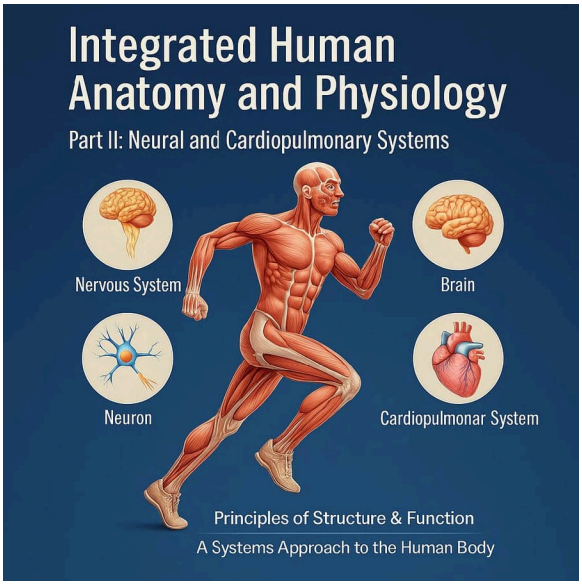
INTEGRATED HUMAN ANATOMY AND PHYSIOLOGY II



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De Anza College

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Lakshmikanta Sengupta and Susmita Sengupta

De Anza College

Thumbnail: Detailed anatomical human figure with exposed muscles, alongside illustrations of cnervous system, brain, neuron, cardiopulmonar system. (AI-generated image created with Nano Banana)

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This text was compiled on 10/13/2026

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Program Page



De Anza College sits in the heart of Silicon Valley, where innovation and opportunity are part of daily life, and also has one of the highest costs of living in the country. For our students, that makes every dollar count, and the price of a textbook can be the difference between staying enrolled or dropping a class. Our Zero Textbook Cost (ZTC) program closes that gap by giving students full access to high-quality, faculty-created course materials at no cost from the first day of class.

De Anza is proud of its diverse student community, and we hold our ZTC materials to the same standard of excellence that defines the college. Faculty across departments write, adapt, and review these resources themselves, keeping them accurate, current, and grounded in the perspectives of the students who use them, with accessibility and inclusive design built in from the start.

This program reflects the time and care our faculty invest in their students. If you have questions, feedback, or come across any issues with a ZTC resource, contact De Anza College's ZTC Grants Coordinator, Shagun Kaur.

Learn more about De Anza ZTC Program [www.deanza.edu/academic-services/oer/]

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Accessibility Statement

Accessibility

De Anza College is dedicated to creating learning materials that are accessible and benefit all students. Our materials follow the Web Content Accessibility Guidelines (WCAG) 2.1 Level AA. Our authors and contracted accessibility specialists checked all resources using this ASCCC OERI checklist, the accessibility tool in LibreTexts, WAVE, JAWS, and other more detailed checks for complex materials such as interactive components.

We selected LibreTexts as our preferred platform because of its commitment to accessibility. In addition to the platform's inherent accessibility, we collaborated with the LibreTexts team to fix any accessibility issues we found.

Our Accessibility Review Process

Accessibility was built into the resource as it was written. This resource went through the following stages of review:

1. **Template check.** The project coordinator and instructional designers checked the first chapter for accessibility as a model to ensure later chapters meet accessibility standards.
2. **Progress checks.** The manuscript was checked at approximately 40%, 60%, and 80% completion to catch and correct accessibility issues in production.
3. **Specialist review.** Once the manuscript was complete, an accessibility expert conducted an accessibility check.
4. **Remediation.** The author and the project manager who conducted the earlier progress checks worked together to remediate the issues identified by the accessibility expert.
5. **Final check.** The resource underwent another full check by the accessibility expert before the book was deemed ready for publication.

This process combines ongoing checks during writing with a final specialist review, so issues are caught early and confirmed fixed before publication.

Report an Accessibility Problem

We are always looking for ways to make our resources more accessible. If you run into a barrier while using this resource, please let us know so we can look into it. When you contact us, it helps to include:

- The name of the resource
- The location of the problem (a web address or page/chapter description)
- A description of the problem you encountered
- The device, browser, and any assistive technology you were using (e.g., Windows 11, Google Chrome, JAWS screen reader)

Contact us: De Anza ZTC team

This statement was last updated August 2026

This resource was project-managed by **Shagun Kaur**, overseeing scope, budget, and schedule; stakeholder coordination via monthly meetings; training and platform support (LibreTexts, troubleshooting, technical support); QA/accessibility check scheduling; and licensing and copyright governance.

A Note on Known Limitations

No accessibility check is perfect. We've made a genuine, sustained effort to catch and fix issues throughout the writing and review process described above, but some barriers may remain, especially in complex or interactive content. We'd rather be upfront about that than claim a standard of perfection we can't guarantee.

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About the Book

About Integrated Human Anatomy and Physiology, Part II

Philosophy and Approach

Integrated Human Anatomy and Physiology, Part II is the second volume in a three-part textbook series developed for undergraduate students in nursing, allied health, medical laboratory technology, physical therapy, respiratory therapy, and pre-medical pathways. The book is designed to support CSU- and UC-transferable coursework and align with Medical Laboratory Technology Associate degree outcomes.

The text uses a student-centered, conversational style, includes intuitive figures, and emphasizes 21st-century critical thinking skills. Its goal is to make complex anatomy and physiology concepts understandable, relevant, and usable for students preparing for a range of health professions.

An equity-focused approach guides the design of the book. Many students using this text are first-generation college students, returning adults, or learners for whom English or scientific language is not their first language. The writing, examples, and learning supports are intended to reduce avoidable barriers, honor diverse experiences, and connect course content to real health and community contexts.

Chapter Organization and Scaffolding

Student-Centered Framework

Integrated Human Anatomy and Physiology, Part II is intentionally structured around the needs and realities of undergraduate students who may be encountering college-level science, medical vocabulary, and high-stakes transfer requirements for the first time. The book is organized into chapters, or modules, that build from orientation to deeper system-level understanding.

Wiring the Body: Basics of the Nervous System

- Anatomical and functional divisions of the nervous system
- Neurons and glial cells as the basic building blocks
- Sensation, integration, and response
- Action potentials and neuronal communication
- Foundational vocabulary and review questions for self-assessment

Central and Peripheral Nervous System

- Support and protection of the brain
- Major brain regions and their functions
- Cranial nerves, spinal cord, and spinal nerves
- Integration of central and peripheral control
- Key terms and review questions for self-assessment

Sensory Information and Coordinated Motor Responses

- General and special senses
- Touch, taste, smell, hearing, balance, and vision
- Somatic integration and motor responses
- Sensory processing and body movement
- Key terms and review questions for self-assessment

The Autonomic Nervous System

- Sympathetic and parasympathetic divisions
- Autonomic synapses, effects, and reflexes
- Central control of autonomic function
- Drugs that affect the autonomic system
- Critical thinking on regulation and imbalance
- Key terms and review questions for self-assessment

The Body's Transport and Defense System

- Overview of blood and its components
- Formation of blood cells
- Erythrocytes, leukocytes, and platelets
- Hemostasis and blood typing
- Key terms and review questions for self-assessment

From Heart Anatomy to Cardiac Output

- Detailed heart anatomy
- Cardiac muscle and electrical activity
- Cardiac cycle and physiology
- Developmental overview of the heart
- Key terms and review questions for self-assessment

Blood Vessels and Circulatory Pathways

- Structure and function of blood vessels
- Blood flow, blood pressure, and resistance
- Capillary exchange and vascular regulation
- Major circulatory pathways
- Fetal circulation and developmental concepts
- Key terms and review questions for self-assessment

The Respiratory System

- Organs and structures of the respiratory system
- Lung anatomy and ventilation
- Mechanics of breathing
- Gas exchange and transport of gases
- Developmental aspects of the respiratory system
- Key terms and review questions for self-assessment

Central Objective

The central objective of the textbook is to support student success by making clear:

- what students are expected to learn and do in each chapter
- why these concepts and skills matter for future courses, clinical practice, and health professions
- how students can tell whether they are meeting expectations and where they may need more practice

Learning Progression

Students are guided through a progression from:

identifying structures → explaining functions → understanding regulation → recognizing homeostatic imbalance → applying knowledge in healthcare contexts

Student-Centered Design Features

To support this progression, the textbook uses several student-centered design features.

Each chapter opens with clearly stated learning objectives written in plain, actionable language. These objectives provide students with a focused roadmap for reading, note-taking, and self-assessment.

Concepts are developed through purpose-driven explanations and examples that connect anatomy and physiology to real-world health, clinical practice, and homeostatic balance.

To support mastery, the text includes transparent criteria for success through review questions, critical thinking prompts, and key terms that align directly with chapter goals. Content is organized in a scaffolded sequence, moving from foundational ideas to more advanced applications and from structure to function to clinical significance. This stepwise progression helps students build confidence and manage complex material in smaller, more manageable parts.

The textbook also provides integrated terminology support in student-friendly language, helping learners build scientific vocabulary and connect terms to observable structures and physiological processes.

Visuals, alternative text descriptions, and accessible formatting further enhance readability and make the material more inclusive for a diverse student audience.

Together, these design choices aim to:

- lower barriers for students with varied academic preparation
- build confidence through clear expectations and frequent, low-stakes opportunities for feedback
- promote durable, transferable skills in anatomical identification, physiological reasoning, scientific communication, and self-directed learning that will support students throughout the full three-part series and into professional training

Integration Across Levels

Integration with the Laboratory Manual

This textbook is designed to function in tandem with a corresponding laboratory manual, especially for sections on the nervous system, circulatory system, and respiratory system. The lecture text provides the conceptual and theoretical foundations for lab activities, preparing students to work confidently with models, microscopes, data, and specimens. It also reinforces lab learning with explanations, diagrams, and consistent terminology.

By aligning readings, figures, and vocabulary with the lab manual, the text supports a cohesive learning experience in which students repeatedly see, name, and explain structures and functions across settings. For example, a student might encounter human heart anatomy first in a figure, then on a model or in a mammalian dissection lab, and later in a case-based question about structure-function relationships. This integration helps connect abstract ideas to concrete observations and supports movement between visual, verbal, and hands-on modes of learning.

Visuals, Accessibility, and Learning Design

Visuals and Images

Visual resources are central to learning anatomy and physiology. Integrated Human Anatomy and Physiology, Part II is designed to make those resources as clear, accurate, and accessible as possible. The book includes high-resolution, clearly labeled diagrams and micrographs, such as anatomical drawings of neurons and action potentials, pulmonary circulation (oxygenation loop) and systemic circulation (delivery loop), the cardiac cycle, conducting and respiratory zones, and the diffusion barrier. It also includes histology images relevant to neurobiology and cardio-respiratory feedback loops and physiological pathways.

These visuals are selected and created to emphasize conceptual clarity and structural accuracy, support transfer-level rigor, and help students connect structure to function and lab to lecture. Whenever possible, the book uses openly licensed Creative Commons visuals and references. This allows instructors and institutions to adapt images, customize them for accessibility, and update content as science and pedagogy evolve.

Accessibility Features

The textbook reflects an accessibility-forward and equity-focused design philosophy. In both print and digital formats, it uses structured headings to support navigation and screen-reader use, readable typography and consistent page layout, and high color contrast for text and key visual elements.

Figures and diagrams are accompanied by meaningful alternative text so that students using screen readers can access the same information as students viewing the images. Hyperlinks are written with descriptive text rather than bare URLs. Tables are simplified, appropriately tagged, and used only when they add clear value. Media are provided with captions and, when applicable, transcripts.

Language throughout the text is as plain as possible without sacrificing scientific accuracy. Explanations and instructions are designed to reduce unnecessary complexity and cognitive load. These choices support students with disabilities, multilingual learners, students reading on mobile devices, and others who may be learning under constrained conditions. They also align with Universal Design for Learning principles and help institutions meet accessibility and accreditation expectations.

By providing zero-cost course materials in accessible formats from the outset, the textbook seeks to promote equitable access and support student persistence and success across diverse backgrounds, learning needs, and life circumstances.

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About the Authors

Lakshmikanta Sengupta



Figure 1: Portrait of coauthor Lakshmikanta Sengupta.

Lakshmikanta Sengupta, Ph.D., is a biology educator with 40 years of experience teaching and mentoring in postsecondary settings. His teaching areas include genetics, biotechnology, human biology, and human anatomy and physiology.

Over four decades in the classroom, he has developed a strong understanding of how undergraduate students think and learn, the misconceptions they often bring to biology, and the explanations and learning activities that best support student success. He views educational institutions as vibrant communities where learning, mentorship, and collaboration can flourish.

Expertise in Biology Education

Dr. Sengupta combines deep disciplinary knowledge with strong teaching practice. He is well suited to author an undergraduate biology textbook because he brings both subject expertise and a student-centered approach to instruction.

His qualifications include:

- broad knowledge of the biological sciences
- familiarity with the history of biology, current debates, and emerging trends
- continued engagement with current scholarship to keep content accurate and relevant
- skill in explaining complex biological ideas in clear, accessible language

He uses engaging narratives, real-world examples, case studies, and carefully designed visuals such as diagrams, charts, and infographics to help students understand unfamiliar material.

Teaching and Learning Approach

Dr. Sengupta's teaching is grounded in evidence-based, student-centered methods. He designs learning activities and assessments that help students monitor their understanding and strengthen scientific reasoning.

Examples include:

- discussion questions

- quizzes
- hands-on activities
- application-focused exercises

He also promotes collaboration among educators and students to share effective practices, improve teaching, and increase the quality and inclusivity of educational materials.

Scholarship of Teaching and Learning

Beyond the classroom, Dr. Sengupta has contributed to the scholarship of teaching and learning in biology. He collaborated with the Community College Biology Faculty Enhancement through Scientific Teaching (CCB FEST), a National Science Foundation-funded program.

He has also coauthored research publications in the following venues:

- Proceedings of the National Academy of Sciences
- CBE—Life Sciences Education

This combination of research engagement and classroom experience informs his textbook authorship and helps ensure that the content is both scientifically rigorous and pedagogically effective.

Broader Interests

Dr. Sengupta's broader interests enrich his teaching with relatable and concrete examples. These interests include:

- macro and micro photography, which highlight the details of natural and anatomical structures
- cooking, which connects biology to nutrition and health
- gardening, which reflects a commitment to sustainability and the natural world

Together, his disciplinary depth, teaching expertise, assessment-driven approach, and long experience working with diverse undergraduate populations support his qualifications as a high-quality author of this undergraduate biology textbook.

Susmita Sengupta



Figure 2: Portrait of coauthor Susmita Sengupta.

Susmita Sengupta, Ph.D., is a scientist and educator who has taught at Bay Area community colleges for nearly two decades. She has more than 29 years of teaching experience at both the undergraduate and graduate levels.

She currently teaches Human Anatomy and Physiology and Human Biology. She has also taught:

- Genetics
- General Biology
- Mammalian Cell Culture
- Gene Expression in Cell Culture

Professional Development and Training

Dr. Sengupta has participated in a number of professional development programs that support effective biology instruction. These include:

- Summer Institute of Functional Genomics and Bioinformatics for Community College Faculty, University of California, Davis (2005)
- Instructional Skills Workshop, De Anza College (2018, 2020)
- RSI training (Winter 2025)

From 2010 to 2014, she was an active participant in CCB FEST (Community College Faculty Enhancement through Scientific Teaching), a National Science Foundation-funded project at San Francisco State University.

Leadership and Service

Dr. Sengupta is currently a member of the Academic Advisory Committee for Explorer's Guide to Biology.

She has also served on several college committees, including:

- Curriculum Committee
- Educational Policies Committee
- Evaluation Committee
- Professional Development Committee

Through this work, she has contributed to program development and instructional quality.

Scholarship and Publications

Dr. Sengupta is the author of more than 26 publications in peer-reviewed journals. She has also developed laboratory manuals to support the courses she teaches.

Her experience in both classroom and laboratory instruction, combined with her long-term engagement in evidence-based teaching and curriculum design, gives her a strong understanding of how students learn biology.

This combination of:

- disciplinary expertise
- pedagogical training
- curriculum experience
- practical teaching experience with diverse student populations

makes her especially well qualified to serve as the author of an undergraduate textbook.

Selected Publications

- 2004: Molecular mechanisms, proteinopathies and therapeutic strategies in neurodegenerative disorders. Current Genomics, 5(4), 327-346.
- 2002: Pharmacogenetic applications of the post genomic era. Current Pharmaceutical Biotechnology, 3(2), 141-150.
- 2001: Bioengineered crops: commercial and ethical considerations. Current Genomics, 2(2), 181-197.
- 1998: Calcium-dependent metabolic regulations in prokaryotes indicate conserved nature of calmodulin gene. Indian Journal

of Experimental Biology, 36(2), 136-147.
- 1997: Phytolectins: the natural molecules with immense biotechnological potential. Indian Journal of Experimental Biology, 35, 11-18.
- 1990: Cytogenetic studies in human populations exposed to gas leak in Bhopal, India. Environmental Health Perspectives, 86, 323-326.

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1: WIRING THE BODY - BASICS OF THE NERVOUS SYSTEM

Chapter Overview

You wake to your alarm, hit snooze without opening your eyes, smell coffee, read a text, and jerk your hand away from a too-hot mug. In just a few moments, your body has detected sound, light, smell, touch, temperature, and pain; you have made decisions, moved muscles, and felt emotions. All of this depends on billions of nerve and support cells working together — rapidly and silently — to collect information, process it, and send out commands.



Figure 1.1: As the neural circuitry of the nervous system has become more fully understood and robotics more sophisticated, it is now possible to integrate technology with the body and restore abilities following traumatic events. At some point in the future, will this type of technology lead to the ability to augment our nervous systems? (Public Domain; via Wikimedia Commons).

This chapter introduces that control network. You will meet the cells that make up nervous tissue, see how they are organized into the central and peripheral nervous systems, and learn how electrical and chemical signals underlie thoughts, memories, movements, and feelings. You will also begin to see how this fast, wired system of control works alongside the slower, hormone-based endocrine system.

The Nervous System and Homeostasis

We will start by placing the nervous system in its homeostatic context. The body must constantly monitor and adjust internal conditions such as temperature, blood pressure, and pH. You will review the basic principles of homeostasis, including negative and positive feedback, and then compare how the nervous and endocrine systems each contribute to maintaining stability. The nervous system uses rapid electrical signals and specific synapses; the endocrine system uses hormones carried in the blood. Understanding where they are similar and where they differ—mechanisms of action, target tissues, speed, and duration of effect—will help you make sense of later chapters on individual organ systems.

Microscopic Anatomy of Nervous Tissue

Next, you will examine the microscopic anatomy of nervous tissue. You will learn to recognize neurons and various types of neuroglia on histology and to relate form to function. You will distinguish neurons, neuroglia, nerves, ganglia, and synapses under the microscope, and compare unipolar, bipolar, and multipolar neurons in terms of structure, typical location, and role. You will survey the major glial cell types (such as astrocytes, oligodendrocytes, Schwann cells, and microglia) and how they support, protect, and insulate neurons. You will also describe myelination, how it alters signal conduction, and how damage to myelin

contributes to selected neurological diseases, and consider the limited regenerative capacity of nervous tissue and why injury to the CNS is often permanent, whereas some PNS neurons can regenerate.

From Cells to Systems: Organization of the Nervous System

You will then zoom out from cells to systems and learn how the nervous system is organized structurally and functionally. Structurally, you will categorize all components into the central nervous system (CNS)—the brain and spinal cord—and the peripheral nervous system (PNS)—all nervous tissue outside the CNS. Functionally, you will differentiate the somatic and autonomic nervous systems and the types of effectors they control, separate sensory (afferent) pathways, which bring information into the CNS, from motor (efferent) pathways, which carry commands out, and contrast the sympathetic and parasympathetic divisions of the autonomic nervous system in terms of anatomy and “fight-or-flight” versus “rest-and-digest” functions.

Neurophysiology: How Neurons Send and Receive Signals

Finally, the chapter focuses on neurophysiology—how neurons generate and transmit electrical signals and how they communicate at synapses. You will learn how the resting membrane potential is established and maintained, including the critical role of the Na⁺/K⁺ pump and ion channels. You will distinguish graded potentials, threshold, and action potentials, and understand where each occurs and how they differ in size, duration, and function. You will follow the steps of action potential generation and propagation along an axon, and see how myelination allows for rapid saltatory conduction. You will examine the sequence of events in synaptic transmission, from arrival of the impulse at the presynaptic terminal, to neurotransmitter release, receptor binding, and postsynaptic responses, and consider how diseases, toxins, and drugs can interfere with or enhance these processes.

Bringing It All Together: Structure, Function, and Body Regulation

By the end of this chapter, you should be able to integrate structure and function - to look at a neuron or a pathway, predict how signals will travel, and explain how the nervous system and endocrine system work together to regulate the body functions. This foundation will be essential as you move into later chapters on the brain, spinal cord, sensory systems, and motor control.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- Describe how the nervous system contributes to the body’s overall control and homeostatic mechanisms, and compare and contrast its methods of communication, speed, specificity, and duration of action with those of the endocrine system.
- Identify the major cell types in nervous tissue (neurons and the principal classes of neuroglia in the CNS and PNS), relate key structural features of each to their functions, and recognize these cells and basic nervous tissue organization (gray vs. white matter, nerves vs. ganglia) in simple histological images.
- Distinguish between the central nervous system (CNS) and peripheral nervous system (PNS), and describe the major subdivisions and pathways of the nervous system in functional terms (somatic vs. autonomic; sympathetic vs. parasympathetic; sensory/afferent vs. motor/efferent), including their primary effectors and general roles.
- Describe, compare, and contrast graded potentials, threshold, and action potentials in terms of where they occur, how they are generated, and how they differ in amplitude, duration, and function; and explain, step by step, how neurons communicate at chemical synapses, including excitatory and inhibitory postsynaptic potentials and summation.
- Predict how alterations in nervous tissue structure—such as demyelination, axonal injury, or loss of specific neuron populations—disrupt normal signal conduction, and correlate these changes with selected clinical conditions affecting the CNS and PNS.
- Categorize key neurotransmitters by major chemical class (e.g., amino acids, biogenic amines, peptides) and summarize their predominant excitatory or inhibitory effects and general functional roles in the nervous system.

1.1: Anatomical and Functional Divisions of the Nervous System
1.2: Neurons and Glial Cells- Building Blocks of the Nervous System
1.3: Sensation, Integration, and Response
1.4: The Action Potential
1.5: Communication Between Neurons
1.6: Terms and Definitions
1.7: Chapter Review Questions
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1.1: Anatomical and Functional Divisions of the Nervous System

The Central and Peripheral Nervous Systems

The nervous system is more than just “brain and spinal cord.” Although these are the two main organs, each contains many specialized regions with distinct functions that often look similar and can only be distinguished with microscopes or electrical recordings. In this way, the nervous system is like a collection of many tightly integrated “mini-organs,” unlike systems such as the digestive system, where organs like the stomach and liver are clearly different to the eye.

For an overview, the nervous system is usually divided into two main parts (Figure 1.1.1):

- the central nervous system (CNS), which includes the brain and spinal cord located within the skull and vertebral column, and
- the peripheral nervous system (PNS), which includes all nervous tissue outside the CNS.

This CNS–PNS division is a useful starting point, even though some peripheral structures lie within the skull or spine and the exact boundary can vary depending on what aspect of the system is being described.

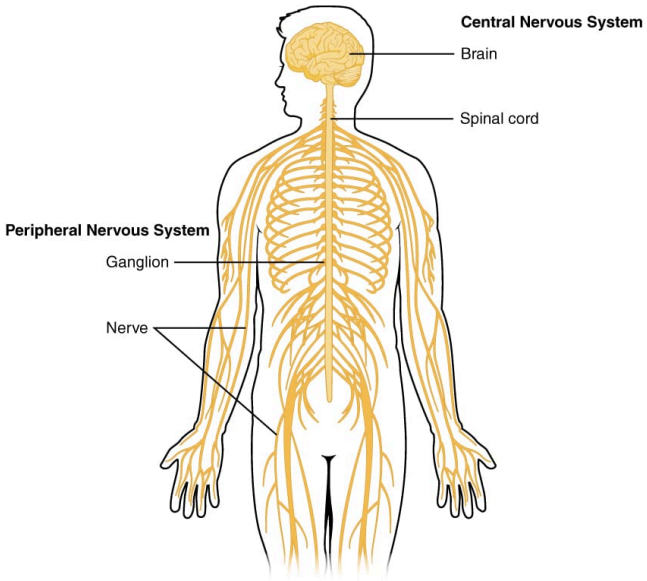


Figure 1.1.1: The structures of the PNS are referred to as ganglia and nerves, which can be seen as distinct structures. The equivalent structures in the CNS are not obvious from this overall perspective and are best examined in prepared tissue under the microscope. (CC BY 4.0; via Basic Structures and Function of the Nervous System). Alternate text description of the Figure 1.1.1.

Nervous tissue in both the CNS and PNS contains two main cell types: neurons and glial cells. Glial cells support and protect neurons. Neurons are the primary signaling cells: each has a cell body (soma), branching dendrites that receive input, and a single axon that carries signals to a target.

Within nervous tissue, areas rich in neuron cell bodies and dendrites are called gray matter, whereas areas dominated by myelinated axons are called white matter. White matter looks pale because myelin is lipid-rich; gray matter appears darker by comparison. This gray – white distinction is especially evident in large CNS structures such as the brain and spinal cord (Figure 1.1.2).

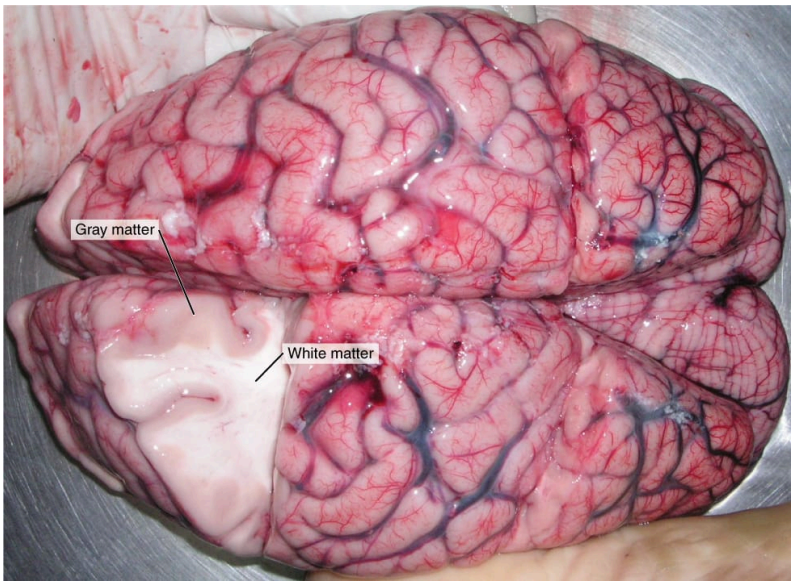


Figure 1.1.2: Autopsy brain with a partial coronal section removed, revealing a pale central region of white matter surrounded by a darker outer layer of gray matter forming the cerebral cortex. The tissue is fresh and unstained: the white matter appears light because of lipid-rich myelin around axons, while the gray matter looks darker by comparison, reflecting its higher density of neuron cell bodies and dendrites. (CC BY 4.0; via Basic Structures and Function of the Nervous System). Alternate text description of the Figure 1.1.2.

Regardless of the appearance of stained or unstained tissue, the cell bodies of neurons or axons can be located in discrete anatomical structures that need to be named. Those names are specific to whether the structure is central or peripheral. A localized collection of neuron cell bodies in the CNS is referred to as a nucleus. In the PNS, a cluster of neuron cell bodies is referred to as a ganglion. Figure 1.1.3 indicates how the term nucleus has a few different meanings within anatomy and physiology. It is the center of an atom, where protons and neutrons are found; it is the center of a cell, where the DNA is found; and it is a center of some function in the CNS. There is also a potentially confusing use of the word ganglion (plural = ganglia) that has a historical explanation. In the central nervous system, there is a group of nuclei that are connected together and were once called the basal ganglia before “ganglion” became accepted as a description for a peripheral structure. Some sources refer to this group of nuclei as the “basal nuclei” to avoid confusion.

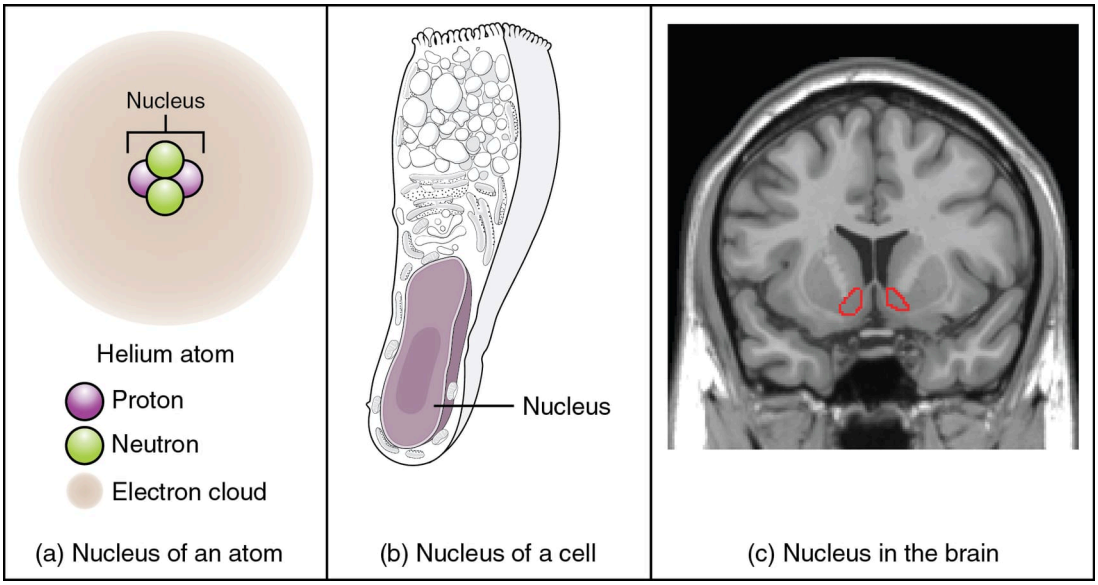


Figure 1.1.3: (a) The nucleus of an atom contains its protons and neutrons. (b) The nucleus of a cell is the organelle that contains DNA. (c) A nucleus in the CNS is a localized center of function with the cell bodies of several neurons, shown here circled in red. (CC BY 4.0; via Basic Structures and Function of the Nervous System). Alternate text description of the Figure 1.1.3.

Terminology applied to bundles of axons also differs depending on location. A bundle of axons, or fibers, found in the CNS is

called a tract whereas the same thing in the PNS would be called a nerve. There is an important point to make about these terms, which is that they can both be used to refer to the same bundle of axons. When those axons are in the PNS, the term is nerve, but if they are CNS, the term is tract. The most obvious example of this is the axons that project from the retina into the brain. Those axons are called the optic nerve as they leave the eye, but when they are inside the cranium, they are referred to as the optic tract. There is a specific place where the name changes, which is the optic chiasm, but they are still the same axons (Figure 1.1.4). In the PNS, they are called the optic nerve, and in the CNS, they are the optic tract. Table 1.1.1 helps to clarify which of these terms apply to the central or peripheral nervous systems.

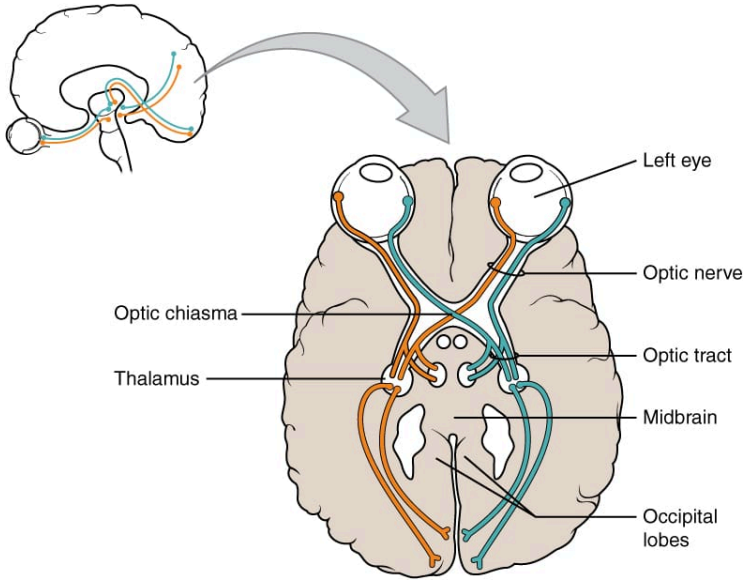


Figure 1.1.4: This drawing of the connections of the eye to the brain shows the optic nerve extending from the eye to the chiasm, where the structure continues as the optic tract. The same axons extend from the eye to the brain through these two bundles of fibers, but the chiasm represents the border between peripheral and central. (CC BY 4.0; via Basic Structures and Function of the Nervous System). Alternate text description of the Figure 1.1.4.

Table 1.1.1: Structures of the CNS and PNS

Structure Type	Central Nervous System (CNS)	Peripheral Nervous System (PNS)
Group of Neuron Cell Bodies (i.e., gray matter)	Nucleus	Ganglion
Bundle of Axons (i.e., white matter)	Tract	Nerve

Functional Divisions of the Nervous System

The nervous system can also be divided on the basis of its functions, but anatomical divisions and functional divisions are different. The CNS and the PNS both contribute to the same functions, but those functions can be attributed to different regions of the brain (such as the cerebral cortex or the hypothalamus) or to different ganglia in the periphery. The problem with trying to fit functional differences into anatomical divisions is that sometimes the same structure can be part of several functions. For example, the optic nerve carries signals from the retina that are either used for the conscious perception of visual stimuli, which takes place in the cerebral cortex, or for the reflexive responses of smooth muscle tissue that are processed through the hypothalamus.

There are two ways to consider how the nervous system is divided functionally. First, the basic functions of the nervous system are sensation, integration, and response. Secondly, control of the body can be somatic or autonomic—divisions that are largely defined by the structures that are involved in the response. There is also a region of the peripheral nervous system that is called the enteric nervous system that is responsible for a specific set of the functions within the realm of autonomic control related to gastrointestinal functions.

Basic Functions

The nervous system has three basic functions: sensation, integration, and response. Sensation is the process of receiving

information about the environment, either outside the body (such as light, sound, touch, chemicals for taste and smell) or inside the body (such as organ stretch or ion concentrations). Sensory receptors detect changes in conditions, called stimuli, and send this information into the nervous system. Integration is the processing of that incoming sensory information within the nervous system. New sensory input is compared and combined with other sensations, past experiences, memories, emotional state, and learning (cognition) to decide what to do. Regions that perform this processing are often called integration or association areas. The process of integration combines sensory perceptions and higher cognitive functions such as memories, learning, and emotion to produce a response. Response is the production of an effect based on the integrated information. The nervous system directs muscles to contract (skeletal, cardiac, or smooth) and glands to secrete substances. Some responses are voluntary and under conscious control, such as moving skeletal muscles, while others are involuntary, such as regulating heart rate, blood vessel diameter, digestion, or sweating.

Together, sensation, integration, and response allow the nervous system to detect changes, make decisions, and act to maintain homeostasis and interact with the environment. Seeing a baseball pitched to a batter will not automatically cause the batter to swing. The trajectory of the ball and its speed will need to be considered. Maybe the count is three balls and one strike, and the batter wants to let this pitch go by in the hope of getting a walk to first base. Or maybe the batter's team is so far ahead, it would be fun to just swing away.

Controlling the Body

The nervous system can be divided into two parts mostly on the basis of a functional difference in responses. The somatic nervous system (SNS) is responsible for conscious perception and voluntary motor responses. Voluntary motor response means the contraction of skeletal muscle, but those contractions are not always voluntary in the sense that you have to want to perform them. Some somatic motor responses are reflexes, and often happen without a conscious decision to perform them. If your friend jumps out from behind a corner and yells "Boo!" you will be startled and you might scream or leap back. You didn't decide to do that, and you may not have wanted to give your friend a reason to laugh at your expense, but it is a reflex involving skeletal muscle contractions. Other motor responses become automatic (in other words, unconscious) as a person learns motor skills (referred to as "habit learning" or "procedural memory").

The autonomic nervous system (ANS) is responsible for involuntary control of the body, usually for the sake of homeostasis (regulation of the internal environment). Sensory input for autonomic functions can be from sensory structures tuned to external or internal environmental stimuli. The motor output extends to smooth and cardiac muscle as well as glandular tissue. The role of the autonomic system is to regulate the organ systems of the body, which usually means to control homeostasis. Sweat glands, for example, are controlled by the autonomic system. When you are hot, sweating helps cool your body down. That is a homeostatic mechanism. But when you are nervous, you might start sweating also. That is not homeostatic, it is the physiological response to an emotional state.

There is another division of the nervous system that describes functional responses. The enteric nervous system (ENS) is responsible for controlling the smooth muscle and glandular tissue in your digestive system. It is a large part of the PNS, and is not dependent on the CNS. It is sometimes valid, however, to consider the enteric system to be a part of the autonomic system because the neural structures that make up the enteric system are a component of the autonomic output that regulates digestion. There are some differences between the two, but for our purposes here there will be a good bit of overlap. See Figure for examples of where these divisions of the nervous system can be found.

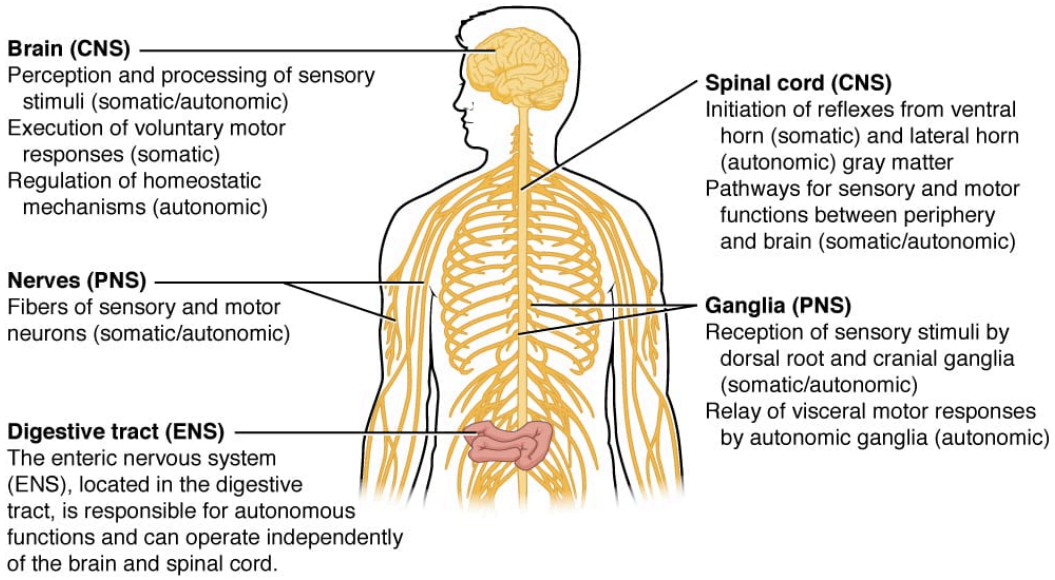


Figure 1.1.5: Somatic structures include the spinal nerves, both motor and sensory fibers, as well as the sensory ganglia (posterior root ganglia and cranial nerve ganglia). Autonomic structures are found in the nerves also, but include the sympathetic and parasympathetic ganglia. The enteric nervous system includes the nervous tissue within the organs of the digestive tract. (CC BY 4.0; via Basic Structures and Function of the Nervous System). Alternate text description of the Figure 1.1.5.

Everyday Connection: How Much of Your Brain Do You Use?

Have you ever heard the claim that humans only use 10 percent of their brains? Maybe you have seen an advertisement on a website saying that there is a secret to unlocking the full potential of your mind—as if there were 90 percent of your brain sitting idle, just waiting for you to use it. If you see an ad like that, don't click. It isn't true. Humans use virtually all of their brain, not just 10%. The idea that we only use 10% of our brain is a persistent myth with no scientific basis.

An easy way to see how much of the brain a person uses is to take measurements of brain activity while performing a task. An example of this kind of measurement is functional magnetic resonance imaging (fMRI), which generates a map of the most active areas and can be generated and presented in three dimensions (Figure 1.1.6). This procedure is different from the standard MRI technique because it is measuring changes in the tissue in time with an experimental condition or event.

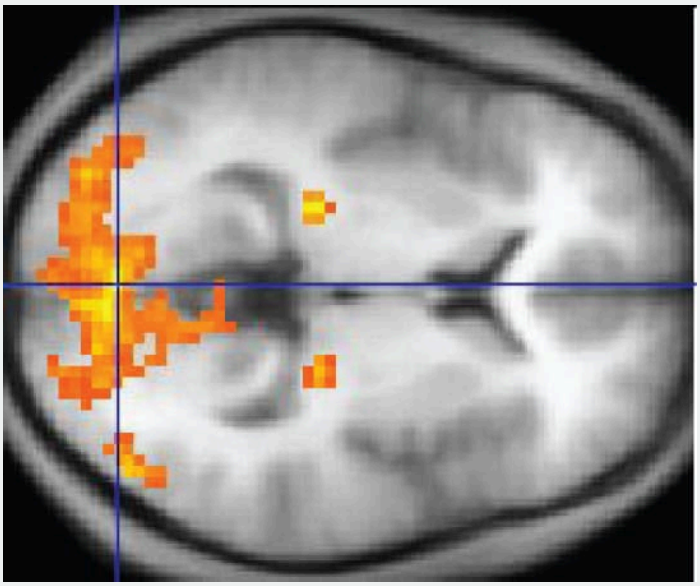


Figure 1.1.6: This functional magnetic resonance imaging (fMRI) shows activation of the visual cortex in response to visual stimuli. (CC BY 4.0; via Basic Structures and Function of the Nervous System). Alternate text description of the Figure 1.1.6.

The underlying assumption is that active nervous tissue will have greater blood flow. By having the subject perform a visual task, activity all over the brain can be measured. Consider this possible experiment: the subject is told to look at a screen with a black dot in the middle (a fixation point). A photograph of a face is projected on the screen away from the center. The subject has to look at the photograph and decipher what it is. The subject has been instructed to push a button if the photograph is of someone they recognize. The photograph might be of a celebrity, so the subject would press the button, or it might be of a random person unknown to the subject, so the subject would not press the button.

In this task, visual sensory areas would be active, integrating areas would be active, motor areas responsible for moving the eyes would be active, and motor areas for pressing the button with a finger would be active. Those areas are distributed all around the brain and the fMRI images would show activity in more than just 10 percent of the brain (some evidence suggests that about 80 percent of the brain is using energy—based on blood flow to the tissue—during well-defined tasks similar to the one suggested above). This task does not even include all of the functions the brain performs. There is no language response, the body is mostly lying still in the MRI machine, and it does not consider the autonomic functions that would be ongoing in the background.

Brain damage to any area usually impairs function, proving no region is "unused. The brain consumes 20% of the body's energy despite being only 2% of body weight. Evolution would have eliminated unused tissue if it existed.

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1.2: Neurons and Glial Cells- Building Blocks of the Nervous System

Neurons

Nervous tissue is composed of two types of cells, neurons and glial cells. Neurons are the primary type of cell that most anyone associates with the nervous system. They are responsible for the computation and communication that the nervous system provides. They are electrically active and release chemical signals to target cells. Glial cells, or glia, are known to play a supporting role for nervous tissue. Ongoing research pursues an expanded role that glial cells might play in signaling, but neurons are still considered the basis of this function. Neurons are important, but without glial support they would not be able to perform their function.

Neurons are the cells considered to be the basis of nervous tissue. They are responsible for the electrical signals that communicate information about sensations, and that produce movements in response to those stimuli, along with inducing thought processes within the brain. An important part of the function of neurons is in their structure, or shape. The three-dimensional shape of these cells makes the immense numbers of connections within the nervous system possible.

Parts of a Neuron

As you learned in the first section, the main part of a neuron is the cell body, which is also known as the soma (soma = "body"). The cell body contains the nucleus and most of the major organelles. But what makes neurons special is that they have many extensions of their cell membranes, which are generally referred to as processes. Neurons are usually described as having one, and only one, axon—a fiber that emerges from the cell body and projects to target cells. That single axon can branch repeatedly to communicate with many target cells. It is the axon that propagates the nerve impulse, which is communicated to one or more cells. The other processes of the neuron are dendrites, which receive information from other neurons at specialized areas of contact called synapses. The dendrites are usually highly branched processes, providing locations for other neurons to communicate with the cell body. Information flows through a neuron from the dendrites, across the cell body, and down the axon. This gives the neuron a polarity—meaning that information flows in this one direction. Figure 1.2.1 shows the relationship of these parts to one another.

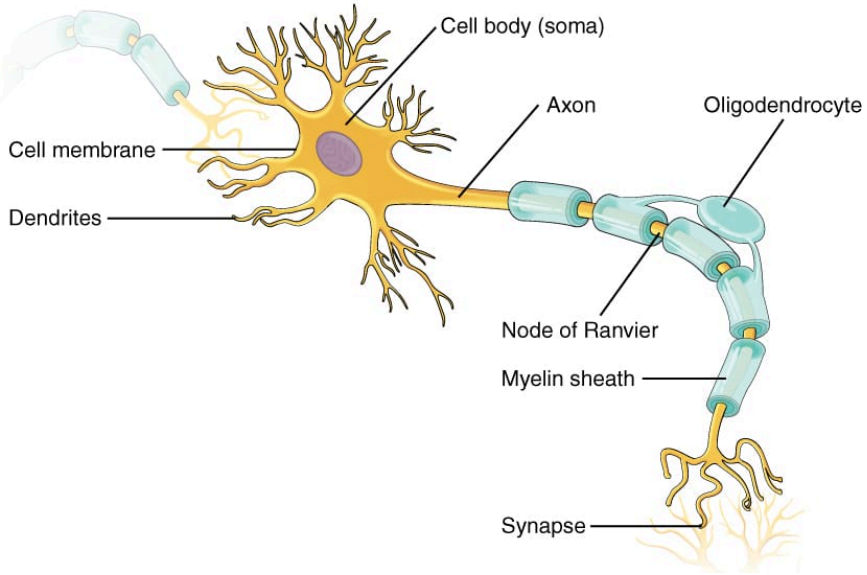


Figure 1.2.1: The major parts of the neuron are labeled on a multipolar neuron from the CNS. (CC BY 4.0; via Nervous Tissue). Alternate text description of the Figure 1.2.1.

Where the axon emerges from the cell body, there is a special region referred to as the axon hillock. This is a tapering of the cell body toward the axon fiber. Within the axon hillock, the cytoplasm changes to a solution of limited components called axoplasm.

Because the axon hillock represents the beginning of the axon, it is also referred to as the initial segment.

A neuron may or may not be myelinated. Many axons are wrapped in a specialized insulating material called myelin, produced by glial cells (oligodendrocytes in the CNS and Schwann cells in the PNS). You can imagine myelin as the biological equivalent of the plastic coating around an electrical wire: it prevents “current” from leaking out and allows the signal to travel faster and farther. Unlike the continuous insulation on most wires, however, myelin is arranged in segments along the axon, with tiny exposed gaps in between. Each gap, called a node of Ranvier, is a critical “booster station” where ions can move across the membrane and regenerate the action potential, allowing the signal to jump rapidly from node to node (saltatory conduction). The myelin-wrapped regions between nodes are the axon segments. At the far end of the axon, it branches into multiple axon terminals, and each branch ends in a swollen synaptic end bulb. These end bulbs are the “plugs” that connect the neuron to its target cell at a synapse, where electrical signals are translated into chemical messages that influence the next cell in the circuit.

Types of Neurons

There are many neurons in the nervous system—a number in the trillions. And there are many different types of neurons. They can be classified by many different criteria. The first way to classify them is by the number of processes attached to the cell body. Using the standard model of neurons, one of these processes is the axon, and the rest are dendrites. Because information flows through the neuron from dendrites or cell bodies toward the axon, these names are based on the neuron's polarity (Figure 1.2.2).

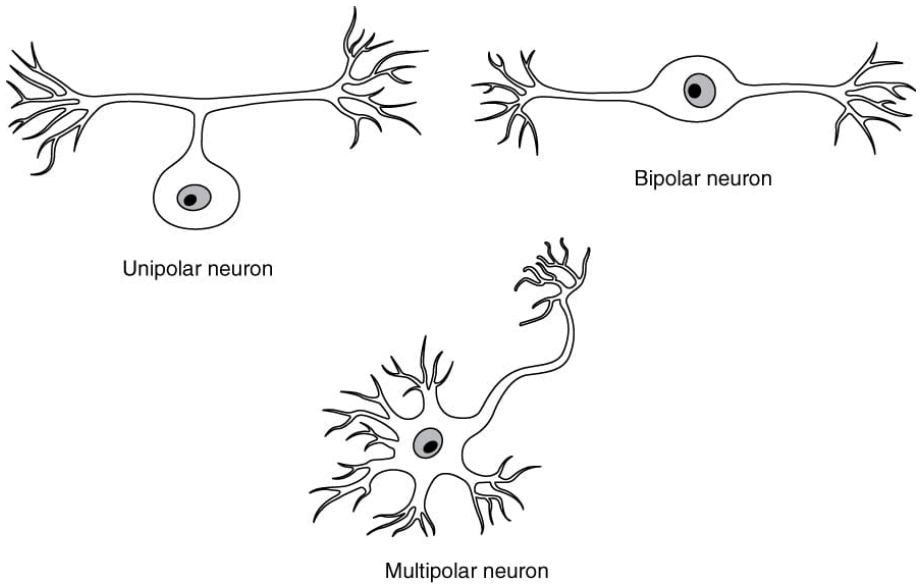


Figure 1.2.2: Unipolar cells have one process that includes both the axon and dendrite. Bipolar cells have two processes, the axon and a dendrite. Multipolar cells have more than two processes, the axon and two or more dendrites. (CC BY 4.0; via Nervous Tissue). Alternate text description of the Figure 1.2.2.

Unipolar cells have only one process emerging from the cell. True unipolar cells are only found in invertebrate animals, so the unipolar cells in humans are more appropriately called “pseudo-unipolar” cells. Invertebrate unipolar cells do not have dendrites. Human unipolar cells have an axon that emerges from the cell body, but it splits so that the axon can extend along a very long distance. At one end of the axon are dendrites, and at the other end, the axon forms synaptic connections with a target. Unipolar cells are exclusively sensory neurons and have two unique characteristics. First, their dendrites are receiving sensory information, sometimes directly from the stimulus itself. Secondly, the cell bodies of unipolar neurons are always found in ganglia. Sensory reception is a peripheral function (those dendrites are in the periphery, perhaps in the skin) so the cell body is in the periphery, though closer to the CNS in a ganglion. The axon projects from the dendrite endings, past the cell body in a ganglion, and into the central nervous system.

Bipolar cells have two processes, which extend from each end of the cell body, opposite to each other. One is the axon and one the dendrite. Bipolar cells are not very common. They are found mainly in the olfactory epithelium (where smell stimuli are sensed), and as part of the retina.

Multipolar neurons are all of the neurons that are not unipolar or bipolar. They have one axon and two or more dendrites (usually many more). With the exception of the unipolar sensory ganglion cells, and the two specific bipolar cells mentioned above, all

other neurons are multipolar. Some cutting edge research suggests that certain neurons in the CNS do not conform to the standard model of “one, and only one” axon. Some sources describe a fourth type of neuron, called an anaxonic neuron. The name suggests that it has no axon (an- = “without”), but this is not accurate. Anaxonic neurons are very small, and if you look through a microscope at the standard resolution used in histology (approximately 400 times to 1000 times total magnification), you will not be able to distinguish any process specifically as an axon or a dendrite. Any of those processes can function as an axon depending on the conditions at any given time. Nevertheless, even if they cannot be easily seen, and one specific process is definitively the axon, these neurons have multiple processes and are therefore multipolar.

Neurons can also be classified on the basis of where they are found, who found them, what they do, or even what chemicals they use to communicate with each other. Some neurons referred to in this section on the nervous system are named on the basis of those sorts of classifications (Figure 1.2.3). For example, a multipolar neuron that has a very important role to play in a part of the brain called the cerebellum is known as a Purkinje (commonly pronounced per-KIN-gee) cell. It is named after the anatomist who discovered it (Jan Evangelista Purkinje, 1787–1869).

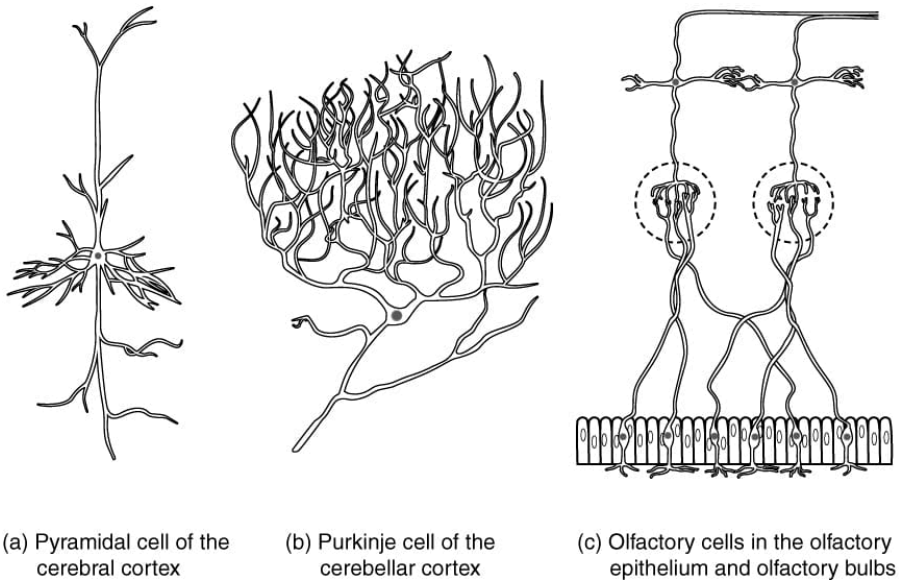


Figure 1.2.3: Three examples of neurons that are classified on the basis of other criteria. (a) The pyramidal cell is a multipolar cell with a cell body that is shaped something like a pyramid. (b) The Purkinje cell in the cerebellum was named after the scientist who originally described it. (c) Olfactory neurons are named for the functional group with which they belong. (CC BY 4.0; via Nervous Tissue). Alternate text description of the Figure 1.2.3.

Glial Cells

Glial cells, or neuroglia or simply glia, are the other type of cell found in nervous tissue. They are considered to be supporting cells, and many functions are directed at helping neurons complete their function for communication. The name glia comes from the Greek word that means “glue,” and was coined by the German pathologist Rudolph Virchow, who wrote in 1856: “This connective substance, which is in the brain, the spinal cord, and the special sense nerves, is a kind of glue (neuroglia) in which the nervous elements are planted.” Today, research into nervous tissue has shown that there are many deeper roles that these cells play. And research may find much more about them in the future.

There are six types of glial cells. Four of them are found in the CNS and two are found in the PNS. Table 1.2.1 outlines some common characteristics and functions.

Table 1.2.1: Glial Cell Types by Location and Basic Function

CNS glia	PNS glia	Basic function
Astrocyte	Satellite cell	Support
Oligodendrocyte	Schwann cell	Insulation, myelination
Microglia	No data available	Immune surveillance and phagocytosis

CNS glia	PNS glia	Basic function
Ependymal cell	No data available	Creating CSF

Glial Cells of the CNS

One cell providing support to neurons of the CNS is the astrocyte, so named because it appears to be star-shaped under the microscope (astro- = “star”). Astrocytes have many processes extending from their main cell body (not axons or dendrites like neurons, just cell extensions). Those processes extend to interact with neurons, blood vessels, or the connective tissue covering the CNS that is called the pia mater (Figure 1.2.4). Generally, they are supporting cells for the neurons in the central nervous system. Some ways in which they support neurons in the central nervous system are by maintaining the concentration of chemicals in the extracellular space, removing excess signaling molecules, reacting to tissue damage, and contributing to the blood-brain barrier (BBB). The blood-brain barrier is a physiological barrier that keeps many substances that circulate in the rest of the body from getting into the central nervous system, restricting what can cross from circulating blood into the CNS. Nutrient molecules, such as glucose or amino acids, can pass through the BBB, but other molecules cannot. This actually causes problems with drug delivery to the CNS. Pharmaceutical companies are challenged to design drugs that can cross the BBB as well as have an effect on the nervous system.

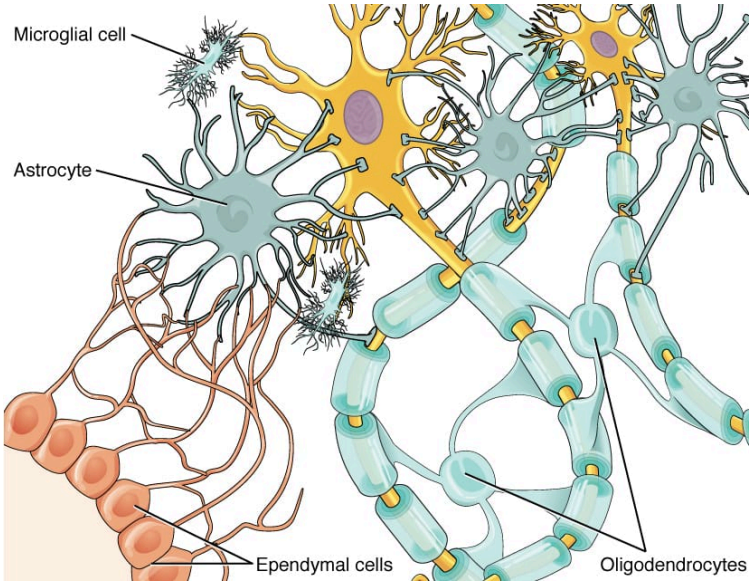


Figure 1.2.4: The CNS has astrocytes, oligodendrocytes, microglia, and ependymal cells that support the neurons of the CNS in several ways. (CC BY 4.0; via Nervous Tissue). Alternate text description of the Figure 1.2.4.

Like a few other parts of the body, the brain has a privileged blood supply. Very little can pass through by diffusion. Most substances that cross the wall of a blood vessel into the CNS must do so through an active transport process. Because of this, only specific types of molecules can enter the CNS. Glucose—the primary energy source—is allowed, as are amino acids. Water and some other small particles, like gases and ions, can enter. But most everything else cannot, including white blood cells, which are one of the body’s main lines of defense. While this barrier protects the CNS from exposure to toxic or pathogenic substances, it also keeps out the cells that could protect the brain and spinal cord from disease and damage. The BBB also makes it harder for pharmaceuticals to be developed that can affect the nervous system. Aside from finding efficacious substances, the means of delivery is also crucial.

Also found in CNS tissue is the oligodendrocyte, sometimes called just “oligo,” which is the glial cell type that insulates axons in the CNS. The name means “cell of a few branches” (oligo- = “few”; dendro- = “branches”; -cyte = “cell”). There are a few processes that extend from the cell body. Each one reaches out and surrounds an axon to insulate it in myelin. One oligodendrocyte will provide the myelin for multiple axon segments, either for the same axon or for separate axons. The function of myelin will be discussed below.

Microglia are, as the name implies, smaller than most of the other glial cells. Ongoing research into these cells, although not entirely conclusive, suggests that they may originate as white blood cells, called macrophages, that become part of the CNS during early development. While their origin is not conclusively determined, their function is related to what macrophages do in

the rest of the body. When macrophages encounter diseased or damaged cells in the rest of the body, they ingest and digest those cells or the pathogens that cause disease. Microglia are the cells in the CNS that can do this in normal, healthy tissue, and they are therefore also referred to as CNS-resident macrophages.

The ependymal cell is a glial cell that filters blood to make cerebrospinal fluid (CSF), the fluid that circulates through the CNS. Because of the privileged blood supply inherent in the BBB, the extracellular space in nervous tissue does not easily exchange components with the blood. Ependymal cells line each ventricle, one of four central cavities that are remnants of the hollow center of the neural tube formed during the embryonic development of the brain. The choroid plexus is a specialized structure in the ventricles where ependymal cells come in contact with blood vessels and filter and absorb components of the blood to produce cerebrospinal fluid. Because of this, ependymal cells can be considered a component of the BBB, or a place where the BBB breaks down. These glial cells appear similar to epithelial cells, making a single layer of cells with little intracellular space and tight connections between adjacent cells. They also have cilia on their apical surface to help move the CSF through the ventricular space. The relationship of these glial cells to the structure of the CNS is seen in Figure 1.2.4.

Glial Cells of the PNS

One of the two types of glial cells found in the PNS is the satellite cell. Satellite cells are found in sensory and autonomic ganglia, where they surround the cell bodies of neurons. This accounts for the name, based on their appearance under the microscope. They provide support, performing similar functions in the periphery as astrocytes do in the CNS—except, of course, for establishing the BBB.

The second type of glial cell is the Schwann cell, which insulate axons with myelin in the periphery. Schwann cells are different than oligodendrocytes, in that a Schwann cell wraps around a portion of only one axon segment and no others. Oligodendrocytes have processes that reach out to multiple axon segments, whereas the entire Schwann cell surrounds just one axon segment. The nucleus and cytoplasm of the Schwann cell are on the edge of the myelin sheath. The relationship of these two types of glial cells to ganglia and nerves in the PNS is seen in Figure 1.2.5.

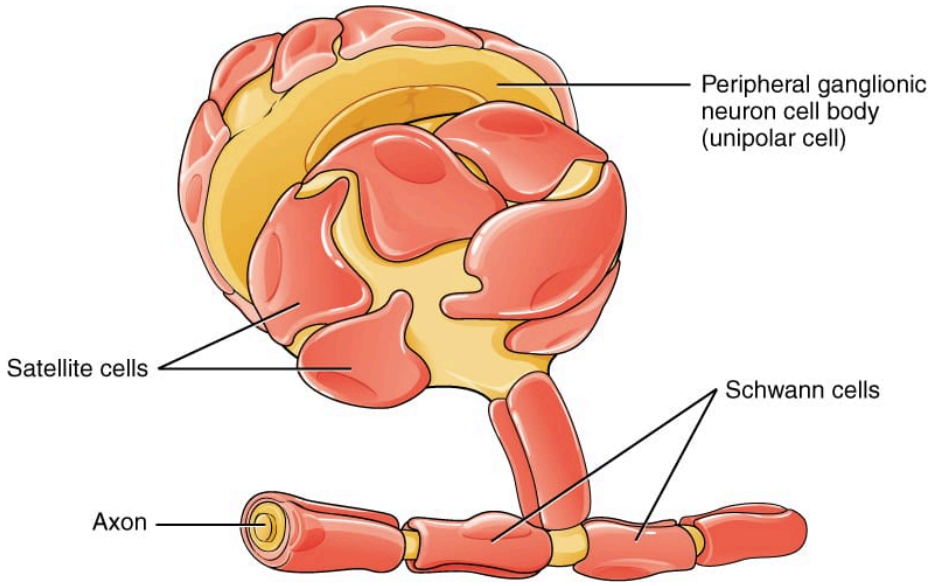


Figure 1.2.5: The PNS has satellite cells and Schwann cells. (CC BY 4.0; via Nervous Tissue). Alternate text description of the Figure 1.2.5.

Myelin

Myelin is a lipid-rich sheath that surrounds the axon and by doing so creates a myelin sheath that facilitates the transmission of electrical signals along the axon. The lipids are essentially the phospholipids of the glial cell membrane. Myelin, however, is more than just the membrane of the glial cell. It also includes important proteins that are integral to that membrane. Some of the proteins help to hold the layers of the glial cell membrane closely together.

The insulation for axons in the nervous system is provided by glial cells, oligodendrocytes in the CNS, and Schwann cells in the PNS. In the CNS and PNS, myelin is like having two different manufacturers making parts for the same kind of high-speed cable.

In the brain and spinal cord, oligodendrocytes act like a central factory that can reach out and insulate many separate axons at once, whereas in peripheral nerves, the appearance of the myelin sheath (by the Schwann cells) can be compared to beads or rings of material wrapped around a string (axon), with short bare stretches of string in between.

The glial cell is wrapped around the axon several times with little to no cytoplasm between the glial cell layers. For oligodendrocytes, the rest of the cell is separate from the myelin sheath as a cell process extends back toward the cell body. A few other processes provide the same insulation for other axon segments in the area. For Schwann cells, the outermost layer of the cell membrane contains cytoplasm and the nucleus of the cell as a bulge on one side of the myelin sheath. During development, the glial cell is loosely or incompletely wrapped around the axon (Figure 1.2.6a). The edges of this loose enclosure extend toward each other, and one end tucks under the other. The inner edge wraps around the axon, creating several layers, and the other edge closes around the outside so that the axon is completely enclosed.

Myelin sheaths can extend for one or two millimeters, depending on the diameter of the axon. Axon diameters can be as small as 1 to 20 micrometers. Because a micrometer is 1/1000 of a millimeter, this means that the length of a myelin sheath can be 100–1000 times the diameter of the axon. Figure 1.2.1, Figure 1.2.4, and Figure 1.2.5 show the myelin sheath surrounding an axon segment, but are not to scale. If the myelin sheath were drawn to scale, the neuron would have to be immense—possibly covering an entire wall of the room in which you are sitting.

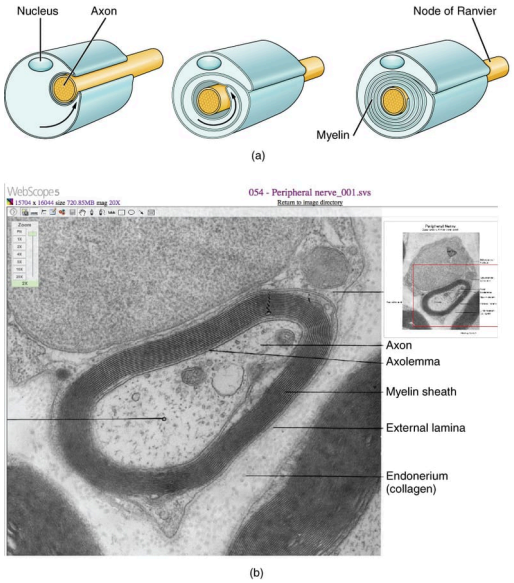


Figure 1.2.6: Myelinating glia wrap several layers of cell membrane around the cell membrane of an axon segment. A single Schwann cell insulates a segment of a peripheral nerve, whereas in the CNS, an oligodendrocyte may provide insulation for a few separate axon segments. EM × 1,460,000. (Micrograph provided by the Regents of University of Michigan Medical School © 2012). (CC BY 4.0; via Nervous Tissue).
Alternate text description of the Figure 1.2.6.

Disorders of the...Nervous Tissue

Nervous tissue depends on healthy neurons, supporting glial cells, and intact myelin sheaths for rapid signaling. When these are damaged, communication in the nervous system slows or fails and characteristic symptoms appear.

A major group of disorders involves demyelination, the loss or damage of the myelin that insulates axons. Demyelination can result from genetic problems, infections, toxins, or autoimmune attacks. Regardless of the cause, the effect is similar: action potentials travel more slowly or are blocked, disrupting normal function.

Multiple sclerosis (MS) is a demyelinating disease of the central nervous system (CNS). It is an autoimmune disorder in which immune cells attack myelin in the brain and spinal cord. This causes inflammation, loss of myelin, and scarring (sclerosis) in white matter. As myelin is destroyed, nerve conduction is impaired. Symptoms can include muscle weakness, problems with coordination and balance, sensory changes (such as numbness or tingling), and autonomic disturbances like bladder dysfunction.

Guillain–Barré syndrome (pronounced gee-YAN bah-RAY) is a demyelinating disease of the peripheral nervous system (PNS). It is usually triggered by an infection and involves an autoimmune attack on myelin in peripheral nerves. Patients often develop rapidly progressing weakness (usually starting in the legs), sensory symptoms such as tingling or pain, and autonomic problems like abnormal heart rhythms or drops in blood pressure that can cause dizziness. As in MS, slowed or blocked conduction in affected nerves underlies the clinical signs.

In addition to demyelinating diseases, other major nervous system disorders include neurodegenerative conditions such as Alzheimer disease, Parkinson disease, and amyotrophic lateral sclerosis (ALS), which primarily damage neurons and synapses rather than myelin. These and other conditions will be discussed in more detail in later chapters.

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1.3: Sensation, Integration, and Response

Functional Organization of Neural Communication

Having looked at the components of nervous tissue, and the basic anatomy of the nervous system, next comes an understanding of how nervous tissue is capable of communicating within the nervous system. Before getting to the nuts and bolts of how this works, an illustration of how the components come together will be helpful. An example is summarized in Figure 1.3.1.

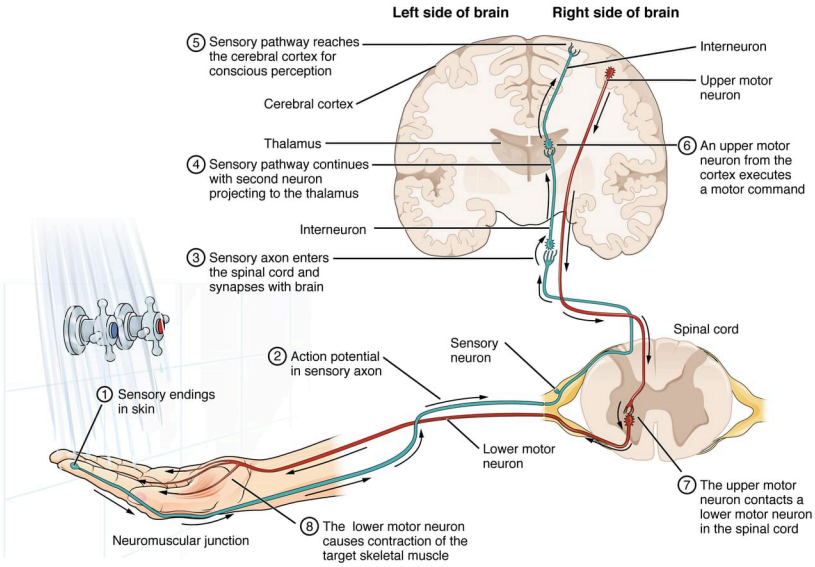


Figure 1.3.1: Sensory input from the hand travels to the brain for perception, then motor output returns to the hand to guide movement. (CC BY 4.0; via The Function of Nervous Tissue). Alternate text description of the Figure 1.3.1.

Imagine you are about to take a shower in the morning before going to school. You have turned on the faucet to start the water as you prepare to get in the shower. After a few minutes, you expect the water to be a temperature that will be comfortable to enter. So you put your hand out into the spray of water. What happens next depends on how your nervous system interacts with the stimulus of the water temperature and what you do in response to that stimulus.

Found in the skin of your fingers or toes is a type of sensory receptor that is sensitive to temperature, called a thermoreceptor. When you place your hand under the shower (Figure 1.3.2), the cell membrane of the thermoreceptors changes its electrical state (voltage). The amount of change is dependent on the strength of the stimulus (how hot the water is). This is called a graded potential. If the stimulus is strong, the voltage of the cell membrane will change enough to generate an electrical signal that will travel down the axon. You have learned about this type of signaling before, with respect to the interaction of nerves and muscles at the neuromuscular junction. The voltage at which such a signal is generated is called the threshold, and the resulting electrical signal is called an action potential. In this example, the action potential travels—a process known as propagation—along the axon from the axon hillock to the axon terminals and into the synaptic end bulbs. When this signal reaches the end bulbs, it causes the release of a signaling molecule called a neurotransmitter.

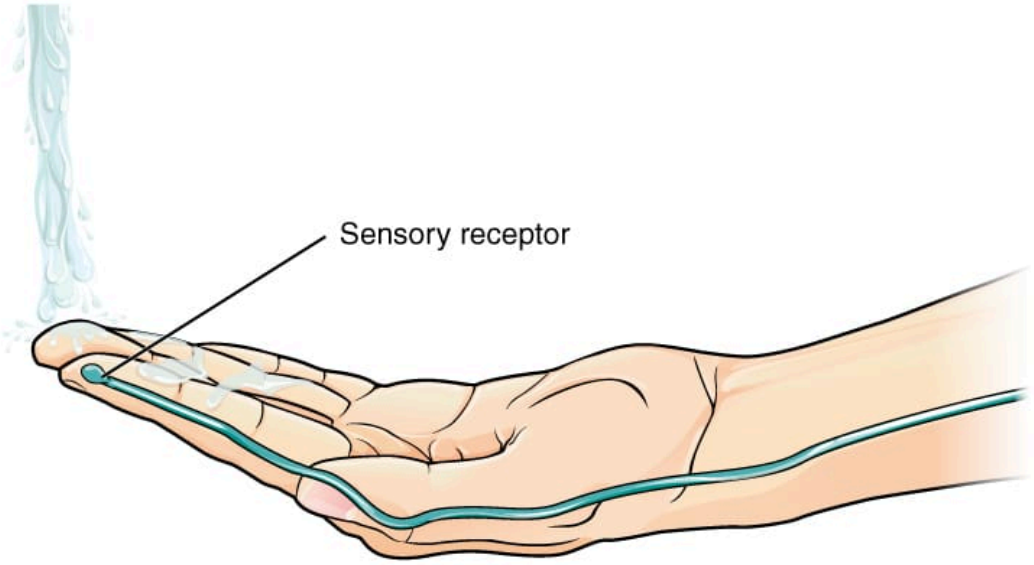


Figure 1.3.2: Receptors in the skin sense the temperature of the water. (CC BY 4.0; via The Function of Nervous Tissue). Alternate text description of the Figure 1.3.2.

The neurotransmitter diffuses across the short distance of the synapse and binds to a receptor protein of the target neuron. When the molecular signal binds to the receptor, the cell membrane of the target neuron changes its electrical state and a new graded potential begins. If that graded potential is strong enough to reach threshold, the second neuron generates an action potential at its axon hillock. The target of this neuron is another neuron in the thalamus of the brain, the part of the CNS that acts as a relay for sensory information. At another synapse, neurotransmitter is released and binds to its receptor. The thalamus then sends the sensory information to the cerebral cortex, the outermost layer of gray matter in the brain, where conscious perception of that water temperature begins.

Within the cerebral cortex, information is processed among many neurons, integrating the stimulus of the water temperature with other sensory stimuli, with your emotional state (you just aren't ready to wake up; the bed is calling to you), memories (perhaps of the lab notes you have to study before a quiz). Finally, a plan is developed about what to do, whether that is to turn the temperature up, turn the whole shower off and go back to bed, or step into the shower. To do any of these things, the cerebral cortex has to send a command out to your body to move muscles (Figure 1.3.3).

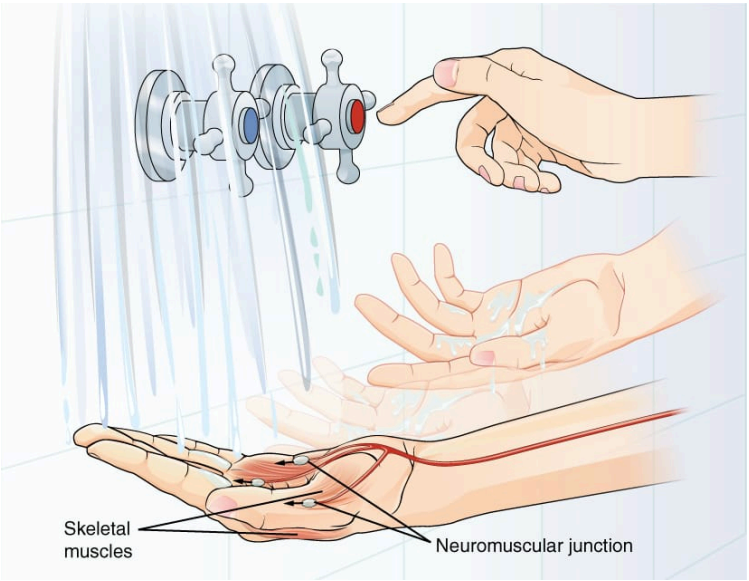


Figure 1.3.3: On the basis of the sensory input and the integration in the CNS, a motor response is formulated and executed. (CC BY 4.0; via The Function of Nervous Tissue). Alternate text description of the Figure 1.3.3.

A region of the cortex is specialized for sending signals down to the spinal cord for movement. The upper motor neuron is in this region, called the precentral gyrus of the frontal cortex, which has an axon that extends all the way down the spinal cord. At the level of the spinal cord at which this axon makes a synapse, a graded potential occurs in the cell membrane of a lower motor neuron. This second motor neuron is responsible for causing muscle fibers to contract. In the manner described in the chapter on muscle tissue, an action potential travels along the motor neuron axon into the periphery. The axon terminates on muscle fibers at the neuromuscular junction. Acetylcholine is released at this specialized synapse, which causes the muscle action potential to begin, following a large potential known as an end plate potential. When the lower motor neuron excites the muscle fiber, it contracts. All of this occurs in a fraction of a second, but this story is the basis of how the nervous system functions.

Overall flow

Stimulus (any change in the external or internal environment) → Sensory Receptors (unimodal; they are specialized to respond to only one type of stimulus energy) in the PNS → Sensory (Afferent) Pathways (neural routes that carry sensory information *toward* the CNS from peripheral receptors) → CNS Integration - brain and spinal cord has crucial ability to receive, process and combine sensory information with motor commands to produce coherent actions, thoughts, and consciousness, involving both subconscious (brainstem, cerebellum for balance/movement) and higher-level (cerebral cortex for decision-making, reasoning, memory) processes, ensuring the body adapts to its environment → Motor (Efferent) Pathways (are nerve tracts that carry commands away from the Central Nervous System (CNS) to muscles and glands, enabling movement and involuntary functions like heart rate, originating from the brain/spinal cord and ending at peripheral "effectors") → Effectors (Muscles - skeletal, cardiac, smooth) and Glands (exocrine, endocrine) → Response (Voluntary/Somatic: skeletal muscle movement; Involuntary (autonomic: heart rate, digestion, glandular secretion, smooth muscle activity).

Career Connection: Neurophysiologist

Understanding how the nervous system works could be a driving force in your career. Studying neurophysiology is a very rewarding path to follow. It means that there is a lot of work to do, but the rewards are worth the effort.

The career path of a research scientist can be straightforward: college, graduate school, postdoctoral research, academic research position at a university. A Bachelor's degree in science will get you started, and for neurophysiology that might be in biology, psychology, computer science, engineering, or neuroscience. But the real specialization comes in graduate school. There are many different programs out there to study the nervous system, not just neuroscience itself. Most graduate programs are doctoral, meaning that a Master's degree is not part of the work. These are usually considered five-year programs, with the first two years dedicated to course work and finding a research mentor, and the last three years dedicated to finding a research topic and pursuing that with a near single-mindedness. The research will usually result in a few publications in scientific journals, which will make up the bulk of a doctoral dissertation. After graduating with a Ph.D., researchers will go on to find specialized work called a postdoctoral fellowship within established labs. In this position, a researcher starts to establish their own research career with the hopes of finding an academic position at a research university.

Other options are available if you are interested in how the nervous system works. Especially for neurophysiology, a medical degree might be more suitable so you can learn about the clinical applications of neurophysiology and possibly work with human subjects. An academic career is not a necessity. Biotechnology firms are eager to find motivated scientists ready to tackle the tough questions about how the nervous system works so that therapeutic chemicals can be tested on some of the most challenging disorders such as Alzheimer's disease or Parkinson's disease, or spinal cord injury.

Others with a medical degree and a specialization in neuroscience go on to work directly with patients, diagnosing and treating mental disorders. You can do this as a psychiatrist, a neuropsychologist, a neuroscience nurse, or a neurodiagnostic technician, among other possible career paths.

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1.4: The Action Potential

Electrically Active Cell Membranes

The three main functions of the nervous system — sensation, integration, and response — depend on how neurons send signals. To understand this, you need to know how an excitable membrane produces electrical signals called action potentials. An action potential is a rapid change in the membrane that acts as a signal. Smaller, local changes called graded potentials will be covered next.

All living cells have a small voltage across their membrane, called the membrane potential. The inside of the cell is slightly negative compared with the outside, usually about –60 to –90 mV. This is caused by unequal ion concentrations (mainly Na^+, K^+, and Cl^-) and by the membrane letting some ions cross more easily than others. Leak channels and the sodium–potassium (Na^+/K^+) pump work all the time to maintain this resting membrane potential. Muscle cells and neurons both use changes in this membrane potential to do their jobs.

The cell membrane is a phospholipid bilayer and acts as a barrier. Only substances that dissolve in the hydrophobic core can cross easily. Charged particles (ions) cannot cross without help. Transmembrane proteins — channel proteins and pumps — allow ions to move across (Figure 1.4.1). Passive channels let ions move down their gradients, while active transporters, especially the Na^+/K^+ pump, use energy to move Na^+ out of the cell and K^+ into the cell. This controlled ion movement creates and maintains the membrane potential and makes action potentials possible.

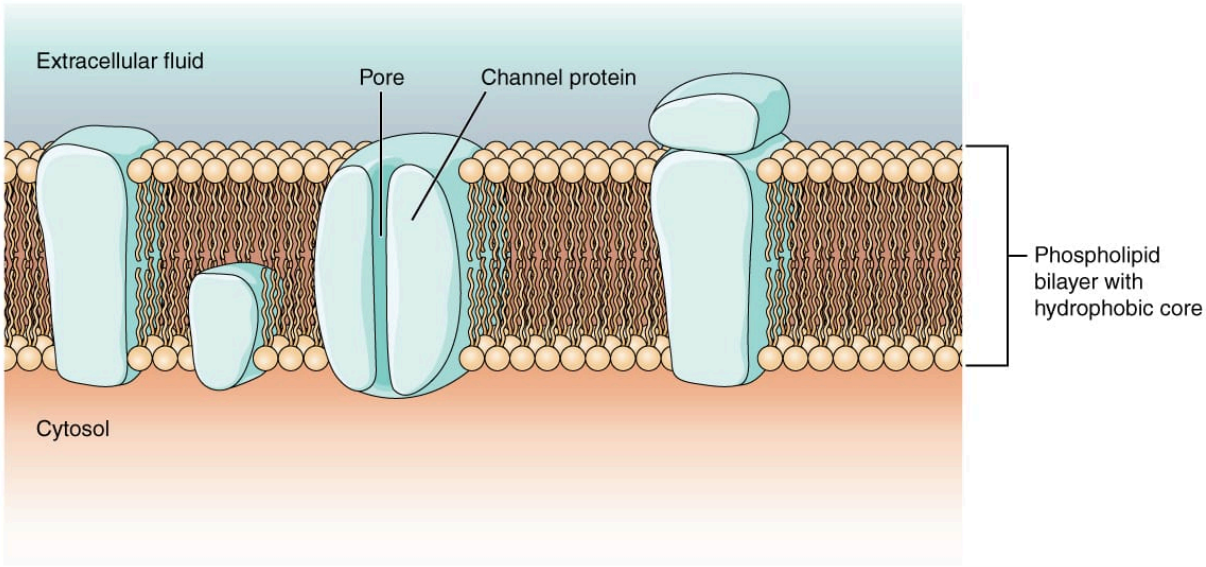


Figure 1.4.1: The cell membrane is composed of a phospholipid bilayer and has many transmembrane proteins, including different types of channel proteins that serve as ion channels. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.1.

The sodium–potassium (Na^+/K^+) pump uses energy from ATP, so it is called an ATPase. It moves Na^+ out of the cell and K^+ into the cell, against their concentration gradients (Na^+ higher outside, K^+ higher inside). This active transport maintains the ion gradients that are needed for the membrane potential.

Ion channels are proteins that let specific ions cross the membrane down their electrochemical gradients. The structure of the channel (its size and charge) makes it selective for certain ions. Together, these channels and the Na^+/K^+ pump create the resting membrane potential, graded potentials, and action potentials—especially in nerve and muscle cells.

There are four main types of ion channels important in neurons:

1. Ligand-gated (chemically gated) channels – open when a specific chemical (like a neurotransmitter) binds to the channel.
2. Mechanically gated channels – open in response to physical deformation, such as stretch, pressure, or vibration.
3. Voltage-gated channels – open or close when the membrane potential changes.
4. Leak (leakage) channels – always open or mostly open; set the resting membrane potential.

Ligand-gated (chemically gated) ion channel

Ligand-gated ion channels (chemically gated) opens when a chemical (ligand) binds on the outside of the cell. Found at synapses. Example: nicotinic ACh receptor at the neuromuscular junction (opens when acetylcholine binds).This type of channel is also known as an ionotropic receptor because when the ligand, known as a neurotransmitter in the nervous system, binds to the protein, ions cross the membrane changing its charge (Figure 1.4.2).

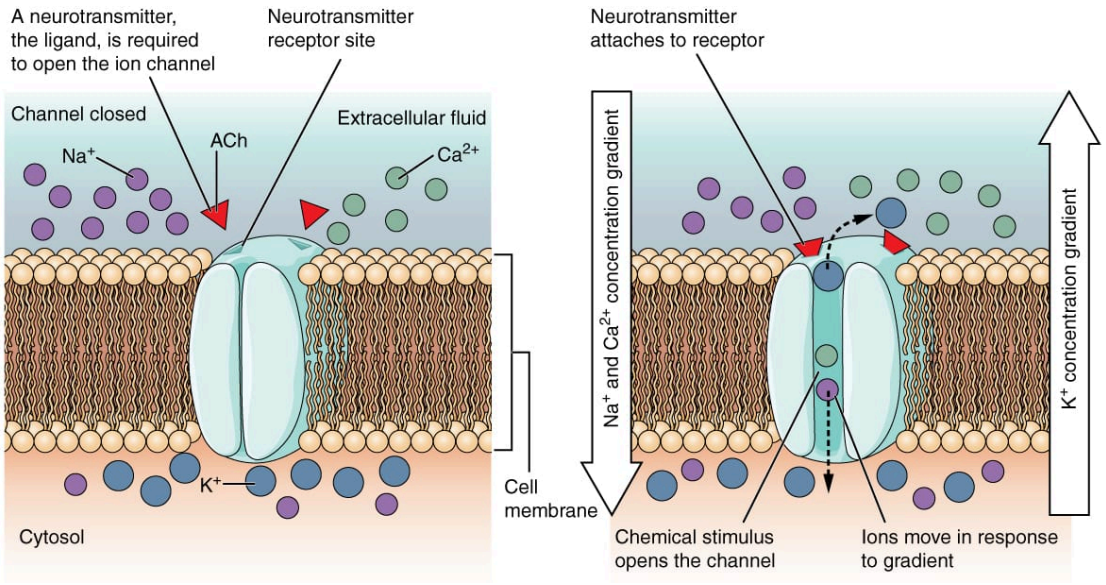


Figure 1.4.2: When the ligand, in this case the neurotransmitter acetylcholine, binds to a specific location on the extracellular surface of the channel protein, the pore opens to allow select ions through. The ions, in this case, are cations of sodium, calcium, and potassium. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.2.

Mechanically-gated ion channel

A mechanically gated ion channel opens when the membrane is stretched, pressed, or bent (physical distortion of the cell membrane). They are mostly located in sensory receptors for touch, pressure, some hearing and balance cells. For example, as pressure is applied to the skin, these channels open and allow ions to enter the cell. Similar to this type of channel would be the channel that opens on the basis of temperature changes, as in testing the water in the shower (Figure 1.4.3).

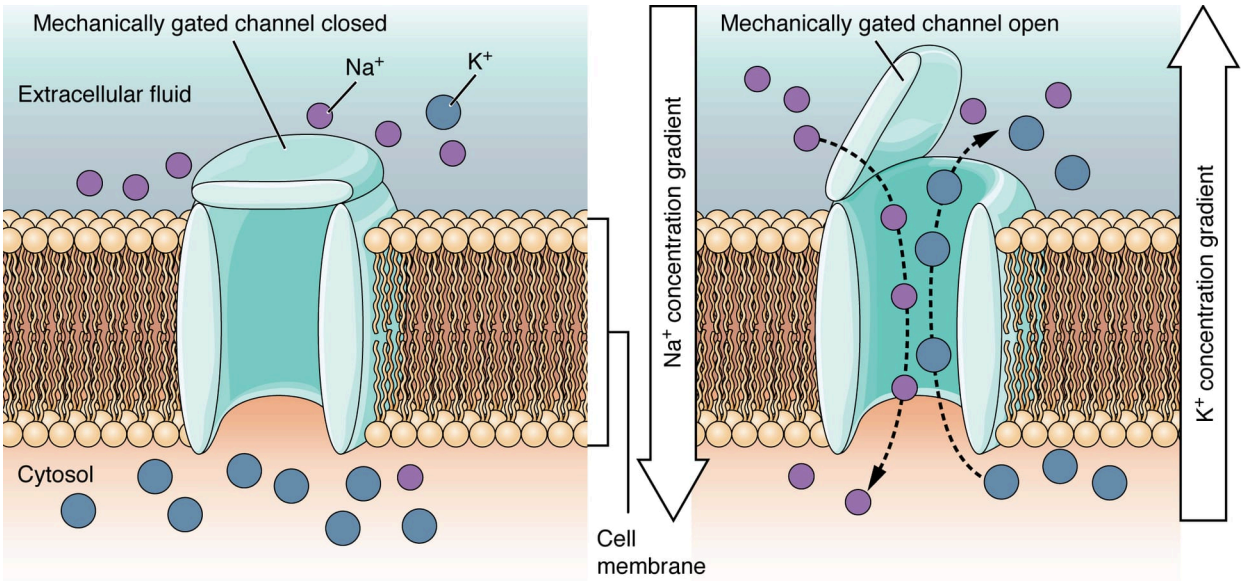


Figure 1.4.3: When a mechanical change occurs in the surrounding tissue, such as pressure or touch, the channel is physically opened. Thermoreceptors work on a similar principle. When the local tissue temperature changes, the protein reacts by physically opening the channel. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.3.

Voltage-gated ion channel

A voltage-gated channel is a channel that responds to changes in the electrical properties of the membrane in which it is embedded. Normally, the inner portion of the membrane is at a negative voltage. When that voltage becomes less negative, the channel begins to allow ions to cross the membrane (Figure 1.4.4).

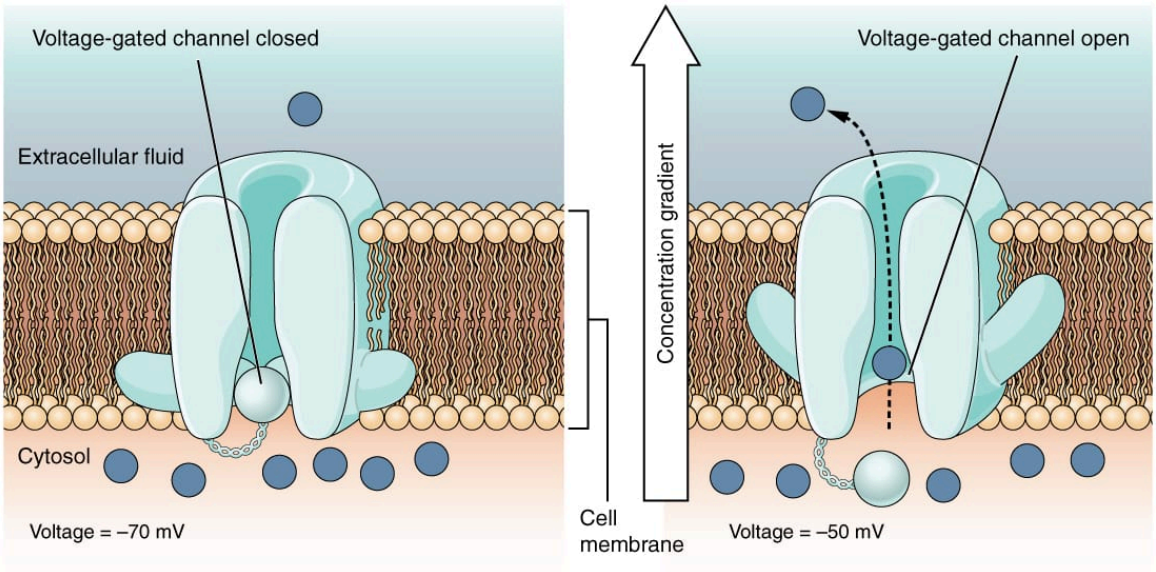


Figure 1.4.4: Voltage-gated channels open when the transmembrane voltage changes around them. Amino acids in the structure of the protein are sensitive to charge and cause the pore to open to the selected ion. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.4.

Leakage Channel

Leak (leakage) channels open and close at random, hence the reference to leaking. It has an intrinsic rate of switching between the open and closed states. Allow ions (especially K leak channels) to move down their gradients. There is no actual event that

opens the channel; instead, it has an intrinsic rate of switching between the open and closed states. Leakage channels contribute to the resting transmembrane voltage (inside negative, outside positive) of the excitable membrane (Figure 1.4.5).

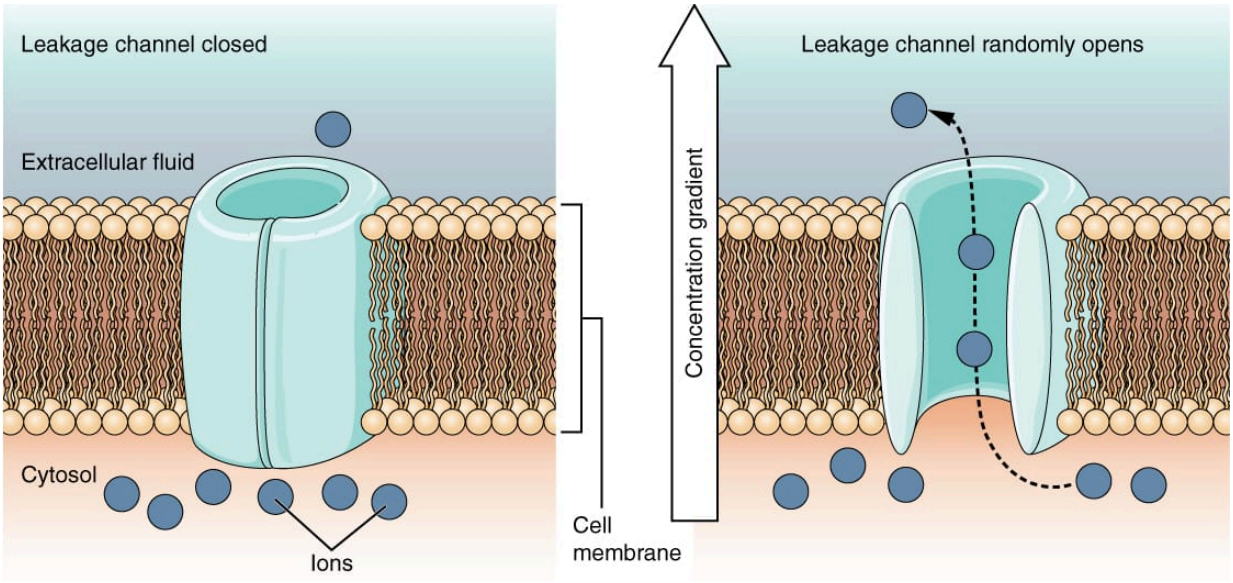


Figure 1.4.5: In certain situations, ions need to move across the membrane randomly. The particular electrical properties of certain cells are modified by the presence of this type of channel. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.5.

In neurons, ligand-gated and mechanically gated channels mainly create graded potentials (local changes), and voltage-gated channels create and spread action potentials. Leak channels and the Na⁺/K⁺ pump keep the resting potential ready between signals.

The Membrane Potential

The electrical state of the cell membrane can have several variations. These are all variations in the membrane potential. A potential is a distribution of charge across the cell membrane, measured in millivolts (mV). The standard is to compare the inside of the cell relative to the outside, so the membrane potential is a value representing the charge on the intracellular side of the membrane based on the outside being zero, relatively speaking (Figure 1.4.6).

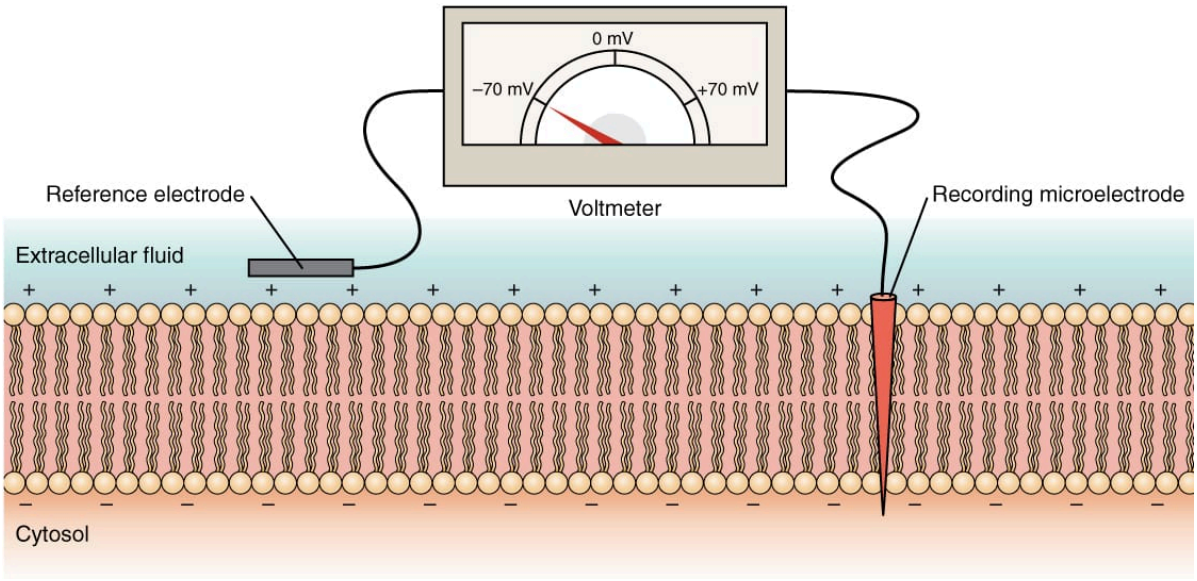


Figure 1.4.6: A recording electrode is inserted into the cell and a reference electrode is outside the cell. By comparing the charge measured by these two electrodes, the transmembrane voltage is determined. It is conventional to express that value for the cytosol relative to the outside. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.6.

The concentration of ions in extracellular and intracellular fluids is largely balanced, with a net neutral charge. However, a slight difference in charge occurs right at the membrane surface, both internally and externally. It is the difference in this very limited region that has all the power in neurons (and muscle cells) to generate graded potentials and action potentials. Graded potentials and action potentials are both changes in a neuron's membrane potential, but they differ in how they are generated, how they spread, and what they do.

Resting Membrane Potential

When a neuron is not sending a signal, it is at rest. In this state, most gated ion channels are closed; only leak channels are open. Ions are then arranged in a typical pattern:

- Sodium (Na⦿) concentration is much higher outside the cell than inside.
- Potassium (K⦿) concentration is higher inside the cell than outside.
- Inside the cell, there are many large negative ions (phosphates and proteins) that cannot cross the membrane easily.

Because of this ion distribution, the inside of the membrane is slightly negative compared with the outside. This voltage difference is called the resting membrane potential and is usually about –70 mV in neurons (Figure 1.4.6).

Leakage channels allow Na^+ to slowly move into the cell or K^+ to slowly move out, and the Na^+/K^+ pump restores them. This may appear to be a waste of energy, but each has a role in maintaining the membrane potential. Na⦿ and K⦿ leak channels allow small, constant ion movement, and the Na⦿/K⦿ pump uses ATP to return Na⦿ and K⦿ to their original sides. Together, these processes maintain the resting membrane potential and prepare the neuron to generate action potentials.

Graded potential

A graded potential is a small, local change in membrane potential, for example from –70 mV to –62 mV. It usually starts when ligand-gated or mechanically gated channels open, such as at a synapse or a sensory ending. Graded potentials can be different sizes and can add together. If a graded depolarization is strong enough to bring the membrane to the threshold, about –55 mV at the axon hillock, it will trigger an action potential.

Threshold and all-or-none

Threshold is the critical membrane voltage (about –55 mV) that must be reached to open enough voltage-gated Na^+ channels. If depolarization does not reach threshold, no action potential is produced. If depolarization reaches threshold or higher, a full action potential always occurs. The action potential is all-or-none: it always has the same size and shape. A stronger stimulus does not make a bigger action potential; instead, it causes more action potentials or makes them fire more often.

The Action Potential

Resting membrane potential is the steady state of the neuron. It is kept stable by a balance of ion leakage and ion pumping, mainly Na^+ and K^+ leak channels and the Na^+/K^+ pump. At rest (about –70 mV), most voltage-gated Na^+ and K^+ channels are closed.

An electrical signal begins when Na^+ channels open. First, a stimulus opens ligand-gated or mechanically gated Na^+ channels.

If the membrane reaches threshold (about –55 mV), voltage-gated Na^+ channels open. Because Na^+ concentration is much higher outside the cell than inside, Na^+ rushes into the cell. Na^+ is positively charged, so the inside becomes less negative. This change toward zero is called depolarization. During depolarization, many voltage-gated Na^+ channels are open.

At about +30 mV, voltage-gated Na^+ channels inactivate (close), and voltage-gated K^+ channels open. K^+ then leaves the cell, carrying positive charge out. The membrane potential moves back toward the resting level. This return toward –70 mV is called repolarization. During repolarization, voltage-gated K^+ channels are open.

Because voltage-gated K^+ channels close a little slowly, extra K^+ leaves the cell and the membrane becomes even more negative than –70 mV for a short time. This is called hyperpolarization and happens while K^+ channels are still open but starting to close.

This full sequence — depolarization (voltage-gated Na^+ channels open), repolarization (voltage-gated K^+ channels open, Na^+ channels inactivated), and brief hyperpolarization (K^+ channels slowly closing) — is the action potential, the basic electrical signal that neurons use to communicate (Figure 1.4.7).

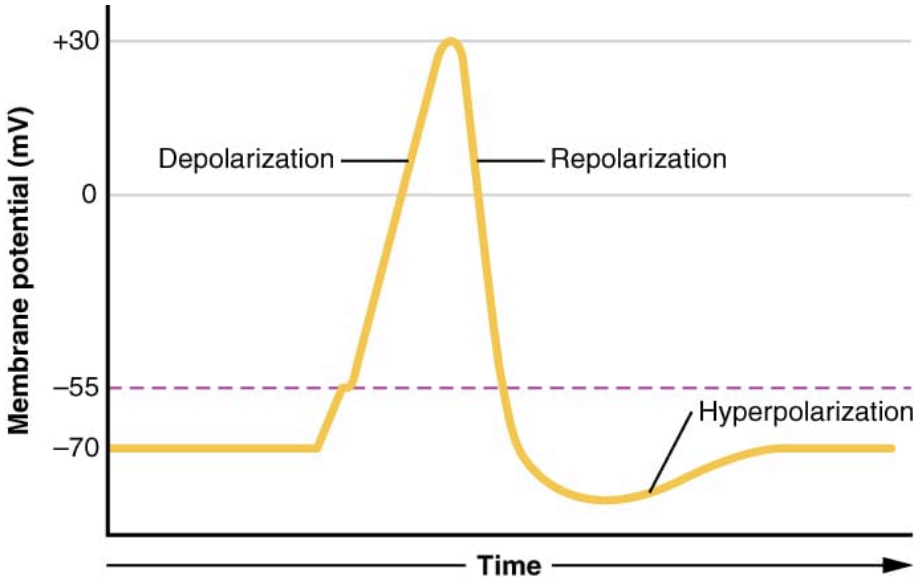


Figure 1.4.7: Plotting voltage measured across the cell membrane against time, the action potential begins with depolarization, followed by repolarization, which goes past the resting potential into hyperpolarization, and finally the membrane returns to rest. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.7.

Depolarization and repolarization depend mainly on two channels: voltage-gated Na+ channels and voltage-gated K+ channels.

Each voltage-gated Na+ channel has two gates. At rest, the activation gate is closed and the inactivation gate is open. When the membrane reaches threshold (–55 mV), the activation gate opens and Na+ rushes in, causing depolarization. Shortly after, at the peak of depolarization, the inactivation gate closes, so Na+ can no longer enter.

The voltage-gated K+ channel has one gate. It opens more slowly, around –50 mV, just as Na+ entry peaks. K+ leaves the cell, causing repolarization. Because these K+ channels close with a slight delay, extra K+ leaves, leading to brief hyperpolarization before the membrane returns to the resting level.

Refractory period

All of these events occur in about 2 milliseconds ((Figure 1.4.8). During an action potential, there is an absolute refractory period, when no new action potential can start because the Na+ inactivation gates are closed. After this, during the relative refractory period, a new action potential is possible, but only if the stimulus is stronger than usual, because K+ is still flowing out and opposing depolarization.

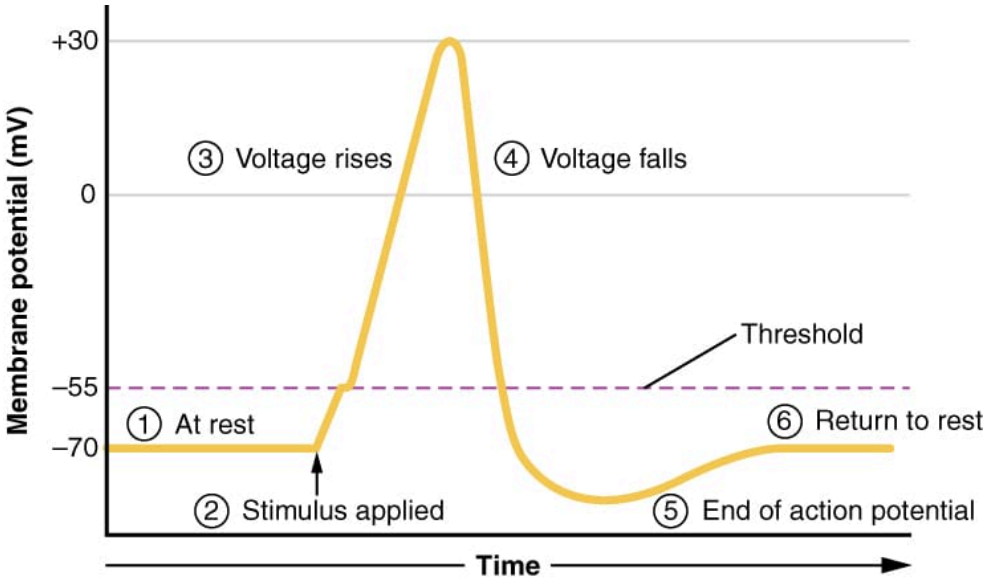


Figure 1.4.8: Plotting voltage measured across the cell membrane against time, the events of the action potential can be related to specific changes in the membrane voltage. (1) At rest, the membrane voltage is -70 mV. (2) The membrane begins to depolarize when an external stimulus is applied. (3) The membrane voltage begins a rapid rise toward +30 mV. (4) The membrane voltage starts to return to a negative value. (5) Repolarization continues past the resting membrane voltage, resulting in hyperpolarization. (6) The membrane voltage returns to the resting value shortly after hyperpolarization. (CC BY 4.0; via The Action Potential). Alternate text description of the Figure 1.4.8.

Propagation of the Action Potential

The action potential starts at the initial segment of the axon, where there is a very high density of voltage-gated Na⁺ channels. When these channels open and cause depolarization, Na⁺ enters and spreads a short distance along the inside of the membrane. This local depolarization opens the next group of voltage-gated Na⁺ channels, so more Na⁺ rushes in and the depolarization moves step by step down the axon toward the axon terminals.

At the peak of depolarization, voltage-gated Na⁺ channels become inactivated. During this absolute refractory period they cannot open again. Because of this, depolarization cannot move backward into the region that just produced an action potential, so the signal travels only in one direction.

In unmyelinated axons, this process happens continuously along the whole length of the membrane and is called continuous conduction. It is relatively slow because new channels must open at every small segment of the axon.

In myelinated axons, voltage-gated Na⁺ channels are concentrated only at the nodes of Ranvier. Na⁺ entering at one node spreads quickly under the myelin to the next node. If the distance between nodes is correct, the membrane at the next node is still depolarized above threshold, so its voltage-gated Na⁺ channels open and a new action potential starts there. This “jumping” of the action potential from node to node is called saltatory conduction (saltare = “to leap”) and is much faster than continuous conduction.

Axon diameter also affects speed. Wider axons have lower internal resistance, so depolarization spreads faster along them—similar to how water flows more easily in a wide river than in a narrow stream.

Homeostatic Imbalances

Glial cells and ion balance

Glial cells, especially astrocytes, help keep the chemical environment of CNS tissue stable. The concentrations of ions in the extracellular fluid are crucial for setting the membrane potential and for normal electrochemical signaling. If this ion balance is disturbed, serious problems can occur.

Normally, K⊡ concentration is higher inside neurons than outside. After an action potential, K⊡ leak channels and the Na⊡/K⊡ pump return Na⊡ and K⊡ to their usual locations. After a stroke or other ischemic event, however, extracellular K⊡ can rise sharply. Astrocytes normally help clear this excess K⊡, but if the imbalance is too large, their normal buffering is overwhelmed.

In these conditions, astrocytes become reactive: they swell, their processes enlarge, and they lose some of their K⊡ buffering capacity. The Na⊡/K⊡ pump may be impaired or even reversed. Sodium can then leak into cells while K⊡ leaks out, disrupting the normal Na⊡/K⊡ balance. This ion imbalance changes the internal chemistry of neurons and glia and can prevent them from functioning or recovering normally.

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1.5: Communication Between Neurons

Graded Potentials

The electrical changes taking place within a neuron, as described in the previous section, are similar to a light switch being turned on. A stimulus starts the depolarization, but the action potential runs on its own once a threshold has been reached. The question is now, “What flips the light switch on?” Temporary changes to the cell membrane voltage can result from neurons receiving information from the environment, or from the action of one neuron on another. These special types of potentials influence a neuron and determine whether an action potential will occur or not. Many of these transient signals originate at the synapse

Local changes in the membrane potential are called graded potentials and are usually associated with the dendrites of a neuron. The amount of change in the membrane potential is determined by the size of the stimulus that causes it. In the example of testing the temperature of the shower, slightly warm water would only initiate a small change in a thermoreceptor, whereas hot water would cause a large amount of change in the membrane potential.

Graded potentials can be of two sorts, either they are depolarizing or hyperpolarizing (Figure 1.5.1). For a membrane at the resting potential, a graded potential represents a change in that voltage either above -70 mV or below -70 mV. Depolarizing graded potentials are often the result of Na^+ or Ca^{2+} entering the cell. Both of these ions have higher concentrations outside the cell than inside; because they have a positive charge, they will move into the cell causing it to become less negative relative to the outside. Hyperpolarizing graded potentials can be caused by K^+ leaving the cell or Cl^- entering the cell. If a positive charge moves out of a cell, the cell becomes more negative; if a negative charge enters the cell, the same thing happens.

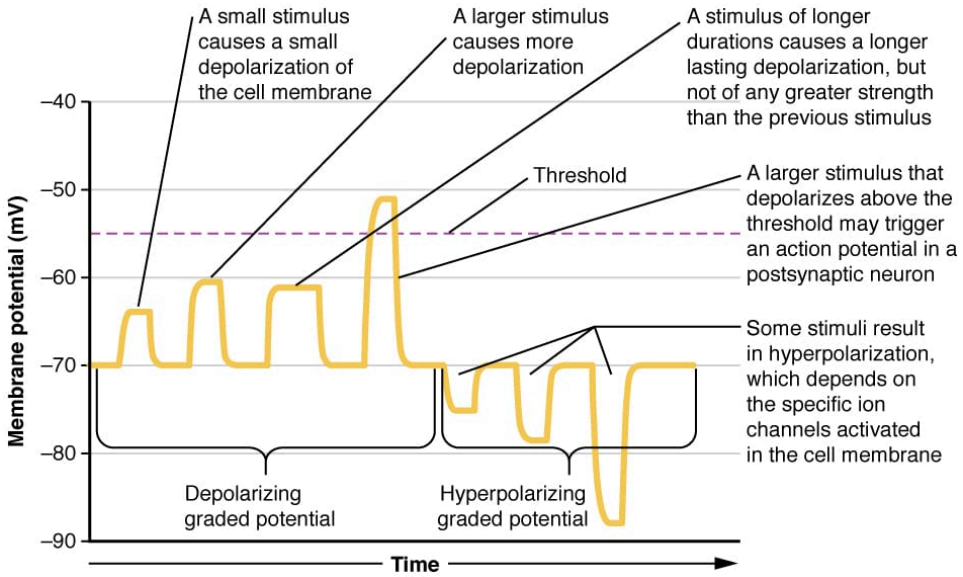


Figure 1.5.1: Graded potentials are temporary changes in the membrane voltage, the characteristics of which depend on the size of the stimulus. Some types of stimuli cause depolarization of the membrane, whereas others cause hyperpolarization. It depends on the specific ion channels that are activated in the cell membrane. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 1.5.1.

Types of Graded Potentials

For the unipolar cells of sensory neurons — both those with free nerve endings and those within encapsulations — graded potentials develop in the dendrites that influence the generation of an action potential in the axon of the same cell. This is called a generator potential. For other sensory receptor cells, such as taste cells or photoreceptors of the retina, graded potentials in their membranes result in the release of neurotransmitters at synapses with sensory neurons. This is called a receptor potential.

A postsynaptic potential (PSP) is the graded potential in the dendrites of a neuron that is receiving synapses from other cells.

Postsynaptic potentials can be depolarizing or hyperpolarizing. Depolarization in a postsynaptic potential is called an excitatory postsynaptic potential (EPSP) because it causes the membrane potential to move toward threshold. Hyperpolarization in a postsynaptic potential is an inhibitory postsynaptic potential (IPSP) because it causes the membrane potential to move away from threshold.

Summation

All types of graded potentials will result in small changes of either depolarization or hyperpolarization in the voltage of a membrane. These changes can lead to the neuron reaching threshold if the changes add together, or summate. The combined effects of different types of graded potentials are illustrated in Figure 1.5.2. If the total change in voltage in the membrane is a positive 15 mV, meaning that the membrane depolarizes from -70 mV to -55 mV, then the graded potentials will result in the membrane reaching threshold.

For receptor potentials, threshold is not a factor because the change in membrane potential for receptor cells directly causes neurotransmitter release. However, generator potentials can initiate action potentials in the sensory neuron axon, and postsynaptic potentials can initiate an action potential in the axon of other neurons. Graded potentials summate at a specific location at the beginning of the axon to initiate the action potential, namely the initial segment. For sensory neurons, which do not have a cell body between the dendrites and the axon, the initial segment is directly adjacent to the dendritic endings. For all other neurons, the axon hillock is essentially the initial segment of the axon, and it is where summation takes place. These locations have a high density of voltage-gated Na^+ channels that initiate the depolarizing phase of the action potential.

Summation can be spatial or temporal, meaning it can be the result of multiple graded potentials at different locations on the neuron, or all at the same place but separated in time. Spatial summation is related to associating the activity of multiple inputs to a neuron with each other. Temporal summation is the relationship of multiple action potentials from a single cell resulting in a significant change in the membrane potential. Spatial and temporal summation can act together, as well.

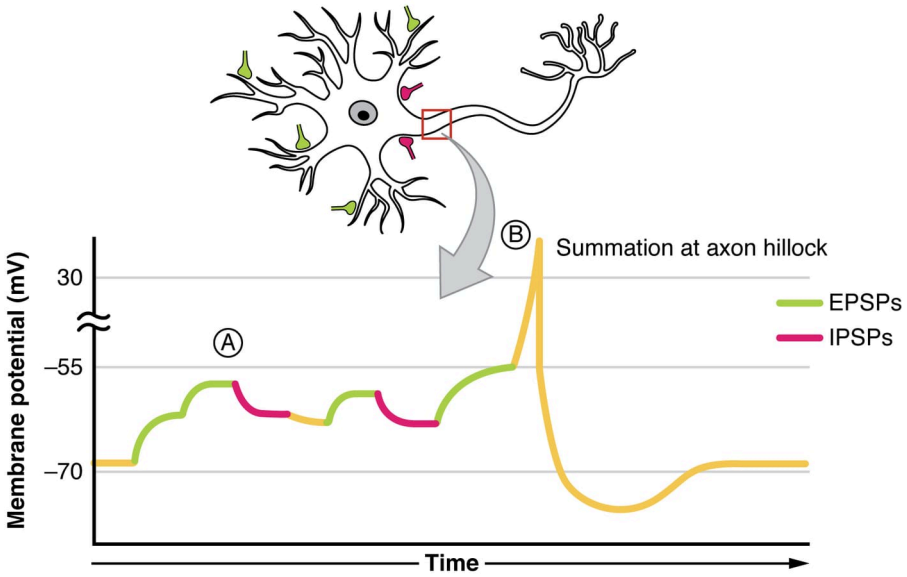


Figure 1.5.2: The result of summation of postsynaptic potentials is the overall change in the membrane potential. At point A, several different excitatory postsynaptic potentials add up to a large depolarization. At point B, a mix of excitatory and inhibitory postsynaptic potentials result in a different end result for the membrane potential. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 1.5.2.

Table 1.5.1: Comparison of graded potentials and action potentials

Feature	Graded Potential	Action Potential
Where they start	Usually on dendrites or cell body	Usually at axon hillock and along the axon

Feature	Graded Potential	Action Potential
Cause (how they start)	Opening of ligand-gated or mechanically-gated channels (sometimes local voltage-gated)	Opening of voltage-gated Na⁺ and K⁺ channels after threshold is reached
Size (amplitude)	Small change; varies with stimulus strength (graded)	Always the same size (all-or-none) once it fires
Can they add together?	Yes. Summation: many small graded potentials can add up	No. Each action potential is separate, same size
Spread with distance	Decreases as it spreads (dies out)	Does not decrease; is regenerated along the axon
Direction of travel	Can go in all directions from the stimulus site	Goes one way along the axon (from hillock to terminals)
Depolarizing or hyperpolarizing?	Can be depolarizing (more positive) or hyperpolarizing (more negative)	Main phase is depolarizing, followed by repolarizing and often slight hyperpolarization
Threshold needed?	No fixed threshold; size depends on stimulus	Needs to reach threshold to start (all-or-none)
Main function	Local signal input; helps decide if neuron will fire an AP	Long-distance, fast signal output along the axon
Examples	EPSPs, IPSPs at synapses, receptor potentials in sensory endings	Nerve impulse in a motor neuron going to muscle

Synapses

Neurons and their targets form synapses. The neuron that generates and conducts the action potential to the target is called a presynaptic cell. The target cell receiving the action potential is called a postsynaptic cell . While the presynaptic cell is always a neuron (because only neurons have axons and can form a synapse), the postsynaptic cell can be a neuron or another type of cell such as skeletal, cardiac or smooth muscle cells, or glands. In synaptic cleft. There are two types of connections between electrically active cells, chemical synapses and electrical synapses.

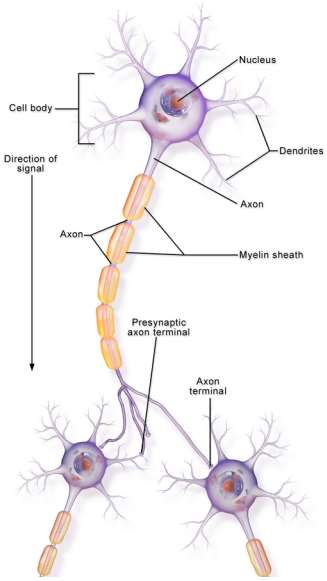


Figure 1.5.3: A neuron showing its major structures, including dendrites, cell body, nucleus, axon, myelin sheath, and axon terminals. Signals travel from the dendrites through the cell body and along the axon to the presynaptic axon terminals, where communication with other neurons occurs. (CC BY-SA 4.0; via Neuron Part 1). Alternate text description of the Figure 1.5.3.

Chemical Synapse

Chemical synapses use chemical signals to pass information from one cell to another. They are the most common type of synapse in the nervous system. In a chemical synapse, the presynaptic cell releases a chemical signal called a neurotransmitter, which then binds to receptors on the postsynaptic cell.

There are many neurotransmitters, such as acetylcholine, serotonin, dopamine, adrenaline, and glutamate. Each neurotransmitter has specific receptors on the postsynaptic membrane, and different combinations of neurotransmitters and receptors produce different responses. Synapses can be named by the neurotransmitter they use (for example, glutamatergic synapses use glutamate).

All chemical synapses share several basic parts: a synaptic end bulb of the presynaptic neuron, neurotransmitter stored in vesicles, a synaptic cleft (the small gap between cells), receptors for the neurotransmitter, and the postsynaptic membrane of the postsynaptic neuron.

Electrical Synapses

In an electrical synapse, there is a direct connection between the presynaptic and postsynaptic cells and the connection is formed by gap junctions. Thus, the electrical charges of an action potential can pass directly from one cell to the next. If one cell delivers an action potential in an electrical synapse, the joined cell will also generate an action potential because the electrical charges will pass between the cells 10 period 4 period 3" data-semantic-speech-none="10 period 4 period 3" data-semantic-summary=" period separated list of length 3" data-semantic-summary-none="period separated list of length 3" data-semantic-type="punctuated" data-speech-node="true"> 10" data-semantic-speech-none="10" data-semantic-summary=" number" data-semantic-summary-none="number" data-semantic-type="number" data-speech-node="true">. Although representing the minority of synapses, electrical synapses are found throughout the nervous system. These synapses also occur between excitable cells other than neurons, for example between smooth muscle cells in the intestines and cardiac muscle cells in the heart.

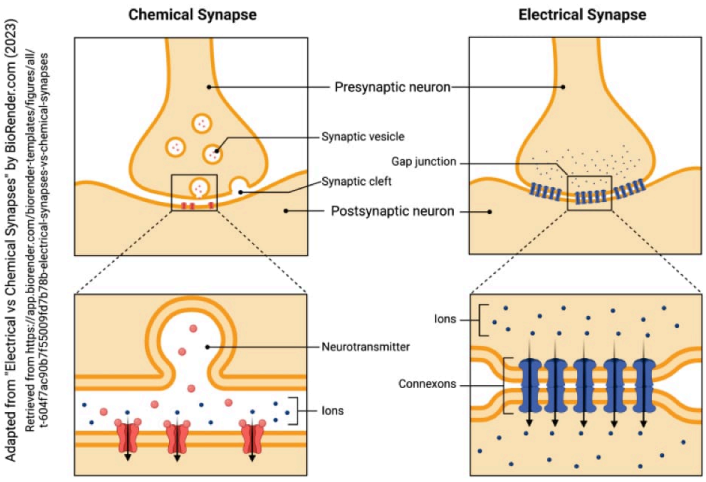


Figure 1.5.4: In a chemical synapse, neurotransmitters are released from synaptic vesicles, cross the synaptic cleft, and bind to receptors on the postsynaptic neuron. In an electrical synapse, ions pass directly between cells through gap junction channels (connexons), allowing rapid electrical communication between neurons. (CC BY-SA 4.0; via Neuron Part 1). Alternate text description of the Figure 1.5.4.

Neurotransmitter Release

When an action potential reaches the axon terminals, voltage-gated Ca^{2+} channels in the membrane of the synaptic end bulb open. The concentration of Ca^{2+} increases inside the end bulb, and the Ca^{2+} ion associates with proteins in the outer surface of neurotransmitter vesicles. The Ca^{2+} facilitates the merging of the vesicle with the presynaptic membrane so that the neurotransmitter is released through exocytosis into the small gap between the cells, known as the synaptic cleft.

Once in the synaptic cleft, the neurotransmitter diffuses the short distance to the postsynaptic membrane and can interact with neurotransmitter receptors. Receptors are specific for the neurotransmitter, and the two fit together like a key and lock. One neurotransmitter binds to its receptor and will not bind to receptors for other neurotransmitters, making the binding a specific chemical event.

Neurotransmitter Systems

There are several systems of neurotransmitters that are found at various synapses in the nervous system. These groups refer to the chemicals that are the neurotransmitters, and within the groups are specific systems.

The first group, which is a neurotransmitter system of its own, is the cholinergic system. It is the system based on acetylcholine. This includes the NMJ as an example of a cholinergic synapse, but cholinergic synapses are found in other parts of the nervous system. They are in the autonomic nervous system, as well as distributed throughout the brain.

The cholinergic system has two types of receptors, the nicotinic receptor is found in the NMJ as well as other synapses. There is also an acetylcholine receptor known as the muscarinic receptor. Both of these receptors are named for drugs that interact with the receptor in addition to acetylcholine. Nicotine will bind to the nicotinic receptor and activate it similar to acetylcholine. Muscarine, a product of certain mushrooms, will bind to the muscarinic receptor. However, nicotine will not bind to the muscarinic receptor and muscarine will not bind to the nicotinic receptor.

Another group of neurotransmitters are amino acids. This includes glutamate (Glu), GABA (gamma-aminobutyric acid, a derivative of glutamate), and glycine (Gly). These amino acids have an amino group and a carboxyl group in their chemical structures. Glutamate is one of the 20 amino acids that are used to make proteins. Each amino acid neurotransmitter would be part of its own system, namely the glutamatergic, GABAergic, and glycinergic systems. They each have their own receptors and do not interact with each other. Amino acid neurotransmitters are eliminated from the synapse by reuptake. A pump in the cell membrane of the presynaptic element, or sometimes a neighboring glial cell, will clear the amino acid from the synaptic cleft so that it can be recycled, repackaged in vesicles, and released again.

Another class of neurotransmitter is the biogenic amine, a group of neurotransmitters that are enzymatically made from amino acids. They have amino groups in them, but no longer have carboxyl groups and are therefore no longer classified as amino acids. Serotonin is made from tryptophan. It is the basis of the serotonergic system, which has its own specific receptors. Serotonin is transported back into the presynaptic cell for repackaging.

Other biogenic amines are made from tyrosine, and include dopamine, norepinephrine, and epinephrine. Dopamine is part of its own system, the dopaminergic system, which has dopamine receptors. Dopamine is removed from the synapse by transport proteins in the presynaptic cell membrane. Norepinephrine and epinephrine belong to the adrenergic neurotransmitter system. The two molecules are very similar and bind to the same receptors, which are referred to as alpha and beta receptors. Norepinephrine and epinephrine are also transported back into the presynaptic cell. The chemical epinephrine (epi- = "on"; "-nephrine" = kidney) is also known as adrenaline (renal = "kidney"), and norepinephrine is sometimes referred to as noradrenaline. The adrenal gland produces epinephrine and norepinephrine to be released into the blood stream as hormones.

A neuropeptide is a neurotransmitter molecule made up of chains of amino acids connected by peptide bonds. This is what a protein is, but the term protein implies a certain length to the molecule. Some neuropeptides are quite short, such as met-enkephalin, which is five amino acids long. Others are long, such as beta-endorphin, which is 31 amino acids long. Neuropeptides are often released at synapses in combination with another neurotransmitter, and they often act as hormones in other systems of the body, such as vasoactive intestinal peptide (VIP) or substance P.

The effect of a neurotransmitter on the postsynaptic element is entirely dependent on the receptor protein. First, if there is no receptor protein in the membrane of the postsynaptic element, then the neurotransmitter has no effect. The depolarizing or hyperpolarizing effect is also dependent on the receptor. When acetylcholine binds to the nicotinic receptor, the postsynaptic cell is depolarized. This is because the receptor is a cation channel and positively charged Na^+ will rush into the cell. However, when acetylcholine binds to the muscarinic receptor, of which there are several variants, it might cause depolarization or hyperpolarization of the target cell.

The amino acid neurotransmitters, glutamate, glycine, and GABA, are almost exclusively associated with just one effect. Glutamate is considered an excitatory amino acid, but only because Glu receptors in the adult cause depolarization of the postsynaptic cell. Glycine and GABA are considered inhibitory amino acids, again because their receptors cause hyperpolarization.

The biogenic amines have mixed effects. For example, the dopamine receptors that are classified as D1 receptors are excitatory whereas D2-type receptors are inhibitory. Biogenic amine receptors and neuropeptide receptors can have even more complex effects because some may not directly affect the membrane potential, but rather have an effect on gene transcription or other metabolic processes in the neuron. The characteristics of the various neurotransmitter systems presented in this section are organized in Table 1.5.2.

The important thing to remember about neurotransmitters, and signaling chemicals in general, is that the effect is entirely dependent on the receptor. Neurotransmitters bind to one of two classes of receptors at the cell surface, ionotropic or metabotropic (Figure 12.28). Ionotropic receptors are ligand-gated ion channels, such as the nicotinic receptor for acetylcholine or the glycine receptor. A metabotropic receptor involves a complex of proteins that result in metabolic changes within the cell. The receptor complex includes the transmembrane receptor protein, a G protein, and an effector protein. The neurotransmitter, referred to as the first messenger, binds to the receptor protein on the extracellular surface of the cell, and the intracellular side of the protein initiates activity of the G protein. The G protein is a guanosine triphosphate (GTP) hydrolase that physically moves from the receptor protein to the effector protein to activate the latter. An effector protein is an enzyme that catalyzes the generation of a new molecule, which acts as the intracellular mediator of the signal that binds to the receptor. This intracellular mediator is called the second messenger.

Different receptors use different second messengers. Two common examples of second messengers are cyclic adenosine monophosphate (cAMP) and inositol triphosphate (IP_3). The enzyme adenylate cyclase (an example of an effector protein) makes cAMP, and phospholipase C is the enzyme that makes IP_3. Second messengers, after they are produced by the effector protein, cause metabolic changes within the cell. These changes are most likely the activation of other enzymes in the cell. In neurons, they often modify ion channels, either opening or closing them. These enzymes can also cause changes in the cell, such as the activation of genes in the nucleus, and therefore the increased synthesis of proteins. In neurons, these kinds of changes are often the basis of stronger connections between cells at the synapse and may be the basis of learning and memory.

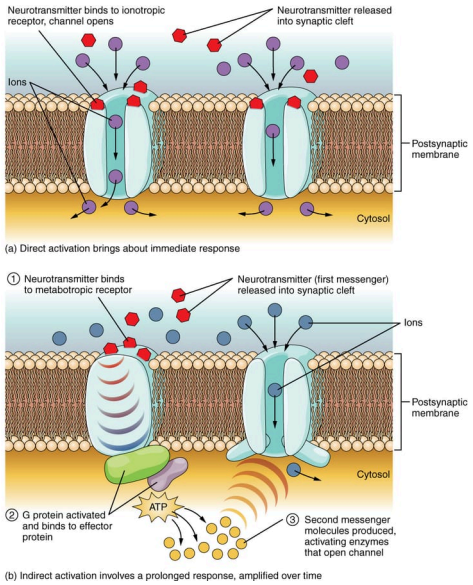


Figure 1.5.5: (a) An ionotropic receptor is a channel that opens when the neurotransmitter binds to it. (b) A metabotropic receptor is a complex that causes metabolic changes in the cell when the neurotransmitter binds to it (1). After binding, the G protein hydrolyzes ATP and moves to the effector protein (2). When the G protein contacts the effector protein, the latter is activated. In the case shown, the effector protein then acts on ATP to generate a second messenger, cAMP (3). The second messenger can then go on to cause changes in the neuron, such as opening or closing ion channels, metabolic changes, and changes in gene transcription.(CC BY 4.0; via LibreTexts). Alternate text description of the Figure 1.5.5.

Table 1.5.2: Source, Action and Examples of dysfunction of major neurotransmitters

Neurotransmitter	Source	Action	Example of Dysfunction
Acetylcholine (major transmitter of the parasympathetic nervous system)	Synthesized in cholinergic neurons (both in the central and peripheral nervous systems) from choline and acetyl-CoA.	At the neuromuscular junction, ACh binds to receptors, causing muscle fibers to contract. In the brain, it's crucial for memory, learning, attention, and executive functions like inhibitory control. Regulates functions like heart rate, digestion, and gland secretion.	• Low Acetylcholine: ◦ Alzheimer's Disease **(**memory loss, confusion, and concentration problems). ◦ Myasthenia Syndrome **(**muscle weakness, particularly in hips and legs). • Excess Acetylcholine (or inhibited breakdown): ◦ Pesticide/Nerve Gas Poisoning: Inhibition of acetylcholinesterase (the enzyme that breaks down ACh) causes ACh buildup, leading to muscle spasms, cramps, paralysis, and potential death.
Dopamine	Substantia nigra and basal ganglia	Typically inhibits and drives attention, memory, motivation, reward, fine motor control, sleep and focus.	Low level causes issues like Parkinson's (tremors, rigidity, balance issues, slow movement. Some aspects of ADHD (lack of focus, reduced motivation, fatigue, inability to feel pleasure. High/dysregulated Dopamine are associated with Schizophrenia (hallucinations, delusions); Impulse Control Disorders (gambling, binge eating, aggression, poor decision-making).

Neurotransmitter	Source	Action	Example of Dysfunction
Serotonin	Hypothalamus, brain stem, and dorsal horn of the spinal cord	Inhibitory effects and helps regulate mood, sleep patterns, sexuality, anxiety, appetite and inhibits pain pathways.	Normal level typically fall between 50 to 200 nanograms per milliliter (ng/mL) in the blood. When too low, are linked to depression, anxiety, sleep issues, OCD, and digestive problems like IBS, while excessively high levels can cause serotonin syndrome, a dangerous condition with symptoms like shivering, diarrhea, muscle rigidity, and fever, often triggered by drug interactions. Decreased levels lead to depression.
Gamma-aminobutyric acid (GABA)	Spinal cord, basal ganglia, cerebellum, and some cortical areas	Inhibitory effect.	Decreased levels lead to seizures. It regulates brain activity to prevent problems in the areas of anxiety, irritability, concentration, sleep, seizures and depression.
Norepinephrine (major transmitter of the sympathetic nervous system)	Brain stem, hypothalamus, and postganglionic neurons of the sympathetic nervous system	Typically excitatory; crucial for focus, mood, and stress response. Affects mood and overall activity levels.	With low levels linked to ADHD, depression, fatigue, and low blood pressure, while high levels are associated with anxiety, hypertension, rapid heart rate, insomnia, and stress-related disorders like PTSD.
Glutamate (Amino acid)	Most abundant neurotransmitter in the brain.	Excitatory; its metabolism is important to maintaining optimal levels within the extracellular space. As such, it is important for cognitive functions like thinking, learning and mood regulation.	Dysfunction interferes with brain processing of information. High levels (excitotoxicity) are associated with neurodegenerative diseases like Alzheimer's, Parkinson's, ALS, MS, stroke, and chronic pain, while low levels are implicated in psychiatric conditions such as depression, schizophrenia, and autism.

Disorders of the... Nervous System

The nervous system includes the brain, spinal cord, and nerves. It controls movement, feeling, thinking, memory, and many automatic functions like breathing and heart rate. When something goes wrong, it can affect almost any part of the body.

- Vascular (blood flow) problems: Stroke happens when a blood vessel in the brain is blocked or bursts. Brain cells die quickly. Symptoms can include sudden weakness on one side, trouble speaking, confusion, or vision loss. A TIA ("mini-stroke") has similar symptoms but they last a short time. Both are emergencies.
- Degenerative (wear-and-tear) diseases: In Parkinson's disease, certain brain cells that make dopamine are lost. This causes shaking at rest, stiff muscles, and slow movements. In Alzheimer's disease, brain cells are damaged, leading to memory loss, confusion, and changes in behavior. ALS (motor neuron disease) affects the nerve cells that control muscles. Over time, muscles become weak and wasted.
- Autoimmune and inflammatory disorders: In multiple sclerosis (MS), the immune system attacks the myelin (insulation) around nerve fibers in the brain and spinal cord. This can cause vision problems, numbness, weakness, and balance difficulty. Guillain-Barré syndrome is when the immune system attacks nerves in the body (peripheral nerves), often after an infection. It usually starts with leg weakness and can move upward but often improves with treatment.

- Seizure disorders: Epilepsy is a condition where sudden bursts of abnormal electrical activity in the brain cause seizures. A person may have shaking, staring spells, or brief loss of awareness. Many people with epilepsy can live normal lives with medication.
- Infections: Meningitis is an infection of the membranes covering the brain and spinal cord. Common signs are headache, fever, stiff neck, and confusion. Encephalitis is an infection of the brain tissue itself and may cause fever, seizures, and behavior changes. Some infections are caused by viruses, others by bacteria.
- Other common nervous system problems: Migraines are strong headaches with throbbing pain, often with nausea and sensitivity to light or sound. Bell's palsy is sudden weakness of the muscles on one side of the face, usually from inflammation of the facial nerve. Carpal tunnel syndrome happens when the median nerve is squeezed at the wrist, causing numbness and tingling in the hand.
- Warning signs: Important symptoms include sudden weakness or numbness, trouble speaking, severe or unusual headache, seizures, sudden vision changes, loss of balance, or strong confusion. These can be emergencies.
- Treatment: Treatment depends on the cause. It may include medicines (for seizures, pain, or to reduce inflammation), surgery (for tumors or bleeding), and rehabilitation (physical, occupational, or speech therapy). Healthy habits such as not smoking, exercising, controlling blood pressure and blood sugar, and wearing helmets and seat belts help protect the nervous system. Early medical help often improves the outcome.

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1.6: Terms and Definitions

Key Terms

Select to View Definitions

Absolute refractory period
time during an action period when another action potential cannot be generated because the voltage-gated Na^+ channel is inactivated

Action potential
change in voltage of a cell membrane in response to a stimulus that results in transmission of an electrical signal; unique to neurons and muscle fibers

Activation gate
part of the voltage-gated Na^+ channel that opens when the membrane voltage reaches threshold

Astrocyte
glial cell type of the CNS that provides support for neurons and maintains the blood-brain barrier

Aautonomic nervous system (ANS)
functional division of the nervous system that is responsible for homeostatic reflexes that coordinate control of cardiac and smooth muscle, as well as glandular tissue

Axon
single process of the neuron that carries an electrical signal (action potential) away from the cell body toward a target cell

Axon hillock
tapering of the neuron cell body that gives rise to the axon

Axon segment
single stretch of the axon insulated by myelin and bounded by nodes of Ranvier at either end (except for the first, which is after the initial segment, and the last, which is followed by the axon terminal)

Axon terminal
end of the axon, where there are usually several branches extending toward the target cell

Axoplasm
cytoplasm of an axon, which is different in composition than the cytoplasm of the neuronal cell body

Biogenic amine
class of neurotransmitters that are enzymatically derived from amino acids but no longer contain a carboxyl group

Bipolar
shape of a neuron with two processes extending from the neuron cell body—the axon and one dendrite

Blood-brain barrier (BBB)
physiological barrier between the circulatory system and the central nervous system that establishes a privileged blood supply, restricting the flow of substances into the CNS

Brain
the large organ of the central nervous system composed of white and gray matter, contained within the cranium and continuous with the spinal cord

Central Nervous System (CNS)
anatomical division of the nervous system located within the cranial and vertebral cavities, namely the brain and

spinal cord

Cerebral cortex
outermost layer of gray matter in the brain, where conscious perception takes place

Cerebrospinal fluid (CSF)
circulatory medium within the CNS that is produced by ependymal cells in the choroid plexus filtering the blood

Chemical synapse
connection between two neurons, or between a neuron and its target, where a neurotransmitter diffuses across a very short distance

Cholinergic system
neurotransmitter system of acetylcholine, which includes its receptors and the enzyme acetylcholinesterase

Choroid plexus
specialized structure containing ependymal cells that line blood capillaries and filter blood to produce CSF in the four ventricles of the brain

Continuous conduction
slow propagation of an action potential along an unmyelinated axon owing to voltage-gated Na^+ channels located along the entire length of the cell membrane

Dendrite
one of many branchlike processes that extends from the neuron cell body and functions as a contact for incoming signals (synapses) from other neurons or sensory cells

Depolarization
change in a cell membrane potential from rest toward zero

Effector protein
enzyme that catalyzes the generation of a new molecule, which acts as the intracellular mediator of the signal that binds to the receptor

Electrical synapse
connection between two neurons, or any two electrically active cells, where ions flow directly through channels spanning their adjacent cell membranes

Electrochemical exclusion
principle of selectively allowing ions through a channel on the basis of their charge

Enteric nervous system (ENS)
neural tissue associated with the digestive system that is responsible for nervous control through autonomic connections

Ependymal cell
glial cell type in the CNS responsible for producing cerebrospinal fluid

Excitable membrane
cell membrane that regulates the movement of ions so that an electrical signal can be generated

Excitatory postsynaptic potential (EPSP)
graded potential in the postsynaptic membrane that is the result of depolarization and makes an action potential more likely to occur

G protein
guanosine triphosphate (GTP) hydrolase that physically moves from the receptor protein to the effector protein to activate the latter

Ganglion
localized collection of neuron cell bodies in the peripheral nervous system

Gated
property of a channel that determines how it opens under specific conditions, such as voltage change or physical deformation

Generator potential
graded potential from dendrites of a unipolar cell which generates the action potential in the initial segment of that cell's axon

Glial cell
one of the various types of neural tissue cells responsible for maintenance of the tissue, and largely responsible for supporting neurons

Graded potential
change in the membrane potential that varies in size, depending on the size of the stimulus that elicits it

Gray matter
regions of the nervous system containing cell bodies of neurons with few or no myelinated axons; actually may be more pink or tan in color, but called gray in contrast to white matter

Inactivation gate
part of a voltage-gated Na^+ channel that closes when the membrane potential reaches +30 mV

Inhibitory postsynaptic potential (IPSP)
graded potential in the postsynaptic membrane that is the result of hyperpolarization and makes an action potential less likely to occur

Initial segment
first part of the axon as it emerges from the axon hillock, where the electrical signals known as action potentials are generated

Integration
nervous system function that combines sensory perceptions and higher cognitive functions (memories, learning, emotion, etc.) to produce a response

Ionotropic receptor
neurotransmitter receptor that acts as an ion channel gate, and opens by the binding of the neurotransmitter

Leakage channel
ion channel that opens randomly and is not gated to a specific event, also known as a non-gated channel

Ligand-gated channels
another name for an ionotropic receptor for which a neurotransmitter is the ligand

Lower motor neuron
second neuron in the motor command pathway that is directly connected to the skeletal muscle

Mechanically gated channel
ion channel that opens when a physical event directly affects the structure of the protein

Membrane potential
distribution of charge across the cell membrane, based on the charges of ions

Metabotropic receptor
neurotransmitter receptor that involves a complex of proteins that cause metabolic changes in a cell

Microglia
glial cell type in the CNS that serves as the resident component of the immune system

Multipolar
shape of a neuron that has multiple processes—the axon and two or more dendrites

Muscarinic receptor
type of acetylcholine receptor protein that is characterized by also binding to muscarine and is a metabotropic receptor

Myelin
lipid-rich insulating substance surrounding the axons of many neurons, allowing for faster transmission of electrical signals

Myelin sheath
lipid-rich layer of insulation that surrounds an axon, formed by oligodendrocytes in the CNS and Schwann cells in the PNS; facilitates the transmission of electrical signals

Nerve
cord-like bundle of axons located in the peripheral nervous system that transmits sensory input and response output to and from the central nervous system

Neuron
neural tissue cell that is primarily responsible for generating and propagating electrical signals into, within, and out of the nervous system

Neuropeptide
neurotransmitter type that includes protein molecules and shorter chains of amino acids

Neurotransmitter
chemical signal that is released from the synaptic end bulb of a neuron to cause a change in the target cell

Nicotinic receptor
type of acetylcholine receptor protein that is characterized by also binding to nicotine and is an ionotropic receptor

Node of Ranvier
gap between two myelinated regions of an axon, allowing for strengthening of the electrical signal as it propagates down the axon

Nonspecific channel
channel that is not specific to one ion over another, such as a nonspecific cation channel that allows any positively charged ion across the membrane

Nucleus
in the nervous system, a localized collection of neuron cell bodies that are functionally related; a “center” of neural function

Oligodendrocyte
glial cell type in the CNS that provides the myelin insulation for axons in tracts

Peripheral nervous system (PNS)
anatomical division of the nervous system that is largely outside the cranial and vertebral cavities, namely all parts except the brain and spinal cord

Postsynaptic potential (PSP)
graded potential in the postsynaptic membrane caused by the binding of neurotransmitter to protein receptors

Precentral gyrus of the frontal cortex
region of the cerebral cortex responsible for generating motor commands, where the upper motor neuron cell body is located

Process
in cells, an extension of a cell body; in the case of neurons, this includes the axon and dendrites

Propagation
movement of an action potential along the length of an axon

Receptor potential
graded potential in a specialized sensory cell that directly causes the release of neurotransmitter without an intervening action potential

Refractory period
time after the initiation of an action potential when another action potential cannot be generated

Relative refractory period
time during the refractory period when a new action potential can only be initiated by a stronger stimulus than the current action potential because voltage-gated K^+ channels are not closed

Repolarization
return of the membrane potential to its normally negative voltage at the end of the action potential

Resistance
property of an axon that relates to the ability of particles to diffuse through the cytoplasm; this is inversely proportional to the fiber diameter

Response
nervous system function that causes a target tissue (muscle or gland) to produce an event as a consequence to stimuli

Resting membrane potential
the difference in voltage measured across a cell membrane under steady-state conditions, typically -70 mV

Saltatory conduction
quick propagation of the action potential along a myelinated axon owing to voltage-gated Na^+ channels being present only at the nodes of Ranvier

Satellite cell
glial cell type in the PNS that provides support for neurons in the ganglia

Schwann cell
glial cell type in the PNS that provides the myelin insulation for axons in nerves

Sensation
nervous system function that receives information from the environment and translates it into the electrical signals of nervous tissue

Size exclusion
principle of selectively allowing ions through a channel on the basis of their relative size

Soma
in neurons, that portion of the cell that contains the nucleus; the cell body, as opposed to the cell processes (axons and dendrites)

Somatic nervous system (SNS)
functional division of the nervous system that is concerned with conscious perception, voluntary movement, and skeletal muscle reflexes

Spatial summation
combination of graded potentials across the neuronal cell membrane caused by signals from separate presynaptic elements that add up to initiate an action potential

Spinal cord
organ of the central nervous system found within the vertebral cavity and connected with the periphery through spinal nerves; mediates reflex behaviors

Stimulus
an event in the external or internal environment that registers as activity in a sensory neuron

Summate
to add together, as in the cumulative change in postsynaptic potentials toward reaching threshold in the membrane, either across a span of the membrane or over a certain amount of time

Synapse
narrow junction across which a chemical signal passes from neuron to the next, initiating a new electrical signal in the target cell

Synaptic cleft
small gap between cells in a chemical synapse where neurotransmitter diffuses from the presynaptic element to the postsynaptic element

Synaptic end bulb
swelling at the end of an axon where neurotransmitter molecules are released onto a target cell across a synapse

Temporal summation
combination of graded potentials at the same location on a neuron resulting in a strong signal from one input

Thalamus
region of the central nervous system that acts as a relay for sensory pathways

Thermoreceptor
type of sensory receptor capable of transducing temperature stimuli into neural action potentials

Threshold
membrane voltage at which an action potential is initiated

Tract
bundle of axons in the central nervous system having the same function and point of origin

Unipolar
shape of a neuron which has only one process that includes both the axon and dendrite

Upper motor neuron
first neuron in the motor command pathway with its cell body in the cerebral cortex that synapses on the lower motor neuron in the spinal cord

Ventricle
central cavity within the brain where CSF is produced and circulates

Voltage-gated channel
ion channel that opens because of a change in the charge distributed across the membrane where it is located

White matter
regions of the nervous system containing mostly myelinated axons, making the tissue appear white because of the high lipid content of myelin

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1.7: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. Which of the following human body cavities contains a component of the central nervous system?
   A. Abdominal
   B. Pelvic
   C. Cranial
   D. Thoracic

2. Which structure predominates in the white matter of the brain?
   A. Myelinated axons
   B. Neuronal cell bodies
   C. Ganglia of the parasympathetic nerves
   D. Bundles of dendrites from the enteric nervous system

3. Which part of a neuron propagates an electrical signal (action potential) away from the cell body toward a target cell?
   A. Dendrites
   B. Synapse
   C. Myelin sheath
   D. Axon

4. Which term describes a bundle of axons in the peripheral nervous system?
   A. Nucleus
   B. Ganglion
   C. Tract
   D. Nerve

5. Which functional division of the nervous system is responsible for the physiological changes seen during exercise (for example, increased heart rate and sweating)?
   A. Somatic
   B. Autonomic
   C. Enteric
   D. Central

6. What type of glial cell provides myelin for the axons in a tract in the CNS?
   A. Oligodendrocyte
   B. Astrocyte
   C. Schwann cell
   D. Satellite cell

7. Which part of a neuron contains the nucleus?
   A. Dendrite
   B. Soma
   C. Axon
   D. Synaptic end bulb

8. Which of the following substances is least able to cross the blood–brain barrier under normal conditions?
   A. Water
   B. Sodium ions
   C. Glucose
   D. White blood cells

9. What type of glial cell is the resident macrophage behind the blood–brain barrier?
   A. Microglia
   B. Astrocyte
   C. Schwann cell
   D. Satellite cell

10. What two types of macromolecules are the main components of myelin?
    A. Carbohydrates and lipids
    B. Proteins and nucleic acids
    C. Lipids and proteins
    D. Carbohydrates and nucleic acids

11. You step into a shower and quickly pull back to adjust the faucet because the water is too hot. At which location is the greatest level of integration for this response taking place?
    A. Skeletal muscle
    B. Spinal cord
    C. Thalamus
    D. Cerebral cortex

12. What ion enters a neuron and causes depolarization of the cell membrane during the rising phase of the action potential?
    A. Sodium
    B. Chloride
    C. Potassium
    D. Phosphate

13. Voltage-gated Na^+ channels open upon reaching what state?
    A. Resting potential
    B. Threshold
    C. Repolarization
    D. Overshoot

14. What does a ligand-gated channel require in order to open?
    A. Increase in concentration of Na^+ ions
    B. Binding of a neurotransmitter
    C. Increase in concentration of K^+ ions
    D. Depolarization of the membrane

15. What does a mechanically gated channel respond to?
    A. Physical stimulus
    B. Chemical stimulus
    C. Increase in resistance
    D. Decrease in resistance

16. Which of the following voltages would most likely be measured during the relative refractory period?
    A. +30 mV
    B. 0 mV
    C. –45 mV
    D. –80 mV

17. Which of the following is likely to propagate an action potential the fastest?
    A. A thin, unmyelinated axon
    B. A thin, myelinated axon
    C. A thick, unmyelinated axon

D. A thick, myelinated axon

18. How much of a depolarization at the axon hillock is typically needed for summation of postsynaptic potentials to reach threshold and generate an action potential (from –70 mV)?
A. +30 mV
B. +15 mV
C. +10 mV
D. 15 mV below rest

19. A channel opens on a postsynaptic membrane and allows a negative ion to enter the cell. What type of graded potential is this most likely to produce?
A. Depolarizing
B. Repolarizing
C. Hyperpolarizing
D. Non-polarizing

20. What neurotransmitter is released at the neuromuscular junction of skeletal muscle?
A. Norepinephrine
B. Serotonin
C. Dopamine
D. Acetylcholine

21. What type of receptor requires an effector protein (such as a G protein) to initiate a signal inside the cell?
A. Biogenic amine
B. Ionotropic receptor
C. Cholinergic system
D. Metabotropic receptor

22. Which of the following neurotransmitters is associated with inhibition almost exclusively in the adult CNS?
A. GABA
B. Acetylcholine
C. Glutamate
D. Norepinephrine

23. A 45-year-old woman presents with progressive muscle weakness and episodes of blurred vision. MRI shows multiple areas of demyelination in the white matter of the brain. Which glial cell type is primarily damaged in this condition?
A. Oligodendrocyte
B. Schwann cell
C. Astrocyte
D. Microglia

24. A patient suffers a deep cut to the forearm and later cannot feel light touch along one finger. Nerve conduction tests show slowed transmission in a peripheral nerve due to loss of myelin. Which cell type is most likely affected?
A. Oligodendrocyte
B. Schwann cell
C. Astrocyte
D. Ependymal cell

25. A 70-year-old man has slow, shuffling movements and a resting tremor. These symptoms are linked to loss of dopamine-producing neurons in a midbrain nucleus. Which neurotransmitter system is primarily disrupted?
A. Cholinergic
B. Glutamatergic
C. Dopaminergic
D. GABAergic

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1.8: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. When you run on a treadmill, your nervous system controls different kinds of tissues in your body. Choose one example for each type of tissue (skeletal muscle, smooth muscle, cardiac muscle, and a gland) and explain briefly what the nervous system makes that tissue do while you are running.
2. Think about eating a meal from start to finish: seeing and smelling the food, chewing, swallowing, and digesting it. Which parts of the nervous system are involved (central vs. peripheral; somatic, autonomic, enteric)? Name each division you use and explain in simple terms what it does in this situation.
3. Multiple sclerosis (MS) is a demyelinating disease in the central nervous system. Which glial cell is mainly damaged in MS, and how does this damage to that cell type interfere with fast communication between neurons?
4. There are different shapes of neurons: multipolar, bipolar, and unipolar (pseudounipolar). Which shape is best for passing information directly from one neuron to another inside the CNS, and what features of its structure make it good at this job?
5. Sensory pathways are called “afferent,” and motor pathways are called “efferent.” Based on these words, what can you say about the direction in which information travels in each pathway, and how does this relate to where the pathway starts and where it ends in the body?
6. A patient cannot move their arm on purpose, but the arm muscles still have normal tone and normal reflexes. Which is more likely damaged—an upper motor neuron or a lower motor neuron? Explain your answer using the role of each type of motor neuron.
7. In your own words, what does it mean to say that an action potential is an “all-or-none” event? How does this property change the way the nervous system shows the strength of a stimulus (for example, weak vs. strong touch)?
8. When you touch something very hot, you quickly pull your hand away, and only after that do you feel the pain clearly. Using what you know about action potential propagation and different pathways in the nervous system, explain why the conscious feeling of pain is slower than the fast reflex movement.
9. A postsynaptic neuron receives input from five presynaptic neurons. Three synapses are excitatory, and two synapses are inhibitory. Explain how spatial summation (inputs from different places) and/or temporal summation (inputs over time) at the axon hillock will decide whether this neuron reaches threshold and produces an action potential.
10. The same neurotransmitter, such as acetylcholine, can excite one cell but inhibit another. Why is the receptor on the postsynaptic cell the key factor that decides the effect of a neurotransmitter? Give a simple example comparing two different receptor types if you can.
    - Hint: Think about what happens inside the cell after the neurotransmitter binds. Does the receptor open an ion channel directly (for example, letting Na^+ in or Cl^- in), or does it activate a G protein and second messengers that can have different effects?

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1.9: Alternate Text Descriptions

Figure 1.1.1: The Human Nervous System

The figure shows the human nervous system divided into two major components: the **central nervous system (CNS)** and the **peripheral nervous system (PNS)**.

1. The **central nervous system** consists of the **brain**, located in the head, and the **spinal cord**, which extends downward from the brain through the trunk.
2. The **peripheral nervous system** consists of numerous **nerves** that branch outward from the spinal cord and extend throughout the body, including the trunk, arms, hands, legs, and feet.
3. The diagram shows the spinal cord as the central pathway connecting the brain to the peripheral nerves.
4. A **ganglion**, identified as a cluster of nerve cell bodies, is labeled along one of the peripheral nerve pathways.
5. The nerves repeatedly divide into smaller branches as they extend away from the spinal cord, illustrating how the peripheral nervous system forms a widespread communication network linking the central nervous system with the rest of the body.

Figure 1.1.2: Human Brain

An anatomical photograph displays a human brain from a superior view, showing the two cerebral hemispheres. A section of the cerebral cortex has been removed to reveal the distinct internal tissue layers. The diagram contains text annotations identifying two primary structural components: gray matter, which forms the outer surface layer of the cortex, and white matter, which lies deep beneath the superficial gray tissue.

Figure 1.1.3: Multiple Meanings of the Term "Nucleus"

This figure is a three-panel educational figure compares the meaning of the term “nucleus” in different scientific contexts.

- Left panel (a): Nucleus of an atom. A helium atom is shown. At the center is the nucleus, made up of two protons and two neutrons represented by colored spheres. Surrounding the nucleus is a larger, faint circular region labeled “electron cloud,” representing the region occupied by the atom's electrons. The word “Nucleus” appears above the central cluster, and a legend identifies the proton, neutron, and electron cloud.
- Middle panel (b): Nucleus of a cell. A diagram of a cell is shown with various internal structures. A large oval-shaped structure labeled “Nucleus” occupies much of the cell's interior. Smaller organelles surround the nucleus within the cell.
- Right panel (c): Nucleus in the brain. A medical image of a human brain is shown. Two small regions near the center of the brain are outlined in red, highlighting examples of nuclei, which are clusters of nerve cell bodies within the central nervous system. The caption below reads “Nucleus in the brain.”

The figure demonstrates that the term “nucleus” can refer to the center of an atom, the membrane-bound control center of a cell, or a cluster of neurons within the brain.

Figure 1.1.4: Visual Pathway from the Eyes to the Brain

The figure illustrates the visual pathway that carries information from the eyes to the brain.

1. The left and right eyes connect to the brain through the optic nerves.
2. The optic nerves meet at the optic chiasma, where some nerve fibers cross to the opposite side while others remain on the same side.
3. Beyond the optic chiasma, the pathways continue as the optic tracts.
4. The optic tracts project to the thalamus and then continue to the occipital lobes at the back of the brain, where visual information is processed.
5. The midbrain is also labeled along the visual pathway.
6. An inset shows a side view of the brain with the visual pathway highlighted. An arrow indicates that the main diagram is an

enlarged view of this region.

The figure demonstrates how visual information from both eyes is partially crossed at the optic chiasma and then distributed to both sides of the brain for processing.

Figure 1.1.5: Major Divisions of the Human Nervous System

The figure shows the major divisions of the human nervous system and their primary functions.

1. The **brain**, part of the **central nervous system (CNS)**, is located in the head and is responsible for processing sensory information, controlling voluntary motor responses, and regulating homeostasis through autonomic functions.
2. The **spinal cord**, also part of the **CNS**, extends downward from the brain through the trunk. It serves as a pathway for sensory and motor information between the brain and the rest of the body and is involved in reflex responses.
3. The **peripheral nervous system (PNS)** consists of nerves that branch from the spinal cord throughout the body. These nerves carry sensory information to the CNS and motor commands from the CNS to body tissues.
4. **Ganglia**, part of the **PNS**, are clusters of nerve cell bodies located along peripheral nerve pathways. They receive sensory information and relay autonomic motor signals.
5. The **enteric nervous system (ENS)** is shown within the digestive tract. It controls digestive functions and can operate independently of the brain and spinal cord.

The figure demonstrates how the central, peripheral, and enteric nervous systems work together to receive sensory information, coordinate responses, regulate body functions, and control digestion.

Figure 1.1.6: Functional Brain Imaging Showing Areas of Neural Activity

The figure shows an axial, or horizontal, brain image from a functional neuroimaging study. Several highlighted regions are overlaid on the brain image, indicating areas of increased neural activity or signal intensity. Most of the highlighted activity is located within one hemisphere, with a few smaller regions appearing closer to the center of the brain.

Two intersecting lines mark a reference point within the image. The underlying grayscale anatomy provides a cross-sectional view of brain structures.

The figure demonstrates the localization of neural activity within specific regions of the brain, illustrating that functional imaging techniques can identify areas that are more active during a particular task or condition.

Figure 1.2.1: Structure of a Neuron and Signal Transmission Pathway

The figure shows the structure of a neuron and the direction of information flow through the cell.

1. **Dendrites** branch from the cell body and receive incoming signals from other neurons.
2. The **cell body (soma)** contains the nucleus and integrates incoming information.
3. The **cell membrane** surrounds the neuron and helps regulate movement of substances into and out of the cell.
4. An **axon** extends from the cell body and carries electrical signals away from the soma.
5. Portions of the axon are covered by **myelin sheaths**, which insulate the axon and increase the speed of signal transmission.
6. Gaps between adjacent myelin sheaths are labeled **nodes of Ranvier**, where the axon is exposed.
7. An **oligodendrocyte** is shown forming multiple myelin sheath segments around the axon.
8. At the end of the axon, the **synapse** represents the site where the neuron communicates with another cell.

The figure demonstrates how signals travel through a neuron from the dendrites to the cell body, along the axon, and ultimately to the synapse, while myelin sheaths and nodes of Ranvier facilitate efficient signal conduction.

Figure 1.2.2: Structural Classification of Neurons: Unipolar, Bipolar, and Multipolar

The figure is a three-part diagram comparing common structural classifications of neurons based on the number of processes

extending from the cell body.

1. **Unipolar neuron:** The neuron has a single process extending from the cell body that branches into two directions. One branch extends toward sensory receptors, and the other extends toward the central nervous system. The cell body is positioned off to the side of the main process.
2. **Bipolar neuron:** The neuron has two processes extending from opposite sides of the cell body. One process functions as a dendrite and the other as an axon, creating a linear arrangement.
3. **Multipolar neuron:** The neuron has a single axon and multiple dendrites extending from the cell body. Numerous branching processes surround the soma, making this the most structurally complex neuron shown.

The figure demonstrates how neurons are classified according to the number of processes attached to the cell body, with unipolar neurons having one process, bipolar neurons having two processes, and multipolar neurons having one axon and multiple dendrites.

Figure 1.2.3: Specialized Neuron Types in the Nervous System

The figure compares three specialized neuron types found in different regions of the nervous system.

1. **Panel (a): Pyramidal cell of the cerebral cortex.** The neuron has a triangular-shaped cell body with multiple branching dendrites extending from its sides. A single prominent dendrite extends upward and branches near the top, while a long axon extends downward from the base of the cell body. The elongated shape reflects its role in communication within the cerebral cortex.
2. **Panel (b): Purkinje cell of the cerebellar cortex.** The neuron has a cell body positioned near the bottom of the panel and a dense, highly branched dendritic network extending upward. The extensive tree-like arrangement of dendrites occupies most of the panel and distinguishes this neuron type. A single axon extends away from the cell body.
3. **Panel (c): Olfactory cells in the olfactory epithelium and olfactory bulbs.** Sensory neurons are shown extending between the olfactory epithelium at the bottom and the olfactory bulb above. Each neuron has processes that detect odor molecules in the epithelium and transmit sensory information to structures within the olfactory bulb. Dashed circles highlight the connections formed within the olfactory bulb.

The figure demonstrates that neurons vary considerably in shape and organization depending on their location and function. Pyramidal cells are characterized by a prominent apical dendrite, Purkinje cells by extensive branching dendrites, and olfactory neurons by specialized sensory connections between the olfactory epithelium and olfactory bulb.

Figure 1.2.4: Major Neuroglial Cell Types and Their Functions

The figure illustrates several major types of neuroglial, or glial, cells and their relationships to neurons within the central nervous system.

1. A large neuron is shown with a cell body, branching dendrites, and a long axon.
2. **Astrocytes** surround the neuron and extend numerous processes that contact both neurons and nearby blood vessels, helping provide structural support and regulate the neuronal environment.
3. **Microglial cells** are smaller cells with highly branched projections located near neurons. These cells serve immune and protective functions within nervous tissue.
4. **Oligodendrocytes** are associated with the neuron's axon and form **myelin sheaths**, shown as segmented coverings wrapped around the axon. These sheaths insulate the axon and increase the speed of nerve signal transmission.
5. **Ependymal cells** form a layer of cells lining a cavity, representing the lining of fluid-filled spaces within the brain and spinal cord.

The figure demonstrates how different types of glial cells support nervous system function by providing structural support, immune protection, insulation of axons, regulation of the neuronal environment, and maintenance of fluid-filled spaces in the central nervous system.

Figure 1.2.5: Peripheral Ganglion Neuron with Satellite Cells and Schwann Cells

The figure shows a peripheral ganglion neuron and the glial cells that support it in the peripheral nervous system.

1. A **peripheral ganglionic neuron cell body (unipolar cell)** is shown at the top of the figure.
2. The neuron cell body is surrounded by **satellite cells**, which form a protective and supportive layer around the neuron.
3. A single **axon** extends from the neuron cell body.
4. The axon is wrapped by multiple **Schwann cells** arranged along its length.
5. Schwann cells form insulating coverings around the axon that support and increase the speed of nerve signal conduction.

The figure demonstrates the organization of glial support cells in the peripheral nervous system. Satellite cells surround and support neuron cell bodies within ganglia, while Schwann cells surround axons and provide insulation that facilitates efficient nerve signal transmission.

Figure 1.2.6: Myelin Formation and Structure of a Myelinated Nerve Fiber

The figure compares a conceptual diagram of myelin formation with a microscopic image of a myelinated nerve fiber.

1. **Panel (a): Formation of the myelin sheath.**
   - A series of three diagrams shows a supporting cell wrapping its cell membrane around an axon.
   - In the first diagram, the cell begins to surround the axon, and the cell nucleus remains visible within the wrapping cell.
   - In the second diagram, additional layers of membrane are wrapped around the axon.
   - In the third diagram, multiple layers form a thick **myelin sheath** around the axon. A gap between adjacent myelin segments is labeled the **node of Ranvier**, where the axon remains exposed.
2. **Panel (b): Electron micrograph of a myelinated nerve fiber.**
   - A magnified image shows an **axon** surrounded by multiple layers of **myelin sheath**.
   - The **axolemma**, or axon membrane, forms the boundary of the axon.
   - Outside the myelin sheath is the **external lamina**.
   - The outermost surrounding tissue is labeled **endoneurium (collagen)**.
   - An inset indicates the location of the magnified region within a larger tissue sample.

The figure demonstrates how myelin is formed by repeated wrapping of a cell membrane around an axon and how this layered structure appears under a microscope. The myelin sheath insulates nerve fibers and supports rapid transmission of nerve impulses, while nodes of Ranvier provide gaps that facilitate signal conduction.

Figure 1.3.1: Sensory and Motor Pathways in Nervous System Processing

The figure illustrates the sequence of events by which the nervous system detects a sensory stimulus, processes the information in the brain, and produces a voluntary motor response.

1. Sensory receptors in the skin of the hand detect the temperature of water flowing over the fingers.
2. A sensory neuron carries the resulting action potential from the hand toward the spinal cord.
3. The sensory neuron enters the spinal cord and synapses with neurons that relay the signal toward the brain.
4. The sensory pathway ascends to the thalamus, which serves as a relay center for sensory information.
5. The sensory signal reaches the cerebral cortex, where conscious perception of the temperature occurs.
6. In response, an upper motor neuron in the cerebral cortex generates a motor command.
7. The motor signal travels downward through the brain and spinal cord, where the upper motor neuron synapses with a lower motor neuron.
8. The lower motor neuron carries the motor signal to the hand and activates skeletal muscle fibers at the neuromuscular junction, producing muscle contraction and movement.

The figure demonstrates how sensory information travels from receptors in the skin to the brain for conscious perception and how the brain generates motor commands that travel through motor pathways to produce a voluntary response.

Figure 1.3.2: Sensory Receptor Detecting a Temperature Stimulus

The figure shows the first stage of sensory processing, in which a sensory receptor detects a stimulus and initiates a nerve signal.

1. A hand is held beneath a stream of flowing water, representing a temperature stimulus applied to the skin.
2. A **sensory receptor** located in the fingertip detects the temperature of the water.
3. A sensory nerve fiber extends from the receptor through the finger and hand toward the forearm.
4. The sensory receptor converts the environmental stimulus into a signal that can be transmitted through the nervous system.

The figure demonstrates how sensory information begins when receptors in the skin detect a stimulus and transmit signals through sensory neurons toward the central nervous system.

Figure 1.3.3: Motor Response Through Skeletal Muscle Activation

The figure illustrates the motor response portion of a nervous system pathway.

1. A hand is positioned near hot and cold water faucet handles, indicating an action to adjust water temperature.
2. A second hand is held beneath a stream of flowing water, representing the body part receiving the stimulus.
3. A **motor neuron** extends from the nervous system to the hand and forearm.
4. The motor neuron connects to **skeletal muscles** through specialized junctions labeled **neuromuscular junctions**.
5. Signals carried by the motor neuron activate skeletal muscle fibers, causing muscle contraction and movement of the hand.
6. The movement allows the individual to adjust the faucet in response to sensory information about the water temperature.

The figure demonstrates how motor commands are transmitted from the nervous system through motor neurons to skeletal muscles, producing a voluntary movement in response to a sensory stimulus.

Figure 1.4.1: Cell Membrane Structure and Channel Proteins

The figure illustrates the structure of the cell membrane and the role of membrane proteins in transporting substances across it.

1. The cell membrane consists of a **phospholipid bilayer**, with phospholipid heads facing the extracellular fluid and cytosol and phospholipid tails forming the membrane interior.
2. The interior of the bilayer is labeled the **hydrophobic core**, which acts as a barrier to many water-soluble substances.
3. The region above the membrane is labeled **extracellular fluid**, and the region below is labeled **cytosol**.
4. Several **channel proteins** are embedded within the phospholipid bilayer.
5. One channel protein contains a **pore** that extends through the membrane, providing a pathway for selected substances to move between the extracellular fluid and the cytosol.
6. Other membrane proteins are shown embedded in the membrane, illustrating that multiple types of proteins can be associated with the phospholipid bilayer.

The figure demonstrates that the phospholipid bilayer forms a selective barrier between the cell interior and the external environment, while channel proteins provide regulated pathways that allow specific substances to cross the membrane.

Figure 1.4.2: Ligand-Gated Ion Channel Opening in Response to Neurotransmitter Binding

The figure compares a ligand-gated ion channel in its closed and open states, illustrating how neurotransmitter binding controls the movement of ions across the cell membrane.

1. **Left panel: Channel closed.**
   - A phospholipid bilayer separates the extracellular fluid from the cytosol.
   - A channel protein spans the membrane but remains closed.
   - A neurotransmitter receptor site is located on the channel protein.
   - Sodium ions (Na^+) and calcium ions (Ca^{2+}) are shown at higher concentrations outside the cell, while potassium ions (K^+) are shown at higher concentrations inside the cell.

- The channel remains closed until a neurotransmitter binds to the receptor.

2. **Right panel: Channel open.**
   - A neurotransmitter binds to the receptor site on the channel protein.
   - This chemical stimulus causes the channel to open, creating a pore through the membrane.
   - Na^+ and Ca^{2+} move into the cell through the open channel.
   - K^+ moves out of the cell through the channel.
   - Ion movement occurs according to each ion's concentration gradient.

The figure demonstrates how ligand-gated ion channels open in response to neurotransmitter binding, allowing specific ions to move across the cell membrane and contributing to changes in the electrical state of the cell.

Figure 1.4.3: Mechanically Gated Ion Channel Response to Mechanical Stimuli

The figure compares a mechanically gated ion channel in its closed and open states, illustrating how a mechanical stimulus allows ions to move across the cell membrane.

1. **Left panel: Mechanically gated channel closed.**
   - A phospholipid bilayer separates the **extracellular fluid** from the **cytosol**.
   - A mechanically gated channel protein spans the membrane but remains closed, preventing ions from passing through.
   - Sodium ions (**Na^+**) are shown at a higher concentration outside the cell, while potassium ions (**K^+**) are shown at a higher concentration inside the cell.
2. **Right panel: Mechanically gated channel open.**
   - A mechanical stimulus causes the channel to open, creating a pore through the membrane.
   - Sodium ions (**Na^+**) move into the cell through the open channel, following their concentration gradient.
   - Potassium ions (**K^+**) move out of the cell through the open channel, following their concentration gradient.
   - Arrows indicate the direction of ion movement across the membrane.

The figure demonstrates how mechanically gated ion channels respond to physical stimuli by opening a pore in the cell membrane. Once the channel opens, ions move according to their concentration gradients, contributing to changes in the cell's electrical state.

Figure 1.4.4: Voltage-Gated Ion Channel Opening in Response to Membrane Depolarization

The figure compares a voltage-gated ion channel in its closed and open states, illustrating how changes in membrane potential regulate ion movement across the cell membrane.

1. **Left panel: Voltage-gated channel closed.**
   - A voltage-gated channel protein spans the cell membrane, separating the **extracellular fluid** from the **cytosol**.
   - The channel is closed when the membrane potential is **−70 mV**.
   - Ions cannot pass through the channel while it remains closed.
2. **Right panel: Voltage-gated channel open.**
   - A change in membrane potential to **−50 mV** causes the channel to open.
   - The opening creates a pore through the membrane that allows ions to move across the cell membrane.
   - An ion is shown passing through the channel in response to its concentration gradient.
   - Arrows indicate ion movement through the open channel.
3. A large arrow labeled **concentration gradient** indicates that ions move according to differences in concentration across the membrane once the channel is open.

The figure demonstrates that voltage-gated ion channels respond to changes in transmembrane voltage. When the membrane potential reaches a specific threshold, the channel changes shape and opens, allowing selected ions to cross the membrane and contribute to electrical signaling in neurons.

Figure 1.4.5: Ion Movement Through a Leakage Channel

The figure compares a leakage ion channel in its closed and open states, illustrating how ions can move across the cell membrane when the channel is open.

1. **Left panel: Leakage channel closed.**
   - A leakage channel protein spans the cell membrane, which separates the **extracellular fluid** from the **cytosol**.
   - The channel is shown in a closed state, preventing ions from moving through it.
   - Ions are present on both sides of the membrane, with a higher concentration shown on one side.
2. **Right panel: Leakage channel randomly opens.**
   - The leakage channel is shown in an open state, creating a pore through the cell membrane.
   - Ions move through the open channel according to their **concentration gradient**.
   - Arrows indicate the direction of ion movement across the membrane.
   - The channel opens without the need for a neurotransmitter, mechanical stimulus, or change in membrane voltage.
3. A large arrow labeled **concentration gradient** emphasizes that ion movement through leakage channels is driven by differences in ion concentration across the membrane.

The figure demonstrates that leakage channels can open spontaneously, allowing ions to move across the cell membrane according to their concentration gradients. These channels contribute to the resting membrane potential by permitting a continuous, regulated movement of ions.

Figure 1.4.6: Measuring Resting Membrane Potential Across a Cell Membrane

The figure illustrates how membrane potential is measured across a cell membrane using electrodes and a voltmeter.

1. A **phospholipid bilayer** separates the **extracellular fluid** from the **cytosol**.
2. Positive charges are shown along the extracellular side of the membrane, while negative charges are shown along the cytosolic side, illustrating a difference in electrical charge across the membrane.
3. A **reference electrode** is positioned in the extracellular fluid outside the cell.
4. A **recording microelectrode** passes through the cell membrane into the cytosol.
5. Both electrodes are connected to a **voltmeter**, which measures the electrical potential difference between the inside and outside of the cell.
6. The voltmeter scale ranges from **–70 mV** to **+70 mV**, with **0 mV** at the midpoint. The needle points toward the negative side of the scale, indicating that the inside of the cell is negative relative to the outside.

The figure demonstrates how resting membrane potential is measured by comparing the electrical charge inside and outside a cell. The separation of positive and negative charges across the membrane creates a voltage difference that can be detected using electrodes and displayed on a voltmeter.

Figure 1.4.7: Phases of an Action Potential

The figure shows a graph of membrane potential over time during an action potential.

1. The vertical axis represents **membrane potential (mV)**, ranging from approximately **–70 mV** to **+30 mV**. The horizontal axis represents **time**.
2. The graph begins at a resting membrane potential of approximately **–70 mV**, shown as a flat line.
3. A dashed horizontal line at **–55 mV** marks the **threshold potential**.
4. Once the membrane potential reaches threshold, the graph rises rapidly in a phase labeled **depolarization**, increasing from –55 mV to a peak of approximately **+30 mV**.
5. Following the peak, the membrane potential decreases rapidly in a phase labeled **repolarization**, returning toward the resting level.
6. The membrane potential briefly falls below the resting level to approximately **–80 mV** in a phase labeled **hyperpolarization**.
7. After hyperpolarization, the membrane potential gradually returns to the resting value of approximately **–70 mV**.

The figure demonstrates the sequence of changes in membrane potential during an action potential. When the membrane

reaches threshold, rapid depolarization occurs, followed by repolarization and a brief period of hyperpolarization before the neuron returns to its resting membrane potential.

Figure 1.4.8: Stages of an Action Potential

The figure shows a graph of membrane potential over time, illustrating the sequence of events in an action potential.

1. **At rest (1):** The membrane potential begins at approximately **–70 mV**, representing the neuron's resting membrane potential.
2. **Stimulus applied (2):** A stimulus causes the membrane potential to begin rising from the resting level.
3. **Voltage rises (3):** The membrane potential reaches the **threshold** value of approximately **–55 mV** and rapidly depolarizes, rising to a peak of about **+30 mV**.
4. **Voltage falls (4):** Following the peak, the membrane potential rapidly decreases during repolarization as the cell returns toward its resting state.
5. **End of action potential (5):** The membrane potential briefly falls below the resting level, creating a period of hyperpolarization.
6. **Return to rest (6):** The membrane potential gradually returns to approximately **–70 mV**, restoring the resting membrane potential.

A dashed horizontal line labeled **Threshold** marks the membrane potential that must be reached to trigger an action potential.

The figure demonstrates the major stages of an action potential: resting state, stimulation, depolarization, repolarization, hyperpolarization, and return to the resting membrane potential.

Figure 1.5.1: Depolarizing and Hyperpolarizing Graded Potentials

The figure shows examples of **graded potentials** and how their size and duration vary with the strength and type of stimulus.

1. The vertical axis represents **membrane potential (mV)**, ranging from approximately **–90 mV** to **–40 mV**. The horizontal axis represents **time**.
2. A dashed horizontal line labeled **Threshold** is shown at approximately **–55 mV**.
3. The membrane potential begins at a resting value of approximately **–70 mV**.

Depolarizing Graded Potentials

1. Several upward deflections from the resting membrane potential illustrate **depolarizing graded potentials**.
2. A small stimulus produces a small depolarization from the resting level.
3. A larger stimulus produces a greater depolarization.
4. A stimulus of longer duration produces a depolarization that lasts longer but does not necessarily reach a greater magnitude.
5. The largest depolarization reaches and slightly exceeds the threshold value, indicating that a sufficiently strong stimulus may trigger an action potential in a postsynaptic neuron.

Hyperpolarizing Graded Potentials

1. Several downward deflections below the resting membrane potential illustrate **hyperpolarizing graded potentials**.
2. These responses make the membrane potential more negative than the resting level.
3. Hyperpolarization varies in magnitude and duration depending on which ion channels are activated.

The figure demonstrates that graded potentials vary in size and duration according to stimulus strength and type. Depolarizing graded potentials move the membrane potential closer to threshold and may initiate an action potential if threshold is reached, whereas hyperpolarizing graded potentials move the membrane potential farther from threshold and reduce the likelihood of generating an action potential.

Figure 1.5.2: Summation of EPSPs and IPSPs at the Axon Hillock

Figure illustrates how excitatory and inhibitory postsynaptic potentials are integrated at the axon hillock and how their combined effects can trigger an action potential.

1. A neuron is shown above the graph with multiple synaptic inputs contacting its dendrites and cell body.
2. Green inputs represent **excitatory postsynaptic potentials (EPSPs)**, which depolarize the membrane and move the membrane potential closer to threshold.
3. Pink inputs represent **inhibitory postsynaptic potentials (IPSPs)**, which hyperpolarize the membrane or oppose depolarization.

A. Postsynaptic Potentials

1. The graph plots **membrane potential (mV)** on the vertical axis and **time** on the horizontal axis.
2. The membrane potential begins near the resting level of **–70 mV**.
3. Several EPSPs produce small upward deflections of the membrane potential, moving it toward threshold.
4. IPSPs produce downward deflections that counteract the effects of EPSPs.
5. The membrane potential fluctuates as excitatory and inhibitory inputs are combined, demonstrating the process of synaptic integration.

B. Summation at the Axon Hillock

1. Near the axon hillock, multiple EPSPs occur in close succession and summate, causing the membrane potential to rise to the **threshold** value of approximately **–55 mV**.
2. Once threshold is reached, an **action potential** is generated, shown as a rapid spike in membrane potential to approximately **+30 mV**.
3. Following the action potential, the membrane potential rapidly decreases, briefly becomes more negative than the resting potential (hyperpolarization), and then gradually returns to the resting level.

The figure demonstrates how neurons integrate excitatory and inhibitory synaptic inputs. The combined effects of EPSPs and IPSPs are summed at the axon hillock, and an action potential is generated only when the membrane potential reaches threshold.

Figure 1.5.3: Neuron Structure and Direction of Neural Signal Transmission

The figure illustrates the structure of a neuron and the direction of signal transmission between neurons.

1. A large neuron occupies the upper portion of the figure. The neuron consists of a **cell body (soma)** containing a **nucleus**, numerous branching **dendrites**, and a single **axon** extending away from the cell body.
2. The **dendrites** branch outward from the cell body and serve as the primary regions for receiving incoming signals from other neurons.
3. The **nucleus** is located within the center of the cell body and is enclosed by the soma.
4. A single **axon** extends from the cell body and carries signals away from the neuron.
5. Portions of the axon are covered by segmented **myelin sheaths**, which insulate the axon and support more rapid transmission of nerve impulses.
6. An arrow labeled **Direction of signal** indicates that information travels from the dendrites and cell body, down the axon, toward the axon terminals.
7. At the end of the axon, multiple branches form **presynaptic axon terminals**.
8. The presynaptic axon terminals make contact with neighboring neurons shown in the lower portion of the figure. These contact points are labeled **axon terminals** and represent sites where signals are transmitted from one neuron to another.
9. Two additional neurons are shown below the first neuron, illustrating how one neuron's axon terminals communicate with the dendrites and cell bodies of other neurons within a neural network.

The figure demonstrates the major structural components of a neuron and the direction of information flow through the nervous system. Signals are received by dendrites, processed in the cell body, conducted along the axon, and transmitted through axon terminals to other neurons.

Figure 1.5.4: Comparison of Chemical and Electrical Synapses

The figure compares the structure and function of **chemical synapses** and **electrical synapses**.

Chemical Synapse

1. A **presynaptic neuron** is separated from a **postsynaptic neuron** by a narrow space labeled the **synaptic cleft**.
2. Within the presynaptic terminal, **synaptic vesicles** contain neurotransmitter molecules.
3. When neurotransmitters are released from the synaptic vesicles, they diffuse across the synaptic cleft.
4. The enlarged inset shows neurotransmitter molecules moving through the synaptic cleft and binding to receptors on the postsynaptic membrane.
5. Binding of neurotransmitters allows ions to move through membrane channels in the postsynaptic cell, generating a postsynaptic response.
6. Communication occurs through the release and detection of chemical messengers.

Electrical Synapse

1. The presynaptic and postsynaptic neurons are connected directly by **gap junctions**.
2. The enlarged inset shows **connexons**, protein channels that form a continuous pathway between the two cells.
3. **Ions** move directly from one cell to the other through the connexons.
4. Because the cells are physically connected, electrical signals pass rapidly between neurons without the release of neurotransmitters.
5. Communication occurs through direct ionic current flow between cells.

The figure demonstrates two mechanisms of communication between neurons. In a **chemical synapse**, signals are transmitted by neurotransmitters crossing a synaptic cleft and activating receptors on the postsynaptic cell. In an **electrical synapse**, ions pass directly between cells through gap junctions, allowing rapid electrical communication.

Figure 1.5.5: Ionotropic and Metabotropic Receptor Signaling Pathways

The figure compares **ionotropic** and **metabotropic** receptor signaling at a postsynaptic membrane.

(a) Ionotropic Receptor: Direct Activation

1. Neurotransmitters are released into the synaptic cleft and bind directly to **ionotropic receptors** located on the postsynaptic membrane.
2. The ionotropic receptor functions as an ion channel.
3. When neurotransmitters bind to the receptor, the channel opens immediately.
4. Ions move through the open channel across the postsynaptic membrane.
5. The direct opening of the channel produces a rapid postsynaptic response.

The top panel demonstrates that ionotropic receptors produce an immediate effect because neurotransmitter binding directly opens an ion channel.

(b) Metabotropic Receptor: Indirect Activation

1. Neurotransmitters, identified as the **first messenger**, are released into the synaptic cleft and bind to a **metabotropic receptor** on the postsynaptic membrane.
2. Binding activates a **G protein**, which interacts with an effector protein on the inner surface of the membrane.
3. Using energy from **ATP**, the activated pathway produces **second messenger molecules** within the cell.
4. The second messengers activate intracellular enzymes and signaling pathways.
5. These signaling events cause an ion channel to open, allowing ions to move across the postsynaptic membrane.
6. Because several signaling steps occur before the channel opens, the response develops more slowly but can be amplified and prolonged.

The bottom panel demonstrates that metabotropic receptors influence ion channels indirectly through G proteins and second messengers, producing a longer-lasting response than direct ion channel activation.

The figure illustrates two mechanisms of postsynaptic signaling. Ionotropic receptors generate rapid responses by directly opening ion channels when neurotransmitters bind. Metabotropic receptors generate slower, longer-lasting responses by activating intracellular signaling pathways that ultimately regulate ion channel activity.

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2: CENTRAL AND PERIPHERAL NERVOUS SYSTEM

Chapter Overview

Phineas Gage's (Figure 2.1) case established a foundational truth in neuroscience: structure determines function. Damage a specific brain structure or its connections, and the functions it supports — personality, judgment, emotional control, social behavior — are specifically and predictably impaired. This principle remains central to understanding brain disorders and brain – behavior relationships today.



Figure 2.1: Phineas Gage was a railroad constructor of the 19th century who lost part of his left frontal lobe to an explosion where an iron rod went through his brain. (CC BY-SA 3.0; via Introduction to the Peripheral Nervous System).

The nervous system is divided into two main parts: the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS includes the brain and spinal cord. The PNS includes all the nerves and ganglia outside the brain and spinal cord. There is a very close link between structure (how each part is built) and function (what it does). Where a structure is located, what it looks like, and how it connects to other parts all help explain its job.

The CNS is mainly responsible for processing information, making decisions, and coordinating responses. The PNS is mainly responsible for carrying information to and from the CNS, connecting it to the rest of the body.

Central Nervous System: Brain

The brain is the control center of the body. Its outer surface is the cerebral cortex, a thin, highly folded layer. The cortex is mostly gray matter, made of neuron cell bodies. The folds increase the surface area, allowing more neurons and more complex processing. The cerebral cortex is essential for higher functions such as thinking, planning, language, personality, conscious movement, and conscious sensation.

Different areas, or lobes, of the cortex have different main roles. The frontal lobe is involved in decision-making, planning, voluntary movement, personality, and social behavior. The parietal lobe processes body sensations such as touch, temperature, and pain, and helps you know where your body is in space. The temporal lobe is involved in hearing, understanding language, and forming memories. The occipital lobe is mainly responsible for vision.

At the very front of the frontal lobe is the prefrontal cortex. This region has many connections to emotion centers in the limbic system and to motor areas. It is important for executive functions such as planning, judgment, impulse control, social behavior, and emotional regulation. Damage to the prefrontal cortex often causes changes in personality and behavior, even if basic

movement and sensation remain normal.

Central Nervous System: Spinal Cord

The spinal cord is a long, thin column of nervous tissue inside the vertebral column (spine). In cross-section, it has gray matter in the center and white matter on the outside. The gray matter contains neuron cell bodies and local circuits that form reflex pathways. The white matter contains myelinated axons that run up and down, forming “highways” for information.

The spinal cord has two major functions. First, it acts as an information highway. Sensory information travels from receptors in the body up the spinal cord to the brain. Motor commands travel from the brain down the spinal cord to muscles and glands. Second, the spinal cord acts as a reflex center. Some fast, automatic responses, such as pulling your hand away from something hot, can be handled at the spinal cord level without waiting for the brain. This allows very rapid, protective reactions.

Peripheral Nervous System: Nerves and Ganglia

The peripheral nervous system includes all nervous tissue outside the brain and spinal cord. Its main parts are nerves and ganglia. A nerve is a bundle of axons wrapped in connective tissue. Nerves may be mostly sensory, mostly motor, or mixed (containing both sensory and motor fibers). Cranial nerves connect directly to the brain. Spinal nerves connect to the spinal cord. The function of nerves is to act as communication lines between the CNS and the rest of the body.

Ganglia are clusters of neuron cell bodies in the PNS. They serve as relay and processing stations, especially in the autonomic nervous system.

Sensory (afferent) fibers in the PNS carry information from receptors in the skin, muscles, joints, and internal organs to the CNS. Motor (efferent) fibers carry commands from the CNS to skeletal muscles, smooth muscle, cardiac muscle, and glands.

Functional Divisions of the PNS: Somatic and Autonomic

The PNS is divided into the somatic nervous system and the autonomic nervous system.

The somatic nervous system is mainly under voluntary control. Its motor neurons go to skeletal muscles and allow conscious movements such as walking, writing, or speaking. Its sensory neurons bring information from the skin, muscles, and joints to the CNS, creating conscious sensations such as touch, pain, temperature, and awareness of limb position.

The autonomic nervous system (ANS) controls internal organs and is mostly involuntary. Its motor neurons go to smooth muscle (for example, in the digestive tract and blood vessels), cardiac muscle (the heart), and glands. The ANS helps regulate heart rate, blood pressure, digestion, pupil size, and many other automatic functions.

The autonomic system has two main divisions: the sympathetic division and the parasympathetic division. The sympathetic division is often called “fight or flight.” It prepares the body for action by increasing heart rate, dilating the airways, and redirecting blood flow to skeletal muscles. The parasympathetic division is often called “rest and digest.” It supports rest, digestion, and energy storage by slowing heart rate, increasing digestive activity, and promoting glandular secretion. These two divisions often have opposite effects on the same organs, which allows fine, balanced control.

Protection and Support of the CNS

The CNS is essential but fragile, so the body provides several layers of protection. The first protection is bone. The skull (cranium) surrounds the brain, and the vertebral column surrounds the spinal cord. These bony structures protect against mechanical injury.

Under the bone are three connective tissue layers called the meninges. The outer layer is the dura mater, a tough, thick membrane attached to the inside of the skull and vertebrae. The dura also forms venous channels called dural sinuses. The middle layer is the arachnoid mater, which has a web-like appearance. Beneath it is the subarachnoid space, filled with cerebrospinal fluid (CSF).

The inner layer is the pia mater, a thin, delicate membrane that closely follows the surface of the brain and spinal cord. Together, the meninges support and stabilize the CNS, divide it into compartments, and create spaces where CSF can circulate.

Blood Supply and the Blood–Brain Barrier

The brain needs a constant blood supply for oxygen and glucose. Arterial blood reaches the brain through the internal carotid arteries and the vertebral arteries. These vessels join to form a circular arterial structure called the circle of Willis. This circle provides alternative pathways for blood flow, helping to maintain perfusion if one part of the system is narrowed or blocked.

The blood–brain barrier (BBB) is a special barrier between the blood and the CNS tissue. It is formed mainly by tight junctions between capillary endothelial cells, supported by glial cells called astrocytes. The BBB limits the exchange of substances between the blood and the interstitial fluid of the brain and spinal cord. It protects the CNS from many toxins and pathogens and from rapid chemical changes in the blood, while still allowing important molecules such as oxygen and glucose to enter. Because of the BBB, the CNS has a "privileged" internal environment that is carefully controlled.

Cerebrospinal Fluid (CSF) and Its Circulation

Cerebrospinal fluid is a clear, watery fluid that surrounds and fills spaces in the brain and spinal cord. It is produced mainly by choroid plexuses, which are networks of capillaries and specialized cells inside the four ventricles of the brain. These structures filter blood to make CSF.

CSF flows from the ventricles through connecting openings into the subarachnoid space between the arachnoid and pia mater. It then circulates around the brain and spinal cord. CSF acts as a shock absorber, cushioning delicate nervous tissue. It also helps maintain a stable chemical environment and carries away metabolic wastes from CNS cells.

Waste removal occurs as CSF is reabsorbed into the venous blood through structures called arachnoid granulations, which project into the dural sinuses. The dural sinuses are venous channels located within the dura mater. From the dural sinuses, blood (now mixed with reabsorbed CSF and dissolved wastes) drains out of the skull through the jugular veins and returns to the heart.

Phineas Gage: Linking Brain Structure and Behavior

The famous case of Phineas Gage shows clearly how specific brain structures are linked to behavior and personality. In 1848, Gage, a 25-year-old railroad construction foreman, suffered a severe accident. An explosion drove a heavy iron rod up through his face and skull, entering below his left cheekbone and exiting through the top of his head. He survived and remained conscious, but the damage to his brain was major.

Modern imaging and modeling suggest that only about 4 percent of his cerebral cortex was directly destroyed, but about 11 percent of his white matter, the brain's "wiring" that connects different regions, was damaged. Important connections between the left frontal lobe, temporal lobe, both frontal lobes, and limbic (emotional) structures were cut. This widespread disconnection of neural pathways was as important as the direct injury to the cortex.

Before the accident, Gage was described as responsible, balanced, and a natural leader. After the accident, his personality changed dramatically. He became impulsive, rude, unreliable, and socially inappropriate. Friends said he was "no longer Gage." These changes match the functions of the prefrontal cortex and its connections with emotion and decision-making centers. Damage to these areas and pathways impaired his judgment, impulse control, and emotional regulation.

Before Gage's case, the frontal lobes were often thought to be "silent" areas with no major function. His case was one of the first strong pieces of evidence that particular brain regions and their connections are necessary for normal personality and higher mental functions. It helped establish the idea that brain structure determines behavioral function and inspired later research into localization of function and the role of the prefrontal cortex.

Today, similar behavioral changes are seen in patients with traumatic brain injuries and in neurodegenerative diseases such as Alzheimer's disease and frontotemporal dementia. These conditions also damage frontal brain areas and their white matter connections. This confirms that both specific regions and the networks connecting them are essential for normal behavior, supporting what was first learned from Phineas Gage's injury.

Summary: How CNS and PNS Work Together

The CNS, with its folded cortex and organized spinal tracts, is specialized for integration, decision-making, and reflex control. It is well protected by bone, meninges, cerebrospinal fluid, and the blood–brain barrier. The PNS, with its long, branching nerves and specialized sensory endings, is specialized for sensing the external and internal environment and carrying out the body's actions.

Sensory information from the PNS enters the CNS, where it is processed and integrated. The CNS then sends motor commands back through the PNS to skeletal muscles and internal organs. The somatic division of the PNS allows voluntary movement and conscious sensation. The autonomic division regulates involuntary functions of the heart, smooth muscles, and glands through its sympathetic and parasympathetic branches.

Together, the structure of the CNS and PNS allows the nervous system to sense, think, decide, and act, and to maintain the body's internal balance (homeostasis) while supporting complex behaviors and mental functions.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- Describe how the skull, vertebrae, meninges, cerebrospinal fluid (CSF), and related connective tissues support and protect the brain and spinal cord, and relate this to vulnerability to injury.
- Identify the major external and internal regions and key landmarks of the brain (cerebrum, diencephalon, brainstem, cerebellum, limbic system) and relate their locations to basic functions.
- Identify the major regions and gross landmarks of the spinal cord (cervical, thoracic, lumbar, sacral regions; enlargements; conus medullaris; cauda equina; filum terminale).
- Map the main functional areas of the cerebral cortex (motor, sensory, association) and summarize their roles in movement, sensation, integration, language, and behavior.
- Explain the concept of brain lateralization and how it influences selected sensory and motor functions (such as language, handedness, and spatial processing).
- Describe the formation, circulation, and reabsorption of CSF, including the roles of ventricles and choroid plexuses, and briefly relate CSF disturbances to example clinical conditions.
- Outline the arterial supply of the brain (internal carotid, vertebral, basilar arteries and Circle of Willis branches) and relate interruption of major cerebral arteries to likely neurological deficits.
- Describe the venous drainage of the brain, including the dural venous sinuses, and trace the pathway of venous blood from brain tissue back to the systemic circulation.
- List the cranial nerves in order of anatomical origin and summarize their main central and peripheral connections.
- List the spinal nerves by vertebral region and indicate which major nerve plexus each contributes to or supplies.

2.1: Support and Protection of the Brain

2.2: Brain- Cerebrum

2.3: Brain- Diencephalon, Brainstem, Cerebellum and Limbic System

2.4: Cranial Nerves

2.5: Spinal Cord and Spinal Nerves

2.6: Terms and Definitions

2.7: Chapter Review Questions

2.8: Critical Thinking Self-Assessment

2.9: Alternate Text Descriptions

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2.1: Support and Protection of the Brain

Protective Coverings of the Brain

The central nervous system (CNS) is crucial to the operation of the body and any compromise of function in the brain and spinal cord can lead to severe difficulties. The brain is protected by multiple structures. First, the bones of the skull enclose and house the brain. Underneath the skeletal structures, the brain is protected by membranes made by connective tissue, called meninges, that surround, support, stabilize and partition the nervous tissue (Figure 2.1.1). In addition, the brain has a privileged blood supply, as suggested by the blood-brain barrier. The function of the tissue is crucial to the survival of the organism, so the contents of the blood cannot simply pass into the central nervous tissue. To protect this region from the toxins and pathogens that may be traveling through the blood stream, there is strict control over what can move out of the general systems and into the brain. Because of this privilege, the brain needs specialized structures for the maintenance of circulation. This begins with a unique arrangement of blood vessels carrying fresh blood into the brain and venous sinuses carrying deoxygenated blood out of the brain. Beyond the supply of blood, the brain filters the blood into cerebrospinal fluid (CSF), which is then circulated through the cavities of the brain, such as the subarachnoid space and the ventricles.

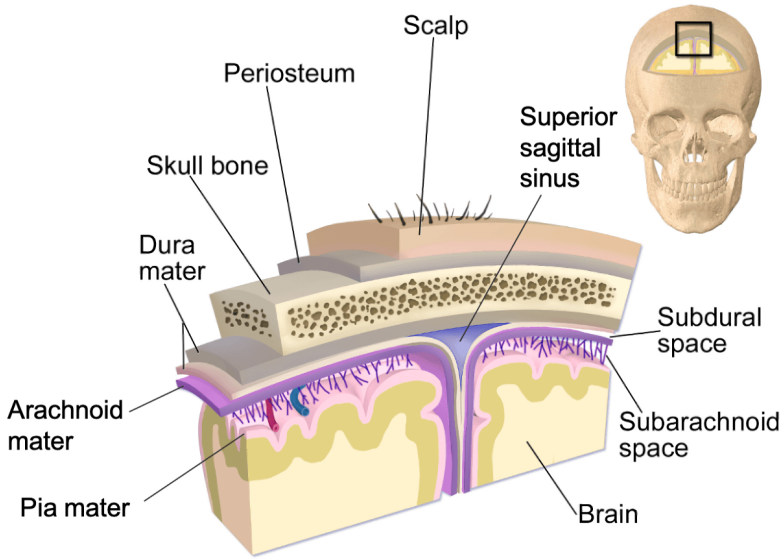


Figure 2.1.1: Superficially, the skin of the scalp and bones of the skull create the first layer of protection. Deep to these layers, the meninges (represented by the dura mater, arachnoid mater and pia mater) cover and partition the brain. Within the meninges, blood inside blood vessels and sinuses, and cerebrospinal fluid within the subarachnoid space and ventricles circulate to support the brain's function. (CC BY 4.0; via original work). Alternate text description of the Figure 2.1.1.

Cranial Meninges

The outer surface of the CNS is covered by a series of membranes composed of connective tissue called the meninges, which protect, stabilize and partition the brain. From superficial to deep, the meningeal layers are the dura mater, arachnoid mater and pia mater. The dura mater is a thick fibrous layer and a strong protective sheath over the entire brain. It is anchored to the inner surface of the cranium and vertebral cavity. The arachnoid mater is a membrane of thin fibrous tissue that forms a loose sac around the brain. Beneath the arachnoid mater, there is a space called the subarachnoid space where a thin, filamentous mesh form the arachnoid trabeculae, that looking like a spider web give this layer its name. Directly adjacent to the surface of the brain is the pia mater, a thin fibrous membrane that follows the superficial convolutions of the brain and fits into other grooves and indentations (Figure 2.1.2).

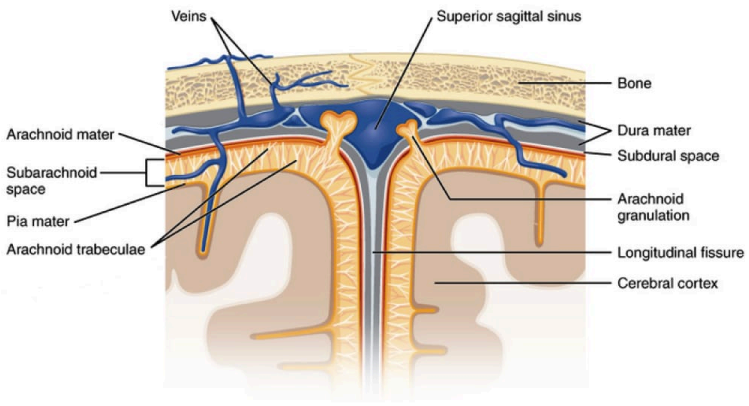


Figure 2.1.2: The layers of the meninges are shown underneath the bones of the skull. The dura mater is adjacent to the inner surface of the cranium. Within it, veins and the superior sagittal sinus lie. The pia mater is adjacent to the surface of the brain. The arachnoid and subarachnoid space are in-between. An arachnoid villus is shown emerging into the dural sinus to allow CSF to filter back into the blood for drainage. (CC BY 4.0; via Support and Protection of the Brain). Alternate text description of the Figure 2.1.2.

Dura Mater

Like a thick cap covering the brain, the dura mater is a tough outer covering. The name comes from the Latin for “tough mother” to represent its physically protective role. It encloses the entire CNS and the major blood vessels that enter the cranium and vertebral cavity. It is directly attached to the inner surface of the bones of the cranium and to the very end of the vertebral cavity. The dura mater of the brain is made by two layers, the periosteal layer which is more superficial and attached to the skull, and the meningeal layer, which lies deep to the first layer. These two layers are usually fused together. However, in some regions of the brain they separate to form large space filled with venous blood called dural sinuses. The dura mater of the spinal cord is composed of only one layer. The region between the bones and the dura mater is called epidural space. In the cranium, the epidural space contains arteries and veins that supply and drain blood from the brain. In normal conditions, the cranial epidural space is a potential space and not a real space. However, trauma can cause the leakage of fluid (hematomas) which accumulates in the epidural space enlarging it and transforming it into a real space. In the vertebral column, the epidural space is a real space that contains areolar and adipose connective tissue for an extra layer of protection, as well as blood vessels. The spinal epidural space at the lumbar level is where epidural injections are administered. Deep to the dura mater, there is another potential space called the subdural space. As the epidural space, this space can be filled with fluid causing a subdural hematoma.

The meningeal layer of the dura mater extends into the cranial cavity at four locations. These flat partitions are called cranial dural septa and separate specific parts of the brain (Figure 2.1.3). The falx cerebri goes through the midline separation of the brain while the falx cerebelli separates the two cerebellar hemispheres. The tentorium cerebelli separates the cerebellum from the superior part of the brain (cerebrum), forming a shelf-like tent. Another partition called diaphragma sellae surrounds the pituitary gland.

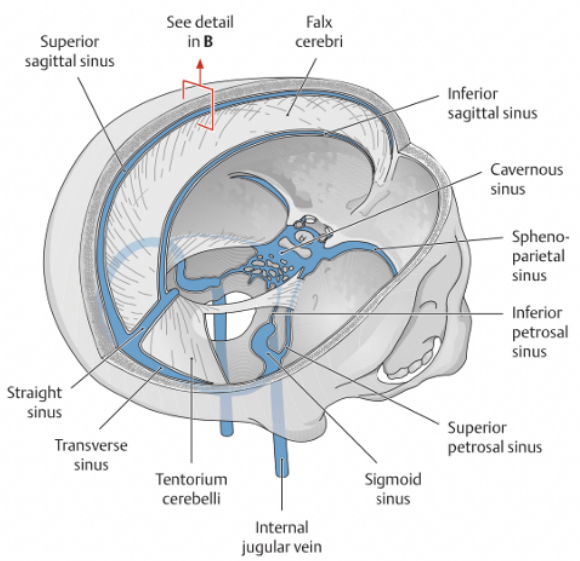


Figure 2.1.3: The dura mater creates fours partitions called cranial dural septa: the falx cerebri, the falx cerebelli, the tentorium cerebelli and the diaphragma sellae (not shown in the figure). The two layers of tissue of the dura mater separate in various regions to create dural sinuses which are filled with venous blood. The main dural sinuses are: the superior sagittal sinus, inferior sagittal sinus, straight sinus, transverse sinus and sigmoid sinus. (CC BY 4.0; via Support and Protection of the Brain). Alternate text description of the Figure 2.1.3.

Arachnoid Mater

Deep to the subdural space lies the middle layer of the meninges called the arachnoid mater. This layer is named for the spider-web–like projections called arachnoid trabeculae between this layer and the pia mater. The arachnoid defines a sac-like enclosure around the CNS. The trabeculae are found in the subarachnoid space, which is filled with circulating CSF, making it a real space. The arachnoid emerges into the dural sinuses as the arachnoid granulations, where the CSF is filtered back into the blood for drainage from the nervous system.

Pia Mater

The outer surface of the CNS is covered in the thin fibrous membrane of the pia mater. It is thought to have a continuous layer of cells providing a fluid-impermeable membrane. The name pia mater comes from the Latin for “tender mother,” suggesting the thin membrane is a gentle covering for the brain. The pia extends into every convolution of the CNS. Blood vessels that are nourishing the central nervous tissue are between the pia mater and the nervous tissue.

Disorders of the Meninges: Meningitis

Meningitis is an inflammation of the meninges, the three layers of fibrous membrane that surround the CNS. Meningitis can be caused by infection by bacteria or viruses. The particular pathogens are not special to meningitis; it is just an inflammation of that specific set of tissues from what might be a broader infection. Bacterial meningitis can be caused by Streptococcus, Staphylococcus, or the tuberculosis pathogen, among many others. Viral meningitis is usually the result of common enteroviruses (such as those that cause intestinal disorders), but may be the result of the herpes virus or West Nile virus. Bacterial meningitis tends to be more severe.

The symptoms associated with meningitis can be fever, chills, nausea, vomiting, light sensitivity, soreness of the neck, or severe headache. More important are the neurological symptoms, such as changes in mental state (confusion, memory deficits, and other dementia-type symptoms). A serious risk of meningitis can be damage to peripheral structures because of the nerves that pass through the meninges. Hearing loss is a common result of meningitis.

The primary test for meningitis is a lumbar puncture. A needle inserted into the lumbar region of the spinal column through the dura mater and arachnoid membrane into the subarachnoid space can be used to withdraw the fluid for chemical testing. Fatality occurs in 5 to 40 percent of children and 20 to 50 percent of adults with bacterial meningitis. Treatment of bacterial meningitis is through antibiotics, but viral meningitis cannot be treated with antibiotics because viruses do not respond to that type of drug. Fortunately, the viral forms are milder.

Blood Supply to the Brain

A lack of oxygen to the CNS can be devastating, and the cardiovascular system has specific regulatory reflexes to ensure that the blood supply is not interrupted. There are multiple routes for blood to get into the CNS, with specializations to protect that blood supply and to maximize the ability of the nervous tissue to get an uninterrupted perfusion.

Arterial Supply to the Brain

The major artery carrying recently oxygenated blood away from the heart is the aorta. The very first branches off the aorta supply the heart with nutrients and oxygen. The next branches give rise to the **common carotid arteries**, which further branch into the **internal carotid arteries**. The external carotid arteries supply blood to the tissues on the surface of the cranium. The internal carotid artery enters the cranium through the carotid canal in the temporal bone.

A second set of vessels that supply the CNS are the **vertebral arteries**, which are protected as they pass through the neck region by the transverse foramina of the cervical vertebrae. The vertebral arteries enter the cranium through the foramen magnum of the occipital bone. The two vertebral arteries then merge into the **basilar artery**, which gives rise to branches to the brainstem and cerebellum. The left and right internal carotid arteries and branches of the basilar artery all become the **circle of Willis**, a confluence of arteries that can maintain perfusion of the brain even if narrowing or a blockage limits flow through one part (Figure 2.1.4).

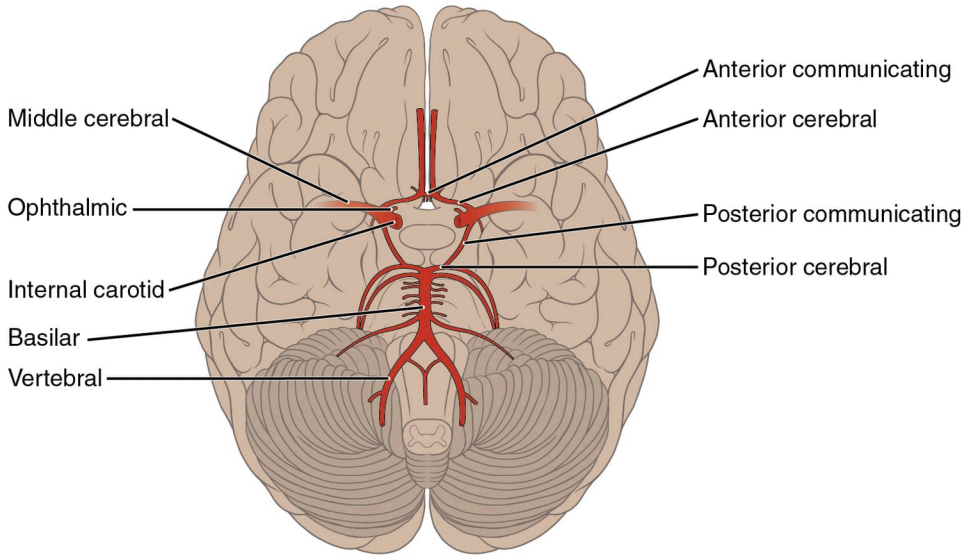


Figure 2.1.4: This inferior view shows the network of arteries serving the brain. The structure is referred to as the circle of Willis and is formed by branches of the internal carotid artery, vertebral artery and basilar artery. (CC BY 3.0; via Arteries of the Brain). Alternate text description of the Figure 2.1.4.

Venous Return from the Brain

After passing through the CNS, blood returns to the circulation through a series of dural sinuses and cerebral veins (Figure 2.1.5). The dural sinuses are housed within the two layers of the dura mater. The superior sagittal sinus runs in the medial groove of the brain, where it absorbs CSF from the meninges. The superior sagittal sinus drains to the confluence of sinuses, along with the occipital sinuses and straight sinus, to then drain into the transverse sinuses. The transverse sinuses connect to the sigmoid sinuses, which then connect to the jugular veins. From there, the blood continues toward the heart to be pumped to the lungs for

reoxygenation.

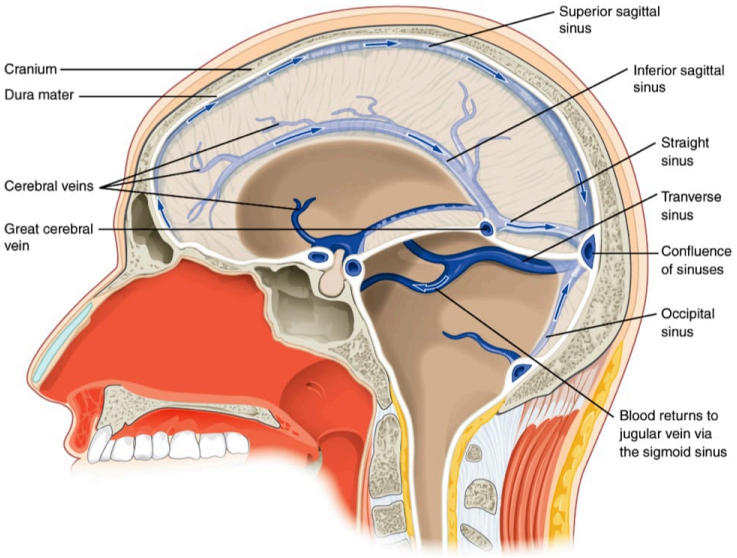


Figure 2.1.5: Blood drains from the brain through a series of sinuses and veins that connect to the jugular veins. (CC BY 3.0; via Arteries of the Brain). Alternate text description of the Figure 2.1.5.

Ventricular System

Cerebrospinal fluid (CSF) is produced by a type of specialized membrane made of ependymal cells called a choroid plexus. Ependymal cells (one of the types of glial cells described in the introduction to the nervous system) surround blood capillaries and filter the blood to make CSF. The fluid is a clear solution with a limited amount of the constituents of blood. It is essentially water, small molecules, and electrolytes and is continuous with the interstitial fluid. Oxygen and carbon dioxide are dissolved into the CSF, as they are in blood, and can diffuse between the fluid and the nervous tissue. CSF circulates through the nervous tissue to remove metabolic wastes from the interstitial fluids of nervous tissues and return them to the blood stream. The choroid plexus lines open spaces within the brain called ventricles. The CSF circulates through all of the ventricles to eventually emerge into the subarachnoid space where it will be reabsorbed into the blood.

Ventricles

There are four ventricles within the brain, all of which developed from the original hollow space within the neural tube, the central canal. The first two are named the lateral ventricles and are deep within the brain (Figure 2.1.6). The two lateral ventricles are shaped as a C and are located in the left and right hemispheres, and were at one time referred to as the first and second ventricles. These ventricles are connected to the third ventricle by two openings called the interventricular foramina. The third ventricle opens into a canal called the cerebral aqueduct that passes through the midbrain and connects the third ventricle to the fourth ventricle. The fourth ventricle is the space between the cerebellum and the pons and upper medulla. The ventricular system opens up to the subarachnoid space from the fourth ventricle. The single median aperture and the pair of lateral apertures connect to the subarachnoid space so that CSF can flow through the ventricles and around the outside of the CNS (Figure 2.1.7). From the fourth ventricle, CSF can continue down the central canal of the spinal cord.

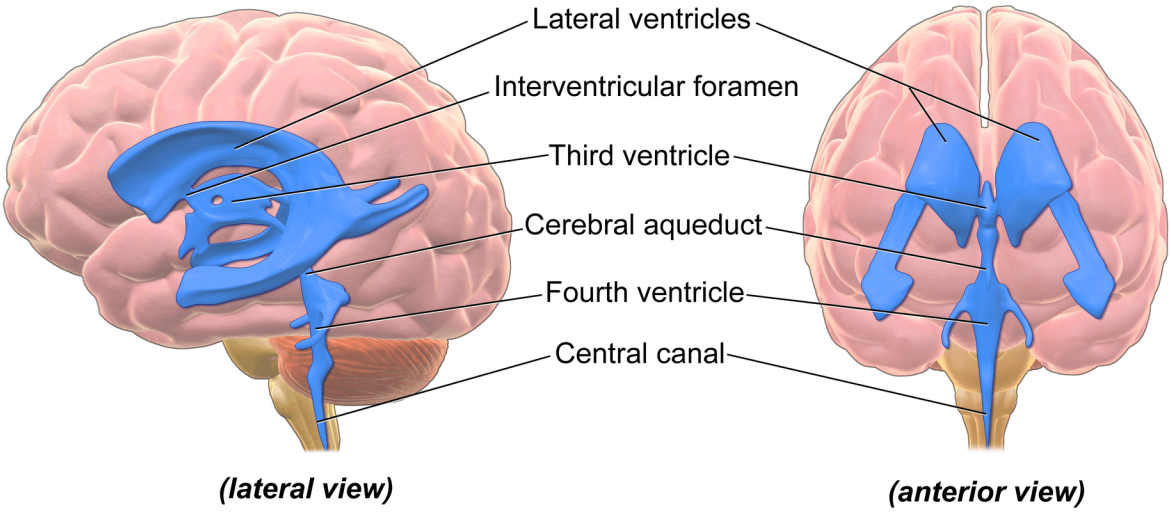


Figure 2.1.6: Within the brain, open spaces called ventricles are lined with ependymal cells that produce cerebrospinal fluid (CSF). Deep within the brain are the lateral ventricles, which are connected to the third ventricle by two openings called interventricular foramina. The third ventricle opens into a canal called the cerebral aqueduct which connects to the fourth ventricle. The fourth ventricle is connected to the central canal of the spinal cord. (CC BY 3.0; via Arteries of the Brain). Alternate text description of the Figure 2.1.6.

Cerebrospinal Fluid Circulation

The choroid plexuses are found in all four ventricles. Observed in dissection, they appear as soft, fuzzy structures that may still be pink, depending on how well the circulatory system is cleared in preparation of the tissue. The CSF is produced from components extracted from the blood, so its flow out of the ventricles is tied to the pulse of cardiovascular circulation.

From the lateral ventricles, the CSF flows into the third ventricle, where more CSF is produced, and then through the cerebral aqueduct into the fourth ventricle where even more CSF is produced (Figure 2.1.7). A very small amount of CSF is filtered at any one of the plexuses, for a total of about 500 milliliters daily, but it is continuously made and pulses through the ventricular system, keeping the fluid moving. From the fourth ventricle, CSF can continue down the central canal of the spinal cord, but this is essentially a cul-de-sac, so more of the fluid leaves the ventricular system and moves into the subarachnoid space through the median and lateral apertures.

Within the subarachnoid space, the CSF flows around all of the CNS, providing two important functions. As with elsewhere in its circulation, the CSF picks up metabolic wastes from the nervous tissue and moves it out of the CNS. It also acts as a liquid cushion for the brain and spinal cord. By surrounding the entire system in the subarachnoid space, it provides a thin buffer around the organs within the strong, protective dura mater. The arachnoid granulations are outpocketings of the arachnoid membrane into the dural sinuses so that CSF can be reabsorbed into the blood, along with the metabolic wastes. From the dural sinuses, blood drains out of the head and neck through the jugular veins, along with the rest of the circulation for blood, to be reoxygenated by the lungs and wastes to be filtered out by the kidneys.

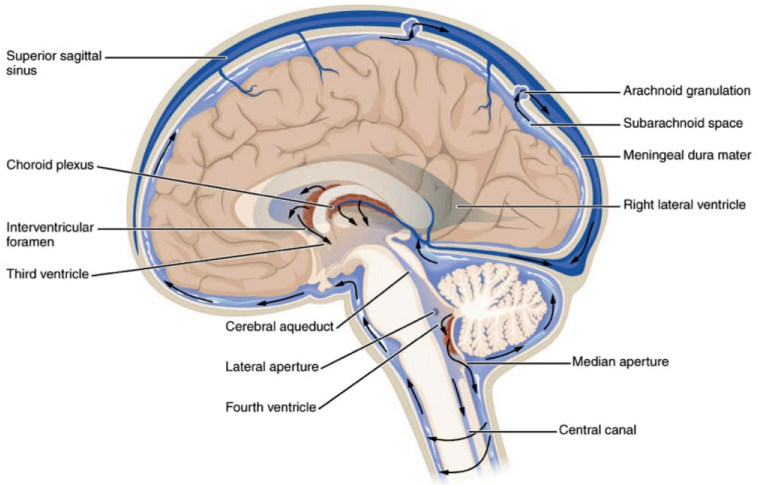


Figure 2.1.7: The choroid plexus in all the ventricles produce CSF, which is circulated through the ventricular system and then enters the subarachnoid space through the median and lateral apertures. The CSF is then reabsorbed into the blood at the arachnoid granulations, where the arachnoid membrane emerges into the dural sinuses. (CC BY 3.0; via Arteries of the Brain). Alternate text description of the Figure 2.1.7.

Disorders of the Central Nervous System: Strokes

The supply of blood to the brain is crucial to its ability to perform many functions. Without a steady supply of oxygen, and to a lesser extent glucose, the nervous tissue in the brain cannot keep up its extensive electrical activity. These nutrients get into the brain through the blood, and if blood flow is interrupted, neurological function is compromised.

The common name for a disruption of blood supply to the brain is a stroke. It is caused by a blockage to an artery in the brain or by blood leaking out of blood vessels (hemorrhagic stroke), although less common. The blockage is caused by some type of embolus: a blood clot, a fat embolus, or an air bubble. When the blood cannot travel through the artery, the surrounding tissue that is deprived starves and dies. Strokes will often result in the loss of very specific functions. A stroke in the lateral medulla, for example, can cause a loss in the ability to swallow. Sometimes, seemingly unrelated functions will be lost because they are dependent on structures in the same region. Along with the swallowing in the previous example, a stroke in that region could affect sensory functions from the face or extremities because important white matter pathways also pass through the lateral medulla. Loss of blood flow to specific regions of the brain can lead to the loss of specific higher functions, from the ability to recognize faces to the ability to move a particular region of the body. Severe or limited memory loss can be the result of a temporal lobe stroke.

Related to strokes are transient ischemic attacks (TIAs), which can also be called “mini-strokes.” These are events in which a physical blockage may be temporary, cutting off the blood supply and oxygen to a region, but not to the extent that it causes cell death in that region. While the neurons in that area are recovering from the event, neurological function may be lost. TIAs usually resolve spontaneously by the body, that is why it is called "transient".

Recovery from a stroke (or TIA) is strongly dependent on the speed of treatment. Often, the person who is present and notices something is wrong must then make a decision. The mnemonic FAST helps people remember what to look for when someone is dealing with sudden losses of neurological function. If someone complains of feeling “funny,” check these things quickly: Look at the person’s face. Does he or she have problems moving Face muscles and making regular facial expressions? Ask the person to raise his or her Arms above the head. Can the person lift one arm but not the other? Has the person’s Speech changed? Is he or she slurring words or having trouble saying things? If any of these things have happened, then it is Time to call for help.

Sometimes, treatment with blood-thinning drugs can alleviate the problem, and recovery is possible. If the tissue is damaged, the amazing thing about the nervous system is that it is adaptable. With physical, occupational, and speech therapy, victims of strokes can recover, or more accurately relearn, functions.

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2.2: Brain- Cerebrum

Cerebrum

The organs of the central nervous system are the brain and spinal cord. The brain is described in terms of four major regions: the cerebrum, the diencephalon, the brainstem, and the cerebellum (Figure 2.2.1). The iconic gray mantle of the human brain, which appears to make up most of the mass of the brain, is the **cerebrum**. Many of the higher neurological functions, such as memory, emotion, and consciousness, are the result of cerebral function. The cerebrum is divided into different regions called lobes. Overall, the functions of the cerebrum are initiation and coordination of movement, processing of general and special senses, and high level functions such as judgment, reasoning, problem solving, and learning

The cerebrum is covered by a continuous layer of gray matter that wraps around either side of the forebrain—the cerebral cortex. This thin, extensive region of wrinkled gray matter is responsible for the higher functions of the nervous system. A gyrus (plural = gyri) is the ridge of one of those wrinkles, and a sulcus (plural = sulci) is the groove between two gyri (Figure 2.2.1). The pattern of these folds of tissue indicates specific regions of the cerebral cortex. The brain must fit inside the cranial cavity of the skull. Extensive folding in the cerebral cortex enables more gray matter to fit into this limited space. If the gray matter of the cortex were peeled off of the cerebrum and laid out flat, its surface area would be roughly equal to one square meter. The folding of the cortex maximizes the amount of gray matter in the cranial cavity. During embryonic development, as the telencephalon expands within the skull, the brain goes through a regular course of growth that results in everyone's brain having a similar pattern of folds.

The surface of the brain can be mapped on the basis of the locations of large gyri and sulci. Three deep sulci are visible: the central sulcus, the lateral sulcus and the parieto-occipital sulcus. Anterior to the central sulcus is the precentral gyrus, while posterior to the central sulcus is the postcentral gyrus (Figure 2.2.1). There is a large separation between the two sides of the cerebrum called the longitudinal fissure (Figure 2.2.2). It separates the cerebrum into two distinct halves, a right and left cerebral hemisphere.

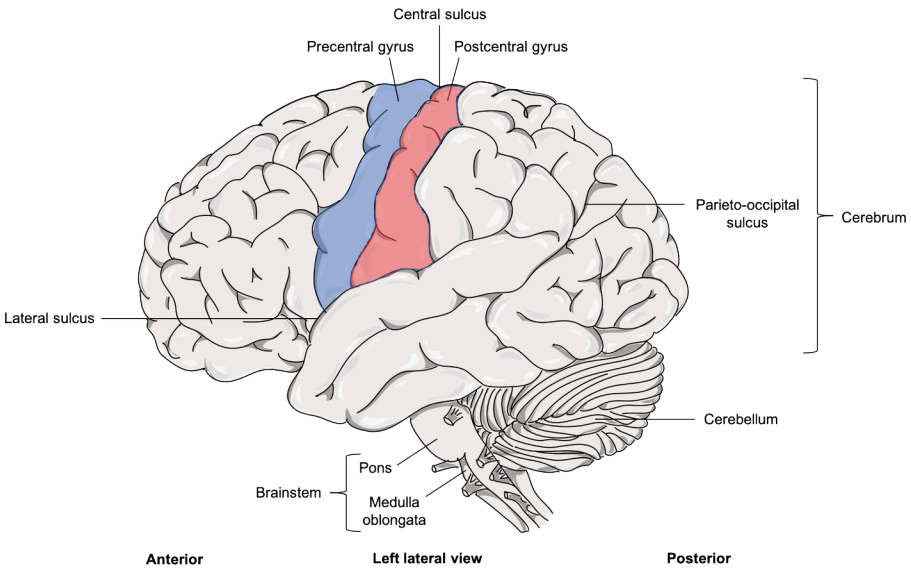


Figure 2.2.1: The regions of the brain visible from a lateral view are the cerebrum, brainstem and cerebellum. The cerebrum presents ridges called gyri and grooves called sulci. The main sulci are the central sulcus, lateral sulcus and parieto-occipital sulcus. The gyrus anterior to the central sulcus is called precentral gyrus, while the posterior one is the postcentral gyrus. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.1.

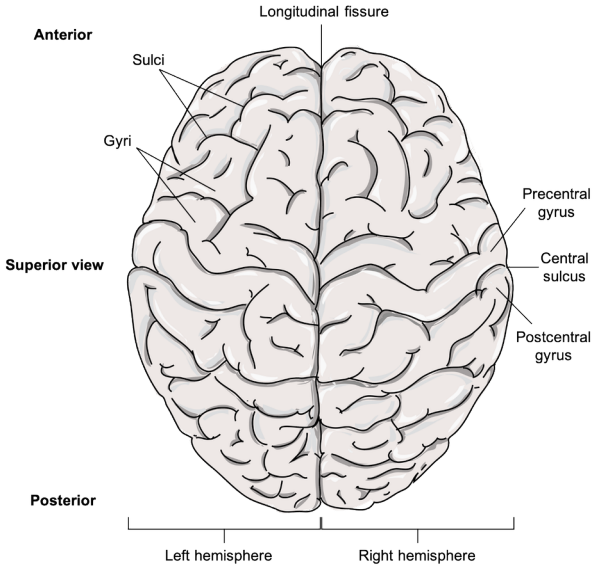


Figure 2.2.2: The cerebrum is medially divided by the longitudinal fissure into left and right hemispheres. The central sulcus is visible cutting the cerebrum in a coronal section. The gyrus anterior to the central sulcus is called precentral gyrus, while the posterior one is the postcentral gyrus. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.2.

The cerebrum comprises the outer wrinkled gray matter that is the cerebral cortex (from the Latin word meaning “bark of a tree”), the inner white matter that forms tracts, and several deep (subcortical) nuclei called basal nuclei (Figure 2.2.3). Deep within the cerebrum, the white matter of the corpus callosum provides the major pathway for communication between the two hemispheres of the cerebral cortex.

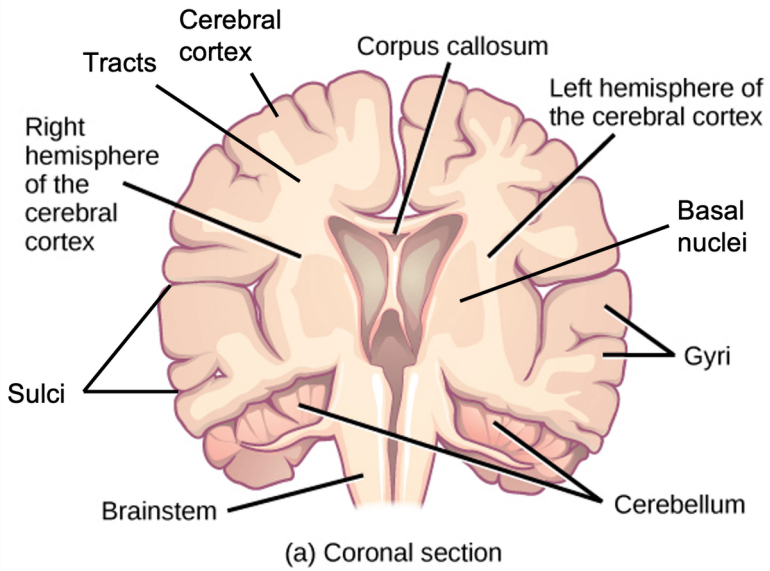


Figure 2.2.3: The coronal section shows the layers of the cerebrum. The superficial gray matter is called cerebral cortex, while the deep gray matter is organized in basal nuclei. The superficial and deep regions are connected by myelinated axons (white matter) called tracts. The corpus callosum is one of these tracts and connect the left and right hemispheres. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.3.

Cerebral Cortex

The cortex can be separated into major regions called lobes (Figure 2.2.4). The lateral sulcus separates the temporal lobe from the other regions. Deep to the lateral sulcus is another lobe called insula (Figure 2.2.5). Superior to the lateral sulcus are the parietal lobe and frontal lobe, which are separated from each other by the central sulcus. The posterior region of the cortex is the occipital lobe, which has no obvious anatomical border between it and the parietal or temporal lobes on the lateral surface of the brain.

From the medial surface, an obvious landmark separating the parietal and occipital lobes is called the parieto-occipital sulcus.

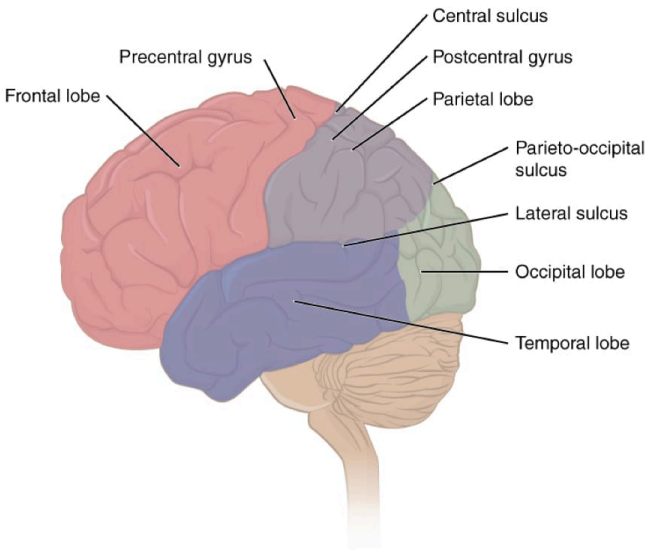


Figure 2.2.4: The cerebral cortex is divided into four lobes by the major sulci. The central sulcus separates the frontal and parietal lobes. The parieto-occipital sulcus separates the parietal and occipital lobes. The lateral sulcus separated the temporal and parietal lobes. The fifth lobe is the insula which is deep to the frontal and temporal lobes. Each lobe corresponds to specific functions of the cerebral cortex. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.4.

Basal Nuclei

Beneath the cerebral cortex are sets of nuclei known as **basal nuclei** that augment cortical processes. The basal nuclei are also referred to as the basal ganglia, although that is potentially confusing because the term ganglia is typically used for peripheral nervous system structures. The basal nuclei include the caudate, putamen, and globus pallidus in the cerebrum, the subthalamic nucleus in the diencephalon, the substantia nigra in the midbrain of the brainstem and the limbic system.

The major structures of the basal nuclei that control movement are the **caudate**, **putamen**, and **globus pallidus**, which are located deep in the cerebrum. The caudate is a long nucleus that follows the basic C-shape of the cerebrum from the frontal lobe, through the parietal and occipital lobes, into the temporal lobe. The putamen is mostly deep in the anterior regions of the frontal and parietal lobes. Together, the caudate and putamen are called the **striatum**. The globus pallidus is a layered nucleus that lies just medial to the putamen; they are called the lenticular nuclei because they look like curved pieces fitting together like lenses. The globus pallidus has two subdivisions, the external and internal segments, which are lateral and medial, respectively. These nuclei are depicted in a frontal section of the brain in Figure 2.2.5. The basal nuclei in the cerebrum are connected with a few more nuclei in the brainstem that together act as a functional group that forms a motor pathway. The basal nuclei are responsible for comparing cortical processing with the general state of activity in the nervous system to influence the likelihood of movement taking place. For example, while a student is sitting in a classroom listening to a lecture, the basal nuclei will keep the urge to jump up and scream from actually happening.

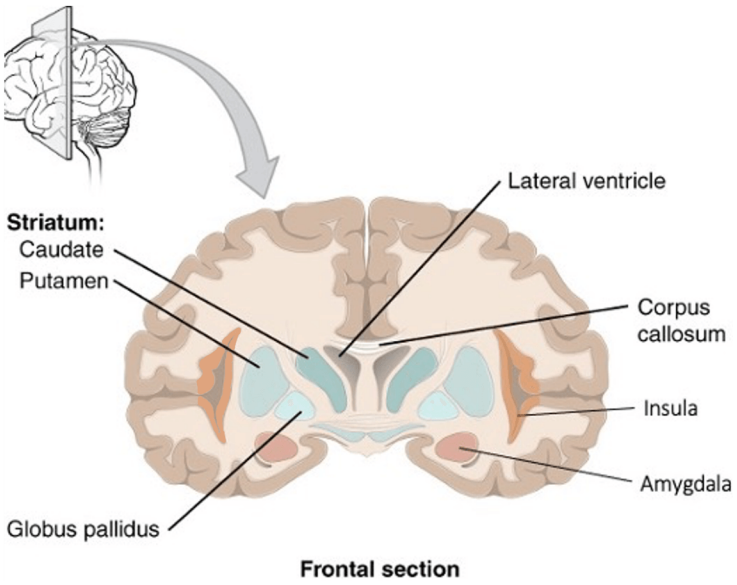


Figure 2.2.5: The major components of the basal nuclei, shown in a frontal section of the brain, are deep within the cerebrum. The caudate is just lateral to the lateral ventricle. The putamen is inferior to the caudate and separated by the large white-matter structure called the internal capsule. The globus pallidus is medial to the putamen. The insula is one of the five lobes of the cerebral cortex and is located deep to the lateral sulcus, between the parietal and temporal lobes. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.5.

White Matter Tracts

The white matter lies deep to the cerebral cortex and is mainly made by myelinated axons, which gives the white appearance. The axons are grouped into bundles called **tracts** that connect different regions of the cerebral cortex to integrate information and motor response. The white matter tracts of the cerebrum can be classified into three groups: **commissural tracts**, **association tracts** and **projection tracts** (Figure 2.2.6).

Commissural tracts extend between the two hemispheres to connect left and right regions of the cerebrum. The most prominent commissural tract, and largest white matter structure, is the **corpus callosum**. Association tracts connect different regions within the same hemisphere. Association tracts can be classified into **arcuate fibers** when they connect gyri of the same lobe or **longitudinal fasciculi** when they connect gyri in different lobes of the same hemisphere. Projection tracts extend from the cerebral cortex to the inferior regions of the brain and to the spinal cord.

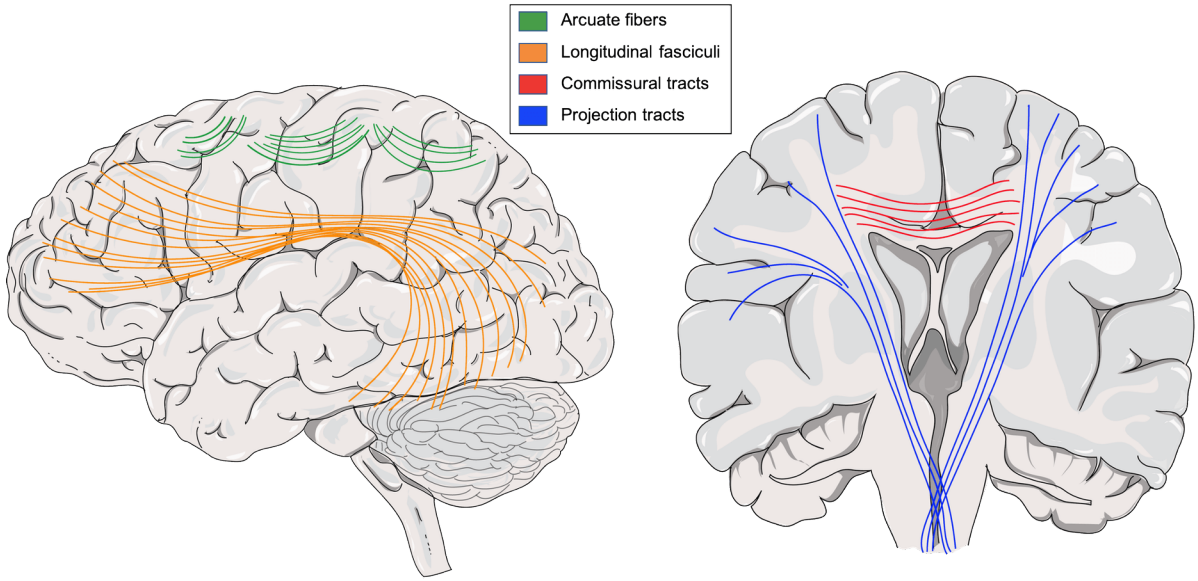


Figure 2.2.6: The white matter of the cerebrum is organized in tracts that connect lobes and gyri within lobes. Association tracts can be divided into arcuate fibers and longitudinal fasciculi. Arcuate fibers connect gyri of the same lobe, while longitudinal fasciculi connect regions of the same hemisphere. Commissural tracts connect the two hemispheres. Projection tracts connect the cerebral cortex to inferior regions of the brain or spinal cord. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.6.

Everyday Connections: Left Brain, Right Brain

Popular media often refer to right-brained and left-brained people, as if the brain were two independent halves that work differently for different people. This is a popular misinterpretation of an important neurological phenomenon. As an extreme measure to deal with a debilitating condition, the corpus callosum may be sectioned to overcome intractable epilepsy. When the connections between the two cerebral hemispheres are cut, interesting effects can be observed.

If a person with an intact corpus callosum is asked to put their hands in their pockets and describe what is there on the basis of what their hands feel, they might say that they have keys in their right pocket and loose change in the left. They may even be able to count the coins in their pocket and say if they can afford to buy a candy bar from the vending machine. If a person with a sectioned corpus callosum is given the same instructions, they will do something quite peculiar. They will only put their right hand in their pocket and say they have keys there. They will not even move their left hand, much less report that there is loose change in the left pocket.

The reason for this is that the language functions of the cerebral cortex are localized to the left hemisphere in 95 percent of the population. Additionally, the left hemisphere is connected to the right side of the body through the corticospinal tract and the ascending tracts of the spinal cord. Motor commands from the precentral gyrus control the opposite side of the body, whereas sensory information processed by the postcentral gyrus is received from the opposite side of the body. For a verbal command to initiate movement of the right arm and hand, the left side of the brain needs to be connected by the corpus callosum. Language is processed in the left side of the brain and directly influences the left brain and right arm motor functions, but is sent to influence the right brain and left arm motor functions through the corpus callosum. Likewise, the left-handed sensory perception of what is in the left pocket travels across the corpus callosum from the right brain, so no verbal report on those contents would be possible if the hand happened to be in the pocket.

Functional Areas of the Cerebrum

The cerebrum is responsible for the perception of sensation and for controlling the skeletal muscles. The cerebrum is also the seat of many of the higher mental functions, such as memory and learning, and language. These higher functions are distributed across various regions of the cortex, and specific locations can be said to be responsible for particular functions.

The frontal lobe is responsible for motor functions, from planning movements through executing commands to be sent to the spinal cord and periphery. The most anterior portion of the frontal lobe is the prefrontal cortex, which is associated with aspects

of personality through its influence on motor responses in decision-making. The other lobes are responsible for sensory functions. The main sensation associated with the parietal lobe is **somatosensation**, meaning the general sensations associated with the body. The occipital lobe is where visual processing begins, although the other parts of the brain can contribute to visual function. The temporal lobe contains the cortical area for auditory processing, but also has regions crucial for memory formation. The insula is responsible for processing of taste sensation.

In the early 1900s, a German neuroscientist named Korbinian Brodmann performed an extensive study of the microscopic anatomy—the cytoarchitecture—of the cerebral cortex and divided the cortex into 52 separate regions on the basis of the histology of the cortex. His work resulted in a system of classification known as **Brodmann's areas**, which is still used today to describe the anatomical distinctions within the cortex (Figure 2.2.7). Continued investigation into these anatomical areas over the subsequent 100 or more years has demonstrated a strong correlation between the structures and the functions attributed to those structures. Today, we more frequently refer to these regions by their function (i.e., primary visual cortex) than by the number Brodmann assigned to them, but in some situations the use of Brodmann numbers persists. Areas 1, 2, 3, 4, 17, and 22 are each described as **primary cortical areas**. Primary areas are where sensory information is initially received for conscious perception, or where descending commands are sent down to the brainstem or spinal cord to execute movements. The regions near the primary areas are each referred to as **association areas**.

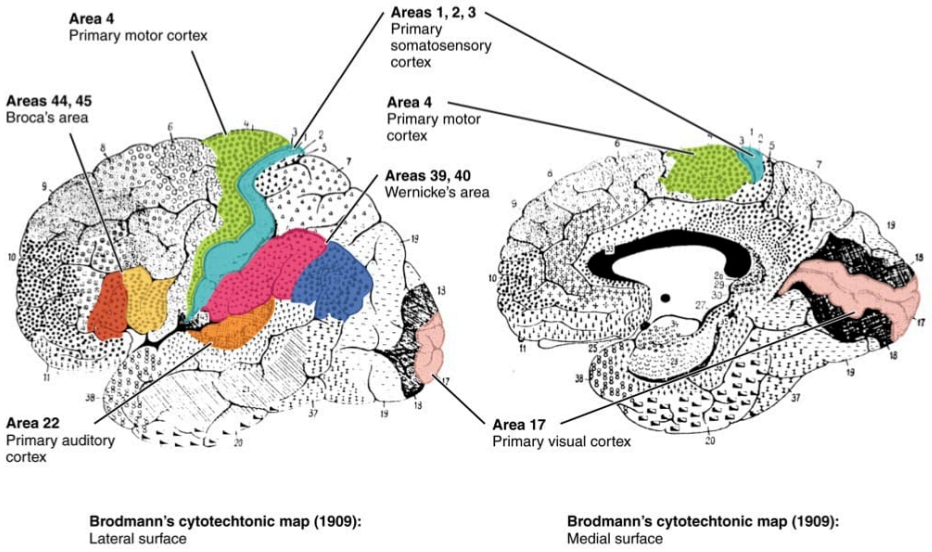


Figure 2.2.7: On the basis of cytoarchitecture, the anatomist Korbinian Brodmann described the extensive array of cortical regions called areas and assigned to them a number, as illustrated in this figure. Subsequent investigations found that these areas corresponded very well to functional differences in the cerebral cortex and associated Brodmann's areas with the various cortices. (CC BY 4.0; via Brain-Cerebrum).
Alternate text description of the Figure 2.2.7.

Conscious Sensation and Voluntary Motor Control

The cortical areas in the parietal, temporal, and occipital lobes are involved in conscious sensation of stimuli. In the postcentral gyrus of the parietal lobe, we find the first three areas in Brodmann's list which compose the **primary somatosensory cortex** (Figure 2.2.8). All of the tactile senses are processed in this area, including touch, pressure, tickle, pain, itch, and vibration, as well as more general senses of the body such as proprioception and kinesthesia, which are the senses of body position and movement, respectively. Next to it in the parietal lobe there is the **sensory association area** which helps the primary area to process somatosensation. In the most posterior part of the occipital lobe (specifically area 17), the **primary visual cortex** receives and processes visual information. Adjacent to that are **visual association areas**, which constitute subsequent regions of visual processing. Area 22 in the temporal lobe is the **primary auditory cortex**, and it is followed by the **auditory association area** (area 23), which further processes auditory information. In the same temporal lobe, the **primary olfactory cortex** is located and processes smell. Finally, the **primary gustatory cortex** is located in the insula and is responsible for processing taste information. The regions that control motor functions are located within the frontal lobes. In the precentral gyrus (area 4) is the **primary motor cortex**. Cells from this region of the cerebral cortex are the upper motor neurons that instruct cells in the spinal cord to move skeletal muscles. Area 6 anterior to it is the motor association area or **premotor cortex**, which aids the primary area in its function. Anterior to the premotor cortex, the **frontal eye field** controls eye movements that require both eyes, such as reading and binocular vision.

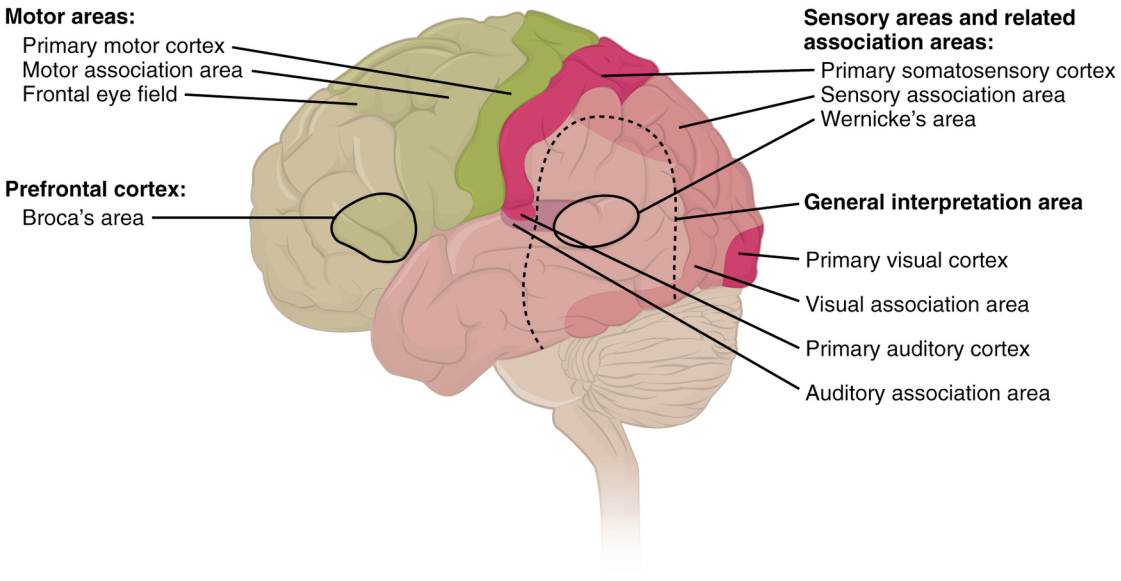


Figure 2.2.8: The cerebral cortex contains primary, association, and integrative regions. Motor areas are located in the frontal lobe, sensory areas in the parietal lobe, visual areas in the occipital lobe, and auditory areas in the temporal lobe. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.8.

The somatosensory and motor cortex provides an example of the mapping of the human body in different regions of the cortex. The term homunculus comes from the Latin word for "little man" and refers to a map of the human body that is laid across a portion of the cerebral cortex. The sensory receptors are mapped onto the somatosensory cortex and this mapping is called a **sensory homunculus** (Figure 2.2.9). In the somatosensory cortex, the external genitals, feet, and lower legs are represented on the medial face of the gyrus within the longitudinal fissure. As the gyrus curves out of the fissure and along the surface of the parietal lobe, the body map continues through the thighs, hips, trunk, shoulders, arms, and hands. The head and face are just lateral to the fingers as the gyrus approaches the lateral sulcus. The representation of the body in this topographical map is medial to lateral from the lower to upper body. Note that this correspondence does not result in a perfectly miniature scale version of the body, but rather exaggerates the more sensitive areas of the body, such as the fingers and lower face. Less sensitive areas of the body, such as the shoulders and back, are mapped to smaller areas on the cortex. Likewise, the motor cortex contains a similar representation of neurons that control the respective body regions. This mapping is called **motor homunculus** (not shown here).

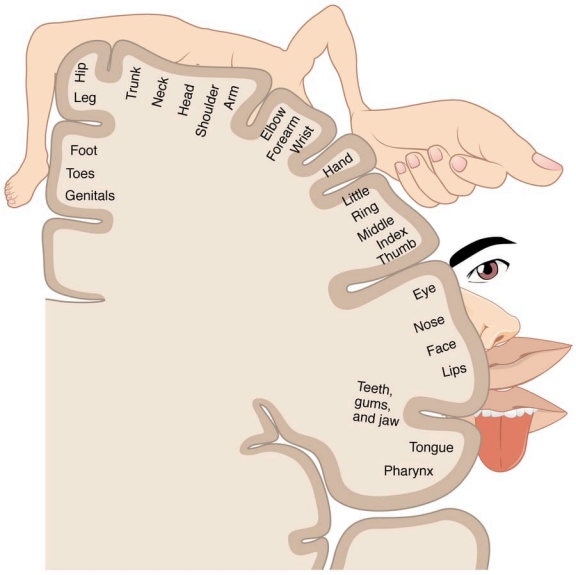


Figure 2.2.9: A cartoon representation of the sensory homunculus is shown. The homunculus refers to a map of the human body that is laid across a portion of the cerebral cortex. In this case, the sensory receptors from various areas of the body are mapped adjacent to the cortical region in which the processing takes place. The most medial region of the somatosensory cortex processes sensory information regarding the toes and feet, while the most lateral region processes sensory information regarding the face, and in particular the mouth. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.9.

A number of other regions, which extend beyond these primary or association areas of the cortex, are referred to as integrative areas. These areas are found in the spaces between the domains for particular sensory or motor functions, and they integrate multisensory information, or process sensory or motor information in more complex ways. Consider, for example, the posterior parietal cortex that lies between the somatosensory cortex and visual cortex regions. This area is called **gnostic area** and has been ascribed to the coordination of visual and motor functions, such as reaching to pick up a glass. The somatosensory function that would be part of this is the proprioceptive feedback from moving the arm and hand. The weight of the glass, based on what it contains, will influence how those movements are executed.

Memory and Learning

There are other cognitive abilities of the cerebral cortex. For example, memory is largely a function of the frontal and temporal lobes, along with structures beneath the cerebral cortex. Short-term memory (also called working or active memory) is localized to the prefrontal lobe. Memory is essentially a sensory function: memories are recalled sensations such as the smell of Mom's baking or the sound of a barking dog. Even memories of movement are really the memory of sensory feedback from those movements, such as stretching muscles or the movement of the skin around a joint. Structures in the temporal lobe are responsible for establishing long-term memory, but the ultimate location of those memories is usually in the region in which the sensory perception was processed.

Language and Speech

Adjacent to the auditory association cortex, at the end of the lateral sulcus just anterior to the visual cortex, is **Wernicke's area** (Figure 2.2.10). Wernicke's area is responsible for the understanding of language, both written and verbal. In the lateral aspect of the frontal lobe, just anterior to the region of the motor cortex associated with the head and neck, is **Broca's area**. Broca's area is responsible for the production of language, or controlling movements responsible for speech. Both area are located only in the left hemisphere in the majority of people. The tendency for some brain functions to be specialized in one side of the brain is called **lateralization of function**. The language areas are not the only example of lateralization. Another example of lateralization is the somatic sensation of one side of the body crossing at the brainstem to send the information to the hemisphere on the opposite side. Exact math calculation and memory retrieval, for example, tend to be on the left parietal lobe. Both regions were originally described on the basis of losses of speech and language, which is called **aphasia**. The aphasia associated with Broca's area is known as an **expressive aphasia**, which means that speech production is compromised. This type of aphasia is often described as non-fluency because the ability to say some words leads to broken or halting speech. Grammar can also appear to be lost. The aphasia associated with Wernicke's area is known as a **receptive aphasia**, which is not a loss of speech production, but a loss of understanding of content. Patients, after recovering from acute forms of this aphasia, report not being able to understand what is

said to them or what they are saying themselves, but they often cannot keep from talking.

The two regions are connected by white matter tracts that run between the posterior temporal lobe and the lateral aspect of the frontal lobe. **Conduction aphasia** associated with damage to this connection refers to the problem of connecting the understanding of language to the production of speech. This is a very rare condition, but is likely to present as an inability to faithfully repeat spoken language.

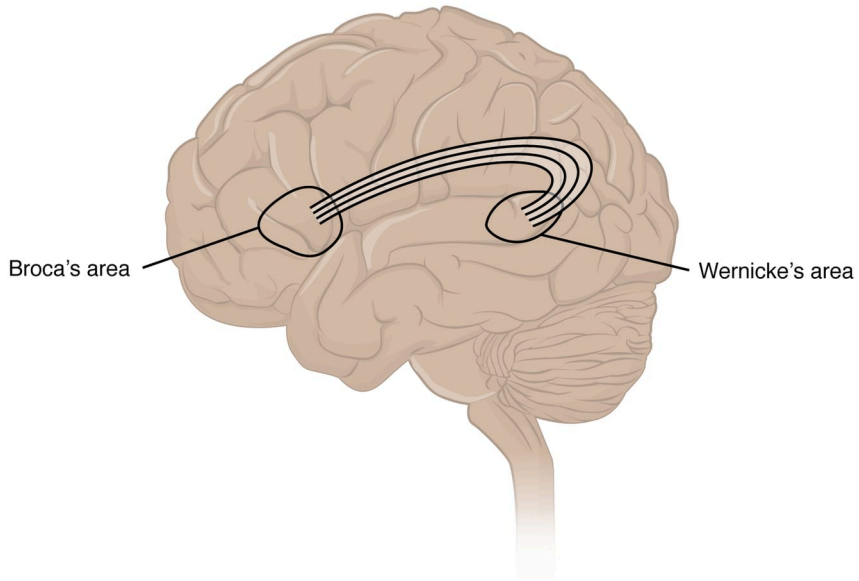


Figure 2.2.10: Two important integration areas of the cerebral cortex associated with language function are Broca's and Wernicke's areas. The two areas are connected through the deep white matter running from the posterior temporal lobe where Wernicke's are is located to the frontal lobe where Broca's area is located. (CC BY 4.0; via Brain-Cerebrum). Alternate text description of the Figure 2.2.10.

Judgment and Abstract Reasoning

Planning and producing responses requires an ability to make sense of the world around us. Making judgments and reasoning in the abstract are necessary to produce movements as part of larger responses. For example, when your alarm goes off, do you hit the snooze button or jump out of bed? Is 10 extra minutes in bed worth the extra rush to get ready for your day? Will hitting the snooze button multiple times lead to feeling more rested or result in a panic as you run late? How you mentally process these questions can affect your whole day.

The prefrontal cortex is responsible for the functions responsible for planning and making decisions. The prefrontal cortex is composed of the regions of the frontal lobe that are not directly related to specific motor functions. The most posterior region of the frontal lobe, the precentral gyrus, is the primary motor cortex. Anterior to that are the premotor cortex, Broca's area, and the frontal eye fields, which are all related to planning certain types of movements. Anterior to what could be described as motor association areas are the regions of the prefrontal cortex. They are the regions in which judgment, abstract reasoning, and working memory are localized. The antecedents to planning certain movements are judging whether those movements should be made, as in the example of deciding whether to hit the snooze button.

A psychiatric practice to deal with various disorders was the **prefrontal lobotomy**. This procedure was common in the 1940s and early 1950s, until antipsychotic drugs became available. The connections between the prefrontal cortex and other regions of the brain were severed. The disorders associated with this procedure included some aspects of what are now referred to as personality disorders, but also included mood disorders and psychoses. Depictions of lobotomies in popular media suggest a link between cutting the white matter of the prefrontal cortex and changes in a patient's mood and personality, though this correlation is not well understood.

Concept Review

The adult brain is separated into four major regions: the cerebrum, the diencephalon, the brainstem, and the cerebellum. The cerebrum is the largest portion and contains the cerebral cortex, subcortical nuclei and white matter tracts. It is divided into two

halves called hemispheres by the longitudinal fissure.

The cerebral cortex is the location of important cognitive functions. It is separated into the frontal, parietal, temporal, and occipital lobes. The frontal lobe is responsible for motor functions, from planning movements through executing commands to be sent to the spinal cord and periphery. The most anterior portion of the frontal lobe is the prefrontal cortex, which is associated with aspects of personality through its influence on motor responses in decision-making. The other lobes are responsible for sensory functions. The parietal lobe is where somatosensation is processed. The occipital lobe is where visual processing begins, although the other parts of the brain can contribute to visual function. The temporal lobe contains the cortical area for auditory processing, but also has regions crucial for memory formation.

Subcortical nuclei beneath the cerebral cortex, known as the subcortical or basal nuclei nuclei, are responsible for augmenting cortical functions. The subcortical or basal nuclei receive input from cortical areas and compare it with the general state of the individual. The output influences the activity of part of the thalamus that can then increase or decrease cortical activity that often results in changes to motor commands.

White matter tracts are bundles of myelinated axons that connect different regions of the brain. Commissural tracts such as the corpus callosum extend between the two hemispheres to connect left and right regions of the cerebrum. Association tracts connect different regions within the same hemisphere. Association tracts can be classified into arcuate fibers when they connect gyri of the same lobe or longitudinal fasciculi when they connect gyri in different lobes of the same hemisphere. Projection tracts extend from the cerebral cortex to the inferior regions of the brain and to the spinal cord.

Losses of language and speech functions, known as aphasias, are associated with damage to the important integration areas in the left hemisphere known as Broca's or Wernicke's areas, as well as the connections in the white matter between them. Different types of aphasia are named for the particular structures that are damaged.

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2.3: Brain- Diencephalon, Brainstem, Cerebellum and Limbic System

Diencephalon

The diencephalon is the one region of the adult brain that retains its name from embryologic dements of the axial musculature, whereas the lateral regions target movements of the appendicular musculature.evelopment. The etymology of the word diencephalon translates to “through brain.” It is the connection between the cerebrum and the rest of the nervous system, with one exception. The rest of the brain, the spinal cord, and the PNS all send information to the cerebrum through the diencephalon. Output from the cerebrum passes through the diencephalon. The single exception is the system associated with olfaction, or the sense of smell, which connects directly with the cerebrum. In the earliest vertebrate species, the cerebrum was not much more than olfactory bulbs that received peripheral information about the chemical environment (to call it smell in these organisms is imprecise because they lived in the ocean).

The diencephalon is deep beneath the cerebrum and constitutes the walls of the third ventricle. The diencephalon can be described as any region of the brain with “thalamus” in its name. The major regions of the diencephalon are the thalamus itself, the hypothalamus, the epithalamus, which contains the pineal gland, and the subthalamus, which includes part of the basal nuclei (Figure 2.3.1).

Epithalamus

The epithalamus forms the posterior roof of the third ventricle and houses the pineal gland, an endocrine gland responsible for the secretion of melatonin. Melatonin regulates day-night cycles. Nuclei within the epithalamus help relay signals to the limbic system and are involved in emotional responses to odors.

Thalamus

The thalamus is a pair of oval-shaped structures that each contain a dozen nuclei, called thalamic nuclei. The thalamus covers the superior and lateral walls of the third ventricle. The right and left thalami are connected by a mass of gray matter called the interthalamic adhesion. All sensory information, except for the sense of smell, passes through the thalamus before processing by the cortex. Axons from the peripheral sensory organs, or intermediate nuclei, synapse in the thalamus, and thalamic neurons project directly to the cerebrum. It is a requisite synapse in any sensory pathway, except for olfaction. The thalamus does not just pass the information on, it also processes that information. For example, the portion of the thalamus that receives visual information will influence what visual stimuli are important, or what receives attention. The cerebrum also sends information down to the thalamus, which usually communicates motor commands. This involves interactions with the cerebellum and other nuclei in the brainstem. Inferior to the thalamus lies the subthalamic nucleus, which is part of the basal nuclei. The subthalamic nucleus connects various basal nuclei and modulate their activity.

Hypothalamus

Inferior and slightly anterior to the thalamus is the hypothalamus, the other major region of the diencephalon. The hypothalamus is a collection of nuclei that are largely involved in regulating homeostasis. The hypothalamus is the executive region in charge of the autonomic nervous system and the endocrine system through its regulation of the anterior pituitary gland. Other parts of the hypothalamus are involved in memory and emotion as part of the limbic system. Two groups of nuclei called the the mammillary bodies are located on the posterior part of the hypothalamus and are important for episodic memory, which is the memory of every day events that you can recollect.

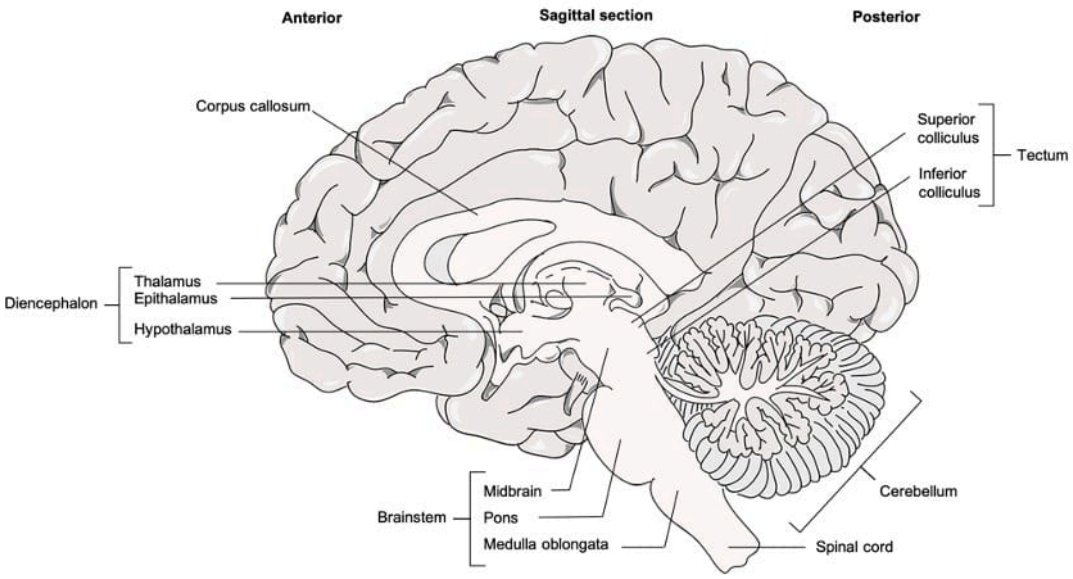


Figure 2.3.1: The diencephalon is composed of the thalamus, hypothalamus, and epithalamus and defines the walls of the third ventricle. The brainstem comprises three regions: the midbrain, the pons, and the medulla. Within the midbrain, the tectum is composed of the superior and inferior colliculi. (CC BY 4.0; via Brain - Diencephalon, Brainstem, Cerebellum and Limbic System). Alternate text description of the Figure 2.3.1 .

Brainstem

The midbrain and hindbrain (composed of the pons and the medulla oblongata) are collectively referred to as the brainstem (Figure 2.3.1 and Figure 2.3.2). The structure emerges from the ventral surface of the forebrain as a tapering cone that connects the brain to the spinal cord. Attached to the brainstem, but considered a separate region of the adult brain, is the cerebellum. The midbrain coordinates sensory representations of the visual, auditory, and somatosensory perceptual spaces. The pons is the main connection with the cerebellum. The pons and the medulla regulate several crucial functions, including the cardiovascular and respiratory systems (such as heart rhythm or breathing rate). The reticular activating system is found in the midbrain, pons, medulla and part of the thalamus and controls levels of wakefulness, maintains consciousness, enables people to pay attention to their environments, and is involved in sleep patterns and regulating the sleep cycle.

The cranial nerves connect through the brainstem and provide the brain with the sensory input and motor output associated with the head and neck, including most of the special senses. The major ascending and descending pathways between the spinal cord and brain, specifically the cerebrum, pass through the brainstem.

Midbrain

The mesencephalon, one of the original region of the embryonic brain, becomes the midbrain, a small region between the thalamus and pons. The cerebral aqueduct passes through the center of the midbrain, such that these regions are the roof and floor of that canal. Indeed, the midbrain is separated into the tectum and tegmentum, from the Latin words for roof and floor, respectively.

The tectum is composed of four bumps known together as the corpora quadrigemina or colliculi (singular = colliculus), which means “little hill” in Latin. The superior colliculus is the superior pair and combines sensory information about visual space, auditory space, and somatosensory space. At this level, the nucleus for the oculomotor nerve (CN III) is found. The inferior colliculus is the inferior pair of these enlargements and is part of the auditory pathway (trochlear nerve, CN IV). Neurons of the inferior colliculus project to the thalamus, which then sends auditory information to the cerebrum for the conscious perception of sound.

The tegmentum is continuous with the gray matter of the rest of the brainstem. The substantia nigra consists of basal nuclei with a black appearance due to melanin production. These nuclei control movement, emotional response, and the ability to experience pleasure and pain. Degeneration of the neurons within the substantia nigra is a hallmark of Parkinson disease.

The midbrain is connected to both the cerebrum and the cerebellum. On the anterolateral surfaces of the midbrain, motor tracts

called **cerebral peduncles** run from the cerebrum to the spinal cord. Moreover, the midbrain is connected to the cerebellum through the **superior cerebellar peduncle (SCP)**, a bundle of myelinated axons.

Pons

The pons is visible on the anterior surface of the brainstem as the thick bundle of white matter attached to the cerebellum. The name of the pons is derived from its connection to the cerebellum. The word means “bridge” and refers to the thick bundle of myelinated axons that form a bulge on its ventral surface. Those fibers are axons that project from the gray matter of the pons into the contralateral cerebellar cortex. These fibers make up the middle cerebellar peduncle (MCP) and are the major physical connection of the cerebellum to the brainstem (Figure 2.3.3). Gray matter regions of the pons contain neurons receiving descending input from the forebrain that is sent to the cerebellum. The pons houses autonomic nuclei important for respiration. The pons is also involved in sensory and motor information of some cranial nerves and houses the nuclei for cranial nerve V to VIII.

Medulla Oblongata

The medulla oblongata is the region known as the myelencephalon in the embryonic brain. The initial portion of the name, “myel,” refers to the significant white matter found in this region—especially on its exterior, which is continuous with the white matter of the spinal cord. The anterior region of the medulla oblongata shows two longitudinal ridges called pyramids, which house motor projection tracts (Figure 2.3.2). In the posterior region of the medulla oblongata, these pyramids cross to the opposite side at a point called the decussation of pyramids. Due to this crossing, each hemisphere controls motor responses of the opposite side of the body. Inferior to each pyramid, a nucleus called inferior olive, is located and functions as a relay point for information on proprioception coming from the spinal cord and going into the cerebellum. Indeed, the medulla oblongata send information to the cerebellum through tracts called inferior cerebellar peduncle (ICP) (Figure 2.3.3). The medulla oblongata contains several nuclei that are associated with last five cranial nerves (CN VIII to XII). It also contains autonomic nuclei important for vital functions such as respiration, heart rate, and blood pressure, among others.

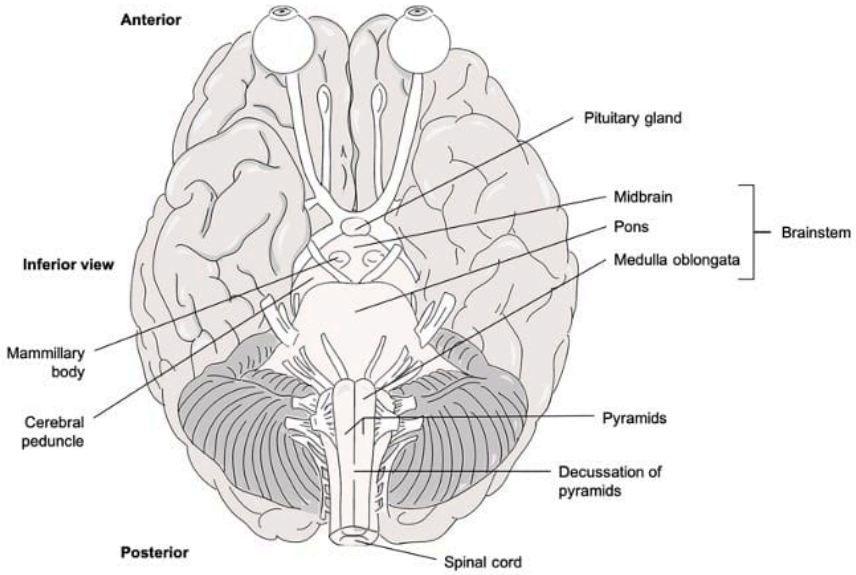


Figure 2.3.2: The inferior view of the brain shows the brainstem, cerebellum and cranial nerves. From anterior to posterior, the midbrain, pons and medulla oblongata form the brainstem. The cerebral peduncles of the midbrain and pyramids and their decussation of the medulla oblongata are also visible. The pituitary gland is attached to the hypothalamus, as are the mammillary bodies. (CC BY 4.0; via Brain - Diencephalon, Brainstem, Cerebellum and Limbic System). Alternate text description of the Figure 2.3.2.

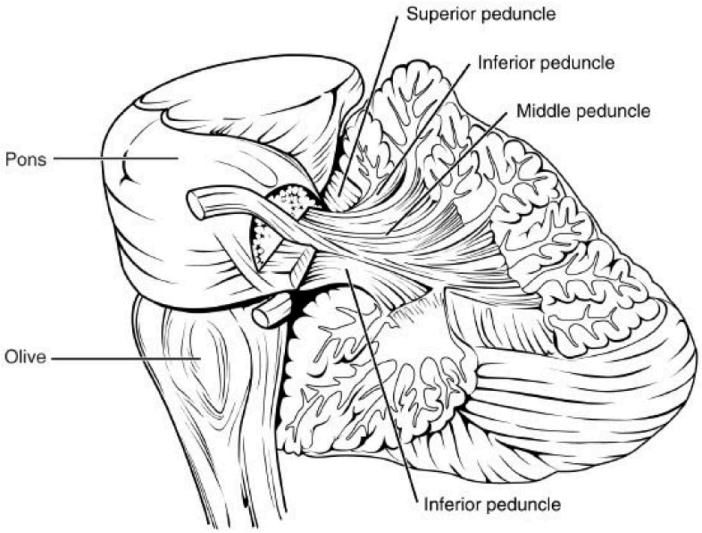


Figure 2.3.3:The connections to the cerebellum are the three cerebellar peduncles, which are close to each other. The superior cerebellar peduncle projects into the midbrain. The middle cerebellar peduncle is the ventral surface of the pons. The inferior cerebellar peduncle arises from the medulla—specifically from the inferior olive, which is visible as a bulge on the ventral surface of the brainstem. (CC BY 4.0; via Brain - Diencephalon, Brainstem, Cerebellum and Limbic System). Alternate text description of the Figure 2.3.3.

DISORDERS OF THE... Basal Nuclei: Parkinson's Disease

Parkinson's disease is a disorder of the basal nuclei, specifically of the substantia nigra, that demonstrates the effects of the direct and indirect pathways. Parkinson's disease is the result of neurons in the substantia nigra pars compacta dying. These neurons release dopamine into the striatum. Without that modulatory influence, the basal nuclei are stuck in the indirect pathway, without the direct pathway being activated. The direct pathway is responsible for increasing cortical movement commands. The increased activity of the indirect pathway results in the hypokinetic disorder of Parkinson's disease.

Parkinson's disease is neurodegenerative, meaning that neurons die that cannot be replaced, so there is no cure for the disorder. Treatments for Parkinson's disease are aimed at increasing dopamine levels in the striatum. Currently, the most common way of doing that is by providing the amino acid L-DOPA, which is a precursor to the neurotransmitter dopamine and can cross the blood-brain barrier. With levels of the precursor elevated, the remaining cells of the substantia nigra pars compacta can make more neurotransmitter and have a greater effect. Unfortunately, the patient will become less responsive to L-DOPA treatment as time progresses, and it can cause increased dopamine levels elsewhere in the brain, which are associated with psychosis or schizophrenia.

Cerebellum

The **cerebellum**, as the name suggests, is the "little brain". Similar to the brain, the cerebellum contains an outer layer of gray matter, an inner layer of white matter and deep **cerebellar nuclei** (Figure 2.3.4). The white matter of the cerebellum is called **arbor vitae** ("tree of life") for its appearance that reminds that of branches of a tree. The folds of the cerebellar cortex are called **folia**, which means "leaf".

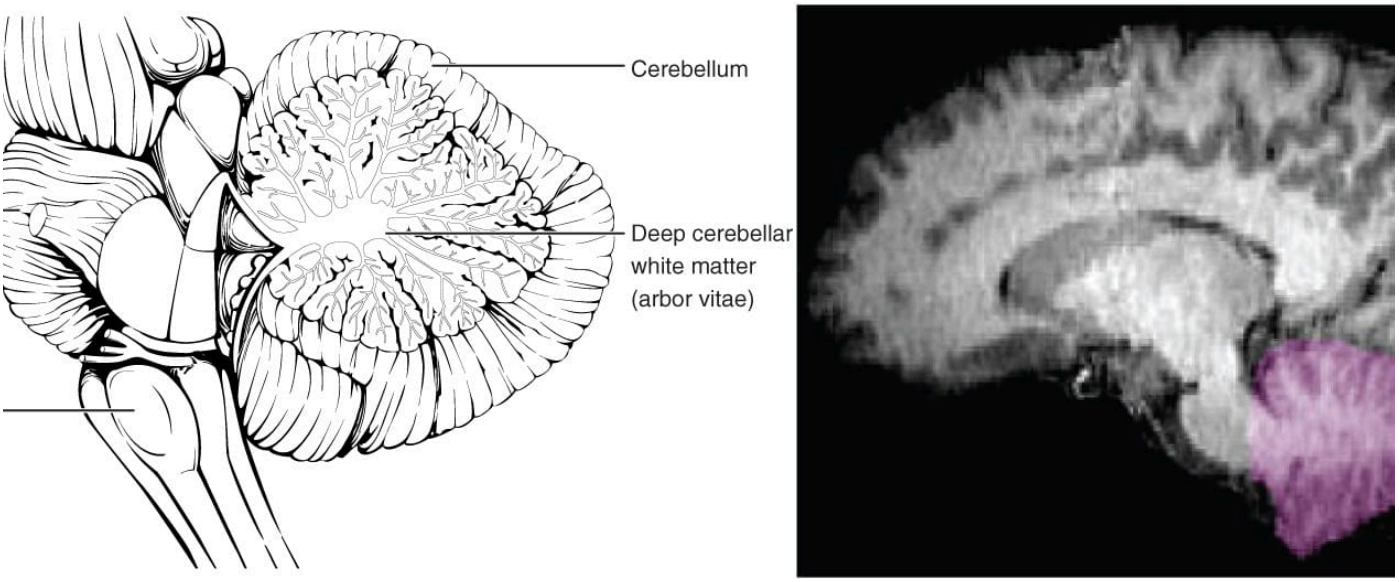


Figure 2.3.4: The cerebellum is situated on the posterior surface of the brainstem. This lateral view of a section of cerebellum shows the deep white matter forming the arbor vitae, which means "tree of life", while the cerebellar cortex is superficial. Descending input from the cerebellum enters through the large white matter structure of the pons. Ascending input from the periphery and spinal cord enters through the fibers of the inferior olive. Output goes to the midbrain, which sends a descending signal to the spinal cord. (CC BY 4.0; via Brain - Diencephalon, Brainstem, Cerebellum and Limbic System). Alternate text description of the Figure 2.3.4.

The cerebellum is divided into regions that are based on the particular functions and connections involved. The midline region of the cerebellum, called the **vermis**, is involved in comparing visual information, equilibrium, and proprioceptive feedback to maintain balance and coordinate movements such as walking, or gait (Figure 2.3.5). The vermis connects the two **cerebellar hemispheres**. Each hemisphere consists of two lobes, the **anterior lobe** and the **posterior lobe**, separated by the **primary fissure**. The lateral hemispheres are primarily concerned with planning motor functions through frontal lobe inputs that are returned through the thalamic projections back to the premotor and motor cortices. Processing in the midline regions targets movements of the axial musculature, whereas the lateral regions target movements of the appendicular musculature.

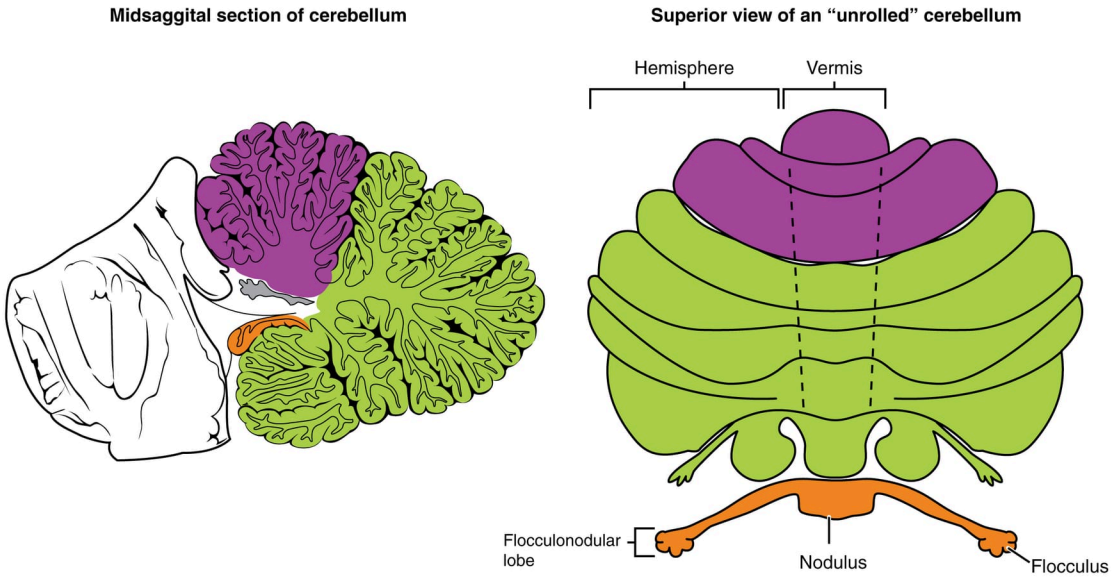


Figure 2.3.5: The cerebellum can be divided into two basic regions: the vermis, which is the midline, and the hemispheres, which are lateral to the vermis. Each cerebellar hemisphere consists of two lobes. The anterior lobe (in purple) and posterior lobe (in green) are separated by the primary fissure. (CC BY 4.0; via Brain - Diencephalon, Brainstem, Cerebellum and Limbic System). Alternate text description of the Figure 2.3.5.

The cerebellum is responsible for coordination of movement, alternating movement of arms and legs, balance, posture and gait. If the primary motor cortex of the frontal lobe sends a command down to the spinal cord to initiate walking, a copy of that instruction is sent to the cerebellum. Sensory feedback from the muscles and joints, proprioceptive information about the movements of

walking, and sensations of balance are sent to the cerebellum through the inferior olive and the cerebellum compares them. If walking is not coordinated, perhaps because the ground is uneven or a strong wind is blowing, then the cerebellum sends out a corrective command to compensate for the difference between the original cortical command and the sensory feedback. The output of the cerebellum is into the midbrain, which then sends a descending input to the spinal cord to correct the messages going to skeletal muscles.

There is also strong evidence of the cerebellar role in procedural memory. The two are not incompatible; in fact, procedural memory is motor memory, such as learning to ride a bicycle. Significant work has been performed to describe the connections within the cerebellum that result in learning.

DISORDERS OF THE... Cerebellum: Ataxia

A movement disorder of the cerebellum is referred to as **ataxia**. It presents as a loss of coordination in voluntary movements. Ataxia can also refer to sensory deficits that cause balance problems, primarily in proprioception and equilibrium. When the problem is observed in movement, it is ascribed to cerebellar damage. Sensory and vestibular ataxia would likely also present with problems in gait and station.

Ataxia is often the result of exposure to exogenous substances, focal lesions, or a genetic disorder. Focal lesions include strokes affecting the cerebellar arteries, tumors that may impinge on the cerebellum, trauma to the back of the head and neck, or MS. Alcohol intoxication or drugs such as ketamine cause ataxia, but it is often reversible. Mercury in fish can cause ataxia as well. Hereditary conditions can lead to degeneration of the cerebellum or spinal cord, as well as malformation of the brain, or the abnormal accumulation of copper seen in Wilson's disease.

Limbic System

The **limbic system** is a collection of structures of the cerebrum and diencephalon involved in emotion, motivation and memory associated with emotions. Although still debated, the structures mostly recognized in this system are the cingulate gyrus, hippocampus, amygdala, olfactory structures, and various nuclei of the diencephalon. Here we will focus on the cingulate gyrus, the hippocampus and amygdala as major structures (Figure 2.3.6). The **cingulate gyrus** is located in the longitudinal fissure, superior to the corpus callosum, surrounding the diencephalon. The cingulate gyrus focuses the attention to events that are emotionally important. The **hippocampus** is a nucleus shaped like a seahorse, hence its name. The hippocampus is essential in forming memories and storing them long-term. Lastly, the **amygdala** (or amygdaloid body) is connected to the hippocampus and is involved in multiple aspects of emotions, especially fear. It helps encoding memories based on the state of emotion that the person is in.

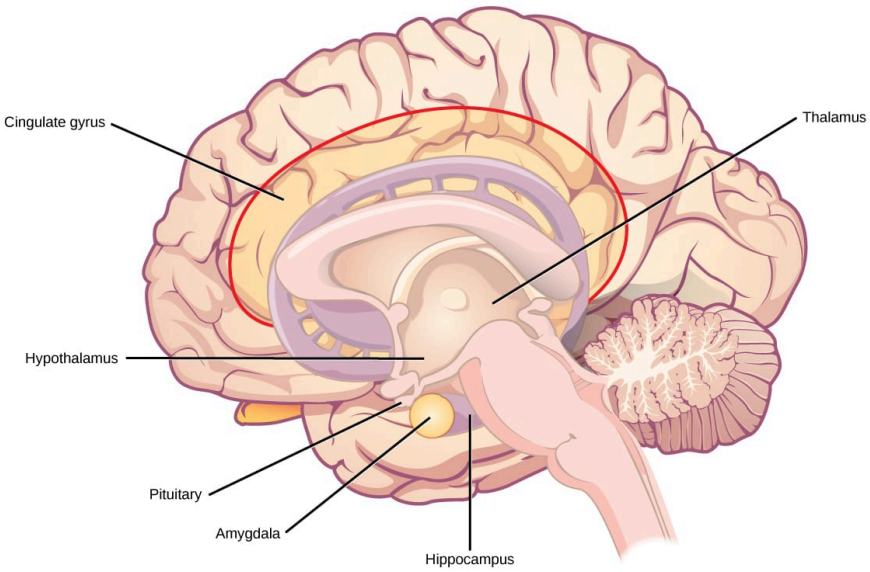


Figure 2.3.6: The limbic system regulates emotion and other behaviors. It includes parts of the cerebral cortex located near the center of the brain, including the cingulate gyrus and the hippocampus as well as the thalamus, hypothalamus and amygdala. The amygdala is inferior to the hypothalamus. (CC BY 4.0; via Brain - Diencephalon, Brainstem, Cerebellum and Limbic System). Alternate text description of the Figure 2.3.6.

Concept Review

The diencephalon includes the thalamus and the hypothalamus, along with some other structures. The thalamus is a relay between the cerebrum and the rest of the nervous system. The hypothalamus coordinates homeostatic functions through the autonomic and endocrine systems.

The brainstem is composed of the midbrain, pons, and medulla. It controls the head and neck region of the body through the cranial nerves. There are control centers in the brainstem that regulate the cardiovascular and respiratory systems.

The cerebellum is connected to the brainstem, primarily at the pons, where it receives a copy of the descending input from the cerebrum to the spinal cord. It can compare this with sensory feedback input through the medulla and send output through the midbrain that can correct motor commands for coordination. The cerebellum is an important part of motor function in the nervous system. It apparently plays a role in procedural learning, which would include motor skills such as riding a bike or throwing a football. The basis for these roles is likely to be tied into the role the cerebellum plays as a comparator for voluntary movement.

The limbic system includes structures of the cerebral cortex and diencephalon that are responsible for emotion and memory. The main structures are the cingulate gyrus, hippocampus and amygdala.

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2.4: Cranial Nerves

Nerves

The PNS is not as contained as the CNS because it is defined as everything that is not the CNS. Some peripheral structures are incorporated into the other organs of the body. In describing the anatomy of the PNS, it is necessary to describe the common structures, the nerves and the ganglia, as they are found in various parts of the body. Many of the neural structures that are incorporated into other organs are features of the digestive system; these structures are known as the enteric nervous system and are a special subset of the PNS.

Bundles of axons in the PNS are referred to as nerves. These structures in the periphery are different than the central counterpart, called a tract. Nerves are composed of more than just nervous tissue. They have connective tissues invested in their structure, as well as blood vessels supplying the tissues with nourishment. The outer surface of a nerve is a surrounding layer of fibrous connective tissue called the **epineurium**. Within the nerve, axons are further bundled into **fascicles**, which are each surrounded by their own layer of fibrous connective tissue called **perineurium**. Finally, individual axons are surrounded by loose connective tissue called the **endoneurium** (Figure 2.4.1 and Figure 2.4.2). These three layers are similar to the connective tissue sheaths for muscles. Nerves are associated with the region of the CNS to which they are connected, either as cranial nerves connected to the brain or spinal nerves connected to the spinal cord.

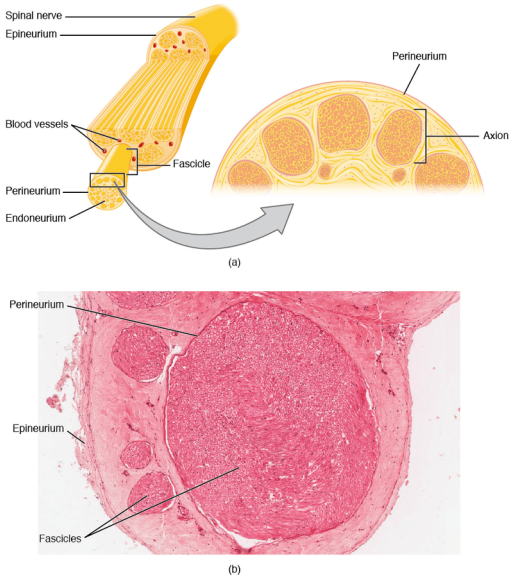


Figure 2.4.1: In (a) diagram of the organization of a nerve. The structure of a nerve is organized by the layers of connective tissue on the outside, around each fascicle, and surrounding the individual nerve fibers. In (b), micrograph of a nerve showing fascicles covered by perineurium, and the nerve covered by epineurium (tissue source: simian). LM x 40. (CC BY 3.0; via Cranial Nerves). Alternate text description of the Figure 2.4.1.



Figure 2.4.2: Magnified micrograph of a nerve trunk where individual axons are visible as circular structures covered by endoneurium (tissue source: simian). Axons are bundled into a fascicle covered by perineurium. LM x 1600. (CC BY 3.0; via Cranial Nerves). Alternate text description of the Figure 2.4.2.

Cranial Nerves

The nerves attached to the brain are the cranial nerves, which are primarily responsible for the sensory and motor functions of the head and neck (with the exception of one that targets organs in the thoracic and abdominal cavities as part of the parasympathetic nervous system). There are twelve cranial nerves, which are designated CNI through CNXII for "Cranial Nerve," using Roman numerals for 1 through 12, based on the anatomical location on the inferior view of the brain, from anterior to posterior (Figure 2.4.3). They can be classified as sensory nerves, motor nerves, or a combination of both, meaning that the axons in these nerves originate out of sensory ganglia external to the cranium or motor nuclei within the brainstem. Sensory axons enter the brain to synapse in a nucleus. Motor axons connect to skeletal muscles of the head or neck. Three of the nerves are solely composed of sensory fibers; five are strictly motor; and the remaining four are mixed nerves.

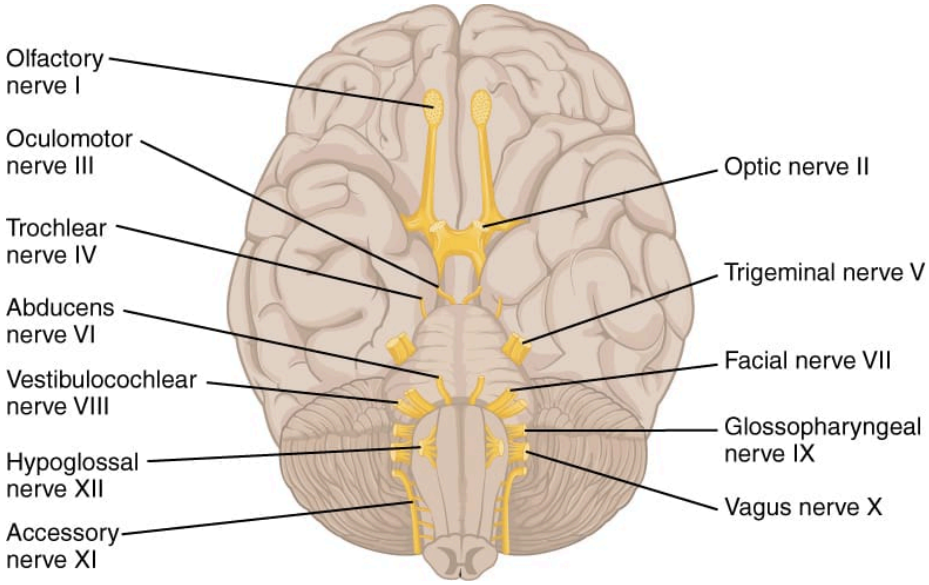


Figure 2.4.3: The anatomical arrangement of the roots of the cranial nerves observed from an inferior view of the brain. Cranial nerves are numbered with Roman numbers from 1 to 12 based on their exit from the brain, from anterior to posterior. (CC BY 3.0; via Cranial Nerves). Alternate text description of the Figure 2.4.3.

Learning the cranial nerves is a tradition in anatomy courses, and students have always used mnemonic devices to remember

the nerve names. A traditional mnemonic is the rhyming couplet, “Oh Oh Oh To Touch And Feel Very Good Velvet Ah Heaven,” in which the initial letter of each word corresponds to the initial letter in the name of each nerve. The names of the nerves have changed over the years to reflect current usage and more accurate naming. An exercise to help learn this sort of information is to generate a mnemonic using words that have personal significance. The names of the cranial nerves are listed in Table 2.4.1 along with a brief description of their function, their source (sensory ganglion or motor nucleus), and their target (sensory nucleus or skeletal muscle). The **olfactory nerve** and **optic nerve** are responsible for the sense of smell and vision, respectively. The **oculomotor nerve** is responsible for eye movements by controlling four of the extraocular muscles. It is also responsible for lifting the upper eyelid when the eyes point up, and for pupillary constriction. The **trochlear nerve** and the **abducens nerve** are both responsible for eye movement, but do so by controlling different extraocular muscles. The **trigeminal nerve** is responsible for cutaneous sensations of the face and controlling the muscles of mastication. The **facial nerve** is responsible for the muscles involved in facial expressions, as well as part of the sense of taste and the production of saliva. The **vestibulocochlear nerve** is responsible for the senses of hearing and balance. The **glossopharyngeal nerve** is responsible for controlling muscles in the oral cavity and upper throat, as well as part of the sense of taste and the production of saliva. The **vagus nerve** is responsible for contributing to homeostatic control of the organs of the thoracic and upper abdominal cavities. The **accessory nerve** is responsible for controlling the muscles of the neck, along with cervical spinal nerves. The **hypoglossal nerve** is responsible for controlling the muscles of the lower throat and tongue.

Four of these cranial nerves make up the cranial component of the autonomic nervous system responsible for pupillary constriction (oculomotor nerve), salivation and lacrimation (facial and glossopharyngeal nerves), and the regulation of the organs of the thoracic and upper abdominal cavities (vagus nerve).

Three of the cranial nerves also contain autonomic fibers, and a fourth is almost purely a component of the autonomic system. The oculomotor, facial, and glossopharyngeal nerves contain fibers that contact autonomic ganglia. The oculomotor fibers initiate pupillary constriction, whereas the facial and glossopharyngeal fibers both initiate salivation. The vagus nerve primarily targets autonomic ganglia in the thoracic and upper abdominal cavities.

Another important aspect of the cranial nerves that lends itself to a mnemonic is the functional role each nerve plays. The nerves fall into one of three basic groups. They are sensory, motor, or both (see Table 2.4.1). The sentence, “Some Say Marry Money But My Brother Says Brains Beauty Matter More,” corresponds to the basic function of each nerve. The first, second, and eighth nerves are purely sensory: the olfactory (CNI), optic (CNII), and vestibulocochlear (CNVIII) nerves. The three eye-movement nerves are all motor: the oculomotor (CNIII), trochlear (CNIV), and abducens (CNVI). The accessory (CNXI) and hypoglossal (CNXII) nerves are also strictly motor. The remainder of the nerves contain both sensory and motor fibers. They are the trigeminal (CNV), facial (CNVII), glossopharyngeal (CNIX), and vagus (CNX) nerves. The nerves that convey both are often related to each other. The trigeminal and facial nerves both concern the face; one concerns the sensations and the other concerns the muscle movements. The facial and glossopharyngeal nerves are both responsible for conveying gustatory, or taste, sensations as well as controlling salivary glands. The vagus nerve is involved in visceral responses to taste, namely the gag reflex. This is not an exhaustive list of what these combination nerves do, but there is a thread of relation between them.

Table 2.4.1: Cranial Nerves

Mnemonic	Number	Name	Function (S/M/B)	Central connection (nuclei)	Peripheral connection (ganglion or muscle)
Oh	I	Olfactory	Smell (S)	Olfactory bulb	Olfactory epithelium
Oh	II	Optic	Vision (S)	Hypothalamus/thalamus/midbrain	Retina (retinal ganglion cells)
Oh	III	Oculomotor	Eye movements (M)	Oculomotor nucleus	Extraocular muscles (other 4), levator palpebrae superioris, ciliary ganglion (autonomic)
To	IV	Trochlear	Eye movements (M)	Trochlear nucleus	Superior oblique muscle
Touch	V	Trigeminal	Sensory/motor – face (B)	Trigeminal nuclei in the midbrain, pons, and medulla	Trigeminal
And	VI	Abducens	Eye movements (M)	Abducens nucleus	Lateral rectus muscle

Mnemonic	Number	Name	Function (S/M/B)	Central connection (nuclei)	Peripheral connection (ganglion or muscle)
Feel	VII	Facial	Motor – face, Taste (B)	Facial nucleus, solitary nucleus, superior salivatory nucleus	Facial muscles, Geniculate ganglion, Pterygopalatine ganglion (autonomic)
Very	VIII	Vestibulocochlear	Hearing/balance (S)	Cochlear nucleus, Vestibular nucleus/ cerebellum	Spiral ganglion (hearing), Vestibular ganglion (balance)
Good	IX	Glossopharyngeal	Motor – throat Taste (B)	Solitary nucleus, inferior salivatory nucleus, nucleus ambiguus	Pharyngeal muscles, Geniculate ganglion, Otic ganglion (autonomic)
Velvet	X	Vagus	Motor/sensory – viscera (autonomic) (B)	Medulla	Terminal ganglia serving thoracic and upper abdominal organs (heart and small intestines)
Ah	XI	Accessory	Motor – head and neck (M)	Spinal accessory nucleus	Neck muscles
Heaven	XII	Hypoglossal	Motor – lower throat (M)	Hypoglossal nucleus	Muscles of the larynx and lower pharynx

Cranial Nerve Ganglia

A ganglion (ganglia for plural) is a group of neuron cell bodies in the peripheral nervous system. Ganglia can be categorized, for the most part, as either sensory ganglia or autonomic ganglia, referring to their primary functions. A type of sensory ganglion is a **cranial nerve ganglion**. The roots of cranial nerves are within the cranium, whereas the ganglia are outside the skull. For example, the **trigeminal ganglion** is superficial to the temporal bone whereas its associated nerve is attached to the mid-pons region of the brainstem. The neurons of cranial nerve ganglia are also unipolar in shape with associated satellite cells.

AGING AND THE...Nervous System: Anosmia

Anosmia is the loss of the sense of smell. It is often the result of the olfactory nerve being severed, usually because of blunt force trauma to the head. The sensory neurons of the olfactory epithelium have a limited lifespan of approximately one to four months, and new ones are made on a regular basis. The new neurons extend their axons into the CNS by growing along the existing fibers of the olfactory nerve. The ability of these neurons to be replaced is lost with age. Age-related anosmia is not the result of impact trauma to the head, but rather a slow loss of the sensory neurons with no new neurons born to replace them.

Smell is an important sense, especially for the enjoyment of food. There are only five tastes sensed by the tongue, and two of them are generally thought of as unpleasant tastes (sour and bitter). The rich sensory experience of food is the result of odor molecules associated with the food, both as food is moved into the mouth, and therefore passes under the nose, and when it is chewed and molecules are released to move up the pharynx into the posterior nasal cavity. Anosmia results in a loss of the enjoyment of food.

As the replacement of olfactory neurons declines with age, anosmia can set in. Without the sense of smell, many sufferers complain of food tasting bland. Often, the only way to enjoy food is to add seasoning that can be sensed on the tongue, which usually means adding table salt. The problem with this solution, however, is that this increases sodium intake, which can lead to cardiovascular problems through water retention and the associated increase in blood pressure.

Concept Review

The PNS is composed of the groups of neurons (ganglia) and bundles of axons (nerves) that are outside of the brain and spinal cord. Nerves are organized into structures by layers of connective tissue that cover them. The epineurium covers the nerve, the perineurium covers the fascicles and the endoneurium covers the individual axon.

Nerves are classified as cranial nerves or spinal nerves on the basis of their connection to the brain or spinal cord, respectively. The twelve cranial nerves can be strictly sensory in function, strictly motor in function, or a combination of the two functions. The olfactory nerve (CN I) and optic nerve (CN II) are responsible for the sense of smell and vision, respectively. The oculomotor nerve (CN III) is responsible for eye movements, lifting the upper eyelid and size of the pupil. The trochlear nerve (CN IV) and the abducens nerve (CN VI) are both responsible for eye movement, but do so by controlling different extraocular muscles. The trigeminal nerve (CN V) is responsible for cutaneous sensations of the face and controlling the muscles of mastication. The facial nerve (VII) is responsible for the muscles involved in facial expressions, as well as part of the sense of taste and the production of saliva. The vestibulocochlear nerve (VIII) is responsible for the senses of hearing and balance. The glossopharyngeal nerve (IX) is responsible for controlling muscles in the oral cavity and upper throat, as well as part of the sense of taste and the production of saliva. The vagus nerve (CN X) is responsible for contributing to homeostatic control of the organs of the thoracic and upper abdominal cavities. The accessory nerve (CN XI) is responsible for controlling the muscles of the neck, along with cervical spinal nerves. The hypoglossal nerve (CN XII) is responsible for controlling the muscles of the lower throat and tongue.

Ganglia are of two types, sensory or autonomic. Sensory ganglia contain unipolar sensory neurons and are associated with many of the cranial nerves.

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2.5: Spinal Cord and Spinal Nerves

Protective Coverings of the Spinal Cord

The spinal cord is continuous with the brain and transmits sensory information from the periphery of the body to the brain, and motor information from the brain to the periphery. It is also a center for coordination of reflexes, as well as control of reflexes independently from the brain. Along the spinal cord, spinal nerves emerge that carry both sensory and motor information.

The spinal cord, as the brain, is protected by meninges, which are continuous with the cranial meninges. From outermost to innermost, the spinal meninges are: **dura mater**, **arachnoid mater** and **pia mater**. The spinal meninges are similar to the cranial ones, except for the dura mater. The spinal dura mater consists of a single layer of connective tissue, differing from the cranial dura mater. Moreover, at each intervertebral foramen, the dura mater extends and fuses with the connective tissue that covers spinal nerves.

One space between the spinal meninges also differs slightly from the ones present around the brain. The **epidural space** between the periosteum and dura mater is a real space around the spinal cord and houses areolar and adipose connective tissues, and blood vessels (Figure 2.5.2). Epidural anesthesia is injected in this space. The subarachnoid space is filled with circulating CSF, which also provides a liquid cushion to the brain and spinal cord. Similar to clinical blood work, a sample of CSF can be withdrawn to find chemical evidence of neuropathology or metabolic traces of the biochemical functions of nervous tissue. Because the spinal cord does not extend through the lower lumbar region of the vertebral column, a needle can be inserted through the dura and arachnoid layers to withdraw CSF. This procedure is called a **lumbar puncture** and avoids the risk of damaging the central tissue of the spinal cord.

Blood Supply to the Spinal Cord

The spinal cord is supported by an extensive collateral supply and drainage of blood. Three longitudinal **spinal arteries** supply the anterior and posterior regions of the spinal cord. The anterior and posterior spinal arteries are connected by cross-connections of pia mater called the **arterial vasocorona** which encircles the cord and supplies the lateral portion of the spinal cord. The blood supply is reinforced by numerous arteries that pass through the intervertebral foramina and divide into anterior and posterior **radicular arteries** and have variable connections with the spinal arteries. Numerous veins and venous plexuses surround the spinal cord and drain the blood from it. These veins share the same names as the arteries that supply the spinal cord.

Spinal Cord

The description of the CNS is concentrated on the structures of the brain, but the spinal cord is another major organ of the system. The length of the spinal cord is divided into regions that correspond to the regions of the vertebral column. As shown in Figure 2.5.1, immediately inferior to the medulla oblongata is the **cervical region** of the spinal cord that contains motor neurons whose axons, together with sensory axons, extend into cervical spinal nerves. Inferior to the cervical region is the **thoracic region**, that contains neurons for the thoracic spinal nerves. The **lumbar region** is a short segment that contains neurons for the lumbar spinal nerves. Finally the **sacral region** contain neurons for the sacral nerves. The spinal cord is not the full length of the vertebral column because the spinal cord does not grow significantly longer after the first or second year, but the skeleton continues to grow. For this reason, the names of the spinal cord regions and vertebral regions to not match up. The lumbar and sacral spinal cord segments are found between vertebral levels T9 and L2. The spinal cord ends around the L1/L2 vertebral level, forming a tapered structure known as the **conus medullaris**, which corresponds to the sacral spinal cord. The nerves that emerge from the spinal cord pass through the intervertebral foramina at the corresponding vertebral segments. For example, lumbar nerves exit the vertebral canal at the respective lumbar vertebrae. As the vertebral column grows, these nerves grow with it and result in a long bundle of nerves inferior to the conus medullaris that resembles a horse's tail and therefore is named the **cauda equina**. Within the cauda equina, there is a thin strand of pia mater called **filum terminale** that helps anchoring the conus medullaris to the coccyx. At each intervertebral foramen, the spinal dura mater extends between vertebrae and surrounds the spinal nerves.

The spinal cord has a diameter of approximately 3/4 of an inch (just less than 2 cm) and its length ranges between 16 to 18 inches (41-45 cm). However, its diameter changes between the different regions because the ratio of white and gray matter changes to reflect the functions of the different regions. The cervical and lumbar regions of the spinal cord show a larger diameter compared

to the rest of the regions. The **cervical enlargement** and **lumbar enlargement** represent an increased amount of neurons in the gray matter and axons in the white matter that serve the upper limbs and lower limbs, respectively.

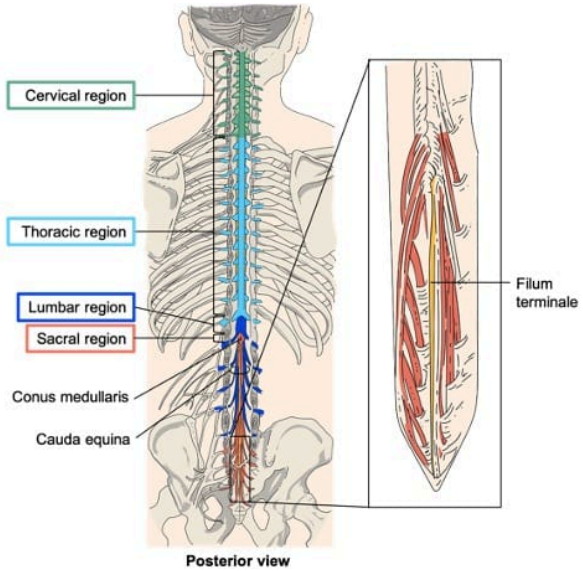


Figure 2.5.1: The spinal cord is divided into four regions: cervical, thoracic, lumbar and sacral. The sacral region has a tapered end called the conus medullaris. The bundle of axons inferior to the conus medullaris is the cauda equina. The cauda equina is anchored to the coccyx by the filum terminale, a thin strand of pia mater. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.1.

Whereas the brain develops out of expansions of the neural tube into primary and then secondary vesicles, the spinal cord maintains the tube structure and is only specialized into certain regions. As the spinal cord continues to develop in the newborn, anatomical features mark its surface. Viewed in cross section, the anterior midline is marked by the **anterior median fissure**, and the posterior midline is marked by the **posterior median sulcus**. As shown in Figure 2.5.2, axons coming from the **dorsal (posterior) root ganglion** enter the posterior side through the **dorsal (posterior) nerve root**, which marks the **posterolateral sulcus** on either side. The axons emerging from the anterior side do so through the **ventral (anterior) nerve root**. Note that it is common to see the terms dorsal (dorsal = "back") and ventral (ventral = "belly") used interchangeably with posterior and anterior, particularly in reference to nerves and the structures of the spinal cord. You should learn to be comfortable with both. The posterior and anterior nerve roots fuse together to form the **spinal nerves**. In the central region of the spinal cord, a **central canal** is the continuation of the fourth ventricle of the brain and contains cerebrospinal fluid (CSF).

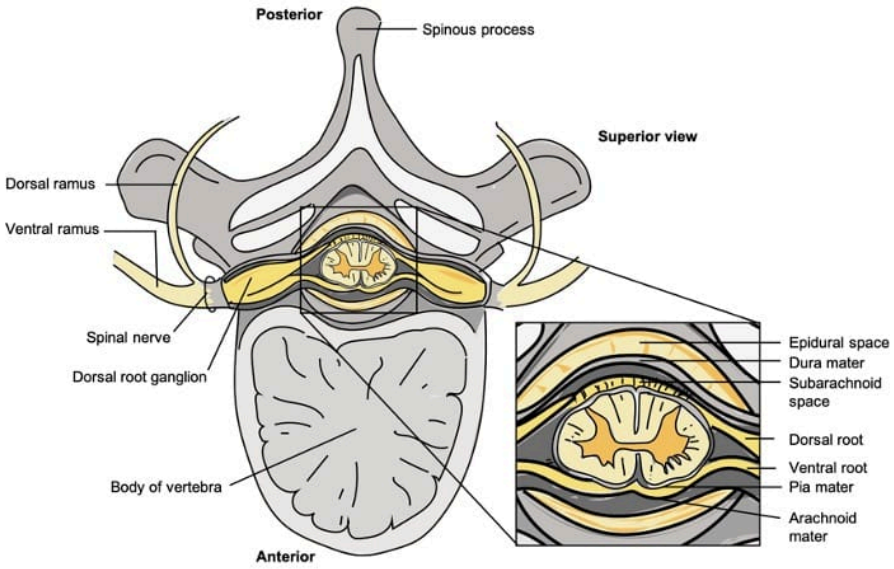


Figure 2.5.2: The superior view of a cross section of spinal cord within the vertebra shows the dorsal and ventral roots emerging from the spinal cord and fusing to form the spinal nerve. On the dorsal root, a dorsal root ganglion contain sensory neurons. The spinal cord is wrapped in meninges, as shown in the figure enlargement. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.2.

Gray Horns

In cross-section, the gray matter of the spinal cord has the appearance of an ink-blot test, with the spread of the gray matter on one side replicated on the other—a shape reminiscent of a bulbous capital "H." As shown in Figure 2.5.3, the gray matter is subdivided into regions that are referred to as horns. The **posterior horn** is responsible for sensory processing and contain the axons of sensory neurons and the cell bodies of interneurons. The axons of sensory neurons are bundled into the posterior nerve root and are coming directly from the posterior root ganglion which contains cell bodies of sensory neurons. The **lateral horn**, which is only found in the thoracic, upper lumbar, and sacral regions, is a central component of the autonomic nervous system and contains cell bodies of autonomic motor neurons which innervate cardiac muscle, smooth muscle and glands.The **anterior horn** contains the cell bodies of somatic motor neurons that control skeletal muscles. The somatic motor neurons are the largest neurons of the body and are classified as multipolar neurons. The fibers that cause contraction of skeletal muscles are the axons of these neurons, which exit the spinal cord through the anterior nerve root. The posterior and anterior nerve roots fuse to form the spinal nerve, which contains mixed sensory and motor information.The motor neuron that causes contraction of the big toe, for example, is located in the sacral spinal cord. The axon that has to reach all the way to the belly of that muscle may be a meter in length. The neuronal cell body that maintains that long fiber must be quite large, possibly several hundred micrometers in diameter, making it one of the largest cells in the body. The neurons of the spinal cord are organized into functional groups called nuclei representing their respective functions (somatic sensory, visceral sensory, somatic motor and autonomic motor). Surrounding the central canal, a horizontal line of gray matter called the **gray commissure** connects the left and right sides of the spinal cord.

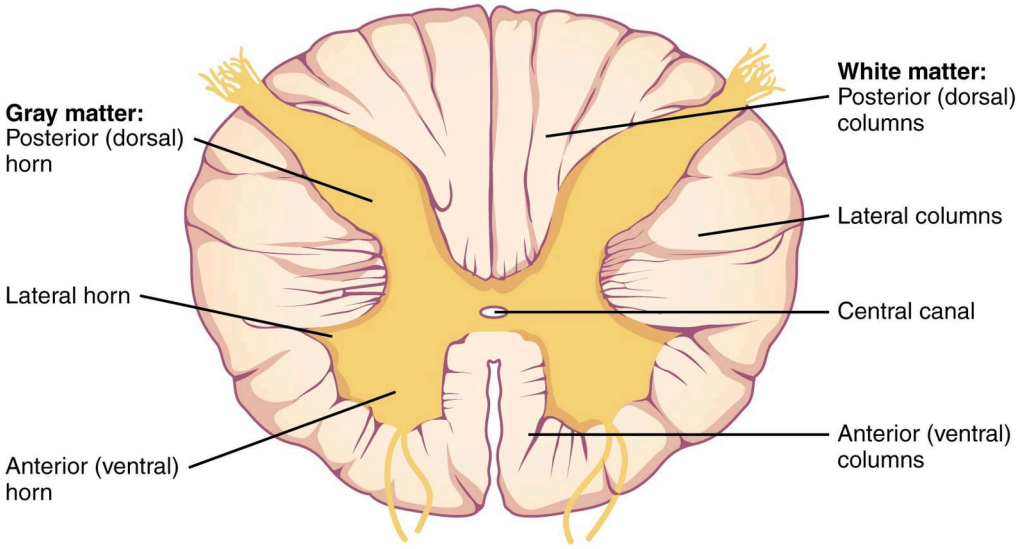


Figure 2.5.3: The cross-section of a thoracic spinal cord segment shows the posterior, anterior, and lateral horns of gray matter, as well as the posterior, anterior, and lateral columns of white matter. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.3.



Figure 2.5.4: A micrograph shows the histology of the spinal cord. LM × 40. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.4.

White Columns

Just as the gray matter is separated into horns, the white matter of the spinal cord is separated into columns. **Ascending tracts** of nervous system fibers in these columns carry sensory information up to the brain, whereas **descending tracts** carry motor commands from the brain. Looking at the spinal cord longitudinally, the columns extend along its length as continuous bands of white matter. Between the two posterior horns of gray matter are the **posterior columns**. The posterior columns are composed of axons of ascending tracts. Between the two anterior horns, and bounded by the axons of motor neurons emerging from that gray matter area, are the **anterior columns**. The white matter on either side of the spinal cord, between the posterior horn and the axons of the anterior horn neurons, are the **lateral columns**. The anterior and lateral columns are composed of many different groups of axons of both ascending and descending tracts—the latter carrying motor commands down from the brain to the spinal cord to control output to the periphery. The anterior columns are connected by a bundle of axons called **white commissure** that crosses the central region of the spinal cord.

Dorsal Root Ganglion

A ganglion (ganglia for plural) is a group of neuron cell bodies in the peripheral nervous system. Ganglia can be categorized, for the most part, as either sensory ganglia or autonomic ganglia, referring to their primary functions. The most common type of sensory ganglion is a dorsal (posterior) root ganglion. These ganglia are the cell bodies of neurons with axons that are sensory endings in the periphery, such as in the skin, and that extend into the CNS through the dorsal nerve root. The ganglion is an enlargement of the nerve root. Under microscopic inspection, it can be seen to include the cell bodies of the neurons, as well as bundles of fibers that are the posterior nerve root (Figure 2.5.5). The cells of the dorsal root ganglion are unipolar cells. Also, the small round nuclei of satellite cells can be seen surrounding—as if they were orbiting—the neuron cell bodies (Figure 2.5.6).

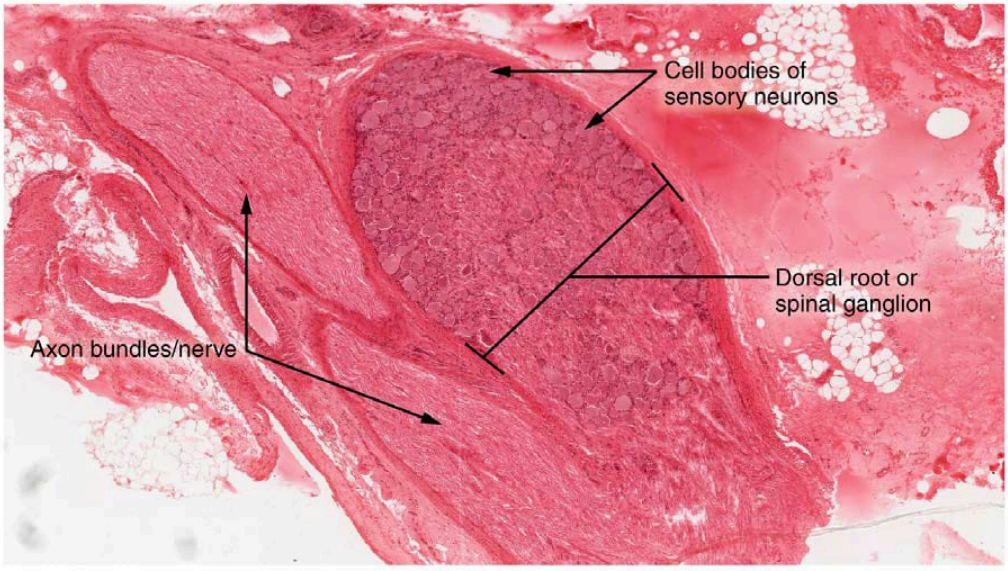


Figure 2.5.5: The cell bodies of sensory neurons, which are unipolar neurons by shape, are seen in this micrograph. Also, the fibrous region is composed of the axons of these neurons that are passing through the ganglion to be part of the dorsal nerve root (tissue source: canine). LM x 40. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.5.

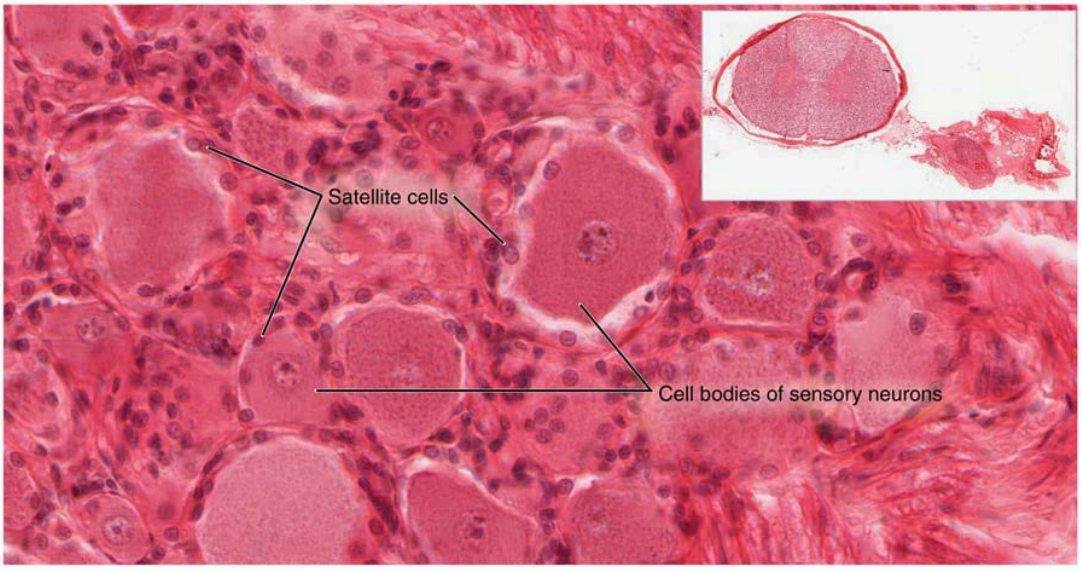


Figure 2.5.6: The micrograph includes both a cross-section of the lumbar spinal cord and a magnified section of the dorsal root ganglion showing cell bodies of sensory neurons surrounded by satellite cells (tissue source: canine). LM x 1600. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.6.

Spinal Nerves

The nerves connected to the spinal cord are the spinal nerves. The arrangement of these nerves is much more regular than that of the cranial nerves. All of the spinal nerves are combined sensory and motor axons that separate into two nerve roots. The sensory axons enter the spinal cord as the dorsal nerve root. The motor fibers, both somatic and autonomic, emerge as the ventral nerve root.

There are 31 spinal nerves, named for the level of the spinal cord at which each one emerges. There are eight pairs of cervical nerves designated C1 to C8, twelve thoracic nerves designated T1 to T12, five pairs of lumbar nerves designated L1 to L5, five pairs of sacral nerves designated S1 to S5, and one pair of coccygeal nerves. The nerves are numbered from the superior to inferior positions, and each emerges from the vertebral column through the intervertebral foramen at its level. The first nerve, C1, emerges between the first cervical vertebra and the occipital bone. The second nerve, C2, emerges between the first and second cervical vertebrae. The same occurs for C3 to C7, but C8 emerges between the seventh cervical vertebra and the first thoracic

vertebra. For the thoracic and lumbar nerves, each one emerges between the vertebra that has the same designation and the next vertebra in the column. The sacral nerves emerge from the sacral foramina along the length of that unique vertebra.

Spinal nerves extend outward from the vertebral column to innervate the periphery. The nerves in the periphery are not straight continuations of the spinal nerves, but rather the reorganization of the axons in those nerves to follow different courses. Axons from different spinal nerves will come together into a **systemic nerve**. This occurs at four places along the length of the vertebral column, each identified as a **nerve plexus**, whereas the other spinal nerves directly correspond to nerves at their respective levels. In this instance, the word plexus is used to describe networks of nerve fibers with no associated cell bodies.

After exiting the intervertebral foramen, each spinal nerve splits into two branches called **rami** (ramus for singular) (Figure 2.5.7). On the posterior side, the **dorsal ramus** is a short nerve that innervates the deep muscle and skin of the back. On the anterior side, the **ventral ramus** splits into multiple branches that eventually can form nerve plexuses. The ventral ramus innervates the anterior and lateral portions of the trunk, the upper and the lower limbs. Spinal nerves are also associated with other branches called **rami communicantes** which are formed by the gray and white rami and connect the spinal nerve to the autonomic ganglia.

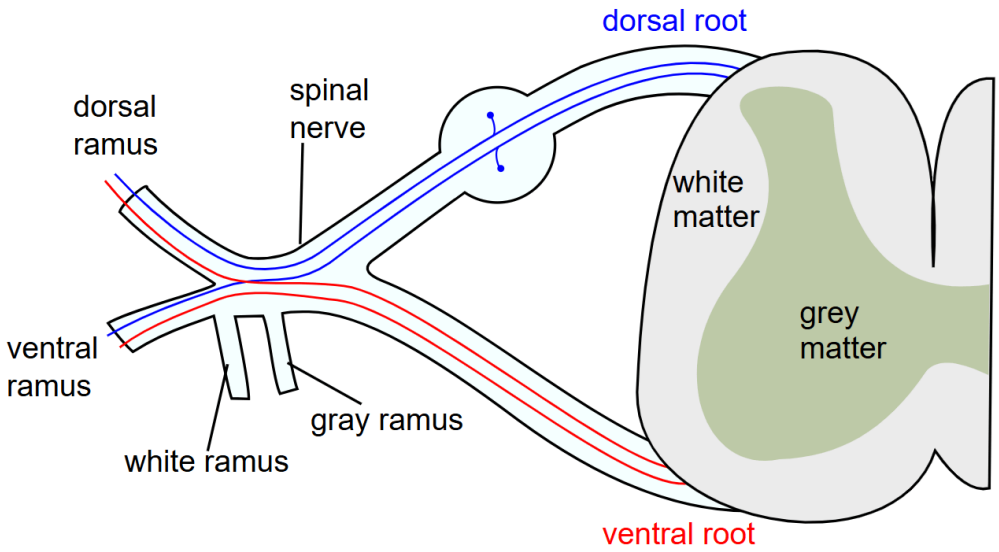


Figure 2.5.7: The ventral and dorsal nerve roots exiting a cross section of spinal cord fuse together to form a spinal nerve, which then splits into the dorsal and ventral rami. The white and gray ramus form the rami communicantes that connect spinal nerves to autonomic ganglia. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.7.

Of the four nerve plexuses, two are found at the cervical level, one at the lumbar level, and one at the sacral level (Figure 2.5.7). The **cervical plexus** is composed of axons from spinal nerves C1 through C5 and branches into nerves in the posterior neck and head, as well as the **phrenic nerve**, which connects to the diaphragm at the base of the thoracic cavity.

The other plexus from the cervical level is the **brachial plexus**. Spinal nerves C5 through T1 reorganize through this plexus to give rise to the nerves of the arms, as the name brachial suggests. The ventral rami of the brachial plexus rearrange to form the superior, middle and inferior **trunks**. At the clavicle, the trunks split into an **anterior division** and **posterior division**, which innervate the anterior and posterior parts of the upper limbs, respectively. At the axilla, the divisions converge to form the posterior, medial and lateral **cords**. Finally, the cords split into five **terminal branches**: the axillary, median, musculocutaneous, radial and ulnar nerves. The **axillary nerve** innervates the deltoid and teres minor muscles, and receives sensation from the superolateral side of the arm. The **median nerve** innervates the most anterior forearm muscles as well as muscles that move the thumb. It receives sensation from the palmar region and dorsal tips of the lateral 3 and a half digits. The **musculocutaneous nerve** innervate the anterior arm muscles and receives sensation from the lateral region of the forearm. The **radial nerve** innervates muscles and receives sensation from the posterior arm and forearm, including the dorsal region of the lateral three digits (not including their tips). The **ulnar nerve** innervates anterior forearm muscles as well as intrinsic hand muscles. It receives sensation from the dorsal and palmar regions of the medial one and a half digits.

The **lumbar plexus** arises from all the lumbar spinal nerves (L1 to L5) and gives rise to nerves innervating the pelvic region and the anterior leg. The **femoral nerve** is one of the major nerves from this plexus, which gives rise to the **saphenous nerve** as a branch that extends through the anterior lower leg. The femoral nerve innervates anterior thigh muscles and receives sensory information from the anterior and medial aspects of the thigh.

The **sacral plexus** comes from the lower lumbar nerves L4 and L5 and the sacral nerves S1 to S4. The most significant systemic nerve to come from this plexus is the **sciatic nerve**, which is a combination of the **tibial nerve** and the **fibular nerve**. The sciatic nerve extends across the hip joint and is the largest and longest nerve of the body. Through the tibial and fibular nerves, the sciatic nerve innervates the muscles and receives sensation of the posterior thigh and lower leg. It is most commonly associated with the condition **sciatica**, which is the result of compression or irritation of the nerve or any of the spinal nerves giving rise to it.

Spinal nerves of the thoracic region, T2 through T11, are not part of the plexuses but rather emerge and give rise to the **intercostal nerves** found between the ribs, which articulate with the vertebrae surrounding the spinal nerve. The intercostal nerves innervate the intercostal muscles and receive sensation from the skin of the anterior and lateral sides of the thoracic region. The last nerves (T7 to T12) also innervate the abdominal muscles and their overlying skin.

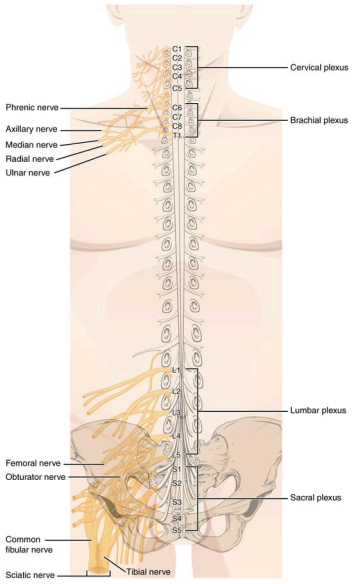


Figure 2.5.8: There are four main nerve plexuses in the human body. The cervical plexus supplies nerves to the posterior head and neck, as well as to the diaphragm. The brachial plexus supplies nerves to the arm. The lumbar plexus supplies nerves to the anterior leg. The sacral plexus supplies nerves to the posterior leg. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.8.

Dermatomes

The spinal nerves, which contain sensory fibers with endings in the skin, connect with the skin in a topographically organized manner, illustrated as **dermatomes** (Figure 2.5.9). For example, the fibers of eighth cervical nerve innervate the medial surface of the forearm and extend out to the fingers. All spinal nerves except C1 innervates a region on the skin. Dermatomes are important in a clinical setting as they can help identify potential damage to spinal nerves. In a sensory exam, the dermatome of a specific area is tested for sensation (for example the stroking of a cotton ball) to evaluate the functioning of the spinal nerve connected to that dermatome. Dermatomes are also associated with referred visceral pain, as the spinal nerves innervated the regions of the skin also innervate internal organs. Pain from the internal organ can then be mistakenly referred to a dermatome. For example, myocardial infarction is perceived as referred pain in the chest, left shoulder and left arm. This is because the sensory fibers that innervate the cardiac tissue travel through the T1-T4 spinal nerves, which also innervate the medial and anterior side of the body.

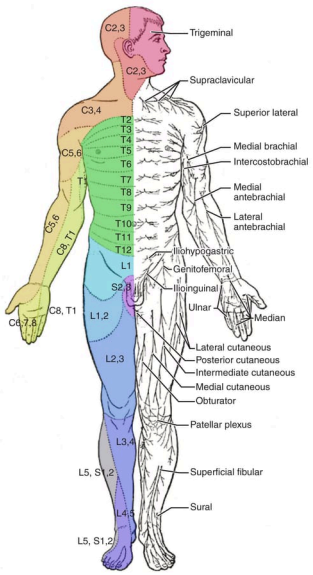


Figure 2.5.9: The surface of the skin can be divided into topographic regions that relate to the location of sensory endings in the skin based on the spinal nerve that contains those fibers. (CC BY 3.0; via Spinal Cord and Spinal Nerves). Alternate text description of the Figure 2.5.9.

Concept Review

The spinal cord is covered and protected by the spinal meninges, similar to the cranial ones. However, the spinal dura mater presents only one layer of tissue. Moreover, the epidural space is a real space that houses areolar and adipose connective tissues, and blood vessels.

The spinal cord is divided into segments that take the same name as the vertebral regions. However, the sacral spinal cord ends at the vertebral lumbar level (L1/L2) with the conus medullaris, while the spinal nerves continue exiting at their respective region of the vertebral column. Inferior to the conus medullaris, the spinal nerves form a bundle called the cauda equina. The spinal cord presents two enlargements at the cervical and lumbar level.

In cross section, the gray matter is deep within the spinal cord and is separated into horns. The sensory axons coming from the posterior root ganglia enter the spinal cord through the posterior nerve root and synapse in the posterior horn, where interneurons are found. Autonomic motor neurons are found in the lateral horns, while somatic motor neurons are found in the anterior horns. Motor neurons send their axons out of the spinal cord through the anterior nerve root. The posterior and anterior root fuse to form the spinal nerve, which contains mixed sensory and motor information. The white matter is separated into columns which contain axons of sensory and motor neurons. The posterior columns contain ascending sensory tracts which are bundles of axons going towards the brain. The lateral and anterior column contain ascending sensory tracts as well as descending motor tracts, which carry the motor responses from the brain. Overall, the posterior regions of the spinal cord are responsible for ascending sensory information, while the anterior regions are responsible for descending motor information.

Spinal nerves are all mixed nerves with both sensory and motor fibers. Spinal nerves emerge from the spinal cord and reorganize through plexuses, which then give rise to systemic nerves. Thoracic spinal nerves are not part of any plexus, but give rise to the intercostal nerves directly.

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2.6: Terms and Definitions

Key Terms

Select to View Definition

arachnoid granulation
outpocket of the arachnoid membrane into the dural sinuses that allows for reabsorption of CSF into the blood

arachnoid mater
middle layer of the meninges named for the spider-web–like trabeculae that extend between it and the pia mater

arachnoid trabeculae
filaments between the arachnoid and pia mater within the subarachnoid space

basilar artery
blood vessel from the merged vertebral arteries that runs along the dorsal surface of the brainstem

central canal
hollow space within the spinal cord that is the remnant of the center of the neural tube

cerebral aqueduct
connection of the ventricular system between the third and fourth ventricles located in the midbrain

choroid plexus
specialized structures containing ependymal cells lining blood capillaries that filter blood to produce CSF in the four ventricles of the brain

circle of Willis
unique anatomical arrangement of blood vessels around the base of the brain that maintains perfusion of blood into the brain even if one component of the structure is blocked or narrowed

common carotid artery
blood vessel that branches off the aorta (or the brachiocephalic artery on the right) and supplies blood to the head and neck

confluence of sinuses
connecting point of the superior sagittal sinus, straight sinus, and occipital sinus

cranial dura septa
extensions of meningeal dura mater into the cranial cavity

diaphragma sellae
extension of the dura that separates the pituitary from the neural structures located superiorly including the optic chiasm

dura mater
tough, fibrous, outer layer of the meninges that is attached to the inner surface of the cranium and vertebral column and surrounds the entire CNS

dural sinus
any of the venous structures surrounding the brain, enclosed within the dura mater, which drain blood from the CNS to the common venous return of the jugular veins

epidural space
area between the dura mater and the above structures

falx cerebelli
extension of dura mater, projecting to the cerebellum

falx cerebri
extension of dura mater that lies between the cerebral hemispheres, in the longitudinal fissure

fourth ventricle
the portion of the ventricular system that is in the region of the brainstem and opens into the subarachnoid space through the median and lateral apertures

internal carotid artery
branch from the common carotid artery that enters the cranium and supplies blood to the brain

interventricular foramina
openings between the lateral ventricles and third ventricle allowing for the passage of CSF

jugular veins
blood vessels that return “used” blood from the head and neck

lateral apertures
pair of openings from the fourth ventricle to the subarachnoid space on either side and between the medulla and cerebellum

lateral ventricles
portions of the ventricular system that are deep within the brain

median aperture
singular opening from the fourth ventricle into the subarachnoid space at the midline between the medulla and cerebellum

meningeal layer
one of the two layers of the cranial dura mater

meninges
protective outer coverings of the CNS composed of connective tissue

occipital sinuses
dural sinuses along the edge of the most posterior region of the brain

periosteal layer
one of the two layers of the cranial dura mater

pia mater
thin, innermost membrane of the meninges that directly covers the surface of the CNS

sigmoid sinuses
dural sinuses that drain directly into the jugular veins

straight sinus
dural sinus that drains blood from the deep center of the brain to collect with the other sinuses

subarachnoid space
space between the arachnoid mater and pia mater that contains CSF and the fibrous connections of the arachnoid trabeculae

subdural space
space between the dura mater and arachnoid mater

superior sagittal sinus
dural sinus that runs along the top of the longitudinal fissure and drains blood from the majority of the outer brain

tentorium cerebelli
: extension of dura mater that covers the upper surface of the cerebellum

third ventricle
: portion of the ventricular system that is in the region of the diencephalon

transverse sinuses
: dural sinuses that drain along either side of the occipital–cerebellar space

ventricles
: remnants of the hollow center of the neural tube that are spaces for cerebrospinal fluid to circulate through the brain

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2.7: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. Which structure is part of the central nervous system (CNS)?
   A. Spinal cord
   B. Spinal nerve
   C. Cranial nerve V
   D. Neuromuscular junction

2. Which structure is part of the peripheral nervous system (PNS)?
   A. Cerebrum
   B. Cerebellum
   C. Spinal cord
   D. Sciatic nerve

3. The main function of the CNS is to:
   A. Detect stimuli in the environment
   B. Integrate and process information
   C. Carry signals to muscles and glands only
   D. Produce hormones for long-term control

4. The main function of the PNS is to:
   A. Store memories
   B. Coordinate reflexes in the spinal cord
   C. Connect receptors and effectors to the CNS
   D. Control the endocrine glands directly

5. Sensory (afferent) neurons in the PNS carry information:
   A. From the CNS to skeletal muscles
   B. From receptors to the CNS
   C. From the CNS to glands only
   D. Only within the brain

6. Motor (efferent) neurons in the PNS carry information:
   A. From receptors to the spinal cord
   B. From the brain to sensory receptors
   C. From the CNS to muscles and glands
   D. Only between different brain regions

7. Which division of the PNS controls voluntary movement of skeletal muscles?
   A. Autonomic nervous system
   B. Somatic nervous system
   C. Enteric nervous system
   D. Sympathetic nervous system

8. Which division of the PNS mainly controls internal organs such as the heart, smooth muscle, and glands?
   A. Somatic nervous system
   B. Autonomic nervous system
   C. Central nervous system
   D. Sensory nervous system

9. The sympathetic division of the autonomic nervous system is most active during:
   A. Deep sleep only
   B. “Rest and digest” activities
   C. “Fight or flight” responses
   D. Voluntary movement of limbs

10. The parasympathetic division of the autonomic nervous system is most active during:
   A. Intense exercise
   B. Acute stress
   C. “Rest and digest” activities, such as after a meal
   D. Strong voluntary muscle contraction

11. Which statement best describes a nerve?
   A. A bundle of cell bodies inside the CNS
   B. A bundle of axons in the PNS
   C. A single long axon in the CNS
   D. A cluster of dendrites in the PNS

12. Which statement best describes a tract?
   A. A bundle of axons in the CNS
   B. A bundle of axons in the PNS
   C. A cluster of neuron cell bodies in the PNS
   D. A cluster of glial cells in the CNS

13. A ganglion is:
   A. A bundle of dendrites in the brain
   B. A cluster of neuron cell bodies in the PNS
   C. A cluster of neuron cell bodies in the spinal cord
   D. A bundle of axons in the spinal cord

14. White matter in the CNS appears white mainly because it contains many:
   A. Neuronal cell bodies
   B. Unmyelinated dendrites
   C. Myelinated axons
   D. Blood vessels

15. A patient has a lesion in the spinal cord and loses the ability to feel touch and pain from the right leg, but motor function is partly preserved. Which system is MOST directly affected?
   A. CNS sensory pathways from the leg
   B. PNS motor neurons to the leg
   C. Autonomic fibers to blood vessels in the leg
   D. Enteric nervous system supplying the intestines

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2.8: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. The brain and spinal cord are protected by bone (skull and vertebrae). Why is this protection important for the central nervous system (CNS)? What might happen to body functions if this protection is damaged?
   Hint: Think about how important the brain and spinal cord are for controlling movement, sensation, and vital functions like breathing. What happens if they are injured?

2. Sensory (afferent) neurons bring information to the CNS, and motor (efferent) neurons carry commands away from the CNS. Explain why it is important to separate these two pathways. What problems could occur if signals were mixed or confused?
   Hint: Imagine a “two-way road” with cars going in both directions. What if there were no rules or lanes? Apply this idea to nerve signals going in and out of the CNS.

3. The sympathetic and parasympathetic systems often have opposite effects (for example, on heart rate or digestion). Why is it useful for the body to have this kind of “push–pull” control? Give one example and explain your reasoning.
   Hint: Think about times when you need to be very active (running, stress) versus times when you are resting and digesting food. Why do you need different settings for these situations?

4. Imagine a person steps on a sharp object and quickly withdraws the foot before feeling pain. Explain how this reflex uses both the peripheral nervous system (PNS) and the CNS. Why is this fast response important for survival?
   Hint: Follow the path: receptors in the skin → sensory neuron → spinal cord → motor neuron → muscle. Why is it faster to use a spinal reflex than to wait for full conscious awareness?

5. Damage to the spinal cord at different levels causes different problems (for example, paralysis of legs vs. whole body). Using your understanding of how the CNS is organized, explain why the level of injury changes the symptoms.
   Hint: Remember that nerves for the arms leave the spinal cord higher up, and nerves for the legs leave lower down. What body parts are controlled below the point of injury?

6. The autonomic nervous system controls internal organs without conscious control (for example, heart rate, breathing, digestion). Discuss one advantage and one possible disadvantage of this system working mostly without our awareness.
   Hint: Think of something good about not having to think about every heartbeat or breath, and something that could be a problem if the system makes changes without you noticing.

7. Suppose a drug affects only the peripheral nervous system but not the central nervous system. Predict one possible effect this drug might have on the body and explain which part of the PNS it could be acting on (somatic vs. autonomic).
   Hint: If it affects the somatic system, think about voluntary muscles and reflexes. If it affects the autonomic system, think about heart rate, blood vessels, glands, or digestion.

8. Neurons in the CNS do not regenerate easily after injury, while some peripheral nerves can recover. Why is this difference important for treatment and rehabilitation of patients with nervous system damage?
   Hint: Compare the chances of recovery after a brain/spinal cord injury with those after a peripheral nerve injury. How would this change what doctors and therapists focus on?

9. The brain has specialized regions (for example, those for movement, vision, speech). Why might it be more efficient for the CNS to have specialized areas instead of every part doing every job? What is one possible risk of this specialization?
   Hint: Think about a team where each person has a special role (doctor, nurse, technician). What is good about this? What happens if one key team member is missing?

10. The CNS must process a huge amount of information coming from the PNS at every moment. Explain one way the nervous system might “filter” or prioritize information. Why is this filtering important for normal daily activities?
    Hint: Consider why you do not feel your clothes all the time, or why you can focus on one conversation in a noisy room. How does ignoring some input help you function?

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2.9: Alternate Text Descriptions

Figure 2.1.1: Protective Layers Surrounding the Brain and Meninges

The figure shows a cross-sectional view of the protective layers surrounding the brain, progressing from the outside of the head inward.

1. The outermost layer is the **scalp**, which covers the top of the head.
2. Beneath the scalp is the **periosteum**, a connective tissue layer that covers the outer surface of the skull.
3. The **skull bone** lies below the periosteum and forms a rigid protective barrier around the brain.
4. Deep to the skull is the **dura mater**, the outermost meningeal layer. The dura mater lines the inner surface of the skull and forms a protective covering around the brain.
5. Between folds of the dura mater is the **superior sagittal sinus**, a large venous channel that drains blood from the brain.
6. Beneath the dura mater is the **subdural space**.
7. The **arachnoid mater** forms the middle meningeal layer and lies below the subdural space.
8. Between the arachnoid mater and the pia mater is the **subarachnoid space**, which contains cerebrospinal fluid and blood vessels that travel along the brain's surface.
9. The **pia mater** is the innermost meningeal layer and closely follows the contours of the brain, extending into its grooves and folds.
10. The **brain** lies beneath the pia mater.

An inset image of a skull indicates the location from which the cross-sectional view was taken.

The figure demonstrates how the brain is protected by multiple layers, including the scalp, skull, meninges, and cerebrospinal fluid-filled spaces. The meninges form three protective coverings around the brain: the dura mater, arachnoid mater, and pia mater.

Figure 2.1.2: Meninges, Cerebral Venous Drainage, and Arachnoid Granulations

The figure shows a cross-sectional view of the meninges, venous drainage pathways, and cerebrospinal fluid (CSF) reabsorption structures surrounding the brain.

1. The outermost layer is the **skull bone**, shown as a protective bony covering above the brain.
2. Beneath the skull is the **dura mater**, the outermost meningeal layer. Within the dura mater is a large venous channel labeled the **superior sagittal sinus**.
3. Several **cerebral veins** extend upward from the surface of the brain and drain into the superior sagittal sinus.
4. Below the dura mater is the **subdural space**.
5. The **arachnoid mater** forms the middle meningeal layer and lies beneath the subdural space.
6. The **subarachnoid space** lies between the arachnoid mater and the pia mater. This space contains **arachnoid trabeculae**, web-like connective tissue strands that span the space, and blood vessels traveling along the brain surface.
7. Small projections labeled **arachnoid granulations** extend through the dura mater into the superior sagittal sinus. These structures allow cerebrospinal fluid to be reabsorbed from the subarachnoid space into the venous circulation.
8. The **pia mater** is the innermost meningeal layer and closely follows the contours of the brain surface.
9. Beneath the pia mater lies the **cerebral cortex**, shown with folds and grooves characteristic of the brain surface.
10. A vertical groove between the cerebral hemispheres is labeled the **longitudinal fissure**.

The figure demonstrates the anatomical relationships among the meninges, cerebral veins, and the superior sagittal sinus. It also illustrates how cerebrospinal fluid in the subarachnoid space is returned to the bloodstream through arachnoid granulations while venous blood from the brain drains into the superior sagittal sinus.

Figure 2.1.3: Major Dural Venous Sinuses of the Brain

The figure shows a sagittal, or side, view of the head illustrating the major **dural venous sinuses**, which collect and drain venous blood from the brain.

1. The **superior sagittal sinus** runs along the upper midline of the cranial cavity within the dura mater.
2. Below it, the **inferior sagittal sinus** follows the lower edge of the **falx cerebri**, a dural fold that separates the cerebral hemispheres.
3. Posteriorly, the inferior sagittal sinus joins the **straight sinus**, which carries blood toward the back of the skull.
4. The straight sinus drains into the **transverse sinus**, which extends horizontally along the posterior cranial cavity.
5. The transverse sinus continues into the **sigmoid sinus**, which curves downward toward the base of the skull.
6. The sigmoid sinus empties into the **internal jugular vein**, which carries blood away from the brain and exits the skull through the neck.
7. Near the base of the brain, the **cavernous sinus** receives venous blood and connects with surrounding venous channels.
8. Additional pathways include the **sphenoparietal sinus**, **superior petrosal sinus**, and **inferior petrosal sinus**, which connect the cavernous sinus with other dural venous sinuses.
9. The **tentorium cerebelli**, a dural fold separating the cerebrum from the cerebellum, is shown beneath the cerebral hemispheres.
10. The **falx cerebri** is shown extending vertically between the hemispheres and contains the superior and inferior sagittal sinuses along its borders.

The figure demonstrates the network of dural venous sinuses that collect blood from the brain and direct it toward the internal jugular vein for return to the systemic circulation.

Figure 2.1.4: Circle of Willis and Major Cerebral Arteries

The figure shows the **Circle of Willis**, a ring-like network of arteries located at the base of the brain, viewed from below.

1. Two **vertebral arteries** ascend along the brainstem and join to form the **basilar artery**.
2. The **basilar artery** continues forward and divides into the left and right **posterior cerebral arteries**, which supply the posterior regions of the brain.
3. On each side, an **internal carotid artery** enters the cranial cavity and contributes to the arterial network.
4. Each internal carotid artery gives rise to an **anterior cerebral artery**, which extends toward the midline, and a **middle cerebral artery**, which extends laterally along the side of the brain.
5. The left and right anterior cerebral arteries are connected by a short vessel called the **anterior communicating artery**.
6. Each **posterior cerebral artery** is connected to the corresponding internal carotid artery by a **posterior communicating artery**.
7. Together, the anterior cerebral arteries, anterior communicating artery, posterior communicating arteries, and posterior cerebral arteries form the circular arterial arrangement known as the **Circle of Willis**.
8. An **ophthalmic artery** branches from each internal carotid artery and extends toward the eye region.

The figure demonstrates how the Circle of Willis creates interconnected pathways for blood flow between the internal carotid and vertebrobasilar systems. This arrangement provides collateral circulation, allowing blood to be redistributed to brain tissue if one arterial pathway becomes narrowed or blocked.

Figure 2.1.5: Dural Venous Sinuses and Venous Drainage of the Brain

The figure shows a sagittal, or side, cross-sectional view of the head illustrating the major dural venous sinuses and the pathways by which venous blood drains from the brain.

1. The **cranium** forms the outer bony covering of the head. Immediately beneath it is the **dura mater**, the outermost meningeal layer.
2. **Cerebral veins** on the surface of the brain collect venous blood from brain tissue and drain into larger venous sinuses.
3. The **superior sagittal sinus** runs along the upper midline of the cranial cavity. Arrows indicate blood flowing posteriorly through this sinus toward the back of the head.
4. The **inferior sagittal sinus** runs along the lower edge of a dural fold within the cranial cavity and also directs blood posteriorly.

5. Deep within the brain, the **great cerebral vein** drains blood from internal brain structures and joins the inferior sagittal sinus.
6. The great cerebral vein and inferior sagittal sinus unite to form the **straight sinus**, which carries blood toward the posterior cranial cavity.
7. At the back of the head, the straight sinus drains into the **confluence of sinuses**, a meeting point for several major venous channels.
8. The **transverse sinus** extends laterally from the confluence of sinuses and carries blood toward the side of the skull.
9. An **occipital sinus** is shown draining into the confluence from the posterior region.
10. Blood continues from the transverse sinus into the **sigmoid sinus**, which curves downward toward the base of the skull.
11. The sigmoid sinus exits the cranial cavity and becomes the **internal jugular vein**, which returns blood from the brain to the systemic circulation.
12. Arrows throughout the venous channels indicate the direction of blood flow from cerebral veins through the dural venous sinuses and ultimately into the internal jugular vein.

The figure demonstrates the major pathways of cerebral venous drainage. Venous blood collected by cerebral veins enters the dural venous sinuses, flows through interconnected channels including the superior sagittal, straight, transverse, and sigmoid sinuses, and ultimately leaves the skull through the internal jugular vein.

Figure 2.1.6: Ventricular System of the Brain

The figure shows the ventricular system of the brain in two views: a **lateral, or side, view** on the left and an **anterior, or front, view** on the right. The ventricles are highlighted to illustrate the interconnected spaces that contain and circulate cerebrospinal fluid, or **CSF**.

1. The **lateral ventricles** are the largest ventricles and are located within the cerebral hemispheres. They appear as paired cavities extending through the upper regions of the brain.
2. Each lateral ventricle connects to the **third ventricle** through an opening called the **interventricular foramen**.
3. The **third ventricle** is a narrow cavity located along the midline of the brain between the two halves of the diencephalon.
4. Extending downward from the third ventricle is the **cerebral aqueduct**, a narrow channel that connects the third ventricle to the fourth ventricle.
5. The **fourth ventricle** is located between the brainstem and the cerebellum in the lower portion of the brain.
6. Inferior to the fourth ventricle, the ventricular pathway continues into the **central canal**, which extends through the spinal cord.
7. The lateral view emphasizes the position and continuity of the ventricles from the cerebral hemispheres through the brainstem and into the spinal cord.
8. The anterior view shows the symmetrical arrangement of the paired lateral ventricles and their connections to the midline ventricular structures.

The figure demonstrates the organization of the ventricular system and the pathway through which cerebrospinal fluid flows from the lateral ventricles to the third ventricle, through the cerebral aqueduct to the fourth ventricle, and then into the central canal of the spinal cord.

Figure 2.1.7: Cerebrospinal Fluid Production, Circulation, and Reabsorption

The figure illustrates the production, circulation, and reabsorption of cerebrospinal fluid, or **CSF**, within the ventricular system and surrounding spaces of the brain and spinal cord.

1. CSF is produced by the **choroid plexus** located within the **right lateral ventricle**.
2. From the lateral ventricle, CSF flows through the **interventricular foramen** into the **third ventricle**, located near the midline of the brain.
3. CSF then travels through the narrow **cerebral aqueduct**, which connects the third ventricle to the **fourth ventricle**.
4. From the fourth ventricle, CSF exits the ventricular system through the **median aperture** and **lateral apertures**.
5. After leaving the fourth ventricle, CSF enters the **subarachnoid space**, where it circulates around the brain and spinal cord.
6. A portion of the CSF also continues into the **central canal** of the spinal cord.
7. Arrows indicate CSF flowing through the subarachnoid space around the surface of the brain.
8. The **arachnoid granulations** project into the **superior sagittal sinus**, a large venous channel within the dura mater.

9. CSF is reabsorbed through the arachnoid granulations into the venous blood of the superior sagittal sinus.
10. The diagram also labels the **meningeal dura mater**, **subarachnoid space**, and ventricular structures to show their relationship to the CSF circulation pathway.

The figure demonstrates the continuous circulation of cerebrospinal fluid: production by the choroid plexus, movement through the ventricles and subarachnoid space, and reabsorption into the venous system through arachnoid granulations.

Figure 2.2.1: Primary Motor and Somatosensory Cortices of the Cerebrum

The figure shows a lateral view of the brain highlighting the relationship between the primary motor cortex and the primary somatosensory cortex.

1. A prominent groove called the **central sulcus** extends across the lateral surface of the cerebrum and separates two adjacent functional regions of the cerebral cortex.
2. The **primary motor cortex** is located immediately anterior to the central sulcus within the **precentral gyrus**. This region initiates and controls voluntary skeletal muscle movements.
3. The **primary somatosensory cortex** is located immediately posterior to the central sulcus within the **postcentral gyrus**. This region receives and processes sensory information from the body, including touch, pressure, temperature, pain, and proprioception.
4. The **cerebellum** is located posterior and inferior to the cerebrum. It coordinates balance, posture, and the precision of voluntary movements.
5. The **brainstem** extends inferiorly from the base of the brain and connects the brain to the spinal cord. It regulates essential functions such as breathing, heart rate, and reflex activity.

The figure demonstrates the close anatomical relationship between the brain regions responsible for voluntary motor control and sensory perception. The central sulcus serves as a landmark separating the primary motor cortex, which generates movement commands, from the primary somatosensory cortex, which receives sensory information from the body.

Figure 2.2.2: Superior View of the Cerebral Hemispheres and Cortical Landmarks

The figure shows a superior, or top-down, view of the cerebrum, highlighting the two cerebral hemispheres and major surface features of the cerebral cortex.

1. The **left hemisphere** and **right hemisphere** are separated by a deep groove called the **longitudinal fissure**, which extends along the midline from the anterior to the posterior region of the brain.
2. The surface of each hemisphere contains numerous raised ridges called **gyri** and shallow grooves called **sulci**. These folds increase the surface area of the cerebral cortex.
3. In the left hemisphere, labels identify examples of **gyri** and **sulci**, emphasizing the folded organization of the cerebral cortex.
4. In the right hemisphere, the **central sulcus** is labeled. This prominent groove separates the frontal lobe from the parietal lobe.
5. The **precentral gyrus** is located immediately anterior to the central sulcus and contains the primary motor cortex, which controls voluntary skeletal muscle movements.
6. The **postcentral gyrus** is located immediately posterior to the central sulcus and contains the primary somatosensory cortex, which receives and processes sensory information from the body.

The figure demonstrates the division of the cerebrum into left and right hemispheres and highlights major cortical landmarks, including gyri, sulci, the central sulcus, and the adjacent motor and sensory regions of the cerebral cortex.

Figure 2.2.3: Coronal Section of the Cerebrum and Associated Brain Structures

The figure shows a **coronal, or front-facing, section** of the cerebrum and adjacent brain structures, revealing both the outer cortex and deeper internal regions of the brain.

1. The cerebrum is divided into **right and left cerebral hemispheres**, which are visible on either side of the midline.
2. The outer layer of each hemisphere is the **cerebral cortex**, which forms a continuous covering around the brain surface and contains numerous folds.
3. The surface of the cerebral cortex contains raised ridges called **gyri** and grooves called **sulci**. These folds increase the surface area available for neural processing.
4. Beneath the cerebral cortex are **tracts**, which are bundles of nerve fibers that connect different regions of the brain and carry information between them.
5. At the center of the brain, the **corpus callosum** forms a broad band of nerve fibers connecting the left and right hemispheres, allowing communication between the two sides of the cerebrum.
6. Deep within each hemisphere are the **basal nuclei**, groups of gray matter involved in the regulation and coordination of movement.
7. The lower portion of the image shows the **cerebellum**, located inferior and posterior to the cerebrum. The cerebellum contributes to balance, posture, and coordination of movement.
8. Extending downward from the cerebrum is the **brainstem**, which connects the brain to the spinal cord and contains centers that regulate essential functions such as breathing, heart rate, and reflexes.

The figure demonstrates the organization of the cerebrum in a coronal section, highlighting the relationship between the cerebral cortex, underlying white matter tracts, corpus callosum, basal nuclei, cerebellum, and brainstem.

Figure 2.2.4: Major Lobes and Functional Landmarks of the Cerebrum

The figure shows a lateral view of the cerebrum with its major lobes and anatomical landmarks labeled.

1. The **frontal lobe** occupies the anterior portion of the cerebrum. It is associated with voluntary movement, planning, decision-making, and other higher cognitive functions.
2. Near the posterior border of the frontal lobe is the **precentral gyrus**, which contains the primary motor cortex responsible for initiating voluntary skeletal muscle movements.
3. The **central sulcus** is a prominent groove that separates the frontal lobe from the parietal lobe and marks the boundary between primary motor and primary somatosensory regions.
4. Immediately posterior to the central sulcus is the **postcentral gyrus**, which contains the primary somatosensory cortex. This region receives and processes sensory information from the body, including touch, pressure, temperature, pain, and proprioception.
5. The **parietal lobe** lies posterior to the central sulcus and is involved in integrating sensory information and supporting spatial awareness.
6. The **parieto-occipital sulcus** marks the boundary between the parietal and occipital lobes.
7. The **occipital lobe** forms the posterior portion of the cerebrum and is primarily responsible for visual processing.
8. The **lateral sulcus** separates the temporal lobe from the frontal and parietal lobes.
9. The **temporal lobe**, located inferior to the lateral sulcus, is involved in auditory processing, language comprehension, memory, and other cognitive functions.

The figure demonstrates how major landmarks, including the central sulcus and lateral sulcus, divide the cerebrum into functional lobes. It also highlights the close relationship between the primary motor cortex in the precentral gyrus and the primary somatosensory cortex in the postcentral gyrus.

Figure 2.2.5: Coronal Section Showing the Basal Nuclei and Other Deep Cerebral Structures

The figure shows a **frontal, or coronal, section** of the cerebrum highlighting deep brain structures involved in communication between the hemispheres, motor regulation, and emotional processing.

1. The **left and right cerebral hemispheres** are visible on either side of the midline.
2. Near the center of the brain are the paired **lateral ventricles**, fluid-filled spaces that contain cerebrospinal fluid.
3. Above the lateral ventricles is the **corpus callosum**, a large bundle of nerve fibers that connects the two cerebral hemispheres and enables communication between them.
4. Deep within each hemisphere is the **striatum**, which consists of the **caudate nucleus** and **putamen**. These structures are components of the basal nuclei and participate in planning and regulating movement.

5. Adjacent to the putamen is the **globus pallidus**, another component of the basal nuclei that works with the striatum to regulate voluntary motor activity.
6. The **insula** is located deep within the lateral portion of each hemisphere beneath the cerebral cortex. It is involved in sensory integration, emotion, and awareness of internal body states.
7. The **amygdala** is located in the inferior portion of each hemisphere and is associated with emotional processing, learning, and memory.
8. An inset diagram indicates the location of the coronal section within the brain.

The figure demonstrates the spatial relationship between the ventricles, corpus callosum, basal nuclei, insula, and amygdala. These internal structures contribute to communication between cerebral hemispheres, regulation of movement, processing of emotions, and integration of sensory information.

Figure 2.2.6: Association, Commissural, and Projection Fiber Pathways in the Cerebrum

The figure shows two views of the cerebrum illustrating the major categories of white matter fibers that connect different regions of the nervous system.

1. The left panel presents a lateral view of a cerebral hemisphere and highlights **association fibers**, which connect regions within the same hemisphere. Some association fibers connect nearby cortical areas, while others extend between more distant areas of the same hemisphere.
2. The right panel shows a frontal, or coronal, section of the cerebrum and highlights two additional categories of white matter fibers.
3. **Commissural fibers** cross the midline and connect corresponding regions of the left and right cerebral hemispheres. These fibers allow information to be exchanged between the two sides of the brain.
4. **Projection fibers** extend vertically between the cerebral cortex and lower regions of the central nervous system. These pathways carry sensory information toward the cerebrum and motor commands from the cerebrum to the brainstem and spinal cord.
5. The frontal section demonstrates how commissural fibers travel horizontally between hemispheres, while projection fibers travel between the cerebrum and deeper neural structures.

The figure demonstrates the three major classes of cerebral white matter connections: **association fibers**, which connect regions within a hemisphere; **commissural fibers**, which connect the two hemispheres; and **projection fibers**, which connect the cerebrum with lower parts of the brain and spinal cord. Together, these pathways enable communication within a hemisphere, between hemispheres, and between the brain and the rest of the body.

Figure 2.2.7: Brodmann Areas and Functional Regions of the Cerebral Cortex

Figure shows **Brodmann's cytoarchitectonic map (1909)** on the cerebral cortex in two views: a **lateral surface view** and a **medial surface view**. The map divides the cerebral cortex into numbered regions based on differences in cellular organization, with several functional areas highlighted.

Lateral Surface View

1. **Area 4 (Primary motor cortex)** is located in the precentral gyrus and is responsible for initiating voluntary skeletal muscle movements.
2. **Areas 1, 2, and 3 (Primary somatosensory cortex)** are located immediately posterior to the primary motor cortex and receive sensory information related to touch, pressure, pain, temperature, and body position.
3. **Areas 44 and 45 (Broca's area)** are located in the inferior frontal region and are associated with speech production and language expression.
4. **Area 22 (Primary auditory cortex)** is located in the temporal lobe and is responsible for processing auditory information.
5. **Areas 39 and 40 (Wernicke's area)** are located in the posterior language-processing region and are involved in language comprehension.

Medial Surface View

1. **Area 4 (Primary motor cortex)** extends onto the medial surface of the cerebral hemisphere.
2. **Areas 1, 2, and 3 (Primary somatosensory cortex)** are also visible on the medial surface adjacent to the motor cortex.
3. **Area 17 (Primary visual cortex)** is located in the posterior portion of the occipital lobe and receives and processes visual information.

The figure demonstrates how specific regions of the cerebral cortex are specialized for different functions. Brodmann's map identifies cortical areas associated with voluntary movement, sensory processing, language production, language comprehension, hearing, and vision. The lateral and medial views illustrate the distribution of these functional regions across the surface of the cerebral hemispheres.

Figure 2.2.8: Functional Areas of the Cerebral Cortex

The figure is a lateral view of the brain identifying functional regions involved in motor control, sensory processing, language, vision, hearing, and higher-level integration. The diagram groups regions into motor areas, prefrontal cortex, and sensory and association areas.

1. The **primary motor cortex** initiates voluntary skeletal muscle movement.
2. The **motor association area** plans and coordinates complex movements.
3. The **frontal eye field** controls voluntary eye movements.
4. Within the **prefrontal cortex**, **Broca's area** is involved in speech production and language expression.
5. The **primary somatosensory cortex** receives and processes sensory input such as touch, pressure, temperature, and body position.
6. The **sensory association area** interprets and integrates sensory information.
7. **Wernicke's area** is responsible for understanding spoken and written language.
8. The **general interpretation area** integrates information from multiple sensory modalities to support complex functions such as comprehension and reasoning.
9. The **primary visual cortex** processes incoming visual information.
10. The **visual association area** interprets and gives meaning to visual input.
11. The **primary auditory cortex** processes sound.
12. The **auditory association area** interprets auditory information.

The diagram shows how specialized regions of the cortex work together to support movement, sensation, communication, and cognition.

Labels included: Motor areas; Primary motor cortex; Motor association area; Frontal eye field; Prefrontal cortex; Broca's area; Sensory areas and related association areas; Primary somatosensory cortex; Sensory association area; Wernicke's area; General interpretation area; Primary visual cortex; Visual association area; Primary auditory cortex; Auditory association area.

Figure 2.2.9: Somatosensory Homunculus of the Primary Somatosensory Cortex

The figure illustrates the **somatosensory homunculus**, a representation of how different body regions are mapped onto the primary somatosensory cortex. The body parts are arranged along a strip of cortex according to the amount of sensory input they provide rather than their actual physical size. Regions with greater sensory sensitivity occupy larger areas of the cortex and are therefore depicted as exaggerated body parts.

1. At the superior and medial portion of the cortical map are representations of the **hip**, **leg**, **foot**, **toes**, and **genitals**.
2. Moving along the cortical surface, the map continues through the **trunk**, **neck**, **head**, **shoulder**, **arm**, **elbow**, **forearm**, and **wrist**.
3. A disproportionately large **hand** occupies a substantial portion of the map, reflecting the high density of sensory receptors in the hand.
4. Individual representations of the **little finger**, **ring finger**, **middle finger**, **index finger**, and **thumb** are shown, emphasizing the detailed sensory processing devoted to the fingers.
5. Inferior to the hand region are representations of the **eye**, **nose**, **face**, **lips**, and **teeth, gums, and jaw**.

6. The largest facial representations are the **lips** and **tongue**, indicating extensive cortical areas devoted to sensory information from these structures.
7. The most inferior portions of the map represent the **tongue** and **pharynx**.
8. The cortical map is illustrated as a distorted human figure wrapped along the contour of the somatosensory cortex. Body regions with greater sensory acuity, such as the hands, lips, and tongue, appear much larger than regions such as the trunk and legs.

The figure demonstrates that the primary somatosensory cortex allocates different amounts of cortical area to different body regions. Areas requiring fine sensory discrimination, including the hands, lips, and tongue, are represented by disproportionately large cortical regions, whereas less sensitive body regions occupy smaller areas.

Figure 2.2.10: Broca's Area, Wernicke's Area, and the Arcuate Fasciculus

The figure shows a lateral view of the cerebral hemisphere highlighting two major language-related regions and the neural pathway that connects them.

1. **Broca's area** is located in the frontal lobe, toward the anterior portion of the brain. This region is involved in the planning and production of speech and language expression.
2. **Wernicke's area** is located in the posterior region of the cerebrum near the temporal and parietal lobes. This region is involved in the comprehension and interpretation of spoken and written language.
3. A bundle of curved nerve fibers extends between Broca's area and Wernicke's area. These fibers represent the **arcuate fasciculus**, a white matter tract that connects the two language regions.
4. The arcuate fasciculus allows information related to language comprehension to be transmitted between Wernicke's area and Broca's area, supporting the coordination of language understanding and speech production.

The figure demonstrates the functional relationship between two major language centers of the brain. Wernicke's area processes and interprets language, Broca's area contributes to language production, and the arcuate fasciculus provides the communication pathway that links these regions.

Figure 2.3.1: Sagittal Section of the Brain Showing Major Internal Structures

The figure shows a **sagittal, or side, section** of the brain, revealing major internal structures along the midline and their anatomical relationships from the anterior to posterior regions of the brain.

1. The **corpus callosum** arches through the upper central portion of the brain and forms a major pathway connecting the left and right cerebral hemispheres.
2. Beneath the corpus callosum is the **diencephalon**, which consists of three major regions:
   - The **thalamus**, positioned centrally, serves as a relay center for sensory information traveling to the cerebral cortex.
   - The **epithalamus**, located superior and posterior to the thalamus.
   - The **hypothalamus**, located inferior to the thalamus and involved in regulating homeostasis and autonomic functions.
3. Posterior to the diencephalon is the **tectum**, a region of the midbrain composed of:
   - The **superior colliculus**, associated with visual reflexes and orientation to visual stimuli.
   - The **inferior colliculus**, associated with auditory reflexes and processing of sound-related information.
4. Inferior to the diencephalon and tectum is the **brainstem**, which connects the brain with the spinal cord. The brainstem is divided into:
   - The **midbrain** at its superior end.
   - The **pons**, located below the midbrain.
   - The **medulla oblongata**, the most inferior portion of the brainstem.
5. Posterior to the brainstem is the **cerebellum**, shown with its highly folded surface. The cerebellum coordinates balance, posture, and the precision of voluntary movements.
6. Inferior to the medulla oblongata, the **spinal cord** continues downward from the brainstem and serves as the major pathway for communication between the brain and the rest of the body.
7. The figure labels the anterior and posterior orientation of the brain and identifies the image as a sagittal section through the midline.

The figure demonstrates the organization and spatial relationships of major brain regions, including the diencephalon, midbrain tectum, brainstem, cerebellum, and spinal cord. It highlights how sensory relay centers, autonomic control centers, reflex centers, and motor coordination regions are arranged around the central pathway connecting the brain to the spinal cord.

Figure 2.3.2: Inferior View of the Brainstem and Ventral Brain Structures

The figure shows an **inferior, or bottom, view** of the brain highlighting structures visible on the ventral surface and the continuation of the brain into the spinal cord.

1. Near the anterior portion of the brain is the **pituitary gland**, a small endocrine structure connected to the hypothalamus that regulates hormone secretion throughout the body.
2. Posterior to the pituitary gland are the paired **mammillary bodies**, structures associated with memory processing and components of the limbic system.
3. On either side of the midline, the **cerebral peduncles** extend through the ventral surface of the midbrain. These fiber tracts carry motor information between the cerebrum and lower regions of the nervous system.
4. The **brainstem** is visible along the midline and is composed of three regions:
    - The **midbrain**, located superiorly.
    - The **pons**, located below the midbrain and appearing as a prominent bulge.
    - The **medulla oblongata**, located inferior to the pons and continuous with the spinal cord.
5. On the ventral surface of the medulla oblongata are the **pyramids**, which contain descending motor pathways involved in voluntary movement.
6. Near the lower end of the medulla is the **decussation of the pyramids**, where many motor fibers cross from one side of the nervous system to the other.
7. Inferior to the medulla oblongata, the **spinal cord** extends downward and serves as the major communication pathway between the brain and the rest of the body.
8. The figure includes orientation labels indicating the **anterior** and **posterior** directions and identifies the view as an **inferior view** of the brain.

The figure demonstrates the anatomical organization of the ventral brainstem and related structures, highlighting pathways that connect the cerebrum with the spinal cord, the location of endocrine and memory-related structures, and the crossing of motor fibers within the medulla oblongata.

Figure 2.3.3: Cerebellar Peduncles Connecting the Cerebellum and Brainstem

The figure shows the cerebellum and adjacent brainstem structures, highlighting the major fiber pathways that connect the cerebellum to the rest of the central nervous system.

1. The **pons** is located anterior to the cerebellum and forms a prominent part of the brainstem. It serves as a relay center that transmits information between the cerebrum and the cerebellum.
2. Inferior to the pons is the **olive**, a structure on the medulla oblongata associated with motor learning and coordination.
3. The cerebellum is connected to the brainstem by three pairs of fiber bundles called **cerebellar peduncles**.
4. The **superior cerebellar peduncle** extends from the upper portion of the cerebellum toward the midbrain. This pathway primarily carries output signals from the cerebellum to other regions of the brain involved in motor control.
5. The **middle cerebellar peduncle** is the largest of the three peduncles and connects the cerebellum to the pons. This pathway carries information from the cerebral cortex to the cerebellum.
6. The **inferior cerebellar peduncle** connects the cerebellum with the medulla oblongata and spinal cord. This pathway carries sensory and balance-related information into the cerebellum.
7. The cerebellum is shown with its highly folded internal structure, illustrating its large surface area for processing motor and sensory information.

The figure demonstrates how the cerebellum communicates with the brainstem through the superior, middle, and inferior cerebellar peduncles. These pathways enable the cerebellum to receive sensory and motor information, coordinate movement, maintain balance and posture, and contribute to motor learning.

Figure 2.3.4: Arbor Vitae and Brainstem Connections of the Cerebellum

The figure shows a section of the **cerebellum** and adjacent **brainstem** structures, highlighting the internal organization of cerebellar white matter.

1. The **cerebellum** occupies the right side of the figure and is shown in section, revealing its highly folded outer cortex and branching internal white matter.
2. Within the cerebellum, the **deep cerebellar white matter**, or **arbor vitae**, forms a distinctive tree-like pattern that extends from the center into the folds of the cerebellum. The arbor vitae contains nerve fibers that carry information into and out of the cerebellar cortex.
3. Anterior to the cerebellum is the **pons**, a part of the brainstem that serves as a major relay center between the cerebrum, cerebellum, and other regions of the central nervous system.
4. Inferior to the pons is the **inferior olive**, or **inferior olivary nucleus**, of the medulla oblongata. This structure contributes to motor learning and coordination by providing input to the cerebellum.
5. The cerebellum is connected to the brainstem through fiber pathways that carry sensory and motor information used to coordinate balance, posture, and voluntary movement.

The figure demonstrates the relationship between the cerebellum and brainstem and highlights the branching arrangement of the **arbor vitae**, the internal white matter network that distributes information throughout the cerebellum.

Figure 2.3.5: Major Divisions of the Cerebellum: Vermis, Hemispheres, and Flocculonodular Lobe

The figure compares two views of the cerebellum to illustrate its major anatomical divisions: a **midsagittal section** on the left and a **superior view of an "unrolled" cerebellum** on the right.

Left Panel: Midsagittal Section of the Cerebellum

1. A midsagittal section reveals the internal structure of the cerebellum, including its highly folded cortex and branching white matter.
2. The central portion of the cerebellum is the **vermis**, shown in purple. The vermis lies along the midline and connects the two cerebellar hemispheres.
3. On either side of the vermis are the **cerebellar hemispheres**, shown in green. These regions make up the majority of the cerebellum.
4. A small region on the inferior surface, shown in orange, represents the **flocculonodular lobe**.
5. The folded internal pattern of the cerebellum reflects its extensive cortical surface area for processing sensory and motor information.

Right Panel: Superior View of an "Unrolled" Cerebellum

1. The cerebellum is shown conceptually unfolded to emphasize its major subdivisions.
2. The central midline region is labeled the **vermis**.
3. The large paired regions on either side are labeled the **hemispheres**.
4. The inferior orange region represents the **flocculonodular lobe**, which consists of:
    - The **nodulus**, located at the midline.
    - The paired **flocculi**, located laterally on each side.
5. The diagram demonstrates how the vermis occupies the center of the cerebellum, the hemispheres extend laterally, and the flocculonodular lobe forms a distinct inferior subdivision.

The figure illustrates the major anatomical divisions of the cerebellum: the **vermis**, **cerebellar hemispheres**, and **flocculonodular lobe**. The sagittal view shows their spatial relationships within the cerebellum, while the unrolled view emphasizes how these regions are organized across the cerebellar surface. The vermis is associated with posture and trunk control, the hemispheres contribute to coordination of voluntary movements, and the flocculonodular lobe is involved in balance and eye movements.

Figure 2.3.6: Major Components of the Limbic System

The figure shows a midsagittal view of the brain highlighting major components of the **limbic system**, a network of structures involved in emotion, memory, motivation, and regulation of internal body functions.

1. A large ring-shaped region outlined around the central part of the brain identifies the major structures of the limbic system.
2. The **cingulate gyrus** arches above the corpus callosum along the medial surface of the cerebrum. It is associated with emotional processing, motivation, learning, and attention.
3. The **thalamus** is located near the center of the brain. It serves as a major relay center that processes and distributes sensory and other information to the cerebral cortex.
4. Inferior to the thalamus is the **hypothalamus**, which regulates homeostasis and autonomic functions, including body temperature, hunger, thirst, endocrine activity, and emotional responses.
5. The **pituitary gland** projects inferiorly from the hypothalamus and functions as a major endocrine gland that controls hormone release throughout the body.
6. The **hippocampus** is located within the medial temporal lobe. It plays a critical role in the formation and consolidation of new memories and supports spatial learning.
7. The **amygdala** is positioned near the anterior end of the hippocampus. It is involved in emotional processing, particularly responses related to fear, motivation, and emotional memory.
8. The structures are shown in close anatomical relationship, demonstrating how regions involved in memory, emotion, hormonal regulation, and autonomic control are interconnected.

The figure demonstrates the organization of key limbic system components and their relationships to nearby diencephalic structures. Together, the cingulate gyrus, hippocampus, amygdala, hypothalamus, thalamus, and pituitary gland contribute to emotional processing, memory formation, motivation, behavior, and maintenance of homeostasis.

Figure 2.4.1: Connective Tissue Organization of a Spinal Nerve

The figure compares a schematic illustration and a histological cross-section of a spinal nerve, showing the connective tissue layers that organize and protect peripheral nerve fibers.

Panel (a): Anatomical Diagram of a Spinal Nerve

1. A spinal nerve is shown in longitudinal view, enclosed by an outer connective tissue layer called the **epineurium**.
2. Within the spinal nerve are multiple bundles of nerve fibers called **fascicles**, which run parallel to one another along the length of the nerve.
3. **Blood vessels** travel within the connective tissue between the fascicles, supplying nutrients and oxygen to the nerve.
4. An enlarged view of a fascicle shows the **perineurium**, a connective tissue sheath that surrounds and protects the entire fascicle.
5. Within the fascicle, individual nerve fibers are surrounded by **endoneurium**, a delicate connective tissue layer that supports individual axons.
6. A second enlarged cross-sectional view shows multiple fascicles within the spinal nerve. Each fascicle contains numerous **axons** grouped together and enclosed by perineurium.

Panel (b): Histological Cross-Section

1. A microscope image shows a cross-section of a peripheral nerve stained pink.
2. Several **fascicles** appear as densely packed circular or oval regions within the nerve.
3. Each fascicle is surrounded by a distinct **perineurium**.
4. The outer connective tissue surrounding all fascicles is labeled **epineurium**.
5. The image demonstrates how multiple fascicles are bundled together within a single nerve and protected by layers of connective tissue.

The figure demonstrates the hierarchical organization of a peripheral nerve. Individual **axons** are surrounded by **endoneurium**, groups of axons form **fascicles** enclosed by **perineurium**, and multiple fascicles together form a **spinal nerve** surrounded by **epineurium**. Blood vessels travel within the nerve to support the metabolic needs of the nerve fibers.

Figure 2.4.2: Histological Cross-Section of a Peripheral Nerve Showing Perineurium and Endoneurium

The figure shows a high-magnification histological cross-section of a peripheral nerve, highlighting connective tissue layers that organize and support nerve fibers.

1. A thick band of connective tissue labeled **perineurium** runs through the center of the image. The perineurium forms the protective sheath surrounding a nerve fascicle and separates adjacent bundles of nerve fibers.
2. On the right side of the image, a large **fascicle** contains numerous tightly packed circular and oval profiles representing cross-sections of individual nerve fibers, or axons.
3. The connective tissue surrounding and supporting individual nerve fibers within the fascicle is labeled **endoneurium**.
4. Additional portions of neighboring fascicles are visible on the left side of the image, separated from one another by connective tissue layers.
5. Throughout the tissue, connective tissue appears pink, while cell nuclei appear as small, dark-stained elongated structures scattered within the tissue.
6. An inset image in the upper-right corner shows the entire peripheral nerve cross-section at lower magnification, with a highlighted box indicating the region displayed in the enlarged view.

The figure demonstrates the organization of connective tissue within a peripheral nerve. The **endoneurium** surrounds individual nerve fibers, while the **perineurium** encloses groups of fibers into fascicles, providing structural support and protection for nerve tissue.

Figure 2.4.3: Inferior View of the Brain Showing the Cranial Nerves (CN I-XII)

The figure shows an **inferior, or ventral, view of the brain** with the **cranial nerves** highlighted as they emerge from the brain and brainstem. The nerves are labeled using both their names and Roman numerals.

1. The **olfactory nerve (CN I)** extends from the anterior portion of the brain and carries information related to smell.
2. The **optic nerve (CN II)** emerges near the base of the forebrain and carries visual information from the eyes to the brain.
3. The **oculomotor nerve (CN III)** arises from the midbrain and controls most eye movements as well as pupil constriction.
4. The **trochlear nerve (CN IV)** emerges from the midbrain and innervates a muscle that moves the eye.
5. The **trigeminal nerve (CN V)** emerges laterally from the pons and is the largest cranial nerve. It carries sensory information from the face and provides motor control to muscles involved in chewing.
6. The **abducens nerve (CN VI)** arises near the junction of the pons and medulla and controls lateral movement of the eye.
7. The **facial nerve (CN VII)** emerges from the brainstem lateral to the abducens nerve and carries motor, sensory, and parasympathetic functions, including facial expression.
8. The **vestibulocochlear nerve (CN VIII)** emerges near the facial nerve and carries information related to hearing and balance.
9. The **glossopharyngeal nerve (CN IX)** arises from the medulla oblongata and contributes to taste, swallowing, and autonomic functions.
10. The **vagus nerve (CN X)** emerges from the medulla and extends inferiorly to provide sensory and parasympathetic innervation to many thoracic and abdominal organs.
11. The **accessory nerve (CN XI)** arises from the medulla and upper spinal cord and supplies muscles of the neck and shoulders.
12. The **hypoglossal nerve (CN XII)** emerges from the medulla oblongata and controls tongue movements.

The figure demonstrates the locations where the twelve cranial nerves emerge from the brain and brainstem. These nerves provide sensory, motor, and autonomic connections between the brain and structures of the head, neck, and other regions of the body.

Figure 2.5.1: Posterior View of the Spinal Cord Showing the Conus Medullaris, Cauda Equina, and Filum Terminale

The figure shows a **posterior view of the spinal cord** within the vertebral column, highlighting its major regions and specialized

structures at its inferior end.

1. The spinal cord extends vertically through the vertebral canal and is divided into four major regions:
   - The **cervical region** in the neck.
   - The **thoracic region** in the upper and middle back.
   - The **lumbar region** in the lower back.
   - The **sacral region** near the pelvis.
2. The spinal cord gradually narrows inferiorly into a cone-shaped structure called the **conus medullaris**, which marks the terminal end of the spinal cord.
3. Inferior to the conus medullaris, numerous spinal nerve roots continue downward within the vertebral canal. These nerve roots form a bundle called the **cauda equina**, named for its resemblance to a horse's tail.
4. An enlarged inset provides a closer view of the lower spinal canal. Within the cauda equina, a thin fibrous strand called the **filum terminale** extends downward from the conus medullaris.
5. The **filum terminale** helps anchor the spinal cord within the vertebral canal while the surrounding spinal nerve roots continue toward their points of exit from the spine.

The figure demonstrates the regional organization of the spinal cord and the anatomical structures found at its inferior end. It highlights the transition from the spinal cord proper to the **conus medullaris**, the collection of descending nerve roots known as the **cauda equina**, and the anchoring **filum terminale**.

Figure 2.5.2: Transverse Section of the Vertebral Canal Showing the Spinal Cord, Spinal Nerves, and Meninges

The figure shows a **superior, or transverse, view** of a vertebra and spinal canal with the spinal cord in cross-section. The image illustrates the relationship between the vertebra, spinal cord, spinal nerve roots, spinal nerves, and the meningeal coverings surrounding the spinal cord.

Vertebral Structures

1. The vertebra surrounds and protects the spinal cord within the vertebral canal.
2. The **spinous process** projects posteriorly from the vertebral arch.
3. The **body of the vertebra** is located anterior to the spinal canal.
4. Orientation labels identify **posterior** at the top of the figure and **anterior** at the bottom.

Spinal Cord and Nerve Pathways

1. The **spinal cord** occupies the center of the vertebral canal and is shown in cross-section, with gray matter surrounded by white matter.
2. On each side of the spinal cord, a **dorsal root** extends laterally and contains a **dorsal root ganglion**, which houses the cell bodies of sensory neurons.
3. A **ventral root** emerges from the anterior aspect of the spinal cord and contains motor nerve fibers.
4. The dorsal and ventral roots join to form a **spinal nerve**.
5. Each spinal nerve quickly divides into a smaller **dorsal ramus** and a larger **ventral ramus**, which distribute sensory and motor fibers to different regions of the body.

Meningeal Layers

1. An enlarged inset shows the protective coverings surrounding the spinal cord.
2. From outermost to innermost, the layers are:
   - **Epidural space**
   - **Dura mater**
   - **Arachnoid mater**
   - **Subarachnoid space**
   - **Pia mater**
3. The **pia mater** closely adheres to the surface of the spinal cord.

4. The **subarachnoid space** surrounds the spinal cord and contains cerebrospinal fluid.

The figure demonstrates the organization of the spinal cord within the vertebral canal and the arrangement of spinal nerve roots. Sensory information enters the spinal cord through the **dorsal roots** and dorsal root ganglia, while motor signals leave through the **ventral roots**. The figure also illustrates the layers of connective tissue and fluid-filled spaces that protect the spinal cord within the vertebral column.

Figure 2.5.3: Anatomical Diagram

This diagram shows a **transverse (cross-sectional) view of the spinal cord**.

At the center is a **butterfly-shaped region of gray matter**. In the middle of this gray matter is a small **central canal**.

The gray matter is divided into three paired regions:

- **Posterior (dorsal) horns**, extending toward the back
- **Lateral horns**, projecting outward from each side
- **Anterior (ventral) horns**, extending toward the front

Surrounding the gray matter is **white matter**, which is divided into:

- **Posterior (dorsal) columns**
- **Lateral columns**
- **Anterior (ventral) columns**

The diagram identifies the major anatomical regions of **gray matter** and **white matter** in the spinal cord and shows their spatial relationships within a transverse section.

Figure 2.5.4: Histological Micrograph

1. A stained microscopic cross-section of the spinal cord is shown.
2. A butterfly-shaped region of gray matter occupies the center of the section.
3. The gray matter forms dorsal and ventral horns that extend toward the outer edge of the spinal cord.
4. A small central canal is visible near the center of the gray matter.
5. White matter surrounds the gray matter and occupies the outer portion of the spinal cord.
6. The white matter contains bundles of myelinated axons, while the gray matter contains neuron cell bodies.

This micrograph demonstrates the organization of spinal cord tissue in cross-section, showing the central gray matter and surrounding white matter. It helps viewers identify the gray matter horns, central canal, and outer white matter regions in an actual histological specimen.

Figure 2.5.5: Histological Section of a Dorsal Root Ganglion and Peripheral Nerve

The figure shows a histological section of a **dorsal root, or spinal, ganglion** and an associated peripheral nerve.

1. The central oval structure is labeled **dorsal root or spinal ganglion**. This ganglion contains the cell bodies of sensory neurons.
2. Within the ganglion, numerous large, circular neuron cell bodies are visible. These structures are labeled **cell bodies of sensory neurons** and are clustered together within the ganglion.
3. Extending away from the ganglion is a bundle of nerve fibers labeled **axon bundles or nerve**. These axons carry sensory information between peripheral tissues and the spinal cord.
4. The ganglion is enclosed by connective tissue and is positioned adjacent to the nerve fiber bundles, demonstrating the relationship between sensory neuron cell bodies and their axons.
5. The surrounding tissue contains connective tissue, blood vessels, and adipose tissue typical of the area surrounding peripheral nerves.

The figure demonstrates the organization of a dorsal root ganglion. The cell bodies of sensory neurons are grouped within the ganglion, while their axons extend into peripheral nerves that transmit sensory information between the body and the central nervous system.

Figure 2.5.6: High-Magnification Histological View of a Dorsal Root Ganglion

This figure is a high-magnification histological light micrograph shows a cross-section of a **dorsal root ganglion**. The image highlights the microscopic structure of peripheral nervous tissue and identifies two key cellular components.

1. **Cell bodies of sensory neurons** appear as multiple large, rounded pseudounipolar neuron cell bodies, or somas. Each cell body contains a prominent central nucleus and abundant cytoplasm.
2. **Satellite cells** form a supportive layer around the outer perimeter of each large sensory neuron cell body. Their small, dense, rounded nuclei create a continuous ring closely surrounding each soma.
3. The image emphasizes the close relationship between the large sensory neuron cell bodies and the surrounding satellite cells, which provide structural and metabolic support.
4. An inset in the upper-right corner provides a low-magnification overview showing a spinal cord cross-section connected to the adjacent dorsal root ganglion, indicating the anatomical origin of the high-magnification image.

The figure demonstrates the microscopic organization of a dorsal root ganglion, where the cell bodies of sensory neurons are clustered together and individually surrounded by satellite cells that support and protect them.

Figure 2.5.7: Sensory and Motor Pathways of a Spinal Nerve

The figure illustrates the organization of sensory and motor pathways associated with a spinal nerve and their connections to the spinal cord.

1. A cross-section of the spinal cord is shown on the right. The central **gray matter** is surrounded by **white matter**.
2. A **dorsal root** extends from the posterior side of the spinal cord. Along this root is a **dorsal root ganglion**, which contains the cell bodies of sensory neurons.
3. Sensory nerve fibers, shown in blue, travel from the peripheral nerve through the dorsal root ganglion and enter the spinal cord through the dorsal root. These fibers carry sensory information from the body to the central nervous system.
4. A **ventral root** extends from the anterior side of the spinal cord. Motor nerve fibers, shown in red, leave the spinal cord through the ventral root.
5. The dorsal root and ventral root join to form a mixed **spinal nerve**, which contains both sensory and motor fibers.
6. Distal to the spinal nerve, branches extend toward peripheral tissues, carrying sensory information to the spinal cord and motor commands from the spinal cord to muscles and other target tissues.
7. The color coding distinguishes pathway function: blue pathways represent sensory, or afferent, fibers entering the spinal cord, and red pathways represent motor, or efferent, fibers leaving the spinal cord.

The figure demonstrates the functional organization of a spinal nerve. Sensory information enters the spinal cord through the dorsal root and dorsal root ganglion, while motor signals exit through the ventral root. The two roots combine to form a spinal nerve that contains both sensory and motor fibers.

Figure 2.5.8: Major Spinal Nerve Plexuses and Their Branches

The figure shows the major **spinal nerve plexuses** and several of their principal peripheral nerves in a posterior view of the body. The spinal cord is shown within the vertebral column, with spinal nerve roots emerging from cervical, thoracic, lumbar, and sacral spinal levels.

1. The **cervical plexus** is formed by nerves arising from the upper cervical spinal cord, **C1 through C5**. A major branch, the **phrenic nerve**, descends into the thoracic region and provides motor innervation to the diaphragm.
2. The **brachial plexus** is formed primarily by spinal nerves **C5 through T1**. This plexus supplies the upper limb and gives rise to several major nerves, including:
    - The **axillary nerve**
    - The **radial nerve**

    - The **median nerve**
    - The **ulnar nerve**
3. Thoracic spinal nerves are shown extending from the thoracic region. Unlike the cervical, lumbar, and sacral regions, most thoracic nerves do not form a plexus and instead continue as individual intercostal nerves.
4. The **lumbar plexus** is located in the lower back and is formed primarily by spinal nerves **L1 through L4**. Major branches include:
    - The **femoral nerve**
    - The **obturator nerve**
5. Inferior to the lumbar plexus is the **sacral plexus**, formed by contributions from lower lumbar and sacral spinal nerves, **L4 through S4**.
6. The sacral plexus gives rise to the **sciatic nerve**, the largest peripheral nerve in the body.
7. Distally, the sciatic nerve divides into the:
    - **Tibial nerve**
    - **Common fibular, or peroneal, nerve**
8. Labels along the vertebral column identify spinal cord levels from the cervical region through the sacral region, illustrating the origins of each plexus.

The figure demonstrates how spinal nerves are organized into interconnected networks called **plexuses**. These plexuses redistribute nerve fibers from multiple spinal cord levels into peripheral nerves that supply the neck, upper limbs, pelvis, and lower limbs.

Figure 2.5.9: Comparison of Dermatomes and Cutaneous Nerve Territories of the Anterior Body

The figure compares two maps of sensory innervation on the anterior surface of the body. The left half illustrates **dermatomes**, which are areas of skin supplied by individual spinal nerve roots, while the right half illustrates the distribution of major **cutaneous nerves**, which are peripheral nerves containing fibers from multiple spinal nerve roots.

Left Side: Dermatome Map

1. The head and face are supplied by the **trigeminal nerve**, while the posterior head and neck are supplied primarily by **C2 through C3** spinal nerves.
2. The neck and shoulder region are supplied by **C3 through C4**.
3. The upper limb is supplied by spinal nerves **C5 through T1**:
    - **C5 through C6** supply the shoulder and lateral upper arm.
    - **C6 through C8** extend through the forearm and hand.
    - **C8 through T1** supply portions of the medial forearm and hand.
4. The trunk is arranged in horizontal bands representing thoracic dermatomes:
    - **T2 through T12** extend from the upper chest to the lower abdomen.
5. The lower abdominal and inguinal region is supplied primarily by **L1**.
6. The lower limb is supplied by lumbar and sacral dermatomes:
    - **L1 through L2** supply the upper thigh.
    - **L2 through L3** supply the anterior thigh.
    - **L3 through L4** supply the medial knee and leg.
    - **L4 through L5** extend to the lower leg and foot.
    - **L5 through S2** supply portions of the lateral leg and foot.
    - **S2 through S3** supply the perineal region.

Right Side: Cutaneous Nerve Distribution

1. The skin is also labeled according to the sensory territories of major peripheral nerves, including:
    - Supraclavicular
    - Superior lateral
    - Medial brachial
    - Intercostobrachial
    - Medial antebrachial

- Lateral antebrachial
- Iliohypogastric
- Genitofemoral
- Ilioinguinal
- Ulnar
- Median
- Lateral cutaneous
- Posterior cutaneous
- Intermediate cutaneous
- Medial cutaneous
- Obturator
- Patellar plexus
- Superficial fibular
- Sural

The figure demonstrates two related but distinct ways of mapping sensory innervation of the skin. **Dermatomes** represent areas supplied by individual spinal nerve roots, whereas **cutaneous nerve territories** represent areas supplied by named peripheral nerves formed from multiple spinal roots. Comparing the two maps illustrates how sensory information from the skin is organized both at the spinal cord level and within the peripheral nervous system.

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3: SENSORY INFORMATION AND COORDINATED MOTOR RESPONSES

Chapter Overview



Figure 3.1: When high temperature is sensed in the skin, a withdrawal is initiated by the muscles of the arm. Sensory neurons are activated by a stimulus, which is sent to the central nervous system, and a motor response is sent out to the skeletal muscles that control this movement. (CC BY 4.0 ; via Introduction).

This chapter explains how the nervous system detects changes inside and outside the body, turns them into electrical signals, and uses that information to plan and carry out movements. You will learn about the main types of sensory receptors, how general (somatic and visceral) and special senses are organized, how sensory information is sent to and processed in the central nervous system, and how motor commands are generated, from simple reflexes to complex voluntary actions. The goal is to help you follow the full path: stimulus → receptor → sensory pathway → central processing → motor pathway → movement.

Types of Sensory Receptors: How Stimuli Become Signals

Sensory receptors are specialized cells or nerve endings that detect specific kinds of energy, such as light, sound, pressure, chemicals, or temperature, and convert them into nerve impulses. Their structure and location are closely related to the kind of information they provide.

You will first look at structural types of receptors, including free nerve endings, encapsulated nerve endings, and specialized receptor cells. Free nerve endings are bare dendrites that detect pain, temperature, and some touch. Encapsulated endings are wrapped in connective tissue and are usually involved in touch, pressure, or vibration. Specialized receptor cells, such as photoreceptors in the eye or hair cells in the inner ear, are not neurons themselves but communicate with sensory neurons.

You will then consider location-based types of receptors. Exteroceptors are near the surface of the body and detect the external environment, such as light, sound, and touch. Interoceptors are in internal organs and detect conditions inside the body, such as stretch, chemical changes, and blood pressure. Proprioceptors are in muscles, tendons, and joints, and give information about body position and movement.

Finally, you will study functional types of receptors. Mechanoreceptors respond to mechanical forces such as touch, pressure, vibration, stretching, hearing, and balance. Thermoreceptors respond to temperature changes. Chemoreceptors respond to chemicals in the environment or body fluids and include receptors for smell, taste, and blood chemistry, as well as osmoreceptors that detect changes in fluid concentration. Nociceptors detect tissue damage and are responsible for pain. Photoreceptors in the

retina respond to light and allow vision. For each type, you will relate what kind of stimulus it detects, what information it sends to the nervous system, and which sensory pathways it activates.

General (Somatic and Visceral) Senses

General senses use receptors that are spread throughout the body, not concentrated in special organs. You will learn to distinguish between somatic and visceral general senses, and to follow their pathways into the central nervous system.

Somatic senses include touch, pressure, vibration, temperature, proprioception, and pain from skin, muscles, joints, and connective tissues. These senses give you detailed information about the external world and your body position, allowing you to interact with your environment and control movement.

Visceral senses come from internal organs. They include stretch of hollow organs, chemical changes in blood and body fluids, and certain kinds of deep pain. Many visceral sensations do not reach conscious awareness, but they are important for reflex control of functions such as blood pressure, digestion, and breathing. Some strong visceral stimuli, such as severe organ damage or ischemia, can be felt as pain, often referred to body surface areas.

You will follow how somatic sensory information travels through spinal and cranial nerves, enters the spinal cord or brainstem, and is relayed upward to higher centers, such as the thalamus and somatosensory cortex, for conscious perception. You will also see how some signals are used locally for reflex responses without reaching conscious awareness.

Special Senses: Dedicated Organs and Pathways

Special senses rely on specific organs and highly organized pathways to the brain. In this chapter, you will survey the anatomy and basic physiology of smell, taste, vision, hearing, and equilibrium, and see how each has a “dedicated line” to specific brain regions.

For olfaction (smell), you will study receptor cells in the nasal epithelium, which detect odor molecules and send signals directly to the olfactory bulb, olfactory cortex, and limbic system. This direct link explains why smells are often strongly connected to emotions and memories.

For gustation (taste), you will look at taste buds on the tongue and other oral structures. Taste receptor cells respond to different basic tastes and send information via cranial nerves to the brainstem, then to the thalamus and gustatory cortex.

For vision, you will review the eye and retina, especially the photoreceptors (rods and cones) that convert light into nerve signals. These signals travel along the optic nerve to visual relay centers in the thalamus and then to the primary visual cortex and association areas, where images are analyzed and interpreted.

For audition (hearing), you will examine the cochlea of the inner ear, where hair cells detect sound vibrations. Signals travel along the auditory nerve to brainstem nuclei that begin to process sound frequency, intensity, and location before continuing to higher auditory centers in the midbrain, thalamus, and cortex.

For equilibrium (balance), you will study vestibular receptors in the semicircular canals and otolith organs, which detect head movement and position. Their signals go to vestibular nuclei in the brainstem and to the cerebellum, where they are combined with visual and proprioceptive input to help maintain posture and coordinate eye movements. For each special sense, you will connect receptor structure to its function and trace the main central processing routes.

Central Processing of Sensory Information

Sensory inputs from general and special senses are integrated in the central nervous system to create perception and to guide action. In this section you will compare the major ascending pathways that carry somatic sensory information to the brain, and see how they are organized.

You will compare the dorsal column–medial lemniscus pathway, which carries fine touch, vibration, and proprioception, with the spinothalamic pathways, which carry pain, temperature, and crude touch. You will also study the trigeminal system, which carries similar information from the face. For each pathway, you will note where the fibers cross the midline and where they synapse on their way to the thalamus and cortex.

You will examine the central role of the thalamus as the primary relay station for most sensory modalities, except olfaction. The thalamus filters and routes incoming information to appropriate cortical areas. You will then see how different cortical regions, such as the primary somatosensory cortex, visual cortex, auditory cortex, vestibular areas, and multimodal association areas, work together to form a coherent picture of the body and the environment.

Planning and Initiating Motor Responses

Once sensory information is processed, the brain must decide what to do and how to do it. In this section, you will study how different cortical areas plan and initiate motor responses.

You will see how the prefrontal cortex and association cortices are involved in deciding on actions, setting goals, and planning strategies based on current sensory input, memories, and expectations. Premotor and supplementary motor areas organize movement sequences, such as reaching, grasping, or speaking, and prepare the motor system for action.

You will then focus on the primary motor cortex, which sends commands down corticospinal and corticobulbar tracts to activate lower motor neurons in the spinal cord and brainstem. These lower motor neurons directly control skeletal muscles. You will learn the concept of a motor unit (a single motor neuron and all the muscle fibers it innervates) and how changing the number and type of motor units recruited controls the force and precision of movements. Throughout, you will see how continuous sensory feedback, especially from proprioceptors and the vestibular system, updates the brain about the success of the movement and allows quick corrections.

Posture, Coordination, and Extrapyramidal Pathways

Not all motor output is under conscious control. Many pathways work automatically to maintain posture, balance, and smooth, coordinated movement. This section introduces the main brainstem and subcortical systems that support these functions.

You will explore brainstem motor pathways such as the vestibulospinal, reticulospinal, rubrospinal, and tectospinal tracts. These pathways adjust muscle tone, head and body position, and postural reflexes in response to sensory input from the vestibular apparatus, visual system, and proprioceptors.

You will also examine the contributions of the basal nuclei and cerebellum. The basal nuclei help select and initiate appropriate movements and inhibit unwanted ones. The cerebellum compares intended movements with actual performance, using sensory feedback, and makes rapid corrections to improve accuracy, timing, and coordination. You will see how these systems work together and with the cerebral cortex to produce smooth, adaptive, and efficient movement.

Somatic Reflexes: Rapid, Protective Circuits

Reflexes are fast, automatic responses to specific stimuli that help protect the body and maintain posture without requiring conscious thought. In this section, you will analyze the basic components of a reflex arc: the receptor that detects the stimulus, the sensory neuron that carries the signal into the CNS, the integration center (often within the spinal cord), the motor neuron that carries the command out, and the effector, usually a skeletal muscle, that produces the response.

You will learn to differentiate simple spinal reflexes, such as the stretch reflex and withdrawal reflex, from more complex, polysynaptic reflexes that coordinate multiple muscle groups on one or both sides of the body. You will relate specific receptors, such as muscle spindles and Golgi tendon organs, to the reflexes they mediate and to their roles in posture control and protection against muscle damage.

By the end of this chapter, you should be able to link specific receptor types to the sensory modalities they detect, trace how information from general and special senses reaches the brain, and explain how the central nervous system transforms that information into appropriate, coordinated motor responses. You will understand how rapid reflexes, automatic postural systems, and voluntary actions all depend on accurate sensory input and precise central processing.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- After completing this chapter, the student will be able to:
- Describe the major types of sensory receptors and explain how they convert different stimuli into nerve signals.
- Distinguish general somatic, general visceral, and special senses, and outline their basic receptor locations and pathways.
- Summarize how sensory information from general and special senses is routed and processed in key central nervous system regions.
- Explain how the brain uses sensory information to plan and initiate voluntary movements.
- Describe how brainstem pathways, basal nuclei, and cerebellum contribute to posture, balance, and coordination.
- Describe the components of a basic somatic reflex arc and differentiate simple spinal reflexes from more complex reflexes.

3.1: Touch, Taste and Smell

3.2: Hearing, Balance and Vision

3.3: Integration of Somatic Functions

3.4: Somatic Motor Responses

3.5: Terms and Definitions

3.6: Chapter Review Questions

3.7: Critical Thinking Self-Assessment

3.8: Alternate Text Descriptions

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3.1: Touch, Taste and Smell

Sensory Receptors

A major role of sensory receptors is to help us learn about the environment around us, or about the state of our internal environment. Stimuli from varying sources, and of different types, are received and changed into the electrochemical signals of the nervous system. This occurs when a stimulus changes the cell membrane of a sensory neuron. The stimulus causes the sensory cell to produce an action potential that is relayed into the central nervous system (CNS), where it is integrated with other sensory information—or sometimes higher cognitive functions—to become a conscious perception of that stimulus. The central integration may then lead to a motor response.

Describing sensory function with the term sensation or perception is a deliberate distinction. Sensation is the activation of sensory receptor cells at the level of the stimulus. Perception is the central processing of sensory stimuli into a meaningful pattern. Perception is dependent on sensation, but not all sensations are perceived. Receptors are the cells or structures that detect sensations. A receptor cell is changed directly by a stimulus, such as opening of ion channels in the plasma membrane.

Stimuli in the environment activate specialized receptor cells in the peripheral nervous system. Different types of stimuli are sensed by different types of receptor cells. Receptor cells can be classified into types on the basis of three different criteria: cell type, position, and function. Receptors can be classified structurally on the basis of cell type and their position in relation to stimuli they sense. They can also be classified functionally on the basis of the **transduction** of stimuli, or how the mechanical stimulus, light, or chemical changed the cell membrane electrical properties.

Structural Receptor Types

The first structural classification of sensory receptors is based on the cell type. The cells that interpret information about the environment can be either (1) a neuron that has a **free nerve ending**, with dendrites embedded in tissue that would receive a sensation; (2) a neuron that has an **encapsulated ending** in which the sensory nerve endings are covered in connective tissue that enhances their sensitivity; or (3) a specialized **receptor cell**, which has distinct structural components that interpret a specific type of stimulus (Figure 3.1.1). The pain and temperature receptors in the dermis of the skin are examples of neurons that have free nerve endings. Also located in the dermis of the skin are lamellated corpuscles, neurons with encapsulated nerve endings that respond to pressure and touch. The cells in the retina that respond to light stimuli are an example of a specialized receptor, a **photoreceptor**.

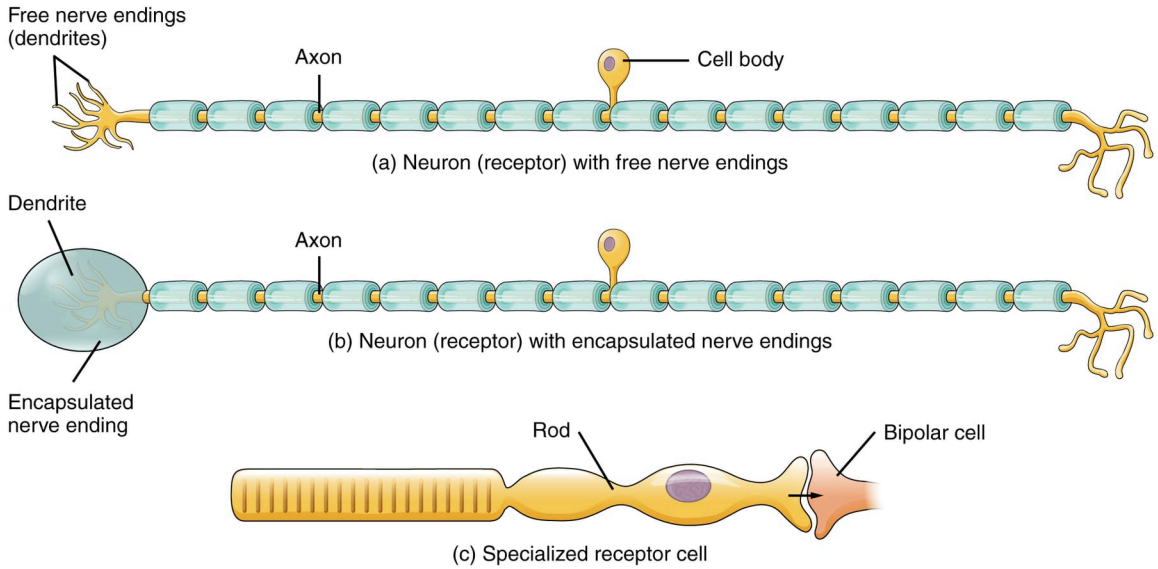


Figure 3.1.1: Sensory receptors can be classified on the basis of their structure. Sensory neurons can have either free nerve endings on their dendritic side such as in a or encapsulated endings where dendrites are covered by connective tissue such as in b. In c, photoreceptors in the eyes, such as rod cells depicted here, are examples of specialized receptor cells. These cells release neurotransmitters onto a bipolar cell, which then synapses with the optic nerve neurons. (CC BY 4.0; via Touch, Taste and Smell). Alternative text description of the Figure 3.1.1.

Functional Receptor Types

A third classification of receptors is by how the receptor transduces stimuli into electrical changes. Stimuli are of three general types. Some stimuli are ions and macromolecules that affect transmembrane receptor proteins when these chemicals diffuse across the cell membrane, for example the ones found in food. Some stimuli are physical variations in the environment that affect the electrical properties of the receptor cell membrane, for example touch. Other stimuli include the electromagnetic radiation from visible light. For humans, the only electromagnetic energy that is perceived by our eyes is visible light. Some other organisms have receptors that humans lack, such as the heat sensors of snakes, the ultraviolet light sensors of bees, or magnetic receptors in migratory birds.

Receptor cells can be further categorized on the basis of the type of stimuli they transduce. Chemical stimuli can be interpreted by a **chemoreceptor** that interprets chemical stimuli, such as an object's taste or smell. **Osmoreceptors** respond to solute concentrations of body fluids. Additionally, pain is primarily a chemical sense that interprets the presence of chemicals from tissue damage, or similar intense stimuli, through a **nociceptor**. Physical stimuli, such as pressure and vibration, as well as the sensation of sound and body position (balance), are interpreted through a **mechanoreceptor**. Another physical stimulus that has its own type of receptor is temperature, which is sensed through a **thermoreceptor** that is either sensitive to temperatures above (heat) or below (cold) normal body temperature.

Sensory Modalities

Ask anyone what the senses are, and they are likely to list the five major senses—taste, smell, touch, hearing, and sight. However, these are not all of the senses. The most obvious omission from this list is balance. Also, what is referred to simply as touch can be further subdivided into pressure, vibration, stretch, and hair-follicle position, on the basis of the type of mechanoreceptors that perceive these touch sensations. Other overlooked senses include temperature perception by thermoreceptors and pain perception by nociceptors.

Senses can be classified as either general or special. A **general sense** is one that is distributed throughout the body and has receptor cells within the structures of other organs. Mechanoreceptors in the skin, muscles, or the walls of blood vessels are examples of this type. General senses often contribute to the sense of touch, as described above, or to **proprioception** (awareness of body position) and **kinesthesia** (awareness of body movement), or to a **visceral sense**, which is most important to autonomic functions. A **special sense** is one that has a specific organ devoted to it, namely the eye, inner ear, tongue, or nose.

Each of the senses is referred to as a **sensory modality**. Modality refers to the way that information is encoded, which is similar to the idea of transduction. The main sensory modalities can be described on the basis of how each is transduced.

The chemical senses are taste and smell. The general sense that is usually referred to as touch includes chemical sensation in the form of nociception, or pain. Pressure, vibration, muscle stretch, and the movement of hair by an external stimulus, are all sensed by mechanoreceptors. Hearing and balance are also sensed by mechanoreceptors. Finally, vision involves the activation of photoreceptors.

Listing all the different sensory modalities, which can number as many as 17, involves separating the five major senses into more specific categories, or **submodalities**, of the larger sense. An individual sensory modality represents the sensation of a specific type of stimulus. For example, the general sense of touch, which is known as **somatosensation**, can be separated into light pressure, deep pressure, vibration, itch, pain, temperature, or hair movement.

Somatosensation (Touch)

Somatosensation is considered a general sense, as opposed to the special senses discussed in this section. Somatosensation is the group of sensory modalities that are associated with touch, proprioception, and interoception. These modalities include pressure, vibration, light touch, tickle, itch, temperature, pain, proprioception, and kinesthesia. This means that its receptors are not associated with a specialized organ, but are instead spread throughout the body in a variety of organs. Many of the somatosensory receptors are located in the skin, but receptors are also found in muscles, tendons, joint capsules, ligaments, and in the walls of visceral organs.

Two types of somatosensory signals that are transduced by free nerve endings are pain and temperature. These two modalities use thermoreceptors and nociceptors to transduce temperature and pain stimuli, respectively. Temperature receptors are stimulated when local temperatures differ from body temperature. Some thermoreceptors are sensitive to just cold and others to just heat. Nociception is the sensation of potentially damaging stimuli. Mechanical, chemical, or thermal stimuli beyond a set threshold will elicit painful sensations. Stressed or damaged tissues release chemicals that activate receptor proteins in the nociceptors.

If you drag your finger across a textured surface, the skin of your finger will vibrate. Such low frequency vibrations are sensed by mechanoreceptors called Merkel cells, also known as type I cutaneous mechanoreceptors. Merkel cells are located in the stratum basale of the epidermis. Deep pressure and vibration is transduced by lamellated (Pacinian) corpuscles, which are receptors with encapsulated endings found deep in the dermis, or subcutaneous tissue. Light touch is transduced by the encapsulated endings known as tactile (Meissner) corpuscles. Follicles are also wrapped in a plexus of nerve endings known as the hair follicle plexus. These nerve endings detect the movement of hair at the surface of the skin, such as when an insect may be walking along the skin. Stretching of the skin is transduced by stretch receptors known as bulbous corpuscles. Bulbous corpuscles are also known as Ruffini corpuscles, or type II cutaneous mechanoreceptors.

Other somatosensory receptors are found in the joints and muscles. Stretch receptors monitor the stretching of tendons, muscles, and the components of joints. For example, have you ever stretched your muscles before or after exercise and noticed that you can only stretch so far before your muscles spasm back to a less stretched state? This spasm is a reflex that is initiated by stretch receptors to avoid muscle tearing. Such stretch receptors can also prevent over-contraction of a muscle. In skeletal muscle tissue, these stretch receptors are called muscle spindles. Golgi tendon organs similarly transduce the stretch levels of tendons. Bulbous corpuscles are also present in joint capsules, where they measure stretch in the components of the skeletal system within the joint. The types of nerve endings, their locations, and the stimuli they transduce are presented in Table 3.1.1.

Table 3.1.1: Mechanoreceptors of Somatosensation

Name	Location(s)	Stimuli
Free nerve endings	Dermis, cornea, tongue, joint capsules, visceral organs	Pain, temperature, mechanical deformation
Mechanoreceptors or Merkel's discs	Epidermal–dermal junction, mucosal membranes	Low frequency vibration (5–15 Hz)
Bulbous (or Ruffini's) corpuscle	Dermis, joint capsules	Stretch
Tactile (or Meissner's) corpuscle	Papillary dermis, especially in the fingertips and lips	Light touch, vibrations below 50 Hz
Lamellated (or Pacinian) corpuscle	Deep dermis, subcutaneous tissue	Deep pressure, high-frequency vibration (around 250 Hz)

Name	Location(s)	Stimuli
Hair follicle plexus	Wrapped around hair follicles in the dermis	Movement of hair
Muscle spindle	In line with skeletal muscle fibers	Muscle contraction and stretch
Tendon stretch (or Golgi tendon) organ	In line with tendons	Stretch of tendon

Gustation (Taste)

Only a few recognized submodalities exist within the sense of taste, or **gustation**. Until recently, only four tastes were recognized: sweet, salty, sour, and bitter. Research at the turn of the 20th century led to recognition of the fifth taste, umami, during the mid-1980s. **Umami** is a Japanese word that means "delicious taste," and is often translated to mean savory. Very recent research has suggested that there may also be a sixth taste for fats, or lipids.

Gustation is the special sense associated with the tongue. The surface of the tongue, along with the rest of the oral cavity, is lined by a stratified squamous epithelium. Raised bumps called **papillae** (singular = papilla) contain the structures for gustatory transduction. There are four types of papillae, based on their appearance (Figure 3.1.2): circumvallate, foliate, filiform, and fungiform. Circumvallate papillae are the largest papillae on the tongue and are located towards the posterior surface of the tongue. They contain the majority of the taste buds. Foliate papillae are located on the posterolateral region of the tongue and they are mostly used during infancy and childhood. Filiform papillae are shaped like bristles and do not contain taste buds. They are important for detecting texture and manipulating food. Fungiform papillae are located on the tip and sides of the tongues and contain few taste buds. Within the structure of the papillae are **taste buds** which are recessed into a **taste pore** and are shaped like an onion. Taste buds contain a variety of cells. **Basal cells** are undifferentiated stem cells located near the base of the taste bud that undergo continuous regeneration to replace the other cells of the taste buds. **Transitional cells** are scattered throughout the taste buds and support the other cells. Specialized **gustatory receptor cells** have microvilli called **taste hairs** and can absorb the chemicals contained within foods that are ingested. These cells release neurotransmitters based on the amount of the chemical in the food. These cells are not neurons but they are in contact with the dendrites of sensory neurons.

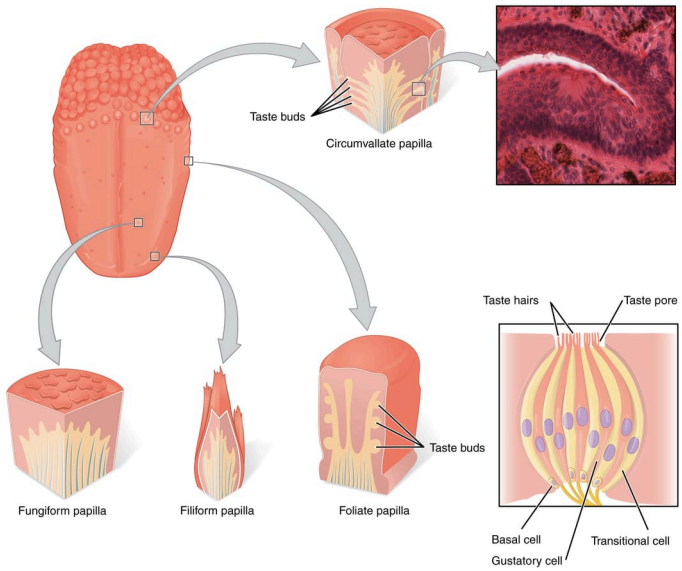


Figure 3.1.2: The tongue is covered with small bumps, called papillae, which contain taste buds that are sensitive to chemicals in ingested food or drink. Different types of papillae are found in different regions of the tongue. From anterior to posterior we find filiform papillae, fungiform papillae, foliate papillae, and circumvallate papillae. Taste buds within the papillae have a recessed taste pore and contain three types of cells: specialized gustatory receptor cells, basal cells and transitional cells. LM × 1600. (CC BY 4.0; via Touch, Taste and Smell). Alternative text description of the Figure 3.1.2.

Once the gustatory cells are activated by the taste molecules, they release neurotransmitters onto the dendrites of sensory neurons. These neurons are part of the facial (CN VII) and glossopharyngeal (CN IX) cranial nerves, as well as a component within the vagus nerve (CN X) dedicated to the gag reflex. The facial nerve connects to taste buds in the anterior third of the tongue. The glossopharyngeal nerve connects to taste buds in the posterior two thirds of the tongue. The vagus nerve connects to taste buds

in the extreme posterior of the tongue, verging on the pharynx, which are more sensitive to noxious stimuli such as bitterness. Gustatory information is then transmitted through these cranial nerves to the brain, through the **thalamus**, which acts as a relay station, and finally into the **primary gustatory cortex** of the **insula**. It is here that this information is processed and perception is initiated.

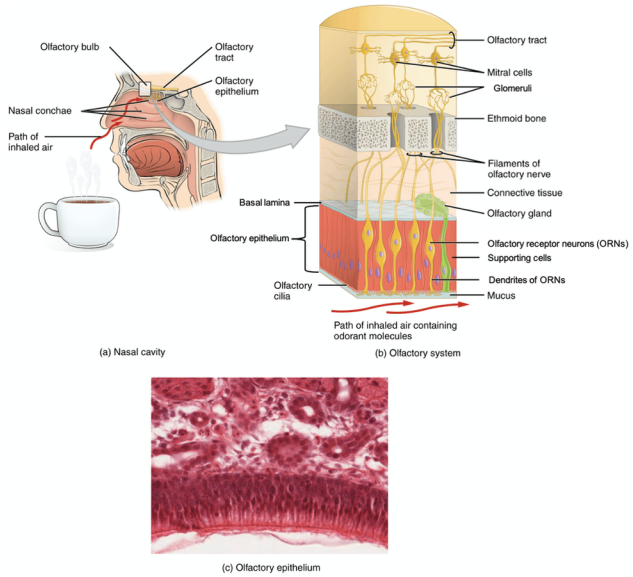
Novel studies have shown that gustatory cells are not only present on the tongue but also in the guts. In humans, sweet, umami and bitter taste cells are also present in the intestines and used to "taste" the ingested food and regulate its variations.

Olfaction (Smell)

Like taste, the sense of smell, or **olfaction**, is also responsive to chemical stimuli. Inhaled air containing **odorant molecules** (smells) enters the nasal cavity and passes by the nasal conchae. The **olfactory receptor neurons (ORNs)** are located in a small region within the superior nasal cavity (Figure $3.1.3$). This region is referred to as the **olfactory epithelium** and also contains **supporting cells** and **basal cells** (not shown), with similar functions as the homonyms in the taste buds. Each olfactory receptor neuron has dendrites that extend from the apical surface of the epithelium into the mucus lining the cavity. **Olfactory glands** within the basal lamina at the interface between the olfactory epithelium and the superior connective tissue produce the mucus that lines the olfactory epithelium, The olfactory sensory neurons present nonmotile cilia called **olfactory cilia** (or hairs) that contain the chemoreceptor for a specific odorant molecule. As airborne molecules are inhaled through the nose, they pass over the olfactory epithelial region and dissolve into the mucus. These odorant molecules bind to proteins that keep them dissolved in the mucus and help transport them to the olfactory dendrites. The odorant–protein complex binds to a receptor protein within the cell membrane of an olfactory dendrite, producing an electrical change in the olfactory neurons.

The axon of an olfactory neuron extends from the basal surface of the epithelium towards the cribriform plate of the ethmoid bone forming the **olfactory nerve (CN I)**. The olfactory fibers go through an olfactory foramen in the cribriform plate and converge to form spherical structures called **glomeruli** in the **olfactory bulb**, which resides on the ventral surface of the frontal lobe. Within the glomeruli, the olfactory receptor neurons synapse with mitral cells which in turn extend their axons to form the **olfactory tract**. From there, the axons split to travel to several brain regions. Some travel to the cerebrum, specifically to the **primary olfactory cortex** that is located in the inferior and medial areas of the temporal lobe. Others project to structures within the limbic system and hypothalamus, where smells become associated with long-term memory and emotional responses. This is how certain smells trigger emotional memories, such as the smell of food associated with one's birthplace. Smell is the one sensory modality that does not synapse in the thalamus before connecting to the cerebral cortex. This intimate connection between the olfactory system and the cerebral cortex is one reason why smell can be a potent trigger of memories and emotion.

The olfactory system can recognize seven primary odors (musky, putrid, pungent, camphoraceous, ethereal, floral, peppermint) as well as thousands of odorant molecules that produce various scents. The nasal epithelium, including the olfactory cells, can be harmed by airborne toxic chemicals. Therefore, the olfactory neurons are one of the few types of neurons that undergo mitosis and is able to replace old cells. Olfactory neurons are regularly replaced within the nasal epithelium, after which the axons of the new neurons must find their appropriate connections in the olfactory bulb. These new axons grow along the axons that are already in place in the cranial nerve.



(c) Olfactory epithelium

Figure 3.1.3: In (a) inhaled air passes by the nasal conchae to reach the olfactory epithelium in the superior nasal cavity. In (b), the olfactory epithelium is composed of olfactory receptor neurons and supporting cells. The dendrites of olfactory receptors neurons contain olfactory cilia that are embedded in the mucus lining the nasal cavity. The mucus is produced by olfactory glands located at the basal lamina. Axons of olfactory receptor neurons extend into the connective tissue and through the cribriform plate of the ethmoid bone, to synapse onto mitral cells in the glomeruli of the olfactory bulb. Axons of mitral cells extend towards the brain to form the olfactory tract. In (c), an histological slide (tissue source: simian) shows the olfactory epithelium. LM × 812. (CC BY 4.0; via Touch, Taste and Smell). Alternative text description of the Figure 3.1.3.

DISORDERS OF THE OLFACTORY SYSTEM: ANOSMIA

Blunt force trauma to the face, such as that common in many car accidents, can lead to the loss of the olfactory nerve, and subsequently, loss of the sense of smell. This condition is known as anosmia. When the frontal lobe of the brain moves relative to the ethmoid bone, the olfactory tract axons may be sheared apart. Professional fighters often experience anosmia because of repeated trauma to face and head. In addition, certain pharmaceuticals, such as antibiotics, can cause anosmia by killing all the olfactory neurons at once. If no axons are in place within the olfactory nerve, then the axons from newly formed olfactory neurons have no guide to lead them to their connections within the olfactory bulb. There are temporary causes of anosmia, as well, such as those caused by inflammatory responses related to respiratory infections or allergies.

Loss of the sense of smell can result in food tasting bland. A person with an impaired sense of smell may require additional spice and seasoning levels for food to be tasted. Anosmia may also be related to some presentations of mild depression, because the loss of enjoyment of food may lead to a general sense of despair.

The ability of olfactory neurons to replace themselves decreases with age, leading to age-related anosmia. This explains why some elderly people salt their food more than younger people do. However, this increased sodium intake can increase blood volume and blood pressure, increasing the risk of cardiovascular diseases in the elderly.

Concept Review

The cells that transduce sensory stimuli into the electrochemical signals of the nervous system are classified on the basis of structural or functional aspects of the cells. The structural classifications are either based on the anatomy of the cell that is interacting with the stimulus (free nerve endings, encapsulated endings, or specialized receptor cell), or where the cell is located relative to the stimulus (interoceptor, exteroceptor, proprioceptor). Thirdly, the functional classification is based on how the cell transduces the stimulus into a neural signal. Chemoreceptors respond to chemical stimuli and are the basis for olfaction and gustation. Related to chemoreceptors are osmoreceptors and nociceptors for fluid balance and pain reception, respectively. Mechanoreceptors respond to mechanical stimuli and are the basis for most aspects of somatosensation, as well as being the basis of audition and equilibrium in the inner ear. Thermoreceptors are sensitive to temperature changes, and photoreceptors are sensitive to light energy.

The senses are somatosensation (sensations associated with the skin and body), gustation (taste), olfaction (smell), audition (hearing), equilibrium (balance), and vision. With the exception of somatosensation, this list represents the special senses, or those systems of the body that are associated with specific organs such as the tongue or eye. The general senses can be divided into somatosensation, which is commonly considered touch, but includes tactile, pressure, vibration, temperature, and pain perception. The general senses also include the visceral senses, which are separate from the somatic nervous system function in that they do not normally rise to the level of conscious perception. The special senses are all primarily part of the somatic nervous system in that they are consciously perceived through cerebral processes, though some special senses contribute to autonomic function.

Somatosensation belongs to the general senses, which are those sensory structures that are distributed throughout the body and in the walls of various organs. Gustation and olfaction belong to the special senses. Gustation transduction is achieved by raised bumps on the tongue called papillae that contain taste buds, which in turn contain specialized gustatory receptor cells for chemical stimuli (”tastes”). Olfactory receptor neurons (unipolar) located in olfactory epithelium respond to chemical stimuli (“odorants”) of the nasal cavity. Their dendrites extend from the apical surface of the epithelium into the mucus lining the cavity and have receptors for odorants. Axons extend into the cribriform plate of the ethmoid bone to reach the olfactory bulb, forming the olfactory nerve (CN I). Once the olfactory nerve crosses the CNS, it becomes the olfactory tract and sends axons to several brain regions. The axons do not synapse onto the thalamus (unlike the other special senses).

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3.2: Hearing, Balance and Vision

Audition (Hearing)

Hearing, or **audition**, is the transduction of sound waves into a neural signal that is made possible by the structures of the ear (Figure 3.2.1). The large, fleshy structure on the lateral aspect of the head is known as the **auricle**. Some sources will also refer to this structure as the pinna, though that term is more appropriate for a structure that can be moved, such as the external ear of a cat. The C-shaped curves of the auricle direct sound waves toward the **auditory canal**, also called **ear canal**. The canal enters the skull through the external auditory meatus of the temporal bone. At the end of the auditory canal is the **tympanic membrane**, or ear drum, which vibrates after it is struck by sound waves. The auricle, ear canal, and tympanic membrane are often referred to as the **external ear**. The **middle ear** consists of a space spanned by three small bones called the **ossicles**. The three ossicles are the **malleus**, **incus**, and **stapes**, which are Latin names that roughly translate to hammer, anvil, and stirrup. The malleus is attached to the tympanic membrane and articulates with the incus. The incus, in turn, articulates with the stapes. The stapes is then attached to the **inner ear**, where the sound waves will be transduced into a neural signal. The middle ear is connected to the pharynx through the **Eustachian (or auditory) tube**, which helps equilibrate air pressure across the tympanic membrane. The tube is normally closed but will pop open when the muscles of the pharynx contract during swallowing or yawning. This helps to equalize the pressure on both sides of the ear drum with change in altitude, such as driving up the mountain or in a plane.

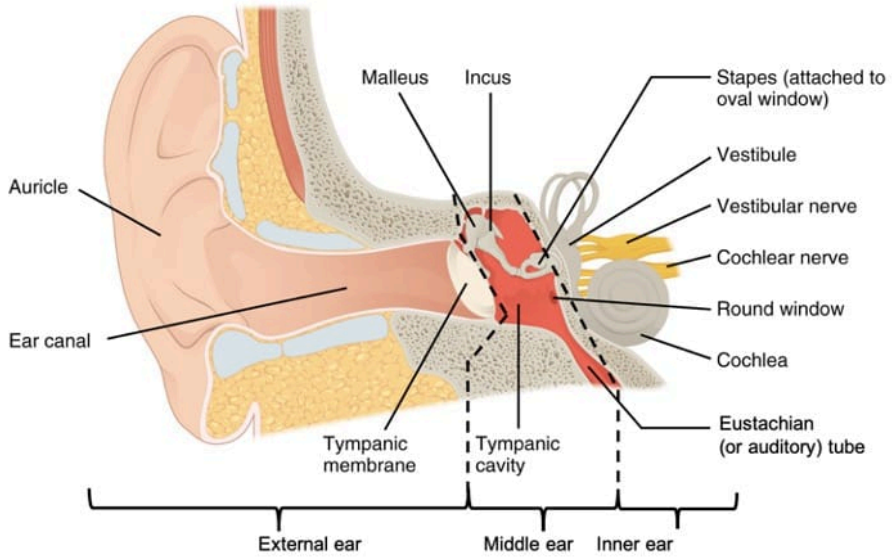


Figure 3.2.1: The external ear contains the auricle, ear canal, and tympanic membrane. The middle ear contains the ossicles and is connected to the pharynx by the Eustachian (or auditory) tube. The inner ear contains the cochlea and vestibule, which are responsible for audition and equilibrium, respectively. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.1.

Sound is transduced into neural signals within the cochlear region of the inner ear, which contains the sensory neurons of the **spiral ganglia**. These ganglia are located within the spiral-shaped cochlea of the inner ear. The cochlea is attached to the stapes through the **oval window**. The oval window is located at the beginning of a fluid-filled tube within the cochlea called the **scala vestibuli**. The scala vestibuli extends from the oval window, traveling above the **cochlear duct**, which is the central cavity of the cochlea that contains the sound-transducing neurons. At the uppermost tip of the cochlea, the scala vestibuli curves over the top of the cochlear duct. The fluid-filled tube, now called the **scala tympani**, returns to the base of the cochlea, this time traveling under the cochlear duct. The scala tympani ends at the **round window**, which is covered by a membrane that contains the fluid within the scala.

A cross-sectional view of the cochlea shows that the scala vestibuli and scala tympani run along both sides of the cochlear duct (Figure 3.2.2 and Figure 3.2.3). The cochlear duct contains several **organs of Corti**, which transduce the wave motion of the two scala into neural signals. The organs of Corti lie on top of the **basilar membrane**, which is the side of the cochlear duct located between the organs of Corti and the scala tympani. Another membrane called the **vestibular membrane** separates the cochlear duct from the scala vestibuli.

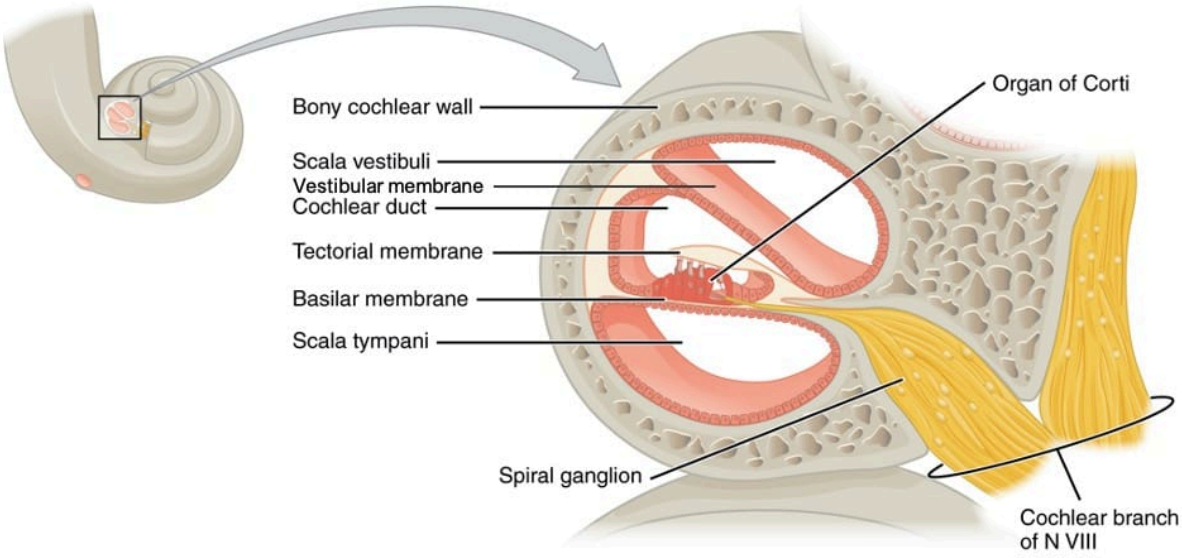


Figure 3.2.2: The membranous labyrinth separates the cochlea in three major regions. The scala vestibuli and scala tympani lie on either side of the cochlear duct. The roof of the cochlear duct is formed by the vestibular memrbane while the base of it is formed by the basilar membrane. Within the cochlear duct, the organ of Corti sits atop the basilar membrane and its cells extend towards the tectorial membrane. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.2.

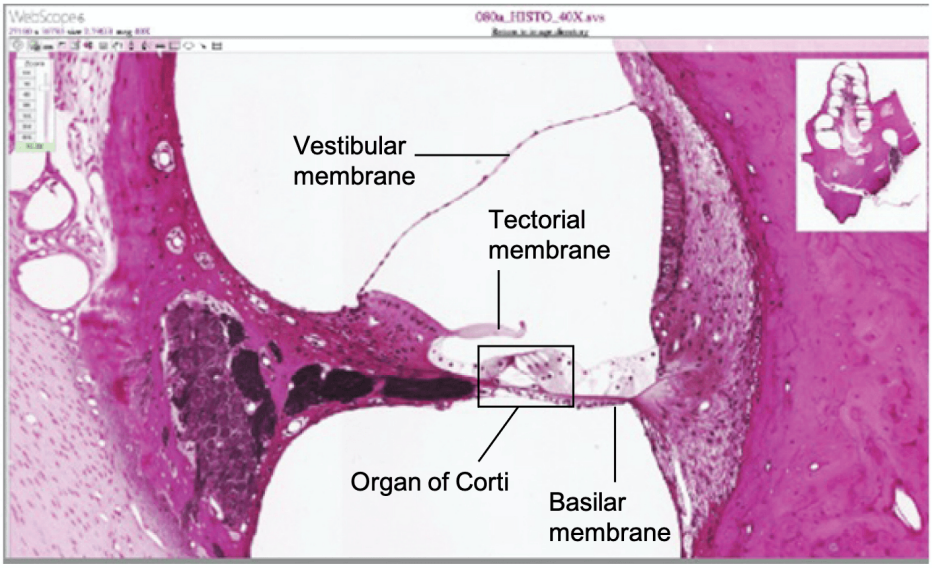


Figure 3.2.3: The organ of Corti sits on the basilar membrane and extends towards the tectorial membrane. The vestibular membrane forms the roof of the cochlear duct. LM × 412. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.3.

The organs of Corti contain **hair cells**, which are named for the hair-like **stereocilia** extending from the cell's apical surfaces (Figure 3.2.4). The stereocilia are an array of microvilli-like structures arranged from tallest to shortest. Protein fibers tether adjacent hairs together within each array, such that the array will bend in response to movements of the basilar membrane. The stereocilia extend up from the hair cells to the overlying **tectorial membrane**, which is attached medially to the organ of Corti.

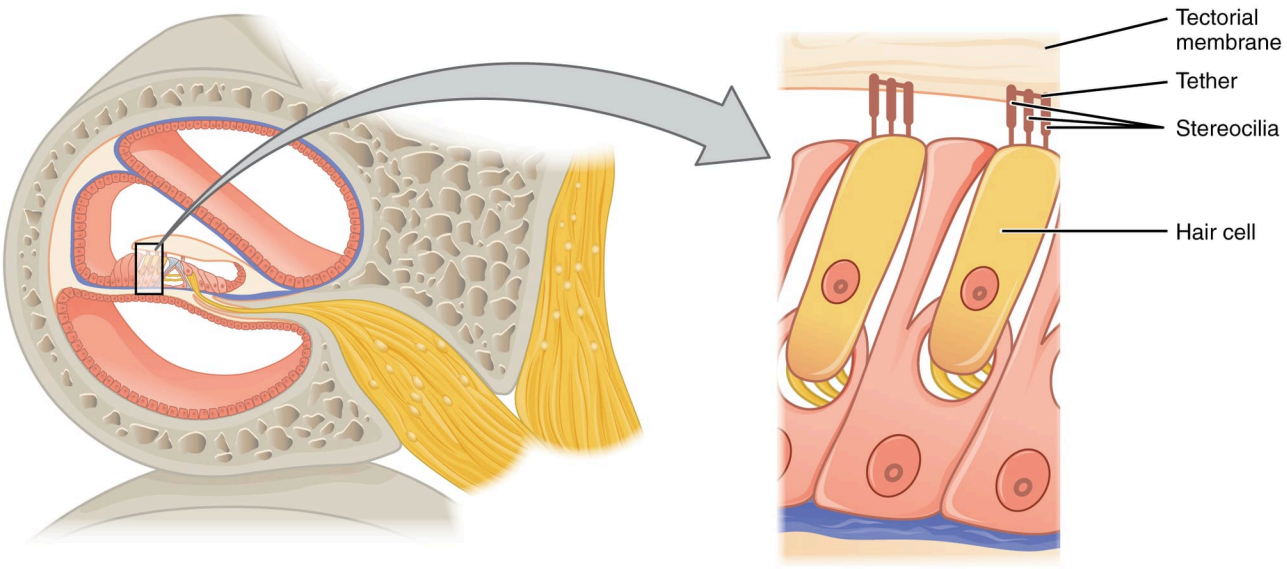


Figure 3.2.4: The hair cell is a mechanoreceptor with an array of stereocilia emerging from its apical surface. The stereocilia extend towards the tectorial membrane. The stereocilia are tethered together by proteins and bend in response to movements of the basilar membrane. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.4.

The transmission and transduction of sound require the participation of the majority of the structures of the ear. Each sound wave has a specific frequency, which depends on its pitch, and amplitude, which depends on its loudness. Sound waves are funneled into the ear canal by the auricle and reach the tympanic membrane (Figure 3.2.5). The vibration of the tympanic membrane is amplified across the ossicles. As vibrations of the ossicles travel through the oval window, the fluid of the scala vestibuli and scala tympani moves in a wave-like motion. The frequency of the fluid waves match the frequencies of the sound waves. The membrane covering the round window will bulge out or pucker in with the movement of the fluid within the scala tympani. As the fluid waves move through the scala vestibuli and scala tympani, the basilar membrane moves at a specific spot of the cochlea, depending on the frequency of the waves. Higher frequency waves move the region of the basilar membrane that is close to the base of the cochlea. Lower frequency waves move the region of the basilar membrane that is near the tip of the cochlea. When the fluid waves from the scala move the basilar membrane, the tectorial membrane slides across the stereocilia. This bends the stereocilia either toward or away from the tallest member of each array of stereocilia. When the stereocilia bend toward the tallest member of their array, tension in the protein tethers opens ion channels in the hair cell membrane. This will electrically change the hair cell membrane, triggering nerve impulses that travel down the afferent nerve fibers attached to the hair cells. When the stereocilia bend toward the shortest member of their array, the tension on the tethers slackens and the ion channels close. The relative movement of different arrays of stereocilia along the length of the basilar membrane allows the brain to perceive frequency, or pitch. The greater force of louder sounds causes an increase in the range of movement of the stereocilia of the hair cells, determining the loudness of a sound. The hair cells then transmit neural signals through the release of neurotransmitters to the **cochlear nerve** which travels within the **vestibulocochlear nerve (CN VIII)**. The nerve carries the auditory information into the brain, through the **thalamus**, and into the **primary auditory cortex** of the temporal lobe.

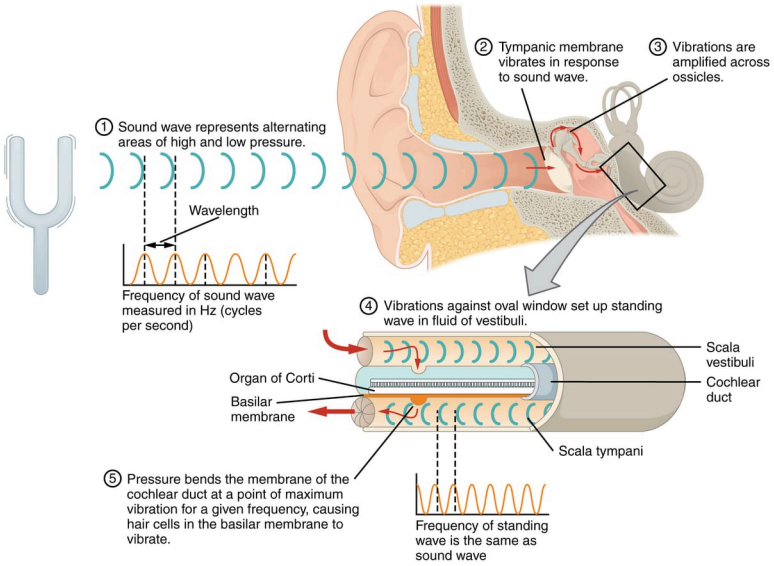


Figure 3.2.5: The transmission is represented in five steps. 1) Sound wave represents alternating areas of high and low pressure, whose frequency can be measured in Hz (cycles per second). 2) Tympanic membrane vibrates in response to sound wave. 3) Vibrations are amplified across the ossicles, in order malleus, incus, and stapes. 4) Vibrations against oval window set up standing wave in fluid of the scala vestibuli and scala tympani. The frequency of the standing wave is the same as the initial sound wave. 5) Pressure bends the membrane of the cochlear duct at a point of maximum vibration for a give frequency, causing hair cells of the Organ of Corti in the basilar membrane to vibrate. The complexity of the pressure waves is determined by the changes in amplitude and frequency of the sound waves entering the ear. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.5.

Equilibrium (Balance)

Along with audition, the inner ear is responsible for encoding information about **equilibrium**, the sense of balance. A similar mechanoreceptor—a hair cell with stereocilia—senses head position, head movement, and whether our bodies are in motion. These cells are located in the **vestibule** and **semicircular canals** within the inner ear. Vestibule and semicircular canals compose the **vestibular system**. The vestibule is the central part of the bony labyrinth, and is located posterior to the cochlea and anterior to the semicircular canals. Within the vestibule, the membranous labyrinth forms two chambers, the **utricle** and **saccule**, which contain endolymph. The utricle and saccule are interconnected by a narrow **endolymphatic duct** and their endolymph is confluent with that of the semicircular ducts and cochlear duct. Head position is sensed by the utricle and saccule, whereas head movement is sensed by the semicircular canals.

The utricle and saccule are both largely composed of **macula** tissue (plural = maculae). The macula is composed of hair cells surrounded by **supporting cells**. The stereocilia of the hair cells extend into a viscous gel called the **otolithic membrane** (Figure 3.2.6). On top of the otolithic membrane is a layer of calcium carbonate crystals, called **otoliths**. The otoliths essentially make the otolithic membrane top-heavy. The otolithic membrane moves separately from the macula in response to head movements. Tilting the head causes the otolithic membrane to slide over the macula in the direction of gravity. The moving otolithic membrane, in turn, bends the sterocilia, causing the hair cells to elicit electrical changes. The exact position of the head is interpreted by the brain based on the pattern of hair-cell activation.

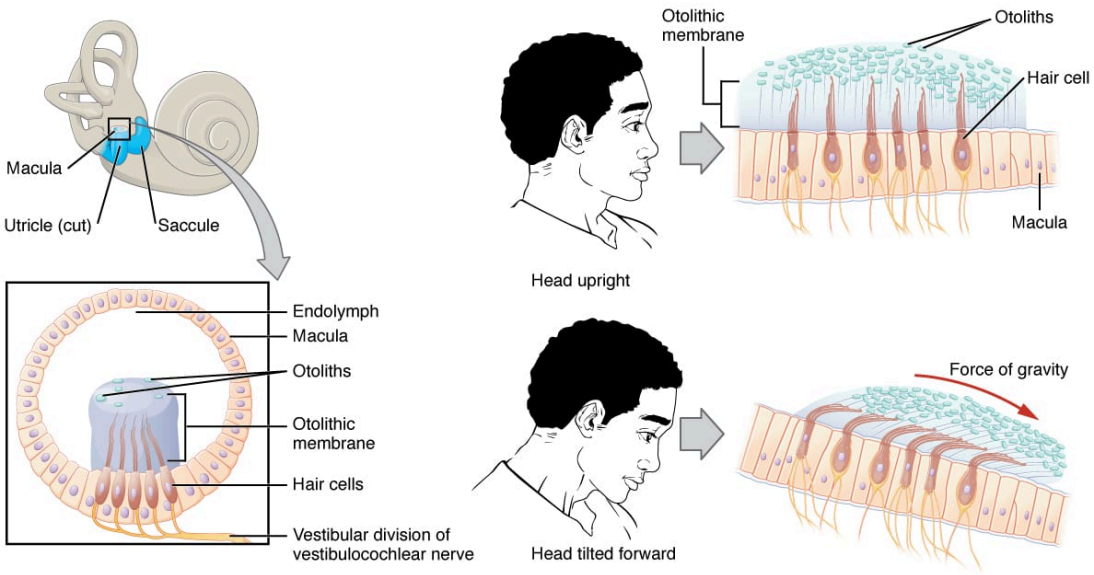


Figure 3.2.6: The maculae within the utricle and saccule are specialized structures lined with supporting cells and hair cells and filled with endolymph. The hair cells have stereocilia that are embedded in a gel-like layer called the otolithic membrane for the presence of heavy crystals called otoliths. The maculae sense linear acceleration, such as when gravity acts on the tilting head, or if the head starts moving in a straight line. The difference in inertia between the hair cell stereocilia and the otolithic membrane in which they are embedded leads to a shearing force that causes the stereocilia to bend in the direction of that linear acceleration. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.6.

The semicircular canals are three ring-like extensions of the vestibule. One is oriented in the horizontal plane, whereas the other two are oriented in the vertical plane. The anterior and posterior vertical canals are oriented at approximately 45 degrees relative to the sagittal plane (Figure 3.2.7). The base of each semicircular canal, where it meets with the vestibule, connects to an enlarged region known as the **ampulla**. The ampulla contains the hair cells that respond to rotational movement, such as turning the head while saying “no.” The stereocilia of these hair cells extend into the **cupula**, a gelatinous membrane that attaches to the top of the ampulla. As the head rotates in a plane parallel to the semicircular canal, the fluid lags, deflecting the cupula in the direction opposite to the head movement. The semicircular canals contain several ampullae, with some oriented horizontally and others oriented vertically. By comparing the relative movements of both the horizontal and vertical ampullae, the vestibular system can detect the direction of most head movements within three-dimensional (3-D) space.

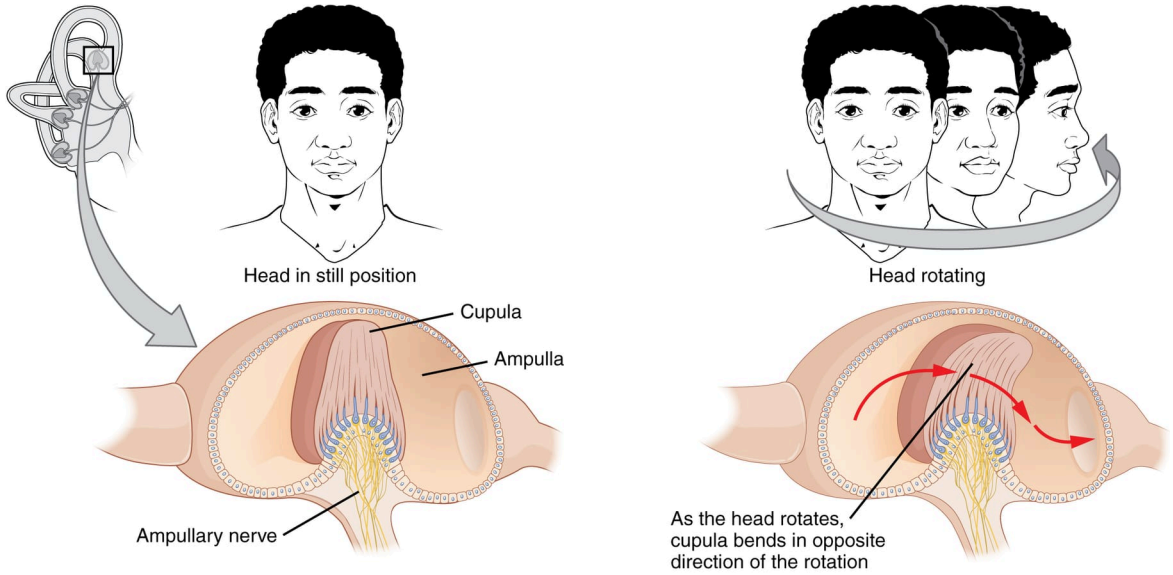


Figure 3.2.7: Rotational movement of the head is encoded by the hair cells contained in ampullae at the base of the semicircular canals. The ampulla is filled with endolymph and contains hair cells that have stereocilia embedded in a gelatinous substance called the cupula. As one of the canals moves in an arc with the head, the endolymph moves in the opposite direction, causing the cupula and stereocilia to bend. The movement of two canals within a plane results in information about the direction in which the head is moving, and activation of all six canals can give a very precise indication of head movement in three dimensions. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.7.

The neural signals generated in the vestibule and semicircular canals are transmitted through the **vestibular nerve** which travels within the **vestibulocochlear nerve (CN VIII)**. The nerve carries the equilibrium information into the brain, through the **thalamus**, and to **cerebral nuclei**, **brainstem** and **cerebellum**.

Vision

Vision is the special sense of sight that is based on the transduction of light stimuli received through the eyes. The eyes are located within either orbit in the skull. Supportive structures protect the eye, prevent objects from entering the eye, keep its surface lubricated and moist and provide superficial covering.

The bony orbits surround the eyeballs, protecting them and anchoring the soft tissues of the eye (Figure 3.2.8). The eyelids, with lashes at their leading edges, help to protect the eye from abrasions by blocking particles that may land on the surface of the eye. Each eyelid contains a thin covering of skin, a fibrous core and tarsal muscles and glands. The **tarsal muscles** is composed of smooth muscle innervated by the sympathetic nervous system and helps maintaining the elevation of the superior eyelid. The **tarsal glands** are sebaceous glands that produce an oily substance which prevents the eyelids from attaching together and prevents the overflow of tears from the open eye. The inner lid is a thin membrane known as the **palpebral conjunctiva**. The palpebral conjunctiva connects the eyelids to the eyeball. At the connection with the eyeball, a thin layer called the **ocular conjunctiva** forms a continuous layer on the external and anterior surface of the eye. The most anterior part of the eye is called the **cornea** and the ocular conjunctiva is absent here. Underneath the ocular conjunctiva lies a white layer called the **sclera**. The conjunctiva contains blood vessels and nerves that support the avascular sclera and detect foreign particles entering the eye. Bacteria and viruses can infect the conjunctiva, which then becomes red and swollen, as in pink eye (another name for pink eye is conjunctivitis).

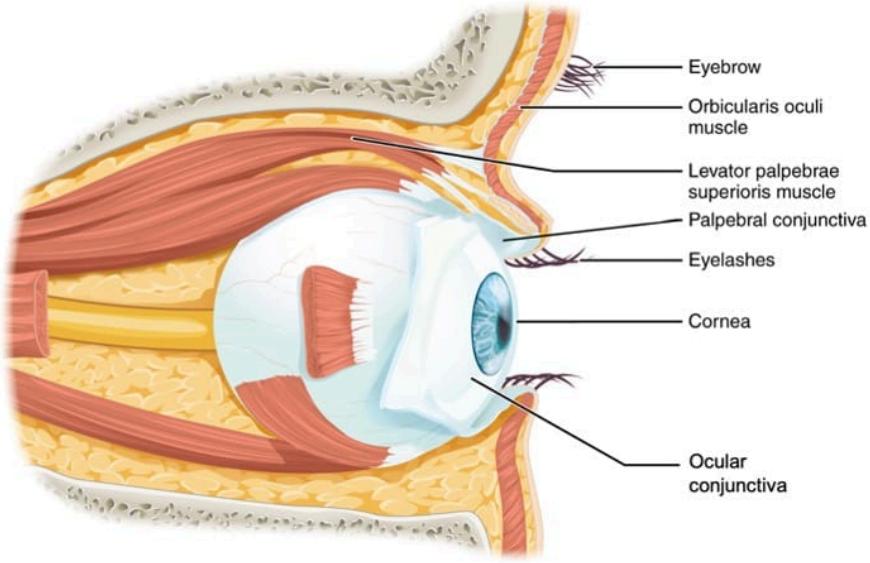


Figure 3.2.8: The eye is located within the orbit and surrounded by soft tissues such as muscles and the conjunctiva that protect and support its function. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.8.

The lacrimal apparatus associated with each eye is responsible for the production and drainage of lacrimal fluid (tears). The lacrimal fluid helps protect the eye by reducing friction of the eyelids, continuously cleaning the anterior surface of the eye, and preventing bacterial infection through the action of lysozyme. Tears are produced by the **lacrimal gland**, located within the superolateral depression of each orbit (Figure 3.2.9). Tears produced by this gland are dispersed on the external surface of the eye by the movement of the eyelids and then flow towards the medial surface of the eye. The **superior and inferior lacrimal puncta** (plural of "punctum") are small openings to drain the lacrimal fluid into channels called the **superior and inferior lacrimal canaliculi**. From there, the lacrimal fluid enters the **lacrimal sac** and drains into the **nasolacrimal duct** that delivers it into the nasal cavity where it mixes with the mucus.

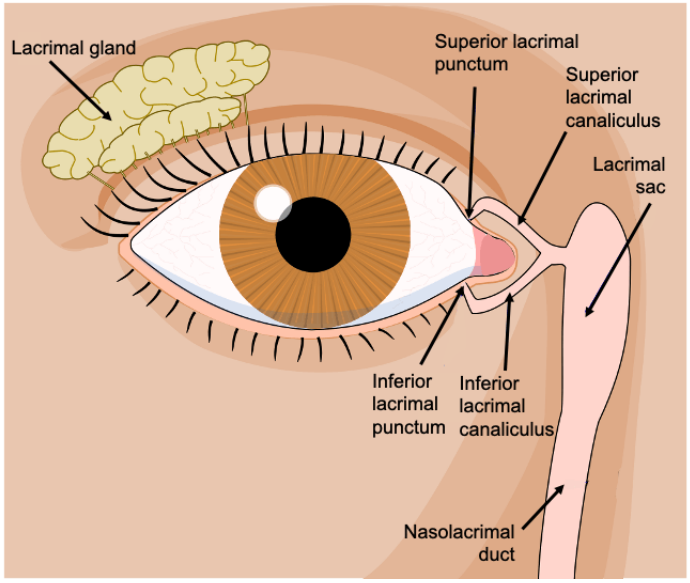


Figure 3.2.9: The lacrimal system produces lacrimal fluid that cleanses and moisturizes the anterior surface of the eye. Tears are produced by the lacrimal gland and dispersed on the external surface of the eye by the movement of the eyelids. The superior and inferior medial lacrimal puncta are small openings to drain the lacrimal fluid into channels called the superior and inferior lacrimal canaliculi. From there, the lacrimal fluid enters the lacrimal sac and drains into the nasolacrimal duct that delivers it into the nasal cavity where it mixes with the mucus. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.9.

Movement of the eye within the orbit is accomplished by the contraction of six **extraocular muscles** that originate from the bones of the orbit and insert into the surface of the eyeball (Figure 3.2.10). Four of the muscles are arranged at opposite sides of a cross around the eye and are named for those locations. They are the **superior rectus**, **medial rectus**, **inferior rectus**, and

lateral rectus and originate at the **common tendinous ring**. When each of these muscles contract, the eye to moves toward the contracting muscle. For example, when the superior rectus contracts, the eye rotates to look up. The **superior oblique** originates at the posterior orbit, near the origin of the four rectus muscles. However, the tendon of the superior oblique muscles threads through a pulley-like piece of cartilage known as the **trochlea**. The tendon inserts obliquely into the superior surface of the eye. The angle of the tendon through the trochlea means that contraction of the superior oblique abducts, depresses, and internally rotates (intorsion) the eye. The **inferior oblique** muscle originates from the floor of the orbit and inserts into the inferolateral surface of the eye. When it contracts, it abducts, elevates, and externally rotates (extorsion) the eye. Rotation of the eye by the two oblique muscles is necessary because the eye is not perfectly aligned on the sagittal plane. When the eye looks up or down, the eye must also rotate slightly to compensate for the superior rectus pulling at approximately a 20-degree angle, rather than straight up. The same is true for the inferior rectus, which is compensated by contraction of the inferior oblique. A seventh muscle in the orbit is the **levator palpebrae superioris**, which is responsible for elevating and retracting the upper eyelid, a movement that usually occurs in concert with elevation of the eye by the superior rectus (see Figure 3.2.10).

The extraocular muscles are innervated by three cranial nerves. The lateral rectus, which causes abduction of the eye, is innervated by the **abducens nerve (CN VI)**. The superior oblique is innervated by the **trochlear nerve (CN IV)**. All of the other muscles are innervated by the **oculomotor nerve (CN III)**, as is the levator palpebrae superioris. The motor nuclei of these cranial nerves connect to the brainstem, which coordinates eye movements.

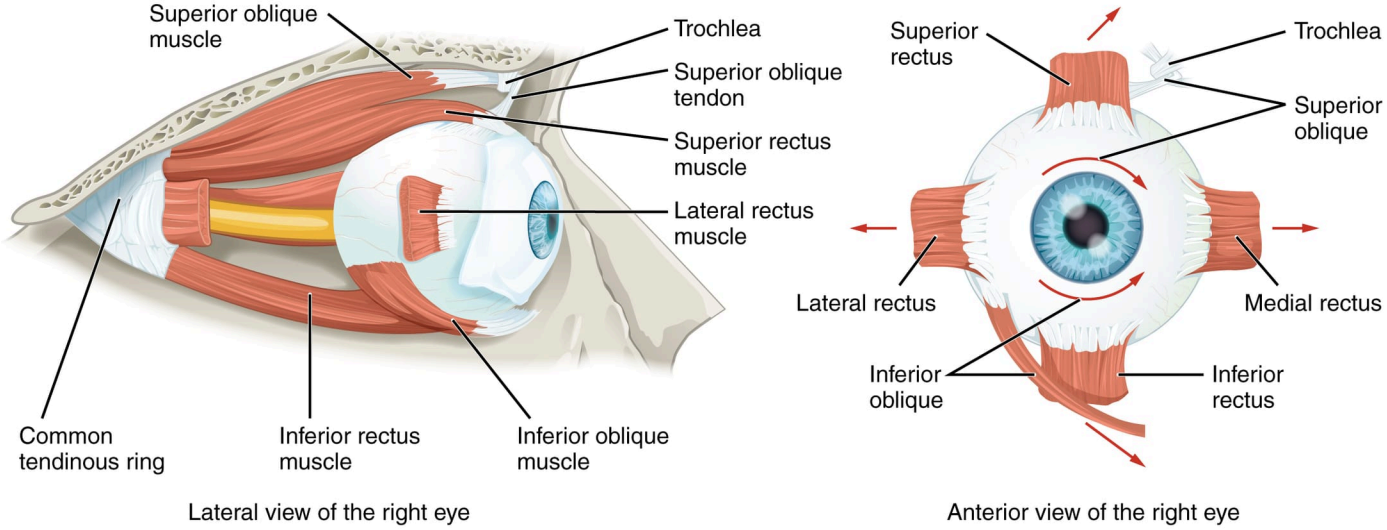


Figure 3.2.10: The extraocular muscles move the eye within the orbit. A lateral view of the right eye shows the common tendinous ring, where the four rectus muscles originate from. The superior oblique muscle originate from the posterior orbit and its tendon threads through the trochlea to insert into the superior surface of the eye. The inferior oblique muscle originate from the inferior portion of the orbit and inserts into the inferolateral surface of the eye. An anterior view of the right eye shows the movements of the extraocular muscles. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.10.

The eye itself is a hollow sphere composed of three layers of tissue. The outermost layer is the **fibrous tunic**, which includes the white sclera posteriorly and clear cornea anteriorly. The sclera accounts for five sixths of the surface of the eye, most of which is not visible, though humans are unique compared with many other species in having so much of the “white of the eye” visible (Figure 3.2.11). The transparent cornea covers the anterior tip of the eye and allows light to enter the eye. The middle layer of the eye is the **vascular tunic**, which is mostly composed of the choroid, ciliary body, and iris. The **choroid** is a layer of highly vascularized connective tissue that provides a blood supply to the eyeball. The choroid is posterior to the **ciliary body**, a muscular structure that is attached to the **lens** by **suspensory ligaments**. These two structures pull the lens, allowing it to focus light on the back of the eye. Overlaying the ciliary body, and visible in the anterior eye, is the **iris**—the colored part of the eye. The innermost layer of the eye is the **neural tunic**, or **retina**, which contains the nervous tissue responsible for photo reception. The retina consists of two layers: a pigmented layer and a neural layer. The **pigmented layer** is a layer of pigmented cells that absorb light after it passes through the retina and provides vital metabolic support for the photoreceptors of the retina. The **neural layer** consists of photoreceptors and blood vessels that supply the neural layer.

The eye is also divided into two cavities: the **anterior cavity** and the **posterior cavity**. The anterior cavity is the space between the cornea and lens, including the iris and ciliary body. It can be further divided into **anterior and posterior chambers** by the iris. These chambers are filled with a watery fluid called the **aqueous humor**. The posterior cavity is the space behind the lens that extends to the posterior side of the interior eyeball, where the retina is located. The posterior cavity is filled with a more

viscous fluid called the **vitreous humor**. Aqueous humor is continually produced at the **ciliary processes** of the ciliary body. The aqueous humor passes through the pupil into the anterior chamber and drains into the **scleral venous sinus** at the edge of the cornea.

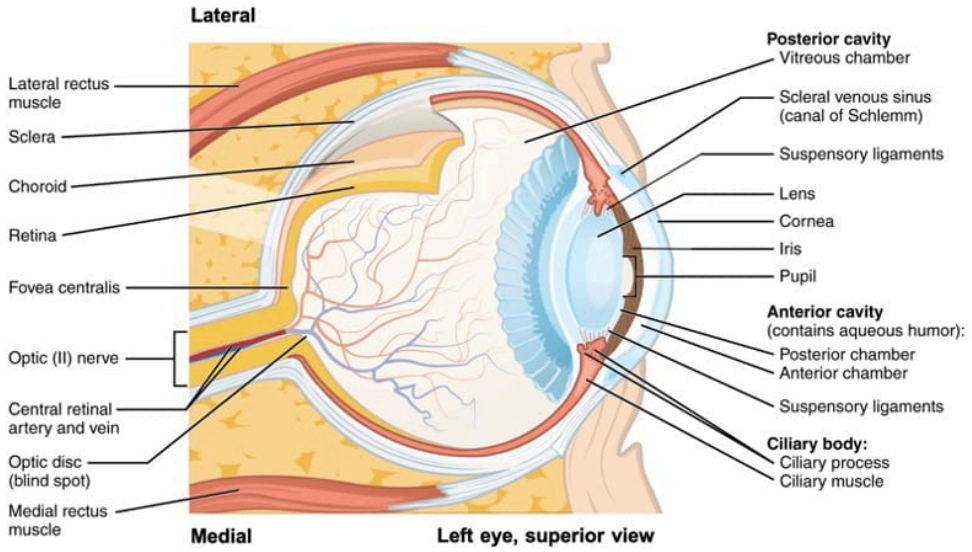


Figure 3.2.11: A superior view of the left eye shows the tissues and chambers composing the eye. The sphere of the eye can be divided into anterior and posterior cavities. The anterior cavity is the space between the cornea and the lens. It contains aqueous humor and is divided in anterior and posterior chambers by the iris, which forms the pupil. The posterior cavity is the space posterior to the lens and is filled with vitreous humor. The wall of the eye is composed of three layers: the fibrous tunic, vascular tunic, and neural tunic. The fibrous tunic is composed of the sclera and cornea. The vascular tunic is composed of the choroid, ciliary body and iris. The neural tunic is composed of the pigmented and neural layers. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.11.

The large interior space behind the lens is the **posterior cavity (vitreous chamber)**, which contains vitreous humor. At the front of the eye, the transparent **cornea** covers the eye and allows light to enter. Light then passes through the **pupil**, the opening in the center of the **iris**, before reaching the **lens**. The lens is suspended by **suspensory ligaments** attached to the **ciliary body**, which contains the **ciliary muscle** and **ciliary processes** that help control lens shape during focusing. The **anterior cavity**, which contains aqueous humor, is divided into the **anterior chamber** and **posterior chamber**. The diagram also labels the **scleral venous sinus (canal of Schlemm)**, which drains aqueous humor from the eye. Surrounding the eyeball are the **medial rectus** and **lateral rectus muscles**, which control horizontal eye movements. The image demonstrates the spatial relationships among the eye's optical, vascular, neural, and supporting structures.

The iris is composed of pigmented layers that give the eye its color and two layers of smooth muscle that opens or closes the **pupil**, which is the hole at the center of the eye that allows light to enter. The layers of smooth muscle are organized circularly into the **sphincter pupillae** and radially into the **dilator pupillae** (Figure 3.2.12). The sphincter pupillae constricts the pupil, while the dilator pupillae dilates the pupil. The iris constricts the pupil in response to bright light and dilates the pupil in response to dim light.

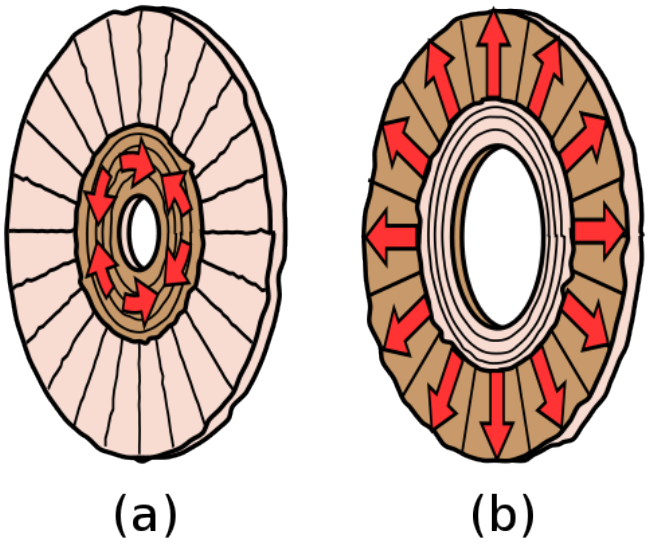


Figure 3.2.12: In (a), the sphincter pupillae is the inner circular muscular layer surrounding the pupil (hole in the center) and is responsible for constricting it. In (b), the dilator pupillae is the outer longitudinal muscular layer and is responsible for dilating the pupil. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.12.

The retina is composed of several layers and contains specialized cells for the initial processing of visual stimuli. The photoreceptors change their membrane electrical properties when stimulated by light energy. Light falling on the retina causes chemical changes to pigment molecules in the photoreceptors. There are two types of photoreceptors called rods and cones. **Cones** in human eyes are specialized in perceiving three different primary colors: red, green, and blue. **Rods** are sensitive to vision in low light conditions, for example in a darkened room. The change in electrical properties of these photoreceptors alters the amount of neurotransmitter that the photoreceptor cells release onto **bipolar cells** (see Figure 3.2.13). It is the bipolar cell in the retina that connects a photoreceptor to a **retinal ganglion cell (RGC)**. There, **amacrine cells** and **horizontal cells** additionally contribute to retinal processing before an action potential is produced by the RGC. The axons of RGCs, which lie at the innermost layer of the retina, collect at the **optic disc** and leave the eye as the **optic nerve**. Because these axons pass through the retina, there are no photoreceptors at the very back of the eye, where the optic nerve begins. This creates a "blind spot" in the retina, and a corresponding blind spot in our visual field. The visual information of the optic nerve will be transmitted through the **thalamus** and sent to the **primary visual cortex** of the occipital lobe.

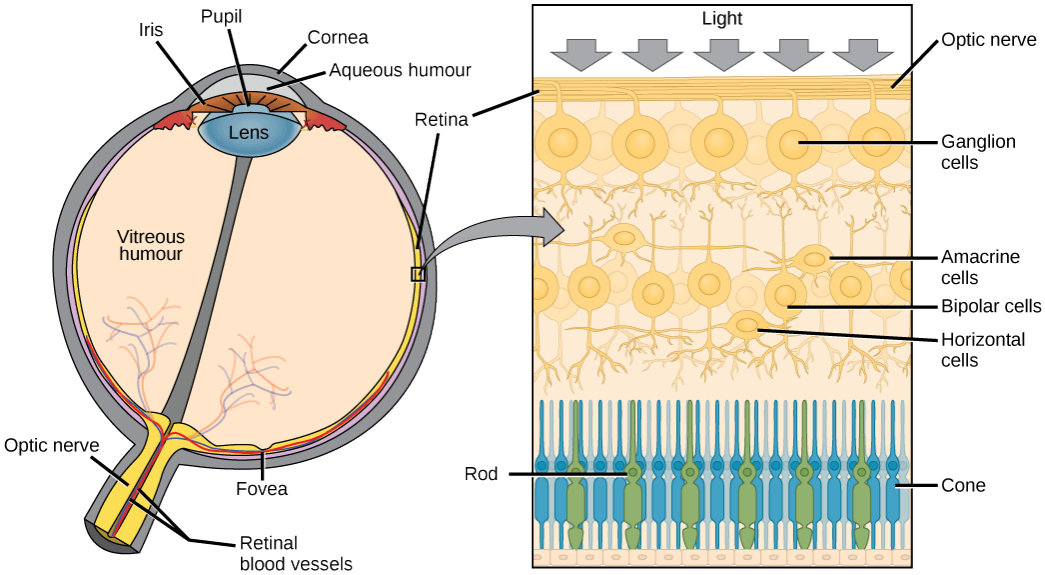


Figure 3.2.13: The retina is composed of several layers of cells that perceive changes in light and color. First, the light goes through the axons of ganglion cells that collect at the optic disc and leave the eye as the optic nerve. Ganglion cells synapse with bipolar cells. Amacrine and horizontal cells are scattered along the layer of bipolar cells. Bipolar cells synapse with the photoreceptors, composed of rods and cones. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.13.

Note that the photoreceptors in the retina (rods and cones) are located behind the axons, RGCs, bipolar cells, and retinal blood vessels. A significant amount of light is absorbed by these structures before the light reaches the photoreceptor cells. However, at the exact center of the retina is a small region known as the **macula lutea** at which center there is a depression called the **fovea** (Figure 3.2.14). In these regions, the retina lacks the supporting cells and blood vessels, and only contains cones. Therefore, **visual acuity**, or the sharpness of vision, is greatest at the fovea. As one moves in either direction from this central point of the retina, visual acuity drops significantly. In addition, each photoreceptor cell of the fovea is connected to a single RGC. Therefore, this RGC does not have to integrate inputs from multiple photoreceptors, which reduces the accuracy of visual transduction. Toward the edges of the retina, several photoreceptors converge on RGCs (through the bipolar cells) up to a ratio of 50 to 1. The difference in visual acuity between the fovea and peripheral retina is easily evidenced by looking directly at a word in the middle of this paragraph. The visual stimulus in the middle of the field of view falls on the fovea and is in the sharpest focus. Without moving your eyes off that word, notice that words at the beginning or end of the paragraph are not in focus. The images in your peripheral vision are focused by the peripheral retina, and have vague, blurry edges and words that are not as clearly identified. As a result, a large part of the neural function of the eyes is concerned with moving the eyes and head so that important visual stimuli are centered on the fovea.

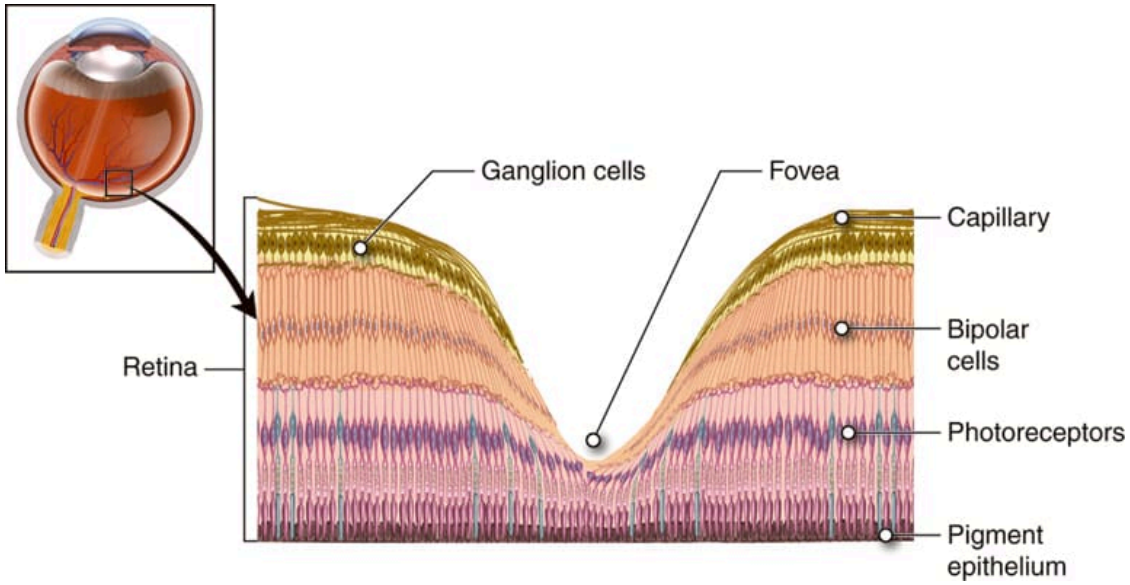


Figure 3.2.14: At the exact center of the retina is a region called the macula lutea where there is a depression called fovea. At the fovea there are no blood vessels, ganglion cells or bipolar cells to interrupt light reaching the receptor cells and only cones are present. Because more light passes to the receptor cells at the fovea, it is in this region that visual acuity is the greatest. (CC BY 4.0 via Hearing, Balance and Vision). Alternative text description of the Figure 3.2.14.

Concept Review

Hearing and balance belong to the special senses and its specialized organ is the ear. The ear is divided in three regions: external, middle and inner ear. In the external ear, the auricle is the fleshy structure that convey sounds into the auditory canal. At the end of the canal is the tympanic membrane that vibrates and transforms sound waves into mechanical waves. The middle ear consists of a space spanned by three small bones called ossicles (malleus, incus, and stapes) that conduct the mechanical waves. The middle ear is connected to the pharynx through the Eustachian (or auditory) tube, which helps equilibrate air pressure across the tympanic membrane. The inner ear is made of a bony labyrinth lined with a membranous labyrinth that separate tubes and spaces. The space between the two labyrinths is filled with a fluid called perilymph. Inside the membranous labyrinth the space is filled with endolymph. The inner ear is responsible for transforming mechanical waves into electrical signals, which are then sent to the brain through the vestibulocochlear nerve (CN VIII).

The cochlea is a spiral-shaped tube, divided into three compartments: the scala vestibuli, scala tympani and cochlear duct. All compartments are filled with endolymph. The scala vestibuli starts at the oval window, curves over the top of the cochlear duct and becomes the scala tympani, that returns to the base of the cochlea, travelling under the cochlear duct and ends at the round window. As vibrations of the ossicles travel through the oval window, the fluid of the scala vestibuli and scala tympani moves in a wave-like motion. The cochlear duct contains several organs of Corti, which tranduce the wave motion of the two scala into neural signals. The organs of Corti lie on top of the basilar membrane, which is the side of the cochlear duct located between the organs of Corti and the scala tympani. The organs of Corti contain hair cells, which are named for the hair-like stereocilia

extending from the cell's apical surfaces. The stereocilia extend up from the hair cells to the overlying tectorial membrane, which is attached medially to the organ of Corti. When the pressure waves from the scala move the basilar membrane, the tectorial membrane slides across the stereocilia. This bends the stereocilia either toward or away from the tallest member of each array, which causes an electrical signal to be generated.

The vestibule and the semicircular canals are responsible for the sense of equilibrium. The vestibule is composed of the utricle and saccule. Hair cells are located in maculae of the utricle and saccule. The stereocilia of the hair cells extend into a viscous gel called the otolithic membrane, on top of which is a layer of calcium carbonate crystals, called otoliths. The vestibule senses the linear acceleration of the head and gravity. When the head moves, the otoliths move and the otolithic membrane bends the stereocilia of the hair cells. Hair cells are also located in ampullae at the base of the three semicircular canals. One is oriented in the horizontal plane, whereas the other two are oriented in the vertical plane. The stereocilia of these hair cells extend into the cupula, a membrane that attaches to the top of the ampulla. The semicircular canals sense the head rotation. As the head rotates in a plane parallel to the semicircular canal, the fluid lags, deflecting the cupula in the direction opposite to the head movement, and bending the stereocilia of the hair cells.

Vision belongs to the special senses and its specialized organ is the eye. The bony orbits, eyelids, tarsal glands and lacrimal apparatus protect the eyes. The inner eyelid is a thin membrane known as the palpebral conjunctiva. The conjunctiva connects the eyelids to the eyeball. At the connection with the eyeball, a thin layer called the ocular conjunctiva forms a continuous layer on the external and anterior surface of the eye (except for the corneal region), superior to the white part of the eye, the sclera. The conjunctiva contains blood vessels and nerves that support the avascular sclera and detect foreign particles entering the eye.

The lacrimal apparatus is responsible for the production and drainage of lacrimal fluid (tears). Tears reduce friction of the eyelids, continuously clean the anterior surface of the eye, and prevent bacterial infection through the action of lysozyme. Tears are produced by the lacrimal gland, located within the superolateral depression of each orbit. The superior and inferior lacrimal puncta are small openings to drain the lacrimal fluid into channels called the superior and inferior lacrimal canaliculi. From there, the lacrimal fluid enters the lacrimal sac and drains into the nasolacrimal duct that delivers it into the nasal cavity where it mixes with the mucus.

There are six extraocular muscles innervated by the abducens nerve (CN VI), trochlear nerve (CN IV) and oculomotor nerve (CN III). The superior rectus elevates the eye, the medial rectus adducts the eye, the inferior rectus depresses the eye, the lateral rectus abducts the eye. The superior oblique abducts, depresses, and internally rotates (intorsion) the eye. The inferior oblique abducts, elevates, and externally rotates (extorsion) the eye.

The eye walls are made by three layers of tissue called tunics. The fibrous tunic includes the white sclera and the clear cornea. The vascular tunic composed of the choroid, ciliary body attached to the lens by suspensory ligaments and iris (colored part of the eye). The iris is composed of pigmented layers and two layers of smooth muscle called the sphincter pupillae and dilator pupillae, that open and close the pupil. The neural tunic (or retina) includes a pigmented layer and a neural layer that contains the photoreceptors. The eye is divided in two cavities: the anterior cavity between the cornea and lens, filled with aqueous humor, and the posterior cavity behind the lens, filled with vitreous humor. The retina is composed of several layers of cells, connected to each other, that support vision transduction. The photoreceptors are rods for light vision and cones for color vision. Retinal ganglion cells (RGCs) have axons that collect at the optic disc and leave the eye as the optic nerve (CN II). At the exact center of the retina is a small area known as the macula lutea with a depression in the middle called the fovea. At the fovea, the retina lacks the supporting cells and blood vessels, and only contains cones. Therefore, visual acuity, or the sharpness of vision, is greatest at the fovea.

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3.3: Integration of Somatic Functions

Sensory Nerves

Once any sensory cell transduces a stimulus into a nerve impulse, that impulse has to travel along axons to reach the CNS. In many of the special senses, the axons leaving the sensory receptors have a **topographical** arrangement, meaning that the location of the sensory receptor relates to the location of the axon in the nerve. For example, in the retina, axons from RGCs in the fovea are located at the center of the optic nerve, where they are surrounded by axons from the more peripheral RGCs.

Spinal Nerves

Generally, **spinal nerves** contain afferent axons from sensory receptors in the periphery, such as from the skin, mixed with efferent axons traveling to the muscles or other effector organs. As the spinal nerve nears the spinal cord, it splits into dorsal and ventral roots. The dorsal root contains only the axons of sensory neurons, whereas the ventral roots contain only the axons of the motor neurons. Some of the branches will synapse with local neurons in the dorsal root ganglion, posterior (dorsal) horn, or even the anterior (ventral) horn, at the level of the spinal cord where they enter. Other branches will travel a short distance up or down the spine to interact with neurons at other levels of the spinal cord. A branch may also turn into the posterior (dorsal) column of the white matter to connect with the brain. Typically, spinal nerve systems that connect to the brain are **contralateral**, in that the right side of the body is connected to the left side of the brain and the left side of the body to the right side of the brain.

Cranial Nerves

Cranial nerves convey specific sensory information from the head and neck directly to the brain. For sensations below the neck, the right side of the body is connected to the left side of the brain and the left side of the body to the right side of the brain. Whereas spinal information is contralateral, cranial nerve systems are mostly **ipsilateral**, meaning that a cranial nerve on the right side of the head is connected to the right side of the brain. Some cranial nerves contain only sensory axons, such as the olfactory, optic, and vestibulocochlear nerves. Other cranial nerves contain both sensory and motor axons, including the trigeminal, facial, glossopharyngeal, and vagus nerves (however, the vagus nerve is not associated with the somatic nervous system). The general senses of somatosensation for the face travel through the trigeminal system.

There are few cranial nerves that are not ipsilateral: optic nerve (CN II), trochlear nerve (CN IV), facial nerve (CN VII), hypoglossal nerves (CN XII). Of these, the optic nerve and facial nerve carry sensory information, while the others carry motor information only.

The connections of the **optic nerve** are more complicated than those of other cranial nerves. Instead of the connections being between each eye and the brain, visual information is segregated between the left and right sides of the visual field. In addition, some of the information from one side of the visual field projects to the opposite side of the brain. Within each eye, the axons projecting from the medial side of the retina cross at the **optic chiasm** and create new bundles called the **optic tract** which synapse onto the **thalamus** and extend to **occipital lobes**. For example, the axons from the medial retina of the left eye cross over to the right side of the brain at the optic chiasm. However, within each eye, the axons projecting from the lateral side of the retina do not cross. For example, the axons from the lateral retina of the right eye project back to the right side of the brain. Therefore the left field of view of each eye is processed on the right side of the brain, whereas the right field of view of each eye is processed on the left side of the brain (Figure 3.3.1).

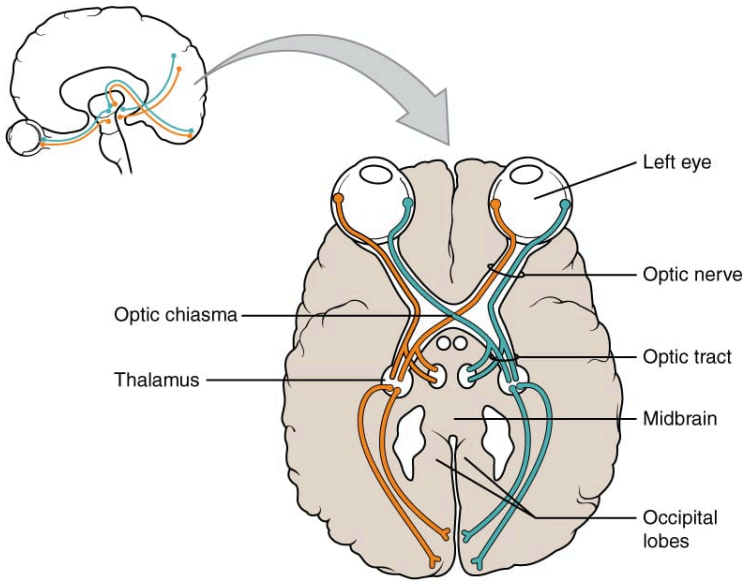


Figure 3.3.1: Optic nerves carry visual information to the brain. Axons within the optic nerves project both contralaterally and ipsilaterally, depending on the visual field. Contralateral visual field information from the lateral retina projects to the ipsilateral brain, whereas ipsilateral visual field information has to cross at the optic chiasm to reach the opposite side of the brain. (CC BY 4.0; via LibreTexts). Alternative text description of the Figure 3.3.1.

Disorders Of The Vision: Bilateral Hemianopia

A unique clinical presentation that relates to this anatomic arrangement is the loss of lateral peripheral vision, known as bilateral hemianopia. This is different from "tunnel vision" because the superior and inferior peripheral fields are not lost. Visual field deficits can be disturbing for a patient, but in this case, the cause is not within the visual system itself. A growth of the pituitary gland presses against the optic chiasm and interferes with signal transmission. However, the axons projecting to the same side of the brain are unaffected. Therefore, the patient loses the outermost areas of their field of vision and cannot see objects to their right and left.

Sensory Pathways

Specific regions of the CNS coordinate different somatic processes using sensory inputs and motor outputs of peripheral nerves. A simple case is a reflex caused by a synapse between a dorsal sensory neuron axon and a motor neuron in the ventral horn. More complex arrangements are possible to integrate peripheral sensory information with higher processes. The important regions of the CNS that play a role in somatic processes can be separated into the spinal cord, brainstem, diencephalon, and cerebrum (cortex and subcortical structures).

Spinal Cord and Brainstem

A sensory pathway that carries peripheral sensations to the brain is referred to as an **ascending pathway**, or ascending tract. Various sensory modalities follow specific pathways through the CNS. Tactile and other somatosensory stimuli activate receptors in the skin, muscles, tendons, and joints throughout the entire body. However, the somatosensory pathways are divided into two separate systems on the basis of the location of the receptor neurons. Somatosensory stimuli from below the neck pass along the sensory pathways of the spinal cord, whereas somatosensory stimuli from the head and neck travel through the cranial nerves—specifically, the trigeminal system.

The **dorsal column system** (sometimes referred to as the dorsal column–medial lemniscus) and the **spinothalamic tract** are two major pathways that bring sensory information to the brain (Figure 3.3.2). The sensory pathways in each of these systems are composed of three successive neurons.

The dorsal column system begins with the axon of a dorsal root ganglion neuron entering the dorsal root and joining the dorsal column white matter in the spinal cord. As axons of this pathway enter the dorsal column, they take on a positional arrangement

so that axons from lower levels of the body position themselves medially, whereas axons from upper levels of the body position themselves laterally. The dorsal column is separated into two component tracts, the **fasciculus gracilis** that contains axons from the legs and lower body, and the **fasciculus cuneatus** that contains axons from the upper body and arms.

The axons in the dorsal column terminate in the nuclei of the medulla, where each synapses with the second neuron in their respective pathway. The **nucleus gracilis** is the target of fibers in the fasciculus gracilis, whereas the **nucleus cuneatus** is the target of fibers in the fasciculus cuneatus. The second neuron in the system projects from one of the two nuclei and then **decussates**, or crosses the midline, in the medulla. These axons then continue to ascend the brainstem as a bundle called the **medial lemniscus**. These axons terminate in the thalamus, where each synapses with the third neuron in their respective pathway. The third neuron in the system projects its axons to the postcentral gyrus of the cerebral cortex, where somatosensory stimuli are initially processed and the conscious perception of the stimulus occurs.

The spinothalamic tract also begins with neurons in a dorsal root ganglion. These neurons extend their axons to the dorsal horn, where they synapse with the second neuron in their respective pathway. The name “spinothalamic” comes from this second neuron, which has its cell body in the spinal cord gray matter and connects to the thalamus. Axons from these second neurons then decussate within the spinal cord and ascend to the brain and enter the thalamus, where each synapses with the third neuron in its respective pathway. The neurons in the thalamus then project their axons to the spinothalamic tract, which synapses in the postcentral gyrus of the cerebral cortex.

These two systems are similar in that they both begin with dorsal root ganglion cells, as with most general sensory information. The dorsal column system is primarily responsible for touch sensations and proprioception, whereas the spinothalamic tract pathway is primarily responsible for pain and temperature sensations. Another similarity is that the second neurons in both of these pathways project across the midline to the other side of the brain or spinal cord, so that sensation is perceived contralaterally. In the dorsal column system, this decussation takes place in the brainstem; in the spinothalamic pathway, it takes place in the spinal cord at the same spinal cord level at which the information entered. The third neurons in the two pathways are essentially the same. In both, the second neuron synapses in the thalamus, and the thalamic neuron projects to the somatosensory cortex.

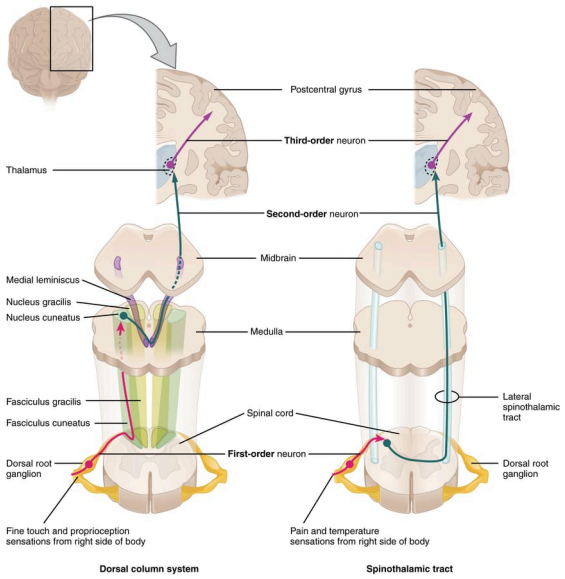


Figure 3.3.2: The dorsal column–medial lemniscus pathway and spinothalamic tract are the principal ascending pathways that transmit somatosensory information from the periphery to the cerebral cortex. The dorsal column pathway carries fine touch and proprioceptive information through the fasciculi gracilis and cuneatus to the medulla, where fibers decussate and ascend to the thalamus before projecting to the postcentral gyrus. The spinothalamic tract carries pain and temperature information; fibers decussate within the spinal cord and ascend through the brainstem to the thalamus, which relays signals to the postcentral gyrus. The figure highlights the first-, second-, and third-order neurons and the distinct sites of decussation for each pathway. (CC BY 4.0; via LibreTexts). Alternative text description of the Figure 3.3.2.

The trigeminal pathway carries somatosensory information from the face, head, mouth, and nasal cavity. As with the previously discussed nerve tracts, the sensory pathways of the trigeminal pathway each involve three successive neurons. First, axons from the trigeminal ganglion enter the brainstem at the level of the pons. These axons project to one of three locations. The **spinal trigeminal nucleus** of the medulla receives information similar to that carried by spinothalamic tract, such as pain and temperature sensations. Other axons go to either the **chief sensory nucleus** in the pons or the **mesencephalic nuclei** in the

midbrain. These nuclei receive information like that carried by the dorsal column system, such as touch, pressure, vibration, and proprioception. Axons from the second neuron decussate and ascend to the thalamus along the trigeminothalamic tract. In the thalamus, each axon synapses with the third neuron in its respective pathway. Axons from the third neuron then project from the thalamus to the primary somatosensory cortex of the cerebrum.

The sensory pathway for gustation travels along the facial and glossopharyngeal cranial nerves, which synapse with neurons of the **solitary nucleus** in the brainstem. Axons from the solitary nucleus then project to the **ventral posterior nucleus** of the thalamus. Finally, axons from the ventral posterior nucleus project to the gustatory cortex of the cerebral cortex, where taste is processed and consciously perceived.

The sensory pathway for audition travels along the vestibulocochlear nerve, which synapses with neurons in the **cochlear nuclei** of the superior medulla. Within the brainstem, input from either ear is combined to extract location information from the auditory stimuli. Whereas the initial auditory stimuli received at the cochlea strictly represent the frequency—or pitch—of the stimuli, the locations of sounds can be determined by comparing information arriving at both ears. Auditory processing continues on to a nucleus in the midbrain called the **inferior colliculus**. Axons from the inferior colliculus project to two locations, the thalamus and the **superior colliculus**. The **medial geniculate nucleus** of the thalamus receives the auditory information and then projects that information to the auditory cortex in the temporal lobe of the cerebral cortex. The superior colliculus receives input from the visual and somatosensory systems, as well as the ears, to initiate stimulation of the muscles that turn the head and neck toward the auditory stimulus.

Balance is coordinated through the vestibular system, the nerves of which are composed of axons from the vestibular ganglion that carries information from the utricle, saccule, and semicircular canals. The system contributes to controlling head and neck movements in response to vestibular signals. An important function of the vestibular system is coordinating eye and head movements to maintain visual attention. Most of the axons terminate in the **vestibular nuclei** of the medulla. Some axons project from the vestibular ganglion directly to the cerebellum, with no intervening synapse in the vestibular nuclei. The cerebellum is primarily responsible for initiating movements on the basis of equilibrium information. Neurons in the vestibular nuclei project their axons to targets in the brainstem. One target is the reticular formation, which influences respiratory and cardiovascular functions in relation to body movements. A second target of the axons of neurons in the vestibular nuclei is the spinal cord, which initiates the spinal reflexes involved with posture and balance. To assist the visual system, fibers of the vestibular nuclei project to the oculomotor, trochlear, and abducens nuclei to influence signals sent along the cranial nerves.

The sensory pathway for vision starts with the optic tract. The optic tract has three major targets, two in the diencephalon and one in the midbrain. The connection between the eyes and diencephalon is demonstrated during development, in which the neural tissue of the retina differentiates from that of the diencephalon by the growth of the secondary vesicles. The connections of the retina into the CNS are a holdover from this developmental association. The majority of the connections of the optic tract are to the thalamus—specifically, the **lateral geniculate nucleus**. Axons from this nucleus then project to the visual cortex of the cerebrum, located in the occipital lobe. Another target of the optic tract is the superior colliculus. In addition, a very small number of retinal ganglion cells (RGCs) axons project from the optic chiasm to the **suprachiasmatic nucleus** of the hypothalamus. These RGCs are photosensitive, in that they respond to the presence or absence of light. Unlike the photoreceptors, however, these photosensitive RGCs cannot be used to perceive images. By simply responding to the absence or presence of light, these RGCs can send information about day length. The perceived proportion of sunlight to darkness establishes the **circadian rhythm** of our bodies, allowing certain physiological events to occur at approximately the same time every day.

Diencephalon

The diencephalon is beneath the cerebrum and includes the thalamus and hypothalamus. In the somatic nervous system, the **thalamus** is an important relay for communication between the cerebrum and the rest of the nervous system. The **hypothalamus** has both somatic and autonomic functions. In addition, the hypothalamus communicates with the limbic system, which controls emotions and memory functions.

Sensory input to the thalamus comes from most of the special senses and ascending somatosensory tracts. Each sensory system is relayed through a particular nucleus in the thalamus. The thalamus is a required transfer point for most sensory tracts that reach the cerebral cortex, where conscious sensory perception begins. The one exception to this rule is the olfactory system. The olfactory tract axons from the olfactory bulb project directly to the cerebral cortex, along with the limbic system and hypothalamus.

The thalamus is a collection of several nuclei that can be categorized into three anatomical groups. White matter running through the thalamus defines the three major regions of the thalamus, which are an **anterior nucleus**, a **medial nucleus**, and a **lateral group of nuclei**. The anterior nucleus serves as a relay between the hypothalamus and the emotion and memory-producing limbic

system. The medial nuclei serve as a relay for information from the limbic system and basal ganglia to the cerebral cortex. This allows memory creation during learning, but also determines alertness. The special and somatic senses connect to the lateral nuclei, where their information is relayed to the appropriate sensory cortex of the cerebrum.

Cerebrum

As described earlier, many of the sensory axons are positioned in the same way as their corresponding receptor cells in the body. This allows identification of the position of a stimulus on the basis of which receptor cells are sending information. The cerebral cortex also maintains this **sensory topography** in the particular areas of the cortex that correspond to the position of the receptor cells. This mapping is often depicted using a sensory homunculus.

Likewise, the topographic relationship between the retina and the visual cortex is maintained throughout the visual pathway. The visual field is projected onto the two retinae, as described above, with sorting at the optic chiasm. The right peripheral visual field falls on the medial portion of the right retina and the lateral portion of the left retina. The right medial retina then projects across the midline through the optic chiasm. This results in the right visual field being processed in the left visual cortex. Likewise, the left visual field is processed in the right visual cortex. Though the chiasm is helping to sort right and left visual information, superior and inferior visual information is maintained topographically in the visual pathway. Light from the superior visual field falls on the inferior retina, and light from the inferior visual field falls on the superior retina. This topography is maintained such that the superior region of the visual cortex processes the inferior visual field and vice versa. Therefore, the visual field information is inverted and reversed as it enters the visual cortex—up is down, and left is right (Figure 3.3.3). However, the cortex processes the visual information such that the final conscious perception of the visual field is correct. The topographic relationship is evident in that information from the foveal region of the retina is processed in the center of the primary visual cortex. Information from the peripheral regions of the retina are correspondingly processed toward the edges of the visual cortex. Similar to the exaggerations in the sensory homunculus of the somatosensory cortex, the foveal-processing area of the visual cortex is disproportionately larger than the areas processing peripheral vision.

In an experiment performed in the 1960s, subjects wore prism glasses so that the visual field was inverted before reaching the eye. On the first day of the experiment, subjects would duck when walking up to a table, thinking it was suspended from the ceiling. However, after a few days of acclimation, the subjects behaved as if everything were represented correctly. Therefore, the visual cortex is somewhat flexible in adapting to the information it receives from our eyes.

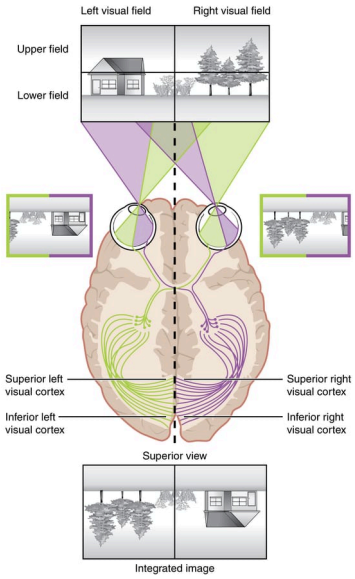


Figure 3.3.3: The visual field projects onto the retina through the lenses and falls on the retinae as an inverted, and reversed image where left is right and superior is inferior, and vice versa. The topography of this image is maintained as the visual information travels through the visual pathway to the visual cortex of the occipital lobe, resulting into an integrated image being inverted and reversed. (CC BY 4.0; via LibreTexts).
Alternative text description of the Figure 3.3.3.

The cortex has been described as having specific regions that are responsible for processing specific information; there is the visual cortex, somatosensory cortex, gustatory cortex, etc. However, our experience of these senses is not divided. Instead, we experience what can be referred to as a seamless percept. Our perceptions of the various sensory modalities—though distinct in

their content—are integrated by the brain so that we experience the world as a continuous whole.

In the cerebral cortex, sensory processing begins at the **primary sensory cortex**, then proceeds to an **association area**, and finally, into a **multimodal integration area**. For example, the visual pathway projects from the retinae through the thalamus to the primary visual cortex in the occipital lobe. This area is primarily in the medial wall within the longitudinal fissure. Here, visual stimuli begin to be recognized as basic shapes. Edges of objects are recognized and built into more complex shapes. Also, inputs from both eyes are compared to extract depth information. Because of the overlapping field of view between the two eyes, the brain can begin to estimate the distance of stimuli based on **binocular depth cues**.

Concept Review

The nerves that convey sensory information from the periphery to the CNS are either spinal nerves, connected to the spinal cord, or cranial nerves, connected to the brain. Spinal nerves have mixed populations of fibers; some are motor fibers and some are sensory. The sensory fibers connect to the spinal cord through the dorsal root, which is attached to the dorsal root ganglion. Sensory information from the body that is conveyed through spinal nerves will project to the opposite side of the brain to be processed by the cerebral cortex. The cranial nerves can be strictly sensory fibers, such as the olfactory, optic, and vestibulocochlear nerves, or mixed sensory and motor nerves, such as the trigeminal, facial, glossopharyngeal, and vagus nerves. The cranial nerves are connected to the same side of the brain from which the sensory information originates.

Sensory input to the brain enters through pathways that travel through either the spinal cord (for somatosensory input from the body) or the brainstem (for everything else, except the visual and olfactory systems) to reach the diencephalon. In the diencephalon, sensory pathways reach the thalamus. This is necessary for all sensory systems to reach the cerebral cortex, except for the olfactory system that is directly connected to the frontal and temporal lobes.

The two major tracts in the spinal cord, originating from sensory neurons in the dorsal root ganglia, are the dorsal column system and the spinothalamic tract. The major differences between the two are in the type of information that is relayed to the brain and where the tracts decussate. The dorsal column system primarily carries information about touch and proprioception and crosses the midline in the medulla. The spinothalamic tract is primarily responsible for pain and temperature sensation and crosses the midline in the spinal cord at the level at which it enters. The trigeminal nerve adds similar sensation information from the head to these pathways.

The auditory pathway passes through multiple nuclei in the brainstem in which additional information is extracted from the basic frequency stimuli processed by the cochlea. Sound localization is made possible through the activity of these brainstem structures. The vestibular system enters the brainstem and influences activity in the cerebellum, spinal cord, and cerebral cortex.

The visual pathway segregates information from the two eyes so that one half of the visual field projects to the other side of the brain. Within visual cortical areas, the perception of the stimuli and their location is passed along two streams, one ventral and one dorsal. The ventral visual stream connects to structures in the temporal lobe that are important for long-term memory formation. The dorsal visual stream interacts with the somatosensory cortex in the parietal lobe, and together they can influence the activity in the frontal lobe to generate movements of the body in relation to visual information.

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3.4: Somatic Motor Responses

Cortical Responses

The defining characteristic of the somatic nervous system is that it controls skeletal muscles. Somatic senses inform the nervous system about the external environment, but the response to that is through voluntary muscle movement. The term “voluntary” suggests that there is a conscious decision to make a movement. However, some aspects of the somatic system use voluntary muscles without conscious control. One example is the ability of our breathing to switch to unconscious control while we are focused on another task. However, the muscles that are responsible for the basic process of breathing are also utilized for speech, which is entirely voluntary.

The information on sensory stimuli registered through receptor cells is relayed to the CNS along ascending pathways. In the cerebral cortex, the initial processing of sensory perception can lead to the incorporation of sensory perceptions into memory, but more importantly, it leads to a response. The completion of cortical processing through the primary, associative, and integrative sensory areas initiates a similar progression of motor processing, usually in different cortical areas.

Prefrontal Cortex

Whereas the sensory cortical areas are located in the occipital, temporal, and parietal lobes, motor functions are largely controlled by the frontal lobe. The most anterior regions of the frontal lobe—the prefrontal areas—are important for **executive functions**, which are cognitive functions that lead to goal-directed behaviors. These higher cognitive processes include **working memory** that can help organize and represent information that is not in the immediate environment. The prefrontal lobe is responsible for aspects of attention, such as inhibiting distracting thoughts and actions so that a person can focus on a goal and direct behavior toward achieving that goal. The functions of the prefrontal cortex are integral to the personality of an individual, because it is largely responsible for what a person intends to do and how they accomplish those plans.

Secondary Motor Cortices

In generating motor responses, the executive functions of the prefrontal cortex will need to initiate actual movements. One way to define the prefrontal area is any region of the frontal lobe that does not elicit movement when electrically stimulated. These are primarily in the anterior part of the frontal lobe. The regions of the frontal lobe that remain are the regions of the cortex that produce movement. The prefrontal areas project into the secondary motor cortices, which include the **premotor cortex** and the **supplemental motor area**. The premotor area aids in controlling movements of the core muscles to maintain posture during movement, whereas the supplemental motor area is hypothesized to be responsible for planning and coordinating movement. The supplemental motor area also manages sequential movements that are based on prior experience (that is, learned movements). Neurons in these areas are most active leading up to the initiation of movement. For example, these areas might prepare the body for the movements necessary to drive a car in anticipation of a traffic light changing.

Adjacent to these two regions are two specialized motor planning centers. The **frontal eye fields** are responsible for moving the eyes in response to visual stimuli. There are direct connections between the frontal eye fields and the superior colliculus. Also, anterior to the premotor cortex and primary motor cortex is **Broca’s area**. This area is responsible for controlling movements of the structures of speech production.

Primary Motor Cortex

The primary motor cortex is located in the precentral gyrus of the frontal lobe. The primary motor cortex receives input from several areas that aid in planning movement, and its principle output stimulates spinal cord neurons to stimulate skeletal muscle contraction. The primary motor cortex is arranged in a similar fashion to the primary somatosensory cortex, in that it has a topographical map of the body, creating a motor homunculus. The neurons responsible for musculature in the feet and lower legs are in the medial wall of the precentral gyrus, with the thighs, trunk, and shoulder at the crest of the longitudinal fissure. The hand and face are in the lateral face of the gyrus. Also, the relative space allotted for the different regions is exaggerated in muscles that have greater enervation. The greatest amount of cortical space is given to muscles that perform fine, agile movements, such as the muscles of the fingers and the lower face. The “power muscles” that perform coarser movements, such as the buttock and

back muscles, occupy much less space on the motor cortex.

Descending Pathways

The motor output from the cortex descends into the brainstem and to the spinal cord to control the musculature through motor neurons. Neurons located in the primary motor cortex, named **Betz cells**, are large cortical neurons that synapse with lower motor neurons in the spinal cord or the brainstem. The two descending pathways traveled by the axons of Betz cells are the **corticospinal tract** and the **corticobulbar tract**. Both tracts are named for their origin in the cortex and their targets—either the spinal cord or the brainstem (the term “bulbar” refers to the brainstem as the bulb, or enlargement, at the top of the spinal cord).

These two descending pathways are responsible for the conscious or voluntary movements of skeletal muscles. Any motor command from the primary motor cortex is sent down the axons of the Betz cells to activate upper motor neurons in either the cranial motor nuclei or in the ventral horn of the spinal cord. The axons of the corticobulbar tract are ipsilateral, meaning they project from the cortex to the cranial motor nucleus on the same side of the nervous system. The corticobulbar tract controls the movement of muscles in the face, head and neck. Conversely, the axons of the corticospinal tract are largely contralateral, meaning that they cross the midline of the brainstem or spinal cord and synapse on the opposite side of the body. The corticospinal tract controls movement of muscles of limbs and trunk. Therefore, the right motor cortex of the cerebrum controls muscles on the left arm, for example, and vice versa.

The corticospinal tract descends from the cortex through the deep white matter of the cerebrum. It then passes between the caudate nucleus and putamen of the basal nuclei as a bundle called the **internal capsule**. The tract then passes through the midbrain as the **cerebral peduncles**, after which it burrows through the pons. Upon entering the medulla, the tracts make up the large white matter tract referred to as the **pyramids** (Figure 3.4.1). The defining landmark of the medullary-spinal border is the **pyramidal decussation**, which is where most of the fibers in the corticospinal tract cross over to the opposite side of the brain. At this point, the tract separates into two parts, which have control over different domains of the musculature.

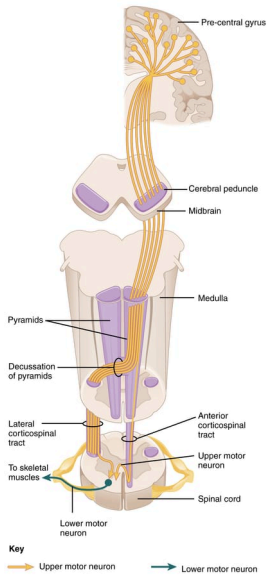


Figure 3.4.1: The major descending tract that controls skeletal muscle movements is the corticospinal tract. It is composed of two neurons, the upper motor neuron and the lower motor neuron. The upper motor neuron has its cell body in the primary motor cortex located in the precentral gyrus of the frontal lobe. Its axon descend through the cerebral peduncle of the midbrain and crosses the midline at the medulla through the decussation of pyramids. Traveling down through the anterior and lateral corticospinal tracts, it then synapses on the lower motor neuron, which is in the ventral horn of the spinal cord and projects to the skeletal muscle in the periphery. (CC BY 4.0; via LibreTexts).
Alternative text description of the Figure 3.4.1.

Appendicular Control

The **lateral corticospinal tract** is composed of the fibers that cross the midline at the pyramidal decussation (see Figure 3.4.1). The axons cross over from the anterior position of the pyramids in the medulla to the lateral column of the spinal cord. These axons are responsible for controlling appendicular muscles.

This influence over the appendicular muscles means that the lateral corticospinal tract is responsible for moving the muscles of the arms and legs. The lower cervical spinal cord and the lumbar spinal cord both have wider ventral horns, representing the greater number of muscles controlled by these motor neurons. The **cervical enlargement** is particularly large because there is greater control over the fine musculature of the upper limbs, particularly of the fingers. The **lumbar enlargement** is not as significant in appearance because there is less fine motor control of the lower limbs.

Axial Control

The **anterior corticospinal tract** is responsible for controlling the muscles of the body trunk (see Figure 3.4.1). These axons do not decussate in the medulla. Instead, they remain in an anterior position as they descend the brainstem and enter the spinal cord. These axons then travel to the spinal cord level at which they synapse with a lower motor neuron. Upon reaching the appropriate level, the axons decussate, entering the ventral horn on the opposite side of the spinal cord from which they entered. In the ventral horn, these axons synapse with their corresponding lower motor neurons. The lower motor neurons are located in the medial regions of the ventral horn, because they control the axial muscles of the trunk.

Because movements of the body trunk involve both sides of the body, the anterior corticospinal tract is not entirely contralateral. Some collateral branches of the tract will project into the ipsilateral ventral horn to control synergistic muscles on that side of the body, or to inhibit antagonistic muscles through interneurons within the ventral horn. Through the influence of both sides of the body, the anterior corticospinal tract can coordinate postural muscles in broad movements of the body. These coordinating axons in the anterior corticospinal tract are often considered bilateral, as they are both ipsilateral and contralateral.

Extrapyramidal System

Other descending connections between the brain and the spinal cord are called the **extrapyramidal system**. The name comes from the fact that this system is outside the corticospinal pathway, which includes the pyramids in the medulla. A few pathways originating from the brainstem contribute to this system.

The **tectospinal tract** projects from the midbrain to the spinal cord and is important for postural movements that are driven by the superior colliculus (Figure 3.4.2). The name of the tract comes from an alternate name for the superior colliculus, which is the tectum. The **reticulospinal tract** connects the reticular system, a diffuse region of gray matter in the brainstem, with the spinal cord. This tract influences trunk and proximal limb muscles related to posture and locomotion. The reticulospinal tract also contributes to muscle tone and influences autonomic functions. The **vestibulospinal tract** connects the brainstem nuclei of the vestibular system with the spinal cord. This allows posture, movement, and balance to be modulated on the basis of equilibrium information provided by the vestibular system. The pathways of the extrapyramidal system are influenced by subcortical structures. For example, connections between the secondary motor cortices and the extrapyramidal system modulate spine and cranium movements. The basal nuclei, which are important for regulating movement initiated by the CNS, influence the extrapyramidal system as well as its thalamic feedback to the motor cortex.

Figure 3.4.2 summarizes both ascending and descending pathways.

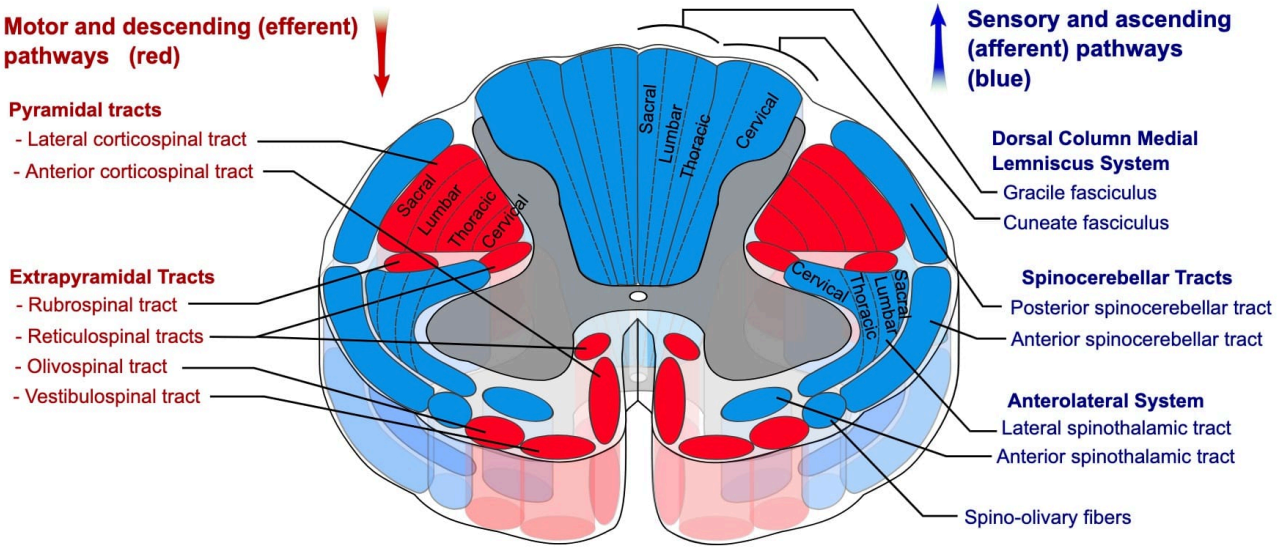


Figure 3.4.2: A cross section of the spinal cord shows the sensory ascending tracts and motor descending tracts. Ascending sensory pathways include the dorsal column medial lemniscus system, the spinocerebellar tracts and anterolateral system. Descending motor pathways include pyramidal tracts and extrapyramidal tracts. (CC BY 4.0; via LibreTexts). Alternative text description of the Figure 3.4.2.

Ventral Horn Output

The somatic nervous system provides output strictly to skeletal muscles. The lower motor neurons, which are responsible for the contraction of these muscles, are found in the ventral horn of the spinal cord. These large, multipolar neurons have a corona of dendrites surrounding the cell body and an axon that extends out of the ventral horn. This axon travels through the ventral nerve root to join the emerging spinal nerve. The axon is relatively long because it needs to reach muscles in the periphery of the body. The diameters of cell bodies may be on the order of hundreds of micrometers to support the long axon; some axons are a meter in length, such as the lumbar motor neurons that innervate muscles in the first digits of the feet.

The axons will also branch to innervate multiple muscle fibers. Together, the motor neuron and all the muscle fibers that it controls make up a motor unit. Motor units vary in size. Some may contain up to 1000 muscle fibers, such as in the quadriceps, or they may only have 10 fibers, such as in an extraocular muscle. The number of muscle fibers that are part of a motor unit corresponds to the precision of control of that muscle. Also, muscles that have finer motor control have more motor units connecting to them, and this requires a larger topographical field in the primary motor cortex.

Somatic Reflexes

This chapter began by introducing reflexes as an example of the basic elements of the somatic nervous system. Simple somatic reflexes do not include the higher centers discussed for conscious or voluntary aspects of movement. Reflexes can be spinal or cranial, depending on the nerves and central components that are involved.

The example described at the beginning of the chapter involved heat and pain sensations from a hot stove causing withdrawal of the arm through a connection in the spinal cord that leads to contraction of the biceps brachii (Figure 3.4.3). The description of this **withdrawal reflex** was simplified, for the sake of the introduction, to emphasize the parts of the somatic nervous system. But to consider reflexes fully, more attention needs to be given to this example.

As you withdraw your hand from the stove, you do not want to slow that reflex down. Consequently, as the biceps brachii contracts, the antagonistic triceps brachii needs to relax. In the hot-stove withdrawal reflex, this occurs through an interneuron in the spinal cord. The interneuron's cell body is located in the dorsal horn of the spinal cord. The interneuron receives a synapse from the axon of the sensory neuron that detects that the hand is being burned. In response to this stimulation from the sensory neuron, the interneuron then inhibits the motor neuron that controls the triceps brachii. Without the antagonistic contraction, withdrawal from the hot stove is faster and keeps further tissue damage from occurring.

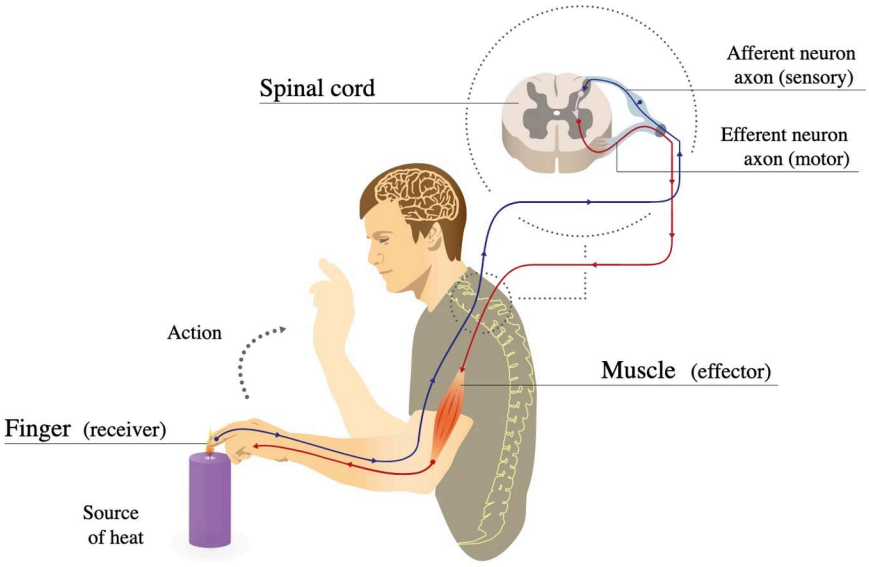


Figure 3.4.3: Nerve impulse of a stimulus is sensed by two or more neurons in the skin of the fingers. The spinal cord receives sensory impulses of the body and, through an interneuron, sends impulses to the arm muscle through spinal nerves. After receiving the order, the muscle runs the command, in this case to remove the finger from the stimulus. (CC BY 4.0; via LibreTexts). Alternative text description of the Figure 3.4.3.

Another type of reflex is a **stretch reflex** shown in Figure 3.4.4. In this reflex, when a skeletal muscle is stretched, a muscle spindle receptor is activated. The axon from this receptor structure will cause direct contraction of the muscle. A collateral of the muscle spindle fiber will also inhibit the motor neuron of the antagonist muscles. A common example of this reflex is the knee jerk that is elicited by a rubber hammer struck against the patellar ligament in a physical exam. The muscle is quickly stretched, resulting in activation of the muscle spindle that sends a signal into the spinal cord through the dorsal root. The fiber synapses directly on the ventral horn motor neuron that activates the muscle, causing contraction. The reflexes are physiologically useful for stability. If a muscle is stretched, it reflexively contracts to return the muscle to compensate for the change in length. In the context of the neurological exam, reflexes indicate that the lower motor neuron is functioning properly.

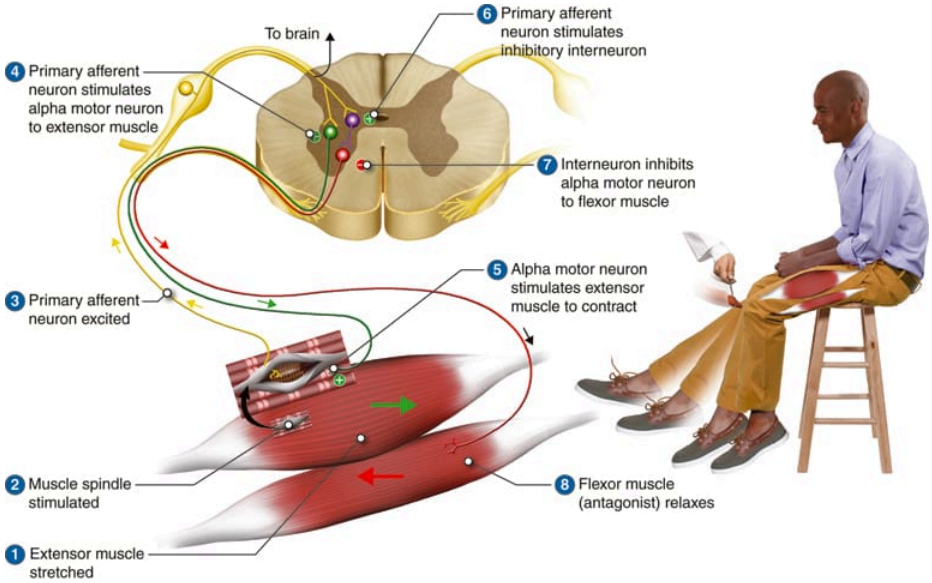


Figure 3.4.4: When an extensor muscle is stretched (1), muscle spindles are stimulated (2) and send information to the spinal cord through a primary afferent neuron (3). The primary afferent neurons synapses on alpha motor neuron of the same extensor muscle (4) causing it to contract (5). At the same time, the primary afferent neuron stimulates an inhibitory interneuron (6) which inhibits the alpha motor neuron to the flexor muscle, preventing its contraction (7). Consequently, the flexor muscle (antagonist) relaxes. (CC BY 4.0; via LibreTexts). Alternative text description of the Figure 3.4.4.

A specialized reflex to protect the surface of the eye is the **corneal reflex**, or the eye blink reflex. When the cornea is stimulated

by a tactile stimulus, or even by bright light in a related reflex, blinking is initiated. The sensory component travels through the trigeminal nerve, which carries somatosensory information from the face, or through the optic nerve, if the stimulus is bright light. The motor response travels through the facial nerve and innervates the orbicularis oculi on the same side. This reflex is commonly tested during a physical exam using an air puff or a gentle touch of a cotton-tipped applicator.

Concept Review

The motor components of the somatic nervous system begin with the frontal lobe of the brain, where the prefrontal cortex is responsible for higher functions such as working memory. The integrative and associate functions of the prefrontal lobe feed into the secondary motor areas, which help plan movements. The premotor cortex and supplemental motor area then feed into the primary motor cortex that initiates movements. Large Betz cells project through the corticobulbar and corticospinal tracts to synapse on lower motor neurons in the brainstem and ventral horn of the spinal cord, respectively. These connections are responsible for generating movements of skeletal muscles.

The extrapyramidal system includes projections from the brainstem and higher centers that influence movement, mostly to maintain balance and posture, as well as to maintain muscle tone. The superior colliculus and red nucleus in the midbrain, the vestibular nuclei in the medulla, and the reticular formation throughout the brainstem each have tracts projecting to the spinal cord in this system. Descending input from the secondary motor cortices, basal nuclei, and cerebellum connect to the origins of these tracts in the brainstem.

All of these motor pathways project to the spinal cord to synapse with motor neurons in the ventral horn of the spinal cord. These lower motor neurons are the cells that connect to skeletal muscle and cause contractions. These neurons project through the spinal nerves to connect to the muscles at neuromuscular junctions. One motor neuron connects to multiple muscle fibers within a target muscle. The number of fibers that are innervated by a single motor neuron varies on the basis of the precision necessary for that muscle and the amount of force necessary for that motor unit. The quadriceps, for example, have many fibers controlled by single motor neurons for powerful contractions that do not need to be precise. The extraocular muscles have only a small number of fibers controlled by each motor neuron because moving the eyes does not require much force, but needs to be very precise.

Reflexes are the simplest circuits within the somatic nervous system. A withdrawal reflex from a painful stimulus only requires the sensory fiber that enters the spinal cord and the motor neuron that projects to a muscle. Antagonist and postural muscles can be coordinated with the withdrawal, making the connections more complex. The simple, single neuronal connection is the basis of somatic reflexes. The corneal reflex is contraction of the orbicularis oculi muscle to blink the eyelid when something touches the surface of the eye. Stretch reflexes maintain a constant length of muscles by causing a contraction of a muscle to compensate for a stretch that can be sensed by a specialized receptor called a muscle spindle.

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3.5: Terms and Definitions

Key Terms

Select to View Definition

Alkaloid
substance, usually from a plant source, that is chemically basic with respect to pH and will stimulate bitter receptors

Amacrine cell
type of cell in the retina that connects to the bipolar cells near the outer synaptic layer and provides the basis for early image processing within the retina

Ampulla
in the ear, the structure at the base of a semicircular canal that contains the hair cells and cupula for transduction of rotational movement of the head

Anosmia
loss of the sense of smell; usually the result of physical disruption of the first cranial nerve

Anterior corticospinal tract
division of the corticospinal pathway that travels through the ventral (anterior) column of the spinal cord and controls axial musculature through the medial motor neurons in the ventral (anterior) horn

Aqueous humor
watery fluid that fills the anterior chamber containing the cornea, iris, ciliary body, and lens of the eye

Ascending pathway
fiber structure that relays sensory information from the periphery through the spinal cord and brain stem to other structures of the brain

Association area
region of cortex connected to a primary sensory cortical area that further processes the information to generate more complex sensory perceptions

Audition
sense of hearing

Auricle
fleshy external structure of the ear

Basilar membrane
in the ear, the floor of the cochlear duct on which the organ of Corti sits

Betz cells
giant pyramidal neuron, crucial for voluntary movement by directly connecting to spinal cord motor neurons via the corticospinal tract

Binocular depth cues
indications of the distance of visual stimuli on the basis of slight differences in the images projected onto either retina

Bipolar cell
cell type in the retina that connects the photoreceptors to the RGCs

Broca's area
region of the frontal lobe associated with the motor commands necessary for speech production

Capsaicin
molecule that activates nociceptors by interacting with a temperature-sensitive ion channel and is the basis for "hot" sensations in spicy food

Cerebral peduncles
segments of the descending motor pathway that make up the white matter of the ventral midbrain

Cervical enlargement
region of the ventral (anterior) horn of the spinal cord that has a larger population of motor neurons for the greater number of and finer control of muscles of the upper limb

Chemoreceptor
sensory receptor cell that is sensitive to chemical stimuli, such as in taste, smell, or pain

Chief sensory nucleus
component of the trigeminal nuclei that is found in the pons

Choroid
highly vascular tissue in the wall of the eye that supplies the outer retina with blood

Ciliary body
smooth muscle structure on the interior surface of the iris that controls the shape of the lens through the zonule fibers

Circadian rhythm
internal perception of the daily cycle of light and dark based on retinal activity related to sunlight

Cochlea
auditory portion of the inner ear containing structures to transduce sound stimuli

Cochlear duct
space within the auditory portion of the inner ear that contains the organ of Corti and is adjacent to the scala tympani and scala vestibuli on either side

Cone photoreceptor
one of the two types of retinal receptor cell that is specialized for color vision through the use of three photopigments distributed through three separate populations of cells

Contralateral
word meaning "on the opposite side," as in axons that cross the midline in a fiber tract

Cornea
fibrous covering of the anterior region of the eye that is transparent so that light can pass through it

Corneal reflex
protective response to stimulation of the cornea causing contraction of the orbicularis oculi muscle resulting in blinking of the eye

Corticobulbar tract
connection between the cortex and the brain stem responsible for generating movement

Corticospinal tract
connection between the cortex and the spinal cord responsible for generating movement

Cupula
specialized structure within the base of a semicircular canal that bends the stereocilia of hair cells when the head rotates by way of the relative movement of the enclosed fluid

Decussate
to cross the midline, as in fibers that project from one side of the body to the other

Dorsal column system
ascending tract of the spinal cord associated with fine touch and proprioceptive sensations

Dorsal stream
connections between cortical areas from the occipital to parietal lobes that are responsible for the perception of visual motion and guiding movement of the body in relation to that motion

Encapsulated ending
configuration of a sensory receptor neuron with dendrites surrounded by specialized structures to aid in transduction of a particular type of sensation, such as the lamellated corpuscles in the deep dermis and subcutaneous tissue

Equilibrium
sense of balance that includes sensations of position and movement of the head

Executive functions
cognitive processes of the prefrontal cortex that lead to directing goal-directed behavior, which is a precursor to executing motor commands

External ear
structures on the lateral surface of the head, including the auricle and the ear canal back to the tympanic membrane

Exteroceptor
sensory receptor that is positioned to interpret stimuli from the external environment, such as photoreceptors in the eye or somatosensory receptors in the skin

Extraocular muscle
one of six muscles originating out of the bones of the orbit and inserting into the surface of the eye which are responsible for moving the eye

Extrapyramidal system
pathways between the brain and spinal cord that are separate from the corticospinal tract and are responsible for modulating the movements generated through that primary pathway

Fasciculus cuneatus
lateral division of the dorsal column system composed of fibers from sensory neurons in the upper body

Fasciculus gracilis
medial division of the dorsal column system composed of fibers from sensory neurons in the lower body

Fibrous tunic
outer layer of the eye primarily composed of connective tissue known as the sclera and cornea

Fovea
exact center of the retina at which visual stimuli are focused for maximal acuity, where the retina is thinnest, at which there is nothing but photoreceptors

Free nerve ending
configuration of a sensory receptor neuron with dendrites in the connective tissue of the organ, such as in the dermis of the skin, that are most often sensitive to chemical, thermal, and mechanical stimuli

Frontal eye fields
area of the prefrontal cortex responsible for moving the eyes to attend to visual stimuli

General sense
any sensory system that is distributed throughout the body and incorporated into organs of multiple other systems, such as the walls of the digestive organs or the skin

Gustation
sense of taste

Gustatory receptor cells
sensory cells in the taste bud that transduce the chemical stimuli of gustation

Hair cells
mechanoreceptor cells found in the inner ear that transduce stimuli for the senses of hearing and balance

Incus
(also, anvil) ossicle of the middle ear that connects the malleus to the stapes

Inferior colliculus
last structure in the auditory brainstem pathway that projects to the thalamus and superior colliculus

Inferior oblique
extraocular muscle responsible for lateral rotation of the eye

Inferior rectus
extraocular muscle responsible for looking down

Inner ear
structure within the temporal bone that contains the sensory apparati of hearing and balance

Inner segment
in the eye, the section of a photoreceptor that contains the nucleus and other major organelles for normal cellular functions

Inner synaptic layer
layer in the retina where bipolar cells connect to RGCs

Interaural intensity difference
cue used to aid sound localization in the horizontal plane that compares the relative loudness of sounds at the two ears, because the ear closer to the sound source will hear a slightly more intense sound

Interaural time difference
cue used to help with sound localization in the horizontal plane that compares the relative time of arrival of sounds at the two ears, because the ear closer to the sound source will receive the stimulus microseconds before the other ear

Internal capsule
segment of the descending motor pathway that passes between the caudate nucleus and the putamen

Interoceptor
sensory receptor that is positioned to interpret stimuli from internal organs, such as stretch receptors in the wall of blood vessels

Ipsilateral
word meaning on the same side, as in axons that do not cross the midline in a fiber tract

Iris
colored portion of the anterior eye that surrounds the pupil

Kkinesthesia
sense of body movement based on sensations in skeletal muscles, tendons, joints, and the skin

Lacrimal duct
duct in the medial corner of the orbit that drains tears into the nasal cavity

Lacrimal gland
gland lateral to the orbit that produces tears to wash across the surface of the eye

Lateral corticospinal tract
division of the corticospinal pathway that travels through the lateral column of the spinal cord and controls appendicular musculature through the lateral motor neurons in the ventral (anterior) horn

Lateral geniculate nucleus
thalamic target of the RGCs that projects to the visual cortex

Lateral rectus
extraocular muscle responsible for abduction of the eye

Lens
component of the eye that focuses light on the retina

Levator palpebrae superioris
muscle that causes elevation of the upper eyelid, controlled by fibers in the oculomotor nerve

Lumbar enlargement
region of the ventral (anterior) horn of the spinal cord that has a larger population of motor neurons for the greater number of muscles of the lower limb

Macula
enlargement at the base of a semicircular canal at which transduction of equilibrium stimuli takes place within the ampulla

Malleus
(also, hammer) ossicle that is directly attached to the tympanic membrane

Mechanoreceptor
receptor cell that transduces mechanical stimuli into an electrochemical signal

Medial geniculate nucleus
thalamic target of the auditory brain stem that projects to the auditory cortex

Medial lemniscus
fiber tract of the dorsal column system that extends from the nuclei gracilis and cuneatus to the thalamus, and decussates

Medial rectus
extraocular muscle responsible for adduction of the eye

Mesencephalic nucleus
component of the trigeminal nuclei that is found in the midbrain

Middle ear
space within the temporal bone between the ear canal and bony labyrinth where the ossicles amplify sound waves from the tympanic membrane to the oval window

Multimodal integration area
region of the cerebral cortex in which information from more than one sensory modality is processed to arrive at higher level cortical functions such as memory, learning, or cognition

Neural tunic
layer of the eye that contains nervous tissue, namely the retina

Nociceptor
receptor cell that senses pain stimuli

Nucleus cuneatus
medullary nucleus at which first-order neurons of the dorsal column system synapse specifically from the upper body and arms

Nucleus gracilis
: medullary nucleus at which first-order neurons of the dorsal column system synapse specifically from the lower body and legs

Odorant molecules
: volatile chemicals that bind to receptor proteins in olfactory neurons to stimulate the sense of smell

Olfaction
: sense of smell

Olfactory bulb
: central target of the first cranial nerve; located on the ventral surface of the frontal lobe in the cerebrum

Olfactory epithelium
: region of the nasal epithelium where olfactory neurons are located

Olfactory sensory neuron
: receptor cell of the olfactory system, sensitive to the chemical stimuli of smell, the axons of which compose the first cranial nerve

Opsin
: protein that contains the photosensitive cofactor retinal for phototransduction

Optic chiasm
: decussation point in the visual system at which medial retina fibers cross to the other side of the brain

Optic disc
: spot on the retina at which RGC axons leave the eye and blood vessels of the inner retina pass

Optic nerve
: second cranial nerve, which is responsible visual sensation

Optic tract
: name for the fiber structure containing axons from the retina posterior to the optic chiasm representing their CNS location

Organ of Corti
: structure in the cochlea in which hair cells transduce movements from sound waves into electrochemical signals

Osmoreceptor
: receptor cell that senses differences in the concentrations of bodily fluids on the basis of osmotic pressure

Ossicles
: three small bones in the middle ear

Otolith
: layer of calcium carbonate crystals located on top of the otolithic membrane

Otolithic membrane
: gelatinous substance in the utricle and saccule of the inner ear that contains calcium carbonate crystals and into which the stereocilia of hair cells are embedded

Outer segment
: in the eye, the section of a photoreceptor that contains opsin molecules that transduce light stimuli

Outer synaptic layer
: layer in the retina at which photoreceptors connect to bipolar cells

Oval window
: membrane at the base of the cochlea where the stapes attaches, marking the beginning of the scala vestibuli

Palpebral conjunctiva
membrane attached to the inner surface of the eyelids that covers the anterior surface of the cornea

Papilla
for gustation, a bump-like projection on the surface of the tongue that contains taste buds

Photoisomerization
chemical change in the retinal molecule that alters the bonding so that it switches from the 11-*cis*-retinal isomer to the all-*trans*-retinal isomer

Photon
individual “packet” of light

Photoreceptor
receptor cell specialized to respond to light stimuli

Premotor cortex
cortical area anterior to the primary motor cortex that is responsible for planning movements

Primary sensory cortex
region of the cerebral cortex that initially receives sensory input from an ascending pathway from the thalamus and begins the processing that will result in conscious perception of that modality

Proprioception
sense of position and movement of the body

Proprioceptor
receptor cell that senses changes in the position and kinesthetic aspects of the body

Pupil
open hole at the center of the iris that light passes through into the eye

Pyramidal decussation
location at which corticospinal tract fibers cross the midline and segregate into the anterior and lateral divisions of the pathway

Pyramids
segment of the descending motor pathway that travels in the anterior position of the medulla

Receptor cell
cell that transduces environmental stimuli into neural signals

Red nucleus
midbrain nucleus that sends corrective commands to the spinal cord along the rubrospinal tract, based on disparity between an original command and the sensory feedback from movement

Reticulospinal tract
extrapyramidal connections between the brain stem and spinal cord that modulate movement, contribute to posture, and regulate muscle tone

Retina
nervous tissue of the eye at which phototransduction takes place

Retinal
cofactor in an opsin molecule that undergoes a biochemical change when struck by a photon (pronounced with a stress on the last syllable)

Retinal ganglion cell (RGC)
neuron of the retina that projects along the second cranial nerve

Rhodopsin
photopigment molecule found in the rod photoreceptors

Rod photoreceptor
one of the two types of retinal receptor cell that is specialized for low-light vision

Round window
membrane that marks the end of the scala tympani

Rubrospinal tract
descending motor control pathway, originating in the red nucleus, that mediates control of the limbs on the basis of cerebellar processing

Saccule
structure of the inner ear responsible for transducing linear acceleration in the vertical plane

Scala tympani
portion of the cochlea that extends from the apex to the round window

Scala vestibuli
portion of the cochlea that extends from the oval window to the apex

Sclera
white of the eye

Semicircular canals
structures within the inner ear responsible for transducing rotational movement information

Sensory homunculus
topographic representation of the body within the somatosensory cortex demonstrating the correspondence between neurons processing stimuli and sensitivity

Sensory modality
a particular system for interpreting and perceiving environmental stimuli by the nervous system

Solitary nucleus
medullar nucleus that receives taste information from the facial and glossopharyngeal nerves

Somatosensation
general sense associated with modalities lumped together as touch

Special sense
any sensory system associated with a specific organ structure, namely smell, taste, sight, hearing, and balance

Spinal trigeminal nucleus
component of the trigeminal nuclei that is found in the medulla

Spinothalamic tract
ascending tract of the spinal cord associated with pain and temperature sensations

Spiral ganglion
location of neuronal cell bodies that transmit auditory information along the eighth cranial nerve

Stapes
(also, stirrup) ossicle of the middle ear that is attached to the inner ear

Stereocilia
array of apical membrane extensions in a hair cell that transduce movements when they are bent

Stretch reflex
response to activation of the muscle spindle stretch receptor that causes contraction of the muscle to maintain a constant length

Submodality
specific sense within a broader major sense such as sweet as a part of the sense of taste, or color as a part of

vision

Superior colliculus
structure in the midbrain that combines visual, auditory, and somatosensory input to coordinate spatial and topographic representations of the three sensory systems

Superior oblique
extraocular muscle responsible for medial rotation of the eye

Superior rectus
extraocular muscle responsible for looking up

Supplemental motor area
cortical area anterior to the primary motor cortex that is responsible for planning movements

Suprachiasmatic nucleus
hypothalamic target of the retina that helps to establish the circadian rhythm of the body on the basis of the presence or absence of daylight

Taste buds
structures within a papilla on the tongue that contain gustatory receptor cells

Tectorial membrane
component of the organ of Corti that lays over the hair cells, into which the stereocilia are embedded

Tectospinal tract
extrapyramidal connections between the superior colliculus and spinal cord

Thermoreceptor
sensory receptor specialized for temperature stimuli

Topographical
relating to positional information

Transduction
process of changing an environmental stimulus into the electrochemical signals of the nervous system

Trochlea
cartilaginous structure that acts like a pulley for the superior oblique muscle

Tympanic membrane
ear drum

Umami
taste submodality for sensitivity to the concentration of amino acids; also called the savory sense

Utricle
structure of the inner ear responsible for transducing linear acceleration in the horizontal plane

Vascular tunic
middle layer of the eye primarily composed of connective tissue with a rich blood supply

Ventral posterior nucleus
nucleus in the thalamus that is the target of gustatory sensations and projects to the cerebral cortex

Ventral stream
connections between cortical areas from the occipital lobe to the temporal lobe that are responsible for identification of visual stimuli

Vestibular ganglion
location of neuronal cell bodies that transmit equilibrium information along the eighth cranial nerve

Vestibular nuclei
targets of the vestibular component of the eighth cranial nerve

Vestibule
in the ear, the portion of the inner ear responsible for the sense of equilibrium

Vestibulo-ocular reflex (VOR)
reflex based on connections between the vestibular system and the cranial nerves of eye movements that ensures images are stabilized on the retina as the head and body move

Vestibulospinal tract
extrapyramidal connections between the vestibular nuclei in the brain stem and spinal cord that modulate movement and contribute to balance on the basis of the sense of equilibrium

Visceral sense
sense associated with the internal organs

Vision
special sense of sight based on transduction of light stimuli

Visual acuity
property of vision related to the sharpness of focus, which varies in relation to retinal position

Vitreous humor
viscous fluid that fills the posterior chamber of the eye

Working memory
function of the prefrontal cortex to maintain a representation of information that is not in the immediate environment

Zonule fibers
fibrous connections between the ciliary body and the lens

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3.6: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. What type of receptor cell is responsible for transducing pain stimuli?
   A. Mechanoreceptor
   B. Nociceptor
   C. Osmoreceptor
   D. Photoreceptor

2. Which cranial nerve is part of the gustatory (taste) system?
   A. Olfactory (CN I)
   B. Trochlear (CN IV)
   C. Trigeminal (CN V)
   D. Facial (CN VII)

3. Which submodality of taste is most sensitive to the pH (acidity) of saliva?
   A. Umami
   B. Sour
   C. Bitter
   D. Sweet

4. Axons from which retinal neurons form the optic nerve?
   A. Amacrine cells
   B. Photoreceptors
   C. Bipolar cells
   D. Retinal ganglion cells

5. What type of receptor cell is involved in the sensations of sound and balance?
   A. Photoreceptor
   B. Chemoreceptor
   C. Mechanoreceptor
   D. Nociceptor

6. Which of these sensory modalities does not pass through the ventral posterior nucleus of the thalamus?
   A. Gustation (taste)
   B. Proprioception (body position)
   C. Audition (hearing)
   D. Nociception (pain)

7. Which nucleus in the medulla oblongata sends auditory information to the inferior colliculus?
   A. Solitary nucleus
   B. Vestibular nucleus
   C. Chief sensory nucleus
   D. Cochlear nucleus

8. Visual stimuli in the upper-left visual field will be processed in which region of the primary visual cortex?
   A. Inferior right
   B. Inferior left
   C. Superior right
   D. Superior left

9. Which location on the body has the largest area of representation in the primary somatosensory cortex (sensory homunculus)?
   A. Lips
   B. Thigh
   C. Elbow
   D. Neck

10. What is the name for the topographical “map” of the body on the somatosensory cortex?
    A. Homunculus
    B. Homo sapiens
    C. Postcentral gyrus
    D. Primary cortex

11. Which of the following is a direct target of neurons in the vestibular ganglion?
    A. Superior colliculus
    B. Cerebellum
    C. Thalamus
    D. Optic chiasm

12. Which region of the frontal lobe directly initiates voluntary movement by sending axons to cranial and spinal motor neurons?
    A. Prefrontal cortex
    B. Supplemental motor area
    C. Premotor cortex
    D. Primary motor cortex

13. Which extrapyramidal tract combines equilibrium (balance) information with motor commands to help maintain posture?
    A. Tectospinal tract
    B. Vestibulospinal tract
    C. Reticulospinal tract
    D. Corticospinal tract

14. Which region of gray matter in the spinal cord contains the cell bodies of motor neurons that innervate skeletal muscle?
    A. Ventral horn
    B. Dorsal horn
    C. Lateral horn
    D. Lateral column

15. What type of spinal reflex protects the foot when a painful stimulus is sensed (for example, stepping on a sharp object)?
    A. Stretch reflex
    B. Gag reflex
    C. Withdrawal (flexor) reflex
    D. Corneal reflex

16. Which term best describes a receptor that responds to the position and movement of joints and muscles?
    A. Nociceptor
    B. Proprioceptor
    C. Chemoreceptor
    D. Photoreceptor

17. A patient reports loss of fine touch and vibration sense in the right hand, but pain and temperature are normal. Which pathway is most likely affected?
    A. Spinothalamic tract

B. Dorsal column–medial lemniscus pathway
C. Vestibulospinal tract
D. Corticospinal tract

18. Which motor pathway is most important for precise, voluntary movements of the distal limbs (for example, fingers)?
A. Vestibulospinal tract
B. Reticulospinal tract
C. Corticospinal tract
D. Tectospinal tract

19. During a stretch reflex at the knee (patellar reflex), which sequence best describes the basic pathway?
A. Receptor → motor neuron → spinal cord → muscle
B. Receptor → spinal cord → motor neuron → muscle
C. Receptor → brain → spinal cord → motor neuron → muscle
D. Receptor → sensory neuron → brain → muscle

20. Which part of the brain is most directly responsible for smooth coordination of movements and balance (for example, walking on a narrow beam)?
A. Medulla oblongata
B. Cerebellum
C. Thalamus
D. Hypothalamus

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3.7: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. When you pick up a hot cup, you feel the heat and then decide to put it down. Explain how sensory information from your hand leads to a coordinated motor response.
   Hint: Think of the steps: receptors in skin → sensory neuron → brain → motor neuron → muscles of hand and arm.

2. A person is walking on uneven ground in the dark but does not fall. How do different senses (for example, vision, touch, balance) and motor responses work together to keep the person upright?
   Hint: Think about the role of feet (pressure), inner ear (balance), and muscles making small corrections.

3. You can touch your nose with your eyes closed. Explain what kind of sensory information helps you do this accurately and how the nervous system uses it to guide movement.
   Hint: Think about proprioception (sense of body position) from muscles and joints, not just vision.

4. Imagine a person has damage to the part of the brain that interprets visual information, but their eyes are normal. How might this affect their motor responses to objects they see, such as catching a ball?
   Hint: Consider that the eyes can send signals, but the brain cannot “understand” them well to plan movement.

5. Reflexes are very fast and automatic, but many movements (like playing piano) are slower and more controlled. Explain why the body needs both fast reflexes and slower, planned motor responses.
   Hint: Think about protection from danger (reflex) versus movements that need practice and precision (voluntary control).

6. If sensory feedback from a limb is reduced or lost (for example, numbness in a hand), how might this change the person’s ability to perform fine motor tasks like writing or buttoning a shirt?
   Hint: Think about how you adjust grip strength and finger position when you can feel the pen or button.

7. When learning a new sport skill (like shooting a basketball), you often start clumsy and then improve. Explain how repeated sensory feedback helps the nervous system produce more coordinated motor responses over time.
   Hint: Think about trial and error: you see and feel what went wrong, then the brain adjusts the commands to muscles.

8. Sometimes the brain must ignore certain sensory information to make a smooth movement (for example, ignoring background noise while speaking). Give one example and explain why filtering out some sensory input is helpful for coordinated actions.
   Hint: Consider what would happen if you tried to pay attention to every sound, sight, and touch at the same time.

9. A person steps on a rolling object (like a small ball) and quickly shifts their weight to avoid falling. Describe how rapid sensory input from the foot and balance organs leads to changes in muscle activity in the legs and trunk.

Hint: Follow the path: receptors in skin and joints + inner ear → CNS → activation of many muscles to change posture.

10. Strong emotions (like fear or anxiety) can change both how we sense things and how we move (for example, shaking hands, "freezing"). Explain one way emotion might alter sensory processing and one way it might alter motor responses.
Hint: Think about increased sensitivity to sounds or touch, faster heart rate, muscle tension, or difficulty making smooth movements.

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3.8: Alternate Text Descriptions

Figure 3.1.1: Types of Sensory Receptors

This figure compares three structural types of sensory receptors and illustrates how sensory information is detected and transmitted to the nervous system. The figure is organized into three labeled panels arranged vertically.

Panel (a): Neuron (receptor) with free nerve endings

The top panel shows a sensory neuron that functions directly as a receptor. On the left side, multiple branching **free nerve endings (dendrites)** extend outward without any surrounding capsule or specialized structure. These free dendritic branches connect to a long **axon** that runs horizontally across the figure. Approximately midway along the neuron, a small rounded **cell body** projects upward from the axon. The axon continues to the right and ends in several terminal branches. Labels identify the free nerve endings, axon, and cell body. This panel demonstrates a receptor neuron in which the dendrites themselves detect stimuli from the environment.

Panel (b): Neuron (receptor) with encapsulated nerve endings

The middle panel shows another sensory neuron that functions directly as a receptor. At the left end of the neuron, the dendritic branches are enclosed within a large oval structure labeled **encapsulated nerve ending**. A label identifies the enclosed **dendrite** within the capsule. The encapsulated ending connects to a long **axon** that extends horizontally across the figure. A small rounded **cell body** projects upward from the axon, similar to the neuron shown in panel (a). The axon continues to the right and terminates in several branches. Labels identify the dendrite, encapsulated nerve ending, axon, and cell body. This panel demonstrates a receptor neuron whose sensory endings are surrounded by a specialized capsule that assists in detecting specific types of stimuli.

Panel (c): Specialized receptor cell

The bottom panel depicts a **specialized receptor cell** rather than a receptor neuron. On the left is a cylindrical sensory structure labeled **rod**. The rod is connected to an elongated receptor cell containing a visible oval nucleus. The receptor cell forms a synaptic connection on the right with a neighboring **bipolar cell**. An arrow points from the receptor cell toward the bipolar cell, indicating the direction of signal transmission. Unlike the receptors shown in panels (a) and (b), the receptor cell does not send an action potential directly along its own axon. Instead, it detects a stimulus and passes information to the adjacent bipolar neuron.

The figure illustrates three ways sensory stimuli can be detected and conveyed to the nervous system:

1. **Free nerve endings** are exposed dendritic endings of sensory neurons that directly detect stimuli.
2. **Encapsulated nerve endings** are sensory neuron dendrites enclosed within specialized structures that enhance detection of particular stimulus types.
3. **Specialized receptor cells**, such as photoreceptors, detect stimuli and then relay information to a separate sensory neuron, represented here by a bipolar cell.

Figure 3.1.2: Papillae and Taste Buds of the Tongue

This figure illustrates the locations and structures of papillae and taste buds on the tongue and shows the cellular organization of a taste bud involved in the sense of taste (gustation). The figure is composed of a central tongue diagram with enlarged views of different papilla types and a detailed cross-sectional view of a taste bud.

Overview of Papillae on the Tongue

At the center-left is a dorsal view of the tongue. Three highlighted regions on the tongue are connected by arrows to enlarged diagrams showing different types of papillae.

Circumvallate papilla

Near the posterior region of the tongue, a highlighted area is enlarged to show a cross-sectional view of a **circumvallate papilla**. Several structures labeled **taste buds** are embedded within the sides of the papilla. An additional arrow leads to a photomicrograph showing microscopic tissue containing numerous taste buds within the epithelial lining. This enlargement demonstrates that circumvallate papillae contain many taste buds.

Fungiform papilla

A highlighted region near the anterior portion of the tongue is enlarged to show a **fungiform papilla**. The papilla appears as a rounded projection extending from the tongue surface. The illustration indicates the presence of sensory innervation within the papilla. Fungiform papillae are located primarily on the anterior portion of the tongue.

Filiform papilla

Another highlighted region is enlarged to show a **filiform papilla**. The structure appears narrow and pointed with a tapered shape. No taste buds are shown within this papilla. The illustration distinguishes filiform papillae from the other papilla types by emphasizing their structural role rather than a taste-sensory function.

Foliate papilla

A highlighted area on the lateral side of the tongue is enlarged to show a **foliate papilla**. Multiple folds extend into the tissue, and several structures labeled **taste buds** are located along the sides of the folds. This enlargement demonstrates that foliate papillae contain taste buds within the grooves of the tissue.

Structure of a Taste Bud

On the right side of the figure is a highly enlarged cross-sectional view of a single **taste bud** embedded within the tongue epithelium.

At the top of the taste bud is a small opening labeled **taste pore**, which connects the taste bud to the surface of the tongue.

Projecting through the taste pore are slender hairlike structures labeled **taste hairs**. These structures extend from the sensory cells into the opening and are exposed to dissolved chemicals in food and beverages.

Within the taste bud are elongated cells containing oval nuclei. Two cell types are identified:

- **Gustatory cells**, the sensory receptor cells responsible for detecting taste stimuli.
- **Transitional cells**, supporting cells located within the taste bud.

At the base of the taste bud are **basal cells**, which serve as progenitor cells that replace worn or damaged taste receptor cells.

Nerve fibers enter the bottom of the taste bud and make contact with the gustatory cells, allowing sensory information to be transmitted to the nervous system.

The figure demonstrates how structures on the tongue support the sense of taste. It shows that:

1. The tongue contains several types of papillae, including circumvallate, fungiform, filiform, and foliate papillae.
2. Circumvallate and foliate papillae contain numerous taste buds, while filiform papillae are shown without taste buds.
3. Taste buds are specialized sensory organs composed of gustatory cells, supporting transitional cells, and regenerative basal cells.
4. Taste hairs project through the taste pore to detect chemicals dissolved in saliva.
5. When chemical tastants contact the taste hairs, gustatory cells are stimulated and transmit sensory information through associated nerve fibers, initiating the perception of taste.

Figure 3.1.3: The Olfactory System

This figure illustrates the anatomy and function of the olfactory (smell) system, showing the location of the olfactory epithelium within the nasal cavity, the microscopic organization of olfactory sensory tissue, and a histological image of the olfactory epithelium. The figure contains three panels labeled **(a) Nasal cavity**, **(b) Olfactory system**, and **(c) Olfactory epithelium**.

(a) Nasal cavity

The left panel presents a sagittal section of the head focused on the nasal cavity. A cup emitting vapor is shown below the nose to represent odor-containing air entering during inhalation.

A red arrow labeled **path of inhaled air** traces the movement of air through the nasal cavity toward the superior region of the nasal passage. The arrow passes beneath structures labeled **nasal conchae**, indicating that inhaled air is directed toward the upper nasal cavity. Near the roof of the nasal cavity is the **olfactory epithelium**, the specialized sensory tissue responsible for detecting odors. Above the olfactory epithelium are the **olfactory bulb** and **olfactory tract**, components of the central nervous system that process smell information.

An arrow links this overview to a magnified view of the olfactory system shown in panel (b).

(b) Olfactory system

The center panel provides an enlarged cross-sectional view of the olfactory region.

At the top of the diagram is the **olfactory bulb**, containing spherical structures labeled **glomeruli** and neurons labeled **mitral cells**. The mitral cells give rise to fibers that form the **olfactory tract**, which carries olfactory information deeper into the brain.

Below the olfactory bulb is the **ethmoid bone**. Bundles of nerve fibers labeled **filaments of the olfactory nerve** pass through openings in the ethmoid bone, connecting sensory neurons in the nasal cavity to structures within the olfactory bulb.

Below the bone is **connective tissue**, which contains an **olfactory gland**. The gland produces secretions that contribute to the mucus layer covering the sensory surface.

The sensory region of the nasal cavity is identified as the **olfactory epithelium**. A **basal lamina** separates the epithelium from the underlying connective tissue.

Within the olfactory epithelium are several cell types:

- **Olfactory receptor neurons (ORNs)**, shown as elongated sensory neurons extending from the epithelial surface toward the olfactory nerve filaments.
- **Supporting cells**, located between the receptor neurons and providing structural and metabolic support.
- **Dendrites of ORNs**, which extend toward the epithelial surface.

At the surface of the epithelium is a layer labeled **mucus**. Extending from the dendrites into the mucus are hairlike structures labeled **olfactory cilia**.

At the bottom of the panel, red arrows labeled **path of inhaled air containing odorant molecules** indicate airflow across the mucus-covered surface. The diagram shows odorant molecules reaching the olfactory cilia, where detection occurs.

(c) Olfactory epithelium

The lower panel is a histological micrograph of olfactory epithelium. The image shows densely packed layers of cells arranged in a pseudostratified epithelial pattern. Numerous elongated cell nuclei are visible within the epithelial layer. Above the epithelium are glandular and connective tissue structures corresponding to those shown in the schematic diagram. The micrograph provides a microscopic view of the sensory tissue responsible for odor detection.

The figure demonstrates how the olfactory system detects odors and transmits smell information to the brain.

1. Inhaled air carrying **odorant molecules** enters the nasal cavity and is directed toward the olfactory epithelium.
2. Odorant molecules dissolve in the mucus covering the sensory surface.
3. The odorants interact with receptors located on the **olfactory cilia** of olfactory receptor neurons.
4. Olfactory receptor neurons convert the chemical signals into nerve impulses.
5. Axons from the receptor neurons form the **olfactory nerve filaments**, which pass through the **ethmoid bone** and enter the **olfactory bulb**.
6. Within the olfactory bulb, the axons synapse in **glomeruli** with **mitral cells**.
7. Mitral cell axons form the **olfactory tract**, which carries smell information to the brain for perception and interpretation.

Figure 3.2.1: The Human Ear

This figure is a labeled cross-sectional diagram of the human ear divided into three main regions: the **external ear**, **middle ear**, and **inner ear**.

The **external ear** includes:

- the **auricle** (outer ear)
- the **ear canal**, which carries sound inward
- the **tympanic membrane (eardrum)**, which marks the boundary between the external and middle ear

The **middle ear** includes:

- the **tympanic cavity**
- the small auditory bones, including the **malleus** and **incus**
- the **stapes**, which connects to the **oval window**
- the **Eustachian (auditory) tube**, which extends downward and helps equalize air pressure

The **inner ear** includes:

- the **vestibule**
- the **cochlea**, a spiral-shaped structure involved in hearing
- the **round window**
- the **vestibular nerve**, which carries balance information
- the **cochlear nerve**, which carries sound information to the brain

Brackets beneath the image label the three main regions—external ear, middle ear, and inner ear—showing the organization of the ear and the pathway through which sound is transmitted and processed.
The diagram helps learners identify the major anatomical structures of the ear and understand how sound moves from the outer ear through the middle ear to the inner ear, where auditory signals are processed and sent to the brain.

Figure 3.2.2: Cross Section of the Cochlea

This figure shows a cross-sectional view of the cochlea and highlights the structures involved in hearing, including the cochlear chambers, membranes, the organ of Corti, and the pathway by which auditory information is transmitted to the brain.

The figure consists of a small inset showing the location of the cross section within the spiral cochlea and a larger enlarged cross-sectional diagram of the cochlea.

The enlarged diagram displays a slice through the cochlea enclosed by the **bony cochlear wall**. Within the cochlea are three fluid-filled chambers arranged vertically:

- The **scala vestibuli** occupies the upper chamber.
- The **cochlear duct** occupies the middle chamber.
- The **scala tympani** occupies the lower chamber.

These compartments are separated by specialized membranes.

Cochlear Membranes and Duct

The **vestibular membrane** forms the boundary between the scala vestibuli and the cochlear duct.

The **basilar membrane** forms the floor of the cochlear duct and separates the cochlear duct from the scala tympani.

Suspended within the cochlear duct is the **tectorial membrane**, a ribbon-like structure that extends over the sensory cells of the hearing apparatus.

Organ of Corti

Located on top of the basilar membrane within the cochlear duct is the **organ of Corti**, the sensory organ responsible for hearing. The organ of Corti contains the auditory receptor cells (hair cells), which convert mechanical vibrations into electrical signals. The figure identifies the organ of Corti as being positioned between the basilar membrane below and the tectorial membrane above.

Auditory Nerve Connections

Nerve fibers extend from the organ of Corti toward a cluster of neuron cell bodies labeled the **spiral ganglion**. The spiral ganglion contains the sensory neurons that carry auditory information from the receptor cells.

Axons from the spiral ganglion converge to form the **cochlear branch of cranial nerve VIII (vestibulocochlear nerve)**, shown as a large bundle of nerve fibers leaving the cochlea and traveling toward the brain.

Functional Relationship of the Structures

The figure illustrates how sound is detected within the cochlea:

1. Sound vibrations entering the inner ear create fluid movements within the scala vestibuli, cochlear duct, and scala tympani.
2. Movement of cochlear fluids causes displacement of the **basilar membrane**.
3. Movement of the basilar membrane shifts the **organ of Corti** relative to the **tectorial membrane**.
4. This mechanical interaction bends the hair cells within the organ of Corti, initiating sensory transduction.
5. Hair cells convert mechanical vibration into electrical signals.
6. These signals are transmitted to neurons within the **spiral ganglion**.
7. Axons from the spiral ganglion form the **cochlear branch of cranial nerve VIII**, which carries auditory information to the brain for perception of sound.

The figure demonstrates the anatomical organization of the cochlea and the pathway by which sound is converted into neural signals. It shows the three cochlear chambers (scala vestibuli, cochlear duct, and scala tympani), the vestibular, basilar, and tectorial membranes, the location of the organ of Corti, the spiral ganglion, and the cochlear branch of the vestibulocochlear nerve. The figure emphasizes that hearing occurs when sound-induced movement within the cochlea stimulates hair cells in the organ of Corti, which then transmit auditory information to the brain through the cochlear nerve.

Figure 3.2.3: Histological Micrograph of the Cochlea

This figure is a histological micrograph of a cross section through the cochlea of the inner ear. The image highlights the anatomical relationships among the **vestibular membrane**, **tectorial membrane**, **organ of Corti**, and **basilar membrane**, which together form the sensory apparatus responsible for hearing.

The micrograph is stained in shades of pink and purple, allowing various tissues and membranes within the cochlea to be distinguished. A small inset image in the upper-right corner shows the location of the section within the overall cochlear structure.

Anatomical Structures

Near the upper center of the image, a thin membrane labeled **vestibular membrane** extends diagonally across the cochlear chamber. This membrane forms part of the boundary separating fluid-filled compartments within the cochlea.

Below the vestibular membrane is a lighter-staining region containing the **tectorial membrane**, which appears as a delicate curved structure extending over the sensory epithelium.

Directly beneath the tectorial membrane is the **organ of Corti**, outlined by a rectangular box. The organ of Corti sits on the upper surface of the basilar membrane and contains the sensory receptor cells responsible for detecting sound vibrations. Although individual hair cells are not identified in this image, the labeled region represents the location of these auditory receptor cells.

The **basilar membrane** forms the floor of the organ of Corti and appears as a thin supporting structure extending horizontally across the image. It separates the cochlear duct from the fluid-filled chamber below.

Surrounding these structures are the walls of the cochlea and supporting connective tissues, visible as densely stained pink and purple regions.

Spatial Relationships

From top to bottom, the labeled structures are arranged in the following order:

1. Vestibular membrane
2. Tectorial membrane
3. Organ of Corti
4. Basilar membrane

This arrangement illustrates the position of the sensory hearing apparatus within the cochlear duct.

Function of the Structures

The figure demonstrates how the structural components of the cochlea work together to detect sound:

- Sound waves entering the inner ear generate pressure waves within the cochlear fluids.
- Fluid movement causes the **basilar membrane** to vibrate.
- As the basilar membrane moves, the **organ of Corti** moves relative to the overlying **tectorial membrane**.
- This relative motion bends the stereocilia of auditory hair cells located within the organ of Corti.
- Bending of the stereocilia converts mechanical energy into electrical signals through sensory transduction.
- These electrical signals are transmitted to auditory neurons and ultimately carried to the brain for sound perception.

The figure provides a microscopic view of the hearing apparatus within the cochlea. It identifies the vestibular membrane, tectorial membrane, organ of Corti, and basilar membrane and shows their spatial arrangement within the cochlear duct. The image illustrates that hearing depends on vibration of the basilar membrane and stimulation of sensory hair cells in the organ of Corti, which convert sound-induced mechanical movement into neural signals that can be interpreted by the brain as sound.

Figure 3.2.4: Auditory Hair Cells and the Organ of Corti

This medical illustration depicts the microscopic anatomy and functional mechanics of the inner ear's auditory receptor system. It features two main components: a cross-sectional view of the cochlear duct on the left, and an enlarged, detailed diagram of auditory hair cells on the right, connected by a large gray callout arrow.

Left Panel: Cochlear Cross-Section

The left side shows a cross-section of a single turn of the bony, snail shell-shaped cochlea. Inside the porous, beige bone structure are fluid-filled chambers lined with specialized membranes:

- **Chambers:** Three separate compartments are visible. The upper chamber (scala vestibuli) and lower chamber (scala tympani) enclose a central, triangular compartment known as the cochlear duct (scala media).
- **Organ of Corti:** Located along the base of the central triangular duct is a specialized cellular layer. A small rectangular box outlines a segment of this layer, with a prominent gray arrow pointing to the right panel to signal a highly magnified view.
- **Auditory Nerve:** A thick bundle of yellow nerve fibers extends from the base of the cellular layer, merging into a larger nerve

trunk that carries signals toward the brain.

Right Panel: Magnified Auditory Hair Cells

The right side provides a high-magnification look inside the rectangular box from the left panel, showcasing the cellular structure responsible for sensory transduction:

- **Hair Cells:** Two rows of elongated, yellowish-beige sensory cells (hair cells) are embedded within a supportive tissue matrix at the bottom. Each cell contains a visible reddish-pink nucleus.
- **Stereocilia:** At the top surface of each hair cell are multiple hair-like projections arranged in graduated heights, labeled as **Stereocilia**.
- **Tethers:** Small vertical lines, labeled as **Tether** (clinically known as tip links), connect the tips of shorter stereocilia to the sides of adjacent taller stereocilia.
- **Tectorial Membrane:** Positioned directly above the stereocilia is a rigid, beige structure labeled as the **Tectorial Membrane**. The tallest stereocilia are physically embedded into or touch the lower surface of this membrane.

The diagram illustrates how mechanical sound vibrations are converted into neural signals. When fluid waves pass through the cochlear chambers, they cause the membranes to vibrate. This movement forces the **stereocilia** to bend against the overlying stationary **tectorial membrane**. The tension on the microscopic **tethers** opens mechanically-gated ion channels, exciting the **hair cell** and sending an electrical signal down the auditory nerve to the brain.

Figure 3.2.5: Transmission of Sound Through the Ear

This figure illustrates the process by which sound waves are transmitted through the ear and converted into neural signals for hearing. The diagram presents a step-by-step sequence, labeled 1 through 5, showing the pathway of sound from the external environment to the sensory receptors within the cochlea.

Step 1: Sound Waves Travel Through the Air

On the left side of the figure is a tuning fork, representing a source of sound. A series of curved blue wave fronts extend from the tuning fork toward the ear.

The figure explains that a **sound wave consists of alternating regions of high and low pressure** moving through the air. Below the wave fronts is a graph illustrating the wave pattern. Dashed vertical lines mark successive peaks, and a double-headed arrow between peaks is labeled **wavelength**.

The graph is labeled **"Frequency of sound wave measured in Hz (cycles per second)."** Frequency represents the number of wave cycles occurring each second and determines the perceived pitch of the sound.

Step 2: Tympanic Membrane Vibrates

The middle portion of the figure shows a cross-sectional view of the external, middle, and inner ear.

As sound waves enter the external auditory canal, they reach the **tympanic membrane (eardrum)**.

A label states, **"Tympanic membrane vibrates in response to sound wave."** Arrows indicate sound energy striking the membrane and causing it to move back and forth in synchrony with the incoming pressure waves.

Step 3: Vibrations Are Amplified by the Ossicles

Behind the tympanic membrane are the auditory ossicles of the middle ear.

A label states, **"Vibrations are amplified across ossicles."** Arrows indicate the transmission of mechanical vibrations through the ossicular chain toward the inner ear.

The ossicles increase the force of vibration before transferring it to the oval window, allowing sound energy to move efficiently

from air into the fluid-filled cochlea.

Step 4: Vibrations Produce Fluid Waves in the Cochlea

A large arrow connects the middle ear to an enlarged cross-sectional view of the cochlea.

The cochlear enlargement identifies three major compartments:

- **Scala vestibuli** (upper chamber)
- **Cochlear duct** (middle chamber)
- **Scala tympani** (lower chamber)

Within the cochlear duct are the **Organ of Corti** and the **Basilar membrane**.

The figure states, **"Vibrations against the oval window set up standing wave in fluid of vestibuli."**

Blue wave symbols and directional arrows indicate that vibration of the oval window creates pressure waves that travel through the fluid contained within the cochlea. These waves move through the scala vestibuli and scala tympani and generate movement within the cochlear duct.

A graph below the cochlear illustration shows a standing wave pattern and is labeled **"Frequency of standing wave is the same as sound wave."** Dashed lines identify repeated wave peaks, emphasizing that the cochlear vibration frequency matches the original sound frequency.

Step 5: Hair Cells Are Stimulated

At the bottom left of the cochlear diagram, a label explains:

"Pressure bends the membrane of the cochlear duct at a point of maximum vibration for a given frequency, causing hair cells in the basilar membrane to vibrate."

The figure shows a localized area of maximum displacement along the basilar membrane. This movement stimulates sensory receptor cells located within the Organ of Corti.

Different sound frequencies produce maximal vibration at different locations along the basilar membrane, allowing the auditory system to distinguish pitch.

The figure demonstrates the sequence of events involved in auditory transduction:

1. Sound waves travel through the air as alternating pressure fluctuations.
2. The tympanic membrane vibrates in response to those pressure changes.
3. The middle-ear ossicles amplify and transmit the vibrations.
4. Vibrations at the oval window generate pressure waves within cochlear fluids.
5. These waves create frequency-specific movement of the basilar membrane.
6. Hair cells within the Organ of Corti bend in response to basilar membrane movement.
7. Mechanical energy is converted into electrical signals by the hair cells.
8. The resulting neural signals are transmitted to the brain, where they are perceived as sound.

The figure explains how sound energy is converted from airborne pressure waves into mechanical vibrations within the ear and ultimately into neural signals by the sensory hair cells of the Organ of Corti, enabling the perception of sound and pitch.

Figure 3.2.6: Maculae of the Utricle and Saccule

This figure illustrates the structure and function of the **maculae** within the **utricle** and **saccule** of the vestibular system and demonstrates how these sensory organs detect head position and linear acceleration through the action of gravity on otoliths.

The figure is composed of four related diagrams showing the location of the macula, its microscopic structure, and its response to changes in head position.

Location of the Macula

In the upper left, a simplified diagram of the inner ear identifies the **utricle** and **saccule**, two vestibular chambers located between the semicircular canals and the cochlea. A sensory region labeled **macula** is highlighted within these structures. An arrow points from this location to an enlarged cross-sectional view of the sensory tissue.

Structure of the Macula

The enlarged cross-sectional view in the lower left shows the organization of the macula within a fluid-filled chamber containing **endolymph**.

The sensory epithelium of the macula contains several labeled structures:

- **Hair cells**, which serve as the sensory receptors.
- **Otolithic membrane**, a gelatinous layer situated above the hair cells.
- **Otoliths**, small calcium carbonate crystals embedded within the otolithic membrane.
- **Vestibular division of the vestibulocochlear nerve**, which receives signals from the hair cells and transmits them to the brain.

The stereocilia of the hair cells project upward into the otolithic membrane. The otoliths rest on top of the membrane, adding mass and making the membrane responsive to gravitational forces and linear movements of the head.

Head Upright Position

In the upper right, a person is shown with the **head upright**. An enlarged view of the macula demonstrates the arrangement of sensory structures during this position.

The **macula** forms a flat sensory surface. Hair cells extend upward into the **otolithic membrane**, which is topped by numerous **otoliths**. When the head is upright and stationary, the membrane remains relatively centered over the hair cells, and the stereocilia are not substantially deflected.

Labels identify the macula, hair cells, otolithic membrane, and otoliths.

Head Tilted Forward

In the lower right, the head is shown **tilted forward**. The corresponding enlargement illustrates the effect of gravity on the sensory apparatus.

A curved arrow labeled **force of gravity** indicates that the weight of the otoliths causes the otolithic membrane to shift relative to the underlying sensory epithelium. As the membrane moves, it bends the hair cell stereocilia in the direction of displacement. The hair cells are shown tilted rather than upright, demonstrating mechanical deformation caused by movement of the otolithic membrane.

This bending changes the activity of the hair cells and alters the signals sent through vestibular nerve fibers.

The figure demonstrates how the utricle and saccule detect head position and linear acceleration.

1. The **macula** contains sensory hair cells embedded within an **otolithic membrane**.
2. The membrane contains dense **otoliths**, which increase its sensitivity to gravity and acceleration.
3. When the head is upright, the otolithic membrane exerts relatively balanced pressure on the hair cells.
4. When the head tilts or undergoes linear acceleration, gravity or inertia causes the otolithic membrane to shift relative to the sensory epithelium.
5. Movement of the membrane bends the stereocilia of the hair cells.
6. Bending of the stereocilia changes the electrical activity of the hair cells.
7. Signals are transmitted through the **vestibular division of the vestibulocochlear nerve** to the brain.
8. The brain interprets these signals to determine head position relative to gravity and detect linear movements.

The figure explains the anatomical organization of the maculae in the utricle and saccule and demonstrates how otolith-mediated displacement of the otolithic membrane bends hair cells, allowing the vestibular system to detect head tilt, static equilibrium, and linear acceleration.

Figure 3.2.7: Dynamic Equilibrium in the Semicircular Canals

This figure explains the **physiology of dynamic equilibrium** in the inner ear. It shows how the **semicircular canals** detect **head rotation** by comparing a resting state on the left with a rotating state on the right.

Left side: Head in still position

The left side shows the vestibular system under resting conditions.

Macroscopic context

In the upper-left corner, a small diagram of the inner ear shows the **semicircular canals**. A black square highlights one of the enlarged bases of a canal, called the **ampulla**.

A large gray arrow points downward to a magnified view of this structure.

Head illustration

Below the inner ear reference is a line drawing of a man with the **head in a still position**, facing forward.

Magnified ampulla diagram

At the bottom is a detailed cross-sectional view of the **ampulla**.

The following structures are shown:

- **Cupula**, a tall, dome-shaped gelatinous structure positioned vertically in the fluid-filled chamber
- **Hair cells**, sensory receptor cells located at the base of the cupula
- **Ampullary nerve**, a bundle of nerve fibers extending downward from the sensory region

The hair bundles of the hair cells project upward into the **cupula**.

In the resting position, the cupula is upright and the hair cells are not strongly deflected.

Right side: Head rotating

The right side shows what happens during head movement.

Head illustration

At the top is a sequence of three overlapping views of the same head, showing the **head rotating** from a forward-facing position to a profile turned to the right.

A large curved gray arrow around the neck shows the direction of rotation.

Magnified ampulla diagram

At the bottom is a second cross-sectional view of the ampulla showing the response to rotation.

Red arrows indicate the relative movement of the surrounding fluid, called **endolymph**, within the ampulla.

The **cupula** is shown bending in the direction opposite the head rotation. Because the hair bundles are embedded in the cupula, this movement bends the hair cells as well.

A caption explains that **as the head rotates, the cupula bends in the opposite direction of the rotation**.

This bending changes the activity of the hair cells and alters the signals sent through the **ampullary nerve**.

This figure demonstrates how the semicircular canals detect **rotational acceleration**.

It shows that:

1. The **ampulla** contains sensory hair cells embedded in the **cupula**.
2. When the head is still, the cupula remains upright and the hair cells are not significantly bent.
3. When the head rotates, the **endolymph** lags behind because of inertia.
4. This relative fluid movement pushes against the cupula.
5. The cupula bends in the direction opposite the head movement.
6. Bending of the cupula deflects the hair bundles of the sensory cells.
7. This changes the electrical activity of the hair cells.
8. Signals are sent through the **ampullary nerve** to the brain, which helps track motion and maintain balance.

The figure shows how the **semicircular canals** detect head rotation by using the movement of **endolymph** to bend the **cupula** and the embedded **hair cells**. This mechanical deformation is converted into nerve signals that help the brain monitor **dynamic equilibrium**, balance, and rotational movement.

Figure 3.2.8: Accessory Structures of the Eye

This figure shows a sagittal (side) cross-sectional view of the anterior portion of the eye and its associated protective structures. The illustration identifies the muscles, membranes, and external features that protect the eye, maintain eyelid function, and help preserve a moist and clear ocular surface.

The eye occupies the central portion of the image and is surrounded by connective tissue, muscles, and the bony orbit. Labels identify the eyebrow, eyelids, conjunctiva, cornea, and muscles involved in eyelid movement.

Anatomical Structures

At the top of the image is the **eyebrow**, located above the eye along the superior margin of the orbit. The eyebrow overlies the tissues surrounding the eye and serves as a protective feature.

Immediately beneath the eyebrow is the **orbicularis oculi muscle**, a circular skeletal muscle that surrounds the eye and extends into the upper and lower eyelids. This muscle is responsible for closing the eyelids during blinking and protective eye closure.

Above the eye is the **levator palpebrae superioris muscle**, which extends from the posterior orbit toward the upper eyelid. This muscle elevates the upper eyelid, allowing the eye to open.

The inner surface of the eyelids is lined by the **palpebral conjunctiva**, a thin mucous membrane that covers the inside of both the upper and lower eyelids.

At the margins of the eyelids are the **eyelashes**, projecting outward from both the upper and lower lids. Eyelashes form a protective barrier at the entrance to the eye.

Covering the visible white portion of the eye is the **ocular conjunctiva** (bulbar conjunctiva). This thin transparent membrane extends from the eyelids onto the anterior surface of the eyeball.

At the front of the eye is the **cornea**, a transparent, dome-shaped structure that covers the anterior portion of the eye and forms the primary refractive surface through which light enters.

Spatial Relationships

The figure demonstrates how the conjunctiva forms a continuous membrane. The **palpebral conjunctiva** lines the inner eyelids and reflects onto the eyeball as the **ocular conjunctiva**, creating a protective continuous surface around the front of the eye.

The cornea is shown projecting anteriorly and is not covered by the ocular conjunctiva, allowing it to remain transparent for vision.

The levator palpebrae superioris muscle is positioned above the eye and inserts into the upper eyelid, while the orbicularis oculi muscle surrounds the eye and eyelids.

The figure illustrates the protective and supportive functions of the accessory structures of the eye:

- The **eyebrow** helps divert sweat, moisture, and debris away from the eye.
- The **eyelashes** help prevent dust and foreign particles from reaching the ocular surface and can trigger protective blinking reflexes.
- The **orbicularis oculi muscle** closes the eyelids during blinking and protects the eye from injury and excessive exposure.
- The **levator palpebrae superioris muscle** raises the upper eyelid, allowing visual access to the environment.
- The **palpebral and ocular conjunctivae** produce and maintain a moist, lubricated surface that protects the eye and reduces friction between the eyelids and eyeball.
- The **cornea** serves as a transparent window that permits light to enter the eye while also contributing significantly to focusing incoming light.

The figure depicts the major accessory structures of the eye, including the eyebrow, eyelashes, conjunctiva, cornea, orbicularis oculi muscle, and levator palpebrae superioris muscle. It demonstrates how these structures work together to protect the eye, maintain lubrication, regulate eyelid movement, keep the ocular surface free of debris, and allow light to enter through the transparent cornea for vision. The overall purpose of the figure is to show the anatomy and functional relationships of the tissues that support and protect the eye.

Figure 3.2.9: Lacrimal Apparatus

This figure illustrates the **lacrimal apparatus**, the system responsible for producing, distributing, collecting, and draining tears from the surface of the eye into the nasal cavity. The diagram shows the anatomical structures involved in tear flow and the pathway tears follow after being secreted.

The eye is shown from an anterior view with surrounding tear-producing and tear-drainage structures labeled.

Anatomical Structures

At the upper outer portion of the eye (upper left of the figure) is the **lacrimal gland**, a lobulated gland located above the eyeball. The lacrimal gland produces tears that help lubricate, clean, and protect the surface of the eye.

Along the inner corner (medial canthus) of the eye are two small openings:

- **Superior lacrimal punctum**, located on the upper eyelid margin.
- **Inferior lacrimal punctum**, located on the lower eyelid margin.

These puncta serve as entry points for tears leaving the eye surface.

Extending from each punctum are small channels:

- **Superior lacrimal canaliculus**
- **Inferior lacrimal canaliculus**

These canaliculi transport tears from the puncta toward the lacrimal sac.

Medial to the eye is the **lacrimal sac**, a dilated collecting chamber that receives tears from the superior and inferior canaliculi.

Inferior to the lacrimal sac is the **nasolacrimal duct**, a tubular passage that carries tears downward into the nasal cavity.

Tear Flow Pathway

The structures are arranged to demonstrate the normal pathway of tear movement:

1. Tears are produced by the **lacrimal gland**.
2. Blinking spreads tears across the anterior surface of the eye, creating a thin protective tear film.
3. Tears migrate toward the inner (medial) corner of the eye.
4. Tears enter the **superior lacrimal punctum** and **inferior lacrimal punctum**.
5. Tears pass through the **superior** and **inferior lacrimal canaliculi**.
6. The canaliculi empty into the **lacrimal sac**.
7. Tears then drain into the **nasolacrimal duct**.
8. The nasolacrimal duct delivers tears into the nasal cavity.

The figure demonstrates how the lacrimal system protects and maintains the eye:

- The **lacrimal gland** produces tears that lubricate the cornea and conjunctiva.
- Tears help remove dust, debris, and microorganisms from the eye surface.
- Antimicrobial substances within tears contribute to protection against infection.
- Blinking distributes tears evenly across the eye.
- The puncta, canaliculi, lacrimal sac, and nasolacrimal duct provide a drainage pathway that prevents excess tears from accumulating on the eye surface.
- Drainage of tears into the nasal cavity explains why increased tear production, such as during crying, may cause a runny nose.

The figure shows the anatomy and function of the lacrimal apparatus, including the lacrimal gland, superior and inferior lacrimal puncta, superior and inferior lacrimal canaliculi, lacrimal sac, and nasolacrimal duct. It illustrates the complete pathway of tear production and drainage, demonstrating how tears are produced, spread across the eye, collected at the medial corner, and transported into the nasal cavity. The overall purpose of the figure is to explain how the lacrimal apparatus maintains eye lubrication, protects the ocular surface, and removes excess tears through an efficient drainage system.

Figure 3.2.10: Extrinsic Muscles of the Right Eye

This figure illustrates the **extrinsic muscles of the right eye** and demonstrates how these muscles attach to the eyeball and produce eye movements. The figure contains two views: a **lateral (side) view of the right eye** on the left and an **anterior (front) view of the right eye** on the right.

Lateral View of the Right Eye

The left panel shows the right eyeball positioned within the orbit and surrounded by several skeletal muscles that control eye movement.

At the posterior portion of the orbit is the **common tendinous ring**, a fibrous structure that serves as the origin for several extraocular muscles.

The following muscles are identified:

- **Superior rectus muscle**, located on the superior surface of the eye and extending from the common tendinous ring to the superior aspect of the eyeball.
- **Inferior rectus muscle**, located on the inferior surface of the eye and extending from the common tendinous ring to the inferior aspect of the eyeball.
- **Lateral rectus muscle**, positioned along the lateral side of the eyeball and attached to the outer surface of the eye.
- **Superior oblique muscle**, located along the superior portion of the orbit. Its tendon passes through a pulley-like structure called the **trochlea** before attaching to the superior surface of the eyeball.
- **Superior oblique tendon**, the continuation of the superior oblique muscle after passing through the trochlea.
- **Inferior oblique muscle**, arising from the anterior floor of the orbit and passing beneath the eye to insert on its posterolateral surface.

The **trochlea** is shown at the anterior superior portion of the orbit. It acts as a pulley that changes the direction of pull of the

superior oblique muscle.

Anterior View of the Right Eye

The right panel presents a frontal view of the right eye with the extraocular muscles arranged around the globe.

The muscles identified include:

- **Superior rectus** at the top of the eye.
- **Inferior rectus** at the bottom of the eye.
- **Medial rectus** along the nasal side of the eye.
- **Lateral rectus** along the temporal side of the eye.
- **Superior oblique**, whose tendon passes through the **trochlea** before attaching to the superior aspect of the globe.
- **Inferior oblique**, which courses beneath the eye and attaches to its inferior-lateral surface.

Red arrows indicate the primary directions of eye movement produced by contraction of these muscles.

- The **superior rectus** elevates the eye (upward movement).
- The **inferior rectus** depresses the eye (downward movement).
- The **medial rectus** turns the eye medially toward the nose (adduction).
- The **lateral rectus** turns the eye laterally away from the nose (abduction).
- The **superior oblique** contributes to downward and rotational movements of the eye.
- The **inferior oblique** contributes to upward and rotational movements of the eye.

Curved arrows around the iris illustrate rotational actions produced by the oblique muscles.

The figure demonstrates how the extraocular muscles work together to control the position and movement of the eye.

1. The four **rectus muscles** primarily move the eye up, down, medially, and laterally.
2. The **superior oblique muscle** passes through the trochlea, which redirects its pulling force and allows it to produce depression and rotational movement of the eye.
3. The **inferior oblique muscle** produces elevation and rotational movement from below the eyeball.
4. Coordinated contraction of these muscles allows the eye to track moving objects, maintain visual fixation, and align both eyes on the same target.
5. The muscles work together to generate precise eye movements necessary for binocular vision, depth perception, reading, and visual scanning.

The figure shows the anatomical arrangement of the extraocular muscles of the right eye and illustrates how the rectus and oblique muscles, acting through their specific attachment points and directions of pull, produce controlled movements of the eyeball in multiple directions.

Figure 3.2.11: Cross Section of the Left Eye

This figure presents a **superior (top) cross-sectional view of the left eye**, illustrating the major anatomical structures involved in vision, support, nourishment, focusing of light, and transmission of visual information to the brain. The image shows the eye within the orbit and identifies the layers of the eyeball, internal chambers, refractive structures, and neural components.

Overall Organization of the Eye

The eyeball is shown as a roughly spherical structure with distinct outer, middle, and inner layers surrounding internal chambers. The front (anterior) portion of the eye contains structures responsible for focusing light, while the posterior portion contains the retina and optic nerve, which process and transmit visual information.

External Eye Muscles

Two extraocular muscles are visible on opposite sides of the eyeball:

- The **lateral rectus muscle** is attached to the lateral side of the eye and helps move the eye away from the midline (abduction).
- The **medial rectus muscle** is attached to the medial side of the eye and helps move the eye toward the midline (adduction).

These muscles work together with other extraocular muscles to position the eye and coordinate visual tracking.

Layers of the Eyeball

The wall of the eyeball consists of three major layers:

Sclera

- The outermost white fibrous layer surrounding most of the eye.
- Provides structural support, protection, and attachment sites for eye muscles.

Choroid

- Located immediately internal to the sclera.
- Contains blood vessels that supply oxygen and nutrients to the retina and absorb excess light within the eye.

Retina

- The innermost sensory layer lining the back of the eye.
- Contains photoreceptors and neural cells responsible for converting light into electrical signals.

Retina and Visual Processing Structures

Several important retinal structures are labeled:

Fovea centralis

- A small depression within the retina.
- Contains the highest concentration of cone photoreceptors and is responsible for the sharpest central vision and greatest visual acuity.

Optic disc (blind spot)

- Region where nerve fibers leave the eye to form the optic nerve.
- Lacks photoreceptors and therefore cannot detect light.

Central retinal artery and vein

- Blood vessels that enter and leave the eye through the optic disc.
- Supply and drain the retina.

Optic (II) nerve

- Also known as the optic nerve.
- Carries visual information from the retina to the brain for processing and perception.

Posterior Cavity

The large compartment behind the lens is labeled the **posterior cavity (vitreous chamber)**.

This chamber is filled with vitreous humor, a clear gel-like substance that:

- Maintains the shape of the eye.
- Supports the retina.
- Allows light to pass through to the retina with minimal distortion.

Anterior Structures Involved in Vision

At the front of the eye are several structures involved in focusing incoming light.

Cornea

- The transparent anterior surface of the eye.
- Acts as the primary refractive structure and bends incoming light toward the retina.

Iris

- Pigmented ring of tissue behind the cornea.
- Controls the size of the pupil.

Pupil

- Central opening within the iris.
- Regulates the amount of light entering the eye.

Lens

- Transparent, flexible structure located behind the iris and pupil.
- Further focuses light onto the retina.

Suspensory Ligaments and Ciliary Body

The lens is held in position by **suspensory ligaments**, which connect it to the **ciliary body**.

The ciliary body consists of:

Ciliary muscle

- Changes the tension on the suspensory ligaments.
- Alters lens shape during accommodation for near and distant vision.

Ciliary processes

- Folded structures that produce aqueous humor.

The figure shows suspensory ligaments extending from the ciliary body to the outer margins of the lens.

Anterior Cavity

The space between the cornea and lens is identified as the **anterior cavity**, which contains aqueous humor.

The anterior cavity is subdivided into:

Anterior chamber

- Located between the cornea and the iris.

Posterior chamber

- Located between the iris and the lens.

Aqueous humor circulates through these chambers, supplying nutrients to avascular structures and helping maintain intraocular pressure.

Drainage System

Near the junction of the cornea and iris is the **scleral venous sinus (canal of Schlemm)**.

This structure:

- Collects aqueous humor from the anterior chamber.
- Drains fluid into the venous circulation.
- Helps maintain normal intraocular pressure.

The figure demonstrates how the anatomical structures of the eye work together to produce vision:

1. Light enters through the **cornea**.
2. The **iris** regulates the amount of incoming light by adjusting the size of the **pupil**.
3. The **lens** fine-tunes focus so that light reaches the retina.
4. The **ciliary muscle** and **suspensory ligaments** alter lens shape to accommodate objects at different distances.
5. Light passes through the vitreous-filled posterior cavity and reaches the **retina**.
6. Photoreceptors in the retina convert light into neural signals.
7. Signals are processed within retinal neurons and converge at the **optic disc**.
8. The **optic nerve (cranial nerve II)** carries visual information to the brain.
9. The **choroid**, retinal blood vessels, aqueous humor, and vitreous humor provide structural support and nourishment necessary for normal visual function.

The figure shows the anatomical organization of the eye and illustrates how its optical, vascular, muscular, and neural structures work together to focus light, maintain ocular health, convert light into nerve impulses, and transmit visual information to the brain for perception.

Figure 3.2.12: Accommodation of the Lens

This figure compares the behavior of the **ciliary muscle** during accommodation for near and distant vision. Two cross-sectional diagrams of the eye's lens and surrounding ciliary body are shown side by side. Red arrows indicate the direction of muscular force and the resulting effect on the lens.

Left Diagram: Relaxed Ciliary Muscle (Distant Vision)

The left image shows the ciliary body arranged around the lens in a relatively expanded position. Red arrows within the ciliary region point inward toward the center, indicating reduced tension generated by the ciliary muscle.

The lens appears flatter and less rounded. In this state, the suspensory ligaments (zonular fibers), although not labeled, would be under greater tension, pulling outward on the lens capsule and maintaining the flattened lens shape.

This configuration is associated with viewing distant objects. A flatter lens has less refractive power and bends incoming light rays to a lesser degree, allowing parallel light rays from distant objects to focus on the retina.

Right Diagram: Contracted Ciliary Muscle (Near Vision)

The right image shows the ciliary muscle in a contracted state. Red arrows point outward from the center toward the periphery, illustrating contraction of the ciliary muscle ring.

As the ciliary muscle contracts, the diameter of the ciliary ring decreases, reducing tension on the suspensory ligaments. With less outward tension, the elastic lens becomes thicker and more rounded.

The lens in this diagram appears more convex than in the left image, reflecting its increased curvature.

This configuration is associated with viewing nearby objects. The rounded lens has greater refractive power and bends incoming light rays more strongly, bringing divergent light from close objects into focus on the retina.

The figure demonstrates the process of **accommodation**, the mechanism by which the eye changes lens shape to focus objects at different distances.

- **Distant Vision (Left):**
  - Ciliary muscle relaxed.
  - Suspensory ligament tension increased.
  - Lens becomes flatter.
  - Refractive power decreases.
  - Light from distant objects is focused on the retina.
- **Near Vision (Right):**
  - Ciliary muscle contracted.
  - Suspensory ligament tension decreases.
  - Lens becomes more rounded.
  - Refractive power increases.
  - Light from nearby objects is focused on the retina.

The figure illustrates how contraction and relaxation of the ciliary muscle alter lens shape during accommodation. By changing the curvature of the lens, the eye adjusts its refractive power to maintain clear vision at different viewing distances. The figure emphasizes the relationship between ciliary muscle activity, lens shape, and the focusing of near versus distant objects onto the retina.

Figure 3.2.13: Internal Structure of the Eye and Layers of the Retina

This figure illustrates the internal structure of the eye and the layered organization of the retina, highlighting the pathway by which light is detected and converted into neural signals. The figure consists of a cross-sectional view of the eye on the left and an enlarged view of the retinal layers on the right.

Overall Structure of the Eye

The left panel shows a sagittal cross section of the eye.

At the front of the eye is the **lens**, positioned behind the anterior structures of the eye. The lens focuses incoming light toward the posterior portion of the eye.

The large central cavity of the eye is filled with **vitreous humor**, a transparent gel that helps maintain the shape of the eyeball and allows light to pass through to the retina.

The inner lining of the posterior eye contains the **retina**, shown as a thin sensory layer along the inner wall of the eyeball.

At the back of the eye, nerve fibers converge and exit the eye through the **optic nerve**, which carries visual information to the brain. Blood vessels are shown traveling through the optic nerve and spreading across the retinal surface to supply retinal tissues.

A highlighted region of the retina is enlarged and displayed in the right panel.

Enlargement of the Retina

The right panel shows the microscopic layers of the retina.

At the top of the enlarged image, arrows labeled **Light** indicate that light enters the retina from the vitreous side and passes through several layers of neurons before reaching the photoreceptors.

The upper retinal layers contain neuronal cell bodies and synaptic connections involved in visual processing.

Visible cell types include:

- **Ganglion cells**, located nearest the vitreous humor. Their axons form the optic nerve.
- **Bipolar cells**, positioned in the middle retinal layers and acting as relays between photoreceptors and ganglion cells.
- Additional interneurons and synaptic networks are shown connecting retinal neurons.

At the bottom of the retinal enlargement are the **photoreceptors**, depicted in two forms:

- **Rods**, shown as long, slender photoreceptors.
- **Cones**, shown as broader photoreceptors interspersed among the rods.

The photoreceptors are located adjacent to a supporting epithelial layer at the outermost portion of the retina.

Direction of Information Flow

The figure demonstrates that the retina is arranged in an inverted orientation relative to incoming light.

1. Light enters the inner retinal surface.
2. Light passes through ganglion cells, bipolar cells, and other retinal layers.
3. Light reaches the rods and cones at the outer retina.
4. Photoreceptors convert light energy into electrical signals.
5. Signals pass from rods and cones to bipolar cells.
6. Bipolar cells transmit information to ganglion cells.
7. Axons of ganglion cells converge to form the optic nerve.
8. The optic nerve carries visual information to the brain for processing and perception.

Function of Major Retinal Cells

- **Rods** are specialized for vision in dim light and are highly sensitive to low levels of illumination.
- **Cones** function primarily in bright light and are responsible for color vision and visual acuity.
- **Bipolar cells** relay sensory information from photoreceptors to ganglion cells.
- **Ganglion cells** generate action potentials that travel through the optic nerve to the brain.

The figure illustrates the anatomical organization of the retina and the pathway of visual information processing. Light enters through the inner surface of the retina, passes through multiple neuronal layers, and reaches photoreceptors located at the outer retina. Rods and cones convert light into neural signals, which are relayed through bipolar cells to ganglion cells. The ganglion cell axons form the optic nerve, transmitting visual information to the brain. The overall purpose of the figure is to demonstrate how retinal structure supports phototransduction and the initial processing of visual information necessary for vision.

Figure 3.2.14: Retina at the Fovea

This figure illustrates the microscopic organization of the **retina** at the **fovea**, the region responsible for the highest visual acuity. The image combines a small inset showing the location of the fovea within the eye and an enlarged cross-sectional view of the retinal layers surrounding the fovea.

Location of the Fovea

In the upper left corner, a small cross-sectional diagram of the eye identifies a highlighted region in the central retina. An arrow points from this highlighted area to the enlarged retinal cross section, indicating that the larger image depicts the structure of the retina at and around the **fovea**.

Retinal Layers

The enlarged image shows the retina as a layered tissue extending from left to right. Several cellular layers and supporting structures are labeled.

Near the inner retinal surface are **ganglion cells**, whose cell bodies form the ganglion cell layer. These neurons receive visual information from other retinal neurons and send their axons toward the optic nerve.

Below the ganglion cell layer are **bipolar cells**, which function as intermediate neurons that relay information from photoreceptors to ganglion cells.

Deeper within the retina are the **photoreceptors**, the sensory receptor cells responsible for detecting light. These cells convert light energy into electrical signals that can be processed by the nervous system.

At the outermost retinal boundary is the **pigment epithelium**, a layer of supporting cells adjacent to the photoreceptors. This layer nourishes photoreceptors, absorbs excess light, and supports normal visual function.

A **capillary** is shown within the inner retinal layers, representing the blood supply that provides oxygen and nutrients to retinal tissues.

Structure of the Fovea

At the center of the image is a depression labeled **fovea**. The retinal surface dips inward, creating a pit-like structure.

Within the fovea, the inner retinal layers, including ganglion cells and much of the bipolar cell layer, are displaced laterally away from the center. This creates a thinner pathway for incoming light to reach the photoreceptors.

The photoreceptor layer remains concentrated within the fovea and occupies the deepest portion of the depression.

Functional Significance of the Fovea

The displacement of inner retinal layers at the fovea minimizes scattering and obstruction of incoming light before it reaches the photoreceptors.

As a result:

- Light has a more direct path to the photoreceptors.
- Visual resolution is increased.
- Fine detail can be detected more accurately.
- Central vision is optimized for tasks such as reading, recognizing faces, and observing small objects.

The fovea contains the highest density of cone photoreceptors and is the region of the retina responsible for the sharpest color vision and greatest visual acuity.

Direction of Visual Information Processing

The figure demonstrates the pathway by which visual information is processed:

1. Light enters the eye and reaches the retinal surface.
2. Light passes through the inner retinal layers.
3. Light reaches the photoreceptors located near the pigment epithelium.

4. Photoreceptors convert light into electrical signals.
5. Signals are transmitted to bipolar cells.
6. Bipolar cells relay information to ganglion cells.
7. Ganglion cell axons transmit visual information to the brain through the optic nerve.

The figure depicts the microscopic structure of the retina at the fovea and illustrates why this region provides the clearest vision. The central depression of the fovea displaces ganglion and bipolar cell layers away from the optical path, allowing light to reach photoreceptors more directly. The figure emphasizes the relationship between retinal anatomy and function by showing how the specialized structure of the fovea supports high-acuity central vision and efficient transmission of visual information to the brain.

Figure 3.3.1: Visual Pathway

This figure illustrates the **visual pathway**, showing how information from both eyes travels through the optic nerves, optic chiasma, optic tracts, thalamus, and ultimately to the visual processing centers in the occipital lobes. The diagram emphasizes the partial crossing of visual fibers at the optic chiasma and demonstrates how visual information from each visual field is processed by the opposite cerebral hemisphere.

The figure consists of a ventral (inferior) view of the brain with both eyes and their associated neural pathways. An inset at the upper left shows the pathway within a sagittal view of the brain.

Structures of the Visual Pathway

At the top of the diagram are the two eyes. The **left eye** is labeled, and each eye is connected to the brain by an **optic nerve**.

The optic nerves from both eyes extend toward the midline and meet at a structure labeled the **optic chiasma**. At this location, some fibers cross to the opposite side while others remain on the same side.

Posterior to the optic chiasma, the pathways continue as the **optic tracts**.

The optic tracts project to structures labeled **thalamus**, representing the thalamic relay nuclei that receive visual input before transmitting information to the cerebral cortex.

Fibers continue posteriorly from the thalamus toward the **occipital lobes**, the primary visual processing regions of the brain.

A **midbrain** structure is also labeled, indicating that some visual fibers project to midbrain centers involved in visual reflexes and eye movements.

Organization of Visual Information

Two color-coded pathways illustrate how visual information is distributed:

- One color represents fibers originating from one half of the retina in each eye.
- The second color represents fibers originating from the opposite half of the retina.

At the **optic chiasma**, fibers arising from the medial (nasal) portion of each retina cross to the opposite side of the brain, while fibers arising from the lateral (temporal) portion remain on the same side.

As a result:

- Visual information from the **left visual field** of both eyes is directed to the **right side of the brain**.
- Visual information from the **right visual field** of both eyes is directed to the **left side of the brain**.

The color-coded tracts demonstrate this redistribution of visual information after crossing at the optic chiasma.

Projections to the Thalamus and Occipital Lobe

The optic tracts terminate primarily in the **thalamus**, where visual information is organized and relayed.

From the thalamus, visual signals are transmitted to the **occipital lobes**, where conscious visual perception occurs. The occipital cortex integrates information received from both eyes to produce a unified visual image.

Midbrain Connections

Some fibers branch toward the **midbrain** rather than continuing exclusively to the thalamus and occipital lobe.

These pathways participate in visual reflex functions, including responses of the pupils to light and reflexive movements of the eyes and head toward visual stimuli.

The figure demonstrates the pathway by which visual information reaches the brain:

1. Light stimulates receptors in both retinas.
2. Retinal signals travel through the **optic nerves**.
3. At the **optic chiasma**, nasal retinal fibers cross to the opposite side while temporal retinal fibers remain on the same side.
4. The reorganized fibers continue through the **optic tracts**.
5. Most fibers synapse in the **thalamus**.
6. Thalamic neurons relay the information to the **occipital lobes** for conscious visual processing.
7. Additional fibers project to the **midbrain** to participate in visual reflexes.
8. Information from corresponding visual fields of both eyes is combined within the opposite cerebral hemisphere, supporting binocular vision and depth perception.

This figure depicts the anatomical visual pathway from the eyes to the brain. It shows the optic nerves converging at the optic chiasma, the partial crossing of retinal fibers, the continuation of fibers as optic tracts, relay through the thalamus, and termination in the occipital lobes. The figure illustrates how visual information from each visual field is processed in the contralateral cerebral hemisphere and highlights both conscious visual processing and reflex pathways associated with vision.

Figure 3.3.2: Major Ascending Sensory Pathways

This figure compares two major ascending sensory pathways that carry somatic sensory information from the body to the brain: the **dorsal column–medial lemniscus system** (left side) and the **spinothalamic tract** (right side). Both pathways are illustrated using sensory input originating from the **right side of the body** and demonstrate the locations of first-order, second-order, and third-order neurons as sensory information travels from peripheral receptors to the primary somatosensory cortex in the **postcentral gyrus**.

An inset at the top left highlights the location of the cerebral cortex being enlarged in the figure.

Left Panel: Dorsal Column System

The left side of the figure depicts the pathway responsible for transmitting **fine touch, vibration, pressure, and conscious proprioception** from the right side of the body.

First-Order Neuron

Sensory information enters the nervous system through a receptor in the right side of the body and travels along a **first-order neuron** whose cell body is located in the **dorsal root ganglion**.

The first-order axon enters the spinal cord and ascends without crossing to the opposite side. It travels within the posterior white columns:

- **Fasciculus gracilis**, carrying information from the lower body.
- **Fasciculus cuneatus**, carrying information from the upper body.

The pathway ascends ipsilaterally through the spinal cord to the medulla.

Second-Order Neuron

Within the **medulla**, the first-order neuron synapses in either the:

- **Nucleus gracilis**
- **Nucleus cuneatus**

The resulting **second-order neuron** crosses to the opposite side of the nervous system in the medulla and then ascends through a fiber tract labeled the **medial lemniscus**.

The medial lemniscus carries the sensory information upward through the brainstem toward the thalamus.

Third-Order Neuron

The second-order neuron synapses within the **thalamus**.

A **third-order neuron** then projects from the thalamus to the **postcentral gyrus**, the location of the primary somatosensory cortex.

At this point, sensory information becomes consciously perceived and interpreted.

Right Panel: Spinothalamic Tract

The right side of the figure depicts the pathway responsible for transmitting **pain and temperature sensations** from the right side of the body.

First-Order Neuron

Sensory receptors detect pain or temperature stimuli and send signals through a **first-order neuron** whose cell body is located in the **dorsal root ganglion**.

The axon enters the spinal cord and quickly synapses within the spinal cord gray matter.

Second-Order Neuron

A **second-order neuron** originates within the spinal cord and crosses to the opposite side almost immediately after entering the spinal cord.

After crossing, the axon ascends through the **lateral spinothalamic tract** located in the anterolateral white matter of the spinal cord.

The pathway continues upward through the brainstem toward the thalamus.

Third-Order Neuron

The second-order neuron synapses within the **thalamus**.

A **third-order neuron** then projects from the thalamus to the **postcentral gyrus**, where conscious perception and localization of pain and temperature occur.

Comparison of the Two Pathways

The figure highlights several important differences:

Dorsal Column-Medial Lemniscus System

- Carries fine touch, vibration, pressure, and conscious proprioception.
- First-order neurons ascend in the spinal cord without crossing.
- Fibers cross in the **medulla**.
- Travels through the **fasciculus gracilis**, **fasciculus cuneatus**, and **medial lemniscus**.
- Projects to the thalamus and then the postcentral gyrus.

Spinothalamic Tract

- Carries pain and temperature information.
- First-order neurons synapse shortly after entering the spinal cord.
- Fibers cross within the **spinal cord**.
- Ascends through the **lateral spinothalamic tract**.
- Projects to the thalamus and then the postcentral gyrus.

The figure demonstrates how somatic sensory information reaches the cerebral cortex through a three-neuron chain:

1. **First-order neurons** carry sensory signals from receptors to the spinal cord or medulla.
2. **Second-order neurons** relay information toward the thalamus and typically cross to the opposite side of the nervous system.
3. **Third-order neurons** project from the thalamus to the primary somatosensory cortex in the postcentral gyrus.

The figure also emphasizes that different sensory modalities travel in distinct pathways. Fine touch and proprioceptive information ascend through the dorsal column–medial lemniscus system and cross in the medulla, whereas pain and temperature information ascend through the spinothalamic tract and cross within the spinal cord.

This figure compares the dorsal column–medial lemniscus system and the spinothalamic tract. It shows the locations of first-order, second-order, and third-order neurons; the fasciculus gracilis, fasciculus cuneatus, medial lemniscus, and lateral spinothalamic tract; and the relay through the thalamus to the postcentral gyrus. The figure demonstrates how different sensory modalities travel from the right side of the body to the contralateral cerebral cortex, where conscious sensory perception occurs.

Figure 3.3.3: Visual Fields and Cortical Processing

This figure illustrates how visual information from the **left and right visual fields** is projected onto the retinas, transmitted to the visual cortex, and integrated into a single visual perception. The diagram demonstrates the organization of visual pathways and the mapping of upper and lower visual fields onto specific regions of the occipital cortex.

The figure is arranged from top to bottom, beginning with the visual scene, continuing through the eyes and visual pathways, and ending with the integrated image perceived by the brain.

Visual Scene

At the top of the figure, the visual environment is divided by vertical and horizontal lines into four quadrants:

- **Left visual field**
- **Right visual field**
- **Upper field**
- **Lower field**

The scene contains a house positioned in the left visual field and trees positioned in the right visual field.

The horizontal line separates objects located in the upper visual field from those located in the lower visual field.

Colored pathways extend from each visual field to the retinas, illustrating how visual information is projected into the eyes.

Projection onto the Retinas

The figure demonstrates that images entering the eyes are inverted and reversed when projected onto the retinas:

- Information from the **left visual field** projects onto the right half of each retina.
- Information from the **right visual field** projects onto the left half of each retina.
- Information from the **upper visual field** projects onto the lower retinal regions.
- Information from the **lower visual field** projects onto the upper retinal regions.

Small inset images positioned beside each eye show inverted retinal images. The house and trees appear upside down on the retina, illustrating the optical inversion produced by the lens.

Visual Pathways to the Brain

Colored pathways extend from the retinas through the visual system and into the occipital lobes.

The figure shows that:

- Information from the **left visual field** is transmitted to the **right visual cortex**.
- Information from the **right visual field** is transmitted to the **left visual cortex**.

The pathways cross at the optic chiasm so that visual information from the same visual field is processed together in the contralateral cerebral hemisphere.

Mapping in the Visual Cortex

The occipital lobes are shown from a superior view and labeled:

- **Superior left visual cortex**
- **Inferior left visual cortex**
- **Superior right visual cortex**
- **Inferior right visual cortex**

The figure demonstrates retinotopic organization within the visual cortex:

- Information from the **upper visual field** projects to the **inferior portions** of the visual cortex.
- Information from the **lower visual field** projects to the **superior portions** of the visual cortex.

Thus, visual information is represented in the cortex in an inverted arrangement relative to the original visual scene.

Integrated Visual Perception

At the bottom of the figure is an image labeled **Integrated image**.

This image illustrates the final visual perception generated by the brain after combining information from both eyes and both hemispheres. The integrated scene contains both the house and the trees as parts of a single continuous visual field. The image remains inverted relative to the original scene shown at the top, emphasizing the retinotopic organization of the visual system. In normal visual perception, higher-order processing interprets these signals so that objects are perceived in their correct orientation.

The figure demonstrates how visual information is organized and processed within the nervous system:

1. Light from the environment enters both eyes.
2. The optical system of the eye projects an inverted image onto the retina.
3. Information from the left and right visual fields is distributed to opposite halves of both retinas.
4. Retinal signals travel through visual pathways, with fibers crossing at the optic chiasm.
5. Information from the left visual field is transmitted to the right occipital cortex, and information from the right visual field is

transmitted to the left occipital cortex.
6. Upper and lower visual fields are mapped to specific superior and inferior regions of the visual cortex.
7. The brain combines information from both eyes to create a unified representation of the visual environment.

This figure illustrates the relationship between the visual fields, retinal image formation, and cortical processing of vision. It shows that images are inverted on the retina, that visual information from each visual field is processed in the opposite cerebral hemisphere, and that upper and lower visual fields project to specific regions of the visual cortex. The figure emphasizes how the brain integrates information from both eyes to produce a single coherent visual perception of the environment.

Figure 3.4.1: Corticospinal Motor Pathway

This figure illustrates the **corticospinal (pyramidal) motor pathway**, showing how voluntary motor commands travel from the **primary motor cortex (precentral gyrus)** in the brain to **skeletal muscles** through a series of upper and lower motor neurons. The diagram traces the pathway from the cerebral cortex through the brainstem into the spinal cord and demonstrates where most fibers cross to the opposite side of the body.

A color key identifies **upper motor neurons** as yellow pathways and **lower motor neurons** as teal pathways.

Origin in the Cerebral Cortex

At the top of the figure, multiple neurons originate in the **precentral gyrus**, the location of the primary motor cortex. The cell bodies of these neurons give rise to long axons that descend from the cerebral cortex and converge into a compact bundle.

These descending fibers represent the **upper motor neurons**, which carry voluntary motor commands from the brain toward the spinal cord.

Passage Through the Midbrain

As the upper motor neuron axons descend, they pass through the **midbrain** and enter a structure labeled the **cerebral peduncle**. The cerebral peduncle serves as a major conduit carrying motor signals from the cerebral cortex to lower regions of the central nervous system.

The fibers remain grouped together as they continue through the brainstem.

Passage Through the Medulla

The descending fibers enter the **medulla**, where they form prominent longitudinal bundles called the **pyramids**. These pyramids contain the corticospinal fibers that convey voluntary motor commands.

Near the lower portion of the medulla, the figure identifies the **decussation of pyramids**, a crossing point where the majority of corticospinal fibers pass from one side of the nervous system to the opposite side.

Formation of the Corticospinal Tracts

After the pyramidal decussation, the descending motor fibers separate into two pathways:

Lateral Corticospinal Tract

Most upper motor neuron fibers cross at the pyramidal decussation and descend within the **lateral corticospinal tract** on the opposite side of the spinal cord.

This pathway is responsible primarily for controlling precise, skilled, voluntary movements of the limbs and distal musculature.

Anterior Corticospinal Tract

A smaller proportion of fibers do not cross at the pyramidal decussation. These fibers continue into the **anterior corticospinal tract**, located in the anterior portion of the spinal cord.

This pathway primarily contributes to control of axial and trunk muscles involved in posture and gross body movements.

Synapse with Lower Motor Neurons

Within the spinal cord, the descending upper motor neurons terminate by forming synapses with **lower motor neurons** located in the anterior horn of the spinal cord.

The figure identifies an **upper motor neuron** ending within the spinal cord and connecting with a **lower motor neuron**.

Lower motor neurons represent the final common pathway between the central nervous system and skeletal muscles.

Exit to Skeletal Muscles

The axons of the lower motor neurons leave the spinal cord through peripheral nerves and travel to **skeletal muscles**, as indicated by the teal pathway and arrow.

When lower motor neurons are activated, they stimulate muscle fibers to contract, producing voluntary movement.

The figure demonstrates the pathway through which the brain controls voluntary movement:

1. Motor commands originate in neurons of the **precentral gyrus (primary motor cortex)**.
2. **Upper motor neuron** axons descend through the **cerebral peduncles** of the midbrain.
3. The fibers continue through the **medulla**, forming the **pyramids**.
4. Most fibers cross to the opposite side at the **decussation of the pyramids**.
5. Crossed fibers descend in the **lateral corticospinal tract**, while a smaller number continue in the **anterior corticospinal tract**.
6. Upper motor neurons synapse with **lower motor neurons** in the spinal cord.
7. Lower motor neuron axons leave the spinal cord and travel to **skeletal muscles**.
8. Activation of skeletal muscles produces voluntary movement.

Because most corticospinal fibers cross at the pyramidal decussation, the **left cerebral hemisphere controls most voluntary movements of the right side of the body**, and the **right cerebral hemisphere controls most voluntary movements of the left side of the body**.

This figure depicts the corticospinal motor pathway from the precentral gyrus to skeletal muscles. It shows upper motor neurons descending through the cerebral peduncles, medullary pyramids, and pyramidal decussation before entering the lateral and anterior corticospinal tracts. The pathway ends when upper motor neurons synapse with lower motor neurons in the spinal cord, whose axons project to skeletal muscles. The figure illustrates how voluntary motor commands are transmitted from the brain to the body and emphasizes the crossing of motor fibers that produces contralateral motor control.

Figure 3.4.2: White Matter Pathways of the Spinal Cord

This figure is a cross-sectional view of the **spinal cord white matter**, illustrating the locations of major **ascending sensory (afferent) pathways** and **descending motor (efferent) pathways**. The pathways are color-coded to distinguish their functions: **blue** indicates ascending sensory tracts carrying information toward the brain, and **red** indicates descending motor tracts carrying commands from the brain to the body. The figure also demonstrates the somatotopic organization of several tracts by labeling regions associated with **sacral, lumbar, thoracic, and cervical** levels of the body.

Overall Organization

The spinal cord is shown in transverse section with gray matter located centrally in a butterfly-shaped arrangement and white

matter surrounding it. Multiple colored regions within the white matter identify specific neural pathways.

A color scale at the top left labels **motor and descending (efferent) pathways** in red. A color scale at the top right labels **sensory and ascending (afferent) pathways** in blue.

Descending Motor Pathways (Red)

The red regions represent pathways that carry motor commands from the brain to lower motor neurons in the spinal cord.

Pyramidal Tracts

These pathways originate primarily in the cerebral cortex and are responsible for voluntary movement.

Lateral corticospinal tract

- Located in the lateral white column.
- The largest descending motor pathway.
- Controls precise, skilled movements, particularly of distal limb muscles.
- Displays somatotopic organization with fibers serving cervical, thoracic, lumbar, and sacral regions arranged within the tract.

Anterior corticospinal tract

- Located in the anterior white column near the midline.
- Primarily controls axial and trunk musculature.
- Contributes to posture and coordinated movements of the body.

Extrapyramidal Tracts

These pathways originate primarily in brainstem nuclei and help regulate posture, muscle tone, balance, and automatic movements.

Rubrospinal tract

- Located in the lateral funiculus.
- Helps coordinate limb movements and motor control.

Reticulospinal tracts

- Located in anterior and lateral regions of the spinal cord.
- Influence posture, locomotion, and muscle tone.

Olivospinal tract

- Shown as a small descending tract.
- Historically described as contributing to motor control, though its existence and functional significance in humans are debated.

Vestibulospinal tract

- Located within the anterior portion of the white matter.
- Helps maintain balance and posture by facilitating activity in antigravity muscles.

Ascending Sensory Pathways (Blue)

The blue regions represent sensory information traveling from the body toward the brain.

Dorsal Column-Medial Lemniscus System

Located in the posterior (dorsal) columns of the spinal cord.

Gracile fasciculus

- Occupies the medial portion of the dorsal column.
- Carries sensory information from the lower trunk and lower limbs.
- Transmits fine touch, vibration, pressure, and conscious proprioception.

Cuneate fasciculus

- Occupies the lateral portion of the dorsal column.
- Carries similar sensory information from the upper trunk and upper limbs.

The dorsal columns are shown with somatotopic organization, labeled from **sacral** medially to **cervical** laterally.

Spinocerebellar Tracts

Located primarily along the lateral aspects of the spinal cord.

Posterior spinocerebellar tract

- Carries unconscious proprioceptive information from muscles and joints to the cerebellum.
- Contributes to coordination of movement.

Anterior spinocerebellar tract

- Also carries proprioceptive information to the cerebellum.
- Provides information related to movement and posture.

Anterolateral System

Located in the lateral and anterior white columns.

Lateral spinothalamic tract

- Carries pain and temperature sensations to the brain.

Anterior spinothalamic tract

- Carries crude touch and pressure sensations.

Together these pathways form the **anterolateral system**, which transmits protective sensory information important for detecting potentially harmful stimuli.

Spino-olivary Fibers

Small ascending fibers shown within the lateral white matter.

- Convey proprioceptive and sensory information to the inferior olivary nucleus.
- Participate in motor learning pathways involving the cerebellum.

Somatotopic Organization

Several tracts display a specific spatial arrangement of fibers based on the body region served.

Labels identify:

- **Sacral**
- **Lumbar**
- **Thoracic**
- **Cervical**

The figure shows that fibers from different body regions occupy distinct positions within both sensory and motor pathways. This organization allows the nervous system to maintain precise information about the origin and destination of neural signals.

The figure demonstrates how the spinal cord serves as a major communication pathway between the body and the brain:

1. **Ascending sensory pathways (blue)** carry information such as touch, vibration, proprioception, pain, temperature, and pressure from peripheral receptors to higher centers in the brain.
2. **Descending motor pathways (red)** carry voluntary and involuntary motor commands from the brain to muscles and glands.
3. The **dorsal column-medial lemniscus system** transmits fine touch, vibration, and conscious proprioception.
4. The **spinocerebellar tracts** provide proprioceptive information needed for movement coordination.
5. The **anterolateral system** conveys pain, temperature, and crude touch sensations.
6. The **corticospinal tracts** control voluntary skeletal muscle movement.
7. **Extrapyramidal tracts** regulate posture, balance, muscle tone, and automatic motor functions.
8. Somatotopic organization preserves information about the specific body regions involved in sensory and motor signaling.

This figure maps the major white matter tracts of the spinal cord and distinguishes sensory pathways from motor pathways. Blue ascending tracts carry sensory information from the body to the brain, while red descending tracts convey motor commands from the brain to the body. The figure emphasizes the locations, functions, and somatotopic organization of the dorsal column-medial lemniscus system, spinocerebellar tracts, anterolateral system, corticospinal tracts, and extrapyramidal pathways, illustrating how the spinal cord integrates and transmits information required for sensation, movement, posture, and coordination.

Figure 3.4.3: Withdrawal Reflex

This figure illustrates a **withdrawal reflex (flexor reflex)**, demonstrating how a painful stimulus applied to the skin produces a rapid, automatic withdrawal of a body part before conscious processing occurs. The diagram shows both the sensory and motor pathways involved in the reflex arc and includes an inset cross section of the spinal cord where neural processing takes place.

The main image depicts a person touching the flame of a candle with a finger. Arrows identify the pathways that carry sensory information to the spinal cord and motor commands back to the muscles of the arm.

Painful Stimulus

At the lower left of the figure, a finger comes into contact with the flame of a lit candle. The flame represents a potentially damaging thermal stimulus.

Specialized sensory receptors in the skin detect the painful heat and generate nerve impulses. These impulses travel along a **sensory (afferent) neuron**, represented by a blue pathway extending from the finger toward the spinal cord.

Sensory Input to the Spinal Cord

The blue afferent pathway enters the spinal cord through a dorsal root and carries information into the gray matter of the spinal cord.

An enlarged spinal cord cross section in the upper right shows the sensory neuron entering the spinal cord and synapsing with interneurons. The sensory information remains within the spinal cord for rapid processing rather than first traveling to the brain.

Integration Within the Spinal Cord

Inside the spinal cord, interneurons receive the incoming sensory signal and connect it to motor neurons.

The reflex is processed at the spinal cord level, allowing a response to occur quickly. The figure illustrates that the sensory signal activates motor pathways responsible for withdrawing the stimulated limb.

Although the reflex is coordinated within the spinal cord, ascending pathways also transmit sensory information toward the brain, enabling conscious awareness of pain after the withdrawal response has begun.

Motor Output

A **motor (efferent) neuron**, represented by a red pathway, exits the spinal cord through the ventral root and travels to skeletal muscles in the arm.

The motor neuron carries signals to the flexor muscles of the upper limb. In the figure, the muscle in the upper arm is highlighted to indicate activation.

When these muscles contract, the forearm bends at the elbow, moving the hand away from the painful stimulus.

Withdrawal Response

A curved arrow near the arm indicates the direction of movement. The hand is shown lifting away from the candle flame as a result of muscle contraction.

This rapid withdrawal helps minimize tissue injury by removing the body part from the source of pain before conscious decision-making is required.

The figure demonstrates the sequence of events in a withdrawal reflex:

1. A painful heat stimulus activates sensory receptors in the skin.
2. Sensory neurons carry the information to the spinal cord through an afferent pathway.
3. Interneurons within the spinal cord process the signal.
4. Motor neurons are activated without requiring initial input from the brain.
5. Motor commands travel through efferent pathways to skeletal muscles.
6. Flexor muscles contract and withdraw the limb from the harmful stimulus.
7. Sensory information simultaneously travels to higher brain centers, allowing conscious perception of pain after the reflex has been initiated.

This figure illustrates a spinal withdrawal reflex in response to a painful thermal stimulus. It shows a sensory neuron carrying pain information from the finger to the spinal cord, interneurons integrating the signal, and a motor neuron activating arm muscles to pull the hand away from a candle flame. The figure emphasizes that reflexes provide a rapid protective response by using spinal cord circuits to produce movement before conscious awareness and voluntary control occur.

Figure 3.4.4: Stretch Reflex and Reciprocal Inhibition

This figure illustrates the **stretch reflex (patellar reflex or knee-jerk reflex)** and the associated process of **reciprocal inhibition**. The diagram combines a real-world example of a patellar tendon tap with a schematic representation of the neural pathways involved in the reflex. Numbered labels (1 through 8) identify the sequence of events from muscle stretch to muscle contraction and relaxation.

Overview

On the right side of the figure, an individual is seated while a reflex hammer strikes the **patellar tendon** just below the knee. The lower leg is shown extending forward in response to the tendon tap, demonstrating the knee-jerk reflex.

The remainder of the figure shows the sensory receptors, spinal cord circuitry, and motor pathways responsible for producing this automatic response.

Step 1: Extensor Muscle Is Stretched

The reflex begins when the patellar tendon is tapped. The force briefly stretches the **extensor muscle** of the thigh (quadriceps muscle group).

The stretched muscle is labeled **"Extensor muscle stretched."**

Step 2: Muscle Spindle Is Stimulated

Within the extensor muscle is a sensory receptor called a **muscle spindle**. Stretching the muscle elongates the muscle spindle and activates sensory nerve endings within the receptor.

The muscle spindle detects changes in muscle length and the rate of stretch.

Step 3: Primary Afferent Neuron Is Excited

Activation of the muscle spindle generates nerve impulses in a **primary afferent neuron** (sensory neuron).

The sensory signal travels from the muscle toward the spinal cord through the afferent pathway.

Step 4: Primary Afferent Neuron Stimulates Alpha Motor Neuron to Extensor Muscle

Within the spinal cord, the sensory neuron forms a direct excitatory synapse with an **alpha motor neuron** that innervates the same extensor muscle.

This monosynaptic connection allows the reflex to occur rapidly without requiring processing by higher brain centers.

The figure also shows that sensory information simultaneously travels upward **to the brain**, providing awareness of the event after the reflex has begun.

Step 5: Alpha Motor Neuron Stimulates Extensor Muscle to Contract

The activated alpha motor neuron sends motor impulses back to the extensor muscle.

As a result, the extensor muscle contracts, indicated by a green arrow within the muscle. Contraction of the quadriceps muscle causes extension of the lower leg at the knee joint.

This contraction opposes the initial stretch and helps restore muscle length.

Step 6: Primary Afferent Neuron Stimulates an Inhibitory Interneuron

The sensory neuron also activates an **inhibitory interneuron** within the spinal cord.

This interneuron participates in reciprocal inhibition by suppressing activity in the motor neurons controlling the opposing muscle group.

Step 7: Interneuron Inhibits Alpha Motor Neuron to Flexor Muscle

The inhibitory interneuron synapses with an alpha motor neuron that innervates the **flexor muscle**, the antagonist of the extensor muscle.

The interneuron decreases motor neuron activity, preventing contraction of the flexor muscle during the reflex.

Step 8: Flexor Muscle (Antagonist) Relaxes

Because the motor neurons supplying the flexor muscle are inhibited, the **flexor muscle relaxes**.

Relaxation of the antagonist muscle reduces resistance to movement and allows the extensor muscle to produce an efficient extension of the leg.

A red arrow within the flexor muscle indicates reduced activity and relaxation.

The figure demonstrates two closely related processes:

Stretch Reflex

1. A tendon tap stretches the extensor muscle.
2. Muscle spindles detect the stretch.
3. Sensory neurons transmit the signal to the spinal cord.
4. Sensory neurons directly stimulate alpha motor neurons.
5. Alpha motor neurons cause the stretched muscle to contract.
6. The contraction opposes the stretch and helps maintain muscle length and posture.

Reciprocal Inhibition

1. The sensory neuron simultaneously activates an inhibitory interneuron.
2. The interneuron inhibits motor neurons supplying the antagonist muscle.
3. The antagonist (flexor) muscle relaxes.
4. Relaxation of the opposing muscle allows the reflex movement to occur more effectively.

This figure illustrates the neural circuitry of the **stretch reflex** and **reciprocal inhibition** using the patellar reflex as an example. A tendon tap stretches the extensor muscle, activating muscle spindles and sensory neurons. The sensory neurons directly excite alpha motor neurons that cause the extensor muscle to contract while simultaneously activating inhibitory interneurons that suppress motor activity in the opposing flexor muscle. The coordinated contraction of the extensor muscle and relaxation of the antagonist muscle produces the characteristic knee-jerk response and helps maintain posture, muscle tone, and joint stability.

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4: THE AUTONOMIC NERVOUS SYSTEM

Chapter Overview



Figure 4.1: Fight or Flight? Though the threats that modern humans face are not large predators, the autonomic nervous system is adapted to this type of stimulus. The modern world presents stimuli that trigger the same response. (CC BY 4.0; via LibreTexts).

The autonomic nervous system (ANS) is the part of the nervous system that automatically regulates the body's internal environment. Alongside the endocrine system, it maintains homeostasis by adjusting heart rate, blood pressure, breathing, digestion, temperature, and many other functions without conscious effort. The ANS has two main divisions with generally opposing effects: the sympathetic division, which prepares the body for "fight-or-flight" during stress or threat, and the parasympathetic division, which supports "rest-and-digest" activities when the body is relaxed. Understanding the ANS requires following its output pathways, how it controls target organs, how it is regulated by reflexes and higher brain centers, and how drugs can alter its function.

Sympathetic Division: Thoracolumbar Outflow and Fight-or-Flight

Sympathetic output arises from neurons in the lateral horn of the thoracolumbar spinal cord. Preganglionic axons leave via the ventral root and spinal nerve to synapse in sympathetic chain ganglia or collateral ganglia. These preganglionic fibers release acetylcholine (ACh) onto nicotinic receptors, exciting ganglionic neurons. Postganglionic fibers then project to target organs and usually release norepinephrine onto adrenergic receptors, producing widespread, coordinated responses such as increased heart rate, bronchodilation, and pupil dilation. An important exception is the sympathetic innervation of sweat glands and some blood vessels in skeletal muscle, where postganglionic fibers release ACh onto muscarinic receptors. The sympathetic system also has a specialized pathway to the adrenal medulla: preganglionic fibers synapse on chromaffin cells, triggering release of epinephrine and norepinephrine into the bloodstream. This hormonal component allows rapid, body-wide amplification of the sympathetic response.

Parasympathetic Division: Craniosacral Outflow and Rest-and-Digest

Parasympathetic output originates in specific nuclei of the brain stem and in the sacral spinal cord. Preganglionic neurons project via cranial nerves (especially the vagus nerve) and sacral nerves to terminal (intramural) ganglia located close to or within

the walls of target organs. These preganglionic fibers also release ACh onto nicotinic receptors to excite ganglionic neurons. Short postganglionic fibers within or near the organ then release ACh onto muscarinic receptors on target tissues, promoting rest-and-digest activities such as decreased heart rate, increased glandular secretion, and enhanced digestive motility. Because many parasympathetic ganglia are close to their effectors and pathways are less divergent, parasympathetic effects tend to be more localized and specific than sympathetic effects.

Autonomic Signaling: Neurotransmitters, Hormones, and Varicosities

The ANS uses a small set of signaling molecules—primarily ACh, norepinephrine, and epinephrine—which may act as neurotransmitters at synapses or as hormones when released into the bloodstream. For example, norepinephrine released from a sympathetic postganglionic axon acts as a neurotransmitter; the same molecule released from the adrenal medulla into the blood acts as a hormone. Autonomic synapses also differ structurally from the classic neuromuscular junction. Many postganglionic fibers form a series of swellings called varicosities along their length, creating a network of release sites that bathe the target tissues in neurotransmitter over a broader area.

Visceral Reflexes and Autonomic Control of Organs

Autonomic function is organized around visceral reflexes, which resemble somatic reflexes but use a two-neuron efferent pathway.

A central (preganglionic) neuron in the spinal cord or brain stem synapses on a ganglionic (postganglionic) neuron, which then innervates the effector. The afferent (sensory) limb of visceral reflexes is similar to that of somatic reflexes, and many somatic and special senses can trigger autonomic responses. There are also visceral sensory inputs—such as distension or ischemia—that normally do not reach conscious awareness. When visceral pain is intense, it may be perceived, often as referred pain (for example, left shoulder and arm pain during a myocardial infarction) because the sensory cortex represents internal organs less precisely than body surface structures.

Autonomic Tone and Sympathetic vs. Parasympathetic Balance

The sympathetic and parasympathetic divisions usually provide opposing influences to the same organs, creating a dynamic balance called autonomic tone. The sympathetic division generally increases heart rate and dilates pupils, whereas the parasympathetic division slows heart rate and constricts pupils. At rest, heart rate is normally under parasympathetic (vagal) tone, which keeps it lower than the intrinsic rate of the pacemaker cells. In contrast, many blood vessels are under sympathetic tone because they lack significant parasympathetic innervation; sympathetic activity maintains a slight baseline vasoconstriction that supports blood pressure. Some effectors, such as most blood vessels and sweat glands, receive only sympathetic input, so their activity is regulated by increases or decreases in sympathetic firing rather than by direct parasympathetic opposition.

Central Control of the Autonomic Nervous System

Autonomic output is integrated with sensory information and higher brain functions in central autonomic networks. The hypothalamus is the principal coordinating center, regulating both sympathetic and parasympathetic efferent pathways to control cardiovascular, respiratory, digestive, thermoregulatory, and other homeostatic functions. Major hypothalamic outputs travel through the medial forebrain bundle and the dorsal longitudinal fasciculus to brain stem and spinal autonomic centers. The medial forebrain bundle also connects the hypothalamus with the limbic system, allowing emotional states to influence visceral responses (for example, increased heart rate during anxiety). The amygdala, a key limbic structure, modulates hypothalamic activity and thus links emotion, autonomic responses, and endocrine function.

Brain Stem Autonomic Centers

Brain stem nuclei, especially in the medulla oblongata, provide important reflex control of autonomic function. The cardiovascular center (a group of medullary nuclei) regulates heart rate, contractility, and vascular tone based on sensory input from the heart, aorta, and carotid sinuses. The solitary nucleus receives this visceral sensory information and can increase sympathetic output via cardiac accelerator and vasomotor pathways. The nucleus ambiguus and dorsal motor nucleus of the vagus contribute parasympathetic fibers to the vagus nerve, slowing heart rate and influencing other thoracic and abdominal organs. These centers work continuously with the hypothalamus to maintain blood pressure, cardiac output, and other vital parameters.

Drugs and the Autonomic Nervous System

Many exogenous substances, both therapeutic and illicit, alter autonomic function by mimicking or blocking endogenous neurotransmitters or their receptors. Studying these drugs helps clarify how the ANS works and how its pathways can be targeted clinically.

Nicotine and General Ganglionic Stimulation

Nicotine (used primarily in tobacco products and some smoking-cessation therapies) stimulates nicotinic ACh receptors at autonomic ganglia, broadly activating both sympathetic and parasympathetic divisions. In many organ systems, these opposing postganglionic influences partially cancel each other. However, because most systemic blood vessels lack parasympathetic innervation, nicotine-induced sympathetic stimulation increases vascular tone and blood pressure, contributing to cardiovascular risk. In the heart, where intrinsic pacemaker activity is modulated rather than initiated by autonomic input, simultaneous sympathetic and parasympathetic stimulation can disturb rhythm and promote arrhythmias—another risk factor for heart disease.

Sympathomimetic and Sympatholytic Drugs

Drugs that mimic the actions of norepinephrine and epinephrine are called sympathomimetic agents. For example, phenylephrine binds to adrenergic receptors and produces effects similar to sympathetic activation (such as vasoconstriction and nasal decongestion). Sympatholytic drugs block adrenergic receptors or interfere with norepinephrine release, reducing sympathetic influence on target organs; many antihypertensive medications act in this way.

Parasympathomimetic and Anticholinergic Drugs

Drugs that enhance parasympathetic signaling, such as muscarinic agonists (parasympathomimetics), act like ACh released from postganglionic parasympathetic fibers, increasing rest-and-digest activities. In contrast, anticholinergic drugs block muscarinic receptors and suppress parasympathetic effects, leading to responses such as increased heart rate, reduced glandular secretions, and pupil dilation. These agents are widely used in medicine but can also produce significant side effects because parasympathetic pathways are so broadly involved in normal function.

Together, these topics provide a framework for understanding how the autonomic nervous system maintains internal stability, coordinates rapid responses to changing conditions, and serves as an important target for many commonly used drugs and toxins.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- Describe the overall role of the autonomic nervous system (ANS) in maintaining internal homeostasis and distinguish it from somatic motor control.
- Compare the sympathetic and parasympathetic divisions in terms of origins, pathways, neurotransmitters, and typical “fight-or-flight” versus “rest-and-digest” effects.
- Explain how autonomic signaling works at effectors, including the roles of acetylcholine, norepinephrine, epinephrine, and varicosities.
- Describe the basic organization of visceral reflexes and how autonomic pathways control target organs.
- Explain the concept of autonomic tone and how sympathetic–parasympathetic balance regulates key organs such as the heart and blood vessels.
- Summarize how hypothalamus and brain stem centers integrate and regulate autonomic function and link it to emotional states.

- Give examples of how selected drugs (e.g., nicotine, sympathomimetics, parasympathomimetics, and their blockers) modify autonomic activity and related physiological effects.

4.1: Divisions of the Autonomic Nervous System
4.2: Autonomic Synapses, Effects and Reflexes
4.3: Central Control Autonomic Function
4.4: Drugs that Affect the Autonomic System
4.5: Terms and Definitions
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4.1: Divisions of the Autonomic Nervous System

Comparison between the Somatic and Autonomic Nervous System

The nervous system can be divided into two functional parts: the **somatic nervous system (SNS)** and the **autonomic nervous system (ANS)**. The major difference between these two systems is based on whether you are conscious of its process. The somatic nervous system consciously detects sensory stimuli from the special senses, skin and proprioceptors. The autonomic nervous system reflexively responds to visceral sensory stimuli, such as levels of carbon dioxide concentration in the blood or stretch caused by blood pressure, that you are not consciously aware of. Moreover, the motor efferent branches of these two systems innervate different **target effectors**. While the somatic motor neurons innervate and cause contraction of skeletal muscles, autonomic motor neurons innervate and control cardiac and smooth muscle, as well as glandular tissue. Thus, the motor response of the somatic nervous system is voluntary while the one of the autonomic nervous system is involuntary.

Another major difference between these two systems lies within the number of lower motor neurons that are involved in the response. In the somatic nervous system, a single lower somatic motor neuron of the brainstem or spinal cord extends from the CNS towards a skeletal muscle through a cranial or spinal nerve, respectively. These somatic motor neurons have large myelinated axons that release acetylcholine (ACh) at neuromuscular junctions. In comparison, the autonomic nervous system is composed of a chain of two lower motor neurons. The cell body of the first of the two ANS motor neurons is located in the brainstem or spinal cord and is called a **preganglionic neuron**. The axon of the preganglionic neuron extends outside of the CNS through cranial or spinal nerves forming a **preganglionic fiber**. This fiber projects to an **autonomic ganglion** of the peripheral nervous system. Preganglionic neurons have small myelinated axons that release acetylcholine (ACh) to excite a second motor neuron. The second motor neuron is called a **ganglionic neuron**. The cell body of the ganglionic neuron resides within the autonomic ganglion and its axon extends to an effector (cardiac muscle, smooth muscle, or gland) forming a **postganglionic fiber**. Ganglionic neurons have small unmyelinated axons that release either acetylcholine (ACh) or norepinephrine (NE) to either excite or inhibit an effector, depending on the type of receptors present on the effector. Since preganglionic and postganglionic axons are small or unmyelinated, the propagation of autonomic electrical impulses is slower compared to the somatic motor axons.

Divisions of the Autonomic Nervous System

The autonomic nervous system regulates many of the internal organs through a balance of two aspects, or divisions. The two divisions of the autonomic nervous system are the **sympathetic division** and the **parasympathetic division**. From a functional point of view, the sympathetic system is associated with the **fight-or-flight response**, while the parasympathetic activity is referred to by the epithet **rest-and-digest**. Homeostasis is the balance between the two divisions since one system complements the other. For example, the parasympathetic division will be more active when you need to conserve energy and replenish nutrient stores. The sympathetic division will activate when during exercise, stress or emergency situations. Many autonomic target effectors have dual innervation by both divisions of the autonomic nervous system, which determines their activity. For example, the heart receives connections from both the sympathetic and parasympathetic divisions: one causes heart rate to increase, whereas the other causes heart rate to decrease. From an anatomical point of view, both divisions use preganglionic and ganglionic neurons to innervate cardiac muscle, smooth muscle and glands. However, the location of preganglionic neurons within the CNS is different between the two divisions. Moreover, the location of the ganglia as well as the length of preganglionic and postganglionic axons differ in the two divisions. In the following sections, you will examine the functional and anatomical features of the sympathetic and parasympathetic divisions.

Sympathetic Division

To respond to a threat—to fight or to run away—the sympathetic system causes diverse effects as many different effector organs are activated together for a common purpose. More oxygen needs to be inhaled and delivered to skeletal muscle. The respiratory, cardiovascular, and musculoskeletal systems are all activated together. Additionally, sweating keeps the excess heat that comes from muscle contraction from causing the body to overheat. The digestive system shuts down so that blood is not absorbing nutrients when it should be delivering oxygen to skeletal muscles. To coordinate all these responses, the connections in the sympathetic system diverge from a limited region of the central nervous system (CNS) to a wide array of ganglia that project to the many effector organs simultaneously. The complex set of structures that compose the output of the sympathetic system make

it possible for these disparate effectors to come together in a coordinated, systemic change.

The sympathetic division of the autonomic nervous system influences various organ systems of the body through connections emerging from the first thoracic (T1) and second lumbar (L2) spinal segments (Figure 4.1.1). It is referred to as the **thoracolumbar system** to reflect this anatomical basis. Sympathetic preganglionic neurons are located in the lateral horns of any of these spinal regions and project to ganglia adjacent to the vertebral column through the ventral roots of the spinal cord.

Sympathetic Neurons, Ganglia and Nerves

The majority of ganglia of the sympathetic system belong to a network of **sympathetic chain (or trunk) ganglia** that runs lateral to the vertebral column and anterior to the paired spinal nerves (Figure 4.1.1). For this reason, these ganglia can also be called **paravertebral ganglia**. The ganglia appear as a series of clusters of neurons linked by ascending and descending axonal bridges called **sympathetic trunks**. There are typically 23 ganglia in the sympathetic chain on either side of the spinal column. Three correspond to the cervical region, 12 are in the thoracic region, four are in the lumbar region, and four correspond to the sacral region. The thoracic and lumbar sympathetic preganglionic fibers travel sequentially through ventral roots, spinal nerves and bundles of myelinated axons called **white rami communicantes** (singular = ramus communicans) to reach the correspondent paravertebral ganglia (Figure 4.1.2). Here preganglionic sympathetic fibers either synapse with ganglionic neurons or often pass on through the sympathetic chain ganglion into one of its emerging nerves to synapse with ganglionic neurons elsewhere. Postganglionic fibers then travel through additional nerves to their destination in one of the organs. Many of the fibers from the postganglionic neurons in the sympathetic chain ganglia pass back into the spinal nerves through **gray rami communicantes** composed of unmyelinated axons and carry sympathetic information through the spinal nerves.

The cervical and sacral paravertebral ganglia are not connected to the spinal cord directly through the spinal nerves, but through sympathetic trunks. Among the cervical ganglia, the **superior cervical ganglion** contains ganglionic neurons that innervate structures of the head and neck such the dilator pupillae and superior tarsal muscles of the eye, the lacrimal gland, mucous membranes of the nose, palate and mouth, and salivary glands. The **middle and inferior cervical ganglia** contain ganglionic neurons that innervate neck and thoracic organs such as the larynx, trachea, pharynx, smooth muscle of arteries and heart.

Preganglionic sympathetic axons extending from T5-L2 do not synapse in a sympathetic chain ganglion and instead continue through the chain anteriorly towards the abdominal and pelvic organs (Figure 4.1.1). These axons form **splanchnic nerves** and typically terminate in three autonomic ganglia called **prevertebral (or collateral) ganglia**. They are referred to as prevertebral because they are anterior to the vertebral column and descending aorta. The prevertebral ganglia are associated with controlling organs in the abdominal cavity, and are also considered part of the enteric nervous system. The **greater splanchnic nerves** originate from T5-T9 spinal nerves and synapse into the prevertebral **celiac ganglia**. Postganglionic axons from these ganglia innervate stomach, abdominal blood vessels, liver, gallbladder, part of the pancreas and small intestine. The **lesser splanchnic nerves** originate from T9-T11 spinal nerves and project to prevertebral **superior mesenteric ganglia**. Postganglionic axons from these ganglia innervate the large intestine. The **least splanchnic nerves** that extend from T12 spinal nerves project to and terminate in the prevertebral renal ganglia (not shown here). Postganglionic axons from these ganglia innervate the remainder of the pancreas and small intestine, the proximal part of the large intestine, the kidneys and proximal ureters. **Lumbar splanchnic nerves** that extend from L1-L2 spinal nerves terminate to the **inferior mesenteric ganglia**. Postganglionic axons from these ganglia project to and innervate the distal part of the large intestine, rectum, kidneys, urinary bladder, gonads and external genitalia. In addition to the above splanchnic nerves, there are also small **sacral splanchnic nerves** that originate from the sacral sympathetic ganglia that are not directly connected to the spinal cord and terminate into urinary and reproductive organs.

The neurons of the sympathetic autonomic ganglia are multipolar in shape, with dendrites radiating out around the cell body where synapses from the spinal cord neurons are made. Because the sympathetic ganglia are adjacent to the vertebral column, preganglionic sympathetic fibers are relatively short, and they are myelinated. Compared with the preganglionic fibers, postganglionic sympathetic fibers are long because of the relatively greater distance from the ganglion to the target effector. These postganglionic fibers are unmyelinated. A diagram that shows the connections of the sympathetic system is somewhat like a circuit diagram that shows the electrical connections between different receptacles and devices. In Figure 4.1.1, the “circuits” of the sympathetic system are intentionally simplified.

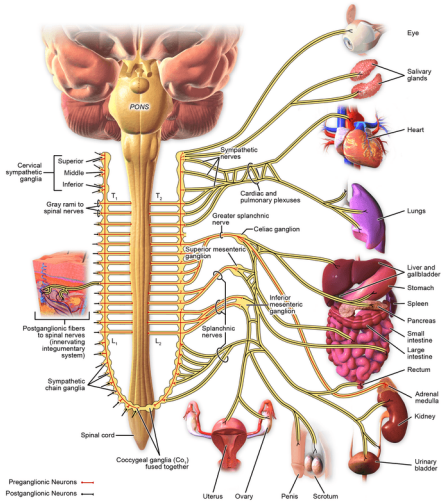


Figure 4.1.1: Neurons from the lateral horn of the spinal cord extend their preganglionic nerve fibers to the sympathetic chain ganglia on either side of the vertebral column or to prevertebral (collateral) ganglia that are anterior to the vertebral column in the abdominal cavity. Axons from these ganglionic neurons called postganglionic nerve fibers then project to target effectors throughout the body. (CC BY 3.0; "Blausen 0838 Sympathetic Innervation" by BruceBlaus). Alternate text description of the Figure 4.1.1.

Sympathetic Pathways

A sympathetic preganglionic axon leaving the lateral horn of the thoracolumbar spinal cord enters the sympathetic chain ganglia, where it branches toward 10-20 targets. To continue with the analogy of the circuit diagram, there are four different types of "junctions" that connect the sympathetic preganglionic axons with their effectors. The type of pathway is determined by the location and type of target effector organ being innervated. In all cases, the preganglionic axon extends into the spinal nerve at the same level as its spinal cord segment. Then it can then either (a) synapse in the paravertebral ganglion and carry information through the spinal nerve at the same level (spinal nerve pathway), (b) ascend to a more superior or descend to a more inferior paravertebral ganglion, synapse there and carry information through sympathetic nerves (sympathetic nerve pathway), (c) descend to a prevertebral (collateral) ganglion, synapse there and carry information through a splanchnic nerve (splanchnic nerve pathway) or (d) project directly to the adrenal medulla (adrenal medulla pathway). All of these branches mean that one preganglionic neuron can influence different regions of the sympathetic system very broadly, by acting on widely distributed organs. In the following paragraphs, you will examine these four different pathways. Except for the adrenal medulla pathway, these connections are represented in Figure 4.1.2.

The **spinal nerve pathway** is the most direct connection. The sympathetic preganglionic nerve projects to the sympathetic chain ganglion at the same level as the target effector. The myelinated preganglionic fiber extending from the lateral horns of the spinal cord projects to the sympathetic chain ganglion through the ventral root and spinal nerve. Through the white ramus communicans, the fiber reaches and synapses with the ganglionic neuron in the sympathetic chain ganglion. The postganglionic fiber then projects to the target effector via the gray ramus communicans, which is formed by unmyelinated axons. An example of this type is spinal nerve T1 that synapses with the T1 sympathetic chain ganglion to innervate the skin (Figure 4.1.2a). Indeed, this pathway generally innervates integumentary structures such as sweat glands, arrector pili muscles, and blood vessels of the skin in the neck, torso and limbs.

The **postganglionic sympathetic nerve pathway** occurs when the target effectors are located superior or inferior to the spinal segment at which the sympathetic preganglionic fiber emerges. With respect to the "wiring" involved, the synapse with the ganglionic neuron occurs at sympathetic chain ganglia superior or inferior to the location of the preganglionic neuron. In order to do this, the preganglionic fiber travels through sympathetic trunks to reach the superior or inferior sympathetic chain ganglion. The postganglionic fiber does not leave the ganglion through the gray ramus communicans. Instead, it extends away from the ganglion through a sympathetic nerve. An example of this is spinal nerve T1 that innervates the eye. The spinal nerve tracks up through the sympathetic trunks until it reaches the superior cervical ganglion, where it synapses with the ganglionic neuron and projects to the eye through a sympathetic nerve (Figure 4.1.2b). This pathway innervates viscera of head (sweat glands, arrector pili muscles, blood vessels of the skin; dilator pupillae, tarsal muscle and gland of the eye; salivary glands) and neck, and thoracic organs such as esophagus, heart, lungs, thoracic blood vessels.

Not all axons from the central neurons terminate in the sympathetic chain ganglia. The **splanchnic nerve pathway** include branches from the ventral nerve root that continue through the sympathetic chain ganglion and on to one of the prevertebral (collateral) ganglia as the greater splanchnic nerve or lesser splanchnic nerve. For example, the greater splanchnic nerve at the level of T5 synapses with a prevertebral (collateral) ganglion outside the sympathetic chain before making the connection to the postganglionic nerves that innervate the stomach (Figure 4.1.2*c*). This pathway innervates abdominopelvic organs such as stomach, intestines, kidneys, ureters, urinary bladder and reproductive organs.

There is one additional way that preganglionic sympathetic fibers can control their effector organs and it is through the **adrenal medulla pathway**. However, in this pathway the preganglionic fiber does not terminate in a ganglion but instead projects to the **adrenal medulla**, the interior portion of the adrenal gland (Figure 4.1.1). These axons are still referred to as preganglionic fibers, but the target is not a ganglion per se. The cells in the adrenal medulla that are contacted by the preganglionic fibers are called **chromaffin cells**. These cells are neurosecretory cells that develop from the neural crest along with the sympathetic chain ganglia. These cells in the adrenal medulla release epinephrine and norepinephrine into the bloodstream, rather than using axons to communicate with target structures. This hormonal component means that the sympathetic chemical signal can spread throughout the body very quickly and affect many organ systems at once. At the same time, these hormones remain in the bloodstream longer than neurotransmitters, prolonging the sympathetic effects.

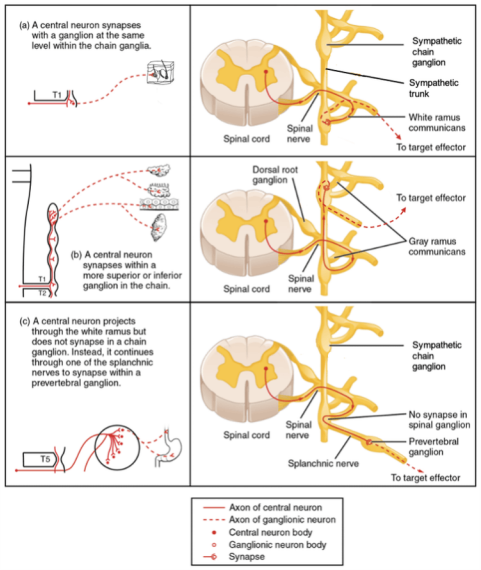


Figure 4.1.2: The axon from a central sympathetic neuron in the spinal cord can project to the periphery in a number of different ways. (a) represents the spinal nerve pathway where the preganglionic fiber projects out and synapses onto a ganglionic neuron at the same level which then carries the information out through the spinal nerve. (b) represents the postganglionic sympathetic nerve pathway where a preganglionic branch projects and synapses onto a more superior or inferior ganglion in the chain which then carries the information out through a symapthetic nerve. (c) represents the splanchnic nerve pathway where a preganglionic branch extends through the white ramus communicans, but does not terminate on a ganglionic neuron in the chain. Instead, it projects through one of the splanchnic nerves to a prevertebral (collateral) ganglion. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 4.1.2.

Parasympathetic Division

The parasympathetic division of the autonomic nervous system is named because its central neurons are located away from (para- = “apart from”) the thoracolumbar region of the spinal cord which is dedicated to the sympathetic division. Indeed, the parasympathetic system can also be referred to as the **craniosacral system** because the preganglionic neurons are located in nuclei of the brainstem and the lateral horn of the sacral spinal cord (S2 to S4).

Parasympathetic Neurons, Ganglia and Nerves

The connections, or “circuits,” of the parasympathetic division are similar to the general layout of the sympathetic division with a few specific differences. The parasympathetic preganglionic fibers from the cranial region travel in cranial nerves, whereas parasympathetic preganglionic fibers from the sacral region travel in spinal nerves. The targets of these fibers are **terminal ganglia**, which are located near the target effector, and **intramural ganglia**, which are found within the walls of the target organ. Terminal ganglia receive input from cranial nerves or sacral spinal nerves. The terminal ganglia that receive input from cranial

nerves are found in the head and neck, as well as the thoracic and upper abdominal cavities, whereas the terminal ganglia that receive sacral input are in the lower abdominal and pelvic cavities. The postganglionic fiber projects from the terminal ganglia a short distance to the target effector, or to the specific target tissue within the organ.

The cranial nerves associated with the parasympathetic system are the oculomotor nerve (CN III), facial nerve (VII), glossopharyngeal nerve (CN IX) and vagus nerve (CN X). These nerves generate from particular nuclei of the brainstem. Nuclei in the midbrain are part of the oculomotor complex, and parasympathetic axons from those neurons travel in the **oculomotor nerve (CN III)** with the somatic motor fibers that innervate the extraocular muscles. The parasympathetic preganglionic fibers within the oculomotor nerve terminate in the **ciliary ganglion**, which is located in the posterior orbit. The postganglionic parasympathetic fibers then project to the sphincter pupillae and ciliary muscle of the iris to control the size of the pupil and the shape of the lens.

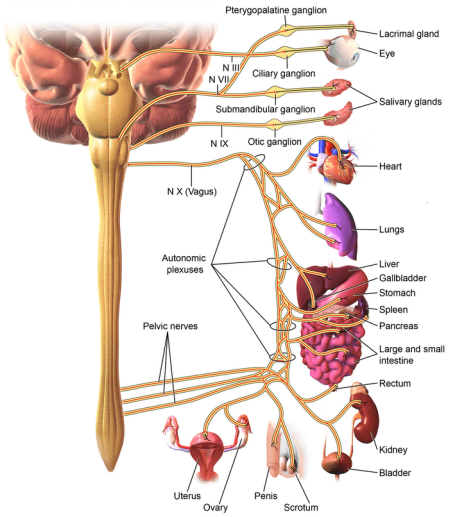
Parasympathetic preganglionic fibers exits the pons and travel through the **facial nerve (CN VII)** to control the secretions of the lacrimal apparatus, nasal epithelium and salivary glands. Two branches exit the facial nerve. The first branch terminates at the **pterygopalatine ganglion**. Postganglionic fibers from this ganglion extend to the lacrimal gland and glands of the nasal cavity, oral cavity, and palate. The second branch terminates at the **submandibular ganglion**. Postganglionic fibers from this ganglion project to submandibular and sublingual salivary glands.

Parasympathetic preganglionic fibers exits the medulla oblongata and travel through the **glossopharyngeal nerve (CN IX)** to the **otic ganglion**. Postganglionic fibers from this ganglion terminate to the parotid salivary glands.

The majority of parasympathetic preganglionic axons travel through the **vagus nerve (CN X)** that innervates thoracic and abdominal organs as well as the gonads (ovaries and testes). Autonomic parasympathetic neurons in the medulla oblongata project through the vagus nerve to the terminal and intramural ganglia of target effectors such as heart, airways, esophagus, stomach, liver, gallbladder, pancreas, small and large intestine, kidneys, ureters, and gonads.

The remaining parasympathetic preganglionic axons originate from neurons of the lateral horns of the S2-S4 segments of the spinal cord. These axons form the **pelvic splanchnic nerves** that project to terminal or intramural ganglia of abdominal and pelvic organs. The main target effectors are the distal portion of the large intestine, rectum, urinary bladder, and most of reproductive organs.

Comparing the relative lengths of axons in the parasympathetic system, the preganglionic fibers are long and the postganglionic fibers are short because the ganglia are close to—and sometimes within—the target effectors. Parasympathetic preganglionic axons tend to have fewer than 4 branches. The lack of divergent branches in parasympathetic preganglionic axons prevents a systemic response and facilitates discrete and localized effects on one group of organs at a time.



Parasympathetic Innervation

Figure 4.1.3: Neurons from brain-stem nuclei, or from the lateral horn of the sacral spinal cord, extend their preganglionic nerve fibers (shown in red) to terminal ganglia near the effector organs or to intramural ganglia within the walls of the effector organs. Postganglionic axons (shown in black) from these ganglionic neurons then project the short distance to target effectors. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 4.1.3.

Everyday Connections

Fight or Flight? What About Fright and Freeze?

The original usage of the epithet “fight or flight” comes from a scientist named Walter Cannon who worked at Harvard in 1915. The concept of homeostasis and the functioning of the sympathetic system had been introduced in France in the previous century. Cannon expanded the idea, and introduced the idea that an animal responds to a threat by preparing to stand and fight or run away. The nature of this response was thoroughly explained in a book on the physiology of pain, hunger, fear, and rage.

When students learn about the sympathetic system and the fight-or-flight response, they often stop and wonder about other responses. If you were faced with a lioness running toward you as pictured at the beginning of this chapter, would you run or would you stand your ground? Some people would say that they would freeze and not know what to do. So isn’t there really more to what the autonomic system does than fight, flight, rest, or digest. What about fear and paralysis in the face of a threat?

The common epithet of “fight or flight” is being enlarged to be “fight, flight, or fright” or even “fight, flight, fright, or freeze.” Cannon’s original contribution was a catchy phrase to express some of what the nervous system does in response to a threat, but it is incomplete. The sympathetic system is responsible for the physiological responses to emotional states. The name “sympathetic” can be said to mean that (sym- = “together”; -pathos = “pain,” “suffering,” or “emotion”).

Concept Review

The nervous system can be divided into two functional parts: the somatic nervous system (SNS) and the autonomic nervous system (ANS). The differences between these two systems lie on multiple features: the conscious level of their processes, their targets, the number of lower motor neurons involved and the neurotransmitters used. The autonomic nervous system reflexively responds to visceral sensory stimuli, such as levels of carbon dioxide concentration in the blood or stretch caused by blood pressure, that you are not consciously aware of and involuntarily controls cardiac and smooth muscle, as well as glandular tissue. The autonomic nervous system has a chain of two lower autonomic motor neurons. The first neuron is called a preganglionic neuron and resides in the brainstem or lateral horns of the spinal cord. This neuron releases ACh to a second neuron called a ganglionic neuron that is located in ganglia. The axons of ganglionic neurons are called postganglionic fibers. These nerves extend to target effectors and release either ACh or norepinephrine (NE).

The primary responsibilities of the autonomic nervous system are to regulate homeostatic mechanisms in the body. The way we respond to the world around us, to manage the internal environment on the basis of the external environment, is divided between two parts of the autonomic nervous system. The sympathetic division responds to threats and produces a readiness to confront the threat or to run away: the fight-or-flight response. The parasympathetic division plays the opposite role. When the external environment does not present any immediate danger, a restful mode descends on the body, and the digestive system is more active.

The sympathetic output of the nervous system originates out of the lateral horn of the thoracolumbar spinal cord. An axon from one of these central neurons projects by way of the ventral spinal nerve root, spinal nerve and white rami communicantes to a sympathetic chain (paravertebral) ganglion. The preganglionic fibers can synapse on ganglionic neurons here or extend to one of the prevertebral (collateral) ganglia via the splanchnic nerves (splanchnic nerve pathway). Postganglionic fibers of sympathetic chain ganglia can either return to the spinal nerve through the gray rami communicantes (spinal nerve pathway) or extend away from the ganglion through a sympathetic nerve (postganglionic sympathetic pathway). The sympathetic system also has a specialized preganglionic connection to the adrenal medulla that causes epinephrine and norepinephrine to be released into the bloodstream rather than exciting a neuron that contacts an organ directly (adrenal medulla pathway).

The parasympathetic output is based in the brainstem and sacral spinal cord. Neurons from particular nuclei in the brainstem carry parasympathetic information through four cranial nerves: oculomotor nerve (CN III), facial nerve (VII), glossopharyngeal nerve (CN IX) and vagus nerve (CN X). In the sacral spinal cord, preganglionic neurons of the lateral horn project out through pelvic splanchnic nerves. Cranial and sacral preganglionic fibers extend to terminal and intramural ganglia located close to or within the wall of target effectors. The postganglionic fibers of the ganglionic neurons then contact the target tissues within the organ to induce rest-and-digest responses. Due to the fact that parasympathetic ganglia are either close to or within the target organ, parasympathetic preganglionic axons are longer and postganglionic axons are shorter, compared to the sympathetic division.

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4.2: Autonomic Synapses, Effects and Reflexes

Autonomic Synapses

Where an autonomic neuron connects with a target, there is a synapse. What are referred to here as synapses may not fit the strictest definition of synapse. The structure of postganglionic connections are not the typical synaptic end bulb that is found at the neuromuscular junction, but rather are chains of swellings along the length of a postganglionic fiber. This place of connection is called a **varicosity** and is typical of the autonomic nervous system (ANS) (Figure 4.2.1). Each postganglionic axon has a high number of these endings resembling a string of beads. At each varicosity, synaptic vesicles containing **neurotransmitters** are present and ready to merge with the plasma membrane of the axon to release the neurotransmitters. On the target organ (smooth muscle, cardiac muscle or glands) **neurotransmitter receptors** are present.

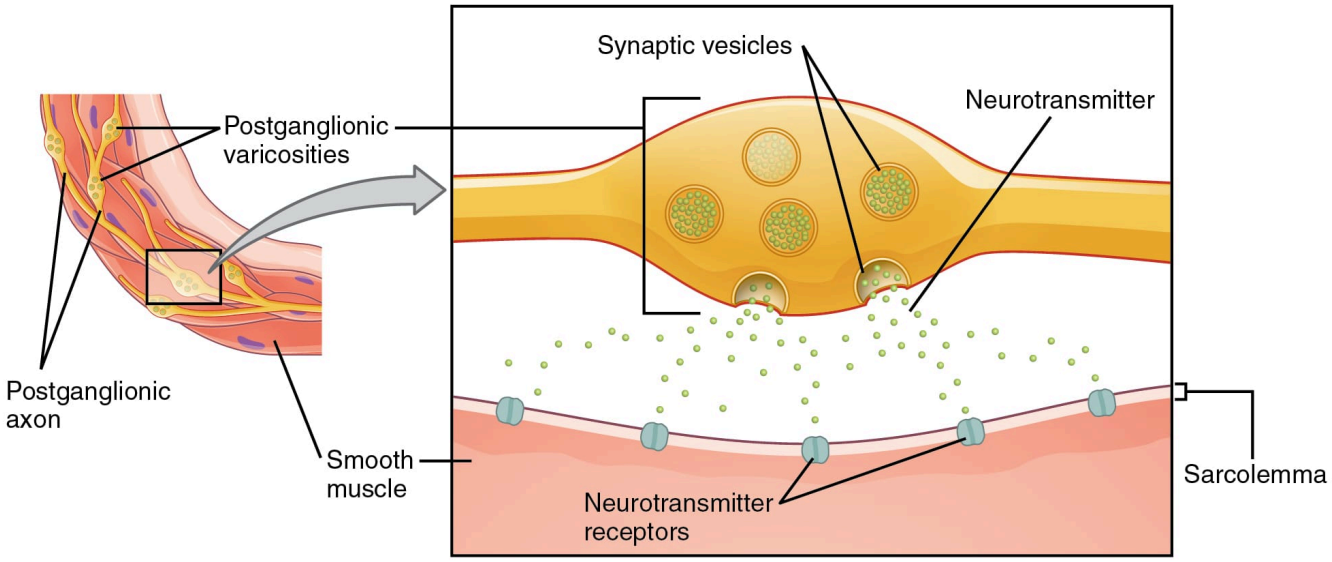


Figure 4.2.1: The connection between autonomic postganglionic fibers and target effectors is called a varicosity. Multiple varicosities are present along the length of postganglionic axons forming an extended network of connections on the target effector. Inside the varicosity, synaptic vesicles containing the neurotransmitters are ready to merge with the plasma membrane and release their content. In this picture, the target effector is smooth muscle and receptors for the neurotransmitters are present on its sarcolemma. (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 4.2.1.

Autonomic Neurotransmitters

Signaling molecules can belong to two broad groups. Neurotransmitters are released at synapses, whereas hormones are released into the bloodstream. However, the same molecule (for example epinephrine) can be released by neurons within the brain as a neurotransmitter or by the adrenal medulla into the bloodstream as a hormone, blurring the lines between these definitions. The main signaling molecules of the autonomic nervous system are **acetylcholine** (ACh) and **norepinephrine** (NE, also called noradrenaline). The adrenal medulla which is a modified sympathetic ganglion releases **epinephrine** (or adrenaline). These molecules will bind to specific receptors on the target cells. Cells that release acetylcholine are called **cholinergic**, while cells that release norepinephrine or epinephrine are called **adrenergic**. Acetylcholine can bind to both **nicotinic receptors** and **muscarinic receptors**. Norepinephrine and epinephrine can bind to **adrenergic receptors**. For each of these classes of receptors, there are multiple subtypes (which we will not discuss in this book) that make the cells respond differently to the binding of the same molecule. Depending on the receptor type and subtype, the molecules released will cause either stimulation or inhibition. Thus, the effects of the autonomic divisions depend on the type of signaling molecule and receptor involved.

All preganglionic fibers, both sympathetic and parasympathetic, are cholinergic and release ACh (Table 4.2.1). All ganglionic neurons (the targets of these preganglionic fibers) have nicotinic receptors in their cell membranes, which leads to the stimulation and firing of an action potential from the ganglionic fibers in both sympathetic and parasympathetic divisions. Most sympathetic

postganglionic fibers are adrenergic and release norepinephrine. The adrenal medulla which is part of the sympathetic division is also adrenergic and releases epinephrine. However, parasympathetic postganglionic fibers are cholinergic and release ACh. This pattern assures that effector organs under the control of both sympathetic and parasympathetic systems can respond differently. Target cells can contain various types and subtypes of receptors and their response will vary depending on the type of receptor and on the neurotransmitter released on them.

There is one exception to this pattern in the sympathetic system. The sympathetic postganglionic fibers that contact the blood vessels within skeletal muscle and sweat glands in the integument release ACh instead of norepinephrine. This does not create any problem because there is no parasympathetic input to these organs. Blood vessels of skeletal muscles and sweat glands have muscarinic receptors and vasodilate (increase diameter size) and produce and secrete sweat, respectively, in response to ACh.

At most of the other autonomic targets that are innervate by both divisions of the autonomic system, the effector response is based on which neurotransmitter is released and what receptor is present. For example, regions of the heart that establish heart rate are contacted by postganglionic fibers from both systems. If norepinephrine is released, it binds to an adrenergic receptor which causes the heart rate to increase. If ACh is released, it binds to a muscarinic receptor that causes the heart rate to slow. Without this parasympathetic input, the heart would work at a rate of approximately 100 beats per minute (bpm). The sympathetic system speeds that up, as it would during exercise, to 120–140 bpm, for example. The parasympathetic system slows it down to the resting heart rate of 60–80 bpm.

Table 4.2.1: Comparison of Sympathetic and Parasympathetic Signaling Molecules

Site of Action	Sympathetic Division	Parasympathetic Division
Preganglionic axons	release ACh (cholinergic)	release ACh (cholinergic)
Ganglionic neurons	contain nicotinic (ACh) receptors	contain nicotinic (ACh) receptors
Postganglionic axons	release NE (adrenergic); release ACh (cholinergic) only in blood vessels of skeletal muscle and sweat glands	release ACh (cholinergic)
Adrenal medulla	contain muscarinic (ACh) receptors; releases epinephrine (adrenergic)	Not Present

Autonomic Effects

Organ systems are balanced between the input from the sympathetic and parasympathetic divisions. When something upsets that balance, the homeostatic mechanisms strive to return it to its regular state. For each organ system, there may be more of a sympathetic or parasympathetic tendency to the resting state, which is known as the **autonomic tone** of the system. For example, the resting heart rate is the result of the parasympathetic system slowing the heart down from its intrinsic rate of 100 bpm, consequently the heart can be said to be in parasympathetic tone.

Many effector organs of the autonomic nervous system have **dual innervation**, meaning that they receive competing inputs from the sympathetic and parasympathetic divisions. These divisions each play a role in effecting change, usually in competing directions. At the level of the target effector, the signal of which system is sending the message is strictly chemical and depending on the division involved and neurotransmitter released, the effects would be diverse. For example, the heart and the eye are examples of organs with dual innervation. The sympathetic system increases heart rate, whereas the parasympathetic system decreases heart rate. The sympathetic system dilates the pupil of the eye, whereas the parasympathetic system constricts the pupil.

In some organs, opposing effects are achieved without dual innervation. For example, the arrector pili muscles, sweat glands, and blood vessels to skeletal muscles and skin are primarily under sympathetic control. Blood pressure is partially determined by the contraction of smooth muscle in the walls of blood vessels. The parasympathetic system has no significant input to the systemic blood vessels, so the sympathetic system determines their tone. The sympathetic system causes vasoconstriction of blood vessels. However, the increasing metabolic activity of muscles causes (paracrine) vasodilation. This allows for blood flow to increase for those skeletal muscles that will be active in the fight-or-flight response.

Not always the sympathetic and parasympathetic divisions have opposite effects and, in a few cases, the two systems cooperate. The best example of cooperative effects occurs in the male sexual function. In reproductive organs, blood vessels of erectile tissues are innervate with parasympathetic projection, making them an exception. Acetylcholine released by these postganglionic parasympathetic fibers cause the vessels to dilate, leading to the engorgement of the erectile tissue and penile erection. At the same time, the sympathetic nervous system stimulates ejaculation by causing the contractions of the seminal vesicle and prostate gland. This synergistic effect facilitates reproduction. Table 4.2.2 summarizes the effects on different organs of the sympathetic and parasympathetic divisions.

Table 4.2.2: Effects of Sympathetic and Parasympathetic Divisions

Target Effector	Sympathetic Effect	Parasympathetic Effect
Arrector pili muscles	Contraction to cause hair erection	None
Sweat glands	Secretion	None
Salivary glands	Inhibits	Stimulates
Pupils	Dilation	Constriction
Ciliary muscle	None	Contraction for near vision
Heart	Increases heart rate	Decreases heart rate
Blood vessels of the heart (coronary)	Vasoconstriction or vasodilation	Vasodilation
Blood vessels to skeletal muscles	Vasodilation	None
Blood vessels to skin and other organs	Vasoconstriction to increase blood pressure	None
Blood vessels to gastrointestinal (GI) tract	Vasoconstriction	Vasodilation
Bronchi of lungs	Dilation	Constriction
Gastrointestinal (GI) tract gland secretion	Inhibits	Stimulates
Gallbladder	Inhibits	Stimulates
Peristalsis (motility)	Inhibits	Stimulates
Sphincters	Contraction (close)	Relaxation (open)
Urinary bladder	Relaxation	Contraction
Internal urethral sphincter	Contraction (close)	Relaxation (open)
Penis	Stimulates ejaculation	Stimulates erection
Clitoris	None	Stimulates erection

Autonomic Reflexes

The autonomic nervous system regulates many organ systems using neural circuits that are conceptually similar to the reflexes of the somatic nervous system.

A somatic reflex, such as the withdrawal reflex, involves skeletal muscle: a painful stimulus activates receptors, the signal travels along a sensory (afferent) neuron to the spinal cord, is processed in an integration center, and then a motor (efferent) neuron triggers a rapid, involuntary contraction of skeletal muscles.

An autonomic (visceral) reflex also uses a reflex arc, but its effectors are smooth muscle, cardiac muscle, or glands. In response to internal or external stimuli, autonomic reflexes adjust organ function without conscious control. Examples include the initiation of urination (micturition reflex), changes in heart rate or blood pressure, regulation of digestive activity, and changes in pupil diameter.

Somatic and visceral reflexes share common features — such as receptors, afferent pathways, integration centers, efferent pathways, and effectors — but they differ in the type of effector tissues involved, the pathways used (single vs. two-neuron efferent chains in autonomic reflexes), and the types of stimuli they typically respond to.

One similarity between a somatic reflex and a visceral reflex is the **afferent branch**, that brings information towards the CNS. Sensory neurons receiving input from the periphery—with cell bodies in the sensory ganglia, either of a cranial nerve or a dorsal root ganglion adjacent to the spinal cord—project into the CNS to initiate the reflex.

Many of the inputs to visceral reflexes are from special or somatic senses, but particular senses are associated with the viscera that are not part of the conscious perception of the environment through the somatic nervous system. For example, there is a specific type of mechanoreceptor, called a **baroreceptor**, in the walls of the aorta and carotid sinuses that senses the stretch of those organs when blood volume or pressure increases. You do not have a conscious perception of having high blood pressure, but that is an important afferent branch of the cardiovascular and, particularly, vasomotor reflexes. The sensory neuron is essentially the same as any other general sensory neuron, a unipolar neuron that has a cell body in a sensory ganglion. The baroreceptors from the carotid arteries have axons in the glossopharyngeal nerve, and those from the aorta have axons in the vagus nerve.

Though visceral senses are not primarily a part of conscious perception, those sensations sometimes make it to conscious awareness. If a visceral sense is strong enough, it will be perceived. The sensory homunculus—the representation of the body in the primary somatosensory cortex—only has a small region allotted for the perception of internal stimuli. If you swallow a large bolus of food, for instance, you will probably feel the lump of that food as it pushes through your esophagus, or even if your stomach is distended after a large meal. If you inhale especially cold air, you can feel it as it enters your larynx and trachea. These sensations are not the same as feeling high blood pressure or blood sugar levels.

When particularly strong visceral sensations rise to the level of conscious perception, the sensations are often felt in unexpected places. For example, strong visceral sensations of the heart will be felt as pain in the left shoulder and left arm. This irregular pattern of projection of conscious perception of visceral sensations is called **referred pain**. Depending on the organ system affected, the referred pain will project to different areas of the body (Figure 4.2.2). The location of referred pain is not random, but a definitive explanation of the mechanism has not been established. The most broadly accepted theory for this phenomenon is that the visceral sensory fibers enter into the same level of the spinal cord as the somatosensory fibers of the referred pain location. By this explanation, the visceral sensory fibers from the mediastinal region, where the heart is located, would enter the spinal cord at the same level as the spinal nerves from the shoulder and arm, so the brain misinterprets the sensations from the mediastinal region as being from the axillary and brachial regions.

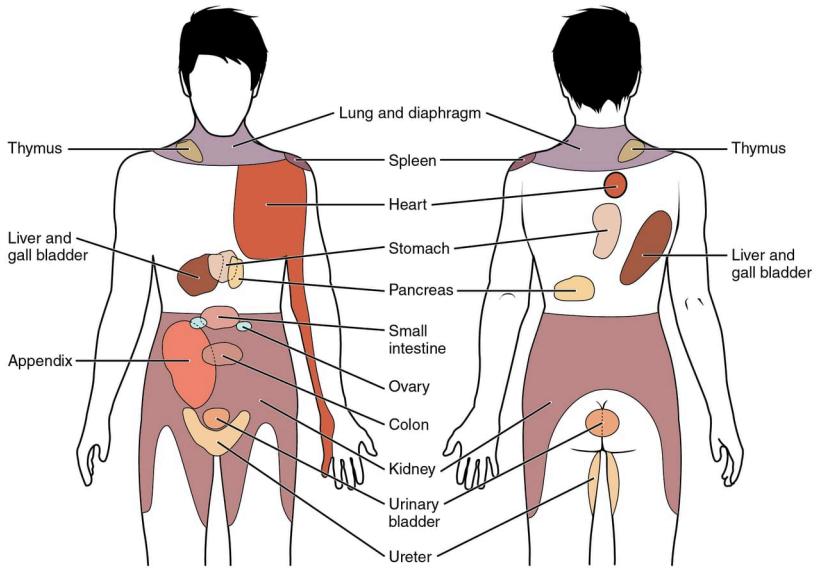


Figure 4.2.2: Referred pain is the conscious perception of visceral sensations. Represented here is a map that associates the pain perceived in certain regions with the visceral organs. Some sensations are felt locally, whereas others are perceived as affecting areas that are quite distant from the involved organ. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 4.2.2.

One difference between a somatic and visceral reflex is in the **efferent branch**. The output of a somatic reflex is the lower motor neuron in the ventral horn of the spinal cord that projects directly to a skeletal muscle to cause its contraction. The output of a visceral reflex is a two-step pathway starting with the preganglionic fiber emerging from a lateral horn neuron in the spinal cord, or a cranial nucleus neuron in the brainstem, to a ganglion—followed by the postganglionic fiber projecting to a target effector (Figure 4.2.3).

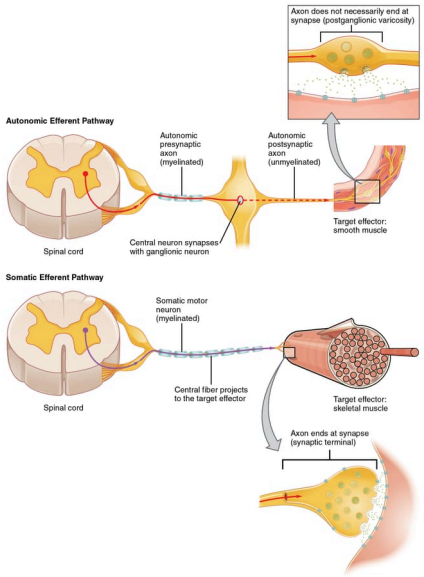


Figure 4.2.3: The efferent branches of somatic and visceral reflexes are different. (a) Visceral reflexes involve a projection from the central autonomic neuron to a ganglion through autonomic preganglionic (presynaptic) axons which are myelinated. This is followed by a second projection from the ganglion to the target effector (in this case smooth muscle) through autonomic postganglionic (postsynaptic) axons which are unmyelinated. These axons end in postganglionic varicosities. (b) Somatic reflexes involve a direct connection from the ventral horn of the spinal cord to the skeletal muscle through a myelinated central fiber. This axon ends in a synaptic terminal. (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 4.2.3.

Short and Long Reflexes

Somatic reflexes involve sensory neurons that connect sensory receptors to the central nervous system (CNS) and motor neurons that project back out to the skeletal muscles. Visceral reflexes that involve the sympathetic or parasympathetic systems share similar connections. However, there are reflexes that do not need to involve any CNS components. A **long reflex** has afferent branches that enter the spinal cord or brain and involve the efferent branches. A **short reflex** is completely peripheral with sensory neurons synapsing with motor neurons in autonomic ganglia; the CNS is not involved (Figure 4.2.4).

A division of the nervous system that is related to the autonomic nervous system is the enteric nervous system. The word enteric refers to the digestive organs, so this represents the nervous tissue that is part of the digestive system. There are a few myenteric plexuses in which the nervous tissue in the wall of the digestive tract organs can directly influence digestive function. If stretch receptors in the stomach are activated by the filling and distension of the stomach, a short reflex will directly activate the smooth muscle fibers of the stomach wall to increase motility to digest the excessive food in the stomach. No CNS involvement is needed because the stretch receptor is directly activating a neuron in the wall of the stomach that causes the smooth muscle to contract. That neuron, connected to the smooth muscle, is a ganglionic parasympathetic neuron that can be controlled by a fiber found in the vagus nerve.

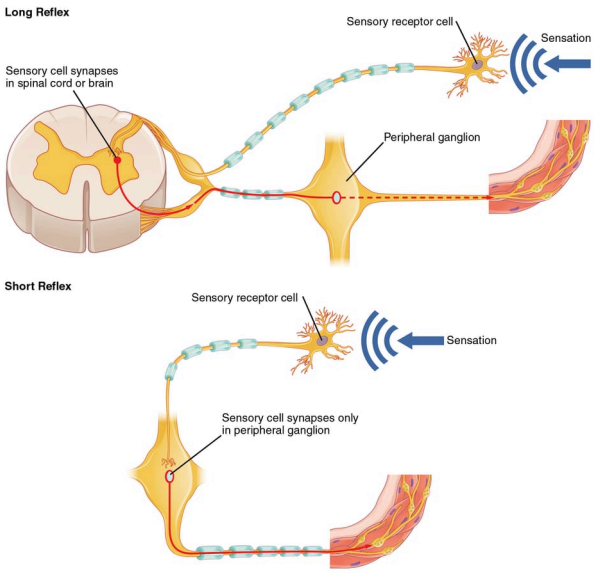


Figure 4.2.4: Sensory input can stimulate either a short or a long reflex. A sensory neuron can project to the brain or spinal cord or to an autonomic ganglion. The long reflex involves integration in the spinal cord or brain. In this reflex, a sensory signal coming from a sensory receptor cell reaches a central motor neuron, which then extends a preganglionic axon to a peripheral ganglion. The postganglionic axons extend to the organ effector. A short reflex involves the direct stimulation of a ganglionic neuron by the sensory neuron. In this reflex, a sensory receptor cell synapses onto a ganglionic neuron, whose axons extend to the organ effector. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 4.2.4.

DISORDERS OF THE...Nervous System: Kehr's Sign

Kehr's sign is the presentation of pain in the left shoulder, chest, and neck regions following rupture of the spleen. The spleen is in the upper-left abdominopelvic quadrant, but the pain is more in the shoulder and neck. How can this be? The sympathetic fibers connected to the spleen are from the celiac ganglion, which would be from the mid-thoracic to lower thoracic region whereas parasympathetic fibers are found in the vagus nerve, which connects in the medulla of the brainstem. However, the neck and shoulder would connect to the spinal cord at the mid-cervical level of the spinal cord. These connections do not fit with the expected correspondence of visceral and somatosensory fibers entering at the same level of the spinal cord.

The incorrect assumption would be that the visceral sensations are coming from the spleen directly. In fact, the visceral fibers are coming from the diaphragm. The nerve connecting to the diaphragm takes a special route. The phrenic nerve is connected to the spinal cord at cervical levels 3 to 5. The motor fibers that make up this nerve are responsible for the muscle contractions that drive ventilation. These fibers have left the spinal cord to enter the phrenic nerve, meaning that spinal cord damage below the mid-cervical level is not fatal by making ventilation impossible. Therefore, the visceral fibers from the diaphragm enter the spinal cord at the same level as the somatosensory fibers from the neck and shoulder.

The diaphragm plays a role in Kehr's sign because the spleen is just inferior to the diaphragm in the upper-left quadrant of the abdominopelvic cavity. When the spleen ruptures, blood spills into this region. The accumulating hemorrhage then puts pressure on the diaphragm. The visceral sensation is actually in the diaphragm, so the referred pain is in a region of the body that corresponds to the diaphragm, not the spleen

Concept Review

The synapses in the autonomic system are not always the typical type of connection first described in the neuromuscular junction. Instead of having synaptic end bulbs at the very end of an axonal fiber, they may have swellings called varicosities along the length of a fiber so that it makes a network of connections within the target tissue. All preganglionic fibers are cholinergic and release acetylcholine (ACh). All ganglionic neurons (the targets of these preganglionic fibers) have nicotinic receptors in their cell membranes. Most sympathetic postganglionic fibers are adrenergic and release norepinephrine. The adrenal medulla which is part of the sympathetic division is also adrenergic and releases epinephrine. Parasympathetic postganglionic fibers are cholinergic and release ACh. Target cells can contain various types and subtypes of receptors and their response will vary

depending on the type of receptor and on the neurotransmitter released on them. The sympathetic postganglionic fibers that contact the blood vessels within skeletal muscle and sweat glands in the integument release ACh instead of norepinephrine. This does not create any problem because there is no parasympathetic input to these organs.

For each organ system, there may be more of a sympathetic or parasympathetic tendency to the resting state, which is known as the autonomic tone of the system. Many effector organs of the autonomic nervous system have dual innervation, meaning that they receive competing inputs from the sympathetic and parasympathetic divisions. The sympathetic system increases heart rate, whereas the parasympathetic system decreases heart rate. The sympathetic system dilates the pupil of the eye, whereas the parasympathetic system constricts the pupil. The competing inputs can contribute to the resting tone of the organ system. Heart rate is normally under parasympathetic tone, whereas blood pressure is normally under sympathetic tone. The heart rate is slowed by the autonomic system at rest, whereas blood vessels retain a slight constriction at rest. In a few systems of the body, the competing input from the two divisions is not the norm. The sympathetic tone of blood vessels is caused by the lack of parasympathetic input to the systemic circulatory system. Only certain regions receive parasympathetic input that relaxes the smooth muscle wall of the blood vessels. Sweat glands are another example of organs that only receive input from the sympathetic system. Not always the sympathetic and parasympathetic divisions have opposite effects and, in a few cases, the two systems cooperate.

Autonomic nervous system function is based on the visceral reflex, which is similar to the somatic reflex. Like the somatic reflex, the afferent branch is composed of sensory neurons receiving input from the periphery project into the CNS to initiate the reflex. The cell bodies of these sensory neurons are located in the sensory ganglia, either of a cranial nerve or a dorsal root ganglion adjacent to the spinal cord. However, the efferent branch of a visceral reflex is composed of two neurons. The central neuron projects from the spinal cord or brainstem to synapse on the ganglionic neuron that projects to the effector. The afferent branch of the somatic and visceral reflexes is very similar, as many somatic and special senses activate autonomic responses. However, there are visceral senses that do not form part of conscious perception. If a visceral sensation, such as cardiac pain, is strong enough, it will rise to the level of consciousness. However, the sensory homunculus does not provide a representation of the internal structures to the same degree as the surface of the body, so visceral sensations are often experienced as referred pain, such as feelings of pain in the left shoulder and arm in connection with a heart attack.

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4.3: Central Control Autonomic Function

Central Control of Autonomic Reflexes

Autonomic control is based on the visceral reflexes, composed of the afferent and efferent branches. These homeostatic mechanisms are based on the balance between the two divisions of the autonomic system, which results in tone for various organs that is based on the predominant input from the sympathetic or parasympathetic systems. Coordinating that balance requires integration that begins with forebrain structures like the hypothalamus and continues into the brainstem and spinal cord.

The autonomic nervous system is controlled and influenced by the cerebral cortex, hypothalamus, limbic system, medulla oblongata and spinal cord. In the next sections we will focus on the inputs from the hypothalamus, amygdala and medulla oblongata.

Hypothalamus

The **hypothalamus** is the control center for many homeostatic mechanisms. It regulates both autonomic function and endocrine function. The optic nerve projects primarily to the thalamus, which is the necessary relay to the occipital cortex for conscious visual perception. Another projection of the optic nerve, however, goes to the hypothalamus. The hypothalamus then uses this visual system input to drive the **pupillary reflexes**. If the retina is activated by high levels of light, the hypothalamus stimulates the parasympathetic response which stimulates the circular fibers of the iris to contract and constrict the pupil. If the optic nerve message shows that low levels of light are falling on the retina, the hypothalamus activates the sympathetic response which dilates the pupil. When light hits the retina in one eye, both pupils contract. When that light is removed, both pupils dilate again back to the resting position. When the stimulus is unilateral (presented to only one eye), the response is bilateral (both eyes). The same is not true for somatic reflexes. If you touch a hot radiator, you only pull that arm back, not both. Central control of autonomic reflexes is different than for somatic reflexes.

The hypothalamus also receives input from other areas of the forebrain through the **medial forebrain bundle** (Figure 4.3.1). The olfactory cortex, the basal nuclei, and the amygdala project into the hypothalamus through the medial forebrain bundle. These forebrain structures inform the hypothalamus about the state of the nervous system and can influence the regulatory processes of homeostasis. A good example of this is found in the amygdala, which is found beneath the cerebral cortex of the temporal lobe and plays a role in our ability to remember and feel emotions.

Output from the hypothalamus follows two main tracts, the **dorsal longitudinal fasciculus** and the medial forebrain bundle (Figure 4.3.1). Along these two tracts, the hypothalamus can influence major parasympathetic nuclei in the brainstem such as the **oculomotor complex**, the **dorsal motor nucleus** and the **nucleus ambiguus**, and preganglionic (central) neurons of the thoracolumbar spinal cord. Neurons in the dorsal nucleus of the vagus nerve and the nucleus ambiguus project through the vagus nerve (cranial nerve X) to the terminal ganglia of the thoracic and abdominal cavities.

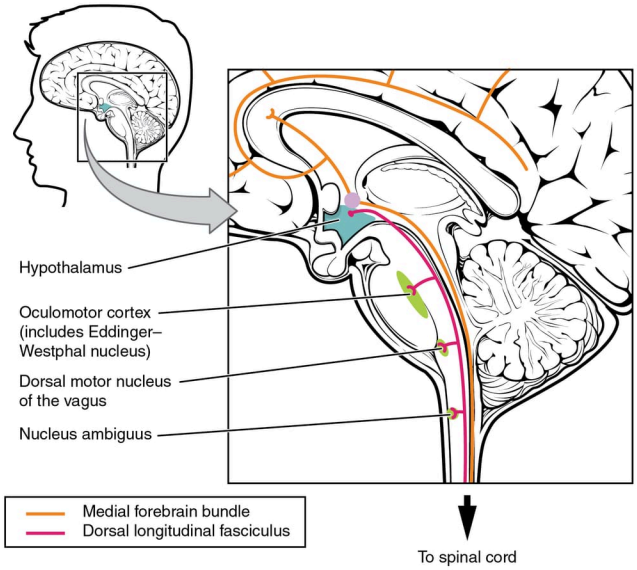


Figure 4.3.1: The hypothalamus is the source of most of the central control of autonomic function. It receives input from cerebral structures through the medial forebrain bundle. The hypothalamus then projects to brainstem and spinal cord structures through the medial forebrain bundle and the dorsal longitudinal fasciculus. to regulate the balance of sympathetic and parasympathetic input to the organ systems of the body. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 4.3.1.

Amygdala

The **amygdala** is a group of nuclei in the medial region of the temporal lobe that is part of the **limbic lobe**, a C-shaped region of the cortex on the medial side of each cerebral hemisphere, consisting of portions of the temporal, parietal, and frontal lobes (Figure 4.3.2). The limbic lobe includes structures that are involved in emotional responses, as well as structures that contribute to memory function. The limbic lobe has strong connections with the hypothalamus and influences the state of its activity on the basis of emotional state. For example, when you are anxious or scared, the amygdala will send signals to the hypothalamus along the medial forebrain bundle that will stimulate the sympathetic fight-or-flight response. The hypothalamus will also stimulate the release of stress hormones through its control of the endocrine system in response to amygdala input.

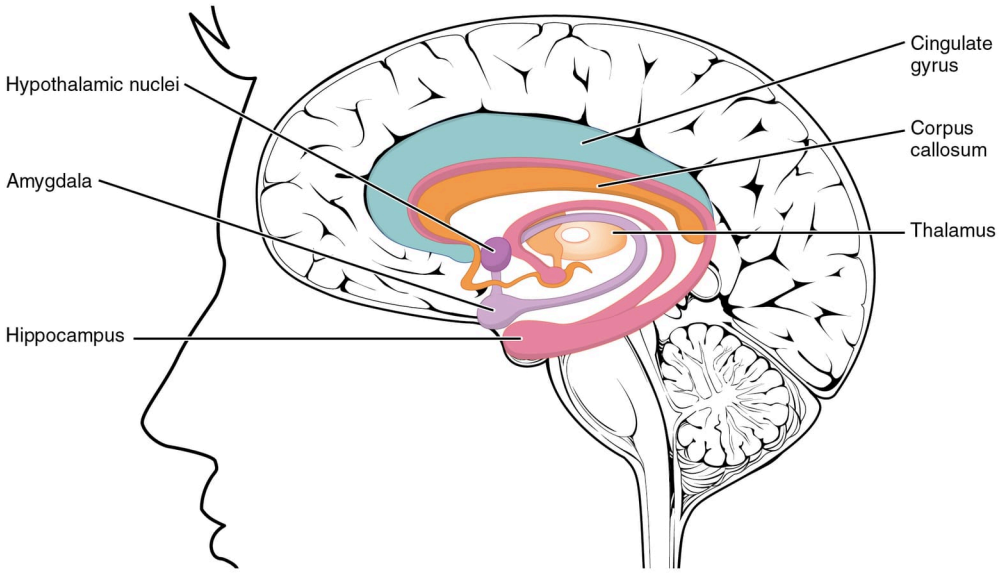


Figure 4.3.2: Structures arranged around the edge of the cerebrum constitute the limbic lobe, which includes the amygdala, hippocampus, and cingulate gyrus, and connects to the hypothalamus, which sits inferior and anterior to the thalamus. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 4.3.2.

Medulla oblongata

The **medulla oblongata** contains nuclei referred to as the **cardiovascular center**, which controls the smooth and cardiac muscle of the cardiovascular system through autonomic connections. When the homeostasis of the cardiovascular system shifts, such as when blood pressure changes, the coordination of the autonomic system can be accomplished within this region. Furthermore, when descending inputs from the hypothalamus stimulate this area, the sympathetic system can increase activity in the cardiovascular system, such as in response to anxiety or stress. The preganglionic sympathetic fibers that are responsible for increasing heart rate are referred to as the **cardiac accelerator nerves**, whereas the preganglionic sympathetic fibers responsible for constricting blood vessels compose the **vasomotor nerves**.

Several brainstem nuclei are important for the visceral control of major organ systems. One brainstem nucleus involved in cardiovascular function is the **solitary nucleus**. It receives sensory input about blood pressure and cardiac function from the glossopharyngeal and vagus nerves, and its output will activate sympathetic stimulation of the heart or blood vessels through the upper thoracic lateral horn. Another brainstem nucleus important for visceral control is the **dorsal motor nucleus of the vagus nerve**, which is the motor nucleus for the parasympathetic functions ascribed to the vagus nerve, including decreasing the heart rate, relaxing bronchial tubes in the lungs, and activating digestive function through the enteric nervous system. The **nucleus ambiguus**, which is named for its ambiguous histology, also contributes to the parasympathetic output of the vagus nerve and targets muscles in the pharynx and larynx for swallowing and speech, as well as contributing to the parasympathetic tone of the heart along with the dorsal motor nucleus of the vagus.

EVERYDAY CONNECTIONS

Exercise and the Autonomic System

In addition to its association with the fight-or-flight response and rest-and-digest functions, the autonomic system is responsible for certain everyday functions. For example, it comes into play when homeostatic mechanisms dynamically change, such as the physiological changes that accompany exercise. Getting on the treadmill and putting in a good workout will cause the heart rate to increase, breathing to be stronger and deeper, sweat glands to activate, and the digestive system to suspend activity. These are the same physiological changes associated with the fight-or-flight response, but there is nothing chasing you on that treadmill.

This is not a simple homeostatic mechanism at work because “maintaining the internal environment” would mean getting all those changes back to their set points. Instead, the sympathetic system has become active during exercise so that your body can cope with what is happening. A homeostatic mechanism is dealing with the conscious decision to push the body away from a resting state. The heart, actually, is moving away from its homeostatic set point. Without any input from the autonomic system, the heart would beat at approximately 100 bpm, and the parasympathetic system slows that down to the resting rate of approximately 70 bpm. But in the middle of a good workout, you should see your heart rate at 120–140 bpm. You could say that the body is stressed because of what you are doing to it. Homeostatic mechanisms are trying to keep blood pH in the normal range, or to keep body temperature under control, but those are in response to the choice to exercise.

Concept Review

The autonomic system integrates sensory information and higher cognitive processes to generate output, which balances homeostatic mechanisms. The central autonomic structure is the hypothalamus, which coordinates sympathetic and parasympathetic efferent pathways to regulate activities of the organ systems of the body. The majority of hypothalamic output travels through the medial forebrain bundle and the dorsal longitudinal fasciculus to influence brainstem and spinal components of the autonomic nervous system. The medial forebrain bundle also connects the hypothalamus with higher centers of the limbic system where emotion can influence visceral responses. The amygdala is a structure within the limbic system that influences the hypothalamus in the regulation of the autonomic system, as well as the endocrine system.

These higher centers have descending control of the autonomic system through brainstem centers, primarily in the medulla, such as the cardiovascular center. This collection of medullary nuclei regulates cardiac function, as well as blood pressure. Sensory input from the heart, aorta, and carotid sinuses project to these regions of the medulla. The solitary nucleus increases sympathetic tone of the cardiovascular system through the cardiac accelerator and vasomotor nerves. The nucleus ambiguus and the dorsal motor nucleus both contribute fibers to the vagus nerve, which exerts parasympathetic control of the heart by decreasing heart rate.

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4.4: Drugs that Affect the Autonomic System

Autonomic Pharmacology

An important way to understand the effects of native neurochemicals in the autonomic system is in considering the effects of pharmaceutical drugs. This can be considered in terms of how drugs change autonomic function. These effects will primarily be based on how drugs act at the receptors of the autonomic system neurochemistry. The signaling molecules of the nervous system interact with proteins in the cell membranes of various target cells. In fact, no effect can be attributed to just the signaling molecules themselves without considering the receptors. A chemical that the body produces to interact with those receptors is called an endogenous chemical, whereas a chemical introduced to the system from outside is an exogenous chemical. Exogenous chemicals may be of a natural origin, such as a plant extract, or they may be synthetically produced in a pharmaceutical laboratory.

Broad Autonomic Effects

One important drug that affects the autonomic system broadly is not a pharmaceutical therapeutic agent associated with the system. This drug is nicotine. The effects of nicotine on the autonomic nervous system are important in considering the role smoking can play in health.

All ganglionic neurons of the autonomic system, in both sympathetic and parasympathetic ganglia, are activated by ACh released from preganglionic fibers. The ACh receptors on these neurons are of the nicotinic type, meaning that they are ligand-gated ion channels. When the neurotransmitter released from the preganglionic fiber binds to the receptor protein, a channel opens to allow positive ions to cross the cell membrane. The result is depolarization of the ganglia. Nicotine acts as an ACh analog at these synapses, so when someone takes in the drug, it binds to these ACh receptors and activates the ganglionic neurons, causing them to depolarize.

Ganglia of both divisions are activated equally by the drug. For many target organs in the body, this results in no net change. The competing inputs to the system cancel each other out and nothing significant happens. For example, the sympathetic system will cause sphincters in the digestive tract to contract, limiting digestive propulsion, but the parasympathetic system will cause the contraction of other muscles in the digestive tract, which will try to push the contents of the digestive system along. The end result is that the food does not really move along and the digestive system has not appreciably changed.

The system in which this can be problematic is in the cardiovascular system, which is why smoking is a risk factor for cardiovascular disease. First, there is no significant parasympathetic regulation of blood pressure. Only a limited number of blood vessels are affected by parasympathetic input, so nicotine will preferentially cause the vascular tone to become more sympathetic, which means blood pressure will be increased. Second, the autonomic control of the heart is special. Unlike skeletal or smooth muscles, cardiac muscle is intrinsically active, meaning that it generates its own action potentials. The autonomic system does not cause the heart to beat, it just speeds it up (sympathetic) or slows it down (parasympathetic). The mechanisms for this are not mutually exclusive, so the heart receives conflicting signals, and the rhythm of the heart can be affected (Figure 4.4.1).

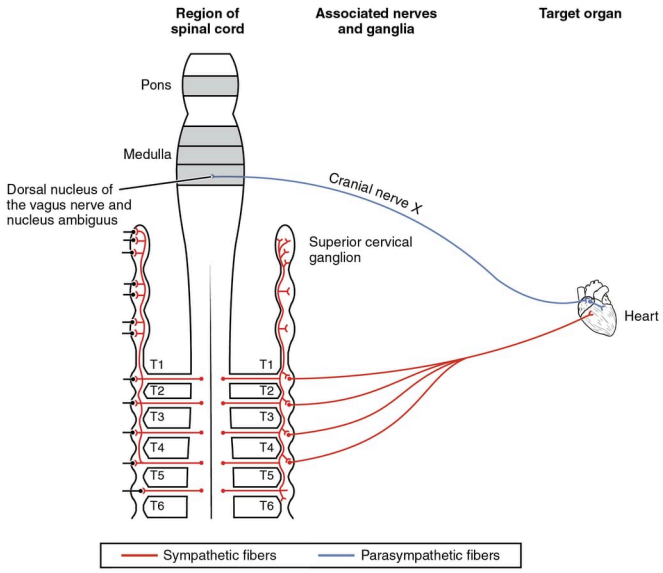


Figure 4.4.1: The nicotinic receptor is found on all autonomic ganglia, but the cardiovascular connections are particular, and do not conform to the usual competitive projections that would just cancel each other out when stimulated by nicotine. The opposing signals to the heart would both depolarize and hyperpolarize the heart cells that establish the rhythm of the heartbeat, likely causing arrhythmia. Only the sympathetic system governs systemic blood pressure so nicotine would cause an increase. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 4.4.1.

Sympathetic Effect

The neurochemistry of the sympathetic system is based on the adrenergic system. Norepinephrine and epinephrine influence target effectors by binding to the α-adrenergic or β-adrenergic receptors. Drugs that affect the sympathetic system affect these chemical systems. The drugs can be classified by whether they enhance the functions of the sympathetic system or interrupt those functions. A drug that enhances adrenergic function is known as a sympathomimetic drug, whereas a drug that interrupts adrenergic function is a sympatholytic drug.

Sympathomimetic Drugs

When the sympathetic system is not functioning correctly or the body is in a state of homeostatic imbalance, these drugs act at postganglionic terminals and synapses in the sympathetic efferent pathway. These drugs either bind to particular adrenergic receptors and mimic norepinephrine at the synapses between sympathetic postganglionic fibers and their targets, or they increase the production and release of norepinephrine from postganglionic fibers. Also, to increase the effectiveness of adrenergic chemicals released from the fibers, some of these drugs may block the removal or reuptake of the neurotransmitter from the synapse.

A common sympathomimetic drug is phenylephrine, which is a common component of decongestants. It can also be used to dilate the pupil and to raise blood pressure. Phenylephrine is known as an α_1-adrenergic agonist, meaning that it binds to a specific adrenergic receptor, stimulating a response. In this role, phenylephrine will bind to the adrenergic receptors in bronchioles of the lungs and cause them to dilate. By opening these structures, accumulated mucus can be cleared out of the lower respiratory tract. Phenylephrine is often paired with other pharmaceuticals, such as analgesics, as in the “sinus” version of many over-the-counter drugs, such as Tylenol Sinus® or Excedrin Sinus®, or in expectorants for chest congestion such as in Robitussin CF®.

A related molecule, called pseudoephedrine, was much more commonly used in these applications than was phenylephrine, until the molecule became useful in the illicit production of amphetamines. Phenylephrine is not as effective as a drug because it can be partially broken down in the digestive tract before it is ever absorbed. Like the adrenergic agents, phenylephrine is effective in dilating the pupil, known as mydriasis (Figure 4.4.2). Phenylephrine is used during an eye exam in an ophthalmologist’s or optometrist’s office for this purpose. It can also be used to increase blood pressure in situations in which cardiac function is compromised, such as under anesthesia or during septic shock.



Figure 4.4.2: The sympathetic system causes pupillary dilation when norepinephrine binds to an adrenergic receptor in the radial fibers of the iris smooth muscle. Phenylephrine mimics this action by binding to the same receptor when drops are applied onto the surface of the eye in a doctor's office. (CC BY 4.0; via LibreTexts).

Other drugs that enhance adrenergic function are not associated with therapeutic uses, but affect the functions of the sympathetic system in a similar fashion. Cocaine primarily interferes with the uptake of dopamine at the synapse and can also increase adrenergic function. Caffeine is an antagonist to a different neurotransmitter receptor, called the adenosine receptor. Adenosine will suppress adrenergic activity, specifically the release of norepinephrine at synapses, so caffeine indirectly increases adrenergic activity. There is some evidence that caffeine can aid in the therapeutic use of drugs, perhaps by potentiating (increasing) sympathetic function, as is suggested by the inclusion of caffeine in over-the-counter analgesics such as Excedrin®.

Sympatholytic Drugs

Drugs that interfere with sympathetic function are referred to as sympatholytic, or sympathoplegic, drugs. They primarily work as an antagonist to the adrenergic receptors. They block the ability of norepinephrine or epinephrine to bind to the receptors so that the effect is "cut" or "takes a blow," to refer to the endings "-lytic" and "-plegic," respectively. The various drugs of this class will be specific to α-adrenergic or β-adrenergic receptors, or to their receptor subtypes.

Possibly the most familiar type of sympatholytic drug are the β-blockers. These drugs are often used to treat cardiovascular disease because they block the β-receptors associated with vasoconstriction and cardioacceleration. By allowing blood vessels to dilate, or keeping heart rate from increasing, these drugs can improve cardiac function in a compromised system, such as for a person with congestive heart failure or who has previously suffered a heart attack. A couple of common versions of β-blockers are metoprolol, which specifically blocks the β_1-receptor, and propanolol, which nonspecifically blocks β-receptors. There are other drugs that are α-blockers and can affect the sympathetic system in a similar way.

Other uses for sympatholytic drugs are as antianxiety medications. A common example of this is clonidine, which is an α-agonist. The sympathetic system is tied to anxiety to the point that the sympathetic response can be referred to as "fight, flight, or fright." Clonidine is used for other treatments aside from hypertension and anxiety, including pain conditions and attention deficit hyperactivity disorder.

Parasympathetic Effects

Drugs affecting parasympathetic functions can be classified into those that increase or decrease activity at postganglionic terminals. Parasympathetic postganglionic fibers release ACh, and the receptors on the targets are muscarinic receptors. There are several types of muscarinic receptors, M1–M5, but the drugs are not usually specific to the specific types. Parasympathetic drugs can be either muscarinic agonists or antagonists, or have indirect effects on the cholinergic system. Drugs that enhance cholinergic effects are called parasympathomimetic drugs, whereas those that inhibit cholinergic effects are referred to as anticholinergic drugs.

Pilocarpine is a nonspecific muscarinic agonist commonly used to treat disorders of the eye. It reverses mydriasis, such as is caused by phenylephrine, and can be administered after an eye exam. Along with constricting the pupil through the smooth

muscle of the iris, pilocarpine will also cause the ciliary muscle to contract. This will open perforations at the base of the cornea, allowing for the drainage of aqueous humor from the anterior compartment of the eye and, therefore, reducing intraocular pressure related to glaucoma.

Atropine and scopolamine are part of a class of muscarinic antagonists that come from the *Atropa* genus of plants that include belladonna or deadly nightshade (Figure 4.4.3). The name of one of these plants, belladonna, refers to the fact that extracts from this plant were used cosmetically for dilating the pupil. The active chemicals from this plant block the muscarinic receptors in the iris and allow the pupil to dilate, which is considered attractive because it makes the eyes appear larger. Humans are instinctively attracted to anything with larger eyes, which comes from the fact that the ratio of eye-to-head size is different in infants (or baby animals) and can elicit an emotional response. The cosmetic use of belladonna extract was essentially acting on this response. Atropine is no longer used in this cosmetic capacity for reasons related to the other name for the plant, which is deadly nightshade. Suppression of parasympathetic function, especially when it becomes systemic, can be fatal. Autonomic regulation is disrupted and anticholinergic symptoms develop. The berries of this plant are highly toxic, but can be mistaken for other berries. The antidote for atropine or scopolamine poisoning is pilocarpine.



Figure 4.4.3: The plant from the genus *Atropa*, which is known as belladonna or deadly nightshade, was used cosmetically to dilate pupils, but can be fatal when ingested. The berries on the plant may seem attractive as a fruit, but they contain the same anticholinergic compounds as the rest of the plant. (CC BY 4.0; via LibreTexts).

Table 4.4.1: Sympathetic and Parasympathetic Effects of Different Drug Types

Drug type	Example(s)	Sympathetic effect	Parasympathetic effect	Overall result
Nicotinic agonists	Nicotine	Mimic ACh at preganglionic synapses, causing activation of postganglionic fibers and the release of norepinephrine onto the target organ	Mimic ACh at preganglionic synapses, causing activation of postganglionic fibers and the release of ACh onto the target organ	Most conflicting signals cancel each other out, but cardiovascular system is susceptible to hypertension and arrhythmias
Sympathomimetic drugs	Phenylephrine	Bind to adrenergic receptors or mimics sympathetic action in some other way	No effect	Increase sympathetic tone
Sympatholytic drugs	β-blockers such as propanolol or metoprolol; α-agonists such as clonidine	Block binding to adrenergic drug or decrease adrenergic signals	No effect	Increase parasympathetic tone

Drug type	Example(s)	Sympathetic effect	Parasympathetic effect	Overall result
Parasympatho-mimetics/ muscarinic agonists	Pilocarpine	No effect, except on sweat glands	Bind to muscarinic receptor, similar to ACh	Increase parasympathetic tone
Anticholinergics/ muscarinic antagonists	Atropine, scopolamine, dimenhydrinate	No effect	Block muscarinic receptors and parasympathetic function	Increase sympathetic tone

Disorders of the Autonomic Nervous System

Approximately 33 percent of people experience a mild problem with motion sickness, whereas up to 66 percent experience motion sickness under extreme conditions, such as being on a tossing boat with no view of the horizon. Connections between regions in the brain stem and the autonomic system result in the symptoms of nausea, cold sweats, and vomiting.

The part of the brain responsible for vomiting, or emesis, is known as the area postrema. It is located next to the fourth ventricle and is not restricted by the blood–brain barrier, which allows it to respond to chemicals in the bloodstream—namely, toxins that will stimulate emesis. There are significant connections between this area, the solitary nucleus, and the dorsal motor nucleus of the vagus nerve. These autonomic system and nuclei connections are associated with the symptoms of motion sickness.

Motion sickness is the result of conflicting information from the visual and vestibular systems. If motion is perceived by the visual system without the complementary vestibular stimuli, or through vestibular stimuli without visual confirmation, the brain stimulates emesis and the associated symptoms. The area postrema, by itself, appears to be able to stimulate emesis in response to toxins in the blood, but it is also connected to the autonomic system and can trigger a similar response to motion.

Autonomic drugs are used to combat motion sickness. Though it is often described as a dangerous and deadly drug, scopolamine is used to treat motion sickness. A popular treatment for motion sickness is the transdermal scopolamine patch. Scopolamine is one of the substances derived from the *Atropa* genus along with atropine. At higher doses, those substances are thought to be poisonous and can lead to an extreme sympathetic syndrome. However, the transdermal patch regulates the release of the drug, and the concentration is kept very low so that the dangers are avoided. For those who are concerned about using “The Most Dangerous Drug,” as some websites will call it, antihistamines such as dimenhydrinate (Dramamine®) can be used.

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4.5: Terms and Definitions

Key Terms

Select to View Definition

acetylcholine (ACh)
neurotransmitter that binds at a motor end-plate to trigger depolarization

adrenal medulla
interior portion of the adrenal (or suprarenal) gland that releases epinephrine and norepinephrine into the bloodstream as hormones

adrenergic
synapse where norepinephrine is released, which binds to α- or β-adrenergic receptors

afferent branch
component of a reflex arc that represents the input from a sensory neuron, for either a special or general sense

agonist
any exogenous substance that binds to a receptor and produces a similar effect to the endogenous ligand

alpha (α)-adrenergic receptor
one of the receptors to which epinephrine and norepinephrine bind, which comes in two subtypes: α_1 and α_2

antagonist
any exogenous substance that binds to a receptor and produces an opposing effect to the endogenous ligand

anticholinergic drugs
drugs that interrupt or reduce the function of the parasympathetic system

autonomic tone
tendency of an organ system to be governed by one division of the autonomic nervous system over the other, such as heart rate being lowered by parasympathetic input at rest

baroreceptor
mechanoreceptor that senses the stretch of blood vessels to indicate changes in blood pressure

beta (β)-adrenergic receptor
one of the receptors to which epinephrine and norepinephrine bind, which comes in three subtypes: β_1, β_2, and β_3

cardiac accelerator nerves
preganglionic sympathetic fibers that cause the heart rate to increase when the cardiovascular center in the medulla initiates a signal

cardiovascular center
region in the medulla that controls the cardiovascular system through cardiac accelerator nerves and vasomotor nerves, which are components of the sympathetic division of the autonomic nervous system

celiac ganglion
one of the collateral ganglia of the sympathetic system that projects to the digestive system

central neuron
specifically referring to the cell body of a neuron in the autonomic system that is located in the central nervous system, specifically the lateral horn of the spinal cord or a brain stem nucleus

cholinergic
synapse at which acetylcholine is released and binds to the nicotinic or muscarinic receptor

chromaffin cells
neuroendocrine cells of the adrenal medulla that release epinephrine and norepinephrine into the bloodstream as part of sympathetic system activity

ciliary ganglion
one of the terminal ganglia of the parasympathetic system, located in the posterior orbit, axons from which project to the iris

collateral ganglia
ganglia outside of the sympathetic chain that are targets of sympathetic preganglionic fibers, which are the celiac, inferior mesenteric, and superior mesenteric ganglia

craniosacral system
alternate name for the parasympathetic division of the autonomic nervous system that is based on the anatomical location of central neurons in brain-stem nuclei and the lateral horn of the sacral spinal cord; also referred to as craniosacral outflow

dorsal longitudinal fasciculus
major output pathway of the hypothalamus that descends through the gray matter of the brain stem and into the spinal cord

dorsal nucleus of the vagus nerve
location of parasympathetic neurons that project through the vagus nerve to terminal ganglia in the thoracic and abdominal cavities

Edinger–Westphal nucleus
location of parasympathetic neurons that project to the ciliary ganglion

efferent branch
component of a reflex arc that represents the output, with the target being an effector, such as muscle or glandular tissue

endogenous
describes substance made in the human body

endogenous chemical
substance produced and released within the body to interact with a receptor protein

epinephrine
signaling molecule released from the adrenal medulla into the bloodstream as part of the sympathetic response

exogenous
describes substance made outside of the human body

exogenous chemical
substance from a source outside the body, whether it be another organism such as a plant or from the synthetic processes of a laboratory, that binds to a transmembrane receptor protein

fight-or-flight response
set of responses induced by sympathetic activity that lead to either fleeing a threat or standing up to it, which in the modern world is often associated with anxious feelings

G protein–coupled receptor
membrane protein complex that consists of a receptor protein that binds to a signaling molecule—a G protein—that is activated by that binding and in turn activates an effector protein (enzyme) that creates a second-messenger molecule in the cytoplasm of the target cell

ganglionic neuron
: specifically refers to the cell body of a neuron in the autonomic system that is located in a ganglion

gray rami communicantes
: (singular = ramus communicans) unmyelinated structures that provide a short connection from a sympathetic chain ganglion to the spinal nerve that contains the postganglionic sympathetic fiber

greater splanchnic nerve
: nerve that contains fibers of the central sympathetic neurons that do not synapse in the chain ganglia but project onto the celiac ganglion

inferior mesenteric ganglion
: one of the collateral ganglia of the sympathetic system that projects to the digestive system

intramural ganglia
: terminal ganglia of the parasympathetic system that are found within the walls of the target effector

lesser splanchnic nerve
: nerve that contains fibers of the central sympathetic neurons that do not synapse in the chain ganglia but project onto the inferior mesenteric ganglion

ligand-gated cation channel
: ion channel, such as the nicotinic receptor, that is specific to positively charged ions and opens when a molecule such as a neurotransmitter binds to it

limbic lobe
: structures arranged around the edges of the cerebrum that are involved in memory and emotion

long reflex
: reflex arc that includes the central nervous system

medial forebrain bundle
: fiber pathway that extends anteriorly into the basal forebrain, passes through the hypothalamus, and extends into the brain stem and spinal cord

mesenteric plexus
: nervous tissue within the wall of the digestive tract that contains neurons that are the targets of autonomic preganglionic fibers and that project to the smooth muscle and glandular tissues in the digestive organ

muscarinic receptor
: type of acetylcholine receptor protein that is characterized by also binding to muscarine and is a metabotropic receptor

mydriasis
: dilation of the pupil; typically the result of disease, trauma, or drugs

nicotinic receptor
: type of acetylcholine receptor protein that is characterized by also binding to nicotine and is an ionotropic receptor

norepinephrine
: signaling molecule released as a neurotransmitter by most postganglionic sympathetic fibers as part of the sympathetic response, or as a hormone into the bloodstream from the adrenal medulla

nucleus ambiguus
: brain-stem nucleus that contains neurons that project through the vagus nerve to terminal ganglia in the thoracic cavity; specifically associated with the heart

parasympathetic division
: division of the autonomic nervous system responsible for restful and digestive functions

parasympathomimetic drugs
drugs that enhance or mimic the function of the parasympathetic system

paravertebral ganglia
autonomic ganglia superior to the sympathetic chain ganglia

postganglionic fiber
axon from a ganglionic neuron in the autonomic nervous system that projects to and synapses with the target effector; sometimes referred to as a postganglionic neuron

preganglionic fiber
axon from a central neuron in the autonomic nervous system that projects to and synapses with a ganglionic neuron; sometimes referred to as a preganglionic neuron

prevertebral ganglia
autonomic ganglia that are anterior to the vertebral column and functionally related to the sympathetic chain ganglia

referred pain
the conscious perception of visceral sensation projected to a different region of the body, such as the left shoulder and arm pain as a sign for a heart attack

reflex arc
circuit of a reflex that involves a sensory input and motor output, or an afferent branch and an efferent branch, and an integrating center to connect the two branches

rest and digest
set of functions associated with the parasympathetic system that lead to restful actions and digestion

short reflex
reflex arc that does not include any components of the central nervous system

somatic reflex
reflex involving skeletal muscle as the effector, under the control of the somatic nervous system

superior cervical ganglion
one of the paravertebral ganglia of the sympathetic system that projects to the head

superior mesenteric ganglion
one of the collateral ganglia of the sympathetic system that projects to the digestive system

sympathetic chain ganglia
series of ganglia adjacent to the vertebral column that receive input from central sympathetic neurons

sympathetic division
division of the autonomic nervous system associated with the fight-or-flight response

sympatholytic drug
drug that interrupts, or “lyses,” the function of the sympathetic system

sympathomimetic drug
drug that enhances or mimics the function of the sympathetic system

target effector
organ, tissue, or gland that will respond to the control of an autonomic or somatic or endocrine signal

terminal ganglia
ganglia of the parasympathetic division of the autonomic system, which are located near or within the target effector, the latter also known as intramural ganglia

thoracolumbar system
alternate name for the sympathetic division of the autonomic nervous system that is based on the anatomical

location of central neurons in the lateral horn of the thoracic and upper lumbar spinal cord

varicosity
: structure of some autonomic connections that is not a typical synaptic end bulb, but a string of swellings along the length of a fiber that makes a network of connections with the target effector

vasomotor nerves
: preganglionic sympathetic fibers that cause the constriction of blood vessels in response to signals from the cardiovascular center

visceral reflex
: reflex involving an internal organ as the effector, under the control of the autonomic nervous system

white rami communicantes
: (singular = ramus communicans) myelinated structures that provide a short connection from a sympathetic chain ganglion to the spinal nerve that contains the preganglionic sympathetic fiber

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4.6: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. Which of these physiological changes is typical of the sympathetic “fight-or-flight” response?

   A. Increased heart rate
   B. Increased stomach motility
   C. Increased saliva for digestion
   D. Constriction of the pupil

2. Which type of autonomic fiber is generally the longest?
   A. Preganglionic parasympathetic fiber
   B. Preganglionic sympathetic fiber
   C. Postganglionic parasympathetic fiber
   D. Postganglionic sympathetic fiber

3. Which signaling molecule is most directly linked to “rest-and-digest” activity in the gut (for example, increased digestive activity)?
   A. Epinephrine
   B. Norepinephrine
   C. Acetylcholine
   D. Adrenaline

4. Which of these cranial nerves carries preganglionic parasympathetic fibers?
   A. Optic nerve (CN II)
   B. Facial nerve (CN VII)
   C. Trigeminal nerve (CN V)
   D. Hypoglossal nerve (CN XII)

5. Sympathetic preganglionic fibers commonly synapse in which structure?
   A. Intramural (terminal) ganglion
   B. Prevertebral (collateral) ganglion
   C. Parasympathetic ganglion in the organ wall
   D. Dorsal root ganglion

6. Baroreceptors (pressure receptors in blood vessels) send sensory information that is important for which system?
   A. Only the somatic nervous system
   B. Only the autonomic nervous system
   C. Both the somatic and autonomic systems
   D. Neither somatic nor autonomic systems

7. What is the name for an autonomic reflex that is processed entirely within the enteric nervous system, without going to the brain or spinal cord?
   A. Long reflex
   B. Visceral reflex

C. Somatic reflex
D. Short reflex

8. Which neurotransmitter, acting on the iris sphincter muscle through parasympathetic fibers, causes constriction of the pupil (miosis)?
A. Norepinephrine
B. Acetylcholine
C. Epinephrine
D. Dopamine

9. Which gland releases epinephrine and norepinephrine into the blood during a fight-or-flight response?
A. Adrenal medulla
B. Thyroid gland
C. Pituitary gland
D. Pancreas

10. Which pairing of neurotransmitter and effect is correct for sympathetic activity?
A. Norepinephrine increases heart rate
B. Acetylcholine dilates the pupil
C. Epinephrine slows the heart rate
D. Acetylcholine causes bronchodilation

11. Which forebrain structure is the main control center for autonomic function and endocrine regulation of homeostasis?
A. Hypothalamus
B. Thalamus
C. Amygdala
D. Hippocampus

12. Which nerve carries visual information, including light levels, from the retina to the brain and helps set circadian rhythms?
A. Glossopharyngeal nerve (CN IX)
B. Oculomotor nerve (CN III)
C. Optic nerve (CN II)
D. Vagus nerve (CN X)

13. Which region of the limbic system is especially important for fear and stress responses that activate the hypothalamus and autonomic output?
A. Hippocampus
B. Amygdala
C. Mammillary bodies
D. Cingulate gyrus

14. What is a common name for the preganglionic sympathetic fibers that project to the heart and increase heart rate and contractility?
A. Solitary tract
B. Vasomotor nerve
C. Vagus nerve
D. Cardiac accelerator nerve

15. Which central fiber tract connects limbic and forebrain areas with the hypothalamus to coordinate emotional states and autonomic responses?
A. Medial forebrain bundle
B. Corticospinal tract
C. Optic radiation
D. Dorsal column pathway

16. Nicotinic acetylcholine receptors in the autonomic nervous system are found mainly at which location?
A. On target organs of sympathetic fibers
B. On target organs of parasympathetic fibers

C. In autonomic ganglia on postganglionic neurons
D. Only in skeletal muscle at the neuromuscular junction

17. A drug is called an agonist when it:
A. Blocks a receptor so the neurotransmitter cannot bind
B. Prevents breakdown of the neurotransmitter
C. Acts like the natural neurotransmitter and activates the receptor
D. Increases reuptake of the neurotransmitter

18. Atropine blocks muscarinic acetylcholine receptors. Which type of drug would help reverse atropine poisoning?
A. Nicotinic agonist
B. Additional anticholinergic drug
C. Muscarinic agonist
D. β-adrenergic blocker

19. A drug that reduces anxiety by lowering heart rate and blood pressure usually acts as a:
A. Nicotinic agonist
B. α-adrenergic blocker
C. Muscarinic agonist
D. Sympathomimetic drug

20. A drug used to treat asthma by causing bronchodilation (opening the airways) would most likely be:
A. Sympathomimetic drug that stimulates β-adrenergic receptors
B. Sympatholytic drug that blocks all sympathetic effects
C. Parasympathomimetic drug that stimulates muscarinic receptors
D. Drug that increases parasympathetic activity in the lungs

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4.7: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. The autonomic nervous system (ANS) has two main parts: sympathetic and parasympathetic. Why is it important for the body to have both “fight or flight” and “rest and digest” systems?
   Hint: Think about what the body needs during danger (running, fighting) and what it needs during rest (digesting food, saving energy).

2. Imagine you suddenly see a car coming toward you while crossing the street. Describe how your sympathetic nervous system might change your body functions in the next few seconds.
   Hint: Think about heart rate, breathing, pupils, blood flow to muscles, and sweating.

3. After a big meal, the parasympathetic system becomes more active. Explain why this is helpful for the body and what might happen if the sympathetic system stayed dominant instead.
   Hint: Think about digestion, blood flow to the intestines, and energy use.

4. Many organs receive both sympathetic and parasympathetic nerve supply. Why is this “dual innervation” useful for fine control of organ function?
   Hint: Think of a car with both an accelerator and a brake. How does this help with smooth control, not just on/off?

5. The ANS works mostly without conscious control. Describe one situation where this automatic control protects you, and one situation where it might cause a problem (for example, in chronic stress or anxiety).
   Hint: Think about automatic changes in heart rate and blood pressure; also think about what happens if these changes are too strong or too long.

6. Some people faint when they stand up quickly because blood pressure drops and the ANS cannot adjust fast enough. Explain how a healthy ANS normally responds when a person stands up from lying down.
   Hint: Think about gravity, blood pooling in the legs, and how the sympathetic system changes heart rate and blood vessel diameter.

7. Drugs like beta-blockers are used to treat high blood pressure by affecting part of the sympathetic nervous system. Predict one effect these drugs might have on exercise ability and explain why.
   Hint: Think about how the sympathetic system normally changes heart rate and force of contraction during exercise.

8. Long-term stress can keep the sympathetic system more active than normal. Explain two possible effects this might have on different body systems (for example, heart, digestion, immune system).

Hint: Think about what happens if “fight or flight” reactions continue for days or weeks, not just a few minutes.

9. The ANS controls smooth muscle, cardiac muscle, and glands. Choose one of these (for example, glands) and explain how autonomic control of it helps the body maintain homeostasis.
   Hint: For glands, think about sweating, saliva, or hormone release and how these help keep internal conditions stable.

10. Some people can slow their heart rate and breathing through relaxation techniques or meditation, even though these are autonomic functions. What does this suggest about possible connections between the ANS and higher brain centers?
    Hint: Think about how emotions, thoughts, and conscious practices might influence autonomic output coming from the brain.

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4.8: Alternate Text Descriptions

Figure 4.1.1: Sympathetic Innervation of the Body

The figure illustrates the **sympathetic division of the autonomic nervous system**, showing the pathways by which sympathetic nerve fibers travel from the spinal cord to organs throughout the body.

1. The **spinal cord** is shown centrally, extending from the brainstem to the lower spinal regions.
2. On either side of the spinal cord is a chain of interconnected **sympathetic chain ganglia**, extending from the cervical region to the coccyx. The most inferior ganglia are fused to form the **coccygeal ganglion**.
3. In the neck, the sympathetic chain includes the **superior**, **middle**, and **inferior cervical sympathetic ganglia**, which provide sympathetic innervation to structures in the head and neck.
4. Sympathetic fibers arise from thoracic and upper lumbar spinal cord segments and enter the sympathetic chain through spinal nerves and **gray rami**.
5. The diagram distinguishes:
    - **Preganglionic neurons**, shown in one color, extending from the spinal cord to sympathetic ganglia.
    - **Postganglionic neurons**, shown in a different color, extending from ganglia to target organs.
6. Sympathetic fibers travel to structures of the head, including the **eye** and **salivary glands**.
7. Fibers from the cervical and upper thoracic regions contribute to the **cardiac and pulmonary plexuses**, which distribute sympathetic innervation to the **heart** and **lungs**.
8. Some preganglionic fibers travel through **greater splanchnic nerves** and other **splanchnic nerves** to prevertebral ganglia located anterior to the vertebral column.
9. Major prevertebral ganglia shown include:
    - **Celiac ganglion**
    - **Superior mesenteric ganglion**
    - **Inferior mesenteric ganglion**
10. Postganglionic fibers from these ganglia project to abdominal organs, including the:

- Liver and gallbladder
- Stomach
- Spleen
- Pancreas
- Small intestine
- Large intestine
- Rectum

11. Additional sympathetic pathways extend to the:

- Adrenal medulla
- Kidneys
- Urinary bladder

12. Sympathetic fibers also project to reproductive organs, including the:

- Uterus
- Ovaries
- Penis
- Scrotum

13. An inset of the skin illustrates postganglionic sympathetic fibers traveling through spinal nerves to the **integumentary system**, where they innervate structures such as sweat glands, blood vessels, and arrector pili muscles.

The figure demonstrates the widespread distribution of the sympathetic nervous system. Preganglionic neurons originating in the thoracic and upper lumbar spinal cord synapse in sympathetic chain ganglia or prevertebral ganglia, and postganglionic neurons distribute signals to organs throughout the head, thorax, abdomen, pelvis, and skin. These pathways support the body's **fight-or-**

flight responses by regulating cardiac activity, respiration, circulation, digestion, sweating, and other autonomic functions.

Figure 4.1.2: Three Pathways of Sympathetic Nervous System Innervation

The figure illustrates three pathways through which sympathetic preganglionic neurons reach postganglionic neurons and target organs. Each panel shows a transverse section of the spinal cord, a spinal nerve, the sympathetic trunk, and associated ganglia.

Panel (a): Synapse at the Same Level in the Sympathetic Chain

1. A preganglionic sympathetic neuron originates in the spinal cord and exits through a spinal nerve.
2. The neuron enters the sympathetic trunk through the **white ramus communicans**.
3. The preganglionic neuron synapses within a **sympathetic chain ganglion** at the same spinal level.
4. A postganglionic neuron then exits the ganglion and travels to a target effector organ.
5. This pathway demonstrates sympathetic innervation in which synapse occurs in the ganglion nearest the spinal cord segment of origin.

Panel (b): Synapse in a Higher or Lower Sympathetic Chain Ganglion

1. A preganglionic sympathetic neuron exits the spinal cord and enters the sympathetic trunk.
2. Instead of synapsing immediately, the neuron ascends or descends within the sympathetic chain.
3. The neuron synapses in a sympathetic chain ganglion located at a different spinal level.
4. A postganglionic neuron leaves the ganglion through a **gray ramus communicans** or other sympathetic pathway.
5. The postganglionic fiber proceeds to a target effector organ.
6. This pathway allows sympathetic signals from thoracolumbar spinal cord segments to reach structures located in the head, neck, or lower body.

Panel (c): Synapse in a Prevertebral Ganglion

1. A preganglionic sympathetic neuron exits the spinal cord and enters the sympathetic trunk through the **white ramus communicans**.
2. The neuron passes through the sympathetic chain ganglion without synapsing.
3. The preganglionic fiber continues as a **splanchnic nerve**.
4. The splanchnic nerve travels to a **prevertebral ganglion** located anterior to the vertebral column.
5. The preganglionic neuron synapses within the prevertebral ganglion.
6. A postganglionic neuron then projects to a target effector organ, typically within the abdominal or pelvic cavities.

The figure demonstrates three major routes used by sympathetic fibers after leaving the spinal cord:

- Synapsing in a sympathetic chain ganglion at the same spinal level
- Ascending or descending within the sympathetic chain before synapsing
- Passing through the sympathetic chain and synapsing in a prevertebral ganglion via a splanchnic nerve

These pathways allow the sympathetic nervous system to distribute autonomic signals to organs throughout the head, neck, thorax, abdomen, pelvis, and body wall.

Figure 4.1.3: Parasympathetic Innervation of the Body

The figure illustrates the **parasympathetic division of the autonomic nervous system**, showing the pathways that originate in the brainstem and sacral spinal cord and extend to organs throughout the body. Preganglionic fibers arise from specific cranial nerves and pelvic nerves, travel to ganglia located near or within target organs, and synapse with postganglionic neurons that regulate organ function.

1. Parasympathetic pathways originate from cranial nerves **III (oculomotor)**, **VII (facial)**, **IX (glossopharyngeal)**, and **X (vagus)**, as well as from **pelvic nerves** arising from the sacral spinal cord.
2. The **oculomotor nerve (CN III)** projects to the **ciliary ganglion**, which sends fibers to the **eye** and controls pupil

constriction and lens accommodation.
3. The **facial nerve (CN VII)** projects to:
    - The **pterygopalatine ganglion**, which supplies the **lacrimal gland**.
    - The **submandibular ganglion**, which supplies the **salivary glands**.
4. The **glossopharyngeal nerve (CN IX)** projects to the **otic ganglion**, which provides parasympathetic innervation to a salivary gland.
5. The **vagus nerve (CN X)** provides extensive parasympathetic innervation through autonomic plexuses and supplies:
    - The **heart**
    - The **lungs**
    - The **liver**
    - The **gallbladder**
    - The **stomach**
    - The **spleen**
    - The **pancreas**
    - The **small intestine**
    - Much of the **large intestine**
6. Parasympathetic fibers from the sacral spinal cord travel through **pelvic nerves** and autonomic plexuses to innervate:
    - The distal **large intestine**
    - The **rectum**
    - The **urinary bladder**
    - Reproductive organs, including the **uterus**, **ovaries**, **penis**, and associated structures
7. Several **autonomic plexuses** distribute parasympathetic fibers to thoracic, abdominal, and pelvic organs.
8. Unlike sympathetic pathways, most parasympathetic ganglia are located close to or within the target organs, resulting in relatively short postganglionic fibers.

The figure demonstrates the widespread distribution of parasympathetic innervation throughout the body. Parasympathetic pathways regulate functions associated with the **rest-and-digest** state, including slowing heart rate, promoting digestion, stimulating glandular secretions, supporting urinary function, and regulating reproductive organs.

Figure 4.2.1: Autonomic Neuroeffector Junction Between a Postganglionic Axon and Smooth Muscle

The figure illustrates an autonomic neuroeffector junction between a **postganglionic autonomic nerve fiber** and **smooth muscle**.

1. On the left, a **postganglionic axon** travels through smooth muscle tissue. Along the axon are several enlarged regions labeled **postganglionic varicosities**.
2. The enlarged panel shows one varicosity in detail. The varicosity contains numerous **synaptic vesicles** filled with neurotransmitter molecules.
3. Neurotransmitters are released from the varicosity into the surrounding extracellular space rather than into a single, specialized synaptic cleft.
4. The released neurotransmitter molecules diffuse through the tissue toward the target smooth muscle cells.
5. The surface membrane of the smooth muscle cell is labeled the **sarcolemma**.
6. Embedded within the sarcolemma are **neurotransmitter receptors**, shown distributed across the membrane.
7. Neurotransmitter molecules bind to these receptors, allowing the autonomic nerve fiber to influence the activity of the smooth muscle.
8. Multiple varicosities along the length of the postganglionic axon can release neurotransmitter, allowing signals to be distributed over a broader area of smooth muscle tissue.

The figure demonstrates how autonomic postganglionic neurons communicate with smooth muscle. Unlike the highly localized neuromuscular junction of skeletal muscle, autonomic axons release neurotransmitters from multiple varicosities along their length, enabling diffuse regulation of smooth muscle activity through receptors distributed across the target tissue.

Figure 4.2.2: Referred Pain Patterns of Internal Organs

The figure shows anterior and posterior views of the human body illustrating the **referred pain patterns of internal organs**.

Colored regions on the skin indicate areas where pain originating from specific organs may be perceived, even though the source of the pain is located internally.

Anterior View

1. The **lung and diaphragm** are associated with pain referred to the neck and shoulder region.
2. The **heart** is associated with pain referred to the left side of the chest, left shoulder, and along the medial aspect of the left upper limb.
3. The **liver and gallbladder** are associated with pain in the right upper abdomen and lower chest region.
4. The **stomach** is associated with pain in the upper central abdomen.
5. The **pancreas** is associated with pain in the upper abdominal region.
6. The **small intestine** is associated with pain around the umbilical region.
7. The **appendix** is associated with pain in the lower right abdominal region.
8. The **colon** is associated with pain across portions of the lower abdomen.
9. The **ovary** is associated with pain in the lower pelvic region.
10. The **kidney** is associated with pain in the flank and lower abdominal regions.
11. The **urinary bladder** is associated with pain in the lower midline pelvic region.
12. The **ureter** is associated with pain radiating from the lower abdomen into the groin region.
13. The **thymus** is shown in the upper chest region.
14. The **spleen** is identified near the left upper abdominal region.

Posterior View

1. The **lung and diaphragm** are associated with referred pain across the posterior neck and shoulder regions.
2. The **heart** is associated with pain referred to the upper thoracic back.
3. The **stomach** is associated with pain in the mid-back region.
4. The **liver and gallbladder** are associated with pain in the right upper back and shoulder area.
5. The **pancreas** is associated with pain in the upper lumbar region.
6. The **kidney** is associated with pain in the lower back and flank regions.
7. The **large and small intestines** are associated with pain in the lower abdominal and corresponding posterior trunk regions.
8. The **rectum**, **bladder**, and reproductive organs are associated with pain in the lower pelvic and sacral regions.

The figure demonstrates **referred pain**, a phenomenon in which pain from an internal organ is perceived in a specific area of the body surface. Different organs produce characteristic patterns of referred pain because sensory pathways from visceral organs and somatic structures converge within the nervous system. These patterns can help clinicians identify the likely source of internal pain.

Figure 4.2.3: Comparison of Autonomic and Somatic Efferent Motor Pathways

The figure compares the organization of the **autonomic efferent pathway** and the **somatic efferent pathway**, highlighting differences in the number of neurons, myelination, target tissues, and synaptic connections.

Top Panel: Autonomic Efferent Pathway

1. An autonomic motor pathway begins with a neuron in the spinal cord.
2. A **preganglionic, or presynaptic, autonomic neuron** extends from the spinal cord to an autonomic ganglion. This axon is labeled **myelinated**.
3. Within the autonomic ganglion, the preganglionic neuron synapses with a **ganglionic, or postganglionic, neuron**.
4. The **postganglionic axon** extends from the ganglion to the target tissue and is labeled **unmyelinated**.
5. The target effector is **smooth muscle**.
6. An enlarged inset shows a **postganglionic varicosity**, an enlarged region along the axon containing neurotransmitter-filled vesicles.
7. Neurotransmitters are released from the varicosities and diffuse across the surrounding tissue to reach receptors on the target smooth muscle.

8. The inset emphasizes that the autonomic axon does **not necessarily end at a specialized synapse**. Instead, neurotransmitter release occurs from multiple varicosities distributed along the axon.

Bottom Panel: Somatic Efferent Pathway

1. A somatic motor pathway begins with a **somatic motor neuron** located in the spinal cord.
2. A single **myelinated axon** extends directly from the spinal cord to the target tissue.
3. No peripheral ganglion is present between the spinal cord and the effector.
4. The target effector is **skeletal muscle**.
5. The axon terminates at a specialized **neuromuscular junction** on the skeletal muscle fiber.
6. An enlarged inset shows the **synaptic terminal** at the end of the axon.
7. Neurotransmitter is released into a localized synaptic cleft directly adjacent to receptors on the muscle fiber membrane.
8. The inset emphasizes that the somatic motor axon **ends at a well-defined synapse**, creating a highly localized connection with the skeletal muscle fiber.

The figure demonstrates the key differences between autonomic and somatic motor pathways. The **autonomic pathway** uses a two-neuron chain consisting of a myelinated preganglionic neuron and an unmyelinated postganglionic neuron that influences smooth muscle through diffuse neurotransmitter release from varicosities. In contrast, the **somatic pathway** uses a single myelinated motor neuron that projects directly from the spinal cord to skeletal muscle and forms a specialized neuromuscular junction at the end of its axon.

Figure 4.2.4: Comparison of Long and Short Autonomic Reflex Pathways

The figure compares **long reflexes** and **short reflexes** in the autonomic nervous system, illustrating the pathways by which sensory information leads to autonomic responses.

Long Reflex

1. A **sensory receptor cell** detects a stimulus, identified by arrows labeled **sensation**.
2. A sensory neuron carries the signal toward the central nervous system.
3. The sensory neuron's cell body is located in a **peripheral ganglion**.
4. The sensory axon enters the **spinal cord** or brain, where the sensory neuron synapses with neurons of the central nervous system.
5. Central processing occurs within the spinal cord or brain before an autonomic response is generated.
6. Motor output returns through autonomic pathways to target tissues, represented by smooth muscle and associated autonomic nerve fibers.
7. The label indicates that the **sensory cell synapses in the spinal cord or brain**, demonstrating involvement of the central nervous system in the reflex pathway.

Short Reflex

1. A **sensory receptor cell** detects a stimulus and initiates a sensory signal.
2. The sensory neuron carries the signal to a **peripheral ganglion**.
3. The sensory neuron synapses within the peripheral ganglion rather than projecting to the spinal cord or brain.
4. Integration and processing occur entirely within the peripheral autonomic ganglion.
5. A motor neuron within the ganglion sends signals directly to the target tissue.
6. The label indicates that the **sensory cell synapses only in the peripheral ganglion**, demonstrating that the central nervous system is not directly involved in this reflex pathway.

The figure compares two autonomic reflex pathways. In a **long reflex**, sensory information travels to the spinal cord or brain for processing before an autonomic response is produced. In a **short reflex**, both sensory input and motor output are integrated within a peripheral autonomic ganglion, allowing the reflex to occur without direct involvement of the central nervous system.

Figure 4.3.1: Hypothalamic Pathways to Brainstem and Spinal Autonomic Centers

The figure shows a midsagittal view of the brain highlighting pathways through which the **hypothalamus** communicates with autonomic control centers in the brainstem and spinal cord.

1. An inset on the left shows the location of the sagittal section within the brain. The main image enlarges the midline structures of the forebrain and brainstem.
2. The **hypothalamus** is highlighted near the base of the diencephalon. It serves as a major control center for autonomic and homeostatic functions.
3. Two major fiber pathways originate from or pass through the hypothalamus:
   - The **medial forebrain bundle**, shown in orange
   - The **dorsal longitudinal fasciculus**, shown in pink
4. The **medial forebrain bundle** extends between the hypothalamus and other regions of the forebrain and brainstem, providing communication among autonomic and limbic structures.
5. The **dorsal longitudinal fasciculus** descends from the hypothalamus through the brainstem and continues toward the spinal cord, carrying autonomic control signals.
6. Several autonomic nuclei within the brainstem are labeled:
   - The **oculomotor nucleus complex**, including the **Edinger-Westphal nucleus**, which contributes parasympathetic control of the eye
   - The **dorsal motor nucleus of the vagus**, which provides parasympathetic output to thoracic and abdominal organs
   - The **nucleus ambiguus**, which contributes autonomic control of the heart, pharynx, and larynx
7. Connections from the hypothalamus reach these brainstem nuclei through the descending autonomic pathways.
8. An arrow labeled **To spinal cord** indicates that autonomic signals continue downward from the brainstem into the spinal cord, where they influence sympathetic and parasympathetic pathways.

The figure demonstrates how the hypothalamus coordinates autonomic function by communicating with cranial nerve nuclei in the brainstem and with autonomic centers in the spinal cord through the **medial forebrain bundle** and **dorsal longitudinal fasciculus**. These pathways help regulate functions such as pupil constriction, cardiovascular activity, digestion, and other homeostatic processes.

Figure 4.3.2: Major Structures of the Limbic System

The figure shows a midsagittal view of the brain highlighting major components of the **limbic system** and their relationships to nearby structures involved in emotion, memory, motivation, and autonomic regulation.

1. The **cingulate gyrus** forms a curved band along the medial surface of the cerebrum above the corpus callosum. It is a component of the limbic system involved in emotional processing, learning, attention, and behavior.
2. The **corpus callosum** lies beneath the cingulate gyrus and forms a large fiber tract connecting the left and right cerebral hemispheres.
3. The **thalamus** is located near the center of the brain and serves as a major relay center for sensory and other information traveling to the cerebral cortex.
4. The **hypothalamic nuclei** are located inferior to the thalamus. These nuclei help regulate autonomic function, endocrine activity, homeostasis, and emotional responses.
5. The **hippocampus** is located in the medial temporal lobe and forms a curved structure extending along the inferior portion of the limbic system. It plays a key role in learning, memory formation, and spatial navigation.
6. The **amygdala** is positioned near the anterior end of the hippocampus. It is involved in emotional processing, particularly fear, motivation, and emotional memory.
7. Colored pathways illustrate the interconnected nature of these limbic structures and their communication with one another.
8. An arrow leading toward the hypothalamic region indicates integration of emotional, behavioral, and autonomic responses within the limbic system.

The figure demonstrates the anatomical organization of the limbic system and emphasizes the connections among the cingulate gyrus, hippocampus, amygdala, hypothalamus, and thalamus. Together, these structures contribute to memory, emotion, motivation, learning, and regulation of autonomic and endocrine functions.

Figure 4.4.1: Sympathetic and Parasympathetic Innervation of the Heart

The figure illustrates the autonomic innervation of the **heart**, comparing sympathetic and parasympathetic pathways from their origins in the central nervous system to their target organ.

1. The diagram is organized into three columns: **region of the central nervous system**, **associated nerves and ganglia**, and **target organ**.
2. The **heart** is shown on the right as the target organ receiving both sympathetic and parasympathetic autonomic input.

Parasympathetic Pathway

1. Parasympathetic fibers originate in the **medulla oblongata**, specifically from the **dorsal motor nucleus of the vagus nerve** and the **nucleus ambiguus**.
2. These fibers travel through **cranial nerve X, the vagus nerve,** toward the heart.
3. The parasympathetic pathway is represented by a blue line extending directly from the medulla to the heart.
4. Parasympathetic stimulation generally slows heart rate and reduces cardiac activity.

Sympathetic Pathway

1. Sympathetic fibers originate from neurons located in the upper thoracic spinal cord, primarily at spinal cord levels **T1 through T6**.
2. Preganglionic fibers enter the **sympathetic chain** and synapse within sympathetic chain ganglia, including the **superior cervical ganglion** and upper thoracic ganglia.
3. Postganglionic sympathetic fibers leave these ganglia and project to the heart.
4. The sympathetic pathways are represented by red lines extending from the thoracic spinal cord through the sympathetic chain ganglia to the heart.
5. Sympathetic stimulation generally increases heart rate, force of contraction, and cardiac output.

The figure demonstrates the dual autonomic control of the heart. **Parasympathetic fibers** originate in the medulla and reach the heart through the **vagus nerve**, while **sympathetic fibers** originate in the upper thoracic spinal cord and travel through the sympathetic chain ganglia before reaching the heart. The coordinated activity of these pathways helps regulate heart rate and cardiac function according to the body's physiological needs.

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5: THE BODY'S TRANSPORT AND DEFENSE SYSTEM

Chapter Overview

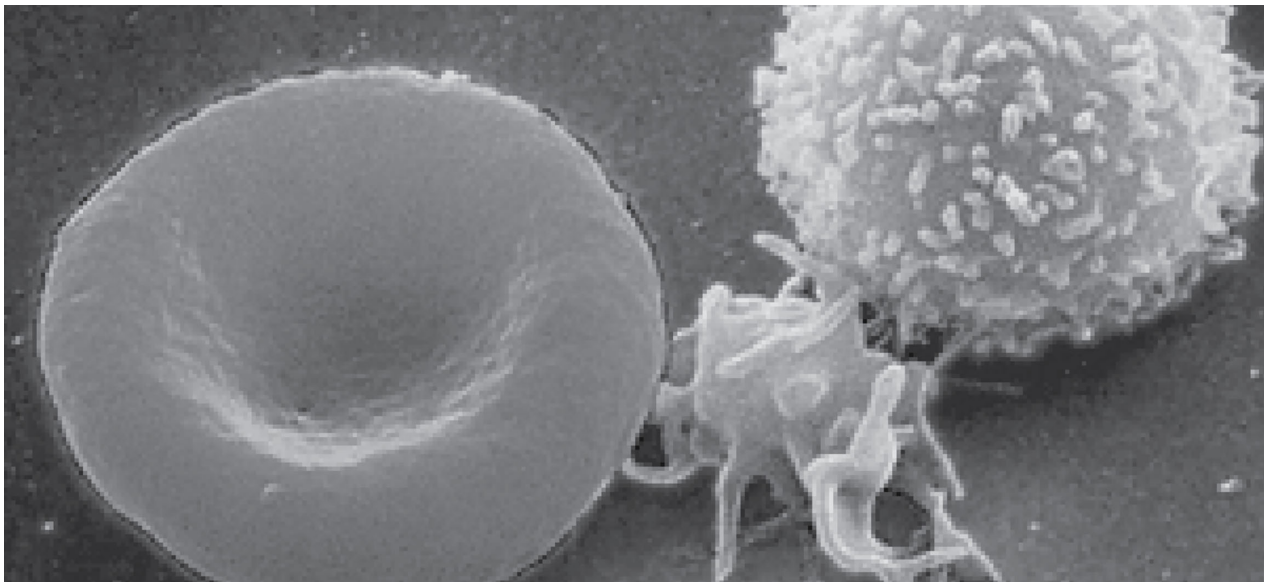


Figure 5.1: A single drop of blood contains millions of red blood cells, white blood cells, and platelets. One of each type is shown here, isolated from a scanning electron micrograph. (CC BY 4.0; via LibreTexts).

Blood is a fluid connective tissue essential for survival. It transports nutrients, gases, hormones, and wastes; defends the body against infection and other threats; and helps maintain homeostasis of pH, temperature, and fluid balance. Blood consists of formed elements—erythrocytes (red blood cells), leukocytes (white blood cells), and platelets—and a fluid extracellular matrix called plasma. More than 90 percent of plasma is water; the rest is primarily plasma proteins (albumin, globulins, fibrinogen) and dissolved solutes such as glucose, lipids, electrolytes, and gases. Because of its cells, proteins, and solutes, blood is thicker and stickier than water, slightly alkaline, and slightly warmer than core body temperature.

Production of the Formed Elements (Hemopoiesis)

Through hemopoiesis, the formed elements are continually replaced as older cells wear out and are removed. This process occurs mainly in red bone marrow, where hematopoietic stem cells give rise to two major lineages: myeloid stem cells, which produce most formed elements (erythrocytes, platelets, neutrophils, eosinophils, basophils, and monocytes), and lymphoid stem cells, which produce lymphocytes (B cells, T cells, and NK cells). Hematopoietic growth factors—including erythropoietin, thrombopoietin, colony-stimulating factors, and interleukins—regulate proliferation and differentiation along these pathways.

Erythrocytes: Structure, Function, and Turnover

Erythrocytes are the most abundant formed element. Their biconcave shape and lack of nucleus maximize space for hemoglobin, the oxygen-carrying protein. Each hemoglobin molecule consists of four globin chains, each bound to a heme group containing iron. In the lungs, iron binds oxygen for transport to tissues; globin simultaneously carries some carbon dioxide from tissues back to the lungs. Erythrocytes live about 120 days and are then removed by macrophages, primarily in the spleen and liver. Hemoglobin is broken down and recycled: globin is degraded to amino acids, iron is stored or reused in new erythrocytes, and heme is converted to bilirubin and excreted in bile or urine. Disorders of red cell number or hemoglobin, such as anemia (deficiency) and polycythemia (excess), alter oxygen-carrying capacity and blood viscosity.

Leukocytes and Platelets: Defense and Repair

Leukocytes provide defense against pathogens, abnormal cells, and other threats. They leave the bloodstream by diapedesis, move through tissues, and concentrate where they are needed. Granular leukocytes (neutrophils, eosinophils, basophils) and monocytes arise from the myeloid lineage; lymphocytes (B, T, and NK cells) arise from the lymphoid lineage. Neutrophils are the most numerous and are rapid responders to bacterial infection. Lymphocytes, which make up about 20–30 percent of leukocytes, are central to specific (adaptive) immunity. Malignancies of leukocytes include leukemia and lymphoma. Platelets are small cell fragments derived from megakaryocytes in the bone marrow. Circulating platelets, along with those stored in the spleen, are essential for hemostasis and release growth factors that support tissue repair.

Hemostasis: Stopping Bleeding and Controlling Clotting

Hemostasis is the process that prevents excessive blood loss after vessel injury. It involves three main steps: vascular spasm (immediate vasoconstriction), platelet plug formation, and coagulation, in which a cascade of clotting factors leads to conversion of fibrinogen to fibrin and formation of a stable clot. Later, fibrinolysis dissolves the clot as healing progresses. Endogenous anticoagulants limit clot formation to the injury site. Inadequate clotting may result from too few platelets or deficient clotting factors, as seen in hemophilia. Excessive clotting (thrombosis) involves the formation of a thrombus (stationary clot) that can break free and become an embolus, potentially blocking critical vessels in the lungs, brain, or heart.

Blood Typing: ABO and Rh Systems and Their Clinical Importance

Surface antigens on erythrocytes determine an individual's blood type and are central to the safety of transfusions and pregnancy. Antigens are "nonself" molecules that can provoke immune responses; when incompatible blood is transfused, antibodies bind donor erythrocyte antigens, causing agglutination and hemolysis.

In the ABO system, individuals with type A blood have A antigens on their erythrocytes; type B has B antigens; type AB has both; and type O has neither. Plasma naturally contains preformed antibodies against the A or B antigens that a person's own cells lack. Transfusing incompatible ABO blood can cause life-threatening transfusion reactions, so ABO typing and cross-matching are essential before transfusion.

The Rh system, especially the D antigen, is equally important. People who are Rh+ express the D antigen; those who are Rh– do not. Rh– individuals do not normally have anti-Rh antibodies but can develop them after exposure to Rh+ blood. In pregnancy, an Rh– person carrying an Rh+ fetus can form anti-Rh antibodies; in a subsequent Rh+ pregnancy, these antibodies can cross the placenta and destroy fetal erythrocytes, causing hemolytic disease of the newborn. Prophylactic administration of RhoGAM to Rh– pregnant individuals prevents sensitization. In emergencies when there is no time for full testing, type O– blood—lacking A, B, and Rh D antigens—is used as universal donor red cells.

Together, understanding the composition of blood, the production and function of its formed elements, the mechanisms of hemostasis, and the principles of ABO and Rh blood typing provides the foundation for interpreting common laboratory tests, managing bleeding and clotting disorders, and ensuring safe blood transfusions and pregnancies.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- Describe the basic composition and physical properties of blood, including plasma and formed elements, and relate these to its major transport and regulatory functions.
- Summarize how formed elements are produced in red bone marrow (hemopoiesis) and outline the myeloid and lymphoid lineages.

- Describe the structure, function, and life cycle of erythrocytes and relate common red cell disorders (e.g., anemia, polycythemia) to altered oxygen transport.
- Summarize the main types and functions of leukocytes and platelets in defense, hemostasis, and tissue repair.
- Outline the major steps of hemostasis (vascular spasm, platelet plug, coagulation, fibrinolysis) and distinguish between inadequate and excessive clotting.
- Explain the basis of ABO and Rh blood groups, and relate blood typing and compatibility to transfusion safety and hemolytic disease of the newborn.

5.1: An Overview of Blood
5.2: Production of the Formed Elements
5.3: Erythrocytes
5.4: Leukocytes and Platelets
5.5: Hemostasis
5.6: Blood Typing
5.7: Terms and Definitions
5.8: Chapter Review Questions
5.9: Critical Thinking Self-Assessment
5.10: Alternate Text Descriptions

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5.1: An Overview of Blood

Functions of Blood

Recall that blood is a connective tissue. Like all connective tissues, it is made up of cellular elements and an extracellular matrix. The cellular elements—referred to as the formed elements—include red blood cells (RBCs), white blood cells (WBCs), and cell fragments called platelets. The extracellular matrix, called plasma, makes blood unique among connective tissues because it is fluid. This fluid, which is mostly water, perpetually suspends the formed elements and enables them to circulate throughout the body within the cardiovascular system.

The primary function of blood is to deliver oxygen and nutrients to and remove wastes from body cells, but that is only the beginning of the story. The specific functions of blood also include defense, distribution of heat, and maintenance of homeostasis.

Transportation

Nutrients from the foods you eat are absorbed in the digestive tract. Most of these travel in the bloodstream directly to the liver, where they are processed and released back into the bloodstream for delivery to body cells. Oxygen from the air you breathe diffuses into the blood, which moves from the lungs to the heart, which then pumps it out to the rest of the body. Moreover, endocrine glands scattered throughout the body release their products, called hormones, into the bloodstream, which carries them to distant target cells. Blood also picks up cellular wastes and byproducts, and transports them to various organs for removal. For instance, blood moves carbon dioxide to the lungs for exhalation from the body, and various waste products are transported to the kidneys and liver for excretion from the body in the form of urine or bile.

Defense

Many types of WBCs protect the body from external threats, such as disease-causing bacteria that have entered the bloodstream in a wound. Other WBCs seek out and destroy internal threats, such as cells with mutated DNA that could multiply to become cancerous, or body cells infected with viruses.

When damage to the vessels results in bleeding, blood platelets and certain proteins dissolved in the plasma, the fluid portion of the blood, interact to block the ruptured areas of the blood vessels involved. This protects the body from further blood loss.

Maintenance of Homeostasis

Recall that body temperature is regulated via a classic negative-feedback loop. If you were exercising on a warm day, your rising core body temperature would trigger several homeostatic mechanisms, including increased transport of blood from your core to your body periphery, which is typically cooler. As blood passes through the vessels of the skin, heat would be dissipated to the environment, and the blood returning to your body core would be cooler. In contrast, on a cold day, blood is diverted away from the skin to maintain a warmer body core. In extreme cases, this may result in frostbite.

Blood also helps to maintain the chemical balance of the body. Proteins and other compounds in blood act as buffers, which thereby help to regulate the pH of body tissues. Blood also helps to regulate the water content of body cells.

Composition of Blood

You have probably had blood drawn from a superficial vein in your arm, which was then sent to a lab for analysis. Some of the most common blood tests—for instance, those measuring lipid or glucose levels in plasma—determine which substances are present within blood and in what quantities. Other blood tests check for the composition of the blood itself, including the quantities and types of formed elements.

One such test, called a hematocrit, measures the percentage of RBCs, clinically known as erythrocytes, in a blood sample. It is performed by spinning the blood sample in a specialized centrifuge, a process that causes the heavier elements suspended within the blood sample to separate from the lightweight, liquid plasma (Figure 5.1.1). Because the heaviest elements in blood

are the erythrocytes, these settle at the very bottom of the hematocrit tube. Located above the erythrocytes is a pale, thin layer composed of the remaining formed elements of blood. These are the WBCs, clinically known as leukocytes, and the platelets, cell fragments also called thrombocytes. This layer is referred to as the buffy coat because of its color; it normally constitutes less than 1 percent of a blood sample. Above the buffy coat is the blood plasma, normally a pale, straw-colored fluid, which constitutes the remainder of the sample.

The volume of erythrocytes after centrifugation is also commonly referred to as packed cell volume (PCV). In normal blood, about 45 percent of a sample is erythrocytes. The hematocrit of any one sample can vary significantly, however, about 36–50 percent, according to gender and other factors. Normal hematocrit values for females range from 37 to 47, with a mean value of 41; for males, hematocrit ranges from 42 to 52, with a mean of 47. The percentage of other formed elements, the WBCs and platelets, is extremely small so it is not normally considered with the hematocrit. So the mean plasma percentage is the percent of blood that is not erythrocytes: for females, it is approximately 59 (or 100 minus 41), and for males, it is approximately 53 (or 100 minus 47).

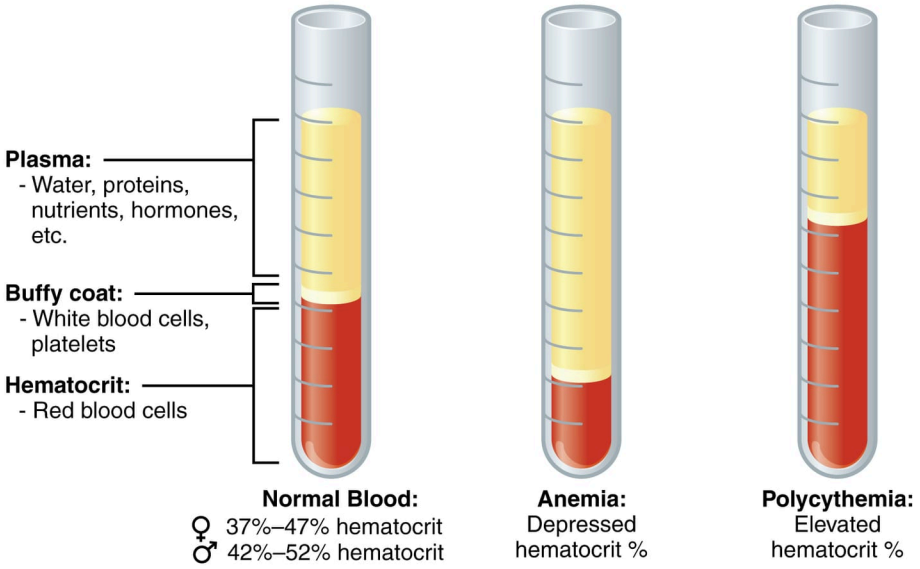


Figure 5.1.1: The cellular elements of blood include a vast number of erythrocytes and comparatively fewer leukocytes and platelets. Plasma is the fluid in which the formed elements are suspended. A sample of blood spun in a centrifuge reveals that plasma is the lightest component. It floats at the top of the tube separated from the heaviest elements, the erythrocytes, by a buffy coat of leukocytes and platelets. Hematocrit is the percentage of the total sample that is comprised of erythrocytes. Depressed and elevated hematocrit levels are shown for comparison. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.1.1.

Characteristics of Blood

When you think about blood, the first characteristic that probably comes to mind is its color. Blood that has just taken up oxygen in the lungs is bright red, and blood that has released oxygen in the tissues is a more dusky red. This is because hemoglobin is a pigment that changes color, depending upon the degree of oxygen saturation.

Blood is viscous and somewhat sticky to the touch. It has a viscosity approximately five times greater than water. Viscosity is a measure of a fluid's thickness or resistance to flow, and is influenced by the presence of the plasma proteins and formed elements within the blood. The viscosity of blood has a dramatic impact on blood pressure and flow. Consider the difference in flow between water and honey. The more viscous honey would demonstrate a greater resistance to flow than the less viscous water. The same principle applies to blood.

The normal temperature of blood is slightly higher than normal body temperature—about 38 °C (or 100.4 °F), compared to 37 °C (or 98.6 °F) for an internal body temperature reading, although daily variations of 0.5 °C are normal. Although the surface of blood vessels is relatively smooth, as blood flows through them, it experiences some friction and resistance, especially as vessels age and lose their elasticity, thereby producing heat. This accounts for its slightly higher temperature.

The pH of blood averages about 7.4; however, it can range from 7.35 to 7.45 in a healthy person. Blood is therefore somewhat more basic (alkaline) on a chemical scale than pure water, which has a pH of 7.0. Blood contains numerous buffers that actually help to regulate pH.

Blood constitutes approximately 8 percent of adult body weight. Adult males typically average about 5 to 6 liters of blood. Females average 4–5 liters.

Blood Plasma

Like other fluids in the body, plasma is composed primarily of water: In fact, it is about 92 percent water. Dissolved or suspended within this water is a mixture of substances, most of which are proteins. There are literally hundreds of substances dissolved or suspended in the plasma, although many of them are found only in very small quantities.

Plasma Proteins

About 7 percent of the volume of plasma—nearly all that is not water—is made of proteins. These include several plasma proteins (proteins that are unique to the plasma), plus a much smaller number of regulatory proteins, including enzymes and some hormones. The major components of plasma are summarized in Figure 5.1.1.

The three major groups of plasma proteins are as follows:

- Albumin is the most abundant of the plasma proteins. Manufactured by the liver, albumin molecules serve as binding proteins—transport vehicles for fatty acids and steroid hormones. Recall that lipids are hydrophobic; however, their binding to albumin enables their transport in the watery plasma. Albumin is also the most significant contributor to the osmotic pressure of blood; that is, its presence holds water inside the blood vessels and draws water from the tissues, across blood vessel walls, and into the bloodstream. This in turn helps to maintain both blood volume and blood pressure. Albumin normally accounts for approximately 54 percent of the total plasma protein content, in clinical levels of 3.5–5.0 g/dL blood.
- The second most common plasma proteins are the globulins. A heterogeneous group, there are three main subgroups known as alpha, beta, and gamma globulins. The alpha and beta globulins transport iron, lipids, and the fat-soluble vitamins A, D, E, and K to the cells; like albumin, they also contribute to osmotic pressure. The gamma globulins are proteins involved in immunity and are better known as an antibodies or immunoglobulins. Although other plasma proteins are produced by the liver, immunoglobulins are produced by specialized leukocytes known as plasma cells. (Seek additional content for more information about immunoglobulins.) Globulins make up approximately 38 percent of the total plasma protein volume, in clinical levels of 1.0–1.5 g/dL blood.
- Fibrinogen is the third of the three major groups of plasma proteins. Like albumin and the alpha and beta globulins, fibrinogen is produced by the liver. It is essential for blood clotting, a process described later in this chapter. Fibrinogen accounts for about 7 percent of the total plasma protein volume, in clinical levels of 0.2–0.45 g/dL blood.

Other Plasma Solutes

In addition to proteins, plasma contains a wide variety of other substances. These include various electrolytes, such as sodium, potassium, and calcium ions; dissolved gases, such as oxygen, carbon dioxide, and nitrogen; various organic nutrients, such as vitamins, lipids, glucose, and amino acids; and metabolic wastes. All of these nonprotein solutes combined contribute approximately 1 percent to the total volume of plasma.

Table 5.1.1: Components of blood and their major functions

Blood Component (% of Blood)	Subcomponent (% of Component)	Type / Percentage	Site of Production	Major Function(s)
Plasma (46–63%)	Water (92%)	Fluid	Absorbed by intestinal tract or produced by metabolism	Transport medium
Plasma (46–63%)	Plasma proteins (7%)	Albumin (54–60%)	Liver	Maintains osmotic concentration; transports lipid molecules
Plasma (46–63%)	Plasma proteins (7%)	Alpha globulins	Liver	Transport; maintain osmotic concentration

Blood Component (% of Blood)	Subcomponent (% of Component)	Type / Percentage	Site of Production	Major Function(s)
Plasma (46–63%)	Plasma proteins (7%)	Beta globulins	Liver	Transport; maintain osmotic concentration
Plasma (46–63%)	Plasma proteins (7%)	Gamma globulins (immunoglobulins)	Plasma cells	Immune responses
Plasma (46–63%)	Plasma proteins (7%)	Fibrinogen (4–7%)	Liver	Blood clotting (hemostasis)
Plasma (46–63%)	Regulatory proteins (<1%)	Hormones and enzymes	Various sources	Regulate body functions
Plasma (46–63%)	Other solutes (1%)	Nutrients, gases, and wastes	Absorbed by the intestinal tract, exchanged in the respiratory system, or produced by cells	Numerous and varied
Formed Elements (37–54%)	Erythrocytes (99%)	Erythrocytes	Red bone marrow	Transport oxygen and some carbon dioxide
Formed Elements (37–54%)	Leukocytes (<1%)	Granular leukocytes: neutrophils, eosinophils, basophils	Red bone marrow	Nonspecific immunity
Formed Elements (37–54%)	Leukocytes (<1%)	Lymphocytes	Bone marrow and lymphatic tissue	Specific immunity
Formed Elements (37–54%)	Leukocytes (<1%)	Monocytes	Red bone marrow	Nonspecific immunity
Formed Elements (37–54%)	Platelets (<1%)	Platelets (derived from megakaryocytes)	Red bone marrow	Hemostasis

Career Connection

Phlebotomy and Medical Lab Technology

Phlebotomists are professionals trained to draw blood (phleb- = “a blood vessel”; -tomy = “to cut”). When more than a few drops of blood are required, phlebotomists perform a venipuncture, typically of a surface vein in the arm. They perform a capillary stick on a finger, an earlobe, or the heel of an infant when only a small quantity of blood is required. An arterial stick is collected from an artery and used to analyze blood gases. After collection, the blood may be analyzed by medical laboratories or perhaps used for transfusions, donations, or research. While many allied health professionals practice phlebotomy, the American Society of Phlebotomy Technicians issues certificates to individuals passing a national examination, and some large labs and hospitals hire individuals expressly for their skill in phlebotomy.

Medical or clinical laboratories employ a variety of individuals in technical positions:

- Medical technologists (MT), also known as clinical laboratory technologists (CLT), typically hold a bachelor’s degree and certification from an accredited training program. They perform a wide variety of tests on various body fluids, including blood. The information they provide is essential to the primary care providers in determining a diagnosis and in monitoring the course of a disease and response to treatment.
- Medical laboratory technicians (MLT) typically have an associate’s degree but may perform duties similar to those of an MT.
- Medical laboratory assistants (MLA) spend the majority of their time processing samples and carrying out routine assignments within the lab. Clinical training is required, but a degree may not be essential to obtaining a position.

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5.2: Production of the Formed Elements

Hemopoiesis: Blood Cell Formation

The lifespan of the formed elements is very brief. Although one type of leukocyte called memory cells can survive for years, most erythrocytes, leukocytes, and platelets normally live only a few hours to a few weeks. Thus, the body must form new blood cells and platelets quickly and continuously. When you donate a unit of blood during a blood drive (approximately 475 mL, or about 1 pint), your body typically replaces the donated plasma within 24 hours, but it takes about 4 to 6 weeks to replace the blood cells. This restricts the frequency with which donors can contribute their blood. The process by which this replacement occurs is called hemopoiesis, or hematopoiesis (from the Greek root haima- = “blood”; -poiesis = “production”).

Sites of Hemopoiesis

Prior to birth, hemopoiesis occurs in a number of tissues, beginning with the yolk sac of the developing embryo, and continuing in the fetal liver, spleen, lymphatic tissue, and eventually the red bone marrow. Following birth, most hemopoiesis occurs in the red marrow, a connective tissue within the spaces of spongy (cancellous) bone tissue. In children, hemopoiesis can occur in the medullary cavity of long bones; in adults, the process is largely restricted to the cranial and pelvic bones, the vertebrae, the sternum, and the proximal epiphyses of the femur and humerus.

Throughout adulthood, the liver and spleen maintain their ability to generate the formed elements. This process is referred to as extramedullary hemopoiesis (meaning hemopoiesis outside the medullary cavity of adult bones). When a disease such as bone cancer destroys the bone marrow, causing hemopoiesis to fail, extramedullary hemopoiesis may be initiated.

Differentiation of Formed Elements from Stem Cells

All formed elements arise from stem cells of the red bone marrow. Recall that stem cells undergo mitosis plus cytokinesis (cellular division) to give rise to new daughter cells: One of these remains a stem cell and the other differentiates into one of any number of diverse cell types. Stem cells may be viewed as occupying a hierarchal system, with some loss of the ability to diversify at each step. The totipotent stem cell is the zygote, or fertilized egg. The totipotent (toti- = “all”) stem cell gives rise to all cells of the human body. The next level is the pluripotent stem cell, which gives rise to multiple types of cells of the body and some of the supporting fetal membranes. Beneath this level, the mesenchymal cell is a stem cell that develops only into types of connective tissue, including fibrous connective tissue, bone, cartilage, and blood, but not epithelium, muscle, and nervous tissue. One step lower on the hierarchy of stem cells is the hematopoietic stem cell, or hemocytoblast. All of the formed elements of blood originate from this specific type of cell.

Hemopoiesis begins when the hematopoietic stem cell is exposed to appropriate chemical stimuli collectively called hemopoietic growth factors, which prompt it to divide and differentiate. One daughter cell remains a hematopoietic stem cell, allowing hemopoiesis to continue. The other daughter cell becomes either of two types of more specialized stem cells (Figure $5.2.1$):

- Lymphoid stem cells give rise to a class of leukocytes known as lymphocytes, which include the various T cells, B cells, and natural killer (NK) cells, all of which function in immunity. However, hemopoiesis of lymphocytes progresses somewhat differently from the process for the other formed elements. In brief, lymphoid stem cells quickly migrate from the bone marrow to lymphatic tissues, including the lymph nodes, spleen, and thymus, where their production and differentiation continues. B cells are so named since they mature in the bone marrow, while T cells mature in the thymus.
- Myeloid stem cells give rise to all the other formed elements, including the erythrocytes; megakaryocytes that produce platelets; and a myeloblast lineage that gives rise to monocytes and three forms of granular leukocytes: neutrophils, eosinophils, and basophils.

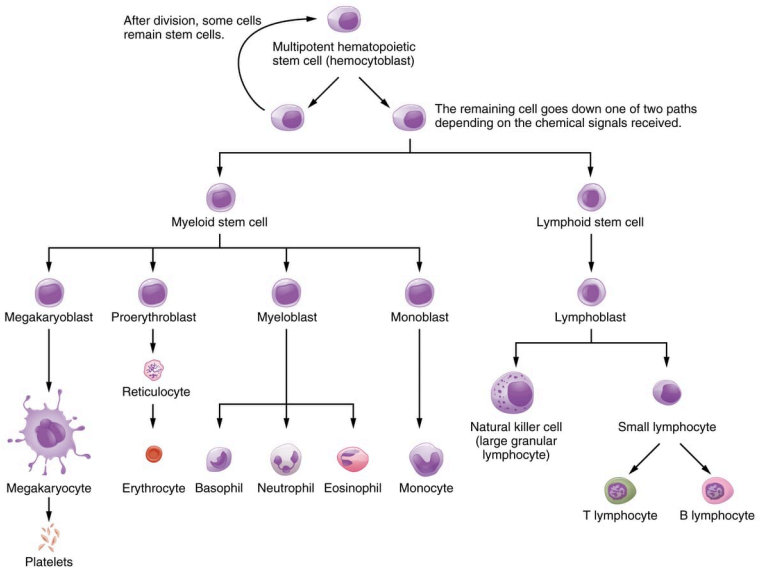


Figure 5.2.1: Hemopoiesis is the proliferation and differentiation of the formed elements of blood. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.2.1.

Lymphoid and myeloid stem cells do not immediately divide and differentiate into mature formed elements. As you can see in Figure 5.2.1, there are several intermediate stages of precursor cells (literally, forerunner cells), many of which can be recognized by their names, which have the suffix -blast. For instance, megakaryoblasts are the precursors of megakaryocytes, and proerythroblasts become reticulocytes, which eject their nucleus and most other organelles before maturing into erythrocytes.

Hemopoietic Growth Factors

Development from stem cells to precursor cells to mature cells is again initiated by hemopoietic growth factors. These include the following:

- Erythropoietin (EPO) is a glycoprotein hormone secreted by the interstitial fibroblast cells of the kidneys in response to low oxygen levels. It prompts the production of erythrocytes. Some athletes use synthetic EPO as a performance-enhancing drug (called blood doping) to increase RBC counts and subsequently increase oxygen delivery to tissues throughout the body. EPO is a banned substance in most organized sports, but it is also used medically in the treatment of certain anemia, specifically those triggered by certain types of cancer, and other disorders in which increased erythrocyte counts and oxygen levels are desirable.
- Thrombopoietin, another glycoprotein hormone, is produced by the liver and kidneys. It triggers the development of megakaryocytes into platelets.
- Cytokines are glycoproteins secreted by a wide variety of cells, including red bone marrow, leukocytes, macrophages, fibroblasts, and endothelial cells. They act locally as autocrine or paracrine factors, stimulating the proliferation of progenitor cells and helping to stimulate both nonspecific and specific resistance to disease. There are two major subtypes of cytokines known as colony-stimulating factors and interleukins.
    - Colony-stimulating factors (CSFs) are glycoproteins that act locally, as autocrine or paracrine factors. Some trigger the differentiation of myeloblasts into granular leukocytes, namely, neutrophils, eosinophils, and basophils. These are referred to as granulocyte CSFs. A different CSF induces the production of monocytes, called monocyte CSFs. Both granulocytes and monocytes are stimulated by GM-CSF; granulocytes, monocytes, platelets, and erythrocytes are stimulated by multi-CSF. Synthetic forms of these hormones are often administered to patients with various forms of cancer who are receiving chemotherapy to revive their WBC counts.
    - Interleukins are another class of cytokine signaling molecules important in hemopoiesis. They were initially thought to be secreted uniquely by leukocytes and to communicate only with other leukocytes, and were named accordingly, but are now known to be produced by a variety of cells including bone marrow and endothelium. Researchers now suspect that interleukins may play other roles in body functioning, including differentiation and maturation of cells, producing immunity and inflammation. To date, more than a dozen interleukins have been identified, with others likely to follow. They are generally numbered IL-1, IL-2, IL-3, etc.

Everyday Connection

Blood Doping

In its original intent, the term blood doping was used to describe the practice of injecting by transfusion supplemental RBCs into an individual, typically to enhance performance in a sport. Additional RBCs would deliver more oxygen to the tissues, providing extra aerobic capacity, clinically referred to as VO_2 max. The source of the cells was either from the recipient (autologous) or from a donor with compatible blood (homologous). This practice was aided by the well-developed techniques of harvesting, concentrating, and freezing of the RBCs that could be later thawed and injected, yet still retain their functionality. These practices are considered illegal in virtually all sports and run the risk of infection, significantly increasing the viscosity of the blood and the potential for transmission of blood-borne pathogens if the blood was collected from another individual.

With the development of synthetic EPO in the 1980s, it became possible to provide additional RBCs by artificially stimulating RBC production in the bone marrow. Originally developed to treat patients suffering from anemia, renal failure, or cancer treatment, large quantities of EPO can be generated by recombinant DNA technology. Synthetic EPO is injected under the skin and can increase hematocrit for many weeks. It may also induce polycythemia and raise hematocrit to 70 or greater. This increased viscosity raises the resistance of the blood and forces the heart to pump more powerfully; in extreme cases, it has resulted in death. Other drugs such as cobalt II chloride have been shown to increase natural EPO gene expression. Blood doping has become problematic in many sports, especially cycling. Lance Armstrong, winner of seven Tour de France and many other cycling titles, was stripped of his victories and admitted to blood doping in 2013.

Bone Marrow Sampling and Transplants

Sometimes, a healthcare provider will order a bone marrow biopsy, a diagnostic test of a sample of red bone marrow, or a bone marrow transplant, a treatment in which a donor's healthy bone marrow—and its stem cells—replaces the faulty bone marrow of a patient. These tests and procedures are often used to assist in the diagnosis and treatment of various severe forms of anemia, such as thalassemia major and sickle cell anemia, as well as some types of cancer, specifically leukemia.

In the past, when a bone marrow sample or transplant was necessary, the procedure would have required inserting a large-bore needle into the region near the iliac crest of the pelvic bones (os coxae). This location was preferred, since its location close to the body surface makes it more accessible, and it is relatively isolated from most vital organs. Unfortunately, the procedure is quite painful.

Now, direct sampling of bone marrow can often be avoided. In many cases, stem cells can be isolated in just a few hours from a sample of a patient's blood. The isolated stem cells are then grown in culture using the appropriate hemopoietic growth factors, and analyzed or sometimes frozen for later use.

For an individual requiring a transplant, a matching donor is essential to prevent the immune system from destroying the donor cells—a phenomenon known as tissue rejection. To treat patients with bone marrow transplants, it is first necessary to destroy the patient's own diseased marrow through radiation and/or chemotherapy. Donor bone marrow stem cells are then intravenously infused. From the bloodstream, they establish themselves in the recipient's bone marrow.

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5.3: Erythrocytes

Structure and Function of Erythrocytes

The erythrocyte, commonly known as a red blood cell (or RBC), is by far the most common formed element: A single drop of blood contains millions of erythrocytes and just thousands of leukocytes. Specifically, males have about 5.4 million erythrocytes per microliter (μL) of blood, and females have approximately 4.8 million per μL. In fact, erythrocytes are estimated to make up about 25 percent of the total cells in the body. As you can imagine, they are quite small cells, with a mean diameter of only about 7–8 micrometers (μm) (Table 5.3.1). The primary functions of erythrocytes are to pick up inhaled oxygen from the lungs and transport it to the body's tissues, and to pick up some (about 24 percent) carbon dioxide waste at the tissues and transport it to the lungs for exhalation. Erythrocytes remain within the vascular network. Although leukocytes typically leave the blood vessels to perform their defensive functions, movement of erythrocytes from the blood vessels is abnormal.

Table 5.3.1: Characteristics of the formed elements of blood.

Formed Element	Subtype	Average Count per µL (Range)	Appearance	Function	Comments
Erythrocytes (red blood cells)	No data available	5.2 million (4.4–6.0 million)	Flattened biconcave disc; no nucleus; pale red color	Transport oxygen and some carbon dioxide between tissues and lungs	Lifespan approximately 120 days
Leukocytes (white blood cells)	All leukocytes	7,000 (5,000–10,000)	Prominent dark-staining nucleus	Body defense and immunity	Exit capillaries and move into tissues; lifespan usually a few hours to days
Leukocytes	Granulocytes (overall)	4,360 (1,800–9,950)	Abundant cytoplasmic granules; lobed nucleus	Nonspecific (innate) immunity	Classified by membrane-bound granules in cytoplasm
Leukocytes	Neutrophils	4,150 (1,800–7,300)	Multilobed nucleus; pale lilac granules	Phagocytosis; especially effective against bacteria	Most common leukocyte; lifespan minutes to days
Leukocytes	Eosinophils	165 (0–700)	Two-lobed nucleus; bright red-orange granules	Defense against parasites; active in allergies; release antihistamines	Lifespan minutes to days
Leukocytes	Basophils	44 (0–150)	Dense dark purple granules often obscure nucleus	Promote inflammation	Least common leukocyte; lifespan unknown
Leukocytes	Agranulocytes (overall)	2,640 (1,700–4,950)	Few visible granules; simple or indented nucleus	Body defense	Includes lymphocytes and monocytes
Leukocytes	Lymphocytes	2,185 (1,500–4,000)	Large nucleus with little cytoplasm	Specific (adaptive) immunity; includes B cells, T cells, and natural killer cells	Memory cells may persist for years
Leukocytes	Monocytes	455 (200–950)	Largest leukocyte; indented or horseshoe-shaped nucleus	Phagocytosis and antigen presentation	Become macrophages after leaving circulation

Formed Element	Subtype	Average Count per µL (Range)	Appearance	Function	Comments
Platelets	No data available	350,000 (150,000–500,000)	Cell fragments surrounded by a plasma membrane and containing granules	Hemostasis and release of growth factors for tissue repair	Derived from megakaryocytes in red bone marrow

Shape and Structure of Erythrocytes

As an erythrocyte matures in the red bone marrow, it extrudes its nucleus and most of its other organelles. During the first day or two that it is in the circulation, an immature erythrocyte, known as a reticulocyte, will still typically contain remnants of organelles. Reticulocytes should comprise approximately 1–2 percent of the erythrocyte count and provide a rough estimate of the rate of RBC production, with abnormally low or high rates indicating deviations in the production of these cells. These remnants, primarily of networks (reticulum) of ribosomes, are quickly shed, however, and mature, circulating erythrocytes have few internal cellular structural components. Lacking mitochondria, for example, they rely on anaerobic respiration. This means that they do not utilize any of the oxygen they are transporting, so they can deliver it all to the tissues. They also lack endoplasmic reticula and do not synthesize proteins. Erythrocytes do, however, contain some structural proteins that help the blood cells maintain their unique structure and enable them to change their shape to squeeze through capillaries. This includes the protein spectrin, a cytoskeletal protein element.

Erythrocytes are biconcave disks; that is, they are plump at their periphery and very thin in the center (Figure 5.3.1). Since they lack most organelles, there is more interior space for the presence of the hemoglobin molecules that, as you will see shortly, transport gases. The biconcave shape also provides a greater surface area across which gas exchange can occur, relative to its volume; a sphere of a similar diameter would have a lower surface area-to-volume ratio. In the capillaries, the oxygen carried by the erythrocytes can diffuse into the plasma and then through the capillary walls to reach the cells, whereas some of the carbon dioxide produced by the cells as a waste product diffuses into the capillaries to be picked up by the erythrocytes. Capillary beds are extremely narrow, slowing the passage of the erythrocytes and providing an extended opportunity for gas exchange to occur. However, the space within capillaries can be so minute that, despite their own small size, erythrocytes may have to fold in on themselves if they are to make their way through. Fortunately, their structural proteins like spectrin are flexible, allowing them to bend over themselves to a surprising degree, then spring back again when they enter a wider vessel. In wider vessels, erythrocytes may stack up much like a roll of coins, forming a rouleaux, from the French word for “roll.”

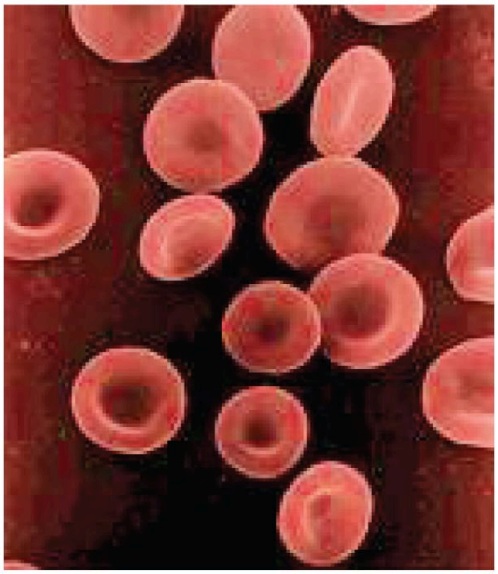


Figure 5.3.1: Erythrocytes are biconcave discs with very shallow centers. This shape optimizes the ratio of surface area to volume, facilitating gas exchange. It also enables them to fold up as they move through narrow blood vessels. (CC BY 4.0; via LibreTexts).

Hemoglobin

Hemoglobin is a large molecule made up of proteins and iron. It consists of four folded chains of a protein called globin, designated alpha 1 and 2, and beta 1 and 2 (Figure 5.3.2*a*). Each of these globin molecules is bound to a red pigment molecule called heme, which contains an ion of iron (Fe^{2+}) (Figure 5.3.2*b*).

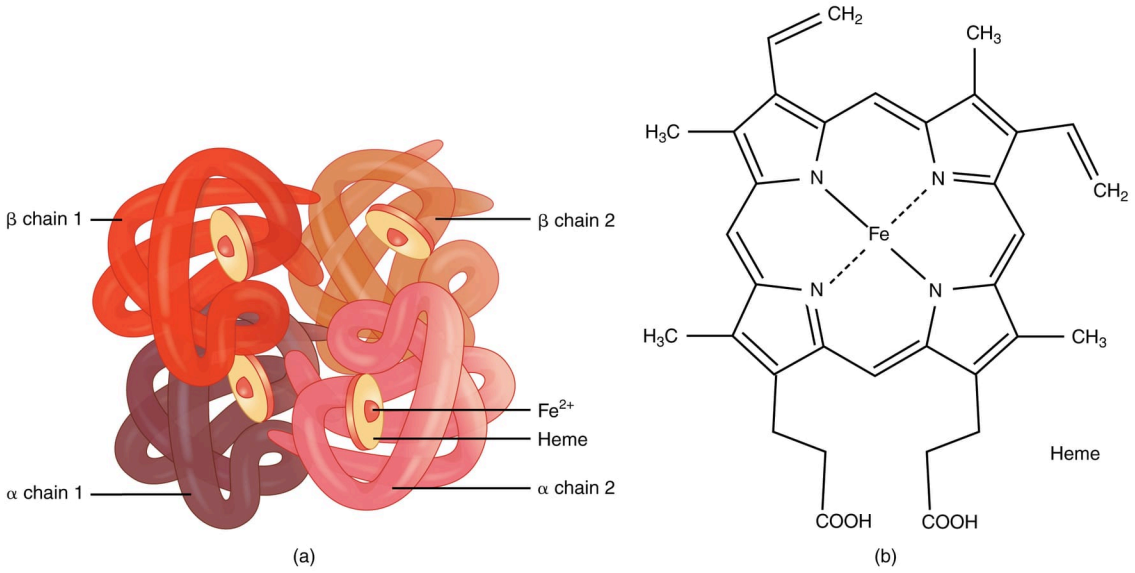


Figure 5.3.2: Hemoglobin (a) A molecule of hemoglobin contains four globin proteins, each of which is bound to one molecule of the iron-containing pigment heme. (b) A single erythrocyte can contain 300 million hemoglobin molecules, and thus more than 1 billion oxygen molecules. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.3.2.

Each iron ion in the heme can bind to one oxygen molecule; therefore, each hemoglobin molecule can transport four oxygen molecules. An individual erythrocyte may contain about 300 million hemoglobin molecules, and therefore can bind to and transport up to 1.2 billion oxygen molecules.

In the lungs, hemoglobin picks up oxygen, which binds to the iron ions, forming oxyhemoglobin. The bright red, oxygenated hemoglobin travels to the body tissues, where it releases some of the oxygen molecules, becoming darker red deoxyhemoglobin, sometimes referred to as reduced hemoglobin. Oxygen release depends on the need for oxygen in the surrounding tissues, so hemoglobin rarely if ever leaves all of its oxygen behind. In the capillaries, carbon dioxide enters the bloodstream. About 76 percent dissolves in the plasma, some of it remaining as dissolved CO_2, and the remainder forming bicarbonate ion. About 23–24 percent of it binds to the amino acids in hemoglobin, forming a molecule known as carbaminohemoglobin. From the capillaries, the hemoglobin carries carbon dioxide back to the lungs, where it releases it for exchange of oxygen.

Changes in the levels of RBCs can have significant effects on the body's ability to effectively deliver oxygen to the tissues. Ineffective hematopoiesis results in insufficient numbers of RBCs and results in one of several forms of anemia. An overproduction of RBCs produces a condition called polycythemia. The primary drawback with polycythemia is not a failure to directly deliver enough oxygen to the tissues, but rather the increased viscosity of the blood, which makes it more difficult for the heart to circulate the blood.

In patients with insufficient hemoglobin, the tissues may not receive sufficient oxygen, resulting in another form of anemia. In determining oxygenation of tissues, the value of greatest interest in healthcare is the percent saturation; that is, the percentage of hemoglobin sites occupied by oxygen in a patient's blood. Clinically this value is commonly referred to simply as "percent sat."

Percent saturation is normally monitored using a device known as a pulse oximeter, which is applied to a thin part of the body, typically the tip of the patient's finger. The device works by sending two different wavelengths of light (one red, the other infrared) through the finger and measuring the light with a photodetector as it exits. Hemoglobin absorbs light differentially depending upon its saturation with oxygen. The machine calibrates the amount of light received by the photodetector against the amount absorbed by the partially oxygenated hemoglobin and presents the data as percent saturation. Normal pulse oximeter readings range from 95–100 percent. Lower percentages reflect hypoxemia, or low blood oxygen. The term hypoxia is more generic and simply refers to low oxygen levels. Oxygen levels are also directly monitored from free oxygen in the plasma typically following an

arterial stick. When this method is applied, the amount of oxygen present is expressed in terms of partial pressure of oxygen or simply P_{O2} or PO2 and is typically recorded in units of millimeters of mercury, mm Hg.

The kidneys filter about 180 liters (~380 pints) of blood in an average adult each day, or about 20 percent of the total resting volume, and thus serve as ideal sites for receptors that determine oxygen saturation. In response to hypoxemia, less oxygen will exit the vessels supplying the kidney, resulting in hypoxia (low oxygen concentration) in the tissue fluid of the kidney where oxygen concentration is actually monitored. Interstitial fibroblasts within the kidney secrete EPO, thereby increasing erythrocyte production and restoring oxygen levels. In a classic negative-feedback loop, as oxygen saturation rises, EPO secretion falls, and vice versa, thereby maintaining homeostasis. Populations dwelling at high elevations, with inherently lower levels of oxygen in the atmosphere, naturally maintain a hematocrit higher than people living at sea level. Consequently, people traveling to high elevations may experience symptoms of hypoxemia, such as fatigue, headache, and shortness of breath, for a few days after their arrival. In response to the hypoxemia, the kidneys secrete EPO to step up the production of erythrocytes until homeostasis is achieved once again. To avoid the symptoms of hypoxemia, or altitude sickness, mountain climbers typically rest for several days to a week or more at a series of camps situated at increasing elevations to allow EPO levels and, consequently, erythrocyte counts to rise. When climbing the tallest peaks, such as Mt. Everest and K2 in the Himalayas, many mountain climbers rely upon bottled oxygen as they near the summit.

Lifecycle of Erythrocytes

Production of erythrocytes in the marrow occurs at the staggering rate of more than 2 million cells per second. For this production to occur, a number of raw materials must be present in adequate amounts. These include the same nutrients that are essential to the production and maintenance of any cell, such as glucose, lipids, and amino acids. However, erythrocyte production also requires several trace elements:

- Iron. We have said that each heme group in a hemoglobin molecule contains an ion of the trace mineral iron. On average, less than 20 percent of the iron we consume is absorbed. Heme iron, from animal foods such as meat, poultry, and fish, is absorbed more efficiently than non-heme iron from plant foods. Upon absorption, iron becomes part of the body's total iron pool. The bone marrow, liver, and spleen can store iron in the protein compounds ferritin and hemosiderin. Ferroportin transports the iron across the intestinal cell plasma membranes and from its storage sites into tissue fluid where it enters the blood. When EPO stimulates the production of erythrocytes, iron is released from storage, bound to transferrin, and carried to the red marrow where it attaches to erythrocyte precursors.
- Copper. A trace mineral, copper is a component of two plasma proteins, hephaestin and ceruloplasmin. Without these, hemoglobin could not be adequately produced. Located in intestinal villi, hephaestin enables iron to be absorbed by intestinal cells. Ceruloplasmin transports copper. Both enable the oxidation of iron from Fe^{2+} to Fe^{3+}, a form in which it can be bound to its transport protein, transferrin, for transport to body cells. In a state of copper deficiency, the transport of iron for heme synthesis decreases, and iron can accumulate in tissues, where it can eventually lead to organ damage.
- Zinc. The trace mineral zinc functions as a co-enzyme that facilitates the synthesis of the heme portion of hemoglobin.
- Erythrocytes live up to 120 days in the circulation, after which the worn-out cells are removed by a type of myeloid phagocytic cell called a macrophage, located primarily within the bone marrow, liver, and spleen. The components of the degraded erythrocytes' hemoglobin are further processed as follows:
  - Globin, the protein portion of hemoglobin, is broken down into amino acids, which can be sent back to the bone marrow to be used in the production of new erythrocytes. Hemoglobin that is not phagocytized is broken down in the circulation, releasing alpha and beta chains that are removed from circulation by the kidneys.
  - The iron contained in the heme portion of hemoglobin may be stored in the liver or spleen, primarily in the form of ferritin or hemosiderin, or carried through the bloodstream by transferrin to the red bone marrow for recycling into new erythrocytes.
- The breakdown pigments formed from the destruction of hemoglobin can be seen in a variety of situations. At the site of an injury, biliverdin from damaged RBCs produces some of the dramatic colors associated with a bruise. With a failing liver, bilirubin cannot be removed effectively from circulation and causes the body to assume a yellowish tinge associated with jaundice. Stercobilins within the feces produce the typical brown color associated with this waste. And the yellow of urine is associated with the urobilins.

  The erythrocyte lifecycle is summarized in Figure 5.3.3.

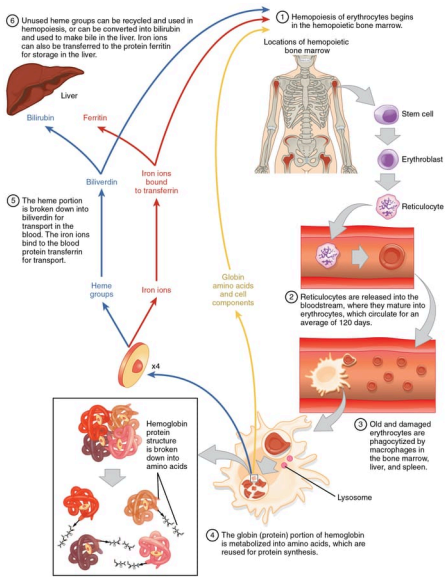


Figure 5.3.3: Erythrocytes are produced in the bone marrow and sent into the circulation. At the end of their lifecycle, they are destroyed by macrophages, and their components are recycled. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.3.3.

Disorders of Erythrocytes

The size, shape, and number of erythrocytes, and the number of hemoglobin molecules can have a major impact on a person's health. When the number of RBCs or hemoglobin is deficient, the general condition is called anemia. There are more than 400 types of anemia and more than 3.5 million Americans suffer from this condition. Anemia can be broken down into three major groups: those caused by blood loss, those caused by faulty or decreased RBC production, and those caused by excessive destruction of RBCs. Clinicians often use two groupings in diagnosis: The kinetic approach focuses on evaluating the production, destruction, and removal of RBCs, whereas the morphological approach examines the RBCs themselves, paying particular emphasis to their size. A common test is the mean corpuscle volume (MCV), which measures size. Normal-sized cells are referred to as normocytic, smaller-than-normal cells are referred to as microcytic, and larger-than-normal cells are referred to as macrocytic. Reticulocyte counts are also important and may reveal inadequate production of RBCs. The effects of the various anemias are widespread, because reduced numbers of RBCs or hemoglobin will result in lower levels of oxygen being delivered to body tissues. Since oxygen is required for tissue functioning, anemia produces fatigue, lethargy, and an increased risk for infection. An oxygen deficit in the brain impairs the ability to think clearly, and may prompt headaches and irritability. Lack of oxygen leaves the patient short of breath, even as the heart and lungs work harder in response to the deficit.

Blood loss anemias are fairly straightforward. In addition to bleeding from wounds or other lesions, these forms of anemia may be due to ulcers, hemorrhoids, inflammation of the stomach (gastritis), and some cancers of the gastrointestinal tract. The excessive use of aspirin or other nonsteroidal anti-inflammatory drugs such as ibuprofen can trigger ulceration and gastritis. Excessive menstruation and loss of blood during childbirth are also potential causes.

Anemias caused by faulty or decreased RBC production include sickle cell anemia, iron deficiency anemia, vitamin deficiency anemia, and diseases of the bone marrow and stem cells.



Figure 5.3.4: Sickle cell anemia is caused by a mutation in one of the hemoglobin genes. Erythrocytes produce an abnormal type of hemoglobin, which causes the cell to take on a sickle or crescent shape. (CC BY 4.0; via LibreTexts).

In contrast to anemia, an elevated RBC count is called polycythemia and is detected in a patient's elevated hematocrit. It can occur transiently in a person who is dehydrated; when water intake is inadequate or water losses are excessive, the plasma volume falls. As a result, the hematocrit rises. For reasons mentioned earlier, a mild form of polycythemia is chronic but normal in people living at high altitudes. Some elite athletes train at high elevations specifically to induce this phenomenon. Finally, a type of bone marrow disease called polycythemia vera (from the Greek vera = "true") causes an excessive production of immature erythrocytes. Polycythemia vera can dangerously elevate the viscosity of blood, raising blood pressure and making it more difficult for the heart to pump blood throughout the body. It is a relatively rare disease that occurs more often in males than in females, and is more likely to be present in elderly patients those over 60 years of age.

- A characteristic change in the shape of erythrocytes is seen in sickle cell disease (also referred to as sickle cell anemia). A genetic disorder, it is caused by production of an abnormal type of hemoglobin, called hemoglobin S, which delivers less oxygen to tissues and causes erythrocytes to assume a sickle (or crescent) shape, especially at low oxygen concentrations (Figure 5.3.4). These abnormally shaped cells can then become lodged in narrow capillaries because they are unable to fold in on themselves to squeeze through, blocking blood flow to tissues and causing a variety of serious problems from painful joints to delayed growth and even blindness and cerebrovascular accidents (strokes). Sickle cell anemia is a genetic condition particularly found in individuals of African descent.
- Iron deficiency anemia is the most common type and results when the amount of available iron is insufficient to allow production of sufficient heme. This condition can occur in individuals with a deficiency of iron in the diet and is especially common in teens and children as well as in vegans and vegetarians. Additionally, iron deficiency anemia may be caused by either an inability to absorb and transport iron or slow, chronic bleeding.
- Vitamin-deficient anemias generally involve insufficient vitamin B12 and folate.
  - Megaloblastic anemia involves a deficiency of vitamin B12 and/or folate, and often involves diets deficient in these essential nutrients. Lack of meat or a viable alternate source, and overcooking or eating insufficient amounts of vegetables may lead to a lack of folate.
  - Pernicious anemia is caused by poor absorption of vitamin B12 and is often seen in patients with Crohn's disease (a severe intestinal disorder often treated by surgery), surgical removal of the intestines or stomach (common in some weight loss surgeries), intestinal parasites, and AIDS.
  - Pregnancies, some medications, excessive alcohol consumption, and some diseases such as celiac disease are also associated with vitamin deficiencies. It is essential to provide sufficient folic acid during the early stages of pregnancy to reduce the risk of neurological defects, including spina bifida, a failure of the neural tube to close.
- Assorted disease processes can also interfere with the production and formation of RBCs and hemoglobin. If myeloid stem cells are defective or replaced by cancer cells, there will be insufficient quantities of RBCs produced.
  - Aplastic anemia is the condition in which there are deficient numbers of RBC stem cells. Aplastic anemia is often inherited, or it may be triggered by radiation, medication, chemotherapy, or infection.
  - Thalassemia is an inherited condition typically occurring in individuals from the Middle East, the Mediterranean, African, and Southeast Asia, in which maturation of the RBCs does not proceed normally. The most severe form is

called Cooley's anemia.
    - Lead exposure from industrial sources or even dust from paint chips of iron-containing paints or pottery that has not been properly glazed may also lead to destruction of the red marrow.
- Various disease processes also can lead to anemias. These include chronic kidney diseases often associated with a decreased production of EPO, hypothyroidism, some forms of cancer, lupus, and rheumatoid arthritis.

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5.4: Leukocytes and Platelets

White Blood Cells and Platelets

The leukocyte, commonly known as a white blood cell (or WBC), is a major component of the body's defenses against disease. Leukocytes protect the body against invading microorganisms and body cells with mutated DNA, and they clean up debris. Platelets are essential for the repair of blood vessels when damage to them has occurred; they also provide growth factors for healing and repair.

Characteristics of Leukocytes

Although leukocytes and erythrocytes both originate from hematopoietic stem cells in the bone marrow, they are very different from each other in many significant ways. For instance, leukocytes are far less numerous than erythrocytes: Typically there are only 5000 to 10,000 per μL. They are also larger than erythrocytes and are the only formed elements that are complete cells, possessing a nucleus and organelles. And although there is just one type of erythrocyte, there are many types of leukocytes. Most of these types have a much shorter lifespan than that of erythrocytes, some as short as a few hours or even a few minutes in the case of acute infection.

One of the most distinctive characteristics of leukocytes is their movement. Whereas erythrocytes spend their days circulating within the blood vessels, leukocytes routinely leave the bloodstream to perform their defensive functions in the body's tissues. For leukocytes, the vascular network is simply a highway they travel and soon exit to reach their true destination. When they arrive, they are often given distinct names, such as macrophage or microglia, depending on their function. As shown in Figure 18.10, they leave the capillaries—the smallest blood vessels—or other small vessels through a process known as emigration (from the Latin for "removal") or diapedesis (dia- = "through"; -pedan = "to leap") in which they squeeze through adjacent cells in a blood vessel wall.

Once they have exited the capillaries, some leukocytes will take up fixed positions in lymphatic tissue, bone marrow, the spleen, the thymus, or other organs. Others will move about through the tissue spaces very much like amoebas, continuously extending their plasma membranes, sometimes wandering freely, and sometimes moving toward the direction in which they are drawn by chemical signals. This attracting of leukocytes occurs because of positive chemotaxis (literally "movement in response to chemicals"), a phenomenon in which injured or infected cells and nearby leukocytes emit the equivalent of a chemical "911" call, attracting more leukocytes to the site. In clinical medicine, the differential counts of the types and percentages of leukocytes present are often key indicators in making a diagnosis and selecting a treatment.

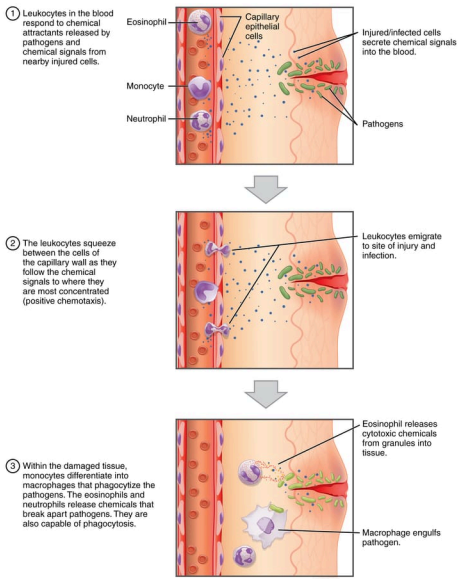


Figure 5.4.1: Leukocytes exit the blood vessel and then move through the connective tissue of the dermis toward the site of a wound. Some leukocytes, such as the eosinophil and neutrophil, are characterized as granular leukocytes. They release chemicals from their granules that destroy pathogens; they are also capable of phagocytosis. The monocyte, an agranular leukocyte, differentiates into a macrophage that then phagocytizes the pathogens. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.4.1.

Classification of Leukocytes

When scientists first began to observe stained blood slides, it quickly became evident that leukocytes could be divided into two groups, according to whether their cytoplasm contained highly visible granules:

- Granular leukocytes contain abundant granules within the cytoplasm. They include neutrophils, eosinophils, and basophils (you can view their lineage from myeloid stem cells in Figure 5.4.2).
- While granules are not totally lacking in agranular leukocytes, they are far fewer and less obvious. Agranular leukocytes include monocytes, which mature into macrophages that are phagocytic, and lymphocytes, which arise from the lymphoid stem cell line.

Granular Leukocytes

We will consider the granular leukocytes in order from most common to least common. All of these are produced in the red bone marrow and have a short lifespan of hours to days. They typically have a lobed nucleus and are classified according to which type of stain best highlights their granules.

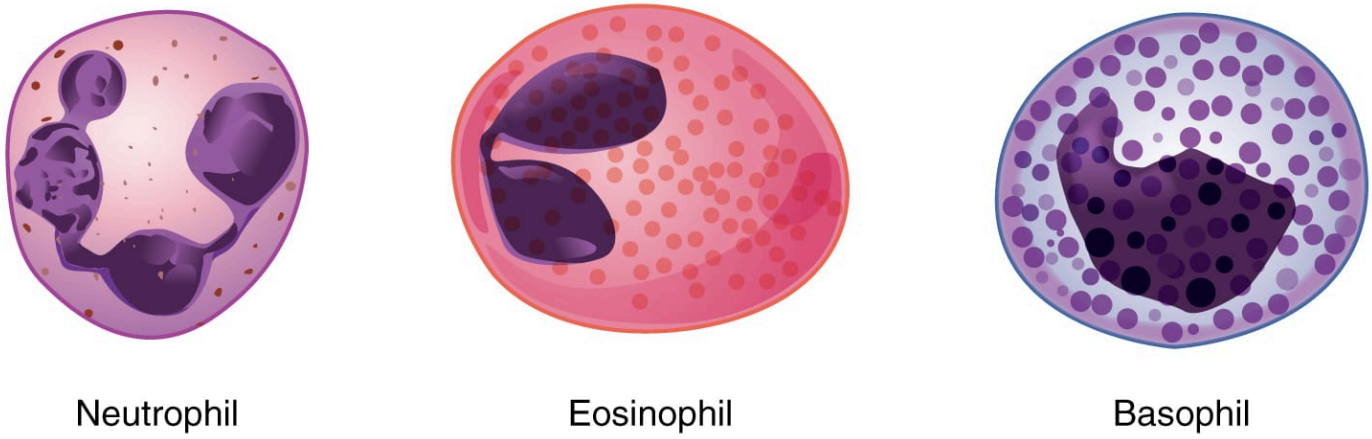


Figure 5.4.2: A neutrophil has small granules that stain light lilac and a nucleus with two to five lobes. An eosinophil's granules are slightly larger and stain reddish-orange, and its nucleus has two to three lobes. A basophil has large granules that stain dark blue to purple and a two-lobed nucleus. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.4.2.

The most common of all the leukocytes, neutrophils will normally comprise 50–70 percent of total leukocyte count. They are 10–12 μm in diameter, significantly larger than erythrocytes. They are called neutrophils because their granules show up most clearly with stains that are chemically neutral (neither acidic nor basic). The granules are numerous but quite fine and normally appear light lilac. The nucleus has a distinct lobed appearance and may have two to five lobes, the number increasing with the age of the cell. Older neutrophils have increasing numbers of lobes and are often referred to as polymorphonuclear (a nucleus with many forms), or simply "polys." Younger and immature neutrophils begin to develop lobes and are known as "bands."

Neutrophils are rapid responders to the site of infection and are efficient phagocytes with a preference for bacteria. Their granules include lysozyme, an enzyme capable of lysing, or breaking down, bacterial cell walls; oxidants such as hydrogen peroxide; and defensins, proteins that bind to and puncture bacterial and fungal plasma membranes, so that the cell contents leak out. Abnormally high counts of neutrophils indicate infection and/or inflammation, particularly triggered by bacteria, but are also found in burn patients and others experiencing unusual stress. A burn injury increases the proliferation of neutrophils in order to fight off infection that can result from the destruction of the barrier of the skin. Low counts may be caused by drug toxicity and other disorders, and may increase an individual's susceptibility to infection.

Eosinophils typically represent 2–4 percent of total leukocyte count. They are also 10–12 μm in diameter. The granules of eosinophils stain best with an acidic stain known as eosin. The nucleus of the eosinophil will typically have two to three lobes and, if stained properly, the granules will have a distinct red to orange color.

The granules of eosinophils include antihistamine molecules, which counteract the activities of histamines, inflammatory chemicals produced by basophils and mast cells. Some eosinophil granules contain molecules toxic to parasitic worms, which can enter the body through the integument, or when an individual consumes raw or undercooked fish or meat. Eosinophils are also capable of phagocytosis and are particularly effective when antibodies bind to the target and form an antigen-antibody complex. High counts of eosinophils are typical of patients experiencing allergies, parasitic worm infestations, and some autoimmune diseases. Low counts may be due to drug toxicity and stress.

Basophils are the least common leukocytes, typically comprising less than one percent of the total leukocyte count. They are slightly smaller than neutrophils and eosinophils at 8–10 μm in diameter. The granules of basophils stain best with basic (alkaline) stains. Basophils contain large granules that pick up a dark blue stain and are so common they may make it difficult to see the two-lobed nucleus.

In general, basophils intensify the inflammatory response. They share this trait with mast cells. In the past, mast cells were considered to be basophils that left the circulation. However, this appears not to be the case, as the two cell types develop from different lineages.

The granules of basophils release histamines, which contribute to inflammation, and heparin, which opposes blood clotting. High counts of basophils are associated with allergies, parasitic infections, and hypothyroidism. Low counts are associated with pregnancy, stress, and hyperthyroidism.

Agranular Leukocytes

Agranular leukocytes contain smaller, less-visible granules in their cytoplasm than do granular leukocytes. The nucleus is simple in shape, sometimes with an indentation but without distinct lobes. There are two major types of agranulocytes: lymphocytes and monocytes.

Lymphocytes are the only formed element of blood that arises from lymphoid stem cells. Although they form initially in the bone marrow, much of their subsequent development and reproduction occurs in the lymphatic tissues. Lymphocytes are the second most common type of leukocyte, accounting for about 20–30 percent of all leukocytes, and are essential for the immune response. The size range of lymphocytes is quite extensive, with some authorities recognizing two size classes and others three. Typically, the large cells are 10–14 μm and have a smaller nucleus-to-cytoplasm ratio and more granules. The smaller cells are typically 6–9 μm with a larger volume of nucleus to cytoplasm, creating a "halo" effect. A few cells may fall outside these ranges, at 14–17 μm. This finding has led to the three size range classification.

The three major groups of lymphocytes include natural killer cells, B cells, and T cells. Natural killer (NK) cells are capable of recognizing cells that do not express "self" proteins on their plasma membrane or that contain foreign or abnormal markers. These "nonself" cells include cancer cells, cells infected with a virus, and other cells with atypical surface proteins. Thus, they provide generalized, nonspecific immunity. The larger lymphocytes are typically NK cells.

B cells and T cells, also called B lymphocytes and T lymphocytes, play prominent roles in defending the body against specific pathogens (disease-causing microorganisms) and are involved in specific immunity. One form of B cells (plasma cells) produces the antibodies or immunoglobulins that bind to specific foreign or abnormal components of plasma membranes. This is also referred to as humoral (body fluid) immunity. T cells provide cellular-level immunity by physically attacking foreign or diseased cells. A memory cell is a variety of both B and T cells that forms after exposure to a pathogen and mounts rapid responses upon subsequent exposures. Unlike other leukocytes, memory cells live for many years. B cells undergo a maturation process in the bone marrow, whereas T cells undergo maturation in the thymus. This site of the maturation process gives rise to the name B and T cells. The functions of lymphocytes are complex and will be covered in detail in the chapter covering the lymphatic system and immunity. Smaller lymphocytes are either B or T cells, although they cannot be differentiated in a normal blood smear.

Abnormally high lymphocyte counts are characteristic of viral infections as well as some types of cancer. Abnormally low lymphocyte counts are characteristic of prolonged (chronic) illness or immunosuppression, including that caused by HIV infection and drug therapies that often involve steroids.

Monocytes originate from myeloid stem cells. They normally represent 2–8 percent of the total leukocyte count. They are typically easily recognized by their large size of 12–20 μm and indented or horseshoe-shaped nuclei. Macrophages are monocytes that have left the circulation and phagocytize debris, foreign pathogens, worn-out erythrocytes, and many other dead, worn out, or damaged cells. Macrophages also release antimicrobial defensins and chemotactic chemicals that attract other leukocytes to the site of an infection. Some macrophages occupy fixed locations, whereas others wander through the tissue fluid.

Abnormally high counts of monocytes are associated with viral or fungal infections, tuberculosis, and some forms of leukemia and other chronic diseases. Abnormally low counts are typically caused by suppression of the bone marrow.

Lifecycle of Leukocytes

Most leukocytes have a relatively short lifespan, typically measured in hours or days. Production of all leukocytes begins in the bone marrow under the influence of CSFs and interleukins. Secondary production and maturation of lymphocytes occurs in specific regions of lymphatic tissue known as germinal centers. Lymphocytes are fully capable of mitosis and may produce clones of cells with identical properties. This capacity enables an individual to maintain immunity throughout life to many threats that have been encountered in the past.

Disorders of Leukocytes

Leukopenia is a condition in which too few leukocytes are produced. If this condition is pronounced, the individual may be unable to ward off disease. Excessive leukocyte proliferation is known as leukocytosis. Although leukocyte counts are high, the cells themselves are often nonfunctional, leaving the individual at increased risk for disease.

Leukemia is a cancer involving an abundance of leukocytes. It may involve only one specific type of leukocyte from either

the myeloid line (myelocytic leukemia) or the lymphoid line (lymphocytic leukemia). In chronic leukemia, mature leukocytes accumulate and fail to die. In acute leukemia, there is an overproduction of young, immature leukocytes. In both conditions the cells do not function properly.

Lymphoma is a form of cancer in which masses of malignant T and/or B lymphocytes collect in lymph nodes, the spleen, the liver, and other tissues. As in leukemia, the malignant leukocytes do not function properly, and the patient is vulnerable to infection. Some forms of lymphoma tend to progress slowly and respond well to treatment. Others tend to progress quickly and require aggressive treatment, without which they are rapidly fatal.

Platelets

You may occasionally see platelets referred to as thrombocytes, but because this name suggests they are a type of cell, it is not accurate. A platelet is not a cell but rather a fragment of the cytoplasm of a cell called a megakaryocyte that is surrounded by a plasma membrane. Megakaryocytes are descended from myeloid stem cells (see Figure 18.4) and are large, typically 50–100 μm in diameter, and contain an enlarged, lobed nucleus. As noted earlier, thrombopoietin, a glycoprotein secreted by the kidneys and liver, stimulates the proliferation of megakaryoblasts, which mature into megakaryocytes. These remain within bone marrow tissue (Figure 5.4.3) and ultimately form platelet-precursor extensions that extend through the walls of bone marrow capillaries to release into the circulation thousands of cytoplasmic fragments, each enclosed by a bit of plasma membrane. These enclosed fragments are platelets. Each megakarocyte releases 2000–3000 platelets during its lifespan. Following platelet release, megakaryocyte remnants, which are little more than a cell nucleus, are consumed by macrophages.

Platelets are relatively small, 2–4 μm in diameter, but numerous, with typically 150,000–160,000 per μL of blood. After entering the circulation, approximately one-third migrate to the spleen for storage for later release in response to any rupture in a blood vessel. They then become activated to perform their primary function, which is to limit blood loss. Platelets remain only about 10 days, then are phagocytized by macrophages.

Platelets are critical to hemostasis, the stoppage of blood flow following damage to a vessel. They also secrete a variety of growth factors essential for growth and repair of tissue, particularly connective tissue. Infusions of concentrated platelets are now being used in some therapies to stimulate healing.

Disorders of Platelets

Thrombocytosis is a condition in which there are too many platelets. This may trigger formation of unwanted blood clots (thrombosis), a potentially fatal disorder. If there is an insufficient number of platelets, called thrombocytopenia, blood may not clot properly, and excessive bleeding may result.

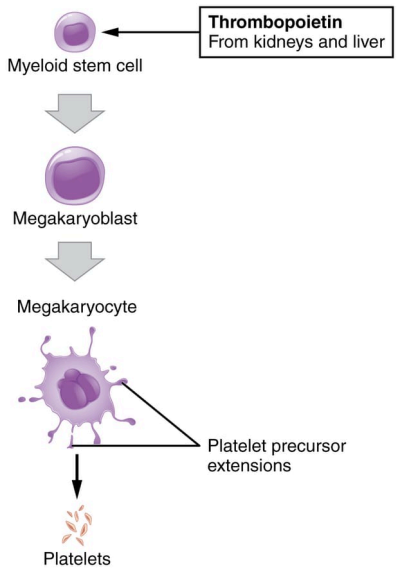


Figure 5.4.3: Platelets are derived from cells called megakaryocytes. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.4.3.

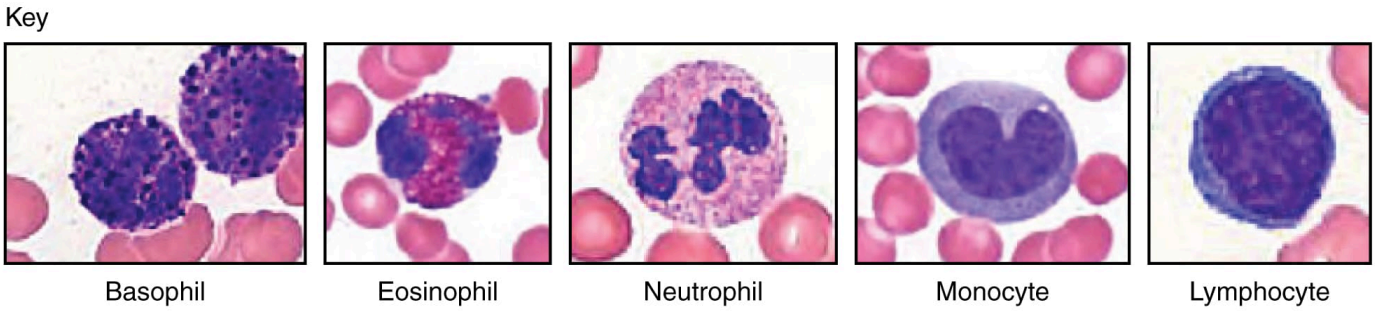


Figure 5.4.4: Reference images of the five major leukocyte types showing their characteristic nuclear shapes and staining patterns: basophil, eosinophil, neutrophil, monocyte, and lymphocyte. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.4.4.

Interactive Link

View University of Michigan Webscopes and explore the blood slides in greater detail. The Webscope feature allows you to move the slides as you would with a mechanical stage. You can increase and decrease the magnification. There is a chance to review each of the leukocytes individually after you have attempted to identify them from the first two blood smears. In addition, there are a few multiple choice questions.

Are you able to recognize and identify the various formed elements? You will need to do this is a systematic manner, scanning along the image. The standard method is to use a grid, but this is not possible with this resource. Try constructing a simple table with each leukocyte type and then making a mark for each cell type you identify. Attempt to classify at least 50 and perhaps as many as 100 different cells. Based on the percentage of cells that you count, do the numbers represent a normal blood smear or does something appear to be abnormal?

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- RNase with four intrachain disulfide bonds (1KF5) has no license indicated.

5.5: Hemostasis

Blood Clot Formation

Platelets are key players in hemostasis, the process by which the body seals a ruptured blood vessel and prevents further loss of blood. Although rupture of larger vessels usually requires medical intervention, hemostasis is quite effective in dealing with small, simple wounds. There are three steps to the process: vascular spasm, the formation of a platelet plug, and coagulation (blood clotting). Failure of any of these steps will result in hemorrhage—excessive bleeding.

Vascular Spasm

When a vessel is severed or punctured, or when the wall of a vessel is damaged, vascular spasm occurs. In vascular spasm, the smooth muscle in the walls of the vessel contracts dramatically. This smooth muscle has both circular layers; larger vessels also have longitudinal layers. The circular layers tend to constrict the flow of blood, whereas the longitudinal layers, when present, draw the vessel back into the surrounding tissue, often making it more difficult for a surgeon to locate, clamp, and tie off a severed vessel. The vascular spasm response is believed to be triggered by several chemicals called endothelins that are released by vessel-lining cells and by pain receptors in response to vessel injury. This phenomenon typically lasts for up to 30 minutes, although it can last for hours.

Formation of the Platelet Plug

In the second step, platelets, which normally float free in the plasma, encounter the area of vessel rupture with the exposed underlying connective tissue and collagenous fibers. The platelets begin to clump together, become spiked and sticky, and bind to the exposed collagen and endothelial lining. This process is assisted by a glycoprotein in the blood plasma called von Willebrand factor, which helps stabilize the growing platelet plug. As platelets collect, they simultaneously release chemicals from their granules into the plasma that further contribute to hemostasis. Among the substances released by the platelets are:

- adenosine diphosphate (ADP), which helps additional platelets to adhere to the injury site, reinforcing and expanding the platelet plug
- serotonin, which maintains vasoconstriction
- prostaglandins and phospholipids, which also maintain vasoconstriction and help to activate further clotting chemicals, as discussed next

A platelet plug can temporarily seal a small opening in a blood vessel. Plug formation, in essence, buys the body time while more sophisticated and durable repairs are being made. In a similar manner, even modern naval warships still carry an assortment of wooden plugs to temporarily repair small breaches in their hulls until permanent repairs can be made.

Coagulation

Those more sophisticated and more durable repairs are collectively called coagulation, the formation of a blood clot. The process is sometimes characterized as a cascade, because one event prompts the next as in a multi-level waterfall. The result is the production of a gelatinous but robust clot made up of a mesh of fibrin—an insoluble filamentous protein derived from fibrinogen, the plasma protein introduced earlier—in which platelets and blood cells are trapped. Figure $5.5.1$ summarizes the three steps of hemostasis.

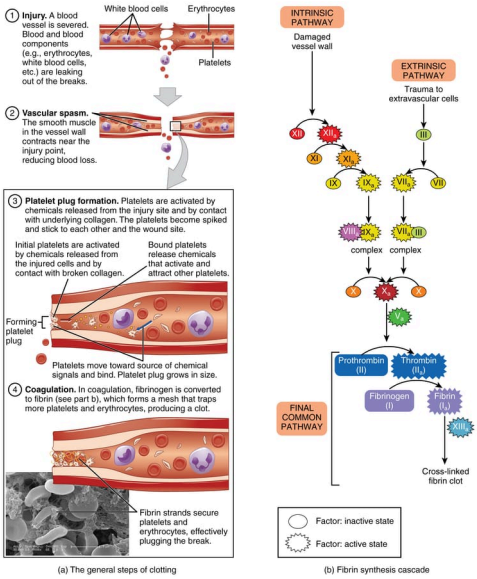


Figure 5.5.1: (a) An injury to a blood vessel initiates the process of hemostasis. Blood clotting involves three steps. First, vascular spasm constricts the flow of blood. Next, a platelet plug forms to temporarily seal small openings in the vessel. Coagulation then enables the repair of the vessel wall once the leakage of blood has stopped. (b) The synthesis of fibrin in blood clots involves either an intrinsic pathway or an extrinsic pathway, both of which lead to a common pathway. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.5.1.

Clotting Factors Involved in Coagulation

In the coagulation cascade, chemicals called clotting factors (or coagulation factors) prompt reactions that activate still more coagulation factors. The process is complex, but is initiated along two basic pathways:

- The extrinsic pathway, which normally is triggered by trauma.
- The intrinsic pathway, which begins in the bloodstream and is triggered by internal damage to the wall of the vessel.

Both of these merge into a third pathway, referred to as the common pathway (Figure 5.5.1*b*). All three pathways are dependent upon the 12 known clotting factors, including Ca^{2+} and vitamin K (Table 5.5.1). Clotting factors are secreted primarily by the liver and the platelets. The liver requires the fat-soluble vitamin K to produce many of them. Vitamin K (along with biotin and folate) is somewhat unusual among vitamins in that it is not only consumed in the diet but is also synthesized by bacteria residing in the large intestine. The calcium ion, considered factor IV, is derived from the diet and from the breakdown of bone. Some recent evidence indicates that activation of various clotting factors occurs on specific receptor sites on the surfaces of platelets.

The 12 clotting factors are numbered I through XIII according to the order of their discovery. Factor VI was once believed to be a distinct clotting factor, but is now thought to be identical to factor V. Rather than renumber the other factors, factor VI was allowed to remain as a placeholder and also a reminder that knowledge changes over time.

Table 5.5.1: Clotting Factors

Factor number	Name	Type of molecule	Source	Pathway(s)
I	Fibrinogen	Plasma protein	Liver	Common; converted into fibrin
II	Prothrombin	Plasma protein	Liver*	Common; converted into thrombin
III	Tissue thromboplastin or tissue factor	Lipoprotein mixture	Damaged cells and platelets	Extrinsic
IV	Calcium ions	Inorganic ions in plasma	Diet, platelets, bone matrix	Entire process
V	Proaccelerin	Plasma protein	Liver, platelets	Extrinsic and intrinsic
VI	Not used	Not used	Not used	Not used

Factor number	Name	Type of molecule	Source	Pathway(s)
VII	Proconvertin	Plasma protein	Liver *	Extrinsic
VIII	Antihemolytic factor A	Plasma protein factor	Platelets and endothelial cells	Intrinsic; deficiency results in hemophilia A
IX	Antihemolytic factor B (plasma thromboplastin component)	Plasma protein	Liver*	Intrinsic; deficiency results in hemophilia B
X	Stuart–Prower factor (thrombokinase)	Protein	Liver*	Extrinsic and intrinsic
XI	Antihemolytic factor C (plasma thromboplastin antecedent)	Plasma protein	Liver	Intrinsic; deficiency results in hemophilia C
XII	Hageman factor	Plasma protein	Liver	Intrinsic; initiates clotting in vitro also activates plasmin
XIII	Fibrin-stabilizing factor	Plasma protein	Liver, platelets	Stabilizes fibrin; slows fibrinolysis

Extrinsic Pathway

The quicker responding and more direct extrinsic pathway (also known as the tissue factor pathway) begins when damage occurs to the surrounding tissues, such as in a traumatic injury. Upon contact with blood plasma, the damaged extravascular cells, which are extrinsic to the bloodstream, release factor III (thromboplastin). Sequentially, Ca^{2+} then factor VII (proconvertin), which is activated by factor III, are added, forming an enzyme complex. This enzyme complex leads to activation of factor X (Stuart–Prower factor), which activates the common pathway discussed below. The events in the extrinsic pathway are completed in a matter of seconds.

Intrinsic Pathway

The intrinsic pathway (also known as the contact activation pathway) is longer and more complex. In this case, the factors involved are intrinsic to (present within) the bloodstream. The pathway can be prompted by damage to the tissues, resulting from internal factors such as arterial disease; however, it is most often initiated when factor XII (Hageman factor) comes into contact with foreign materials, such as when a blood sample is put into a glass test tube. Within the body, factor XII is typically activated when it encounters negatively charged molecules, such as inorganic polymers and phosphate produced earlier in the series of intrinsic pathway reactions. Factor XII sets off a series of reactions that in turn activates factor XI (antihemolytic factor C or plasma thromboplastin antecedent) then factor IX (antihemolytic factor B or plasma thromboplasmin). In the meantime, chemicals released by the platelets increase the rate of these activation reactions. Finally, factor VIII (antihemolytic factor A) from the platelets and endothelial cells combines with factor IX (antihemolytic factor B or plasma thromboplasmin) to form an enzyme complex that activates factor X (Stuart–Prower factor or thrombokinase), leading to the common pathway. The events in the intrinsic pathway are completed in a few minutes.

Common Pathway

Both the intrinsic and extrinsic pathways lead to the common pathway, in which fibrin is produced to seal off the vessel. Once factor X has been activated by either the intrinsic or extrinsic pathway, the enzyme prothrombinase converts factor II, the inactive enzyme prothrombin, into the active enzyme thrombin. (Note that if the enzyme thrombin were not normally in an inactive form, clots would form spontaneously, a condition not consistent with life.) Then, thrombin converts factor I, the soluble fibrinogen, into the insoluble fibrin protein strands. Factor XIII then stabilizes the fibrin clot.

Fibrinolysis

The stabilized clot is acted upon by contractile proteins within the platelets. As these proteins contract, they pull on the fibrin threads, bringing the edges of the clot more tightly together, somewhat as we do when tightening loose shoelaces (Figure $5.5.1a$). This process also wrings out of the clot a small amount of fluid called serum, which is blood plasma without its clotting factors.

To restore normal blood flow as the vessel heals, the clot must eventually be removed. Fibrinolysis is the gradual degradation of

the clot. Again, there is a fairly complicated series of reactions that involves factor XII and protein-catabolizing enzymes. During this process, the inactive protein plasminogen is converted into the active plasmin, which gradually breaks down the fibrin of the clot. Additionally, bradykinin, a vasodilator, is released, reversing the effects of the serotonin and prostaglandins from the platelets. This allows the smooth muscle in the walls of the vessels to relax and helps to restore the circulation.

Plasma Anticoagulants

An anticoagulant is any substance that opposes coagulation. Several circulating plasma anticoagulants play a role in limiting the coagulation process to the region of injury and restoring a normal, clot-free condition of blood. For instance, a cluster of proteins collectively referred to as the protein C system inactivates clotting factors involved in the intrinsic pathway. TFPI (tissue factor pathway inhibitor) inhibits the conversion of the inactive factor VII to the active form in the extrinsic pathway. Antithrombin inactivates factor X and opposes the conversion of prothrombin (factor II) to thrombin in the common pathway. And as noted earlier, basophils release heparin, a short-acting anticoagulant that also opposes prothrombin. Heparin is also found on the surfaces of cells lining the blood vessels. A pharmaceutical form of heparin is often administered therapeutically, for example, in surgical patients at risk for blood clots.

View these animations to explore the intrinsic, extrinsic, and common pathways that are involved the process of coagulation. The coagulation cascade restores hemostasis by activating coagulation factors in the presence of an injury. How does the endothelium of the blood vessel walls prevent the blood from coagulating as it flows through the blood vessels?

Disorders of Clotting

Either an insufficient or an excessive production of platelets can lead to severe disease or death. As discussed earlier, an insufficient number of platelets, called thrombocytopenia, typically results in the inability of blood to form clots. This can lead to excessive bleeding, even from minor wounds.

Another reason for failure of the blood to clot is the inadequate production of functional amounts of one or more clotting factors. This is the case in the genetic disorder hemophilia, which is actually a group of related disorders, the most common of which is hemophilia A, accounting for approximately 80 percent of cases. This disorder results in the inability to synthesize sufficient quantities of factor VIII. Hemophilia B is the second most common form, accounting for approximately 20 percent of cases. In this case, there is a deficiency of factor IX. Both of these defects are linked to the X chromosome and are typically passed from a healthy (carrier) female to their male offspring, since males are XY. Females would need to inherit a defective gene from each parent to manifest the disease, since they are XX. Patients with hemophilia bleed from even minor internal and external wounds, and leak blood into joint spaces after exercise and into urine and stool. Hemophilia C is a rare condition that is triggered by an autosomal (not sex) chromosome that renders factor XI nonfunctional. It is not a true recessive condition, since even individuals with a single copy of the mutant gene show a tendency to bleed. Regular infusions of clotting factors isolated from healthy donors can help prevent bleeding in hemophiliac patients. At some point, genetic therapy will become a viable option.

In contrast to the disorders characterized by coagulation failure is thrombocytosis, also mentioned earlier, a condition characterized by excessive numbers of platelets that increases the risk for excessive clot formation, a condition known as thrombosis. A thrombus (plural = thrombi) is an aggregation of platelets, erythrocytes, and even WBCs typically trapped within a mass of fibrin strands. While the formation of a clot is normal following the hemostatic mechanism just described, thrombi can form within an intact or only slightly damaged blood vessel. In a large vessel, a thrombus will adhere to the vessel wall and decrease the flow of blood, and is referred to as a mural thrombus. In a small vessel, it may actually totally block the flow of blood and is termed an occlusive thrombus. Thrombi are most commonly caused by vessel damage to the endothelial lining, which activates the clotting mechanism. These may include venous stasis, when blood in the veins, particularly in the legs, remains stationary for long periods. This is one of the dangers of long airplane flights in crowded conditions and may lead to deep vein thrombosis. Thrombophilia, also called hypercoagulation, is a condition in which there is a tendency to form thrombosis. This may be familial (genetic) or acquired. Acquired forms include the autoimmune disease lupus, immune reactions to heparin, polycythemia vera, thrombocytosis, sickle cell disease, pregnancy, and even obesity. A thrombus can seriously impede blood flow to or from a region and will cause a local increase in blood pressure. If flow is to be maintained, the heart will need to generate a greater pressure to overcome the resistance.

When a portion of a thrombus breaks free from the vessel wall and enters the circulation, it is referred to as an embolus. An embolus that is carried through the bloodstream can be large enough to block a vessel critical to a major organ. When it becomes trapped, an embolus is called an embolism. In the heart, brain, or lungs, an embolism may accordingly cause a heart attack, a stroke, or a pulmonary embolism. These are medical emergencies.

Among the many known biochemical activities of aspirin is its role as an anticoagulant. Aspirin (acetylsalicylic acid) is very effective at inhibiting the aggregation of platelets. It is routinely administered during a heart attack or stroke to reduce the adverse effects. Physicians sometimes recommend that patients at risk for cardiovascular disease take a low dose of aspirin on a daily basis as a preventive measure. However, aspirin can also lead to serious side effects, including increasing the risk of ulcers. A patient is well advised to consult a physician before beginning any aspirin regimen.

A class of drugs collectively known as thrombolytic agents can help speed up the degradation of an abnormal clot. If a thrombolytic agent is administered to a patient within 3 hours following a thrombotic stroke, the patient's prognosis improves significantly. However, some strokes are not caused by thrombi, but by hemorrhage. Thus, the cause must be determined before treatment begins. Tissue plasminogen activator is an enzyme that catalyzes the conversion of plasminogen to plasmin, the primary enzyme that breaks down clots. It is released naturally by endothelial cells but is also used in clinical medicine. New research is progressing using compounds isolated from the venom of some species of snakes, particularly vipers and cobras, which may eventually have therapeutic value as thrombolytic agents.

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5.6: Blood Typing

Blood Groups and Transfusion Reactions

Blood transfusions in humans were risky procedures until the discovery of the major human blood groups by Karl Landsteiner, an Austrian biologist and physician, in 1900. Until that point, physicians did not understand that death sometimes followed blood transfusions, when the type of donor blood infused into the patient was incompatible with the patient's own blood. Blood groups are determined by the presence or absence of specific marker molecules on the plasma membranes of erythrocytes. With their discovery, it became possible for the first time to match patient-donor blood types and prevent transfusion reactions and deaths.

Antigens, Antibodies, and Transfusion Reactions

Antigens are substances that the body does not recognize as belonging to the "self" and that therefore trigger a defensive response from the leukocytes of the immune system. (Seek more content for additional information on immunity.) Here, we will focus on the role of immunity in blood transfusion reactions. With RBCs in particular, you may see the antigens referred to as isoantigens or agglutinogens (surface antigens) and the antibodies referred to as isoantibodies or agglutinins. In this chapter, we will use the more common terms antigens and antibodies.

Antigens are generally large proteins, but may include other classes of organic molecules, including carbohydrates, lipids, and nucleic acids. Following an infusion of incompatible blood, erythrocytes with foreign antigens appear in the bloodstream and trigger an immune response. Proteins called antibodies (immunoglobulins), which are produced by certain B lymphocytes called plasma cells, attach to the antigens on the plasma membranes of the infused erythrocytes and cause them to adhere to one another.

- Because the arms of the Y-shaped antibodies attach randomly to more than one nonself erythrocyte surface, they form clumps of erythrocytes. This process is called agglutination.
- The clumps of erythrocytes block small blood vessels throughout the body, depriving tissues of oxygen and nutrients.
- As the erythrocyte clumps are degraded, in a process called hemolysis, their hemoglobin is released into the bloodstream. This hemoglobin travels to the kidneys, which are responsible for filtration of the blood. However, the load of hemoglobin released can easily overwhelm the kidney's capacity to clear it, and the patient can quickly develop kidney failure.

More than 50 antigens have been identified on erythrocyte membranes, but the most significant in terms of their potential harm to patients are classified in two groups: the ABO blood group and the Rh blood group.

The ABO Blood Group

Although the ABO blood group name consists of three letters, ABO blood typing designates the presence or absence of just two antigens, A and B. Both are glycoproteins. People whose erythrocytes have A antigens on their erythrocyte membrane surfaces are designated blood type A, and those whose erythrocytes have B antigens are blood type B. People can also have both A and B antigens on their erythrocytes, in which case they are blood type AB. People with neither A nor B antigens are designated blood type O. ABO blood types are genetically determined.

The body must be exposed to a foreign antigen before an antibody can be produced. ABO blood group antigens are found in foods and microbes throughout nature. Thus, the human immune system is exposed to A and B antigens at an early age and antibodies are formed naturally. Individuals with type A blood—without any prior exposure to incompatible blood—have naturally-formed antibodies to the B antigen circulating in their blood plasma. These antibodies, referred to as anti-B antibodies, will cause agglutination and hemolysis if they ever encounter erythrocytes with B antigens. Similarly, an individual with type B blood has naturally-formed anti-A antibodies. Individuals with type AB blood, which has both antigens, do not have naturally-formed antibodies to either of these. People with type O blood lack antigens A and B on their erythrocytes, but both anti-A and anti-B antibodies circulate in their blood plasma.

Rh Blood Groups

The Rh blood group is classified according to the presence or absence of a second erythrocyte antigen identified as Rh. (It was first discovered in a type of primate known as a rhesus macaque, which is often used in research, because its blood is similar to that of humans.) Although dozens of Rh antigens have been identified, only one, designated D, is clinically important. Those who have the Rh D antigen present on their erythrocytes—about 85 percent of Americans—are described as Rh positive (Rh^+) and those who lack it are Rh negative (Rh^-). Note that the Rh group is distinct from the ABO group, so any individual, no matter their ABO blood type, may have or lack this Rh antigen. When identifying a patient's blood type, the Rh group is designated by adding the word positive or negative to the ABO type. For example, A positive (A^+) means ABO group A blood with the Rh antigen present, and AB negative (AB^-) means ABO group AB blood without the Rh antigen. Table 5.6.1 summarizes the distribution of the ABO and Rh blood types within the United States.

Table 5.6.1: Summary of ABO and Rh Blood Types within the United States

Blood Type	Asian	Black non-Hispanic	Hispanic	North American Indian	White non-Hispanic
A^+	27.3	24.0	28.7	31.3	33.0
A^-	0.5	1.9	2.4	3.8	6.8
B^+	25.0	18.4	9.2	7.0	9.1
B^-	0.4	1.3	0.7	0.9	1.8
AB^+	7.0	4.0	2.3	2.2	3.4
AB^-	0.1	0.3	0.2	0.3	0.7
O^+	39.0	46.6	52.6	50.0	37.2
O^-	0.7	3.6	3.9	4.7	8.0

In contrast to the ABO group antibodies, which are preformed, antibodies to the Rh antigen are produced only in Rh^- individuals after exposure to the antigen. This process, called sensitization, occurs following a transfusion with Rh-incompatible blood or, more commonly, with the birth of an Rh^+ baby to an Rh^- person. Problems are rare in a first pregnancy, since the baby's Rh^+ cells rarely cross the placenta (the organ of gas and nutrient exchange between the fetus and the pregnant person). However, during or immediately after birth, the Rh^- parent can be exposed to the baby's Rh^+ cells (Figure 5.6.1). Research has shown that this occurs in about 13–14 percent of such pregnancies. After exposure, the immune system of the person who has given birth begins to generate anti-Rh antibodies. If the same person should then become pregnant with another Rh^+ baby, the Rh antibodies they have produced can cross the placenta into the fetal bloodstream and destroy the fetal RBCs. This condition, known as hemolytic disease of the newborn (HDN) or erythroblastosis fetalis, may cause anemia in mild cases, but the agglutination and hemolysis can be so severe that without treatment the fetus may die in the womb or shortly after birth.

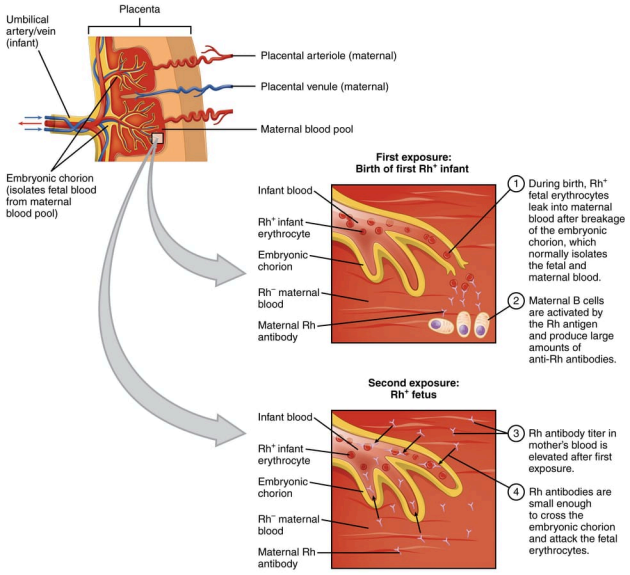


Figure 5.6.1: The first exposure of an Rh^- person to Rh^+ erythrocytes during pregnancy induces sensitization. Anti-Rh antibodies begin to circulate in the pregnant person's bloodstream. A second exposure occurs with a subsequent pregnancy with an Rh^+ fetus in the uterus. During that subsequent pregnancy, the pregnant person's anti-Rh antibodies may cross the placenta and enter the fetal bloodstream, causing agglutination and hemolysis of fetal erythrocytes. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.6.1.

A drug known as RhoGAM, short for Rh immune globulin, can temporarily prevent the development of Rh antibodies in the Rh^- parent, thereby averting this potentially serious disease for the fetus. RhoGAM antibodies destroy any fetal Rh^+ erythrocytes that may cross the placental barrier. RhoGAM is normally administered to Rh^- pregnant people during weeks 26–28 of pregnancy and within 72 hours following birth. It has proven remarkably effective in decreasing the incidence of HDN. Earlier we noted that the incidence of HDN in an Rh^+ subsequent pregnancy to an Rh^- person is about 13–14 percent without preventive treatment. Since the introduction of RhoGAM in 1968, the incidence has dropped to about 0.1 percent in the United States.

Determining ABO Blood Types

Clinicians are able to determine a patient's blood type quickly and easily using commercially prepared antibodies. An unknown blood sample is allocated into separate wells. Into one well a small amount of anti-A antibody is added, and to another a small amount of anti-B antibody. If the antigen is present, the antibodies will cause visible agglutination of the cells (Figure 5.6.2). The blood should also be tested for Rh antibodies.

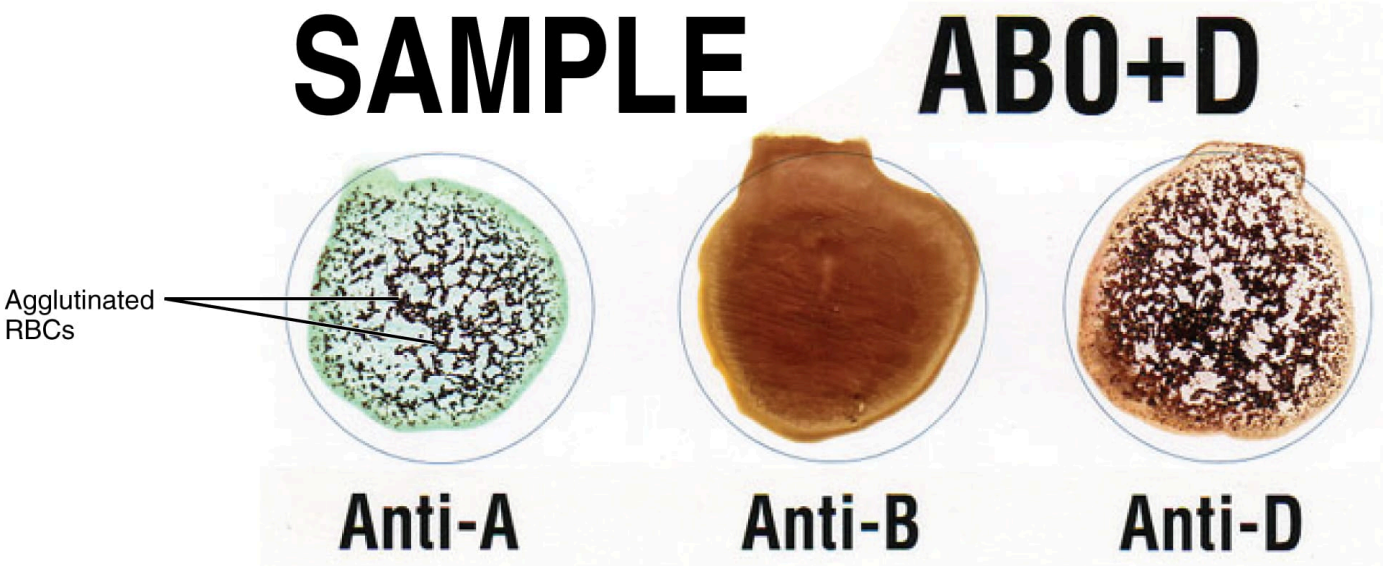


Figure 5.6.2: This sample of a commercially produced “bedside” card enables quick typing of both a recipient’s and donor’s blood before transfusion. The card contains three reaction sites or wells. One is coated with an anti-A antibody, one with an anti-B antibody, and one with an anti-D antibody (tests for the presence of Rh factor D). Mixing a drop of blood and saline into each well enables the blood to interact with a preparation of type-specific antibodies, also called anti-seras. Agglutination of RBCs in a given site indicates a positive identification of the blood antigens, in this case A and Rh antigens for blood type A^+. For the purpose of transfusion, the donor’s and recipient’s blood types must match. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.6.2.

ABO Transfusion Protocols

To avoid transfusion reactions, it is best to transfuse only matching blood types; that is, a type B^+ recipient should ideally receive blood only from a type B^+ donor and so on. That said, in emergency situations, when acute hemorrhage threatens the patient’s life, there may not be time for cross matching to identify blood type. In these cases, blood from a universal donor—an individual with type O^- blood—may be transfused. Recall that type O erythrocytes do not display A or B antigens. Thus, anti-A or anti-B antibodies that might be circulating in the patient’s blood plasma will not encounter any erythrocyte surface antigens on the donated blood and therefore will not be provoked into a response. One problem with this designation of universal donor is if the O^- individual had prior exposure to Rh antigen, Rh antibodies may be present in the donated blood. Also, introducing type O blood into an individual with type A, B, or AB blood will nevertheless introduce antibodies against both A and B antigens, as these are always circulating in the type O blood plasma. This may cause problems for the recipient, but because the volume of blood transfused is much lower than the volume of the patient’s own blood, the adverse effects of the relatively few infused plasma antibodies are typically limited. Rh factor also plays a role. If Rh^- individuals receiving blood have had prior exposure to Rh antigen, antibodies for this antigen may be present in the blood and trigger agglutination to some degree. Although it is always preferable to cross match a patient’s blood before transfusing, in a true life-threatening emergency situation, this is not always possible, and these procedures may be implemented.

A patient with blood type AB^+ is known as the universal recipient. This patient can theoretically receive any type of blood, because the patient’s own blood—having both A and B antigens on the erythrocyte surface—does not produce anti-A or anti-B antibodies. In addition, an Rh^+ patient can receive both Rh^+ and Rh^- blood. However, keep in mind that the donor’s blood will contain circulating antibodies, again with possible negative implications. Figure 5.6.3 summarizes the blood types and compatibilities.

At the scene of multiple-vehicle accidents, military engagements, and natural or human-caused disasters, many victims may suffer simultaneously from acute hemorrhage, yet type O blood may not be immediately available. In these circumstances, medics may at least try to replace some of the volume of blood that has been lost. This is done by intravenous administration of a saline solution that provides fluids and electrolytes in proportions equivalent to those of normal blood plasma. Research is ongoing to develop a safe and effective artificial blood that would carry out the oxygen-carrying function of blood without the RBCs, enabling transfusions in the field without concern for incompatibility. These blood substitutes normally contain hemoglobin- as well as perfluorocarbon-based oxygen carriers.

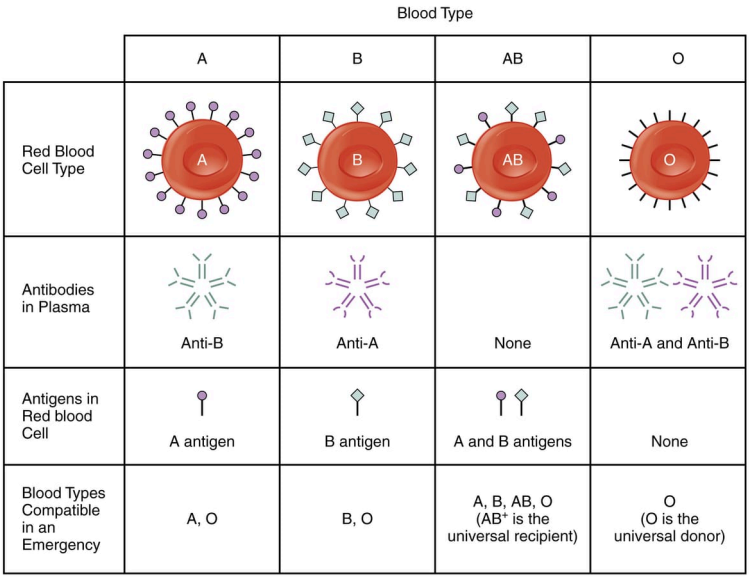


Figure 5.6.3: This chart summarizes the characteristics of the blood types in the ABO blood group. See the text for more on the concept of a universal donor or recipient. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 5.6.3.

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5.7: Terms and Definitions

Key Terms

Select to View Definition

ABO Blood Group
: blood-type classification based on the presence or absence of A and B glycoproteins on the erythrocyte membrane surface

Agglutination
: clustering of cells into masses linked by antibodies

Agranular Leukocytes
: leukocytes with few granules in their cytoplasm; specifically, monocytes, lymphocytes, and NK cells

Albumin
: most abundant plasma protein, accounting for most of the osmotic pressure of plasma

Anemia
: deficiency of red blood cells or hemoglobin

Antibodies
: (also, immunoglobulins or gamma globulins) antigen-specific proteins produced by specialized B lymphocytes that protect the body by binding to foreign objects such as bacteria and viruses

Anticoagulant
: substance such as heparin that opposes coagulation

Antithrombin
: anticoagulant that inactivates factor X and opposes the conversion of prothrombin (factor II) into thrombin in the common pathway

B Lymphocytes
: (also, B cells) lymphocytes that defend the body against specific pathogens and thereby provide specific immunity

Basophils
: granulocytes that stain with a basic (alkaline) stain and store histamine and heparin

Bilirubin
: yellowish bile pigment produced when iron is removed from heme and is further broken down into waste products

Biliverdin
: green bile pigment produced when the non-iron portion of heme is degraded into a waste product; converted to bilirubin in the liver

Blood
: liquid connective tissue composed of formed elements—erythrocytes, leukocytes, and platelets—and a fluid extracellular matrix called plasma; component of the cardiovascular system

Bone Marrow Biopsy
: diagnostic test of a sample of red bone marrow

Bone Marrow Transplant
: treatment in which a donor's healthy bone marrow with its stem cells replaces diseased or damaged bone marrow of a patient

Bruise
localized bleeding under the skin due to damaged blood vessels

Buffy Coat
thin, pale layer of leukocytes and platelets that separates the erythrocytes from the plasma in a sample of centrifuged blood

Carbaminohemoglobin
compound of carbon dioxide and hemoglobin, and one of the ways in which carbon dioxide is carried in the blood

Clotting Factors
group of 12 identified substances active in coagulation

Coagulation
formation of a blood clot; part of the process of hemostasis

Colony-Stimulating Factors (CSFs)
glycoproteins that trigger the proliferation and differentiation of myeloblasts into granular leukocytes (basophils, neutrophils, and eosinophils)

Common Pathway
final coagulation pathway activated either by the intrinsic or the extrinsic pathway, and ending in the formation of a blood clot

Cross Matching
blood test for identification of blood type using antibodies and small samples of blood

Cytokines
class of proteins that act as autocrine or paracrine signaling molecules; in the cardiovascular system, they stimulate the proliferation of progenitor cells and help to stimulate both nonspecific and specific resistance to disease

Defensins
antimicrobial proteins released from neutrophils and macrophages that create openings in the plasma membranes to kill cells

Deoxyhemoglobin
molecule of hemoglobin without an oxygen molecule bound to it

Diapedesis
(also, emigration) process by which leukocytes squeeze through adjacent cells in a blood vessel wall to enter tissues

Embolus
thrombus that has broken free from the blood vessel wall and entered the circulation

Emigration
(also, diapedesis) process by which leukocytes squeeze through adjacent cells in a blood vessel wall to enter tissues

Eosinophils
granulocytes that stain with eosin; they release antihistamines and are especially active against parasitic worms

Erythrocyte
(also, red blood cell) mature myeloid blood cell that is composed mostly of hemoglobin and functions primarily in the transportation of oxygen and carbon dioxide

Erythropoietin (EPO)
glycoprotein that triggers the bone marrow to produce RBCs; secreted by the kidney in response to low oxygen levels

Extrinsic Pathway
initial coagulation pathway that begins with tissue damage and results in the activation of the common pathway

Ferritin
protein-containing storage form of iron found in the bone marrow, liver, and spleen

Fibrin
insoluble, filamentous protein that forms the structure of a blood clot

Fibrinogen
plasma protein produced in the liver and involved in blood clotting

Fibrinolysis
gradual degradation of a blood clot

Formed Elements
cellular components of blood; that is, erythrocytes, leukocytes, and platelets

Globin
heme-containing globular protein that is a constituent of hemoglobin

Globulins
heterogeneous group of plasma proteins that includes transport proteins, clotting factors, immune proteins, and others

Granular Leukocytes
leukocytes with abundant granules in their cytoplasm; specifically, neutrophils, eosinophils, and basophils

Hematocrit
(also, packed cell volume) volume percentage of erythrocytes in a sample of centrifuged blood

Hematopoietic Stem Cell
type of pluripotent stem cell that gives rise to the formed elements of blood (hemocytoblast)

Heme
red, iron-containing pigment to which oxygen binds in hemoglobin

Hemocytoblast
hematopoietic stem cell that gives rise to the formed elements of blood

Hemoglobin
oxygen-carrying compound in erythrocytes

Hemosiderin
protein-containing storage form of iron found in the bone marrow, liver, and spleen

Hemostasis
physiological process by which bleeding ceases

Heparin
short-acting anticoagulant stored in mast cells and released when tissues are injured, opposes prothrombin

Hypoxemia
below-normal level of oxygen saturation of blood (typically <95 percent)

Immunoglobulins
(also, antibodies or gamma globulins) antigen-specific proteins produced by specialized B lymphocytes that protect the body by binding to foreign objects such as bacteria and viruses

Interleukins
signaling molecules that may function in hemopoiesis, inflammation, and specific immune responses

Intrinsic Pathway
initial coagulation pathway that begins with vascular damage or contact with foreign substances, and results in the activation of the common pathway

Jaundice
yellowing of the skin or whites of the eyes due to excess bilirubin in the blood

Leukemia
cancer involving leukocytes

Leukocyte
(also, white blood cell) colorless, nucleated blood cell, the chief function of which is to protect the body from disease

Leukocytosis
excessive leukocyte proliferation

Leukopenia
below-normal production of leukocytes

Lymphocytes
agranular leukocytes of the lymphoid stem cell line, many of which function in specific immunity

Lymphoid Stem Cells
type of hematopoietic stem cells that gives rise to lymphocytes, including various T cells, B cells, and NK cells, all of which function in immunity

Lymphoma
form of cancer in which masses of malignant T and/or B lymphocytes collect in lymph nodes, the spleen, the liver, and other tissues

Lysozyme
digestive enzyme with bactericidal properties

Macrophage
phagocytic cell of the myeloid lineage; a matured monocyte

Megakaryocyte
bone marrow cell that produces platelets

Memory Cell
type of B or T lymphocyte that forms after exposure to a pathogen

Monocytes
agranular leukocytes of the myeloid stem cell line that circulate in the bloodstream; tissue monocytes are macrophages

Myeloid Stem Cells
type of hematopoietic stem cell that gives rise to some formed elements, including erythrocytes, megakaryocytes that produce platelets, and a myeloblast lineage that gives rise to monocytes and three forms of granular leukocytes (neutrophils, eosinophils, and basophils)

Natural Killer (NK) Cells
cytotoxic lymphocytes capable of recognizing cells that do not express “self” proteins on their plasma membrane or that contain foreign or abnormal markers; provide generalized, nonspecific immunity

Neutrophils
granulocytes that stain with a neutral dye and are the most numerous of the leukocytes; especially active against bacteria

Oxyhemoglobin
molecule of hemoglobin to which oxygen is bound

Packed Cell Volume (PCV)
(also, hematocrit) volume percentage of erythrocytes present in a sample of centrifuged blood

Plasma
in blood, the liquid extracellular matrix composed mostly of water that circulates the formed elements and dissolved materials throughout the cardiovascular system

Plasmin
blood protein active in fibrinolysis

Platelet Plug
accumulation and adhesion of platelets at the site of blood vessel injury

Platelets
(also, thrombocytes) one of the formed elements of blood that consists of cell fragments broken off from megakaryocytes

Pluripotent Stem Cell
stem cell that derives from totipotent stem cells and is capable of differentiating into many, but not all, cell types

Polycythemia
elevated level of hemoglobin, whether adaptive or pathological

Polymorphonuclear
having a lobed nucleus, as seen in some leukocytes

Positive Chemotaxis
process in which a cell is attracted to move in the direction of chemical stimuli

Red Blood Cells (RBCs)
(also, erythrocytes) one of the formed elements of blood that transports oxygen

Reticulocyte
immature erythrocyte that may still contain fragments of organelles

Rh Blood Group
blood-type classification based on the presence or absence of the antigen Rh on the erythrocyte membrane surface

Serum
blood plasma that does not contain clotting factors

Sickle Cell Disease
(also, sickle cell anemia) inherited blood disorder in which hemoglobin molecules are malformed, leading to the breakdown of RBCs that take on a characteristic sickle shape

T Lymphocytes
(also, T cells) lymphocytes that provide cellular-level immunity by physically attacking foreign or diseased cells

Thalassemia
inherited blood disorder in which maturation of RBCs does not proceed normally, leading to abnormal formation of hemoglobin and the destruction of RBCs

Thrombin
enzyme essential for the final steps in formation of a fibrin clot

Thrombocytes
platelets, one of the formed elements of blood that consists of cell fragments broken off from megakaryocytes

Thrombocytopenia
condition in which there are too few platelets, resulting in abnormal bleeding (hemophilia)

Thrombocytosis
condition in which there are too many platelets, resulting in abnormal clotting (thrombosis)

Thrombopoietin
hormone secreted by the liver and kidneys that prompts the development of megakaryocytes into thrombocytes (platelets)

Thrombosis
excessive clot formation

Thrombus
aggregation of fibrin, platelets, and erythrocytes in an intact artery or vein

Tissue Factor
protein thromboplastin, which initiates the extrinsic pathway when released in response to tissue damage

Totipotent Stem Cell
embryonic stem cell that is capable of differentiating into any and all cells of the body; enabling the full development of an organism

Transferrin
plasma protein that binds reversibly to iron and distributes it throughout the body

Universal Donor
individual with type O^- blood

Universal Recipient
individual with type AB^+ blood

Vascular Spasm
initial step in hemostasis, in which the smooth muscle in the walls of the ruptured or damaged blood vessel contracts

White Blood Cells (WBCs)
(also, leukocytes) one of the formed elements of blood that provides defense against disease agents and foreign materials

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5.8: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. Which of the following statements about blood is true?
   A. Blood is about 92 percent water.
   B. Blood is slightly more acidic than water.
   C. Blood is slightly more viscous (thicker) than water.
   D. Blood is slightly more salty than seawater.

2. Which of the following statements about albumin is true?
   A. It draws water out of the blood vessels and into the body's tissues.
   B. It is the most abundant plasma protein.
   C. It is produced by specialized leukocytes called plasma cells.
   D. All of the above are true.

3. Which of the following plasma proteins is NOT produced by the liver?
   A. Fibrinogen
   B. Alpha globulin
   C. Beta globulin
   D. Immunoglobulin (antibody)

4. Which of the formed elements arises from myeloid stem cells?
   A. B cells
   B. Natural killer (NK) cells
   C. Platelets
   D. All of the above

5. Which of the following statements about erythropoietin (EPO) is true?
   A. It stimulates proliferation and differentiation of the erythrocyte lineage.
   B. It is a hormone produced mainly by the thyroid gland.
   C. It is a growth factor that prompts lymphoid stem cells to leave the bone marrow.
   D. Both A and B are true.

6. Interleukins are primarily associated with which of the following?
   A. Production of various lymphocytes
   B. Immune responses
   C. Inflammation
   D. All of the above

7. Which of the following statements about mature, circulating erythrocytes is true?
   A. They have no nucleus.
   B. They are packed with mitochondria.
   C. They survive for an average of about 4 days.
   D. All of the above

8. A molecule of hemoglobin:
   A. Is shaped like a biconcave disk packed almost entirely with iron
   B. Contains four glycoprotein units studded with oxygen
   C. Consists of four globin proteins, each bound to one heme group
   D. Can carry up to 120 molecules of oxygen

9. The production of healthy erythrocytes depends on the availability of:
   A. Copper
   B. Zinc
   C. Vitamin B12
   D. Copper, zinc, and vitamin B12

10. Aging and damaged erythrocytes are removed from the circulation mainly by:
    A. Myeloblasts
    B. Monocytes in circulating blood
    C. Macrophages in the spleen and liver
    D. Mast cells in connective tissue

11. A patient has been suffering for 2 months with chronic, watery diarrhea and is now very dehydrated. Which blood finding is most likely?
    A. A hematocrit below 30 percent
    B. Hypoxemia (low blood oxygen)
    C. Anemia
    D. Apparent polycythemia from loss of plasma volume

12. The process by which leukocytes squeeze between cells of a blood vessel wall to leave the bloodstream is called:
    A. Leukocytosis
    B. Positive chemotaxis
    C. Emigration (diapedesis)
    D. Cytoplasmic extending

13. Which of the following best describes a neutrophil?
    A. Abundant, agranular, especially effective against cancer cells
    B. Abundant, granular, especially effective against bacteria
    C. Rare, agranular, releases antimicrobial defensins
    D. Rare, granular, contains many granules packed with histamine

14. T and B lymphocytes:
    A. Are polymorphonuclear cells
    B. Are involved in specific (adaptive) immune function
    C. Proliferate excessively in leukopenia
    D. Are most active against parasitic worms

15. A patient has severe, persistent allergy symptoms (runny nose, itching) that improve with an antihistamine. Before treatment, which leukocyte type was probably especially active?
    A. Basophils
    B. Neutrophils
    C. Monocytes
    D. Natural killer (NK) cells

16. Thrombocytes are more accurately called:
    A. Clotting factors
    B. Megakaryoblasts
    C. Megakaryocytes
    D. Platelets

17. The first step in hemostasis is:
    A. Vascular spasm (vasoconstriction)
    B. Conversion of fibrinogen to fibrin

C. Activation of the intrinsic pathway
D. Activation of the common pathway

18. Prothrombin is converted to thrombin during the:
A. Intrinsic pathway
B. Extrinsic pathway
C. Common pathway
D. Formation of the platelet plug

19. Hemophilia is characterized by:
A. Inadequate production of heparin
B. Inadequate production or function of clotting factors
C. Excessive production of fibrinogen
D. Excessive production of platelets

20. The process in which antibodies bind to antigens on cells, causing clumping into visible masses, is called:
A. Sensitization
B. Coagulation
C. Agglutination
D. Hemolysis

21. People with ABO blood type O:
A. Have both antigens A and B on their erythrocytes
B. Lack both antigens A and B on their erythrocytes
C. Have neither anti-A nor anti-B antibodies in their plasma
D. Are considered universal recipients

22. Hemolytic disease of the newborn is a risk in a later pregnancy when:
A. A type AB woman is carrying a type O fetus
B. A type O woman is carrying a type AB fetus
C. An Rh+ woman is carrying an Rh– fetus
D. An Rh– woman is carrying a second Rh+ fetus

23. A 5-year-old boy is pale and tired. Lab tests show low hemoglobin, low RBC count, and normal WBC and platelet counts. Which condition is most consistent with these findings?
A. Leukemia
B. Isolated anemia
C. Thrombocytopenia
D. Polycythemia vera

24. A 60-year-old woman with long-term smoking history has a very high hematocrit and high RBC count, but normal total blood volume. This change is most likely a response to:
A. Chronic hypoxia stimulating erythropoietin release
B. Excessive iron intake
C. Liver failure reducing albumin
D. Vitamin K deficiency

25. After a deep cut, a patient's bleeding stops quickly and a firm clot forms. Which part of hemostasis is MOST directly responsible for forming the stable fibrin mesh?
A. Platelet plug alone
B. Conversion of fibrinogen to fibrin by thrombin
C. Vasoconstriction of the damaged vessel
D. Emigration of neutrophils into the wound

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5.9: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. Blood is often called a “connective tissue.” Based on its components and functions, explain why blood fits this classification, even though it is a fluid.
   Hint: Think about what all connective tissues do (transport, support, protection) and how plasma and formed elements help connect different organs.

2. A patient has a hematocrit of 60%. What might this tell you about the relative amounts of red blood cells and plasma, and how could this affect blood flow and the heart?
   Hint: High hematocrit means more red blood cells and thicker blood. Think about how easily the blood can move through vessels.

3. Red blood cells have no nucleus and very few organelles. Explain one advantage and one disadvantage of this special structure.
   Hint: Think about space for hemoglobin and oxygen transport, and also what is lost when a cell cannot divide or repair itself.

4. A person with chronic kidney disease often has low red blood cell counts (anemia). Using your knowledge of blood physiology, explain why a kidney problem can lead to a blood problem.
   Hint: Remember which hormone from the kidney controls red blood cell production in the bone marrow.

5. Compare how blood would change in:
   a) acute blood loss from an accident, and
   b) long-term iron deficiency.
   Explain how each situation affects red blood cells and oxygen-carrying capacity.
   Hint: Think about speed of onset, total blood volume, and whether the body has time and materials to make normal RBCs.

6. A patient’s white blood cell count is very low after chemotherapy. Explain how this changes their risk of infection, even if their skin and mucous membranes are normal.
   Hint: Consider the role of leukocytes in recognizing and destroying pathogens that get past physical barriers.

7. Platelets and plasma clotting factors work together to stop bleeding. Describe what might happen if:
   a) platelet number is very low, but clotting proteins are normal,
   b) clotting proteins (like fibrinogen) are very low, but platelet number is normal.
   Hint: Think about the difference between forming a temporary platelet plug and making a stable fibrin clot.

8. A patient has long-standing, uncontrolled high blood pressure. Explain how this might increase their risk of forming dangerous blood clots, and how that relates to the normal functions of blood.

Hint: Consider how high pressure affects vessel walls, platelets, and flow patterns, and how clots can block blood supply.

9. Someone has type A negative (A–) blood. Explain which ABO and Rh antigens are present (or absent) on their red blood cells, and which types of donor blood they can safely receive in an emergency.
   Hint: Think separately about A/B antigens and the Rh factor. In emergency situations, consider both antigen match and risk of reaction.

10. During a severe infection, a patient's blood test shows high neutrophils and an increased total white blood cell count. Explain why this pattern is expected, and how it helps the body fight the infection.
    Hint: Recall which type of leukocyte is the main first responder to bacterial infection and how increasing their number is useful.

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5.10: Alternate Text Descriptions

Figure 5.1.1: Comparison of Hematocrit in Normal Blood, Anemia, and Polycythemia

The figure compares centrifuged blood samples representing **normal blood**, **anemia**, and **polycythemia**, illustrating differences in hematocrit, the percentage of blood volume occupied by red blood cells.

1. Each test tube contains blood separated into three layers after centrifugation.
2. The upper yellow layer is labeled **plasma**, which contains water, proteins, nutrients, hormones, and other dissolved substances.
3. A thin middle layer, labeled the **buffy coat**, contains **white blood cells and platelets**.
4. The lower red layer is labeled **hematocrit** and consists of **red blood cells**.

Normal Blood

1. The left test tube represents **normal blood**.
2. The red blood cell layer occupies a typical proportion of the tube.
3. Reference values shown are:
    - **37% to 47% hematocrit** for females
    - **42% to 52% hematocrit** for males
4. The plasma layer occupies the remaining majority of the blood volume, with the buffy coat appearing as a thin band between plasma and red blood cells.

Anemia

1. The middle test tube represents **anemia**.
2. The red blood cell layer is noticeably reduced in size compared with normal blood.
3. The plasma layer occupies a larger proportion of the tube.
4. The figure identifies anemia as having a **depressed hematocrit percentage**, indicating a reduced proportion of red blood cells.

Polycythemia

1. The right test tube represents **polycythemia**.
2. The red blood cell layer occupies a much larger proportion of the blood volume than in the normal sample.
3. The plasma layer is correspondingly reduced in proportion.
4. The figure identifies polycythemia as having an **elevated hematocrit percentage**, indicating an increased proportion of red blood cells.

The figure demonstrates how blood composition can be assessed using hematocrit measurements. **Anemia** is characterized by a decreased proportion of red blood cells, while **polycythemia** is characterized by an increased proportion of red blood cells. The comparison highlights the relationship between plasma volume, the buffy coat, and red blood cell volume in different hematologic conditions.

Figure 5.2.1: Hematopoiesis: Development of Blood Cells from Hematopoietic Stem Cells

The figure illustrates **hematopoiesis**, the process by which all blood cells develop from a common stem cell in the bone marrow.

1. At the top of the diagram is a **multipotent hematopoietic stem cell, or hemocytoblast**. After division, some daughter cells

remain hematopoietic stem cells, maintaining the stem cell population.
2. Other stem cells differentiate into one of two major lineages depending on chemical signals they receive:
    - **Myeloid stem cell**
    - **Lymphoid stem cell**

Myeloid Lineage

1. The **myeloid stem cell** gives rise to several precursor cells that produce formed elements of the blood.
2. A **megakaryoblast** develops into a **megakaryocyte**, which releases **platelets**.
3. A **proerythroblast** develops into a **reticulocyte**, which matures into an **erythrocyte**, or **red blood cell**.
4. A **myeloblast** differentiates into three types of granulocytes:
    - **Basophil**
    - **Neutrophil**
    - **Eosinophil**
5. A **monoblast** develops into a **monocyte**.

Lymphoid Lineage

1. The **lymphoid stem cell** develops into a **lymphoblast**.
2. The lymphoblast can differentiate into:
    - A **natural killer, or NK, cell**, also identified as a large granular lymphocyte
    - A **small lymphocyte**
3. The small lymphocyte further differentiates into:
    - A **T lymphocyte, or T cell**
    - A **B lymphocyte, or B cell**

The figure demonstrates that all blood cells originate from a common **hematopoietic stem cell**. Differentiation along the **myeloid lineage** produces erythrocytes, platelets, granulocytes, and monocytes, whereas differentiation along the **lymphoid lineage** produces lymphocytes, including B cells, T cells, and natural killer cells. This process continuously replenishes the cellular components of blood throughout life.

Figure 5.3.2: Structure of Hemoglobin and the Heme Group

The figure compares the three-dimensional structure of **hemoglobin** with the chemical structure of its **heme** group.

Panel (a): Hemoglobin Structure

1. Hemoglobin is shown as a large protein composed of **four polypeptide subunits** arranged together in a compact structure.
2. The protein contains:
    - **Alpha chain 1 (α chain 1)**
    - **Alpha chain 2 (α chain 2)**
    - **Beta chain 1 (β chain 1)**
    - **Beta chain 2 (β chain 2)**
3. Each globin chain contains a **heme group**, shown as a disc-shaped structure embedded within the protein.
4. At the center of each heme group is an **iron ion (Fe2+)**.
5. The four subunits and their heme groups form the functional hemoglobin molecule that can bind and transport oxygen in the blood.

Panel (b): Heme Structure

1. The heme group is shown as a chemical structure consisting of a large ring-shaped organic molecule.
2. Four nitrogen-containing regions are arranged around a central **iron atom (Fe)**.
3. The iron atom is coordinated within the center of the ring and serves as the binding site for oxygen.
4. Chemical side groups attached to the ring include methyl (CH3), vinyl (CH2), and carboxyl (COOH) groups.
5. The diagram emphasizes the arrangement of the iron atom within the heme ring and the molecular structure that supports

oxygen binding.

The figure demonstrates that hemoglobin is composed of **two alpha chains and two beta chains**, each containing a **heme group** with a central **iron ion (Fe2+)**. Oxygen binds to the iron atom within each heme group, allowing a single hemoglobin molecule to transport up to four oxygen molecules through the bloodstream.

Figure 5.3.3: Life Cycle and Recycling of Erythrocytes (Red Blood Cells)

The figure illustrates the life cycle of **erythrocytes**, or **red blood cells**, including their production, circulation, breakdown, and recycling of hemoglobin components.

1. **Erythropoiesis begins in red bone marrow.**
   - Hematopoietic stem cells develop into **erythroblasts**, which mature into **reticulocytes**.
   - The diagram highlights major locations of hematopoietic bone marrow within the skeleton.
2. **Reticulocytes enter the bloodstream and mature into erythrocytes.**
   - Newly released reticulocytes complete maturation in the circulation.
   - Mature erythrocytes circulate in the blood for approximately **120 days**.
3. **Aging erythrocytes are removed from circulation.**
   - Old or damaged erythrocytes are phagocytized by **macrophages** in the bone marrow, liver, and spleen.
   - Lysosomes within macrophages break down the erythrocyte components.
4. **Globin is recycled.**
   - The globin portion of hemoglobin is degraded into **amino acids**.
   - These amino acids are reused in protein synthesis and other cellular processes.
5. **Heme is separated into iron and non-iron components.**
   - Each hemoglobin molecule contains four heme groups.
   - Iron ions are removed from heme and bind to **transferrin** for transport in the bloodstream.
   - The remaining heme structure is converted to **biliverdin**.
6. **Iron and heme breakdown products are reused or stored.**
   - Iron transported by transferrin can be returned to the bone marrow and reused in erythropoiesis.
   - Iron may also be stored in the liver bound to **ferritin**.
   - Biliverdin is converted to **bilirubin**, which is transported to the liver and becomes a component of bile.
   - Unused heme components can be recycled for future hemoglobin synthesis.

The figure demonstrates the continuous cycle of erythrocyte production, circulation, removal, and recycling. Components of hemoglobin are recovered and reused whenever possible, allowing iron and amino acids to contribute to the formation of new red blood cells while heme breakdown products are processed by the liver.

Figure 5.4.1: Leukocyte Migration and Immune Response at a Site of Infection

The figure illustrates the sequence of events by which **leukocytes** respond to tissue injury or infection and migrate from the bloodstream into affected tissues.

Step 1: Detection of Chemical Signals

1. A capillary containing circulating leukocytes is shown adjacent to injured tissue containing pathogens.
2. Leukocytes within the bloodstream include an **eosinophil**, **monocyte**, and **neutrophil**.
3. **Injured or infected cells** release chemical signals into the surrounding tissue and bloodstream.
4. **Pathogens** are present at the site of injury.
5. The chemical signals form a concentration gradient that attracts leukocytes toward the affected area.

Step 2: Leukocyte Emigration, or Diapedesis

1. Leukocytes migrate toward the highest concentration of chemical signals through a process called **positive chemotaxis**.
2. The leukocytes squeeze between adjacent **capillary endothelial cells** and pass through the capillary wall.

3. As they leave the bloodstream, the leukocytes move into the surrounding tissue toward the site of injury and infection.

Step 3: Response Within the Tissue

1. After entering the tissue, **monocytes** differentiate into **macrophages**.
2. Macrophages engulf and digest pathogens through **phagocytosis**.
3. **Neutrophils** and **eosinophils** also migrate to the site of infection.
4. Eosinophils release cytotoxic substances from intracellular granules into the tissue.
5. Neutrophils release antimicrobial substances and can also engulf pathogens by phagocytosis.
6. Together, macrophages, neutrophils, and eosinophils help eliminate invading microorganisms and damaged cellular material.

The figure demonstrates the leukocyte response to tissue injury and infection. Chemical signals released by damaged tissue and pathogens attract leukocytes from the bloodstream. The leukocytes exit capillaries by passing between endothelial cells, migrate toward the source of the signals, and participate in immune defense through phagocytosis and the release of antimicrobial chemicals.

Figure 5.4.2: Comparison of Neutrophils, Eosinophils, and Basophils

The figure compares three types of **granulocytes**: a **neutrophil**, **eosinophil**, and **basophil**, highlighting differences in nuclear shape and cytoplasmic granules.

1. **Neutrophil**
   - The neutrophil has a **multilobed nucleus** consisting of several interconnected lobes.
   - The cytoplasm contains numerous small, pale granules that are less prominent than those of the other granulocytes.
   - The segmented nucleus is the most distinctive feature of this cell type.
2. **Eosinophil**
   - The eosinophil has a **bilobed nucleus** with two connected lobes.
   - The cytoplasm is filled with large, evenly distributed granules.
   - The granules are prominent and occupy much of the space surrounding the nucleus.
3. **Basophil**
   - The basophil contains numerous large granules that densely fill the cytoplasm.
   - The granules partially obscure the nucleus, making its shape difficult to distinguish.
   - The nucleus appears irregularly shaped beneath the granules.

The figure demonstrates the distinguishing microscopic characteristics of the three granulocyte types. **Neutrophils** are identified by a multilobed nucleus, **eosinophils** by a bilobed nucleus and prominent granules, and **basophils** by large dense granules that often obscure the nucleus.

Figure 5.4.3: Thrombopoiesis: Formation of Platelets from Megakaryocytes

The figure illustrates the process of **thrombopoiesis**, the production of **platelets** from myeloid stem cells under the influence of **thrombopoietin**.

1. **Thrombopoietin**, a hormone produced primarily by the **liver and kidneys**, stimulates platelet production in the bone marrow.
2. A **myeloid stem cell** responds to thrombopoietin and differentiates into a **megakaryoblast**.
3. The **megakaryoblast** matures into a **megakaryocyte**, a very large bone marrow cell.
4. The mature megakaryocyte develops long cytoplasmic projections labeled **platelet precursor extensions**.
5. Portions of these extensions fragment and separate from the megakaryocyte.
6. The released fragments become **platelets**, or **thrombocytes**, small cell fragments that circulate in the blood and participate in blood clotting.

The figure demonstrates the developmental pathway of platelet formation. Under stimulation by **thrombopoietin**, myeloid stem cells differentiate into **megakaryoblasts** and then **megakaryocytes**. Cytoplasmic extensions from megakaryocytes fragment to produce platelets, which enter the circulation and contribute to **hemostasis**.

Figure 5.4.4: Major Types of Leukocytes (White Blood Cells)

The figure presents representative micrographs of the five major types of **leukocytes**, or **white blood cells**, allowing comparison of their size, nuclear shape, and cytoplasmic granules.

1. **Basophil**
   - The basophil contains numerous large, dark-staining cytoplasmic granules.
   - The granules densely fill the cell and partially obscure the nucleus.
   - The nucleus is difficult to distinguish because it is covered by the abundant granules.
2. **Eosinophil**
   - The eosinophil contains a bilobed nucleus with two connected lobes.
   - The cytoplasm is filled with large, prominent granules distributed throughout the cell.
   - The granules are readily visible and distinguish the eosinophil from other leukocytes.
3. **Neutrophil**
   - The neutrophil has a segmented, multilobed nucleus composed of several connected lobes.
   - Fine granules are present in the cytoplasm but are less conspicuous than those of eosinophils and basophils.
   - The multilobed nucleus is the most prominent identifying feature.
4. **Monocyte**
   - The monocyte is the largest leukocyte shown.
   - It contains a large nucleus that is kidney-shaped, or horseshoe-shaped.
   - The cytoplasm surrounds the nucleus and lacks the prominent granules seen in granulocytes.
5. **Lymphocyte**
   - The lymphocyte contains a large, round nucleus that occupies most of the cell.
   - Only a thin rim of cytoplasm surrounds the nucleus.
   - The cell appears smaller and more compact than the monocyte.

The figure demonstrates the distinguishing microscopic features of the five major leukocyte types. **Basophils**, **eosinophils**, and **neutrophils** are **granulocytes** characterized by cytoplasmic granules, while **monocytes** and **lymphocytes** are **agranulocytes** distinguished primarily by the shape and size of their nuclei.

Figure 5.5.1: Hemostasis and the Coagulation Cascade

The figure consists of two panels illustrating the process of **hemostasis**, or **blood clotting**, and the **coagulation cascade** that produces a fibrin clot.

Panel (a): General Steps of Clotting

1. **Injury**
   - A blood vessel is damaged, creating an opening in the vessel wall.
   - Blood components, including **erythrocytes**, or **red blood cells**, **white blood cells**, and **platelets**, leak through the break.
2. **Vascular Spasm**
   - Smooth muscle in the vessel wall contracts near the site of injury.
   - Constriction of the blood vessel reduces blood flow and limits blood loss.
3. **Platelet Plug Formation**
   - Platelets are activated by chemicals released from damaged tissue and by exposure to underlying collagen.
   - Activated platelets adhere to the damaged vessel wall and to one another.
   - Bound platelets release additional chemical signals that attract and activate more platelets.
   - As additional platelets accumulate, a **platelet plug** begins to form and enlarge at the site of injury.
4. **Coagulation**
   - The coagulation process converts soluble **fibrinogen** into insoluble **fibrin**.
   - Fibrin strands form a meshwork around the platelet plug.
   - The fibrin network traps platelets, erythrocytes, and other blood components, producing a stable clot that seals the damaged vessel.
   - An inset micrograph shows fibrin strands surrounding and securing blood cells within a clot.

Panel (b): Fibrin Synthesis Cascade

1. The diagram shows the series of clotting factor activations that lead to fibrin formation.
2. The **intrinsic pathway** is initiated by damage to the vessel wall and involves sequential activation of clotting factors **XII, XI, IX, and VIII**.
3. The **extrinsic pathway** is initiated by trauma to tissues outside the blood vessel and involves **tissue factor (III)** and **factor VII**.
4. Both pathways converge at the activation of **factor X**.
5. Activated **factor X**, together with **factor V**, converts **prothrombin (factor II)** into **thrombin (factor IIa)**.
6. Thrombin converts **fibrinogen (factor I)** into **fibrin (factor Ia)**.
7. Activated **factor XIII (factor XIIIa)** cross-links fibrin strands, forming a stable **cross-linked fibrin clot**.
8. The legend indicates that oval symbols represent inactive clotting factors and star-shaped symbols represent activated clotting factors.

The figure demonstrates the body's response to blood vessel injury. Hemostasis begins with vascular constriction and platelet plug formation and is reinforced by the coagulation cascade. The intrinsic and extrinsic pathways converge to generate **thrombin**, which converts **fibrinogen** into **fibrin**. Cross-linked fibrin forms a stable clot that seals the damaged vessel and limits blood loss.

Figure 5.6.1: Rh Incompatibility and Hemolytic Disease of the Newborn

The figure illustrates **Rh incompatibility**, also called **hemolytic disease of the newborn**, and shows how exposure of an **Rh-negative mother** to **Rh-positive fetal blood** can lead to the production of maternal antibodies that affect subsequent **Rh-positive pregnancies**.

Placenta and Separation of Maternal and Fetal Blood

1. The upper left diagram shows the **placenta**, where maternal and fetal circulations are closely associated but normally remain separate.
2. Fetal blood travels through the **umbilical artery and vein** and branches within the placenta.
3. Maternal blood enters through **placental arterioles**, circulates through a **maternal blood pool**, and leaves through **placental venules**.
4. The **embryonic chorion** forms a barrier that normally separates fetal blood from maternal blood while allowing exchange of gases, nutrients, and wastes.

First Exposure: Birth of the First Rh-Positive Infant

1. During the birth of an **Rh-positive infant** to an **Rh-negative mother**, the embryonic chorion may be disrupted, allowing some **Rh-positive fetal erythrocytes** to enter the maternal circulation.
2. The maternal immune system recognizes the **Rh antigen** on the fetal erythrocytes as foreign.
3. Maternal **B lymphocytes** become activated and produce **anti-Rh antibodies**.
4. The first **Rh-positive infant** is usually not significantly affected because antibody production occurs after fetal blood enters the maternal circulation.

Second Exposure: Subsequent Rh-Positive Pregnancy

1. During a later pregnancy with another **Rh-positive fetus**, the mother's **anti-Rh antibody** concentration is already elevated because of the previous exposure.
2. Maternal **anti-Rh antibodies** circulate in the maternal blood and are small enough to cross the placental barrier.
3. These antibodies enter the fetal circulation and bind to **Rh-positive fetal erythrocytes**.
4. Antibody-coated fetal erythrocytes are targeted for destruction, leading to **hemolysis** and reduced numbers of circulating red blood cells in the fetus.

The figure demonstrates the mechanism of **Rh sensitization** and **hemolytic disease of the newborn**. Initial exposure of an **Rh-negative mother** to **Rh-positive fetal blood** stimulates production of **anti-Rh antibodies**. During a subsequent **Rh-positive pregnancy**, these maternal antibodies can cross the placenta and attack fetal erythrocytes, potentially causing **fetal anemia** and other complications.

Figure 5.6.2: ABO and Rh Blood Typing Test Showing A Positive (A+) Blood Type

The figure shows the results of an **ABO and Rh (D) blood typing test** using three wells containing a blood sample mixed with different antibodies: **Anti-A**, **Anti-B**, and **Anti-D**.

1. The **Anti-A** well shows visible **agglutination**, or **clumping**, of red blood cells. The clumped appearance indicates that the red blood cells possess the **A antigen** and have reacted with the **Anti-A** reagent.
2. The **Anti-B** well shows a smooth, uniform appearance with **no agglutination**. This indicates that the red blood cells do not possess the **B antigen** and therefore do not react with the **Anti-B** reagent.
3. The **Anti-D** well shows visible **agglutination** of red blood cells. This reaction indicates the presence of the **Rh (D) antigen** on the red blood cells.
4. The image includes a label identifying the clumped cells in the **Anti-A** well as **agglutinated red blood cells**.
5. Because agglutination occurs in the **Anti-A** and **Anti-D** wells, but not in the **Anti-B** well, the blood sample contains the **A antigen** and the **Rh (D) antigen**, but lacks the **B antigen**.

The figure demonstrates how blood typing is determined by observing agglutination reactions with specific antibodies. The pattern of reactions shown identifies the sample as **blood type A positive, or A-positive**.

Figure 5.6.3: ABO Blood Types, Antigens, Antibodies, and Transfusion Compatibility

The figure summarizes the characteristics of the four major **ABO blood types** by comparing red blood cell antigens, antibodies present in plasma, and compatibility for emergency blood transfusions.

Blood Type A

1. Red blood cells display **A antigens** on their surface.
2. Plasma contains **anti-B antibodies**.
3. Individuals with type A blood can generally receive **type A** or **type O** blood in an emergency transfusion.

Blood Type B

1. Red blood cells display **B antigens** on their surface.
2. Plasma contains **anti-A antibodies**.
3. Individuals with type B blood can generally receive **type B** or **type O** blood in an emergency transfusion.

Blood Type AB

1. Red blood cells display both **A antigens** and **B antigens** on their surface.
2. Plasma contains **neither anti-A nor anti-B antibodies**.
3. Individuals with type AB blood can receive **type A, type B, type AB, or type O** blood in an emergency transfusion.
4. The figure identifies **AB-positive** as the **universal recipient**.

Blood Type O

1. Red blood cells do not display **A** or **B antigens**.
2. Plasma contains both **anti-A** and **anti-B antibodies**.
3. Individuals with type O blood can receive **type O** blood.
4. The figure identifies **type O** as the **universal donor**.

The figure demonstrates the relationship between **ABO blood group antigens** present on red blood cells and the **antibodies** present in plasma. Blood transfusion compatibility depends on avoiding reactions between recipient antibodies and donor red blood cell antigens. **Type A** blood contains **A antigens**, **type B** contains **B antigens**, **type AB** contains both antigens, and **type**

O contains neither antigen. These differences determine transfusion compatibility among the ABO blood groups.

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6: FROM HEART ANATOMY TO CARDIAC OUTPUT

Chapter Overview

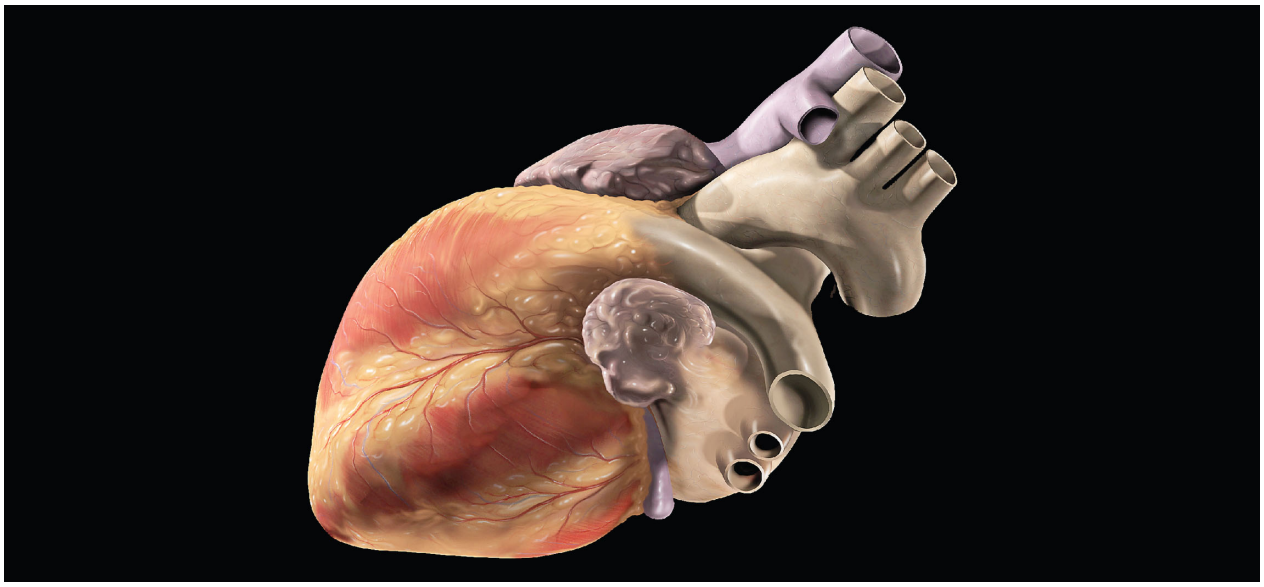


Figure 6.1: Anterior-lateral view of the heart illustrating an abnormal mass within the left atrium adjacent to the pulmonary vein openings. (CC BY 4.0; via LibreTexts).

This chapter explains how the heart is built, how it generates and conducts electrical signals, how it pumps blood through the pulmonary and systemic circuits, and how its activity is regulated and changes over the lifespan. You will connect gross anatomy to microscopic structure, normal electrical activity to the electrocardiogram (ECG), and basic physiology to clinical concepts such as exercise responses and fetal heart development.

External and Internal Anatomy of the Heart

You will learn to identify and describe the external landmarks and internal chambers of the heart, including its position, size, and shape within the thoracic cavity. This section covers the major surfaces and borders of the heart, the great vessels attached to it, and the internal features of the atria and ventricles such as valves, chordae tendineae, and papillary muscles. You will also trace the coronary circulation that supplies blood to the myocardium itself.

Path of Blood Through the Heart and Circulatory Circuits

You will follow the flow of blood through the right and left sides of the heart and through the pulmonary and systemic circuits. Venous blood returns to the right atrium, passes to the right ventricle, and is pumped to the lungs through the pulmonary trunk and arteries. Oxygenated blood returns from the lungs via the pulmonary veins to the left atrium, flows into the left ventricle, and is ejected into the aorta and systemic arteries. Understanding this sequence will help you visualize how oxygen-poor and oxygen-rich blood are kept separate and how valves maintain one-way flow.

Cardiac Muscle and the Conduction System

You will compare cardiac muscle to skeletal and smooth muscle in terms of cell structure, junctions, and electrical properties. Cardiac muscle fibers are branched and interconnected by intercalated discs containing gap junctions and desmosomes, forming a functional syncytium. You will then explore the cardiac conduction system, including the sinoatrial node, atrioventricular node, atrioventricular bundle, bundle branches, and Purkinje fibers, and how these specialized structures generate and coordinate the electrical impulses that time atrial and ventricular contraction.

Electrical Activity and the Electrocardiogram

You will connect intrinsic electrical activity of cardiac cells to the surface electrocardiogram. This section explains how ion movements generate pacemaker and action potentials and how these electrical events appear as the P wave, QRS complex, and T wave on an ECG tracing. You will learn the basic process and purpose of recording an ECG and how it is used clinically to assess heart rhythm, conduction, and some types of ischemia or damage.

The Cardiac Cycle and Cardiac Output

You will examine the sequence of mechanical events in one heartbeat, known as the cardiac cycle. This includes ventricular filling, isovolumetric contraction, ventricular ejection, and isovolumetric relaxation, and how these phases relate to changes in chamber pressure, valve status, and heart sounds. You will learn how to define and calculate cardiac output as the product of heart rate and stroke volume, and how stroke volume is influenced by preload, contractility, and afterload.

Regulation of Heart Rate and the Effects of Exercise

You will study how heart rate and cardiac output are regulated by neural and hormonal mechanisms and by changing physiological demands. This section describes the autonomic centers in the medulla oblongata that control sympathetic and parasympathetic output to the heart, the roles of baroreceptors and chemoreceptors, and the influence of hormones, ions, body temperature, age, and fitness. You will also examine how acute exercise increases heart rate and cardiac output and how long-term training can enhance cardiac efficiency and alter resting heart rate.

Development of the Fetal and Neonatal Heart

You will trace the major stages of heart development, from formation of the primitive heart tube through looping and partitioning into four chambers, and the development of valves and great vessels. This section explains the special features of fetal circulation, including the foramen ovale and ductus arteriosus, which divert blood away from the nonfunctional fetal lungs. You will then learn how these shunts function before birth and what changes occur at and after birth as the lungs expand, pressures shift, and the fetal shunts close or remodel.

By the end of this chapter, you should be able to identify key structures of the heart, describe the path of blood through its chambers and circuits, explain how electrical and mechanical events are coordinated, calculate cardiac output, discuss how the heart responds to neural, hormonal, and exercise-related influences, and outline the basic steps of heart development.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- After completing this chapter, the student will be able to:
- Identify the major external and internal structures of the heart and trace coronary circulation.
- Trace the path of blood through the heart and distinguish between pulmonary and systemic circuits.
- Compare cardiac muscle to other muscle types and outline the components and function of the cardiac conduction system.
- Relate cardiac electrical activity to the main waves of the electrocardiogram (P wave, QRS complex, T wave) and explain the basic clinical use of an ECG.
- Describe the phases of the cardiac cycle and define and calculate cardiac output, including key determinants (heart rate and stroke volume).
- Summarize how autonomic, hormonal, and physiological factors (including exercise) regulate heart rate and cardiac output.
- Outline the major steps of heart development, key features of fetal circulation, and the changes that occur at and after birth.

6.1: Heart Anatomy
6.2: Cardiac Muscle and Electrical Activity
6.3: Cardiac Cycle
6.4: Cardiac Physiology
6.5: Development of the Heart
6.6: Terms and Definitions
6.7: Chapter Review Questions
6.8: Critical Thinking Self-Assessment
6.9: Alternate Text Descriptions

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- 15: Glucose, Glycogen, and Their Metabolic Regulation by Henry Jakubowski and Patricia Flatt has no license indicated.

6.1: Heart Anatomy

Structure and Function of the Heart

The vital importance of the heart is obvious. If one assumes an average rate of contraction of 75 contractions per minute, a human heart would contract approximately 108,000 times in one day, more than 39 million times in one year, and nearly 3 billion times during a 75-year lifespan. Each of the major pumping chambers of the heart ejects approximately 70 mL blood per contraction in a resting adult. This would be equal to 5.25 liters of fluid per minute and approximately 14,000 liters per day. Over one year, that would equal 10,000,000 liters or 2.6 million gallons of blood sent through roughly 60,000 miles of vessels. In order to understand how that happens, it is necessary to understand the anatomy and physiology of the heart.

Location of the Heart

The human heart is located within the thoracic cavity, medially between the lungs in the space known as the mediastinum. Figure 6.1.1 shows the position of the heart within the thoracic cavity. Within the mediastinum, the heart is separated from the other mediastinal structures by a tough membrane known as the pericardium, or pericardial sac, and sits in its own space called the pericardial cavity. The dorsal surface of the heart lies near the bodies of the vertebrae, and its anterior surface sits deep to the sternum and costal cartilages. The great veins, the superior and inferior venae cavae, and the great arteries, the aorta and pulmonary trunk, are attached to the superior surface of the heart, called the base. The base of the heart is located at the level of the third costal cartilage, as seen in Figure 6.1.1. The inferior tip of the heart, the apex, lies just to the left of the sternum between the junction of the fourth and fifth ribs near their articulation with the costal cartilages. The right side of the heart is deflected anteriorly, and the left side is deflected posteriorly. It is important to remember the position and orientation of the heart when placing a stethoscope on the chest of a patient and listening for heart sounds, and also when looking at images taken from a midsagittal perspective. The slight deviation of the apex to the left is reflected in a depression in the medial surface of the inferior lobe of the left lung, called the cardiac notch.

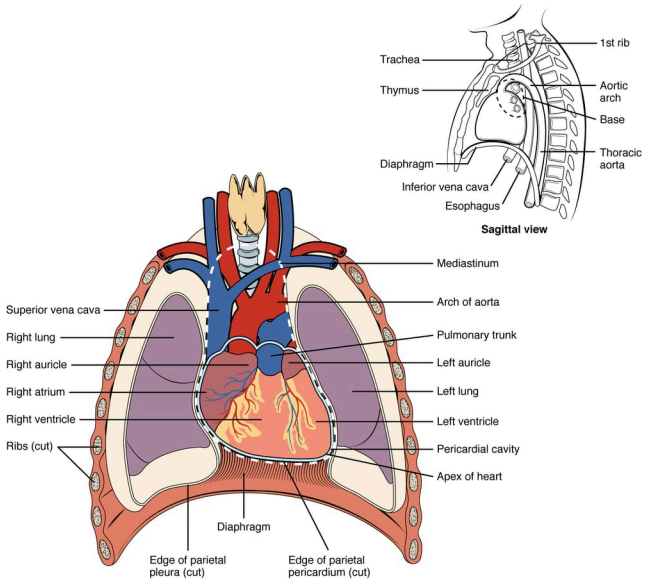


Figure 6.1.1: The heart is located within the thoracic cavity, medially between the lungs in the mediastinum. It is about the size of a fist, is broad at the top, and tapers toward the base. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.1.

Everyday Connection

CPR

The position of the heart in the torso between the vertebrae and sternum (see Figure 6.1.1 for the position of the heart within the thorax) allows for individuals to apply an emergency technique known as cardiopulmonary resuscitation (CPR) if the heart of a patient should stop. By applying pressure with the flat portion of one hand on the sternum in the area between the line at T4 and T9 (Figure 6.1.2), it is possible to manually compress the blood within the heart enough to push some of the blood within it into the pulmonary and systemic circuits. This is particularly critical for the brain, as irreversible damage and death of neurons occur within minutes of loss of blood flow. Current standards call for compression of the chest at least 5 cm deep and at a rate of 100 compressions per minute, a rate equal to the beat in "Staying Alive," recorded in 1977 by the Bee Gees. If you are unfamiliar with this song, a version is available on www.youtube.com. At this stage, the emphasis is on performing high-quality chest compressions, rather than providing artificial respiration. CPR is generally performed until the patient regains spontaneous contraction or is declared dead by an experienced healthcare professional.

When performed by untrained or overzealous individuals, CPR can result in broken ribs or a broken sternum, and can inflict additional severe damage on the patient. It is also possible, if the hands are placed too low on the sternum, to manually drive the xiphoid process into the liver, a consequence that may prove fatal for the patient. Proper training is essential. This proven life-sustaining technique is so valuable that virtually all medical personnel as well as concerned members of the public should be certified and routinely recertified in its application. CPR courses are offered at a variety of locations, including colleges, hospitals, the American Red Cross, and some commercial companies. They normally include practice of the compression technique on a mannequin.

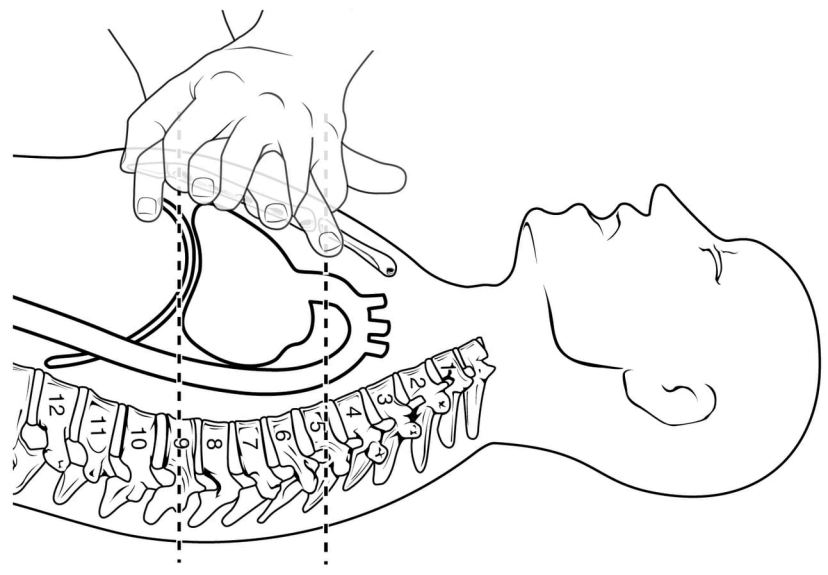


Figure 6.1.2: If the heart should stop, CPR can maintain the flow of blood until the heart resumes beating. By applying pressure to the sternum, the blood within the heart will be squeezed out of the heart and into the circulation. Proper positioning of the hands on the sternum to perform CPR would be between the lines at T4 and T9. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.2.

Interactive Link

Visit the American Heart Association website to help locate a course near your home in the United States. There are also many other national and regional heart associations that offer the same service, depending upon the location.

Shape and Size of the Heart

The shape of the heart is similar to a pinecone, rather broad at the superior surface and tapering to the apex (Figure 6.1.1). A typical heart is approximately the size of your fist: 12 cm (5 in) in length, 8 cm (3.5 in) wide, and 6 cm (2.5 in) in thickness. Given the size difference between most members of the sexes, the weight of a female heart is approximately 250–300 grams (9 to 11 ounces), and the weight of a male heart is approximately 300–350 grams (11 to 12 ounces). The heart of a well-trained athlete, especially one specializing in aerobic sports, can be considerably larger than this. Cardiac muscle responds to exercise in a manner similar to that of skeletal muscle. That is, exercise results in the addition of protein myofilaments that increase the size of the individual cells without increasing their numbers, a concept called hypertrophy. Hearts of athletes can pump blood

more effectively at lower rates than those of nonathletes. Enlarged hearts are not always a result of exercise; they can result from pathologies, such as hypertrophic cardiomyopathy. The cause of an abnormally enlarged heart muscle is unknown, but the condition is often undiagnosed and can cause sudden death in apparently otherwise healthy young people.

Chambers and Circulation through the Heart

The human heart consists of four chambers: The left side and the right side each have one atrium and one ventricle. Each of the upper chambers, the right atrium (plural = atria) and the left atrium, acts as a receiving chamber and contracts to push blood into the lower chambers, the right ventricle and the left ventricle. The ventricles serve as the primary pumping chambers of the heart, propelling blood to the lungs or to the rest of the body.

There are two distinct but linked circuits in the human circulation called the pulmonary and systemic circuits. Although both circuits transport blood and everything it carries, we can initially view the circuits from the point of view of gases. The pulmonary circuit transports blood to and from the lungs, where it picks up oxygen and delivers carbon dioxide for exhalation. The systemic circuit transports oxygenated blood to virtually all of the tissues of the body and returns relatively deoxygenated blood and carbon dioxide to the heart to be sent back to the pulmonary circulation.

The right ventricle pumps deoxygenated blood into the pulmonary trunk, which leads toward the lungs and bifurcates into the left and right pulmonary arteries. These vessels in turn branch many times before reaching the pulmonary capillaries, where gas exchange occurs: Carbon dioxide exits the blood and oxygen enters. The pulmonary trunk arteries and their branches are the only arteries in the post-natal body that carry relatively deoxygenated blood. Highly oxygenated blood returning from the pulmonary capillaries in the lungs passes through a series of vessels that join together to form the pulmonary veins—the only post-natal veins in the body that carry highly oxygenated blood. The pulmonary veins conduct blood into the left atrium, which pumps the blood into the left ventricle, which in turn pumps oxygenated blood into the aorta and on to the many branches of the systemic circuit. Eventually, these vessels will lead to the systemic capillaries, where exchange with the tissue fluid and cells of the body occurs. In this case, oxygen and nutrients exit the systemic capillaries to be used by the cells in their metabolic processes, and carbon dioxide and waste products will enter the blood.

The blood exiting the systemic capillaries is lower in oxygen concentration than when it entered. The capillaries will ultimately unite to form venules, joining to form ever-larger veins, eventually flowing into the two major systemic veins, the superior vena cava and the inferior vena cava, which return blood to the right atrium. The blood in the superior and inferior venae cavae flows into the right atrium, which pumps blood into the right ventricle. This process of blood circulation continues as long as the individual remains alive. Understanding the flow of blood through the pulmonary and systemic circuits is critical to all health professions (Figure 6.1.3).

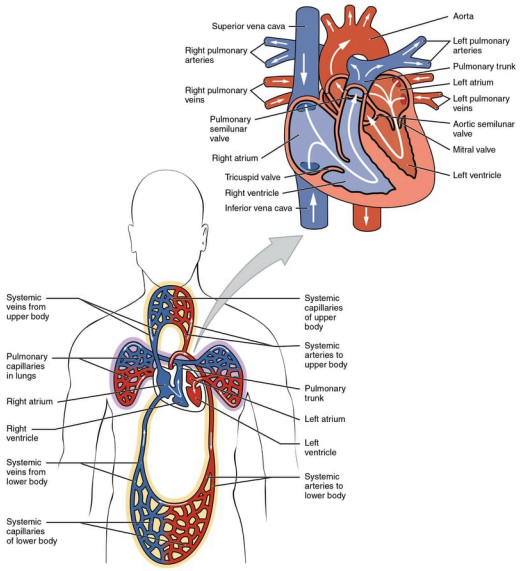


Figure 6.1.3: Blood flows from the right atrium to the right ventricle, where it is pumped into the pulmonary circuit. The blood in the pulmonary artery branches is low in oxygen but relatively high in carbon dioxide. Gas exchange occurs in the pulmonary capillaries (oxygen into the blood, carbon dioxide out), and blood high in oxygen and low in carbon dioxide is returned to the left atrium. From here, blood enters the left ventricle, which pumps it into the systemic circuit. Following exchange in the systemic capillaries (oxygen and nutrients out of the capillaries and carbon dioxide and wastes in), blood returns to the right atrium and the cycle is repeated. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.3.

Membranes, Surface Features, and Layers

Our exploration of more in-depth heart structures begins by examining the membrane that surrounds the heart, the prominent surface features of the heart, and the layers that form the wall of the heart. Each of these components plays its own unique role in terms of function.

Membranes

The membrane that directly surrounds the heart and defines the pericardial cavity is called the pericardium or pericardial sac. It also surrounds the “roots” of the major vessels, or the areas of closest proximity to the heart. The pericardium, which literally translates as “around the heart,” consists of two distinct sublayers: the sturdy outer fibrous pericardium and the inner serous pericardium. The fibrous pericardium is made of tough, dense connective tissue that protects the heart and maintains its position in the thorax. The more delicate serous pericardium consists of two layers: the parietal pericardium, which is fused to the fibrous pericardium, and an inner visceral pericardium, or epicardium, which is fused to the heart and is part of the heart wall. The pericardial cavity, filled with lubricating serous fluid, lies between the epicardium and the pericardium.

In most organs within the body, visceral serous membranes such as the epicardium are microscopic. However, in the case of the heart, it is not a microscopic layer but rather a macroscopic layer, consisting of a simple squamous epithelium called a mesothelium, reinforced with loose, irregular, or areolar connective tissue that attaches to the pericardium. This mesothelium secretes the lubricating serous fluid that fills the pericardial cavity and reduces friction as the heart contracts. Figure 6.1.4 illustrates the pericardial membrane and the layers of the heart.

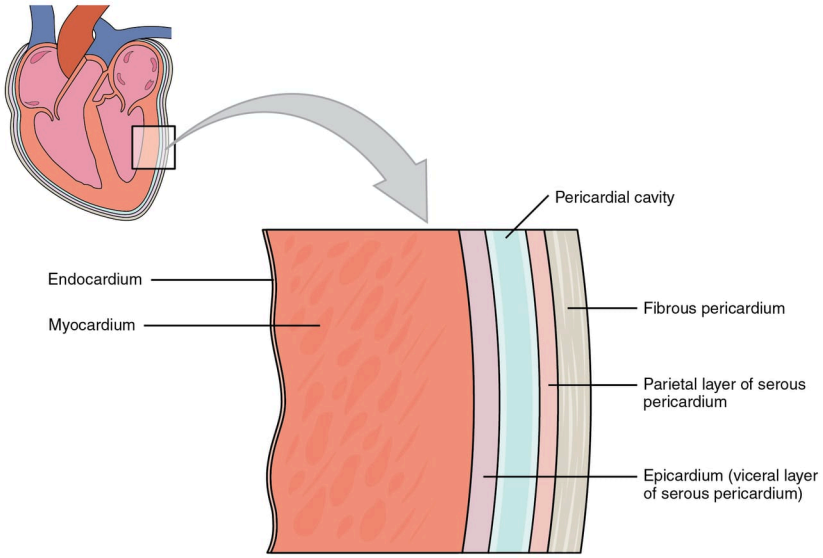


Figure 6.1.4: The pericardial membrane that surrounds the heart consists of three layers and the pericardial cavity. The heart wall also consists of three layers. The pericardial membrane and the heart wall share the epicardium. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.4.

Disorders of the Heart: Cardiac Tamponade

If excess fluid builds within the pericardial space, it can lead to a condition called cardiac tamponade, or pericardial tamponade. With each contraction of the heart, more fluid—in most instances, blood—accumulates within the pericardial cavity. In order to fill with blood for the next contraction, the heart must relax. However, the excess fluid in the pericardial cavity puts pressure on the heart and prevents full relaxation, so the chambers within the heart contain slightly less blood as they begin each heart cycle. Over time, less and less blood is ejected from the heart. If the fluid builds up slowly, as in hypothyroidism, the pericardial cavity may be able to expand gradually to accommodate this extra volume. Some cases of fluid in excess of one liter within the pericardial cavity have been reported. Rapid accumulation of as little as 100 mL of fluid following trauma may trigger cardiac tamponade. Other common causes include myocardial rupture, pericarditis, cancer, or even cardiac surgery. Removal of this excess fluid requires insertion of drainage tubes into the pericardial cavity. Premature removal of these drainage tubes, for example, following cardiac surgery, or clot formation within these tubes are causes of this condition. Untreated, cardiac tamponade can lead to death.

Surface Features of the Heart

Inside the pericardium, the surface features of the heart are visible, including the four chambers. There is a superficial leaf-like extension of the atria near the superior surface of the heart, one on each side, called an auricle—a name that means “ear like”—because its shape resembles the external ear of a human (Figure 6.1.5). Auricles are relatively thin-walled structures that can fill with blood and empty into the atria or upper chambers of the heart. You may also hear them referred to as atrial appendages. Also prominent is a series of fat-filled grooves, each of which is known as a sulcus (plural = sulci), along the superior surfaces of the heart. Major coronary blood vessels are located in these sulci. The deep coronary sulcus is located between the atria and ventricles. Located between the left and right ventricles are two additional sulci that are not as deep as the coronary sulcus. The anterior interventricular sulcus is visible on the anterior surface of the heart, whereas the posterior interventricular sulcus is visible on the posterior surface of the heart. Figure 6.1.5 illustrates anterior and posterior views of the surface of the heart.

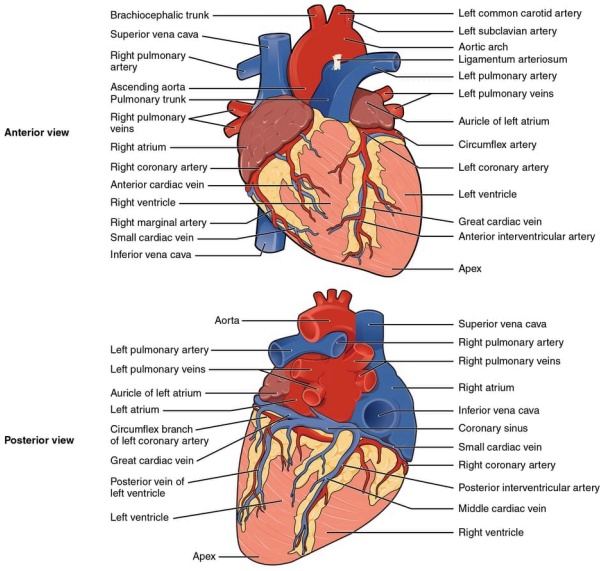


Figure 6.1.5: Inside the pericardium, the surface features of the heart are visible. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.5.

Layers

The wall of the heart is composed of three layers of unequal thickness. From superficial to deep, these are the epicardium, the myocardium, and the endocardium (see Figure 6.1.4). The outermost layer of the wall of the heart is also the innermost layer of the pericardium, the epicardium, or the visceral pericardium discussed earlier.

The middle and thickest layer is the myocardium, made largely of cardiac muscle cells. It is built upon a framework of collagenous fibers, plus the blood vessels that supply the myocardium and the nerve fibers that help regulate the heart. It is the contraction of the myocardium that pumps blood through the heart and into the major arteries. The muscle pattern is elegant and complex, as the muscle cells swirl and spiral around the chambers of the heart. They form a figure 8 pattern around the atria and around the bases of the great vessels. Deeper ventricular muscles also form a figure 8 around the two ventricles and proceed toward the apex. More superficial layers of ventricular muscle wrap around both ventricles. This complex swirling pattern allows the heart to pump blood more effectively than a simple linear pattern would. Figure 6.1.6 illustrates the arrangement of muscle cells.

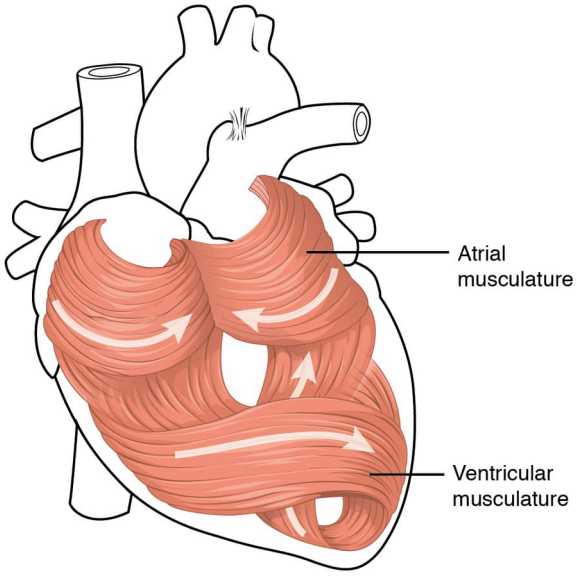


Figure 6.1.6: The swirling pattern of cardiac muscle tissue contributes significantly to the heart's ability to pump blood effectively. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.6.

Although the ventricles on the right and left sides pump the same amount of blood per contraction, the muscle of the left ventricle is much thicker and better developed than that of the right ventricle. In order to overcome the high resistance required to pump blood into the long systemic circuit, the left ventricle must generate a great amount of pressure. The right ventricle does not need to generate as much pressure, since the pulmonary circuit is shorter and provides less resistance. Figure 6.1.7 illustrates the differences in muscular thickness needed for each of the ventricles.

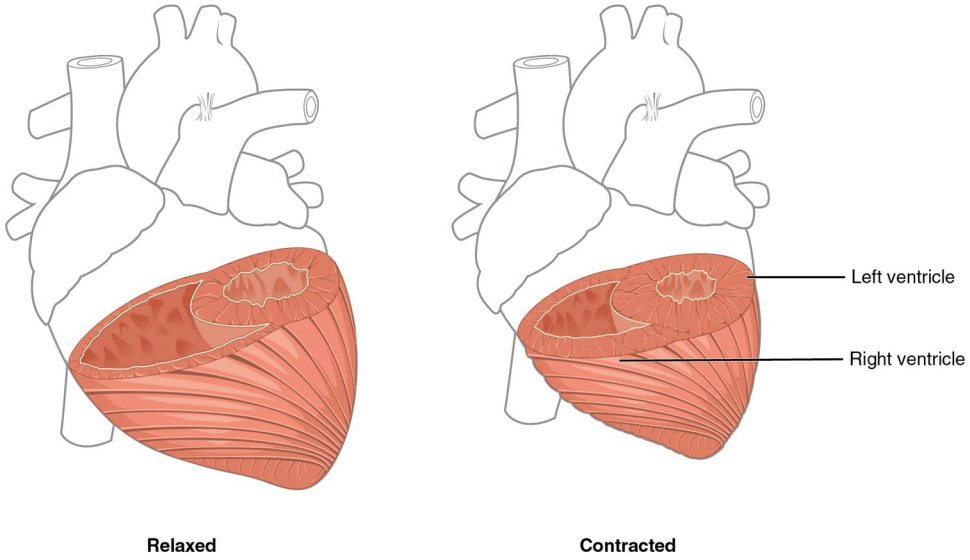


Figure 6.1.7: The myocardium in the left ventricle is significantly thicker than that of the right ventricle. Both ventricles pump the same amount of blood, but the left ventricle must generate a much greater pressure to overcome greater resistance in the systemic circuit. The ventricles are shown in both relaxed and contracting states. Note the differences in the relative size of the lumens, the region inside each ventricle where the blood is contained. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.7.

The innermost layer of the heart wall, the endocardium, is joined to the myocardium with a thin layer of connective tissue. The endocardium lines the chambers where the blood circulates and covers the heart valves. It is made of simple squamous epithelium called endothelium, which is continuous with the endothelial lining of the blood vessels.

Once regarded as a simple lining layer, recent evidence indicates that the endothelium of the endocardium and the coronary capillaries may play active roles in regulating the contraction of the muscle within the myocardium. The endothelium may also regulate the growth patterns of the cardiac muscle cells throughout life, and the endothelins it secretes create an environment in the surrounding tissue fluids that regulates ionic concentrations and states of contractility. Endothelins are potent vasoconstrictors and, in a normal individual, establish a homeostatic balance with other vasoconstrictors and vasodilators.

Internal Structure of the Heart

Recall that the heart's contraction cycle follows a dual pattern of circulation—the pulmonary and systemic circuits—because of the pairs of chambers that pump blood into the circulation. In order to develop a more precise understanding of cardiac function, it is first necessary to explore the internal anatomical structures in more detail.

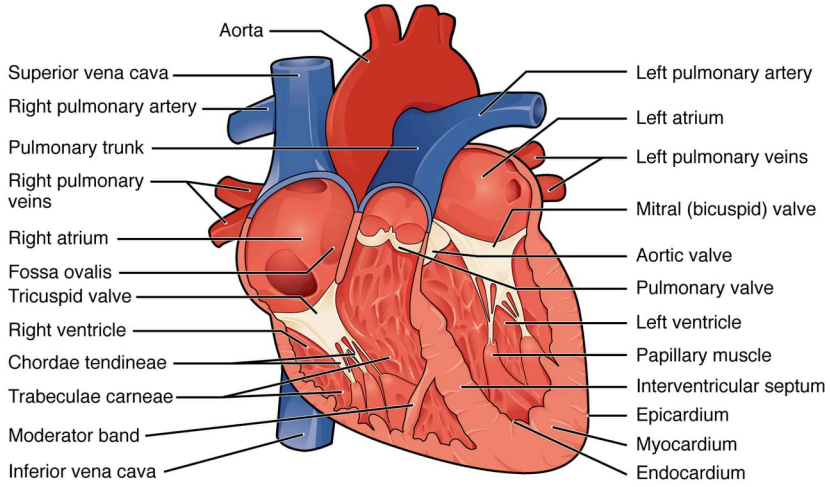
Septa of the Heart

The word septum is derived from the Latin for "something that encloses;" in this case, a septum (plural = septa) refers to a wall or partition that divides the heart into chambers. The septa are physical extensions of the myocardium lined with endocardium. Located between the two atria is the interatrial septum. Normally in an adult heart, the interatrial septum bears an oval-shaped depression known as the fossa ovalis, a remnant of an opening in the fetal heart known as the foramen ovale. The foramen ovale allowed blood in the fetal heart to pass directly from the right atrium to the left atrium, allowing some blood to bypass the pulmonary circuit. Within seconds after birth, a flap of tissue known as the septum primum that previously acted as a valve closes the foramen ovale and establishes the typical cardiac circulation pattern.

Between the two ventricles is a second septum known as the interventricular septum. Unlike the interatrial septum, the interventricular septum is normally intact after its formation during fetal development. It is substantially thicker than the interatrial

septum, since the ventricles generate far greater pressure when they contract.

The septum between the atria and ventricles is known as the atrioventricular septum. It is marked by the presence of four openings that allow blood to move from the atria into the ventricles and from the ventricles into the pulmonary trunk and aorta. Located in each of these openings between the atria and ventricles is a valve, a specialized structure that ensures one-way flow of blood. The valves between the atria and ventricles are known generically as atrioventricular valves. The valves at the openings that lead to the pulmonary trunk and aorta are known generically as semilunar valves. The interventricular septum is visible in Figure 6.1.8. In this figure, the atrioventricular septum has been removed to better show the bicuspid and tricuspid valves; the interatrial septum is not visible, since its location is covered by the aorta and pulmonary trunk. Since these openings and valves structurally weaken the atrioventricular septum, the remaining tissue is heavily reinforced with dense connective tissue called the cardiac skeleton, or skeleton of the heart. It includes four rings that surround the openings between the atria and ventricles, and the openings to the pulmonary trunk and aorta, and serve as the point of attachment for the heart valves. The cardiac skeleton also provides an important boundary in the heart electrical conduction system.



Anterior view

Figure 6.1.8: This anterior view of the heart shows the four chambers, the major vessels and their early branches, as well as the valves. The presence of the pulmonary trunk and aorta covers the interatrial septum, and the atrioventricular septum is cut away to show the atrioventricular valves. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.8.

Disorders of the Heart: Heart Defects

One very common form of interatrial septum pathology is patent foramen ovale, which occurs when the septum primum does not close at birth, and the fossa ovalis is unable to fuse. The word patent is from the Latin root patens for “open.” It may be benign or asymptomatic, perhaps never being diagnosed, or in extreme cases, it may require surgical repair to close the opening permanently. As much as 20–25 percent of the general population may have a patent foramen ovale, but fortunately, most have the benign, asymptomatic version. Patent foramen ovale is normally detected by auscultation of a heart murmur (an abnormal heart sound) and confirmed by imaging with an echocardiogram. Despite its prevalence in the general population, the causes of patent ovale are unknown, and there are no known risk factors. In nonlife-threatening cases, it is better to monitor the condition than to risk heart surgery to repair and seal the opening.

Coarctation of the aorta is a congenital abnormal narrowing of the aorta that is normally located at the insertion of the ligamentum arteriosum, the remnant of the fetal shunt called the ductus arteriosus. If severe, this condition drastically restricts blood flow through the primary systemic artery, which is life threatening. In some individuals, the condition may be fairly benign and not detected until later in life. Detectable symptoms in an infant include difficulty breathing, poor appetite, trouble feeding, or failure to thrive. In older individuals, symptoms include dizziness, fainting, shortness of breath, chest pain, fatigue, headache, and nosebleeds. Treatment involves surgery to resect (remove) the affected region or angioplasty to open the abnormally narrow passageway. Studies have shown that the earlier the surgery is performed, the better the chance of survival.

A patent ductus arteriosus is a congenital condition in which the ductus arteriosus fails to close. The condition may range from severe to benign. Failure of the ductus arteriosus to close results in blood flowing from the higher pressure aorta into the lower pressure pulmonary trunk. This additional fluid moving toward the lungs increases pulmonary pressure and makes respiration difficult. Symptoms include shortness of breath (dyspnea), tachycardia, enlarged heart, a widened pulse pressure, and poor weight gain in infants. Treatments include surgical closure (ligation), manual closure using platinum coils or specialized mesh inserted via the femoral artery or vein, or nonsteroidal anti-inflammatory drugs to block the synthesis of prostaglandin E2, which maintains the vessel in an open position. If untreated, the condition can result in congestive heart failure.

Septal defects are not uncommon in individuals and may be congenital or caused by various disease processes. Tetralogy of Fallot is a congenital condition that may also occur from exposure to unknown environmental factors; it occurs when there is an opening in the interventricular septum caused by blockage of the pulmonary trunk, normally at the pulmonary semilunar valve. This allows blood that is relatively low in oxygen from the right ventricle to flow into the left ventricle and mix with the blood that is relatively high in oxygen. Symptoms include a distinct heart murmur, low blood oxygen percent saturation, dyspnea or difficulty in breathing, polycythemia, broadening (clubbing) of the fingers and toes, and in children, difficulty in feeding or failure to grow and develop. It is the most common cause of cyanosis following birth. The term “tetralogy” is derived from the four components of the condition, although only three may be present in an individual patient: pulmonary infundibular stenosis (rigidity of the pulmonary valve), overriding aorta (the aorta is shifted above both ventricles), ventricular septal defect (opening), and right ventricular hypertrophy (enlargement of the right ventricle). Other heart defects may also accompany this condition, which is typically confirmed by echocardiography imaging. Tetralogy of Fallot occurs in approximately 400 out of one million live births. Normal treatment involves extensive surgical repair, including the use of stents to redirect blood flow and replacement of valves and patches to repair the septal defect, but the condition has a relatively high mortality. Survival rates are currently 75 percent during the first year of life; 60 percent by 4 years of age; 30 percent by 10 years; and 5 percent by 40 years.

In the case of severe septal defects, including both tetralogy of Fallot and patent foramen ovale, failure of the heart to develop properly can lead to a condition commonly known as a “blue baby.” Regardless of normal skin pigmentation, individuals with this condition have an insufficient supply of oxygenated blood, which leads to cyanosis, a blue or purple coloration of the skin, especially when active.

Septal defects are commonly first detected through auscultation, listening to the chest using a stethoscope. In this case, instead of hearing normal heart sounds attributed to the flow of blood and closing of heart valves, unusual heart sounds may be detected. This is often followed by medical imaging to confirm or rule out a diagnosis. In many cases, treatment may not be needed. Some common congenital heart defects are illustrated in Figure 6.1.9.

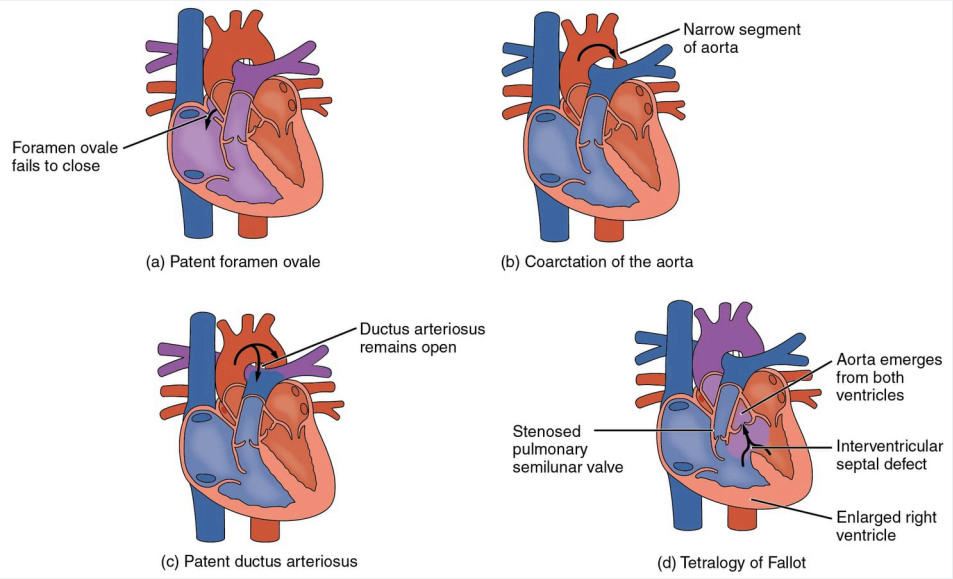


Figure 6.1.9: Congenital Heart Defects (a) A patent foramen ovale defect is an abnormal opening in the interatrial septum, or more commonly, a failure of the foramen ovale to close. (b) Coarctation of the aorta is an abnormal narrowing of the aorta. (c) A patent ductus arteriosus is the failure of the ductus arteriosus to close. (d) Tetralogy of Fallot includes an abnormal opening in the interventricular septum. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.9.

Right Atrium

The right atrium serves as the receiving chamber for blood returning to the heart from the systemic circulation. The two major systemic veins, the superior and inferior venae cavae, and the large coronary vein called the coronary sinus that drains the heart myocardium empty into the right atrium. The superior vena cava drains blood from regions superior to the diaphragm: the head, neck, upper limbs, and the thoracic region. It empties into the superior and posterior portions of the right atrium. The inferior vena cava drains blood from areas inferior to the diaphragm: the lower limbs and abdominopelvic region of the body. It, too, empties into the posterior portion of the atria, but inferior to the opening of the superior vena cava. Immediately superior and slightly medial to the opening of the inferior vena cava on the posterior surface of the atrium is the opening of the coronary sinus. This thin-walled vessel drains most of the coronary veins that return systemic blood from the heart. The majority of the internal heart structures discussed in this and subsequent sections are illustrated in Figure 6.1.8.

While the bulk of the internal surface of the right atrium is smooth, the depression of the fossa ovalis is medial, and the anterior surface demonstrates prominent ridges of muscle called the pectinate muscles. The right auricle also has pectinate muscles. The left atrium does not have pectinate muscles except in the auricle.

The atria receive venous blood on a nearly continuous basis, preventing venous flow from stopping while the ventricles are contracting. While most ventricular filling occurs while the atria are relaxed, they do demonstrate a contractile phase and actively pump blood into the ventricles just prior to ventricular contraction. The opening between the atrium and ventricle is guarded by the tricuspid valve.

Right Ventricle

The right ventricle receives blood from the right atrium through the tricuspid valve. Each flap of the valve is attached to strong strands of connective tissue, the chordae tendineae, literally “tendinous cords,” or sometimes more poetically referred to as “heart strings.” There are several chordae tendineae associated with each of the flaps. They are composed of approximately 80 percent collagenous fibers with the remainder consisting of elastic fibers and endothelium. They connect each of the flaps to a papillary muscle that extends from the inferior ventricular surface. There are three papillary muscles in the right ventricle, called the anterior, posterior, and septal muscles, which correspond to the three sections of the valves.

When the myocardium of the ventricle contracts, pressure within the ventricular chamber rises. Blood, like any fluid, flows from higher pressure to lower pressure areas, in this case, toward the pulmonary trunk and the atrium. To prevent any potential backflow, the papillary muscles also contract, generating tension on the chordae tendineae. This prevents the flaps of the valves from being forced into the atria and regurgitation of the blood back into the atria during ventricular contraction. Figure 6.1.10 shows papillary muscles and chordae tendineae attached to the tricuspid valve.

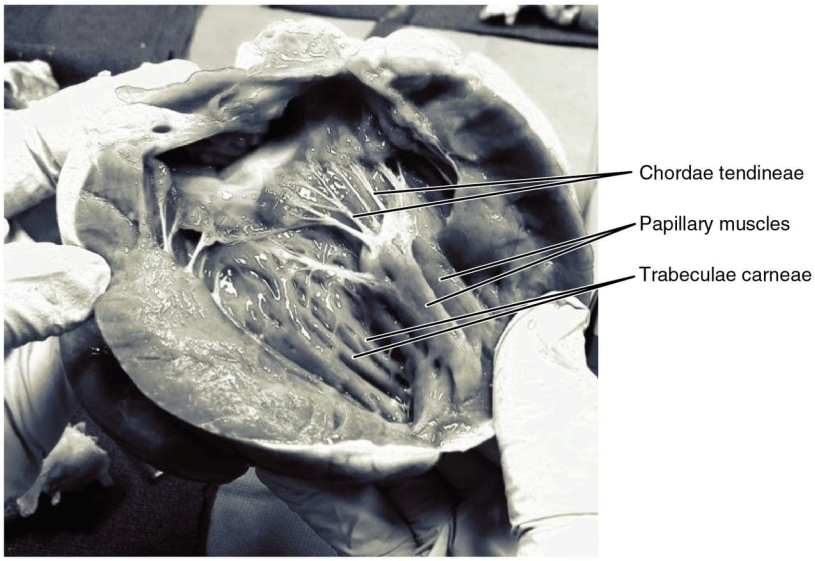


Figure 6.1.10: In this frontal section, you can see papillary muscles attached to the tricuspid valve on the right as well as the mitral valve on the left via chordae tendineae. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.10.

The walls of the ventricle are lined with trabeculae carneae, ridges of cardiac muscle covered by endocardium. In addition to these muscular ridges, a band of cardiac muscle, also covered by endocardium, known as the moderator band reinforces the thin walls of the right ventricle and plays a crucial role in cardiac conduction. It arises from the inferior portion of the interventricular septum and crosses the interior space of the right ventricle to connect with the inferior papillary muscle.

When the right ventricle contracts, it ejects blood into the pulmonary trunk, which branches into the left and right pulmonary arteries that carry it to each lung. The superior surface of the right ventricle begins to taper as it approaches the pulmonary trunk. At the base of the pulmonary trunk is the pulmonary semilunar valve that prevents backflow from the pulmonary trunk.

Left Atrium

After exchange of gases in the pulmonary capillaries, blood returns to the left atrium high in oxygen via one of the four pulmonary veins. While the left atrium does not contain pectinate muscles, it does have an auricle that includes these pectinate ridges. Blood flows nearly continuously from the pulmonary veins back into the atrium, which acts as the receiving chamber, and from here through an opening into the left ventricle. Most blood flows passively into the heart while both the atria and ventricles are relaxed, but toward the end of the ventricular relaxation period, the left atrium will contract, pumping blood into the ventricle. This atrial contraction accounts for approximately 20 percent of ventricular filling. The opening between the left atrium and ventricle is guarded by the mitral valve.

Left Ventricle

Recall that, although both sides of the heart will pump the same amount of blood, the muscular layer is much thicker in the left ventricle compared to the right (see Figure 6.1.7). Like the right ventricle, the left also has trabeculae carneae, but there is no moderator band. The mitral valve is connected to papillary muscles via chordae tendineae. There are two papillary muscles on the left—the anterior and posterior—as opposed to three on the right.

The left ventricle is the major pumping chamber for the systemic circuit; it ejects blood into the aorta through the aortic semilunar valve.

Heart Valve Structure and Function

A transverse section through the heart slightly above the level of the atrioventricular septum reveals all four heart valves along the same plane (Figure 6.1.11). The valves ensure unidirectional blood flow through the heart. Between the right atrium and the right ventricle is the right atrioventricular valve, or tricuspid valve. It typically consists of three flaps, or leaflets, made of endocardium reinforced with additional connective tissue. The flaps are connected by chordae tendineae to the papillary muscles, which control the opening and closing of the valves.

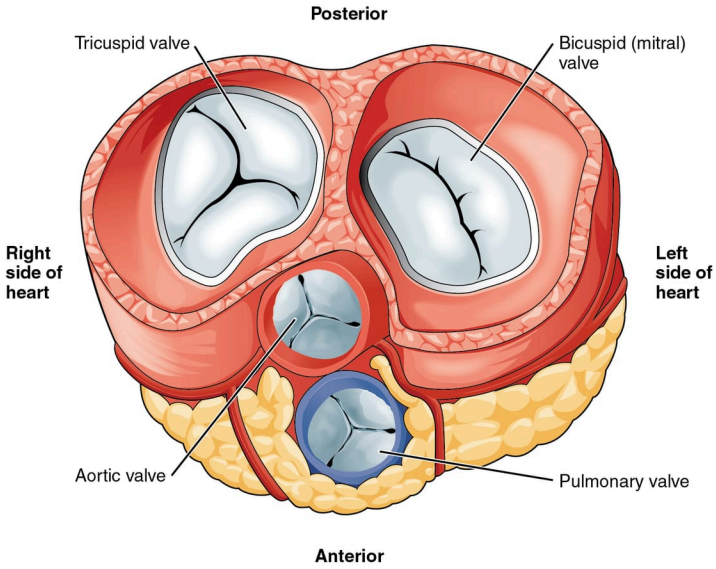


Figure 6.1.11: With the atria and major vessels removed, all four valves are clearly visible, although it is difficult to distinguish the three separate cusps of the tricuspid valve. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.11.

Emerging from the right ventricle at the base of the pulmonary trunk is the pulmonary semilunar valve, or the pulmonary valve; it is also known as the pulmonic valve or the right semilunar valve. The pulmonary valve is comprised of three small flaps of endothelium reinforced with connective tissue. When the ventricle relaxes, the pressure differential causes blood to flow back into the ventricle from the pulmonary trunk. This flow of blood fills the pocket-like flaps of the pulmonary valve, causing the valve to close and producing an audible sound. Unlike the atrioventricular valves, there are no papillary muscles or chordae tendineae associated with the pulmonary valve.

Located at the opening between the left atrium and left ventricle is the mitral valve, also called the bicuspid valve or the left atrioventricular valve. Structurally, this valve consists of two cusps, compared to the three cusps of the tricuspid valve. In a clinical setting, the valve is referred to as the mitral valve, rather than the bicuspid valve. The two cusps of the mitral valve are attached by chordae tendineae to two papillary muscles that project from the wall of the ventricle.

At the base of the aorta is the aortic semilunar valve, or the aortic valve, which prevents backflow from the aorta. It normally is composed of three flaps. When the ventricle relaxes and blood attempts to flow back into the ventricle from the aorta, blood will fill the cusps of the valve, causing it to close and producing an audible sound.

In Figure 6.1.12a, the two atrioventricular valves are open and the two semilunar valves are closed. This occurs when both atria and ventricles are relaxed and when the atria contract to pump blood into the ventricles. Figure 6.1.12b shows a frontal view. Although only the left side of the heart is illustrated, the process is virtually identical on the right.

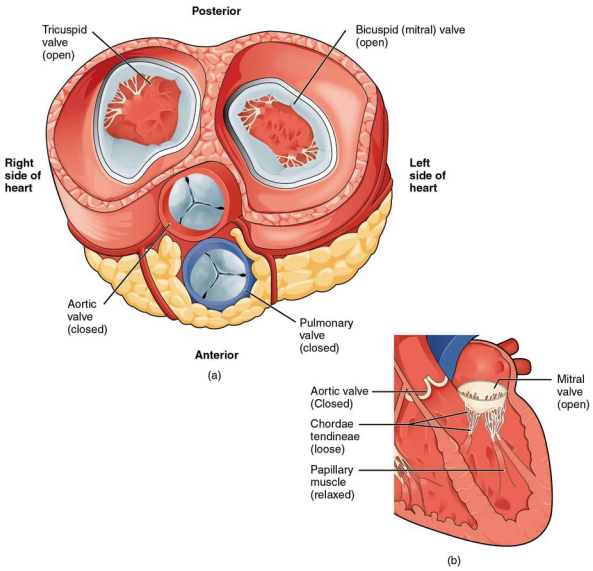


Figure 6.1.12: (a) A transverse section through the heart illustrates the four heart valves. The two atrioventricular valves are open; the two semilunar valves are closed. The atria and vessels have been removed. (b) A frontal section through the heart illustrates blood flow through the mitral valve. When the mitral valve is open, it allows blood to move from the left atrium to the left ventricle. The aortic semilunar valve is closed to prevent backflow of blood from the aorta to the left ventricle. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.12.

Figure 6.1.13a shows the atrioventricular valves closed while the two semilunar valves are open. This occurs when the ventricles contract to eject blood into the pulmonary trunk and aorta. Closure of the two atrioventricular valves prevents blood from being forced back into the atria. This stage can be seen from a frontal view in Figure 6.1.13b.

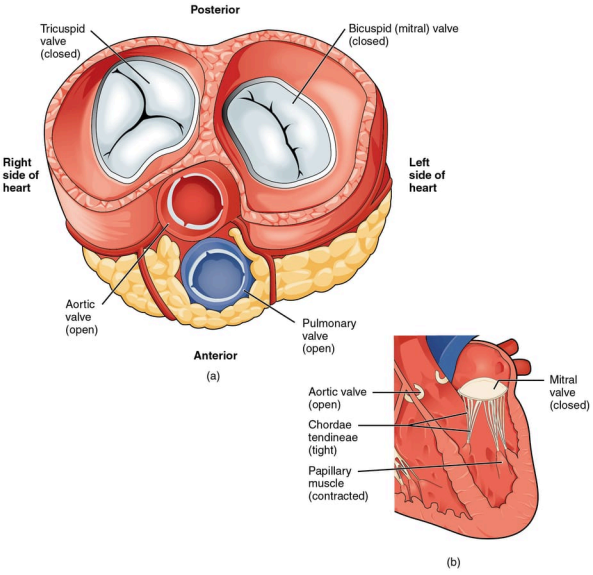


Figure 6.1.13: (a) A transverse section through the heart illustrates the four heart valves during ventricular contraction. The two atrioventricular valves are closed, but the two semilunar valves are open. The atria and vessels have been removed. (b) A frontal view shows the closed mitral (bicuspid) valve that prevents backflow of blood into the left atrium. The aortic semilunar valve is open to allow blood to be ejected into the aorta. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.13.

When the ventricles begin to contract, pressure within the ventricles rises and blood flows toward the area of lowest pressure, which is initially in the atria. This backflow causes the cusps of the tricuspid and mitral (bicuspid) valves to close. These valves are tied down to the papillary muscles by chordae tendineae. During the relaxation phase of the cardiac cycle, the papillary muscles are also relaxed and the tension on the chordae tendineae is slight (Figure 6.1.14a). However, as the myocardium of the ventricle contracts, so do the papillary muscles. This creates tension on the chordae tendineae (Figure 6.1.14b), helping to hold the cusps of the atrioventricular valves in place and preventing them from being blown back into the atria.

The aortic and pulmonary semilunar valves lack the chordae tendineae and papillary muscles associated with the atrioventricular valves. Instead, they consist of pocket-like folds of endocardium reinforced with additional connective tissue. When the ventricles relax and the change in pressure forces the blood toward the ventricles, the blood presses against these cusps and seals the openings.

Disorders of the Heart Valves

When heart valves do not function properly, they are often described as incompetent and result in valvular heart disease, which can range from benign to lethal. Some of these conditions are congenital, that is, the individual was born with the defect, whereas others may be attributed to disease processes or trauma. Some malfunctions are treated with medications, others require surgery, and still others may be mild enough that the condition is merely monitored since treatment might trigger more serious consequences.

Valvular disorders are often caused by carditis, or inflammation of the heart. One common trigger for this inflammation is rheumatic fever, or scarlet fever, an autoimmune response to the presence of a bacterium, *Streptococcus pyogenes*, normally a disease of childhood.

While any of the heart valves may be involved in valve disorders, mitral regurgitation is the most common, detected in approximately 2 percent of the population, and the pulmonary semilunar valve is the least frequently involved. When a valve malfunctions, the flow of blood to a region will often be disrupted. The resulting inadequate flow of blood to this region will be described in general terms as an insufficiency. The specific type of insufficiency is named for the valve involved: aortic insufficiency, mitral insufficiency, tricuspid insufficiency, or pulmonary insufficiency.

If one of the cusps of the valve is forced backward by the force of the blood, the condition is referred to as a prolapsed valve. Prolapse may occur if the chordae tendineae are damaged or broken, causing the closure mechanism to fail. The failure of the valve to close properly disrupts the normal one-way flow of blood and results in regurgitation, when the blood flows backward from its normal path. Using a stethoscope, the disruption to the normal flow of blood produces a heart murmur.

Stenosis is a condition in which the heart valves become rigid and may calcify over time. The loss of flexibility of the valve interferes with normal function and may cause the heart to work harder to propel blood through the valve, which eventually weakens the heart. Aortic stenosis affects approximately 2 percent of the population over 65 years of age, and the percentage increases to approximately 4 percent in individuals over 85 years. Occasionally, one or more of the chordae tendineae will tear or the papillary muscle itself may die as a component of a myocardial infarction (heart attack). In this case, the patient's condition will deteriorate dramatically and rapidly, and immediate surgical intervention may be required.

Auscultation, or listening to a patient's heart sounds, is one of the most useful diagnostic tools, since it is proven, safe, and inexpensive. The term auscultation is derived from the Latin for "to listen," and the technique has been used for diagnostic purposes as far back as the ancient Egyptians. Valve and septal disorders will trigger abnormal heart sounds. If a valvular disorder is detected or suspected, a test called an echocardiogram, or simply an "echo," may be ordered. Echocardiograms are sonograms of the heart and can help in the diagnosis of valve disorders as well as a wide variety of heart pathologies.

Career Connection

Cardiologist

Cardiologists are medical doctors that specialize in the diagnosis and treatment of diseases of the heart. After completing 4 years of medical school, cardiologists complete a three-year residency in internal medicine followed by an additional three or more years in cardiology. Following this 10-year period of medical training and clinical experience, they qualify for a rigorous two-day examination administered by the Board of Internal Medicine that tests their academic training and clinical abilities, including diagnostics and treatment. After successful completion of this examination, a physician becomes a board-certified cardiologist. Some board-certified cardiologists may be invited to become a Fellow of the American College of Cardiology (FACC). This professional recognition is awarded to outstanding physicians based upon merit, including outstanding credentials, achievements, and community contributions to cardiovascular medicine.

Career Connection

Cardiovascular Technologist/Technician

Cardiovascular technologists/technicians are trained professionals who perform a variety of imaging techniques, such as sonograms or echocardiograms, used by physicians to diagnose and treat diseases of the heart. Nearly all of these positions require an associate degree, and these technicians earn a median salary of $49,410 as of May 2010, according to the U.S. Bureau of Labor Statistics. Growth within the field is fast, projected at 29 percent from 2010 to 2020.

There is a considerable overlap and complementary skills between cardiac technicians and vascular technicians, and so the term cardiovascular technician is often used. Special certifications within the field require documenting appropriate experience and completing additional and often expensive certification examinations. These subspecialties include Certified Rhythm Analysis Technician (CRAT), Certified Cardiographic Technician (CCT), Registered Congenital Cardiac Sonographer (RCCS), Registered Cardiac Electrophysiology Specialist (RCES), Registered Cardiovascular Invasive Specialist (RCIS), Registered Cardiac Sonographer (RCS), Registered Vascular Specialist (RVS), and Registered Phlebology Sonographer (RPhS).

Coronary Circulation

You will recall that the heart is a remarkable pump composed largely of cardiac muscle cells that are incredibly active throughout life. Like all other cells, a cardiomyocyte requires a reliable supply of oxygen and nutrients, and a way to remove wastes, so it needs a dedicated, complex, and extensive coronary circulation. And because of the critical and nearly ceaseless activity of the heart throughout life, this need for a blood supply is even greater than for a typical cell. However, coronary circulation is not continuous; rather, it cycles, reaching a peak when the heart muscle is relaxed and nearly ceasing while it is contracting.

Coronary Arteries

Coronary arteries supply blood to the myocardium and other components of the heart. The first portion of the aorta after it arises from the left ventricle gives rise to the coronary arteries. There are three dilations in the wall of the aorta just superior to the aortic semilunar valve. Two of these, the left posterior aortic sinus and anterior aortic sinus, give rise to the left and right coronary arteries, respectively. The third sinus, the right posterior aortic sinus, typically does not give rise to a vessel. Coronary vessel branches that remain on the surface of the artery and follow the sulci are called epicardial coronary arteries.

The left coronary artery distributes blood to the left side of the heart, the left atrium and ventricle, and the interventricular septum. The circumflex artery arises from the left coronary artery and follows the coronary sulcus to the left. Eventually, it will fuse with the small branches of the right coronary artery. The larger anterior interventricular artery, also known as the left anterior descending artery (LAD), is the second major branch arising from the left coronary artery. It follows the anterior interventricular sulcus around the pulmonary trunk. Along the way it gives rise to numerous smaller branches that interconnect with the branches of the posterior interventricular artery, forming anastomoses. An anastomosis is an area where vessels unite to form interconnections that normally allow blood to circulate to a region even if there may be partial blockage in another branch. The anastomoses in the heart are very small. Therefore, this ability is somewhat restricted in the heart so a coronary artery blockage often results in death of the cells (myocardial infarction) supplied by the particular vessel.

The right coronary artery proceeds along the coronary sulcus and distributes blood to the right atrium, portions of both ventricles, and the heart conduction system. Normally, one or more marginal arteries arise from the right coronary artery inferior to the right atrium. The marginal arteries supply blood to the superficial portions of the right ventricle. On the posterior surface of the heart, the right coronary artery gives rise to the posterior interventricular artery, also known as the posterior descending artery. It runs along the posterior portion of the interventricular sulcus toward the apex of the heart, giving rise to branches that supply the interventricular septum and portions of both ventricles. Figure 6.1.14 presents views of the coronary circulation from both the anterior and posterior views.

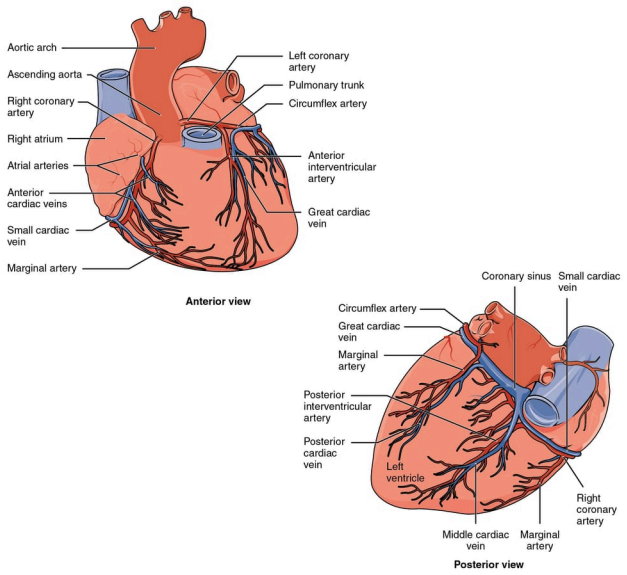


Figure 6.1.14: The anterior view of the heart shows the prominent coronary surface vessels. The posterior view of the heart shows the prominent coronary surface vessels. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.14.

Diseases of the Heart: Myocardial Infraction

Myocardial infarction (MI) is the formal term for what is commonly referred to as a heart attack. It normally results from a lack of blood flow (ischemia) and oxygen (hypoxia) to a region of the heart, resulting in death of the cardiac muscle cells. An MI often occurs when a coronary artery is blocked by the buildup of atherosclerotic plaque consisting of lipids, cholesterol and fatty acids, and white blood cells, primarily macrophages. It can also occur when a portion of an unstable atherosclerotic plaque travels through the coronary arterial system and lodges in one of the smaller vessels. The resulting blockage restricts the flow of blood and oxygen to the myocardium and causes death of the tissue. MIs may be triggered by excessive exercise, in which the partially occluded artery is no longer able to pump sufficient quantities of blood, or severe stress, which may induce spasm of the smooth muscle in the walls of the vessel.

In the case of acute MI, there is often sudden pain beneath the sternum (retrosternal pain) called angina pectoris, often radiating down the left arm in males but not in female patients. Until this anomaly between the sexes was discovered, many female patients suffering MIs were misdiagnosed and sent home. In addition, patients typically present with difficulty breathing and shortness of breath (dyspnea), irregular heartbeat (palpations), nausea and vomiting, sweating (diaphoresis), anxiety, and fainting (syncope), although not all of these symptoms may be present. Many of the symptoms are shared with other medical conditions, including anxiety attacks and simple indigestion, so differential diagnosis is critical. It is estimated that between 22 and 64 percent of MIs present without any symptoms.

An MI can be confirmed by examining the patient's ECG, which frequently reveals alterations in the ST and Q components. Some classification schemes of MI are referred to as ST-elevated MI (STEMI) and non-elevated MI (non-STEMI). In addition, echocardiography or cardiac magnetic resonance imaging may be employed. Common blood tests indicating an MI include elevated levels of creatine kinase MB (an enzyme that catalyzes the conversion of creatine to phosphocreatine, consuming ATP) and cardiac troponin (the regulatory protein for muscle contraction), both of which are released by damaged cardiac muscle cells.

Immediate treatments for MI are essential and include administering supplemental oxygen, aspirin that helps to break up clots, and nitroglycerine administered sublingually (under the tongue) to facilitate its absorption. Despite its unquestioned success in treatments and use since the 1880s, the mechanism of nitroglycerine is still incompletely understood but is believed to involve the release of nitric oxide, a known vasodilator, and endothelium-derived releasing factor, which also relaxes the smooth muscle in the tunica media of coronary vessels. Longer-term treatments include injections of thrombolytic agents such as streptokinase that dissolve the clot, the anticoagulant heparin, balloon angioplasty and stents to open blocked vessels, and bypass surgery to allow blood to pass around the site of blockage. If the damage is extensive, coronary

replacement with a donor heart or coronary assist device, a sophisticated mechanical device that supplements the pumping activity of the heart, may be employed. Despite the attention, development of artificial hearts to augment the severely limited supply of heart donors has proven less than satisfactory but will likely improve in the future.

MIs may trigger cardiac arrest, but the two are not synonymous. Important risk factors for MI include cardiovascular disease, age, smoking, high blood levels of the low-density lipoprotein (LDL, often referred to as "bad" cholesterol), low levels of high-density lipoprotein (HDL, or "good" cholesterol), hypertension, diabetes mellitus, obesity, lack of physical exercise, chronic kidney disease, excessive alcohol consumption, and use of illegal drugs.

Coronary Veins

Coronary veins drain the heart and generally parallel the large surface arteries (see Figure 6.1.14). The great cardiac vein can be seen initially on the surface of the heart following the interventricular sulcus, but it eventually flows along the coronary sulcus into the coronary sinus on the posterior surface. The great cardiac vein initially parallels the anterior interventricular artery and drains the areas supplied by this vessel. It receives several major branches, including the posterior cardiac vein, the middle cardiac vein, and the small cardiac vein. The posterior cardiac vein parallels and drains the areas supplied by the marginal artery branch of the circumflex artery. The middle cardiac vein parallels and drains the areas supplied by the posterior interventricular artery. The small cardiac vein parallels the right coronary artery and drains the blood from the posterior surfaces of the right atrium and ventricle. The coronary sinus is a large, thin-walled vein on the posterior surface of the heart lying within the atrioventricular sulcus and emptying directly into the right atrium. The anterior cardiac veins parallel the small cardiac arteries and drain the anterior surface of the right ventricle. Unlike these other cardiac veins, it bypasses the coronary sinus and drains directly into the right atrium.

Disorders of the Heart: Coronary Artery Disease

Coronary artery disease is the leading cause of death worldwide. It occurs when the buildup of plaque—a fatty material including cholesterol, connective tissue, white blood cells, and some smooth muscle cells—within the walls of the arteries obstructs the flow of blood and decreases the flexibility or compliance of the vessels. This condition is called atherosclerosis, a hardening of the arteries that involves the accumulation of plaque. As the coronary blood vessels become occluded, the flow of blood to the tissues will be restricted, a condition called ischemia that causes the cells to receive insufficient amounts of oxygen, called hypoxia. Figure 6.1.15 shows the blockage of coronary arteries highlighted by the injection of dye. Some individuals with coronary artery disease report pain radiating from the chest called angina pectoris, but others remain asymptomatic. If untreated, coronary artery disease can lead to MI or a heart attack.

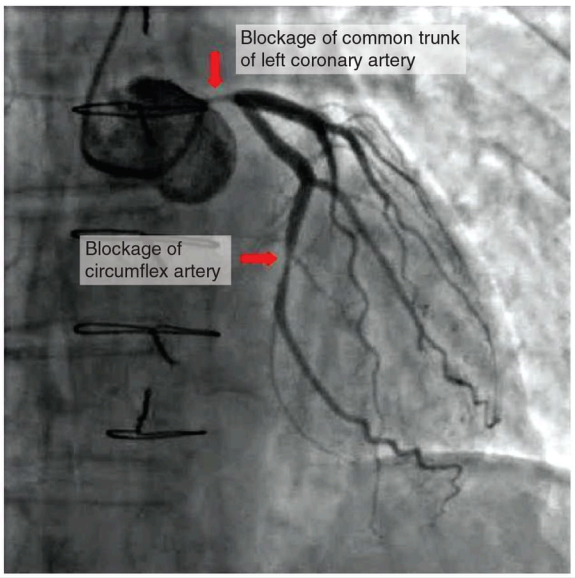


Figure 6.1.15: In this coronary angiogram (X-ray), the dye makes visible two occluded coronary arteries. Such blockages can lead to decreased blood flow (ischemia) and insufficient oxygen (hypoxia) delivered to the cardiac tissues. If uncorrected, this can lead to cardiac muscle death (myocardial infarction). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.1.15.

The disease progresses slowly and often begins in children and can be seen as fatty “streaks” in the vessels. It then gradually progresses throughout life. Well-documented risk factors include smoking, family history, hypertension, obesity, diabetes, high alcohol consumption, lack of exercise, stress, and hyperlipidemia or high circulating levels of lipids in the blood. Treatments may include medication, changes to diet and exercise, angioplasty with a balloon catheter, insertion of a stent, or coronary bypass procedure.

Angioplasty is a procedure in which the occlusion is mechanically widened with a balloon. A specialized catheter with an expandable tip is inserted into a superficial vessel, normally in the leg, and then directed to the site of the occlusion. At this point, the balloon is inflated to compress the plaque material and to open the vessel to increase blood flow. Then, the balloon is deflated and retracted. A stent consisting of a specialized mesh is typically inserted at the site of occlusion to reinforce the weakened and damaged walls. Stent insertions have been routine in cardiology for more than 40 years.

Coronary bypass surgery may also be performed. This surgical procedure grafts a replacement vessel obtained from another, less vital portion of the body to bypass the occluded area. This procedure is clearly effective in treating patients experiencing a MI, but overall does not increase longevity. Nor does it seem advisable in patients with stable although diminished cardiac capacity since frequently loss of mental acuity occurs following the procedure. Long-term changes to behavior, emphasizing diet and exercise plus a medicine regime tailored to lower blood pressure, lower cholesterol and lipids, and reduce clotting are equally as effective.

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6.2: Cardiac Muscle and Electrical Activity

Heart Conduction and Rhythm

Recall that cardiac muscle shares a few characteristics with both skeletal muscle and smooth muscle, but it has some unique properties of its own. Not the least of these exceptional properties is its ability to initiate an electrical potential at a fixed rate that spreads rapidly from cell to cell to trigger the contractile mechanism. This property is known as autorhythmicity. Neither smooth nor skeletal muscle can do this. Even though cardiac muscle has autorhythmicity, heart rate is modulated by the endocrine and nervous systems.

There are two major types of cardiac muscle cells: myocardial contractile cells and myocardial conducting cells. The myocardial contractile cells constitute the bulk (99 percent) of the cells in the atria and ventricles. Contractile cells conduct impulses and are responsible for contractions that pump blood through the body. The myocardial conducting cells (1 percent of the cells) form the conduction system of the heart. Except for Purkinje cells, they are generally much smaller than the contractile cells and have few of the myofibrils or filaments needed for contraction. Their function is similar in many respects to neurons, although they are specialized muscle cells. Myocardial conduction cells initiate and propagate the action potential (the electrical impulse) that travels throughout the heart and triggers the contractions that propel the blood.

Structure of Cardiac Muscle

Compared to the giant cylinders of skeletal muscle, cardiac muscle cells, or cardiomyocytes, are considerably shorter with much smaller diameters. Cardiac muscle also demonstrates striations, the alternating pattern of dark A bands and light I bands attributed to the precise arrangement of the myofilaments and fibrils that are organized in sarcomeres along the length of the cell (Figure $6.2.1a$). These contractile elements are virtually identical to skeletal muscle. T (transverse) tubules penetrate from the surface plasma membrane, the sarcolemma, to the interior of the cell, allowing the electrical impulse to reach the interior. The T tubules are only found at the Z discs, whereas in skeletal muscle, they are found at the junction of the A and I bands. Therefore, there are one-half as many T tubules in cardiac muscle as in skeletal muscle. In addition, the sarcoplasmic reticulum stores few calcium ions, so most of the calcium ions must come from outside the cells. The result is a slower onset of contraction. Mitochondria are plentiful, providing energy for the contractions of the heart. Typically, cardiomyocytes have a single, central nucleus, but two or more nuclei may be found in some cells.

Cardiac muscle cells branch freely. A junction between two adjoining cells is marked by a critical structure called an intercalated disc, which helps support the synchronized contraction of the muscle (Figure $6.2.1b$). The sarcolemmas from adjacent cells bind together at the intercalated discs. They consist of desmosomes, specialized linking proteoglycans, tight junctions, and large numbers of gap junctions that allow the passage of ions between the cells and help to synchronize the contraction (Figure 19.17**c**). Intercellular connective tissue also helps to bind the cells together. The importance of strongly binding these cells together is necessitated by the forces exerted by contraction.

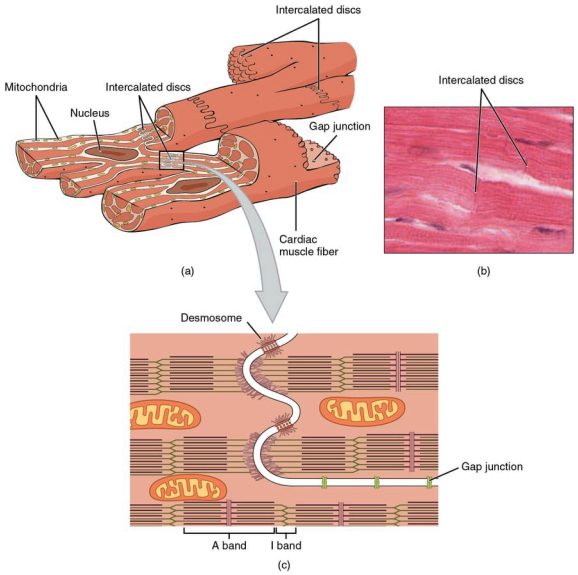


Figure 6.2.1: (a) Cardiac muscle cells have myofibrils composed of myofilaments arranged in sarcomeres, T tubules to transmit the impulse from the sarcolemma to the interior of the cell, numerous mitochondria for energy, and intercalated discs that are found at the junction of different cardiac muscle cells. (b) A photomicrograph of cardiac muscle cells shows the nuclei and intercalated discs. (c) An intercalated disc connects cardiac muscle cells and consists of desmosomes and gap junctions. LM × 1600. (Micrograph provided by the Regents of the University of Michigan Medical School © 2012). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.1.

Cardiac muscle undergoes aerobic respiration patterns, primarily metabolizing lipids and carbohydrates. Myoglobin, lipids, and glycogen are all stored within the cytoplasm. Cardiac muscle cells undergo twitch-type contractions with long refractory periods followed by brief relaxation periods. The relaxation is essential so the heart can fill with blood for the next cycle. The refractory period is very long to prevent the possibility of tetany, a condition in which muscle remains involuntarily contracted. In the heart, tetany is not compatible with life, since it would prevent the heart from pumping blood.

Everyday Connection

Repair and Replacement

Damaged cardiac muscle cells have extremely limited abilities to repair themselves or to replace dead cells via mitosis. Recent evidence indicates that at least some stem cells remain within the heart that continue to divide and at least potentially replace these dead cells. However, newly formed or repaired cells are rarely as functional as the original cells, and cardiac function is reduced. In the event of a heart attack or MI, dead cells are often replaced by patches of scar tissue. Autopsies performed on individuals who had successfully received heart transplants show some proliferation of original cells. If researchers can unlock the mechanism that generates new cells and restore full mitotic capabilities to heart muscle, the prognosis for heart attack survivors will be greatly enhanced. To date, myocardial cells produced within the patient (*in situ*) by cardiac stem cells seem to be nonfunctional, although those grown in Petri dishes (*in vitro*) do beat. Perhaps soon this mystery will be solved, and new advances in treatment will be commonplace.

Conduction System of the Heart

If embryonic heart cells are separated into a Petri dish and kept alive, each is capable of generating its own electrical impulse followed by contraction. When two independently beating embryonic cardiac muscle cells are placed together, the cell with the higher inherent rate sets the pace, and the impulse spreads from the faster to the slower cell to trigger a contraction. As more cells are joined together, the fastest cell continues to assume control of the rate. A fully developed adult heart maintains the capability of generating its own electrical impulse, triggered by the fastest cells, as part of the cardiac conduction system. The components of the cardiac conduction system include the sinoatrial node, the atrioventricular node, the atrioventricular bundle, the atrioventricular bundle branches, and the Purkinje cells (Figure 6.2.2).

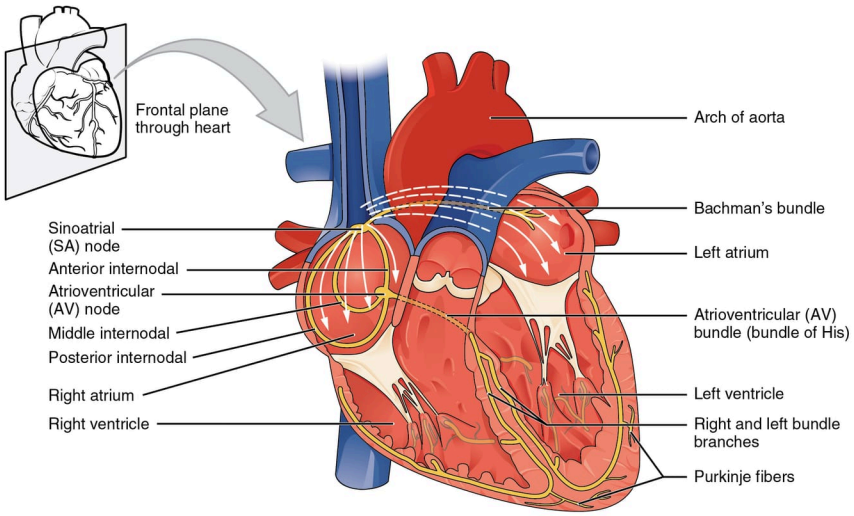


Figure 6.2.2: Specialized conducting components of the heart include the sinoatrial node, the internodal pathways, the atrioventricular node, the atrioventricular bundle, the right and left bundle branches, and the Purkinje fibers. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.2.

Sinoatrial (SA) Node

Normal cardiac rhythm is established by the sinoatrial (SA) node, a specialized clump of myocardial conducting cells located in the superior and posterior walls of the right atrium in close proximity to the orifice of the superior vena cava. The SA node has the highest inherent rate of depolarization and is known as the pacemaker of the heart. It initiates the sinus rhythm, or normal electrical pattern followed by contraction of the heart.

This impulse spreads from its initiation in the SA node throughout the atria through specialized internodal pathways, to the atrial myocardial contractile cells and the atrioventricular node. The internodal pathways consist of three bands (anterior, middle, and posterior) that lead directly from the SA node to the next node in the conduction system, the atrioventricular node (see Figure 6.2.2). The impulse takes approximately 50 ms (milliseconds) to travel between these two nodes. The relative importance of this pathway has been debated since the impulse would reach the atrioventricular node simply following the cell-by-cell pathway through the contractile cells of the myocardium in the atria. In addition, there is a specialized pathway called Bachmann's bundle or the interatrial band that conducts the impulse directly from the right atrium to the left atrium. Regardless of the pathway, as the impulse reaches the atrioventricular septum, the connective tissue of the cardiac skeleton prevents the impulse from spreading into the myocardial cells in the ventricles except at the atrioventricular node. Figure 6.2.3 illustrates the initiation of the impulse in the SA node that then spreads the impulse throughout the atria to the atrioventricular node.

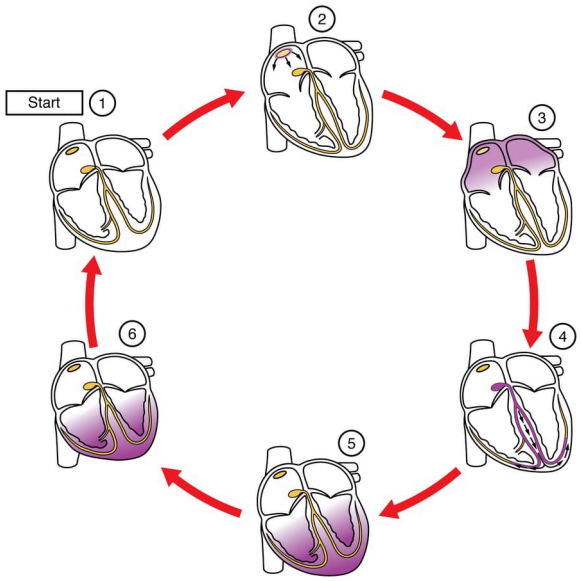


Figure 6.2.3: (1) The sinoatrial (SA) node and the remainder of the conduction system are at rest. (2) The SA node initiates the action potential, which sweeps across the atria. (3) After reaching the atrioventricular node, there is a delay of approximately 100 ms that allows the atria to complete pumping blood before the impulse is transmitted to the atrioventricular bundle. (4) Following the delay, the impulse travels through the atrioventricular bundle and bundle branches to the Purkinje fibers, and also reaches the right papillary muscle via the moderator band. (5) The impulse spreads to the contractile fibers of the ventricle. (6) Ventricular contraction begins. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.3.

The electrical event, the wave of depolarization, is the trigger for muscular contraction. The wave of depolarization begins in the right atrium, and the impulse spreads across the superior portions of both atria and then down through the contractile cells. The contractile cells then begin contraction from the superior to the inferior portions of the atria, efficiently pumping blood into the ventricles.

Atrioventricular (AV) Node

The atrioventricular (AV) node is a second clump of specialized myocardial conductive cells, located in the inferior portion of the right atrium within the atrioventricular septum. The septum prevents the impulse from spreading directly to the ventricles without passing through the AV node. There is a critical pause before the AV node depolarizes and transmits the impulse to the atrioventricular bundle (see Figure 6.2.3, step 3). This delay in transmission is partially attributable to the small diameter of the cells of the node, which slow the impulse. Also, conduction between nodal cells is less efficient than between conducting cells. These factors mean that it takes the impulse approximately 100 ms to pass through the node. This pause is critical to heart function, as it allows the atrial cardiomyocytes to complete their contraction that pumps blood into the ventricles before the impulse is transmitted to the cells of the ventricle itself. With extreme stimulation by the SA node, the AV node can transmit impulses maximally at 220 per minute. This establishes the typical maximum heart rate in a healthy young individual. Damaged hearts or those stimulated by drugs can contract at higher rates, but at these rates, the heart can no longer effectively pump blood.

Atrioventricular Bundle (Bundle of His), Bundle Branches, and Purkinje Fibers

Arising from the AV node, the atrioventricular bundle, or bundle of His, proceeds through the interventricular septum before dividing into two atrioventricular bundle branches, commonly called the left and right bundle branches. The left bundle branch has two fascicles. The left bundle branch supplies the left ventricle, and the right bundle branch the right ventricle. Since the left ventricle is much larger than the right, the left bundle branch is also considerably larger than the right. Portions of the right bundle branch are found in the moderator band and supply the right papillary muscles. Because of this connection, each papillary muscle receives the impulse at approximately the same time, so they begin to contract simultaneously just prior to the remainder of the myocardial contractile cells of the ventricles. This is believed to allow tension to develop on the chordae tendineae prior to right ventricular contraction. There is no corresponding moderator band on the left. Both bundle branches descend and reach the apex of the heart where they connect with the Purkinje fibers (see Figure 6.2.3, step 4). This passage takes approximately 25 ms.

The Purkinje fibers are additional myocardial conductive fibers that spread the impulse to the myocardial contractile cells in the

ventricles. They extend throughout the myocardium from the apex of the heart toward the atrioventricular septum and the base of the heart. The Purkinje fibers have a fast inherent conduction rate, and the electrical impulse reaches all of the ventricular muscle cells in about 75 ms (see Figure 6.2.3, step 5). Since the electrical stimulus begins at the apex, the contraction also begins at the apex and travels toward the base of the heart, similar to squeezing a tube of toothpaste from the bottom. This allows the blood to be pumped out of the ventricles and into the aorta and pulmonary trunk. The total time elapsed from the initiation of the impulse in the SA node until depolarization of the ventricles is approximately 225 ms.

Membrane Potentials and Ion Movement in Cardiac Conductive Cells

Action potentials are considerably different between cardiac conductive cells and cardiac contractive cells. While Na^+ and K^+ play essential roles, Ca^{2+} is also critical for both types of cells. Unlike skeletal muscles and neurons, cardiac conductive cells do not have a stable resting potential. Conductive cells contain a series of sodium ion channels that allow a normal and slow influx of sodium ions that causes the membrane potential to rise slowly from an initial value of –60 mV up to about –40 mV. The resulting movement of sodium ions creates spontaneous depolarization (or prepotential depolarization). At this point, calcium ion channels open and Ca^{2+} enters the cell, further depolarizing it at a more rapid rate until it reaches a value of approximately +15 mV. At this point, the calcium ion channels close and K^+ channels open, allowing outflux of K^+ and resulting in repolarization. When the membrane potential reaches approximately –60 mV, the K^+ channels close and Na^+ channels open, and the prepotential phase begins again. This phenomenon explains the autorhythmicity properties of cardiac muscle (Figure 6.2.4).

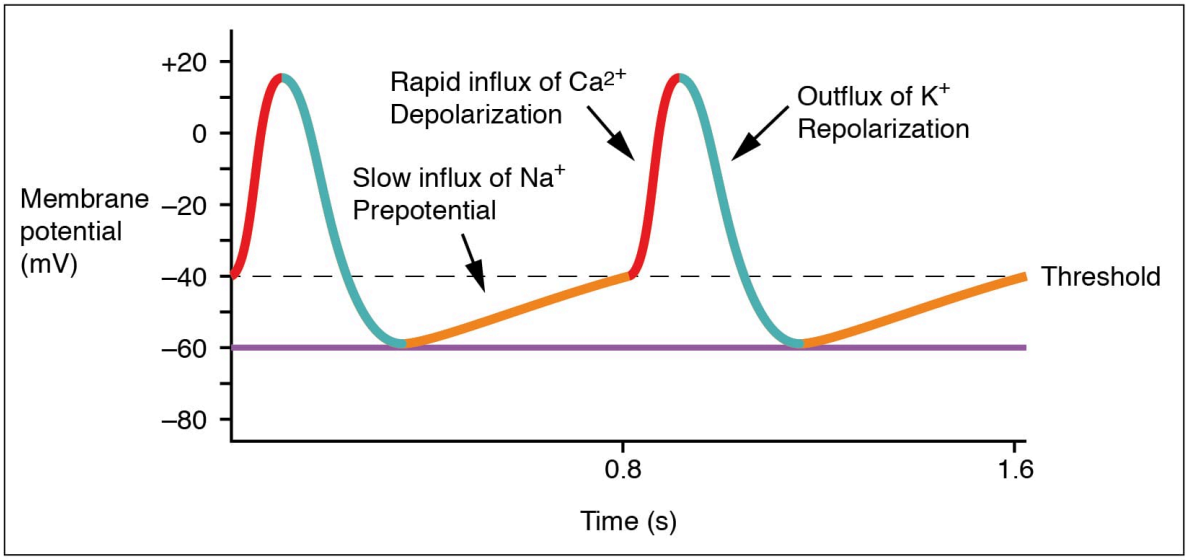


Figure 6.2.4: The prepotential is due to a slow influx of sodium ions until the threshold is reached followed by a rapid depolarization and repolarization. The prepotential accounts for the membrane reaching threshold and initiates the spontaneous depolarization and contraction of the cell. Note the lack of a resting potential. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.4.

Membrane Potentials and Ion Movement in Cardiac Contractile Cells

There is a distinctly different electrical pattern involving the contractile cells. In this case, there is a rapid depolarization, followed by a plateau phase and then repolarization. This phenomenon accounts for the long refractory periods required for the cardiac muscle cells to pump blood effectively before they are capable of firing for a second time. These cardiac myocytes normally do not initiate their own electrical potential but rather wait for an impulse to reach them.

Contractile cells demonstrate a much more stable resting phase than conductive cells at approximately –80 mV for cells in the atria and –90 mV for cells in the ventricles. Despite this initial difference, the other components of their action potentials are virtually identical. In both cases, when stimulated by an action potential, voltage-gated channels rapidly open, beginning the positive-feedback mechanism of depolarization. This rapid influx of positively charged ions raises the membrane potential to approximately +30 mV, at which point the sodium channels close. The rapid depolarization period typically lasts 3–5 ms. Depolarization is followed by the plateau phase, in which membrane potential declines relatively slowly. This is due in large part to the opening of the slow Ca^{2+} channels, allowing Ca^{2+} to enter the cell while few K^+ channels are open, allowing K^+ to exit the

cell. The relatively long plateau phase lasts approximately 175 ms. Once the membrane potential reaches approximately zero, the Ca^{2+} channels close and K^+ channels open, allowing K^+ to exit the cell. The repolarization lasts approximately 75 ms. At this point, membrane potential drops until it reaches resting levels once more and the cycle repeats. The entire event lasts between 250 and 300 ms (Figure 6.2.5).

The absolute refractory period for cardiac contractile muscle lasts approximately 200 ms, and the relative refractory period lasts approximately 50 ms, for a total of 250 ms. This extended period is critical, since the heart muscle must contract to pump blood effectively and the contraction must follow the electrical events. Without extended refractory periods, premature contractions would occur in the heart and would not be compatible with life.

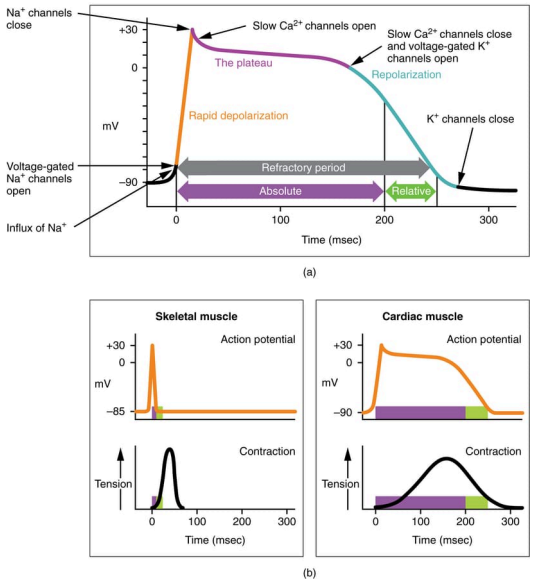


Figure 6.2.5: (a) Note the long plateau phase due to the influx of calcium ions. The extended refractory period allows the cell to fully contract before another electrical event can occur. (b) The action potential for heart muscle is compared to that of skeletal muscle. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.5.

Calcium Ions

Calcium ions play two critical roles in the physiology of cardiac muscle. Their influx through slow calcium channels accounts for the prolonged plateau phase and absolute refractory period that enable cardiac muscle to function properly. Calcium ions also combine with the regulatory protein troponin in the troponin-tropomyosin complex; this complex removes the inhibition that prevents the heads of the myosin molecules from forming cross bridges with the active sites on actin that provide the power stroke of contraction. This mechanism is virtually identical to that of skeletal muscle. Approximately 20 percent of the calcium required for contraction is supplied by the influx of Ca^{2+} during the plateau phase. The remaining Ca^{2+} for contraction is released from storage in the sarcoplasmic reticulum.

Comparative Rates of Conduction System Firing

The pattern of prepotential or spontaneous depolarization, followed by rapid depolarization and repolarization just described, are seen in the SA node and a few other conductive cells in the heart. Since the SA node is the pacemaker, it reaches threshold faster than any other component of the conduction system. It will initiate the impulses spreading to the other conducting cells. The SA node, without nervous or endocrine control, would initiate a heart impulse approximately 80–100 times per minute. Although each component of the conduction system is capable of generating its own impulse, the rate progressively slows as you proceed from the SA node to the Purkinje fibers. Without the SA node, the AV node would generate a heart rate of 40–60 beats per minute. If the AV node were blocked, the atrioventricular bundle would fire at a rate of approximately 30–40 impulses per minute. The bundle branches would have an inherent rate of 20–30 impulses per minute, and the Purkinje fibers would fire at 15–20 impulses per minute. While a few exceptionally trained aerobic athletes demonstrate resting heart rates in the range of 30–40 beats per minute (the lowest recorded figure is 28 beats per minute for Miguel Indurain, a cyclist), for most individuals, rates lower than 50 beats per minute would indicate a condition called bradycardia. Depending upon the specific individual, as rates fall much below this level, the heart would be unable to maintain adequate flow of blood to vital tissues, initially resulting in decreasing loss of

function across the systems, unconsciousness, and ultimately death.

Electrocardiogram

By careful placement of surface electrodes on the body, it is possible to record the complex, compound electrical signal of the heart. This tracing of the electrical signal is the electrocardiogram (ECG), also commonly abbreviated EKG (K coming kardiology, from the German term for cardiology). Careful analysis of the ECG reveals a detailed picture of both normal and abnormal heart function, and is an indispensable clinical diagnostic tool. The standard electrocardiograph (the instrument that generates an ECG) uses 3, 5, or 12 leads. The greater the number of leads an electrocardiograph uses, the more information the ECG provides. The term "lead" may be used to refer to the cable from the electrode to the electrical recorder, but it typically describes the voltage difference between two of the electrodes. The 12-lead electrocardiograph uses 10 electrodes placed in standard locations on the patient's skin (Figure 6.2.6). In continuous ambulatory electrocardiographs, the patient wears a small, portable, battery-operated device known as a Holter monitor, or simply a Holter, that continuously monitors heart electrical activity, typically for a period of 24 hours during the patient's normal routine.

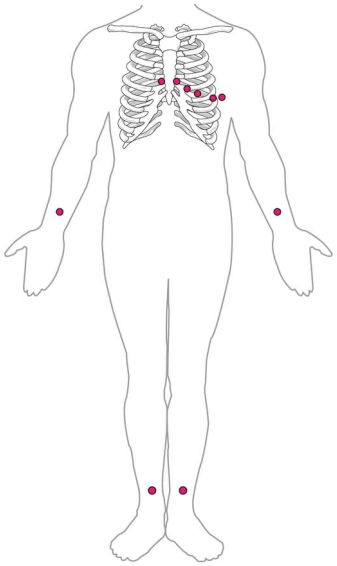


Figure 6.2.6: In a 12-lead ECG, six electrodes are placed on the chest, and four electrodes are placed on the limbs. (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 6.2.6.

A normal ECG tracing is presented in Figure 6.2.7. Each component, segment, and interval is labeled and corresponds to important electrical events, demonstrating the relationship between these events and contraction in the heart.

There are five prominent points on the ECG: the P wave, the QRS complex, and the T wave. The small P wave represents the depolarization of the atria. The atria begin contracting approximately 25 ms after the start of the P wave. The large QRS complex represents the depolarization of the ventricles, which requires a much stronger electrical signal because of the larger size of the ventricular cardiac muscle. The ventricles begin to contract as the QRS reaches the peak of the R wave. Lastly, the T wave represents the repolarization of the ventricles. The repolarization of the atria occurs during the QRS complex, which masks it on an ECG.

The major segments and intervals of an ECG tracing are indicated in Figure 6.2.7. Segments are defined as the regions between two waves. Intervals include one segment plus one or more waves. For example, the PR segment begins at the end of the P wave and ends at the beginning of the QRS complex. The PR interval starts at the beginning of the P wave and ends with the beginning of the QRS complex. The PR interval is more clinically relevant, as it measures the duration from the beginning of atrial depolarization (the P wave) to the initiation of the QRS complex. Since the Q wave may be difficult to view in some tracings, the measurement is often extended to the R that is more easily visible. Should there be a delay in passage of the impulse from the SA node to the AV node, it would be visible in the PR interval. Figure 19.24 correlates events of heart contraction to the corresponding segments and intervals of an ECG.

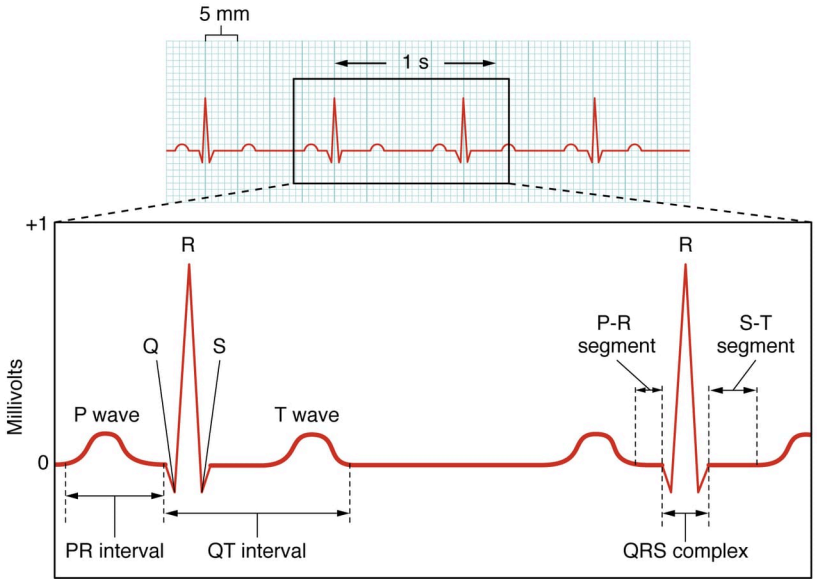


Figure 6.2.7: A normal tracing shows the P wave, QRS complex, and T wave. Also indicated are the PR, QT, QRS, and ST intervals, plus the P-R and S-T segments. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.7.

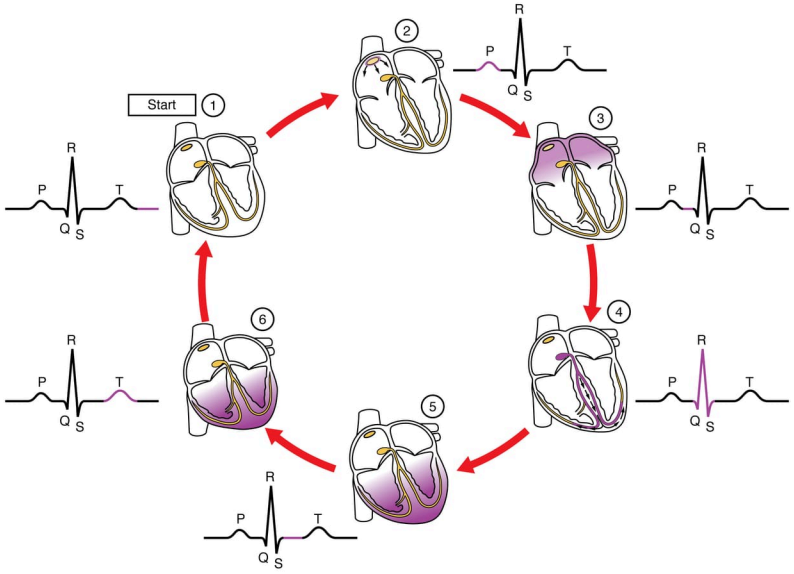


Figure 6.2.8: This diagram correlates an ECG tracing with the electrical and mechanical events of a heart contraction. Each segment of an ECG tracing corresponds to one event in the cardiac cycle. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.8.

Everyday Connection

ECG Abnormalities

Occassionally, an area of the heart other than the SA node will initiate an impulse that will be followed by a premature contraction. Such an area, which may actually be a component of the conduction system or some other contractile cells, is known as an ectopic focus or ectopic pacemaker. An ectopic focus may be stimulated by localized ischemia; exposure to certain drugs, including caffeine, digitalis, or acetylcholine; elevated stimulation by both sympathetic or parasympathetic divisions of the autonomic nervous system; or a number of disease or pathological conditions. Occasional occurences are generally transitory and nonlife threatening, but if the condition becomes chronic, it may lead to either an arrhythmia, a deviation from the normal pattern of impulse conduction and contraction, or to fibrillation, an uncoordinated beating of the heart.

While interpretation of an ECG is possible and extremely valuable after some training, a full understanding of the complexities and intricacies generally requires several years of experience. In general, the size of the electrical variations, the duration of the events, and detailed vector analysis provide the most comprehensive picture of cardiac function. For example, an amplified P wave may indicate enlargement of the atria, an enlarged Q wave may indicate a MI, and an enlarged suppressed or inverted Q wave often indicates enlarged ventricles. T waves often appear flatter when insufficient oxygen is being delivered to the myocardium. An elevation of the ST segment above baseline is often seen in patients with an acute MI, and may appear depressed below the baseline when hypoxia is occurring.

As useful as analyzing these electrical recordings may be, there are limitations. For example, not all areas suffering a MI may be obvious on the ECG. Additionally, it will not reveal the effectiveness of the pumping, which requires further testing, such as an ultrasound test called an echocardiogram or nuclear medicine imaging. It is also possible for there to be pulseless electrical activity, which will show up on an ECG tracing, although there is no corresponding pumping action. Common abnormalities that may be detected by the ECGs are shown in Figure 6.2.9.

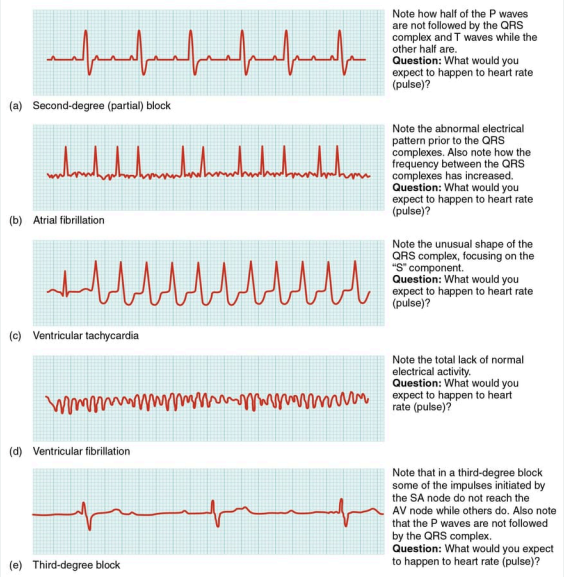


Figure 6.2.9: (a) In a second-degree or partial block, one-half of the P waves are not followed by the QRS complex and T waves while the other half are. (b) In atrial fibrillation, the electrical pattern is abnormal prior to the QRS complex, and the frequency between the QRS complexes has increased. (c) In ventricular tachycardia, the shape of the QRS complex is abnormal. (d) In ventricular fibrillation, there is no normal electrical activity. (e) In a third-degree block, there is no correlation between atrial activity (the P wave) and ventricular activity (the QRS complex). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.9.

Everyday Connection

External Automated Defibrillators

In the event that the electrical activity of the heart is severely disrupted, cessation of electrical activity or fibrillation may occur. In fibrillation, the heart beats in a wild, uncontrolled manner, which prevents it from being able to pump effectively. Atrial fibrillation (see Figure 6.2.9*b*) is a serious condition, but as long as the ventricles continue to pump blood, the patient's life may not be in immediate danger. Ventricular fibrillation (see Figure 6.2.9*d*) is a medical emergency that requires life support, because the ventricles are not effectively pumping blood. In a hospital setting, it is often described as "code blue." If untreated for as little as a few minutes, ventricular fibrillation may lead to brain death. The most common treatment is defibrillation, which uses special paddles to apply a charge to the heart from an external electrical source in an attempt to establish a normal sinus rhythm (Figure 6.2.10). A defibrillator effectively stops the heart so that the SA node can trigger a normal conduction cycle. Because of their effectiveness in reestablishing a normal sinus rhythm, external automated defibrillators (EADs) are being placed in areas frequented by large numbers of people, such as schools, restaurants, and airports. These devices contain simple and direct verbal instructions that can be followed by nonmedical personnel in an attempt to save a life.



(a) (b)

Figure 6.2.10: (a) An external automatic defibrillator can be used by nonmedical personnel to reestablish a normal sinus rhythm in a person with fibrillation. (b) Defibrillator paddles are more commonly used in hospital settings. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.2.10.

A heart block refers to an interruption in the normal conduction pathway. The nomenclature for these is very straightforward. SA nodal blocks occur within the SA node. AV nodal blocks occur within the AV node. Infra-Hisian blocks involve the bundle of His. Bundle branch blocks occur within either the left or right atrioventricular bundle branches. Hemiblocks are partial and occur within one or more fascicles of the atrioventricular bundle branch. Clinically, the most common types are the AV nodal and infra-Hisian blocks.

AV blocks are often described by degrees. A first-degree or partial block indicates a delay in conduction between the SA and AV nodes. This can be recognized on the ECG as an abnormally long PR interval. A second-degree or incomplete block occurs when some impulses from the SA node reach the AV node and continue, while others do not. In this instance, the ECG would reveal some P waves not followed by a QRS complex, while others would appear normal. In the third-degree or complete block, there is no correlation between atrial activity (the P wave) and ventricular activity (the QRS complex). Even in the event of a total SA block, the AV node will assume the role of pacemaker and continue initiating contractions at 40–60 contractions per minute, which is adequate to maintain consciousness. Second- and third-degree blocks are demonstrated on the ECG presented in Figure 6.2.9.

When arrhythmias become a chronic problem, the heart maintains a junctional rhythm, which originates in the AV node. In order to speed up the heart rate and restore full sinus rhythm, a cardiologist can implant an artificial pacemaker, which delivers electrical impulses to the heart muscle to ensure that the heart continues to contract and pump blood effectively. These artificial pacemakers are programmable by the cardiologists and can either provide stimulation temporarily upon demand or on a continuous basis. Some devices also contain built-in defibrillators.

Cardiac Muscle Metabolism

Normally, cardiac muscle metabolism is entirely aerobic. Oxygen from the lungs is brought to the heart, and every other organ, attached to the hemoglobin molecules within the erythrocytes. Heart cells also store appreciable amounts of oxygen in myoglobin. Normally, these two mechanisms, circulating oxygen and oxygen attached to myoglobin, can supply sufficient oxygen to the heart, even during peak performance.

Fatty acids and glucose from the circulation are broken down within the mitochondria to release energy in the form of ATP. Both fatty acid droplets and glycogen are stored within the sarcoplasm and provide additional nutrient supply. (Seek additional content for more detail about metabolism.)

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6.3: Cardiac Cycle

Blood Flow Through the Heart

The period of time that begins with contraction of the atria and ends with ventricular relaxation is known as the cardiac cycle (Figure 6.3.1). The period of contraction that the heart undergoes while it pumps blood into circulation is called systole. The period of relaxation that occurs as the chambers fill with blood is called diastole. Both the atria and ventricles undergo systole and diastole, and it is essential that these components be carefully regulated and coordinated to ensure blood is pumped efficiently to the body.

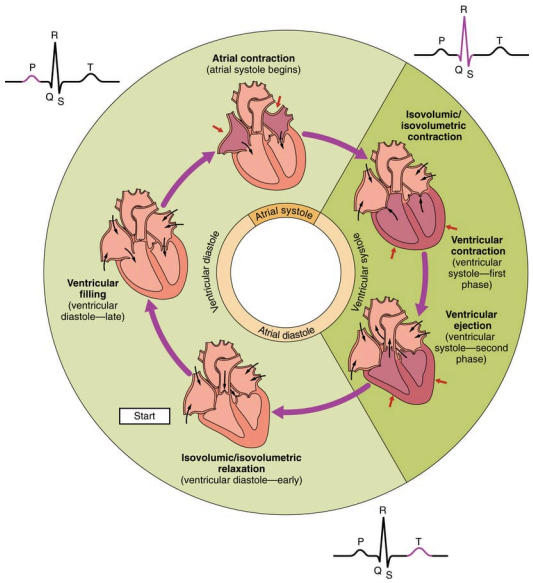


Figure 6.3.1: The cardiac cycle begins with atrial systole and progresses to ventricular systole, atrial diastole, and ventricular diastole, when the cycle begins again. Correlations to the ECG are highlighted. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.3.1.

Pressures and Flow

Fluids, whether gases or liquids, are materials that flow according to pressure gradients—that is, they move from regions that are higher in pressure to regions that are lower in pressure. Accordingly, when the heart chambers are relaxed (diastole), blood will flow into the atria from the veins, which are higher in pressure. As blood flows into the atria, the pressure will rise, so the blood will initially move passively from the atria into the ventricles. When the action potential triggers the muscles in the atria to contract (atrial systole), the pressure within the atria rises further, pumping blood into the ventricles. During ventricular systole, pressure rises in the ventricles, pumping blood into the pulmonary trunk from the right ventricle and into the aorta from the left ventricle. Again, as you consider this flow and relate it to the conduction pathway, the elegance of the system should become apparent.

Phases of the Cardiac Cycle

At the beginning of the cardiac cycle, both the atria and ventricles are relaxed (diastole). Blood is flowing into the right atrium from the superior and inferior venae cavae and the coronary sinus. Blood flows into the left atrium from the four pulmonary veins. The two atrioventricular valves, the tricuspid and mitral valves, are both open, so blood flows unimpeded from the atria and into the ventricles. Approximately 70–80 percent of ventricular filling occurs by this method. The two semilunar valves, the pulmonary and aortic valves, are closed, preventing backflow of blood into the right and left ventricles from the pulmonary trunk on the right and the aorta on the left.

Atrial Systole and Diastole

Contraction of the atria follows depolarization, represented by the P wave of the ECG. As the atrial muscles contract from the superior portion of the atria toward the atrioventricular septum, pressure rises within the atria and blood is pumped into the ventricles through the open atrioventricular (tricuspid, and mitral or bicuspid) valves. At the start of atrial systole, the ventricles are normally filled with approximately 70–80 percent of their capacity due to inflow during diastole. Atrial contraction, also referred to as the "atrial kick," contributes the remaining 20–30 percent of filling (see Figure 6.3.1). Atrial systole lasts approximately 100 ms and ends prior to ventricular systole, as the atrial muscle returns to diastole and blood flow into the atria resumes.

Ventricular Systole

Ventricular systole (see Figure 6.3.1) follows the depolarization of the ventricles and is represented by the QRS complex in the ECG. It may be conveniently divided into two phases, lasting a total of 270 ms. At the end of atrial systole and just prior to ventricular contraction, the ventricles contain approximately 130 mL blood in a resting adult in a standing position. This volume is known as the end diastolic volume (EDV) or preload.

Initially, as the muscles in the ventricle contract, the pressure of the blood within the chamber rises, but it is not yet high enough to open the semilunar (pulmonary and aortic) valves and be ejected from the heart. However, blood pressure quickly rises above that of the atria that are now relaxed and in diastole. This increase in pressure causes blood to flow back toward the atria, closing the tricuspid and mitral valves. Since blood is not being ejected from the ventricles at this early stage, the volume of blood within the chamber remains constant. Consequently, this initial phase of ventricular systole is known as isovolumic contraction, also called isovolumetric contraction (see Figure 6.3.1).

In the second phase of ventricular systole, the ventricular ejection phase, the contraction of the ventricular muscle has raised the pressure within the ventricle to the point that it is greater than the pressures in the pulmonary trunk and the aorta. Blood is pumped from the heart, pushing open the pulmonary and aortic semilunar valves. Pressure generated by the left ventricle will be appreciably greater than the pressure generated by the right ventricle, since the existing pressure in the aorta will be so much higher. Nevertheless, both ventricles pump the same amount of blood. This quantity is referred to as stroke volume. Stroke volume will normally be in the range of 70–80 mL. Since ventricular systole began with an EDV of approximately 130 mL of blood, this means that there is still 50–60 mL of blood remaining in the ventricle following contraction. This volume of blood is known as the end systolic volume (ESV).

Ventricular Diastole

Ventricular relaxation, or diastole, follows repolarization of the ventricles and is represented by the T wave of the ECG. It too is divided into two distinct phases and lasts approximately 430 ms.

During the early phase of ventricular diastole, as the ventricular muscle relaxes, pressure on the remaining blood within the ventricle begins to fall. When pressure within the ventricles drops below pressure in both the pulmonary trunk and aorta, blood flows back toward the heart, producing the dicrotic notch (small dip) seen in blood pressure tracings. The semilunar valves close to prevent backflow into the heart. Since the atrioventricular valves remain closed at this point, there is no change in the volume of blood in the ventricle, so the early phase of ventricular diastole is called the isovolumic ventricular relaxation phase, also called isovolumetric ventricular relaxation phase (see Figure 6.3.1).

In the second phase of ventricular diastole, called late ventricular diastole, as the ventricular muscle relaxes, pressure on the blood within the ventricles drops even further. Eventually, it drops below the pressure in the atria. When this occurs, blood flows from the atria into the ventricles, pushing open the tricuspid and mitral valves. As pressure drops within the ventricles, blood flows from the major veins into the relaxed atria and from there into the ventricles. Both chambers are in diastole, the atrioventricular valves are open, and the semilunar valves remain closed (see Figure 6.3.1). The cardiac cycle is complete.

Figure 6.3.2 illustrates the relationship between the cardiac cycle and the ECG.

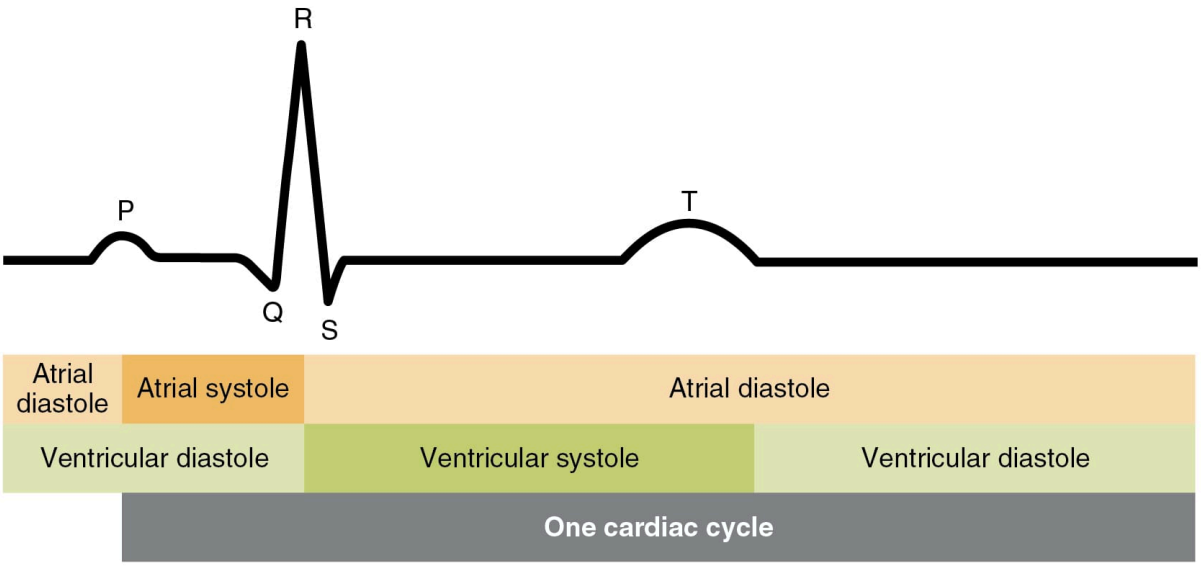


Figure 6.3.2: Initially, both the atria and ventricles are relaxed (diastole). The P wave represents depolarization of the atria and is followed by atrial contraction (systole). Atrial systole extends until the QRS complex, at which point, the atria relax. The QRS complex represents depolarization of the ventricles and is followed by ventricular contraction. The T wave represents the repolarization of the ventricles and marks the beginning of ventricular relaxation. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.3.2.

Heart Sounds

One of the simplest, yet effective, diagnostic techniques applied to assess the state of a patient's heart is auscultation using a stethoscope.

In a normal, healthy heart, there are only two audible heart sounds: S_1 and S_2. S_1 is the sound created by the closing of the atrioventricular valves during ventricular contraction and is normally described as a "lub," or first heart sound. The second heart sound, S_2, is the sound of the closing of the semilunar valves during ventricular diastole and is described as a "dub" (Figure 6.3.3). In both cases, as the valves close, the openings within the atrioventricular septum guarded by the valves will become reduced, and blood flow through the opening will become more turbulent until the valves are fully closed. There is a third heart sound, S_3, but it is rarely heard in healthy individuals. It may be the sound of blood flowing into the atria, or blood sloshing back and forth in the ventricle, or even tensing of the chordae tendineae. S_3 may be heard in youth, some athletes, and pregnant people. If the sound is heard later in life, it may indicate congestive heart failure, warranting further tests. Some cardiologists refer to the collective S_1, S_2, and S_3 sounds as the "Kentucky gallop," because they mimic those produced by a galloping horse. The fourth heart sound, S_4, results from the contraction of the atria pushing blood into a stiff or hypertrophic ventricle, indicating failure of the left ventricle. S_4 occurs prior to S_1 and the collective sounds S_4, S_1, and S_2 are referred to by some cardiologists as the "Tennessee gallop," because of their similarity to the sound produced by a galloping horse with a different gait. A few individuals may have both S_3 and S_4, and this combined sound is referred to as S_7.

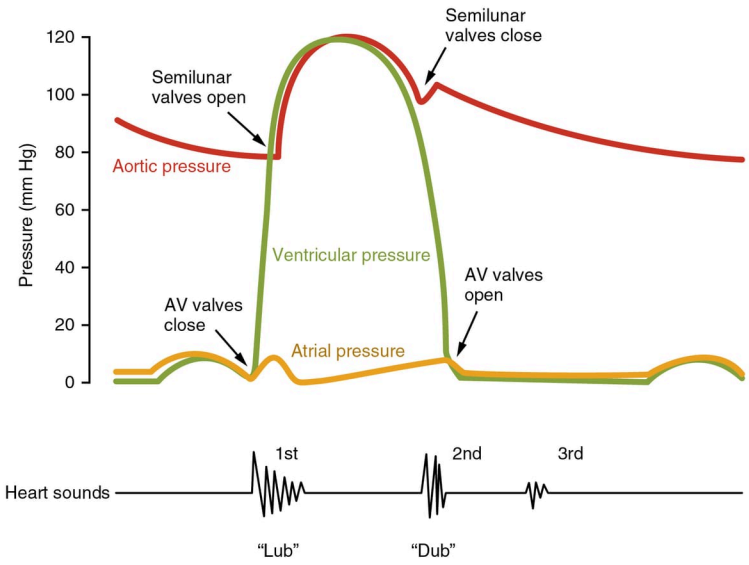


Figure 6.3.3: In this illustration, the x-axis reflects time with a recording of the heart sounds. The y-axis represents pressure. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.3.3.

The term murmur is used to describe an unusual sound coming from the heart that is caused by the turbulent flow of blood. Murmurs are graded on a scale of 1 to 6, with 1 being the most common, the most difficult sound to detect, and the least serious. The most severe is a 6. Phonocardiograms or auscultograms can be used to record both normal and abnormal sounds using specialized electronic stethoscopes.

During auscultation, it is common practice for the clinician to ask the patient to breathe deeply. This procedure not only allows for listening to airflow, but it may also amplify heart murmurs. Inhalation increases blood flow into the right side of the heart and may increase the amplitude of right-sided heart murmurs. Expiration partially restricts blood flow into the left side of the heart and may amplify left-sided heart murmurs. Figure 6.3.4 indicates proper placement of the bell of the stethoscope to facilitate auscultation.

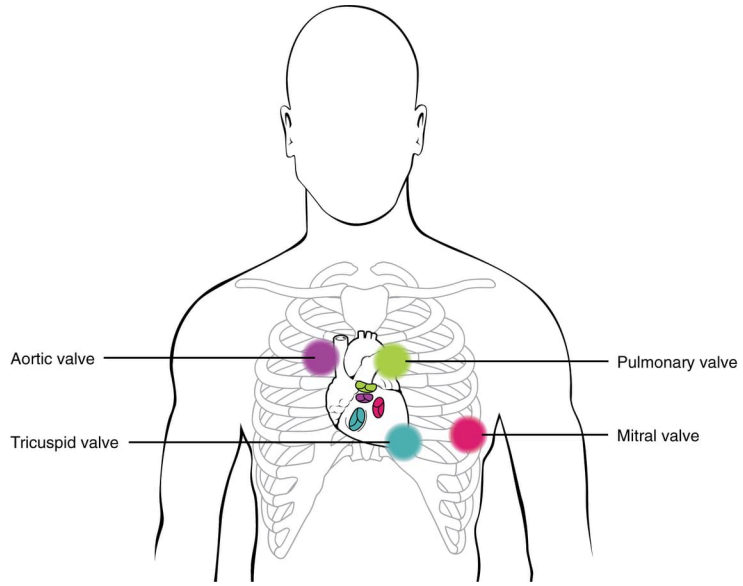


Figure 6.3.4: Proper placement of the bell of the stethoscope facilitates auscultation. At each of the four locations on the chest, a different valve can be heard. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.3.4.

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6.4: Cardiac Physiology

Regulation of Cardiac Function

The autorhythmicity inherent in cardiac cells keeps the heart beating at a regular pace; however, the heart is regulated by and responds to outside influences as well. Neural and endocrine controls are vital to the regulation of cardiac function. In addition, the heart is sensitive to several environmental factors, including electrolytes.

Resting Cardiac Output

Cardiac output (CO) is a measurement of the amount of blood pumped by each ventricle in one minute. To calculate this value, multiply stroke volume (SV), the amount of blood pumped by each ventricle, by heart rate (HR), in contractions per minute (or beats per minute, bpm). It can be represented mathematically by the following equation:

$$CO = HR \times SV$$

SV is normally measured using an echocardiogram to record EDV and ESV, and calculating the difference: SV = EDV – ESV. SV can also be measured using a specialized catheter, but this is an invasive procedure and far more dangerous to the patient. A mean SV for a resting 70-kg (150-lb) individual would be approximately 70 mL. There are several important variables, including size of the heart, physical and mental condition of the individual, sex, contractility, duration of contraction, preload or EDV, and afterload or resistance. Normal range for SV would be 55–100 mL. An average resting HR would be approximately 75 bpm but could range from 60–100 in some individuals.

Using these numbers, the mean CO is 5.25 L/min, with a range of 4.0–8.0 L/min. Remember, however, that these numbers refer to CO from each ventricle separately, not the total for the heart. Factors influencing CO are summarized in Figure 6.4.1.

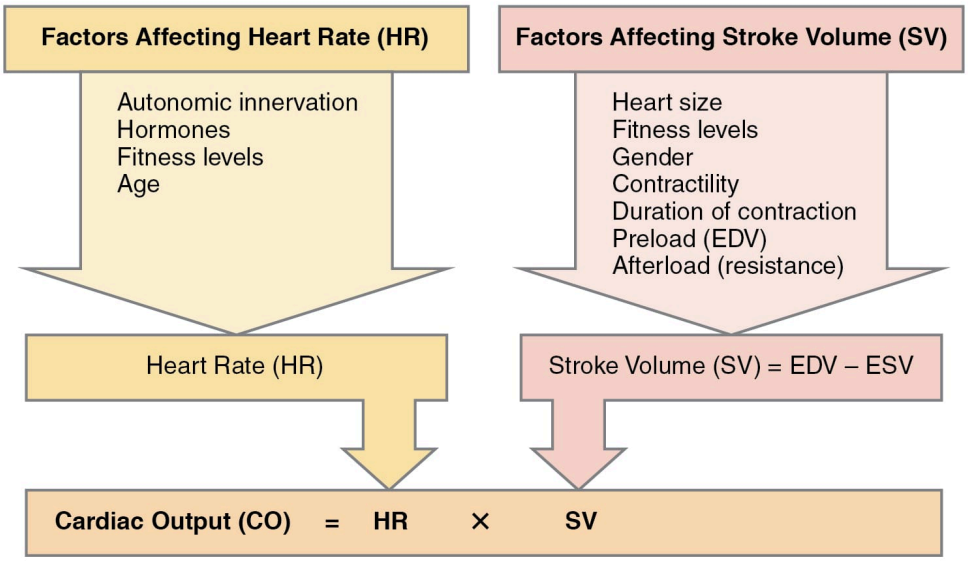


Figure 6.4.1: Cardiac output is influenced by heart rate and stroke volume, both of which are also variable. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.4.1.

SVs are also used to calculate ejection fraction, which is the portion of the blood that is pumped or ejected from the heart with each contraction. To calculate ejection fraction, SV is divided by EDV. Despite the name, the ejection fraction is normally expressed as a percentage. Ejection fractions range from approximately 55–70 percent, with a mean of 58 percent.

Exercise and Maximum Cardiac Output

In healthy young individuals, HR may increase to 150 bpm during exercise. SV can also increase from 70 to approximately 130 mL due to increased strength of contraction. This would increase CO to approximately 19.5 L/min, 4–5 times the resting rate. Top cardiovascular athletes can achieve even higher levels. At their peak performance, they may increase resting CO by 7–8 times.

Since the heart is a muscle, exercising it increases its efficiency. The difference between maximum and resting CO is known as the cardiac reserve. It measures the residual capacity of the heart to pump blood.

Heart Rates

HRs vary considerably, not only with exercise and fitness levels, but also with age. Newborn resting HRs may be 120 bpm. HR gradually decreases until young adulthood and then gradually increases again with age.

Maximum HRs are normally in the range of 200–220 bpm, although there are some extreme cases in which they may reach higher levels. As one ages, the ability to generate maximum rates decreases. This may be estimated by taking the maximal value of 220 bpm and subtracting the individual's age. So a 40-year-old individual would be expected to hit a maximum rate of approximately 180, and a 60-year-old person would achieve a HR of 160.

Disorders of the Heart: Abnormal Heart Rates

For an adult, normal resting HR will be in the range of 60–100 bpm. Bradycardia is the condition in which resting rate drops below 60 bpm, and tachycardia is the condition in which the resting rate is above 100 bpm. Trained athletes typically have very low HRs. If the patient is not exhibiting other symptoms, such as weakness, fatigue, dizziness, fainting, chest discomfort, palpitations, or respiratory distress, bradycardia is not considered clinically significant. However, if any of these symptoms are present, they may indicate that the heart is not providing sufficient oxygenated blood to the tissues. The term relative bradycardia may be used with a patient who has a HR in the normal range but is still suffering from these symptoms. Most patients remain asymptomatic as long as the HR remains above 50 bpm.

Bradycardia may be caused by either inherent factors or causes external to the heart. While the condition may be inherited, typically it is acquired in older individuals. Inherent causes include abnormalities in either the SA or AV node. If the condition is serious, a pacemaker may be required. Other causes include ischemia to the heart muscle or diseases of the heart vessels or valves. External causes include metabolic disorders, pathologies of the endocrine system often involving the thyroid, electrolyte imbalances, neurological disorders including inappropriate autonomic responses, autoimmune pathologies, over-prescription of beta blocker drugs that reduce HR, recreational drug use, or even prolonged bed rest. Treatment relies upon establishing the underlying cause of the disorder and may necessitate supplemental oxygen.

Tachycardia is not normal in a resting patient but may be detected in pregnant people or individuals experiencing extreme stress. In the latter case, it would likely be triggered by stimulation from the limbic system or disorders of the autonomic nervous system. In some cases, tachycardia may involve only the atria. Some individuals may remain asymptomatic, but when present, symptoms may include dizziness, shortness of breath, lightheadedness, rapid pulse, heart palpations, chest pain, or fainting (syncope). While tachycardia is defined as a HR above 100 bpm, there is considerable variation among people. Further, the normal resting HRs of children are often above 100 bpm, but this is not considered to be tachycardia. Many causes of tachycardia may be benign, but the condition may also be correlated with fever, anemia, hypoxia, hyperthyroidism, hypersecretion of catecholamines, some cardiomyopathies, some disorders of the valves, and acute exposure to radiation. Elevated rates in an exercising or resting patient are normal and expected. Resting rate should always be taken after recovery from exercise. Treatment depends upon the underlying cause but may include medications, implantable cardioverter defibrillators, ablation, or surgery.

Correlation Between Heart Rates and Cardiac Output

Initially, physiological conditions that cause HR to increase also trigger an increase in SV. During exercise, the rate of blood returning to the heart increases. However as the HR rises, there is less time spent in diastole and consequently less time for the ventricles to fill with blood. Even though there is less filling time, SV will initially remain high. However, as HR continues to increase, SV gradually decreases due to decreased filling time. CO will initially stabilize as the increasing HR compensates for the decreasing SV, but at very high rates, CO will eventually decrease as increasing rates are no longer able to compensate for the

decreasing SV. Consider this phenomenon in a healthy young individual. Initially, as HR increases from resting to approximately 120 bpm, CO will rise. As HR increases from 120 to 160 bpm, CO remains stable, since the increase in rate is offset by decreasing ventricular filling time and, consequently, SV. As HR continues to rise above 160 bpm, CO actually decreases as SV falls faster than HR increases. So although aerobic exercises are critical to maintain the health of the heart, individuals are cautioned to monitor their HR to ensure they stay within the target heart rate range of between 120 and 160 bpm, so CO is maintained. The target HR is loosely defined as the range in which both the heart and lungs receive the maximum benefit from the aerobic workout and is dependent upon age.

Cardiovascular Centers

Nervous control over HR is centralized within the two paired cardiovascular centers of the medulla oblongata (Figure 6.4.2). The cardioaccelerator regions stimulate activity via sympathetic stimulation of the cardioaccelerator nerves, and the cardioinhibitory centers decrease heart activity via parasympathetic stimulation as one component of the vagus nerve, cranial nerve X. During rest, both centers provide slight stimulation to the heart, contributing to autonomic tone. This is a similar concept to tone in skeletal muscles. Normally, vagal stimulation predominates as, left unregulated, the SA node would initiate a sinus rhythm of approximately 100 bpm.

Both sympathetic and parasympathetic stimulations flow through a paired complex network of nerve fibers known as the cardiac plexus near the base of the heart. The cardioaccelerator center also sends additional fibers, forming the cardiac nerves via sympathetic ganglia (the cervical ganglia plus superior thoracic ganglia T1–T4) to both the SA and AV nodes, plus additional fibers to the atria and ventricles. The ventricles are more richly innervated by sympathetic fibers than parasympathetic fibers. Sympathetic stimulation causes the release of the neurotransmitter norepinephrine (NE) at the neuromuscular junction of the cardiac nerves. NE shortens the repolarization period, thus speeding the rate of depolarization and contraction, which results in an increase in HR. It opens chemical- or ligand-gated sodium and calcium ion channels, allowing an influx of positively charged ions.

NE binds to the beta-1 receptor. Some cardiac medications (for example, beta blockers) work by blocking these receptors, thereby slowing HR and are one possible treatment for hypertension. Overprescription of these drugs may lead to bradycardia and even stoppage of the heart.

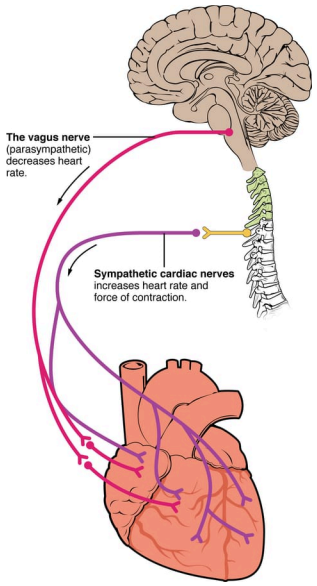


Figure 6.4.2: Cardioaccelerator and cardioinhibitory areas are components of the paired cardiac centers located in the medulla oblongata of the brain. They innervate the heart via sympathetic cardiac nerves that increase cardiac activity and vagus (parasympathetic) nerves that slow cardiac activity. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.4.2.

Parasympathetic stimulation originates from the cardioinhibitory region with impulses traveling via the vagus nerve (cranial nerve X). The vagus nerve sends branches to both the SA and AV nodes, and to portions of both the atria and ventricles. Parasympathetic stimulation releases the neurotransmitter acetylcholine (ACh) at the neuromuscular junction. ACh slows HR by opening chemical- or ligand-gated potassium ion channels to slow the rate of spontaneous depolarization, which extends repolarization and increases the time before the next spontaneous depolarization occurs. Without any nervous stimulation, the SA

node would establish a sinus rhythm of approximately 100 bpm. Since resting rates are considerably less than this, it becomes evident that parasympathetic stimulation normally slows HR. This is similar to an individual driving a car with one foot on the brake pedal. To speed up, one need merely remove one's foot from the break and let the engine increase speed. In the case of the heart, decreasing parasympathetic stimulation decreases the release of ACh, which allows HR to increase up to approximately 100 bpm. Any increases beyond this rate would require sympathetic stimulation. Figure 6.4.3 illustrates the effects of parasympathetic and sympathetic stimulation on the normal sinus rhythm.

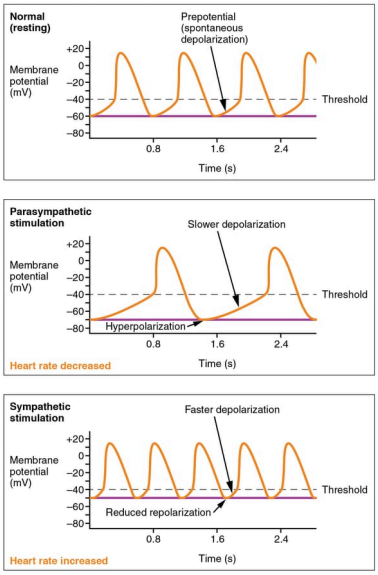


Figure 6.4.3: The wave of depolarization in a normal sinus rhythm shows a stable resting HR. Following parasympathetic stimulation, HR slows. Following sympathetic stimulation, HR increases. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.4.3.

Input to the Cardiovascular Center

The cardiovascular center receives input from a series of visceral receptors with impulses traveling through visceral sensory fibers within the vagus and sympathetic nerves via the cardiac plexus. Among these receptors are various proprioreceptors, baroreceptors, and chemoreceptors, plus stimuli from the limbic system. Collectively, these inputs normally enable the cardiovascular centers to regulate heart function precisely, a process known as cardiac reflexes. Increased physical activity results in increased rates of firing by various proprioreceptors located in muscles, joint capsules, and tendons. Any such increase in physical activity would logically warrant increased blood flow. The cardiac centers monitor these increased rates of firing, and suppress parasympathetic stimulation and increase sympathetic stimulation as needed in order to increase blood flow.

Similarly, baroreceptors are stretch receptors located in the aortic sinus, carotid bodies, the venae cavae, and other locations, including pulmonary vessels and the right side of the heart itself. Rates of firing from the baroreceptors represent blood pressure, level of physical activity, and the relative distribution of blood. The cardiac centers monitor baroreceptor firing to maintain cardiac homeostasis, a mechanism called the baroreceptor reflex. With increased pressure and stretch, the rate of baroreceptor firing increases, and the cardiac centers decrease sympathetic stimulation and increase parasympathetic stimulation. As pressure and stretch decrease, the rate of baroreceptor firing decreases, and the cardiac centers increase sympathetic stimulation and decrease parasympathetic stimulation.

There is a similar reflex, called the atrial reflex or Bainbridge reflex, associated with varying rates of blood flow to the atria. Increased venous return stretches the walls of the atria where specialized baroreceptors are located. However, as the atrial baroreceptors increase their rate of firing and as they stretch due to the increased blood pressure, the cardiac center responds by increasing sympathetic stimulation and inhibiting parasympathetic stimulation to increase HR. The opposite is also true.

Increased metabolic byproducts associated with increased activity, such as carbon dioxide, hydrogen ions, and lactic acid, plus falling oxygen levels, are detected by a suite of chemoreceptors innervated by the glossopharyngeal and vagus nerves. These chemoreceptors provide feedback to the cardiovascular centers about the need for increased or decreased blood flow, based on the relative levels of these substances.

The limbic system can also significantly impact HR related to emotional state. During periods of stress, it is not unusual to

identify higher than normal HRs, often accompanied by a surge in the stress hormone cortisol. Individuals experiencing extreme anxiety may manifest panic attacks with symptoms that resemble those of heart attacks. These events are typically transient and treatable. Meditation techniques have been developed to ease anxiety and have been shown to lower HR effectively. Doing simple deep and slow breathing exercises with one's eyes closed can also significantly reduce this anxiety and HR.

Disorders of the Heart: Broken Heart Syndrome

Extreme stress from such life events as the death of a loved one, an emotional break up, loss of income, or foreclosure of a home may lead to a condition commonly referred to as broken heart syndrome. This condition may also be called Takotsubo cardiomyopathy, transient apical ballooning syndrome, apical ballooning cardiomyopathy, stress-induced cardiomyopathy, Gebrochenes-Herz syndrome, and stress cardiomyopathy. The recognized effects on the heart include congestive heart failure due to a profound weakening of the myocardium not related to lack of oxygen. This may lead to acute heart failure, lethal arrhythmias, or even the rupture of a ventricle. The exact etiology is not known, but several factors have been suggested, including transient vasospasm, dysfunction of the cardiac capillaries, or thickening of the myocardium—particularly in the left ventricle—that may lead to the critical circulation of blood to this region. While many patients survive the initial acute event with treatment to restore normal function, there is a strong correlation with death. Careful statistical analysis by the Jaap Spreeuw and Iqbal Owadally of Bayes Business School in London, revealed that within one year of the death of a loved one, women are more than twice as likely to die and men are six times as likely to die as would otherwise be expected. The same study indicated that longer relationships also had a greater incidence of a person dying soon after the death of their spouse.

Other Factors Influencing Heart Rate

Using a combination of autorhythmicity and innervation, the cardiovascular center is able to provide relatively precise control over HR. However, there are a number of other factors that have an impact on HR as well, including epinephrine, NE, and thyroid hormones; levels of various ions including calcium, potassium, and sodium; body temperature; hypoxia; and pH balance (Table 6.4.1 and Table 6.4.2). After reading this section, the importance of maintaining homeostasis should become even more apparent.

Table 6.4.1: Major Factors Increasing Heart Rate and Force of Contraction

Factor	Effect
Cardioaccelerator nerves	Release of norepinephrine by cardioaccelerator nerves
Proprioreceptors	Increased firing rates of proprioreceptors (e.g. during exercise)
Chemoreceptors	Chemoreceptors sensing decreased levels of O_2 or increased levels of H^+, CO_2 and lactic acid
Baroreceptors	Decreased firing rates of baroreceptors (indicating falling blood volume/pressure)
Limbic system	Anticipation of physical exercise or strong emotions by the limbic system
Catecholamines	Decreased epinephrine and norepinephrine release by the adrenal glands
Thyroid hormones	Increased T_3 and T_4 in the blood (released by thyroid)
Calcium	Decrease in calcium ions in the blood
Potassium	Decrease in potassium ions in the blood
Sodium	Decrease in sodium ions in the blood
Body temperature	Increase in body temperature
Nicotine and caffeine	Presence of nicotine, caffeine or other stimulants

Table 6.4.2: Factors Decreasing Heart Rate and Force of Contraction

Factor	Effect
Cardioinhibitor nerves (vagus)	Release of acetylcholine by cardioinhibitor nerves
Proprioreceptors	Decreased firing rates of proprioreceptors (e.g. during rest)

Factor	Effect
Chemoreceptors	Chemoreceptors sensing increased levels of O_2 or decreased levels of H^+, CO_2 and lactic acid
Baroreceptors	Increased firing rates of baroreceptors (indicating rising blood volume/pressure)
Limbic system	Anticipation of relaxation by the limbic system
Catecholamines	Decreased epinephrine and norepinephrine release by the adrenal glands
Thyroid hormones	Decreased T_3 and T_4 in the blood (released by thyroid)
Calcium	Decrease in calcium ions in the blood
Potassium	Increase in potassium ions in the blood
Sodium	Increase in sodium ions in the blood
Body temperature	Decrease in body temperature
Opiates and tranquilizers	Presence of opiates (heroin), tranquilizers or other depressants

Epinephrine and Norepinephrine

The catecholamines, epinephrine and NE, secreted by the adrenal medulla form one component of the extended fight-or-flight mechanism. The other component is sympathetic stimulation. Epinephrine and NE have similar effects: binding to the beta-1 receptors, and opening sodium and calcium ion chemical- or ligand-gated channels. The rate of depolarization is increased by this additional influx of positively charged ions, so the threshold is reached more quickly and the period of repolarization is shortened. However, massive releases of these hormones coupled with sympathetic stimulation may actually lead to arrhythmias. There is no parasympathetic stimulation to the adrenal medulla.

Thyroid Hormones

In general, increased levels of thyroid hormone, or thyroxin, increase cardiac rate and contractility. The impact of thyroid hormone is typically of a much longer duration than that of the catecholamines. The physiologically active form of thyroid hormone, T_3 or triiodothyronine, has been shown to directly enter cardiomyocytes and alter activity at the level of the genome. It also impacts the beta adrenergic response similar to epinephrine and NE described above. Excessive levels of thyroxin may trigger tachycardia.

Calcium

Calcium ion levels have great impacts upon both HR and contractility; as the levels of calcium ions increase, so do HR and contractility. High levels of calcium ions (hypercalcemia) may be implicated in a short QT interval and a widened T wave in the ECG. The QT interval represents the time from the start of depolarization to repolarization of the ventricles, and includes the period of ventricular systole. Extremely high levels of calcium may induce cardiac arrest. Drugs known as calcium channel blockers slow HR by binding to these channels and blocking or slowing the inward movement of calcium ions.

Caffeine and Nicotine

Caffeine and nicotine are not found naturally within the body. Both of these nonregulated drugs have an excitatory effect on membranes of neurons in general and have a stimulatory effect on the cardiac centers specifically, causing an increase in HR. Caffeine works by increasing the rates of depolarization at the SA node, whereas nicotine stimulates the activity of the sympathetic neurons that deliver impulses to the heart.

Although it is the world's most widely consumed psychoactive drug, caffeine is legal and not regulated. While precise quantities have not been established, "normal" consumption is not considered harmful to most people, although it may cause disruptions to sleep and acts as a diuretic. Its consumption by pregnant people is cautioned against, although no evidence of negative effects has been confirmed. Tolerance and even physical and mental addiction to the drug result in individuals who routinely consume the substance.

Nicotine, too, is a stimulant and produces addiction. While legal and nonregulated, concerns about nicotine's safety and

documented links to respiratory and cardiac disease have resulted in warning labels on cigarette packages.

Factors Decreasing Heart Rate

HR can be slowed when a person experiences altered sodium and potassium levels, hypoxia, acidosis, alkalosis, and hypothermia (see Table 6.4.1). The relationship between electrolytes and HR is complex, but maintaining electrolyte balance is critical to the normal wave of depolarization. Of the two ions, potassium has the greater clinical significance. Initially, both hyponatremia (low sodium levels) and hypernatremia (high sodium levels) may lead to tachycardia. Severely high hypernatremia may lead to fibrillation, which may cause CO to cease. Severe hyponatremia leads to both bradycardia and other arrhythmias. Hypokalemia (low potassium levels) also leads to arrhythmias, whereas hyperkalemia (high potassium levels) causes the heart to become weak and flaccid, and ultimately to fail.

Acidosis is a condition in which excess hydrogen ions are present, and the patient's blood expresses a low pH value. Alkalosis is a condition in which there are too few hydrogen ions, and the patient's blood has an elevated pH. Normal blood pH falls in the range of 7.35–7.45, so a number lower than this range represents acidosis and a higher number represents alkalosis. Recall that enzymes are the regulators or catalysts of virtually all biochemical reactions; they are sensitive to pH and will change shape slightly with values outside their normal range. These variations in pH and accompanying slight physical changes to the active site on the enzyme decrease the rate of formation of the enzyme-substrate complex, subsequently decreasing the rate of many enzymatic reactions, which can have complex effects on HR. Severe changes in pH will lead to denaturation of the enzyme.

The last variable is body temperature. Elevated body temperature is called hyperthermia, and suppressed body temperature is called hypothermia. Slight hyperthermia results in increasing HR and strength of contraction. Hypothermia slows the rate and strength of heart contractions. This distinct slowing of the heart is one component of the larger diving reflex that diverts blood to essential organs while submerged. If sufficiently chilled, the heart will stop beating, a technique that may be employed during open heart surgery. In this case, the patient's blood is normally diverted to an artificial heart-lung machine to maintain the body's blood supply and gas exchange until the surgery is complete, and sinus rhythm can be restored. Excessive hyperthermia and hypothermia will both result in death, as enzymes drive the body systems to cease normal function, beginning with the central nervous system.

Stroke Volume

Many of the same factors that regulate HR also impact cardiac function by altering SV. While a number of variables are involved, SV is ultimately dependent upon the difference between EDV and ESV. The three primary factors to consider are preload, or the stretch on the ventricles prior to contraction; the contractility, or the force or strength of the contraction itself; and afterload, the force the ventricles must generate to pump blood against the resistance in the vessels. These factors are summarized in Table 6.4.1 and Table 6.4.2

Preload

Preload is another way of expressing EDV. Therefore, the greater the EDV is, the greater the preload is. One of the primary factors to consider is filling time, or the duration of ventricular diastole during which filling occurs. The more rapidly the heart contracts, the shorter the filling time becomes, and the lower the EDV and preload are. This effect can be partially overcome by increasing the second variable, contractility, and raising SV, but over time, the heart is unable to compensate for decreased filling time, and preload also decreases.

With increasing ventricular filling, both EDV or preload increase, and the cardiac muscle itself is stretched to a greater degree. At rest, there is little stretch of the ventricular muscle, and the sarcomeres remain short. With increased ventricular filling, the ventricular muscle is increasingly stretched and the sarcomere length increases. As the sarcomeres reach their optimal lengths, they will contract more powerfully, because more of the myosin heads can bind to the actin on the thin filaments, forming cross bridges and increasing the strength of contraction and SV. If this process were to continue and the sarcomeres stretched beyond their optimal lengths, the force of contraction would decrease. However, due to the physical constraints of the location of the heart, this excessive stretch is not a concern.

The relationship between ventricular stretch and contraction has been stated in the well-known Frank-Starling mechanism or simply Starling's Law of the Heart. This principle states that, within physiological limits, the force of heart contraction is directly proportional to the initial length of the muscle fiber. This means that the greater the stretch of the ventricular muscle (within limits), the more powerful the contraction is, which in turn increases SV. Therefore, by increasing preload, you increase the second

variable, contractility.

Otto Frank (1865–1944) was a German physiologist; among his many published works are detailed studies of this important heart relationship. Ernest Starling (1866–1927) was an important English physiologist who also studied the heart. Although they worked largely independently, their combined efforts and similar conclusions have been recognized in the name “Frank-Starling mechanism.”

Any sympathetic stimulation to the venous system will increase venous return to the heart, which contributes to ventricular filling, and EDV and preload. While much of the ventricular filling occurs while both atria and ventricles are in diastole, the contraction of the atria, the atrial kick, plays a crucial role by providing the last 20–30 percent of ventricular filling.

Contractility

It is virtually impossible to consider preload or ESV without including an early mention of the concept of contractility. Indeed, the two parameters are intimately linked. Contractility refers to the force of the contraction of the heart muscle, which controls SV, and is the primary parameter for impacting ESV. The more forceful the contraction is, the greater the SV and smaller the ESV are. Less forceful contractions result in smaller SVs and larger ESVs. Factors that increase contractility are described as positive inotropic factors, and those that decrease contractility are described as negative inotropic factors (ino- = “fiber;” -tropic = “turning toward”).

Not surprisingly, sympathetic stimulation is a positive inotrope, whereas parasympathetic stimulation is a negative inotrope. Sympathetic stimulation triggers the release of NE at the neuromuscular junction from the cardiac nerves and also stimulates the adrenal cortex to secrete epinephrine and NE. In addition to their stimulatory effects on HR, they also bind to both alpha and beta receptors on the cardiac muscle cell membrane to increase metabolic rate and the force of contraction. This combination of actions has the net effect of increasing SV and leaving a smaller residual ESV in the ventricles. In comparison, parasympathetic stimulation releases ACh at the neuromuscular junction from the vagus nerve. The membrane hyperpolarizes and inhibits contraction to decrease the strength of contraction and SV, and to raise ESV. Since parasympathetic fibers are more widespread in the atria than in the ventricles, the primary site of action is in the upper chambers. Parasympathetic stimulation in the atria decreases the atrial kick and reduces EDV, which decreases ventricular stretch and preload, thereby further limiting the force of ventricular contraction. Stronger parasympathetic stimulation also directly decreases the force of contraction of the ventricles.

Several synthetic drugs, including dopamine and isoproterenol, have been developed that mimic the effects of epinephrine and NE by stimulating the influx of calcium ions from the extracellular fluid. Higher concentrations of intracellular calcium ions increase the strength of contraction. Excess calcium (hypercalcemia) also acts as a positive inotropic agent. The drug digitalis lowers HR and increases the strength of the contraction, acting as a positive inotropic agent by blocking the sequestering of calcium ions into the sarcoplasmic reticulum. This leads to higher intracellular calcium levels and greater strength of contraction. In addition to the catecholamines from the adrenal medulla, other hormones also demonstrate positive inotropic effects. These include thyroid hormones and glucagon from the pancreas.

Negative inotropic agents include hypoxia, acidosis, hyperkalemia, and a variety of synthetic drugs. These include numerous beta blockers and calcium channel blockers. Early beta blocker drugs include propranolol and pronethalol, and are credited with revolutionizing treatment of cardiac patients experiencing angina pectoris. There is also a large class of dihydropyridine, phenylalkylamine, and benzothiazepine calcium channel blockers that may be administered decreasing the strength of contraction and SV.

Afterload

Afterload refers to the tension that the ventricles must develop to pump blood effectively against the resistance in the vascular system. Any condition that increases resistance requires a greater afterload to force open the semilunar valves and pump the blood. Damage to the valves, such as stenosis, which makes them harder to open will also increase afterload. Any decrease in resistance decreases the afterload. Figure 6.4.4 summarizes the major factors influencing SV, Figure 6.4.5 summarizes the major factors influencing CO, and Table 6.4.3 and Table 6.4.4 summarize cardiac responses to increased and decreased blood flow and pressure in order to restore homeostasis.

	Factors Affecting Stroke Volume (SV)		
	Preload	**Contractility**	**Afterload**
Raised due to:	• increased filling time • increased venous return **Increases end diastolic volume,** **Increases stroke volume**	• sympathetic stimulation • epinephrine and norepinephrine • high intracellular calcium ions • high blood calcium level • thyroid hormones • glucagon **Decreases end systolic volume,** **Increases stroke volume**	• increased vascular resistance • semilunar valve damage **Increases end systolic volume** **Decreases stroke volume**
Lowered due to:	• decreased thyroid hormones • decreased calcium ions • high or low potassium ions • high or low sodium • low body temperature • hypoxia • abnormal pH balance • drugs (i.e., calcium channel blockers) **Decreases end diastolic volume,** **Decreases stroke volume**	• parasympathetic stimulation • acetylcholine • hypoxia • hyperkalemia **Increases end systolic volume** **Decreases stroke volume**	• decreased vascular resistance **Decreases end systolic volume** **Increases stroke volume**

Figure 6.4.4: Multiple factors impact preload, afterload, and contractility, and are the major considerations influencing SV. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.4.4.

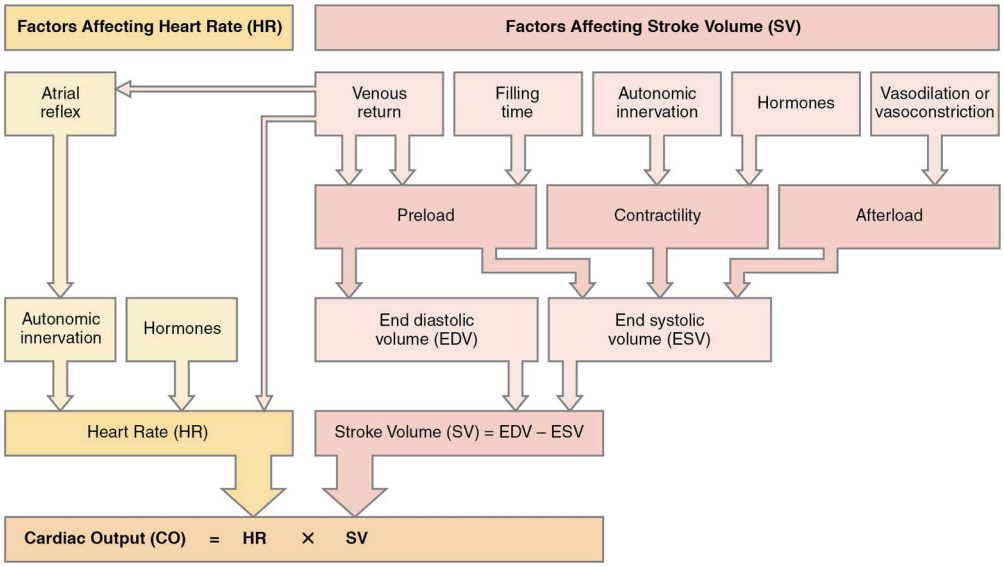


Figure 6.4.5: The primary factors influencing HR include autonomic innervation plus endocrine control. Not shown are environmental factors, such as electrolytes, metabolic products, and temperature. The primary factors controlling SV include preload, contractility, and afterload. Other factors such as electrolytes may be classified as either positive or negative inotropic agents. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.4.5.

Table 6.4.3: Cardiac Response to Decreasing Blood Flow and Pressure Due to Decreasing Cardiac Output

Response	Baroreceptors (aorta, carotid arteries, venae cavae, and atria)	Chemoreceptors (both central nervous system and in proximity to baroreceptors)
Sensitive to	Decreasing stretch	Decreasing O_2 and increasing CO_2, H^+, and lactic acid
Target	Parasympathetic stimulation suppressed	Sympathetic stimulation increased
Response of heart	Increasing heart rate and increasing stroke volume	Increasing heart rate and increasing stroke volume
Overall effect	Increasing blood flow and pressure due to increasing cardiac output; homeostasis restored	Increasing blood flow and pressure due to increasing cardiac output; homeostasis restored

Table 6.4.4: Cardiac Response to Increasing Blood Flow and Pressure Due to Increasing Cardiac Output

Response Component	Baroreceptors (aorta, carotid arteries, venae cavae, and atria)	Chemoreceptors (both central nervous system and in proximity to baroreceptors)
Sensitive to	Increasing stretch	Increasing O_2 and decreasing CO_2, H^+, and lactic acid
Target	Parasympathetic stimulation increased	Sympathetic stimulation suppressed
Response of heart	Decreasing heart rate and decreasing stroke volume	Decreasing heart rate and decreasing stroke volume
Overall effect	Decreasing blood flow and pressure due to decreasing cardiac output; homeostasis restored	Decreasing blood flow and pressure due to decreasing cardiac output; homeostasis restored

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- 15.2: Glycogenesis by Henry Jakubowski and Patricia Flatt has no license indicated.

6.5: Development of the Heart

Fetal Heart Development

The human heart is the first functional organ to develop. It begins beating and pumping blood around day 21 or 22, a mere three weeks after fertilization. This emphasizes the critical nature of the heart in distributing blood through the vessels and the vital exchange of nutrients, oxygen, and wastes both to and from the developing baby. The critical early development of the heart is reflected by the prominent heart bulge that appears on the anterior surface of the embryo.

The heart forms from an embryonic tissue called mesoderm around 18 to 19 days after fertilization. Mesoderm is one of the three primary germ layers that differentiates early in development that collectively gives rise to all subsequent tissues and organs. The heart begins to develop near the head of the embryo in a region known as the cardiogenic area. Following chemical signals called factors from the underlying endoderm (another of the three primary germ layers), the cardiogenic area begins to form two strands called the cardiogenic cords (Figure 6.5.1). As the cardiogenic cords develop, a lumen rapidly develops within them. At this point, they are referred to as endocardial tubes. The two tubes migrate together and fuse to form a single primitive heart tube. The primitive heart tube quickly forms five distinct regions. From head to tail, these include the truncus arteriosus, bulbus cordis, primitive ventricle, primitive atrium, and the sinus venosus. Initially, all venous blood flows into the sinus venosus, and contractions propel the blood from tail to head, or from the sinus venosus to the truncus arteriosus. This is a very different pattern from that of an adult.

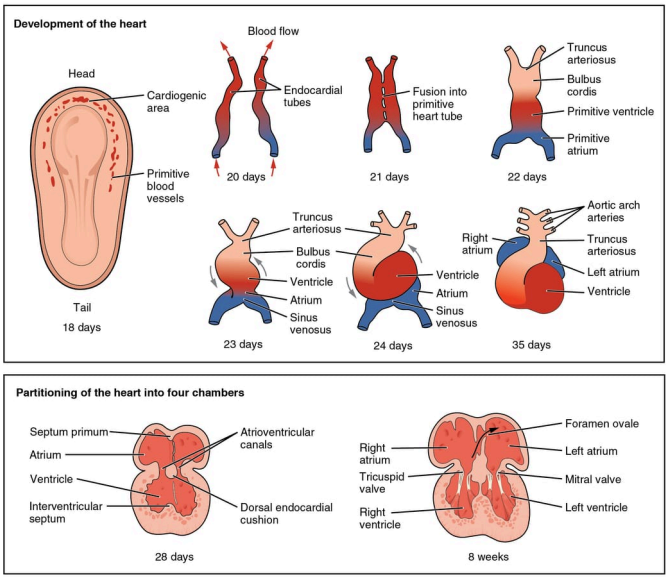


Figure 6.5.1: This diagram outlines the embryological development of the human heart during the first eight weeks and the subsequent formation of the four heart chambers. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 6.5.1.

The five regions of the primitive heart tube develop into recognizable structures in a fully developed heart. The truncus arteriosus will eventually divide and give rise to the ascending aorta and pulmonary trunk. The bulbus cordis develops into the right ventricle. The primitive ventricle forms the left ventricle. The primitive atrium becomes the anterior portions of both the right and left atria, and the two auricles. The sinus venosus develops into the posterior portion of the right atrium, the SA node, and the coronary sinus.

As the primitive heart tube elongates, it begins to fold within the pericardium, eventually forming an S shape, which places the chambers and major vessels into an alignment similar to the adult heart. This process occurs between days 23 and 28. The remainder of the heart development pattern includes development of septa and valves, and remodeling of the actual chambers. Partitioning of the atria and ventricles by the interatrial septum, interventricular septum, and atrioventricular septum is complete by the end of the fifth week, although the fetal blood shunts remain until birth or shortly after. The atrioventricular valves form between weeks five and eight, and the semilunar valves form between weeks five and nine.

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6.6: Terms and Definitions

Key Terms

Select to View Definition

Afterload
: force the ventricles must develop to effectively pump blood against the resistance in the vessels

Anastomosis
: (plural = anastomoses) area where vessels unite to allow blood to circulate even if there may be partial blockage in another branch

Anterior Cardiac Veins
: vessels that parallel the small cardiac arteries and drain the anterior surface of the right ventricle; bypass the coronary sinus and drain directly into the right atrium

Anterior Interventricular Artery
: (also, left anterior descending artery or LAD) major branch of the left coronary artery that follows the anterior interventricular sulcus

Anterior Interventricular Sulcus
: sulcus located between the left and right ventricles on the anterior surface of the heart

Aortic Valve
: (also, aortic semilunar valve) valve located at the base of the aorta

Artificial Pacemaker
: medical device that transmits electrical signals to the heart to ensure that it contracts and pumps blood to the body

Atrial Reflex
: (also called Bainbridge reflex) autonomic reflex that responds to stretch receptors in the atria that send impulses to the cardioaccelerator area to increase HR when venous flow into the atria increases

Atrioventricular (AV) Node
: clump of myocardial cells located in the inferior portion of the right atrium within the atrioventricular septum; receives the impulse from the SA node, pauses, and then transmits it into specialized conducting cells within the interventricular septum

Atrioventricular Bundle
: (also, bundle of His) group of specialized myocardial conductive cells that transmit the impulse from the AV node through the interventricular septum and form the left and right atrioventricular bundle branches

Atrioventricular Bundle Branches
: (also, left or right bundle branches) specialized myocardial conductive cells that arise from the bifurcation of the atrioventricular bundle and pass through the interventricular septum; lead to the Purkinje fibers and also to the right papillary muscle via the moderator band

Atrioventricular Septum
: cardiac septum located between the atria and ventricles; atrioventricular valves are located here

Atrioventricular Valves
: one-way valves located between the atria and ventricles; the valve on the right is the tricuspid valve and the one on the left is the mitral or bicuspid valve

Atrium
(plural = atria) upper or receiving chamber of the heart that pumps blood into the lower chambers just prior to their contraction; the right atrium receives blood from the systemic circuit and the left atrium receives blood from the pulmonary circuit

Auricle
extension of an atrium visible on the superior surface of the heart

Autonomic Tone
contractile state during resting cardiac activity produced by mild sympathetic and parasympathetic stimulation

Autorhythmicity
ability of cardiac muscle to initiate its own electrical impulse that triggers contraction without nervous or endocrine control

Bachmann's Bundle
(also, interatrial band) group of specialized conducting cells that transmit the impulse directly from the SA node in the right atrium to the left atrium

Bainbridge Reflex
(also called atrial reflex) autonomic reflex that responds to stretch receptors in the atria that send impulses to the cardioaccelerator area to increase HR when venous flow into the atria increases

Coronary Sinus
large, thin-walled vein on the posterior surface of the heart that lies within the atrioventricular sulcus and drains the heart myocardium directly into the right atrium

Coronary Sulcus
sulcus that marks the boundary between the atria and ventricles

Coronary Veins
vessels that drain the heart and generally parallel the large surface arteries

Diastole
period of time when the heart muscle is relaxed and the chambers fill with blood

Ejection Fraction
portion of the blood that is pumped or ejected from the heart with each contraction; mathematically represented by SV divided by EDV

Electrocardiogram (ECG)
surface recording of the electrical activity of the heart that can be used for diagnosis of irregular heart function; also abbreviated as EKG

End Diastolic Volume (EDV)
(also, preload) amount of blood in the ventricles at the end of atrial systole just prior to ventricular contraction

End Systolic Volume (ESV)
amount of blood remaining in each ventricle following systole

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6.7: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. Which structure is most directly responsible for preventing backflow of blood from a ventricle into an atrium?
   A. Endocardium
   B. AV (atrioventricular) valves
   C. Aortic arch
   D. Coronary sinus

2. Which valve separates the left atrium from the left ventricle?
   A. Mitral (bicuspid) valve
   B. Tricuspid valve
   C. Pulmonary semilunar valve
   D. Aortic semilunar valve

3. In correct order, which valves does a red blood cell pass as it travels from the right atrium to the aorta?
   A. Tricuspid → pulmonary semilunar → mitral → aortic semilunar
   B. Tricuspid → aortic semilunar → mitral → pulmonary semilunar
   C. Mitral → tricuspid → pulmonary semilunar → aortic semilunar
   D. Pulmonary semilunar → tricuspid → aortic semilunar → mitral

4. Which heart chamber receives blood directly from the systemic circulation (via the venae cavae)?
   A. Left atrium
   B. Left ventricle
   C. Right atrium
   D. Right ventricle

5. In which chamber is the myocardium (muscle layer) the thickest?
   A. Left atrium
   B. Left ventricle
   C. Right atrium
   D. Right ventricle

6. Which layer of the heart wall is in direct contact with the blood in the chambers?
   A. Epicardium (visceral pericardium)
   B. Myocardium
   C. Endocardium
   D. Fibrous pericardium

7. Cardiac muscle cells are able to generate their own action potentials in some regions of the heart. What is this property called?
   A. Contractility
   B. Autorhythmicity
   C. Refractoriness
   D. Plasticity

8. The plateau phase of the cardiac contractile cell action potential is mainly caused by influx of which ion?
   A. Sodium (Na^+)
   B. Potassium (K^+)
   C. Calcium (Ca^{2+})
   D. Chloride (Cl^-)

9. Which wave or complex of the ECG represents ventricular depolarization?
   A. P wave
   B. QRS complex
   C. T wave
   D. U wave

10. Which statement best explains why atrial repolarization is not seen as a separate wave on a typical ECG tracing?
    A. It occurs at the same time as ventricular depolarization and is hidden in the QRS complex.
    B. It is too small to be recorded by surface electrodes.
    C. It occurs only during sleep.
    D. It takes place after the T wave.

11. Which part of the cardiac conduction system normally has the fastest intrinsic firing rate and acts as the main pacemaker?
    A. Atrioventricular (AV) node
    B. Purkinje fibers
    C. Sinoatrial (SA) node
    D. Bundle branches

12. During which phase of the cardiac cycle are the ventricles contracting while all valves are closed and no blood is being ejected?
    A. Atrial systole
    B. Isovolumic (isovolumetric) contraction
    C. Ventricular ejection
    D. Ventricular filling

13. Most of the blood that fills the ventricles enters during which phase?
    A. Atrial systole (atrial contraction)
    B. Passive ventricular filling during atrial diastole
    C. Ventricular systole
    D. Isovolumic relaxation

14. The first heart sound ("lub," S_1) is mainly produced by which event?
    A. Closing of the semilunar valves
    B. Closing of the AV (mitral and tricuspid) valves
    C. Opening of the AV valves
    D. Opening of the semilunar valves

15. Ventricular relaxation (the beginning of ventricular diastole) begins immediately after which electrical event?
    A. Atrial depolarization
    B. Ventricular depolarization
    C. Ventricular repolarization
    D. Atrial repolarization

16. The term "afterload" refers to:
    A. The amount of blood in the ventricle at the end of filling
    B. The resistance the ventricle must overcome to eject blood into the arteries
    C. The number of heartbeats per minute
    D. The volume of blood pumped per minute

17. Which brain region contains the main cardiovascular centers that help control heart rate and blood vessel diameter?
    A. Cerebral cortex
    B. Cerebellum
    C. Medulla oblongata

D. Hypothalamus

18. In a healthy young adult, what usually happens to cardiac output when heart rate rises above about 160 beats per minute?
A. It increases steadily as heart rate increases.
B. It decreases because there is not enough time for the ventricles to fill.
C. It stays exactly the same.
D. It becomes zero.

19. Venoconstriction (constriction of systemic veins) has what main effect on preload?
A. Decreases preload
B. Increases preload
C. Has no effect on preload
D. First decreases, then increases preload

20. A 22-year-old athlete has a resting heart rate of 48 beats per minute and a stroke volume of 100 mL per beat. A non-athletic friend has a resting heart rate of 72 beats per minute and a stroke volume of 70 mL per beat. Which statement about their resting cardiac outputs is correct?
A. The athlete's cardiac output is lower than his friend's.
B. The athlete's cardiac output is higher than his friend's.
C. The two cardiac outputs are approximately the same.
D. Cardiac output cannot be estimated from heart rate and stroke volume.

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6.8: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. The left ventricular wall is much thicker than the right ventricular wall. Explain why this difference in muscle thickness is necessary for normal blood flow in the body.
   Hint: Think about where each ventricle pumps blood (near vs. far, low vs. high pressure) and how much force is needed.

2. The atrioventricular (AV) valves (tricuspid and mitral) and the semilunar valves (aortic and pulmonary) open and close at different times in the cardiac cycle. Explain how proper timing of these valves prevents backflow and supports efficient pumping.
   Hint: Imagine what should happen to blood when the ventricles relax vs. when they contract. When should each type of valve be open or closed?

3. A blockage in the left coronary artery can damage the left ventricle. Explain how this damage might affect cardiac output and the person's ability to exercise.
   Hint: Remember that cardiac output = heart rate × stroke volume. How would a weak left ventricle change stroke volume?

4. The sinoatrial (SA) node is often called the "pacemaker" of the heart. Describe what might happen to heart rhythm and cardiac output if the SA node stops working and another part of the conduction system takes over.
   Hint: Think about changes in heart rate (faster or slower?) and how regular or irregular the beats might become.

5. During exercise, both heart rate and stroke volume usually increase. Explain how each change (HR and SV) helps to meet the higher oxygen needs of the muscles.
   Hint: Use the formula for cardiac output and think about how quickly and how strongly the heart must pump.

6. A patient has a leaky mitral valve (mitral regurgitation), so some blood flows backward into the left atrium during ventricular contraction. Explain how this could affect stroke volume and cardiac output over time.
   Hint: Consider how much blood actually leaves the left ventricle into the aorta vs. how much is lost backward.

7. Compare the effects on cardiac output of:
   a) a sudden, large loss of blood (for example, from trauma), and
   b) a very fast heart rate (severe tachycardia) with normal blood volume.
   Explain how each situation can reduce cardiac output.
   Hint: For blood loss, think about filling of the heart (preload). For very fast HR, think about filling time between beats.

8. The Frank–Starling law of the heart says that the more the heart muscle is stretched by filling (to a point), the stronger the contraction will be. Explain how this helps the heart automatically adjust cardiac output when venous return increases.
   Hint: Think about what happens when more blood returns to the heart from the veins. How does the ventricle respond without

changing nerve input?

9. The autonomic nervous system can change heart rate and contractility. Describe one situation where sympathetic stimulation of the heart is helpful, and one situation where long-term excessive sympathetic activity could be harmful.
Hint: Think about short-term needs (stress, exercise) vs. long-term effects on workload, oxygen demand, and possible heart damage.

10. A person has a narrowed (stenotic) aortic valve, so it is harder for blood to leave the left ventricle. Explain how this increased resistance affects left ventricular work and may change cardiac output at rest and during exercise.
Hint: Consider how much pressure the ventricle must generate, how this affects muscle thickness, and whether it can keep up during higher demands.

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6.9: Alternate Text Descriptions

Figure 6.1.1: Heart Position Within the Mediastinum and Thoracic Cavity

This figure illustrates the location of the **heart** within the **thoracic cavity** and its relationship to surrounding anatomical structures.

1. In the frontal view, the heart is shown within the **mediastinum**, the central compartment of the thorax located between the right and left lungs.
2. The heart is enclosed by the **pericardial cavity** and rests superior to the **diaphragm**.
3. The lungs occupy the spaces on either side of the mediastinum, demonstrating that the heart lies between the lungs rather than within either lung.
4. The accompanying sagittal view shows the mediastinum from the side and highlights the heart's position relative to other thoracic structures.
5. The heart lies inferior to the **trachea** and **thymus**, anterior to the **esophagus** and **thoracic aorta**, and superior to the **diaphragm**.
6. Together, the two views demonstrate how the mediastinum serves as the central region of the thoracic cavity that contains the heart and other major vessels and organs.

The instructional purpose of this figure is to show the anatomical location of the heart within the mediastinum and emphasize its spatial relationships to the lungs, diaphragm, and neighboring thoracic structures.

Figure 6.1.2: Surface Anatomy of the Heart and Anatomical Landmarks for Cardiopulmonary Resuscitation (CPR)

This figure demonstrates the anatomical relationship between the **heart**, **sternum**, and **vertebral column** and identifies the proper hand position for performing chest compressions during **cardiopulmonary resuscitation, or CPR**.

1. The illustration shows a side view of the head, neck, and thorax with the **cervical** and **upper thoracic vertebrae** labeled.
2. The heart is positioned within the thoracic cavity **posterior to the sternum** and **anterior to the vertebral column**.
3. Dashed vertical reference lines indicate the superior and inferior boundaries of the heart's projection onto the chest wall.
4. A transparent overlay of a hand is shown resting on the anterior chest over the **lower half of the sternum**.
5. The image emphasizes that chest compressions are applied to the **sternum directly above the heart**, rather than over the neck, upper chest, or abdomen.
6. The relationship between the hand placement and the underlying heart demonstrates how external compressive force is transmitted through the sternum to the heart during CPR.

The primary instructional purpose of the figure is to show the location of the heart within the thorax and to illustrate the anatomical landmark used to position the hands for effective chest compressions during CPR. By aligning the hand placement with the surface anatomy of the chest, the figure helps learners understand where compressive force should be applied to maximize circulation during resuscitation efforts.

Figure 6.1.3: Pulmonary and Systemic Circulation Through the Heart and Body

This figure illustrates the pathway of blood flow through the **heart**, **lungs**, and **body**, highlighting the relationship between **pulmonary circulation** and **systemic circulation**.

1. The lower portion of the figure shows the two interconnected circulatory circuits.
2. **Oxygen-poor blood**, represented in blue, returns from the upper and lower body through **systemic veins** and enters the **right atrium**.
3. Blood then flows into the **right ventricle** and is pumped through the **pulmonary trunk** and **pulmonary arteries** to the **lungs**.

4. Within the **pulmonary capillaries** of the lungs, gas exchange occurs, adding oxygen to the blood and removing carbon dioxide.
5. **Oxygen-rich blood**, represented in red, returns from the lungs through the **pulmonary veins** and enters the **left atrium**.
6. Blood then flows into the **left ventricle**, which pumps it into the **aorta** and **systemic arteries**.
7. The blood is distributed to capillary networks throughout the upper and lower body, where oxygen and nutrients are delivered to tissues before the blood returns to the heart through the systemic veins.
8. An enlarged inset of the heart identifies the major chambers, valves, and blood vessels involved in this circulation pathway.
9. The right side of the heart receives oxygen-poor blood from the **superior vena cava** and **inferior vena cava** and pumps it to the lungs through the **pulmonary semilunar valve**, **pulmonary trunk**, and **pulmonary arteries**.
10. The left side of the heart receives oxygen-rich blood from the **pulmonary veins** and pumps it through the **mitral valve**, **aortic semilunar valve**, and **aorta** to the body.
11. Arrows indicate the direction of blood flow through the heart and circulatory system.

The instructional purpose of this figure is to demonstrate how blood moves in a continuous cycle between the heart, lungs, and body. It emphasizes that the **right side of the heart** pumps blood to the lungs for oxygenation, while the **left side of the heart** pumps oxygenated blood to the body's tissues through systemic circulation.

Figure 6.1.4: Layers of the Heart Wall and Pericardial Membranes

This figure illustrates the anatomical layers that form the wall of the **heart** and the surrounding **pericardial sac**. An inset of the heart identifies the location from which the enlarged cross-sectional view is taken.

1. The enlarged view shows the layers arranged from the interior of the heart outward.
2. The innermost layer is the **endocardium**, which lines the chambers of the heart.
3. External to the endocardium is the **myocardium**, the thick muscular layer responsible for generating the force of cardiac contraction and pumping blood throughout the circulatory system.
4. Surrounding the heart wall is the **epicardium**, which is also identified as the **visceral layer of the serous pericardium**.
5. External to the epicardium is the **pericardial cavity**, a narrow fluid-filled space that reduces friction as the heart beats.
6. The outer boundary of the cavity is formed by the **parietal layer of the serous pericardium**.
7. The most external layer shown is the **fibrous pericardium**, a tough connective tissue layer that helps stabilize and protect the heart within the thorax.

The instructional purpose of this figure is to demonstrate the structural organization of the heart wall and its surrounding protective membranes. The figure emphasizes the sequence and relationships among the **endocardium**, **myocardium**, **epicardium**, **pericardial cavity**, **parietal serous pericardium**, and **fibrous pericardium**, helping learners understand how these layers contribute to heart function, protection, and friction-free movement during cardiac activity.

Figure 6.1.5: Anterior and Posterior Views of the Heart Showing Major Blood Vessels and Coronary Circulation

This figure presents **anterior, or front,** and **posterior, or back,** views of the **heart** to illustrate its external anatomy, the major blood vessels connected to it, and the **coronary arteries and veins** that supply and drain the heart muscle.

Anterior View

1. The heart is shown with the **right atrium**, **right ventricle**, and **left ventricle** visible on the surface.
2. Major vessels connected to the heart include the:
    - **Superior vena cava**
    - **Inferior vena cava**
    - **Pulmonary trunk**
    - **Right and left pulmonary arteries**
    - **Pulmonary veins**
    - **Ascending aorta**
    - **Aortic arch**
3. Branches arising from the **aortic arch** include the:
    - **Brachiocephalic trunk**

    - **Left common carotid artery**
    - **Left subclavian artery**
4. Coronary vessels visible on the anterior surface include the:
    - **Right coronary artery**
    - **Left coronary artery**
    - **Anterior interventricular artery**
    - **Circumflex artery**
    - **Right marginal artery**
    - **Anterior cardiac vein**
    - **Small cardiac vein**
    - **Great cardiac vein**
5. The **apex of the heart** is identified at the inferior tip.

Posterior View

1. The **left atrium** and portions of both ventricles are visible.
2. The **pulmonary veins** entering the left atrium and the **superior** and **inferior vena cava** entering the right atrium can be seen more clearly from this perspective.
3. The **coronary sinus**, which collects blood from the cardiac veins, is visible on the posterior surface.
4. Additional coronary vessels shown include the:
    - **Posterior interventricular artery**
    - **Great cardiac vein**
    - **Middle cardiac vein**
    - **Posterior vein of the left ventricle**
    - **Circumflex branch of the left coronary artery**
    - **Right coronary artery**

The figure demonstrates that the heart receives its blood supply through **coronary arteries** branching from the **aorta** and returns deoxygenated blood from the heart muscle through **cardiac veins** that drain into the **coronary sinus**. Comparing anterior and posterior views highlights how the arrangement of chambers, great vessels, arteries, and veins differs depending on the viewing angle.

Figure 6.1.6: Myocardial Fiber Orientation in the Atria and Ventricles

This figure illustrates the arrangement of **cardiac muscle fibers** within the walls of the **atria** and **ventricles**. The heart is shown with the outer layers of muscle highlighted to emphasize the direction and organization of **myocardial fibers**.

Atria

1. In the **atria**, the muscle fibers are arranged primarily in **circular patterns** that wrap around the atrial chambers.
2. Curved arrows indicate the orientation of these fibers around each atrium.
3. This arrangement supports coordinated contraction of the atria, helping move blood into the ventricles.

Ventricles

1. In the **ventricles**, the muscle fibers form a more complex **spiral pattern** that wraps around the heart and extends toward the **apex**.
2. Arrows show the **helical orientation** of the ventricular musculature and its changing direction across the ventricular walls.
3. The spiral arrangement allows ventricular contraction to produce a **twisting, or wringing, motion** that efficiently ejects blood from the heart into the **pulmonary trunk** and **aorta**.

The figure contrasts the relatively simple circular fiber organization of the atria with the layered, spiral organization of the ventricles. The differing orientations reflect the distinct functions of the two cardiac regions:

- **Atrial contraction** moves blood into the ventricles.
- **Ventricular contraction** generates the greater force required to propel blood through the pulmonary and systemic

circulatory circuits.

Figure 6.1.7: Changes in Ventricular Shape During Cardiac Contraction

This figure compares the appearance of the **ventricles** in relaxed and contracted states to illustrate how ventricular contraction changes the size and shape of the ventricular chambers during the **cardiac cycle**.

Left Panel: Relaxed

1. The left panel, labeled **Relaxed**, shows the ventricles with relatively large internal chamber volumes.
2. The ventricular walls are less compressed, and the **apex of the heart** appears broader.
3. The relaxed state represents **ventricular diastole**, when the ventricles are filling with blood.

Right Panel: Contracted

1. The right panel, labeled **Contracted**, shows the ventricles after the ventricular muscle fibers have shortened and tightened.
2. The internal ventricular chambers are noticeably smaller, reflecting a reduction in volume as blood is expelled from the heart.
3. Labels identify the **right ventricle** and **left ventricle**.
4. The ventricular walls appear thicker because the **myocardium** is contracted, and the overall shape of the ventricles becomes more compact.
5. The apex is drawn upward and inward, demonstrating the twisting and squeezing action produced by the spiral arrangement of ventricular muscle fibers.

By comparing the two panels, the figure demonstrates that **ventricular contraction** decreases chamber volume while increasing pressure within the ventricles. This pressure forces blood out of the **right ventricle** into the **pulmonary circulation** and out of the **left ventricle** into the **systemic circulation**.

Figure 6.1.8: Internal Anatomy of the Heart (Anterior View)

This figure presents an **anterior view of the heart** with the front wall partially removed to reveal the internal chambers, valves, supporting structures, and major blood vessels. The image is designed to show how blood moves through the heart and how the anatomical structures support that function.

Right Side of the Heart

1. The **right side of the heart** receives oxygen-poor blood from the body through the **superior vena cava** and **inferior vena cava**, which empty into the **right atrium**.
2. A depression in the wall of the right atrium, the **fossa ovalis**, is visible.
3. Blood passes from the right atrium through the **tricuspid valve** into the **right ventricle**.
4. Within the right ventricle, the **chordae tendineae** connect the valve cusps to **papillary muscles**, helping prevent valve prolapse during ventricular contraction.
5. Irregular muscular ridges called **trabeculae carneae** line the ventricular wall, and the **moderator band** extends across the right ventricle.
6. From the right ventricle, blood is pumped through the **pulmonary valve** into the **pulmonary trunk** and then into the **right and left pulmonary arteries**, which carry blood to the lungs.

Left Side of the Heart

1. Oxygen-rich blood returns from the lungs through the **right and left pulmonary veins** and enters the **left atrium**.
2. Blood then flows through the **mitral, or bicuspid, valve** into the **left ventricle**.
3. The **left ventricular wall** is noticeably thicker than the right ventricular wall, reflecting its role in generating the higher pressure required to pump blood throughout the body.
4. Blood exits the left ventricle through the **aortic valve** and enters the **aorta** for systemic distribution.

Shared and Supporting Structures

1. The **interventricular septum** separates the right and left ventricles.
2. The layers of the heart wall are shown, including:
    - The **endocardium**, lining the chambers
    - The **myocardium**, forming the muscular wall
    - The **epicardium**, covering the outer surface of the heart

The instructional purpose of this figure is to help learners identify the major internal structures of the heart and understand how the chambers, valves, supporting tissues, and blood vessels work together to direct **one-way blood flow** through the **pulmonary** and **systemic circulatory circuits**.

Figure 6.1.9: Examples of Congenital Heart Defects Affecting Cardiac Structure and Blood Flow

This figure illustrates four **congenital heart defects** and highlights the structural abnormalities that alter normal blood flow through the heart and major vessels.

(a) Patent Foramen Ovale

1. The upper-left panel shows a **foramen ovale** that remains open after birth.
2. The opening persists in the wall between the **right atrium** and **left atrium**, allowing blood to pass between the atrial chambers.
3. The figure emphasizes the failure of normal postnatal closure of the fetal opening.

(b) Coarctation of the Aorta

1. The upper-right panel shows a localized narrowing of the **aorta**.
2. The narrowed segment reduces the diameter of the vessel through which blood leaves the **left ventricle** for systemic circulation.
3. The figure highlights the site of constriction within the aorta.

(c) Patent Ductus Arteriosus

1. The lower-left panel depicts a **ductus arteriosus** that remains open after birth.
2. This persistent connection links the **pulmonary trunk** and the **aorta**, allowing blood to flow between these vessels when the fetal vessel would normally be closed.
3. Arrows indicate the abnormal pathway through the persistent connection.

(d) Tetralogy of Fallot

1. The lower-right panel shows four structural abnormalities occurring together.
2. These include:
    - Narrowing of the **pulmonary semilunar valve**, or **pulmonary stenosis**
    - An **interventricular septal defect** between the ventricles
    - An **aorta** positioned so that it receives blood from both ventricles
    - Enlargement of the **right ventricle**
3. The figure highlights how these defects alter the normal separation of oxygen-poor and oxygen-rich blood and affect blood flow leaving the heart.

The instructional purpose of this figure is to compare common congenital heart defects and demonstrate how developmental abnormalities can change the structure of the heart and great vessels. The figure emphasizes the specific anatomical changes associated with each condition and their effects on the normal pathways of blood flow through the cardiovascular system.

Figure 6.1.10: Internal Structures of a Ventricle: Chordae Tendineae, Papillary Muscles, and Trabeculae Carneae

This photograph shows the interior of a dissected **ventricle** with key structures involved in ventricular contraction and **atrioventricular valve** function labeled. The heart has been opened to expose the ventricular chamber and its internal muscular features.

1. Thin, cord-like **chordae tendineae** extend from the **atrioventricular valve leaflets** to the ventricular wall.
2. These fibrous cords attach to prominent muscular projections called **papillary muscles**.
3. During ventricular contraction, the papillary muscles tense the chordae tendineae, helping prevent the valve leaflets from prolapsing into the atrium and supporting **one-way blood flow** through the heart.
4. The inner surface of the ventricle contains irregular muscular ridges known as **trabeculae carneae**.
5. These ridges form a textured network along the ventricular wall and contribute to the internal structure of the chamber.
6. The photograph provides a real anatomical example of these internal ventricular components and demonstrates their physical relationships.
7. The chordae tendineae connect the valve apparatus to the papillary muscles, while the trabeculae carneae form the ridged lining of the ventricular wall.

The instructional purpose of this figure is to help learners identify the major internal structures of a ventricle and understand how **papillary muscles** and **chordae tendineae** work together to support valve function during ventricular contraction, while **trabeculae carneae** contribute to the structural organization of the ventricular chamber.

Figure 6.1.11: Superior View of the Four Heart Valves

This figure shows the **four heart valves** as viewed from above, illustrating their relative positions and structural differences within the heart.

1. Orientation labels indicate the **anterior** and **posterior** aspects of the heart, as well as the anatomical **right** and **left** sides.
2. The two larger openings are the **atrioventricular valves**.
3. The **tricuspid valve** is located on the right side of the heart and has **three cusps**.
4. The **bicuspid, or mitral, valve** is located on the left side of the heart and has **two cusps**.
5. These atrioventricular valves regulate blood flow between the **atria** and **ventricles**.
6. Between the atrioventricular valves are the smaller **semilunar valves**.
7. The **aortic valve** is positioned centrally and posteriorly.
8. The **pulmonary valve** is positioned more anteriorly.
9. These semilunar valves regulate blood flow from the ventricles into the **great arteries**.
10. The figure demonstrates that the atrioventricular valves occupy larger openings between the heart chambers, whereas the semilunar valves are smaller structures located at the **ventricular outflow tracts**.
11. The arrangement of the valves reflects their role in directing blood through the heart in a **single direction**.

The instructional purpose of this figure is to compare the locations and anatomy of the four heart valves and distinguish the **atrioventricular valves** from the **semilunar valves** within the overall structure of the heart.

Figure 6.1.12: Heart Valves During Ventricular Filling (Diastole)

This figure illustrates the positions of the heart valves during ventricular filling (diastole), when blood flows from the atria into the ventricles.

In panel (a), a superior view of the heart shows that the tricuspid and mitral (bicuspid) valves are open, creating pathways for blood to flow from the atria into the ventricles. At the same time, the aortic and pulmonary semilunar valves are closed, preventing blood from flowing back into the ventricles from the great arteries. The arrangement demonstrates how valve actions direct blood forward through the heart during ventricular filling.

In panel (b), an enlarged view of the left ventricle shows the mitral valve in the open position and the aortic valve closed. The papillary muscles are relaxed and the chordae tendineae are not under tension, allowing the valve leaflets to remain open while blood enters the ventricle from the left atrium.

The figure demonstrates how coordinated opening and closing of the atrioventricular and semilunar valves maintains one-way blood flow during diastole. Open atrioventricular valves allow ventricular filling, while closed semilunar valves prevent backflow from the aorta and pulmonary trunk into the ventricles.

Instructional purpose is to show the functional relationship between heart valve position and blood flow during ventricular filling and to illustrate the role of the mitral valve, chordae tendineae, and papillary muscles when the ventricles are relaxed.

Figure 6.1.13: Coronary Arteries and Cardiac Veins: Anterior and Posterior Views

This figure illustrates the positions of the heart valves during ventricular contraction (systole), when blood is ejected from the ventricles into the pulmonary trunk and aorta. The figure includes a superior view of the valves and an enlarged view of the left ventricle showing the relationship among the mitral valve, chordae tendineae, and papillary muscles during contraction.

In panel (a), a superior view of the heart shows that the tricuspid and mitral (bicuspid) valves are closed, preventing blood from flowing backward into the atria. At the same time, the aortic and pulmonary semilunar valves are open, allowing blood to leave the ventricles and enter the systemic and pulmonary circulations. The arrangement demonstrates how valve closure and opening maintain one-way blood flow during ventricular contraction.

In panel (b), an enlarged view of the left ventricle shows the mitral valve closed and the aortic valve open. The papillary muscles are contracted, placing tension on the chordae tendineae. This tension helps keep the mitral valve leaflets closed and prevents them from prolapsing into the left atrium as ventricular pressure rises during systole.

The figure demonstrates how coordinated valve function, contraction of papillary muscles, and tension in the chordae tendineae work together to support efficient blood ejection while preventing backflow into the atria.

Instructional purpose is to show the functional relationship between heart valve position and blood flow during ventricular contraction and to illustrate the role of the mitral valve, chordae tendineae, and papillary muscles in maintaining one-way blood flow during systole.

Figure 6.1.14: Coronary Circulation of the Heart: Anterior and Posterior Views

This figure shows the major coronary arteries and cardiac veins on the surface of the heart from anterior (front) and posterior (back) views. The coronary vessels form a network that supplies oxygen and nutrients to the myocardium and removes deoxygenated blood from cardiac tissues.

In the anterior view, the right and left coronary arteries originate near the base of the aorta and branch across the surface of the heart. Major branches extend over the ventricles and atria, illustrating how arterial blood is distributed throughout the myocardium. Cardiac veins accompany many of these arteries and collect deoxygenated blood from the heart muscle.

In the posterior view, additional coronary vessels are visible on the back of the heart. The coronary sinus, a large venous channel on the posterior surface, receives blood from several cardiac veins. The coronary sinus then returns this blood to the right atrium. The posterior view demonstrates how coronary vessels extend across the entire surface of the heart to provide circulation to regions not visible from the front.

Together, the two views show that coronary arteries deliver oxygenated blood to the myocardium, while cardiac veins return deoxygenated blood to the coronary sinus and ultimately to the right atrium. Comparing the anterior and posterior surfaces emphasizes the extensive distribution of coronary vessels required to support the metabolic demands of cardiac muscle.

Instructional purpose is to illustrate the organization of coronary circulation and show how coronary arteries and cardiac veins work together to supply and drain the myocardium throughout the entire heart.

Figure 6.1.15: Coronary Angiogram Showing Blockages in the Left Coronary Artery and Circumflex Artery

This image is a **coronary angiogram**, a medical imaging study that uses **contrast dye** and **X-ray imaging** to visualize blood flow through the **coronary arteries**. The image shows the **left coronary artery** and its branches on the surface of the heart.

1. Dark, contrast-filled vessels extend from the main trunk of the **left coronary artery** into branching coronary arteries.
2. Two sites of arterial narrowing are highlighted with red arrows and text labels.
3. One arrow identifies a **blockage in the common trunk of the left coronary artery**, near the origin of the vessel before it divides into its major branches.
4. A second arrow identifies a **blockage in the circumflex artery**, one of the primary branches of the left coronary artery that supplies blood to portions of the **left atrium** and **left ventricle**.
5. The contrast dye outlines the vessel lumen, allowing areas of narrowing to be visualized as regions where the diameter of the artery is reduced.
6. The labeled blockages indicate locations where blood flow through the coronary circulation may be restricted due to narrowing of the arterial lumen.

The instructional purpose of this figure is to demonstrate how **coronary angiography** can be used to identify obstructed coronary arteries and assess **coronary artery disease**. The image highlights examples of clinically significant narrowing in the **left coronary circulation**, illustrating how arterial blockages can reduce blood flow to the **myocardium** and increase the risk of **myocardial ischemia** or **infarction**.

Figure 6.2.1: Intercalated Discs and Cellular Connections in Cardiac Muscle

This figure illustrates the structural features that enable **cardiac muscle cells** to function as a coordinated, electrically connected tissue. The figure includes a schematic drawing of cardiac muscle fibers, a micrograph of cardiac muscle tissue, and an enlarged view of the cellular structures found within **intercalated discs**.

Panel (a): Schematic of Cardiac Muscle Fibers

1. Branching **cardiac muscle fibers** are shown connected end-to-end by **intercalated discs**.
2. Each cell contains a centrally located **nucleus** and numerous **mitochondria**.
3. The intercalated discs form specialized junctions between adjacent cardiac muscle cells, creating both **mechanical** and **electrical connections**.
4. **Gap junctions** within the intercalated discs provide pathways for electrical signals to pass directly from one cell to another.

Panel (b): Micrograph of Cardiac Muscle Tissue

1. A micrograph of cardiac muscle tissue shows **intercalated discs** as dark bands crossing adjacent muscle fibers.
2. The image demonstrates that these junctions are visible structural features of cardiac muscle and occur at the boundaries between neighboring cells.

Panel (c): Enlarged View of an Intercalated Disc

1. An enlarged view of an **intercalated disc** highlights its major components.
2. **Desmosomes** mechanically anchor adjacent cells together, helping the tissue withstand the forces generated during repeated contractions.
3. **Gap junctions** create channels between neighboring cells that allow ions and electrical impulses to pass rapidly from cell to cell.
4. The arrangement of cardiac muscle contractile elements, including the **A bands** and **I bands**, is also shown within the connected cells.

Together, the panels demonstrate that **cardiac muscle cells** are physically linked and electrically coupled through **intercalated**

discs. These specialized connections allow the **myocardium** to contract as a coordinated unit while maintaining the structural integrity needed to withstand continuous cardiac activity.

Instructional purpose is to illustrate the structure and function of **intercalated discs** and explain how **desmosomes** and **gap junctions** enable cardiac muscle cells to remain connected mechanically and communicate electrically, supporting coordinated contraction of the heart.

Figure 6.2.2: Cardiac Conduction System and the Pathway of Electrical Impulses Through the Heart

This figure illustrates the cardiac conduction system and the sequence by which electrical impulses spread through the heart to coordinate atrial and ventricular contraction. The image shows a frontal section of the heart with specialized conduction tissues highlighted and arrows indicating the direction of electrical signal transmission.

The electrical impulse originates in the sinoatrial (SA) node, located in the wall of the right atrium. From the SA node, impulses spread through the atrial myocardium and travel along internodal pathways. A branch known as Bachmann's bundle conducts the impulse to the left atrium, allowing both atria to depolarize and contract in a coordinated manner.

The impulse then reaches the atrioventricular (AV) node at the junction between the atria and ventricles. After a brief delay at the AV node, the signal enters the atrioventricular (AV) bundle, also called the bundle of His, which provides the normal electrical connection between the atria and ventricles.

The AV bundle divides into right and left bundle branches that descend through the interventricular septum toward the apex of the heart. These branches give rise to Purkinje fibers, which distribute the electrical impulse throughout the ventricular myocardium. This conduction pattern causes ventricular depolarization to spread from the apex upward through the ventricular walls, producing coordinated ventricular contraction and efficient ejection of blood.

The figure emphasizes that the conduction system generates and distributes electrical signals in an organized sequence, ensuring that the atria contract before the ventricles and that the ventricles contract as a coordinated pumping unit.

Instructional purpose is to illustrate the components of the cardiac conduction system and demonstrate how electrical impulses travel through the heart to coordinate the timing and sequence of atrial and ventricular contractions.

Figure 6.2.3: Sequence of Electrical Conduction and Depolarization Through the Heart

This figure illustrates the progression of electrical activity through the heart during a cardiac cycle. Six sequential panels show how an electrical impulse originates in the cardiac conduction system and spreads through the atria and ventricles to coordinate contraction.

Panel 1: The heart is at rest before depolarization begins. The sinoatrial (SA) node and other components of the conduction system are visible, but no chamber is depolarized.

Panel 2: The electrical impulse originates in the sinoatrial (SA) node and begins spreading across the atria through conduction pathways. Arrows indicate the direction of impulse propagation.

Panel 3: Depolarization spreads throughout both atria, shown in purple. This stage corresponds to atrial activation and precedes atrial contraction.

Panel 4: The impulse reaches the atrioventricular (AV) node and then travels rapidly through the atrioventricular bundle, bundle branches, and Purkinje fibers toward the apex of the heart. The conduction pathways within the ventricles are highlighted.

Panel 5: Ventricular depolarization begins near the apex and spreads upward through the ventricular myocardium. Most of the ventricular tissue is shown as depolarized.

Panel 6: Depolarization has spread throughout both ventricles, indicating complete ventricular activation. Following this coordinated electrical activation, ventricular contraction can occur to eject blood into the pulmonary trunk and aorta.

The sequence emphasizes that electrical activity begins in the SA node, spreads through the atria, pauses briefly at the AV node, and then travels through specialized conduction pathways to activate the ventricles from the apex upward. This ordered pattern ensures that atrial contraction occurs before ventricular contraction and that the ventricles contract as an efficient pumping unit.

Instructional purpose is to demonstrate the normal sequence of cardiac electrical conduction and show how depolarization spreads through the atria and ventricles to coordinate the timing of the heartbeat.

Figure 6.2.4: Pacemaker Action Potential in Sinoatrial (SA) Nodal Cells

This graph illustrates the spontaneous action potential generated by sinoatrial (SA) nodal cells, the heart's primary pacemaker cells. Membrane potential is plotted against time, showing the repeating cycle of depolarization, repolarization, and spontaneous depolarization that enables the SA node to initiate the heartbeat without external stimulation.

The cycle begins at a membrane potential of approximately −60 mV. Unlike contractile cardiac muscle cells, SA nodal cells do not maintain a stable resting membrane potential. Instead, a gradual depolarization, labeled the **prepotential**, occurs as sodium ions slowly enter the cell, causing the membrane potential to become less negative until it reaches the threshold of approximately −40 mV.

Once threshold is reached, rapid depolarization occurs as calcium ions enter the cell through calcium channels. The membrane potential rises to approximately +15 to +20 mV, generating an action potential.

Following depolarization, potassium channels open and potassium ions leave the cell, producing repolarization. The membrane potential returns to approximately −60 mV, completing the cycle.

As repolarization ends, the membrane potential again begins to slowly depolarize toward threshold, initiating another action potential. The repeating pattern shown in the graph demonstrates the automatic rhythmic activity of pacemaker cells.

The figure highlights three key phases of the SA nodal action potential: the prepotential caused primarily by slow sodium influx, depolarization caused by calcium influx, and repolarization caused by potassium efflux. Unlike ventricular contractile cells, SA nodal cells lack a prolonged plateau phase and continuously cycle between action potentials.

Instructional purpose is to illustrate how pacemaker cells in the sinoatrial node generate spontaneous action potentials and to demonstrate the ion movements responsible for the automatic initiation and rhythmic control of the heartbeat.

Figure 6.2.5: Cardiac Muscle Action Potential, Refractory Periods, and Comparison with Skeletal Muscle

This figure compares the electrical activity and contraction characteristics of cardiac muscle and skeletal muscle. Panel (a) illustrates the phases of a cardiac muscle action potential and associated refractory periods, while panel (b) compares the timing of action potentials, refractory periods, and contractions in skeletal and cardiac muscle.

In panel (a), membrane potential is plotted against time for a cardiac contractile cell. The action potential begins with rapid depolarization caused by the opening of voltage-gated sodium channels and influx of sodium ions. After reaching a positive membrane potential, sodium channels close and slow calcium channels open, producing a prolonged plateau phase. During the plateau, calcium influx helps maintain depolarization and supports muscle contraction. Repolarization begins when calcium channels close and potassium channels open, allowing potassium to leave the cell and return the membrane potential toward its resting level. Once repolarization is complete, potassium channels close and the cell returns to its resting membrane potential. The graph also shows a prolonged refractory period consisting of an absolute refractory period followed by a shorter relative refractory period, both of which occupy most of the action potential and contraction cycle.

In panel (b), skeletal muscle and cardiac muscle are compared. In skeletal muscle, the action potential and refractory period are very short relative to the duration of contraction. As a result, additional stimuli can occur before the muscle has fully relaxed, allowing summation and tetanic contractions. In cardiac muscle, the action potential includes an extended plateau phase that greatly lengthens the refractory period. The refractory period overlaps almost entirely with the contraction, preventing another action potential from triggering a second contraction before relaxation is complete.

The figure demonstrates that the prolonged plateau phase and refractory period of cardiac muscle prevent sustained tetanic

contractions and ensure that the heart undergoes alternating cycles of contraction and relaxation necessary for effective pumping of blood.

Instructional purpose is to illustrate the phases of the cardiac muscle action potential, explain the relationship between ion channel activity and membrane potential changes, and compare cardiac and skeletal muscle refractory periods to show why cardiac muscle cannot undergo tetanus.

Figure 6.2.6: Surface Locations for Cardiac Auscultation

This figure shows the standard locations on the body surface where healthcare providers listen to heart sounds using a stethoscope. The listening points, or auscultation sites, are positioned on the chest wall where sounds produced by blood flow through specific heart valves are transmitted most clearly. Additional pulse points are shown at the wrists and ankles for comparison.

Several markers are located on the anterior chest near the sternum and between the ribs. These sites correspond to the aortic, pulmonic, tricuspid, and mitral valve auscultation areas. Although the markers are associated with specific valves, they are not positioned directly over the valves themselves. Instead, they represent locations where valve sounds are best heard after sound waves travel through blood and surrounding tissues.

The aortic area is located at the right second intercostal space adjacent to the sternum. The pulmonic area is located at the left second intercostal space adjacent to the sternum. The tricuspid area is located along the lower left sternal border. The mitral area, also called the apical area, is located at the left fifth intercostal space near the midclavicular line. Additional markers illustrate intermediate auscultation points commonly used during a comprehensive cardiac examination.

Markers at the wrists and ankles identify peripheral pulse assessment sites that may be examined alongside heart auscultation to evaluate cardiovascular function and blood flow.

Instructional purpose is to identify the primary surface landmarks used for cardiac auscultation and demonstrate where heart valve sounds are most effectively heard during a physical examination.

Figure 6.2.7: Electrocardiogram (ECG) Waveform and Measurement Intervals

This figure illustrates the major components of a normal electrocardiogram (ECG) tracing and the standard intervals and segments used to evaluate cardiac electrical activity. An inset at the top shows an ECG tracing recorded on grid paper, with markings indicating the standard scale used to measure time and voltage.

The enlarged tracing identifies the principal ECG waves and intervals within a cardiac cycle. The **P wave** represents atrial depolarization as the electrical impulse spreads through the atria. The **QRS complex** represents ventricular depolarization and consists of three deflections: a small downward Q wave, a large upward R wave, and a downward S wave. The **T wave** represents ventricular repolarization as the ventricles return to their resting electrical state.

Several clinically important measurements are highlighted. The **PR interval** extends from the beginning of the P wave to the beginning of the QRS complex and reflects conduction of the electrical impulse from the atria through the atrioventricular conduction system to the ventricles. The **QT interval** extends from the beginning of the QRS complex to the end of the T wave and represents the total time required for ventricular depolarization and repolarization. The **PR segment** lies between the end of the P wave and the beginning of the QRS complex, while the **ST segment** lies between the end of the QRS complex and the beginning of the T wave.

The inset demonstrates how ECG grid paper is used to measure waveform duration. A 5 mm division and a one-second interval are shown, illustrating how time measurements are determined from the recording.

The figure emphasizes that an ECG records the electrical events associated with the cardiac cycle. By analyzing the waves, segments, and intervals, clinicians can evaluate the timing and conduction of electrical impulses through the heart.

Instructional purpose is to identify the major components of a normal ECG tracing and demonstrate how ECG waves, segments, and intervals correspond to electrical events occurring in the atria and ventricles during the cardiac cycle.

Figure 6.2.8: Relationship Between Cardiac Electrical Activity and the Electrocardiogram (ECG)

This figure illustrates how the spread of electrical activity through the heart corresponds to the waves recorded on an electrocardiogram (ECG). Six sequential panels show depolarization of the atria and ventricles, while adjacent ECG tracings highlight the portion of the waveform produced by each electrical event.

Panel 1: The heart is electrically at rest following ventricular repolarization. The ECG tracing is at the baseline between the end of the T wave and the beginning of the next P wave.

Panel 2: The sinoatrial (SA) node initiates an electrical impulse that begins spreading through the atria. This early atrial depolarization corresponds to the beginning of the **P wave** on the ECG.

Panel 3: Depolarization spreads throughout both atria. The atria are fully activated, and the **P wave** is completed. The impulse is approaching the atrioventricular (AV) node before entering the ventricles.

Panel 4: The electrical impulse travels rapidly through the AV bundle, bundle branches, and Purkinje fibers, initiating ventricular depolarization. This event corresponds to the **QRS complex** on the ECG. Because atrial repolarization occurs simultaneously, it is obscured by the much larger QRS complex and is not visible as a separate wave.

Panel 5: Ventricular depolarization continues through the ventricular myocardium. The ventricles are activated and begin contracting. This stage corresponds to the **ST segment**, when the ventricles remain depolarized.

Panel 6: The ventricles begin repolarizing and returning to their resting electrical state. This process corresponds to the **T wave** on the ECG. As repolarization is completed, the ECG returns to the baseline and the cycle repeats.

The figure demonstrates that the ECG records the electrical events that precede cardiac contraction. The P wave reflects atrial depolarization, the QRS complex reflects ventricular depolarization, and the T wave reflects ventricular repolarization.

Instructional purpose is to illustrate the relationship between the spread of electrical activity through the heart and the corresponding waves and segments of a normal electrocardiogram.

Figure 6.2.9: Examples of Abnormal Electrocardiogram (ECG) Rhythms and Conduction Disorders

This figure presents five ECG tracings that illustrate common abnormalities of cardiac conduction and rhythm. Each panel demonstrates a different pattern of electrical activity and its effect on the coordination of cardiac contraction.

(a) Second-degree atrioventricular (AV) block: Some P waves are not followed by QRS complexes, indicating that electrical impulses generated in the atria are intermittently blocked before reaching the ventricles. As a result, some atrial contractions are not followed by ventricular contractions, producing a slower ventricular rate than the atrial rate.

(b) Atrial fibrillation: Normal, discrete P waves are absent and replaced by rapid, irregular electrical activity in the atria. The QRS complexes occur at irregular intervals, reflecting irregular conduction of impulses to the ventricles. This pattern indicates uncoordinated atrial depolarization and loss of effective atrial contraction.

(c) Ventricular tachycardia: A rapid series of broad, abnormal QRS complexes is present with little or no normal atrial activity visible. The ventricles are being activated by an abnormal electrical focus, resulting in a fast ventricular rhythm that can reduce the heart's pumping efficiency.

(d) Ventricular fibrillation: The tracing shows chaotic, disorganized electrical activity without identifiable P waves, QRS complexes, or T waves. Electrical impulses are occurring in an uncoordinated manner throughout the ventricles, preventing effective ventricular contraction and blood ejection.

(e) Third-degree atrioventricular (complete) block: Atrial and ventricular electrical activities occur independently of one another. P waves and QRS complexes are present but are not consistently associated. Because impulses from the atria do not reach the ventricles, the ventricles rely on a slower escape rhythm to maintain contraction.

Together, these tracings demonstrate different types of cardiac arrhythmias and conduction abnormalities, ranging from delayed or blocked impulse conduction to severe disruptions of coordinated electrical activity.

Instructional purpose is to compare normal impulse conduction with common conduction blocks and arrhythmias and to demonstrate how abnormalities in cardiac electrical activity alter ECG waveforms and affect the heart's ability to maintain coordinated contractions and effective blood flow.

Figure 6.2.10: Automated External Defibrillator (AED) and Defibrillation Paddles

This figure shows equipment used to deliver an electrical shock to the heart during certain life-threatening cardiac arrhythmias.

In panel (a), an automated external defibrillator (AED) is stored inside a clearly marked wall-mounted protective cabinet. The device is designed for rapid access during cardiac emergencies and can be used to analyze heart rhythm and, when appropriate, deliver a defibrillation shock. The cabinet includes a heart-and-lightning-bolt symbol commonly associated with defibrillators.

In panel (b), a handheld defibrillation paddle is shown. Defibrillation paddles or adhesive electrode pads are placed on specific locations of the patient's chest to deliver a controlled electrical shock through the heart. The shock can depolarize a large portion of the myocardium simultaneously, interrupting abnormal electrical activity and allowing the heart's normal conduction system to reestablish an organized rhythm.

Together, the images illustrate both the emergency device used to provide defibrillation and a component used to transmit electrical energy to the patient's heart during treatment.

Instructional purpose is to identify equipment used in cardiac resuscitation and demonstrate how defibrillation devices are used to treat certain abnormal heart rhythms by restoring coordinated electrical activity in the heart.

Figure 6.3.1: Mechanical Events of the Cardiac Cycle and Their Relationship to the ECG

This figure illustrates the major phases of the cardiac cycle and the associated changes in chamber contraction, blood flow, and electrical activity. The cycle is presented as a continuous sequence progressing clockwise through atrial systole, ventricular systole, and ventricular diastole. Representative ECG tracings are positioned around the diagram to show the electrical events associated with each phase.

The cycle begins with **late ventricular diastole**, when the ventricles are relaxed and filling with blood from the atria through open atrioventricular valves. Blood flows passively into the ventricles.

Next, **atrial contraction (atrial systole)** occurs. The atria contract and force additional blood into the ventricles, completing ventricular filling. This phase follows atrial depolarization represented by the **P wave** on the ECG.

As ventricular depolarization begins, represented by the **QRS complex**, the ventricles enter **isovolumetric (isovolumic) contraction**. Ventricular pressure rises, causing the atrioventricular valves to close. Because all valves are closed during this phase, ventricular volume remains constant while pressure increases.

When ventricular pressure exceeds the pressure in the pulmonary trunk and aorta, the semilunar valves open and **ventricular ejection** begins. Blood is expelled from the right ventricle into the pulmonary circulation and from the left ventricle into the systemic circulation. This phase represents the second part of ventricular systole.

Following ventricular ejection, the ventricles begin to repolarize, corresponding to the **T wave** on the ECG. The ventricles then enter **isovolumetric (isovolumic) relaxation**. Ventricular pressure falls, causing the semilunar valves to close. Because all valves are again closed, ventricular volume remains unchanged during the early relaxation phase.

As ventricular pressure drops below atrial pressure, the atrioventricular valves open and **ventricular filling** resumes. Blood once again flows from the atria into the ventricles, returning the heart to late ventricular diastole and completing the cycle.

The figure emphasizes the coordinated relationship between electrical activity recorded on the ECG and the mechanical events

of the cardiac cycle. Electrical depolarization precedes contraction, and repolarization precedes relaxation.

Instructional purpose is to illustrate the sequence of events in the cardiac cycle and demonstrate how atrial contraction, ventricular contraction, ventricular ejection, ventricular relaxation, and ventricular filling correspond to changes in cardiac electrical activity and blood flow through the heart.

Figure 6.3.2: Relationship Between the ECG and Mechanical Events of One Cardiac Cycle

This figure correlates the **electrical activity** recorded on an **electrocardiogram, or ECG,** with the mechanical phases of **atrial** and **ventricular contraction** and **relaxation** during one cardiac cycle.

ECG and Mechanical Timing

1. The ECG tracing displays a **P wave**, **QRS complex**, and **T wave**.
2. Beneath the tracing, color-coded bars indicate the timing of **atrial systole**, **atrial diastole**, **ventricular systole**, and **ventricular diastole** relative to these electrical events.
3. The cycle begins with both the **atria** and **ventricles** in **diastole**.
4. The **P wave** corresponds to **atrial depolarization** and is followed by **atrial systole**, during which the atria contract and complete ventricular filling.
5. While the atria contract, the ventricles remain in **diastole**.
6. The **QRS complex** represents **ventricular depolarization** and marks the beginning of **ventricular systole**.
7. As the ventricles contract and develop pressure, the atria enter **atrial diastole** and remain relaxed for the remainder of the cycle.
8. **Ventricular systole** encompasses both **isovolumetric contraction** and **ventricular ejection**.
9. The **T wave** represents **ventricular repolarization** and is associated with the onset of **ventricular diastole**.
10. Following ventricular relaxation, the ventricles fill with blood as the next cardiac cycle begins.

The figure emphasizes that **electrical events occur immediately before the corresponding mechanical events**. **Atrial depolarization** precedes **atrial contraction**, **ventricular depolarization** precedes **ventricular contraction**, and **ventricular repolarization** precedes **ventricular relaxation**.

Instructional purpose is to demonstrate the timing relationship between **ECG waveforms** and the mechanical phases of **atrial** and **ventricular systole** and **diastole** during a single cardiac cycle.

Figure 6.3.3: Pressure Changes, Valve Events, and Heart Sounds During the Cardiac Cycle

This figure illustrates changes in **aortic**, **ventricular**, and **atrial pressures** during one cardiac cycle and shows how these pressure changes correspond to **valve movements** and **heart sounds**.

Pressure Curves

1. Three pressure curves are plotted over time.
2. The **red curve** represents **aortic pressure**.
3. The **green curve** represents **ventricular pressure**.
4. The **orange curve** represents **atrial pressure**.
5. A tracing below the graph shows the timing of the **first**, **second**, and **third heart sounds**.

Sequence of Events During the Cardiac Cycle

1. At the beginning of the cycle, atrial and ventricular pressures are low, and the **atrioventricular, or AV, valves** are **open**, allowing ventricular filling.
2. As the ventricles begin to contract, **ventricular pressure** rises rapidly.

3. When ventricular pressure exceeds atrial pressure, the **AV valves close**, producing the **first heart sound, S1, or "lub."**
4. During **ventricular systole**, ventricular pressure continues to increase.
5. When ventricular pressure exceeds **aortic pressure**, the **semilunar valves open**, allowing blood to be ejected into the **aorta**.
6. Ventricular and aortic pressures rise together and reach similar peak values during ejection.
7. As ventricular relaxation begins, ventricular pressure falls below aortic pressure, causing the **semilunar valves to close**.
8. Closure of the semilunar valves produces the **second heart sound, S2, or "dub."**
9. A brief increase in aortic pressure following valve closure, known as the **dicrotic notch**, is visible on the aortic pressure curve.
10. During **ventricular diastole**, ventricular pressure continues to decrease.
11. When ventricular pressure falls below atrial pressure, the **AV valves open**, allowing blood to flow from the atria into the ventricles and initiating ventricular filling.
12. A small **third heart sound, S3,** is shown during early ventricular filling.

The figure demonstrates that **heart sounds** are produced primarily by **valve closure** and that the opening and closing of valves are determined by **pressure differences** between the heart chambers and great vessels. It also shows how **atrial**, **ventricular**, and **aortic pressures** change throughout **systole** and **diastole** to maintain blood flow through the heart.

Instructional purpose is to illustrate the relationship among **chamber pressures**, **valve movements**, and **heart sounds** during the cardiac cycle and to demonstrate how **pressure gradients** control blood flow through the heart.

Figure 6.3.4: Cardiac Auscultation Sites for the Four Heart Valves

This figure shows the standard chest locations where heart sounds associated with the **four heart valves** are best heard during **auscultation**. The image includes an anterior view of the **thorax** with the **heart** and **rib cage** shown in the background and color-coded listening areas marked on the chest surface.

Key Features

1. The auscultation sites do not correspond to the anatomical locations of the valves within the heart.
2. Instead, they represent the areas where sounds generated by blood flow through each valve are transmitted most clearly through surrounding tissues.
3. The **aortic valve** auscultation site is located at the **right second intercostal space** adjacent to the **sternum**.
4. The **pulmonary valve** auscultation site is located at the **left second intercostal space** adjacent to the sternum.
5. The **tricuspid valve** auscultation site is located along the **lower left sternal border**, typically near the **fourth or fifth intercostal space**.
6. The **mitral, or bicuspid, valve** auscultation site is located at the **cardiac apex** in the **left fifth intercostal space** near the **midclavicular line**.

The figure demonstrates that clinicians listen at specific **surface landmarks** on the chest to evaluate **valve function**, **heart sounds**, and **cardiac murmurs**, even though the valves themselves are located deeper within the heart.

Instructional purpose is to identify the standard auscultation sites for the **aortic**, **pulmonary**, **tricuspid**, and **mitral** valves and to demonstrate where sounds from each valve are best heard during a cardiac examination.

Figure 6.4.1: Factors Determining Cardiac Output

This flowchart illustrates the relationship between **heart rate**, **stroke volume**, and **cardiac output**. The diagram shows that cardiac output depends on both the number of heartbeats per minute and the amount of blood ejected by the heart during each contraction.

Heart Rate Factors

1. The left side of the figure identifies factors that influence **heart rate, or HR**.
2. These factors include:
   - **Autonomic nervous system activity**
   - **Circulating hormones**

- **Fitness level**
- **Age**

3. These variables affect how frequently the heart contracts.

Stroke Volume Factors

1. The right side of the figure identifies factors that influence **stroke volume, or SV**, which is the amount of blood ejected by a ventricle during a single heartbeat.
2. Factors affecting stroke volume include:
   - **Heart size**
   - **Fitness level**
   - **Biological sex**
   - **Myocardial contractility**
   - **Duration of contraction**
   - **Preload**, or **end-diastolic volume, EDV**
   - **Afterload**, or **vascular resistance**
3. The figure also indicates that stroke volume is calculated as **end-diastolic volume minus end-systolic volume**.
4. In abbreviated form, **SV = EDV − ESV**.

Cardiac Output Relationship

1. Arrows from the heart rate and stroke volume sections converge on a final equation showing that **cardiac output, or CO, equals heart rate multiplied by stroke volume**.
2. In abbreviated form, **CO = HR × SV**.
3. The diagram emphasizes that changes in either heart rate or stroke volume will alter the total volume of blood pumped by the heart each minute.

Instructional purpose: To demonstrate the factors that influence **heart rate** and **stroke volume** and explain how these variables interact to determine **cardiac output**, the volume of blood pumped by the heart per minute.

Figure 6.4.2: Autonomic Nervous System Regulation of Heart Rate and Cardiac Contractility

This figure illustrates how the **autonomic nervous system** regulates heart function through **parasympathetic** and **sympathetic innervation** of the heart. The diagram shows pathways extending from the **brainstem** and **spinal cord** to the heart and identifies their effects on **heart rate** and **cardiac contraction**.

Parasympathetic Pathway

1. The **parasympathetic pathway**, represented by the **vagus nerve**, originates in the **brainstem** and extends directly to the heart.
2. Parasympathetic fibers primarily innervate the **sinoatrial, or SA, node** and **atrioventricular, or AV, node**.
3. Activation of the vagus nerve decreases **heart rate** by slowing the generation and conduction of electrical impulses within the **cardiac conduction system**.

Sympathetic Pathway

1. The **sympathetic pathway** originates from the **thoracic region of the spinal cord** and travels through **sympathetic cardiac nerves** to the heart.
2. Sympathetic fibers innervate the **cardiac conduction system** and the **myocardium**.
3. Activation of sympathetic cardiac nerves increases **heart rate** and increases the **force of ventricular contraction**, allowing the heart to pump more blood during periods of increased physiological demand.

The figure contrasts the opposing influences of the two autonomic divisions. **Parasympathetic stimulation** slows cardiac activity, whereas **sympathetic stimulation** accelerates cardiac activity and enhances pumping strength. Together, these

pathways provide continuous neural regulation of heart function to help maintain **cardiovascular homeostasis**.

Instructional purpose is to demonstrate how **parasympathetic** and **sympathetic autonomic pathways** innervate the heart and regulate **heart rate** and the **force of cardiac contraction** through opposing physiological effects.

Figure 6.4.3: Effects of Autonomic Stimulation on Sinoatrial (SA) Node Pacemaker Activity

This figure compares **pacemaker action potentials** in **sinoatrial, or SA, nodal cells** under **resting conditions**, **parasympathetic stimulation**, and **sympathetic stimulation**. **Membrane potential** is plotted against **time**, with a dashed line indicating the **threshold** for action potential generation.

Top Panel: Normal Resting Activity

1. The **top panel**, labeled **Normal resting**, shows the regular spontaneous activity of **SA nodal cells**.
2. Following each **repolarization**, the membrane potential gradually depolarizes during the **prepotential** phase until it reaches threshold.
3. Reaching threshold triggers the next action potential.
4. This repeating cycle establishes the normal **intrinsic heart rate**.

Middle Panel: Parasympathetic Stimulation

1. The **middle panel**, labeled **Parasympathetic stimulation**, shows the effects of **vagal stimulation** on SA nodal cells.
2. The membrane potential becomes more negative following repolarization, a change described as **hyperpolarization**.
3. The rate of spontaneous depolarization is reduced.
4. As a result, threshold is reached less frequently and action potentials occur farther apart in time.
5. This change decreases the firing rate of the **SA node** and lowers **heart rate**.

Bottom Panel: Sympathetic Stimulation

1. The **bottom panel**, labeled **Sympathetic stimulation**, shows the effects of sympathetic stimulation on SA nodal cells.
2. The slope of spontaneous depolarization becomes **steeper**.
3. Repolarization is reduced so that the membrane potential remains closer to threshold.
4. As a result, threshold is reached more quickly and action potentials occur more frequently.
5. This change increases the firing rate of the **SA node** and raises **heart rate**.

The figure demonstrates that **autonomic regulation** alters heart rate by changing the rate at which **pacemaker cells** reach threshold. **Parasympathetic stimulation** slows spontaneous depolarization and increases the time between action potentials, whereas **sympathetic stimulation** accelerates spontaneous depolarization and shortens the interval between action potentials.

Instructional purpose is to illustrate how **parasympathetic** and **sympathetic stimulation** modify **SA nodal pacemaker activity** and to demonstrate the cellular mechanisms by which the **autonomic nervous system** decreases or increases heart rate.

Figure 6.4.4: Factors That Increase or Decrease Stroke Volume

This table summarizes the major factors that influence **stroke volume, or SV,** and groups them into three categories: **preload**, **contractility**, and **afterload**. For each category, the figure identifies conditions that increase or decrease the factor and shows the resulting effect on stroke volume.

Preload

1. **Preload** refers to the degree of **ventricular filling** before contraction and is closely related to **end-diastolic volume, or EDV**.
2. Increased **filling time** and increased **venous return** raise preload.

3. Higher preload results in a larger end-diastolic volume and an **increase in stroke volume**.
4. The figure also identifies conditions associated with reduced preload, including:
    - Reduced **thyroid hormone** levels
    - Abnormal **calcium**, **potassium**, or **sodium** concentrations
    - Low **body temperature**
    - **Hypoxia**
    - Abnormal **blood pH**
    - Certain **medications**
5. Reduced preload decreases end-diastolic volume and lowers stroke volume.

Contractility

1. **Contractility** refers to the strength of **ventricular contraction** independent of ventricular filling.
2. Factors that increase contractility include:
    - **Sympathetic stimulation**
    - **Epinephrine**
    - **Norepinephrine**
    - Increased **intracellular calcium**
    - Elevated **blood calcium** levels
    - **Thyroid hormones**
    - **Glucagon**
3. Greater contractility reduces **end-systolic volume, or ESV**, causing more blood to be ejected and increasing stroke volume.
4. Factors that decrease contractility include:
    - **Parasympathetic stimulation**
    - **Acetylcholine**
    - **Hypoxia**
    - **Hyperkalemia**
5. Reduced contractility increases end-systolic volume and decreases stroke volume.

Afterload

1. **Afterload** refers to the resistance the ventricles must overcome to eject blood.
2. Increased **vascular resistance** or **semilunar valve damage** raises afterload, making ventricular ejection more difficult.
3. Higher afterload increases end-systolic volume and decreases stroke volume.
4. Reduced vascular resistance lowers afterload, decreases end-systolic volume, and increases stroke volume.

The figure emphasizes that **stroke volume** depends on the balance among **ventricular filling** or preload, the **strength of contraction** or contractility, and **resistance to ejection** or afterload. Changes in any of these factors alter the amount of blood ejected by the ventricle with each heartbeat.

Instructional purpose is to compare the physiological factors that affect **stroke volume** and demonstrate how changes in **preload**, **contractility**, and **afterload** influence **end-diastolic volume**, **end-systolic volume**, and the amount of blood ejected during each cardiac contraction.

Figure 6.4.5: Integrated Regulation of Cardiac Output by Heart Rate and Stroke Volume

This flowchart illustrates the physiological factors that regulate **cardiac output, or CO,** through their effects on **heart rate, or HR,** and **stroke volume, or SV**. Arrows show how multiple cardiovascular mechanisms influence these variables and ultimately determine the volume of blood pumped by the heart each minute.

Heart Rate Regulation

1. The left side of the diagram focuses on factors affecting **heart rate**.
2. The **atrial reflex** responds to increased **venous return** and contributes to adjustments in heart rate.

3. **Autonomic innervation** and **hormones** also influence heart rate by altering the activity of the heart's **pacemaker** and **conduction systems**.
4. These factors converge to determine the overall heart rate.

Stroke Volume Regulation

1. The right side of the diagram focuses on factors affecting **stroke volume**.
2. Stroke volume is determined by the difference between **end-diastolic volume, or EDV,** and **end-systolic volume, or ESV**.
3. In equation form, **SV = EDV − ESV**.
4. Three primary factors influence stroke volume:
   - **Preload**
   - **Contractility**
   - **Afterload**

Effects of Preload, Contractility, and Afterload

1. **Preload** is affected by **venous return** and **filling time**.
2. Preload influences **end-diastolic volume** by determining how much blood fills the ventricles before contraction.
3. **Contractility** is influenced by **autonomic innervation** and **hormones**.
4. Contractility affects the force of ventricular contraction and therefore influences **end-systolic volume**.
5. **Afterload** is influenced by **vasodilation** and **vasoconstriction**.
6. Afterload affects the resistance the ventricles must overcome to eject blood and also influences **end-systolic volume**.
7. Changes in preload alter **EDV**, while changes in contractility and afterload alter **ESV**.

Cardiac Output Relationship

1. The lower portion of the figure shows that **cardiac output** is calculated as **heart rate multiplied by stroke volume**.
2. In equation form, **CO = HR × SV**.
3. The diagram emphasizes that cardiac output can be increased or decreased through changes in **heart rate**, **stroke volume**, or both.

Instructional purpose is to demonstrate how **heart rate** and **stroke volume** are regulated by interconnected physiological factors and to illustrate how these variables combine to determine **cardiac output**.

Figure 6.5.1: Development and Septation of the Embryonic Heart

This figure illustrates the major stages of **embryonic heart development**, beginning with formation of the **primitive heart tube** and ending with separation of the heart into **four chambers**.

Upper Panel: Early Heart Development

1. The upper panel follows heart development from approximately **day 18 to day 35** of embryonic development.
2. Early in development, the **cardiogenic region** forms near the cranial end of the embryo, where paired **endocardial tubes** develop.
3. By approximately **day 21**, these tubes fuse to form a single **primitive heart tube**.
4. The primitive heart tube differentiates into distinct regions that will contribute to the mature heart, including the:
   - **Truncus arteriosus**
   - **Bulbus cordis**
   - **Primitive ventricle**
   - **Primitive atrium**
   - **Sinus venosus**
5. As development continues, the heart tube lengthens and undergoes **looping**.
6. Looping brings future atrial regions **superiorly and posteriorly** while positioning ventricular regions **inferiorly and anteriorly**.
7. By approximately **day 35**, recognizable atrial and ventricular chambers are present, and outflow structures are connected to

the developing **aortic arch arteries**.

Lower Panel: Formation of the Four-Chambered Heart

1. The lower panel illustrates formation of the **four-chambered heart**.
2. At approximately **28 days**, internal **septa** begin dividing the primitive atrium and ventricle.
3. The **septum primum** grows downward within the atrium.
4. The **interventricular septum** develops between the ventricles.
5. **Endocardial cushions** contribute to formation of the **atrioventricular canals** and future **cardiac valves**.
6. By approximately **8 weeks**, the heart has become partitioned into the **right and left atria** and **right and left ventricles**.
7. The **tricuspid valve** and **mitral valve** separate the atria from their corresponding ventricles.
8. The **foramen ovale** remains present between the atria as part of normal **fetal circulation**, allowing blood to pass from the **right atrium** to the **left atrium** before birth.

The figure demonstrates how a simple linear **heart tube** undergoes **looping**, **chamber formation**, and **septation** to produce the **four-chambered heart** capable of supporting fetal and postnatal circulation.

Instructional purpose is to illustrate the sequence of embryonic heart development, including formation and looping of the **primitive heart tube** and partitioning of the heart into four chambers through the development of **septa**, **endocardial cushions**, and **valves**.

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7: BLOOD VESSELS AND CIRCULATORY PATHWAYS

Chapter Overview



Figure Figure 7.1: While most blood vessels are located deep from the surface and are not visible, the superficial veins of the upper limb provide an indication of the extent, prominence, and importance of these structures to the body. (CC BY 4.0; via LibreTexts).

Blood pumped by the heart flows through a series of vessels—arteries, arterioles, capillaries, venules, and veins—before returning to the heart. Arteries transport blood away from the heart and branch into smaller arterioles, which deliver blood to capillary beds, the main sites of exchange with body tissues. Capillaries then drain into venules, which merge into larger veins that return blood to the heart.

Comparing Arteries, Veins, and Capillaries

The arterial system is a relatively high-pressure system, so arteries have thick, muscular walls and usually appear round in cross section. The venous system is lower pressure and contains veins with larger lumens and thinner walls that often appear flattened. Arteries, arterioles, venules, and veins share three layers (tunics): the tunica intima, tunica media, and tunica externa. Capillaries consist only of a tunica intima. The tunica intima is a thin layer of simple squamous epithelium (endothelium) and a small amount of connective tissue. The tunica media is a thicker middle layer made of variable amounts of smooth muscle and connective tissue; it is the thickest layer in all but the largest arteries. The tunica externa is primarily connective tissue and, in veins, also contains some smooth muscle. Changes in smooth muscle tone in vessel walls (vasoconstriction and vasodilation) can dramatically influence vessel diameter and blood flow.

Systemic Circulation: Supplying the Body Tissues

Systemic circulation carries oxygen-rich blood from the left side of the heart to all tissues of the body and returns oxygen-poor blood to the right side of the heart. Blood leaves the left ventricle through the aorta and its branches, travels through systemic arteries, arterioles, and capillary beds where exchange with tissues occurs, and then returns via venules, veins, and the superior and inferior venae cavae to the right atrium. Key pathways such as the coronary circulation (to the heart) and the hepatic portal circulation (from digestive organs to the liver) illustrate how specific organs are supplied and drained.

Pulmonary Circulation: Gas Exchange in the Lungs

Pulmonary circulation carries oxygen-poor blood from the right side of the heart to the lungs and returns oxygen-rich blood to the left side of the heart. Blood is pumped from the right ventricle into the pulmonary trunk and pulmonary arteries, then into pulmonary capillaries that surround the alveoli, where gas exchange occurs. Oxygenated blood then returns via the pulmonary veins to the left atrium. Pulmonary vessels support efficient oxygen uptake and carbon dioxide removal and differ structurally and functionally from systemic vessels, for example in their responses to changes in oxygen levels.

Blood Flow, Blood Pressure, and Vascular Resistance

Blood flow is the movement of blood through a vessel, tissue, or organ, and resistance is anything that slows or obstructs this movement. Blood pressure is the force that blood exerts on the walls of blood vessels or heart chambers. Systolic pressure results from ventricular contraction; diastolic pressure reflects ventricular relaxation. Pulse pressure is the difference between systolic and diastolic values, and mean arterial pressure is the “average” effective pressure driving blood into tissues. Pulse, the expansion and recoil of an artery, reflects the heartbeat. In systemic circulation, blood flow and blood pressure are influenced by cardiac output, vessel compliance, blood volume, blood viscosity, and vessel length and diameter. In arterioles, slight vasodilation greatly decreases resistance and increases flow, whereas slight vasoconstriction greatly increases resistance and decreases flow. Thus, as resistance rises in the arterial system, blood pressure increases and flow decreases. In veins, constriction raises venous pressure and helps drive blood back to the heart; by making the lumen more rounded, venoconstriction also decreases resistance and improves flow. Venoconstriction works together with the skeletal muscle pump, the respiratory pump, and venous valves to promote venous return.

Capillary Exchange: Filtration and Reabsorption

Small molecules cross capillary walls by simple or facilitated diffusion, and larger molecules may cross in vesicles or through clefts, fenestrations, or gaps between endothelial cells. Most fluid movement, however, occurs by bulk flow via filtration and reabsorption. Filtration, the movement of fluid out of capillaries into the interstitial space, is driven primarily by capillary hydrostatic pressure (CHP). Reabsorption, the movement of interstitial fluid back into capillaries, is driven by blood colloid osmotic pressure (BCOP). Filtration predominates at the arterial end of a capillary; in the mid-capillary region, net forces are nearly balanced; and reabsorption predominates at the venous end. Under healthy conditions, hydrostatic and colloid osmotic pressures in the interstitial fluid are negligible.

Homeostatic Regulation of the Vascular System

Blood flow, blood pressure, and tissue perfusion are regulated by neural, endocrine, and local (autoregulation) mechanisms. Neural control involves cardiovascular centers in the medulla oblongata; baroreceptors in the aorta, carotid arteries, and right atrium; and chemoreceptors that monitor blood levels of oxygen, carbon dioxide, and hydrogen ions. Endocrine mechanisms include epinephrine and norepinephrine, antidiuretic hormone (ADH), the renin–angiotensin–aldosterone system, atrial natriuretic hormone (ANH), and erythropoietin (EPO). Autoregulation refers to local control of arteriolar diameter by chemical signals from tissues and by the intrinsic myogenic response of vascular smooth muscle. Regular exercise enhances cardiovascular function and lowers the risk of disease, including hypertension, a major cause of heart attacks and strokes. Severe blood loss can lead to hypovolemic shock, a form of circulatory shock; other forms of shock may result from sepsis, vascular obstruction, or widespread inflammation.

Development of Blood Vessels and Fetal Circulation

Blood vessels develop from embryonic mesoderm. Precursor cells called hemangioblast differentiate into angioblasts, which give rise to blood vessels, and into pluripotent stem cells, which form the cellular elements of blood. These cells form scattered blood islands that connect via vascular tubes to create a primitive vascular network. As the embryo grows, the placenta forms to supply oxygen- and nutrient-rich blood to the fetus via the umbilical vein and to remove wastes through the umbilical arteries. Three major shunts modify fetal circulation: the foramen ovale and ductus arteriosus divert blood away from the nonfunctional fetal lungs into the systemic circuit, and the ductus venosus channels highly oxygenated blood from the umbilical vein toward the fetal heart.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- Compare and contrast the anatomical structure and functional roles of arteries, arterioles, capillaries, venules, and veins.
- Accurately describe the hydrostatic and osmotic forces that drive capillary exchange and explain how these forces determine movement of fluid between blood and interstitial spaces.
- List the major factors that influence blood flow, blood pressure, and peripheral resistance (including vessel diameter, blood viscosity, and vessel length).
- Explain how blood flow, blood pressure, and resistance are mathematically and physiologically interrelated, and predict how changes in one variable affect the others.
- Discuss how neural mechanisms (autonomic nervous system reflexes) and endocrine mechanisms (e.g., hormones such as epinephrine, angiotensin II, ADH, ANP) work together to maintain vascular homeostasis.
- Describe how the cardiovascular system interacts with other body systems (such as respiratory, renal, endocrine, nervous, and muscular systems) to support overall homeostasis.
- Label the major blood vessels of the pulmonary and systemic circulations on a diagram and relate them to the organs and regions they supply or drain.
- Identify the components of the hepatic portal system and describe its role in directing blood from the digestive tract to the liver for processing.
- Describe the embryonic development of major blood vessels and the key features of fetal circulation, including specialized fetal structures.
- Compare fetal circulation to postnatal circulation, explaining the structural and functional changes that occur at and after birth.

7.1: Structure and Function of Blood Vessels

7.2: Blood Flow, Blood Pressure, and Resistance

7.3: Capillary Exchange

7.4: Homeostatic Regulation of the Vascular System

7.5: Circulatory Pathways

7.6: Development of Blood Vessels and Fetal Circulation

7.7: Terms and Definitions

7.8: Chapter Review Questions

7.9: Critical Thinking Self-Assessment

7.10: Alternate Text Descriptions

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7.1: Structure and Function of Blood Vessels

Blood Vessel Anatomy

Blood is carried through the body via blood vessels. An artery is a blood vessel that carries blood away from the heart, where it branches into ever-smaller vessels. Eventually, the smallest arteries, vessels called arterioles, further branch into tiny capillaries, where nutrients and wastes are exchanged, and then combine with other vessels that exit capillaries to form venules, small blood vessels that carry blood to a vein, a larger blood vessel that returns blood to the heart.

Arteries and veins transport blood in two distinct circuits: the systemic circuit and the pulmonary circuit (Figure 7.1.1). Systemic arteries provide blood rich in oxygen to the body's tissues. The blood returned to the heart through systemic veins has less oxygen, since much of the oxygen carried by the arteries has been delivered to the cells. In contrast, in the pulmonary circuit, arteries carry blood low in oxygen exclusively to the lungs for gas exchange. Pulmonary veins then return freshly oxygenated blood from the lungs to the heart to be pumped back out into systemic circulation. Although arteries and veins differ structurally and functionally, they share certain features.

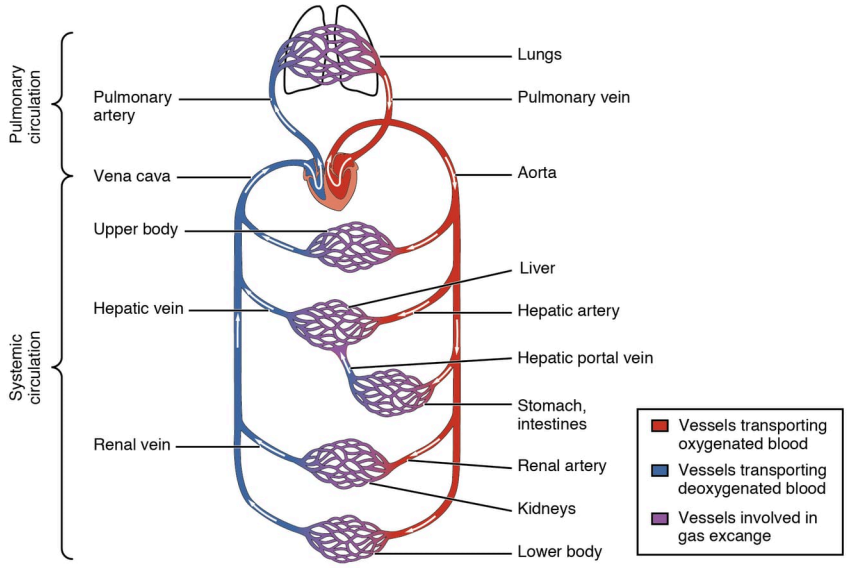


Figure Figure 7.1.1: The pulmonary circuit moves blood from the right side of the heart to the lungs and back to the heart. The systemic circuit moves blood from the left side of the heart to the head and body and returns it to the right side of the heart to repeat the cycle. The arrows indicate the direction of blood flow, and the colors show the relative levels of oxygen concentration. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.1.1.

Shared Structures

Different types of blood vessels vary slightly in their structures, but they share the same general features. Arteries and arterioles have thicker walls than veins and venules because they are closer to the heart and receive blood that is surging at a far greater pressure (Figure 7.1.2). Each type of vessel has a lumen—a hollow passageway through which blood flows. Arteries have smaller lumens than veins, a characteristic that helps to maintain the pressure of blood moving through the system. Together, their thicker walls and smaller diameters give arterial lumens a more rounded appearance in cross section than the lumens of veins.

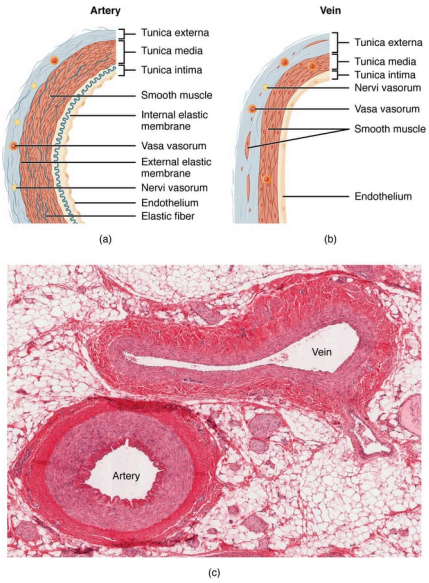


Figure 7.1.2: Structure of Blood Vessels (a) Arteries and (b) veins share the same general features, but the walls of arteries are much thicker because of the higher pressure of the blood that flows through them. (c) A micrograph shows the relative differences in thickness. LM × 160. (Micrograph provided by the Regents of the University of Michigan Medical School © 2012). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.1.2.

By the time blood has passed through capillaries and entered venules, the pressure initially exerted upon it by heart contractions has diminished. In other words, in comparison to arteries, venules and veins withstand a much lower pressure from the blood that flows through them. Their walls are considerably thinner and their lumens are correspondingly larger in diameter, allowing more blood to flow with less vessel resistance. In addition, many veins of the body, particularly those of the limbs, contain valves that assist the unidirectional flow of blood toward the heart. This is critical because blood flow becomes sluggish in the extremities, as a result of the lower pressure and the effects of gravity.

The walls of arteries and veins are largely composed of living cells and their products (including collagenous and elastic fibers); the cells require nourishment and produce waste. Since blood passes through the larger vessels relatively quickly, there is limited opportunity for blood in the lumen of the vessel to provide nourishment to or remove waste from the vessel's cells. Further, the walls of the larger vessels are too thick for nutrients to diffuse through to all of the cells. Larger arteries and veins contain small blood vessels within their walls known as the vasa vasorum—literally "vessels of the vessel"—to provide them with this critical exchange. Since the pressure within arteries is relatively high, the vasa vasorum must function in the outer layers of the vessel or the pressure exerted by the blood passing through the vessel would collapse it, preventing any exchange from occurring. The lower pressure within veins allows the vasa vasorum to be located closer to the lumen. The restriction of the vasa vasorum to the outer layers of arteries is thought to be one reason that arterial diseases are more common than venous diseases, since its location makes it more difficult to nourish the cells of the arteries and remove waste products. There are also minute nerves within the walls of both types of vessels that control the contraction and dilation of smooth muscle. These minute nerves are known as the nervi vasorum.

Both arteries and veins have the same three distinct tissue layers, called tunics (from the Latin term tunica), for the garments first worn by ancient Romans; the term tunic is also used for some modern garments. From the most interior layer to the outer, these tunics are the tunica intima, the tunica media, and the tunica externa (Figure 7.1.2). Table 7.1.1 compares and contrasts the tunics of the arteries and veins.

Table 7.1.1: Comparison of Tunics in Arteries and Veins

Vessel Wall Feature	Arteries	Veins
General appearance	Thick walls with small lumens Generally appear rounded	Thin walls with large lumens Generally appear flattened
Tunica intima	Endothelium usually appears wavy due to constriction of smooth muscle Internal elastic membrane present in larger vessels	Endothelium appears smooth Internal elastic membrane absent

Vessel Wall Feature	Arteries	Veins
Tunica media	Normally the thickest layer in arteries Smooth muscle cells and elastic fibers predominate (the proportions of these vary with distance from the heart) External elastic membrane present in larger vessels	Normally thinner than the tunica externa Smooth muscle cells and collagenous fibers predominate Nervi vasorum and vasa vasorum present External elastic membrane absent
Tunica externa	Normally thinner than the tunica media in all but the largest arteries Collagenous and elastic fibers Nervi vasorum and vasa vasorum present	Normally the thickest layer in veins Collagenous and smooth fibers predominate Some smooth muscle fibers Nervi vasorum and vasa vasorum present

Tunica Intima

The tunica intima (also called the tunica interna) is composed of epithelial and connective tissue layers. Lining the tunica intima is the specialized simple squamous epithelium called the endothelium, which is continuous throughout the entire vascular system, including the lining of the chambers of the heart. Damage to this endothelial lining and exposure of blood to the collagenous fibers beneath is one of the primary causes of clot formation. Until recently, the endothelium was viewed simply as the boundary between the blood in the lumen and the walls of the vessels. Recent studies, however, have shown that it is physiologically critical to such activities as helping to regulate capillary exchange and altering blood flow. The endothelium releases local chemicals called endothelins that can constrict the smooth muscle within the walls of the vessel to increase blood pressure. Uncompensated overproduction of endothelins may contribute to hypertension (high blood pressure) and cardiovascular disease.

Next to the endothelium is the basement membrane, or basal lamina, that effectively binds the endothelium to the connective tissue. The basement membrane provides strength while maintaining flexibility, and it is permeable, allowing materials to pass through it. The thin outer layer of the tunica intima contains a small amount of areolar connective tissue that consists primarily of elastic fibers to provide the vessel with additional flexibility; it also contains some collagenous fibers to provide additional strength.

In larger arteries, there is also a thick, distinct layer of elastic fibers known as the internal elastic membrane (also called the internal elastic lamina) at the boundary with the tunica media. Like the other components of the tunica intima, the internal elastic membrane provides structure while allowing the vessel to stretch. It is permeated with small openings that allow exchange of materials between the tunics. The internal elastic membrane is not apparent in veins. In addition, many veins, particularly in the lower limbs, contain valves formed by sections of thickened endothelium that are reinforced with connective tissue, extending into the lumen.

Under the microscope, the lumen and the entire tunica intima of a vein will appear smooth, whereas those of an artery will normally appear wavy because of the partial constriction of the smooth muscle in the tunica media, the next layer of blood vessel walls.

Tunica Media

The tunica media is the substantial middle layer of the vessel wall (Figure 7.1.2). It is generally the thickest layer in arteries, and it is much thicker in arteries than it is in veins. The tunica media consists of layers of smooth muscle supported by connective tissue that is primarily made up of elastic fibers, most of which are arranged in circular sheets. Toward the outer portion of the tunic, there are also layers of longitudinal muscle. Contraction and relaxation of the circular muscles decrease and increase the diameter of the vessel lumen, respectively. Specifically in arteries, vasoconstriction decreases blood flow as the smooth muscle in the walls of the tunica media contracts, making the lumen narrower and increasing blood pressure. Similarly, vasodilation increases blood flow as the smooth muscle relaxes, allowing the lumen to widen and blood pressure to drop. Both vasoconstriction and vasodilation are regulated in part by small vascular nerves, known as nervi vasorum, or “nerves of the vessel,” that run within the walls of blood vessels. These are generally all sympathetic fibers, although some trigger vasodilation and others induce vasoconstriction, depending upon the nature of the neurotransmitter and receptors located on the target cell. Parasympathetic

stimulation does trigger vasodilation as well as erection during sexual arousal in the external genitalia of both sexes. Nervous control over vessels tends to be more generalized than the specific targeting of individual blood vessels. Local controls, discussed later, account for this phenomenon. (k additional content for more information on these dynamic aspects of the autonomic nervous system.) Hormones and local chemicals also control blood vessels. Together, these neural and chemical mechanisms reduce or increase blood flow in response to changing body conditions, from exercise to hydration. Regulation of both blood flow and blood pressure is discussed in detail later in this chapter.

The smooth muscle layers of the tunica media are supported by a framework of collagenous fibers that also binds the tunica media to the inner and outer tunics. Along with the collagenous fibers are large numbers of elastic fibers that appear as wavy lines in prepared slides. Separating the tunica media from the outer tunica externa in larger arteries is the external elastic membrane (also called the external elastic lamina), which also appears wavy in slides. This structure is not usually n in smaller arteries, nor is it n in veins.

Tunica Externa

The outer tunic, the tunica externa (also called the tunica adventitia), is a substantial sheath of connective tissue composed primarily of collagenous fibers. Some bands of elastic fibers are found here as well. The tunica externa in veins also contains groups of smooth muscle fibers. This is normally the thickest tunic in veins and may be thicker than the tunica media in some larger arteries. The outer layers of the tunica externa are not distinct but rather blend with the surrounding connective tissue outside the vessel, helping to hold the vessel in relative position. If you are able to palpate some of the superficial veins on your upper limbs and try to move them, you will find that the tunica externa prevents this. If the tunica externa did not hold the vessel in place, any movement would likely result in disruption of blood flow.

Arteries

An artery is a blood vessel that conducts blood away from the heart. All arteries have relatively thick walls that can withstand the high pressure of blood ejected from the heart. However, those close to the heart have the thickest walls, containing a high percentage of elastic fibers in all three of their tunics. This type of artery is known as an elastic artery (Figure 7.1.3). Vessels larger than 10 mm in diameter are typically elastic. Their abundant elastic fibers allow them to expand, as blood pumped from the ventricles passes through them, and then to recoil after the surge has passed. If artery walls were rigid and unable to expand and recoil, their resistance to blood flow would greatly increase and blood pressure would rise to even higher levels, which would in turn require the heart to pump harder to increase the volume of blood expelled by each pump (the stroke volume) and maintain adequate pressure and flow. Artery walls would have to become even thicker in response to this increased pressure. The elastic recoil of the vascular wall helps to maintain the pressure gradient that drives the blood through the arterial system. An elastic artery is also known as a conducting artery, because the large diameter of the lumen enables it to accept a large volume of blood from the heart and conduct it to smaller branches.

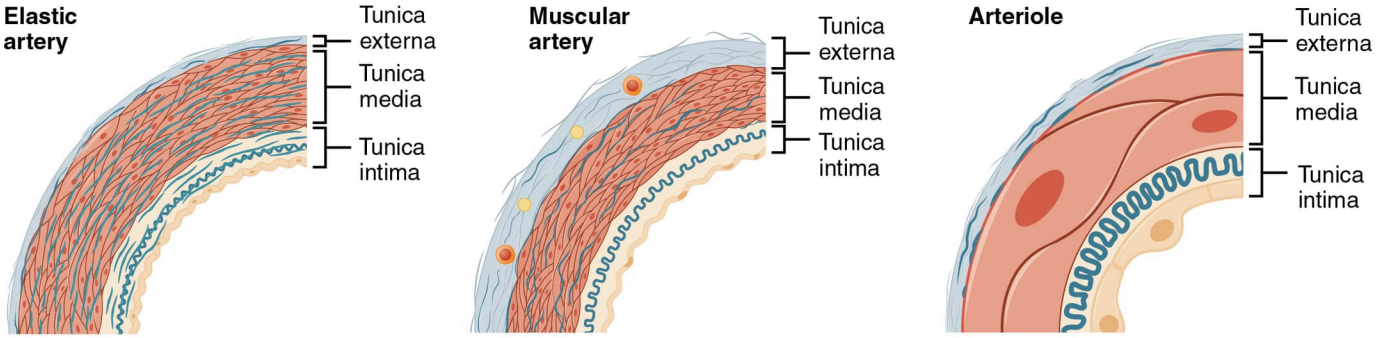


Figure 7.1.3: Comparison of the walls of an elastic artery, a muscular artery, and an arteriole is shown. In terms of scale, the diameter of an arteriole is measured in micrometers compared to millimeters for elastic and muscular arteries. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.1.3.

Farther from the heart, where the surge of blood has dampened, the percentage of elastic fibers in an artery's tunica intima decreases and the amount of smooth muscle in its tunica media increases. The artery at this point is described as a muscular artery. The diameter of muscular arteries typically ranges from 0.1 mm to 10 mm. Their thick tunica media allows muscular arteries to play a leading role in vasoconstriction. In contrast, their decreased quantity of elastic fibers limits their ability to expand. Fortunately, because the blood pressure has eased by the time it reaches these more distant vessels, elasticity has become less

important.

Notice that although the distinctions between elastic and muscular arteries are important, there is no “line of demarcation” where an elastic artery suddenly becomes muscular. Rather, there is a gradual transition as the vascular tree repeatedly branches. In turn, muscular arteries branch to distribute blood to the vast network of arterioles. For this reason, a muscular artery is also known as a distributing artery.

Arterioles

An arteriole is a very small artery that leads to a capillary. Arterioles have the same three tunics as the larger vessels, but the thickness of each is greatly diminished. The critical endothelial lining of the tunica intima is intact. The tunica media is restricted to one or two smooth muscle cell layers in thickness. The tunica externa remains but is very thin (Figure 7.1.3).

With a lumen averaging 30 micrometers or less in diameter, arterioles are critical in slowing down—or resisting—blood flow and, thus, causing a substantial drop in blood pressure. Because of this, you may them referred to as resistance vessels. The muscle fibers in arterioles are normally slightly contracted, causing arterioles to maintain a consistent muscle tone—in this case referred to as vascular tone—in a similar manner to the muscular tone of skeletal muscle. In reality, all blood vessels exhibit vascular tone due to the partial contraction of smooth muscle. The importance of the arterioles is that they will be the primary site of both resistance and regulation of blood pressure. The precise diameter of the lumen of an arteriole at any given moment is determined by neural and chemical controls, and vasoconstriction and vasodilation in the arterioles are the primary mechanisms for distribution of blood flow.

Capillaries

A capillary is a microscopic channel that supplies blood to the tissues, a process called perfusion. Exchange of gases and other substances occurs between the blood in capillaries and the surrounding cells and their tissue fluid (interstitial fluid). The diameter of a capillary lumen ranges from 5–10 micrometers; the smallest are just barely wide enough for an erythrocyte to squeeze through. Flow through capillaries is often described as microcirculation.

The wall of a capillary consists of the endothelial layer surrounded by a basement membrane with occasional smooth muscle fibers. There is some variation in wall structure: In a large capillary, several endothelial cells bordering each other may line the lumen; in a small capillary, there may be only a single cell layer that wraps around to contact itself.

For capillaries to function, their walls must be leaky, allowing substances to pass through. There are three major types of capillaries, which differ according to their degree of “leakiness:” continuous, fenestrated, and sinusoid capillaries (Figure 7.1.4).

Continuous Capillaries

The most common type of capillary, the continuous capillary, is found in almost all vascularized tissues. Continuous capillaries are characterized by a complete endothelial lining with tight junctions between endothelial cells. Although a tight junction is usually impermeable and only allows for the passage of water and ions, they are often incomplete in capillaries, leaving intercellular clefts that allow for exchange of water and other very small molecules between the blood plasma and the interstitial fluid. Substances that can pass between cells include metabolic products, such as glucose, water, and small hydrophobic molecules like gases and hormones, as well as various leukocytes. Continuous capillaries not associated with the brain are rich in transport vesicles, contributing to either endocytosis or exocytosis. Those in the brain are part of the blood-brain barrier. Here, there are tight junctions and no intercellular clefts, plus a thick basement membrane and astrocyte extensions called end feet; these structures combine to prevent the movement of nearly all substances.

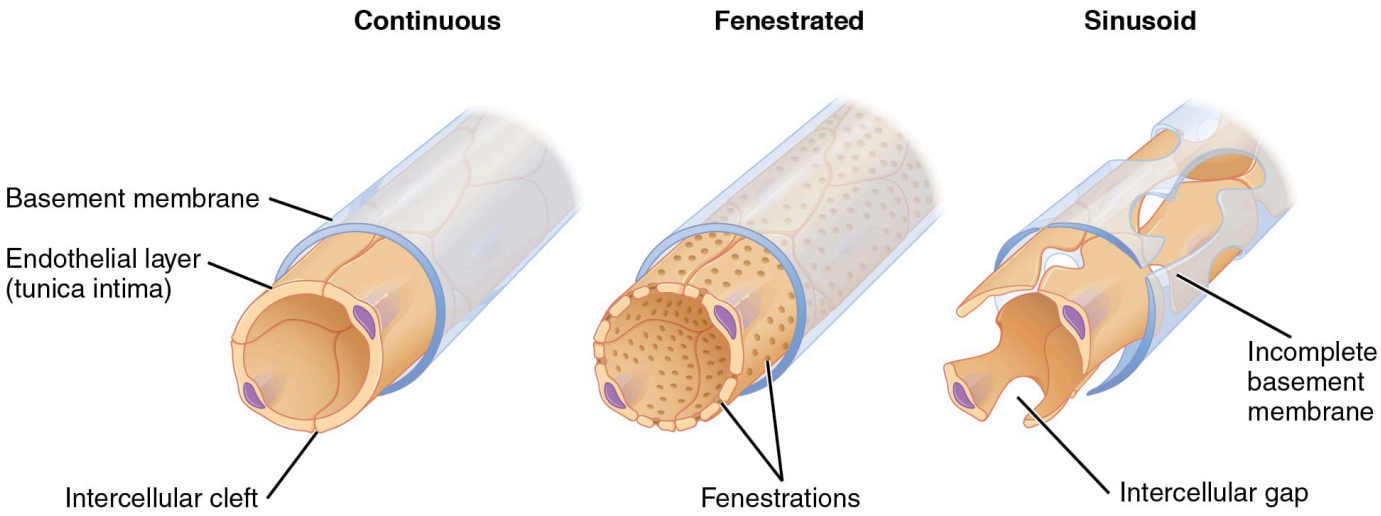


Figure 7.1.4: The three major types of capillaries: continuous, fenestrated, and sinusoid. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.1.4.

Fenestrated Capillaries

A fenestrated capillary is one that has pores (or fenestrations) in addition to tight junctions in the endothelial lining. These make the capillary permeable to larger molecules. The number of fenestrations and their degree of permeability vary, however, according to their location. Fenestrated capillaries are common in the small intestine, which is the primary site of nutrient absorption, as well as in the kidneys, which filter the blood. They are also found in the choroid plexus of the brain and many endocrine structures, including the hypothalamus, pituitary, pineal, and thyroid glands.

Sinusoid Capillaries

A sinusoid capillary (or sinusoid) is the least common type of capillary. Sinusoid capillaries are flattened, and they have extensive intercellular gaps and incomplete basement membranes, in addition to intercellular clefts and fenestrations. This gives them an appearance not unlike Swiss cheese. These very large openings allow for the passage of the largest molecules, including plasma proteins and even cells. Blood flow through sinusoids is very slow, allowing more time for exchange of gases, nutrients, and wastes. Sinusoids are found in the liver and spleen, bone marrow, lymph nodes (where they carry lymph, not blood), and many endocrine glands including the pituitary and adrenal glands. Without these specialized capillaries, these organs would not be able to provide their myriad of functions. For example, when bone marrow forms new blood cells, the cells must enter the blood supply and can only do so through the large openings of a sinusoid capillary; they cannot pass through the small openings of continuous or fenestrated capillaries. The liver also requires extensive specialized sinusoid capillaries in order to process the materials brought to it by the hepatic portal vein from both the digestive tract and spleen, and to release plasma proteins into circulation.

Metarterioles and Capillary Beds

A metarteriole is a type of vessel that has structural characteristics of both an arteriole and a capillary. Slightly larger than the typical capillary, the smooth muscle of the tunica media of the metarteriole is not continuous but forms rings of smooth muscle (sphincters) prior to the entrance to the capillaries. Each metarteriole arises from a terminal arteriole and branches to supply blood to a capillary bed that may consist of 10–100 capillaries.

The precapillary sphincters, circular smooth muscle cells that surround the capillary at its origin with the metarteriole, tightly regulate the flow of blood from a metarteriole to the capillaries it supplies. Their function is critical: If all of the capillary beds in the body were to open simultaneously, they would collectively hold every drop of blood in the body and there would be none in the arteries, arterioles, venules, veins, or the heart itself. Normally, the precapillary sphincters are closed. When the surrounding tissues need oxygen and have excess waste products, the precapillary sphincters open, allowing blood to flow through and exchange to occur before closing once more (Figure 7.1.5). If all of the precapillary sphincters in a capillary bed are closed, blood will flow from the metarteriole directly into a thoroughfare channel and then into the venous circulation, bypassing the capillary bed entirely. This creates what is known as a vascular shunt. In addition, an arteriovenous anastomosis may bypass the capillary

bed and lead directly to the venous system.

Although you might expect blood flow through a capillary bed to be smooth, in reality, it moves with an irregular, pulsating flow. This pattern is called vasomotion and is regulated by chemical signals that are triggered in response to changes in internal conditions, such as oxygen, carbon dioxide, hydrogen ion, and lactic acid levels. For example, during strenuous exercise when oxygen levels decrease and carbon dioxide, hydrogen ion, and lactic acid levels all increase, the capillary beds in skeletal muscle are open, as they would be in the digestive system when nutrients are present in the digestive tract. During sleep or rest periods, vessels in both areas are largely closed; they open only occasionally to allow oxygen and nutrient supplies to travel to the tissues to maintain basic life processes.

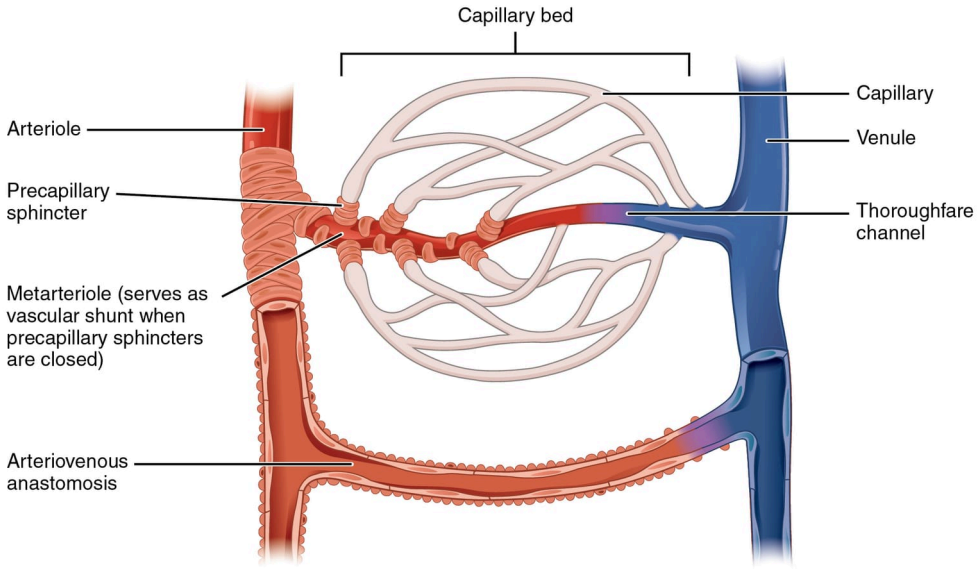


Figure 7.1.5: In a capillary bed, arterioles give rise to metarterioles. Precapillary sphincters located at the junction of a metarteriole with a capillary regulate blood flow. A thoroughfare channel connects the metarteriole to a venule. An arteriovenous anastomosis, which directly connects the arteriole with the venule, is shown at the bottom. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.1.5.

Venules

A venule is an extremely small vein, generally 8–100 micrometers in diameter. Postcapillary venules join multiple capillaries exiting from a capillary bed. Multiple venules join to form veins. The walls of venules consist of endothelium, a thin middle layer with a few muscle cells and elastic fibers, plus an outer layer of connective tissue fibers that constitute a very thin tunica externa. Venules as well as capillaries are the primary sites of emigration or diapedesis, in which the white blood cells adhere to the endothelial lining of the vessels and then squeeze through adjacent cells to enter the tissue fluid.

Veins

A vein is a blood vessel that conducts blood toward the heart. Compared to arteries, veins are thin-walled vessels with large and irregular lumens (Figure 7.1.6). Because they are low-pressure vessels, larger veins are commonly equipped with valves that promote the unidirectional flow of blood toward the heart and prevent backflow toward the capillaries caused by the inherent low blood pressure in veins as well as the pull of gravity. Table 7.1.2 compares the features of arteries and veins.

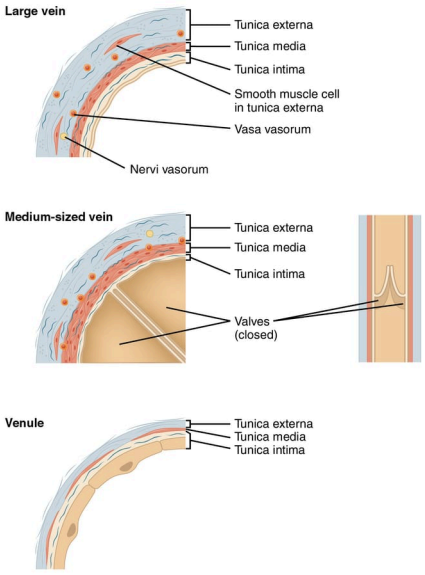


Figure 7.1.6: Many veins have valves to prevent back flow of blood, whereas venules do not. In terms of scale, the diameter of a venule is measured in micrometers compared to millimeters for veins. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.1.6.

Table 7.1.2: Comparison of Arteries and Veins

Characteristic	Arteries	Veins
Direction of blood flow	Conducts blood away from the heart	Conducts blood toward the heart
General appearance	Rounded	Irregular, often collapsed
Pressure	High	Low
Wall thickness	Thick	Thin
Relative oxygen concentration	Higher in systemic arteries Lower in pulmonary arteries	Lower in systemic veins Higher in pulmonary veins
Valves	Not present	Present most commonly in limbs and in veins inferior to the heart

Disorders of the Cardiovascular System: Edema and Varicose Veins

Despite the presence of valves and the contributions of other anatomical and physiological adaptations we will cover shortly, over the course of a day, some blood will inevitably pool, especially in the lower limbs, due to the pull of gravity. Any blood that accumulates in a vein will increase the pressure within it, which can then be reflected back into the smaller veins, venules, and eventually even the capillaries. Increased pressure will promote the flow of fluids out of the capillaries and into the interstitial fluid. The presence of excess tissue fluid around the cells leads to a condition called edema.

Most people experience a daily accumulation of tissue fluid, especially if they spend much of their work life on their feet (like most health professionals). However, clinical edema goes beyond normal swelling and requires medical treatment. Edema has many potential causes, including hypertension and heart failure, severe protein deficiency, renal failure, and many others. In order to treat edema, which is a sign rather than a discrete disorder, the underlying cause must be diagnosed and alleviated.



Figure 7.1.7: Varicose veins are commonly found in the lower limbs. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.1.7.

Edema may be accompanied by varicose veins, especially in the superficial veins of the legs (Figure 7.1.7). This disorder arises when defective valves allow blood to accumulate within the veins, causing them to distend, twist, and become visible on the surface of the integument. Varicose veins may occur in all people, but are more common in females and are often related to pregnancy. More than simple cosmetic blemishes, varicose veins are often painful and sometimes itchy or throbbing. Without treatment, they tend to grow worse over time. The use of support hose, as well as elevating the feet and legs whenever possible, may be helpful in alleviating this condition. Laser surgery and interventional radiologic procedures can reduce the size and severity of varicose veins. Severe cases may require conventional surgery to remove the damaged vessels. As there are typically redundant circulation patterns, that is, anastomoses, for the smaller and more superficial veins, removal does not typically impair the circulation. There is evidence that patients with varicose veins suffer a greater risk of developing a thrombus or clot.

Veins as Blood Reservoirs

In addition to their primary function of returning blood to the heart, veins may be considered blood reservoirs, since systemic veins contain approximately 64 percent of the blood volume at any given time (Table 7.1.3). Their ability to hold this much blood is due to their high capacitance, that is, their capacity to distend (expand) readily to store a high volume of blood, even at a low pressure. The large lumens and relatively thin walls of veins make them far more distensible than arteries; thus, they are said to be capacitance vessels.

Table 7.1.3: Distribution of Blood Volume in the Circulatory System

Major Category	Subcategory	Percentage of Total Blood Volume
Systemic Circulation	**Total Systemic Circulation**	84%
Systemic Circulation	**Systemic Veins**	64%
Systemic Circulation	**Large Veins**	18%
Systemic Circulation	**Large Venous Networks (Liver, Bone Marrow, and Integument)**	21%
Systemic Circulation	**Venules and Medium-Sized Veins**	25%
Systemic Circulation	**Systemic Arteries**	13%
Systemic Circulation	**Arterioles**	2%
Systemic Circulation	**Muscular Arteries**	5%
Systemic Circulation	**Elastic Arteries**	4%
Systemic Circulation	**Aorta**	2%

Major Category	Subcategory	Percentage of Total Blood Volume
Systemic Circulation	**Systemic Capillaries**	7%
Pulmonary Circulation	**Total Pulmonary Circulation**	9%
Pulmonary Circulation	**Pulmonary Veins**	4%
Pulmonary Circulation	**Pulmonary Capillaries**	2%
Pulmonary Circulation	**Pulmonary Arteries**	3%
Heart	**Heart**	7%

When blood flow needs to be redistributed to other portions of the body, the vasomotor center located in the medulla oblongata sends sympathetic stimulation to the smooth muscles in the walls of the veins, causing constriction—or in this case, venoconstriction. Less dramatic than the vasoconstriction n in smaller arteries and arterioles, venoconstriction may be likened to a "stiffening" of the vessel wall. This increases pressure on the blood within the veins, speeding its return to the heart. As you will note in Figure 7.1.8, approximately 21 percent of the venous blood is located in venous networks within the liver, bone marrow, and integument. This volume of blood is referred to as venous reserve. Through venoconstriction, this "reserve" volume of blood can get back to the heart more quickly for redistribution to other parts of the circulation.

Career Connection

Vascular Surgeons and Technicians

Vascular surgery is a specialty in which the physician deals primarily with diseases of the vascular portion of the cardiovascular system. This includes repair and replacement of diseased or damaged vessels, removal of plaque from vessels, minimally invasive procedures including the insertion of venous catheters, and traditional surgery. Following completion of medical school, the physician generally completes a 5-year surgical residency followed by an additional 1 to 2 years of vascular specialty training. In the United States, most vascular surgeons are members of the Society of Vascular Surgery.

Vascular technicians are specialists in imaging technologies that provide information on the health of the vascular system. They may also assist physicians in treating disorders involving the arteries and veins. This profession often overlaps with cardiovascular technology, which would also include treatments involving the heart. Although recognized by the American Medical Association, there are currently no licensing requirements for vascular technicians, and licensing is voluntary. Vascular technicians typically have an Associate's degree or certificate, involving 18 months to 2 years of training. The United States Bureau of Labor projects this profession to grow by 29 percent from 2010 to 2020.

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7.2: Blood Flow, Blood Pressure, and Resistance

Hemodynamics: Blood Flow and Blood Pressure

Blood flow refers to the movement of blood through a vessel, tissue, or organ, and is usually expressed in terms of volume of blood per unit of time. It is initiated by the contraction of the ventricles of the heart. Ventricular contraction ejects blood into the major arteries, resulting in flow from regions of higher pressure to regions of lower pressure, as blood encounters smaller arteries and arterioles, then capillaries, then the venules and veins of the venous system. This section discusses a number of critical variables that contribute to blood flow throughout the body. It also discusses the factors that impede or slow blood flow, a phenomenon known as resistance.

As noted earlier, hydrostatic pressure is the force exerted by a fluid due to gravitational pull, usually against the wall of the container in which it is located. One form of hydrostatic pressure is blood pressure, the force exerted by blood upon the walls of the blood vessels or the chambers of the heart. Blood pressure may be measured in capillaries and veins, as well as the vessels of the pulmonary circulation; however, the term blood pressure without any specific descriptors typically refers to systemic arterial blood pressure—that is, the pressure of blood flowing in the arteries of the systemic circulation. In clinical practice, this pressure is measured in mm Hg and is usually obtained using the brachial artery of the arm.

Components of Arterial Blood Pressure

Arterial blood pressure in the larger vessels consists of several distinct components (Figure 7.2.1): systolic and diastolic pressures, pulse pressure, and mean arterial pressure.

Systolic and Diastolic Pressures

When systemic arterial blood pressure is measured, it is recorded as a ratio of two numbers (e.g., 120/80 is a normal adult blood pressure), expressed as systolic pressure over diastolic pressure. The systolic pressure is the higher value (typically around 120 mm Hg) and reflects the arterial pressure resulting from the ejection of blood during ventricular contraction, or systole. The diastolic pressure is the lower value (usually about 80 mm Hg) and represents the arterial pressure of blood during ventricular relaxation, or diastole.

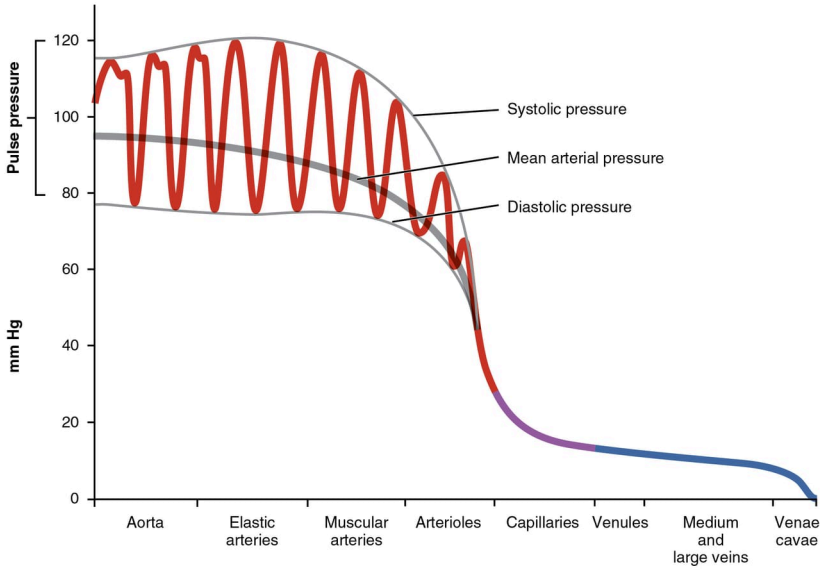


Figure 7.2.1: The graph shows the components of blood pressure throughout the blood vessels, including systolic, diastolic, mean arterial, and pulse pressures. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.2.1.

Pulse Pressure

As shown in Figure 7.2.1, the difference between the systolic pressure and the diastolic pressure is the pulse pressure. For example, an individual with a systolic pressure of 120 mm Hg and a diastolic pressure of 80 mm Hg would have a pulse pressure of 40 mmHg.

Generally, a pulse pressure should be at least 25 percent of the systolic pressure. A pulse pressure below this level is described as low or narrow. This may occur, for example, in patients with a low stroke volume, which may be seen in congestive heart failure, stenosis of the aortic valve, or significant blood loss following trauma. In contrast, a high or wide pulse pressure is common in healthy people following strenuous exercise, when their resting pulse pressure of 30–40 mm Hg may increase temporarily to 100 mm Hg as stroke volume increases. A persistently high pulse pressure at or above 100 mm Hg may indicate excessive resistance in the arteries and can be caused by a variety of disorders. Chronic high resting pulse pressures can degrade the heart, brain, and kidneys, and warrant medical treatment.

Mean Arterial Pressure

Mean arterial pressure (MAP) represents the "average" pressure of blood in the arteries, that is, the average force driving blood into vessels that serve the tissues. Mean is a statistical concept and is calculated by taking the sum of the values divided by the number of values. Although complicated to measure directly and complicated to calculate, MAP can be approximated by adding the diastolic pressure to one-third of the pulse pressure or systolic pressure minus the diastolic pressure:

$$\text{MAP} = \text{ diastolic BP} + \frac{(\text{systolic BP-diastolic BP })}{3} \tag{7.2.1}$$

or

$$\text{MAP} = \text{ diastolic BP} + \frac{(\text{pulse pressure})}{3} \tag{7.2.2}$$

In Figure \(\PageIndex{1}\), this value is approximately 80 + (120 – 80) / 3, or 93.33. Normally, the MAP falls within the range of 70–110 mm Hg. If the value falls below 60 mm Hg for an extended time, blood pressure will not be high enough to ensure circulation to and through the tissues, which results in ischemia, or insufficient blood flow. A condition called hypoxia, inadequate oxygenation of tissues, commonly accompanies ischemia. The term hypoxemia refers to low levels of oxygen in systemic arterial blood. Neurons are especially sensitive to hypoxia and may die or be damaged if blood flow and oxygen supplies are not quickly restored.

Pulse

After blood is ejected from the heart, elastic fibers in the arteries help maintain a high-pressure gradient as they expand to accommodate the blood, then recoil. This expansion and recoiling effect, known as the pulse, can be palpated manually or measured electronically. Although the effect diminishes over distance from the heart, elements of the systolic and diastolic components of the pulse are still evident down to the level of the arterioles.

Because pulse indicates heart rate, it is measured clinically to provide clues to a patient's state of health. It is recorded as beats per minute. Both the rate and the strength of the pulse are important clinically. A high or irregular pulse rate can be caused by physical activity or other temporary factors, but it may also indicate a heart condition. The pulse strength indicates the strength of ventricular contraction and cardiac output. If the pulse is strong, then systolic pressure is high. If it is weak, systolic pressure has fallen, and medical intervention may be warranted.

Pulse can be palpated manually by placing the tips of the fingers across an artery that runs close to the body surface and pressing lightly. While this procedure is normally performed using the radial artery in the wrist or the common carotid artery in the neck, any superficial artery that can be palpated may be used (Figure 7.2.2). Common sites to find a pulse include temporal and facial arteries in the head, brachial arteries in the upper arm, femoral arteries in the thigh, popliteal arteries behind the knees, posterior tibial arteries near the medial tarsal regions, and dorsalis pedis arteries in the feet. A variety of commercial electronic devices are also available to measure pulse.

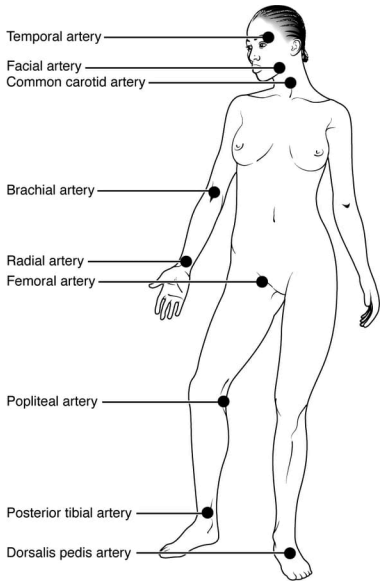


Figure 7.2.2: The pulse is most readily measured at the radial artery, but can be measured at any of the pulse points shown. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.2.2.

Measurement of Blood Pressure

Blood pressure is one of the critical parameters measured on virtually every patient in every healthcare setting. The technique used today was developed more than 100 years ago by a pioneering Russian physician, Dr. Nikolai Korotkoff. Turbulent blood flow through the vessels can be heard as a soft ticking while measuring blood pressure; these sounds are known as Korotkoff sounds. The technique of measuring blood pressure requires the use of a sphygmomanometer (a blood pressure cuff attached to a measuring device) and a stethoscope. The technique is as follows:

- The clinician wraps an inflatable cuff tightly around the patient's arm at about the level of the heart.
- The clinician squeezes a rubber pump to inject air into the cuff, raising pressure around the artery and temporarily cutting off blood flow into the patient's arm.
- The clinician places the stethoscope on the patient's antecubital region and, while gradually allowing air within the cuff to escape, listens for the Korotkoff sounds.

Although there are five recognized Korotkoff sounds, only two are normally recorded. Initially, no sounds are heard since there is no blood flow through the vessels, but as air pressure drops, the cuff relaxes, and blood flow returns to the arm. As shown in Figure 7.2.3, the first sound heard through the stethoscope—the first Korotkoff sound—indicates systolic pressure. As more air is released from the cuff, blood is able to flow freely through the brachial artery and all sounds disappear. The point at which the last sound is heard is recorded as the patient's diastolic pressure.

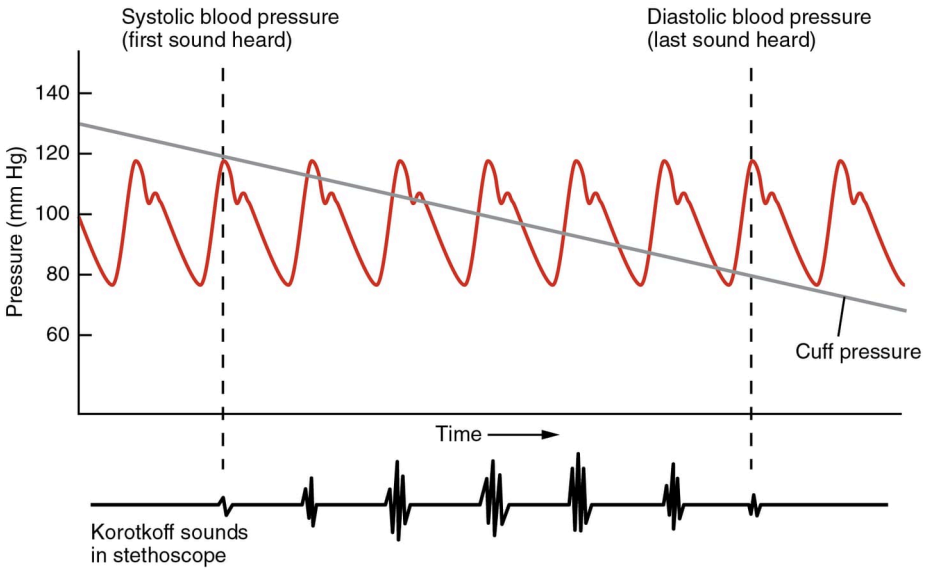


Figure 7.2.3: When pressure in a sphygmomanometer cuff is released, a clinician can hear the Korotkoff sounds. In this graph, a blood pressure tracing is aligned to a measurement of systolic and diastolic pressures. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.2.3.

The majority of hospitals and clinics have automated equipment for measuring blood pressure that work on the same principles. An even more recent innovation is a small instrument that wraps around a patient's wrist. The patient then holds the wrist over the heart while the device measures blood flow and records pressure.

Variables Affecting Blood Flow and Blood Pressure

Five variables influence blood flow and blood pressure:

- Cardiac output
- Compliance
- Volume of the blood
- Viscosity of the blood
- Blood vessel length and diameter

Recall that blood moves from higher pressure to lower pressure. It is pumped from the heart into the arteries at high pressure. If you increase pressure in the arteries (afterload), and cardiac function does not compensate, blood flow will actually decrease. In the venous system, the opposite relationship is true. Increased pressure in the veins does not decrease flow as it does in arteries, but actually increases flow. Since pressure in the veins is normally relatively low, for blood to flow back into the heart, the pressure in the atria during atrial diastole must be even lower. It normally approaches zero, except when the atria contract (see Figure 7.2.1).

Cardiac Output

Cardiac output is the measurement of blood flow from the heart through the ventricles, and is usually measured in liters per minute. Any factor that causes cardiac output to increase, by elevating heart rate or stroke volume or both, will elevate blood pressure and promote blood flow. These factors include sympathetic stimulation, the catecholamines epinephrine and norepinephrine, thyroid hormones, and increased calcium ion levels. Conversely, any factor that decreases cardiac output, by decreasing heart rate or stroke volume or both, will decrease arterial pressure and blood flow. These factors include parasympathetic stimulation, elevated or decreased potassium ion levels, decreased calcium levels, anoxia, and acidosis.

Compliance

Compliance is the ability of any compartment to expand to accommodate increased content. A metal pipe, for example, is not compliant, whereas a balloon is. The greater the compliance of an artery, the more effectively it is able to expand to accommodate

surges in blood flow without increased resistance or blood pressure. Veins are more compliant than arteries and can expand to hold more blood. When vascular disease causes stiffening of arteries, compliance is reduced and resistance to blood flow is increased. The result is more turbulence, higher pressure within the vessel, and reduced blood flow. This increases the work of the heart.

A Mathematical Approach to Factors Affecting Blood Flow

Jean Louis Marie Poiseuille was a French physician and physiologist who devised a mathematical equation describing blood flow and its relationship to known parameters. The same equation also applies to engineering studies of the flow of fluids. Although understanding the math behind the relationships among the factors affecting blood flow is not necessary to understand blood flow, it can help solidify an understanding of their relationships. Please note that even if the equation looks intimidating, breaking it down into its components and following the relationships will make these relationships clearer, even if you are weak in math. Focus on the three critical variables: radius (r), vessel length (λ), and viscosity (η).

Poiseuille's equation:

$$\text{Blood flow} = \frac{\pi \Delta P r^4}{8 \eta \lambda} \tag{7.2.3}$$

- π is the Greek letter pi, used to represent the mathematical constant that is the ratio of a circle's circumference to its diameter. It may commonly be represented as 3.14, although the actual number extends to infinity.
- ΔP represents the difference in pressure.
- r^4 is the radius (one-half of the diameter) of the vessel to the fourth power.
- η is the Greek letter eta and represents the viscosity of the blood.

One of several things this equation allows us to do is calculate the resistance in the vascular system. Normally this value is extremely difficult to measure, but it can be calculated from this known relationship:

$$\text{Blood flow} = \frac{\Delta P}{\text{Resistance}} \tag{7.2.4}$$

If we rearrange this slightly,

$$\text{Resistance} = \frac{\Delta P}{\text{Blood flow}} \tag{7.2.5}$$

Then by substituting Pouseille's equation for blood flow

$$\text{Resistance} = \frac{8 \eta \lambda}{\pi r^4} \tag{7.2.6}$$

By examining this equation, you can see that there are only three variables: viscosity, vessel length, and radius, since 8 and π are both constants. The important thing to remember is this: Two of these variables, viscosity and vessel length, will change slowly in the body. Only one of these factors, the radius, can be changed rapidly by vasoconstriction and vasodilation, thus dramatically impacting resistance and flow. Further, small changes in the radius will greatly affect flow, since it is raised to the fourth power in the equation.

We have briefly considered how cardiac output and blood volume impact blood flow and pressure; the next step is to see how the other variables (contraction, vessel length, and viscosity) articulate with Pouseille's equation and what they can teach us about the impact on blood flow.

Blood Volume

The relationship between blood volume, blood pressure, and blood flow is intuitively obvious. Water may merely trickle along a creek bed in a dry season, but rush quickly and under great pressure after a heavy rain. Similarly, as blood volume decreases,

pressure and flow decrease. As blood volume increases, pressure and flow increase.

Under normal circumstances, blood volume varies little. Low blood volume, called hypovolemia, may be caused by bleeding, dehydration, vomiting, severe burns, or some medications used to treat hypertension. It is important to recognize that other regulatory mechanisms in the body are so effective at maintaining blood pressure that an individual may be asymptomatic until 10–20 percent of the blood volume has been lost. Treatment typically includes intravenous fluid replacement.

Hypervolemia, excessive fluid volume, may be caused by retention of water and sodium, as seen in patients with heart failure, liver cirrhosis, some forms of kidney disease, hyperaldosteronism, and some glucocorticoid steroid treatments. Restoring homeostasis in these patients depends upon reversing the condition that triggered the hypervolemia.

Blood Viscosity

Viscosity is the thickness of fluids that affects their ability to flow. Clean water, for example, is less viscous than mud. The viscosity of blood is directly proportional to resistance and inversely proportional to flow; therefore, any condition that causes viscosity to increase will also increase resistance and decrease flow. For example, imagine sipping milk, then a milkshake, through the same size straw. You experience more resistance and therefore less flow from the milkshake. Conversely, any condition that causes viscosity to decrease (such as when the milkshake melts) will decrease resistance and increase flow.

Normally the viscosity of blood does not change over short periods of time. The two primary determinants of blood viscosity are the formed elements and plasma proteins. Since the vast majority of formed elements are erythrocytes, any condition affecting erythropoiesis, such as polycythemia or anemia, can alter viscosity. Since most plasma proteins are produced by the liver, any condition affecting liver function can also change the viscosity slightly and therefore alter blood flow. Liver abnormalities such as hepatitis, cirrhosis, alcohol damage, and drug toxicities result in decreased levels of plasma proteins, which decrease blood viscosity. While leukocytes and platelets are normally a small component of the formed elements, there are some rare conditions in which severe overproduction can impact viscosity as well.

Vessel Length and Diameter

The length of a vessel is directly proportional to its resistance: the longer the vessel, the greater the resistance and the lower the flow. As with blood volume, this makes intuitive sense, since the increased surface area of the vessel will impede the flow of blood. Likewise, if the vessel is shortened, the resistance will decrease and flow will increase.

The length of our blood vessels increases throughout childhood as we grow, of course, but is unchanging in adults under normal physiological circumstances. Further, the distribution of vessels is not the same in all tissues. Adipose tissue does not have an extensive vascular supply. One pound of adipose tissue contains approximately 200 miles of vessels, whereas skeletal muscle contains more than twice that. Overall, vessels decrease in length only during loss of mass or amputation. An individual weighing 150 pounds has approximately 60,000 miles of vessels in the body. Gaining about 10 pounds adds from 2000 to 4000 miles of vessels, depending upon the nature of the gained tissue. One of the great benefits of weight reduction is the reduced stress to the heart, which does not have to overcome the resistance of as many miles of vessels.

In contrast to length, the diameter of blood vessels changes throughout the body, according to the type of vessel, as we discussed earlier. The diameter of any given vessel may also change frequently throughout the day in response to neural and chemical signals that trigger vasodilation and vasoconstriction. The vascular tone of the vessel is the contractile state of the smooth muscle and the primary determinant of diameter, and thus of resistance and flow. The effect of vessel diameter on resistance is inverse: Given the same volume of blood, an increased diameter means there is less blood contacting the vessel wall, thus lower friction and lower resistance, subsequently increasing flow. A decreased diameter means more of the blood contacts the vessel wall, and resistance increases, subsequently decreasing flow.

The influence of lumen diameter on resistance is dramatic: A slight increase or decrease in diameter causes a huge decrease or increase in resistance. This is because resistance is inversely proportional to the radius of the blood vessel (one-half of the vessel's diameter) raised to the fourth power ($R = 1/r^4$). This means, for example, that if an artery or arteriole constricts to one-half of its original radius, the resistance to flow will increase 16 times. And if an artery or arteriole dilates to twice its initial radius, then resistance in the vessel will decrease to 1/16 of its original value and flow will increase 16 times.

The Roles of Vessel Diameter and Total Area in Blood Flow and Blood Pressure

Recall that we classified arterioles as resistance vessels, because given their small lumen, they dramatically slow the flow of blood from arteries. In fact, arterioles are the site of greatest resistance in the entire vascular network. This may seem surprising, given that capillaries have a smaller size. How can this phenomenon be explained?

Figure 7.2.4 compares vessel diameter, total cross-sectional area, average blood pressure, and blood velocity through the systemic vessels. Notice in parts (a) and (b) that the total cross-sectional area of the body's capillary beds is far greater than any other type of vessel. Although the diameter of an individual capillary is significantly smaller than the diameter of an arteriole, there are vastly more capillaries in the body than there are other types of blood vessels. Part (c) shows that blood pressure drops unevenly as blood travels from arteries to arterioles, capillaries, venules, and veins, and encounters greater resistance. However, the site of the most precipitous drop, and the site of greatest resistance, is the arterioles. This explains why vasodilation and vasoconstriction of arterioles play more significant roles in regulating blood pressure than do the vasodilation and vasoconstriction of other vessels.

Part (d) shows that the velocity (speed) of blood flow decreases dramatically as the blood moves from arteries to arterioles to capillaries. This slow flow rate allows more time for exchange processes to occur. As blood flows through the veins, the rate of velocity increases, as blood is returned to the heart.

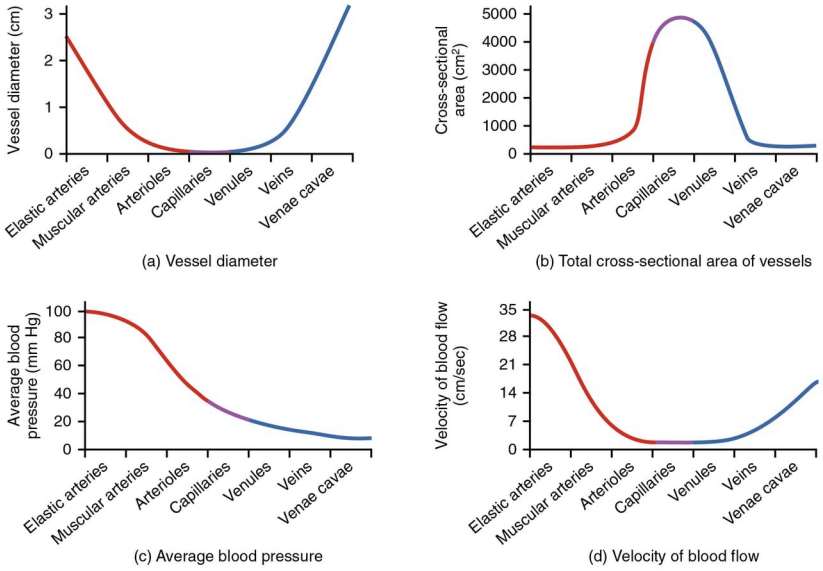


Figure 7.2.4: The relationships among blood vessels that can be compared include (a) vessel diameter, (b) total cross-sectional area, (c) average blood pressure, and (d) velocity of blood flow. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.2.4.

Disorders of the Cardiovascular System: Arteriosclerosis

Compliance allows an artery to expand when blood is pumped through it from the heart, and then to recoil after the surge has passed. This helps promote blood flow. In arteriosclerosis, compliance is reduced, and pressure and resistance within the vessel increase. This is a leading cause of hypertension and coronary heart disease, as it causes the heart to work harder to generate a pressure great enough to overcome the resistance.

Arteriosclerosis begins with injury to the endothelium of an artery, which may be caused by irritation from high blood glucose, infection, tobacco use, excessive blood lipids, and other factors. Artery walls that are constantly stressed by blood flowing at high pressure are also more likely to be injured—which means that hypertension can promote arteriosclerosis, as well as result from it.

Recall that tissue injury causes inflammation. As inflammation spreads into the artery wall, it weakens and scars it, leaving it stiff (sclerotic). As a result, compliance is reduced. Moreover, circulating triglycerides and cholesterol can seep between the damaged lining cells and become trapped within the artery wall, where they are frequently joined by leukocytes, calcium, and cellular debris. Eventually, this buildup, called plaque, can narrow arteries enough to impair blood flow. The term for this condition, atherosclerosis (athero- = "porridge") describes the mealy deposits (Figure 7.2.5).

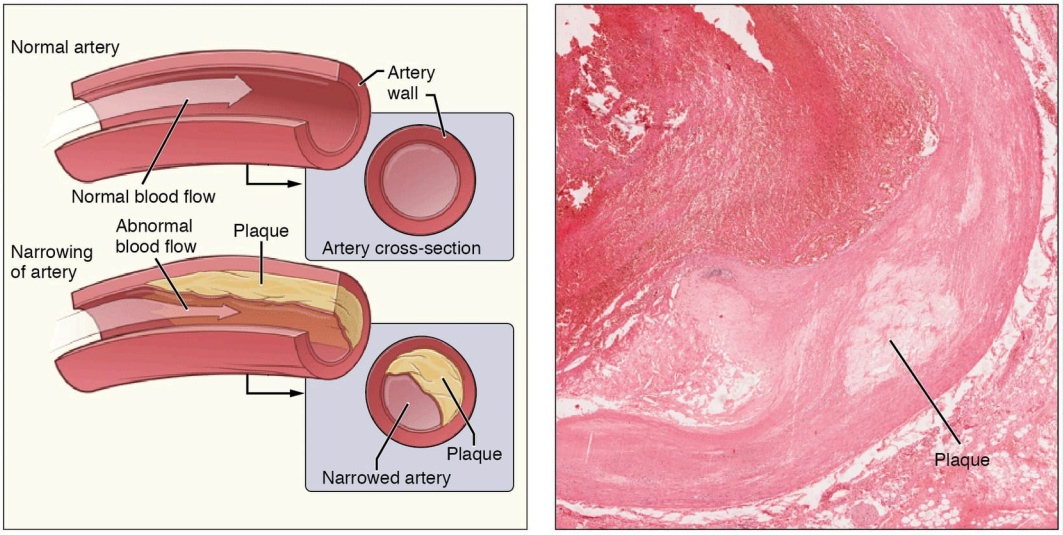


Figure 7.2.5: (a) Atherosclerosis can result from plaques formed by the buildup of fatty, calcified deposits in an artery. (b) Plaques can also take other forms, as shown in this micrograph of a coronary artery that has a buildup of connective tissue within the artery wall. LM × 40. (Micrograph provided by the Regents of University of Michigan Medical School © 2012). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.2.5.

Sometimes a plaque can rupture, causing microscopic tears in the artery wall that allow blood to leak into the tissue on the other side. When this happens, platelets rush to the site to clot the blood. This clot can further obstruct the artery and—if it occurs in a coronary or cerebral artery—cause a sudden heart attack or stroke. Alternatively, plaque can break off and travel through the bloodstream as an embolus until it blocks a more distant, smaller artery.

Even without total blockage, vessel narrowing leads to ischemia—reduced blood flow—to the tissue region "downstream" of the narrowed vessel. Ischemia in turn leads to hypoxia—decreased supply of oxygen to the tissues. Hypoxia involving cardiac muscle or brain tissue can lead to cell death and severe impairment of brain or heart function.

A major risk factor for both arteriosclerosis and atherosclerosis is advanced age, as the conditions tend to progress over time. Arteriosclerosis is normally defined as the more generalized loss of compliance, "hardening of the arteries," whereas atherosclerosis is a more specific term for the build-up of plaque in the walls of the vessel and is a specific type of arteriosclerosis. There is also a distinct genetic component, and pre-existing hypertension and/or diabetes also greatly increase the risk. However, obesity, poor nutrition, lack of physical activity, and tobacco use all are major risk factors.

Treatment includes lifestyle changes, such as weight loss, smoking cessation, regular exercise, and adoption of a diet low in sodium and saturated fats. Medications to reduce cholesterol and blood pressure may be prescribed. For blocked coronary arteries, surgery is warranted. In angioplasty, a catheter is inserted into the vessel at the point of narrowing, and a second catheter with a balloon-like tip is inflated to widen the opening. To prevent subsequent collapse of the vessel, a small mesh tube called a stent is often inserted. In an endarterectomy, plaque is surgically removed from the walls of a vessel. This operation is typically performed on the carotid arteries of the neck, which are a prime source of oxygenated blood for the brain. In a coronary bypass procedure, a non-vital superficial vessel from another part of the body (often the great saphenous vein) or a synthetic vessel is inserted to create a path around the blocked area of a coronary artery.

Venous System

The pumping action of the heart propels the blood into the arteries, from an area of higher pressure toward an area of lower pressure. If blood is to flow from the veins back into the heart, the pressure in the veins must be greater than the pressure in

the atria of the heart. Two factors help maintain this pressure gradient between the veins and the heart. First, the pressure in the atria during diastole is very low, often approaching zero when the atria are relaxed (atrial diastole). Second, two physiologic "pumps" increase pressure in the venous system. The use of the term "pump" implies a physical device that speeds flow. These physiological pumps are less obvious.

Skeletal Muscle Pump

In many body regions, the pressure within the veins can be increased by the contraction of the surrounding skeletal muscle. This mechanism, known as the skeletal muscle pump (Figure 7.2.6), helps the lower-pressure veins counteract the force of gravity, increasing pressure to move blood back to the heart. As leg muscles contract, for example during walking or running, they exert pressure on nearby veins with their numerous one-way valves. This increased pressure causes blood to flow upward, opening valves superior to the contracting muscles so blood flows through. Simultaneously, valves inferior to the contracting muscles close; thus, blood should not seep back downward toward the feet. Military recruits are trained to flex their legs slightly while standing at attention for prolonged periods. Failure to do so may allow blood to pool in the lower limbs rather than returning to the heart. Consequently, the brain will not receive enough oxygenated blood, and the individual may lose consciousness.

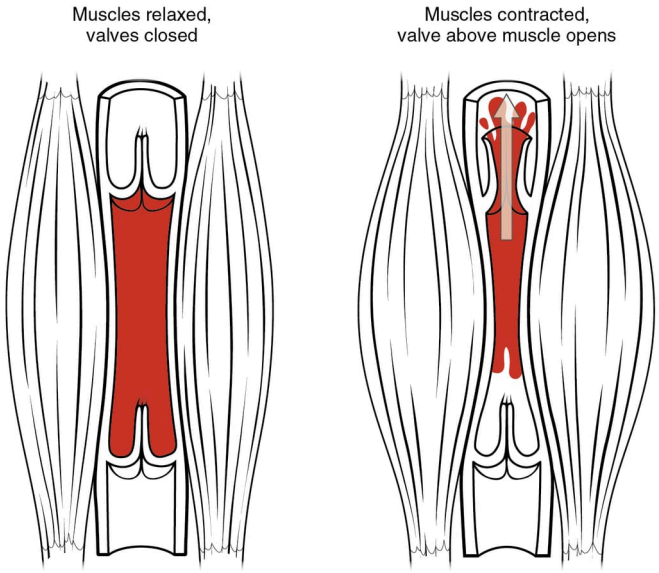


Figure 7.2.6: The contraction of skeletal muscles surrounding a vein compresses the blood and increases the pressure in that area. This action forces blood closer to the heart where venous pressure is lower. Note the importance of the one-way valves to assure that blood flows only in the proper direction. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.2.6.

Respiratory Pump

The respiratory pump aids blood flow through the veins of the thorax and abdomen. During inhalation, the volume of the thorax increases, largely through the contraction of the diaphragm, which moves downward and compresses the abdominal cavity. The elevation of the chest caused by the contraction of the external intercostal muscles also contributes to the increased volume of the thorax. The volume increase causes air pressure within the thorax to decrease, allowing us to inhale. Additionally, as air pressure within the thorax drops, blood pressure in the thoracic veins also decreases, falling below the pressure in the abdominal veins. This causes blood to flow along its pressure gradient from veins outside the thorax, where pressure is higher, into the thoracic region, where pressure is now lower. This in turn promotes the return of blood from the thoracic veins to the atria. During exhalation, when air pressure increases within the thoracic cavity, pressure in the thoracic veins increases, speeding blood flow into the heart while valves in the veins prevent blood from flowing backward from the thoracic and abdominal veins.

Pressure Relationships in the Venous System

Although vessel diameter increases from the smaller venules to the larger veins and eventually to the venae cavae (singular = vena cava), the total cross-sectional area actually decreases (see Figure 7.2.2a and Figure 7.2.2b). The individual veins are larger in diameter than the venules, but their total number is much lower, so their total cross-sectional area is also lower.

Also notice that, as blood moves from venules to veins, the average blood pressure drops, but the blood velocity actually increases. This pressure gradient drives blood back toward the heart. Again, the presence of one-way valves and the skeletal muscle and respiratory pumps contribute to this increased flow. Since approximately 64 percent of the total blood volume resides in systemic veins, any action that increases the flow of blood through the veins will increase venous return to the heart. Maintaining vascular tone within the veins prevents the veins from merely distending, dampening the flow of blood, and as you will see, vasoconstriction actually enhances the flow.

The Role of Venoconstriction in Resistance, Blood Pressure, and Flow

As previously discussed, vasoconstriction of an artery or arteriole decreases the radius, increasing resistance and pressure, but decreasing flow. Venoconstriction, on the other hand, has a very different outcome. The walls of veins are thin but irregular; thus, when the smooth muscle in those walls constricts, the lumen becomes more rounded. The more rounded the lumen, the less surface area the blood encounters, and the less resistance the vessel offers. Vasoconstriction increases pressure within a vein as it does in an artery, but in veins, the increased pressure increases flow. Recall that the pressure in the atria, into which the venous blood will flow, is very low, approaching zero for at least part of the relaxation phase of the cardiac cycle. Thus, venoconstriction increases the return of blood to the heart. Another way of stating this is that venoconstriction increases the preload or stretch of the cardiac muscle and increases contraction.

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7.3: Capillary Exchange

Mechanisms of Capillary Exchange

The primary purpose of the cardiovascular system is to circulate gases, nutrients, wastes, and other substances to and from the cells of the body. Small molecules, such as gases, lipids, and lipid-soluble molecules, can diffuse directly through the membranes of the endothelial cells of the capillary wall. Glucose, amino acids, and ions—including sodium, potassium, calcium, and chloride—use transporters to move through specific channels in the membrane by facilitated diffusion. Glucose, ions, and larger molecules may also leave the blood through intercellular clefts. Larger molecules can pass through the pores of fenestrated capillaries, and even large plasma proteins can pass through the great gaps in the sinusoids. Some large proteins in blood plasma can move into and out of the endothelial cells packaged within vesicles by endocytosis and exocytosis. Water moves by osmosis.

Bulk Flow

The mass movement of fluids into and out of capillary beds requires a transport mechanism far more efficient than mere diffusion. This movement, often referred to as bulk flow, involves two pressure-driven mechanisms: Volumes of fluid move from an area of higher pressure in a capillary bed to an area of lower pressure in the tissues via filtration. In contrast, the movement of fluid from an area of higher pressure in the tissues into an area of lower pressure in the capillaries is reabsorption. Two types of pressure interact to drive each of these movements: hydrostatic pressure and osmotic pressure.

Hydrostatic Pressure

The primary force driving fluid transport between the capillaries and tissues is hydrostatic pressure, which can be defined as the pressure of any fluid enclosed in a space. Blood hydrostatic pressure is the force exerted by the blood confined within blood vessels or heart chambers. Even more specifically, the pressure exerted by blood against the wall of a capillary is called capillary hydrostatic pressure (CHP), and is the same as capillary blood pressure. CHP is the force that drives fluid out of capillaries and into the tissues.

As fluid exits a capillary and moves into tissues, the hydrostatic pressure in the interstitial fluid correspondingly rises. This opposing hydrostatic pressure is called the interstitial fluid hydrostatic pressure (IFHP). Generally, the CHP originating from the arterial pathways is considerably higher than the IFHP, because lymphatic vessels are continually absorbing excess fluid from the tissues. Thus, fluid generally moves out of the capillary and into the interstitial fluid. This process is called filtration.

Osmotic Pressure

The net pressure that drives reabsorption—the movement of fluid from the interstitial fluid back into the capillaries—is called osmotic pressure (sometimes referred to as oncotic pressure). Whereas hydrostatic pressure forces fluid out of the capillary, osmotic pressure draws fluid back in. Osmotic pressure is determined by osmotic concentration gradients, that is, the difference in the solute-to-water concentrations in the blood and tissue fluid. A region higher in solute concentration (and lower in water concentration) draws water across a semipermeable membrane from a region higher in water concentration (and lower in solute concentration).

As we discuss osmotic pressure in blood and tissue fluid, it is important to recognize that the formed elements of blood do not contribute to osmotic concentration gradients. Rather, it is the plasma proteins that play the key role. Solutes also move across the capillary wall according to their concentration gradient, but overall, the concentrations should be similar and not have a significant impact on osmosis. Because of their large size and chemical structure, plasma proteins are not truly solutes, that is, they do not dissolve but are dispersed or suspended in their fluid medium, forming a colloid rather than a solution.

The pressure created by the concentration of colloidal proteins in the blood is called the blood colloidal osmotic pressure (BCOP). Its effect on capillary exchange accounts for the reabsorption of water. The plasma proteins suspended in blood cannot move across the semipermeable capillary cell membrane, and so they remain in the plasma. As a result, blood has a higher colloidal concentration and lower water concentration than tissue fluid. It therefore attracts water. We can also say that the BCOP is higher than the interstitial fluid colloidal osmotic pressure (IFCOP), which is always very low because interstitial fluid contains few

proteins. Thus, water is drawn from the tissue fluid back into the capillary, carrying dissolved molecules with it. This difference in colloidal osmotic pressure accounts for reabsorption.

Interaction of Hydrostatic and Osmotic Pressures

The normal unit used to express pressures within the cardiovascular system is millimeters of mercury (mm Hg). When blood leaving an arteriole first enters a capillary bed, the CHP is quite high—about 35 mm Hg. Gradually, this initial CHP declines as the blood moves through the capillary so that by the time the blood has reached the venous end, the CHP has dropped to approximately 18 mm Hg. In comparison, the plasma proteins remain suspended in the blood, so the BCOP remains fairly constant at about 25 mm Hg throughout the length of the capillary and considerably above the osmotic pressure in the interstitial fluid.

The net filtration pressure (NFP) represents the interaction of the hydrostatic and osmotic pressures, driving fluid out of the capillary. It is equal to the difference between the CHP and the BCOP. Since filtration is, by definition, the movement of fluid out of the capillary, when reabsorption is occurring, the NFP is a negative number.

NFP changes at different points in a capillary bed (Figure 7.3.1). Close to the arterial end of the capillary, it is approximately 10 mm Hg, because the CHP of 35 mm Hg minus the BCOP of 25 mm Hg equals 10 mm Hg. Recall that the hydrostatic and osmotic pressures of the interstitial fluid are essentially negligible. Thus, the NFP of 10 mm Hg drives a net movement of fluid out of the capillary at the arterial end. At approximately the middle of the capillary, the CHP is about the same as the BCOP of 25 mm Hg, so the NFP drops to zero. At this point, there is no net change of volume: Fluid moves out of the capillary at the same rate as it moves into the capillary. Near the venous end of the capillary, the CHP has dwindled to about 18 mm Hg due to loss of fluid. Because the BCOP remains steady at 25 mm Hg, water is drawn into the capillary, that is, reabsorption occurs. Another way of expressing this is to say that at the venous end of the capillary, there is an NFP of –7 mm Hg.

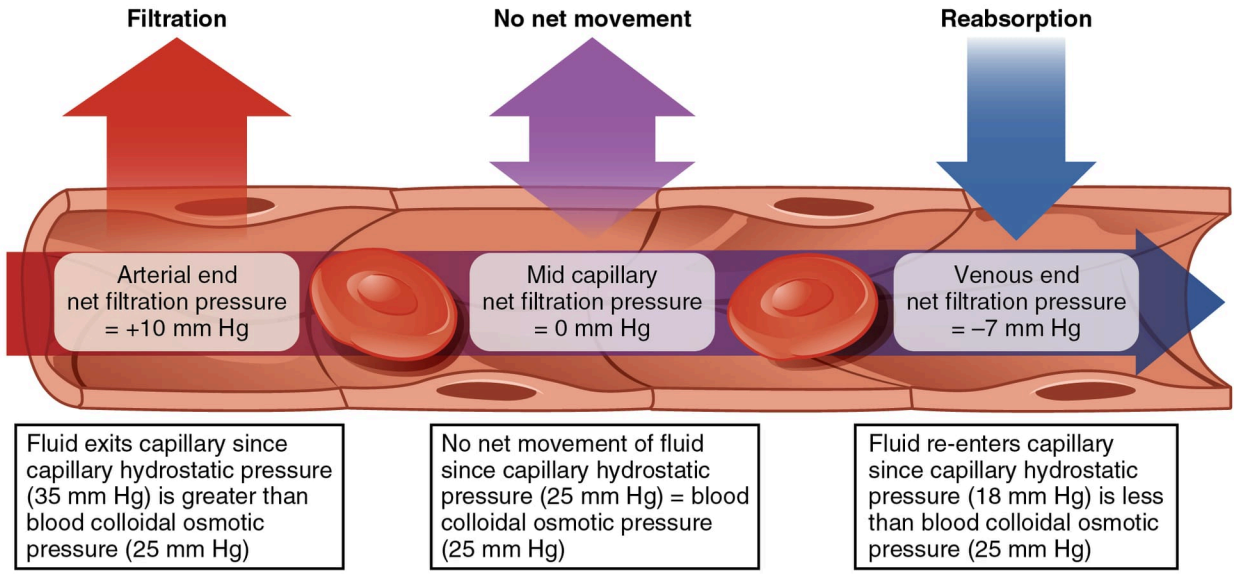


Figure 7.3.1: Net filtration occurs near the arterial end of the capillary since capillary hydrostatic pressure (CHP) is greater than blood colloidal osmotic pressure (BCOP). There is no net movement of fluid near the midpoint since CHP = BCOP. Net reabsorption occurs near the venous end since BCOP is greater than CHP. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.3.1.

The Role of Lymphatic Capillaries

Since overall CHP is higher than BCOP, it is inevitable that more net fluid will exit the capillary through filtration at the arterial end than enters through reabsorption at the venous end. Considering all capillaries over the course of a day, this can be quite a substantial amount of fluid: Approximately 24 liters per day are filtered, whereas 20.4 liters are reabsorbed. This excess fluid is picked up by capillaries of the lymphatic system. These extremely thin-walled vessels have copious numbers of valves that ensure unidirectional flow through ever-larger lymphatic vessels that eventually drain into the subclavian veins in the neck. An important function of the lymphatic system is to return the fluid (lymph) to the blood. Lymph may be thought of as recycled blood plasma. (Seek additional content for more detail on the lymphatic system.)

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7.4: Homeostatic Regulation of the Vascular System

Regulation of Blood Pressure and Blood Flow

In order to maintain homeostasis in the cardiovascular system and provide adequate blood to the tissues, blood flow must be redirected continually to the tissues as they become more active. In a very real sense, the cardiovascular system engages in resource allocation, because there is not enough blood flow to distribute blood equally to all tissues simultaneously. For example, when an individual is exercising, more blood will be directed to skeletal muscles, the heart, and the lungs. Following a meal, more blood is directed to the digestive system. Only the brain receives a more or less constant supply of blood whether you are active, resting, thinking, or engaged in any other activity.

Table 7.4.1 provides the distribution of systemic blood at rest and during exercise. Although most of the data appears logical, the values for the distribution of blood to the integument may seem surprising. During exercise, the body distributes more blood to the body surface where it can dissipate the excess heat generated by increased activity into the environment.

Table 7.4.1: Systemic Blood Flow During Rest, Mild Exercise, and Maximal Exercise in a Healthy Young Individual

Organ	Resting (mL/min)	Mild exercise (mL/min)	Maximal exercise (mL/min)
Skeletal muscle	1200	4500	12,500
Heart	250	350	750
Brain	750	750	750
Integument	500	1500	1900
Kidney	1100	900	600
Gastrointestinal	1400	1100	600
Others (i.e., liver, spleen)	600	400	400
Total	5800	9500	17,500

Three homeostatic mechanisms ensure adequate blood flow, blood pressure, distribution, and ultimately perfusion: neural, endocrine, and autoregulatory mechanisms. They are summarized in Table ({7{.}4{.}\eatSpaces{2} \endSpaces}\).

Table 7.4.2: Mechanisms Regulating Blood Flow and Blood Pressure

Regulatory Mechanism	Trigger	Response	Outcome
Autoregulation	Changes in local blood flow detected by tissue receptors	Vasodilators relax precapillary sphincters.	Increased blood flow; homeostasis restored.
Autoregulation	Changes in local blood flow detected by tissue receptors	Vasoconstrictors constrict precapillary sphincters.	Decreased blood flow; homeostasis restored.
Neural Regulation	Changes in blood pressure	Sympathetic stimulation increases cardiac output and blood flow; parasympathetic stimulation decreases cardiac output and blood flow.	Homeostasis restored.
Neural Regulation	Changes in blood chemistry	Cardiac and vasomotor centers regulate vessel diameter through vasoconstriction and vasodilation.	Homeostasis restored.

Regulatory Mechanism	Trigger	Response	Outcome
Endocrine Regulation	Kidneys	Erythropoietin and the renin-angiotensin-aldosterone system regulate blood volume and pressure.	Homeostasis restored.

Neural Regulation

The nervous system plays a critical role in the regulation of vascular homeostasis. The primary regulatory sites include the cardiovascular centers in the brain that control both cardiac and vascular functions. In addition, more generalized neural responses from the limbic system and the autonomic nervous system are factors.

The Cardiovascular Centers in the Brain

Neurological regulation of blood pressure and flow depends on the cardiovascular centers located in the medulla oblongata. This cluster of neurons responds to changes in blood pressure as well as blood concentrations of oxygen, carbon dioxide, and hydrogen ions. The cardiovascular center contains three distinct paired components:

- The cardioaccelerator centers stimulate cardiac function by regulating heart rate and stroke volume via sympathetic stimulation from the cardiac accelerator nerve.
- The cardioinhibitor centers slow cardiac function by decreasing heart rate and stroke volume via parasympathetic stimulation from the vagus nerve.
- The vasomotor centers control vessel tone or contraction of the smooth muscle in the tunica media. Changes in diameter affect peripheral resistance, pressure, and flow, which affect cardiac output. The majority of these neurons act via the release of the neurotransmitter norepinephrine from sympathetic neurons.

Although each center functions independently, they are not anatomically distinct.

There is also a small population of neurons that control vasodilation in the vessels of the brain and skeletal muscles by relaxing the smooth muscle fibers in the vessel tunics. Many of these are cholinergic neurons, that is, they release acetylcholine, which in turn stimulates the vessels' endothelial cells to release nitric oxide (NO), which causes vasodilation. Others release norepinephrine that binds to β_2 receptors. A few neurons release NO directly as a neurotransmitter.

Recall that mild stimulation of the skeletal muscles maintains muscle tone. A similar phenomenon occurs with vascular tone in vessels. As noted earlier, arterioles are normally partially constricted: With maximal stimulation, their radius may be reduced to one-half of the resting state. Full dilation of most arterioles requires that this sympathetic stimulation be suppressed. When it is, an arteriole can expand by as much as 150 percent. Such a significant increase can dramatically affect resistance, pressure, and flow.

Baroreceptor Reflexes

Baroreceptors are specialized stretch receptors located within thin areas of blood vessels and heart chambers that respond to the degree of stretch caused by the presence of blood. They send impulses to the cardiovascular center to regulate blood pressure. Vascular baroreceptors are found primarily in sinuses (small cavities) within the aorta and carotid arteries: The aortic sinuses are found in the walls of the ascending aorta just superior to the aortic valve, whereas the carotid sinuses are in the base of the internal carotid arteries. There are also low-pressure baroreceptors located in the walls of the venae cavae and right atrium.

When blood pressure increases, the baroreceptors are stretched more tightly and initiate action potentials at a higher rate. At lower blood pressures, the degree of stretch is lower and the rate of firing is slower. When the cardiovascular center in the medulla oblongata receives this input, it triggers a reflex that maintains homeostasis (Table 7.4.3):

- When blood pressure rises too high, the baroreceptors fire at a higher rate and trigger parasympathetic stimulation of the heart. As a result, cardiac output falls. Sympathetic stimulation of the peripheral arterioles will also decrease, resulting in vasodilation. Combined, these activities cause blood pressure to fall.
- When blood pressure drops too low, the rate of baroreceptor firing decreases. This will trigger an increase in sympathetic stimulation of the heart, causing cardiac output to increase. It will also trigger sympathetic stimulation of the peripheral

vessels, resulting in vasoconstriction. Combined, these activities cause blood pressure to rise.

Table 7.4.3: Baroreceptor Reflex Response to Changes in Blood Pressure

Initial Change	Baroreceptor Response	Control Center Response	Cardiovascular Response	Effect on Blood Pressure	Outcome
Increased blood pressure	Increased baroreceptor firing	Increased cardiac inhibitor center activity	Decreased cardiac output (heart rate and stroke volume)	Blood pressure drops	Homeostasis restored
Increased blood pressure	Increased baroreceptor firing	Decreased cardiac accelerator center activity	Decreased cardiac output (heart rate and stroke volume)	Blood pressure drops	Homeostasis restored
Increased blood pressure	Increased baroreceptor firing	Decreased vasomotor center activity	Increased vasodilation	Blood pressure drops	Homeostasis restored
Decreased blood pressure	Decreased baroreceptor firing	Decreased cardiac inhibitor center activity	Increased cardiac output (heart rate and stroke volume)	Blood pressure increases	Homeostasis restored
Decreased blood pressure	Decreased baroreceptor firing	Increased cardiac accelerator center activity	Increased cardiac output (heart rate and stroke volume)	Blood pressure increases	Homeostasis restored
Decreased blood pressure	Decreased baroreceptor firing	Increased vasomotor center activity	Increased vasoconstriction	Blood pressure increases	Homeostasis restored

The baroreceptors in the venae cavae and right atrium monitor blood pressure as the blood returns to the heart from the systemic circulation. Normally, blood flow into the aorta is the same as blood flow back into the right atrium. If blood is returning to the right atrium more rapidly than it is being ejected from the left ventricle, the atrial receptors will stimulate the cardiovascular centers to increase sympathetic firing and increase cardiac output until homeostasis is achieved. The opposite is also true. This mechanism is referred to as the atrial reflex.

Chemoreceptor Reflexes

In addition to the baroreceptors are chemoreceptors that monitor levels of oxygen, carbon dioxide, and hydrogen ions (pH), and thereby contribute to vascular homeostasis. Chemoreceptors monitoring the blood are located in close proximity to the baroreceptors in the aortic and carotid sinuses. They signal the cardiovascular center as well as the respiratory centers in the medulla oblongata.

Since tissues consume oxygen and produce carbon dioxide and acids as waste products, when the body is more active, oxygen levels fall and carbon dioxide levels rise as cells undergo cellular respiration to meet the energy needs of activities. This causes more hydrogen ions to be produced, causing the blood pH to drop. When the body is resting, oxygen levels are higher, carbon dioxide levels are lower, more hydrogen is bound, and pH rises. (Seek additional content for more detail about pH.)

The chemoreceptors respond to increasing carbon dioxide and hydrogen ion levels (falling pH) by stimulating the cardioaccelerator and vasomotor centers, increasing cardiac output and constricting peripheral vessels. The cardioinhibitor centers are suppressed. With falling carbon dioxide and hydrogen ion levels (increasing pH), the cardioinhibitor centers are stimulated, and the cardioaccelerator and vasomotor centers are suppressed, decreasing cardiac output and causing peripheral vasodilation. In order to maintain adequate supplies of oxygen to the cells and remove waste products such as carbon dioxide, it is essential that the respiratory system respond to changing metabolic demands. In turn, the cardiovascular system will transport these gases to the lungs for exchange, again in accordance with metabolic demands. This interrelationship of cardiovascular and respiratory control cannot be overemphasized.

Other neural mechanisms can also have a significant impact on cardiovascular function. These include the limbic system that links physiological responses to psychological stimuli, as well as generalized sympathetic and parasympathetic stimulation.

Endocrine Regulation

Endocrine control over the cardiovascular system involves the catecholamines, epinephrine and norepinephrine, as well as several hormones that interact with the kidneys in the regulation of blood volume.

Epinephrine and Norepinephrine

The catecholamines epinephrine and norepinephrine are released by the adrenal medulla, and enhance and extend the body's sympathetic or "fight-or-flight" response. They increase heart rate and force of contraction, while temporarily constricting blood vessels to organs not essential for flight-or-fight responses and redirecting blood flow to the liver, muscles, and heart.

Antidiuretic Hormone

Antidiuretic hormone (ADH), also known as vasopressin, is secreted by the cells in the hypothalamus and transported via the hypothalamic-hypophyseal tracts to the posterior pituitary where it is stored until released upon nervous stimulation. The primary trigger prompting the hypothalamus to release ADH is increasing osmolarity of tissue fluid, usually in response to significant loss of blood volume. ADH signals its target cells in the kidneys to reabsorb more water, thus preventing the loss of additional fluid in the urine. This will increase overall fluid levels and help restore blood volume and pressure. In addition, ADH constricts peripheral vessels.

Renin-Angiotensin-Aldosterone Mechanism

The renin-angiotensin-aldosterone mechanism has a major effect upon the cardiovascular system (Table 7.4.4). Renin is an enzyme, although because of its importance in the renin-angiotensin-aldosterone pathway, some sources identify it as a hormone. Specialized cells in the kidneys , called juxtaglomerular (JG) cells, respond to decreased blood pressure by secreting renin into the blood. Renin converts the plasma protein angiotensinogen, which is produced by the liver, into its active form—angiotensin I. Angiotensin I circulates in the blood and is then converted into angiotensin II in the lungs. This reaction is catalyzed by the enzyme angiotensin-converting enzyme (ACE).

Angiotensin II is a powerful vasoconstrictor, greatly increasing blood pressure. It also stimulates the release of ADH and aldosterone, a hormone produced by the adrenal cortex. Aldosterone increases the reabsorption of sodium into the blood by the kidneys. Since water follows sodium, this increases the reabsorption of water. This in turn increases blood volume, raising blood pressure. Angiotensin II also stimulates the thirst center in the hypothalamus, so an individual will likely consume more fluids, again increasing blood volume and pressure.

Table 7.4.4: Short-Term and Long-Term Responses to Decreased Blood Pressure and/or Blood Volume

Time Frame	Initial Stimulus	Physiological Response	Intermediate Effect	Outcome	Final Result
Short Term	Decreased blood pressure and/or blood volume	Sympathetic activation; adrenal glands release epinephrine and norepinephrine	Increased cardiac output and increased peripheral vasoconstriction	Increased blood pressure	Homeostasis restored
Long Term	Decreased blood pressure and/or blood volume	Kidneys produce renin	Angiotensin II activated	Antidiuretic hormone released and aldosterone secreted	Homeostasis restored
Long Term	Decreased blood pressure and/or blood volume	Kidneys produce renin	Angiotensin II activated	Thirst stimulated	Increased blood volume → Homeostasis restored
Long Term	Decreased blood pressure and/or blood volume	Kidneys produce erythropoietin	Increased red blood cell formation	Increased blood volume	Homeostasis restored

Erythropoietin

Erythropoietin (EPO) is released by the kidneys when blood flow and/or oxygen levels decrease. EPO stimulates the production of erythrocytes within the bone marrow. Erythrocytes are the major formed element of the blood and may contribute 40 percent or more to blood volume, a significant factor of viscosity, resistance, pressure, and flow. In addition, EPO is a vasoconstrictor. Overproduction of EPO or excessive intake of synthetic EPO, often to enhance athletic performance, will increase viscosity, resistance, and pressure, and decrease flow in addition to its contribution as a vasoconstrictor.

Atrial Natriuretic Hormone

Secreted by cells in the atria of the heart, atrial natriuretic hormone (ANH) (also known as atrial natriuretic peptide) is secreted when blood volume is high enough to cause extreme stretching of the cardiac cells. Cells in the ventricle produce a hormone with similar effects, called B-type natriuretic hormone. Natriuretic hormones are antagonists to angiotensin II. They promote loss of sodium and water from the kidneys, and suppress renin, aldosterone, and ADH production and release. All of these actions promote loss of fluid from the body, so blood volume and blood pressure drop.

Autoregulation of Perfusion

As the name would suggest, autoregulation mechanisms require neither specialized nervous stimulation nor endocrine control. Rather, these are local, self-regulatory mechanisms that allow each region of tissue to adjust its blood flow—and thus its perfusion. These local mechanisms include chemical signals and myogenic controls.

Chemical Signals Involved in Autoregulation

Chemical signals work at the level of the precapillary sphincters to trigger either constriction or relaxation. As you know, opening a precapillary sphincter allows blood to flow into that particular capillary, whereas constricting a precapillary sphincter temporarily shuts off blood flow to that region. The factors involved in regulating the precapillary sphincters include the following:

- Opening of the sphincter is triggered in response to decreased oxygen concentrations; increased carbon dioxide concentrations; increasing levels of lactic acid or other byproducts of cellular metabolism; increasing concentrations of potassium ions or hydrogen ions (falling pH); inflammatory chemicals such as histamines; and increased body temperature. These conditions in turn stimulate the release of NO, a powerful vasodilator, from endothelial cells (see Table 7.4.2).
- Contraction of the precapillary sphincter is triggered by the opposite levels of the regulators, which prompt the release of endothelins, powerful vasoconstricting peptides secreted by endothelial cells. Platelet secretions and certain prostaglandins may also trigger constriction.

Again, these factors alter tissue perfusion via their effects on the precapillary sphincter mechanism, which regulates blood flow to capillaries. Since the amount of blood is limited, not all capillaries can fill at once, so blood flow is allocated based upon the needs and metabolic state of the tissues as reflected in these parameters. Bear in mind, however, that dilation and constriction of the arterioles feeding the capillary beds is the primary control mechanism.

The Myogenic Response

The myogenic response is a reaction to the stretching of the smooth muscle in the walls of arterioles as changes in blood flow occur through the vessel. This may be viewed as a largely protective function against dramatic fluctuations in blood pressure and blood flow to maintain homeostasis. If perfusion of an organ is too low (ischemia), the tissue will experience low levels of oxygen (hypoxia). In contrast, excessive perfusion could damage the organ's smaller and more fragile vessels. The myogenic response is a localized process that serves to stabilize blood flow in the capillary network that follows that arteriole.

When blood flow is low, the vessel's smooth muscle will be only minimally stretched. In response, it relaxes, allowing the vessel to dilate and thereby increase the movement of blood into the tissue. When blood flow is too high, the smooth muscle will contract in response to the increased stretch, prompting vasoconstriction that reduces blood flow.

Table 7.4.5 summarizes the effects of nervous, endocrine, and local controls on arterioles.

Table 7.4.5: Factors Affecting Vasoconstriction and Vasodilation

Control Mechanism	Factor	Effects on Vasoconstriction	Effects on Vasodilation
Neural	Sympathetic stimulation	Arterioles within integument, abdominal viscera, and mucosa membrane; skeletal muscle (at high levels); varied effects in veins and venules	Arterioles within heart; skeletal muscles at low to moderate levels
Neural	Parasympathetic stimulation	No known innervation for most vessels	Arterioles in external genitalia; no known innervation for most other arterioles or veins
Endocrine	Epinephrine	Similar to sympathetic stimulation for extended fight-or-flight responses; at high levels, binds to specialized	

Effect of Exercise on Vascular Homeostasis

The heart is a muscle and, like any muscle, it responds dramatically to exercise. For a healthy young adult, cardiac output (heart rate × stroke volume) increases in the nonathlete from approximately 5.0 liters (5.25 quarts) per minute to a maximum of about 20 liters (21 quarts) per minute. Accompanying this will be an increase in blood pressure from about 120/80 to 185/75. However, well-trained aerobic athletes can increase these values substantially. For these individuals, cardiac output soars from approximately 5.3 liters (5.57 quarts) per minute resting to more than 30 liters (31.5 quarts) per minute during maximal exercise. Along with this increase in cardiac output, blood pressure increases from 120/80 at rest to 200/90 at maximum values.

In addition to improved cardiac function, exercise increases the size and mass of the heart. The average weight of the heart for the nonathlete is about 300 g, whereas in an athlete it will increase to 500 g. This increase in size generally makes the heart stronger and more efficient at pumping blood, increasing both stroke volume and cardiac output.

Tissue perfusion also increases as the body transitions from a resting state to light exercise and eventually to heavy exercise. These changes result in selective vasodilation in the skeletal muscles, heart, lungs, liver, and integument. Simultaneously, vasoconstriction occurs in the vessels leading to the kidneys and most of the digestive and reproductive organs. The flow of blood to the brain remains largely unchanged whether at rest or exercising, since the vessels in the brain largely do not respond to regulatory stimuli, in most cases, because they lack the appropriate receptors.

As vasodilation occurs in selected vessels, resistance drops and more blood rushes into the organs they supply. This blood eventually returns to the venous system. Venous return is further enhanced by both the skeletal muscle and respiratory pumps. As blood returns to the heart more quickly, preload rises and the Frank-Starling principle tells us that contraction of the cardiac muscle in the atria and ventricles will be more forceful. Eventually, even the best-trained athletes will fatigue and must undergo a period of rest following exercise. Cardiac output and distribution of blood then return to normal.

Regular exercise promotes cardiovascular health in a variety of ways. Because an athlete's heart is larger than a nonathlete's, stroke volume increases, so the athletic heart can deliver the same amount of blood as the nonathletic heart but with a lower heart rate. This increased efficiency allows the athlete to exercise for longer periods of time before muscles fatigue and places less stress on the heart. Exercise also lowers overall cholesterol levels by removing from the circulation a complex form of cholesterol, triglycerides, and proteins known as low-density lipoproteins (LDLs), which are widely associated with increased risk of cardiovascular disease. Although there is no way to remove deposits of plaque from the walls of arteries other than specialized surgery, exercise does promote the health of vessels by decreasing the rate of plaque formation and reducing blood pressure, so the heart does not have to generate as much force to overcome resistance.

Generally as little as 30 minutes of noncontinuous exercise over the course of each day has beneficial effects and has been shown to lower the rate of heart attack by nearly 50 percent. While it is always advisable to follow a healthy diet, stop smoking, and lose weight, studies have clearly shown that fit, overweight people may actually be healthier overall than sedentary slender people. Thus, the benefits of moderate exercise are undeniable.

Clinical Considerations in Vascular Homeostasis

Any disorder that affects blood volume, vascular tone, or any other aspect of vascular functioning is likely to affect vascular homeostasis as well. That includes hypertension, hemorrhage, and shock.

Hypertension and Hypotension

New guidelines in 2017 from the American College of Cardiology and American Heart Association list normal blood pressure (BP) as less than 120/80 mm Hg and elevated BP as systolic P between 120–129 and diastolic P less than 80 mm Hg. Chronically elevated blood pressure is known clinically as hypertension. The new guidelines list hypertension that should be treated as 130/80 mm Hg. Tens of millions of Americans currently suffer from hypertension. Unfortunately, hypertension is typically a silent disorder; therefore, hypertensive patients may fail to recognize the seriousness of their condition and fail to follow their treatment plan. The result is often a heart attack or stroke. Hypertension may also lead to an aneurysm (ballooning of a blood vessel caused by a weakening of the wall), peripheral arterial disease (obstruction of vessels in peripheral regions of the body), chronic kidney disease, or heart failure.

Listen to this CDC podcast to learn about hypertension, often described as a “silent killer.” What steps can you take to reduce your risk of a heart attack or stroke?

Hemorrhage

Minor blood loss is managed by hemostasis and repair. Hemorrhage is a loss of blood that cannot be controlled by hemostatic mechanisms. Initially, the body responds to hemorrhage by initiating mechanisms aimed at increasing blood pressure and maintaining blood flow. Ultimately, however, blood volume will need to be restored, either through physiological processes or through medical intervention.

In response to blood loss, stimuli from the baroreceptors trigger the cardiovascular centers to stimulate sympathetic responses to increase cardiac output and vasoconstriction. This typically prompts the heart rate to increase to about 180–200 contractions per minute, restoring cardiac output to normal levels. Vasoconstriction of the arterioles increases vascular resistance, whereas constriction of the veins increases venous return to the heart. Both of these steps will help increase blood pressure. Sympathetic stimulation also triggers the release of epinephrine and norepinephrine, which enhance both cardiac output and vasoconstriction. If blood loss were less than 20 percent of total blood volume, these responses together would usually return blood pressure to normal and redirect the remaining blood to the tissues.

Additional endocrine involvement is necessary, however, to restore the lost blood volume. The angiotensin-renin-aldosterone mechanism stimulates the thirst center in the hypothalamus, which increases fluid consumption to help restore the lost blood. More importantly, it increases renal reabsorption of sodium and water, reducing water loss in urine output. The kidneys also increase the production of EPO, stimulating the formation of erythrocytes that not only deliver oxygen to the tissues but also increase overall blood volume. Table 7.4.6 summarizes the responses to loss of blood volume.

Table 7.4.6: Neural and Endocrine Responses to Decreased Blood Pressure and Blood Volume

Initial Stimulus	Control Mechanism	Intermediate Response	Physiological Effect	Outcome
Decreased blood pressure and blood volume	Endocrine mechanism	Release of antidiuretic hormone (ADH), angiotensin II, aldosterone, and erythropoietin (EPO)	Increased blood volume	Homeostasis restored
Decreased blood pressure and blood volume	Neural mechanism	Baroreceptors and chemoreceptors stimulated	Increased cardiac output; peripheral vasoconstriction; increased blood pressure; decreased venous reserve	Homeostasis restored

Initial Stimulus	Control Mechanism	Intermediate Response	Physiological Effect	Outcome
Decreased blood pressure and blood volume	Neural mechanism	Cardiovascular centers stimulated	Increased cardiac output; peripheral vasoconstriction; increased blood pressure; decreased venous reserve	Homeostasis restored
Decreased blood pressure and blood volume	Neural mechanism	General sympathetic activation and release of norepinephrine and epinephrine	Increased cardiac output; peripheral vasoconstriction; increased blood pressure; decreased venous reserve	Homeostasis restored

Circulatory Shock

The loss of too much blood may lead to circulatory shock, a life-threatening condition in which the circulatory system is unable to maintain blood flow to adequately supply sufficient oxygen and other nutrients to the tissues to maintain cellular metabolism. It should not be confused with emotional or psychological shock. Typically, the patient in circulatory shock will demonstrate an increased heart rate but decreased blood pressure, but there are cases in which blood pressure will remain normal. Urine output will fall dramatically, and the patient may appear confused or lose consciousness. Urine output less than 1 mL/kg body weight/hour is cause for concern. Unfortunately, shock is an example of a positive-feedback loop that, if uncorrected, may lead to the death of the patient.

There are several recognized forms of shock:

- Hypovolemic shock in adults is typically caused by hemorrhage, although in children it may be caused by fluid losses related to severe vomiting or diarrhea. Other causes for hypovolemic shock include extensive burns, exposure to some toxins, and excessive urine loss related to diabetes insipidus or ketoacidosis. Typically, patients present with a rapid, almost tachycardic heart rate; a weak pulse often described as “thready;” cool, clammy skin, particularly in the extremities, due to restricted peripheral blood flow; rapid, shallow breathing; hypothermia; thirst; and dry mouth. Treatments generally involve providing intravenous fluids to restore the patient to normal function and various drugs such as dopamine, epinephrine, and norepinephrine to raise blood pressure.
- Cardiogenic shock results from the inability of the heart to maintain cardiac output. Most often, it results from a myocardial infarction (heart attack), but it may also be caused by arrhythmias, valve disorders, cardiomyopathies, cardiac failure, or simply insufficient flow of blood through the cardiac vessels. Treatment involves repairing the damage to the heart or its vessels to resolve the underlying cause, rather than treating cardiogenic shock directly.
- Vascular shock occurs when arterioles lose their normal muscular tone and dilate dramatically. It may arise from a variety of causes, and treatments almost always involve fluid replacement and medications, called inotropic or pressor agents, which restore tone to the muscles of the vessels. In addition, eliminating or at least alleviating the underlying cause of the condition is required. This might include antibiotics and antihistamines, or select steroids, which may aid in the repair of nerve damage. A common cause is sepsis (or septicemia), also called “blood poisoning,” which is a widespread bacterial infection that results in an organismal-level inflammatory response known as septic shock. Neurogenic shock is a form of vascular shock that occurs with cranial or spinal injuries that damage the cardiovascular centers in the medulla oblongata or the nervous fibers originating from this region. Anaphylactic shock is a severe allergic response that causes the widespread release of histamines, triggering vasodilation throughout the body.
- Obstructive shock, as the name would suggest, occurs when a significant portion of the vascular system is blocked. It is not always recognized as a distinct condition and may be grouped with cardiogenic shock, including pulmonary embolism and cardiac tamponade. Treatments depend upon the underlying cause and, in addition to administering fluids intravenously, often include the administration of anticoagulants, removal of fluid from the pericardial cavity, or air from the thoracic cavity, and surgery as required. The most common cause is a pulmonary embolism, a clot that lodges in the pulmonary vessels and interrupts blood flow. Other causes include stenosis of the aortic valve; cardiac tamponade, in which excess fluid in the pericardial cavity interferes with the ability of the heart to fully relax and fill with blood (resulting in decreased preload); and a pneumothorax, in which an excessive amount of air is present in the thoracic cavity, outside of the lungs, which interferes with venous return, pulmonary function, and delivery of oxygen to the tissues.

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7.5: Circulatory Pathways

Major Blood Vessels and Circulatory Routes

Virtually every cell, tissue, organ, and system in the body is impacted by the circulatory system. This includes the generalized and more specialized functions of transport of materials, capillary exchange, maintaining health by transporting white blood cells and various immunoglobulins (antibodies), hemostasis, regulation of body temperature, and helping to maintain acid-base balance. In addition to these shared functions, many systems enjoy a unique relationship with the circulatory system. Figure 7.5.1 summarizes these relationships.

System	Role of Circulatory System
Digestive	Absorbs nutrients and water; delivers nutrients (except most lipids) to liver for processing by hepatic portal vein; provides nutrients essential for hematopoiesis and building hemoglobin
Endocrine	Delivers hormones: atrial natriuretic hormone (peptide) secreted by the heart atrial cells to help regulate blood volumes and pressures; epinephrine, ANH, angiotensin II, ADH, and thyroxine to help regulate blood pressure; estrogen to promote vascular health in women and men
Integumentary	Carries clotting factors, platelets, and white blood cells for hemostasis, fighting infection, and repairing damage; regulates temperature by controlling blood flow to the surface, where heat can be dissipated; provides some coloration of integument; acts as a blood reservoir
Lymphatic	Transports various white blood cells, including those produced by lymphatic tissue, and immunoglobulins (antibodies) throughout the body to maintain health; carries excess tissue fluid not able to be reabsorbed by the vascular capillaries back to the lymphatic system for processing
Muscular	Provides nutrients and oxygen for contraction; removes lactic acid and distributes heat generated by contraction; muscular pumps aid in venous return; exercise contributes to cardiovascular health and helps to prevent atherosclerosis
Nervous	Produces cerebrospinal fluid (CSF) within choroid plexuses; contributes to blood–brain barrier; cardiac and vasomotor centers regulate cardiac output and blood flow through vessels via autonomic system
Reproductive	Aids in erection of genitalia in both sexes during sexual arousal; transports gonadotropic hormones that regulate reproductive functions
Respiratory	Provides blood for critical exchange of gases to carry oxygen needed for metabolic reactions and carbon dioxide generated as byproducts of these processes
Skeletal	Provides calcium, phosphate, and other minerals critical for bone matrix; transports hormones regulating buildup and absorption of matrix including growth hormone (somatotropin), thyroid hormone, calcitonins, and parathyroid hormone; erythropoietin stimulates myeloid cell hematopoiesis; some level of protection for select vessels by bony structures
Urinary	Delivers 20% of resting circulation to kidneys for filtering, reabsorption of useful products, and secretion of excesses; regulates blood volume and pressure by regulating fluid loss in the form of urine and by releasing the enzyme renin that is essential in the renin-angiotensin-aldosterone mechanism

Figure 7.5.1: Interaction of the Circulatory System with Other Body Systems. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.1.

As you learn about the vessels of the systemic and pulmonary circuits, notice that many arteries and veins share the same names, parallel one another throughout the body, and are very similar on the right and left sides of the body. These pairs of vessels will be traced through only one side of the body. Where differences occur in branching patterns or when vessels are singular, this will be indicated. For example, you will find a pair of femoral arteries and a pair of femoral veins, with one vessel on each side of the body. In contrast, some vessels closer to the midline of the body, such as the aorta, are unique. Moreover, some superficial veins, such as the great saphenous vein in the femoral region, have no arterial counterpart. Another phenomenon that can make the study of vessels challenging is that names of vessels can change with location. Like a street that changes name as it passes through an intersection, an artery or vein can change names as it passes an anatomical landmark. For example, the left subclavian artery becomes the axillary artery as it passes through the body wall and into the axillary region, and then becomes the brachial artery as it flows from the axillary region into the upper arm (or brachium). You will also find examples of anastomoses where two blood vessels that previously branched reconnect. Anastomoses are especially common in veins, where they help maintain blood flow even when one vessel is blocked or narrowed, although there are some important ones in the arteries supplying the brain.

As you read about circular pathways, notice that there is an occasional, very large artery referred to as a trunk, a term indicating that the vessel gives rise to several smaller arteries. For example, the celiac trunk gives rise to the left gastric, common hepatic, and splenic arteries.

As you study this section, imagine you are on a “Voyage of Discovery” similar to Lewis and Clark’s expedition in 1804–1806, which followed rivers and streams through unfamiliar territory, seeking a water route from the Atlantic to the Pacific Ocean. You might envision being inside a miniature boat, exploring the various branches of the circulatory system. This simple approach has proven effective for many students in mastering these major circulatory patterns. Another approach that works well for many students is to create simple line drawings similar to the ones provided, labeling each of the major vessels. It is beyond the scope of this text to name every vessel in the body. However, we will attempt to discuss the major pathways for blood and acquaint you with the

major named arteries and veins in the body. Also, please keep in mind that individual variations in circulation patterns are not uncommon.

Pulmonary Circulation

Recall that blood returning from the systemic circuit enters the right atrium via the superior and inferior venae cavae and the coronary sinus, which drains the blood supply of the heart muscle. These vessels will be described more fully later in this section. This blood is relatively low in oxygen and relatively high in carbon dioxide, since much of the oxygen has been extracted for use by the tissues and the waste gas carbon dioxide was picked up to be transported to the lungs for elimination. From the right atrium, blood moves into the right ventricle, which pumps it to the lungs for gas exchange. This system of vessels is referred to as the pulmonary circuit.

The single vessel exiting the right ventricle is the pulmonary trunk. At the base of the pulmonary trunk is the pulmonary semilunar valve, which prevents backflow of blood into the right ventricle during ventricular diastole. As the pulmonary trunk reaches the superior surface of the heart, it curves posteriorly and rapidly bifurcates (divides) into two branches, a left and a right pulmonary artery. To prevent confusion between these vessels, it is important to refer to the vessel exiting the heart as the pulmonary trunk, rather than also calling it a pulmonary artery. The pulmonary arteries in turn branch many times within the lung, forming a series of smaller arteries and arterioles that eventually lead to the pulmonary capillaries. The pulmonary capillaries surround lung structures known as alveoli that are the sites of oxygen and carbon dioxide exchange.

Once gas exchange is completed, oxygenated blood flows from the pulmonary capillaries into a series of pulmonary venules that eventually lead to a series of larger pulmonary veins. Four pulmonary veins, two on the left and two on the right, return blood to the left atrium. At this point, the pulmonary circuit is complete. Table 7.5.1 defines the major arteries and veins of the pulmonary circuit discussed in the text.

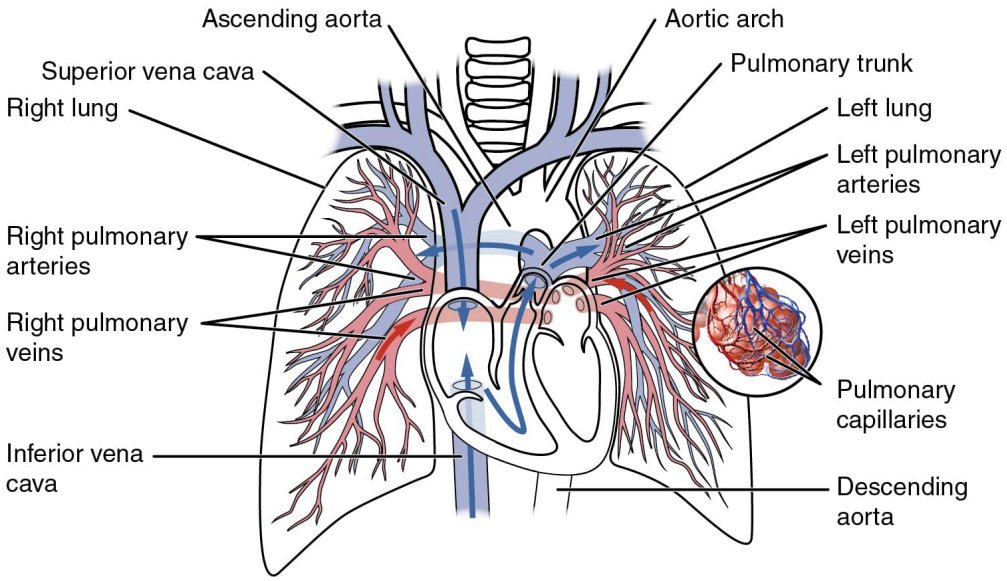


Figure 7.5.2: Blood exiting from the right ventricle flows into the pulmonary trunk, which bifurcates into the two pulmonary arteries. These vessels branch to supply blood to the pulmonary capillaries, where gas exchange occurs within the lung alveoli. Blood returns via the pulmonary veins to the left atrium. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.2.

Table 7.5.1: Pulmonary Arteries and Veins

Vessel	Description
Pulmonary trunk	Single large vessel exiting the right ventricle that divides to form the right and left pulmonary arteries
Pulmonary arteries	Left and right vessels that form from the pulmonary trunk and lead to smaller arterioles and eventually to the pulmonary capillaries
Pulmonary veins	Two sets of paired vessels—one pair on each side—that are formed from the small venules, leading away from the pulmonary capillaries to flow into the left atrium

Overview of Systemic Arteries

Blood relatively high in oxygen concentration is returned from the pulmonary circuit to the left atrium via the four pulmonary veins. From the left atrium, blood moves into the left ventricle, which pumps blood into the aorta. The aorta and its branches—the systemic arteries—send blood to virtually every organ of the body (Figure 7.5.3).

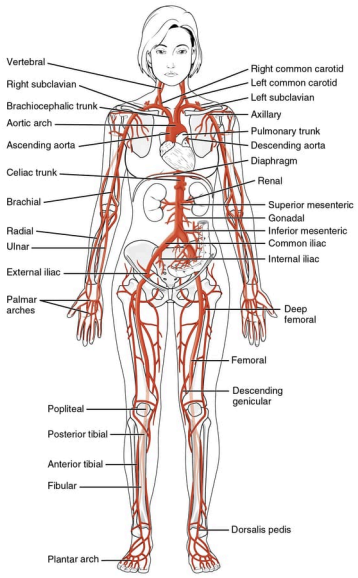


Figure 7.5.3: The major systemic arteries shown here deliver oxygenated blood throughout the body. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.3.

The Aorta

The aorta is the largest artery in the body. It arises from the left ventricle and eventually descends to the abdominal region, where it bifurcates at the level of the fourth lumbar vertebra into the two common iliac arteries. The aorta consists of the ascending aorta, the aortic arch, and the descending aorta, which passes through the diaphragm and a landmark that divides into the superior thoracic and inferior abdominal components. Arteries originating from the aorta ultimately distribute blood to virtually all tissues of the body. At the base of the aorta is the aortic semilunar valve that prevents backflow of blood into the left ventricle while the heart is relaxing. After exiting the heart, the ascending aorta moves in a superior direction for approximately 5 cm and ends at the sternal angle. Following this ascent, it reverses direction, forming a graceful arc to the left, called the aortic arch. The aortic arch descends toward the inferior portions of the body and ends at the level of the intervertebral disk between the fourth and fifth thoracic vertebrae. Beyond this point, the descending aorta continues close to the bodies of the vertebrae and passes through an opening in the diaphragm known as the aortic hiatus. Superior to the diaphragm, the aorta is called the thoracic aorta, and inferior to the diaphragm, it is called the abdominal aorta. The abdominal aorta terminates when it bifurcates into the two common iliac arteries at the level of the fourth lumbar vertebra.

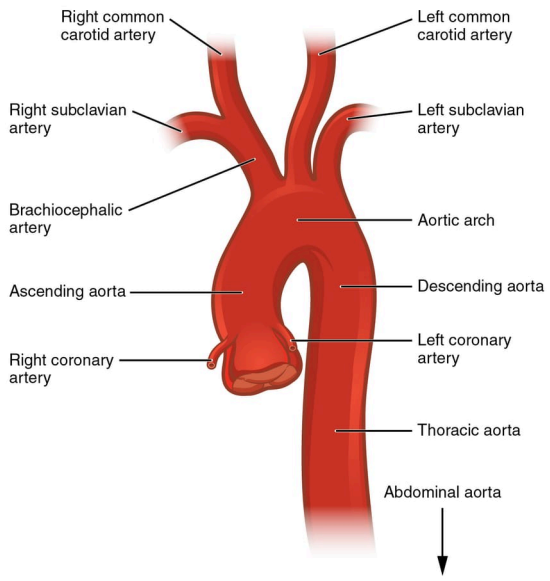


Figure 7.5.4: The aorta has distinct regions, including the ascending aorta, aortic arch, and the descending aorta, which includes the thoracic and abdominal regions. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.4.

Table 7.5.2: Components of the Aorta

Vessel	Description
Aorta	Largest artery in the body, originating from the left ventricle and descending to the abdominal region, where it bifurcates into the common iliac arteries at the level of the fourth lumbar vertebra; arteries originating from the aorta distribute blood to virtually all tissues of the body
Ascending aorta	Initial portion of the aorta, rising superiorly from the left ventricle for a distance of approximately 5 cm
Aortic arch	Graceful arc to the left that connects the ascending aorta to the descending aorta; ends at the intervertebral disk between the fourth and fifth thoracic vertebrae
Descending aorta	Portion of the aorta that continues inferiorly past the end of the aortic arch; subdivided into the thoracic aorta and the abdominal aorta
Thoracic aorta	Portion of the descending aorta superior to the aortic hiatus
Abdominal aorta	Portion of the aorta inferior to the aortic hiatus and superior to the common iliac arteries

Coronary Circulation

The first vessels that branch from the ascending aorta are the paired coronary arteries, which arise from two of the three sinuses in the ascending aorta just superior to the aortic semilunar valve. These sinuses contain the aortic baroreceptors and chemoreceptors critical to maintain cardiac function. The left coronary artery arises from the left posterior aortic sinus. The right coronary artery arises from the anterior aortic sinus. Normally, the right posterior aortic sinus does not give rise to a vessel.

The coronary arteries encircle the heart, forming a ring-like structure that divides into the next level of branches that supplies blood to the heart tissues. (Seek additional content for more detail on cardiac circulation.)

Aortic Arch Branches

There are three major branches of the aortic arch: the brachiocephalic artery, the left common carotid artery, and the left subclavian (literally “under the clavicle”) artery. As you would expect based upon proximity to the heart, each of these vessels is classified as an elastic artery.

The brachiocephalic artery is located only on the right side of the body; there is no corresponding artery on the left. The brachiocephalic artery branches into the right subclavian artery and the right common carotid artery. The left subclavian and left common carotid arteries arise independently from the aortic arch but otherwise follow a similar pattern and distribution to the corresponding arteries on the right side.

Each subclavian artery supplies blood to the arms, chest, shoulders, back, and central nervous system. It then gives rise to three major branches: the internal thoracic artery, the vertebral artery, and the thyrocervical artery. The internal thoracic artery, or mammary artery, supplies blood to the thymus, the pericardium of the heart, and the anterior chest wall. The vertebral artery passes through the vertebral foramen in the cervical vertebrae and then through the foramen magnum into the cranial cavity to supply blood to the brain and spinal cord. The paired vertebral arteries join together to form the large basilar artery at the base of the medulla oblongata. This is an example of an anastomosis. The subclavian artery also gives rise to the thyrocervical artery that provides blood to the thyroid, the cervical region of the neck, and the upper back and shoulder.

The common carotid artery divides into internal and external carotid arteries. The right common carotid artery arises from the brachiocephalic artery and the left common carotid artery arises directly from the aortic arch. The external carotid artery supplies blood to numerous structures within the face, lower jaw, neck, esophagus, and larynx. These branches include the lingual, facial, occipital, maxillary, and superficial temporal arteries. The internal carotid artery initially forms an expansion known as the carotid sinus, containing the carotid baroreceptors and chemoreceptors. Like their counterparts in the aortic sinuses, the information provided by these receptors is critical to maintaining cardiovascular homeostasis.

The internal carotid arteries along with the vertebral arteries are the two primary suppliers of blood to the human brain. Given the central role and vital importance of the brain to life, it is critical that blood supply to this organ remains uninterrupted. Recall that blood flow to the brain is remarkably constant, with approximately 20 percent of blood flow directed to this organ at any given time. When blood flow is interrupted, even for just a few seconds, a transient ischemic attack (TIA), or mini-stroke, may occur, resulting in loss of consciousness or temporary loss of neurological function. In some cases, the damage may be permanent. Loss of blood flow for longer periods, typically between 3 and 4 minutes, will likely produce irreversible brain damage or a stroke, also called a cerebrovascular accident (CVA). The locations of the arteries in the brain not only provide blood flow to the brain tissue but also prevent interruption in the flow of blood. Both the carotid and vertebral arteries branch once they enter the cranial cavity, and some of these branches form a structure known as the arterial circle (or circle of Willis), an anastomosis that is remarkably like a traffic circle that sends off branches (in this case, arterial branches to the brain). As a rule, branches to the anterior portion of the cerebrum are normally fed by the internal carotid arteries; the remainder of the brain receives blood flow from branches associated with the vertebral arteries.

The internal carotid artery continues through the carotid canal of the temporal bone and enters the base of the brain through the carotid foramen where it gives rise to several branches. One of these branches is the anterior cerebral artery that supplies blood to the frontal lobe of the cerebrum. Another branch, the middle cerebral artery, supplies blood to the temporal and parietal lobes, which are the most common sites of CVAs. The ophthalmic artery, the third major branch, provides blood to the eyes.

The right and left anterior cerebral arteries join together to form an anastomosis called the anterior communicating artery. The initial segments of the anterior cerebral arteries and the anterior communicating artery form the anterior portion of the arterial circle. The posterior portion of the arterial circle is formed by a left and a right posterior communicating artery that branches from the posterior cerebral artery, which arises from the basilar artery. It provides blood to the posterior portion of the cerebrum and brain stem. The basilar artery is an anastomosis that begins at the junction of the two vertebral arteries and sends branches to the cerebellum and brain stem. It flows into the posterior cerebral arteries. Table 7.5.3 summarizes the aortic arch branches, including the major branches supplying the brain.

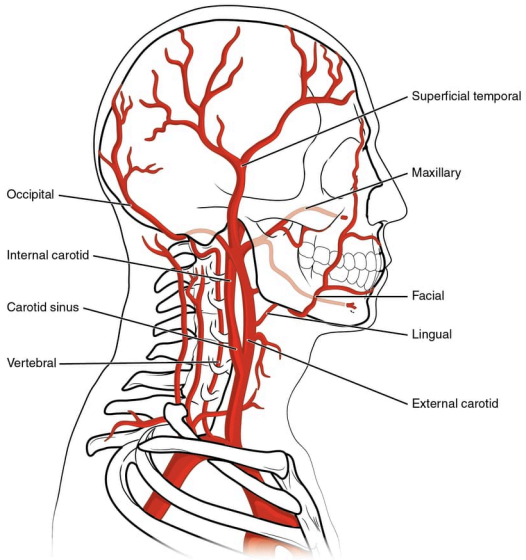


Figure 7.5.5: The common carotid artery gives rise to the external and internal carotid arteries. The external carotid artery remains superficial and gives rise to many arteries of the head. The internal carotid artery first forms the carotid sinus and then reaches the brain via the carotid canal and carotid foramen, emerging into the cranium via the foramen lacerum. The vertebral artery branches from the subclavian artery and passes through the transverse foramen in the cervical vertebrae, entering the base of the skull at the vertebral foramen. The subclavian artery continues toward the arm as the axillary artery. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.5.

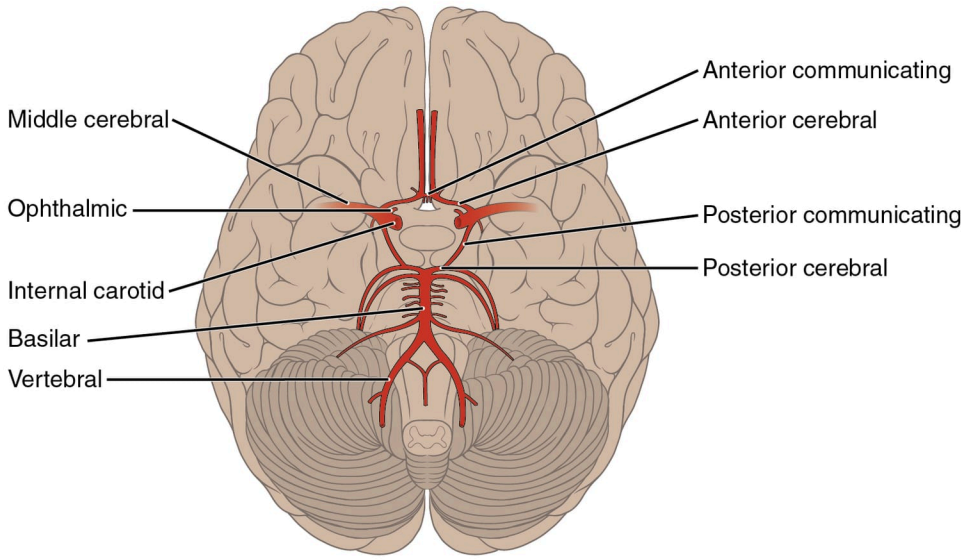


Figure 7.5.6: This inferior view shows the network of arteries serving the brain. The structure is referred to as the arterial circle or circle of Willis. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.6.

Table 7.5.3: Aortic Arch Branches and Brain Circulation

Vessel	Description
Brachiocephalic artery	Single vessel located on the right side of the body; the first vessel branching from the aortic arch; gives rise to the right subclavian artery and the right common carotid artery; supplies blood to the head, neck, upper limb, and wall of the thoracic region
Subclavian artery	The right subclavian artery arises from the brachiocephalic artery while the left subclavian artery arises from the aortic arch; gives rise to the internal thoracic, vertebral, and thyrocervical arteries; supplies blood to the arms, chest, shoulders, back, and central nervous system
Internal thoracic artery	Also called the mammary artery; arises from the subclavian artery; supplies blood to the thymus, pericardium of the heart, and anterior chest wall

Vessel	Description
Vertebral artery	Arises from the subclavian artery and passes through the vertebral foramen through the foramen magnum to the brain; joins with the internal carotid artery to form the arterial circle; supplies blood to the brain and spinal cord
Thyrocervical artery	Arises from the subclavian artery; supplies blood to the thyroid, the cervical region, the upper back, and shoulder
Common carotid artery	The right common carotid artery arises from the brachiocephalic artery and the left common carotid artery arises from the aortic arch; each gives rise to the external and internal carotid arteries; supplies the respective sides of the head and neck
External carotid artery	Arises from the common carotid artery; supplies blood to numerous structures within the face, lower jaw, neck, esophagus, and larynx
Internal carotid artery	Arises from the common carotid artery and begins with the carotid sinus; goes through the carotid canal of the temporal bone to the base of the brain; combines with the branches of the vertebral artery, forming the arterial circle; supplies blood to the brain
Arterial circle or circle of Willis	An anastomosis located at the base of the brain that ensures continual blood supply; formed from the branches of the internal carotid and vertebral arteries; supplies blood to the brain
Anterior cerebral artery	Arises from the internal carotid artery; supplies blood to the frontal lobe of the cerebrum
Middle cerebral artery	Another branch of the internal carotid artery; supplies blood to the temporal and parietal lobes of the cerebrum
Ophthalmic artery	Branch of the internal carotid artery; supplies blood to the eyes
Anterior communicating artery	An anastomosis of the right and left internal carotid arteries; supplies blood to the brain
Posterior communicating artery	Branches of the posterior cerebral artery that form part of the posterior portion of the arterial circle; supplies blood to the brain
Posterior cerebral artery	Branch of the basilar artery that forms a portion of the posterior segment of the arterial circle of Willis; supplies blood to the posterior portion of the cerebrum and brain stem
Basilar artery	Formed from the fusion of the two vertebral arteries; sends branches to the cerebellum, brain stem, and the posterior cerebral arteries; the main blood supply to the brain stem

Thoracic Aorta and Major Branches

The thoracic aorta begins at the level of vertebra T5 and continues through to the diaphragm at the level of T12, initially traveling within the mediastinum to the left of the vertebral column. As it passes through the thoracic region, the thoracic aorta gives rise to several branches, which are collectively referred to as visceral branches and parietal branches (Figure 7.5.7). Those branches that supply blood primarily to visceral organs are known as the visceral branches and include the bronchial arteries, pericardial arteries, esophageal arteries, and the mediastinal arteries, each named after the tissues it supplies. Each bronchial artery (typically two on the left and one on the right) supplies systemic blood to the lungs and visceral pleura, in addition to the blood pumped to the lungs for oxygenation via the pulmonary circuit. The bronchial arteries follow the same path as the respiratory branches, beginning with the bronchi and ending with the bronchioles. There is considerable, but not total, intermingling of the systemic and pulmonary blood at anastomoses in the smaller branches of the lungs. This may sound incongruous—that is, the mixing of systemic arterial blood high in oxygen with the pulmonary arterial blood lower in oxygen—but the systemic vessels also deliver nutrients to the lung tissue just as they do elsewhere in the body. The mixed blood drains into typical pulmonary veins, whereas the bronchial artery branches remain separate and drain into bronchial veins described later. Each pericardial artery supplies blood to the pericardium, the esophageal artery provides blood to the esophagus, and the mediastinal artery provides blood to the mediastinum. The remaining thoracic aorta branches are collectively referred to as parietal branches or somatic branches, and include the intercostal and superior phrenic arteries. Each intercostal artery provides blood to the muscles of the

thoracic cavity and vertebral column. The superior phrenic artery provides blood to the superior surface of the diaphragm. Table 7.5.4 lists the arteries of the thoracic region.

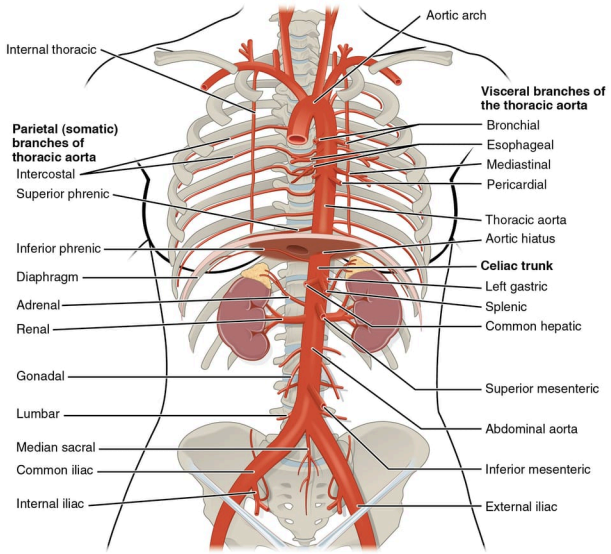


Figure 7.5.7: The thoracic aorta gives rise to the arteries of the visceral and parietal branches. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.7.

Table 7.5.4: Arteries of the Thoracic Region

Vessel	Description
Visceral branches	A group of arterial branches of the thoracic aorta; supplies blood to the viscera (i.e., organs) of the thorax
Bronchial artery	Systemic branch from the aorta that provides oxygenated blood to the lungs; this blood supply is in addition to the pulmonary circuit that brings blood for oxygenation
Pericardial artery	Branch of the thoracic aorta; supplies blood to the pericardium
Esophageal artery	Branch of the thoracic aorta; supplies blood to the esophagus
Mediastinal artery	Branch of the thoracic aorta; supplies blood to the mediastinum
Parietal branches	Also called somatic branches, a group of arterial branches of the thoracic aorta; include those that supply blood to the thoracic wall, vertebral column, and the superior surface of the diaphragm
Intercostal artery	Branch of the thoracic aorta; supplies blood to the muscles of the thoracic cavity and vertebral column
Superior phrenic artery	Branch of the thoracic aorta; supplies blood to the superior surface of the diaphragm

Abdominal Aorta and Major Branches

After crossing through the diaphragm at the aortic hiatus, the thoracic aorta is called the abdominal aorta. This vessel remains to the left of the vertebral column and is embedded in adipose tissue behind the peritoneal cavity. It formally ends at approximately the level of vertebra L4, where it bifurcates to form the common iliac arteries. Before this division, the abdominal aorta gives rise to several important branches. A single celiac trunk (artery) emerges and divides into the left gastric artery to supply blood to the stomach and esophagus, the splenic artery to supply blood to the spleen, and the common hepatic artery, which in turn gives rise to the hepatic artery proper to supply blood to the liver, the right gastric artery to supply blood to the stomach, the

cystic artery to supply blood to the gall bladder, and several branches, one to supply blood to the duodenum and another to supply blood to the pancreas. Two additional single vessels arise from the abdominal aorta. These are the superior and inferior mesenteric arteries. The superior mesenteric artery arises approximately 2.5 cm after the celiac trunk and branches into several major vessels that supply blood to the small intestine (duodenum, jejunum, and ileum), the pancreas, and a majority of the large intestine. The inferior mesenteric artery supplies blood to the distal segment of the large intestine, including the rectum. It arises approximately 5 cm superior to the common iliac arteries.

In addition to these single branches, the abdominal aorta gives rise to several significant paired arteries along the way. These include the inferior phrenic arteries, the adrenal arteries, the renal arteries, the gonadal arteries, and the lumbar arteries. Each inferior phrenic artery is a counterpart of a superior phrenic artery and supplies blood to the inferior surface of the diaphragm. The adrenal artery supplies blood to the adrenal (suprarenal) glands and arises near the superior mesenteric artery. Each renal artery branches approximately 2.5 cm inferior to the superior mesenteric arteries and supplies a kidney. The right renal artery is longer than the left since the aorta lies to the left of the vertebral column and the vessel must travel a greater distance to reach its target. Renal arteries branch repeatedly to supply blood to the kidneys. Each gonadal artery supplies blood to the gonads, or reproductive organs, and is also described as either an ovarian artery or a testicular artery (internal spermatic), depending upon the sex of the individual. An ovarian artery supplies blood to an ovary, uterine (Fallopian) tube, and the uterus, and is located within the suspensory ligament of the uterus. It is considerably shorter than a testicular artery, which ultimately travels outside the body cavity to the testes, forming one component of the spermatic cord. The gonadal arteries arise inferior to the renal arteries and are generally retroperitoneal. The ovarian artery continues to the uterus where it forms an anastomosis with the uterine artery that supplies blood to the uterus. Both the uterine arteries and vaginal arteries, which distribute blood to the vagina, are branches of the internal iliac artery. The four paired lumbar arteries are the counterparts of the intercostal arteries and supply blood to the lumbar region, the abdominal wall, and the spinal cord. In some instances, a fifth pair of lumbar arteries emerges from the median sacral artery.

The aorta divides at approximately the level of vertebra L4 into a left and a right common iliac artery but continues as a small vessel, the median sacral artery, into the sacrum. The common iliac arteries provide blood to the pelvic region and ultimately to the lower limbs. They split into external and internal iliac arteries approximately at the level of the lumbar-sacral articulation. Each internal iliac artery sends branches to the urinary bladder, the walls of the pelvis, the external genitalia, and the medial portion of the femoral region. In females, they also provide blood to the uterus and vagina. The much larger external iliac artery supplies blood to each of the lower limbs.

Table 7.5.5: Major Branches of the Thoracic and Abdominal Aorta and Their Areas of Supply

Aortic Region	Artery	Branch Type	Structures Supplied
Thoracic Aorta	Bronchial arteries	Unpaired	Air passages of the respiratory tract and lung tissue
Thoracic Aorta	Pericardial arteries	Unpaired	Pericardium
Thoracic Aorta	Esophageal arteries	Unpaired	Esophagus
Thoracic Aorta	Mediastinal arteries	Unpaired	Mediastinal structures
Thoracic Aorta	Intercostal arteries	Paired	Vertebrae, spinal cord, back muscles, body wall, and skin
Thoracic Aorta	Superior phrenic arteries	Paired	Vertebrae, spinal cord, back muscles, body wall, skin, and diaphragm
Abdominal Aorta (via Celiac Trunk)	Left gastric artery	Unpaired	Stomach and adjacent portion of the esophagus
Abdominal Aorta (via Celiac Trunk)	Splenic artery	Unpaired	Spleen, stomach, and pancreas
Abdominal Aorta (via Celiac Trunk)	Common hepatic artery	Unpaired	Liver, stomach, gallbladder, duodenum, and pancreas
Abdominal Aorta	Superior mesenteric artery	Unpaired	Pancreas, small intestine, appendix, and first two-thirds of the large intestine

Aortic Region	Artery	Branch Type	Structures Supplied
Abdominal Aorta	Inferior mesenteric artery	Unpaired	Last one-third of the large intestine
Abdominal Aorta	Inferior phrenic arteries	Paired	Diaphragm and inferior portion of the esophagus
Abdominal Aorta	Adrenal arteries	Paired	Adrenal glands

Table 7.5.6: Vessels of the Abdominal Aorta

Vessel	Description
Celiac trunk	Also called the celiac artery; a major branch of the abdominal aorta; gives rise to the left gastric artery, the splenic artery, and the common hepatic artery that forms the hepatic artery to the liver, the right gastric artery to the stomach, and the cystic artery to the gall bladder
Left gastric artery	Branch of the celiac trunk; supplies blood to the stomach
Splenic artery	Branch of the celiac trunk; supplies blood to the spleen
Common hepatic artery	Branch of the celiac trunk that forms the hepatic artery, the right gastric artery, and the cystic artery
Hepatic artery proper	Branch of the common hepatic artery; supplies systemic blood to the liver
Right gastric artery	Branch of the common hepatic artery; supplies blood to the stomach
Cystic artery	Branch of the common hepatic artery; supplies blood to the gall bladder
Superior mesenteric artery	Branch of the abdominal aorta; supplies blood to the small intestine (duodenum, jejunum, and ileum), the pancreas, and a majority of the large intestine
Inferior mesenteric artery	Branch of the abdominal aorta; supplies blood to the distal segment of the large intestine and rectum
Inferior phrenic arteries	Branches of the abdominal aorta; supply blood to the inferior surface of the diaphragm
Adrenal artery	Branch of the abdominal aorta; supplies blood to the adrenal (suprarenal) glands
Renal artery	Branch of the abdominal aorta; supplies each kidney
Gonadal artery	Branch of the abdominal aorta; supplies blood to the gonads or reproductive organs; also described as ovarian arteries or testicular arteries, depending upon the sex of the individual
Ovarian artery	Branch of the abdominal aorta; supplies blood to ovary, uterine (Fallopian) tube, and uterus
Testicular artery	Branch of the abdominal aorta; ultimately travels outside the body cavity to the testes and forms one component of the spermatic cord

Vessel	Description
Lumbar arteries	Branches of the abdominal aorta; supply blood to the lumbar region, the abdominal wall, and spinal cord
Common iliac artery	Branch of the aorta that leads to the internal and external iliac arteries
Median sacral artery	Continuation of the aorta into the sacrum
Internal iliac artery	Branch from the common iliac arteries; supplies blood to the urinary bladder, walls of the pelvis, external genitalia, and the medial portion of the femoral region; in females, also provides blood to the uterus and vagina
External iliac artery	Branch of the common iliac artery that leaves the body cavity and becomes a femoral artery; supplies blood to the lower limbs

Arteries Serving the Upper Limbs

As the subclavian artery exits the thorax into the axillary region, it is renamed the axillary artery. Although it does branch and supply blood to the region near the head of the humerus (via the humeral circumflex arteries), the majority of the vessel continues into the upper arm, or brachium, and becomes the brachial artery. The brachial artery supplies blood to much of the brachial region and divides at the elbow into several smaller branches, including the deep brachial arteries, which provide blood to the posterior surface of the arm, and the ulnar collateral arteries, which supply blood to the region of the elbow. As the brachial artery approaches the coronoid fossa, it bifurcates into the radial and ulnar arteries, which continue into the forearm, or antebrachium. The radial artery and ulnar artery parallel their namesake bones, giving off smaller branches until they reach the wrist, or carpal region. At this level, they fuse to form the superficial and deep palmar arches that supply blood to the hand, as well as the digital arteries that supply blood to the digits. Figure 7.5.8 shows the distribution of systemic arteries from the heart into the upper limb. Table 7.5.7 summarizes the arteries serving the upper limbs.

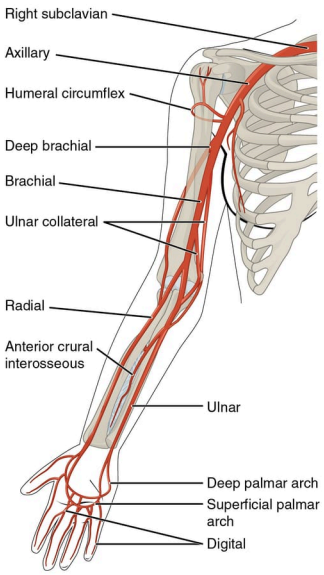


Figure 7.5.8: The right subclavian artery continues as the axillary and brachial arteries, which branch into the radial and ulnar arteries. These vessels form the superficial and deep palmar arches and supply the digits through the digital arteries. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.8.

Table 7.5.7: Major Arteries of the Head, Neck, and Upper Limb

Origin	Branch	Supplies
Left ventricle	Aortic arch	Distributes oxygenated blood to the head, neck, upper limbs, and thorax
Aortic arch	Brachiocephalic trunk	Gives rise to the right common carotid artery and right subclavian artery

Origin	Branch	Supplies
Aortic arch	Left common carotid artery	Head and neck
Aortic arch	Left subclavian artery	Left upper limb and associated branches
Right subclavian artery	Right vertebral artery	Spinal cord, cervical vertebrae; joins the left vertebral artery to form the basilar artery
Left subclavian artery	Left vertebral artery	Spinal cord, cervical vertebrae; joins the right vertebral artery to form the basilar artery
Right subclavian artery	Right thyrocervical trunk	Neck muscles and skin, thyroid gland, shoulder region, and upper back
Left subclavian artery	Left thyrocervical trunk	Neck muscles and skin, thyroid gland, shoulder region, and upper back
Right subclavian artery	Right internal thoracic artery	Chest wall, abdominal wall, mammary gland, and pericardium
Left subclavian artery	Left internal thoracic artery	Chest wall, abdominal wall, mammary gland, and pericardium
Right subclavian artery	Right axillary artery	Pectoral region and axilla
Left subclavian artery	Left axillary artery	Pectoral region and axilla
Right axillary artery	Right brachial artery	Arm and elbow
Left axillary artery	Left brachial artery	Arm and elbow
Right brachial artery	Right radial artery	Lateral (thumb-side) forearm and hand
Right brachial artery	Right ulnar artery	Medial forearm and hand
Left brachial artery	Left radial artery	Lateral (thumb-side) forearm and hand
Left brachial artery	Left ulnar artery	Medial forearm and hand
Radial and ulnar arteries	Digital arteries	Fingers

Table 7.5.8: Arteries Serving the Upper Limbs

Vessel	Description
Axillary artery	Continuation of the subclavian artery as it penetrates the body wall and enters the axillary region; supplies blood to the region near the head of the humerus (humeral circumflex arteries); the majority of the vessel continues into the brachium and becomes the brachial artery
Brachial artery	Continuation of the axillary artery in the brachium; supplies blood to much of the brachial region; gives off several smaller branches that provide blood to the posterior surface of the arm in the region of the elbow; bifurcates into the radial and ulnar arteries at the coronoid fossa
Radial artery	Formed at the bifurcation of the brachial artery; parallels the radius; gives off smaller branches until it reaches the carpal region where it fuses with the ulnar artery to form the superficial and deep palmar arches; supplies blood to the lower arm and carpal region
Ulnar artery	Formed at the bifurcation of the brachial artery; parallels the ulna; gives off smaller branches until it reaches the carpal region where it fuses with the radial artery to form the superficial and deep palmar arches; supplies blood to the lower arm and carpal region
Palmar arches (superficial and deep)	Formed from anastomosis of the radial and ulnar arteries; supply blood to the hand and digital arteries

Vessel	Description
Digital arteries	Formed from the superficial and deep palmar arches; supply blood to the digits

Arteries Serving the Lower Limbs

The external iliac artery exits the body cavity and enters the femoral region of the lower leg (Figure 7.5.9). As it passes through the body wall, it is renamed the femoral artery. It gives off several smaller branches as well as the lateral deep femoral artery that in turn gives rise to a lateral circumflex artery. These arteries supply blood to the deep muscles of the thigh as well as ventral and lateral regions of the integument. The femoral artery also gives rise to the genicular artery, which provides blood to the region of the knee. As the femoral artery passes posterior to the knee near the popliteal fossa, it is called the popliteal artery. The popliteal artery branches into the anterior and posterior tibial arteries.

The anterior tibial artery is located between the tibia and fibula, and supplies blood to the muscles and integument of the anterior tibial region. Upon reaching the tarsal region, it becomes the dorsalis pedis artery, which branches repeatedly and provides blood to the tarsal and dorsal regions of the foot. The posterior tibial artery provides blood to the muscles and integument on the posterior surface of the tibial region. The fibular or peroneal artery branches from the posterior tibial artery. It bifurcates and becomes the medial plantar artery and lateral plantar artery, providing blood to the plantar surfaces. There is an anastomosis with the dorsalis pedis artery, and the medial and lateral plantar arteries form two arches called the dorsal arch (also called the arcuate arch) and the plantar arch, which provide blood to the remainder of the foot and toes.

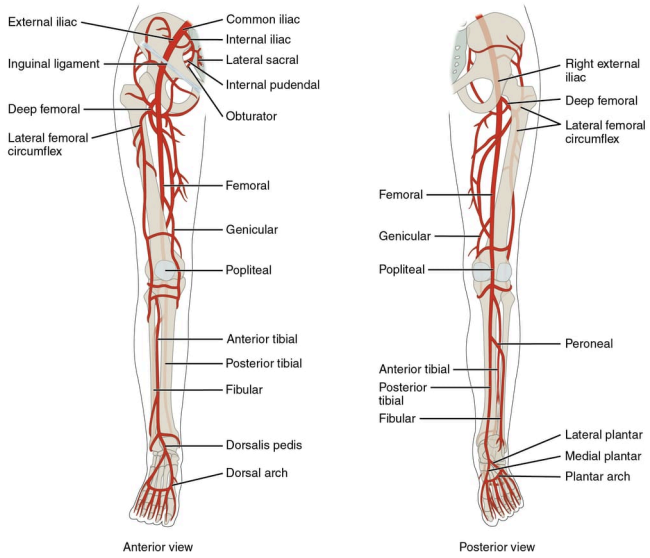


Figure 7.5.9: Major arteries serving the lower limb are shown in anterior and posterior views. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.9.

Table 7.5.9: Arteries Serving the Lower Limbs

Vessel	Description
Femoral artery	Continuation of the external iliac artery after it passes through the body cavity; divides into several smaller branches, the lateral deep femoral artery, and the genicular artery; becomes the popliteal artery as it passes posterior to the knee
Deep femoral artery	Branch of the femoral artery; gives rise to the lateral circumflex arteries
Lateral circumflex artery	Branch of the deep femoral artery; supplies blood to the deep muscles of the thigh and the ventral and lateral regions of the integument

Vessel	Description
Genicular artery	Branch of the femoral artery; supplies blood to the region of the knee
Popliteal artery	Continuation of the femoral artery posterior to the knee; branches into the anterior and posterior tibial arteries
Anterior tibial artery	Branches from the popliteal artery; supplies blood to the anterior tibial region; becomes the dorsalis pedis artery
Dorsalis pedis artery	Forms from the anterior tibial artery; branches repeatedly to supply blood to the tarsal and dorsal regions of the foot
Posterior tibial artery	Branches from the popliteal artery and gives rise to the fibular or peroneal artery; supplies blood to the posterior tibial region
Medial plantar artery	Arises from the bifurcation of the posterior tibial arteries; supplies blood to the medial plantar surfaces of the foot
Lateral plantar artery	Arises from the bifurcation of the posterior tibial arteries; supplies blood to the lateral plantar surfaces of the foot
Dorsal or arcuate arch	Formed from the anastomosis of the dorsalis pedis artery and the medial and plantar arteries; branches supply the distal portions of the foot and digits
Plantar arch	Formed from the anastomosis of the dorsalis pedis artery and the medial and plantar arteries; branches supply the distal portions of the foot and digits

Overview of Systemic Veins

Systemic veins return blood to the right atrium. Since the blood has already passed through the systemic capillaries, it will be relatively low in oxygen concentration. In many cases, there will be veins draining organs and regions of the body with the same name as the arteries that supplied these regions and the two often parallel one another. This is often described as a “complementary” pattern. However, there is a great deal more variability in the venous circulation than normally occurs in the arteries. For the sake of brevity and clarity, this text will discuss only the most commonly encountered patterns. However, keep this variation in mind when you move from the classroom to clinical practice.

In both the neck and limb regions, there are often both superficial and deeper levels of veins. The deeper veins generally correspond to the complementary arteries. The superficial veins do not normally have direct arterial counterparts, but in addition to returning blood, they also make contributions to the maintenance of body temperature. When the ambient temperature is warm, more blood is diverted to the superficial veins where heat can be more easily dissipated to the environment. In colder weather, there is more constriction of the superficial veins and blood is diverted deeper where the body can retain more of the heat.

The “Voyage of Discovery” analogy and stick drawings mentioned earlier remain valid techniques for the study of systemic veins, but veins present a more difficult challenge because there are numerous anastomoses and multiple branches. It is like following a river with many tributaries and channels, several of which interconnect. Tracing blood flow through arteries follows the current in the direction of blood flow, so that we move from the heart through the large arteries and into the smaller arteries to the capillaries. From the capillaries, we move into the smallest veins and follow the direction of blood flow into larger veins and back to the heart.

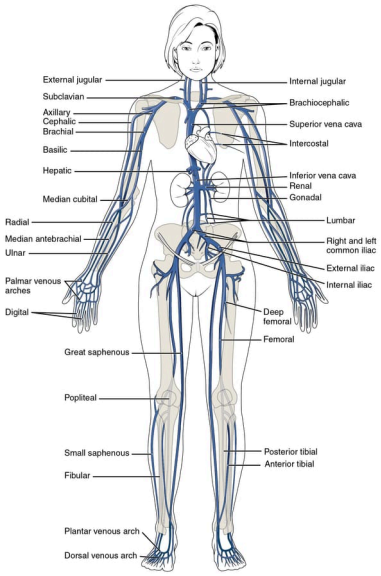


Figure 7.5.10: The major systemic veins of the body are shown here in an anterior view. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.10.

The right atrium receives all of the systemic venous return. Most of the blood flows into either the superior vena cava or inferior vena cava. If you draw an imaginary line at the level of the diaphragm, systemic venous circulation from above that line will generally flow into the superior vena cava; this includes blood from the head, neck, chest, shoulders, and upper limbs. The exception to this is that most venous blood flow from the coronary veins flows directly into the coronary sinus and from there directly into the right atrium. Beneath the diaphragm, systemic venous flow enters the inferior vena cava, that is, blood from the abdominal and pelvic regions and the lower limbs.

The Superior Vena Cava

The superior vena cava drains most of the body superior to the diaphragm (Figure 7.5.11). On both the left and right sides, the subclavian vein forms when the axillary vein passes through the body wall from the axillary region. It fuses with the external and internal jugular veins from the head and neck to form the brachiocephalic vein. Each vertebral vein also flows into the brachiocephalic vein close to this fusion. These veins arise from the base of the brain and the cervical region of the spinal cord, and flow largely through the intervertebral foramina in the cervical vertebrae. They are the counterparts of the vertebral arteries. Each internal thoracic vein, also known as an internal mammary vein, drains the anterior surface of the chest wall and flows into the brachiocephalic vein.

The remainder of the blood supply from the thorax drains into the azygos vein. Each intercostal vein drains muscles of the thoracic wall, each esophageal vein delivers blood from the inferior portions of the esophagus, each bronchial vein drains the systemic circulation from the lungs, and several smaller veins drain the mediastinal region. Bronchial veins carry approximately 13 percent of the blood that flows into the bronchial arteries; the remainder intermingles with the pulmonary circulation and returns to the heart via the pulmonary veins. These veins flow into the azygos vein, and with the smaller hemiazygos vein (hemi- = “half”) on the left of the vertebral column, drain blood from the thoracic region. The hemiazygos vein does not drain directly into the superior vena cava but enters the brachiocephalic vein via the superior intercostal vein.

The azygos vein passes through the diaphragm from the thoracic cavity on the right side of the vertebral column and begins in the lumbar region of the thoracic cavity. It flows into the superior vena cava at approximately the level of T2, making a significant contribution to the flow of blood. It combines with the two large left and right brachiocephalic veins to form the superior vena cava.

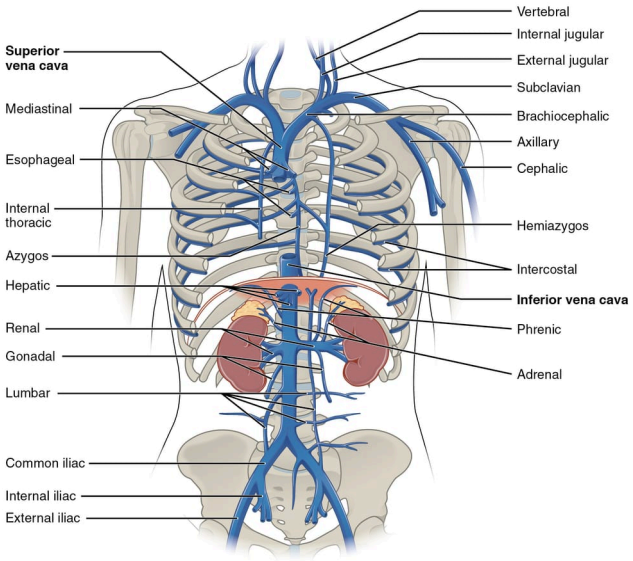


Figure 7.5.11: Veins of the thoracic and abdominal regions drain blood from the area above the diaphragm, returning it to the right atrium via the superior vena cava. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.11.

Table 7.5.10: Veins of the Thoracic Region

Vessel	Description
Superior vena cava	Large systemic vein; drains blood from most areas superior to the diaphragm; empties into the right atrium
Subclavian vein	Located deep in the thoracic cavity; formed by the axillary vein as it enters the thoracic cavity from the axillary region; drains the axillary and smaller local veins near the scapular region and leads to the brachiocephalic vein
Brachiocephalic veins	Pair of veins that form from a fusion of the external and internal jugular veins and the subclavian vein; subclavian, external and internal jugulars, vertebral, and internal thoracic veins flow into it; drain the upper thoracic region and lead to the superior vena cava
Vertebral vein	Arises from the base of the brain and the cervical region of the spinal cord; passes through the intervertebral foramina in the cervical vertebrae; drains smaller veins from the cranium, spinal cord, and vertebrae, and leads to the brachiocephalic vein; counterpart of the vertebral artery
Internal thoracic veins	Also called internal mammary veins; drain the anterior surface of the chest wall and lead to the brachiocephalic vein
Intercostal vein	Drains the muscles of the thoracic wall and leads to the azygos vein
Esophageal vein	Drains the inferior portions of the esophagus and leads to the azygos vein
Bronchial vein	Drains the systemic circulation from the lungs and leads to the azygos vein
Azygos vein	Originates in the lumbar region and passes through the diaphragm into the thoracic cavity on the right side of the vertebral column; drains blood from the intercostal veins, esophageal veins, bronchial veins, and other veins draining the mediastinal region, and leads to the superior vena cava
Hemiazygos vein	Smaller vein complementary to the azygos vein; drains the esophageal veins from the esophagus and the left intercostal veins, and leads to the brachiocephalic vein via the superior intercostal vein

Veins of the Head and Neck

Blood from the brain and the superficial facial vein flow into each internal jugular vein. Blood from the more superficial portions of the head, scalp, and cranial regions, including the temporal vein and maxillary vein, flow into each external jugular vein. Although the external and internal jugular veins are separate vessels, there are anastomoses between them close to the thoracic region. Blood from the external jugular vein empties into the subclavian vein. Table 7.5.11 summarizes the major veins of the head and

neck.

Table 7.5.11: Major Veins of the Head and Neck

Vessel	Description
Internal jugular vein	Parallel to the common carotid artery, which is more or less its counterpart, and passes through the jugular foramen and canal; primarily drains blood from the brain, receives the superficial facial vein, and empties into the subclavian vein
Temporal vein	Drains blood from the temporal region and flows into the external jugular vein
Maxillary vein	Drains blood from the maxillary region and flows into the external jugular vein
External jugular vein	Drains blood from the more superficial portions of the head, scalp, and cranial regions, and leads to the subclavian vein

Venous Drainage of the Brain

Circulation to the brain is both critical and complex. Many smaller veins of the brain stem and the superficial veins of the cerebrum lead to larger vessels referred to as intracranial sinuses. These include the superior and inferior sagittal sinuses, straight sinus, cavernous sinuses, left and right sinuses, the petrosal sinuses, and the occipital sinuses. Ultimately, sinuses will lead back to either the inferior jugular vein or vertebral vein.

Most of the veins on the superior surface of the cerebrum flow into the largest of the sinuses, the superior sagittal sinus. It is located midsagittally between the meningeal and periosteal layers of the dura mater within the falx cerebri and, at first glance in images or models, can be mistaken for the subarachnoid space. Most reabsorption of cerebrospinal fluid occurs via the chorionic villi (arachnoid granulations) into the superior sagittal sinus. Blood from most of the smaller vessels originating from the inferior cerebral veins flows into the great cerebral vein and into the straight sinus. Other cerebral veins and those from the eye socket flow into the cavernous sinus, which flows into the petrosal sinus and then into the internal jugular vein. The occipital sinus, sagittal sinus, and straight sinuses all flow into the left and right transverse sinuses near the lambdoid suture. The transverse sinuses in turn flow into the sigmoid sinuses that pass through the jugular foramen and into the internal jugular vein. The internal jugular vein flows parallel to the common carotid artery and is more or less its counterpart. It empties into the brachiocephalic vein. The veins draining the cervical vertebrae and the posterior surface of the skull, including some blood from the occipital sinus, flow into the vertebral veins. These parallel the vertebral arteries and travel through the transverse foramina of the cervical vertebrae. The vertebral veins also flow into the brachiocephalic veins. Table 7.5.11 summarizes the major veins of the brain.

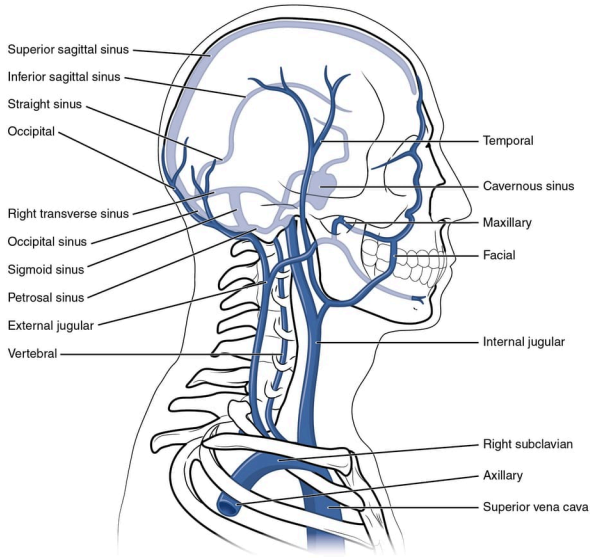


Figure 7.5.12: This left lateral view shows the veins of the head and neck, including the intercranial sinuses. (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 7.5.12.

Table 7.5.12: Major Veins of the Brain

Vessel	Description
Superior sagittal sinus	Enlarged vein located midsagittally between the meningeal and periosteal layers of the dura mater within the falx cerebri; receives most of the blood drained from the superior surface of the cerebrum and leads to the inferior jugular vein and the vertebral vein
Great cerebral vein	Receives most of the smaller vessels from the inferior cerebral veins and leads to the straight sinus
Straight sinus	Enlarged vein that drains blood from the brain; receives most of the blood from the great cerebral vein and leads to the left or right transverse sinus
Cavernous sinus	Enlarged vein that receives blood from most of the other cerebral veins and the eye socket, and leads to the petrosal sinus
Petrosal sinus	Enlarged vein that receives blood from the cavernous sinus and leads into the internal jugular veins
Occipital sinus	Enlarged vein that drains the occipital region near the falx cerebelli and leads to the left and right transverse sinuses, and also the vertebral veins
Transverse sinuses	Pair of enlarged veins near the lambdoid suture that drains the occipital, sagittal, and straight sinuses, and leads to the sigmoid sinuses
Sigmoid sinuses	Enlarged vein that receives blood from the transverse sinuses and leads through the jugular foramen to the internal jugular vein

Veins Draining the Upper Limbs

The digital veins in the fingers come together in the hand to form the palmar venous arches. From here, the veins come together to form the radial vein, the ulnar vein, and the median antebrachial vein. The radial vein and the ulnar vein parallel the bones of the forearm and join together at the antebrachium to form the brachial vein, a deep vein that flows into the axillary vein in the brachium.

The median antebrachial vein parallels the ulnar vein, is more medial in location, and joins the basilic vein in the forearm. As the basilic vein reaches the antecubital region, it gives off a branch called the median cubital vein that crosses at an angle to join the cephalic vein. The median cubital vein is the most common site for drawing venous blood in humans. The basilic vein continues

through the arm medially and superficially to the axillary vein.

The cephalic vein begins in the antebrachium and drains blood from the superficial surface of the arm into the axillary vein. It is extremely superficial and easily seen along the surface of the biceps brachii muscle in individuals with good muscle tone and in those without excessive subcutaneous adipose tissue in the arms.

The subscapular vein drains blood from the subscapular region and joins the cephalic vein to form the axillary vein. As it passes through the body wall and enters the thorax, the axillary vein becomes the subclavian vein.

Many of the larger veins of the thoracic and abdominal region and upper limb are further represented in the flow chart in Figure 7.5.13. Table 7.5.12 summarizes the veins of the upper limbs.

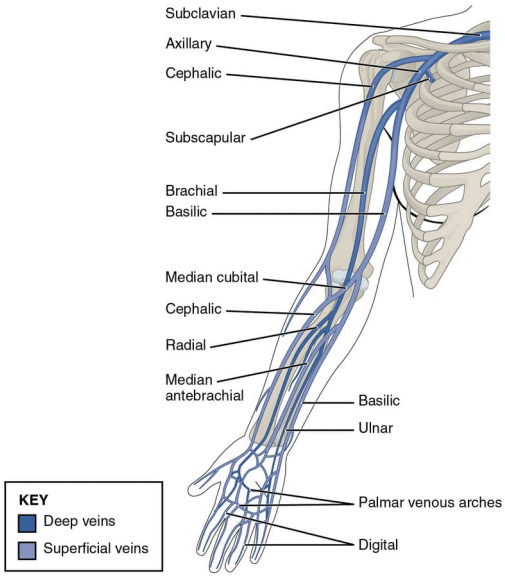


Figure 7.5.13: This anterior view shows the veins that drain the upper limb. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.13.

Table 7.5.13: Veins of the Upper Limbs

Vessel	Description
Digital veins	Drain the digits and lead to the palmar arches of the hand and dorsal venous arch of the foot
Palmar venous arches	Drain the hand and digits, and lead to the radial vein, ulnar veins, and the median antebrachial vein
Radial vein	Vein that parallels the radius and radial artery; arises from the palmar venous arches and leads to the brachial vein
Ulnar vein	Vein that parallels the ulna and ulnar artery; arises from the palmar venous arches and leads to the brachial vein
Brachial vein	Deeper vein of the arm that forms from the radial and ulnar veins in the lower arm; leads to the axillary vein
Median antebrachial vein	Vein that parallels the ulnar vein but is more medial in location; intertwines with the palmar venous arches; leads to the basilic vein
Basilic vein	Superficial vein of the arm that arises from the median antebrachial vein, intersects with the median cubital vein, parallels the ulnar vein, and continues into the upper arm; along with the brachial vein, it leads to the axillary vein
Median cubital vein	Superficial vessel located in the antecubital region that links the cephalic vein to the basilic vein in the form of a v; a frequent site from which to draw blood
Cephalic vein	Superficial vessel in the upper arm; leads to the axillary vein

Vessel	Description
Subscapular vein	Drains blood from the subscapular region and leads to the axillary vein
Axillary vein	The major vein in the axillary region; drains the upper limb and becomes the subclavian vein

The Inferior Vena Cava

Other than the small amount of blood drained by the azygos and hemiazygos veins, most of the blood inferior to the diaphragm drains into the inferior vena cava before it is returned to the heart. Lying just beneath the parietal peritoneum in the abdominal cavity, the inferior vena cava parallels the abdominal aorta, where it can receive blood from abdominal veins. The lumbar portions of the abdominal wall and spinal cord are drained by a series of lumbar veins, usually four on each side. The ascending lumbar veins drain into either the azygos vein on the right or the hemiazygos vein on the left, and return to the superior vena cava. The remaining lumbar veins drain directly into the inferior vena cava.

Blood supply from the kidneys flows into each renal vein, normally the largest veins entering the inferior vena cava. A number of other, smaller veins empty into the left renal vein. Each adrenal vein drains the adrenal or suprarenal glands located immediately superior to the kidneys. The right adrenal vein enters the inferior vena cava directly, whereas the left adrenal vein enters the left renal vein.

From the male reproductive organs, each testicular vein flows from the scrotum, forming a portion of the spermatic cord. Each ovarian vein drains an ovary in females. Each of these veins is generically called a gonadal vein. The right gonadal vein empties directly into the inferior vena cava, and the left gonadal vein empties into the left renal vein.

Each side of the diaphragm drains into a phrenic vein; the right phrenic vein empties directly into the inferior vena cava, whereas the left phrenic vein empties into the left renal vein. Blood supply from the liver drains into each hepatic vein and directly into the inferior vena cava. Since the inferior vena cava lies primarily to the right of the vertebral column and aorta, the left renal vein is longer, as are the left phrenic, adrenal, and gonadal veins. The longer length of the left renal vein makes the left kidney the primary target of surgeons removing this organ for donation.

Table 7.5.14: Major Veins of the Abdominal Region

Vessel	Description
Inferior vena cava	Large systemic vein that drains blood from areas largely inferior to the diaphragm; empties into the right atrium
Lumbar veins	Series of veins that drain the lumbar portion of the abdominal wall and spinal cord; the ascending lumbar veins drain into the azygos vein on the right or the hemiazygos vein on the left; the remaining lumbar veins drain directly into the inferior vena cava
Renal vein	Largest vein entering the inferior vena cava; drains the kidneys and flows into the inferior vena cava
Adrenal vein	Drains the adrenal or suprarenal; the right adrenal vein enters the inferior vena cava directly and the left adrenal vein enters the left renal vein
Testicular vein	Drains the testes and forms part of the spermatic cord; the right testicular vein empties directly into the inferior vena cava and the left testicular vein empties into the left renal vein
Ovarian vein	Drains the ovary; the right ovarian vein empties directly into the inferior vena cava and the left ovarian vein empties into the left renal vein
Gonadal vein	Generic term for a vein draining a reproductive organ; may be either an ovarian vein or a testicular vein, depending on the sex of the individual
Phrenic vein	Drains the diaphragm; the right phrenic vein flows into the inferior vena cava and the left phrenic vein empties into the left renal vein
Hepatic vein	Drains systemic blood from the liver and flows into the inferior vena cava

Veins Draining the Lower Limbs

The superior surface of the foot drains into the digital veins, and the inferior surface drains into the plantar veins, which flow into a complex series of anastomoses in the feet and ankles, including the dorsal venous arch and the plantar venous arch. From the dorsal venous arch, blood supply drains into the anterior and posterior tibial veins. The anterior tibial vein drains the area near the tibialis anterior muscle and combines with the posterior tibial vein and the fibular vein to form the popliteal vein. The posterior tibial vein drains the posterior surface of the tibia and joins the popliteal vein. The fibular vein drains the muscles and integument in proximity to the fibula and also joins the popliteal vein. The small saphenous vein located on the lateral surface of the leg drains blood from the superficial regions of the lower leg and foot, and flows into to the popliteal vein. As the popliteal vein passes behind the knee in the popliteal region, it becomes the femoral vein. It is palpable in patients without excessive adipose tissue.

Close to the body wall, the great saphenous vein, the deep femoral vein, and the femoral circumflex vein drain into the femoral vein. The great saphenous vein is a prominent surface vessel located on the medial surface of the leg and thigh that collects blood from the superficial portions of these areas. The deep femoral vein, as the name suggests, drains blood from the deeper portions of the thigh. The femoral circumflex vein forms a loop around the femur just inferior to the trochanters and drains blood from the areas in proximity to the head and neck of the femur.

As the femoral vein penetrates the body wall from the femoral portion of the upper limb, it becomes the external iliac vein, a large vein that drains blood from the leg to the common iliac vein. The pelvic organs and integument drain into the internal iliac vein, which forms from several smaller veins in the region, including the umbilical veins that run on either side of the bladder. The external and internal iliac veins combine near the inferior portion of the sacroiliac joint to form the common iliac vein. In addition to blood supply from the external and internal iliac veins, the middle sacral vein drains the sacral region into the common iliac vein. Similar to the common iliac arteries, the common iliac veins come together at the level of L5 to form the inferior vena cava.

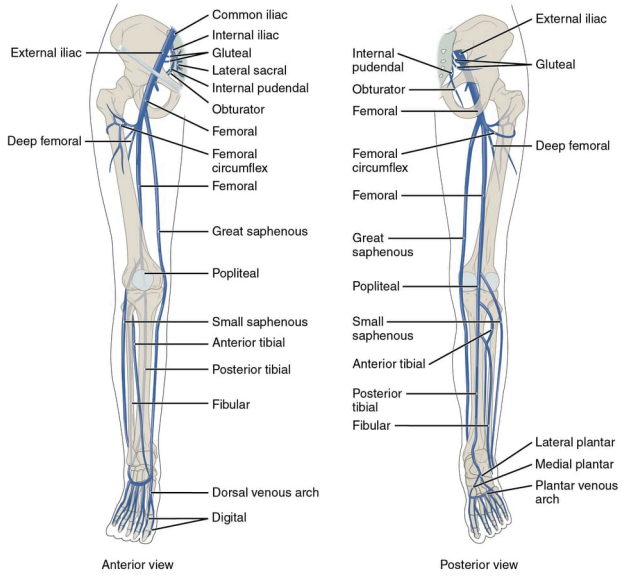


Figure 7.5.14: Anterior and posterior views show the major veins that drain the lower limb into the inferior vena cava. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.5.14.

Table 7.5.15: Veins of the Lower Limbs

Vessel	Description
Plantar veins	Drain the foot and flow into the plantar venous arch
Dorsal venous arch	Drains blood from digital veins and vessels on the superior surface of the foot
Plantar venous arch	Formed from the plantar veins; flows into the anterior and posterior tibial veins through anastomoses

Vessel	Description
Anterior tibial vein	Formed from the dorsal venous arch; drains the area near the tibialis anterior muscle and flows into the popliteal vein
Posterior tibial vein	Formed from the dorsal venous arch; drains the area near the posterior surface of the tibia and flows into the popliteal vein
Fibular vein	Drains the muscles and integument near the fibula and flows into the popliteal vein
Small saphenous vein	Located on the lateral surface of the leg; drains blood from the superficial regions of the lower leg and foot, and flows into the popliteal vein
Popliteal vein	Drains the region behind the knee and forms from the fusion of the fibular, anterior, and posterior tibial veins; flows into the femoral vein
Great saphenous vein	Prominent surface vessel located on the medial surface of the leg and thigh; drains the superficial portions of these areas and flows into the femoral vein
Deep femoral vein	Drains blood from the deeper portions of the thigh and flows into the femoral vein
Femoral circumflex vein	Forms a loop around the femur just inferior to the trochanters; drains blood from the areas around the head and neck of the femur; flows into the femoral vein
Femoral vein	Drains the upper leg; receives blood from the great saphenous vein, the deep femoral vein, and the femoral circumflex vein; becomes the external iliac vein when it crosses the body wall
External iliac vein	Formed when the femoral vein passes into the body cavity; drains the legs and flows into the common iliac vein
Internal iliac vein	Drains the pelvic organs and integument; formed from several smaller veins in the region; flows into the common iliac vein
Middle sacral vein	Drains the sacral region and flows into the left common iliac vein
Common iliac vein	Flows into the inferior vena cava at the level of L5; the left common iliac vein drains the sacral region; formed from the union of the external and internal iliac veins near the inferior portion of the sacroiliac joint

Hepatic Portal System

The liver is a complex biochemical processing plant. It packages nutrients absorbed by the digestive system; produces plasma proteins, clotting factors, and bile; and disposes of worn-out cell components and waste products. Instead of entering the circulation directly, absorbed nutrients and certain wastes (for example, materials produced by the spleen) travel to the liver for processing. They do so via the hepatic portal system. Portal systems begin and end in capillaries. In this case, the initial capillaries from the stomach, small intestine, large intestine, and spleen lead to the hepatic portal vein and end in specialized capillaries within the liver, the hepatic sinusoids. You saw the only other portal system with the hypothalamic-hypophyseal portal vessel in the endocrine chapter.

The hepatic portal system consists of the hepatic portal vein and the veins that drain into it. The hepatic portal vein itself is relatively short, beginning at the level of L2 with the confluence of the superior mesenteric and splenic veins. It also receives branches from the inferior mesenteric vein, plus the splenic veins and all their tributaries. The superior mesenteric vein receives blood from the small intestine, two-thirds of the large intestine, and the stomach. The inferior mesenteric vein drains the distal third of the large intestine, including the descending colon, the sigmoid colon, and the rectum. The splenic vein is formed from branches from the spleen, pancreas, and portions of the stomach, and the inferior mesenteric vein. After its formation, the hepatic portal vein also receives branches from the gastric veins of the stomach and cystic veins from the gall bladder. The hepatic portal vein delivers materials from these digestive and circulatory organs directly to the liver for processing.

Because of the hepatic portal system, the liver receives its blood supply from two different sources: from normal systemic circulation via the hepatic artery and from the hepatic portal vein. The liver processes the blood from the portal system to remove

certain wastes and excess nutrients, which are stored for later use. This processed blood, as well as the systemic blood that came from the hepatic artery, exits the liver via the right, left, and middle hepatic veins, and flows into the inferior vena cava. Overall systemic blood composition remains relatively stable, since the liver is able to metabolize the absorbed digestive components.

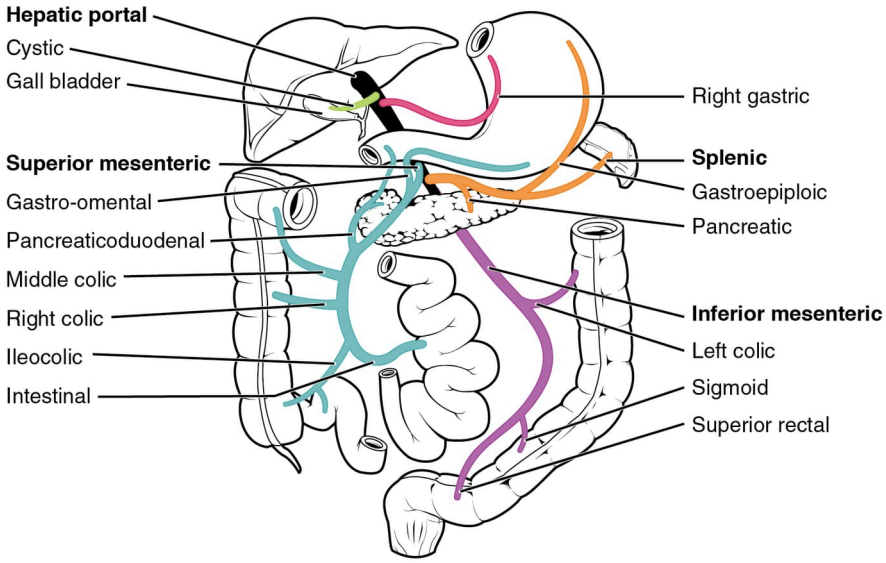


Figure 7.5.15: The liver receives blood from the normal systemic circulation via the hepatic artery. It also receives and processes blood from other organs, delivered via the veins of the hepatic portal system. All blood exits the liver via the hepatic vein, which delivers the blood to the inferior vena cava. (Different colors are used to help distinguish among the different vessels in the system.). (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 7.5.15.

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7.6: Development of Blood Vessels and Fetal Circulation

Embryonic and Fetal Circulation

In a developing embryo,the heart has developed enough by day 21 post-fertilization to begin beating. Circulation patterns are clearly established by the fourth week of embryonic life. It is critical to the survival of the developing human that the circulatory system forms early to supply the growing tissue with nutrients and gases, and to remove waste products. Blood cells and vessel production in structures outside the embryo proper called the yolk sac, chorion, and connecting stalk begin about 15 to 16 days following fertilization. Development of these circulatory elements within the embryo itself begins approximately 2 days later. You will learn more about the formation and function of these early structures when you study the chapter on development. During those first few weeks, blood vessels begin to form from the embryonic mesoderm. The precursor cells are known as hemangioblasts. These in turn differentiate into angioblasts, which give rise to the blood vessels and pluripotent stem cells, which differentiate into the formed elements of blood. (Seek additional content for more detail on fetal development and circulation.) Together, these cells form masses known as blood islands scattered throughout the embryonic disc. Spaces appear on the blood islands that develop into vessel lumens. The endothelial lining of the vessels arise from the angioblasts within these islands. Surrounding mesenchymal cells give rise to the smooth muscle and connective tissue layers of the vessels. While the vessels are developing, the pluripotent stem cells begin to form the blood.

Vascular tubes also develop on the blood islands, and they eventually connect to one another as well as to the developing, tubular heart. Thus, the developmental pattern, rather than beginning from the formation of one central vessel and spreading outward, occurs in many regions simultaneously with vessels later joining together. This angiogenesis—the creation of new blood vessels from existing ones—continues as needed throughout life as we grow and develop.

Blood vessel development often follows the same pattern as nerve development and travels to the same target tissues and organs. This occurs because the many factors directing growth of nerves also stimulate blood vessels to follow a similar pattern. Whether a given vessel develops into an artery or a vein is dependent upon local concentrations of signaling proteins.

As the embryo grows within the mother's uterus, its requirements for nutrients and gas exchange also grow. The placenta—a circulatory organ unique to pregnancy—develops jointly from the embryo and uterine wall structures to fill this need. Emerging from the placenta is the umbilical vein, which carries oxygen-rich blood from the pregnant person to the fetal inferior vena cava via the ductus venosus to the heart that pumps it into fetal circulation. Two umbilical arteries carry oxygen-depleted fetal blood, including wastes and carbon dioxide, to the placenta. Remnants of the umbilical arteries remain in the adult. (Seek additional content for more information on the role of the placenta in fetal circulation.)

There are three major shunts—alternate paths for blood flow—found in the circulatory system of the fetus. Two of these shunts divert blood from the pulmonary to the systemic circuit, whereas the third connects the umbilical vein to the inferior vena cava. The first two shunts are critical during fetal life, when the lungs are compressed, filled with amniotic fluid, and nonfunctional, and gas exchange is provided by the placenta. These shunts close shortly after birth, however, when the newborn begins to breathe. The third shunt persists a bit longer but becomes nonfunctional once the umbilical cord is severed. The three shunts are as follows (Figure 7.6.1):

- The foramen ovale is an opening in the interatrial septum that allows blood to flow from the right atrium to the left atrium. A valve associated with this opening prevents backflow of blood during the fetal period. As the newborn begins to breathe and blood pressure in the atria increases, this shunt closes. The fossa ovalis remains in the interatrial septum after birth, marking the location of the former foramen ovale.
- The ductus arteriosus is a short, muscular vessel that connects the pulmonary trunk to the aorta. Most of the blood pumped from the right ventricle into the pulmonary trunk is thereby diverted into the aorta. Only enough blood reaches the fetal lungs to maintain the developing lung tissue. When the newborn takes the first breath, pressure within the lungs drops dramatically, and both the lungs and the pulmonary vessels expand. As the amount of oxygen increases, the smooth muscles in the wall of the ductus arteriosus constrict, sealing off the passage. Eventually, the muscular and endothelial components of the ductus arteriosus degenerate, leaving only the connective tissue component of the ligamentum arteriosum.
- The ductus venosus is a temporary blood vessel that branches from the umbilical vein, allowing much of the freshly oxygenated blood from the placenta—the organ of gas exchange between the pregnant person and fetus—to bypass the fetal liver and go directly to the fetal heart. The ductus venosus closes slowly during the first weeks of infancy and degenerates to become the ligamentum venosum.

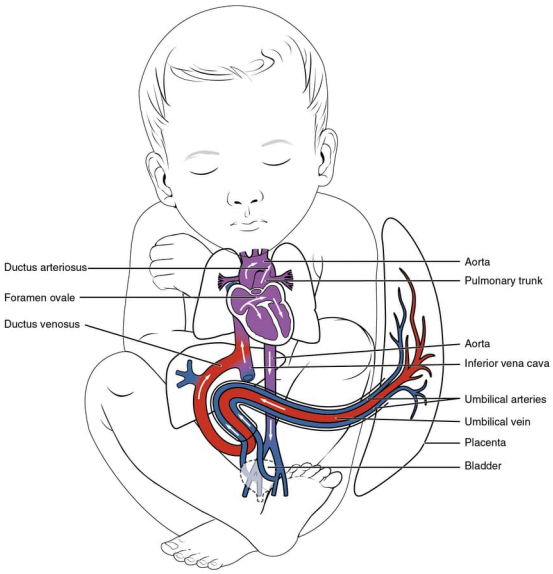


Figure 7.6.1: The foramen ovale in the interatrial septum allows blood to flow from the right atrium to the left atrium. The ductus arteriosus is a temporary vessel, connecting the aorta to the pulmonary trunk. The ductus venosus links the umbilical vein to the inferior vena cava largely through the liver. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 7.6.1.

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7.7: Terms and Definitions

Key Terms

Select to View Definitions

Abdominal Aorta
Portion of the aorta inferior to the aortic hiatus and superior to the common iliac arteries.

Adrenal Artery
Branch of the abdominal aorta; supplies blood to the adrenal (suprarenal) glands.

Adrenal Vein
Drains the adrenal glands superior to the kidneys; the right adrenal vein enters the inferior vena cava directly and the left adrenal vein enters the left renal vein.

Anaphylactic Shock
Type of shock that follows a severe allergic reaction and results from massive vasodilation.

Angioblasts
Stem cells that give rise to blood vessels.

Angiogenesis
Development of new blood vessels from existing vessels.

Anterior Cerebral Artery
Arises from the internal carotid artery; supplies the frontal lobe of the cerebrum.

Anterior Communicating Artery
Anastomosis of the right and left internal carotid arteries; supplies blood to the brain.

Anterior Tibial Artery
Branches from the popliteal artery; supplies blood to the anterior tibial region and becomes the dorsalis pedis artery.

Anterior Tibial Vein
Forms from the dorsal venous arch; drains the area near the tibialis anterior muscle and leads to the popliteal vein.

Aorta
Largest artery in the body, originating from the left ventricle and distributing blood to virtually all tissues.

Aortic Arch
Arc connecting the ascending aorta to the descending aorta.

Aortic Hiatus
Opening in the diaphragm that allows passage of the thoracic aorta into the abdominal region.

Aortic Sinuses
Small pockets in the ascending aorta that contain baroreceptors and chemoreceptors involved in vascular homeostasis.

Arterial Circle
Also called the Circle of Willis; an anastomosis at the base of the brain that helps ensure continuous blood supply.

Arteriole
Very small artery that leads to a capillary; also called a resistance vessel.

Arteriovenous Anastomosis
Short vessel connecting an arteriole directly to a venule, bypassing capillary beds.

Artery
Blood vessel that conducts blood away from the heart.

Ascending Aorta
Initial portion of the aorta arising from the left ventricle.

Atrial Reflex
Mechanism that helps maintain vascular homeostasis through atrial baroreceptors.

Axillary Artery
Continuation of the subclavian artery that supplies the axillary region and upper limb.

Axillary Vein
Major vein of the axillary region that drains the upper limb and becomes the subclavian vein.

Azygos Vein
Vein on the right side of the vertebral column that drains thoracic structures and leads to the superior vena cava.

Basilar Artery
Formed by the fusion of the vertebral arteries; supplies the brain stem and cerebellum.

Basilic Vein
Superficial vein of the arm that contributes to formation of the axillary vein.

Blood Colloidal Osmotic Pressure (BCOP)
Pressure exerted by plasma proteins and other colloids within the blood.

Blood Flow
Movement of blood through a vessel, tissue, or organ.

Blood Hydrostatic Pressure
Force exerted by blood against the walls of blood vessels or heart chambers.

Blood Islands
Masses of developing blood vessels and formed elements in the embryonic disc.

Blood Pressure
Force exerted by blood against a vessel wall.

Brachial Artery
Continuation of the axillary artery that supplies the arm and divides into the radial and ulnar arteries.

Brachial Vein
Deep vein of the arm that drains into the axillary vein.

Brachiocephalic Artery
First branch of the aortic arch that gives rise to the right subclavian and right common carotid arteries.

Brachiocephalic Vein
Vein formed by the union of the jugular and subclavian veins; drains into the superior vena cava.

Bronchial Artery
Systemic branch of the aorta that supplies oxygenated blood to the lungs.

Bronchial Vein
Vein that drains the systemic circulation of the lungs into the azygos system.

Capacitance
Ability of a vein to distend and store blood.

Capacitance Vessels
Veins that function as blood reservoirs.

Capillary
Smallest blood vessel where exchange occurs between blood and tissues.

Capillary Bed
Network of capillaries connecting arterioles to venules.

Capillary Hydrostatic Pressure (CHP)
Force exerted by blood against capillary walls.

Cardiogenic Shock
Type of shock resulting from the inability of the heart to maintain adequate cardiac output.

Carotid Sinuses
Small pockets near the base of the internal carotid arteries that are the locations of baroreceptors and chemoreceptors involved in regulating vascular homeostasis.

Cavernous Sinus
Enlarged vein that receives blood from most cerebral veins and the eye socket and drains into the petrosal sinus.

Celiac Trunk
(Also called the celiac artery) Major branch of the abdominal aorta that gives rise to the left gastric, splenic, and common hepatic arteries.

Cephalic Vein
Superficial vessel in the upper arm that drains into the axillary vein.

Cerebrovascular Accident (CVA)
Blockage of blood flow to the brain; also called a stroke.

Circle of Willis
(Also called the arterial circle) Anastomosis at the base of the brain that ensures continual blood supply to the brain.

Circulatory Shock
Life-threatening condition in which the circulatory system cannot provide adequate blood flow and oxygen delivery to tissues.

Common Carotid Artery
Major artery of the head and neck that divides into the external and internal carotid arteries.

Common Hepatic Artery
Branch of the celiac trunk that forms the hepatic artery, right gastric artery, and cystic artery.

Common Iliac Artery
Branch of the aorta that divides into the internal and external iliac arteries.

Common Iliac Vein
Vein that flows into the inferior vena cava at the level of L5 and divides into the external and internal iliac veins.

Compliance
Degree to which a blood vessel can stretch rather than remain rigid.

Continuous Capillary
Most common type of capillary, containing small gaps in the endothelial lining that permit exchange.

Cystic Artery
Branch of the common hepatic artery that supplies blood to the gallbladder.

Deep Femoral Artery
Branch of the femoral artery that gives rise to the lateral circumflex arteries.

Deep Femoral Vein
Vein that drains the deeper portions of the thigh and leads to the femoral vein.

Descending Aorta
Portion of the aorta that continues inferiorly beyond the aortic arch and includes the thoracic and abdominal aorta.

Diastolic Pressure
Lower arterial blood pressure value measured during ventricular relaxation.

Digital Arteries
Arteries formed from the superficial and deep palmar arches that supply blood to the digits.

Digital Veins
Veins that drain the digits and empty into the palmar arches of the hand and dorsal venous arch of the foot.

Dorsal Arch
(Also called the arcuate arch) Arterial arch formed by an anastomosis of the dorsalis pedis and plantar arteries that supplies the distal foot and digits.

Dorsal Venous Arch
Venous arch that drains blood from digital veins and vessels on the superior surface of the foot.

Dorsalis Pedis Artery
Continuation of the anterior tibial artery that supplies the tarsal and dorsal regions of the foot.

Ductus Arteriosus
Fetal shunt connecting the pulmonary trunk to the aorta.

Ductus Venosus
Fetal shunt that allows oxygenated blood to bypass the liver and flow into the inferior vena cava.

Elastic Artery
(Also called a conducting artery) Artery rich in elastic fibers that helps maintain pressure and conduct blood from the heart to smaller vessels.

Esophageal Artery
Branch of the thoracic aorta that supplies blood to the esophagus.

Esophageal Vein
Vein that drains the inferior portions of the esophagus and empties into the azygos vein.

External Carotid Artery
Branch of the common carotid artery that supplies the face, lower jaw, neck, esophagus, and larynx.

External Elastic Membrane
Layer of elastic fibers that separates the tunica media from the tunica externa in larger arteries.

External Iliac Artery
Branch of the common iliac artery that becomes the femoral artery and supplies the lower limbs.

External Iliac Vein
Vein formed when the femoral vein enters the body cavity; drains the legs and joins the common iliac vein.

External Jugular Vein
Major superficial vein of the neck that drains the scalp, head, and cranial regions into the subclavian vein.

Femoral Artery
Continuation of the external iliac artery that supplies the thigh and becomes the popliteal artery behind the knee.

Femoral Circumflex Vein
Vein that encircles the femur and drains blood from the regions surrounding the head and neck of the femur.

Femoral Vein
Major vein of the thigh that receives blood from several tributaries and becomes the external iliac vein.

Fenestrated Capillary
Capillary containing pores in the endothelium that allow rapid exchange of small substances.

Fibular Vein
Vein that drains tissues near the fibula and empties into the popliteal vein.

Filtration
Movement of fluid and dissolved substances from a capillary into the interstitial fluid due to pressure differences.

Foramen Ovale
Fetal opening between the right and left atria that diverts blood away from the pulmonary circulation.

Genicular Artery
Branch of the femoral artery that supplies the knee region.

Gonadal Artery
Branch of the abdominal aorta that supplies the ovaries or testes.

Gonadal Vein
General term for a vein that drains a reproductive organ, such as an ovarian or testicular vein.

Great Cerebral Vein
Large vein that receives blood from inferior cerebral veins and drains into the straight sinus.

Great Saphenous Vein
Large superficial vein on the medial leg and thigh that drains into the femoral vein.

Hemangioblasts
Embryonic stem cells that give rise to angioblasts and pluripotent stem cells.

Hemiazygos Vein
Vein on the left side of the vertebral column that drains into the brachiocephalic vein through the superior intercostal vein.

Hepatic Artery Proper
Branch of the common hepatic artery that supplies oxygenated blood to the liver.

Hepatic Portal System
Specialized circulatory pathway that transports blood from digestive organs to the liver for processing.

Hepatic Vein
Vein that drains blood from the liver into the inferior vena cava.

Hypertension
Chronic elevation of arterial blood pressure, typically 140/90 mm Hg or higher.

Hypervolemia
Abnormally high blood or fluid volume in the body.

Hypovolemia
Abnormally low blood or fluid volume in the body.

Hypovolemic Shock
Circulatory shock caused by severe loss of blood or body fluids.

Hypoxia
Insufficient oxygen supply to body tissues.

Inferior Mesenteric Artery
Branch of the abdominal aorta that supplies the distal large intestine and rectum.

Inferior Phrenic Artery
Branch of the abdominal aorta that supplies the inferior surface of the diaphragm.

Inferior Vena Cava
Large vein that returns blood from regions inferior to the diaphragm to the right atrium.

Intercostal Artery
Branch of the thoracic aorta that supplies the thoracic wall and vertebral column.

Intercostal Vein
Vein that drains the thoracic wall and empties into the azygos vein.

Internal Carotid Artery
Major artery supplying the brain; contributes to the circle of Willis.

Internal Elastic Membrane
Elastic tissue layer that separates the tunica intima from the tunica media in larger arteries.

Internal Iliac Artery
Branch of the common iliac artery that supplies pelvic organs, pelvic walls, and external genitalia.

Internal Iliac Vein
Vein that drains the pelvis and empties into the common iliac vein.

Internal Jugular Vein
Major vein of the neck that drains blood from the brain and empties into the subclavian vein.

Internal Thoracic Artery
Branch of the subclavian artery that supplies the thymus, pericardium, and anterior chest wall.

Internal Thoracic Vein
Vein that drains the anterior chest wall and empties into the brachiocephalic vein.

Interstitial Fluid Colloidal Osmotic Pressure (IFCOP)
Pressure exerted by proteins and other colloids within the interstitial fluid.

Interstitial Fluid Hydrostatic Pressure (IFHP)
Pressure exerted by fluid within the tissue spaces.

Ischemia
Insufficient blood flow to tissues.

Korotkoff Sounds
Sounds produced by turbulent blood flow during blood pressure measurement.

Lateral Circumflex Artery
Branch of the deep femoral artery that supplies the thigh and surrounding tissues.

Lateral Plantar Artery
Branch of the posterior tibial artery that supplies the lateral plantar surface of the foot.

Left Gastric Artery
Branch of the celiac trunk that supplies the stomach.

Lumbar Arteries
Branches of the abdominal aorta that supply the lumbar region, abdominal wall, and spinal cord.

Lumbar Veins
Veins that drain the lumbar region and spinal cord.

Lumen
Interior space of a tubular structure through which fluid passes.

Maxillary Vein
Vein that drains the maxillary region and empties into the external jugular vein.

Mean Arterial Pressure (MAP)
Average pressure driving blood through the systemic circulation.

Medial Plantar Artery
Branch of the posterior tibial artery that supplies the medial plantar surface of the foot.

Median Antebrachial Vein
Medial forearm vein associated with the palmar venous arches.

Median Cubital Vein
Superficial vein connecting the cephalic and basilic veins; common site for venipuncture.

Median Sacral Artery
Continuation of the aorta into the sacral region.

Mediastinal Artery
Branch of the thoracic aorta that supplies the mediastinum.

Metarteriole
Short vessel connecting a terminal arteriole to a capillary bed.

Microcirculation
Blood flow through capillaries and associated microvessels.

Middle Cerebral Artery
Branch of the internal carotid artery that supplies the temporal and parietal lobes.

Middle Sacral Vein
Vein that drains the sacral region and empties into the left common iliac vein.

Muscular Artery
(Also called a distributing artery) Artery with abundant smooth muscle that distributes blood to organs and tissues.

Myogenic Response
Constriction or dilation of arterioles in response to changes in blood pressure to maintain consistent blood flow.

Nervi Vasorum
Small nerve fibers found in arteries and veins that trigger contraction of the smooth muscle in their walls.

Net Filtration Pressure (NFP)
Force driving fluid out of the capillary and into the tissue spaces; equal to the difference of the capillary hydrostatic pressure and the blood colloidal osmotic pressure.

Neurogenic Shock
Type of shock that occurs with cranial or high spinal injuries that damage the cardiovascular centers in the medulla oblongata or the nervous fibers originating from this region.

Obstructive Shock
Type of shock that occurs when a significant portion of the vascular system is blocked.

Occipital Sinus
Enlarged vein that drains the occipital region near the falx cerebelli and flows into the left and right transverse sinuses, and also into the vertebral veins.

Ophthalmic Artery
Branch of the internal carotid artery; supplies blood to the eyes.

Ovarian Artery
Branch of the abdominal aorta; supplies blood to the ovary, uterine (Fallopian) tube, and uterus.

Ovarian Vein
Drains the ovary; the right ovarian vein leads to the inferior vena cava and the left ovarian vein leads to the left renal vein.

Palmar Arches
Superficial and deep arches formed from anastomoses of the radial and ulnar arteries; supply blood to the hand and digital arteries.

Palmar Venous Arches
Drain the hand and digits, and feed into the radial and ulnar veins.

Parietal Branches
(Also, somatic branches) group of arterial branches of the thoracic aorta; includes those that supply blood to the thoracic cavity, vertebral column, and the superior surface of the diaphragm.

Perfusion
Distribution of blood into the capillaries so the tissues can be supplied.

Pericardial Artery
Branch of the thoracic aorta; supplies blood to the pericardium.

Petrosal Sinus
Enlarged vein that receives blood from the cavernous sinus and flows into the internal jugular vein.

Phrenic Vein
Drains the diaphragm; the right phrenic vein flows into the inferior vena cava and the left phrenic vein leads to the left renal vein.

Plantar Arch
Formed from the anastomosis of the dorsalis pedis artery and medial and plantar arteries; branches supply the distal portions of the foot and digits.

Plantar Veins
Drain the foot and lead to the plantar venous arch.

Plantar Venous Arch
Formed from the plantar veins; leads to the anterior and posterior tibial veins through anastomoses.

Popliteal Artery
Continuation of the femoral artery posterior to the knee; branches into the anterior and posterior tibial arteries.

Popliteal Vein
Continuation of the femoral vein behind the knee; drains the region behind the knee and forms from the fusion of the fibular and anterior and posterior tibial veins.

Posterior Cerebral Artery
Branch of the basilar artery that forms a portion of the posterior segment of the arterial circle; supplies blood to the posterior portion of the cerebrum and brain stem.

Posterior Communicating Artery
Branch of the posterior cerebral artery that forms part of the posterior portion of the arterial circle; supplies blood to the brain.

Posterior Tibial Artery
Branch from the popliteal artery that gives rise to the fibular or peroneal artery; supplies blood to the posterior

tibial region.

Posterior Tibial Vein
Forms from the dorsal venous arch; drains the area near the posterior surface of the tibia and leads to the popliteal vein.

Precapillary Sphincters
Circular rings of smooth muscle that surround the entrance to a capillary and regulate blood flow into that capillary.

Pulmonary Artery
One of two branches, left and right, that divides off from the pulmonary trunk and leads to smaller arterioles and eventually to the pulmonary capillaries.

Pulmonary Circuit
System of blood vessels that provide gas exchange via a network of arteries, veins, and capillaries that run from the heart, through the body, and back to the lungs.

Pulmonary Trunk
Single large vessel exiting the right ventricle that divides to form the right and left pulmonary arteries.

Pulmonary Veins
Two sets of paired vessels, one pair on each side, that are formed from the small venules leading away from the pulmonary capillaries that flow into the left atrium.

Pulse
Alternating expansion and recoil of an artery as blood moves through the vessel; an indicator of heart rate.

Pulse Pressure
Difference between the systolic and diastolic pressures.

Radial Artery
Formed at the bifurcation of the brachial artery; parallels the radius; gives off smaller branches until it reaches the carpal region where it fuses with the ulnar artery to form the superficial and deep palmar arches; supplies blood to the lower arm and carpal region.

Radial Vein
Parallels the radius and radial artery; arises from the palmar venous arches and leads to the brachial vein.

Reabsorption
In the cardiovascular system, the movement of material from the interstitial fluid into the capillaries.

Renal Artery
Branch of the abdominal aorta; supplies each kidney.

Renal Vein
Largest vein entering the inferior vena cava; drains the kidneys and leads to the inferior vena cava.

Resistance
Any condition or parameter that slows or counteracts the flow of blood.

Respiratory Pump
Increase in the volume of the thorax during inhalation that decreases air pressure, enabling venous blood to flow into the thoracic region, then exhalation increases pressure, moving blood into the atria.

Right Gastric Artery
Branch of the common hepatic artery; supplies blood to the stomach.

Sepsis
(Also, septicemia) organismal-level inflammatory response to a massive infection.

Septic Shock
(Also, blood poisoning) type of shock that follows a massive infection resulting in organism-wide inflammation.

Sigmoid Sinuses
Enlarged veins that receive blood from the transverse sinuses; flow through the jugular foramen and into the internal jugular vein.

Sinusoid Capillary
Rarest type of capillary, which has extremely large intercellular gaps in the basement membrane in addition to clefts and fenestrations; found in areas such as the bone marrow and liver where passage of large molecules occurs.

Skeletal Muscle Pump
Effect on increasing blood pressure within veins by compression of the vessel caused by the contraction of nearby skeletal muscle.

Small Saphenous Vein
Located on the lateral surface of the leg; drains blood from the superficial regions of the lower leg and foot, and leads to the popliteal vein.

Sphygmomanometer
Blood pressure cuff attached to a device that measures blood pressure.

Splenic Artery
Branch of the celiac trunk; supplies blood to the spleen.

Straight Sinus
Enlarged vein that drains blood from the brain; receives most of the blood from the great cerebral vein and flows into the left or right transverse sinus.

Subclavian Artery
Right subclavian arises from the brachiocephalic artery, whereas the left subclavian artery arises from the aortic arch; gives rise to the internal thoracic, vertebral, and thyrocervical arteries; supplies blood to the arms, chest, shoulders, back, and central nervous system.

Subclavian Vein
Located deep in the thoracic cavity; becomes the axillary vein as it enters the axillary region; drains the axillary and smaller local veins near the scapular region; leads to the brachiocephalic vein.

Subscapular Vein
Drains blood from the subscapular region and leads to the axillary vein.

Superior Mesenteric Artery
Branch of the abdominal aorta; supplies blood to the small intestine (duodenum, jejunum, and ileum), the pancreas, and a majority of the large intestine.

Superior Phrenic Artery
Branch of the thoracic aorta; supplies blood to the superior surface of the diaphragm.

Superior Sagittal Sinus
Enlarged vein located midsagittally between the meningeal and periosteal layers of the dura mater within the falx cerebri; receives most of the blood drained from the superior surface of the cerebrum and leads to the inferior jugular vein and the vertebral vein.

Superior Vena Cava
Large systemic vein; drains blood from most areas superior to the diaphragm; empties into the right atrium.

Systolic Pressure
Larger number recorded when measuring arterial blood pressure; represents the maximum value following ventricular contraction.

Temporal Vein
Drains blood from the temporal region and leads to the external jugular vein.

Testicular Artery
Branch of the abdominal aorta; will ultimately travel outside the body cavity to the testes and form one component of the spermatic cord.

Testicular Vein
Drains the testes and forms part of the spermatic cord; the right testicular vein empties directly into the inferior vena cava and the left testicular vein empties into the left renal vein.

Thoracic Aorta
Portion of the descending aorta superior to the aortic hiatus.

Thoroughfare Channel
Continuation of the metarteriole that enables blood to bypass a capillary bed and flow directly into a venule, creating a vascular shunt.

Thyrocervical Artery
Arises from the subclavian artery; supplies blood to the thyroid, the cervical region, the upper back, and shoulder.

Transient Ischemic Attack (TIA)
Temporary loss of neurological function caused by a brief interruption in blood flow; also known as a mini-stroke.

Transverse Sinuses
Pair of enlarged veins near the lambdoid suture that drain the occipital, sagittal, and straight sinuses, and leads to the sigmoid sinuses.

Trunk
Large vessel that gives rise to smaller vessels.

Tunica Externa
(Also, tunica adventitia) outermost layer or tunic of a vessel (except capillaries).

Tunica Intima
(Also, tunica interna) innermost lining or tunic of a vessel.

Tunica Media
Middle layer or tunic of a vessel (except capillaries).

Ulnar Artery
Formed at the bifurcation of the brachial artery; parallels the ulna; gives off smaller branches until it reaches the carpal region where it fuses with the radial artery to form the superficial and deep palmar arches; supplies blood to the lower arm and carpal region.

Ulnar Vein
Parallels the ulna and ulnar artery; arises from the palmar venous arches and leads to the brachial vein.

Umbilical Arteries
Pair of vessels that runs within the umbilical cord and carries fetal blood low in oxygen and high in waste to the placenta for exchange with maternal blood.

Umbilical Vein
Single vessel that originates in the placenta and runs within the umbilical cord, carrying oxygen- and nutrient-rich blood to the fetal heart.

Vasa Vasorum
Small blood vessels located within the walls or tunics of larger vessels that supply nourishment to and remove wastes from the cells of the vessels.

Vascular Shock
Type of shock that occurs when arterioles lose their normal muscular tone and dilate dramatically.

Vascular Shunt
Continuation of the metarteriole and thoroughfare channel that allows blood to bypass the capillary beds to flow directly from the arterial to the venous circulation.

Vascular Tone
Contractile state of smooth muscle in a blood vessel.

Vascular Tubes
Rudimentary blood vessels in a developing fetus.

Vasoconstriction
Constriction of the smooth muscle of a blood vessel, resulting in a decreased vascular diameter.

Vasodilation
Relaxation of the smooth muscle in the wall of a blood vessel, resulting in an increased vascular diameter.

Vasomotion
Irregular, pulsating flow of blood through capillaries and related structures.

Vein
Blood vessel that conducts blood toward the heart.

Venous Reserve
Volume of blood contained within systemic veins in the integument, bone marrow, and liver that can be returned to the heart for circulation, if needed.

Venule
Small vessel leading from the capillaries to veins.

Vertebral Artery
Arises from the subclavian artery and passes through the vertebral foramen through the foramen magnum to the brain; joins with the internal carotid artery to form the arterial circle; supplies blood to the brain and spinal cord.

Vertebral Vein
Arises from the base of the brain and the cervical region of the spinal cord; passes through the intervertebral foramina in the cervical vertebrae; drains smaller veins from the cranium, spinal cord, and vertebrae, and leads to the brachiocephalic vein; counterpart of the vertebral artery.

Visceral Branches
Branches of the descending aorta that supply blood to the viscera.

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7.8: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. The endothelium is found in the:
   A. Tunica intima
   B. Tunica media
   C. Tunica externa
   D. Lumen

2. Nervi vasorum control:
   A. Vasoconstriction
   B. Vasodilation
   C. Capillary permeability
   D. Both vasoconstriction and vasodilation

3. Closer to the heart, arteries would be expected to have a higher percentage of:
   A. Endothelium
   B. Smooth muscle fibers
   C. Elastic fibers
   D. Collagenous fibers

4. Which of the following best describes veins?
   A. Thick walled, small lumens, low pressure, lack valves
   B. Thin walled, large lumens, low pressure, have valves
   C. Thin walled, small lumens, high pressure, have valves
   D. Thick walled, large lumens, high pressure, lack valves

5. An especially leaky type of capillary found in the liver and certain other tissues is called a:
   A. Capillary bed
   B. Fenestrated capillary
   C. Sinusoid capillary
   D. Metarteriole

6. In a blood pressure measurement of 110/70, the number 70 is the:
   A. Systolic pressure
   B. Diastolic pressure
   C. Pulse pressure
   D. Mean arterial pressure

7. A healthy elastic artery:
   A. Is compliant
   B. Reduces blood flow
   C. Is a resistance artery
   D. Has a thin wall and irregular lumen

8. Which of the following statements is true about blood flow and resistance?
   A. The longer the vessel, the lower the resistance and the greater the flow
   B. As blood volume decreases, blood pressure and blood flow also decrease
   C. Increased viscosity increases blood flow
   D. All of the above are true

9. Slight vasodilation in an arteriole will cause a:
   A. Slight increase in resistance
   B. Huge increase in resistance
   C. Slight decrease in resistance
   D. Huge decrease in resistance

10. Venoconstriction increases which of the following?
    A. Blood pressure within the vein
    B. Blood flow within the vein
    C. Return of blood to the heart (venous return)
    D. All of the above

11. Hydrostatic pressure is:
    A. Greater than colloid osmotic pressure at the venous end of the capillary bed
    B. The pressure exerted by a fluid in an enclosed space
    C. About zero at the midpoint of a capillary bed
    D. All of the above

12. Net filtration pressure (NFP) across a capillary wall is calculated by:
    A. Adding the capillary hydrostatic pressure to the interstitial fluid hydrostatic pressure
    B. Subtracting the fluid drained by lymphatic vessels from the total interstitial fluid
    C. Adding the blood colloid osmotic pressure to the capillary hydrostatic pressure
    D. Subtracting the blood colloid osmotic pressure from the capillary hydrostatic pressure

13. Which of the following statements about fluid movement in capillary beds is true?
    A. In one day, more fluid exits the capillaries by filtration than enters by reabsorption
    B. In one day, approximately 35 mm of blood are filtered and 7 mm are reabsorbed
    C. In one day, the capillaries of the lymphatic system absorb about 20.4 liters of fluid
    D. None of the above are true

14. Clusters of neurons in the medulla oblongata that regulate blood pressure are known collectively as:
    A. Baroreceptors
    B. Angioreceptors
    C. The cardiomotor mechanism
    D. The cardiovascular center

15. In the renin–angiotensin–aldosterone mechanism:
    A. Decreased blood pressure prompts the release of renin from the liver
    B. Aldosterone prompts increased urine output
    C. Aldosterone prompts the kidneys to reabsorb sodium
    D. All of the above

16. In the myogenic response:
    A. Muscle contraction promotes venous return to the heart
    B. Ventricular contraction strength is decreased
    C. Vascular smooth muscle responds directly to stretch
    D. Endothelins dilate muscular arteries

17. A form of circulatory shock common in young children with severe diarrhea or vomiting is:
    A. Hypovolemic shock
    B. Anaphylactic shock
    C. Obstructive shock
    D. Hemorrhagic shock

18. The coronary arteries branch off of the:
    A. Aortic valve
    B. Ascending aorta
    C. Aortic arch
    D. Thoracic aorta

19. Which of the following statements about systemic arteries is true?
    A. The left and right common carotid arteries both branch off the brachiocephalic trunk
    B. The brachial artery is the distal continuation of the axillary artery
    C. The radial and ulnar arteries join to form the palmar arch
    D. All of the above are true

20. Arteries serving the stomach, pancreas, and liver all branch from the:
    A. Superior mesenteric artery
    B. Inferior mesenteric artery
    C. Celiac trunk
    D. Splenic artery

21. The right and left brachiocephalic veins:
    A. Drain blood from the right and left internal jugular veins
    B. Drain blood from the right and left subclavian veins
    C. Drain into the superior vena cava
    D. All of the above are true

22. The hepatic portal system delivers blood from the digestive organs to the:
    A. Liver
    B. Hypothalamus
    C. Spleen
    D. Left atrium

23. Blood islands are:
    A. Clusters of blood-filtering cells in the placenta
    B. Masses of pluripotent stem cells in fetal bone marrow
    C. Vascular tubes that give rise to the embryonic tubular heart
    D. Masses of developing blood vessels and formed elements in the embryonic disc

24. Which of the following statements about fetal circulation is true?
    A. Two umbilical veins carry oxygen-poor blood from the fetus to the placenta
    B. One umbilical vein carries oxygen-rich blood from the placenta to the fetal heart
    C. Two umbilical arteries carry oxygen-rich blood to the fetal lungs
    D. None of the above are true

25. The ductus venosus is a fetal shunt that allows:
    A. Blood to flow from the right atrium to the left atrium
    B. Blood to flow from the right ventricle to the left ventricle
    C. Most freshly oxygenated blood from the umbilical vein to flow into the fetal inferior vena cava and heart
    D. Most oxygen-poor fetal blood to flow directly into the pulmonary trunk

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7.9: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. Arteries and veins both carry blood, but their walls have different thickness and amounts of smooth muscle and elastic tissue. Explain how these structural differences relate to the different pressures and jobs of arteries and veins.
   Hint: Think about which vessels receive blood directly from the heart under high pressure, and which carry blood back at lower pressure (often against gravity).

2. Capillaries are the smallest blood vessels and have very thin walls. Explain why this thin structure is important for their function, and why capillaries are found in almost all tissues of the body.
   Hint: Think about exchange of gases, nutrients, and wastes between blood and cells, and how distance and wall thickness affect this.

3. When a person stands up quickly, blood tends to pool in the veins of the legs. Explain how the structure of veins and the action of skeletal muscles help return blood to the heart in this position.
   Hint: Consider venous valves, low pressure, and the “muscle pump” when leg muscles contract and relax.

4. Compare the systemic circulation and the pulmonary circulation in terms of pressure and resistance. Explain why the right ventricle can have a thinner wall than the left ventricle.
   Hint: Think about the distance and type of vessels blood travels through to the lungs vs. to the rest of the body.

5. A patient has an atherosclerotic plaque that narrows a coronary artery (an artery that supplies the heart muscle). Explain how this local narrowing can affect the entire circulatory system, especially during exercise.
   Hint: Consider blood flow to the myocardium, the heart’s ability to pump, and what happens to cardiac output if the heart muscle is short of oxygen.

6. Blood pressure is higher in arteries and much lower in veins. Describe one short-term mechanism the body can use to increase blood pressure if it suddenly drops (for example, due to blood loss).
   Hint: Think about changes in diameter of arterioles, heart rate, and the role of the sympathetic nervous system.

7. In the systemic circuit, blood flows from the left ventricle to the aorta and then through many arteries and arterioles before reaching capillary beds. Explain why branching into many smaller vessels is helpful, even though it slows the flow of blood.
   Hint: Consider total cross-sectional area, time for exchange, and distribution of blood to different organs.

8. The hepatic portal circulation takes blood from the digestive organs to the liver before it returns to the heart. Explain why this special pathway is important for maintaining homeostasis after you eat a meal.
   Hint: Think about nutrients, toxins, and the liver’s role in processing and storing substances absorbed from the intestine.

9. Long-term high blood pressure (hypertension) often damages small arteries and arterioles. Explain how this damage can lead to problems in organs such as the kidneys, brain, or eyes.
   Hint: Consider how thickening or narrowing of small vessels affects blood flow and oxygen delivery to sensitive tissues.

10. During exercise, blood flow to skeletal muscles increases, but blood flow to some other organs (such as the digestive system) decreases. Explain how changes in blood vessel diameter help redistribute blood, and why this redistribution is useful.
    Hint: Think about vasodilation and vasoconstriction in different vascular beds, and the body's priority to supply working muscles with oxygen.

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7.10: Alternate Text Descriptions

Figure 7.1.1: Pulmonary and Systemic Circulation Pathways

This diagram illustrates the movement of blood through the **pulmonary circulation** and **systemic circulation** and shows how the heart connects these two circuits. The pulmonary circulation carries blood between the heart and lungs for **gas exchange**, while the systemic circulation carries blood between the heart and the rest of the body to deliver **oxygen** and **nutrients** and collect wastes.

Pulmonary Circulation

1. Blood returning from the body enters the **right side of the heart** through the **vena cava**.
2. The right side of the heart pumps **oxygen-poor blood** through the **pulmonary arteries** to the lungs.
3. Within the **pulmonary capillary network**, gas exchange occurs as **carbon dioxide** leaves the blood and **oxygen** enters it.
4. **Oxygen-rich blood** then returns to the **left side of the heart** through the **pulmonary veins**.

Systemic Circulation

1. The left side of the heart pumps **oxygen-rich blood** into the **aorta**.
2. The aorta distributes blood throughout the **systemic circulation**.
3. Arterial blood travels to capillary networks in the:
    - **Upper body**
    - **Liver**
    - **Digestive organs**
    - **Kidneys**
    - **Lower body**
4. In these capillary networks, oxygen and nutrients are exchanged with tissues.
5. Blood then returns through **systemic veins** to the **vena cava** and reenters the right side of the heart, completing the cycle.

Hepatic Portal System

1. The diagram also highlights the **hepatic portal system**.
2. Blood from the **stomach** and **intestines** first travels through the **hepatic portal vein** to the **liver**.
3. Blood then returns to the systemic venous circulation through the **hepatic vein**.
4. The liver additionally receives oxygenated blood through the **hepatic artery**.

Instructional purpose is to demonstrate the pathways of **pulmonary** and **systemic circulation**, show how blood moves through the **heart**, **lungs**, and **body** in a continuous circuit, and illustrate the special role of the **hepatic portal circulation** in directing blood from the digestive organs to the liver before it returns to the heart.

Figure 7.1.2: Structural Organization of Arteries and Veins

This figure compares the structure of an **artery** and a **vein** and shows how differences in their wall architecture relate to their functions in the circulatory system. The figure includes schematic cross-sections of an artery and a vein and a histological image showing both vessel types in tissue.

Panel (a): Artery

1. A cross-section of an **artery** is shown.
2. The arterial wall consists of three layers:
    - **Tunica intima**
    - **Tunica media**

- **Tunica externa**

3. The **tunica media** is the thickest layer and contains abundant **smooth muscle** and **elastic fibers**.
4. **Internal** and **external elastic membranes** separate the tunica media from adjacent layers.
5. Small blood vessels called **vasa vasorum** and nerves called **nervi vasorum** supply and innervate the outer vessel wall.
6. The thick muscular and elastic wall helps arteries withstand and regulate the **high pressures** generated by cardiac contraction.

Panel (b): Vein

1. A cross-section of a **vein** is shown.
2. Veins also contain a:
   - **Tunica intima**
   - **Tunica media**
   - **Tunica externa**
3. The **tunica media** is thinner and contains less **smooth muscle** and **elastic tissue** than that of arteries.
4. **Vasa vasorum** and **nervi vasorum** are present in the outer wall.
5. The thinner wall reflects the **lower pressures** within the venous system and allows veins to serve as vessels that return blood to the heart.

Panel (c): Histological Comparison

1. A histological section shows an **artery** and a **vein** within the same tissue sample.
2. The artery has a **thicker wall** and a **smaller, more circular lumen**.
3. The vein has a **thinner wall** and a **larger, more irregularly shaped lumen**.
4. These structural differences distinguish arteries from veins in microscopic sections.

The figure demonstrates that arteries and veins share a common **three-layer wall organization** but differ in the relative thickness of those layers. **Arteries** have thicker muscular and elastic walls adapted for **higher pressures**, while **veins** have thinner walls and larger lumens adapted for **lower-pressure blood return**.

Instructional purpose is to compare the structural organization of **arteries** and **veins** and demonstrate how differences in **wall thickness**, **elastic tissue**, **smooth muscle**, and **lumen shape** support their distinct roles in transporting blood through the circulatory system.

Figure 7.1.3: Comparison of Elastic Arteries, Muscular Arteries, and Arterioles

This figure compares the structure of three types of arteries: an **elastic artery**, a **muscular artery**, and an **arteriole**. Each vessel is shown in cross section with its wall layers labeled to illustrate how vessel structure changes as arteries branch and decrease in diameter.

Shared Structural Features

1. All three vessel types contain the same three basic layers:
   - **Tunica intima**, lining the lumen
   - **Tunica media**, composed primarily of smooth muscle and elastic tissue
   - **Tunica externa**, the outer connective tissue layer
2. However, the relative thickness and composition of these layers differ among vessel types.

Elastic Artery

1. The **elastic artery** has a thick wall with a particularly thick **tunica media** containing numerous **elastic fibers** arranged throughout the vessel wall.
2. This abundance of elastic tissue allows the vessel to **expand** when blood is ejected from the heart and **recoil** as blood moves through the circulation.

3. This helps maintain blood flow and pressure between heartbeats.

Muscular Artery

1. The **muscular artery** has a prominent **tunica media** dominated by **smooth muscle** and fewer elastic fibers than the elastic artery.
2. Smooth muscle makes up most of the vessel wall.
3. This allows the artery to regulate blood distribution by changing its diameter through **vasoconstriction** and **vasodilation**.

Arteriole

1. The **arteriole** is substantially smaller and has a much thinner wall.
2. Its **tunica media** consists of only a few layers of **smooth muscle** surrounding a relatively small lumen.
3. Although simple in structure, arterioles are the primary **resistance vessels** of the circulatory system.
4. They play a major role in controlling blood flow into **capillary beds** and regulating **systemic blood pressure**.

The figure demonstrates the gradual transition from large **elastic arteries** specialized for **pressure buffering**, to **muscular arteries** specialized for **blood distribution**, to **arterioles** specialized for regulating **vascular resistance** and **tissue perfusion**.

Instructional purpose is to compare the structural organization of **elastic arteries**, **muscular arteries**, and **arterioles** and demonstrate how differences in the **tunica media**, **elastic tissue**, and **vessel size** relate to their roles in maintaining blood flow, distributing blood, and regulating vascular resistance.

Figure 7.1.4: Structural Types of Capillaries

This figure compares the three major types of **capillaries**: **continuous capillaries**, **fenestrated capillaries**, and **sinusoids**, also called **discontinuous capillaries**. The diagrams illustrate differences in **endothelial structure**, **basement membrane continuity**, and **permeability**.

Continuous Capillary

1. The **continuous capillary** has a continuous **endothelial lining** and a continuous **basement membrane**.
2. Small spaces called **intercellular clefts** are present between adjacent endothelial cells.
3. These clefts allow limited movement of **water** and **small solutes**.
4. This structure creates the **least permeable** capillary type and provides the greatest control over substance exchange between blood and surrounding tissues.

Fenestrated Capillary

1. The **fenestrated capillary** contains pores, or **fenestrations**, within the endothelial cells.
2. It still maintains a continuous **basement membrane**.
3. These openings increase permeability and allow more rapid exchange of water and certain dissolved substances between the blood and surrounding tissues than occurs in continuous capillaries.

Sinusoid

1. The **sinusoid**, or **discontinuous capillary**, has large **intercellular gaps** between endothelial cells.
2. It also has an incomplete or discontinuous **basement membrane**.
3. The **lumen** is wider and more irregular than in the other capillary types.
4. These structural features make sinusoids the **most permeable** capillaries.
5. They permit the passage of **larger molecules** and, in some locations, even **cells** between the blood and surrounding tissues.

The figure demonstrates a progression from **continuous capillaries**, which provide the greatest barrier to exchange, to **fenestrated capillaries**, which permit increased exchange, and finally to **sinusoids**, which allow extensive movement of substances across the vessel wall. The differences in structure reflect each capillary type's specialized role in regulating exchange

between the bloodstream and body tissues.

Instructional purpose is to compare the structural features of **continuous**, **fenestrated**, and **sinusoidal capillaries** and demonstrate how differences in **endothelial openings** and **basement membrane integrity** affect capillary permeability and the exchange of materials between blood and tissues.

Figure 7.1.5: Structure and Regulation of a Capillary Bed

This figure illustrates the organization of a **capillary bed** and the pathways through which blood moves from an **arteriole** to a **venule**. It highlights the roles of **metarterioles**, **capillaries**, **precapillary sphincters**, **thoroughfare channels**, and **arteriovenous anastomoses** in regulating blood flow through tissues.

Blood Flow Through the Capillary Bed

1. Blood enters the capillary bed from an **arteriole**, which gives rise to a **metarteriole**.
2. Branching from the metarteriole are numerous **capillaries** that form an interconnected capillary bed.
3. At the entrance to each capillary is a ring of smooth muscle called a **precapillary sphincter**.
4. These sphincters regulate blood flow into individual capillaries by opening or closing in response to tissue needs.

Open and Closed Precapillary Sphincters

1. When precapillary sphincters are **open**, blood flows through the capillary network.
2. This allows exchange of **gases**, **nutrients**, **wastes**, and other substances between the blood and surrounding tissues.
3. When precapillary sphincters are **closed**, blood bypasses most capillaries and continues through the **metarteriole** into a **thoroughfare channel**.
4. The thoroughfare channel connects directly to the **venule**.
5. In this situation, the metarteriole functions as a **vascular shunt** that reduces blood flow through the exchange vessels.

Arteriovenous Anastomosis

1. The figure also shows an **arteriovenous anastomosis**, a direct connection between an **arteriole** and a **venule**.
2. This pathway bypasses the capillary bed entirely.
3. It can rapidly redirect blood flow and contributes to the regulation of **tissue perfusion** and **heat exchange** in certain regions of the body.

The diagram demonstrates that blood flow through **capillary beds** is not constant but is regulated by **precapillary sphincters** and **vascular shunts**. These mechanisms allow tissues to receive blood according to their changing **metabolic demands**.

Instructional purpose is to illustrate the structural components of a **capillary bed** and demonstrate how **precapillary sphincters**, **metarterioles**, **thoroughfare channels**, and **arteriovenous anastomoses** regulate blood flow and capillary exchange within tissues.

Figure 7.1.6: Structural Organization of Veins and Venules

This figure compares the wall structure of a **large vein**, a **medium-sized vein**, and a **venule**, highlighting differences in vessel wall composition and the presence of **venous valves**.

Large Vein

1. The **large vein** has three distinct layers:
   - **Tunica intima**
   - **Tunica media**
   - **Tunica externa**
2. The **tunica externa** is the thickest layer.
3. It contains **smooth muscle cells**, small blood vessels called **vasa vasorum**, and nerves called **nervi vasorum** that supply

and regulate the vessel wall.
4. In contrast, the **tunica media** is relatively thin compared with that of arteries.

Medium-Sized Vein

1. The **medium-sized vein** also contains the three vascular layers.
2. It is characterized by the presence of **venous valves**, which are folds of the **tunica intima** that project into the lumen.
3. The enlarged inset shows a valve in the **closed position**.
4. These valves help prevent **backflow of blood** and promote **one-way movement** of blood toward the heart, particularly in regions where blood must travel against gravity.

Venule

1. The **venule** has a much thinner wall than either type of vein.
2. Its **tunica intima**, **tunica media**, and **tunica externa** are only minimally developed.
3. This reflects its role as a **low-pressure vessel** that collects blood from **capillary beds** and returns it to progressively larger veins.

The figure demonstrates that **veins** and **venules** share the same basic **three-layer organization** found throughout the vascular system but differ in **wall thickness** and **specialization**. **Large veins** possess a prominent **tunica externa**, **medium-sized veins** contain valves that aid **venous return**, and **venules** have thin walls adapted for collecting blood from capillary networks.

Instructional purpose is to compare the structural organization of **large veins**, **medium-sized veins**, and **venules** and to illustrate how features such as a thick **tunica externa** and **venous valves** support the return of low-pressure blood to the heart.

Figure 7.1.7: Varicose Veins of the Lower Limb

This clinical photograph shows the lower limb of an individual with prominent **varicose veins**.

Visible Features

1. Multiple enlarged, tortuous **superficial veins** are visible beneath the skin along the **calf** and **lower leg**.
2. The affected veins appear **bulging** and **irregular**, forming raised, winding structures that protrude from the surface of the leg.
3. The most prominent dilated veins are located along the **medial** and **posterior** aspects of the calf.
4. Several interconnected venous enlargements create a **rope-like appearance**.
5. The skin overlying the veins remains **intact**, and body hair is visible across the leg.
6. No anatomical labels are present in the image.

The photograph demonstrates the characteristic appearance of **varicose veins**, which develop when **venous valves** become ineffective and allow blood to accumulate within superficial veins. As pressure increases within the vessel, the vein enlarges, elongates, and becomes visibly twisted beneath the skin.

Instructional purpose is to illustrate the gross appearance of **varicose veins** and demonstrate how **venous valve dysfunction** can lead to **dilation**, **tortuosity**, and **pooling of blood** within superficial veins of the lower limb.

Figure 7.2.1: Changes in Blood Pressure and Pulse Pressure Throughout the Circulatory System

This graph illustrates how **blood pressure** changes as blood moves through different types of blood vessels, from the **aorta** to the **venae cavae**. The horizontal axis represents the major segments of the vascular system, including the **aorta**, **elastic arteries**, **muscular arteries**, **arterioles**, **capillaries**, **venules**, **medium and large veins**, and **venae cavae**. The vertical axis shows pressure in **millimeters of mercury, or mm Hg**.

Pressure Patterns in the Arterial System

1. Three pressure measurements are displayed in the arterial system:
   - **Systolic pressure**
   - **Diastolic pressure**
   - **Mean arterial pressure**
2. A red pulsating waveform represents the pressure fluctuations produced by each heartbeat.
3. In the **aorta** and large arteries, pressure oscillates between systolic and diastolic values, creating a substantial **pulse pressure**.
4. As blood moves through **elastic** and **muscular arteries**, the pulsations gradually decrease in amplitude.

Pressure Drop Across the Arterioles

1. The greatest decline in pressure occurs across the **arterioles**, where **vascular resistance** is highest.
2. In this region, **systolic**, **diastolic**, and **mean arterial pressures** all decrease rapidly.
3. The pulsatile nature of blood flow is also greatly reduced.

Pressure in Capillaries and Veins

1. By the time blood reaches the **capillaries**, pressure is much lower and pulsations are essentially absent.
2. Pressure continues to decline through the **venules** and **veins**.
3. It approaches **zero** in the **venae cavae** as blood returns to the heart.

The figure demonstrates two important principles of **vascular hemodynamics**:

1. **Arterial pressure** steadily decreases as blood travels farther from the heart.
2. **Pulse pressure** is progressively dampened so that blood flow in capillaries becomes relatively continuous rather than strongly pulsatile.

The graph also highlights the critical role of **arterioles** as the primary site of **resistance** and **pressure reduction** within the systemic circulation.

Instructional purpose is to illustrate how **systolic**, **diastolic**, **mean arterial**, and **pulse pressures** change throughout the vascular system and to demonstrate that the largest pressure drop occurs in the **arterioles** while pulsatile flow is progressively reduced before blood reaches the **capillaries**.

Figure 7.2.2: Major Arterial Pulse Palpation Points

This black-and-white medical line drawing shows the primary **arterial pulse palpation points** on the human body. The figure presents a simplified **front view of a standing woman**. Solid black circles mark locations where a pulse can be measured. Horizontal lines extend from each marked point to labels on the left side of the diagram.

The labeled pulse sites, from head to foot, are:

- **Temporal artery**: on the side of the head at the temple, above and slightly outside the eyebrow
- **Facial artery**: on the lower face along the jawline, near the corner of the mouth
- **Common carotid artery**: on the side of the neck, below the jawline and near the trachea
- **Brachial artery**: at the inner elbow, in the antecubital region
- **Radial artery**: at the wrist on the thumb side
- **Femoral artery**: in the groin, where the thigh meets the lower torso
- **Popliteal artery**: at the knee region, shown near the medial or posterior aspect of the joint
- **Posterior tibial artery**: on the inner side of the ankle, just behind the medial malleolus
- **Dorsalis pedis artery**: on the top of the foot, near the midline between the first and second toes

The diagram identifies the major anatomical sites where arterial pulses can be palpated. It helps learners locate common pulse points used in health assessment and clinical practice.

Figure 7.2.3: Measurement of Systolic and Diastolic Blood Pressure Using Korotkoff Sounds

This figure illustrates how **systolic** and **diastolic blood pressures** are determined during manual blood pressure measurement using an inflatable **cuff** and a **stethoscope**. The graph plots **pressure**, measured in **millimeters of mercury, or mm Hg**, on the vertical axis and **time** on the horizontal axis as cuff pressure is gradually released.

Graph Features

1. The **red waveform** represents pulsatile **arterial blood pressure** generated by the cardiac cycle.
2. A **gray line** represents **cuff pressure**, which steadily decreases over time as air is released from the cuff.
3. At the bottom of the figure, a tracing shows the appearance and disappearance of **Korotkoff sounds** heard through the stethoscope.

Sequence of Blood Pressure Measurement

1. Initially, cuff pressure exceeds **systolic arterial pressure**, completely occluding the artery and preventing blood flow.
2. At this stage, no **Korotkoff sounds** are heard.
3. As cuff pressure falls to the level of systolic arterial pressure, blood begins to pass through the partially compressed artery during **systole**.
4. The **first Korotkoff sound** becomes audible, marking the measurement of **systolic blood pressure**.
5. As cuff pressure continues to decrease between systolic and diastolic pressures, blood flow through the partially compressed artery becomes **turbulent**.
6. This turbulent flow produces repeated **Korotkoff sounds** that are heard with each heartbeat.
7. When cuff pressure falls below **diastolic arterial pressure**, the artery remains open throughout the cardiac cycle and blood flow becomes **smooth**.
8. The **Korotkoff sounds disappear**, and the **last sound heard** marks the measurement of **diastolic blood pressure**.

The figure demonstrates that **systolic pressure** corresponds to the cuff pressure at which blood flow first becomes detectable, whereas **diastolic pressure** corresponds to the cuff pressure at which turbulent flow and **Korotkoff sounds** disappear.

Instructional purpose is to demonstrate how **systolic** and **diastolic blood pressures** are determined during **auscultatory blood pressure measurement** and to explain the relationship among **cuff pressure**, **arterial pressure**, and **Korotkoff sounds**.

Figure 7.2.4: Changes in Vessel Diameter, Cross-Sectional Area, Blood Pressure, and Blood Flow Velocity Across the Vascular System

This figure presents four graphs comparing how **vessel diameter**, **total cross-sectional area**, **average blood pressure**, and **blood flow velocity** change as blood moves through the systemic circulation from **elastic arteries** to the **venae cavae**.

Panel (a): Vessel Diameter

1. The graph shows that **vessel diameter** is greatest in the large **elastic arteries**.
2. Diameter progressively decreases through **muscular arteries** and **arterioles**.
3. It reaches its minimum in the **capillaries**.
4. Diameter then increases again through **venules** and **veins**.
5. It becomes largest in the **venae cavae** as blood returns to the heart.

Panel (b): Total Cross-Sectional Area of Vessels

1. The graph shows that total **cross-sectional area** increases dramatically from arteries to capillaries.
2. This occurs because blood is distributed among a very large number of capillaries.
3. Cross-sectional area reaches its maximum within the **capillary beds**.
4. It then decreases through **venules** and **veins** as vessels merge into progressively larger channels.

Panel (c): Average Blood Pressure

1. The graph demonstrates a continuous decline in **blood pressure** throughout the vascular system.
2. Pressure is highest in the **elastic arteries** near the heart and decreases gradually through arteries.
3. The most substantial drop occurs across the **arterioles**, reflecting their role as the major **resistance vessels**.
4. Pressure continues to decrease through **capillaries**, **venules**, and **veins**.
5. It reaches its lowest values in the **venae cavae**.

Panel (d): Velocity of Blood Flow

1. The graph shows that **blood flow velocity** is highest in the large arteries near the heart.
2. As blood enters smaller vessels and the total cross-sectional area increases, velocity decreases.
3. Flow is slowest within the **capillary beds**, where reduced velocity supports efficient exchange of **gases**, **nutrients**, and **wastes** between blood and tissues.
4. Velocity then increases again through **venules** and **veins** as total cross-sectional area decreases and blood returns to the heart.

Taken together, the four graphs demonstrate the relationship between **vessel structure** and **blood flow dynamics**. The extensive **cross-sectional area** of capillary networks slows blood flow and facilitates exchange, while **arterioles** produce the greatest **resistance** and **pressure drop** within the circulation.

Instructional purpose is to compare changes in **vessel diameter**, **total cross-sectional area**, **blood pressure**, and **blood flow velocity** throughout the vascular system and demonstrate how these variables contribute to efficient **blood distribution**, **capillary exchange**, and **venous return**.

Figure 7.2.5: Atherosclerosis and Plaque Formation in an Artery

This figure compares a **normal artery** with an artery narrowed by **atherosclerotic plaque** and includes a histological image of plaque within an arterial wall.

Panel (a): Normal and Atherosclerotic Artery

1. The upper illustration shows a **normal artery** with a wide, unobstructed **lumen** that allows blood to flow freely.
2. A cross-sectional view demonstrates the circular lumen surrounded by the arterial wall.
3. The lower illustration shows an artery affected by **atherosclerosis**.
4. A **plaque deposit** has accumulated within the arterial wall and projects into the lumen, narrowing the passageway available for blood flow.
5. The reduction in lumen diameter disrupts normal blood flow and increases resistance to circulation.
6. A corresponding cross-sectional view shows the plaque occupying part of the lumen, leaving a substantially smaller opening for blood to pass through.

Panel (b): Histological View of Plaque

1. A histological section of an artery shows an **atherosclerotic plaque** within the vessel wall.
2. The plaque appears as a thickened region projecting inward toward the lumen.
3. The image demonstrates how plaque accumulation can distort the normal structure of the arterial wall and reduce the size of the vessel lumen.

Together, the panels show that **atherosclerosis** involves the buildup of **plaque** within arterial walls, leading to progressive narrowing of the vessel. As the lumen becomes smaller, blood flow may become restricted, reducing the delivery of **oxygen** and **nutrients** to downstream tissues.

Instructional purpose is to illustrate the development of **atherosclerotic plaque** within an artery and demonstrate how plaque accumulation narrows the **vessel lumen**, alters **blood flow**, and increases **resistance** within the cardiovascular system.

Figure 7.2.6: Skeletal Muscle Pump and Venous Valves in Venous Return

This figure illustrates how contractions of surrounding **skeletal muscles** assist the movement of blood through **veins** and how **venous valves** prevent backflow.

Left Panel: Relaxed Skeletal Muscles

1. The left panel shows a vein located between **relaxed skeletal muscles**.
2. Venous valves above and below a segment of blood are **closed**, preventing backward movement of blood within the vessel.
3. In this resting state, blood movement toward the heart is limited because the surrounding muscles are not compressing the vein.

Right Panel: Contracting Skeletal Muscles

1. The right panel shows the same vein during **skeletal muscle contraction**.
2. As the surrounding muscles compress the vein, pressure within the vessel increases and pushes blood upward toward the heart.
3. The valve above the compressed segment **opens**, allowing blood to move forward.
4. The valve below remains **closed**, preventing backflow.
5. An arrow indicates the direction of blood movement through the open valve.

Together, the panels demonstrate the **skeletal muscle pump**, a mechanism that promotes **venous return** by intermittently compressing veins during muscle activity. The figure emphasizes that **venous valves** ensure **one-way blood flow** toward the heart by opening in response to forward-moving blood and closing when blood attempts to flow backward.

Instructional purpose is to illustrate how **skeletal muscle contractions** and **venous valves** work together to facilitate **venous return** and maintain one-way blood flow toward the heart, particularly in the limbs where blood must travel against gravity.

Figure 7.3.1: Capillary Filtration and Reabsorption Driven by Hydrostatic and Osmotic Pressures

This figure illustrates the movement of fluid across a **capillary wall** as blood travels from the **arterial end** to the **venous end** of a capillary. The diagram demonstrates how the balance between **capillary hydrostatic pressure** and **blood colloid osmotic pressure** determines whether fluid leaves or enters the capillary.

Arterial End of the Capillary

1. At the **arterial end** of the capillary, **capillary hydrostatic pressure** is **35 mm Hg**.
2. **Blood colloid osmotic pressure** is **25 mm Hg**.
3. Because hydrostatic pressure is greater than osmotic pressure, there is a **net filtration pressure of +10 mm Hg**.
4. As a result, fluid moves **out of the capillary** and into the surrounding tissues.
5. An outward-pointing arrow represents this **filtration** process.

Midpoint of the Capillary

1. At the **midpoint** of the capillary, capillary hydrostatic pressure has decreased to **25 mm Hg**.
2. This matches the blood colloid osmotic pressure of **25 mm Hg**.
3. Because the opposing pressures are equal, the **net filtration pressure is 0 mm Hg**.
4. There is no net movement of fluid across the capillary wall.

Venous End of the Capillary

1. At the **venous end** of the capillary, capillary hydrostatic pressure has fallen to **18 mm Hg**.
2. **Blood colloid osmotic pressure** remains **25 mm Hg**.

3. Osmotic pressure now exceeds hydrostatic pressure, producing a **net filtration pressure of –7 mm Hg**.
4. As a result, fluid moves from the surrounding tissues **back into the capillary**.
5. An inward-pointing arrow represents this **reabsorption** process.

The figure demonstrates that **capillary hydrostatic pressure** decreases from the arterial end to the venous end of the capillary, whereas **blood colloid osmotic pressure** remains relatively constant. This shift in the balance of forces causes **filtration** to predominate at the arterial end and **reabsorption** to predominate at the venous end.

Instructional purpose is to illustrate how **hydrostatic** and **osmotic pressures** regulate fluid exchange across capillary walls and to demonstrate why fluid typically leaves the capillary at the arterial end and reenters at the venous end.

Figure 7.5.1: Interactions Between the Circulatory System and Other Body Systems

This table summarizes how the **circulatory system** interacts with and supports multiple **organ systems** throughout the body. The left column identifies each body system, and the right column describes the circulatory system's role in maintaining the functions of that system.

Body System Interactions

1. Digestive System
   - The circulatory system absorbs and transports **water** and **nutrients** from the digestive tract.
   - Nutrients are delivered to the **liver** through the **hepatic portal circulation** for processing and distribution.
   - These nutrients support **tissue growth**, **maintenance**, and **hemoglobin production**.
2. Endocrine System
   - The circulatory system transports **hormones** throughout the body.
   - These include hormones produced by the **heart**, **adrenal glands**, **thyroid gland**, and other endocrine organs.
   - These hormones help regulate **blood volume**, **blood pressure**, **metabolism**, and other physiological processes.
3. Integumentary System
   - The circulatory system transports **platelets**, **clotting factors**, and **immune cells** involved in wound repair and defense.
   - Blood flow to the skin helps regulate **body temperature**.
   - Blood flow also contributes to **skin coloration**.
4. Lymphatic System
   - The circulatory system works with the **lymphatic system** to transport **immune cells** and **antibodies**.
   - Excess fluid that leaves blood vessels and enters tissues is collected by **lymphatic vessels**.
   - This fluid is eventually returned to the circulatory system.
5. Muscular System
   - The circulatory system delivers **oxygen** and **nutrients** needed for muscle contraction.
   - It removes **metabolic wastes** produced during activity.
   - Blood flow helps distribute **heat** generated by muscle activity.
   - Muscle contractions also assist **venous return** to the heart.
6. Nervous System
   - The circulatory system supplies **oxygen** and **nutrients** to nervous tissue.
   - It supports the **blood-brain barrier**.
   - Neural control centers regulate **cardiac output** and **blood vessel diameter** through **autonomic nervous system** activity.
7. Reproductive System
   - The circulatory system supplies blood flow necessary for **reproductive function**.
   - It also transports hormones that regulate reproductive processes.
8. Respiratory System
   - The circulatory system transports **oxygen** from the lungs to body tissues.
   - It returns **carbon dioxide** to the lungs for removal.
   - Together, the respiratory and circulatory systems support **gas exchange** required for cellular metabolism.
9. Skeletal System
   - The circulatory system transports **minerals**, **hormones**, and other substances involved in bone growth and maintenance.
   - **Bone marrow** contributes to **blood cell production**.

- Skeletal structures provide protection for some blood vessels.

10. Urinary System
    - The circulatory system delivers blood to the **kidneys** for filtration and removal of wastes.
    - The kidneys help regulate **blood volume** and **blood pressure**.
    - They also produce signals that influence **blood cell production** and **cardiovascular function**.

The figure demonstrates that the **circulatory system** supports all major body systems by transporting **gases**, **nutrients**, **hormones**, **wastes**, **immune components**, and **heat**. These interactions help maintain **homeostasis** throughout the body.

Instructional purpose is to summarize the functional relationships between the **circulatory system** and other body systems and demonstrate how blood transport supports **nutrient delivery**, **gas exchange**, **waste removal**, **hormone distribution**, **immune defense**, **temperature regulation**, and overall **homeostasis**.

Figure 7.5.2: Pulmonary Circulation and Gas Exchange in the Lungs

This figure illustrates the pathway of **blood flow** between the **heart** and **lungs** and highlights the role of **pulmonary circulation** in **gas exchange**. The heart is shown centrally between the right and left lungs, with arrows indicating the direction of blood flow through the pulmonary vessels.

Blood Flow to the Lungs

1. **Oxygen-poor blood** returns to the **right side of the heart** through the **superior vena cava** and **inferior vena cava**.
2. From the **right ventricle**, blood is pumped into the **pulmonary trunk**.
3. The pulmonary trunk divides into the **right pulmonary artery** and **left pulmonary artery**.
4. These arteries carry oxygen-poor blood from the heart to the lungs.

Gas Exchange in the Lungs

1. Within the lungs, the pulmonary arteries branch into progressively smaller vessels.
2. These vessels terminate in **pulmonary capillary networks** surrounding the **alveoli**.
3. An inset highlights these pulmonary capillaries, where **gas exchange** occurs.
4. In this region, **carbon dioxide** diffuses from the blood into the lungs to be exhaled.
5. At the same time, **oxygen** diffuses from the lungs into the blood.

Blood Flow Back to the Heart

1. After gas exchange, **oxygen-rich blood** returns to the heart through the **right pulmonary vein** and **left pulmonary vein**.
2. These veins empty into the **left atrium**, completing the pulmonary circuit.
3. Blood is then pumped from the **left ventricle** into the **ascending aorta**, through the **aortic arch**, and into the **descending aorta** for distribution to the **systemic circulation**.

The figure emphasizes that, unlike most arteries and veins in the body, **pulmonary arteries** carry **oxygen-poor blood** to the lungs and **pulmonary veins** carry **oxygen-rich blood** back to the heart. Together, the **heart**, **pulmonary vessels**, **lungs**, and **pulmonary capillaries** form the pulmonary circulation responsible for **oxygenating blood** and removing **carbon dioxide**.

Instructional purpose is to illustrate the flow of blood through the **pulmonary circulation** and demonstrate how blood travels between the **heart** and **lungs** for gas exchange before returning to the heart for distribution to the rest of the body.

Figure 7.5.3: Major Arteries of the Systemic Circulation

This figure presents an anterior view of the body's major systemic arteries. The arterial system is shown branching from the aorta and distributing oxygenated blood from the heart to the head, neck, upper limbs, thoracic organs, abdominal organs, pelvis, and lower limbs.

At the center of the figure, the **ascending aorta** emerges from the heart and gives rise to the **aortic arch**. Major branches of the

aortic arch include the **brachiocephalic trunk**, **right common carotid artery**, **left common carotid artery**, **right subclavian artery**, and **left subclavian artery**, which supply the head, neck, and upper extremities. The **vertebral arteries** ascend through the neck and contribute to blood flow to the brain.

The aorta continues inferiorly as the **descending aorta**, which gives rise to branches that supply thoracic and abdominal organs. Abdominal branches shown include the **celiac trunk**, **superior mesenteric artery**, **renal arteries**, **gonadal arteries**, and **inferior mesenteric artery**.

Arteries of the upper limbs are illustrated as a continuous pathway from the **axillary artery** to the **brachial artery**, which divides into the **radial** and **ulnar arteries**. These vessels contribute to the **palmar arches** of the hand.

Near the pelvis, the descending aorta divides into the **common iliac arteries**, which branch into the **internal iliac arteries** supplying pelvic structures and the **external iliac arteries** supplying the lower limbs. In the thigh, the **femoral artery** and **deep femoral artery** provide blood to the lower extremity. Distally, blood continues through the **popliteal artery** behind the knee and into the **anterior tibial**, **posterior tibial**, and **fibular arteries** of the leg. Arterial blood reaches the foot through branches that contribute to the **dorsalis pedis artery** and **plantar arch**.

The figure emphasizes the hierarchical branching pattern of the arterial system, beginning with the aorta and progressively dividing into smaller arteries that distribute oxygenated blood throughout the body.

Instructional purpose is to identify the major arteries of the systemic circulation and demonstrate how oxygenated blood is distributed from the aorta to the organs, upper limbs, pelvis, and lower limbs through an organized branching network of arteries.

Figure 7.5.4: Major Branches of the Aorta and Aortic Arch

This figure illustrates the major regions of the aorta and its primary branches as blood leaves the left ventricle and enters the systemic circulation. The diagram focuses on the aortic arch and the vessels that supply the head, neck, upper limbs, and body.

The **ascending aorta** emerges from the heart and gives rise to the **right coronary artery** and **left coronary artery**, which supply blood to the myocardium. The ascending aorta then curves superiorly to form the **aortic arch**.

Three major branches arise from the aortic arch. The first branch is the **brachiocephalic trunk**, which divides into the **right common carotid artery** and **right subclavian artery**. The second branch is the **left common carotid artery**, and the third is the **left subclavian artery**. Together, these vessels supply oxygenated blood to the head, neck, and upper extremities.

Beyond the arch, the aorta continues inferiorly as the **descending aorta**. Within the thorax, this vessel is referred to as the **thoracic aorta**. After passing through the diaphragm, it becomes the **abdominal aorta**, which carries blood to abdominal organs, the pelvis, and the lower limbs.

The figure demonstrates the continuity of the aorta from its origin at the heart through its major regions and highlights the branching pattern that distributes oxygenated blood to the upper body before continuing to the rest of the systemic circulation.

Instructional purpose is to identify the major regions of the aorta and the principal branches of the aortic arch and demonstrate how oxygenated blood is distributed from the heart to the head, neck, upper limbs, and the remainder of the body through the systemic arterial system.

Figure 7.5.5: Major Arteries of the Head and Neck

This figure illustrates the major **arteries** that supply the **head**, **neck**, **face**, and **brain**. A lateral view of the head and neck shows the branching pattern of the **common carotid**, **external carotid**, **internal carotid**, and **vertebral arteries**.

Common Carotid Artery

1. The **common carotid artery** ascends through the neck.
2. It divides into the **internal carotid artery** and **external carotid artery** near the **carotid sinus**.
3. The **carotid sinus** is a widened region at the bifurcation that contains receptors involved in monitoring **blood pressure**.

Internal Carotid and Vertebral Arteries

1. The **internal carotid artery** travels into the skull.
2. It contributes to the arterial supply of the **brain**.
3. The **vertebral artery** ascends through openings in the **cervical vertebrae**.
4. It also supplies blood to the **brain**.
5. Together, the **internal carotid** and **vertebral arteries** provide the major arterial blood supply to the **central nervous system**.

External Carotid Artery and Its Branches

1. The **external carotid artery** gives rise to branches that supply structures of the **face**, **scalp**, and **neck**.
2. Branches shown include:
    - **Facial artery**, which supplies facial tissues
    - **Lingual artery**, which supplies the tongue
    - **Maxillary artery**, which supplies deep facial structures
    - **Superficial temporal artery**, which supplies the scalp in the temporal region
    - **Occipital artery**, which supplies the posterior scalp

The figure demonstrates the division of blood flow from the **common carotid artery** into separate pathways serving the **brain** and the superficial structures of the **head** and **neck**. It also highlights the contribution of the **vertebral arteries** to **cerebral circulation**.

Instructional purpose is to identify the major arteries of the **head and neck** and demonstrate how the **common carotid arteries** branch into **internal** and **external carotid arteries** that supply the **brain**, **face**, **scalp**, and **neck**, while the **vertebral arteries** provide an additional route of blood supply to the brain.

Figure 7.5.6: Circle of Willis and Major Arteries Supplying the Brain

This figure shows the arterial circulation at the base of the **brain**, highlighting the **Circle of Willis**, an interconnected network of arteries that links blood flow from the **internal carotid arteries** and **vertebral arteries**. The inferior, or underside, view of the brain illustrates how multiple arterial pathways provide blood to the **cerebrum** and other brain structures.

Vertebral and Basilar Arteries

1. The **right vertebral artery** and **left vertebral artery** ascend along the **brainstem**.
2. They merge to form the **basilar artery**.
3. The basilar artery travels along the ventral surface of the brainstem.
4. It divides into the **right posterior cerebral artery** and **left posterior cerebral artery**.
5. These arteries supply posterior regions of the brain.

Internal Carotid Arteries

1. The **right internal carotid artery** and **left internal carotid artery** enter the cranial cavity.
2. They contribute to the anterior circulation of the brain.
3. Each internal carotid artery gives rise to an **ophthalmic artery**, which supplies structures associated with the **orbit**.
4. Each internal carotid artery then continues into the cerebral circulation.

Anterior and Middle Cerebral Arteries

1. The **anterior cerebral arteries** extend toward the midline.
2. They are connected by the **anterior communicating artery**.
3. The **middle cerebral arteries** branch laterally from the internal carotid arteries.
4. They supply large portions of the lateral **cerebral hemispheres**.

Posterior Communicating Arteries and Circle of Willis

1. The **posterior communicating arteries** connect the internal carotid arteries to the posterior cerebral arteries.
2. These connections complete the arterial ring known as the **Circle of Willis**.
3. This network creates multiple pathways for blood flow between the anterior and posterior circulations of the brain.

The figure demonstrates how the **Circle of Willis** provides **collateral circulation**, allowing blood to be redistributed if blood flow through one contributing vessel is reduced or obstructed. This arrangement helps maintain **cerebral perfusion** by linking the **carotid** and **vertebrobasilar arterial systems**.

Instructional purpose is to identify the major arteries forming the **Circle of Willis** and demonstrate how connections among the **internal carotid**, **vertebral**, **basilar**, **cerebral**, and **communicating arteries** provide an alternative route for blood flow to the brain.

Figure 7.5.7: Major Branches of the Thoracic and Abdominal Aorta

This figure illustrates the major branches of the **descending aorta** as it travels through the **thoracic** and **abdominal cavities**. The diagram shows the relationship between the aorta and the **organs**, **body walls**, and **lower limbs** supplied by its branches.

Thoracic Aorta

1. The **aortic arch** gives rise to vessels supplying the **head**, **neck**, and **upper extremities** before continuing as the **thoracic aorta**.
2. Within the thorax, the thoracic aorta gives off both **parietal, or somatic, branches** and **visceral branches**.
3. The parietal branches include:
    - **Intercostal arteries**
    - **Superior phrenic arteries**
4. These vessels supply the **thoracic wall** and **diaphragm**.
5. The visceral branches include:
    - **Bronchial arteries**
    - **Esophageal arteries**
    - **Mediastinal arteries**
    - **Pericardial arteries**
6. These vessels supply structures within the thoracic cavity such as the **lungs**, **esophagus**, **mediastinum**, and **pericardium**.

Abdominal Aorta

1. The thoracic aorta passes through the **aortic hiatus** of the diaphragm and continues inferiorly as the **abdominal aorta**.
2. Just below the diaphragm, the **celiac trunk** branches into the:
    - **Left gastric artery**
    - **Splenic artery**
    - **Common hepatic artery**
3. These branches supply the **stomach**, **spleen**, **liver**, and associated digestive organs.
4. Additional major abdominal branches include the:
    - **Superior mesenteric artery**
    - **Inferior mesenteric artery**
5. These arteries supply portions of the **intestines**.

Paired Branches and Terminal Branches

1. The abdominal aorta also gives rise to paired branches, including:
    - **Renal arteries**, supplying the kidneys
    - **Adrenal arteries**, supplying the adrenal glands
    - **Gonadal arteries**, supplying the reproductive organs
    - **Lumbar arteries**, supplying the posterior abdominal wall
2. Near its termination, the **median sacral artery** descends toward the **sacrum**.

3. At the level of the pelvis, the abdominal aorta divides into the **right common iliac artery** and **left common iliac artery**.
4. Each common iliac artery further divides into the:
    - **Internal iliac artery**, which supplies pelvic organs and structures
    - **External iliac artery**, which continues toward the lower limbs

The figure demonstrates the progressive branching pattern of the **descending aorta** and shows how blood is distributed from a single large arterial trunk to the **thoracic organs**, **abdominal organs**, **pelvic structures**, and **lower extremities**.

Instructional purpose is to identify the major branches of the **thoracic** and **abdominal aorta** and demonstrate how the descending aorta distributes **oxygenated blood** to the **thoracic cavity**, **abdominal organs**, **pelvis**, and **lower limbs** through a hierarchical network of arterial branches.

Figure 7.5.8: Major Arteries of the Upper Limb and Hand

This figure illustrates the major **arteries** supplying the **right upper limb**, showing the pathway of blood flow from the **subclavian artery** to the arteries of the **hand** and **fingers**. The diagram follows the arterial network through the **shoulder**, **arm**, **forearm**, **wrist**, and **hand**.

Shoulder Region

1. The **right subclavian artery** passes beneath the **clavicle**.
2. It becomes the **axillary artery** as it enters the **axillary region**.
3. Within the shoulder region, branches including the **humeral circumflex arteries** supply structures surrounding the **shoulder joint** and **proximal humerus**.

Arm

1. As the vessel continues into the arm, the **axillary artery** becomes the **brachial artery**.
2. The brachial artery is the principal artery of the **upper arm**.
3. A major branch, the **deep brachial artery**, supplies the muscles and structures of the **posterior arm**.
4. Additional branches, including the **ulnar collateral arteries**, contribute to arterial networks around the **elbow**.

Forearm

1. Near the elbow, the **brachial artery** divides into the:
    - **Radial artery**
    - **Ulnar artery**
2. These arteries travel through the forearm on opposite sides.
3. An **anterior interosseous artery** branches within the forearm.
4. It courses between the **radius** and **ulna** to supply deeper forearm structures.

Wrist and Hand

1. At the wrist and hand, the **radial** and **ulnar arteries** connect through arterial **anastomoses**.
2. They form the:
    - **Deep palmar arch**
    - **Superficial palmar arch**
3. These interconnected arches provide **collateral circulation** within the hand.
4. They distribute blood to the **palm** and **fingers**.

Digital Arteries

1. Branches arising from the palmar arches form the **digital arteries**.
2. These vessels extend into the **fingers** and supply the tissues of the **digits**.

The figure emphasizes the continuity of blood flow from the **subclavian artery** through the **axillary**, **brachial**, **radial**, and **ulnar arteries**. It also demonstrates how the **palmar arches** create an interconnected arterial network that helps ensure blood supply to the hand and fingers.

Instructional purpose is to identify the major arteries of the **upper limb** and demonstrate how blood is distributed from the **subclavian artery** to the **shoulder**, **arm**, **forearm**, **hand**, and **fingers** through a branching network of arteries and **palmar arterial arches**.

Figure 7.5.9: Major Veins of the Lower Extremity and Pelvis

The figure shows anterior (left) and posterior (right) views of the venous system of the lower extremity and pelvis.

In both views, veins are shown from the pelvis to the foot and labeled by name. At the pelvis, the common iliac vein divides into the external iliac vein and several pelvic tributaries, including the internal iliac, gluteal, lateral sacral, internal pudendal, and obturator veins. The external iliac vein continues into the thigh as the femoral vein.

In the thigh, the femoral vein receives blood from the deep femoral vein and femoral circumflex veins. The great saphenous vein is shown as a superficial vein extending from the foot along the medial leg and thigh to join the femoral vein.

At the knee, the femoral vein continues as the popliteal vein. Below the knee, the anterior tibial, posterior tibial, and fibular veins drain the leg. The small saphenous vein ascends along the posterior calf and joins the popliteal vein.

The anterior view shows the dorsal venous arch and digital veins on the top of the foot. The posterior view shows the plantar venous arch and the medial and lateral plantar veins on the sole of the foot.

The figure functions as a reference map of the major superficial and deep veins that return blood from the lower limbs and pelvis to the larger veins of the trunk.

Figure 7.5.10: Major Veins of the Human Body

In the head and neck, the **external jugular** and **internal jugular veins** drain into the **brachiocephalic veins**, which join to form the **superior vena cava**. Veins of the chest include the **subclavian**, **axillary**, **cephalic**, **brachial**, and **intercostal veins**.

In the upper limbs, the **cephalic**, **basilic**, **brachial**, **radial**, **ulnar**, and **median antebrachial veins** are shown, along with the **median cubital vein** at the elbow. The **palmar venous arches** and **digital veins** drain the hand and fingers.

In the abdomen, the **hepatic**, **renal**, **gonadal**, and **lumbar veins** drain into the **inferior vena cava**.

In the pelvis, the **right and left common iliac veins** receive blood from the **external iliac** and **internal iliac veins** before joining to form the inferior vena cava.

In the lower limbs, the **femoral**, **deep femoral**, **great saphenous**, **popliteal**, **small saphenous**, **anterior tibial**, **posterior tibial**, and **fibular veins** are labeled. Veins of the foot include the **dorsal venous arch** and **plantar venous arch**.

The figure functions as a reference map of the major systemic veins that return deoxygenated blood from the body to the superior and inferior vena cava and ultimately to the heart.

Figure 7.5.11: Major Systemic Veins Draining into the Venae Cavae

In the upper body, the **vertebral**, **internal jugular**, **external jugular**, and **subclavian veins** drain into the **brachiocephalic veins**, which merge to form the **superior vena cava**. Additional thoracic tributaries include the **axillary**, **cephalic**, **internal thoracic**, **intercostal**, **mediastinal**, **esophageal**, **azygos**, and **hemiazygos veins**.

In the abdomen, the **inferior vena cava** receives blood from the **hepatic**, **renal**, **gonadal**, **lumbar**, **phrenic**, and **adrenal veins**.

In the pelvis, the **right and left common iliac veins** unite to form the inferior vena cava. Each common iliac vein is formed by the **internal iliac** and **external iliac veins**.

The figure emphasizes the major venous pathways that return deoxygenated blood from the head, neck, upper limbs, thorax, abdomen, and pelvis to the heart through the superior and inferior vena cava.

Figure 7.5.12: Venous Drainage of the Brain, Head, and Neck

Within the cranial cavity, the labeled dural venous sinuses include the **superior sagittal sinus**, **inferior sagittal sinus**, **straight sinus**, **transverse sinus**, **occipital sinus**, **sigmoid sinus**, **petrosal sinus**, and **cavernous sinus**. These sinuses collect blood from the brain and direct it toward the **internal jugular vein**.

Superficial veins of the face and scalp include the **temporal**, **maxillary**, and **facial veins**. The **internal jugular vein** descends through the neck as the primary drainage pathway from the cranial sinuses, while the **external jugular** and **vertebral veins** drain additional regions of the head and neck.

At the base of the neck, the **internal jugular vein** joins the **right subclavian vein**. Venous return then continues through larger vessels that connect to the **superior vena cava**. Additional labeled vessels include the **axillary vein** and **superior vena cava**.

The figure illustrates the pathways by which venous blood drains from the brain, face, and neck into the major veins that return blood to the heart.

Figure 7.5.13: Major Veins of the Upper Limb

At the shoulder, the **subclavian vein** continues into the **axillary vein**, which receives tributaries including the **cephalic** and **subscapular veins**. The axillary vein continues into the arm as the **brachial vein**.

In the arm and forearm, the superficial **cephalic** and **basilic veins** course along the lateral and medial sides, respectively. At the elbow, the **median cubital vein** connects the cephalic and basilic veins. The **median antebrachial vein** is shown along the forearm.

Deep veins of the forearm include the **radial** and **ulnar veins**, which accompany the arteries of the same names and join proximally to contribute to the brachial vein.

In the hand, the **palmar venous arches** collect blood from the **digital veins** of the fingers.

The figure functions as a reference diagram showing the arrangement of superficial and deep veins that return blood from the hand, forearm, and arm to the axillary and subclavian veins.

Figure 7.5.14: Veins of the Lower Limb and Pelvis

In both views, the **common iliac vein** receives blood from the **internal iliac** and **external iliac veins** and continues into the thigh as the **femoral vein**. Pelvic tributaries include the **gluteal**, **lateral sacral**, **internal pudendal**, and **obturator veins**. The **deep femoral** and **femoral circumflex veins** drain into the femoral vein.

A superficial **great saphenous vein** extends from the foot along the medial side of the leg and thigh to join the femoral vein. At the knee, the femoral vein becomes the **popliteal vein**.

Below the knee, the **anterior tibial**, **posterior tibial**, and **fibular veins** drain the leg. The posterior view also shows the **small saphenous vein**, which ascends along the back of the leg and drains into the popliteal vein.

The anterior view labels the **dorsal venous arch** and **digital veins** of the foot. The posterior view labels the **plantar venous arch** and the **medial** and **lateral plantar veins**.

The figure illustrates the major pathways that return blood from the foot, leg, thigh, and pelvis toward the common iliac veins and the inferior vena cava.

Figure 7.5.15: Hepatic Portal Venous System

The **hepatic portal vein** enters the liver and receives blood from three major venous tributaries:

- The **splenic vein**, which drains the spleen and receives blood from the **gastroepiploic** and **pancreatic veins**
- The **superior mesenteric vein**, which drains the small intestine and portions of the large intestine through tributaries including the **intestinal**, **ileocolic**, **right colic**, **middle colic**, **pancreaticoduodenal**, and **gastro-omental veins**
- The **inferior mesenteric vein**, which drains the distal large intestine through the **left colic**, **sigmoid**, and **superior rectal veins**

Additional labeled vessels include the **right gastric vein** draining the stomach and the **cystic vein** draining the **gallbladder**.

The figure illustrates how venous blood from the stomach, intestines, pancreas, spleen, and gallbladder is collected into the hepatic portal vein and transported to the liver for processing before returning to the general circulation.

Figure 7.6.1: Fetal Circulation and Specialized Vascular Shunts

This figure illustrates the pathway of **blood flow** in the **fetus** and highlights the three specialized shunts that direct circulation before birth: the **ductus venosus**, **foramen ovale**, and **ductus arteriosus**. The fetus is shown connected to the **placenta** through the **umbilical cord**, with arrows indicating the direction of blood flow.

Blood Flow from the Placenta to the Fetus

1. **Oxygenated blood** from the **placenta** travels through the **umbilical vein** toward the fetus.
2. Most of this blood bypasses the **liver** through the **ductus venosus**.
3. The ductus venosus is a vascular shunt that directs blood into the **inferior vena cava**.
4. Blood then enters the **right atrium** of the heart.

Foramen Ovale

1. Because the fetal lungs are not yet functioning in **gas exchange**, much of the blood entering the **right atrium** passes through the **foramen ovale**.
2. The foramen ovale is an opening between the **right atrium** and **left atrium**.
3. This pathway allows blood to bypass the **pulmonary circulation** and enter the **left atrium**.
4. Blood then flows into the **left ventricle** and is pumped into the **aorta** for distribution to the **head**, **heart**, and **upper body**.

Ductus Arteriosus

1. Blood that enters the **right ventricle** is pumped into the **pulmonary trunk**.
2. Due to high resistance in the fetal lungs, most of this blood bypasses the lungs through the **ductus arteriosus**.
3. The ductus arteriosus is a vessel connecting the **pulmonary trunk** to the **aorta**.
4. This shunt directs blood into the **systemic circulation** rather than the pulmonary circulation.

Blood Return to the Placenta

1. After circulating through the fetal body, **oxygen-poor blood** returns to the placenta through the paired **umbilical arteries**.
2. In the placenta, **carbon dioxide** and other wastes are exchanged for **oxygen** and **nutrients** supplied by the maternal circulation.

The figure demonstrates that **fetal circulation** differs from **postnatal circulation** by using three vascular shunts to bypass organs that are not yet fully functional before birth.

1. The **ductus venosus** bypasses much of the **liver**.
2. The **foramen ovale** bypasses the **lungs** by directing blood between the atria.
3. The **ductus arteriosus** bypasses the pulmonary circulation by connecting the **pulmonary trunk** to the **aorta**.

Instructional purpose is to illustrate the pathway of **fetal blood flow** between the fetus and placenta and demonstrate how the **ductus venosus**, **foramen ovale**, and **ductus arteriosus** enable blood to bypass the **liver** and **lungs** before birth.

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8: THE RESPIRATORY SYSTEM

Chapter Review



Figure 8.1: Mountain Climbers The thin air at high elevations can strain the human respiratory system. (CC BY 4.0; via LibreTexts).

The respiratory system is responsible for obtaining oxygen and getting rid of carbon dioxide, and aiding in speech production and in sensing odors. From a functional perspective, the respiratory system can be divided into two major areas: the conducting zone and the respiratory zone. The conducting zone consists of all of the structures that provide passageways for air to travel into and out of the lungs: the nasal cavity, pharynx, trachea, bronchi, and most bronchioles. The nasal passages contain the conchae and meatuses that expand the surface area of the cavity, which helps to warm and humidify incoming air, while removing debris and pathogens. The pharynx is composed of three major sections: the nasopharynx, which is continuous with the nasal cavity; the oropharynx, which borders the nasopharynx and the oral cavity; and the laryngopharynx, which borders the oropharynx, trachea, and esophagus. The respiratory zone includes the structures of the lung that are directly involved in gas exchange: the terminal bronchioles and alveoli.

Histology of the Conducting and Respiratory Zones

The lining of the conducting zone is composed mostly of pseudostratified ciliated columnar epithelium with goblet cells. The mucus traps pathogens and debris, whereas beating cilia move the mucus superiorly toward the throat, where it is swallowed. As the bronchioles become smaller and smaller, and nearer the alveoli, the epithelium thins and is simple squamous epithelium in the alveoli. The endothelium of the surrounding capillaries, together with the alveolar epithelium, forms the respiratory membrane. This is a blood-air barrier through which gas exchange occurs by simple diffusion.

Gross Structure of the Lungs and Pleura

The lungs are the major organs of the respiratory system and are responsible for performing gas exchange. The lungs are paired and separated into lobes; the left lung consists of two lobes, whereas the right lung consists of three lobes. Blood circulation is very important, as blood is required to transport oxygen from the lungs to other tissues throughout the body. The function of the pulmonary circulation is to aid in gas exchange. The pulmonary artery provides deoxygenated blood to the capillaries that form respiratory membranes with the alveoli, and the pulmonary veins return newly oxygenated blood to the heart for further transport throughout the body. The lungs are innervated by the parasympathetic and sympathetic nervous systems, which coordinate the bronchodilation and bronchoconstriction of the airways. The lungs are enclosed by the pleura, a membrane that is composed of visceral and parietal pleural layers. The space between these two layers is called the pleural cavity. The mesothelial cells of the pleural membrane create pleural fluid, which serves as both a lubricant (to reduce friction during breathing) and as an adhesive to adhere the lungs to the thoracic wall (to facilitate movement of the lungs during ventilation).

Pressures and Gas Laws in Pulmonary Ventilation

Pulmonary ventilation is the process of breathing, which is driven by pressure differences between the lungs and the atmosphere. Atmospheric pressure is the force exerted by gases present in the atmosphere. The force exerted by gases within the alveoli is called intra-alveolar (intrapulmonary) pressure, whereas the force exerted by gases in the pleural cavity is called intrapleural pressure. Typically, intrapleural pressure is lower, or negative to, intra-alveolar pressure. The difference in pressure between intrapleural and intra-alveolar pressures is called transpulmonary pressure. In addition, intra-alveolar pressure will equalize with the atmospheric pressure. Pressure is determined by the volume of the space occupied by a gas and is influenced by resistance. Air flows when a pressure gradient is created, from a space of higher pressure to a space of lower pressure. Boyle's law describes the relationship between volume and pressure. A gas is at lower pressure in a larger volume because the gas molecules have more space in which to move. The same quantity of gas in a smaller volume results in gas molecules crowding together, producing increased pressure.

Resistance, Surface Tension, and Lung Compliance

Resistance is created by inelastic surfaces, as well as the diameter of the airways. Resistance reduces the flow of gases. The surface tension of the alveoli also influences pressure, as it opposes the expansion of the alveoli. However, pulmonary surfactant helps to reduce the surface tension so that the alveoli do not collapse during expiration. The ability of the lungs to stretch, called lung compliance, also plays a role in gas flow. The more the lungs can stretch, the greater the potential volume of the lungs. The greater the volume of the lungs, the lower the air pressure within the lungs.

Mechanics of Breathing: Inspiration and Expiration

Pulmonary ventilation consists of the process of inspiration (or inhalation), where air enters the lungs, and expiration (or exhalation), where air leaves the lungs. During inspiration, the diaphragm and external intercostal muscles contract, causing the rib cage to expand and move outward, and expanding the thoracic cavity and lung volume. This creates a lower pressure within the lung than that of the atmosphere, causing air to be drawn into the lungs. During expiration, the diaphragm and intercostals relax, causing the thorax and lungs to recoil. The air pressure within the lungs increases to above the pressure of the atmosphere, causing air to be forced out of the lungs. However, during forced exhalation, the internal intercostals and abdominal muscles may be involved in forcing air out of the lungs.

Respiratory Volumes, Capacities, and Dead Space

Respiratory volume describes the amount of air in a given space within the lungs, or which can be moved by the lung, and is dependent on a variety of factors. Tidal volume refers to the amount of air that enters the lungs during quiet breathing, whereas inspiratory reserve volume is the amount of air that enters the lungs when a person inhales past the tidal volume. Expiratory reserve volume is the extra amount of air that can leave with forceful expiration, following tidal expiration. Residual volume is the amount of air that is left in the lungs after expelling the expiratory reserve volume. Respiratory capacity is the combination of two or more volumes. Anatomical dead space refers to the air within the respiratory structures that never participates in gas exchange, because it does not reach functional alveoli. Respiratory rate is the number of breaths taken per minute, which may change during certain diseases or conditions.

Neural and Chemical Control of Breathing

Both respiratory rate and depth are controlled by the respiratory centers of the brain, which are stimulated by factors such as chemical and pH changes in the blood. These changes are sensed by central chemoreceptors, which are located in the brain, and peripheral chemoreceptors, which are located in the aortic arch and carotid arteries. A rise in carbon dioxide or a decline in oxygen levels in the blood stimulates an increase in respiratory rate and depth.

Gas Laws and Alveolar Gas Exchange

The behavior of gases can be explained by the principles of Dalton's law and Henry's law, both of which describe aspects of gas exchange. Dalton's law states that each specific gas in a mixture of gases exerts force (its partial pressure) independently of the other gases in the mixture. Henry's law states that the amount of a specific gas that dissolves in a liquid is a function of its partial pressure. The greater the partial pressure of a gas, the more of that gas will dissolve in a liquid, as the gas

moves toward equilibrium. Gas molecules move down a pressure gradient; in other words, gas moves from a region of high pressure to a region of low pressure. The partial pressure of oxygen is high in the alveoli and low in the blood of the pulmonary capillaries. As a result, oxygen diffuses across the respiratory membrane from the alveoli into the blood. In contrast, the partial pressure of carbon dioxide is high in the pulmonary capillaries and low in the alveoli. Therefore, carbon dioxide diffuses across the respiratory membrane from the blood into the alveoli. The amount of oxygen and carbon dioxide that diffuses across the respiratory membrane is similar.

Ventilation–Perfusion Relationships and External vs. Internal Respiration

Ventilation is the process that moves air into and out of the alveoli, and perfusion affects the flow of blood in the capillaries. Both are important in gas exchange, as ventilation must be sufficient to create a high partial pressure of oxygen in the alveoli. If ventilation is insufficient and the partial pressure of oxygen drops in the alveolar air, the capillary is constricted and blood flow is redirected to alveoli with sufficient ventilation. External respiration refers to gas exchange that occurs in the alveoli, whereas internal respiration refers to gas exchange that occurs in the tissue. Both are driven by partial pressure differences.

Oxygen Transport and the Oxygen–Hemoglobin Dissociation Curve

Oxygen is primarily transported through the blood by erythrocytes. These cells contain a metalloprotein called hemoglobin, which is composed of four subunits with a ring-like structure. Each subunit contains one atom of iron bound to a molecule of heme. Heme binds oxygen so that each hemoglobin molecule can bind up to four oxygen molecules. When all of the heme units in the blood are bound to oxygen, hemoglobin is considered to be saturated. Hemoglobin is partially saturated when only some heme units are bound to oxygen. An oxygen–hemoglobin saturation/dissociation curve is a common way to depict the relationship of how easily oxygen binds to or dissociates from hemoglobin as a function of the partial pressure of oxygen. As the partial pressure of oxygen increases, the more readily hemoglobin binds to oxygen. At the same time, once one molecule of oxygen is bound by hemoglobin, additional oxygen molecules more readily bind to hemoglobin. Other factors such as temperature, pH, the partial pressure of carbon dioxide, and the concentration of 2,3-bisphosphoglycerate can enhance or inhibit the binding of hemoglobin and oxygen as well. Fetal hemoglobin has a different structure than adult hemoglobin, which results in fetal hemoglobin having a greater affinity for oxygen than adult hemoglobin.

Carbon Dioxide Transport and the Haldane Effect

Carbon dioxide is transported in blood by three different mechanisms: as dissolved carbon dioxide, as bicarbonate, or as carbaminohemoglobin. A small portion of carbon dioxide remains dissolved. The largest amount of transported carbon dioxide is as bicarbonate, formed in erythrocytes. For this conversion, carbon dioxide is combined with water with the aid of an enzyme called carbonic anhydrase. This combination forms carbonic acid, which spontaneously dissociates into bicarbonate and hydrogen ions. As bicarbonate builds up in erythrocytes, it is moved across the membrane into the plasma in exchange for chloride ions by a mechanism called the chloride shift. At the pulmonary capillaries, bicarbonate re-enters erythrocytes in exchange for chloride ions, and the reaction with carbonic anhydrase is reversed, recreating carbon dioxide and water. Carbon dioxide then diffuses out of the erythrocyte and across the respiratory membrane into the air. An intermediate amount of carbon dioxide binds directly to hemoglobin to form carbaminohemoglobin. The partial pressures of carbon dioxide and oxygen, as well as the oxygen saturation of hemoglobin, influence how readily hemoglobin binds carbon dioxide. The less saturated hemoglobin is and the lower the partial pressure of oxygen in the blood is, the more readily hemoglobin binds to carbon dioxide. This is an example of the Haldane effect.

Respiratory System Disorders

A variety of diseases can affect the respiratory system and interfere with normal gas exchange. Common examples include asthma, emphysema, chronic obstructive pulmonary disease (COPD), and lung cancer. Although these conditions differ in their causes and specific changes to lung tissue, they all reduce the efficiency with which oxygen enters the blood and carbon dioxide is removed. As a result, affected individuals often experience labored breathing, shortness of breath, decreased exercise tolerance, and other respiratory difficulties that can significantly affect quality of life.

Respiratory Adjustments to Exercise and High Altitude

Normally, the respiratory centers of the brain maintain a consistent, rhythmic breathing cycle. However, in certain cases, the

respiratory system must adjust to situational changes in order to supply the body with sufficient oxygen. For example, exercise results in increased ventilation, and chronic exposure to a high altitude results in a greater number of circulating erythrocytes. Hyperpnea, an increase in the rate and depth of ventilation, appears to be a function of three neural mechanisms that include a psychological stimulus, motor neuron activation of skeletal muscles, and the activation of proprioceptors in the muscles, joints, and tendons. As a result, hyperpnea related to exercise is initiated when exercise begins, as opposed to when tissue oxygen demand actually increases.

Effects of Acute and Chronic High Altitude: AMS and Acclimatization

In contrast, acute exposure to a high altitude, particularly during times of physical exertion, does result in low blood and tissue levels of oxygen. This change is caused by a low partial pressure of oxygen in the air, because the atmospheric pressure at high altitudes is lower than the atmospheric pressure at sea level. This can lead to a condition called acute mountain sickness (AMS) with symptoms that include headaches, disorientation, fatigue, nausea, and lightheadedness. Over a long period of time, a person's body will adjust to the high altitude, a process called acclimatization. During acclimatization, the low tissue levels of oxygen will cause the kidneys to produce greater amounts of the hormone erythropoietin, which stimulates the production of erythrocytes. Increased levels of circulating erythrocytes provide an increased amount of hemoglobin that helps supply an individual with more oxygen, preventing the symptoms of AMS.

Development of the Respiratory System

The development of the respiratory system in the fetus begins at about 4 weeks and continues into childhood. Ectodermal tissue in the anterior portion of the head region invaginates posteriorly, forming olfactory pits, which ultimately fuse with endodermal tissue of the early pharynx. At about this same time, a protrusion of endodermal tissue extends anteriorly from the foregut, producing a lung bud, which continues to elongate until it forms the laryngotracheal bud. The proximal portion of this structure will mature into the trachea, whereas the bulbous end will branch to form two bronchial buds. These buds then branch repeatedly, so that at about week 16, all major airway structures are present. Development progresses after week 16 as respiratory bronchioles and alveolar ducts form, and extensive vascularization occurs. Alveolar type I cells also begin to take shape. Type II pulmonary cells develop and begin to produce small amounts of surfactant. As the fetus grows, the respiratory system continues to expand as more alveoli develop and more surfactant is produced. Beginning at about week 36 and lasting into childhood, alveolar precursors mature to become fully functional alveoli. At birth, compression of the thoracic cavity forces much of the fluid in the lungs to be expelled. The first inhalation inflates the lungs. Fetal breathing movements begin around week 20 or 21, and occur when contractions of the respiratory muscles cause the fetus to inhale and exhale amniotic fluid. These movements continue until birth and may help to tone the muscles in preparation for breathing after birth and are a sign of good health.

Chapter Objectives

To achieve these chapter objectives, actively engage with the material. Learning tissues is not a passive process. Your understanding will grow as you interact with the content, peers, and your instructor. Regularly check your thinking against feedback. Revisit complex concepts until you can confidently explain and apply them on your own.

After completing this chapter, the student will be able to:

- Distinguish the conducting and respiratory zones of the respiratory system and identify their major structures and basic histology.
- Describe the gross structure of the lungs and pleura, including pulmonary circulation and autonomic control of airway diameter.
- Explain how pressure differences, resistance, surface tension, and lung compliance govern ventilation (inspiration and expiration).
- Define key respiratory volumes and capacities and relate them to ventilation and dead space.
- Summarize neural and chemical regulation of breathing, including the roles of central and peripheral chemoreceptors.
- Apply basic gas laws to alveolar and tissue gas exchange and explain ventilation–perfusion relationships in external and internal respiration.
- Describe how oxygen and carbon dioxide are transported in the blood, including the oxygen–hemoglobin dissociation curve and the Haldane effect.
- Recognize major respiratory disorders and explain, in general terms, how they impair gas exchange and ventilation.

- Describe respiratory adjustments to exercise and high altitude, including acute mountain sickness and acclimatization.
- Outline the major stages of respiratory system development from fetal life through birth and early childhood.

8.1: Organs and Structures of the Respiratory System
8.2: The Lungs
8.3: The Process of Breathing
8.4: Gas Exchange
8.5: Transport of Gases
8.6: Modifications in Respiratory Functions
8.7: Embryonic Development of the Respiratory System
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8.1: Organs and Structures of the Respiratory System

Anatomy of the Respiratory System

The major organs of the respiratory system function primarily to provide oxygen to body tissues for cellular respiration, remove the waste product carbon dioxide, and help to maintain acid-base balance. Portions of the respiratory system are also used for non-vital functions, such as sensing odors, speech production, and for straining, such as during childbirth or coughing (Figure 8.1.1).

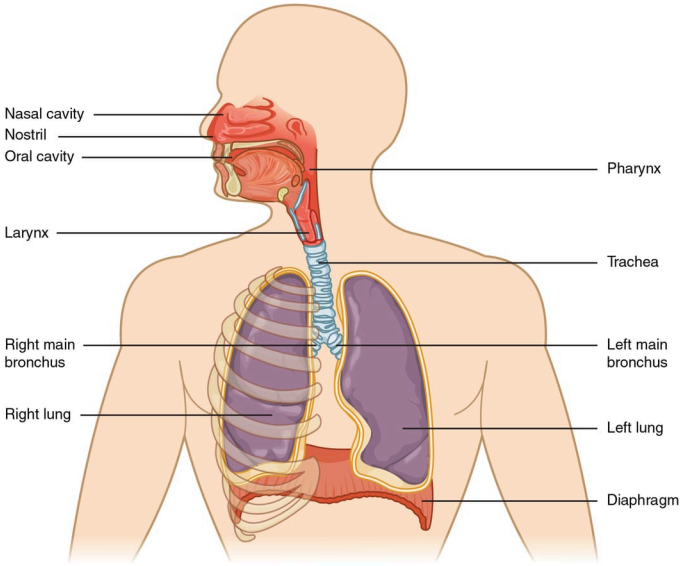


Figure 8.1.1: The major respiratory structures span the nasal cavity to the diaphragm. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.1.

Functionally, the respiratory system can be divided into a conducting zone and a respiratory zone. The conducting zone of the respiratory system includes the organs and structures not directly involved in gas exchange. The gas exchange occurs in the respiratory zone.

Conducting Zone

The major functions of the conducting zone are to provide a route for incoming and outgoing air, remove debris and pathogens from the incoming air, and warm and humidify the incoming air. Several structures within the conducting zone perform other functions as well. The epithelium of the nasal passages, for example, is essential to sensing odors, and the bronchial epithelium that lines the lungs can metabolize some airborne carcinogens.

The Nose and its Adjacent Structures

The major entrance and exit for the respiratory system is through the nose. When discussing the nose, it is helpful to divide it into two major sections: the external nose, and the nasal cavity or internal nose.

The external nose consists of the surface and skeletal structures that result in the outward appearance of the nose and contribute to its numerous functions (Figure 8.1.2). The root is the region of the nose located between the eyebrows. The bridge is the part of the nose that connects the root to the rest of the nose. The dorsum nasi is the length of the nose. The apex is the tip of the nose. On either side of the apex, the nostrils are formed by the alae (singular = ala). An ala is a cartilaginous structure that forms the lateral side of each naris (plural = nares), or nostril opening. The philtrum is the concave surface that connects the apex of the nose to the upper lip.

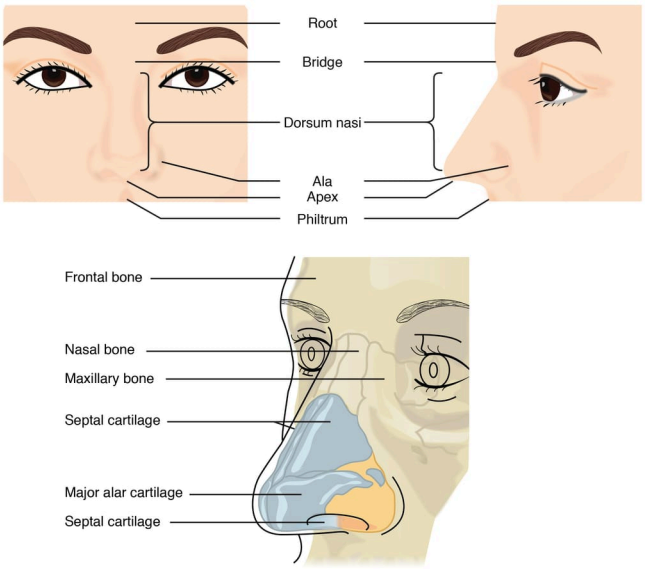


Figure 8.1.2: This illustration shows features of the external nose (top) and skeletal features of the nose (bottom). (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 8.1.2.

Underneath the thin skin of the nose are its skeletal features (see Figure 8.1.2, lower illustration). While the root and bridge of the nose consist of bone, the protruding portion of the nose is composed of cartilage. As a result, when looking at a skull, the nose is missing. The nasal bone is one of a pair of bones that lies under the root and bridge of the nose. The nasal bone articulates superiorly with the frontal bone and laterally with the maxillary bones. Septal cartilage is flexible hyaline cartilage connected to the nasal bone, forming the dorsum nasi. The alar cartilage consists of the apex of the nose; it surrounds the naris.

The nares open into the nasal cavity, which is separated into left and right sections by the nasal septum (Figure 8.1.3). The nasal septum is formed anteriorly by a portion of the septal cartilage (the flexible portion you can touch with your fingers) and posteriorly by the perpendicular plate of the ethmoid bone (a cranial bone located just posterior to the nasal bones) and the thin vomer bones (whose name refers to its plough shape). Each lateral wall of the nasal cavity has three bony projections, called the superior, middle, and inferior nasal conchae. The inferior conchae are separate bones, whereas the superior and middle conchae are portions of the ethmoid bone. Conchae serve to increase the surface area of the nasal cavity and to disrupt the flow of air as it enters the nose, causing air to bounce along the epithelium, where it is cleaned and warmed. The conchae and meatuses also conserve water and prevent dehydration of the nasal epithelium by trapping water during exhalation. The floor of the nasal cavity is composed of the palate. The hard palate at the anterior region of the nasal cavity is composed of bone. The soft palate at the posterior portion of the nasal cavity consists of muscle tissue. Air exits the nasal cavities via the internal nares and moves into the pharynx.

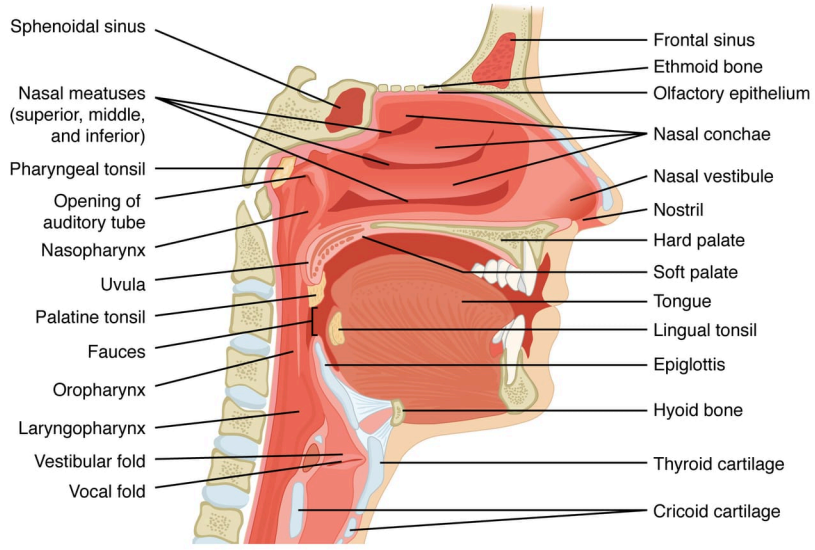


Figure 8.1.3: Sagittal view of the nasal cavity, pharynx, oral cavity, and larynx showing major anatomical structures involved in respiration, olfaction, phonation, and the passage of air and food. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.3.

Several bones that help form the walls of the nasal cavity have air-containing spaces called the paranasal sinuses, which serve to warm and humidify incoming air. Sinuses are lined with a mucosa. Each paranasal sinus is named for its associated bone: frontal sinus, maxillary sinus, sphenoidal sinus, and ethmoidal sinus. The sinuses produce mucus and lighten the weight of the skull.

The nares and anterior portion of the nasal cavities are lined with mucous membranes, containing sebaceous glands and hair follicles that serve to prevent the passage of large debris, such as dirt, through the nasal cavity. An olfactory epithelium used to detect odors is found deeper in the nasal cavity.

The conchae, meatuses, and paranasal sinuses are lined by respiratory epithelium composed of pseudostratified ciliated columnar epithelium (Figure 8.1.4). The epithelium contains goblet cells, one of the specialized, columnar epithelial cells that produce mucus to trap debris. The cilia of the respiratory epithelium help remove the mucus and debris from the nasal cavity with a constant beating motion, sweeping materials towards the throat to be swallowed. Interestingly, cold air slows the movement of the cilia, resulting in accumulation of mucus that may in turn lead to a runny nose during cold weather. This moist epithelium functions to warm and humidify incoming air. Capillaries located just beneath the nasal epithelium warm the air by convection. Serous and mucus-producing cells also secrete the lysozyme enzyme and proteins called defensins, which have antibacterial properties. Immune cells that patrol the connective tissue deep to the respiratory epithelium provide additional protection.



Figure 8.1.4: Respiratory epithelium is pseudostratified ciliated columnar epithelium. Seromucous glands provide lubricating mucus. LM × 680. (Micrograph provided by the Regents of University of Michigan Medical School © 2012). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.4.

Pharynx

The pharynx is a tube formed by skeletal muscle and lined by mucous membrane that is continuous with that of the nasal cavities. The pharynx is divided into three major regions: the nasopharynx, the oropharynx, and the laryngopharynx (Figure 8.1.5).

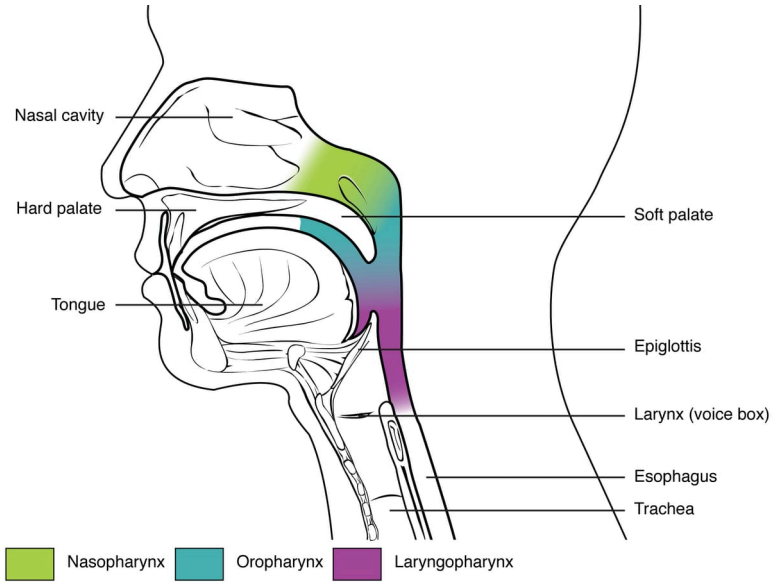


Figure 8.1.5: The pharynx is divided into three regions: the nasopharynx, the oropharynx, and the laryngopharynx. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.5.

The nasopharynx is flanked by the conchae of the nasal cavity, and it serves only as an airway. At the top of the nasopharynx are the pharyngeal tonsils. A pharyngeal tonsil, also called an adenoid, is an aggregate of lymphoid reticular tissue similar to a lymph node that lies at the superior portion of the nasopharynx. The function of the pharyngeal tonsil is not well understood, but it contains a rich supply of lymphocytes and is covered with ciliated epithelium that traps and destroys invading pathogens that enter during inhalation. The pharyngeal tonsils are large in children, but interestingly, tend to regress with age and may even disappear. The uvula is a small bulbous, teardrop-shaped structure located at the apex of the soft palate. Both the uvula and soft palate move like a pendulum during swallowing, swinging upward to close off the nasopharynx to prevent ingested materials from entering the nasal cavity. In addition, auditory (Eustachian) tubes that connect to each middle ear cavity open into the

nasopharynx. This connection is why colds often lead to ear infections.

The oropharynx is a passageway for both air and food. The oropharynx is bordered superiorly by the nasopharynx and anteriorly by the oral cavity. The fauces is the opening at the connection between the oral cavity and the oropharynx. As the nasopharynx becomes the oropharynx, the epithelium changes from pseudostratified ciliated columnar epithelium to stratified squamous epithelium. The oropharynx contains two distinct sets of tonsils, the palatine and lingual tonsils. A palatine tonsil is one of a pair of structures located laterally in the oropharynx in the area of the fauces. The lingual tonsil is located at the base of the tongue. Similar to the pharyngeal tonsil, the palatine and lingual tonsils are composed of lymphoid tissue, and trap and destroy pathogens entering the body through the oral or nasal cavities.

The laryngopharynx is inferior to the oropharynx and posterior to the larynx. It continues the route for ingested material and air until its inferior end, where the digestive and respiratory systems diverge. The stratified squamous epithelium of the oropharynx is continuous with the laryngopharynx. Anteriorly, the laryngopharynx opens into the larynx, whereas posteriorly, it enters the esophagus.

Larynx

The larynx is a cartilaginous structure inferior to the laryngopharynx that connects the pharynx to the trachea and helps regulate the volume of air that enters and leaves the lungs (Figure 8.1.6). The structure of the larynx is formed by several pieces of cartilage. Three large cartilage pieces—the thyroid cartilage (anterior), epiglottis (superior), and cricoid cartilage (inferior)—form the major structure of the larynx. The thyroid cartilage is the largest piece of cartilage that makes up the larynx. The thyroid cartilage consists of the laryngeal prominence, or “Adam’s apple,” which tends to be more prominent in males. The thick cricoid cartilage forms a ring, with a wide posterior region and a thinner anterior region. Three smaller, paired cartilages—the arytenoids, corniculates, and cuneiforms—attach to the epiglottis and the vocal cords and muscle that help move the vocal cords to produce speech.

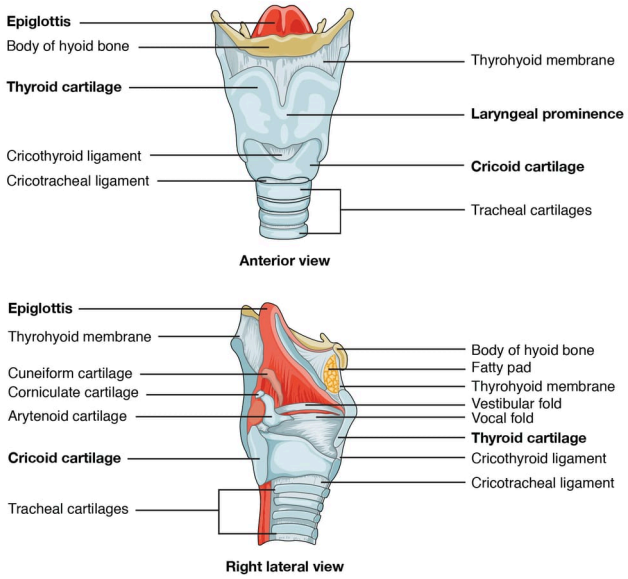


Figure 8.1.6: The larynx extends from the laryngopharynx and the hyoid bone to the trachea. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.6.

The epiglottis, attached to the thyroid cartilage, is a very flexible piece of elastic cartilage that covers the opening of the trachea (see Figure 8.1.3). When in the “closed” position, the unattached end of the epiglottis rests on the glottis. The glottis is composed of the vestibular folds, the true vocal cords, and the space between these folds (Figure 8.1.7). A vestibular fold, or false vocal cord, is one of a pair of folded sections of mucous membrane. A true vocal cord is one of the white, membranous folds attached by muscle to the thyroid and arytenoid cartilages of the larynx on their outer edges. The inner edges of the true vocal cords are free, allowing oscillation to produce sound. The size of the membranous folds of the true vocal cords differs between individuals, producing voices with different pitch ranges. Folds in males tend to be larger than those in females, which create a deeper voice. The act of swallowing causes the pharynx and larynx to lift upward, allowing the pharynx to expand and the epiglottis of the larynx to swing downward, closing the opening to the trachea. These movements produce a larger area for food to pass through, while preventing food and beverages from entering the trachea.

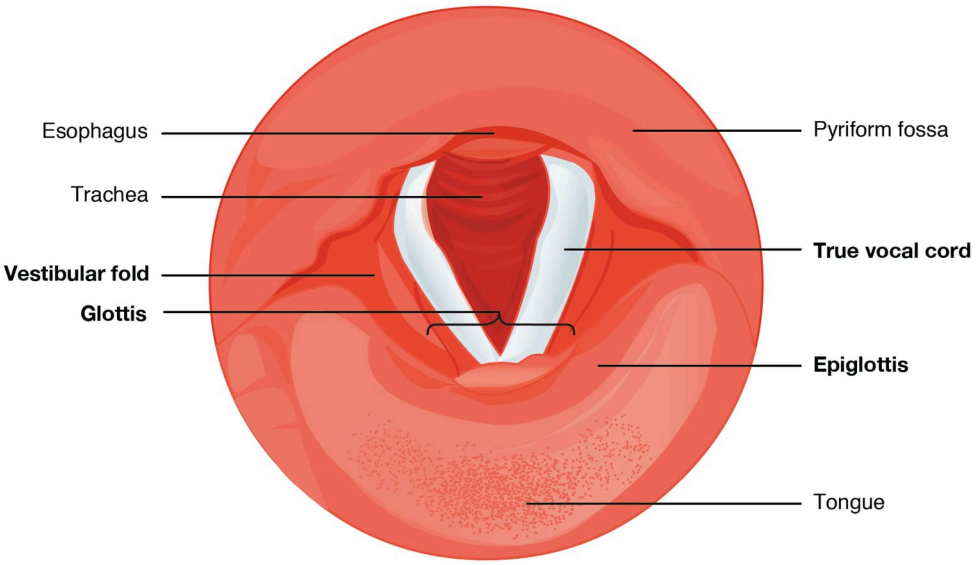


Figure 8.1.7: The true vocal cords and vestibular folds of the larynx are viewed inferiorly from the laryngopharynx. (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 8.1.7.

Continuous with the laryngopharynx, the superior portion of the larynx is lined with stratified squamous epithelium, transitioning into pseudostratified ciliated columnar epithelium that contains goblet cells. Similar to the nasal cavity and nasopharynx, this specialized epithelium produces mucus to trap debris and pathogens as they enter the trachea. The cilia beat the mucus upward towards the laryngopharynx, where it can be swallowed down the esophagus.

Trachea

The trachea (windpipe) extends from the larynx toward the lungs (Figure 8.1.8*a*). The trachea is formed by 16 to 20 stacked, C-shaped pieces of hyaline cartilage that are connected by dense connective tissue. The trachealis muscle and elastic connective tissue together form the fibroelastic membrane, a flexible membrane that closes the posterior surface of the trachea, connecting the C-shaped cartilages. The fibroelastic membrane allows the trachea to stretch and expand slightly during inhalation and exhalation, whereas the rings of cartilage provide structural support and prevent the trachea from collapsing. In addition, the trachealis muscle can be contracted to force air through the trachea during exhalation. The trachea is lined with pseudostratified ciliated columnar epithelium, which is continuous with the larynx. The esophagus borders the trachea posteriorly.

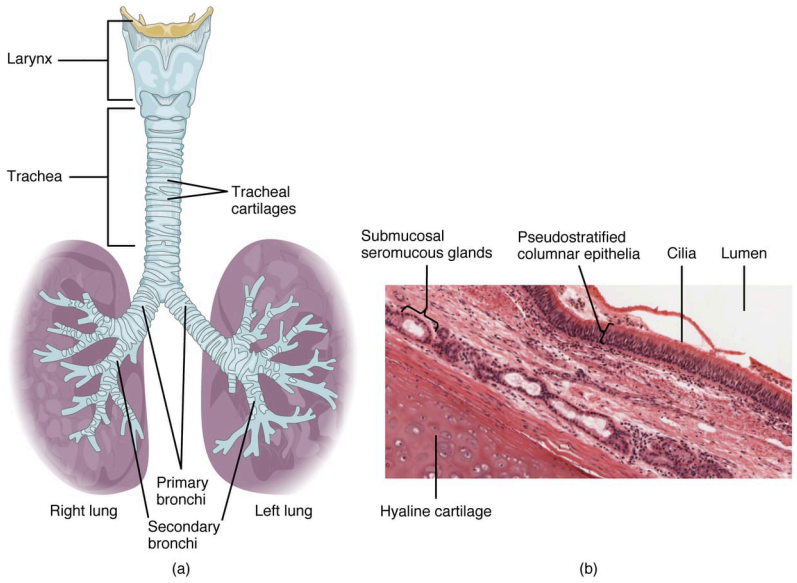


Figure 8.1.8: (a) The tracheal tube is formed by stacked, C-shaped pieces of hyaline cartilage. (b) The layer visible in this cross-section of tracheal wall tissue between the hyaline cartilage and the lumen of the trachea is the mucosa, which is composed of pseudostratified ciliated columnar epithelium that contains goblet cells. LM × 1220. (Micrograph provided by the Regents of University of Michigan Medical School © 2012). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.8.

Bronchial Tree

The trachea branches into the right and left primary bronchi at the carina. These bronchi are also lined by pseudostratified ciliated columnar epithelium containing mucus-producing goblet cells (Figure 8.1.8*b*). The carina is a raised structure that contains specialized nervous tissue that induces violent coughing if a foreign body, such as food, is present. Rings of cartilage, similar to those of the trachea, support the structure of the bronchi and prevent their collapse. The primary bronchi enter the lungs at the hilum, a concave region where blood vessels, lymphatic vessels, and nerves also enter the lungs. The bronchi continue to branch into a bronchial tree. A bronchial tree (or respiratory tree) is the collective term used for these multiple-branched bronchi. The main function of the bronchi, like other conducting zone structures, is to provide a passageway for air to move into and out of each lung. In addition, the mucous membrane traps debris and pathogens.

A bronchiole branches from the tertiary bronchi. Bronchioles, which are about 1 mm in diameter, further branch until they become the tiny terminal bronchioles, which lead to the structures of gas exchange. There are more than 1000 terminal bronchioles in each lung. The muscular walls of the bronchioles do not contain cartilage like those of the bronchi. This muscular wall can change the size of the tubing to increase or decrease airflow through the tube.

Respiratory Zone

In contrast to the conducting zone, the respiratory zone includes structures that are directly involved in gas exchange. The respiratory zone begins where the terminal bronchioles join a respiratory bronchiole, the smallest type of bronchiole (Figure 8.1.9), which then leads to an alveolar duct, opening into a cluster of alveoli.

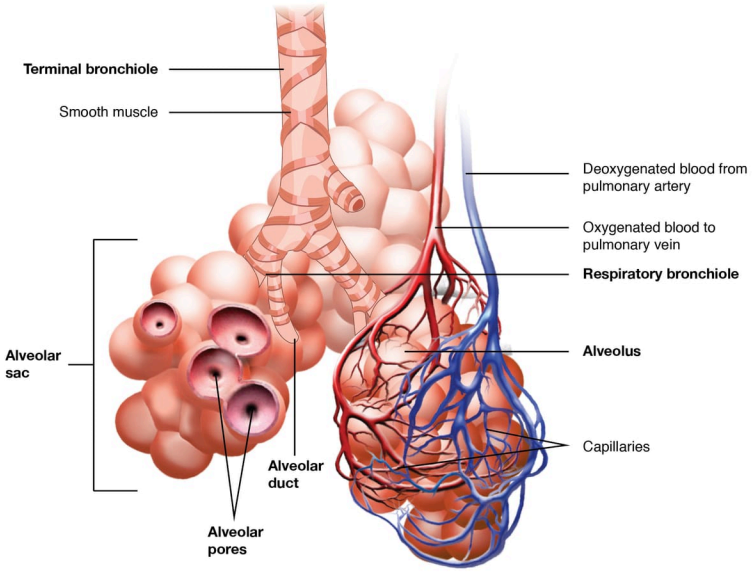


Figure 8.1.9: Bronchioles lead to alveolar sacs in the respiratory zone, where gas exchange occurs. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.9.

Alveoli

An alveolar duct is a tube composed of smooth muscle and connective tissue, which opens into a cluster of alveoli. An alveolus is one of the many small, grape-like sacs that are attached to the alveolar ducts.

An alveolar sac is a cluster of many individual alveoli that are responsible for gas exchange. An alveolus is approximately 200 µm in diameter with elastic walls that allow the alveolus to stretch during air intake, which greatly increases the surface area available for gas exchange. Alveoli are connected to their neighbors by alveolar pores, which help maintain equal air pressure throughout the alveoli and lung (Figure 8.1.10).

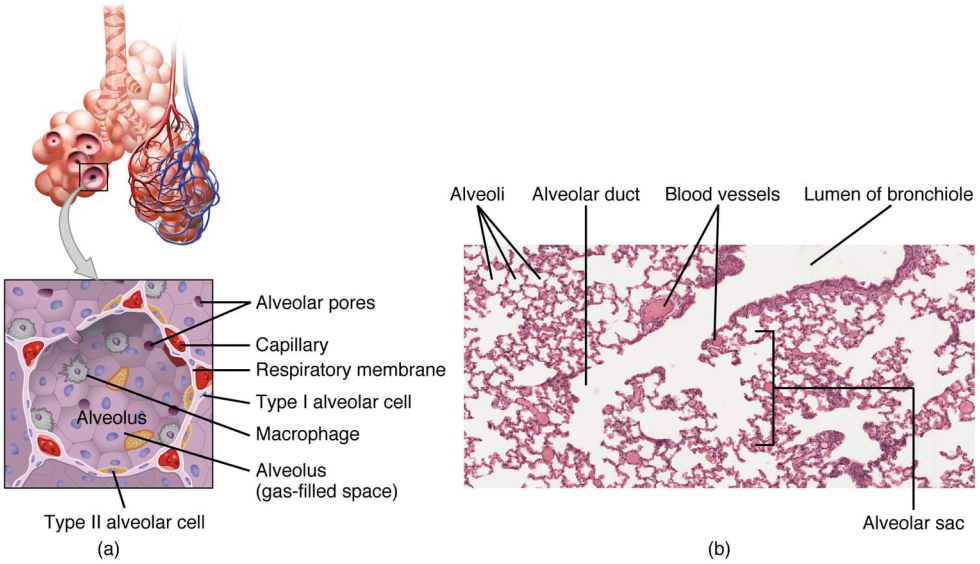


Figure 8.1.10: (a) The alveolus is responsible for gas exchange. (b) A micrograph shows the alveolar structures within lung tissue. LM × 178. (Micrograph provided by the Regents of University of Michigan Medical School © 2012). (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.10.

The alveolar wall consists of three major cell types: type I alveolar cells, type II alveolar cells, and alveolar macrophages. A type I alveolar cell is a squamous epithelial cell of the alveoli, which constitute up to 97 percent of the alveolar surface area. These

cells are about 25 nm thick and are highly permeable to gases. A type II alveolar cell is interspersed among the type I cells and secretes pulmonary surfactant, a substance composed of phospholipids and proteins that reduces the surface tension of the alveoli. Roaming around the alveolar wall is the alveolar macrophage, a phagocytic cell of the immune system that removes debris and pathogens that have reached the alveoli.

The simple squamous epithelium formed by type I alveolar cells is attached to a thin, elastic basement membrane. This epithelium is extremely thin and borders the endothelial membrane of capillaries. Taken together, the alveoli and capillary membranes form a respiratory membrane that is approximately 0.5 µm (micrometers) thick. The respiratory membrane allows gases to cross by simple diffusion, allowing oxygen to be picked up by the blood for transport and CO_2 to be released into the air of the alveoli.

Diseases of the Respiratory system: Asthma

Asthma is common condition that affects the lungs in both adults and children. Approximately 8.2 percent of adults (18.7 million) and 9.4 percent of children (7 million) in the United States suffer from asthma. In addition, asthma is the most frequent cause of hospitalization in children.

Asthma is a chronic disease characterized by inflammation and edema of the airway, and bronchospasms (that is, constriction of the bronchioles), which can inhibit air from entering the lungs. In addition, excessive mucus secretion can occur, which further contributes to airway occlusion (Figure 8.1.11). Cells of the immune system, such as eosinophils and mononuclear cells, may also be involved in infiltrating the walls of the bronchi and bronchioles.

Bronchospasms occur periodically and lead to an “asthma attack.” An attack may be triggered by environmental factors such as dust, pollen, pet hair, or dander, changes in the weather, mold, tobacco smoke, and respiratory infections, or by exercise and stress.

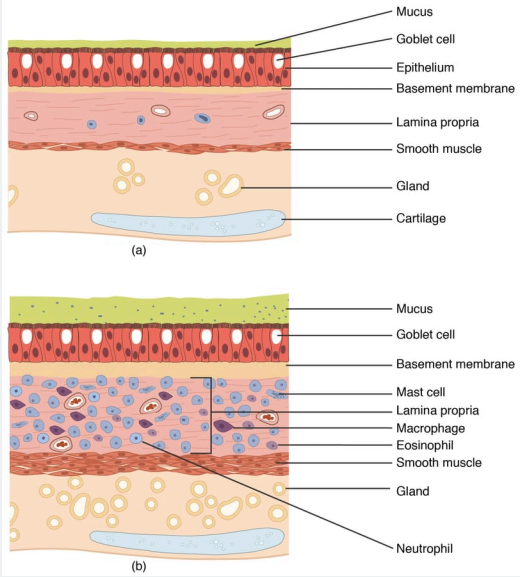


Figure 8.1.11: (a) Normal lung tissue does not have the characteristics of lung tissue during (b) an asthma attack, which include thickened mucosa, increased mucus-producing goblet cells, and eosinophil infiltrates. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.1.11.

Symptoms of an asthma attack involve coughing, shortness of breath, wheezing, and tightness of the chest. Symptoms of a severe asthma attack that requires immediate medical attention would include difficulty breathing that results in blue (cyanotic) lips or face, confusion, drowsiness, a rapid pulse, sweating, and severe anxiety. The severity of the condition, frequency of attacks, and identified triggers influence the type of medication that an individual may require. Longer-term treatments are used for those with more severe asthma. Short-term, fast-acting drugs that are used to treat an asthma attack are typically administered via an inhaler. For young children or individuals who have difficulty using an inhaler, asthma medications can be administered via a nebulizer.

In many cases, the underlying cause of the condition is unknown. However, recent research has demonstrated that certain viruses,

such as human rhinovirus C (HRVC), and the bacteria *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* that are contracted in infancy or early childhood, may contribute to the development of many cases of asthma.

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8.2: The Lungs

Structure and Function of Lungs

A major organ of the respiratory system, each lung houses structures of both the conducting and respiratory zones. The main function of the lungs is to perform the exchange of oxygen and carbon dioxide with air from the atmosphere. To this end, the lungs exchange respiratory gases across a very large epithelial surface area—about 70 square meters—that is highly permeable to gases.

Gross Anatomy of the Lungs

The lungs are pyramid-shaped, paired organs that are connected to the trachea by the right and left bronchi; on the inferior surface, the lungs are bordered by the diaphragm. The diaphragm is a dome-shaped muscle located at the base of the lungs and thoracic cavity. The lungs are enclosed by the pleurae, which are attached to the mediastinum. The right lung is shorter and wider than the left lung, and the left lung occupies a smaller volume than the right. The cardiac notch is an indentation on the surface of the left lung, and it allows space for the heart (Figure 8.2.1). The apex of the lung is the superior region, whereas the base is the opposite region near the diaphragm. The costal surface of the lung borders the ribs. The mediastinal surface faces the midline.

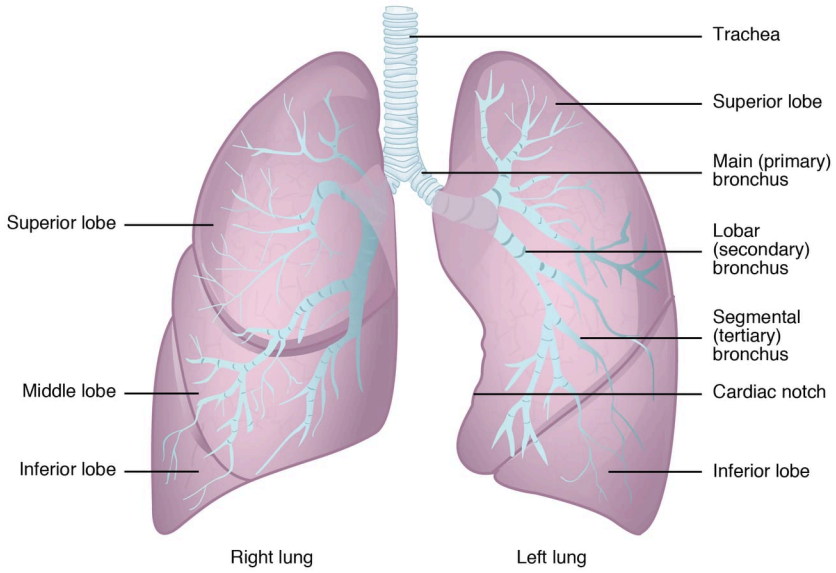


Figure 8.2.1: Gross Anatomy of the Lungs. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.2.1.

Each lung is composed of smaller units called lobes. Fissures separate these lobes from each other. The right lung consists of three lobes: the superior, middle, and inferior lobes. The left lung consists of two lobes: the superior and inferior lobes. A bronchopulmonary segment is a division of a lobe, and each lobe houses multiple bronchopulmonary segments. Each segment receives air from its own tertiary bronchus and is supplied with blood by its own artery. Some diseases of the lungs typically affect one or more bronchopulmonary segments, and in some cases, the diseased segments can be surgically removed with little influence on neighboring segments. A pulmonary lobule is a subdivision formed as the bronchi branch into bronchioles. Each lobule receives its own large bronchiole that has multiple branches. An interlobular septum is a wall, composed of connective tissue, which separates lobules from one another.

Blood Supply and Nervous Innervation of the Lungs

The blood supply of the lungs plays an important role in gas exchange and serves as a transport system for gases throughout the body. In addition, innervation by the both the parasympathetic and sympathetic nervous systems provides an important level of control through dilation and constriction of the airway.

Blood Supply

The major function of the lungs is to perform gas exchange, which requires blood from the pulmonary circulation. This blood supply contains deoxygenated blood and travels to the lungs where erythrocytes, also known as red blood cells, pick up oxygen to be transported to tissues throughout the body. The pulmonary artery is an artery that arises from the pulmonary trunk and carries deoxygenated, arterial blood to the alveoli. The pulmonary artery branches multiple times as it follows the bronchi, and each branch becomes progressively smaller in diameter. One arteriole and an accompanying venule supply and drain one pulmonary lobule. As they near the alveoli, the pulmonary arteries become the pulmonary capillary network. The pulmonary capillary network consists of tiny vessels with very thin walls that lack smooth muscle fibers. The capillaries branch and follow the bronchioles and structure of the alveoli. It is at this point that the capillary wall meets the alveolar wall, creating the respiratory membrane. Once the blood is oxygenated, it drains from the alveoli by way of multiple pulmonary veins, which exit the lungs through the hilum.

Nervous Innervation

Dilation and constriction of the airway are achieved through nervous control by the parasympathetic and sympathetic nervous systems. The parasympathetic system causes bronchoconstriction, whereas the sympathetic nervous system stimulates bronchodilation. Reflexes such as coughing, and the ability of the lungs to regulate oxygen and carbon dioxide levels, also result from this autonomic nervous system control. Sensory nerve fibers arise from the vagus nerve, and from the second to fifth thoracic ganglia. The pulmonary plexus is a region on the lung root formed by the entrance of the nerves at the hilum. The nerves then follow the bronchi in the lungs and branch to innervate muscle fibers, glands, and blood vessels.

Pleura of the Lungs

Each lung is enclosed within a cavity that is surrounded by the pleura. The pleura (plural = pleurae) is a serous membrane that surrounds the lung. The right and left pleurae, which enclose the right and left lungs, respectively, are separated by the mediastinum. The pleurae consist of two layers. The visceral pleura is the layer that is superficial to the lungs, and extends into and lines the lung fissures (Figure 8.2.2). In contrast, the parietal pleura is the outer layer that connects to the thoracic wall, the mediastinum, and the diaphragm. The visceral and parietal pleurae connect to each other at the hilum. The pleural cavity is the space between the visceral and parietal layers.

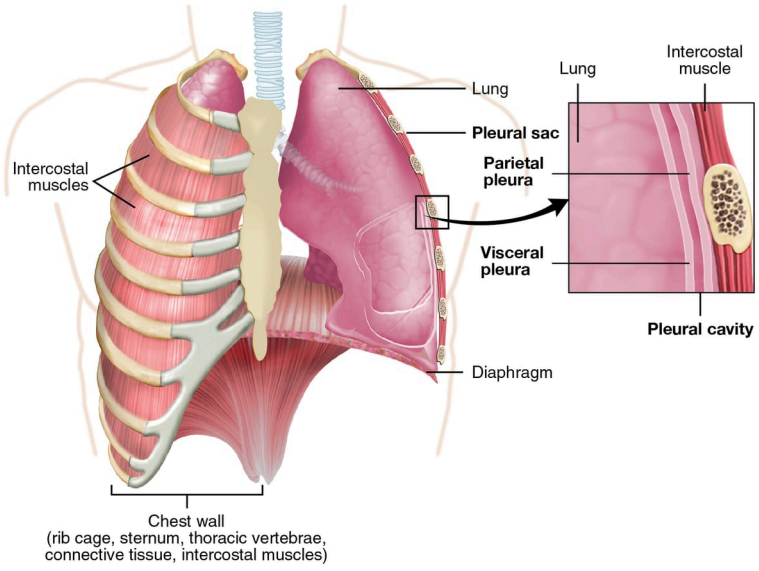


Figure 8.2.2: Parietal and Visceral Pleurae of the Lungs. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.2.2

The pleurae perform two major functions: They produce pleural fluid and create cavities that separate the major organs. Pleural fluid is secreted by mesothelial cells from both pleural layers and acts to lubricate their surfaces. This lubrication reduces friction between the two layers to prevent trauma during breathing, and creates surface tension that helps maintain the position of the lungs against the thoracic wall. This adhesive characteristic of the pleural fluid causes the lungs to enlarge when the thoracic wall expands during ventilation, allowing the lungs to fill with air. The pleurae also create a division between major organs that prevents interference due to the movement of the organs, while preventing the spread of infection.

Everyday Connection

The Effects of Second-Hand Tobacco Smoke

The burning of a tobacco cigarette creates multiple chemical compounds that are released through mainstream smoke, which is inhaled by the smoker, and through sidestream smoke, which is the smoke that is given off by the burning cigarette. Second-hand smoke, which is a combination of sidestream smoke and the mainstream smoke that is exhaled by the smoker, has been demonstrated by numerous scientific studies to cause disease. At least 40 chemicals in sidestream smoke have been identified that negatively impact human health, leading to the development of cancer or other conditions, such as immune system dysfunction, liver toxicity, cardiac arrhythmias, pulmonary edema, and neurological dysfunction. Furthermore, second-hand smoke has been found to harbor at least 250 compounds that are known to be toxic, carcinogenic, or both. Some major classes of carcinogens in second-hand smoke are polyaromatic hydrocarbons (PAHs), N-nitrosamines, aromatic amines, formaldehyde, and acetaldehyde.

Tobacco and second-hand smoke are considered to be carcinogenic. Exposure to second-hand smoke can cause lung cancer in individuals who are not tobacco users themselves. It is estimated that the risk of developing lung cancer is increased by up to 30 percent in nonsmokers who live with an individual who smokes in the house, as compared to nonsmokers who are not regularly exposed to second-hand smoke. Children are especially affected by second-hand smoke. Children who live with an individual who smokes inside the home have a larger number of lower respiratory infections, which are associated with hospitalizations, and higher risk of sudden infant death syndrome (SIDS). Second-hand smoke in the home has also been linked to a greater number of ear infections in children, as well as worsening symptoms of asthma.

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8.3: The Process of Breathing

Mechanisms of Breathing

Pulmonary ventilation is the act of breathing, which can be described as the movement of air into and out of the lungs. The major mechanisms that drive pulmonary ventilation are atmospheric pressure (P_{atm}); the air pressure within the alveoli, called intra-alveolar pressure (P_{alv}); and the pressure within the pleural cavity, called intrapleural pressure (P_{ip}).

The intra-alveolar and intrapleural pressures are dependent on certain physical features of the lung. However, the ability to breathe—to have air enter the lungs during inspiration and air leave the lungs during expiration—is dependent on the air pressure of the atmosphere and the air pressure within the lungs.

Pressure Relationships

Inspiration (or inhalation) and expiration (or exhalation) are dependent on the differences in pressure between the atmosphere and the lungs. In a gas, pressure is a force created by the movement of gas molecules that are confined. For example, a certain number of gas molecules in a two-liter container has more room than the same number of gas molecules in a one-liter container (Figure 8.3.1). In this case, the force exerted by the movement of the gas molecules against the walls of the two-liter container is lower than the force exerted by the gas molecules in the one-liter container. Therefore, the pressure is lower in the two-liter container and higher in the one-liter container. At a constant temperature, changing the volume occupied by the gas changes the pressure, as does changing the number of gas molecules. Boyle's law describes the relationship between volume and pressure in a gas at a constant temperature. Boyle discovered that the pressure of a gas is inversely proportional to its volume: If volume increases, pressure decreases. Likewise, if volume decreases, pressure increases. Pressure and volume are inversely related ($P = k/V$). Therefore, the pressure in the one-liter container (one-half the volume of the two-liter container) would be twice the pressure in the two-liter container. Boyle's law is expressed by the following formula:

$$P_1V_1 = P_2V_2 \tag{8.3.1}$$

In this formula, P_1 represents the initial pressure and V_1 represents the initial volume, whereas the final pressure and volume are represented by P_2 and V_2, respectively. If the two- and one-liter containers were connected by a tube and the volume of one of the containers were changed, then the gases would move from higher pressure (lower volume) to lower pressure (higher volume).

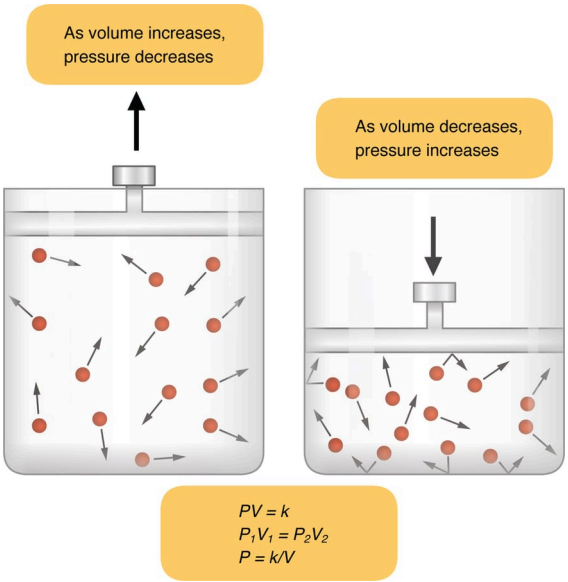


Figure 8.3.1: When the volume of a closed container increases, gas pressure decreases; when the volume decreases, gas pressure increases. This inverse relationship between pressure and volume underlies the pressure changes that drive pulmonary ventilation. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.3.1.

Pulmonary ventilation is dependent on three types of pressure: atmospheric, intra-alveolar, and intrapleural. Atmospheric pressure is the amount of force that is exerted by gases in the air surrounding any given surface, such as the body. Atmospheric pressure can be expressed in terms of the unit atmosphere, abbreviated atm, or in millimeters of mercury (mm Hg). One atm is equal to 760 mm Hg, which is the atmospheric pressure at sea level. Typically, for respiration, other pressure values are discussed in relation to atmospheric pressure. Therefore, negative pressure is pressure lower than the atmospheric pressure, whereas positive pressure is pressure that it is greater than the atmospheric pressure. A pressure that is equal to the atmospheric pressure is expressed as zero.

Intra-alveolar pressure (intrapulmonary pressure) is the pressure of the air within the alveoli, which changes during the different phases of breathing (Figure 8.3.2). Because the alveoli are connected to the atmosphere via the tubing of the airways (similar to the two- and one-liter containers in the example above), the intrapulmonary pressure of the alveoli always equalizes with the atmospheric pressure.

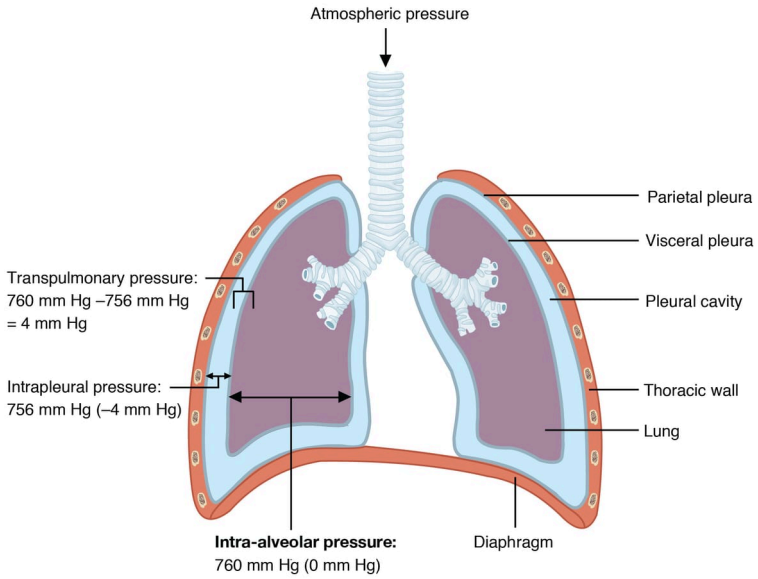


Figure 8.3.2: Intra-alveolar pressure changes during the different phases of the cycle. It equalizes at 760 mm Hg but does not remain at 760 mm Hg. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.3.2.

Intrapleural pressure is the pressure of the air within the pleural cavity, between the visceral and parietal pleurae. Similar to intra-alveolar pressure, intrapleural pressure also changes during the different phases of breathing. However, due to certain characteristics of the lungs, the intrapleural pressure is always lower than, or negative to, the intra-alveolar pressure (and therefore also to atmospheric pressure). Although it fluctuates during inspiration and expiration, intrapleural pressure remains approximately –4 mm Hg throughout the breathing cycle.

Competing forces within the thorax cause the formation of the negative intrapleural pressure. One of these forces relates to the elasticity of the lungs themselves—elastic tissue pulls the lungs inward, away from the thoracic wall. Surface tension of alveolar fluid, which is mostly water, also creates an inward pull of the lung tissue. This inward tension from the lungs is countered by opposing forces from the pleural fluid and thoracic wall. Surface tension within the pleural cavity pulls the lungs outward. Too much or too little pleural fluid would hinder the creation of the negative intrapleural pressure; therefore, the level must be closely monitored by the mesothelial cells and drained by the lymphatic system. Since the parietal pleura is attached to the thoracic wall, the natural elasticity of the chest wall opposes the inward pull of the lungs. Ultimately, the outward pull is slightly greater than the inward pull, creating the –4 mm Hg intrapleural pressure relative to the intra-alveolar pressure. Transpulmonary pressure is the difference between the intrapleural and intra-alveolar pressures, and it determines the size of the lungs. A higher transpulmonary pressure corresponds to a larger lung.

Physical Factors Affecting Ventilation

In addition to the differences in pressures, breathing is also dependent upon the contraction and relaxation of muscle fibers of both the diaphragm and thorax. The lungs themselves are passive during breathing, meaning they are not involved in creating the movement that helps inspiration and expiration. This is because of the adhesive nature of the pleural fluid, which allows the lungs to be pulled outward when the thoracic wall moves during inspiration. The recoil of the thoracic wall during expiration

causes compression of the lungs. Contraction and relaxation of the diaphragm and intercostals muscles (found between the ribs) cause most of the pressure changes that result in inspiration and expiration. These muscle movements and subsequent pressure changes cause air to either rush in or be forced out of the lungs.

Other characteristics of the lungs influence the effort that must be expended to ventilate. Resistance is a force that slows motion, in this case, the flow of gases. The size of the airway is the primary factor affecting resistance. A small tubular diameter forces air through a smaller space, causing more collisions of air molecules with the walls of the airways. The following formula helps to describe the relationship between airway resistance and pressure changes:

$$F = \Delta P/R$$

As noted earlier, there is surface tension within the alveoli caused by water present in the lining of the alveoli. This surface tension tends to inhibit expansion of the alveoli. However, pulmonary surfactant secreted by type II alveolar cells mixes with that water and helps reduce this surface tension. Without pulmonary surfactant, the alveoli would collapse during expiration.

Thoracic wall compliance is the ability of the thoracic wall to stretch while under pressure. This can also affect the effort expended in the process of breathing. In order for inspiration to occur, the thoracic cavity must expand. The expansion of the thoracic cavity directly influences the capacity of the lungs to expand. If the tissues of the thoracic wall are not very compliant, it will be difficult to expand the thorax to increase the size of the lungs.

Pulmonary Ventilation

The difference in pressures drives pulmonary ventilation because air flows down a pressure gradient, that is, air flows from an area of higher pressure to an area of lower pressure. Air flows into the lungs largely due to a difference in pressure; atmospheric pressure is greater than intra-alveolar pressure, and intra-alveolar pressure is greater than intrapleural pressure. Air flows out of the lungs during expiration based on the same principle; pressure within the lungs becomes greater than the atmospheric pressure.

Pulmonary ventilation comprises two major steps: inspiration and expiration. Inspiration is the process that causes air to enter the lungs, and expiration is the process that causes air to leave the lungs (Figure 8.3.3). A respiratory cycle is one sequence of inspiration and expiration. In general, two muscle groups are used during normal inspiration: the diaphragm and the external intercostal muscles. Additional muscles can be used if a bigger breath is required. When the diaphragm contracts, it moves inferiorly toward the abdominal cavity, creating a larger thoracic cavity and more space for the lungs. Contraction of the external intercostal muscles moves the ribs upward and outward, causing the rib cage to expand, which increases the volume of the thoracic cavity. Due to the adhesive force of the pleural fluid, the expansion of the thoracic cavity forces the lungs to stretch and expand as well. This increase in volume leads to a decrease in intra-alveolar pressure, creating a pressure lower than atmospheric pressure. As a result, a pressure gradient is created that drives air into the lungs.

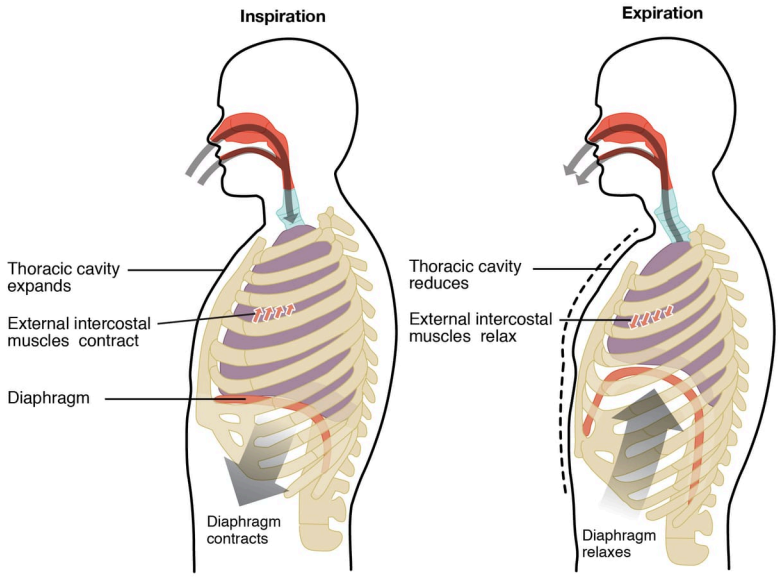


Figure 8.3.3: Inspiration and Expiration Inspiration and expiration occur due to the expansion and contraction of the thoracic cavity, respectively. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.3.3.

The process of normal expiration is passive, meaning that energy is not required to push air out of the lungs. Instead, the elasticity of the lung tissue causes the lung to recoil, as the diaphragm and intercostal muscles relax following inspiration. In turn, the thoracic cavity and lungs decrease in volume, causing an increase in intrapulmonary pressure. The intrapulmonary pressure rises above atmospheric pressure, creating a pressure gradient that causes air to leave the lungs.

There are different types, or modes, of breathing that require a slightly different process to allow inspiration and expiration. Quiet breathing, also known as eupnea, is a mode of breathing that occurs at rest and does not require the cognitive thought of the individual. During quiet breathing, the diaphragm and external intercostals must contract.

A deep breath, called diaphragmatic breathing, requires the diaphragm to contract. As the diaphragm relaxes, air passively leaves the lungs. A shallow breath, called costal breathing, requires contraction of the intercostal muscles. As the intercostal muscles relax, air passively leaves the lungs.

In contrast, forced breathing, also known as hyperpnea, is a mode of breathing that can occur during exercise or actions that require the active manipulation of breathing, such as singing. During forced breathing, inspiration and expiration both occur due to muscle contractions. In addition to the contraction of the diaphragm and intercostal muscles, other accessory muscles must also contract. During forced inspiration, muscles of the neck, including the scalenes, contract and lift the thoracic wall, increasing lung volume. During forced expiration, accessory muscles of the abdomen, including the obliques, contract, forcing abdominal organs upward against the diaphragm. This helps to push the diaphragm further into the thorax, pushing more air out. In addition, accessory muscles (primarily the internal intercostals) help to compress the rib cage, which also reduces the volume of the thoracic cavity.

Respiratory Volumes and Capacities

Respiratory volume is the term used for various volumes of air moved by or associated with the lungs at a given point in the respiratory cycle. There are four major types of respiratory volumes: tidal, residual, inspiratory reserve, and expiratory reserve (Figure 8.3.4). Tidal volume (TV) is the amount of air that normally enters the lungs during quiet breathing, which is about 500 milliliters. Expiratory reserve volume (ERV) is the amount of air you can forcefully exhale past a normal tidal expiration, up to 1200 milliliters for males. Inspiratory reserve volume (IRV) is produced by a deep inhalation, past a tidal inspiration. This is the extra volume that can be brought into the lungs during a forced inspiration. Residual volume (RV) is the air left in the lungs if you exhale as much air as possible. The residual volume makes breathing easier by preventing the alveoli from collapsing. Respiratory volume is dependent on a variety of factors, and measuring the different types of respiratory volumes can provide important clues about a person's respiratory health (Table 8.3.1).

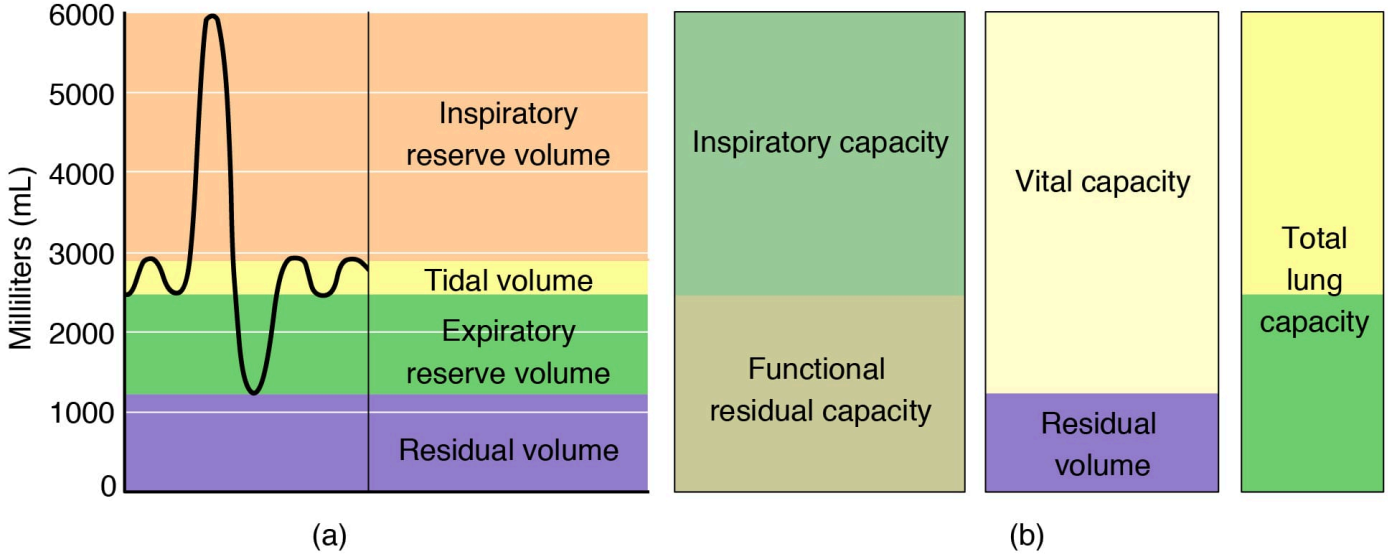


Figure 8.3.4: Respiratory Volumes and Capacities These two graphs show (a) respiratory volumes and (b) the combination of volumes that results in respiratory capacity. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.3.4.

Table 8.3.1: Pulmonary Function Tests

Pulmonary Function Test	Instrument	Measure	Function
Spirometry	Spirometer	Forced Vital Capacity (FVC)	Volume of air exhaled after maximum inhalation
Spirometry	Spirometer	Forced Expiratory Volume (FEV)	Volume of air exhaled during one forced breath
Spirometry	Spirometer	Forced Expiratory Flow, 25–75%	Airflow during the middle portion of exhalation
Spirometry	Spirometer	Peak Expiratory Flow (PEF)	Rate of exhalation
Spirometry	Spirometer	Maximum Voluntary Ventilation (MVV)	Volume of air inspired and expired in 1 minute
Spirometry	Spirometer	Slow Vital Capacity (SVC)	Volume of air slowly exhaled after inhaling beyond tidal volume
Spirometry	Spirometer	Total Lung Capacity (TLC)	Volume of air in the lungs after maximum inhalation
Spirometry	Spirometer	Functional Residual Capacity (FRC)	Volume of air remaining in the lungs after normal expiration
Spirometry	Spirometer	Residual Volume (RV)	Volume of air remaining in the lungs after maximum exhalation
Spirometry	Spirometer	Total Lung Capacity (TLC, Maximum)	Maximum volume of air the lungs can hold
Spirometry	Spirometer	Expiratory Reserve Volume (ERV)	Volume of air exhaled beyond normal expiration
Gas Diffusion	Blood Gas Analyzer	Arterial Blood Gases	Concentration of oxygen and carbon dioxide in the blood

Respiratory capacity is the combination of two or more selected volumes, which further describes the amount of air in the lungs during a given time. For example, total lung capacity (TLC) is the sum of all of the lung volumes (TV, ERV, IRV, and RV), which represents the total amount of air a person can hold in the lungs after a forceful inhalation. TLC is about 6000 mL air for males, and about 4200 mL for females. Vital capacity (VC) is the amount of air a person can move into or out of their lungs, and is the sum of all of the volumes except residual volume (TV, ERV, and IRV), which is between 4000 and 5000 milliliters. Inspiratory capacity (IC) is the maximum amount of air that can be inhaled past a normal tidal expiration, is the sum of the tidal volume and inspiratory reserve volume. On the other hand, the functional residual capacity (FRC) is the amount of air that remains in the lung after a normal tidal expiration; it is the sum of expiratory reserve volume and residual volume. In addition to the air that creates respiratory volumes, the respiratory system also contains anatomical dead space, which is air that is present in the airway that never reaches the alveoli and therefore never participates in gas exchange. Alveolar dead space involves air found within alveoli that are unable to function, such as those affected by disease or abnormal blood flow. Total dead space is the anatomical dead space and alveolar dead space together, and represents all of the air in the respiratory system that is not being used in the gas exchange process.

Respiratory Rate and Control of Ventilation

Breathing usually occurs without thought, although at times you can consciously control it, such as when you swim under water, sing a song, or blow bubbles. The respiratory rate is the total number of breaths, or respiratory cycles, that occur each minute. Respiratory rate can be an important indicator of disease, as the rate may increase or decrease during an illness or in a disease condition. The respiratory rate is controlled by the respiratory center located within the medulla oblongata in the brain, which responds primarily to changes in carbon dioxide, oxygen, and pH levels in the blood.

The normal respiratory rate of a child decreases from birth to adolescence. A child under 1 year of age has a normal respiratory rate between 30 and 60 breaths per minute, but by the time a child is about 10 years old, the normal rate is closer to 18 to 30. By adolescence, the normal respiratory rate is similar to that of adults, 12 to 18 breaths per minute.

Ventilation Control Centers

The control of ventilation is a complex interplay of multiple regions in the brain that signal the muscles used in pulmonary ventilation to contract (Table 8.3.2). The result is typically a rhythmic, consistent ventilation rate that provides the body with sufficient amounts of oxygen, while adequately removing carbon dioxide.

Table 8.3.2: Summary of Ventilation Regulation

System component	Function
Medullary respiratory center	Sets the basic rhythm of breathing
Ventral respiratory group (VRG)	Generates the breathing rhythm and integrates data coming into the medulla
Dorsal respiratory group (DRG)	Integrates input from the stretch receptors and the chemoreceptors in the periphery
Pontine respiratory group (PRG)	Influences and modifies the medulla oblongata's functions
Aortic body	Monitors blood PCO_2, PO_2, and pH
Carotid body	Monitors blood PCO_2, PO_2, and pH
Hypothalamus	Monitors emotional state and body temperature
Cortical areas of the brain	Control voluntary breathing
Proprioceptors	Send impulses regarding joint and muscle movements
Pulmonary irritant reflexes	Protect the respiratory zones of the system from foreign material
Inflation reflex	Protects the lungs from over-inflating

Neurons that innervate the muscles of the respiratory system are responsible for controlling and regulating pulmonary ventilation. The major brain centers involved in pulmonary ventilation are the medulla oblongata and the pontine respiratory group (Figure 8.3.5).

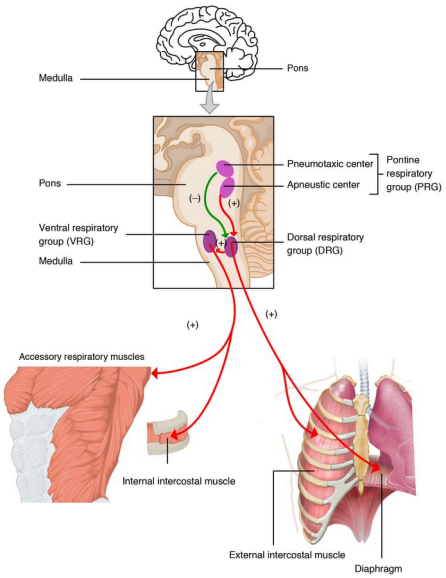


Figure 8.3.5: Respiratory Centers of the Brain. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.3.5.

The medulla oblongata contains the dorsal respiratory group (DRG) and the ventral respiratory group (VRG). The DRG is involved in maintaining a constant breathing rhythm by stimulating the diaphragm and intercostal muscles to contract, resulting in inspiration. When activity in the DRG ceases, it no longer stimulates the diaphragm and intercostals to contract, allowing them to relax, resulting in expiration. The VRG is involved in forced breathing, as the neurons in the VRG stimulate the accessory muscles involved in forced breathing to contract, resulting in forced inspiration. The VRG also stimulates the accessory muscles involved in forced expiration to contract.

The second respiratory center of the brain is located within the pons, called the pontine respiratory group, and consists of the apneustic and pneumotaxic centers. The apneustic center is a double cluster of neuronal cell bodies that stimulate neurons in the DRG, controlling the depth of inspiration, particularly for deep breathing. The pneumotaxic center is a network of neurons that inhibits the activity of neurons in the DRG, allowing relaxation after inspiration, and thus controlling the overall rate.

Factors That Affect the Rate and Depth of Respiration

The respiratory rate and the depth of inspiration are regulated by the medulla oblongata and pons; however, these regions of the brain do so in response to systemic stimuli. It is a dose-response, negative-feedback relationship in which the greater the stimulus, the greater the response. Thus, increasing stimuli results in forced breathing. Multiple systemic factors are involved in stimulating the brain to produce pulmonary ventilation.

The major factor that stimulates the medulla oblongata and pons to produce respiration is surprisingly not oxygen concentration, but rather the concentration of carbon dioxide in the blood. As you recall, carbon dioxide is a waste product of cellular respiration and can be toxic. Concentrations of chemicals are sensed by chemoreceptors. A central chemoreceptor is one of the specialized receptors that are located in the brain and brainstem, whereas a peripheral chemoreceptor is one of the specialized receptors located in the carotid arteries and aortic arch. Concentration changes in certain substances, such as carbon dioxide or hydrogen ions, stimulate these receptors, which in turn signal the respiration centers of the brain. In the case of carbon dioxide, as the concentration of CO_2 in the blood increases, it readily diffuses across the blood-brain barrier, where it collects in the extracellular fluid. As will be explained in more detail later, increased carbon dioxide levels lead to increased levels of hydrogen ions, decreasing pH. The increase in hydrogen ions in the brain triggers the central chemoreceptors to stimulate the respiratory centers to initiate contraction of the diaphragm and intercostal muscles. As a result, the rate and depth of respiration increase, allowing more carbon dioxide to be expelled, which brings more air into and out of the lungs promoting a reduction in the blood levels of carbon dioxide, and therefore hydrogen ions, in the blood. In contrast, low levels of carbon dioxide in the blood cause low levels of hydrogen ions in the brain, leading to a decrease in the rate and depth of pulmonary ventilation, producing shallow, slow breathing.

Another factor involved in influencing the respiratory activity of the brain is systemic arterial concentrations of hydrogen ions. Increasing carbon dioxide levels can lead to increased H^+ levels, as mentioned above, as well as other metabolic activities, such as lactic acid accumulation after strenuous exercise. Peripheral chemoreceptors of the aortic arch and carotid arteries sense arterial levels of hydrogen ions. When peripheral chemoreceptors sense decreasing, or more acidic, pH levels, they stimulate an increase in ventilation to remove carbon dioxide from the blood at a quicker rate. Removal of carbon dioxide from the blood helps to reduce hydrogen ions, thus increasing systemic pH.

Blood levels of oxygen are also important in influencing respiratory rate. The peripheral chemoreceptors are responsible for sensing large changes in blood oxygen levels. If blood oxygen levels become quite low—about 60 mm Hg or less—then peripheral chemoreceptors stimulate an increase in respiratory activity. The chemoreceptors are only able to sense dissolved oxygen molecules, not the oxygen that is bound to hemoglobin. As you recall, the majority of oxygen is bound by hemoglobin; when dissolved levels of oxygen drop, hemoglobin releases oxygen. Therefore, a large drop in oxygen levels is required to stimulate the chemoreceptors of the aortic arch and carotid arteries.

The hypothalamus and other brain regions associated with the limbic system also play roles in influencing the regulation of breathing by interacting with the respiratory centers. The hypothalamus and other regions associated with the limbic system are involved in regulating respiration in response to emotions, pain, and temperature. For example, an increase in body temperature causes an increase in respiratory rate. Feeling excited or the fight-or-flight response will also result in an increase in respiratory rate.

Disorders of the Respiratory System: Sleep Apnea

Sleep apnea is a chronic disorder that can occur in children or adults, and is characterized by the cessation of breathing during sleep. These episodes may last for several seconds or several minutes, and may differ in the frequency with which they are experienced. Sleep apnea leads to poor sleep, which is reflected in the symptoms of fatigue, evening napping, irritability, memory problems, and morning headaches. In addition, many individuals with sleep apnea experience a dry throat in the morning after waking from sleep, which may be due to excessive snoring.

There are two types of sleep apnea: obstructive sleep apnea and central sleep apnea. Obstructive sleep apnea is caused by an obstruction of the airway during sleep, which can occur at different points in the airway, depending on the underlying cause of the obstruction. For example, the tongue and throat muscles of some individuals with obstructive sleep apnea may relax excessively, causing the muscles to push into the airway. Another example is obesity, which is a known risk factor for sleep apnea, as excess adipose tissue in the neck region can push the soft tissues towards the lumen of the airway, causing the trachea to narrow.

In central sleep apnea, the respiratory centers of the brain do not respond properly to rising carbon dioxide levels and therefore do not stimulate the contraction of the diaphragm and intercostal muscles regularly. As a result, inspiration does not occur and breathing stops for a short period. In some cases, the cause of central sleep apnea is unknown. However, some medical conditions, such as stroke and congestive heart failure, may cause damage to the pons or medulla oblongata. In addition, some pharmacologic agents, such as morphine, can affect the respiratory centers, causing a decrease in the respiratory rate. The symptoms of central sleep apnea are similar to those of obstructive sleep apnea.

A diagnosis of sleep apnea is usually done during a sleep study, where the patient is monitored in a sleep laboratory for several nights. The patient's blood oxygen levels, heart rate, respiratory rate, and blood pressure are monitored, as are brain activity and the volume of air that is inhaled and exhaled. Treatment of sleep apnea commonly includes the use of a device called a continuous positive airway pressure (CPAP) machine during sleep. The CPAP machine has a mask that covers the nose, or the nose and mouth, and forces air into the airway at regular intervals. This pressurized air can help to gently force the airway to remain open, allowing more normal ventilation to occur. Other treatments include lifestyle changes to decrease weight, eliminate alcohol and other sleep apnea–promoting drugs, and changes in sleep position. In addition to these treatments, patients with central sleep apnea may need supplemental oxygen during sleep.

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8.4: Gas Exchange

Principles of Gas Exchange

The purpose of the respiratory system is to perform gas exchange. Pulmonary ventilation provides air to the alveoli for this gas exchange process. At the respiratory membrane, where the alveolar and capillary walls meet, gases move across the membranes, with oxygen entering the bloodstream and carbon dioxide exiting. It is through this mechanism that blood is oxygenated and carbon dioxide, the waste product of cellular respiration, is removed from the body.

In order to understand the mechanisms of gas exchange in the lung, it is important to understand the underlying principles of gases and their behavior. In addition to Boyle's law, several other gas laws help to describe the behavior of gases.

Gas Laws and Air Composition

Gas molecules exert force on the surfaces with which they are in contact; this force is called pressure. In natural systems, gases are normally present as a mixture of different types of molecules. For example, the atmosphere consists of oxygen, nitrogen, carbon dioxide, and other gaseous molecules, and this gaseous mixture exerts a certain pressure referred to as atmospheric pressure (Table 8.4.1). Partial pressure (P_x) is the pressure of a single type of gas in a mixture of gases. For example, in the atmosphere, oxygen exerts a partial pressure, and nitrogen exerts another partial pressure, independent of the partial pressure of oxygen (Figure 8.4.1). Total pressure is the sum of all the partial pressures of a gaseous mixture. Dalton's law describes the behavior of nonreactive gases in a gaseous mixture and states that a specific gas type in a mixture exerts its own pressure; thus, the total pressure exerted by a mixture of gases is the sum of the partial pressures of the gases in the mixture.

Table 8.4.1: Partial Pressures of Atmospheric Gases

Gas	Percent of total composition	Partial pressure (mm Hg)
Nitrogen (N_2)	78.6	597.4
Oxygen (O_2)	20.9	158.8
Water (H_2O)	0.4	3.0
Carbon dioxide (CO_2)	0.04	0.3
Others	0.06	0.5
Total composition/total atmospheric pressure	100%	760.0

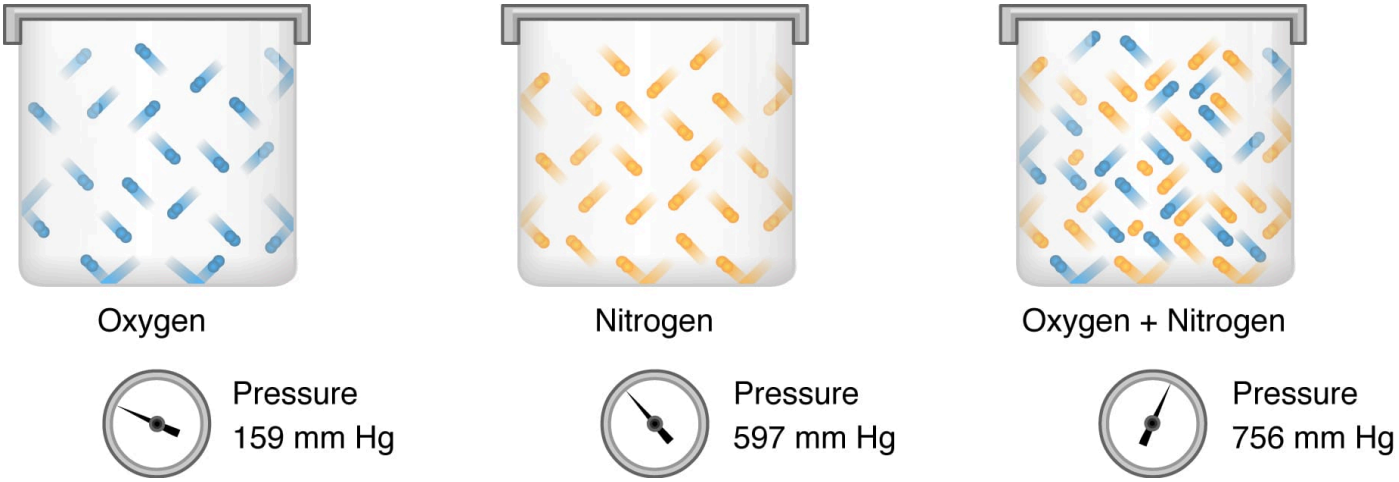


Figure 8.4.1: Partial pressure is the force exerted by a gas. The sum of the partial pressures of all the gases in a mixture equals the total pressure. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.4.1.

Partial pressure is extremely important in predicting the movement of gases. Recall that gases tend to equalize their pressure in two regions that are connected. A gas will move from an area where its partial pressure is higher to an area where its partial pressure is lower. In addition, the greater the partial pressure difference between the two areas, the more rapid is the movement of gases.

Solubility of Gases in Liquids

Henry's law describes the behavior of gases when they come into contact with a liquid, such as blood. Henry's law states that the concentration of gas in a liquid is directly proportional to the solubility and partial pressure of that gas. The greater the partial pressure of the gas, the greater the number of gas molecules that will dissolve in the liquid. The concentration of the gas in a liquid is also dependent on the solubility of the gas in the liquid. For example, although nitrogen is present in the atmosphere, very little nitrogen dissolves into the blood, because the solubility of nitrogen in blood is very low. The exception to this occurs in scuba divers; the composition of the compressed air that divers breathe causes nitrogen to have a higher partial pressure than normal, causing it to dissolve in the blood in greater amounts than normal. Too much nitrogen in the bloodstream results in a serious condition that can be fatal if not corrected. Gas molecules establish an equilibrium between those molecules dissolved in liquid and those in air.

The composition of air in the atmosphere and in the alveoli differs. In both cases, the relative concentration of gases is nitrogen > oxygen > water vapor > carbon dioxide. The amount of water vapor present in alveolar air is greater than that in atmospheric air (Table 8.4.2). Recall that the respiratory system works to humidify incoming air, thereby causing the air present in the alveoli to have a greater amount of water vapor than atmospheric air. In addition, alveolar air contains a greater amount of carbon dioxide and less oxygen than atmospheric air. This is no surprise, as gas exchange removes oxygen from and adds carbon dioxide to alveolar air. Both deep and forced breathing cause the alveolar air composition to be changed more rapidly than during quiet breathing. As a result, the partial pressures of oxygen and carbon dioxide change, affecting the diffusion process that moves these materials across the membrane. This will cause oxygen to enter and carbon dioxide to leave the blood more quickly.

Table 8.4.2: Composition and Partial Pressures of Alveolar Air

Gas	Percent of total composition	Partial pressure (mm Hg)
Nitrogen (N_2)	74.9	569
Oxygen (O_2)	13.7	104
Water (H_2O)	6.2	40
Carbon dioxide (CO_2)	5.2	47
Total composition/total alveolar pressure	100%	760.0

Ventilation and Perfusion

Two important aspects of gas exchange in the lung are ventilation and perfusion. Ventilation is the movement of air into and out of the lungs, and perfusion is the flow of blood in the pulmonary capillaries. For gas exchange to be efficient, the volumes involved in ventilation and perfusion should be compatible. However, factors such as regional gravity effects on blood, blocked alveolar ducts, or disease can cause ventilation and perfusion to be imbalanced.

The partial pressure of oxygen in alveolar air is about 104 mm Hg, whereas the partial pressure of oxygenated blood in pulmonary veins is about 100 mm Hg. When ventilation is sufficient, oxygen enters the alveoli at a high rate, and the partial pressure of oxygen in the alveoli remains high. In contrast, when ventilation is insufficient, the partial pressure of oxygen in the alveoli drops. Without the large difference in partial pressure between the alveoli and the blood, oxygen does not diffuse efficiently across the respiratory membrane. The body has mechanisms that counteract this problem. In cases when ventilation is not sufficient for an alveolus, the body redirects blood flow to alveoli that are receiving sufficient ventilation. This is achieved by constricting the pulmonary arterioles that serves the dysfunctional alveolus, which redirects blood to other alveoli that have sufficient ventilation. At the same time, the pulmonary arterioles that serve alveoli receiving sufficient ventilation vasodilate, which brings in greater blood flow. Factors such as carbon dioxide, oxygen, and pH levels can all serve as stimuli for adjusting blood flow in the capillary networks associated with the alveoli.

Ventilation is regulated by the diameter of the airways, whereas perfusion is regulated by the diameter of the blood vessels. The

diameter of the bronchioles is sensitive to the partial pressure of carbon dioxide in the alveoli. A greater partial pressure of carbon dioxide in the alveoli causes the bronchioles to increase their diameter as will a decreased level of oxygen in the blood supply, allowing carbon dioxide to be exhaled from the body at a greater rate. As mentioned above, a greater partial pressure of oxygen in the alveoli causes the pulmonary arterioles to dilate, increasing blood flow.

Gas Exchange

Gas exchange occurs at two sites in the body: in the lungs, where oxygen is picked up and carbon dioxide is released at the respiratory membrane, and at the tissues, where oxygen is released and carbon dioxide is picked up. External respiration is the exchange of gases with the external environment, and occurs in the alveoli of the lungs. Internal respiration is the exchange of gases with the internal environment, and occurs in the tissues. The actual exchange of gases occurs due to simple diffusion. Energy is not required to move oxygen or carbon dioxide across membranes. Instead, these gases follow pressure gradients that allow them to diffuse. The anatomy of the lung maximizes the diffusion of gases: The respiratory membrane is highly permeable to gases; the respiratory and blood capillary membranes are very thin; and there is a large surface area throughout the lungs.

External Respiration

The pulmonary artery carries deoxygenated blood into the lungs from the heart, where it branches and eventually becomes the capillary network composed of pulmonary capillaries. These pulmonary capillaries create the respiratory membrane with the alveoli (Figure 8.4.2). As the blood is pumped through this capillary network, gas exchange occurs. Although a small amount of the oxygen is able to dissolve directly into plasma from the alveoli, most of the oxygen is picked up by erythrocytes (red blood cells) and binds to a protein called hemoglobin, a process described later in this chapter. Oxygenated hemoglobin is red, causing the overall appearance of bright red oxygenated blood, which returns to the heart through the pulmonary veins. Carbon dioxide is released in the opposite direction of oxygen, from the blood to the alveoli. Some of the carbon dioxide is returned on hemoglobin, but can also be dissolved in plasma or is present as a converted form, also explained in greater detail later in this chapter.

External respiration occurs as a function of partial pressure differences in oxygen and carbon dioxide between the alveoli and the blood in the pulmonary capillaries.

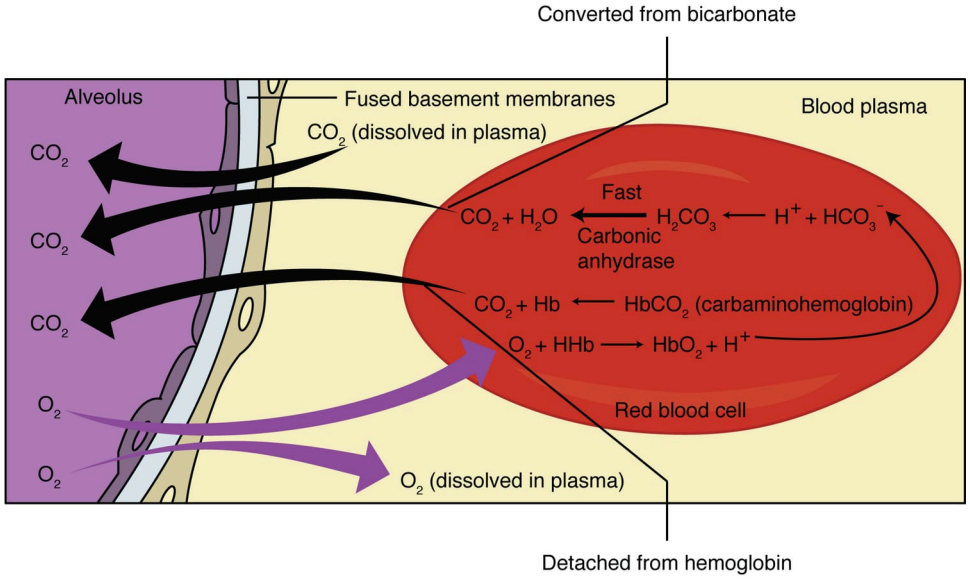


Figure 8.4.2: In external respiration, oxygen diffuses across the respiratory membrane from the alveolus to the capillary, whereas carbon dioxide diffuses out of the capillary into the alveolus. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.4.2.

Although the solubility of oxygen in blood is not high, there is a drastic difference in the partial pressure of oxygen in the alveoli versus in the blood of the pulmonary capillaries. This difference is about 64 mm Hg: The partial pressure of oxygen in the alveoli is about 104 mm Hg, whereas its partial pressure in the blood of the capillary is about 40 mm Hg. This large difference in partial pressure creates a very strong pressure gradient that causes oxygen to rapidly cross the respiratory membrane from the alveoli into the blood.

The partial pressure of carbon dioxide is also different between the alveolar air and the blood of the capillary. However, the

partial pressure difference is less than that of oxygen, about 5 mm Hg. The partial pressure of carbon dioxide in the blood of the capillary is about 45 mm Hg, whereas its partial pressure in the alveoli is about 40 mm Hg. However, the solubility of carbon dioxide is much greater than that of oxygen—by a factor of about 20—in both blood and alveolar fluids. As a result, the relative concentrations of oxygen and carbon dioxide that diffuse across the respiratory membrane are similar.

Internal Respiration

Internal respiration is gas exchange that occurs at the level of body tissues (Figure 8.4.3). Similar to external respiration, internal respiration also occurs as simple diffusion due to a partial pressure gradient. However, the partial pressure gradients are opposite of those present at the respiratory membrane. The partial pressure of oxygen in tissues is low, about 40 mm Hg, because oxygen is continuously used for cellular respiration. In contrast, the partial pressure of oxygen in the blood is about 100 mm Hg. This creates a pressure gradient that causes oxygen to dissociate from hemoglobin, diffuse out of the blood, cross the interstitial space, and enter the tissue. Hemoglobin that has little oxygen bound to it loses much of its brightness, so that blood returning to the heart is more burgundy in color.

Considering that cellular respiration continuously produces carbon dioxide, the partial pressure of carbon dioxide is lower in the blood than it is in the tissue, causing carbon dioxide to diffuse out of the tissue, cross the interstitial fluid, and enter the blood. It is then carried back to the lungs either bound to hemoglobin, dissolved in plasma, or in a converted form. By the time blood returns to the heart, the partial pressure of oxygen has returned to about 40 mm Hg, and the partial pressure of carbon dioxide has returned to about 45 mm Hg. The blood is then pumped back to the lungs to be oxygenated once again during external respiration.

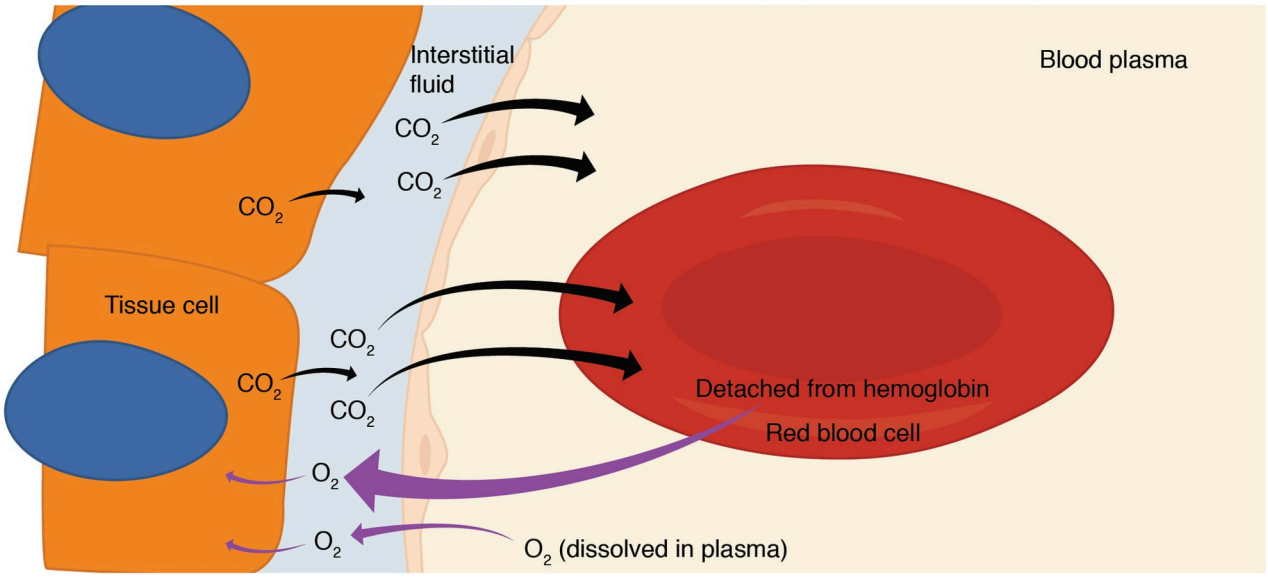


Figure 8.4.3: Oxygen diffuses out of the capillary and into cells, whereas carbon dioxide diffuses out of cells and into the capillary. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.4.3.

Everyday Connection

Hyperbaric Chamber Treatment

A type of device used in some areas of medicine that exploits the behavior of gases is hyperbaric chamber treatment. A hyperbaric chamber is a unit that can be sealed and expose a patient to either 100 percent oxygen with increased pressure or a mixture of gases that includes a higher concentration of oxygen than normal atmospheric air, also at a higher partial pressure than the atmosphere. There are two major types of chambers: monoplace and multiplace. Monoplace chambers are typically for one patient, and the staff tending to the patient observes the patient from outside of the chamber (Figure 22.24). Some facilities have special monoplace hyperbaric chambers that allow multiple patients to be treated at once, usually in a sitting or reclining position, to help ease feelings of isolation or claustrophobia. Multiplace chambers are large enough for multiple patients to be treated at one time, and the staff attending these patients is present inside the chamber. In a multiplace chamber, patients are often treated with air via a mask or hood, and the chamber is pressurized.



Figure 8.4.4: Hyperbaric Chamber. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.4.4.

Hyperbaric chamber treatment is based on the behavior of gases. As you recall, gases move from a region of higher partial pressure to a region of lower partial pressure. In a hyperbaric chamber, the atmospheric pressure is increased, causing a greater amount of oxygen than normal to diffuse into the bloodstream of the patient. Hyperbaric chamber therapy is used to treat a variety of medical problems, such as wound and graft healing, anaerobic bacterial infections, and carbon monoxide poisoning. Exposure to and poisoning by carbon monoxide is difficult to reverse, because hemoglobin's affinity for carbon monoxide is much stronger than its affinity for oxygen, causing carbon monoxide to replace oxygen in the blood. Hyperbaric chamber therapy can treat carbon monoxide poisoning, because the increased atmospheric pressure causes more oxygen to diffuse into the bloodstream. At this increased pressure and increased concentration of oxygen, carbon monoxide is displaced from hemoglobin. Another example is the treatment of anaerobic bacterial infections, which are created by bacteria that cannot or prefer not to live in the presence of oxygen. An increase in blood and tissue levels of oxygen helps to kill the anaerobic bacteria that are responsible for the infection, as oxygen is toxic to anaerobic bacteria. For wounds and grafts, the chamber stimulates the healing process by increasing energy production needed for repair. Increasing oxygen transport allows cells to ramp up cellular respiration and thus ATP production, the energy needed to build new structures.

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8.5: Transport of Gases

Oxygen Transport in the Blood

The other major activity in the lungs is the process of respiration, the process of gas exchange. The function of respiration is to provide oxygen for use by body cells during cellular respiration and to eliminate carbon dioxide, a waste product of cellular respiration, from the body. In order for the exchange of oxygen and carbon dioxide to occur, both gases must be transported between the external and internal respiration sites. Although carbon dioxide is more soluble than oxygen in blood, both gases require a specialized transport system for the majority of the gas molecules to be moved between the lungs and other tissues.

Even though oxygen is transported via the blood, you may recall that oxygen is not very soluble in liquids. A small amount of oxygen does dissolve in the blood and is transported in the bloodstream, but it is only about 1.5% of the total amount. The majority of oxygen molecules are carried from the lungs to the body's tissues by a specialized transport system, which relies on the erythrocyte—the red blood cell. Erythrocytes contain a metalloprotein, hemoglobin, which serves to bind oxygen molecules to the erythrocyte (Figure 8.5.1). Heme is the portion of hemoglobin that contains iron, and it is heme that binds oxygen. Each hemoglobin molecule contains four iron-containing heme molecules, and because of this, one hemoglobin molecule is capable of carrying up to four molecules of oxygen. As oxygen diffuses across the respiratory membrane from the alveolus to the capillary, it also diffuses into the red blood cell and is bound by hemoglobin. The following reversible chemical reaction describes the production of the final product, oxyhemoglobin (HbO_2), which is formed when oxygen binds to hemoglobin. Oxyhemoglobin is a bright red-colored molecule that contributes to the bright red color of oxygenated blood.

$$\mathrm{Hb} + \mathrm{O_2} \rightleftharpoons \mathrm{HbO_2}$$

In this formula, Hb represents reduced hemoglobin, that is, hemoglobin that does not have oxygen bound to it. There are multiple factors involved in how readily heme binds to and dissociates from oxygen, which will be discussed in the subsequent sections.

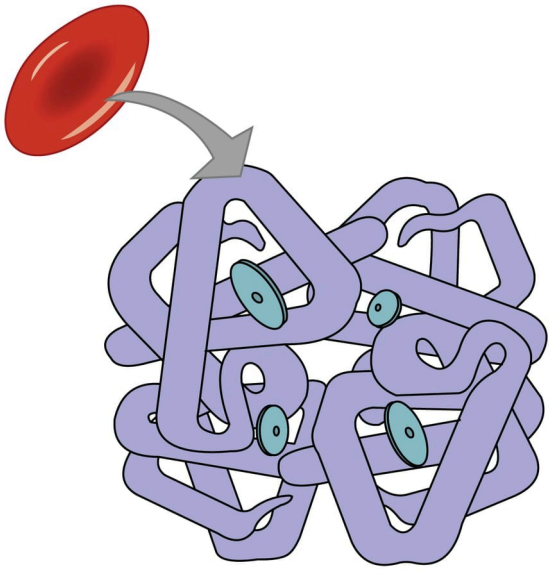


Figure 8.5.1: Hemoglobin consists of four subunits, each of which contains one molecule of iron. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.5.1.

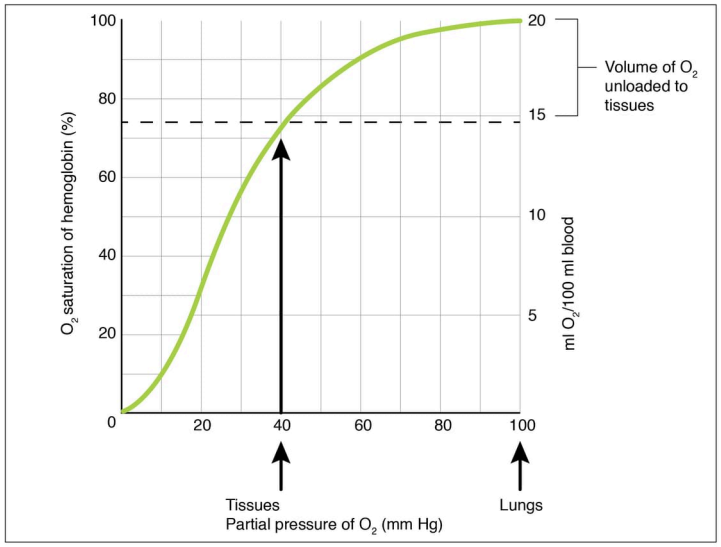
Function of Hemoglobin

Hemoglobin is composed of subunits, a protein structure that is referred to as a quaternary structure. Each of the four subunits that make up hemoglobin is arranged in a ring-like fashion, with an iron atom covalently bound to the heme in the center of each subunit. Binding of the first oxygen molecule causes a conformational change in hemoglobin that allows the second molecule of oxygen to bind more readily. As each molecule of oxygen is bound, it further facilitates the binding of the next molecule, until all four heme sites are occupied by oxygen. The opposite occurs as well: After the first oxygen molecule dissociates and is "dropped

off" at the tissues, the next oxygen molecule dissociates more readily. When all four heme sites are occupied, the hemoglobin is said to be saturated. When one to three heme sites are occupied, the hemoglobin is said to be partially saturated. Therefore, when considering the blood as a whole, the percent of the available heme units that are bound to oxygen at a given time is called hemoglobin saturation. Hemoglobin saturation of 100 percent means that every heme unit in all of the erythrocytes of the body is bound to oxygen. In a healthy individual with normal hemoglobin levels, hemoglobin saturation generally ranges from 95 percent to 99 percent.

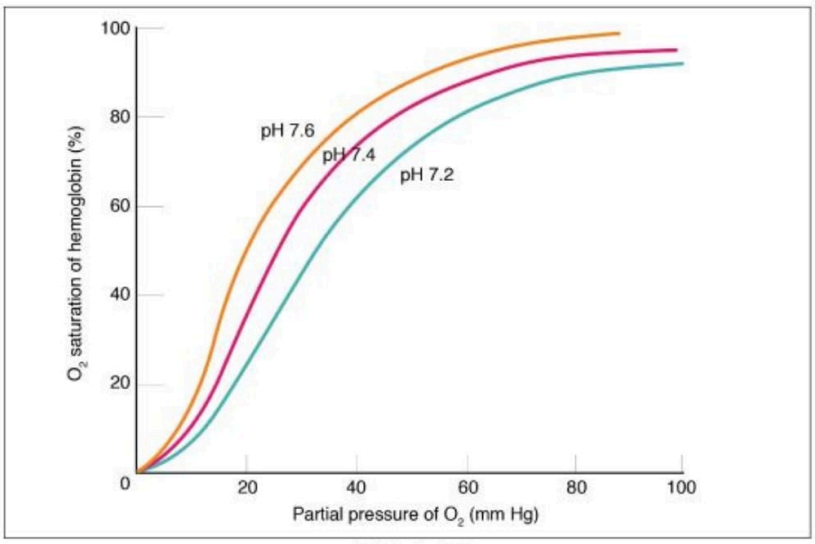
Oxygen Dissociation from Hemoglobin

Partial pressure is an important aspect of the binding of oxygen to and disassociation from heme. An oxygen–hemoglobin dissociation curve is a graph that describes the relationship of partial pressure to the binding of oxygen to heme and its subsequent dissociation from heme. Remember that gases travel from an area of higher partial pressure to an area of lower partial pressure. In addition, the affinity of an oxygen molecule for heme increases as more oxygen molecules are bound. Therefore, in the oxygen–hemoglobin saturation curve, as the partial pressure of oxygen increases, a proportionately greater number of oxygen molecules are bound by heme. Not surprisingly, the oxygen–hemoglobin saturation/dissociation curve also shows that the lower the partial pressure of oxygen, the fewer oxygen molecules are bound to heme. As a result, the partial pressure of oxygen plays a major role in determining the degree of binding of oxygen to heme at the site of the respiratory membrane, as well as the degree of dissociation of oxygen from heme at the site of body tissues.



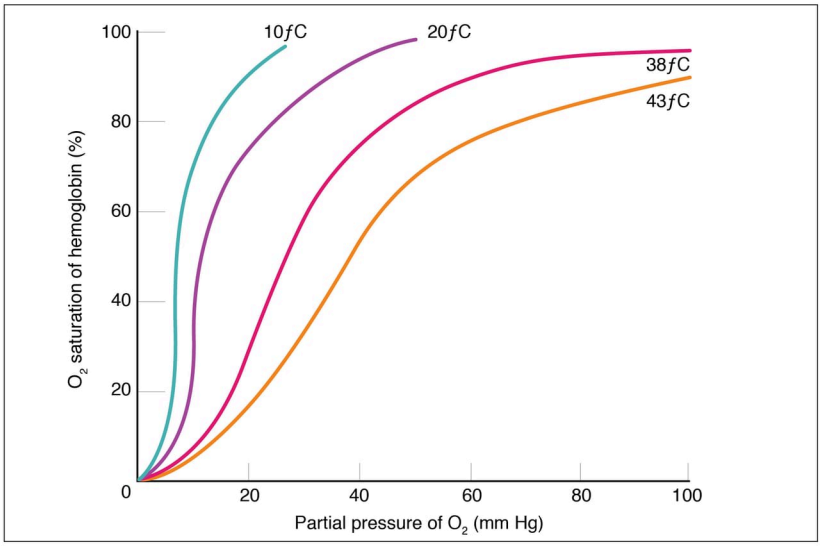
(a) Partial pressure of oxygen and hemoglobin saturation

Figure 8.5.2: Oxygen-hemoglobin dissociation curve showing the relationship between the partial pressure of oxygen (PO_2) and hemoglobin oxygen saturation. The curve demonstrates how oxygen loading occurs in the lungs and oxygen unloading occurs in the tissues. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.5.2.



(b) Effect of pH

Figure 8.5.3: Decreases in blood pH shift the curve to the right, reducing hemoglobin's affinity for oxygen and promoting oxygen release to tissues, whereas increases in pH shift the curve to the left, increasing oxygen affinity and reducing oxygen unloading. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.5.3.



(c) Effect of temperature

Figure 8.5.4: Increasing temperature shifts the curve to the right, reducing hemoglobin's affinity for oxygen and promoting oxygen unloading to tissues, whereas decreasing temperature shifts the curve to the left, increasing oxygen affinity and reducing oxygen release. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.5.4.

The mechanisms behind the oxygen–hemoglobin saturation/dissociation curve also serve as automatic control mechanisms that regulate how much oxygen is delivered to different tissues throughout the body. This is important because some tissues have a higher metabolic rate than others. Highly active tissues, such as muscle, rapidly use oxygen to produce ATP, lowering the partial pressure of oxygen in the tissue to about 20 mm Hg. The partial pressure of oxygen inside capillaries is about 100 mm Hg, so the difference between the two becomes quite high, about 80 mm Hg. As a result, a greater number of oxygen molecules dissociate from hemoglobin and enter the tissues. The reverse is true of tissues, such as adipose (body fat), which have lower metabolic rates. Because less oxygen is used by these cells, the partial pressure of oxygen within such tissues remains relatively high, resulting in fewer oxygen molecules dissociating from hemoglobin and entering the tissue interstitial fluid. Although venous blood is said to be deoxygenated, some oxygen is still bound to hemoglobin in its red blood cells. This provides an oxygen reserve that can be used when tissues suddenly demand more oxygen.

Factors other than partial pressure also affect the oxygen–hemoglobin saturation/dissociation curve. For example, a higher temperature promotes hemoglobin and oxygen to dissociate faster, whereas a lower temperature inhibits dissociation (see Figure

8.5.3). However, the human body tightly regulates temperature, so this factor may not affect gas exchange throughout the body. The exception to this is in highly active tissues, which may release a larger amount of energy than is given off as heat. As a result, oxygen readily dissociates from hemoglobin, which is a mechanism that helps to provide active tissues with more oxygen.

Certain hormones, such as androgens, epinephrine, thyroid hormones, and growth hormone, can affect the oxygen–hemoglobin saturation/disassociation curve by stimulating the production of a compound called 2,3-bisphosphoglycerate (BPG) by erythrocytes. BPG is a byproduct of glycolysis. Because erythrocytes do not contain mitochondria, glycolysis is the sole method by which these cells produce ATP. BPG promotes the disassociation of oxygen from hemoglobin. Therefore, the greater the concentration of BPG, the more readily oxygen dissociates from hemoglobin, despite its partial pressure.

The pH of the blood is another factor that influences the oxygen–hemoglobin saturation/dissociation curve (see Figure 8.5.2). The Bohr effect is a phenomenon that arises from the relationship between pH and oxygen's affinity for hemoglobin: A lower, more acidic pH promotes oxygen dissociation from hemoglobin. In contrast, a higher, or more basic, pH inhibits oxygen dissociation from hemoglobin. The greater the amount of carbon dioxide in the blood, the more molecules that must be converted, which in turn generates hydrogen ions and thus lowers blood pH. Furthermore, blood pH may become more acidic when certain byproducts of cell metabolism, such as lactic acid, carbonic acid, and carbon dioxide, are released into the bloodstream.

Hemoglobin of the Fetus

The fetus has its own circulation with its own erythrocytes; however, it is dependent on the pregnant person for oxygen. Blood is supplied to the fetus by way of the umbilical cord, which is connected to the placenta and separated from maternal blood by the chorion. The mechanism of gas exchange at the chorion is similar to gas exchange at the respiratory membrane. However, the partial pressure of oxygen is lower in the maternal blood in the placenta, at about 35 to 50 mm Hg, than it is in maternal arterial blood. The difference in partial pressures between maternal and fetal blood is not large, as the partial pressure of oxygen in fetal blood at the placenta is about 20 mm Hg. Therefore, there is not as much diffusion of oxygen into the fetal blood supply. The fetus' hemoglobin overcomes this problem by having a greater affinity for oxygen than maternal hemoglobin (Figure 8.5.5). Both fetal and adult hemoglobin have four subunits, but two of the subunits of fetal hemoglobin have a different structure that causes fetal hemoglobin to have a greater affinity for oxygen than does adult hemoglobin.

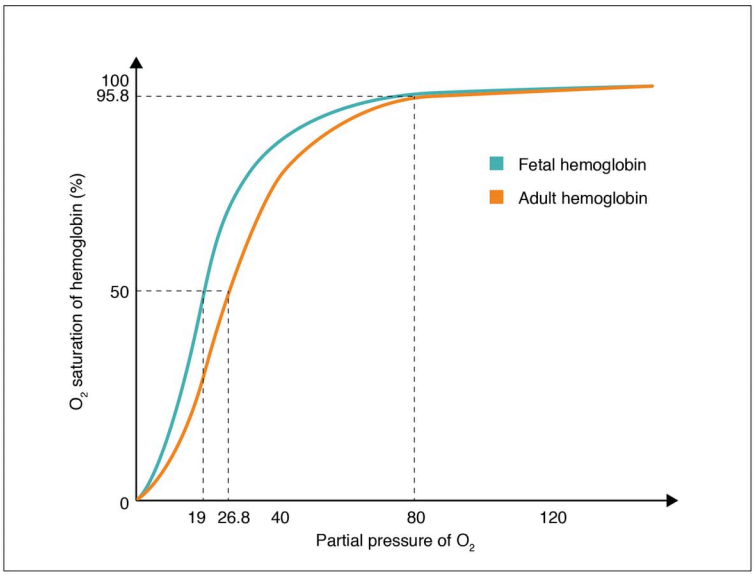


Figure 8.5.5 Oxygen-Hemoglobin Dissociation Curves in Fetus and Adult Fetal hemoglobin has a greater affinity for oxygen than does adult hemoglobin. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.5.5.

Carbon Dioxide Transport in the Blood

Carbon dioxide is transported by three major mechanisms. The first mechanism of carbon dioxide transport is by blood plasma, as some carbon dioxide molecules dissolve in the blood. The second mechanism is transport in the form of bicarbonate (HCO_3^-), which also dissolves in plasma. The third mechanism of carbon dioxide transport is similar to the transport of oxygen by erythrocytes (Figure 8.5.6).

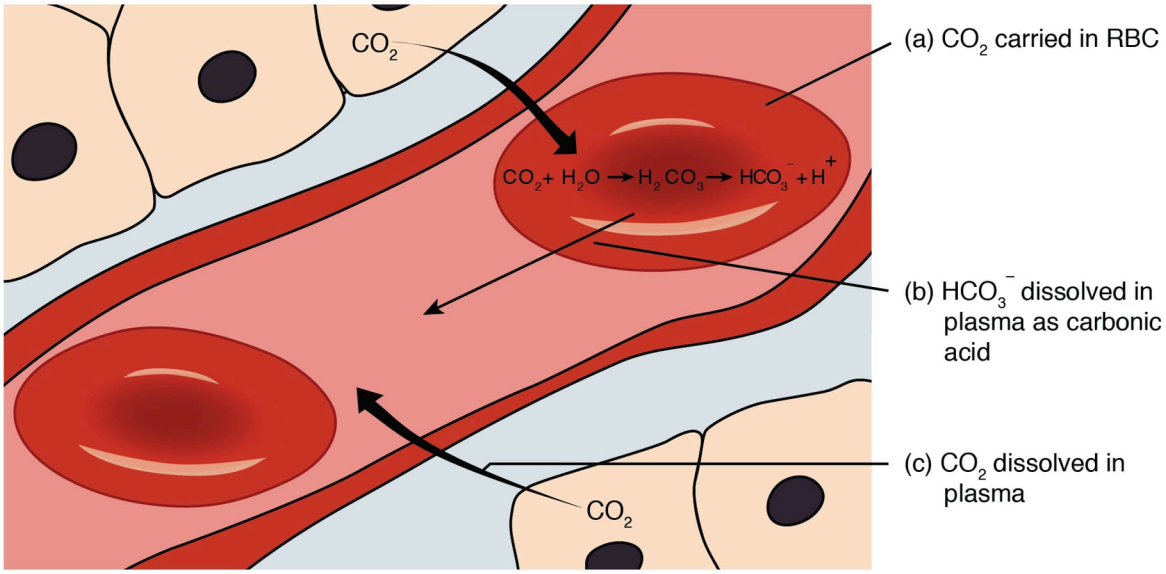


Figure 8.5.6: Carbon dioxide is transported by three different methods: (a) in erythrocytes; (b) after forming carbonic acid (H_2CO_3), which is dissolved in plasma; (c) and in plasma. (CC BY 4.0; via LibreTexts). Alternate text description of the Figure 8.5.6.

Dissolved Carbon Dioxide

Although carbon dioxide is not considered to be highly soluble in blood, a small fraction—about 7 to 10 percent—of the carbon dioxide that diffuses into the blood from the tissues dissolves in plasma. The dissolved carbon dioxide then travels in the bloodstream and when the blood reaches the pulmonary capillaries, the dissolved carbon dioxide diffuses across the respiratory membrane into the alveoli, where it is then exhaled during pulmonary ventilation.

Bicarbonate Buffer

A large fraction—about 70 percent—of the carbon dioxide molecules that diffuse into the blood is transported to the lungs as bicarbonate. Most bicarbonate is produced in erythrocytes after carbon dioxide diffuses into the capillaries, and subsequently into red blood cells. Carbonic anhydrase (CA) causes carbon dioxide and water to form carbonic acid (H_2CO_3), which dissociates into two ions: bicarbonate ions (HCO_3^-) and hydrogen ions (H^+). The following formula depicts this reaction:

$$CO_2 + H_2O \xrightarrow{CA} H_2CO_3 \rightleftharpoons H^+ + CO_3^-$$

Bicarbonate tends to build up in the erythrocytes, so that there is a greater concentration of bicarbonate in the erythrocytes than in the surrounding blood plasma. As a result, some of the bicarbonate will leave the erythrocytes and move down its concentration gradient into the plasma in exchange for chloride (Cl^-) ions. This phenomenon is referred to as the **chloride shift** and occurs because by exchanging one negative ion for another negative ion, neither the electrical charge of the erythrocytes nor that of the blood is altered.

At the pulmonary capillaries, the chemical reaction that produced bicarbonate (shown above) is reversed, and carbon dioxide and water are the products. Much of the bicarbonate in the plasma re-enters the erythrocytes in exchange for chloride ions. Hydrogen ions and bicarbonate ions join to form carbonic acid, which is converted into carbon dioxide and water by carbonic anhydrase. Carbon dioxide diffuses out of the erythrocytes and into the plasma, where it can further diffuse across the respiratory membrane into the alveoli to be exhaled during pulmonary ventilation.

Carbaminohemoglobin

About 20 percent of carbon dioxide is bound by hemoglobin and is transported to the lungs. Carbon dioxide does not bind to iron as oxygen does; instead, carbon dioxide binds amino acid moieties on the globin portions of hemoglobin to form carbaminohemoglobin, which forms when hemoglobin and carbon dioxide bind. When hemoglobin is not transporting oxygen, it tends to have a bluish-purple tone to it, creating the darker maroon color typical of deoxygenated blood. The following formula depicts this reversible reaction:

$$CO_2 + Hb \rightleftharpoons HbCO_2$$

Similar to the transport of oxygen by heme, the binding and dissociation of carbon dioxide to and from hemoglobin is dependent on the partial pressure of carbon dioxide. Because carbon dioxide is released from the lungs, blood that leaves the lungs and reaches body tissues has a lower partial pressure of carbon dioxide than is found in the tissues. As a result, carbon dioxide leaves the tissues because of its higher partial pressure, enters the blood, and then moves into red blood cells, binding to hemoglobin. In contrast, in the pulmonary capillaries, the partial pressure of carbon dioxide is high compared to within the alveoli. As a result, carbon dioxide dissociates readily from hemoglobin and diffuses across the respiratory membrane into the air.

In addition to the partial pressure of carbon dioxide, the oxygen saturation of hemoglobin and the partial pressure of oxygen in the blood also influence the affinity of hemoglobin for carbon dioxide. The Haldane effect is a phenomenon that arises from the relationship between the partial pressure of oxygen and the affinity of hemoglobin for carbon dioxide. Hemoglobin that is saturated with oxygen does not readily bind carbon dioxide. However, when oxygen is not bound to heme and the partial pressure of oxygen is low, hemoglobin readily binds to carbon dioxide.

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8.6: Modifications in Respiratory Functions

Oxygen Transport by Hemoglobin

At rest, the respiratory system performs its functions at a constant, rhythmic pace, as regulated by the respiratory centers of the brain. At this pace, ventilation provides sufficient oxygen to all the tissues of the body. However, there are times that the respiratory system must alter the pace of its functions in order to accommodate the oxygen demands of the body.

Hyperpnea

Hyperpnea is an increased depth and rate of ventilation to meet an increase in oxygen demand as might be seen in exercise or disease, particularly diseases that target the respiratory or digestive tracts. This does not significantly alter blood oxygen or carbon dioxide levels, but merely increases the depth and rate of ventilation to meet the demand of the cells. In contrast, hyperventilation is an increased ventilation rate that is independent of the cellular oxygen needs and leads to abnormally low blood carbon dioxide levels and high (alkaline) blood pH.

Interestingly, exercise does not cause hyperpnea as one might think. Muscles that perform work during exercise do increase their demand for oxygen, stimulating an increase in ventilation. However, hyperpnea during exercise appears to occur before a drop in oxygen levels within the muscles can occur. Therefore, hyperpnea must be driven by other mechanisms, either instead of or in addition to a drop in oxygen levels. The exact mechanisms behind exercise hyperpnea are not well understood, and some hypotheses are somewhat controversial. However, in addition to low oxygen, high carbon dioxide, and low pH levels, there appears to be a complex interplay of factors related to the nervous system and the respiratory centers of the brain.

First, a conscious decision to partake in exercise, or another form of physical exertion, results in a psychological stimulus that may trigger the respiratory centers of the brain to increase ventilation. In addition, the respiratory centers of the brain may be stimulated through the activation of motor neurons that innervate muscle groups that are involved in the physical activity. Finally, physical exertion stimulates proprioceptors, which are receptors located within the muscles, joints, and tendons, which sense movement and stretching; proprioceptors thus create a stimulus that may also trigger the respiratory centers of the brain. These neural factors are consistent with the sudden increase in ventilation that is observed immediately as exercise begins. Because the respiratory centers are stimulated by psychological, motor neuron, and proprioceptor inputs throughout exercise, the fact that there is also a sudden decrease in ventilation immediately after the exercise ends when these neural stimuli cease, further supports the idea that they are involved in triggering the changes of ventilation.

High Altitude Effects

An increase in altitude results in a decrease in atmospheric pressure. Although the proportion of oxygen relative to gases in the atmosphere remains at 21 percent, its partial pressure decreases (Table 8.6.1). As a result, it is more difficult for a body to achieve the same level of oxygen saturation at high altitude than at low altitude, due to lower atmospheric pressure. In fact, hemoglobin saturation is lower at high altitudes compared to hemoglobin saturation at sea level. For example, hemoglobin saturation is about 67 percent at 19,000 feet above sea level, whereas it reaches about 98 percent at sea level.

Table 8.6.1: Partial Pressure of Oxygen at Different Altitudes

Example location	Altitude (feet above sea level)	Atmospheric pressure (mm Hg)	Partial pressure of oxygen (mm Hg)
New York City, New York	0	760	159
Boulder, Colorado	5000	632	133
Aspen, Colorado	8000	565	118
Pike's Peak, Colorado	14,000	447	94
Denali (Mt. McKinley), Alaska	20,000	350	73

Example location	Altitude (feet above sea level)	Atmospheric pressure (mm Hg)	Partial pressure of oxygen (mm Hg)
Mt. Everest	29,032	253	53

As you recall, partial pressure is extremely important in determining how much gas can cross the respiratory membrane and enter the blood of the pulmonary capillaries. A lower partial pressure of oxygen means that there is a smaller difference in partial pressures between the alveoli and the blood, so less oxygen crosses the respiratory membrane. As a result, fewer oxygen molecules are bound by hemoglobin. Despite this, the tissues of the body still receive a sufficient amount of oxygen during rest at high altitudes. This is due to two major mechanisms. First, the number of oxygen molecules that enter the tissue from the blood is nearly equal between sea level and high altitudes. At sea level, hemoglobin saturation is higher, but only a quarter of the oxygen molecules are actually released into the tissue. At high altitudes, a greater proportion of molecules of oxygen are released into the tissues. Secondly, at high altitudes, a greater amount of BPG is produced by erythrocytes, which enhances the dissociation of oxygen from hemoglobin. Physical exertion, such as skiing or hiking, can lead to altitude sickness due to the low amount of oxygen reserves in the blood at high altitudes. At sea level, there is a large amount of oxygen reserve in venous blood (even though venous blood is thought of as “deoxygenated”) from which the muscles can draw during physical exertion. Because the oxygen saturation is much lower at higher altitudes, this venous reserve is small, resulting in pathological symptoms of low blood oxygen levels. You may have heard that it is important to drink more water when traveling at higher altitudes than you are accustomed to. This is because your body will increase micturition (urination) at high altitudes to counteract the effects of lower oxygen levels. By removing fluids, blood plasma levels drop but not the total number of erythrocytes. In this way, the overall concentration of erythrocytes in the blood increases, which helps tissues obtain the oxygen they need.

Acute mountain sickness (AMS), or altitude sickness, is a condition that results from acute exposure to high altitudes due to a low partial pressure of oxygen at high altitudes. AMS typically can occur at 2400 meters (8000 feet) above sea level. AMS is a result of low blood oxygen levels, as the body has acute difficulty adjusting to the low partial pressure of oxygen. In serious cases, AMS can cause pulmonary or cerebral edema. Symptoms of AMS include nausea, vomiting, fatigue, lightheadedness, drowsiness, feeling disoriented, increased pulse, and nosebleeds. The only treatment for AMS is descending to a lower altitude; however, pharmacologic treatments and supplemental oxygen can improve symptoms. AMS can be prevented by slowly ascending to the desired altitude, allowing the body to acclimate, as well as maintaining proper hydration.

Acclimatization

Especially in situations where the ascent occurs too quickly, traveling to areas of high altitude can cause AMS. Acclimatization is the process of adjustment that the respiratory system makes due to chronic exposure to a high altitude. Over a period of time, the body adjusts to accommodate the lower partial pressure of oxygen. The low partial pressure of oxygen at high altitudes results in a lower oxygen saturation level of hemoglobin in the blood. In turn, the tissue levels of oxygen are also lower. As a result, the kidneys are stimulated to produce the hormone erythropoietin (EPO), which stimulates the production of erythrocytes, resulting in a greater number of circulating erythrocytes in an individual at a high altitude over a long period. With more red blood cells, there is more hemoglobin to help transport the available oxygen. Even though there is low saturation of each hemoglobin molecule, there will be more hemoglobin present, and therefore more oxygen in the blood. Over time, this allows the person to partake in physical exertion without developing AMS.

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8.7: Embryonic Development of the Respiratory System

Time Line

Development of the respiratory system begins early in the fetus. It is a complex process that includes many structures, most of which arise from the endoderm. Towards the end of development, the fetus can be observed making breathing movements. Until birth, however, the pregnant person provides all of the oxygen to the fetus as well as removes all of the fetal carbon dioxide via the placenta.

The development of the respiratory system begins at about week 4 of gestation. By week 28, enough alveoli have matured that a baby born prematurely at this time can usually breathe on its own. The respiratory system, however, is not fully developed until early childhood, when a full complement of mature alveoli is present.

Weeks 4–7

Respiratory development in the embryo begins around week 4. Ectodermal tissue from the anterior head region invaginates posteriorly to form olfactory pits, which fuse with endodermal tissue of the developing pharynx. An olfactory pit is one of a pair of structures that will enlarge to become the nasal cavity. At about this same time, the lung bud forms. The lung bud is a dome-shaped structure composed of tissue that bulges from the foregut. The foregut is endoderm just inferior to the pharyngeal pouches. The laryngotracheal bud is a structure that forms from the longitudinal extension of the lung bud as development progresses. The portion of this structure nearest the pharynx becomes the trachea, whereas the distal end becomes more bulbous, forming bronchial buds. A bronchial bud is one of a pair of structures that will eventually become the bronchi and all other lower respiratory structures (Figure 8.7.1).

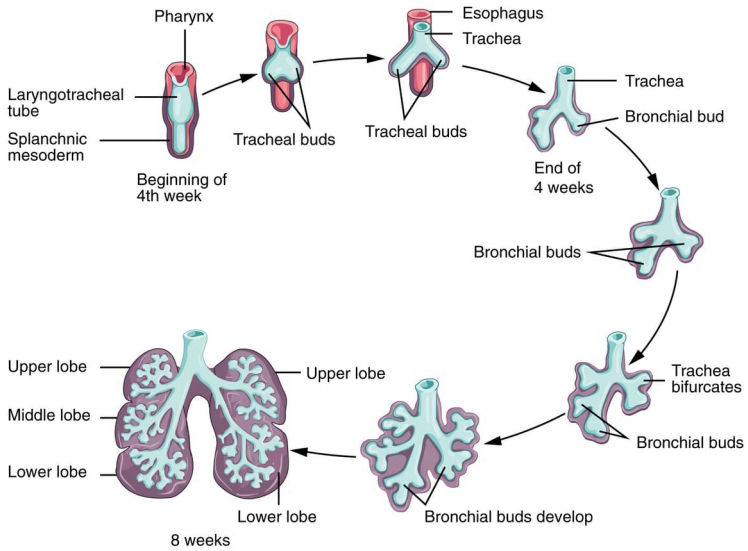


Figure 8.7.1: Embryonic development of the respiratory system from the laryngotracheal tube to the developing lungs. Sequential branching of the tracheal and bronchial buds forms the trachea, bronchi, and lung lobes during the early weeks of development. (CC BY 4.0; via LibreTexts).
Alternate text description of the Figure 8.7.1.

Weeks 7–16

Bronchial buds continue to branch as development progresses until all of the segmental bronchi have been formed. Beginning around week 13, the lumens of the bronchi begin to expand in diameter. By week 16, respiratory bronchioles form. The fetus now has all major lung structures involved in the airway.

Weeks 16–24

Once the respiratory bronchioles form, further development includes extensive vascularization, or the development of the blood vessels, as well as the formation of alveolar ducts and alveolar precursors. At about week 19, the respiratory bronchioles have formed. In addition, cells lining the respiratory structures begin to differentiate to form type I and type II pneumocytes. Once type II cells have differentiated, they begin to secrete small amounts of pulmonary surfactant. Around week 20, fetal breathing movements may begin.

Weeks 24–Term

Major growth and maturation of the respiratory system occurs from week 24 until term. More alveolar precursors develop, and larger amounts of pulmonary surfactant are produced. Surfactant levels are not generally adequate to create effective lung compliance until about the eighth month of pregnancy. The respiratory system continues to expand, and the surfaces that will form the respiratory membrane develop further. At this point, pulmonary capillaries have formed and continue to expand, creating a large surface area for gas exchange. The major milestone of respiratory development occurs at around week 28, when sufficient alveolar precursors have matured so that a baby born prematurely at this time can usually breathe on its own. However, alveoli continue to develop and mature into childhood. A full complement of functional alveoli does not appear until around 8 years of age.

Fetal “Breathing”

Although the function of fetal breathing movements is not entirely clear, they can be observed starting at 20–21 weeks of development. Fetal breathing movements involve muscle contractions that cause the inhalation of amniotic fluid and exhalation of the same fluid, with pulmonary surfactant and mucus. Fetal breathing movements are not continuous and may include periods of frequent movements and periods of no movements. Maternal factors can influence the frequency of breathing movements. For example, high blood glucose levels, called hyperglycemia, can boost the number of breathing movements. Conversely, low blood glucose levels, called hypoglycemia, can reduce the number of fetal breathing movements. Tobacco use is also known to lower fetal breathing rates. Fetal breathing may help tone the muscles in preparation for breathing movements once the fetus is born. It may also help the alveoli to form and mature. Fetal breathing movements are considered a sign of robust health.

Birth

Prior to birth, the lungs are filled with amniotic fluid, mucus, and surfactant. As the fetus is squeezed through the birth canal, the fetal thoracic cavity is compressed, expelling much of this fluid. Some fluid remains, however, but is rapidly absorbed by the body shortly after birth. The first inhalation occurs within 10 seconds after birth and not only serves as the first inspiration, but also acts to inflate the lungs. Pulmonary surfactant is critical for inflation to occur, as it reduces the surface tension of the alveoli. Preterm birth around 26 weeks frequently results in severe respiratory distress, although with current medical advancements, some babies may survive. Prior to 26 weeks, sufficient pulmonary surfactant is not produced, and the surfaces for gas exchange have not formed adequately; therefore, survival is low.

Disorders of the Respiratory System: Respiratory Distress Syndrome

Respiratory distress syndrome (RDS) primarily occurs in infants born prematurely. Up to 50 percent of infants born between 26 and 28 weeks and fewer than 30 percent of infants born between 30 and 31 weeks develop RDS. RDS results from insufficient production of pulmonary surfactant, thereby preventing the lungs from properly inflating at birth. A small amount of pulmonary surfactant is produced beginning at around 20 weeks; however, this is not sufficient for inflation of the lungs. As a result, dyspnea occurs and gas exchange cannot be performed properly. Blood oxygen levels are low, whereas blood carbon dioxide levels and pH are high.

The primary cause of RDS is premature birth, which may be due to a variety of known or unknown causes. Other risk factors include gestational diabetes, cesarean delivery, second-born twins, and family history of RDS. The presence of RDS can lead to other serious disorders, such as septicemia (infection of the blood) or pulmonary hemorrhage. Therefore, it is important that RDS is immediately recognized and treated to prevent death and reduce the risk of developing other disorders.

Medical advances have resulted in an improved ability to treat RDS and support the infant until proper lung development can occur. At the time of delivery, treatment may include resuscitation and intubation if the infant does not breathe on their own. These infants would need to be placed on a ventilator to mechanically assist with the breathing process. If spontaneous breathing occurs, application of nasal continuous positive airway pressure (CPAP) may be required. In addition, pulmonary surfactant is typically administered. Death due to RDS has been reduced by 50 percent due to the introduction of pulmonary surfactant therapy. Other therapies may include corticosteroids, supplemental oxygen, and assisted ventilation. Supportive therapies, such as temperature regulation, nutritional support, and antibiotics, may be administered to the premature infant as well.

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8.8: Terms and Definitions

Key Terms

Select to View Definitions

Acclimatization
process of adjustment that the respiratory system makes due to chronic exposure to high altitudes

Acute Mountain Sickness (AMS)
condition that occurs a result of acute exposure to high altitude due to a low partial pressure of oxygen

Ala
(plural = alae) small, flaring structure of a nostril that forms the lateral side of the nares

Alar Cartilage
cartilage that supports the apex of the nose and helps shape the nares; it is connected to the septal cartilage and connective tissue of the alae

Alveolar Dead Space
air space within alveoli that are unable to participate in gas exchange

Alveolar Duct
small tube that leads from the terminal bronchiole to the respiratory bronchiole and is the point of attachment for alveoli

Alveolar Macrophage
immune system cell of the alveolus that removes debris and pathogens

Alveolar Pore
opening that allows airflow between neighboring alveoli

Alveolar Sac
cluster of alveoli

Alveolus
small, grape-like sac that performs gas exchange in the lungs

Anatomical Dead Space
air space present in the airway that never reaches the alveoli and therefore never participates in gas exchange

Apex
tip of the external nose

Apneustic Center
network of neurons within the pons that stimulate the neurons in the dorsal respiratory group; controls the depth of inspiration

Atmospheric Pressure
amount of force that is exerted by gases in the air surrounding any given surface

Bohr Effect
relationship between blood pH and oxygen dissociation from hemoglobin

Boyle's Law
relationship between volume and pressure as described by the formula: $P_1V_1 = P_2V_2$

Bridge
portion of the external nose that lies in the area of the nasal bones

Bronchial Bud
structure in the developing embryo that forms when the laryngotracheal bud extends and branches to form two bulbous structures

Bronchial Tree
collective name for the multiple branches of the bronchi and bronchioles of the respiratory system

Bronchiole
branch of bronchi that are 1 mm or less in diameter and terminate at alveolar sacs

Bronchoconstriction
decrease in the size of the bronchiole due to relaxation of the muscular wall

Bronchodilation
increase in the size of the bronchiole due to contraction of the muscular wall

Bronchus
tube connected to the trachea that branches into many subsidiaries and provides a passageway for air to enter and leave the lungs

Carbaminohemoglobin
bound form of hemoglobin and carbon dioxide

Carbonic Anhydrase (CA)
enzyme that catalyzes the reaction that causes carbon dioxide and water to form carbonic acid

Cardiac Notch
indentation on the surface of the left lung that allows space for the heart

Central Chemoreceptor
one of the specialized receptors that are located in the brain that sense changes in hydrogen ion, oxygen, or carbon dioxide concentrations in the brain

Chloride Shift
facilitated diffusion that exchanges bicarbonate (HCO_3^-) with chloride (Cl^-) ions

Conducting Zone
region of the respiratory system that includes the organs and structures that provide passageways for air and are not directly involved in gas exchange

Cricoid Cartilage
portion of the larynx composed of a ring of cartilage with a wide posterior region and a thinner anterior region; attached to the esophagus

Dalton's Law
statement of the principle that a specific gas type in a mixture exerts its own pressure, as if that specific gas type was not part of a mixture of gases

Dorsal Respiratory Group (DRG)
region of the medulla oblongata that stimulates the contraction of the diaphragm and intercostal muscles to induce inspiration

Dorsum Nasi
intermediate portion of the external nose that connects the bridge to the apex and is supported by the nasal bone

Epiglottis
leaf-shaped piece of elastic cartilage that is a portion of the larynx that swings to close the trachea during swallowing

Expiration
(also, exhalation) process that causes the air to leave the lungs

Expiratory Reserve Volume (ERV)
amount of air that can be forcefully exhaled after a normal tidal exhalation

External Nose
region of the nose that is easily visible to others

External Respiration
gas exchange that occurs in the alveoli

Fauces
portion of the posterior oral cavity that connects the oral cavity to the oropharynx

Fibroelastic Membrane
specialized membrane that connects the ends of the C-shape cartilage in the trachea; contains smooth muscle fibers

Forced Breathing
(also, hyperpnea) mode of breathing that occurs during exercise or by active thought that requires muscle contraction for both inspiration and expiration

Foregut
endoderm of the embryo towards the head region

Functional Residual Capacity (FRC)
sum of ERV and RV, which is the amount of air that remains in the lungs after a tidal expiration

Glottis
opening between the vocal folds through which air passes when producing speech

Haldane Effect
relationship between the partial pressure of oxygen and the affinity of hemoglobin for carbon dioxide

Henry's Law
statement of the principle that the concentration of gas in a liquid is directly proportional to the solubility and partial pressure of that gas

Hilum
concave structure on the mediastinal surface of the lungs where blood vessels, lymphatic vessels, nerves, and a bronchus enter the lung

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8.9: Chapter Review Questions

Multiple-Choice Section

- Use the end-of-chapter multiple-choice section as a regular self-check tool, not just before exams.
- Read each item carefully, think about what is really being tested (concept, definition, application, or calculation), and choose the option (A, B, C, or D) that best reflects what you have learned in this chapter.
  - After you read through the chapter, attempt all items without looking at your notes; then return to the chapter to review any you missed or felt unsure about.
  - Explain why the correct option is right and why the others are not. Repeat the multiple-choice section a few days later to reinforce learning.
- Students who consistently use these prompts typically perform better because they turn reading into active practice.

1. Which of the following anatomical structures is not part of the conducting zone?
   A. Pharynx
   B. Nasal cavity
   C. Alveoli
   D. Bronchi

2. What is the function of the conchae in the nasal cavity?
   A. Increase surface area
   B. Exchange gases
   C. Maintain surface tension
   D. Maintain air pressure

3. The fauces connects which of the following structures to the oropharynx?
   A. Nasopharynx
   B. Laryngopharynx
   C. Nasal cavity
   D. Oral cavity

4. Which of the following are structural features of the trachea?
   A. C-shaped cartilage
   B. Smooth muscle fibers
   C. Cilia
   D. All of the above

5. Which of the following structures is not part of the bronchial tree?
   A. Alveoli
   B. Bronchi
   C. Terminal bronchioles
   D. Respiratory bronchioles

6. What is the main role of alveolar macrophages?
   A. To secrete pulmonary surfactant
   B. To secrete antimicrobial proteins into the mucus
   C. To remove pathogens and debris by phagocytosis
   D. To directly facilitate gas exchange

7. Which of the following structures separates the lung into lobes?
   A. Mediastinum
   B. Fissure
   C. Root
   D. Pleura

8. A section of the lung that receives its own tertiary (segmental) bronchus is called the:
   A. Bronchopulmonary segment
   B. Pulmonary lobule
   C. Interpulmonary segment
   D. Respiratory segment

9. The _____ circulation picks up oxygen for the body and releases carbon dioxide for removal.
   A. Pulmonary
   B. Interlobular
   C. Respiratory
   D. Bronchial

10. The pleura that surrounds the lungs consists of two layers, the:
    A. Visceral and parietal pleurae
    B. Mediastinum and parietal pleurae
    C. Visceral and mediastinum pleurae
    D. None of the above

11. Which of the following processes does atmospheric pressure directly help drive?
    A. Pulmonary ventilation (breathing)
    B. Production of pulmonary surfactant
    C. Airway resistance
    D. Surface tension in the alveoli

12. According to Boyle's law, a decrease in thoracic volume leads to a(n) _____ in pressure in the lungs.
    A. Decrease
    B. Equalization
    C. Increase
    D. Zero

13. The pressure difference between the intra-alveolar pressure and the intrapleural pressure is called:
    A. Atmospheric pressure
    B. Pulmonary pressure
    C. Negative pressure
    D. Transpulmonary pressure

14. Gas flow in the airways decreases as which factor increases?
    A. Resistance
    B. Pressure gradient
    C. Airway diameter
    D. Friction with the chest wall only

15. Contraction of the external intercostal muscles causes which of the following to occur?
    A. The diaphragm moves downward
    B. The rib cage is compressed
    C. The thoracic cavity volume decreases
    D. The ribs and sternum move upward

16. Which of the following helps prevent the alveoli from collapsing between breaths?
    A. Residual volume
    B. Tidal volume
    C. Expiratory reserve volume
    D. Inspiratory reserve volume

17. Gas moves from an area of _____ partial pressure to an area of _____ partial pressure.
    A. Low; high
    B. Low; low
    C. High; high

D. High; low

18. When ventilation to an alveolus is not sufficient (poor airflow), which of the following local responses occurs in its nearby pulmonary arterioles?
A. The capillary (arteriole) constricts
B. The capillary dilates
C. The partial pressure of oxygen in that alveolus increases
D. The bronchioles dilate

19. Gas exchange that occurs between systemic capillaries and body tissues is called:
A. External respiration
B. Interpulmonary respiration
C. Internal respiration
D. Pulmonary ventilation

20. The partial pressure of carbon dioxide is 45 mm Hg in the blood and 40 mm Hg in the alveoli. What happens to the carbon dioxide?
A. It diffuses into the blood
B. It diffuses into the alveoli
C. The gradient is too small for carbon dioxide to diffuse
D. It decomposes into carbon and oxygen

21. Oxyhemoglobin forms by a chemical reaction between:
A. Hemoglobin and carbon dioxide
B. Carbonic anhydrase and carbon dioxide
C. Hemoglobin and oxygen
D. Carbonic anhydrase and oxygen

22. Which of the following factors all play a role in shifting the oxygen–hemoglobin saturation (dissociation) curve?
A. Temperature
B. pH
C. BPG (2,3-bisphosphoglycerate)
D. All of the above

23. Which of the following occurs during the chloride shift in systemic capillaries?
A. Chloride is removed from the erythrocyte
B. Chloride is exchanged for bicarbonate entering or leaving the erythrocyte
C. Bicarbonate is removed only from the erythrocyte into the tissues
D. Bicarbonate is removed from the blood completely

24. A low partial pressure of oxygen in the blood promotes hemoglobin binding to carbon dioxide. This is an example of the:
A. Haldane effect
B. Bohr effect
C. Dalton's law
D. Henry's law

25. Increased ventilation that results in a rise in blood pH (more basic) is called:
A. Hyperventilation
B. Hyperpnea
C. Acclimatization
D. Apnea

26. Exercise at high altitude can trigger symptoms of acute mountain sickness (AMS) mainly because of:
A. Low partial pressure of oxygen in the air
B. Low atmospheric pressure with unchanged oxygen fraction
C. Abnormal neural signals from the motor cortex
D. Small venous reserve of oxygen at sea level

27. Which of the following most directly stimulates the production of new erythrocytes (RBCs)?
    A. AMS
    B. High blood levels of carbon dioxide alone
    C. Low atmospheric pressure alone
    D. Erythropoietin (EPO)

28. The olfactory pits (primitive nasal cavities) form from which embryonic germ layer?
    A. Mesoderm
    B. Cartilage
    C. Ectoderm
    D. Endoderm

29. A full complement of mature alveoli is usually present by:
    A. Early childhood, around 8 years of age
    B. Birth
    C. 37 weeks of gestation
    D. 16 weeks of gestation

30. If a baby is born prematurely before type II cells produce sufficient pulmonary surfactant, which of the following is most likely?
    A. Difficulty expressing (expelling) fetal lung fluid
    B. Difficulty inflating the lungs due to high surface tension
    C. Difficulty with pulmonary capillary blood flow
    D. No difficulty, because type I cells can produce enough surfactant

31. When do fetal breathing movements (practice breaths) usually begin?
    A. Around week 20 of gestation
    B. Around week 37 of gestation
    C. Around week 16 of gestation
    D. Only after birth

32. What happens to the fluid that remains in the lungs after birth?
    A. It reduces the surface tension of the alveoli
    B. It is expelled from the lungs shortly after birth
    C. It is absorbed into the tissue and blood shortly after birth
    D. It lubricates the pleurae long-term

Case-based questions

33. A 65-year-old man with long-term smoking history has a chronic cough and difficulty exhaling air. His chest X-ray shows hyperinflated lungs with flattened diaphragms. Which of the following changes best explains his problem with exhalation?
    A. Increased elastic recoil of the lung tissue
    B. Decreased elastic recoil and destruction of alveolar walls
    C. Increased thickness of the respiratory membrane for diffusion
    D. Decreased airway resistance in the bronchioles

34. A 25-year-old woman has an asthma attack after exposure to cat dander. She is wheezing and has difficulty moving air, especially when exhaling. Which of the following is the main immediate cause of her increased airway resistance?
    A. Loss of surfactant in the alveoli
    B. Constriction of the smooth muscle in the bronchioles
    C. Collapse of the tracheal cartilage rings
    D. Thickening of the pleural membranes

35. A healthy 20-year-old at rest has a tidal volume of 500 mL, a respiratory rate of 12 breaths per minute, and an anatomic dead space of 150 mL. Which value is closest to this person's alveolar ventilation per minute (ml/min)?
    A. 6,000 mL/min

B. 4,200 mL/min
C. 3,000 mL/min
D. 1,800 mL/min

36. A 50-year-old man with severe pneumonia has thick fluid filling parts of his alveoli. His arterial PO_2 is low even though he is breathing room air. Which change in his lungs most directly explains the reduced oxygen diffusion into the blood?
A. Decreased total surface area for gas exchange
B. Decreased thickness of the respiratory membrane
C. Increased ventilation–perfusion matching
D. Increased alveolar PO_2

37. A mountain climber quickly travels from sea level to 3,500 m altitude and develops headache, dizziness, and rapid breathing. What is the main immediate reason her arterial oxygen saturation falls at high altitude?
A. Higher body temperature at altitude
B. Lower fraction of oxygen in the air
C. Lower atmospheric pressure, so lower PO_2 despite same oxygen fraction
D. Increased thickness of her alveolar walls

38. A patient has chronic kidney disease and cannot excrete acid effectively. His blood pH is low (acidosis), and his arterial PCO_2 is also low. Which respiratory response is the body using to help compensate for the metabolic acidosis?
A. Hypoventilation to retain CO_2
B. Hyperventilation to blow off CO_2
C. Apnea to prevent oxygen loss
D. Decreased tidal volume with unchanged rate

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8.10: Critical Thinking Self-Assessment

Challenge Yourself

- Extended response questions are assessment items that require students to construct written answers of substantial length - typically several sentences to multiple paragraphs - rather than selecting from predefined options. They represent a form of constructed response assessment designed to measure complex, higher-order thinking skills.
- First, try answering without using your notes. Then review reliable resources and revise your response if needed. Make a note of any areas where you felt unsure and revisit those topics.

Writing Guidelines

- Answer each question in your own words.
- Write 4–8 clear, complete sentences per question, organized into short paragraphs (not bullet points).
- Focus on explaining your reasoning, not just stating the answer.
- Use correct scientific vocabulary, and briefly define terms when needed.
- For questions that ask you to compare, contrast, or explain why/how:
  - Clearly state similarities or differences
  - Support your ideas with a brief explanation or example

1. The wall of an alveolus is extremely thin and is surrounded by many capillaries. Explain how this structure helps with gas exchange between air and blood.
   Hint: Think about the distance oxygen and carbon dioxide must travel, and why a thin, moist surface with a large area is important.

2. Most of the airway (trachea and bronchi) has ciliated epithelium and mucus. Explain how these structures help protect the lungs from dust and microorganisms.
   Hint: Think about what mucus does to particles you breathe in, and how cilia move that mucus.

3. The left lung has two lobes, and the right lung has three lobes. Suggest one possible advantage of having the lungs divided into separate lobes and segments.
   Hint: Think about what happens if one area is infected or collapsed. Can other parts still work?

4. During quiet breathing, inspiration (breathing in) is usually active, and expiration (breathing out) is usually passive. Explain what this means in terms of muscles and energy use.
   Hint: Think about the diaphragm and intercostal muscles, and what happens to the lungs and chest when these muscles relax.

5. A person has a blockage in a main bronchus leading to one lung. Explain how this would affect:
   a) ventilation (air movement) in that lung, and
   b) gas exchange in the body as a whole.
   Hint: Consider what happens to oxygen levels in blood if one lung cannot receive fresh air.

6. At high altitude, the air has lower oxygen pressure, even though the percentage of oxygen is the same. Explain why a person may feel short of breath and tired more easily at high altitude.
   Hint: Think about diffusion of oxygen from alveoli to blood and how lower pressure affects this movement.

7. Carbon dioxide is transported in blood in three main forms: dissolved in plasma, bound to hemoglobin, and as bicarbonate ions. Explain why changing CO_2 levels in the blood can also change blood pH.
   Hint: Remember that CO_2 + water can form an acid; think about what happens if CO_2 is too high or too low.

8. During exercise, both breathing rate and depth increase. Explain how these changes in ventilation help match the needs of working muscles.
   Hint: Use the ideas of oxygen delivery and carbon dioxide removal; think about why just faster breaths or just deeper breaths might not be enough.

9. In asthma, the smooth muscles in the bronchioles constrict and the airways become narrowed. Explain how this affects airflow and the effort needed to breathe.
   Hint: Think about resistance to airflow in small tubes and how hard the respiratory muscles must work to move air.

10. The respiratory system and the cardiovascular system must work closely together to maintain homeostasis. Describe one situation (for example, heavy exercise, blood loss, or lung disease) and explain how a problem in one of these systems affects the other.
    Hint: Think about what happens to heart rate, breathing rate, and blood oxygen levels when one system is not working well.

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8.11: Alternate Text Descriptions

Figure 8.1.1: Major Structures of the Respiratory System

This figure illustrates the major structures of the **human respiratory system** and the pathway air follows from the external environment to the **lungs**. The diagram presents a frontal anatomical view of the **head**, **neck**, and **thorax** with labels identifying key respiratory structures.

Pathway of Air Through the Respiratory System

1. Air enters the respiratory system through the **nostrils** and passes into the **nasal cavity**.
2. Air may also enter through the **oral cavity**.
3. Both passages connect to the **pharynx**, located behind the nasal and oral cavities.
4. The pharynx serves as a passageway that directs air toward the lower respiratory tract.
5. Inferior to the pharynx is the **larynx**, which connects the upper respiratory tract to the **trachea**.
6. The **trachea** extends downward through the **neck** into the **thoracic cavity**.
7. At the level of the lungs, the trachea divides into the **right main bronchus** and **left main bronchus**.
8. These bronchi conduct air into the **right lung** and **left lung**, respectively.

Lungs and Airways

1. The **right lung** and **left lung** occupy most of the **thoracic cavity**.
2. They are the primary organs of **gas exchange**.
3. The main bronchi branch within the lungs into progressively smaller airways, although these smaller branches are not shown in the figure.

Diaphragm

1. Beneath both lungs is the **diaphragm**, a dome-shaped muscle that separates the **thoracic cavity** from the **abdominal cavity**.
2. Contraction of the diaphragm increases thoracic cavity volume and helps draw air into the lungs during **inhalation**.
3. Relaxation of the diaphragm decreases thoracic cavity volume and assists **exhalation**.

Labeled Structures

1. Nasal cavity
2. Nostril
3. Oral cavity
4. Pharynx
5. Larynx
6. Trachea
7. Right main bronchus
8. Left main bronchus
9. Right lung
10. Left lung
11. Diaphragm

The figure shows the major structures of the **respiratory system** and demonstrates the route air follows from the external environment into the **lungs**, where gas exchange will ultimately occur.

Figure 8.1.2: External and Structural Anatomy of the Nose

This figure presents the external anatomy and underlying structural framework of the human nose using three labeled views. The upper portion includes a frontal view (left) and a lateral view (right) showing surface landmarks of the nose. The lower portion shows a lateral anatomical view illustrating the bones and cartilages that support the nasal structure.

In the frontal and lateral views, labels identify key external anatomical landmarks. The **root** is the superior portion of the nose located between the eyes where the nose begins. Inferior to the root is the **bridge**, the upper narrow region that extends downward along the midline. The **dorsum nasi** refers to the length of the external nose extending from the bridge toward the tip. The **ala** forms the rounded, lateral wings of the nose surrounding the nostrils. The **apex** is the tip of the nose. The **philtrum** is the vertical groove located between the base of the nose and the upper lip.

The lower anatomical view illustrates the skeletal and cartilaginous structures that support the external nose. The superior portion of the nose is supported by the **frontal bone**, which forms part of the forehead. Inferior to the frontal bone is the **nasal bone**, which contributes to the upper bony framework of the nose. The **maxillary bone** forms part of the facial skeleton adjacent to the nasal region and provides structural support.

The lower and anterior portions of the nose are supported by cartilage rather than bone. The **septal cartilage** forms the anterior portion of the nasal septum and helps maintain the shape of the nasal midline. The **major alar cartilage** supports the lateral walls and tip of the nose, contributing to the shape of the nostrils and the apex. A second label identifies the inferior portion of the **septal cartilage** near the base of the nose.

Together, these structures create the shape of the external nose and provide support for the nasal passages, which conduct air from the external environment into the nasal cavity for filtration, warming, and humidification before air enters the lower respiratory tract.

Labels shown in the figure: Root, Bridge, Dorsum nasi, Ala, Apex, Philtrum, Frontal bone, Nasal bone, Maxillary bone, Septal cartilage (two locations), and Major alar cartilage.

Figure 8.1.3: Midsagittal View of the Nasal Cavity, Pharynx, Oral Cavity, and Larynx

This figure shows a midsagittal (side) section of the head and neck, illustrating the upper respiratory tract, portions of the digestive tract, and associated structures. Labels identify anatomical features of the nasal cavity, pharynx, oral cavity, and larynx, demonstrating the pathways for airflow, smell, speech production, and swallowing.

Air enters the respiratory system through the **nostril** and passes through the **nasal vestibule** into the nasal cavity. Within the nasal cavity are three curved bony shelves called the **nasal conchae**, which increase surface area and help warm, humidify, and filter inhaled air. The spaces beneath the conchae are the **nasal meatuses** (superior, middle, and inferior), which direct airflow through the cavity. The **olfactory epithelium**, located near the roof of the nasal cavity, contains sensory receptors responsible for the sense of smell.

Several surrounding structures are shown. The **frontal sinus** lies within the frontal bone superior to the nasal cavity, while the **sphenoidal sinus** is positioned posteriorly within the sphenoid bone. The **ethmoid bone** contributes to the roof and walls of the nasal cavity.

Posterior to the nasal cavity is the **nasopharynx**, the superior portion of the pharynx. The **pharyngeal tonsil** is located on the posterior wall of the nasopharynx. The **opening of the auditory tube** connects the nasopharynx to the middle ear and helps equalize air pressure.

Inferior to the nasal cavity is the oral cavity. The anterior roof of the mouth is formed by the **hard palate**, while the posterior muscular portion is the **soft palate**. Extending from the soft palate is the **uvula**. The **tongue** occupies much of the oral cavity and assists with speech, chewing, and swallowing. The **lingual tonsil** is located at the base of the tongue. The opening between the oral cavity and the pharynx is identified as the **fauces**. The **palatine tonsil** is located along the lateral wall of the oropharynx.

The pharynx is divided into three regions. The **nasopharynx** lies posterior to the nasal cavity, the **oropharynx** lies posterior to the oral cavity, and the **laryngopharynx** lies posterior to the larynx. These regions serve as passageways for air and, in the lower

portions, for food and liquids.

Inferior to the tongue is the **hyoid bone**, which supports the tongue and associated muscles. The **epiglottis** is a leaf-shaped structure that helps prevent food and liquid from entering the airway during swallowing by covering the laryngeal inlet.

The larynx contains the **vestibular fold** (false vocal fold) and the **vocal fold** (true vocal fold), which are involved in airway protection and sound production. The framework of the larynx includes the **thyroid cartilage** and the **cricoid cartilage**, which support and maintain the airway.

Overall, the figure demonstrates the anatomical relationships between the nasal passages, oral cavity, pharynx, and larynx, highlighting how inhaled air travels from the nostril through the nasal cavity and pharynx to the lower respiratory tract, while also showing structures involved in smell, speech, hearing pressure regulation, and swallowing.

Labels shown: Sphenoidal sinus, Nasal meatuses (superior, middle, and inferior), Pharyngeal tonsil, Opening of auditory tube, Nasopharynx, Uvula, Palatine tonsil, Fauces, Oropharynx, Laryngopharynx, Vestibular fold, Vocal fold, Frontal sinus, Ethmoid bone, Olfactory epithelium, Nasal conchae, Nasal vestibule, Nostril, Hard palate, Soft palate, Tongue, Lingual tonsil, Epiglottis, Hyoid bone, Thyroid cartilage, and Cricoid cartilage

Figure 8.1.4: Histology of the Tracheal Lining and Mucociliary Apparatus

This figure is a light micrograph of the tracheal lining and underlying tissues. It highlights the structure of the respiratory mucosa and demonstrates the components of the mucociliary apparatus that help protect the lower respiratory tract.

The upper white region is labeled as the **lumen of the trachea**, the open space through which air travels. Forming the boundary between the lumen and the underlying tissue is a layer of **pseudostratified columnar epithelium**, the characteristic epithelial lining of much of the conducting respiratory tract. Although the nuclei appear at different heights, all epithelial cells rest on the basement membrane, creating the appearance of multiple layers.

Projecting from the apical surface of the epithelial cells are numerous **cilia**, visible as fine hair-like extensions facing the lumen. The coordinated beating of these cilia moves mucus and trapped particles toward the pharynx, where the material can be swallowed or expelled.

Scattered among the epithelial cells are **goblet cells**, specialized mucus-secreting cells. Goblet cells produce mucus that coats the epithelial surface. This mucus traps inhaled dust, microorganisms, and other airborne particles, helping prevent them from reaching the lungs.

Beneath the epithelial layer is connective tissue containing a **seromucous gland in the submucosa**. These glands secrete both mucus and watery fluid, contributing to the moisture of the airway lining and supporting the formation of the protective mucus layer.

Together, the goblet cells, submucosal glands, and cilia form the **mucociliary escalator**, a defense mechanism that filters inhaled air and transports debris away from the lower respiratory tract.

Labels shown in the figure: Goblet cell, Lumen of trachea, Cilia, Pseudostratified columnar epithelia, and Seromucous gland in submucosa.

Figure 8.1.5: Anatomical Divisions of the Pharynx

This figure is a midsagittal (side) view of the head and neck illustrating the anatomical divisions of the pharynx. The pharynx is color-coded to distinguish its three regions: the **nasopharynx** (green), **oropharynx** (blue), and **laryngopharynx** (purple). Surrounding structures of the respiratory and digestive systems are labeled to show their relationship to each pharyngeal region.

The **nasal cavity** occupies the superior portion of the airway and opens posteriorly into the **nasopharynx**, the uppermost region of the pharynx. The nasopharynx is located posterior to the nasal cavity and superior to the soft palate. This region functions primarily as an airway, conducting air from the nasal cavity toward the lower respiratory tract.

Inferior to the nasal cavity is the **hard palate**, which forms the anterior bony roof of the mouth. Posterior to the hard palate is the **soft palate**, a muscular structure that separates the nasopharynx from the oral cavity during swallowing.

The **tongue** occupies much of the oral cavity. Posterior to the oral cavity is the **oropharynx**, shown in blue. The oropharynx extends from the soft palate to the upper border of the epiglottis and serves as a common passageway for both air and food.

Inferior to the oropharynx is the **laryngopharynx**, shown in purple. This region extends from the level of the epiglottis to the entrance of the esophagus and larynx. The laryngopharynx serves as a shared pathway where air continues toward the respiratory tract and food is directed toward the digestive tract.

The **epiglottis** is a flap-like structure located at the entrance to the larynx. During swallowing, it helps prevent food and liquids from entering the airway. Anterior to the laryngopharynx is the **larynx (voice box)**, which connects to the **trachea**, the airway leading to the lungs. Posterior to the larynx is the **esophagus**, the muscular tube that carries food and liquids to the stomach.

The figure emphasizes the anatomical organization of the pharynx and demonstrates how the upper airway connects the nasal cavity and oral cavity with the lower respiratory and digestive tracts. The nasopharynx functions primarily in respiration, whereas the oropharynx and laryngopharynx serve both respiratory and digestive functions.

Labels shown in the figure: Nasal cavity, Hard palate, Tongue, Soft palate, Epiglottis, Larynx (voice box), Esophagus, Trachea, Nasopharynx, Oropharynx, and Laryngopharynx.

Figure 8.1.6: Anatomy of the Larynx in Anterior and Right Lateral Views

This figure presents the anatomy of the **larynx (voice box)** in two views. The upper illustration shows an **anterior view** of the larynx and upper trachea, while the lower illustration shows a **right lateral view**. The labeled structures demonstrate how the larynx connects the pharynx to the trachea and functions in breathing, swallowing, and phonation.

Anterior View

At the superior end of the larynx is the **epiglottis**, a leaf-shaped elastic cartilage that helps prevent food and liquids from entering the airway during swallowing.

Immediately inferior to the epiglottis is the **body of the hyoid bone**, a U-shaped bone that supports the tongue and serves as an attachment site for muscles associated with swallowing and speech. The hyoid bone is connected to the larynx by the **thyrohyoid membrane**.

The largest cartilage of the larynx is the **thyroid cartilage**, which forms the anterior and lateral walls of the larynx. On its anterior surface is the **laryngeal prominence**, commonly known as the Adam's apple.

Inferior to the thyroid cartilage is the **cricothyroid ligament**, which connects the thyroid cartilage to the **cricoid cartilage**. The cricoid cartilage forms a complete ring around the airway and provides structural support to the inferior portion of the larynx.

Below the cricoid cartilage is the **cricotracheal ligament**, which connects the larynx to the trachea. The airway continues inferiorly through the **tracheal cartilages**, a series of cartilaginous rings that help maintain an open passage for airflow.

Right Lateral View

The lateral view provides additional detail of the internal and supporting structures of the larynx.

Superiorly, the **epiglottis** extends upward behind the hyoid bone and folds over the laryngeal inlet during swallowing to protect the lower respiratory tract. The **thyrohyoid membrane** spans the space between the hyoid bone and thyroid cartilage. A **fatty pad** is shown adjacent to the thyrohyoid membrane.

Several paired cartilages that contribute to laryngeal function are visible posteriorly. The **arytenoid cartilages** provide attachment points for the vocal folds and muscles that regulate the opening of the airway and tension of the vocal cords. Situated superior to the arytenoid cartilages are the **corniculate cartilages**, and superior to these are the **cuneiform cartilages**, both of which help support surrounding soft tissues.

Within the larynx are two folds extending into the airway. The superior fold is the **vestibular fold** (false vocal fold), which helps protect the airway. Inferior to it is the **vocal fold** (true vocal fold), which vibrates as air passes through the larynx to produce

sound.

The **thyroid cartilage**, **cricothyroid ligament**, **cricoid cartilage**, and **cricotracheal ligament** are also visible in the lateral view and maintain the structural integrity of the larynx while connecting it to the trachea below. Inferiorly, the **tracheal cartilages** continue into the conducting airways of the respiratory system.

Functional Significance

The figure illustrates how the laryngeal cartilages, ligaments, membranes, and folds work together to:

- Maintain an open airway for respiration.
- Protect the lower respiratory tract during swallowing.
- Regulate airflow through the larynx.
- Enable voice production through vibration of the vocal folds.

Labels shown in the figure: Epiglottis, Body of hyoid bone, Thyrohyoid membrane, Thyroid cartilage, Laryngeal prominence, Cricothyroid ligament, Cricoid cartilage, Cricotracheal ligament, Tracheal cartilages, Cuneiform cartilage, Corniculate cartilage, Arytenoid cartilage, Fatty pad, Vestibular fold, and Vocal fold.

Figure 8.1.7: Superior View of the Larynx and Vocal Folds

This figure shows a superior view looking downward into the larynx from above the throat. The image illustrates the relationship between the vocal folds and the airway, as well as adjacent structures involved in respiration, swallowing, and phonation.

At the center of the figure are the paired **true vocal cords** (true vocal folds), shown as two pale bands extending from the anterior portion of the larynx toward the posterior wall. The vocal cords form the boundaries of the **glottis**, the opening between the vocal folds through which air passes during breathing and speech. Changes in the position and tension of the vocal cords alter airflow and enable sound production.

Located superior and lateral to the true vocal cords are the **vestibular folds** (false vocal folds). These folds do not normally participate in sound production but help protect the airway and contribute to closure of the laryngeal inlet during swallowing.

Anterior to the glottis is the **epiglottis**, a leaf-shaped structure that helps prevent food and liquids from entering the airway. During swallowing, the epiglottis folds posteriorly over the laryngeal opening, directing material toward the digestive tract.

Posterior to the laryngeal opening is the **esophagus**, the muscular tube that carries food and liquids from the pharynx to the stomach. Anterior and inferior to the glottis is the **trachea**, the airway that conducts air between the larynx and the lungs.

Lateral to the laryngeal inlet on both sides are the **pyriform fossae** (pyriform recesses), shallow channels within the laryngopharynx that guide food and liquids around the laryngeal opening and toward the esophagus during swallowing.

The **tongue** occupies the inferior portion of the image and lies anterior to the laryngeal structures. The tongue helps manipulate food in the oral cavity and initiates swallowing.

The figure demonstrates the anatomical relationship between the respiratory and digestive pathways. Air moves through the glottis into the trachea, while food and liquids are directed around the laryngeal opening through the pyriform fossae and into the esophagus. The epiglottis and vestibular folds help protect the lower respiratory tract during swallowing, while the true vocal cords produce sound.

Labels shown in the figure: Esophagus, Trachea, Vestibular fold, Glottis, Pyriform fossa, True vocal cord, Epiglottis, and Tongue.

Figure 8.1.8: Gross and Microscopic Anatomy of the Tracheobronchial Tree

This figure consists of two panels illustrating both the gross anatomy and microscopic structure of the conducting airways of the respiratory system.

(a) Gross Anatomy of the Tracheobronchial Tree

The left panel shows the **larynx**, **trachea**, and bronchial tree extending into the lungs. The larynx is located superiorly and continues inferiorly as the **trachea**, a tube supported by a series of **tracheal cartilages** that help keep the airway open during breathing.

At the inferior end of the trachea, the airway divides into the **right and left primary bronchi**, each entering its respective lung. Within the lungs, the primary bronchi branch into **secondary bronchi**, which continue to divide into progressively smaller airways (not shown).

The **right lung** and **left lung** are shown surrounding the branching bronchial tree. The figure demonstrates the pathway through which air travels from the larynx, through the trachea, into the bronchi, and ultimately toward the gas-exchange regions of the lungs.

(b) Microscopic Structure of a Bronchus

The right panel is a histological section of a bronchus showing the organization of tissues that line and support the airway.

The central open space is the **lumen**, through which air passes. The lumen is lined by **pseudostratified columnar epithelium**, a characteristic respiratory epithelium composed of cells that all contact the basement membrane but appear to form multiple layers because their nuclei are located at different heights.

Extending from the apical surface of the epithelial cells are **cilia**, fine hair-like projections that beat in coordinated waves to move mucus and trapped particles toward the pharynx for removal.

Beneath the epithelial lining are **submucosal seromucous glands**. These glands produce mucus and watery secretions that moisten the airway and help trap inhaled dust, microorganisms, and other foreign particles.

Deeper in the bronchial wall is **hyaline cartilage**, which provides structural support and helps maintain airway patency by preventing collapse during respiration.

The figure illustrates how the conducting airways transport and condition inhaled air. The trachea and bronchi provide a passageway for airflow, while cartilage maintains airway openness. The respiratory epithelium, cilia, and seromucous glands work together as part of the **mucociliary escalator**, a protective mechanism that traps and removes debris before it can reach the delicate respiratory surfaces of the lungs.

Labels shown in the figure: Larynx, Trachea, Tracheal cartilages, Primary bronchi, Secondary bronchi, Right lung, Left lung, Submucosal seromucous glands, Pseudostratified columnar epithelia, Cilia, Lumen, and Hyaline cartilage.

Figure 8.1.9: Respiratory Zone of the Lungs and Alveolar Gas Exchange

This figure illustrates the distal portion of the respiratory tract where gas exchange occurs. The diagram shows the branching pathway from a **terminal bronchiole** into the respiratory zone of the lungs and highlights the close relationship between the alveoli and the pulmonary capillary network.

At the top of the figure is a **terminal bronchiole**, the smallest conducting airway. The wall of the terminal bronchiole contains **smooth muscle**, which can adjust airway diameter and regulate airflow. The terminal bronchiole branches into smaller **respiratory bronchioles**, which represent the beginning of the respiratory zone and participate in both air conduction and gas exchange.

The respiratory bronchioles lead into **alveolar ducts**, elongated passageways whose walls are lined with openings into numerous alveoli. The alveolar ducts terminate in clusters of alveoli called **alveolar sacs**.

Individual **alveoli** are shown as small, thin-walled, balloon-like air chambers. Their walls provide a large surface area for gas exchange. Small openings between adjacent alveoli, labeled **alveolar pores**, allow air movement between neighboring alveoli and help equalize air pressure throughout the alveolar network.

Surrounding the alveoli is an extensive network of **capillaries**. Blood arriving from the pulmonary circulation through branches of

the **pulmonary artery** is shown as **deoxygenated blood**. These vessels form a dense capillary bed closely associated with the alveolar walls.

Across the thin respiratory membrane, oxygen diffuses from air within the alveoli into the blood, while carbon dioxide diffuses from the blood into the alveoli to be exhaled. After gas exchange occurs, the blood becomes oxygen-rich and leaves the capillary network as **oxygenated blood** that drains into branches of the **pulmonary vein**.

The figure emphasizes the structural organization of the respiratory zone and demonstrates how the branching airways, alveoli, and surrounding capillary network work together to maximize the efficiency of gas exchange between the lungs and the bloodstream.

Labels shown in the figure: Terminal bronchiole, Smooth muscle, Respiratory bronchiole, Alveolar duct, Alveolar sac, Alveolus, Alveolar pores, Capillaries, Deoxygenated blood from pulmonary artery, and Oxygenated blood to pulmonary vein.

Figure 8.1.10: Microscopic Anatomy of the Alveoli

This figure consists of two panels that illustrate the microscopic anatomy of the alveoli, the primary sites of gas exchange in the lungs.

(a) Alveolar Structure

The left panel is a detailed schematic of a single **alveolus** and its surrounding wall. An inset indicates that the enlarged view is taken from an alveolar sac within the respiratory zone of the lung.

The central cavity of the alveolus is labeled **alveolus (gas-filled space)**. This air-filled chamber receives inhaled air and serves as the location where oxygen and carbon dioxide are exchanged between the air and blood.

The alveolar wall is composed primarily of **Type I alveolar cells (type I pneumocytes)**, which form a thin epithelial lining specialized for rapid diffusion of gases. Embedded within the wall are **Type II alveolar cells (type II pneumocytes)**, which produce pulmonary surfactant that reduces surface tension and helps prevent alveolar collapse during exhalation.

Surrounding the alveolar wall are **capillaries** containing red blood cells. The thin barrier separating air in the alveolus from blood in the capillaries is labeled the **respiratory membrane**. This membrane consists of the alveolar epithelium, fused basement membranes, and capillary endothelium and serves as the site of gas exchange.

Small openings called **alveolar pores** connect adjacent alveoli, allowing airflow between neighboring air spaces and helping equalize pressure throughout the alveolar network.

Also present within the alveolar lumen is a **macrophage**, an immune cell that engulfs inhaled particles, microorganisms, and cellular debris to help maintain alveolar health.

(b) Histological View of Lung Tissue

The right panel is a light micrograph of lung tissue. Numerous thin-walled **alveoli** appear as small interconnected air spaces separated by delicate alveolar septa. The large number of alveoli creates extensive surface area for gas exchange.

Several alveoli open into a common chamber labeled **alveolar sac**. Multiple alveolar sacs connect to an **alveolar duct**, a passageway that conducts air through the respiratory zone.

Within the alveolar walls are **blood vessels** that supply pulmonary capillaries for gas exchange. The micrograph also shows the **lumen of a bronchiole**, representing an airway that conducts air into the respiratory portion of the lung.

Together, the two panels demonstrate how alveolar anatomy supports efficient gas exchange. Thin Type I alveolar cells and the respiratory membrane minimize diffusion distance, Type II alveolar cells help maintain alveolar inflation through surfactant production, capillaries provide close contact between blood and air, macrophages provide immune defense, and the extensive network of alveoli, alveolar ducts, and alveolar sacs maximizes surface area for the exchange of oxygen and carbon dioxide.

Labels shown in the figure: Alveolar pores, Capillary, Respiratory membrane, Type I alveolar cell, Type II alveolar cell, Macrophage, Alveolus, Alveolus (gas-filled space), Alveoli, Alveolar duct, Blood vessels, Lumen of bronchiole, and Alveolar sac.

Figure 8.1.11: Comparison of Normal and Inflamed Airway Mucosa

This figure compares the microscopic structure of a normal airway wall (a) with an inflamed airway wall (b). Both panels show the layers of the airway mucosa and underlying tissues, including the epithelium, basement membrane, lamina propria, smooth muscle, glands, and cartilage. The comparison highlights changes that occur during inflammation.

(a) Normal Airway Mucosa

The upper panel illustrates a healthy airway lining. A thin layer of mucus covers the luminal surface of the airway. Beneath the mucus is the epithelium, which contains scattered goblet cells that produce mucus. The epithelium rests on a basement membrane, a thin supporting layer that separates the epithelial cells from the underlying tissue.

Below the basement membrane is the lamina propria, a connective tissue layer containing relatively few cells. Beneath the lamina propria is a layer of smooth muscle, which helps regulate airway diameter through contraction and relaxation.

Deeper in the airway wall are glands that contribute secretions to the airway surface. A region of cartilage provides structural support and helps maintain airway patency.

Overall, the normal airway exhibits a relatively thin mucus layer and limited numbers of inflammatory cells within the connective tissue.

(b) Inflamed Airway Mucosa

The lower panel illustrates an inflamed airway. A thicker layer of mucus covers the epithelial surface, indicating increased mucus production. The epithelium contains goblet cells, which contribute to mucus secretion.

As in the normal airway, the epithelium rests on a basement membrane, with the lamina propria located beneath it. However, the lamina propria contains a markedly increased number of immune and inflammatory cells. Labeled cell types include mast cells, macrophages, eosinophils, and neutrophils. These cells accumulate within the tissue as part of the inflammatory response.

The smooth muscle layer remains present beneath the lamina propria. Deeper structures include the glands, which may contribute to the increased mucus production, and cartilage, which supports the airway wall.

The figure demonstrates how airway inflammation alters the structure of the respiratory mucosa. Compared with the normal airway, the inflamed airway contains a thicker mucus layer and a greater concentration of inflammatory cells within the lamina propria. These changes can contribute to airway narrowing, increased mucus accumulation, and impaired airflow.

Labels shown in the figure: Mucus, Goblet cell, Epithelium, Basement membrane, Lamina propria, Smooth muscle, Gland, Cartilage, Mast cell, Macrophage, Eosinophil, and Neutrophil.

Figure 8.2.1: Gross Anatomy of the Lungs and Bronchial Tree

This figure illustrates the gross anatomy of the lungs and the branching bronchial tree in an anterior view. The trachea extends downward from the neck and divides into the right and left main bronchi, which enter the corresponding lungs. Within each lung, the bronchi branch into progressively smaller airways.

At the superior center of the figure is the **trachea**, a cartilaginous airway that conducts air between the larynx and the lungs. Near its inferior end, the trachea divides into the **main (primary) bronchi**, one entering each lung.

Within the lungs, the primary bronchi branch into **lobar (secondary) bronchi**, which supply individual lobes of the lungs. The secondary bronchi further divide into **segmental (tertiary) bronchi**, which distribute air to smaller bronchopulmonary segments.

The **right lung**, shown on the left side of the figure, is divided into three lobes:

- **Superior lobe**
- **Middle lobe**
- **Inferior lobe**

The right main bronchus branches into lobar bronchi that supply each of these three lobes.

The **left lung**, shown on the right side of the figure, is divided into two lobes:

- **Superior lobe**
- **Inferior lobe**

The left lung contains a prominent indentation along its medial border called the **cardiac notch**, which accommodates the position of the heart. The left main bronchus branches into lobar bronchi supplying the superior and inferior lobes, and these continue to divide into segmental bronchi.

The figure demonstrates the hierarchical organization of the conducting airways, beginning with the trachea and progressing through the primary, secondary, and tertiary bronchi as air is distributed throughout the lungs. It also highlights the anatomical differences between the lungs, including the presence of three lobes in the right lung, two lobes in the left lung, and the cardiac notch in the left lung.

Labels shown in the figure: Trachea, Main (primary) bronchus, Lobar (secondary) bronchus, Segmental (tertiary) bronchus, Superior lobe, Middle lobe, Inferior lobe, Cardiac notch, Right lung, and Left lung.

Figure 8.2.2: Pleural Membranes, Pleural Cavity, and Lung within the Thoracic Wall

This figure illustrates the relationship between the lungs, thoracic wall, diaphragm, and pleural membranes. A portion of the chest wall is removed to reveal the lung enclosed within the pleural sac. An enlarged inset provides a detailed view of the pleural layers and pleural cavity.

The **chest wall**, consisting of the rib cage, sternum, thoracic vertebrae, connective tissue, and **intercostal muscles**, surrounds and protects the thoracic cavity. The intercostal muscles occupy the spaces between adjacent ribs and assist with breathing by helping expand and contract the thoracic cavity.

Within the thoracic cavity lies a **lung**, shown enclosed by a **pleural sac**. The pleural sac is composed of two continuous serous membranes:

- The **parietal pleura** lines the inner surface of the chest wall, diaphragm, and mediastinum.
- The **visceral pleura** adheres directly to the external surface of the lung and follows its contours.

Between the parietal and visceral pleura is a thin potential space called the **pleural cavity**. Although depicted as a visible space for illustration purposes, the pleural cavity normally contains only a small amount of lubricating pleural fluid. This fluid allows the pleural membranes to slide against one another during breathing while maintaining close contact between the lungs and thoracic wall.

Inferior to the lungs is the **diaphragm**, a dome-shaped skeletal muscle that forms the floor of the thoracic cavity. During inhalation, contraction of the diaphragm increases thoracic cavity volume and contributes to lung expansion.

The enlarged inset shows the pleural membranes in greater detail. From superficial to deep, the layers are the **intercostal muscle**, **parietal pleura**, **pleural cavity**, **visceral pleura**, and **lung**. The inset emphasizes that the pleural cavity lies between the two pleural membranes and serves as a low-friction interface that allows the lungs to expand and recoil smoothly during respiration.

The figure demonstrates how the pleural membranes and pleural fluid reduce friction during breathing and help couple lung movement to movement of the thoracic wall. As the chest wall and diaphragm expand during inhalation, the visceral pleura and lungs follow because of the close relationship between the pleural layers, allowing efficient ventilation.

Labels shown in the figure: Lung, Pleural sac, Parietal pleura, Visceral pleura, Pleural cavity, Intercostal muscles, Intercostal

muscle, Diaphragm, Trachea, and Chest wall (rib cage, sternum, thoracic vertebrae, connective tissue, intercostal muscles).

Figure 8.3.1: Boyle's Law: Relationship Between Gas Volume and Pressure

This figure illustrates **Boyle's law** using two piston-and-cylinder models containing gas particles. The figure demonstrates the inverse relationship between gas volume and gas pressure.

On the left, a piston is shown moving upward, indicated by an upward arrow. As the piston rises, the volume of the container increases. The gas particles are spread farther apart within the enlarged space and collide less frequently with the walls of the container. A text box above the container states, **"As volume increases, pressure decreases."**

On the right, a piston is shown moving downward, indicated by a downward arrow. As the piston descends, the volume of the container decreases. The gas particles occupy a smaller space and collide more frequently with the walls of the container. A text box above the container states, **"As volume decreases, pressure increases."**

Arrows attached to individual gas particles in both containers indicate random particle motion. The figure visually compares the different particle densities associated with larger and smaller volumes.

At the bottom of the figure are mathematical expressions describing Boyle's law:

- $PV = k$
- $P_1V_1 = P_2V_2$
- $P = k/V$

These equations indicate that pressure (**P**) and volume (**V**) are inversely proportional when temperature and the amount of gas remain constant. The product of pressure and volume remains constant (**k**), meaning that an increase in volume results in a decrease in pressure, while a decrease in volume results in an increase in pressure.

Figure 8.3.2: Pressure Relationships in the Thoracic Cavity During Quiet Breathing at Rest

This figure illustrates the lungs within the thoracic cavity and demonstrates the pressure relationships between the atmosphere, alveoli, pleural cavity, and thoracic wall during quiet breathing at rest.

The **trachea** descends into the thoracic cavity and branches into the airways of the right and left lungs. Each **lung** is enclosed by pleural membranes and lies within the thoracic cavity above the **diaphragm**, which forms the floor of the thoracic cavity.

The outer pleural membrane, the **parietal pleura**, lines the inner surface of the thoracic wall. The inner pleural membrane, the **visceral pleura**, adheres directly to the surface of the lung. Between these two membranes is the **pleural cavity**, a thin fluid-filled potential space that allows the pleural surfaces to slide during respiration while maintaining close contact between the lungs and chest wall.

The figure identifies the **thoracic wall**, which surrounds the lungs and includes the rib cage and associated tissues. The thoracic wall tends to recoil outward, while the lungs tend to recoil inward because of their elastic properties. These opposing forces create a pressure difference within the pleural cavity.

An arrow above the trachea indicates **atmospheric pressure** acting on the respiratory system. The pressure within the alveoli, labeled **intra-alveolar pressure**, is shown as **760 mm Hg (0 mm Hg relative to atmospheric pressure)**. At rest, alveolar pressure equals atmospheric pressure, so no net airflow occurs.

The pressure within the pleural cavity, labeled **intrapleural pressure**, is shown as **756 mm Hg (–4 mm Hg relative to atmospheric pressure)**. Because intrapleural pressure is lower than alveolar pressure, the lungs remain expanded rather than collapsing.

The difference between alveolar pressure and intrapleural pressure is labeled **transpulmonary pressure** and is calculated as:

760 mm Hg – 756 mm Hg = 4 mm Hg

This positive transpulmonary pressure acts across the lung wall and keeps the lungs inflated by opposing their natural elastic recoil.

The figure demonstrates that lung expansion depends on the pressure difference between the alveoli and the pleural cavity. The negative intrapleural pressure generates a positive transpulmonary pressure that holds the lungs against the thoracic wall and maintains inflation. Loss of this pressure difference, such as when air enters the pleural cavity, can cause partial or complete lung collapse.

Labels shown in the figure: Atmospheric pressure, Parietal pleura, Visceral pleura, Pleural cavity, Thoracic wall, Lung, Diaphragm, Trachea, Intra-alveolar pressure (760 mm Hg; 0 mm Hg relative), Intrapleural pressure (756 mm Hg; –4 mm Hg relative), and Transpulmonary pressure (4 mm Hg).

Figure 8.3.3: Movements of the Thoracic Cavity During Inspiration and Expiration

This figure compares the movements of the thoracic cavity, diaphragm, and respiratory muscles during **inspiration** (left) and **expiration** (right). Arrows illustrate the direction of airflow and movement of the thoracic structures responsible for ventilation.

Inspiration

The left panel depicts inspiration, the process by which air enters the lungs.

Airflow arrows show air moving through the nose and mouth into the respiratory tract. During inspiration, the **external intercostal muscles contract**, causing the ribs to elevate and move outward. This movement expands the rib cage and increases the dimensions of the thoracic cavity.

At the same time, the **diaphragm contracts**. The figure shows the diaphragm moving downward and flattening, indicated by a large downward arrow. This action increases the vertical dimension of the thoracic cavity.

Together, contraction of the external intercostal muscles and diaphragm causes the **thoracic cavity to expand**. As thoracic volume increases, intrapulmonary pressure decreases below atmospheric pressure, allowing air to flow into the lungs.

Labels in the inspiration panel include:

- Thoracic cavity expands
- External intercostal muscles contract
- Diaphragm
- Diaphragm contracts

Expiration

The right panel depicts expiration, the process by which air leaves the lungs.

Airflow arrows show air moving outward through the nose and mouth. During expiration, the **external intercostal muscles relax**, allowing the rib cage to move downward and inward. A dashed outline indicates the larger thoracic position during inspiration, highlighting the reduction in thoracic volume during expiration.

Simultaneously, the **diaphragm relaxes** and returns to its dome-shaped resting position, moving upward into the thoracic cavity. An upward-directed arrow illustrates this movement.

These actions cause the **thoracic cavity to reduce in size**. As thoracic volume decreases, intrapulmonary pressure rises above atmospheric pressure, causing air to flow out of the lungs.

Labels in the expiration panel include:

- Thoracic cavity reduces
- External intercostal muscles relax

- Diaphragm relaxes

The figure demonstrates the mechanical basis of breathing. Inspiration is an active process driven primarily by contraction of the diaphragm and external intercostal muscles, which increases thoracic cavity volume and draws air into the lungs. Expiration during quiet breathing is primarily passive, occurring when these muscles relax and the elastic recoil of the lungs and thoracic wall decreases thoracic volume and forces air out of the lungs.

Text shown in the figure: Inspiration, Expiration, Thoracic cavity expands, External intercostal muscles contract, Diaphragm contracts, Thoracic cavity reduces, External intercostal muscles relax, and Diaphragm relaxes.

Figure 8.3.4: Major Lung Volumes and Capacities

This figure illustrates the major lung volumes and capacities used to assess pulmonary function. The figure consists of two panels. Panel (a) presents a spirogram tracing and the individual lung volumes, while panel (b) shows how these volumes combine to form larger respiratory capacities.

(a) Lung Volumes

The left panel displays a graph with lung volume on the vertical axis, measured in milliliters (mL), ranging from 0 to 6000 mL. A spirogram tracing shows changes in lung volume during normal and forced breathing.

During quiet breathing, the tracing oscillates within a narrow range representing the **tidal volume**, located between approximately 2500 and 3000 mL. Tidal volume is the amount of air inhaled or exhaled during a normal breath.

Above the tidal volume is the **inspiratory reserve volume (IRV)**, extending from the top of a normal inspiration to approximately 6000 mL. Inspiratory reserve volume represents the additional amount of air that can be inhaled after a normal inspiration.

Below the tidal volume is the **expiratory reserve volume (ERV)**, extending from the end of a normal expiration to approximately 1200 mL. Expiratory reserve volume represents the additional amount of air that can be forcefully exhaled after a normal expiration.

The lowest portion of the graph represents the **residual volume (RV)**, extending from 0 to approximately 1200 mL. Residual volume is the amount of air that remains in the lungs after a maximal forced expiration and cannot be voluntarily expelled.

The spirogram includes a deep inspiration reaching nearly 6000 mL, followed by a forceful expiration reaching approximately 1200 mL, illustrating the reserve volumes above and below normal tidal breathing.

(b) Lung Capacities

The right panel shows four vertical bars representing lung capacities composed of combinations of the individual lung volumes.

Inspiratory Capacity (IC) is shown as the sum of tidal volume and inspiratory reserve volume. It represents the maximum volume of air that can be inhaled after a normal expiration.

Functional Residual Capacity (FRC) is shown as the sum of expiratory reserve volume and residual volume. It represents the volume of air remaining in the lungs after a normal expiration.

Vital Capacity (VC) is shown as the sum of inspiratory reserve volume, tidal volume, and expiratory reserve volume. It represents the maximum volume of air that can be moved into and out of the lungs through voluntary effort.

Total Lung Capacity (TLC) is shown as the sum of all lung volumes, including inspiratory reserve volume, tidal volume, expiratory reserve volume, and residual volume. It represents the total amount of air the lungs can contain after a maximal inspiration.

The figure demonstrates how lung volumes describe individual components of breathing, while lung capacities represent combinations of these volumes that provide information about respiratory function. These measurements are commonly used to evaluate lung health, ventilation, and respiratory disorders.

Labels shown in the figure: Inspiratory reserve volume, Tidal volume, Expiratory reserve volume, Residual volume, Inspiratory capacity, Functional residual capacity, Vital capacity, Total lung capacity, and Milliliters (mL).

Figure 8.3.5: Brainstem Respiratory Control Centers and Neural Regulation of Breathing

This figure illustrates the regions of the brainstem involved in respiratory control and their neural connections to the muscles responsible for breathing. The diagram shows the **pons** and **medulla** within the brainstem, expanded in an enlarged inset that identifies the major respiratory control centers and their interactions.

Within the **pons** are two components of the **pontine respiratory group (PRG)**: the **pneumotaxic center** and the **apneustic center**. The pneumotaxic center provides inhibitory influences on respiratory activity, while the apneustic center provides excitatory influences that promote inspiration. Arrows indicate communication between these pontine centers and the respiratory centers of the medulla.

Within the **medulla** are two major respiratory control regions: the **dorsal respiratory group (DRG)** and the **ventral respiratory group (VRG)**. The DRG and VRG communicate with one another and receive modulatory input from the pontine respiratory centers. Positive (+) and negative (–) symbols indicate excitatory and inhibitory influences between these regions.

The **dorsal respiratory group (DRG)** primarily functions as the inspiratory center and generates the basic rhythm of quiet breathing. Descending neural pathways from the DRG project to muscles involved in inspiration, including the **external intercostal muscles** and the **diaphragm**.

The **ventral respiratory group (VRG)** becomes particularly active during increased ventilatory demand and contributes to both inspiration and forced expiration. Descending pathways from the VRG project to additional respiratory muscles, including the **internal intercostal muscle** and various **accessory respiratory muscles**.

At the bottom right of the figure, the thoracic cavity is shown with labeled **external intercostal muscle** and **diaphragm**. Neural signals from the respiratory centers stimulate these muscles to contract during inspiration, expanding the thoracic cavity and drawing air into the lungs.

At the lower center, a cross-sectional view of the **internal intercostal muscle** is shown. Activation of these muscles contributes to forced expiration by helping pull the ribs downward and inward.

At the lower left, a group of **accessory respiratory muscles** is shown. These muscles are recruited during periods of increased respiratory effort, such as exercise or respiratory distress, to assist in expanding or compressing the thoracic cavity.

The figure demonstrates how respiratory rhythm is generated and regulated by the brainstem. The medullary respiratory centers establish the basic breathing pattern, while pontine centers modify and smooth respiratory activity. Through motor output to the diaphragm, intercostal muscles, and accessory respiratory muscles, these neural circuits coordinate the movements required for effective ventilation.

Labels shown in the figure: Medulla, Pons, Pneumotaxic center, Apneustic center, Pontine respiratory group (PRG), Dorsal respiratory group (DRG), Ventral respiratory group (VRG), Accessory respiratory muscles, Internal intercostal muscle, External intercostal muscle, and Diaphragm.

Figure 8.4.1: Dalton’s Law of Partial Pressures

This figure illustrates **Dalton's law of partial pressures** using three sealed containers containing oxygen gas, nitrogen gas, or a mixture of both gases. Pressure gauges beneath each container display the pressure exerted by the gas molecules.

The left container contains only **oxygen** molecules, represented by blue diatomic particles moving randomly throughout the container. The pressure gauge below the container indicates a pressure of **159 mm Hg**. This value represents the **partial pressure of oxygen** when oxygen is present alone.

The middle container contains only **nitrogen** molecules, represented by orange diatomic particles moving randomly throughout the container. The pressure gauge below the container indicates a pressure of **597 mm Hg**. This value represents the **partial**

pressure of nitrogen when nitrogen is present alone.

The right container contains a mixture of both **oxygen and nitrogen** molecules. Blue oxygen molecules and orange nitrogen molecules are intermixed and move independently within the same volume. The pressure gauge below the container indicates a total pressure of **756 mm Hg**.

The figure demonstrates that each gas contributes independently to the total pressure. The total pressure in the mixed-gas container equals the sum of the individual partial pressures:

- Partial pressure of oxygen ($\mathbf{PO_2}$) = **159 mm Hg**
- Partial pressure of nitrogen ($\mathbf{PN_2}$) = **597 mm Hg**
- Total pressure = **756 mm Hg**

Thus:

PO_2 + PN_2 = Total Pressure

159 mm Hg + 597 mm Hg = 756 mm Hg

The figure illustrates the principle that each gas in a mixture exerts pressure independently of the other gases present. In the respiratory system, oxygen, carbon dioxide, nitrogen, and water vapor each have individual partial pressures. Gas exchange in the lungs and tissues occurs because oxygen and carbon dioxide diffuse from regions of higher partial pressure to regions of lower partial pressure.

Text shown in the figure: Oxygen, Nitrogen, Oxygen + Nitrogen, Pressure 159 mm Hg, Pressure 597 mm Hg, and Pressure 756 mm Hg.

Figure 8.4.2: Gas Exchange Between an Alveolus and a Pulmonary Capillary

This figure illustrates the exchange of oxygen and carbon dioxide between an **alveolus** and a pulmonary capillary. It also shows the chemical reactions occurring within a **red blood cell** that facilitate gas transport in the blood.

The **alveolus** is shown on the left side of the figure, while the surrounding capillary containing blood plasma and a red blood cell is shown on the right. Separating the alveolar air space from the blood is the thin respiratory membrane, which includes the **fused basement membranes** of the alveolar and capillary walls.

Two large purple arrows indicate that **oxygen (O_2)** diffuses from the alveolus across the respiratory membrane into the blood. Some oxygen enters the plasma as **O_2 dissolved in plasma**, while much of it enters red blood cells.

Within the red blood cell, oxygen binds to deoxygenated hemoglobin according to the reaction:

O_2 + HHb → HbO_2 + H⃝

This reaction forms **oxyhemoglobin (HbO_2)** and releases hydrogen ions (H⃝). A label indicates that oxygen has **detached from hemoglobin** on the tissue side and is now reassociating with hemoglobin in the lungs.

Three large black arrows indicate that **carbon dioxide (CO_2)** moves in the opposite direction, diffusing from the blood into the alveolus to be exhaled.

Carbon dioxide reaches the alveolus from three sources:

1. **Dissolved CO_2 in plasma** diffuses directly into the alveolar air space.
2. **Carbaminohemoglobin ($HbCO_2$)** releases carbon dioxide according to the reaction:
   $HbCO_2$ → CO_2 + Hb
3. Bicarbonate-derived carbon dioxide is regenerated within the red blood cell. Bicarbonate ions (**HCO_3⃝**) combine with hydrogen ions (**H⃝**) to form carbonic acid (**H_2CO_3**), which is then rapidly converted to carbon dioxide and water by the enzyme **carbonic anhydrase**:

$H^{\oplus} + HCO_3^{\ominus} \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O$

A label indicates that this carbon dioxide is **converted from bicarbonate**. The enzyme **carbonic anhydrase** accelerates this conversion, allowing carbon dioxide to diffuse efficiently from the blood into the alveolus.

The figure emphasizes that oxygen loading and carbon dioxide unloading occur simultaneously in the pulmonary capillaries. Oxygen enters the blood and binds to hemoglobin, while carbon dioxide is released from dissolved, bicarbonate, and hemoglobin-bound forms and diffuses into the alveoli for exhalation.

The figure demonstrates the mechanisms by which gas exchange occurs in the lungs. Oxygen moves from alveolar air into the blood because alveolar oxygen partial pressure is higher than blood oxygen partial pressure. Carbon dioxide moves from blood to alveolar air because blood carbon dioxide partial pressure is higher than alveolar carbon dioxide partial pressure. Conversion of bicarbonate to carbon dioxide and release of carbon dioxide from carbaminohemoglobin allow most transported carbon dioxide to be eliminated through the lungs.

Labels shown in the figure: Alveolus, Fused basement membranes, Blood plasma, Red blood cell, CO_2 (dissolved in plasma), O_2 (dissolved in plasma), Carbonic anhydrase, Converted from bicarbonate, Detached from hemoglobin, $H^{\oplus} + HCO_3^{\ominus}$, H_2CO_3, $CO_2 + H_2O$, $HbCO_2$ (carbaminohemoglobin), $CO_2 + Hb$, $O_2 + HHb$, and $HbO_2 + H^{\oplus}$.

Figure 8.4.3: Gas Exchange Between Systemic Capillary Blood and Body Tissues

This figure illustrates the exchange of oxygen and carbon dioxide between systemic capillary blood and body tissues. A **tissue cell** is shown on the left, separated from a blood vessel by the **interstitial fluid**. On the right are **blood plasma** and a **red blood cell** within a systemic capillary.

Two purple arrows indicate the movement of **oxygen (O_2)** from the blood toward the tissues. Some oxygen is shown as **O_2 dissolved in plasma**, diffusing from the blood plasma through the interstitial fluid and into the tissue cells. A larger arrow labeled **"Detached from hemoglobin"** shows oxygen being released from hemoglobin within the red blood cell before diffusing into the tissues. This oxygen delivery supports cellular respiration and other metabolic activities within the tissue cells.

Several black arrows indicate the movement of **carbon dioxide (CO_2)** in the opposite direction. Carbon dioxide produced by cellular metabolism leaves the tissue cells and enters the **interstitial fluid**. From there, carbon dioxide diffuses across the capillary wall into the blood plasma and red blood cell.

The figure emphasizes the opposite directions of oxygen and carbon dioxide transport at the tissues: oxygen leaves the blood and enters cells, while carbon dioxide leaves cells and enters the blood. These diffusion movements occur because oxygen partial pressure is higher in the blood than in the tissues, whereas carbon dioxide partial pressure is higher in the tissues than in the blood.

This figure demonstrates **internal respiration**, the exchange of gases between systemic blood and body tissues. Oxygen unloading from hemoglobin allows tissues to receive the oxygen required for cellular metabolism. At the same time, carbon dioxide generated as a metabolic waste product enters the blood, where it will be transported to the lungs for elimination during exhalation.

Labels shown in the figure: Tissue cell, Interstitial fluid, Blood plasma, Red blood cell, Detached from hemoglobin, O_2 dissolved in plasma, O_2, and CO_2.

Figure 8.4.4: Hyperbaric Oxygen Therapy Chambers in a Clinical Treatment Room

This photograph shows a clinical hyperbaric oxygen therapy facility containing multiple **hyperbaric chambers** arranged in a treatment room. The chambers are large, cylindrical devices designed to accommodate a patient lying in a reclined position while receiving oxygen under increased atmospheric pressure.

In the foreground is a beige-colored hyperbaric chamber with a transparent cylindrical enclosure extending horizontally from a

rounded access door. The chamber contains a mattress and bedding for patient comfort. Control panels with gauges, dials, and monitoring instruments are located along the side of the unit. The access door includes a handle and several circular fittings used to seal and operate the chamber.

A second hyperbaric chamber is visible behind the first, positioned parallel to it. This chamber also contains pillows and bedding and has similar control instrumentation and transparent walls, allowing observation of the chamber interior during treatment.

The treatment room has red carpeting and contains additional medical equipment and furnishings, including a chair and a wheeled cart. Posters or educational displays are mounted on the walls in the background, and a window with blinds is visible along one wall. Overhead lighting illuminates the room and reflects from the transparent chamber surfaces.

The image demonstrates the equipment used for **hyperbaric oxygen therapy (HBOT)**, a treatment in which patients breathe oxygen in a pressurized environment. Increasing environmental pressure allows more oxygen to dissolve directly into the blood plasma, increasing oxygen availability to body tissues and supporting treatment of selected medical conditions.

Hyperbaric oxygen chambers are used to increase the amount of oxygen dissolved in the blood beyond what can be achieved under normal atmospheric conditions. The higher oxygen delivery may promote tissue healing, support recovery in areas with reduced blood supply, and assist in the treatment of conditions such as decompression sickness, carbon monoxide poisoning, and certain nonhealing wounds.

Visible elements in the image: Hyperbaric chambers, transparent chamber enclosures, access doors, control gauges, control panels, mattresses, pillows, treatment room furnishings, wall posters, window blinds, and medical equipment.

Figure 8.5.1: Structure of Hemoglobin and Its Oxygen-Binding Sites

This figure illustrates the structure of a **hemoglobin** molecule and its role in oxygen transport within red blood cells. A red blood cell is shown in the upper left corner with an arrow pointing toward an enlarged representation of a hemoglobin molecule.

The hemoglobin molecule is depicted as a complex protein composed of four folded protein subunits arranged together to form a quaternary structure. The subunits are shown as intertwined purple ribbon-like structures. Embedded within each subunit is a small blue-green circular structure representing a **heme group**.

The figure shows a total of **four heme groups**, one associated with each protein subunit. Each heme group contains an iron ion that can reversibly bind oxygen. The arrangement illustrates how a single hemoglobin molecule possesses four potential oxygen-binding sites.

The arrow connecting the red blood cell to the hemoglobin molecule indicates that hemoglobin is located inside red blood cells and serves as the primary oxygen-transport protein in the blood.

Hemoglobin binds oxygen in the lungs and releases oxygen in the tissues. Because each hemoglobin molecule contains four heme groups, it can bind up to four oxygen molecules simultaneously. This greatly increases the oxygen-carrying capacity of blood compared with oxygen dissolved in plasma alone.

The figure emphasizes the relationship between red blood cells and hemoglobin and highlights the structural basis for oxygen transport: four globin subunits, each containing a heme group capable of binding oxygen.

Elements shown in the figure: Red blood cell, hemoglobin molecule, four globin protein subunits, four heme groups (oxygen-binding sites), and an arrow indicating hemoglobin located within the red blood cell.

Figure 8.5.2: Oxygen-Hemoglobin Dissociation Curve

This figure presents the **oxygen-hemoglobin dissociation curve**, which illustrates how the percentage of hemoglobin saturated with oxygen changes as the **partial pressure of oxygen (PO_2)** increases.

The horizontal axis represents **partial pressure of oxygen (mm Hg)** and ranges from **0 to 100 mm Hg**. The left side of the axis is labeled **Tissues** at approximately **40 mm Hg**, and the right side is labeled **Lungs** at approximately **100 mm Hg**.

The left vertical axis represents **O_2 saturation of hemoglobin (%)**, ranging from **0% to 100%**. A second vertical axis on the

right represents the oxygen content of blood, ranging from **0 to 20 mL O_2 per 100 mL blood**.

A green S-shaped (sigmoidal) curve begins near the origin, rises steeply between approximately **20 and 60 mm Hg**, and gradually levels off as it approaches 100% saturation near **100 mm Hg**.

At the **lung PO_2 of approximately 100 mm Hg**, the curve is nearly at its maximum, indicating that hemoglobin is almost completely saturated with oxygen, approaching **100% saturation** and carrying approximately **20 mL O_2 per 100 mL blood**.

At the **tissue PO_2 of approximately 40 mm Hg**, the curve intersects a horizontal dashed line at approximately **75% hemoglobin saturation**. A vertical arrow extends upward from the 40 mm Hg mark to this point on the curve, highlighting the typical oxygen saturation of blood reaching systemic tissues.

A bracket on the right side of the graph extends from approximately **20 mL O_2 per 100 mL blood** down to **15 mL O_2 per 100 mL blood** and is labeled **"Volume of O_2 unloaded to tissues."** This illustrates that about **5 mL O_2 per 100 mL blood** is released from hemoglobin as blood passes through resting tissues.

The steep middle portion of the curve indicates that relatively small decreases in PO_2 produce substantial oxygen unloading, whereas the flattened upper portion indicates that hemoglobin remains highly saturated across a broad range of alveolar oxygen pressures.

The figure demonstrates how hemoglobin efficiently transports oxygen. In the lungs, where PO_2 is high, hemoglobin becomes nearly fully saturated with oxygen. In the tissues, where PO_2 is lower, hemoglobin releases a portion of its bound oxygen. The sigmoidal shape of the curve promotes oxygen loading in the lungs while facilitating oxygen unloading where tissues require it.

Labels shown in the figure: O_2 saturation of hemoglobin (%), Partial pressure of O_2 (mm Hg), Tissues, Lungs, mL O_2/100 mL blood, Volume of O_2 unloaded to tissues, and Partial pressure of oxygen and hemoglobin saturation.

Figure 8.5.3: Effect of Blood pH on the Oxygen-Hemoglobin Dissociation Curve

This graph illustrates the effect of **blood pH** on the relationship between the **partial pressure of oxygen (PO_2)** and **hemoglobin oxygen saturation**. Three oxygen-hemoglobin dissociation curves are shown for blood pH values of **7.6**, **7.4**, and **7.2**.

The horizontal axis represents **partial pressure of oxygen (PO_2)** in millimeters of mercury (mm Hg), ranging from **0 to 100 mm Hg**. The vertical axis represents **oxygen saturation of hemoglobin (%)**, ranging from **0% to 100%**.

All three curves have a sigmoidal (S-shaped) appearance, reflecting cooperative oxygen binding by hemoglobin. However, the position of each curve differs depending on blood pH.

The **pH 7.6 curve** is shifted farthest to the left. At any given PO_2, hemoglobin exhibits the highest oxygen saturation among the three curves, indicating an increased affinity for oxygen. Hemoglobin binds oxygen more readily and releases less oxygen to tissues.

The **pH 7.4 curve**, representing normal arterial blood pH, occupies an intermediate position. It demonstrates the typical balance between oxygen loading in the lungs and oxygen unloading in peripheral tissues.

The **pH 7.2 curve** is shifted to the right. At any given PO_2, hemoglobin saturation is lower than at higher pH values, indicating a reduced affinity for oxygen. This rightward shift promotes oxygen release from hemoglobin to the tissues.

The graph demonstrates that as pH decreases from **7.6 to 7.2**, the oxygen-hemoglobin dissociation curve progressively shifts to the right. Conversely, increasing pH shifts the curve to the left.

This figure demonstrates the **Bohr effect**, in which changes in blood pH alter hemoglobin's affinity for oxygen. In tissues with high metabolic activity, increased carbon dioxide production leads to a decrease in pH. The resulting rightward shift of the curve promotes oxygen unloading, helping meet tissue oxygen demands. In contrast, higher pH increases hemoglobin's affinity for oxygen, favoring oxygen binding and retention.

The pH-dependent shift in the oxygen-hemoglobin dissociation curve helps match oxygen delivery to the metabolic needs of

tissues throughout the body.

Labels shown in the figure: O_2 saturation of hemoglobin (%), Partial pressure of O_2 (mm Hg), pH 7.6, pH 7.4, pH 7.2, and Effect of pH.

Figure 8.5.4: Effect of Temperature on the Oxygen-Hemoglobin Dissociation Curve

This graph illustrates the effect of **temperature** on the relationship between the **partial pressure of oxygen (PO_2)** and **hemoglobin oxygen saturation**. Four oxygen-hemoglobin dissociation curves are shown, representing temperatures of **10°C**, **20°C**, **38°C**, and **43°C**.

The horizontal axis represents **partial pressure of oxygen (PO_2)** in millimeters of mercury (mm Hg), ranging from **0 to 100 mm Hg**. The vertical axis represents **oxygen saturation of hemoglobin (%)**, ranging from **0% to 100%**.

All four curves have a sigmoidal (S-shaped) appearance but differ in position along the horizontal axis.

The **10°C curve** is shifted farthest to the left. At any given PO_2, hemoglobin exhibits the highest oxygen saturation among the four curves, indicating a greater affinity for oxygen. Hemoglobin binds oxygen readily and releases it less easily to tissues.

The **20°C curve** is also left-shifted relative to normal body temperature. Hemoglobin remains highly saturated at relatively low oxygen partial pressures, reflecting increased oxygen affinity compared with warmer temperatures.

The **38°C curve**, which approximates normal body temperature, occupies an intermediate position. Hemoglobin demonstrates a balance between oxygen loading in the lungs and oxygen unloading in the tissues.

The **43°C curve** is shifted farthest to the right. At any given PO_2, hemoglobin saturation is lower than at cooler temperatures, indicating reduced oxygen affinity. More oxygen is released from hemoglobin to the tissues at the same oxygen partial pressure.

The graph shows that as temperature increases from 10°C to 43°C, the dissociation curve progressively shifts to the right. Conversely, decreasing temperature shifts the curve to the left.

The figure demonstrates how temperature influences oxygen transport by hemoglobin. Increased temperature, such as during exercise or in metabolically active tissues, decreases hemoglobin's affinity for oxygen and promotes oxygen unloading where oxygen demand is high. Decreased temperature increases hemoglobin affinity for oxygen, allowing oxygen to remain more tightly bound to hemoglobin and reducing oxygen release to tissues.

This temperature-dependent shift helps match oxygen delivery to tissue metabolic needs. Warm, active tissues receive more oxygen because hemoglobin releases oxygen more readily, while cooler tissues receive less oxygen unloading.

Labels shown in the figure: O_2 saturation of hemoglobin (%), Partial pressure of O_2 (mm Hg), 10°C, 20°C, 38°C, 43°C, and Effect of temperature.

Figure 8.5.5: Comparison of Fetal and Adult Hemoglobin Oxygen Dissociation Curves

This graph compares the oxygen-binding properties of **fetal hemoglobin** and **adult hemoglobin** by showing their oxygen-hemoglobin dissociation curves.

The horizontal axis represents the **partial pressure of oxygen (PO_2)** and extends from approximately **0 to 140 mm Hg**. The vertical axis represents **oxygen saturation of hemoglobin (%)**, ranging from **0% to 100%**.

Two sigmoidal (S-shaped) curves are plotted:

- A **teal curve** labeled **Fetal hemoglobin**.
- An **orange curve** labeled **Adult hemoglobin**.

The fetal hemoglobin curve is shifted to the **left** of the adult hemoglobin curve. At nearly every oxygen partial pressure, fetal hemoglobin exhibits a higher percentage saturation than adult hemoglobin, indicating a greater affinity for oxygen.

Dashed reference lines highlight the partial pressure of oxygen at which each hemoglobin type is **50% saturated**. The graph shows:

- **Fetal hemoglobin P50 ≈ 19 mm Hg**
- **Adult hemoglobin P50 ≈ 26.8 mm Hg**

Because fetal hemoglobin reaches 50% saturation at a lower PO_2 than adult hemoglobin, it binds oxygen more readily.

A second dashed horizontal line marks approximately **95.8% oxygen saturation**. At a PO_2 of about **80 mm Hg**, both fetal and adult hemoglobin approach this high level of saturation, and their curves begin to plateau. As oxygen partial pressure continues to increase, both curves approach nearly complete saturation near **100%**.

The graph emphasizes that the fetal hemoglobin curve remains above the adult hemoglobin curve throughout most of the physiological range of oxygen pressures.

The figure demonstrates that fetal hemoglobin has a higher affinity for oxygen than adult hemoglobin. This difference enables fetal blood to extract oxygen from maternal blood in the placenta, even though both blood supplies are exposed to relatively similar oxygen partial pressures. The leftward shift of the fetal hemoglobin curve facilitates oxygen loading by the fetus and supports oxygen delivery during prenatal development.

Labels Shown in the Figure

- O_2 saturation of hemoglobin (%)
- Partial pressure of O_2
- Fetal hemoglobin
- Adult hemoglobin
- 19 mm Hg (P50 for fetal hemoglobin)
- 26.8 mm Hg (P50 for adult hemoglobin)
- 95.8% oxygen saturation
- 100% oxygen saturation

Figure 8.5.6: Transport of Carbon Dioxide from Tissues in the Blood

This figure illustrates the movement of **carbon dioxide (CO_2)** from body tissues into the blood and the major forms in which carbon dioxide is transported in the circulation. Tissue cells surround a small blood vessel containing plasma and red blood cells.

Several arrows show **CO_2** diffusing from the surrounding tissue cells across the interstitial fluid and into the bloodstream. Once in the blood, carbon dioxide is transported by three different mechanisms, labeled (a), (b), and (c).

(a) CO_2 Carried in Red Blood Cells

A large arrow indicates carbon dioxide entering a **red blood cell (RBC)**. Inside the red blood cell, carbon dioxide combines with water according to the reaction:

$CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow HCO_3^- + H^+$

Carbon dioxide is first converted to **carbonic acid (H_2CO_3)** and then dissociates into **bicarbonate ions (HCO_3^-)** and **hydrogen ions (H^+)**. This conversion occurs within red blood cells and represents the primary pathway for carbon dioxide transport from tissues.

(b) HCO_3^- Transported in Plasma

An arrow extending from the red blood cell into the surrounding blood plasma indicates the movement of **bicarbonate ions (HCO_3^-)** out of the red blood cell and into the plasma. Bicarbonate dissolved in plasma represents the largest fraction of carbon

dioxide transport in the blood.

(c) CO_2 Dissolved in Plasma

Another arrow shows a portion of carbon dioxide entering the bloodstream and remaining as **CO_2 dissolved directly in plasma**. This dissolved carbon dioxide is transported without chemical conversion.

The figure depicts tissue cells as the source of carbon dioxide produced during cellular respiration. Carbon dioxide diffuses down its concentration gradient from the tissues into the blood, where it is carried toward the lungs for elimination during exhalation.

The figure demonstrates the three major forms of carbon dioxide transport in blood:

1. **As bicarbonate ions (HCO_3^-) in plasma** after conversion within red blood cells.
2. **Within red blood cells** as part of the carbonic acid-bicarbonate system and carbon dioxide reactions.
3. **Dissolved directly in plasma.**

These transport mechanisms allow carbon dioxide generated by cellular metabolism to be efficiently carried from tissues to the lungs, where it is released and exhaled.

Labels shown in the figure: CO_2 carried in RBC, HCO_3^- dissolved in plasma as carbonic acid, CO_2 dissolved in plasma, CO_2 + H_2O, H_2CO_3, HCO_3^- + H^+, and tissue cells.

Figure 8.7.1: Embryonic Development of the Respiratory System

This figure illustrates the early embryonic development of the respiratory system from the beginning of the fourth week through approximately eight weeks of development. A series of diagrams connected by arrows shows the progressive formation and branching of the respiratory tract.

At the **beginning of the fourth week**, a tube-like structure called the **laryngotracheal tube** extends from the **pharynx** and is surrounded by **splanchnic mesoderm**. This structure represents the early respiratory diverticulum from which the lower respiratory tract will develop.

As development proceeds, the distal end of the laryngotracheal tube enlarges and forms paired **tracheal buds**. These buds project laterally from the lower end of the developing airway and mark the beginning of lung development.

The next stage shows separation of the developing **esophagus** and **trachea**. The tracheal buds continue to enlarge and extend outward from the trachea. The figure emphasizes the establishment of separate respiratory and digestive pathways.

By the **end of the fourth week**, the trachea is identifiable and each lateral outgrowth is labeled a **bronchial bud**. These paired bronchial buds represent the future right and left primary bronchi.

Subsequent stages show continued branching of the bronchial buds. The **trachea bifurcates**, producing increasingly complex branches. Additional bronchial buds emerge from the primary branches, representing the formation of the bronchial tree.

As development continues, numerous **bronchial buds develop**, creating a progressively branched respiratory network within the growing lung tissue. Repeated branching establishes the framework of the future conducting airways.

By approximately **eight weeks**, the lungs have expanded considerably and distinct lobes are visible. The **right lung** is divided into:

- Upper lobe
- Middle lobe
- Lower lobe

The **left lung** is divided into:

- Upper lobe
- Lower lobe

Within both lungs, extensive branching of the developing airways is visible, demonstrating the formation of the bronchial tree.

The figure demonstrates how the lower respiratory tract develops from a simple embryonic tube through repeated branching morphogenesis. The laryngotracheal tube gives rise to the trachea and primary bronchi, while successive branching of the bronchial buds forms the bronchial tree and establishes the lobar organization of the lungs.

Labels shown in the figure: Pharynx, Laryngotracheal tube, Splanchnic mesoderm, Tracheal buds, Esophagus, Trachea, Bronchial bud, Trachea bifurcates, Bronchial buds develop, Upper lobe, Middle lobe, Lower lobe, Beginning of 4th week, End of 4 weeks, and 8 weeks.

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GLOSSARY

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