allergies, and the oral inhaler is used for long-term control of asthma. Fluticasone is also used in a combination product with salmeterol. It decreases the frequency and severity of asthma attacks and improves overall asthma symptoms. See Figures 5.14-16
"Fluticasone Propionate Nasal Spray" by _BuBBy_ is licensed under CC BY 2.0
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"Fluticasone.JPG" by James Heilman, MD is licensed under CC BY-SA 4.0
,
"Asthmatic Control" by David Camerer is licensed under CC BY-NC-ND 2.0
for images of different formulations of fluticasone.

Oral prednisone prevents the release of substances in the body that cause inflammation. It also suppresses the immune system.

Methylprednisolone IV prevents the release of substances in the body that cause inflammation. It also suppresses the immune system. Methylprednisolone requires reconstitution before administration. See Figure 5.17
"Methylprednisolone vial.jpg" by Intropin is licenced under CC BY 3.0
for an image of methylprednisolone.

**Indications for Use**

Fluticasone inhalers are used to prevent asthma attacks. In respiratory conditions, oral prednisone is used to control severe or incapacitating allergic conditions that are unresponsive to adequate trials of conventional treatment for seasonal or perennial allergic rhinitis, bronchial asthma, contact dermatitis, atopic dermatitis, serum sickness, and drug hypersensitivity reactions. Methylprednisolone IV is used to rapidly control these same conditions.

**Nursing Considerations Across the Lifespan**

Fluticasone is safe for 4 years and older. Prednisone and methylprednisolone are safe for all ages.

**Adverse/Side Effects**

Fluticasone can cause hoarseness, dry mouth, cough, sore throat, and oropharyngeal candidiasis. Patients should rinse their mouths after use to prevent candidiasis (thrush).

Prednisone and methylprednisolone: See more information about adverse effects of corticosteroids in the "Endocrine" chapter. Cardiovascular symptoms can include fluid retention, edema, and hypertension. Imbalances such as hypernatremia (↑Na), hypokalemia (↓ K+), and increased blood glucose with associated weight gain can occur. CNS symptoms include mood swings and euphoria. GI symptoms can include nausea, vomiting, and GI bleed. In long- term therapy, bone resorption occurs, which increases the risk for fractures; the skin may bruise easily and become paper thin; wound healing is delayed; infections can be masked; and the risk for infection increases. Long-term corticosteroid therapy should never be stopped abruptly because adrenal insufficiency may occur.
Frandsen, G. & Pennington, S. (2018). *Abrams' clinical drug: Rationales for nursing practice* (11th ed.). Wolters Kluwer.

Figure 5.14 Fluticasone nasal spray formulation

Figure 5.15 Fluticasone oral inhaler

formulation


Figure 5.16 Fluticasone combination formulation


Figure 5.17 Methylprednisolone requires reconstitution before administration

**Patient Teaching & Education**

Patients should be advised that corticosteroids are not used to treat an acute asthma attack. They can cause immunosuppression and suppress signs of infection. Corticosteroids can also cause an increase in blood glucose levels. Patients may experience weight gain, swelling, increased fatigue, bruising, and behavioral changes. These occurrences should be reported to one's healthcare provider.
uCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral

Now let's take a closer look at the medication grid for fluticasone, prednisone, and methylprednisolone in Table 5.12.


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Frandsen, G. & Pennington, S. (2018). *Abrams' clinical drug: Rationales for nursing practice* (11th ed.). Wolters Kluwer.

,



Table 5.12 Fluticasone, Prednisone, and Methylprednisolone Medication Grid

| Class/Subclass | Prototype/Generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Corticosteroids** | fluticasone | Rinse mouth after use<br><br>Do not use as a "rescue" medication | Nasal spray: Used for management of the nasal symptoms of perennial nonallergic rhinitis<br><br>Inhaler: Used to improve the control of asthma by reducing inflammation in the airways | Hoarseness, dry mouth, cough, sore throat, and oropharyngeal candidiasis |
| **Corticosteroids** | prednisone | Do not use if signs of a systemic infection<br><br>When using more than 10 days, the dose must be slowly tapered<br><br>May increase blood glucose levels | Used to control severe or incapacitating allergic or respiratory conditions | CV: fluid retention, edema, and hypertension<br><br>Electrolytes: ↑Na, ↓K+, ↑Ca, ↑BG<br><br>CNS: mood swings and euphoria in high doses<br><br>GI: Nausea/ Vomiting, GI bleed |

| | | | | |
|---|---|---|---|---|
| | | | | MS: bone resorption<br><br>Skin: acne, paper thin, bruises, infections, and delayed healing<br><br>Weight gain<br><br>Adrenal suppression<br><br>Increased risk for infection and infections can be masked<br><br>Long-term use may result in Cushing's syndrome |
| **Corticosteroids** | methylprednisolone | May increase blood glucose levels | Used to rapidly control severe or incapacitating allergic or respiratory conditions, in sepsis to reduce systemic inflammation, and to treat adrenal insufficiency | Same as prednisone |

# 5.13 Leukotriene Receptor Antagonists

Open Resources for Nursing (Open RN)

Montelukast is a leukotriene antagonist medication with a distinctly shaped tablet. See Figure 5.18.


**Mechanism of Action**

Montelukast blocks leukotriene receptors and decreases inflammation.

**Indications for Use**

Montelukast is used for the long-term control of asthma and for decreasing the frequency of asthma attacks. It is also indicated for exercise-induced bronchospasm and allergic rhinitis.

**Nursing Considerations Across the Lifespan**

The medication is safe for children 12 months and older. It is available in granule packets and chewable tablets, as well as regular tablets.

**Adverse/Side Effects**

Montelukast can cause headache, cough, nasal congestion, nausea, and hepatotoxicity.
Frandsen, G. & Pennington, S. (2018). *Abrams' clinical drug: Rationales for nursing practice* (11th ed.). Wolters Kluwer.

Figure 5.18 Montelukast Tablets

**Patient Teaching & Education**

Patients should be instructed to take medications at the same time each day and at least two hours prior to exercise. They should not discontinue medications without notifying the healthcare provider.
uCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral

Now let's take a closer look at the medication grid on montelukast in Table 5.13.


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Frandsen, G. & Pennington, S. (2018). *Abrams' clinical drug: Rationales for nursing practice* (11th ed.). Wolters Kluwer.

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Adams, M., Holland, N., & Urban, C. (2020). *Pharmacology for nurses: A pathophysiologic approach* (6th ed.). pp. 622-63 & 626. Pearson.

Table 5.13 Montelukast Medication Grid

| Class/Subclass | Prototype/ Generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Leukotriene inhibitor** | montelukast | Use as directed; not to be used as "rescue" medication | Prevention and treatment of asthma and exercise-induced bronchoconstriction | Headache<br>Cough |

| | |
|---|---|
| | Nasal congestion |
| Typically 3-7 days to reach effectiveness | Nausea |
| | Hepatotoxicity |

# 5.14 Xanthine Derivatives

Open Resources for Nursing (Open RN)

Theophylline is a xanthine derivative.

**Mechanism of Action**

Theophylline relaxes bronchial smooth muscle by inhibition of the enzyme phosphodiesterase and suppresses airway responsiveness to stimuli that cause bronchoconstriction.

**Indications for Use**

Theophylline is used for the long-term management of persistent asthma that is unresponsive to beta agonists or inhaled corticosteroids.

**Adverse/Side Effects**

Theophylline can cause nausea, vomiting, CNS stimulation, nervousness, and insomnia.
Frandsen, G. & Pennington, S. (2018). *Abrams' clinical drug: Rationales for nursing practice* (11th ed.). Wolters Kluwer.

**Patient Teaching & Education**

Patients should be sure to take medications as prescribed at appropriate intervals. They should avoid irritants and drink fluids to help thin secretions. Patients will need serum blood levels tested every six to twelve months.
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Now let's take a closer look at the medication grid on theophylline in Table 5.14.


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Frandsen, G. & Pennington, S. (2018). *Abrams' clinical drug: Rationales for nursing practice* (11th ed.). Wolters Kluwer.

,



Table 5.14 Theophylline Medication Grid

| Class/ Subclass | Prototype/ Generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Xanthine** | theophylline | Avoid caffeine<br><br>Requires evaluation of therapeutic blood level to prevent toxicity | Long-term treatment of chronic asthma and COPD unresponsive to other treatment | GI: Nausea, vomiting<br><br>CNS stimulation<br><br>Nervousness and insomnia |

# 5.15 Module Learning Activities

Open Resources for Nursing (Open RN)

## Image of lightbulb
## Lightbulb Momentin a circle

Let's apply what you have learned in the respiratory unit.

**Asthma Scenario**

An adult patient presents to the emergency department with complaints of shortness of breath and increased work of breathing. The patient is alert and oriented times 3, skin is pink, warm and dry, BP 148/88, T 98, P92, R 24, pulse oximetry 91% on room air. Assessment of the lung reveals expiratory wheezing throughout the lung fields. The patient has a past medical history of asthma, hypertension, and diabetes.

1. The nurse anticipates which of the following medications will be initially administered to the patient?

    a) Theophylline
    b) Montelukast
    c) Albuterol
    d) Salmeterol

2. List the steps the nurse should take to safely administer the medication.

3. What assessments should the nurse plan to complete after administering the medication?

4. The nurse plans on teaching the patient about using the albuterol inhaler at home. What information should be included?

5. What is the best method for the nurse to use to ensure that the patient is correctly using an inhaler?

**Allergy Scenario**

A pediatric patient presents to the emergency department with complaints of shortness of breath, increased work of breathing, and a cough. The patient is alert and oriented times 3, skin is pink, warm and dry, BP 112/68, T 99, P106, R 32, pulse oximetry 90% on room air. Assessment of the lung sounds reveals diminished lung sounds throughout all lung fields. The patient has a past medical history of peanut allergy. The mother tells you that they were at a birthday party and after consumption of a cupcake, the symptoms started.

6. The nurse anticipates that which of the following medication will be likely ordered for this patient?

a. Diphenhydramine

b. Epinephrine

c. Cetirizine

d. Guaifenesin

Note: Answers to the Lightbulb Moment can be found in the "Answer Key" sections at the end of the book.

## Interactive Activity

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## Interactive Activity

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## Interactive Activity

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# V. Glossary

Open Resources for Nursing (Open RN)

**Allergies:** Allergies occur when the immune system reacts to a foreign substance and makes antibodies that identify a particular allergen as harmful, even though it isn't.

**Anaphylaxis:** A severe, potentially life-threatening allergic reaction. It can occur within seconds or minutes of exposure to something you're allergic to, such as peanuts or bee stings.

**Cyanotic:** A bluish or purplish discoloration (as of skin) due to deficient oxygenation of the blood.

**Gas Exchange:** The process at the alveoli level where blood is oxygenated and carbon dioxide, the waste product of cellular respiration, is removed from the body.

**Pallor:** A deficiency of color especially of the face; paleness.

**Paradoxical Effect:** An effect that is opposite to what is expected.

**Respiratory Rate:** The total number of breaths, or respiratory cycles, that occur each minute. 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.

**Sputum:** Matter expectorated from the respiratory system and especially the lungs that is composed of

mucus but may contain pus, blood, fibrin, or microorganisms (such as bacteria) in diseased states.

VI

# Cardiovascular & Renal System

# 6.1 Cardiovascular and Renal System Introduction

Open Resources for Nursing (Open RN)

## Learning Objectives

- Cite the classifications and actions of cardiovascular drugs
- Cite the classifications and actions of renal system drugs
- Give examples of when, how, and to whom cardiovascular system drugs may be administered
- Give examples of when, how, and to whom renal system drugs may be administered
- Identify the side effects and special considerations associated with cardiovascular and renal system drug therapy
- Identify considerations and implications of using cardiovascular system medications across the life span
- Identify considerations and implications of using renal system medications across the life span
- Apply evidence-based concepts when using the nursing process
- Identify and interpret related laboratory tests

The heart is the powerhouse of the body, providing oxygenated blood to organs so that they can conduct the vital processes needed to keep the body functioning. Without a properly functioning heart to ensure blood flow, cells are in jeopardy of oxygenation starvation, impairment, and subsequent death.

Did you know that the average adult human heart contracts 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 heartbeat ejects approximately 70 mL blood, resulting in 5.25 liters of fluid per minute and approximately 14,000 liters per day. Over one year, that means over 2.6 million gallons of blood are sent through roughly 60,000 miles of vessels in the adult body.



It is no wonder that the heart is the most important muscle of the body! This chapter will review important concepts and disorders related to the heart and cardiovascular system before discussing common medication classes. It is vital for nurses to understand how these cardiovascular medications work to provide safe, effective care to the patients who take them.

# 6.2 Review of Basic Concepts

Open Resources for Nursing (Open RN)

To understand the effects of various cardiovascular medications, it is important to first understand the basic anatomy and physiology of the cardiovascular and renal system.

## Location of the Heart

The human heart is located within the thoracic cavity, medially between the lungs in the space known as the mediastinum. 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.



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. It is important to remember the position of the heart when placing a stethoscope on the chest of a patient and listening for heart sounds.

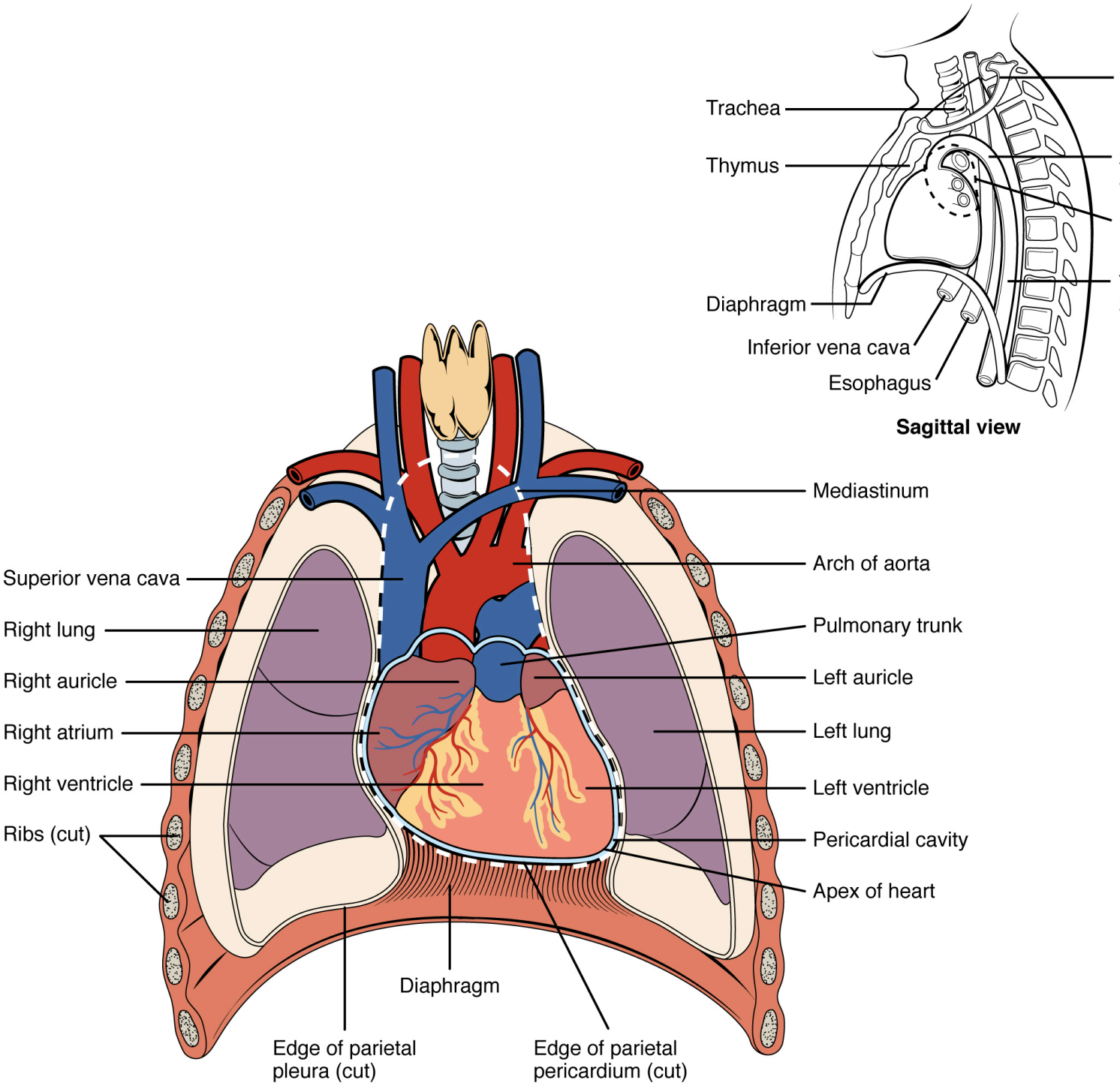



Figure 6.1 Position of the heart in the thoracic cavity

## Chambers and Circulation Through the Heart

The heart consists of four chambers: two atria and two ventricles. The right atrium receives deoxygenated blood from the systemic circulation, and the left atrium receives oxygenated blood from the lungs. The atria contract to push blood into the lower chambers, the right ventricle and the left ventricle. The right ventricle contracts to push blood into the lungs, and the left ventricle is the primary pump that propels blood to the rest of the body.

There are two distinct but linked circuits in the human circulation called the pulmonary and systemic circuits. 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 deoxygenated blood and carbon dioxide to the heart to be sent back to the pulmonary circulation. See Figure 6.2



for an illustration of blood flow through the heart and blood circulation throughout the body.

Figure 6.2 Chambers of the heart and blood circulation to the lungs and throughout the body

Blood also circulates through the coronary arteries with each beat of the heart. The left coronary artery distributes blood to the left side of the heart, and the right coronary distributes blood to the right atrium, portions of both ventricles, and the heart conduction system. See Figure 6.3



for an illustration of the coronary arteries. When a patient has a myocardial infarction, a blood clot lodges in one of these coronary arteries that perfuse the heart tissue. If a significant area of muscle tissue dies from lack of perfusion, the heart is no longer able to pump.

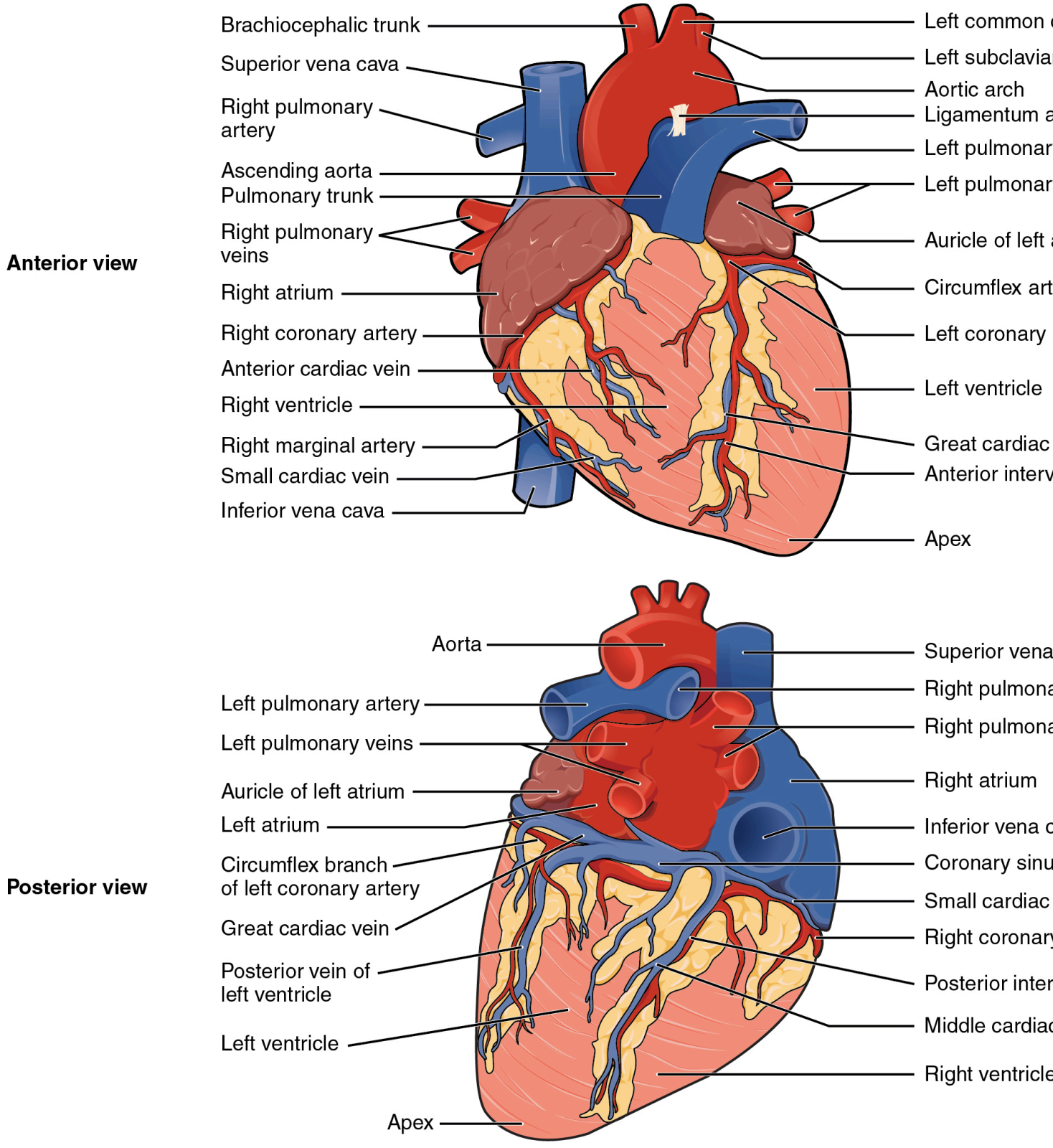



Figure 6.3 Coronary arteries of the heart

## Conduction System of the Heart

Contractions of the heart are stimulated by the electrical conduction system. The components of the cardiac conduction system include the sinoatrial (SA) node, the atrioventricular (AV) node, the left and right bundle branches, and the Purkinje fibers. (See Figure 6.4 for an image of the conduction system of the heart.



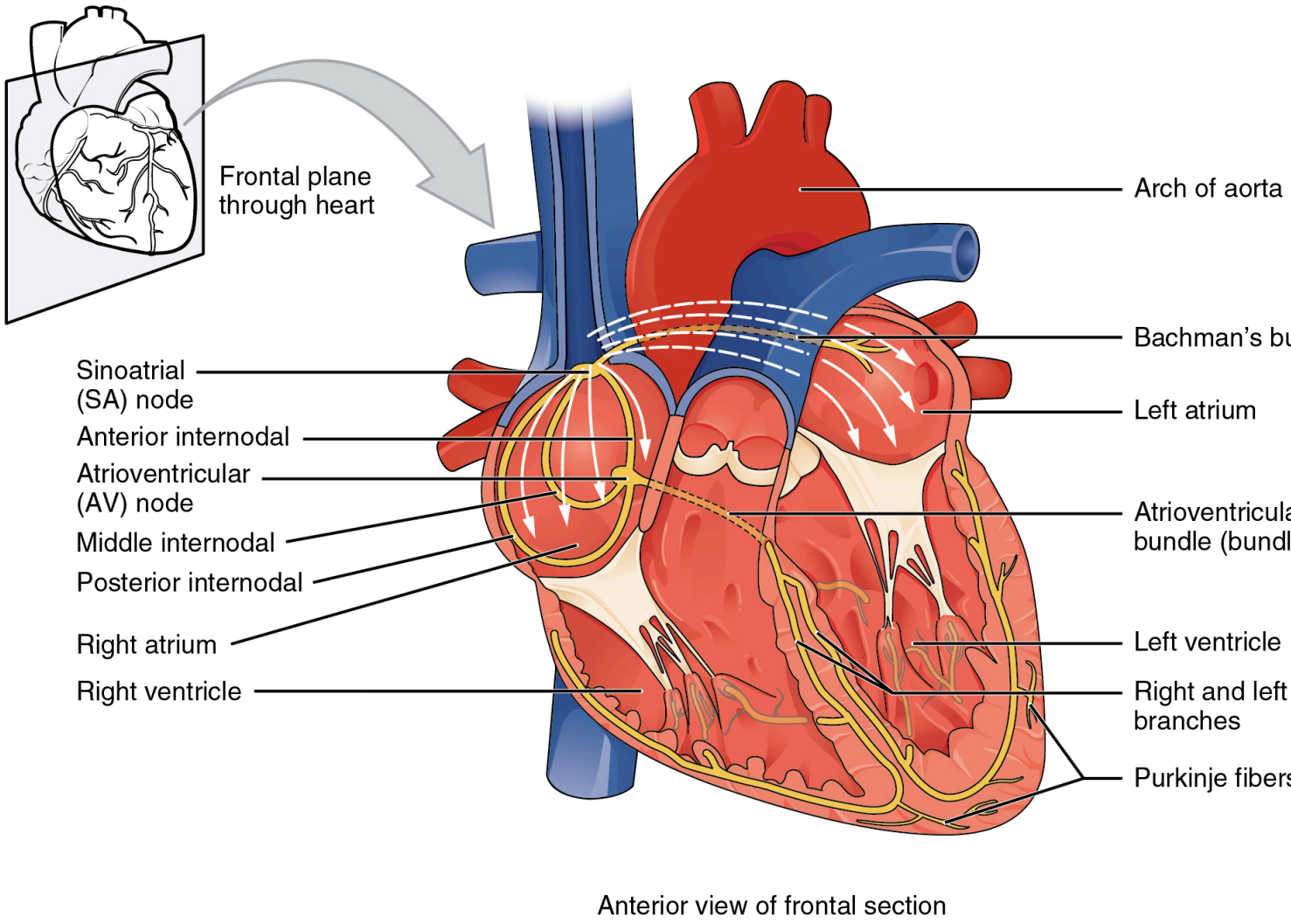



Figure 6.4 Components of the cardiac conduction system

Normal cardiac rhythm is established by the **sinoatrial (SA) node**. The SA node has the highest 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. The SA node initiates the action potential, which sweeps across the atria through the AV node to the bundle branches and Purkinje fibers, and then spreads to the contractile fibers of the ventricle to stimulate the contraction of the ventricle.

## Cardiac Conductive Cells

Sodium (Na ), potassium (K ) and calcium (Ca2 ) ions play critical roles in cardiac conducting cells in the conduction system of the heart. 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 influx of sodium ions that cause the membrane potential to rise slowly and eventually cause spontaneous depolarization. At this point, calcium ion channels open and Ca2 enters the cell, further depolarizing it. As the calcium ion channels then close, the K channels open, 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. Calcium ions play two critical roles in the physiology of cardiac muscle. In addition to depolarization, calcium ions also cause myosin to form cross bridges with the muscle cells that then provide the power stroke of contraction. Medications called calcium channel blockers thus affect both the conduction and contraction roles of calcium in the heart.

The autorhythmicity inherent in cardiac cells keeps the heart beating at a regular pace. However, the heart is regulated by other neural and endocrine controls, and it is sensitive to other factors, including electrolytes. These factors are further discussed in the homeostatic section below.



## Focus on Clinical Practice: The ECG

Surface electrodes placed on specific anatomical sites on the body can record the heart's electrical signals. This tracing of the electrical signal is called an electrocardiogram (ECG), also historically abbreviated EKG. Careful analysis of the ECG reveals a detailed picture of both normal and abnormal heart function and is an indispensable clinical diagnostic tool. A normal ECG tracing is presented in Figure 6.5.



Each component, segment, and the interval is labeled and corresponds to important electrical events.

There are five prominent components of the ECG: the P wave, the Q, R, and S components, and the T wave. The small P wave represents the depolarization of the atria. The large QRS complex represents the depolarization of the ventricles, which requires a much stronger impulse 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 ventricle. Several cardiac disorders can cause abnormal ECG readings called "dysrhythmias," also called "arrhythmias," and there are several types of antidysrhythmic medications used to treat these disorders that will be discussed later in this chapter.

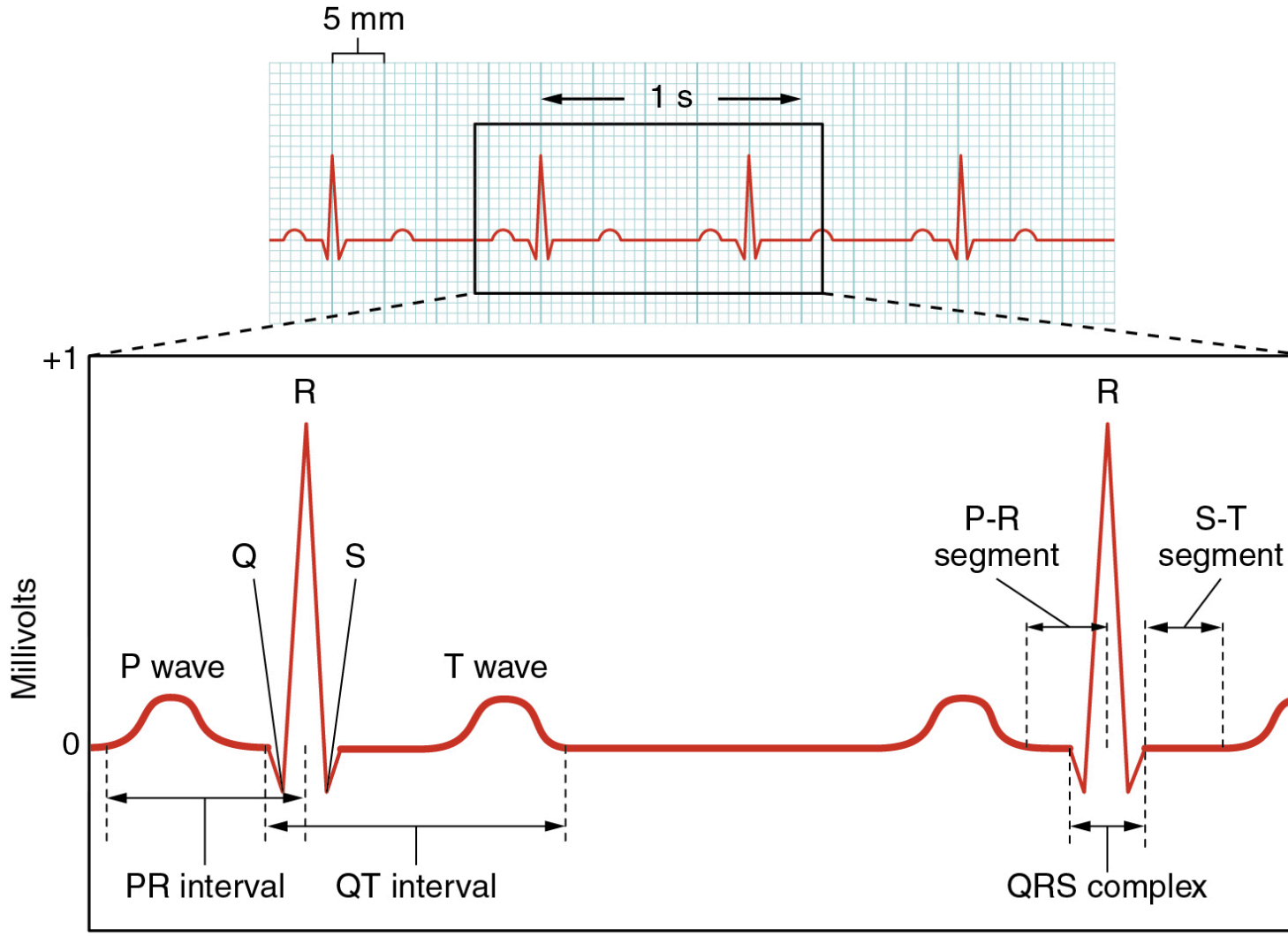



Figure 6.5 Components of an ECG reading

## Cardiac Cycle

The period of time that begins with contraction of the atria and ends with ventricular relaxation is known as the cardiac cycle. 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**.

## 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 into the left atrium from the four pulmonary veins. Contraction of the atria follows depolarization, which is represented by the P wave of the ECG. Just prior to atrial contraction, the ventricles contain approximately 130 mL blood in a resting adult. This volume is known as the end diastolic volume or **preload**. As the atrial muscles contract, pressure rises within the atria and blood is pumped into the ventricles.

Ventricular systole follows the depolarization of the ventricles and is represented by the QRS complex

in the ECG. During the ventricular ejection phase, the contraction of the ventricular muscle causes blood to be pumped out of the heart. This quantity of blood is referred to as **stroke volume (SV)**. Ventricular relaxation, or diastole, follows repolarization of the ventricles and is represented by the T wave of the ECG.



## 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 the heart rate (HR) in beats per minute. It can be represented mathematically by the following equation: CO = HR × SV. Factors influencing CO are summarized in Figure 6.6



and include autonomic innervation by the sympathetic and parasympathetic nervous system, hormones such as epinephrine, preload, contractility, and afterload. Each of these factors is further discussed below.



SV is also used to calculate ejection fraction, which is the portion of the blood that is pumped or ejected from the heart with each contraction.

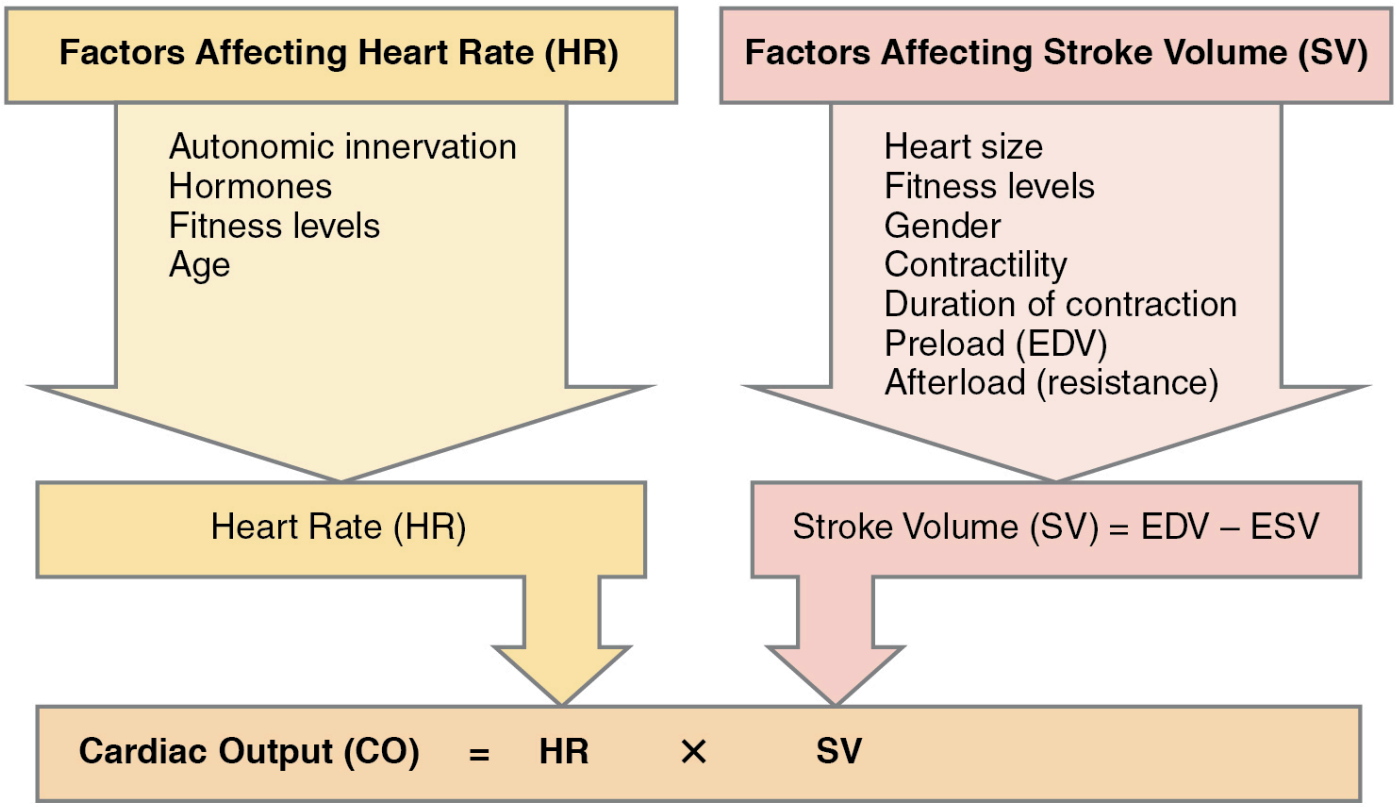



Figure 6.6 Factors affecting cardiac output

## Heart Rate

Heart rate (HR) can vary considerably, not only with exercise and fitness levels, but also with age. Newborn resting HRs may be 120 -160 bpm. HR gradually decreases until young adulthood and then gradually increases again with age. 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.

## Correlation Between Heart Rates and Cardiac Output

Conditions that cause increased HR also trigger an initial increase in SV. However, as the HR rises, there is less time spent in diastole and, consequently, less time for the ventricles to fill with blood. As HR continues to increase, SV gradually decreases due to less filling time. In this manner, tachycardia will eventually cause decreased cardiac output.

## Cardiovascular Centers

Sympathetic stimulation increases the heart rate and contractility, whereas parasympathetic stimulation decreases the heart rate. (See Figure 6.7 for an illustration of the ANS stimulation of the heart.

) Sympathetic stimulation causes the release of the neurotransmitter norepinephrine (NE), which shortens the repolarization period, thus speeding the rate of depolarization and contraction and increasing the HR. It also opens 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 lowering blood pressure. However, an overdose of beta blockers can lead to bradycardia and even stop the heart.

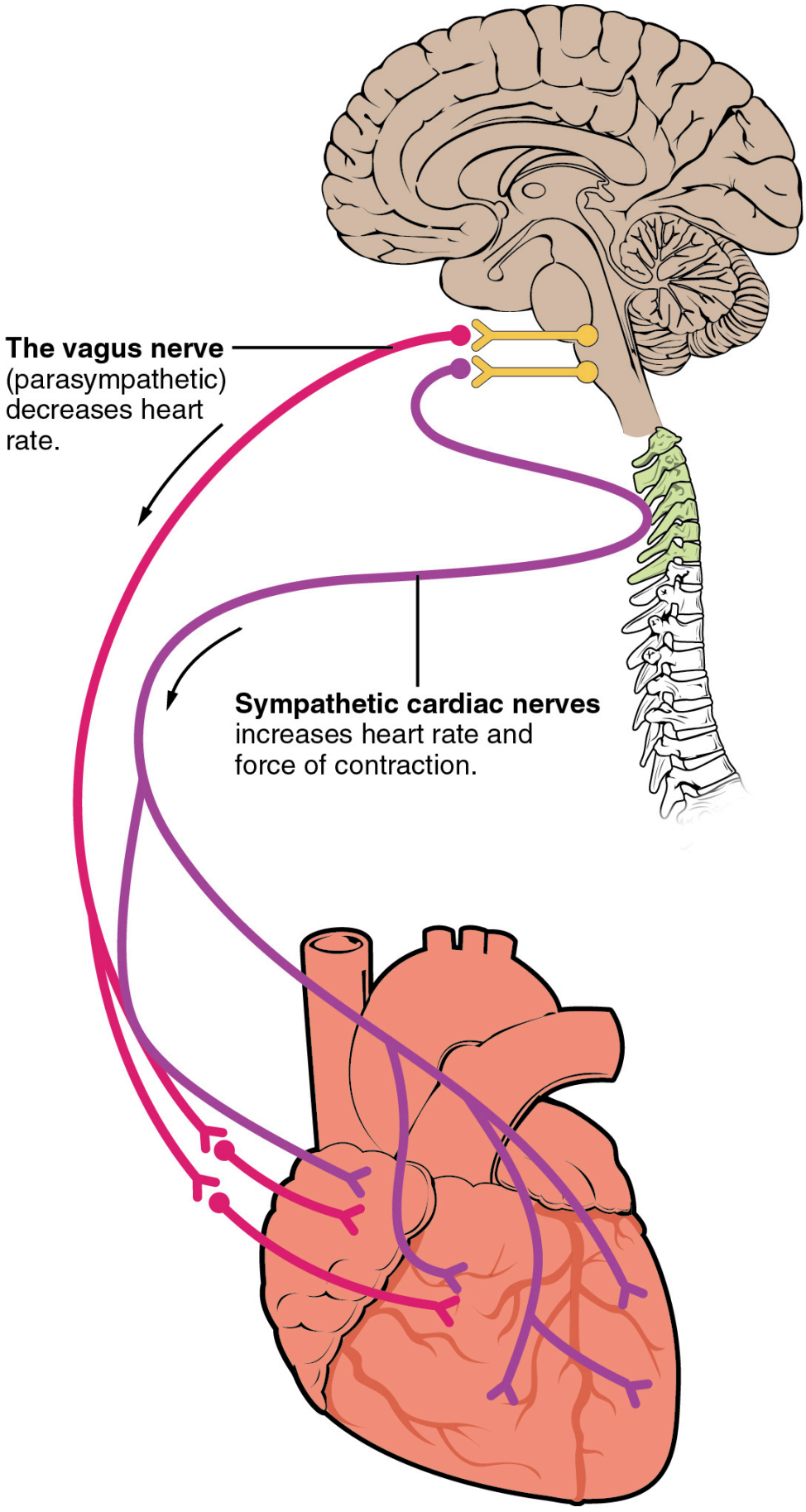

**The vagus nerve** (parasympathetic) decreases heart rate.
**Sympathetic cardiac nerves** increases heart rate and force of contraction.

Figure 6.7 ANS stimulation of the heart includes sympathetic and parasympathetic stimulation

## Stroke Volume

Many of the same factors that regulate HR also impact cardiac function by altering SV. Three primary factors that affect stroke volume are preload, or the stretch on the ventricles prior to contraction; **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. Many cardiovascular medications affect cardiac output by affecting preload, contractility, or afterload.


## Preload

Preload is another way of expressing end diastolic volume (EDV). Therefore, the greater the EDV is, the greater the preload is. One of the primary factors to consider is filling time, the duration of ventricular diastole during which filling occurs. Any sympathetic stimulation to the venous system will also increase venous return to the heart, which contributes to ventricular filling and preload. Medications such as diuretics decrease preload by causing the kidneys to excrete more water, thus decreasing blood volume.

## Contractility

Contractility refers to the force of the contraction of the heart muscle, which controls SV. Factors that increase contractility are described as **positive inotropic factors,** and those that decrease contractility are described as **negative inotropic factors**.

Not surprisingly, sympathetic stimulation is a positive inotrope, whereas parasympathetic stimulation is a negative inotrope. The drug digoxin is used to lower HR and increase the strength of the contraction. It works by inhibiting the activity of an enzyme (ATPase) that controls movement of calcium, sodium, and potassium into heart muscle. Inhibiting ATPase increases calcium in heart muscle and, therefore, increases the force of heart contractions.

Negative inotropic agents include hypoxia, acidosis, hyperkalemia, and a variety of medications such as beta blockers and calcium channel blockers.

## Afterload

Afterload refers to the force 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, which decreases cardiac output. On the other hand, any decrease in resistance reduces the afterload and thus increases cardiac output. Figure 6.8

summarizes the major factors influencing cardiac output. Calcium channel blockers such as amlodipine, verapamil, nifedipine, and diltiazem can be used to reduce afterload and thus increase cardiac output.

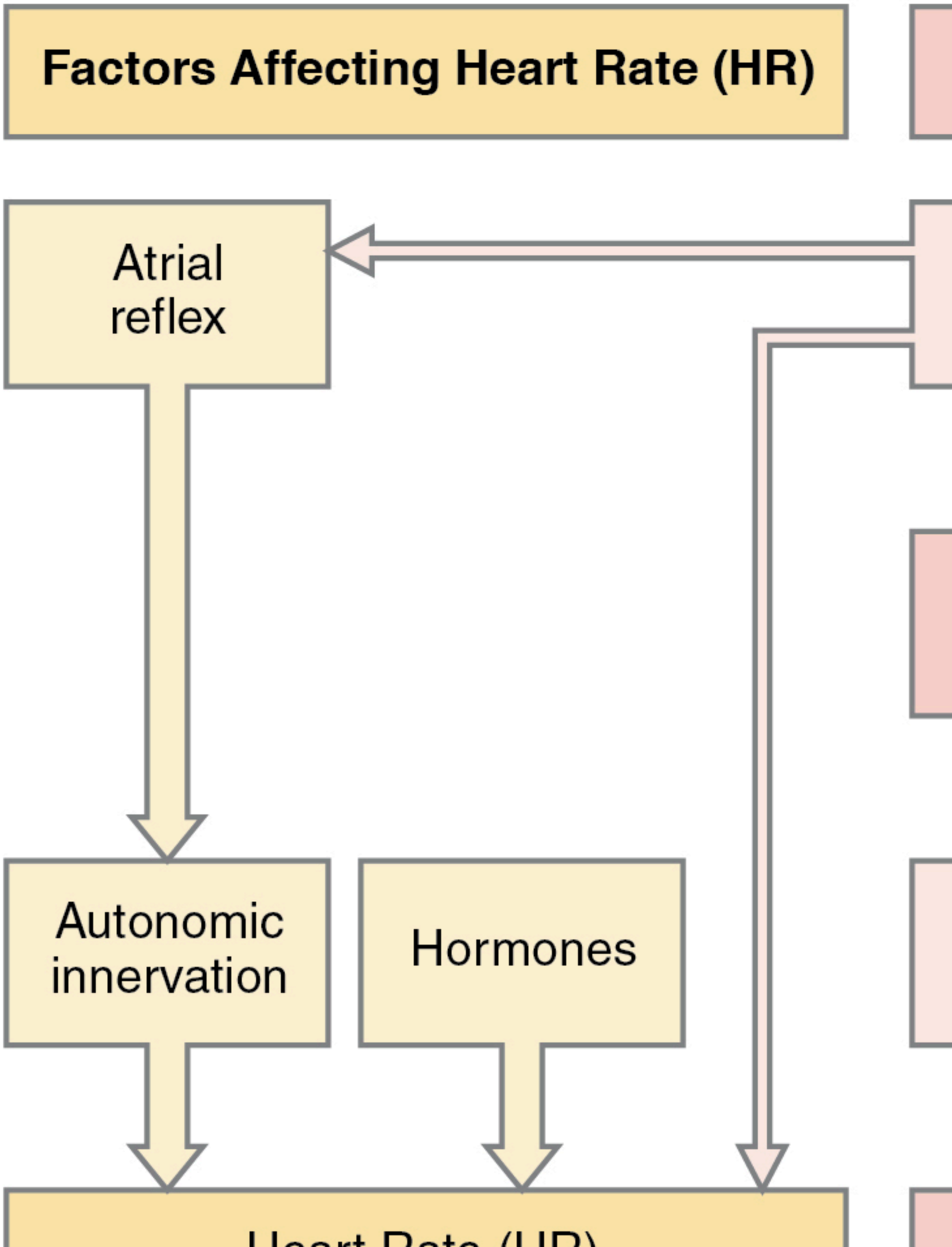

Factors Affecting Heart Rate (HR)
Atrial reflex
Autonomic innervation
Hormones

Figure 6.8 Factors affecting cardiac output

## Systemic Circulation: Blood Vessels

After blood is pumped out of the ventricles, it 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 and eventually into tiny **capillaries** where nutrients and wastes are exchanged at the cellular level. Capillaries then combine with other small blood vessels that carry blood to a **vein**, a larger blood vessel that returns blood to the heart. Compared to arteries, veins are thin-walled, low-pressure vessels. Larger veins are also equipped with valves that promote the unidirectional flow of blood toward the heart and prevent backflow caused by the inherent low blood pressure in veins as well as the pull of gravity.

In addition to their primary function of returning blood to the heart, veins may be considered blood reservoirs because systemic veins contain approximately 64 percent of the blood volume at any given time. 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.

Nitroglycerin is an example of a medication that causes arterial and venous vasodilation. It is used for patients with angina to decrease cardiac workload and increase the amount of oxygen available to the heart. By causing vasodilation of the veins, nitroglycerin decreases the amount of blood returned to the heart, and thus decreases preload. It also reduces afterload by causing vasodilation of the arteries and reducing peripheral vascular resistance.



## Edema

Despite the presence of valves within larger veins, over the course of a day, some blood will inevitably pool in the lower limbs, due to the pull of gravity. Any blood that accumulates in a vein will increase the pressure within it. 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**. See Figure 6.9



for an image of a patient with pitting edema.

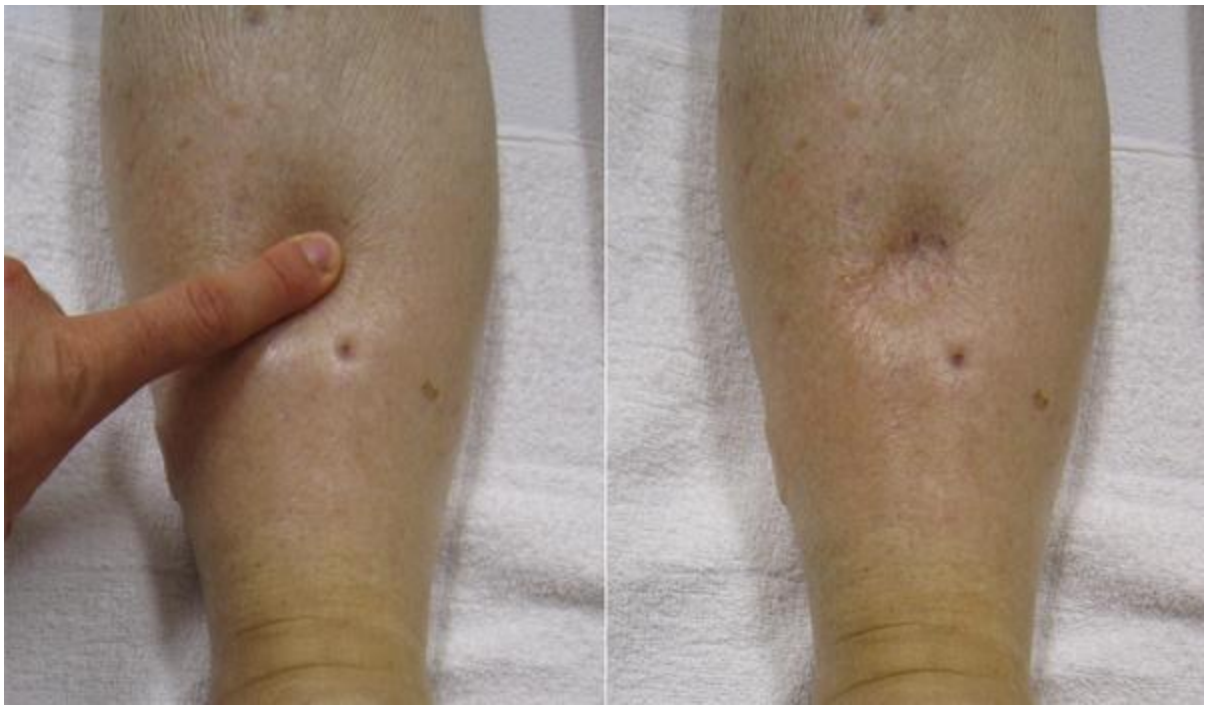


Figure 6.9 Pitting edema

Most people experience a daily accumulation of fluid in their tissues, especially if they spend much of their time 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, and renal failure. Diuretics such as furosemide are used to treat edema by causing the kidneys to eliminate sodium and water.



## Blood Flow and Blood Pressure

Blood flow refers to the movement of blood through a vessel, tissue, or organ. **Blood pressure** is the force exerted by blood on the walls of the blood vessels. In clinical practice, this pressure is measured in mm Hg and is typically obtained using a sphygmomanometer (a blood pressure cuff) on the brachial artery of the arm. When systemic arterial blood pressure is measured, it is recorded as a ratio of two numbers expressed as systolic pressure over diastolic pressure (e.g., 120/80 is a normal adult blood 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.

Three primary variables influence blood flow and blood pressure:

- Cardiac output
- Compliance
- Volume of the blood

Any factor that causes cardiac output to increase will elevate blood pressure and promote blood flow. Conversely, any factor that decreases cardiac output will decrease blood flow and blood pressure. See

the previous section on cardiac output for more information about factors that affect cardiac output.

**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. When vascular disease causes stiffening of arteries, called arteriosclerosis, compliance is reduced and resistance to blood flow is increased. The result is higher blood pressure within the vessel and reduced blood flow. Arteriosclerosis is a common cardiovascular disorder that is a leading cause of hypertension and coronary heart disease because it causes the heart to work harder to generate a pressure great enough to overcome the resistance.

There is a relationship between blood volume, blood pressure, and blood flow. As an example, 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, blood pressure and flow decrease, but when blood volume increases, blood pressure and flow increase.

Low blood volume, called **hypovolemia**, may be caused by bleeding, dehydration, vomiting, severe burns, or by diuretics used to treat hypertension. Treatment typically includes intravenous fluid replacement. Excessive fluid volume, called **hypervolemia**, is caused by retention of water and sodium, as seen in patients with heart failure, liver cirrhosis, and some forms of kidney disease. Treatment may include the use of diuretics that cause the kidneys to eliminate sodium and water.



## Homeostatic Regulation of the Cardiovascular System

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. For example, when an individual is exercising, more blood will be directed to skeletal muscles, the heart, and the lungs. On the other hand, following a meal, more blood is directed to the digestive system. Only the brain receives a constant supply of blood regardless of rest or activity. Three homeostatic mechanisms ensure adequate blood flow and ultimately perfusion of tissues: neural, endocrine, and autoregulatory mechanisms.

## Neural Regulation

The nervous system plays a critical role in the regulation of vascular homeostasis based on baroreceptors and chemoreceptors. Baroreceptors are specialized stretch receptors located within the aorta and carotid arteries that respond to the degree of stretch caused by the presence of blood and then send impulses to the cardiovascular center to regulate blood pressure. In addition to the baroreceptors, chemoreceptors monitor levels of oxygen, carbon dioxide, and hydrogen ions (pH). When the cardiovascular center in the brain receives this input, it triggers a reflex that maintains homeostasis.

## Endocrine Regulation

Endocrine control over the cardiovascular system involves 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 are a part of 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.

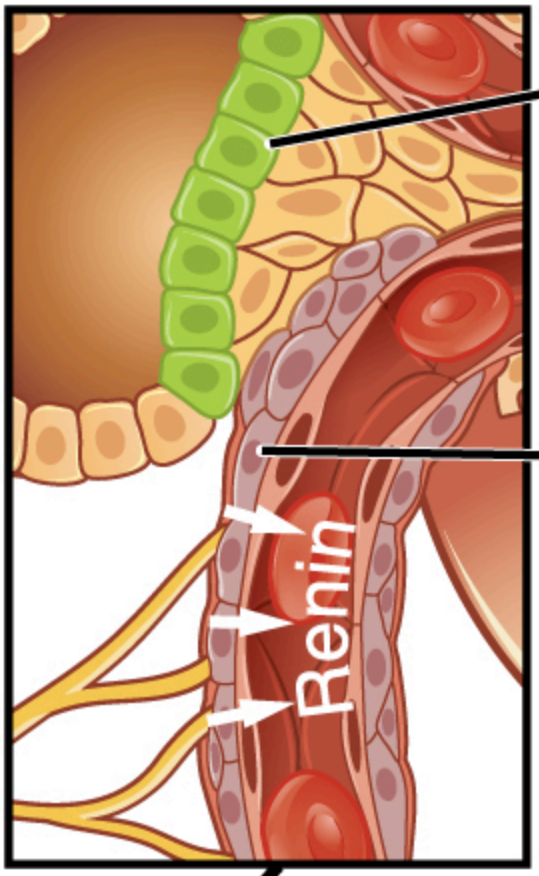
## Antidiuretic Hormone

Antidiuretic hormone (ADH), also known as vasopressin, is secreted by the hypothalamus. 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.

## Renin-Angiotensin-Aldosterone System

The **renin-angiotensin-aldosterone system** (RAAS) has a major effect on the cardiovascular system. Specialized cells in the kidneys respond to decreased blood flow by secreting renin into the blood. Renin converts the plasma protein angiotensinogen 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 called angiotensin-converting enzyme (ACE). Medications called ACE inhibitors such as lisinopril target this step in the RAAS in an effort to decrease blood pressure.

Angiotensin II is a powerful vasoconstrictor that greatly increases blood pressure. It also stimulates the release of ADH and aldosterone, a hormone produced by the adrenal cortex. Aldosterone then increases the reabsorption of sodium into the blood by the kidneys. Because water follows sodium, there is an increase in the reabsorption of water, which increases blood volume and blood pressure. See Figure 6.10 for an illustration of the renin-angiotensin-aldosterone system and Figure 6.11 for a summary of the effect of hormones involved in renal control of blood pressure.




Macula densa senses low fluid flow or low $Na^+$ concentration

Juxtaglomerular cells secrete renin

Kidney releases enzyme renin into blood

Enzyme reaction → Angiotensin I → Enzy react

Liver releases angiotensinogen into blood

Figure 6.10 The renin-angiotensin-aldosterone system

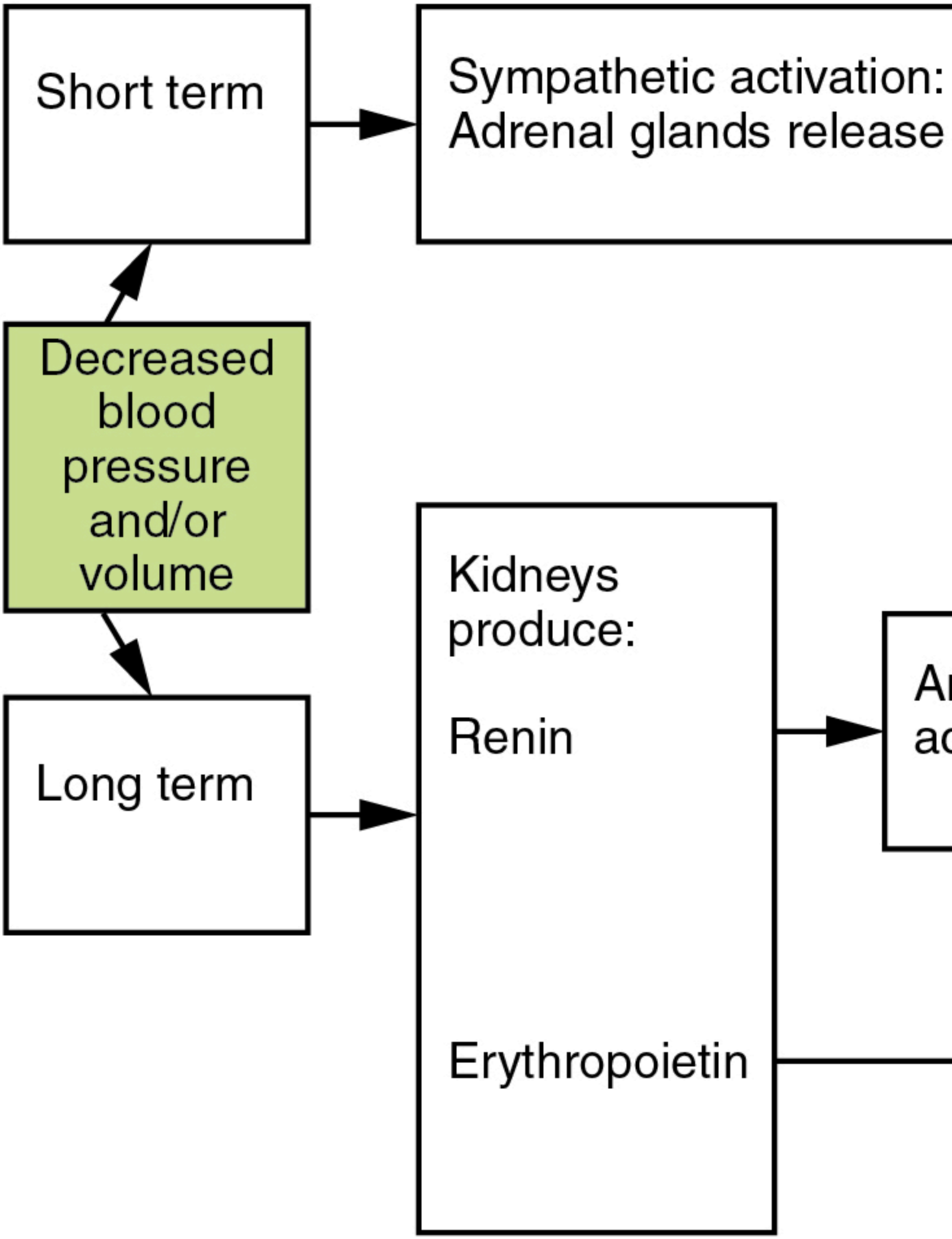

Short term
Sympathetic activation:
Adrenal glands release
Decreased blood pressure and/or volume
Long term
Kidneys produce:
Renin
Erythropoietin

Figure 6.11 Hormones involved in renal control of blood pressure

## Autoregulation of Perfusion

Local, self-regulatory mechanisms allow each region of tissue to adjust its blood flow—and thus its perfusion. These mechanisms are affected by sympathetic and parasympathetic stimulation, as well as endocrine factors. See Figure 6.12 for a summary of these factors and their effects.



| Control | Factor | Vasoconstriction | Vasodilation |
|---|---|---|---|
| **Neural** | Sympathetic stimulation | Arterioles within integument abdominal viscera and mucosa membrane; skeletal muscles (at high levels); varied in veins and venules | Arterioles within heart; skeletal muscles at low to moderate levels |
| | Parasympathetic | No known innervation for most | Arterioles in external genitalia; no known innervation for most other arterioles or veins |
| **Endocrine** | Epinephrine | Similar to sympathetic stimulation for extended flight-or-fight responses; at high levels, binds to specialized alpha (α) receptors | Similar to sympathetic stimulation for extended fight-or-flight responses; at low to moderate levels, binds to specialized beta (β) receptors |
| | Norepinephrine | Similar to epinephrine | Similar to epinephrine |
| | Angiotensin II | Powerful generalized vasoconstrictor; also stimulates release of aldosterone and ADH | n/a |
| | ANH (peptide) | n/a | Powerful generalized vasodilator; also promotes loss of fluid volume from kidneys, hence reducing blood volume, pressure, and flow |
| | ADH | Moderately strong generalized vasoconstrictor; also causes body to retain more fluid via kidneys, increasing blood volume and pressure | n/a |
| **Other factors** | Decreasing levels of oxygen | n/a | Vasodilation, also opens precapillary sphincters |
| | Decreasing pH | n/a | Vasodilation, also opens precapillary sphincters |

| | | |
|---|---|---|
| Increasing levels of carbon dioxide | n/a | Vasodilation, also opens precapillary sphincters |
| Increasing levels of potassium ion | n/a | Vasodilation, also opens precapillary sphincters |
| Increasing levels of prostaglandins | Vasoconstriction, closes precapillary sphincters | Vasodilation, opens precapillary sphincters |
| Increasing levels of adenosine | n/a | Vasodilation |
| Increasing levels of lactic acid and other metabolites | n/a | Vasodilation, also opens precapillary sphincters |
| Increasing levels of endothelins | Vasoconstriction | n/a |
| Increasing levels of platelet secretions | Vasoconstriction | n/a |
| Increasing hypothermia | n/a | Vasodilation |
| Stretching of vascular wall (myogenic) | Vasoconstriction | n/a |
| Increasing levels of histamines from basophils and mast cells | n/a | Vasodilation |

*Figure 6.12 The effects of nervous, endocrine, and local controls on the vasoconstriction and vasodilation of arterioles*

## Kidney Function Review

As discussed earlier, the kidney helps to regulate blood pressure, along with the heart and blood vessels, primarily through the Renin-Angiotensin-Aldosterone System (RAAS). In addition to cardiovascular medications affecting the RAAS system, there are also medications called diuretics that reduce blood volume by working at the nephron level. This section will review the basic concepts of kidney function at the nephron level to promote understanding of the mechanism of action of various cardiovascular medications.

The kidney receives blood from the circulatory system via the renal artery. The renal artery branches into smaller and smaller arterioles until the smallest arteriole, the afferent arteriole, services the nephrons. There are about 1.3 million nephrons in each kidney. Nephrons filter the blood and modify it

into urine by accomplishing three principal functions—filtration, reabsorption, and secretion. They also have additional secondary functions in regulating blood pressure (via the production of renin) and producing red blood cells (via the hormone erythropoietin).


The initial filtering of the blood takes place in the glomerulus, a cluster of capillaries surrounded by the glomerular capsule. The rate at which this filtering occurs is called the glomerular filtration rate (GFR) and is used to gauge how well the kidneys are functioning. The rate at which blood flows into the glomerulus is controlled by afferent arterioles and the blood vessels flowing out of the glomerulus. These blood vessels are called called efferent arterioles.
McCuistion, L., Vuljoin-DiMaggio, K., Winton, M, & Yeager, J. (2018). *Pharmacology: A patient-centered nursing process approach.* pp. 443-454. Elsevier.
See Figure 6.13

for an illustration of blood flow through the kidney and nephrons.

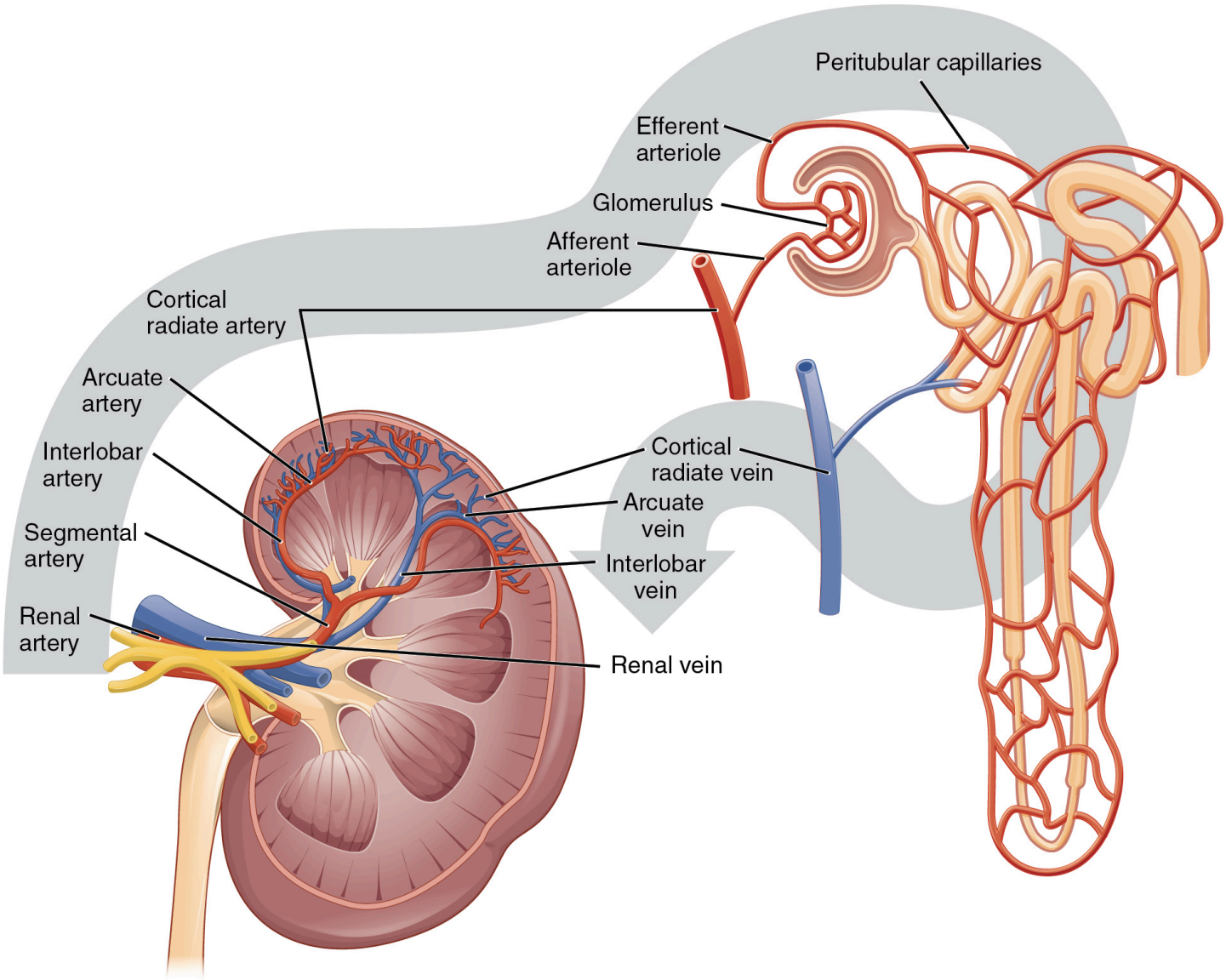


Figure 6.13 Blood flow through the kidney and nephrons

Lying just outside the glomerulus is the juxtaglomerular apparatus (JGA). One function of the JGA is to regulate renin release as part of the RAAS system discussed earlier in this chapter.

See Figure 6.14

for an illustration of nephron structure. From the glomerulus (1), the proximal tubule (2) returns 60-70% of the sodium and water back into the bloodstream. From the proximal tubule, the filtrate flows into the descending loop of Henle (3) and then the ascending loop of Henle (4). Another 20-25% of sodium is reabsorbed in the ascending loop of Henle, and this is the site of action of loop diuretics. Filtrate then enters the distal tubule (5), where sodium is actively filtered in exchange for potassium or hydrogen ions, a process regulated by the hormone aldosterone. This is the site of action for thiazide diuretics. The collecting duct (6) is the final pathway; this is where antidiuretic hormone (ADH) acts to increase the absorption of water back into the bloodstream, thereby preventing it from being lost in the urine.
McCuistion, L., Vuljoin-DiMaggio, K., Winton, M, & Yeager, J. (2018). *Pharmacology: A patient-centered nursing process approach.* pp. 443-454. Elsevier.

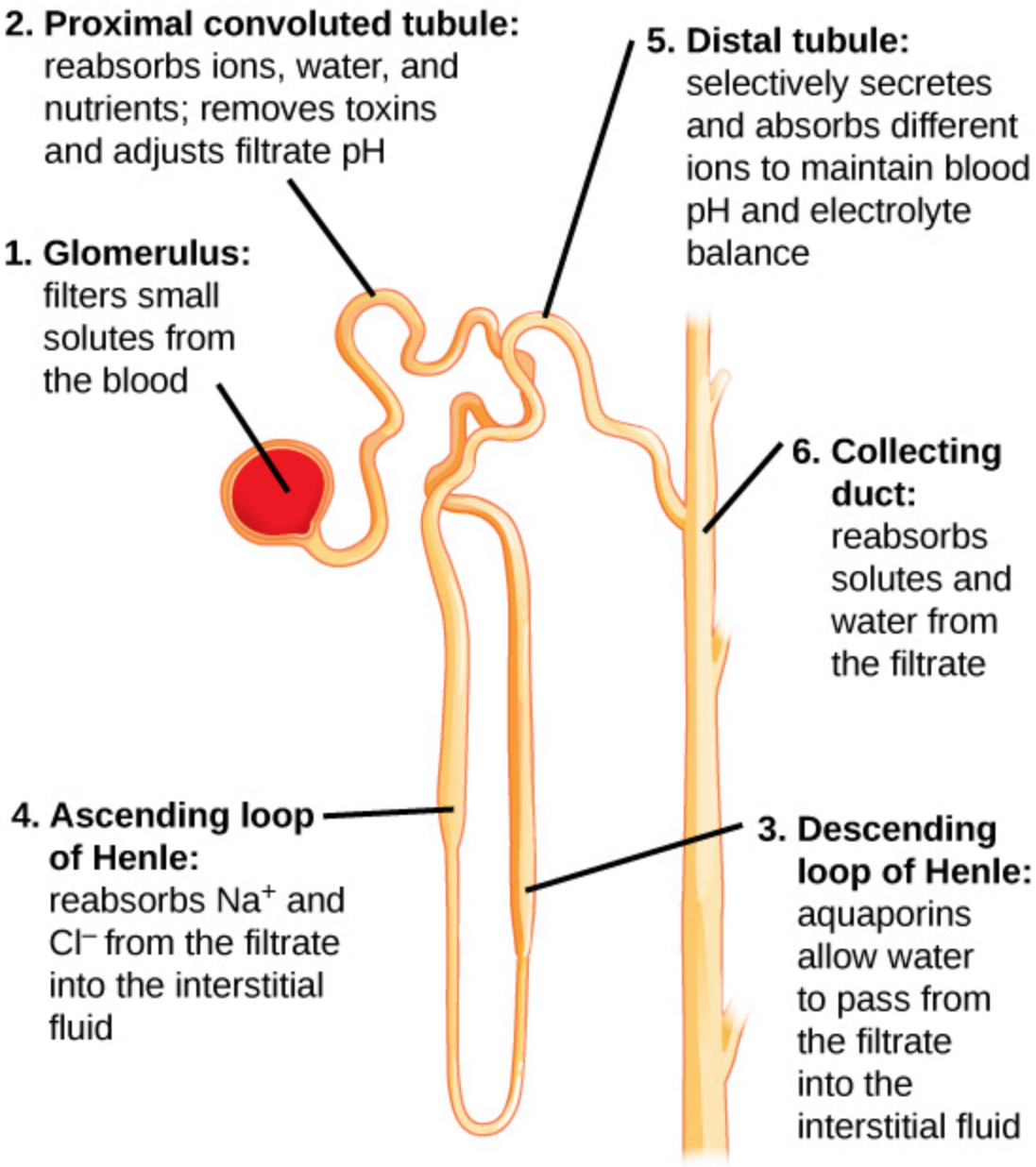



Figure 6.14 Nephron structure

## Elimination of Drugs and Hormones

Water-soluble drugs may be excreted in the urine and are influenced by one or all of the following processes: glomerular filtration, tubular secretion, or tubular reabsorption. Drugs that are structurally small can be filtered by the glomerulus with the filtrate. However, large drug molecules such as heparin or those that are bound to plasma proteins cannot be filtered and are not readily eliminated. Some drugs can be eliminated by carrier proteins that enable secretion of the drug into the tubule (such as dopamine or histamine).

## Blood and Coagulation

Now that we have reviewed the functions of the heart, blood vessels, and kidneys, we will review coagulation. As we discussed, the primary function of blood as it moves through the blood vessels in the body is to deliver oxygen and nutrients and remove wastes as it is filtered by the kidney, but that is only the beginning of the story. Cellular elements of blood include red blood cells (RBCs), white blood cells (WBCs), and platelets, and each element has its own function. Red blood cells carry oxygen; white blood cells assist with the immune response; and platelets are key players in **hemostasis**, the process by which the body seals a small ruptured blood vessel and prevents further loss of blood. There are three steps to the hemostasis 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). Each of these steps will be further discussed below.



## 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. 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

A platelet plug can temporarily seal a small opening in a blood vessel, thus buying the body more time while more sophisticated and durable repairs are being made.

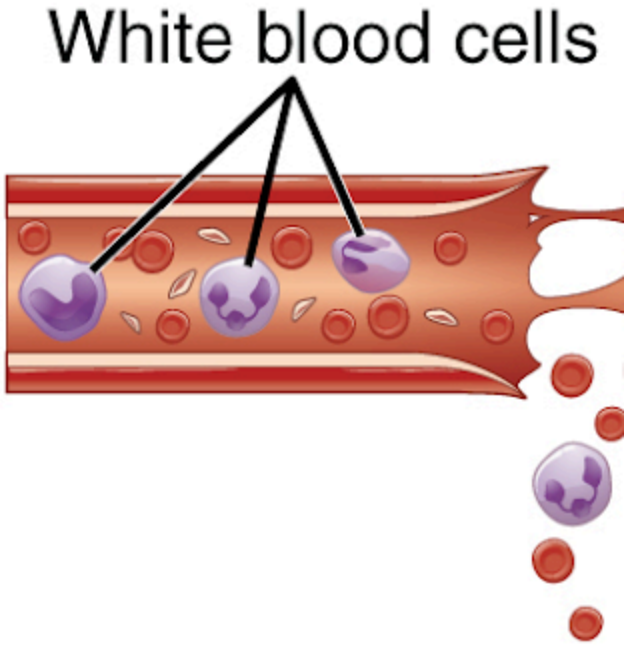


## Coagulation

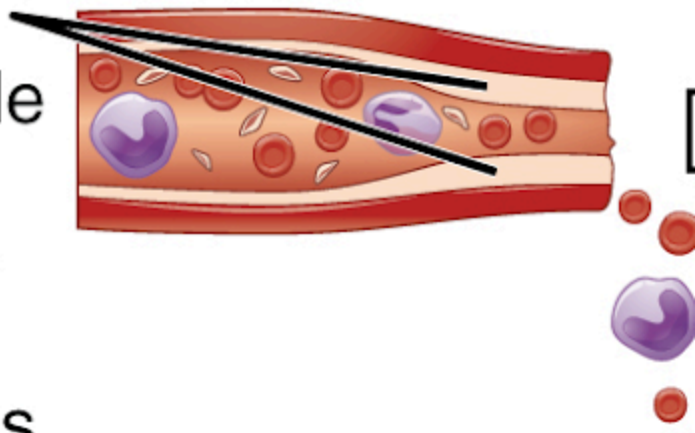
The more sophisticated and more durable repairs are called **coagulation**, or the formation of a blood clot. The process is sometimes characterized as a cascade because one event prompts the next as in a multi-level waterfall. The result is the production of a gelatinous but robust clot made up of a mesh of

fibrin in which platelets and blood cells are trapped. Figure 6.15
"1909 Blood Clotting.jpg" by OpenStax College is licensed under CC BY 4.0 Access for free at https://openstax.org/books/anatomy-and-physiology/pages/18-5-hemostasis
summarizes the three steps of hemostasis when an injury to a blood vessel occurs. 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. The synthesis of fibrin in blood clots involves either an intrinsic pathway or an extrinsic pathway, both of which lead to a common pathway creating a clot.

1 **Injury.** A blood vessel is severed. Blood and blood components (e.g., erythrocytes, white blood cells, etc.) are leaking out of the breaks.




2 **Vascular spasm.** The smooth muscle in the vessel wall contracts near the injury point, reducing blood loss.



3 **Platelet plug formation.** Platelets are
chemicals released from the injury site
with underlying collagen. The platelets

Figure 6.15 The steps of hemostasis

## 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. The events in the extrinsic pathway are completed in a matter of seconds.

## Intrinsic Pathway

The intrinsic 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 or resulting from internal factors such as arterial disease. 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, where fibrin is produced to seal off the vessel. Once Factor X has been activated by either the intrinsic or extrinsic pathway, Factor II, the inactive enzyme prothrombin, is converted into the active enzyme thrombin. 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 on 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. 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, antithrombin inactivates Factor X and opposes the conversion of prothrombin (Factor II) to thrombin in the common pathway. Basophils release heparin, a short-acting anticoagulant that also opposes prothrombin. A pharmaceutical form of heparin is often administered therapeutically to prevent or treat blood clots.

A **thrombus** 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. In a small vessel, it may actually totally block the flow of blood and is termed an occlusive thrombus.

There are several medications that impact the coagulation cascade. For example, aspirin (acetylsalicylic acid) is very effective at inhibiting the aggregation of platelets. Patients at risk for cardiovascular disease often take a low dose of aspirin on a daily basis as a preventive measure. It is also routinely administered during a heart attack or stroke to reduce the formation of the platelet plug. Anticoagulant medications such as warfarin and heparin prevent the formation of clots by affecting the intrinsic or extrinsic pathways. Another class of drugs known as thrombolytic agents are used to dissolve an abnormal clot. If a thrombolytic agent is administered to a patient within a few hours following a thrombotic stroke or myocardial infarction, the patient's prognosis improves significantly. Tissue plasminogen activator (TPA) is an example of a medication that is released naturally by endothelial cells but is also used in clinical medicine to break down a clot.



## Video Review of Basic Concepts

For additional video review of the basic anatomy and physiology concepts of the cardiovascular and renal system, see the supplementary videos below.

## Blood Vessels



One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-1

## Muscle Contraction



One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-2

## Fluids and Electrolytes: Potassium and Aldosterone



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## Fluid and Electrolytes: Sodium

permission. https://youtu.be/ar-WrfC7SJs

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-4

## Anatomy of the Heart

Forciea, B. (2015, May 20). Anatomy of the Heart (v2.0). [Video]. YouTube. All rights reserved. Video used with permission. https://youtu.be/d8RSvcc8koo

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-5

## The Blood

Forciea, B. (2015, May 19). Anatomy and Physiology: The Blood. [Video]. YouTube. All rights reserved. Video used with permission. https://youtu.be/bjfcOSoDSzg

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-6

## Anatomy of Urinary System

Forciea, B. (2015, May 13). Urinary System Anatomy (v2.0) [Video]. YouTube. All rights reserved. Video used with permission. https://youtu.be/2Wd45Zmq_Ck

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-7

## Renin-Angiotensin System

Forciea, B. (2015, May 13). Renin-Angiotensin System for Anatomy and Physiology (v2.0) [Video]. YouTube. All rights reserved. Video used with permission. https://youtu.be/iin4lbAKv7Q

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-8

## Introduction to ECG

Forciea, B. (2015, May 12). Introduction to the Electrocardiogram (ECG) V2.0. [Video]. YouTube. All rights reserved. Video used with permission. https://youtu.be/mAN0GK7O9yU

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-9

## Circulatory System Anatomy

Forciea, B. (2015, May 12). Circulatory System Anatomy (v2.0). [Video]. YouTube. All rights reserved. Video used with permission. https://youtu.be/nBSHhkOEKHA

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://wtcs.pressbooks.pub/pharmacology/?p=2635#oembed-10

# 6.3 Common Cardiac Disorders

Open Resources for Nursing (Open RN)

Now that we have reviewed the basic anatomy and physiology concepts of the cardiovascular and renal system, let's discuss some common cardiac disorders.

## Hyperlipidemia

Cholesterol is a fat (also called a lipid) that your body needs to work properly. However, too much bad cholesterol can increase the risk for heart disease, stroke, and peripheral vascular disease. The medical term for high blood cholesterol is **hyperlipidemia**. There are many types of cholesterol (see Figure 6.16 for basic types of cholesterol.
"máu nhiễm mỡ - cholesterol" by LÊ VĂN THẢO is licensed under CC BY-SA 2.0
)

- **Total cholesterol**: All the cholesterols combined
- **High density lipoprotein (HDL) cholesterol:** Often called "good" cholesterol because it promotes the excretion of cholesterol. Exercise helps to increase HDL and remove cholesterol from the bloodstream
- **Low density lipoprotein (LDL) cholesterol:** Often called "bad" cholesterol because it stores cholesterol in the bloodstream, which contributes to atherosclerosis

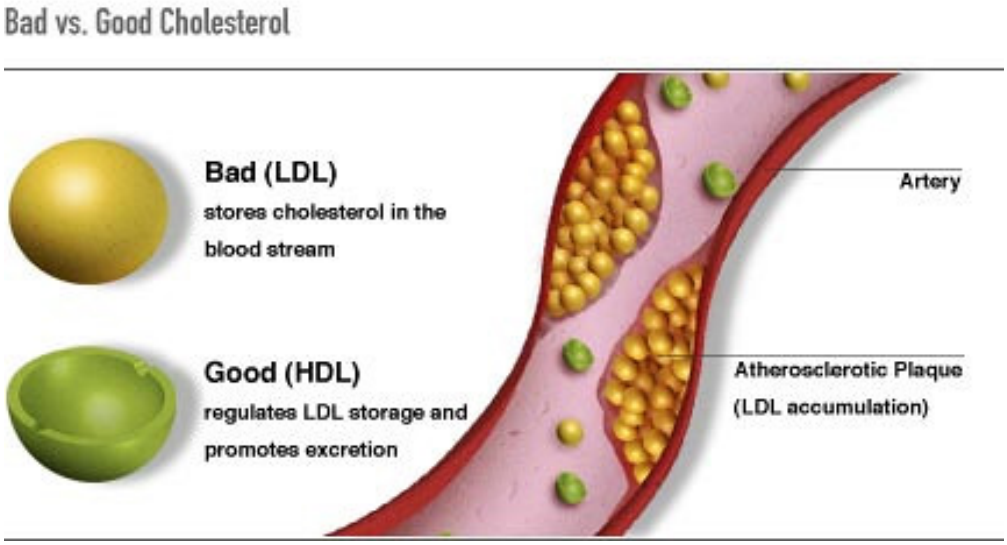



Figure 6.16 A comparison of LDL (bad cholesterol) and HDL (good cholesterol)

For many people, abnormal cholesterol levels are partly due to lifestyle choices, including a diet that is high in fat, being overweight, or lack of exercise. However, disorders can also be passed down through families that lead to abnormal cholesterol and triglyceride levels.
A.D.A.M. Medical Encyclopedia [Internet]. Atlanta (GA): A.D.A.M., Inc.; c2019. High blood cholesterol levels; [reviewed

2018 February 22; updated 2018 March 28; cited 2019 November 29]. https://medlineplus.gov/ency/article/000403.htm.
In addition to lifestyle modifications such as a low-fat diet and exercise, hyperlipidemia is treated with antilipidemic medication such as atorvastatin (Lipitor) to help prevent long-term complications.

## Hypertension

Chronically elevated blood pressure is known clinically as hypertension. High blood pressure is treated with lifestyle changes and medication. New American Heart Association guidelines state that hypertension should be treated at 130/80 mm Hg rather than the previous standard of 140/90.
Whelton, P.K., Carey R.M., Aronow W.S., et. al. (2018). 2017 Guideline for high blood pressure in adults. *Journal of the American College of Cardiology, 71*. https://www.acc.org/latest-in-cardiology/ten-points-to-remember/2017/11/09/11/41/2017-guideline-for-high-blood-pressure-in-adults.
See Figure 6.17
"Monthly check up." by Bryan Mason is licensed under CC BY 2.0
for an image of a health care professional obtaining an accurate blood pressure reading that will be used to determine a treatment plan for the patient.

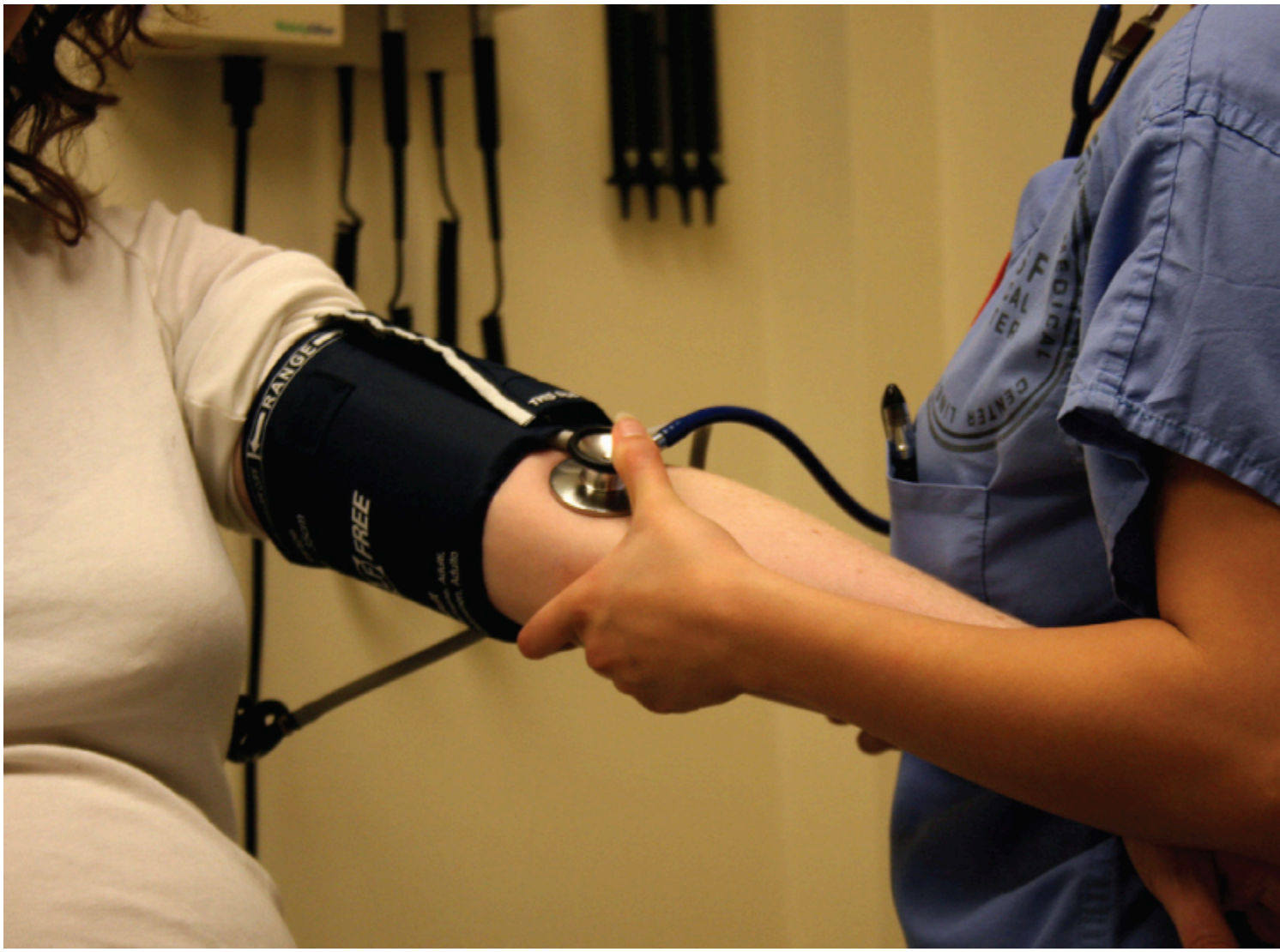


Figure 6.17 It is critical to obtain an accurate blood pressure that will be used for the development of a treatment pla
hypertension

About 68 million Americans currently suffer from hypertension. Unfortunately, hypertension is often a silent disorder, meaning no symptoms occur until complications happen, so 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), myocardial infarction, chronic kidney disease, or heart failure.


Many cardiovascular medications are commonly used to treat hypertension such as diuretics, ACE inhibitors, beta blockers, and calcium channel blockers.

## Thrombi and Emboli

Thrombi are most commonly caused by vessel damage to the endothelial lining, which activates the clotting mechanism. A thrombus can seriously impede blood flow to tissue or organs. Deep vein thrombosis (DVT) can occur when blood in the veins, particularly in the legs, remains stationary for long periods, such as during and after surgery. See Figure 6.18

for an image of a patient experiencing typical symptoms of a DVT, including unilateral edema and redness.

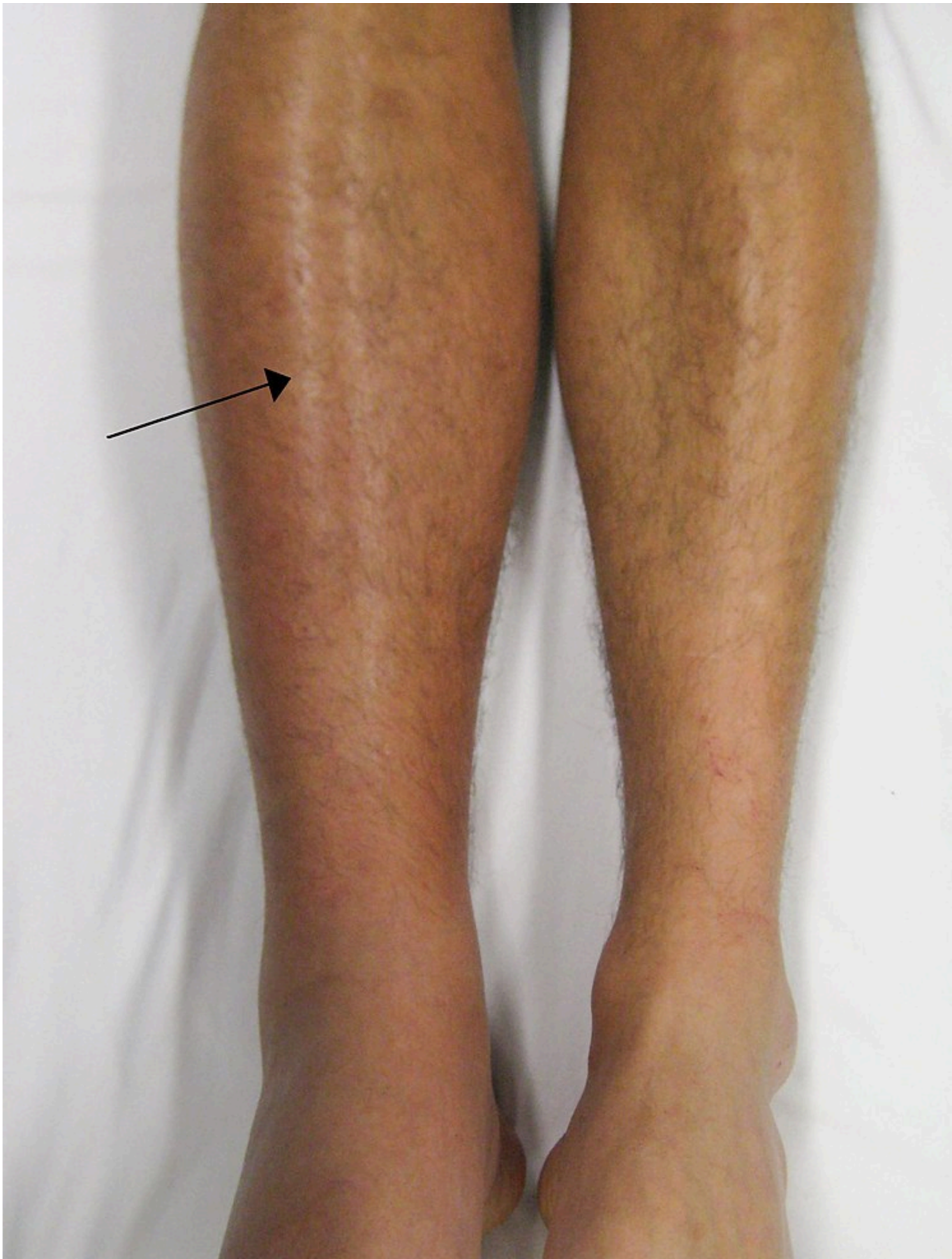


Figure 6.18 Typical signs of a DVT include unilateral edema and redness

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 cause a heart attack, a cerebrovascular accident (CVA) or otherwise known as a stroke, or a pulmonary embolism. These are medical emergencies.

Medications such as aspirin and warfarin are used to prevent the formation of clots in people who are at risk. Heparin is a medication that can be used to prevent or treat clots, and tPA is used to dissolve severe clots causing ischemia in the brain, heart, or lungs.



## Atherosclerosis

**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. Injured artery walls causes inflammation. As inflammation spreads into the artery wall, it weakens and scars it, leaving it stiff. Circulating triglycerides and cholesterol can seep between the damaged lining cells and become trapped within the artery wall, where they are 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, describes the plaque deposits. See Figure 6.19



for an illustration of atherosclerosis.

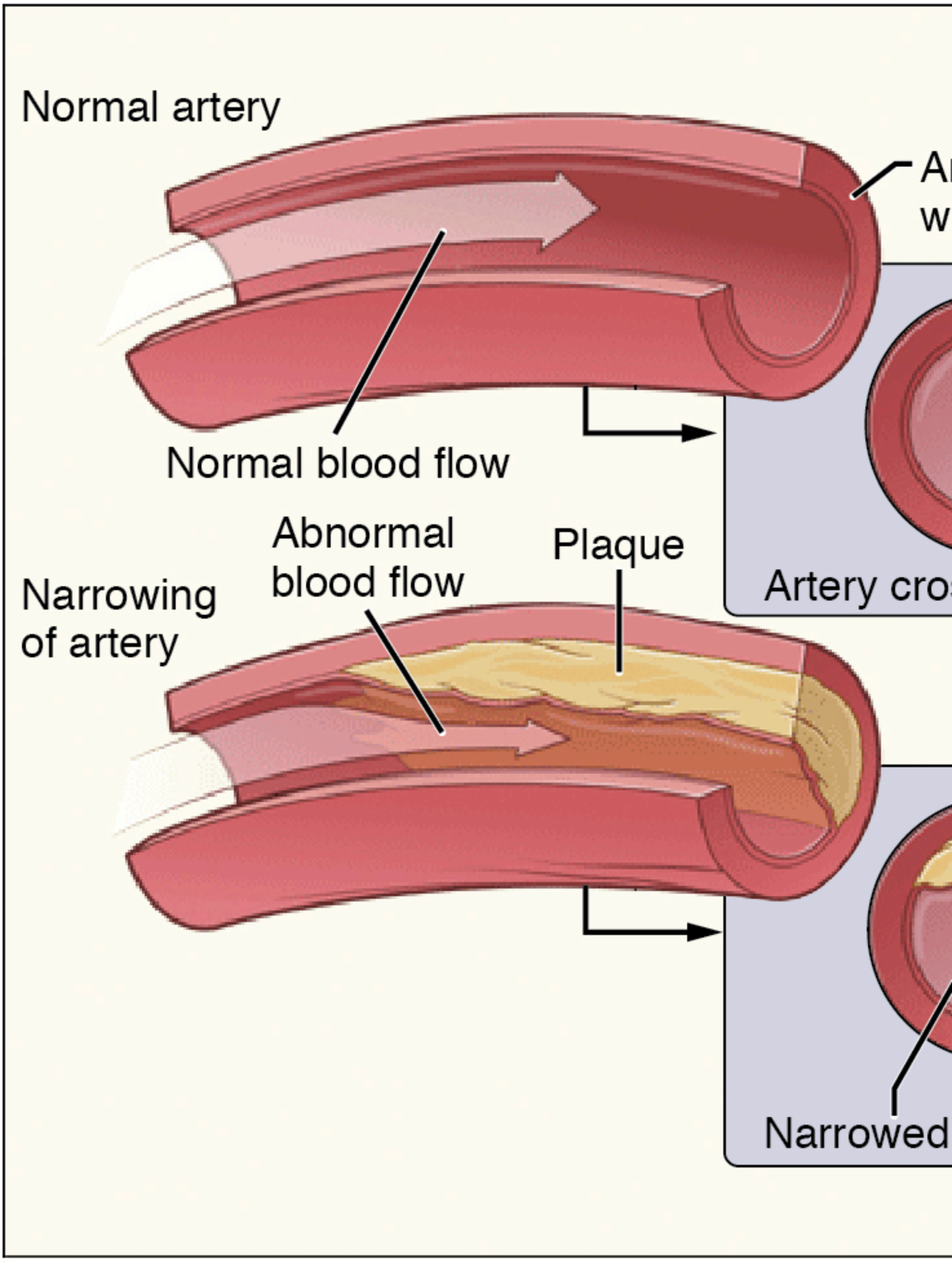

Normal artery
Normal blood flow
Narrowing of artery
Abnormal blood flow
Plaque
Narrowed

Figure 6.19 Atherosclerosis

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 also break off and travel through the bloodstream as an **embolus** until it blocks a more distant, smaller artery.

Even without total blockage, narrowed vessels lead to **ischemia** (reduced blood flow to the tissue region "downstream" of the narrowed vessel). Ischemia can lead to hypoxia (decreased supply of oxygen to the tissues), causing a myocardial infarction or cerebrovascular accident.

Treatment of atherosclerosis includes lifestyle changes, such as weight loss, smoking cessation, regular exercise, and adoption of a diet low in sodium and saturated fats. Antilipemic medications such as atorvastatin are prescribed to reduce cholesterol and help prevent atherosclerosis.

## Coronary Artery Disease

Coronary artery disease is the leading cause of death worldwide. It occurs when atherosclerosis within the walls of the coronary arteries obstructs blood flow. As the coronary blood vessels become blocked with plaque, the flow of blood to the tissues is restricted, causing the cardiac cells to receive insufficient amounts of oxygen, which can cause pain called angina. Figure 6.20

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, but others, especially women, may remain asymptomatic or have alternative symptoms of neck, jaw, shoulder, upper back, or abdominal pain. If untreated, coronary artery disease can lead to a **myocardial infarction** (heart attack). Risk factors include smoking, family history, hypertension, obesity, diabetes, lack of exercise, stress, and hyperlipidemia. Treatments may include medication, changes to diet and exercise, a coronary angioplasty with a balloon catheter, insertion of a stent, or coronary bypass procedure.

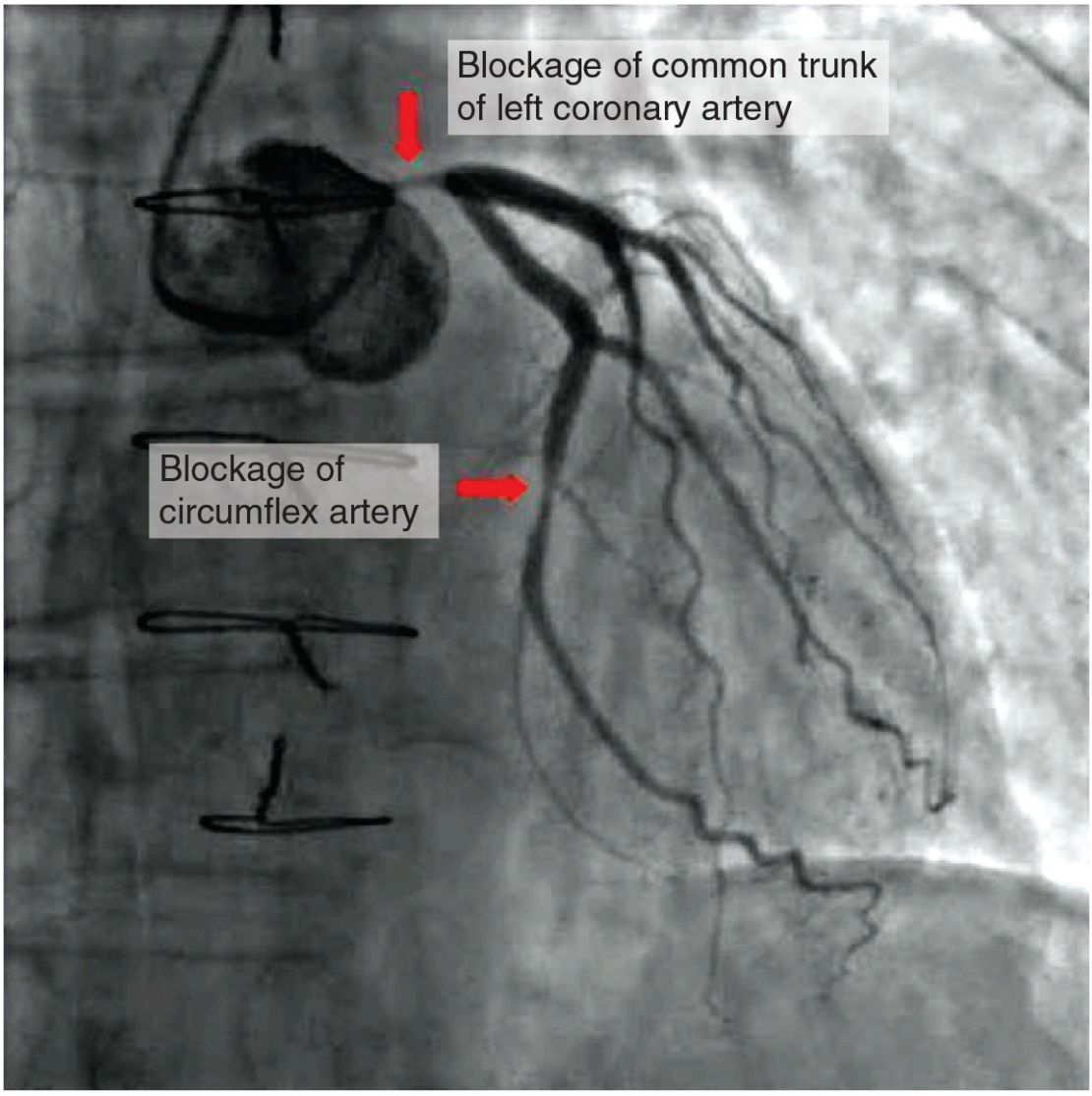



Figure 6.20 Image of blocked coronary arteries highlighted by the injection of dye during a coronary angiogram

## Myocardial Infarction

Myocardial infarction (MI) is the medical term for what is commonly referred to as a "heart attack." It results from a lack of blood flow and oxygen 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 and becomes a thrombus or when a portion of an unstable atherosclerotic plaque travels through the coronary arterial system and lodges in one of the smaller vessels.

In the case of acute MI, there is often sudden pain beneath the sternum (retrosternal pain) called angina, often radiating down the left arm in male patients, but not as commonly in female patients (see Figure 6.21).



In addition, patients typically present with difficulty breathing and shortness of breath (dyspnea), irregular heartbeat (palpitations), 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 accurate diagnosis is critical for survival.

An MI can be confirmed by examining the patient's ECG, which frequently reveals alterations in the ST and Q components. Immediate treatments for MI are required and include administering supplemental oxygen, aspirin, and nitroglycerin. Longer-term treatments may include injections of thrombolytic agents such as tPA that dissolve the clot, along with the anticoagulant heparin, a balloon angioplasty with stents to open blocked vessels, or bypass surgery to allow blood to pass around the site of blockage.

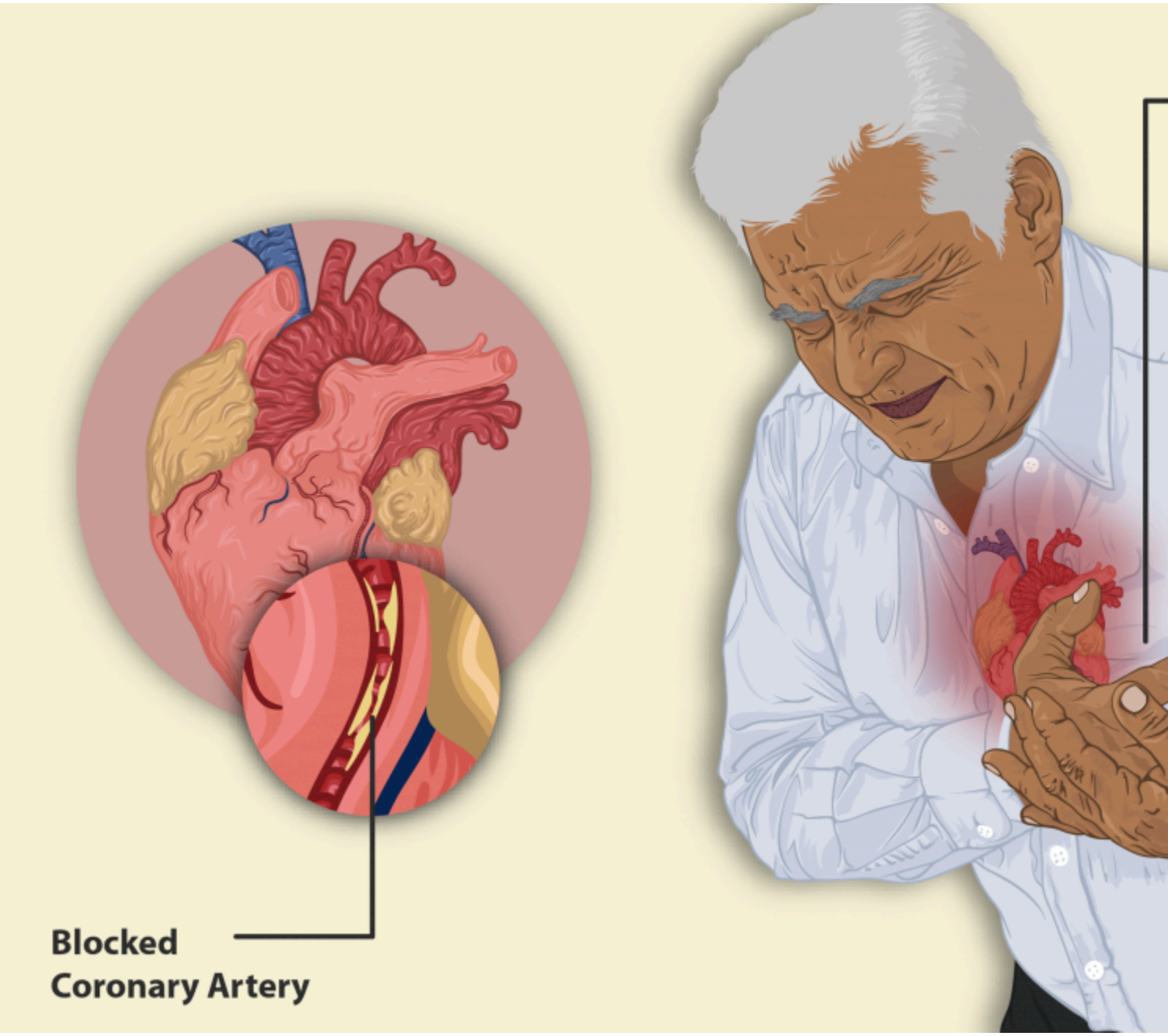




Figure 6.21 Male patients often describe chest pain associated with MI "like something is sitting on my chest," but f
GI upset

## Cerebrovascular Accident (CVA)

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. However, blood flow may become obstructed due to atherosclerosis or an emboli that has traveled from elsewhere in the blood. For example, an arrhythmia called atrial fibrillation can cause clots to form in the heart that then move to the brain. 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. Loss of blood flow for longer periods produce irreversible brain damage or a stroke, also called a **cerebrovascular accident (CVA)**.



There are two types of cerebrovascular accidents: ischemia and hemorrhagic. Ischemic strokes are caused by atherosclerosis or a blood clot that blocks the flow of blood to the brain (see Figure 6.22).



Eighty percent of strokes are ischemic. Hemorrhagic strokes are caused by a blood vessel that ruptures and bleeds into the brain. Risk factors for a stroke include smoking, high blood pressure, and cardiac arrhythmias. Treatment of a stroke depends on the cause.

Anderson, P. & Townsend, T. (2015) Preventing high-alert medication errors in hospital patients. *Nurse Today, 10*(5). https://www.americannursetoday.com/wp-content/uploads/2015/05/ant5-CE-421.pdf

Ischemic strokes are treated with thrombolytic medication such as tPA to dissolve the clot, whereas hemorrhagic strokes often require surgery to stop the bleeding.

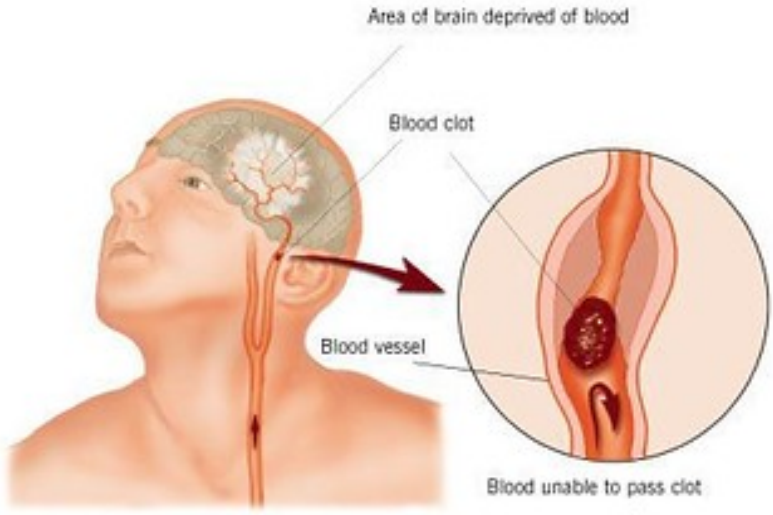



Figure 6.22 Ischemic Stroke

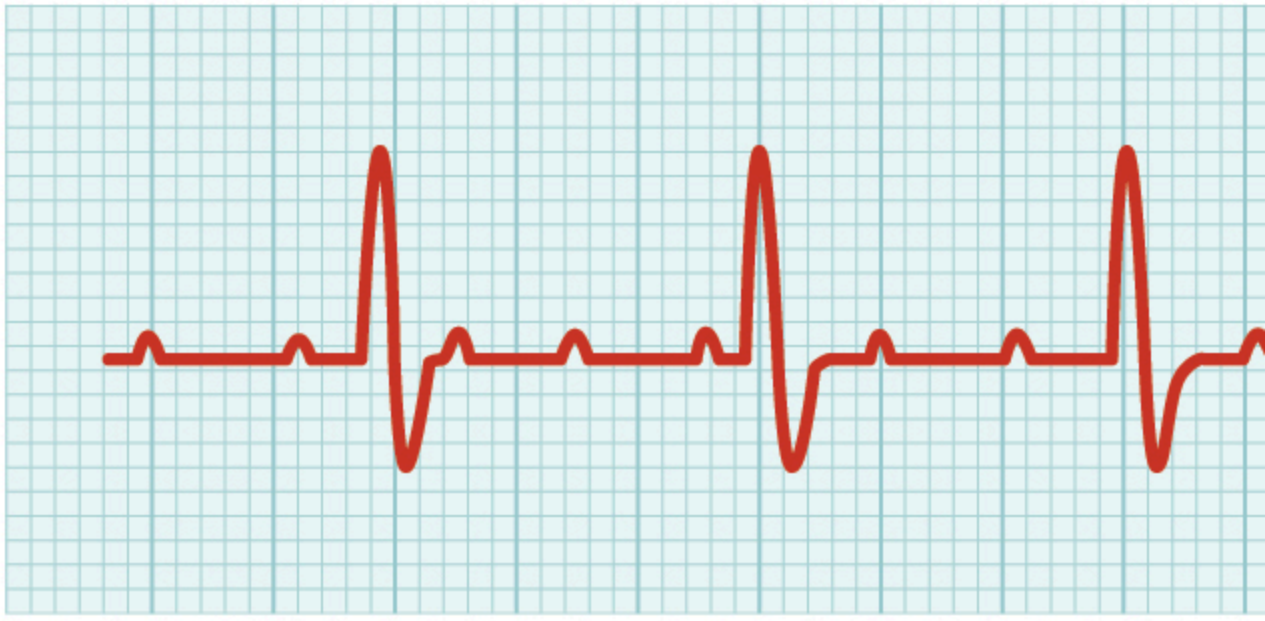
## Arrhythmias

Occasionally, 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 is known as an ectopic focus. An ectopic focus may be stimulated by localized ischemia, exposure to certain drugs, elevated stimulation by both sympathetic or parasympathetic divisions of the autonomic nervous system, or several diseases or pathological

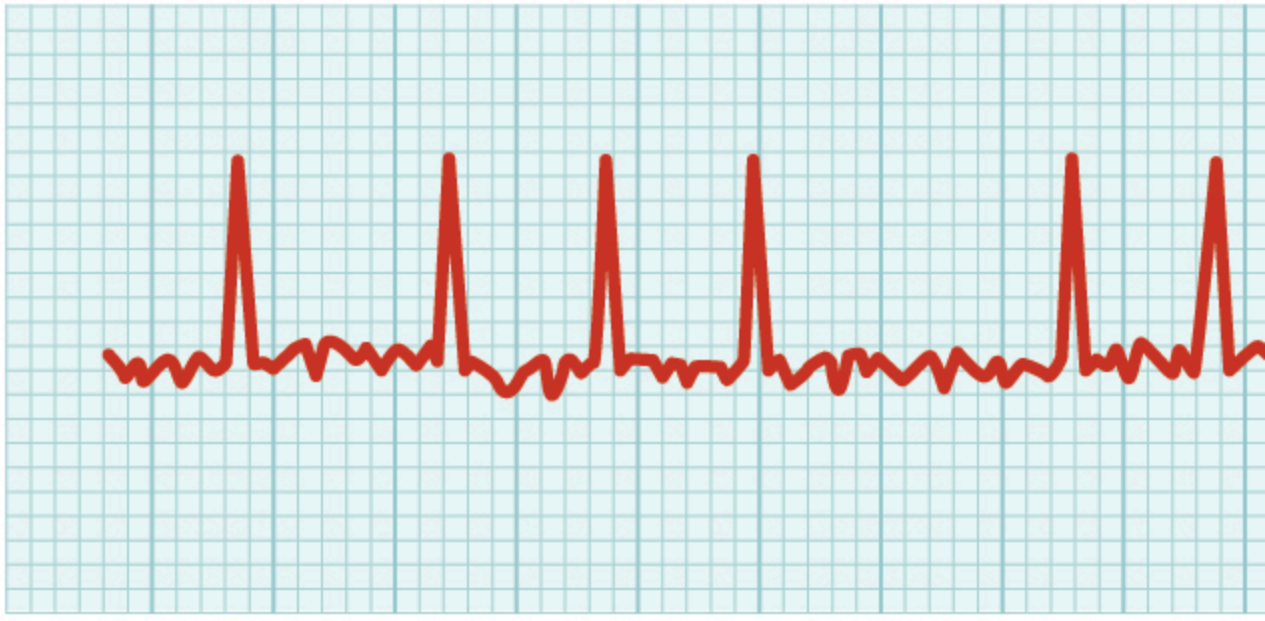
conditions. Occasional occurrences are generally transitory and nonlife threatening, but if the condition becomes chronic, it may lead to either an **arrhythmia,** a deviation from the normal pattern of impulse conduction and contraction, or to **fibrillation,** an uncoordinated beating of the heart. Severe arrhythmias can lead to cardiac arrest, which is fatal if not treated within a few minutes. Abnormalities that may be detected by the ECGs are shown in Figure 6.23.



Antiarrhythmic medications such as sotalol, diltiazem, and amiodarone are used to treat arrhythmias.



(a) Second-degree (partial) block



(b) Atrial fibrillation

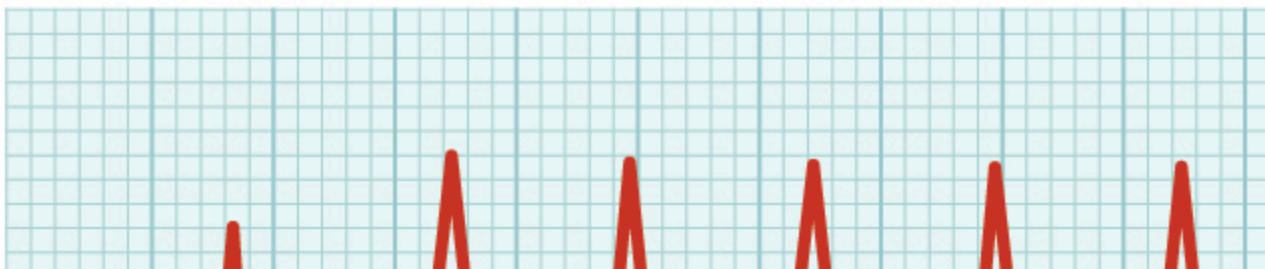


Figure 6.23 Sample arrhythmias: a) In a second-degree or partial block, one-half of the P waves are not followed by increased. (c) In ventricular tachycardia, the shape of the QRS complex is abnormal. (d) In ventricular fibrillation, th

### Heart Failure

Heart failure is a condition in which the heart can't pump enough blood to meet the body's needs. Right-side heart failure occurs if the heart can't pump enough blood to the lungs to pick up oxygen, whereas left-side heart failure occurs if the heart can't pump enough oxygen-rich blood to the rest of the body. Heart failure is a very common condition with over 5.7 million people in the United States having this chronic condition. There is no cure, but the symptoms can be managed for several years with lifestyle modifications and several different types of medications. Causes of heart failure include hypertension, myocardial infarction, and other cardiac and respiratory diseases. Common symptoms of heart failure include peripheral edema and shortness of breath that occur as a result of fluid overload. Many patients are treated with diuretics to manage the symptoms of fluid overload and with antihypertensives to manage the blood pressure. Other medications such as digoxin and dobutamine may also be used to increase the contractility of the heart.
National Heart, Lung, and Blood, National Institute of Health (2019). *Heart failure.* https://www.nhlbi.nih.gov/health-topics/heart-failure

# 6.4 Nursing Process Related to Cardiovascular and Renal Medications

Open Resources for Nursing (Open RN)

### Assessment

Understanding the mechanism of action of a cardiac medication will help a nurse choose the proper assessments to perform on a patient. It is important for a nurse to complete a full cardiac assessment to fully understand the health status of the patient, the safe implementation of the medication, and the expected effectiveness of the medication.

Many cardiovascular medications alter a patient's blood pressure or heart rate, such as antiarrhythmics, cardiac glycosides, antihypertensives, or diuretics. Therefore, it is important for a nurse to assess a patient's blood pressure and heart rate prior to administration. Medication parameters are often included in the order by a healthcare provider. For example, a common medication parameter is to hold a beta blocker if a patient's heart rate is less than 60 beats per minute. Additionally, antiarrhythmic medication will alter the electrical conduction of the heart, so intermittent or continuous ECG monitoring may be required during initial therapy or dose changes.

Electrolytes can play a large role in cardiac conduction and muscle function. Medications that alter electrolytes, such as loop diuretics, require a review of laboratory values before administration. Loop diuretics such as furosemide (Lasix) often cause a depletion of potassium. If a nurse administers a loop diuretic to a patient who already has low serum potassium levels (called hypokalemia), worsening symptoms of hypokalemia will occur, which can cause a life-threatening arrhythmia.

Monitoring kidney function is also important when administering many cardiovascular medications. For example, diuretics can cause renal injury. A nurse should be aware of cardiovascular medications that

are affected by impaired renal function or cause renal injury. In addition, a nurse must appropriately assess and report abnormal laboratory values such as worsening serum creatinine and glomerular filtration rates (GFR). It is also important to assess for signs of dehydration, as well as intake and output in patients taking diuretics.

Anticoagulant medications cause serious risk for bleeding that can be life threatening. Prior to administering medication that alters a patient's coagulation, it is important to assess for signs and symptoms of unusual bleeding or bruising. Laboratory values, such as INR, PTT, or platelets, may also require review prior to administering an anticoagulant medication. Any new abnormal lab values or signs of increased bleeding and internal bleeding should be immediately reported.

## Implementation

Before administration of any cardiovascular medication, it is vital for the nurse to determine if this particular cardiac medication is safe for this patient at this time. For example, if the patient's heart rate or blood pressure is below the anticipated parameters, the medication should be withheld and the prescribing provider notified.

It is also important to consider the effect of the medication before administering it at the ordered time. For example, if a diuretic is prescribed before a patient is sent to a diagnostic test, the test may be disrupted by the need for the patient to urinate, and the dosage should be rescheduled for a later time. A more significant safety concern arises when a patient who is scheduled for surgery is prescribed aspirin or an anticoagulant. The nurse should consider these types of upcoming events before administering medications as they are ordered.

## Evaluation

It is always important to evaluate the patient's response to a medication compared to what is expected. Many medications require dose adjustments to produce desired effect. For example, IV heparin is administered based on a protocol that requires dose adjustment based on PTT or aPTT lab results to achieve therapeutic range (and avoid overdosage that can cause life-threatening bleeding).

It is also important to evaluate the patient's understanding of the purpose and proper use of their cardiac medications, as well as when they should notify their provider of changing symptoms. Additional patient education before discharge home is often required, especially if new medications are prescribed.

Nurses should continue to monitor a patient's blood pressure, heart rate, intake and output, edema, or other cardiac assessments to evaluate if ordered cardiac agents are effective or if further treatment or dosage adjustment is required. The patient should be continually monitored for potential adverse effects of medication, some of which can be life threatening and require prompt notification to the prescribing provider.

# 6.5 Cardiovascular and Renal System Medications

Open Resources for Nursing (Open RN)

If you have not done so already, be sure to read the “Review of Basic Concepts” section earlier in this chapter. To truly understand the mechanism of actions of various cardiovascular and renal system medications and their potential adverse effects, it is vital to have a solid understanding of the anatomy and physiology underlying the cardiovascular system.

The remaining sections of this chapter will review classes of medications related to the cardiovascular and renal systems, including administration considerations, therapeutic effects, adverse/side effects, and patient education.

# 6.6 Antiarrhythmics

Open Resources for Nursing (Open RN)

## Antiarrhythmics

An arrhythmia is any deviation from the normal rate or pattern of a heartbeat. This includes heart rates that are too slow (bradycardia), too fast (tachycardia), or are irregular. The terms dysrhythmia (disturbed heart rhythm) and arrhythmia (absence of heart rhythm) are traditionally used interchangeably in clinical practice despite their difference in meaning.

The ECG is used to identify and monitor an arrhythmia. See more information about ECGs in the “Review of Basic Concepts” section and an overview of arrhythmias in the “Common Cardiac Disorders” section.

Antiarrhythmic medications regulate heart rate and rhythm by manipulating the conduction of electrical signals to change the heart rate or to attempt to revert an arrhythmia to a normal sinus rhythm. All antiarrhythmic medications have a risk of producing an arrhythmia. Some antiarrhythmic medications are used during emergency situations such as cardiac arrest, whereas others are used long-term, such as those used to control atrial fibrillation. Monitoring electrolytes and the ECG patterns are very important assessments for the nurse administering these types of medications.

### Class I – Sodium Channel Blockers

Class I antidysrhythmic medications slow conduction and prolong depolarization by decreasing sodium influx into cardiac cells. There are three subgroups of sodium channel blockers: Class IA, IB, and IC. Quinidine is an example of a Class IA antidysrhythmic. Lidocaine is an example of a Class IB medication that is also used as a local anesthetic. Flecainide is an example of a class IC antidysrhythmic.

**Mechanism of Action**

Quinidine slows conduction and prolongs depolarization by decreasing sodium influx into cardiac cells. The conduction rate and automaticity are decreased. This medication also has alpha-antagonistic properties that cause peripheral vasodilation.

**Indications for Use**

This medication is typically used for life-threatening ventricular dysrhythmias such as ventricular tachycardia or for conversion of atrial fibrillation that has not responded to other therapy.

**Nursing Considerations Across the Lifespan**

Sodium channel blockers are contraindicated in patients who have a history of thrombocytopenia or myasthenia gravis. Use cautiously with patients who have a serious heart block rhythm and do not have an artificial pacemaker, such as a 2nd degree heart block.

There is an increased risk for toxicity with patients who have heart failure and renal or hepatic dysfunction due to drug accumulation. This medication's safety has not been thoroughly evaluated in children and geriatric patients. Grapefruit juice should be avoided by patients taking this medication.

**Adverse/Side Effects**

Quinidine may prolong QT interval leading to ventricular arrhythmias, such as ventricular tachycardia or torsades de pointes.

Quinidine may induce thrombocytopenia. Routine lab work may be evaluated by a patient's health care provider. Common side effects of this medication are nausea, vomiting, diarrhea, fever, chills, abnormal ECG/arrhythmias, and headache.

In many research trials, use of antiarrhythmic therapy for non-life-threatening arrhythmias actually resulted in increased risk of death compared to placebo.[footnote]This work is a derivative of Daily Med by U.S. National Library of Medicine in the public domain. [/footnote]

**Patient Teaching & Education**

Patients should be instructed regarding the significance of compliance with therapeutic drug regimen and take medications as prescribed, even if not symptomatic. Patients or family members may need instruction on how to take pulse rate and parameters regarding reporting to their healthcare provider.

Some antiarrhythmic medications may cause dizziness and may increase sensitivity to light.
uCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral

Now let's take a closer look at the medication grid for quinidine in Table 6.6a.
This work is a derivative of Daily Med by U.S. National Library of Medicine in the public domain.
Medication grids are intended to assist students to learn key points about each medication class. Basic information related to a common generic medication in this class is outlined, including administration considerations, therapeutic effects, and side effects/adverse effects. Prototype/generic medication listed in the medication grid is also hyperlinked directly to a free resource from the U.S. National Library of

Medicine called Daily Med. Because information about medication is constantly changing, nurses should always consult evidence-based resources to review current recommendations before administering specific medication.

Table 6.6a Quinidine Medication Grid

| Class/Subclass | Prototype/ Generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Antiarrhythmic Class I** | quinidine | Monitor blood pressure, heart rate, and QT with administration<br><br>Avoid grapefruit juice<br><br>Maintain consistent sodium intake<br><br>Health care provider should review medications, as this medication may interact with many medications | Control supraventricular arrhythmias | Lengthen the QT interval, arrhythmia, dizziness, and headache<br><br>Thrombocytopenia<br><br>GI: Nausea, diarrhea, and vomiting |

## Class II – Beta Blockers

Class II medications are beta blockers that are used to decrease conduction velocity, automaticity, and the refractory period of the cardiac conduction cycle. Sotalol is a Beta-1 and Beta-2 blocker that also has Class III antiarrhythmic properties. Recall that other types of beta blockers, such as metoprolol, are also used to treat hypertension. See the "Antihypertensives" section later in this chapter for more information about the use of beta blockers to treat hypertension.

### Mechanism of Action

Sotalol is a non-selective beta-adrenergic blocker that prolongs the cardiac action potential.

### Indications for Use

Sotalol is given to patients for life-threatening arrhythmias, such as ventricular arrhythmias or supraventricular arrhythmias. It is not recommended for patients with less than severe arrhythmias.

### Nursing Considerations Across the Lifespan

Titration of this medication is done by evaluating renal function and monitoring QTc on the ECG 2-4 hours after each medication upon initiation. Patients with decreased renal function require dosage adjustment. Sotalol is contraindicated for patients with decreased serum potassium, bradycardia, 2nd or 3rd degree heart block, heart failure, and conditions leading to bronchospasm.

### Adverse/Side Effects

Black Box Warning: This drug can cause arrhythmias. This medication lengthens a patient's QTc interval. Initiation of this medication requires a patient to be in a facility to determine baseline QT and intermittent QT interval checks. QT interval checks are done 2-4 hours after each dose. If the QT corrected interval is greater than 500 msec, the dosing must be changed.

Common side effects for sotalol are arrhythmias, chest pain, palpitations, fatigue, dizziness, hypotension, bradycardia, heart failure, cardiac ischemia, bronchospasm, thyroid abnormalities, and hypoglycemia.


**Patient Teaching & Education**

Patients should be instructed regarding the significance of compliance with therapeutic drug regimen and take medications as prescribed, even if not symptomatic. Patients or family members may need instruction on how to take pulse rate and blood pressure. They should receive parameters regarding reporting to their healthcare provider. They should report any pulse rate less than 50 bpm and significant changes in blood pressure.

Patients should also be advised that these medications may cause dizziness and visual changes. Patients may also notice orthostatic blood pressure decrease with position changes and should be advised to change positions slowly. If the patient notices irregular, fast heart rate or experiences any fainting episodes, they should notify their healthcare provider immediately.

Additionally, these medications may also mask the signs of hypoglycemia, so diabetic patients must use extra caution to monitor for low blood sugar. They may also increase cold sensitivity.[footnoteuCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral[/footnote]

Now let's take a closer look at the medication grid for sotalol in Table 6.6b.


Table 6.6b Sotalol Medication Grid

| Class/Subclass | Prototype/ Generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Antiarrhythmic Class 2** | sotalol | Black Box Warning: Drug induced arrhythmias<br><br>Strict QTc monitoring with initiation of therapy<br><br>Do not double dose<br><br>Monitor blood pressure and heart rate | Treatment of life threatening arrhythmias | Arrhythmias due to lengthening QTc<br><br>Chest pain, palpitations, dizziness, fatigue, hypotension, heart failure, cardiac ischemia, and bradycardia<br><br>Bronchospasm<br><br>Thyroid abnormalities |

# Critical Thinking Activity 6.6a
# Image of lightbulb
# in a circle

1. What should a nurse assess before and after administration of sotalol?

## Class III – Potassium Channel Blockers

Class III medications prolong repolarization by blocking the potassium channels in cardiac cells that are responsible for repolarization. They are used for emergency treatment of ventricular dysrhythmias. Amiodarone is an example of an antidysrhythmic that has predominantly Class III properties.

### Mechanism of Action

Class III medications prolong repolarization by blocking the potassium channels in cardiac cells that are responsible for repolarization. Amiodarone also antagonizes alpha and beta receptors.

### Indications for Use

Amiodarone is indicated only for the treatment of life-threatening recurrent ventricular arrhythmias when these have not responded to documented adequate doses of other available antiarrhythmics or when alternative agents could not be tolerated.

### Nursing Considerations Across the Lifespan

Amiodarone can cause fetal injury when administered to a pregnant patient. Use cautiously with the geriatric population who may have decreased hepatic, cardiac, or renal function. Read drug label information carefully due to several potential drug interactions.

### Adverse/Side Effects

Black Box Warnings: Amiodarone has several fatal toxicities such as pulmonary toxicity, exacerbation of arrhythmia, liver injury, and heart block. Patients who require initiation of this therapy should be hospitalized and monitored closely. Neurological impairments (such as fatigue, tremors, involuntary movements, poor coordination, and gait) and GI disturbances are common adverse effects. Vision

changes/loss of vision and photosensitivity may also occur.

**Patient Teaching & Education**

Patients should be advised to closely follow the recommended dosing regimen. If one dose of medication is missed, the patient should follow the normal dosing schedule and resume with the next dose. If more than one dose of medication is missed, the patient should call the healthcare provider for guidance. Patients should be compliant with all follow-up appointments and monitoring.

Patients should avoid drinking grapefruit juice during medication therapy. Some patients may experience photosensitivity and protective measures should be taken.
uCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral

Now let's take a closer look at the medication grid for amiodarone in Table 6.6c.
This work is a derivative of Daily Med by U.S. National Library of Medicine in the public domain.

Table 6.6c Amiodarone Medication Grid

| Class/Subclass | Prototype/ Generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Antiarrhythmic Class 3** | amiodarone | Black Box Warning: Fatal toxicities<br><br>Read drug label information due to several drug interactions<br><br>Monitor blood pressure and heart rate for profound hypotension and bradycardia<br><br>Initiation of therapy typically requires patients to be hospitalized to receive a loading dose | Treatment of life-threatening ventricular arrhythmia | Fatal toxicities<br><br>Neurological impairments<br><br>GI upset<br><br>Worsening arrhythmia, bradycardia, hypotension<br><br>Thyroid abnormalities<br><br>Vision changes<br><br>Photosensitivity |

## Class IV – Calcium Channel Blockers

Class IV medications include the calcium channel blockers verapamil and diltiazem. These medications increase the refractory period of the AV node by slowing the influx of calcium ions, thus decreasing the ventricular response and decreasing the heart rate. This medication may be used to control heart rate associated with supraventricular tachycardias. Calcium channel blockers are also used to treat hypertension because they relax smooth muscle and cause vasodilation. See the "Antihypertensives" section later in this chapter for more information about their use in treating hypertension.

**Mechanism of Action**

Diltiazem inhibits calcium during depolarization to decrease the workload of the heart and increase

oxygen supply to the myocardium. This medication will relax smooth muscle and decrease peripheral resistance.

**Indications for Use**

Diltiazem is used to treat angina, hypertension, and supraventricular tachycardias.

**Nursing Considerations Across the Lifespan**

This medication is not given to hypotensive patients, patients with acute myocardial infarction, or patients with 2nd or 3rd degree heart block or sick sinus syndrome.

**Adverse/Side Effects**

Diltiazem can potentially worsen signs and symptoms of heart failure due to the negative inotropic effect. Patients may experience bradycardia, worsening 1st degree AV block, syncope, edema, hypotension, headache, dizziness, or hepatic injury.


**Patient Teaching & Education**

Patients should be advised to closely follow the recommended dosing regimen. Patients or family members may need instruction on how to take a pulse rate and should report any pulse less than 50 bpm. Patients should also be advised that this medication may cause dizziness and visual changes. Patients may also notice orthostatic blood pressure decrease with position changes and should be advised to change positions slowly.

Patients should be advised to avoid grapefruit juice during medication therapy. They should also monitor for gingival sensitivity and be sure to maintain good oral hygiene. Patients may also notice increased photosensitivity and should take protective measures.[footnoteuCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral[/footnote]

Now let's take a closer look at the medication grid for diltiazem in Table 6.6.d.


Table 6.6d Diltiazem Medication Grid

| Class/Subclass | Prototype/Generic | Administration Considerations | |
|---|---|---|---|
| **Antiarrhythmic Class 4** | diltiazem | Monitor blood pressure and heart rate | Reduc to myo |

## Adenosine

Adenosine is a unique medication given to patients who are experiencing paroxysmal supraventricular tachycardia. It is given all at once as a bolus in either a 6 or 12 mg dose to slow electrical conduction to restore a normal sinus rhythm.

### Mechanism of Action

Adenosine will slow conduction through the AV node to restore normal sinus rhythm.

### Indications for Use

Adenosine is used to treat paroxysmal supraventricular tachycardia.

### Nursing Considerations Across the Lifespan

This medication is an emergent type of medication. Use cautiously with geriatric patients with decreased cardiac function.

This medication is contraindicated with patients who have 2nd or 3rd degree AV block, sinus node disease, or any known hypersensitivity.

At time of administration, a nurse may see no electrical activity on an ECG for a brief few seconds before normal sinus rhythm is restored. It is important to warn the patient about an extremely uncomfortable feeling during this short period of time.

### Adverse/Side Effects

Patients receiving adenosine may experience prolonged asystole, arrhythmias, palpitations, facial flushing, hypotension, bronchospasm, shortness of breath, dizziness, seizures, loss of consciousness, numbness and tingling to upper extremities, and nausea.


### Patient Teaching & Education

Patients should be advised to closely follow the recommended dosing regimen. Patients or family members may need instruction on how to take a pulse rate and should report any abnormalities. Patients should also be advised that this medication may cause dizziness and visual changes. Patients may also notice orthostatic blood pressure decrease with position changes and should be advised to change positions slowly.

Patients should be advised to avoid grapefruit juice during medication therapy. They should also monitor for gingival sensitivity and be sure to maintain good oral hygiene. Patients may also notice increased photosensitivity and should take protective measures.


Now let's take a closer look at the medication grid for adenosine in Table 6.6e.


Table 6.6e Adenosine Medication Grid

| Class/Subclass | Prototype/ Generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Antiarrhythmic** | adenosine | Place the patient in a supine position and inject medication rapidly followed by saline flush | Restore normal sinus rhythm | Prolonged asystole, arrhythmias, palpitations, facial flushing, hypotension, bronchospasm, shortness of breath, and dizziness<br><br>Seizures, loss of consciousness, numbness, and tingling to upper extremities<br><br>Nausea |

# 6.7 Cardiac Glycosides

Open Resources for Nursing (Open RN)

## Digoxin

Digoxin is a cardiac glycoside medication that has been used for centuries to treat heart failure. It has three effects on heart muscle: positive inotropic action (increases contractility, stroke volume and, thus, cardiac output), negative chronotropic action (decreases heart rate), and negative dromotropic action (decrease conduction of cardiac cells).
McCuistion, L., Vuljoin-DiMaggio, K., Winton, M, & Yeager, J. (2018). *Pharmacology: A patient-centered nursing process approach.* pp. 443-454. Elsevier.

### Mechanism of Action

Digoxin works by inhibiting the sodium and potassium pump, which results in an increase in intracellular sodium and an influx of calcium into cardiac cells, causing the cardiac muscle fibers to contract more efficiently and increase cardiac output.
McCuistion, L., Vuljoin-DiMaggio, K., Winton, M, & Yeager, J. (2018). *Pharmacology: A patient-centered nursing process approach*. pp. 443-454. Elsevier.

### Indications for Use

This medication is used as second-line treatment for patients who have heart failure or atrial fibrillation. Due to the risk for digoxin toxicity, the clinical use of digoxin has decreased and alternative, safer medications are being used.

### Nursing Considerations Across the Lifespan

Apical pulse should be taken for a full minute before administration of this medication. If the apical pulse is less than 60, the dose should be withheld and the prescribing provider notified.

Serum digoxin levels should be monitored, with a normal therapeutic range from 0.8 to 2 ng/mL.

Serum potassium levels should also be closely monitored for patients on digoxin because hypokalemia increases the effect of digoxin and can result in digoxin toxicity. Normal potassium level is 3.5 to 5.0 mEq/L, and a result less than 3.5 should be immediately reported to the provider.

Nurses should closely monitor signs of digoxin toxicity. Geriatric patients have an increased risk for developing digoxin toxicity. Digibind is used to treat digoxin toxicity.

**Adverse/Side Effects**

Overdose or accumulation of digoxin causes digoxin toxicity. Signs and symptoms of digoxin toxicity are bradycardia (heart rate less than 60), nausea, vomiting, visual changes (halos), and arrhythmias. Cardiotoxicity is a serious adverse effect with ventricular dysrhythmias. Toxicity of this medication typically occurs at greater than 2 ng/mL, but some patients may have signs and symptoms at lower levels. Pediatric patients typically present with bradycardia or arrhythmias if toxicity is occuring.

Decreased renal function, hypokalemia, hypercalcemia, and hypomagnesemia may increase risk for digoxin toxicity.

Common side effects include GI symptoms, headache, weakness, dizziness, anxiety, depression, delirium, and hallucination.


**Patient Teaching & Education**

The patient should be instructed to follow the prescribed dosing regimen and take medications at the same time each day. The patient should be cautious not to double up on medication doses. Additionally, the patient should consult the healthcare provider if two or more doses of medication are missed for follow-up instruction.

Patients should receive education regarding pulse rate monitoring and report any pulse rate less than 60. If the patient experiences signs of digoxin toxicity, this should be reported to the provider immediately. The medication should be stored in its original container and care should be taken not to mix the medication with other medications.


Now let's take a closer look at the medication grid for digoxin in Table 6.7a.


Table 6.7a Digoxin Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Cardiac** | digoxin | Assess apical heart rate | Increased cardiac output | Digoxin toxicity; |

| | | | | |
|---|---|---|---|---|
| **glycosides** | | Assess serum digoxin and potassium levels<br><br>Assess for signs and symptoms of digoxin toxicity | | early signs include nausea, vomiting, and diarrhea<br><br>Bradycardia and arrhythmias<br><br>Headache, weakness, dizziness, and mental changes such as anxiety or hallucinations<br><br>Gynecomastia (with prolonged use) |

# Critical Thinking Activities 6.7a
# Image of lightbulb in a circle

1. Why should a nurse assess the apical pulse for 1 full minute before administering digoxin?
2. How does a nurse evaluate if digoxin is effective?
3. Why must the nurse monitor serum potassium levels as well as digoxin levels?
4. A nurse enters a patient's room and the patient complains "My vision seems strange and I feel nauseated." What is the nurse's next best action?

Note: Answers to the Critical Thinking activities can be found in the "Answer Key" sections at the end of the book.

# Digibind

Digibind is used to treat digoxin toxicity.

**Mechanism of Action**

Digibind binds to digoxin molecules, reducing free digoxin.

**Indications for Use**

This medication is the antidote for digoxin. Digibind will be administered when a patient is experiencing life-threatening digoxin toxicity.

**Nursing Considerations Across the Lifespan**

There are no contraindications when using digibind.

**Adverse/Side Effects**

The most common effects a patient may experience are to have worsening heart failure, worsening atrial fibrillation, and hypokalemia.


**Patient Teaching & Education**

The patient should report any signs of worsening heart failure, atrial fibrillation, or hypokalemia immediately to the healthcare provider.


Now let's take a closer look at the medication grid for digibind in Table 5.6b.


Table 5.6b Medication Grid for Digibind.

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Antidote** | digoxin immune fab (Digibind) | Give when patients are experiencing life- threatening digoxin toxicity | Reduce free digoxin | Worsening heart failure<br><br>Worsening atrial fibrillation<br><br>Hypokalemia |

# 6.8 Antianginal - Nitrates

Open Resources for Nursing (Open RN)

Antianginal medication is used to treat angina pectoris. Angina is chest pain caused by inadequate blood flow, resulting in hypoxia of the cardiac tissue. Angina can be chronic pain caused by atherosclerosis in coronary artery disease or acute pain caused by a myocardial infarction.

Antianginals increase blood flow to the heart or decrease oxygen demand by the heart. Nitrates promote vasodilation of coronary arteries and veins. Beta blockers and calcium channel blockers are also used to decrease workload of the heart and decrease oxygen demands.

Nitrates may come in a variety of routes, such as sublingual, extended-release tablets, creams, transdermal patches, and intravenously. The grid below focuses on administration via sublingual tablets. Sublingual tablets are prescribed PRN ("as needed") for patients who are experiencing chronic, stable angina due to coronary artery disease.

**Mechanism of Action**

Nitroglycerin relieves angina by relaxing vascular smooth muscle, resulting in vasodilation.

**Indications for Use**

Nitroglycerin is used to relieve angina due to coronary artery disease, during times of an acute attack, or prophylactically.

**Nursing Considerations Across the Lifespan**

Patients taking sildenafil (Viagra) or similiar medications for erectile dysfunction in the previous 24 hours may not take nitroglycerin as this may result in a dangerous drop in blood pressure.

Nitroglycerin should not be used in pregnant women or those who are breastfeeding.

Nitroglycerin is contraindicated in patients who have severe anemia, increased intracranial pressure, hypersensitivity, or circulatory failure.

**Adverse/Side Effects**

Patients taking nitroglycerin may experience hypotension, palpitations, headache, weakness, sweating, flushing, nausea, vomiting, or dizziness.

Patients should allow medication to dissolve under their tongue. This route allows immediate absorption into the circulation and avoids first-pass metabolism by the liver. Patients may take up to one tablet every 5 minutes, up to 3 sublingual tablets within 15 minutes to relieve chest pain. If chest pain is not relieved after the first dose, 911 should be called. Nitroglycerin may also be used prophylactically 5 to 10 minutes prior to engaging in activities that might precipitate an acute attack.

**Patient Teaching & Education**

Instruct patients to avoid eating or smoking during administration as this may alter absorption. Patients should sit during administration to decrease the risk for injury due to the possibility of hypotension, dizziness, and weakness. Nitroglycerin decomposes when exposed to heat or light, so it should be stored in the original, airtight glass container. See Figure 6.24

for an image of nitroglycerin containers.
This work is a derivative of Daily Med by U.S. National Library of Medicine in the public domain.

Historically, patients have been taught to seek emergency help (call 911) if pain persists after the 3rd dose of medication. However, new guidelines from the American Heart Association urge patients to call 911 after the first dose if symptoms are not improved or become worse.
O'Gara, P., Kushner, F. , Ascheim, D. , Casey, D., Chung, M., de Lemos, J., Ettinger, S., Fang, J, Fesmire, F., Franklin, B., Granger, C., Krumholz, H., Linderbaum, J., Morrow, D., Newby, L., Ornato, J., Ou, N., Radford, M., Tamis-Holland, J., Tommaso, C., Tracy, C., Woo, Y., & Zhao, D. (2013). ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. *Circulation, 127*(4). https://www.ahajournals.org/doi/full/10.1161/CIR.0b013e3182742cf6?url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org&rfr_dat=cr_pub%3Dpubmed

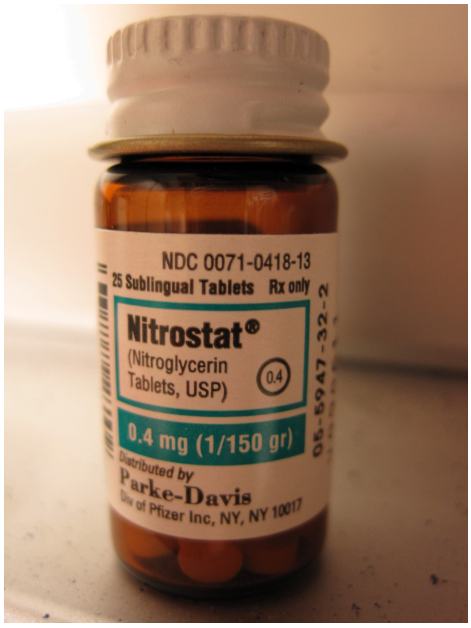


Figure 6.24 Sublingual nitroglycerin should be stored in its original, air tight glass container

Now let's take a closer look at the medication grid for nitroglycerin in Table 6.8.
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Table 6.8 Nitroglycerine Medication Grid

| Class/ Subclass | Prototype- generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Nitrate** | nitroglycerin | Patients may take up to 3 sublingual tablets within 15 minutes (1 every 5 minutes) to relieve chest pain | Decrease chest pain | Hypotension and palpitations<br><br>Headache, weakness, |

| | | | | |
|---|---|---|---|---|
| | | If symptoms are not improved after the first dose or become worse, or if the pain persists after the 3rd dose of medication, seek emergency help (call 911). Nurses should check BP after each dose<br><br>No eating or smoking during administration of SL tablet<br><br>Do not chew or crush SL tablet<br><br>Advise patients to sit while taking this medication | | sweating, flushing, nausea, vomiting, and dizziness |

## Critical Thinking Activity 6.8
## Image of lightbulb in a circle

A patient was administered the first dose of nitroglycerin at 1305 for acute angina. What should the nurse evaluate after administration?

Note: Answers to the Critical Thinking activities can be found in the "Answer Key" sections at the end of the book.

# 6.9 Diuretics

Open Resources for Nursing (Open RN)

Diuretics are used to decrease blood pressure and to decrease symptoms of fluid overload such as edema. There are many classifications of diuretics. We will discuss loop, thiazide, and potassium-sparing diuretics. Other diuretics, such as osmotic diuretics, are used to decrease fluid from cerebrospinal fluid and the brain.

Diuretics cause diuresis (increased urine flow) by inhibiting sodium and water reabsorption from the kidney tubules. By eliminating excess water, blood volume and blood pressure, as well as preload, are decreased.

Diuretics are often used in combination with other antihypertensive agents to reduce a patient's blood pressure.

## Furosemide

### Mechanism of Action

Loop diuretics inhibit absorption of sodium and chloride in the loop of henle and proximal and distal tubules, thus causing fluid loss, along with sodium, potassium, calcium, and magnesium losses. Loop diuretics are very potent diuretics and are used when a patient has an exacerbation of fluid overload.

### Indications for Use

Furosemide is used to treat patients with edema and is also used to treat hypertension. IV furosemide is used to urgently treat pulmonary edema.

### Nursing Considerations Across the Lifespan

The onset of diuresis following oral administration is within 1 hour. The peak effect occurs within the first or second hour. The duration of diuretic effect is 6 to 8 hours. When possible, loop diuretics should be administered in the morning, and evening doses should be avoided (unless urgent) so that sleep is not disturbed.

Nurses should continually monitor for dehydration and electrolyte imbalances that can occur with excessive diuresis, such as dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, arrhythmia, or gastrointestinal disturbances such as nausea and vomiting.

Use cautiously in the geriatric population who have decreased renal function. Kidney function should be monitored closely for all patients because this is a potent medication that works within the kidney tubules.

Monitor the patient closely for hypokalemia if furosemide is used concomitantly with digoxin. Hypokalemia may increase the risk of digoxin toxicity.

### Adverse/Side Effects

Adverse effects include dehydration, hypotension, and electrolyte imbalances such as hypokalemia. Health care providers may add potassium to a patient's scheduled medication list to decrease risk of hypokalemia. If using IV route, the administration must be given slowly to reduce the risk of the patient

developing ototoxicity.


**Patient Teaching & Education**

Advise patients to change position slowly as they may experience orthostatic changes. Patients should also report weight gain of more than three pounds in a day to their healthcare provider. Patients should also be encouraged to enjoy potassium-rich foods during loop diuretic drug therapy.


Now let's take a closer look at the medication grid for furosemide in Table 6.9a.


Table 6.9a Furosemide Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Loop diuretic** | furosemide | Assess blood pressure<br><br>Monitor electrolytes (potassium)<br><br>Promote potassium-rich diet<br><br>Assess renal function<br><br>Assess for dehydration, intake and output<br><br>Monitor weight | Based on indication; decreased blood pressure or edema | Dehydration<br><br>Electrolyte depletion (especially potassium)<br><br>Ototoxicity with rapid IV infusion<br><br>Renal impairment |

# Critical Thinking Activity 6.9

# Image of lightbulb in a circle

Mrs. Smith is a 79-year-old widow who has lived alone for the past 5 years. Three years ago she was hospitalized for an MI, which resulted in heart failure. She is compliant with her medications, which include digoxin (Lanoxin) 0.125 mg daily, furosemide (Lasix) 40 mg daily, and potassium (K-Dur) 20 mEq daily.

Recently Mrs. Smith ran out of her potassium and thought that because it was "just a supplement," it would be OK to go without it until the next time she went to town to fill the prescription. She has not taken her potassium for a week.

Today she comes into the clinic with generalized weakness, fatigue, nausea, and diarrhea. Her BP is 104/62, pulse 98 bpm and slightly irregular, RR 20, and temp 97.2 F. Blood is drawn and shows serum sodium level of 150 mEq/L, digoxin level of 2.6ng/ml and potassium level of 3.2 mEq/L.

1. What assessments should a nurse do before and after administering a diuretic?

2. What are the signs and symptoms of digoxin toxicity? What can happen to a patient who has toxic levels of digoxin?

3. What is the normal range for serum potassium level?

4. What classification of medication is furosemide (Lasix)?

5. Is dehydration a risk for patients on furosemide (Lasix)? Why or why not?

6. How would you assess for dehydration?

7. What electrolyte imbalance(s) can occur in patients taking furosemide (Lasix)?

8. What relationship exists between this patient's furosemide, digoxin, and potassium levels?

Note: Answers to the Critical Thinking activities can be found in the "Answer Key" sections at the end of the book.

## Hydrochlorothiazide

### Mechanism of Action

Thiazide diuretics work near the distal tubule to promote the excretion of sodium and water, thus causing diuresis. They are not effective for immediate diuresis.

**Indications for Use**

Hydrochlorothiazide diuretics are used to manage hypertension and edema.

**Nursing Considerations Across the Lifespan**

Thiazide diuretics are contraindicated for patients who have anuria or hypersensitivity.

After oral use, diuresis begins within 2 hours, peaks in about 4 hours, and lasts about 6 to 12 hours.

Use with caution in severe renal disease.

**Adverse/Side Effects**

Patients who are taking thiazide diuretics should be monitored for electrolyte depletion, dehydration, weakness, hypotension, renal impairment, and hypersensitivities.


**Patient Teaching & Education**

Patients should be instructed to take these medications at the same time each day and notify their healthcare provider if they experience significant changes in weight. Thiazide diuretics may cause orthostatic changes so individuals should change positions slowly. Additionally, some patients may note increased photosensitivity so protective measures should be taken. Patients should monitor their blood pressure and comply with interventions to reduce hypertension.
uCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral

Now let's take a closer look at the medication grid for hydrochlorothiazide in Table 6.9b.


Table 6.9b Hydrochlorothiazide Medication Grid

| Class/ Subclass | Prototype- generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|

| | | | | |
|---|---|---|---|---|
| **Thiazide diuretics** | hydrochlorothiazide | Assess blood pressure<br><br>Monitor electrolytes (potassium)<br><br>Promote potassium-rich diet<br><br>Assess renal function<br><br>Assess for dehydration, intake and output<br><br>Monitor weight | Decrease blood pressure<br><br>Decrease edema | Electrolyte depletion<br><br>Dehydration and weakness<br><br>Hypotension<br><br>Renal impairment<br><br>Hypersensitivity (vasculitis, respiratory distress, photosensitivity, rash) |

## Spironolactone

Spironolactone is a potassium sparing diuretic that is used as a mild diuretic or in combination with another diuretic.

### Mechanism of Action

Spironolactone acts primarily through competitive binding of receptors at the aldosterone-dependent sodium-potassium exchange site in the distal convoluted renal tubule. Spironolactone causes increased amounts of sodium and water to be excreted, while potassium is retained.

### Indications for Use

Spironolactone is used to treat hypertension and to control edema for patients with heart failure or liver dysfunction.

### Nursing Considerations Across the Lifespan

This medication may cause hyperkalemia. Monitor urine output and report if less than 30 ml/hour. Use cautiously with patients who have renal impairment due to increased risk for hyperkalemia. Use cautiously in patients with liver impairment. Administer in the morning to avoid nocturia.

### Adverse/Side Effects

Hyperkalemia, hyperglycemia, hyperuricemia, dehydration, hypotension, renal impairment, hypersensitivity, and gynecomastia. This medication may increase risk for lithium toxicity.

**Patient Teaching & Education**

Patients should be instructed to take these medications at the same time each day and notify their healthcare provider if they experience significant changes in weight. Diuretics may cause orthostatic changes so individuals should change positions slowly. Patients should be advised to avoid salt substitutes and foods that contain high levels of potassium.
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Now let's take a closer look at the medication grid for spironolactone in Table 6.9c


Table 6.9c Spironolactone Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Potassium Sparing diuretics** | spironolactone | Assess blood pressure<br><br>Monitor electrolytes (potassium)<br><br>Assess renal function<br><br>Assess for dehydration, intake and output<br><br>Monitor weight | Decrease blood pressure<br><br>Decrease edema | Hyperkalemia, hyperglycemia,hyperuricemia<br><br>Dehydration<br><br>Hypotension<br><br>Renal impairment<br><br>Hypersensitivity (vasculitis, fever, anaphylactic reactions, rash)<br><br>Gynecomastia |

# 6.10 Antihypertensives

Open Resources for Nursing (Open RN)

Many different medication classifications are used to treat **hypertension**. It is important to understand the different mechanisms of action for different classes of antihypertensives because patients are often

on a combination of medications that work synergistically to manage blood pressure. These medications are also discussed in the "Autonomic Nervous System" chapter, with more information provided regarding the specific receptors they affect.

## Alpha-2 Agonist

**Clonidine** is an Alpha-2 agonist. You can read more information about Alpha-2 agonists in the "Autonomic Nervous System" chapter.

### Mechanism of Action

Clonidine stimulates the alpha-adrenergic receptors, resulting in vasodilation and decreased blood pressure, thus decreasing peripheral resistance, increased blood flow to the kidneys, and decreased afterload.

### Indications for Use

Clonidine is used to treat hypertension and ADHD.

### Nursing Considerations Across the Lifespan

Monitor BP and pulse rate. Dosage is usually adjusted to patient's blood pressure because it can cause hypotension, bradycardia, and sedation. Rebound hypertension may occur if stopped abruptly.


### Patient Teaching & Education

Patients should be compliant with medication therapy and take the medication at the same time each day. They should be careful not to take more than the prescribed dose within a 24-hour period. Do not abruptly cease medication as rebound hypertension might occur. Medications may cause orthostatic changes so individuals should change positions slowly. Additionally, medications may cause dry mouth and dry eyes. Individuals should also avoid the use of alcohol and other CNS depressants while taking these medications.


Now let's take a closer look at the medication grid for clonidine in Table 6.10a.


Table 6.10a Clonidine Medication Grid

| Class | Prototype | Administration Considerations | Therapeutic Effects | Adverse Effects |
|---|---|---|---|---|
| **Alpha-2 Agonist** | **clonidine** | Monitor blood pressure and pulse rate frequently<br><br>Dosage is usually adjusted to patient's BP and tolerance | Treat hypertension or ADHD | Hypotension<br><br>Bradycardia<br><br>Sedation |

| | | | | |
|---|---|---|---|---|
| | | | | Rebound HTN if stopped abruptly |

## Beta-1 Antagonist

**Metoprolol** is a selective Beta-1 blocker. You can read more information about Beta-1 antagonists in the "Autonomic Nervous System" chapter.

### Mechanism of Action

Metoprolol primarily blocks Beta-1 receptors in the heart, causing decreased heart rate and decreased blood pressure. However, higher doses can also block Beta-2 receptors in the lungs, causing bronchoconstriction.

### Indications for Use

Metoprolol is commonly used to treat high blood pressure, chest pain due to poor blood flow to the heart, and several heart conditions involving an abnormally fast heart rate. It is used as an early intervention during myocardial infarction (MI) to reduce workload of the heart.

### Nursing Considerations Across the Lifespan

ER formulations should not be crushed. Assess patient's apical pulse rate before administering; if it is less than 60 beats/minute, withhold the drug and call the prescriber immediately, unless other parameters are provided. In diabetic patients, monitor glucose level closely because the drug masks common signs and symptoms of hypoglycemia.

### Adverse Effects

The most serious potential adverse effects are shortness of breath, bradycardia, and worsening heart failure. Other adverse effects include fatigue, dizziness, depression, insomnia, nightmares, GI upset, erectile dysfunction, dyspnea, and wheezing. Black Box Warning: When stopping therapy, taper dosage over 1 to 2 weeks because abrupt discontinuation may cause chest pain or MI.


### Patient Teaching & Education

Patients should be compliant with medication therapy and take the medication at the same time each day. Do not abruptly cease medication as arrhythmias, hypertension, or ischemia may develop. Patients and families should be instructed to check pulse and blood pressure and report abnormalities to the healthcare provider. Additionally, these medications may cause side effects of dizziness and cold sensitivity.


Now let's take a closer look at the medication grid for metoprolol in Table 6.10b.

Table 6.10b Medication Grid for Metoprolol

| Class | Prototype – generic | Administration Considerations | Therapeutic Effects | Adverse Effects |
|---|---|---|---|---|
| **Beta-1 Antagonist** | Selective B blocker: **metoprolol** | Do not crush ER formulations<br><br>Always assess apical HR and if less than 60, do not administer and call the prescriber unless other parameters are provided<br><br>Monitor blood sugar in diabetic patients because drug can mask symptoms of hypoglycemia | Decreases blood pressure or controls rapid heart rate | Most serious: hypotension, bradycardia, and worsening HF<br><br>Other:<br><br>CNS: fatigue, dizziness, depression, insomnia, nightmares<br><br>GI upset<br><br>GU: erectile dysfunction<br><br>Respiratory: dyspnea and wheezing |

## ACE Inhibitor (Angiotensin Converting Enzyme)

Captopril is an example of an ACE (angiotensin converting enzyme) inhibitor.

**Mechanism of Action**

This medication blocks the conversion of Angiotensin I to Angiotensin II in the renin-angiotensin-aldosterone system. This will lead to vasodilation and sodium and water excretion by blocking aldosterone. See more information about the renin-angiotensin-aldosterone system in the “Review of Basic Concepts” section of this chapter.

**Indications for Use**

Captopril is used to treat hypertension and heart failure. This medication also helps reduce diabetic nephropathy.

**Nursing Considerations Across the Lifespan**

Do not administer to patients who are pregnant. Use with caution with patients who have diabetes.

Avoid use with other medications that increase potassium. This medication may increase risk for lithium toxicity.

**Adverse/Side Effects**

Black Box Warning: Patients who become pregnant should discontinue this medication due to the risk of fetal harm or fetal death.

Patients taking this medication may experience hypotension, cough, hyperkalemia, increased risk for infection, angioedema, anaphylactoid reactions, or proteinuria. Patients who experience increased facial swelling or difficulty swallowing or breathing should seek emergency medical attention. Report a persistent cough or angioedema to the health care provider.


**Patient Teaching & Education**

Medications should be taken as directed. Patients taking ACE inhibitors should be cautioned to avoid salt substitutes or foods high in potassium. Additionally, the medication may alter the sense of taste, but this generally resolves within 2-3 months of medication therapy.

Patients taking ACE inhibitors may also experience a persistent cough throughout the duration of medication therapy.
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Now let's take a closer look at the medication grid for captopril in Table 6.10c.


Table 6.10c Captopril Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **ACE Inhibitor** | captopril | Black Box Warning: Do not use while pregnant<br>Monitor blood pressure<br>Report cough<br>Assess for facial swelling or difficulty breathing | Decrease blood pressure<br>Decrease fluid volume status | Hypotension<br>Cough<br>Hyperkalemia<br>Neutropenia or agranulocytosis<br>Angioedema |

| | | | | |
|---|---|---|---|---|
| | | | | Anaphylactoid reactions<br><br>Proteinuria |

## Angiotensin II Receptor Blocker (ARB)

Losartan is an example of an Angiotensin II receptor blocker, also referred to as an ARB. ARBs are similar to ACE inhibitors in that they act on the renin-angiotensin-aldosterone system (RAAS). However, the difference is that they block Angiotensin II and cause vasodilation and decreased peripheral resistance, but are not likely to cause the cough that ACE inhibitors can.

### Mechanism of Action

Losartan blocks Angiotensin II in the renin-angiotensin-aldosterone system to produce vasodilation.

### Indications for Use

ARB is used for treatment of hypertension and to prevent nephropathy in diabetic patients.

### Nursing Considerations Across the Lifespan

Do not administer to patients who are pregnant. It is not recommended for children under 6. Anticipate dosage adjustment with hepatic impairment. This drug can cause renal impairment and hyperkalemia.

### Adverse/Side Effects

Black Box Warning: Patients who become pregnant should discontinue this medication due to the risk of fetal harm or fetal death.

Patients taking this medication may experience hypotension, dizziness, increased risk for infection, angioedema, or proteinuria. Patients who experience increased facial swelling or difficulty swallowing or breathing should seek emergency medical attention.

### Patient Teaching & Education

Medications should be taken as directed at the same time each day. Patients should not discontinue therapy unless directed to by their healthcare provider. Patients should be careful to avoid salt substitutes and foods with high levels of potassium. ARBs may cause orthostatic changes and patients should be cautioned to change positions slowly.
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Now let's take a closer look at the medication grid for losartan in Table 6.10d.

Table 6.10d Medication Grid for Losartan

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **ARB** | losartan (Cozaar) | Black Box Warning: Do not use while pregnant<br><br>Monitor blood pressure | Decrease blood pressure | Hypotension and dizziness<br><br>Hyperkalemia<br><br>Proteinuria |

# Critical Thinking Activity 6.10
# Image of lightbulb in a circle

A male 65-year-old patient has the following medications ordered: metoprolol succinate 100 mg daily, lisinopril 5 mg daily, verapamil ER 100 mg daily, and hydrochlorothiazide 25 mg daily. He has a history of hyperlipidemia, hypertension, and coronary artery disease. The patient asks the nurse, "Why do I have to take so many medications?"

1. What is the class and mechanism of action of each of these medications?
2. What is the nurse's best response to the patient's question?

Note: Answers to the Critical Thinking activities can be found in the "Answer Key" sections at the end of the book.

## Vasodilator

Hydralazine is an example of a direct vasodilator.

### Mechanism of Action

Hydralazine's direct mechanism of action is unknown, but it causes vasodilation via direct relaxation of

vascular smooth muscle. Peripheral vasodilation results in a reduction of blood pressure and decreased vascular resistance, resulting in increased cardiac output.

**Indications for Use**

Vasodilators are used to treat hypertension.

**Nursing Considerations Across the Lifespan**

Use with caution in patients with coronary artery disease, mitral valve rheumatic heart disease, and cerebral vascular accidents.

This medication should only be used in pregnancy if the benefits outweigh the risks due to lack of safety studies.

**Adverse/Side Effects**

Patients should be monitored for infection and are at risk of developing systemic lupus erythematosus (SLE). SLE is a chronic disease that causes inflammation in connective tissues. The signs and symptoms of SLE vary among affected individuals and can involve many organs and systems, including the skin, joints, kidneys, lungs, central nervous system, and blood-forming (hematopoietic) system. A characteristic sign of SLE is a flat, red rash across the cheeks and bridge of the nose. This rash is called a "butterfly rash" because of its shape.

Hypotension, palpitations, angina, tremors, numbness, tingling, disorientation, nasal congestion, headache, nausea, vomiting, and diarrhea are effects associated with hydralazine.


**Patient Teaching & Education**

Patients should remain compliant with the therapeutic dosing regimen, even if symptoms resolve. The patient should be cautious not to double up on medication doses. Additionally, the patient should consult the healthcare provider if two or more doses of medication are missed for follow-up instruction. Patients should be instructed to monitor their weight and assess for fluid retention in the feet and ankles. Additionally, the medication can cause side effects of orthostatic hypotension and drowsiness.
uCentral from Unbound Medicine. https://www.unboundmedicine.com/ucentral

Now let's take a closer look at the medication grid on hydralazine in Table 6.10e.


Table 6.10e Medication grid for Hydralazine

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Vasodilator** | hydralazine (Apresoline) | Monitor blood pressure | Reduce blood pressure | Systemic lupus erythematosus |

| | | | | |
|---|---|---|---|---|
| | | Obtain complete blood count (CBC) and antibody titers prior to beginning this medication<br><br>Report signs and symptoms of infection | | (SLE)<br><br>Hypotension, palpitations, and angina<br><br>Tremors, numbness, tingling, and disorientation<br><br>Nasal congestion<br><br>Headache, nausea, vomiting, and diarrhea |

# 6.11 Antilipemics

Open Resources for Nursing (Open RN)

Antilipemic agents reduce hyperlipidemia that may lead to additional health problems such as stroke, myocardial infarction, angina, and heart failure. Medications should be used in adjunct with a healthy diet and exercise regime approved by the patient's health care provider.

## Atorvastatin

### Mechanism of Action

Atorvastatin inhibits HMG-CoA reductase and cholesterol synthesis, which reduces LDL (low density lipoprotein).

### Indications for Use

This medication is used for hyperlipidemia and the prevention of cardiovascular disease.

### Nursing Considerations Across the Lifespan

Do not use with patients who have hepatic disease.

This medication is contraindicated with patients who are pregnant or breastfeeding. Do not give to patients under 10 years of age.

Use caution with geriatric patients due to increased risk for myopathy.

**Adverse/Side Effects**

Patients who are pregnant or breastfeeding should not take this medication. A health care provider will assess routine liver function for a patient taking atorvastatin. Nausea, diarrhea, dyspepsia, increase in blood glucose, rhabdomyolysis, myalgia, or muscle spasms may be produced by taking this medication. Rhabdomyolysis is a condition in which damaged skeletal muscle breaks down rapidly, causing muscle pain and weakness. Some of the muscle breakdown products are harmful to the kidneys and can cause kidney failure. There may be tea-colored urine or an irregular heartbeat with rhabdomyolysis.


**Patient Teaching & Education**

Patients should take the prescribed medication as directed and avoid consuming grapefruit juice during drug therapy. The medication should be used with dietary modifications. If the patient experiences muscle pain, tenderness, or weakness, these should be reported to the healthcare provider.


Now let's take a closer look at the medication grid on atrovastatin in Table 6.11a.


Table 6.11a Atorvastatin Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **HMG-CoA Reductase Inhibitors** | atorvastatin | Take at the same time each day, with or without food<br><br>Report muscle weakness, feeling tired, abdominal pain, or yellowing of skin or eyes | Reduce LDL | Rhabdomyolysis, myalgia, and muscle spasms<br><br>Abnormal liver enzymes<br><br>May increase blood glucose<br><br>Nausea, diarrhea, and dyspepsia |

## Ezetimibe

**Mechanism of Action**

Ezetimibe blocks the absorption of cholesterol in the small intestines to reduce LDL.

**Indications for Use**

This medication is used for treatment of hyperlipidemia and familial hypercholesterolemia.

**Nursing Considerations Across the Lifespan**

If medication is combined with HMG-CoA reductase inhibitors, do not give to pregnant or breastfeeding patients.

**Adverse/Side Effects**

Use with caution when ezetimibe is combined with additional medication. Patients may experience arthralgia, rhabdomyolysis, hepatic impairment, dizziness, upper respiratory infections, or diarrhea if they are taking this medication. Minimal side effects were reported with monotherapy.


**Patient Teaching & Education**

Patients should take the prescribed medication as directed and avoid consuming grapefruit juice during drug therapy. The medication should be used with dietary modifications. If the patient experiences muscle pain, tenderness, or weakness, this should be reported to the healthcare provider.


Now let's take a closer look at the medication grid for ezetimibe in Table 6.11b.


Table 6.11b Ezetimibe Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Cholesterol Absorption Inhibitor** | ezetimibe | Take at the same time each day, with or without food<br><br>Report muscle weakness, feeling tired, abdominal pain, or yellowing of skin or eyes | Reduce LDL | Arthralgia, rhabdomyolysis<br><br>Hepatic impairment<br><br>Dizziness<br><br>Upper respiratory infection<br><br>Diarrhea |

# 6.12 Blood Coagulation Modifiers

Open Resources for Nursing (Open RN)

This section will discuss medications that affect blood coagulation and includes several types of medications including anticoagulants, antiplatelets, and thrombolytics, as well as their associated reversal agents.

**Anticoagulants** prevent the formation of a clot by inhibiting certain types of clotting factors. Anticoagulants include the following drug classes: heparins or unfractionated heparin and low molecular weight heparin (LMWH), warfarin (Coumadin), selective factor Xa inhibitors (rivaroxaban), and direct thrombin inhibitors (dabigatran). Antiplatelets include aspirin and other aggregation inhibitors such as clopidogrel, and thrombolytics include alteplase (tPA). All these types of medications are included on the List of High Alert Medications (HAMs) by the Institute for Safe Medication Practices (ISMP) that require special safeguards to reduce the risk of errors or adverse effects.
Institute for Safe Medication Practices (ISMP). (2018). *ISMP List of High-Alert Medications in Acute Care Settings.* https://www.ismp.org/sites/default/files/attachments/2018-08/highAlert2018-Acute-Final.pdf.

The most common anticoagulant errors in acute hospital settings are administration mistakes, including incorrect dosage calculation and infusion rates. The Health Research and Educational Trust focuses on reducing harm related to HAMs by 50% and recommends the following interventions to achieve this goal:

- Educate staff based on evidence and best practices.
- Use standardized order sets and protocols.
- Perform medication reconciliation at all transitions.

Specific interventions regarding anticoagulant therapy include standardization of protocols for withholding and restarting warfarin perioperatively, including pharmacists on rounds to provide decision support for staff administering HAMs and to reduce prescribing errors, pharmacist monitoring of anticoagulants, and pharmacist notification when rescue medications are given.
Anderson, P. & Townsend, T. (2015) Preventing high-alert medication errors in hospital patients. *Nurse Today, 10*(5). https://www.americannursetoday.com/wp-content/uploads/2015/05/ant5-CE-421.pdf

According to the *Institute for Safe Medication Practices (ISMP) 2016 Annual Report*, there is also a high risk of acute injuries for patients taking anticoagulants outside of the hospital setting. Anticoagulants are commonly used by the elderly to reduce the risk of ischemic stroke, with an estimated 3.8 million people taking oral anticoagulants in 2016. CDC data show that adverse effects of oral anticoagulants account for more emergency department visits than any other class of drugs. Adverse effects range from gastrointestinal bleeding to cerebral hemorrhages, resulting in over 3,000 deaths in 2016.
Institute for Safe Medication Practices (ISMP). (2017). Q*uarterWatch™ (2016 Annual Report) Part II: Oral Anticoagulants—The Nation's Top Risk of Acute Injury from Drugs.* https://www.ismp.org/resources/quarterwatchtm-2016-annual-report-part-ii-oral-anticoagulants-nations-top-risk-acute.

Since 1954, warfarin has been a standard but hazardous treatment for preventing blood clots. Warfarin requires close laboratory monitoring and individual dose adjustments based on PT and INR lab results.

When the pharmaceutical industry began marketing modern replacements for warfarin, including dabigatran (Pradaxa), rivaroxaban (Xarelto), and apixaban (Eliquis), they designed them to be easier to use than warfarin because no laboratory monitoring was required, but not necessarily safer. It is vital for nurses to provide thorough patient and caregiver education for patients prescribed anticoagulants at home. Suggested patient education topics are included for each type of medication below.

## Heparin Sodium

Heparin sodium is an anticoagulant that can be injected or used intravenously and is formulated in several dosages. (See Figure 6.25.)



Due to heparin being a high-alert medication, hospitals use several processes for storing and labeling the medication to help prevent errors. It is also important to note that there is a type of heparin flush often referred to as "Hep-Lock" that is used to maintain the patency of central lines. The dosage of heparin in heparin IV flushes is much different than the heparin dose used as an intravenous medication to prevent or treat a blood clot.

Most hospitals have weight-based protocols for IV heparin administration that titrate a patient's dosage to be within a therapeutic range based on the results of a lab test called **Partial thromboplastin time (PTT)**. PTT is a blood test that looks at how long it takes for blood to clot. Patients receiving heparin subcutaneous injections to prevent DVTs (deep vein thrombosis) do not require PTT monitoring.


Figure 6.25 Heparin comes in many dosages, and overdose can be deadly, so it is important for the nurse to use safeguards to prevent potential medication errors

### Mechanism of Action

Heparin inhibits the activated coagulation factors involved in the clotting sequence, particularly Xa and

IIa. Heparin also prevents the formation of a stable fibrin clot by inhibiting the activation of the fibrin stabilizing factor. Heparin does not have fibrinolytic activity; therefore, it will not breakdown existing clots.

**Indications for Use**

IV heparin is commonly indicated for the treatment of deep venous thromboembolism (DVT) or pulmonary embolism. It is also indicated for use during an acute myocardial infarction. Subcutaneous heparin is commonly indicated to prevent DVT or embolization caused by atrial fibrillation. Heparin IV flushes ("Hep-Locks") are used to maintain the patency of central IV lines.

**Nursing Considerations Across the Lifespan**

When bleeding requires the reversal of heparinization, protamine sulfate by slow infusion will neutralize heparin sodium.

A higher incidence of bleeding has been reported in patients over 60 years of age, especially women.

Fatal hemorrhages have occurred due to medication errors. Carefully examine all heparin products to confirm the correct dose prior to the administration of the drug.

IV heparin therapy requires close monitoring of frequent partial thromboplastin time (PTT) results to ensure dosage is in therapeutic range and to reduce the risk of overdose with associated bleeding. Dosage is considered adequate when the activated partial thromboplastin time (APTT) is 1.5 to 2 times the normal or when the whole blood clotting time is elevated approximately 2.5 to 3 times the control value.

This drug is contraindicated in patients with a history of Heparin-Induced Thrombocytopenia (HIT) and Heparin-Induced Thrombocytopenia and Thrombosis (HITT). HIT is a condition where platelets drop 30% or more below a patient's baseline after heparin is administered and can lead to HITT where thrombi are formed.

Use with caution with medication that affects the coagulation cascade due to additive effects that increase the risk of bleeding. When a patient is receiving IV heparin therapy to treat a blood clot, it may be overlapped with oral warfarin to establish anticoagulation therapy after discharge. See more information about this process under the "Warfarin" section.

**Adverse/Side Effects**

There is a high risk of bleeding that can lead to hemorrhaging. Notify prescribing provider immediately of new signs of bleeding or bruising or sudden changes in vital signs that indicate internal bleeding, such as decreasing blood pressure with an associated increase in heart rate.

Some patients may develop Heparin-Induced Thrombocytopenia (HIT) or Heparin-Induced Thrombocytopenia and Thrombosis (HITT); therefore, heparin should be immediately discontinued.

**Patient Teaching & Education**

Notify health care staff immediately of new signs of bleeding or bruising. Remind physicians and

dentists that they are receiving heparin before any surgery or invasive procedure is scheduled.

Patients should avoid medications containing aspirin or NSAIDS. Bleeding precautions should be taken, including the avoidance of IM injections, use of a soft toothbrush, and elective razor.


Now let's take a closer look at the medication grid for heparin in Table 6.12a.


Table 6.12a Heparin Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Anticoagulant** | heparin | Injection (subq) or IV<br><br>Carefully examine all heparin products to confirm the correct choice prior to administration<br><br>Closely monitor PTT levels in IV therapy to ensure in therapeutic range<br><br>Protamine sulfate is the reversal agent | Prevent or treat clots | High risk of bleeding<br><br>Risk of gastrointestinal or cerebral hemorrhage, especially in elderly<br><br>Risk of Heparin-Induced Thrombocytopenia (HIT) and Heparin-Induced Thrombocytopenia and Thrombosis (HITT) |

## Low Molecular Weight Heparin (LMWH)

Enoxaparin (Lovenox) is a low molecular weight heparin (LMWH) that is supplied in a prefilled syringe (see Figure 6.26).

LMWH heparin formulations do not require lab monitoring.

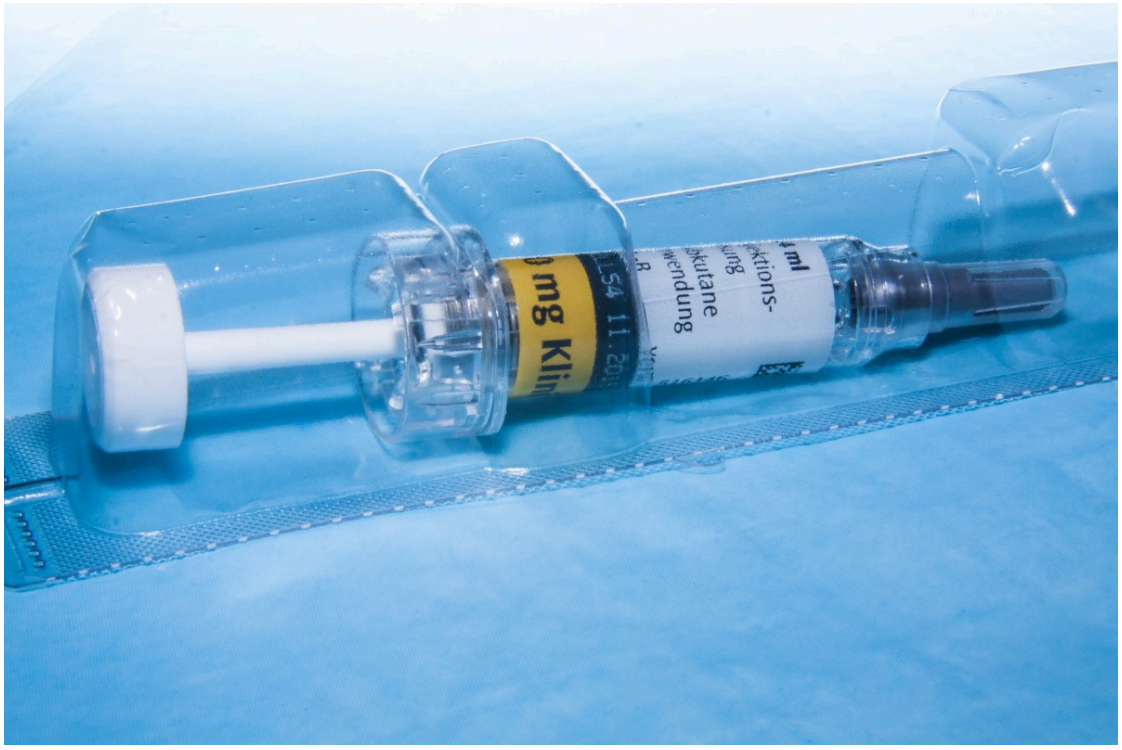

Figure 6.26 Enoxaparin in a prefilled syringe

**Mechanism of Action**

Enoxaparin is a low molecular weight heparin, which has antithrombotic properties with a higher ratio of anti-Factor Xa to anti-Factor IIa activity compared to heparin.

**Indications for Use**

It is indicated for the prevention and treatment of deep vein thrombosis (DVT), which may lead to pulmonary embolism (PE). It is also used in combination with aspirin for the treatment of acute myocardial infarction.

**Nursing Considerations Across the Lifespan**

Enoxaparin is administered subcutaneously and preferably in the abdomen for best absorption.

Safety and effectiveness have not been established in pediatric patients. The risk of bleeding increases with age, especially if used concurrently with antiplatelet medications.

Use with caution in patients with renal impairment; risk of bleeding is increased. A dosage adjustment is recommended for patients with severe renal impairment.

Overdosage can be neutralized with a slow IV infusion of protamine sulfate.

**Adverse/Side Effects**

Black Box Warning: Epidural or spinal hematomas may occur in patients who are anticoagulated with low molecular weight heparins (LMWH) and are receiving neuraxial anesthesia or undergoing spinal

puncture. These hematomas may result in long-term or permanent paralysis.

There is a risk of bleeding and hemorrhaging, especially following percutaneous coronary revascularization procedures or with concurrent medication conditions such as recent GI ulcer. It may cause Heparin-Induced Thrombocytopenia (HIT) or Heparin-Induced Thrombocytopenia with Thrombosis (HITT).


**Patient Teaching & Education**

Notify health care staff immediately of new signs of bleeding or bruising. Remind physicians and dentists that they are receiving heparin before any surgery or invasive procedure is scheduled.

Patients should avoid medications containing aspirin or NSAIDS.


Now let's take a closer look at the medication grid on enoxaparin in Table 6.12b.


Table 6.12b Enoxaparin Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Anticoagulant** | enoxaparin | Use with caution in patients with kidney disease.<br><br>If used for a patient undergoing neuraxial anesthesia or a spinal puncture, monitor frequently for neurological impairment. If neurological compromise is noted, urgent treatment is necessary | Prevention or treatment of DVT or PE | Bleeding<br><br>Risk of hemorrhage<br><br>Thrombocytopenia, HIT, or HITT |



## Warfarin

Warfarin (Coumadin) is an oral anticoagulant formulated in various strengths in different colors to help prevent errors when patients self-administer different dosages at home (see Figure 6.27

). Close monitoring of **prothrombin time (PT)** or **international normalized ratio (INR)** is required.

Figure 6.27 Warfarin is an oral pill with various strengths in different colors

**Mechanism of Action**

Warfarin acts by inhibiting the synthesis of vitamin K-dependent clotting factors, which include Factors II, VII, IX, and X and the anticoagulant proteins C and S.

**Indications for Use**

Warfarin is indicated for the following:

- Prophylaxis and treatment of venous thrombosis and its extension, pulmonary embolism (PE).
- Prophylaxis and treatment of thromboembolic complications associated with atrial fibrillation (AF) and/or cardiac valve replacement.
- Reduction in the risk of death, recurrent myocardial infarction (MI), and thromboembolic events such as stroke or systemic embolization after myocardial infarction.

**Nursing Considerations Across the Lifespan**

Warfarin is contraindicated in pregnant women except for those with mechanical heart valves; it can cause fetal harm.

Vitamin K is the reversal agent. Fresh frozen plasma may be considered if the requirement to reverse the effects of warfarin sodium is urgent.

Close monitoring of prothrombin time (PT) or international normalized ratio (INR) is required. Therapeutic INR ranges from 2.0 to 3.5, depending on the indication.

In hospitalized patients receiving heparin therapy, there is often a period of overlap where the patient is prescribed both IV heparin and warfarin until the INR reaches therapeutic range. At that point, the IV heparin is discontinued.

Warfarin has significant interactions with many medications; read drug label information before

administering.

Warfarin sodium is contraindicated in patients with many conditions, including, but not limited to:

- Hemorrhagic tendencies or blood dyscrasias
- Recent or contemplated surgery of the central nervous system or eye, or traumatic surgery resulting in large open surfaces

Bleeding tendencies associated with:

- Active ulceration or overt bleeding of the gastrointestinal, genitourinary, or respiratory tracts
- Central nervous system hemorrhage
- Cerebral aneurysms and dissecting aorta
- Pericarditis and pericardial effusions
- Bacterial endocarditis

**Adverse/Side Effects**

- Black Box Warnings: Warfarin can cause major or fatal bleeding. Perform regular monitoring of INR in all treated patients. Drugs, dietary changes, and other factors affect INR levels achieved with warfarin therapy. Instruct patients about prevention measures to minimize risk of bleeding and to report signs and symptoms of bleeding. Warfarin can cause acute kidney injury and bleeding risks are increased in patients with liver disease.

**Patient Education**

Advise patients to:

- Avoid alcohol, cranberries, and grapefruit as they increase the effect of warfarin and the risk for bleeding.
- Strictly adhere to the prescribed dosage schedule.
- Follow INR monitoring guidelines as provided by the prescriber.
- Avoid any activity or sport that may result in traumatic injury.
- Tell their provider if they experience frequent falls because warfarin can increase their risk for bleeding in the brain.
- Eat a normal, balanced diet to maintain a consistent intake of vitamin K (such as green, leafy vegetables).
- Tell all health care professionals and dentists that they are taking warfarin, especially before surgery or dental procedures.
- Use electric razors instead of straight razors.
- Carry identification stating that they are taking warfarin.
- Notify their provider immediately if any unusual bleeding or symptoms occur, such as pain, swelling or discomfort, prolonged bleeding from cuts, increased menstrual flow or vaginal bleeding, nosebleeds, bleeding of gums from brushing, unusual bleeding or bruising, red or dark brown urine, red or tar black stools, headache, dizziness, or weakness.



Now let's take a closer look at the medication grid on warfarin in Table 6.12c.

Table 6.12c Warfarin Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Anticoagulant** | warfarin | Oral route<br><br>Vitamin K is the antidote<br><br>Monitor INR results before administering medication<br><br>Use with caution in patients with liver disease | Prevent DVT or PE | Bleeding<br><br>Hemorrhage |

# Critical Thinking Activity 6.12
# Image of lightbulb in a circle

A patient who was treated in the hospital for DVT in his left leg has been prescribed warfarin.

1. The patient asks, “Will the warfarin dissolve the clot in my leg?” What is the nurse’s best response?

The nurse plans to assess the patient’s lab work before administering the warfarin.

2. What blood test(s) are important to monitor for patients taking warfarin, and what is the therapeutic range?

The nurse knows that the patient will need to monitor his diet when taking warfarin.

3. What dietary instructions should be provided to the patient?

The nurse plans to provide patient education regarding this newly prescribed medication.

4. Outline the topics to cover with this high-risk medication.

5. What is the reversal agent for warfarin?

Note: Answers to the Critical Thinking activities can be found in the "Answer Key" sections at the end of the book.

## Rivaroxaban

Rivaroxaban (Xarelto) is a selective Xa inhibitor.

### Mechanism of Action

Rivaroxaban is a selective inhibitor of factor Xa and indirectly inhibits platelet aggregation induced by thrombin.

### Indications for Use

Rivaroxaban is indicated for prevention or treatment of DVT and PE. In combination with aspirin, it is indicated to reduce the risk of major cardiovascular events such as cardiovascular (CV) death, myocardial infarction (MI) and stroke and in patients with chronic coronary artery disease (CAD) or peripheral artery disease (PAD).

### Nursing Considerations Across the Lifespan

For overdose, activated charcoal can be used to reduce absorption and Andexxa is a reversal agent.

Avoid in patients with moderate to severe liver impairment. Report any unusual bleeding or bruising.

### Adverse/Side Effects

Black Box Warning: Epidural or spinal hematomas may occur in patients who are anticoagulated with rivaroxaban and are receiving neuraxial anesthesia or undergoing spinal puncture. These hematomas may result in long-term or permanent paralysis.

Risk of bleeding can be fatal.


### Patient Teaching & Education

Patients should report any signs of unusual bleeding or bruising to the healthcare provider. The patient should also notify the provider of all prescriptions, OTC medications, vitamins, and herbal products.


Now let's take a closer look at the medication grid on rivaroxaban in Table 6.12d.


Table 6.12d Rivaroxaban Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Selective Xa Inhibitors** | rivaroxaban | Activated charcoal or Andexxa can be used in overdose<br><br>Avoid in patients with liver impairment | Prevent DVT and PE and and risk of severe cardiovascular events | Bleeding<br><br>Epidural or spinal hematomas if neuraxial anesthesia or spinal puncture |

## Dabigatran

Dabigatran (Pradaxa) is a direct-acting thrombin inhibitor.

### Mechanism of Action

Dabigatran is a competitive, direct thrombin inhibitor. Because thrombin enables the conversion of fibrinogen into fibrin during the coagulation cascade, its inhibition prevents the development of a thrombus.

### Indications for Use

This drug is used to prevent or treat deep vein thromboses (DVT) or pulmonary emboli (PE).

### Nursing Considerations Across the Lifespan

Overdose: Idarucizumab, a specific reversal agent, is available.

Safety and effectiveness in pediatric patients have not been established.

### Adverse/Side Effects

Black Box Warning: Epidural or spinal hematomas may occur in patients who are anticoagulated with dabigatran and are receiving neuraxial anesthesia or undergoing spinal puncture. These hematomas may result in long-term or permanent paralysis**.**

Risk of bleeding can be fatal.



### Patient Teaching & Education

Patients should report any signs of unusual bleeding or bruising to the healthcare provider. Additionally, dabigatran bottles should be disposed of four months after opening. The patient should also notify the provider of all prescriptions, OTC medications, vitamins, and herbal products.
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Now let's take a closer look at the medication grid for dabigatran in Table 6.12e.
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Table 6.12e Dabigatran Medication Grid

| Class/ Subclass | Prototype-generic | Administration Considerations | Therapeutic Effects | Adverse/Side Effects |
|---|---|---|---|---|
| **Direct-acting thrombin inhibitors** | dabigatran | Idarucizumab is a specific reversal agent | Prevent or treat DVT or PE | Risk of bleeding that can be fatal<br><br>Epidural or spinal hematomas if receiving neuraxial anesthesia or undergoing spinal puncture |

## Antiplatelets

Acetylsalicylic acid (aspirin) and clopidogrel (Plavix) are antiplatelet medications.

During an active myocardial infarction (heart attack), chewable aspirins are used due to their rapid absorption (see Figure 6.28
"Bayer Aspirin Low Dose" by Mike Mozart is licensed under CC BY 2.0
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