Circulatory System: The Heart

- Overview of Cardiovascular System
- Gross Anatomy of the Heart
- Cardiac Conduction System and Cardiac Muscle
- Electrical and Contractile Activity of Heart
- Blood Flow, Heart Sounds, and Cardiac Cycle
- Cardiac Output

Circulatory System: The Heart

- **cardiology** – the scientific study of the heart and the treatment of its disorders
- **cardiovascular system**
  - heart and blood vessels
- **circulatory system**
  - heart, blood vessels, and the blood
- major divisions of circulatory system
  - **pulmonary circuit** - right side of heart
    - carries blood to lungs for gas exchange and back to heart
  - **systemic circuit** - left side of heart
    - supplies oxygenated blood to all tissues of the body and returns it to the heart

Cardiovascular System Circuit

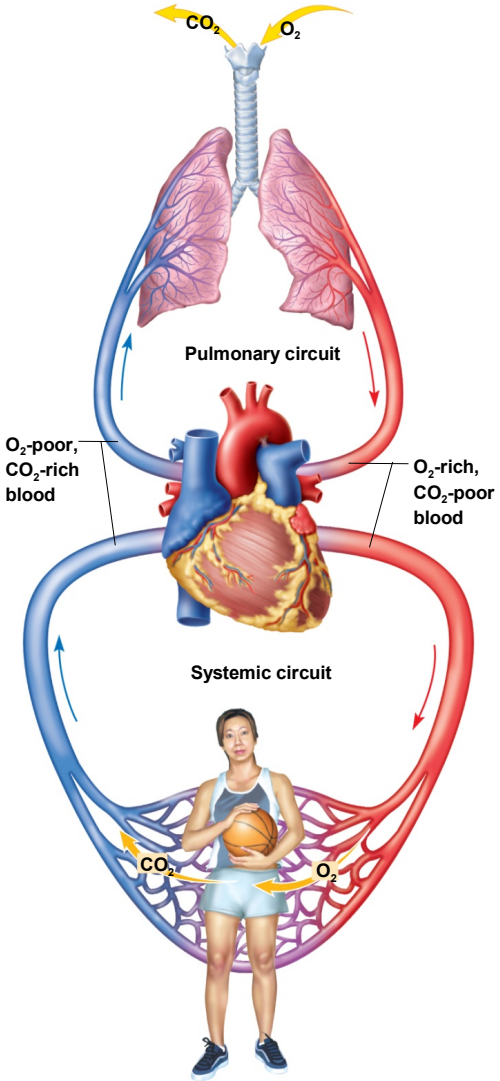


Figure 19.1

- left side of heart
  - fully oxygenated blood arrives from lungs via pulmonary veins
  - blood sent to all organs of the body via aorta
- right side of heart
  - lesser oxygenated blood arrives from inferior and superior vena cava
  - blood sent to lungs via pulmonary trunk

Position, Size, and Shape

- heart located in mediastinum, between lungs
- **base** – wide, superior portion of heart, blood vessels attach here
- **apex** - inferior end, tilts to the left, tapers to point
- 3.5 in. wide at base, 5 in. from base to apex and 2.5 in. anterior to posterior; weighs 10 oz.

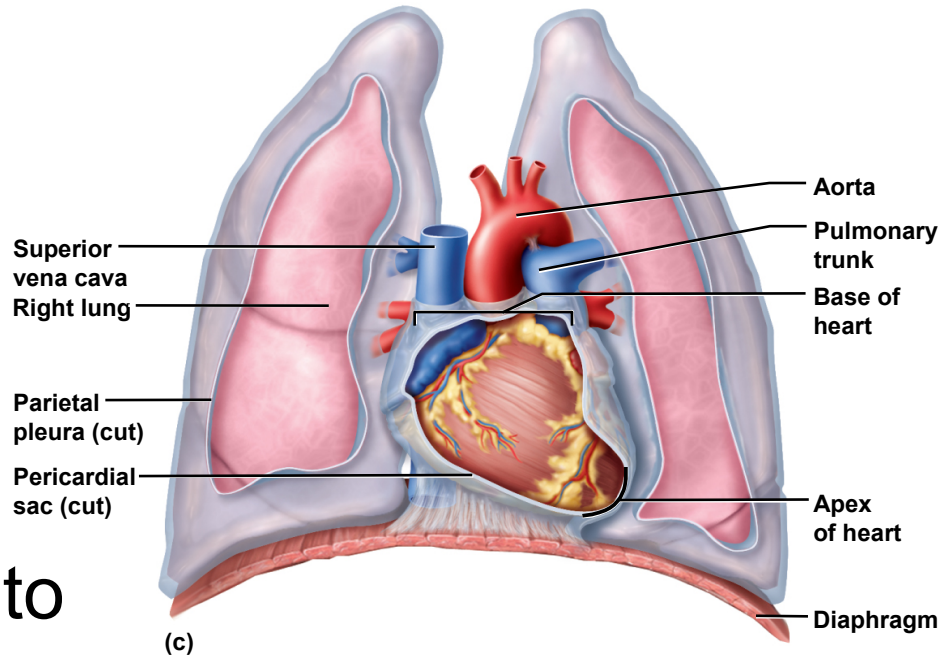


Figure 19.2c

Heart Position

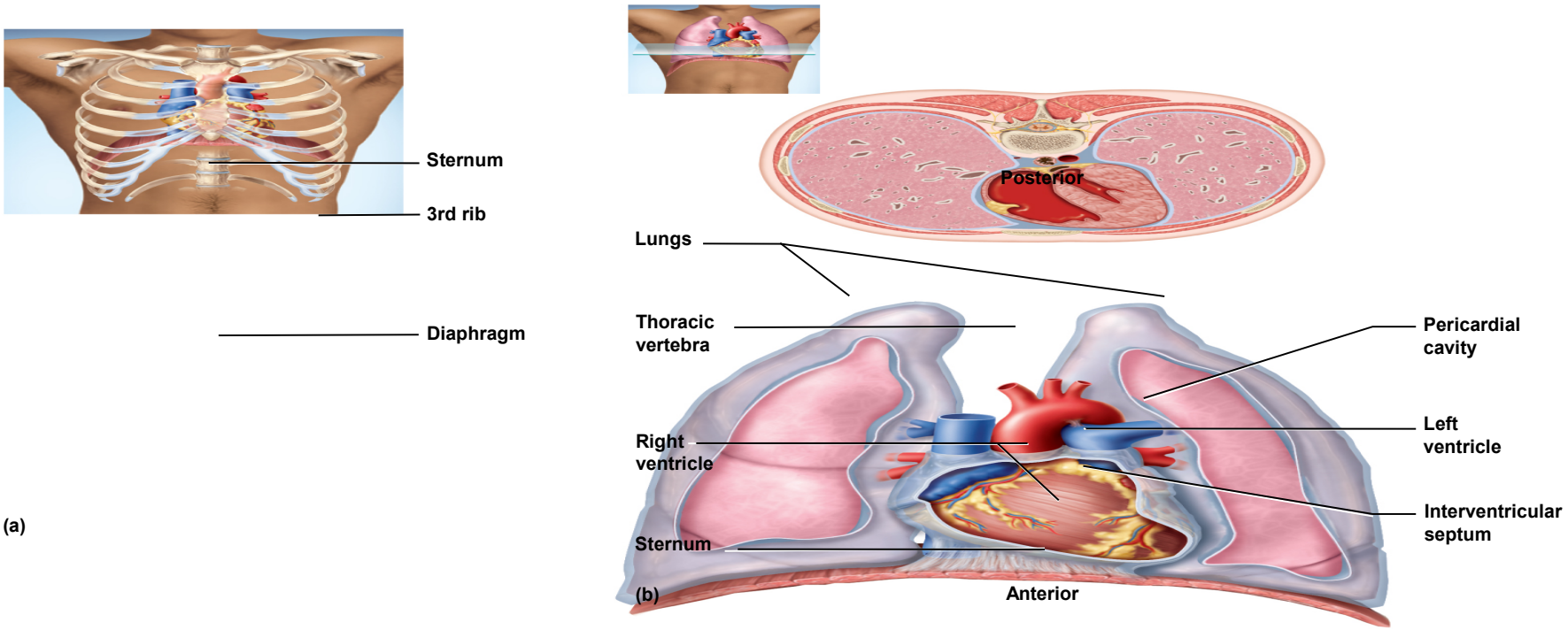


Figure 19.2 a-b

Pericardium

- **pericardium** - double-walled sac (pericardial sac) that encloses the heart
  - allows heart to beat without friction, provides room to expand, yet resists excessive expansion
  - anchored to diaphragm inferiorly and sternum anteriorly
- **parietal pericardium** – outer wall of sac
  - superficial **fibrous layer** of connective tissue
  - a deep, thin **serous layer**
- **visceral pericardium (epicardium)** – heart covering
  - serous lining of sac turns inward at base of heart to cover the heart surface
- **pericardial cavity** - space inside the pericardial sac filled with 5 - 30 mL of pericardial fluid
- **pericarditis** – inflammation of the membranes
  - painful friction rub with each heartbeat

Pericardium and Heart Wall

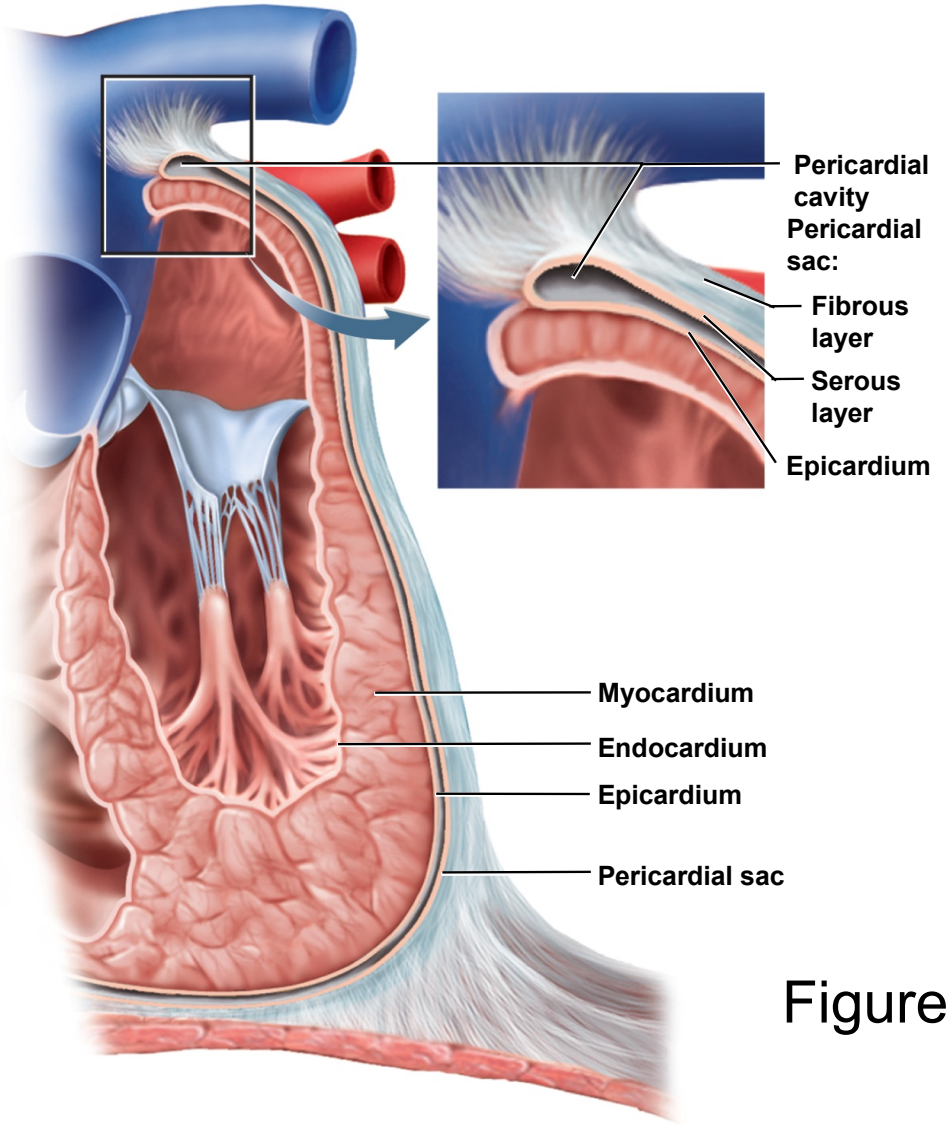
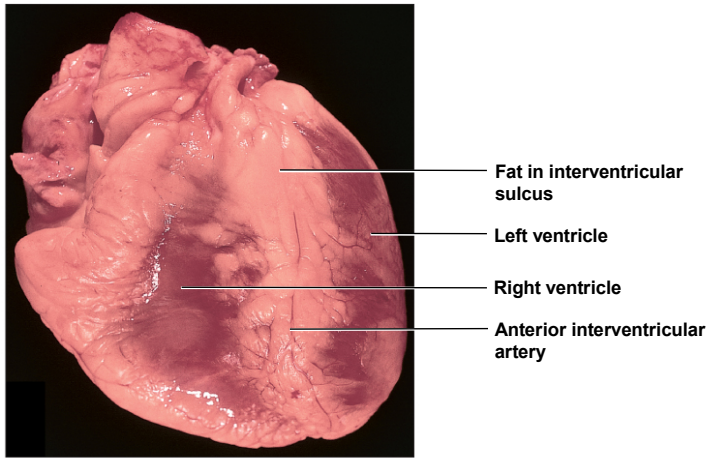
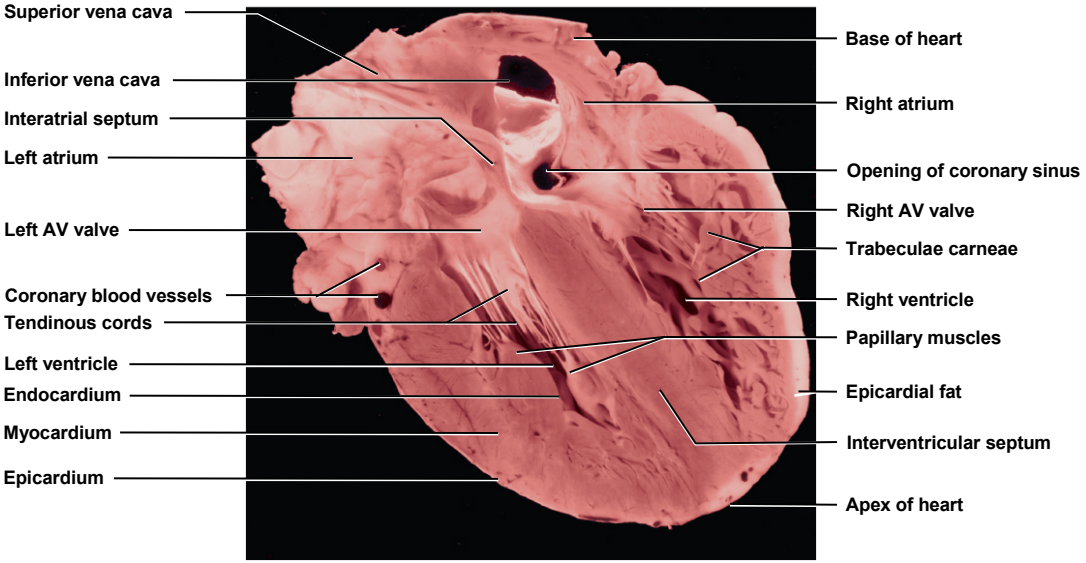


Figure 19.3

Cadaver Heart



(a) Anterior view, external anatomy



(b) Posterior view, internal anatomy

Figure 19.4 a-b

Heart Wall

- **epicardium** (visceral pericardium)
  - serous membrane covering heart
  - adipose in thick layer in some places
  - coronary blood vessels travel through this layer
- **endocardium**
  - smooth inner lining of heart and blood vessels
  - covers the valve surfaces and continuous with endothelium of blood vessels
- **myocardium**
  - layer of **cardiac muscle** proportional to work load
    - muscle spirals around heart which produces wringing motion
  - **fibrous skeleton of the heart -** framework of **collagenous and elastic fibers**
    - provides structural support and attachment for cardiac muscle and anchor for valve tissue
    - electrical insulation between atria and ventricles important in timing and coordination of contractile activity

Heart Chambers

- **four chambers**
  - **right and left atria**
    - two superior chambers
    - receive blood returning to heart
    - auricles (seen on surface) enlarge chamber
  - **right and left ventricles**
    - two inferior chambers
    - pump blood into arteries

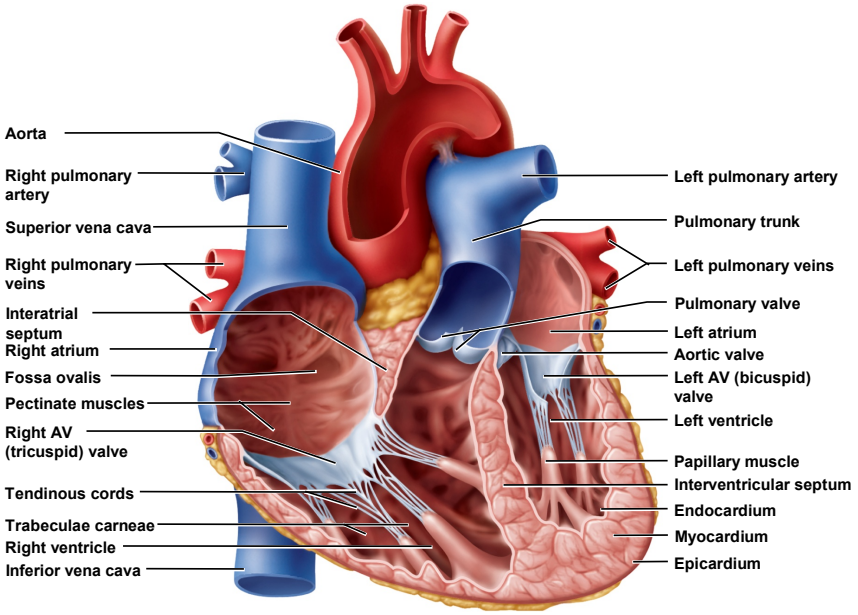
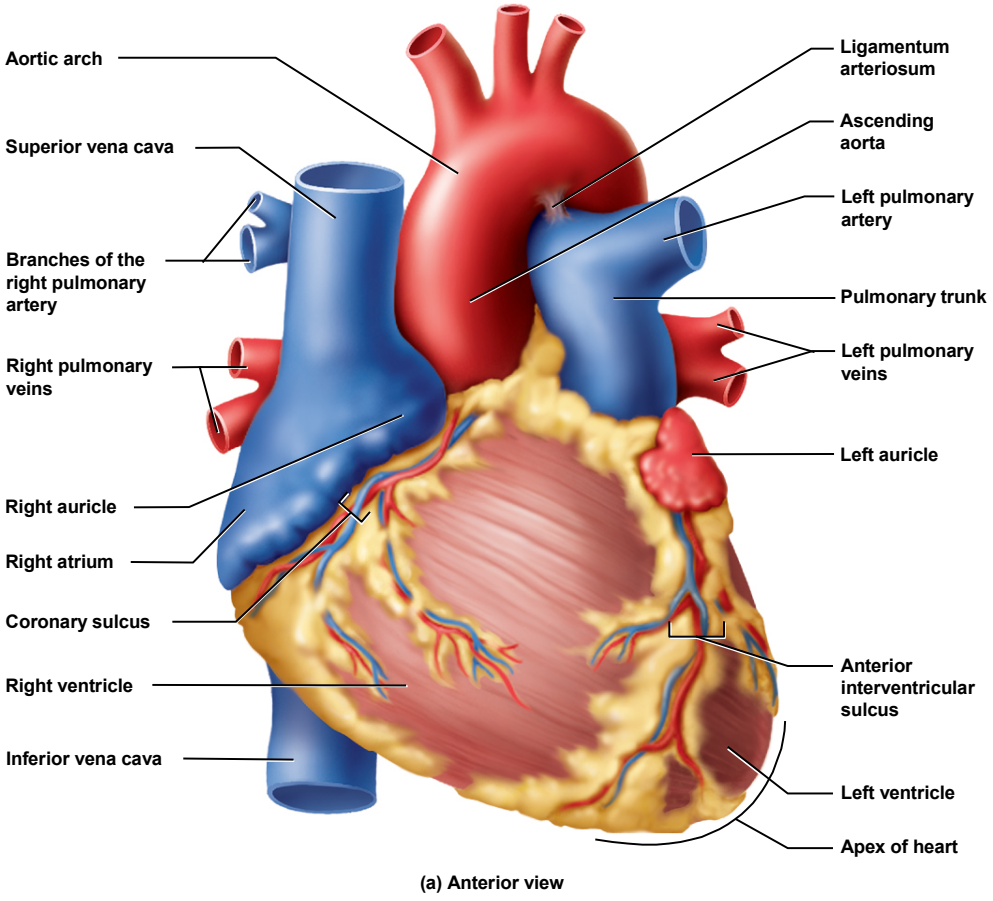


Figure 19.7

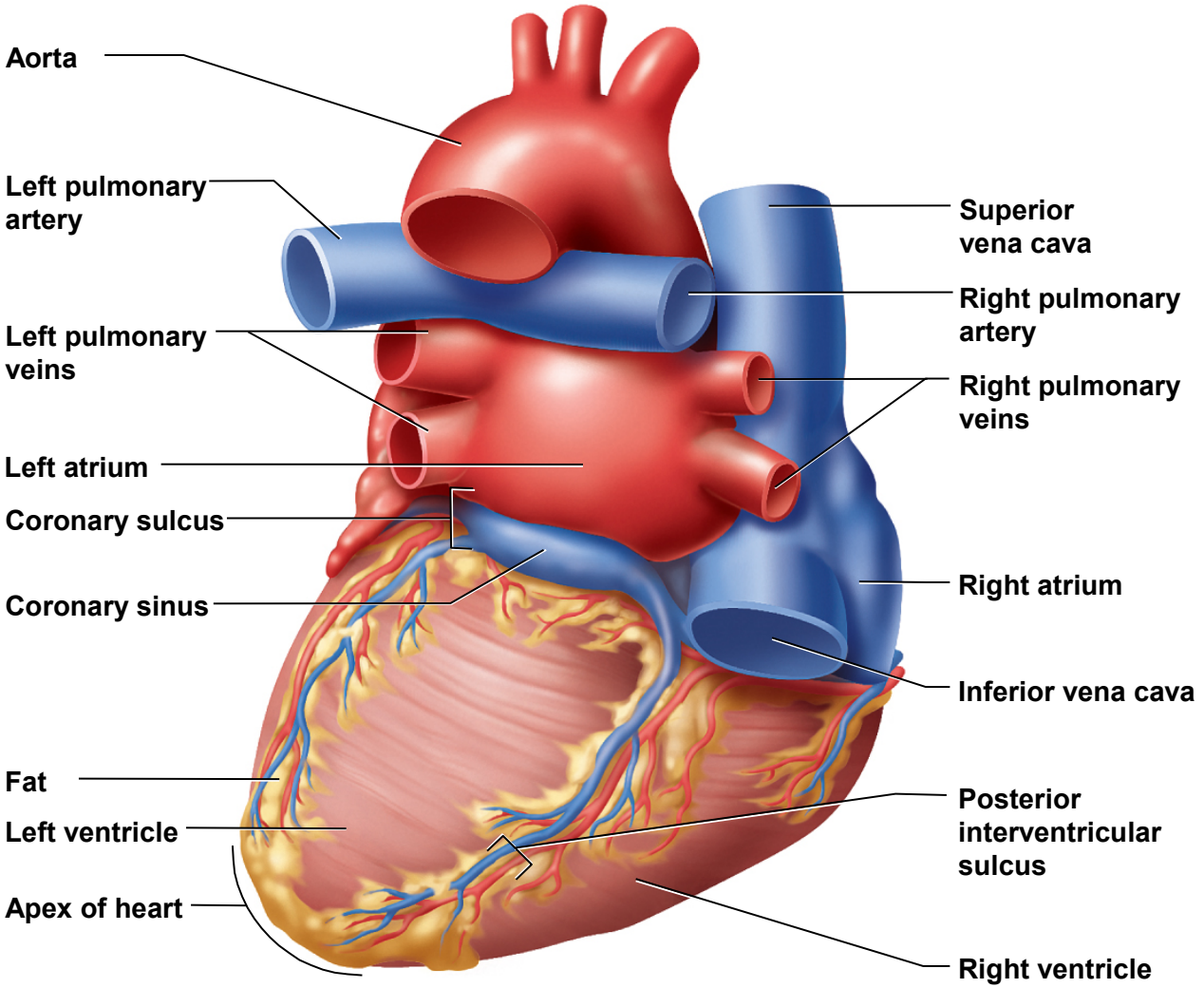
External Anatomy - Anterior



- **atrioventricular sulcus** - separates atria and ventricles
- **interventricular sulcus** - overlies the interventricular septum that divides the right ventricle from the left
- sulci contain coronary arteries

Figure 19.5a

External Anatomy - Posterior



(b) Posterior view

Figure 19.5b

Heart Chambers - Internal

- **interatrial septum**
  - wall that separates atria
- **pectinate muscles**
  - internal ridges of myocardium in right atrium and both auricles
- **interventricular septum**
  - muscular wall that separates ventricles
- **trabeculae carneae**
  - internal ridges in both ventricles

Internal Anatomy - Anterior

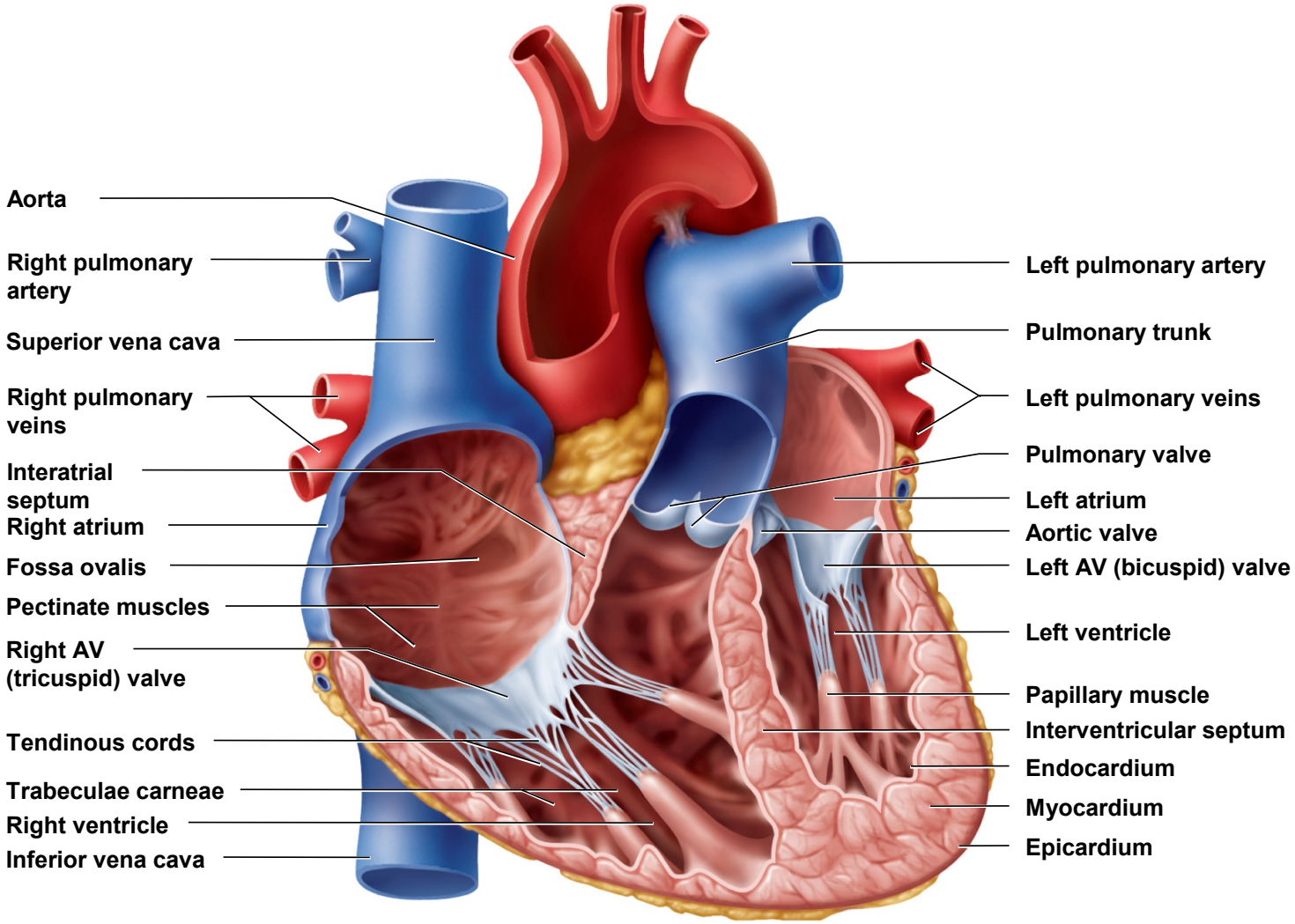
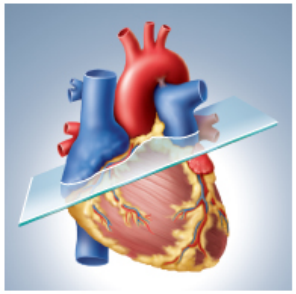


Figure 19.7

Heart Valves

- valves ensure a one-way flow of blood through the heart
- **atrioventricular (AV) valves** – controls blood flow between atria and ventricles
  - **right AV valve** has 3 cusps (**tricuspid valve**)
  - **left AV valve** has 2 cusps (**mitral or bicuspid valve**)
  - **chordae tendineae** - cords connect AV valves to **papillary muscles** on floor of ventricles
    - prevent AV valves from flipping inside out or bulging into the atria when the ventricles contract
- **semilunar valves** - control flow into great arteries – open and close because of blood flow and pressure
  - **pulmonary semilunar valve** - in opening between right ventricle and pulmonary trunk
  - **aortic semilunar valve** in opening between left ventricle and aorta

Heart Valves



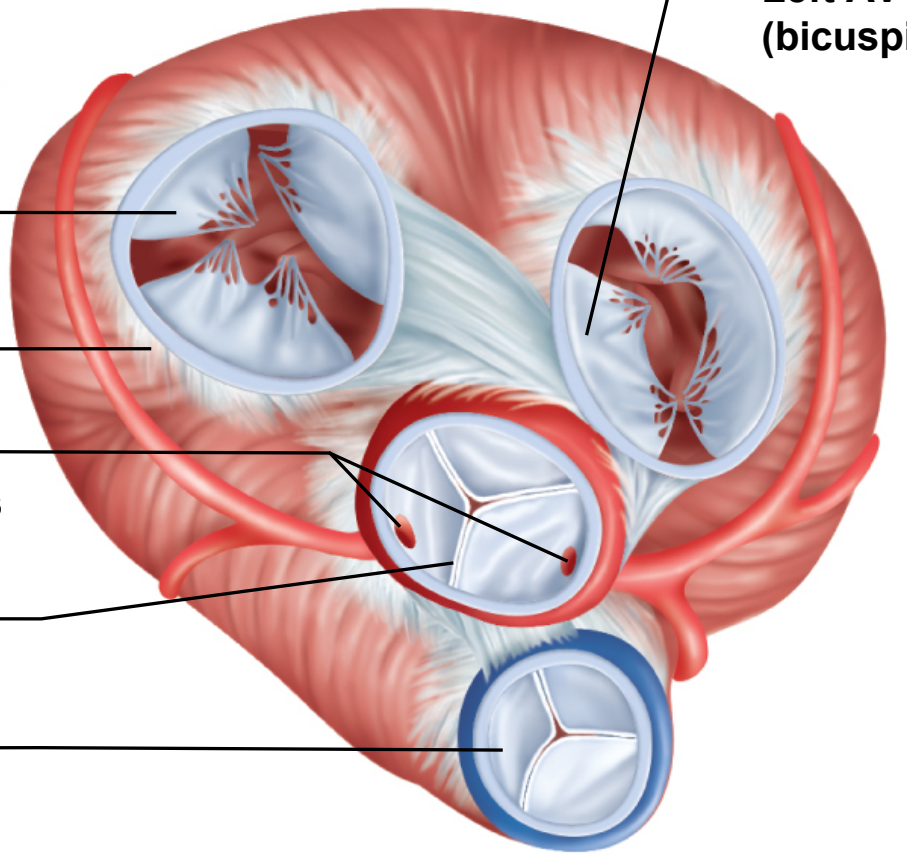
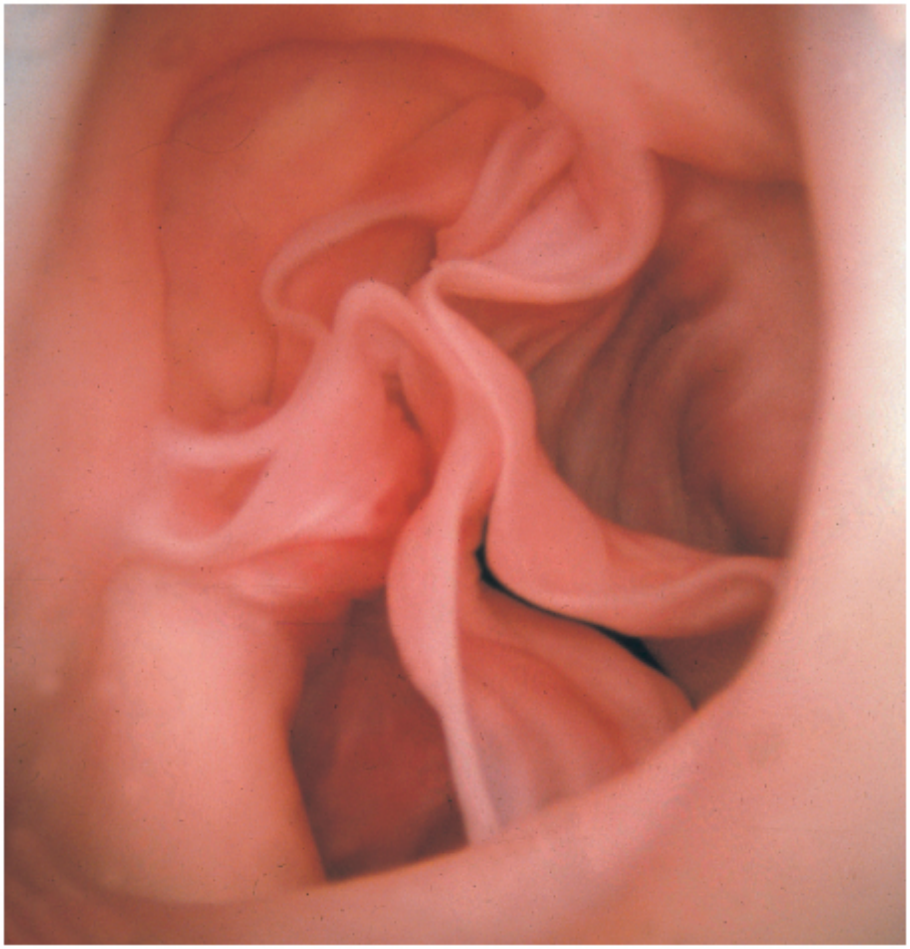


Figure 19.8a

(a)

Endoscopic View of Heart Valve



(b)

Figure 19.8b

Heart Valves

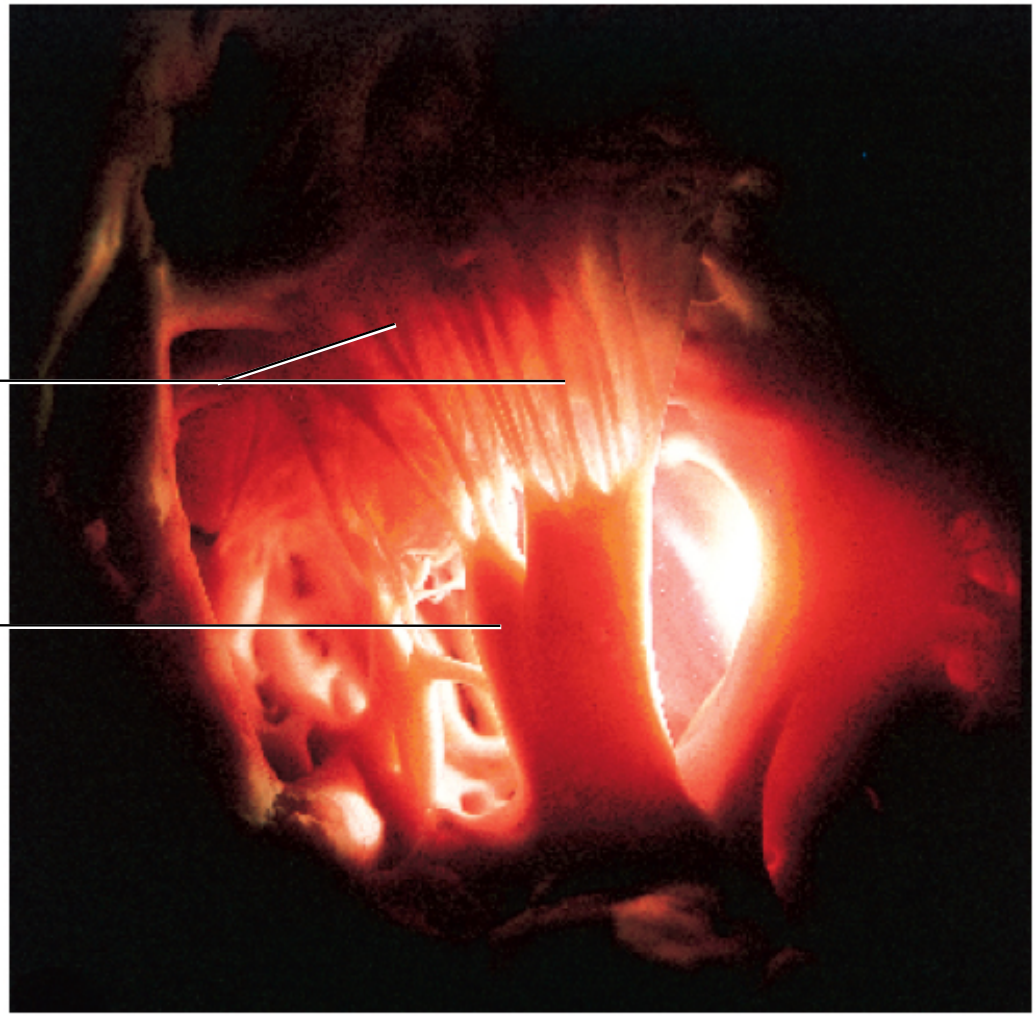


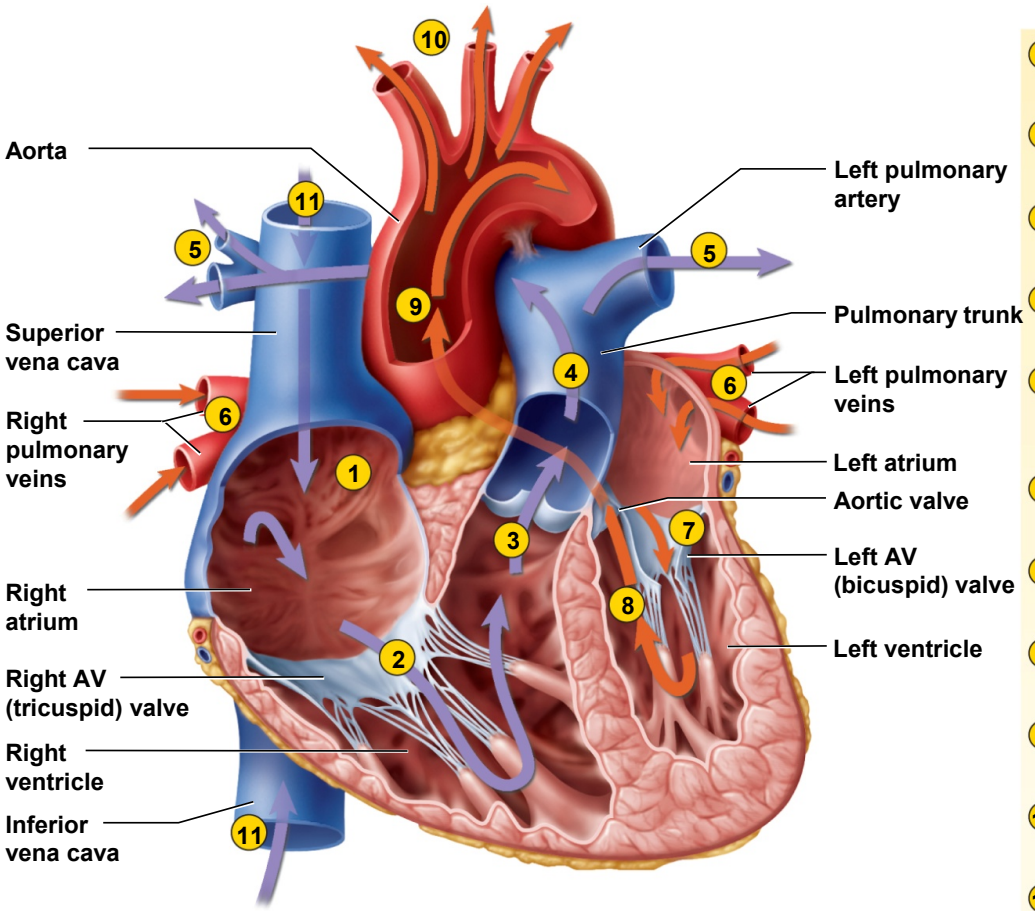
Figure 19.8c

(c)

AV Valve Mechanics

- ventricles relax
  - pressure drops inside the ventricles
  - semilunar valves close as blood attempts to back up into the ventricles from the vessels
  - AV valves open
  - blood flows from atria to ventricles
- ventricles contract
  - AV valves close as blood attempts to back up into the atria
  - pressure rises inside of the ventricles
  - semilunar valves open and blood flows into great vessels

Blood Flow Through Heart



1. Blood enters right atrium from superior and inferior venae cavae.
2. Blood in right atrium flows through right AV valve into right ventricle.
3. Contraction of right ventricle forces pulmonary valve open.
4. Blood flows through pulmonary valve into pulmonary trunk.
5. Blood is distributed by right and left pulmonary arteries to the lungs, where it unloads CO_2 and loads O_2.
6. Blood returns from lungs via pulmonary veins to left atrium.
7. Blood in left atrium flows through left AV valve into left ventricle.
8. Contraction of left ventricle (simultaneous with step 3) forces aortic valve open.
9. Blood flows through aortic valve into ascending aorta.
10. Blood in aorta is distributed to every organ in the body, where it unloads O_2 and loads CO_2.
11. Blood returns to heart via venae cavae.

Figure 19.9

blood pathway travels from the right atrium through the body and back to the starting point

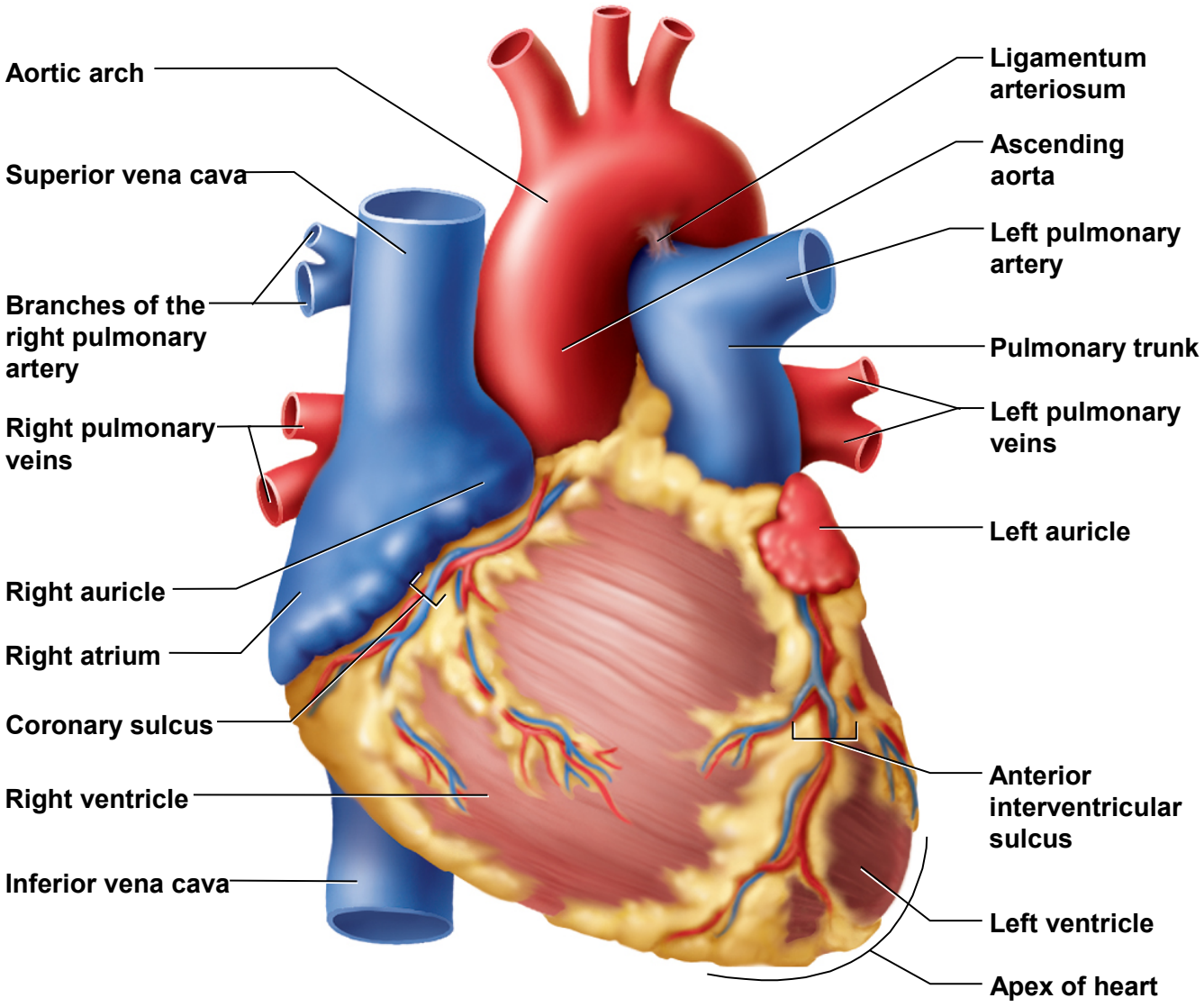
Coronary Circulation

- 5% of blood pumped by heart is pumped to the heart itself through the coronary circulation to sustain its strenuous workload
  - 250 ml of blood per minute
  - needs abundant O_2 and nutrients

- **left coronary artery** (LCA) branch off the ascending aorta
  - **anterior interventricular branch**
    - supplies blood both ventricles and anterior two-thirds of the interventricular septum
  - **circumflex branch**
    - passes around left side of heart in coronary sulcus
    - gives off **left marginal branch** and then ends on the posterior side of the heart
    - supplies left atrium and posterior wall of left ventricle

- **right coronary artery** (RCA) branch off the ascending aorta
  - supplies right atrium and sinoatrial node (pacemaker)
  - **right marginal branch**
    - supplies lateral aspect of right atrium and ventricle
  - **posterior interventricular branch**
    - supplies posterior walls of ventricles

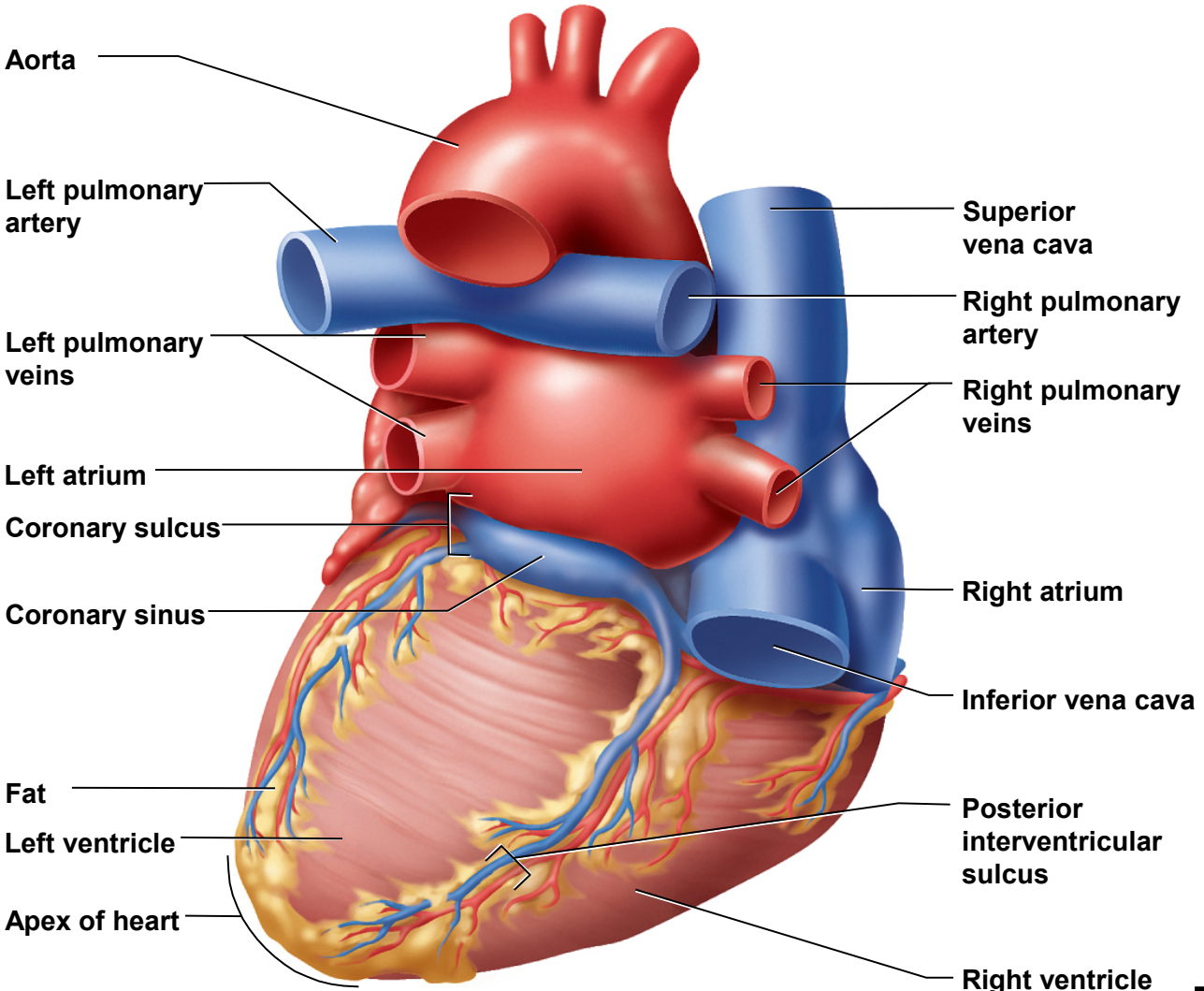
Coronary Vessels - Anterior



(a) Anterior view

Figure 19.5a

Coronary Vessels - Posterior



(b) Posterior view

Figure 19.5b

Coronary Blood Flow

- blood flow to the heart muscle during ventricular contraction is slowed, unlike the rest of the body

- three reasons:
  - contraction of the myocardium compresses the coronary arteries and obstructs blood flow
  - opening of the aortic valve flap during ventricular systole covers the openings to the coronary arteries blocking blood flow into them
  - during ventricular diastole, blood in the aorta surges back toward the heart and into the openings of the coronary arteries
    - blood flow to the myocardium increases during ventricular relaxation

Angina and Heart Attack

- **angina pectoris** – chest pain from partial obstruction of coronary blood flow
  - pain caused by ischemia of cardiac muscle
  - obstruction partially blocks blood flow
  - myocardium shifts to anaerobic fermentation producing lactic acid stimulating pain
- **myocardial infarction** – sudden death of a patch of myocardium resulting from long-term obstruction of coronary circulation
  - atheroma (blood clot or fatty deposit) often obstruct coronary arteries
  - cardiac muscle downstream of the blockage dies
  - heavy pressure or squeezing pain radiating into the left arm
  - some painless heart attacks may disrupt electrical conduction pathways, lead to fibrillation and cardiac arrest
    - silent heart attacks occur in diabetics & elderly
  - MI responsible for about half of all deaths in the United States

Venous Drainage of Heart

- 5 -10% drains directly into heart chambers, right atrium and right ventricle, by way of the **thebesian veins**
- the rest returns to right atrium by way of the coronary sinus:
  - **great cardiac vein, middle cardiac vein, left marginal vein**
    - empty into coronary sinus
- **coronary sinus**
    - large transverse vein in coronary sulcus on posterior side of heart
    - collects blood and empties into right atrium

Structure of Cardiac Muscle

- **cardiocytes** - striated, short, thick, branched cells, one central nucleus surrounded by light staining mass of glycogen
- **intercalated discs -** join cardiocytes end to end
  - **electrical junctions** - **gap junctions** allow ions to flow between cells – can stimulate neighbors
    - entire myocardium of either two atria or two ventricles acts like single unified cell
- repair of damage of cardiac muscle is almost entirely by **fibrosis** (scarring)

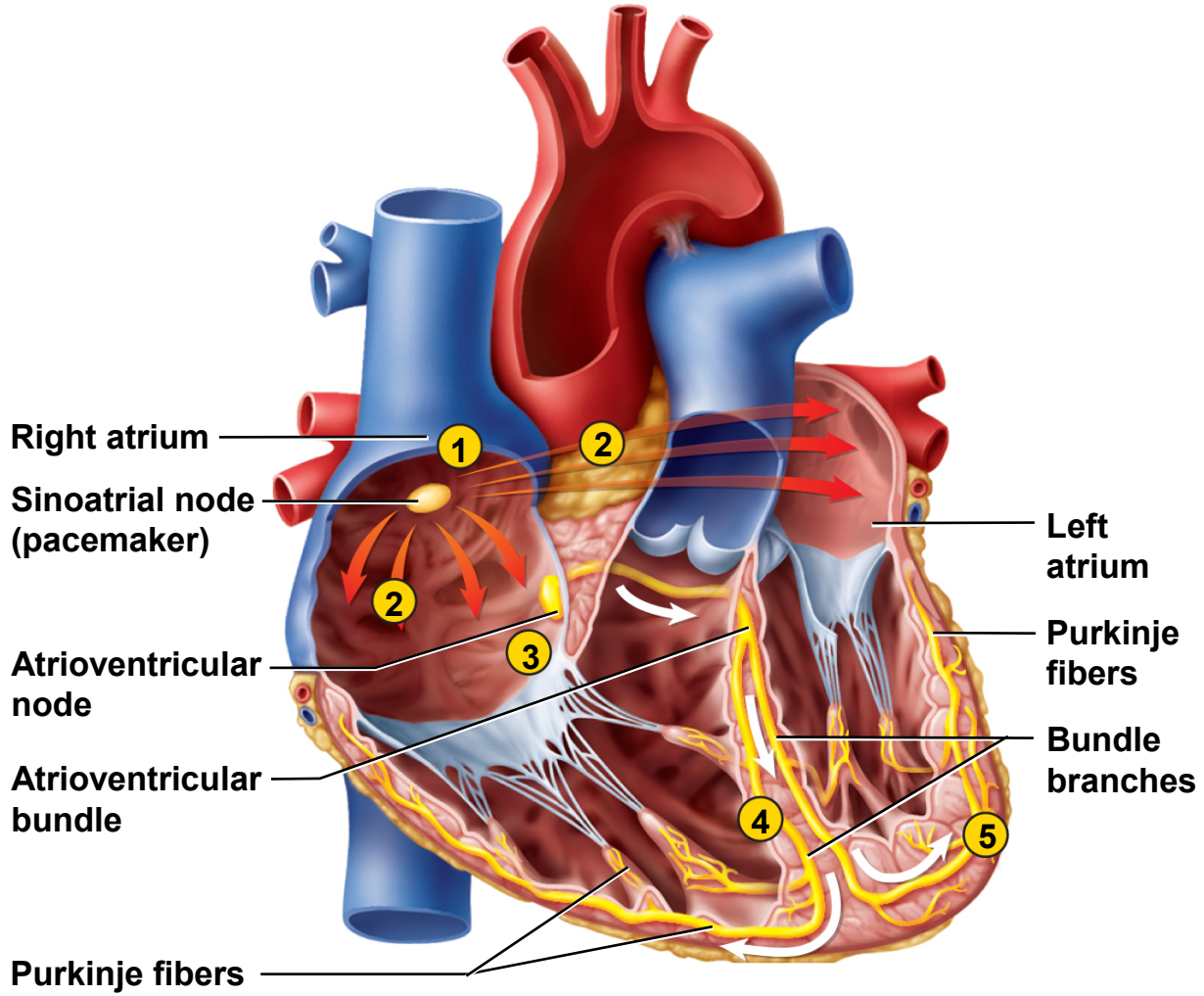
Metabolism of Cardiac Muscle

- **cardiac muscle** depends almost exclusively on **aerobic respiration** used to make ATP
  - rich in myoglobin and glycogen
  - huge mitochondria – fill 25% of cell
- **adaptable to organic fuels** used
  - fatty acids (60%), glucose (35%), ketones, lactic acid and amino acids (5%)
  - more vulnerable to oxygen deficiency than lack of a specific fuel
- **fatigue resistant** since makes little use of anaerobic fermentation or oxygen debt mechanisms
  - does not fatigue for a lifetime

Cardiac Conduction System

- coordinates the heartbeat
  - composed of an **internal pacemaker** and **nervelike conduction pathways** through myocardium
  - generates and conducts rhythmic electrical signals in the following order:
- **sinoatrial (SA) node** - modified cardiocytes
  - initiates each heartbeat and determines heart rate
  - signals spread throughout atria
  - **pacemaker** in right atrium near base of superior vena cava
- **atrioventricular (AV) node**
  - located near the right AV valve at lower end of interatrial septum
  - electrical gateway to the ventricles
  - fibrous skeleton acts as an insulator to prevent currents from getting to the ventricles from any other route
- **atrioventricular (AV) bundle (bundle of His)**
  - bundle forks into right and left bundle branches
  - these branches pass through interventricular septum toward apex
- **Purkinje fibers**
  - nervelike processes spread throughout ventricular myocardium
- signal pass from cell to cell through **gap junctions**

Cardiac Conduction System



1. SA node fires.
2. Excitation spreads through atrial myocardium.
3. AV node fires.
4. Excitation spreads down AV bundle.
5. Purkinje fibers distribute excitation through ventricular myocardium.

Figure 19.12

Cardiac Rhythm

- cycle of events in heart – special names
  - **systole** – atrial or ventricular contraction
  - **diastole** – atrial or ventricular relaxation
- **sinus rhythm** - normal heartbeat triggered by the SA node
  - set by SA node at 60 – 100 bpm
  - adult at rest is 70 to 80 bpm
- **ectopic focus** - another parts of heart fires before SA node
  - caused by hypoxia, electrolyte imbalance, or caffeine, nicotine, and other drugs

Abnormal Heart Rhythms

- spontaneous firing from some part of heart not the SA node
  - **ectopic foci** - region of spontaneous firing
    - **nodal rhythm** – if SA node is damaged, heart rate is set by AV node, 40 to 50 bpm
    - **intrinsic ventricular rhythm** – if both SA and AV nodes are not functioning, rate set at 20 to 40 bpm
      - this requires pacemaker to sustain life

- **arrhythmia** – any abnormal cardiac rhythm
  - failure of conduction system to transmit signals (heart block)
    - bundle branch block
    - total heart block (damage to AV node)

Cardiac Arrhythmias

- **atrial flutter** – ectopic foci in atria
  - atrial fibrillation
  - atria beat 200 - 400 times per minute
- **premature ventricular contractions** (PVCs)
  - caused by stimulants, stress or lack of sleep
- **ventricular fibrillation**
  - serious arrhythmia caused by electrical signals reaching different regions at widely different times
    - heart can't pump blood and no coronary perfusion
  - kills quickly if not stopped
    - **defibrillation** - strong electrical shock whose intent is to depolarize the entire myocardium, stop the fibrillation, and reset SA nodes to sinus rhythm

Impulse Conduction to Myocardium

- signal from **SA node** stimulates two atria to contract almost simultaneously
  - reaches AV node in 50 msec
- signal slows down through **AV node**
  - thin cardiocytes have fewer gap junctions
  - delays signal 100 msec which allows the ventricles to fill
- signals travel very quickly through **AV bundle** and **Purkinje fibers**
  - entire ventricular myocardium depolarizes and contracts in near unison
    - papillary muscles contract an instant earlier than the rest, tightening slack in chordae tendineae
- ventricular systole progresses up from the apex of the heart
  - spiral arrangement of cardiocytes twists ventricles slightly
  - like someone wringing out a towel

Electrocardiogram (ECG or EKG)

- composite of all action potentials of nodal and myocardial cells detected, amplified and recorded by electrodes on arms, legs and chest

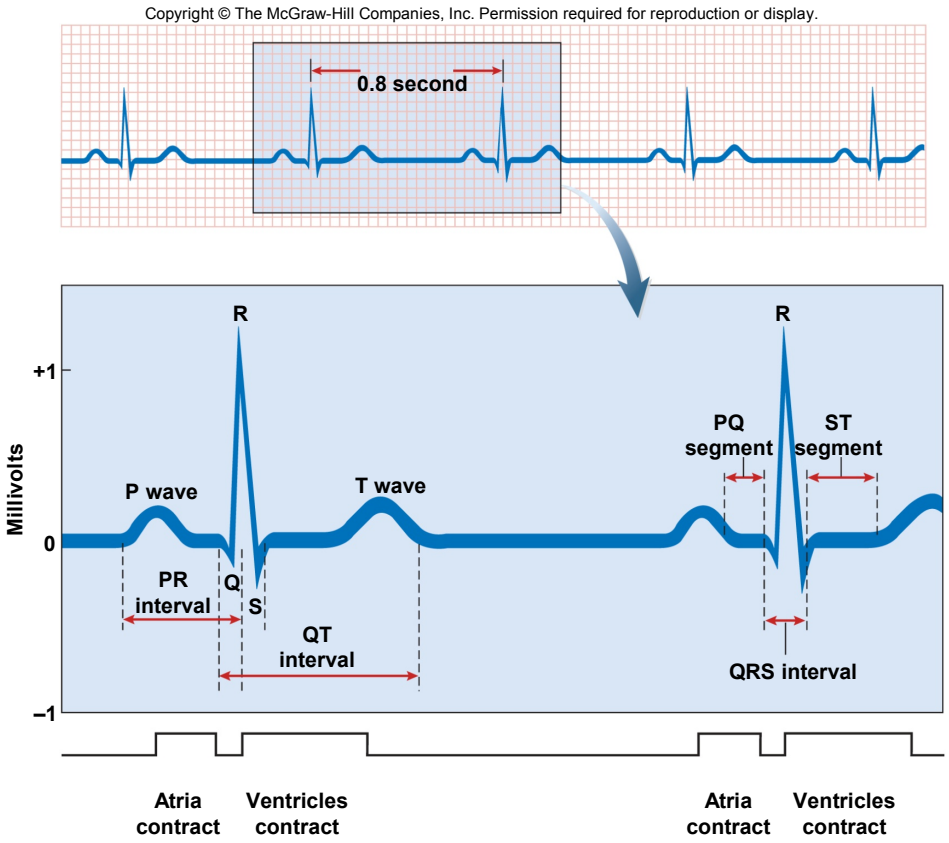


Figure 19.15

ECG Deflections

- **P wave**
  - SA node fires, **atria depolarize** and contract
  - atrial systole begins 100 msec after SA signal
- **QRS complex**
  - **ventricular depolarization**
  - complex shape of spike due to different thickness and shape of the two ventricles
- **ST segment** - **ventricular systole**
  - plateau in myocardial action potential
- **T wave**
  - **ventricular repolarization** and relaxation

Normal Electrocardiogram (ECG)

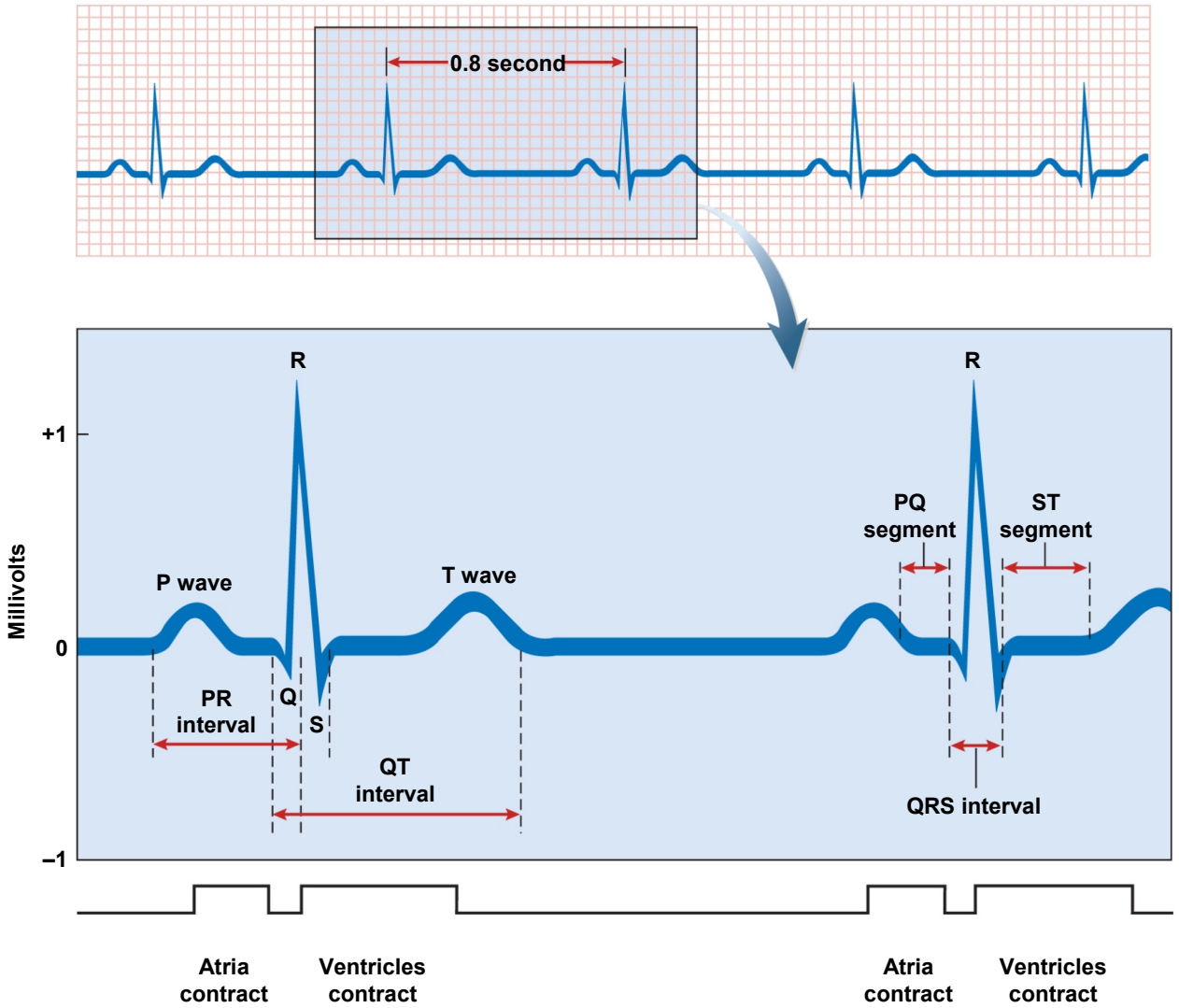


Figure 19.15

Electrical Activity of Myocardium

1) atrial depolarization begins
2) atrial depolarization complete (atria contracted)
1) ventricles begin to depolarize at apex; atria repolarize (atria relaxed)
1) ventricular depolarization complete (ventricles contracted)
1) ventricles begin to repolarize at apex
6) ventricular repolarization complete (ventricles relaxed)

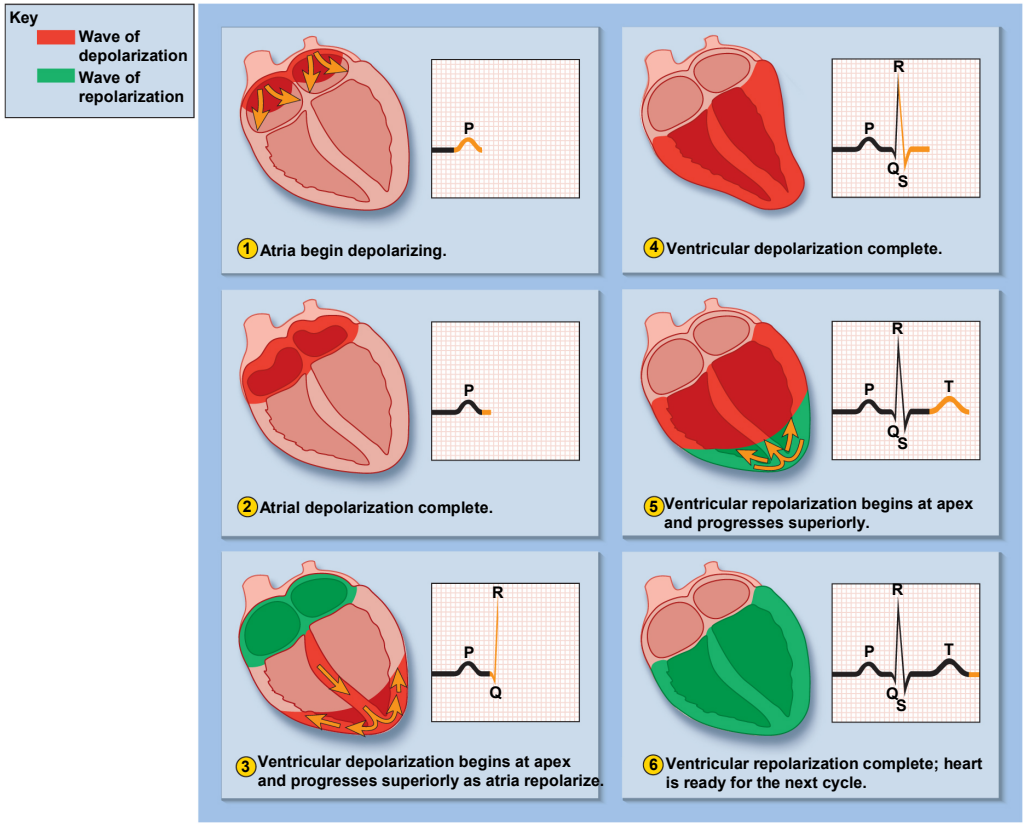
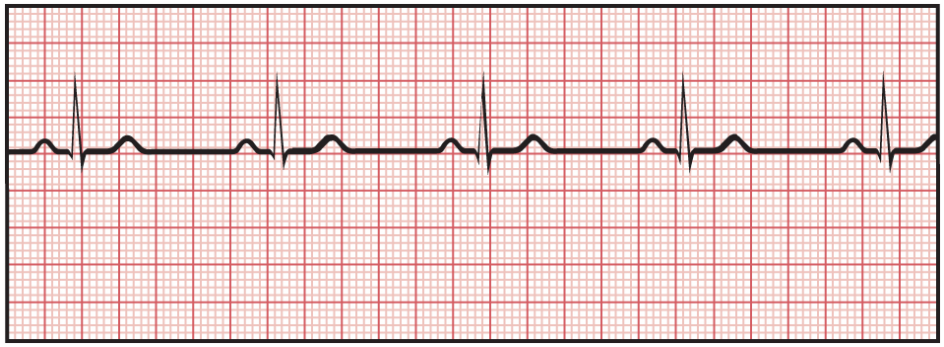


Figure 19.16

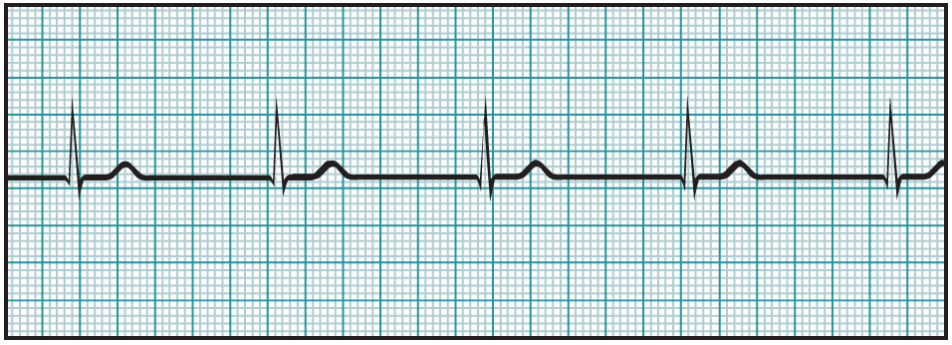
Diagnostic Value of ECG

- abnormalities in conduction pathways
- myocardial infarction
- nodal damage
- heart enlargement
- electrolyte and hormone imbalances

ECGs: Normal and Abnormal



(a) Sinus rhythm (normal)



(b) Nodal rhythm—no SA node activity

- abnormalities in conduction pathways
- myocardial infarction
- heart enlargement
- electrolyte and hormone imbalances

Figure 19.17 a-b

Cardiac Cycle

- **cardiac cycle -** one complete contraction and relaxation of all four chambers of the heart
- atrial **systole** (contraction) occurs while ventricles are in **diastole** (relaxation)
- atrial **diastole** occurs while ventricles in **systole**
- **quiescent period** all four chambers relaxed at same time
- questions to solve – *how does pressure affect blood flow?* and *how are heart sounds produced?*

Operation of Heart Valves

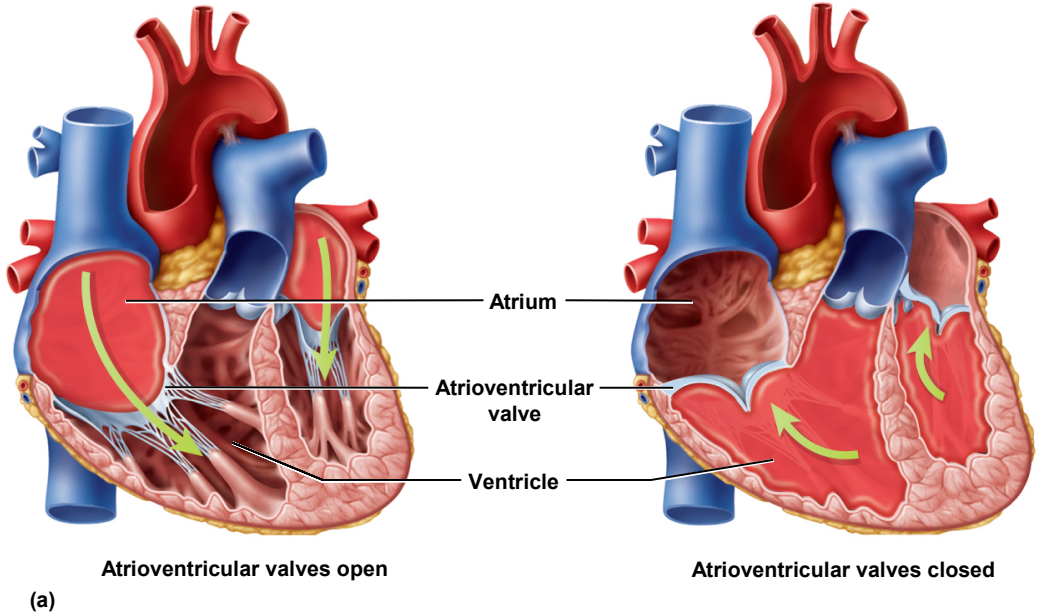


Figure 19.19

Valvular Insufficiency

- **valvular insufficiency** (incompetence) - any failure of a valve to prevent reflux (regurgitation) the backward flow of blood
  - **valvular stenosis** – cusps are stiffened and opening is constricted by scar tissue
    - result of **rheumatic fever** autoimmune attack on the mitral and aortic valves
    - heart overworks and may become enlarged
    - **heart murmur** – abnormal heart sound produced by regurgitation of blood through incompetent valves
  - **mitral valve prolapse** – insufficiency in which one or both mitral valve cusps bulge into atria during ventricular contraction
    - hereditary in 1 out of 40 people
    - may cause chest pain and shortness of breath

Heart Sounds

- **auscultation** - listening to sounds made by body
- **first heart sound** (S_1), louder and longer "lubb", occurs with closure of AV valves, turbulence in the bloodstream, and movements of the heart wall
- second heart sound (S_2), softer and sharper "dupp" occurs with closure of semilunar valves, turbulence in the bloodstream, and movements of the heart wall
- S_3 - rarely heard in people over 30
- exact cause of each sound is not known with certainty

Phases of Cardiac Cycle

- ventricular filling
- isovolumetric contraction
- ventricular ejection
- isovolumetric relaxation

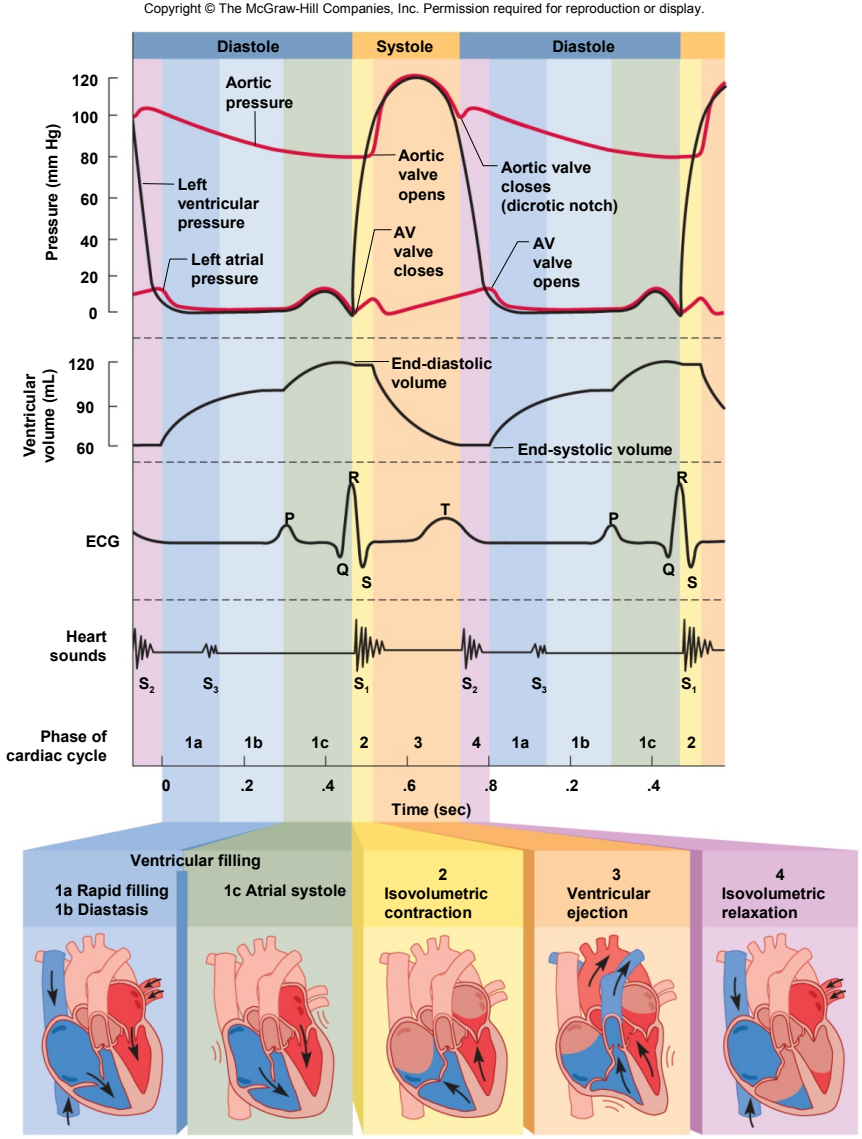
- all the events in the cardiac cycle are completed in less than one second!

Timing of Cardiac Cycle

- in a resting person
  - **atrial systole** last about **0.1 sec**
  - **ventricular systole** about **0.3 sec**
  - **quiescent period**, when all four chambers are in diastole, **0.4 sec**

- **total duration of the cardiac cycle** is therefore **0.8 sec** in a heart beating **75 bpm**

Major Events of Cardiac Cycle



- ventricular filling
- isovolumetric contraction
- ventricular ejection
- isovolumetric relaxation

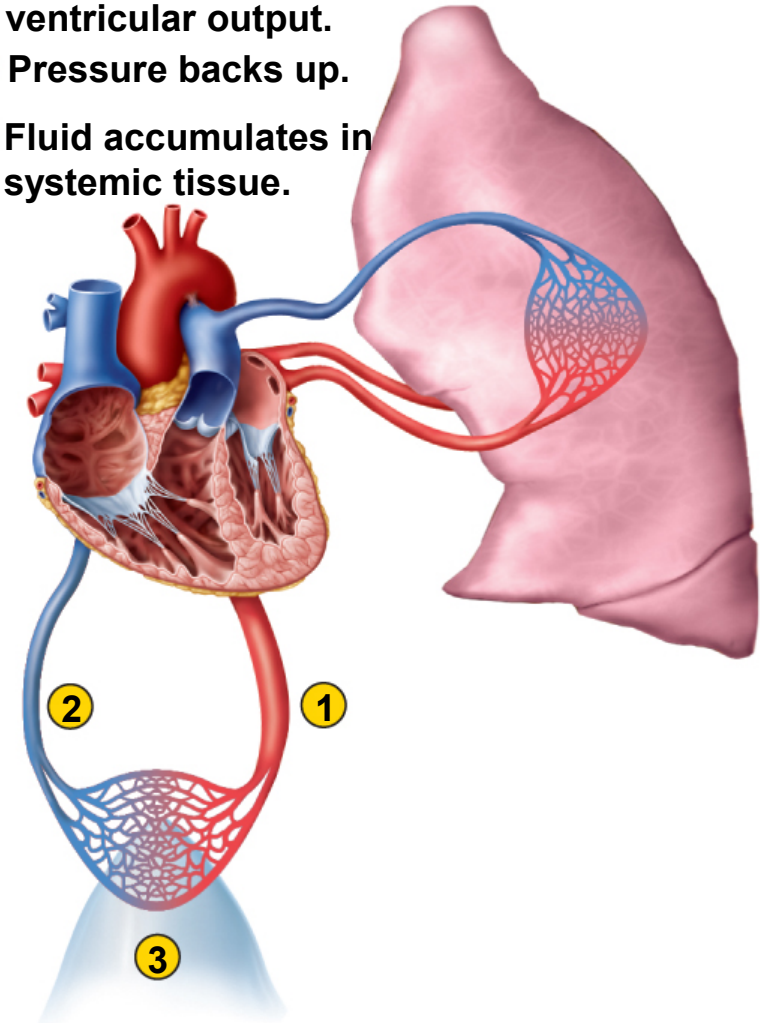
Figure 19.20

Congestive Heart Failure

- **congestive heart failure** (CHF) - results from the failure of either ventricle to eject blood effectively
  - usually due to a heart weakened by myocardial infarction, chronic hypertension, valvular insufficiency, or congenital defects in heart structure.
- **left ventricular failure** – blood backs up into the lungs causing pulmonary edema
  - shortness of breath or sense of suffocation
- **right ventricular failure** – blood backs up in the vena cava causing systemic or generalized edema
  - enlargement of the liver, ascites (pooling of fluid in abdominal cavity), distension of jugular veins, swelling of the fingers, ankles, and feet
- eventually leads to total heart failure

Unbalanced Ventricular Output

1. Left ventricular output exceeds right ventricular output.
2. Pressure backs up.
3. Fluid accumulates in systemic tissue.



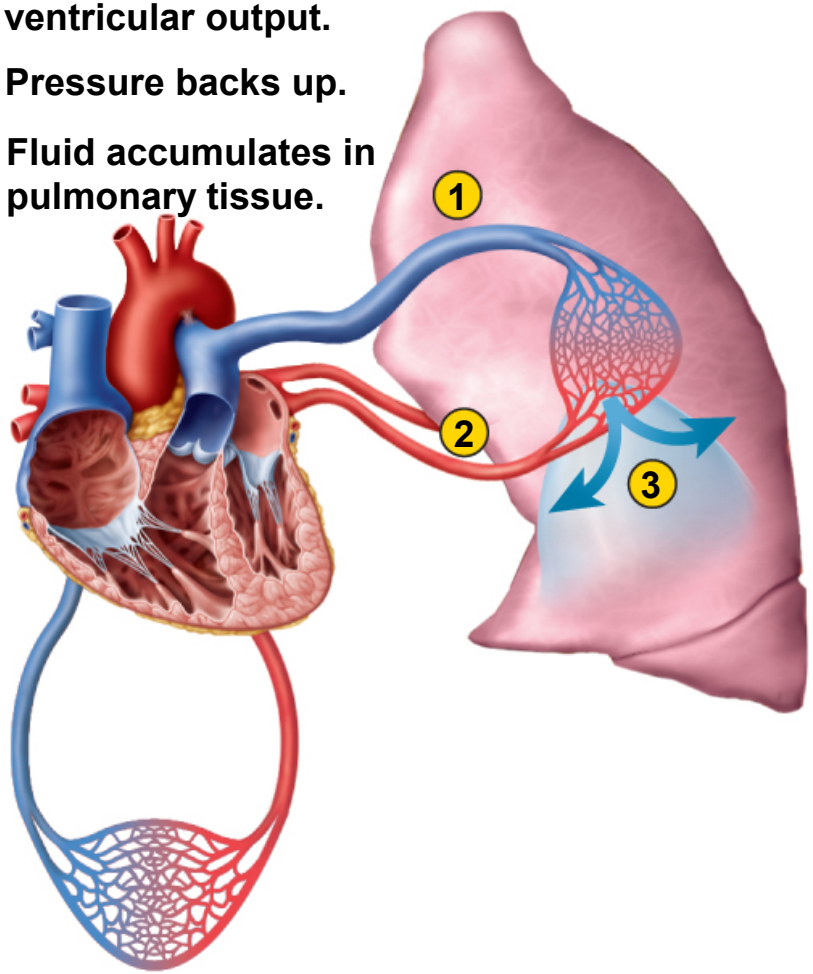
(b) Systemic edema

peripheral edema

Figure 19.21b

Unbalanced Ventricular Output

1. Right ventricular output exceeds left ventricular output.
2. Pressure backs up.
3. Fluid accumulates in pulmonary tissue.



(a) Pulmonary edema

pulmonary edema

Figure 19.21a

Cardiac Output (CO)

- **cardiac output** (CO) – the amount ejected by ventricle in 1 minute
- **cardiac output = heart rate x stroke volume**
  - about **4 to 6 L/min at rest**
  - a RBC leaving the left ventricle will arrive back at the left ventricle in about 1 minute
  - vigorous exercise increases CO to 21 L/min for fit person and up to 35 L/min for world class athlete
- **cardiac reserve** – the difference between a person's maximum and resting CO
  - increases with fitness, decreases with disease

Heart Rate

- **pulse** – surge of pressure produced by each heart beat that can be felt by palpating a superficial artery with the fingertips
  - infants have HR of 120 bpm or more
  - young adult females avg. 72 - 80 bpm
  - young adult males avg. 64 to 72 bpm
  - heart rate rises again in the elderly
- **tachycardia** - resting adult heart rate above 100 bpm
  - stress, anxiety, drugs, heart disease, or fever
  - loss of blood or damage to myocardium
- **bradycardia** - resting adult heart rate of less than 60 bpm
  - in sleep, low body temperature, and endurance trained athletes

Chronotropic Effects of the Autonomic Nervous System

- **sympathetic**
  - release **norepinephrine**
  - binds to **β-adrenergic fibers** in the heart
  - **accelerated depolarization of SA node**
  - cardiocytes **relax more quickly**
  - by accelerating both contraction and relaxation, heart rate increases as high as 230 bpm
  - diastole becomes too brief for adequate filling
  - both stroke volume and cardiac output are reduced

Chronotropic Effects of the Autonomic Nervous System

- **parasympathetic vagus nerves**
  - **Release acetylcholine (ACh)** binds to **muscarinic receptors**
  - **hyperpolarized** nodal cells fire less frequently
  - **heart slows down**
  - parasympathetics work on the heart faster than sympathetics
    - parasympathetics do not need a second messenger system
- without influence from the cardiac centers, the heart has **a intrinsic "natural" firing rate of 100 bpm**
- **vagal tone** – holds down this heart rate to 70 – 80 bpm at rest
  - steady background firing rate of the **vagus nerves**

Inputs to Cardiac Center

- **cardiac centers in the medulla** receive input from many sources and integrate it into the 'decision' to speed or slow the heart
- **higher brain centers** affect heart rate
  - **cerebral cortex**, **limbic system**, **hypothalamus**
    - sensory or emotional stimuli
- medulla also receives input from muscles, joints, arteries, and brainstem
  - **proprioceptors** in the muscles and joints
    - inform cardiac center about changes in activity, HR increases before metabolic demands of muscle arise
  - **baroreceptors** signal cardiac center
    - pressure sensors in **aorta and internal carotid arteries**
    - blood pressure decreases, signal rate drops, cardiac center increases heart rate
    - if blood pressure increases, signal rate rises, cardiac center decreases heart rate

Chronotropic Chemicals

- chemicals affect heart rate as well as neurotransmitters from cardiac nerves
  - blood born adrenal catecholamines (NE and epinephrine) are potent cardiac stimulants
- **drugs** that stimulate heart
  - **nicotine** stimulates catecholamine secretion
  - **thyroid hormone** increases number adrenergic receptors on heart so more responsive to sympathetic stimulation
  - **caffeine** inhibits cAMP breakdown prolonging adrenergic effect

Chronotropic Chemicals

- electrolytes
  - K^+ has greatest chronotropic effect
    - **hyperkalemia** – excess K^+ in cardiocytes
      - myocardium less excitable, heart rate slows and becomes irregular
    - **hypokalemia** – deficiency K^+ in cardiocytes
      - cells hyperpolarized, require increased stimulation
  - calcium
    - **hypercalcemia** – excess of Ca^{2+}
      - decreases heart rate and contraction strength
    - **hypocalcemia** – deficiency of Ca^{2+}
      - increases heart rate and contraction strength

Stroke Volume (SV)

- the other factor that in cardiac output, besides heart rate, is **stroke volume**
- three variables govern stroke volume:
  1. preload
  2. contractility
  3. afterload
- example
  - increased preload or contractility causes increases stroke volume
  - increased afterload causes decrease stroke volume

Exercise and Cardiac Output

- exercise makes the heart work harder and increases cardiac output
- **proprioceptors** signal cardiac center
  - at beginning of exercise, signals from joints and muscles reach the cardiac center of brain
  - sympathetic output from cardiac center increases cardiac output
- increased muscular activity increases venous return
  - increases preload and ultimately cardiac output
- increase in heart rate and stroke volume cause an increase in cardiac output
- exercise produces ventricular hypertrophy
  - increased stroke volume allows heart to beat more slowly at rest
  - athletes with increased cardiac reserve can tolerate more exertion than a sedentary person

Coronary Artery Disease

- **coronary artery disease** (CAD) – a constriction of the coronary arteries
  - usually the result of **atherosclerosis** – accumulation of lipid deposits that degrade the arterial wall and obstruct the lumen
  - endothelium damaged by hypertension, virus, diabetes or other causes
  - monocytes penetrate walls of damaged vessels and transform into macrophages
    - absorb cholesterol and fats to be called **foam cells**
      - look like fatty streak on vessel wall
      - can grow into atherosclerotic plaques (**atheromas**)
  - platelets adhere to damaged areas and secrete platelet-derived growth factor
    - attracting immune cells and promoting mitosis of muscle and fibroblasts, and the deposition of collagen
- bulging mass grows to obstruct arterial lumen

Affects of Atheromas

- causes **angina pectoris**, intermittent chest pain, by obstructing 75% or more of the blood flow
- immune cells of atheroma stimulate inflammation – may rupture – traveling clots or fatty emboli may result
- inflammation transforms atheroma into a hardened complicated plaque called **arteriosclerosis**

Risk

- major risk factor for atherosclerosis is excess of low-density lipoprotein (LDL) in the blood combined with defective LDL receptors in the arterial walls
  - protein-coated droplets of cholesterol, neutral fats, free fatty acids and phospholipids
- most cells have LDL receptors that take up these droplets from blood by receptor-mediated endocytosis
  - dysfunctional receptors in arterial cells accumulate excess cholesterol
- familial hypercholesterolemia
  - dominant gene makes no receptors for LDL
    - heterozygous individual suffer heart attacks by 35
    - homozygous individuals suffer heart attacks by 2
- unavoidable risk factors - heredity, aging, being male
- avoidable risk factors – obesity, smoking, lack of exercise, anxious personality, stress, aggression, and diet

Prevention and Treatment

- treatment
  - coronary bypass surgery
    - great saphenous vein
  - balloon angioplasty
  - laser angioplasty