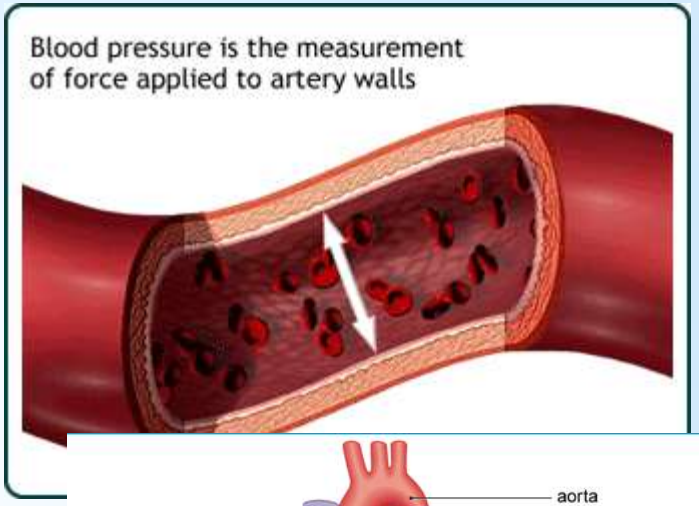
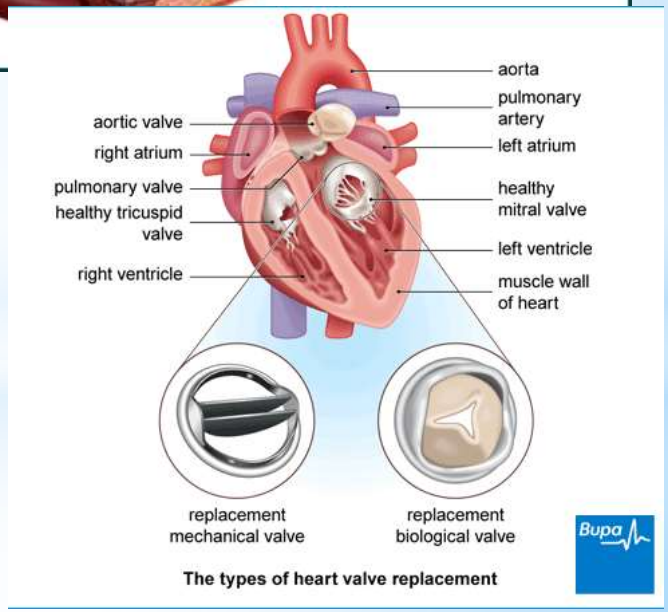
Functions of the Heart

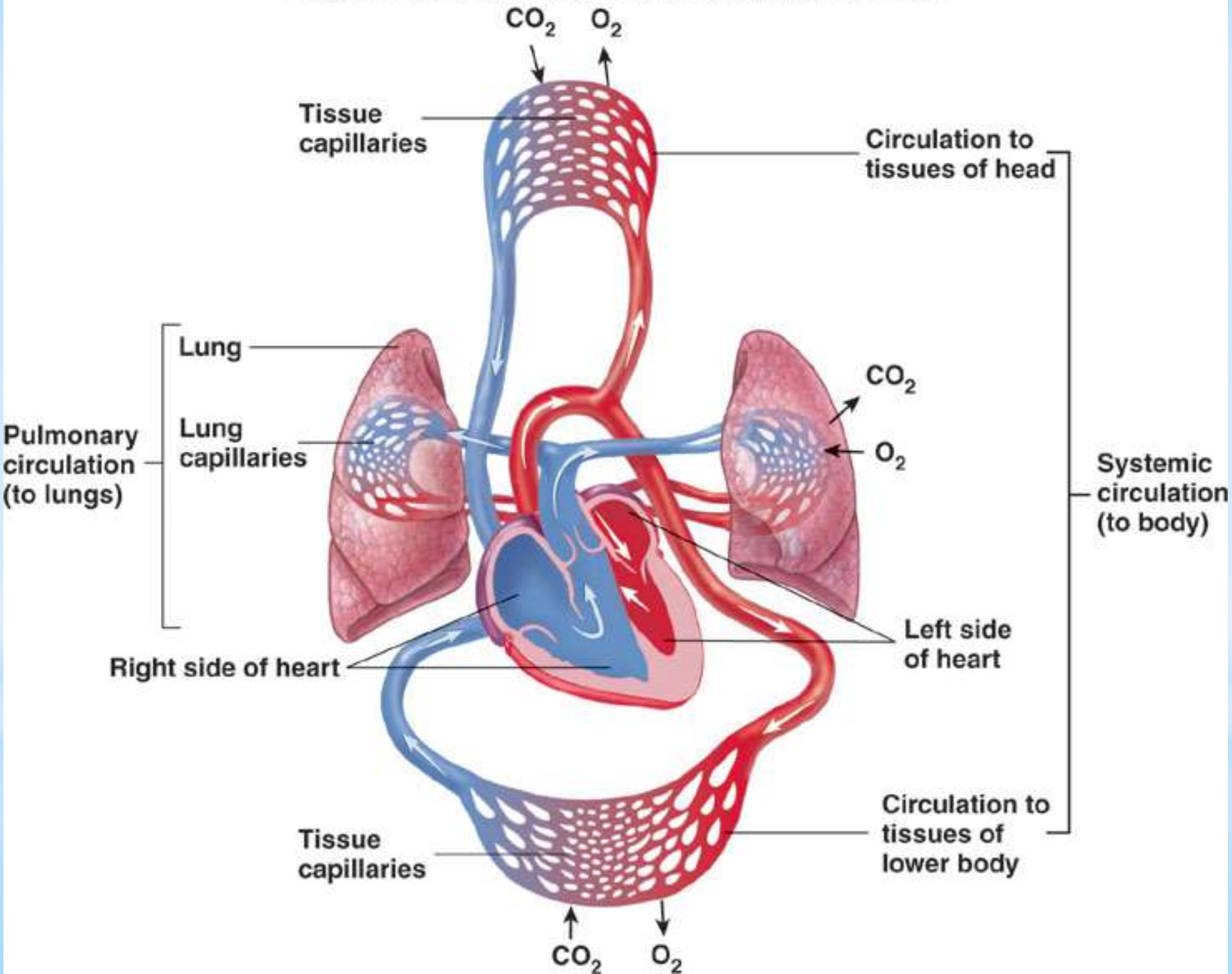
- Generating blood pressure
- Routing blood: separates pulmonary and systemic circulations
- Ensuring one-way blood flow: valves
- Regulating blood supply
  - Changes in contraction rate and force match blood delivery to changing metabolic needs





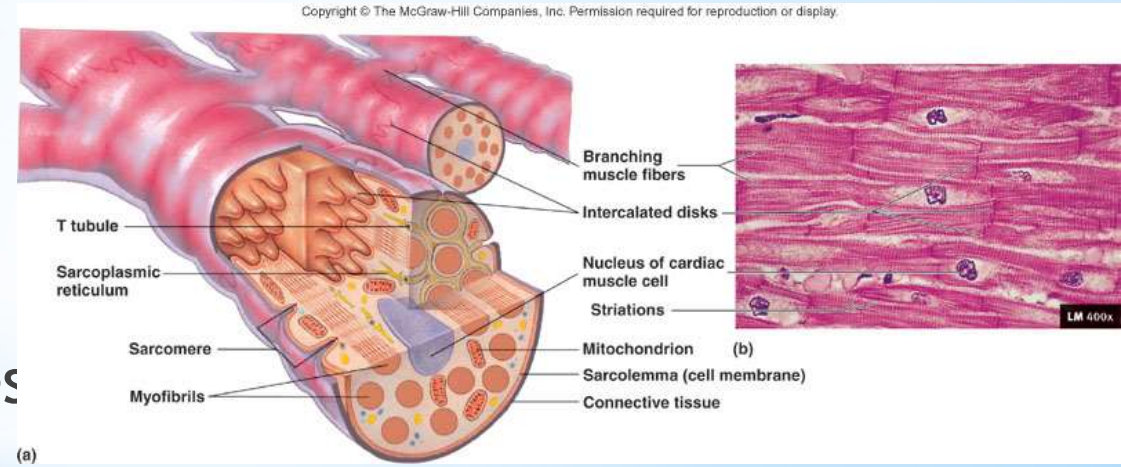
The cardiovascular system is divided into two circuits

- Pulmonary circuit
  - blood to and from the lungs
- Systemic circuit
  - blood to and from the rest of the body
- Vessels carry the blood through the circuits
  - Arteries carry blood away from the heart
  - Veins carry blood to the heart
  - Capillaries permit exchange



Cardiac Muscle

- Elongated, branching cells containing 1-2 centrally located nuclei
- Contains actin and myosin myofilaments
- **Intercalated disks**: specialized cell-cell contacts.
- Electrically, cardiac muscle of the atria and of the ventricles behaves as single unit



- Mitochondria comprise 30% of volume of the cell vs. 2% in skeletal

Cardiac Muscle Contraction

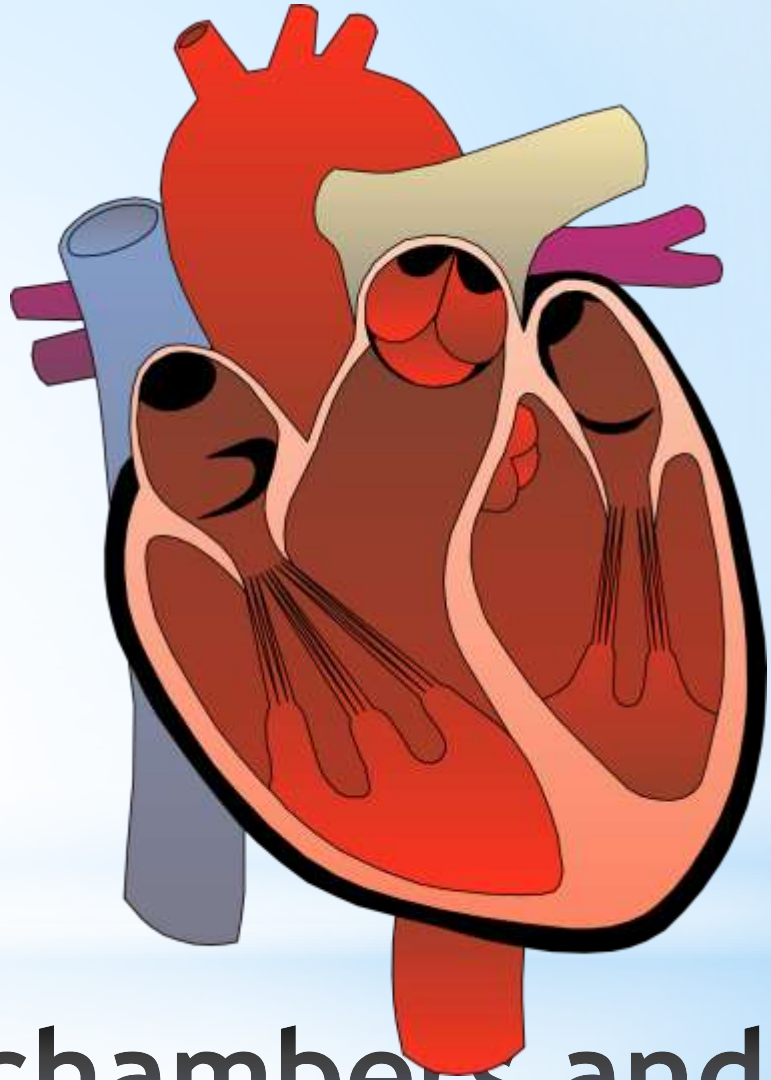
- Heart muscle:
  - Is stimulated by nerves and is self-excitable (automaticity)
  - Contracts as a unit; no *motor units*
- Cardiac muscle contraction is similar to skeletal muscle contraction, i.e., sliding-filaments

Heart chambers and valves

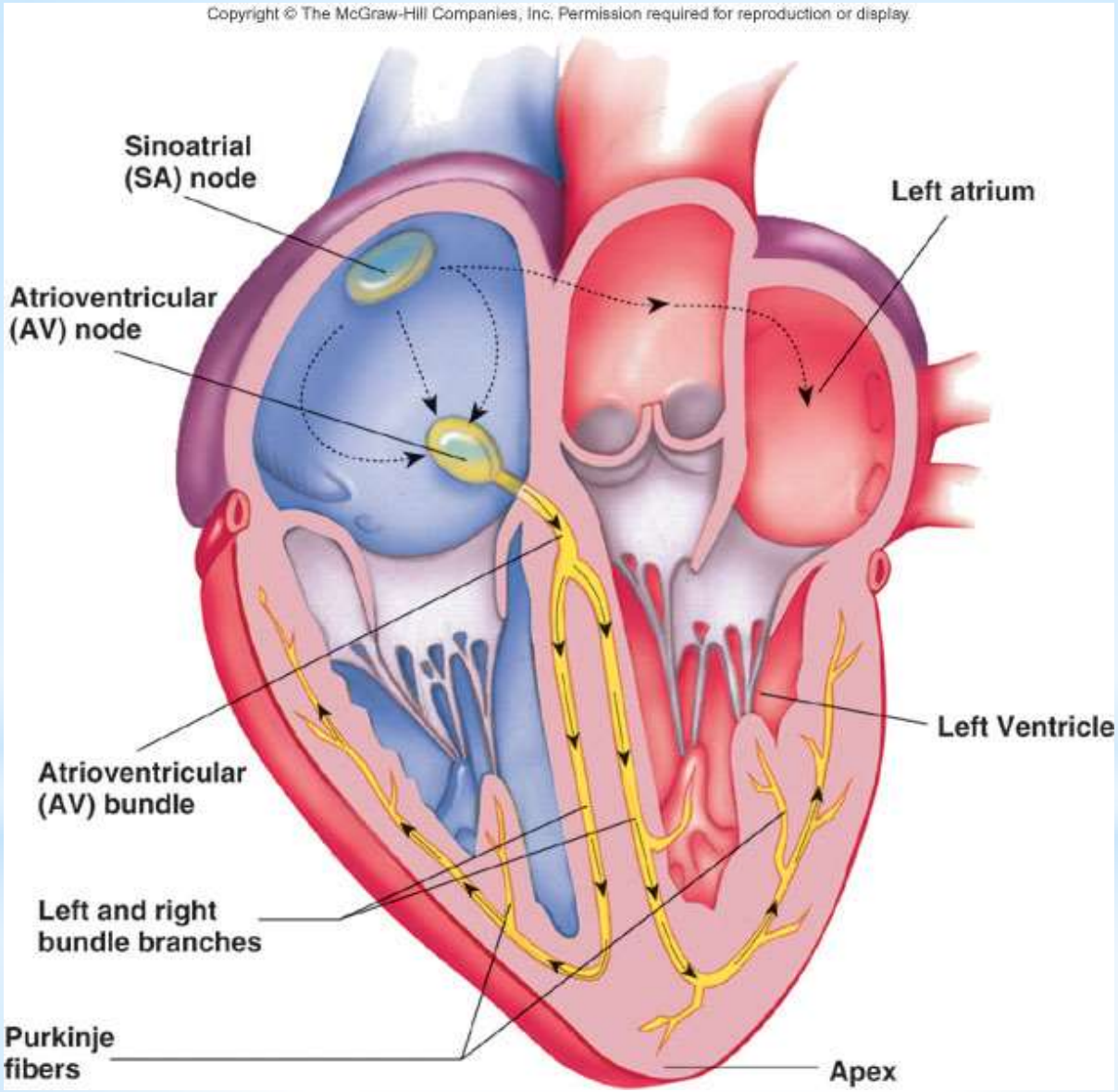
* Heart has 4 internal chambers (atria, ventricles)

* Structural Differences in heart chambers
  * The left side of the heart is more muscular than the right side

* Functions of valves (4 valves)
  * AV valves prevent backflow of blood from the ventricles to the atria
  * Semilunar valves prevent backflow into the ventricles from the pulmonary trunk and aorta



* Conducting System of Heart



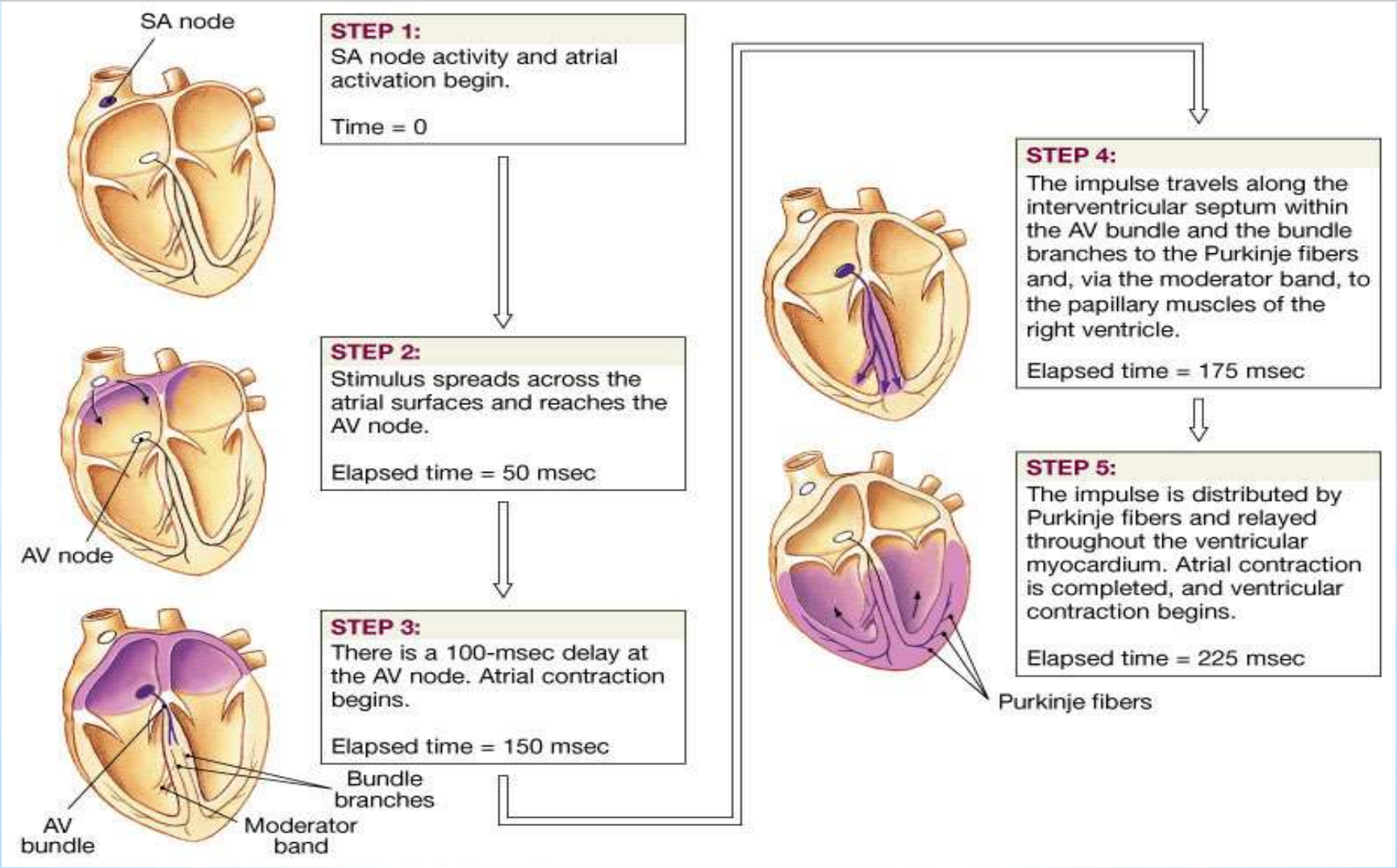
*Conduction System of the Heart

* **SA node**: sinoatrial node. The pacemaker.
  * Specialized cardiac muscle cells.
  * Generate spontaneous action potentials *(autorhythmic tissue)*.
* **AV node**: atrioventricular node.
  * Action potentials conducted more slowly here than in any other part of system.
  * Ensures ventricles receive signal to contract after atria have contracted
* **Right and left bundle branches**: extend beneath endocardium THROUGH SEPTUM to APEX of right and left ventricles
* **Purkinje fibers**:
  * Conduct action potential to ventricular muscle cells (myocardium)

* Please turn in your Alien Heart and take out your notebook!

* Heart Test: Wednesday

Impulse Conduction through the Heart



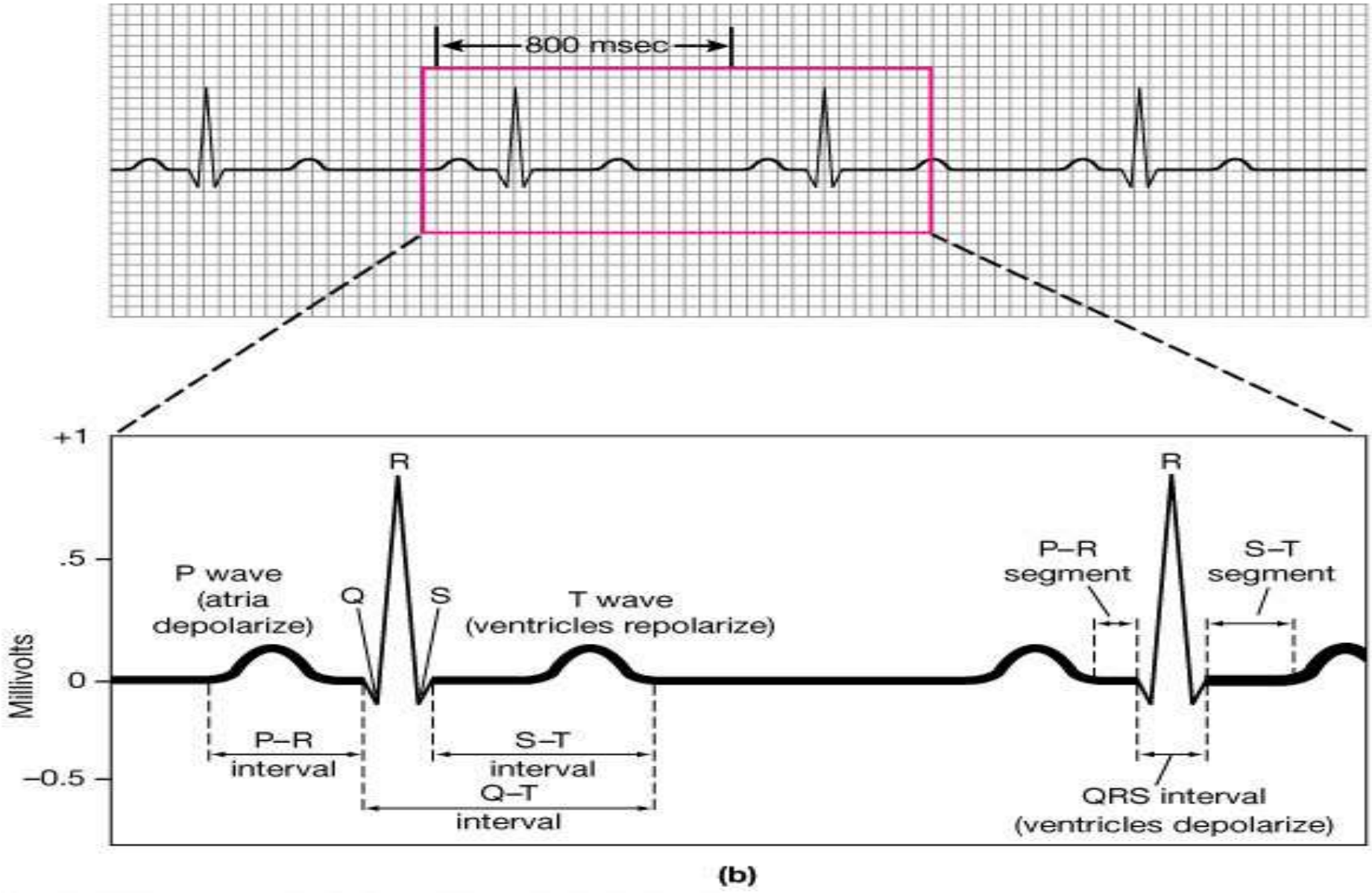
*Electrocardiogram

* Record of electrical events in the myocardium that can be correlated with mechanical events
* **P wave**: depolarization of atrial myocardium.
  * Signals onset of atrial contraction

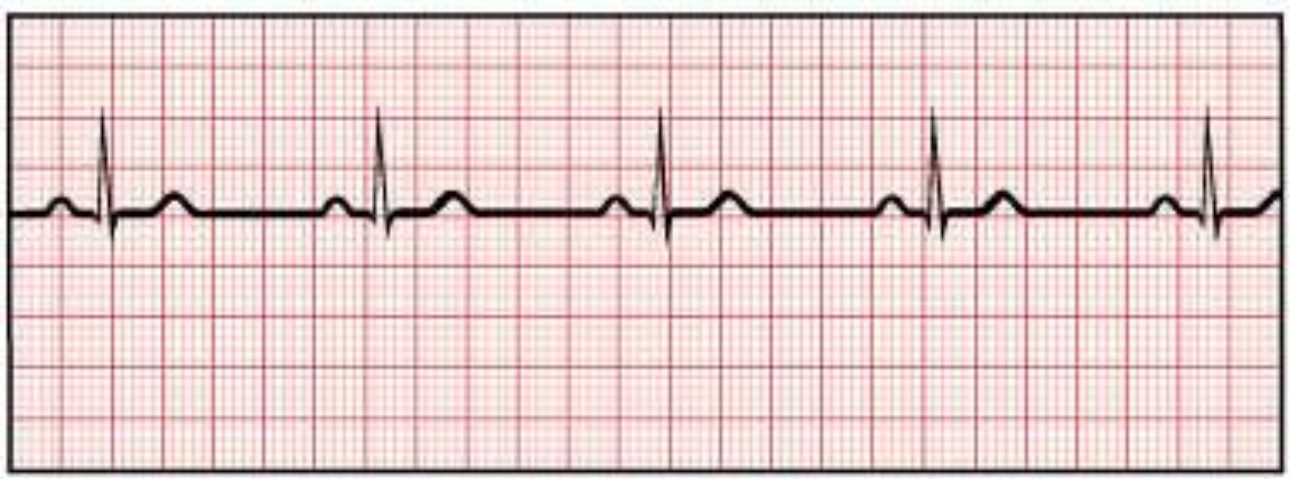
* **QRS complex**: ventricular depolarization
  * Signals onset of ventricular contraction..
* **T wave**: repolarization of ventricles
  * Can be lengthened by electrolyte disturbances, conduction problems,

* **Q-T interval**: time required for ventricles to undergo a single cycle of depolarization and repolarization
  * Can be lengthened by electrolyte disturbances, conduction problems, myocardial damage

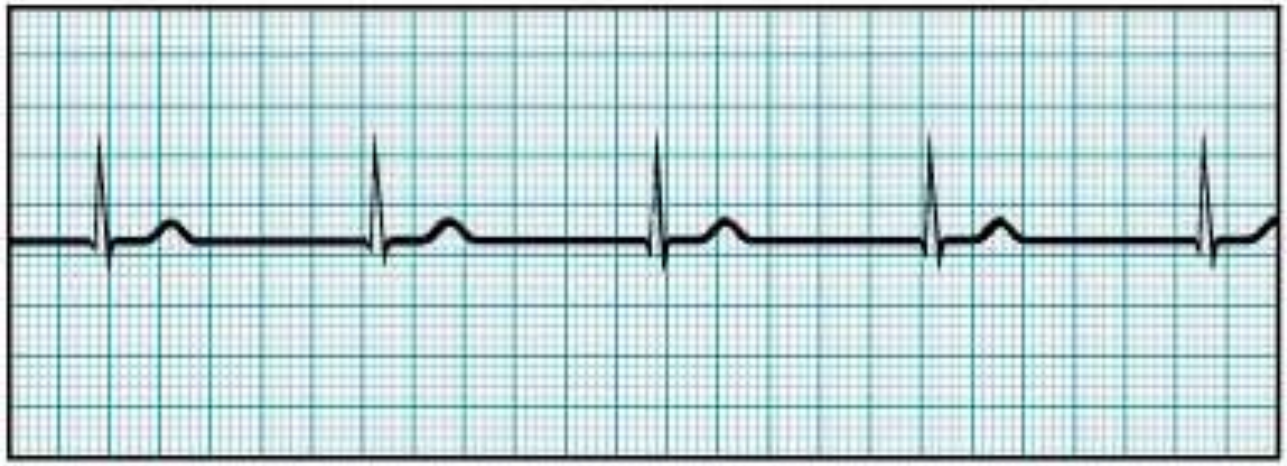
*An Electrocardiogram



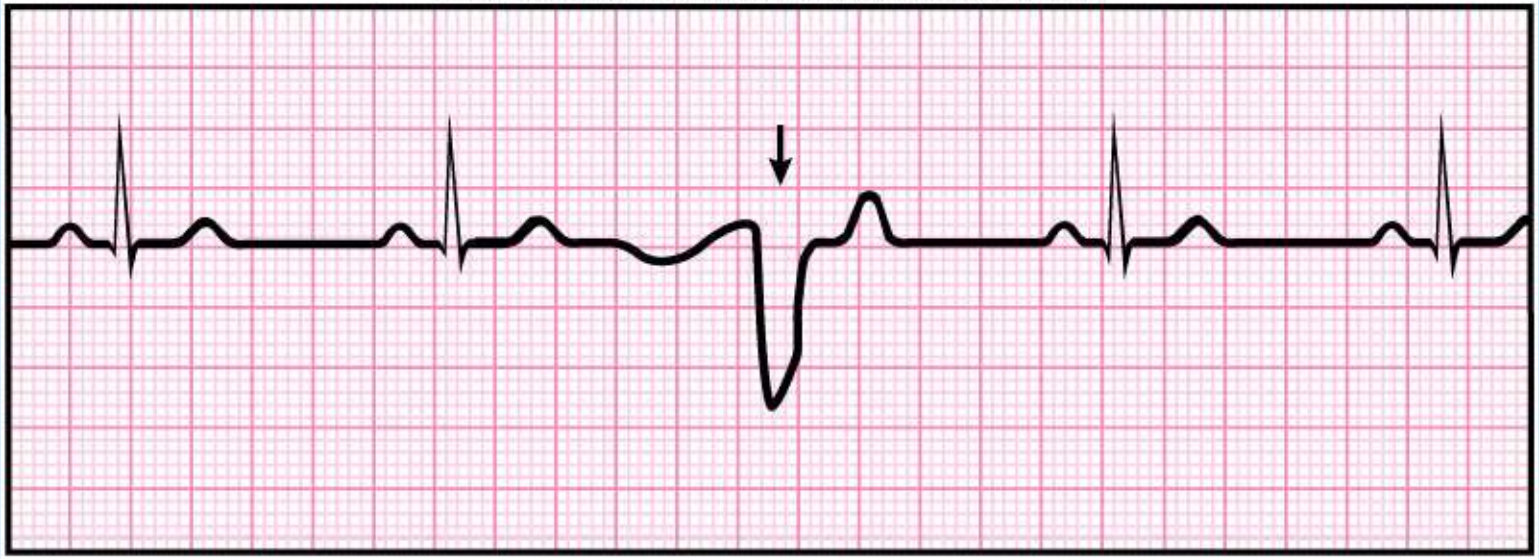
ECGs, Normal and Abnormal



(a) Sinus rhythm (normal)



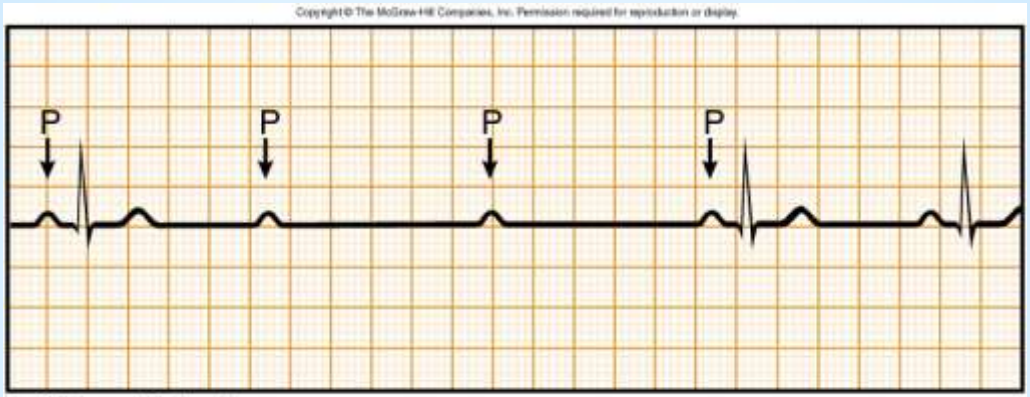
(b) Nodal rhythm – no SA node activity



(d) Premature ventricular contraction

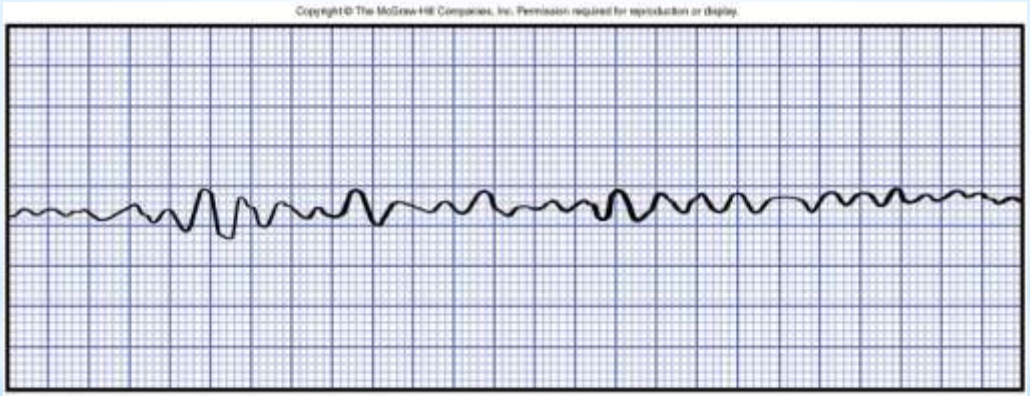
ECGs, Abnormal

Extrasystole : note inverted QRS complex, misshapen QRS and T and absence of a P wave preceding this contraction.



(c) Heart block

Arrhythmia: conduction failure at AV node



(e) Ventricular fibrillation

No pumping action occurs

*ECGs, Abnormal

The Cardiac Cycle

- Cardiac cycle refers to all events associated with blood flow through the heart from the start of one heartbeat to the beginning of the next
- During a cardiac cycle
  - Each heart chamber goes through systole and diastole
  - Correct pressure relationships are dependent on careful timing of contractions

Phases of the Cardiac Cycle

- Atrial diastole and systole -
  - Blood flows into and passively out of atria (80% of total)
    - AV valves open
  - Atrial systole pumps only about 20% of blood into ventricles
- Ventricular filling: mid-to-late diastole
  - Heart blood pressure is low as blood enters atria and flows into ventricles
  - 80% of blood enters ventricles *passively*
  - AV valves are open, then atrial systole occurs
  - Atrial systole pumps remaining 20% of blood into ventricles

Phases of the Cardiac Cycle

- Ventricular systole
  - Atria relax
  - Rising ventricular pressure results in closing of AV valves (1st heart sound - ‘lubb’)
  - Isovolumetric contraction phase
    - Ventricles are contracting but no blood is leaving
    - Ventricular pressure not great enough to open semilunar valves
  - *Ventricular ejection* phase opens semilunar valves
    - Ventricular pressure now greater than pressure in arteries (aorta and pulmonary trunk)

Phases of the Cardiac Cycle

- Ventricular diastole
  - Ventricles relax
  - Backflow of blood in aorta and pulmonary trunk closes semilunar valves (2nd hear sound - “dubb
    - Dicrotic notch – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves
  - Blood once again flowing into relaxed atria and passively into ventricles

Cardiac Output (CO) and Cardiac Reserve

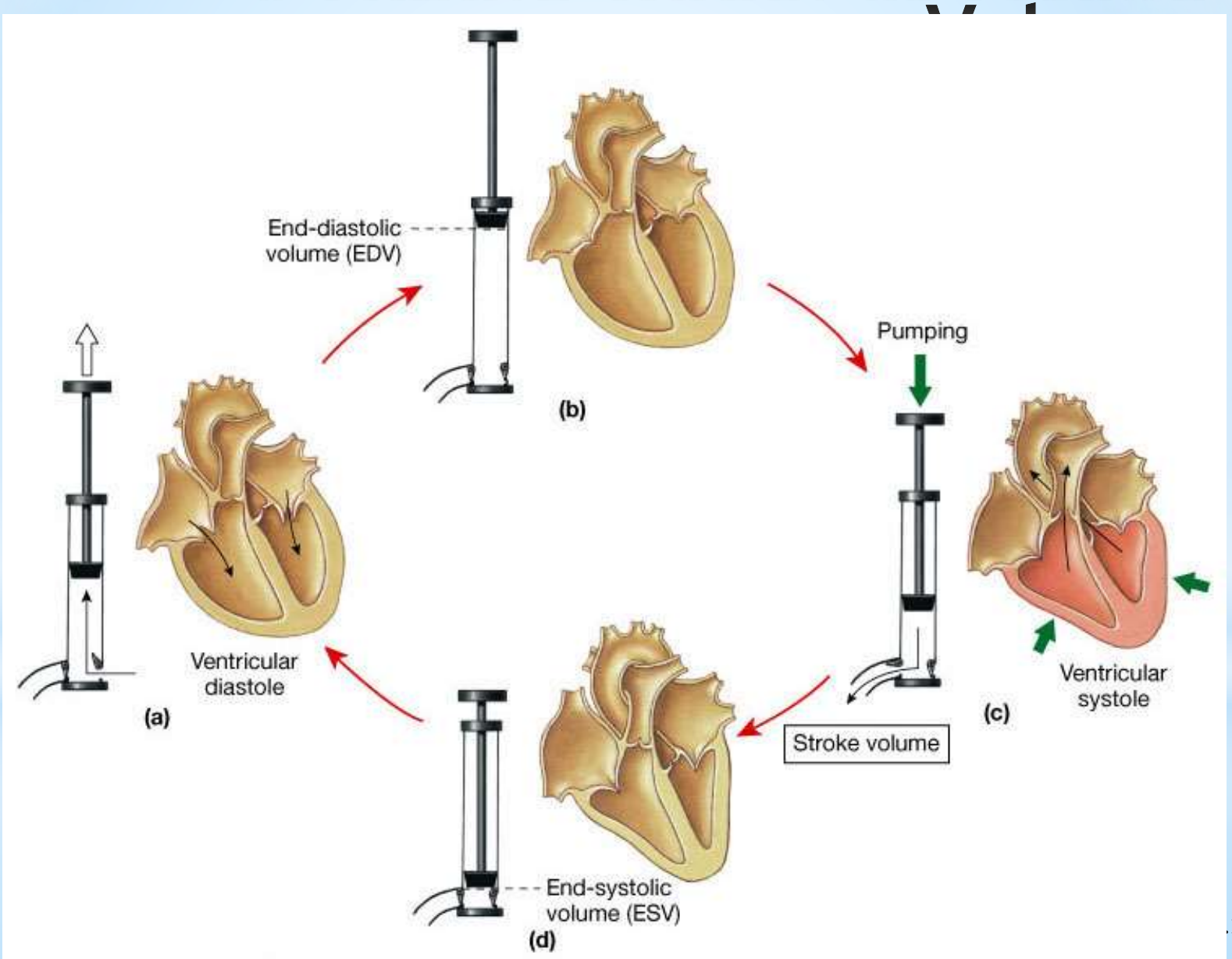
* CO is the amount of blood pumped by each ventricle in one minute
* CO is the product of heart rate (HR) and stroke volume (SV)

CO = HR x SV

(ml/min) = (beats/min) x ml/beat

* HR is the number of heart beats per minute
* SV is the amount of blood pumped out by a ventricle with each beat
* Cardiac reserve is the difference between resting and maximal CO

*A Simple Model of Stroke

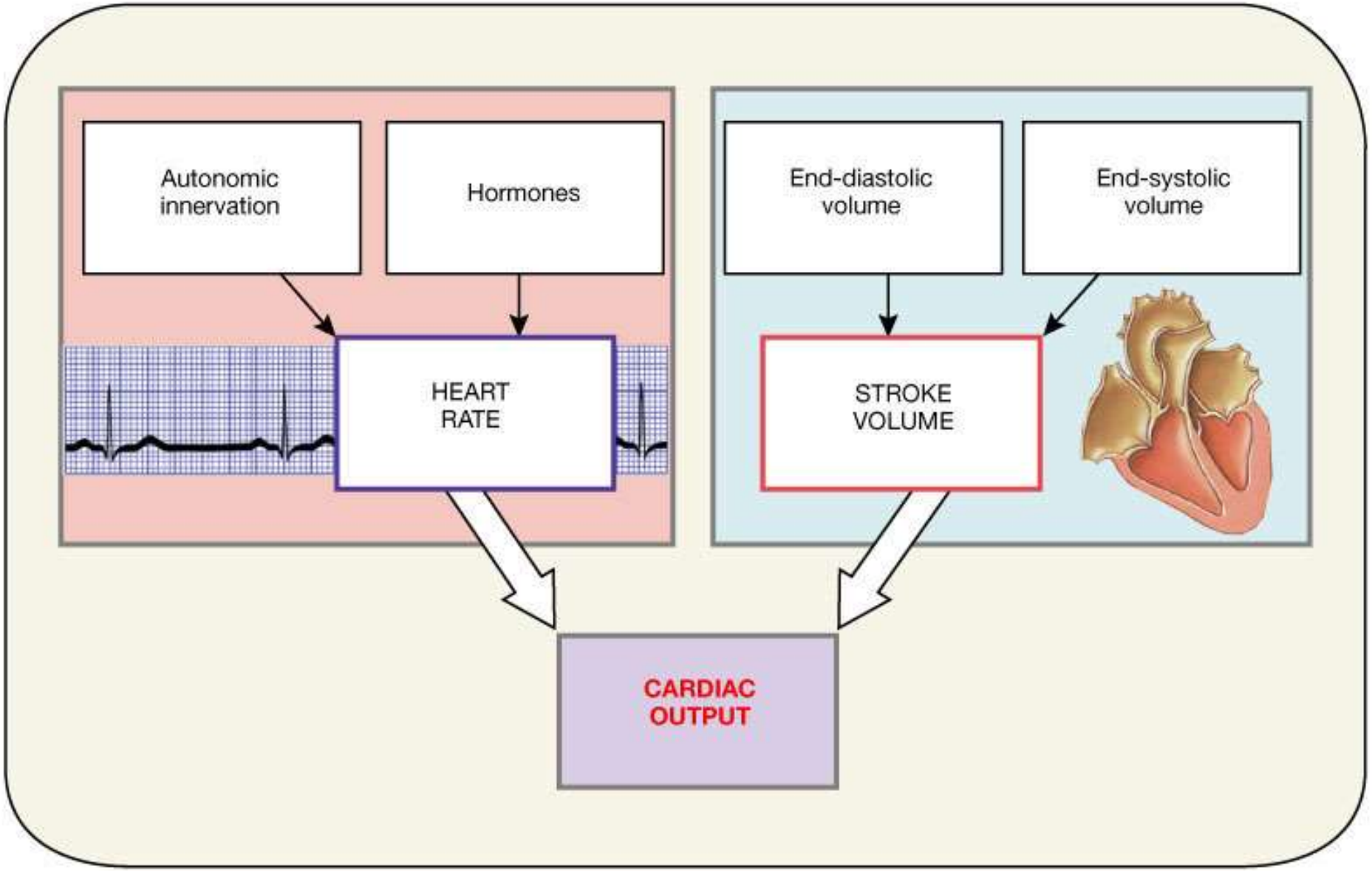


19a-d

Cardiac Output: An Example

- CO (ml/min) = HR (75 beats/min) x SV (70 ml/beat)
- CO = 5250 ml/min (5.25 L/min)
- If HR increases to 150 b/min and SV increases to 120 ml/beat, then
  - CO = 150 b/min x 120 ml/beat
  - CO = 18,000 ml/min or 18 L/min (WOW is right!!)

* Factors Affecting Cardiac Output



Heart Rate

- Pulse = surge of pressure in artery
  - infants have HR of 120 bpm or more
  - young adult females avg. 72 - 80 bpm
  - young adult males avg. 64 to 72 bpm
  - HR rises again in the elderly
- Tachycardia: resting adult HR above 100
  - stress, anxiety, drugs, heart disease or ↑ body temp.
- Bradycardia: resting adult HR < 60
  - in sleep and endurance trained athletes

Regulation of Heart Rate

* Positive chronotropic factors increase heart rate
  * *Chrono* - time
* Negative chronotropic factors decrease heart rate

*Extrinsic Innervation of the

*Vital centers of medulla

1. Cardiac Center

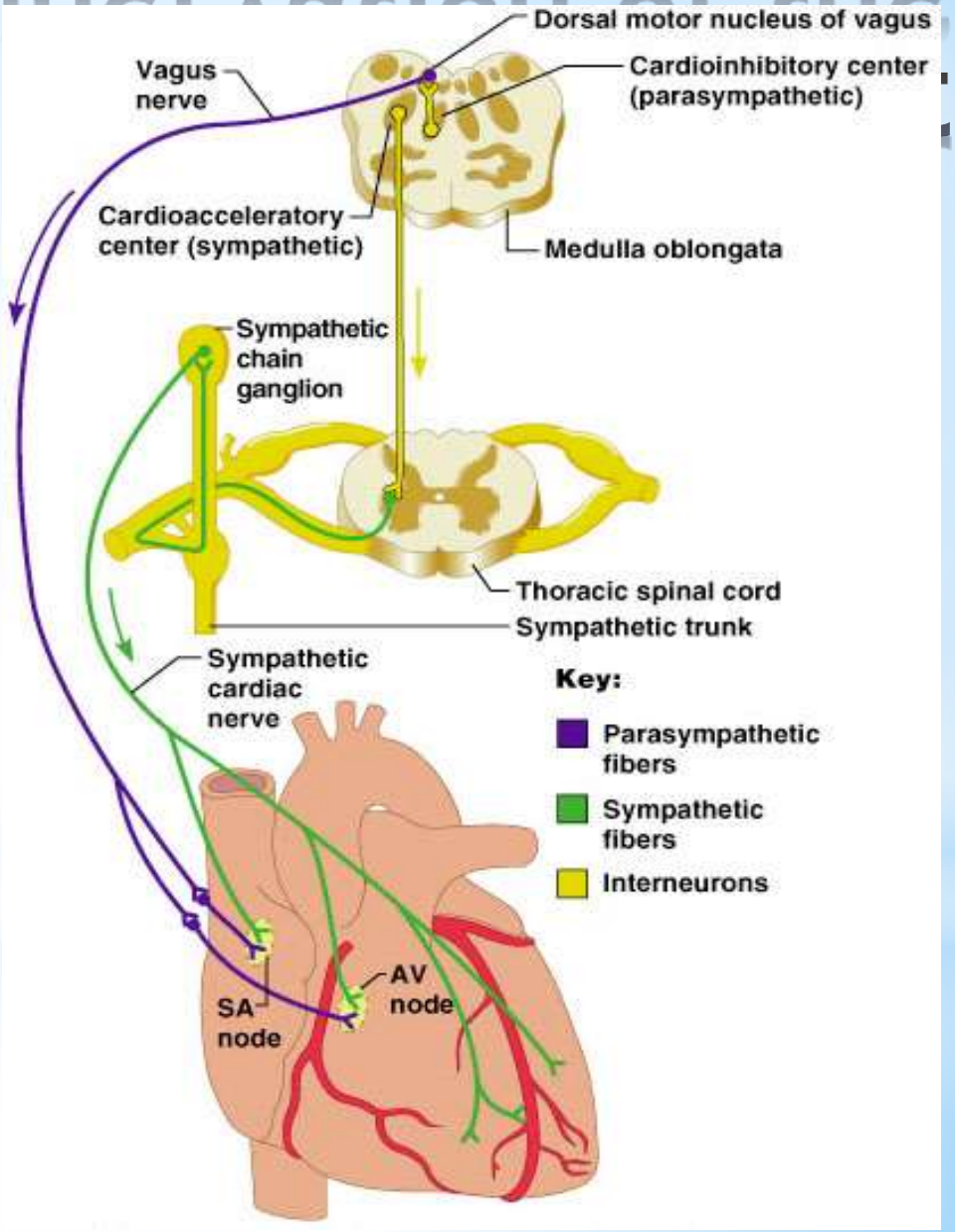
*Cardioaccelerator center

*Activates sympathetic neurons that increase HR

*Cardioinhibitory center

*Activates parasympathetic neurons that decrease HR

*Cardiac center receives input from higher centers (hypothalamus), monitoring blood pressure and dissolved gas concentrations



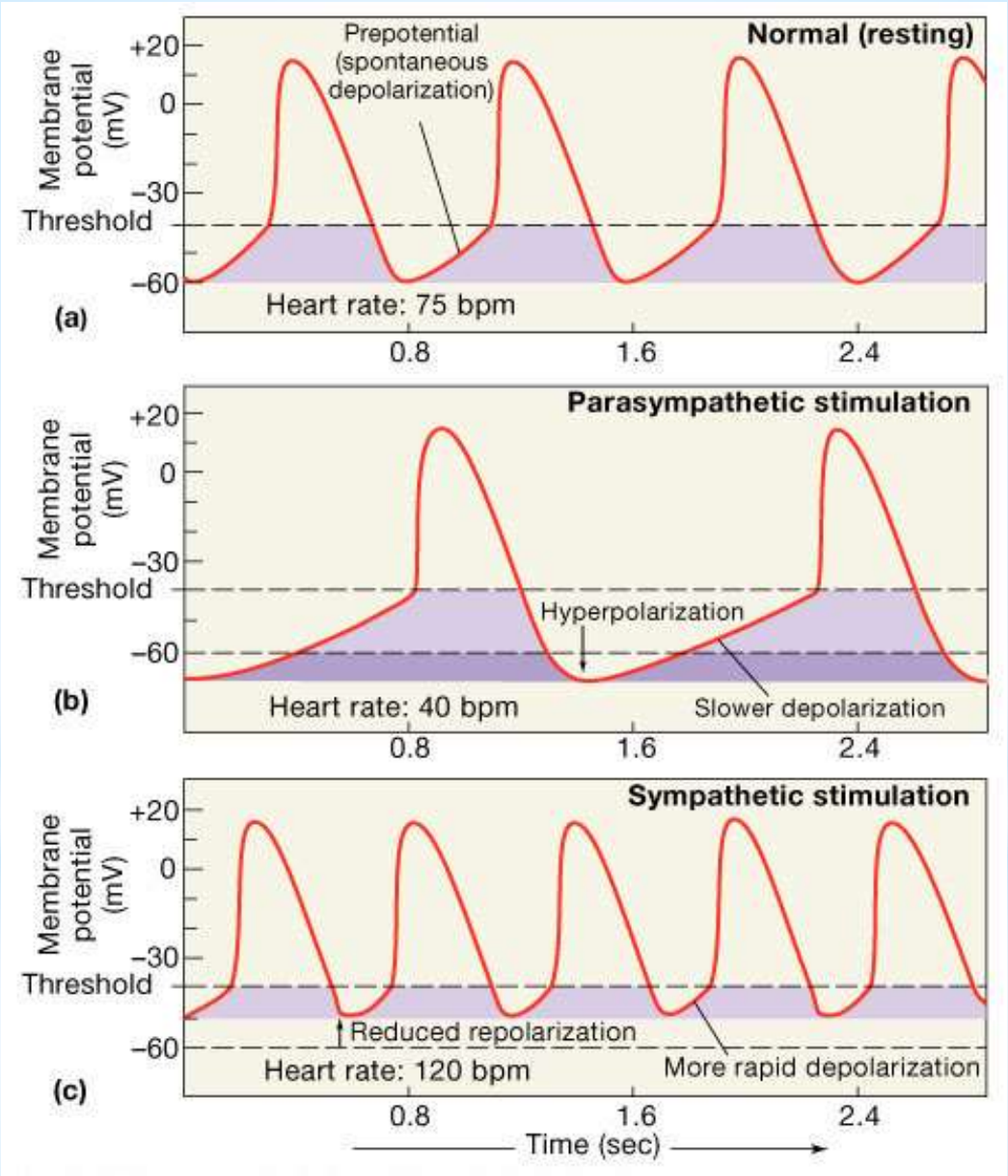
Regulation of the Heart

- **Neural regulation**
  - Parasympathetic stimulation - a negative chronotropic factor
    - Supplied by vagus nerve, decreases heart rate, acetylcholine is secreted and hyperpolarizes the heart
  - Sympathetic stimulation - a positive chronotropic factor
    - Supplied by cardiac nerves.
    - Innervate the SA and AV nodes, and the atrial and ventricular myocardium.
    - Increases heart rate and force of contraction.
    - Epinephrine and norepinephrine released.
    - Increased heart beat causes increased cardiac output. Increased force of contraction causes a lower end-systolic volume; heart empties to a greater extent. Limitations: heart has to have time to fill.
- **Hormonal regulation**
  - Epinephrine and norepinephrine from the adrenal medulla.
    - Occurs in response to increased physical activity, emotional excitement, stress

Basic heart rate established by pacemaker cells

- SA node establishes baseline (sinus rhythmn)
- Modified by ANS
- If all ANS nerves to heart are cut, heart rate jumps to about 100 b/min
  - What does this tell you about which part of the ANS is most dominant during normal period?

*Pacemaker Function



* The hormones epinephrine and thyroxine increase heart rate
* Intra- and extracellular ion concentrations must be maintained for normal heart function

* Chemical Regulation of the Heart

Regulation of Stroke Volume

- SV: volume of blood pumped by a ventricle per beat
  - SV= end diastolic volume (EDV) minus end systolic volume (ESV); SV = EDV - ESV
- EDV = end diastolic volume
  - amount of blood in a ventricle at end of diastole
- ESV = end systolic volume
  - amount of blood remaining in a ventricle after contraction
- Ejection Fraction - % of EDV that is pumped by the ventricle; important clinical parameter
  - Ejection fraction should be about 55-60% or higher

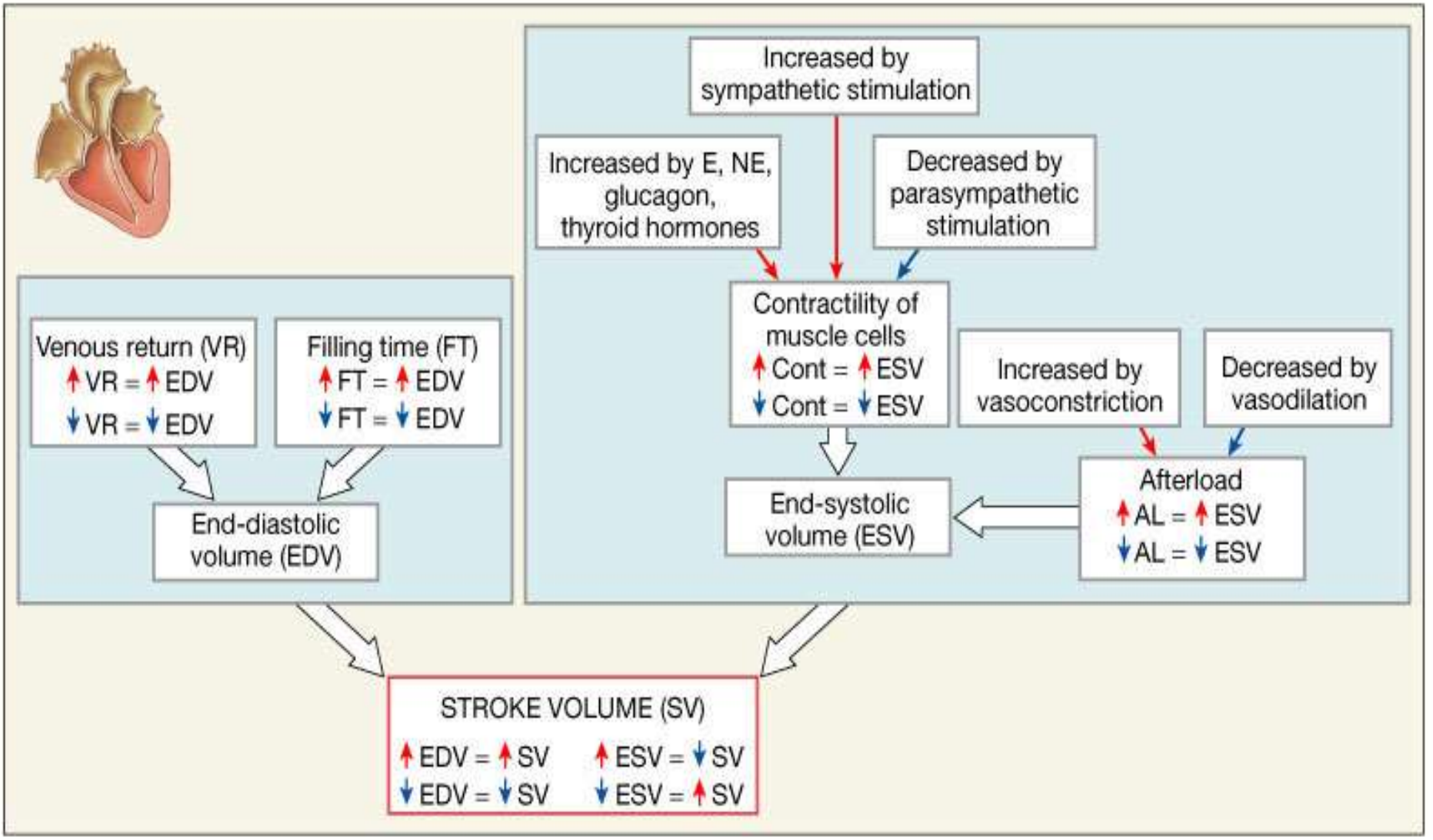
Factors Affecting Stroke Volume

- EDV - affected by
  - Venous return - vol. of blood returning to heart
  - Preload – amount ventricles are stretched by blood (=EDV)
- ESV - affected by
  - Contractility – myocardial contractile force due to factors other than EDV
  - Afterload – back pressure exerted by blood in the large arteries leaving the heart

Frank-Starling Law of the Heart

- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume; ↑EDV leads to ↑stretch of myocard.
  - ↑preload → ↑stretch of muscle → ↑force of contraction → ↑SV
  - Unlike skeletal fibers, cardiac fibers contract MORE FORCEFULLY when stretched thus ejecting MORE BLOOD (↑SV)
  - If SV is increased, then ESV is decreased!!
- Slow heartbeat and exercise increase venous return (VR) to the heart, increasing SV
  - VR changes in response to blood volume, skeletal muscle activity, alterations in cardiac output
  - ↑VR → ↑EDV and ↓in VR → ↓ in EDV
  - Any ↓ in EDV → ↓ in SV
- Blood loss and extremely rapid heartbeat decrease

*Factors Affecting Stroke Volume



Extrinsic Factors Influencing Stroke Volume

* Contractility is the increase in contractile strength, independent of stretch and EDV
* Referred to as extrinsic since the influencing factor is from some *external source*
* Increase in contractility comes from:
  * Increased sympathetic stimuli
  * Certain hormones
  * Ca^{2+} and some drugs
* Agents/factors that decrease contractility include:
  * Acidosis
  * Increased extracellular K^{+}
  * Calcium channel blockers

Effects of Autonomic Activity on Contractility

- Sympathetic stimulation
  - Release norepinephrine from symp. postganglionic fiber
  - Also, EP and NE from adrenal medulla
  - Have positive ionotropic effect
  - Ventricles contract more forcefully, increasing SV, increasing ejection fraction and decreasing ESV
- Parasympathetic stimulation via Vagus Nerve -CNX
  - Releases ACh
  - Has a negative inotropic effect
    - Hyperpolarization and inhibition
  - Force of contractions is reduced, ejection fraction decreased

*Contractility and e

* Sympathetic stimulation releases norepinephrine and initiates a cyclic AMP 2nd-messenger system

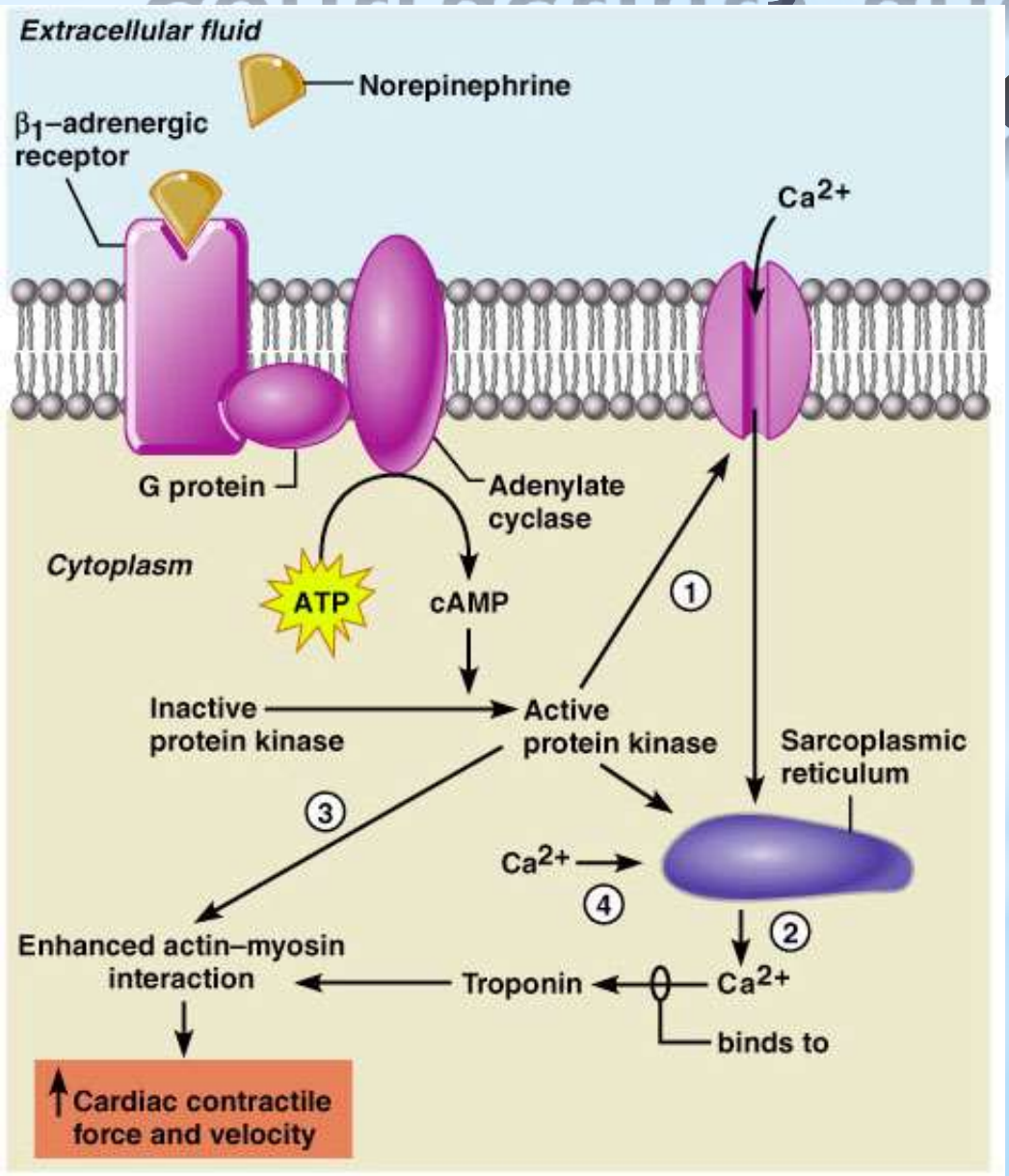


Figure 18.22

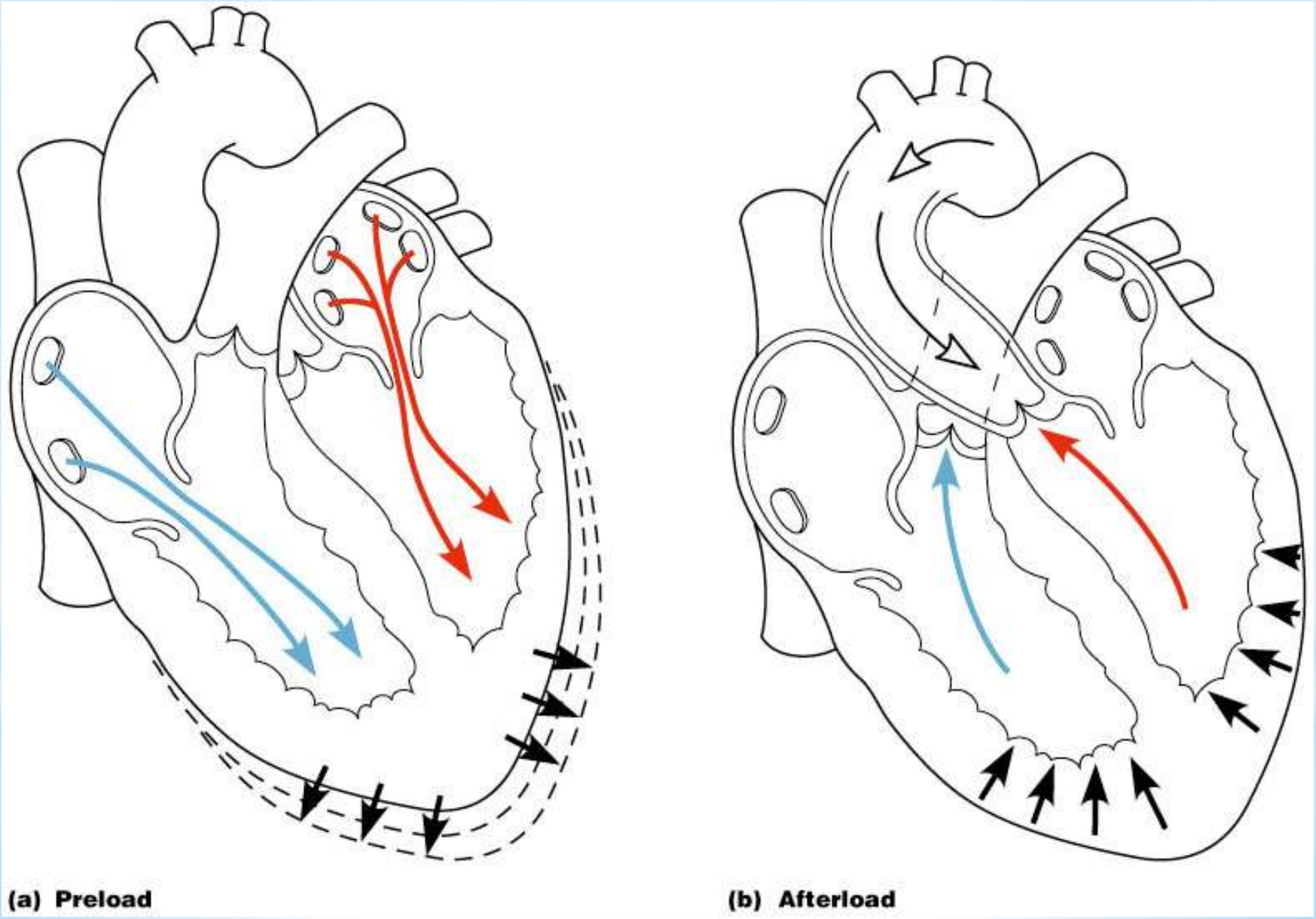
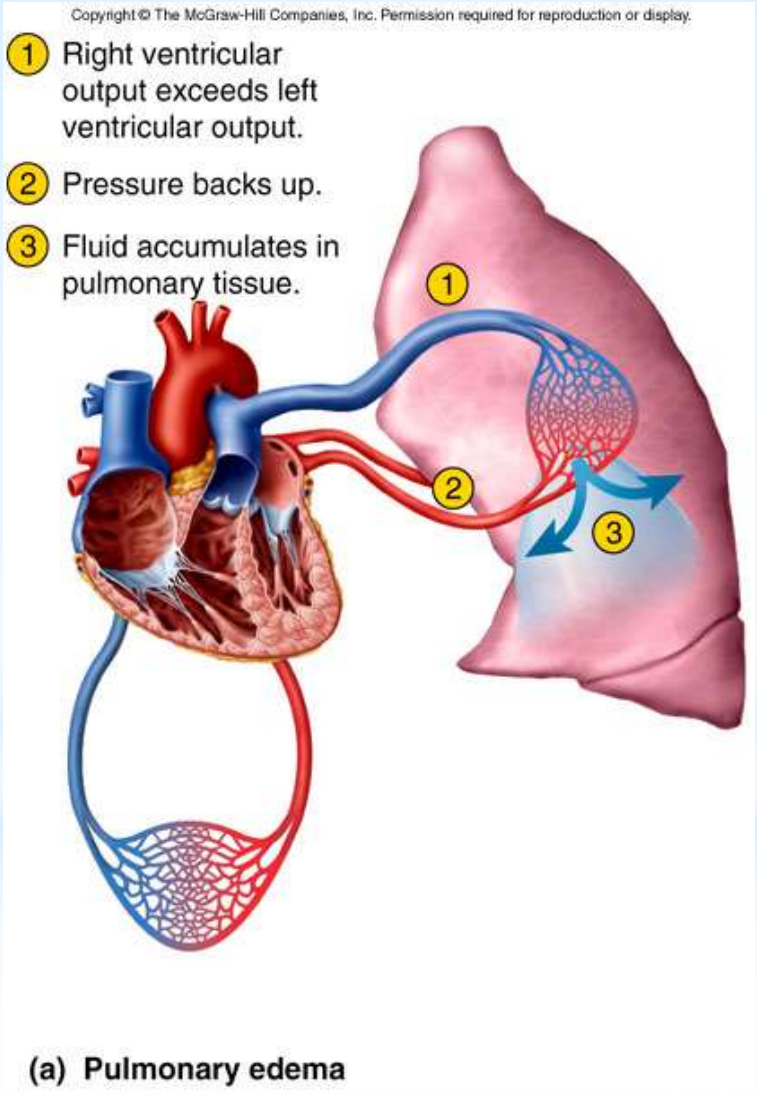


Figure 18.21

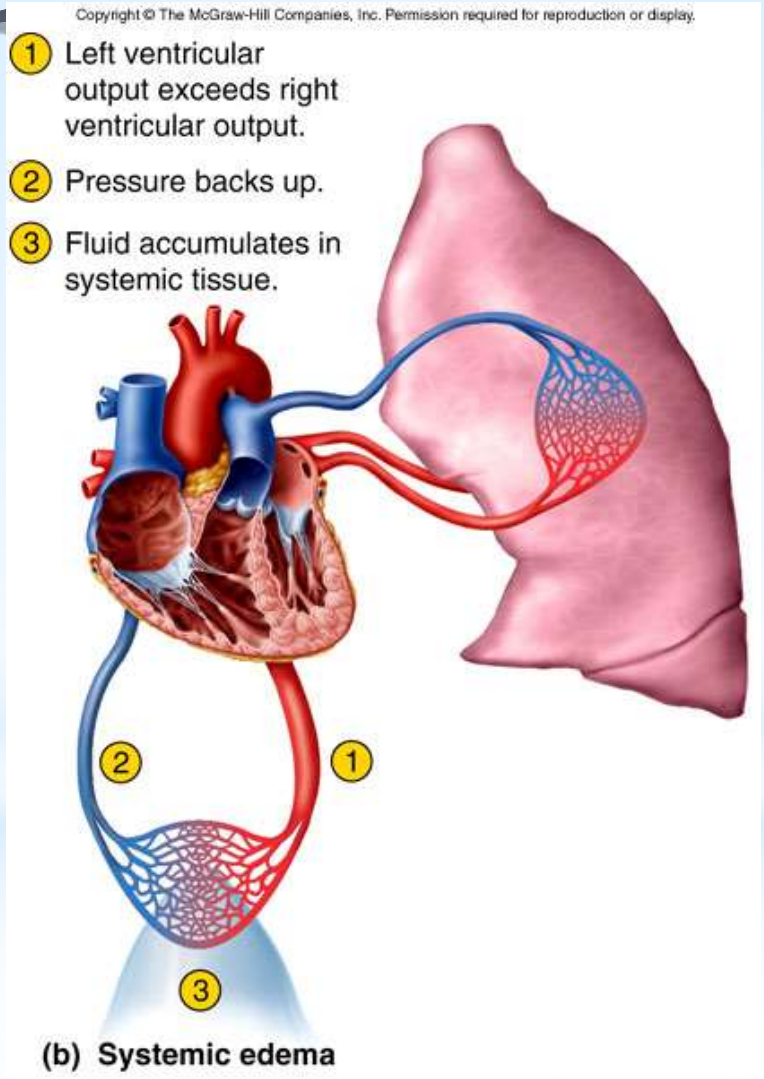
Effects of Hormones on Contractility

- Epi, NE, and Thyroxine all have positive ionotropic effects and thus ↑contractility
- Digitalis elevates intracellular Ca^{++} concentrations by interfering with its removal from sarcoplasm of cardiac cells
- Beta-blockers (*propanolol*, *timolol*) block beta-receptors and prevent sympathetic stimulation of heart (neg. chronotropic effect)

Unbalanced Ventricular Output



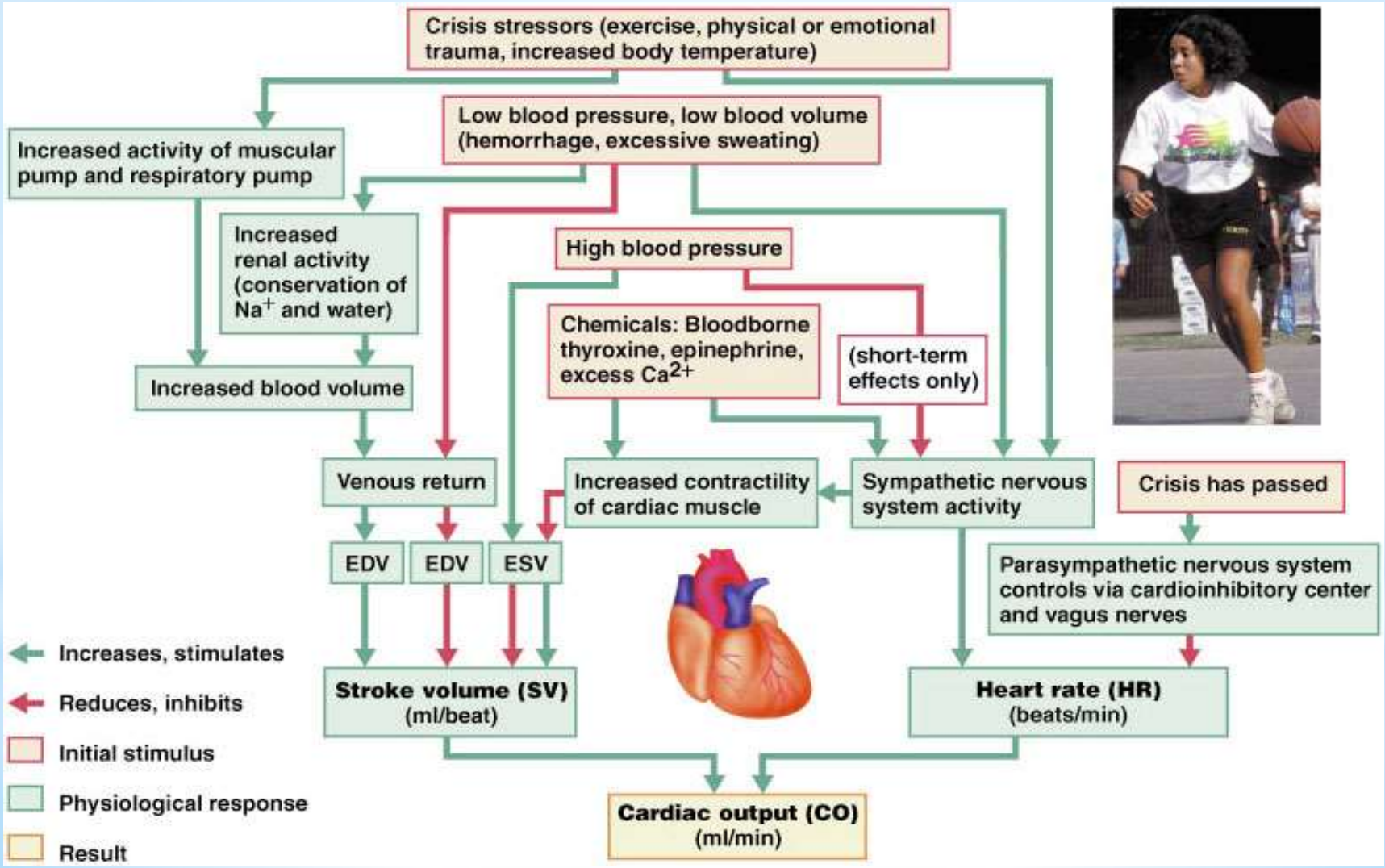
Unbalanced Ventricular Output



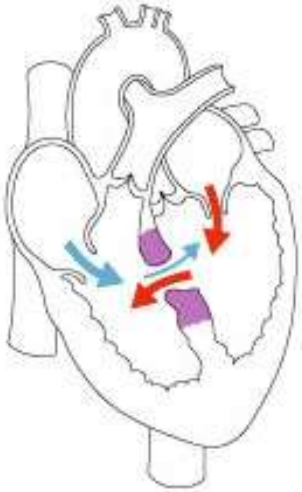
Exercise and Cardiac Output

* Proprioceptors
  * HR ↑ at beginning of exercise due to signals from joints, muscles
* Venous return
  * muscular activity ↑ venous return causes ↑ SV
* ↑ HR and ↑ SV cause ↑CO
* Exercise produces ventricular hypertrophy
  * ↑ SV allows heart to beat more slowly at rest
  * ↑ cardiac reserve

Factors Involved in Regulation of Cardiac Output

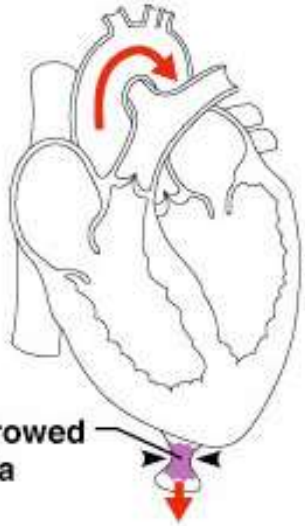


*Examples of Congenital Heart Defects



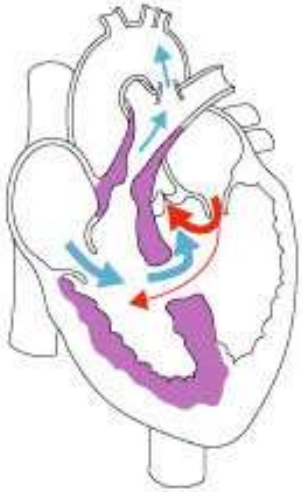
Occurs in about 1 in every 500 births

(a) Ventricular septal defect. The superior part of the interventricular septum fails to form; thus, blood mixes between the two ventricles, but because the left ventricle is stronger, more blood is shunted from left to right.



Occurs in about 1 in every 1500 births

(b) Coarctation of the aorta. A part of the aorta is narrowed, increasing the workload on the left ventricle.



Occurs in about 1 in every 2000 births

(c) Tetralogy of Fallot. Multiple defects (*tetra* = four): Pulmonary trunk too narrow and pulmonary valve stenosed, resulting in a hypertrophied right ventricle; ventricular septal defect; aorta opens from both ventricles; wall of right ventricle thickened from overwork.

Figure 18.25