Semey State Medical University

SIW

Physiology of the Heart

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Plan:

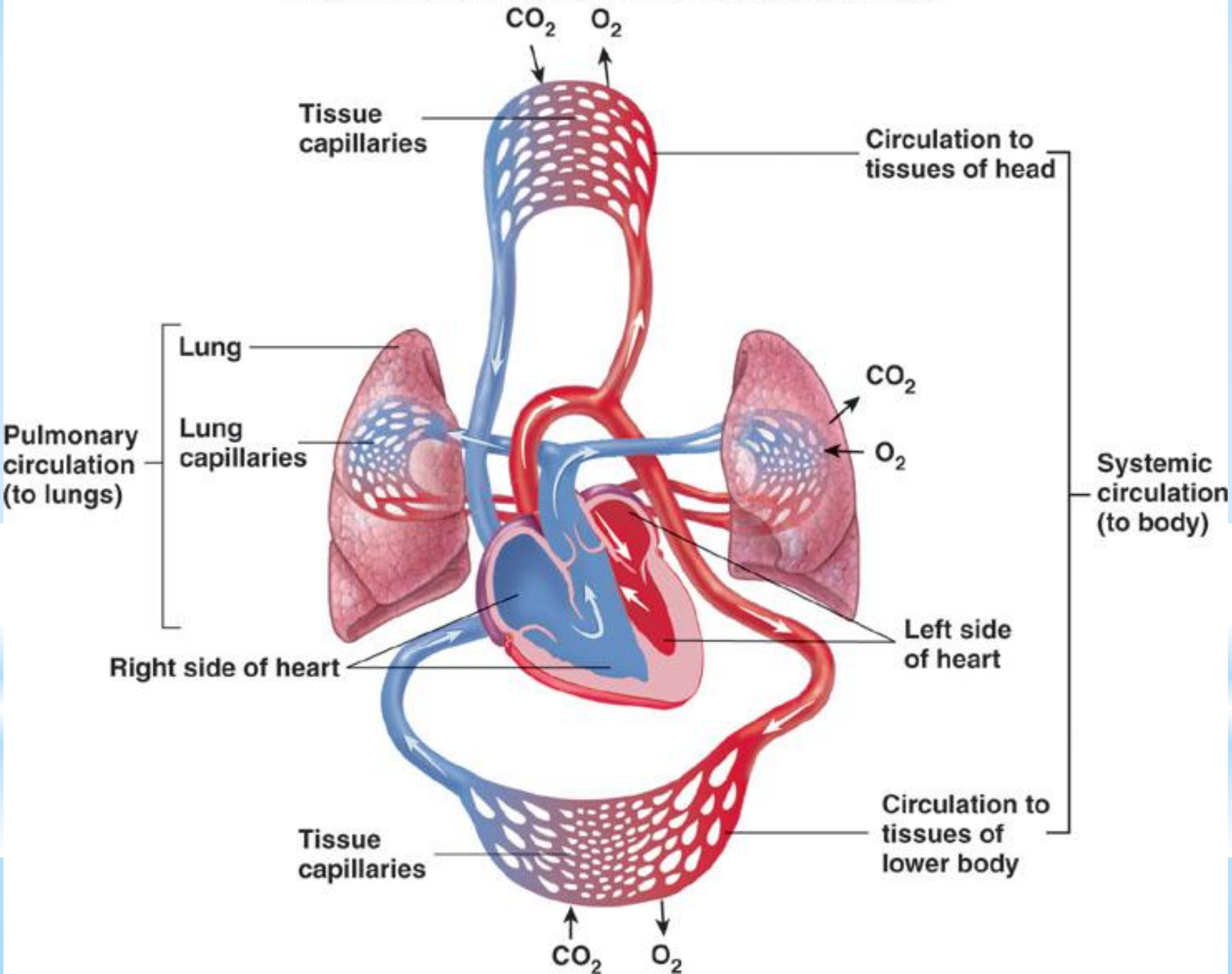
* Functions of the Heart
* Conducting System of Heart
* An Electrocardiogram
* The Cardiac Cycle
* Regulation of the Heart

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

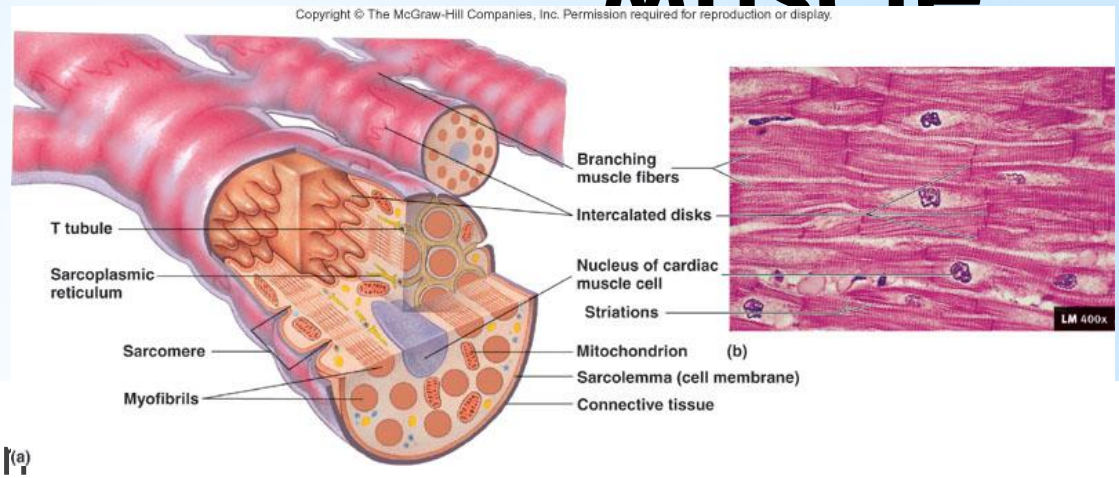
The cardiovascular system is divided into two circuits

- 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.
  * Cell membranes interdigitate
  * Desmosomes hold cells together
  * Gap junctions allow action potentials to move from one cell to the next.
* 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

Heart chambers and valves

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

Cardiac Muscle Contraction

- Heart muscle:
  - Is stimulated by nerves and is self-excitable (automaticity)
  - Contracts as a unit; no *motor units*
  - Has a long (250 ms) absolute refractory period
- Cardiac muscle contraction is similar to skeletal muscle contraction, i.e., sliding-filaments

Differences Between Skeletal and Cardiac Muscle Physiology

- Action Potential
  - Cardiac: Action potentials conducted from cell to cell.
  - Skeletal, action potential conducted along length of single fiber
- Rate of Action Potential Propagation
  - Slow in cardiac muscle because of gap junctions and small diameter of fibers.
  - Faster in skeletal muscle due to larger diameter fibers.
- Calcium release
  - Calcium-induced calcium release (CICR) in cardiac
    - Movement of extracellular Ca^{2+} through plasma membrane and T tubules into sarcoplasm stimulates release of Ca^{2+} from sarcoplasmic reticulum
  - Action potential in T-tubule stimulates Ca^{++} release from

*The Action Potential in Skeletal and Cardiac Muscle

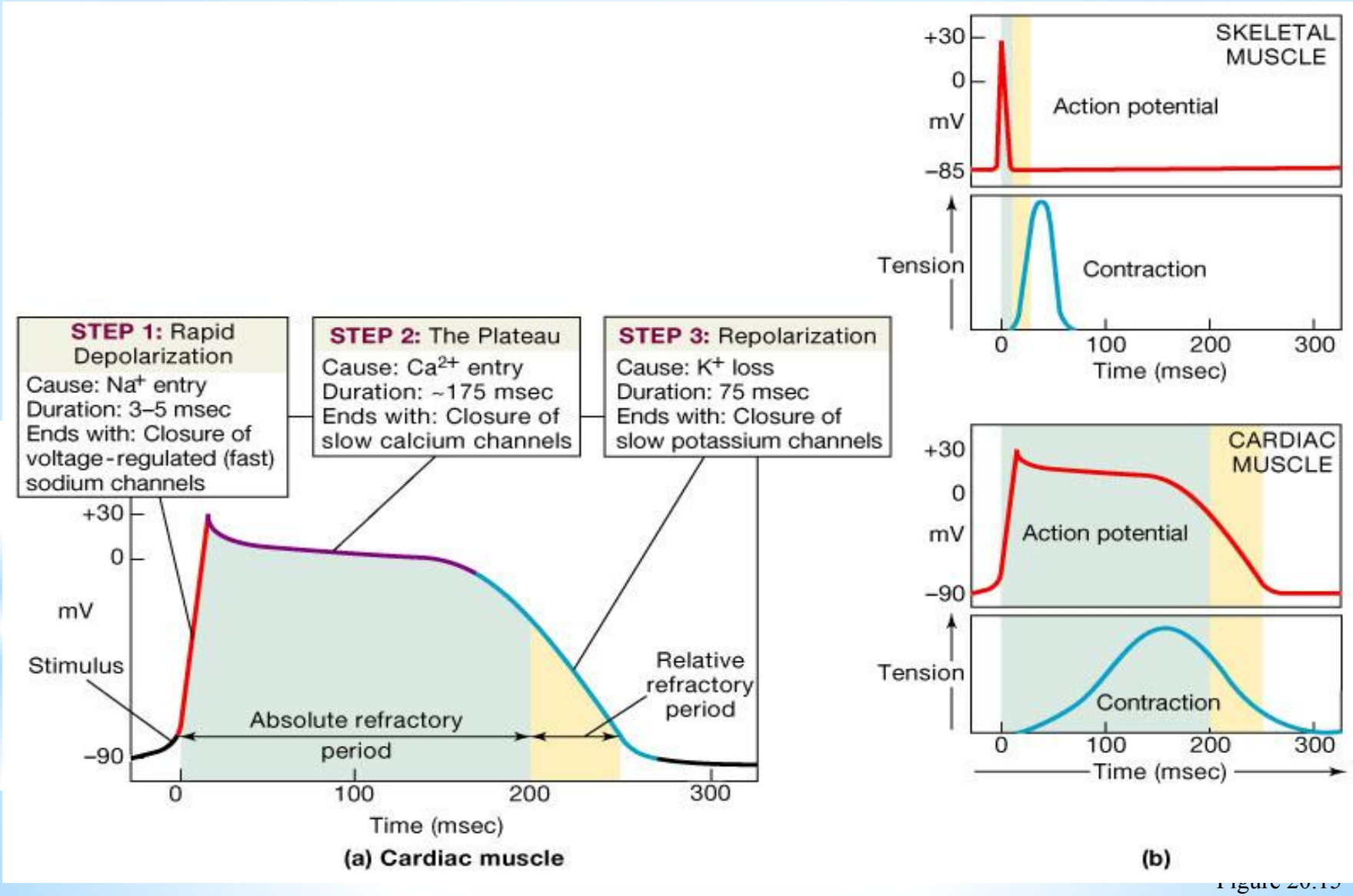
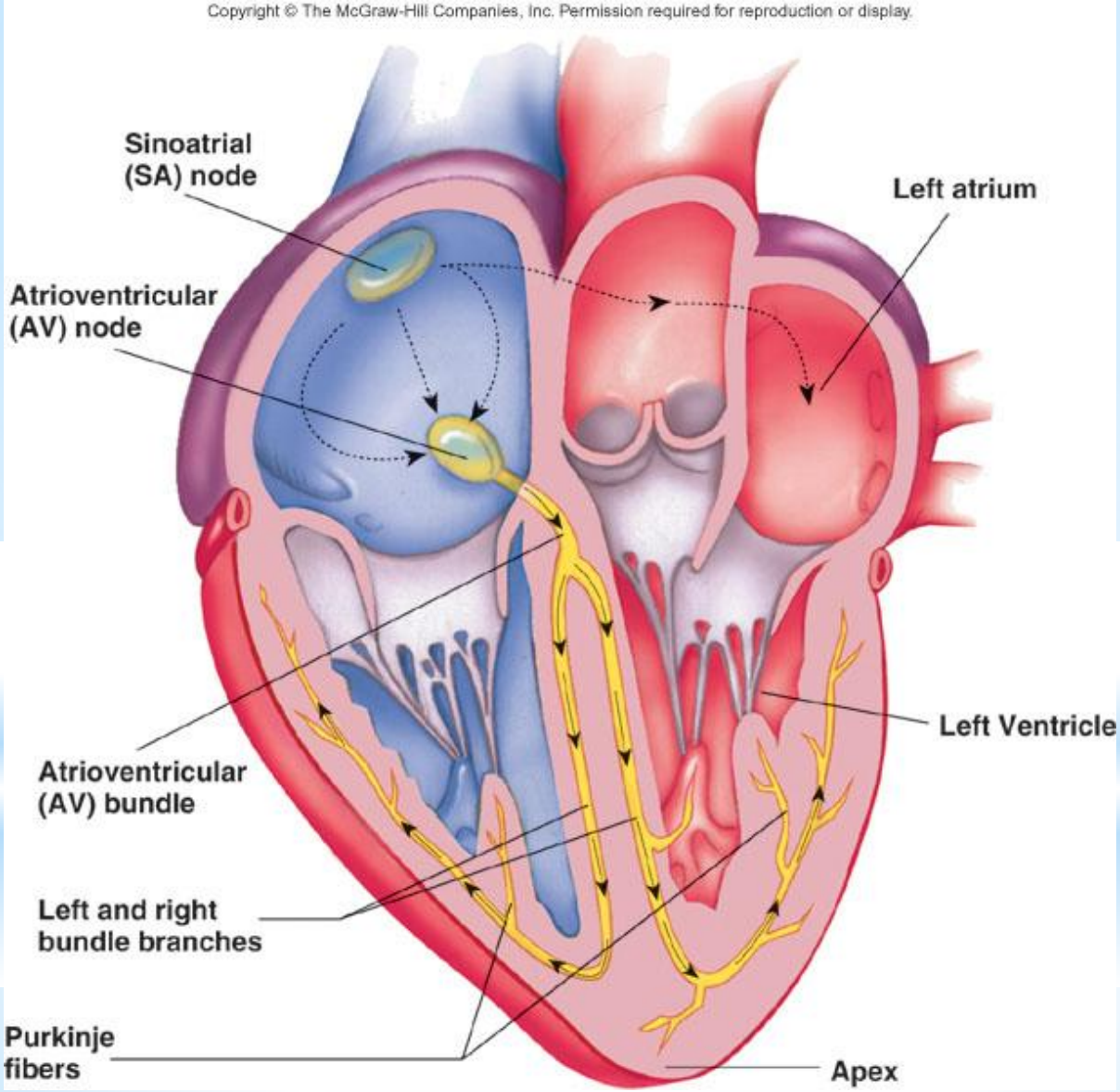


Figure 20.19

* Electrical Properties of Myocardial Fibers

1. Rising phase of action potential
   - Due to opening of fast Na^+ channels
2. Plateau phase
   - Closure of sodium channels
   - Opening of calcium channels
   - Slight increase in K^+ permeability
   - Prevents summation and thus tetanus of cardiac muscle
3. Repolarization phase
   - Calcium channels closed
   - Increased K^+ permeability

* 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)*.
  - Action potentials pass to atrial muscle cells and to the AV node
- **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
- **AV bundle**: passes through hole in cardiac skeleton to reach interventricular septum
- **Right and left bundle branches**: extend beneath endocardium to apices of right and left ventricles
- **Purkinje fibers**:
  - Large diameter cardiac muscle cells with few myofibrils.
  - Many gap junctions.

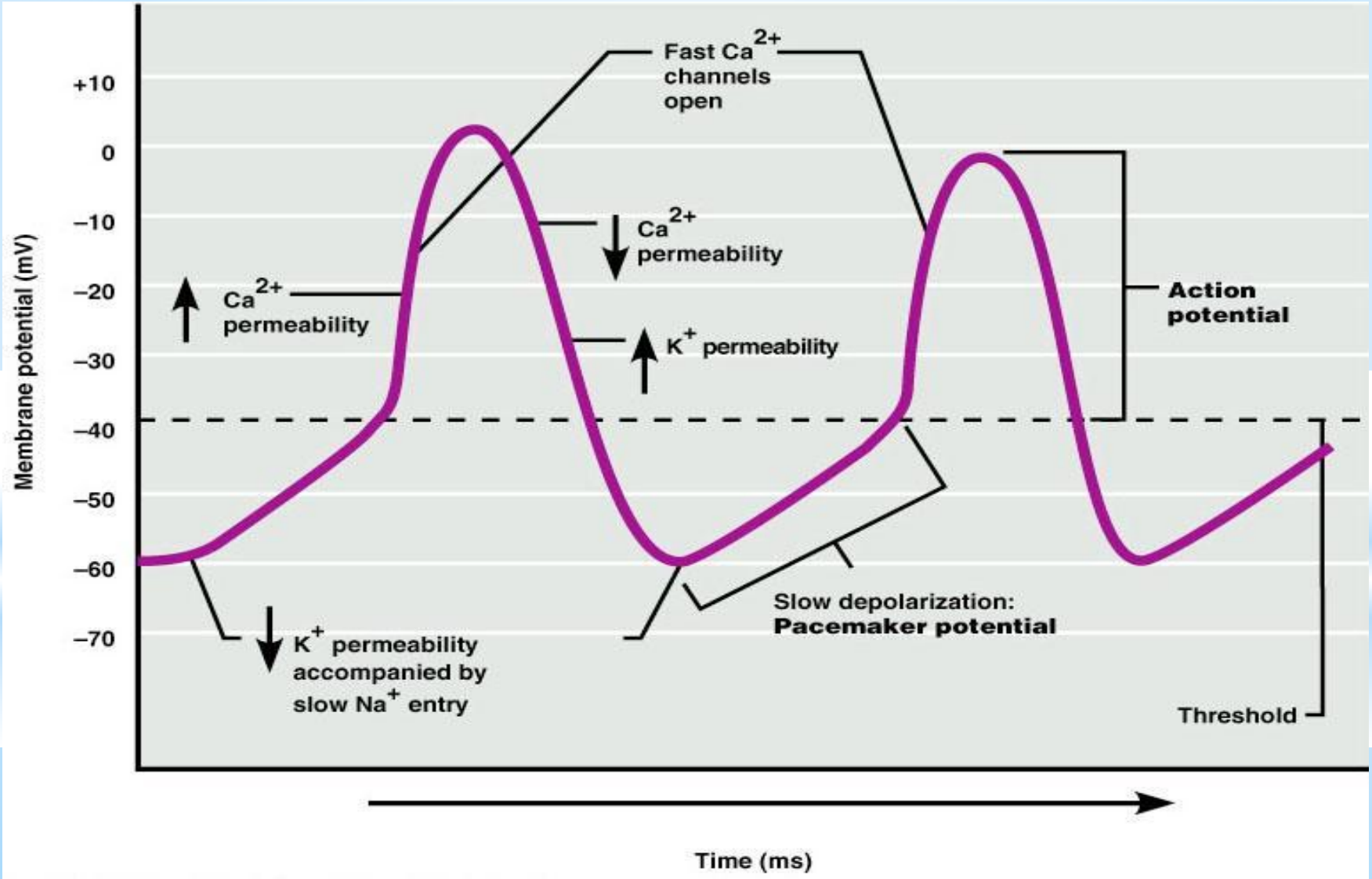
Heart Physiology: Intrinsic Conduction System

- Autorhythmic cells:
  - Initiate action potentials
  - Have unstable resting potentials called pacemaker potentials
  - Use calcium influx (rather than sodium) for rising phase of the action potential

*Depolarization of SA Node

* SA node - no stable resting membrane potential
* Pacemaker potential
  * gradual depolarization *from -60 mV*, slow influx of Na^+
* Action potential
  * occurs at threshold of *-40 mV*
  * depolarizing phase *to 0 mV*
    * fast Ca^{2+} channels open, (Ca^{2+} in)
  * repolarizing phase
    * K^+ channels open, (K^+ out)
    * *at -60 mV* K^+ channels close, pacemaker potential starts over
* Each depolarization creates one heartbeat

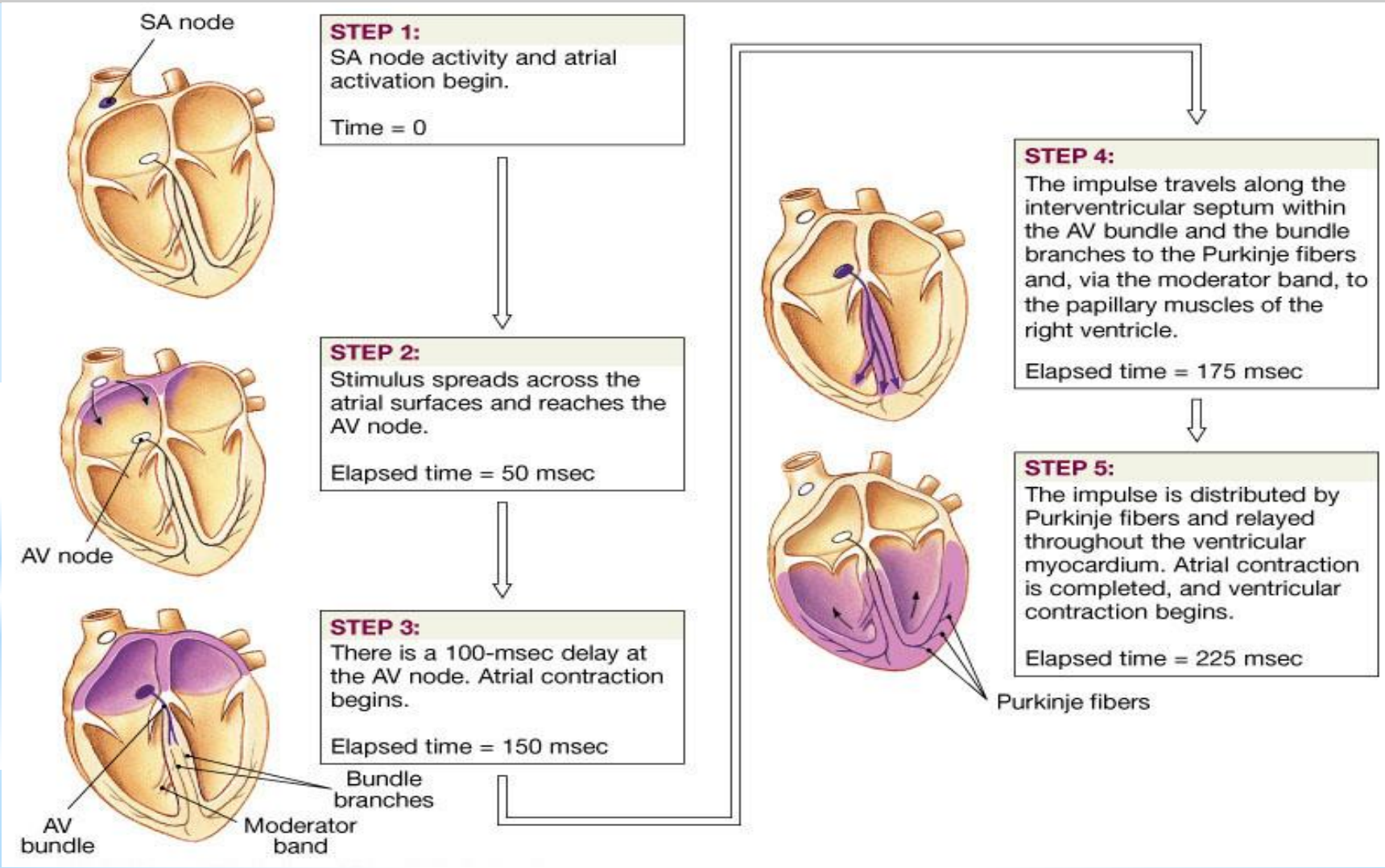
*Pacemaker and Action Potentials of the Heart



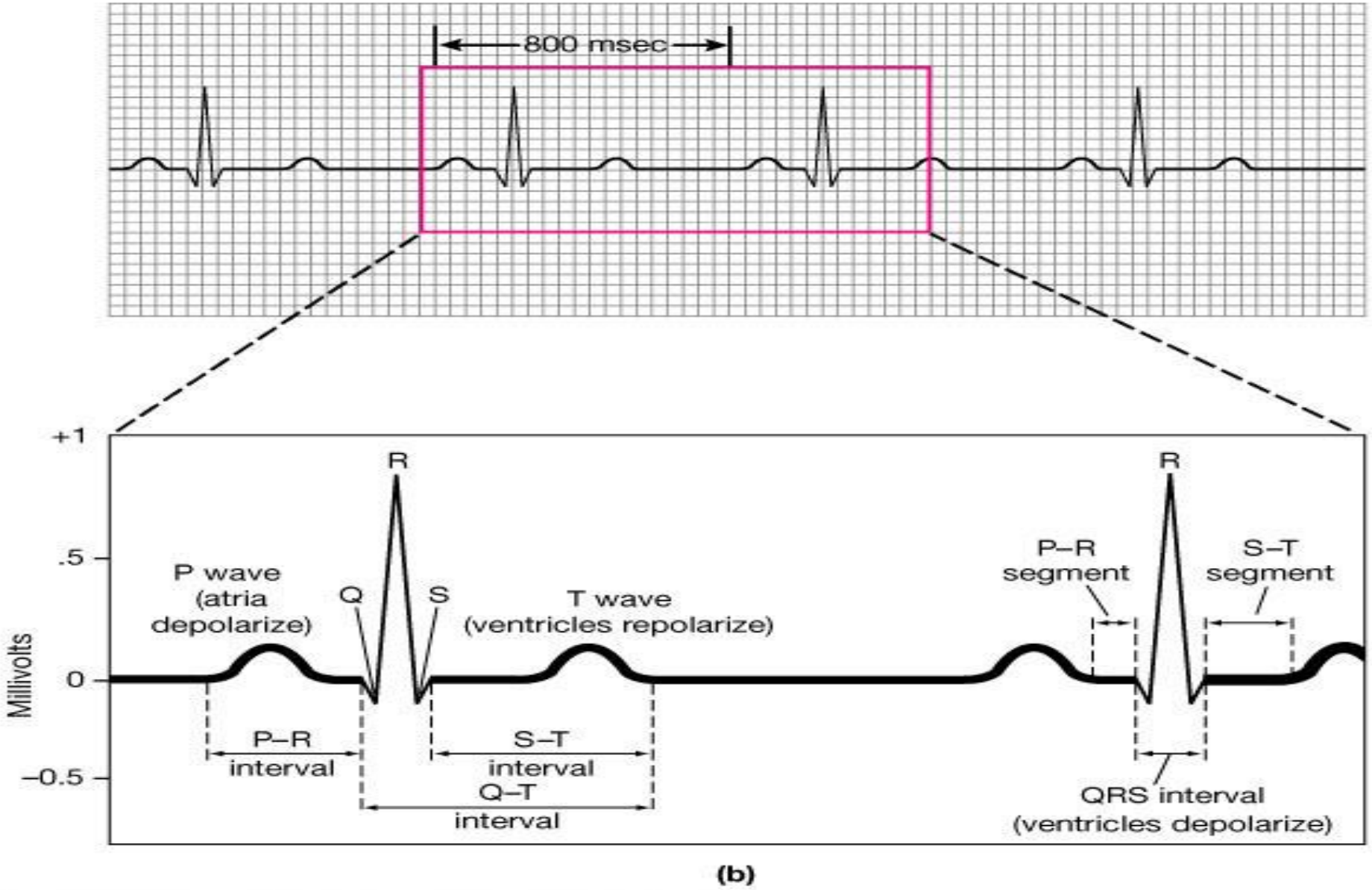
* Sinoatrial (SA) node generates impulses about 75 times/minute
* Atrioventricular (AV) node delays the impulse approximately 0.1 second
* Impulse passes from atria to ventricles via the atrioventricular bundle (bundle of His) to the Purkinje fibers and finally to the myocardial fibers

* Heart Physiology: Sequence of Excitation

*Impulse Conduction through the Heart

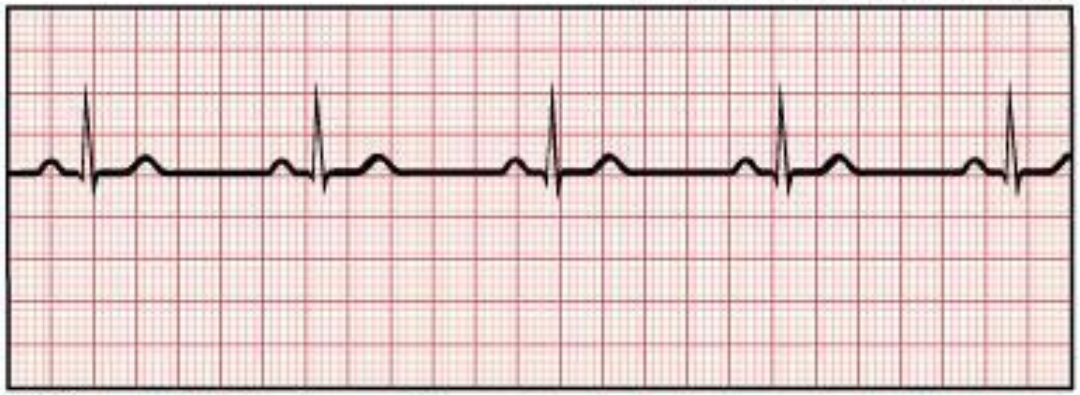


*An Electrocardiogram

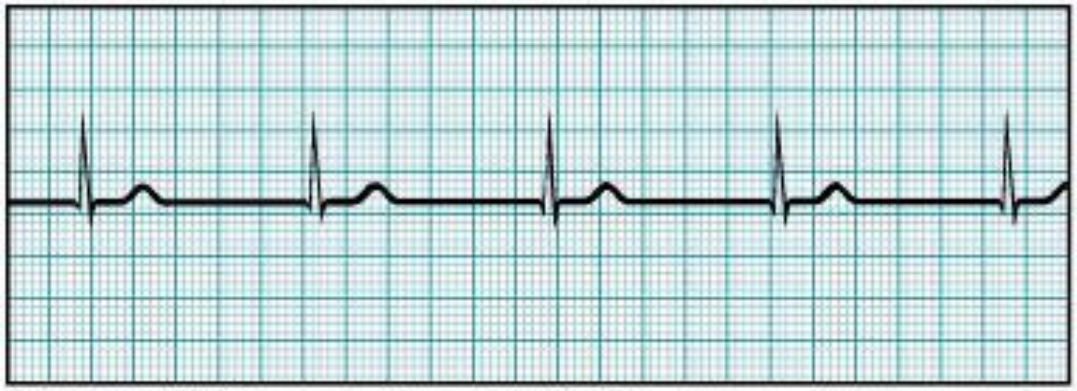


Electrocardiogram

- 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
- **PR interval** or PQ interval: 0.16 sec
  - Extends from start of atrial depolarization to start of ventricular depolarization (QRS complex) contract and begin to relax
  - Can indicate damage to conducting pathway or AV node if greater than 0.20 sec (200 msec)
- **Q-T interval**: time required for ventricles to undergo a single cycle of depolarization and repolarization
  - Can be lengthened by electrolyte disturbances, conduction problems, coronary ischemia, myocardial damage



(a) Sinus rhythm (normal)



(b) Nodal rhythm – no SA node activity

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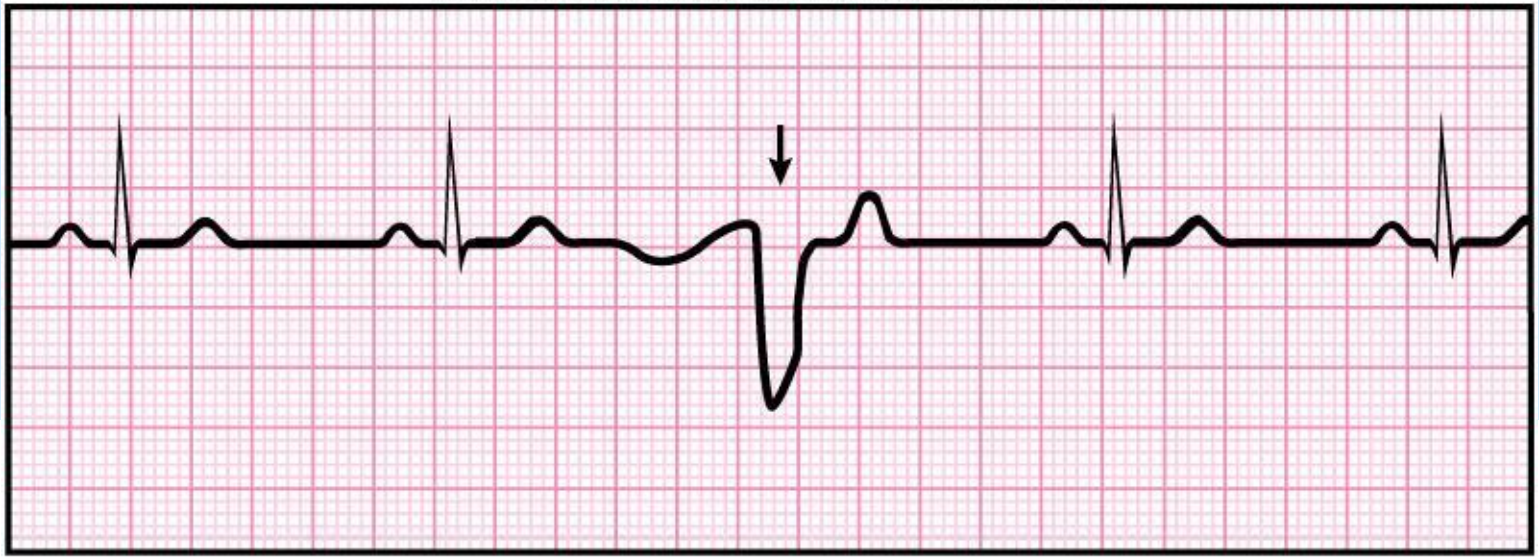
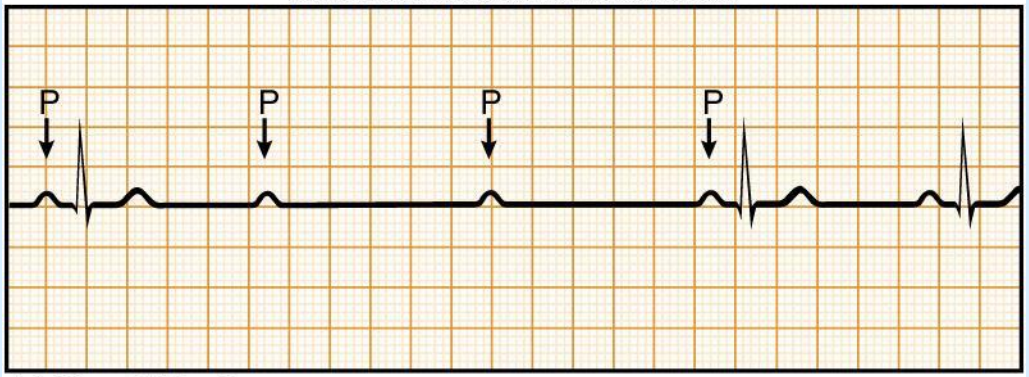


(d) Premature ventricular contraction

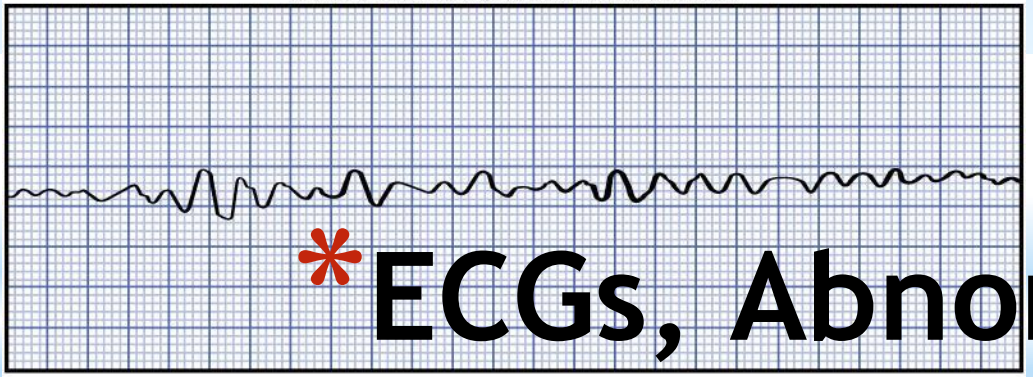
ECGs, Abnormal

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

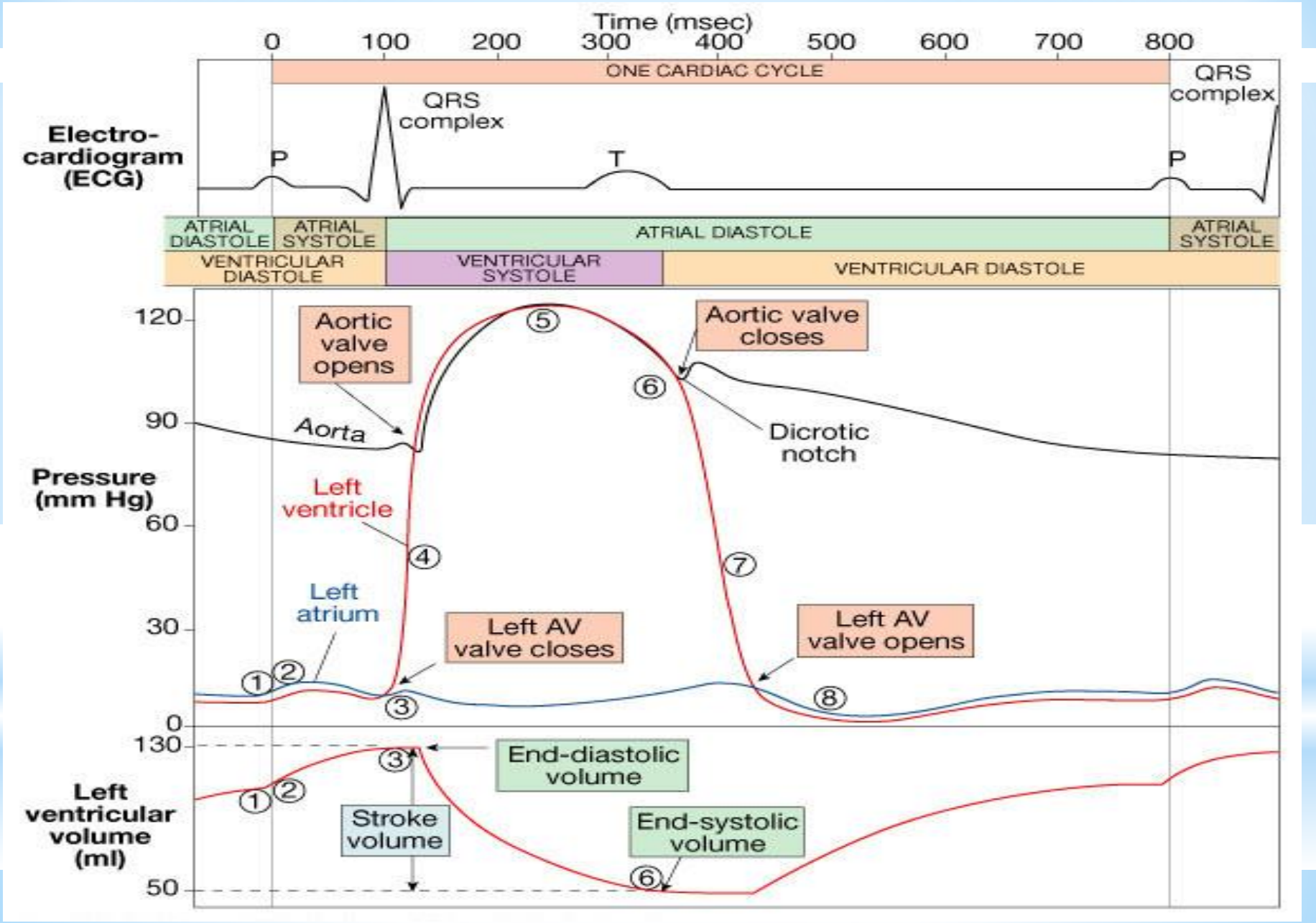
- Atrial diastole and systole -
  - Blood flows into and passively out of atria (80% of total)
    - AV valves open
  - Atrial systole pumps only about 20% of blood into ventricles
- Ventricular filling: mid-to-late diastole
  - Heart blood pressure is low as blood enters atria and flows into ventricles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

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



Pressure and Volume Relationships in the Cardiac Cycle

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 Volume

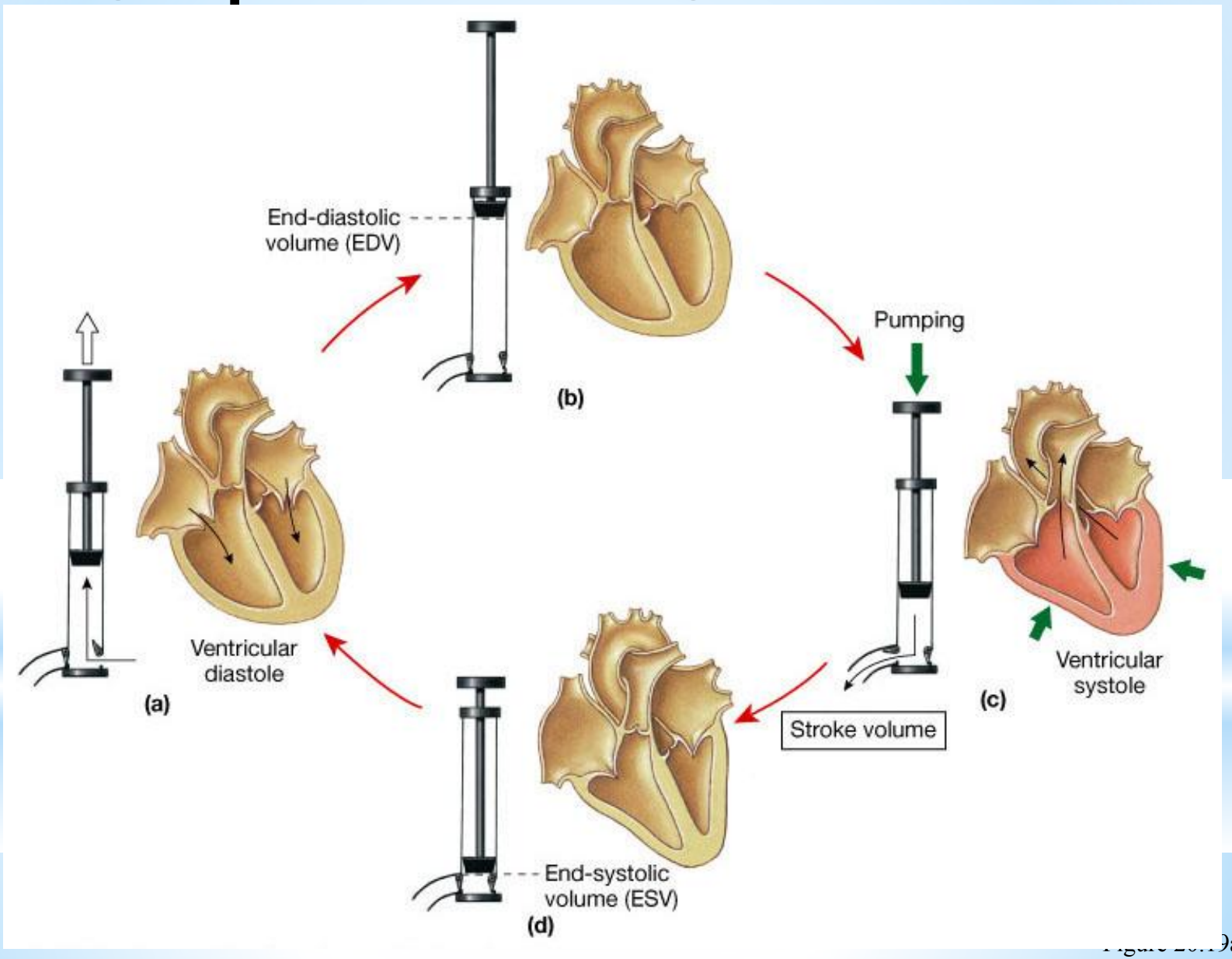
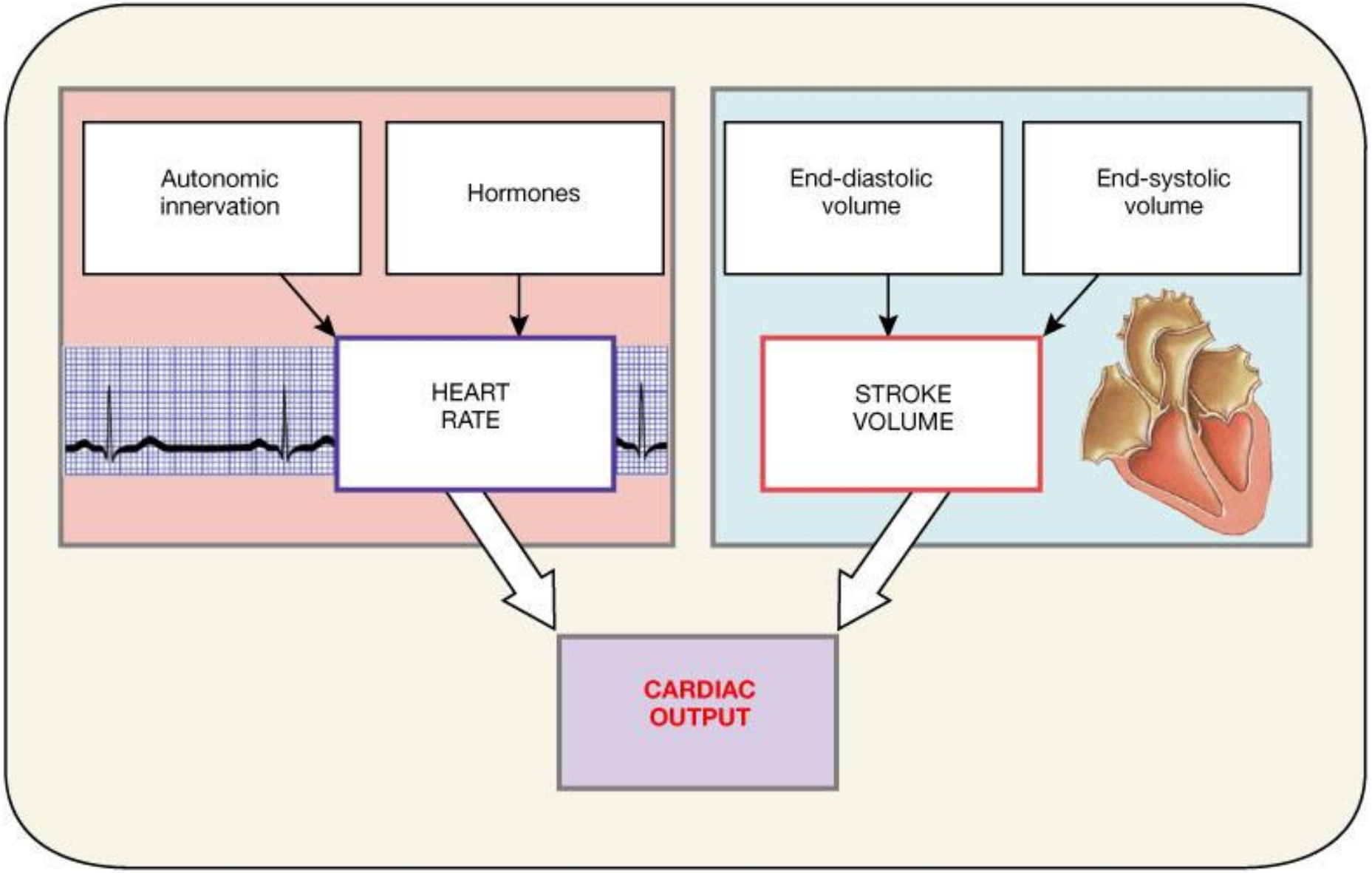


Figure 20.19a-d

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



*Extrinsic Innervation of the

*Vital centers of medulla

1. Cardiac Center

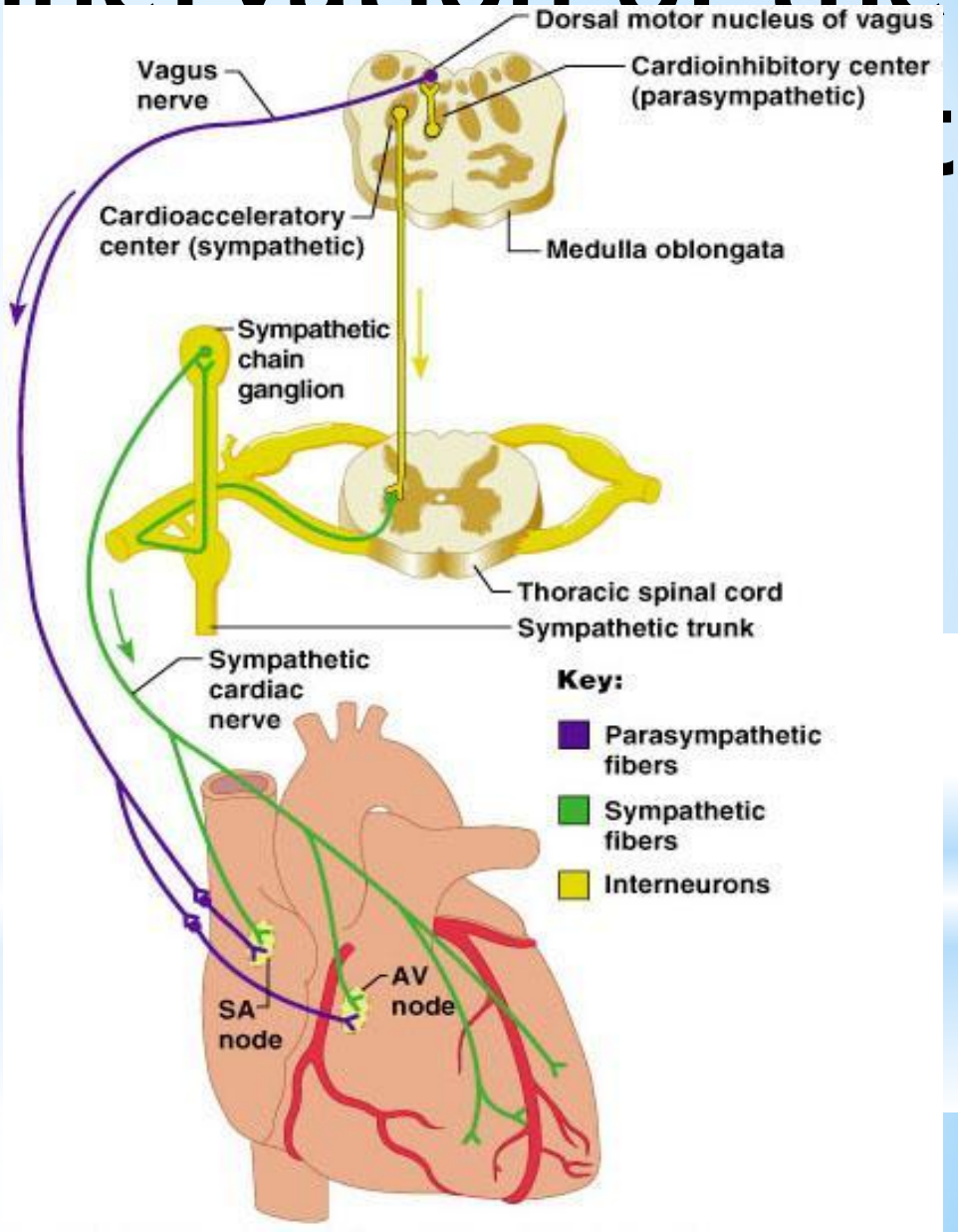
*_Cardioaccelerator center_

*Activates sympathetic neurons that increase HR

*_Cardioinhibitory center_

*Activates parasympathetic neurons that decrease HR

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



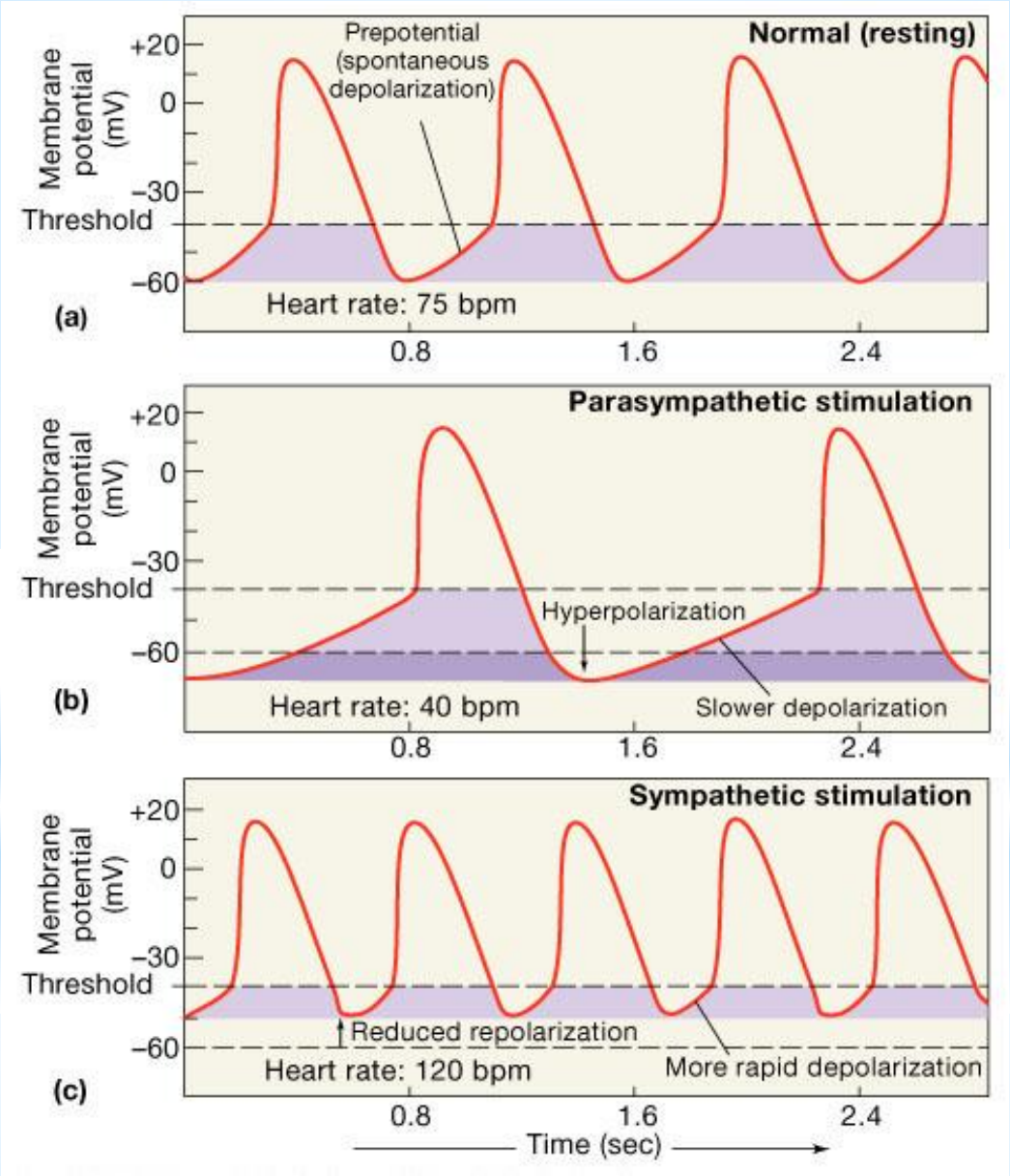
Regulation of the Heart

- **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

* 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?

* Basic heart rate established by pacemaker cells

*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

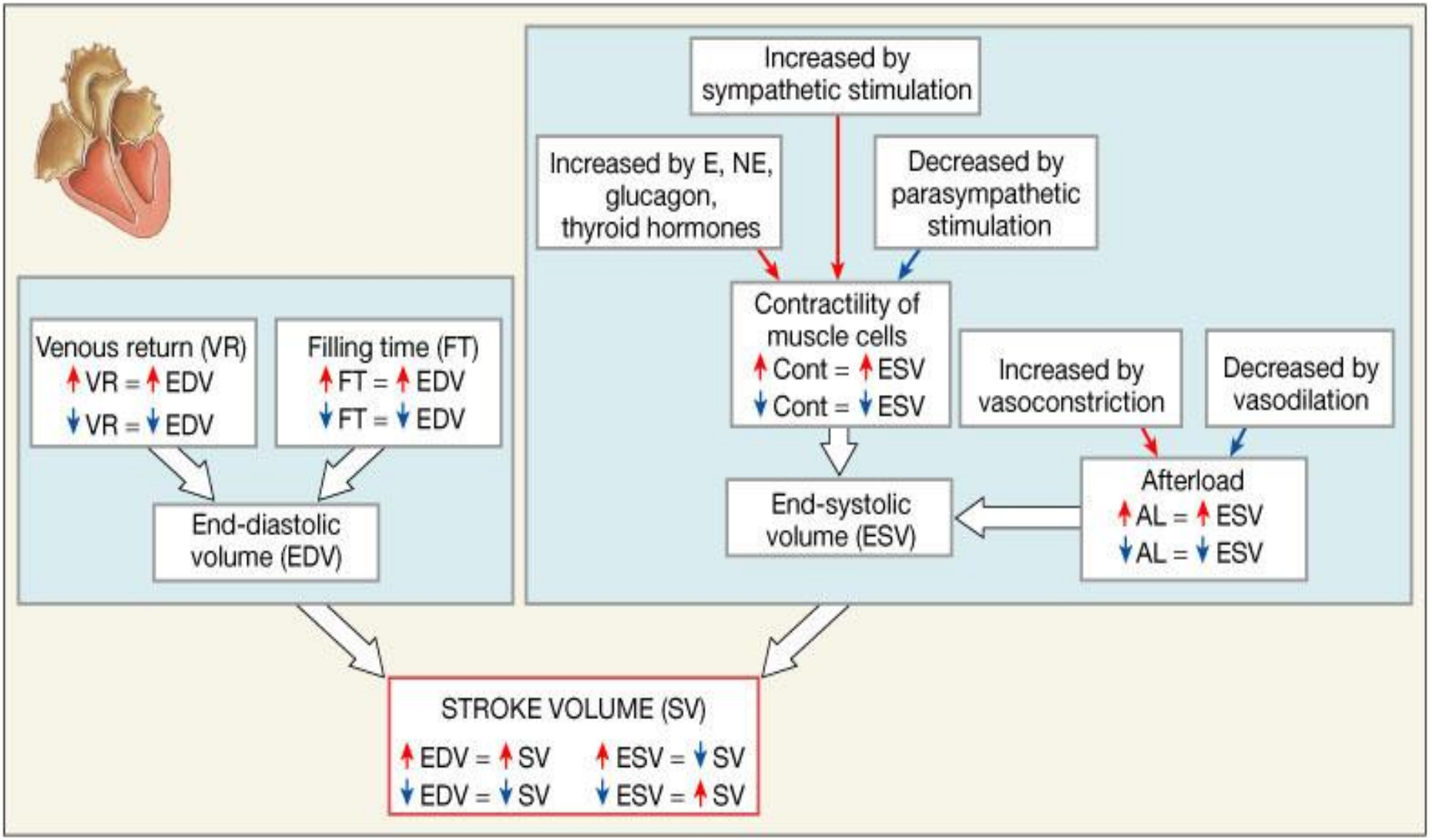
Factors Affecting Stroke Volume

- 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 SV

*Factors Affecting Stroke Volume



Extrinsic Factors Influencing Stroke Volume

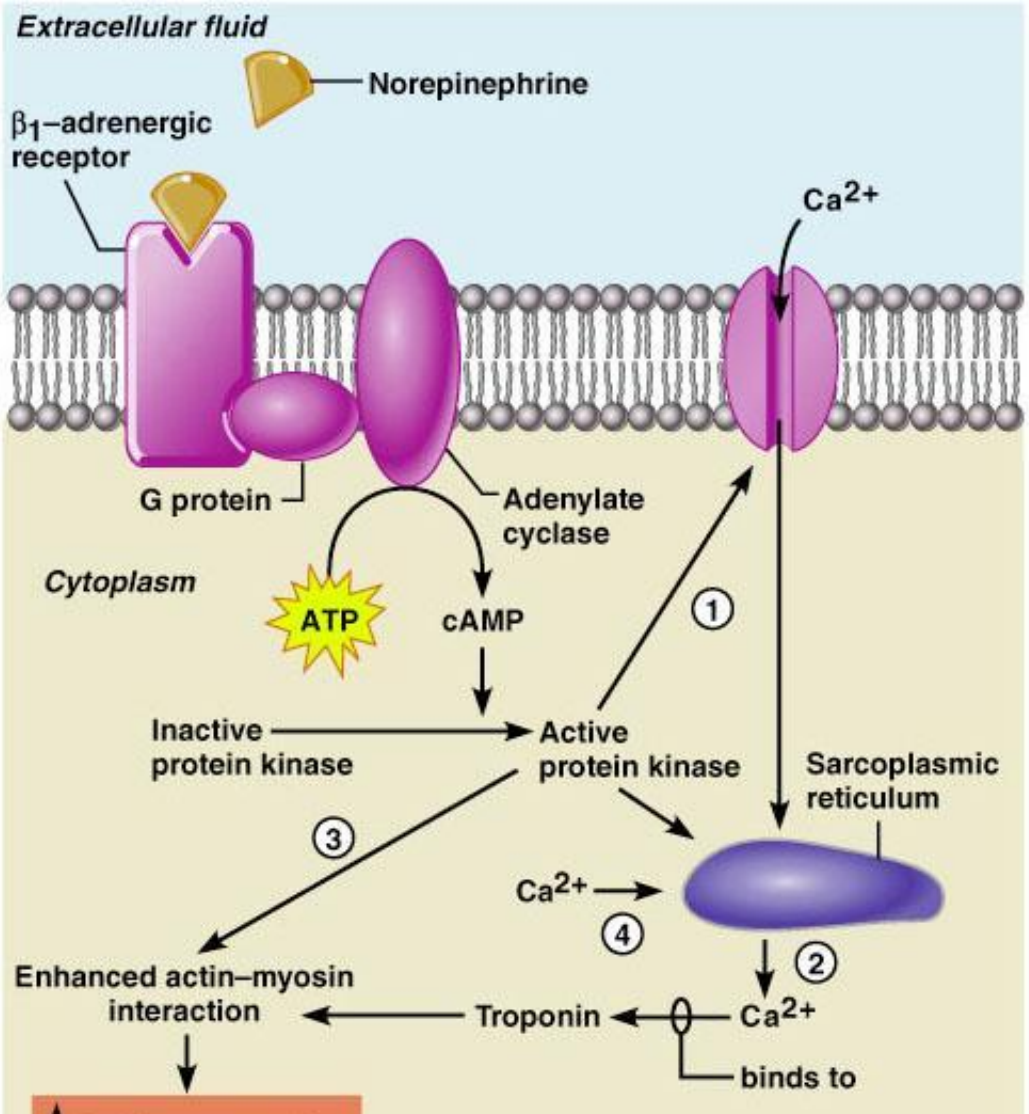
- Contractility is the increase in contractile strength, independent of stretch and EDV
- Referred to as extrinsic since the influencing factor is from some *external source*
- Increase in contractility comes from:
  - Increased sympathetic stimuli
  - Certain hormones
  - Ca^{2+} and some drugs
- Agents/factors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

*Contractility and Norepinephrine

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



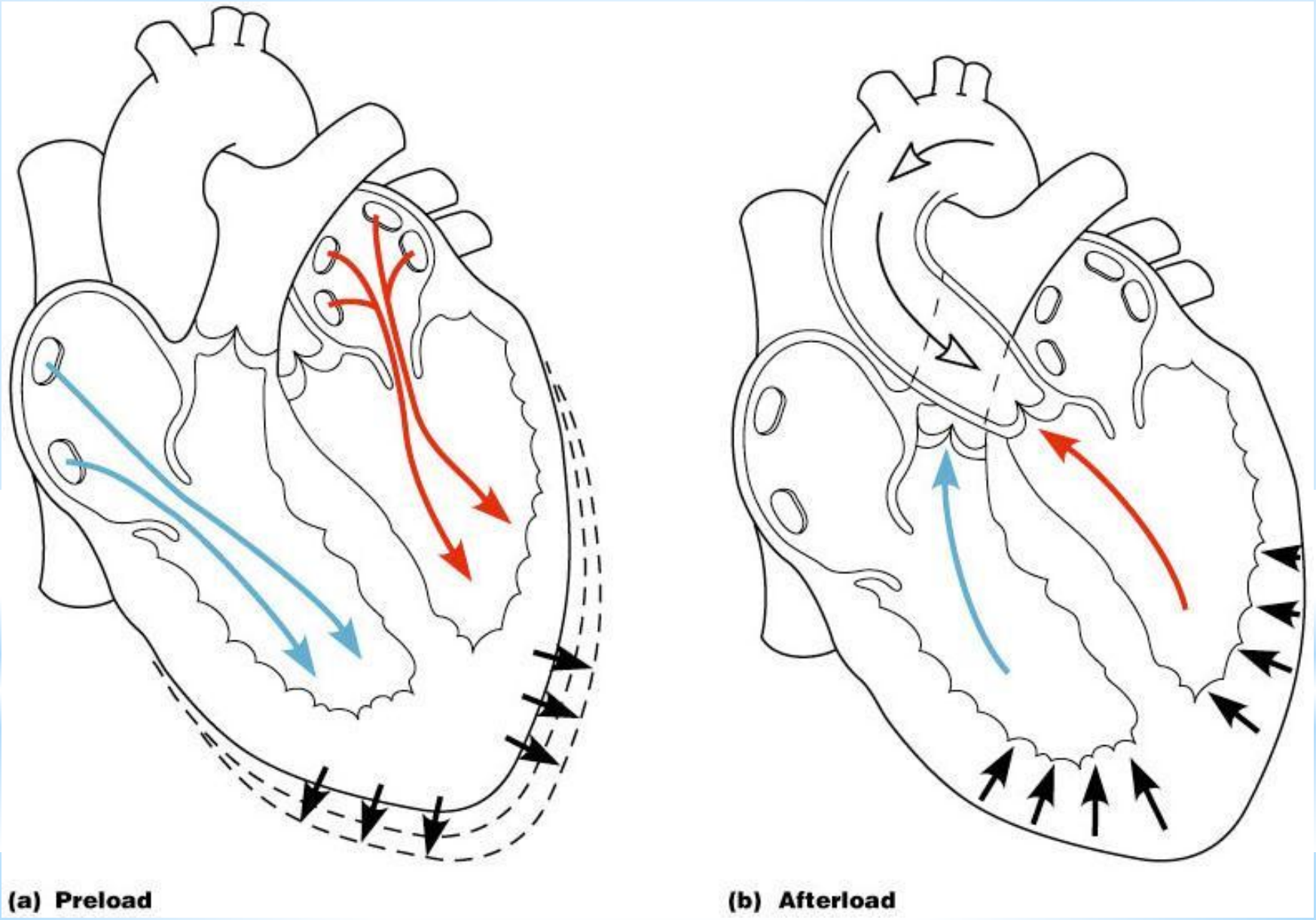


Figure 18.21

*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)

References

* Internet resources
* Textbook of Marya

Human phisiology