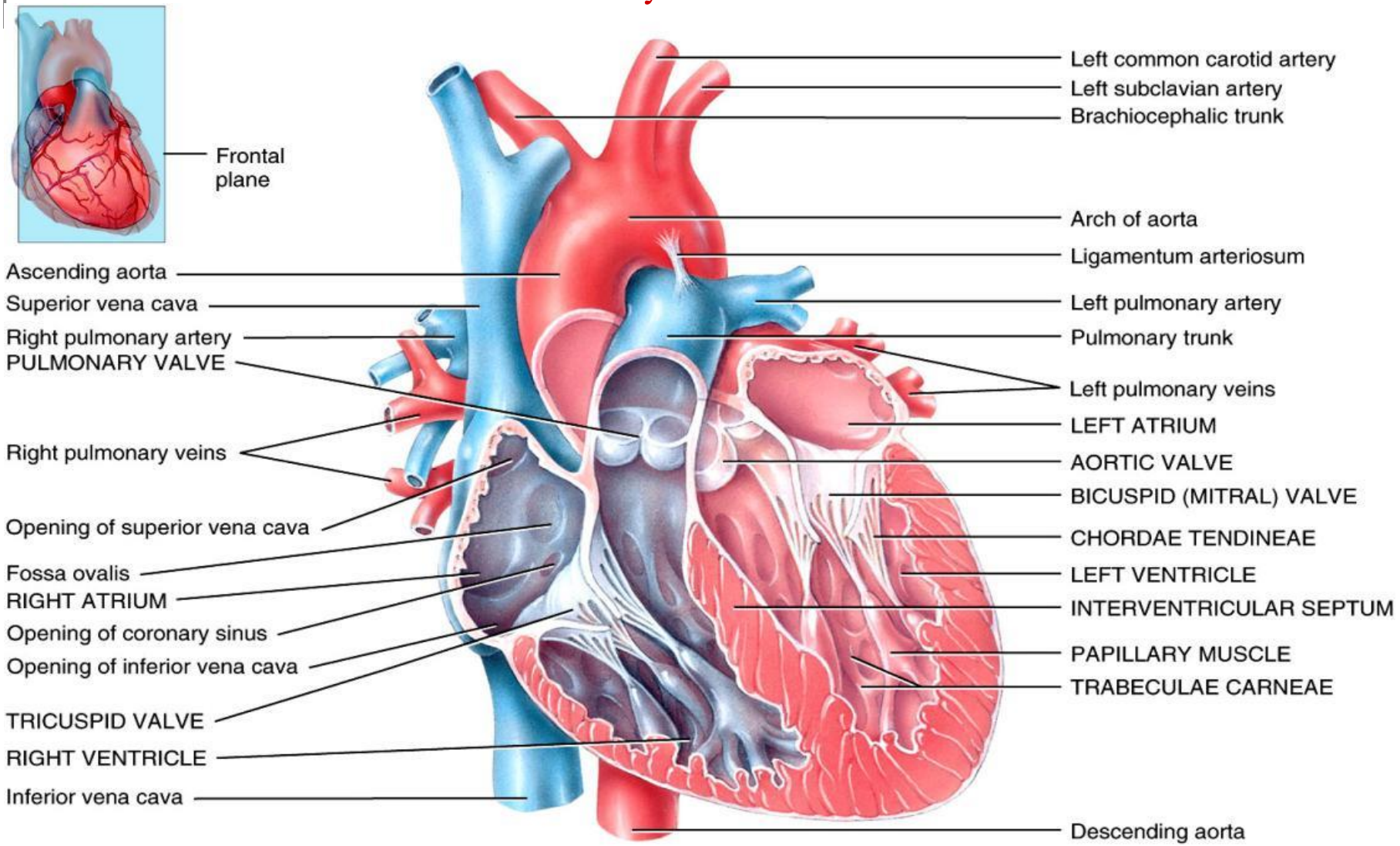
The Heart

Internal Anatomy of the Heart



(a) Anterior view of frontal section showing internal anatomy

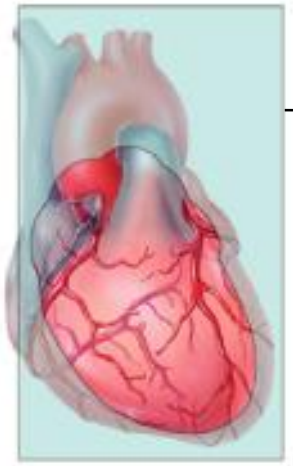
Figure 20.04a Tortora - PAP 12/e

Auto Rhythmic Fibers

- Specialized cardiac muscle fibers
- Self-excitable
- Repeatedly generate action potentials that trigger heart contractions
- 2 important functions
  - ✓ Act as pacemaker
  - ✓ Form conduction system

Conduction system

1. Begins in sinoatrial (SA) node in right atrial wall
   - Propagates through atria via gap junctions
   - Atria contract
2. Reaches atrioventricular (AV) node in inter atrial septum
3. Enters atrioventricular (AV) bundle (Bundle of His)
   - Only site where action potentials can conduct from atria to ventricles due to fibrous skeleton
4. Enters right and left bundle branches which extends through inter ventricular septum toward apex
5. Finally, large diameter Purkinje fibers conduct action potential to remainder of ventricular myocardium
   - Ventricles contract



Frontal plane

Right atrium

1 SINOATRIAL (SA) NODE

2 ATRIOVENTRICULAR (AV) NODE

3 ATRIOVENTRICULAR (AV) BUNDLE (BUNDLE OF HIS)

4 RIGHT AND LEFT BUNDLE BRANCHES

Right ventricle

5 PURKINJE FIBERS

Left atrium

Left ventricle

Anterior view of frontal section

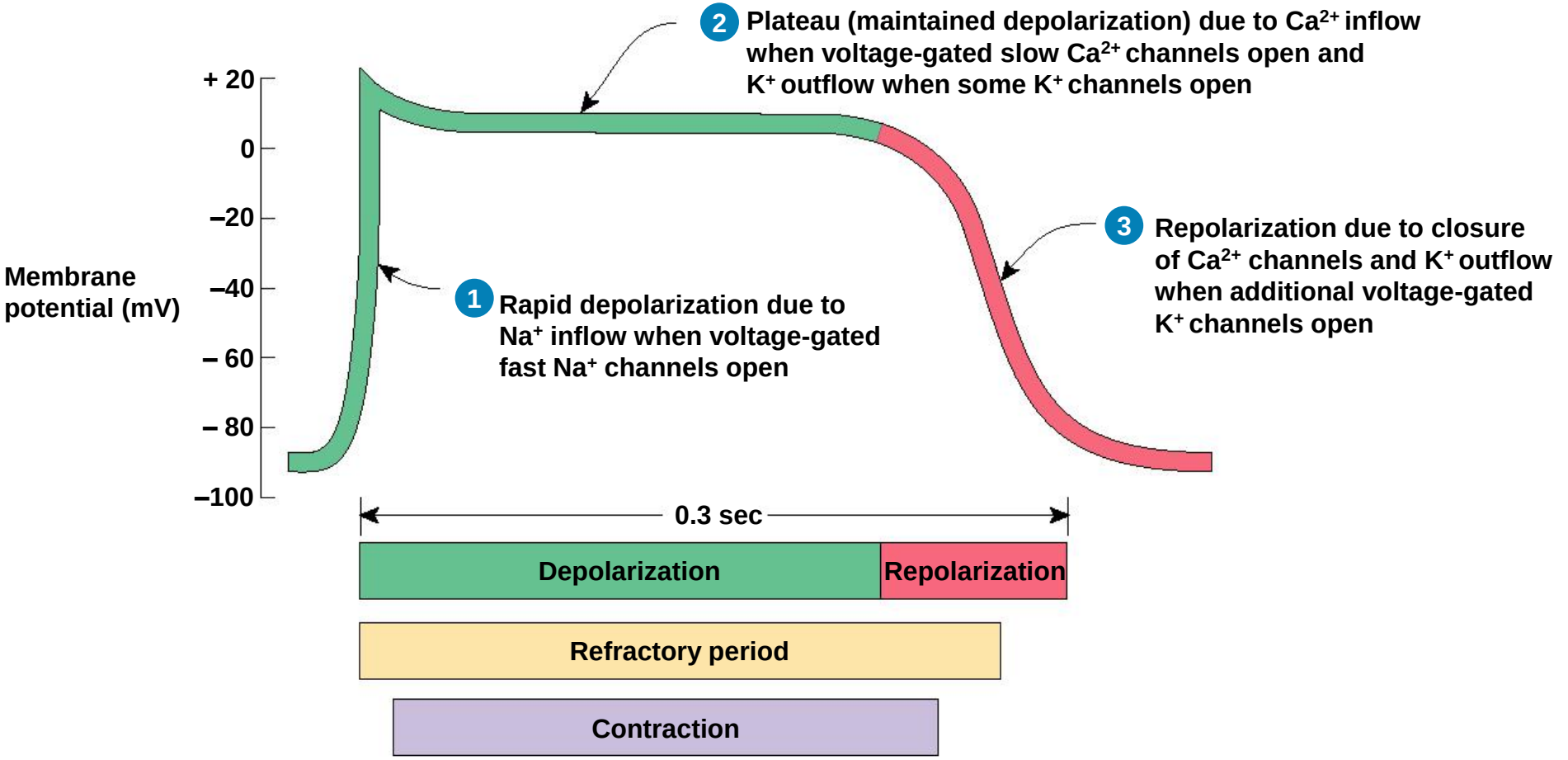
Conduction System

- SA node acts as natural pacemaker
  - Faster than other autorhythmic fibers
- Nerve impulses from autonomic nervous system (ANS) and hormones (epinephrine) modify timing and strength of each heartbeat
  - Do not establish fundamental rhythm

Action Potentials and Contraction

- Action potential initiated by SA node spreads out to excite "working" fibers called contractile fibers

1. Depolarization
2. Plateau
3. Repolarization


2 Plateau (maintained depolarization) due to Ca^{2+} inflow when voltage-gated slow Ca^{2+} channels open and K^+ outflow when some K^+ channels open
3 Repolarization due to closure of Ca^{2+} channels and K^+ outflow when additional voltage-gated K^+ channels open
1 Rapid depolarization due to Na^+ inflow when voltage-gated fast Na^+ channels open
Membrane potential (mV)
+ 20
0
–20
–40
– 60
– 80
–100
0.3 sec
Depolarization
Repolarization
Refractory period
Contraction

Action Potentials and Contraction

Depolarization – contractile fibers have stable resting membrane potential: -90mv

- Voltage-gated fast Na^+ channels open – Na^+ flows in
- Are referred to as "fast" because they open very rapidly in response to a threshold-level depolarization
- Inflow of Na down the electrochemical gradient produces a rapid depolarization
- Then deactivate and Na^+ inflow decreases

Action Potentials and Contraction

Plateau – period of maintained depolarization

- Due in part to opening of voltage gated slow Ca^{2+} channels – Ca^{2+} moves from interstitial fluid into cytosol
- Inflow of Ca^{2+} causes even more Ca^{2+} to pour out of the sarcoplasmic reticulum into the cytosol through additional Ca^{2+} channels in the sarcoplasmic reticulum membrane
- Ultimately triggers contraction
- Depolarization sustained due to voltage gated K^{+} channels balancing Ca^{2+} inflow with K^{+} outflow
- The plateau phase lasts for about 0.25 sec

Action Potentials and Contraction

Repolarization – recovery of resting membrane potential

- Resembles that in other excitable cells
- Additional voltage-gated K^+ channels open
- Outflow K^+ of restores negative resting membrane potential
- Closing Calcium channels - contributes to Repolarization

Action Potentials and Contraction

- Refractory period – time interval during which second contraction cannot be triggered
  - Lasts longer than contraction itself
  - another contraction cannot begin until relaxation is well underway
  - Tetanus (maintained contraction) cannot occur in cardiac muscle as it can in skeletal muscle
  - If heart muscle could undergo tetanus, blood flow would cease

ATP Production in Cardiac Muscle

- It relies almost exclusively on aerobic cellular respiration in its numerous mitochondria
- The needed oxygen diffuses from blood in the coronary circulation and is released from myoglobin inside cardiac muscle fibers
- Cardiac muscle fibers use several fuels to power mitochondrial ATP production
- The heart's ATP comes mainly from oxidation of fatty acids (60%) and glucose (35%),with smaller contributions from lactic acid, amino acids, and ketone bodies.

Cardiac Cycle

- All events associated with one heartbeat
- Systole and diastole of atria and ventricles
- In each cycle, atria and ventricles alternately contract and relax
  - During atrial systole, ventricles are relaxed
  - During ventricle systole, atria are relaxed
- Forces blood from higher pressure to lower pressure
- When heart rate is 75 beats/min, a cardiac cycle lasts 0.8 sec
- During relaxation period, both atria and ventricles are relaxed
  - The faster the heart beats, the shorter the relaxation period
  - Systole and diastole lengths shorten slightly

Atrial Systole

- During atrial systole, which lasts about 0.1 sec, the atria are contracting. At the same time, the ventricles are relaxed.
- Atrial systole contributes a final 25 mL of blood to the volume already in each ventricle (about 105 mL).
- The end of atrial systole is also the end of ventricular diastole (relaxation).
- Thus, each ventricle contains about 130 mL at the end of its relaxation period (diastole).
- This blood volume is called the end-diastolic volume (EDV).

Ventricular Systole

- During ventricular systole, which lasts about 0.3 sec, the ventricles are contracting.
- At the same time, the atria are relaxed in atrial diastole.
- As ventricular systole begins, pressure rises inside the ventricles and pushes blood up against the atrioventricular (AV) valves, forcing them shut.
- For about 0.05 seconds, both the SL (semilunar) and AV valves are closed.
- This is the period of isovolumetric contraction

Isovolumetric contraction

- During this interval, cardiac muscle fibers are contracting and exerting force but are not yet shortening.
- Thus, the muscle contraction is isometric (same length).
- Because all four valves are closed, ventricular volume remains the same (isovolumic).

Ventricular Systole

- Continued contraction of the ventricles causes pressure inside the chambers to rise sharply.
- When left ventricular pressure surpasses aortic pressure at about 80 millimeters of mercury (mmHg) and right ventricular pressure rises above the pressure in the pulmonary trunk (about 20 mmHg), both Semi Lunar valves open.

Ventricular Systole

- At this point, ejection of blood from the heart begins.
- The period when the SL valves are open is ventricular ejection and lasts for about 0.25 sec.
- The pressure in the left ventricle continues to rise to about 120 mmHg, whereas the pressure in the right ventricle climbs to about 25–30 mmHg.

Ventricular Systole

- The left ventricle ejects about 70 mL of blood into the aorta and the right ventricle ejects the same volume of blood into the pulmonary trunk.
- The volume remaining in each ventricle at the end of systole, about 60 mL, is the end-systolic volume (ESV).
- Stroke volume, the volume ejected per beat from each ventricle, equals end-diastolic volume minus end systolic volume
- SV= EDV-ESV, 130-60= 70ml

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Relaxation Period

- During the relaxation period, which lasts about 0.4 sec, the atria and the ventricles are both relaxed.
- Ventricular repolarization causes ventricular diastole.
- As the ventricles relax, pressure within the chambers falls, and blood in the aorta and pulmonary trunk begins to flow backward toward the regions of lower pressure in the ventricles

Relaxation Period

- The aortic valve closes at a pressure of about 100 mmHg.
- After the SL valves close, there is a brief interval when ventricular blood volume does not change because all four valves are closed.
- This is the period of isovolumetric relaxation.

Cardiac Output

- CO = volume of blood ejected from left (or right) ventricle into aorta (or pulmonary trunk) each minute
- CO = stroke volume (SV) x heart rate (HR)
- In typical:
  - 5.25L/min = 70mL/beat x 75 beats/min
- Entire blood volume flows through pulmonary and systemic circuits each minute

Regulation of stroke volume

- 3 factors ensure left and right ventricles pump equal volumes of blood

1. Preload
2. Contractility
3. Afterload

Preload

- Degree of stretch on the heart before it contracts
- Greater preload increases the force of contraction
- Frank-Starling law of the heart – the more the heart fills with blood during diastole, the greater the force of contraction during systole
  - Preload proportional to end-diastolic volume (EDV)
- 2 factors determine EDV
  1. Duration of ventricular diastole
  2. Venous return – volume of blood returning to right ventricle

Contractility

- Strength of contraction at any given preload
- Positive inotropic agents increase contractility
  - Often promote Ca^{2+} inflow during cardiac action potential
  - Increases stroke volume
  - Epinephrine, Norepinephrine, Digitalis
- Negative inotropic agents decrease contractility
  - Anoxia, acidosis, some anesthetics, and increased K^{+} in interstitial fluid

Afterload

- The pressure that must be overcome before a semilunar valve can open is termed the afterload.
- Increase in afterload causes stroke volume to decrease
  - Blood remains in ventricle at the end of systole
- Hypertension and atherosclerosis increase afterload

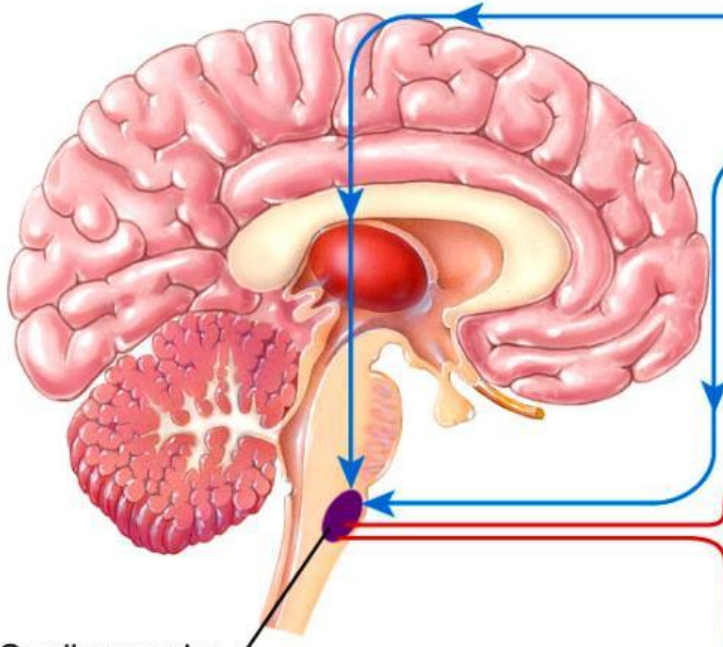
Regulation of Heart Beat

- Cardiac output depends on heart rate and stroke volume
- Adjustments in heart rate is important in short-term control of cardiac output and blood pressure
- Autonomic nervous system and epinephrine/ norepinephrine most important

Autonomic regulation

- Originates in cardiovascular center of medulla oblongata
- Increases or decreases frequency of nerve impulses in both sympathetic and parasympathetic branches of ANS
- Noreprinephrine has 2 separate effects
  - In SA and AV node speeds rate of spontaneous depolarization
  - In contractile fibers enhances Ca^{2+} entry increasing contractility
- Parasympathetic nerves release acetylcholine which decreases heart rate by slowing rate of spontaneous depolarization

Nervous System Control of the Heart



Cardiovascular (CV) center

INPUT TO CARDIOVASCULAR CENTER

From higher brain centers: cerebral cortex, limbic system, and hypothalamus

From sensory receptors:
Proprioceptors–monitor movements
Chemoreceptors–monitor blood chemistry
Baroreceptors–monitor blood pressure

Cardiac accelerator nerves (sympathetic)

Vagus nerves (cranial nerve X, parasympathetic)

OUTPUT TO HEART

Increased rate of spontaneous depolarization in SA node (and AV node) increases heart rate

Increased contractility of atria and ventricles increases stroke volume

Decreased rate of spontaneous depolarization in SA node (and AV node) decreases heart rate

Figure 20.16 Tortora - PAP 12/e

Chemical regulation of heart rate

- Hormones
  - Epinephrine and norepinephrine increase heart rate and contractility
  - Thyroid hormones also increase heart rate and contractility
- Cations
  - Ionic imbalance can compromise pumping effectiveness
  - Relative concentration of K^+, Ca^{2+} and Na^+ important