Heart Physiology 1

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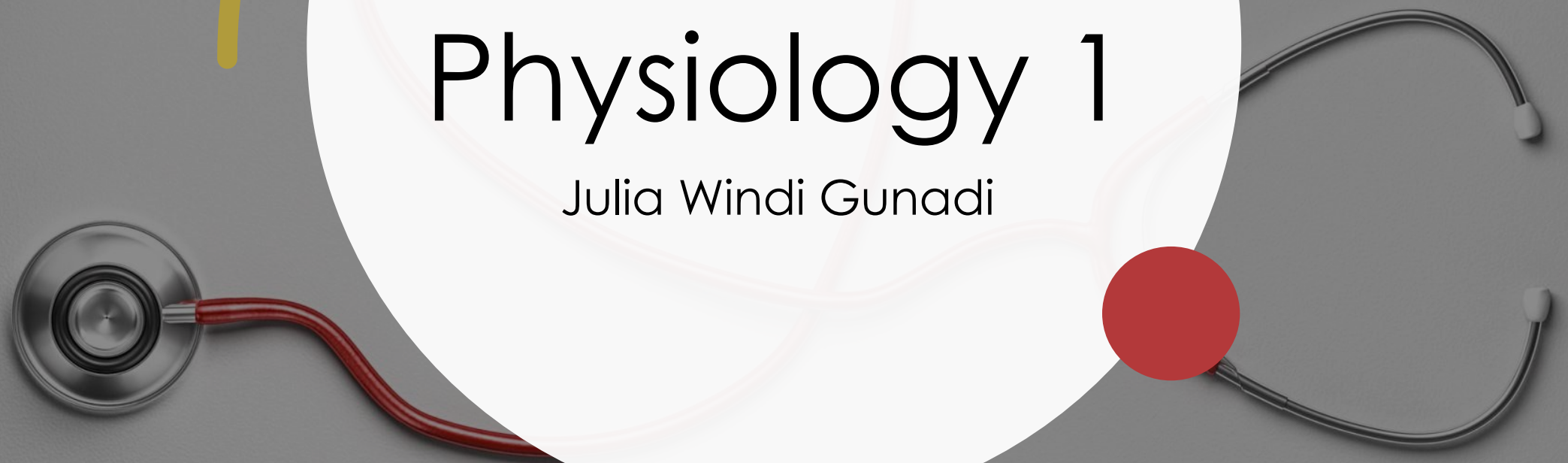
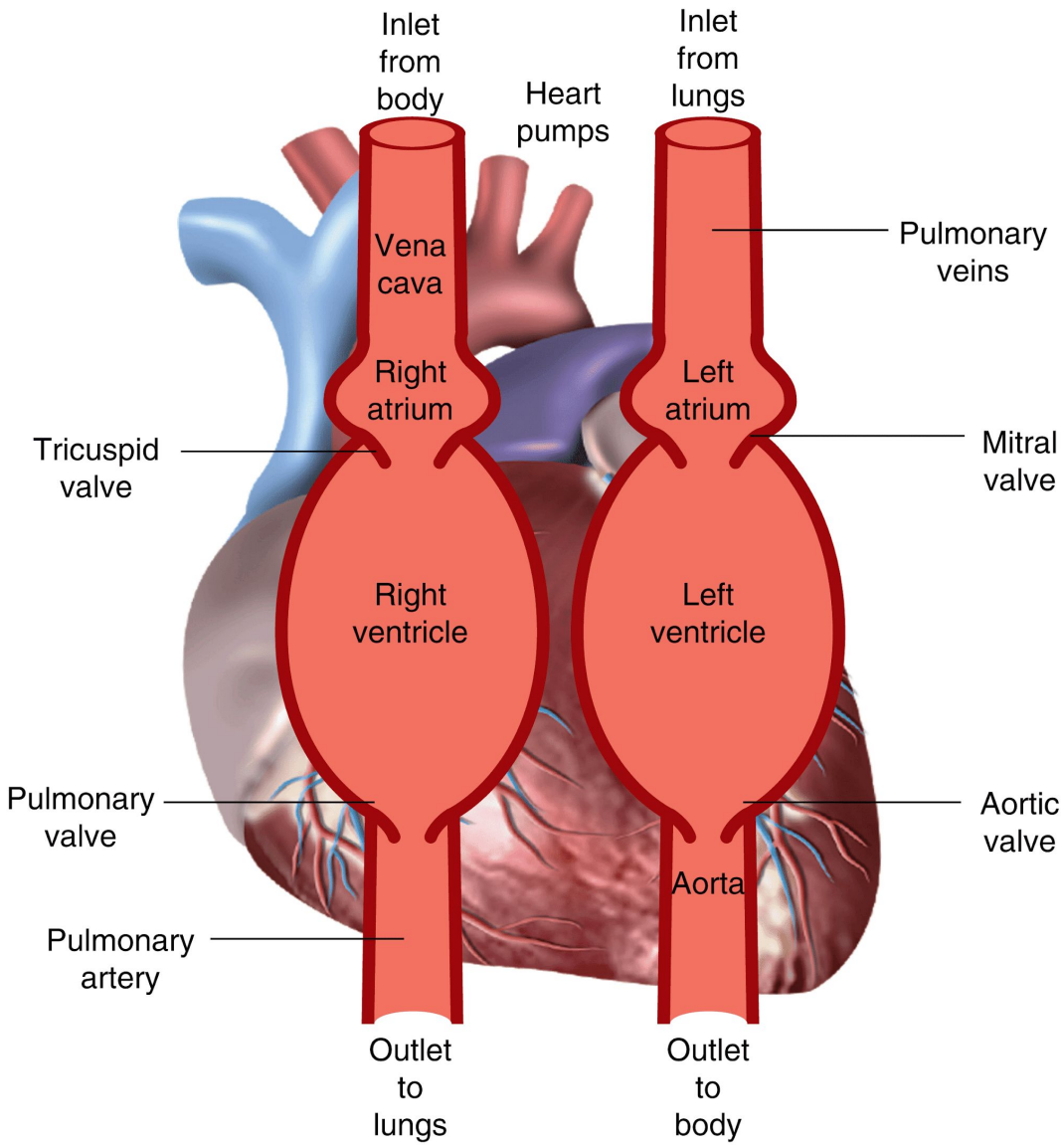


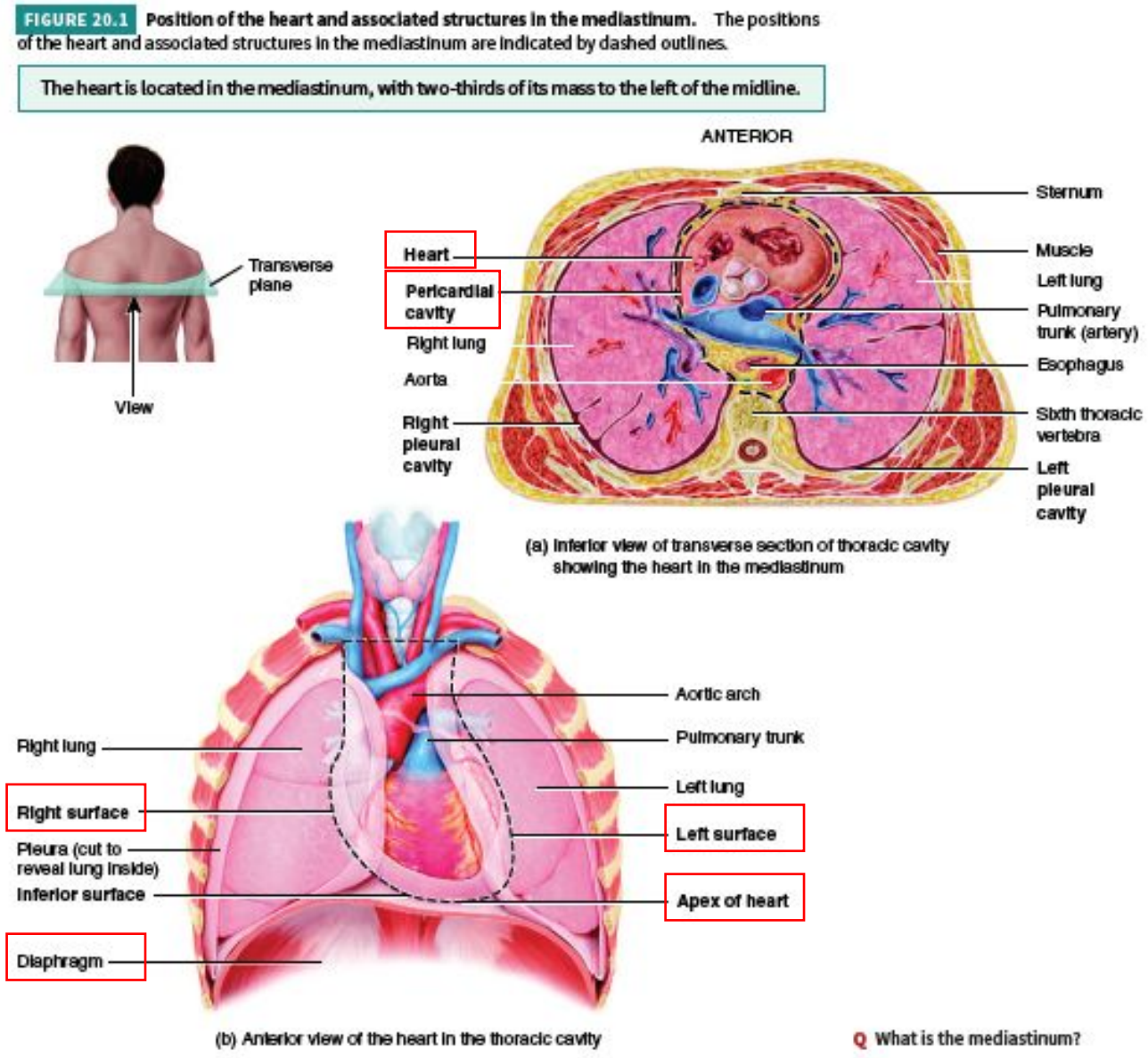
Highlights

- Heart Anatomy
- Pulmonal and Cardiac Circulation
- Cardiac Muscle Excitation Contraction Coupling
- Conduction System
- Regulation of Heart Rate
- Effect of HR on Contractility
- Effect of Cardiac Glycosides on Contractility
- Cardiac Cycle



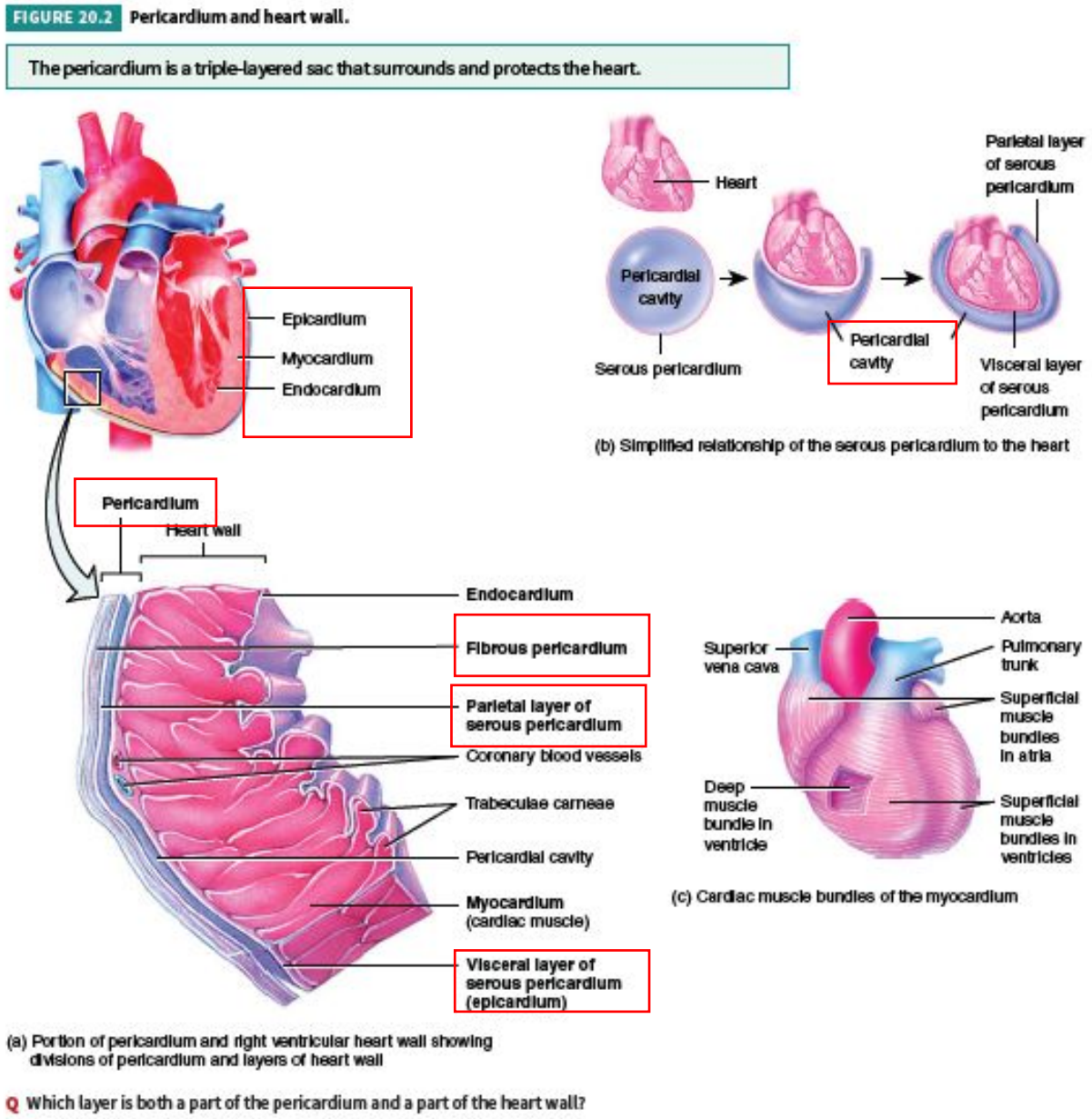
Heart as a pump

- Heart beats 100.000x/day, 35 million beats/year, 2.5 billion in average lifetime
- The left side heart pumps blood 100.000 (60.000 ml), equivalent to travelling around the earth's equator about three times
- Heart pumps 14.000 L of blood/day, 5 million/year
- You don't spend all your time sleeping, so the heart pumps more vigorously when you active



Heart Anatomy

- Sebesar kepalan tangan manusia
- Panjang 12 cm, lebar 9 cm, tebal 6 cm, berat 250/300 gram
- Berada dalam diafragma, dekat midline cavum thoraks
- 2/3 massa jantung berada di bagian kiri midline
- Apex inferior, anteriorly to the left, Basis opposite to apex and posterior, formed mostly by left atrium



Pericardium and Heart wall

- Perikardium tdd 3 lapisan:
- Fibrous jaringan penyokong yg kuat, menempel pd diafragma (fusi dengan tendon) proteksi, cegah overstretching, membuat jantung tertanam dlm mediastinum
- Parietal layer of serous membatasi bag dalam fibrous
- Visceral/Inner layer of serous = epicardium
- Di antaranya (cavum pericardial) terdapat cairan pericardium mengurangi friksi antara lapisan serous saat jantung bergerak
- Heart wall tdd epicardium, myocardium, dan endocardium

A prodrome of fever, malaise, and myalgia may herald the chief complaint of chest pain.

History

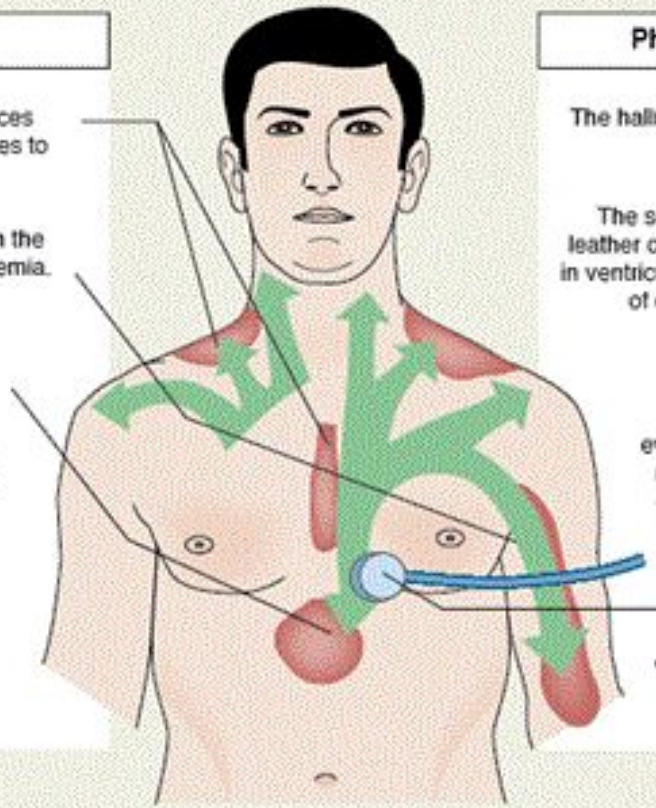
Typically, acute pericarditis produces sharp retrosternal *pain* that radiates to the trapezius ridge.

Pericardial pain may radiate down the left arm and suggest cardiac ischemia.

Pain may be localized to the epigastrium and mimic an acute abdomen.

Pericardial pain is aggravated by lying down and relieved by sitting up, and often worsens with inspiration.

Patients with acute pericarditis may complain of dyspnea and (less commonly) cough, dysphagia, and/or hiccups.



Physical examination

The hallmark of acute pericarditis is the pericardial *friction rub*.

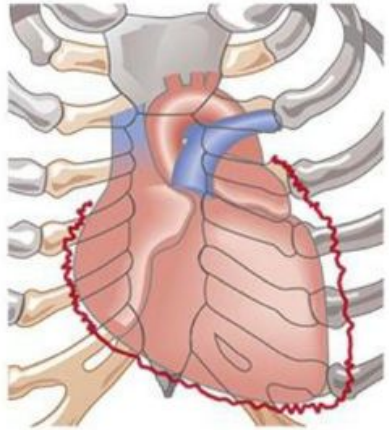
The sound resembles "the squeak of leather of a new saddle under the rider" in ventricular systole, atrial systole (70% of cases), and ventricular diastole (<70% of cases).

Pericardial friction rubs are evanescent, usually change with respiration and with changes in position, and frequently coexist with pleural rubs.

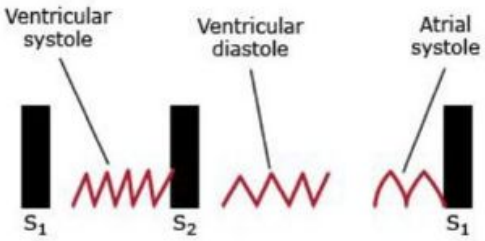
The stethoscope diaphragm should be placed firmly on the chest wall, usually between the lower left sternal border and the cardiac apex.

Clinical note: The quality, severity, and location of pain vary greatly. Repeat examinations often prove necessary to detect friction rubs, which may be confused with cardiac murmurs, with sounds due to pneumomediastinum, and, most commonly, with artifacts produced by skin rubbing against a loosely placed stethoscope head.

Pericardial Friction Rub



- Superficial scratchy/squeaky quality
- Generated by friction between layers of the pericardium
- Loudest on left sternal border



INFECTIVE ENDOCARDITIS (IE) – AETIOLOGY

Native valve
- Viridans group streptococci
- Enterococci
- Other streptococci
- *Staphylococcus aureus*
- Coagulase-negative staphylococci
- Fastidious Gram-negatives

Culture-negative endocarditis
- NB serological diagnosis
- Previous antibiotic therapy
- *Chlamydophila pneumoniae/ Chlamydia psittaci*
- *Coxiella burnetti* (Q fever)
- *Mycoplasma*

Prosthetic valve – Early
- Coagulase-negative staphylococci
- *Staphylococcus aureus*
- Viridans group streptococci
- Enterococci and other streptococci
- Fungi

Late – as for native valve

Right sided
- Nutritionally deficient strains
- *Staphylococcus aureus*
- Mixed infections
- Fungi

IE – PATHOGENESIS

Damage and roughening of endothelium

↓

Fibrin and platelet deposition

↓ Bacteraemia – oropharynx/gut/ urinary tract

Colonization of deposit

↓

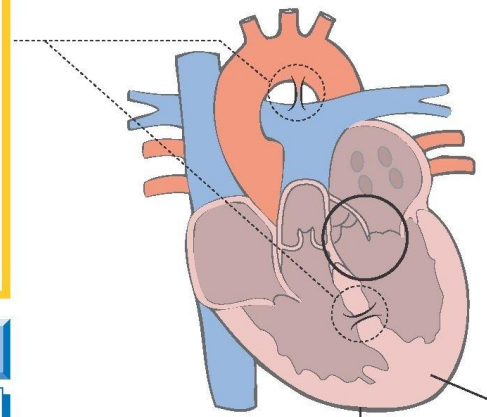
Bacterial multiplication, further fibrin and platelet deposition, immune activation

↓

Systemic signs of infection, development of vegetation, toxic, embolic and immune complex phenomena

IE – PREDISPOSING FACTORS

- Atherosclerosis/ischaemic changes
- Degenerative changes
- Congenital abnormalities, e.g. VSD, coarctation
- Rheumatic fever
- Prosthetic material, e.g. valves, pacing wires, patches/ grafts, central venous lines
- i.v. drug abusers – right sided



PERICARDITIS – AETIOLOGY

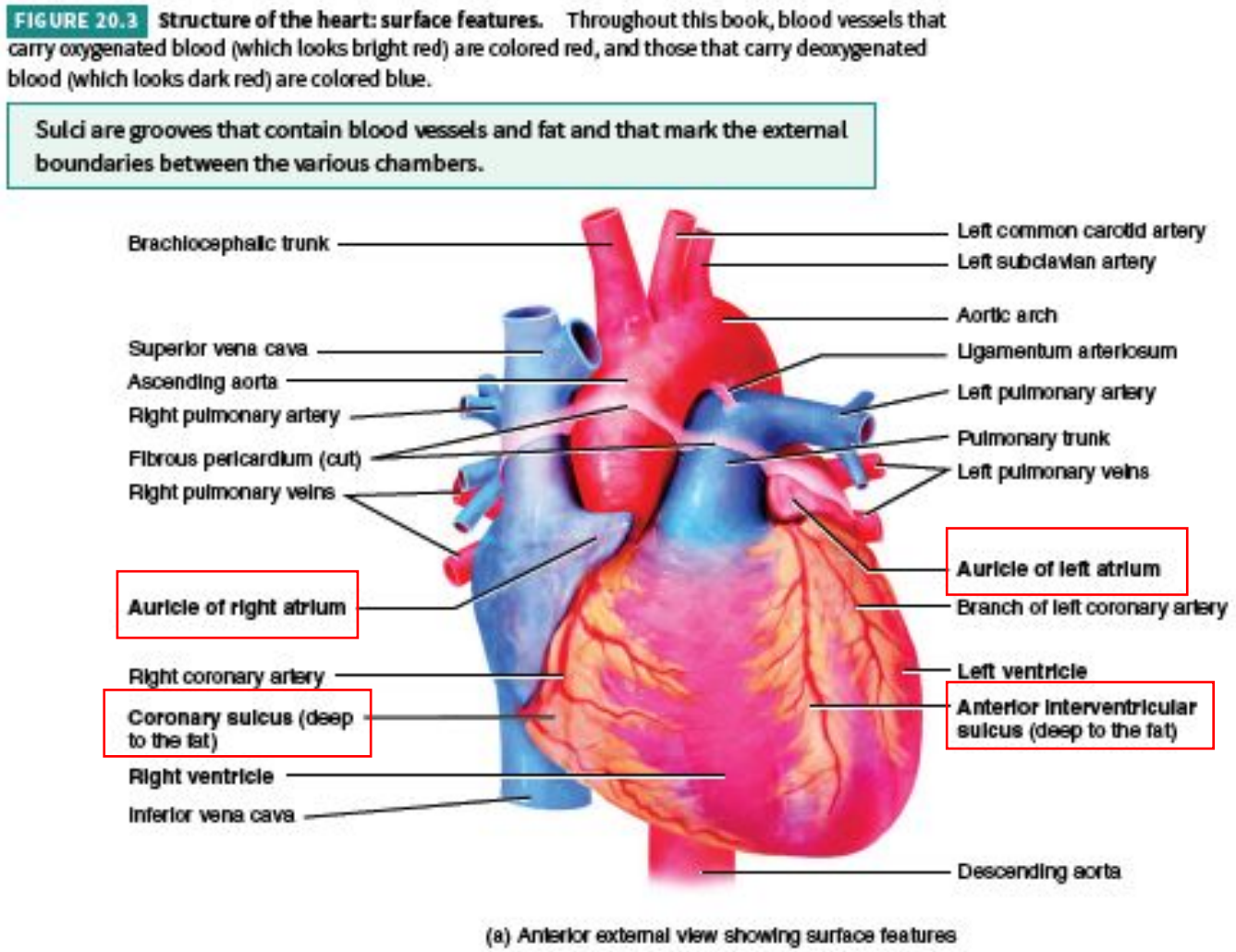
- Pneumonia, e.g. Pneumococcus, S. aureus, M. tuberculosis
- Enterovirus
- Influenza
- Mycoplasma

MYOCARDITIS – AETIOLOGY

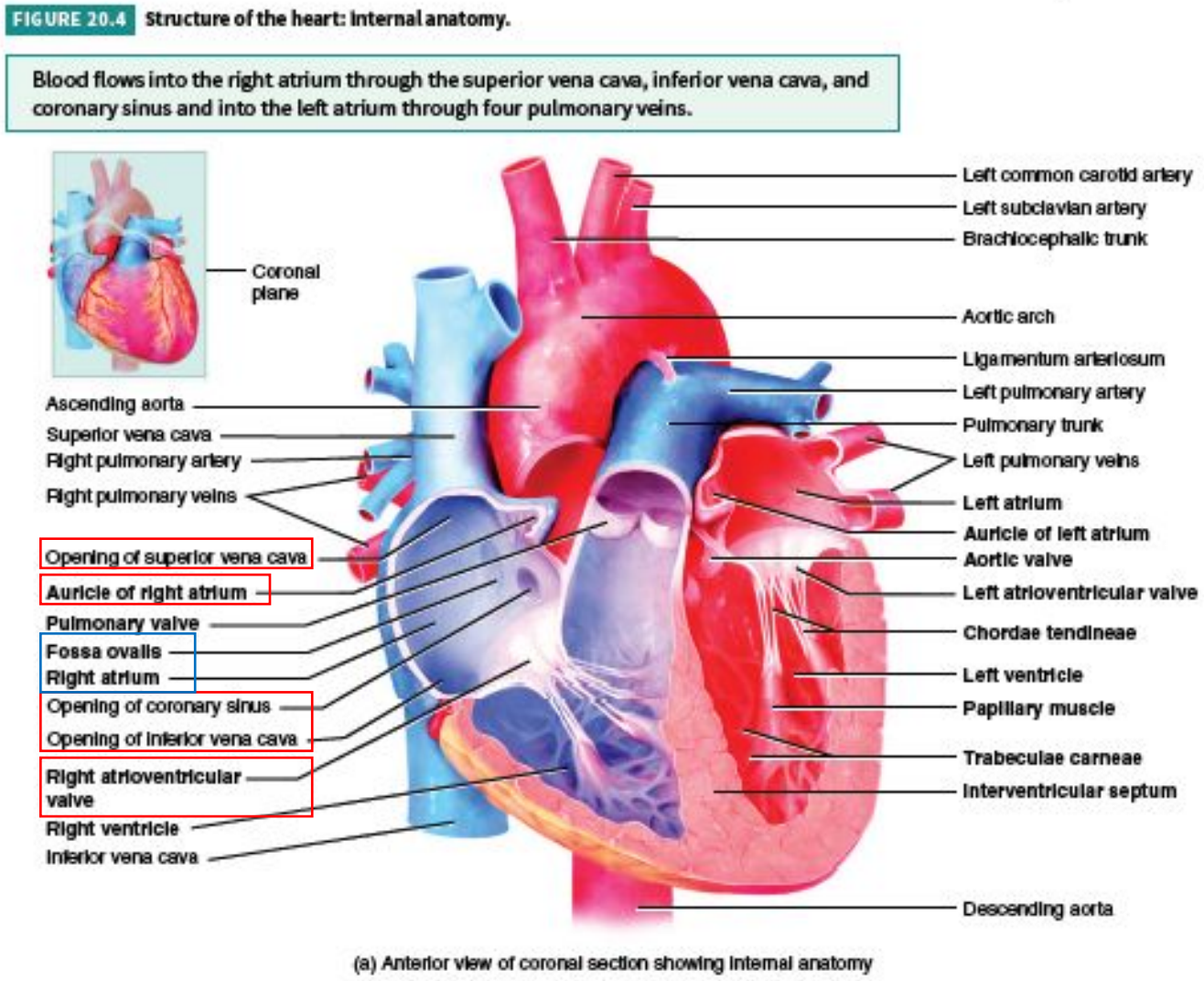
- Coxsackievirus
- Echovirus
- Adenovirus
- Rubella
- Mycoplasma
- Toxic – septicaemia – diphtheria

N.B. Immune mediated

Heart Chambers



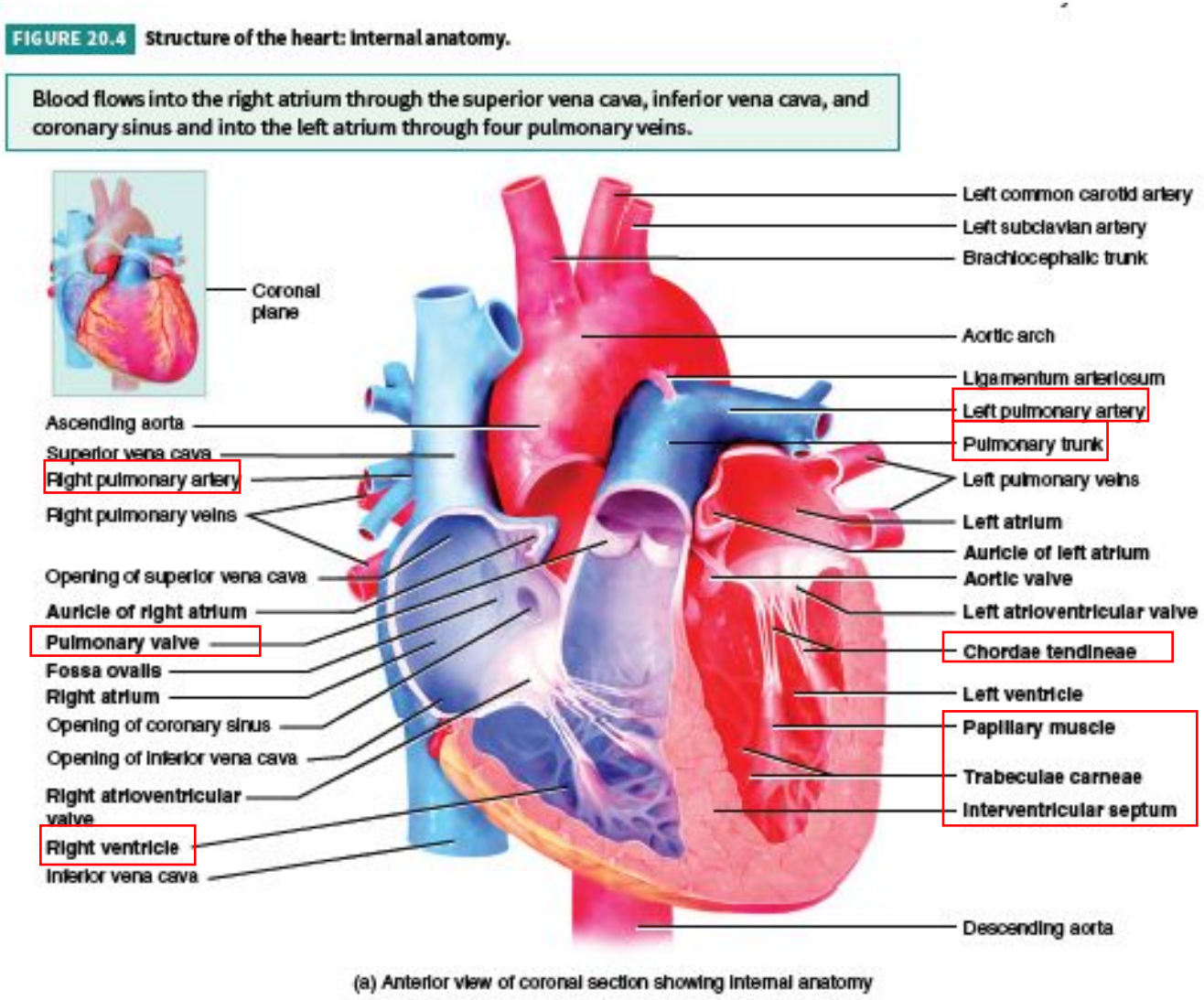
- Tdd 2 atrium dan 2 ventrikel
- Auricula atrium (spt telinga anjing) meningkatkan kapasitas atrium menampung darah
- Sulcus coronary menandai perbatasan antara atrium dan ventrikel
- Sulcus interventricular (anterior dan posterior) menandai perbatasan antar ventrikel



Atrium Kanan

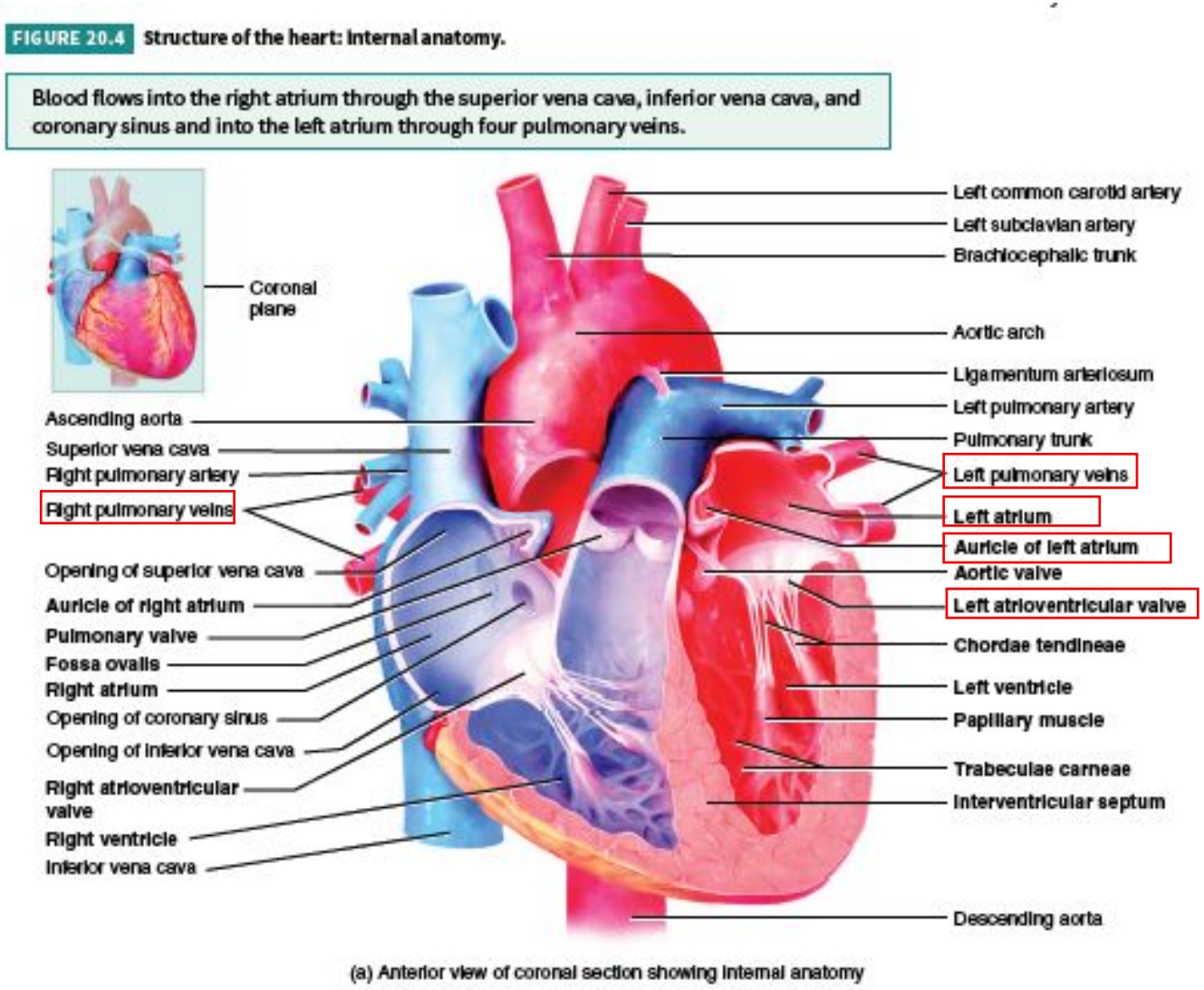
- Membentuk permukaan kanan jantung
- Menerima darah dari v. cava inf, v.cava sup, dan sinus coronarius
- Tebal 2-3 mm
- Dinding posterior smooth, anterior rough krn ada m. pectinatus yg ekstensi sampai ke auricula
- Septum interatrial memisahkan atrium kanan dan kiri, terdapat fossa ovalis (sisa dari foramen ovale yg menutup saat lahir)
- Katup tricuspid (3 leaflets/cusps) antara atrium kiri dan kanan

Ventrikel Kanan



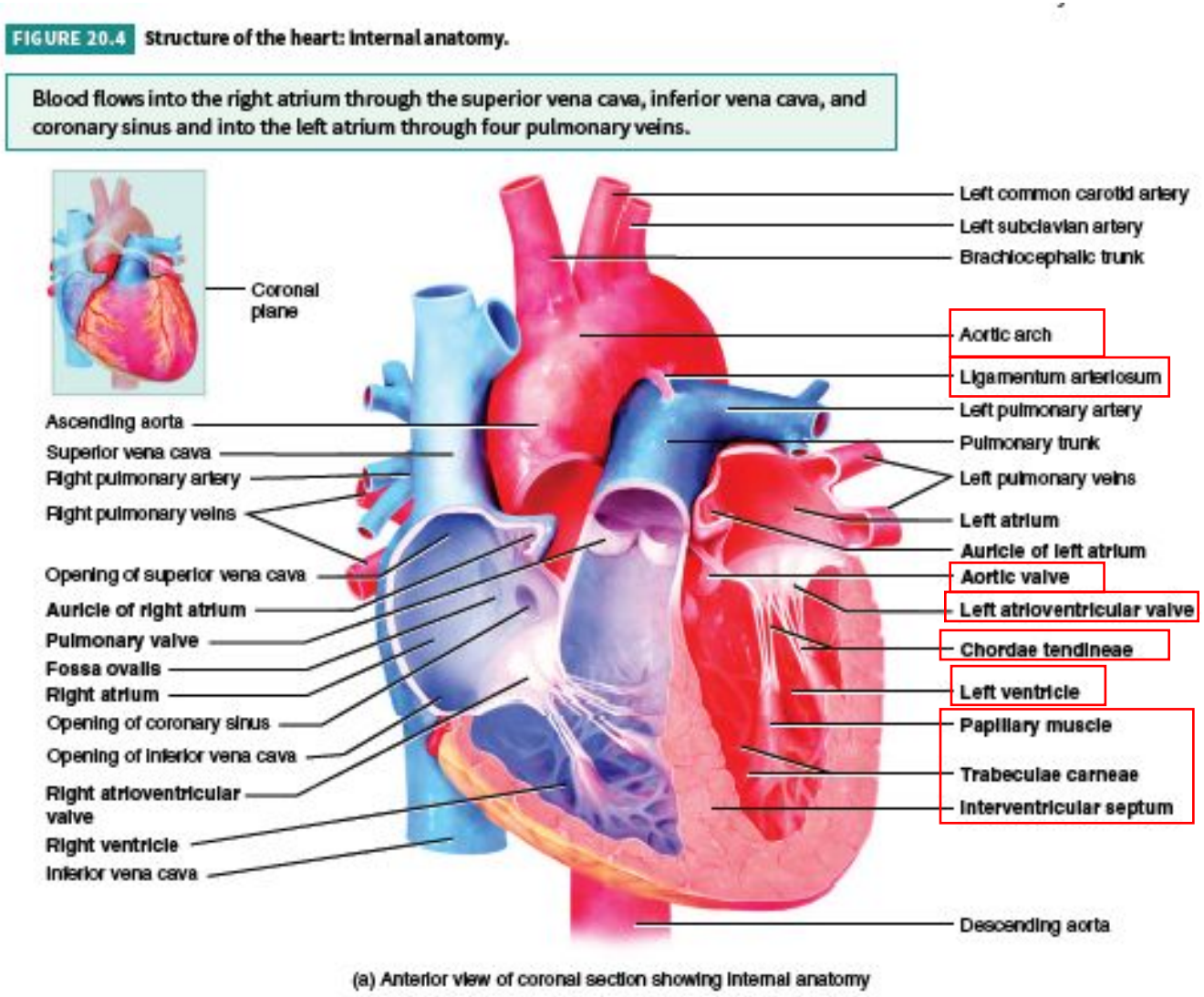
- Membentuk Sebagian besar perm anterior jantung
- Tebal 4-5 mm
- Dinding dalam: Trabeculae carneae (muscle fibers) sistem konduksi
- Katup tricuspid terhubung dengan korda tendinae yg terhubung dengan m. papilaris
- Septum interventricular: memisahkan ventrikel kanan dan kiri
- Darah dari ventrikel kanan melalui katup pulmonalis ke truncus pulmonalis (a. pulmonalis dextra et sinistra)

Atrium kiri



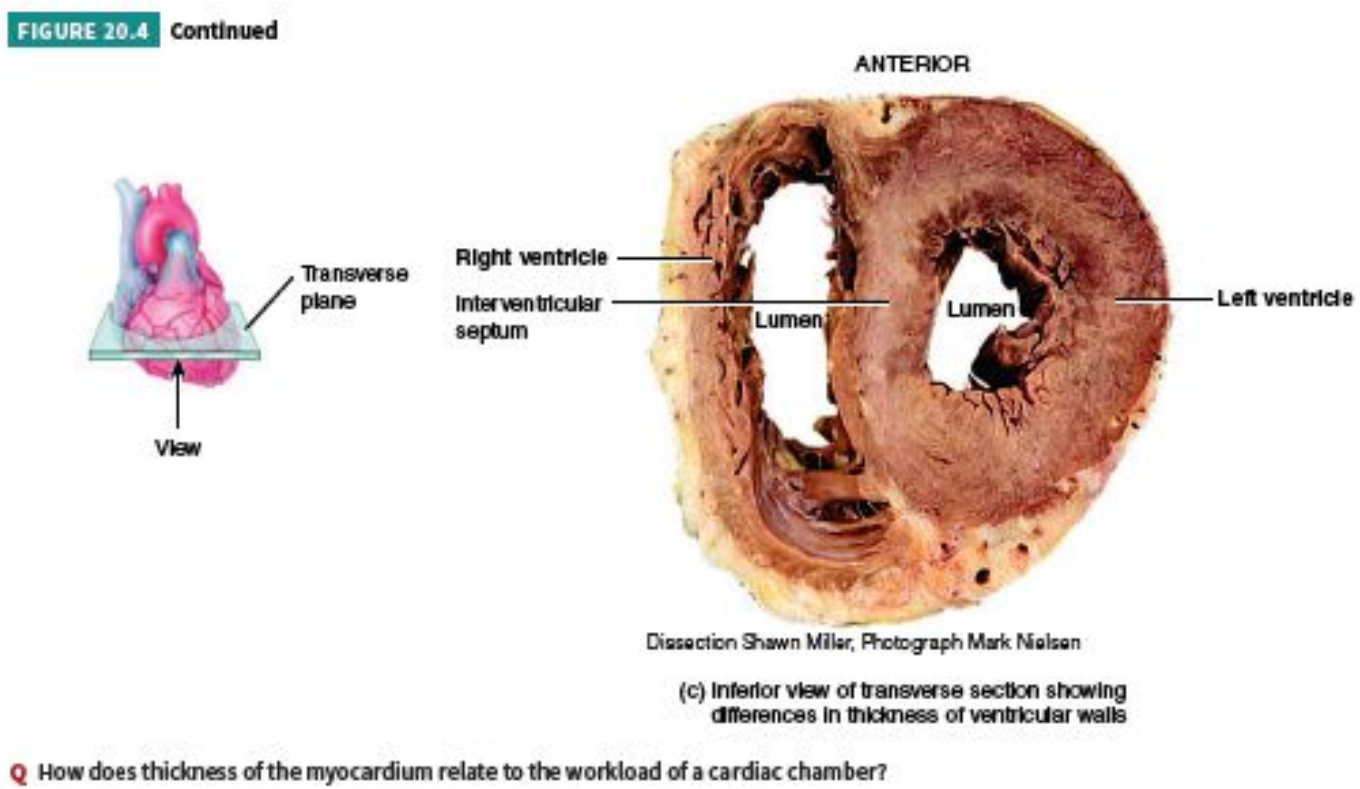
- Membentuk basis jantung
- Tebal = atrium kanan
- Menerima darah dari paru melalui v pulmonalis
- Dinding anterior dan posterior smooth karena m pectinatus terbatas pada aurikula atrium kiri
- Katup antara atrium kiri dan ventrikel kiri bicuspid/mitral
- Mitral topi uskup

Ventrikel Kiri



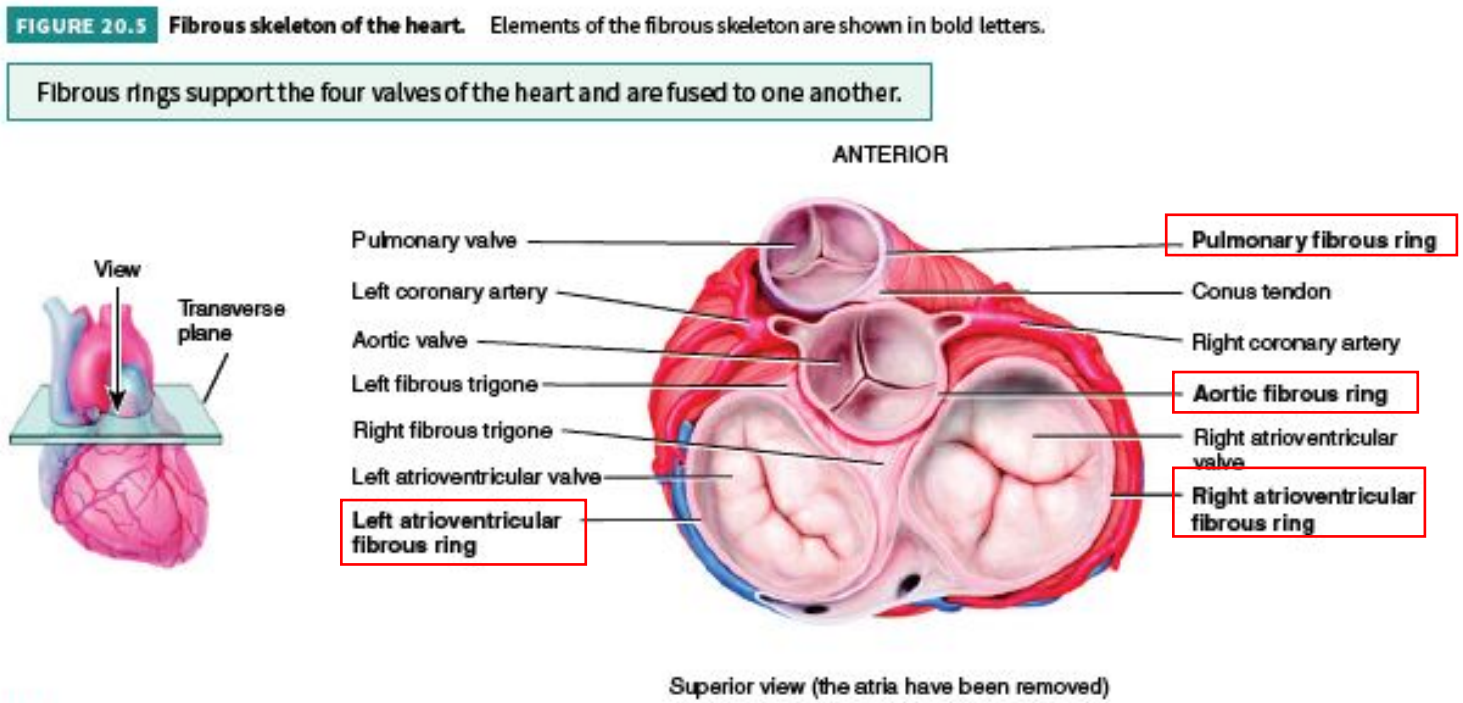
- Membentuk apex jantung
- Tebal 10-15 mm
- Dinding dalam: Trabeculae carneae dan korda tendinae yg terhubung dengan m. papilaris
- Darah dari ventrikel kiri melalui katup aorta ke aorta asenden, dan Sebagian menuju ke a. coronarius. Sisanya menuju ke aorta desenden (aorta thorcica dan aorta abdominalis)
- Ductus arteriosus, shunt darah dari truncus pulmonalis ke aorta, menutup saat lahir ligamentum arteriosum

Perbedaan dinding ventrikel kanan dan kiri



- Dinding ventrikel kanan lebih tipis drpd ventrikel kiri
- Ventrikel kanan smaller workload (shorter distance, smaller resistance of blood flow)
- Ventrikel kiri bigger workload (longer distance, higher resistance of blood flow)
- Lumen vent kanan berbentuk bulan sabit/crescent
- Lumen vent kiri bulat/sirkular

Fibrous skeleton of the heart



- Ada 4 cincin fibrosa (dibentuk oleh dense connective tissues)
- Mengelilingi katup jantung, berfusi satu dengan lainnya, merge dg septum interventricular
- Fungsi:

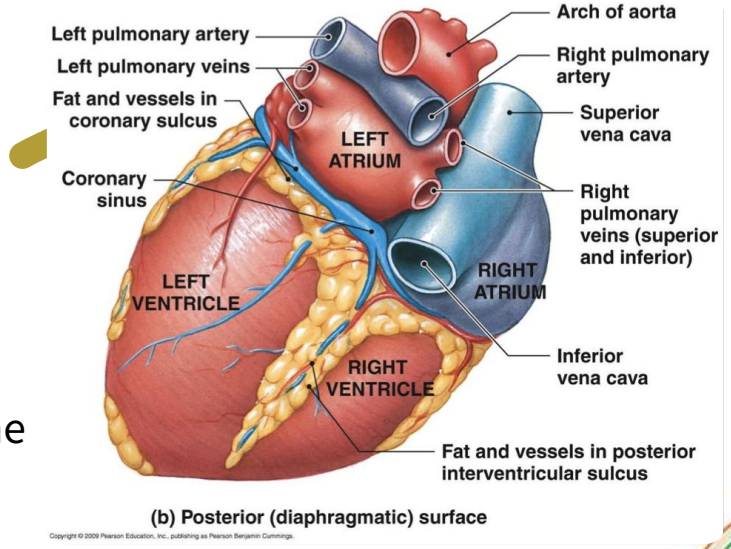
-membentuk dasar struktur katup jantung

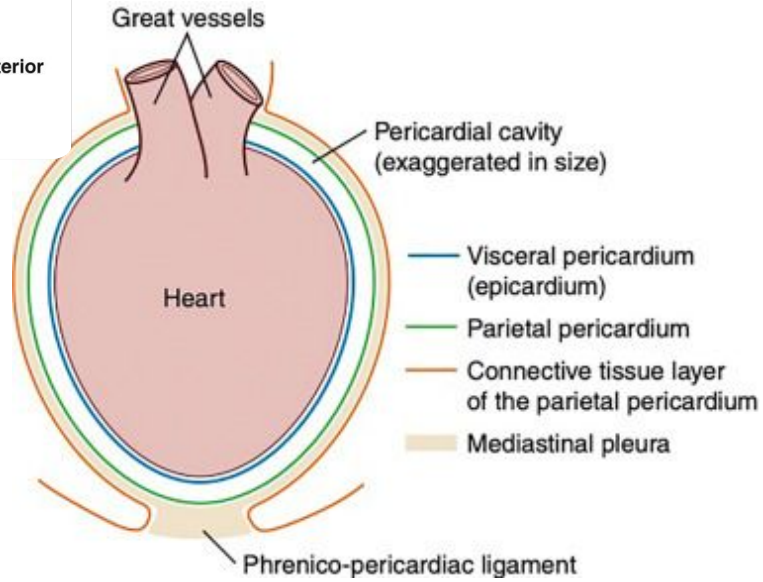
-mencegah overstretching katup saat darah melewatinya
-point of insertion utk bundles serabut otot jantung

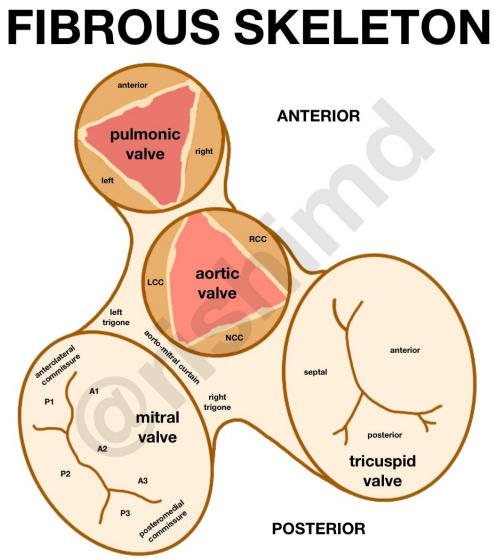
-insulator elektrik antara atrium dan ventrikel

Checkpoint

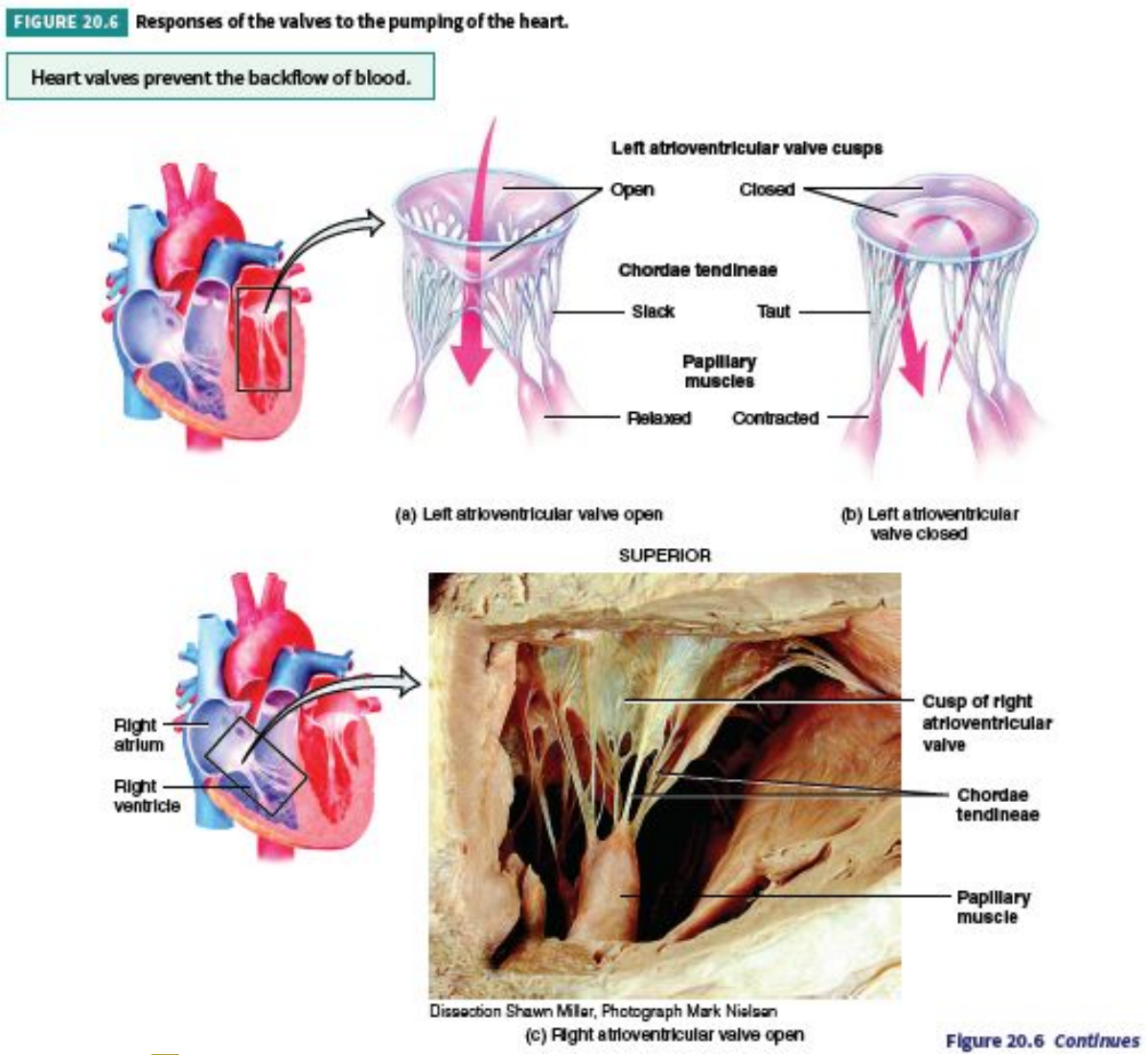
1. Define each of the following external features of the heart: auricle, coronary sulcus, anterior interventricular sulcus, and posterior interventricular sulcus.
2. Describe the structure of the pericardium and the layers of the wall of the heart.
3. What are the characteristic internal features of each chamber of the heart?
4. Which blood vessels deliver blood to the right and left atria?
5. What is the relationship between wall thickness and function among the various chambers of the heart?
6. What type of tissue composes the fibrous skeleton of the heart, and how is it organized?





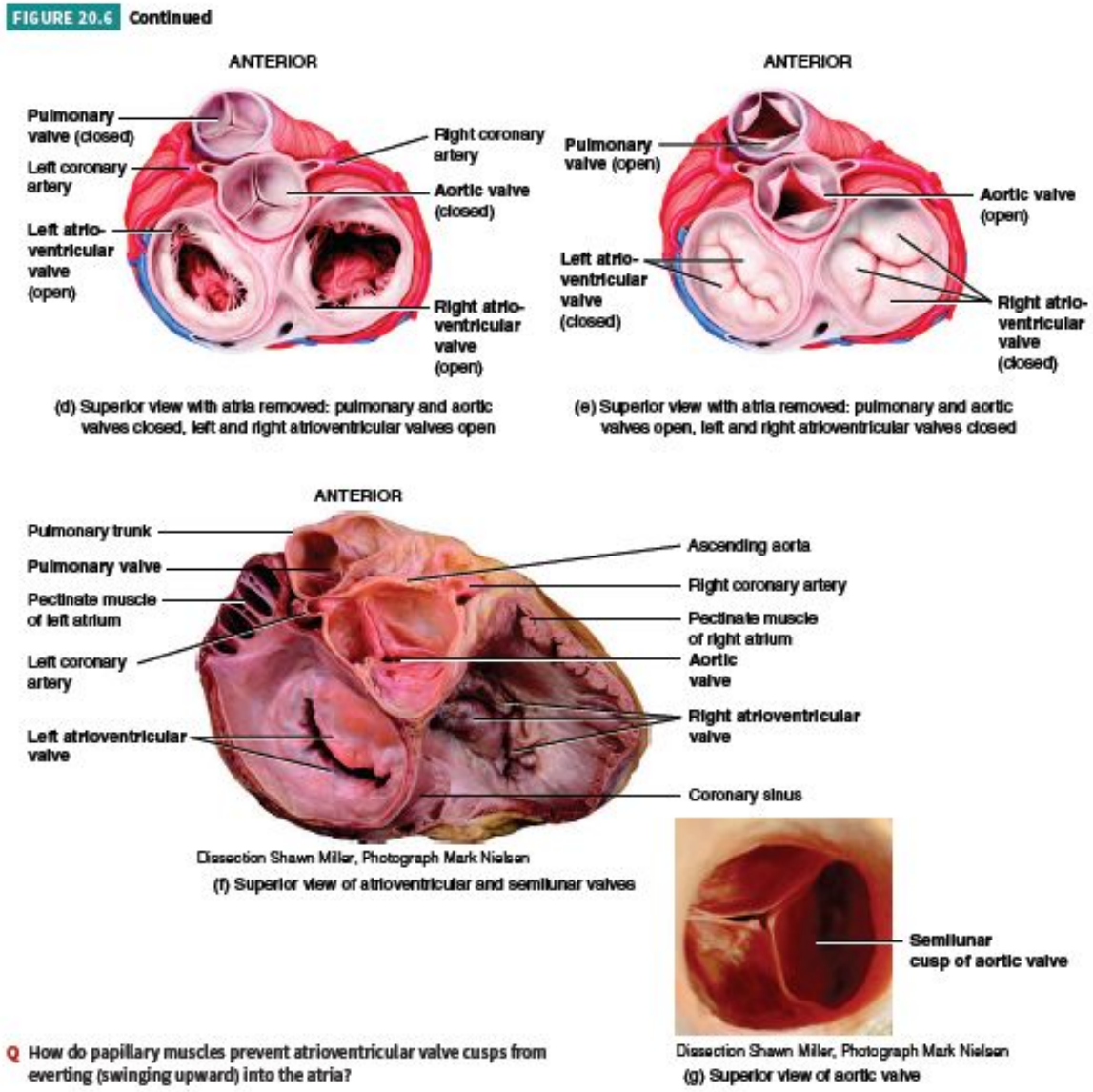


Operation of AV Valve



- Saat katup AV terbuka (relaksasi ventrikel), ujung bundar puncak (cusp) katup terprojeksi ke dalam ventrikel, m papilaris relaksasi, korda tendinae kendur, darah mengalir dari tek atrium yang lebih tinggi ke ventrikel (gravitasi)
- Saat katup AV tertutup (kontraksi ventrikel), ujung bundar katup terdorong ke atas sampai ujungnya bertemu sehingga katup menutup, m papilaris kontraksi, korda tendinae tegang □ mencegah terbukanya katup ke dalam atrium/regurgitasi

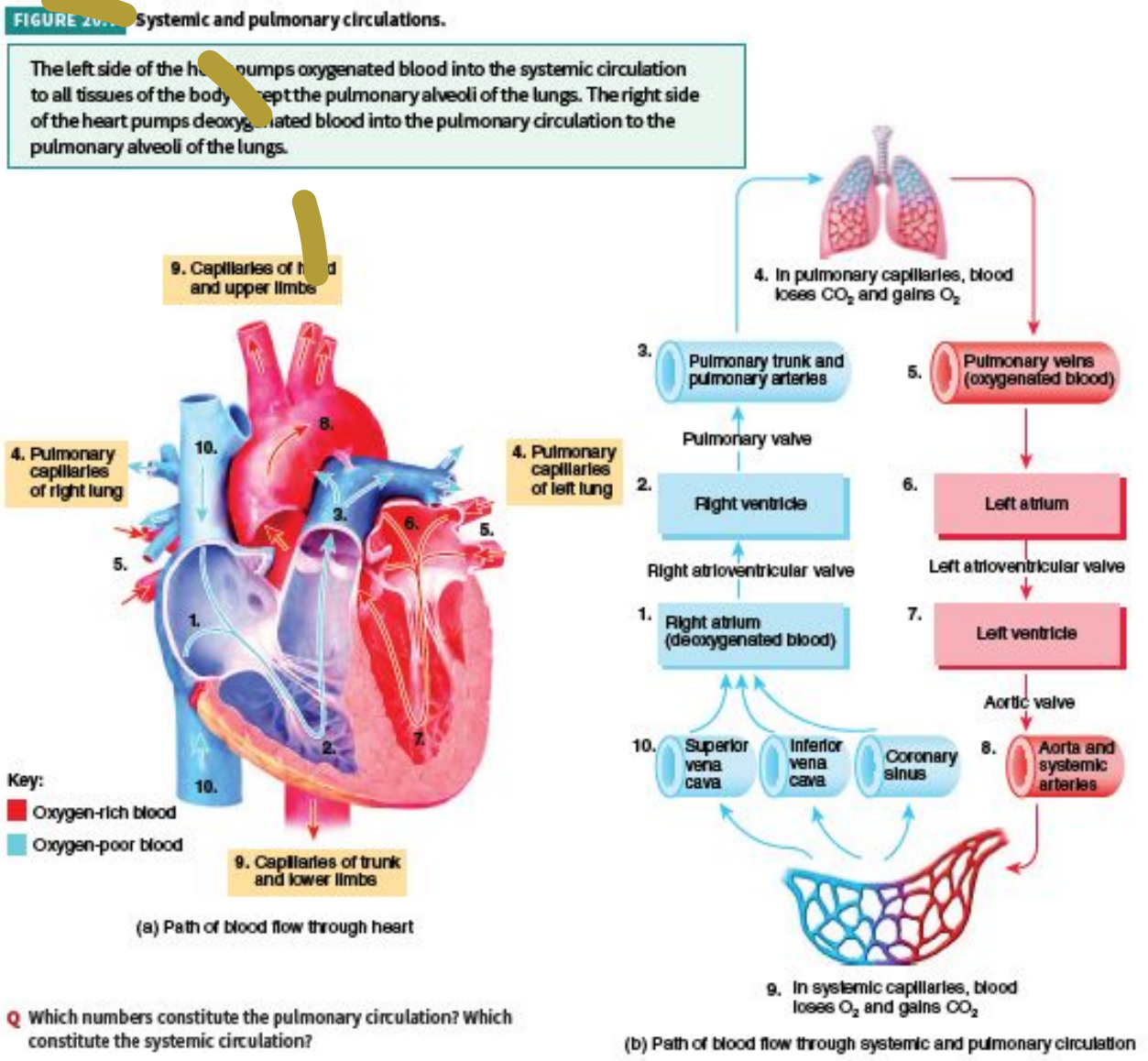
Operation of Semilunar Valve



- Semilunar (tdd dari 3 katup berbentuk spt setengah bulan), menempel pada dinding arteri
- Katup ini membuat darah dari jantung masuk ke arteri dan mencegah backflow/darah kembali ke ventrikel
- Ujung bebasnya terprojeksi ke lumen arteri
- Vent kontraksi tek vent lebih tinggi dari tek arteri ejeksi ventrikel trunkus pulmonalis dan aorta
- Vent relaksasi darah kembali ke darah free edges dari katup terisi darah katup menutup

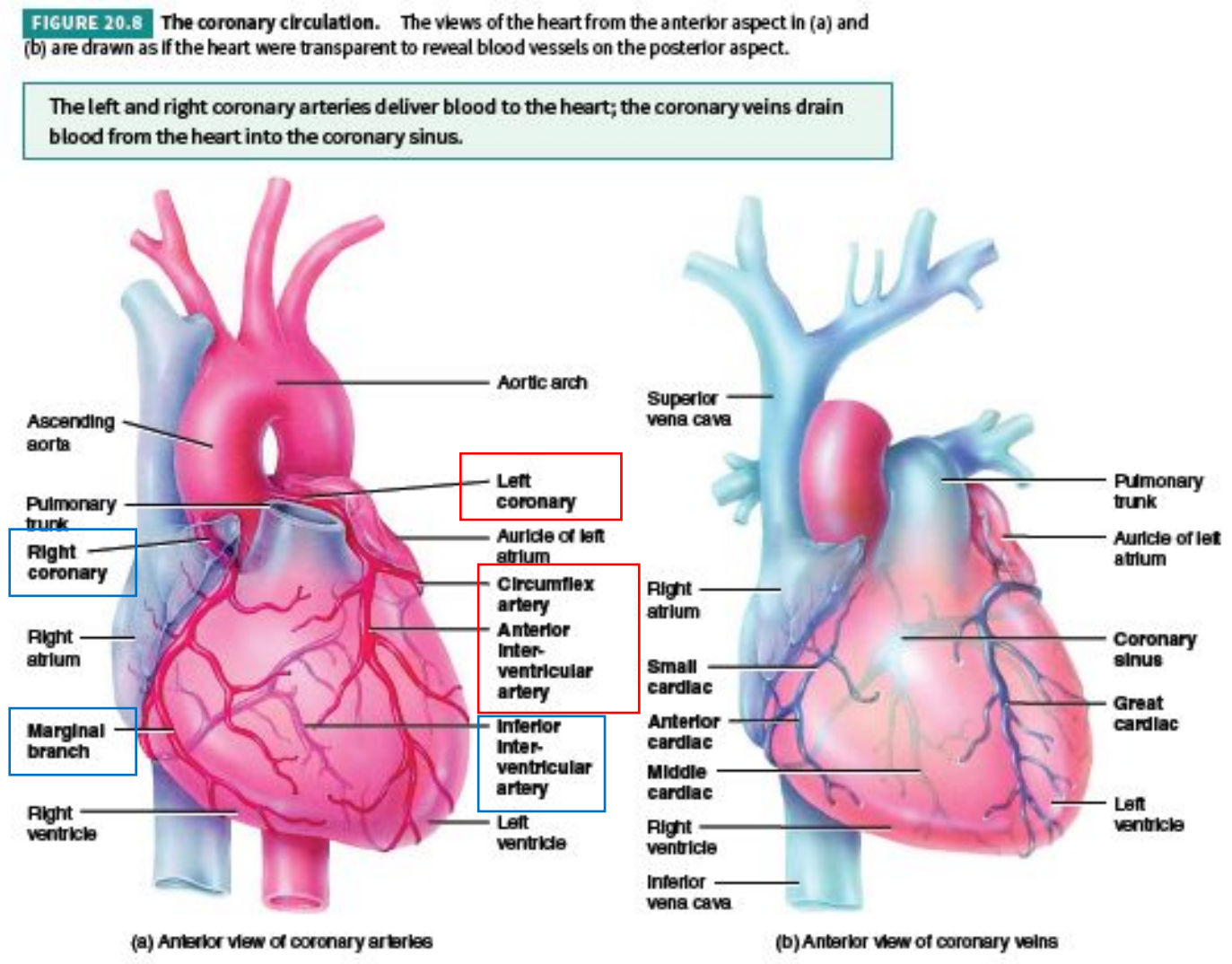
Sirkulasi Sistemik dan Pulmonal

- Sirkulasi sistemik: Ventrikel kiri (oxygen rich)-aorta-arteri sistemik-arteriol-kapiler-exchange nutrients and gases-venula (oxygen-poor)-vena sistemik-atrium kanan
- Sirkulasi pulmonal: ventrikel kanan (oxygen-poor)-trunkus pulmonalis-arteri pulmonalis-paru kanan dan kiri-kapiler pulmonal-unload CO2 dan picks up O2-vena pulmonalis-atrium kiri



Sirkulasi Koroner

- Arteri coroner (coron=crown), jad arteri ini bercabang dari aorta asenden dan melingkari jantung seperti mahkota/crown
- Saat jantung kontraksi, aliran darah coroner sedikit krn tertekan
- Saat jantung relaksasi, tek darah yg tinggi di aorta mengalirkan darah ke art koroner-kapiler-vena coroner
- Tdd art coronaria sinistra dan dextra



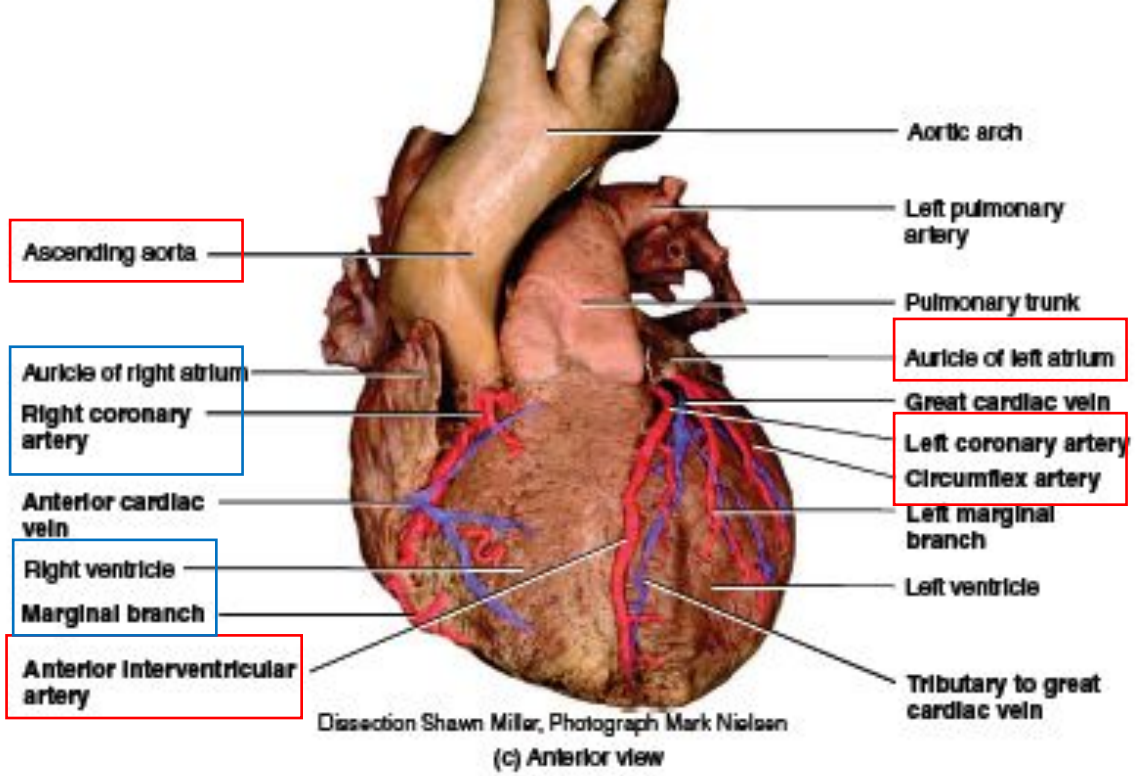
Sirkulasi Koroner

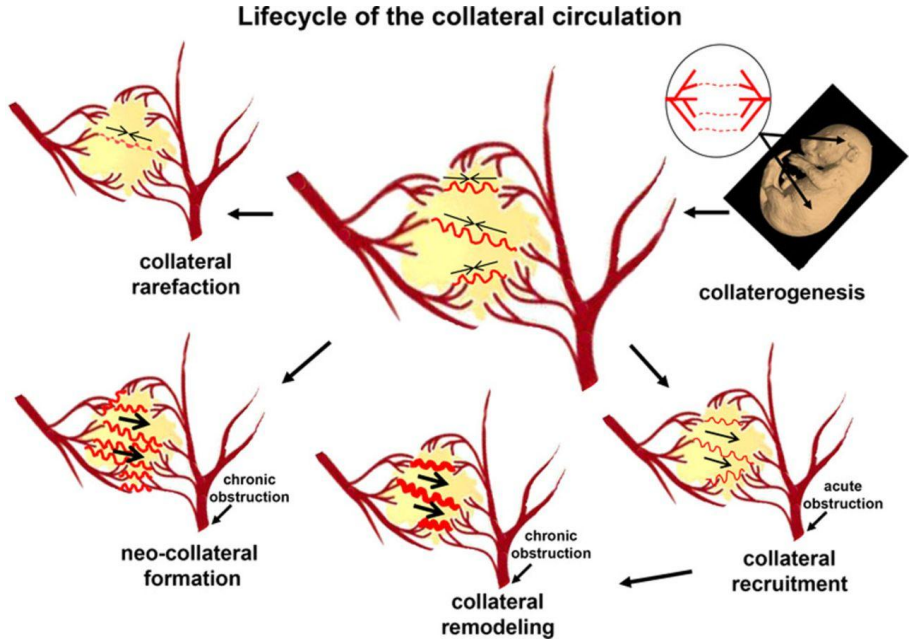
- Art coroner:

-Kiri: ke inferior menuju auricula kiri, terbagi menjadi interventricular anterior/Left anterior descending pd dinding anterior sulkus interventricular; dan circumflexa pd sulkus koronarius atrium dan vent kiri

-Kanan: suplai atrial branches ke atrium kanan; ke inferior menuju aurikula kanan, terbagi menjadi interventricular posterior pd sulkus interven post suplai ventrikel dg darah oxygen rich; marginal pd sulkus koronarius batas kanan jantung ventrikel kanan

- Anastomosis collateral circulation detour if coronary obstructed





Sirkulasi Koroner

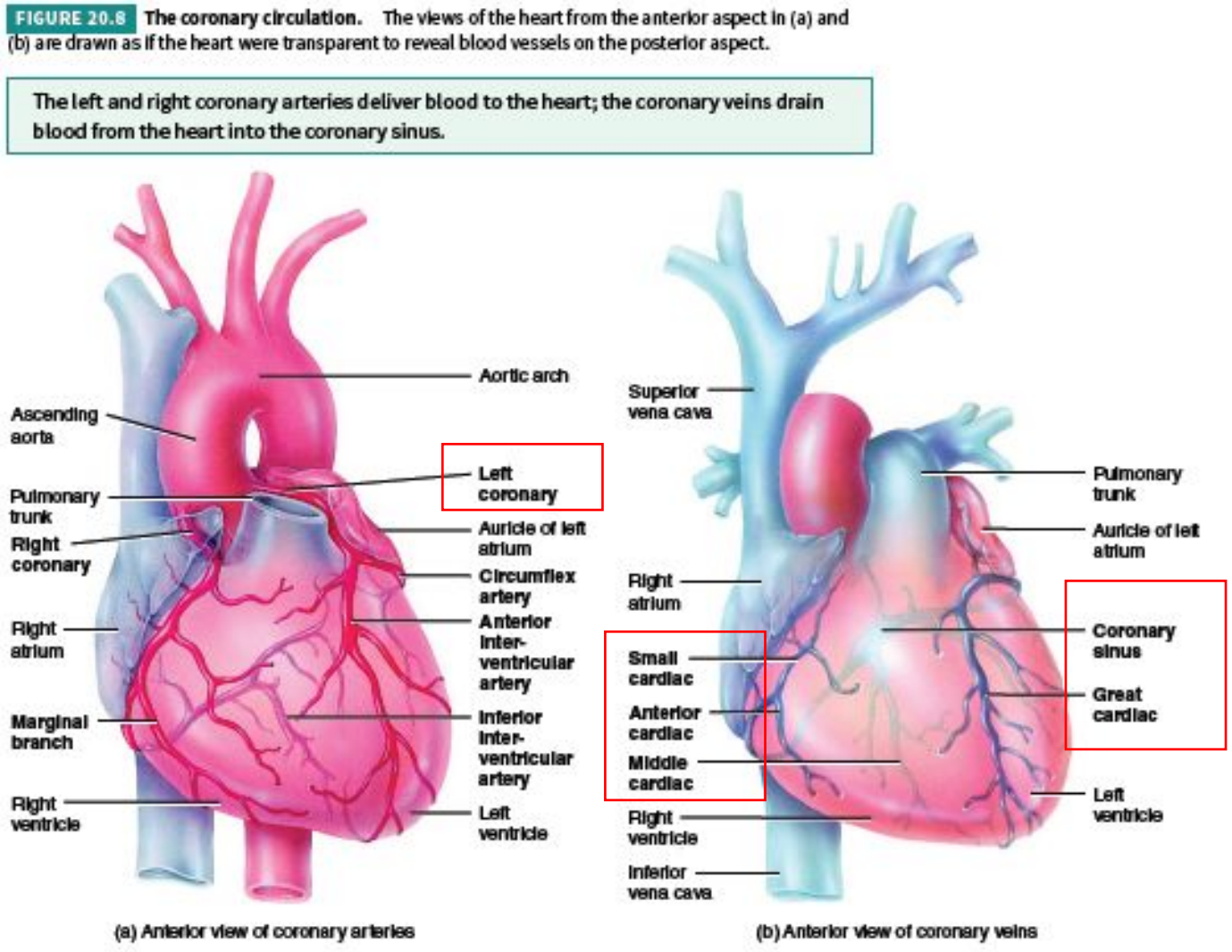
- Darah dari art coroner kapiler delivers O2, nutrient ke otot jtg CO2 dan waste vena coroner
- Sebagian besar darah oxygen poor masuk ke sinus koronarius atrium kanan
- Vena yang membawa darah menuju sinus koronarius:

-Vena cardiaca magna/great cardiac vein di sulkus intervent ant

-Vena cardiaca media/middle cardiac vein di sulkus intervent post

-Vena cardiaca parva/small cardiac vein di sulkus koronarius

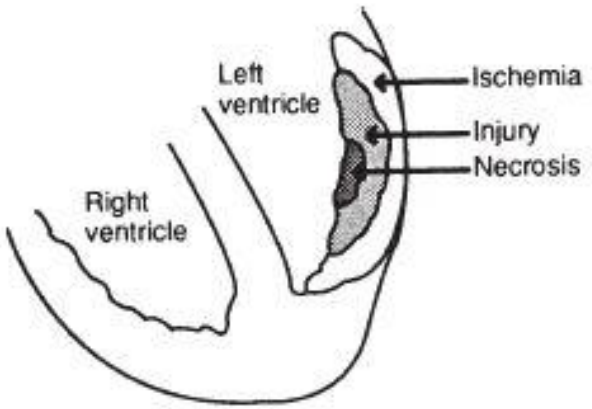
-Vena cardiaca anterior, mengalirkan darah dari vent kanan



Zones of Anatomic or Functional Loss

Necrosis	Injury	Ischemia
No O_2 or blood supply	Little or no O_2 and blood supply	Minimal O_2 and blood supply
Noncontractile	Noncontractile	Poor contractility
Electrically inert	Electrically unstable (arrhythmias)	Electrically unstable (arrhythmias)
ECG = deep or wide Q wave	ECG = elevated ST segment	ECG = inverted T-wave

Note: Myocardial cells begin to die (necrosis) if ischemic areas are not reperfused within 20 minutes.

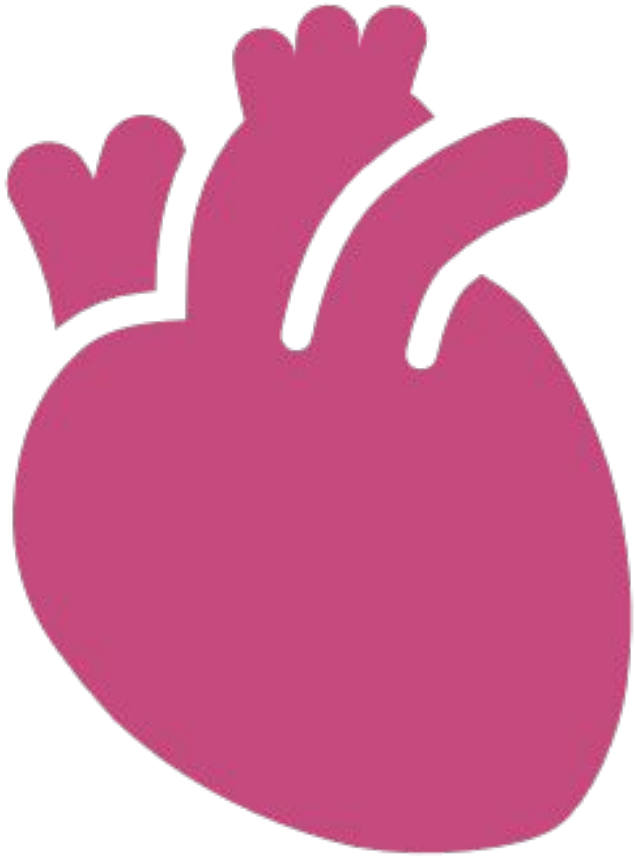


Clinical Connection

Myocardial Ischemia and Infarction

Partial obstruction of blood flow in the coronary arteries may cause **myocardial ischemia** (is-KĒ-mē-a; *ische-* = to obstruct; *-emia* = in the blood), a condition of reduced blood flow to the myocardium. Usually, ischemia causes **hypoxia** (hī-POKS-ē-a = reduced oxygen supply), which may weaken cells without killing them. **Angina pectoris** (an-JĪ-na, or AN-ji-na, PEK-tō-ris), which literally means "strangled chest," is a severe pain that usually accompanies myocardial ischemia. Typically, sufferers describe it as a tightness or squeezing sensation, as though the chest were in a vise. The pain associated with angina pectoris is often referred to the neck, chin, or down the left arm to the elbow. **Silent myocardial ischemia,** ischemic episodes without pain, is particularly dangerous because the person has no forewarning of an impending heart attack.

A complete obstruction to blood flow in a coronary artery may result in a **myocardial infarction (MI)** (in-FARK-shun), commonly called a *heart attack*. *Infarction* is the death of an area of tissue because of interrupted blood supply. Because the heart tissue distal to the obstruction dies and is replaced by noncontractile scar tissue, the heart muscle loses some of its strength. Depending on the size and location of the infarcted (dead) area, an infarction may disrupt the conduction system of the heart and cause sudden death by triggering ventricular fibrillation. Treatment for a myocardial infarction may involve injection of a thrombolytic (clot-dissolving) agent such as streptokinase or tPA, plus heparin (an anticoagulant), or performing coronary angioplasty or coronary artery bypass grafting. Fortunately, heart muscle can remain alive in a resting person if it receives as little as 10–15% of its normal blood supply.



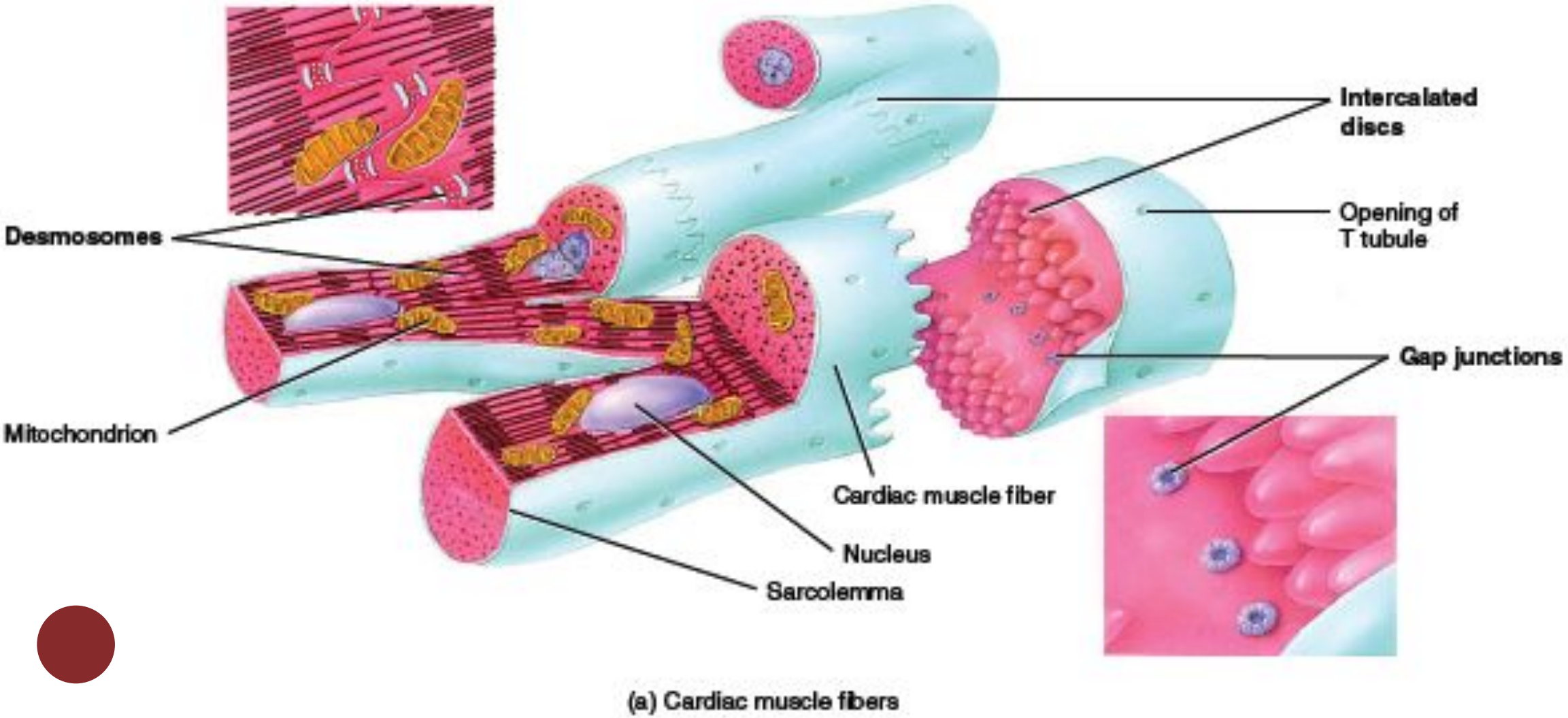
Checkpoint

1. What causes the heart valves to open and to close? What supporting structures ensure that the valves operate properly?
2. In correct sequence, which heart chambers, heart valves, and blood vessels would a drop of blood encounter as it flows from the right atrium to the aorta?
3. Which arteries deliver oxygenated blood to the myocardium of the left and right ventricles?

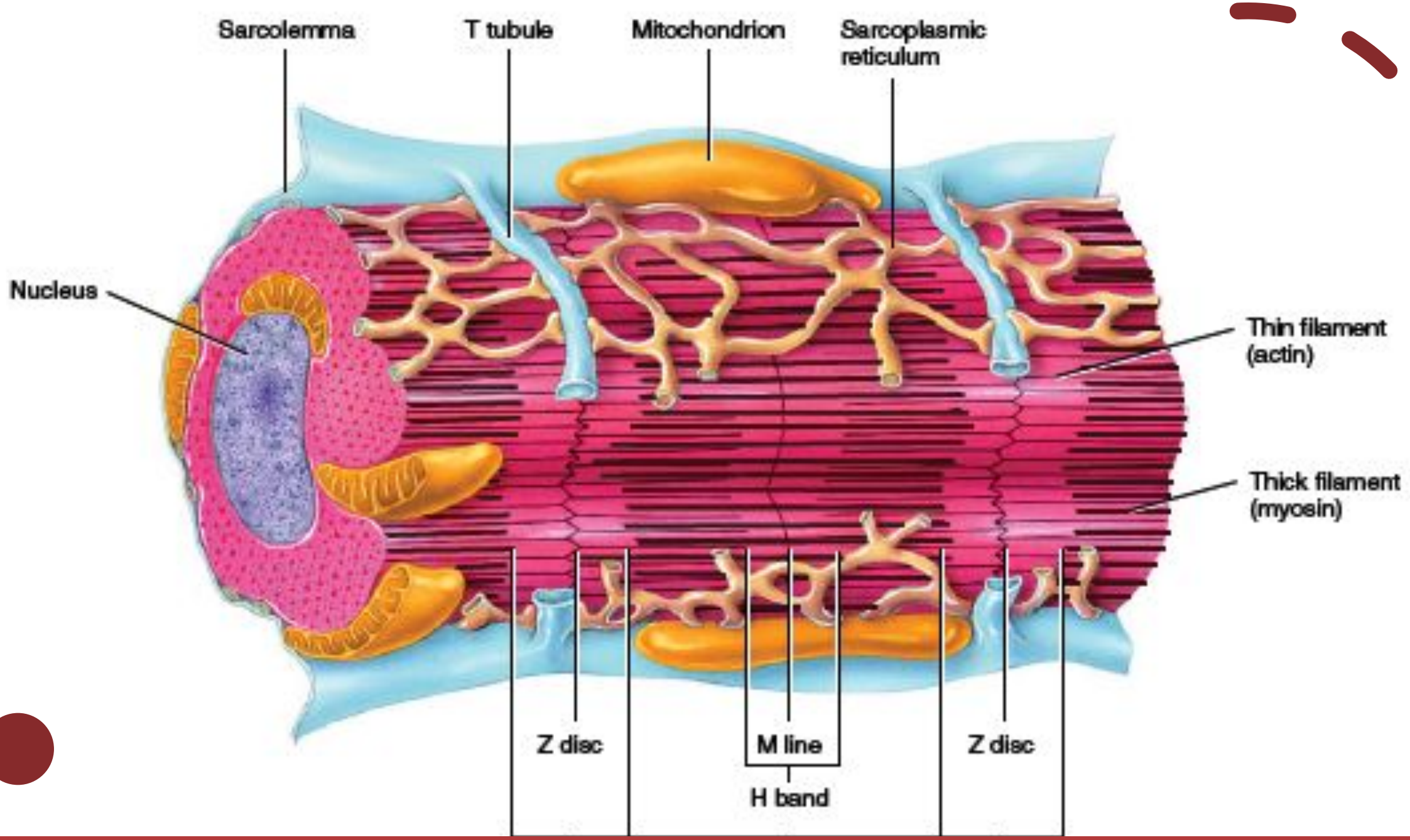
Cardiac Muscle compared to skeletal muscle

- Serabut otot jantung lebih pendek, lebih kurang bundar drpd otot skelet
- Panjang 50-100μm, diameter 14 μm
- 1 Nucleus di tengah/sentral, terkadang ada 2 nuclei
- Ujungnya terhubung dengan serabut oleh diskus intercalatus (tdd desmosome dan gap junctions)
- Mitokondria lebih besar dan banyak (25% dari ruang sitosol), pada otot skelet hanya 2%
- Tdd actin dan myosin, same bands, zones, and Z discs
- Tubulus T lebih lebar dan lebih sedikit, 1 tubulus per sarkomer terletak pada Z disc
- Retikulum sarkoplasma lebih kecil smaller intracellular reserve of Ca2+

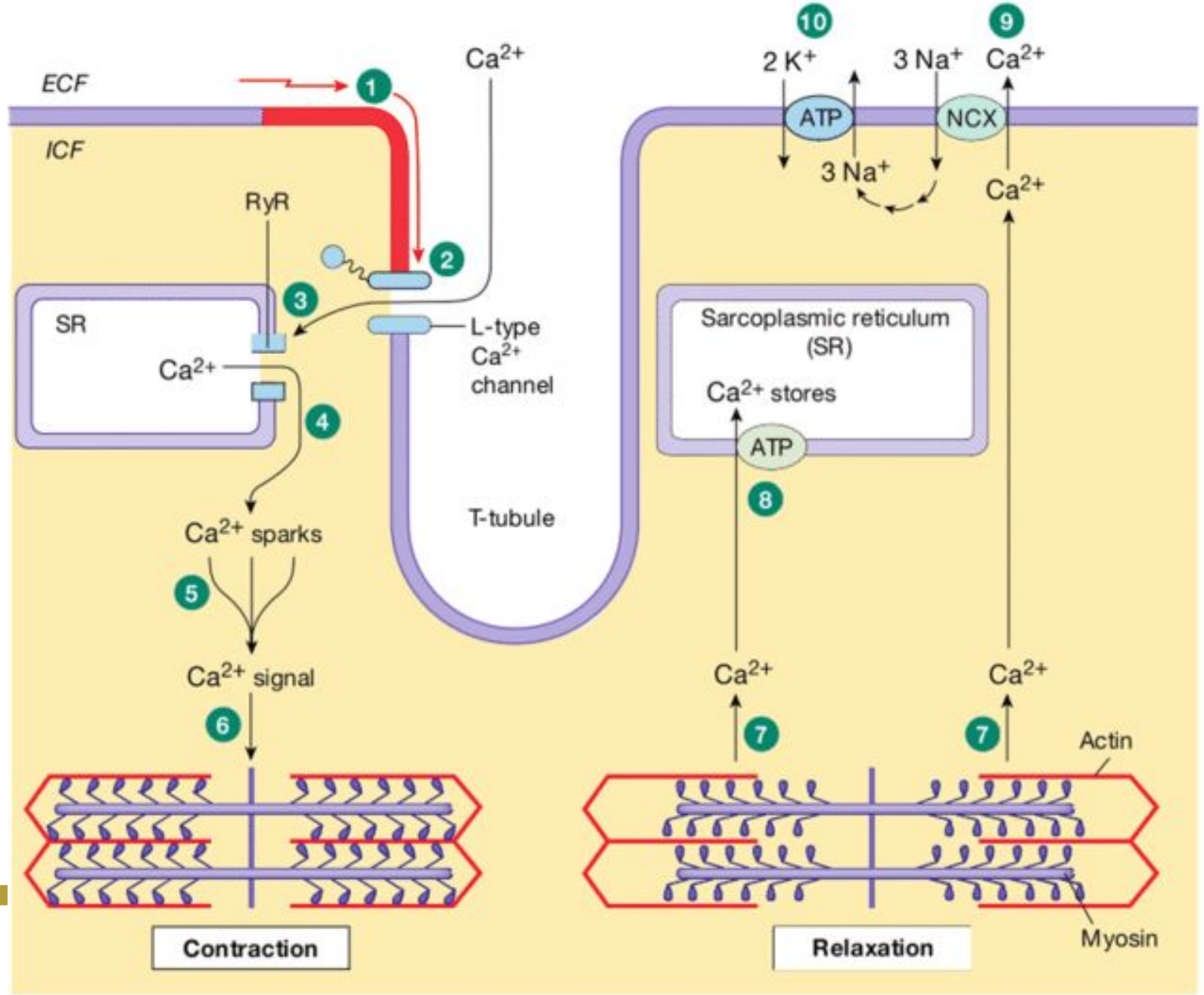
Cardiac muscle fibers connect to neighboring fibers by intercalated discs, which contain desmosomes and gap junctions.



(a) Cardiac muscle fibers

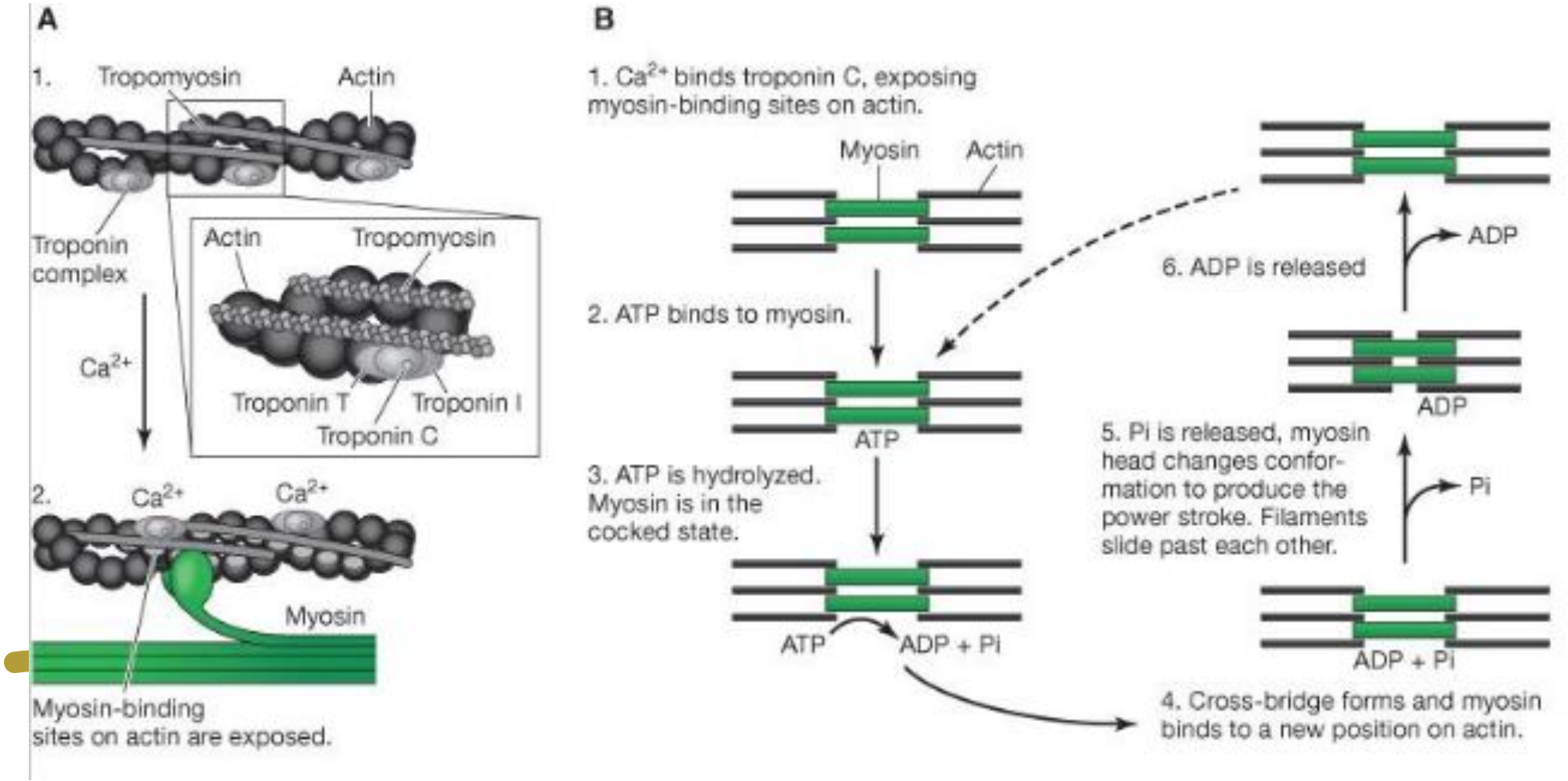

Sarcolemma
T tubule
Mitochondrion
Sarcoplasmic reticulum
Nucleus
Thin filament (actin)
Thick filament (myosin)
Z disc
M line
H band
Z disc

Excitation-Contraction Coupling



1. Action potential enters from adjacent cell.
2. Voltage-gated Ca^{2+} channels open. Ca^{2+} enters cell.
3. Ca^{2+} induces Ca^{2+} release through ryanodine receptor-channels (RyR).
4. Local release causes Ca^{2+} spark.
5. Summed Ca^{2+} sparks create a Ca^{2+} signal.
6. Ca^{2+} ions bind to troponin to initiate contraction.
7. Relaxation occurs when Ca^{2+} unbinds from troponin.
8. Ca^{2+} is pumped back into the sarcoplasmic reticulum for storage.
9. Ca^{2+} is exchanged with Na^+ by the NCX antiporter.
10. Na^+ gradient is maintained by the Na^+-K^+-ATPase.

Cardiac Muscle Contraction



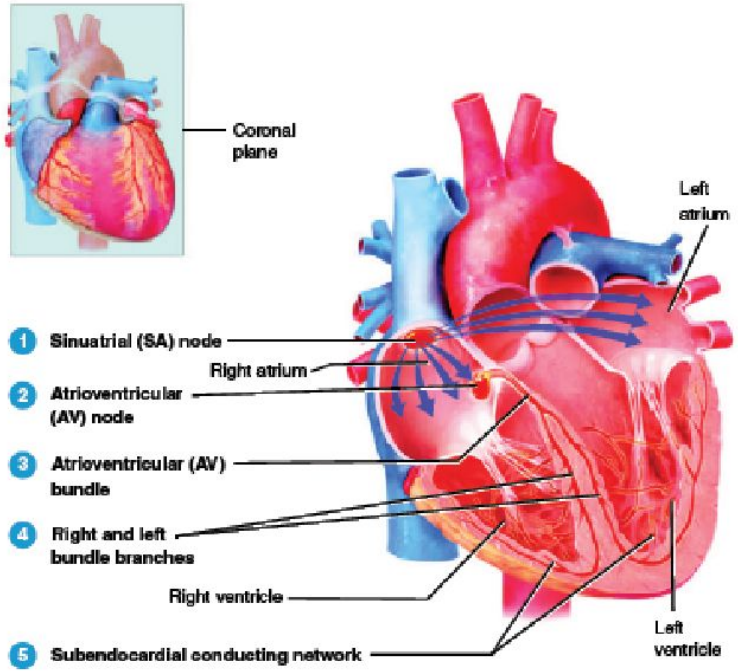
Cardiac Excitation Contraction Coupling

Sistem Konduksi: Serabut autoritmik

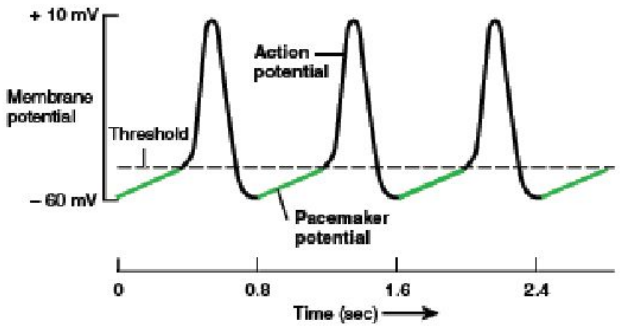
- Jantung dapat bertahan sepanjang hidup karena memiliki aktivitas listrik yang inherent (permanen) dan ritmikal
- Sumber aktivitas listrik ini berasal dari serabut autoritmik (karena self-excitable), secara berulang menghasilkan potensial aksi yang merangsang kontraksi jantung
- Saat jantung dikeluarkan dari tubuh dan semua sarafnya dipotong, ia dpt tetap terstimulasi (misalkan saat transplantasi jantung). Ahli bedah tidak menempelkan kembali saraf jantung saat transplantasi. Jadi ahli bedah jantung lebih tepat disebut 'plumbers' daripada 'electricians'
- Selama perkembangan embrio, hanya 1% serabut otot jantung yang berubah menjadi serabut autoritmik (sbg natural pacemaker, dan pembentuk sistem konduksi jantung)

FIGURE 20.10 **The conduction system of the heart.** Autorhythmic fibers in the SA node, located in the right atrial wall (a), act as the heart's pacemaker, initiating cardiac action potentials (b) that cause contraction of the heart's chambers.

The conduction system ensures that the chambers of the heart contract in a coordinated manner.



(a) Anterior view of coronal section

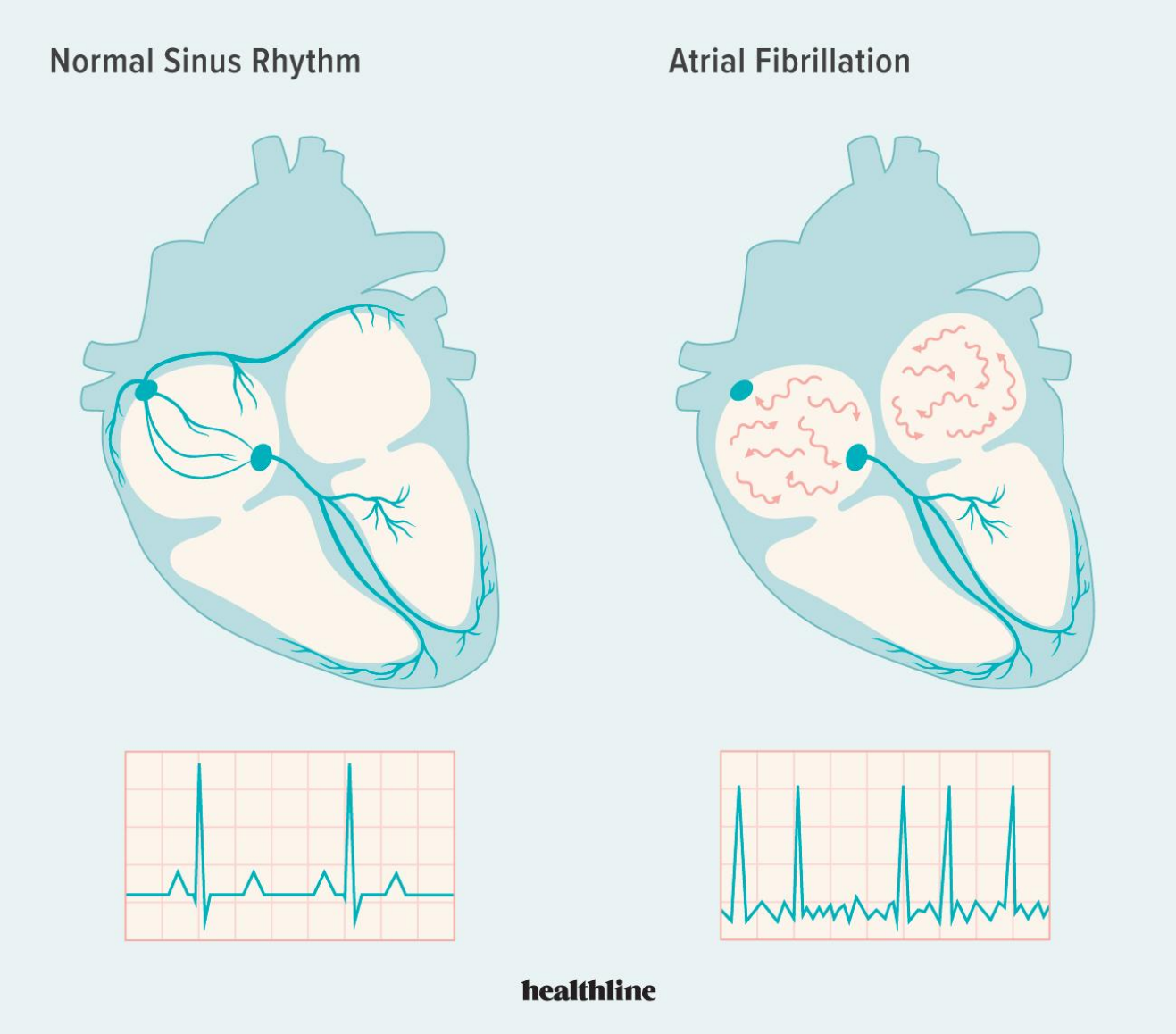


(b) Pacemaker potentials (green) and action potentials (black) in autorhythmic fibers of SA node

Q Which component of the conduction system provides the only electrical connection between the atria and the ventricles?

Sistem Konduksi Jantung

1. SA Node, sbg pacemaker, berlokasi di dinding atrium kanan. Aksi potensial diteruskan ke kedua atrium via atrial internodal tracts
2. AV Node, di septum interatrial, slows karena perbedaan variasi struktur sel delay utk atrium mengosongkan darah
3. AV Bundle/His, aksi potensial diteruskan ke ventrikel, sedangkan di tempat lain fibrous skeleton mjd isolator listrik
4. Left and right bundle branches septum interventricular apex jantung
5. Purkinje fibers cepat mengkonduksi aksi potensial dari apex ke atas menuju seluruh ventrikel kontraksi ventrikel

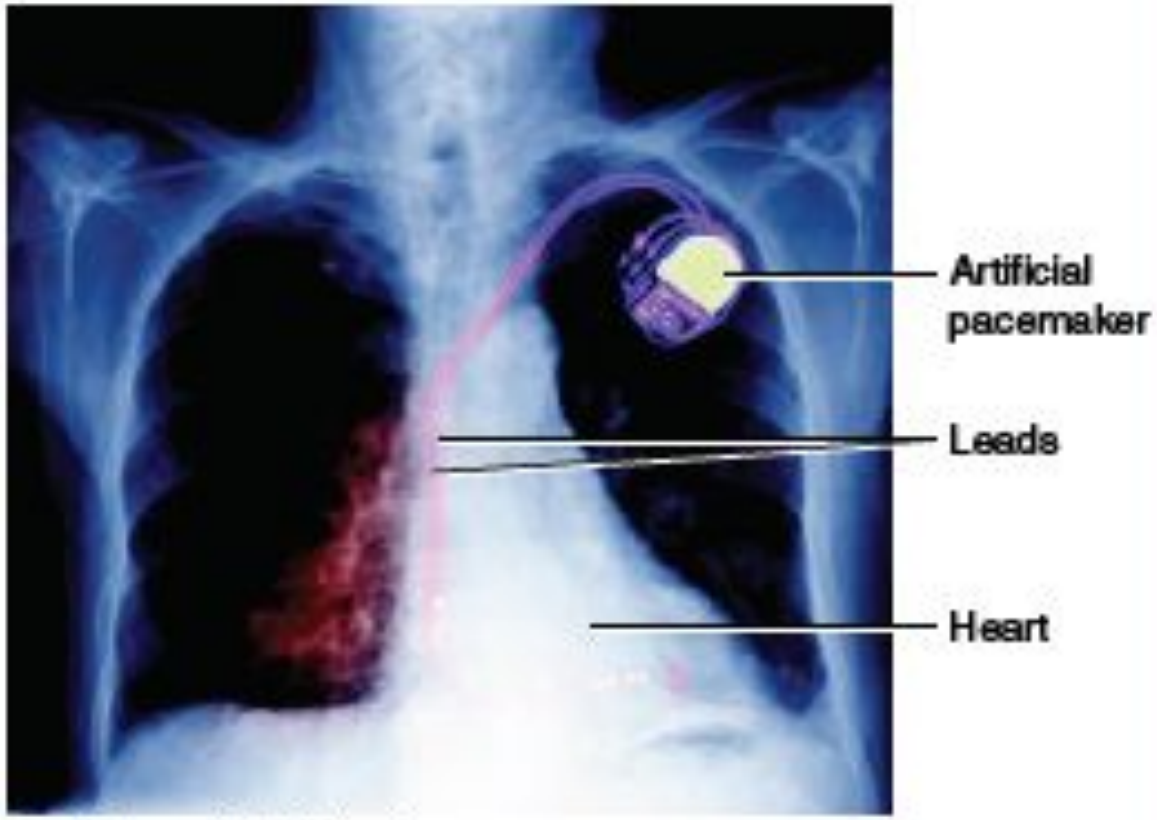


Normal Sinus Rhythm

1. The action potential must originate in the SA node.
2. The SA nodal impulses must occur regularly at a rate of 60 to 100 impulses per minute.
3. The activation of the myocardium must occur in the correct sequence and with the correct timing and delays.

Clinical Connection

Artificial Pacemakers



Scott Camazine/Medical Images

When heart rate is abnormally low, normal heart rhythm can be restored and maintained by surgically implanting an **artificial pacemaker**, a device that sends out small electrical currents to stimulate the heart to contract. An artificial pacemaker consists of a battery and impulse generator and is usually implanted beneath the skin just inferior to the clavicle. The pacemaker is connected to one or two flexible leads (wires) that are threaded through the superior vena cava and then passed into the various chambers of the heart. Many of the newer pacemakers, referred to as *activity-adjusted pacemakers*, automatically speed up the heartbeat during exercise.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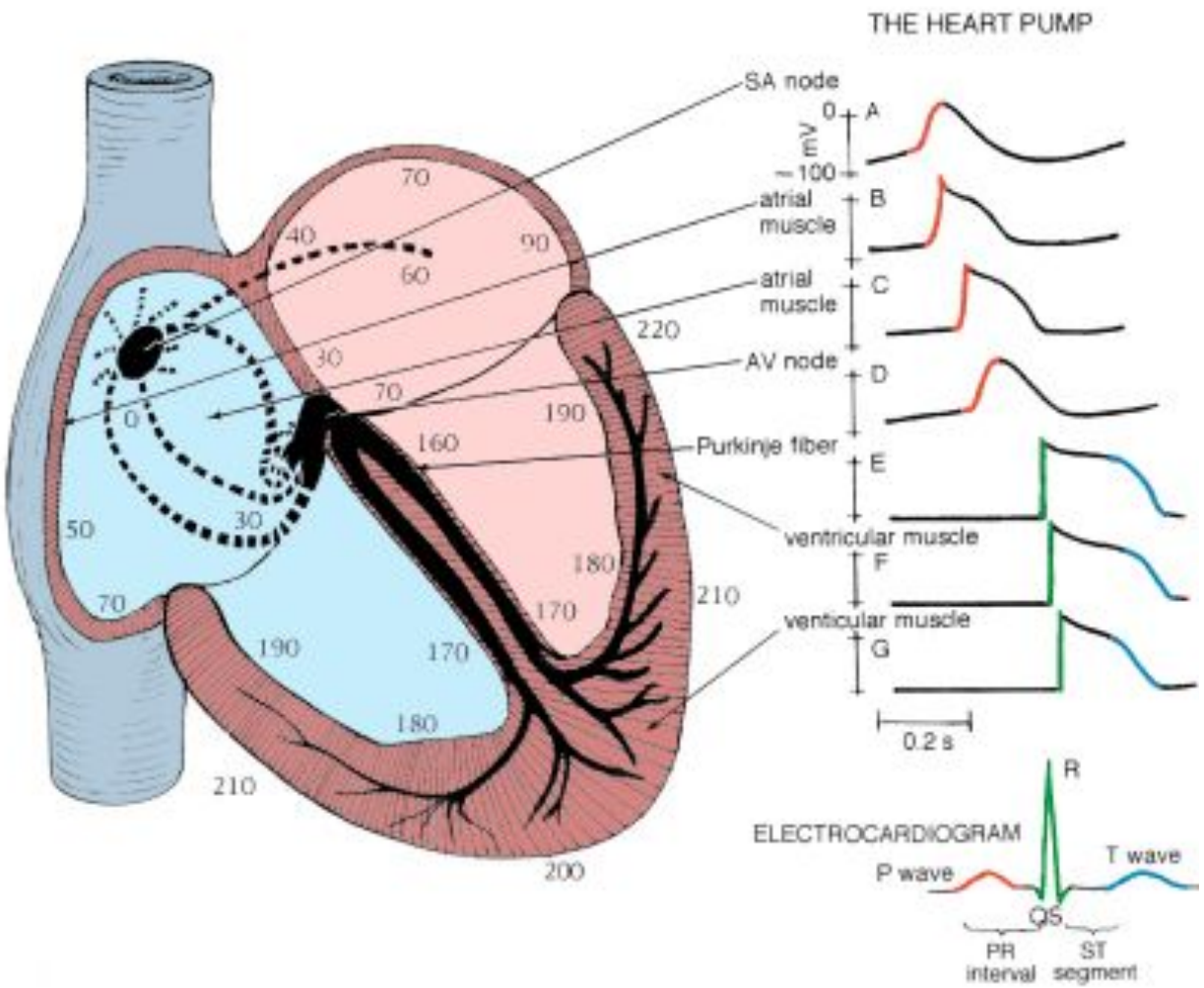
Cardiac Action Potential



Depolarization-repolarization cycle

The depolarization-repolarization cycle consists of the following phases:

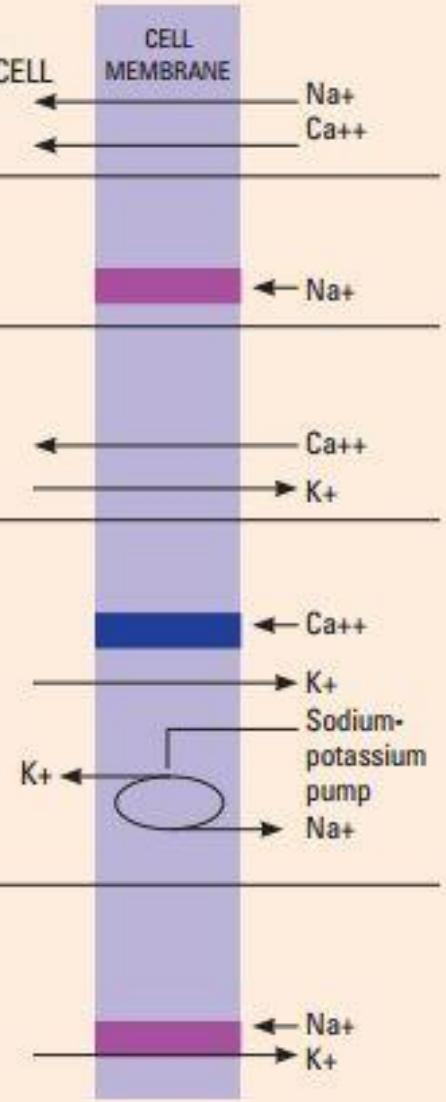
Phase 0:
Rapid depolarization
- Sodium (Na^+) moves rapidly into cell.
- Calcium (Ca^{++}) moves slowly into cell.

Phase 1:
Early repolarization
- Sodium channels close.

Phase 2:
Plateau phase
- Calcium continues to flow in.
- Potassium (K^+) continues to flow out.

Phase 3:
Rapid repolarization
- Calcium channels close.
- Potassium flows out rapidly.
- Active transport via the sodium-potassium pump begins restoring potassium to the inside of the cell and sodium to the outside of the cell.

Phase 4:
Resting phase
- Cell membrane is impermeable to sodium.
- Potassium moves out of the cell.



Perbandingan Aksi Potensial di SA Node, Atrium, Ventrikel

Table 4–2 Comparison of Action Potentials in Cardiac Tissues

Cardiac Tissue	Action Potential Duration (msec)	Upstroke	Plateau	Phase 4 Depolarization
Sinoatrial node	150	Inward Ca^{2+} current Ca^{2+} channels	None	Inward Na^{+} current (I_f) Normal pacemaker
Atrium	150	Inward Na^{+} current	Inward Ca^{2+} current (slow inward current) L-type Ca^{2+} channels	None
Ventricle	250	Inward Na^{+} current	Inward Ca^{2+} current (slow inward current) L-type Ca^{2+} channels	None
Purkinje fibers	300	Inward Na^{+} current	Inward Ca^{2+} current (slow inward current) L-type Ca^{2+} channels	Latent pacemaker

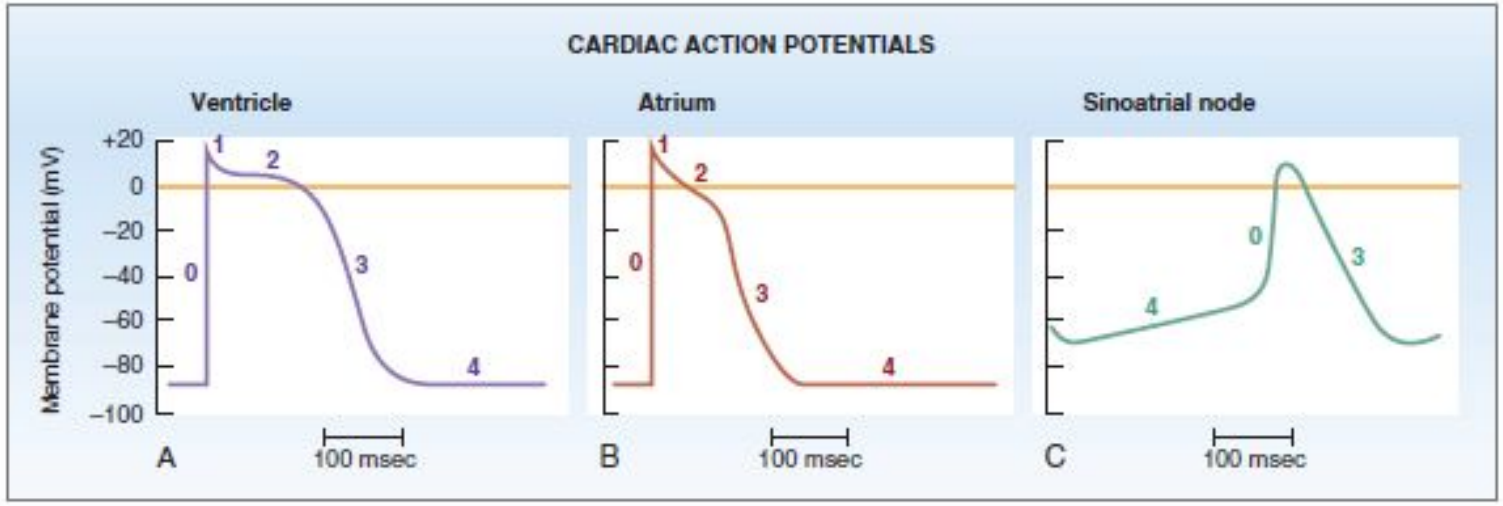


Figure 4–12 **Cardiac action potentials in the ventricle, atrium, and sinoatrial node. A–C,** The numbers correspond to the phases of the action potentials.

SA NODE ACTION POTENTIAL

0. Rapid depolarization: Ca inflow (inward)
1. Absent
2. Absent
3. Repolarisasi: K outflow
4. Spontaneous depolarization/pacemaker potential: paling lama, menentukan kemampuan automatisasi. Maximum diastolic potential:-65 mV. Terjadi Na inflow depolarisasi lambat

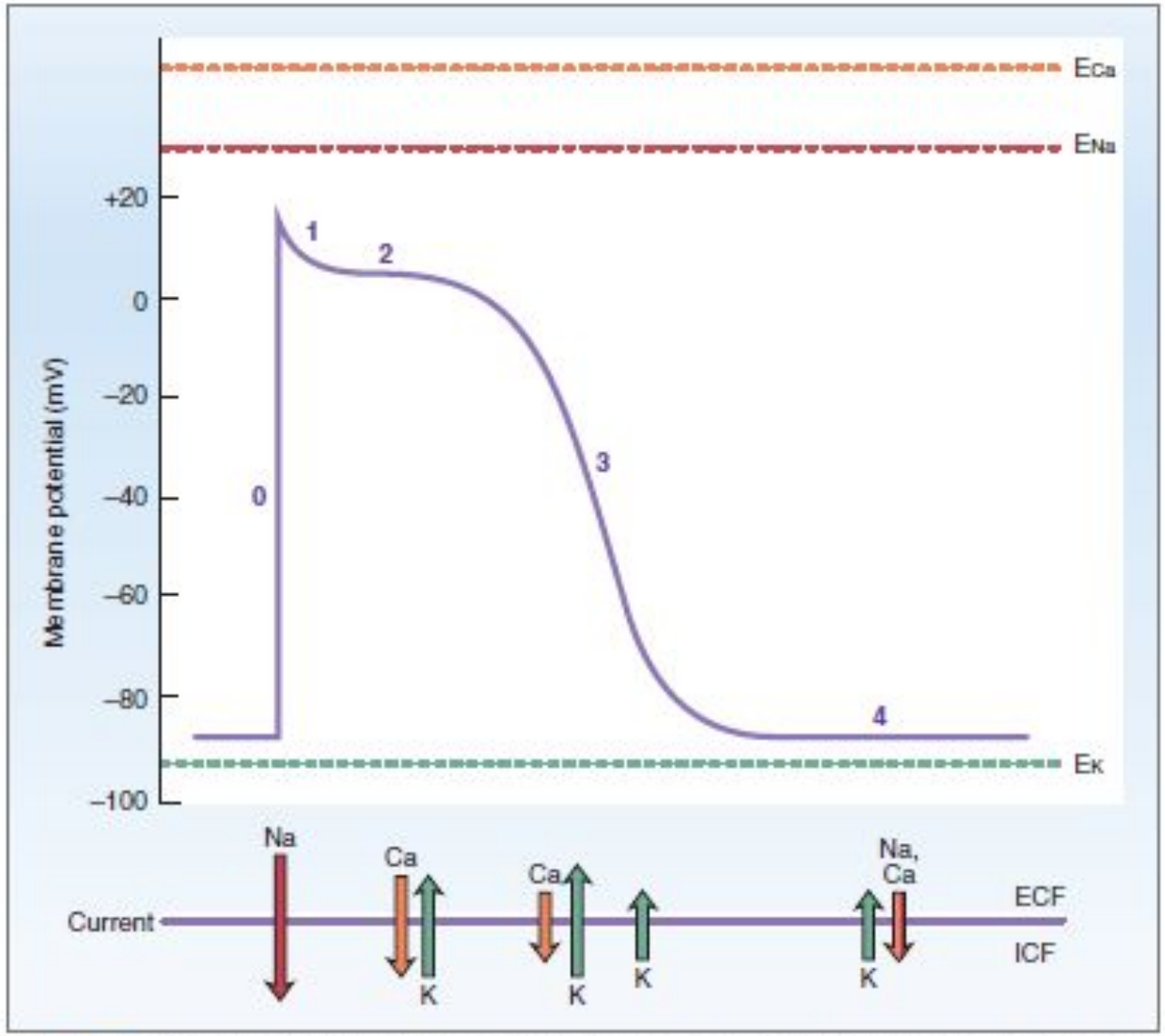


Figure 4–13 Currents responsible for ventricular action potential. The length of the *arrows* shows the relative size of each ionic current. E, Equilibrium potential; ECF, extracellular fluid; ICF, intracellular fluid.

Ventricle Action Potential

0. Rapid depolarization: Na+ inflow (inward)
1. Initial repolarization: K+ outflow (outward)
2. Plateau: Ca inflow dan K outflow
3. Repolarisasi: K outflow, Ca channel tertutup
4. Resting membrane potential/Electric diastole: dipertahankan oleh equilibrium K, Na, dan Ca

Mengapa SA Node menjadi pacemaker?

Table 4-3 Firing Rate of Sinoatrial Node and Latent Pacemakers in the Heart

Location	Intrinsic Firing Rate (impulses/min)
Sinoatrial node	70–80
Atrioventricular node	40–60
Bundle of His	40
Purkinje fibers	15–20

Latent Pacemakers

The cells in the SA node are not the only myocardial cells with intrinsic automaticity; other cells, called **latent pacemakers,** also have the capacity for spontaneous phase 4 depolarization. Latent pacemakers include the cells of the **AV node**, **bundle of His**, **and Purkinje fibers.** Although each of these cells has the potential for automaticity, it normally is not expressed.

The rule is that *the pacemaker with the fastest rate of phase 4 depolarization controls the heart rate.* Normally, the SA node has the fastest rate of phase 4 depolarization, and therefore, it sets the heart rate (Table 4-3). Recall also that, of all myocardial cells, the SA nodal cells have the shortest action potential duration (i.e., the shortest refractory periods). Therefore, SA nodal cells recover faster and are ready to fire another action potential before the other cell types are ready.

Kapan latent pacemaker menjadi pacemaker menggantikan SA Node?

Mengapa kecepatan konduksi berbeda-beda ?

Conduction Velocity	
Atria	1 m/sec
AV node	0.01–0.05 m/sec
His-Purkinje	2–4 m/sec
Ventricle	1 m/sec

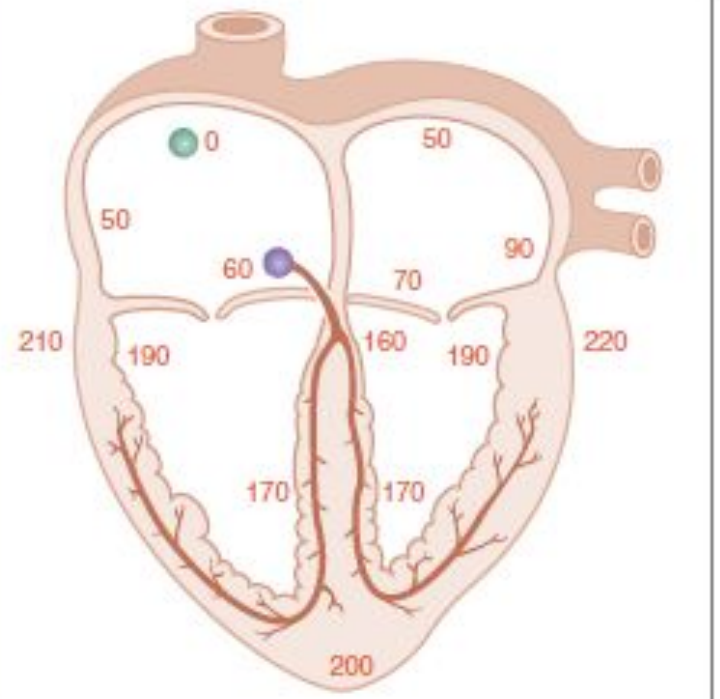


Figure 4–14 Timing of activation of the myocardium. The numbers superimposed on the myocardium indicate the cumulative time, in msec, from the initiation of the action potential in the sinoatrial node.

- Kecepatan konduksi di miokardium berbeda-beda, paling lambat di AV Node (0.01-0.05 m/s), paling cepat di His-Purkinje (2-4 m/s)
- Konduksi dimulai dari SA Node (0), menuju ke titik terjauh di ventrikel selama 220 ms.
- AV delay: konduksi di AV node selama 100 ms (1/2 dari waktu total) memastikan agar ventrikel tidak teraktivasi terlalu awal (terisi penuh dulu)
- Purkinje fast conduction: Aktivasi ventrikel cepat utk ejeksi ventrikel yg efektif

Refractory Period

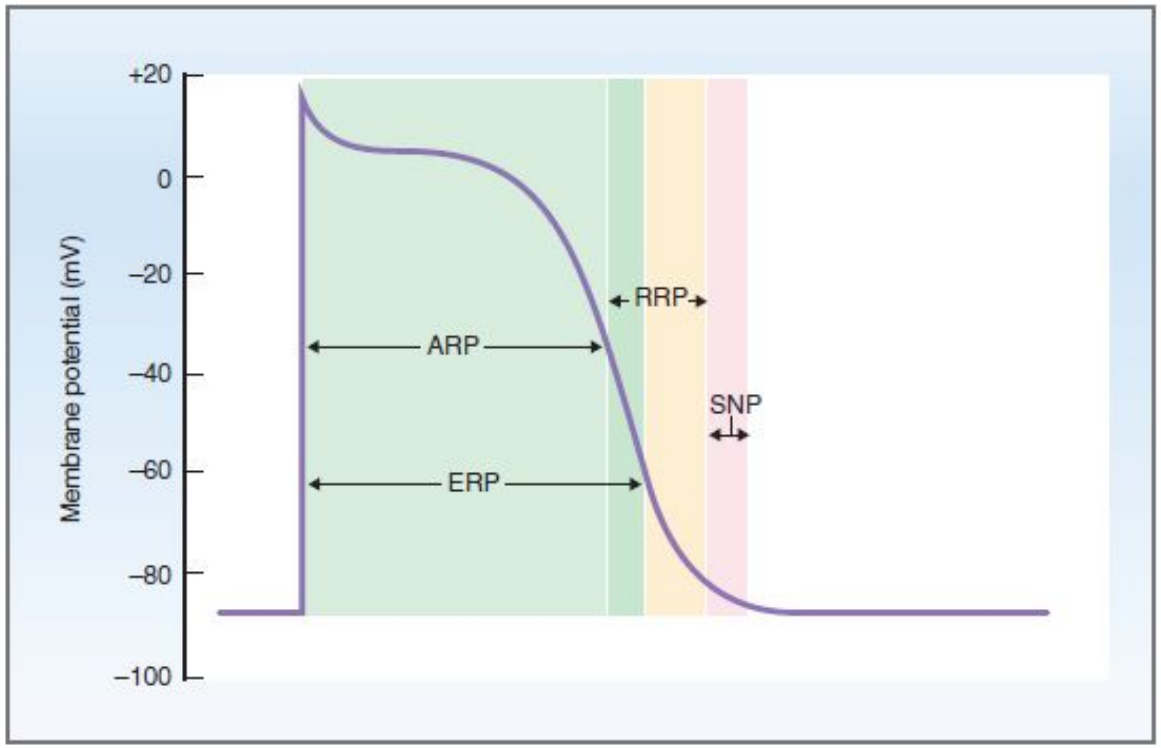


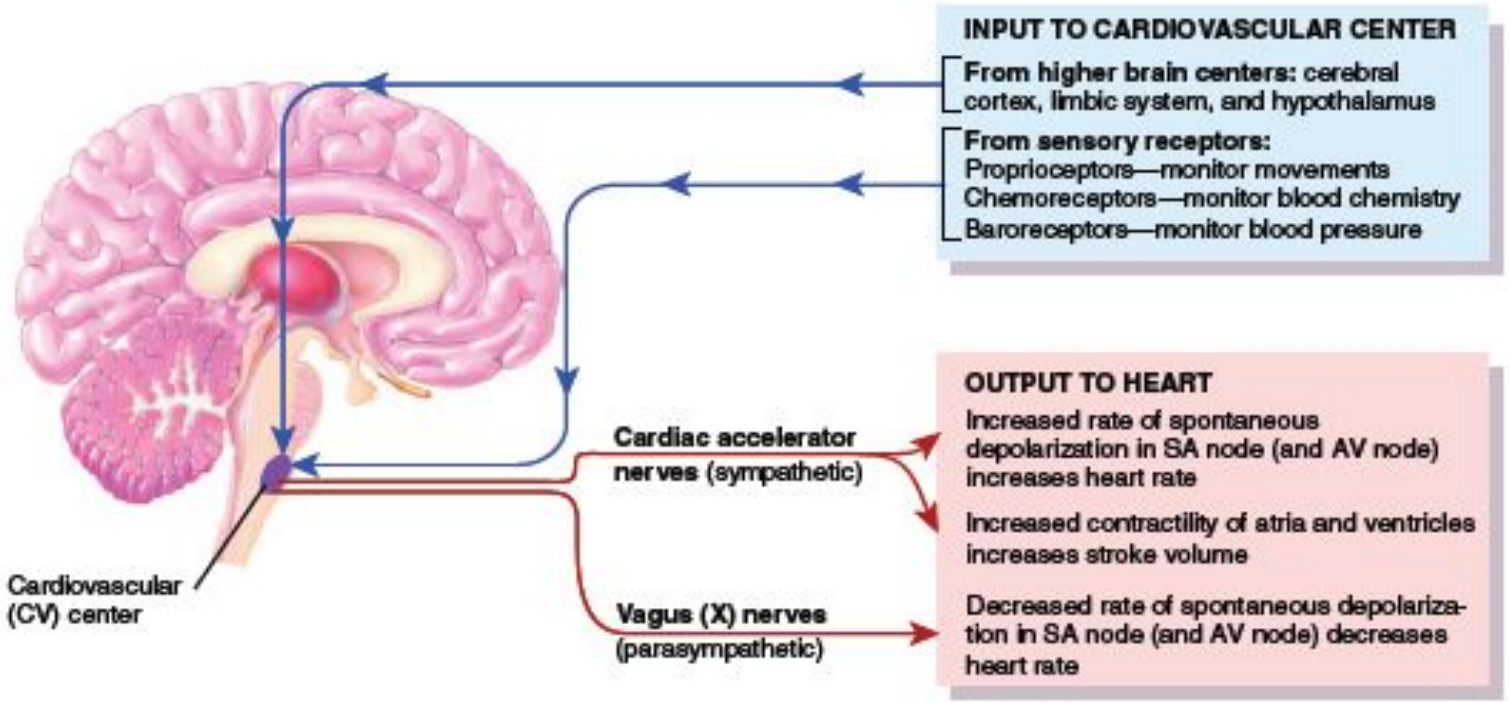
Figure 4–15 Refractory periods of the ventricular action potential. The effective refractory period (ERP) includes the absolute refractory period (ARP) and the first half of the relative refractory period (RRP). The RRP begins when the absolute refractory period ends and includes the last portion of the effective refractory period. The supranormal period (SNP) begins when the relative refractory period ends.

- Absolute Refractory Period (ARP): fase 0-sebag fase 3, tidak ada stimulus yg bisa menimbulkan aksi potensial
- Effective Refractory Period (EFP): sedikit lebih lama dari ARP, aksi potensial tdk bisa ditimbulkan (tdk ada inward yg cukup walau Na mulai recover)
- Relative Refractory Period (RRP): makin bnyak Na yg recover, butuh stimulus lebih besar
- Supranormal Period (SNP): mulai dari -70 sd -85 mV, lebih mudah dirangsang (stimulus yg dibutuhkan tidak sebesar pada fase RRP

Regulation of Heart Rate

FIGURE 20.16 **Nervous system control of the heart.**

The cardiovascular center in the medulla oblongata controls both sympathetic (blue) and parasympathetic nerves (red) that innervate the heart.



Q Which region of the heart is innervated by the sympathetic division but not by the parasympathetic division?

- Diatur oleh saraf otonom, CV center di medulla oblongata
- Menerima input dari sistem limbik, korteks serebri, proprioseptor, kemoreseptor, (arcus aorta dan arteri carotid)

Efek Sistem Saraf Otonom terhadap Jantung

Table 4–4 Effects of Autonomic Nervous System on the Heart and Blood Vessels

	Sympathetic			Parasympathetic		
	Action	*Receptor*	*Mechanism*	*Action*	*Receptor*	*Mechanism*
Heart rate	↑	β_1	↑ I_f ↑ I_{Ca}	↓	M_2	↓ I_f ↑ $I_{K\text{-}ACh}$ ↓ I_{Ca}
Conduction velocity	↑	β_1	↑ I_{Ca}	↓	M_2	↓ I_{Ca} ↑ $I_{K\text{-}ACh}$
Contractility	↑	β_1	↑ I_{Ca} Phosphorylation of phospholamban	↓ (atria only)	M_2	↓ I_{Ca} ↑ $I_{K\text{-}Ach}$
Vascular smooth muscle (skin, renal, and splanchnic)	Constriction	α_1	—	Dilation (releases EDRF)	M_3	—
Vascular smooth muscle (skeletal muscle)	Dilation	β_2	—	Dilation (releases EDRF)	M_3	—
	Constriction	α_1	—			

AV, Atrioventricular; EDRF, endothelial-derived relaxing factor; M, muscarinic.

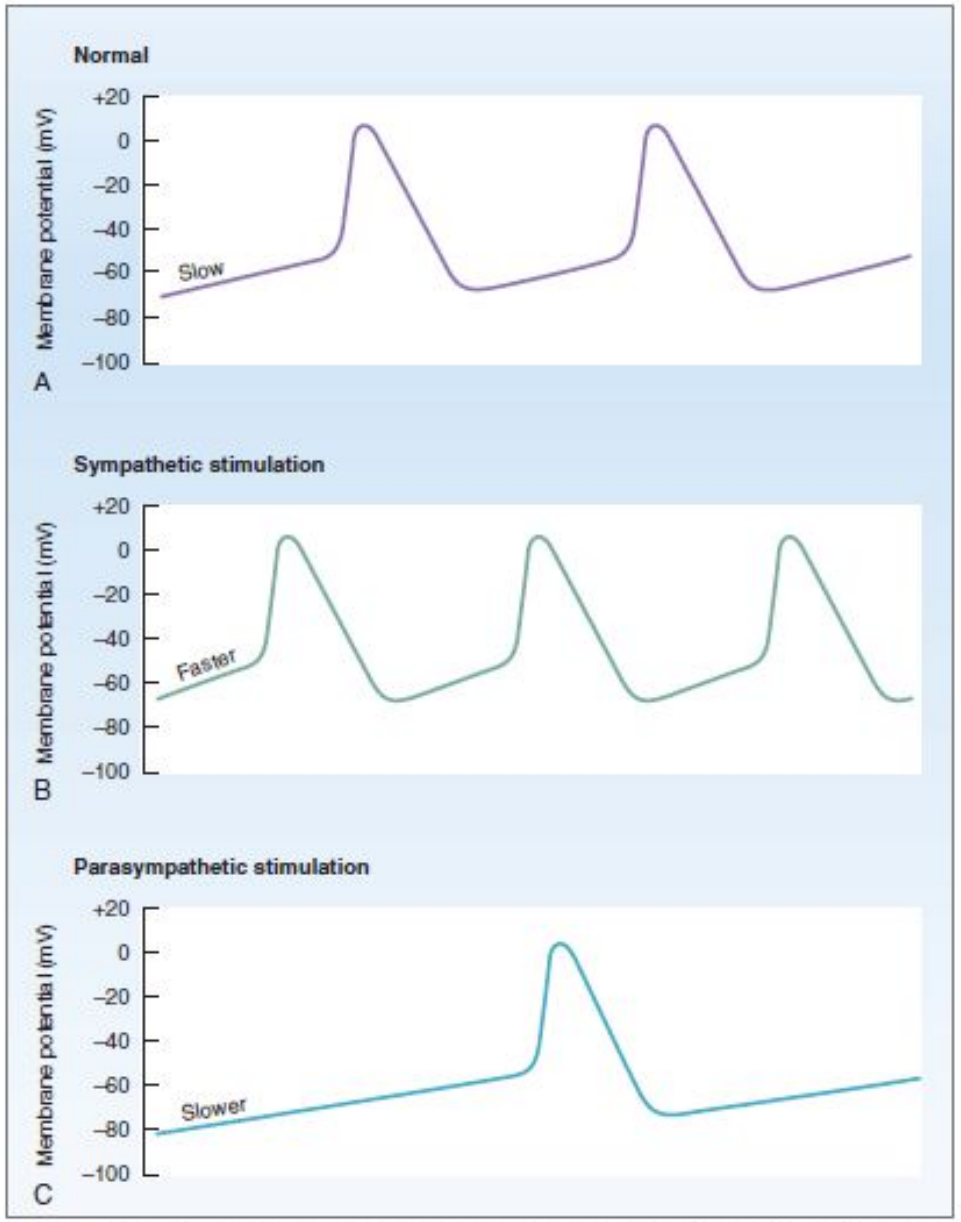


Figure 4–16 Effect of sympathetic and parasympathetic stimulation on the SA node action potential. A, The normal firing pattern of the SA node is shown. **B,** Sympathetic stimulation increases the rate of phase 4 depolarization and increases the frequency of action potentials. **C,** Parasympathetic stimulation decreases the rate of phase 4 depolarization and hyperpolarizes the maximum diastolic potential to decrease the frequency of action potentials.

Efek Simpatis dan Parasimpatis on HR (SA Node)

- Efek Simpatis pada reseptor β1: Chronotropic effect increased HR, CV via

  - increase If (Na inflow di fase 4) increase rate

  - increase Ica (Ca di dalam cukup less depolarization required to reach threshold)

- Efek Parasimpatis pada reseptor M2:

  - slowing the rate of phase 4 depolarization

  - hyperpolarizing the maximum diastolic potential so that more inward current is required to reach threshold potential

  - increasing the threshold potential.

1897 TW Engelmann described:

- **Inotropy:** Contractility
- **Chronotropy:** Rate (SA node)
- **Dromotropy:** Conduction (AV node)
- **Bathmotropy:** Excitability

1982 described:

- **Lusitropy:** Relaxation (active)