Blood circulation and its regulation. The peculiarities of circulation and its regulation in maxillofacial region.

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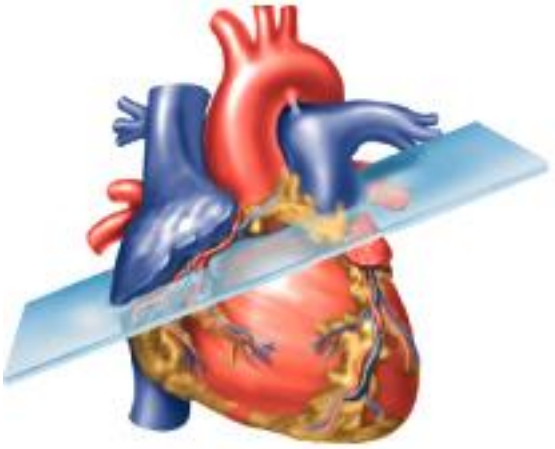
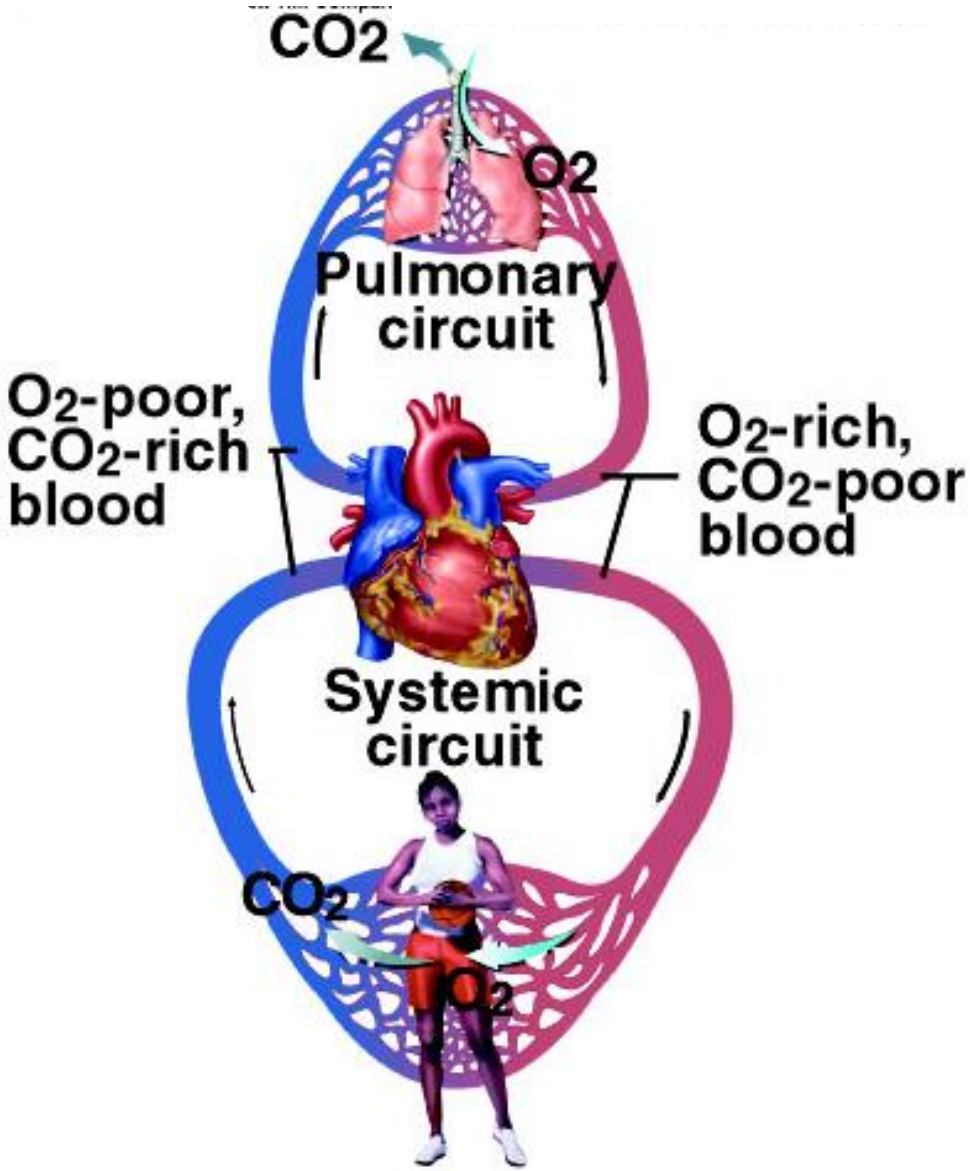
The Heart



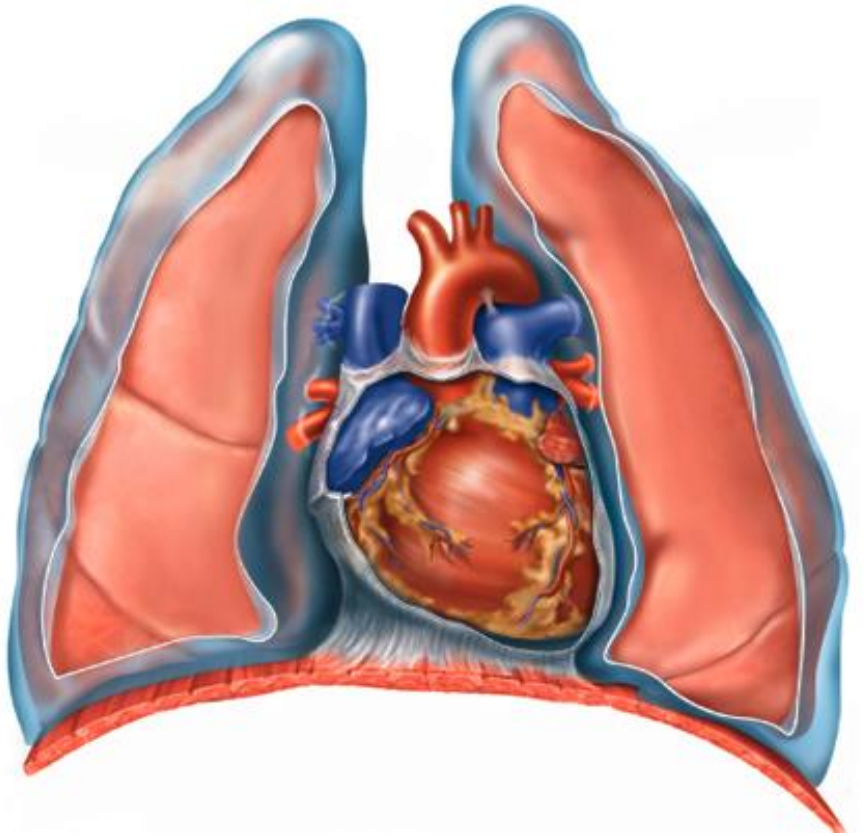
- Circulatory system
  - heart, blood vessels and blood
- Cardiovascular system
  - heart, arteries, veins and capillaries; 2 major divisions
- Pulmonary circuit - right side of heart
  - carries blood to lungs for gas exchange
- Systemic circuit - left side of heart
  - supplies blood to all organs of the body

Cardiovascular System Circuit

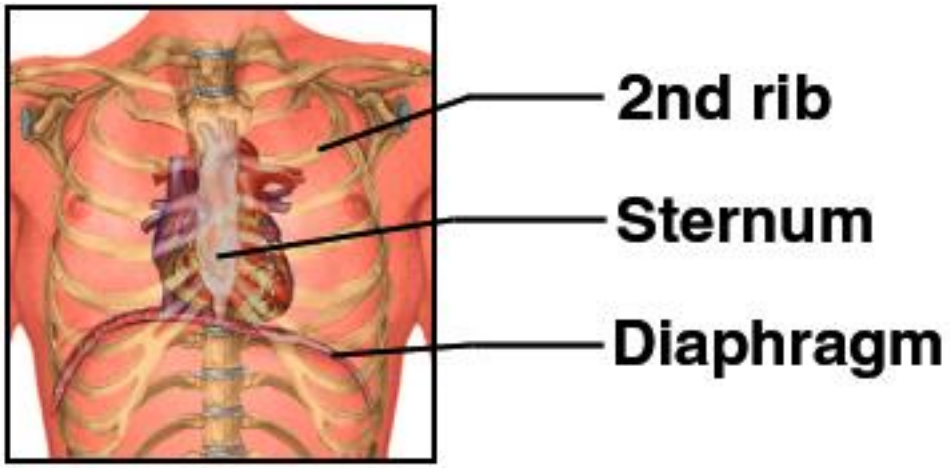


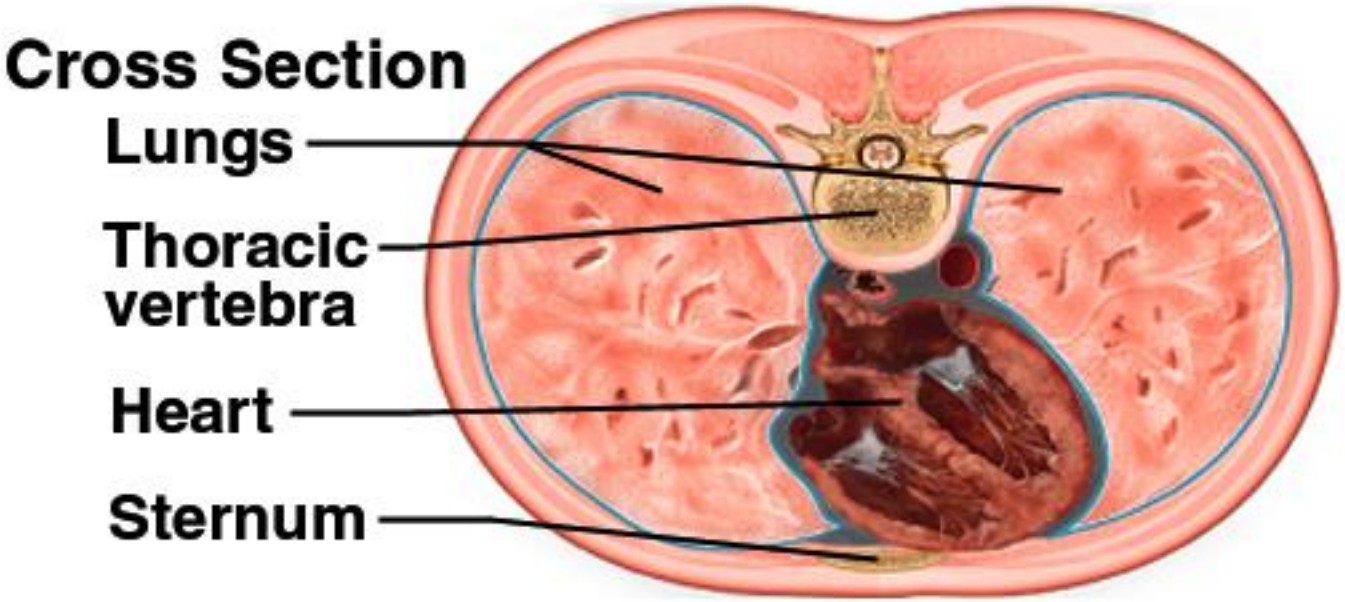
Size, Shape and Position

- Located in mediastinum, between lungs
- Base - broad superior portion of heart
- 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



Heart Position





Pericardium

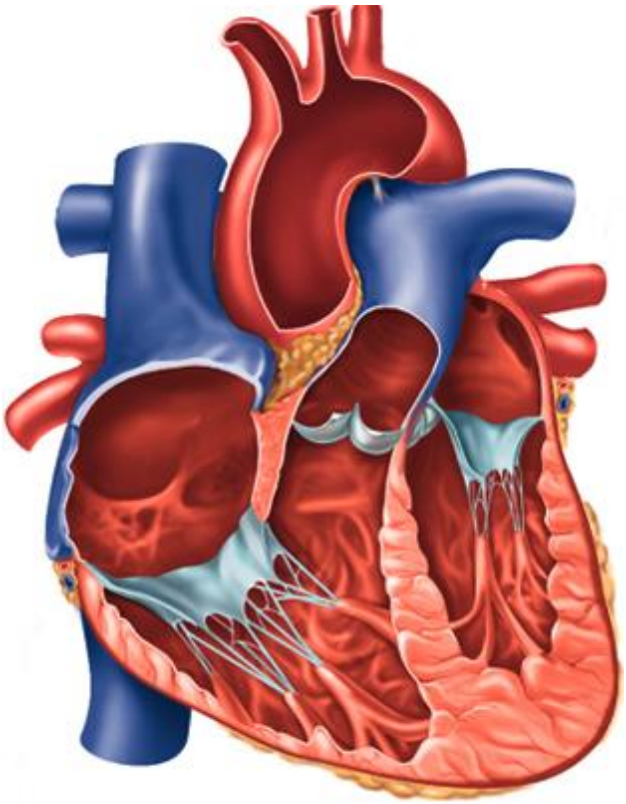
- Allows heart to beat without friction, room to expand and resists excessive expansion
- Parietal pericardium
  - outer, tough, fibrous layer of CT
  - inner, thin, smooth, moist serous layer
- Pericardial cavity
  - filled with pericardial fluid
- Visceral pericardium (a.k.a. epicardium of heart wall)
  - thin, smooth, moist serous layer covers heart surface

Heart Wall

- Epicardium (a.k.a. visceral pericardium)
  - serous membrane covers heart
- Myocardium
  - thick muscular layer
  - fibrous skeleton - network of collagenous and elastic fibers
    - provides structural support
    - attachment for cardiac muscle
    - nonconductor important in coordinating contractile activity
- Endocardium
  - smooth inner lining

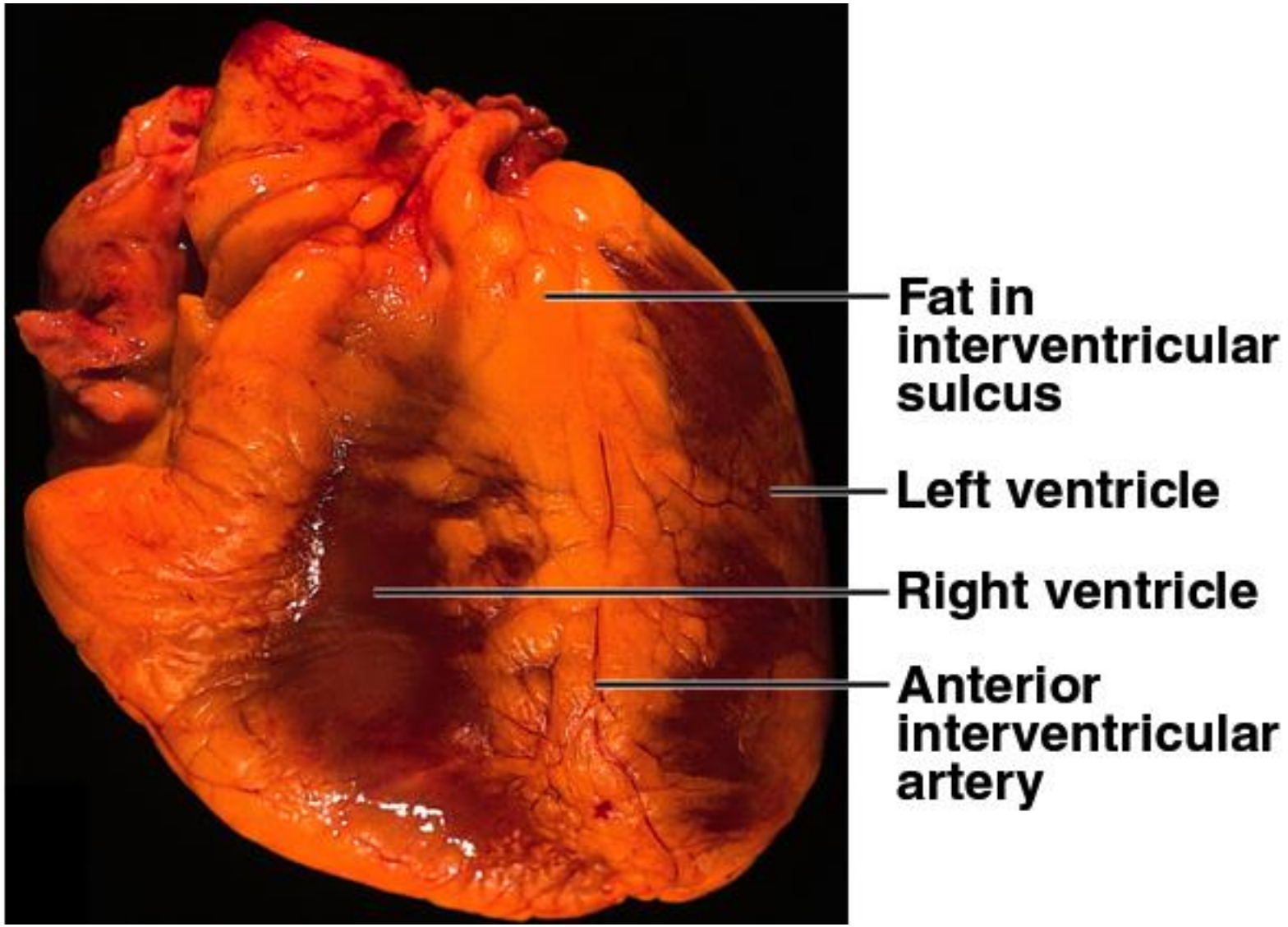
Heart Chambers

- 4 chambers
- Right and left atria
  - 2 superior, posterior chambers
  - receive blood returning to heart
- Right and left ventricles
  - 2 inferior chambers
  - pump blood into arteries

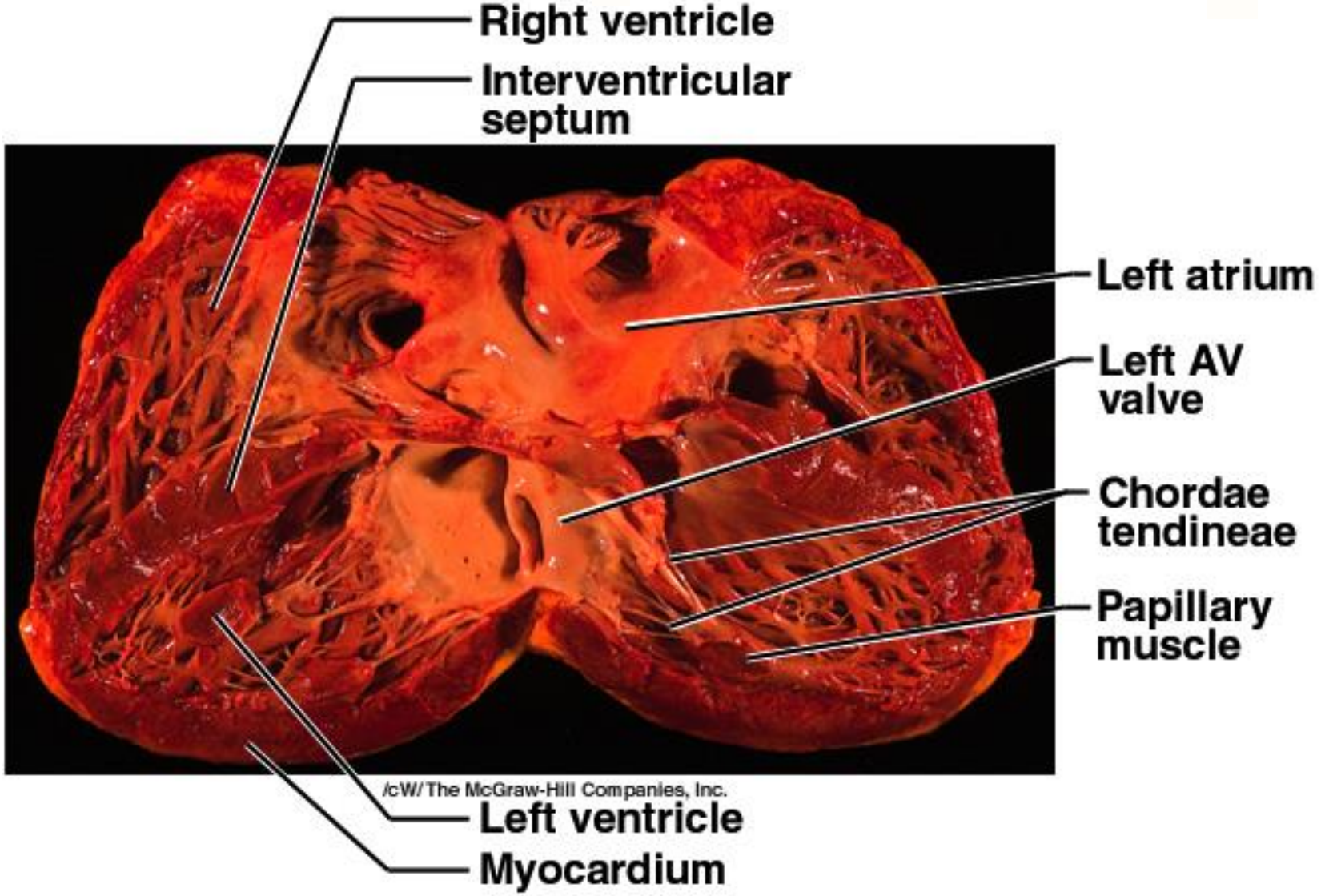


- Atrioventricular sulcus - separates atria, ventricles
- Anterior and posterior sulci - grooves separate ventricles *(next slide)*

Anterior Aspect



Heart Internal Anatomy

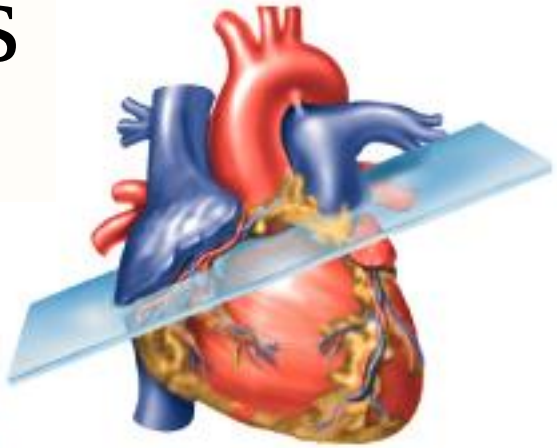


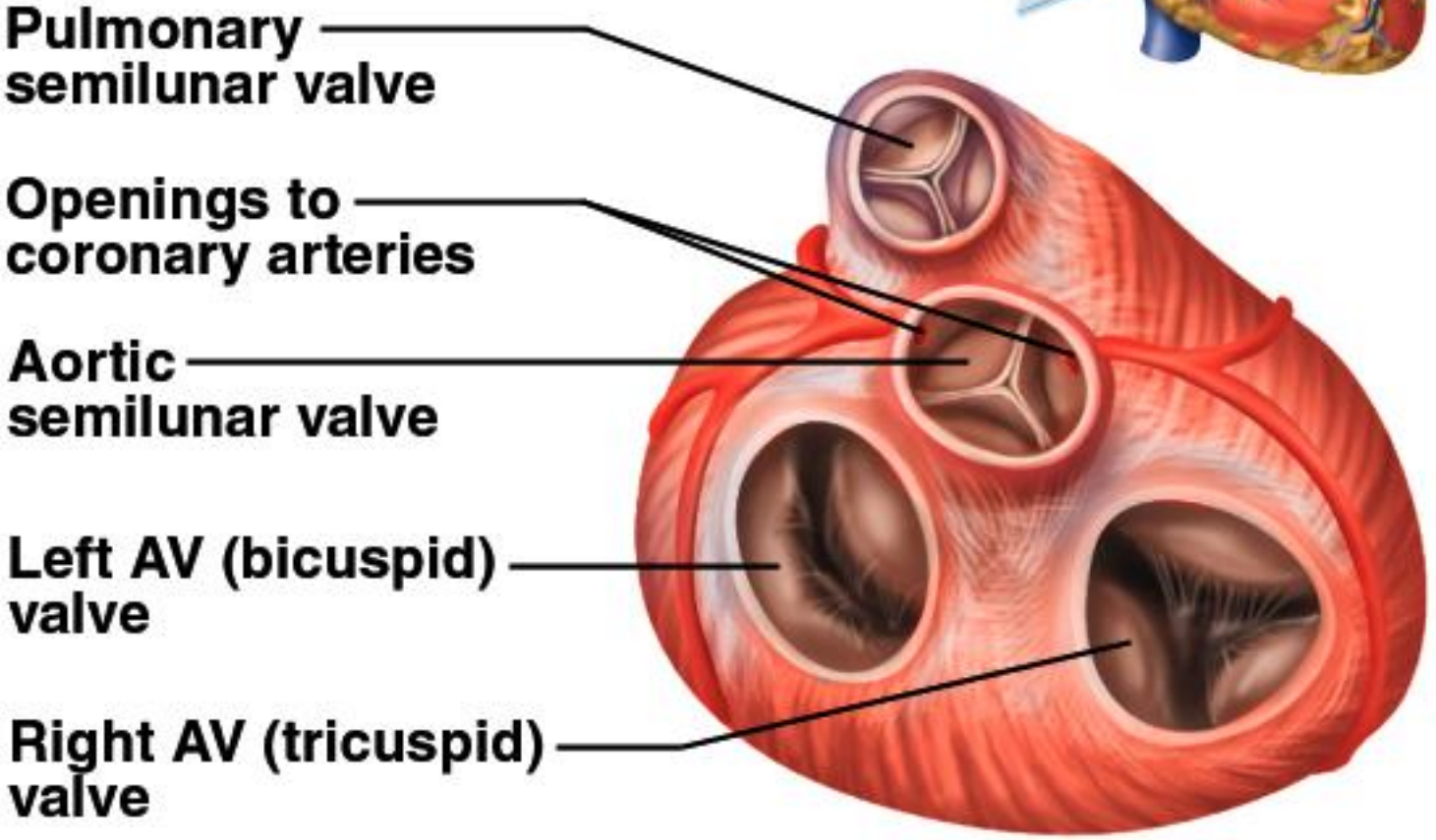
- Heart bisected in frontal plane, opened like a book

Heart Valves

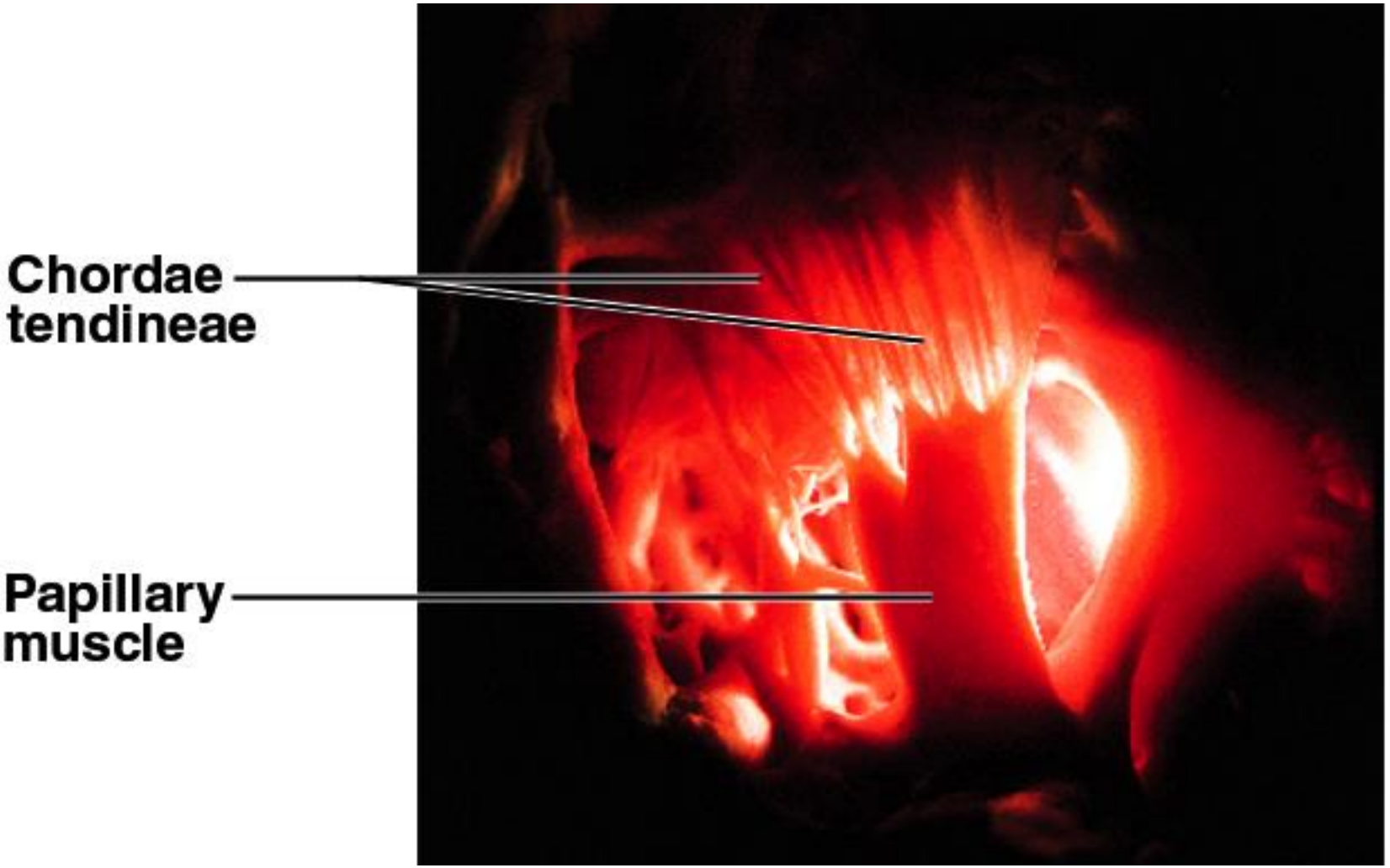
- Ensure one-way blood flow
- Atrioventricular (AV) valves
  - right AV valve has 3 cusps (tricuspid valve)
  - left AV valve has 2 cusps (mitral, bicuspid valve) lamb
  - chordae tendineae - cords connect AV valves to papillary muscles (on floor of ventricles)
- Semilunar valves - control flow into great arteries
  - pulmonary: from right ventricle into pulmonary trunk
  - aortic: from left ventricle into aorta

Heart Valves





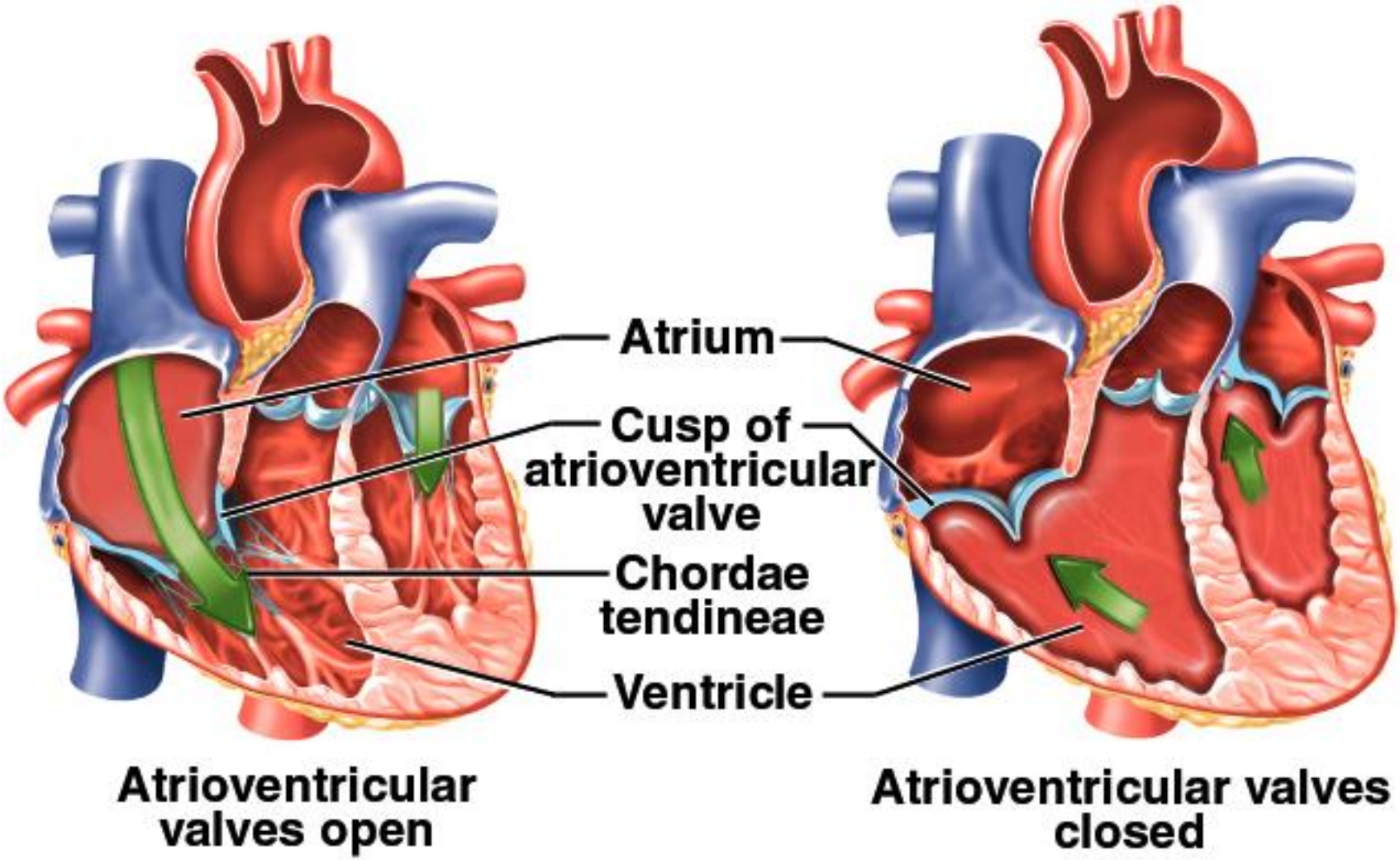
Heart Valves



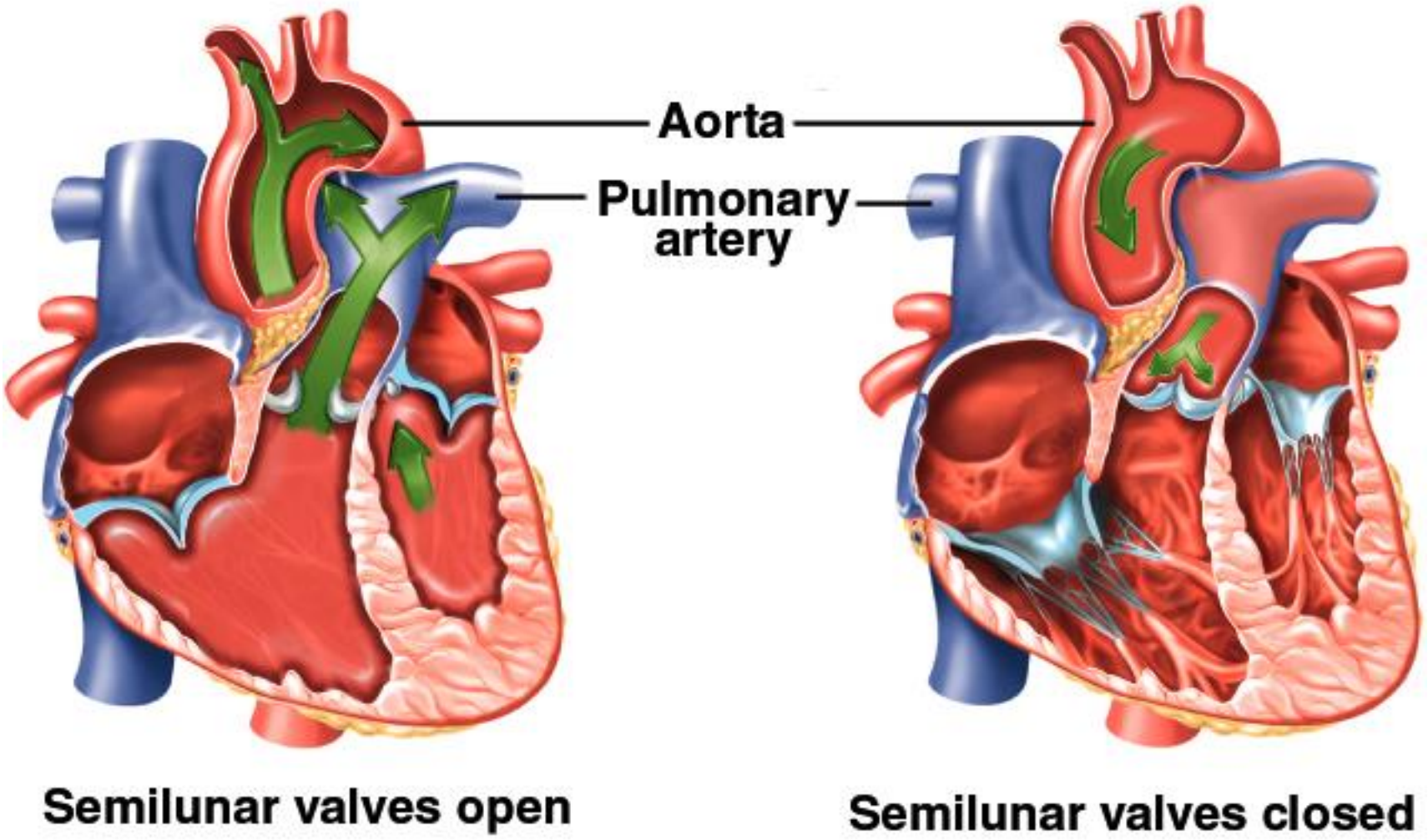
AV Valve Mechanics

- Ventricles relax, *pressure drops*, semilunar valves close, AV valves open, blood flows from atria to ventricles
- Ventricles contract, AV valves close (papillary m. contract and pull on chordae tendineae to prevent prolapse), *pressure rises*, semilunar valves open, blood flows into great vessels

Operation of Atrioventricular Valves



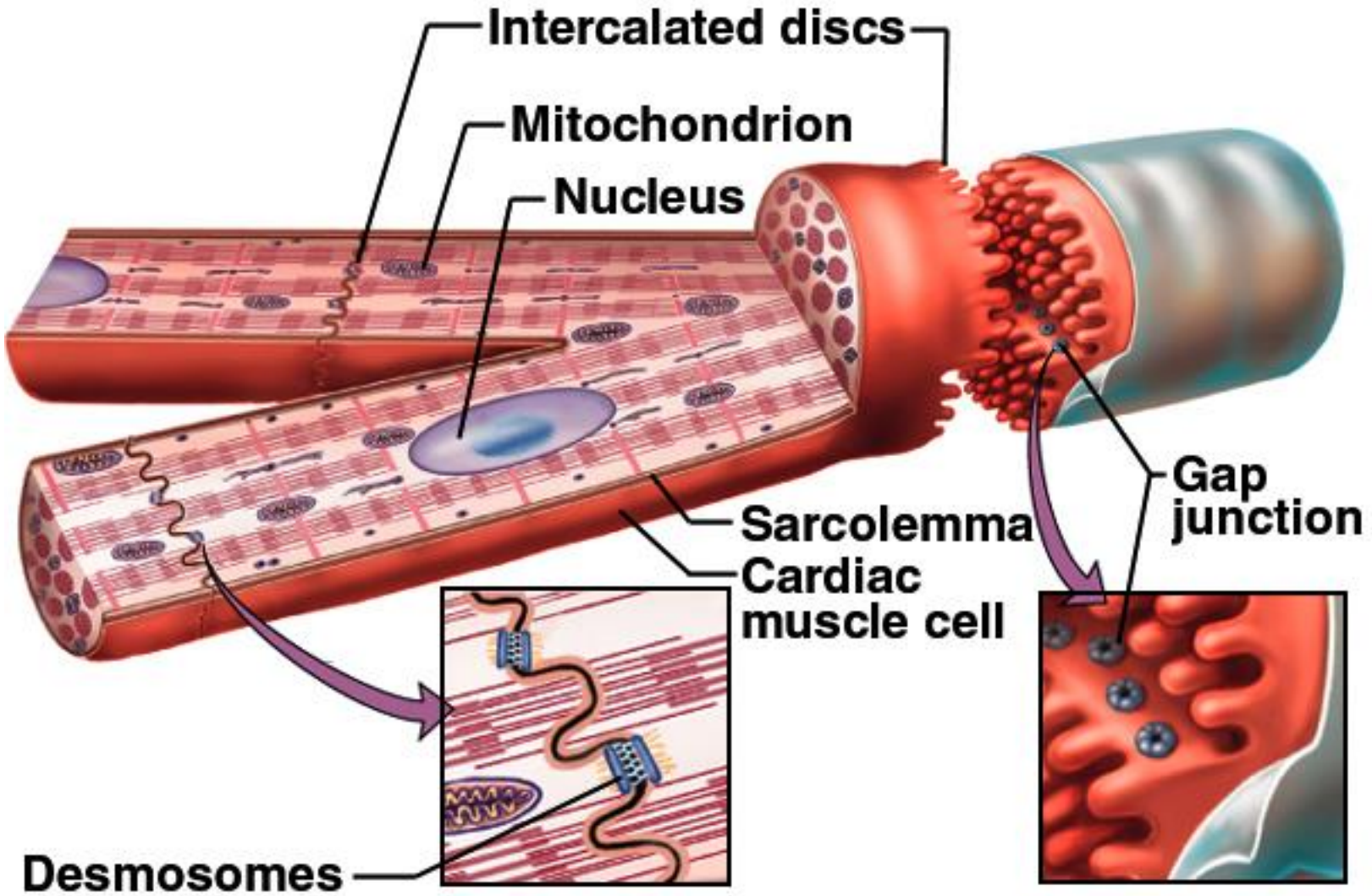
Operation of Semilunar Valves



Structure of Cardiac Muscle

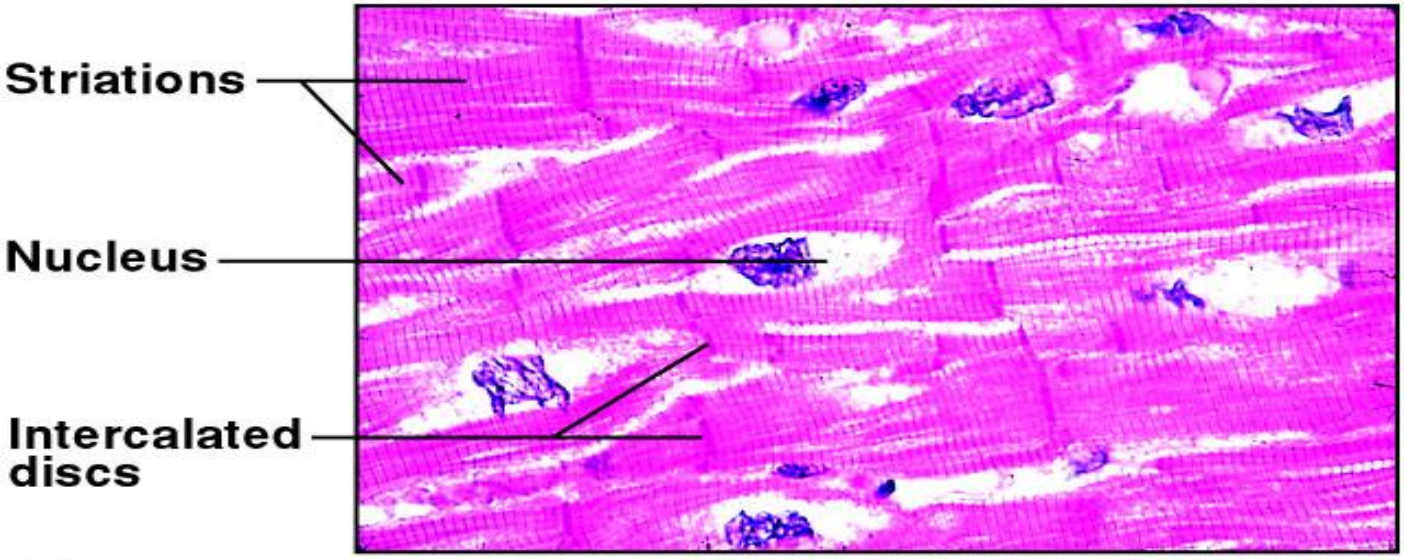
- Short, thick, branched cells, 50 to 100 μm long and 10 to 20 μm wide with one central nucleus
- ↓ Sarcoplasmic reticulum, large T tubules
  - must admit more Ca^{2+} from ECF during excitation
- Intercalated discs, join myocytes end to end
  - interdigitating folds - ↑ surface area
  - mechanical junctions tightly join myocytes
    - fascia adherens: actin anchored to plasma membrane
    - desmosomes
  - electrical junctions - gap junctions form channels allowing ions to flow directly into next cell

Structure of Cardiac Muscle Cell



Metabolism of Cardiac Muscle

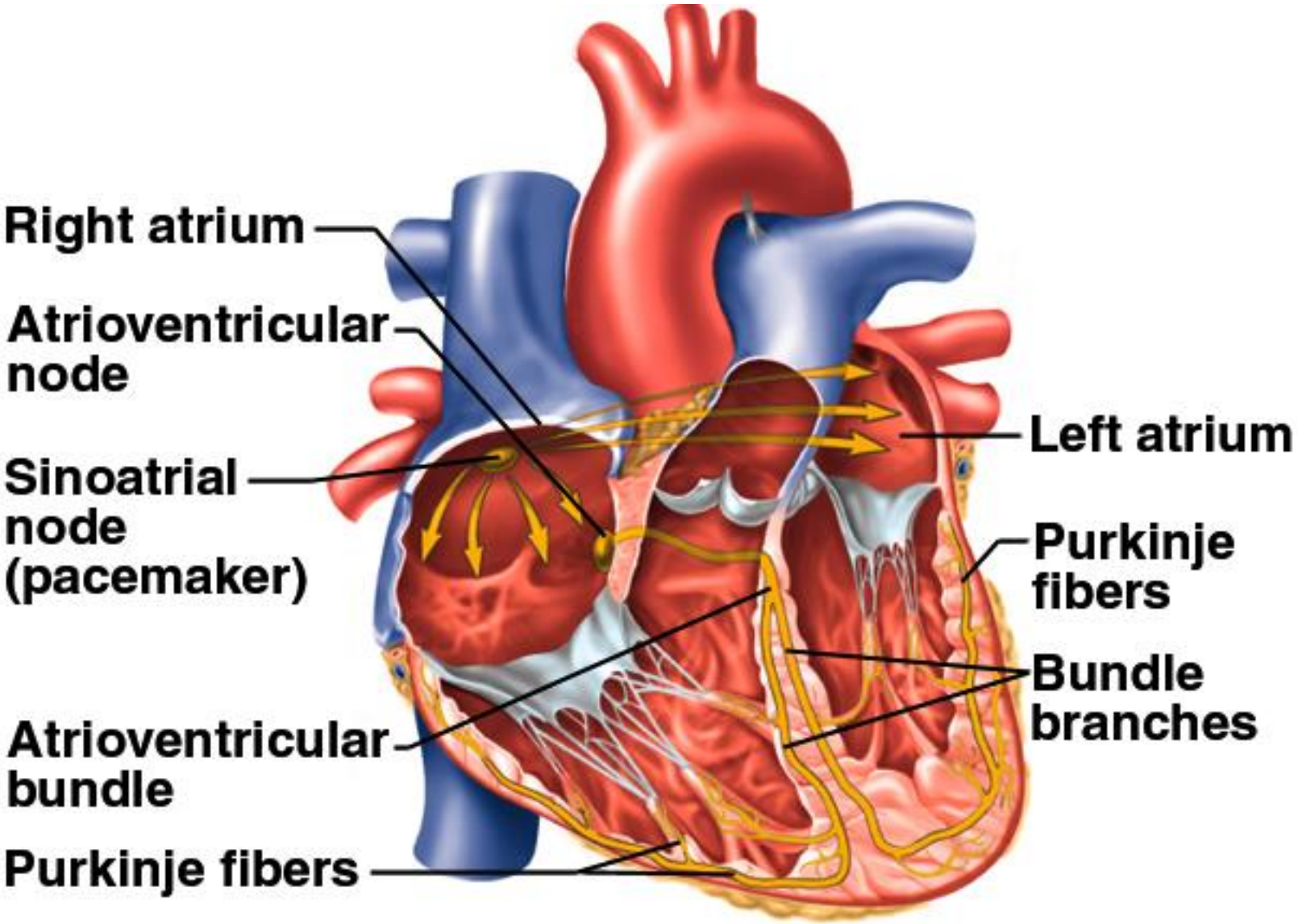
- Aerobic respiration
- Rich in myoglobin and glycogen
- Large mitochondria
- Organic fuels: fatty acids, glucose, ketones
- Fatigue resistant



Cardiac Conduction System

- Myogenic - heartbeat originates within heart
- Autorhythmic – regular, spontaneous depolarization
- Conduction system
  - SA node: pacemaker, initiates heartbeat, sets heart rate
  - *fibrous skeleton insulates atria from ventricles*
  - AV node: electrical gateway to ventricles
  - AV bundle: pathway for signals from AV node
  - Right and left bundle branches: divisions of AV bundle that enter interventricular septum and descend to apex
  - Purkinje fibers: upward from apex spread throughout ventricular myocardium

Cardiac Conduction System



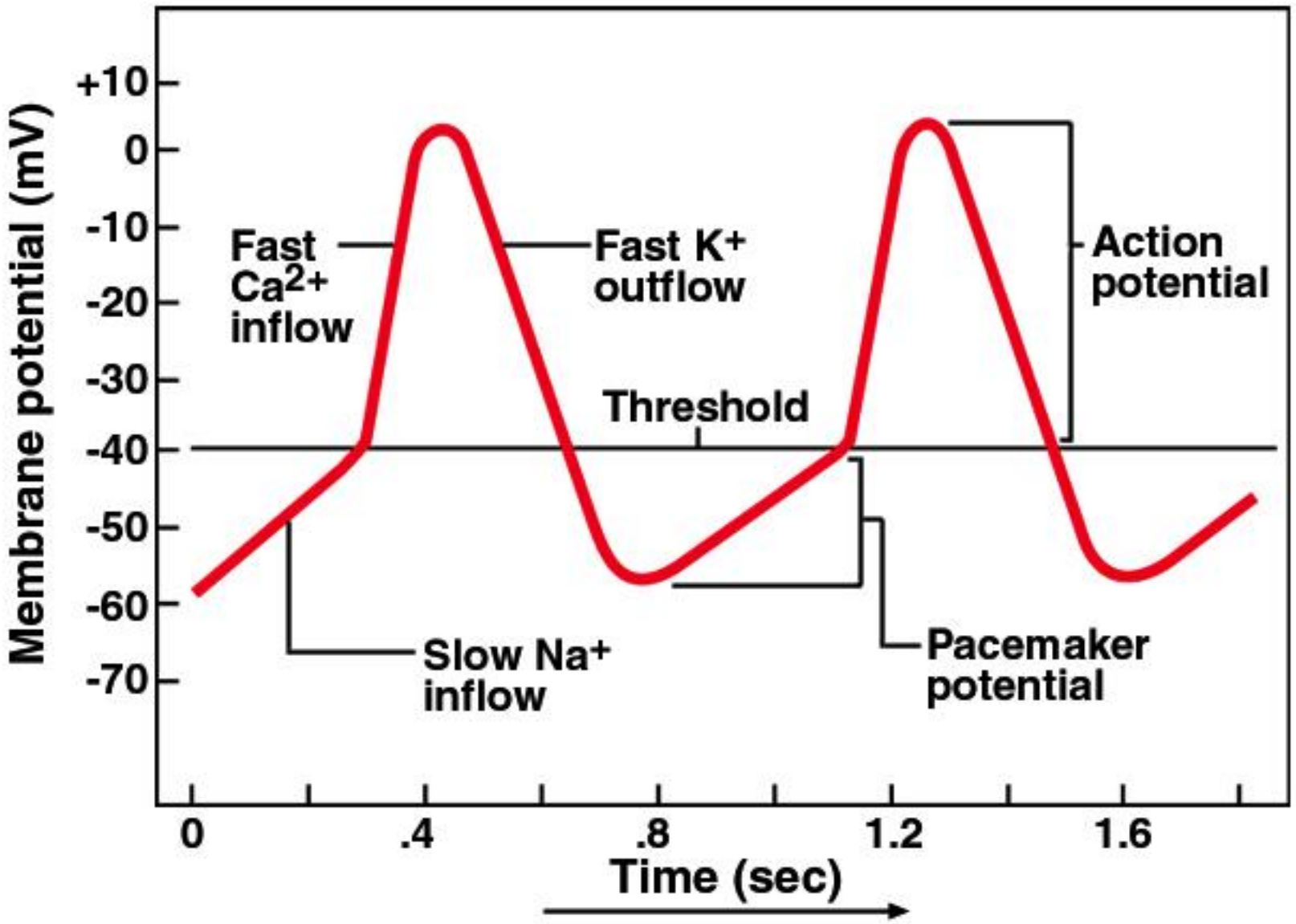
Cardiac Rhythm

- Systole = contraction; diastole = relaxation
- Sinus rhythm
  - set by SA node, adult at rest is 70 to 80 bpm
- Ectopic foci - region of spontaneous firing *(not SA)*
  - nodal rhythm - set by AV node, 40 to 50 bpm
  - intrinsic ventricular rhythm - 20 to 40 bpm
- Arrhythmia - abnormal cardiac rhythm
  - heart block: failure of conduction system
    - bundle branch block
    - total heart block (damage to AV node)

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
  - SA node at rest fires at 0.8 sec, about 75 bpm

SA Node Potentials



Impulse Conduction to Myocardium

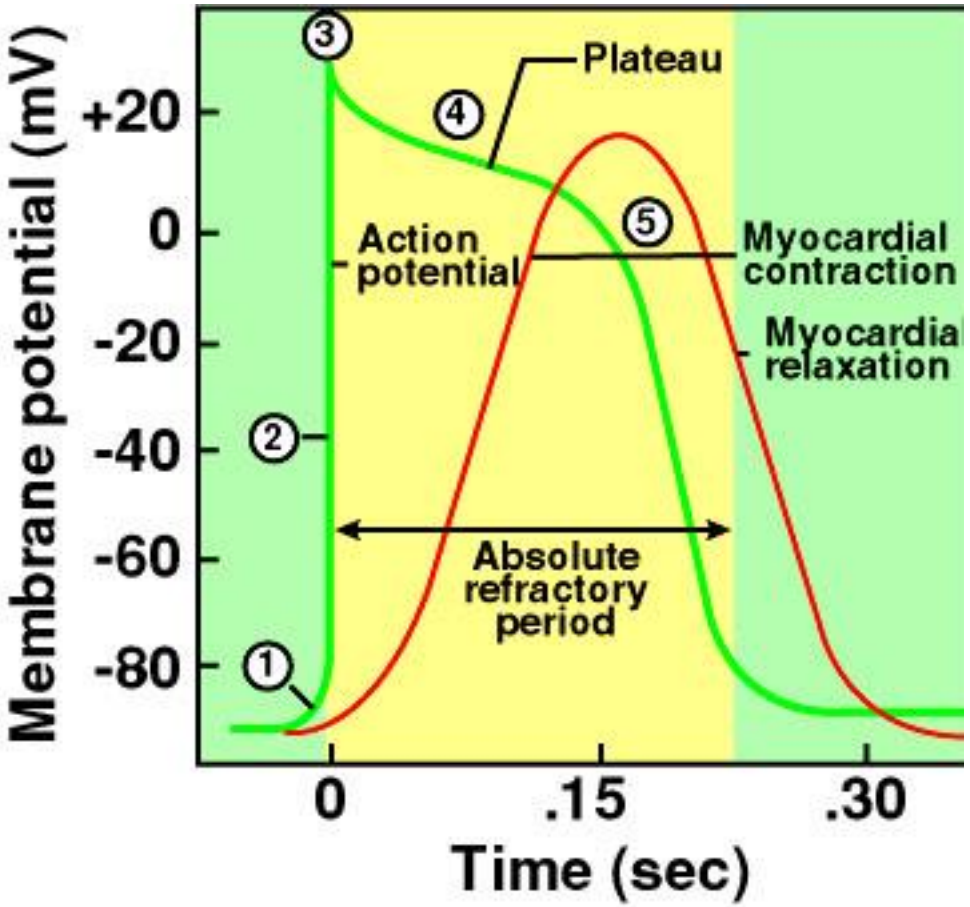
- SA node signal travels at *1 m/sec* through atria
- AV node slows signal to *0.05 m/sec*
  - thin myocytes with fewer gap junctions
  - delays signal 100 msec, allows ventricles to fill
- AV bundle and purkinje fibers
  - speeds signal along at *4 m/sec* to ventricles
- Papillary muscles - get signal first, contraction stabilizes AV valves
- Ventricular systole begins at apex, progresses up
  - spiral arrangement of myocytes twists ventricles slightly

Contraction of Myocardium

- Myocytes have stable resting potential of -90 mV
- Depolarization (very brief)
  - stimulus opens voltage regulated Na^+ gates, (Na^+ rushes in) membrane depolarizes rapidly
  - action potential peaks at +30 mV
  - Na^+ gates close quickly
- Plateau - 200 to 250 msec, sustains contraction
  - slow Ca^{+2} channels open, Ca^{+2} binds to fast Ca^{+2} channels on SR, releases $\uparrow Ca^{+2}$ into cytosol: contraction
- Repolarization - Ca^{+2} channels close, K^+ channels open, rapid K^+ out returns to resting potential

Myocardial Contraction & Action Potential

1) Na^+ gates open
2) Rapid depolarization
3) Na^+ gates close
4) Ca^{+2} channels open
5) Ca^{+2} channels close
   K^+ channels open



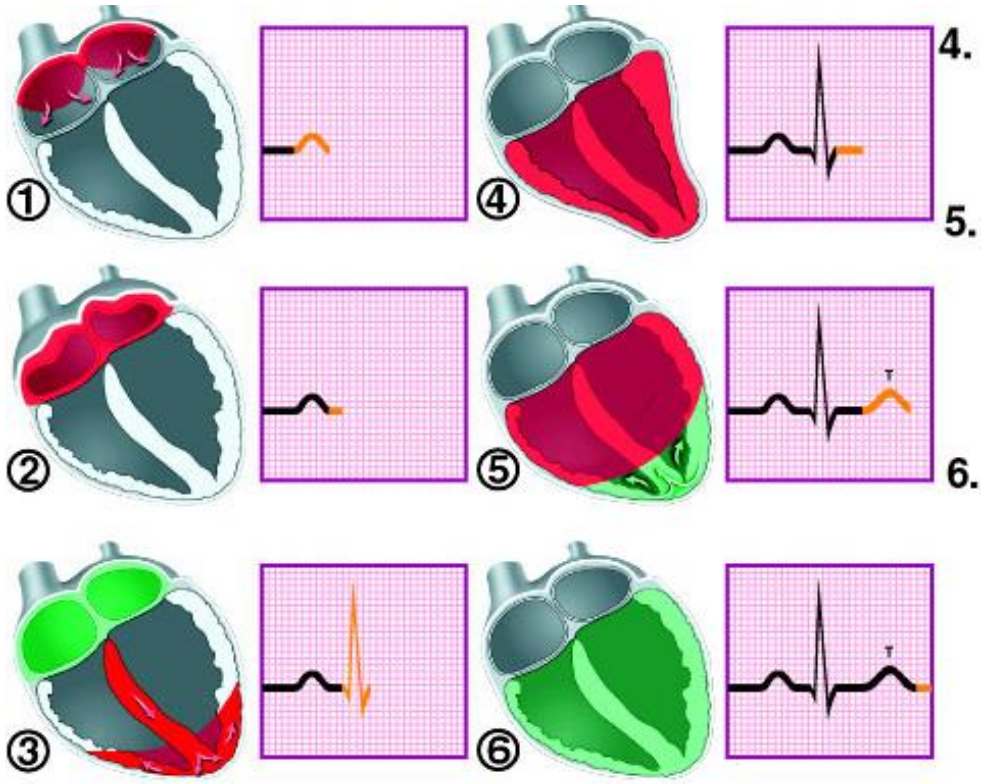
Electrical Activity of Myocardium

1) **atrial** depolarization begins

2) atrial depolarization complete (atria contracted)

3) **ventricles** begin to depolarize at apex; atria repolarize (atria relaxed)

4) ventricular depolarization complete (ventricles contracted)

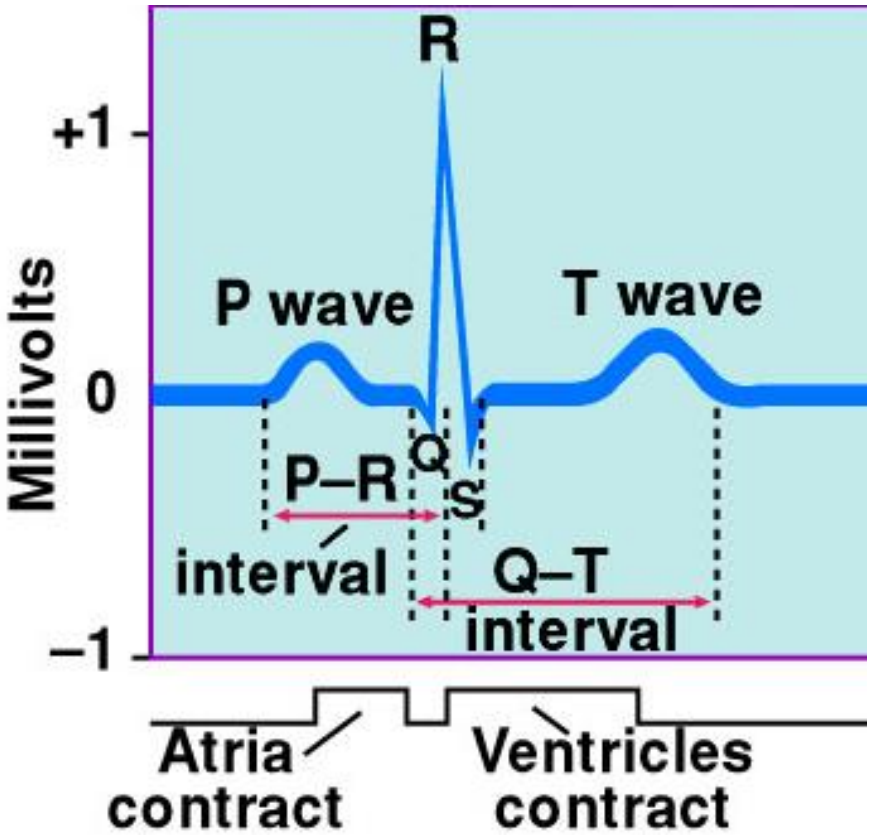


5) ventricles begin to repolarize at apex

6) ventricular repolarization complete (ventricles relaxed)

Electrocardiogram (ECG)

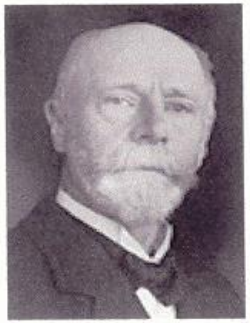
- Composite of all action potentials of nodal and myocardial cells detected, amplified and recorded by electrodes on arms, legs and chest



The EKG is not only the oldest but, in fact, over 100 years after its introduction, continues as the most commonly used cardiovascular laboratory procedure.

The Nobel Prize in Physiology or Medicine 1924

"for his discovery of the mechanism of the electrocardiogram"



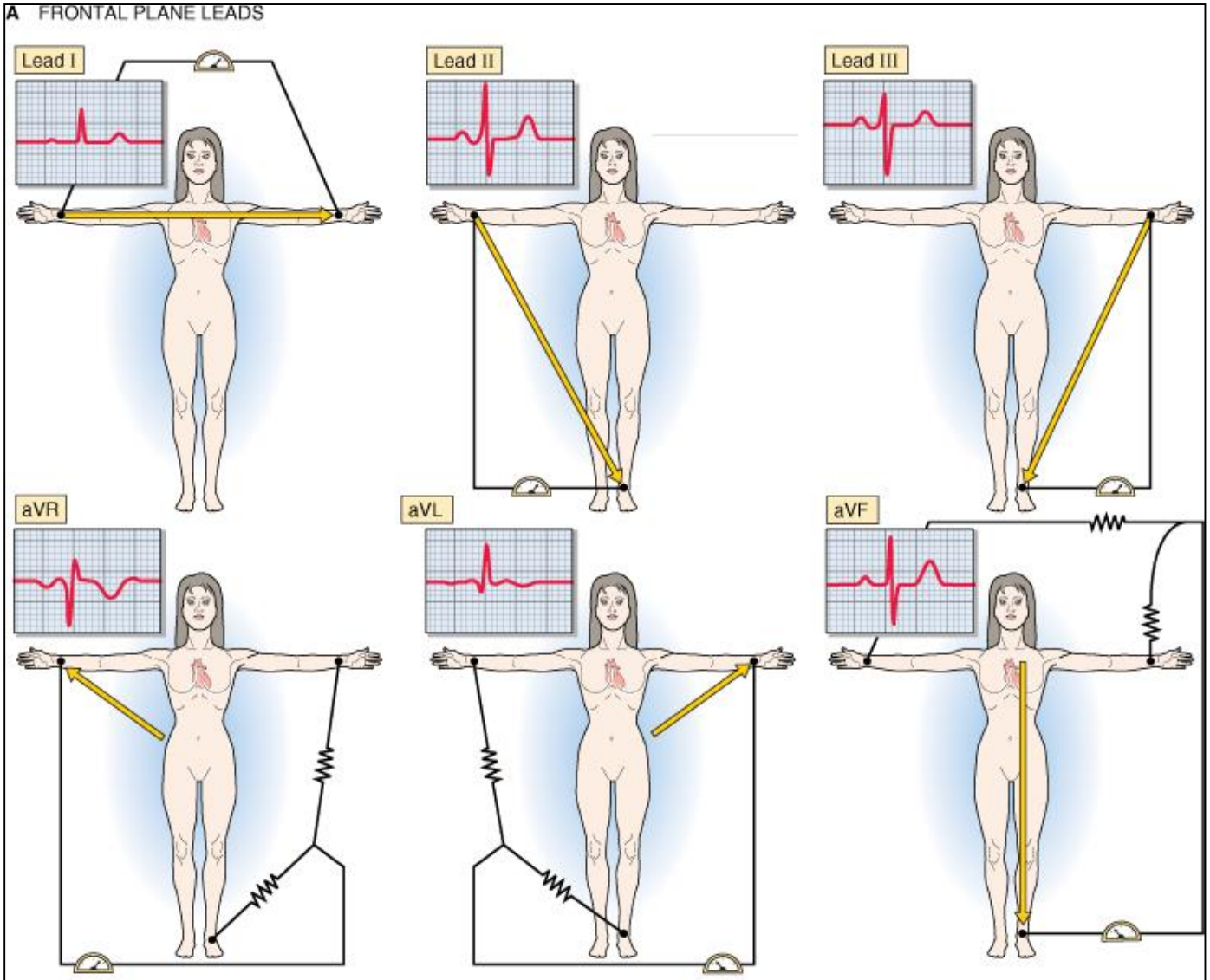
Willem Einthoven

the Netherlands

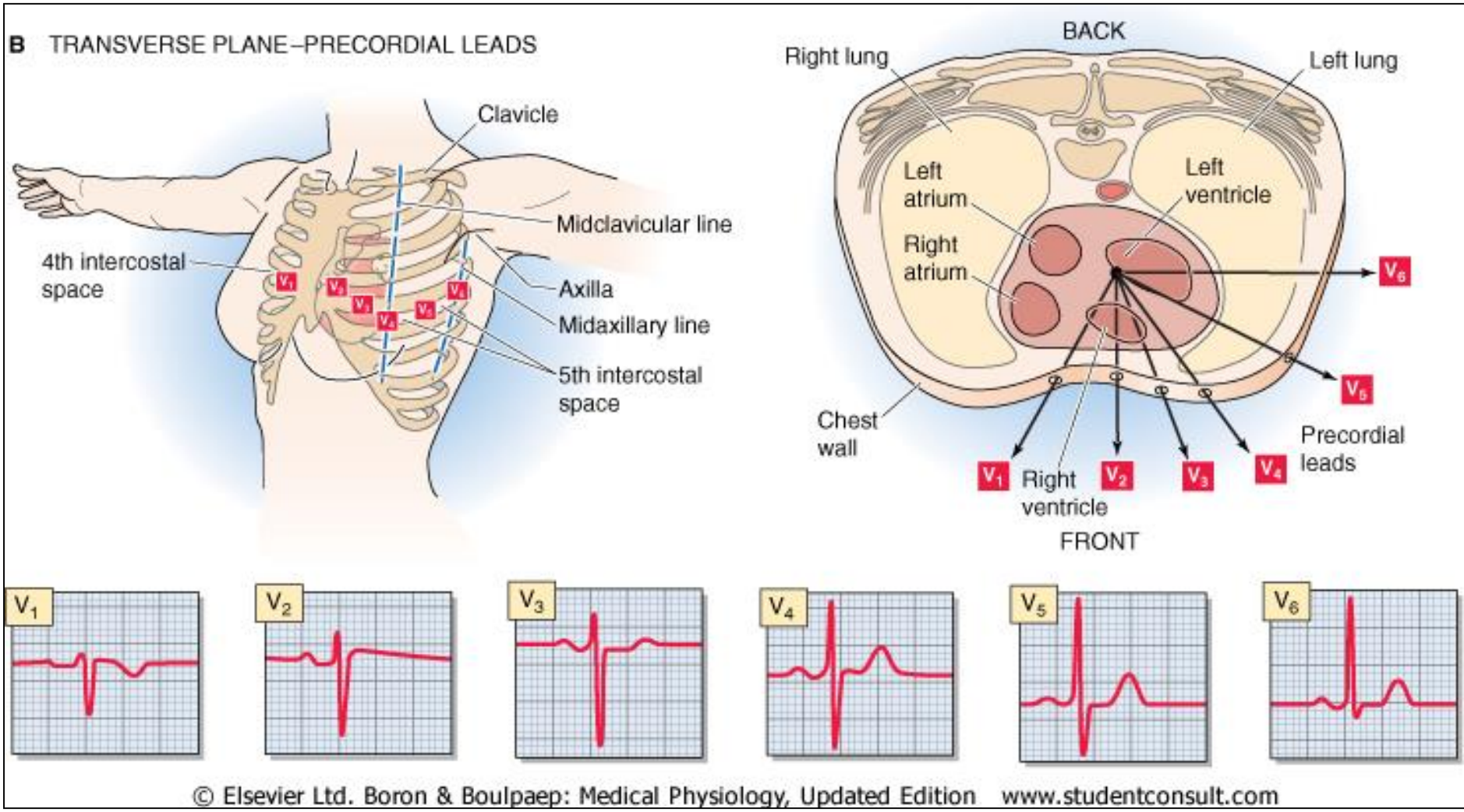
Leiden University
Leiden, the Netherlands

b.1860
(in Semarang, Java, then Dutch East Indies)
d.1927

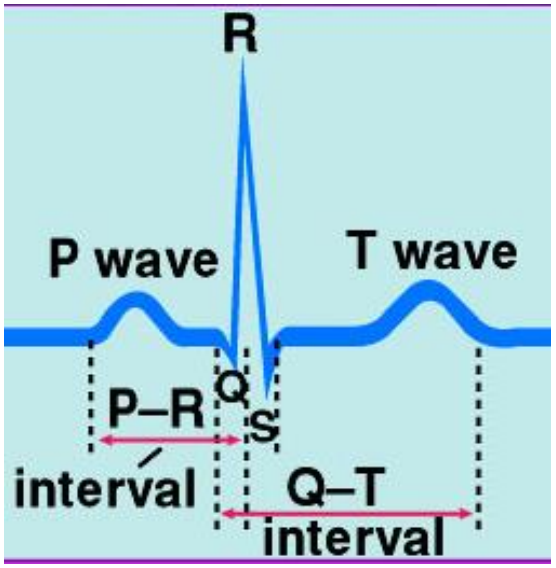
Summary of Limb Leads



The Precordial (Chest) Leads

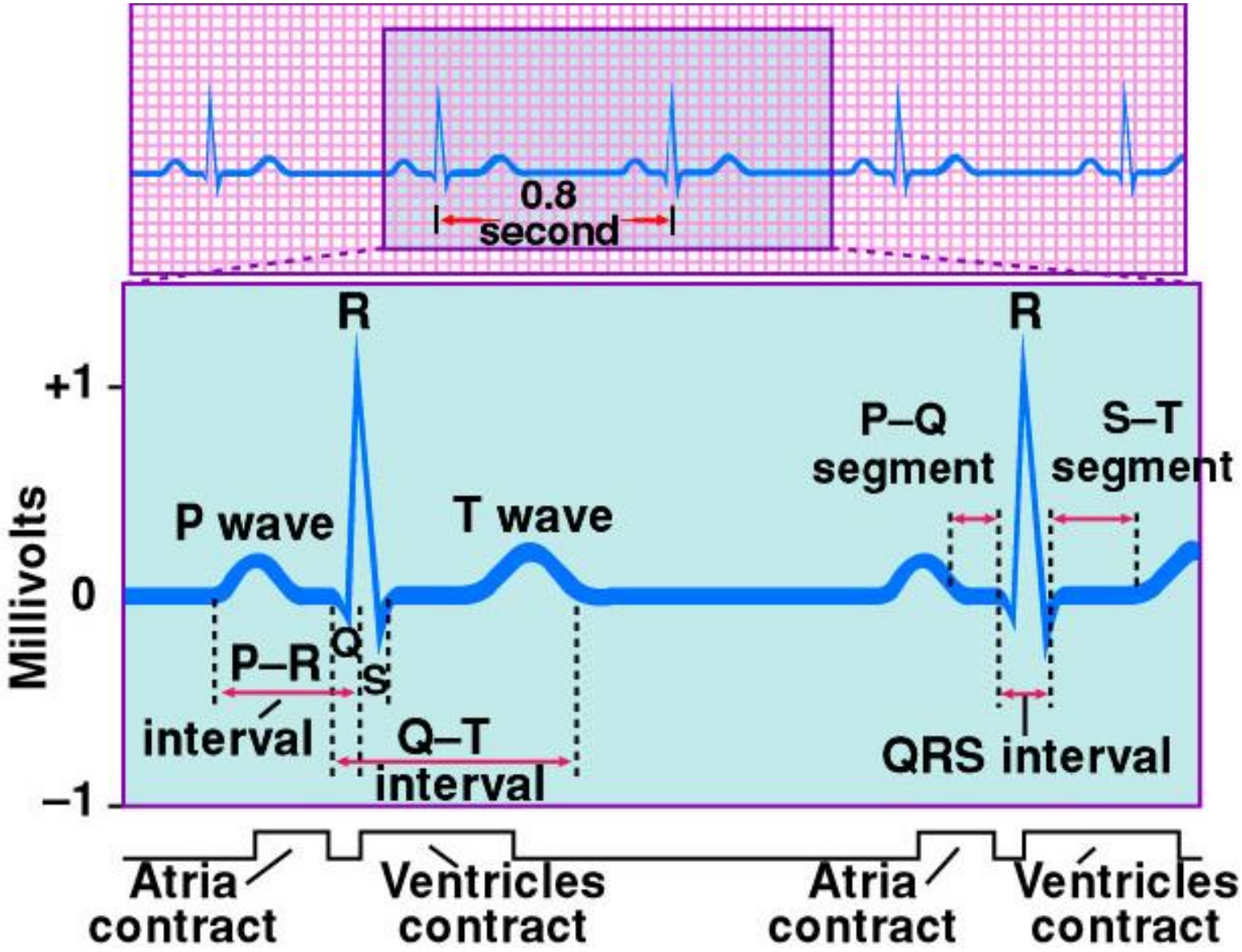


ECG

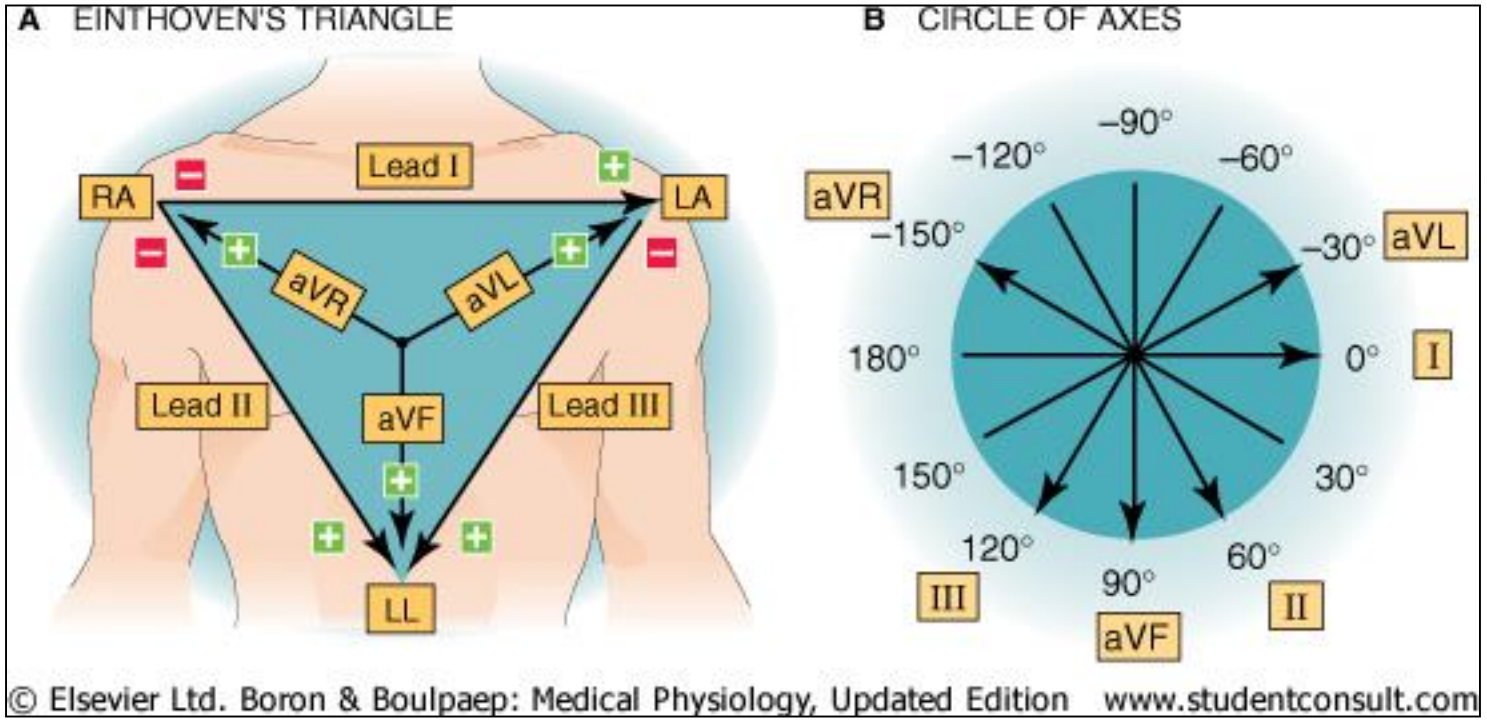


- P wave
  - SA node fires, atrial depolarization
  - atrial systole
- QRS complex
  - AV node fires, ventricular depolarization
  - ventricular systole
  - (atrial repolarization and diastole - signal obscured)
- T wave
  - ventricular repolarization

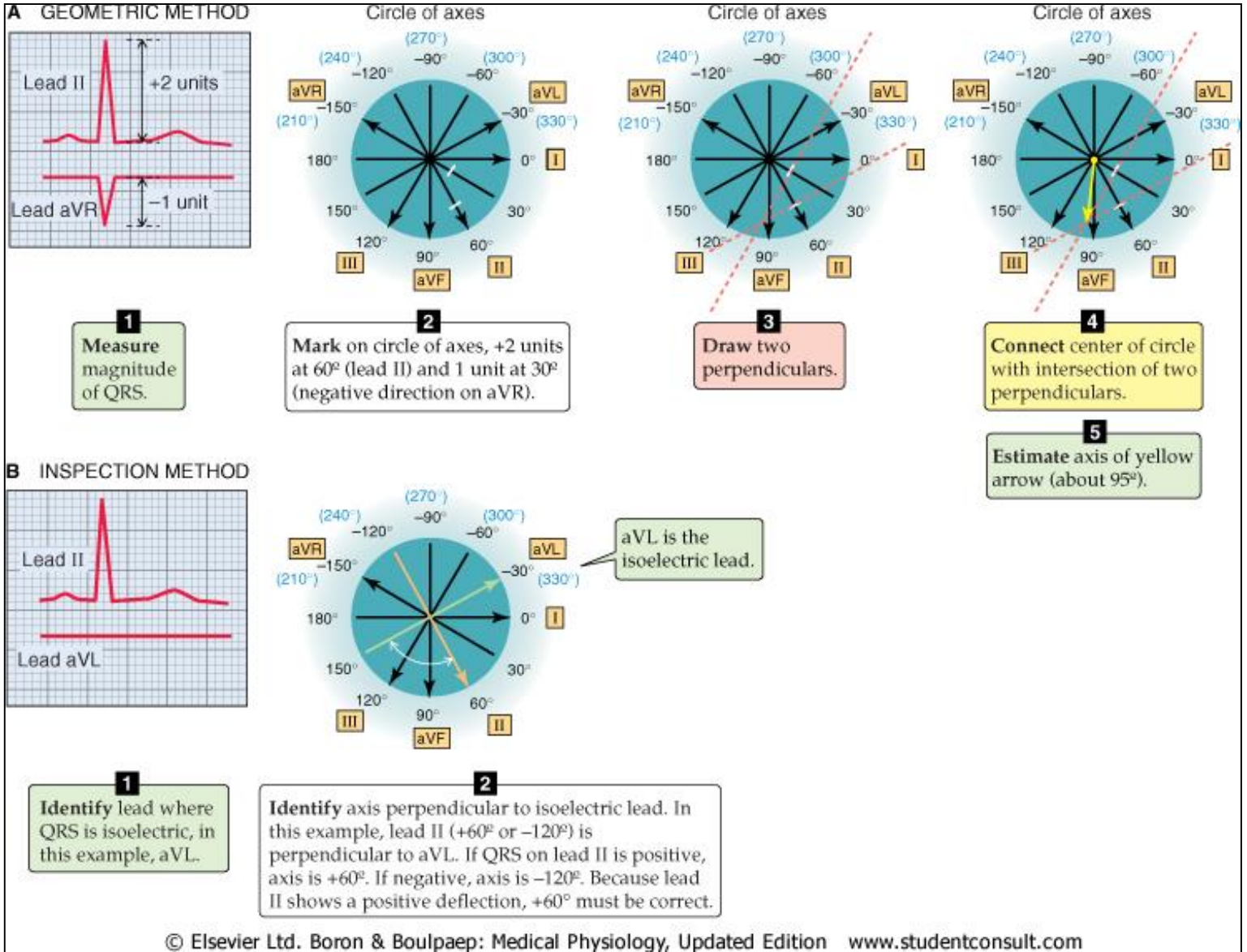
Normal Electrocardiogram (ECG)



Vector Analysis, cont'd



Vector Algebraic Analysis

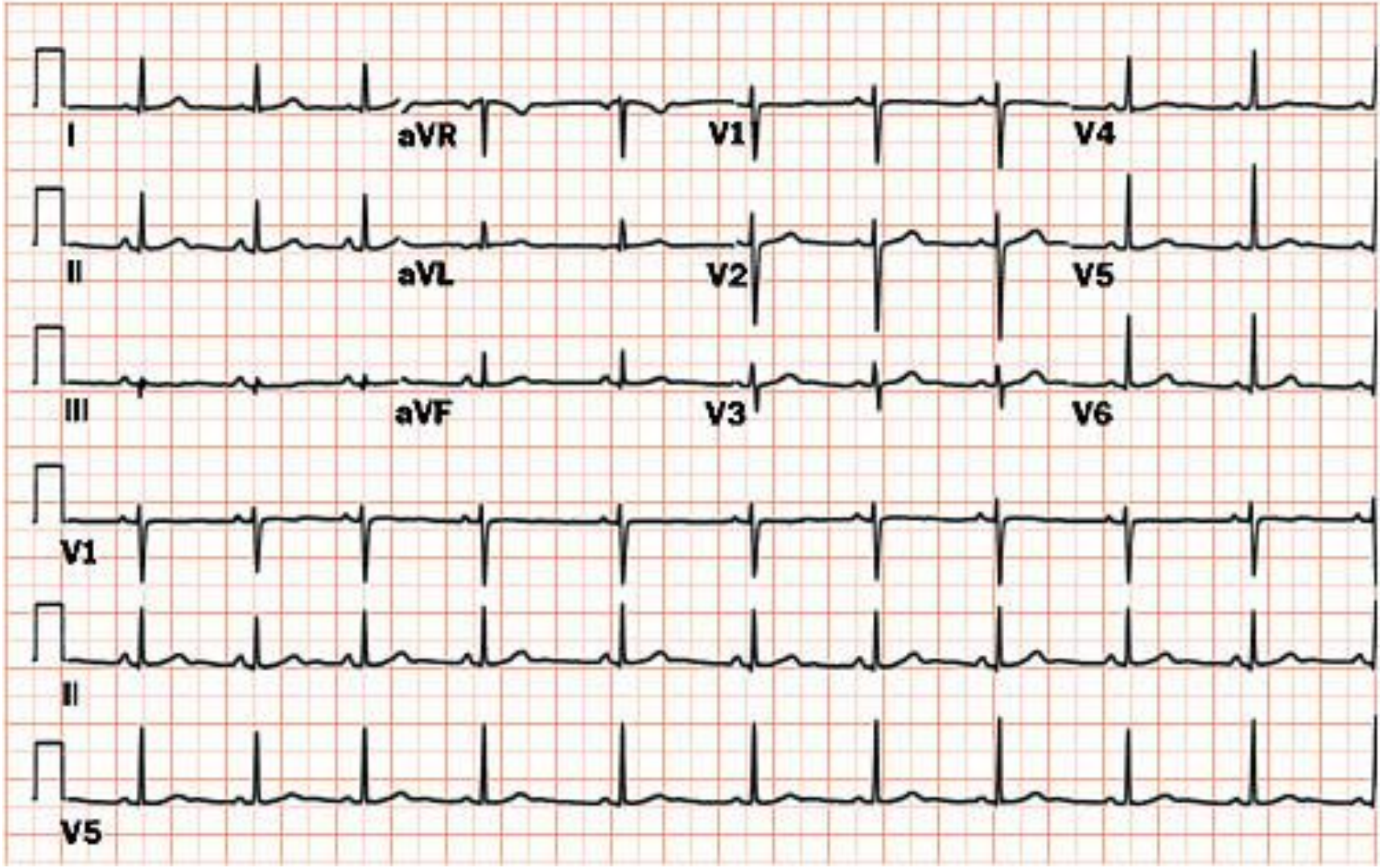


Diagnostic Value of ECG

- Invaluable for diagnosing abnormalities in conduction pathways, MI, heart enlargement and electrolyte and hormone imbalances

Sinus rhythm

This image taken from the *Textbook of Cardiovascular Medicine*, 2nd Ed.



ECG analysis performance

1. **Cardiac rhythm and conductivity analysis.**

- Heart contraction regularity asessment.

  Interval R-R is usually measured between R denses apexes.

- Heart regular **sinus** rhythm is diagnosed when measured R-R intervals duration is equal or difference is ±10% from R-R average duration.

2. Cardiac contractions number (heart contraction rate – HCR) counting. It is assessed at correct rhythm by formule:

- **HCR=60: (R-R)**

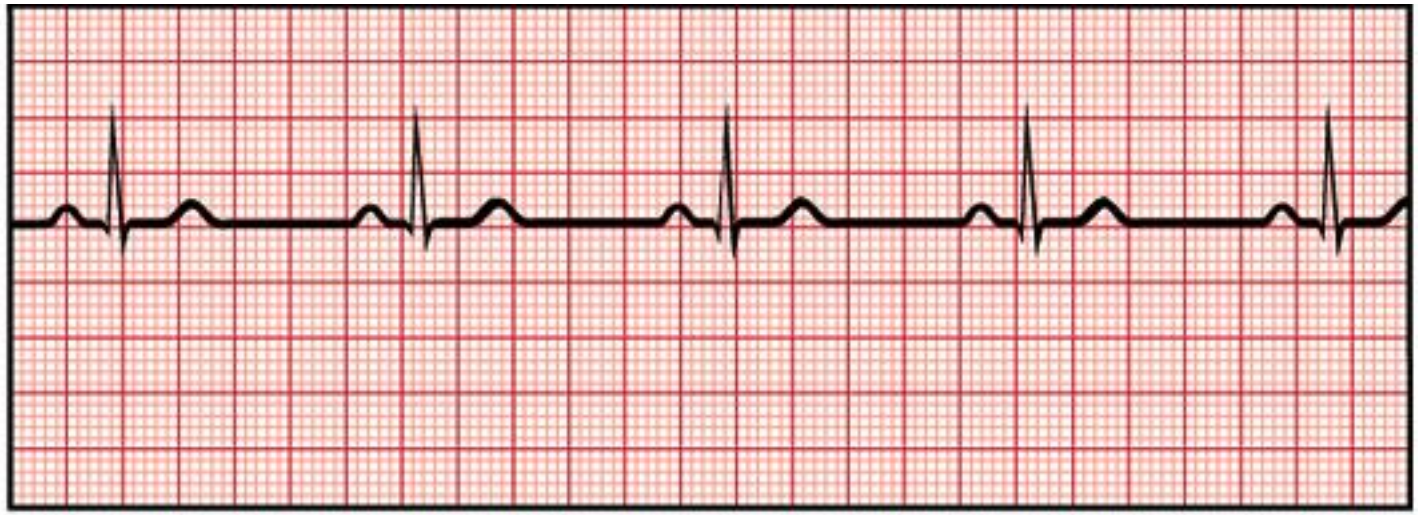
where:

- 60 is seconds number in 1 minute;
- R-R – interval duration expressed in seconds.

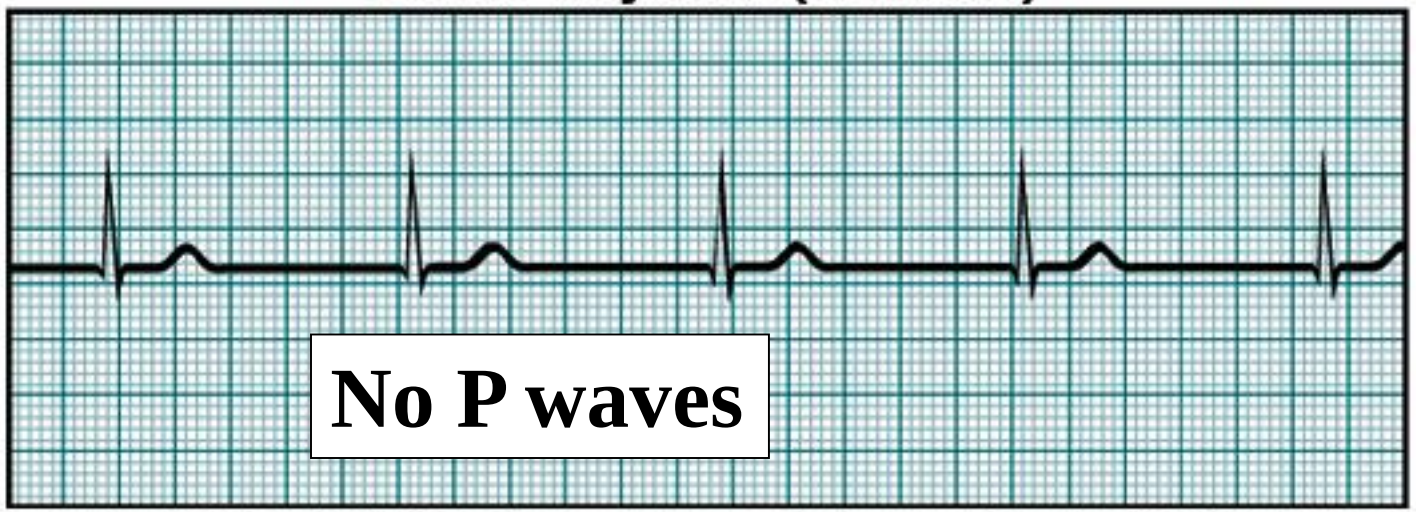
3. Excitement source determining.

- Sinus rhythm is characterized by:
- positive P-denses in II standard lead and these P-denses come before every complex QRS;
- all P-denses constant equal shape in one and the same lead.

ECGs, Normal & Abnormal



Sinus rhythm (normal)

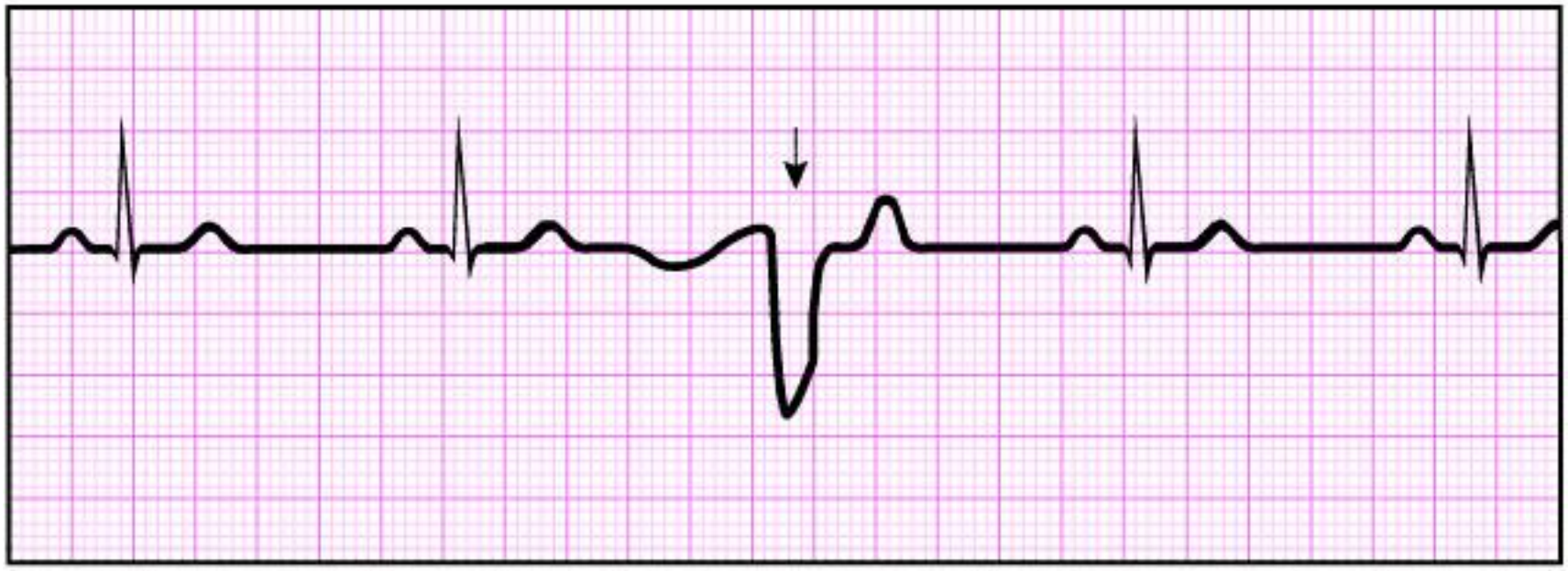


Nodal rhythm – no SA node activity

Excitement source determining.

- ***Atrial rhythms*** (from atria inferior parts) are characterized by negative denses P
- in II and III and non-changed QRS complexes following after them.
- ***Rhythms from AV-binding*** are characterized by:
- P-dense absence – this dense coincides to usual non-changed QRS-dense or
  - negative P-denses located after usual non-changed QRS complexes.
- ***Ventricular (idio-ventricular) rhythm*****is** characterized by:
  - slow ventricular rhythm (less than 40 beatings per 1 min);
  - presence of dilated and deformed QRS complexes;
  - absence of usual connection between QRS complex and

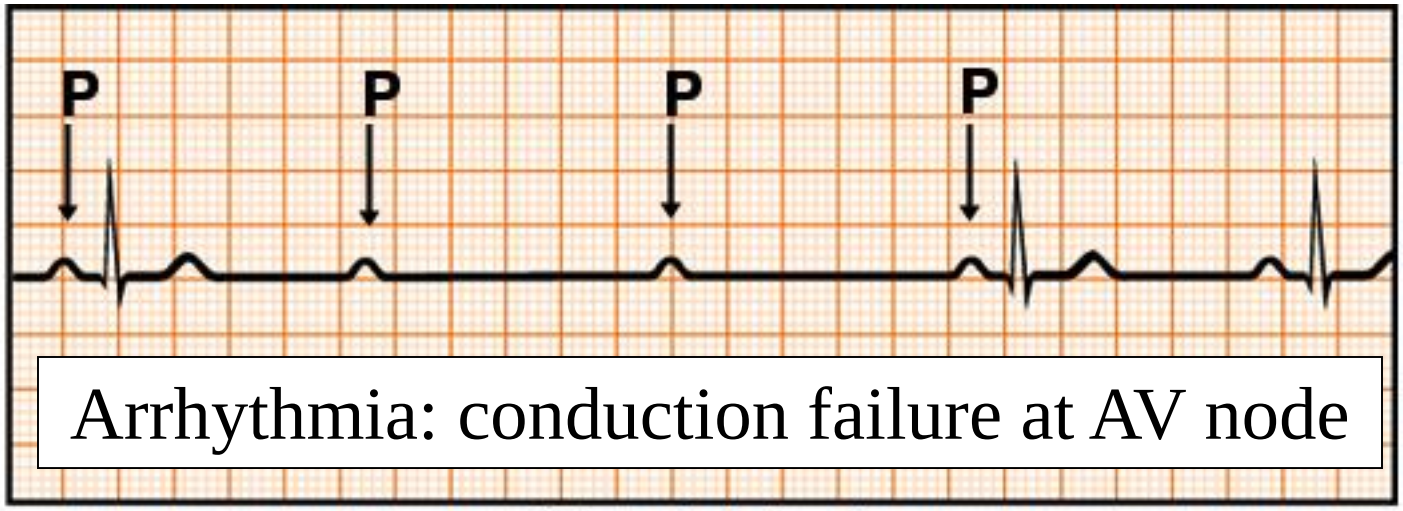
ECGs, Abnormal



Premature ventricular contraction

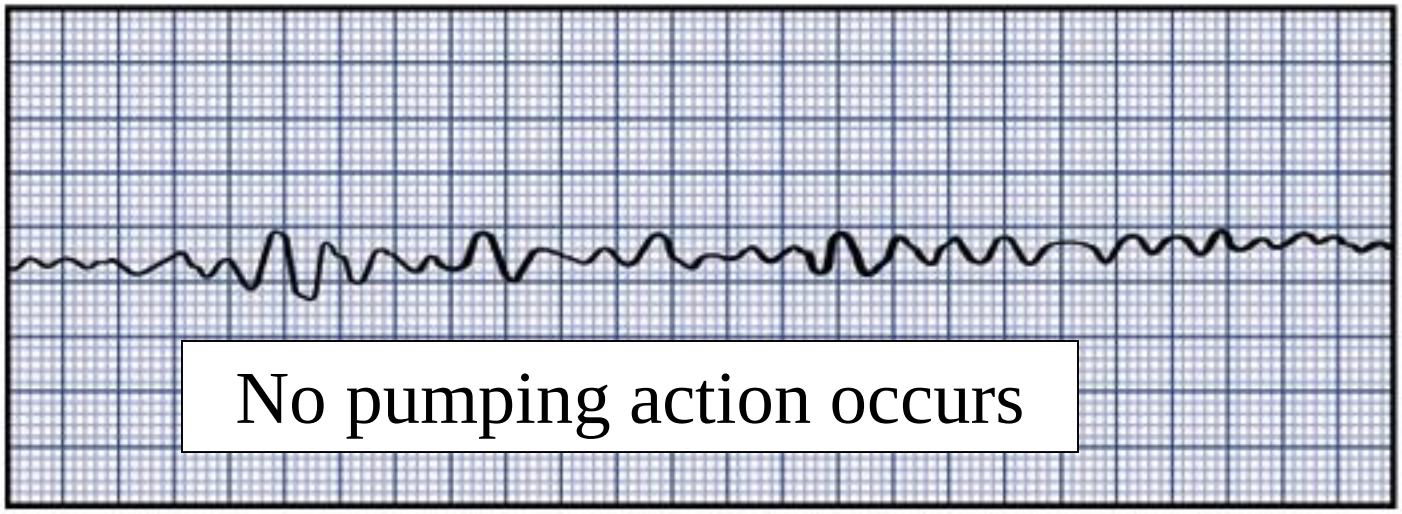
Extrasystole : note the inverted QRS complex, misshapen QRS and T and absence of a P wave preceding this contraction.

ECGs, Abnormal



Arrhythmia: conduction failure at AV node

Heart block



No pumping action occurs

Fibrillation

4.Determining heart turnings round

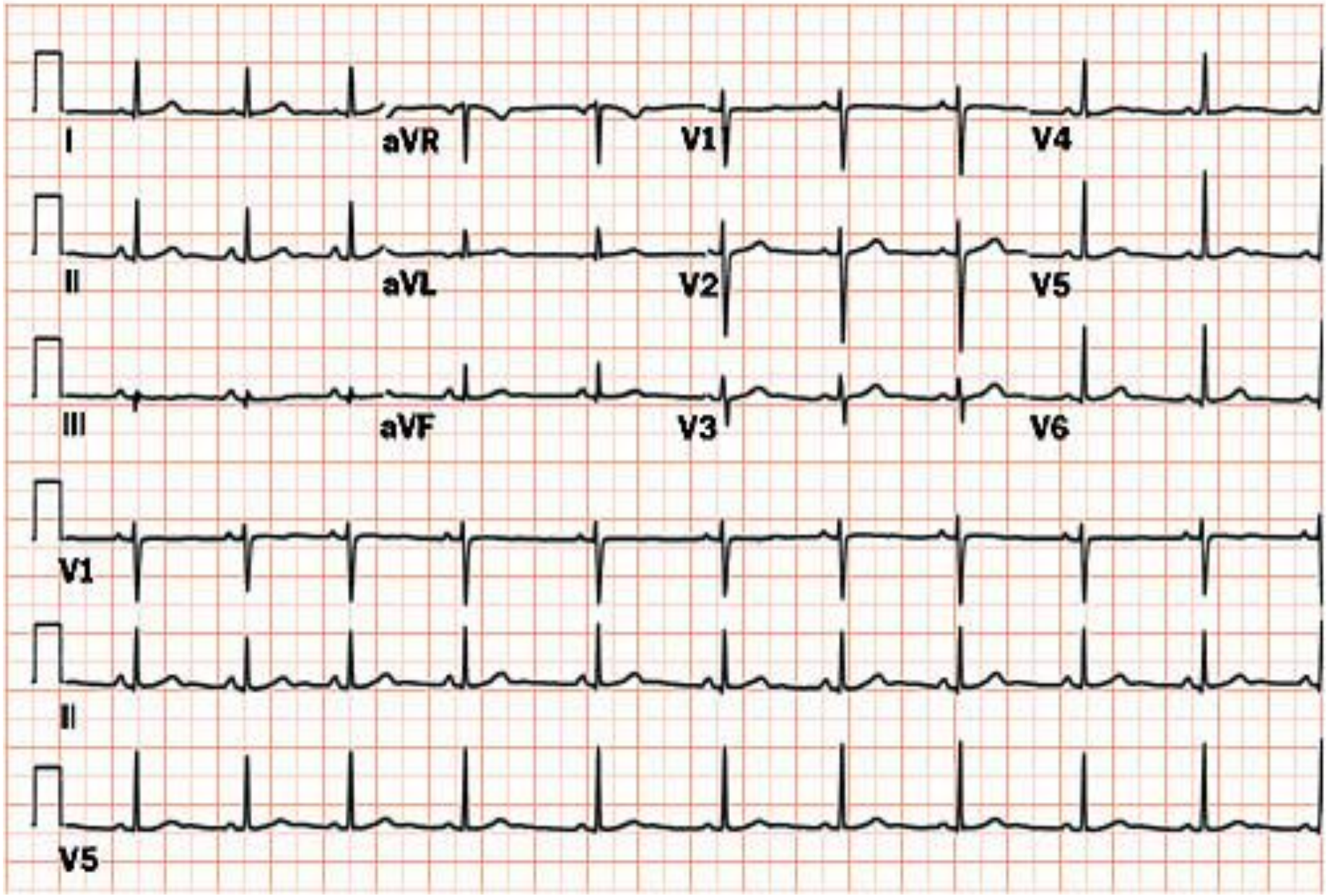
- Heart turnings:
  - round anterior-posterior axe;
  - round longitudinal axe;
  - round transversal axe.

Anterior-posterior axes

- Heart electrical axe position:

standard and augmented leads

- ***normal position*** **when angle α is from +30° till +69°;**
- ***vertical position*** **when angle α is from +70° till +90°;**
- ***horizontal position*** **when angle α is from 0° till +29°;**
  - ***axe inclination to the right*** **when angle α is from +91° till ±180°;**
  - ***axe inclination to the left*** **when angle α is from 0° till -90°.**
- Normal, horizontal and vertical position of heart electrical axe (from 0° till +90°) can be resent both in healthy people



Heart turnings determining round longitudinal axe

Thoracic leads / horizontal plane

- *ECG sign of heart turning round longitudinal axe clockwise* is the following: transitional zone (denses S and R equality) possible replacement to the left into
- V4-V5.
- *ECG sign of heart turning round longitudinal axe counter clockwise* the following: transitional zone possible replacement to the right in V2.

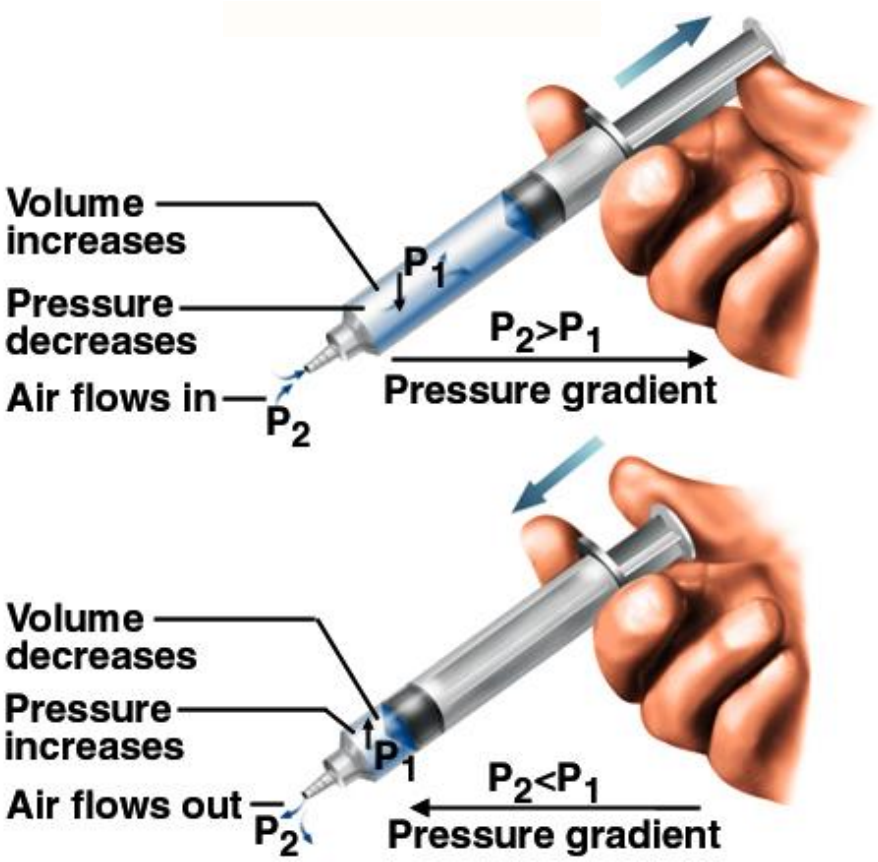
Cardiac Cycle

- One complete contraction and relaxation of all 4 chambers of the heart
- Atrial systole, Ventricle diastole
- Atrial diastole, Ventricle systole

- Quiescent period

Principles of Pressure and Flow

- Measurement: force (mmHg) required to stop flow - sphygmomanometer
- Change in volume creates a pressure gradient
- Opposing pressures
  - great vessels have positive blood pressure
  - ventricular pressure must rise above this pressure for blood to flow into great vessels



Heart Sounds

- Auscultation - listening to sounds made by body
- First heart sound (S1), louder and longer “lubb”, occurs with closure of AV valves
- Second heart sound (S2), softer and sharper “dupp” occurs with closure of semilunar valves
- S3 - rarely heard in people < 30

Phases of Cardiac Cycle

- Quiescent period
  - all chambers relaxed
  - AV valves open
  - blood flowing into ventricles
- Atrial systole
  - SA node fires, atria depolarize
  - P wave appears on ECG
  - atria contract, force additional blood into ventricles
  - ventricles now contain end-diastolic volume (EDV) of about 130 ml of blood

Isovolumetric Contraction of Ventricles

- Atria repolarize and relax
- Ventricles depolarize
- QRS complex appears in ECG
- Ventricles contract
- Rising pressure closes AV valves - heart sound S1 occurs
- No ejection of blood yet (no change in volume)

Ventricular Ejection

- Rising pressure opens semilunar valves
- Rapid ejection of blood
- Reduced ejection of blood (less pressure)
- Stroke volume: amount ejected, 70 ml at rest
- SV/EDV= ejection fraction, at rest ~ 54%, during vigorous exercise as high as 90%, diseased heart < 50%
- End-systolic volume: amount left in heart

Isovolumetric Relaxation of Ventricles

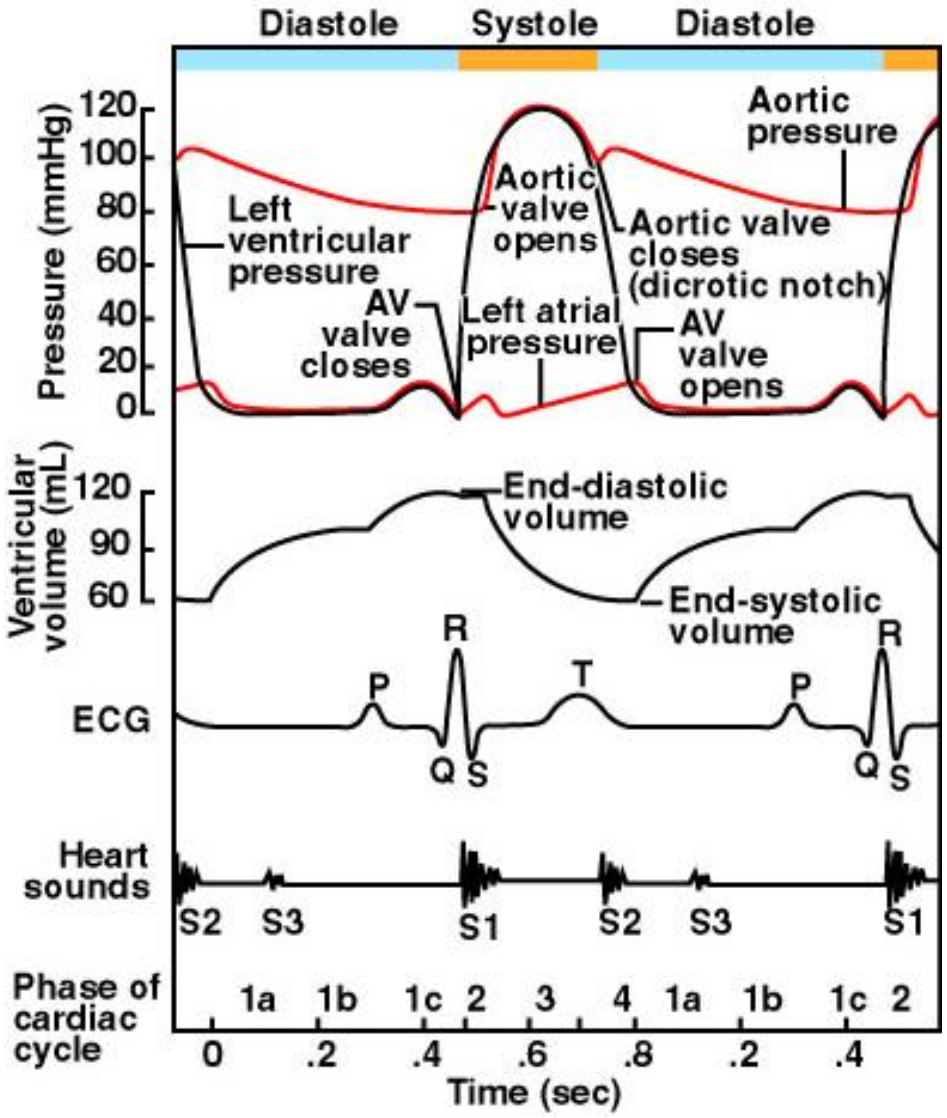
- T wave appears in ECG
- Ventricles repolarize and relax (begin to expand)
- Semilunar valves close (dicrotic notch of aortic press. curve) - heart sound S2 occurs
- AV valves remain closed
- Ventricles expand but do not fill (no change in volume)

Ventricular Filling - 3 phases

- Rapid ventricular filling - AV valves first open ↑pressure
- Diastasis - sustained lower pressure, venous return
- Filling completed by atrial systole

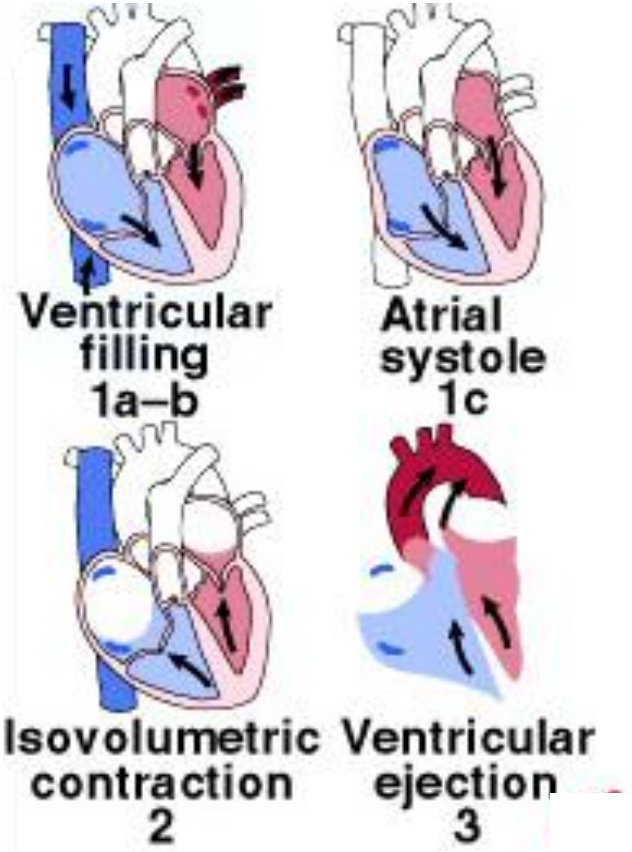
- Heart sound S3 may occur

Major Events of Cardiac Cycle



- Quiescent period
- Ventricular filling
- Isovolumetric contraction
- Ventricular ejection
- Isovolumetric relaxation

Events of the Cardiac Cycle



Phases of cardiac cycle:

1. Ventricular filling
   - 1a. Rapid filling
   - 1b. Diastasis
   - 1c. Atrial systole
2. Isovolumetric contraction
3. Ventricular ejection
4. Isovolumetric relaxation

Rate of Cardiac Cycle

- Atrial systole, 0.1 sec
- Ventricular systole, 0.3 sec
- Quiescent period, 0.4 sec
- Total 0.8 sec, heart rate 75 bpm

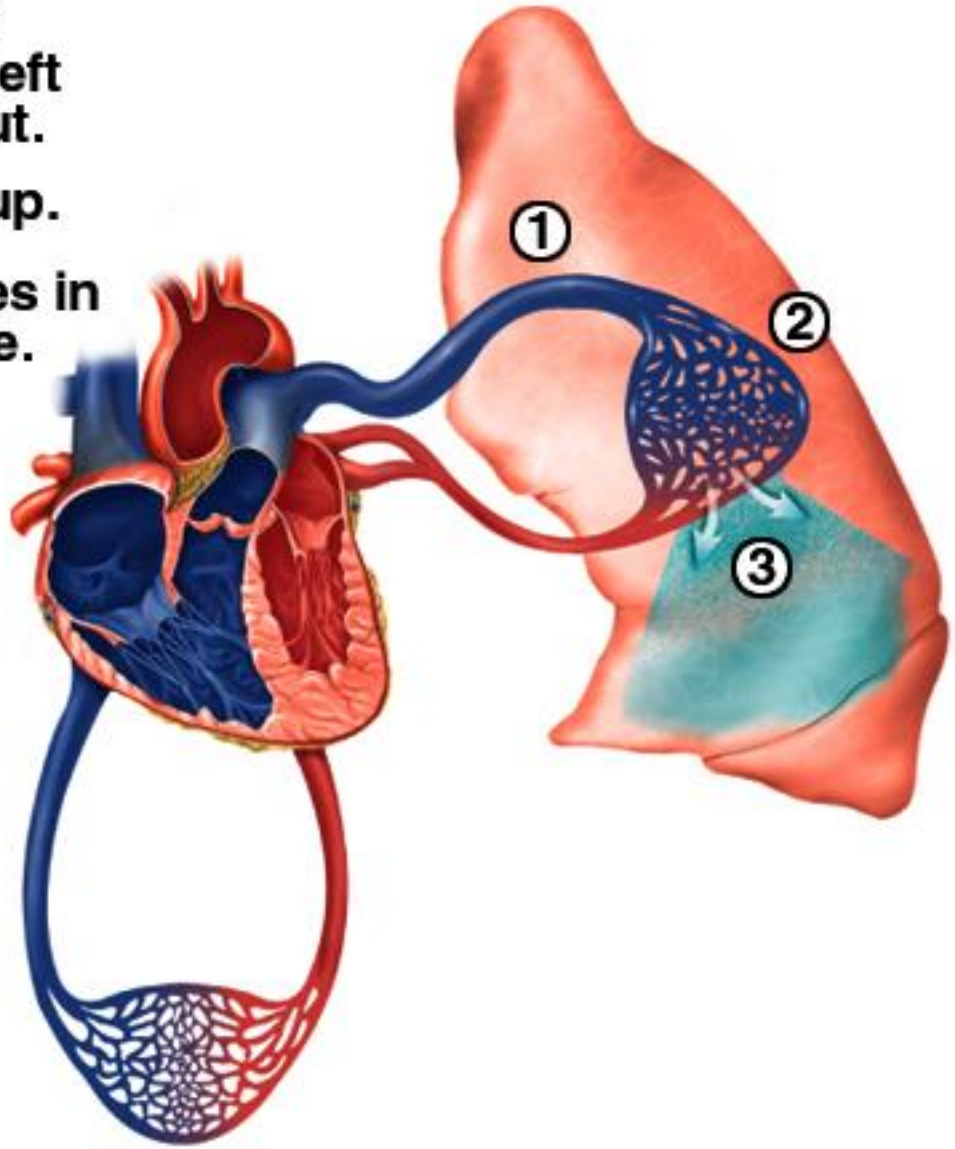
Ventricular Volume Changes at Rest

End-systolic volume (ESV)	60 ml
Passively added to ventricle during atrial diastole	+30 ml
Added by atrial systole	+40 ml
End-diastolic volume (EDV)	130 ml
Stroke volume (SV) ejected by ventricular systole	-70 ml
End-systolic volume (ESV)	60 ml

Both ventricles must eject same amount of blood

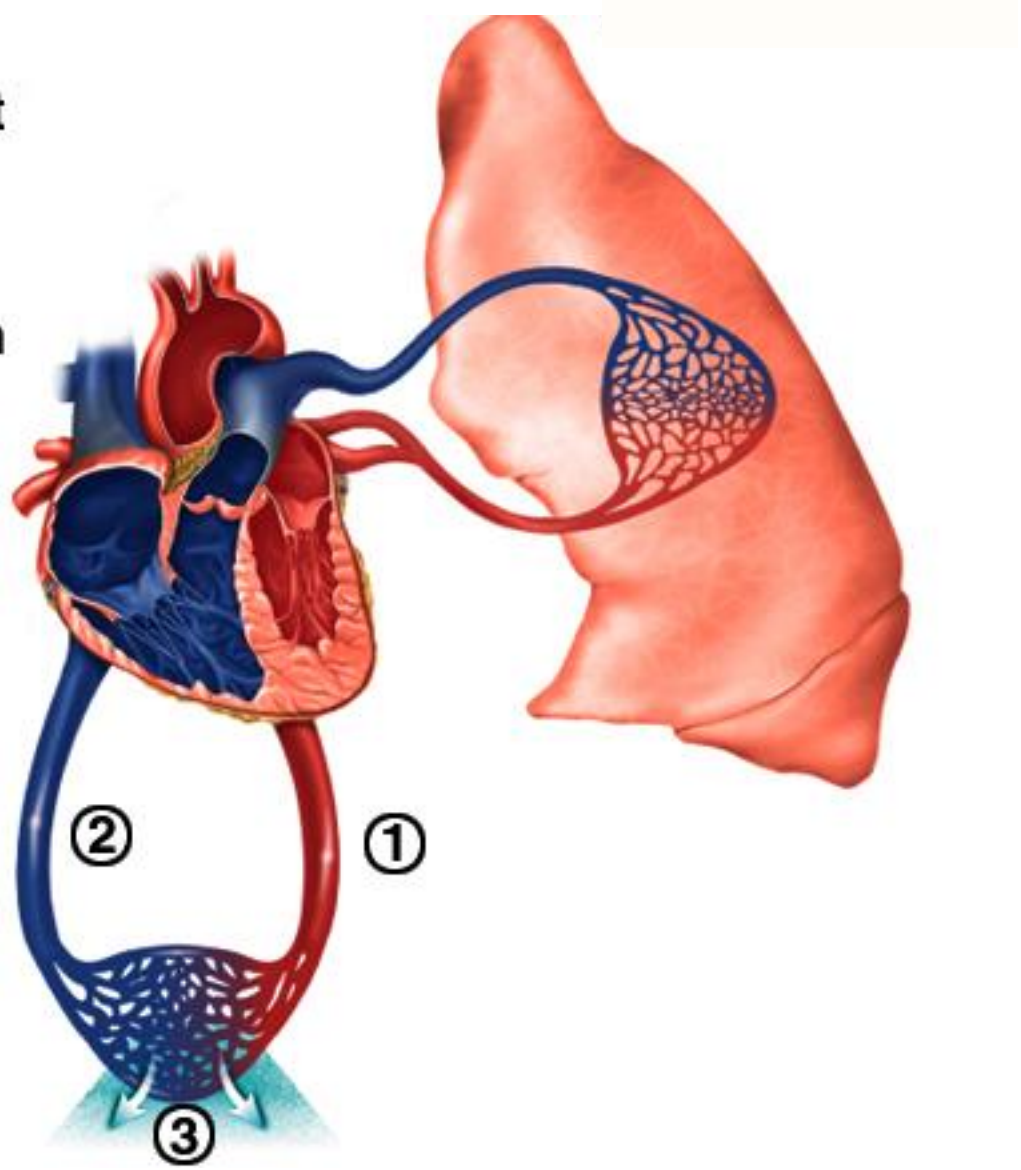
Unbalanced Ventricular Output

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



Unbalanced Ventricular Output

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



Cardiac Output (CO)

- Amount ejected by each ventricle in 1 minute
- CO = HR x SV
- Resting values, CO = 75 beats/min x70 ml/beat = 5,250 ml/min, usually about 4 to 6L/min
- Vigorous exercise ↑ CO to 21 L/min for fit person and up to 35 L/min for world class athlete
- Cardiac reserve: difference between a persons maximum and resting CO
  - ↑ with fitness, ↓ with disease

Heart Rate

- Measured from pulse
- Infants have HR of 120 beats per minute or more
- Young adult females avg. 72 - 80 bpm
- Young adult males avg. 64 to 72 bpm
- HR rises again in the elderly
- Tachycardia: persistent, resting adult HR > 100
  - stress, anxiety, drugs, heart disease or ↑ body temp.
- Bradycardia: persistent, resting adult HR < 60
  - common in sleep and endurance trained athletes (↑ SV)

Chronotropic Effects

- Positive chronotropic agents ↑ HR and negative chronotropic agents ↓ HR
- Cardiac center of medulla oblongata
  - an autonomic control center with 2 neuronal pools: a cardioacceleratory center (sympathetic), and a cardioinhibitory center (parasympathetic)

Sympathetic Nervous System

- Cardioacceleratory center
  - stimulates sympathetic cardiac accelerator nerves to SA node, AV node and myocardium
  - these nerves secrete norepinephrine, which binds to β-adrenergic receptors in the heart (+ chronotropic effect)
  - CO peaks at HR of 160 to 180 bpm
  - Sympathetic n.s. can ↑ HR up to 230 bpm, (limited by refractory period of SA node), but SV and CO ↓ (less filling time)

Parasympathetic Nervous System

- Cardioinhibitory center
  - stimulates vagus nerves
    - right vagus nerve - SA node
    - left vagus nerve - AV node
  - secrete ACH (acetylcholine), binds to muscarinic receptors
    - opens K^+ channels in nodal cells, hyperpolarized, fire less frequently, HR slows down
  - vagal tone: background firing rate holds HR to sinus rhythm of 70 to 80 bpm
    - severed vagus nerves - SA node fires at intrinsic rate-100bpm
    - maximum vagal stimulation ↓ HR as low as 20 bpm

Inputs to Cardiac Center

- Higher brain centers affect HR
  - sensory and emotional stimuli - rollercoaster,
  - cerebral cortex, limbic system, hypothalamus
- Proprioceptors
  - inform cardiac center about changes in activity, HR ↑ before metabolic demands arise
- Baroreceptors
  - pressure sensors in aorta and internal carotid arteries send continual stream of signals to cardiac center
    - if pressure drops, signal rate drops, cardiac center ↑ HR
    - if pressure rises, signal rate rises, cardiac center ↓ HR

Inputs to Cardiac Center 2

- Chemoreceptors
  - sensitive to blood pH, CO_2 and oxygen
  - aortic arch, carotid arteries and medulla oblongata
  - primarily respiratory control, may influence HR
  - ↑ CO_2 (hypercapnia) causes ↑ H^+ levels, may create acidosis (pH < 7.35)
  - Hypercapnia and acidosis stimulates cardiac center to ↑ HR

Chronotropic Chemicals

- Neurotransmitters - with cAMP as 2 messenger
  - norepinephrine and epinephrine (catecholamines) are potent cardiac stimulants
- Drugs
  - caffeine inhibits cAMP breakdown
  - nicotine stimulates catecholamine secretion
- Hormones
  - TH ↑ adrenergic receptors in heart, ↑ sensitivity to sympathetic stimulation, ↑ HR
- Electrolytes - K has greatest chronotropic effects
  - ↑ K myocardium less excitable, HR slow and irregular
  - ↓ K cells hyperpolarized, requires ↑ stimulation

Stroke Volume

- Governed by three factors:
  - preload, contractility and afterload
- ↑ preload or contractility ↑ SV
- ↑ afterload ↓ SV

Preload

- Amount of tension in ventricular myocardium before it contracts
- ↑ preload causes ↑ contraction strength
  - exercise ↑ venous return, stretches myocardium *(↑preload)* , myocytes generate more tension during contraction, ↑ CO matches ↑ venous return
- Frank-Starling law of heart - SV∝ EDV
  - ventricles eject as much blood as they receive; more they are stretched *(↑preload)* the harder they contract

Contractility

- Contraction force for a **given** preload
- Tension caused by factors that adjust myocyte's responsiveness to stimulation
  - factors that ↑ contractility are positive inotropic agents
    - hypercalcemia, catecholamines, glucagon, digitalis
  - factors that ↓ contractility are negative inotropic agents
    - hyperkalemia (K^+), hypocalcemia, hypoxia, hypercapnia

Afterload

- Pressure in arteries above semilunar valves opposes opening of valves
- ↑ afterload ↓ SV
  - any impedance in arterial circulation ↑ afterload
- Continuous ↑ in afterload (lung disease, atherosclerosis, etc.) causes hypertrophy of myocardium, may lead it to weaken and fail

Exercise and Cardiac Output

- Effect of proprioceptors
  - HR ↑ at beginning of exercise due to signals from joints, muscles
- Effect of venous return
  - muscular activity ↑ venous return causes ↑ SV
- ↑ HR and ↑ SV cause ↑CO
- Effect of ventricular hypertrophy
  - caused by sustained program of exercise
  - ↑ SV allows heart to beat more slowly at rest, **40-60bpm**
  - ↑ cardiac reserve, can tolerate more exertion

Coronary Atherosclerosis