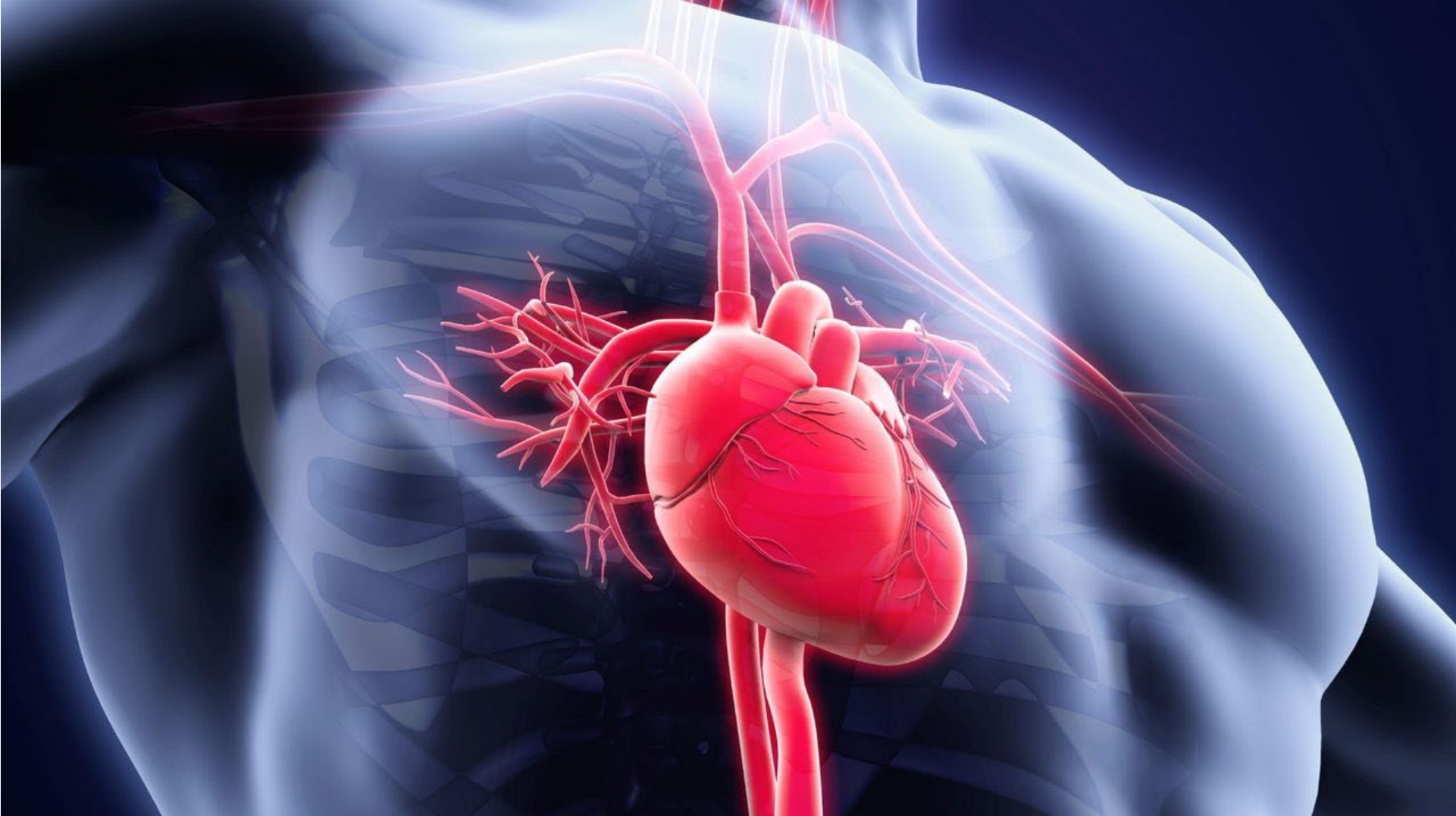


Chapter 19.2

The Heart



Intrinsic Cardiac Conduction System

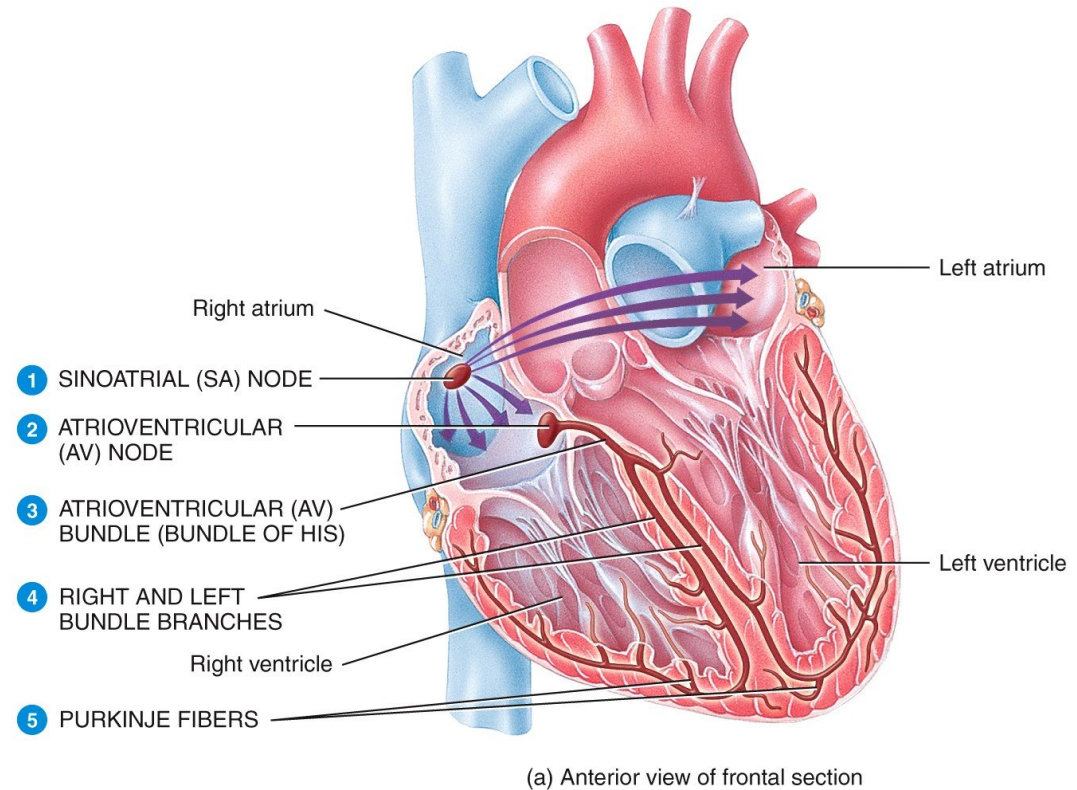


The ICCS coordinate the sequential contraction of the heart chambers during the cardiac cycle

The heart contracts every 0.8 seconds (cardiac cycle)

This generates 75 beats per minute

Each cycle pumps **70 ml of blood (stroke volume)** into the pulmonary trunk and aorta



Cardiac output = 70 ml/beat x 75 beats/min = **5.25 L per minute**

*This rhythmic action of the heart is regulated by **pacemakers**. Sequential contraction of the different heart chambers is regulated by the **intrinsic conduction system**.*

Intrinsic Cardiac Conduction System



Generates and conducts rhythmic electrical signals in the following order

Sinoatrial (SA) node

- these are modified cardiocytes
- initiates each heartbeat and determines heart rate
- *this intrinsic rate is modified by the autonomic nervous system!*
- signals spread throughout atria
- pacemaker in right atrium near base of superior vena cava

Atrioventricular (AV) node

- located near the right AV valve at lower end of inter-atrial septum
- electrical gateway to the ventricles
- **fibrous skeleton acts as an insulator**
- prevent currents from getting to the ventricles from any other route
- action potential delayed at AV node

Cardiac Conduction System



Atrioventricular (AV) bundle (bundle of His)

- bundle forks into right and left bundle branches
- these branches pass through interventricular septum toward apex

Purkinje fibers

- nerve like processes spread throughout ventricular myocardium
- from end of Purkinje Fibers // signal pass from cell to cell through gap junctions

Nodal Tissue and Nerve Supply of the Heart

Myocardocytes are **not able to maintain a resting membrane potential** // these cells allow sodium to leak into their cytoplasm

The myocardocyte with the highest rate of ion leakage (SA node) spontaneously reach threshold and cause an action potential (nodal potential) to occur

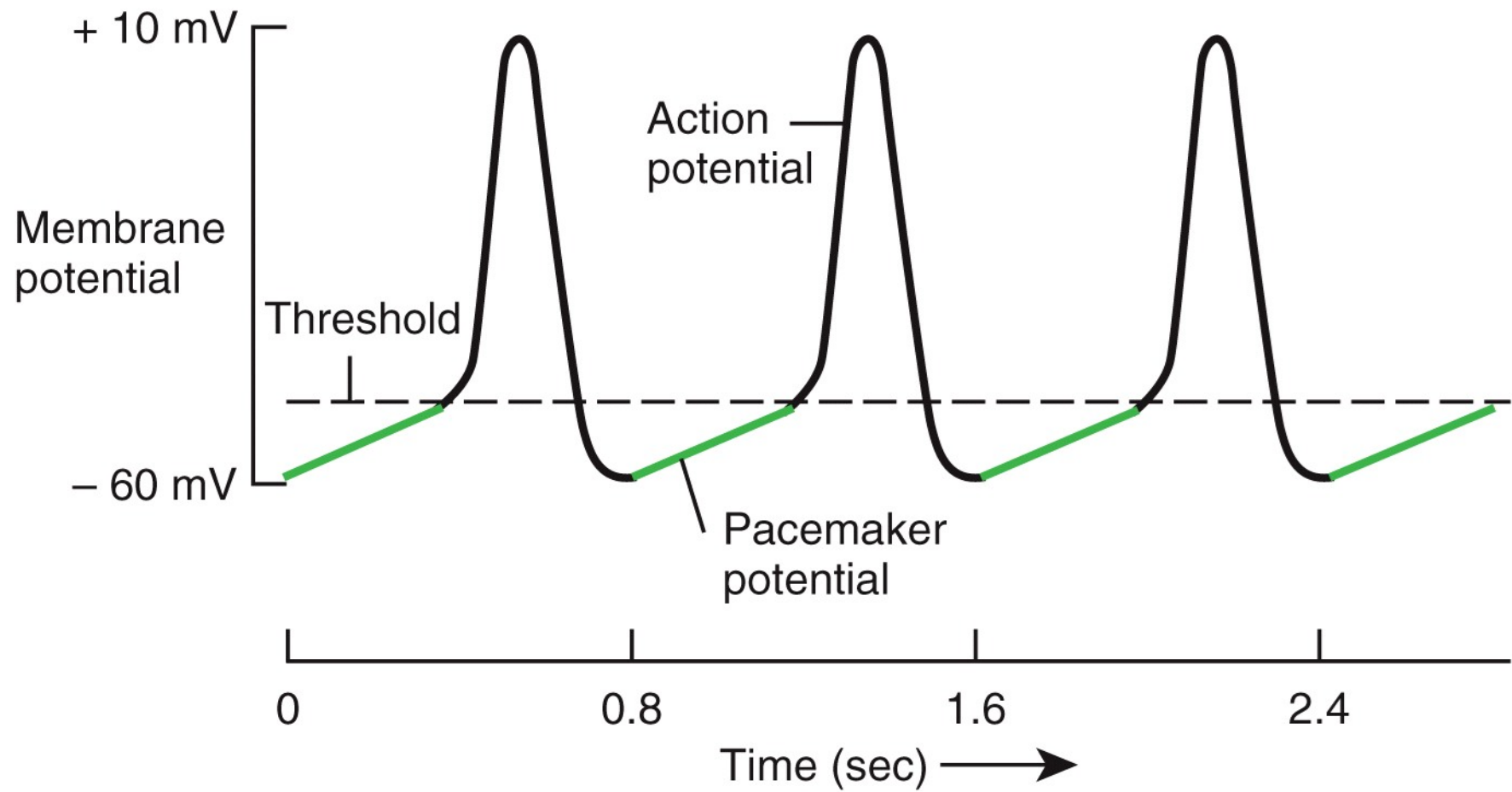
Sinoatrial Node (SA node) and **Atrioventricular Node** (AV node) are two area that **“leak sodium ions”** faster than any other myocaridocytes // SA leaks fastest so it sets the rate for the heart

If the SA node is lost (e.g. virus kills these cells) then AV note will now set the rate of depolarization for all the heart

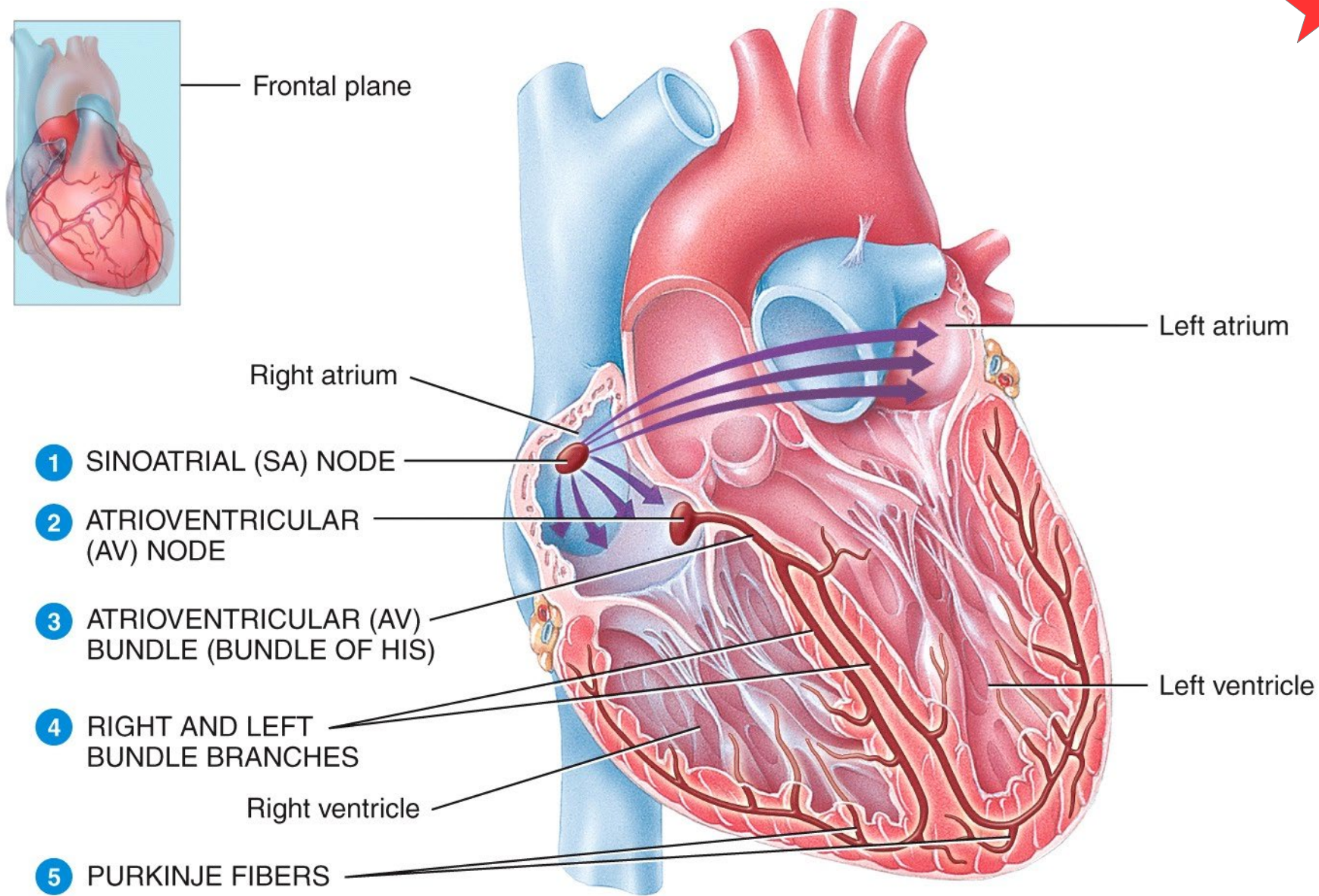
Any myocardocyte removed from the heart may be used to demonstrate the cardiocyte inability to maintain a resting membrane potential

The rate of nodal depolarization is **“modified”** by the autonomic nervous system

- Sympathetic NS - increase rate
- Parasympathetic NS - decrease rate



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



Frontal plane

Left atrium

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 ventricle

(a) Anterior view of frontal section

Significance of AV Delay

Action potential from SA node must completely depolarize right and left atria before ventricles start to depolarize // but the distance between the SA node and far side of left atria is greater than the distance between SA node and AV node

Action potential reaches AV node in 50 msec before action potential reaches far side of the left atria

Therefore – action potential must be delayed at AV node /// delays signal 100 msec

This allows action potential to completely depolarize all of the left atria and allows for the ventricles to receive blood from atria before ventricles start to contract

Atrial ventricular septum blocks flow of action potential because septum does not have gap junctions.

Cardiocytes at AV node have fewer gap junctions than between most cardiocytes // this explains the delay at AV node

Impulse Conduction to Myocardium

Signals travel very quickly through AV bundle and into Purkinje fibers

Depolarization is initiated at apex /// then entire ventricular myocardium depolarizes and contracts in near unison

Papillary muscles contract an instant earlier than the ventricular myocytes
/// tightening the slack in chordae tendineae

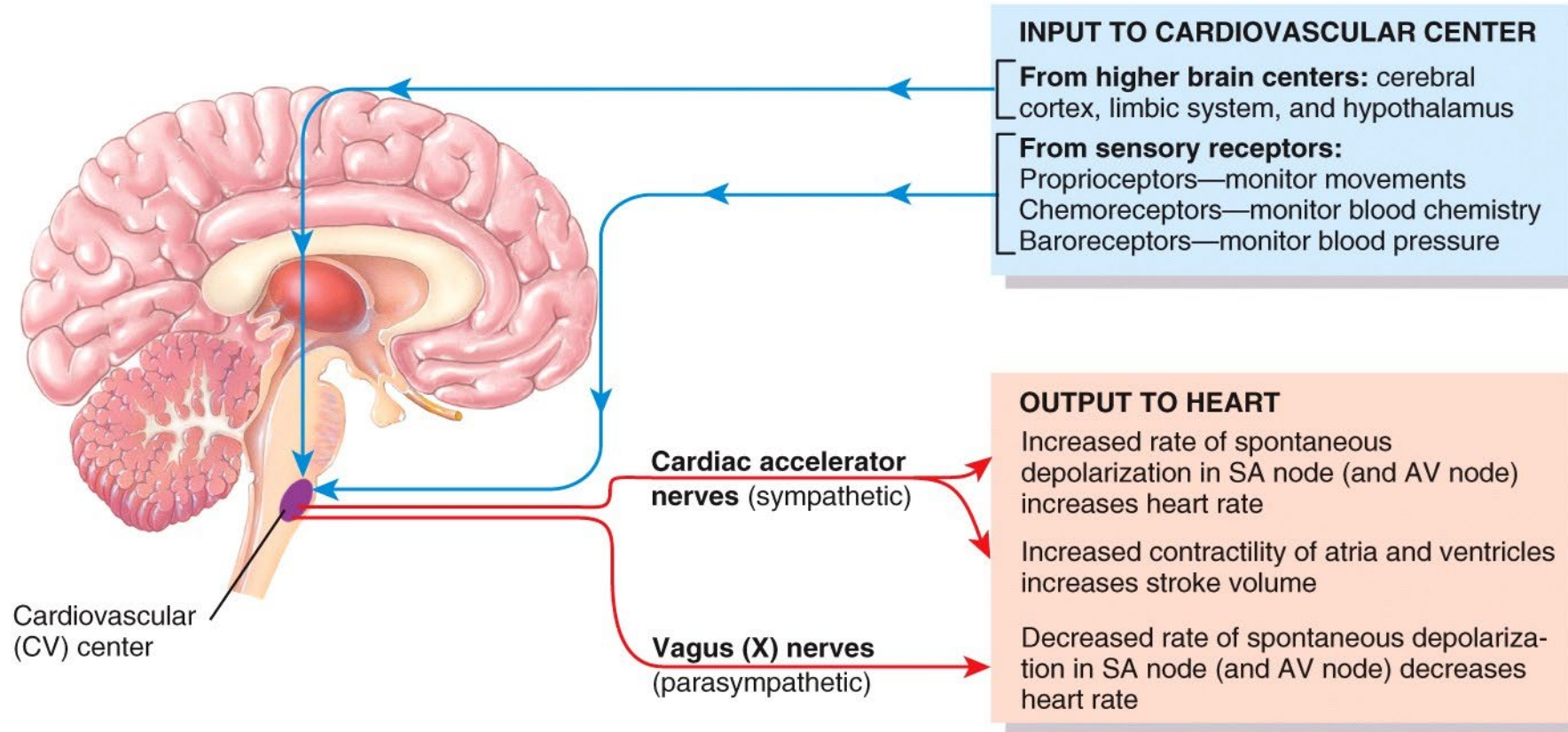
Ventricular systole progresses from the apex of the heart towards the base of the heart

Spiral arrangement of cardiocytes twists ventricles slightly like someone wringing out a towel

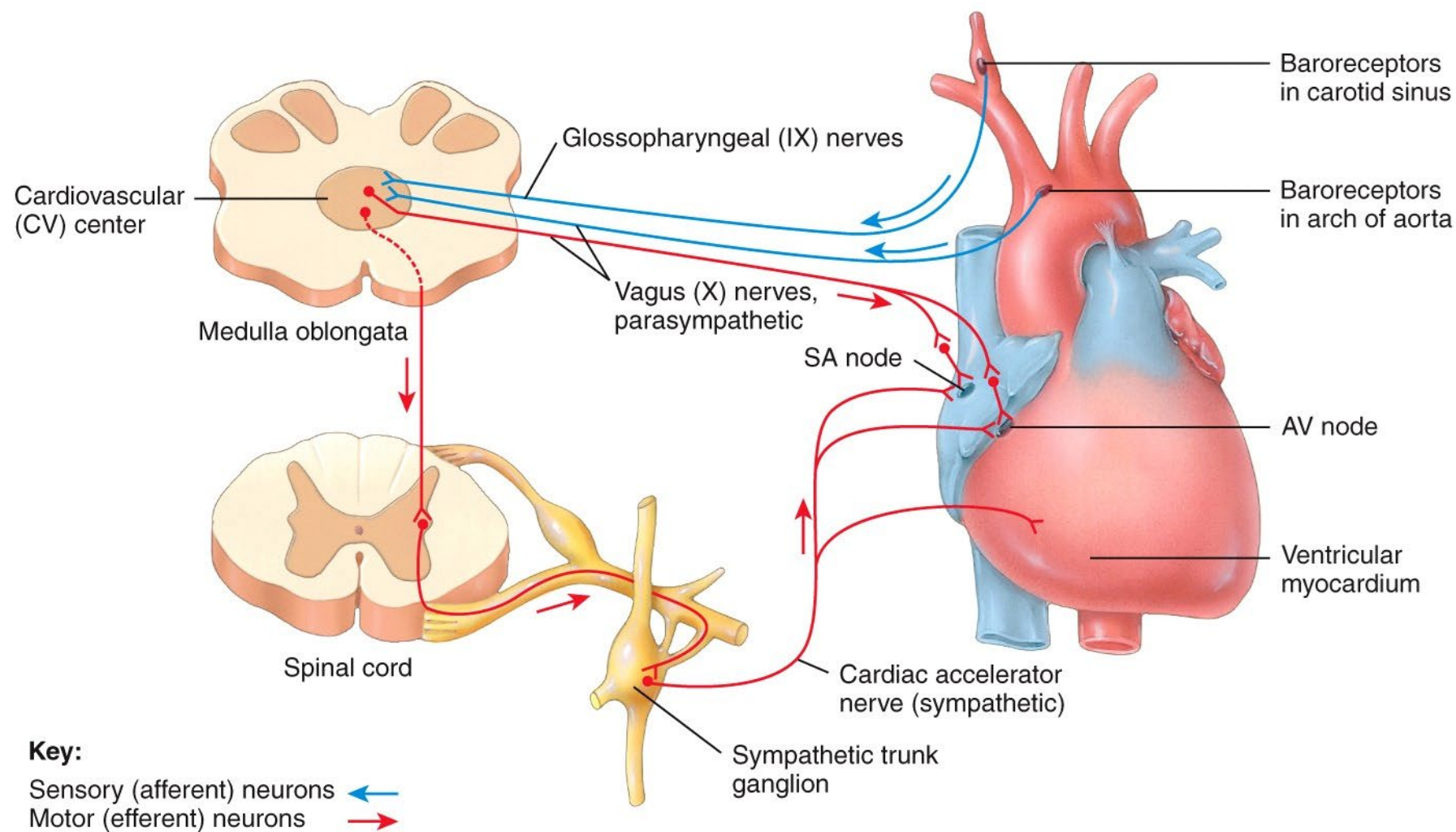
The cardiac control center is located in the **medulla oblongata**.



SA node provide auto rhythm, but the rate of SA node depolarization may be modified by stimuli from different inputs



Note: Sympathetic fibers also dilate coronary arteries!



Regulation (Inputs) to Cardiac Center

Cardiac centers located in the medulla oblongata (reticular formation)

Receive input from many sources and integrate them into 'decisions' to either speed up or slow down the heart /// chemo and baro receptors

Higher brain centers can affect heart rate

Cerebral cortex, limbic system, hypothalamus // somato-sensory or emotional stimuli // may influence these effects using biofeedback and or meditation

Heart rate may start to increase even “before the event starts!” // anticipation of muscular activity

Regulation (Inputs) to Cardiac Center

Proprioception is a type of sensation located in the muscles and joints // receptors are called proprioceptors

- **Medulla also receives input from muscles & joints**
- **Inform cardiac center about changes in activity**
- **This will allow HR to increase before metabolic demands of muscle rises**

Regulation (Inputs) to Cardiac Center

Baroreceptors send signal to cardiac center

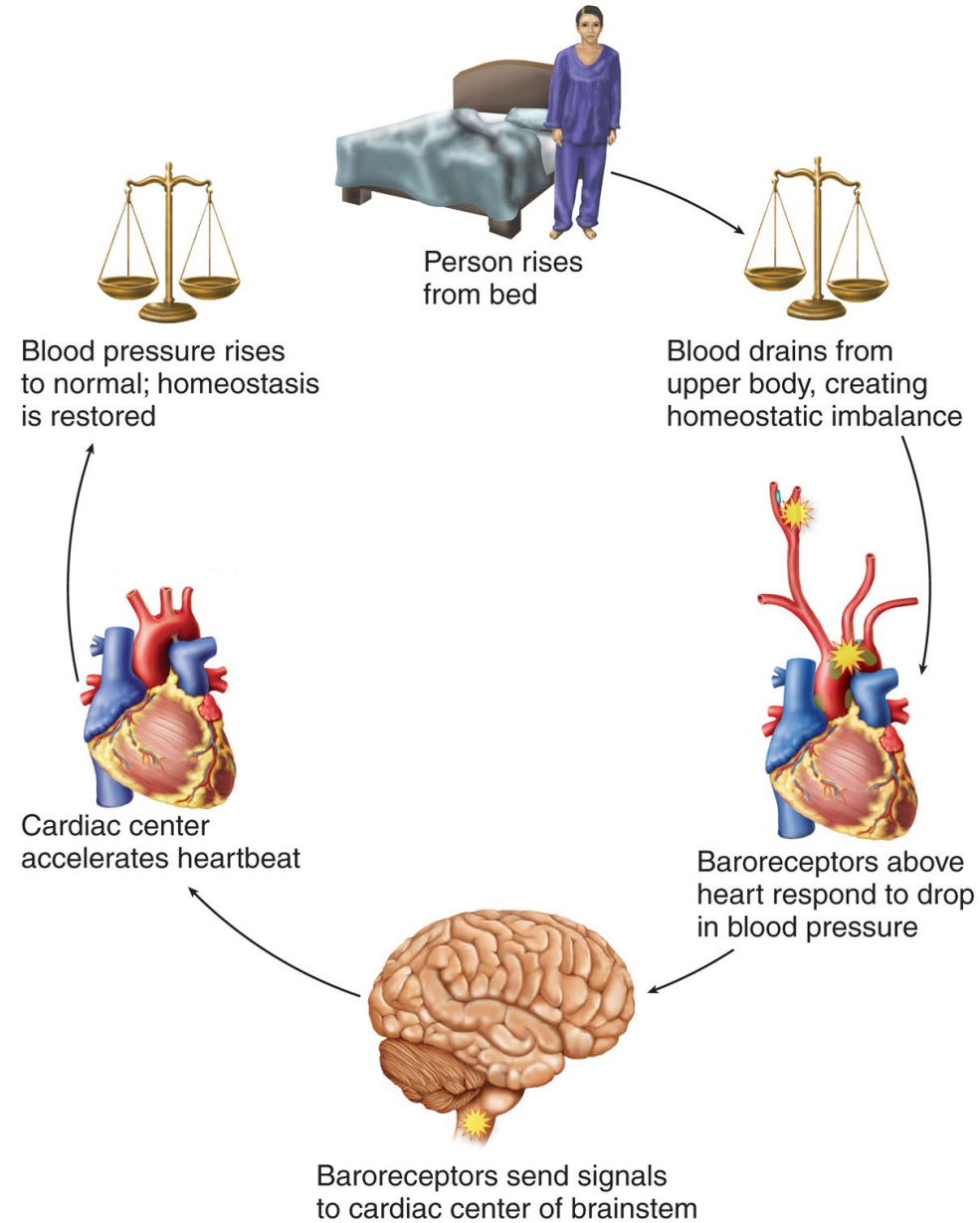
Pressure sensors (**called sinuses**) located: in aortic arch & internal carotid arteries

If blood pressure decreases // cardiac center increases heart rate // more blood pumped into vessels and blood pressure increases

If blood pressure increases /// cardiac center decreases heart rate // less blood pumped into vessels and blood pressure decreases

Blood Pressure & Heart Function

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Regulation (Inputs) into Cardiac Center

Chemoreceptors (three locations):

- aortic arch
- carotid arteries
- medulla oblongata

Sensitive to blood pH, CO₂ and O₂ levels

Chemoreceptors are more important in respiratory control than cardiac control // minor role in heart function

If CO₂ accumulates in blood or CSF (hypercapnia), reacts with water and causes increase in H⁺ levels

H⁺ lowers the pH of the blood possibly creating acidosis (pH < 7.35)

Regulation (Inputs) into Cardiac Center

Hypercapnia (high CO₂) and acidosis stimulate the cardiac center to increase heart rate

Hypoxemia (oxygen deficiency in the blood) usually slows down the heart

Chemoreflexes and baroreflexes use negative feedback loops

Sympathetic Nerve Supply to Heart

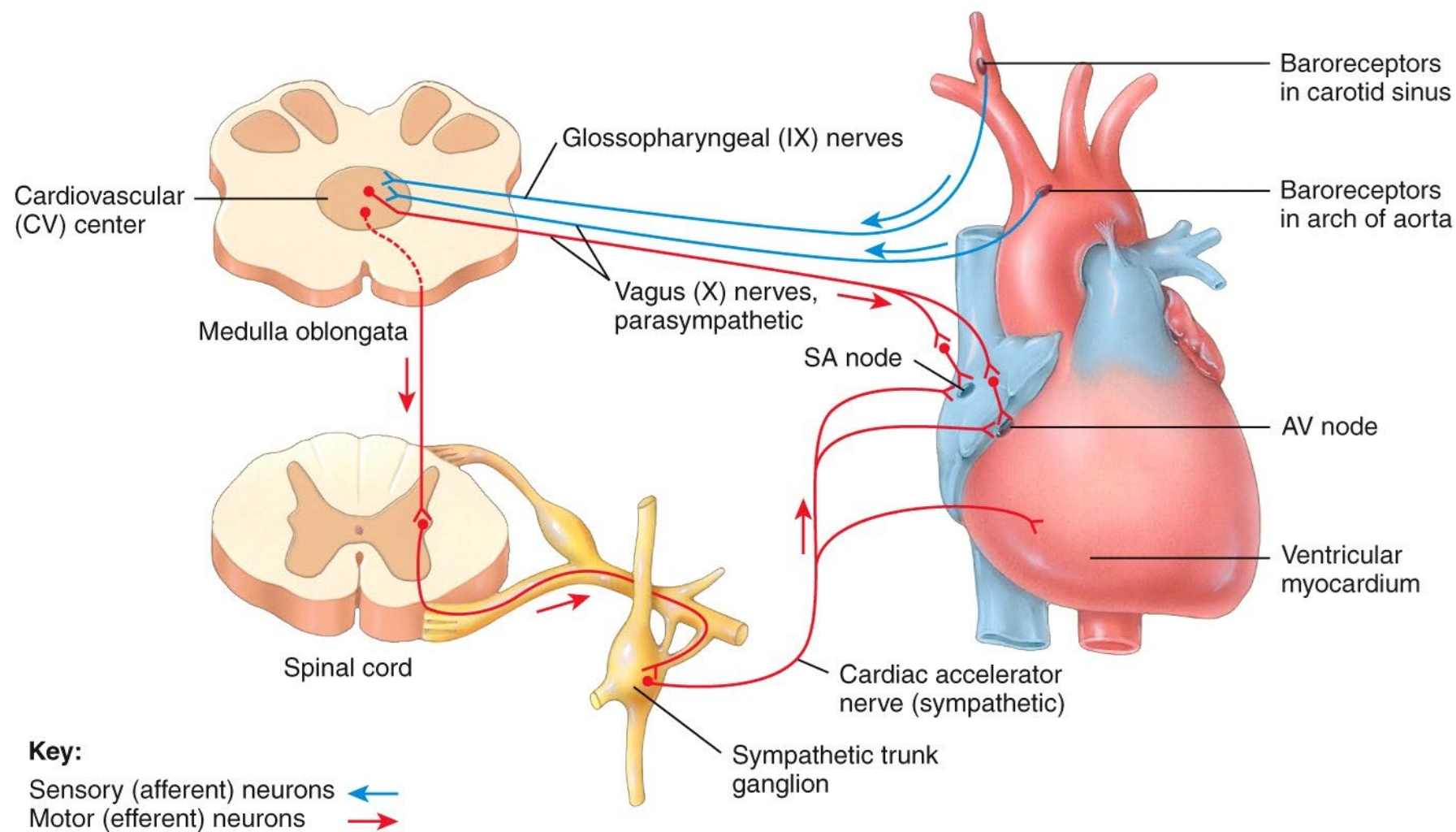
Sympathetic nerves

sympathetic pathway to the heart originates in the lower cervical to upper thoracic segments of the spinal cord

continues to adjacent sympathetic chain ganglia

some pass through **cardiac plexus** in mediastinum

continue as **cardiac nerves** to the heart



Sympathetic Nerve Supply to Heart



Sympathetic nerve fibers terminate in

SA and AV nodes

In atrial and ventricular myocardium

Coronary arteries (as well as the aorta, pulmonary trunk)

Three effects:

1 - increase heart rate

2 - increase contraction strength

3 - dilates coronary arteries

Sympathetic Postganglionic Fibers



Adrenergic fiber release norepinephrine

- Bind to beta 1 receptors
- Use second messenger system // cAMP
- Myocardocytes and nodal cells
- G protein activates enzymes that **open plasma channels** to allow calcium to enter cell // this increases depolarization rate
- cAMP also **increase uptake of calcium back into sarcoplasmic reticulum** // speed up repolarization rate

Overall outcome is to increase heart rate

- From 75 bpm (resting) to maximum of 240 bpm
- Cardiac output will not increase after 160 bpm. **Why? (ventricular filling!)**

Parasympathetic Nerve Supply to Heart

Parasympathetic Nerves

Pathway begins with **nuclei of the vagus nerves** in the medulla oblongata

Extend to **cardiac plexus** and continue to the heart by way of the **cardiac nerves**

Fibers of right vagus nerve lead to the SA node

Fibers of left vagus nerve lead to the AV node

Little or no vagal stimulation of the myocardium

No influence on myocarial blood vessels

Parasympathetic stimulation reduces the heart rate // **slows heart rate**

Parasympathetic Postganglionic Fibers



Cholinergic fibers release acetylcholine

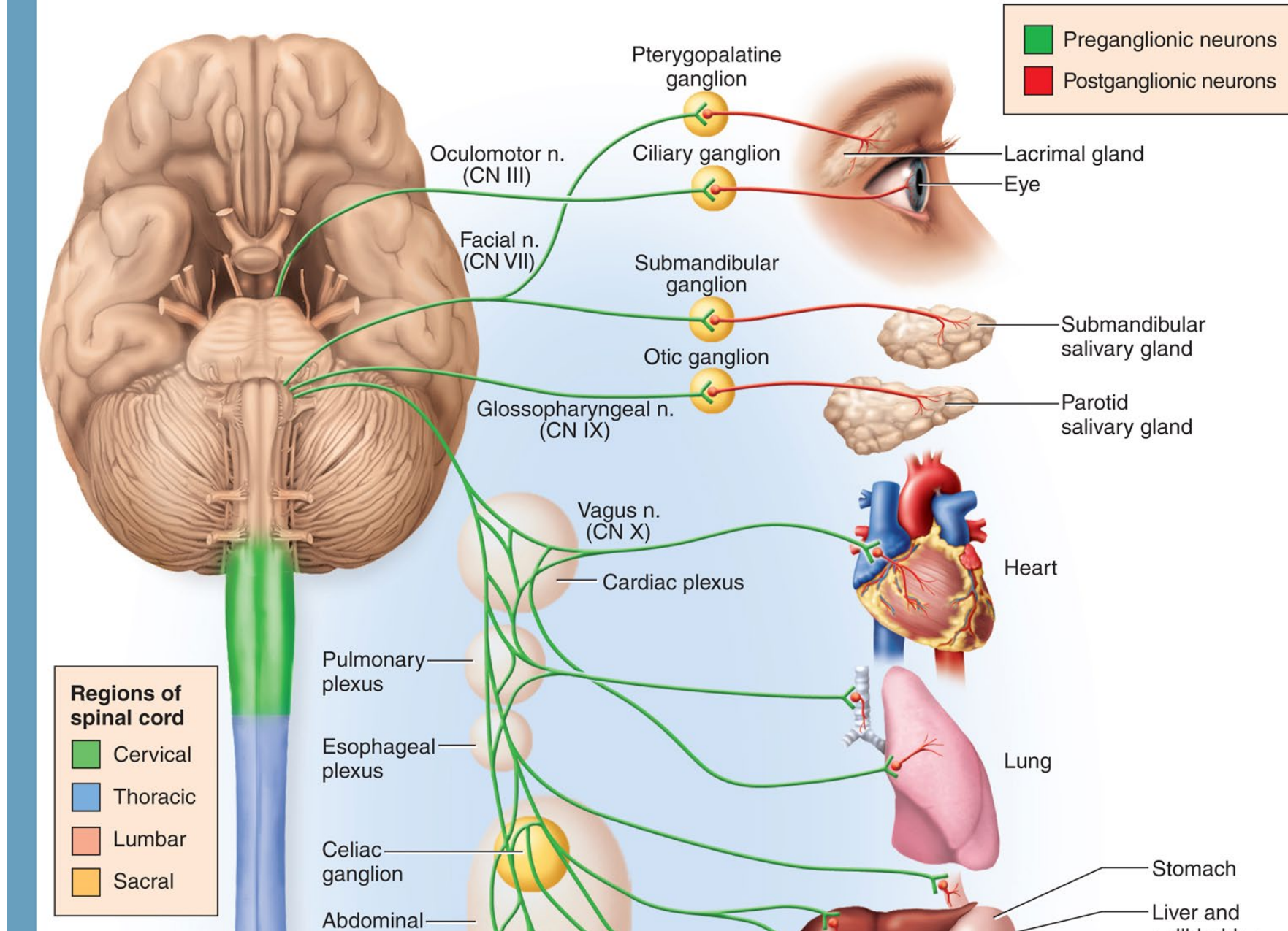
The myocytes do not use ionotropic receptors!

Myocytes use **muscarinic receptors** (metabotropic receptor = second messenger system)

Synapse only on SA and AV nodes

G protein activates enzymes that open plasma membrane channels to **allow potassium to exit cells** // increase repolarization rate

Slows down heart rate



Cardiac Rhythms and Vagal Tone



The state of the cardiac muscle following depolarization and repolarization are called

Systole – this occurs immediately after depolarization of atrial and ventricles (i.e. associated with heart contraction)

Diastole – this occurs immediately after repolarization of atrial and ventricles (i.e. associated with heart relaxation)

Sinus rhythm - normal heartbeat triggered by the SA node

Heart rate benchmark for sinus rhythm is 75 bpm

Vagal tone – vagus nerve under normal conditions suppresses the heart rate /// if all ANS fibers are cut going to heart then heart rate would be 100 bpm /// **Why does this make sense????**



Ectopic focus // caused by myocardial cell that depolarized faster than SA node depolarized

Caused by hypoxia, electrolyte imbalance, caffeine, nicotine, cocaine and other drugs

Nodal rhythm = heart cycle controlled by AV nodal // if SA node is damaged, heart rate is then set by AV node – slower rate of depolarization // Will the atria depolarize? Significance?

40 to 50 bpm (test benchmark – **50 bpm**) // not adequate for active lifestyle but able to survive with nodal rhythm

Cardiac Rhythms / Terminology

Intrinsic ventricular rhythm // occurs if both SA and AV nodes are destroyed

Rate now set by random myocardiocyte with fastest leaking sodium channel within ventricle but this rate is only at 20 to 40 bpm (**test benchmark = 25bpm**)

Cardiac output not sufficient to sustain life /// this requires artificial pacemaker to sustain long term life

Arrhythmia // any abnormal cardiac rhythm

- could be failure of nodal potential(s)
- conduction system to transmit signals
- bundle branch block

Total heart block // occurs because damage to AV node /// Action potential fails to pass AV node

Cardiac Rhythms / Terminology

Premature ventricular contractions (PVCs) // caused by stimulants, stress or lack of sleep

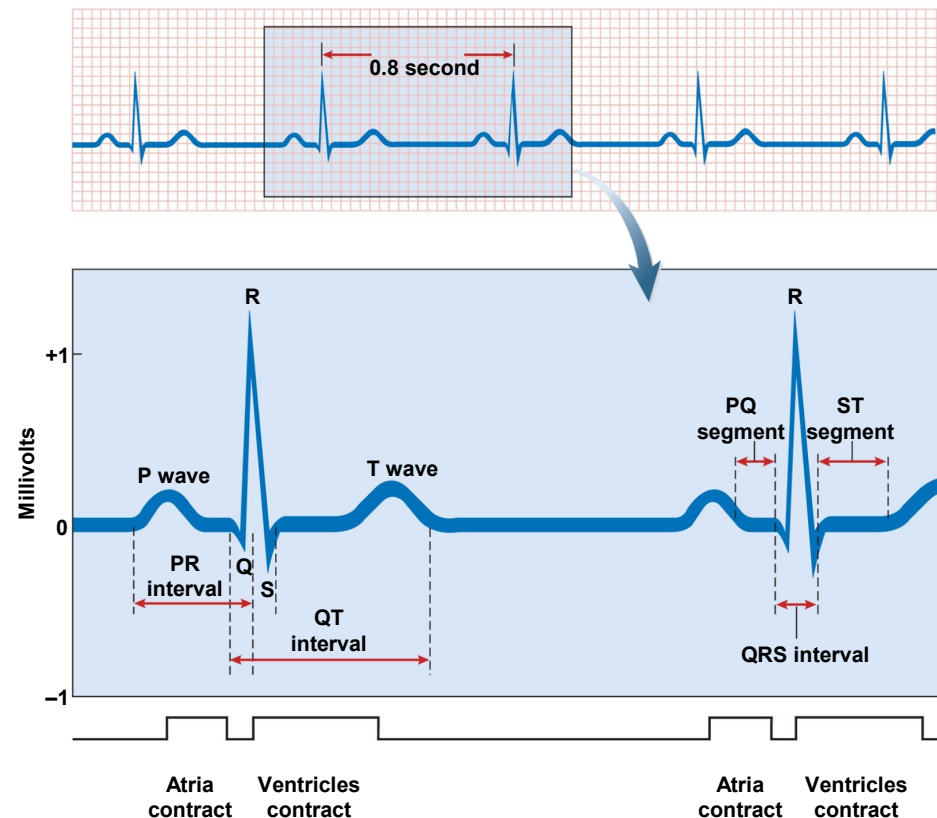
Tachycardia // Persistent resting adult heart rate above 100 bpm

Bradycardia // Persistent resting adult heart rate below 60 bpm

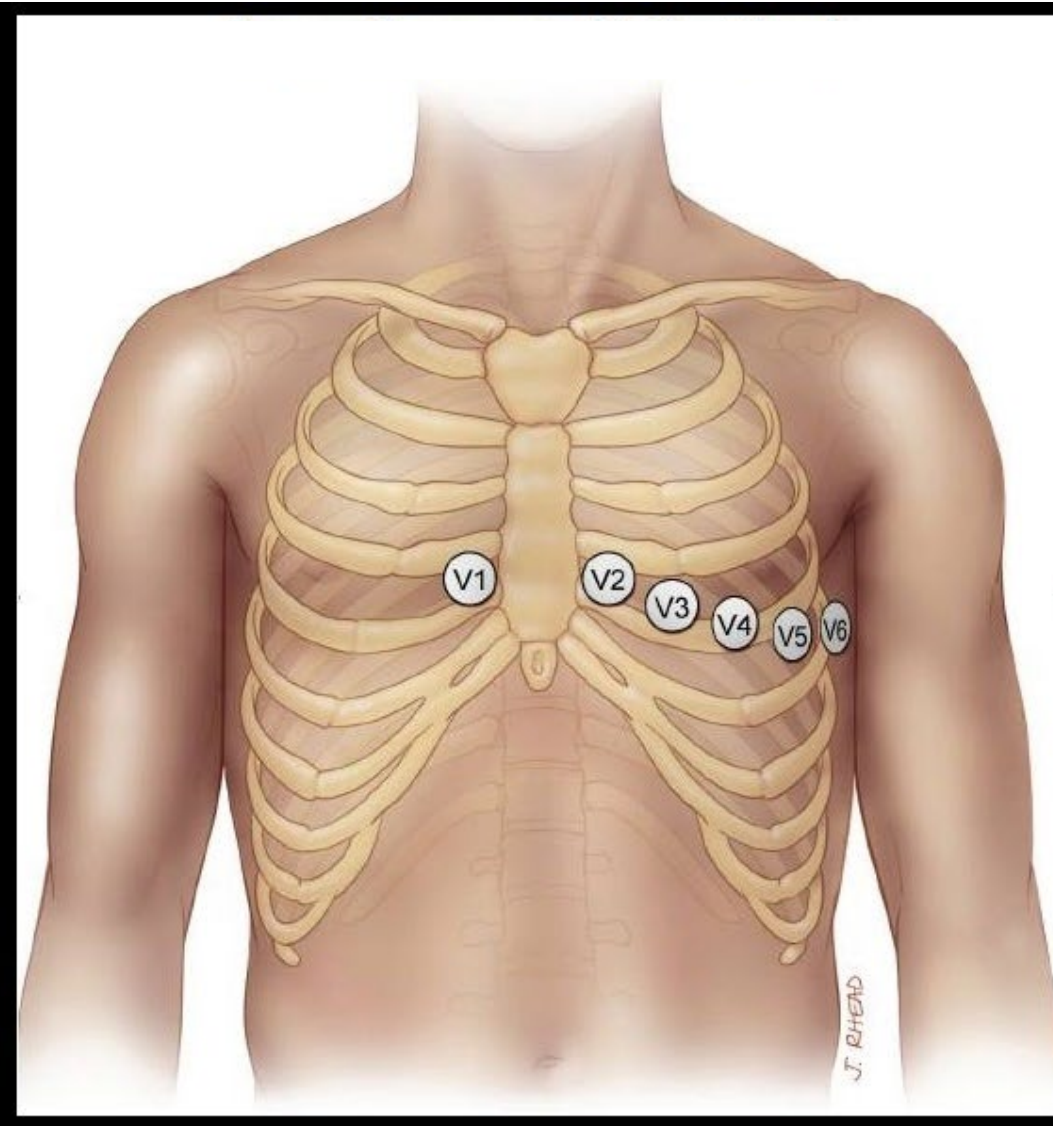
Electrocardiogram (ECG or EKG)



- All the electrical activity from the heart's action potentials (nodal cells and myocardial cells) is carried by “electrolytes” to the skin surface.
- The electrical event is detected, amplified and recorded by electrodes on arms, legs and chest // different methods used in lead placement
- Composite recording then associated with events in cardiac cycle



Proper Placement of 6 Chest Leads EKG



V1 4th Intercostal space to the right of the sternum

V2 4th Intercostal space to the left of the sternum

V3 Midway between V2 and V4

V4 5th Intercostal space at the midclavicular line

V5 Anterior axillary line at the same level as V4

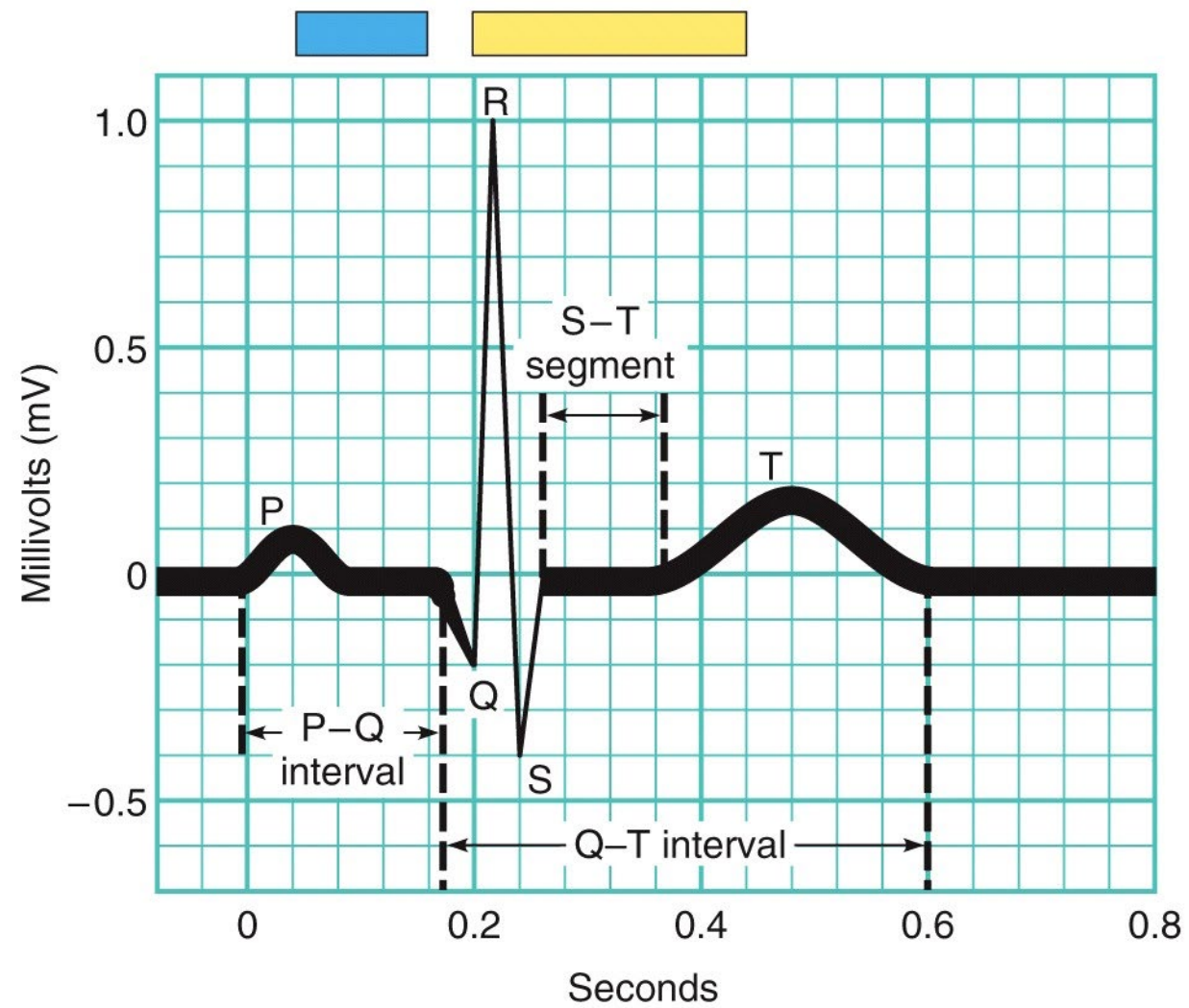
V6 Midaxillary line at the same level as V4 and V5

RL Anywhere above the ankle and below the torso

RA Anywhere between the shoulder and the elbow

LL Anywhere above the ankle and below the torso

LA Anywhere between the shoulder and the elbow



Key:

-  Atrial contraction
-  Ventricular contraction

P wave = SA node depolarizes

PQ segment

- follow atria depolarization
- period of atrial systole
- atrial systole begins 100 msec after SA signal

QRS complex

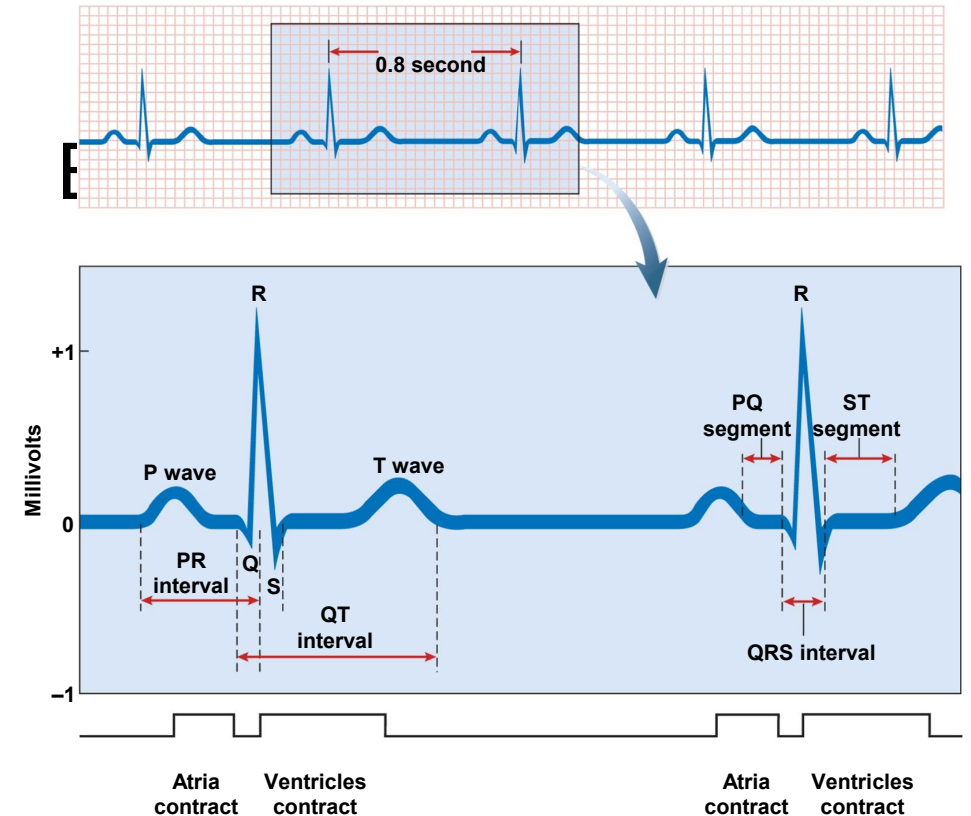
- ventricular depolarization
- complex shape of spike due to different thickness and shape of the two ventricles
- occurs when atria repolarize

ST segment

- ventricular systole
- plateau in myocardial action potential extends the ST segment
- longer than PQ

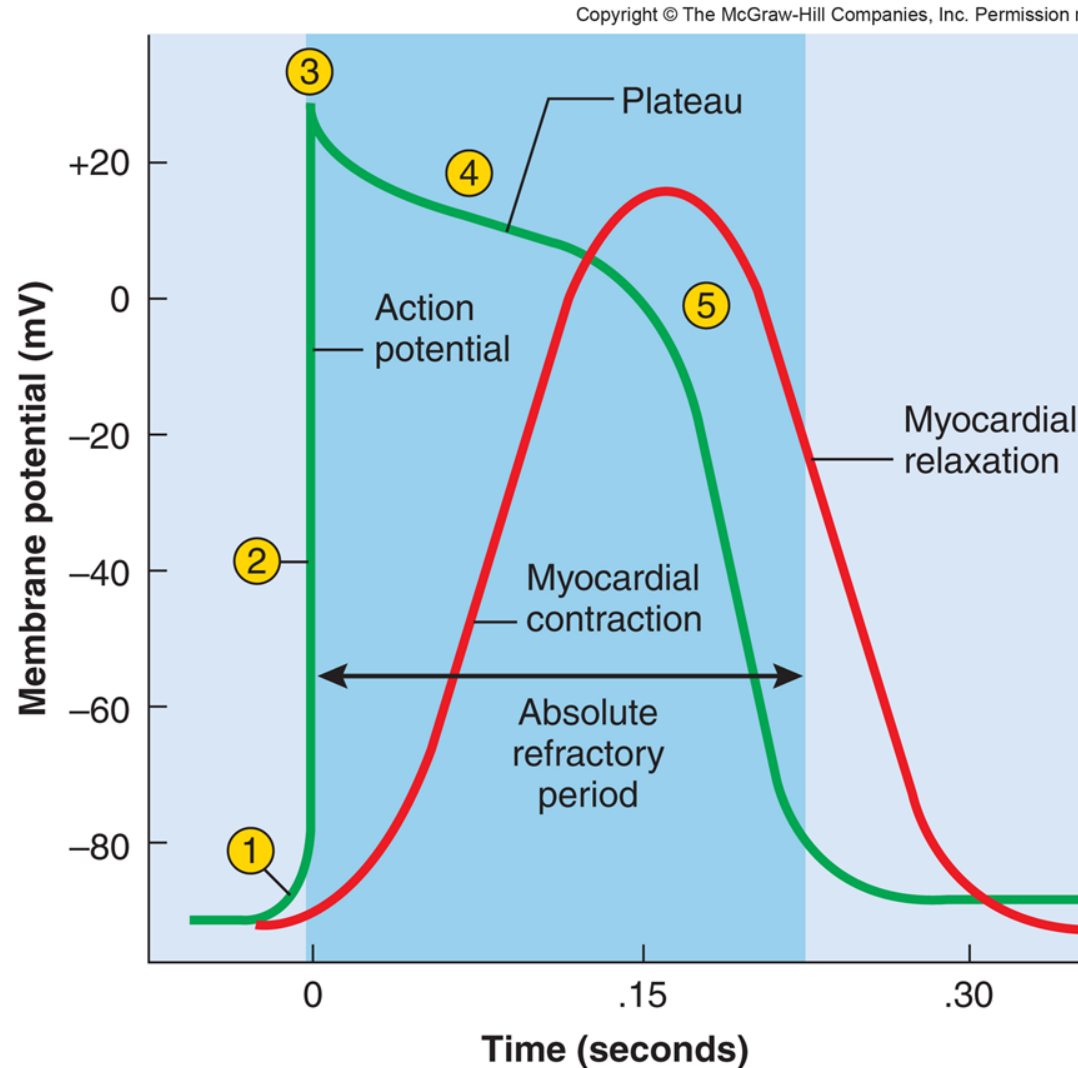
T wave = ventricular repolarization and relaxation

Electrocardiogram

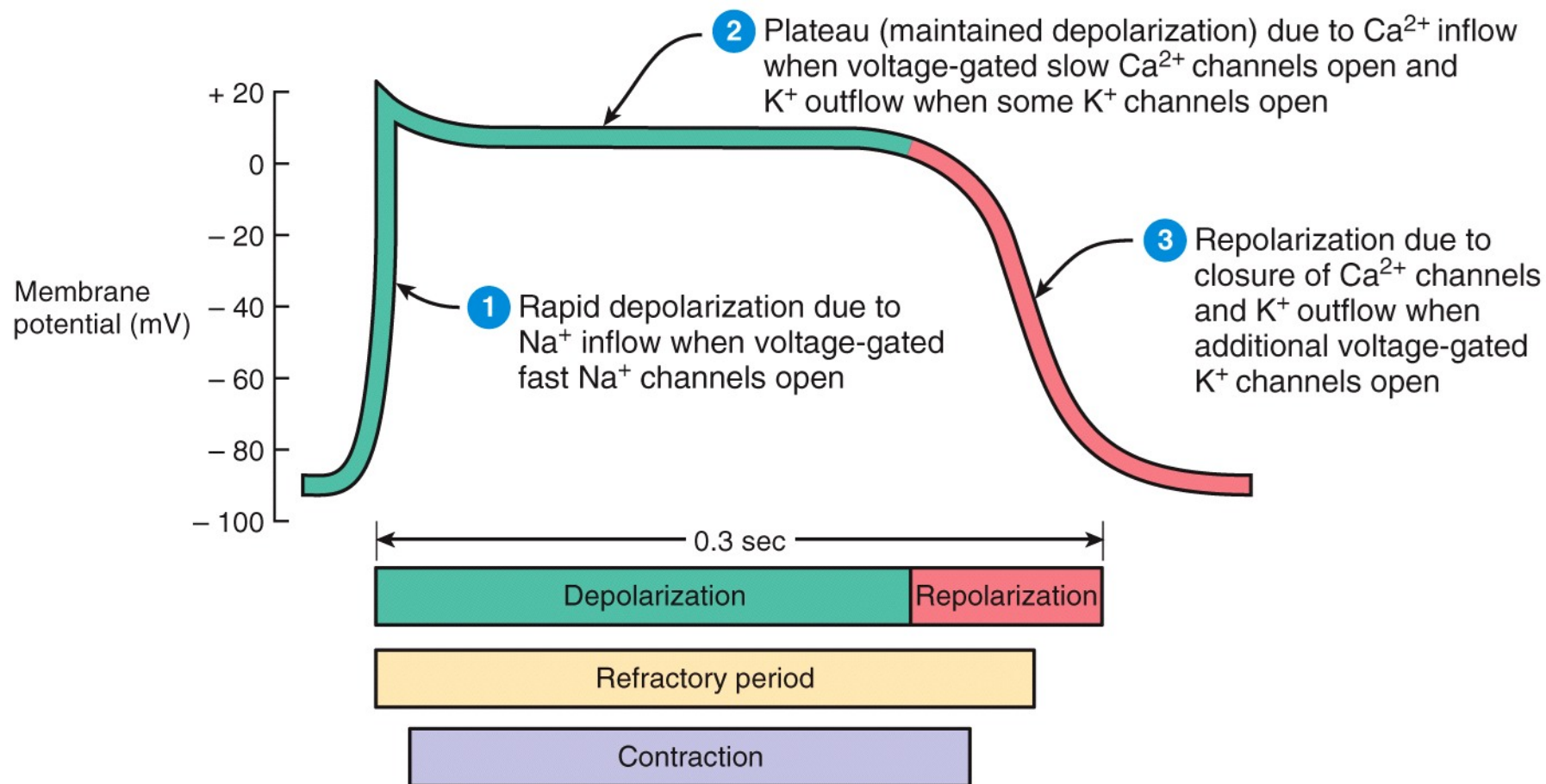


Note: asystole = “flat line” / no contraction of myocardium / requires cardiopulmonary resuscitation (CPR)

Action Potential of Mycardiocyte

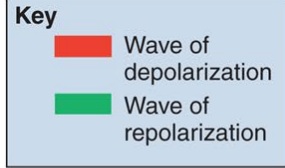


- ① Voltage-gated Na⁺ channels open.
- ② Na⁺ inflow depolarizes the membrane and triggers the opening of still more Na⁺ channels, creating a positive feedback cycle and a rapidly rising membrane voltage.
- ③ Na⁺ channels close when the cell depolarizes, and the voltage peaks at nearly +30 mV.
- ④ Ca²⁺ entering through slow Ca²⁺ channels prolongs depolarization of membrane, creating a plateau. Plateau falls slightly because of some K⁺ leakage, but most K⁺ channels remain closed until end of plateau.
- ⑤ Ca²⁺ channels close and Ca²⁺ is transported out of cell. K⁺ channels open, and rapid K⁺ outflow returns membrane to its resting potential.

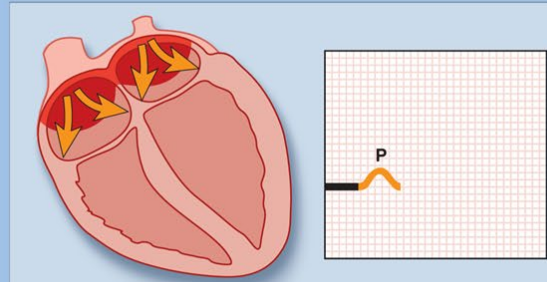


Electrical Activity of Myocardium

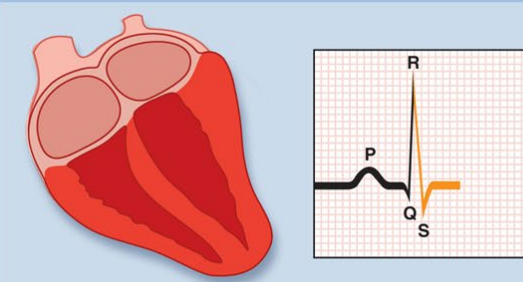
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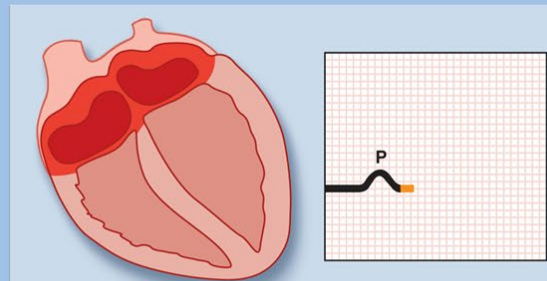
- 1) atrial depolarization begins
- 2) atrial depolarization complete (atria contracted)
- 3) ventricles begin to depolarize at apex; atria repolarize (atria relaxed)
- 4) ventricular depolarization complete (ventricles contracted)
- 5) ventricles begin to repolarize at apex
- 6) ventricular repolarization complete (ventricles relaxed)



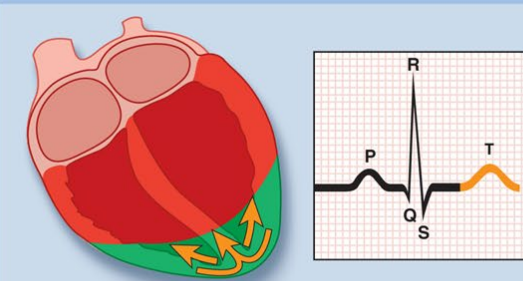
① Atria begin depolarizing.



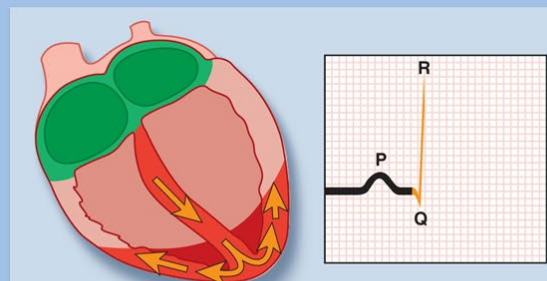
④ Ventricular depolarization complete.



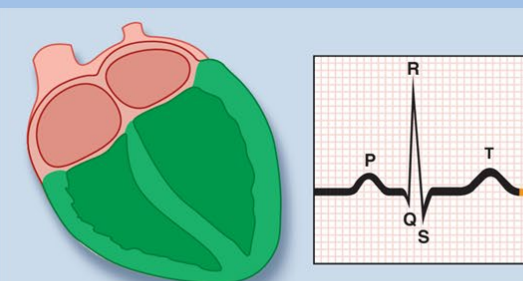
② Atrial depolarization complete.



⑤ Ventricular repolarization begins at apex and progresses superiorly.



③ Ventricular depolarization begins at apex and progresses superiorly as atria repolarize.



⑥ Ventricular repolarization complete; heart is ready for the next cycle.

What is fibrillation?

Fibrillation of the heart refers to an abnormal heart rhythm where the heart chambers contract irregularly and rapidly. There are two main types:

Atrial fibrillation (AFib): Irregular contractions in the upper chambers of the heart (atria).

Ventricular fibrillation (VFib): Irregular contractions in the lower chambers of the heart (ventricles).

Causes:

AFib:

- Heart valve disease
- High blood pressure
- Thyroid disease
- Lung disease
- Age

VFib:

- Heart attack
- Electrolyte imbalances
- Certain medications

Atrial Fibrillation



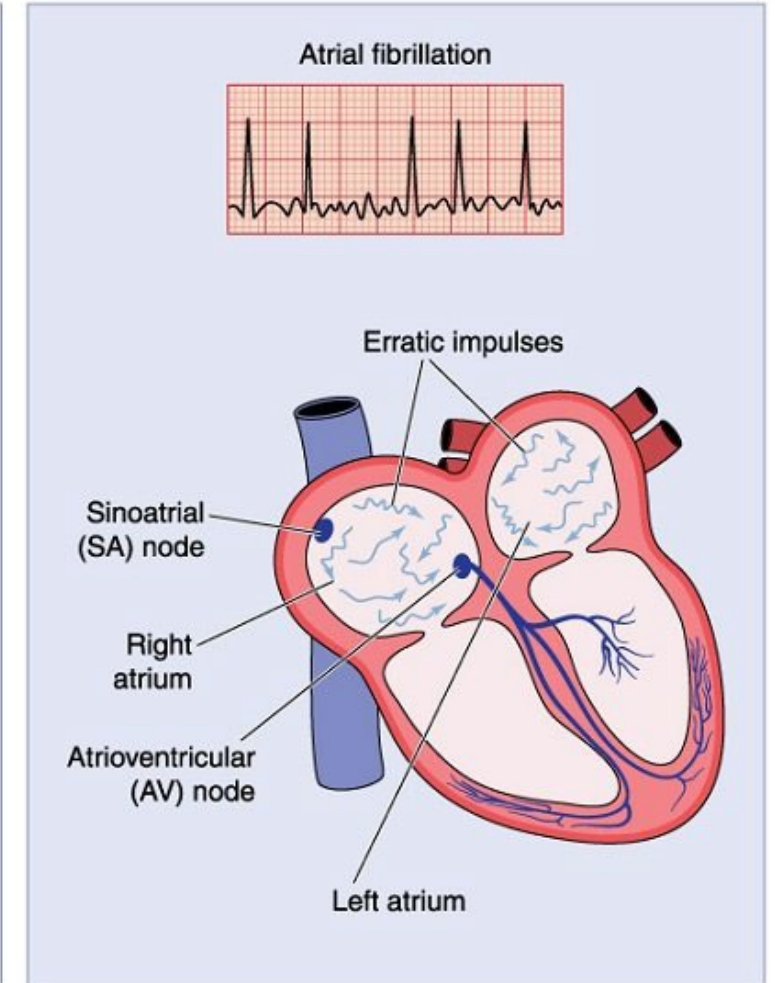
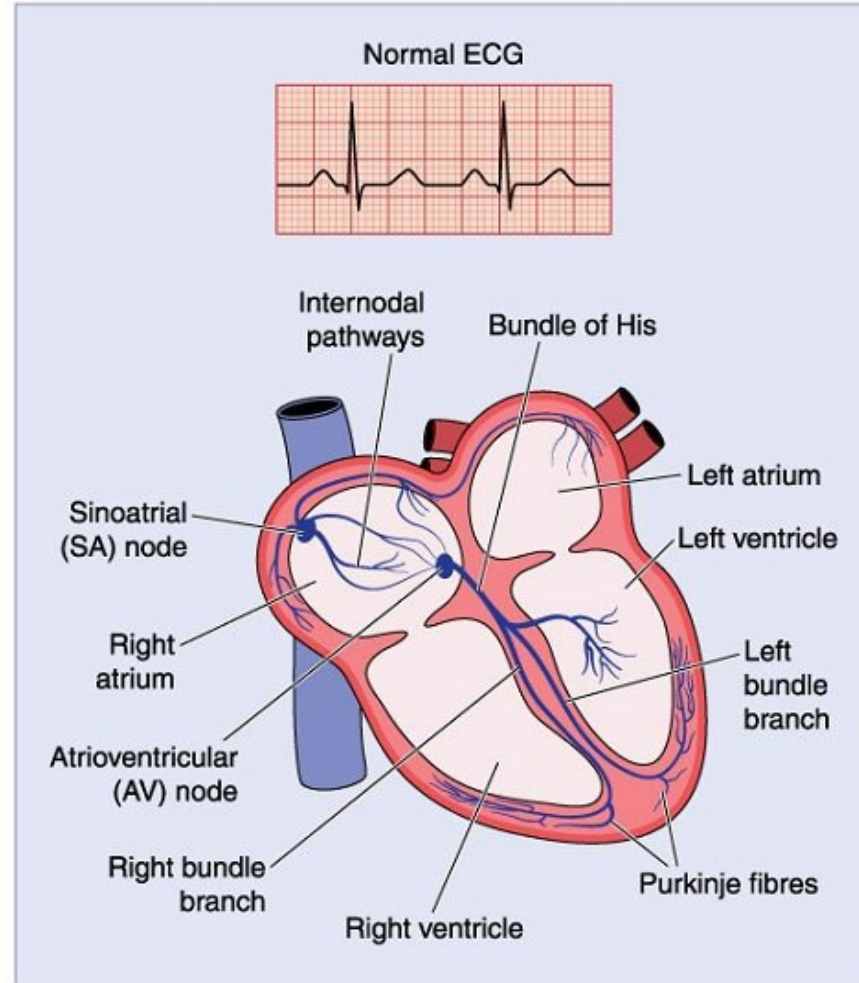
Atrial fibrillation // caused by ectopic foci in atria

occurs when atria beat 200 - 400 times per minute

atria unable to pump blood into ventricles // atria continues to “drop” blood into the ventricles

this may not be fatal

ventricles will still fill with blood // passive ventricular filling



Ventricular Fibrillation



Ventricular fibrillation // serious arrhythmia caused by many electrical signals reaching different regions of ventricles at widely different times

heart can't pump blood into systemic circuit
plus

no coronary circuit // no perfusion

will kill quickly if not stopped

defibrillation - strong electrical shock through heart // intent is to depolarize all myocytes at same time // stop the fibrillation

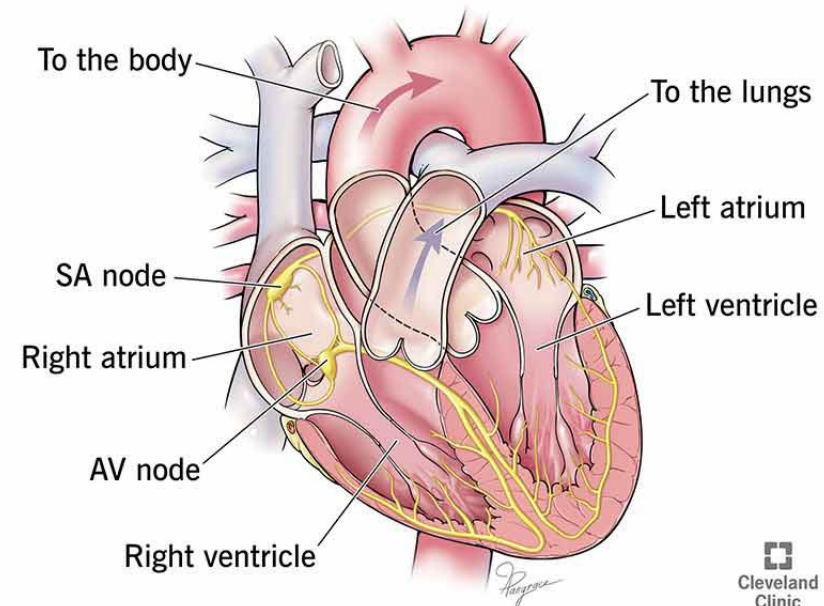
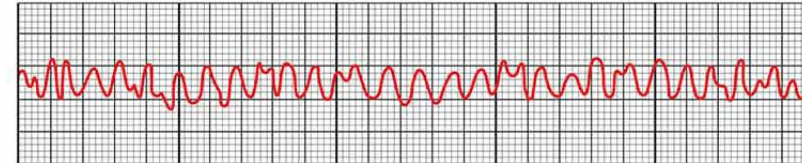
hope to reset the normal SA node function and sinus rhythm

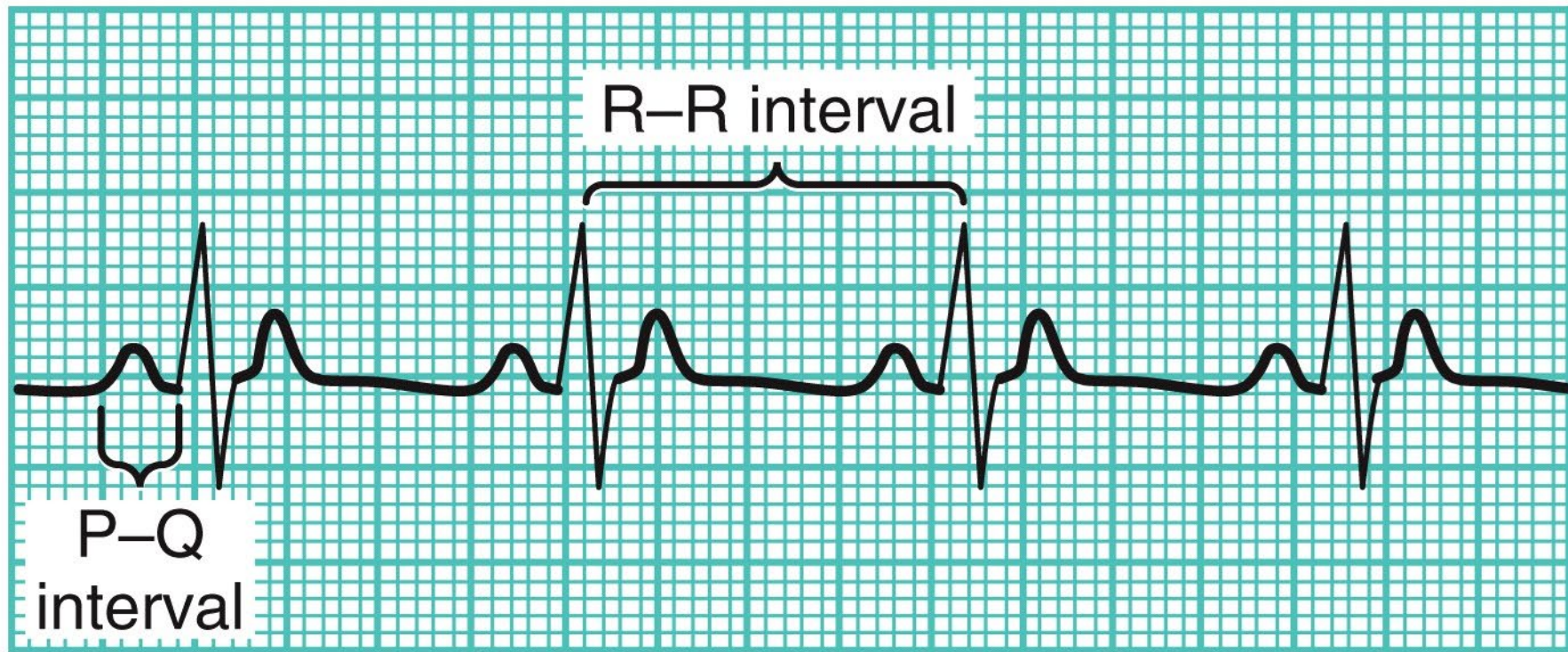
Ventricular arrhythmia

Normal sinus rhythm



Ventricular arrhythmia (fibrillation shown)

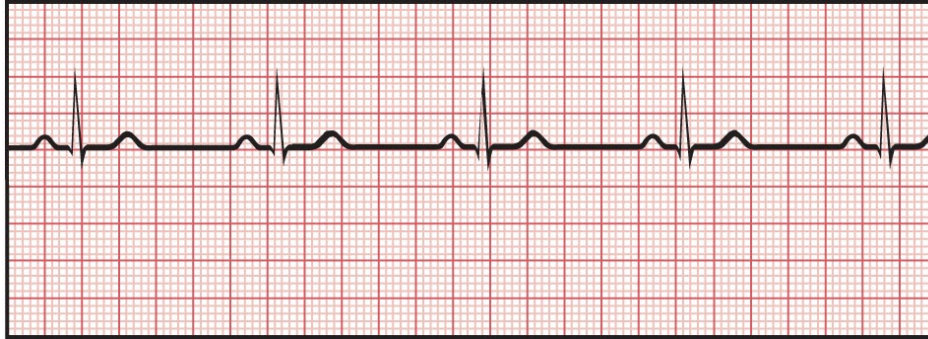




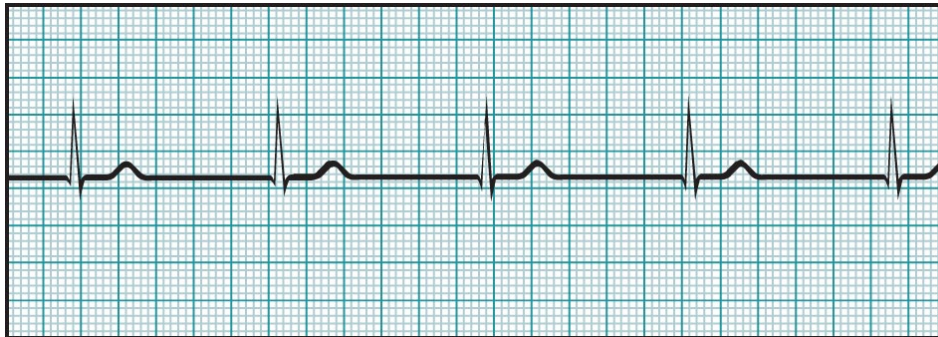
(a) Normal electrocardiogram (ECG)

ECGs: Normal and Abnormal

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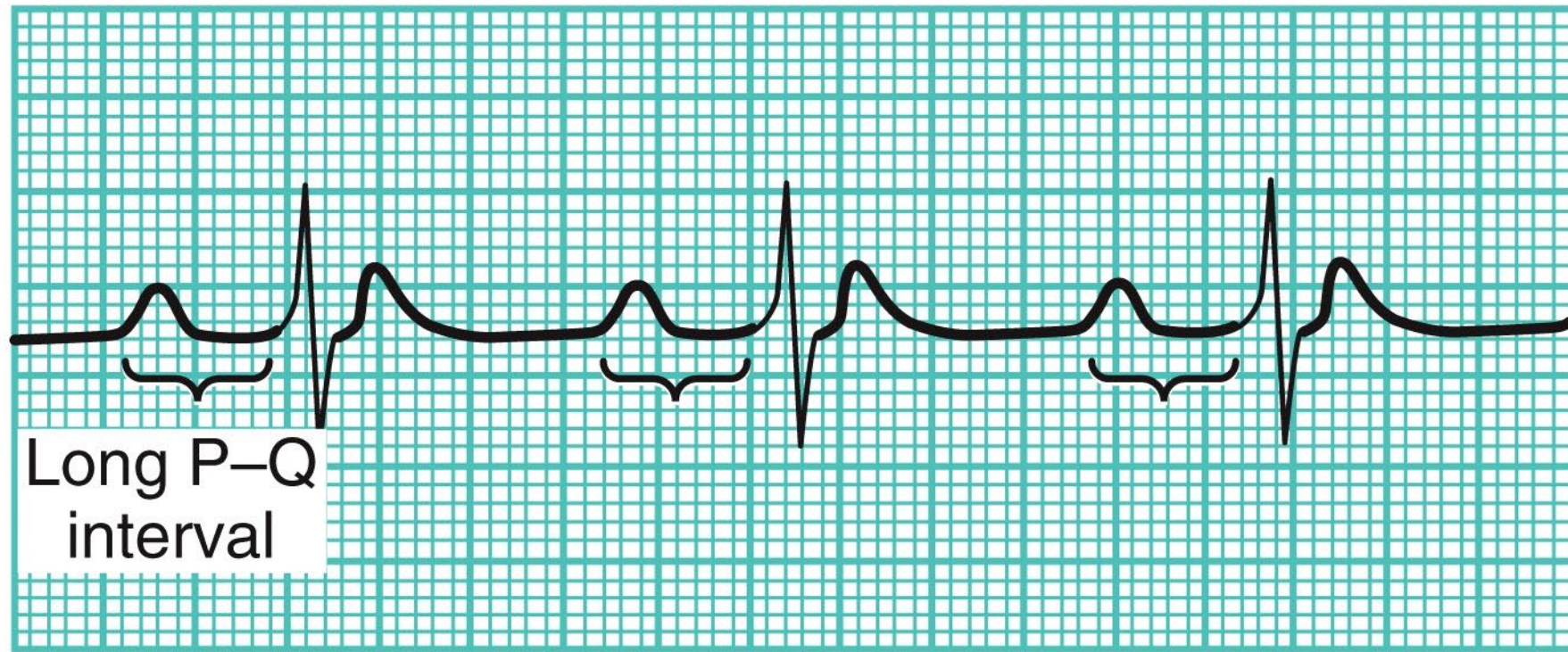


(a) Sinus rhythm (normal)

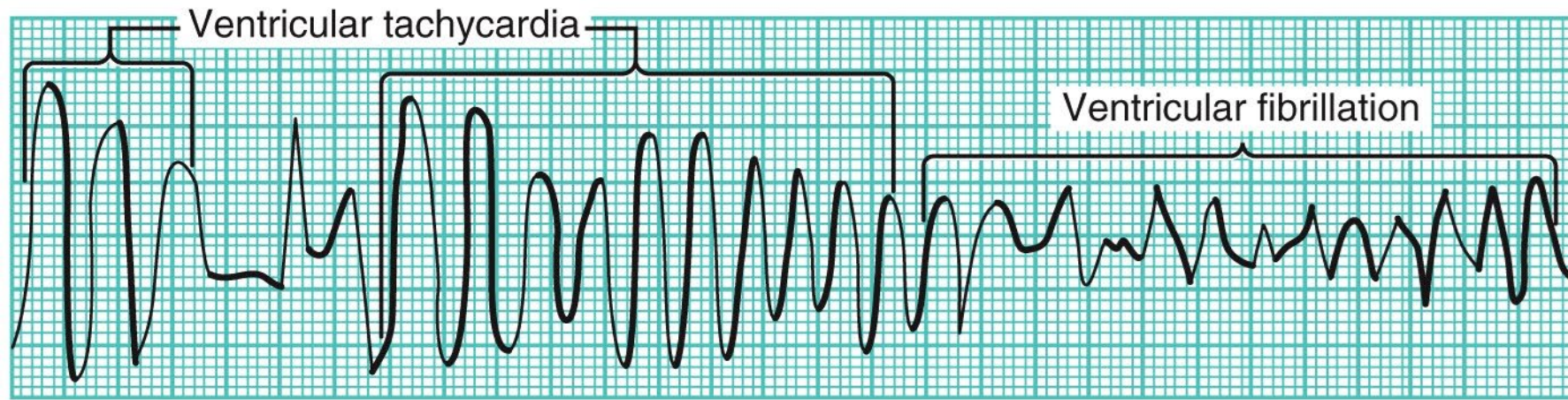


(b) Nodal rhythm—no SA node activity

- abnormalities in conduction pathways
- myocardial infarction
- heart enlargement
- electrolyte and hormone imbalances

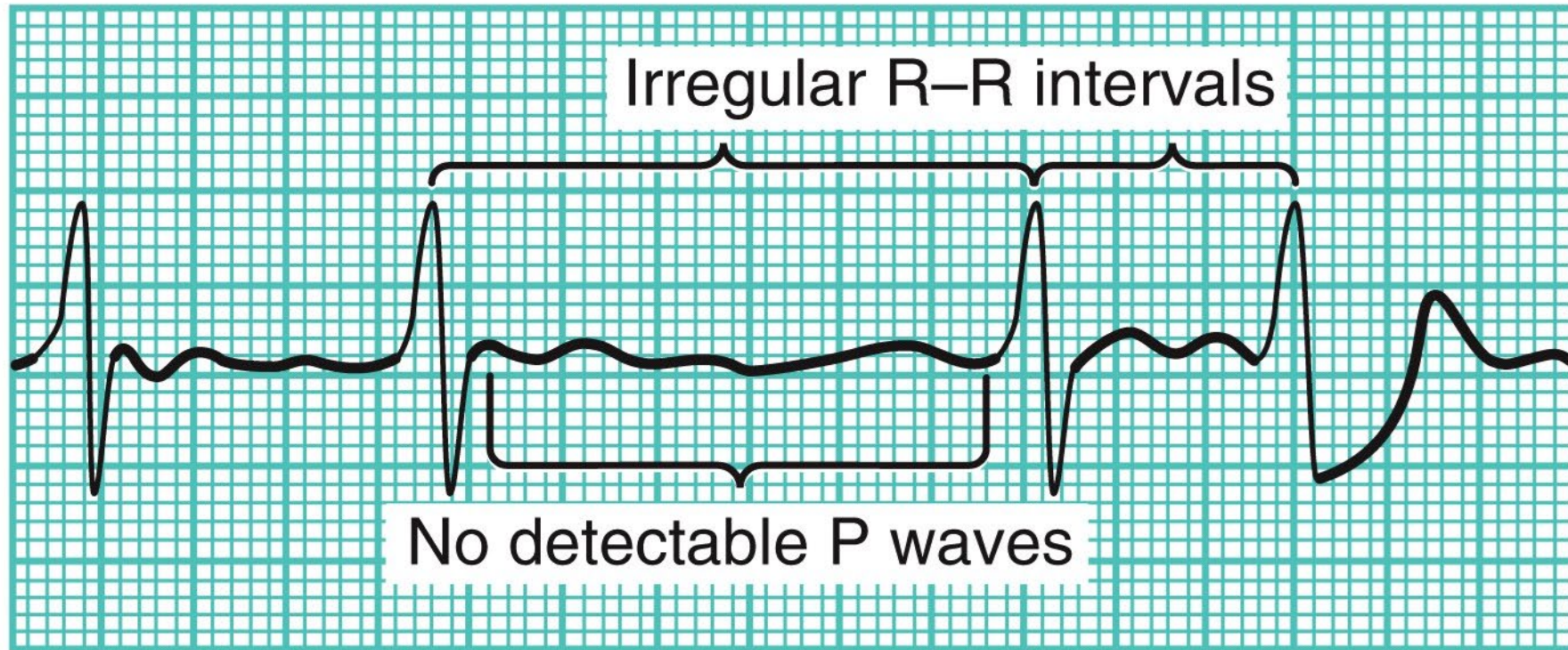


(b) First-degree AV block



(d) Ventricular tachycardia

(e) Ventricular fibrillation



(c) Atrial fibrillation

Diagnostic Value of ECG

- abnormalities in conduction pathways
- myocardial infarction
- nodal damage
- heart enlargement
- electrolyte and hormone imbalances