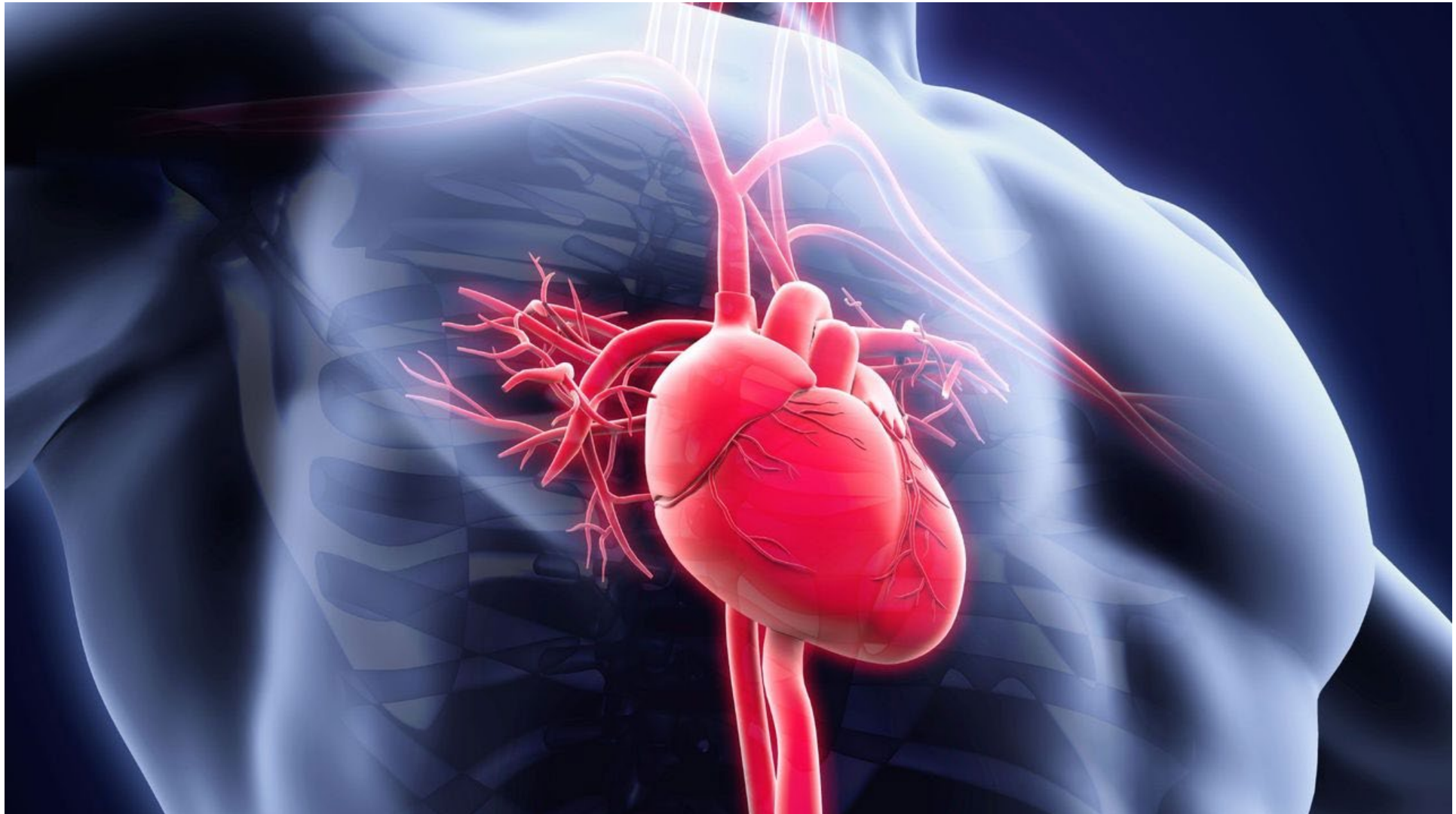
Chapter 19.2

# The Heart's Electrical Conduction System



# Intrinsic Cardiac Conduction System

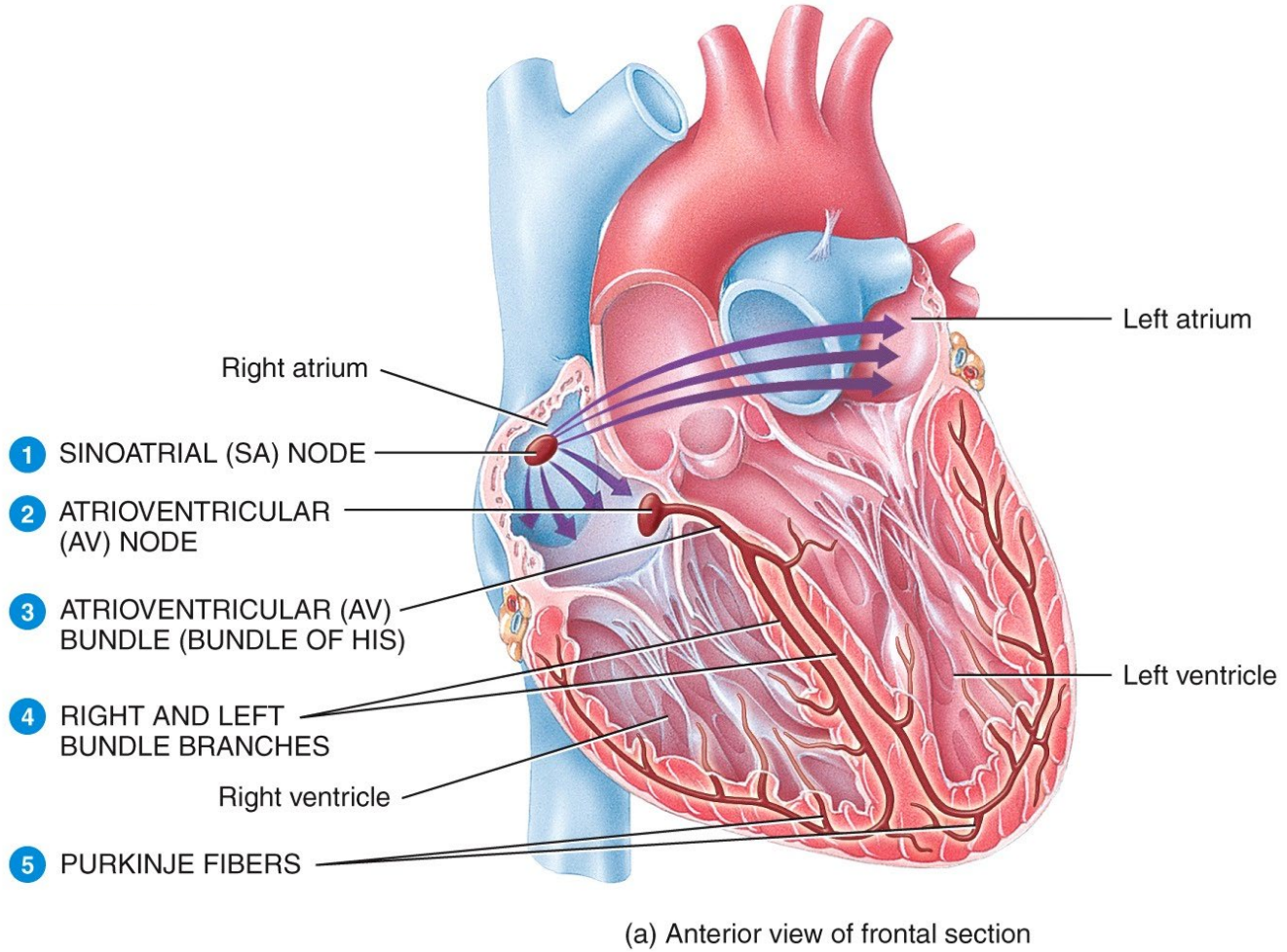
The ICCS coordinate the sequential contraction of the heart chambers during the cardiac cycle

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 truck and aorta

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



(a) Anterior view of frontal section

*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 note

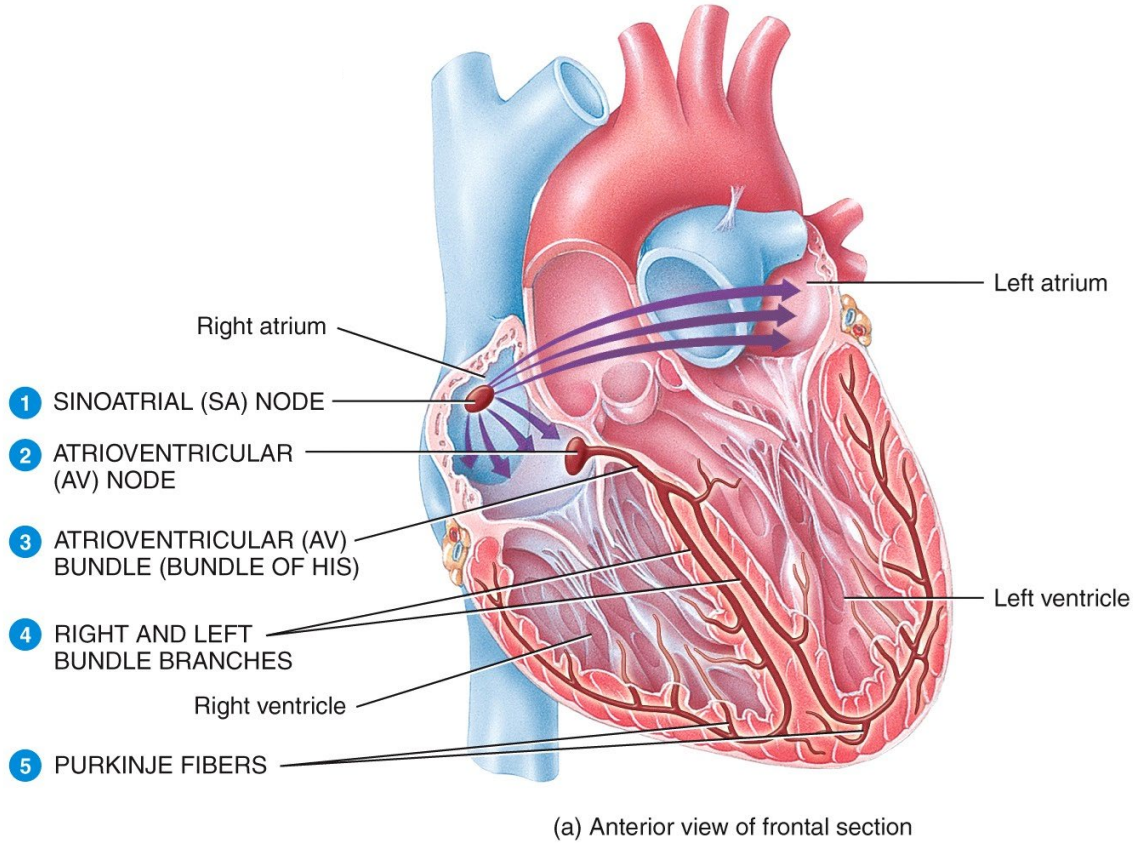
# Cardiac Conduction System

Atrioventricular (AV) bundle (bundle of His)

– bundle forks into right and left bundle branches
– branches pass through interventricular septum
– move towards apex

Purkinje fibers

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



(a) Anterior view of frontal section

# Nodal Tissue and Nerve Supply of the Heart

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

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

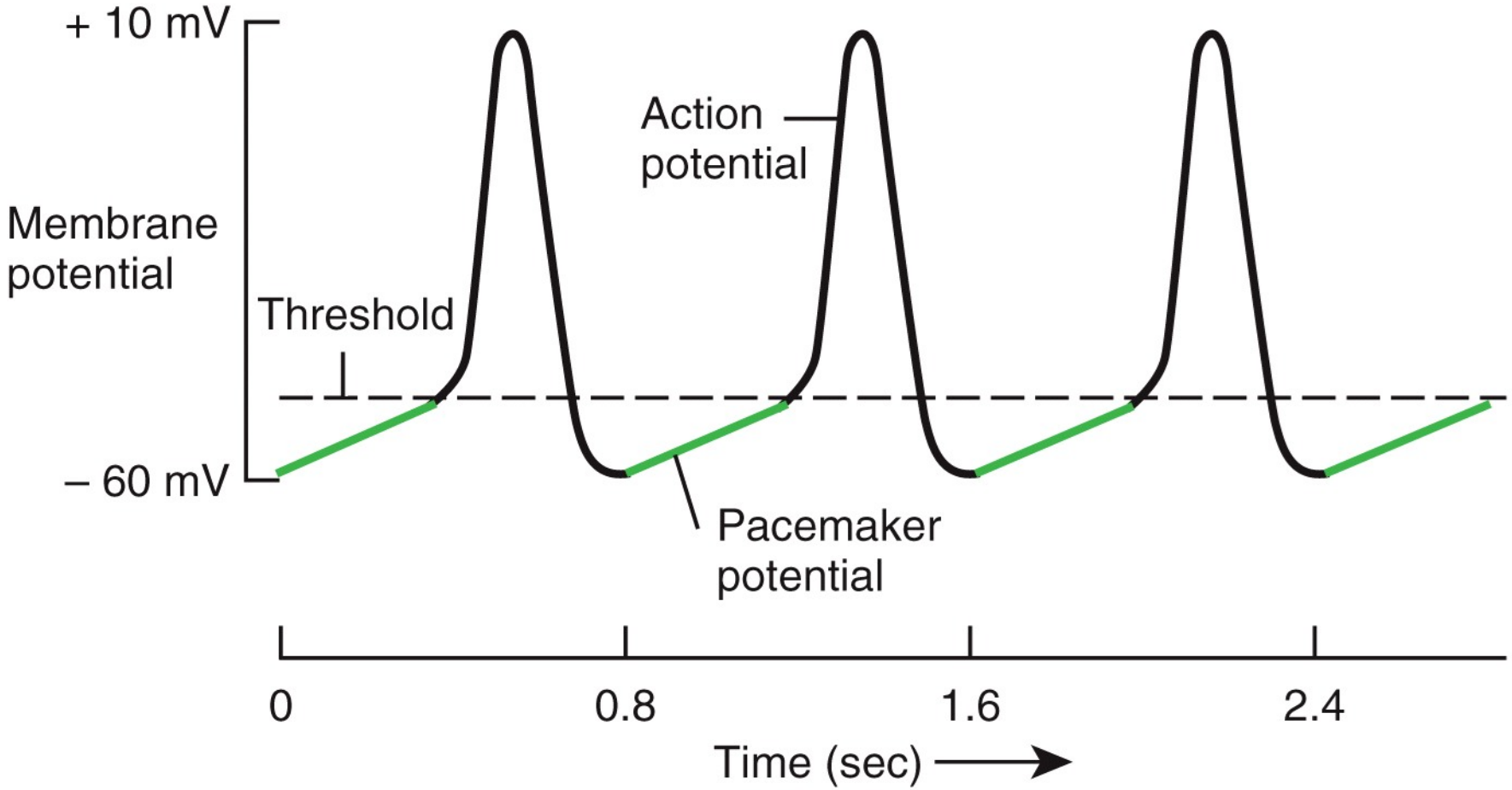
Sinoatrial Node (SA node) and Atrioventricular Node (AV node) are two area that "leak sodium ions" faster than any other myocaridocytes // SA leaks fastest so it sets the rate for the heart

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

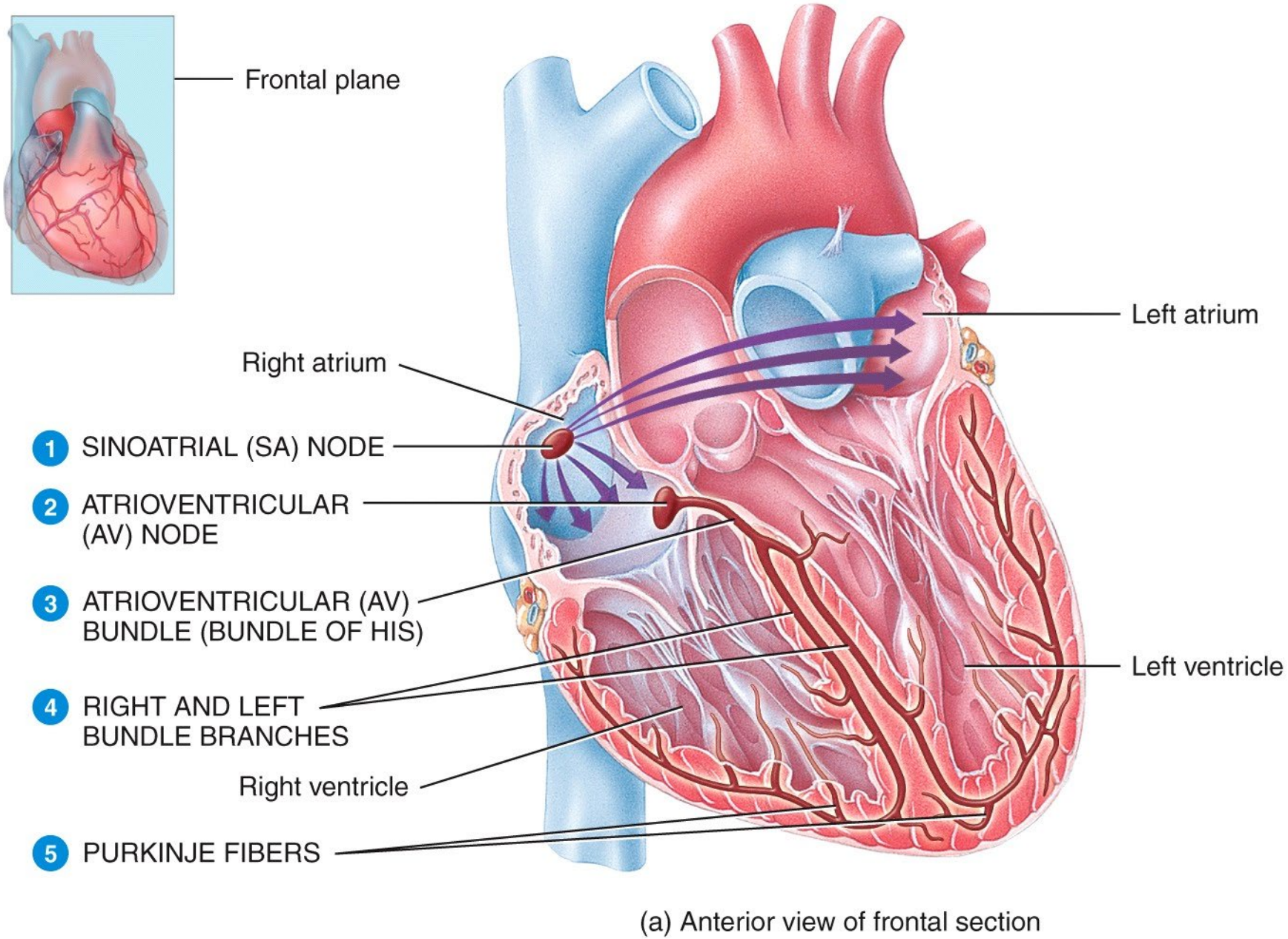
Any myocardiocyte removed from the heart may be used to demonstrate the cardiocyte inability to maintain a resting membrane potential

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




(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 contact*

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

Cardiocytes at AV node have fewer gap junctions then 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

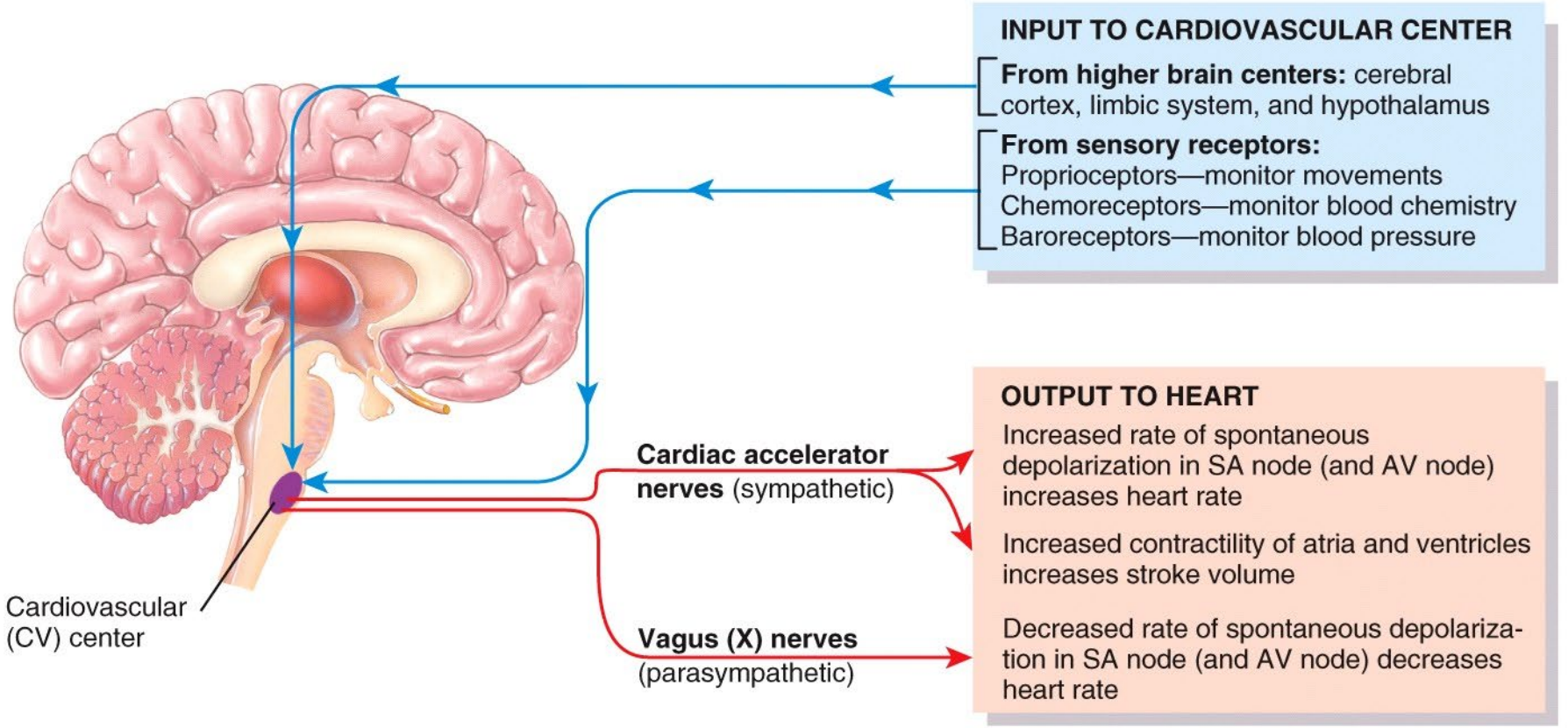
Papillary muscles contract an instant earlier than the ventricular myocardiocytes /// tightening the slack in chordae tendineae

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

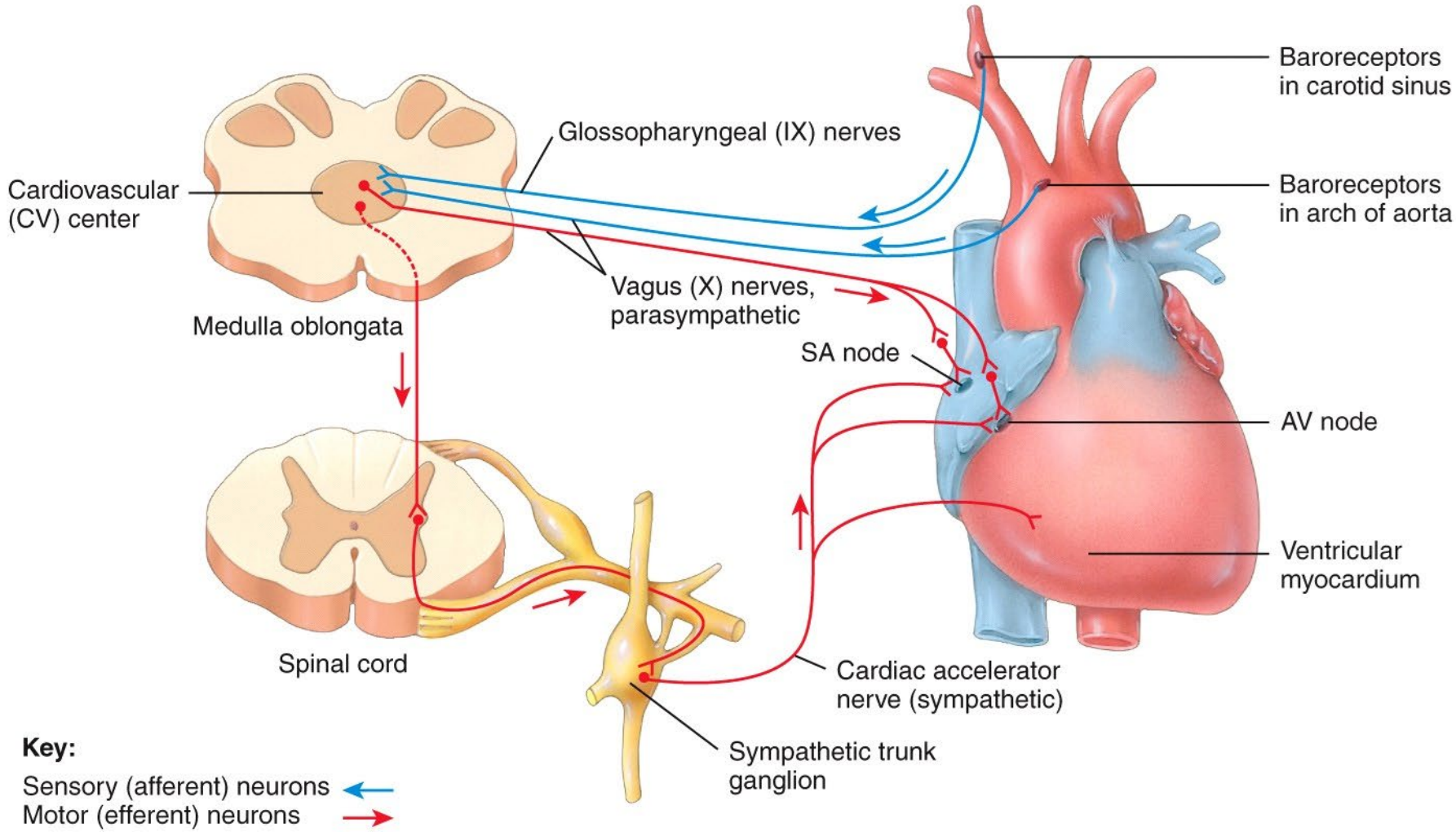
Spiral arrangement of cardiocytes twists ventricles slightly like someone wringing out a towel

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!


Baroreceptors in carotid sinus
Glossopharyngeal (IX) nerves
Cardiovascular (CV) center
Baroreceptors in arch of aorta
Vagus (X) nerves, parasympathetic
Medulla oblongata
SA node
AV node
Ventricular myocardium
Spinal cord
Cardiac accelerator nerve (sympathetic)
Sympathetic trunk ganglion
Key:
Sensory (afferent) neurons
Motor (efferent) neurons

# 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 /// chemorecptors and baroreceptors

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 call 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

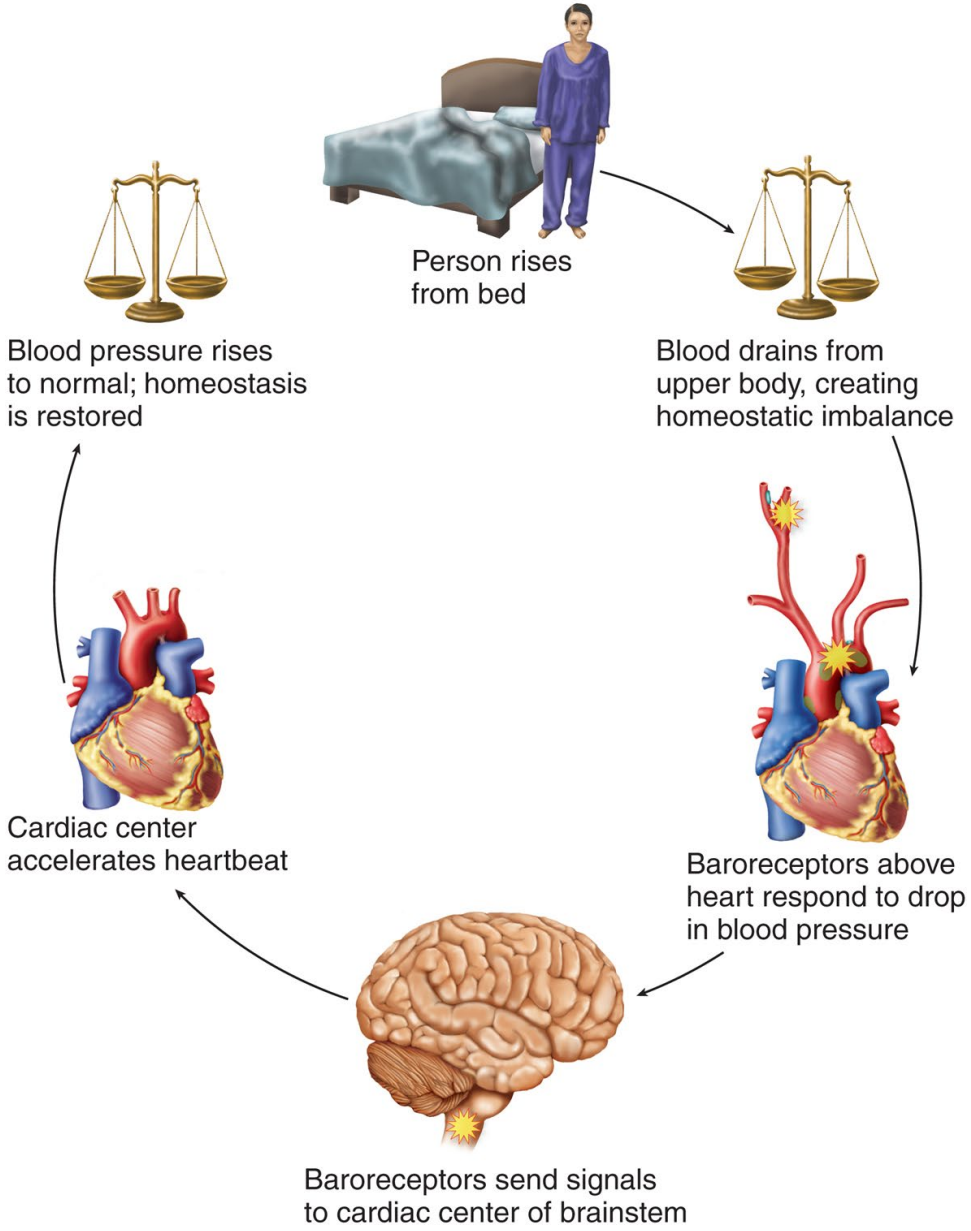
Pressure sensors (called sinuses) located: in aortic arch & internal carotid arteries

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





# Regulation (Inputs) into Cardiac Center

Chemoreceptors (three locations):

- aortic arch
- carotid arteries
- medulla oblongata
- Sensitive to blood pH, $CO_2$ and $O_2$ levels

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

If $CO_2$ accumulates in blood or CSF (hypercapnia), reacts with water and causes increase in $H^+$ levels

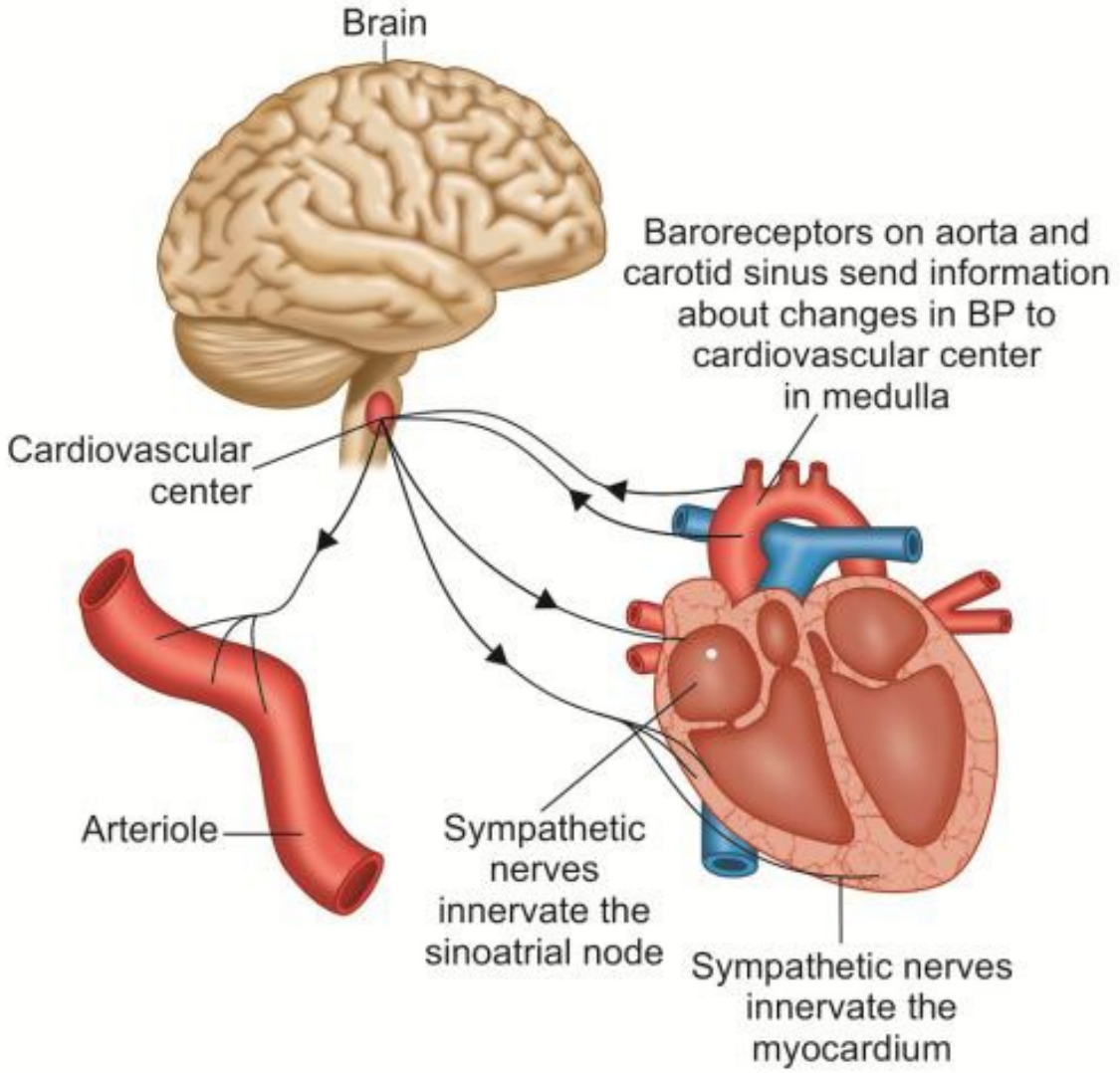
$H^+$ lowers the pH of the blood possibly creating acidosis (pH < 7.35)

# Regulation (Inputs) into Cardiac Center

Hypercapnia (high $CO_2$) 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

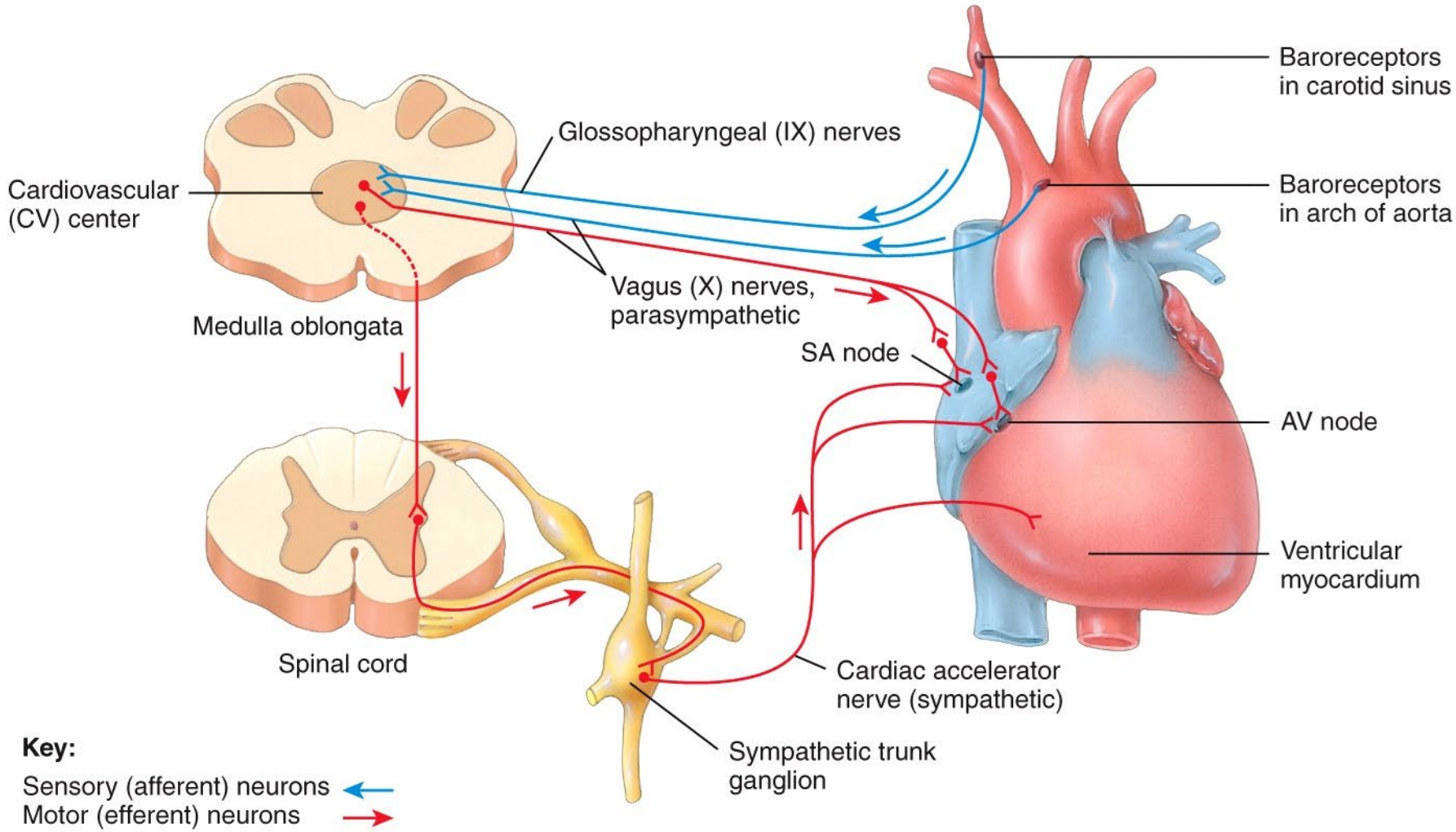
Sympathetic nerves

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

Continues to adjacent sympathetic chain ganglia

Come pass through cardiac plexus in mediastinum

Continue as cardiac nerves to the heart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SA node
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Ventricular myocardium
Spinal cord
Cardiac accelerator nerve (sympathetic)
Sympathetic trunk ganglion
Key:
Sensory (afferent) neurons
Motor (efferent) neurons

# 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
- Synapse on myocardiocytes and nodal cells
- G protein activates enzymes that open plasma channels
- Allow calcium to enter cell // this increases depolarization rate
- cAMP also increase uptake of calcium back into sarcoplasmic reticulumn
- 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

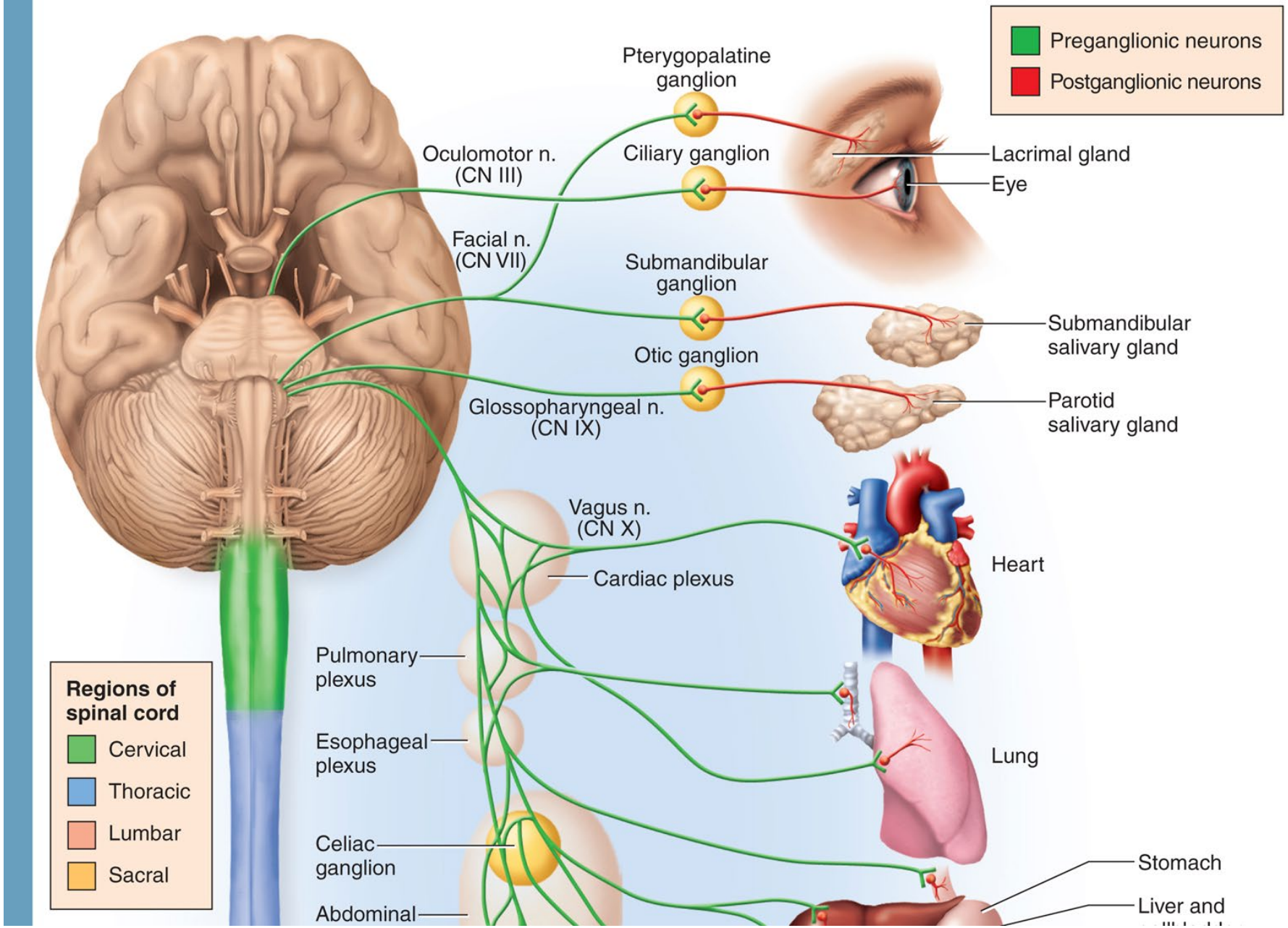
The myocardiocytes do not use ionotropic receptors!

Myocardiocytes use muscurinic 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????

# Cardiac Rhythms / Terminology / Nodal Rhythm

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

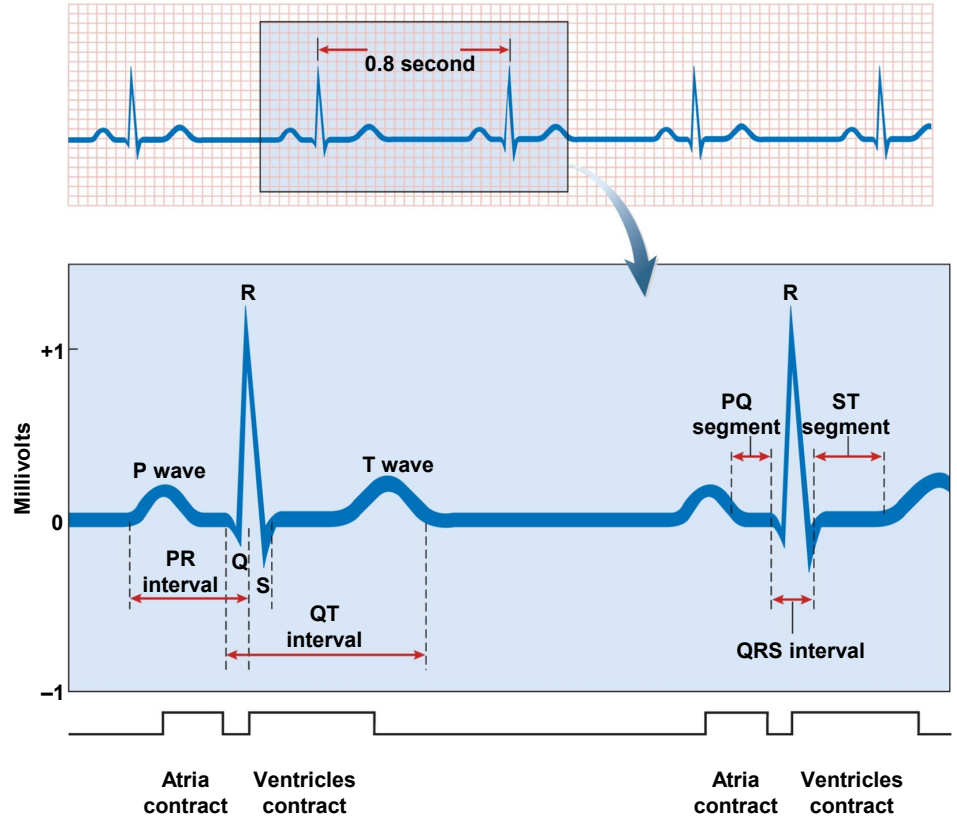
Arrhythmia // any abnormal cardiac rhythm

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

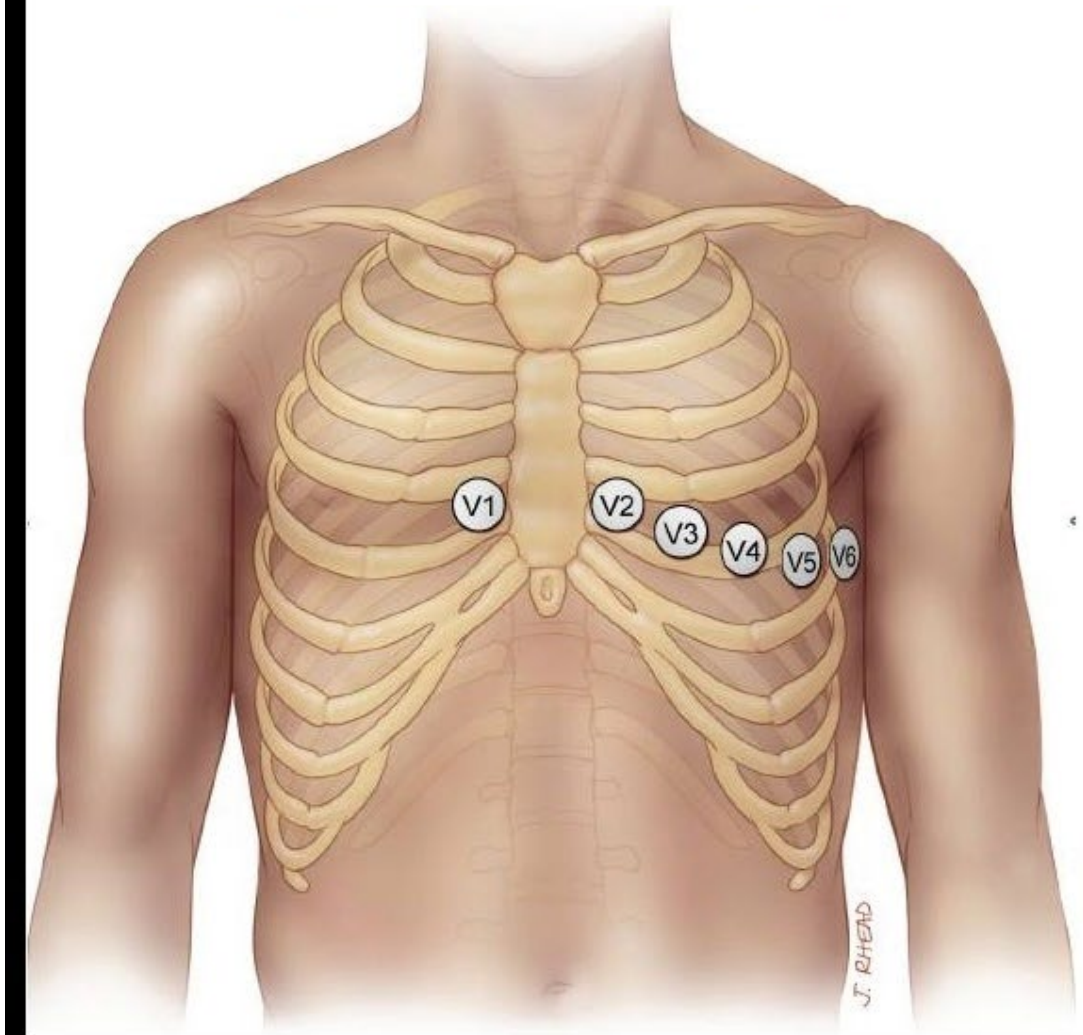
Total heart block // occurs because damage to AV node /// Action potential fails to pass AV node

# Electrocardiogram (ECG or EKG)

- 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
- The composite recording  is associated with events in cardiac cycle



# Proper Placement of Six 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

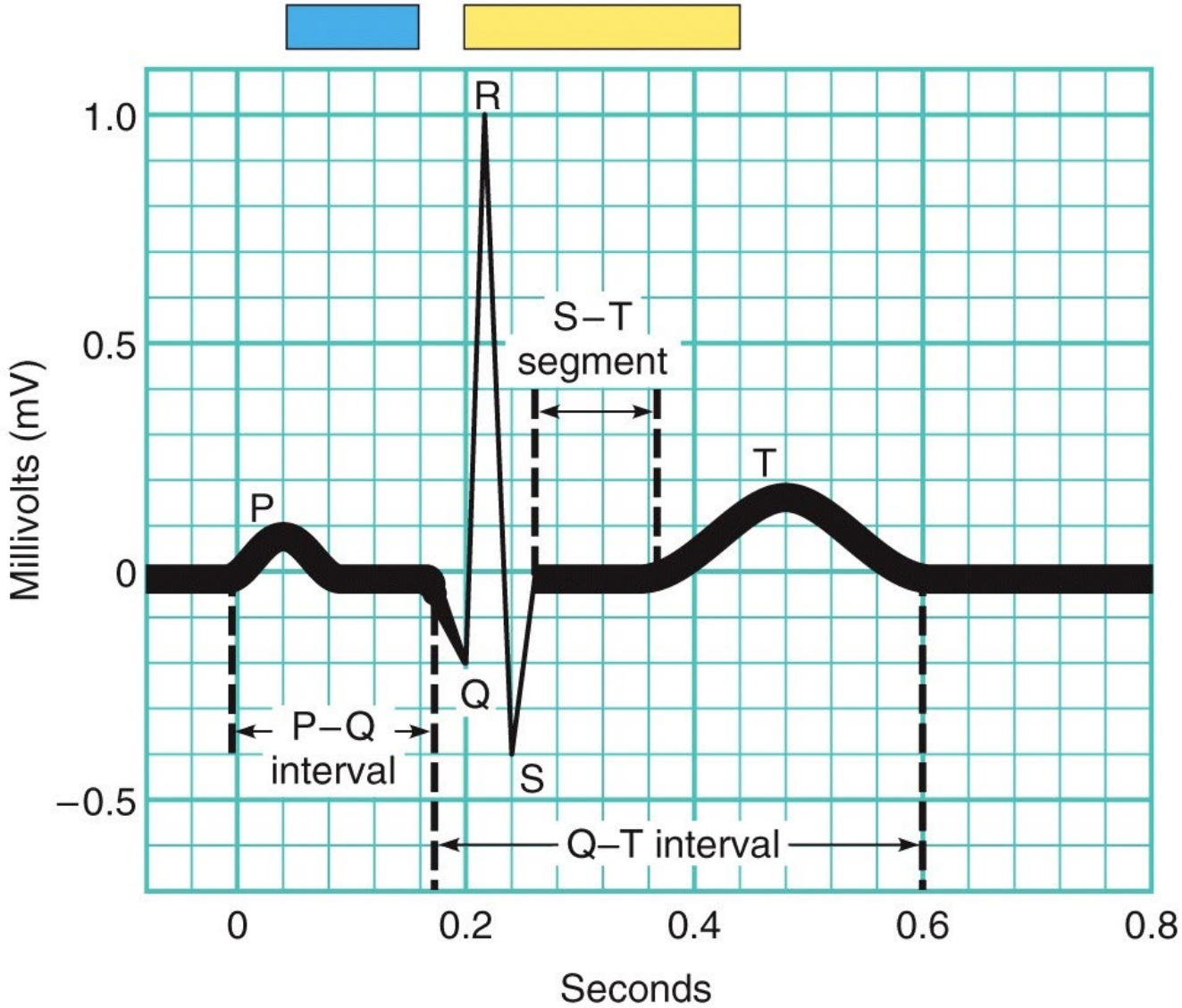
**V6** Midaxillary line at the same level as V4 and V5

**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

– follows atria depolarization

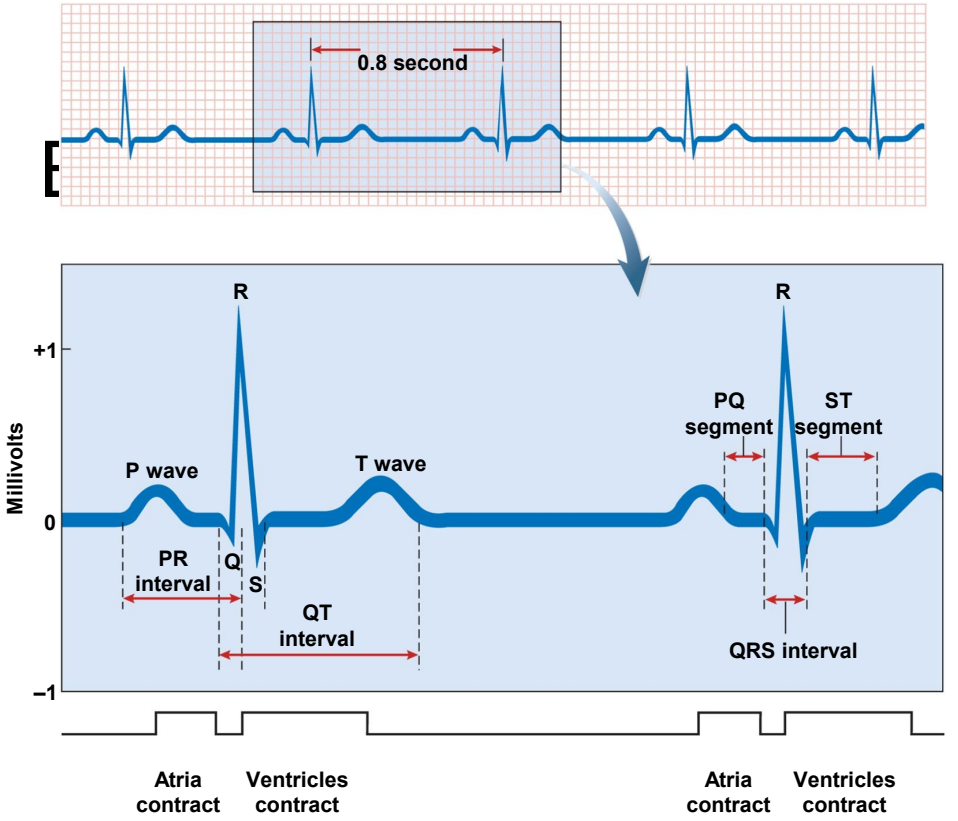
– period of atrial systole (systole is the actual contraction)
– atrial systole begins 100 msec after SA depolarization

QRS complex
– ventricular depolarization
– complex shape due to different thickness/shape of 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

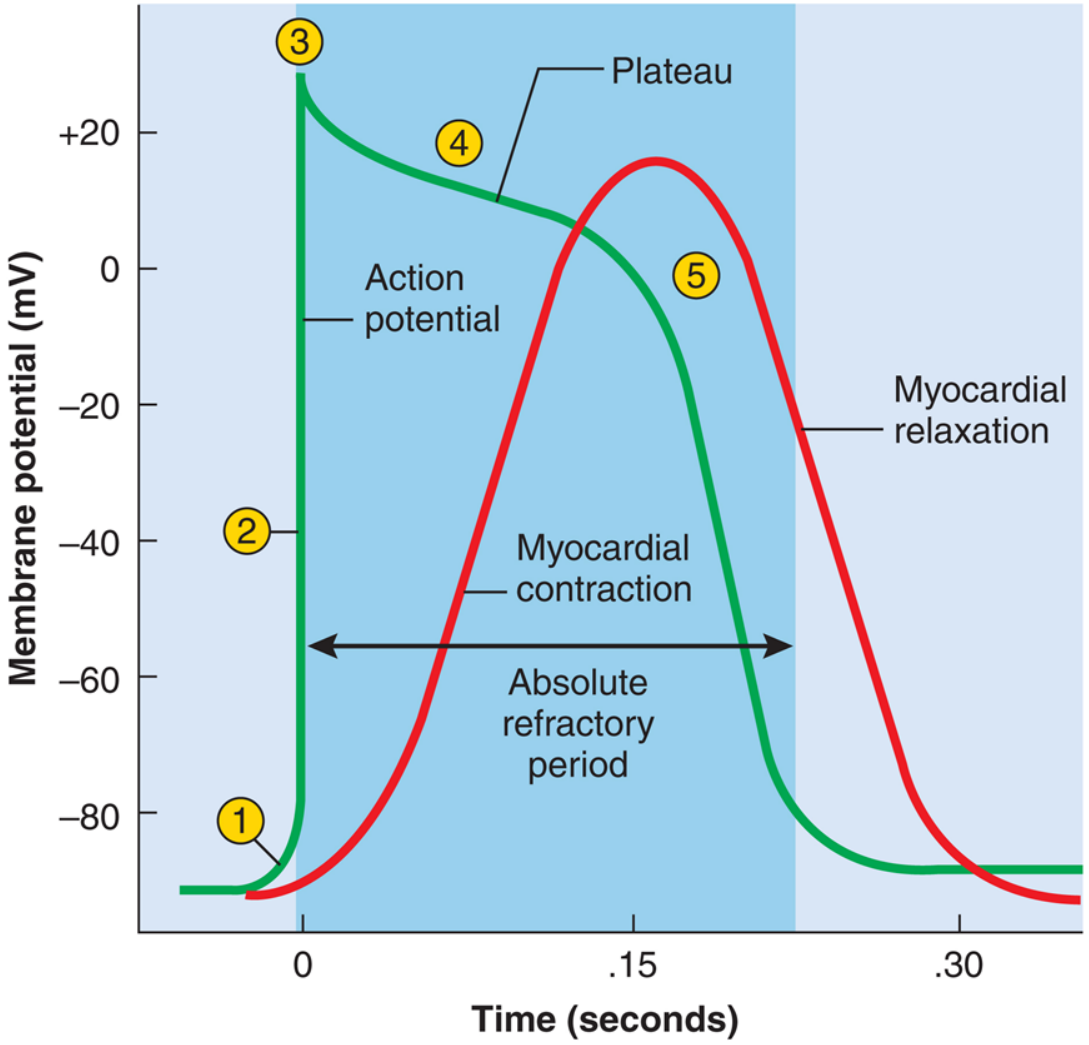
# Electrocardiogram



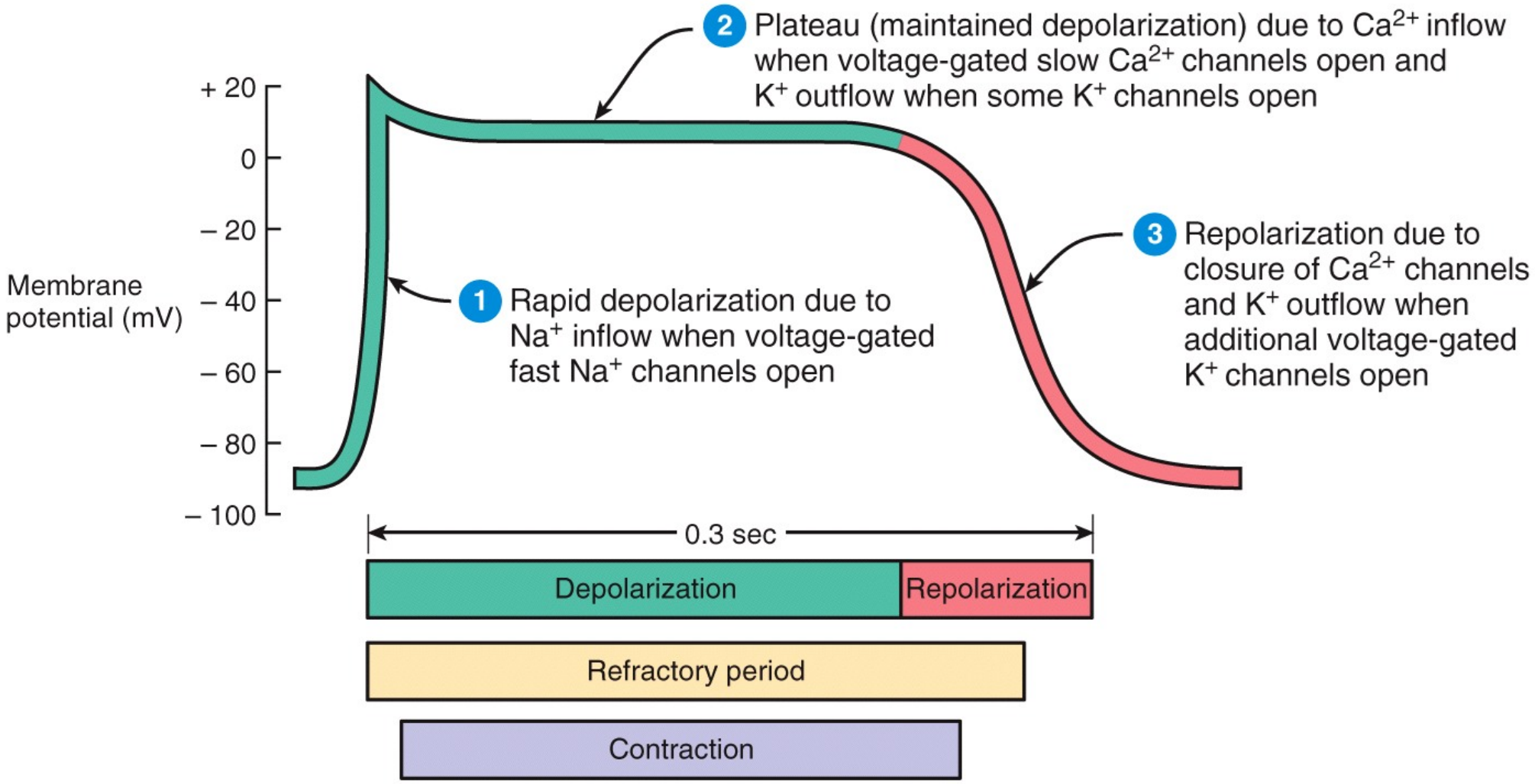

Note: asystole = “flat line” / no contraction of myocardium / requires cardiopulmonary resuscitation (CPR)

# Action Potential of Myocardiocyte






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. $Ca^{2+}$ entering through slow $Ca^{2+}$ 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. $Ca^{2+}$ channels close and $Ca^{2+}$ is transported out of cell. $K^+$ channels open, and rapid $K^+$ outflow returns membrane to its resting potential.




Note the extended “contraction” due to calcium inflow from interstitial space.

# Electrical Activity of Myocardium



1) atrial depolarization begins

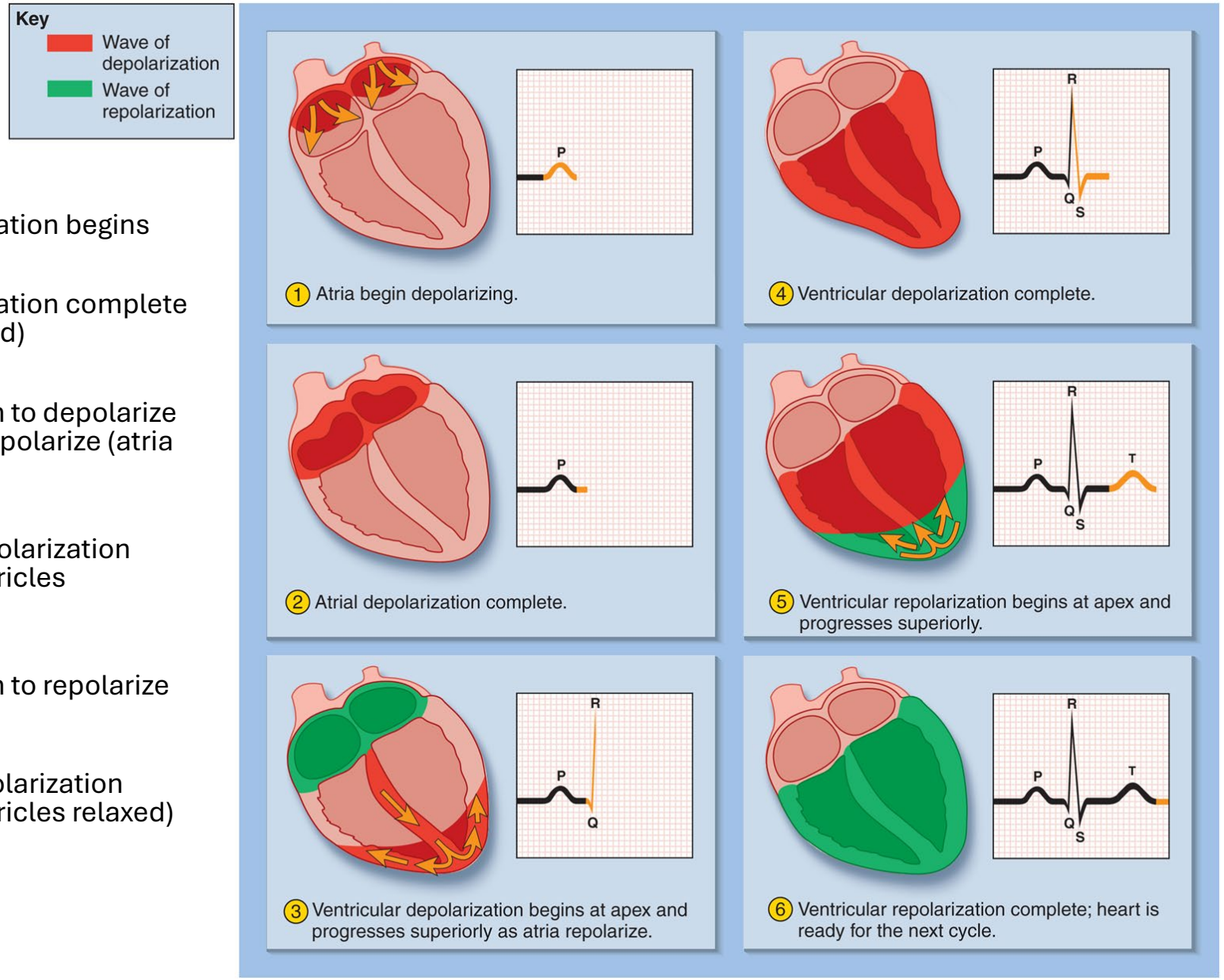
2) atrial depolarization complete (atria contracted)

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)



# 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

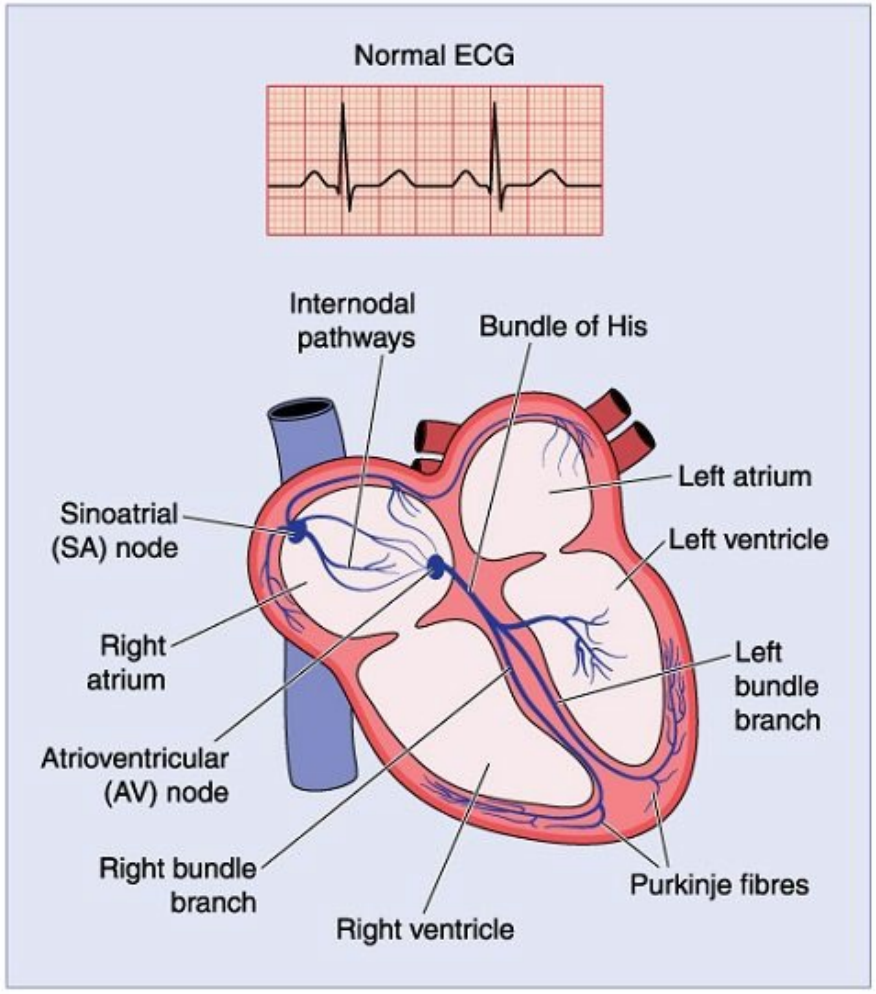
Atrial fibrillation // caused by ectopic foci in atria

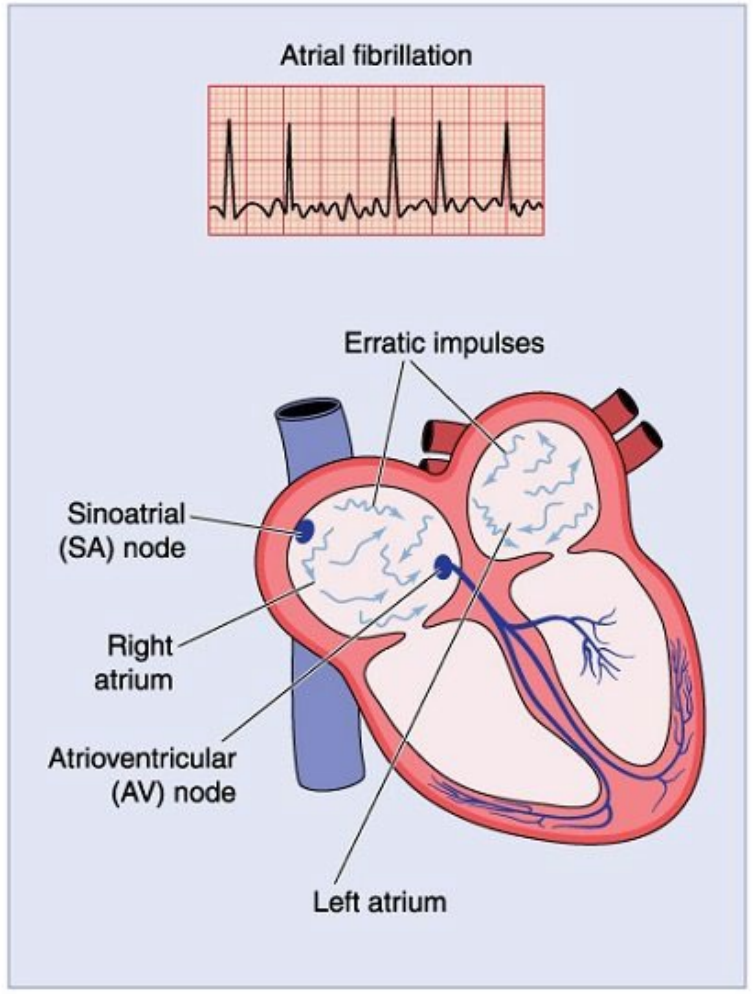
Occurs when atria beat <u>200 - 400 times per minute</u>

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

<u>This may not be fatal but associated with tachycardia and right heart failure.</u>

<u>Ventricles will still fill with blood // incomplete filling by passive ventricular filling</u>



# Ventricular Fibrillation

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

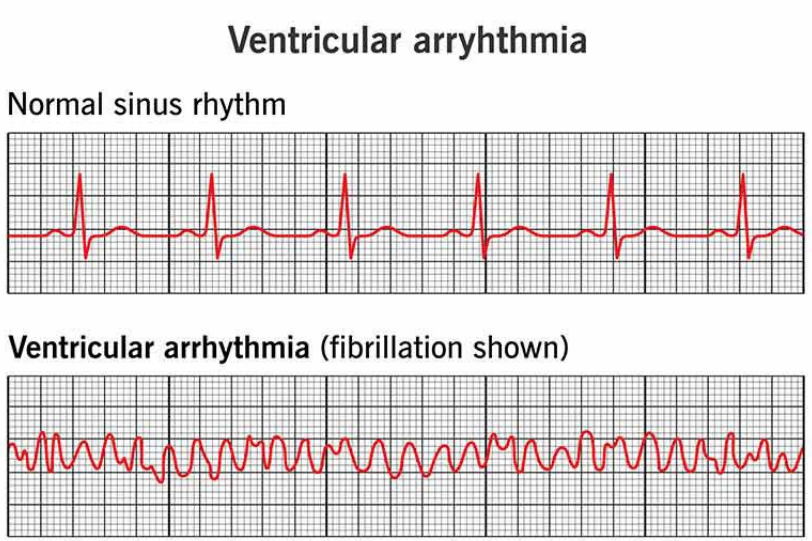
Heart can't pump blood into systemic circuit plus

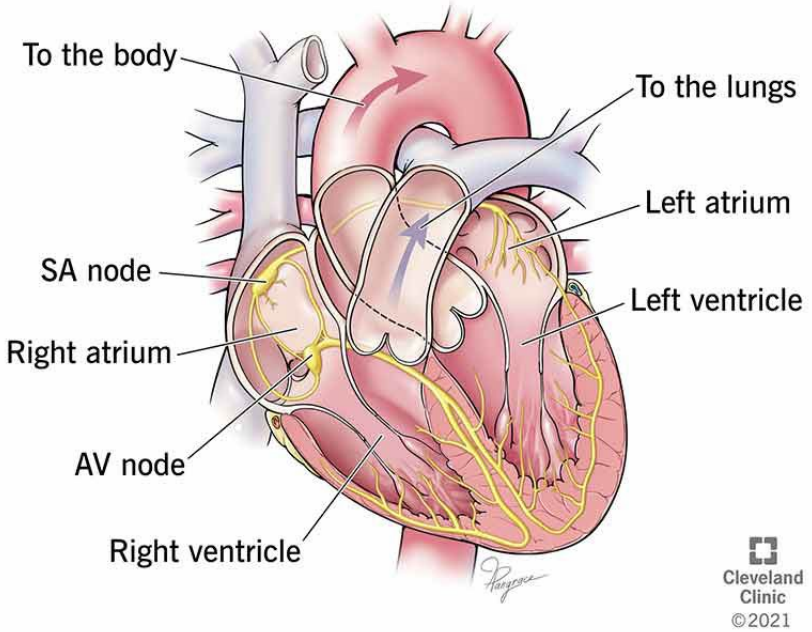
No coronary circuit // no perfusion

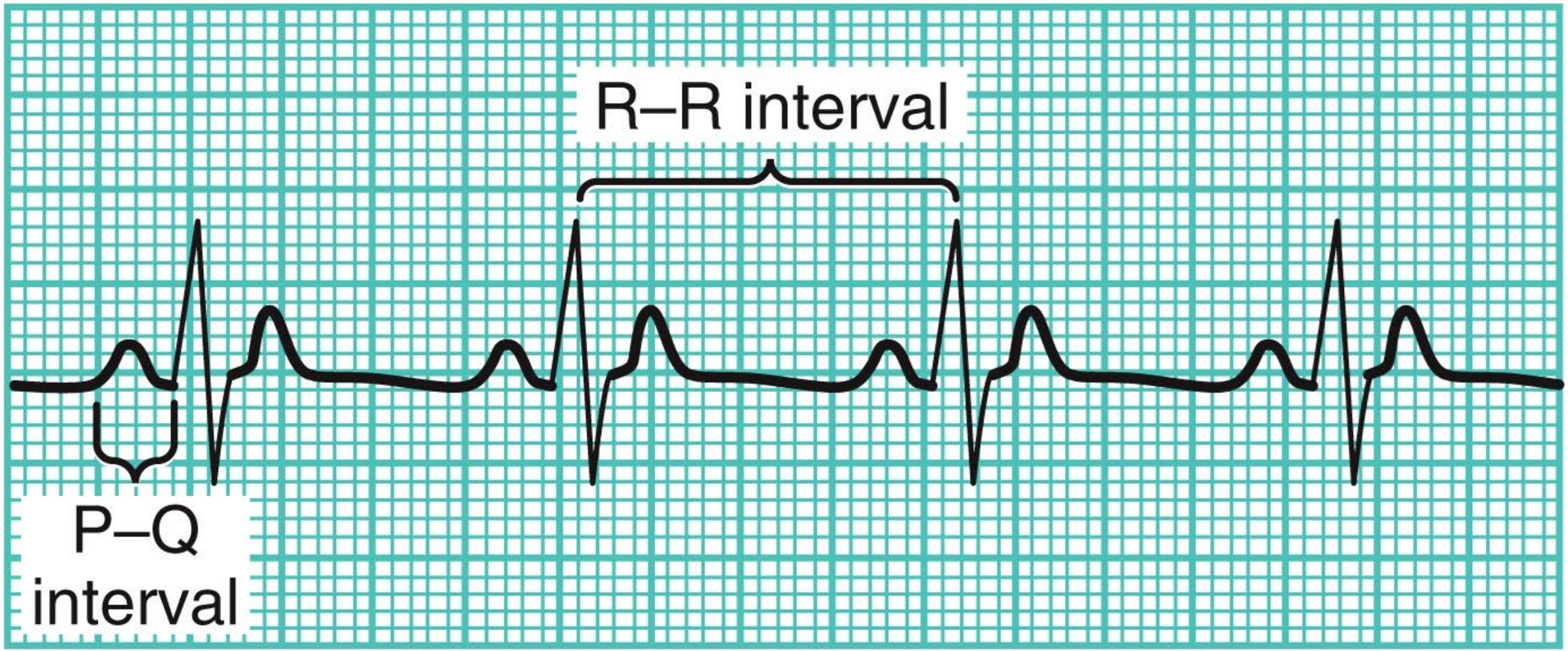
Will kill quickly if not stopped immediately

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

The hope is to reset the normal SA node function and sinus rhythm





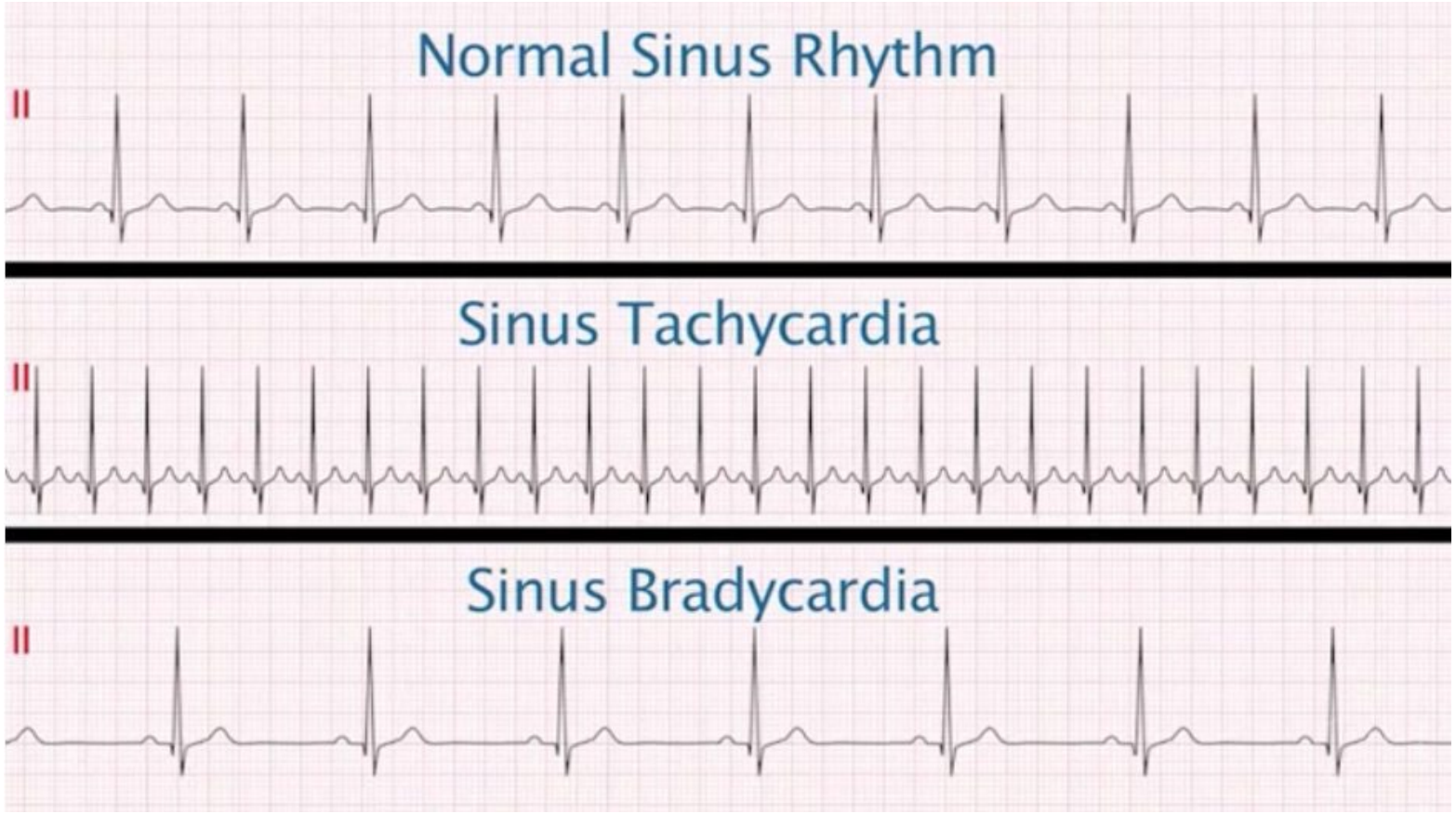

(a) Normal electrocardiogram (ECG)

# Cardiac Rhythms / Terminology

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

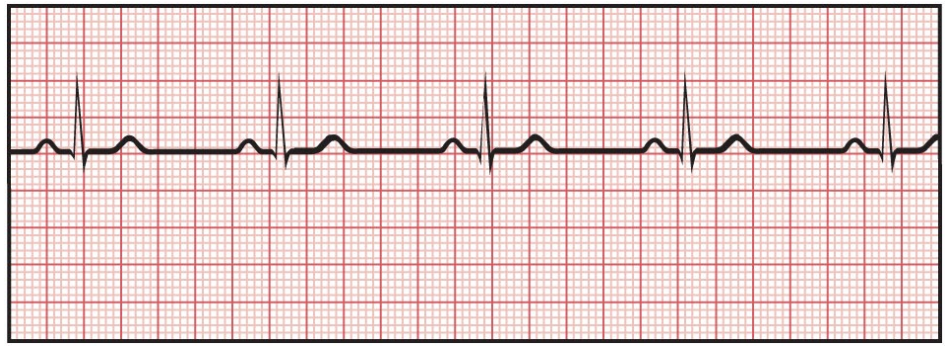
Tachycardia // Persistent resting adult heart rate above 100 bpm

Bradycardia // Persistent resting adult heart rate below 60 bpm

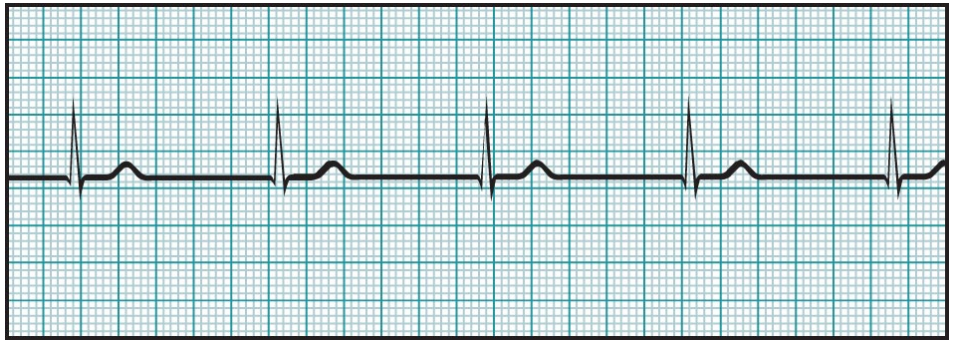


# ECGs: Normal and Abnormal



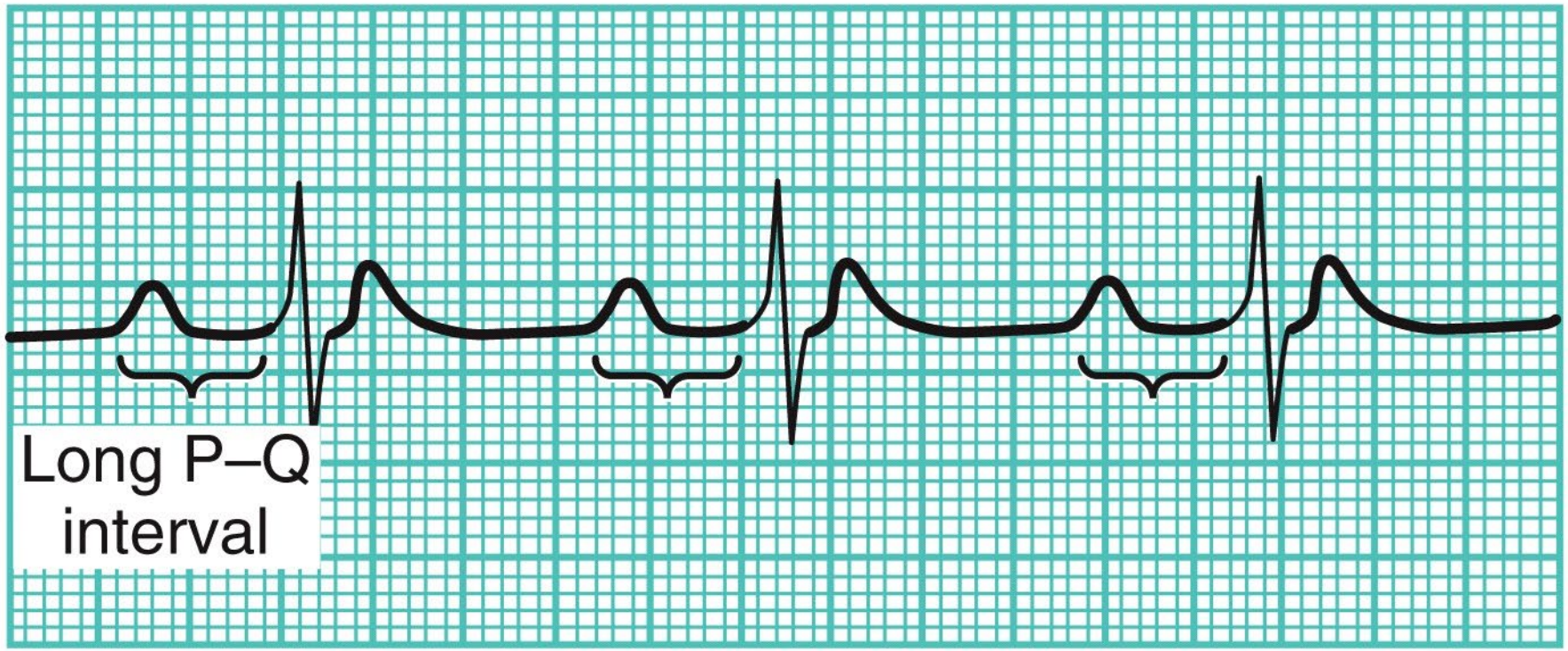


**(a) Sinus rhythm (normal)**

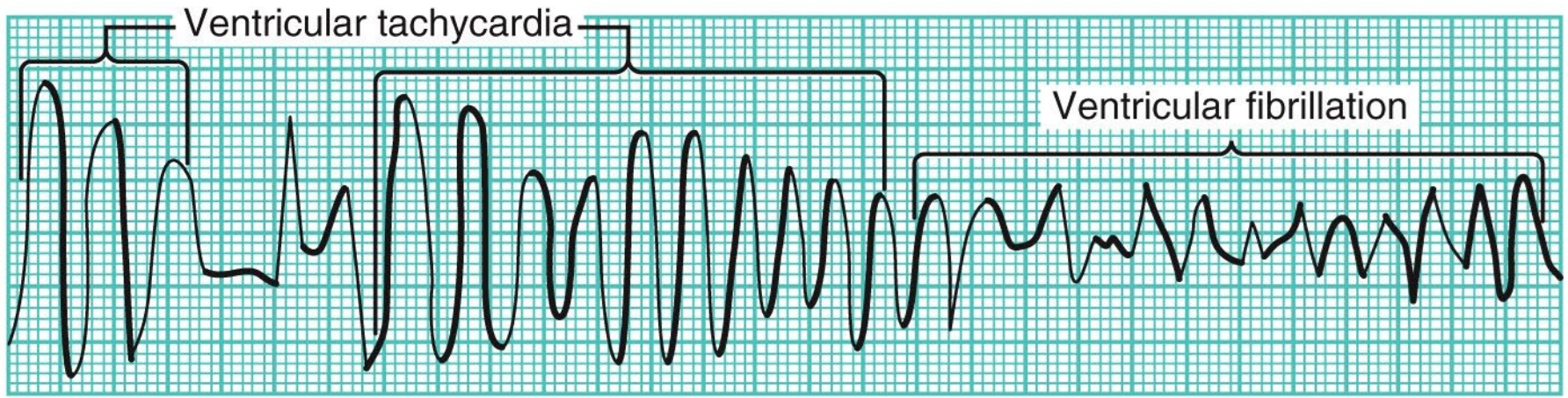


**(b) Nodal rhythm—no SA node activity**

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

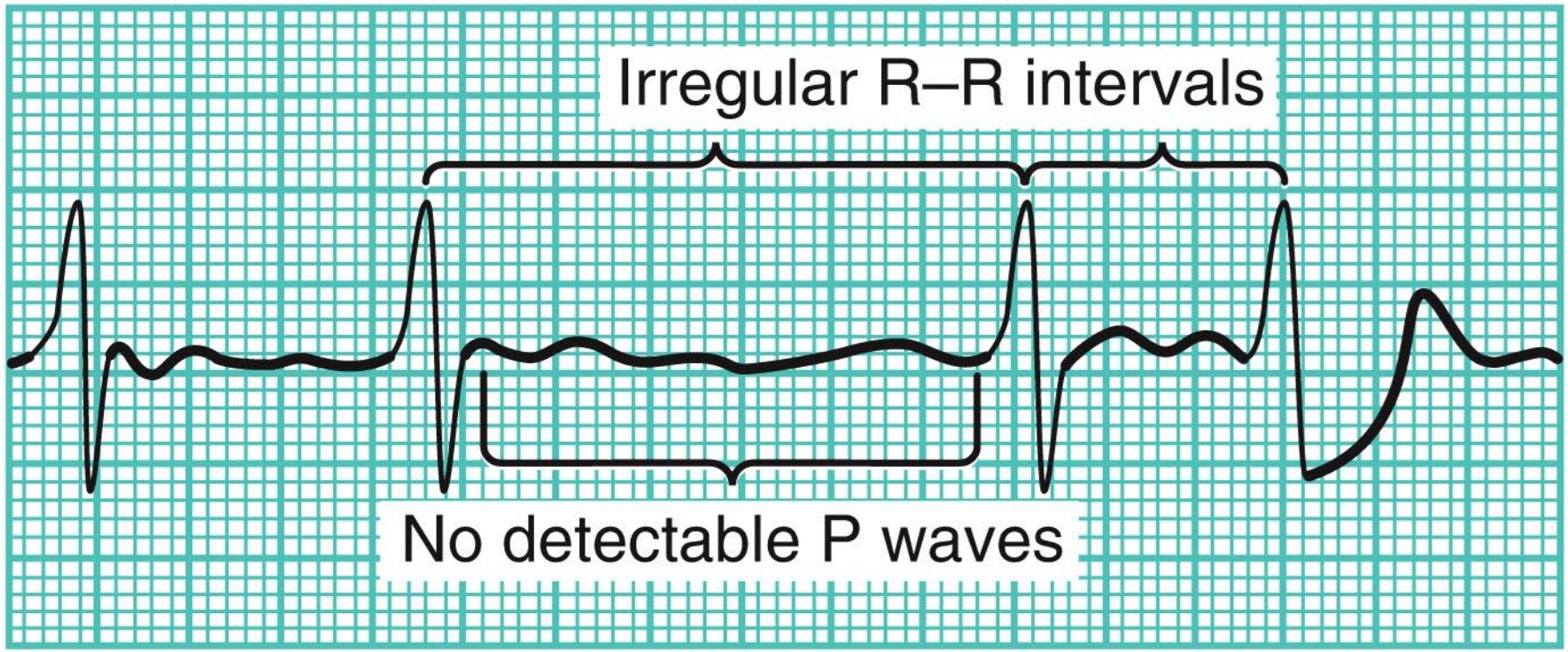



(b) First-degree AV block




(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