Chapter 19.2

The Heart

Learning Objectives

- **Describe the structure and function of the intrinsic cardiac conduction system**
- **Trace an impulse through the conduction system of the heart**

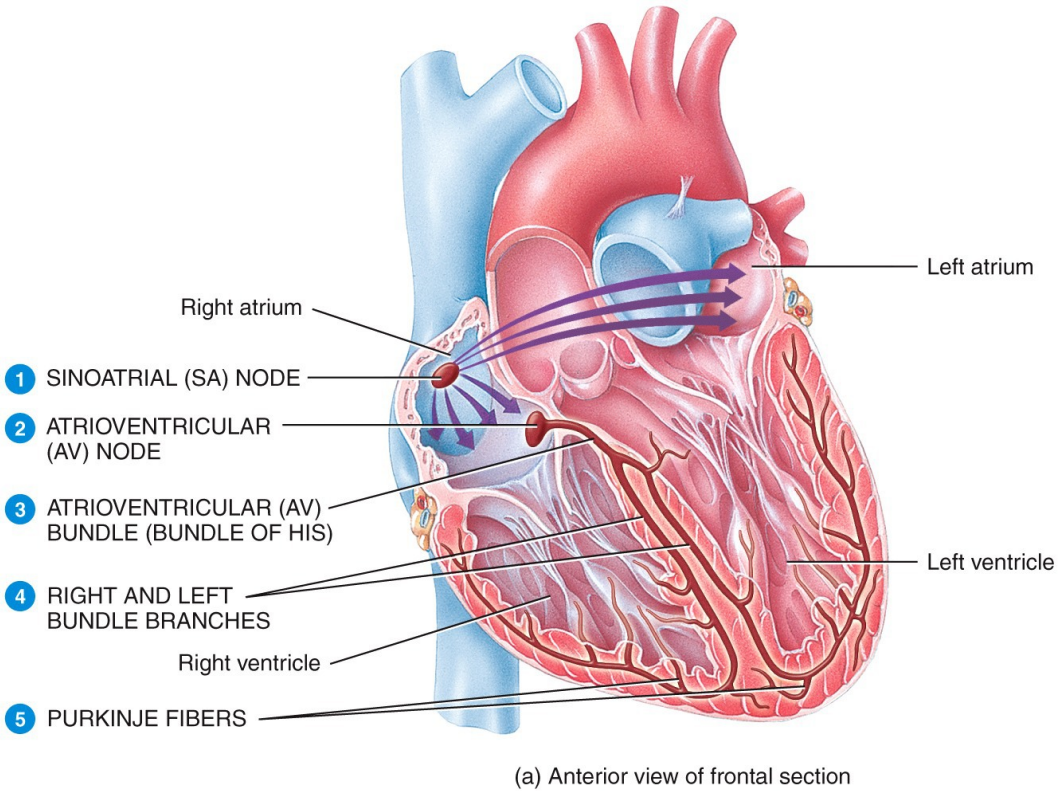
Intrinsic Cardiac Conduction System

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 truck and aorta

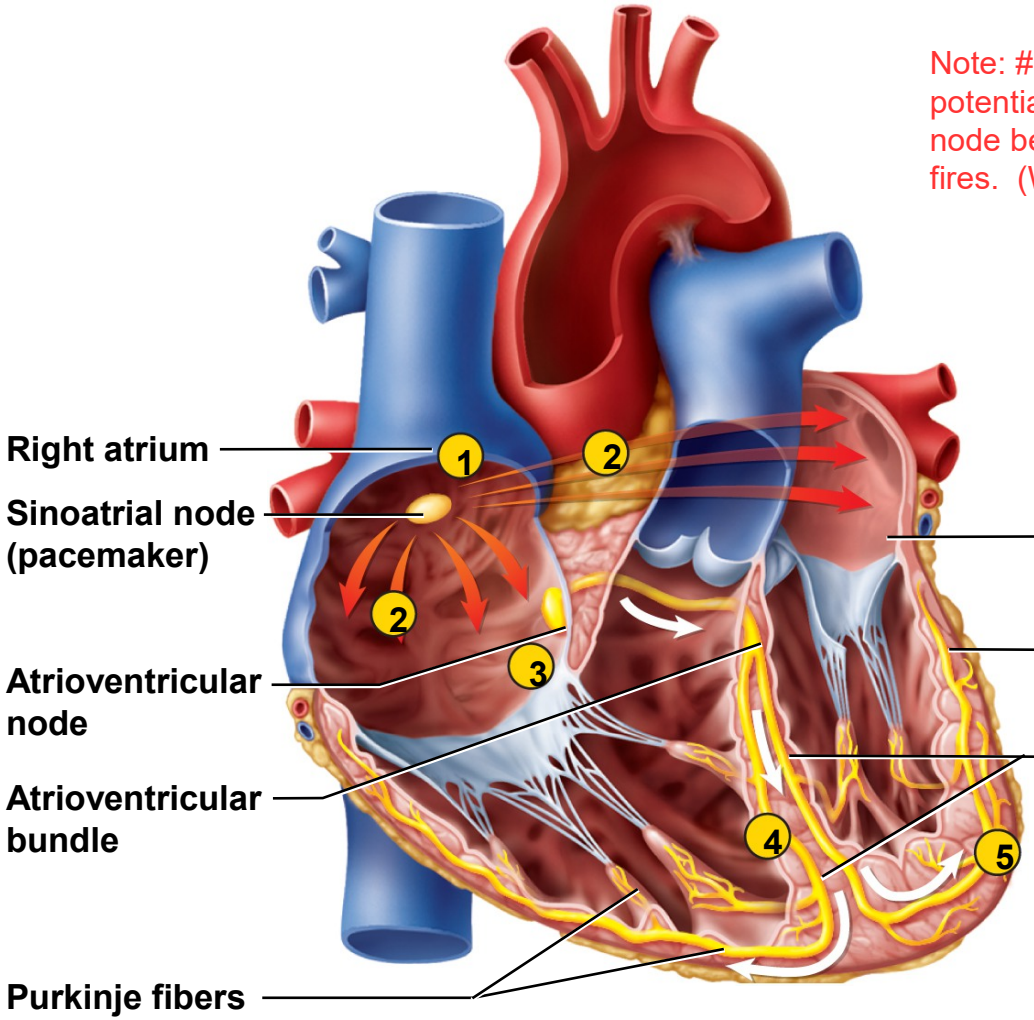


(a) Anterior view of frontal section

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



Note: #3 – action potential is delayed at AV node before AV node fires. (Why?)

1. SA node fires.
2. Excitation spreads through atrial myocardium.
3. AV node fires.
4. Excitation spreads down AV bundle.
5. Purkinje fibers distribute excitation through ventricular myocardium.

Cardiac cycle maintained by an internal pacemaker and by a nerve like conduction pathways which run through the myocardium between the AV note to the apex where it then moves up into walls of the myocardium

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 to prevent currents from getting to the ventricles from any other route
  - action potential delayed at AV note

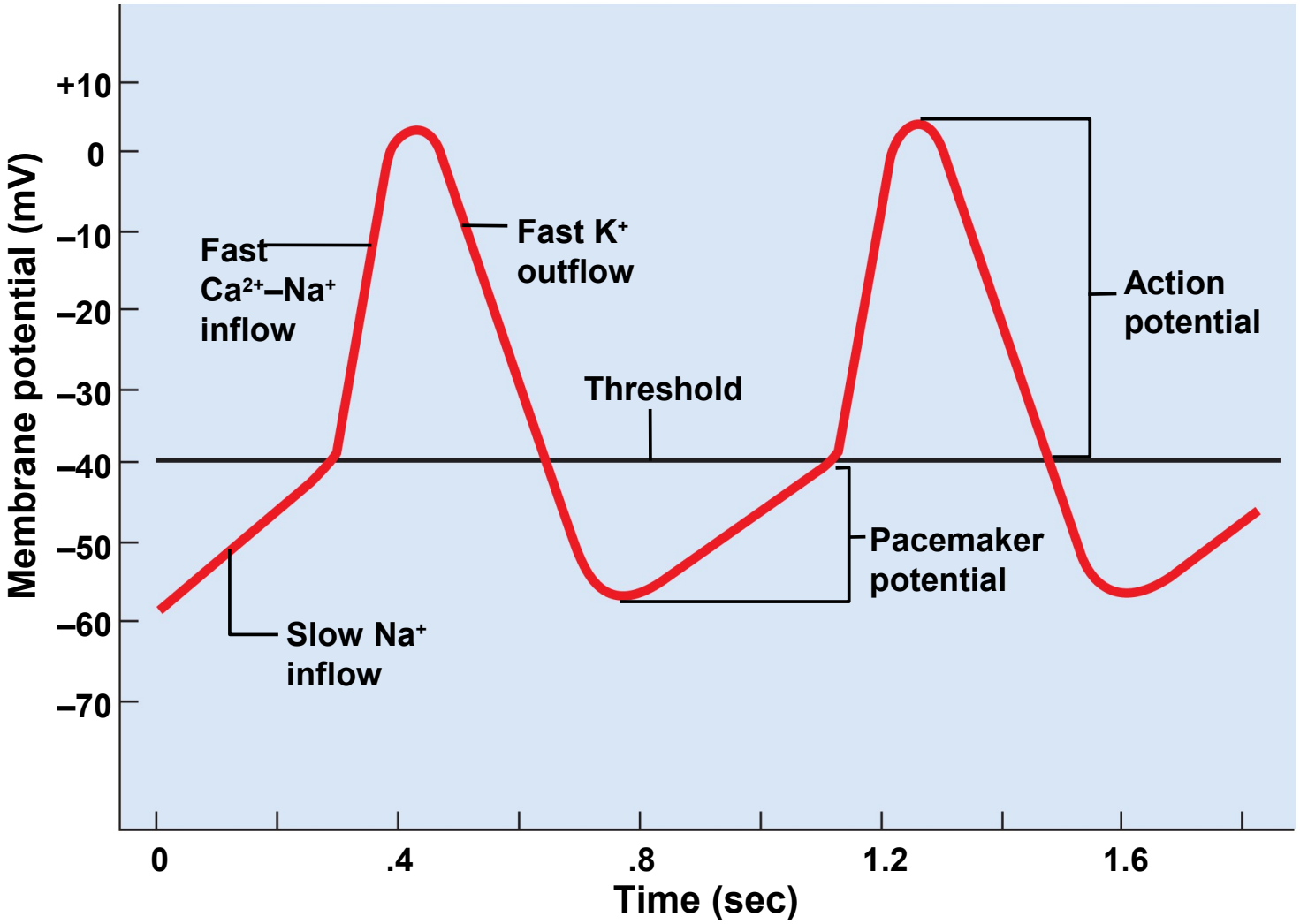
Cardiac Conduction System

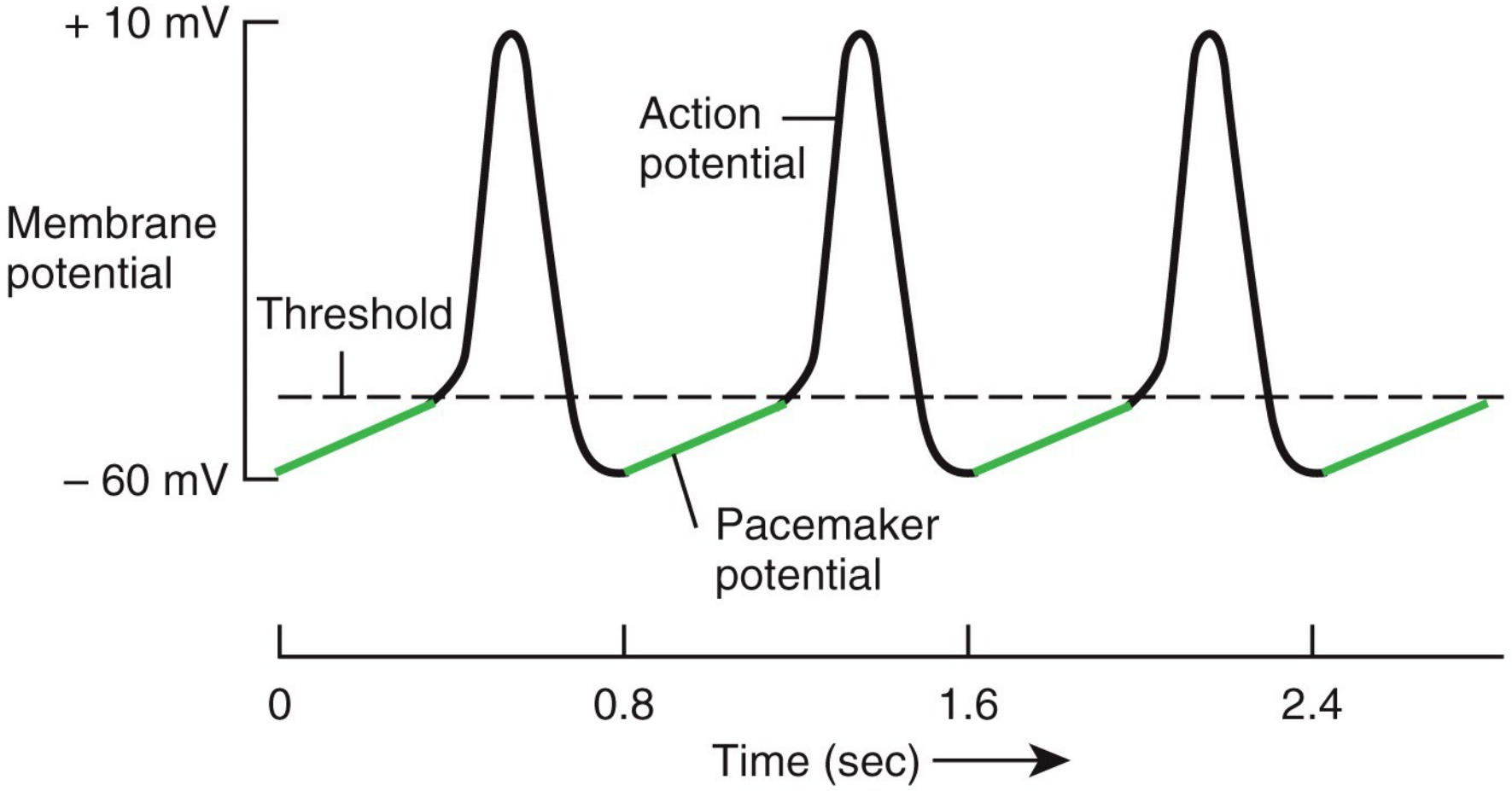
- Atrioventricular (AV) bundle (bundle of His)
  - bundle forks into right and left bundle branches
  - these branches pass through interventricular septum toward apex
- Purkinje fibers
  - Nerve like processes spread throughout ventricular myocardium
  - From end of Purkinje Fibers // signal pass from cell to cell through gap junctions

Nodal Tissue and Nerve Supply of the Heart

- Special myocardiocytes are not able to maintain a resting membrane potential // these cells allow sodium to leak into their cytoplasm // this is why the SA node is a pacemaker.
- The cells 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" (sodium enters node) // 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

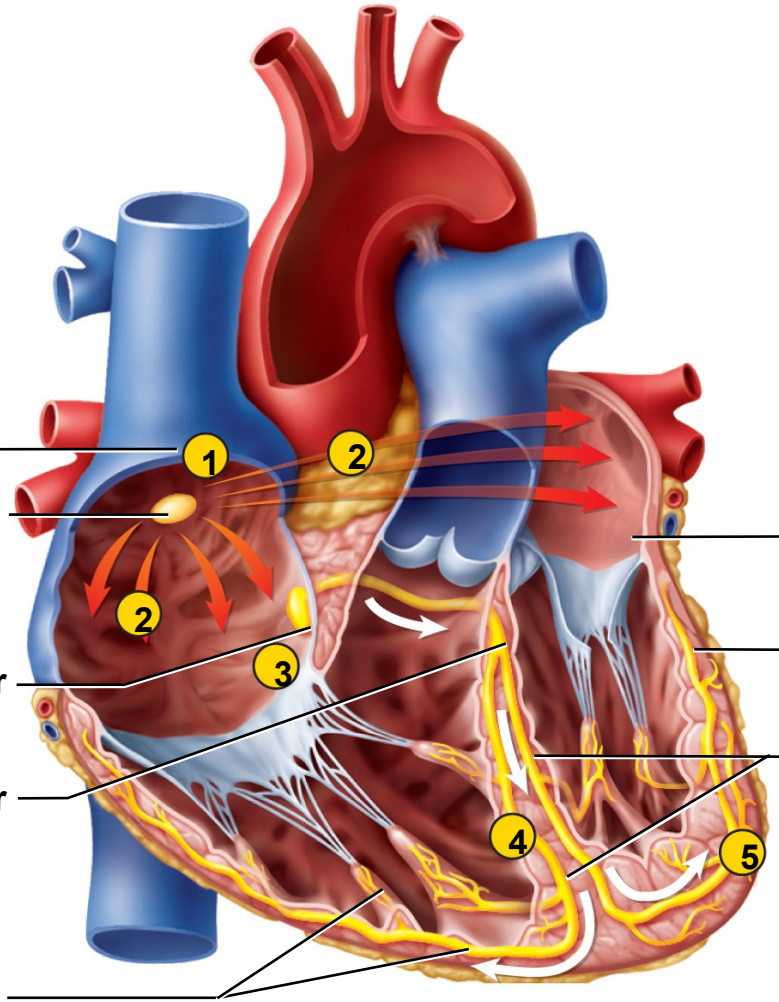
SA Node Potentials





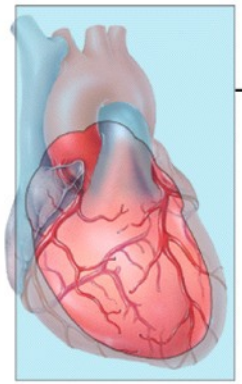
(b) Pacemaker potentials (green) and action potentials (black) in autorhythmic fibers of SA node

Cardiac Conduction System



1. SA node fires.
2. Excitation spreads through atrial myocardium.
3. AV node fires.
4. Excitation spreads down AV bundle.
5. Purkinje fibers distribute excitation through ventricular myocardium.

Note: conduction system extends into papillary muscles. As these muscle contract they pull on the cordae tendinae to prevent prolapse of the AV valves!



Frontal plane

Right atrium

1 SINOATRIAL (SA) NODE

2 ATRIOVENTRICULAR (AV) NODE

3 ATRIOVENTRICULAR (AV) BUNDLE (BUNDLE OF HIS)

4 RIGHT AND LEFT BUNDLE BRANCHES

Right ventricle

5 PURKINJE FIBERS

Left atrium

Left ventricle

(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*

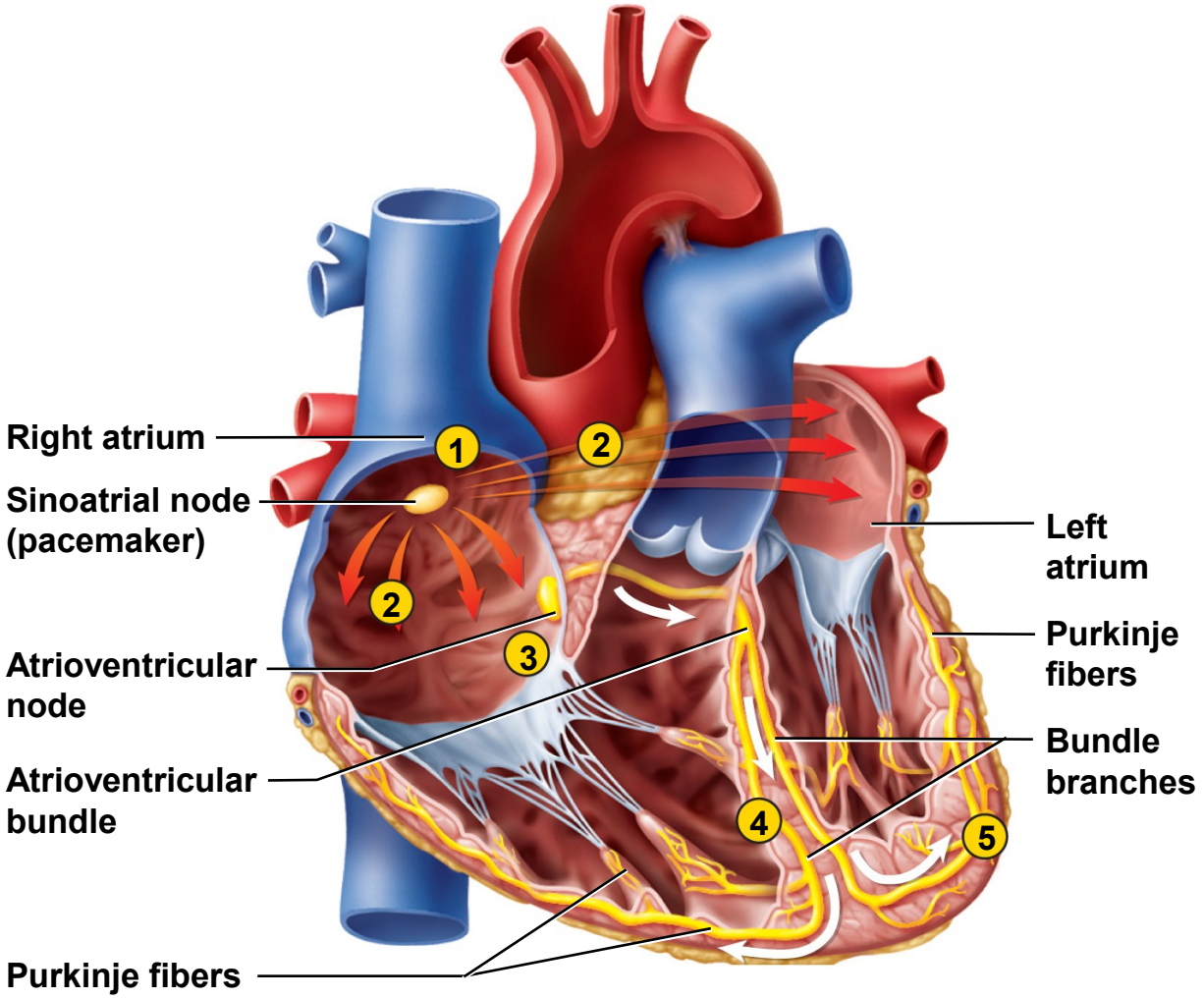
  - *atrial ventricular septum blocks flow of action potential because septum does not have gap junctions.*

  - 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

Cardiac Conduction System



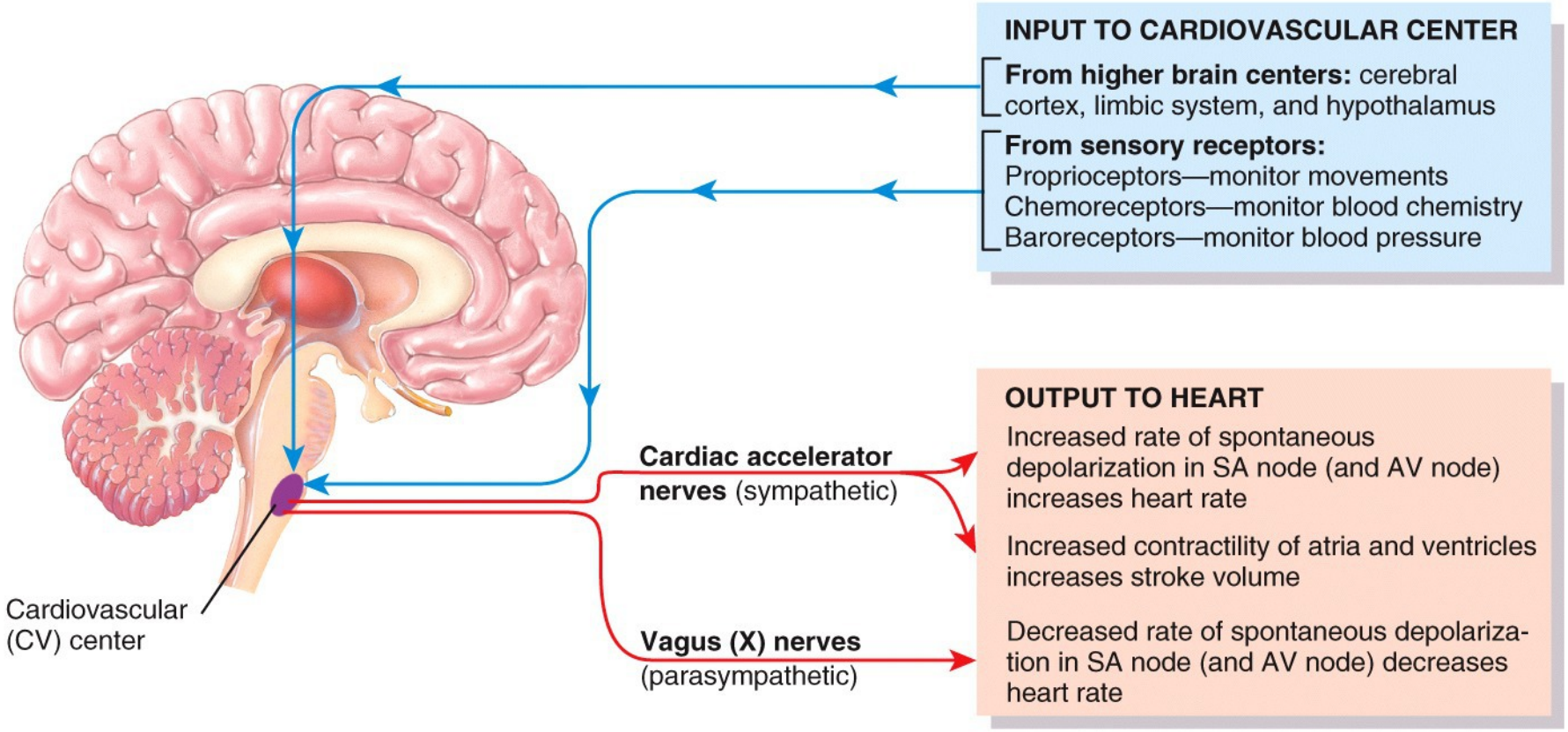
1. SA node fires.
2. Excitation spreads through atrial myocardium.
3. AV node fires.
4. Excitation spreads down AV bundle.
5. Purkinje fibers distribute excitation through ventricular myocardium.

Learning Objectives

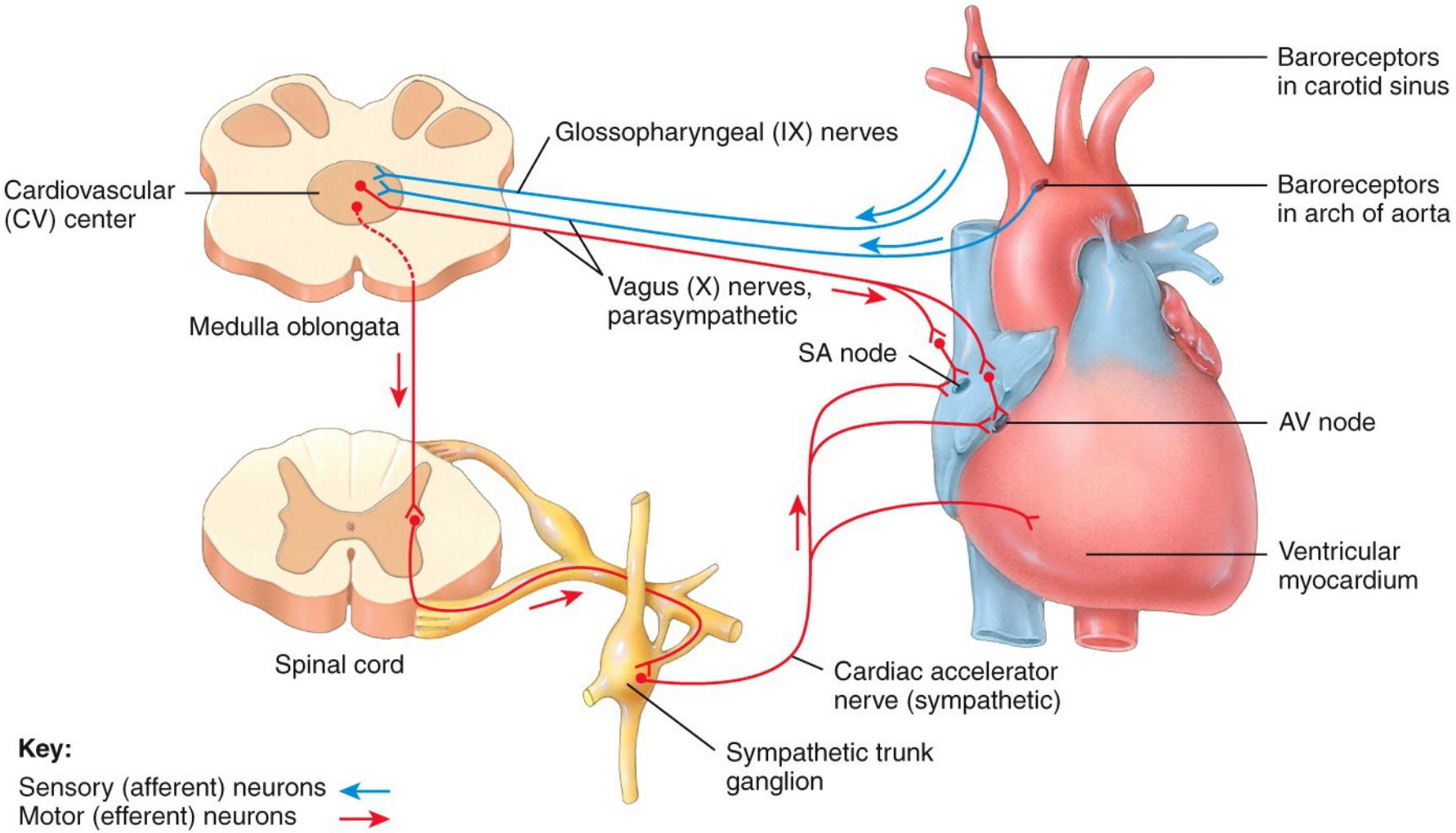
- **Identify the area of the CNS concerned with cardiovascular regulation.**
- **Describe factors which regulate heart rate, including the autonomic nervous system and baroreceptors**
- **Cardiac rhythms & arrhythmia**

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 /// 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

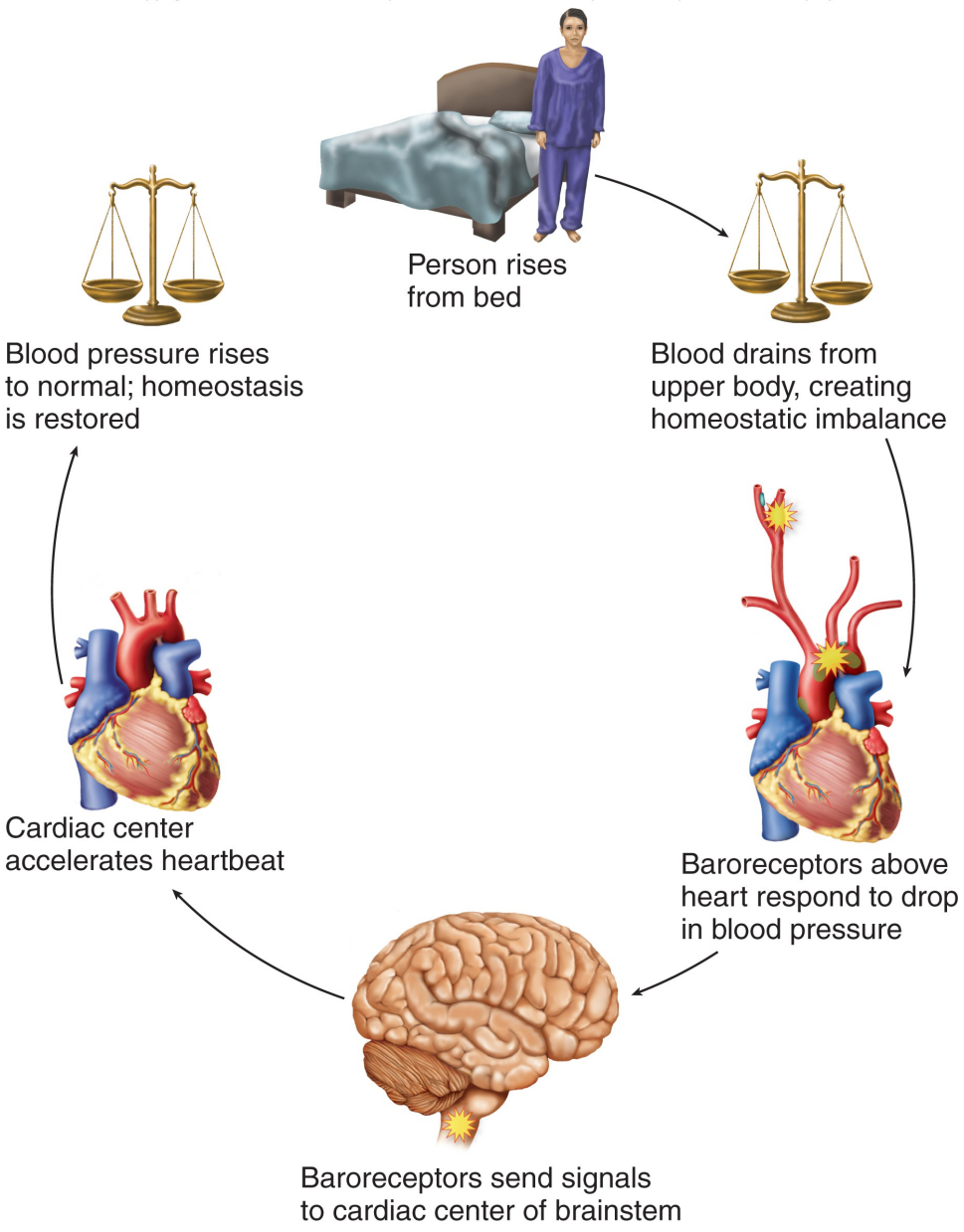
Regulation (Inputs) to Cardiac Center

- **Proprioception is a type of sensation located in the muscles and joints // receptors are 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

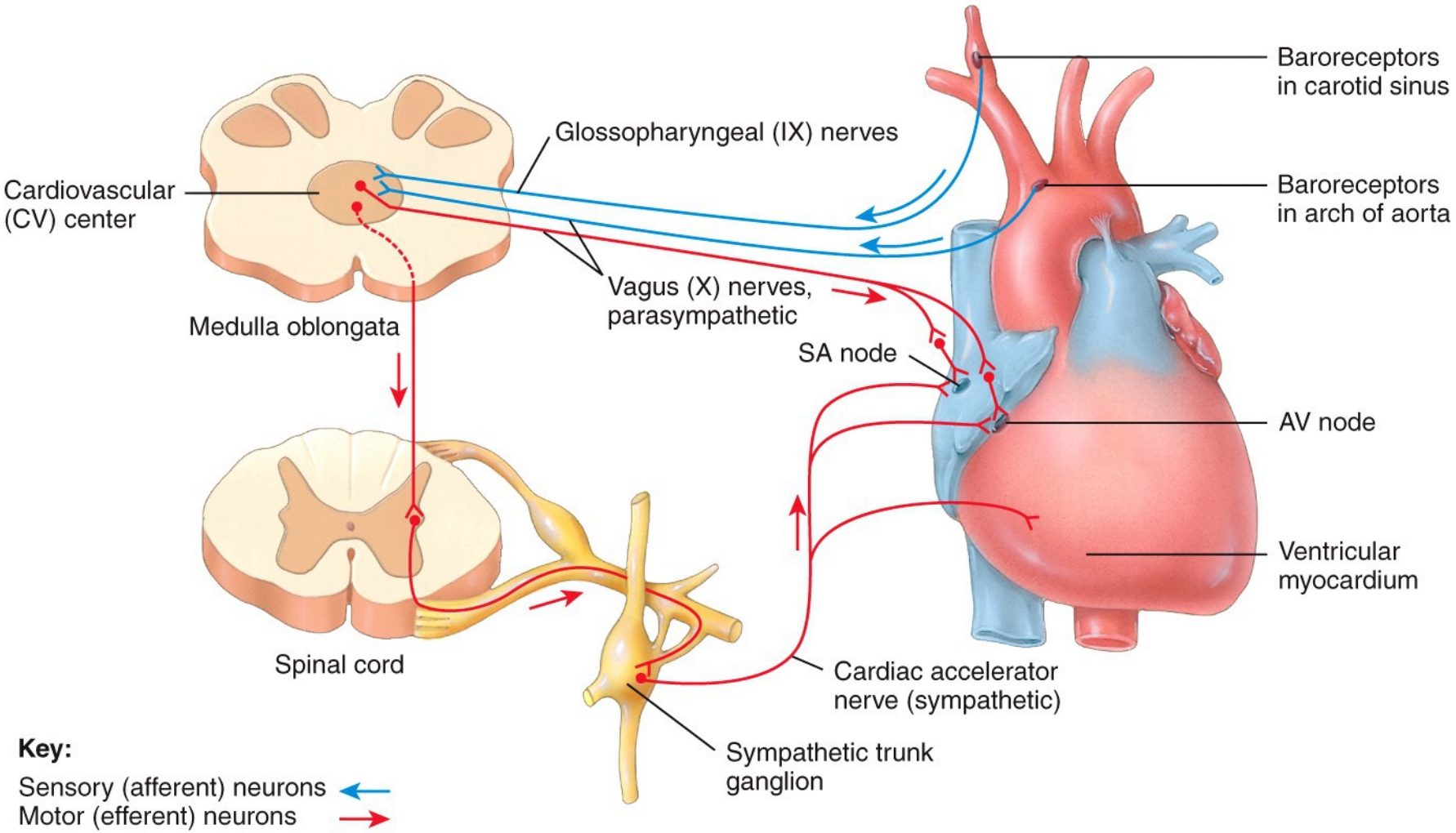
- Chemoreceptors (three locations):
  - aortic arch
  - carotid arteries
  - medulla oblongata
  - sensitive to blood pH, CO_2 and O_2 levels
  - *Chmeoreceptors are more important in respiratory control than cardiac control // minor role in heart function*
    - if CO_2 accumulates in blood or CSF (hypercapnia), 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
  - some pass through cardiac plexus in mediastinum
  - continue as cardiac nerves to the heart


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

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 Postganlionic Fibers

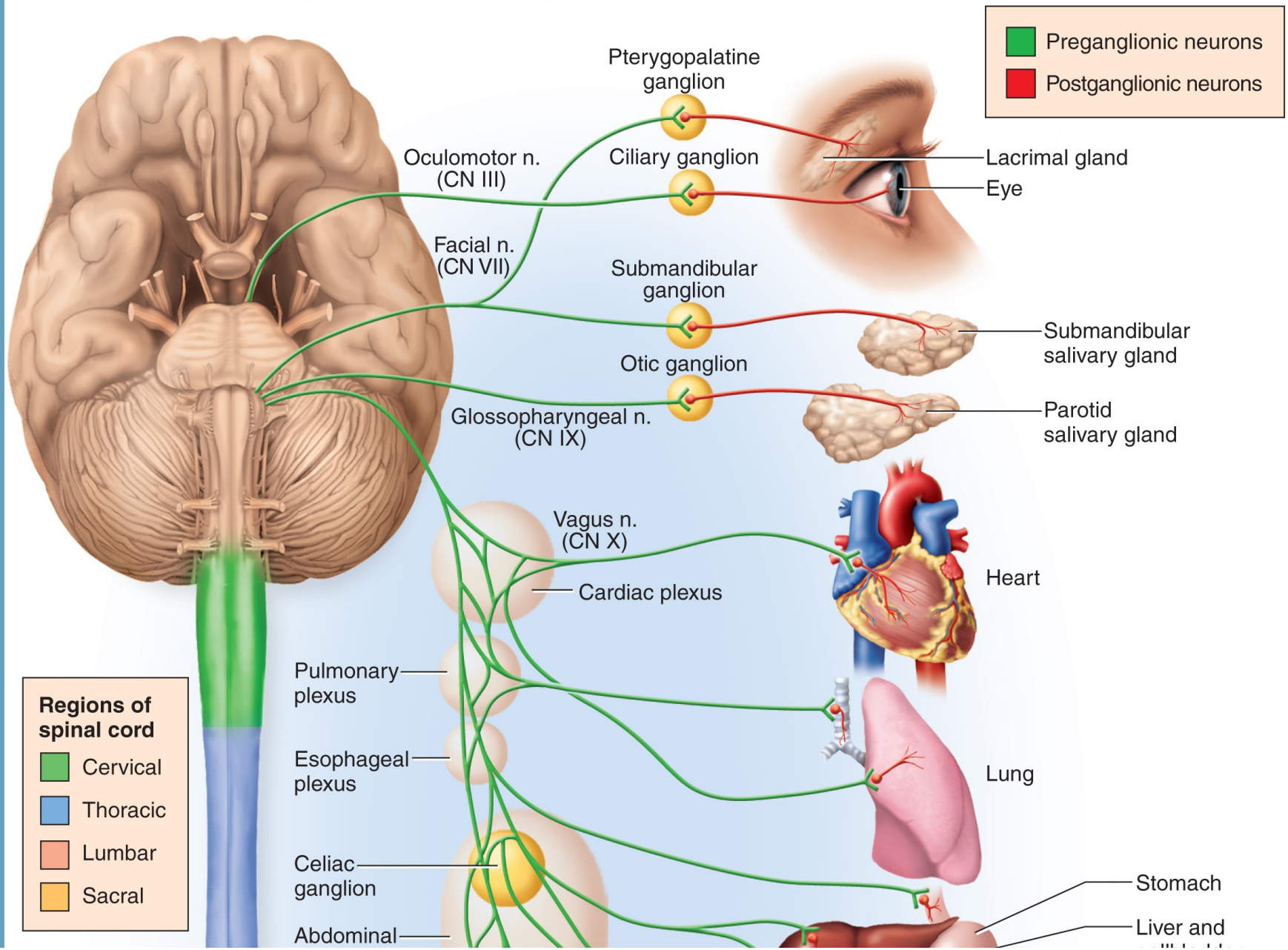
- Adrenergic fiber release norepinphrine
  - Bind to beta 1 receptors
  - Use second messenger system // cAMP
  - Myocardiocytes and nodal cells
  - G protein activates enzymes that open plasma channels to allow calcium to enter cell // this increases depolarization rate
  - cAMP also increase uptake of calcium back into sarcoplasmic reticulumn // speed up repolarization rate
  - Overall outcome is to increase heart rate
  - From 75 bpm (resting) to maximum of 240 bpm
  - Note: cardiac output does not increases after 160 bpm. Why? (think about 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 Postganlionic Fibers

- **Cholinergic fiber 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 reploraization 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 life style 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 /// examples
  - 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

What is fibrillation? Why is it dangerous?

- 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 // 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 myocardiocytes at same time
    - stop the fibrillation
    - hope to reset the normal SA node function and sinus rhythm

Cardiac Rhythms / Terminology

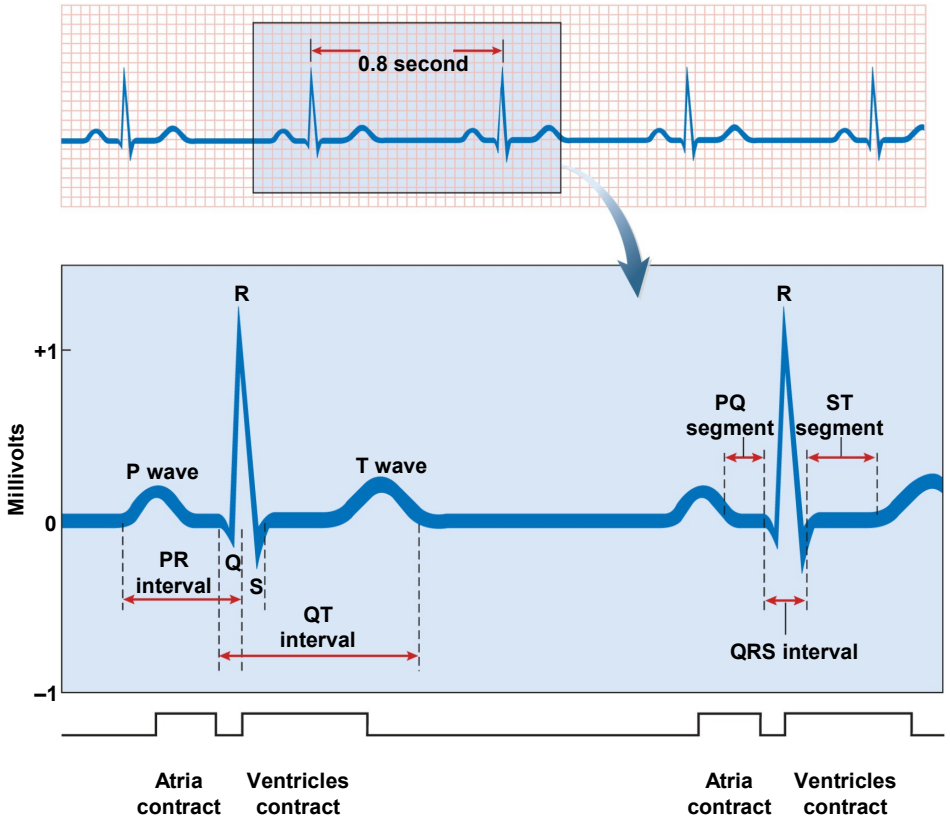
- Premature ventricular contractions (PVCs) // caused by stimulants, stress or lack of sleep
- Tachycardia // Persistent resting adult heart rate above 100 bpm
- Bradycardia // Persistent resting adult heart rate below 60 bpm

Learning Objectives

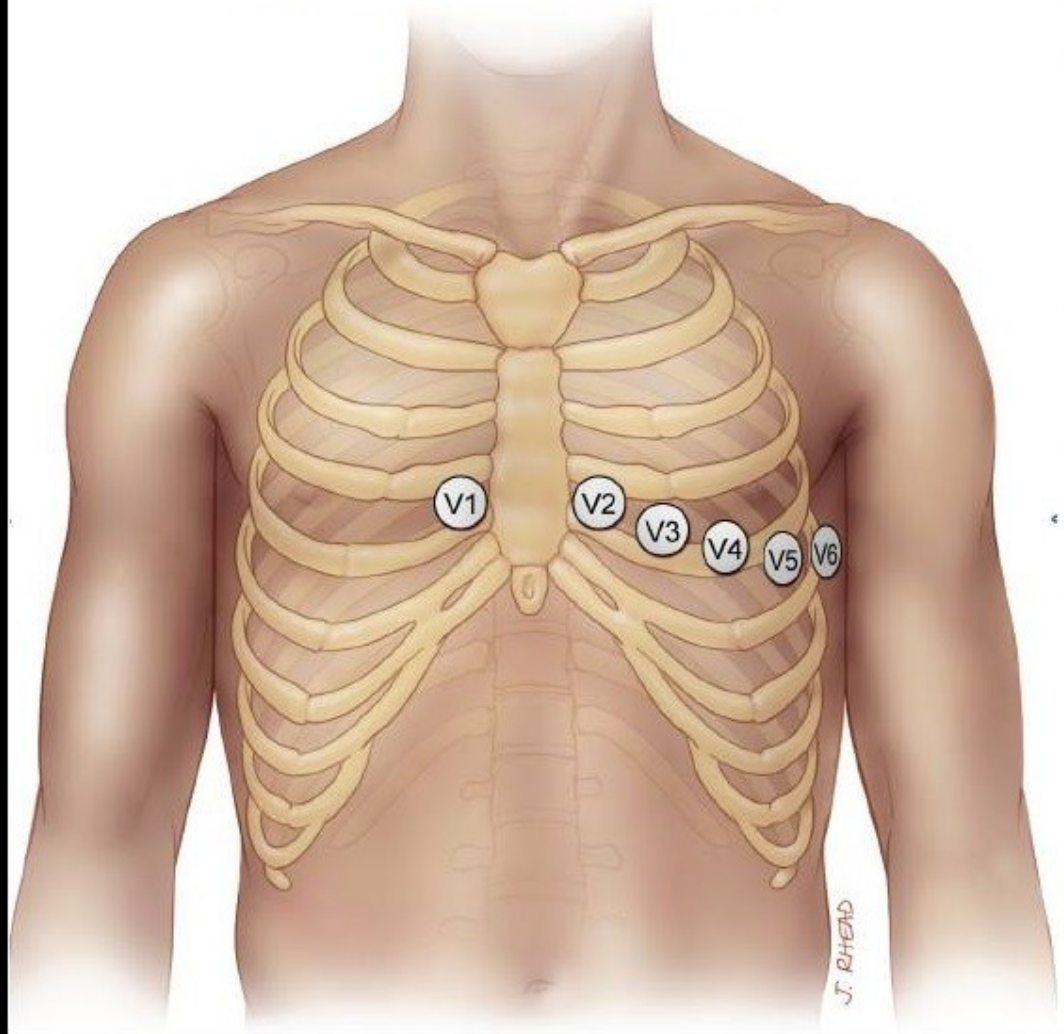
- **Diagram and describe a typical ECG (EKG) pattern and relate it to pressure changes and heart sounds**

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

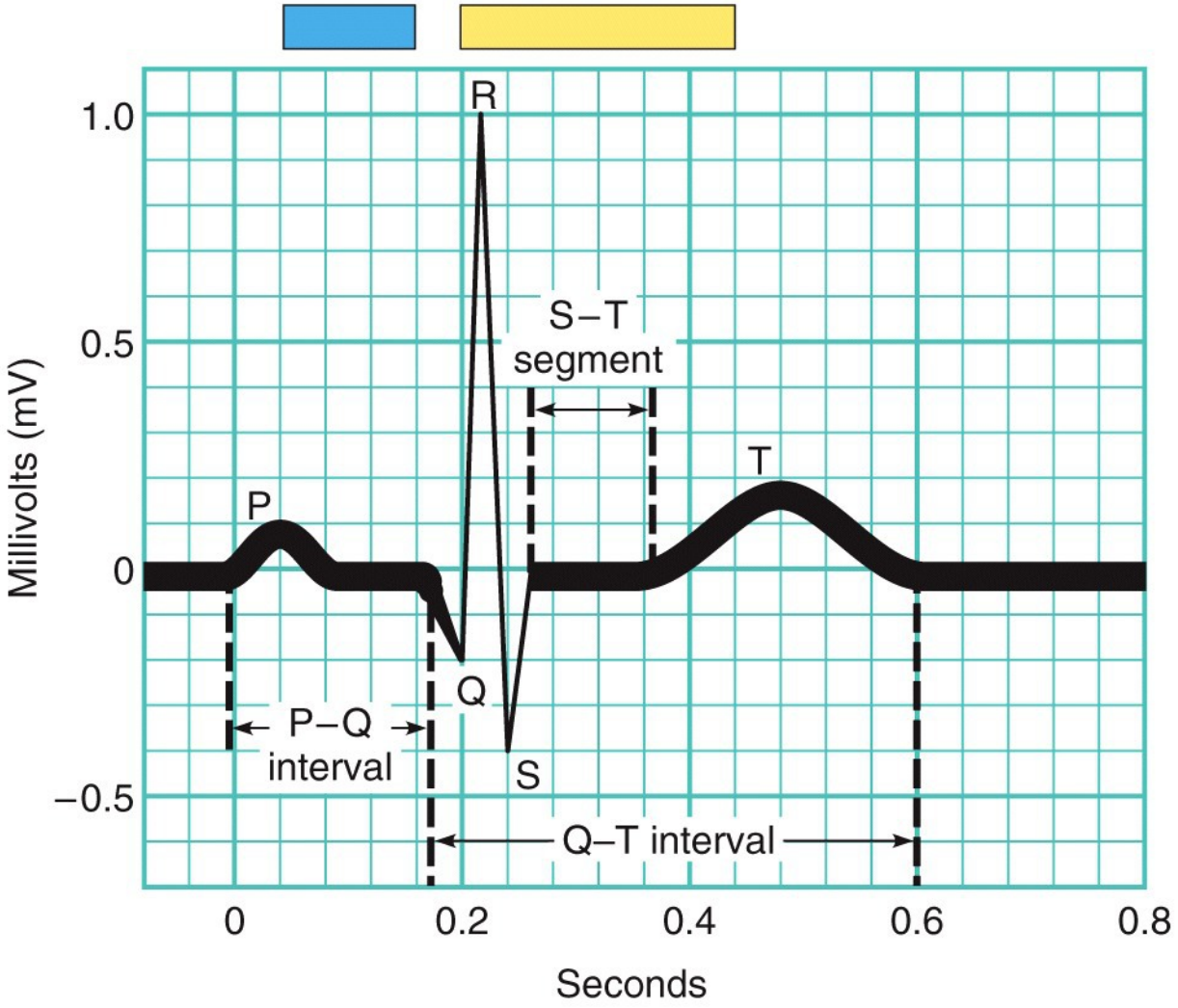
V6 Midaxillary line at the same level as V4 and V5

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



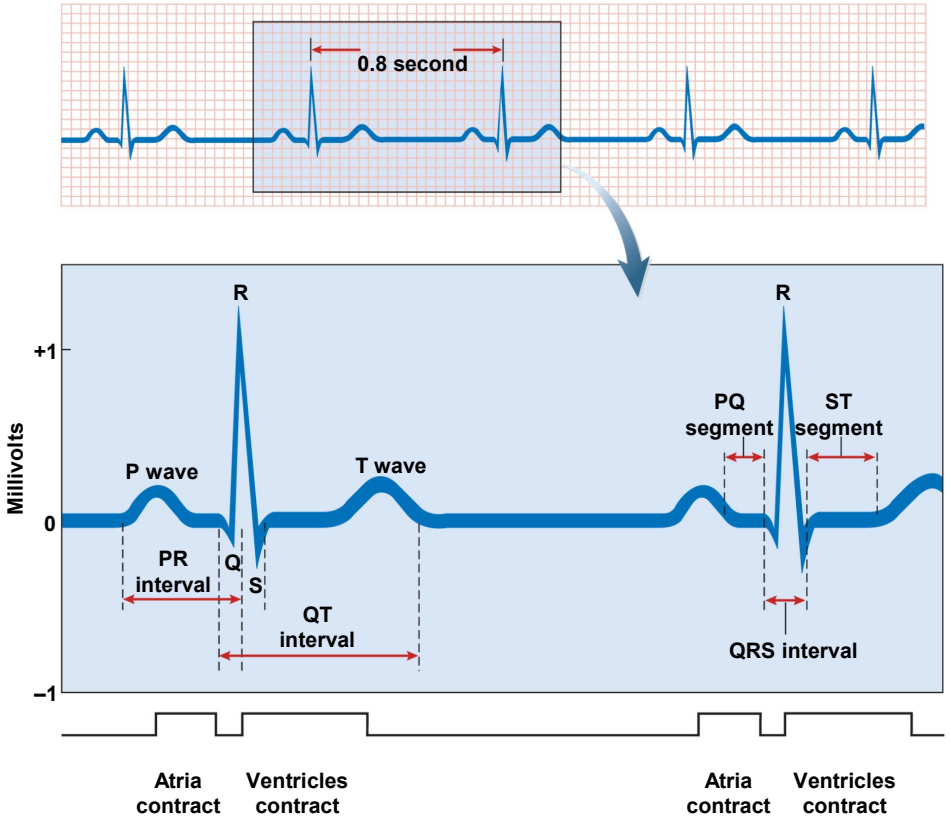
Key:

Atrial contraction

Ventricular contraction

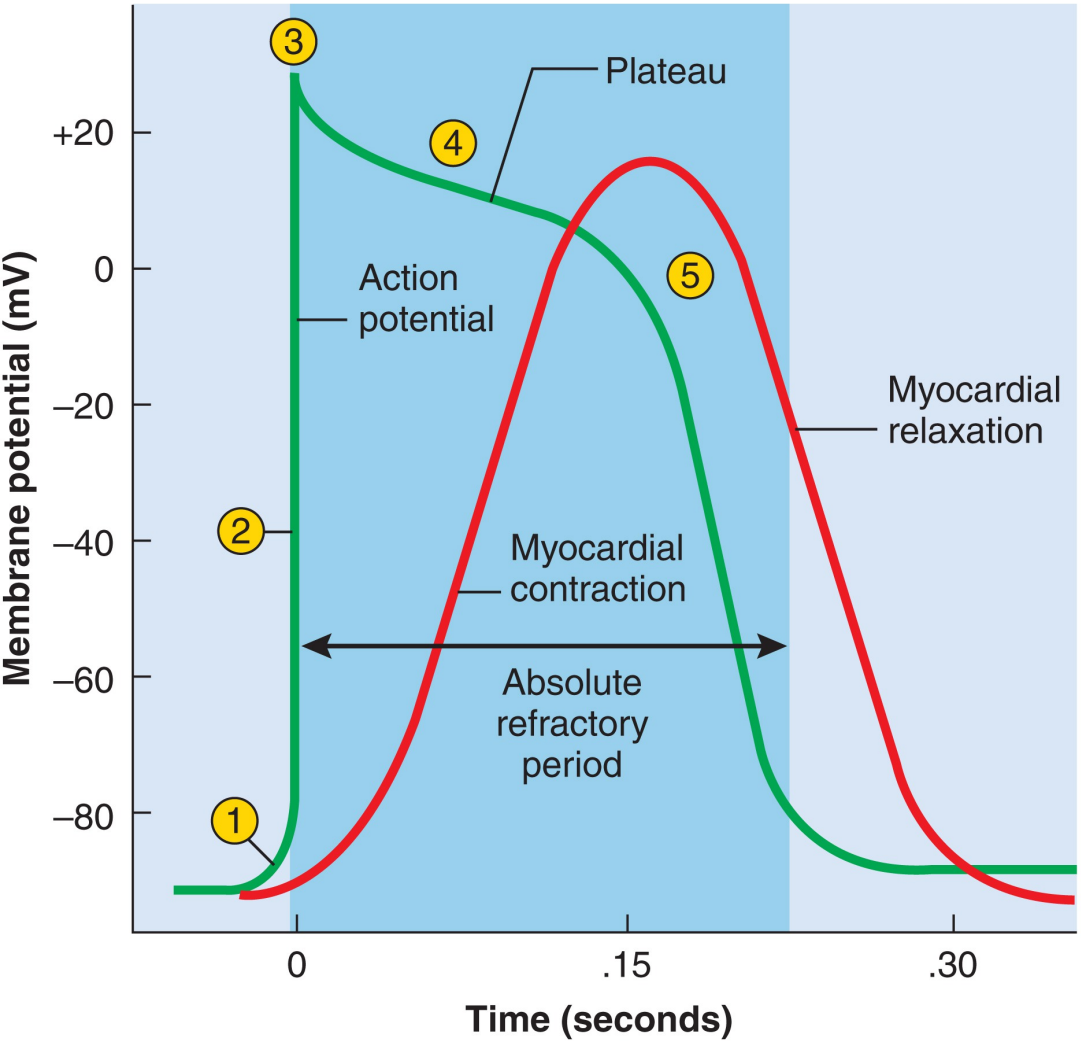
Electrocardiogram

- 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

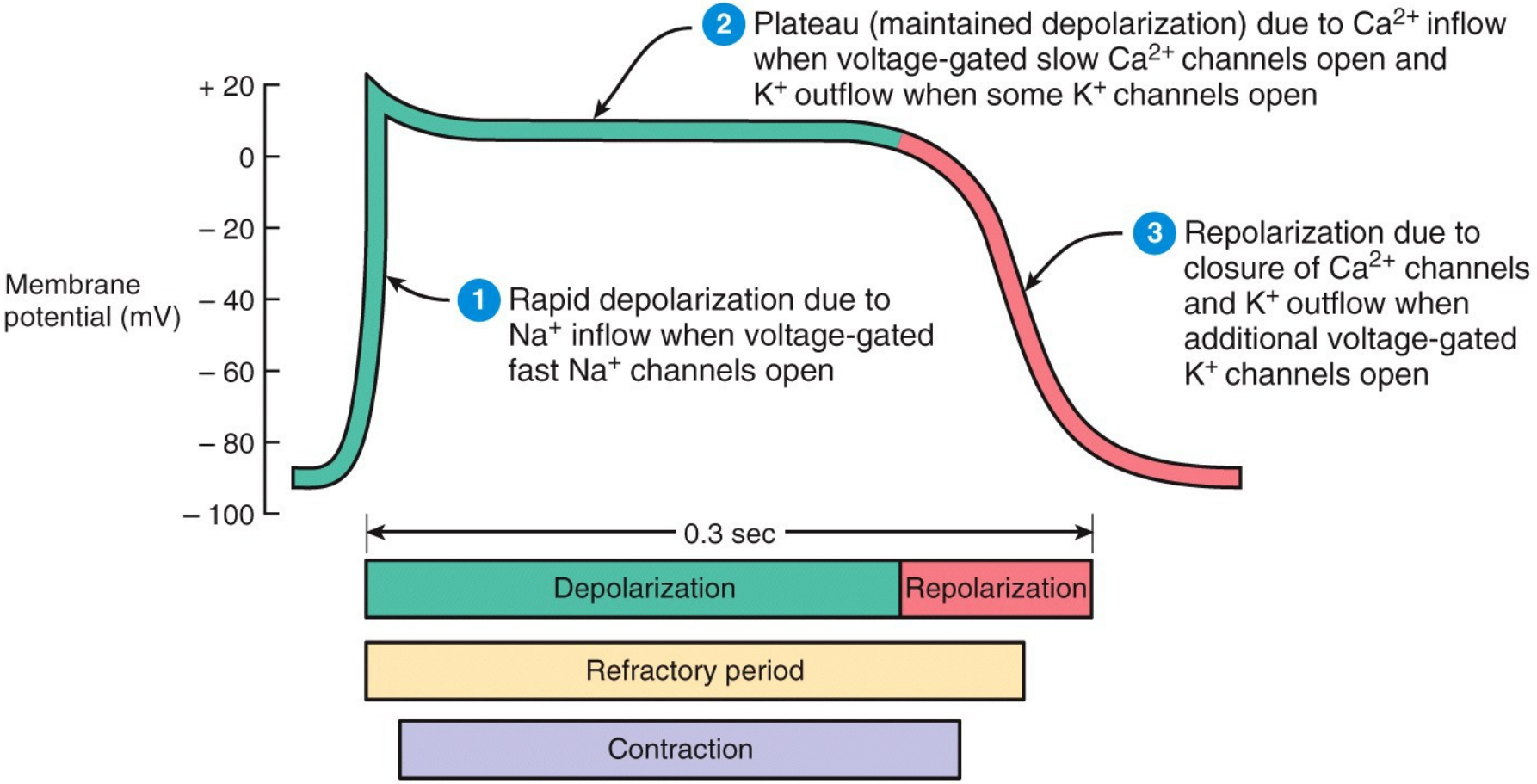


Note: asystole = “flat line” / no contraction of myocardium / requires cardiopulmonary resuscitation (CPR)

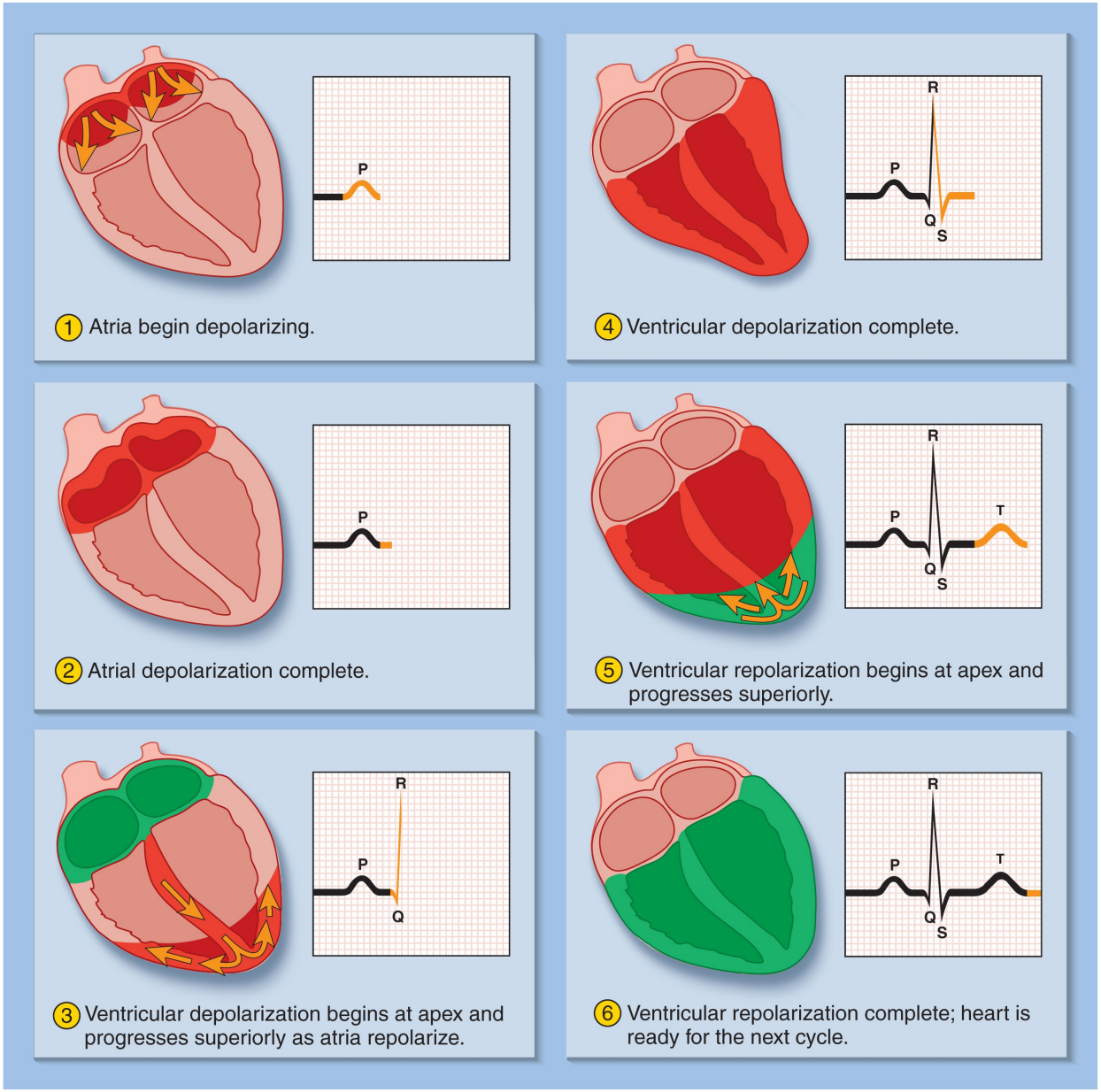
Action Potential of Myocardiocyte



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.


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

Electrical Activity of Myocardium



1) atrial depolarization begins

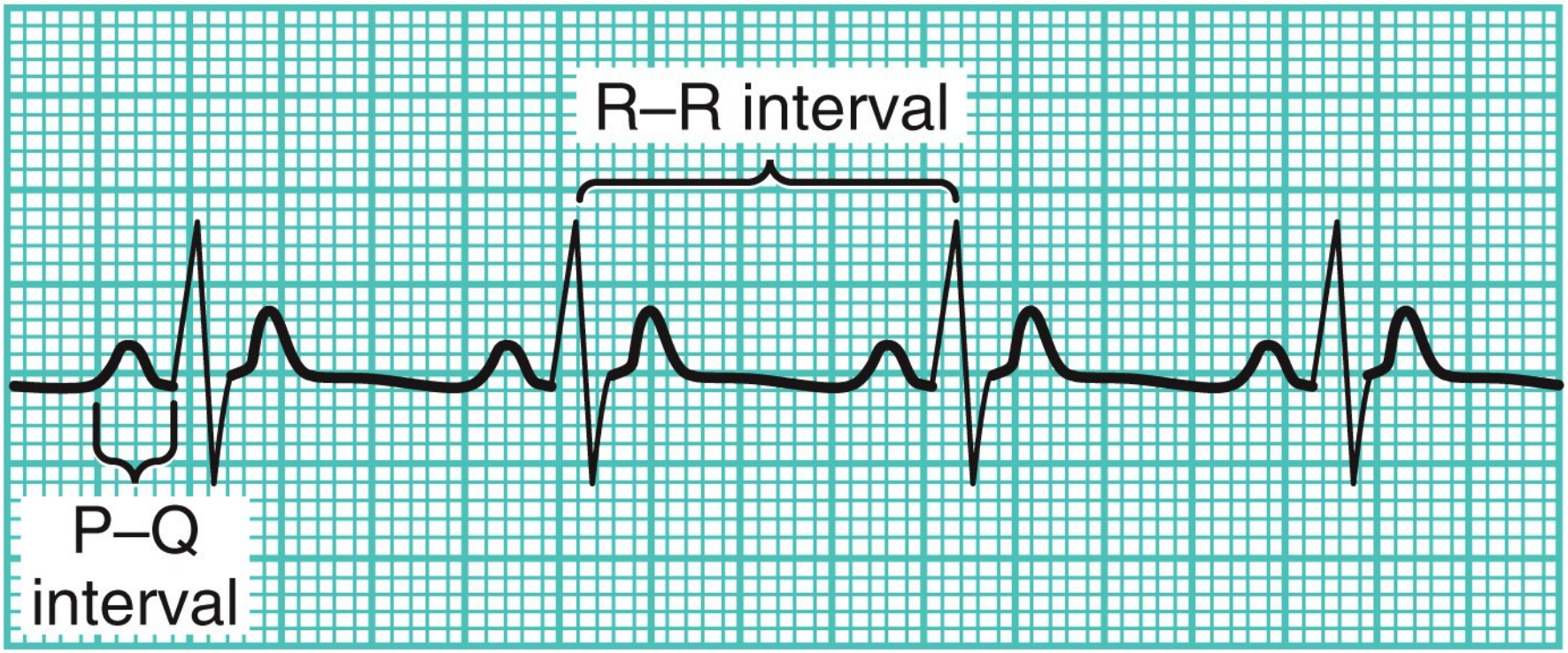
2) atrial depolarization complete (atria contracted)

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

4) ventricular depolarization complete (ventricles contracted)

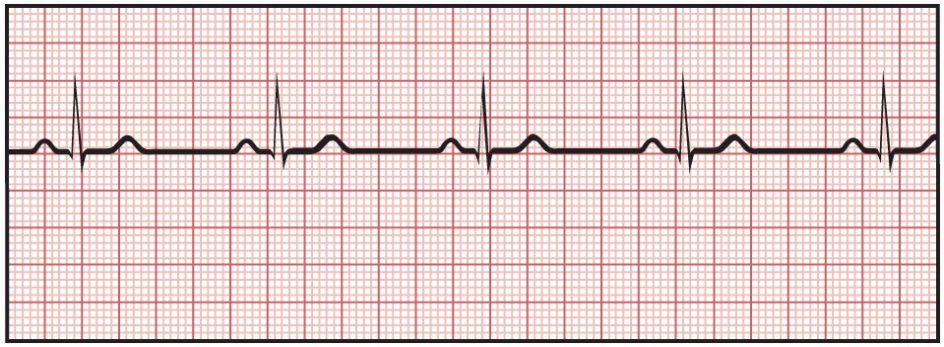
5) ventricles begin to repolarize at apex

6) ventricular repolarization complete (ventricles relaxed)

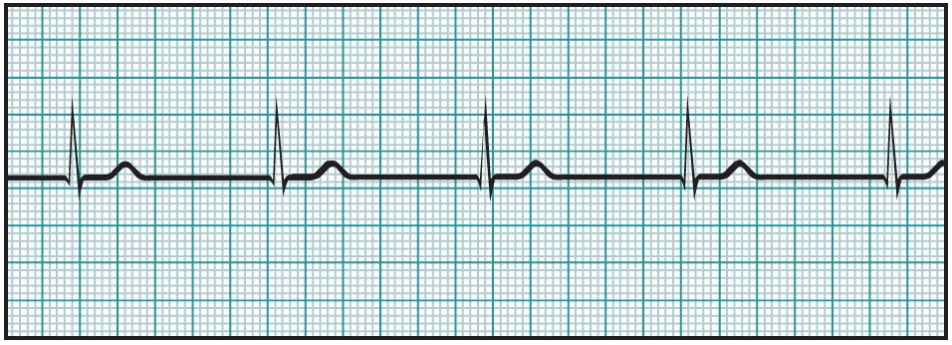


(a) Normal electrocardiogram (ECG)

ECGs: Normal and Abnormal

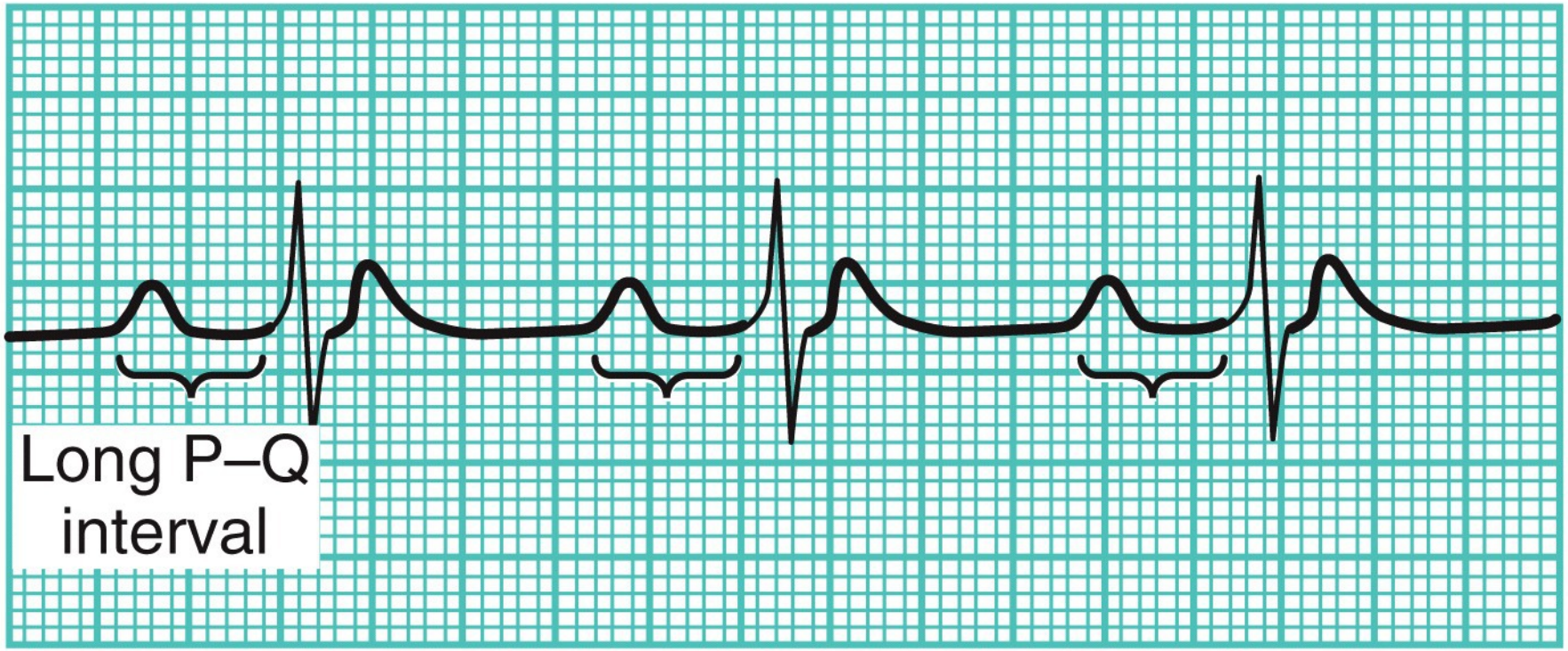


(a) Sinus rhythm (normal)

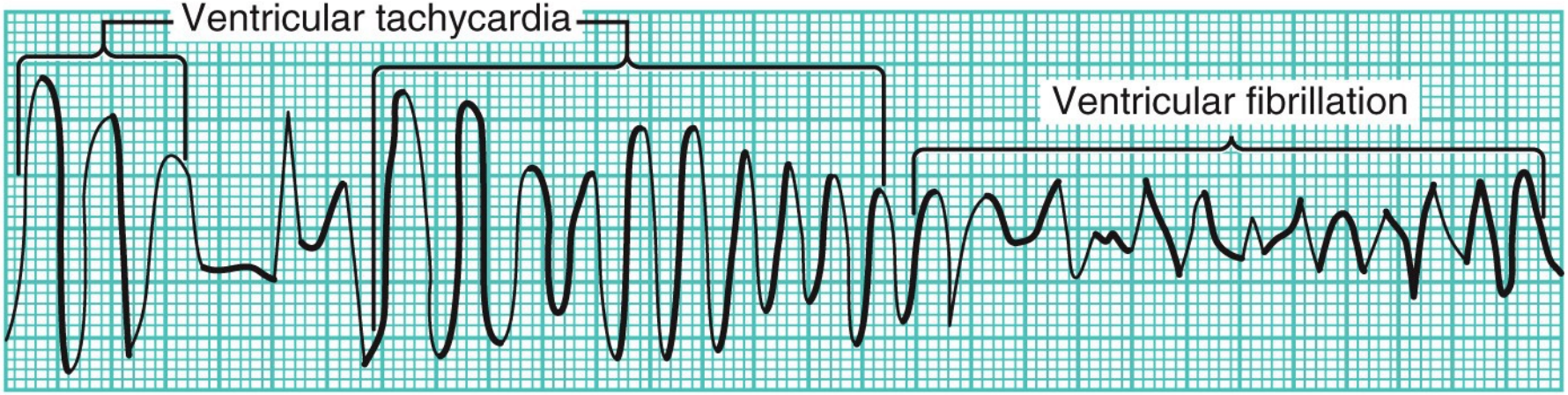


(b) Nodal rhythm—no SA node activity

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

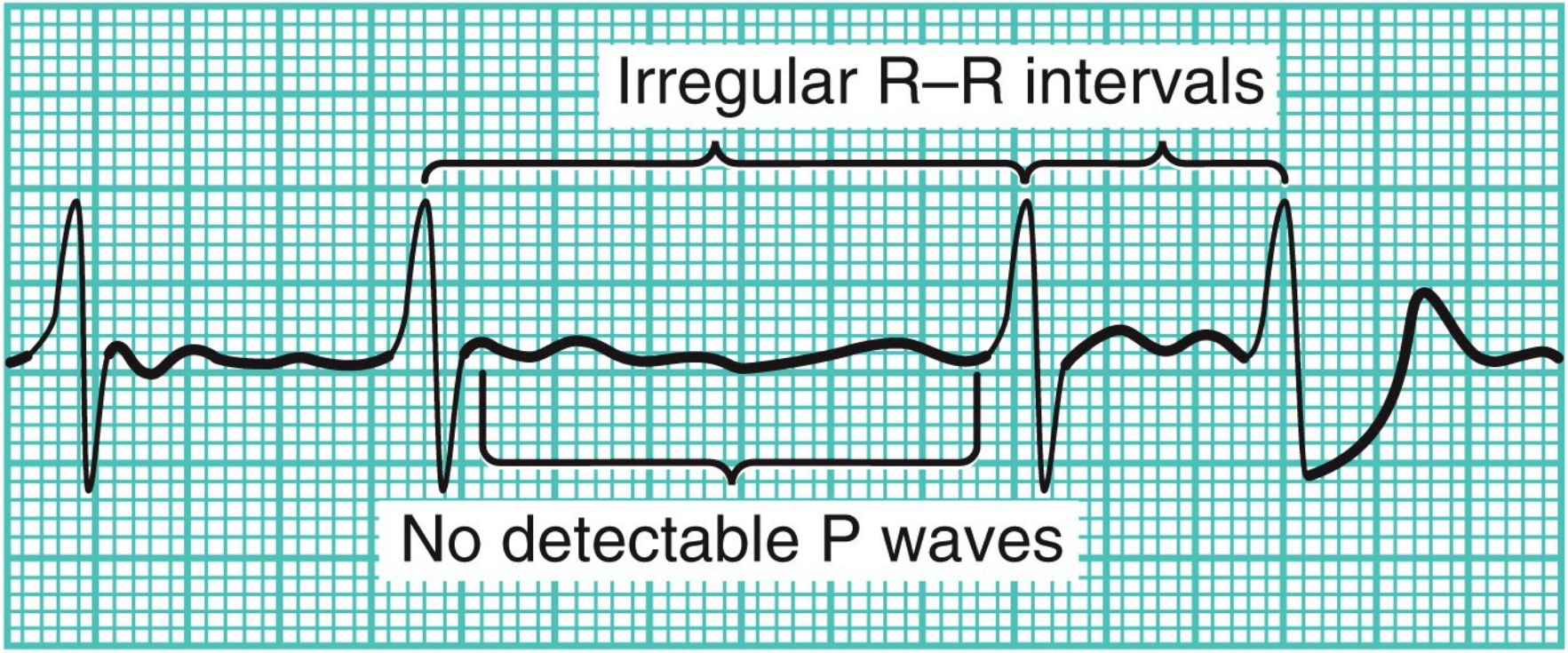


(b) First-degree AV block



(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