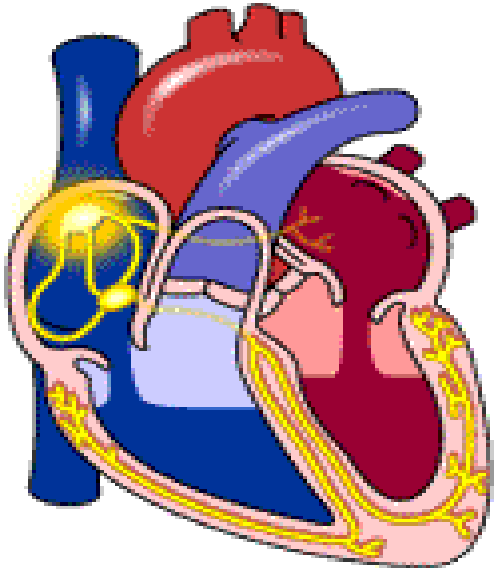
CARDIAC ELECTRIC ACTIVITY: CONDUCTING SYSTEM



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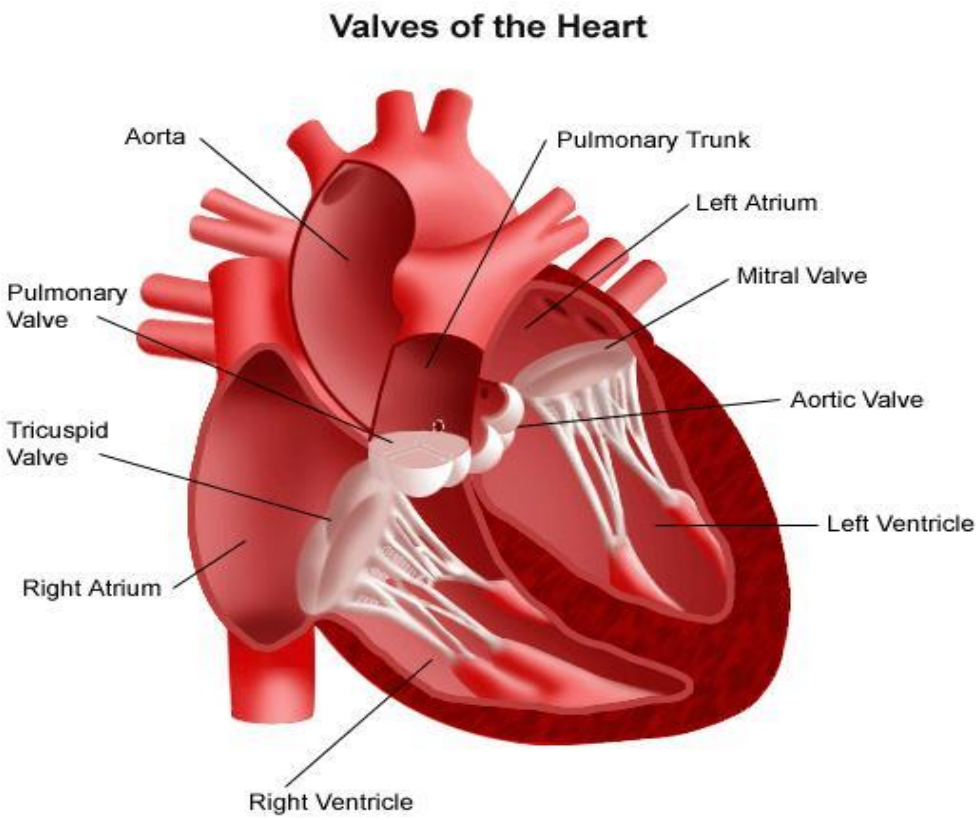
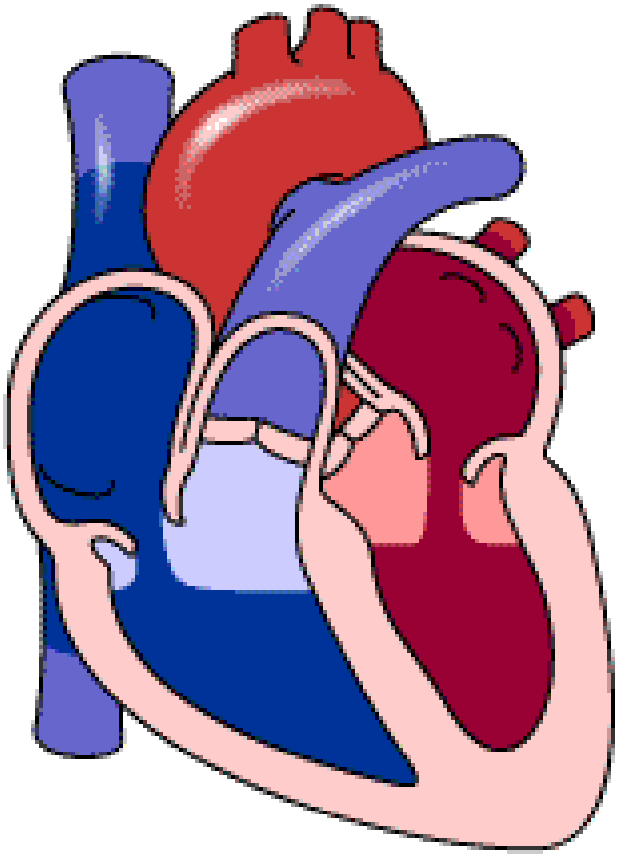
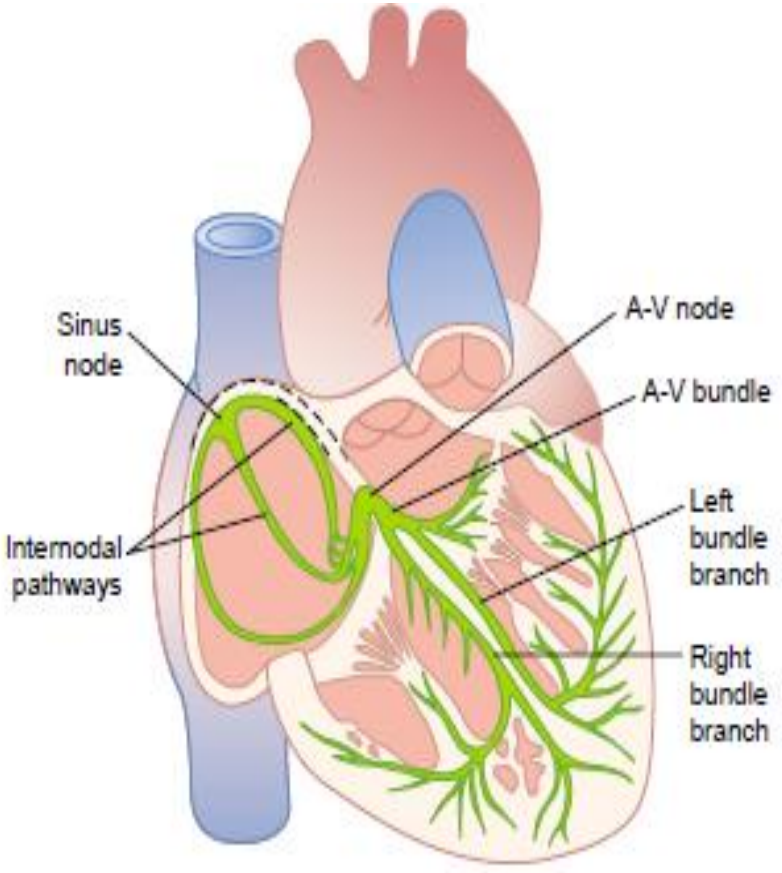
COMPONENTS OF CONDUCTIVE SYSTEM





COMPONENTS OF CONDUCTIVE SYSTEM

- S A Node
- Inter-Nodal Pathway
- A V Node
- A V bundle
- Right bundle branch
- Left bundle branch



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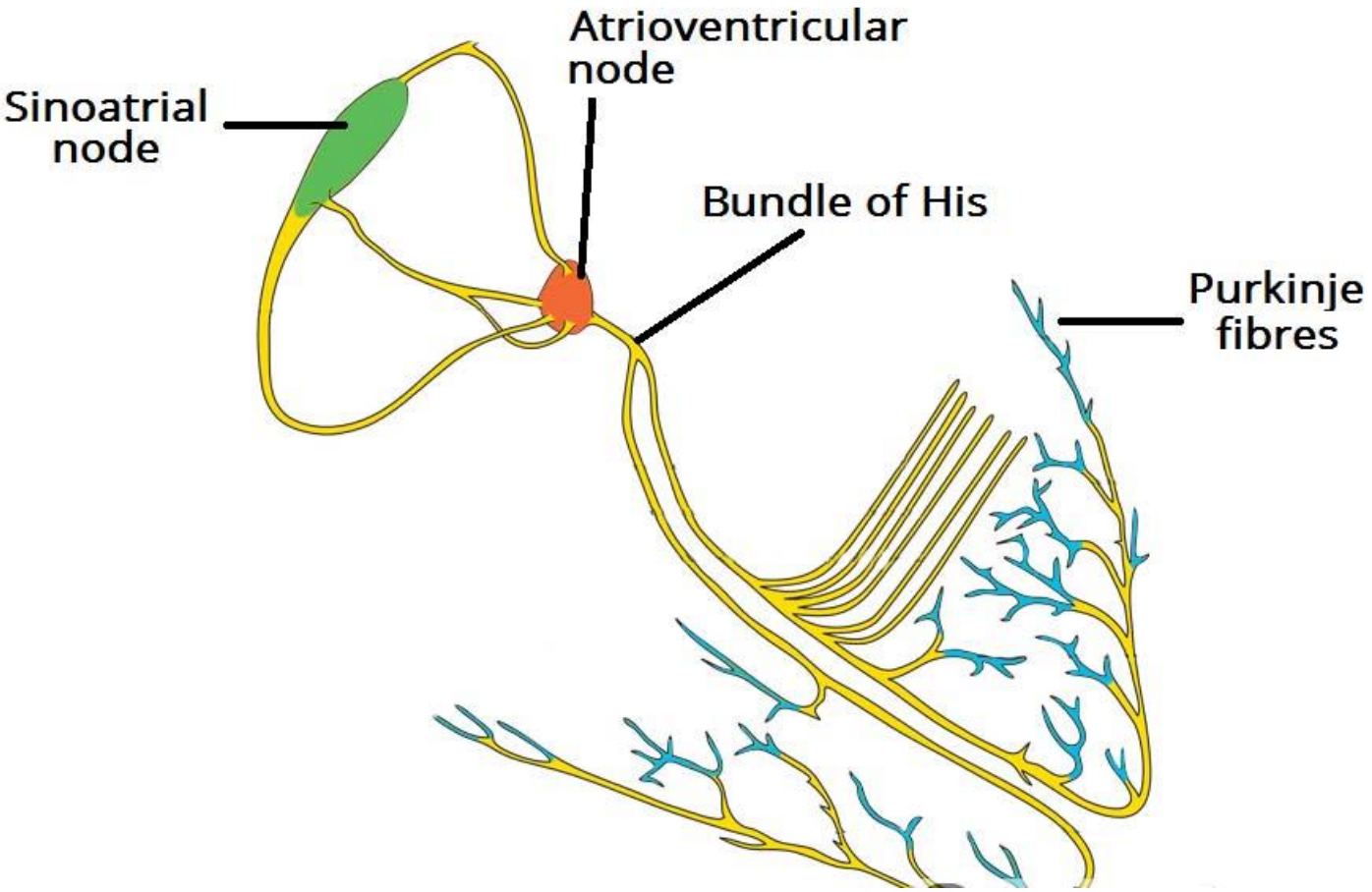

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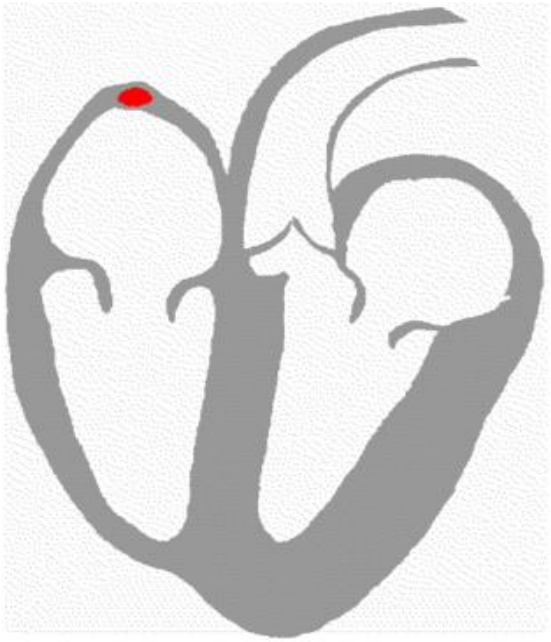

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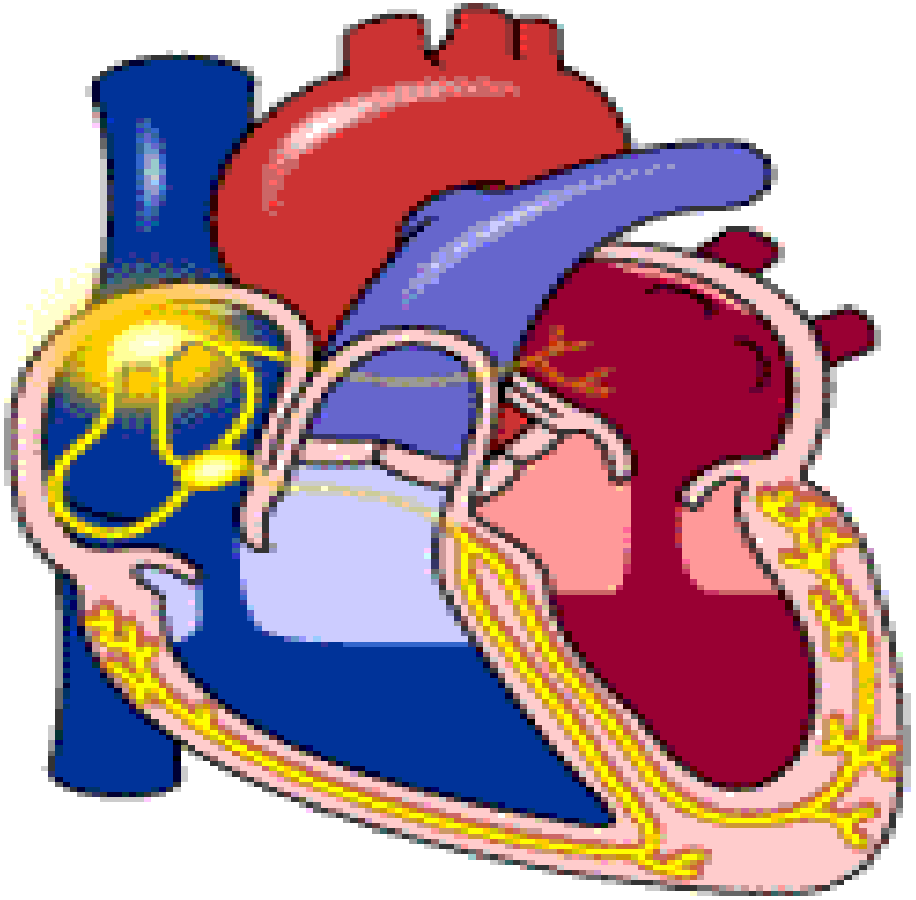
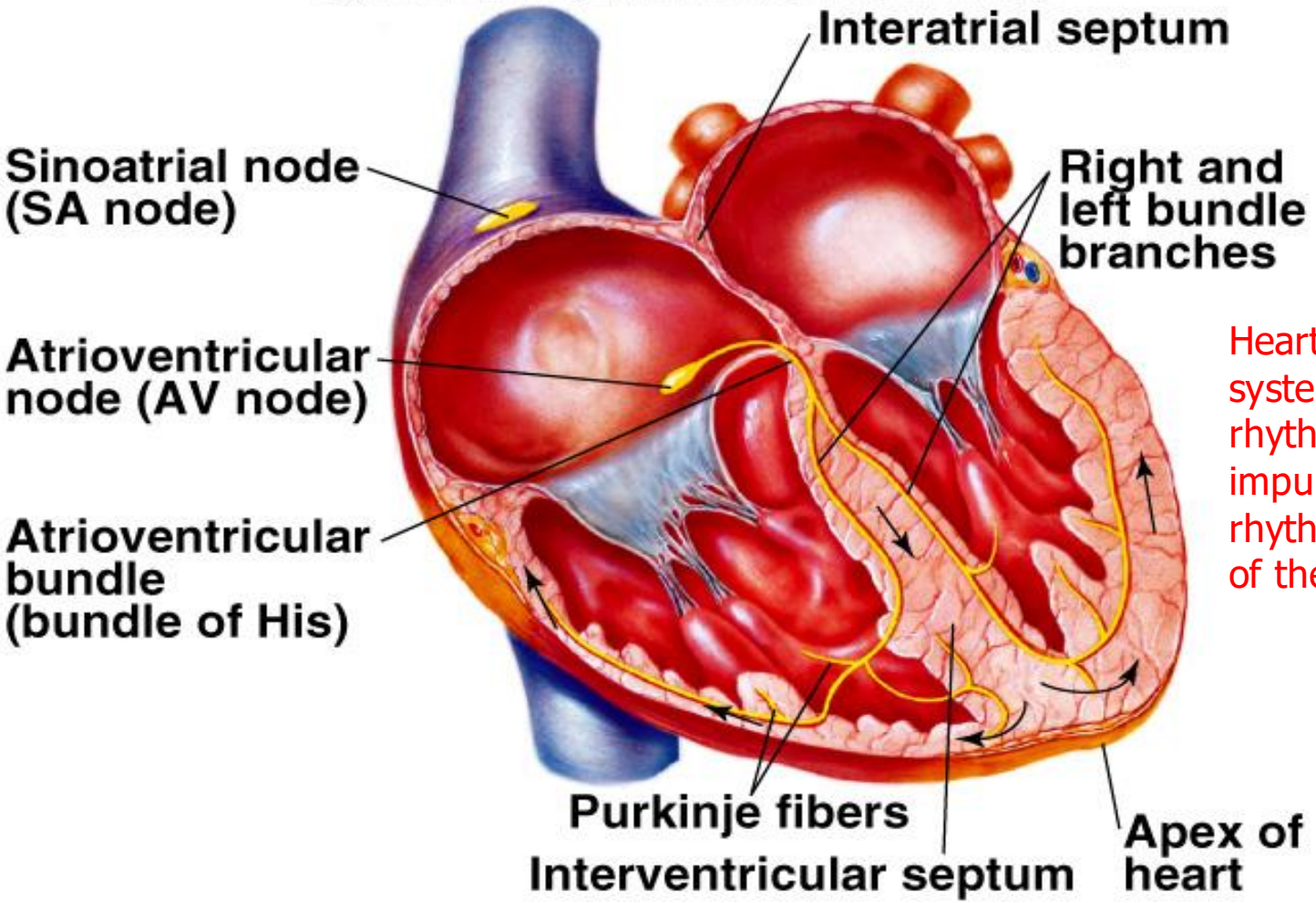
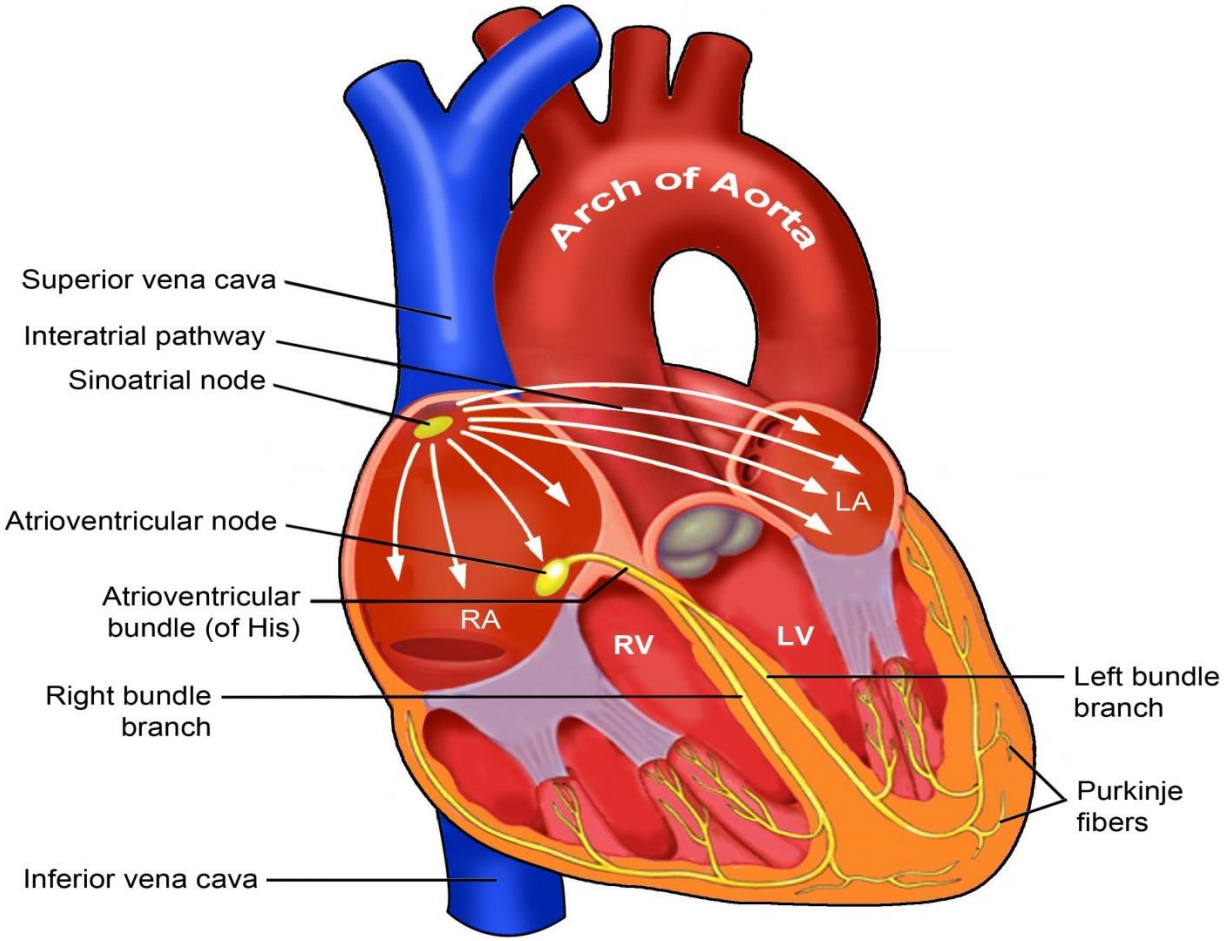


CONDUCTING TISSUES OF THE HEART



Heart has a special system for generating rhythmical electrical impulses to cause rhythmical contraction of the heart muscle.

CONDUCTING TISSUES OF THE HEART



CONDUCTING TISSUES OF THE HEART

Sequence of excitation

Sinus-Atrial Node (SA node)

↓

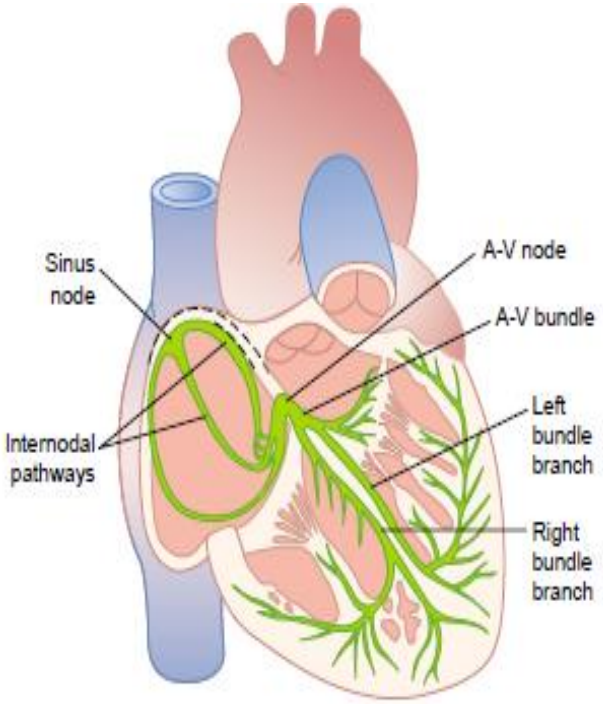
Atria

↓

Atrial-ventricular Node (AV node)

↓

Ventricles



CONDUCTING TISSUES OF THE HEART

Sinoatrial node

↓

Atrial syncytium

↓

Junctional fibers

↓

Atrioventricular node

↓

A-V bundle

↓

Bundle branches

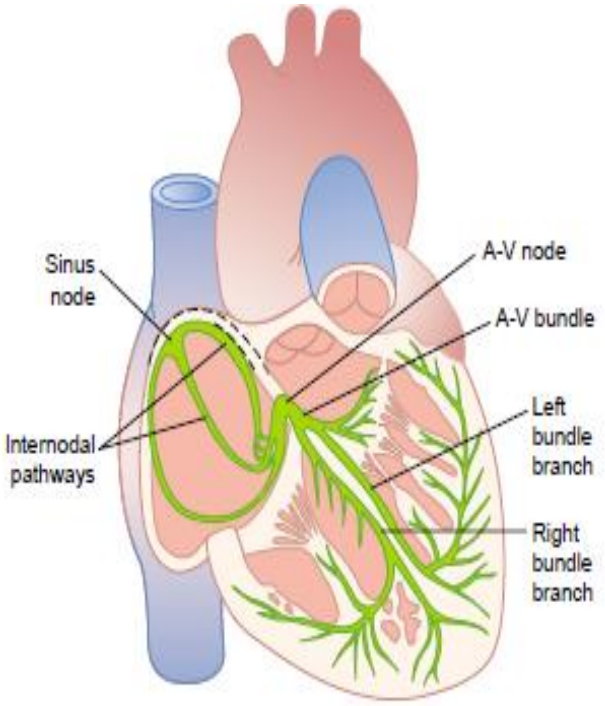
↓

Purkinje fibers

↓

Ventricular syncytium

Sequence of excitation



SA NODE

- The Sinus Node *(Sinoatrial node)* is a small, flattened, specialized cardiac cell
- Known as Pacemaker of the heart.
- Located in the superior posterio-lateral wall of the right atrium
- Responsible for generating the electrical impulses that bring about the mechanical activity i.e contraction of the heart.
- SA node has the fastest rate of autorhythmicity.

CARDIAC IMPULSE FROM SA NODE TO ATRIAL MUSCLE

The cardiac impulse after it's origin in the SA node spreads through out the atrial muscle through two routes

- Ordinary Atrial muscle fibers
- Anterior, middle and posterior conducting bundles
  - Anterior internodal bundle of Bachman
  - Middle internodal bundle of Wenkebach
  - Posterior internodal bundle of Thoral

- These inter nodal pathways conduct the impulses at a faster rate than atrial muscle fibers, because of specialized conduction fibers.

CARDIAC IMPULSE FROM SA NODE TO ATRIAL MUSCLE

- The velocity of conduction in most atrial muscle is about 0.3m/sec.
- In the specialized internodal pathways the conduction velocity may reach upto 1m/sec.
- The impulse after leaving SA node takes 0.03 sec to reach the AV node.

AV NODE

The AV node is located in the posterior wall of the right atrium immediately behind the tricuspid valve.

Slow Conduction in the AV Node:

The cause of slow conduction is mainly diminished number of gap junctions between the successive cells in the conducting pathways.

SIGNIFICANCE OF AV NODAL DELAY

- The cardiac impulse does not travel from the atria to the ventricles too rapidly.
- This delay allows time for the atria to empty their blood into the ventricles before ventricular contraction begins.
- This increases the efficiency of the pumping action of the heart.

PURKINJEE FIBERS

- Purkinje fibers are very large fibers and they transmit AP at a velocity of 1.5 to 4.0 m/sec.
- The rapid transmission of action potentials through the Purkinje fibers is believed to be caused by a very high level of permeability of gap junctions at the intercalated discs between the successive cells of Purkinje fibers.
- The rapid conduction through the purkinje fibers ensures that different parts of ventricles are excited almost simultaneously; this greatly increases the efficiency of heart as a pump.

RIGHT AND LEFT BUNDLE BRANCHES

- Bundle of His splits into two branches which are called right and left bundle branches present on the respective sides of the ventricular septum.

- From the time the cardiac impulse enters the bundle branches until it reaches the terminations of Purkinje fibers , the total time averages only 0.03 sec.

CONDUCTION OF IMPULSE

- APs from SA node spread quickly at rate of 0.8 - 1.0 m/sec.
- Time delay occurs as impulses pass through AV node.
  - Slow conduction of 0.03 – 0.05 m/sec.
- Impulse conduction increases as spread to Purkinje fibers at a velocity of 4.0 m/sec.
  - Ventricular contraction begins 0.1–0.2 sec. after contraction of the atria.

ONE- WAY CONDUCTION THROUGH AV BUNDLE

- A special characteristic of the A-V bundle is it's inability of action potentials to travel backward from the ventricles to the atria.
- This prevents re-entry of cardiac impulse by this route from the ventricles to the atria.
- The atrial muscle is separated from the ventricular muscle by a continuous fibrous barrier which acts as an insulator to prevent the passage of cardiac impulse between the atrial and ventricular muscle

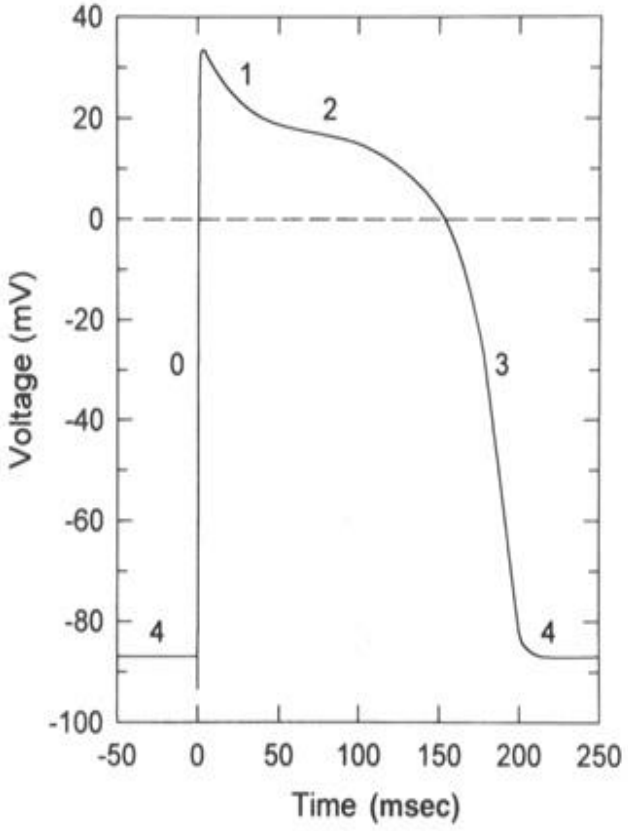
ACTION POTENTIAL IN THE CARDIAC MUSCLE

The cardiac action potential is made of 3 phases:

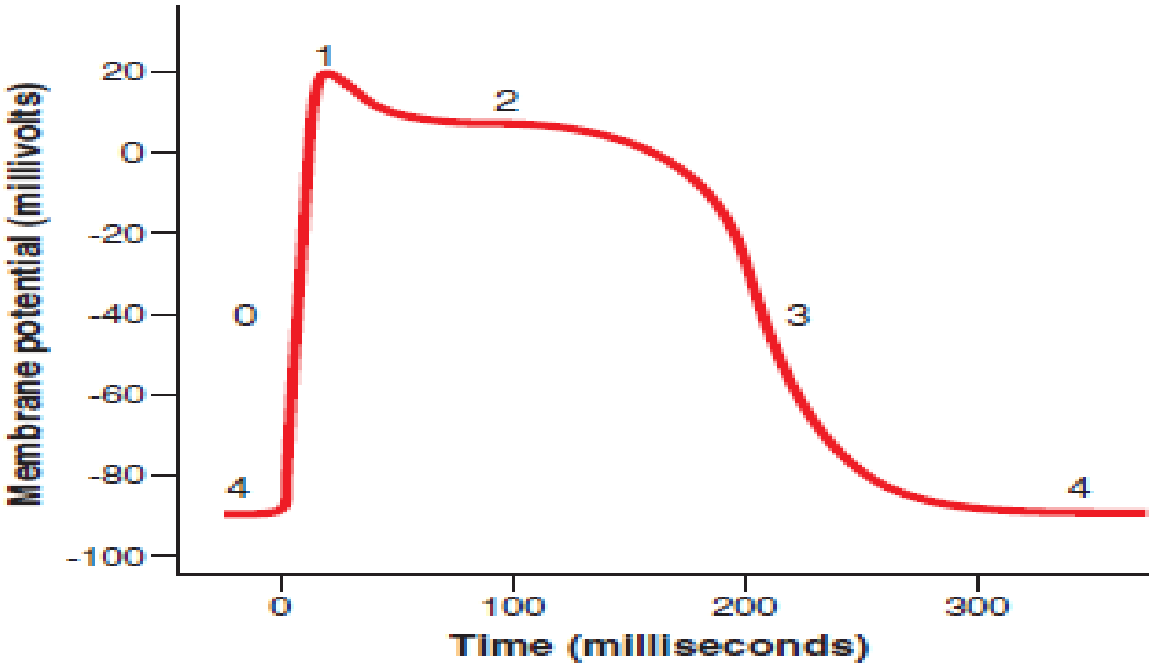
Depolarization: caused by the opening of Fast Na channels & slow Ca channels

Plateau: remaining of slow Ca channels open for several m seconds, drawing large amount of Ca inside which prolong depolarization

Replarization: Opening of potassium channels



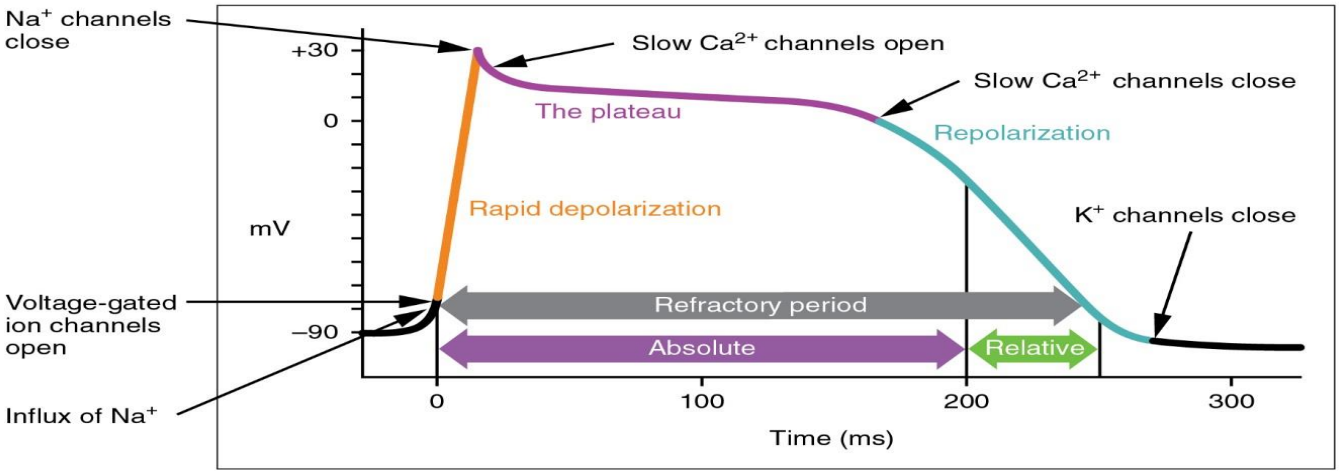
ACTION POTENTIAL IN THE CARDIAC MUSCLE



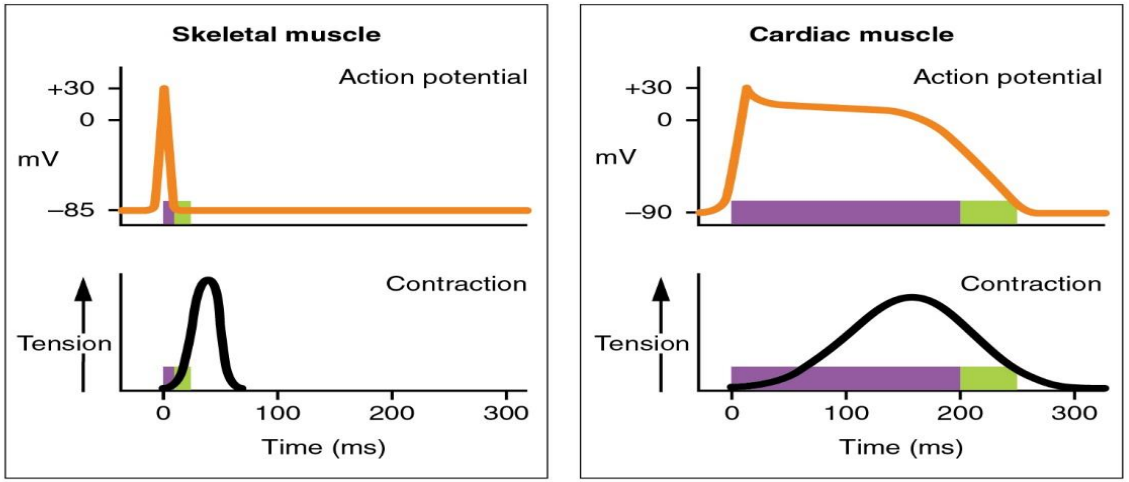
The presence of Plateau in the action potential causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle

Guyton pp 110

ACTION POTENTIAL IN THE CARDIAC MUSCLE



(a)



(b)

ACTION POTENTIAL IN THE CARDIAC MUSCLE

Phase 0 (depolarization): *Fast sodium channels open.* Voltage gated sodium channels (fast sodium channels) open and cell depolarize. Membrane potential reaches about +20 millivolts before the sodium channels close.

Phase 1 (initial repolarization), *fast sodium channels close*. Cell begins to repolarize, and potassium ions leave the cell through open potassium channels.

Guyton and Hall, pp 111

ACTION POTENTIAL IN THE CARDIAC MUSCLE

Phase 2 (Plateau), *calcium channels open and fast potassium channels close*. Initial repolarization Occurs. Potassium ion efflux and increased calcium ion influx causes the action potential to plateau.

Phase 3 (rapid repolarization), *calcium channels close and slow potassium channels open*. The closure of calcium ion channels and increased potassium ion permeability, permitting potassium ions to rapidly exit the cell

Phase 4 (resting membrane potential) *averages about –90 millivolts.*

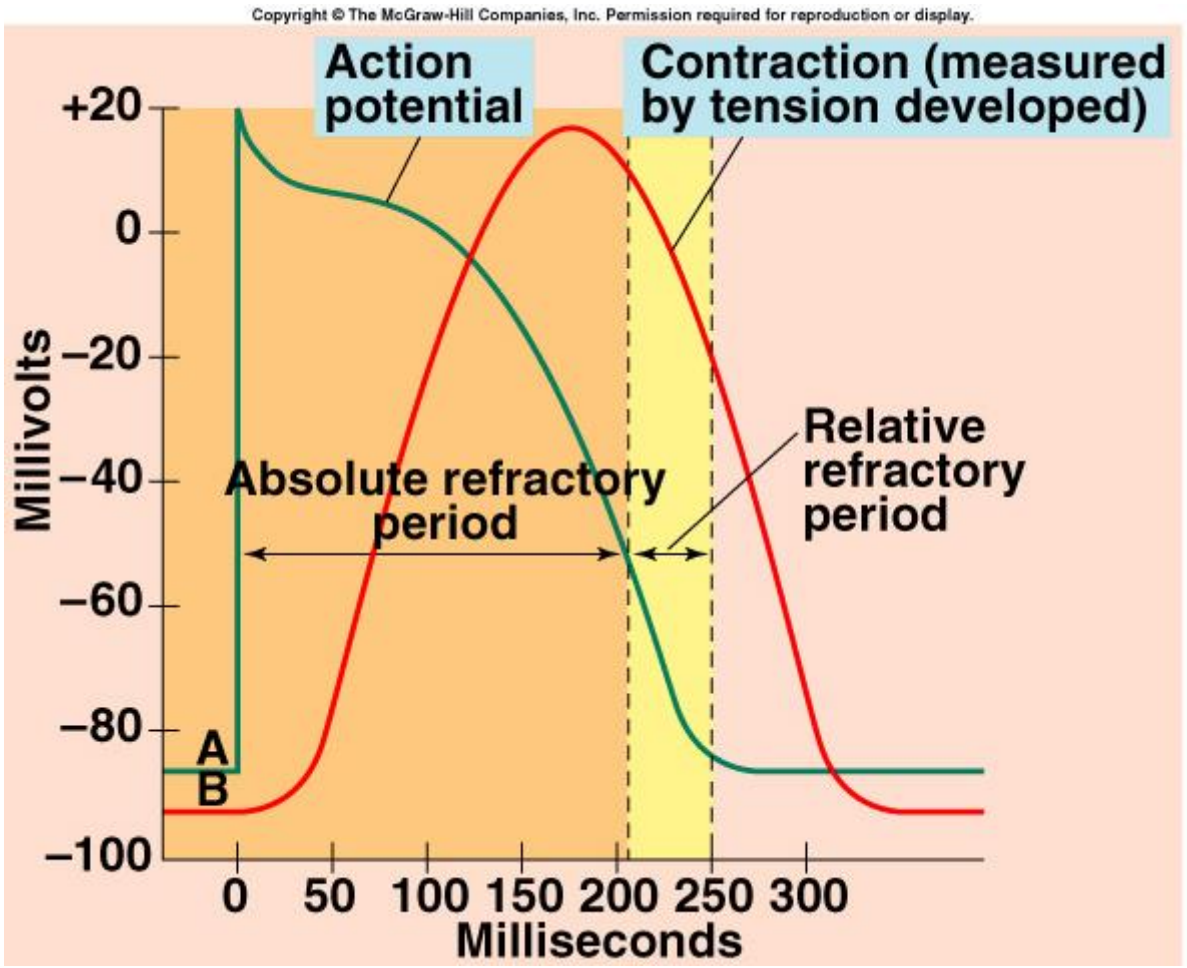
Guyton and Hall, pp 111

WHY PLATEAU OCCURS

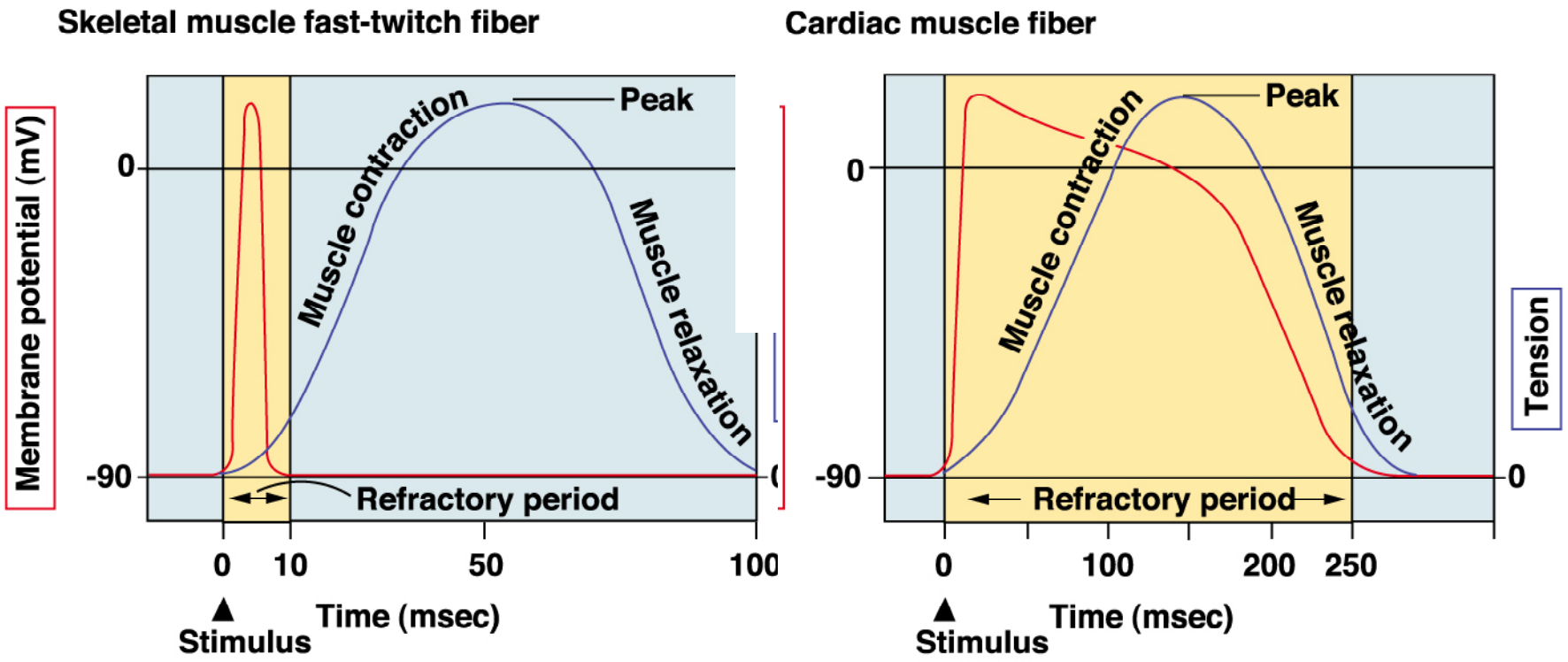
- Voltage activated sodium channels, called *fast channels*
- Voltage-activated calcium-sodium channels *(L-type calcium)*, slow to open, called *slow channels.*
- Opening of fast channels causes spike of AP
- Prolonged opening of the slow calcium-sodium channels allows calcium to enter, cause plateau.
- Moreover, voltage-gated potassium channels are slower to open. This delays the return of the membrane potential to −80 to −90 millivolts.

Refractory Periods

- Heart contracts as syncytium.
- Contraction lasts almost 300 msec.
- Refractory periods last almost as long as contraction.
- Myocardial muscle cannot be stimulated to contract again until it has relaxed.
- Summation cannot occur.



Refractory Periods



- The refractory period is short in skeletal muscle, but very long in cardiac muscle.
- This means that skeletal muscle can undergo summation and tetanus, via repeated stimulation
- Cardiac muscle **CAN NOT** undergo sum action potentials or contractions and can't be tetanized

Refractory Periods

- Long refractory period prevent ventricles from contracting at too high rates so that enough time is allowed for refill of the ventricles
- Because long refractory period occurs in conjunction with prolonged plateau phase, **summation and tetanus of cardiac muscle is impossible**
- Ensures alternate periods of contraction and relaxation which are essential for pumping blood

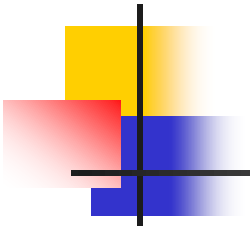
Conduction speed in Cardiac tissues

Tissue	Conduction Rate (m/s)
SA node	0.05
Atrial pathways	1
AV node	0.05
Bundle of His	1
Purkinje system	4
Ventricular muscle	1

Action Potentials: Skeletal Muscle vs Cardiac Muscle

TABLE 14-3 Comparison of Action Potentials in Cardiac and Skeletal Muscle

	SKELETAL MUSCLE	CONTRACTILE MYOCARDIUM	AUTORHYTHMIC MYOCARDIUM
Membrane potential	Stable at −70 mV	Stable at −90 mV	Unstable pacemaker potential; usually starts at −60 mV
Events leading to threshold potential	Net Na^+ entry through ACh-operated channels	Depolarization enters via gap junctions	Net Na^+ entry through I_f channels; reinforced by Ca^{2+} entry
Rising phase of action potential	Na^+ entry	Na^+ entry	Ca^{2+} entry
Repolarization phase	Rapid; caused by K^+ efflux	Extended plateau caused by Ca^{2+} entry; rapid phase caused by K^+ efflux	Rapid; caused by K^+ efflux
Hyperpolarization	Due to excessive K^+ efflux at high K^+ permeability when K^+ channels close; leak of K^+ and Na^+ restores potential to resting state	None; resting potential is −90 mV, the equilibrium potential for K^+	Normally none; when repolarization hits −60 mV, the I_f channels open again. ACh can hyperpolarize the cell.
Duration of action potential	Short: 1–2 msec	Extended: 200+ msec	Variable; generally 150+ msec
Refractory period	Generally brief	Long because resetting of Na^+ channel gates delayed until end of action potential	None



THANK YOU