Cardiac Muscle Action Potential

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Learning Objectives:

1. Explain phases of Cardiac muscle action potential.
2. Describe the characteristics of cardiac action potentials and the role of “slow calcium” channels in causing plateau and its significance.
3. Describe the significance of AV nodal Delay.
4. Define Pacemaker and explain why SA node is the normal pacemaker of the heart.
5. Define various types of refractory periods.
6. Differentiate the refractory period of cardiac muscle with that of skeletal muscle.

Learning Objectives:

7. Describe the significance of prolonged action potential in cardiac muscle.
8. Describe the specialized excitatory and conductive pathway of the cardiac muscle tissue.
9. Describe the effects of Sympathetic and Parasympathetic system on Cardiac conduction, Heart rate and Contractility.

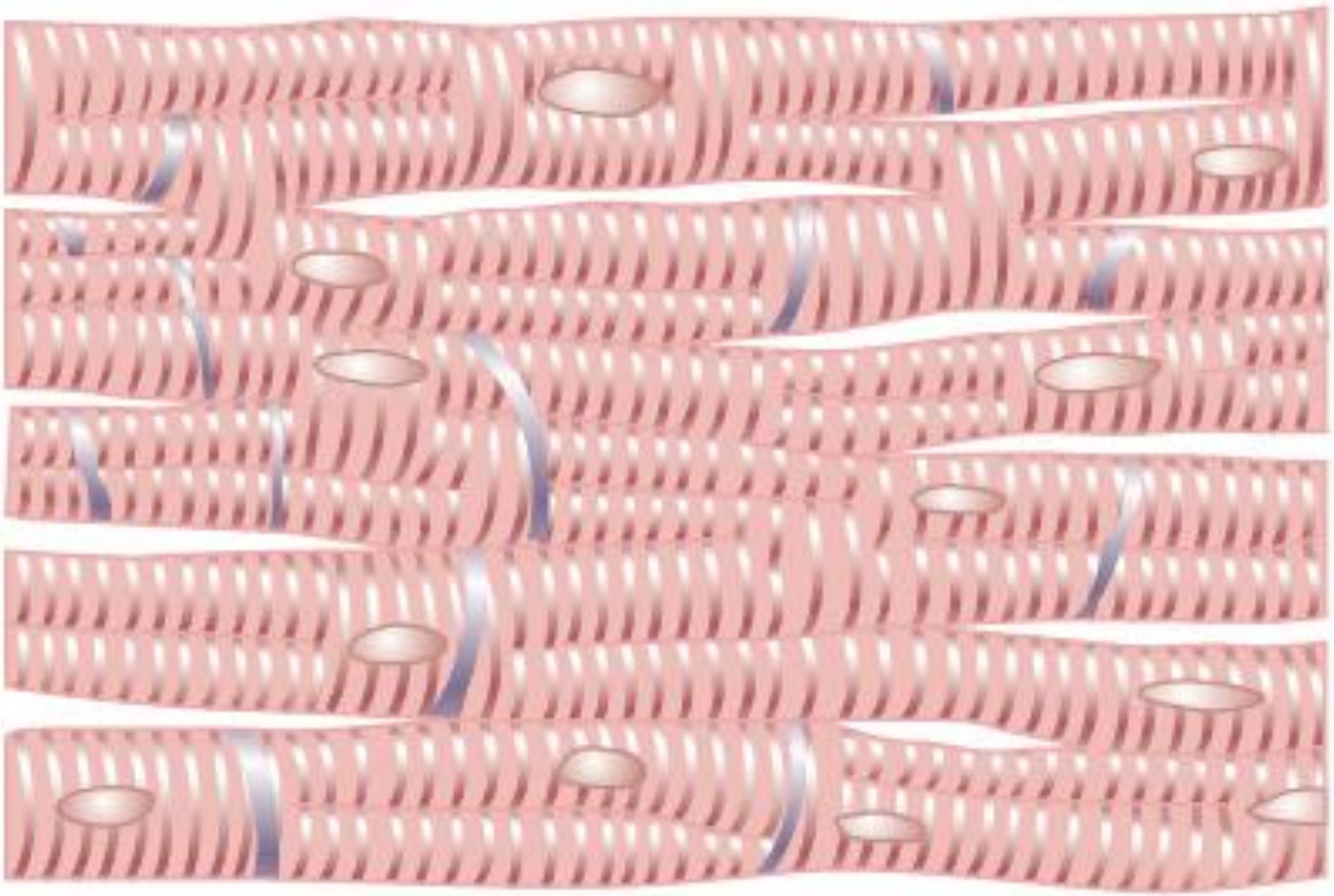
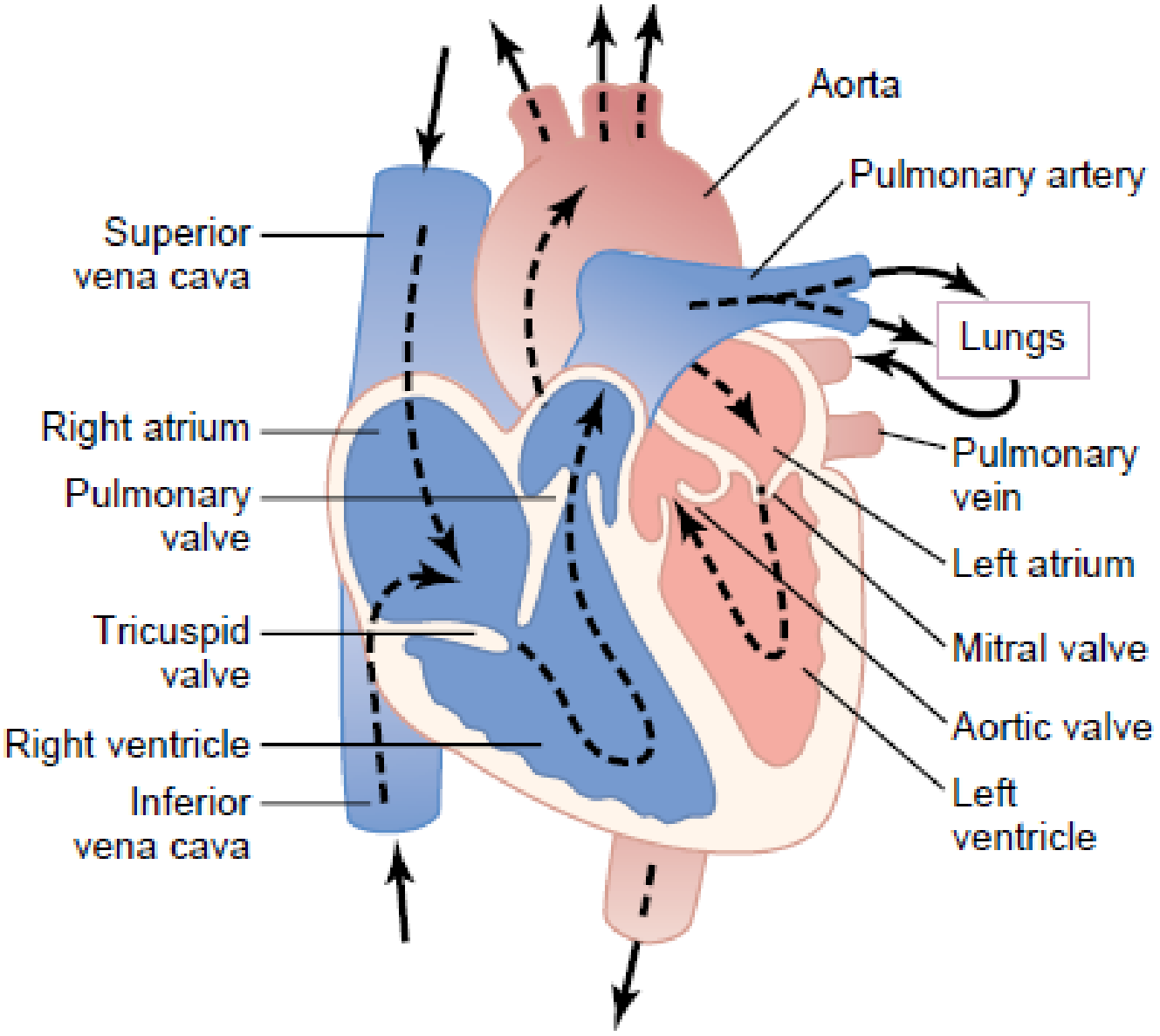


Figure 9–2

"Syncytial," interconnecting nature of cardiac muscle fibers.

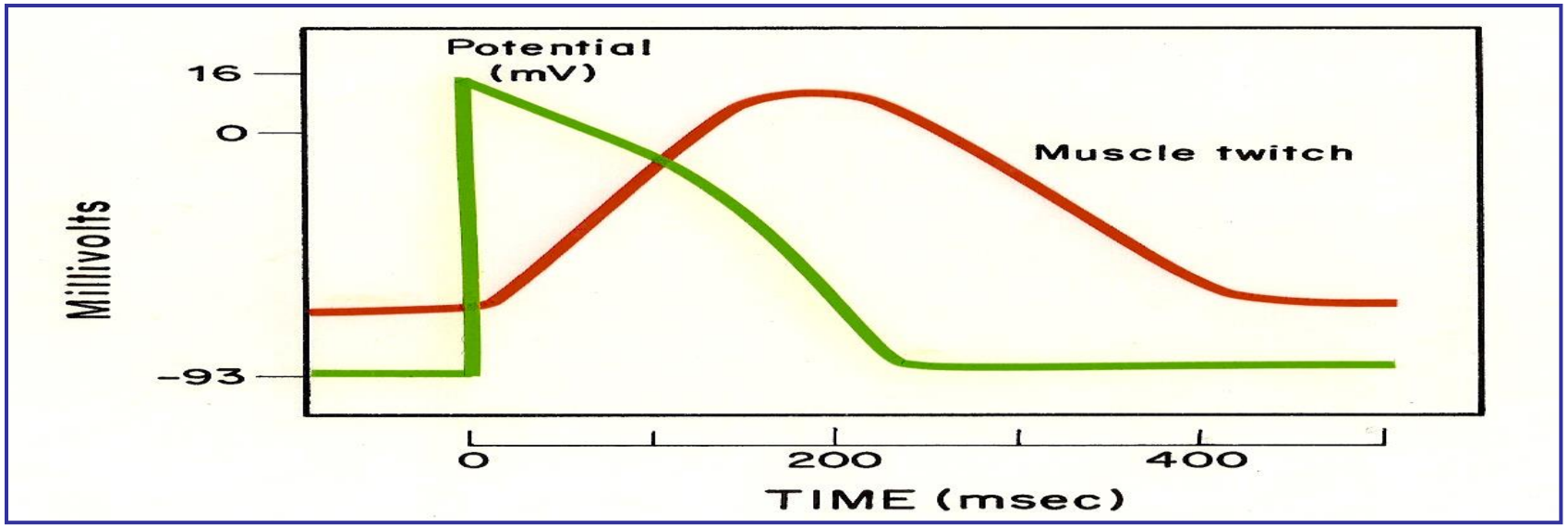

Aorta
Pulmonary artery
Superior vena cava
Lungs
Right atrium
Pulmonary valve
Pulmonary vein
Left atrium
Tricuspid valve
Mitral valve
Right ventricle
Aortic valve
Inferior vena cava
Left ventricle

Properties of the cardiac muscle:

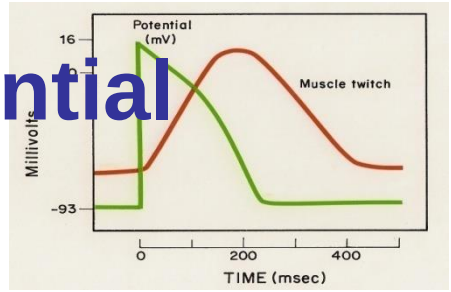
I. Excitability
II. Conductivity
III. Contractility
IV. Rhythmicity

I. Excitability (Irritability):

= the ability of cardiac ms to respond to adequate stimuli by generating an action potential followed by a mechanical contraction.



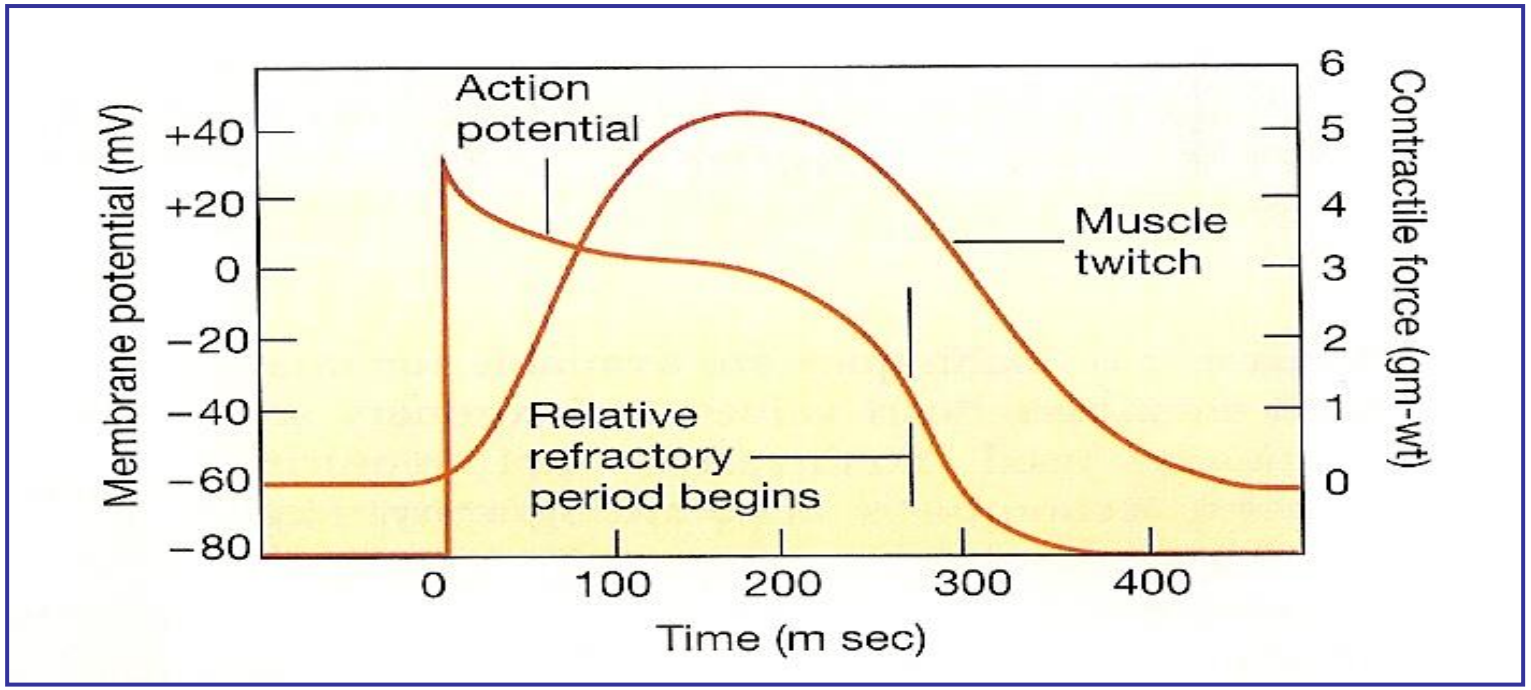
Relation between the action potential & the mechanical response



- The mechanical response consists of contraction (systole) & relaxation (diastole).
- Cardiac ms begins to contract few milliseconds after the AP begins, & continues to contract until few milliseconds after the AP ends.
- Duration of contraction:
  - ≈ 0.2 sec in arial muscle, &
  - ≈ 0.3 sec in ventricular muscle.

Relation between the action potential & the mechanical response

- Diastole begins at the end of the plateau.



Cardiac Action Potential

- RMP is determined by K^+ conductance & approaches the K^+ equilibrium potential

- Inward current---positive charge in---cell depolarize
- Outward current---positive charge out---cell repolarize

Cardiac Action Potential

- Na – K atpase maintain the ionic gradients across the cell membrane

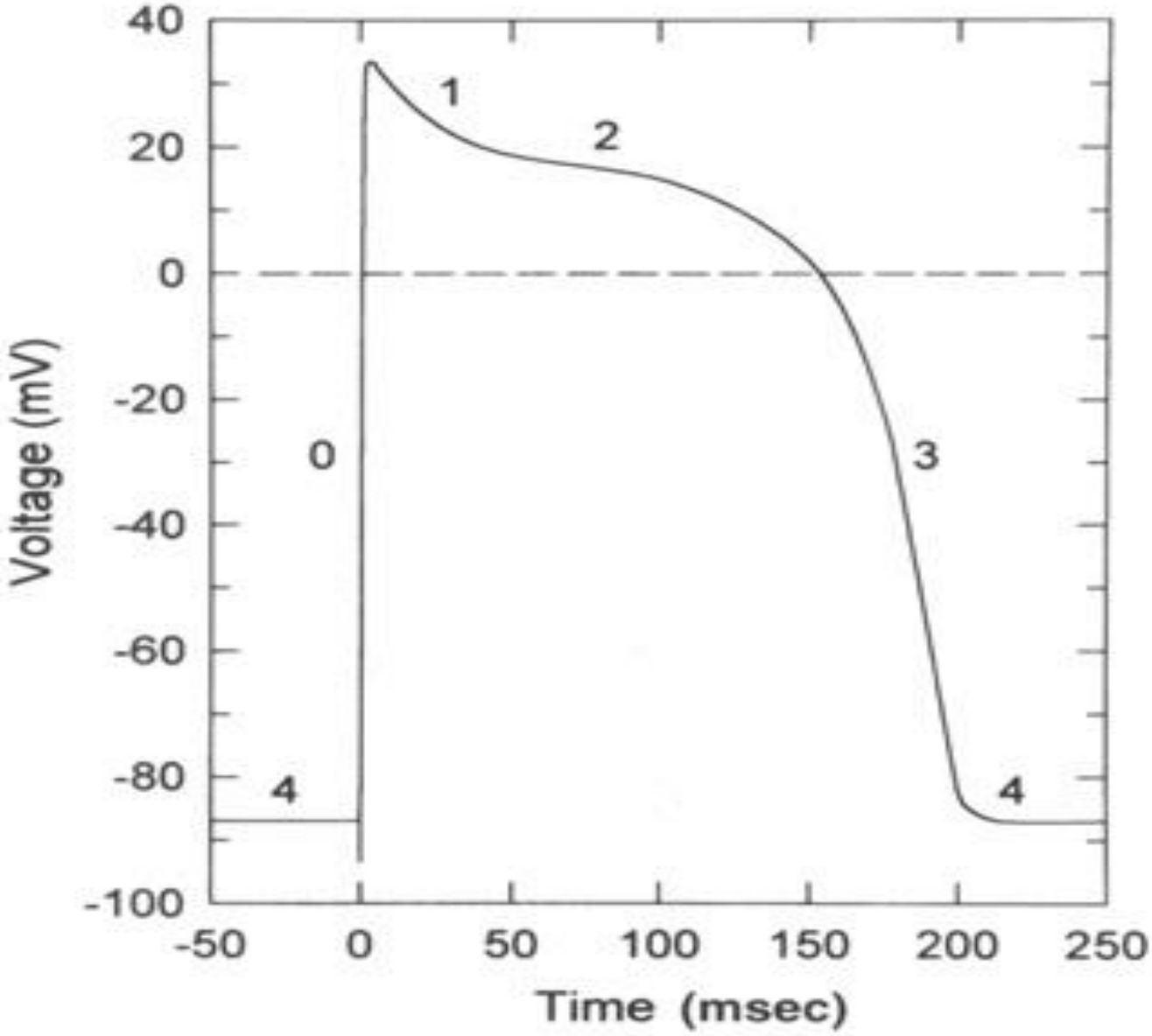
Ventricles, Atria & Purkinje System

- Have stable resting potential of -85 to -95 mv which represents the K+ equlibrium potential.

Phases of Action potential in Ventricular Muscle

PHASE 0:

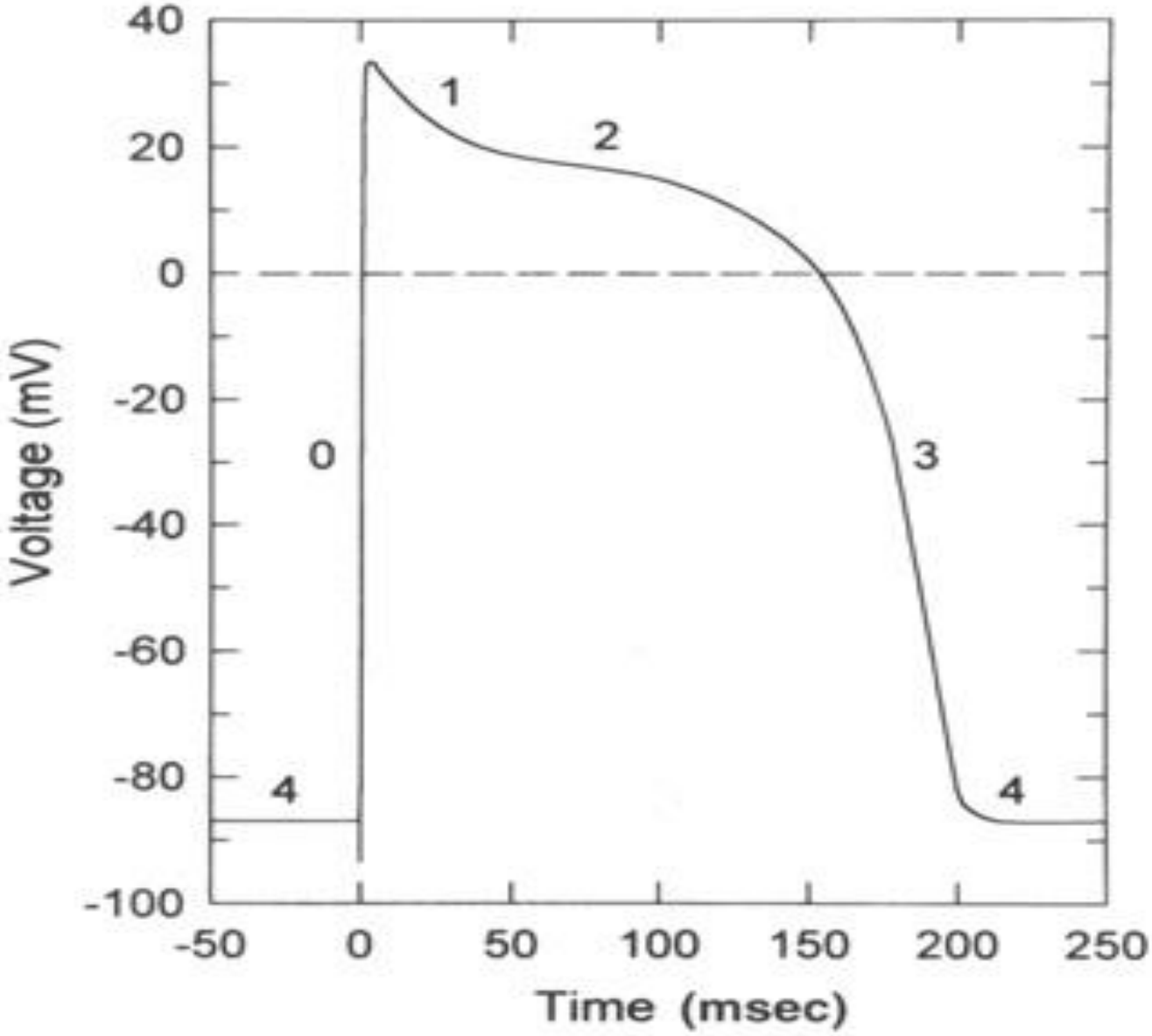
- Upstroke of AP
- Opening of fast Na+ channels
- Inward Na+ current due to increase Na+ conductance.


40
20
0
-20
-40
-60
-80
-100
Voltage (mV)
-50
0
50
100
150
200
250
Time (msec)
0
1
2
3
4
4

Phases of Action Potential in Ventricular Muscle

PHASE 1:

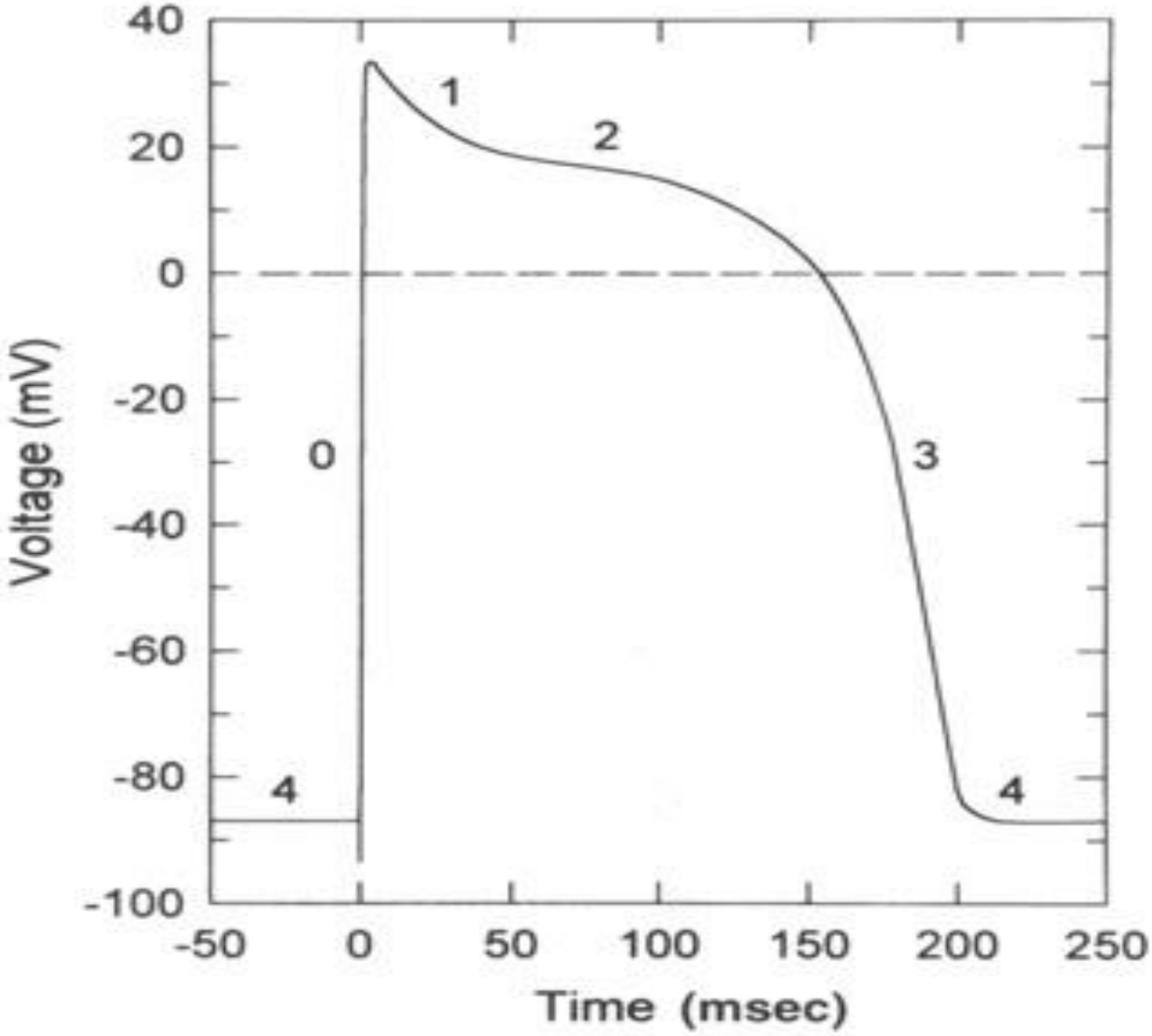
- Brief period of initial repolarization.
- Due to decrease Na conductance.
- Due to an outward current due to K efflux.


40
20
0
-20
-40
-60
-80
-100
Voltage (mV)
-50
0
50
100
150
200
250
Time (msec)
0
1
2
3
4
4

Phases of Action potential

PHASE 2:

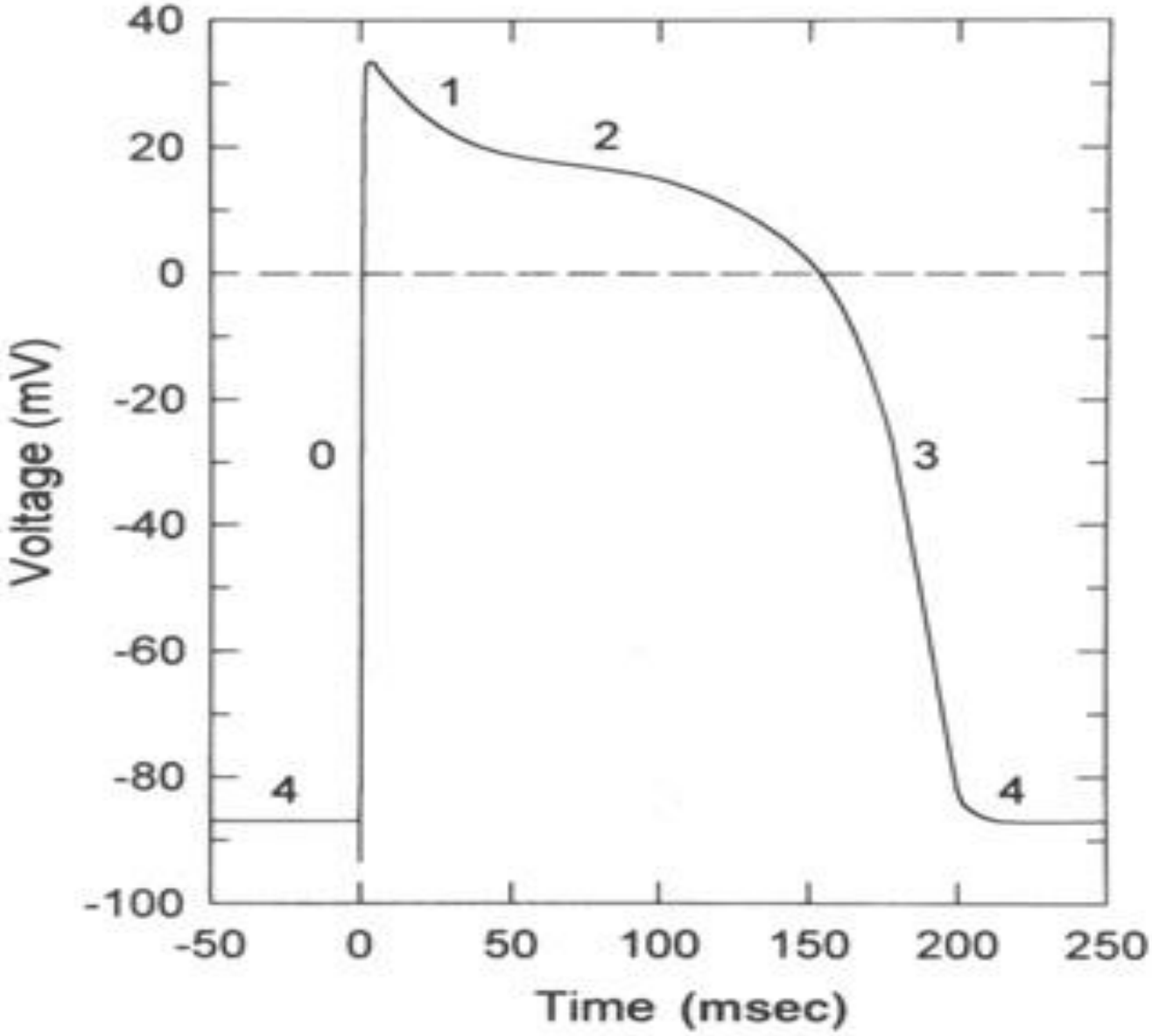
- Plateau of AP
- Opening of slow Na+ & Ca+ channels
- Transient inc in Ca+ & Na+ conductance.
- Inward Ca+ current.


40
20
0
-20
-40
-60
-80
-100
Voltage (mV)
-50
0
50
100
150
200
250
Time (msec)
0
1
2
3
4
4

Phases of Action potential

PHASE 3:

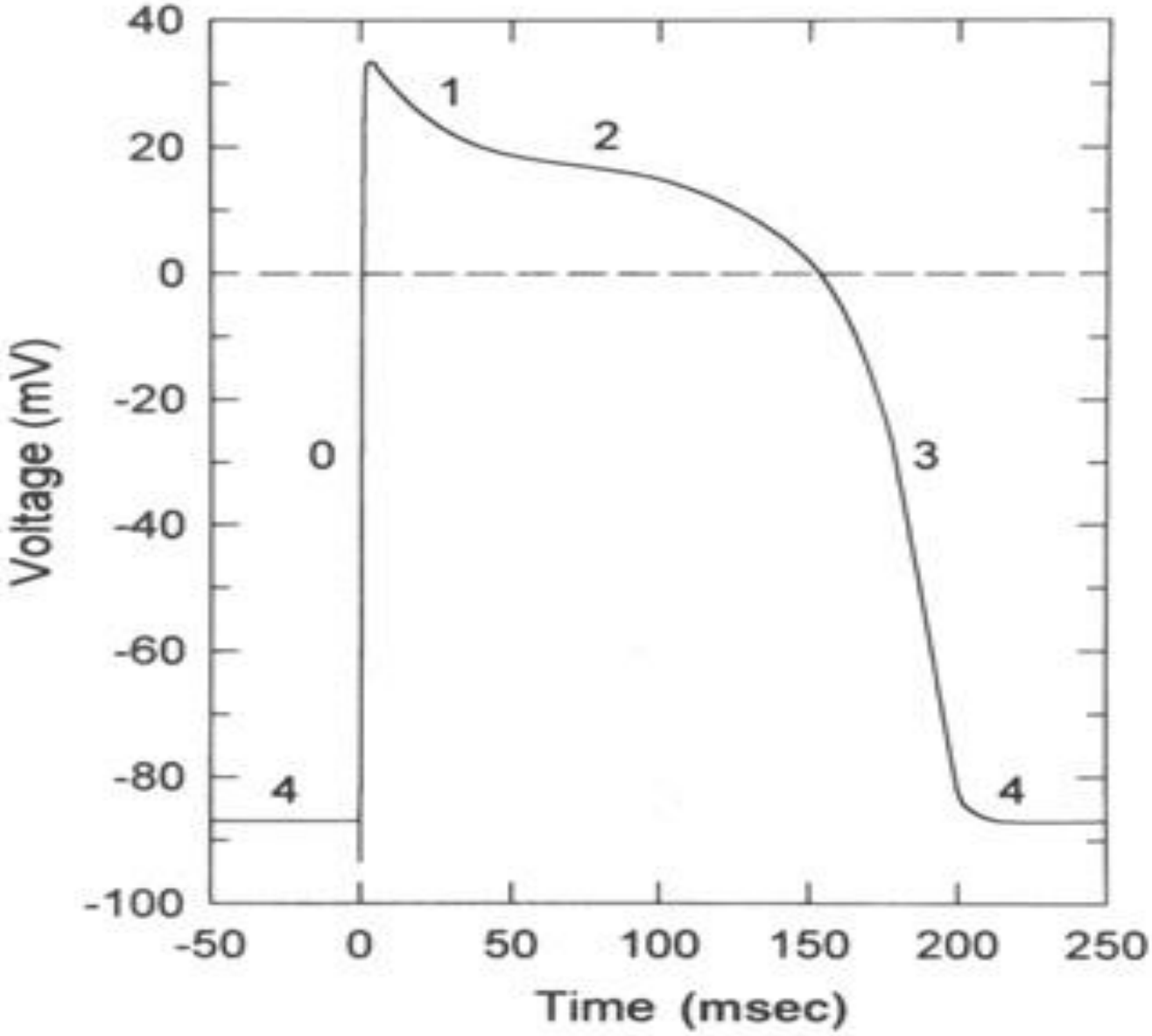
- Is repolarization.
- Ca+ & Na+ conductance dec & K+ conductance inc.
- Large K+ outward current leads to hyperpolarization of the membrane toward K+ equilibrium potential.


40
20
0
-20
-40
-60
-80
-100
Voltage (mV)
-50
0
50
100
150
200
250
Time (msec)
0
1
2
3
4
4

Phases of Action potential

PHASE 4:

- Is resting membrane potential.
- Inward & outward current is equal.
- Membrane potential approaches K+ equilibrium potential.


40
20
0
-20
-40
-60
-80
-100
Voltage (mV)
-50
0
50
100
150
200
250
Time (msec)
0
1
2
3
4
4

AP in Nodal Tissues

- SA node is the pacemaker of the heart have an unstable RMP.
- SA node shows phase 4 depolarization or automaticity.
- AV node, & His- purkinje systems are latent pacemakers that may exhibit automaticity & overide SA node if suppressed.

AP in Nodal Tissues

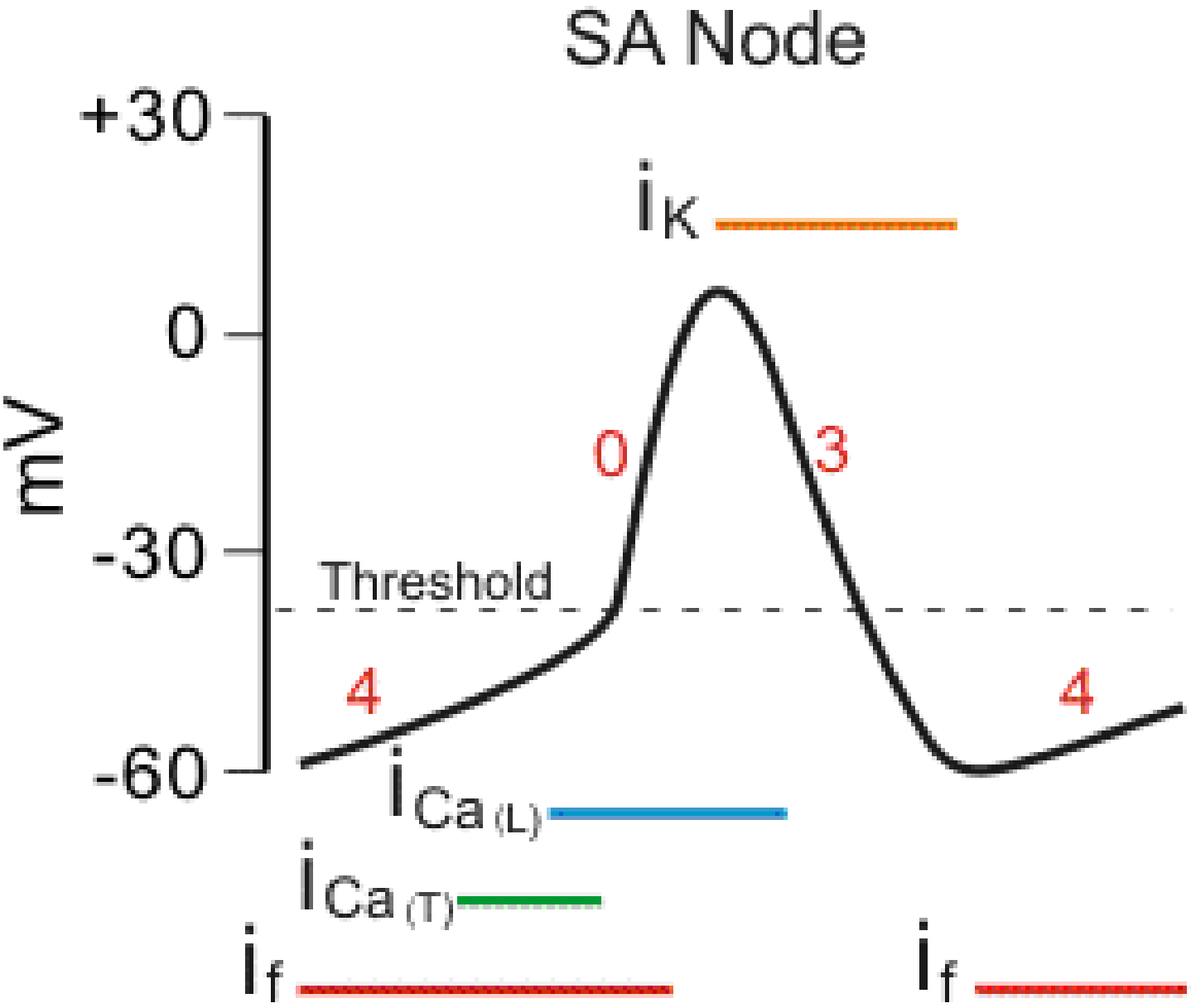
- Intrinsic rate of phase 4 depolarization & heart rate is fastest in the SA node & slowest in the His – purkinje system.

- SA node > AV node > His purkinje

Phases of AP in SA node

PHASE 0:

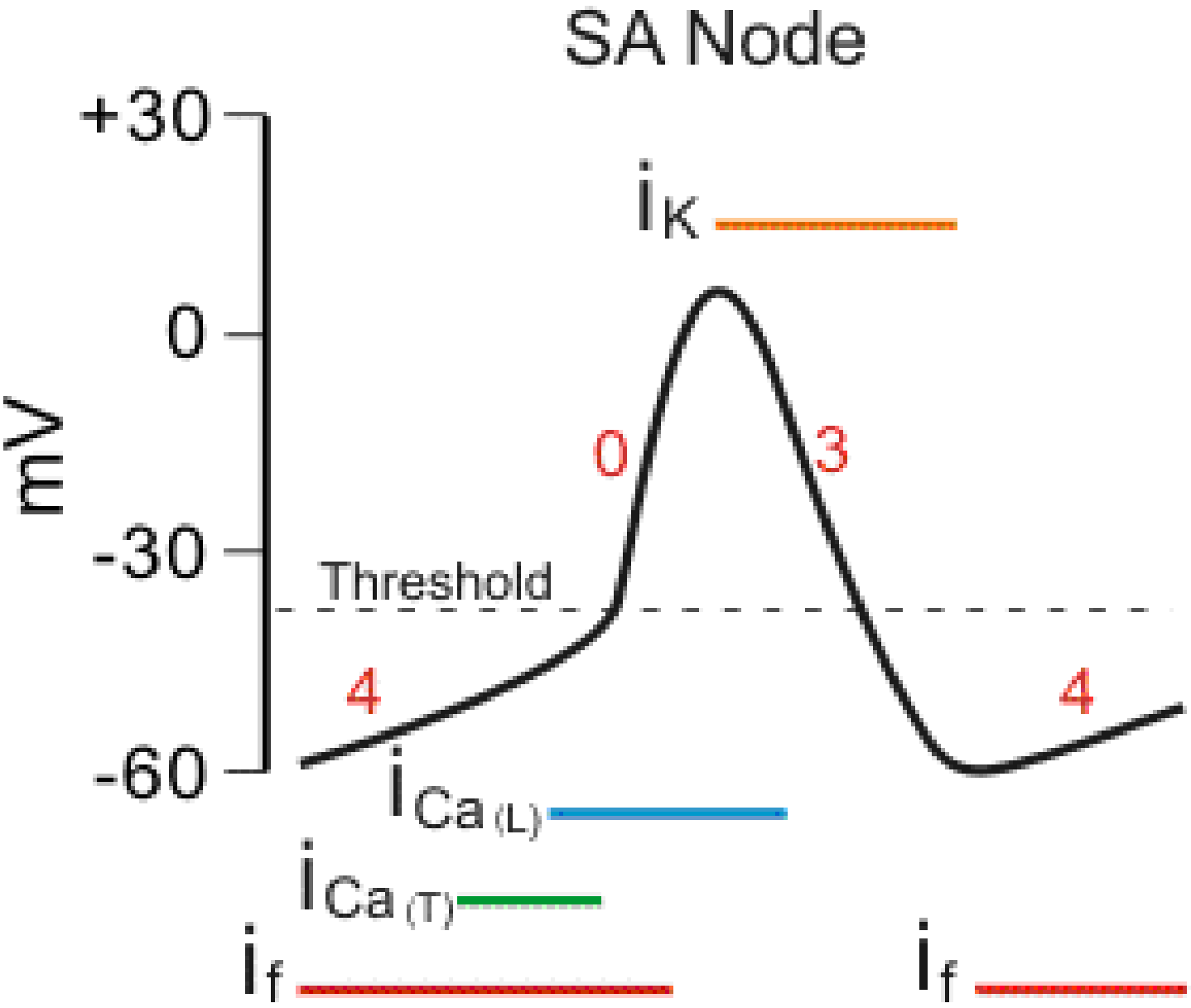
- Upstroke of the AP
- Inc Ca+ conductance
- Inward Ca+ current
- Differ from ventricles, atria & purkinje fibers.


SA Node
+30
0
-30
-60
mV
Threshold
i_K
0
3
4
4
$i_{Ca\ (L)}$
$i_{Ca\ (T)}$
i_f
i_f

Phases of AP in SA node

PHASE 3:

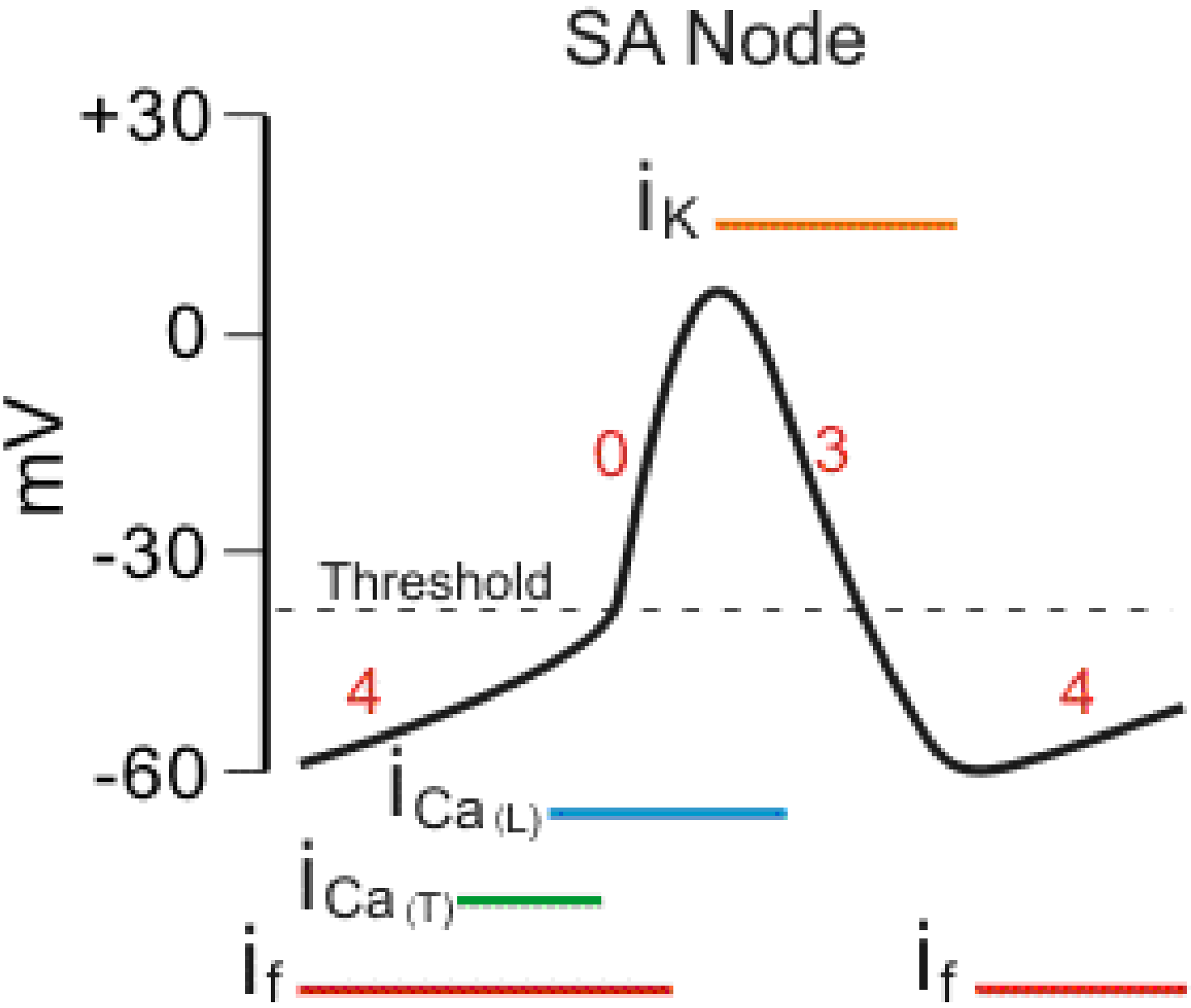
- Repolarization
- Inc K+ conductance
- Outward K+ current


SA Node
+30
0
-30
-60
mV
Threshold
i_K
0
3
4
4
$i_{Ca\ (L)}$
$i_{Ca\ (T)}$
i_f
i_f

Phases of AP in SA node

PHASE 4:

- Slow depolarization
- Accounts for pacemaker activity (automaticity)
- Inc Na+ conductance --- inward Na+ current


SA Node
+30
0
-30
-60
mV
Threshold
i_K
0
3
4
4
$i_{Ca\ (L)}$
$i_{Ca\ (T)}$
i_f
i_f

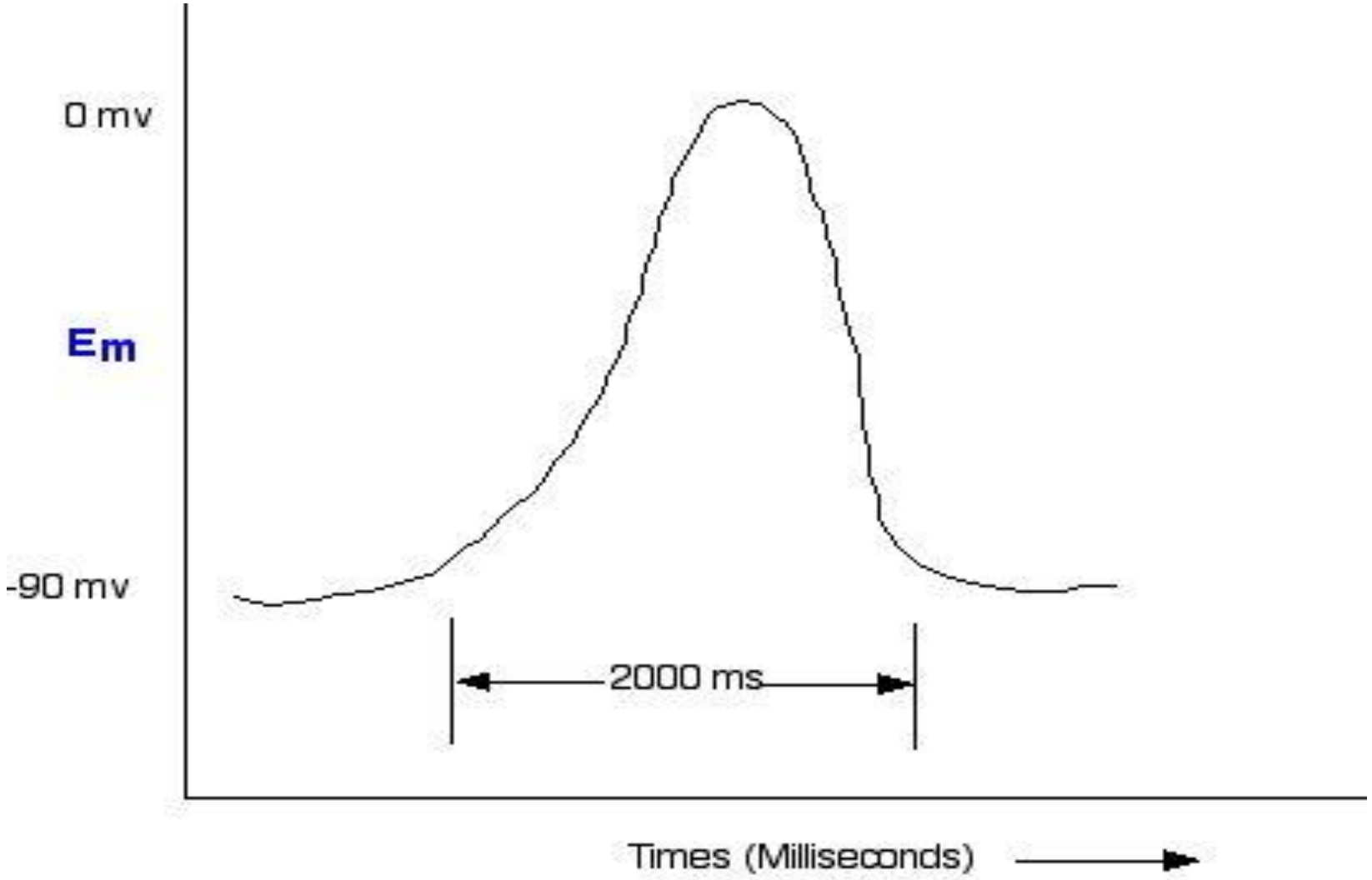
Phases of AP in SA node

PHASE I & II:

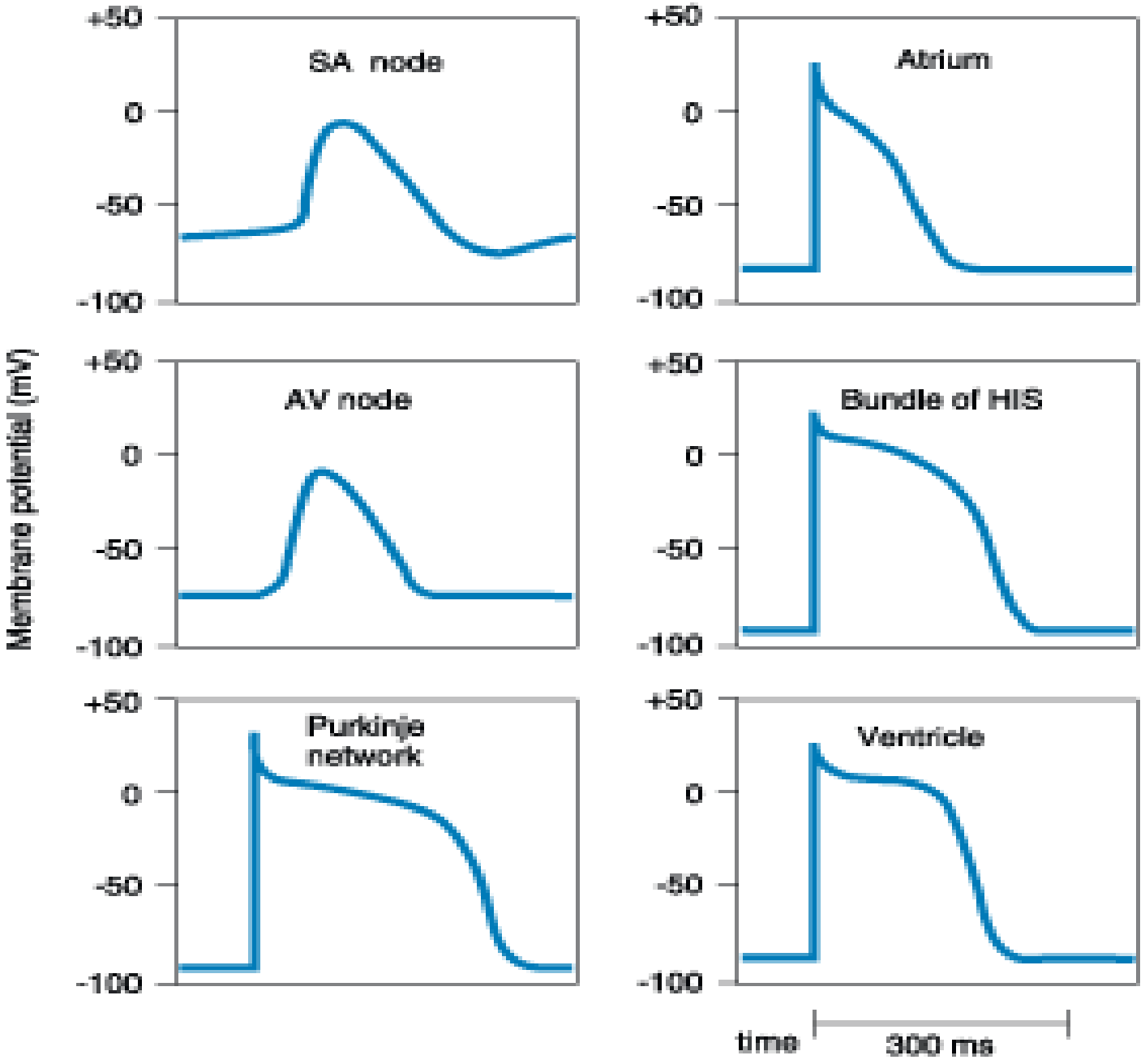
- Absent in SA node

Action Potential in AV node

- Upstroke in AV node is due to the result of inward Ca+ current (as in SA node)



A-V Node Action Potential


Membrane potential (mV)
+50
0
-50
-100
SA node
Atrium
AV node
Bundle of HIS
Purkinje network
Ventricle
time
300 ms

Conduction Velocity

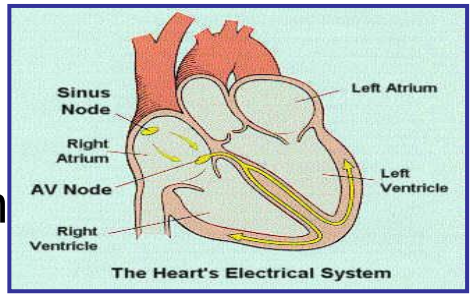
- Time required for excitation to spread throughout the cardiac tissue.
- Depends on the size of inward current
- Larger inward current --- higher conduction velocity.

Conduction Velocity

- Is fastest in purkinje system.
- Is slowest in AV bundle.(Allowing time for ventricular filling before ventricular contraction)

- If CV inc in AV bundle then ventricular filling may be compromised.

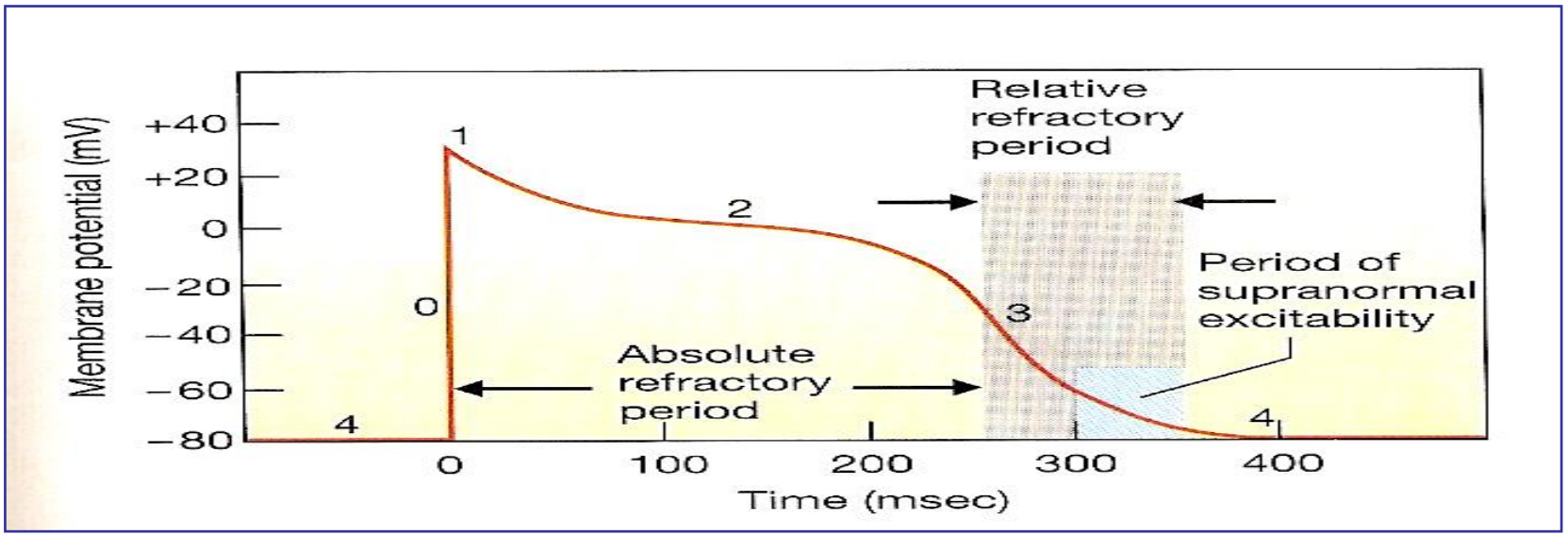
The conduction velocities (con



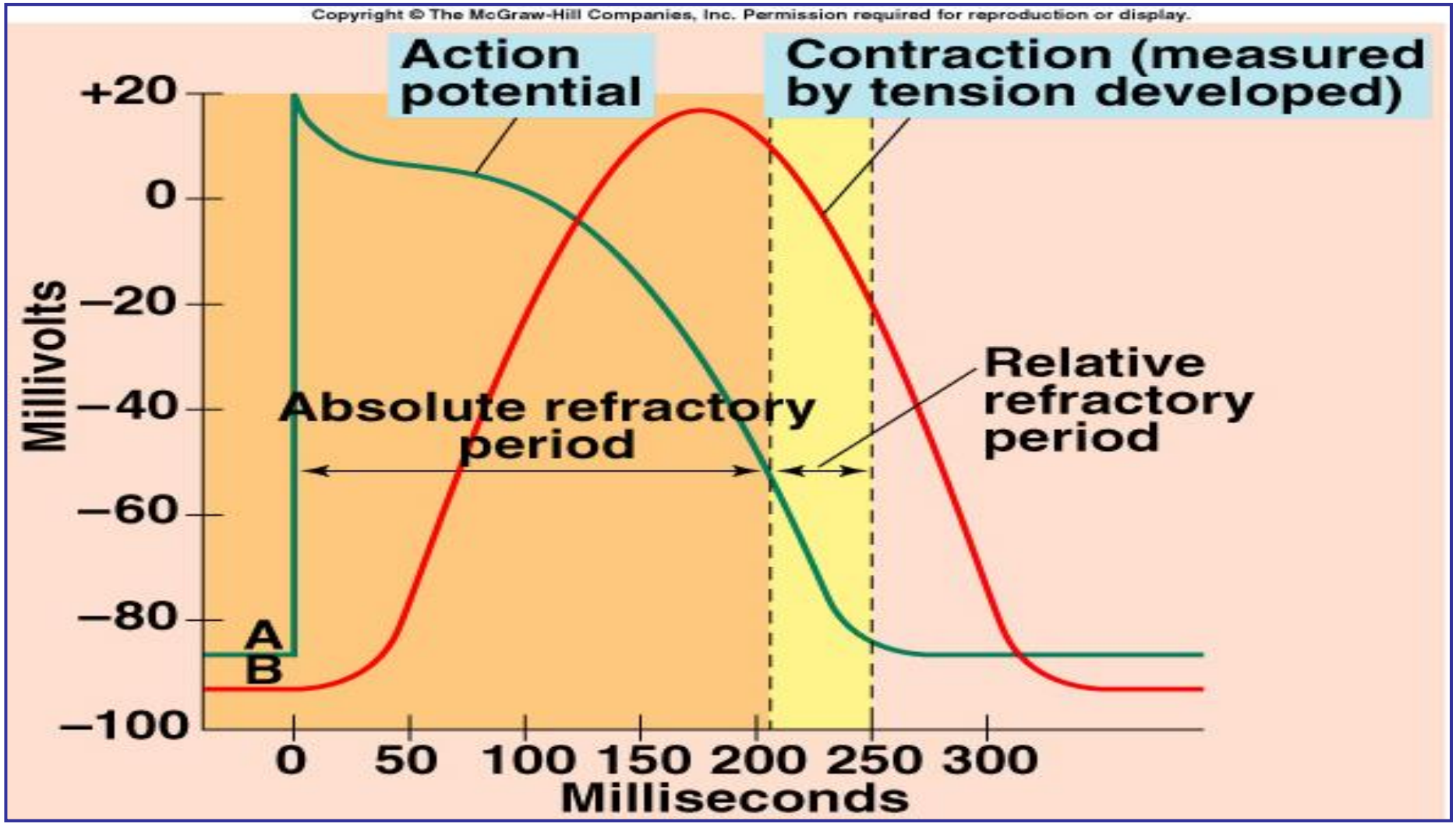
- **The slowest conduction velocity in AV-node:**
  - because it has few no. of intercalated discs.
  - Importance: to allow sufficient time for ventricles to be filled with blood before they contract.
- **The fastest Conduction velocity in Purkinje fibers:**
  - Importance: to allow the 2 ventricles to contract at the same time simultaneously.

Excitability changes during the action potential:

- **Passes through 3 periods:**
  1. Absolute refractory period (ARP / ERP)
  2. Relative refractory period (RRP)
  3. Dangerous period (supranormal period)

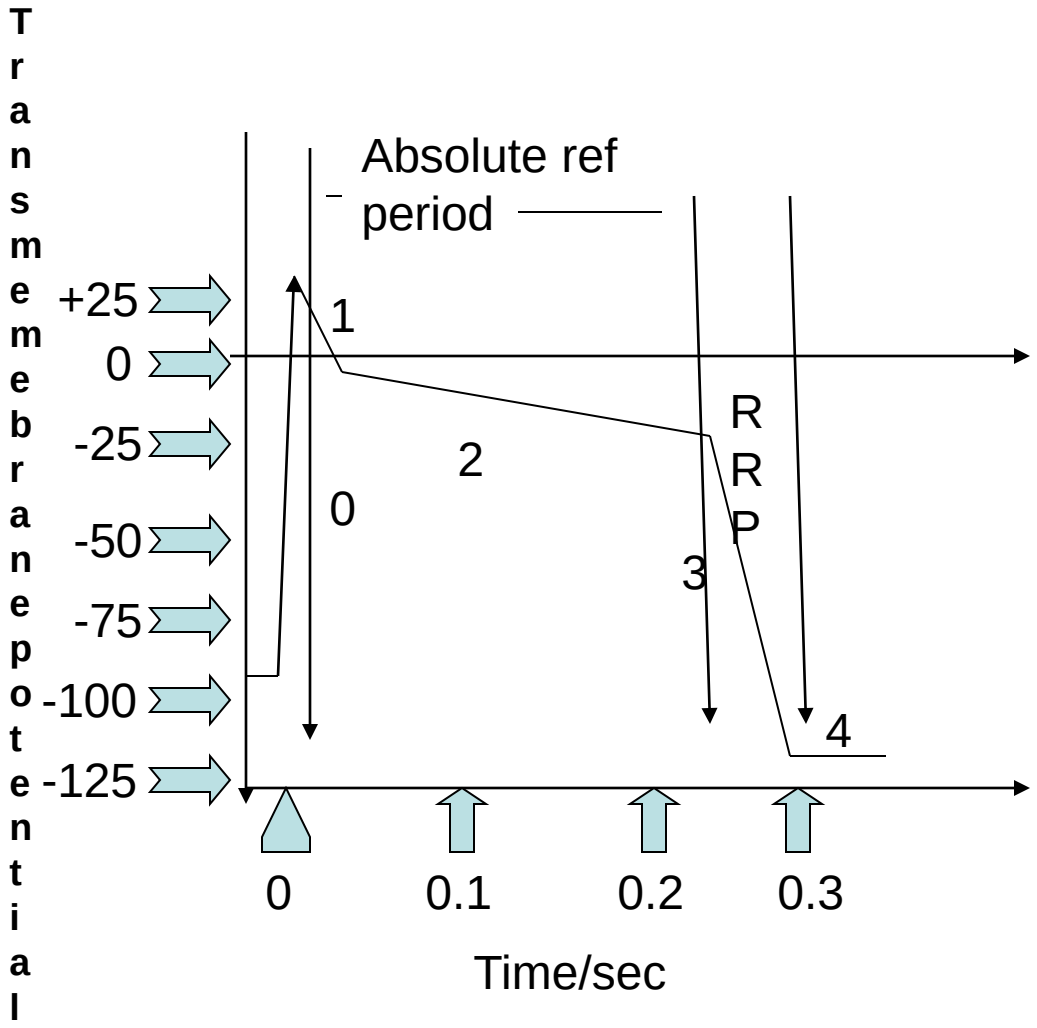


Refractory Periods



REFRACTORY PERIOD

- **Absolute Refractory Period** – regardless of the strength of a stimulus, the cell cannot be depolarized.
- **Relative Refractory Period** – stronger than normal stimulus can induce depolarization.



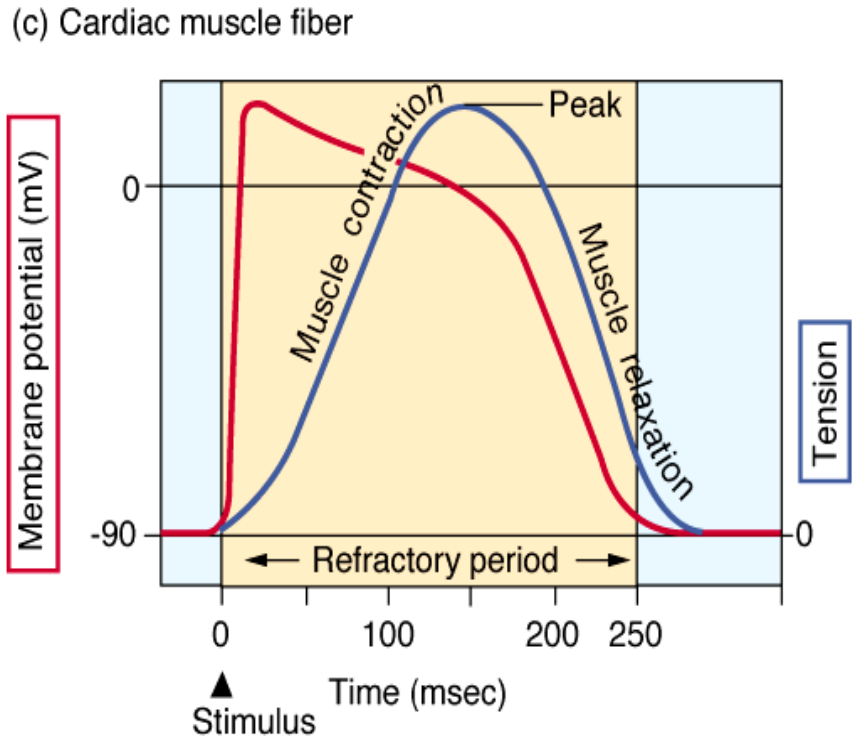
1. Absolute refractory period (ARP):

- The excitability of cardiac ms is completely lost during this period, i.e. doesn't respond to 2nd stimulus.
- V. long.
- Occupies the whole period of systole.
- Corresponds to the period of depolarization (phase 0), & the first 2 phases of repolarization.
- Heart can't be tetanized (continuous contraction), as its ARP occupies the whole contraction phase.

Twitch summation does not occur: Tetanus is not possible

IMPORTANCE OF REFRACTORY PERIOD

- Long refractory period prevents tetanic contractions
- systole and diastole occur alternately.
- It is very important for pumping blood to arteries.

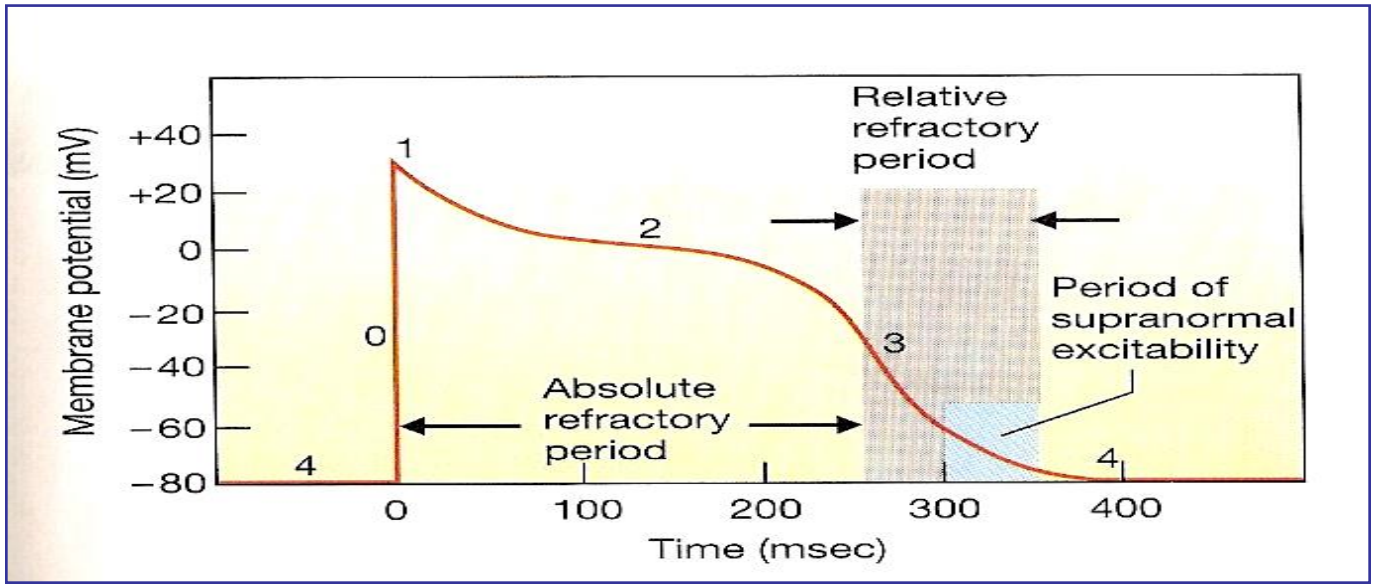


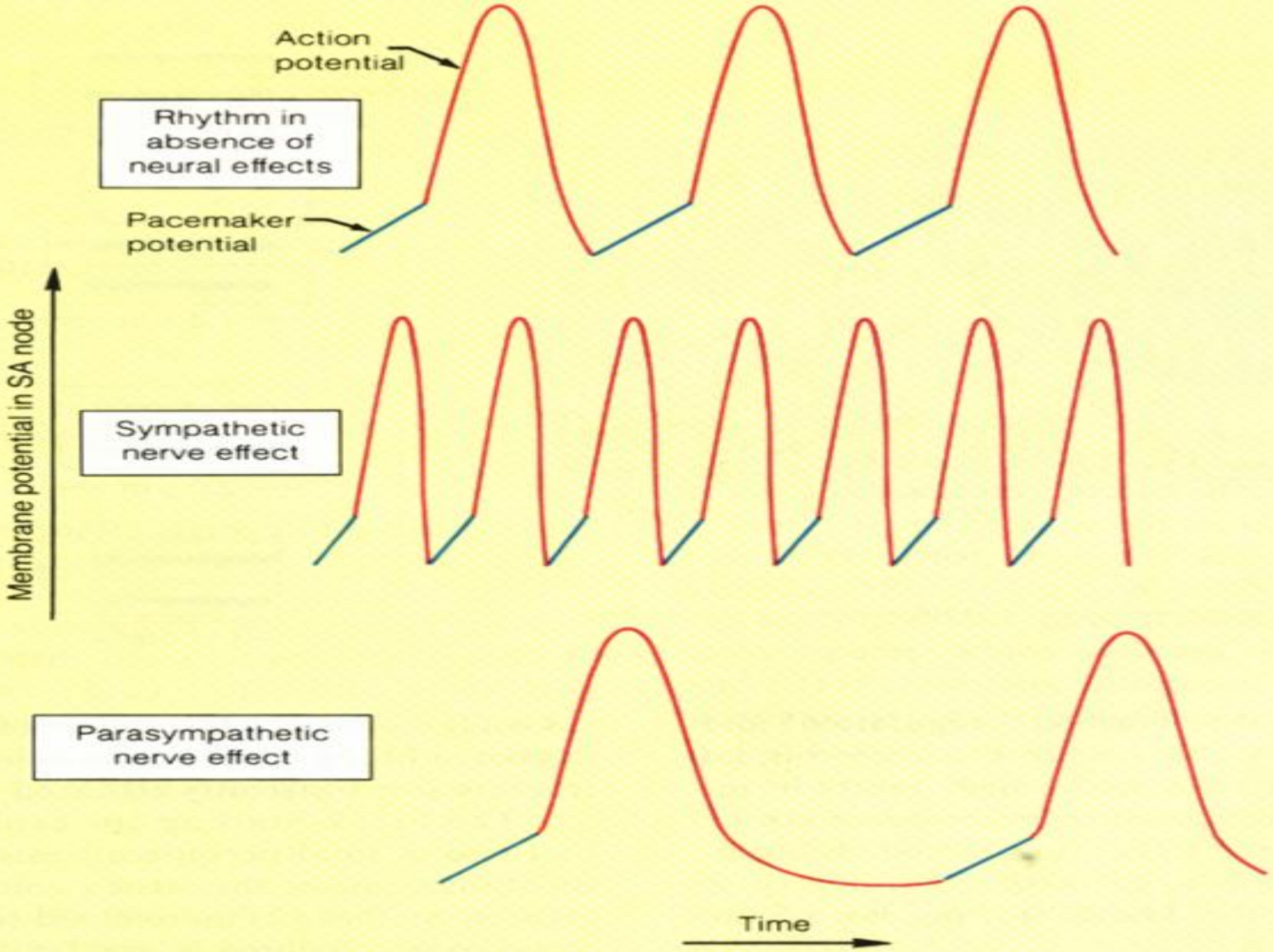
2. Relative Refractory Period (RRP):

- The excitability of cardiac muscle is partially recovered during this period, i.e. stronger stimuli than normal are required to excite the muscle.
- Occupies the time of diastole.
- Corresponds to the 3rd phase of repolarization.

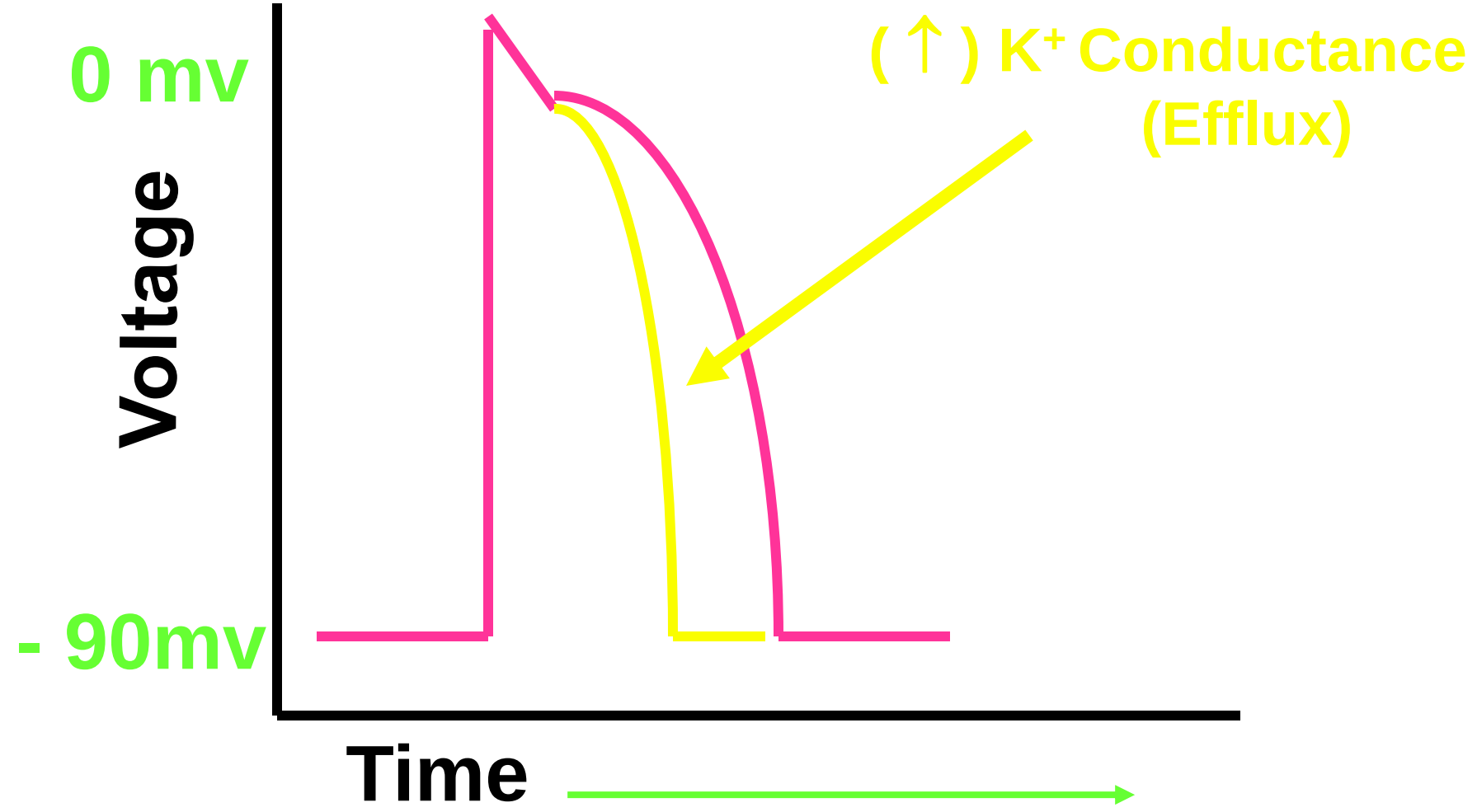
3. Dangerous Period (Supranormal):

- The excitability of cardiac muscle is supra normal just at the end of the AP, i.e. weaker stimuli than normal can excite the cardiac muscle.
- result in ventricular fibrillation.




Action potential
Rhythm in absence of neural effects
Pacemaker potential
Membrane potential in SA node
Sympathetic nerve effect
Parasympathetic nerve effect
Time

Ach ON ATRIAL ACTION POTENTIAL



Autonomic effects on heart rate & conduction velocity

Chronotropic Effects:

- Produces changes in heart rate.

- Negative Chronotropic Effect --- dec heart rate by dec firing rate of the SA node & vice versa.

Autonomic effects on heart rate & conduction velocity

Dromotropic Effects:

- Produces changes in conduction velocity primarily of AV node.

- Negative Dromotropic Effect --- dec Conduction Velocity through the AV node slowing the conduction of action potential from atria to the ventricles & vice versa.

Parasympathetic effects on heart rate & conduction velocity

- SA node, atria & AV node have parasympathetic vagal innervation
- Acetylcholine is the neurotransmiter
- Dec in heart rate
- Dec the rate of phase 4 depolarization
- Dec inward Na current

Parasympathetic effects on heart rate & conduction velocity

- Dec in conduction velocity via AV node
- Action potentials carry more slowly from the atria to the ventricles
- Dec inward ca+ current & inc outward K+ current

Sympathetic effects on heart rate & conduction velocity

- Nor epinephrine is the neurotransmiter
- Inc in heart rate
- Inc the rate of phase 4 depolarization
- Inc inward Na current
- More action potentials occur per unit time

Sympathetic effects on heart rate & conduction velocity

- Inc in conduction velocity via AV node
- Action potentials carry more rapidly from the atria to the ventricles
- Ventricular filling may be compromised
- Inc inward ca+ current

Ionotropic Effects:

- Produces changes in the hearts contractility.
- Related to intracellular Ca+ accumulation

- Positive ionotropic agents increases the contractility & vice versa

Positive Ionotropism

Factors:

- Increased heart rate (more action potentials / unit time --- more Ca+ --- more contraction)

- Sympathetic stimulation by (catecholamines) Inc inward Ca+ current

Negative Ionotropism

Factors:

- Parasympathetic stimulation by (Acytylcholine) Dec inward Ca+ current --- Dec force of contraction

Factors affecting myocardial excitability (continued)

1. **Cardiac Innervation:**
   - Sympathetic NS → ↑ excitability.
   - Parasympathetic NS (vagus) → ↓ excitability.
2. **Effect of ions concentration in ECF:**
   - ↑ Ca^{2+} → ↑ excitability.
   - ↑ K^{+} → ↓ excitability.
3. **Physical factors:**
   - ↑ temperature → ↑ excitability.
   - ↓ temperature → ↓ excitability.

Factors affecting myocardial excitability (continued)

4. **Blood flow:**
   - Insufficient blood flow to cardiac ms ↓ excitability &
     myocardial metabolism for 3 reasons:
     (1) lack of O_2,
     (2) excess accumulation of CO_2, &
     (3) lack of sufficient food nutrients.
5. **Chemical factors (drugs):**
   - Digitalis → ↑ excitability.

Preload:

The load to which a muscle is subjected before shortening / contraction.

Afterload:

The load to which a muscle is subjected after shortening / contraction.

Thank You