Cardiovascular

(CVS)

By

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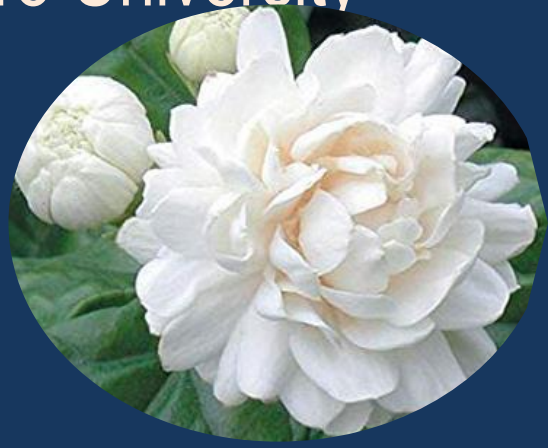

Cardiac properties:

1. Rhythmicity
2. Conductivity
3. Excitability
4. Contractility

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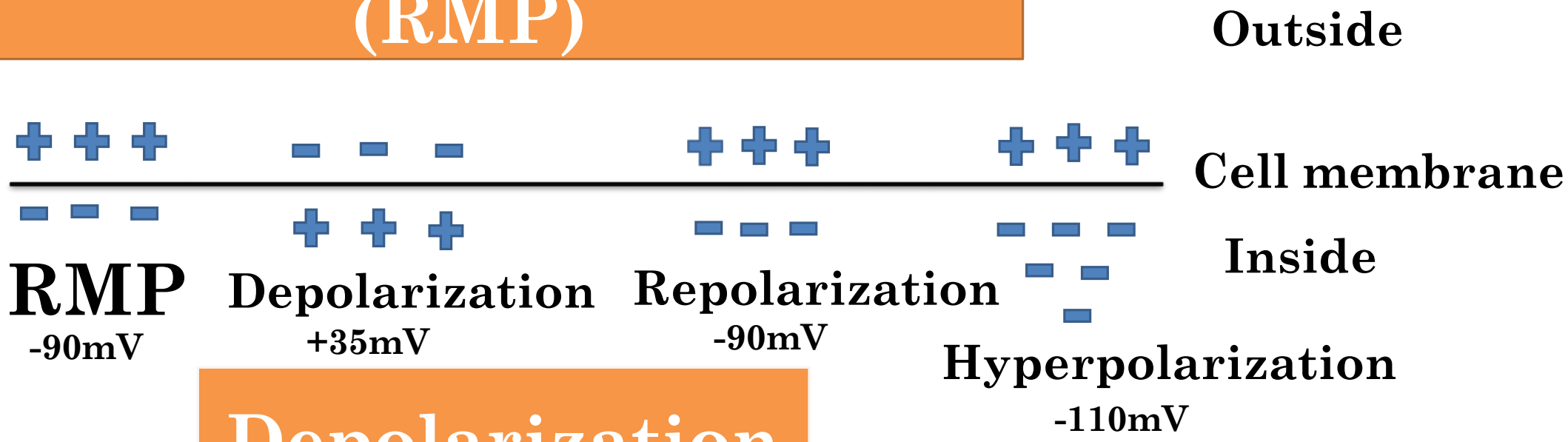
Resting Membrane Potential (RMP)



Depolarization

Repolarization

Hyperpolarization

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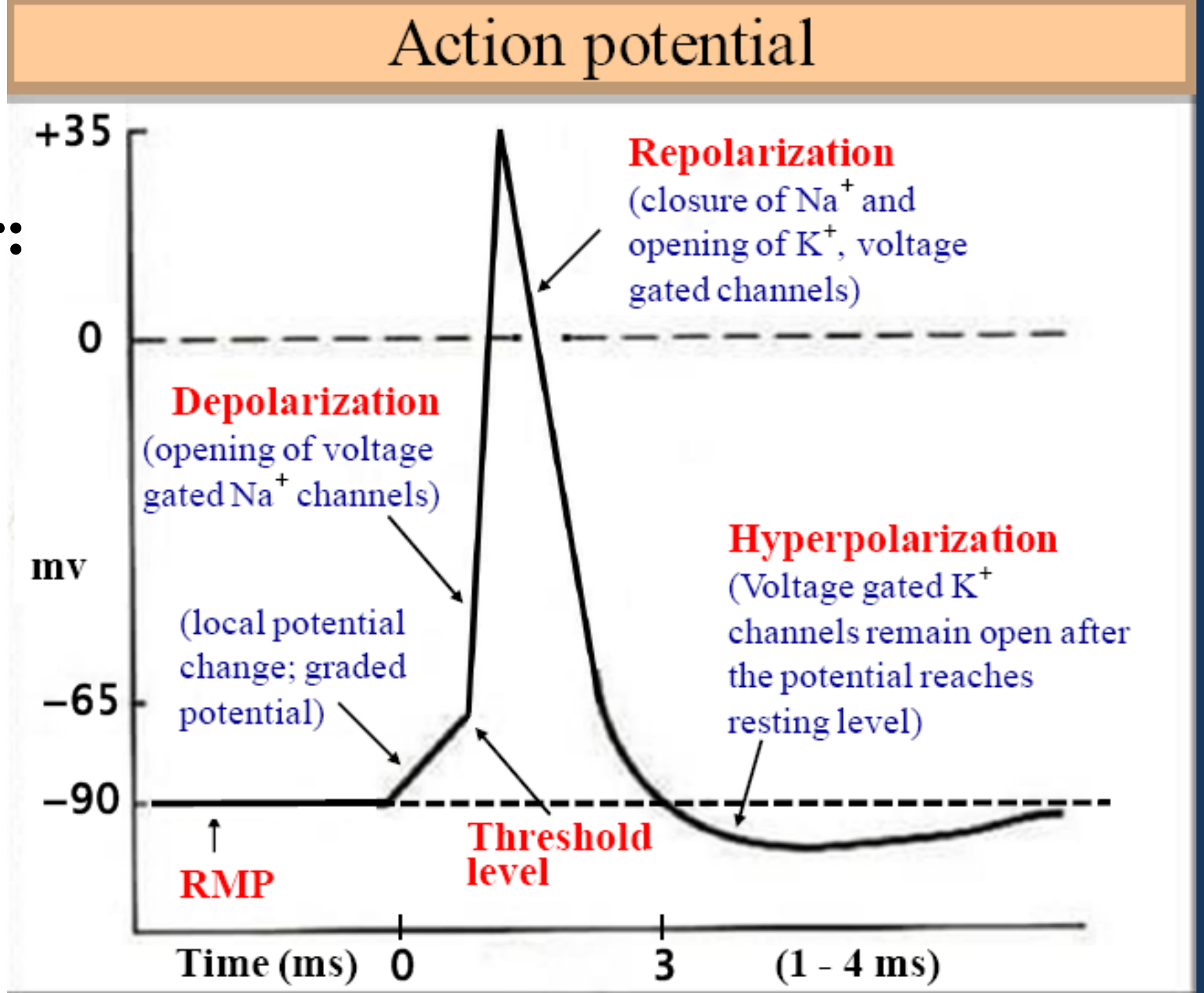
- **To remember: Regular Nerve and Muscle action potential (Fast AP)**



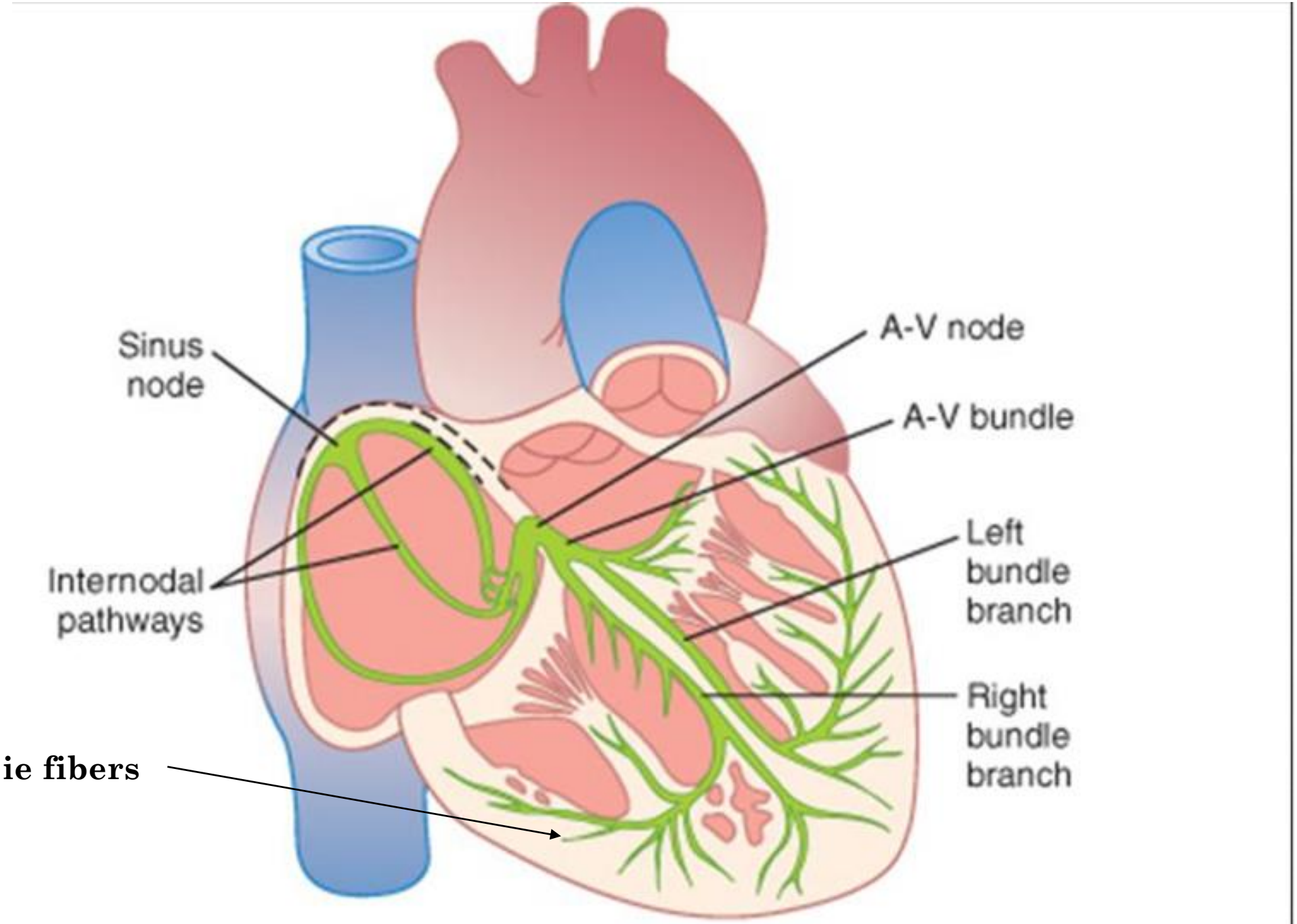
1. Rhythmicity (Automaticity)

It is the ability of the cardiac muscle to beat regularly and to initiate its own regular impulses.

1. Rhythmicity

- It is specific to cardiac muscle (Myogenic in nature)
- Specific fibers concerned with rhythmicity are:

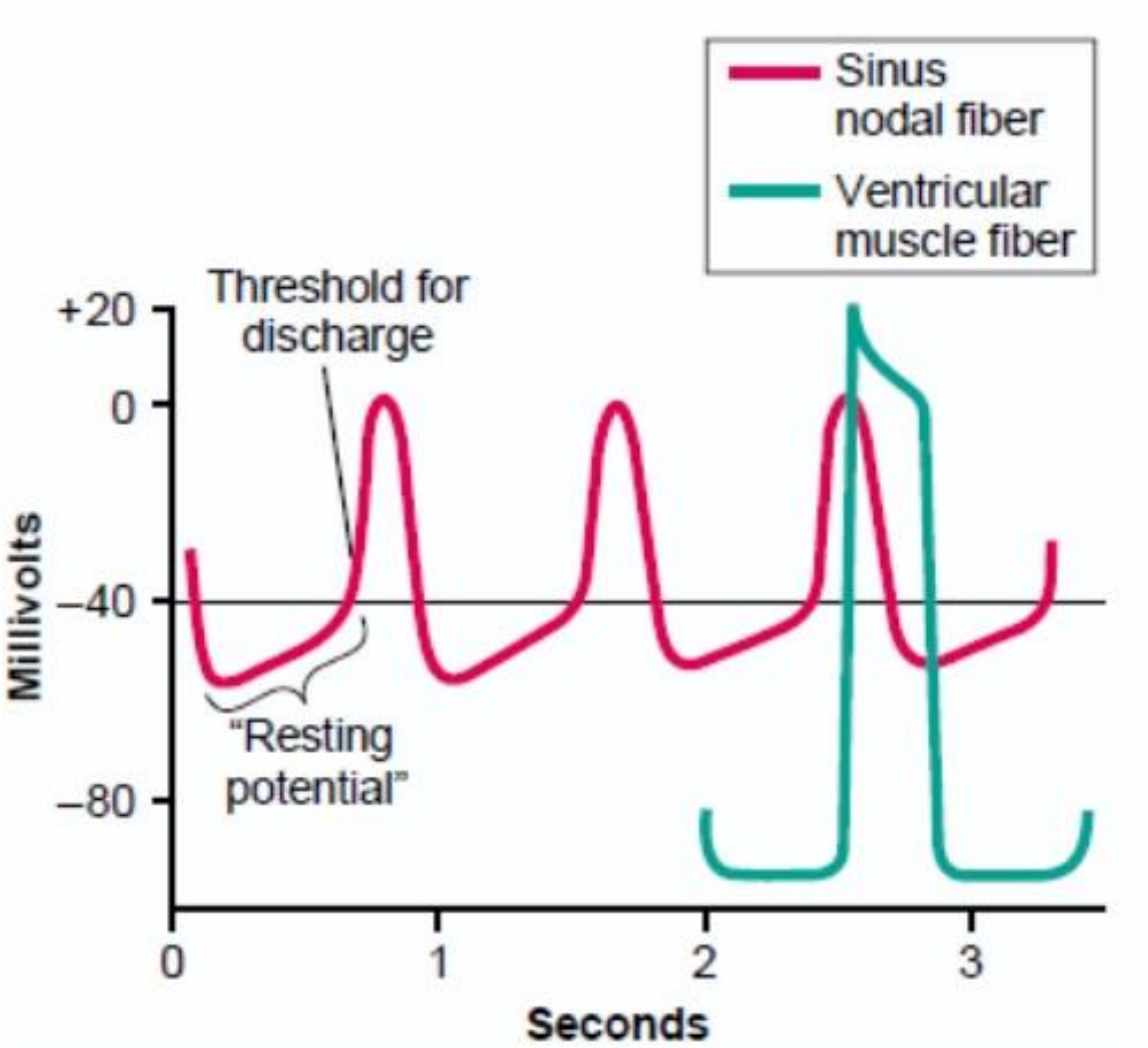
1. **SAN**
2. **AVN**
3. **PF**



Guyton & Hall: Textbook of Medical Physiology 11e - www.studentconsult.com

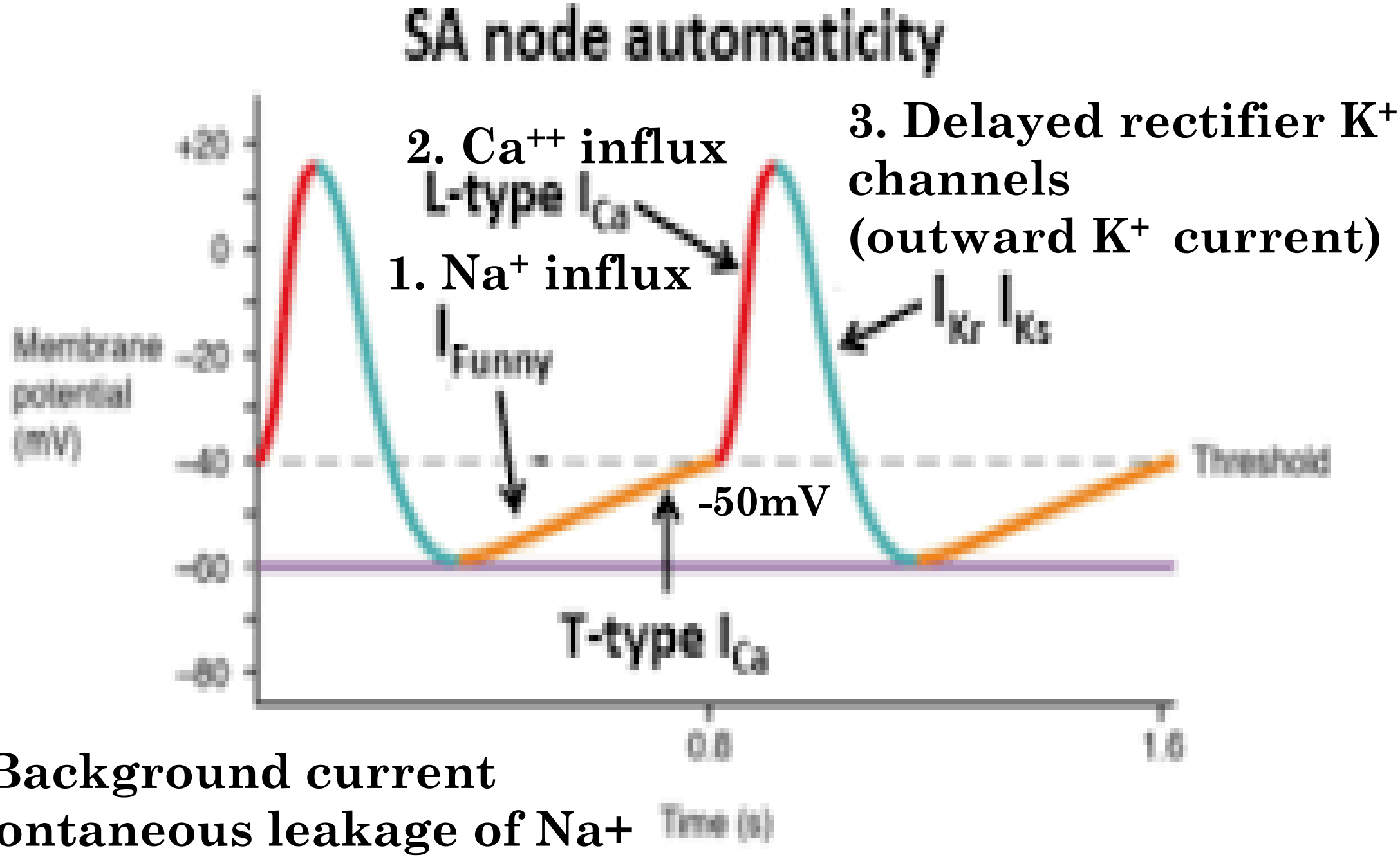
Automatic specific fibers differ from cardiac muscle fibers in:

1. They have unstable RMP
2. Can discharge impulses spontaneously i.e. independent from extrinsic or intrinsic nerve supply
3. Rhythmic
4. Have prepotential (Pacemaker potential)
5. Differ from ventricular action potential as they have no plateau.



Pacemaker potential (Prepotential)

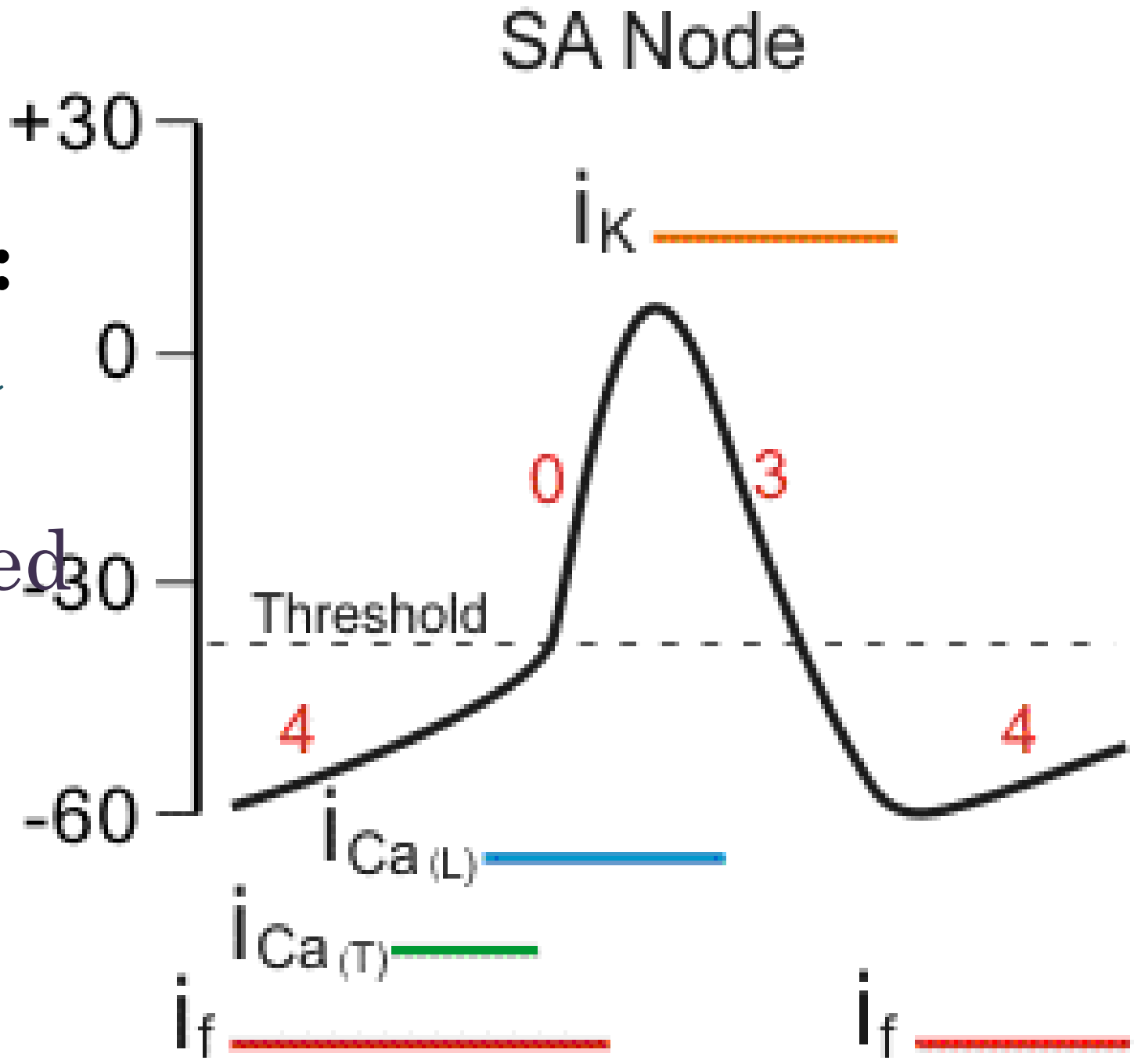
- It is slow partial depolarization
- Diastolic depolarization i.e. occurs during diastole
- Spread allover the cardiac muscle leading to excitability and contractility (Systole) of the cardiac muscle.



4. Background current
Spontaneous leakage of Na+

- **Slow partial depolarization:**

1. Absent fast Na channels
2. Slow inactivated inward Ca^{++} current
3. Slow repolarization



Pacemaker Cells of the heart

- **SAN** (rate of intrinsic discharge of impulses 90 -105/min)
- **AVN** (rate of intrinsic discharge of impulses 40 - 60/min)
- **Purkinje fibers** (rate of intrinsic discharge of impulses 15 -40/min)

- SAN is the normal pace maker of the heart so, it controls the heart rate **(Why?)**

  - **Because:**

it has the fastest rate than the natural self-excitatory discharge rate of either AVN or PF.

  - **AVN** and **PF** can play a role in case of failure of SAN to send them impulses for any reason. It is called **nodal rhythm** and **idioventricular rhythm** respectively

- **Why?**

SAN rate of discharge is 90 discharge per minute while the heart rate is 70 beat per minute
(bpm or beat/min)

Due to

Vagal tone

Vagal Tone:

- Under normal resting conditions, parasympathetic effect has the upper hand over the sympathetic supply of the SAN, leading to decrease rate of discharge inside the body.
- SAN rate of discharge is:

1. At rest inside the body: 70/min
2. Vagi are cut or blocked: 120/min (sympathetic effect)
3. Vagi and Sympathetic nerves are cut or blocked: 90/min

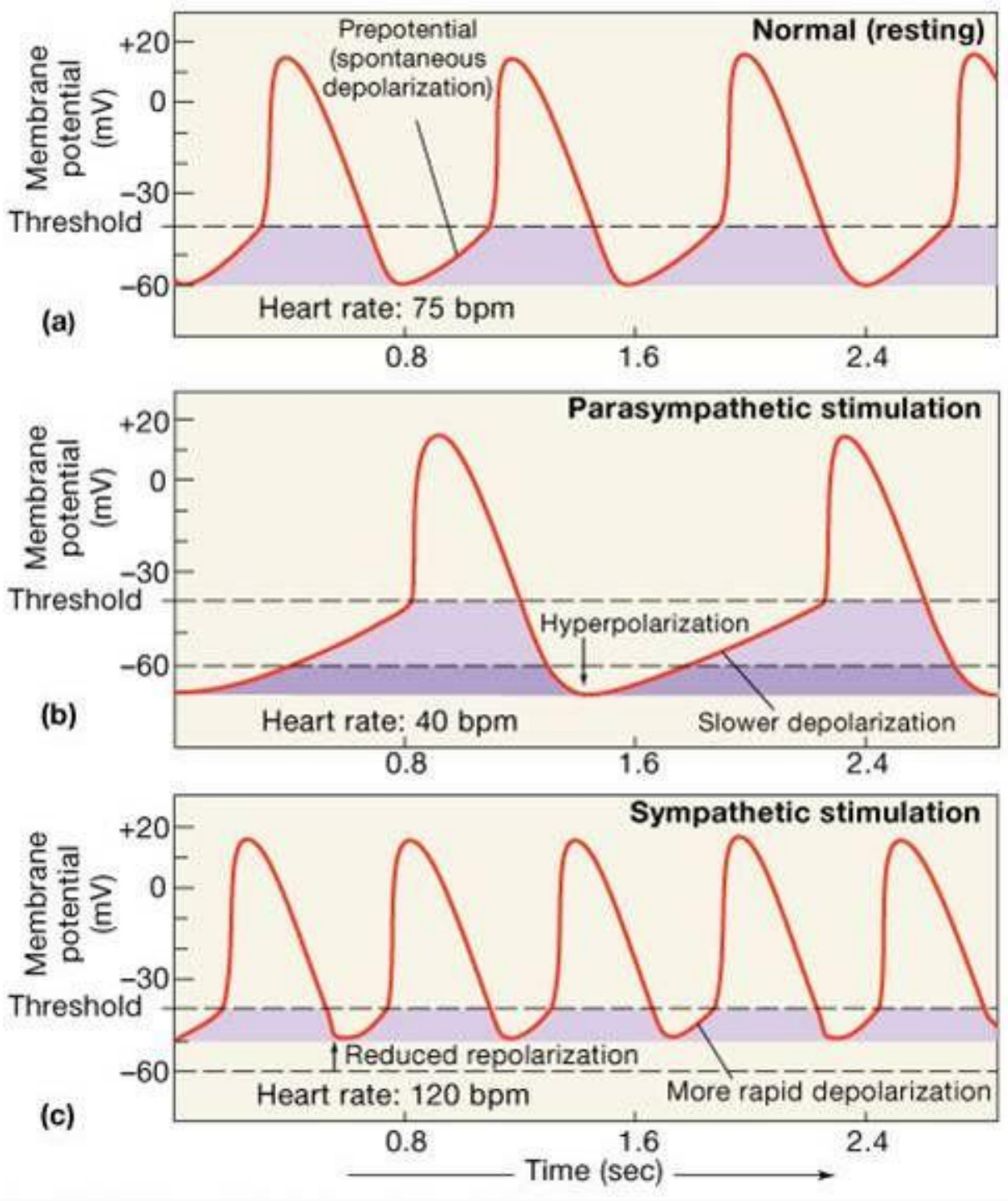
Vagal Tone:

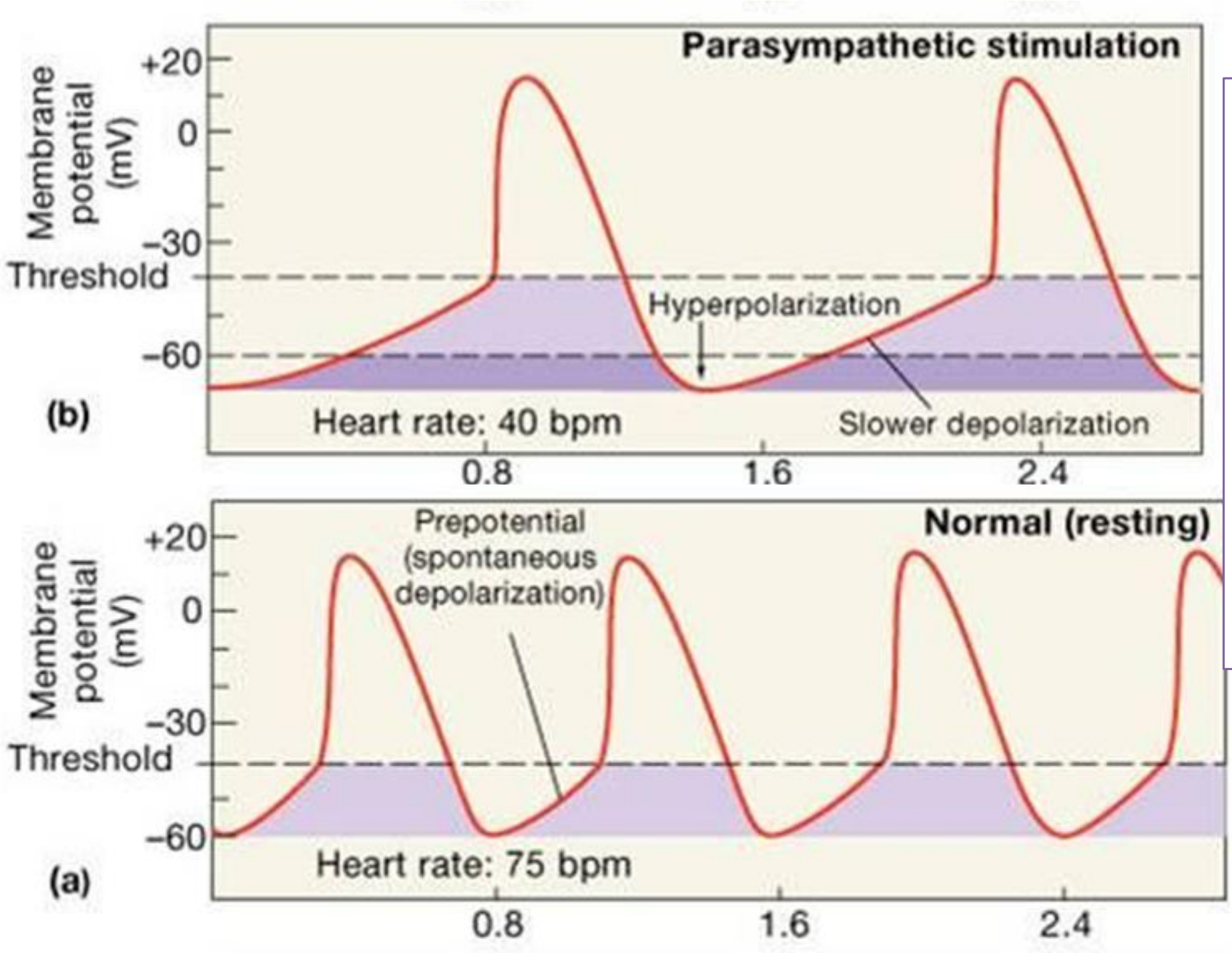
- **<u>Parasympathetic reflex (SRACEER):</u>**
  - **Stimulus:** normal rate of discharge of SAN
  - **receptors:** muscarinic receptors
  - **afferent:** Vagus nerve
  - **efferent:** Vagus nerve
  - **Effector organ:** SAN
  - **response:** normal HR

Normal Heart rate (HR)

Bradycardia = decreased HR (-ve chronotropic effect)

Tachycardia = increased HR (+ve chronotropic effect)





Slope decreases

HR decreases

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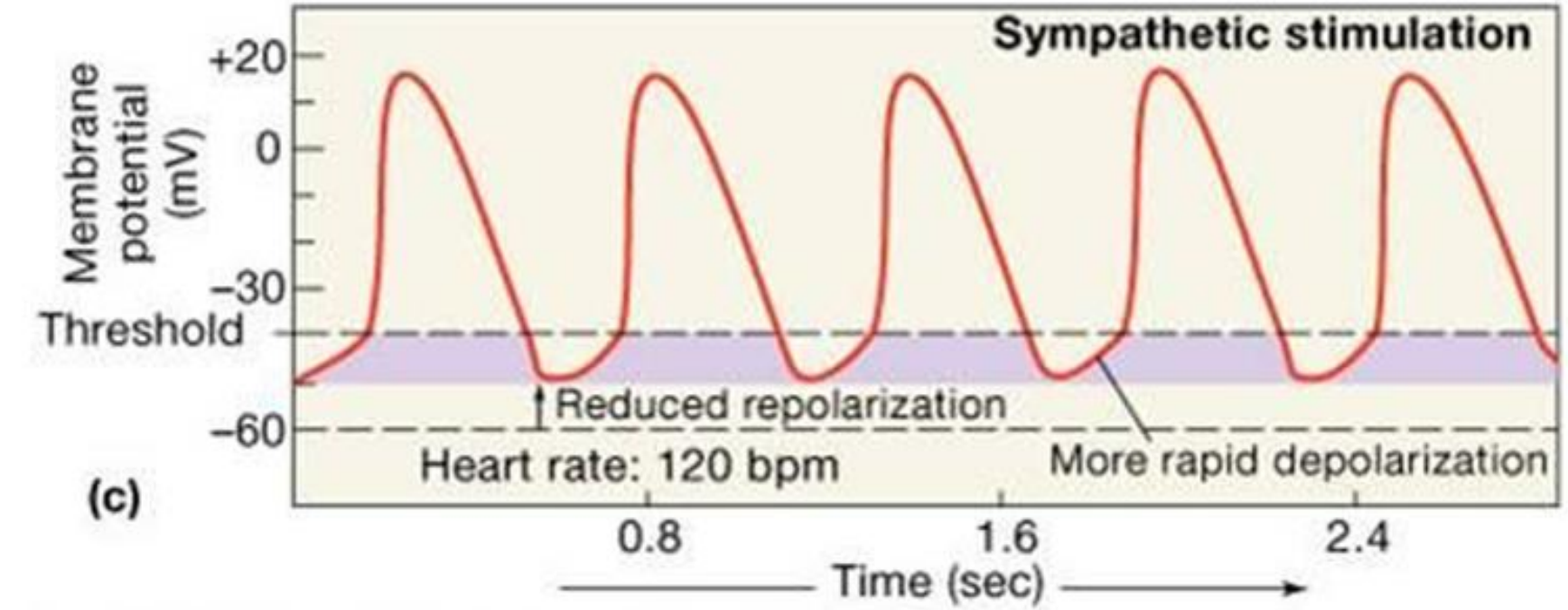

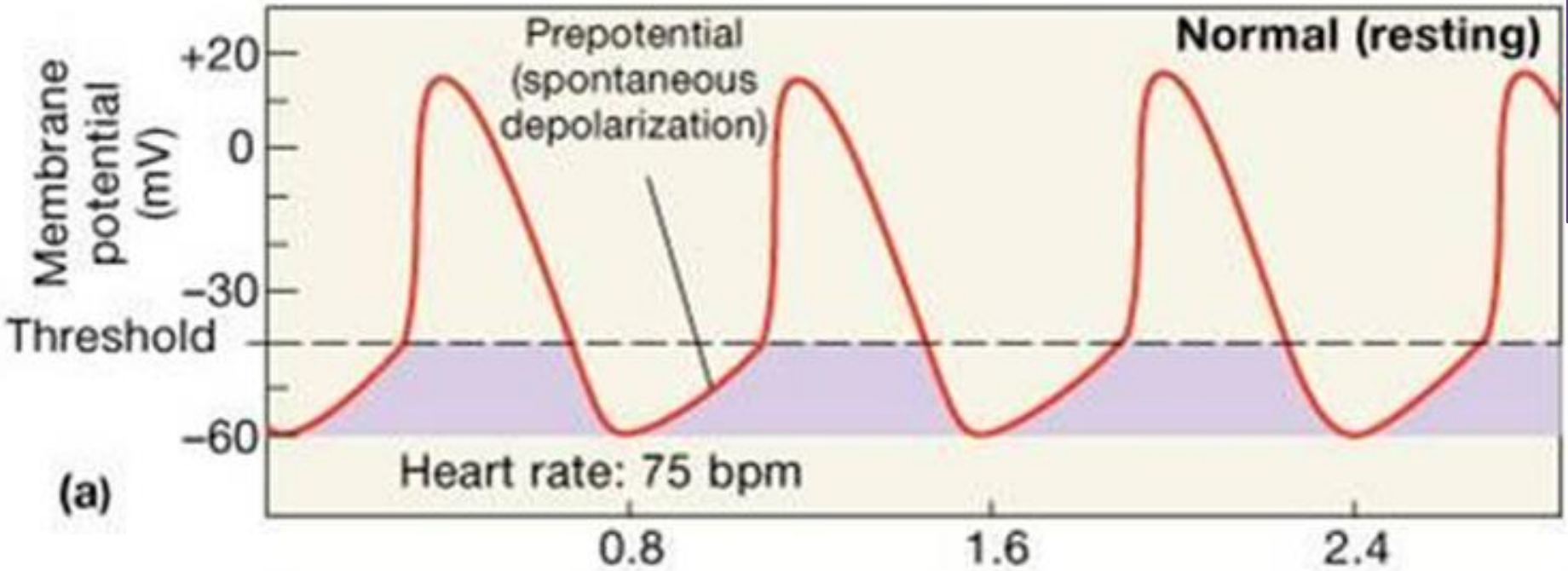

Slope increases

HR increases

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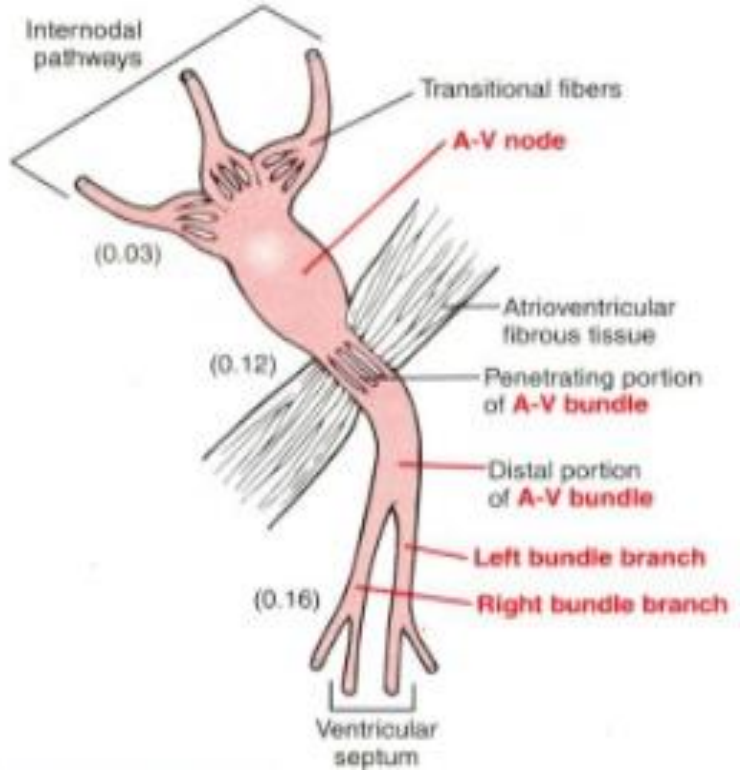
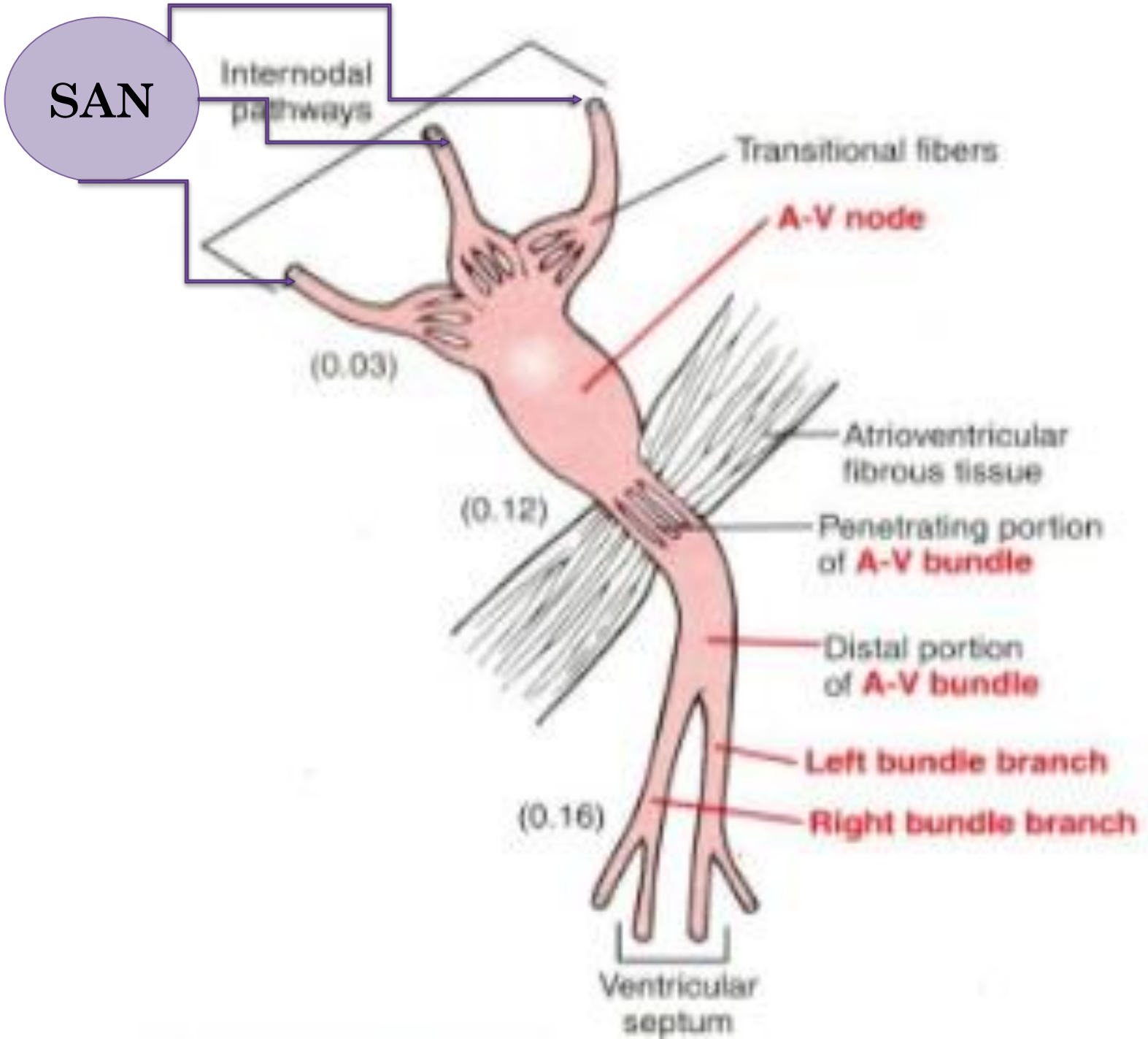


2. Conductivity

It is the ability of the cardiac muscle to transmit and conduct the excitation wave (impulses = action potentials)



Rate of conduction of cardiac impulse in different parts of conductive system:

- **SAN:** 0.5 m/sec
- **Atrial muscle:** 0.3 m/sec
- **Internodal pathways:** 1 m/sec
- **Atrioventricular node:** 0.2 -0.3 m/sec (0.05 m/sec) slowest conduction
- **A-V bundle:** 2m/sec
- **PF: 4 – 5 m/sec** fastest conduction
- **Ventricular muscle:** 0.3 – 0.5 m/sec

The conduction velocities in different parts of cardiac tissue:

Site of conduction	Velocity of conduction
SAN through atrial myocytes	0.5 m/sec
Internodal pathways	1 m/sec
AVN	**0.05 m/sec**
Bundle of His and its branches	2 m/sec
Purkinje fibres	4 m/sec
Ventricular myocytes	0.5 m/sec

Significance of slow AVN conductivity (AV nodal delay):

- It is the only route for the transmission of impulses from the atria to the ventricles as they are separated by AV fibrous tissue.
- **Cause:**

It has the least number of gap junctions so, increasing the resistance to the electrical current flow and delays impulses from reaching the ventricular muscle.

Significance of slow AVN conductivity (AV nodal delay):

- **Physiologically:**

this delay gives enough time for the atria to contract and empty their blood to the ventricle (atrial systole), hence enough time to the ventricular diastole i.e. ventricular filling which plays an important role in efficient cardiac contractility.

- AV nodal delay is 0.1 – 0.2 sec.

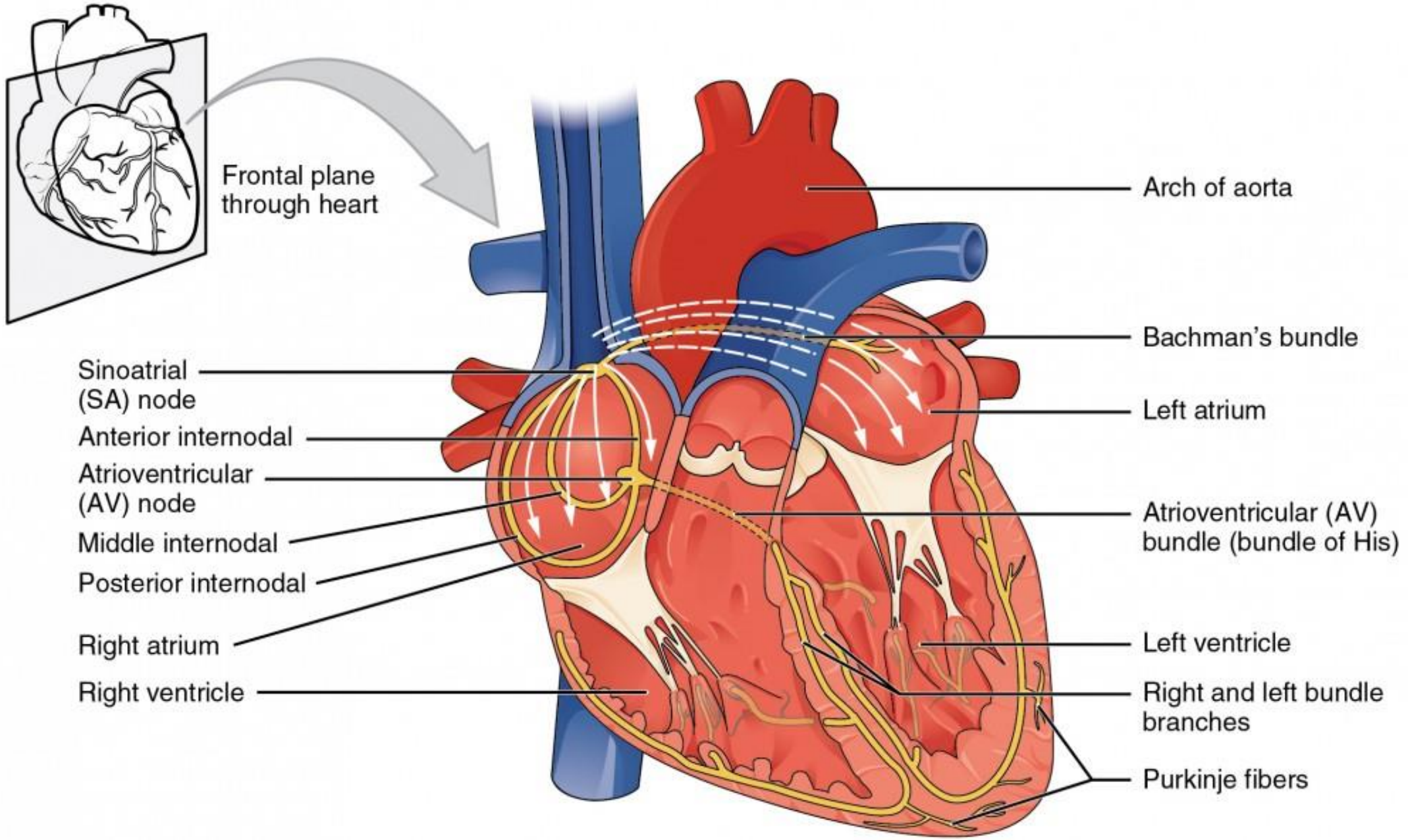
Significance of slow AVN conductivity (AV nodal delay):

- **Pathologically:**

In case of high rhythms as in supraventricular tachycardia causes (except AVN diseases), It can reduce the rate of impulses that can pass from the atria to the ventricles trying to protect ventricular filling.

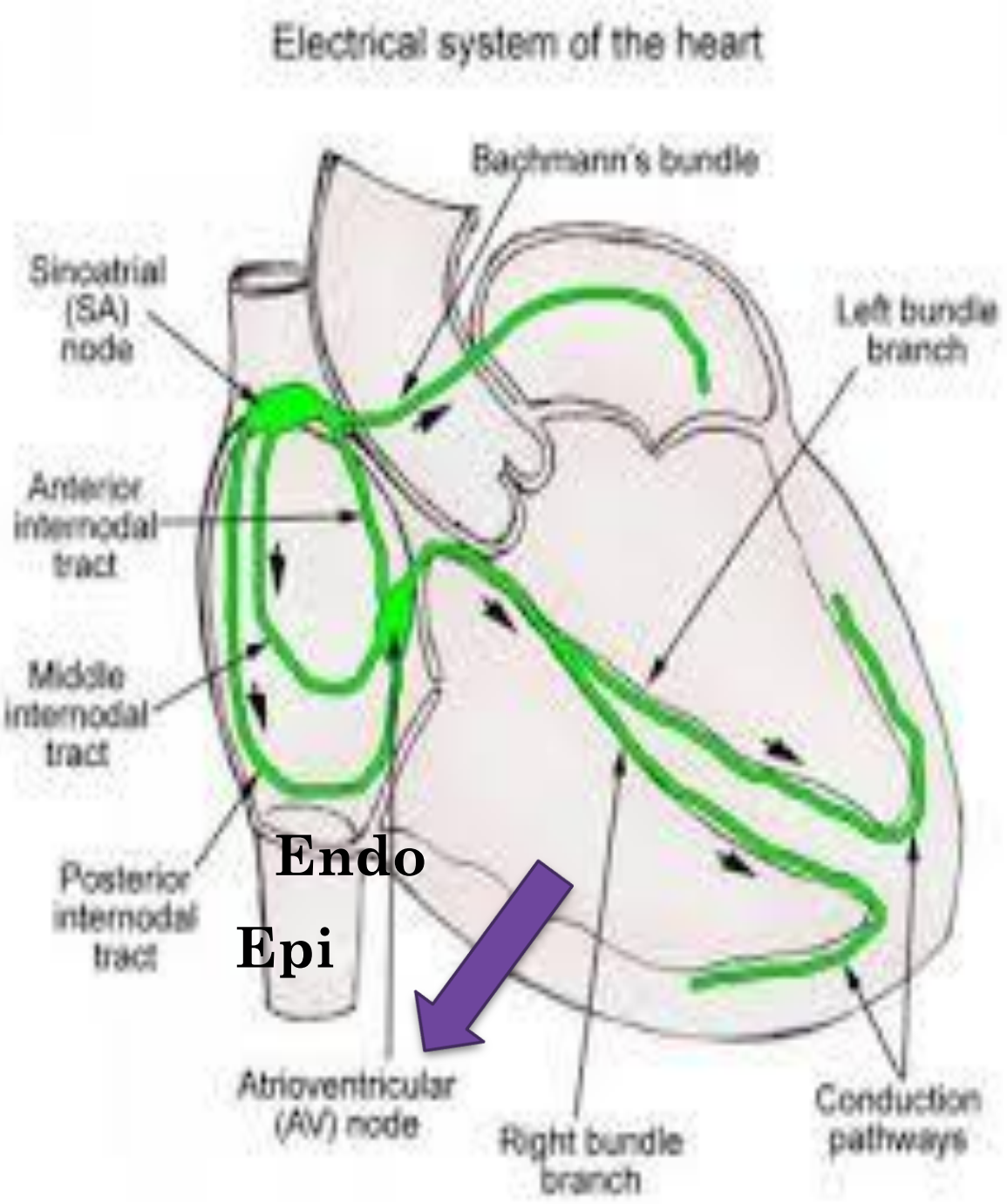
Significance of fast conductivity of Purkinje fibers (PF) and AV bundle branches :

- **Cause:** very high level of gap junctions and the largest cardiac muscle fibers.
- **Importance:** very rapid transmission of the cardiac impulses to all ventricular muscle fibers at the same time so, they contract maximally as one syncytium pumping blood to the whole body against peripheral resistance.



Anterior view of frontal section

- The conduction of cardiac impulses through ventricular muscle is from endocardium to epicardium
- So, the last portion of the ventricular syncytium that receives cardiac excitation wave is the epicardial surface of the base of the left ventricle



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