PHYSIOLOGY

بسم الله الرحمن الرحيم

MID | Lecture 3

Conduction System of The Heart

وَلَقَدْ خَلَقْنَا الْإِنسَانَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِ نَفْسُهُ ۖ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ الْوَرِيدِ

اللهم إنّا نعوذ بك من شرور أنفسنا ومن سيئات أعمالنا

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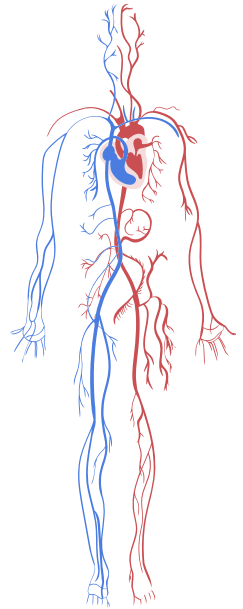
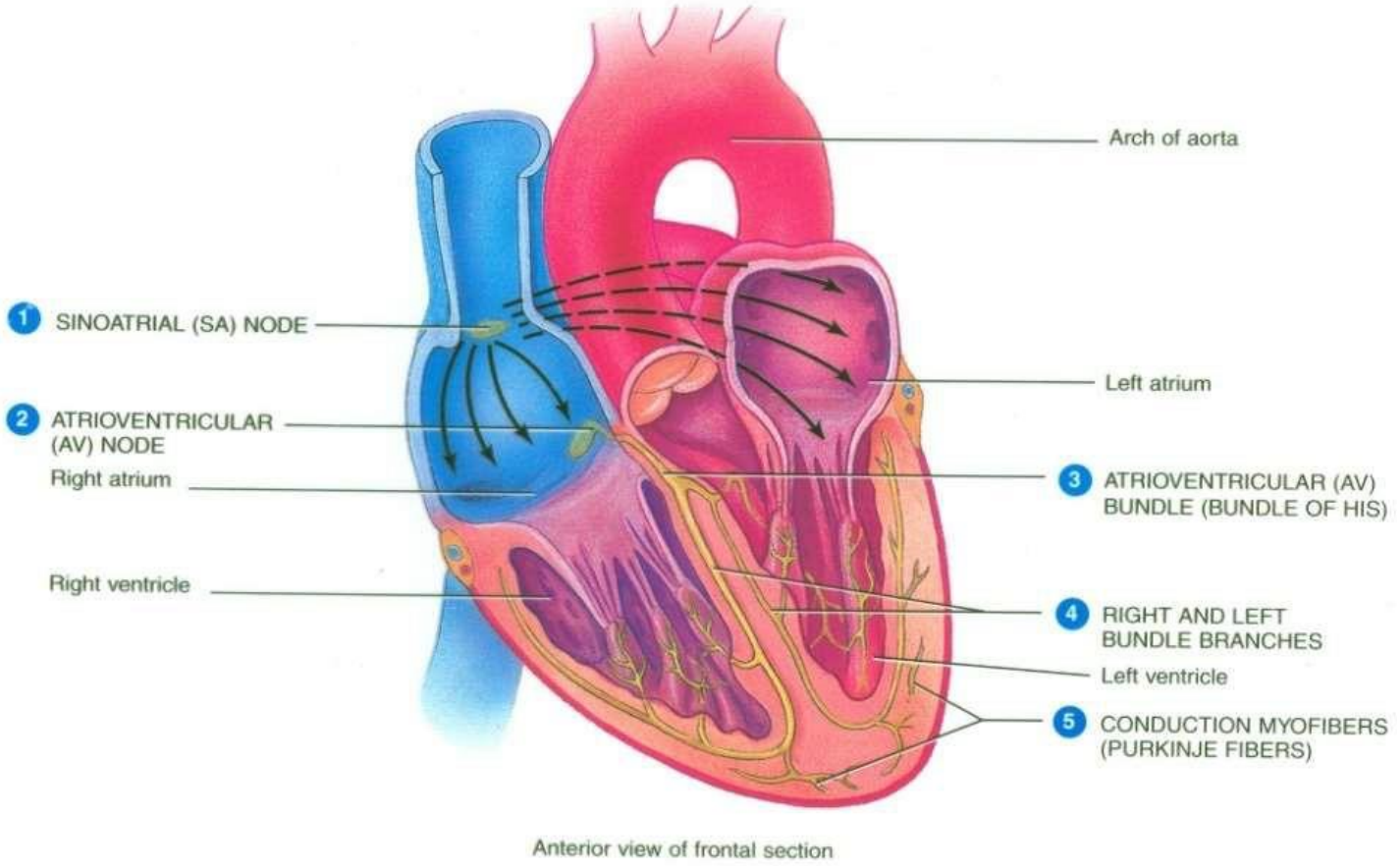


Objectives

- List the parts that comprise the conduction system
- Explain the mechanism of slow response action potential (pacemaker potential)
- Point out the regulation of the conduction system potential by Autonomic Nerves
- Resource: *Guyton's Textbook of Medical Physiology last edition*.

Structures of the Conduction System



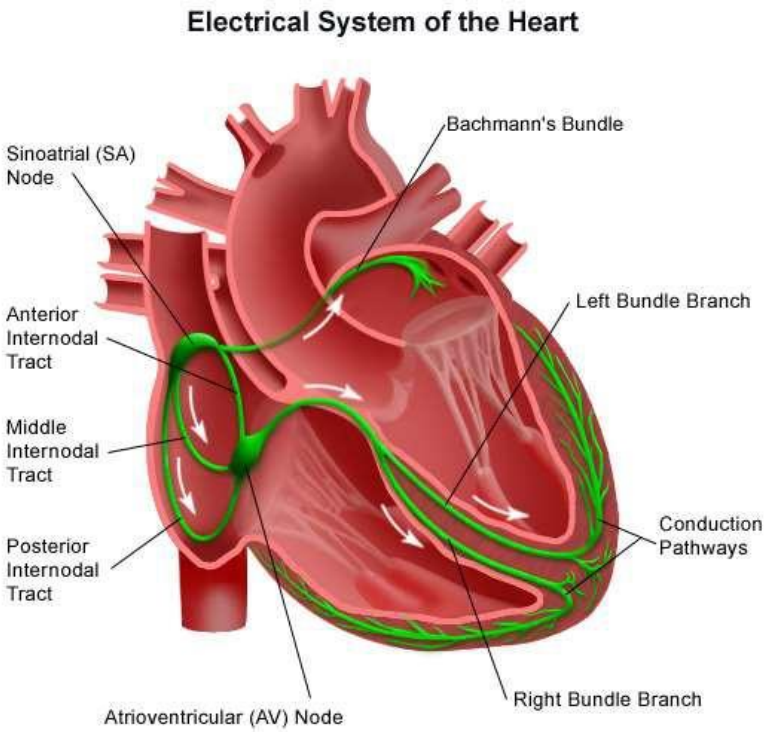
Anterior view of frontal section

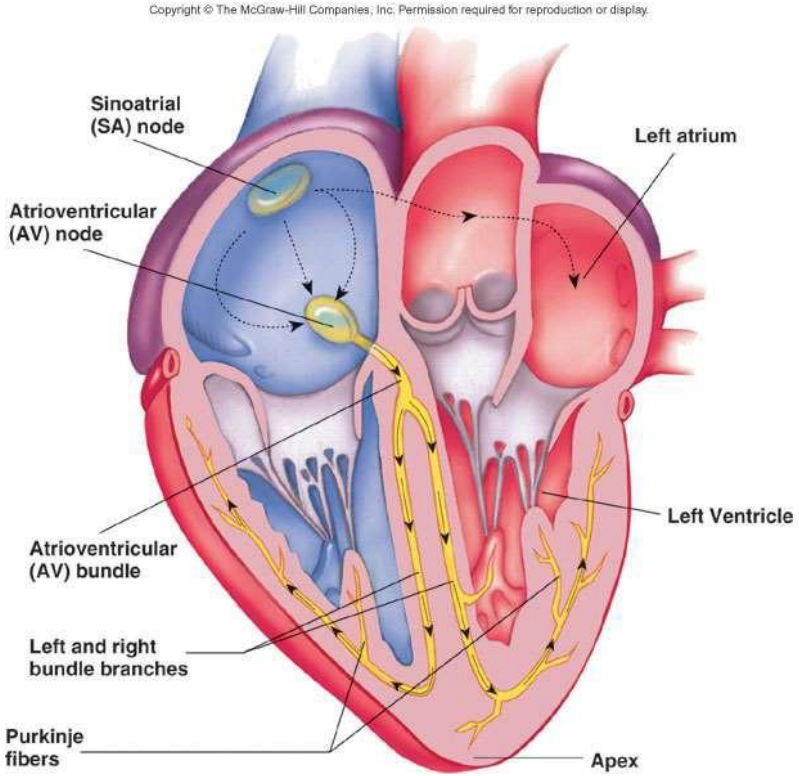
Structures of the Conduction System

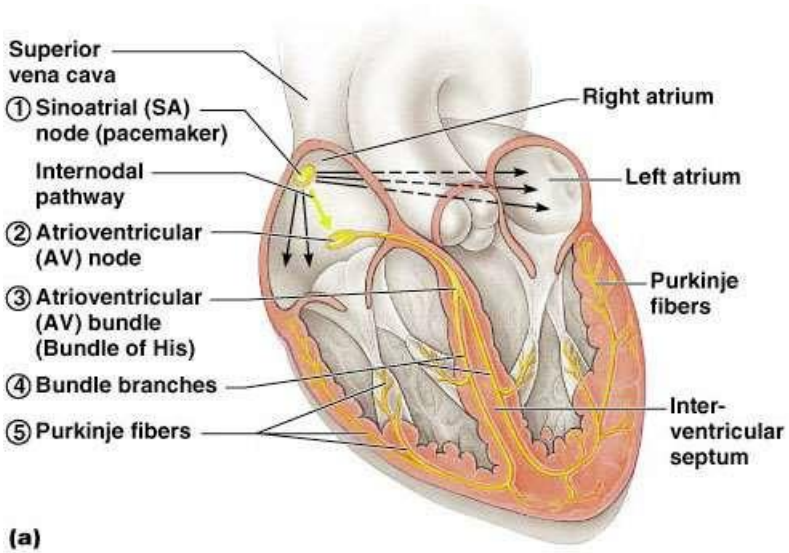
- Cardiac **muscle** is **involuntary**, meaning it is not controlled by **somatic (voluntary)** nerves.
- It is supplied by **autonomic fibers**, which include both the **sympathetic and parasympathetic divisions**.
- However, even if both autonomic inputs are severed, the **heart continues to beat spontaneously**.
- Therefore, the function of these autonomic nerves is **to regulate, not initiate,** the heartbeat.

Structures of the Conduction System

- During a heart transplant, the organ is removed from a **donor** and preserved in a **calcium-rich solution**.
- Extracellular **Ca^{2+}** is essential because the calcium that enters through **voltage-gated slow Ca^{2+} channels** triggers additional calcium release from the **sarcoplasmic reticulum**, sustaining contraction.
- Even when isolated and placed in a **Ca^{2+}-containing solution**, the heart **continues to contract on its own**.
- As you know, **mechanical contraction follows electrical excitation**—there is no contraction without an electrical impulse.
- This electrical impulse arises from a **specialized network within the heart called the cardiac conduction system**, which provides an **intrinsic rhythm** for cardiac activity.







Conducting System of Heart & Sequence of Excitation

Heart Physiology: Sequence of Excitation

- The first component of the conduction system is the **sinoatrial (SA) node**, located just below the **superior vena cava (SVC)** opening on the **posterior wall of the right atrium**.
- All parts of the conduction system consist of **specialized cardiac muscle fibers**, adapted either **structurally or functionally** for impulse generation and conduction.
- Following the SA node, the impulse reaches the **atrioventricular (AV) node**, situated in the **right atrium near the atrioventricular junction**.
- From the AV node, the impulse travels through the **atrioventricular bundle (Bundle of His)**, which **pierces the fibrous skeleton** separating the atria and ventricles — a region that **cannot conduct impulses directly because it is connective tissue, not muscle**.

Heart Physiology: Sequence of Excitation

- Thus, the **only normal pathway** for conduction between the atria and ventricles is via the **AV node and the AV bundle**.
- The Bundle of His then divides into **right and left bundle branches**, which course **subendocardially along the interventricular septum** and extend through approximately **two-thirds of the ventricular myocardium**.
- Finally, these branches terminate as **Purkinje fibers (conduction myofibers)** that distribute the impulse throughout the ventricular walls.

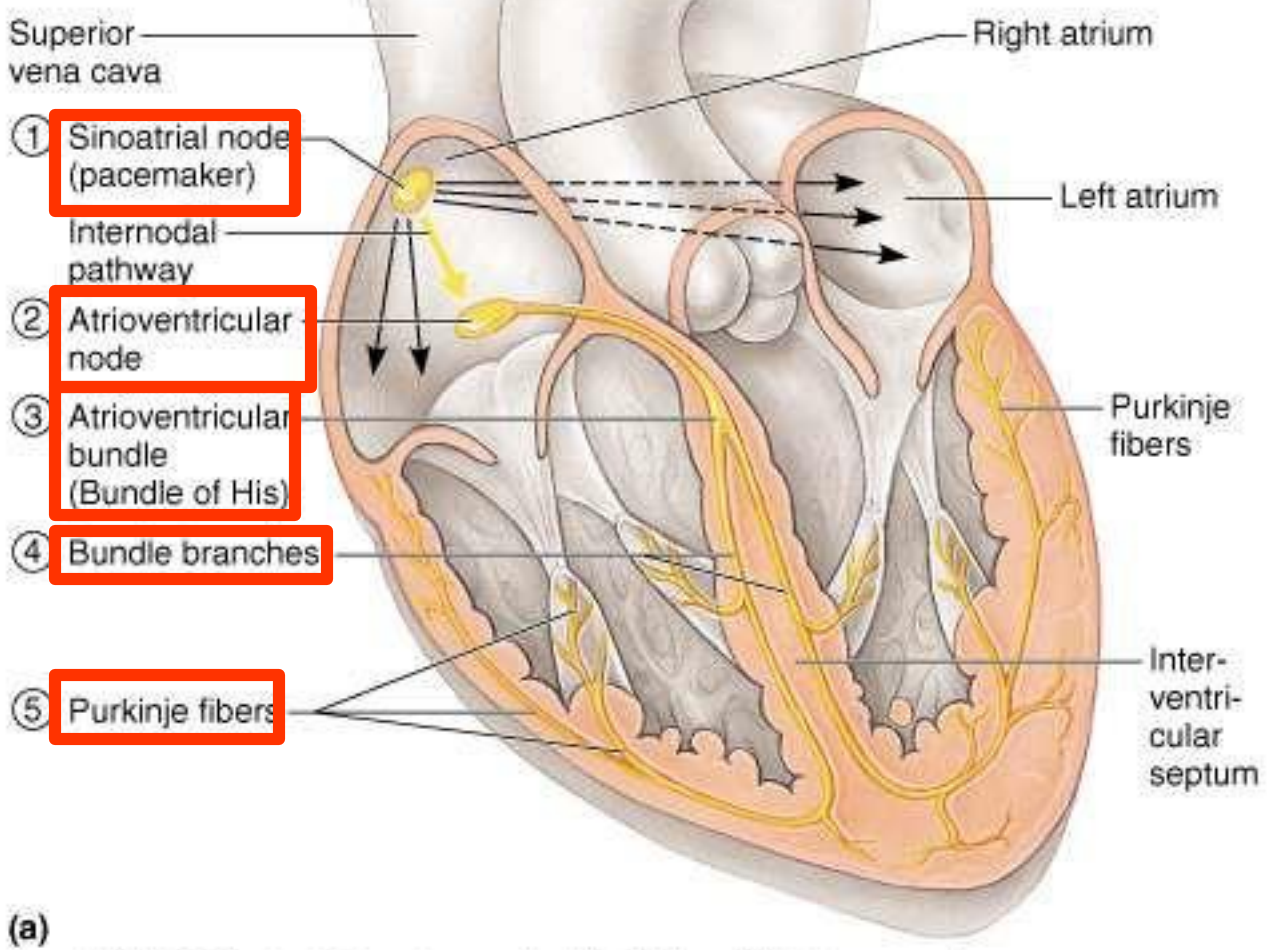
Intrinsic Cardiac Conduction System

Approximately 1% of cardiac muscle cells are autorhythmic rather than contractile

70-80/min

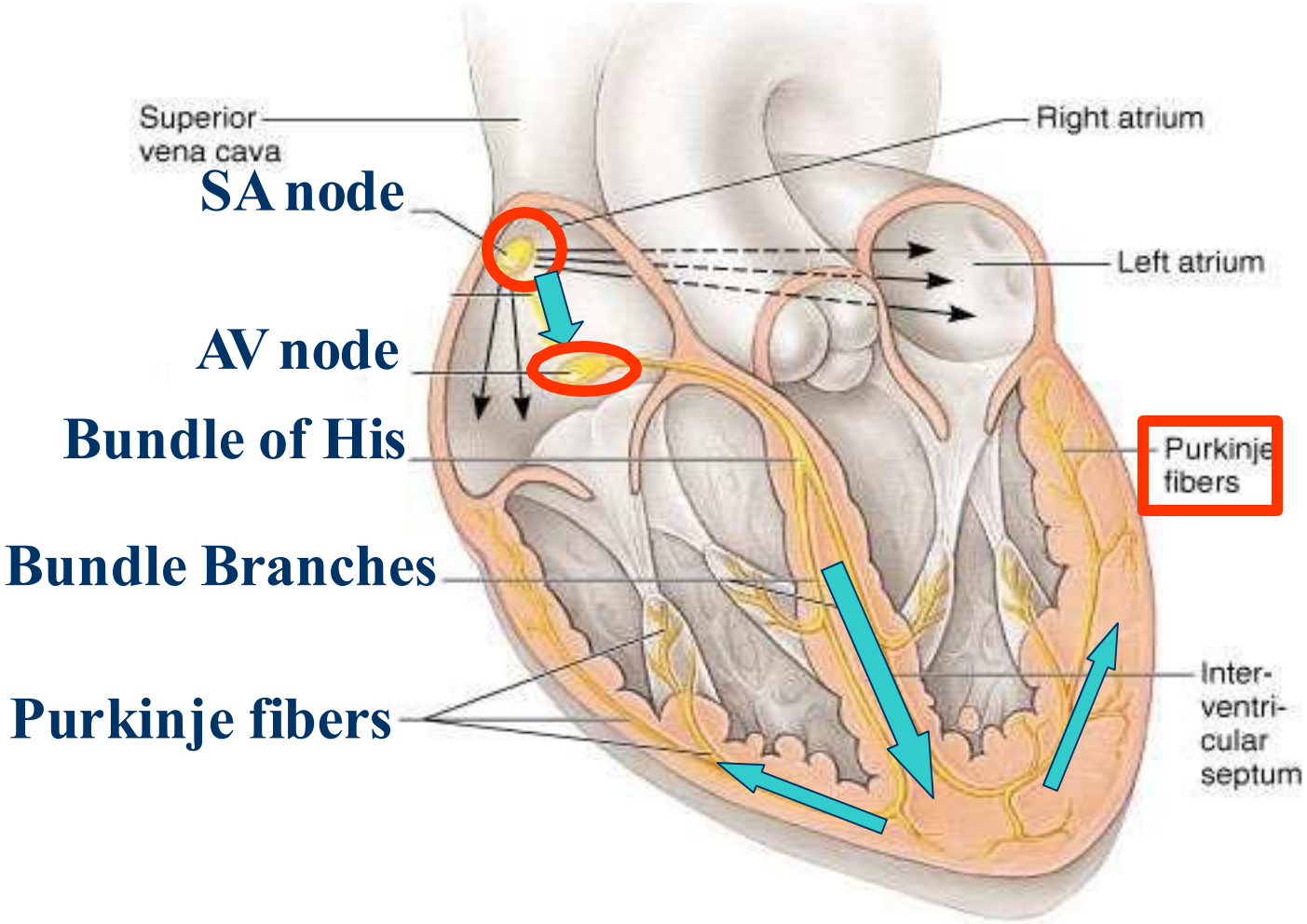
40-60/min

15-40/min



Intrinsic Conduction System

Function: initiate & distribute impulses so heart depolarizes & contracts in orderly manner from atria to ventricles.



Internodal Pathways ??

- Transmits cardiac impulse throughout atria
- Anterior, middle, and posterior internodal pathways
- Anterior interatrial band carries impulses to left atrium
- Some researchers propose that the **sinoatrial (SA) node** and **atrioventricular (AV) node** are connected by **internodal pathways.** However, our doctor **believes** that these distinct fibers **are not anatomically proven**.
- Instead, the impulse generated by the **SA node** spreads throughout the **atrial myocardium**, and because the **AV node lies within this muscle**, it **receives the depolarization directly** through the surrounding atrial fibers.
- The **AV node** then transmits the impulse to the **atrioventricular bundle (Bundle of His)**, which continues into the **right and left bundle branches**, and finally into the **Purkinje fibers.**

Components of the Conduction System of the Heart

- SA (sinoatrial) node (Pacemaker)
- Conduction system parts are **modified** cardiac muscle cells, consisting of:
    1. AV (atrioventricular) node
    2. A-V (atrioventricular) bundle
    3. Bundle branches (right and left bundle branches)
    4. Purkinje fibers
- All components of the conduction system can generate **intrinsic action potentials**, but each at a **different frequency**.
- These conduction fibers represent **less than 1% of all cardiac muscle cells** and are termed **autorhythmic cells**, as they **generate rhythmic impulses rather than contract**.
- The remaining **99%** are **contractile cells** responsible for the mechanical pumping action of the heart.

Pathway of the Heartbeat

1. **Begins in the sinoatrial (S-A) node**
2. **Internodal pathway to atrioventricular (A-V) node ??**
3. **Impulse delayed in A-V node** (allows atria to contract before ventricles)
4. **A-V bundle** takes impulse into ventricles
5. **Left and right bundles of Purkinje fibers** take impulses to all parts of ventricles

Atrioventricular (AV) Nodal Delay and Coordination of Contraction

- In the **atrioventricular (AV) node**, the electrical impulse is **delayed** before it passes to the ventricles.
- After the **sinoatrial (SA) node** generates an action potential, **atrial depolarization** occurs, leading to **atrial contraction (systole)**.
- **Depolarization** represents the **electrical activation** of the muscle, followed by **mechanical contraction**, while **repolarization** corresponds to **relaxation (diastole)**.
- The **AV nodal delay** ensures that the **atria complete their contraction** and fully empty blood into the ventricles **before ventricular contraction begins**.
- If both the atria and ventricles contracted simultaneously, it would result in **inefficient pumping and impaired cardiac function**.
- Thus, the **AV node slows conduction** to coordinate **atrial systole** before **ventricular systole**.

Sinus Node

- Specialized cardiac muscle connected to atrial muscle.
- Acts as pacemaker because membrane leaks **Na^+** and **membrane potential is -55 to -60 mV**.
- When membrane potential reaches **-40 mV**, slow Ca^{++} channels open causing **action potential**.
- After **100–150 msec**, **Ca^{++} channels close** and **K^+ channels open** more, thus returning membrane potential to **-55 mV**.

A-V Node

- **Delays** cardiac impulse
- Most delay is in A-V node
- Delay A-V node — 0.09 sec
- Delay A-V bundle — 0.04 sec

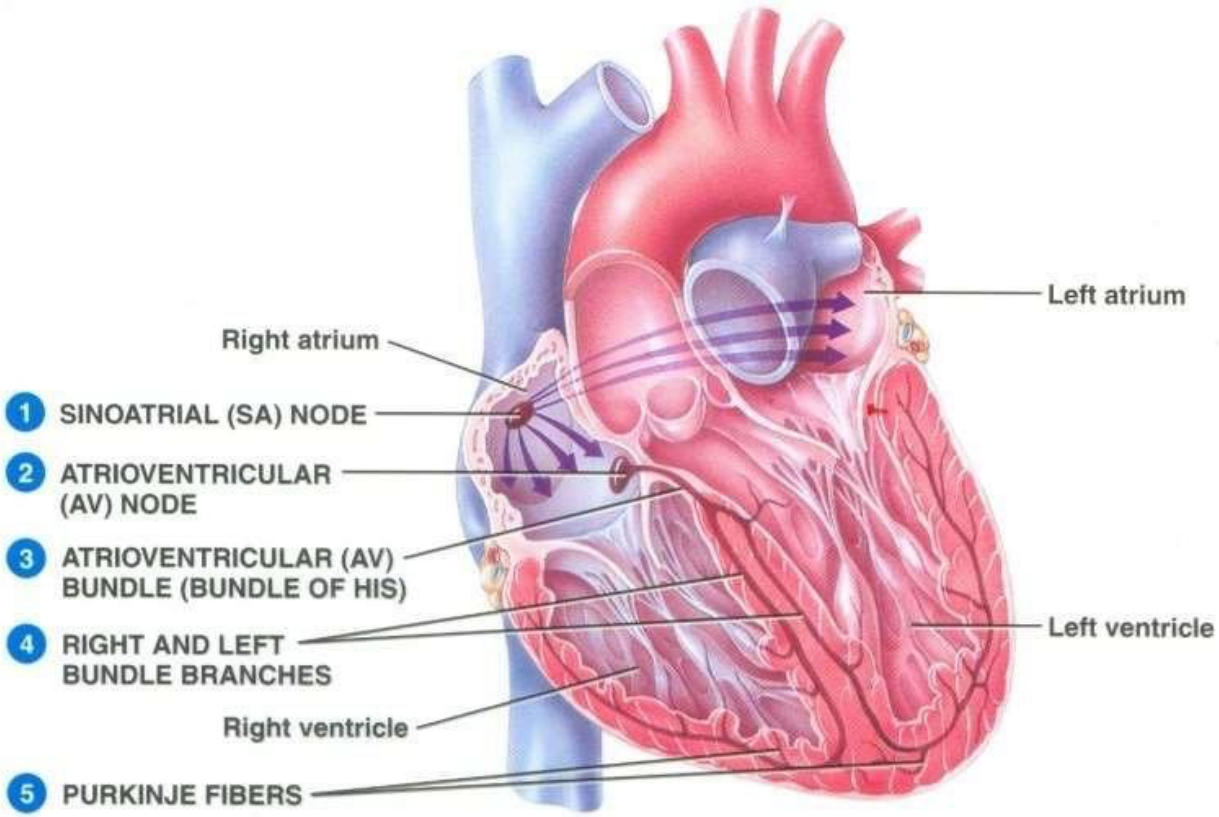
Do NOT memorize the numbers.

Purkinjie System

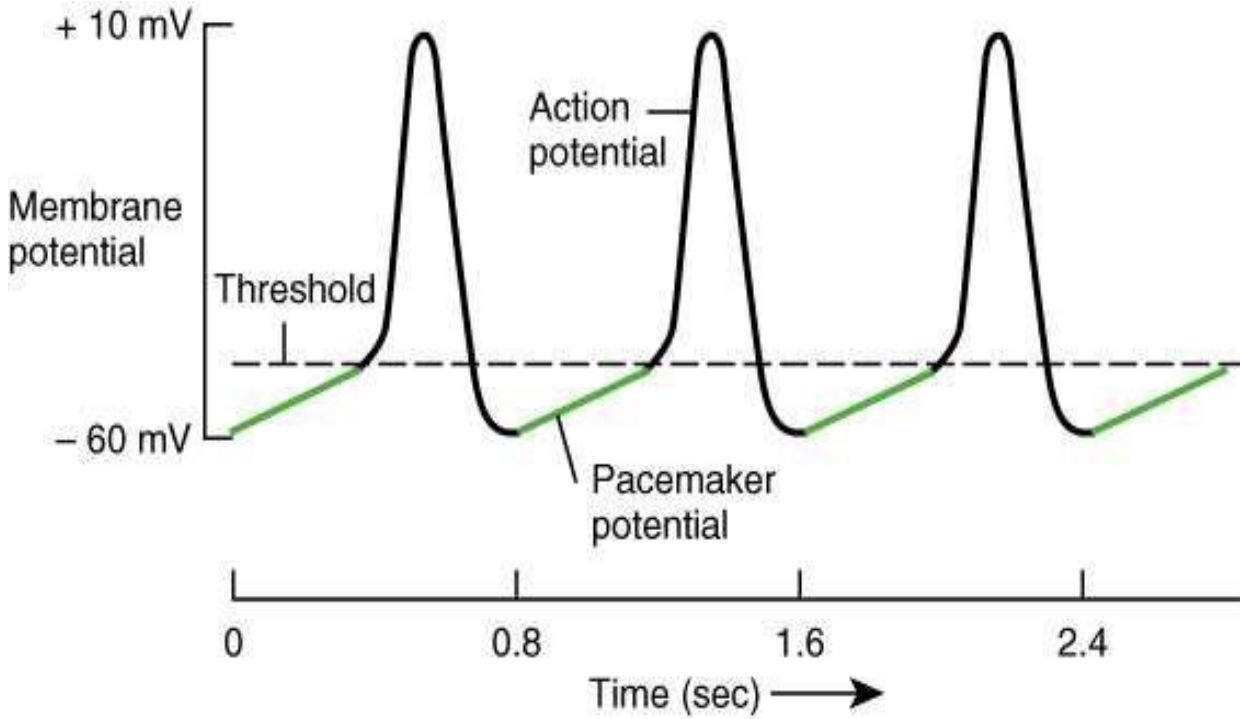
- Fibers extend **from** the A-V node through the A-V bundle **into** the ventricles
- Conduction is **very fast** due to numerous **gap junctions** located at the **intercalated disks**.
- The **Purkinje fibers** have the **lowest intrinsic pacemaker rate**, yet they are the **fastest conducting fibers** in the heart.
- They **resemble ordinary cardiac muscle cells** but possess a **larger diameter**, which **facilitates rapid impulse conduction**.
- Additionally, they contain **numerous gap junctions**, while the **SA and AV nodes** have **fewer gap junctions**, leading to **slower conduction** in those regions.

A-V Bundles

- Normally one-way conduction through the bundles
  - In the **atrioventricular (AV) bundle**, electrical conduction **normally proceeds in one direction only**—from the **atria to the ventricles**.
  - If an impulse were to **travel backward**, it would reach regions of the conduction system that are in the **absolute refractory period**, during which **cardiac cells cannot be re-excited**.
  - This mechanism **ensures one-way conduction** and maintains the **coordinated rhythm of the heartbeat**.
- Only conducting path between atria and ventricles is A-V node → A-V bundle
- Divides into left and right bundles
- Transmission time between A-V bundles and last of ventricular fibers is 0.06 second (QRS time)
  - This means that it takes about **0.06 seconds (60 milliseconds)** for the **electrical impulse** to travel **from the A-V bundle (Bundle of His)** through the **right and left bundle branches** and then through the **Purkinje fibers** to reach **all the ventricular muscle fibers**.



(a) Anterior view of frontal section



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

20.10b

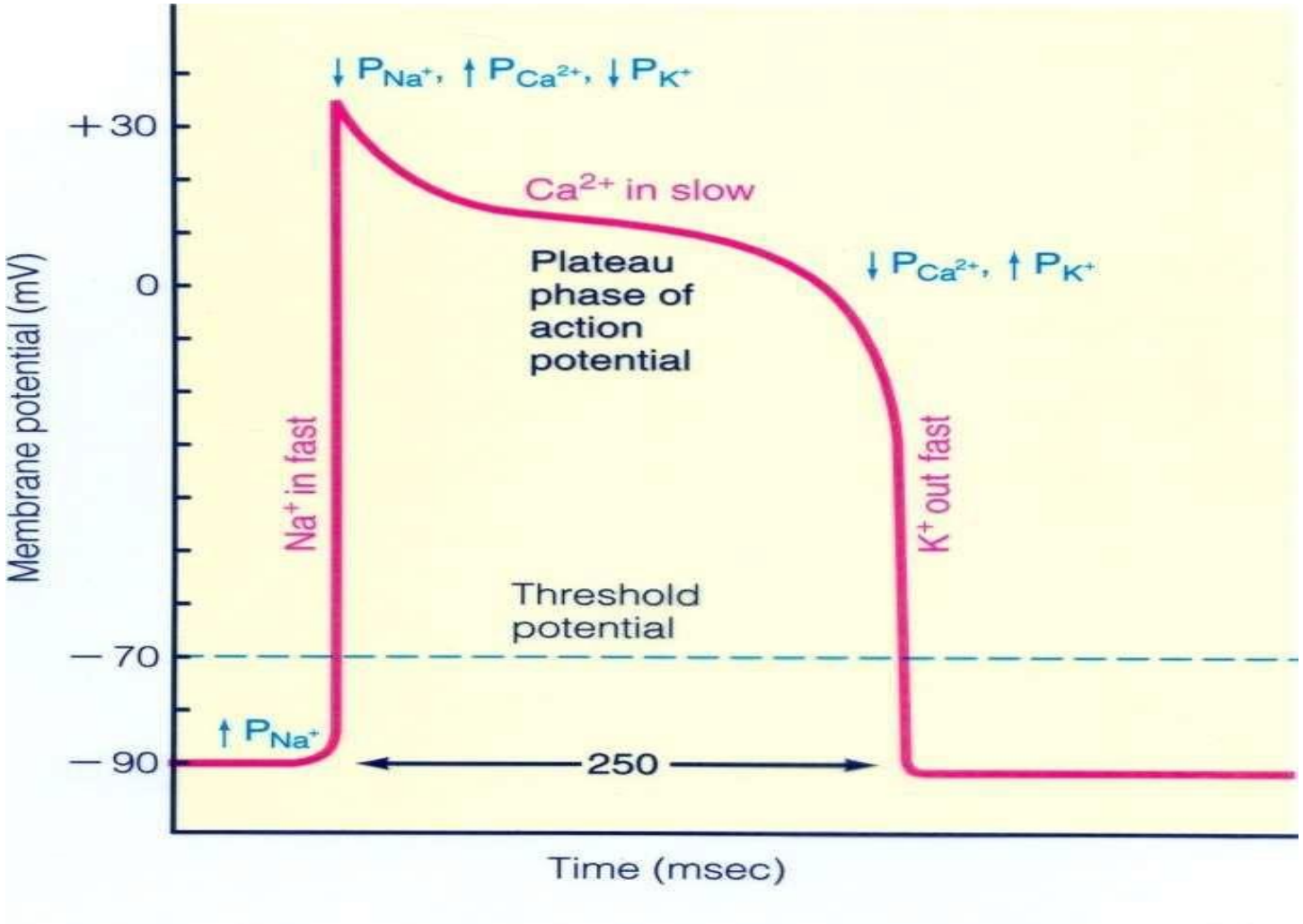
Mechanism of Intrinsic Activity

- **Structurally, autorhythmic cells** are **round**, have **fewer intercalated discs** and **gap junctions**, and **lack contractile filaments**, which means they **cannot contract**.
- **Functionally**, these cells are **leaky to sodium (Na^+)** – during **phase 4**, sodium **slowly enters through "funny" Na^+ channels (If)**.
- Because of this, the **resting membrane potential** does not reach **–90 mV** as in contractile cells but remains around **–60 mV**.
- This **gradual Na^+ influx** causes **slow depolarization** during **phase 4** until the cell reaches **threshold potential**.
- In **contractile cells**, fast depolarization (phase 0) results from **voltage-gated Na^+ channel activation**, involving both **activation** (extracellular) and **inactivation** (intracellular) gates.
- However, in **pacemaker cells**, depolarization occurs **slowly**, allowing the inactivation gates to close before activation occurs – thus, **voltage-gated Na^+ channels do not open**.
- Instead, **slow voltage-gated Ca^{2+} channels** open, producing the **slow depolarization of phase 0 in SA and AV nodal cells**.

Mechanism of Intrinsic Activity

- There is **no plateau phase** in pacemaker cells.
- **Repolarization (phase 3)** follows due to **K^+ efflux**, returning the membrane toward its resting potential.
- This entire cycle forms the **pacemaker potential** or **slow-response action potential**.
- **Resting membrane potentials:**
  - SA node: approximately **–60 mV** (least negative)
  - AV node: slightly **more negative**
  - Purkinje fibers: **most negative**
- **Slope of phase 4 depolarization:**
  - **Steepest** in the **SA node** (fastest rate)
  - **Less steep** in the **AV node**
  - **Shallowest** in the **Purkinje fibers** (slowest rate)

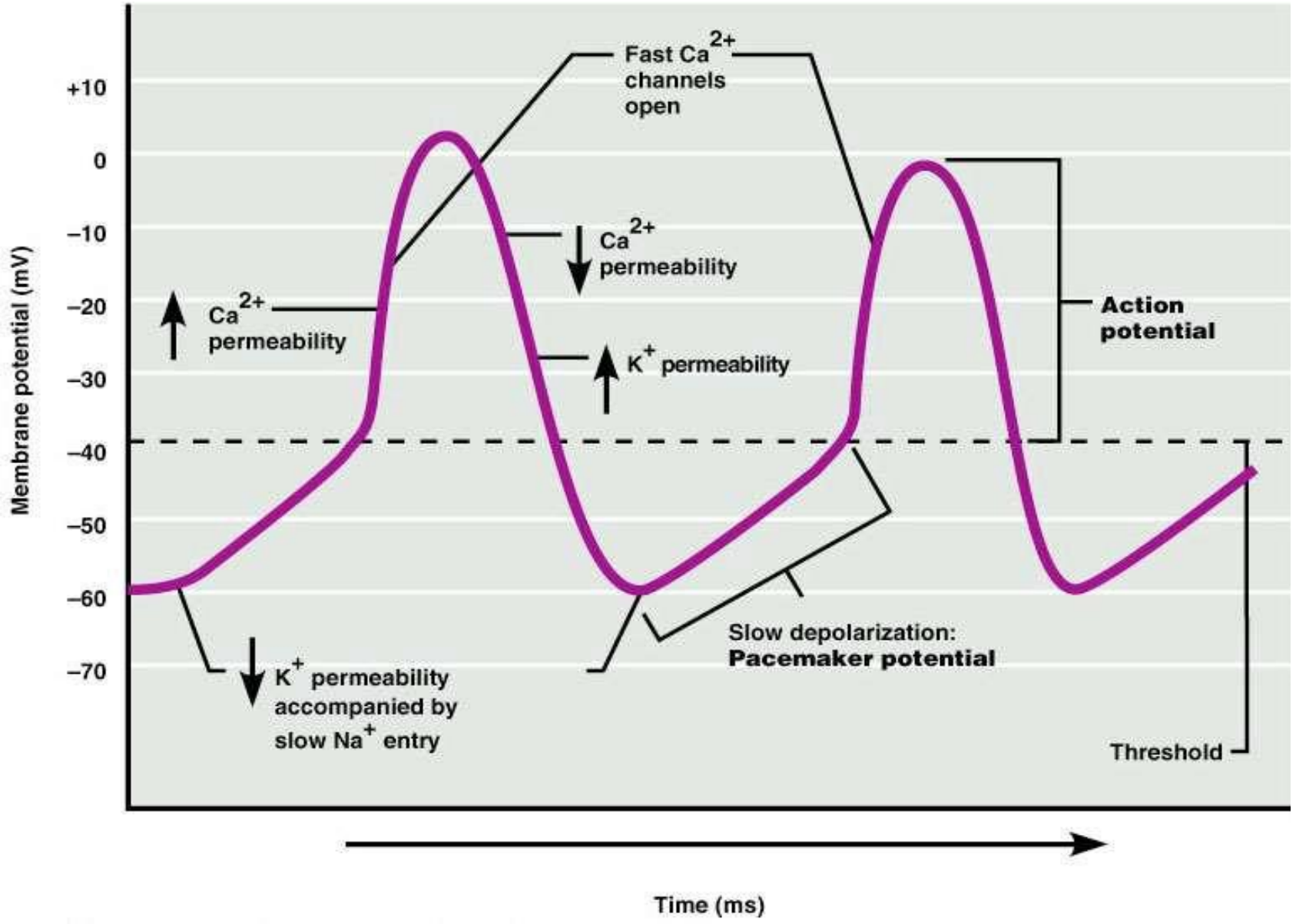
Fast Response Action Potential of Contractile Cardiac Muscle Cell



Fast Response Action Potential of Contractile Cardiac Muscle Cell

- This slide shows the **contractile cardiac muscle (fast-response)** action potential, which has five phases: 0, 1, 2, 3, and 4.
- In **phase 4, the membrane potential is stable (resting) rather than slowly depolarizing**; it is **not driven by a Na^+ leak.**
- **This slide itself illustrates the fast-response action potential typical of contractile cells.**
- **This is not what was said in the lecture.**

Pacemaker & Action Potentials of the Heart



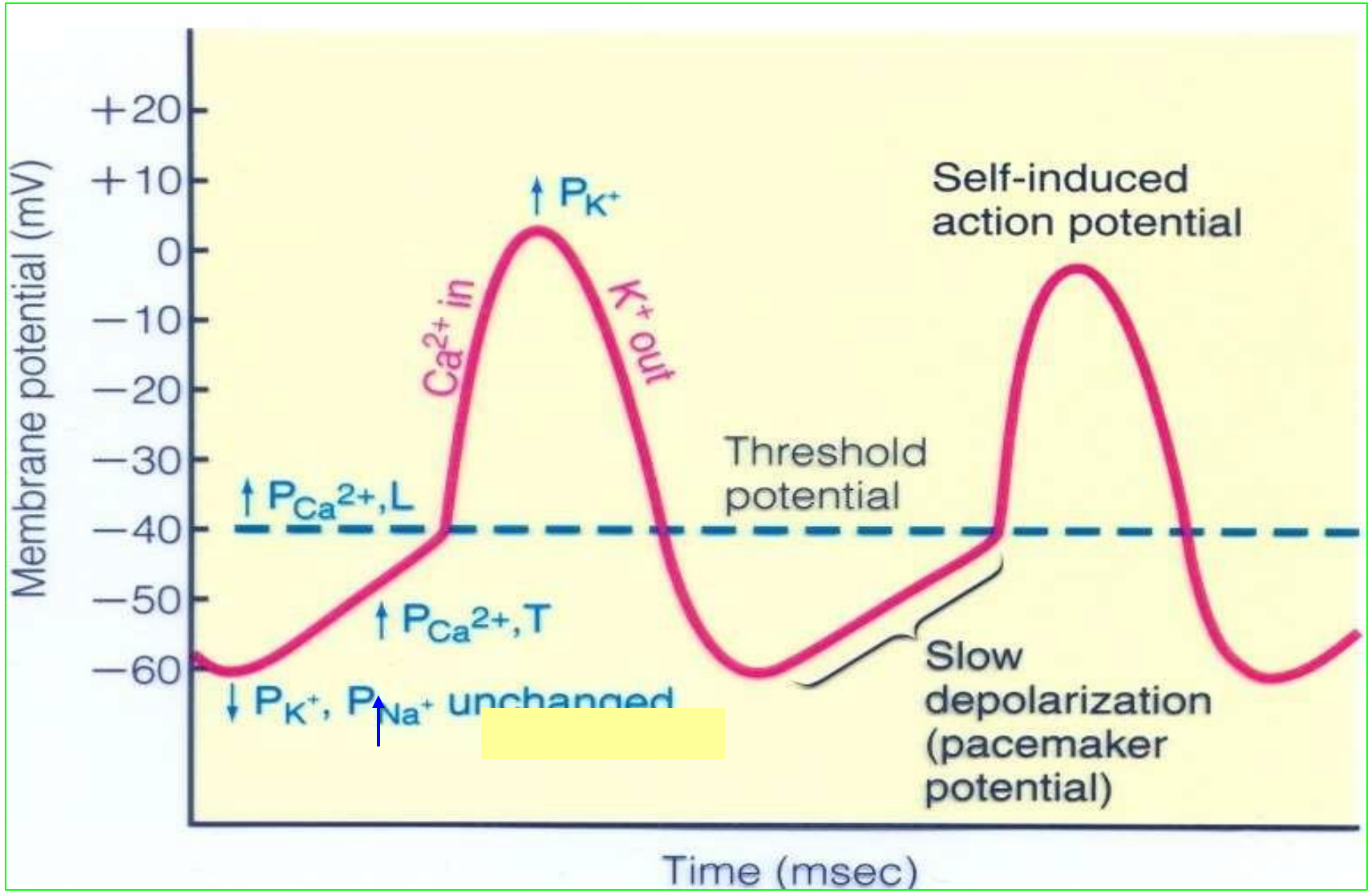
Pacemaker & Action Potentials of the Heart

- This slide shows the **slow-response action potential**, also known as the **pacemaker potential**.
- In this type, the cell reaches **threshold gradually**, because the **inactivation gate closes before the activation gate opens** for the **voltage-gated Na^+ channels**.
- As a result, **Na^+ does not enter the cell through these channels**.
- Instead, **slow voltage-gated Ca^{2+} channels** open, allowing **Ca^{2+} influx during phase 0**.
- **Phase 0:** Increased **Ca^{2+} permeability (conductance)** through **L-type Ca^{2+} channels** causes **slow depolarization**.
- **Phase 4:** There is a **slow Na^+ leak** through **funny Na^+ channels (If)**, producing **gradual depolarization toward threshold**.
- **Phase 3: K^+ permeability increases** and **Ca^{2+} permeability decreases**, leading to **repolarization**.

Pacemaker & Action Potentials of the Heart

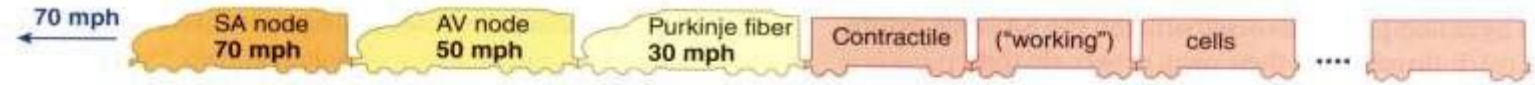
- Because these cells can **generate impulses spontaneously**, they are called **autorhythmic cells**.
- This type of action potential occurs in both the **SA node** and the **AV node**, but with subtle differences:
  - The **AV node** begins from a **more negative potential**,
  - Takes **longer to reach threshold**, and
  - Therefore, **depolarizes more slowly**.
- Although the **SA and AV nodes** share the same basic pattern, the **AV node has a less steep slope** during phase 4.
- The slope ($\Delta V/\Delta T$) represents the **rate of voltage change over time**:
  - The **AV node** has a **smaller slope** than the **SA node**.
  - By contrast, **contractile muscle fibers** have a **very steep slope during phase 0**, reflecting their **rapid depolarization** compared to slow-response cells.

Slow Response Action Potential (Pacemaker Potential)

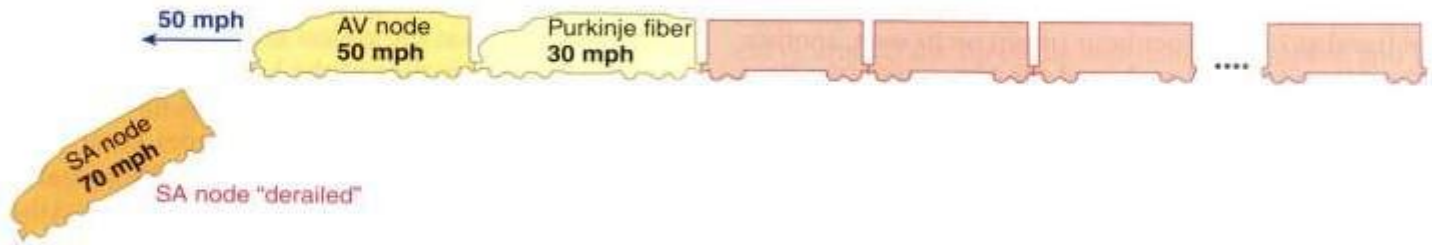


Intrinsic Rate & Speed of Conduction of the Components of the System

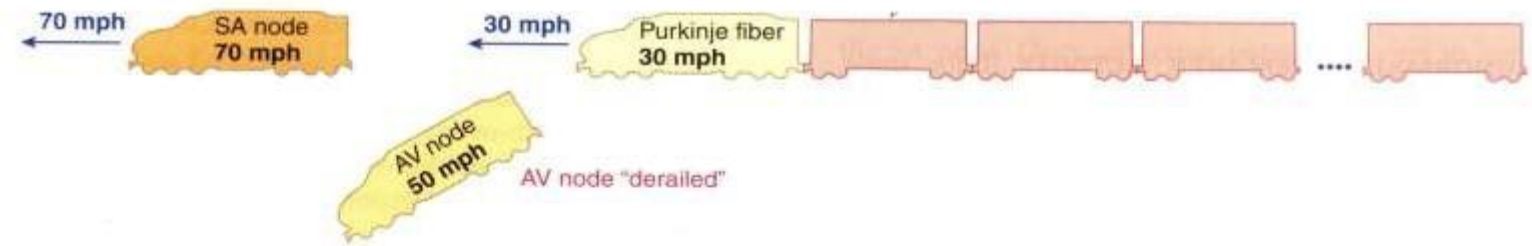
- **SA node:** 60–80 action potentials/min (primary & fastest *Pacemaker*)
- **AV node:** 40–60 action potentials/min
- **Purkinje:** 15–40 action potentials/min (slowest pacemaker)
- Conduction Speed:
  - **SA node:** slow speed of conduction
  - **Ventricular and Atrial muscle** moderate speed
  - **AV node:** slowest speed of conduction
  - **Purkinje fibers:** fastest speed of conduction
- Ectopic Pacemaker – Abnormal site of pacemaker



(a) Normal pacemaker activity: Whole train will go **70 mph** (heart rate set by SA node, the fastest autorhythmic tissue).



(b) Takeover of pacemaker activity by AV node when the SA node is nonfunctional: Train will go **50 mph** (the next fastest autorhythmic tissue, the AV node, will set the heart rate).



(c) Takeover of ventricular rate by the slower ventricular autorhythmic tissue in complete heart block: First part of train will go **70 mph**; last part will go **30 mph** (atria will be driven by SA node; ventricles will assume own, much slower rhythm).

(d) Takeover of pacemaker activity by an ectopic focus: Train will be driven by ectopic focus, which is now going faster than the SA node (the whole heart will be driven more rapidly by an abnormal pacemaker).

Pacemaker Hierarchy & Ectopic Activity in the Heart

- The **sinoatrial (SA) node** is known as the **normal pacemaker of the heart**. as the SA node is the **fastest component** of the conduction system.
- If the SA node becomes **damaged or dysfunctional**, the **atrioventricular (AV) node** can **assume the pacemaker role**.
- In this case, it is termed an **ectopic pacemaker**, since it lies **outside the normal pacemaking site**.
- If the **AV node** is also impaired, the **Purkinje fibers** may generate impulses and act as the pacemaker, though this is **still ectopic** and occurs at a **much slower rate**.
- Occasionally, another **region within the ventricles** may begin firing impulses **faster than the SA node**. This abnormal site is called an **ectopic focus**.
- Although it can cause the **ventricles to contract more rapidly**, it is **not considered normal** because it originates **outside the SA node**.

The term “pace” refers to rate or speed.

Role of Purkinje Fibers in Rapid Ventricular Conduction and Synchronous Contraction

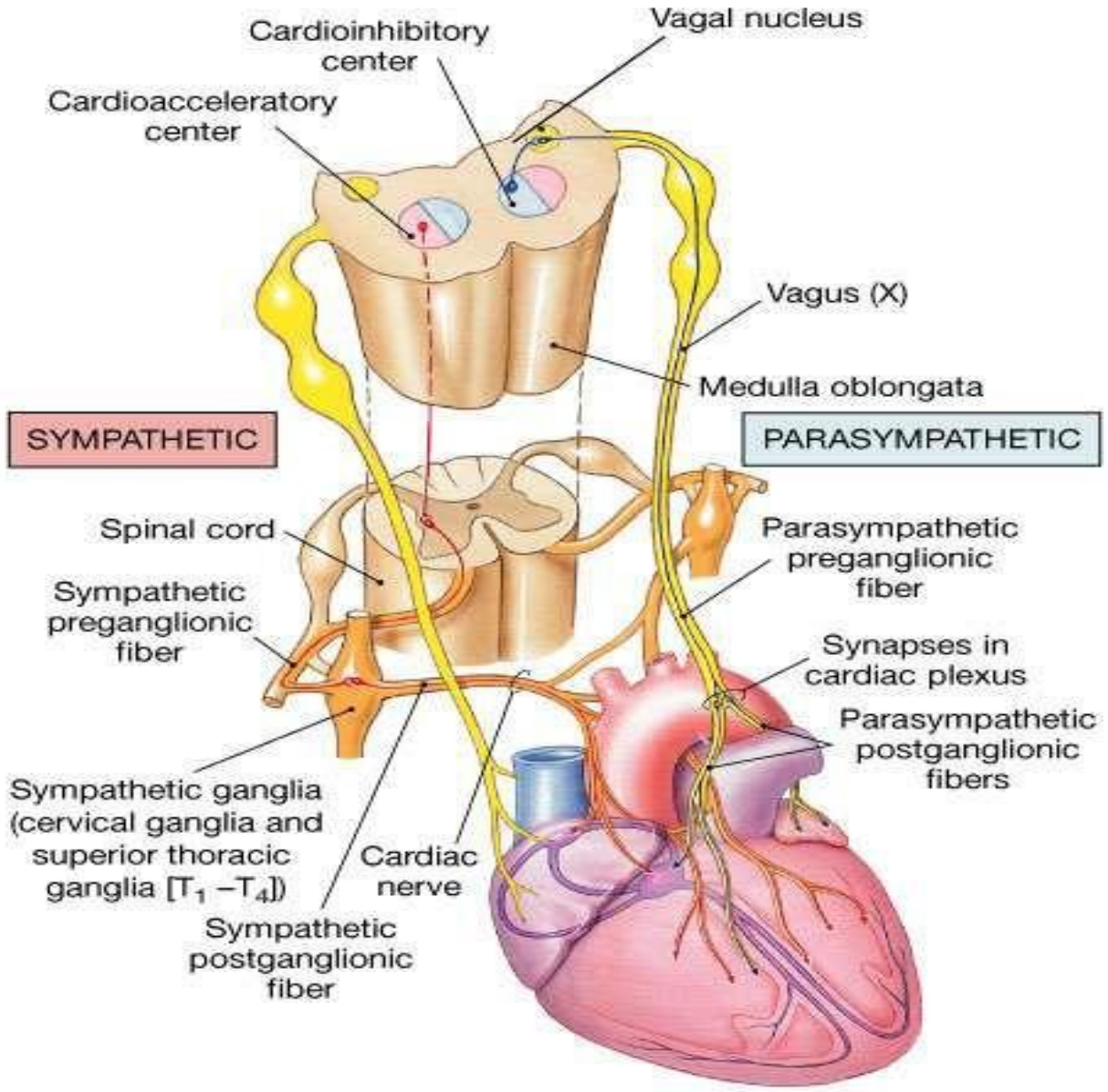
- The **Purkinje fibers** exhibit the **fastest conduction speed** because they possess a **large diameter**, comparable to that of **contractile cardiac muscle fibers**.
- Although their **intrinsic pacemaker rate** is the **slowest**, their **conduction velocity** is the **highest** among all components of the conduction system.
- When the impulse reaches the Purkinje fibers, it is **transmitted rapidly and almost simultaneously** to all regions of the ventricles.
- This ensures that **both ventricles contract together**, allowing the heart to function as a **single, coordinated pump**.
- Simultaneous ventricular contraction generates **high intraventricular pressure**, producing an **effective and powerful pumping action**.

Ventricular Fibrillation and the Importance of Coordinated Conduction

- If the impulse were conducted to different ventricular fibers at **different times**, some regions would **contract while others relax**, leading to **ventricular fibrillation**.
- In **ventricular fibrillation**, the **ventricular muscle fibers contract independently and irregularly**, resulting in a **loss of effective pumping** and is **potentially fatal**.
- This can occur during **myocardial infarction (heart attack)** and requires **immediate medical intervention**.
- As mentioned earlier, the **A-V node introduces a conduction delay**, ensuring that **atrial contraction is completed** before **ventricular contraction begins**.

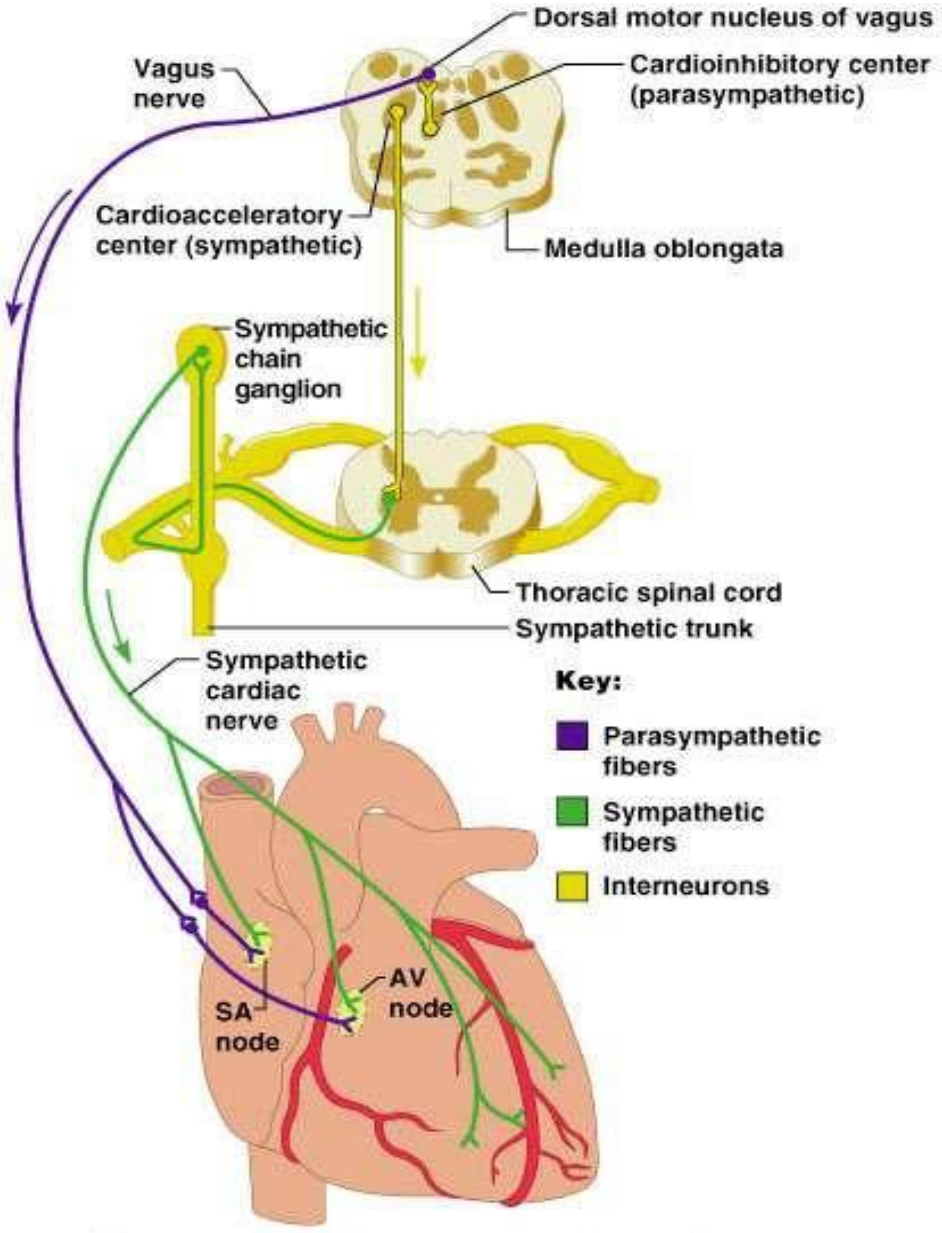
Autonomic Innervation of the Heart

- The heart receives **parasympathetic innervation** from the **vagus nerve (cranial nerve X)**, which primarily supplies the **atria, SA node, and AV node**, with **minimal innervation to the ventricles**.
- In contrast, **sympathetic fibers** arise from the **cardiac plexus**, originating in the **cervical and upper thoracic ganglia (T1–T4)**, and **innervate all regions of the heart**.



Extrinsic Innervation of the Heart

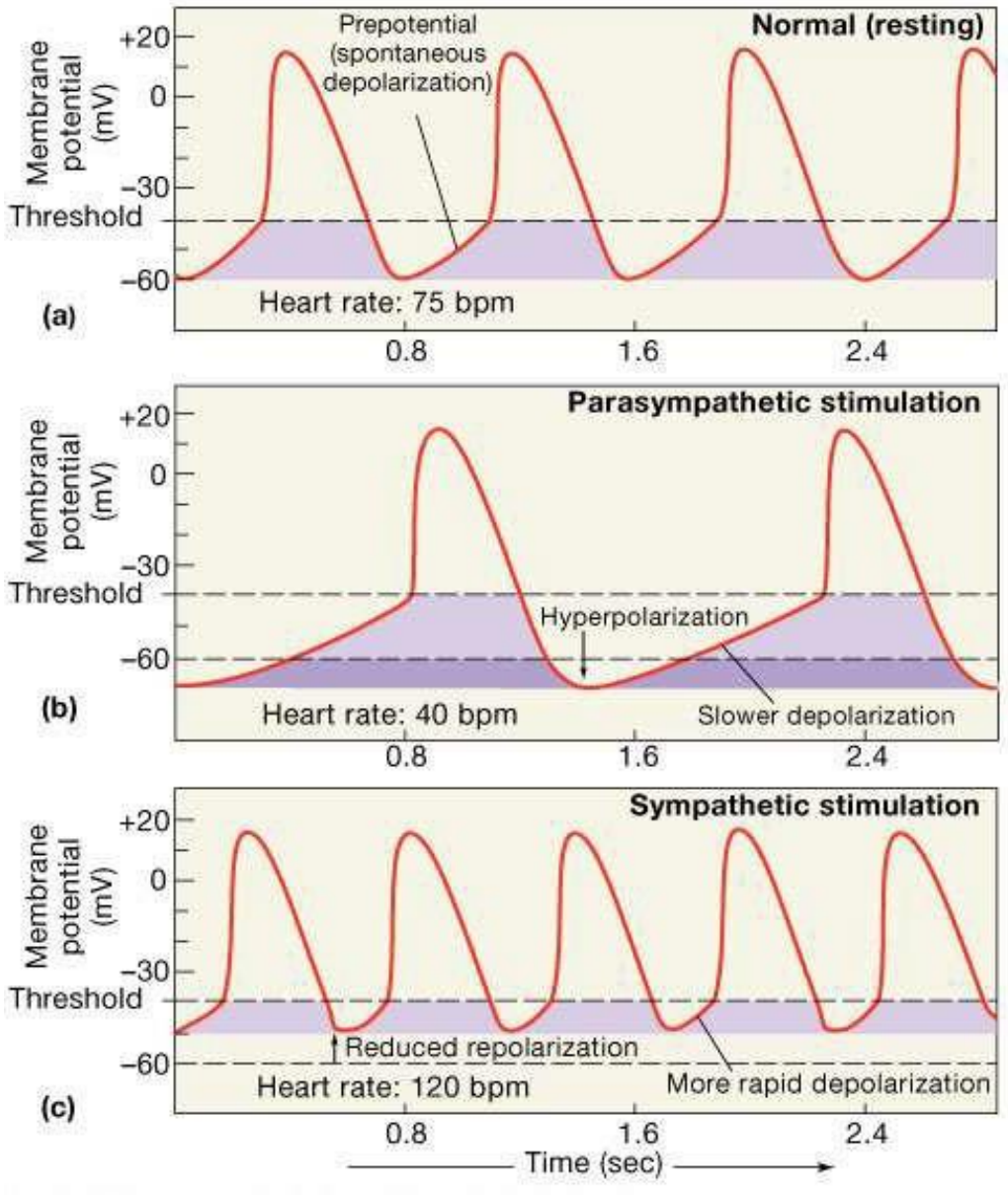
- Vital centers of medulla oblongata
  1. Cardiac Center
     - **Cardioaccelerator center:** Activates sympathetic neurons that increase HR and myocardial contractility.
     - **Cardioinhibitory center:** Activates parasympathetic neurons that decrease HR
- Cardiac center receives input from higher centers (particularly the hypothalamus), which monitors blood pressure and dissolved gas concentrations



Extrinsic Innervation of the Heart

- Both the **sympathetic** and **parasympathetic** nerves that regulate cardiac activity originate from the **medulla oblongata**.
- The **parasympathetic influence on contractility is minimal** because the **vagus nerve** primarily innervates the atria and conduction system, with **little or no supply to the ventricles**.
- Therefore, it mainly affects the **rate of contraction**, not the **force**.

Pacemaker Function



Normal Pacemaker & Parasympathetic Stimulation

1. **Normal Pacemaker**
   - Represents the **normal activity of the sinoatrial (SA) node**, which initiates each heartbeat under resting conditions.
2. **Parasympathetic Stimulation**
   - The **parasympathetic neurotransmitter, acetylcholine (ACh), increases K^+ permeability** and **decreases Na^+ and Ca^{2+} permeability.**
   - **Increased K^+ efflux** makes the membrane potential **more negative (hyperpolarized)**, around **–65 to –70 mV**.
   - **Reduced Na^+ and Ca^{2+} influx** slows **phase 4 depolarization**, delaying the threshold potential.
   - **As a result:**
     - The **rate of depolarization decreases**.
     - The **heart rate decreases (negative chronotropic effect)**.

Normal Pacemaker & Parasympathetic Stimulation

- Since the **vagus nerve does not innervate the ventricles**, **ventricular contractility remains unaffected**.
- **Summary of Parasympathetic Effects:**
  - **Chronotropic (rate):** Negative → ↓ heart rate
  - **Inotropic (contractility):** Minimal effect (atria only)
  - **Dromotropic (conduction):** Negative → ↓ conduction velocity (mainly in atria and AV node)
- Thus, parasympathetic stimulation **reduces SA node firing rate**, **slows AV conduction**, and **decreases atrial contractility**.

Sympathetic Stimulation

3. Sympathetic Stimulation

- The **sympathetic neurotransmitters – norepinephrine and epinephrine – increase Na^+ and Ca^{2+} permeability** while **reducing K^+ permeability.**
- **Consequences:**
  - The **slow depolarization phase (phase 4)** becomes **steeper and faster.**
  - The **resting membrane potential** becomes **less negative (~–55 mV).**
  - The **rate of depolarization** increases, leading to a **faster heart rate (positive chronotropic effect).**
- Because the **sympathetic fibers also innervate the ventricles**, they:
  - **Increase intracellular and sarcoplasmic Ca^{2+} levels,**
  - **Enhance the force of contraction (positive inotropic effect),** and
  - **Increase conduction velocity (positive dromotropic effect).**

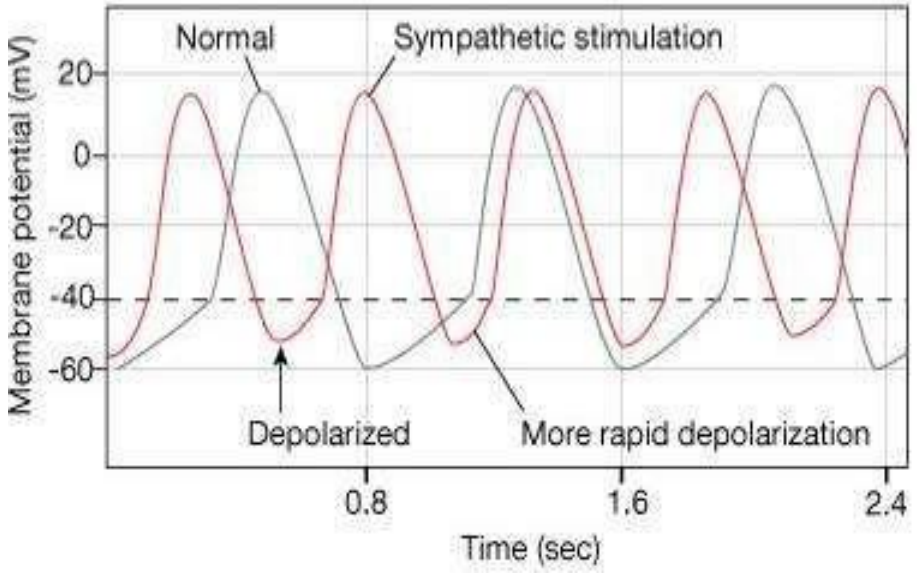
Sympathetic Stimulation

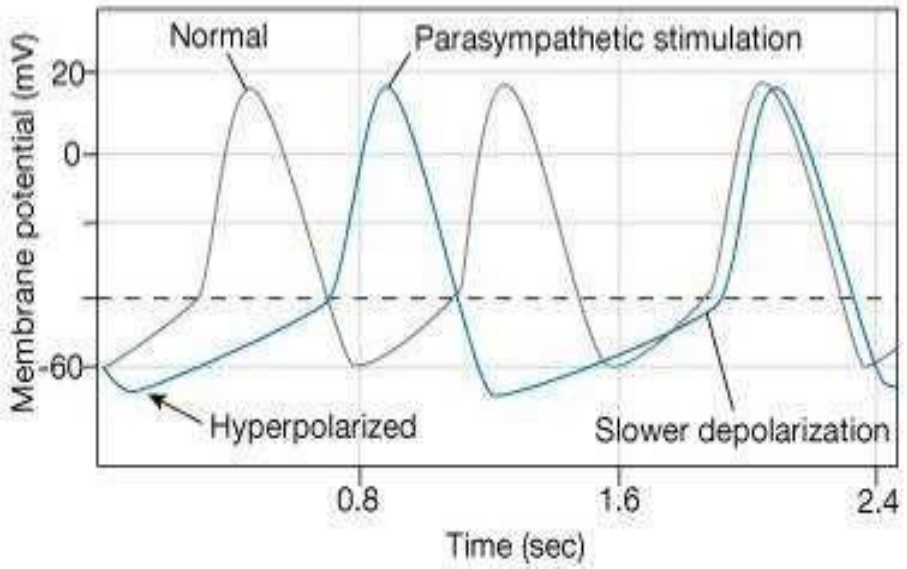
- **Summary of Sympathetic Effects:**
  - **Chronotropic:** Positive → ↑ heart rate
  - **Inotropic:** Positive → ↑ contractility
  - **Dromotropic:** Positive → ↑ conduction speed
- In all cases, the **shape of the action potential peak remains unchanged**.
- The differences appear in:
  - The **resting membrane potential**,
  - The **rate (slope) of phase 4 depolarization**, and
  - The **time required to reach threshold**.

Autonomic Neurotransmitters Affect Ion Flow to Change Rate

- **Sympathetic** – increases heart rate by ↑ Ca^{+2} & I_f channel (net Na^+) flow
- **Parasympathetic** – decreases rate by ↑ K^+ efflux & ↓ Ca^{+2} influx

What part of the graph is not changed by autonomic influences?





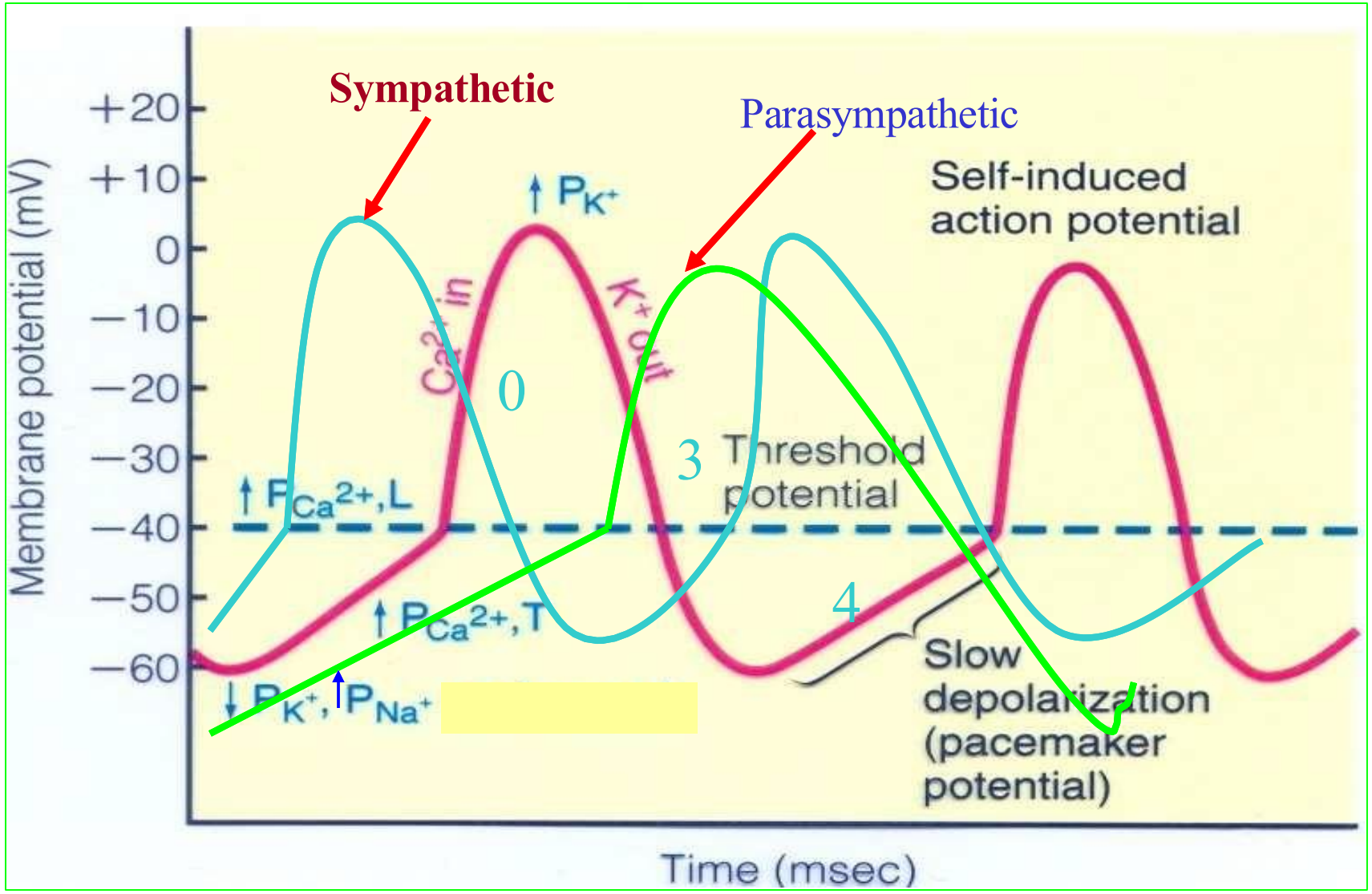
Autonomic neurotransmitters affect ion flow to change rate

- Under parasympathetic stimulation, there is an **increase in K^+ efflux** and a **decrease in Na^+ and Ca^{2+} influx**.
- The **phase 0 of the action potential does not change**.
- However, the **rate of depolarization** and the **resting membrane potential** do change:
- During **parasympathetic activity**, the cell becomes **hyperpolarized** (more negative).
- During **sympathetic activity**, the cell becomes **depolarized** (less negative) due to **increased Na^+ and Ca^{2+} permeability**.

Effect of autonomic nerve activity on the heart

Region affected	Sympathetic Nerve	Parasympathetic Nerve
SA node	Increased rate of diastole depolarization ; increased cardiac rate	Decreased rate of diastole depolarization ; Decreased cardiac rate
AV node	Increase conduction rate	Decreased conduction rate
Atrial muscle	Increase strength of contraction	Decreased strength of contraction
Ventricular muscle	Increased strength of contraction	No significant effect

Effect of Sympathetic & Parasympathetic Stimulation



Regulation of the Heartbeat

- Sympathetic from the cardiac plexus supplies all parts of the heart (atria, ventricle and all parts of the conduction system).
- Parasympathetic from **Vagus** nerves supply mainly the **atria, SA and AV nodes**, very little supply to ventricles.
- Sympathetic: increase the permeability of the cardiac cells to Na^{+} and Ca^{++} i.e. Positive **Chronotropic** and positive **Inotropic** action.
- Parasympathetic: Increase the permeability of the cardiac cells to K^{+} and decrease its permeability to Na^{+} and Ca^{++}.
- Negative **Chronotropic** effect and ?? **Inotropic effect**.
- Ventricular Escape and Overdrive suppression.

Regulation of the Heartbeat

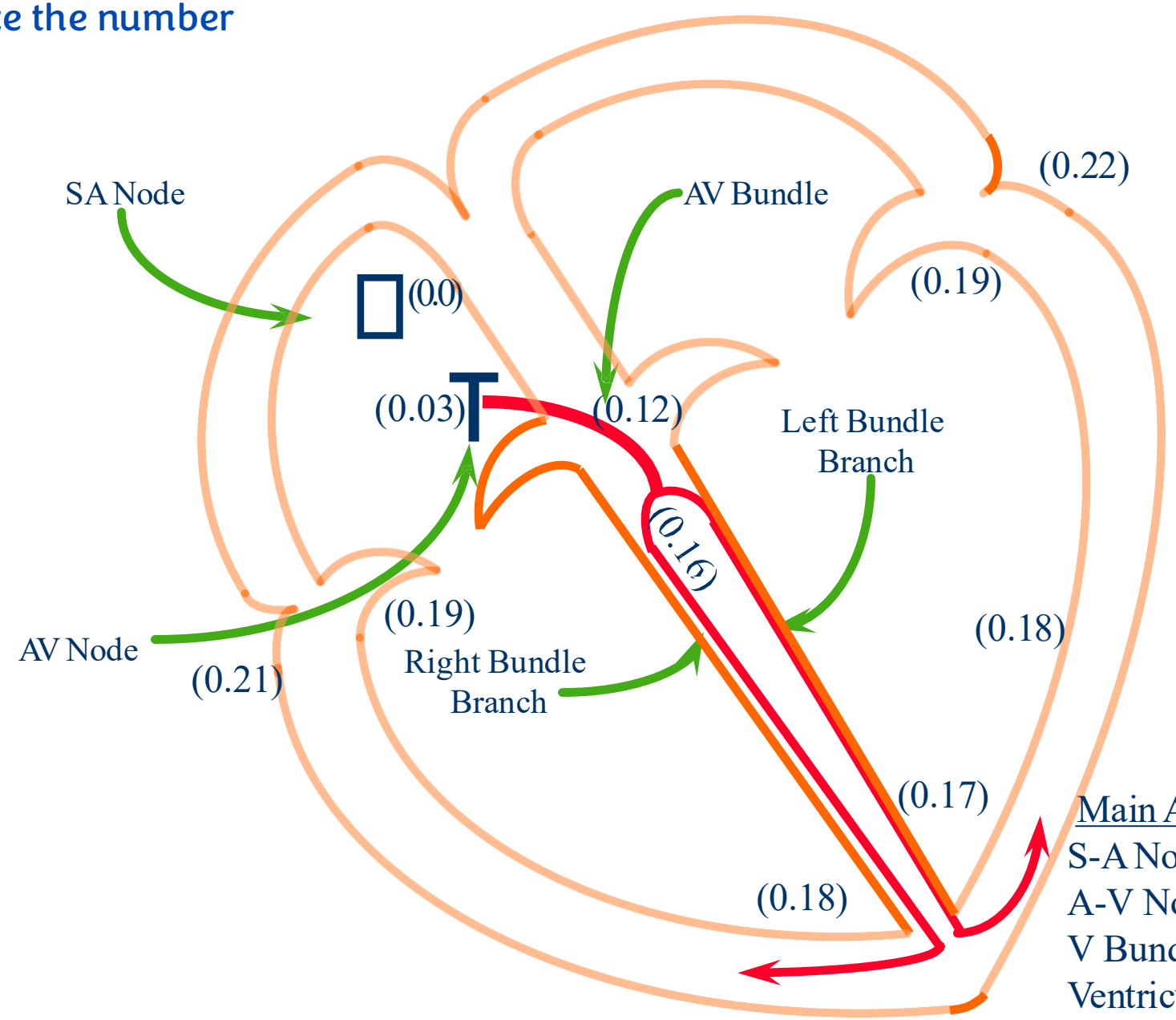
- When the parasympathetic system is strongly stimulated — for example, due to excessive vagus nerve activation — it can cause the heart rate to slow dramatically or even stop temporarily.
- This may occur if pressure is applied to the carotid sinus, which triggers vagal stimulation.
- During strong vagal stimulation:
  - The heart **stops** beating temporarily.
  - The person may **faint** due to the lack of blood flow.
- After about **15–30 seconds**, the heart resumes beating but at a slower rate. This happens because:
  - The **Purkinje fibers** in the ventricles are **not** innervated by the vagus nerve.
  - When the higher pacemaker centers (SA or AV node) stop firing, the Purkinje fibers resume their own intrinsic rate (15–40 beats per minute).

Regulation of the Heartbeat

- Normally, the Purkinje fibers are suppressed by the faster rate of the SA node or AV node – a mechanism known as **overdrive suppression**.
- When the higher pacemakers cease activity, the Purkinje fibers "escape" this inhibition and begin generating their own impulses.
- This phenomenon is known as **ventricular escape**.

Time of Arrival of Cardiac Impulse

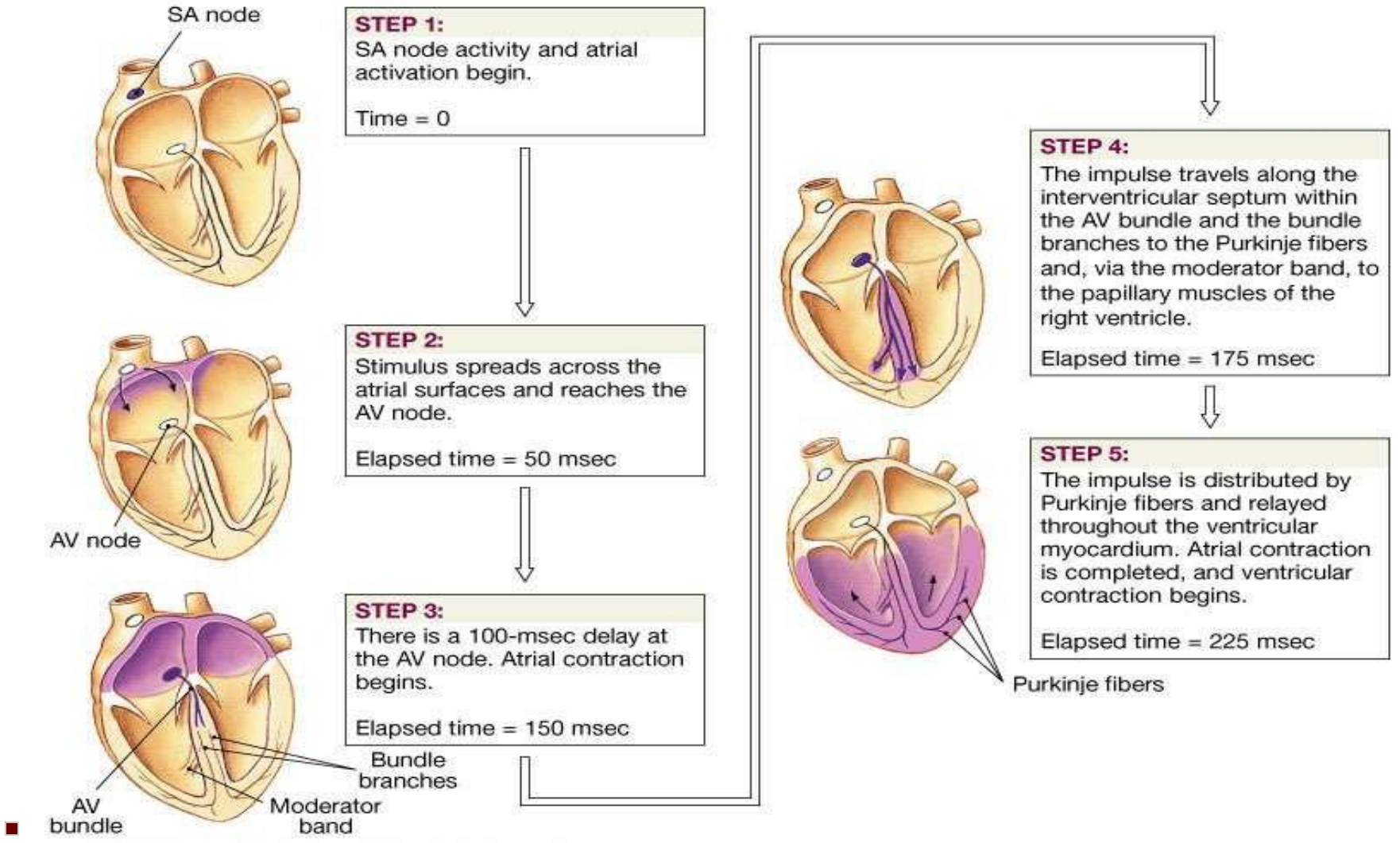
Don’t memorize the number



Main Arrival Times

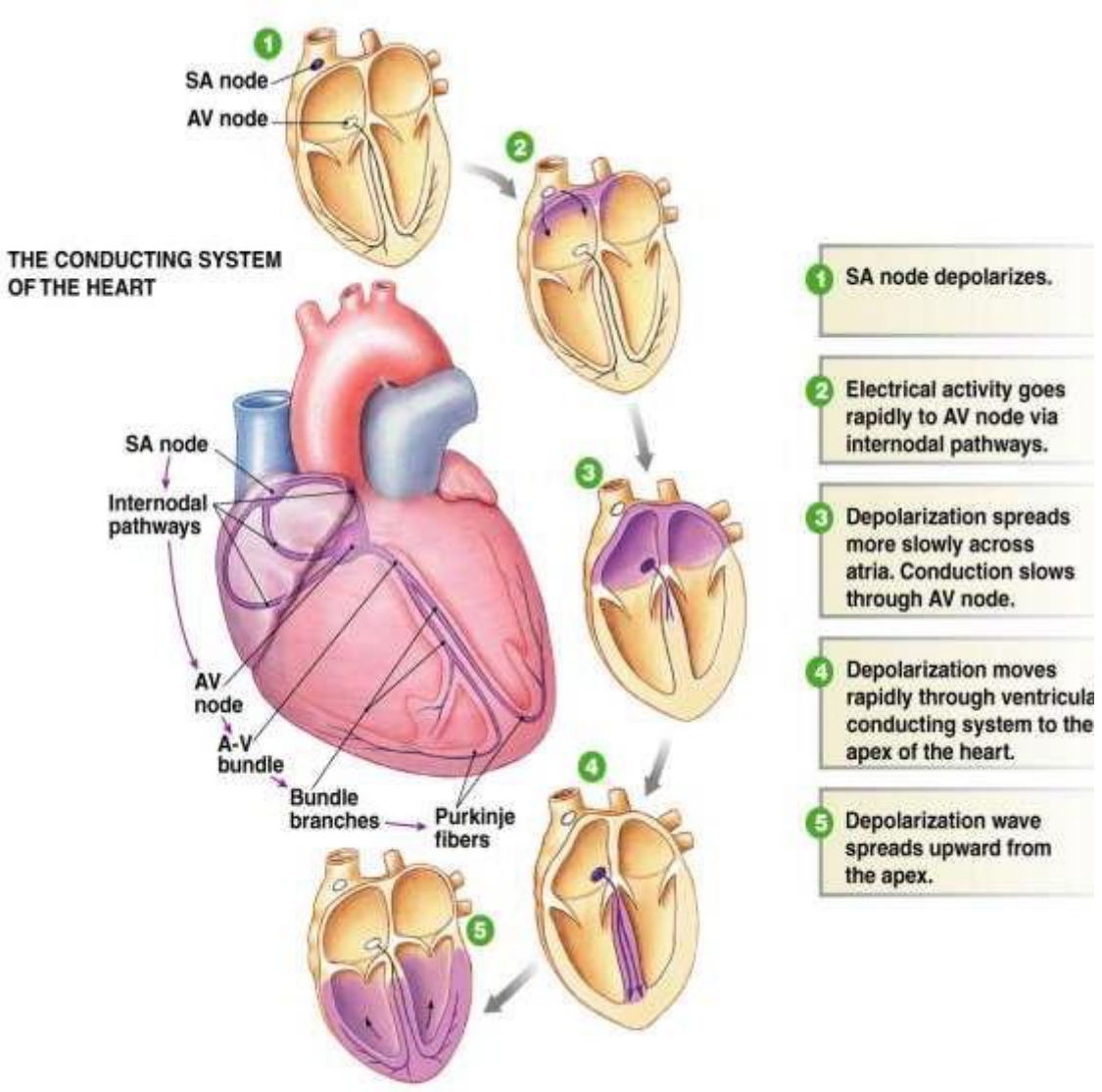
S-A Node	0.00 sec
A-V Node	0.03 sec
A-V Bundle	0.12 sec
Ventricular Septum	0.16 sec

Impulse Conduction through the Heart



The heart is approximately 15 cm long.
The Purkinje fibers are rectangular in shape, resembling normal contractile muscle cells in appearance.

Tissue	Conduction rate (m/s)
Atrial muscle	0.3
Atrial pathways	1
AV node	0.05
Bundle of His	1
Purkinje system	4
Ventricular muscle	0.3-0.5



Sinus Node is Cardiac Pacemaker

- Normal rates of discharge:
  - Sinus node – 70–80/min.
  - A-V node – 40–60/min.
  - Purkinje fibers – 15–40/min.
- Sinus node is pacemaker because of its faster discharge rate
- Intrinsic rate of subsequent parts is suppressed by "Overdrive Suppression"

Ectopic Pacemaker

- This is a portion of the heart with a more rapid discharge than the sinus node.
- Also occurs when transmission from sinus node to A-V node is blocked (A-V block).
- During sudden onset of A-V block, sinus node discharge does not get through, and next fastest area of discharge becomes pacemaker of heartbeat (Purkinje system).
- Delay in pickup of the heartbeat is the “Stokes-Adams” syndrome. New pacemaker is in A-V node or penetrating part of A-V bundle.

Parasympathetic Effects on Heart Rate

- Parasympathetic (vagal) nerves, which release acetylcholine at their endings, innervate S-A node and A-V junctional fibers proximal to A-V node.
- Causes hyperpolarization because of increased K^+ permeability in response to acetylcholine.
- This causes decreased transmission of impulses maybe temporarily stopping heart rate.
- Ventricular escape occurs.

Sympathetic Effects on Heart Rate

- Releases norepinephrine at sympathetic ending.
- Causes increased sinus node discharge (**Chronotropic effect**).
- Increases rate of conduction of impulse (**Dromotropic effect**).
- Increases force of contraction in atria and ventricles (**Inotropic effect**).

PHYSIOLOGY QUIZ

LECTURE 3

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Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			