Cardiac electric activity

Asma Al yahya

MBBS, MSc, PhD

Objectives

- Know The Components of The Conducting System of The Heart, The Conduction Velocities & Spread of Cardiac Impulse Through The Heart
- Understand Control of Excitation and Conduction in the Heart
- Identify The Action Potential of The Pace Maker and The Differences Between Pace Maker Potential & Action Potential of Myocardial Cells
- Describe The Control Of Heart Rhythmicity and Impulse Conduction By The Cardiac Nerves, What is Latent and Abnormal Pacemakers

Abbreviations

Actio potential: AP

Sinoatrial : SA

Atrioventricular: AV

Sodium ion: Na^+

Potassium ion: K+

Calcium ion:Ca+2

Extracellular fluid: ECF

Rhythmical Excitation of the Heart

The heart is endowed with a special system for:

(1) generating rhythmical electrical impulses to cause rhythmical contraction of heart muscle

(2) conducting impulses rapidly through heart.

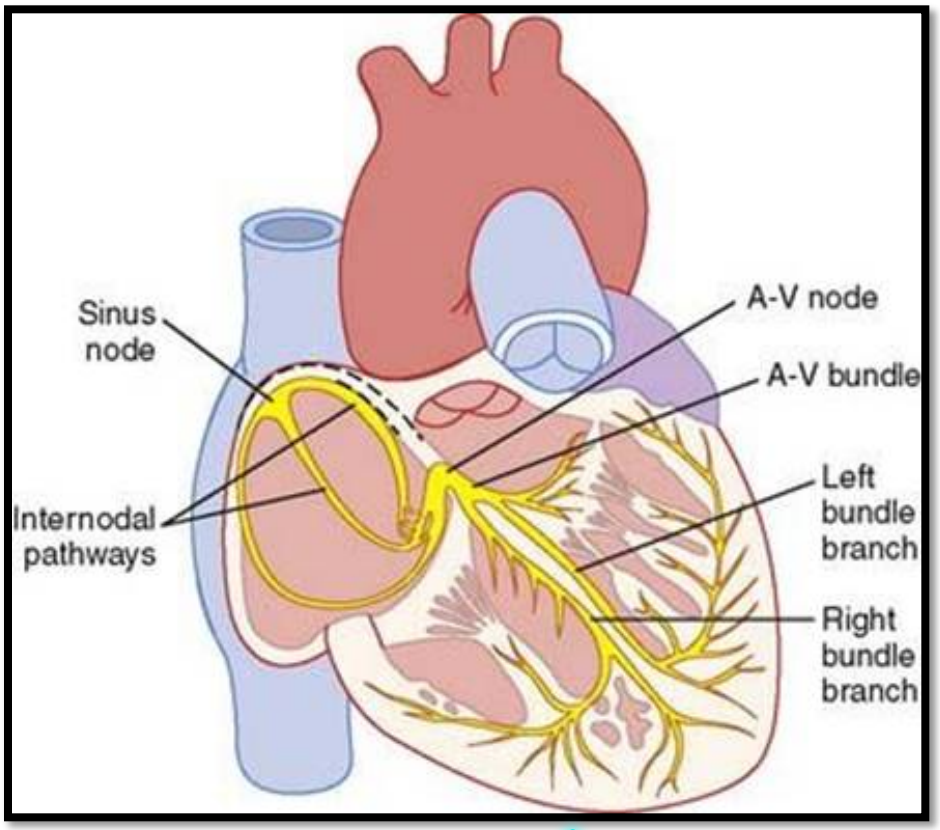
Rhythmical Excitation of the Heart..

When this system functions normally:

- Atria contract 1/6 of a sec ahead of ventricular contraction (allows filling of ventricles).
- All portions of ventricles contract at same time (essential for effective pressure generation in the ventricular chambers)

Specialized Excitatory and Conductive System of the Heart

- Sinus (SA) node
- Internodal pathways
- A-V node
- A-V bundle (Left and right)
- Purkinje fibers



Sequence of excitation

Sinus-Atrial Node (SA node)

↓

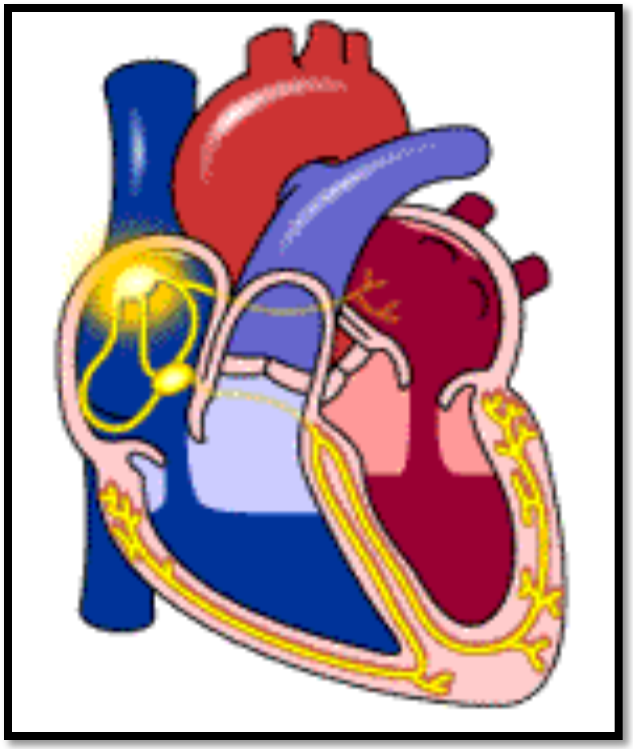
Atria

↓

Atrial-ventricular Node (AV node)

↓

Ventricles



Sinus (Sinoatrial) Node

❖SA node is a small, flat strip of specialized cardiac muscle. Located in superior Posterolateral wall of RA

❖It has few contractile fibers.

❖Its fibers connect directly with atrial muscle.

(*any AP that begins in the SA node spreads immediately into atrial muscle wall).*

❖SA node controls heart rate i.e SA node has the fastest rate of autorhythmicity

Mechanism of Sinus Nodal Rhythmicity

Cardiac muscle has 3 types of membrane ion channels that play important roles in causing voltage changes of AP:

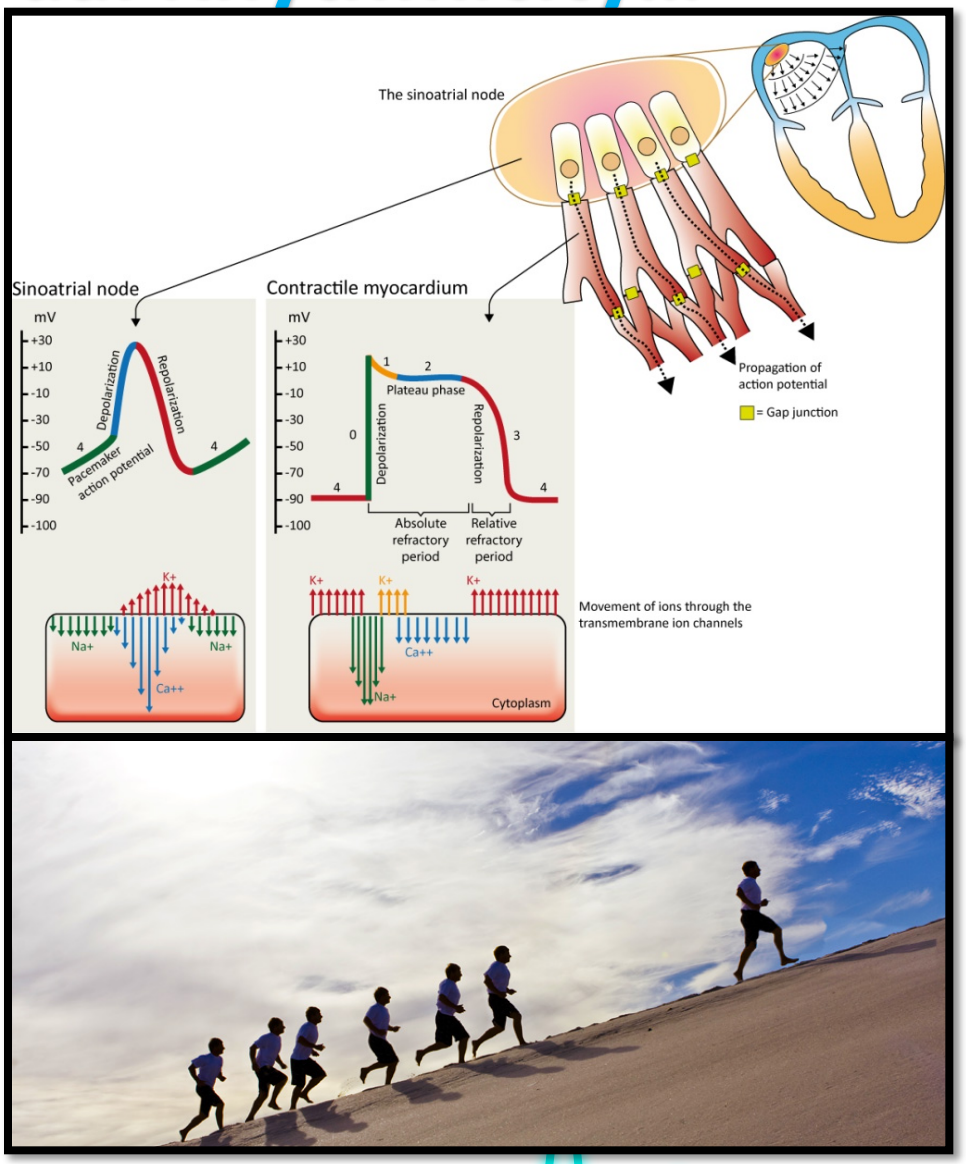
(1) *fast Na^+ channels,*
(2) *slow Na^+-Ca^{+2} channels,*
(3) *K^+ channels.*

Mechanism of Sinus Nodal Rhythmicity…

- Resting membrane potential of SA node has negativity of −55 to −60 mv

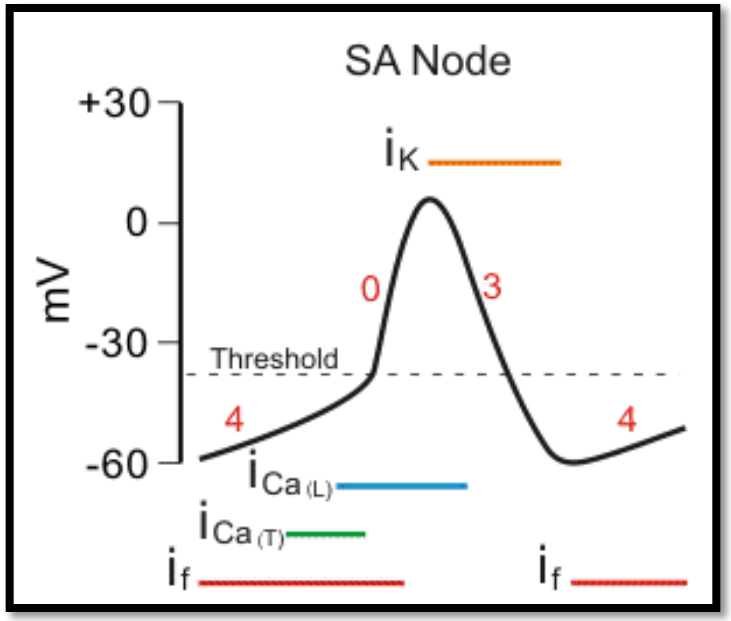
- Why?

  Because Its leaky



SA nodal APs are divided into 3 phases.

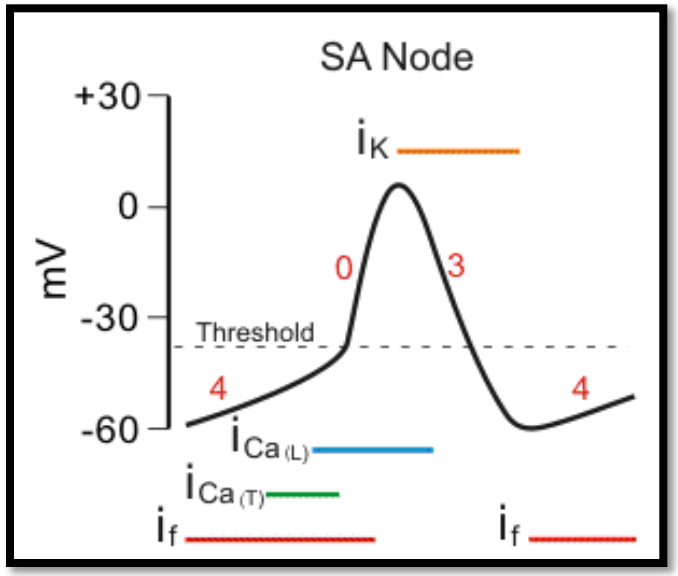
- **Phase 4:** spontaneous depolarization (pacemaker potential) triggers AP at threshold between -40 and -30 mV).
- **Phase 0:** depolarization
- **Phase 3:** repolarization.
- Once the cell is completely repolarized at about -60 mV, the cycle is spontaneously repeated.



Mechanism of pacemaker action potential

Phase 4:

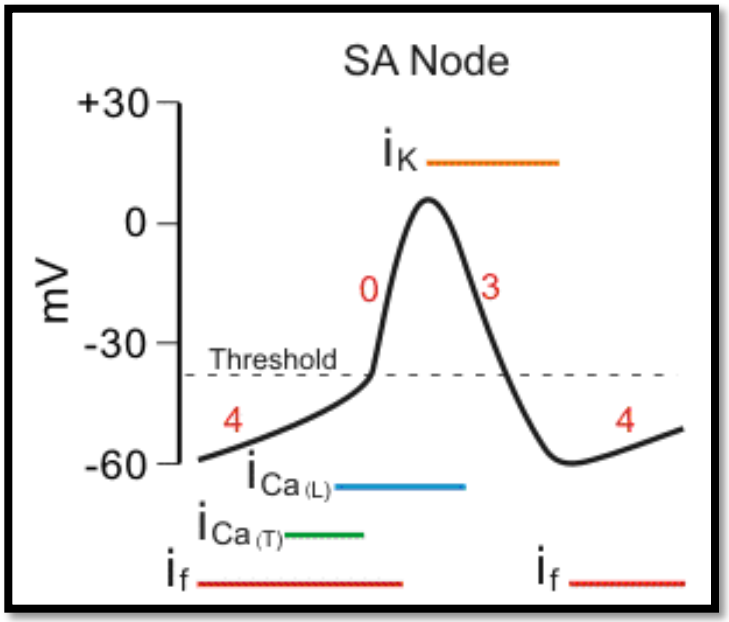
- At -60 mV, Na^+ **"funny" currents** enter (depolarizing) initiating **Phase 4**.
- At -50 mV, transient or **T-type Ca^{++} channel opens** . Ca^{++} enters depolarizing cell.



- At -40 mV, long-lasting, or **L-type Ca^{++} channels** open causes more Ca^{++} to enter and depolarize cell until an AP threshold is reached (between -40 and -30 mV).

Mechanism of pacemaker action potential

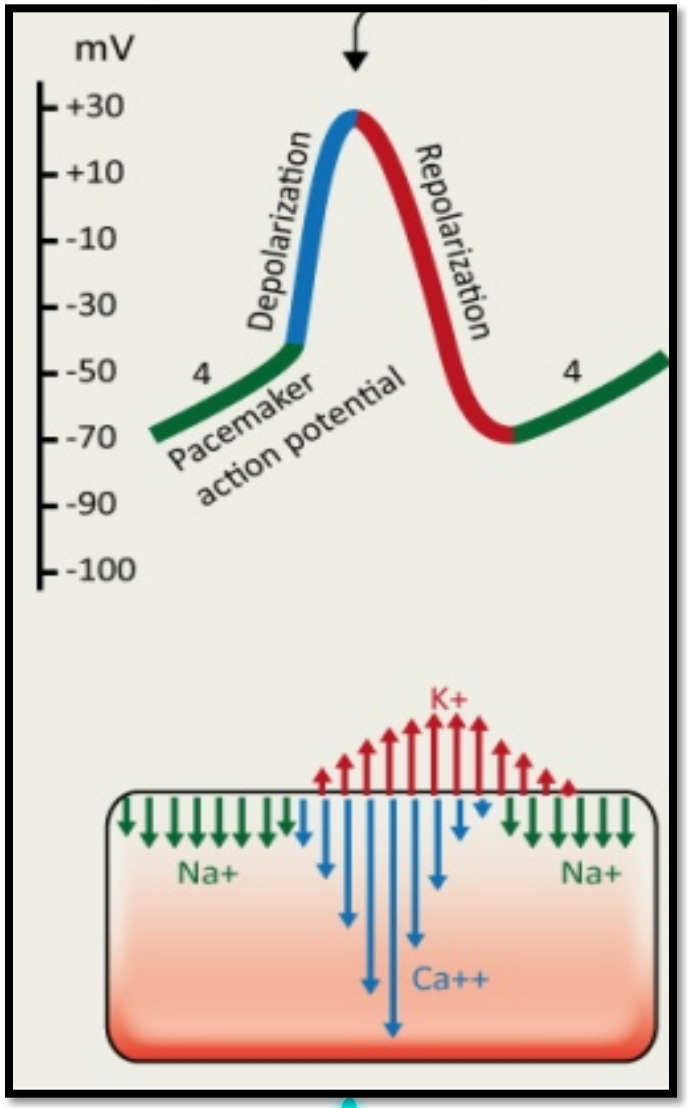
- **Phase 0**: depolarization is caused by increased Ca^{++} through the L-type Ca^{++} channels that began to open toward the end of Phase 4.



Phase 3: Repolarization occurs as K^+ channels open thereby increasing outward directed, hyperpolarizing K^+ currents.

Mechanism of Sinus Nodal Rhythmicity..

❖Return of AP to its negative state occurs slowly, rather than the abrupt return that occurs for ventricular fiber.



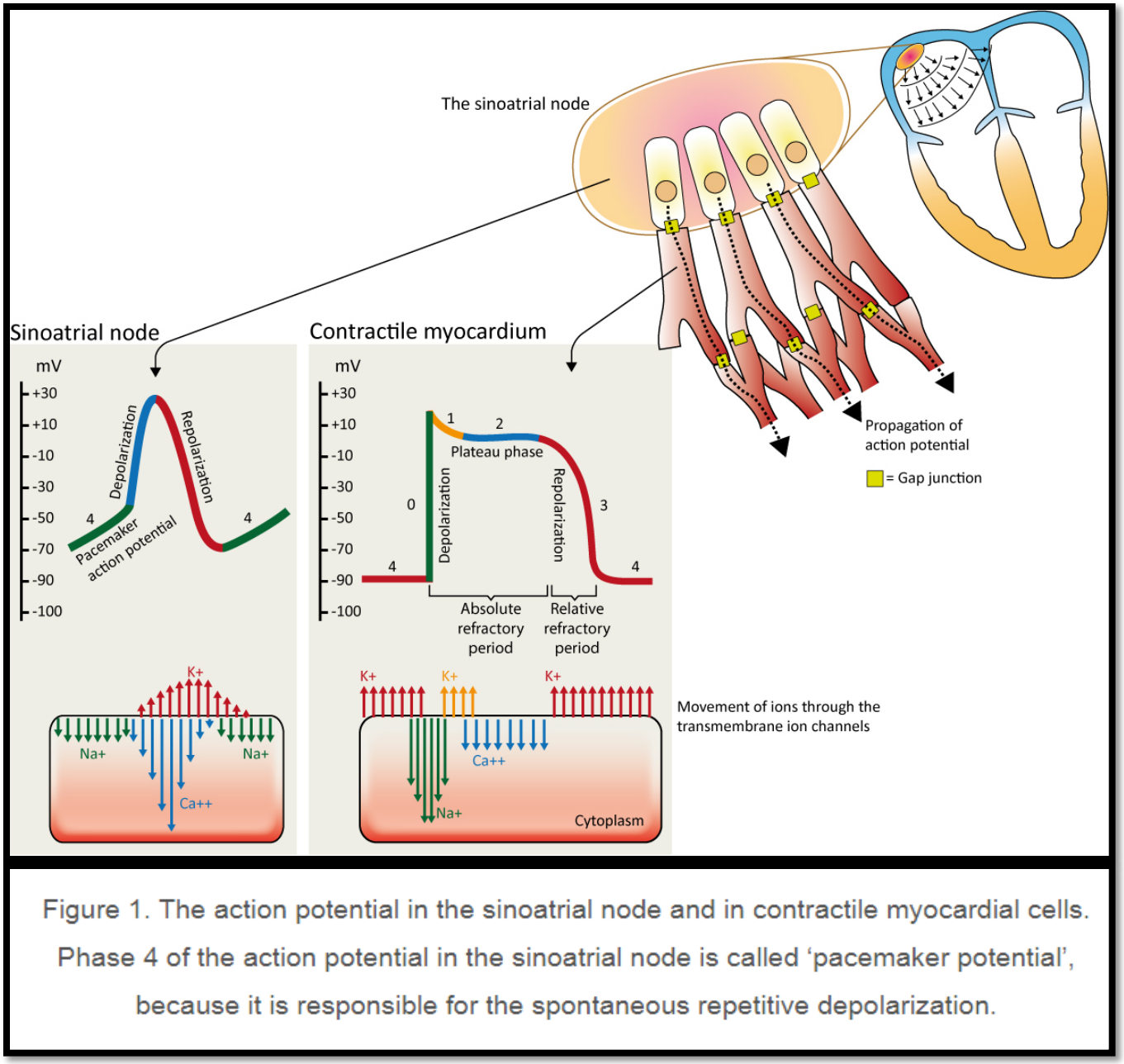
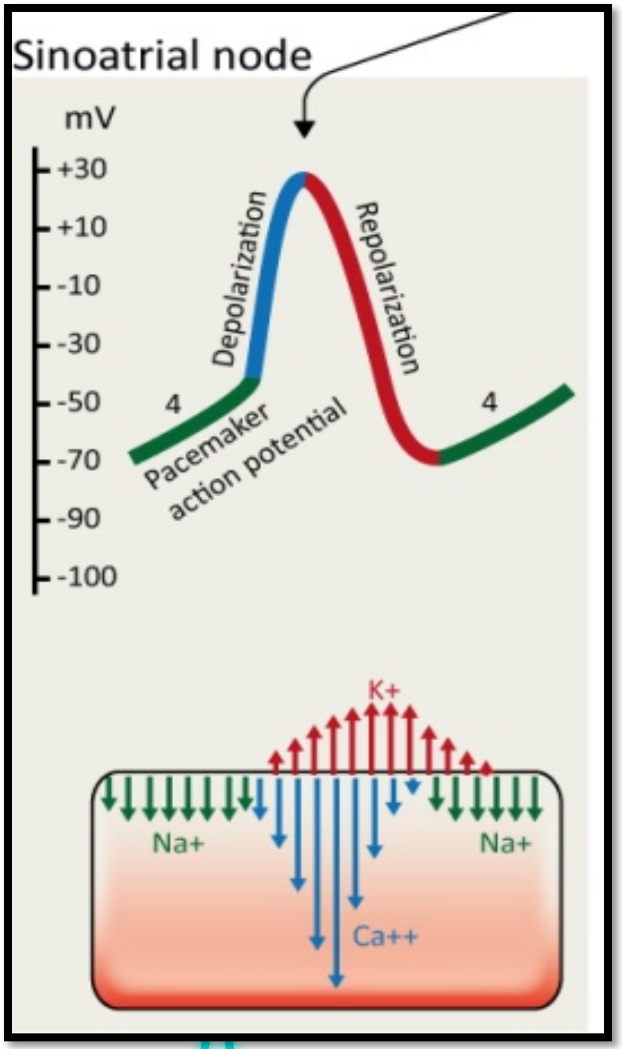


Figure 1. The action potential in the sinoatrial node and in contractile myocardial cells. Phase 4 of the action potential in the sinoatrial node is called 'pacemaker potential', because it is responsible for the spontaneous repetitive depolarization.

Why does leakiness to Na^{+} and Ca+2 ions not cause the SA node fibers to remain depolarized all the time?

❖**Answer:** two events occur during the course of AP to prevent this.

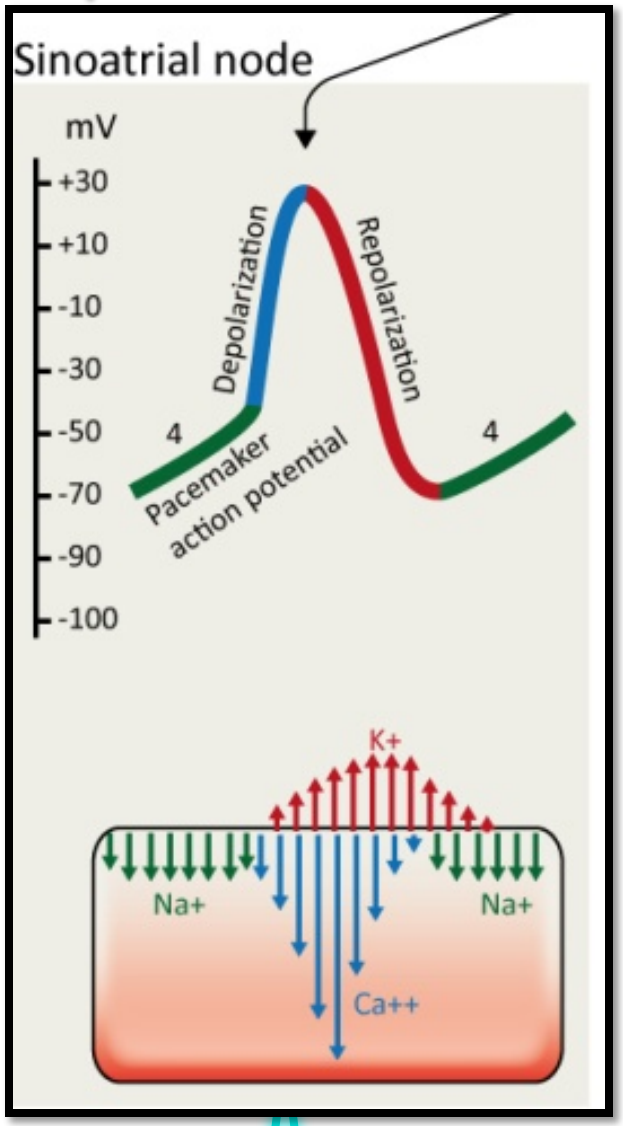
1. Na^{+}- Ca^{+2} channels become inactivated (i.e., they close) within about 100 to 150 millisec after opening,
2. At the same time, increased numbers of K+ channels open.
3. K+ channels remain open with resultant excess negativity inside the fiber causing *hyperpolarization*



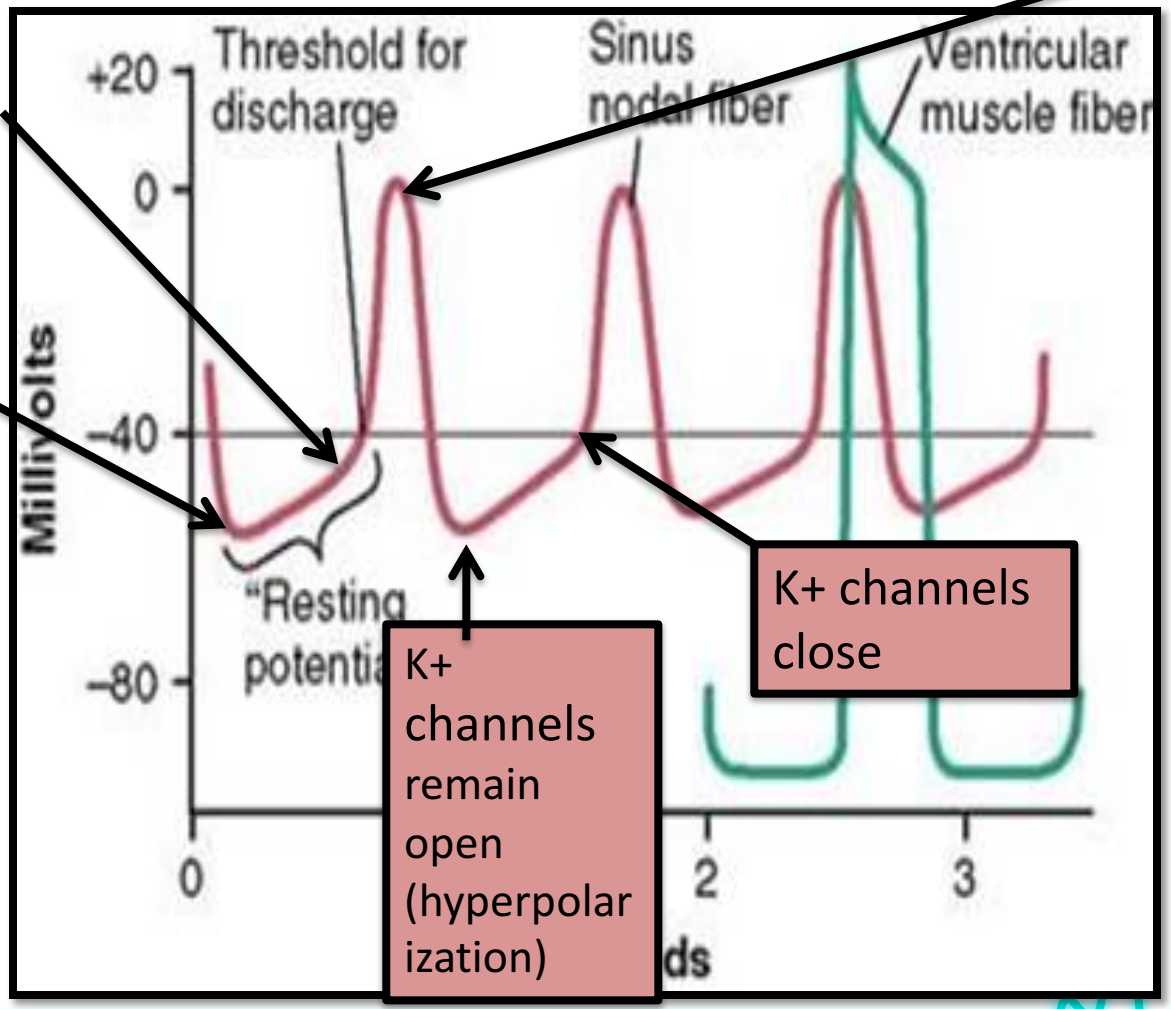
Why is this new state of hyperpolarization not maintained forever?

❖After AP is over, more K+ channels close.

❖Inward-leaking Na^{+} and Ca^{+2} overbalances outward flux of K+, and this causes “resting” potential to drift upward once more, reaching threshold level for discharge at a potential of about –40 mv.



Rhythmical discharge of a sinus nodal fiber compared with that of a ventricular muscle fiber



Entering Na^+ and Ca+2 reduce negativity.

Resting membrane potential of SA fiber is −55 to −60 mv

Closing of Na^+ -Ca+2 channels and opening of K+ channels

K+ channels close

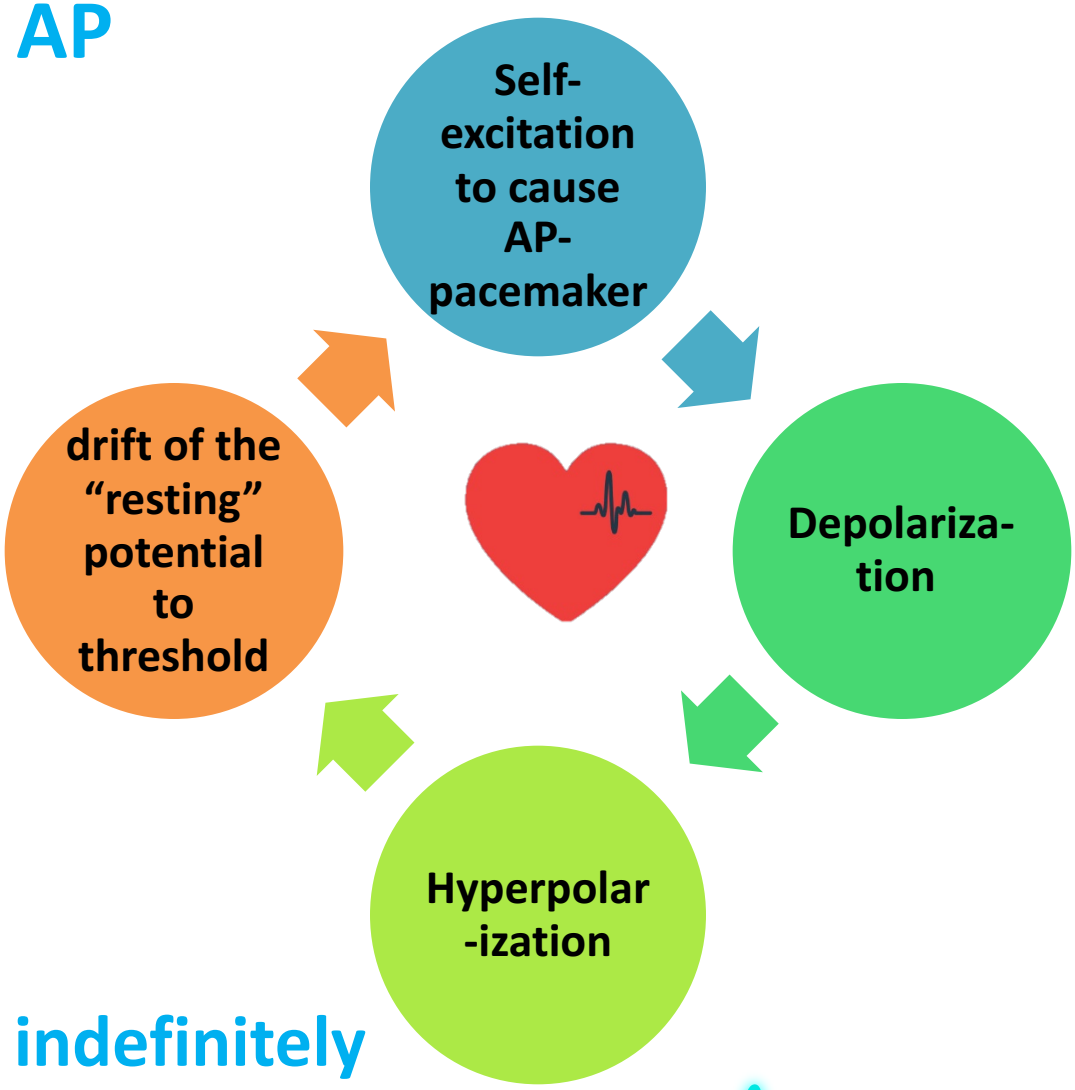
K+ channels remain open (hyperpolarization)

Resting membrane potential of ventricular muscle fiber is −85 to −90 mv

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

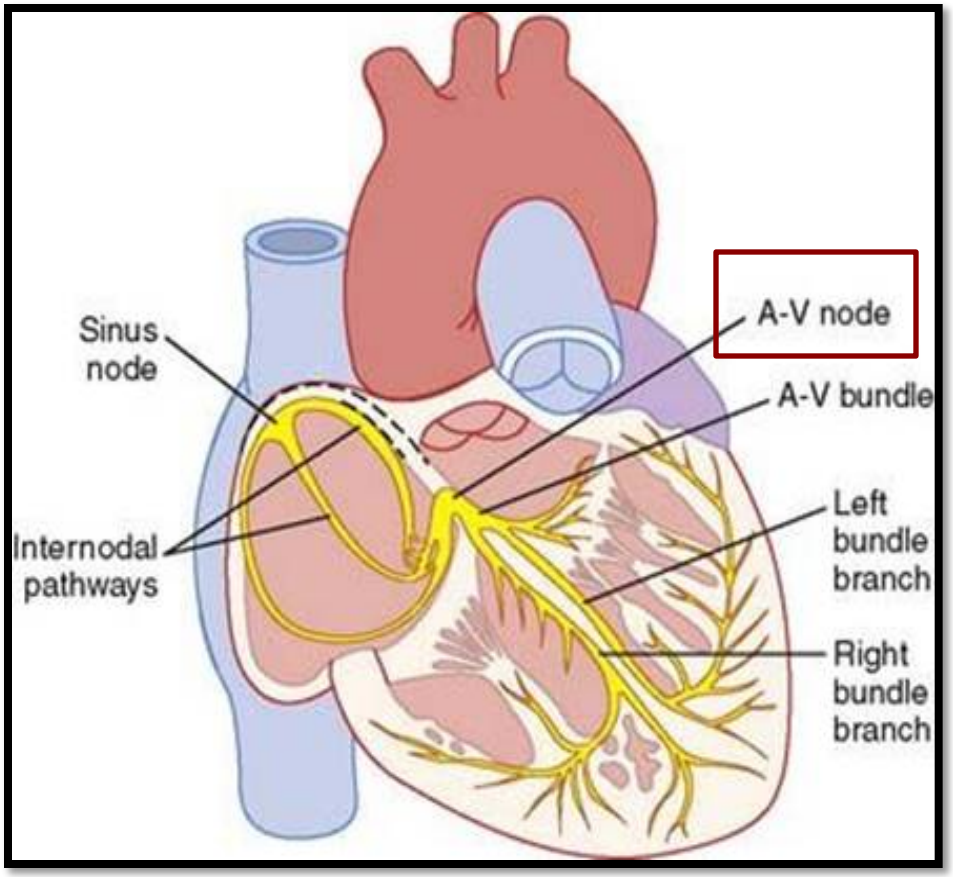
Summary SA node AP



This process continues indefinitely throughout a person's life.

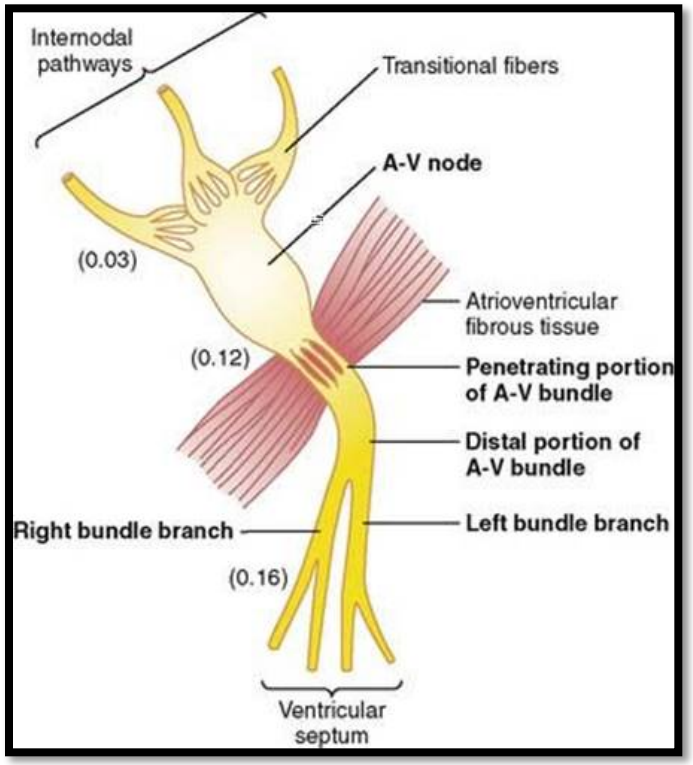
Atrioventricular (AV) Node

A-V node is located in the posterior wall of the right atrium immediately behind the tricuspid valve



Atrioventricular Node and Delay of Impulse Conduction from the Atria to the Ventricle

1. Impulse originates in SA node
2. Reaches AV node **0.03** sec after its origin
3. Delay of **0.09** sec in A-V node
4. Enters A-V bundle, passes into ventricles.
5. Delay of **0.04** sec occurs in A-V bundle
6. Total delay of **0.16 sec**



slow conduction in penetrating A-V bundle fibers is caused by diminished numbers of gap junctions between cells

Transmission in Ventricular Purkinje System

Purkinje fibers

- very large fibers
- Transmit APs at a velocity of **1.5 - 4.0 m/sec** (6 times that of ventricular muscle).
- Allows instant transmission of cardiac impulse throughout entire ventricular muscle.
- Rapid transmission is caused by a high level of permeability of gap junctions

One-Way Conduction Through A-V Bundle

- Is a characteristic of the A-V bundle
- Allows only forward conduction from atria to ventricles.
- Atrial muscle is separated from ventricular muscle by a continuous fibrous barrier except at A-V bundle, allowing only forward conduction through the A-V bundle itself.

Transmission of the Cardiac Impulse in the Ventricular Muscle

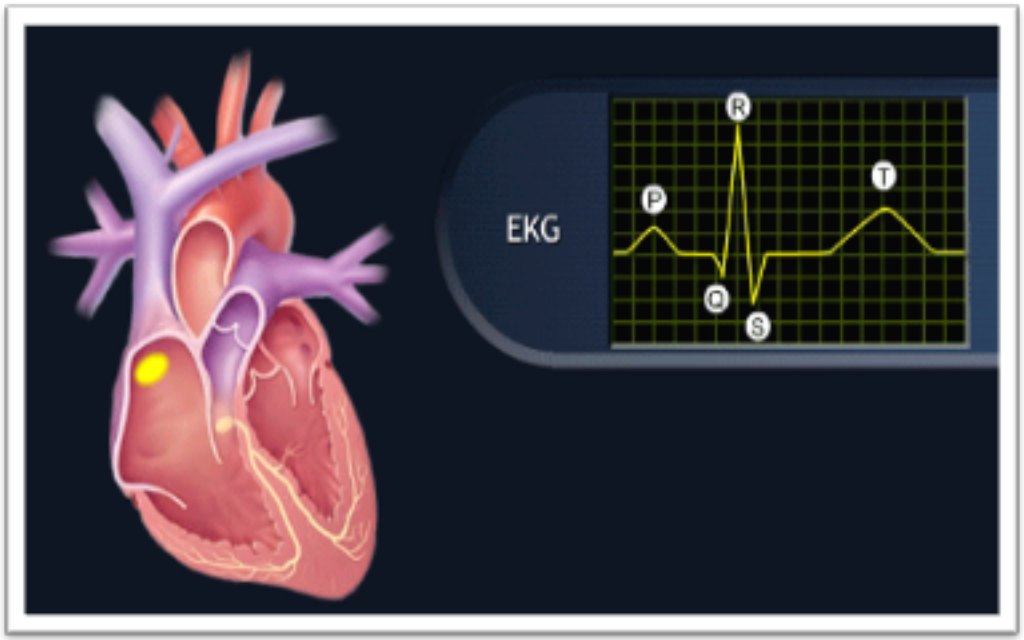
❖Once impulse reaches ends of Purkinje fibers, it is transmitted through ventricular muscle mass by ventricular muscle fibers themselves.

Conduction Velocity in different cardiac tissues

Conduction Velocity (m/sec)	
Atria	0.3-1
AV node	0.01–0.05
Bundle of His	1–2
Purkinje	2-4
Ventricle	1 m/

Conduction velocity is not the same in all myocardial tissues: It is slowest in the AV node and fastest in the Purkinje fibers

Control of Excitation and Conduction in the Heart, Sinus Node as the Pacemaker of the Heart



Why does SA node rather than A-V node or Purkinje fibers control heart's rhythmicity?

Because its rate of rhythmical discharge is faster than any other part of heart.

Effect of Sympathetic Stimulation on Cardiac Rhythm and Conduction

- It increases rate of sinus nodal discharge.
- It increases rate of conduction, as well as the level of excitability in all portions of the heart.

Mechanism of the Sympathetic Effect

Stimulation of the sympathetic nerves releases the hormone norepinephrine

Norepinephrine stimulates *beta-1 adrenergic receptors*

Increases permeability to Na^+ and Ca+2 causing a more **positive resting potential** and increased excitability

increases rate of sinus nodal discharge

Effect of parasympathetic Stimulation on Cardiac Rhythm and Conduction

- It decreases rate of sinus nodal discharge.
- It reduces rate of conduction

Strong stimulation of vagi stops rhythmical excitation by SA node or blocks transmission of cardiac impulse from atria into ventricles.

Mechanism of the Vagal Effects

Parasympathetic Stimulation releases *acetylcholine*

Acetylcholine increases permeability to K+ allowing rapid leakage of K+ out of conductive fibers.

Causes increased negativity inside fibers (hyperpolarization) making tissue less excitable

Decreases rate of rhythm of the sinus node and A-V junctional fibers.

Summary of Effects of Autonomic Nervous System on the Conduction velocity

	Sympathetic	Parasympathetic
Conduction velocity	↑	↓

Effect of sympathetic and parasympathetic stimulation on the SA node action potential.

Sympathetic stimulation increases the rate of phase 4 depolarization and increases the frequency of action potentials

Parasympathetic stimulation decreases the rate of phase 4 depolarization and hyperpolarizes

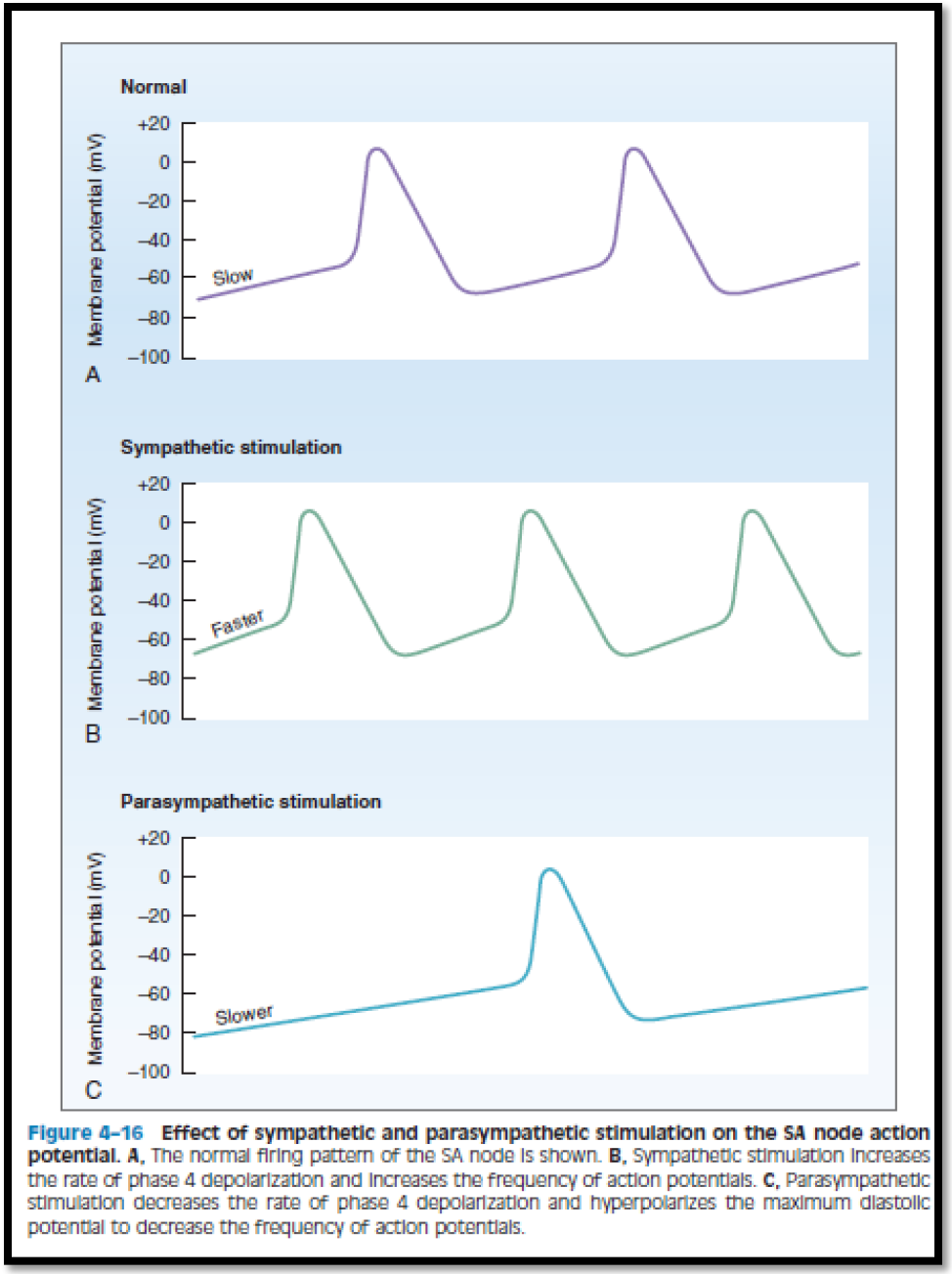


Figure 4–16 Effect of sympathetic and parasympathetic stimulation on the SA node action potential. A, The normal firing pattern of the SA node is shown. **B,** Sympathetic stimulation increases the rate of phase 4 depolarization and increases the frequency of action potentials. **C,** Parasympathetic stimulation decreases the rate of phase 4 depolarization and hyperpolarizes the maximum diastolic potential to decrease the frequency of action potentials.

Latent Pacemakers

Table 4–3 Firing Rate of Sinoatrial Node and Latent Pacemakers in the Heart

Location	Intrinsic Firing Rate (impulses/min)
Sinoatrial node	70–80
Atrioventricular node	40–60
Bundle of His	40
Purkinje fibers	15–20

The lower you go in the heart the lower the rate

If your heart is in rhythm, so is your life