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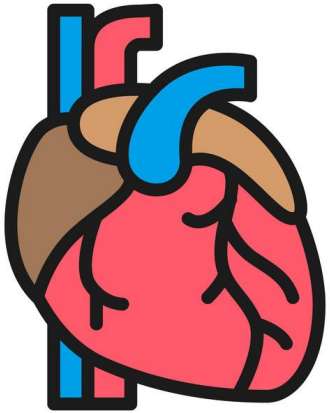

Cardiac electrical activity

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Editing File

Objectives

- Know the components of the conducting system of the 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 Pacemaker and The Differences between Pacemaker Potential & Action Potential of Myocardial Cells
- Describe The Control of Heart Rhythmicity and impulse Conduction by the cardiac Nerves, what is latent and abnormal Pacemaker

Before studying we recommend watching the following videos

Video

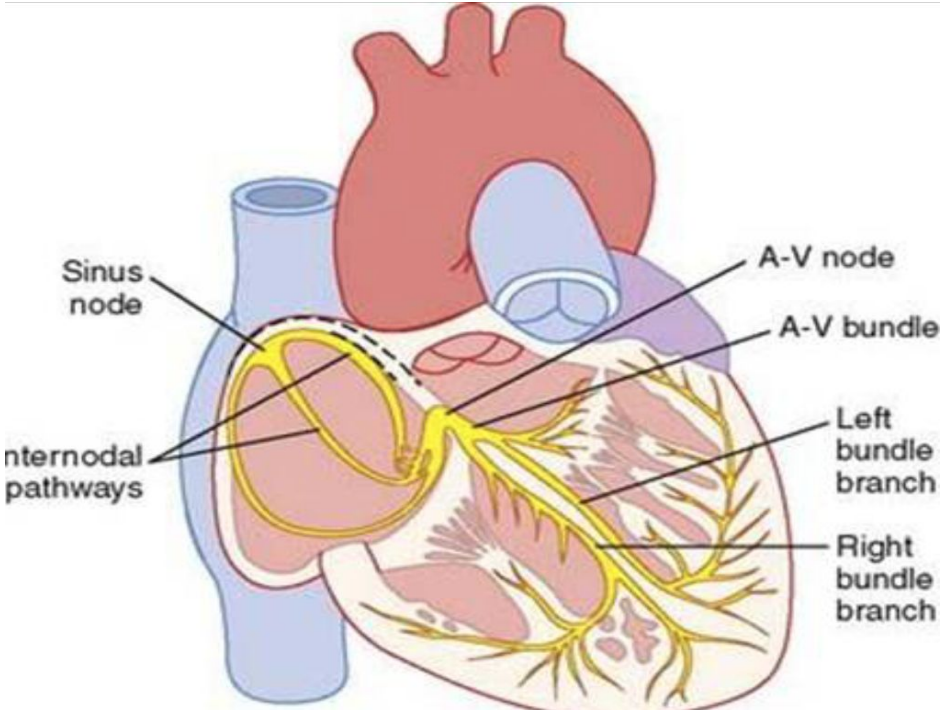
Action potential in pacemaker

Video

Rhythmicity of heart

Abbreviations

- Action potential: AP
- Sinoatrial: SA
- Atrioventricular: AV
- Sodium ion:Na+
- Potassium ion: K+
- Calcium ion:2
- Extracellular fluid: ECF



Rhythmical excitation of the heart

Female slides

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.

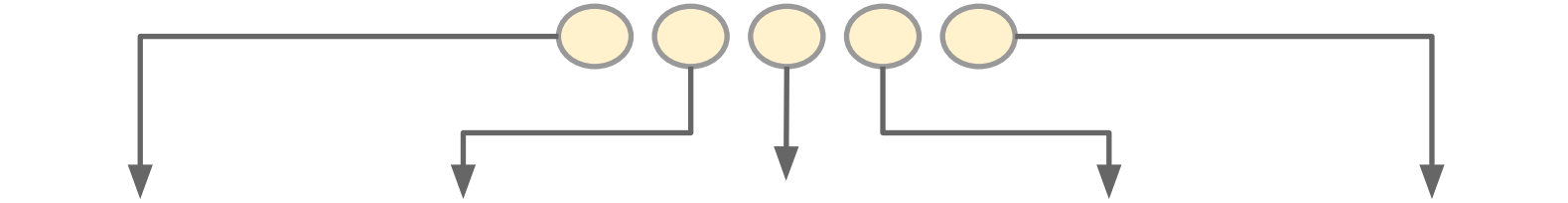
When the system functions normally:

All portions of ventricles contract at same time (essential for effective pressure generation in the ventricular chambers)

Atria contract 1/6 of a sec ahead of ventricular contraction (allows filling of ventricles).

Specialized Excitatory and Conductive System of the Heart

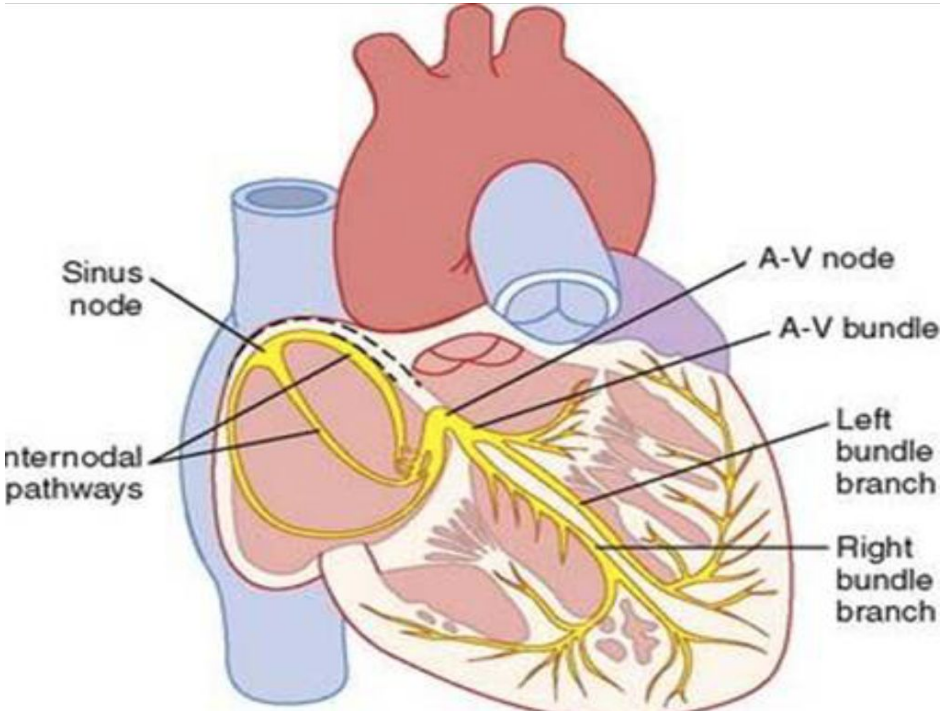
Components of conductive system



sequence of excitation

Sequence of excitation

Male slides



- Sinoatrial node
- Atrial syncytium
- Junctional fibers
- Atrioventricular node
- A-V bundle
- Bundle branches
- Purkinjie fibers
- Ventricular syncytium

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
- Responsible for generating the electrical impulses that bring about the mechanical activity i.e contraction of the heart
- The velocity of conduction in most atrial muscle is about 0.3m/sec.
- the impulse after leaving SA node takes 0.03 sec to reach the AV node.

Internodal pathway

Male slide

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

2.Anterior, middle and posterior conducting bundles

1.Ordinary Atrial muscle fibers

Internodal pathway

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.

In the specialized internodal pathways the conduction velocity may reach upto 1m/sec.

Mechanism of sinus nodal Rhythmicity

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

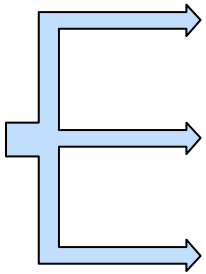
(1) *fast Na+channels*

(2) *slow Na+-Ca+ channels*

(3) *K+ channels*

Resting membrane potential of SA node has negativity of –55 to –60 mv
Why?Because It's 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

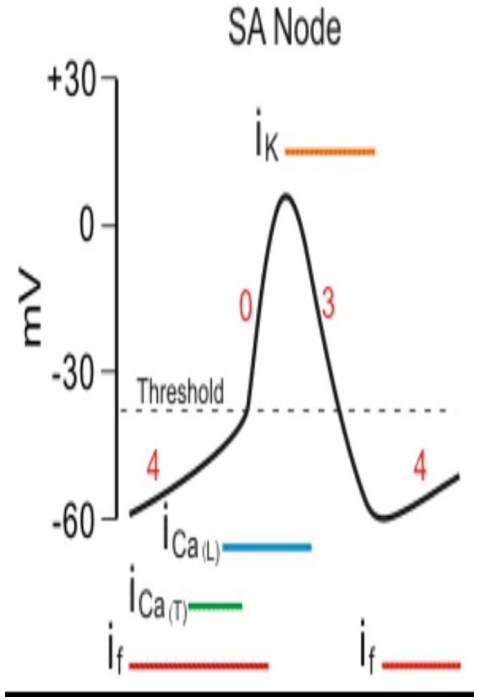
Female slide

1. **Phase 4:** At -60 mV, Na+ **"funny" currents** enter (depolarizing) initiating **Phase 4**.

   At-50mV,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).

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

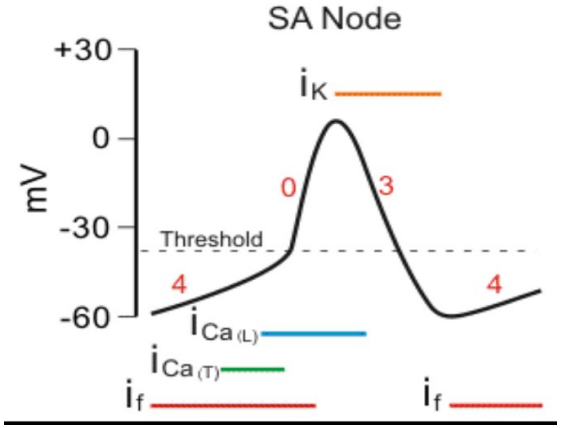


Mechanism of pacemaker action potential

Female slide

3

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



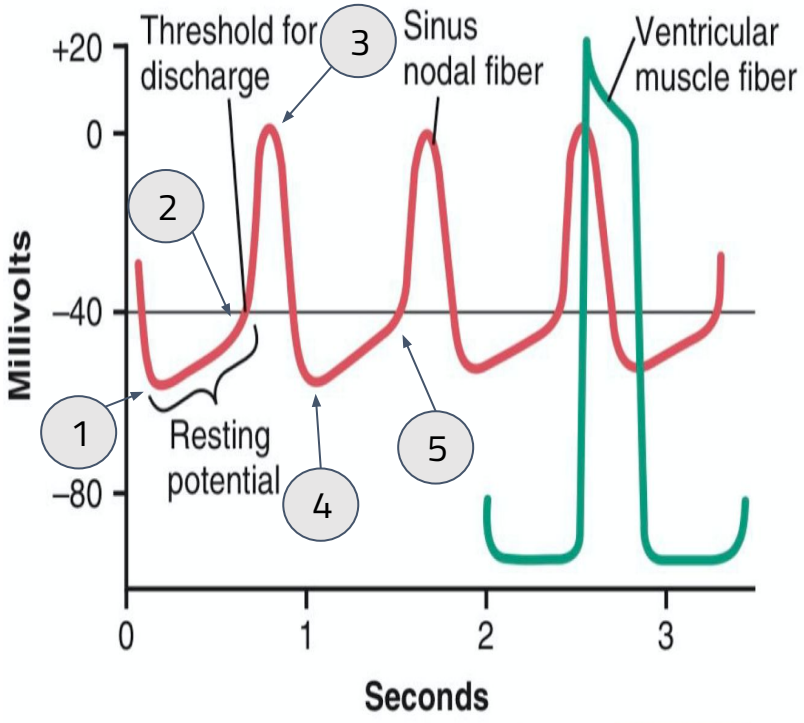
-Return of AP to its negative state occurs slowly, rather than the abrupt return that occurs for ventricular fiber.

-A hyperpolarized state is necessary for the pacemaker chanel to become activated.

Without the membrane voltage becoming very negative at the end of phase 3, pacemaker channels remain inactivated , which suppresses pacemaker currents and decreases the slope of phase 4.

- After repolarization the cycle repeat itself again this process happen in the autorhythmic cells then The electrical impulses goes from the autorhythmic cells to the contractile cells follow up what will happen in the contractile cells in the next slide .

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



1.Resting membrane potential of SA fiber is –55 to –60 mv

2.Entering Na+ and Ca+2 reduce negativity.

3.Closing of Na+ -Ca+2 channels and opening of K+ channels

4.K+ channels remain open (hyperpolar ization)

5.K+ channels close

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

Extra explanation for the whole process:

-the heart has two types if cells (contractile cells - autorhythmic cells)

1.**(autorhythmic cells) goes through 3 phases:**

-Phase4(pacemaker):(opening of Na funny channel at -60mv , T type Ca channel at -50mv , L type Ca channel between -40, -30 mv)

- phase0(Depolarization):increased Ca through the L type Ca channel.

- phase3(hyperpolarization): k channel opens.

Then electrical impulses goes to the contractile cells

The contractile cells goes through 5 phase:

Phase 0(depolarization):fast Na channels open membrane potential from -90 to 20mv.

Phase1(initial repolarization):fast Na channels close, k channels open

Phase2(plateau):CA channels open K channels close .

phase3(rapid repolarization):CA channels close K channels open

Phase4(resting membrane potential):goes back -90 mv.

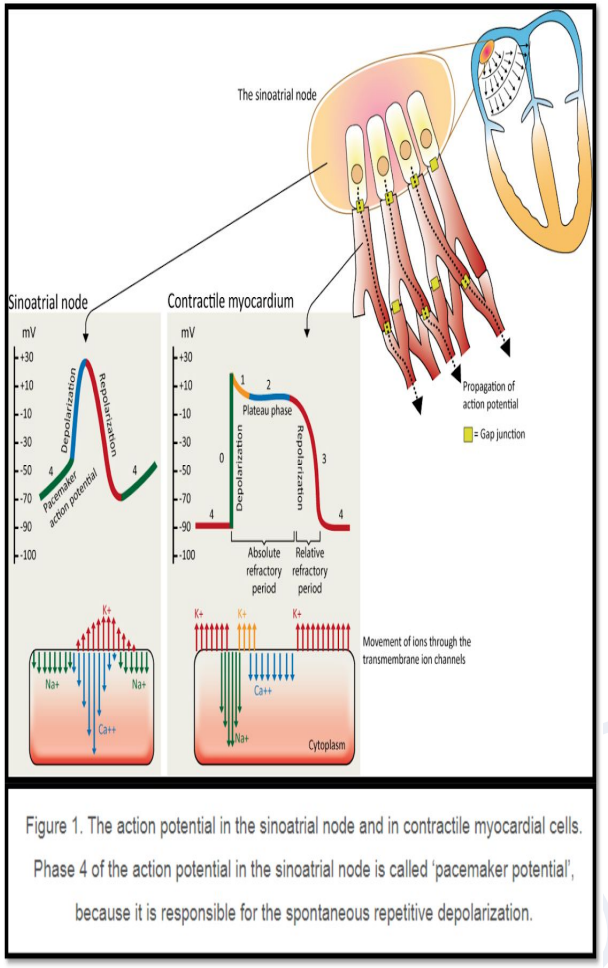
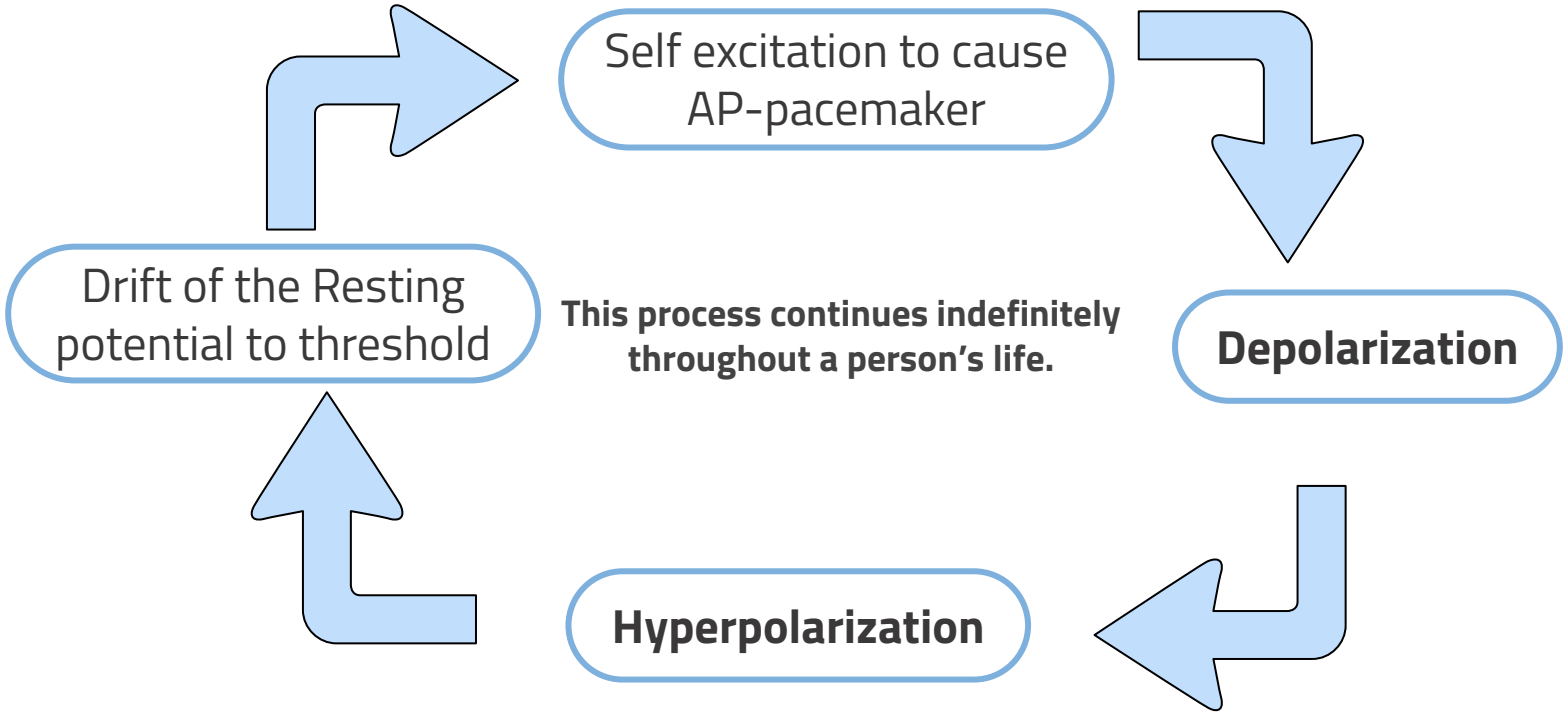


Figure 1. The action potential in the sinoatrial node and in contractile myocardial cells. Phase 4 of the action potential in the sinoatrial node is called 'pacemaker potential', because it is responsible for the spontaneous repetitive depolarization.

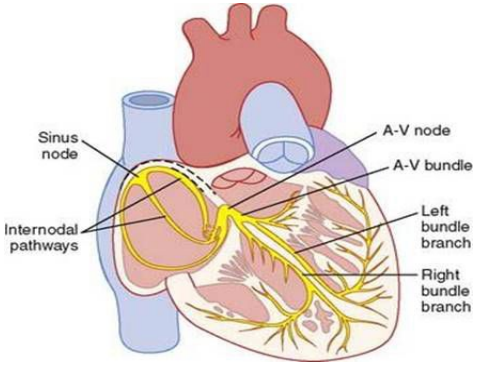
SA node Action potential (AP) Summary

Female Slide



Atrioventricular (AV) Node

Located in the posterior wall of the right atrium immediately behind the tricuspid valve



Significance of AV Nodal Delay:

- The cardiac impulse does not travel from the atria the 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

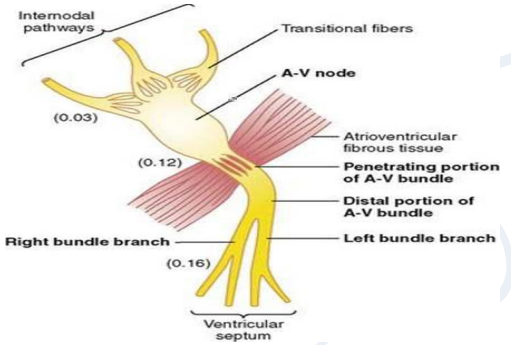
Atrioventricular Node & Delay of Impulse Conduction from the Atria to the Ventricle

Female Slide

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

Total Delay of 0.16 sec

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



Transmission in Ventricular Purkinje system

1. Very **large** fibers
2. **Transmit** APs at a velocity of **1.5 - 4.0 m/sec** (6 times that of ventricular muscle)
3. **Allows** instant transmission of cardiac impulse throughout entire ventricular muscle
4. **Rapid** transmission is caused by a high level of permeability of gap junctions at the intercalated discs between the successive cells of Purkinje fibers
5. 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
And Conduction of Impulse

Male Slide

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

2. 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.

3. Time delay occurs as impulses pass through AV node:
   - Slow conduction of 0.03 - 0.05 m/sec

4. 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 A-V Bundle

Is a **characteristic of the A-V bundle is inability of action potentials to travel backward from the ventricles to the atria**

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\Which acts as an insulator to prevent the passage of cardiac impulse between the atrial and ventricular muscle

This prevents re-entry of cardiac impulse by this route from the ventricles to the atria

Conduction Velocity in different cardiac tissues

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

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 is not the same in all myocardial tissues:

It is slowest in the AV node and fastest in the Purkinje fibers

Conduction Velocity in different cardiac tissues

Extra slides

Cardiac tissue conduction velocity

Fastest

Purkinje system

↓

Atrial muscle

↓

Ventricular muscle

↓

AV node

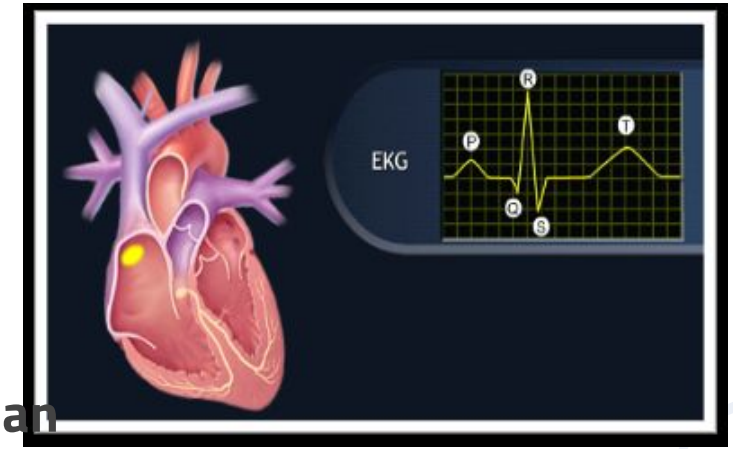
Slowest

Mnemonic: "**P**ark **At** **Vent**ure **Av**enue"

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

Female slides

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

Female Slide

- 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 Effects

Stimulation of the sympathetic nerves release the hormone Norepinephrine

Norepinephrine stimulates β_1 adrenergic receptor

Increase permeability to Na and Ca causing a more positive resting potential and increased excitability

Increase rate of sinus nodal discharge

Effect of parasympathetic Stimulation on Cardiac Rhythm and Conduction

Female Slide

- 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 ventricle

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.

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

Female Slide

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

Latent Pacemakers :

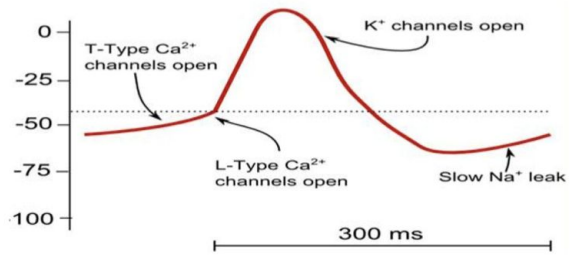
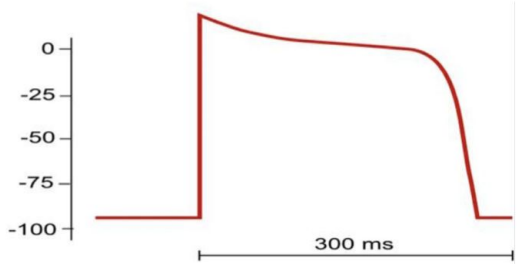
The Lower you go in the Heart
The Lower the rate

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

Pacemaker Vs. Myocardial Cell Action Potential

Thanks to Team439!

Pacemaker Action Potential	Ventricular Muscle Action Potential.
	
Does not need a stimulus	Needs a stimulus (needs electrical impulses)
RMP is -60 mv. it's less negative than ventricular RMP, because it's cell membrane is naturally leaky to NA+ and Ca++ ions.	RMP is -90 mv.
Threshold is about -40 mv.	Threshold is about -70 mv
Max. depolarization is +10 mv.	Max. depolarization is +20 mv
Is of smaller magnitude	Is of larger magnitude.
Has pre-potential stage	Has no pre-potential stage
Depolarization is gradual.	Depolarization is rapid.
Depolarization is due to Ca++	Depolarization is due to Na+.
It has spike, no plateau.	It has plateau, no spike

Comparison of Action potential in Cardiac and skeletal muscle			
	Skeletal muscle	Contractile myocardium	Autorhythmic myocardium
Membrane potential	stable at -70 mV	stable at -90 mV	Unstable peacemaker potential; usually starts at -60 mV
Events leading to threshold potential	Net Na+ entry through ACh- operated channels	Depolarization enters via gap junction	Net Na+ entry through funny channels, reinforced by Ca+ entry
Rising phase of AP	Na+ entry	Na+ entry	Ca+ entry
Repolarization phase	Rapid; caused by K+ efflux	Extended plateau caused by Ca+ 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+ resotres potential to resting state	None; resting potential is -90 mV, the equilibrium potential for k+	Normally; when repolarization hits -60 mV th funny channels open again. ACh can hyperpolarize the cell
Duration of AP	Short: 1-2 mSec	Extended: 200+ mSec	Variable; generally 150+ mSec
Refractory period	Generally brief	Long because resetting of Na+ channels gates delayed until end of AP	None

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Special thanks to Arwa Almobeirek for designing the theme!

Click here for a summary done by the team