Cardiac Electrical Activity

Physiology Team .439

Black: in male / female slides
Red : important
Pink: in female slides only
Blue: in male slides only
Green: Dr' notes
Gray: extra information

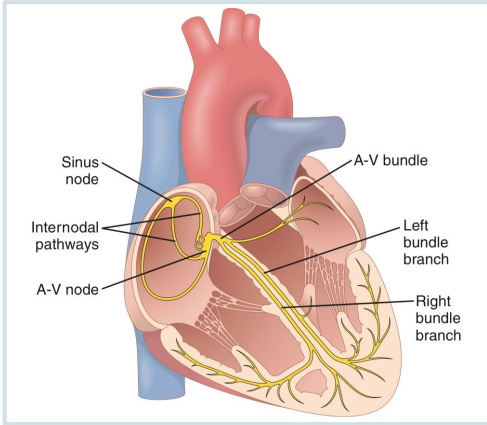
Editing File

Objectives

- Know the components of the conducting system of the Heart, the conduction velocities & spread of the cardiac impulse through the Heart
- Understand the 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

Cardiac Electrical Activity

The heart has a special system for generating rhythmical electrical impulses to cause rhythmical contraction of the heart muscle.



Automaticity of the heart:

the heart is capable of:

1- Generating rhythmical electrical impulses

2- Conducting impulses rapidly through the heart in a specialized conducting system formed of specialized muscle fibers (Not nerve fibers).

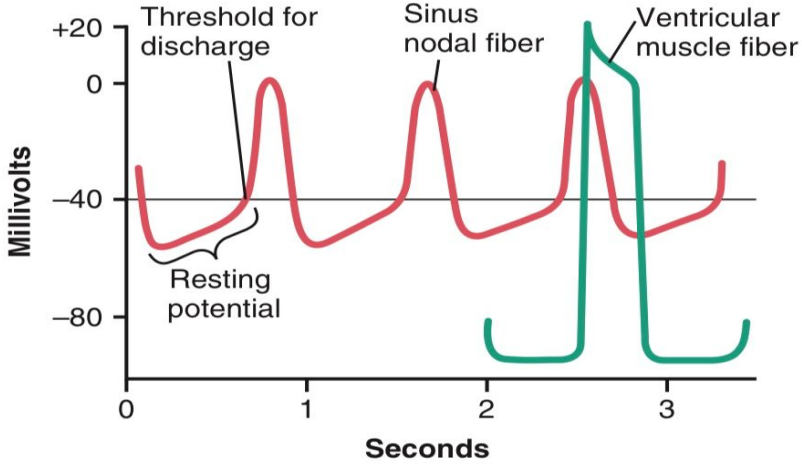
The atria contract about one sixth of a second a head of ventricular contraction, why?

To allow filling of the ventricles before they pump the blood into the circulation.

In the previous lecture, we talked about the action potential of contractile heart muscles.
In this lecture, we'll be talking about the action potential of conductive/nodal cells. Nodal cells send the electrical stimulus to the contractile cells. Then, contractile cells transmit the signal to one another (signals are transmitted through gap junctions). Ultimately, this causes the heart to contraction.

Unlike skeletal muscles which have to be stimulated by nervous system, the heart generates its own electrical stimulation. That is why it can beat even if it's taken of the body. The CNS can make the heart go faster or slower.

EXTRA.



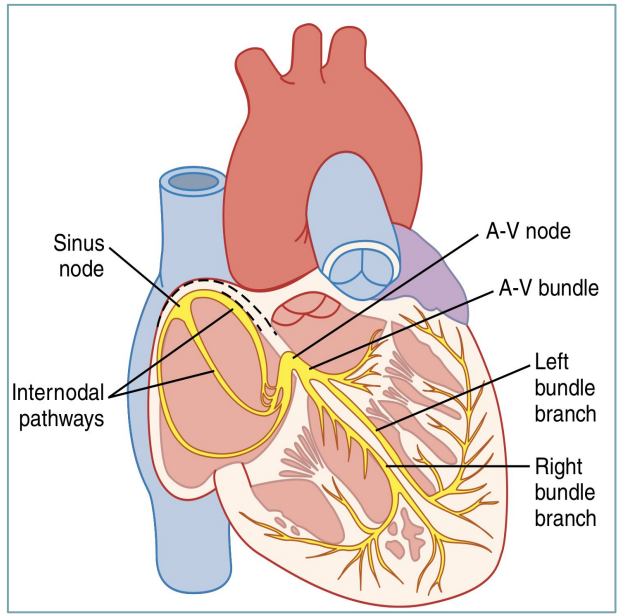
Components of Heart Conduction system

- SA Node
- Inter-Nodal Pathway
- AV Node
- AV Bundle (Bundle of His)
- Right & Left Bundle Branch
- Purkinje Fibers

Sequence of excitation:

This sequence is one-way only.

SA node
↓
Atrial syncytium
↓
Junctional Fibers
↓
AV Node
↓
AV Bundle (Bundle of His)
↓
Right & Left Bundle Branches
↓
Purkinje Fibers
↓
Ventricular Syncytium



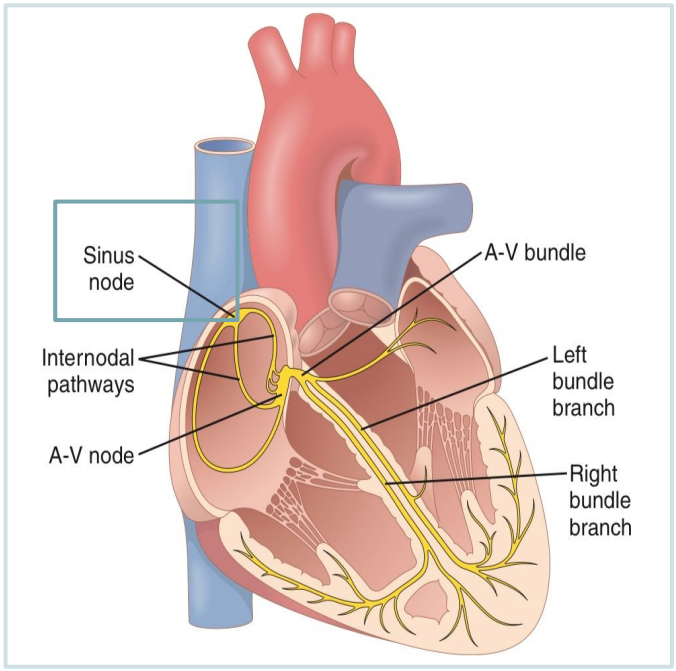
1- Sinoatrial Node (SA Node)

- Small, flattened, specialized cardiac cell located in the superior posterio-lateral wall of the right atrium.
- It is made of modified cardiac muscles
- Its fibers are continuous with atrial fibers.
- Velocity of conduction between its fibers is 0.05 m/s.
- Its membrane potential is unstable, so it is responsible for generating the electrical impulses (action potentials) that bring about the mechanical activity i.e **contraction of the heart**

Known as the normal Pacemaker of the heart, Why?

Because it has the **fastest autorhythmicity** (pacemaker= the place where the first impulse is generated).

(Autorhythmicity= The ability of a cell to self propagate an impulse)



- from Physiology team 437:
S.A node determine the heart rate.

2- Internodal pathway

Action potential can travel from SA node to spread throughout the atrial muscle through two routes:

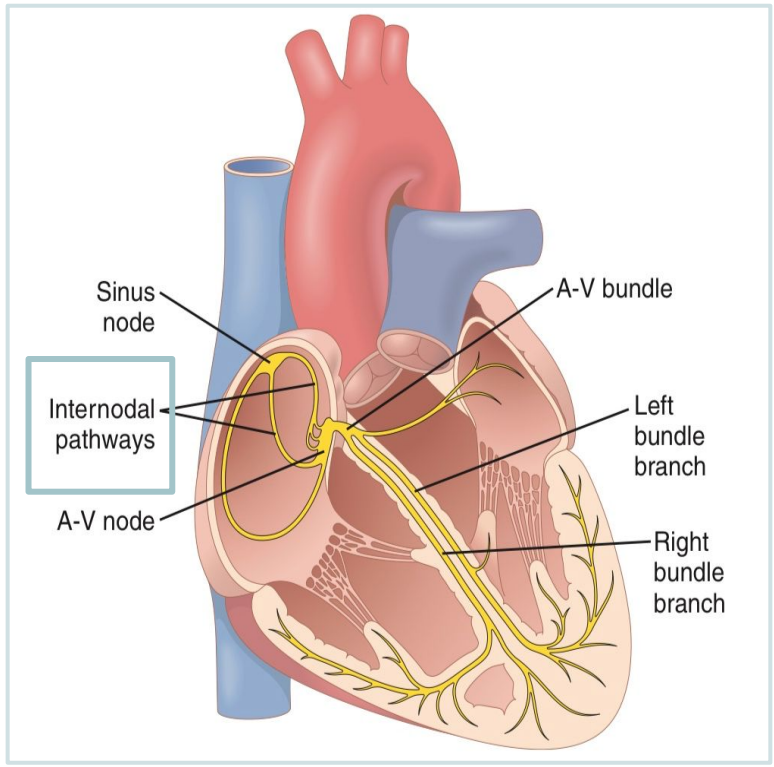
1- Ordinary atrial muscle fibers (conduction velocity is 0.3 m/s).

2- Internodal pathway (conduction velocity is 1m/s) **(faster)**.

These inter nodal pathways conduct the impulses at a faster rate than atrial muscle fibers, because of specialized conduction fibers.

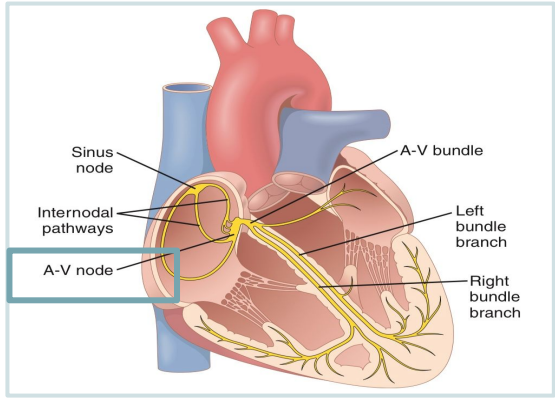
2- Inter-Nodal Pathway:

- **Anterior** internodal bundle of Bachman
- **Middle internodal** bundle of Wenkebach
- **Posterior** internodal bundle of Thoral



3- AV Node

- The A-V node is located in the posterior wall of the right atrium immediately behind the tricuspid valve.
- The impulse after leaving SA node takes 0.03 sec to reach the AV node
- Impulses pass through A-V node at a velocity of 0.01 m/s **(slower)**.
- A-V node receives impulses from S-A node and transmits them to ventricles through A-V bundle.
- Delay in the conduction of impulses occurs at A-V node (0.13 sec)



What is the significance of A-V Nodal delay (0.13 sec)? **(important)**

Cardiac impulse does not travel from the atria to the ventricles too rapidly. The significance of this is:

1- To allow time for the atria to **empty the blood** into the ventricles before ventricular contraction begin and so gives time for **ventricular filling** with blood and **increases the efficiency** of the pumping action of the heart.

2- To **protect** ventricles from pathological high atrial rhythm.

3- (from 437): allows the coronary blood to supply the heart.

What causes A-V Nodal delay?

The **diminished number of gap junctions** between the successive cells makes them slower.

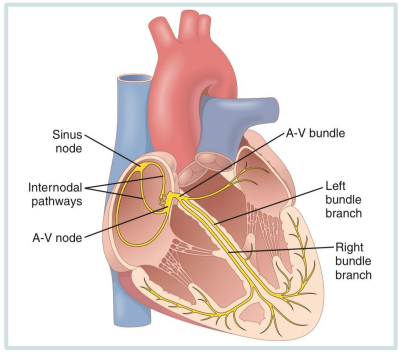
4- AV Bundle (Bundle of His)

The Right & Left Bundle Branches

- A-V node transmits impulses to ventricles through A-V bundle.
- A-V bundle conducts impulses to A-V bundle branch at velocity of 1 m/s.
- A-V bundle splits into two branches (right and left bundle branches) present on the respective sides of the ventricular septum and spread toward the apex of the heart, then reflect on ventricular wall.

The one-way conduction of the AV bundle (Bundle of His):

- A special characteristic of the A-V bundle is its **inability** of action potentials **to travel backward** from the ventricles to the atria. (one way)
- The atrial muscle is separated from the ventricular muscle (except at the A-V bundle) by a continuous fibrous barrier (Atrioventricular fibrous tissue) which acts as an insulator to prevent re-entry of cardiac impulse by this route from the ventricles to the atria. (this barrier is a reason for the one way conduction)
- APs (Action potential) spread from S-A node through internodal pathway quickly at velocity of 0.8-1.0 m/sec.
- Impulses pass through A-V node at a velocity of (0.03-0.05 m/sec) (0.01 m/sec). Time delay occurs (0.13 sec).
  (There were different values among boys & girls slides)
- 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.



5-Purkinje System

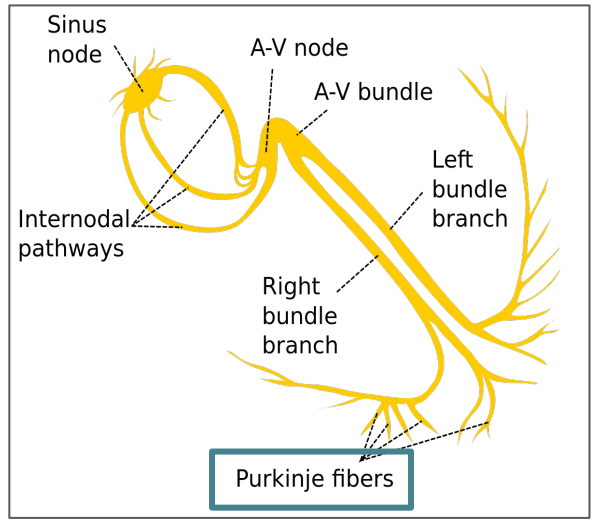
- After the right and left bundle branches reflect on ventricular wall, they divide into small branches **(Purkinje fibers).**
- Purkinje fibers penetrate and become continuous with **ventricular** cardiac muscle fibers.
- From the time the cardiac impulse enters the bundle branches until it reaches the terminations of Purkinje fibers , the total time averages only **0.03 sec**.
- Purkinje fibers are very large fibers. They can Transmit action potentials at a **very high velocity** (1.5-4.0 m/s) (fastest velocity of conduction)

Why do they have high conduction velocity?

It is because they have a very high permeability of **gap junctions** at the intercalated discs between the successive cells of Purkinje fibers → ions are transmitted easily from one cell to the next → enhance the velocity of transmission.

What is the benefit (Significance) of high conduction velocity?

To ensure that different parts of ventricles are excited almost simultaneously, so, all ventricular muscle contract at almost the same time; this greatly increases the efficiency of heart as a pump **(synchronous contraction)**

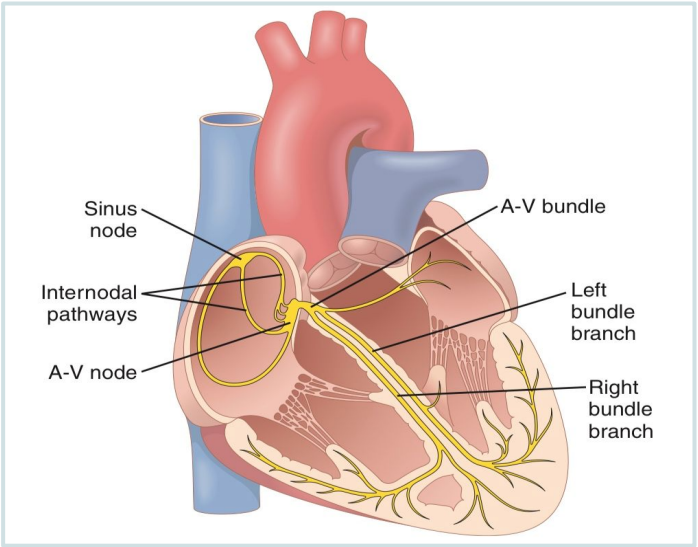


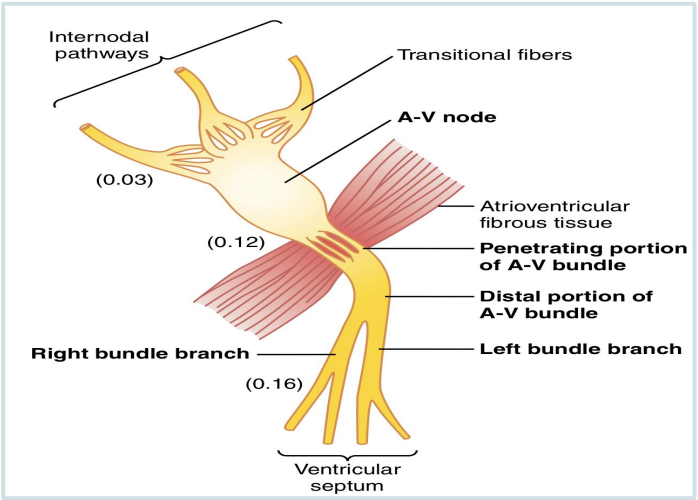
Conduction velocities & Spread of the cardiac impulse through the Heart

Sequence of Excitation & Conduction Velocities through the heart's electrical system:

	Sequence of excitation	Conduction velocity
0.03 sec	Sinoatrial (S-A) node	**0.05 m/sec**
	Atrial muscles	**0.3 m/sec -0.5 m/sec.**
	Internodal pathway	**1.00 m/sec** (2nd fastest)
	Atrioventricular (A-V) node	**0.01 m/sec (slowest)**
0.13 sec delay	A-V Bundle (Bundle of His)	**1.00 m/sec** (2nd fastest)
	Right & Left Bundle Branches	**1.00 m/sec (2nd fastest)**
0.03 sec	**Purkinje Fibers**	**4.00 m/sec (fastest)**
	Ventricular muscles	**0.3-0.5 m/sec**

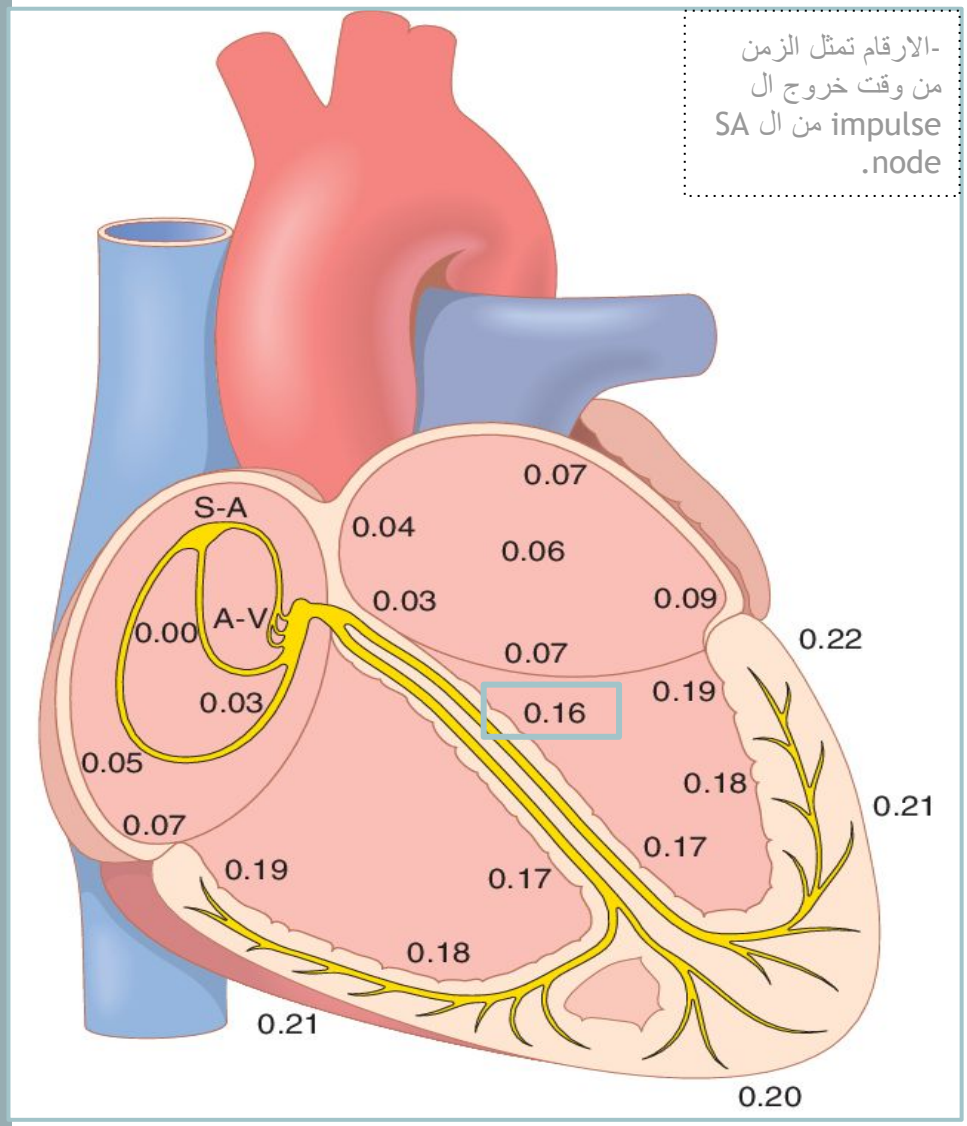
Ventricular contraction begins **0.1–0.2** sec. after contraction of the atria.





Organization of the A-V node. The numbers represent the interval of time from the origin of the impulse in the S-A node.

Extra Explanation



-سرعة اطلاق ال electrical impulse داخل ال SA node الى اطرافها تكون 0.05m/s.

-ثم من اطراف ال SA node تدخل ال impulse الى internodal pathway بسرعة 1m/s حتى تصل الى ال AV node وهذا يكون خلال 0.03s.

-اول ما توصل الى ال AV node تصير سرعتها بطيئة ما بين 0.01 الى 0.05 m/s بسبب ال diminished number of gap junctions at the intercalated disks، وهذا الانخفاض في السرعة يؤدي الى تأخير في الزمن بمقدار 0.09s داخل ال AV node نفسها.

-ثم ال impulse تخرج من ال AV node الى His bundle بسرعة 1m/s, لكن داخل His bundle يصير فيه تأخير آخر في الزمن بمقدار 0.04s, ومع نهاية His bundle تكون ال impulse وصلت لبداية ال ventricular muscle.

-الحين نجي نحسب الوقت من لحظة خروج ال impulse من AV node الى ما وصلت الى ال ventricles:

تأخير صار داخل ال AV node نفسها مقداره (0.09s) + تأخير صار داخل His bundle مقداره (0.04s) = يطلع 0.13s

ثم 0.13s + الوقت ما بين SA و AV مقداره (0.03s) = يطلع كله في النهاية (0.16s)

-طبعا هذي ال 0.16s هي الوقت حتى تصل ال impulse الى بداية ال ventricular muscle لكن تحتاج ال impulse ما يصل الى 0.22s حتى تتوزع بشكل كامل على طول ال purkinje fibers (بسرعة تصل الى 1.5 الى 4 m/s وهي الاسرع) وبالتالي كل ال ventricular muscle.

Control of Excitation and Conduction in the Heart

Female's slides ONLY

The S-A node is the normal Pacemaker of the Heart.

It has pacemaker prepotential (autorhythmic tissue).

It initiates the excitation wave, drives the entire heart and makes the heart rate 105 impulse/min, but it is normally **inhibited** by the right vagus nerve to be 70 impulse/min (vagal tone).
The parasympathetic system (vagus nerve) has the upper hand on the heart.
Its rate of rhythmical discharge is faster than that of any other part of the heart, so it drives rest of the heart.

A-V node, His bundle, and Purkinje fibers have also intrinsic automaticity & ability to set a pace

They are called "latent Pacemakers".
Latent Pacemakers are normally suppressed & function only if the **S-A node is damaged**, **its impulse is blocked**, or i**f the rate of firing of the latent pacemakers increases**.

If S-A node is damaged, A-V node becomes the new pacemaker and heart follows it but at a slower rate (**50-60 impulse/min**) (A-V nodal rhythm).
So for example when we see on report written "he is A-V nodal rhythm" that's mean, his S-A node is damaged

If S-A node or A-V node are damaged, His bundle & Purkinji fibers become the pacemaker with a rhythm of (**28-40 impulse/min**) (idioventricular rhythm). (originating from ventricles)

Rhythmicity is highest in S-A node then A-V node then His bundle & Purkinje fiber:

SA Node:
70 impulse/min

>

AV Node "A-V nodal rhythm":
50-60 impulse/ min

>

Purkinje fibers "Idioventricular rhythm":
28-40 impulses/min

Abnormal (Ectopic) Pacemakers

Female's slides ONLY

- **Ectopic pacemaker:** a pacemaker elsewhere than the SA node. In some cases, Purkinje fibers can become over excited i.e. ectopic focus (purkinje fibers start to generate impulses at a high rate which can reach 140 impulse/min) and cause premature ventricular contraction.
- It can occurs upon excess caffeine, lack of sleep, anxiety, stress or some organic conditions.

Causes of Ectopic Pacemakers

Any other part of the heart develops a rhythmical discharge rate that is more rapid than that of the SA node

Blockage of transmission of the cardiac impulse from the S-A node to the other parts of the heart
Example: A-V block:

Cardiac impulses fails to pass from atria into the ventricles.

↓

The atria continues to beat at the normal rhythm rate of the S-A node.

+

A new pacemaker develops in the Purkinje system with a new rate.

From gayton :
Occasionally some other part of the heart develops a rhythmical discharge rate that is more rapid than that of the sinus node. For instance, this sometimes occurs in the A-V node or in the Purkinje fibers when one of these becomes abnormal. In either case, the pacemaker of the heart shifts from the sinus node to the A-V node or to the excited Purkinje fibers. Under rarer conditions, a place in the atrial or ventricular muscle develops excessive excitability and becomes the pacemaker.

Action potential of the pacemaker (pacemaker potential)

The cell membrane of pacemaker cells is leaky to Na+. This decreases gradually membrane potential . Na+ influx is who start depolarization, if the potential becomes at a certain potential Transient Ca++ will open

Then Ca++ influx d.t opening of transient Ca++ channels(t type) decreases MP from -60 mv to a firing level of -40 mv.

At firing level, long lasting (L type) Ca++ channels open & Ca++ influx occurs causing fast change of MP from -40 to + 10-20 mv. (depolarization)

At peak, K+ outflux begins & MP returns to -60 mv. (repolarization).

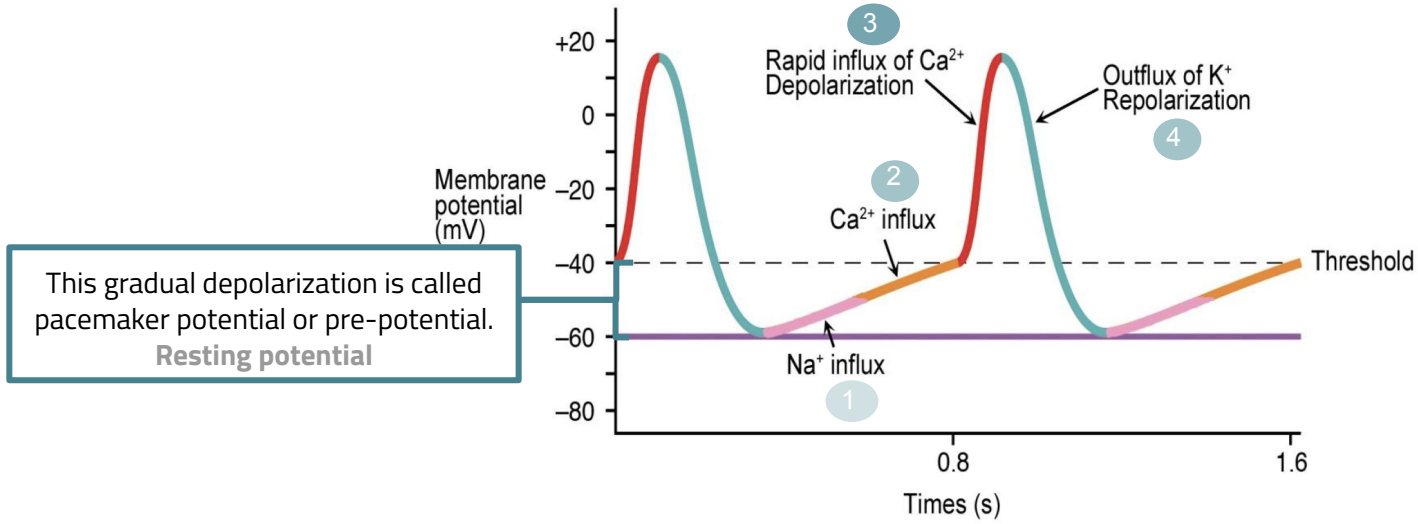
Then the cycle is repeated by self excitation.

(1) Na+ influx

(2) Ca++ influx

(3) rapid Ca++influx

(4) outflux of K+REPOLARIZATION



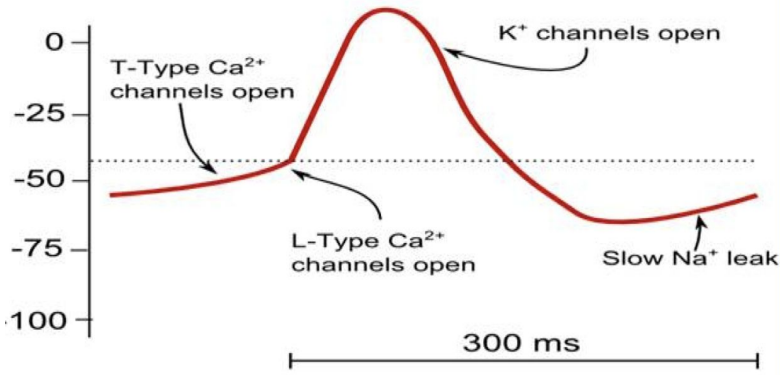
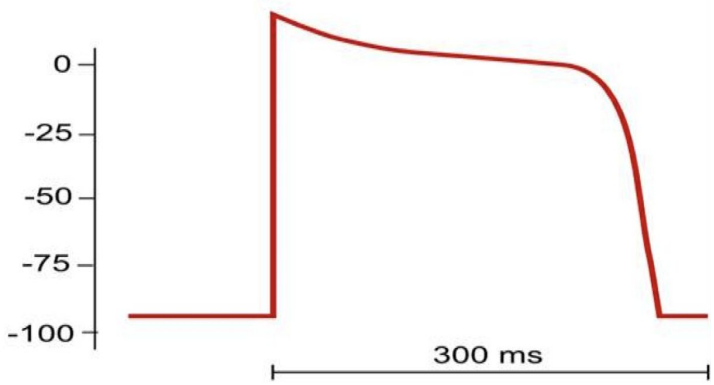
From Gayton:

The "pacemaker" potential gradually rises and becomes less negative between each two heartbeats. When the potential reaches a threshold voltage of about−40 millivolts, the L-type calcium channels become "activated," thus causing the action potential. Therefore, basically, the inherent leakiness of the sinus nodal fibers to sodium and calcium ions causes their self-excitation.

From Linda:

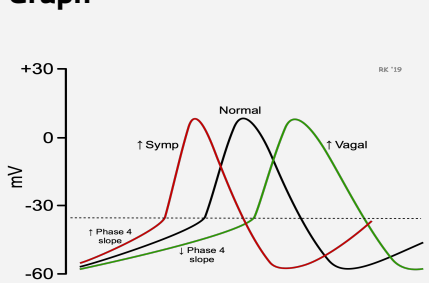
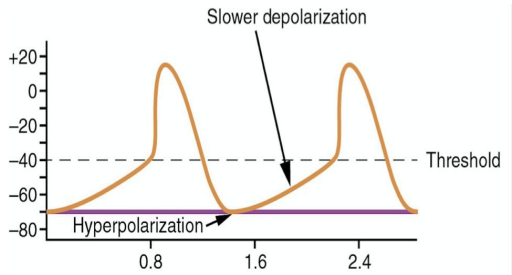
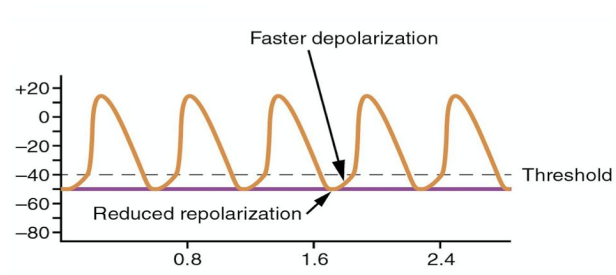
If the rate of the pacemaker potential increases, threshold is reached more quickly, the SA node will fire more action potentials per time, and heart rate will increase. Conversely, if the rate of the pacemaker potential decreases, threshold reached more slowly, the SA node will fire fewer action potentials per time, and heart rate will decrease.

Pacemaker Vs. Myocardial Cell Action Potential

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

Control of Heart Rhythmicity and Impulse Conduction by the Cardiac Nerves

The heart is supplied by both sympathetic and parasympathetic nerves

	Parasympathetic (decrease the rhythmicity)	Sympathetic (increase the rhythmicity)
Rhimicity	Decreased	Increased
Supplied by	**Vagus nerve** N.B. Strong stimulation of the vagi: ▪ Stop completely the rhythmical excitation by the S-A node ▪ Block completely transmission of cardiac impulses from the atria to the ventricle ▪ Some point in the Purkinje fibers develops a rhythm of its own **"Ventricular Escape"**	**Sympathetic trunk**
Affects	Mainly SA and AV nodes (the atrium)	All parts of the heart with strong supply to the ventricles
Slope of Pre-potential (rhythm) **& Ion Permeability of S-A node**	Decreased pre-potential slope of S-A node due to increased the K+ permeability (Away from firing/threshold because it will be more negative. That means it will take more time to reach firing)	Increase the slope of pre-potential of S-A node due to increase the permeability to Na+ & Ca++, (Closer to firing/threshold because it will be more positive. That means it will take less time to reach firing)
Impulses transmission to A-V Node	slows heart rate	Increases heart rate
Force of Contraction	Decrease	Increase
Graph	 	

Summary of Action Potential

Skeletal Muscle vs Cardiac Muscle

Comparison of Action potentials in cardiac and skeletal Muscle

	Skeletal muscle	Contactile Myocardium	Autorhythmic myocardium
Membrane potential	Stable at -70 mV	Stable at -90 mV	Unstable pacemaker potential ; usually start at -60 mV.
Events leading to threshold potential	Net Na+ entry through ACh operated channels	Depolarization enters via Gap junctions	Net N+ entry through I_f Channels;reinforced by Ca2+ 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++ entry; rapid phase caused K+ efflux	Rapid; caused by K^+ efflux
Hyperpolarization phase	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

MCQs:

1-Where is the exact location of the SA Node ?
A) superior posterio-lateral wall of the right atrium
B) superior posterio-lateral lateral wall of the left atrium.
C) inferior lateral wall of the right atrium.
D) superior lateral wall of the left atrium.

2 -A-V bundle conducts impulses to A-V bundle branch at velocity of:
A) 0.1 m/sec.
B) 1 m/sec.
C) 0.3 m/sec.
D) 0.03 m/sec.

3-What makes Purkinje Fibers so fast?
A) Absence of gap junctions
B) high permeability of gap junctions
C) Made of normal cardiac cells
D) Both A&C

4- Velocity of conduction between SA-Nodal fibers is:
A) 0.3 m/sec.
B) 0.03 m/sec.
C) 0.5 m/sec.
D) 0.05 m/sec.

5- Why SA Node is called the Pacemaker of the Heart?
A) Has the lowest autorhythmicity
B) has the fastest rate of autorhythmicity
C) Made of Special Cardiac Fibers
D) None

6- What is the normal Pacemaker of the Heart?
A) Bundle of His
B) The S-A node
C) A-V bundle
D) Purkinje Fibers

7- What's a cause of ectopic focus?
A) Purkinje fibers become overexcited
B) purkinje fibers self-controlled
C) both
D) neither

8- What's the conduction velocity of Purkinje Fibers?
A) 0.05 m/sec
B) 0.3 m/sec
C) 1.00m/sec
D) 4.00 m/sec

9- What starts depolarization at pacemaker potential?
A) Na+ influx
B) transient Ca++ influx
C) long lasting Ca++ influx
D) K+ outflux.

10- Which one of these sentences <u>isn't</u> correct about Pacemaker Action Potential?
A) RMP is -60 mv.
B) Depolarization is gradual.
C) Does not need a stimulus
D) It has plateau

Answer Key: 1A - 2B - 3B - 4D - 5B
6B - 7A - 8D - 9A - 10D

SAQs:

1- Mention the three types of the inter-nodal pathway?

2- The cardiac impulse does not travel from the atria to the ventricles too rapidly, can you explain why ?

3- Describe what happens at the action potential of the pacemaker?

4- What is the importance of plateau?

A1: Anterior internodal bundle of Bachman Middle internodal bundle of Wenkebach Posterior internodal bundle of Thoral

A2: to give enough time for emptying the blood into the ventricles. And to prevent pathological high atrial rhythm.

A3:

1-The cell membrane of pacemaker cells is leaky to Na+. This decreases gradually MP.
2-Then Ca++ influx d.t opening of transient Ca++ channels decreases MP from -60 mv to a firing level of -40 mv.
3-At firing level, long lasting Ca++ channels open & Ca++ influx occurs causing fast change of MP from -40 to + 10-20 mv. (depolarization)
4-At peak, K+ outflux begins & MP returns to - 60 mv. (repolarization).

A4: Maintain Depolarization

Team Leaders

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