



Cardiovascular System Block Cardiac Electrical Activity: Conducting System (Physiology)

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Learning Objectives



Cardiac Electrical Activity

Automaticity of the heart: the heart is capable of:-

- Generating rhythmical electrical impulses
- **Conduct** the 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?





Components of the Conducting System



The Conducting System of the Heart



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

The Sinoatrial Node (S-A node)

- Located in the superior lateral wall of the right atrium. Its fibers are continuous with atrial fibers.
- It is made of modified cardiac muscles.
- 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
- It is the normal Pacemaker of the heart
- SA node has the fastest rate of autorhythmicity.



Representation

Why?

The Internodal Pathway

Action potential can travel from S-A node to spread through out the atrial muscle through two routes:-

- □Ordinary atrial muscle fibers (conduction velocity is 0.3 m/s).
- □Internodal pathway:-
 - Anterior internodal bundle of Bachman
 - Middle internodal bundle of Wenkebach
 - Posterior internodal bundle of Thoral.
 (conduction velocity is 1 m/s.)





The A-V node



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

- The impulse after leaving S-A node takes 0.03 sec to reach the A-V node.
- 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)



Significance Of A-V Nodal Delay (0.13 sec)

The cardiac impulse does not travel from the atria to the ventricles too rapidly:-

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 protects ventricles from pathological high atrial rhythm Why The cause of slow conduction is mainly diminished number of gap junctions between the successive cells

The A-V 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.

 Bundle of His 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.

IV-The 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.





IV-The Purkinje System.....Cont.

- Purkinje fibers are very large fibers
 Transmit action potentials at a very high velocity (1.5-4.0 m/sec)
 - 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
 - \rightarrow enhance the velocity of transmission
- Significance: ensures 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 of Impulse: The One-Way Conduction Through A-V Bundle

- APs spread from S-A node through internodal pathway quickly at velocity of 1.0 m/sec.
- Impulses pass through A-V node at a velocity of 0.01 m/sec.). Time delay occurs (0.13 sec).
- 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.
- A special characteristic of the A-V bundle is inability of action potentials to travel backward from the ventricles to the atria.
- The atrial muscle is separated from the ventricular muscle (except at the A-V bundle) by a continuous fibrous barrier which acts as an insulator to prevent re-entry of cardiac impulse by this route from the ventricles to the atria.



Conduction Velocities & Spread of the Cardiac Impulse Through The Heart



S-A node Internodal pathway A-V node Bundle of His Purkinje system Atrial & Ventricular muscles

0.05 m/sec. 1.00 m/sec. 0.01 m/sec. (slowest) 1.00 m/sec. 4.00 m/sec. (Fastest) 0.3-0.5 m/sec.

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



Control of Excitation and Conduction in the Heart

- The S-A node is the normal **<u>Pacemaker</u>** of the Heart.
- It has pacemaker pre-potential (autorhythmic tissue).
- it initiates the excitation wave, drive whole heart and makes the pace (speed) of heart at a rate of 105 impulse/min, inhibited by right vagus nerve to be 70 impulse/min (vagal tone).
- Its rate of rhythmical discharge is faster than that of any other part of the heart, so it derives rest of the heart.



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

latent Pacemakers

- A-V node, His bundle & 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, or its impulse is blocked, or if the rate of firing of the latent pacemakers increases.
- If S-A node is damaged, A-V node becomes the new pacemaker and heart follow it but at a slower rate (50-60 impulse/min) (A-V nodal rhythm).



latent Pacemakers.....Cont.

- 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).
- Rhythmicity is high in S-A node > A-V node > His bundle & Purkinje fibers.



Reprint

Abnormal (Ectopic) Pacemakers

- Ectopic pacemaker: a pacemaker elsewhere than the SA node
- In some cases, Purkinje fibers can become overexcited = ectopic focus and cause premature ventricular contraction.
- It can occurs upon excess caffeine, lack of sleep, anxiety, stress or some organic conditions.



Causes of Ectopic Pacemakers

- 1- Any other part of the heart develops a rhythmical discharge rate that is more rapid than that of the SA node
- 2- Blockage of transmission of the cardiac impulse from the S-A node to the other parts of the heart
 - Example: A-V block
 - \rightarrow 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

Action potential of the pace maker (pace maker potential)

- The cell membrane of pace maker cells is leaky to Na⁺. This decreases gradually MP.
- Then Ca⁺⁺ influx d.t opening of <u>transient</u>
 <u>Ca⁺⁺ channels</u> decreases MP from -60 mv to a firing level of -40 mv.
- This gradual depolarization is called pace maker potential or pre-potential.
- At firing level, <u>long lasting Ca⁺⁺ channels</u> open & Ca⁺⁺ influx occurs causing fast change of MP from -40 to + 10 mv. (depolarization).
- At peak, K⁺ outflux begins & MP returns to -60 mv. (repolarization).
- Then the cycle is repeated by self excitation.



Differences between pace maker P & AP of myocardial cells

Pacemaker AP	Ventricular AP
Weighting the second se	0- -25- -50- -75- -100- 300 ms
Pace Maker Action Potential	Ventricular Muscle Action Potential.
Does not need a stimulus	Needs a stimulus
RMP is -60 mv.	RMP is -90 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

<u>Control of Heart Rhythmicity and Impulse Conduction by the</u> <u>Cardiac Nerves</u>

- The heart is supplied with both sympathetic and parasympathetic nerves
- <u>Sympathetic nerves</u>: to all parts of the heart with strong supply to the ventricles
- Parasympathetic nerves (vagi): mainly to the S-A and A-V nodes



Sympathetic stimulation of the heart

- the slope of pre-potential of S-A node (i.e ↑ rhythm of the S-A node) due to increase the permeability to Na⁺ & Ca⁺⁺, so accelerate the heart rate.
- ↑ transmission of impulses to the A-V node
- ↑ force of myocardial contraction



Parasympathetic stimulation of the heart

- ↓ the slope of pre-potential of S-A node due to increase the permeability to K⁺ (i.e ↓ rhythm of the S-A node, so slow the heart rate).
- \downarrow transmission of impulses to the A-V node
- 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 VS Parasympathetic stimulation of the heart



For further readings and diagrams:

<u>Textbook of Medical Physiology by Guyton & Hall</u> <u>Chapter 10 (Rhythmical Excitation of the Heart)</u>

