Rhythmical Excitation of the Heart

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Special Excitory and Conductive System of the Heart

Sinus Node (or sinoatrial node or S-A): A small node with almost no contractile muscle, where signals originate.

A-V bundle: The conductive bridge between the atrial and ventricular lobes.

Purkinje Fibers: Transmit the signals to all parts of the ventricles.

Sinus Fibers: The sinus fibers have the ability to self-excite, and maintain the rhythm of the heart.
Mechanisms

Fast Sodium Channels → Slow Sodium Channels → Pottassium Channels

Leakage of sodium ions and self-excitation

Reversion to normal
Transmission of Cardiac Impulse (AV)
Transmission of Cardiac Impulse (Purkinje)

Transmission is 6 times faster than in AV node
Action potentials cannot travel backwards

Control of Excitation and Conduction

- **Pacemaker of the heart (Sinus Node)**
  - cardiac impulse transmission does not always necessarily start in the Sinus node
  - When start in the A-V nodal fibers and discharge at 40 to 60 times per minute or in the purkinje fibers where they discharge between 15 to 40 times per minute
  - regardless, cardiac transmissions mainly discharge from the sinus node
    - 70-80 times per minute
  - it’s discharge also excites the a-v nodal fibers and the purkinje fibers
  - discharges the a-v nodal or purkinje fibers before they can self excite
  - Sinus node controls the beat of the heart as the “pace maker” because it has the fastest rhythmical discharge
Abnormal Pacemaker

- “Ectopic” Pacemaker

  - pacemaker anywhere other than the sinus node, causes abnormal contraction in heart and debility of the heart pumping

  - this happens when another part of the heart such as the a-v node or purkinje fibers develop a rhythmic discharge faster than that of the sinus node

  - signal is blocked from the sinus node and pacemaker starts in the a-v node on the way to the ventricles

  - atria continues to beat at normal rate of sinus node

  - pacemaker develops in purkinje system for the ventricles to contract ventricle muscle

  - purkinje “pacemaker” starts 5-20 seconds later because fibers are suppressed from previous sinus over-load

  - during these 5-20 seconds the ventricle fails to pump blood and the person faints after 4-5 seconds

  - this whole process is called Stokes-Adams syndrome and can lead to death if delay period is too long.
Purkinje System and synchronous contraction

- Purkinje system contractions happen only 0.03 to 0.06 apart
  - contractions are happening at almost the exact same time
  - any slowing in the purkinje fiber signaling will result in irregular contraction of the ventricle
  - this leads to many debilities of the heart and can decrease pumping effectiveness by 20-30 percent
Sympathetic and Parasympathetic Nerves

- Parasympathetic nerves (vagi)
  - mainly distributed to S-A and A-V nodes, and a small amount extend to the atria with even less to the ventricles

- Sympathetic nerves
  - conversely, distributed to all areas of the heart
Parasympathetic stimulation

- Can slow or even block cardiac rhythm
  - parasympathetic stimulation causes release of acetylcholine
    - decreases rate of sinus node, and decreases excitability of the A-V junction fibers slowing contraction of ventricles
    - can block complete transmission from atria to ventricles through A-V node
    - ventricle stops beating for 5 to 20 seconds until purkinje fibers kick in
      - this is called ventricular escape
    - Acetylcholine release increases permeability of nerve endings and allows leakage of potassium causing “hyperpolarization”
    - Because of this, membrane potential takes much longer to reach the threshold potential for excitation
    - Because of this there are moderate delays or possible blocks the rate of rhythmicity of the nodal fibers.
Sympathetic stimulation

- Is responsible for the exact opposite effects of parasympathetic (vagal) stimulation
  - Increases rate of nodal discharge
    - Increases rate of conduction and level of excitability in all nervous fibers
    - Increases force of contraction of all cardiac musculature (atrial/venticle)
    - Basically increases overall function of the heart as much as two fold
Mechanisms of sympathetic stimulation

- Begins with the secretion of norepinephrine in sympathetic nerve endings
  - norepinephrine stimulates beta-1 adrenergic receptors
  - the exact effect is unclear but is believed to increase permeability of fiber membrane causing increase sodium-calcium levels
  - this causes a more positive resting potential increasing the threshold level for self excitation therefore increasing heart rate
  - A-V node and A-V bundles increase in sodium-calcium permeability makes action potential easier and therefore exciting each conducting fiber faster and increasing conduction time from the atria to the ventricles
Atrioventricular (AV) node