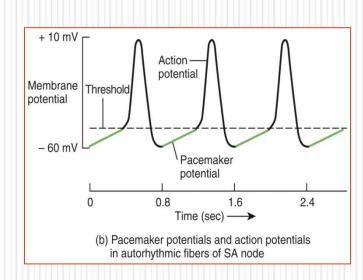
# Cardiovascular Physiology The conducting system of the heart Fall 2019-2020

**Part 3-4** 

Dr. Khalid Maseer



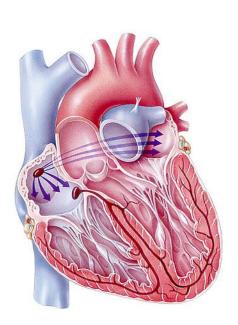
#### Properties of the cardiac muscle

- □ Cardiac muscle contraction is similar to skeletal muscle contraction
- □ The heart has four basic properties which are essential for its functioning as the central pump of the CVS. These are:
- 1. Autorhythmicity
- 2. Conductivity
- 3. Excitability
- 4. Contractility

#### 1. Autorhythmicity

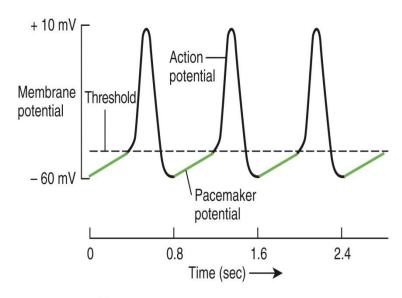
□During embryonic development, about 1% of all of the muscle cells of the heart form a network or pathway called the cardiac conduction system.

□ This specialized group of myocytes called Autorhythmic cells.



#### **Autorhythmic Fibers**

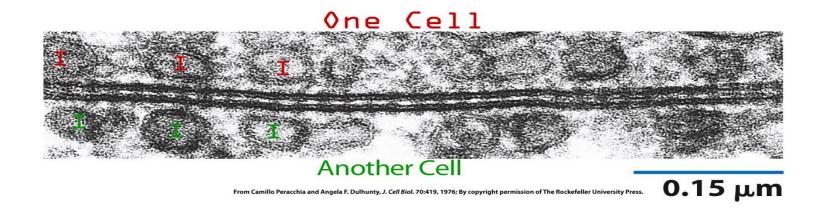
- □ They have the ability to spontaneously depolarize (self-excitable)
- Repeatedly generate action potentials that trigger heart contractions.
- ☐ The rhythmical electrical activity they produce is called autorhythmicity.
- Because heart muscle is autorhythmic, it does not rely on the central nervous system to sustain a lifelong heartbeat.



(b) Pacemaker potentials and action potentials in autorhythmic fibers of SA node

#### **Autorhythmic Fibers**

- □ Autorhythmic cells spontaneously depolarize at a given rate, some groups faster, some groups slower.
- □ Once a group of autorhythmic cells reaches threshold and starts an action potential (AP), ions spread through gap junctions of the Intercalated discs (I) to allows the AP to pass from cell to cell, so all of the cells in that area of the heart also depolarize.



#### **Autorhythmic Fibers**

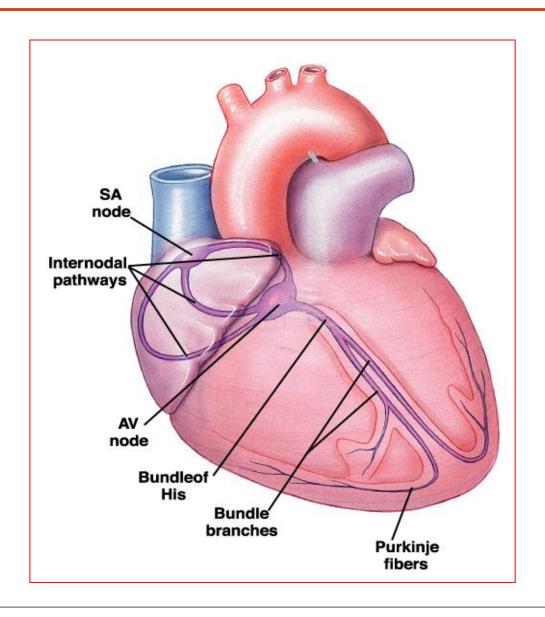
- ☐ The self-excitable myocytes that "act like nerves" have the 2 important roles:
  - ✓ Act as **pacemaker** within that system: setting the rhythm of electrical excitation that causes contraction of the heart.
  - ✓ Forming the **conduction system:** a network of specialized cardiac muscle fibers that provide a path for each cycle of cardiac excitation to progress through the heart, and ensures that cardiac chambers become stimulated to contract in a coordinated manner

## 2. Conductivity

□ Conductivity: Impulses can spread easily between cardiac muscle fibers.

☐ Yet, conduction in the heart is normally carried out by the specialized conducting system to ensure the spread of the excitation wave from the S-A node to all over the heart in certain pattern.

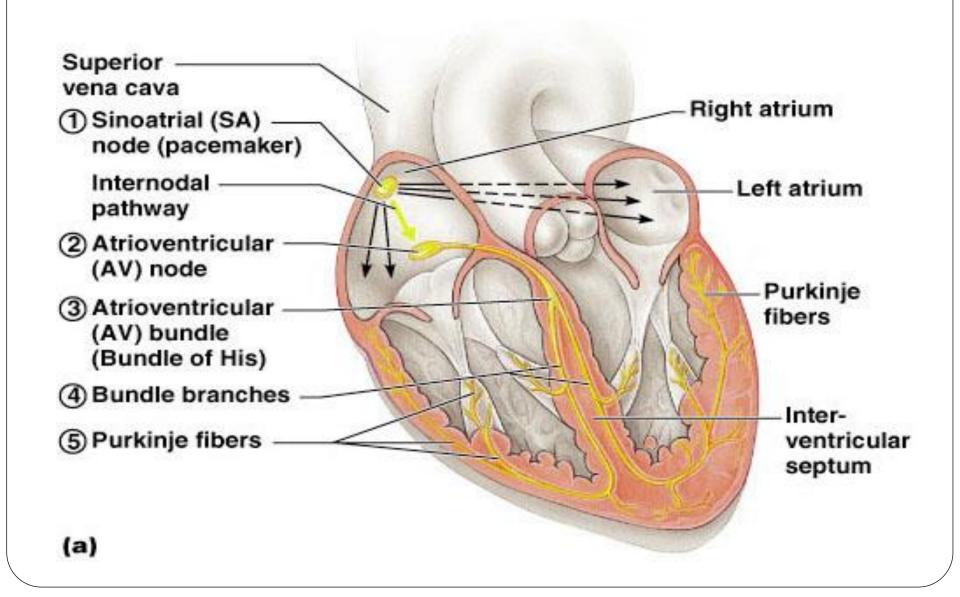
## The conducting system of the heart



## **Conduction system: Sequence of Excitation**

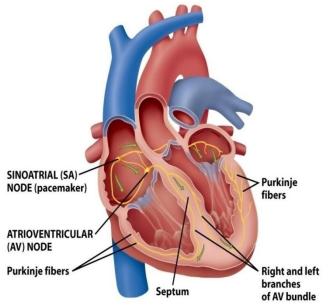
- □ Sinoatrial (SA) node: located in the right atrial wall.
- Atrioventricular (AV) node: located in the interatrial septum, just anterior to the opening of the coronary sinus
- **☐** Atrioventricular bundle (bundle of His):
  - ✓ Only site where action potentials can conduct from atria to ventricles due to fibrous skeleton
  - ✓ AV bundle splits into two pathways in the interventricular septum:
    - 1. Bundle branches which extends through interventricular septum toward apex
    - 2. Purkinje fibers

## **Conduction system: Sequence of Excitation**



## **Cardiac Conduction (SA node)**

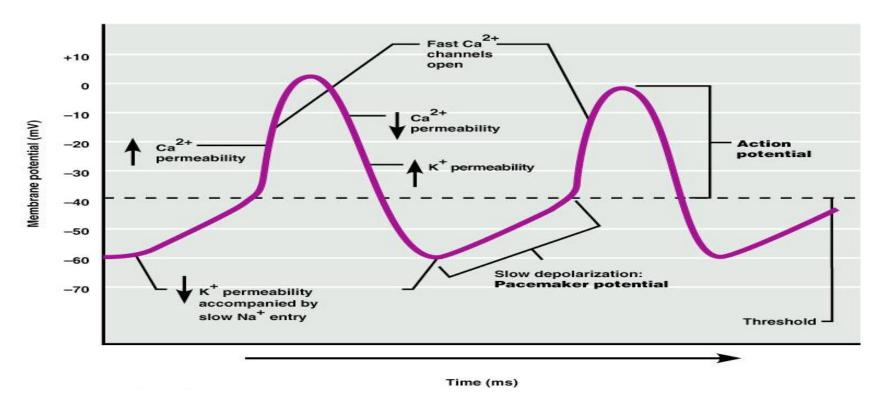
■ Because it has the fastest rate of depolarization, the normal pacemaker of the heart is the sinoatrial (SA) node, located in the right atrial wall just below where the superior vena cava enters the chamber.



#### **Cardiac Conduction (SA node)**

- □ The pacemaker cells are characterized by having an unstable membrane potential. This is the basis for automaticity. SA node acts as natural pacemaker
  - ✓ Faster than other autorhythmic fibers
- □ After firing an action potential, the membrane potential decreases i.e. the membrane depolarizes gradually from a basal value of ~ -60mV to a critical firing level of −45 mV. At this level, an **action potential** is fired and the cycle is repeated.
- □ The gradual depolarization of the S-A nodal cells is called the **pacemaker potential** or the prepotential.

#### **Pacemaker Potentials of the Heart**

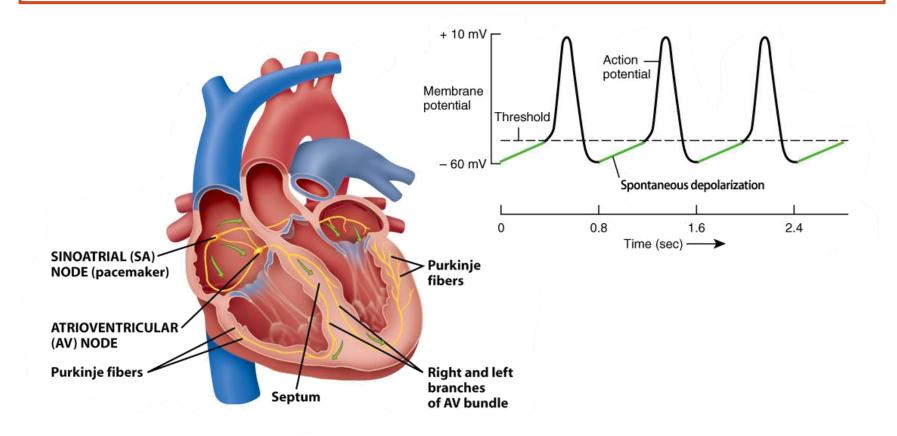


The early part of the pacemaker potential is caused by a decrease in the permeability of the membrane to K<sup>+</sup>. The late part is caused by Ca<sup>++</sup> influx through the transient (T-type) Ca<sup>++</sup>-channels.

#### **Cardiac Conduction (SA node)**

- ■SA node generates impulses about every 0.6 second, or 100 action potential/minute.
  - ✓ Propagates through atria via gap junctions
  - ✓ Atria contact
- □ Nerve impulses from autonomic nervous system (ANS) and hormones modify timing and strength of each heartbeat
  - ✓ Do not establish fundamental rhythm

#### **Cardiac Conduction (SA node)**

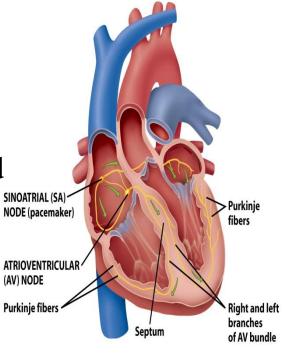


Spontaneous depolarization of autorhythmic fibers in the SA node firing about once every 0.6 seconds, or 100 action potentials per minute

#### **Cardiac Conduction (AV node)**

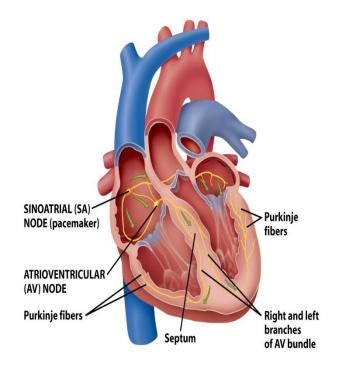
□ The action potential generated from the SA node reaches the next pacemaker by propagating throughout the wall of the atria to the AV node in the interatrial septum.

At the AV node, the signal is slowed approximately 0.1 second, allowing the atrium a chance to mechanically move blood into the ventricles.



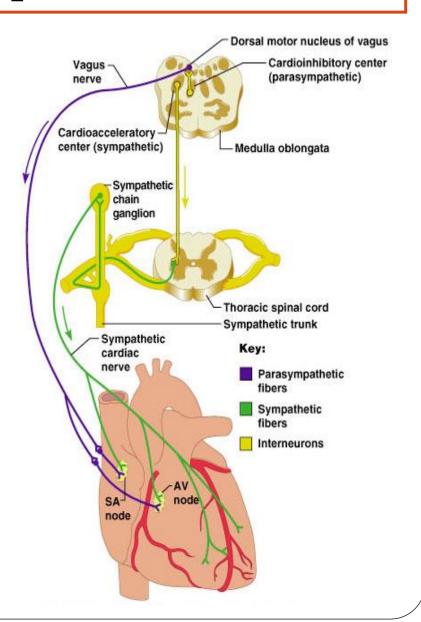
#### Cardiac Conduction (Atrioventricular bundle )

- ☐ From the AV node, the action potential enters the atrioventricular (AV) bundle. This bundle is the only site where action potentials can conduct from the atria to the ventricles.
- ☐ The A-V bundle conducts impulses only in one direction. So impulse pass through the AV bundle to the left and right bundle branches in the interventricular septum towards the apex of the heart.
- ☐ Finally, the **Purkinje fibers** rapidly conduct the action potential throughout the ventricles (0.2 seconds after atrial contraction).
- ☐ Then the **ventricles contract**, pushing the blood upward toward the semilunar valves.



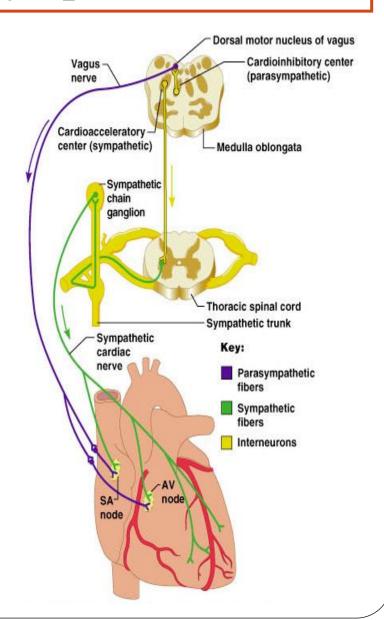
#### **Autonomic regulation (sympathetic)**

- ☐ Heart is stimulated by the sympathetic cardioacceleratory center.
  - ✓ Noreprinephrine has 2 separate effects
    - In SA and AV node speeds rate of spontaneous depolarization
    - In contractile fibers enhances Ca<sup>2+</sup> entry increasing contractility



## **Autonomic regulation (parasympathetic)**

- ☐ Heart is inhibited by the parasympathetic cardioinhibitory center.
- ✓ Parasympathetic nerves release acetylcholine which decreases heart rate by slowing rate of spontaneous depolarization



#### 3. Excitability

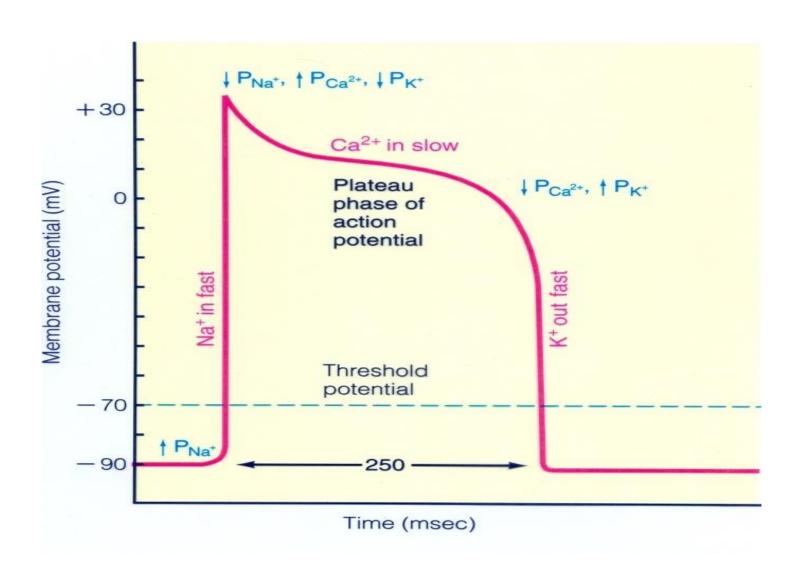
**Excitability** is the ability to respond to stimuli.

□ The resting membrane potential of the contractile fibers is stable at about – 90 mV. When an effective stimulus is applied, a propagated **action potential** is produced in the cell membrane.

#### **Action Potentials and Contraction**

- □ Action potential initiated by SA node spreads out to excite "working" fibers called contractile fibers.
- An action potential occurs in a contractile fiber as follows:
- 1. Depolarization
- 2. Plateau
- 3. Repolarization

#### **Action Potential of Contractile Cardiac Muscle Cell**



#### Rapid depolarization

- **Depolarization:** It is caused by the **rapid influx of Na**<sup>+</sup> into the cell.
- ✓ Unlike autorhythmic fibers, contractile fibers have a stable resting membrane potential that is close to -90 mV.
- ✓ When a contractile fiber is brought to threshold by an action potential from neighboring fibers, its voltage-gated fast Na channels open.
- ✓ These sodium ion channels are referred to as "fast" because they open very rapidly in response to a threshold-level depolarization.

## Rapid depolarization

□ Opening of these channels allows Na inflow because the cytosol of contractile fibers is electrically more negative than interstitial fluid and Na concentration is higher in interstitial fluid.

□ Inflow of Na down the electrochemical gradient produces a **rapid depolarization. Within a few milliseconds**, the fast Na channels automatically inactivate and Na inflow decreases.

#### Plateau

- □ Plateau, a period of maintained depolarization.
- □ It is due in part to **opening of voltage-gated slow Ca<sup>2+</sup> channels** in the sarcolemma. The inward movement of Ca<sup>2+</sup> and the **decreased efflux of K**<sup>+</sup> maintain the membrane potential near zero during this phase of the action potential.
- $\square$  The plateau phase lasts for about 0.25 seconds.
- □ By comparison, depolarization in a neuron or skeletal muscle fiber is much briefer, about 1 msec (0.001 sec), because it lacks a plateau phase.

#### Repolarization

□ Repolarization: due to a reduction of the inward Na+ and Ca2+ currents and a large increase in the outward K+ current.

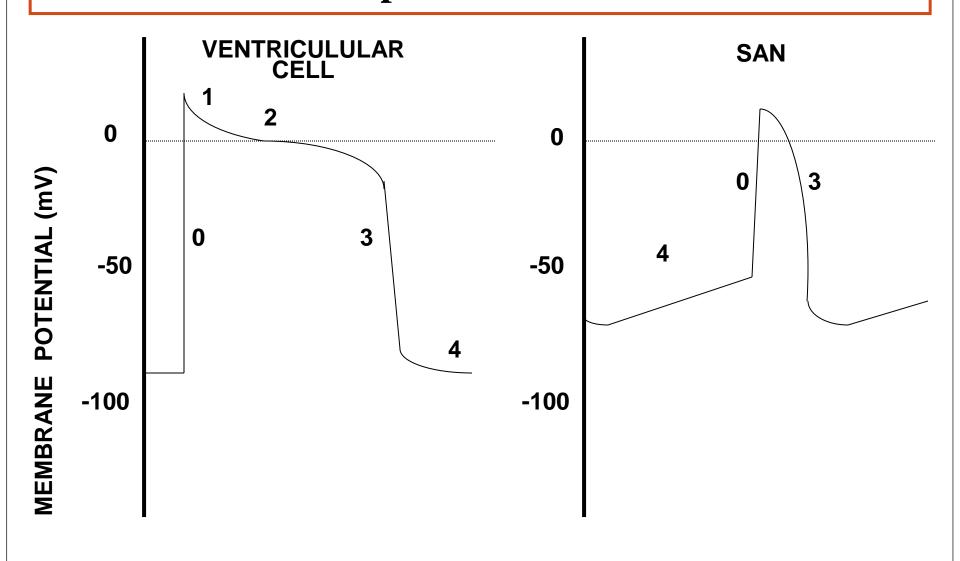
□ The membrane goes back to the resting level (- 90 mV).

□ Na+-K+ pump works to drive the excess Na+ out and the excess K+ in.

#### Differences between pacemaker and contractile cells

- ☐ The pacemaker action potential differs from the action potential of the contractile myocardial cells in the following:
- □ Depolarization phase is mainly due to Ca2+ influx through long-lasting (L-type) Ca2+-channels.
- Depolarization phase is relatively slow to develop.
- ☐ There is no plateau phase. Repolarization immediately follows depolarization.

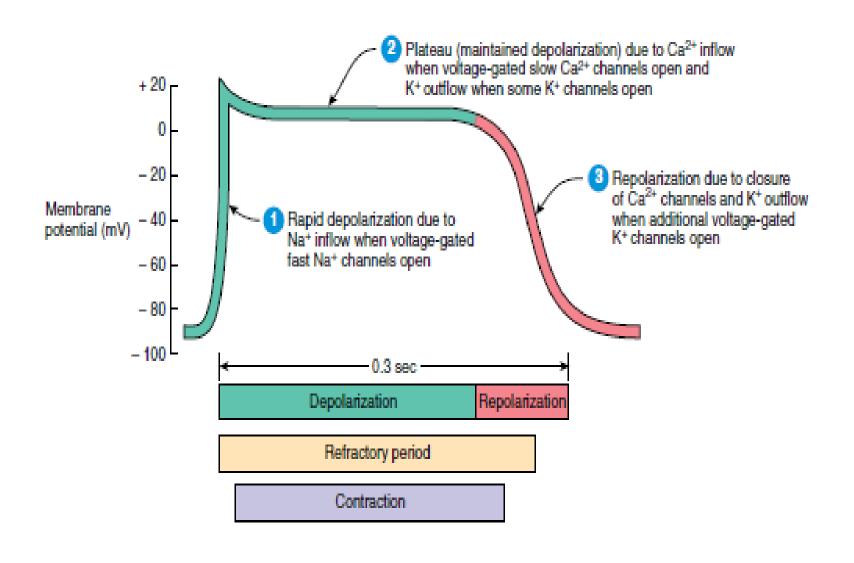
#### Differences between pacemaker and contractile cells



## **Refractory periods**

- ☐ In muscle, the refractory period is the time interval during which a second contraction cannot be triggered.
- ☐ The refractory period of a cardiac muscle fiber lasts longer than the contraction itself. As a result, another contraction cannot begin until relaxation is well under way.
- ☐ This means that the ventricle would not respond to any stimulus until it finishes with its systole and have some diastole.
- □ So refractory period protects the ventricle against tetnization if it receives multiple successive stimuli.

#### **Refractory periods**



#### 4. Contractility

□ Contractility is the ability of the muscle to convert the potential energy into mechanical energy.

#### **ATP Production in Cardiac Muscle**

- Cardiac muscle relies almost exclusively on aerobic cellular respiration in its numerous mitochondria.
- The needed oxygen diffuses from blood in the coronary circulation and is released from myoglobin inside cardiac muscle fibers.

