ELECTRICAL & MECHANICAL ACTIVITY OF THE MYOCARDIAL CELL

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Objectives

By the end of the session the learner should be able to;-

Define

- polarization
- depolarization
- repolarization
- action potential
- refractory periods
- Describe the five (5) phases of the action potential
- Describe regulation of the CVS

Action Potential (AP)

The AP is a **five** phase cycle that reflects the difference in the concentration of ions across the cell membrane at any given time.

A series of events causes the electrical charge inside the cell to change from its *resting state* (negative) to its *depolarized* or stimulated state (positive) and back to its resting state (negative).

Resting Membrane potential

- In a myocardial cell at rest, the normal resting membrane potential is approximately -80 to -90mV.
- The interior of the cell is relatively negative
- The outside of the cell is relatively positive

IONS	EXTRACELLULAR CONC (meq/l)	INTRACELLULAR CONC (meq/l)
K+	4	135
Na+	145	10
Ca++	2	0.1

Depolarization

- When the cardiac muscle cell is stimulated, the cell is said to be depolarized.
- The inside of the cell is said to be more +ve d/t entry of Na+ ions into the cell through Na+ membrane channels.
 - Depolarization occurs from the innermost layer of the heart to the outermost

Repolarization

- After the myocardial cell has depolarized, the diffusion of Na⁺ into the cell stops. K+ is allowed to diffuse out of the cell.
- Thus repolarization occurs d/t the outward diffusion of the K+.
- The membrane potential of the cell returns to its negative resting level.
 - Repolarization proceeds from the outermost layer to the innermost layer.

Phases of the Cardiac Action Potential

There are five (5) phases:Phase 0

- 1. Opening of the fast Na+ channels
- 2. Na+ influx into the cell
- 3. Corresponds with the **QRS complex** on the ECG.

- 1. Brief period of early repolarization
- 2. Fast Na+ channels partially close, K+ moving out as well
- 3. Get a negative deflection on the graph

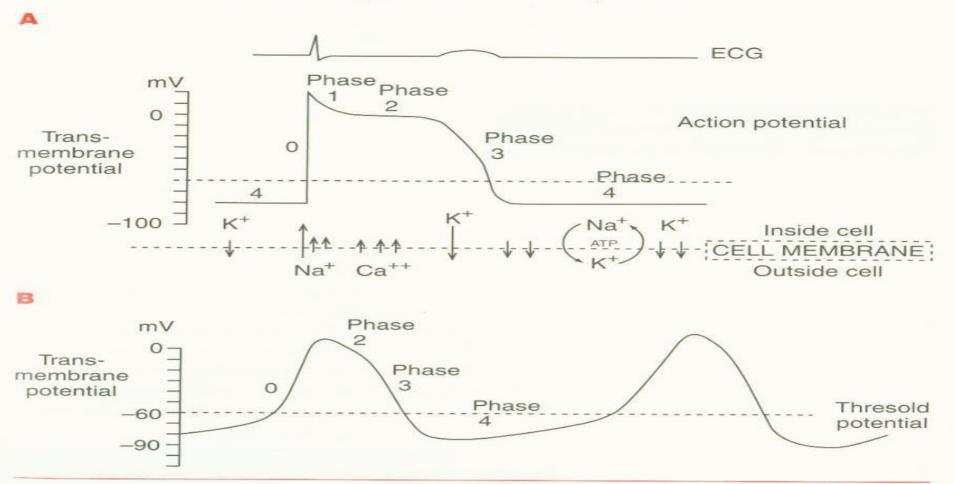
- 1. Slow inward movement of Ca ++
- 2. Slow opening of calcium channels
- 3. Continued outward flow of K⁺
- 4. The Ca ++ entering the cell at this phase causes cardiac contraction
- 5. Phase 2 is responsible for **ST segment** on the ECG
- 6. CC blockers e.g. verapamil and diltiazem inhibit the inward flow of Ca++

- 1. This is the repolarization phase
- 2. K⁺ moves out of the cell
- 3. Ca ++ and Na+ channels close
- 4. It corresponds with ventricular repolarization i.e. **T wave** on the ECG.
- 5. The intracellular environment hence becomes more negative thereby reestablishing RMP.

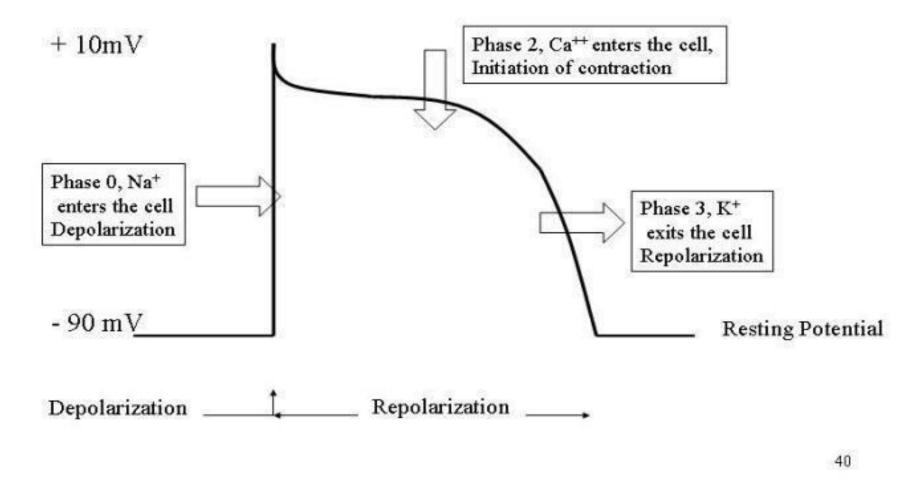
- 1. AP returns to RMP of -80 to -90mV.
- The excess Na⁺ that entered the cell during depolarization is now removed from the cell in exchange for K⁺ by means of Na⁺/K⁺ pump.
- This mechanism returns intracellular concentrations of Na⁺ and K⁺ to levels before depolarization.

Myocardial Cell Action Potential

FIGURE 1. A, Schematic representation of ventricular myocardial working cell action potential. Arrows indicate times of major ionic movement across cell membrane. **B,** Schematic representation of pacemaker cell action potential.



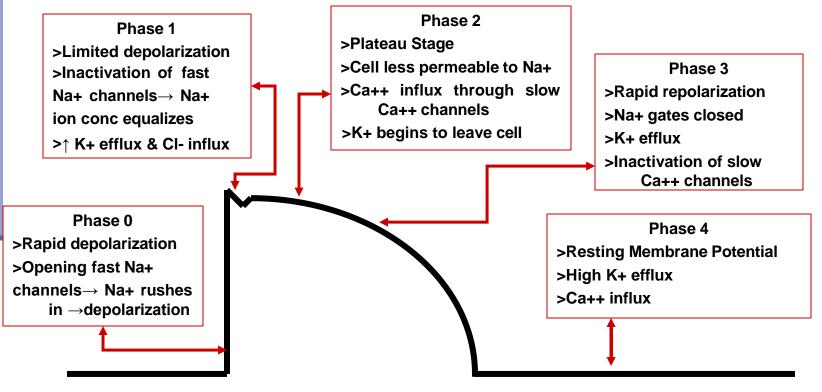
Monophasic Action Potential (Cardiac Muscle Cell)



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Phases of Action Potential

PHASES OF ACTION POTENTIAL



Excitation – contraction coupling (electromechanical coupling)

- Mechanical contraction occurs during phase 2 of the AP
- As the myocardial cell is depolarized, some Ca++ moves from the sarcoplasmic reticulum (SR) to the cytoplasm via Ca++ channels
- The cytoplasmic Ca++ then binds with troponin & tropomyosin (molecules in the Actin filaments) resulting in contraction
- After contraction, Ca++ is taken back into the SR, cytoplasmic Ca++ drops hence muscular relaxation

Excitation –contraction coupling...

Contraction & relaxation are active processes needing energy from ATP; Ca is removed from cell by the **Na+/Ca++ pump**

Refractory Periods

- Refractoriness is a term used to describe the extent to which a cell is able to respond to a stimulus.
- Absolute refractory period- It is the period during which the myocardium will not respond to further stimulation no matter how strong the impulse.

Relative Refractory Period- it is the period during which some cardiac cells have repolarized to their threshold potential and can be stimulated to respond (depolarize) to a stronger than normal stimulus.





Regulation of Heart Function

By M.P .Nguru

REGULATION OF THE HEART BEAT

- The heart is innervated by both the sympathetic and parasympathetic divisions of the autonomic nervous system
- The sympathetic (SNS) division mobilizes the body, allowing the body to function under stress ("fight" or "flight" response)
 - **The parasympathetic (PNS)** division is responsible for the conservation & restoration of body resources ("feed" and "breed" response)

- The PNS and SNS affect the CV function by slowing the HR during periods of calm and increasing it in response to sympathetic stimulation.
- PNS fibers are concentrated mostly near the SA or AV conduction tissue & in the atria.
- SNS nerve fibers have a greater impact on the ventricles.

Intrinsic Regulation

- In addition to the nervous control, there are several reflexes that serve as feedback mechanisms to the brain. They work to maintain even blood flow, oxygenation & perfusion.
- Baroreceptors- are specialized nerve tissue/ stretch sensors located in the internal carotid arteries & the aortic arch. These sensory receptors detect changes in BP and cause reflex response in either the sns or pns of the autonomic nervous system.

- These sensors detect a change in wall conformation, usually as a result of ↑se or ↓se in BP.
- E.g., if the SBP ↓ses, the body's normal compensatory response is;
 - peripheral vasoconstxn,
 - ↑sed HR &
 - ↑sed myocardial contractility.

- Increases in volume or pressure cause parasympathetic stimulation to decrease BP.
- Carotid sinus pressure will decrease sympathetic nervous system activity & increase vagal (parasympathetic) activity to decrease the heart rate & BP.

This is through the alteration in the baroreceptor input to the vasomotor centre in the medulla (brain stem) to cause reflex tachycardia.

- **2. Chemoreceptors-** located in the **internal carotid arteries & at the bifurcation of the aortic arch,** detect changes in the conc. of H⁺, O₂, and CO₂ in blood (i.e. pH<7.4, PO₂ <80mmHg and PCO₂ >40mmHg).
- The primary function of the chemoreceptors is to maintain homeostasis during hypoxemia
 Stimulation of the chemoreceptors cause an ↑se in RR and depth.

- **3. Right Atrial Receptors-** When the pressure in the right atrium rises sufficiently to stimulate these stretch receptors, it causes a reflex tachycardia.
- This reflex protects the right side of the heart from an overload state and to quickly equalize filling pressures of the right and left side of the heart.

- 4. Natriuretic peptide- The heart secretes two major natriuretic peptides. The atrial myocardium secretes Atrial Natriuretic Peptide (ANP), and the ventricular myocardium secretes Brain Natriuretic Peptide (BNP).
 - Both are released in atrial and ventricular chamber stretch.
- Both peptides cause vasodilation, natriuresis and inhibit SNS and RAAS.

5.Renin Angiotensin Aldosterone System (RAAS) is activated by low BP or intravascular volume depletion.

6.Respiratory Influences- The HR usually accelerates on inspiration and decelerates on exhalation

Other factors influencing heart rate

a. Conc. of potassium in the ECF: Hyperkalemia slows the HR and can block conduction of an impulse from atria to ventricles

Hypercalcaemia causes the heart to go into spastic contractions.

Hypocalcaemia causes the heart to become flaccid.

- b. Hormone levels e.g. thyroxin, epinephrine and norepinephrine
- c. Medications- CC blockers
- d. Stress
- e. Anxiety
- f. Fear
- g. Body temperature- HR ↑ses when body temperature ↑ses and vice versa.





Summary

- Defined
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 - repolarization
 - action potential
 - refractory periods
 - Described the five (5) phases of the action potential
- Described regulation of CVS