EXCITATION CONTRACTION COUPLING

(MECHANISM OF CONTRACTION IN RESPONSE TO ACTION POTENTIAL)

LEARNING OBJECTIVES

- Explain the mechanism of calcium role in cardiac muscle contraction
- Define the sources of calcium in cardiac muscle
- Mention the steps of calcium consumption in cardiac muscle

CRITERIA FOR SPREAD OF EXCITATION & EFFICIENT CARDIAC FUNCTION

1. Atrial excitation and contraction should be complete before onset of ventricular contraction
   - ensures complete filling of the ventricles during diastole

2. Excitation of cardiac muscle fibres should be coordinated
   →ensure each heart chamber contracts as a unit
   →accomplish efficient pumping
   - smooth uniform contraction essential to squeeze out blood

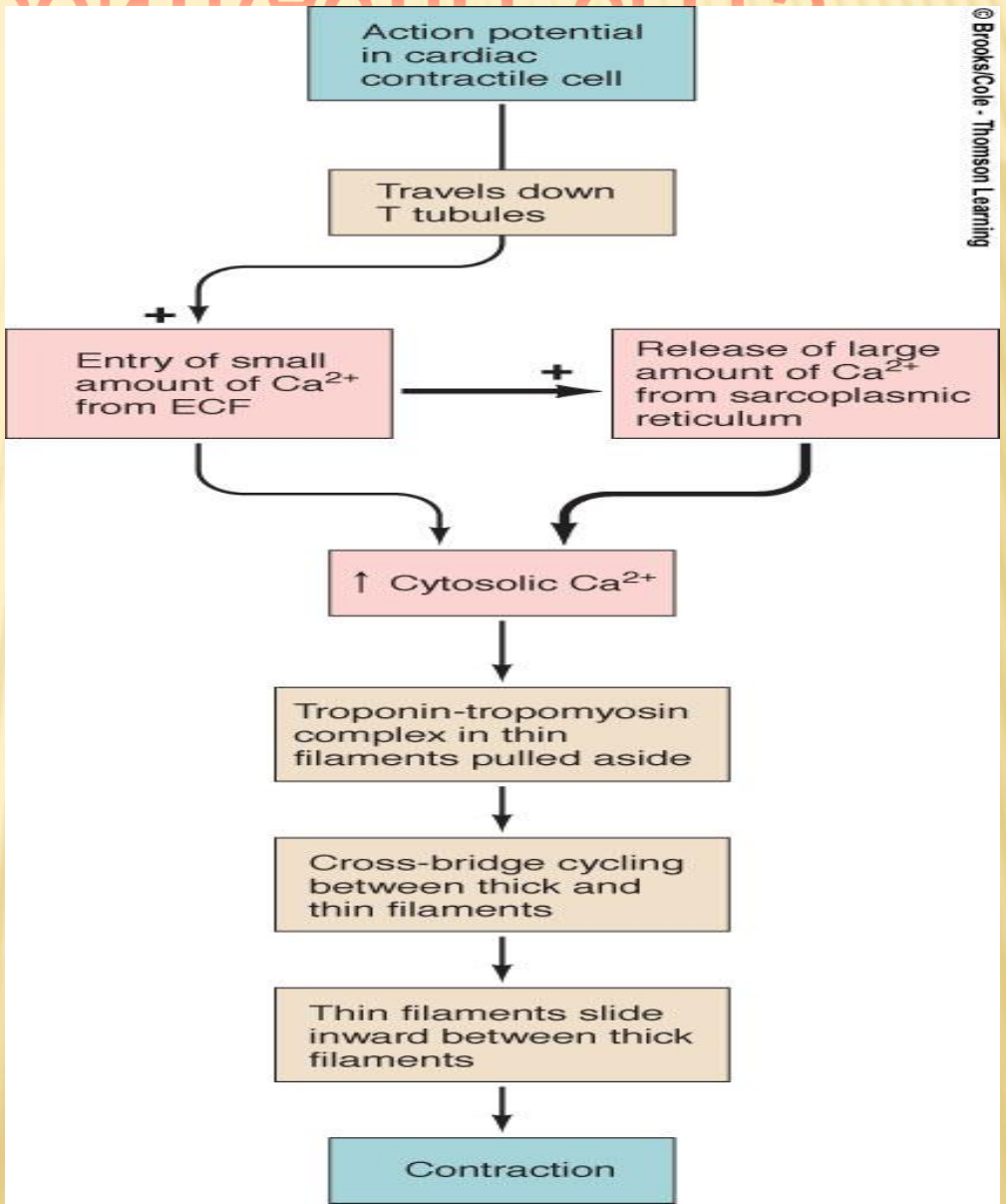
3. Pair of atria & pair of ventricles should be functionally co-ordinated → both members contract simultaneously
   - permits synchronized pumping of blood into pulmonary & systemic circulation

EXCITATION-CONTRACTION COUPLING IN CARDIAC CONTRACTILE CELLS

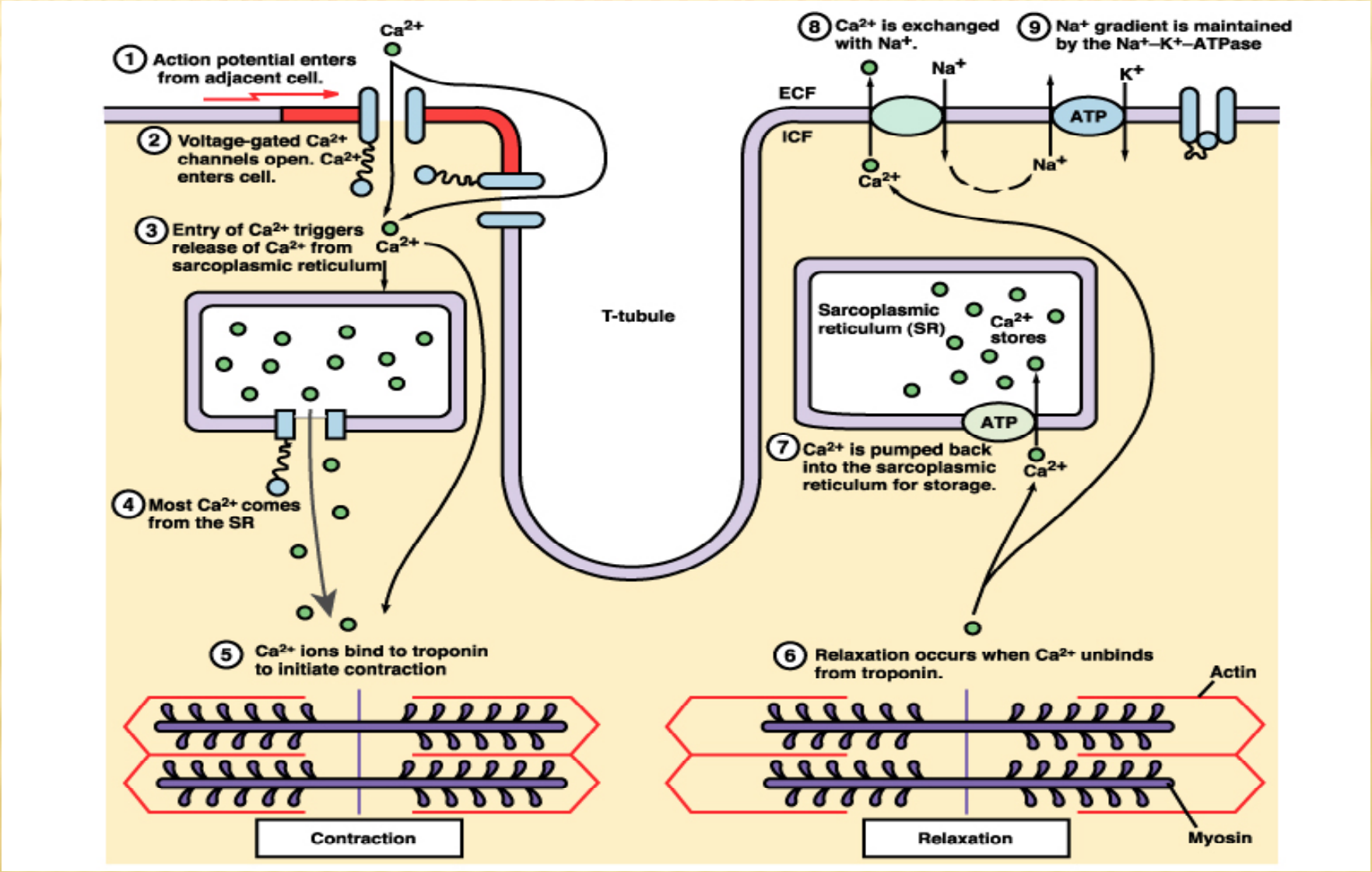
Similar to that in skeletal muscles

BUT in Myocardium:

1. **Ca entry through T tubules.**
2. **5 times more larger T tubules**
3. **Less developed SR**
4. **Extra-cellular Ca concentration affects myocardial contraction more**



EXCITATION-CONTRACTION COUPLING IN CARDIAC CONTRACTILE CELLS



STEPS IN EXCITATION CONTRACTION COUPLING

1. Action potential from adjacent cell membrane
2. Voltage gated Ca channels are opened
3. Ca ions enter into the cells through these channels as well as through T tubules

CONT:
STEPS IN EXCITATION CONTRACTION COUPLING

1. Ca reaches to Sarcoplasmic reticulum, causes the release of stored Ca in SR in cytosol.
2. Ca binds to troponins for contraction
3. Actin and myosin slide on each other and causing contraction of the muscles.
4. When Ca unbinds troponin, relaxation occurs

CONT:
STEPS IN EXCITATION CONTRACTION COUPLING

7. When Ca unbinds troponin, relaxation occurs
8. Ca ions mostly enter into SR
9. Some of the Ca ions goes out with exchange of Na ions
10. Na ions gradient is mantained by

    Na-K ATPase

ACTION POTENTIAL IN SINO-ATRIAL NODE CELL

OR

SELF EXCITATORY CELL

OR

AUTORHYTHMIC CELL

ACTION POTENTIAL OF A CONTRACTILE MYOCARDIAL CELL A TYPICAL VENTRICULAR CELL

- **Depolarization**

- Opening of fast voltage-gated Na+ channels.
- Rapid Influx of Sodium ions leading to rapid depolarization.

- **Small Repolarization**

- Opening of a subclass of Potassium channels which are fast channels.
- Rapid Potassium Efflux.

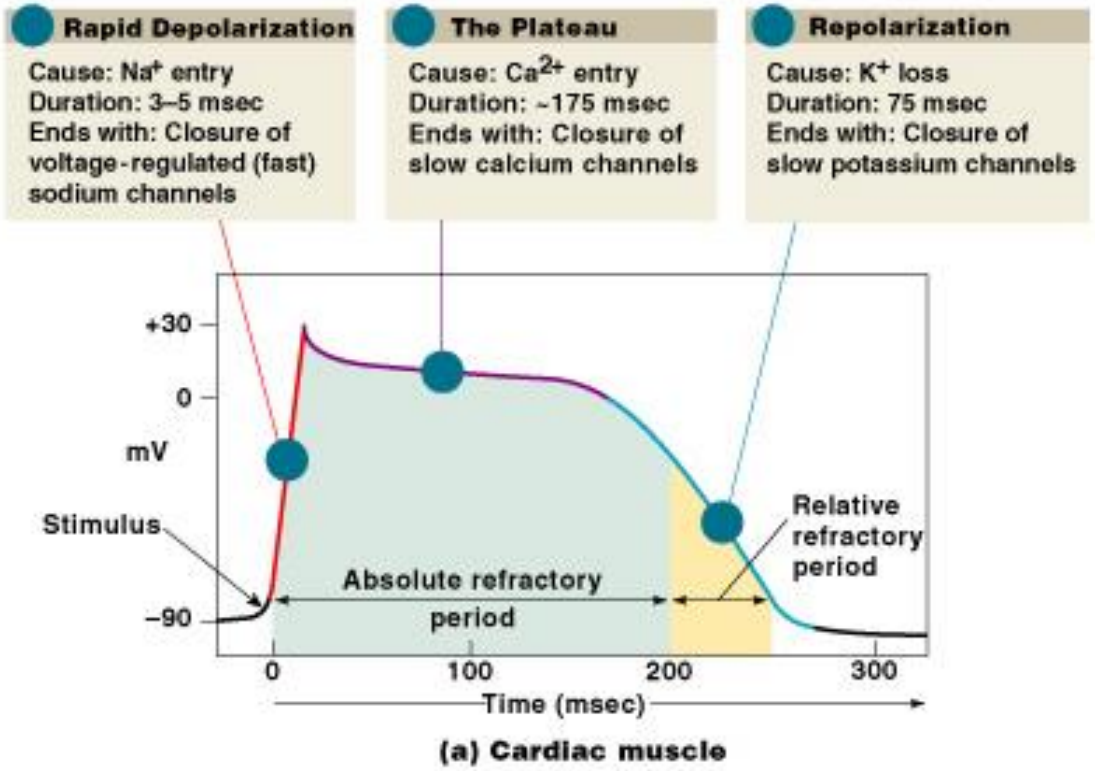
- **Plateau phase**

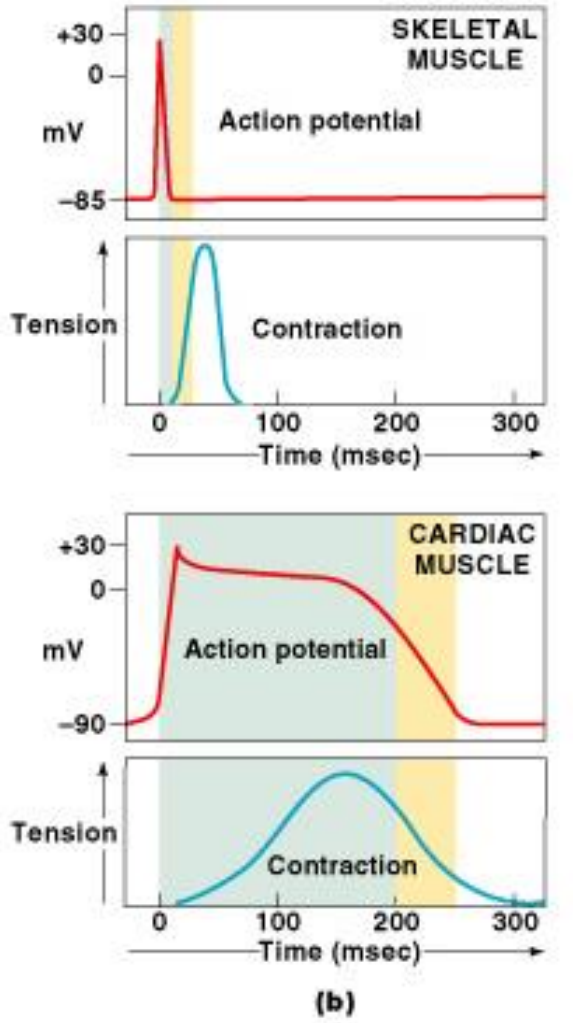
- 250 msec duration (while it is only 1msec in neuron)
- Opening of the L-type voltage-gated slow Calcium channels & Closure of the Fast K+ channels.
- Large Calcium influx
- K^+ Efflux is very small as K^+ permeability decreases & only few K channels are open.

- **Repolarization**

- Opening of the typical, slow, voltage-gated Potassium channels.
- Closure of the L-type, voltage-gated Calcium channels.
- Calcium Influx STOPS
- Potassium Efflux takes place.

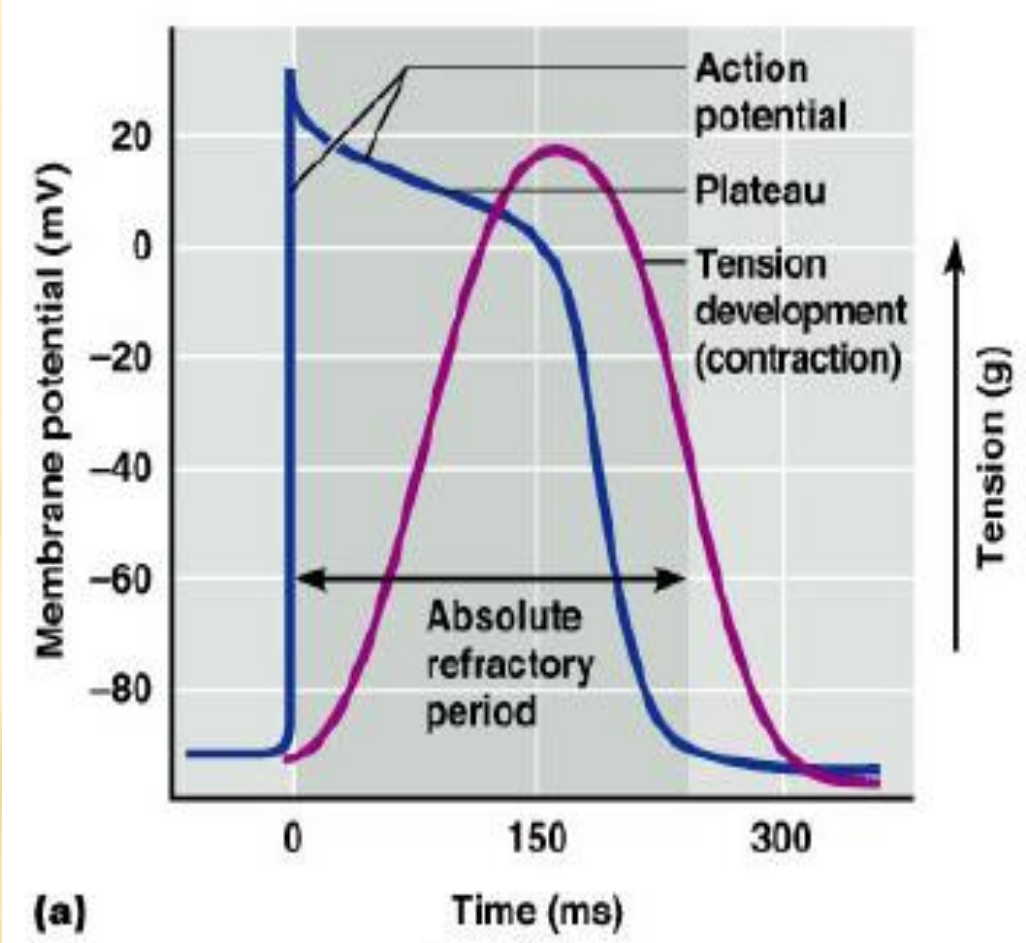
ACTION POTENTIALS IN SKELETAL AND CARDIAC MUSCLE





REFRACTORY PERIOD

- **Long** refractory period (250 msec) compared to skeletal muscle (3msec)
- During this period membrane is refractory to further stimulation until contraction is over.
- It lasts longer than muscle contraction, prevents tetanus
- Gives time to heart to relax after each contraction, prevent fatigue
- It allows time for the heart chambers to fill during diastole before next contraction

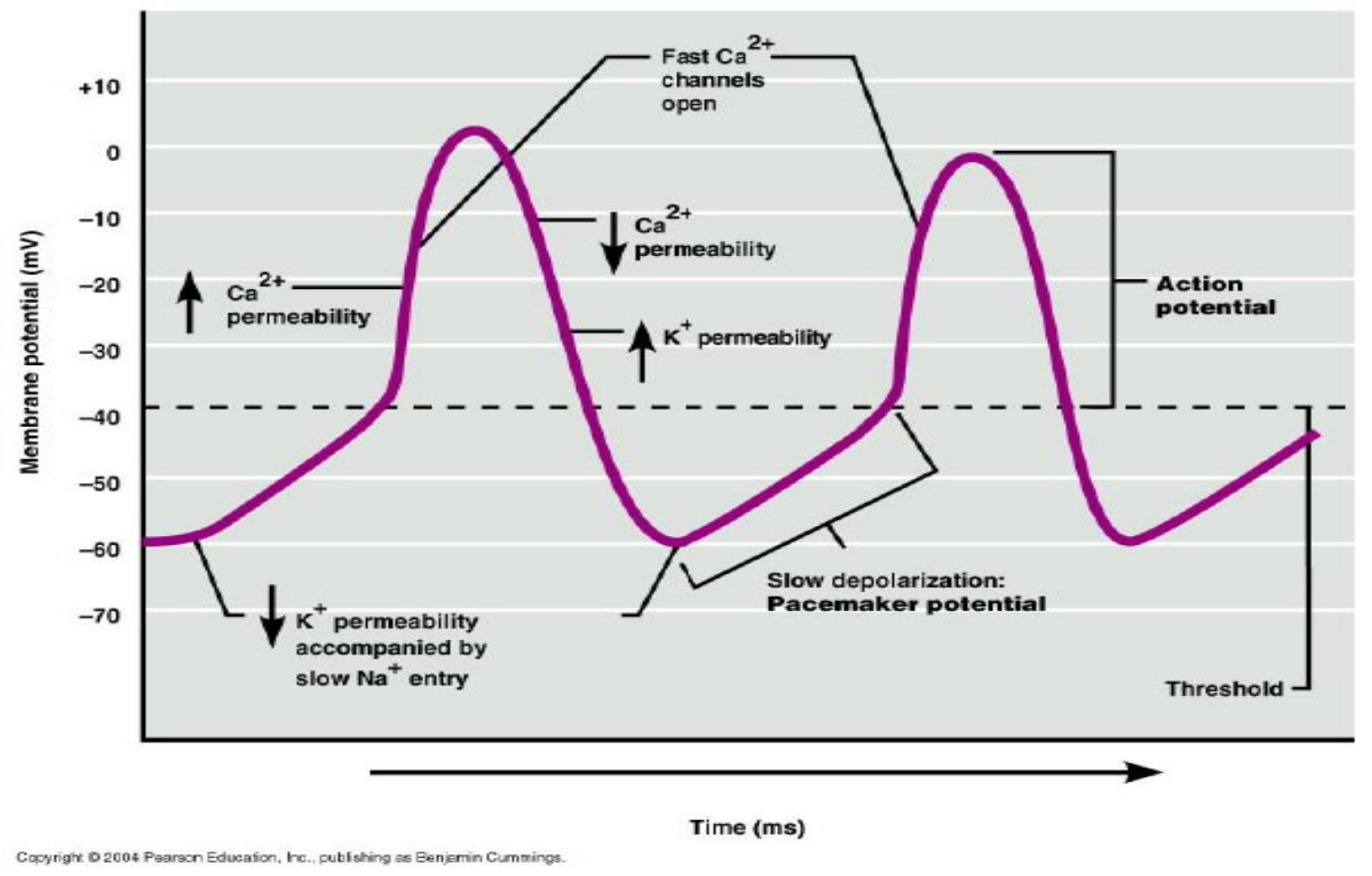


AP in skeletal muscle : 1-5 msec
AP in cardiac muscle :200 -300 msec

OBJECTIVES

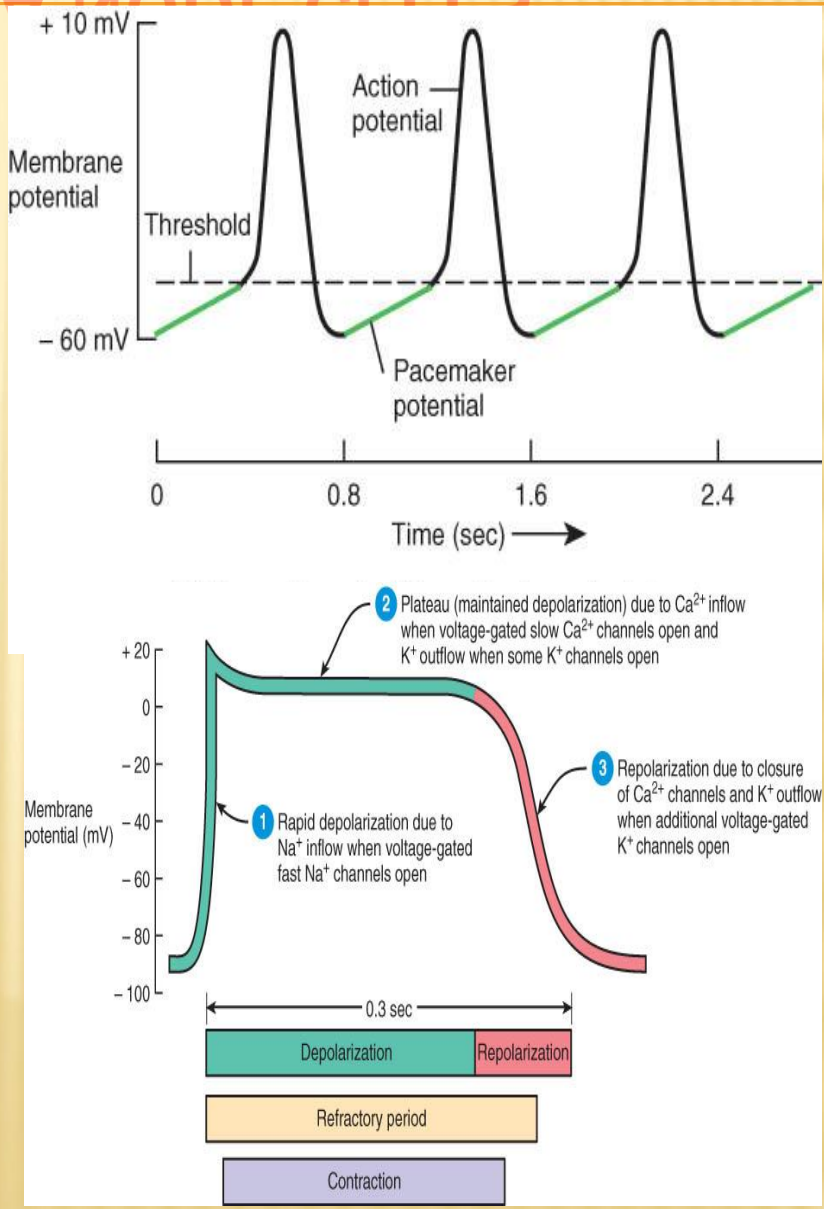
1. Cardiac muscle, structural and functional properties.
2. Define the terms; Rhythmicity, Excitability, Conductivity and Contractility.
3. Describe cardiac syncytium.
4. Outline the normal pathway of the cardiac impulse.
5. Describe the excitation-contraction coupling in cardiac muscles and compare it to excitation-contraction coupling in skeletal muscles.
6. Compare and contrast action potential in sinoatrial node and ventricular muscle.
7. Explain the significance of the plateau and refractory periods in ventricular muscle action potential.

ACTION POTENTIAL IN S.A NODE CELL (SELF EXCITATORY OR AUTORHYTHMIC CELLS)



COMPARISON OF ACTION POTENTIAL IN VENTRICULAR AND SA NODE CELLS

1. Resting membrane potential
2. Permeability to ions
3. Slow depolarization,
4. Slow Na and Ca channels
5. No Plateau formation
6. unstable RMP during pause
7. Longer time period of Action potential in SA node.
8. Self excitation



COMPARISON

- Autorythmic cells do not have stable resting membrane potential (RMP)
- Natural leakiness to Na & Ca→ spontaneous and gradual depolarization
- Unstable resting membrane potential (= pacemaker potential)
- Gradual depolarization reaches threshold (-40 mv) → spontaneous AP generation