

## MYOCARDIAL CELL PROPERTIES

### A. Action and Resting Potentials

#### 1. Ventricular myocardium

a. resting potential: about -80 or -90 mv

- 1) similar to other excitable tissue
- 2) not normally self-excitatory

b. action potential phases (see numbers on figure)

- 0) initial rapid depolarization
- 1) small initial repolarization
- 2) plateau (long)
- 3) repolarization
- 4) resting potential

c. action potential duration

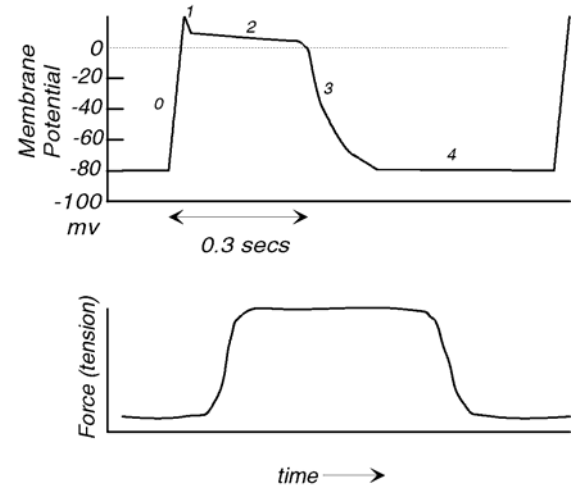
- 1) about 0.3 seconds at rest (ventricular)
- 2) shortens with increasing heart rate

d. ionic basis: channels

- 1) fast  $\text{Na}^+$  channels
  - a) regenerative, Hodgkin cycle
  - b) open briefly upon depolarization, then inactivated (A and I gates)
  - c) remain inactivated until after repolarization
- 2) slow  $\text{Ca}^{2+}$  -  $\text{Na}^+$  channels
  - a) admit both  $\text{Ca}^{2+}$  (mainly) and  $\text{Na}^+$
  - b) open upon depolarization, delayed
- 3) resting  $\text{K}^+$  channels
  - a) open when membrane resting, close upon depolarization
- 4) transient  $\text{K}^+$  channels
  - a) open briefly upon depolarization
- 5) slow (delayed)  $\text{K}^+$  channels
  - a) open later upon depolarization

e. sequence

- 1) resting state:  $P_{\text{K}}$  high,  $P_{\text{Na}}$  &  $P_{\text{Ca}}$  low
- 2) membrane depolarizes to threshold due to conducted excitation
- 3) fast  $\text{Na}^+$  channels open (phase 0), leading to rapid depolarization
- 4) fast  $\text{Na}^+$  channels close and transient  $\text{K}^+$  channels open briefly, leading to small repolarization (phase 1)
- 5) slow  $\text{Ca}^{2+}$  -  $\text{Na}^+$  channels open, leading to an inward current that balances the outward  $\text{K}^+$  current (note that  $P_{\text{K}}$  is reduced from resting); thus plateau (phase 2)
- 6) slow  $\text{K}^+$  channels eventually open, causing repolarization (phase 3); the decreased membrane potential causes  $\text{Ca}^{2+}$  -  $\text{Na}^+$  channels to close
- 7) the membrane returns to its resting potential (phase 4), but remains refractory for several tenths of a second; resting  $\text{K}^+$  channels open, establishing resting potential

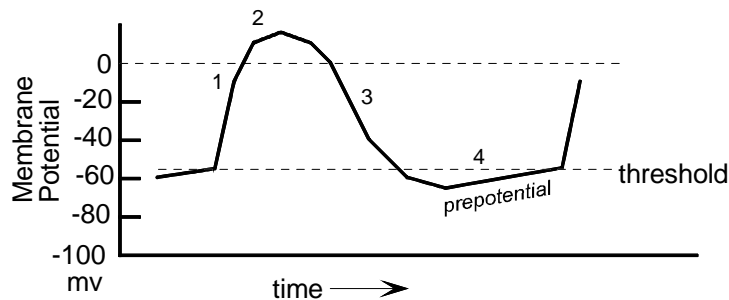


#### 2. Atrial cells: resemble ventricular cells, but shorter AP duration

A. Action and Resting Potentials (continued)

3. Nodal tissue (SA & AV nodes)

- a. resting potential  $\cong$  -60 mv
- b. spontaneous depolarization reaching threshold, given sufficient time
  - 1) termed autorhythmic or self-excitation
  - 2) resting potential depolarization termed prepotential
  - 3) probably due to non-specific  $I_f$  channels (both  $\text{Na}^+$  &  $\text{K}^+$  permeable, with  $\text{Na}^+$  influx  $>$   $\text{K}^+$  efflux) until threshold reached ( $I_f$  = "funny current")
  - 4) rate of prepotential slope depends on the particular cell and external influences (e.g. autonomic & endocrine, temperature)
- c. lacks fast  $\text{Na}^+$  channels
- d. action potential shape
  - 1) depolarization due to opening of  $\text{Ca}^{2+}$  channels (phase 1)
  - 2)  $\text{Ca}^{2+}$  close,  $\text{K}^+$  channels begin to open (phase 2)
  - 3)  $\text{K}^+$  outflux (phase 3)
  - 4) prepotential, slow depolarization due to  $I_f$  channels (phase 4)

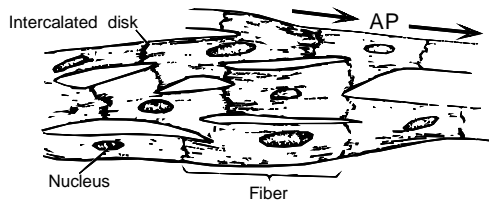


Note: Atrial and ventricular cells, which normally are excited by conduction from adjacent tissue, can generate nodal type APs if hypoxic, subject to certain drugs, etc. That is, atrial and ventricular cells have the potential to become self excitatory (autorhythmic).

## MYOCARDIAL CELL PROPERTIES (continued)

### B. Excitation Conduction

1. Cardiac muscle is an electrical syncytium (acts as a single unit): an action potential, once initiated, travels as far as possible via cell-to-cell conduction through low resistance gap junctions (intercalated disks)



2. Conduction limitation: fibrous A-V skeleton block AP conduction, except through the AV node
3. Conduction velocity

Atrial fibers	80 cm/sec
Ventricular fibers	30
A-V nodal fibers	5 (small diameter)
Purkinje fibers	200

Note: Two possible modes of excitation, conducted and autorhythmic (not neural excitation)

### C. Mechanical Contraction

1. Molecular Basis: similar to skeletal muscle (interaction of thick myosin filaments and thin actin-troponin-tropomyosin filaments)
2. Excitation-Contraction Coupling
  - a. Depolarization => contraction
  - b. Repolarization => relaxation
  - c. Mediated by  $\text{Ca}^{2+}$ , but  $\text{Ca}^{2+}$  entering the cell during AP acts as a trigger for internal  $\text{Ca}^{2+}$  release (contrast with skeletal muscle cells)

Note: there is a delay between the membrane potential changes and the mechanical contraction

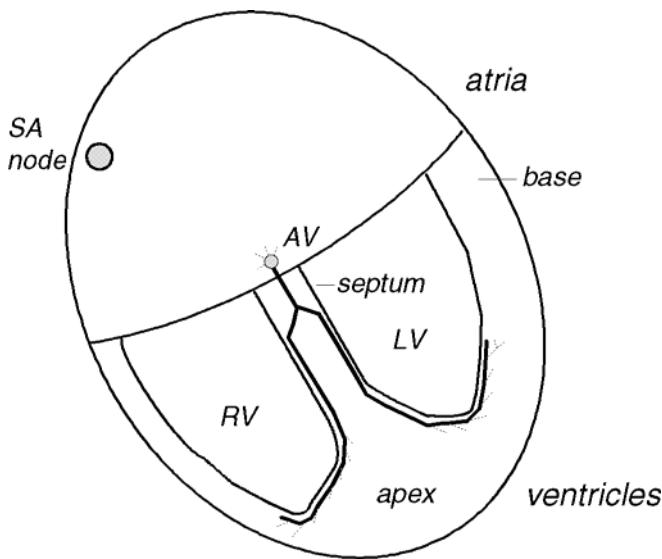
3. Electrical refractory period long; thus mechanical contraction cannot summate

NORMAL SEQUENCE OF CARDIAC ACTIVATION

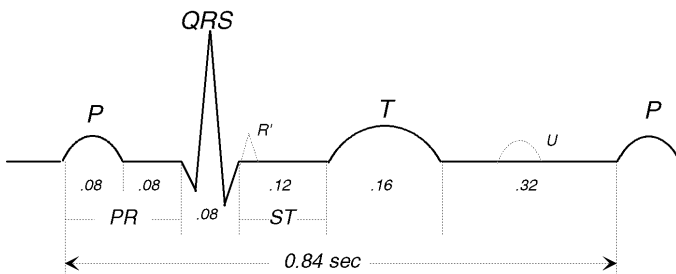
- A. SA node self excitation: pacemaker  
(normally, inherently the most rapid)
- B. Spread of depolarization through the atrium (mainly passive, may involve specialized rapidly conducting fibers to AV node) (ECG P wave)
- C. Conducted excitation through the AV node (slow)
- D. Conducted excitation down the ventricular conduction system: bundle of His (Purkinje fibers); first common bundle, then left & right bundles
- E. Excitation of the ventricular myocardium in the following order (ECG QRS complex)
  - 1. intraventricular septum and papillary muscles
  - 2. ventricular apex
  - 3. base

Note: atrial repolarization occurs during ventricular depolarization

- F. Repolarization of the ventricles (not conducted) (ECG T wave)



- G. This gives rise to the typical electrocardiogram (ECG, EKG)



**P wave:** Atria depolarizing  
**P-R segment** (end of P to start of QRS): Depolarization traveling through the AV node and ventricular rapid conduction system  
**QRS complex:** Ventricles depolarizing (& atria repolarizing)  
**S-T segment:** Ventricles depolarized (& atria repolarized)  
**T wave:** Ventricles repolarizing