EMC 451

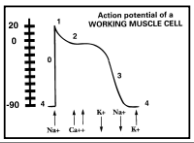
Advanced ECG Interpretation

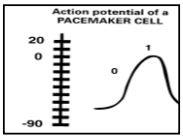
Unit 12
Electrophysiology, Arrhythmiagenesis, and Drug Effects

Unit Objectives

- **Upon completion of this unit, you should be able to:**
  - Describe the normal physiology of electrical conduction of working and pacemaker cells.
  - Describe the role of resting membrane potential, threshold potential, and slope of diastolic depolarization on heart rate and arrhythmiagenesis.
  - Discuss the effects of acetylcholine and epinephrine on resting membrane potential and threshold potential.
  - Describe the effect of ischemia on myocardial depolarization.
  - Describe the enhanced automaticity mechanism of arrhythmiagenesis.
  - Describe retrograde conduction and circus movement mechanisms of re-entrant arrhythmias.
  - Describe the mechanisms of arrhythmia suppression provided by lidocaine.
  - Describe the EKG findings associated with Quinidine, Digitalis, and TCA toxicity.

Normal Action Potential

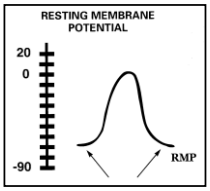


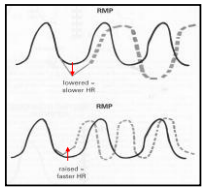


- Working cells can't depolarize themselves. They must be stimulated to fire. This electrical stimulation alters the cell's permeability to sodium.
- Normal resting potential is -90 mv (Phase 4)
- As sodium enters the cell through the fast channel, cell becomes more positive, or "fires" (depolarizes) (Phase 0)
- Sodium channel is closed and calcium channel opens allowing calcium enters the cell (Phase 2)
- Potassium leaks out of the cell (Phase 3)
- Near the bottom of Phase 3, sodium is pumped out of the cell and potassium returns.
- When this is completed, the cell has returned to its resting potential (Phase 4).

- Pacemaker cells have the property of automaticity, or the ability to fire themselves.
- Pacemaker cells have a slightly different action potential.
- The normal resting membrane potential is –70 mv.
- The action potential uses calcium rather than sodium to initiate the impulse.
- As potassium leaks out there is a slow upward-sloping phase 0.
- Changes in the normal resting membrane potential change the rate of firing and may also lead to arrhythmias.

Resting Membrane Potential

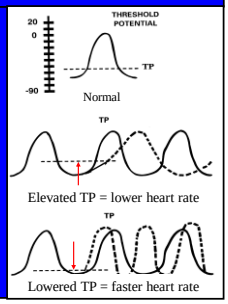




- The normal resting membrane potential is –70 mv, which is less negative than the working cell.
- Because the RMP is normally less negative, it takes less time to depolarize.
- If RMP is lowered, it takes longer to reach threshold and depolarize and thus, heart rate decreases.
- If RMP is raised, it takes less time to reach threshold, meaning that the next impulse arrives earlier, leading to an increase in heart rate.

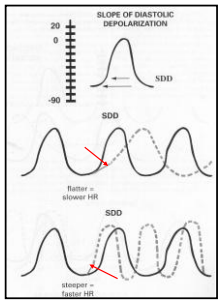
Threshold Potential

- If threshold potential is raised, it takes longer for the action potential to reach threshold, and thus, heart rate is slower.
- If threshold potential is lowered, the action potential reaches threshold sooner and heart rate increases.



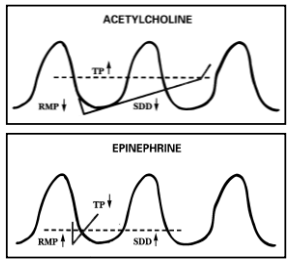
Slope of Diastolic Depolarization

- Slope of diastolic depolarization begins at the most negative point of the action potential (Phase 4), and extends until it reaches the firing threshold. This is the phase 0 slope when potassium is slowly leaking from the cell.
- If the angle of SDD is flattened, it takes longer for the action potential to reach threshold, and the heart rate slows.
- If the angle of SDD is made steeper, threshold is reached sooner and heart rate increases.



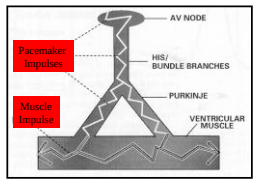
Drugs and Heart Rate

- 3 methods for altering heart rate
  1. Alter threshold potential
  2. Alter resting membrane potential
  3. Alter slope of diastolic depolarization



Arrhythmiagenesis

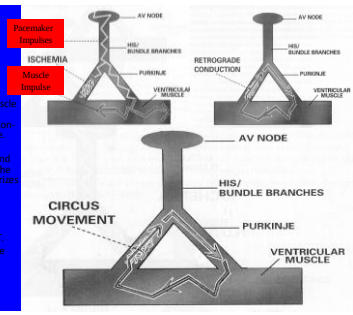
- Pacemaker cells depolarize by slow channel calcium conduction
- When these impulses reach the muscle wall, the electrical stimulus alters the cell permeability of the muscle cells, allowing shifting of sodium and potassium, resulting in a muscle cell depolarization.



Arrhythmiagenesis

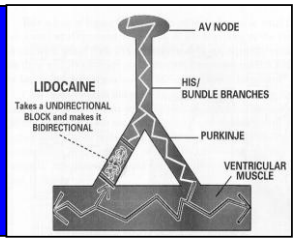
2 Methods of Arrhythmias

1. Enhanced Automaticity
2. Re-entry
   - Impulse travels down one branch of purkinje fiber normally, the other side is blocked by ischemia
   - As impulse depolarizes muscle fiber, the impulse travels retrogradely through the non-depolarized ischemic tissue.
   - If that tissue is polarized (resting), it is depolarized and the impulse travels down the normal branch and depolarizes the muscle wall again.
   - If this happens once, it generates a PVC.
   - If it sets up a cycle (circus movement) it generates VT.
   - Similar mechanisms may be responsible for atrial tachycardias



Lidocaine

- Lidocaine increases the degree of block in ischemic tissue so that it converts a unidirectional block in a bidirectional block, thus eliminating the retrograde conduction arm.
- Lidocaine accomplishes this by lowering the resting membrane potential, flattening the slope of diastolic depolarization, and raises the threshold potential.
- This effects also treat arrhythmias caused by enhanced automaticity, as well as re-entry.



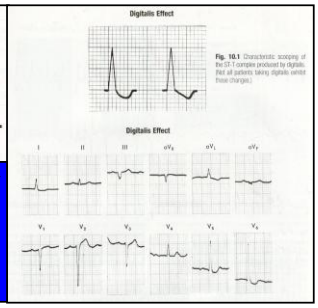
Digitalis

- Cardiac Glycoside – most common drug used is digoxin, but lanoxin is also a digitalis preparation
- Has 2 effects:
  - Mechanical
    - Used to increase strength of contraction in patients with CHF
  - Electrical
    - Increases vagal tone in AV node, thus slowing conduction through the AV node
    - Used to control ventricular rate associated with AF and PSVT
- Very narrow therapeutic range
- S&S of Digitalis Toxicity
  - Weakness
  - Anorexia
  - N&V
  - Altered color perception
  - Cardiac dysrhythmias
    - PVCs
    - VT/VF
    - AV block
    - Junctional
    - AT with block
    - Junctional tachycardia

Digitalis

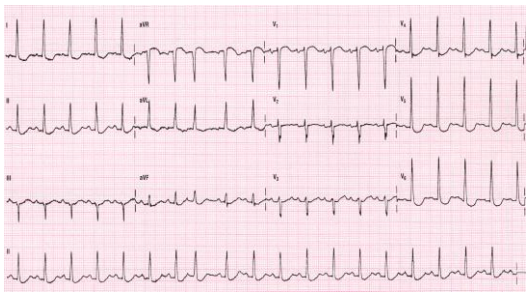
- Predisposing Factors
  - Hypokalemia
  - Hypomagnesemia
  - Hypercalcemia
  - Hypoxemia
  - Chronic lung disease
  - Myocardial infarction
  - Advanced age
  - Renal insufficiency
  - Hypothyroidism
  - WPW syndrome and AF

ECG Effects of Digitalis



- Causes scooping of ST-T complex
- This EKG effect does not imply digitalis toxicity

ECG Effects of Digitalis

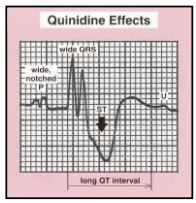


Treatment of Digitalis Toxicity

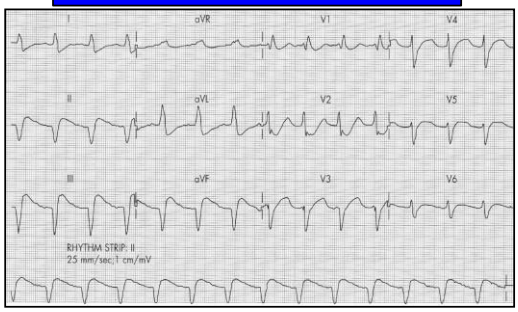
- **Treat dysrhythmias**
- **Cardioversion is very dangerous and may result in fatal VT/VF**
- **Use of quinidine, verapamil, and amiodarone increases digoxin level.**
- **Digitalis toxicity may cause hyperkalemia which must be treated**
- **Severe toxicity may require administration of Digibind, a digitalis-binding antibody**

Quinidine

- Has its effects on the sodium and potassium channels
- Slows depolarization and repolarization through both the atria and ventricles
- EKG effects
  - Wide, notched P wave
  - Widened QRS complex
  - Prolonged QT interval
  - U waves
- May result in Torsades de Pointes



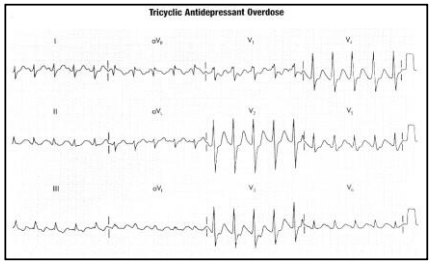
Quinidine



Tricyclic Antidepressants

- Responsible for more drug overdose deaths than any other prescribed medication
- Mortality rate is 5% and is related to cardiotoxicity
- Drugs
  - Amitriptyline
  - Amoxapine
  - Desipramine
  - Doxepin
  - Nortriptyline
- EKG effects
  - Mainly result of sodium channel blockade and potassium channel antagonism
  - Tachycardia due to anticholingergic effect
  - Widened QRS complex
  - Prolonged QT interval from potassium channel effect
  - Prolonged PR interval
  - Bradycardia signifies profound sodium channel blockade
  - Heart block
  - Torsades de Pointes

Tricyclic Antidepressants



Management of Tricyclic Antidepressants

- GI Decontamination
  - NG tube with gastric lavage
  - Activated charcoal
  - No syrup ipecac
- Manage dysrhythmias
- IV hydration
- Sodium bicarbonate
  - Initial 1-2 mEq/kg bolus
  - Infusion (@ amps in 1 L of D5W of ½ NS) to run at 2 ml/kg/hr