

EMC 451

Advanced ECG Interpretation

Unit 12 Electrophysiology, Arrhythmogenesis, and Drug Effects

EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

1

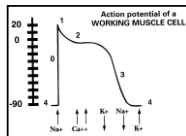
Unit Objectives

- 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 arrhythmogenesis.
 - 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 arrhythmogenesis.
 - 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.

EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

2

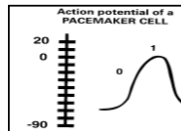
Normal Action Potential



- 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 2).
- 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).

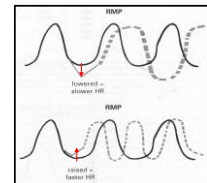
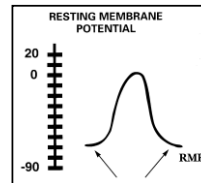
EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

3



- 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



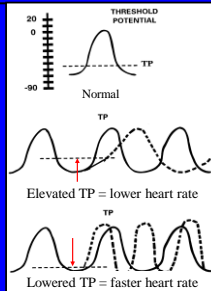
- The normal resting membrane potential is -90 mV, which is less negative than the working cell.
- Because the RMP is normally less negative, it takes less time to depolarize.
- If RMP is lowered, it takes longer to reach threshold and depolarize and thus, heart rate decreases.
- If RMP is raised, it takes less time to reach threshold, meaning that the next impulse arrives earlier, leading to an increase in heart rate.

EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

4

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.

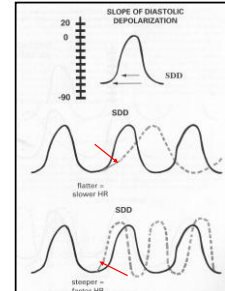


EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

5

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.



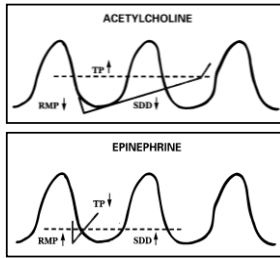
EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

6

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

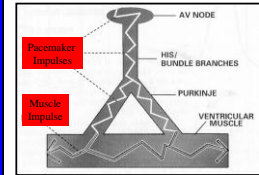


EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

7

Arrhythmogenesis

- 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.



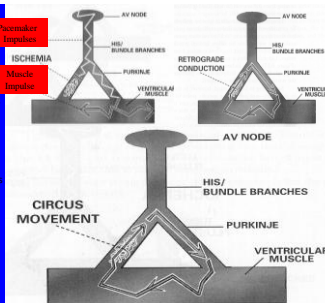
EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

8

Arrhythmogenesis

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 (corus movement) it generates VT.
- Similar mechanisms may be responsible for atrial tachycardias

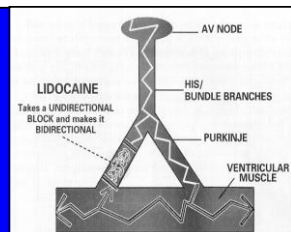


EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

9

Lidocaine

- Lidocaine increases the degree of block in ischemic tissue so that it converts a unidirectional block to 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.



EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

10

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

EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

11

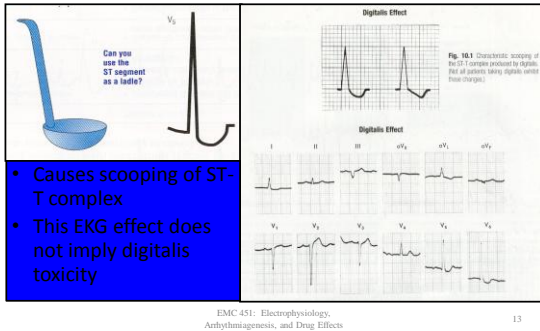
Digitalis

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

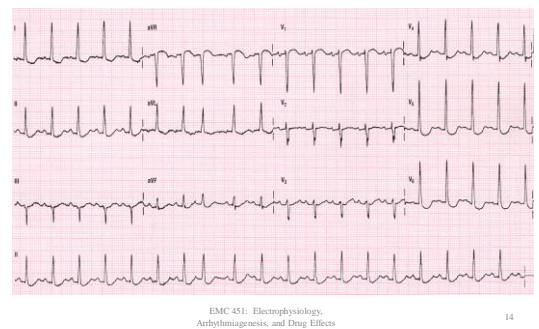
EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

12

ECG Effects of Digitalis



ECG Effects of Digitalis



Treatment of Digitalis Toxicity

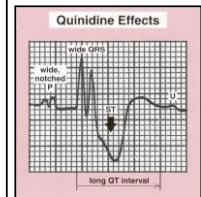
- Treat dysrhythmias
- Cardioversion is very dangerous and may result in fatal VT/VF
- Use of quinidine, verapamil, and amiodarone increases digoxin level.
- Digitalis toxicity may cause hyperkalemia which must be treated
- Severe toxicity may require administration of Digibind, a digitalis-binding antibody

EMC 451: Electrophysiology, Arrhythmogenesis, and Drug Effects

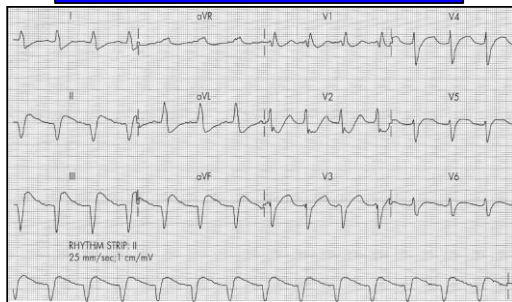
15

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



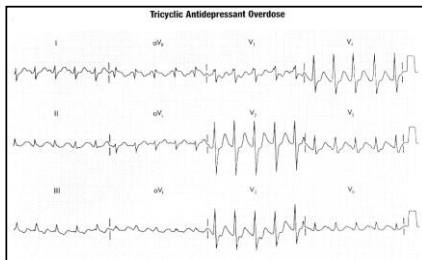
Tricyclic Antidepressants

- 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 anticholinergic 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

EMC 451: Electrophysiology, Arrhythmogenesis, and Drug Effects

18

Tricyclic Antidepressants



EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

19

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

EMC 451: Electrophysiology,
Arrhythmogenesis, and Drug Effects

20