Chapter 26

Drugs for Dysrhythmias

Dysrhythmias

- Abnormalities of electrical conduction or rhythm in heart
- Also known as arrhythmias
- Can range from harmless to life threatening

Diagnosis/Treatment of Dysrhythmias

- Electrocardiogram (ECG)
- Appropriate diagnosis and pharmacotherapy essential

Frequency in Population Difficult to Predict

- Symptoms range from none to sudden death
- Still, dysrhythmias estimated to be quite common
- Persistent/severe dysrhythmias increase risk of stroke and heart failure

Flow of Electrical Impulses through Normal Heart

- Review the property of automaticity
  - Ability to initiate an action potential
- Sinoatrial (SA) node—pacemaker
- Spreads across both atria
- Atrioventricular (AV) node
Flow of Electrical Impulses through the Normal Heart

- Atrioventricular bundle (bundle of His)
- Right and left bundle branches
- Purkinje fibers
- Both ventricles

Conduction System

- Purpose is to regulate heat and maintain cardiac output

Occurrence of Dysrhythmias

- Can occur in both healthy and diseased hearts
- Disrupt regulation of heart
- May decrease cardiac output
- Closely associated with certain conditions
  - Heart disease
  - Myocardial infarction

Classification of Dysrhythmias

- Location
  - Atrial or ventricular
- Type
  - Flutter, fibrillation, block
  - Atrial fibrillation most common

Types of Dysrhythmia

- Premature atrial or premature ventricular contractions (PVCs)
- Atrial or ventricular tachycardia
- Atrial or ventricular flutter and/or fibrillation
- Sinus bradycardia
- Heart block
Action Potentials

- Electrical impulses across myocardium
- Found in neural and cardiac cells
- Created by changes in extra- and intracellular ion polarization
- Cell membrane at rest = polarized
  - Negative membrane potential
  - Na\(^+\) and Ca\(^{++}\) outside cell
  - K\(^+\) inside cell

Generation of Action Potential

- Sodium-ion channels open
  - Sodium ions rush in, cause depolarization
- Calcium-ion channels open
  - Calcium ions enter cell, stimulate cardiac-muscle contraction
  - SA and AV cells depolarize in response to calcium-ion influx

Repolarization

- Return to polarized state
- Sodium pump removes Na\(^+\)
- Potassium-ion channels allow K\(^+\) to move back into cell

Pharmacological Strategies to Terminate Dysrhythmias

- Block potassium-, sodium-, or calcium-ion channels
- Prolong refractory period
  - Brief period in conduction cycle when myocardial cells cannot produce another action potential

Nonpharmacological Therapies for Dysrhythmias

- Cardioversion and defibrillation
  - Serious types of dysrhythmias
  - Electrical shock stops all electrical impulses in heart and allows SA node to regain control
Nonpharmacological Therapies for Dysrhythmias (continued)

• Catheter ablation—identify and destroy aberrant cardiac cells that cause dysrhythmias
• Cardiac pacemaker—paces heart at set rate
• Implantable cardioverter defibrillator (ICD)—combination of pacemaker and defibrillator

Antidysrhythmic Drugs

• Primary Mechanisms of Action
  – Blocking conduction (flow of ions)
  – Altering automaticity (autonomic activity)
• Use is declining significantly
  – Can worsen or create new dysrhythmias
  – Nonpharmacological therapy is improving

Antidysrhythmic Drug Groups

• Five groups
  – Class I: sodium-ion-channel blockers
    • Block sodium-ion channels and suppress ectopic activity
  – Class II: beta-adrenergic antagonists
    • Slow heart rate, decrease conduction velocity through AV node

Antidysrhythmic Drug Groups (continued)

  – Class III: potassium-ion-channel blockers
    • Delay repolarization and lengthens refractory period—stabilizes dysrhythmias
  – Class IV: calcium-ion-channel blockers
    • Reduces automaticity in SA node and slows impulse conduction through AV node; slows heart rate
  – Miscellaneous antidysrhythmic drugs: Slow conduction through AV node and/or decrease automaticity of SA node

Role of Nurse

• Careful monitoring
• Providing education
• Obtaining vital signs, medical and drug history

Sodium-Ion-Channel Blockers

• Monitor ECG for changes
• Monitor for hypotension, changes in level of consciousness, diarrhea
• Teach client to take medication as directed and to avoid alcohol
• Contraindicated when clients have heart failure or renal impairment
Beta-adrenergic Blockers

- Contraindicated in clients with heart block, severe bradycardia, AV block, asthma
- Monitor for hypotension and hypoglycemia
- Elderly clients may show signs of cognitive impairment
- Teach clients to measure heart rate, to rise slowly, to report signs of heart failure

Potassium-Ion-Channel Blockers

- Use cautiously in clients with heart block
- Do not use during pregnancy (category C) or lactation
- Monitor for vision changes, palpitations, jaundice, abdominal pain
- Avoid sun exposure; take with food

Calcium-Ion-Channel Blockers

- Do not use for clients with sick-sinus syndrome, heart block, severe hypotension, cardiogenic shock, or congestive heart failure
- Monitor for hypotension, especially in elderly

Calcium-Ion-Channel Blockers

- Do not use during pregnancy (category C) or lactation
- Report palpitations, blood-pressure changes, edema, shortness of breath

Miscellaneous Drugs for Dysrhythmias

- Monitor heart rate and blood pressure.
- Report symptoms of digoxin toxicity

Sodium-Ion-Channel Blockers (Class I)

- Prototype drug: procainamide (Pronestyl)
- Mechanism of action: to block sodium-ion channels, which slows rate of impulse conduction across heart
Sodium-Ion-Channel Blockers (Class I)

- **Primary use:** to correct atrial and ventricular dysrhythmias
- **Adverse effects:** creates new dysrhythmias or worsens existing ones
  - Lupus effect, nausea, vomiting, abdominal pain, headache
  - High doses can produce CNS effects

Beta-Adrenergic Blockers (Class II)

- **Prototype drug:** propranolol (Inderal)
- **Mechanism of action:** to block beta receptors, which reduces automaticity and slows conduction velocity across myocardium
- **Primary use:** to treat atrial dysrhythmias associated with heart failure
- **Adverse effects:** bradycardia, hypotension with dizziness and fainting
  - Bronchospasms, hypoglycemia, diminished libido

Potassium-Ion-Channel Blockers (Class III)

- **Prototype drug:** amiodarone (Cordarone)
- **Mechanism of action:** to block potassium-ion channels in myocardial cells, which prolongs refractory period of heart
- **Primary use:** to treat resistant ventricular tachycardia, atrial dysrhythmias with heart failure
- **Adverse effects:** blurred vision, pneumonia-like syndrome, bradycardia, hypotension
  - Can create new dysrhythmias or worsen existing ones

Calcium-Ion-Channel Blockers (Class IV)

- **Prototype drug:** verapamil (Calan)
- **Mechanism of action:** to block calcium-ion channels, which reduces automaticity and slows myocardial (AV) conduction velocity
- **Primary use:** to treat supraventricular tachycardia
- **Adverse effects:** bradycardia, hypotension, headache
Miscellaneous Antidysrhythmics

- **Examples:** digoxin (Lanoxin) and adenosine (Adenocard)
- **Mechanism of action:** to decrease automaticity of SA node and slow conduction through AV node but not act by blocking ion channels

Primary use:
- for digoxin—certain types of atrial dysrrhythmias; for adenosine—serious atrial tachycardia
- **Adverse effects:** creates new dysrhythmias or worsens existing ones
  - Digoxin: nausea, vomiting, headache, visual disturbances
  - Adenosine: facial flushing, dyspnea

Sodium-Ion-Channel Blockers (Class I)

- **Largest group of antidysrhythmics**
- **Mechanism of action:** to block sodium-ion channels
  - Slows rate of impulse conduction across heart
- **Primary use:** to correct atrial and ventricular dysrhythmias
- **Examples:** procainamide (Pronestyl), quinidine sulfate (Quinidex), and tocainide (Tonocard)

Beta-adrenergic Blockers (Class II)

- **Mechanism of action:** to block beta receptors
  - Reduce automaticity and slows conduction velocity across myocardium
- **Primary use:** to treat atrial dysrhythmias associated with heart failure
- **Examples:** propranolol (Inderal), esmolol (Brevibloc), acebutolol (Sectral)

Potassium-Ion-Channel Blockers (Class III)

- **Mechanism of action:** to block potassium-ion channels in myocardial cells
  - Prolongs refractory period of heart
- **Primary use:** to treat resistant ventricular tachycardia, atrial dysrhythmias with heart failure
- **Examples:** amiodarone (Cordarone), dofetilide (Tikosyn), and Ibutilide (Corvert)

Calcium-Ion-Channel Blockers (Class IV)

- **Mechanism of action:** to block calcium-ion channels
  - Reduces automaticity and slows myocardial (AV) conduction velocity
- **Primary use:** to treat supraventricular tachycardia
- **Examples:** verapamil (Calan) and diltiazem (Cardizem)
Miscellaneous antidysrhythmics

- **Examples:** digoxin (Lanoxin) and adenosine (Adenocard)
- **Mechanism of action:** to decrease automaticity of SA node and slow conduction through the AV node
  - Do not act by blocking ion channels
- **Primary use:**
  - Digoxin (Lanoxin): to treat certain types of atrial dysrhythmias
  - Adenosine (Adenocard): to treat serious atrial tachycardia

Drug Therapy for Dysrhythmias

- **Assessment**
  - Complete health history
  - Assessment of cardiac output
  - Baseline ECG to compare throughout therapy

- **Nursing diagnoses**
  - Ineffective tissue perfusion
  - Knowledge deficit
  - Risk for injury
  - Decreased cardiac output

- **Planning**
  - Improved cardiac output
  - Understanding of drug therapy
  - Prevention of adverse effects

- **Implementation**
  - Monitor cardiac rate and rhythm
  - Monitor IV site
  - Investigate possible causes of dysrhythmia
  - Observe for correct administration of drugs and adverse effects

- **Evaluation**
  - Ideal outcome criteria
  - Improved cardiac output
  - Client verbalization of understanding of drug therapy
  - Prevention of adverse effects