Chapter 9
Cardiac Arrhythmias

Learning Objectives

- Define electrical therapy
- Explain why electrical therapy is preferred initial therapy over drug administration for cardiac arrest and some arrhythmias
- Discuss medications used to treat symptomatic bradycardia: atropine sulfate, epinephrine, and dopamine

Learning Objectives

- Discuss adenosine (Adenocard) and its role in treatment of supraventricular tachycardia
- List and discuss three classes of antiarrhythmic agents
Learning Objectives

- Discuss the beta blocker atenolol (Tenormin), anticoagulant warfarin (Coumadin), and calcium channel blockers diltiazem (Cardizem) and verapamil (Isoptin)

- Explain benefits of beta blockers in treatment of arrhythmias

Learning Objectives

- Discuss the following ventricular antiarrhythmic agents:
  - Amiodarone (Cordarone)
  - Lidocaine (Xylocaine)
  - Procainamide (Pronestyl)
  - Magnesium sulfate

- Discuss medications used to treat pulseless electrical activity and asystole: epinephrine and vasopressin

Introduction

- Cardiac arrhythmia
  - Loss of abnormality of cardiac rhythms
  - Chronic atrial fibrillation
  - Cardiac arrest caused by ventricular fibrillation
Basic Electrophysiology

- Heart has four small pumps/chambers
  - Each has millions of cardiac muscle cells
  - Each can contract independently
- For the heart to pump blood effectively, muscle cells and four chambers must coordinate

Basic Electrophysiology

- Muscle cells composing the four chambers must contract at precise moments
  - Coordinated by the heart’s electrical system
- Electrical impulse originates in SA node and travels through heart’s electrical system in a predictable way
  - As it moves through the heart, it signals each portion of the heart to contract in required organized fashion

Basic Electrophysiology

- When electrical impulses coordinating cardiac contraction are too slow, too fast, or irregular:
  - Heart chambers do not contract in an organized fashion
  - Amount of blood pumped by the heart drops
  - BP falls
Overview of Arrhythmias

- Sinus bradycardia
  - Cardiac output (CO) is determined by patient’s heart rate (HR) x amount of blood pumped with each heartbeat (stroke volume [SV])
    - \[ CO = HR \times SV \]
  - Decrease in heart rate causes drop in cardiac output
    - Reduced blood flow to brain
    - Dizziness and syncope

Overview of Arrhythmias

- Ventricular fibrillation (VF)
  - Cardiac rhythm when all the cardiac cells are trying to contract independently and not coordinating or communicating with each other
  - Electrical therapy resets the heart
  - Cardiac arrest is usually caused by sudden onset of cardiac arrhythmia
  - VF occurs from chronic myocardial ischemia resulting in electrical instability of the heart
  - Electrical defibrillation cardiac arrest is single best method of treatment
Overview of Arrhythmias

Management

Bradycardia
- Heart rate less than 60 beats/min
- Ensure patent airway
- Provide supplemental O₂
- Monitor cardiac rhythm
- Evaluate O₂ saturation and BP
- Place IV line
- Causes decreased cardiac output

Management

Bradycardia
- Symptoms:
  - Decreased mental status
  - Syncope
  - Hypotension
  - Chest pain
  - Congestive heart failure
  - Dyspnea
  - Seizures
  - Shock
Management

- Bradycardia
  - Treatment
    - Transcutaneous pacing
      - Start external pacing for type II second-degree blocks and third-degree blocks
    - Atropine 0.5 mg IV
    - Epinephrine 2 to 10 mcg/min or dopamine 2 to 10 mcg/kg/min infusions may be used while waiting to start pacing line or if pacing is ineffective
      - Are beta-adrenergic drugs
      - Chronotropic, which results in increased heart rate

Management

- Tachycardia
  - In young patients with SVT, vagal maneuvers and carotid massage can be used to return patient to normal sinus rhythm
    - Carotid massage should never be used in older persons
    - Valsalva maneuver can be used in older persons

Management

- Tachycardia
  - Synchronized cardioversion is indicated if patient is unstable
    - Altered mental status
    - Hypotension
    - Chest pain
Management

- Tachycardia
  - Adenosine (Adenocard) is used for stable patients in SVT
    - Alters movement of potassium in action potential of the heart
    - Creates short period of asystole or ventricular escape beats
    - Only used as antiarrhythmic for SVT
    - Has no effect on ventricular arrhythmias
    - Ultrashort half-life of 5 to 20 sec

Management

- Tachycardia
  - Works only when majority of administered doses reaches the heart quickly
    - Given by rapid IV push, immediately followed by flush solution
    - AHA recommended dose: 6 mg IV, followed by normal saline bolus of 20 mL
    - Extremity should be elevated
  - If SVT does not resolve within 1 to 2 min, dose should be increased to 12 mg and repeated no more than twice
Management

- Antiarrhythmic drugs
  - Calcium channel blockers
    - Block influx of calcium into cardiac cells and arterial smooth muscle cells
    - Slows conduction velocity of cardiac action potential
    - Prolongs period of repolarization
    - Rapid electrical impulses traveling down from atria to ventricles through AV node are slowed

- Calcium channel blockers
  - Cause peripheral arterioles to dilate
  - BP to be monitored closely
  - Clinical applications
    - Useful for controlling and/or converting certain supraventricular arrhythmias
    - Stable, narrow-complex reentry SVT
    - Automatic focus tachycardias not converted or controlled by adenosine or vagal maneuvers

- Do not convert atrial fibrillation or atrial flutter into sinus rhythm
- Used to:
  - Treat hypertension
  - Decrease angina episodes
  - Decrease incidence of migraine headaches
Management

- Antiarrhythmic drugs
  - Calcium channel blockers
    - Various calcium channel blockers have different proportions of vasodilator, antihypertensive, and antiarrhythmic effects
    - Diltiazem (Cardizem)
      - Used for acute ventricular rate control and management of hypertension
    - Verapamil (Isoptin)
      - No longer routinely used for treating arrhythmias
      - Prescribed for BP control or continued rate control in chronic atrial fibrillation
      - Do not administer to patients with heart failure or impaired ventricular function
  - Beta blockers
    - Exert effects on both beta_1 and beta_2 receptors
    - Beta_1 receptors
      - Located in the heart
      - Act as main mediator of rate and contractility
    - Lower BP and O_2 consumption
    - Effects result in lower heart rate, automaticity, and conduction
    - Can dramatically decrease cardiac output
    - Beta_2 blockers prevent vasoconstriction and lower BP
Management

- Antiarrhythmic drugs
  - Beta blockers
    - Highly effective for:
      - Angina
      - Hypertension
      - Decreased mortality rate during and after AMI

- Decrease infarct size
- Reduce risk for recurrent ischemia
- Decrease incidence of sudden death from arrhythmias
- Useful for treatment of dissecting aortic aneurysms
- Can be used as antiarrhythmics, especially in high catecholamine or epinephrine states such as:
  - Alcohol withdrawal
  - Panic attacks
  - Hyperthyroidism

Management

- Antiarrhythmic drugs
  - Beta blockers
    - Control heart rate in SVTs
    - Decrease AV nodal conduction
    - Depress ventricular automaticity
    - May be effective in rate control of atrial fibrillation, SVT, and sinus tachycardia
    - Suppress premature ventricular contractions, especially in patients with angina
Management

- Antiarrhythmic drugs
  - Beta blockers
    - Propranolol (Inderal)
    - Metoprolol (Lopressor)
    - Atenolol (Tenormin)
    - Esmolol (Brevibloc)

Management

- Ventricular antiarrhythmic drugs
  - Divided into classes based on their mechanism of action:
    - Class I
      - Sodium channel blockers
    - Class II
      - Beta blockers
    - Class III
      - Potassium channel blocking agents
    - Class IV
      - Calcium channel blocking agents

Management

- Ventricular antiarrhythmic drugs

  - Amiodarone
    - Only antiarrhythmic drug that has actions from every class of antiarrhythmics
    - Used to treat arrhythmias that originate both above and below AV node
    - Used for cardiac arrest from VF and pulseless ventricular tachycardia
    - Converts acute atrial fibrillation or atrial flutter
Ventricular antiarrhythmic drugs

- Amiodarone
  - Increases duration of action potential
  - Increases refractory period of atria, AV node, and ventricular tissues
  - Reduces risk for developing atrial fibrillation, can convert from atrial fibrillation to sinus rhythm
Management

- Ventricular antiarrhythmic drugs
  - Amiodarone
    * Increasing duration of action potential and refractory period:
      * Specialized tissue that conducts electrical impulses or action potential from SA node through ventricles, past SA node and down into the ventricles
      * The faster the action potential duration, the faster the action potential will move down the conduction system
      * The greater the refractory period, the fewer action potentials will travel down through the heart in 1 min

- Ventricular antiarrhythmic drugs
  - Lidocaine
    * Class 1B antiarrhythmic
    * Blocks sodium channel
    * Decreases amount of time required for repolarization
    * Short-acting anesthetic
    * Converts only about 11% to 12% of cases of VT

- Ventricular antiarrhythmic drugs
  - Procainamide (Pronestyl)
    * Class IA antiarrhythmic
    * Binds to fast sodium channels
    * Slows almost all phases of action potential
    * Slows depolarization, repolarization, and impulse conduction
    * Can prolong PR and QT intervals
Management

- Ventricular antiarrhythmic drugs
  - Procainamide (Pronestyl)
    - At higher doses, can cause QRS complex to widen
    - Can be used for both atrial and ventricular arrhythmias
    - Used as second-line agent of choice for atrial fibrillation, atrial flutter, and SVT
    - Usually drug of choice when amiodarone is not effective
    - Cannot be given rapidly
    - Takes 20 to 40 min to work

Management

- Ventricular antiarrhythmic drugs
  - Magnesium sulfate
    - Electrolyte
    - Extremely effective antiarrhythmic
    - Magnesium is used to treat torsades de pointes
    - Decreases PVC caused by a prolonged QT interval
    - Used in alcoholic and malnourished patients

Management

- Cardiopulmonary arrest
  - Pulseless electrical activity (PEA) and asystole
    - PEA has detectable electrical activity on the monitor but no mechanical cardiac activity
    - Detected by the presence of a pulse or audible heart tones
    - Asystole has no electrical or mechanical cardiac activity
    - Only epinephrine and vasopressin are indicated
Management

- Cardiopulmonary arrest
  - PEA and asystole
    - Epinephrine
      - In arrest: 1 mg IV, IO administered every 3 to 5 min
      - In beta or calcium channel blocker overdose: up to 0.2 mg/kg
      - When cannot establish IV or IO access, administer down ET tube: 2 to 2.5 mg

Management

- Cardiopulmonary arrest
  - PEA and asystole
    - Vasopressin
      - Vasoconstrictor that does not use adrenergic receptors
      - Powerful inotropic and chronotropic
      - Stimulates the heart via different receptor mechanism
      - In cardiac arrest, single dose of 40 units IV or IO

Management

- Cardiopulmonary arrest
  - Pulseless ventricular tachycardia and ventricular fibrillation
    - Epinephrine and vasopressin are indicated
    - Electrical therapy has proven to be most effective treatment of cardiac arrest with these rhythms
    - If patient does not respond to defibrillation, epinephrine or vasopressin, consider amiodarone
    - Lidocaine can be used as substitute
Questions?