Chapter 22
Cardiovascular Disorders

Learning Objectives
- Describe the epidemiology of cardiovascular disease
- Identify risk factors most predisposing to coronary heart disease
- Discuss prevention strategies that may reduce morbidity and mortality rates from coronary heart disease

Learning Objectives (Cont'd)
- Identify the major structures of the vascular system
- Describe heart anatomy, including position in thoracic cavity, heart layers, heart chambers, location and function of cardiac valves
- Identify normal characteristics of point of maximal impulse
Learning Objectives (Cont'd)

- Identify phases of the cardiac cycle
- Identify arterial blood supply to areas of the myocardium
- Compare coronary arterial distribution with major portions of the cardiac conduction system

Learning Objectives (Cont’d)

- Identify structures of ANS, effect on heart rate, rhythm, contractility
- Define and give examples of positive, negative inotropism, chronotropism, dromotropism
- Identify and define components of cardiac output, factors affecting venous return

Learning Objectives (Cont’d)

- Define preload, afterload, left ventricular end-diastolic pressure; relate each to heart failure pathophysiology
- Describe the clinical significance of Starling’s law
- Define the functional properties of the cardiac muscle
Learning Objectives (Cont'd)

- Describe events involved in steps from excitation to contraction of cardiac muscle fibers
- Define events composing cardiac action potential

Learning Objectives (Cont'd)

- Correlate electrophysiological and hemodynamic events occurring throughout the entire cardiac cycle with various ECG waveforms, segments, intervals
- Identify the structure and course of all divisions, subdivisions of the cardiac conduction system

Learning Objectives (Cont'd)

- Identify and describe how the heart’s pacemaking control, rate, rhythm are determined
- Explain the physiological basis of conduction delay in AV node
- Differentiate primary mechanisms responsible for producing cardiac dysrhythmias
Learning Objectives (Cont'd)

- Describe reentry
- Explain the purpose of ECG monitoring
- Identify limitations of an ECG
- Relate cardiac surfaces/areas represented by ECG leads

Learning Objectives (Cont'd)

- Describe the correct anatomic placement of chest leads
- Identify how heart rates, durations, amplitudes are determined from ECG recordings
- Describe how ECG waveforms are produced

Learning Objectives (Cont'd)

- Recognize changes on ECG that reflect myocardial ischemia, injury
- Describe systematic approach to analysis, interpretation of cardiac dysrhythmias
- Describe ECG characteristics, possible causes, signs and symptoms, initial emergency care for dysrhythmias originating in the sinus node
Learning Objectives (Cont'd)

- Describe ECG characteristics, possible causes, signs and symptoms, initial emergency care for dysrhythmias originating in the atria
- Describe aberrant conduction
- Define synchronized cardioversion, discuss indications, methods for this procedure

Learning Objectives (Cont’d)

- Describe the significance of accessory pathways
- Describe ECG characteristics, possible causes, signs and symptoms, initial emergency care for dysrhythmias originating in the atrioventricular junction

Learning Objectives (Cont’d)

- Describe electrocardiographic characteristics, possible causes, signs and symptoms, initial emergency care for dysrhythmias originating in ventricles
- Describe conditions of pulseless electrical activity
- Describe the process and pitfalls in the differentiation of wide QRS complex tachycardias
Learning Objectives (Cont'd)

- Describe dysrhythmias seen in cardiac arrest
- Define defibrillation, discuss indications, methods for procedure
- Describe electrocardiographic characteristics, possible causes, signs and symptoms, initial emergency care of AV block

Learning Objectives (Cont'd)

- Describe characteristics of the implanted pacemaking system
- List causes, implications of pacemaker failure
- Recognize complications of artificial pacemakers on ECG

Learning Objectives (Cont'd)

- Identify additional hazards that interfere with artificial pacemaker function
- Describe artifacts that may cause confusion when evaluating ECG of patient with pacemaker
- Describe components, functions of transcutaneous pacing system
Learning Objectives (Cont’d)

• Identify indications for transcutaneous cardiac pacing
• Describe technique of applying transcutaneous pacing system
• Explain what each setting, indicator on transcutaneous pacing system represents, how settings may be adjusted

Learning Objectives (Cont’d)

• List possible complications of transcutaneous pacing
• Based on pathophysiology, clinical evaluation of patient with suspected acute myocardial infarction (MI), list anticipated clinical problems according to life-threatening potential

Learning Objectives (Cont’d)

• Identify ECG changes characteristically seen during evolution of acute MI
• Recognize limitations of ECG in reflecting evidence of myocardial ischemia, injury
• Describe abnormalities originating within the bundle branch system
Learning Objectives (Cont'd)

- Identify ECG changes characteristically produced by electrolyte imbalances
- Identify and describe components of the focused history of a patient with cardiovascular compromise
- Identify OPQRST of chest pain assessment

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Learning Objectives (Cont'd)

- Explain the clinical significance of paroxysmal nocturnal dyspnea
- Identify patient situations for which ECG rhythm analysis is indicated
- Identify and describe details of inspection, auscultation, palpation specific to the cardiovascular system

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Learning Objectives (Cont'd)

- Identify and define heart sounds
- Relate heart sounds to hemodynamic events in the cardiac cycle
- Describe differences between normal and abnormal heart sounds
Learning Objectives (Cont'd)
- Define pulse deficit, pulsus paradoxus, pulsus alternans
- Describe how to determine if pulsus paradoxus, pulsus alternans, electrical alternans are present
- Describe the clinical significance of unequal arterial blood pressure readings in arms

Learning Objectives (Cont'd)
- Describe the etiology, epidemiology, history, physical findings of acute coronary syndromes
- Using patient history, physical examination findings, ECG analysis, develop treatment plan for acute coronary syndrome
- Describe the incidence of myocardial conduction defects

Learning Objectives (Cont'd)
- List other clinical conditions that may mimic signs and symptoms of acute coronary syndromes
- Describe etiology, epidemiology, history, physical findings of heart failure
- Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for heart failure
Learning Objectives (Cont'd)

- Describe the etiology, epidemiology, history, physical findings of myocarditis
- Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for myocarditis
- Describe the etiology, epidemiology, history, physical findings of cardiogenic shock

Learning Objectives (Cont'd)

- Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for cardiogenic shock
- Describe the etiology, epidemiology, history, physical findings of cardiac arrest
- Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for cardiac arrest

Learning Objectives (Cont'd)

- Assess and manage the adult immediately after resuscitation from cardiac arrest
- Identify, list inclusion, exclusion criteria for termination of resuscitation efforts
- Identify communication, documentation protocols with medical direction, law enforcement used for termination of resuscitation efforts
Learning Objectives (Cont'd)

• Describe the etiology, epidemiology, history, physical findings of hypertensive emergency
• Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for hypertensive emergency

Learning Objectives (Cont'd)

• Describe the etiology, epidemiology, history, physical findings of endocarditis
• Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for endocarditis

Learning Objectives (Cont'd)

• Describe the etiology, epidemiology, history, physical findings of pericarditis
• Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for pericarditis
Learning Objectives (Cont'd)

- Describe the etiology, epidemiology, history, physical findings of pericardial tamponade
- Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for pericardial tamponade

Learning Objectives (Cont'd)

- Describe the etiology, epidemiology, history, physical findings of aortic aneurysm
- Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for aortic aneurysm

Learning Objectives (Cont'd)

- Describe the etiology, epidemiology, history, physical findings of vascular disorders
- Using the patient history, physical examination findings, ECG analysis, develop a treatment plan for vascular disorder
Introduction

- Cardiovascular disorder
  - Diseases, conditions that involve the heart, blood vessels
- Heart disease
  - Conditions affecting the heart

Introduction (Cont’d)

- Coronary heart disease
  - Coronary arteries, resulting complications
    - Angina pectoris, acute MI
- Coronary artery disease
  - Affects arteries that supply heart muscle with blood

Risk Factors and Prevention Strategies

- Risk factors
  - Nonmodifiable (fixed) risk factors
  - Modifiable risk factors
    - High blood pressure
    - Elevated serum cholesterol levels
    - Tobacco use
    - Diabetes
Risk Factors and Prevention Strategies (Cont’d)

- Risk factors
  - Modifiable risk factors
    - Physical inactivity
    - Obesity, body fat distribution
    - Metabolic syndrome

- Contributing risk factors
  - Stress
  - Inflammatory markers
  - Psychosocial factors
  - Alcohol intake

Cardiovascular Anatomy and Physiology
Anatomy Review

- Blood vessels
  - Arteries
  - Arterioles
  - Capillaries
  - Venules
  - Veins

Anatomy Review (Cont’d)

- Heart anatomy
  - Location
    - Mediastinum
    - Behind sternum, above diaphragm
    - Base
    - Apex

Heart Location

- Third costal cartilage
- Sixth costal cartilage
- Second intercostal space
- Fiel
- Intercostal space
- Midsternal line
Anatomy Review (Cont'd)

• Heart anatomy
  ➢ Heart chambers
    ➢ Upper chambers
      ➢ Right, left atria
    ➢ Lower chambers
      ➢ Right, left ventricles

Anatomy Review (Cont'd)

• Heart anatomy
  ➢ Septum
  ➢ Pulmonary circulation
  ➢ Systemic circulation
  ➢ Blood carried from heart to body through arteries, arterioles, capillaries
  ➢ Blood returned to heart through venules, veins
Anatomy Review (Cont'd)

- Heart anatomy
  - Heart layers
    - Endocardium
    - Myocardium
    - Epicardium
    - Pericardium

- Heart anatomy
  - Heart valves
    - AV valves
      - Tricuspid, between the right atrium and right ventricle
      - Mitral/bicuspid, between the left atrium and left ventricle
      - Open when forward pressure forces blood forward
      - Close when backward pressure pushes blood backward

- Heart anatomy
  - Atrial kick
    - Blood flows continuously into atria
    - 70% flows directly through, into ventricles before atria contract
    - When atria contract, additional 30% is added to filling of ventricles
    - When ventricles contract (systole), pressure rises
      - Tricuspid and mitral valves close when pressure within the ventricles exceeds that of atria
Heart anatomy
- Semilunar (SL) valves
  - Pulmonic, aortic valves
  - Prevent backflow of blood from aorta and pulmonary arteries into ventricles
  - Close as ventricular contraction ends, pressure in pulmonary artery, aorta exceeds that of ventricles
  - Chordae tendinae, connective tissue, attached to AV valves underside and papillary muscles

Blood flow through heart
- Enters right atrium via superior, inferior venae cavae, coronary sinus
- Right atrium through tricuspid valve into right ventricle
- Right ventricle expels blood through pulmonic valve into pulmonary trunk
- Flows through pulmonary arteries to lungs

Blood flow through the heart
- Low in O₂, passes through pulmonary capillaries
- From left atrium through mitral valve into left ventricle
- Distributed throughout the body through the aorta and its branches
Anatomy Review (Cont'd)

- Blood flow through heart
  - Tissues of head, neck, upper extremities via superior vena cava
  - Lower body via inferior vena cava
  - Superior and inferior vena cava carry contents into right atrium

Anatomy Review (Cont'd)

- Cardiac cycle
  - Repetitive pumping process, events associated with blood flow through the heart
    - Systole
    - Diastole

Anatomy Review (Cont'd)

- Cardiac cycle
  - Depends on the ability of the cardiac muscle to contract, condition of heart's conduction system
  - Pressure with each chamber rises in systole, falls in diastole
  - Conduction system provides timing of events between atrial and ventricular systole
Anatomy Review (Cont’d)

- Coronary arteries
  - Right, left
  - Main arteries
  - Left anterior descending artery (LAD), circumflex (CX) artery, right coronary artery (RCA)
  - Lie on outer surface of heart

Anatomy Review (Cont’d)

- Coronary veins
  - Travel alongside arteries
  - Coronary sinus, largest vein, drains heart
Anatomy Review (Cont’d)

● Heart rate
  ➢ Affected by sympathetic and parasympathetic ANS
  ➢ Chronotropic effect
  ➢ Inotropic effect
  ➢ Dromotropic effect

Anatomy Review (Cont’d)

● Heart rate
  ➢ Baroreceptors
    • Specialized nerve tissue (sensors)
    • Found in internal carotid arteries, aortic arch
    • Detect changes in blood pressure
    • When stimulated cause sympathetic/parasympathetic response
    • Will “reset” to new “normal” after a few days of exposure to specific pressure

Anatomy Review (Cont’d)

● Heart rate
  ➢ Chemoreceptors
    • In internal carotid arteries, aortic arch, medulla detect changes in concentration of hydrogen ions (pH), O₂, carbon dioxide in blood
Anatomy Review (Cont'd)

- Heart rate
  - Parasympathetic stimulation
    - Parasympathetic fibers supply sinoatrial node, atrial muscle, and AV junction of heart by vagus nerves

- Sympathetic stimulation
  - Sympathetic nerves supply specific areas of the heart's electrical system, atrial muscle, ventricular myocardium
  - When stimulated, norepinephrine is released
  - Increases in heart rate shorten all phases of the cardiac cycle

- Increases in heart rate shorten all phases of the cardiac cycle
- Electrolyte, hormone levels, medications, stress, anxiety, fear, body temperature can influence heart rate
- Heart rate increases when body temperature increases, decreases when body temperature decreases
Heart as Pump

- Venous return
  - The most important factor determining amount of blood pumped out by the heart is the amount of blood flowing into the right heart.

Heart as Pump (Cont’d)

- Cardiac output
  - Amount of blood pumped into the aorta each minute by the heart
  - Defined as stroke volume × heart rate
  - Stroke volume determined by
    - Preload
    - Afterload

Heart as Pump (Cont’d)

- Cardiac output
  - Frank–Starling’s law
    - Greater the volume of blood in heart during diastole (preload), the more forceful cardiac contraction and more blood ventricle will pump (stroke volume)
    - Important that heart adjusts its pumping capacity in response to changes in venous return
    - During exercise, heart muscle fibers stretch in response to increased volume (preload) before contracting
Heart as Pump (Cont’d)

- Cardiac output
  - Frank–Starling’s law
  - Factors that increase cardiac output include increased body metabolism, exercise, age, and size of body
  - Factors that may decrease cardiac output include shock, hypovolemia, heart failure

Electrophysiology of the Heart

Cardiac Cells

- Types
  - Myocardial, contain contractile filaments
  - Pacemaker/conducting/automatic
Cardiac Action Potential

- Polarization
  - Potential energy exists due to an imbalance of charged particles, makes cells excitable
  - Proteins, phosphates unable to pass through cell membrane, results in negative charge inside the cell
  - Membrane potential
  - Electrolytes moved by pumps

Cardiac Action Potential (Cont’d)

- Depolarization
  - Pacemaker cells need flow of electrolytes across cell membrane to produce impulse
  - Cell stimulated, cell membrane changes, becomes permeable to Na⁺, K⁺
  - Inside of cell becomes more positive
  - Must occur before heart can mechanically contract, pump blood
Cardiac Action Potential (Cont’d)

- **Depolarization**
  - Proceeds from endocardium to epicardium
  - Chain reaction occurs from cell to cell in heart’s electrical conduction system until all cells are stimulated, depolarized
  - Impulse spreads from pacemaker cells to working myocardial cells, contract when stimulated

Cardiac Action Potential (Cont’d)

- **Depolarization**
  - When atria stimulated, P wave recorded on ECG
  - When ventricles stimulated, QRS complex recorded on ECG
  - Results in contraction
Cardiac Action Potential (Cont’d)

- Repolarization
  - After depolarization, cell recovers, restores electrical charges to normal
  - Charged particle movement across cell membrane, cell inside restored to negative charge
  - Stops flow of Na+ into cell, allows K+ to leave

- Repolarization
  - Negatively charges particles left inside cell, returned to resting state
  - Proceeds from epicardium to endocardium
  - ST segment, T wave represent ventricular repolarization
Cardiac Action Potential (Cont’d)

Ventricular Muscle Cell Action Potential

Phases

- Phase 0: depolarization
  - Rapid entry of Na⁺ into cell
  - Upstroke, spike, overshoot
  - Begins when cell receives impulse
  - Na⁺ moves into cell, K⁺ leaves, Ca²⁺ moves slowly in

Phases

- Phase 1: early repolarization
  - Na⁺ channels partially close slowing Na⁺ flow into cell
  - Cl enters, K⁺ leaves
Cardiac Action Potential (Cont’d)

- Phases
  - Phase 2: plateau phase
    - Ca++ slowly enters cell, K+ continues to leave
    - Allows cardiac muscle increased contraction period

Cardiac Action Potential (Cont’d)

- Phases
  - Phase 3: final rapid repolarization
    - Action potential downslope
    - Completes repolarization, K+ quickly leaves cell
    - Na+, Ca2+ channels close, stopping entry of Na+, Ca2+
    - Cell inside becomes more electrically negative
    - More sensitive to external stimuli until original sensitivity is restored
    - T wave on ECG

Cardiac Action Potential (Cont’d)

- Phases
  - Phase 4: resting membrane potential
    - Excess of Na+ inside cell, excess of K+ outside cell
    - Na+/K+ pump activated, move Na+ out of the cell, K+ into the cell
    - Heart polarized
Refractory Periods

- Recovery period cells need after discharge to respond to stimulus
- No longer than contraction

Refractory Periods (Cont’d)

- Absolute refractory period
  - Cell will not respond to further stimulation
  - Myocardial cells cannot contract, electrical conduction system cells cannot conduct electrical impulse

Refractory Periods (Cont’d)

- Relative refractory period
  - Some cells repolarized to threshold potential, can be stimulated to respond to stronger than normal stimulus
  - ECG, corresponds to T wave downslope
Refractory Periods (Cont’d)

- Supernormal period
  - Weaker than normal stimulus causes depolarization of cardiac cells
  - From phase 3 end to phase 4 beginning
  - ECG, corresponds to T wave end
  - Dysrhythmia can develop

Conduction System

- Sinoatrial node (SA)
  - Normal heartbeat, result of electrical impulse begins
  - Primary pacemaker, fastest firing rate
  - 60–100 beats/min
  - Upper posterior of right atrium, superior vena cava and right atrium meet

Conduction System (Cont’d)

- Sinoatrial node (SA)
  - Fibers directly connect with atria fibers
  - Impulse leaves, spread from cell to cell, wavelike form across atrial muscle
  - Contraction through AV node begins before depolarization is complete
Conduction System (Cont’d)

- Atrioventricular junction
  - AV node and the nonbranching portion of bundle of His
  - Provides electrical links between atria and ventricles
  - Accessory pathway

Conduction System (Cont’d)

- Atrioventricular junction
  - Atrioventricular node
    - Cells located in right atrium floor, behind tricuspid valve, near coronary sinus opening
    - CX provides blood supply
    - Supplied by sympathetic and parasympathetic nerve fibers
    - Impulse from atria enters AV node, conduction delay of impulse to ventricles
Conduction System (Cont'd)

- **Atrioventricular junction**
  - Bundle of His
    - Common bundle, AV bundle
    - Interventricular septum upper portion
    - Conducts electrical impulse to right and left bundle branches
    - Receives dual blood supply from branches of left anterior and posterior descending coronary arteries
    - Less vulnerable to ischemia
    - His-Purkinje system

Conduction System (Cont'd)

- **Right and left bundle branches**
  - Right innervates right ventricle
  - Left spreads to electrical impulse to interventricular septum, left ventricle

Conduction System (Cont'd)

- **Purkinje fibers**
  - Spread from interventricular septum into papillary muscles, continue downward toward heart apex, penetrates one-third of the way into ventricular muscle mass
  - Pacemaker cells firing rate of 20-40 beats/min
Conduction System (Cont’d)

- Purkinje fibers
  - Electrical impulse spreads rapidly through right and left bundle branches, Purkinje fibers to reach ventricular muscle
  - Spreads from endocardium to myocardium, reach the epicardial surface
  - Wrings blood out of ventricular chambers, forces it into arteries

Causes of Dysrhythmias

- Enhanced automaticity
  - Cardiac cells not normally associated with pacemaker function begin to depolarize
  - Pacemaker site other than the sinoatrial (SA) node increases firing rate beyond normal

Causes of Dysrhythmias (Cont’d)

- Triggered activity
  - Abnormal electrical impulse, after depolarizations
  - Pacemaker cells from site other than the SA node, myocardial working cells depolarize 1+ times after stimulation by single impulse
Causes of Dysrhythmias (Cont’d)

- **Reentry**
  - Impulse spread through tissue already stimulated by same impulse
  - Electrical impulse delayed/blocked in conduction system, delays electrical impulse entering cardiac cells
  - Depolarization can produce single premature beat/repetitive electrical impulses, causing abnormally fast heart rate

Causes of Dysrhythmias (Cont’d)

- **Escape beats/rhythms**
  - SA node slows/fails to initiate depolarization, lower site spontaneously produces electrical impulses
  - Protective mechanism, maintain cardiac output
  - Begin in AV junction, ventricles

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Causes of Dysrhythmias (Cont’d)

- Conduction disturbances
  - Rapid, too slow
  - May occur from trauma, drug toxicity, electrolyte disturbances, myocardial ischemia, infarction

Electrocardiogram (Cont’d)

- Uses
  - Monitor heart rate
  - Evaluate effects of disease/injury on heart function
  - Evaluate pacemaker function
  - Evaluate response to medications
  - Baseline reading before, during, and after medical procedure
  - Evaluate for signs of myocardial ischemia, injury, and infarction

Electrocardiogram (Cont’d)

- Information obtained
  - Orientation of heart in chest
  - Conduction disturbances
  - Electrical effects of medications, electrolytes
  - Mass of cardiac muscle
  - Presence of ischemic change
Electrocardiogram (Cont'd)

- Electrodes
  - Contain conductive media, applied to skin to view heart's electrical activity
  - Ensure conductive jelly not dry, avoid placing electrodes directly over bony areas
  - Monitoring cable attached to electrode and ECG machine, conducts current back to cardiac machine

Skill 22-1 ECG Monitoring

- Connect ECG cable to machine
- Connect lead wires to ECG cable
- Turn power on

Skill 22-1 ECG Monitoring (Cont'd)

- Open ECG electrode package
- Ensure electrode gel is moist
- Attach electrode to each lead wire
Skill 22-1 ECG Monitoring (Cont’d)

- Prepare patient skin
  - Gauze rub
  - Shave

Skill 22-1 ECG Monitoring (Cont’d)

- Place electrodes on patient
- Connect lead wires to electrodes

Skill 22-1 ECG Monitoring (Cont’d)

- Relax patient
- Adjust ECG size
- Feel pulse, compare with monitor
- Set heart rate alarms
Skill 22-1 ECG Monitoring (Cont’d)

- Select print/record
- Interpret rhythm
- Assess patient
- Attach rhythm strip to PCR

Electrocardiogram

- Electrodes
  - Leads
    - Record of electrical activity between two electrodes
    - Each records average current flow
  - Planes
    - Frontal view heart from front of body
    - Horizontal view heart as if body sliced in half horizontally
    - 12-lead, 12 different angles

Electrocardiogram (Cont’d)

- Electrodes
  - Leads
    - Each lead has positive electrode/pole
    - No electrical activity, straight line
      - Baseline
      - Isoelectric line
Electrocardiogram (Cont’d)

Wave of Depolarization

-electrocardiogram (ECG)-

Electrodes

- Leads
  - Waveform
    - Movement away from baseline in positive/forward direction
    - Related to specific electrical event in the heart
    - Wave of depolarization moves away from positive electrode, upright wave
    - Wave of depolarization moves toward positive electrode, waveform inverted
  - Biphasic, partly positive/partly negative

Electrocardiogram (Cont’d)

Electrodes

- Leads
  - Standard limb leads
    - Leads I, II, III
  - Bipolar limb leads
  - Positive electrode at left wrist in lead I
  - Left foot, leads II and III have positive electrode
Electrocardiogram (Cont’d)

- Electrodes
  - Leads
    - Standard limb leads
      - Difference in electrical potential between positive and negative pole measured by each lead
      - Place leads I, II, and III on extremities, avoid bony prominences

Electrocardiogram (Cont’d)

- Electrodes
  - Leads
    - Lead axis
    - Einthoven’s triangle

Electrocardiogram (Cont’d)

- Electrodes
  - Leads
    - Standard limb leads
      - Lead I records differences in electrical potential between left arm (+) and right arm (-) electrodes
      - Lead II records difference in electrical potential between left leg (+) and right arm (-) electrodes
      - Lead III records differences in electrical potential between left leg (+) and left arm (-) electrodes
Electrocardiogram (Cont'd)

Standard and Augmented Limb Leads

- Leads
  - Augmented limb leads
    - aVR, aVL, aVF
    - a = augment
    - V = voltage
    - R/L = right/left arm
    - F = foot/leg
  - Distinctive positive pole, no distinctive negative pole
  - Produced electrical potential normally small

Electrocardiogram (Cont'd)

- Leads
  - Chest leads
    - Unipolar
    - View heart in horizontal plane
    - V1-V6, all positive
Electrocardiogram (Cont’d)

- Leads
  - Chest leads
    - V1: Right side of sternum, fourth intercostal space
    - Views septum
    - V2: Left side of sternum, fourth intercostal space
    - Views septum
    - V3: Midway between V2 and V4
    - Views anterior heart
Electrocardiogram (Cont’d)

Chest Lead Placement

Electrocardiogram (Cont’d)

- Leads
  - Right chest leads
    - Right ventricular MI suspected
    - Placement identical to standard, except on right side of chest
    - V4R lead is the choice if there is no time

Electrocardiogram (Cont’d)

- Leads
  - Right chest leads, placement
    - V1R
      - Lead V5
    - V2R
      - Lead V5
    - V3R
      - Midway between V5R and V3R
Electrocardiogram (Cont’d)

- Leads
  - Right chest leads, placement
    - V4R
      - Right midclavicular line, fifth intercostal space
    - V5R
      - Right anterior axillary line at same level as V4R
    - V6R
      - Right midaxillary line at same level as V4R

Electrocardiogram (Cont’d)

Placement of Right Chest Leads

Electrocardiogram (Cont’d)

- Leads
  - Posterior chest leads
    - Standard 12-lead, no leads look directly at posterior surface
    - Place leads further left, toward back
    - All leads placed on same horizontal line as V4–V6
Electrocardiogram (Cont’d)

- Leads
  - Posterior chest leads
    - Lead V7 placed at posterior axillary line
    - Lead V8 placed at angle of scapula
    - Lead V9 placed over left border of spine

Electrocardiogram (Cont’d)

ECG paper
- Graph paper with small, large boxes measured in millimeters
- Horizontal axis corresponds to time, in seconds
- Vertical axis measures voltage/amplitude of waveform
Electrocardiogram (Cont’d)

- ECG paper
  - Vertical axis measures voltage/amplitude of waveform
  - Positive/negative value
  - Measured in millimeters
  - 1-mV electrical signal produces deflection measuring exactly 10 mm tall, calibrated

Electrocardiogram (Cont’d)

- Waveforms
  - P wave
    - First waveform in cardiac cycle
    - First abrupt/gradual movement away from baseline, ends when returns to baseline
    - Represents depolarization and spread of electrical impulse throughout right and left atria
    - Precedes each QRS complex
Electrocardiogram (Cont'd)

- Waveforms
  - QRS complex
    - Has Q, R, and S wave
    - Spread of electrical impulse through ventricles
    - Depolarization triggers contraction of ventricular tissue
    - Not every complex contains all three waves

Electrocardiogram (Cont'd)

- Waveforms
  - QRS complex
    - R wave, first positive deflection
    - S wave, negative deflection following R wave
    - R and S waves, simultaneous depolarization of right and left ventricles

Electrocardiogram (Cont'd)

- Waveforms
  - QRS complex
    - If complex is entirely positive waveform, R wave
    - If complex is entirely negative waveform, QS wave
    - If 2+ deflections are present in the same complex, second is R prime (R´)
**Electrocardiogram (Cont’d)**

- Waveforms
  - QRS complex
    - If 2 – deflections in the same complex, S prime (S’)
    - Beginning measured from where first complex wave begins to move away from baseline
    - Last wave of complex levels out/distinctly changes direction, at/above/below baseline end of complex
    - Normal duration <0.10 second

- Waveforms
  - T wave
    - Ventricular repolarization
    - Slope of ST wave becomes abruptly/gradually steeper
    - Ends when return to baseline
    - <5 mm in height in any limb lead, >0.5 mm in height in leads I and II
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Electrocardiogram (Cont’d)
• Waveforms
  ➢ T wave
    • If follows abnormal QRS complex, opposite in direction to QRS
    • Seen with ventricular beats/rhythms and bundle branch block
    • Tall, pointed T waves seen in hyperkalemia

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Electrocardiogram (Cont’d)
• Waveforms
  ➢ U wave
    • Small waveform
    • When seen, follows T wave
    • Represents repolarization of Purkinje fibers
    • Seen when heart rate is slow
    • Difficult to identify when rate >90 beats/min
    • Tallest in leads V2 and V3
    • Usually in the same direction as preceding T wave
U Waves

Segments
- PR segment
  - PR interval part
  - Horizontal line between P wave end and QRS complex beginning
  - Represents activation of AV node, bundle of His, bundle branches, Purkinje fibers
  - Atrial repolarization occurs
**Electrocardiogram (Cont’d)**

- **Segments**
  - **TP segment**
    - Between T wave end and following P wave beginning
    - Heart in normal limits, usually isoelectric
    - Rapid heart rates, TP segment unrecognizable

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**Electrocardiogram (Cont’d)**

- **Segments**
  - **ST segment**
    - Between QRS complex and T wave
    - Used whether final wave of QRS complex is R/S wave
    - Early part of repolarization of right and left ventricles
Electrocardiogram (Cont’d)

- Waveforms
  - ST segment
    - Begins at isoelectric line, extends from S wave end, curves upward to beginning of T wave
    - J-point, point at which QRS complex and ST segment meet

- ST segment displacement
  - Myocardial ischemia, injury, infarction
  - Elevated if deviated above baseline
  - Depressed if segment is deviated below
  - Locate J-point

- Use TP and PR segments to estimate position of isoelectric line
- Compare level of ST segment to isoelectric line
- Significant findings, seen in 2+ leads facing same anatomic area of the heart
Electrocardiogram (Cont’d)

- Intervals
  - Waveform and segment
    - PR interval
      - P wave + PR segment = PR interval
      - Changes with heart rate, normally 0.12-0.20 second
  - QT interval
    - Time from ventricular depolarization to repolarization
      - QRS complex beginning to T wave ending
      - Q wave absence, R wave beginning
      - Duration depends on age, gender, heart rate
Electrocardiogram (Cont’d)

- Intervals
  - QT interval
    - Heart rate increases, QT interval shortens
    - Corrected by adjusting to heart rate, QTc
    - Abnormally long if QTc >0.44 second
    - Measure interval between two consecutive R waves (R-R), divide by 2

Electrocardiogram (Cont’d)

- Intervals
  - R-R and P-P intervals
    - Determine rate, regularity of cardiac rhythm
    - Ventricular rhythm regularity, R-R
    - Atrial rhythm regularity, P-P

Electrocardiogram (Cont’d)

- Artifact
  - Distortion by noncardiac electrical activity
  - Loose electrodes, ECG cables, wires
  - Muscle tremor
  - Patient movement
  - External chest compression
  - 60-cycle interference
Analyzing Rhythm Strip

- Assess rhythm, regularity
  - Distance between P waves, QRS complexes
  - R-R intervals/P-P intervals same, regular
  - Variation ± 10% is acceptable
  - Evaluate across entire 6-second rhythm strip

Analyzing Rhythm Strip (Cont’d)

- Assess rhythm, regularity
  - Ventricular rhythm
    - Measure distance between two consecutive R-R intervals
    - If same, regular

Analyzing Rhythm Strip (Cont’d)

- Assess rhythm, regularity
  - Atrial rhythm
    - Measure distance between two consecutive P-P intervals
    - If same, regular
Analyzing Rhythm Strip (Cont’d)

- Assess rate
  - Method 1: 6-second method
    - Ventricular rate
      - Number of complete QRS complexes within 6 seconds
      - \( \times 10 \) = number of complexes in 1 minute

- Assess rate
  - Method 2: large boxes
    - Ventricular rate
      - Number of large boxes between R-R interval, divide into 300
    - Atrial rate
      - Number of large boxes between P-P interval, divide into 300

- Assess rate
  - Method 3: small boxes
    - 1500 boxes = 1 minute
    - Ventricular rate
      - Number of small boxes between R-R interval, divide into 1500
    - Atrial rate
      - Number of small boxes between P-P interval, divide into 1500
Analyzing Rhythm Strip (Cont’d)

Calculating Heart Rate

Assess rate
- Method 4: sequence method
  - Ventricular rate
    - Select R wave on dark vertical line
    - Number next six consecutive dark vertical lines 300, 150, 100, 75, 60, 50
Analyzing Rhythm Strip (Cont’d)

• Identify, examine P waves
  - Left of each QRS complex, precedes
  - Similar in size, shape, position
  - If none, rhythm originated in AV
    junction/ventricles

Analyzing Rhythm Strip (Cont’d)

• Assess intervals (evaluate conduction)
  - PR interval
  - QRS duration
  - QT interval

Analyzing Rhythm Strip (Cont’d)

• Assess intervals (evaluate conduction)
  - PR interval
    - Measure where P wave leaves baseline to QRS
      complex beginning
    - Normal, 0.12-0.20 second
Analyzing Rhythm Strip (Cont’d)

- Assess intervals (evaluate conduction)
  - QRS duration
    - Measure QRS complexes duration
    - Narrow, <0.10 second, supraventricular
    - Wide, >0.10 second

- QT interval
  - Measure QT interval in leads showing largest amplitude T waves
  - If less than half the R-R interval, it is normal

- Evaluate the overall appearance of rhythm
  - ST segment
    - Elevated if the segment deviated above baseline
    - Depressed if the segment deviated below baseline
  - T wave
    - Negative, myocardial ischemia
    - Tall, pointed, hyperkalemia
Analyzing Rhythm Strip (Cont’d)

- Interpret rhythm, evaluate clinical significance
  - Origin site of rhythm
  - Mechanism
  - Ventricular rate

Cardiac Dysrhythmias

Sinus Mechanisms

- Normal rhythm starts in SA node
  - Positive P wave before each QRS complex
  - P waves look alike
  - Constant PR interval
  - Regular atrial, ventricular rhythm
Sinus Mechanisms (Cont’d)

- **Affects**
  - Medications
  - Diseases, conditions cause increased/decreased heart rate, beat irregularly
  - Diseases, conditions delay block impulse from leaving SA node
  - Diseases, conditions prevent impulse being generated in SA node

Sinus Mechanisms (Cont’d)

- **Sinus rhythm**
  - Normal heart rhythm, electrical activity
    - Rate
      - 60-100 beats/min
    - Rhythm
      - P-P interval and R-R interval regular

Sinus Mechanisms (Cont’d)

- **Sinus rhythm**
  - Normal heart rhythm, electrical activity
    - P waves
      - Positive in lead II
      - One precedes each QRS complex
      - Look alike
    - PR interval
      - 0.12-0.20 second, constant from beat to beat
    - QRS duration
      - 0.10 second unless intraventricular conduction delay
Sinus Mechanisms (Cont'd)

- Sinus bradycardia
  - SA nodes fire at slower rate than normal for age
  - Adults, adolescents <60 beats/min
  - Severe <40 beats/min

- Sinus bradycardia
  - Assess tolerance to rhythm
    - No symptoms, no treatment
    - Symptoms, treat
      - O₂
      - IV access
      - Atropine

Sinus Mechanisms (Cont'd)

- Sinus rhythm
  - Characteristics
    - Rate
      - <60 beats/min
    - Rhythm
      - P-P and R-R intervals regular
    - P waves
      - Positive in lead II
      - One precedes each QRS complex
      - Look alike
Sinus Mechanisms (Cont’d)

• Sinus rhythm
  ➢ Characteristics
    • PR interval
      ➢ 0.12-0.20 second, constant from beat to beat
    • QRS duration
      ➢ 0.10 second unless intraventricular conduction delay

Sinus Mechanisms (Cont’d)

• Sinus tachycardia
  ➢ Causes
    • Exercise
    • Fever
    • Pain
    • Fear, anxiety
    • Hypoxia
    • Congestive heart failure
    • Acute MI
    • Infection
    • Sympathetic stimulation
    • Shock
    • Dehydration, hypovolemia
    • Pulmonary embolism
    • Hyperthyroidism
    • Medications
    • Caffeine-containing beverages
    • Nicotine
    • Drugs

Sinus Mechanisms (Cont’d)

• Sinus tachycardia
  ➢ Heart’s demand for O₂ increases as heart rate increases
    • Less time for ventricles to fill
    • Less blood to pump out
    • Decreased cardiac output
  ➢ Warning sign for heart failure, cardiogenic shock, dysrhythmias
Sinus Mechanisms (Cont’d)

- Sinus tachycardia
  - Characteristics
    - Rate: 101-180 beats/min
    - Rhythm: P-P and R-R intervals regular
    - P waves:
      - Positive in lead II
      - One precedes each QRS complex
      - Look alike: At very fast rates, differentiating P wave from T wave difficult
    - PR interval:
      - 0.12-0.20 second (may shorten), constant from beat to beat
    - QRS duration:
      - 0.10 second/less unless intraventricular conduction delay
Sinus Mechanisms (Cont’d)

- Sinus arrhythmia
  - SA node fires irregularly
  - Respiratory sinus arrhythmia
    - Respiration phases, intrathoracic pressure changes
    - Heart increases with inspiration, decreases with expiration
    - No treatment unless with slow heart rate

Sinus Mechanisms (Cont’d)

- Sinus arrhythmia
  - Characteristics
    - Rate
      - 60-100 beats/min
    - Rhythm
      - Irregular, phasic with respiration
    - P waves
      - Positive in lead II
      - One precedes each QRS complex
      - Look alike
      - At very fast rates, differentiating P wave from T wave difficult

Sinus Mechanisms (Cont’d)

- Sinus arrhythmia
  - Characteristics
    - PR interval
      - 0.12-0.20 second (may shorten), constant from beat to beat
    - QRS duration
      - 0.10 second/less unless intraventricular conduction delay
Sinus Mechanisms (Cont'd)

Sinus Arrhythmia

- Sinoatrial block
  - SA node initiates impulse, blocked as it exits SA node
  - Cell failure in SA node to conduct impulse from pacemaker cells to surrounding atrium
  - Falls back into rhythm after one skipped beat

- Causes:
  - Acute coronary syndromes
  - Medications
  - Coronary artery disease
  - Myocarditis
  - Heart failure
  - Carotid sinus sensitivity
  - Increased vagal tone
Sinus Mechanisms (Cont’d)

- Sinoatrial block
  - Characteristics
    - Rate
      - Usually normal, but varies because of pause
    - Rhythm
      - Irregular because of pause; pause same as distance between two other P-P intervals
    - P waves
      - Positive in lead II
      - One precedes each QRS complex
      - Look alike

Sinus Mechanisms (Cont’d)

- Sinoatrial block
  - Characteristics
    - PR interval
      - 0.12-0.20 sec (may shorten), constant from beat to beat
    - QRS duration
      - 0.10 sec/less unless intraventricular conduction delay

Sinus Mechanisms (Cont’d)

- Sinoatrial block

Sinoatrial Block

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Sinus Mechanisms (Cont’d)

- Sinus arrest
  - SA node pacemaker cells fail to initiate electrical impulse for 1+ beats
  - Absent PQRST complexes

- Causes
  - Hypoxia
  - Acute coronary syndrome
  - Hyperkalemia
  - Digitalis toxicity
  - Medication reaction
  - Carotid sinus sensitivity
  - Increased vagal tone

- Characteristics
  - Rate
    - Usually normal, but varies because of pause
  - Rhythm
    - Irregular
  - Pause undetermined length
    - Not the same distance as other P-P intervals
  - P waves
    - Positive in lead II
    - One precedes each QRS complex
    - Look alike
Sinus Mechanisms (Cont’d)

- Sinus arrest
  - Characteristics
    - PR interval
      - 0.12–0.20 second (may shorten), constant from beat to beat
    - QRS duration
      - 0.10 second/less unless intraventricular conduction delay

Atrial Dysrhythmias

- P waves reflect atrial depolarization
  - Begins in atria, P wave shaped differently than when it begins in SA node
  - Follows different conduction pathway to AV node
Atrial Dysrhythmias (Cont’d)

- Premature atrial complexes (PACs)
  - Irritable site within atria fires before next SA node impulse
  - Interrupts sinus rhythm
  - P wave may be biphasic, flattened, notched, pointed

Atrial Dysrhythmias (Cont’d)

- PACs
  - Ectopic complexes in patterns
    - Pairs: two ectopic complexes consecutively
    - Runs/bursts: 3+ ectopic complexes consecutively
    - Bigeminy: ectopic complex every other beat
    - Trigeminy: ectopic complex every third beat
    - Quadrigeminy: ectopic complex every fourth beat

Atrial Dysrhythmias (Cont’d)

- Sinus Tachycardia with PACs

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Atrial Dysrhythmias (Cont’d)

- PACs
  - Characteristics
    - Rate
      - Usually within normal range, depends on underlying rhythm
    - Rhythm
      - Regular with premature beats

- P waves
  - Premature (occurring earlier than next expected P wave)
  - Positive in lead I
  - One before each QRS complex
  - Differ in shape from sinus P waves
    - Flattened, notched, pointed, biphasic, lost in preceding T wave
  - PR interval
    - Normal/prolonged depending on prematurity of beat

- QRS duration
  - Usually 0.10 second/less
  - May be wide/absent
  - QRS similar in shape to those of underlying rhythm unless PAC is abnormally conducted
Atrial Dysrhythmias (Cont’d)

- PACs
  - Aberrantly conducted PACs
    - PAC occurs early, right bundle branch slow to respond
    - Impulse travels down left bundle branch, stimulates right bundle branch
    - QRS wide from ventricular depolarization delay
    - Conduction through ventricles abnormal

Atrial Dysrhythmias (Cont’d)

PACs With/Without Aberrancy

Atrial Dysrhythmias (Cont’d)

- PACs
  - Nonconducted PACs
    - PAC occurs early, close to T wave of preceding beat
    - Only P wave seen with no QRS after
    - P wave occurred too early to be conducted
Atrial Dysrhythmias (Cont’d)

- PACs
  - Causes
    - Emotional stress
    - Heart failure
    - Acute coronary syndromes
    - Mental, physical fatigue
    - Atrial enlargement
  - Valvular heart disease
  - Digitalis toxicity
  - Electrolyte imbalance
  - Hyperthyroidism
  - Stimulants

Atrial Dysrhythmias (Cont’d)

- Do not require treatment if infrequent
  - Frequent
    - Atrial fibrillation
    - Atrial flutter
    - Paroxysmal supraventricular tachycardia
    - Correct underlying cause

Sinus Rhythm with Nonconducted PAC
Atrial Dysrhythmias (Cont'd)

- Wandering atrial pacemaker
  - Multiformed atrial rhythm, updated term
  - P wave size, shape, and direction vary from beat to beat
  - Must have three different P wave configurations in same lead
  - Usually no signs/symptoms

- Characteristics
  - Rate
    - Usually 60-100 beats/min
    - May be slow
    - If >100 beats/min, multifocal
  - Rhythm
    - May be irregular as pacemaker shifts from SA node to ectopic atrial locations and AV junction

- P waves
  - Size, shape, and direction change from beat to beat
  - 3+ configurations needed

- PR interval
  - Variable

- QRS duration
  - 0.10 second/less unless intraventricular conduction delay
Atrial Dysrhythmias (Cont’d)

Wandering Atrial Pacemaker

Atrial Dysrhythmias (Cont’d)

- Multifocal atrial tachycardia
  - Multiformed atrial rhythm with ventricular rate >100 beats/min
  - Multiple ectopic sites stimulate atria

Atrial Dysrhythmias (Cont’d)

- Multifocal atrial tachycardia
  - Conditions
    - Severe chronic obstructive pulmonary disease
    - Hypoxia
    - Acute coronary syndromes
    - Digoxin toxicity
    - Rheumatic heart disease
    - Theophylline toxicity
    - Electrolyte imbalances
Atrial Dysrhythmias (Cont’d)

- Multifocal atrial tachycardia
  - Treatment
    - Underlying cause
    - Vagal maneuver
    - Adenosine IV

Skill 22-2 Carotid Sinus Massage

- Palpate each carotid separately to assess pulse
- Check for carotid bruits with stethoscope
Skill 22-2 Carotid Sinus Massage (Cont'd)

- With two fingers, locate the carotid pulse
- With firm pressure, press the carotid artery toward the cervical vertebrae

Skill 22-2 Carotid Sinus Massage (Cont'd)

- After the procedure, reassess vital signs and ECG rhythm

Atrial Dysrhythmias

- Supraventricular tachycardia (SVT)
  - Begin above bifurcation of bundle of His
  - Supraventricular dysrythmias include rhythms that begin in SA node, atrial tissue, AV junction
Atrial Dysrhythmias (Cont’d)

- SVT
  - Atrial tachycardia (AT)
    - Irritable site in atria automatically fires at rapid rate
    - Overrides SA node, becomes pacemaker
    - Every atrial impulse conducted to ventricles

Atrial Dysrhythmias (Cont’d)

- SVT
  - AT
    - Paroxysmal atrial tachycardia
      - AT suddenly starts/ends
      - AV node filters some impulses to protect the ventricles

Atrial Dysrhythmias (Cont’d)

- SVT
  - AT
    - Triggers
      - Stimulant use
      - Infection
      - Electrolyte imbalance
      - Acute illness with excessive catecholamine release
      - MI
    - Short event, no treatment
    - Sustained
Atrial Dysrhythmias (Cont'd)

- SVT
  - AT characteristics
    - Rate
      - 100-250 beats/min
    - Rhythm
      - Regular
    - P waves
      - One positive P wave precedes each QRS complex in lead II
      - P waves differ in shape from sinus P waves
      - Isoelectric baseline usually present between P waves
  - PR interval
    - Shorter/longer than normal
  - QRS duration
    - 0.10 second/less unless intraventricular conduction delay
Atrial Dysrhythmias (Cont’d)

• SVT
  ➢ Atrioventricular nodal reentrant tachycardia (AVNRT)
    ➢ Fast and slow pathways in AV node form electrical circuit/loop
    ➢ Impulse spins around the AV nodal area
    ➢ Reentry in area of the AV node

• Triggers
  ➢ Hypoxia
  ➢ Stress
  ➢ Overexertion
  ➢ Anxiety
  ➢ Caffeine
  ➢ Smoking
  ➢ Sleep deprivation
  ➢ Medications
Atrial Dysrhythmias (Cont'd)

- SVT
  - AVNRT
    - Treatment, stable
      - O₂ (if indicated), IV access
      - Vagal maneuvers
      - Adenosine
    - Treatment, unstable
      - O₂ (if indicated), IV access
      - Sedation
      - Synchronized cardioversion

Atrial Dysrhythmias (Cont'd)

- SVT
  - AVNRT characteristics
    - Rate
      - 150-250 beats/min
    - Rhythm
      - Regular

Atrial Dysrhythmias (Cont'd)

- SVT
  - AVNRT characteristics
    - P waves
      - P waves often hidden in QRS complex
      - If ventricles are stimulated first then atria, negative P wave appears after QRS in leads II, III, aVF
      - When atria are depolarized after ventricles, P wave distorts end of QRS complex
Atrial Dysrhythmias (Cont’d)

• SVT
  ➢ AVNRT characteristics
    ➢ PR interval
      ➢ P waves not seen before QRS complex
      ➢ PR interval not measurable
    ➢ QRS duration
      ➢ 0.10 second/less unless intraventricular conduction delay
Atrial Dysrhythmias (Cont’d)

- SVT
  - Atrioventricular reentrant tachycardia (AVRT)
    - Impulse begins above the ventricles, travels by pathway other than AV node and bundle of His

Atrial Dysrhythmias (Cont’d)

- SVT
  - AVRT
    - Preexcitation
      - Rhythms originate from above ventricles
      - Impulse travels by pathway other than AV node and bundle of His
      - Supraventricular impulse excites ventricles earlier
      - Prone to AVRT
      - Wolff-Parkinson-White (WPW) syndrome
Atrial Dysrhythmias (Cont’d)

Wolff-Parkinson-White Syndrome

SVT

- AVRT
  - Treatment
    - O₂ (if indicated)
    - IV access
    - Contact physician
    - Do not administer medications that slow or block AV conduction

Skill 22-3 Synchronized Cardioversion

- Give O₂
- Start IV
Skill 22-3 Synchronized Cardioversion (Cont'd)

- Remove hair where electrodes will be placed
- Place pads on chest

- Press “sync” control on defibrillator
- Select lead with optimum QRS complex amplitude and no artifact present

- If patient is awake and time permits, sedation per local protocol
- Select appropriate energy level on defibrillator
Skill 22-3 Synchronized Cardioversion (Cont’d)

- Charge defibrillator
- Place pads on chest, firm pressure
- Call "Clear!"
- No O₂ flowing over chest

Skill 22-3 Synchronized Cardioversion (Cont’d)

- Press and hold both discharge buttons at the same time until shock is delivered
- Release shock controls after shock is delivered

Atrial Dysrhythmias

- Atrial flutter
  - Type I
    - Caused by reentry
    - Impulse circles around large tissue area
    - Typical, classic atrial flutter
    - 250-350 beats/min
Atrial Dysrhythmias (Cont'd)

- Atrial flutter
  - Type II
  - Develop atrial fibrillation
  - 350-450 beats/min

Atrial Dysrhythmias (Cont'd)

- Atrial flutter
  - Conditions
    - Hypoxia
    - Pulmonary embolism
    - Chronic lung disease
    - Mitral/tricuspid valve stenosis/regurgitation
    - Pneumonia
    - Ischemic heart disease
    - Complication of MI
    - Cardiomyopathy
    - Hyperthyroidism
    - Digitalis/quinidine toxicity
    - Cardiac surgery
    - Pericarditis/myocarditis

Atrial Dysrhythmias (Cont'd)

- Atrial flutter
  - Characteristics
    - Rate
      - 250-450 beats/min, typically 300 beats/min
      - Ventricular rate variable, determined by AV blockade, will not exceed 180 beats/min
    - Rhythm
      - Atrial regular
      - Ventricular regular/irregular depending on AV conduction/blockade
Atrial Dysrhythmias (Cont'd)

- Atrial flutter
  - Characteristic
    - P waves
    - No identifiable P waves
    - Saw-toothed “flutter” waves
    - PR interval
    - Not measurable
    - QRS duration
      - 0.10 second/less, widened if flutter waves buried in QRS complex/intraventricular conduction delay

Atrial Dysrhythmias (Cont'd)

- Atrial fibrillation
  - Irritable sites in atria firing at rate of 400-600/min
  - Muscles quiver
  - Ineffective atrial contraction
  - Decreased stroke volume
  - Cardiac output decreases
  - Atrial kick loss
Atrial Dysrhythmias (Cont’d)

- **Atrial fibrillation**
  - Conditions
    - Idiopathic (no clear cause)
    - Hypertension
    - Ischemic heart disease
    - Advanced age
    - Rheumatic heart disease
    - Cardiomyopathy
    - Heart failure
    - Congenital heart disease
    - Sick sinus syndrome
    - Wolff-Parkinson-White syndrome
    - Pericarditis
  - **Conditions**
    - Pulmonary embolism
    - Chronic lung disease
    - After surgery
    - Diabetes
    - Stress
    - Sympathomimetics
    - Excessive caffeine
    - Hypoxia
    - Hypokalemia
    - Hypoglycemia
    - Systemic infection
    - Hyperthyroidism
    - Electrocution

- **Stroke risk**
  - Treatment dependent
  - Ventricular rate
  - Rhythm duration
  - General health
  - Rhythm toleration
Atrial Dysrhythmias (Cont’d)

- Atrial fibrillation
  - Signs/symptoms
    - Lightheadedness
    - Palpations
    - Dyspnea
    - Chest discomfort
    - Low BP
    - Rapid ventricular rate
  - Characteristics
    - Rate: 400-600 beats/min
    - Ventricular rate is variable
    - Rhythm: Ventricular usually irregularly irregular
    - P waves:
      - No identifiable P waves
      - Fibrillatory waves present, erratic, wavy baseline
    - PR interval: Not measurable
    - QRS duration: 0.10 second, widened intraventricular conduction delay
Atrial Dysrhythmias (Cont’d)

Junctional Dysrhythmias
• AV junction paces heart
  ➢ SA node fails to discharge
  ➢ Impulse from SA node generated, blocked as it exits SA node
  ➢ Discharge rate of SA node slower than AV junction
  ➢ SA node impulse generated, conducted through atria, not to ventricles

Junctional Dysrhythmias (Cont’d)
• AV junction paces heart
  ➢ Electrical impulse must travel backward to activate the atria
  ➢ P wave inverted in leads II, III, aVF
  ➢ Atria depolarizes before ventricles, inverted P wave seen before QRS complex
Junctional Dysrhythmias (Cont’d)

- AV junction paces heart
  - PR interval < 0.12 second
  - If atria and ventricles depolarize at the same time, P wave is visible
  - If atria depolarizes after ventricles, inverted P wave will appear after QRS complex

Junctional Dysrhythmias (Cont’d)

- Premature junctional complexes (PJC)
  - Irritable site within AV junction fires before next SA node impulse is due to fire
  - QRS complex < 0.10 second
  - Noncompensatory pause often follows PJC
  - Junctional complexes
Junctional Dysrhythmias (Cont’d)

- Premature junctional complexes
  - PJC causes
    - Heart failure
    - Acute coronary syndrome
    - Mental, physical fatigue
    - Valvular heart disease
    - Digitalis toxicity
    - Electrolyte imbalance
    - Rheumatic heart disease
    - Stimulants

Junctional Dysrhythmias (Cont’d)

Sinus Tachycardia With PJC’s

Junctional Dysrhythmias (Cont’d)

- Premature junctional complexes
  - Characteristics
    - Rate
      - Usually within normal range
    - Rhythm
      - Regular with premature beats
    - P waves
      - Before, during, and after QRS
    - P wave inverted in leads II, III, aVF if seen
Junctional Dysrhythmias (Cont’d)

- Premature junctional complexes
  - Characteristics
    - PR interval
      - If P wave occurs before QRS, PR interval is usually ≤ 0.12 second
      - No P wave before QRS, PR interval is present
    - QRS duration
      - ≤ 0.10 second, unless aberrantly conducted/intraventricular conduction delay

Junctional Dysrhythmias (Cont’d)

- Junctional escape beats/rhythm
  - Sinus arrests, follow pauses of nonconducted PACs
  - Characteristics
    - Rate
      - Usually within normal range
    - Rhythm
      - Regular with late beats
    - P waves
      - Before, during, and after QRS
      - P wave inverted in leads II, III, aVF if seen
Junctional Dysrhythmias (Cont’d)

- Junctional escape beats or rhythm
  - Junctional rhythm
  - Several sequential junctional escape beats
  - Junctional bradycardia

Junctional Dysrhythmias (Cont’d)

- Causes
  - Acute coronary syndrome
  - Hypoxia
  - Rheumatic heart disease
  - Valvular disease
  - SA node disease
  - Increased parasympathetic tone
  - Cardiac surgery
  - Medication effects
Junctional Dysrhythmias (Cont’d)

- Junctional escape beats or rhythm
  - Junctional rhythm, characteristics
    - Rate
      - 40-60 beats/min
    - Rhythm
      - Very regular
    - P waves
      - Before, during, and after QRS
      - P wave inverted in leads II, III, aVF

- PR interval
  - If P wave occurs before QRS, PR interval is usually 0.12 second/less
  - No P wave before QRS, PR interval present

- QRS duration
  - 0.10 second/less, unless aberrantly conducted/intraventricular conduction delay
Junctional Dysrhythmias (Cont’d)

- Accelerated junctional rhythm
  - AV junction speeds up, fires at 60-100 beats/min
  - Ventricular rate increase

Junctional Dysrhythmias (Cont’d)

- Accelerated junctional rhythm
  - Causes
    - Digitalis toxicity
    - Acute MI
    - Cardiac surgery
    - Rheumatic fever
    - COPD
    - Hypokalemia

Junctional Dysrhythmias (Cont’d)

- Accelerated junctional rhythm
  - Characteristics
    - Rate
      - 61-100 beats/min
    - Rhythm
      - Very regular
    - P waves
      - Before, during, and after QRS
      - P wave inverted in leads II, III, aVF if seen
Junctional Dysrhythmias (Cont’d)

- Accelerated junctional rhythm
  - Characteristics
    - PR interval
      - If P wave occurs before QRS, PR interval is usually ≤ 0.12 second/less
      - No P wave before QRS, PR interval present
    - QRS duration
      - ≤ 0.10 second/less, unless aberrantly conducted/intraventricular conduction delay

Junctional Dysrhythmias (Cont’d)

- Junctional tachycardia
  - Ectopic rhythm, begins in pacemaker cells in bundle of His
  - ≥ 3 sequential PJCs > 100 beats/min
  - Ventricular rate for paroxysmal junctional tachycardia > 140 beats/min

Junctional Dysrhythmias (Cont’d)

- Junctional tachycardia
  - Causes
    - Acute coronary syndrome
    - Heart failure
    - Theophylline administration
    - Digitalis toxicity
Junctional Dysrhythmias (Cont’d)

- Junctional tachycardia
  - Treatment
    - Tolerates rhythm, observation
    - Symptomatic
      - O₂, (if indicated )
      - IV access
      - Vagal maneuvers
      - IV adenosine
      - Beta-blocker, calcium channel blocker

Junctional Dysrhythmias (Cont’d)

- Junctional tachycardia
  - Characteristics
    - Rate
      - 101-180 beats/min
    - Rhythm
      - Very regular
    - P waves
      - Before, during, and after QRS
      - P wave inverted in leads II, III, aVF if seen

Junctional Dysrhythmias (Cont’d)

- Junctional tachycardia
  - Characteristics
    - PR interval
      - If P wave occurs before QRS, PR interval is usually 0.12 seconds less
      - No P wave before QRS, PR interval present
    - QRS duration
      - 0.10 secondless, unless aberrantly conducted/intraventricular conduction delay
Ventricular Dysrhythmias

- Ventricles assume pacing responsibility
  - SA node fails to discharge
  - Impulse from SA node generated, blocked as it exits SA node
  - Discharge rate of SA node less than ventricles
  - Irritable site in either ventricle produces early/rapid rhythm
  - QRS complexes abnormally shaped, longer

Ventricular Dysrhythmias (Cont’d)

- Ventricles, heart’s least efficient pacemaker
- Generate impulses at rate of 20-40 beats/min

Ventricular Dysrhythmias (Cont’d)

- Premature ventricular complexes (PVCs)
  - Irritable site within either ventricle
  - Occurs earlier than next expected sinus beat
  - QRS ≥0.12 second
Ventricular Dysrhythmias (Cont’d)

- PVCs
  - Types
    - Uniform PVCs
      - Premature ventricular beats look the same in the same lead, begin from the same anatomic site
    - Multiform PVCs
      - PVCs look different from one another in the same lead
    - Interpolated PVCs
      - Squeezed between two regular complexes, does not disturb underlying rhythm

- PVCs
  - Types
    - R-on-T PVCs
      - R wave of PVC falls on T wave of preceding beat
    - Couplet/paired
      - Two consecutive PVCs
    - Multiform PVCs
      - PVCs look different from one another in the same lead
    - Interpolated PVCs
      - Squeezed between two regular complexes, does not disturb underlying rhythm

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    - Interpolated PVCs
      - Squeezed between two regular complexes, does not disturb underlying rhythm
Ventricular Dysrhythmias (Cont’d)

Sinus Rhythm With PVCs

- PVCs
  - Causes
    - Normal variant
    - Hypoxia
    - Stress, anxiety
    - Exercise
    - Digitalis toxicity
    - Acid-base imbalance
    - Myocardial ischemia
    - Electrolyte imbalance
    - Heart failure
  - Increased sympathetic tone
  - ACS
  - Stimulants (caffeine, tobacco)
  - Medications (sympathomimetics, cyclic antidepressants, phenothiazines)

Ventricular Dysrhythmias (Cont’d)

Sinus Rhythm With VT
**Ventricular Dysrhythmias (Cont’d)**

- Ventricular escape beats/rhythms
  - After pause when supraventricular pacemaker failed to fire
  - Escape beat is late, appears after next sinus beat
  - Protects heart from more extreme slowing, systole

**Ventricular Dysrhythmias (Cont’d)**

- Ventricular escape beats/rhythms
  - Characteristics
    - **Rate**
      - Usually within range, depends on underlying rhythm
    - **Rhythm**
      - Essentially regular with late beats
      - Occurs after next expected sinus beat
    - **P waves**
      - Usually absent with retrograde conduction to atria
      - May appear after QRS

**Ventricular Dysrhythmias (Cont’d)**

- Ventricular escape beats/rhythms
  - Characteristics
    - **PR interval**
      - None
      - Ectopic beat originates in ventricles
    - **QRS duration**
      - >0.12 second
      - Wide and bizarre
      - T wave frequently in direction opposite to QRS complex
Ventricular Dysrhythmias (Cont’d)

- Ventricular escape beats/rhythms
  - Idioventricular rhythm (IVR) occurs when 3+ consecutive beats at 20-40 beats/min
  - Wide and bizarre

Ventricular Dysrhythmias (Cont’d)

- Ventricular escape beats/rhythms
  - Agonal rhythm
    - Ventricular rate slows to <20 beats/min
    - SA node and AV junction fail to initiate electrical impulse
    - Discharge rate of SA node, AV junction is less than the intrinsic ventricle rate
    - Blocked impulses generate by supraventricular pacemaker site
    - MI

Ventricular Dysrhythmias (Cont’d)

- Ventricular escape beats/rhythms
  - Agonal rhythm
    - Digitalis toxicity
    - Metabolic imbalances
    - With pulse and symptomatic from slow rate, transcutaneous pacing
    - Pulseless electrical activity (PEA)
    - Not breathing, no pulse
Ventricular Dysrhythmias (Cont’d)

- Ventricular escape beats/rhythms
  - Idioventricular rhythm characteristics
    - Rate
      - Usually within range, depends on underlying rhythm
    - Rhythm
      - Essentially regular with late beats
      - Occurs after next expected sinus beat
    - P waves
      - Usually absent with retrograde conduction to atria
      - May appear after QRS

- Ventricular escape beats/rhythms
  - Idioventricular rhythm characteristics
    - PR interval
      - None
        - Ectopic beat originates in ventricles
    - QRS duration
      - > 0.12 second
        - Wide and bizarre
        - T wave frequently in direction opposite to QRS complex

Ventricular Dysrhythmias (Cont’d)

Sinus Rhythm With Prolonged PR Interval
Ventricular Dysrhythmias (Cont’d)

- Accelerated idioventricular rhythm (AIVR)
  - 3+ ventricular escape beats occur consecutively at 41-100 beats/min
  - Nonparoxysmal VT
  - Benign escape rhythm
  - Appears when sinus rate slows down, disappears when sinus rate speeds up

- AIVR
  - Last few seconds to minute
  - Seen in first 12 hours of MI
  - After successful reperfusion therapy
  - No treatment
  - Hemodynamic compromise signs, lack of atrial kick
  - Atropine blocks vagus nerve, stimulates SA node to overdrive ventricular rhythm
Ventricular Dysrhythmias (Cont’d)

- AIVR
  - Characteristics
    - Rate
      - 41-100 beats/min
    - Rhythm
      - Essentially regular
    - P waves
      - Usually absent with retrograde conduction to atria
      - May appear after QRS

- AIVR
  - Characteristics
    - PR interval
      - None
    - QRS duration
      - >0.12 second
      - T wave frequently in direction opposite to QRS complex

- AIVR
  

Ventricular Dysrhythmias (Cont’d)
Ventricular Dysrhythmias (Cont’d)

- Ventricular tachycardia (VT)
  - 3+ PVCs occur consecutively at rate of >100 beats/min
  - If it occurs as short run <30 second, nonsustained VT
  - If it persists >30 second, sustained VT
  - Monomorphic VT

Nonsustained Ventricular Tachycardia

VT
- Monomorphic VT characteristics
  - Rate: 101-250 beats/min
  - Rhythm: Essentially regular
  - P waves: May be present/absent
  - If present, no set relation to QRS complexes appearing between QRSs at rate different from that of VT
Ventricular Dysrhythmias (Cont’d)

- VT
  - Monomorphic VT characteristics
    - PR interval
      - None
    - QRS duration
      - >0.12 second
        - Often difficult to differentiate between QRS and T wave

Ventricular Dysrhythmias (Cont’d)

- Monomorphic VT

Ventricular Dysrhythmias (Cont’d)

- VT
  - Causes
    - Acute coronary syndromes
    - Cardiomyopathy
    - Tricyclic antidepressant overdose
    - Digitalis toxicity
  - Valvular heart disease
  - Cocaine abuse
  - Mitral valve prolapsed
  - Acid-base imbalance
  - Trauma
  - Electrolyte imbalance
Ventricular Dysrhythmias (Cont’d)

- VT
  - May occur with or without pulses
  - SVT with intraventricular conduction delay
difficult to distinguish from VT

Ventricular Dysrhythmias (Cont’d)

- VT
  - Polymorphic VT
    - QRS complexes of VT vary in shape and amplitude from beat to beat
    - QRS complexes twist from upright to negative and negative to upright
    - Torsades de pointes

Ventricular Dysrhythmias (Cont’d)

- VT
  - Polymorphic VT characteristics
    - Rate:
      - 150-300 beats/min; typically 200-250 beats/min
    - Rhythm:
      - May be regular/irregular
    - P waves:
      - None
Ventricular Dysrhythmias (Cont’d)

VT
- Polymorphic VT characteristics
  - PR interval
    - None
  - QRS duration
    - >0.12 second
    - Gradual alteration in amplitude and direction of QRS complexes
    - Typical cycle has 5-20 QRS complexes

Ventricular Dysrhythmias (Cont’d)

Ventricular fibrillation (VF)
- Chaotic rhythm, begins in ventricles
- No organized depolarization on ventricle
- Ventricular muscle quivers
- No MI occurs
Ventricular Dysrhythmias (Cont’d)

- **VF**
  - Cardiopulmonary/cardiac arrest
    - Unresponsive
    - Not breathing
    - No pulse
    - Rhythms include VT, VF, asystole

- **VF**
  - Cardiopulmonary/cardiac arrest
    - **PEA**
      - ECG rhythm looks chaotic with deflections that vary in shape, amplitude
      - No normal-looking waveforms are visible
      - Coarse VF, with waves >3 mm high
      - Fine VF, with waves <3 mm high
      - Factors for heart muscle fibrillation

Ventricular Dysrhythmias (Cont’d)

- **VF**
  - Characteristics
    - **Rate**
      - Cannot be determined
      - No waves, complexes discernible to measure
    - **Rhythm**
      - Rapid, chaotic, no pattern or regularity
      - P waves
        - Not discernible
Ventricular Dysrhythmias (Cont’d)

- VF
  - Characteristics
    - PR interval
    - Not discernible
  - QRS duration
    - Not discernible

Ventricular Dysrhythmias (Cont’d)

Coarse VF

Ventricular Dysrhythmias (Cont’d)

Fine VF
Ventricular Dysrhythmias (Cont’d)

- Defibrillation
  - Delivery of electrical current across heart muscle
  - Done to terminate abnormal heart rhythm
  - Also called asynchronous countershock
  - Indications include
    - Polymorphic VT
    - Pulseless VT
    - Ventricular fibrillation
  - Causes momentary stunning of the heart, gives opportunity for heart’s natural pacemakers to resume normal activity

Skill 22-4 Defibrillation

- Standard precautions
- Verify procedure indicated
- Identify rhythm on cardiac monitor

Skill 22-4 Defibrillation (Cont’d)

- Charge defibrillator
- Make sure O₂ is not flowing over the chest
Skill 22-4 Defibrillation (Cont’d)
- Multipurpose adhesive electrodes on chest
- Place pregelled pads on chest
- Recheck ECG rhythm

Skill 22-4 Defibrillation (Cont’d)
- Call "Clear"
- Press shock control
- Recheck rhythm

Ventricular Dysrhythmias
- Asystole (cardiac standstill)
  - Total absence of ventricular electrical activity
  - No ventricular rate/rhythm, pulse, cardiac output
  - Some atrial electrical activity
Ventricular Dysrhythmias (Cont’d)

- Asystole
  - P-wave asystole
  - Atrial electrical activity present
  - Causes same as PEA

Ventricular Dysrhythmias (Cont’d)

- Asystole
  - Characteristics
    - Rate
      - Ventricular usually not discernible
      - Atrial activity may be seen
    - Rhythm
      - Ventricular not discernible
      - Atrial may be discernible
    - P waves
      - Usually not discernible

Ventricular Dysrhythmias (Cont’d)

- Asystole
  - Characteristics
    - PR interval
      - Not measurable
    - QRS duration
      - Absent
Ventricular Dysrhythmias (Cont’d)

Asystole

Atrioventricular Blocks

- Delay/interruption in impulse conduction within AV node, bundle of His, His-Purkinje system, resulting dysrythmia
- PR interval differentiates AV block type
  - QRS complex width
  - In second- and third-degree blocks, rate of escape rhythm

P Wave Asystole
Atrioventricular Blocks (Cont’d)

AV Block Locations

First-degree block
- All components of ECG tracing within normal limits except PR interval
- Electrical impulses travel normally from SA node through atria, impulse conduction delay at AV node level
- Normal finding with no cardiac disease history

First-degree block
- Causes
  - Ischemia/injury to AV node/junction
  - Medications, amiodarone, procainamide, beta-blockers
  - Rheumatic heart disease
  - Hyperkalemia
  - Acute MI
  - Increased vagal tone
Atrioventricular Blocks (Cont’d)

• First-degree block
  Characteristics
  • Rate
    ▶ Usually within normal range
    ▶ Depends on underlying rhythm
  • Rhythm
    ▶ Regular
  • P waves
    ▶ Normal in size and shape
    ▶ One positive P wave before each QRS in leads II, III, aVF

Atrioventricular Blocks (Cont’d)

• First-degree block
  Characteristics
  • PR interval
    ▶ Prolonged but constant
  • QRS duration
    ▶ Usually 0.10 second/less unless intraventricular conduction delay

Atrioventricular Blocks (Cont’d)

First-Degree AV Block
Atrioventricular Blocks (Cont’d)

- Second-degree block, type I
  - Mobitz type I/Wenckebach
  - Progressive lengthening of PR interval followed by P wave with no QRS complex
  - Conduction delay occurs at AV node level
  - RCA occlusions, associated with AV block occurring in AV node

Atrioventricular Blocks (Cont’d)

- Second-degree block, type I
  - Asymptomatic, ventricular rate nearly normal, cardiac output not significantly affected
  - Slow heart rate, atropine
  - With acute MI, observe for increasing AV block

Atrioventricular Blocks (Cont’d)

- Second-degree block, type I
  - Characteristics
    - Rate
      - Atrial rate is greater than ventricular rate
    - Rhythm
      - Atrial regular
      - Ventricular irregular
    - P waves
      - Normal in size and shape
      - Some P waves not followed by QRS complex
Atrioventricular Blocks (Cont’d)

- Second-degree block, type I
  - Characteristics
    - PR interval
      - Lengthens with each cycle until P wave appears without QRS complex
      - PR interval after nonconducted beat shorter than interval preceding nonconducted beat
      - QRS duration
        - Usually 0.10 second/less, periodically dropped

- Second-degree block, type II
  - Conduction delay occurs below AV node
  - More serious than type I, progresses to third degree
  - Ventricular rate normal, asymptomatic
  - Pacing
  - Dopamine / Epinephrine / Isoproterenol drip
Atrioventricular Blocks (Cont’d)

- Second-degree block, type II
  - Characteristics
    - Rate
      - Atrial rate is greater than ventricular rate
      - Ventricular rate often slow
    - Rhythm
      - Atrial regular
      - Ventricular irregular
    - P waves
      - Normal in size and shape
      - Some P waves not followed by QRS complex

Atrioventricular Blocks (Cont’d)

- Second-degree block, type II
  - Characteristics
    - PR interval
      - Within normal limits, slightly prolonged
      - Constant for conducted beats
      - Some shortening of PR interval may follow nonconducted P wave
    - QRS duration
      - Usually 0.10 second/less, periodically absent after P waves

Atrioventricular Blocks (Cont’d)

Second-Degree AV Block Type II
Atrioventricular Blocks (Cont’d)

- Third-degree block
  - Impulses generated by SA node blocked before reaching ventricles, P waves conducted
  - Atria and ventricles beat independently
  - Complete AV block

Atrioventricular Blocks (Cont’d)

- Third-degree block
  - Occurs at AV node, bundle of His, bundle branches
  - Secondary pacemaker stimulates ventricles
  - With inferior MI, result of block above bundle of His

Atrioventricular Blocks (Cont’d)

- Third-degree block
  - With anterior MI, preceded by second-degree AV block type II/intraventricular conduction delay
  - Rhythm usually unstable, escape pacemaker ventricular, with rate <40 beats/min
Atrioventricular Blocks (Cont’d)

• Third-degree block
  • Characteristics
    - Rate
      - Atrial rate is greater than ventricular rate
    - Rhythm
      - Atrial regular
      - Ventricular regular
      - No relation between atrial and ventricular rhythms
  - P waves
    - Normal in size and shape

Atrioventricular Blocks (Cont’d)

• Third-degree block
  • Characteristics
    - PR interval
      - None
      - Atria and ventricles beat independently, no true PR interval
    - QRS duration
      - Narrow/wide depending on escape pacemaker
      - Narrow = junctional pacemaker
      - Wide = ventricular pacemaker
Pacemaker Rhythms

- Pacemaker
  - Artificial pulse generator
  - Delivers electrical current to heart
  - Stimulates depolarization
  - Named to where electrodes are located, route of electrical current to heart
  - Pulse generator
  - Pacing leads

Pacemaker Rhythms (Cont’d)

- Permanent pacemakers
  - Implanted in body
  - Wires surrounded by plastic catheters
  - Circuitry housed in hermetically sealed titanium case

Pacemaker Rhythms (Cont’d)
Pacemaker Rhythms (Cont’d)

- Temporary pacemakers
  - Transvenous
    - Stimulate endocardium of right atrium/ventricle
    - Electrode into central vein

Pacemaker Rhythms (Cont’d)

- Epicardial
  - Placing pacing leads directly onto/through epicardium
  - During surgery
  - Transcutaneous

Pacemaker Rhythms (Cont’d)

- Pacemaker modes
  - Fixed-rate (asynchronous) pacemakers
  - Demand (synchronous, noncompetitive) pacemakers
Pacemaker Rhythms (Cont’d)

- Single-chamber pacemakers
  - Paces atrium/ventricle chamber
  - One lead placed in heart
  - Atrium
    - Electrode in right atrium
    - Stimulation produces spike followed by P wave
    - Used when SA node is diseased, damaged, normal conduction through AV junction and ventricles
    - Ineffective if AV block develops

Pacemaker Rhythms (Cont’d)

Atrial Pacing

Pacemaker Rhythms (Cont’d)

- Single-chamber pacemakers
  - Ventricle
    - Electrode in right ventricle
    - Stimulation produces spike followed by wide QRS
Pacemaker Rhythms (Cont’d)

Ventricular Pacing

- Dual-chamber pacemakers
  - Paces both atrium and ventricle
  - Two-lead system placed in heart

- AV sequential pacemaker
  - Stimulates right atrium first and right ventricle sequentially
  - Mimics normal cardiac physiology, preserves atrial contribution to ventricular filling
Pacemaker Rhythms (Cont’d)

AV Sequential Pacing

Implanted pacemaker malfunction
- Failure to pace
  - Malfunction, fails to deliver correct number of electrical stimulations/minute
  - On ECG, pacemaker spike absence
- Signs/symptoms
  - Syncope
  - Chest pain
  - Bradycardia
  - Hypotension

Causes
- Battery failure
- Pacing lead wire fracture
- Electrode tip displacement
- Pulse generator failure
- Broken/loose connection between lead and pulse generator
- Electromagnetic interference
- Sensitivity setting too high
Pacemaker Rhythms (Cont'd)

- Implanted pacemaker malfunction
  - Failure to pace
    - Treatment
      - Adjust sensitivity setting
      - Replace pulse generator battery
      - Replace lead
      - Replace pulse generator unit
      - Tighten connections
      - Perform electrical check
      - Removing source of electromagnetic interference

- Implanted pacemaker malfunction
  - Failure to capture
    - Inability of pacemaker stimulus to depolarize myocardium
    - Visible pacemaker spikes followed by P waves/QRS complexes

- Implanted pacemaker malfunction
  - Causes
    - Recent defibrillation
    - Battery failure
    - Lead wire fracture
    - Lead wire displacement
    - Myocardium perforation by lead wire
    - Swelling/scar tissue formation at electrode tip
    - Output energy set too low

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Pacemaker Rhythms (Cont’d)

- Implanted pacemaker malfunction
  - Failure to capture
    - Treatment
      - Repositioning patient
      - Slowly increase output setting until capture occurs/proximal setting reached
      - Replace pulse generator battery
      - Replacing/repositioning lead
      - Surgery

Pacemaker Rhythms (Cont’d)

Failure to Capture

Pacemaker Rhythms (Cont’d)

- Implanted pacemaker malfunction
  - Failure to sense (undersensing)
    - Fails to recognize ECG waveforms
    - On ECG, pacemaker spike follows too closely behind QRS complexes
    - R-on-T phenomenon/competition between pacemaker and patient’s own cardiac rhythm
Pacemaker Rhythms (Cont’d)

- Implanted pacemaker malfunction
  - Causes
    - Battery failure
    - Lead wire fracture
    - Electrode tip displacement
    - Loose connections
    - Recent defibrillation
  - Decreased P wave/QRS voltage
  - Circuitry dysfunction
  - Antiarrhythmic medications
  - Severe electrolyte disturbances

- Treatment
  - Increase sensitivity setting
  - Replace pulse generator battery
  - Replace/reposition lead

Pacemaker Rhythms (Cont’d)

- Implanted pacemaker malfunction
  - Oversensing
    - Inappropriate sensing of unrelated electrical signals
    - Pacemaker spike at slower rate than pacemaker’s preset rate/no paced beats
    - Patient should avoid strong electromagnetic fields
    - Treatment, adjust sensitivity setting
Pacemaker Rhythms (Cont’d)

1. Pacemaker-induced tachycardia
   - Uncommon
   - In dual-chamber pacemaker
   - PVC/depolarization conducted backward through AV node to atrium

2. Findings
   - “Racing heart”
   - Lightheadedness
   - Syncope
   - Chest discomfort

3. Treatment
   - Apply magnet over device, reprogram pacemaker
Pacemaker Rhythms (Cont’d)

- Pacemaker artifact
  - Do not rely on cardiac monitor’s heart rate display with implanted/subcutaneous pacemaker
  - Some monitors and defibrillators are equipped with internal pacemaker pulse detector

Pacemaker Rhythms (Cont’d)

- Transcutaneous pacing
  - Use of electrical stimulation through pacing pads positioned on torso, stimulate contraction of heart
  - Current delivered is less that that used for cardioversion/defibrillation
  - Stimulating current, milliamperes
  - Electrical signal exits from negative terminal on the machine, passes through the chest wall to the heart

Pacemaker Rhythms (Cont’d)

- Transcutaneous pacing
  - Indications
    - Bradycardias unresponsive to atropine therapy
    - When atropine is not immediately available/indicated
    - Used as bridge until transvenous pacing is accomplished, bradycardia cause reversed
Pacemaker Rhythms (Cont’d)

- Transcutaneous pacing
  - Complications
    - Coughing
    - Skin burns
    - Interference with sensing from patient agitation, muscle contractions
    - Pain from electrical stimulation

Pacemaker Rhythms (Cont’d)

- Transcutaneous pacing
  - Complications
    - Failure to recognize pacemaker is not capturing
    - Failure to recognize underlying treatable VF
    - Tissue damage, third-degree burns
    - Prolonged pacing, threshold changes, capture failure

Skill 22-5 Transcutaneous Pacing

- O₂, vital signs, IV access
- Apply ECG electrodes
- Identify, verify paceable rhythm
Skill 22-5 Transcutaneous Pacing (Cont'd)

- Apply adhesive placing pads

Skill 22-5 Transcutaneous Pacing (Cont'd)

- Connect pacing cable to pacemaker, pads on patient
- Power on
- Set pacing rate

Skill 22-5 Transcutaneous Pacing (Cont'd)

- After rate is regulated, start the pacemaker
- Increase current slowly until capture is achieved
Skill 22-5 Transcutaneous Pacing (Cont'd)

- Watch for electrical capture
  - Wide QRS & broad T wave

- Assess mechanical capture
- Continue pacing at output level slightly higher than initial electrical capture

- Assess BP, SpO₂, responsiveness level
- Document, record ECG rhythm
Overview of 12-Lead ECG

- Views heart in frontal, horizontal planes
- Views surfaces of left ventricle from 12 angles
  - Recognition of bundle blocks
  - ID of ST-segment and T-wave changes with myocardial ischemia, injury, infarction
  - ID of ECG changes from medications, electrolyte imbalances

Overview of 12-Lead ECG (Cont'd)

- Indications
  - Chest pain/discomfort
  - Assisting in dysrhythmia interpretation
  - Right/left ventricle failure
  - Status before/after electrical therapy

Overview of 12-Lead ECG (Cont'd)

- Indications
  - Syncope, near syncope
  - Electrical injuries
  - Stroke
  - Unstable patient with unknown etiology
  - Known/suspected overdoses
  - Known/suspected electrolyte imbalances
**Skill 22-6 Chest Lead Placement**

- Place finger at notch at top of the sternum
- Move finger slowly downward until slight horizontal ridge/elevation

**Skill 22-6 Chest Lead Placement (Cont'd)**

- Follow angle of Louis to patient's right until articulates with second rib
- Locate second intercostal space

**Skill 22-6 Chest Lead Placement (Cont'd)**

- From second intercostal space, third and fourth intercostal spaces can be found
- V, positioned in fourth intercostal space just to right of the sternum
Skill 22-6 Chest Lead Placement (Cont'd)

- From V₁ position, find corresponding intercostal space on left side of the sternum

Skill 22-6 Chest Lead Placement (Cont'd)

- Place V₂ electrode in fourth intercostal space just to left of the sternum
- From V₂ position, locate fifth intercostal space, follow to midclavicular line

Skill 22-6 Chest Lead Placement (Cont'd)

- Position V₄ electrode in fifth intercostal space in midclavicular line
- V₅ positioned halfway between V₂ and V₄
Skill 22-6 Chest Lead Placement (Cont'd)

- V₆ positioned in midaxillary line, level with V₄
- V₅ positioned in anterior axillary line, level with V₄

Skill 22-7 Right Chest Lead Placement

- Move cables for standard chest leads to electrodes for additional leads
- Place electrode for V₁R in fourth intercostal space, left of the sternum

Skill 22-7 Right Chest Lead Placement (Cont'd)

- From V₁R position, find the corresponding intercostal space on right side of the sternum
- Place V₂R electrode in fourth intercostal space, right of the sternum
Skill 22-7 Right Chest Lead Placement (Cont’d)

- From V₂R position, move fingers down, find the fifth intercostal space, follow to the midclavicular line
- Place V₄R electrode in fifth intercostal space

Skill 22-7 Right Chest Lead Placement (Cont’d)

- Imagine line between V₂R and V₄R
- Position the V₃R electrode halfway between V₂R and V₄R on an imaginary line

Skill 22-7 Right Chest Lead Placement (Cont’d)

- Electrode for V₆R position in right midaxillary line, level with V₄R
- Position V₅R in right anterior axillary line, level with V₄R
Skill 22-7 Right Chest Lead Placement (Cont’d)
- Once leads are printed, correct lead must be handwritten onto ECG to indicate origin of tracing
- Clearly label ECG “right chest ECG”

Skill 22-8 Posterior Chest Lead Placement
- Obtain and print standard 12-lead ECG
- Locate landmarks for posterior leads
- Position patient on side
- Place V₇ electrode on fifth intercostal space
- Attach V₄ lead wire to V₇ electrode
Skill 22-8 Posterior Chest Lead Placement (Cont’d)

- Place V8 on fifth intercostal space of the left midscapular line
- Attach V5 lead wire to V8 electrodes

Skill 22-8 Posterior Chest Lead Placement (Cont’d)

- Place V9 just left of the spinal column at fifth intercostal space
- Attach V6 lead wire to V9 electrode

Skill 22-8 Posterior Chest Lead Placement (Cont’d)

- Obtain and print 12-lead ECG
- Clearly label, “posterior chest ECG”
Overview of 12-Lead ECG

- Axis
  - Leads I, II, III form an equilateral triangle with heart at the center
  - Hexaxial reference system
    - Add limb leads, bisect each other
    - Represents all frontal plane leads
    - Heart in center
    - Expresses location of frontal plane

Overview of 12-Lead ECG (Cont'd)

- Axis
  - Hexaxial reference system
    - 360° circle surrounding the heart
    - Positive end of lead I at 0°
    - Six frontal plane leads divide circle into 30° segments
    - All degrees in upper hemisphere, negative
    - All degrees in lower hemisphere, positive
    - Normal electrical axis between 0 and 90°

Overview of 12-Lead ECG (Cont'd)

- Axis
  - Current flow to right of normal
  - Current flow in opposite direction of normal
  - Current flow to left of normal
  - Leads I, aVF divide heart into four quadrants
Overview of 12-Lead ECG (Cont'd)

- Analyzing 12-lead ECG
  - Assess quality of tracing
  - Identify rate, underlying rhythm
  - Evaluate intervals

- Evaluate waveforms
- Examine each lead for ST-segment displacement
- Determine axis
- Evidence for hypertrophy/chamber enlargement
- Medication effects, electrolyte imbalances
- Interpret findings
Overview of 12-Lead ECG (Cont'd)

- Localization of infarctions
  - Contiguous leads
    - Indicative changes
      - Changes of myocardial ischemia, injury, infarction found in 2+ looking directly at affected area
      - ST-segment elevated at least 1 mm, MI
      - Significant, seen in two anatomically contiguous leads
  - Reciprocal changes
    - Findings in leads opposite affected area
Overview of 12-Lead ECG (Cont'd)

- Localization of infarctions
  - Predicting site and extent of coronary artery occlusion
    - Leads I, aVL, V1-V6 evaluate tissue supplied by left coronary artery

- Potential problems
  - Lateral wall infarction on ECG may be anterior wall infarction
  - Factors may affect perceived location
  - Patient's unique coronary artery distribution pattern and collateral circulation presence can affect infarct location
Epicardial Coronary Arteries

- Localization of infarctions
  - Anterior wall infarctions
    - Leads V3 and V4 face left ventricle anterior wall
    - Left main coronary artery supplies LAD and CX arteries, most of bundle branch tissue
    - LAD artery blockage
    - Anterior wall MI
Overview of 12-Lead ECG (Cont'd)

Extensive Anterior Infarction

Overview of 12-Lead ECG (Cont'd)

- Localization of infarctions
  - Inferior wall infarctions
    - Leads II, III, aVF view left ventricle inferior surface
    - Left ventricle inferior wall supplied by posterior descending branch of RCA
    - Increased PNS activity is common with inferior wall MIs, bradydysrhythmias
    - Conduction delays are usually transient

Overview of 12-Lead ECG (Cont'd)

Inferior Wall Infarction
Overview of 12-Lead ECG (Cont'd)

Inferior Wall Infarction

- Leads II, III, and aVF show ST elevation or depression.
- ST elevation in these leads indicates inferior wall infarction.

Localization of infarctions

- Lateral wall infarctions:
  - Leads I, aVL, V5, V6 view left ventricle lateral wall.
  - Left ventricle lateral wall supplied by CX/LAD arteries/RCA branch.

Overview of 12-Lead ECG (Cont'd)

Lateral Wall Infarction

- Leads I, aVL, V5, V6 show ST elevation.
- ST elevation in these leads indicates lateral wall infarction.

-CX/LAD arteries/RCA branch supply the lateral wall of the left ventricle.

Overview of 12-Lead ECG (Cont'd)

-Localization of infarctions:
  - Lateral wall infarctions:
    - Leads I, aVL, V5, V6 view left ventricle lateral wall.
    - Left ventricle lateral wall supplied by CX/LAD arteries/RCA branch.
Overview of 12-Lead ECG (Cont’d)

- Localization of infarctions
  - Septal infarctions
    - Leads V1, V2 face left ventricle septal area
    - Septum supplied by LAD artery
    - If infarction site is limited to septum, V1-V2 changes
    - If entire anterior wall, V1-V4 changes

- Posterior wall infarctions
  - CX artery supplies left ventricle posterior wall in most patients
  - No leads of 12-lead view posterior wall, additional chest leads used
  - Complications
    - Left ventricular dysfunction
    - Dysrhythmias involving SA node, AV node, bundle of His
Localization of infarctions
  - Right ventricular infarctions
    - Supplied by right ventricular marginal branch of RCA
    - Right ventricular marginal branch occlusion, RVI
    - Occlusion of RCA proximal to right ventricular
      marginal branch, inferior and RVI
  - RVI suspected when ECG changes suggest inferior
    infarction
  - Clinical evidence in 10-15% of patients
  - Left ventricular output reduction decreases BP
Overview of 12-Lead ECG (Cont'd)

- Localization of infarctions
  - Right ventricular infarctions
    - Complications
      - Hypotension
      - Cardiogenic shock
      - AV blocks
      - Atrial flutter/fibrillation
      - PACs

Overview of 12-Lead ECG (Cont'd)

Right Ventricular Infarction

Overview of 12-Lead ECG (Cont'd)

Inferior Infarction, RVI
Intraventricular Conduction Delays

- Bundle branch activation
  - Normal ventricular depolarization
    - Interventricular septum left side is stimulated first
    - Electrical impulse transverses septum to stimulate right side
    - Right bundle branch conducts impulse to right ventricle

Intraventricular Conduction Delays (Cont’d)

- Bundle branch activation
  - Normal ventricular depolarization
    - Left bundle branch conducts impulse to left ventricle
    - Left and right ventricles are depolarized at same time
    - Impulse travels down unblocked branch, stimulates the ventricle

Intraventricular Conduction Delays (Cont’d)

- Bundle branch activation
  - Normal ventricular depolarization
    - Block forces impulse to travel from cell to cell, through myocardium to stimulate the other ventricle
    - Conduction slower, QRS complex widens
    - Ventricle with blocked bundle is last to be depolarized
Intraventricular Conduction Delays (Cont'd)

Determining Direction of Terminal Force

- ECG criteria
  - ID of right/left bundle branch block
  - Select widest QRS complex with discernible beginning and end
  - V1 single best lead when differentiating between right/left BBB

Turn Signal Theory
Intraventricular Conduction Delays
(Cont’d)

ECG criteria

Right BBB
- Electrical impulse travels through AV node, down left bundle branch into interventricular septum
- Septum activated by left posterior fascicle, depolarized in left-to-right direction
- When two BBB criteria are met and V1 displays RSR' pattern, right BBB

Intraventricular Conduction Delays
(Cont’d)

ECG criteria

Left BBB
- Septum and right ventricle depolarized by right bundle branch
- Current away from V1
- Produces QS pattern in V1

Intraventricular Conduction Delays
(Cont’d)

ECG criteria

Differentiation is difficult
- Resembles qR or rS pattern
- Focus on terminal force of QRS complex
ECG Changes for Electrolyte Disturbances and Hypothermia

- Electrolyte disturbances
  - Hypocalcemia
    - ST segment long, flattened
    - QT interval prolonged

ECG Changes for Electrolyte Disturbances and Hypothermia (Cont’d)

- Electrolyte disturbances (Cont’d)
  - Hypercalcemia
    - PR interval prolonged
    - ST segment shortened
    - QT interval shortened

ECG Changes for Electrolyte Disturbances and Hypothermia (Cont’d)

- Electrolyte disturbances
  - Hypokalemia
    - QRS complex widens as level decreases
    - ST segment depressed
    - T wave flattened
    - U wave present
    - QT interval prolonged
ECG Changes for Electrolyte Disturbances and Hypothermia (Cont'd)

- Electrolyte disturbances
  - Hyperkalemia
    - P wave disappears as level increases
    - PR interval normal/prolonged
    - QRS complex widens as level increases
    - ST segment disappears as level increases
    - T wave tall, peaked, tented
    - Heart rate slows

- Hypomagnesemia
  - P wave diminished voltage
  - QRS complex widens as level decreases, diminished voltage
  - ST segment depressed
  - T wave flattened
  - U wave present
  - QT interval prolonged

- Hypermagnesemia
  - PR interval prolonged
  - QRS complex widened
  - T wave tall/elevated
ECG Changes for Electrolyte Disturbances and Hypothermia (Cont’d)

- Hypothermia
  - Dysrhythmias
  - PR interval prolonged
  - QRS duration prolonged
  - QT interval prolonged
  - Core body temperature

Osborn Waves
Initial Assessment

- Scene safety
- Number of patients, additional resources needed

Initial Assessment (Cont'd)

- Primary survey
  - General impression
    - Sight and hearing to assess life-threatening problem
    - Appearance
    - Work of breathing
    - Circulation

- Airway, cervical spine protection
  - Ask questions, answers provide information about condition of airway, mental status
  - Responsive, airway open, evaluate breathing
  - Responsive, cannot talk/cry/cough forcefully, airway obstruction
Initial Assessment (Cont’d)

- Primary survey
  - Airway, cervical spine protection
    - Unresponsive
    - Jaw thrust maneuver without head tilt
    - Airway compromise signs
    - Clear airway, suction
    - Obstructed, not visualized, clear per resuscitation guidelines
    - Insert oral/nasal airway

Initial Assessment (Cont’d)

- Primary survey
  - Breathing, ventilation
    - Breathing adequate/inadequate
      - Increased work of breathing signs
        - Nasal flaring
        - Pursed-lip breathing
        - Accessory muscle use
        - Leaning forward to inhale, retractions
    - Respiratory rate
    - Chest rise/fall, expansion = enough tidal volume to rise

Initial Assessment (Cont’d)
Initial Assessment (Cont’d)

• Primary survey
  ➢ Breathing, ventilation
    ➢ Before stethoscope
      ➢ Breathing quiet, absent, noisy
      ➢ Stridor
      ➢ Wheezing
      ➢ Snoring
      ➢ Gurgling

Initial Assessment (Cont’d)

• Primary survey
  ➢ Breathing, ventilation
    ➢ Presence/absence of breath sounds
      ➢ Clear, equal bilaterally
      ➢ Listen for crackles, pulmonary congestion
    ➢ O₂
      ➢ Absent, begin rescue breathing

Initial Assessment (Cont’d)

• Primary survey
  ➢ Circulation with bleeding control
    ➢ Visible external hemorrhage, control
    ➢ Pulse presence, rate, quality
    ➢ Skin color, temperature, moisture, turgor, mobility
Initial Assessment (Cont’d)

- Primary survey
  - Disability (mental status)
    - Glasgow Coma Scale, establish baseline

Initial Assessment (Cont’d)

- Primary survey
  - Expose/environment
    - Preserve body heat, undress as needed
      - Respect modesty, replace promptly after examination
  - Make transport decision
  - Reassess frequently

Initial Assessment (Cont’d)

- Secondary assessment
  - SAMPLE history
  - Signs/symptoms
    - Chest pain/discomfort
      - Onset
      - Quality
      - Region/radiation
      - Severity
      - Time
Initial Assessment (Cont’d)

- Secondary assessment
  - Signs/symptoms
    - Dyspnea
      - Uncomfortable awareness of breathing
      - Abnormal when at rest
    - Headache
    - Fever, chills
    - Chest pain
    - Pain, swelling in lower legs
    - Throat tightness

Initial Assessment (Cont’d)

- Secondary assessment
  - Signs/symptoms
    - With inspiration, upper airway obstruction
    - With expiration, lower airway obstruction
    - On exertion/at rest
    - Sudden

Initial Assessment (Cont’d)

- Secondary assessment
  - Signs/symptoms
    - Relieved by squatting, caused by tetralogy of Fallot
    - Failing pump sign
    - Orthopnea
    - Paroxysmal nocturnal dyspnea (PND)
Initial Assessment (Cont’d)

- Secondary assessment
  - Signs/symptoms
    - Cough
      - Causes
      - Symptom
      - Chronic, 3+ weeks
      - Complications
      - Sputum production

Initial Assessment (Cont’d)

- Secondary assessment
  - Signs/symptoms
    - Fainting
      - Syncope
      - Brief LOC by temporary decreased blood flow to brain
      - Causes
      - Cardiac causes

Initial Assessment (Cont’d)

- Secondary assessment
  - Signs/symptoms
    - Fainting
      - Patient’s position, activity level before
      - Duration of underresponsiveness
      - Involuntary movements
      - Duration of confusion/disorientation after awakening
      - Fall, other trauma
      - Medical history, medications, previous fainting episodes
      - Medical ID, diabetes, seizures
Initial Assessment (Cont'd)

- Secondary assessment
  - Signs/symptoms
    - Abnormal heartbeat or palpitations
    - Patient descriptions
    - Causes
    - Associated symptoms

- Secondary assessment
  - Signs/symptoms
    - Fatigue
      - Common with impaired cardiovascular function
      - Vague causes
      - Onset, duration
      - Associated symptoms

- Secondary assessment
  - Signs/symptoms
    - Allergies
      - Medications
      - Food
      - Environmental elements
Initial Assessment (Cont’d)

- Secondary assessment
  - Signs/symptoms
    - Medications
      - Current
      - Document
      - Bring containers to hospital
      - Compliance
      - Common cardiac medications
  - Medications
  - Herbal supplements
  - Recreational drugs
  - Sildenafil use in previous 24-48 hours
  - Nonprescribed medications

Initial Assessment (Cont’d)

- Secondary assessment
  - Past medical history
    - Current physician care
    - History of heart attack, angina, heart failure, high BP, abnormal heart rhythm
    - Heart-related medical procedure history
Initial Assessment (Cont’d)

- Secondary assessment
  - Past medical history
    - History of stroke, diabetes, lung, liver, kidney disease
    - Risk factors for heart disease
    - Recent hospitalizations, surgeries

- Vital signs and physical examination
  - Vital signs, pulse oximetry
  - Respiratory rate, effort
  - Pulse

- Last oral intake
- Events leading to incident
Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Cardiac monitor
    - 12-lead indications
    - Assisting in dysrhythmia interpretation
    - Chest pain or discomfort
    - Anginal equivalents
    - Stroke
    - Syncope or near syncope
    - Right or left ventricular failure
    - Known or suspected electrolyte imbalances
    - Known or suspected overdoses
    - Blunt chest trauma
    - Electrical injuries
    - Before and after electrical therapy

Initial Assessment (Cont’d)

- Vital signs and physical examination
  - BP <90 mm Hg, low
    - Cardiogenic shock
  - Elevated BP
    - Aortic dissection
    - Heart failure
    - Stroke

Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Look (inspection)
    - Low cardiac output, inadequate tissue perfusion
    - Heart attack
    - Infection, pericarditis
Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Look (inspection)
    - Pain response
    - Restlessness
    -flushed skin
    - Increased heart rate
    - Increased respiratory rate
    - Elevated BP

Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Look (inspection)
    - Neck veins, jugular vein distention
      - Sitting, 45° angle, JVD present, abnormal
      - Activity of heart’s right side

Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Look (inspection)
    - Chest
      - Scar in center, previous heart surgery
      - Shape, barrel chest, COPD
      - Increased respiratory effort, retractions
Initial Assessment (Cont’d)

• Vital signs and physical examination
  ➢ Look (inspection)
    ➢ Abdomen
      ➢ Pulsations in epigastric area, aortic aneurysm
      ➢ Abdominal distention

Initial Assessment (Cont’d)

• Vital signs and physical examination
  ➢ Look (inspection)
    ➢ Edema
      ➢ Extremities
      ➢ Lower back, pitting

Initial Assessment (Cont’d)

• Vital signs and physical examination
  ➢ Look (inspection)
    ➢ Other signs of cardiac disease
      ➢ Nitroglycerin patch
      ➢ Signs bulge under skin
      ➢ Nail clubbing
Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Listen (auscultation)
    - Breath sounds
      - Apexes, bases
        - Present, diminished, absent
        - Equal, unequal
        - Clear, noisy
        - Crackles, rhonchi, wheezes, gurgling
        - Foamy, blood-tinged sputum
    - Heart sounds
      - Quiet environment
      - Vibrations in heart tissues from heart valves closing
      - Difficult to hear in field
      - S1 and S2, normal heart sounds
      - First sound, "lubb," low-pitched, dull
      - Second sound, "dubb," short, high-pitched, louder than S1
      - Third sound, normal in children, young adults

Anterior Chest View
Initial Assessment (Cont’d)

• Vital signs and physical examination
  ➢ Listen (auscultation)
    • Carotid sinus massage
    • Dysrhythmia treatment
    • Listen for bruits in carotid arteries
    • Contraindicated if bruit is present

Initial Assessment (Cont’d)

• Vital signs and physical examination
  ➢ Feel (palpation)
    • Tenderness, crepitus
    • Palpate abdomen for pulsations, tenderness

Initial Assessment (Cont’d)

Arterial Pulse Palpation
Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Feel (palpation)
    - Peripheral pulses
      - Rate
      - Rhythm
      - Strength
      - Equality

Initial Assessment (Cont’d)

- Vital signs and physical examination
  - Feel (palpation)
    - Apical before medication administration
      - Pulse deficit
      - Pulsus alternans

Initial Assessment (Cont’d)

Pulse Abnormalities
Initial Assessment (Cont'd)

- Vital signs and physical examination
  - Feel (palpation)
    - Peripheral pulses
      - Electrical alternans
  - Pulsus paradoxus
    - Systolic BP falls more than 10 mm Hg with inspiration
    - Mechanisms
    - Indications
    - Dissected thoracic aorta

- Vital signs and physical examination
  - Pulse pressure
    - Stroke volume, arterial elasticity
    - Difference between systolic and diastolic BP
    - Normal = 30-40 mm Hg
    - Widened seen in later stages of shock
    - Narrowed seen in tachycardia, cardiac tamponade
Reassessment

- En route to facility
- Primary complaint
- Medications, monitor response
- Breath sounds

Reassessment (Cont’d)

- ECG
- Vital signs
- Compare to baseline

Cardiovascular Disorders
Acquired Myocardium Diseases

- Acute coronary syndromes
  - Decreased supply of oxygenated blood to body part/organ
  - Temporary/permanent coronary artery blockage
  - Myocardial ischemia/injury
  - Heart muscle death

(Cont’d)

- Acute coronary syndromes
  - Unstable angina
  - Non-ST-segment elevation MI
  - ST-segment elevation MI
  - Sudden cardiac death can occur

(Cont’d)

- Acute coronary syndromes
  - Usual cause
    - Atherosclerotic plaque rupture
  - Arteries have three layers
    - Tunica adventitia
    - Tunica media
    - Tunica intima
Acquired Myocardium Diseases (Cont'd)
Atherosclerosis in Aorta

Acquired Myocardium Diseases (Cont'd)
Vulnerable Plaque

Acquired Myocardium Diseases (Cont'd)
- Acute coronary syndromes
  - Arteriosclerosis
    - Abnormal thickening, hardening of vessel walls
  - Atherosclerosis
  - Atherosclerotic plaques
Acquired Myocardium Diseases
(Cont'd)

- Acute coronary syndromes
  - Angina pectoris
    - Chest discomfort when heart muscle does not receive enough O₂
    - Ischemia from increased myocardial O₂ demand/reduced myocardial O₂ supply
    - Coronary artery disease, uncontrolled high BP, valvular heart disease

Acquired Myocardium Diseases
(Cont'd)

- Acute coronary syndromes
  - Stable angina
    - Constant, predictable in severity, signs/symptoms, precipitating events, treatment response
    - Shortness of breath
    - Palpitations
    - Sweating
    - Nausea, vomiting
    - Lasts 2-5 minutes
    - Prolonged uncommon

Acquired Myocardium Diseases
(Cont'd)

- Acute coronary syndromes
  - Unstable angina
    - Intermediate severity between stable angina and acute MI
    - Symptoms occur at rest ≥20 minutes
    - Symptoms severe and/or of new onset
    - Symptoms that are increasing in:
      - Intensity
      - Duration
      - Frequency
Acquired Myocardium Diseases  
(Cont’d)

- Acute coronary syndromes
  - Prinzmetal’s angina
    - Unstable angina form
    - Intense spasm of coronary artery segment
    - At rest, night/early morning
    - Chest discomfort
    - Difficulty breathing
    - Palpitations

- MI
  - Ischemia prolonged more than a few minutes causes myocardial injury
  - Will die if not corrected
  - Fibrinolytics, coronary angioplasty, coronary artery bypass graft
  - Results from thrombus

- MI and trauma
  - Blunt injury causes myocardial concussion
    - Commotio cordis
    - Myocardial contusion causes bruising
    - Cardiac rupture
Acquired Myocardium Diseases
(Cont’d)

- Acute coronary syndromes
  - MI
    - Ischemic Complications
      - Angina
      - Reinfarction
      - Infarct extension
    - Electrical Complications
      - Bradydysrhythmias
      - Tachydysrhythmias
      - AV blocks
      - Bundle branch and fascicular blocks
      - Sudden cardiac death
  - Mechanical Complications
    - Ventricular aneurysm
    - Ventricular septal rupture
    - Papillary muscle disorders
    - Cardiac wall rupture
    - Right or left ventricular failure
  - Inflammatory Complications
    - Pericarditis
  - Embolic Complications
    - Stroke
    - DVT
    - PE

Acquired Myocardium Diseases
(Cont’d)

- Acute coronary syndromes
  - MI
    - Mechanical Complications
    - Ventricular aneurysm
    - Ventricular septal rupture
    - Papillary muscle disorders
    - Cardiac wall rupture
    - Right or left ventricular failure
  - Inflammatory Complications
    - Pericarditis
  - Embolic Complications
    - Stroke
    - DVT
    - PE

Acquired Myocardium Diseases
(Cont’d)

Myocardial Infarction
Acquired Myocardium Diseases (Cont’d)

- Acute coronary syndromes
  - 785,000 new MIs in U.S. each year
  - 195,000 silent MIs each year
  - American Indians, Alaskan Natives, black
  - 30 patients with stable angina for every infarction
  - Unstable angina occurs more frequently in men
  - 40-60 years, MI risk × 5
  - Average age of first MI is 64.5 yo in men and 70.3 yo in women

Acquired Myocardium Diseases (Cont’d)

- Acute coronary syndromes
  - History
    - Nature of angina symptoms
    - Previous history of CAD
    - Gender
    - Age
    - Number of risk factors present
    - Chest discomfort begins in central/left chest, radiates to arm, wrist, jaw, epigastrium, left shoulder, between shoulder blades

Acquired Myocardium Diseases (Cont’d)

- Acute coronary syndromes
  - History
    - Cardiac-related symptoms
      - Altered mental status
      - Restlessness, anxiety
      - Impending doom
      - Anguished facial expression
      - Nausea/vomiting
      - Profuse sweating
      - Fatigue
    - Palpitations
    - Extremely edema, sacral area
    - Headache
    - Syncope
    - Activity limitations
Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - History
    - Anginal equivalent symptoms
      - Generalized weakness
      - Difficulty breathing
      - Excessive sweating
      - Unexplained nausea and vomiting
      - Dizziness
    - Syncope
      - Palpitations
      - Isolated arm/jaw pain
      - Fatigue
      - Dysrhythmias

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - History
    - Elderly atypical symptoms
      - Mental status changes
      - Generalized weakness
      - Syncope
      - Shortness of breath
      - Fatigue
      - Unexplained nausea
      - Abdominal/epigastric discomfort

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - History
    - Diabetic atypical symptoms
      - Generalized weakness
      - Syncope
      - Lightheadedness
      - Mental status changes
Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - History
    - Women atypical symptoms
    - Aching
    - Tightness
    - Pressure
    - Sharpness
    - Burning
    - Fullness
    - Tingling
    - Back, shoulder, neck discomfort

- Acute coronary syndromes
  - History
    - Frequent acute symptoms
    - Shortness of breath
    - Weakness
    - Unusual fatigue
    - Cold sweats
    - Dizziness
    - Nausea/vomiting

- Acute coronary syndromes
  - Physical findings
    - Restless, anxious, frightened
    - Congested/clear breath sounds
    - Crackles, underlying CAD
    - Skin cool, clammy, pale, ashen
Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Physical findings
    - Rapid heart rate and high BP, ischemia response
    - Measure BP in both arms
    - Pulse deficit, vascular disease, CAD
    - Hypotension, clear lung sounds and JVD, suggests RV1

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - ECG findings
    - Ischemia viewed on ECG as brief changes in ST segments and T waves in leads facing affected area of the ventricle

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Physical findings
    - Cardiovascular causes
      - Aneurysm
      - Aortic dissection
      - Microvascular disease
      - Myocardial ischemia
      - Myocarditis
      - Pericarditis
Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Physical findings
    - Respiratory causes
    - Pleurisy
    - Pneumothorax
    - Pulmonary embolism
    - Respiratory infections

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Physical findings
    - Gastrointestinal causes
      - Cholecystitis
      - Dyspepsia
      - Esophageal spasm
      - Gastroesophageal reflux disease
      - Hiatal hernia
      - Hernia
      - Peptic ulcer disease

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Physical findings
    - Musculoskeletal causes
      - Acromioclavicular disease
      - Chest wall syndrome
      - Chest wall tumors
      - Chest wall trauma
      - Costochondritis
      - Intercostal muscle cramps
Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Therapeutic interventions
    - Minimize infarct size
    - Save ischemic heart muscle
    - Lesson vasoconstriction
    - Reduce myocardial O2 demand

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Therapeutic interventions
    - Prevent, manage complications
    - Improve chances of survival
    - Suspect ACS until ruled out
    - Position of comfort
    - Supplemental O2 if indicated
    - Monitor vital signs continuously
    - Pulse oximeter
    - ECG

Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Therapeutic interventions
    - Possible ACS, 12-lead within 10 minutes of patient contact
    - Repeat ECG with symptom changes
    - May have ACS and normal ECG
    - IV access
    - If no contraindications administer aspirin and nitroglycerin
Acquired Myocardium Diseases (Cont'd)

- Acute coronary syndromes
  - Therapeutic interventions
    - NTG repeated every 5 minutes up to three tablets/sprays
    - Relief of pain primary goal
    - Dysrhythmias common in first few hours of infarction
    - High risk refusal

Acquired Myocardium Diseases (Cont'd)

- Heart failure
  - Heart unable to pump blood, meet metabolic needs of body
  - Acute heart failure
  - Chronic heart failure
  - Right ventricle failure (RVF) result of LVF

Acquired Myocardium Diseases (Cont'd)

- Heart failure
  - Etiology
    - Pericardium, myocardium, endocardium, great vessel disorders
    - Inability of left ventricle to pump blood effectively
    - Any condition that impairs preload, afterload, cardiac contractility, heart rate
    - Starling’s law
Acquired Myocardium Diseases (Cont'd)

- Heart failure
  - Etiology
    - Left ventricular failure and pulmonary edema
      - Blood backs up behind ventricle
      - Blood builds up in lungs
      - Left atrium swells with blood
      - Atrial muscle fibers stretch
      - Pulmonary veins cannot empty into left atrium
      - Pressure within pulmonary muscles increases, forces fluid from pulmonary capillaries across alveolar walls into lung alveoli.

Acquired Myocardium Diseases (Cont'd)

Left-Sided Heart Failure

- Heart failure
  - Etiology
    - Right ventricular failure
      - Must work harder to overcome high pressure and congestion within pulmonary vessels
      - Blood backs up behind right ventricle, increases pressure in right atrium
      - Blood backs up in superior vena cavae, cannot drain into right atrium
Acquired Myocardium Diseases (Cont'd)

- Heart failure
  - Etiology
  - Right ventricular failure
    - Venous return delayed, organs congested with blood
    - Peripheral edema results, generalized body edema/anasarca
    - Cor pulmonale

Acquired Myocardium Diseases (Cont'd)
Right-Sided Heart Failure

Acquired Myocardium Diseases (Cont'd)
Chronic Cor Pulmonale
• Heart failure
  ➢ Etiology
    ➢ Compensatory mechanisms
    ▶ Body attempts to improve cardiac output by manipulating preload, afterload, cardiac contractility, heart rate
    ▶ May worsen heart failure by overdoing other mechanisms

• Heart failure
  ➢ History
    ▶ Shortness of breath
    ▶ Difficulty sleeping
    ▶ PND episodes, suffocation, coughing
    ▶ Tired, weak, “no energy”
    ▶ Recent weight gain

• Heart failure
  ➢ History
    ▶ Progressive lower extremity swelling
    ▶ Difficulty concentrating
    ▶ Nausea, loss of appetite
    ▶ Faintness, palpitations, irregular/rapid pulse
    ▶ Medications
Acquired Myocardium Diseases (Cont'd)

● Heart failure
  ➢ Differential diagnosis
    ▪ Cardiovascular causes
      ➢ Cardiogenic shock
      ➢ Myocardial infarction
      ➢ Myocardial ischemia
      ➢ Cardiogenic pulmonary edema
      ➢ High-altitude pulmonary edema
      ➢ Noncardiogenic pulmonary edema
      ➢ Pulmonary embolism

Acquired Myocardium Diseases (Cont'd)

● Heart failure
  ➢ Differential diagnosis
    ▪ Respiratory causes
      ➢ Acute respiratory distress syndrome
      ➢ Chronic bronchitis
      ➢ Chronic obstructive pulmonary disease
      ➢ Emphysema
      ➢ Pneumonia
      ➢ Pneumothorax
      ➢ Reactive airway disease
      ➢ Respiratory failure

Acquired Myocardium Diseases (Cont'd)

● Heart failure
  ➢ Therapeutic interventions
    ▪ Correct hypoxia
    ▪ Reduce preload
    ▪ Reduce afterload
    ▪ Improve myocardial contractility
    ▪ Position of comfort
    ▪ Sitting position with feet dangling
Acquired Myocardium Diseases (Cont’d)

- Heart failure
  - Therapeutic interventions
    - Supplemental O₂ if indicated
    - Pulse oximeter
    - O₂ saturation >94%
    - Cardiac monitor, 12-lead ECG

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Acquired Myocardium Diseases (Cont’d)

- Heart failure
  - Therapeutic interventions
    - IV access
    - Heparin/saline lock
    - Infuse fluid at to-keep-open rate : 30 mL/hr

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Acquired Myocardium Diseases (Cont’d)

- Heart failure
  - Therapeutic interventions
    - Medications per medical direction
      - Antiarrhythmics treat dysrhythmias
      - Analgesics treat pain
      - Diuretics reduce preload
      - Venodilators reduce preload
      - Positive inotropic agents increase myocardial contractility
      - Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta blockers reduce afterload
Acquired Myocardium Diseases (Cont'd)

- Heart failure
  - Patient and family education
  - Weigh self daily, detect fluid retention
  - Call physician if 2 lb/day, 5 lb/3-4 days
  - Avoid salt
  - Current vaccinations
  - Medication compliance
  - Nonsteroidal antiinflammatory drugs (NSAIDs) avoided
  - Regular exercise

Acquired Myocardium Diseases (Cont'd)

- Myocarditis
  - Inflammation of middle heart/myocardium
  - Constrict heart
  - Benign, self-limiting
  - If widespread in heart muscle, affects heart's ability to pump
  - RVF, LVF, dysrhythmias, death

Acquired Myocardium Diseases (Cont'd)

- Myocarditis
  - Etiology
    - Bacteria, viruses, parasites
    - Most common cause, viral infection
    - Other causes
### Acquired Myocardium Diseases (Cont'd)

**Myocarditis**

- **History**
  - Flu-like symptoms
  - Fatigue
  - Decreased appetite
  - Mild shortness of breath
  - Joint, muscle aches
  - 20% fever
  - Cardiac-related symptoms appear 10-14 days after onset
  - Palpitations

<table>
<thead>
<tr>
<th>Physical findings</th>
</tr>
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<tbody>
<tr>
<td>Tachycardia, tachypnea</td>
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<tr>
<td>ECG, low-voltage QRS complexes, ST-segment elevation</td>
</tr>
<tr>
<td>Atrial/ventricular dysrhythmias</td>
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<tr>
<td>Sinus rhythm</td>
</tr>
<tr>
<td>20%, second-/third-degree AV block</td>
</tr>
<tr>
<td>Left/right BBB</td>
</tr>
<tr>
<td>JVD, crackles, ascites, peripheral edema</td>
</tr>
</tbody>
</table>

- **Physical findings**
  - Newborn/infant signs/symptoms
  - Fever
  - Intrauterine or intra-uterine
  - Periodic episodes of pectoral that precede tachypnea or respiratory distress
  - Diaphoresis
  - Poor feeding; sweating while feeding
  - Mild cyanosis

- Cool, mottled skin from decreased cardiac output
- Rapid, labored respiration; grunting
- Crackles (uncommon)
- Heart failure (tachycardia, gallop rhythm)
Acquired Myocardium Diseases (Cont'd)

- Myocarditis
  - Physical findings
    - Adults signs/symptoms
      - Low-grade fever
      - Fatigue, exhaustion
      - Fatigue
      - Decreased appetite
      - Muscle aches and pain
      - Chest pain
      - Dyspnea with activity
      - Orthopnea and shortness of breath at rest
      - Palpitations (common)
      - Heart failure

Acquired Myocardium Diseases (Cont'd)

- Myocarditis
  - Differential diagnosis
    - Acute coronary syndrome
    - Heart failure
    - Aortic dissection
    - Pneumonia
    - Pulmonary embolism
    - Esophageal perforation, rupture, or tear
    - Kawasaki disease

Acquired Myocardium Diseases (Cont'd)

- Myocarditis
  - Therapeutic interventions
    - Ensure oxygenation and ventilation are effective
    - Positive-pressure ventilation if indicated
    - Pulse oximeter
    - O₂ saturation >94%
    - Supplemental O₂ if indicated
    - IV access
    - Cardiac monitor
Cardiogenic Shock

- Description and definition
  - Heart muscle function severely impaired
  - Decreased cardiac output and inadequate tissue perfusion
  - Common cause, MI

Cardiogenic Shock (Cont’d)

- Etiology
  - Complications of shock
    - Myocardial contractility decreased
    - Prolonged cardiac surgery
    - Ventricular aneurysm
    - Cardiac arrest
    - Ventricular wall rupture

Cardiogenic Shock (Cont’d)

- Etiology
  - Causes
    - Cardiac dysrhythmias
    - Ventricular septum rupture
    - Myocarditis
    - Cardiomyopathy
    - Myocardial trauma
    - Heart failure
    - Hypothermia
    - Severe electrolyte/acid-base imbalances
    - Severe congenital heart disease
Cardiogenic Shock (Cont’d)

• Epidemiology and demographics
  ➢ 7-10% with acute MI
  ➢ 80% have large infarction involving >40% of the left ventricle
  ➢ 10% recent RVI
  ➢ 10% mechanical complications
  ➢ At autopsy, two-thirds have 75% + narrowing of three major coronary arteries

Cardiogenic Shock (Cont’d)

• History
  ➢ Ask the family
  ➢ Cardiomyopathy
  ➢ Congenital heart disease
  ➢ Recent MI
  ➢ Older
  ➢ STEMI
  ➢ Previous MI, heart failure
  ➢ Anterior infarction at time shock develops
  ➢ Dysrhythmia

Cardiogenic Shock (Cont’d)

• Physical findings
  ➢ Mental status is initially normal
  ➢ Breath sounds, crackles in most
  ➢ JVD, absent with hypovolemia
  ➢ Peripheral pulses, weak, rapid
  ➢ Skin, pale and mottled
Cardiogenic Shock (Cont’d)

- Physical findings
  - Extremities, cool and moist
  - ECG, old/new infarctions
  - Systolic BP normal initially, pulse pressure narrowed
  - Cardiac tamponade, heart sounds muffled

Cardiogenic Shock (Cont’d)

- Physical findings
  - Decompensated
    - Altered mental status
    - Unresponsive
    - Breathing rapid and shallow
    - Breath sounds, increased pulmonary congestion, crackles
    - Peripheral pulses are absent
  - Central pulses, weak and rapid
  - Skin pale, mottled, cyanotic
  - Extremities cold and sweaty
  - Ventricular function worsens
  - Cardiac output falls
  - BP decreases

Cardiogenic Shock (Cont’d)

- Differential diagnosis
  - Most common cause, MI
  - Cardiovascular causes
    - Acute coronary syndrome
    - Aortic dissection
    - Myocardial rupture
    - Myocarditis
**Cardiogenic Shock (Cont’d)**

- **Differential diagnosis**
  - Respiratory cause
    - Pulmonary embolism
  - Other causes
    - Hypovolemic shock
    - Sepsis, septic shock

- **Therapeutic interventions**
  - Increase contractility without significant heart rate increases, altering preload/afterload, controlling dysrhythmias
  - ABCs
  - Supplemental O₂ in indicated
  - Pulse oximeter
  - Maintain O₂ saturation >94%

- **Therapeutic interventions**
  - Position of comfort/sitting with feet dangling
  - Limit patient’s physical activity
  - Cardiac monitor, IV access
  - 12-lead ECG
Cardiogenic Shock (Cont’d)

- Therapeutic interventions
  - IV fluids, medication per medical direction
  - Treat dysrhythmias
  - Rapid transport
  - Refusal of care

Cardiopulmonary Arrest

- Absence of cardiac pump function
  - No detectable pulse
  - Unresponsive
  - Apnea/agonal, gasping respirations
Cardiopulmonary Arrest (Cont’d)

- Sudden cardiac death (SCD)
  - Unexpected death from cardiac cause, immediately/within 1 hour of onset of symptoms
  - No warning signs

Cardiopulmonary Arrest (Cont’d)

- Etiology
  - 40%, healed MI
  - 80-90%, narrowing of 1-3 major coronary arteries
  - 50%+, hypertrophic cardiomyopathy and congenital coronary artery anomalies
  - Children, cardiopulmonary arrest from respiratory failure/shock

Cardiopulmonary Arrest (Cont’d)

- Etiology
  - Causes of commotio cordis from blunt trauma to the chest:
    - Baseballs, softballs, lacrosse, cricket, and soccer balls
    - Tennis balls filled with coins for training pitchers
    - Hockey pucks, sticks
    - Play shadow boxing
    - Parent-to-child discipline
    - Gang rituals
    - Scuffle
    - Plastic sledging saucers
Cardiopulmonary Arrest (Cont’d)

• Etiology
  ➢ Onset of terminal event
    • 1 hour-less between sudden changes in cardiovascular status and cardiac arrest
    • Circulatory failure deaths, pulse stops after peripheral circulation is collapsed
  ➢ Most deaths are from dysrhythmias

Cardiopulmonary Arrest (Cont’d)

• Epidemiology and demographics
  ➢ 50% of coronary heart disease deaths are sudden/unexpected
  ➢ In 50% of men and 64% of women, sudden deaths have no previous symptoms
  ➢ Previous heart attack, sudden death rate is four to six times the general population
  ➢ SCD peak incidence, birth to 6 months and 45-75 years
  ➢ SCD, 19% in ages 1-13 years, 30% in ages 14-21 years

Cardiopulmonary Arrest (Cont’d)

• History
  ➢ SCD risk
    • Chest discomfort
    • Dyspnea
    • Weakness/fatigue
    • Palpitations
    • Syncope
    • Nonspecific complaints before MI/SCD
Cardiopulmonary Arrest (Cont’d)

- Cardiac arrest questions
  - Did anyone see the patient collapse?
  - Can you describe what happened?
  - What time did the patient collapse?
  - Did anyone begin CPR?
  - How long before 9-1-1 was called?
  - Was an automated defibrillator used?
  - DNR order?

Cardiopulmonary Arrest (Cont’d)

- Physical findings
  - Cardiopulmonary arrest
    - Central pulses absent
    - Work of breathing absent
    - Unresponsive

Cardiopulmonary Arrest (Cont’d)

- Differential diagnosis
  - Cardiovascular causes
    - Acute coronary syndrome
    - Cardiac tamponade
    - Cardiomyopathy
    - Congenital heart disease
    - Hypovolemia
    - MI
    - Wolff-Parkinson-White syndrome
Cardiopulmonary Arrest (Cont’d)

- Differential diagnosis
  - Respiratory causes
    - Hypoxia
    - Pulmonary embolism
    - Tension pneumothorax

- Other causes
  - Preexisting acidosis
  - Drug overdose
  - Hypoglycemia
  - Hyperkalemia
  - Hypokalemia
  - Hyperthermia
  - Hypothermia

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - En route, determine which team member performs the critical tasks of resuscitation
    - Airway management
    - Chest compressions
    - Monitoring and defibrillation
    - Vascular access and medication administration
Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - En route, determine which team member performs critical tasks of resuscitation
    - Team leader
    - Know resuscitation guidelines
    - Local protocols
    - Patient condition
    - Open to suggestions
    - Team members relay vital signs every 3-5 minutes, ABC changes, completed tasks

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Begin tasks where patient was found unless hazardous
  - ABC assessment
  - Obvious death signs, do not begin CPR
  - Check for DNAR order
  - Cardiac monitor

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Pulseless ventricular tachycardia or ventricular fibrillation
    - Perform high quality CPR
    - Observed collapse, monitor shows VT/VF
    - Defibrillate using manufacturer recommended energy levels
    - When applying cardiac monitor/preparing defibrillator delayed, perform CPR
Therapeutic interventions

- Pulseless ventricular tachycardia or ventricular fibrillation
  - Ventilate with bag-valve-mask, 100% O2, one breath/5-6 seconds
  - Endotracheal CO₂ monitor
  - IV access with normal saline, Ringer's lactate

- Medications per resuscitation guidelines
- Advanced airway if there is time
- Look for and treat reversible causes of arrest
- Rhythm changes, assess pulse

- Asystole
  - No ventricular activity on the cardiac monitor
  - Check leads, cable connections, power on, gain turned up, same rhythm when second lead is checked
Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Pulseless electrical activity (PEA) and asystole
    - PEA
    - Organized electrical activity on ECG
    - Patient is unresponsive, not breathing, no central pulse
  - Poor prognosis
  - Hyperkalemia in renal failure, methanol ingestion, aspirin overdose, TCA overdose
  - Tension pneumothorax
  - Cardiac tamponade

- Hypovolemia
- Hypoxia
- Blood glucose level <60 mg/dL
- Hypothermia
Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Pulseless electrical activity and asystole
    - Hyperkalemia with renal failure
    - Tricyclic antidepressant overdose
    - Narcotic overdose
    - Beta-blocker overdose

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Postresuscitation care
    - Goals
      - Providing cardiorespiratory support to optimize tissue perfusion—especially to the heart, brain, and lungs
      - Transporting the patient to an appropriate facility capable of providing comprehensive post-cardiac arrest care including acute coronary interventions, neurological care, goal-directed critical care, and hypothermia
      - Attempting to identify the precipitating cause of the arrest, start specific treatment if necessary, and take actions to prevent recurrence

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Postresuscitation care
    - Reassess ABCDs
    - Airway
      - Effectiveness of initial airway maneuvers, interventions
      - Advanced airway if needed
Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Postresuscitation care
    - Breathing
      - Assess ventilation adequacy
      - Confirm advanced airway placement
      - Positive-pressure ventilation with O2
      - Effectiveness of ventilations with capnography
      - Pulse oximeter, assess O2 saturation
      - Rule out breathing complications from resuscitation
      - Positive-pressure ventilation for absent/inadequate spontaneous respirations

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Postresuscitation care
    - Circulation
      - Reassess vital signs, skin color
      - IV access with normal saline, lactated Ringer's
      - ECG monitoring, 12-lead ECG
      - Differential diagnosis

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Evaluate IV infusions during resuscitation, currently running/still needed
  - Ensure the family is updated
  - Documentation
  - Acknowledge the efforts of the resuscitation team
  - Post-resuscitation critique
Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Postresuscitation care
    - Temperature regulation
    - Monitor closely
    - Spontaneous circulation return
    - Therapeutic hypothermia

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Glucose control
    - Hypoglycemia signs not obvious in comatose patient

Cardiopulmonary Arrest (Cont’d)

- Therapeutic interventions
  - Field termination of resuscitation
    - NAEMSP recommendations
    - Adult with SCD likely medical, not associated with condition potentially responsive to hospital treatment
Therapeutic interventions
- Field termination of resuscitation
  - NAEMSP recommendations
    - Unwitnessed cardiac arrest with delayed CPR beyond 8 minutes, delayed defibrillation beyond 11 minutes
    - Without advance directives, full resuscitative effort and 20- to 30-minute treatment following advanced cardiac life support guidelines before declaring patient dead
  - Rhythm changes/remains in VF/VT, continued resuscitation
  - Asystole/PEA considered terminal rhythms, resuscitation termination considered
  - Logistics considered
- Consult medical direction before termination
  - EMS professionals, family access to clergy, counselors, social workers
  - Continuous documentation
Hypertension

- Normal BP
  - Systolic <120 mm Hg
  - Diastolic <80 mm Hg
- Prehypertension
  - Systolic, 120-139 mm Hg
  - Diastolic, 80-89 mm Hg

Hypertension (Cont'd)

- Stage 1 high BP
  - Systolic, 140-159 mm Hg
  - Diastolic, 90-99 mm Hg
- Stage 2 high BP
  - Systolic ≥160 mm Hg
  - Diastolic ≥100 mm Hg

Hypertension (Cont'd)

- Hypertension
  - Measured 2+ occasions
  - Not taking medicine for high BP
  - Do not have short-term serious illness
  - Do not have other conditions, diabetes, kidney disease
Hypertension (Cont’d)

- **Etiology**
  - Silent killer, no signs/symptoms
  - Damages heart, brain, eyes, blood vessels, kidneys
  - Uncontrolled
    - Vision problems
    - Increases risk of stroke, heart attack, heart failure, kidney failure

Hypertension (Cont’d)

Hypertensive Heart

Hypertension (Cont’d)

- **Etiology**
  - 90-95% essential/primary hypertension
    - No identifable cause
    - High BP factors
Hypertension (Cont’d)

• Etiology
  ➢ Identifiable cause
    ➢ Medications
      ➢ Amphetamines
      ➢ Anabolic steroids
      ➢ Antidepressants such as desipramine (Norpramin), phenelzine (Nardil), bupropion (Wellbutrin, Zyban), venlafaxine (Effexor)
      ➢ Appetite suppressants

Hypertension (Cont’d)

• Etiology
  ➢ Identifiable cause
    ➢ Medications
      ➢ Cocaine
      ➢ Cold medicines, nasal decongestants
      ➢ Ephedra (ma-huang)
      ➢ Herbal supplements such as ginkgo, ginseng, licorice, St. John’s wort
      ➢ Immunosuppressants such as corticosteroids (Medrol), cyclosporine (Neoral, Sandimmune), tacrolimus (Prograf, Protopic)

Hypertension (Cont’d)

• Etiology
  ➢ Identifiable cause
    ➢ Medications
      ➢ NSAIDs such as ibuprofen (Advil, Motrin), meloxicam (Mobic), naproxen (Naprosyn), naproxen sodium (Aleve)
      ➢ Oral contraceptives
      ➢ Phencyclidine (PCP, “angel dust”)
Hypertension (Cont’d)

- Etiology
  - Identifiable cause
    - Disease/condition
      - Abnormal blood vessels
      - Adrenal disease
      - Coarctation of the aorta
      - Cushing’s syndrome
      - Hyperaldosteronism
      - Hyperparathyroidism

  - Kidney disease
  - Lead poisoning
  - Pheochromocytoma
  - Preeclampsia
  - Quadriplegia
  - Sleep apnea

- Factors that affect BP
  - Increased peripheral resistance
    - Atherosclerosis
    - Pheochromocytoma
    - Cushing’s syndrome
    - Stress
    - Hyperthyroidism
    - Diabetes

  - Cold medicines and nasal decongestants
  - Anorexiants, anabolic steroids, cocaine, pseudoephedrine, phencyclidine
  - Tobacco use

- Increased cardiac output
  - Excessive sodium intake
  - Pheochromocytoma
  - Preeclampsia
  - Stress
  - Hyperthyroidism
  - Kidney disease
  - Hyperaldosteronism
Hypertension (Cont'd)

- Etiology
  - Hypertensive emergencies
    - Hypertensive urgencies
      - Significant BP elevations with nonspecific symptoms, corrected within 24 hours

- Etiology
  - Hypertensive emergencies
    - Require rapid (within 1 hour) BP lowering to prevent/limit organ damage
    - Must have evidence of end-organ failure secondary to hypertension
    - Malignant hypertension

- Etiology
  - Hypertensive emergencies
    - Severe hypertension
    - Headache
    - Vomiting
    - Visual disturbances
    - Mental status changes
    - Seizures
    - Abnormalities of retina of the eye
    - Untreated, death in a few hours
Hypertension (Cont'd)

- Epidemiology and demographics
  - Hypertensive emergencies occur with history of hypertension
  - Medication noncompliance
  - Toxemia from pregnancy

- History
  - Develops rapidly
    - Presentation
      - Severe headache
      - Blurred vision
      - Dizziness
      - Tinnitus
      - Dyspnea
      - Chest pain/lightness
      - Nosebleed
      - Muscle cramps
      - Weakness
      - Palpitations
      - Symptoms of PND, orthopnea

Hypertension (Cont'd)

- History

Hypertension (Cont'd)

- Physical findings
  - Looks sick
  - Possible mental status changes
  - Skin
    - Pales, flushed
    - Normal, feels dry and moist
    - Warm/cold
Hypertension (Cont'd)

- Physical findings
  - Peripheral pulse strong/bounding
  - Check BP in both arms for dissected aorta
  - Diastolic pressure >130 mm Hg
  - Seizures
  - Signs of heart failure
  - Ischemic changes seen on ECG

Hypertension (Cont'd)

- Differential diagnosis
  - Cardiovascular causes
    - Aortic dissection
  - Neurological causes
    - Epileptiform state
    - Head injury
    - Intracranial mass
    - Stroke
    - Subarachnoid hemorrhage

Hypertension (Cont'd)

- Differential diagnosis
  - Genitourinary causes
    - Pheochromocytoma
    - Renal failure
    - Toxemia of pregnancy
Hypertension (Cont'd)

- Differential diagnosis
  - Other causes
    - Acute anxiety
    - Cocaine/amphetamine use
    - Connective tissue disease
    - Drug overdose/withdrawal

Hypertension (Cont'd)

- Therapeutic interventions
  - Lowering BP with IV medications, usually in the hospital
  - Supportive care
  - Do not delay transport
  - Vasodilators, monitor BP closely
  - Treat seizures
  - Rapid transport
  - Refusal of care

Hypertension (Cont'd)

- Patient and family education
  - Control BP
  - Taught to take and record
  - Quit smoking
  - Overweight, lose weight, diet low in salt
  - Vitamins, minerals
  - Medication compliance importance
Endocarditis

- Infection of innermost surface of the heart
  - Chambers
  - 1+ heart valves
  - Septum

Endocarditis (Cont’d)

Infective Endocarditis

Endocarditis (Cont’d)

- Etiology
  - Bacteria lodge in bloodstream, multiply on heart valve, other damaged tissue in heart
  - Untreated, damages heart valve, malfunction
  - Most often with preexisting valvular disease
  - Congenital heart disease
Endocarditis (Cont’d)

- Etiology
  - Right-sided endocarditis
    - Affects tricuspid and pulmonary valves
    - IV drug users
    - Infected venous catheters
    - Dialysis shunts
    - Transvenous pacing wires

- Etiology
  - Organisms
    - Originate from skin, upper airway, genitourinary or gastrointestinal tract
    - Minor skin infection
    - Dental cleaning
    - Upper respiratory infection
    - Major surgery
    - Body piercing, tattooing

Endocarditis (Cont’d)

- Epidemiology and demographics
  - Predisposing factors
    - Artificial heart valve
    - Previous history of endocarditis
    - Heart valves damaged by rheumatic fever
    - Congenital heart valve defects
    - Hypertrophic cardiomyopathy
    - Intravascular catheter
Endocarditis (Cont'd)

- History
  - Fever, chills
  - Headache
  - Appetite loss
  - Weight loss
  - Muscle and joint aches/pains
  - Night sweats

Endocarditis (Cont'd)

- History
  - Shortness of breath
  - Cough
  - Recent dental work in previous 1-2 months
  - IV drug use
  - Recent valvular surgery/disease
  - Rheumatic fever history
  - History of endocarditis

Endocarditis (Cont'd)

- Physical findings
  - 30-40%, CNS involvement
  - Transient ischemic attack
  - Subarachnoid hemorrhage
  - Heart failure signs
Endocarditis (Cont’d)

- Physical findings
  - ECG changes
    - Prolonged PR interval
    - Complete heart block
    - Left BBB
    - Flat, painless, red-to-blue lesions on palms, soles
    - Small, tender nodules on finger/toe pads

Endocarditis (Cont’d)

- Differential diagnosis
  - Neurological causes
    - Encephalitis
    - Meningitis
  - Respiratory causes
    - Lung abscess
    - Pneumonia
    - Pulmonary emboli

Endocarditis (Cont’d)

- Therapeutic interventions
  - Supportive
    - Position of comfort
    - O₂
    - Establish IV
    - Cardiac monitor
    - Pulse oximeter, O₂ saturation >94%
    - Heart failure care per local protocols
Endocarditis (Cont’d)

- Therapeutic interventions
  - Refusal of care
    - Persuade patient to accept care
  - Medical direction for advice
  - Document

Endocarditis (Cont’d)

- Patient and family education
  - Carry wallet card to alert health professionals
  - High risk, prophylactic antibiotics before undergoing dental, surgical, and other invasive procedures

Diseases of the Pericardium

- Pericarditis
  - Inflammation of double-walled sac enclosing the heart
  - Pericardium helps anchor the heart in place
    - Prevents excessive movement of heart in chest with body movement
    - Protects from trauma, infection
Diseases of the Pericardium (Cont'd)

Pericarditis

- **Etiology**
  - Viral, bacterial, fungal
  - May develop days/weeks after heart attack
  - After blunt, penetrating chest trauma, open-heart surgery, coronary angioplasty, implantation of defibrillator/pacemaker
  - Procainamide, hydralazine, radiation therapy

- Kidney disease
- Inflammatory disorders
- Breast/lung cancer
- Lymphomas
- Leukemia
- No cause
Diseases of the Pericardium
(Cont'd)

- Pericarditis
  - History
    - Recent flulike symptoms
    - Recent upper respiratory infection
    - Recent fever with shaking chills, shortness of breath, coughing, skin rash, weight loss
    - Recent MI, leukemia, Hodgkin's disease, lymphoma, lupus, kidney disease, chest trauma, heart surgery

Diseases of the Pericardium
(Cont'd)

- Pericarditis
  - Physical findings
    - Chest discomfort
    - Sharp, stabbing pain
    - Constricting pain radiates to shoulder and arm(s)
    - Pain is worse with deep inspiration, coughing, lying flat

Diseases of the Pericardium
(Cont'd)

- Pericarditis
  - Physical findings
    - Heart sounds
      - Pericardial friction rub, not always present
      - Crephey, grating sounds
      - Lean forward, listen to third and fifth intercostal space to left of the sternum
      - Patient hold breath while listening
Diseases of the Pericardium  
(Cont'd)

- Pericarditis
  - Physical findings
    - Fever
    - Tachycardia
    - Tachypnea
    - Pale
    - JVD indicates pericardial effusion
    - Breath sounds normal
    - ECG: ST-segment elevation in multiple leads, PR-segment depression

Diseases of the Pericardium  
(Cont'd)

- Pericarditis

  ECG

Diseases of the Pericardium  
(Cont'd)

- Pericarditis
  - Differential diagnosis
    - Cardiovascular causes
      - Aortic dissection
      - Cardiomyopathy
      - MI
Diseases of the Pericardium
(Cont'd)

- Pericarditis
  - Differential diagnosis
    - Respiratory causes
      - Pleurisy
      - Pneumothorax
      - Pulmonary embolism
    - Other causes
      - Costochondritis
      - Gastroesophageal reflux disease
      - Lupus

Diseases of the Pericardium
(Cont'd)

- Pericarditis
  - Therapeutic interventions
    - Supportive
      - Position of comfort
      - O₂
      - IV access
    - Cardiac monitor
    - Pulse oximeter, saturation >94%

Diseases of the Pericardium
(Cont'd)

- Pericarditis
  - Therapeutic interventions
    - Nonsteroidal antiinflammatory drugs
    - Viral usually resolves on its own
    - Bacterial, antibiotics
    - Fungal, antifungal medications
    - Transport without lights, sirens
    - Refusal of care
Diseases of the Pericardium (Cont'd)

- Pericardial tamponade
  - Pericardial effusion
    - Increased volume of pericardial fluid surrounding the heart
    - Pressure within the pericardium increases
    - Impairs the heart's ability to fill; depends on rate buildup or ability of the pericardium to stretch, make room for increased volume

Diseases of the Pericardium (Cont'd)

Effects of Pericardial Effusion and Cardiac Tamponade

- Etiology
  - Cardiac tamponade may be caused by same conditions as pericarditis
  - Develops gradually if caused by infection, tumor
Diseases of the Pericardium (Cont'd)

- Pericardial tamponade
  - Causes
    - Pericarditis, pericardial effusion
    - Cardiac rupture after MI
    - Blunt trauma
    - Penetrating trauma
    - Renal disease
    - Hypothyroidism
    - Aortic dissection
    - Radiation induced
    - Heart surgery
    - Hospital procedures

- Pericardial tamponade
  - History
    - Anxious, restless
    - Shortness of breath
    - Chest tightness
    - Dizziness
    - Recent invasive procedure
    - Heart attack
    - Chronic illness
    - Medications

- Pericardial tamponade
  - Physical findings
    - From trauma, chest wall injury
    - JVD
    - Hypotension
    - Muffled heart sounds
Diseases of the Pericardium (Cont'd)

- Pericardial tamponade
  - Physical findings
    - Cold, pale, mottled, cyanotic skin
    - Tachycardia
    - Weak, absent peripheral pulses
    - Narrowing pulse pressure
    - Pulsus paradoxus

- If it develops slowly, signs/symptoms
  - Heart failure
  - Dyspnea
  - Orthopnea
  - JVD

- ECG changes
  - Low-voltage QRS and T waves
  - ST-segment elevation
  - Nonspecific T-wave changes
  - ECG voltage of P wave, QRS complex, T wave
Diseases of the Pericardium (Cont'd)

- Pericardial tamponade
  - Differential diagnosis
    - Cardiovascular causes
      - RVF
      - RVI
    - Respiratory causes
      - Pulmonary embolism
      - Tension pneumothorax

- Therapeutic interventions
  - Trauma, stabilize cervical spine
  - Open airway
  - O₂ saturation >94%
  - Ventilation, oxygenation effective

- Suction
- Control bleeding
- Cardiac monitor
- Maintain body temperature
Diseases of the Pericardium (Cont'd)

- Pericardial tamponade
  - Therapeutic interventions
    - En route
      - IV access
      - IV fluids, medications per medical direction
      - Assess mental status, heart rate, respiratory effort, breath sounds, BP

- Definitive treatment, at hospital
  - Pericardiocentesis
  - Refusal of care
    - Persuade patient to accept care
    - Risk of death
    - Medical direction for advice
    - Document

Cardiovascular Congenital Abnormalities

- Acyanotic heart defects
  - Oxygenated blood shunted from left to right side of heart
  - Increased pulmonary blood flow
    - Atrial septal defect
    - Ventricular septal defect
    - Patent ductus arteriosus
Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Obstruction to blood flow from ventricles
    - Coartation of the aorta
    - Aortic stenosis
    - Pulmonary stenosis

- Atrial septal defect (ASD)
  - Abnormal opening in the wall separating the atrial chambers of heart
  - Some oxygenated blood from left atrium flows through the hole to the right atrium
  - Defect not repaired, pulmonary hypertension

- More in women
- 5-10% of all congenital heart defects
- Murmur
- Risk of atrial dysrhythmias
- Small ASDs (< 8 mm) often close by 18 months
Cardiovascular Congenital Abnormalities (Cont’d)

Atrial Septal Defect

Acyanotic heart defects

Ventricular septal defect (VSD)
- Abnormal opening in wall separating right and left ventricles
- Allows some oxygenated blood to flow from the left ventricle through the hole to the right ventricle and pulmonary artery instead of pumped into aorta
- Stroke volume reduced, affects cardiac output
- Risk for bacterial endocarditis
- Antibiotics before certain dental and surgical procedures

Right ventricle may enlarge

Most common defect, 15-20%

Small, asymptomatic

Moderate to large size

Risk for bacterial endocarditis

Antibiotics before certain dental and surgical procedures
Cardiovascular Congenital Abnormalities (Cont'd)

Ventricular Septal Defect

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Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Patent ductus arteriosus
    - Ductus arteriosus fails to close after birth
    - Failure to close within 10 days, abnormal
    - Common in premature infants
    - Maternal rubella infection

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Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Patent ductus arteriosus
    - High altitude
    - Oxygenated blood traveling through the aorta is shunted from the aorta, across the duct, to the pulmonary artery, mixes with deoxygenated blood
    - Left atrium, ventricle workload increased
    - Less blood is delivered to lungs for oxygenation
Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Patent ductus arteriosus
    - Larger shunt signs/symptoms
    - Wide pulse pressure
    - Bounding peripheral pulses
    - Characteristic murmur
    - Rapid breathing, increased work of breathing
    - Frequent respiratory infections
    - Fatigue/poor growth

Cardiovascular Congenital Abnormalities (Cont'd)

- Patent Ductus Arteriosus

Cardiovascular Congenital Abnormalities (Cont'd)

- Coarctation of the aorta (COA)
  - Aorta pinched/constricted in area of ductus arteriosus, just beyond aorta's branching vessels to head/arms
  - Left ventricle works harder to force blood through narrowed area to lower body
  - Increased BP proximal to defect
Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Coarctation of the aorta (COA)
    - Decreased blood flow distally
    - Severe narrowing, left ventricle not strong enough to perform extra work, heart failure, poor perfusion
    - 8-10% all congenital heart defects
    - Occurs in males twice as frequently as in females

Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Coarctation of the aorta (COA)
    - Physical findings
      - Dyspnea, poor feeding, poor weight gain, signs of shock
      - High BP, bounding pulses in arms
      - Lower BP with weak/absent femoral pulses
      - Cool lower extremities

Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Coarctation of the aorta (COA)
    - Differential cyanosis
    - Signs of heart failure
    - Dizziness, frequent headaches, fainting, nosebleeds
Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Coarctation of the aorta (COA)
    - Risks
      - Ruptured aorta
      - Hypertension
      - Aortic aneurysm
      - Stroke

Cardiovascular Congenital Abnormalities (Cont'd)

Coarctation of the Aorta

Cardiovascular Congenital Abnormalities (Cont'd)

- Acyanotic heart defects
  - Aortic stenosis
    - Aortic valve narrowed
    - Most common, at valve itself
    - Valve may have only one cusp or two cusps
    - Does not open freely, causing left ventricle to work harder to eject blood into the aorta
    - 3-6% of all congenital heart defects
Cardiovascular Congenital Abnormalities (Cont’d)

- Acyanotic heart defects
  - Aortic stenosis
    - Occurs in males four times as frequently as in females
    - Chest pain, dizziness, fainting, unusual tiring
    - Murmur
    - BP usually normal
    - Exercise restriction

Cardiovascular Congenital Abnormalities (Cont’d)

- Pulmonary stenosis
  - Pulmonic valve narrowed
  - Obstruction usually at the valve itself
  - Valve cusps thickened, hinders blood flow from right ventricle
  - Right ventricle increased workload
  - 8-12% all congenital heart defects
Cardiovascular Congenital Abnormalities (Cont’d)

- Acyanotic heart defects
  - Pulmonary stenosis
    - Shortness of breath
    - Chest pain
    - Epigastric pain with exertion
    - Murmur
    - Cyanosis
    - Sudden death possible

Cardiovascular Congenital Abnormalities (Cont’d)

Pulmonary Stenosis

Cardiovascular Congenital Abnormalities (Cont’d)

- Cyanotic heart defects
  - Deoxygenated blood from right side of heart mixes with oxygenated blood from left, enters systemic circulation, bypasses pulmonary circulation
  - Right-to-left shunt
  - Decreased pulmonary blood flow
    - Tetralogy of Fallot
    - Tricuspid atresia
Cyanotic heart defects
- Decreased pulmonary blood flow
- Tetralogy of Fallot
- Tricuspid atresia

- Mixed blood flow
  - Transposition of great vessels
  - Total anomalous pulmonary venous return or communication
  - Truncus arteriosus
  - Hypoplastic heart syndrome

- Tetralogy of Fallot
  - Large ventricular septal defect
  - Narrowing at, below pulmonary valve
  - Right ventricle more muscular than normal
  - Aorta lies directly over ventricular septal defect
Cardiovascular Congenital Abnormalities (Cont’d)

- Cyanotic heart defects
  - Tetralogy of Fallot
  - After ductus closes, cyanosis develops
  - Clubbing occurs
  - Characteristic murmur present
  - May experience tetralogy spell
    - Keep calm
    - Hold in knee-chest position
    - Treat as directed

Tetralogy of Fallot

Cardiovascular Congenital Abnormalities (Cont’d)

- Cyanotic heart defects
  - Transposition of great vessels
    - Positions of pulmonary artery and aorta reversed
    - Aorta connected to right ventricle
    - Pulmonary artery connected to left ventricle
Cardiovascular Congenital Abnormalities (Cont’d)

- Cyanotic heart defects
  - Transposition of great vessels
    - Survival only if 1+ defects allows oxygenated blood to reach systemic circulation
    - Cyanosis
    - Possible murmur
    - Death rate, 90% without surgery <6 months

Circulation Disorders

- Aortic aneurysm and dissecting aneurysm
  - Layers
    - Adventitia, thin outer layer
    - Media, thick, elastic middle layer
    - Intima, thin innermost layer
Circulation Disorders (Cont’d)

- Aortic aneurysm and dissecting aneurysm
  - Aneurysm
    - Constant wall stress weakens, swells
    - Ruptures if stretches too far
  - Aortic dissection
    - Tear in inner aortic lining
    - Blood flows through the tear
    - Blood between layers causes separation
    - Most begin in ascending aorta
    - 65-75% untreated, die within 2 weeks

- Etiology
  - Coronary heart disease
  - High blood cholesterol
  - Hypertension
  - Atherosclerosis, most common cause
  - Cystic medial degeneration
  - Marfan syndrome

Marfan Syndrome
Aortic aneurysm and dissecting aneurysm

- Risk factors
  - Age
  - Gender
  - Family history
  - Smoking
  - Atherosclerosis
  - Hypertension
  - Hyperlipidemia
  - Pregnancy
  - Crack cocaine use
  - Trauma
  - Inflammatory disease
  - Connective tissue disorders

Epidemiology and demographics

- 13% with aortic aneurysm have multiple aneurysms
- 25-28% with thoracic aortic aneurysm have abdominal aneurysm
- 75% occur in abdominal aorta
- Abdominal aortic aneurysms 5-10× as frequent in men
- Frequency increases >55 years men, >70 years women

History

- Symptoms not always present, depend on location of aneurysm
- Dissection, pain begins abruptly, constant, unbearable
- Tearing, ripping, sharp, stabbing, knife-like
- Pain
Circulation Disorders (Cont’d)

• Aortic aneurysm and dissecting aneurysm
  ➢ History
    • 10% have no pain
    • Anxiety, impending doom
    • Extremity weakness, numbness, tingling
    • Myocardial ischemia
    • Less common symptoms

Circulation Disorders (Cont’d)

• Aortic aneurysm and dissecting aneurysm
  ➢ Physical findings
    • Dissecting thoracic aneurysm, BP variations in upper extremities
    • Dissecting abdominal aneurysm, BP variations in lower extremities
    • Peripheral pulses hard to feel
    • Skin pale, diaphoretic

Circulation Disorders (Cont’d)

• Aortic aneurysm and dissecting aneurysm
  ➢ Physical findings
    • Tracheal, bronchial compression
    • Hemothorax signs if dissection ruptures in the pleural cavity
    • Sudden back pain with abdominal aneurysm rupture
    • Possible hypotension
    • Caution when palpating
    • Abdominal distention
Circulation Disorders (Cont’d)

Abdominal Aortic Aneurysm

Circulation Disorders (Cont’d)

- Aortic aneurysm and dissecting aneurysm
  - Differential diagnosis
    - Cardiovascular causes
      - Cardiac tamponade
      - Cardiogenic, hypovolemic, hemorrhagic shock
      - Cardiomyopathy
      - Hypertensive emergencies
      - Myocardial infarction
      - Myocarditis
      - Pericarditis

Circulation Disorders (Cont’d)

- Aortic aneurysm and dissecting aneurysm
  - Differential diagnosis
    - Respiratory causes
      - Pleural effusion
      - Pulmonary embolism
    - Musculoskeletal causes
      - Back pain
      - Costochondritis
Circulation Disorders (Cont'd)

- Aortic aneurysm and dissecting aneurysm
  - Differential diagnosis
    - Gastrointestinal causes
      - Diverticulitis
    - Gastrointestinal bleeding
    - Other causes
      - Hernia
      - Kidney stone

- Therapeutic interventions
  - Medical emergency
  - Supplemental O₂
  - Effective ventilation, oxygenation
  - Pulse oximeter, >94% saturation
  - Cardiac monitor
  - Maintain normal body temperature
  - Do not delay transport for procedures

- IV access, two large-bore catheters
- Fluids, medications per medical direction
- Contact medical direction with dissection suspicion
- En route, reassess every 5 minutes
- Rapid transport
Circulation Disorders (Cont'd)

• Acute arterial occlusion and acute limb ischemia
  ➢ Sudden disruption of arterial blood flow
    • Thrombus
    • Embolus
    • Tumor
    • Direct trauma to artery

Circulation Disorders (Cont'd)

• Acute arterial occlusion and acute limb ischemia
  ➢ Most from embolus
  ➢ Artery blockage from atherosclerosis
  ➢ Intermittent claudication

Circulation Disorders (Cont'd)

• Acute arterial occlusion and acute limb ischemia
  ➢ History
    • Sudden symptoms, embolus likely for ischemia
    • Gradual symptoms, thrombus
    • Five Ps
      • Motor impairment, sensory loss
      • Recent extremity injury
  ➢ IV drug use
  ➢ Heart surgery/attack
  ➢ Clotting disorder
  ➢ Pulmonary embolism
  ➢ AFB
  ➢ Rheumatic heart disease
Circulation Disorders (Cont’d)

● Acute arterial occlusion and acute limb ischemia
  ➢ Physical findings
    • Arterial pulses
    • Skin color pale, mottled
    • Arterially blood flow restricted, foot chalk white when raised
    • Assess all extremities
    • BP in both arms, unequal suggests thoracic aneurysm
    • Bruit

Circulation Disorders (Cont’d)

● Acute arterial occlusion and acute limb ischemia
  ➢ Differential diagnosis
    • Abdominal aneurysm
    • Arthritis
    • Deep vein thrombosis
    • Scleroderma
    • Soft tissue injury
    • Systemic lupus erythematosus

Circulation Disorders (Cont’d)

● Acute arterial occlusion and acute limb ischemia
  ➢ Therapeutic interventions
    • Position of comfort, sitting
    • O₂, IV access, cardiac monitor
    • Pulse oximeter, saturation >94%
    • Medications per medical direction
    • Ambulance compartment warm
    • Do not apply heat/cold to affected limb
    • Rapid transport
    • Refusal of care
Circulation Disorders (Cont’d)

- Acute deep vein thrombosis
  - Thrombophlebitis
  - Superficial thrombophlebitis
  - Deep vein thrombosis

Circulation Disorders (Cont’d)

- Acute deep vein thrombosis
  - Risk factors
    - Increasing age
    - Recent direct trauma to vein
    - Recent prolonged inactivity
    - Recent prolonged bed rest
    - Certain cancers
    - Obesity
    - Oral contraceptives, hormone replacement therapy

Circulation Disorders (Cont’d)

- Acute deep vein thrombosis
  - Risk factors
    - Central venous catheterization
    - Pregnancy, postpartum period
    - Stroke with paralysis
    - Clotting disorder family history
    - DVT, pulmonary embolism history
    - Heart failure
Circulation Disorders (Cont’d)

- Acute deep vein thrombosis
  - Predispositions
    - Venous stasis, sluggish blood flow
    - Vessel inner lining damage
    - Blood clotting disorders

Circulation Disorders (Cont’d)

Venous Stasis Ulcer

Circulation Disorders (Cont’d)

- Acute deep vein thrombosis
  - History
    - Swelling, pain, tenderness in limb
Circulation Disorders (Cont’d)

• Acute deep vein thrombosis
  ➢ Physical findings
    • Assess extremities carefully
    ➢ Compare
    ➢ Inflammation
    ➢ Homan’s sign
    ➢ Do not rub affected limb, dislodge clot

Circulation Disorders (Cont’d)

• Acute deep vein thrombosis
  ➢ Differential diagnosis
    • Arthritis
    • Cellulitis
    • Muscle/soft tissue injury
    • Pulmonary embolism
    • Superficial thrombophlebitis

Circulation Disorders (Cont’d)

• Acute deep vein thrombosis
  ➢ Therapeutic interventions
    • Supportive
      ➢ Position of comfort
      ➢ O₂, IV access, cardiac monitor
      ➢ Pulse oximeter, saturation >94%
      ➢ Monitor for pulmonary embolism
      ➢ Refusal of care
Circulation Disorders (Cont’d)

- Acute deep vein thrombosis
  - Patient and family education
    - Avoid dehydration
    - Avoid constrictive clothing
    - Walking
    - Compression stockings
    - Medical ID bracelet for blood thinners

Chapter Summary

- Cardiovascular disorders are diseases; conditions involve the heart, blood vessels
  - Coronary heart disease refers to disease of the coronary arteries; resulting complications, such as angina pectoris/acute MI, affect arteries that supply the heart muscle with blood

Chapter Summary (Cont’d)

- Risk factors are traits; lifestyle habits may increase a person’s chance of developing disease
  - Cannot be modified are nonmodifiable/fixed risk factors
  - Contributing risk factors lead to increased risk of heart disease, exact role is not defined
Arteries are conductance vessels; they carry blood from the heart under high pressure. Smallest branches of arteries, resistance vessels, smooth muscle in walls, allows vessel to adjust its diameter, controlling amount of blood flow to specific tissues.

Capillaries are smallest blood vessels; they connect arterioles and venules, function as exchange vessels. Venules connect capillaries, veins, veins carry O₂-poor blood from body to right side of heart. Because most of the body's blood is located in them at any one time, veins are capacity (storage) vessels.

Heart has four chambers and two functional pumps. Right atrium, right ventricle. Left atrium, left ventricle. Right side of heart is low-pressure system. Left side of heart is high-pressure pump. Walls of heart are composed of endocardium, myocardium, epicardium.
Chapter Summary (Cont’d)

- Four valves in heart (two AV valves, two SL valves) ensure blood flows in one direction through the heart’s chambers, prevent backflow of blood
- Heart has three major coronary arteries: LAD, CX, RCA

Parasympathetic stimulation of the heart slows firing rate of SA node, slows conduction through AV node, decreases atrial contraction strength, cause small decrease in force of ventricular contraction

Sympathetic stimulation of the heart results in increased force of contraction, increased heart rate, increased BP

Cardiac output is the amount of blood pumped into the aorta each minute by the heart
  - Stroke volume × heart rate
  - Stroke volume is determined by degree of ventricular filling with preload, afterload, contracting/relaxing
Cardiac action potential is a five-phase cycle, reflecting difference in concentration of charged particles across the cell membrane.

- Polarized state is period after repolarization of the myocardial cell (resting state) when the outside of the cell is positive and the interior of the cell is negative.
- Depolarization is the movement of ions across the cell membrane, causing the inside of the cell to become more positive, electrical event expected to result in contraction.
- Repolarization is the movement of ions across the cell membrane in which the inside of the cell is restored to negative charge.

SA node is the heart’s normal pacemaker.

- Built-in rate of SA node, 60-100 beats/min.
- AV junction is AV node, nonbranching portion of bundle of His.
- Bundle of His has pacemaker cells capable of discharging at 40-60 beats/min.
- Purkinje fibers have pacemaker cells capable of firing at 20-40 beats/min.

ECG records electrical activity of large mass of atrial, ventricular cells as specific waveforms, complexes.

- Three types of leads: standard limb leads, augmented limb leads, chest leads.
  - Position of positive electrode on body determines which portion of the left ventricle is seen by each lead.
  - Leads I, II, III are standard limb leads, leads aVR, aVL, aVF are the augmented limb leads, chest leads are identified as V1, V2, V3, V4, V5, V6.
Chapter Summary (Cont’d)

- ECG paper, graph paper with small, large boxes measured in millimeters
  - Horizontal axis of paper corresponds to time
  - Vertical axis of ECG paper measures voltage or amplitude of waveform
    - Voltage measured in millivolts (mV)

- Waveform is movement away from baseline in positive/negative direction
  - Named alphabetically, beginning with P, QRS, T, U
  - Segment is line between waveforms, named by waveform that precedes/follows it
  - Interval is waveform, segment
  - Complex is several waveforms

- P wave represents atrial depolarization, spread of electrical impulse throughout right and left atria
- QRS complex consists of Q wave, R wave, S wave, represents spread of electrical impulse through ventricles (ventricular depolarization)
- T wave represents ventricular repolarization
Chapter Summary (Cont’d)

- PR interval represents interval between onset of atrial depolarization and ventricular depolarization, ST segment represents early part of repolarization of right and left ventricles
  - Point where QRS complex, ST segment meet is junction/J-point
  - TP segment is portion of ECG tracing between end of T wave and beginning of next P wave

Chapter Summary (Cont’d)

- Rhythm that begins in SA node has positive P wave before each QRS complex, P waves that look alike, constant PR interval, regular atrial, ventricular rhythm

Chapter Summary (Cont’d)

- Rhythm that begins in atria has positive P wave shaped differently than P waves that begin in SA node
  - Difference in P-wave configuration occurs because impulse begins in atria, follows a different conduction pathway to AV node
Chapter Summary (Cont’d)

• If AV junction paces the heart, electrical impulse must travel in backward (retrograde) direction to activate atria.
• If P wave is seen, it will be inverted in leads II, III, aVF because impulse is traveling away from the positive electrode.

Chapter Summary (Cont’d)

• If atria depolarize before ventricles, inverted P wave will be seen before QRS complex.
• If atria and ventricles depolarize at the same time, P wave will not be visible because it is hidden in QRS complex.
• If atria depolarize after ventricles, inverted P wave will appear after QRS complex.

Chapter Summary (Cont’d)

• When ectopic site within ventricle assumes responsibility for pacing the heart, electrical impulse bypasses normal intraventricular conduction pathway, results in stimulation of ventricles at slightly different times.
  - Ventricular beats, rhythms usually have QRS complexes that are abnormally shaped, longer than normal (greater than 0.12 second).
Chapter Summary (Cont’d)

• In first-degree AV block, all components of ECG tracing are usually within normal limits except the PR interval
  ▶ Electrical impulses normally travel from the SA node through the atria, but delay occurs in impulse conduction, usually at the level of the AV node

• Second-degree AV blocks are types of incomplete blocks because the AV junction conducts at least some impulses to the ventricles
• Third-degree AV block, impulses generated by SA node are blocked before reaching ventricles, so no P waves are conducted
  ▶ Complete AV block may occur at AV node, bundle of His, bundle branches

• Pacemaker is artificial pulse generator that delivers electrical current to heart to stimulate depolarization
  ▶ Pacemaker systems are usually named according to where the electrodes are located and the route the electrical current takes to the heart
Chapter Summary (Cont’d)

- In right BBB, last portion of QRS complex points up; in left BBB, last portion of QRS complex is directed downward
  - Patients with temperature less than 32°C (89.6°F) may develop a unique ECG pattern called a J wave (also called an Osborn wave), which is seen at the J-point

Chapter Summary (Cont’d)

- Systematic approach to assessment of cardiac patient is important so as not to overlook physical findings/important information pertinent to treatment plan for the patient

Chapter Summary (Cont’d)

- ACSs are conditions caused by a similar sequence of pathological events—temporary/permanent blockage of coronary artery
  - Results in conditions ranging from myocardial ischemia/injury to death (necrosis) of heart muscle
  - ACSs include unstable angina, NSTEMI, STEMI
  - SCD can occur with any of these conditions
Chapter Summary (Cont’d)

- Heart failure, heart is unable to pump enough blood to meet metabolic needs of body
  - Impairs ability of ventricle to fill with/eject blood
  - In acute heart failure, symptoms occur suddenly
  - In chronic heart failure, symptoms develop slowly
  - Often fail together
  - RVF often a result of LVF

Chapter Summary (Cont’d)

- Shock is inadequate tissue perfusion from failure of the cardiovascular system to deliver sufficient O₂, nutrients to sustain vital organ function
  - Underlying cause must be recognized and treated promptly or cell, organ dysfunction, death may result

Chapter Summary (Cont’d)

- Cardiac arrest is the absence of cardiac pump function, confirmed by the absence of a detectable pulse, unresponsiveness, apnea, agonal, gasping respirations
  - May be reversible; death without prompt emergency care
  - SCD is unexpected death from cardiac cause, occurs immediately/within 1 hour of symptom onset
Chapter Summary (Cont’d)

- Some EMS systems have developed protocols that allow field termination of resuscitation efforts in specific circumstances.

Chapter Summary (Cont’d)

- Uncontrolled high BP can lead to vision problems, increased risk of stroke, heart attack, heart failure, kidney failure.
  - Hypertensive urgencies are significant elevations in BP with nonspecific symptoms should be corrected within 24 hours.
  - Hypertensive emergencies are situations that require rapid (within 1 hour) lowering of BP to prevent/limit organ damage.

Chapter Summary (Cont’d)

- Endocarditis occurs when bacteria in the bloodstream lodge, begin to multiply on heart valve/other damaged heart tissue.
  - Untreated, bacteria can damage heart valve, causing malfunction.
Chapter Summary (Cont’d)

- Pericarditis is inflammation of the double-walled sac (pericardium) that encloses the heart
  - Pericardial effusion is increase in volume and/or character of pericardial fluid that surrounds heart
  - Cardiac tamponade occurs when buildup of pericardial fluid compresses heart, impairs contraction, ventricular filling

Chapter Summary (Cont’d)

- Congenital heart defects may obstruct blood flow in heart/vessels near it or cause alteration in normal pattern of blood flow through heart
- Aneurysm is localized dilation/bulging of the blood vessel wall (or wall of heart chamber)
  - Leak/rupture if stretched too far
  - Medical emergency

Chapter Summary (Cont’d)

- Acute arterial occlusion is sudden disruption of arterial blood flow that occurs from thrombus, embolus, tumor, direct trauma to artery
  - Acute limb ischemia results when arterial occlusion suddenly reduces blood flow to arm/leg
  - Intermittent claudication is pain, cramping, muscle tightness, fatigue, weakness of legs when walking/during exercise
Vasculitis is inflammation of the blood vessels; can affect vessels of any type in any organ.
Thrombophlebitis is the development of a clot in the vein with inflammation.
- Superficial thrombophlebitis occurs when a clot develops in a vein near the skin surface.
- DVT if clot develops in deep veins of extremities, increased risk of pulmonary embolism.

Questions?