Chapter 29: Cardiology

Objectives

- Identify risk factors and prevention strategies for cardiovascular disease
- Describe cardiovascular physiology
- Discuss cardiac electrophysiology
- Outline the electrical conduction system of the heart
- Outline cardiovascular assessment

Objectives

- Describe ECG monitoring techniques
- Explain how the ECG tracing relates to the heart's electrical activity
- Describe the steps in ECG interpretation
- Identify features of normal sinus rhythm
- Interpret ECG tracings, including rhythm, site, causes, significance, and prehospital management
Objectives

- Describe assessment and management of patients with cardiovascular disorders
- List relevant information for cardiovascular pharmacological agents
- Identify actions to be taken for prehospital termination of resuscitation

Scenario

An elderly, diabetic female is complaining of weakness and an "aching" feeling in her chest. She is pale, diaphoretic, and has a blood pressure of 100/70 mm Hg, P is 110/min, and blood sugar is 90 mg/dL. You prepare to perform a 12-lead ECG as your partner starts an IV after placing her on oxygen. Suddenly, her eyes roll back and her head slumps limply to the side. As you realize she has no carotid pulse, you note a chaotic rhythm on the monitor and recognize that she is in ventricular fibrillation.
Discussion

- List this patient’s risk factors for cardiovascular disease.
- Describe the steps in initial cardiac care that were indicated on this call?
- What changes on the 12-lead would have confirmed myocardial infarction?
- Outline steps that need to be taken now that her condition has changed.

Cardiovascular Disease

- Age
- Family history
- Diabetes
- Hypertension
- Hyperlipidemia
- High cholesterol
- Cigarette smoking
- Preexisting cardiac disease
- Cocaine use
- Carbohydrate intolerance

Heart Anatomy

- Muscular pump
  - Two atria
  - Two ventricles
- Cone shape
- Size of closed fist
Heart Anatomy

- In mediastinum of thoracic cavity
  - 2/3 of heart's mass lies left of midline of sternum

Pericardium

- Fibrous outer layer, thin inner layer surround heart
- Cavity between layers contains pericardial fluid
  - Reduces friction

Coronary Vessels

- Seven large veins carry blood to the heart
  - Pulmonary veins
  - Superior and inferior vena cavae
- Coronary sinus
**Coronary Vessels**
- Aorta
- Pulmonary trunk
- Coronary arteries supply heart muscle

**Heart Chambers and Valves**
- Septum separates right and left chambers
  - Interventricular septum
  - Interatrial septum

**Atrioventricular Valves**
- Allow blood flow from atria into ventricles
- Prevent backflow
- Tricuspid valve
- Mitral (bicuspid) valve
Semilunar Valves

- Aortic and pulmonary semilunar valves
  - Block blood flow
  - Blood pushes against valves, forcing them open
  - Blood flowing from aorta or pulmonary trunk causes valves to close

Peripheral Circulation

- Flow of blood
  - Ventricles
  - Arteries
  - Arterioles
  - Capillaries
  - Venous system

Capillary Network

- Blood supplied to capillary network by arterioles
- Blood flows through network into venules
  - Regulated precapillary sphincters
- Exchange nutrients and wastes
Arteries and Veins

- Three layers in all blood vessel walls (except capillaries and venules)
  - Tunica intima
    - Inner layer
  - Tunica media
    - Middle layer
  - Tunica adventitia
    - Outer layer

Types of Arteries

- Conducting arteries
  - Large
- Distributing arteries
  - Small to medium
- Arterioles
  - Smallest

Venules

- Similar to capillaries
- Collected blood from capillaries and transport to small veins
- Nutrient exchange across walls of venules
Veins

- Walls are layer of smooth muscle cells
- Medium-sized and large veins
  - Carry blood to venous trunks and then the heart
- Large veins
  - Valves allow blood flow to but not from the heart
  - Prevents backflow of blood

Arteriovenous Anastomoses (AV Shunts)

- Allows blood flow from arteries to veins
  - Without passing through capillaries
- Natural AV shunts
  - Sole of foot and nail beds
  - Regulate body temperature
- Pathological shunts
  - Injury or tumors

Coronary Arteries

- Supply arterial blood to heart muscle
  - Left coronary artery carries about 85% of blood supply to myocardium
  - Right coronary artery carries remainder
- Originate above aortic valve
**Left Coronary Artery**

- Divides into left anterior descending and circumflex arteries
  - Left anterior descending (LAD) supplies:
    - Anterior wall of left ventricle
    - Interventricular septum
  - Circumflex supplies:
    - Lateral and posterior portions of left ventricle
    - Part of right ventricle

**Coronary Arteries**

- Right coronary artery and left anterior descending artery supply:
  - Most of right atrium and ventricle
  - Inferior aspect of left ventricle
- Anastomoses provide collateral circulation

**Coronary Capillaries**

- Exchange nutrients and metabolic wastes
- Merge to form coronary veins
- Coronary sinus empties into right atrium
  - Major vein draining myocardium
Physiology

- Heart is two pumps in one:
  - Low-pressure pump (right atrium and right ventricle)
    - Supplies pulmonary vasculature
  - High-pressure pump (left atrium and left ventricle)
    - Supplies systemic vasculature

Blood Flow through the Heart

- Blood enters right atrium from systemic circulation by inferior and superior vena cavae and from the heart by the coronary sinus
- Blood passes into right ventricle
- Ventricles push blood against tricuspid and semilunar valves
- Blood enters pulmonary trunk

- Pulmonary arteries carry blood to lungs
  - Carbon dioxide released
  - Oxygen picked up
- Blood enters left atrium through pulmonary veins
- Left atrium contracts and fills ventricles
- Blood enters aorta and is sent through body
Cardiac Cycle

- Systole
  - Atrial
  - Ventricular

- Diastole
  - Atrial
  - Ventricular

Heart Action during Atrial Systole

Heart Action during Ventricular Systole
Cardiac Output

- Stroke volume
- Heart rate
- Contractility
- Starling's law

Nervous System Control of the Heart

- Extrinsic control by parasympathetic and sympathetic nerves influences:
  - Heart rate
  - Conductivity
  - Contractility

Nervous System Control of the Heart

- Sympathetic and parasympathetic nerve fibers in atria
- Ventricles mainly have sympathetic nerves
**Sympathetic Control**

- Postganglionic sympathetic fibers release norepinephrine; have effects on myocardium:
  - Inotropic
  - Dromotropic
  - Chronotropic

**Sympathetic Control**

- Sympathetic stimulation of the heart
  - Dilation of coronary blood vessels
  - Constriction of peripheral vessels
  - Increased oxygen demands of the heart met by increase in blood and oxygen supply

**Sympathetic Control**

- Norepinephrine release results from stimulation of alpha- and beta-adrenergic receptors in the heart
- Sympathetic stimulation increases the heart rate
Parasympathetic Control

- Parasympathetic innervation of the heart by vagus nerve
  - Continuous inhibitory influence on the heart by decreasing heart rate and contractility

Hormonal Regulation of the Heart

- Sympathetic impulses are transmitted to adrenal medulla and blood vessels
  - Adrenal medulla secretes epinephrine and norepinephrine

Epinephrine

- Epinephrine increases rate and force of contraction
- Epinephrine also:
  - Constricts blood vessels in skin, kidneys, GI tract, and other organs
  - Dilates skeletal and coronary vessels
Norepinephrine
- Causes constriction of peripheral blood vessels in most areas of the body
- Stimulates cardiac muscle

Role of Electrolytes
- Myocardial cells bathed in electrolyte solution
- Electrolytes that influence cardiac function:
  - Calcium
  - Potassium
  - Sodium
  - Magnesium

Electrophysiology of the Heart
- Two cell types in myocardium
  - Cells of electrical conduction system
    - Formation and conduction of electric current
  - Working myocardial cells
    - Contractility
Electrical Activity of Cardiac Cells

- Ions are charged particles that are electrically positive or negative
  - Cations
  - Anions

Electrical Activity of Cardiac Cells

- Charged particles are like magnets:
  - Need energy to push apart if opposite electrical charges
  - Need energy to join if like electrical charges

Membrane Potentials

- Magnetic-like attraction gives separated particles of opposite charges potential energy
  - Membrane potential between inside and outside of cell
  - Charge between inside and outside of cells expressed in millivolts (mV)
Resting Membrane Potential

- Cell in “resting” state
- Has electrical charge difference
  - Resting membrane potential (RMP)
  - Inside of cell is negative compared to outside of cell membrane

Resting Membrane Potential

- RMP due to difference between intracellular and extracellular potassium ion level

Depolarization

- Sodium—Positively charged ion on outside of cell
  - Chemical and electrical gradient
  - Tends to move intracellularly

Sodium channels remain closed in resting cell membrane
Depolarization

* Depolarization (electrical conduction) takes place when sodium rushes into the cell, making inside more positive compared with outside.

Diffusion through Ion Channels

* Cell membrane is:
  - Relatively permeable to potassium
  - Less permeable to calcium chloride
  - Minimally permeable to sodium
Diffusion through Ion Channels

- Protein-lined channels allow passage of ions through cell membrane
- Permeability influenced by:
  - Electrical charge
  - Size
  - Gating proteins

Sodium-Potassium Pump

- Actively pumps sodium ions out of cell and potassium ions into cell
- Transports three sodium ions out for every two potassium ions taken in
- Returns cell to its resting state

Sodium-Potassium Exchange Pump
Channels in Cardiac Muscle Cells

- Sodium and calcium ions enter cells through two separate channel systems in cell membrane:
  - Fast channels
  - Slow channels

Channels

- Fast channels are sensitive to small changes in membrane potential:
  - As cell nears threshold level:
    - Fast sodium channels open
    - Sodium ions rush intracellularly
    - Rapid depolarization
  - Slow channels are selectively permeable to calcium and sodium

Cell Excitability

- Nerve and muscle cells are capable of producing action potential
  - Threshold potential
**Propagation of Action Potential**
- Action potential on cell membrane stimulates adjacent cell membrane
  - Excitation process is spread along length of cell and on to the next
  - All-or-none principle

---

**Cardiac Action Potential**
- Phase 0 (rapid depolarization phase)
- Phase 1 (early rapid depolarization phase)
- Phase 2 (plateau phase)
- Phase 3 (terminal phase of rapid repolarization)
- Phase 4 (resting period)

---

**Action Potential of Myocardial Cells**
Absolute Refractory Period

- Absolute refractory period
  - Cardiac muscle cell is completely insensitive to stimulation
- Refractory period of ventricles is about same duration as action potential

Relative Refractory Period

- Muscle cell is more difficult than normal to excite but can still be stimulated

Electrical Conduction System

- Sinoatrial node (SA node)
- Atrioventricular (AV) junction
  - AV node
  - Bundle of His
- His-Purkinje system
  - Bundle branches
    - Right
    - Left anterior fascicle
    - Left posterior fascicle
Characteristics of Myocardial Cells

- Automaticity
- Excitability
- Conductivity
- Contractility

Intrinsic Rates

- SA node: 60-100/min
- AV junctional tissue: 40-60/min
- Ventricles (bundle branches and Purkinje fibers): 20-40/min

Ectopic Electrical Impulse Formation

- Ectopic beat results when pacemaker function is assumed by cells other than in SA node
  - Premature beats
  - Early in diastole before SA node is scheduled to discharge
Ectopic Electrical Impulse Formation

- Premature beats
  - Atrial origin—Premature atrial complexes (PACs)
  - Junctional origin—Premature junctional complexes (PJC"
  - Ventricular origin—Premature ventricular complexes (PVC"

- Two mechanisms for ectopic impulse generation in the heart:
  - Enhanced automaticity
  - Reentry

Enhanced Automaticity

- Acceleration in depolarization
- Due to high leakage of sodium ions into cells
  - Cells reach threshold prematurely
  - Rate of impulse formation in potential pacemakers increases beyond their inherent rate
- Causes dysrhythmias in Purkinje fibers
  - Other myocardial cells
**Enhanced Automaticity**

- Excess catecholamines
- Digitalis toxicity
- Hypoxia
- Hypercapnia
- Myocardial ischemia or infarction
- Increased venous return (preload)
- Hypokalemia or electrolyte abnormalities
- Atropine administration

**Reentry**

- Reactivation of myocardial tissue by same impulse
- Occurs when electrical impulse is delayed, blocked in segments of the heart’s electrical conduction system

**Reentry**

Conduction through normal and severely depressed Purkinje fibers
Reentry

- Reentry dysrhythmias can occur in:
  - SA node
  - Atria
  - AV junction
  - Bundle branches
  - Purkinje fibers

Delayed Impulses

- Causes of delayed or blocked impulses:
  - Myocardial ischemia
  - Certain drugs
  - Hyperkalemia

Assessment of Cardiac Patient

- Chief complaint
- History of event and significant past medical history
- Physical exam
Chief Complaint

- Cardiac disease chief complaints
  - Chest pain or discomfort
  - Shoulder, arm, neck, or jaw pain or discomfort
  - Dyspnea
  - Syncope
  - Abnormal heart beat or palpitations
  - May vary

Chest Pain or Discomfort

- Common chief complaint in myocardial infarction
- Noncardiac causes of chest pain
  - Pulmonary embolus
  - Pleurisy
  - Reflux esophagitis
- History of chest pain is important
  - OPQRST method

Dyspnea

- May occur with myocardial infarction
- Symptom of heart failure
- Dyspnea unrelated to heart disease
  - Chronic obstructive pulmonary disease
  - Respiratory infection
  - Pulmonary embolus
  - Asthma
**Dyspnea**

Factors important to differentiate dyspnea:
- Duration
- Circumstances of onset
- What aggravates or relieves, including medications
- Previous episodes
- Associated symptoms
- Orthopnea
- Prior cardiac problems

**Syncope**

- Sudden decrease in cerebral perfusion
- Cardiac causes decrease cardiac output
  - Dysrhythmias

**Syncope**

- Noncardiac causes of syncope
  - Stroke
  - Drug or alcohol intoxication
  - Aortic stenosis
  - Pulmonary embolism
  - Hypoglycemia
Syncope—History

- Aura (nausea, weakness, lightheadedness)
- Circumstances
  - Position before event
  - Pain
  - Stress
- Duration of syncopal episode
- Symptoms before syncope
- Other symptoms
- Previous episodes

Palpitations

- Sometimes normal
- May indicate serious dysrhythmia

Palpitations

- History and physical exam
  - Pulse rate (if obtained)
  - Regular versus irregular rhythm
  - Circumstances
  - Duration
  - Chest pain, diaphoresis, syncope, confusion, dyspnea
  - Previous episodes
  - Medications
Significant Past Medical History

- Is the patient taking prescription medications, particularly cardiac medications?
  - Digoxin
  - Furosemide (or other diuretics)
  - Nitroglycerin
  - Beta blockers

- Is the patient being treated for any other illness?

---

Significant Past Medical History

- Has the patient ever had:
  - Myocardial infarction or angina pectoris
  - Coronary artery bypass procedure or angioplasty
  - Implanted pacemaker or ICD
  - Heart failure
  - Hypertension
  - Diabetes
  - Chronic lung disease

---

Significant Past Medical History

- Allergies

- Other risk factors for cardiac event

- Implanted pacemaker or implantable cardioverter-defibrillator (ICD)
Physical Examination

- Classic presentation of myocardial infarction:
  - Pain or discomfort under sternum for longer than 15 min

- Other signs and symptoms
  - Apprehension
  - Diaphoresis
  - Dyspnea
  - Nausea and vomiting
  - Sense of impending doom

- Atypical presentations

---

Initial Assessment

- Level of consciousness
- Respiration
- Pulse (rate, regularity)
- Blood pressure
- Skin

---

Physical Examination

- “Look-listen-feel” approach

---

**Look**

- Skin color, capillary refill, skin moisture
  - Oxygenation (pulse oximetry)
  - Cardiac function (peripheral perfusion)
- Jugular vein distention (JVD)
  - Evaluate with head elevated to 45 degrees
  - Difficult to assess in obese patients

**Look**

- Peripheral and presacral edema
  - Back-pressure in venous circulation
  - Obvious in dependent areas
  - Nonpitting
    - Minimal depression of tissue after removal of finger pressure
  - Pitting
    - Depression of tissue remains after removal of finger pressure

**Look**

- Indicators of cardiac disease
  - Nitroglycerin patch
  - Midsternal scar from coronary surgery
  - Implanted pacemaker or automatic implantable cardioverter-defibrillator (left upper chest; abdominal wall)
  - Medic alert information
Listen

- Lung sounds
  - Equality
  - Adventitious sounds
    - May indicate pulmonary congestion or edema
- Heart sounds
  - Gallops

Heart Sounds

- Auscultate for:
  - Frequency (pitch)
  - Intensity (loudness)
  - Duration
  - Timing in the cardiac cycle

Auscultating Heart Sounds
Point of Maximal Impulse (PMI)
- Apical impulse
  - Visible and palpable
  - Produced by contraction of left ventricle
- Pulse deficits noted by palpating or auscultating apical impulse and carotid pulse simultaneously

Heart Sounds
- Aortic
  - 2nd intercostal space to right of sternum
- Pulmonic
  - 2nd intercostal space to left of sternum

Heart Sounds
- Tricuspid
  - 5th left intercostal space close to sternal border
- Mitral
  - 5th intercostal space medial to left midclavicular line
  - Over left ventricle
  - Apical area or apex
**S1**

- "Lub" sound
  - Mitral and tricuspid valve closure
  - Beginning of ventricular systole
- Diaphragm of stethoscope at apex of heart
  - 5th intercostal space

**S2**

- "Dub" sound
  - Aortic and pulmonic valve closure
  - End of ventricular systole
- Use diaphragm of stethoscope at 2nd intercostal space to right and left of the sternum
  - Aortic and pulmonic areas

**S3**

- Extra heart sound
  - Rapid ventricular filling
- Common in children, athletes, and young adults
- Abnormal in persons >30 y/o
- Use bell of stethoscope at apex
S3
- Sounds like “Ken-Tuck-Y”
  - Emphasis on “Tuck”
  - “Ken” = S1, “Tuak” = S2, “Y” = S3
- Warning sign of congestive heart failure

S4
- Last of ventricular filling
- Tensing of atrioventricular valves
- Atrial contraction
- Just before S1
- Heard at apex with stethoscope bell
- Sounds like “Ten-nes-see”
  - Emphasis on “Ten”
  - “Ten” = S4, “Nes” = S1, “See” = S2

Feel
- Peripheral or presacral edema
- Pulse
  - Rate
  - Regularity
  - Equality
  - Pulse deficit
  - Pulsus paradoxus
  - Pulsus alternans
Feel

- Skin
  - Diaphoretic, pale skin
  - Peripheral vasoconstriction
  - Sympathetic stimulation
  - Cyanosis
    - Poor oxygenation
  - Fever
    - Infection

ECG Monitoring

- Graphic representation of the heart's electrical activity
- Generated by depolarization and repolarization of atria and ventricles

ECG Monitoring

- Tool to identify cardiac abnormalities:
  - Abnormal heart rates and rhythms
  - Abnormal conduction pathways
  - Hypertrophy or atrophy of portions of the heart
  - Location of ischemic or infarcted cardiac muscle
ECG Monitoring

- ECG tracing shows the heart's electrical activity
- Does not provide information regarding mechanical events (e.g., force of contraction or blood pressure)

Sum of action potentials in heart during cardiac cycle measured on body surface
- Obtained by applying electrodes to skin and connecting them to an ECG machine
- Voltage changes fed to machine
  - Amplified and displayed on screen
  - Printed on ECG paper

Voltage

- Positive
  - Upward deflection on ECG tracing
- Negative
  - Downward deflection on ECG tracing
- Isoelectric
  - Straight baseline on ECG
  - No current detected
ECG Leads

- Two surface electrodes of opposite polarity
  - Bipolar lead
    - Two electrodes of opposite polarity
  - Unipolar lead
    - Single positive electrode and reference point

Leads

- Bipolar leads
  - Limb leads
    - I, II, III
- Unipolar leads
  - Augmented limb leads
    - aVR, aVL, aVF
  - Precordial leads
    - V1 through V6

- Each lead assesses electrical activity from a different angle

Lead Comparison

| I, II, III | Limb lead | Bipolar |
| aVR, aVL, aVF | Limb lead | Unipolar |
| V1-V6 | Chest lead | Unipolar |
### Leads and Cardiac Surfaces

<table>
<thead>
<tr>
<th>Lead</th>
<th>Cardiac Surface Viewed</th>
</tr>
</thead>
<tbody>
<tr>
<td>II, III, aVF</td>
<td>Inferior wall</td>
</tr>
<tr>
<td>V1, V2</td>
<td>Septum</td>
</tr>
<tr>
<td>V3, V4</td>
<td>Anterior wall</td>
</tr>
<tr>
<td>V5, V6, I, aVL</td>
<td>Lateral wall</td>
</tr>
</tbody>
</table>

### Waveforms

- Leads produce different ECG tracings:
  - If depolarization moves toward a positive electrode, ECG shows an upward deflection.
  - If the wave moves away from a positive electrode, a negative deflection appears on the ECG.

### Rule of Electrical Flow

- If the polarity moves toward a positive electrode, an upward deflection appears on the ECG.
- If the polarity moves toward a negative electrode, a downward deflection appears on the ECG.
Standard Limb Leads

- Record difference in electrical potential between left arm, right arm, and left leg electrodes
- Represent axes

Axis

- Average direction of the heart’s electrical activity
- Triaxial reference system

Axis

- Lead I is a lateral (leftward) lead
  - Assesses electrical activity from a viewpoint defined as 0° on a circle divided into an upper negative 180° and a lower positive 180°
Axis

- Leads II and III are inferior leads
  - Assess the heart's electrical activity from vantage points of +60° and +120°

Bipolar Lead Placement

**Limb lead placement**

<table>
<thead>
<tr>
<th>Lead</th>
<th>Positive Electrode</th>
<th>Negative Electrode</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Left arm</td>
<td>Right arm</td>
</tr>
<tr>
<td>II</td>
<td>Left leg</td>
<td>Right arm</td>
</tr>
<tr>
<td>III</td>
<td>Left leg</td>
<td>Left arm</td>
</tr>
</tbody>
</table>

Augmented Limb Leads

- Same electrodes as limb leads
- Record difference in electrical potential between extremity lead sites and a reference point
  - Zero electrical potential
  - At center of the heart’s electrical field
Augmented Limb Leads

- Axis of each lead is formed by line from electrode site to center of the heart.

aVR, aVL, and aVF leads intersect at angles different from those of the standard limb leads.

Produce three other intersecting lines of reference:
- With standard limb leads, these leads make up a hexaxial reference system.

Lead aVR

- Distant recording electrode
- Looks at heart from right shoulder
Lead aVL

- Lateral lead
- Records electrical activity from left shoulder
  - 30°

Lead aVF

- Inferior lead
- Records electrical activity from left lower extremity
  - 90°

Limb Leads

- Leads II, III, aVF
  - Inferior leads
- I, aVL
  - Lateral leads
Modified Lead Recording

- Limb lead placement altered to mimic precordial leads (V₁ through V₆)
  - Modified chest leads
  - MCL₁ to MCL₆
- May help:
  - Distinguish between supraventricular tachycardia with aberration and ventricular tachycardia
  - Diagnose bundle branch blocks

MCL₁

- Positive electrode in V₂ position
  - 4ᵗʰ intercostal space, right of sternum
- Negative electrode placed anteriorly
  - Below lateral end of left clavicle

MCL₆

- Positive electrode on left midaxillary line at 5ᵗʰ intercostal space
  - As for lead V₄
- Negative electrode placed anteriorly, below left shoulder
Routine ECG Monitoring

- Usually lead II or MCL
  - Best leads to visualize P waves
  - Monitor for dysrhythmias

Single-Lead ECG Monitoring

- Information gathered
  - Heart rate
  - Regularity
  - Length of conduction in areas of the heart

- Limitations
  - May fail to reveal abnormalities
  - Particularly ST segment changes that signal myocardial injury or infarction

12-Lead ECG Monitoring

- 10 electrodes
  - Four limb leads (right arm, right leg, left arm, left leg)
    - Leads I, II, and III, and aVF, aVL, and aVR
  - Six chest leads
    - V1 through V6
12-Lead ECG Monitoring

- Leads view left ventricle from position of its positive electrode

12-Lead ECG Monitoring

- Identifies ST segment and T-wave changes
  - Myocardial ischemia, injury, and infarction
- Identifies VT in wide-complex tachycardia
- Determines electrical axis
  - Presence of fascicular blocks
- Determines presence and location of bundle branch blocks

Precordial Leads

- Six precordial leads are projected through anterior chest wall toward back
- Positive leads are placed on chest in reference to thoracic landmarks
  - Record electrical activity in transverse or horizontal plane
Precordial Leads

- V₁ and V₂: Septal leads
- V₃ and V₄: Anterior leads
- V₅ through V₆: Lateral leads

Application of Monitoring Electrodes

- Electrodes are applied to chest wall
- When applying electrodes:
  - Cleanse area to remove moisture and dirt
  - Use inner surfaces of arms and legs
  - Attach ECG cables to electrodes
  - Attach electrodes
  - Turn on ECG monitor
  - Obtain baseline tracing
  - Record tracing at significant events
Monitoring Electrodes

- If poor signal, recheck cable connections and electrode contact

- Other causes of poor signal
  - Excessive body hair
  - Dried conductive gel
  - Poor electrode placement
  - Diaphoresis

12-Lead Electrode Application

Locate the jugular notch
Palpate for the angle of Louis

Follow the angle of Louis to patient's right until it articulates with 2nd rib
Locate the 2nd IC space (immediately below 2nd rib)
12-Lead Electrode Application

From the 2nd IC space, the 3rd and 4th IC spaces can be found.

V1 is positioned in the 4th IC space just right of the sternum.

Place V2 electrode in the 4th IC space just left of the sternum.

From V1, find the corresponding IC space on the left side of the sternum.

12-Lead Electrode Application

From V2 position, locate 5th IC space, follow to the midclavicular line.

Position V4 electrode in 5th IC space in midclavicular line.
12-Lead Electrode Application

Position V₆ in the midaxillary line, level with V₄.
9-Lead from a 3-Lead

- Obtain 9-lead reading from 3-lead monitor
- Enable diagnostic setting (if available)
- Run leads I, II, and III
  - Obtain a strip of each lead and label

9-Lead from a 3-Lead

- Leave monitor in lead III
  - Negative electrode at left shoulder
- Move left leg cable to each of MCL positions (from V₁ to V₆) to obtain a readout
- Label each strip

9-Lead ECG Readout

ECG Graph Paper

- Standardized to allow ECG analysis

- Squares 1 mm in height and width
  - Darker lines every fifth square, vertically and horizontally
  - Large square is 5 mm high and 5 mm wide

---

ECG Graph Paper

- As paper moves past stylus of ECG machine, it measures time and amplitude

- Time measured on horizontal plane
  - Side to side

- ECG recorded at standard speed of 25 mm/sec:
  - Each small square equals 1 mm (0.04 second)
  - Each large square equals 5 mm (0.20 second)

---

ECG Graph Paper

- Amplitude measured on vertical axis (top to bottom) of graph paper

- Each small square of graph paper = 0.1 mV

- Each large square (five small squares) = 0.5 mV
Calibration

- Sensitivity of 12-lead ECG machine is standardized

- When calibrated, a 1 mV electrical signal produces a 10 mm deflection (2 large squares) on ECG tracing

Time Interval Markings

- Denoted by short vertical lines on ECG graph paper

- At standard speed, the distance between each short vertical line is 75 mm (3 sec)
Each waveform represents conduction of an electrical impulse through a specific part of the heart.

Waveforms begin and end at isoelectric line.

Isoelectric line shows absence of electrical activity in cardiac tissue.

Deflections above baseline—positive
- Electrical flow toward positive electrode
Deflections below baseline—negative
- Electrical flow away from positive electrode

Normal ECG consists of a P wave, a QRS complex, and a T wave.

Also evaluate:
- PR interval
- ST segment
- QT interval

Combination of these waves represents a single heartbeat.
- One complete cardiac cycle.
**P Wave**

- First positive (upward) deflection on ECG
- Atrial depolarization
- Rounded and precedes QRS complex
  - Begins with first positive deflection from baseline
  - Ends when wave returns to baseline

**Duration normally**

- **< 0.10 sec**
- **Amplitude normally**
  - **0.5 - 2.5 mm**
- Followed by QRS complex unless conduction disturbances

**PR Interval**

- Time it takes for electrical impulse to be conducted through atria and AV node up to ventricular depolarization
  - Measured from beginning of P wave to beginning of next deflection on baseline
  - **0.12 - 0.20 sec**
PR Interval

- Normal PR interval indicates electrical impulse conducted through atria and AV node, normally and without delay.

QRS Complex

- Three individual waves:
  - Q
  - R
  - S

- Begins where first wave of complex deviates from baseline.

- Ends where last wave of complex begins to flatten at, above, or below baseline.

Direction of QRS complex may be:

- Predominantly positive
  - Upright
- Predominantly negative
  - Inverted
- Biphasic
  - Partly positive, partly negative.
**QRS Complex**

- Normal QRS complex is narrow and sharply pointed
- Duration
  - $\leq 0.08 - 0.10 \text{ sec}$
- Amplitude
  - $< 5 \text{ mm to } >15 \text{ mm}$

**Q Wave**

- First negative (downward) deflection of QRS complex on ECG
  - May not be present in all leads
- Depolarization of interventricular septum

**R Wave**

- First positive deflection after the P wave
- Subsequent positive deflections in the QRS complex that extend above the baseline and that are taller than the first R wave are called R prime ($R'$), R double prime ($R''$), etc.
S Wave

- Negative deflection after R wave
  - Subsequent negative deflections are called S prime (S'), S double prime (S''), R and S waves are electrical forces from depolarization of right and left ventricles

QRS Complex

- Follows P wave
- Approximate beginning of mechanical systole of ventricles
  - Continues through onset of T wave
- Ventricular depolarization
  - Conduction of electrical impulse from AV node through bundle of His, Purkinje fibers, and right and left bundle branches
ST Segment

- Early phase of repolarization of ventricles
- Follows QRS complex
- Ends with onset of T wave

ST Segment

- ST segment “takes off” from the QRS complex at J point

ST Segment

- Position of ST segment is commonly judged using baseline of PR or TP interval for reference
  - ST segment elevation
  - ST segment depression
**ST Segment**
- Abnormal ST segments
  - Infarction
  - Ischemia
  - Pericarditis
  - After digitalis administration
  - Other disease states

**T Wave**
- Repolarization of ventricular cells
- Last part of ventricular systole
- Above or below isoelectric line
- Usually rounded and slightly asymmetrical

**T Wave**
- Deep, symmetrically inverted T waves may suggest cardiac ischemia
- T wave elevated more than half the height of the QRS complex may indicate:
  - Onset of myocardial ischemia
  - Hyperkalemia
**QT Interval**

- Onset of the QRS complex until end of T wave

**Artifact**

- Deflections on ECG produced by factors other than the heart's electrical activity

- Causes
  - Improper grounding of ECG machine
  - Patient movement
  - Loss of electrode contact with skin
  - Patient shivering or tremors
  - External chest compressions

**Artifact—Muscle Tremors**
To determine if potential for life-threatening rhythm disturbances, ask:

- Is the patient sick?
- What is the heart rate?
- Are there normal-looking QRS complexes?
- Are there normal-looking P waves?
- What is the relationship between P waves and QRS complexes?
Step 1: Analyze the QRS Complex

- Analyze for regularity and width
- Supraventricular QRS complexes are ≤0.10 sec wide

Normal QRS Complexes

- Complexes >0.12 sec wide indicate:
  - Conduction abnormality in ventricles
  - Focus that originates in ventricles and is abnormal
Analyzing the QRS Complex

- To evaluate abnormal QRS width, identify lead with widest QRS complex
  - Part of QRS complex may be blended with baseline in some leads

Abnormal QRS Complexes
Step 2: Analyze the P Waves
- Present?
- Regular?
- One P wave for each QRS complex, and a QRS complex follows each P wave?
- Upright or inverted?
- All alike?
Step 3: Analyze the Rate

- Rate < 60 = Bradycardia
- Rate ≥ 100 = Tachycardia

Heart Rate Rulers

- Various manufacturers
- Accurate if rhythm is regular
  - May not be readily available

Heart Rate Calculator Ruler
Triplicate Method
- Memorize two sets of numbers: 300-150-100 and 75-60-50
  - Accurate only if rhythm is regular and >50 bpm

R-R Method 1
- Measure distance in seconds between peaks of two consecutive R waves
- Divide this into 60 to obtain heart rate

R-R Method 2
- Count large squares between peaks of two consecutive R waves
- Divide into 300 to obtain heart rate
R-R Method 3

- Count small squares between peaks of two consecutive R waves
- Divide into 1500 to obtain heart rate

---

6-Second Method

- Least accurate method
- Quickly obtain rate in regular and irregular rhythms
- Count number of QRS complexes in 6-sec interval and multiply number by 10

---

Step 4: Analyze the Rhythm

- To analyze rhythm, compare R-R intervals from left to right.
- If distances between R waves are equal or vary by <0.16 sec, rhythm is regular
- If shortest and longest R-R intervals vary by >0.16 sec, rhythm is irregular
Determining the Rhythm

Regular Rhythm

Step 4: Analyze the Rhythm

- Regularly irregular rhythm
  - Patterned irregularity or group beating
Step 4: Analyze the Rhythm

- Occasionally irregular
  - Only one or two R-R intervals are unequal to the others

Step 4: Analyze the Rhythm

- Irregularly irregular
  - Totally irregular; no relationship between R-R intervals

Step 5: Analyze the PR Interval

- Time it takes for electrical impulse to be conducted through atria and AV node
- Should be constant across ECG tracing
Step 5: Analyze the PR Interval

- **Prolonged PR interval**
  - Delay in impulse conduction through AV node or bundle of His
  - AV block

- **Short PR interval**
  - Impulse progressed from atria to ventricles through pathways other than AV node

Normal PR Intervals

Abnormal PR Intervals

Causes of Cardiac Dysrhythmias
- Myocardial ischemia
- Autonomic nervous system imbalance
- Distention of heart chambers
- Acid-base abnormalities
- Hypoxemia
- Electrolyte imbalance
- Drug effects or toxicity
- Electrical injury
- Hypothermia
- CNS injury
Classification of Dysrhythmias

- Based on a number of factors
  - Changes in automaticity versus disturbances in conduction
  - Cardiac arrest (lethal) rhythms and noncardiac arrest (nonlethal) rhythms
  - Site of origin

Dysrhythmias Originating in the SA Node

- Sinus bradycardia
- Sinus tachycardia
- Sinus dysrhythmia
- Sinus arrest

Dysrhythmias Originating in the Atria

- Wandering pacemaker
- Premature atrial complex (PAC)
- Paroxysmal supraventricular tachycardia (PSVT)
- Atrial flutter
- Atrial fibrillation
Dysrhythmias Originating in the AV Node
- Premature junctional complex (PJC)
- Junctional escape complexes or rhythms
- Accelerated junctional rhythm

Dysrhythmias Originating in the Ventricles
- Ventricular escape complexes or rhythm
- Premature ventricular complex (PVC)
- Ventricular tachycardia (VT)
- Ventricular fibrillation (VF)
- Asystole
- Artificial pacemaker rhythm

Disorders of Conduction
- AV blocks
  - First-degree AV block
  - Second-degree AV block type I
  - Second-degree AV block type II
  - Third-degree AV block
- Disturbances of ventricular conduction
- Pulseless electrical activity (PEA)
- Preexcitation syndrome: Wolff-Parkinson-White (WPW) syndrome
Treatment Guidelines

- Treat patient, not monitor
- Algorithms presume condition persists,
  - In cardiac arrest CPR is always performed
- Apply different interventions if indications exist

Airway, ventilation, oxygenation, chest compression, and defibrillation

- Take precedence over initiating an IV line or administering drugs

Some medications can be administered via endotracheal tube
- Use endotracheal dose 2-2½ times IV dose for adults
- In arrest, with few exceptions IV meds are administered rapidly by bolus method
  - Follow with a 20-30 mL bolus of IV fluid and immediately elevate extremity
Dysrhythmias Originating in the SA Node

- Sinus dysrhythmias
  - Often from increases or decreases in vagal tone
- SA node dysrhythmias
  - SA node gets inhibitory parasympathetic impulses from vagus nerve to keep rate below discharge rate of pacemaker cells
    - If vagal discharge increases, heart rate becomes bradycardic
    - If vagal discharge decreases, sympathetic stimulation results in sinus tachycardia

Dysrhythmias Originating in the SA Node

- SA node dysrhythmias
  - Normal duration of QRS complex
    - If no bundle branch block
  - Upright P waves in lead II
  - P waves similar
  - Normal duration of PR interval
    - If no AV block

Sinus Rhythm
Sinus Bradycardia

- Slowing of pacemaker rate of SA node
  - Adult HR <60 bpm

- Causes
  - Sinus node disease
  - Increased vagal tone
  - Hypoxia
  - Hypothermia
  - Drugs
    - AMI

- Can decrease cardiac output and cause:
  - Hypotension
  - Syncope
  - Angina

Sinus Bradycardia—Management

- Observe, if patient clinically stable

- If unstable, treatment may include:
  - Oxygen
  - Atropine
  - Dopamine infusion
  - Epinephrine infusion
  - Transcutaneous pacing
**Sinus Tachycardia**

- Increase in rate of sinus node discharge
  - Adult HR >100 bpm
- Treat underlying cause

**Causes**
- Exercise
- Fever
- Ingestion of drugs
- Smoking
- Hypovolemia
- Anemia
- Congestive heart failure

**Sinus Dysrhythmia**

- Difference between the longest and shortest R-R intervals is >0.16 sec
- Often normal
- Occurs in heart disease, drug treatment
- No clinical management needed
Sinus Arrest

- Marked depression in SA node automatically
- Sinus node fails, causes periods of cardiac standstill until:
  - Other pacemakers discharge
  - Sinus node resumes normal function
- Causes:
  - Increased vagal tone
  - Hypoxia
  - Ischemia
  - Excess digitalis or propranolol
  - Hyperkalemia
  - Damaged SA node

Sinus Arrest

- May cause decreased cardiac output
- Syncope
- Asystole possible
- Observe
- If symptomatic:
  - Atropine
  - TCP

---

**Dysrhythmias Originating in the Atria**

- Originate in tissues of atria or internodal pathways

- Causes of atrial dysrhythmias
  - Ischemia
  - Hypoxia
  - Atrial dilation caused by:
    - Congestive heart failure
    - Mitral valve abnormalities
    - Increased pulmonary artery pressures

**ECG features common to atrial dysrhythmias** (if no ventricular conduction disturbance):

- Normal QRS complexes
- P waves (if present) that differ in appearance from sinus P waves
- Abnormal, shortened, or prolonged PR intervals
Wandering Atrial Pacemaker

- Transfer of pacemaker sites from sinus node to other pacemaker in atria or AV junction
- Shift in site is usually transient
- May be normal
- Can result from digitalis toxicity
- Not often clinically significant
Premature Atrial Complex

- Single impulse originating in atria
  - Outside sinus node
- Single ectopic pacemaker site or multiple sites in atria
- Enhanced automaticity or reentry mechanism
- Isolated PACs not significant
- Multiple PACs may predispose to SVT

**Causes**
- Increased sympathetic tone
- Stimulant use
- Drugs
- Electrolyte imbalance
- Hypoxia
- Cardiac disease

**Treatment**
- Observation
- If not conducted:
  - May need bradycardia treatment
Supraventricular tachycardias (SVTs)

- Paroxysmal supraventricular tachycardia (PSVT)
- Nonparoxysmal atrial tachycardia
- Multifocal atrial tachycardia
- Junctional tachycardia
- Atrial flutter
- Atrial fibrillation

Paroxysmal supraventricular tachycardia (PSVT)

- Supraventricular tachycardia that begins abruptly
- Atrial origin
- Paroxysmal atrial tachycardia (PAT)
- AV junction
- Paroxysmal junctional tachycardia (PJ T)
- Rapid atrial or junctional depolarization overrides SA node

Often reentry mechanism

- Stress, overexertion, tobacco, caffeine
- Wolff-Parkinson-White syndrome
- May be tolerated briefly if healthy

Can cause:

- Compromised cardiac output
- Hypotension syncope
- CHF
SVT and PSVT—Treatment

- **Stable**
  - Vagal maneuvers
  - Adenosine
  - Calcium Channel Blockers or
  - Beta blockers

- **Unstable**
  - Synchronized cardioversion
    - 50 J (PSVT or Atrial Flutter)
    - 100 J
    - 200 J
    - 360 J
    - Or equivalent biphasic

PSVT

Atrial Flutter

- Usually rapid atrial reentry focus
- Conduction variable
- Sawtooth or picket-fence appearance
- Loss of atrial kick
- May decrease perfusion

Associated with:
- Cardiomyopathy
- Cardiac hypertrophy
- Digitalis toxicity
- Hypoxia
- CHF
- Pericarditis
- Myocarditis

Atrial Flutter

Multiple areas of reentry in atria or ectopic atrial pacemakers

Chaotic impulses too numerous to be conducted by AV node through ventricles
  - AV conduction is random
  - Ventricular response irregular
  - Usually rapid unless patient is on medication to slow ventricular rate

Atrial Fibrillation

P waves absent

F waves

Atrial kick lost

More unstable with rapid ventricular response

Cardiac decompensation

Causes
  - "Holiday heart" syndrome
  - Rheumatic heart disease
  - CHF
  - Cardiac disease
  - Chest trauma

Atrial Fibrillation

Management based on:
- Time of rhythm onset
- Signs and symptoms
- Heart function

Rate control with:
- Diltiazem
- Beta blockers
- Magnesium

Unstable
- Synchronized cardioversion
  - 50 J (Atrial Flutter)
  - 100 J
  - 200 J
  - 300 J
  - 360 J
- Or equivalent biphasic

Atrial Fibrillation and Wolff-Parkinson-White Syndrome

Do not administer:
- Adenosine
- Diltiazem or verapamil
- Beta blockers
- Digoxin
Dysrhythmias of the AV Junction

- If SA node and atria don’t generate electrical impulses, AV node or area surrounding it may assume role of secondary pacemaker

Dysrhythmias of the AV Junction

- May occur because of:
  - Hypoxia
  - Ischemia
  - Myocardial infarction
  - Drug toxicity

- Usually benign arrhythmia

- Assess to determine patient’s response to rhythm

Premature Junctional Complex

- Single electrical impulse
- Originates in AV junction
- Occurs before next expected sinus impulse
- P waves occur before, during, or after QRS
- Abnormal P wave
- PRI < 0.12 sec
Premature Junctional Complex
- Usually no clinical significance
- No treatment needed

Causes
- Medications
- Increased vagal tone
- Hypoxia
- Congestive heart failure
- AV junction damaged

PJC
- Junctional Escape Complex or Rhythm
  - Isolated impulse or rhythm
  - Rate of primary pacemaker falls below AV junction or with SA or AV block
  - P waves may be absent
  - If present before, during, or after QRS
  - PRI <0.16 sec
  - Rate: 40-60/min
**Junctional Escape Complex or Rhythm**

- Decreased cardiac output
  - Usually rates below 50/min
- Stable
  - Observe
- Unstable
  - Treat as bradycardia

**Causes**
- Increased vagal tone
- Skewed SA discharge
- AV block

**Accelerated Junctional Rhythm**

- Increased AV junction automaticity
- Discharges faster than intrinsic rate
  - 40-60 bpm
- Overrides SA node
- Rate: 60-99 bpm
- Usually stable
- Observe

Accelerated Junctional Rhythm

Dysrhythmias Originating in the Ventricles

- Ventricular rhythms often life-threatening
- Failure of atria, AV junction
- Enhanced automaticity
- Reentry
- Associated with myocardial ischemia or infarction
- Least efficient pacemaker

Dysrhythmias Originating in the Ventricles

- QRS complexes >0.12 sec, bizarre appearance
- P waves hidden or superimposed on QRS
- ST deviated from baseline
Ventricular Escape Complexes or Rhythms

- Isolated impulse or rhythm
- Idioventricular rhythm
- Impulses from higher pacemakers
  - Fail
  - Don't reach ventricles
  - Rate of discharge of higher pacemakers is less than that of ventricles
- Compensatory mechanism
  - Prevents cardiac standstill

Ventricular Escape Complexes or Rhythms

- Hypotension
- Decreased cardiac output
- Perfusion to brain
- Syncope
- Shock
- Absent P waves
- Rate 20-40 bpm
- Wide QRS

Management
- Oxygen
- TCP
- Dopamine

Ventricular Escape Rhythm

“Dying Heart” or Agonal Rhythm

Premature Ventricular Complexes
- Single ectopic impulse
- From irritable focus in ventricle
- Earlier than expected sinus beat
- Common
- Occurs with any cardiac rhythm
- Enhanced automaticity or a reentry mechanism

PVCs
Compensatory Pause

- Measure interval between R wave before PVC and R wave after PVC
- If compensatory, distance equals twice R-R interval of underlying rhythm
- Interpolated PVC
  - PVC falls between two sinus beats
  - Doesn’t interrupt rhythm

Interpolated PVC

Unifocal and Multifocal PVCs

- Unifocal PVCs
  - Originate from single site within ventricles
  - Look alike
- Multifocal PVCs
  - Originate from different ventricular sites
  - Varying shapes and sizes
Unifocal and Multifocal PVCs

Fusion Beats
- PVCs occur at the same time as ventricular activation by underlying rhythm
  - Can cause ventricular depolarization simultaneously in two directions
- Fusion beat QRS complex has characteristics of PVC and QRS complex of underlying rhythm

Fusion Beat with PVC
Grouped Beating

- PVCs in patterns of grouped beating
  - Bigeminy
    - Every other complex is PVC
  - Trigeminy
    - Every 3rd complex is PVC
  - Quadrigeminy
    - Every 4th complex is PVC

Ventricular Bigeminy and Trigeminy

PVCs

- PVCs not separated by complex of underlying rhythm
  - Couplets
    - Two PVCs in a row
  - Run of VT
    - ≥3 sequential PVCs
    - Rate: ≥100 bpm
  - R-on-T phenomenon
**PVCs—Causes**

- Healthy individuals
  - Usually no significance

- Pathological PVCs
  - Myocardial ischemia
  - Hypoxia
  - Electrolyte imbalance
  - Congestive heart failure
  - Increased sympathetic tone
  - Stimulants
  - Drugs

**PVCs**

- QRS >0.12 sec

- P waves present or absent
  - No relationship to QRS

- Significant if:
  - Frequent
  - Multifocal
  - R-on-T
  - Grouped beats
PVCs

- Management, if clinically significant:
  - Oxygen
  - Antiarrhythmic drugs
  - Check potassium at hospital
    - Treat hypokalemia if present

Ventricular Tachycardia

- ≥3 consecutive ventricular complexes
- Rate: >100 bpm
- Overrides primary pacemaker
- Starts suddenly, triggered by a PVC
- Atria and ventricles are asynchronous
- If sustained, may lead to unconsciousness and loss of pulse

Monomorphic Ventricular Tachycardia

- Cardiac disease
- Electrolyte imbalances
- CHF
- Increased catecholamines
- Stimulants
- Drugs
- Long QT interval

- Stable but symptomatic:
  - Oxygen
  - Amdorepine
  - Procainamide
- Unstable
  - Cardioversion
  - Pulseless
    - Treat as ventricular fibrillation

Monomorphic VT

Torsades de Pointes

- Prolonged QT interval
  - May or may not be present
- Idiopathic
- Drug induced
- Polymorphic VT

Treatment varies:
- DC drugs that prolong QT
- Magnesium sulfate
- Lidocaine
- Isoproterenol
- Overdrive pacing

If Unstable:
- Uninhibited cardioversion

Torsades de Pointes
Determine axis deviation:
- Leads I, II, III, MCL₁ (V₁), MCL₂ (V₂)
- QRS complex negative in leads I, II, and III (extreme right axis deviation, or "no man's land") and positive in MCL₁ (V₁) indicates VT
  - If not, proceed to step 2

If extreme right axis deviation is not present, assess QRS deflection in MCL₁ (V₁) and MCL₂ (V₂):
- Negative QS complex
- Wide Q wave in MCL₂ (V₂) indicates VT
- Regardless of the QRS deflection in leads I, II, and III
12-Lead Strategies for Wide-Complex Tachycardias

- VT if positive QRS deflections with either:
  - Single peak
  - Taller left “rabbit ear”
  - RS complex with a fat R wave
  - Slurred S wave in MCL1 (V₁)

The presence of right axis deviation (negative QRS complex in lead I; positive QRS complex in leads II and III) and a negative QRS complex in MCL1 (V₁) indicates VT.

Note “Rabbit Ear”
Right Axis Deviation and a Downward MCL\textsubscript{1} Indicates VT

12-Lead Strategies for Wide-Complex Tachycardias

- VT if:
  - All precordial leads (V leads) are either positive or negative
  - Precordial concordance

VT-Concordance
12-Lead Strategies for Wide-Complex Tachycardias

- RS interval >0.10 sec in any V lead indicates VT
- Increased ventricular activation time

VT (RS interval is 0.16 sec)

Ventricular Fibrillation

- Chaotic ventricular rhythm
- Quivering ventricular movements
- No pulse
- Multiple reentry foci in ventricles
Ventricular Fibrillation—Causes

- Myocardial ischemia
- AMI
- Third-degree AV block
- Cardiomyopathy
- Digitalis toxicity
- Acidosis
- Electrolyte imbalance
- Electrical injury
- Drug toxicity
- Hypoxia

Ventricular Fibrillation—Treatment

- If unwitnessed:
  - CPR first then:
- Defibrillation
- Intubation
- Vascular access
- Epinephrine
  - May substitute vasopressin for first or second dose
- Amiodarone
  - Lidocaine as alternative
- Effective CPR

Coarse and Fine VF

Ventricular Asystole
- Absence of ventricular activity
- Confirm in two leads
- Primary event or follows other dysrhythmias
- No cardiac output
- Terminal rhythm

Ventricular Asystole—Treatment
- Effective CPR
- Intubation/IV
- Epinephrine
- Atropine
- Consider and treat other causes
  - Sodium bicarbonate possible

Ventricular Asystole

Artificial Pacemaker Rhythms

- Generate rhythm by electrical stimulation through electrode in the heart
  - Fixed rate or asynchronous
  - Demand pacemakers
  - Atrial synchronous ventricular pacemakers
  - AV sequential pacemakers
  - Rate-responsive pacemakers
Causes of Pacemaker Malfunction

- Battery failure
- Runaway pacemaker
- Failure of sensing device in demand pacemaker
- Failure to capture

Management of Pacemaker Failure

- True emergency
- Immediate recognition
- Rapid transport

Principles to follow:
- Look for battery packs under skin/medical ID tag
- Follow appropriate algorithm
- Manage ventricular irritability
- Defibrillate if needed; do not discharge energy directly over battery pack
- Use TCP if indicated

Heart Blocks

- Delays or complete interruptions in cardiac electrical conduction
- Occur in atria, between SA node and AV node, or in ventricles between AV node and Purkinje fibers
- Caused by:
  - Pathology in conduction system
  - Physiological block

Heart Blocks—Causes

- AV junctional ischemia
- AV junctional necrosis
- Degenerative disease of conduction system
- Electrolyte imbalances
- Drug toxicity
  - Often digitalis

Heart Blocks—Classification

- Classified by:
  - Site of block
    - (e.g., left bundle branch block)
  - Degree of block
    - (e.g., second-degree AV block)
  - Category of AV conduction disturbances
    - (e.g., Type I)

First-Degree AV Block

- Not true block
- Delay in conduction
  - Usually at AV node
- Superimposed on another rhythm
- Identify underlying rhythm
**First-Degree AV Block**
- PRI >0.20 sec
- Usually transient
- Often asymptomatic
- May progress to other block
- Observe

**Second-Degree AV Block Type I (Wenckebach)**
- Intermittent
- AV node
- Conduction delay increases from beat to beat until conduction to ventricle is blocked
  - PRI intervals get progressively longer until a P wave occurs that is not followed by QRS complex
- Pattern
- May be symptoms if rate is very slow
- Treat for bradycardia if symptomatic
Second-Degree AV Block Type I

- Atrial impulses are not conducted to ventricles
- P waves conducted with a constant PR interval before a dropped beat

Second-Degree AV Block Type II

- Intermittent block
- Atrial impulses are not conducted to ventricles
- P waves conducted with a constant PR interval before a dropped beat

- Usually a regular sequence with conduction ratios (P waves to QRS complexes)
  - 2:1, 3:2, and 4:3
Second-Degree AV Block Type II

- Below bundle of His
- Two consecutive impulses (atrial P waves) fail to be conducted to ventricles
- High-grade AV block

3:2 AV Block and 4:3 AV Block

3:1 High-Grade AV Block

Second-Degree AV Block Type II
- High-grade AV blocks have underlying atrial and ventricular rates
- Slow rates and hypoperfusion
- May advance to complete heart block
- Treat symptomatic patients with TCP

Third-Degree Heart Block
- Complete electrical block at or below the AV node
  - Infranodal
- SA node paces atria, and an ectopic focus paces ventricles
- P waves and QRS complexes occur rhythmically, but the rhythms are unrelated to each other
  - AV dissociation
Third-Degree AV Block

**Causes**
- Increased vagal tone
- Septal necrosis
- Myocarditis
- Drug toxicity
- Electrolyte imbalance

**Significance**
- Bradycardia may be severe
- Cardiac output decreased
- Wide complex is ominous sign

---

Third-Degree AV Block—Treatment

- Transvenous pacer is needed

- Initial prehospital care if symptomatic:
  - TCP
  - Dopamine
  - Epinephrine

---

Third-Degree AV Block

---

---
Ventricular Conduction Disturbances

- Bundle branch blocks or hemiblocks
- Delay electrical transmission below bundle of His

Bundle Branch Blocks and Hemiblocks

- Common causes of bundle branch block
  - Ischemic heart disease
  - Acute heart failure
  - Acute myocardial infarction
  - Hyperkalemia
  - Trauma
  - Cardiomyopathy
  - Aortic stenosis
  - Infection

Bundle Branch Anatomy

- Bundle of His divides:
  - Left and right bundle branches
  - Right bundle branch continues toward apex and spreads through right ventricle
  - Left bundle branch subdivides into anterior and posterior fascicles and spreads through left ventricle
- Electrical impulse conduction through Purkinje fibers stimulates ventricular contraction
Normal conduction
- Left side of septum is stimulated first
- Electrical impulse traverses septum to stimulate other side
- Left and right ventricles are then simultaneously stimulated

Normal Ventricular Activation
**Bundle Branch Block—ECG**

- One ventricle depolarizes and contracts before the other.
- Ventricular activation is not simultaneous, therefore QRS complex widens.
  - Slurred or notched appearance
    - "Rabbit ears"
- QRS complex is ≥0.12 sec.

---

**Bundle Branch Block**

- Criteria for bundle branch block
  - QRS complex ≥0.12 sec
  - QRS complexes produced by supraventricular activity.

---

**Bundle Branch Block**

- Leads V₁ and V₆
  - MCL₁ and MCL₆
  - Permit differentiation of right and left bundle branch blocks.
- Normal conduction
  - V₁ (MCL₁) is predominantly negative
  - QRS complex is 0.08-0.10 sec.
Right Bundle Branch Block

- Left bundle branch performs normally
- Activates left side of heart before right
- ECG characteristics
  - Initial negative deflection (S wave)
  - RSR-prime pattern
  - QRS (or in this case, RSR) duration ≥ 0.12 sec

Left Bundle Branch Block

- Fibers that fire interventricular septum are blocked:
  - Alters normal septal activation
  - Sends it in opposite direction
- ECG characteristics
  - Initial Q wave in V1 (MCL1)
  - R wave in V1 (MCL1)
  - Deep, wide S wave (QS pattern)
  - QRS duration ≥ 0.12 sec
Left Bundle Branch Block

Find J point
Draw line back into QRS complex
Fill in triangle created
Note direction triangle points

Left vs. Right BBB

More common than posterior hemiblock
Anterior fascicle of left bundle branch is a longer and thinner structure
Blood supply primarily from left anterior descending (LAD) coronary artery
Anterior hemiblock characterized by left axis deviation in patient with supraventricular rhythm

Anterior Hemiblock

Anterior Hemiblock

- Other ECG findings in anterior hemiblock:
  - Normal QRS complex (<0.12 sec) or a right bundle branch block
  - Small Q wave followed by tall R wave in lead I
  - Small R wave followed by deep S wave in lead III

- High risk to develop complete heart block

Posterior Hemiblock

- Right axis deviation with normal QRS complex or right bundle branch block

- Other ECG findings
  - Small R wave followed by deep S wave in lead I
  - Small Q wave followed by tall R wave in lead III
**Posterior Hemiblock**

Showing 2 of 3 Fascicles Blocked

---

**Bifascicular Block**

- 2 of 3 pathways for ventricular conduction blocked
  - Right bundle branch block with anterior or posterior hemiblock
  - Left bundle branch block

- Compromises myocardial contractility and cardiac output

- May develop complete heart block suddenly

---

**Multilead Determination of Axis and Hemiblocks**

- Identifying axis can be useful in determining the presence of hemiblocks

- Best evaluated by looking at the QRS complexes in leads I, II, and III
Multilead Determination of Axis and Hemiblocks

- Axis is:
  - Normal if QRS deflection is positive in bipolar leads
  - Physiological left (normal in some patients) when QRS deflection is:
    - Positive in leads I and II
    - Negative (inverted) in lead III
  - Pathological left when QRS deflection is:
    - Positive in lead I
    - Negative in leads II and III (indicating an anterior hemiblock)

- Right axis when QRS deflection is:
  - Negative in lead I, negative or positive in lead II
  - Positive in lead III (pathological in any adult)
  - Indicative of posterior hemiblock
- Extreme right ("No man’s land") when QRS deflection is negative in all three leads
  - Rhythm is ventricular in origin

Pulseless Electrical Activity

- Absence of detectable pulse and presence of rhythm other than VT or VF
- Prognosis is poor unless underlying cause is identified and corrected
- Priority of care is to maintain circulation with basic and advanced life support
- Search for correctable cause
Pulseless Electrical Activity

Correctable causes
- Cardiac tamponade
- Tension pneumothorax
- Hypoxemia
- Acidosis
- Hyperkalemia
- Hypothermia
- Drug overdoses

Less correctable causes
- Massive myocardial damage
- Prolonged ischemia
- Profound hypovolemia
- Massive pulmonary embolism
- Profound shock

Management
- Effective CPR
- ALS
- Epinephrine
- Atropine if HR < 60 bpm
- Identify and correct specific causes

Various PEA Rhythms as Seen in Lead II
Preexcitation Syndromes

- Clinical condition with abnormal conduction pathway between atria and ventricles
  - Bypasses AV node and/or bundle of His
  - Allows electrical impulses to depolarize ventricles earlier than usual
  - Several accessory pathways

- Most common preexcitation syndrome is Wolff-Parkinson-White (WPW) syndrome

Wolff-Parkinson-White Syndrome

- Bundle of Kent connects lateral wall of atrium and ventricle
  - Bypasses AV node

- Life-threatening if tachycardia develops

- Rate: Normal unless associated with rapid supraventricular tachycardia

- PR interval <0.12 sec
  - Normal delay at AV node does not occur

Wolff-Parkinson-White Syndrome

- ECG findings
  - Short PR interval
  - Delta wave
  - QRS widening

- Susceptible to PSVTs

- Amiodarone, procainamide

- May be harmful:
  - Adenosine, calcium channel blockers
  - Beta blockers, digoxin
WPW

(A) Appearance of WPW syndrome in leads where QRS complex is upright.

(B) Appearance of WPW syndrome with QRS complex predominantly negative.

---

Acute Coronary Syndromes

- Acute myocardial infarction (AMI)
- Unstable angina (UA)

Treatment goals
- Reduce myocardial necrosis
- Prevent major adverse cardiac events
- Treat acute complications of ACS

---

Atherosclerosis

- Progressive narrowing of lumen of medium and large arteries
  - Aorta and its branches, cerebral arteries, coronary arteries

- Development of thick, hard, atherosclerotic plaques called atheromas or atheromatous lesions
  - Commonly found in areas of turbulent blood flow

---
Atherosclerosis—Risk Factors

- Age
- Earlier in men than women
- Family history
- Diabetes
- Smoking
- Hypertension
- Hypercholesterolemia

Atherosclerosis—Effects

- Disrupts intimal surface, causing loss of vessel elasticity and increase in thrombogenesis
  - Atheroma reduces diameter of vessel lumen
  - Decreases blood supply to tissues

Angina Pectoris

- Symptom of myocardial ischemia
- "Choking" pain in the chest
- Imbalance between myocardial oxygen supply and demand
- Accumulation of lactic acid and carbon dioxide in ischemic tissues of myocardium
  - Metabolites irritate nerve endings and produce pain
**Angina Pectoris**

- **Causes**
  - Atherosclerotic disease of the coronary arteries
  - Temporary occlusion due to coronary artery spasm with or without atherosclerosis
    - Prinzmetal's angina

- **Pain described as pressure, squeezing, heaviness, or tightness in chest**
  - 30% feel pain only in chest
  - Others describe as radiating to shoulders, arms, neck, and jaw and through to back

- **Associated signs and symptoms**
  - Anxiety
  - Shortness of breath
  - Nausea or vomiting
  - Diaphoresis

**Stable Angina**

- Usually caused by physical exertion or emotional stress
- Pain lasts 1-5 min
  - May last as long as 15 min
- Relieved by rest, nitroglycerin, or oxygen
- “Attacks” are usually similar in nature
- Always relieved by same therapy
Unstable Angina

- Preinfarction angina
- Anginal pattern that has changed in its ease of onset, frequency, intensity, duration, or quality
- Includes “new onset” anginal chest pain
- May occur during exercise or at rest
- Pain lasts >10 min
- Less promptly relieved than stable angina

Unstable Angina—Management

- Place patient at rest
- Administer oxygen
- Administer aspirin (per protocol)
- IV therapy
- Pharmacological therapy
  - Nitroglycerin
  - Morphine
- Monitor ECG
- Transport as soon as possible

Myocardial Infarction

- Sudden and total occlusion or near-occlusion of blood flowing through affected coronary artery
- Ischemia, injury, and necrosis of myocardium distal to occlusion
- Often associated with atherosclerotic heart disease (ASHD)
- Precipitating events
Types and Locations of Infarcts

- Infarction distal to occluded artery

- Size of infarct determined by:
  - Metabolic needs of tissue supplied by occluded vessel
  - Collateral circulation
  - Time until flow is reestablished

Types and Locations of Infarcts

- Emergency care
  - Increasing oxygen supply
  - Decreasing metabolic needs
  - Providing collateral circulation
  - Reestablishing perfusion to ischemic myocardium quickly

Types and Locations of Infarcts

- Most AMIs involve ventricle or interventricular septum, which is supplied by either of two major coronary arteries
  - Some patients sustain damage to right ventricle

Types and Locations of Infarcts

- Anterior, lateral, or septal wall infarction
  - Usually left coronary artery occlusion
- Inferior wall infarction
  - Usually right coronary artery occlusion

Myocardial Infarction

- Three ischemic syndromes
- Based on rupture of an unstable plaque in an epicardial artery
  - Unstable angina
  - Non-ST-elevation myocardial infarction
  - ST-elevation myocardial infarction

Infarction

- Unstable angina
  - Thrombus has not completely obstructed coronary flow
  - Intermittent ischemic episode
  - May lead to complete occlusion and AMI
- Non-ST-elevation MI
  - ST-segment depression
  - T-wave abnormalities
**Infarction**
- ST-elevation MI
  - Q-wave MI
  - Pathological Q waves
    - > 5 mm in depth
    - > 0.04 sec in duration in 
      ≥ 2 contiguous leads

**Death of Myocardium**
- After blood flow to myocardium stops, cells switch to anaerobic metabolism
  - Produces ischemic pain (angina)
- Cells begin to swell and depolarize
- If collateral flow and reperfusion are inadequate, much of muscle dies distal to occlusion

**Area of Infarction**
Myocardial Infarction—Deaths

- Lethal dysrhythmias
  - VT
  - VF
  - Cardiac standstill
- Pump failure
  - Cardiogenic shock
  - CHF
- Myocardial tissue rupture
  - Ventricle, septum, or papillary muscle

MI—Signs and Symptoms

- Pain is similar to angina
- May radiate to arms, neck, jaw, or back
- Dyspnea
- Anxiety
- Agitation
- Sense of impending doom
- Nausea and vomiting
- Diaphoresis
- Cyanosis
- Palpitations

MI—Signs and Symptoms

- Chest pain often constant
- Not altered by nitroglycerin or medications, rest, changes in body position, or breathing patterns
  - Onset of pain at rest in >50% of MI patients
  - Most have experienced warning anginal pain (preinfarction angina) hours or days before
Myocardial Infarction—ECG Findings

- Heart muscle unable to contract effectively
- Remains in depolarized state
  - Current flow between pathologically depolarized and normally repolarized areas can produce:
    - Abnormal ST segment elevation
    - Ischemic ST segment depression
    - Normal or non-diagnostic ECG changes

Myocardial Infarction—ECG Findings

- ST-Segment Elevation MI (STEMI)
  - ST segment elevation >1 mm in 2 adjacent leads
  - new LBBB
- High-Risk UA/non-ST-Elevation MI (NSTEMI)
  - ST segment depression >0.5 mm lasting 20 min.
  - T-wave inversion with pain
- Normal or nondiagnostic ECG changes
  - Inconclusive changes

Myocardial Infarction ECG Imposters

- Left bundle branch block
- Some ventricular rhythms
- Left ventricular hypertrophy
- Pericarditis
- Ventricular aneurysm
- Early repolarization
Myocardial Infarction—Management

- Oxygen
- Aspirin
- Nitroglycerin
- Morphine
- 12-lead ECG
- Fibrinolytic screening
- Transport to appropriate facility

---

ST Segment Elevation Likely with Acute Injury

---

ST-Elevation and Infarct Location

<table>
<thead>
<tr>
<th>Lead</th>
<th>Location of Infarction</th>
<th>Coronary Artery Involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>II, III, aVF</td>
<td>Interior wall (most common)</td>
<td>Right</td>
</tr>
<tr>
<td>V1, V2</td>
<td>Septal wall</td>
<td>Left</td>
</tr>
<tr>
<td>V3, V4</td>
<td>Anterior wall (most lethal)</td>
<td>Left</td>
</tr>
<tr>
<td>I, aVL, V5, V6</td>
<td>Lateral wall</td>
<td>Left</td>
</tr>
<tr>
<td>V4R, V5R, V6R</td>
<td>Right ventricle</td>
<td>Right</td>
</tr>
</tbody>
</table>

---

Multilead Assessment of the Heart

Left Ventricular Failure (LVF) and Left Ventricular Failure (LVF) and Pulmonary Edema

- Left ventricle fails to function as an effective forward pump
- Causes back-pressure of blood into pulmonary circulation

LVF and Pulmonary Edema

- Caused by heart disease, including:
  - Ischemic
  - Valvular
  - Hypertensive heart disease
- Untreated LVF leads to pulmonary edema
LVF—Signs and Symptoms

- Respiratory distress
- Apprehension, agitation, confusion
- Cyanosis (if severe)
- Diaphoresis
- Adventitious lung sounds
- JVD
- Abnormal vital signs

Pulmonary Edema—Management

- Oxygen, IV, monitor
- 12-lead ECG
- Nitroglycerin (SBP >100)
- Furosemide
- Morphine
- CPAP
- Reversible causes
- Dobutamine or dopamine for shock

Right Ventricular Failure (RVF)

- Right ventricle fails as effective forward pump
- Back-pressure of blood into systemic venous circulation
**RVF**

- **Causes**
  - Chronic hypertension (LVF precedes RVF)
  - COPD
  - Pulmonary embolism
  - Valvular heart disease
  - Right ventricular infarction

- RVF usually results from LVF

---

**RVF**

- **Signs and symptoms**
  - Tachycardia
  - Venous congestion
    - Engorged liver, spleen, or both
    - Venous distention
    - Peripheral edema
  - Fluid accumulation in serous cavities

- **Management**

---

**Cardiogenic Shock**

- Most extreme form of pump failure

- Left ventricular function is so compromised
  - Heart cannot meet metabolic needs of body

- Extensive myocardial infarction
  - 40% of left ventricle
  - Diffuse ischemia
Cardiac Tamponade

- Impaired diastolic filling of heart
- Increased fluid in pericardial space
- Volume of pericardial fluid encroaches on capacity of atria and ventricles to fill adequately
- Ventricular filling is mechanically limited, and stroke volume is decreased

Cardiac Tamponade

- Acute onset
  - Trauma
- Gradual onset
  - Neoplasm
  - Infectious
  - Renal disease
  - Hypothyroidism
- Management
  - Fluid
  - Pericardiocentesis

Presentation

- Chest pain
- Tachycardia
- Ectopy
- JVD
- Decreased SBP
- Pulsus paradoxus
- Muffled heart sounds
- ECG changes

Thoracic and Abdominal Aortic Aneurysms

- Aneurysm
  - Dilatation of a vessel

- Causes
  - Atherosclerotic disease (most common)
  - Infectious disease (primarily syphilis)
  - Traumatic injury
  - Certain genetic disorders (e.g., Marfan's syndrome)
Aortic Aneurysms—Signs and Symptoms

- Hypotension
- Syncope
- Abdominal or back pain
  - Tearing or ripping
- Low back or flank pain
  - Radiates to thigh, groin, testicle
- Peritoneal irritation
- Urge to defecate
- Pulsatile, tender mass
- Distal pulses present or absent
- GI bleeding

Pathogenesis of Dissecting Aneurysms

- Medial and intimal degeneration in aortic wall
- Hemodynamic forces produce tear
- Dissecting hematoma propagated by pulse wave
Dissecting Aneurysms—Management

- Gentle handling
- Oxygen
- Monitor
- IV fluids
  - Bolus if profound shock

Acute Arterial Occlusion

- Blockage of arterial flow caused by:
  - Trauma
  - Embolus
  - Thrombosis

- Severity of episode depends on:
  - Site of occlusion
  - Collateral circulation

Acute Arterial Occlusion

- Signs and symptoms
  - Pain in extremity
    - May be severe and sudden in onset or absent because of paresthesia
  - Pallor
  - Cool skin distal to occlusion
  - Change in sensory and motor function
  - Diminished or absent pulse distal to injury
  - Bruit over affected vessel
  - Slow capillary filling
  - Sometimes shock
**Arterial Occlusion—Management**

- Extremity occlusion is painful and limb threatening if blood flow is not reestablished within 4-8 hrs
- Immobilize limb and transport
- Patients with mesenteric occlusion
  - Manage for shock:
    - Oxygen
    - IV fluids
  - Analgesics for pain control

---

**Common Sites of Embolic Arterial Occlusion**

---

**Venous Thrombosis**

- Predisposing factors
  - History of trauma
  - Sepsis
  - Stasis or inactivity
  - Recent immobilization
  - Pregnancy
  - Birth control pills
  - Malignancy
  - Coagulopathies
  - Smoking
  - Varicose veins
Acute Deep Vein Thrombosis (DVT)

- Occlusion of deep veins is serious, common problem
- May involve any portion of deep venous system
  - More common in lower extremities

Acute DVT—Risk Factors

- Lower extremity trauma
- Recent surgery
- Advanced age
- Recent MI
- Inactivity
- Previous thrombosis
- Oral contraceptives
- Cancer
- Obesity
- CHF

Acute DVT—Management

- Risk of pulmonary embolus
- Hospitalization
- Bed rest
- Anticoagulants
Resting BP: Consistently >140/90 mm Hg

Several categories of hypertension based on level of blood pressure, symptoms, and urgency of need for intervention

- Cerebral hemorrhage and stroke
- Myocardial infarction
- Renal failure (secondary to vascular changes in the kidney)
- Thoracic and/or abdominal aortic aneurysm
Hypertensive Emergencies

- Blood pressure increase leads to significant, irreversible end-organ damage within hours if not treated
- Organs most at risk are brain, heart, and kidneys

Hypertensive Emergencies

- Myocardial ischemia with hypertension
- Aortic dissection with hypertension
- Pulmonary edema with hypertension
- Hypertensive intracranial hemorrhage
- Toxemia
- Hypertensive encephalopathy

Hypertensive Emergencies—Signs and Symptoms

- Paroxysmal nocturnal dyspnea
- Shortness of breath
- Altered mental status
- Vertigo
- Headache
- Epistaxis
- Tinnitus
- Changes in visual acuity
- Nausea and vomiting
- Seizures
- ECG changes
Hypertensive Encephalopathy

- Severe hypertension produces hypertensive encephalopathy and cerebral hypoperfusion
- Loss of integrity of blood-brain barrier
- Fluid exudation into brain tissue

Hypertensive Encephalopathy

- Progresses from:
  - Severe headache, nausea, vomiting, aphasia, hemiparesis, and transient blindness
- Later
  - Seizures, stupor, coma, and death

Hypertensive Emergencies

- Supportive care
- Oxygen
- IV
- ECG monitoring
- Rapid transport
- Drugs under medical supervision
Techniques for Managing Cardiac Emergencies

- Basic life support
- Mechanical CPR devices
- Monitor-defibrillators
- Implantable cardioverter-defibrillators (ICDs)
- Transcutaneous cardiac pacing (TCP)
- Advanced cardiac life support (ACLS) system

Basic Life Support

- CPR within 4 min
- Compression/ventilation ratio 30:2
- Compressions must be at least 100/min
- Depth of 1-1/2 to 2 inches
- Allow complete chest recoil
- Minimize interruptions of CPR
- Externally supports circulation and respiration

Mechanical CPR Devices

- Designed to:
  - Standardize CPR technique
  - Eliminate rescuer fatigue
  - Free rescuers to perform ACLS procedures
  - Provide adequate compression during patient transport
Impedence Threshold Devices

- Attach to ET or mask
- During Pulseless Arrest:
  - Increases blood flow to heart and brain
  - Doubles systolic pressure
  - Increases survival to hospital
  - Increases defibrillation success
  - AHA Class IIa in intubated patients

Automated External Defibrillators

- Analyze ECG signal
  - Including frequency, amplitude, and wave morphology
- Designed for use by individuals with minimal training

Defibrillation

- Delivery of electrical current through chest wall to terminate VF and pulseless VT
  - Shock depolarizes a large mass of myocardial cells at once
  - If 75% of cells are in resting state (depolarized) after shock is delivered, normal pacemaker may resume discharging
Defibrillation

- Early defibrillation:
  - VF is most frequent initial rhythm in arrest
  - Treatment for VF is electrical defibrillation
  - Chance of successful defibrillation diminishes rapidly over time
  - VF tends to convert to asystole within a few minutes
  - If prehospital arrest is unwitnessed, 2 minutes of CPR may enhance ability to defibrillate

Paddle Electrodes

- Apex or sternum
- Place so that the heart is in path of current and distance between electrodes and the heart is minimized

Stored and Delivered Energy

- Electrical energy measured in joules
  - Watt seconds
- Delivered energy about 80% of stored energy
  - Losses within defibrillator circuitry
  - Resistance to current flow across chest wall
- 80% of stored energy approximates amount of joules delivered to patient
Monitor Defibrillator Pacer

Defibrillator Safety

- Clear all personnel from patient, bed, and defibrillator before shock delivery
- Do not make contact with patient except through defibrillator paddle handles
- Do not use excessive gel or coupling material
- Do not discharge paddles over a pacemaker or ICD generator or nitroglycerin paste
- Remove nitroglycerin patches before defibrillation
**Defibrillator Safety**

- Apply gel or paste before turning on defibrillator
- Do not discharge defibrillator in open air to rid unwanted charge
- Turn defibrillator off to dump charge
- Do not discharge defibrillator with paddles placed together


---

**Defibrillator Safety**

- Do not touch metal electrodes or hold paddles to your body when defibrillator is on
- Clean paddles after use
- Routinely check defibrillator to make sure equipment is functioning properly
  - Including batteries


---

**Defibrillator Use in Special Environments**

- Can defibrillate in wet conditions
  - Keep chest dry between defibrillator electrode sites
  - Keep operator's hands and paddle handles as dry as possible
    - In a rainstorm, finding shelter would be safest

Defibrillator Use in Special Environments

Video Clip: Defibrillation

Implantable Cardioverter-Defibrillator (ICD)

- Implanted through median sternotomy incision
  - Other approaches also used
- ICD monitors patient’s cardiac rhythm, rate, and QRS morphology
**Synchronized Cardioversion**

- Terminates dysrhythmias other than VF and pulseless VT
- Delivers shock about 10 msec after peak of QRS complex
  - Avoid relative refractory period
  - May reduce energy needed to end dysrhythmia
  - Decreases potential for development of secondary complicating dysrhythmias

**Procedure**

---

**Transcutaneous Cardiac Pacing (TCP)**

- External cardiac pacing
- Treatment for bradycardia
- Indications
- Contraindications
TCP—Proper Electrode Placement

Preferred anterior-posterior placement

Alternate anterior-anterior placement

Video Clip: Transcutaneous Pacing

Cardiac Arrest and Sudden Death

- Resuscitation in prehospital setting best chance for survival
- Rapid ACLS protocol initiated without delaying transport to hospital
Termination of Resuscitation

- Inclusion criteria
- Exclusion criteria
- Procedure
- Special considerations

Conclusion

About two thirds of cases of sudden death due to coronary disease take place outside the hospital. This usually occurs within 4 hours after onset of symptoms. It is possible that a large number of these deaths could be prevented by rapid entry into the EMS system, effective early CPR, and early defibrillation.

Questions?