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Acute Coronary Syndrome

Ghana Emergency Medicine Collaborative
Kristen Sarna, RN, BSN
Acute Coronary Syndrome (ACS)

- Primarily caused by atherosclerosis (the build up of plaque that impedes blood flow) often called “hardening of the arteries”
- Disruption of a previously nonsevere lesion
Acute Coronary Syndrome

- Angina
- Unstable Angina
- Prinzmetal’s or variant
- NSTEMI (non ST elevated myocardial infarction)
- STEMI (ST elevated myocardial infarction)
## Risk Factors for ACS

<table>
<thead>
<tr>
<th>Modifiable</th>
<th>Non-modifiable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum lipid levels</td>
<td>Age</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Gender</td>
</tr>
<tr>
<td>Smoking/tobacco use</td>
<td>Ethnicity</td>
</tr>
<tr>
<td>Sedentary lifestyle</td>
<td>Family history</td>
</tr>
<tr>
<td>Obesity</td>
<td>Genetics</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Menopause</td>
</tr>
<tr>
<td>Diet</td>
<td></td>
</tr>
</tbody>
</table>
Angina

- Chest pressure or heaviness that is reproduced by activities or conditions that increase myocardial oxygen demand
- May be described as epigastric pain, indigestion, or anxiety
- Can also have neck pain, arm pain, shortness of breath, weakness, nausea/vomiting, light-headedness, and diaphoresis
Angina
Signs and Symptoms

- Palpitations
- Chest pain
  - Describes as:
    - Heaviness
    - Burning
    - Achy
    - Squeezing
- Exertional dyspnea
- Diaphoresis
- Nausea
Stable vs Unstable

- **Stable Angina:**
  - Episodic pain lasting 5-15 minutes provoked by exertion and relieved by rest and nitroglycerin
  - Usually relieved by nitro and rest

- **Unstable Angina:**
  - Increased pain that is easily induced
  - Increased risk for adverse cardiac events (NSTEMI, STEMI)
  - Not resolved by nitroglycerin administration
  - May have T wave abnormality
Prinzmetal’s or Variant Angina

- Often occurs at rest, usually in response to spasm of a major artery
- Frequently seen in pts with migraine headaches or Reynaud's phenomenon
- May experience angina and transient ST segment elevation
- Treated with calcium channel blockers and/or nitrates
STEMI

- ST-elevation-myocardial infarction
- Sustained ischemia – irreversible cell death
- Usually results from a blockage in the Left anterior descending coronary artery
STEMI Signs and Symptoms

- Chest pain – 20% of the population have no pain
- Jaw, neck and/or arm pain/pressure
- Changes in BP
- Tachycardia or bradycardia
- Palpitations
- Diaphoresis
- Syncope
- Nausea/vomiting
NSTEMI Diagnosis

- ST elevation on EKG
- Location of elevation determines where MI is occurring in the heart
EKG changes

- ST Elevation in leads V1 and V2 is a septal wall MI
  - Caused by blockage in right coronary artery
- ST elevation in leads V3 and V4 is an Anterior wall MI
  - Caused by blockage in left ascending artery
- ST elevation in leads V5, V6, I and aVL is a lateral wall MI
  - Caused by blockage in the left circumflex artery
- ST elevation in leads II, III, and aVF is an inferior wall MI
  - Caused by blockage in the right coronary artery
- Posterior wall MI – get posterior EKG to show leads V7, V8, V9
  - Caused by blockage in the right coronary artery
STEMI Treatment and Management

- **ABC’s**
- **Continuous cardiac monitoring**
- **MONA**
  - Morphine
  - Oxygen
  - Nitroglycerin
  - Aspirin
Treatment and Management con’t

- Heparin or LMWH (low molecular weight heparin)
- Beta blockers
- Antiplatelets
- ACE inhibitors
- Fibrinolytics
- Cath lab
NSTEMI

- Non-ST-elevation-myocardial-infarction
- May have normal EKG
- Diagnosed by lab values
  - Elevated troponin
  - Elevated myoglobin
  - Elevated CK and CK-MB
Right Ventricular Infarction

- Occurs due to Right coronary artery occlusion
- Right ventricular failure and elevated right ventricular filling pressures despite relatively normal left ventricular filling pressures resulting in decreased cardiac output
- Less likely to infarct vs left side due to low pressure and oxygen demand
- Higher mortality rate
Right Ventricular Infarction
Signs and Symptom

- Hypotension
- Hypoxia – due to right to left shunting
- Distended neck veins
- Bradycardia requiring pacing support
- May auscultate 3rd and 4th heart sounds
- Clear lung sounds
Diagnosis

- Chest x-ray
- Echocardiogram
- EKG – serial 12 lead EKG’s may be needed, may be normal or inconclusive during first few hours after an MI.
  - Abnormalities include:
    - Non Q wave MI
    - ST segment elevation
    - Q Waves (represents scarring and necrosis)
Diagnosis

- **Coronary angiography**
  - Reveals coronary artery stenosis or obstruction
  - Shows the condition of the arteries beyond the narrowing
- **Stress Testing**
- **Serial Laboratory studies**
  - Troponins
  - Creatine kinase (CK) especially the CK-MB, specific to the cardiac muscle
  - Lipid profile
Treatment

- Avoid nitroglycerin
- IV fluid
  - Avoid dopamine and phyenlephrine
  - Oxygen
  - Rest
  - Thrombolytic therapy
  - Aspirin
Treatment

- **Positive Inotropes**
  - Dobutamine
  - Milrinone
  - Norepinephrine
  - Low dose vasopressin
Treatment

- Pacing may be required to keep heart rate at a level to perfuse the rest of the body’s organs
- Heart catheterization
  - Coronary artery bypass graft (CABG) may be needed if obstructive lesions are found
On going assessment

- ABC’s- airway, breathing, circulation
- Vital signs
- Cardiac monitoring- rhythm analysis
- Laboratory studies
- Administer and titrate medications as ordered
Heart Failure

- Impaired cardiac pumping (systolic) or impaired cardiac filling (diastole)
- Pathophysiologic changes of vasoconstriction and fluid retention
Pathology of Ventricular Failure

- Systolic failure (impaired pumping)
- Diastolic failure (impaired filling)
Systolic Failure

- Most common
- Inability of the ventricles to pump (contract)
- The left ventricle (LV) loses the ability to generate enough pressure to eject blood forward through the aorta, resulting in decreased EF (ejection fraction)
- LV becomes thin-walled, dilated and hypertrophied
Diastolic failure

- Impaired ability of the ventricles to relax and fill during diastole
- Decreased filling results in decreased stroke volume and cardiac output
- High filling pressures due to stiff or noncompliant ventricles and results in venous engorgement in both the pulmonary and systemic vascular systems
Two types of heart failure

- Left-sided heart failure
- Right-sided heart failure
Left-Sided Heart failure

- Most common type
- Caused by left ventricular dysfunction which prevents normal blood flow and causes blood to back up into the left atrium and into the pulmonary veins
- Increased pulmonary pressures results in fluid extravasation from the pulmonary capillary bed into the interstitium and then the alveoli—which causes pulmonary congestion and edema
Signs and Symptoms of Left sided heart failure

- Weakness
- Fatigue
- Dyspnea
- Shallow respirations
- Dry, hacking cough
- Frothy, pink tinged sputum
Patient assessment

- Tachycardia
- Crackles in the lungs
- S3 and S4 heart sounds
- Pleural effusion
- Change in mental status
- Restlessness/confusion
Right-Sided Heart Failure

- Causes back up of blood into the right atrium and venous circulation.
- Usually caused by left-sided failure:
  - Increased pressure in the blood vessels of the lungs (pulmonary hypertension)
Signs and symptoms of right sided heart failure

- Fatigue
- Anxiety
- Depended bilateral edema
- GI bloating
- Nausea
- Weight gain
Patient assessment

- Murmurs
- Jugular vein distention
- Edema
- Tachycardia
- Ascites
- Generalized peripheral edema
- Hepatomegaly (liver enlargement)
Assessment of the HF patient

- Airway
- Breathing
- Circulation
- Vital signs including pulse oximetry
- EKG monitoring
- Assess for distended neck vein and peripheral edema
Diagnosis

- Past medical history
- Physical assessment
- B-type natriuretic peptide (BNP) – hormone secreted in response to ventricular wall stretch
- Chest x-ray
- Echocardiogram to measure ejection fraction
Interventions

- Maintain high-fowler’s position
- Apply oxygen
- Obtain IV access
- ACE inhibitors to increase cardiac output
- Strict monitoring of I’s and O’s
- Diuretics
- Monitor labs for hyponatremia and hypokalemia
Treatment

- Vasodilators
  - ACE inhibitors
  - Nitrates
- Diuretics
- Positive inotropes
Patient education

- Diet education
  - Low sodium diet
- Fluid restriction
- Weight management
  - Weight self daily
On going assessment

- ABC’s
- Vital signs
- ECG monitoring for arrhythmias
- Urinary output
Pulmonary Edema

- An acute life-threatening event in which the lung alveoli become filled with serosanguinuous fluid
- Most common cause: left sided HF
Cardiogenic Pulmonary Edema

- Inadequate left ventricular pumping, causing increased fluid pressure, which leads to decreased atrial emptying, causing back up of fluid into the pulmonary circulation
- Fluid fills the alveolar space normally occupied by air
- Caused by heart failure or acute coronary syndromes
Signs/Symptoms of Pulmonary Edema

- Severe dyspnea
- Diaphoresis
- Hypertension
- Tachycardia
- Anxiety
- Tachypnea
- Pink, frothy sputum production
Treatment of Pulmonary Edema

- Airway management - intubation may be necessary
- Oxygenation
- Bronchodilators
- Medication therapy to increase contractility of heart.
- Diuretics
- nitroglycerin
On going assessment

- ABC’s
- Vital signs
- Oxygenation
- EKG
- Mental status
Cardiomyopathy

- A group of diseases that directly affects the structural or functional ability of the myocardium

- Three types
  - Dilated
  - Hypertrophic
  - Restrictive
Dilated Cardiomyopathy

- Most common type
- Diffuse inflammation and rapid degeneration of myocardial fibers.
- Results in:
  - ventricular dilation
  - Impairment of systolic function
  - Atrial enlargement
  - Stasis of blood in the left ventricle
Dilated Cardiomyopathy

- Causes:
  - Genetic
  - Hypertension
  - Ischemia
  - Myocarditis
  - Muscular dystrophy
  - Pregnancy
  - Valve disease
  - Cardiotoxic agents
    - Alcohol
    - Cocaine
Signs and symptoms of dilated cardiomyopathy - early signs

- Decreased exercise capacity
- Fatigue
- Dyspnea at rest
- Paroxysmal nocturnal dyspnea
- Orthopnea
Dilated Cardiomyopathy as the disease process advances

- Dry cough
- Palpitations
- Abdominal bloating
- Nausea
- Vomiting
- Anorexia
- Irregular heart beat

- Bradycardia or tachycardia
- Pulmonary crackles
- Edema
- Pallor
- Weak pulses
- JVD
Diagnosis of Dilated Cardiomyopathy

- History and physical exam
- EKG
- Echocardiogram
- Chest xray
- Cardiac catheterization
Treatment/Management of dilated cardiomyopathy

- Similar to heart failure
- Cardiac rehabilitation to reduce symptoms and improve cardiac output
- Usually does not respond well to drug therapy
- LVAD (left ventricular assist device)
- Place AICD/pacemaker
- Heart transplant
Hypertrophic Cardiomyopathy

- Asymmetric left ventricular hypertrophy without ventricular dilation
- The septum between the two ventricles becomes enlarged and obstructs the blood flow from the left ventricle
- Impaired ventricular filling as the ventricle becomes noncompliant and unable to relax
Signs and symptoms of hypertrophic cardiomyopathy

- May be asymptomatic
- Dyspnea
- Fatigue
- Angina
- Syncope
Diagnosis of hypertrophic cardiomyopathy

- Clinical findings may be unremarkable
- Chest palpation
- Auscultation of heart sounds, S4 and murmurs
- EKG
Treatment and Management

- Focused on relieving symptoms and preventing complications
- Provide emotional and psychological support
- Patient education
Treatment and Management

- Beta blockers
- Calcium channel blockers
- Antidysrhythmics if needed
- pacemaker
Restrictive Cardiomyopathy

- Disease of the heart that impairs diastolic filling and stretch
- Etiology unknown: may be caused by:
- Ventricle are resistant to filling and therefore demand high diastolic filling pressures to maintain cardiac output
Signs and symptoms of restrictive cardiomyopathy

- Fatigue
- Exercise intolerance
- Dyspnea
- Angina
- Orthopnea
- Syncope
- Palpitations
- Signs of HF
  - Peripheral edema
  - JVD
  - Ascities
Diagnosis

- Chest xray
- EKG
- Echocardiogram
- CT scan
Treatment and management

- Currently no specific treatment
- Treat symptoms
- Treatment aimed at improving diastolic filling
- Heart transplant
- Patient education
Myocarditis

- Inflammation of the myocardium
Myocarditis

- **Caused by**
  - Virus
  - Bacteria
  - Fungi
  - Radiation therapy
  - Pharmacologic factors
  - Chemical factors
  - Idiopathic
Signs and Symptoms

- Sometimes no symptoms at all
- Can be fatal
- Early signs appear 7 to 10 days post viral infection
<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Clinical manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Fever</td>
<td>• Pleuritic chest pain</td>
</tr>
<tr>
<td>• Fatigue</td>
<td>• Pericardial friction rub</td>
</tr>
<tr>
<td>• Malaise</td>
<td>• Signs of HF</td>
</tr>
<tr>
<td>• Myalgias</td>
<td>◦ S3 heart sound</td>
</tr>
<tr>
<td>• Pharyngitis</td>
<td>◦ Crackles</td>
</tr>
<tr>
<td>• Dyspnea</td>
<td>◦ JVD</td>
</tr>
<tr>
<td>• Nausea/vomiting</td>
<td>◦ Syncope</td>
</tr>
<tr>
<td>• Lymphadenopathy</td>
<td>◦ Peripheral edema</td>
</tr>
<tr>
<td></td>
<td>◦ angina</td>
</tr>
</tbody>
</table>
Diagnosis of Myocarditis

- Good history taking, any recent illness
- EKG may have diffuse ST segment abnormalities
- Dysrhythmias and conduction disturbances may be present
- Labs: leukocytosis, increased ESR and CRP, elevated troponin
- Biopsy during the first 6 weeks of symptoms
Treatment of Myocarditis

- There are no standards of care treatment currently
- Management of symptoms
- Medications to improve cardiac output
- Most patients recover from myocarditis spontaneously
Pericarditis

- Inflammation of the pericardial sac, pericardium
Causes of Pericarditis

- Viral infection
- Bacterial infection
- TB
- Fungal infection
- Uremia
- Acute Myocardial infarction
- Trauma
- Radiation
- Dissecting Aortic Aneurysm
- Drug reactions
<table>
<thead>
<tr>
<th>Complications</th>
<th>Pericardial Effusion</th>
<th>Cardiac Tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Accumulation of excess fluid in the pericardium</strong></td>
<td>• Cough, dyspnea, tachypnea</td>
<td>• Compression of the heart from a build up of fluid</td>
</tr>
<tr>
<td><strong>Can occur rapidly or insidious onset</strong></td>
<td>• Hiccups, hoarseness</td>
<td>• Chest pain, confusion, anxious, restless</td>
</tr>
<tr>
<td><strong>Distant or muffled heart tones</strong></td>
<td>• Distant or muffled heart tones</td>
<td>• Muffled heart tones and pulse pressure is narrowed</td>
</tr>
<tr>
<td><strong>Cough, dyspnea, tachypnea</strong></td>
<td></td>
<td>• Tachypnea, tachycardia, decreased CO</td>
</tr>
<tr>
<td><strong>Hiccups, hoarseness</strong></td>
<td></td>
<td>• JVD and pulsus paradoxus</td>
</tr>
</tbody>
</table>
Diagnosis

- EKG
- Chest X-Ray
- Echocardiogram
- CT scan
- MRI
- Labs:
  - Leukocytosis
  - Elevated ESR, CRP, troponin
Treatment and Management

- Identify and treat underlying problem
- Antibiotics
- NSAIDS
- Aspirin
- Pericardiocentesis
- Bed rest
Ongoing assessment

- ABC’s
- Cardiac monitoring
- Support cardiac function by medications
- Manage pain and anxiety
- Patient education
Infective Endocarditis (IE)

- Infection of the endocardial surface of the heart
- Inner most layer of the heart
- Affects the cardiac valves
<table>
<thead>
<tr>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Prior endocarditis</td>
</tr>
<tr>
<td>• Prosthetic valve</td>
</tr>
<tr>
<td>• Valve disease</td>
</tr>
<tr>
<td>• Cardiac lesions</td>
</tr>
<tr>
<td>• Congenital heart defects</td>
</tr>
<tr>
<td>• Pacemakers</td>
</tr>
<tr>
<td>• Marfans syndrome</td>
</tr>
<tr>
<td>• cardiomyopathy</td>
</tr>
<tr>
<td>• IV drug abuse</td>
</tr>
<tr>
<td>• Intravascular devices</td>
</tr>
<tr>
<td>• Nosocomial bacteremia</td>
</tr>
</tbody>
</table>
Causes of IE

- *Staphlococcus aureus*
- *Streptococcus viridans*
- Fungi
- Viruses
### Signs and Symptoms

<table>
<thead>
<tr>
<th>Acute IE</th>
<th>Sub acute IE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low grade fever</td>
<td>Arthralgias (joint pain)</td>
</tr>
<tr>
<td>Chills</td>
<td>Back pain</td>
</tr>
<tr>
<td>Weakness</td>
<td>Abdominal discomfort</td>
</tr>
<tr>
<td>Malaise</td>
<td>Headache</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Clubbing of fingers</td>
</tr>
<tr>
<td>Anorexia</td>
<td></td>
</tr>
</tbody>
</table>
Clinical symptoms

- New or changing murmur
- Vascular manifestation include:
  - Splinter hemorrhages
  - Petechiae in conjunctiva, buccal mucosa, palate and over the ankles, inner bend of elbows and behind the knee
Diagnosis of IE

- History and Physical exam
- Blood cultures (2 sets)
- May have elevated WBC count
- Murmur
- Echocardiogram
- EKG
- Chest xray
Treatment

- Needs to be treated promptly
- Infection can spread to other parts of the heart and surrounding structures
- Need for prophylaxis treatment
Antibiotic prophylaxis

- Surgical procedures
- Dental procedures
- GI scoping
Treatment

- Accurate identification of the causative agent is imperative to the treatment of IE
- Prosthetic valve replacement
- Aspirin, acetaminophen, ibuprofen for fever/pain
- Fluids
- Rest
EKG interpretation

- P wave: 0.06-0.12 seconds
- PR interval: 0.12-0.20 seconds
- QRS complex: 0.4-0.12 seconds
- ST segment: 0.12 seconds
- T wave: 0.16 seconds
- QT interval: 0.34-0.43 seconds
Normal Sinus Rhythm

- Regular rate and rhythm
- 60-100 beats/minute
- Normal P wave, PR interval, and QRS complex
Sinus Dysrhythmia

- SA node fires less than 60 or greater than 100 beats/min
- Sinus bradycardia
- Sinus tachycardia
Sinus Bradycardia

- Regular rhythm
- Less than 60 beats/min
- Normal P Wave, PR interval, QRS complex
Sinus Bradycardia

- Monitor blood pressure
- Monitor patients ability to tolerate bradycardia
- Signs/symptoms include:
  - Pale, cool skin
  - Hypotension
  - Weakness
  - Dizziness or syncope
  - Confusion
Sinus Bradycardia Treatment

- Administration of atropine
- Pacemaker may be required
Sinus Tachycardia

- Regular rhythm
- Greater than 100 beats/minute
- Normal P wave, PR interval, and QRS interval
Sinus Tachycardia causes

- Fever
- Exercise
- Hypotension
- Hypovolemia
- Fear

- Anemia
- Hypoxia
- Hypoglycemia
- Anxiety
- Myocardial ischemia
Signs and Symptoms

- Dizziness
- Dyspnea
- Hypotension
- Chest pain
Sinus Tachycardia Treatment

- Treat underlying cause
- Beta blockers such as Metoprolol can be used
  - Monitor BP before administering medications
Atrial Dysrhythmias

- Premature Atrial Contraction (PVC)
- Paroxysmal Supraventricular Tachycardia
- Atrial Flutter
- Atrial Fibrillation
Premature Atrial Contraction

- A contraction developed from the atria, not at the SA node
- It can be stopped, delayed (causing longer PR interval), or conducted normally
PAC

- Irregular rhythm
- P wave has different shape
- PR interval may be shorter or longer
- QRS complex normal
PAC

- May be asymptomatic
- Monitor for occurrence
PAC Caused by:

- Emotional or physical fatigue
- Use of caffeine, tobacco, alcohol
- Hypoxia
- Electrolyte imbalances
- COPD
- CAD
PAC treatment

- Treat underlying cause
- Provide oxygen
- Stop the use of caffeine, tobacco, and alcohol
- Beta adrenergic blockers may be useful in decreasing amount of PAC’s
Paroxysmal Supraventricular Tachycardia (PSVT)

- Electrical dysrythmia that develops above the bundle of His
- Hard to determine exact place of origin
- Heart rate between 100-300 beats/min
- No distinguishable P wave, usually hidden in the previous T wave**

**Source Unknown**
PSVT causes

- Over exertion
- Caffeine
- Tobacco
- Stress
- Deep inspiration
- Exercise
PSVT Treatment

- Some spontaneously resolve
- Vagal maneuvers
  - Holding breath and bearing down
  - Ice on face
  - Forceful cough
- Adenosine
  - 6mg, followed by large rapid NS flush
  - Repeat at 12mg if unsuccessful
  - Repeat a third time at 12mg if unsuccessful
Atrial Flutter

- Recurring, regular, sawtooth-shaped flutter (called F waves)
- Originate in the Right atrium from a single ectopic focus

***insert pic of A. Flutter***
Atrial Flutter

- Beats normally 250-300 beats/min, ventricular rate usually around 150 beats/min
- Described by how many atrial beats are between ventricular beats ex: 3:1, or 4:1
- PR interval is unable to be measured
- QRS usually normal
Atrial Flutter

- Usually seen in diseased hearts:
  - CAD
  - HTN
  - PE
  - Chronic lung disease
  - cardiomyopathy
Atrial Flutter

- **Symptoms include**
  - Palpitations
  - Fluttering in chest
  - Shortness of breath
  - Weakness
  - Anxiety
Atrial Flutter Treatment

- Medications such as beta adrenergic blockers or calcium channel blockers
- Electrical cardioversion
Junctional Dysrhythmias

- When the SA node fails to fire, or the electrical signal has been blocked, the AV node takes over and becomes the pacemaker.
- The impulse from the AV node goes backwards, producing an abnormal P wave.
- P wave can be found right before QRS complex, hidden in the QRS complex or right after the QRS complex.
Junctional Dysrhythmias

- **Junctional escape**
  - Heart rate 40-60 beats/minute

- **Accelerated junctional**
  - Heart rate 61-100 beats/minute

- **Junctional tachycardia**
  - Heart rate 101-150 beats/minute
Junctional Dysrhythmias

- Can be associated with:
  - Coronary Artery Disease
  - Heart Failure
  - Cardiomyopathy
  - Electrolyte imbalances
  - Inferior wall MI
  - Certain drugs
Treatment

- Determined by the patient's tolerance of the rhythm and clinical condition
- Treat underlying cause, example digoxin toxicity
- Atropine may be needed if clinically indicated
First Degree AV Block

- Prolongation of the PR interval to greater than 0.20 seconds
- Heart rate is normal and rhythm is regular
- No changes to QRS
First Degree AV Block
First Degree AV Block

- Patients are usually asymptomatic
- No treatment
- Monitor patient for worsening blocks or arrhythmias
Second Degree AV Blocks

- **Second Degree Type I**
  - Also known as Mobitz I or Wenckbach
- **Second Degree Type 2**
  - Also known as Mobitz II
Second Degree Type I

- Gradual lengthening of the PR interval until the atrial impulse is nonconducted, meaning the QRS complex is blocked.
- Atrial rate is normal, ventricular rate may be slower
- Usually results from ischemia or infarction
- Usually transient and well tolerated
Second Degree Type I - EKG

- Rhythm on EKG appears in groups
- Ventricular rhythm is irregular
- P wave has normal shape
- QRS complex is normal
Second Degree Type I
Second Degree Type 1- Treatment

- Atropine if patient is symptomatic
- Pacemaker may be necessary
- If asymptomatic, closely monitor, with transcutaneous pacer on stand-by
Second Degree Type II

- Impulses from the SA node are not conducted through the ventricles, causing a blocked QRS complex on EKG
- Usually occurs in the His-Purkinje system
Second Degree Type II

- Atrial rate is normal
- Ventricular rate may be irregular
- P wave is normal
- PR interval may be normal or prolonged, regular in duration
- QRS complex usually more than 0.12 seconds
Second Degree Type II
Second Degree Type II- Treatment

- This usually progresses to third degree heart block
- Usually will have decreased cardiac output indicating need for permanent pacemaker
Third Degree Heart Block

- Also known as complete heart block
- The atrium and the ventricles are contracting independently
- Associated with coronary artery disease, myocardial infarction, myocarditis, or cardiomyopathy
Third Degree Heart Block

- Atrial and ventricular rhythms are normal, but do not coordinate with each other
- P wave is normal shape
- PR interval is variable
- No time relationship between P wave and the QRS complex
- QRS usually normal shape
Third Degree Heart Block
Third Degree Heart Block
Premature Ventricular Contraction (PVC)

- Premature contraction originating in the ectopic area of the ventricle
- Early QRS complex on EKG
Premature Ventricular Contraction (PVC)

- QRS is wide distorted in shape
- Different shapes when the electrical impulse are from different areas of the ventricle
  - Unifocal: PVC’s that appears to have the same shape
  - Multifocal: PVC’s that have different shapes from each other
Premature Ventricular Contractions

Unifocal  Multifocal

Unifocal:

Multifocal:

Source Unknown
Premature Ventricular Contractions

- Ventricular Bigeminy
  - Every other beat is a PVC
- Ventricular Trigeminy
  - Every third beat is PVC
- Couplet
  - Two consecutive PVCs
Ventricular Bigeminy

Source Unknown
Ventricular Trigeminy
Couplet
PVCs

- **Associated with stimulants**
  - Caffeine, alcohol, nicotine, epinephrine, digoxin

- **Also associated with:**
  - Electrolyte imbalances
  - Hypoxia
  - Fever
  - Stress
  - exercise
PVCs

• Can also be found in disease states:
  - Myocardial infarction
  - Mitral valve prolapse
  - Heart failure
  - Coronary artery disease
PVCs

- Usually benign in a healthy heart
- May reduce cardiac output in the diseased heart
- Treat underlying cause
- Drugs if hemodynamically unstable
  - Procainamide, amiodarone, or lidocaine
Ventricular Dysrhythmias

- Ventricular Tachycardia (V. Tach or VT)
- Ventricular Fibrillation (V. Fib or VF)
Ventricular Tachycardia (VT)

- Three or more PVC’s in a row
- Occurs when an ectopic focus fire repetitively and the ventricle takes over as the pacemaker
- Life threatening dysrhythmia
- Can lead to ventricular fibrillation

Source Unknown
Ventricular Tachycardia
EKG changes

- Rate is 150-250 beats/minute
- Rhythm may be regular or irregular
- P waves occurs independently of the QRS
- QRS complex is distorted, duration longer than 0.12 seconds, ST-T wave in the opposite direction of the QRS
<table>
<thead>
<tr>
<th>Stable</th>
<th>Unstable</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient has a pulse</td>
<td>No pulse</td>
<td></td>
</tr>
<tr>
<td>Sustained VT will lead to decreased cardiac output causing severe hypotension, pulmonary edema, decreased cerebral blood flow and lead to cardiopulmonary arrest</td>
<td><em><strong>START CPR</strong></em></td>
<td></td>
</tr>
</tbody>
</table>
Stable VT - Treatment

- Treat underlying cause:
  - Electrolyte imbalances
  - Ischemia
  - Digitalis toxicity
- Anti-arrhythmics
  - procainamide, sotalol, amiodarone, lidocaine
- Cardioversion
Name: [Name]
ID: [ID]
Age: 40
Sex: [Sex]
12-Lead 10
Date: [Date]

HR 197bpm
PR 0.000s
QRs 0.174s
QRS 0.300s/0.543s
Left axis deviation
Left bundle branch block

*Abnormal ECG **Unconfirmed**

0.05-150Hz 25mm/sec
MEDTRONIC PHYSIO-CONTROL P/N 805319
Unstable VT - Treatment

- ***START CPR*****
- Rapid defibrillation
- Same treatment as ventricular fibrillation
Ventricular Fibrillation (VF)

- Irregular varying shapes and amplitude
- Firing of multiple ectopic foci in the ventricle
- Ventricle is quivering resulting in no cardiac output
Ventricular Fibrillation

EKG

- Heart rate is immeasurable
- Rhythm is irregular and sporadic
- P wave is not visible
- PR interval and QRS are also immeasurable
- Patient is pulseless
Ventricular Fibrillation Treatment

• Cardiopulmonary Resuscitation
• If CPR is not started quickly, pt will die
Asystole

- Total absence of ventricular electrical activity
- Patients are
  - Unresponsive
  - Pulseless
  - Apneic
Asystole Treatment

- CPR
Pulseless Electrical Activity (PEA)

- Electrical activity is seen, however no ventricular movement or contraction
- Patient has no pulse
- EKG may look like NSR
PEA - treatment

- Same as pulseless VT and VF
- Start CPR
<table>
<thead>
<tr>
<th>Hypertensive Urgency</th>
<th>Hypertensive Emergency</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Elevated BP usually systolic greater than 180 and diastolic greater than 120</td>
<td>• Same as hypertensive urgency, however hypertension results in internal organ damage</td>
</tr>
<tr>
<td>• Immediate treatment necessary</td>
<td></td>
</tr>
</tbody>
</table>
Hypertensive emergencies
Sign/symptoms

- Headache
- Nausea
- Vomiting
- Seizures
- Confusion
- encephalopathy
Hypertensive Urgency

- May be able to be treated with oral anti-hypertensives
- May require one dose of IV anti-hypertensives
Hypertensive Emergency treatment

- **IV anti-hypertensives**
  - **Vasodilators**
    - Nitroprusside
    - Nitroglycerin
    - Fenoldopam
    - Hydralazine
    - Nicardipine
Acute Aortic Dissection

- A tear in the inner most layer of the arterial wall of the aorta
- Most commonly found in the thoracic aorta
Aortic Dissection

- The tear in the innermost layer (intimal) of the artery allows blood to track between the intima and media and creates a false lumen of blood flow. As the heart contracts, each systolic pulsation causes increased pressure on the damaged area, which further increases the dissection.

Reference:
Aortic Dissection

- As the dissection moves upward or downward, it can occlude major branches of the aorta and cause complete cut off of circulation to the brain, kidneys, abdominal organs, spinal cord and extremities
Aortic Dissection

- Sudden severe pain to anterior chest with pain radiating to back, between the shoulder blades, or pain radiating down the spinal cord or abdomen
- Pain often described as: ripping or tearing
Diagnosis

- Chest X-Ray
- Transesophageal echocardiogram
- MRI or CT scan of the chest
Treatment

- Keep BP low, use IV anti-hypertensives
- Treat pain
- If stable, may not require surgery
- If symptoms are present, surgical intervention may be necessary
## Peripheral Venous Thrombosis

<table>
<thead>
<tr>
<th><strong>Superficial thrombophlebitis</strong></th>
<th><strong>Deep vein thrombosis (DVT)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Inflammation of the superficial vein</td>
<td>• Disorder involving a thrombus (clot) in a deep vein</td>
</tr>
<tr>
<td>• Occurs in 65% of patients receiving IV therapy</td>
<td>• Most commonly found in the iliac and femoral vein</td>
</tr>
<tr>
<td></td>
<td>• More serious d/t risk of embolization of thrombi to the lung</td>
</tr>
</tbody>
</table>
Risk factors for DVT

1. Venous Stasis
   - Prolonged immobility
   - A. fib
   - Chronic heart failure

2. Endothelial damage
   - Trauma
   - Fracture that causes damage to blood vessels
   - Contaminated IV equipment

3. Hypercoagulability
   - Clotting disorders
   - Cigarette smoking
   - Malignancies
DVT- clinical manifestations

- Unilateral leg edema
- Extremity pain
- Warm skin
- Errythema
- Systemic temperature $> 100.4^\circ F (38^\circ C)$
DVT- diagnosis and treatment

- Venous doppler (US to view blood flow through veins)
- Treatment
  - Bedrest
  - Elevation of affected extremity
  - Anticoagulation
Anticoagulation

- Most common treatment is low-molecular-weight-heparin (LMWH) and warfarin (coumadin)
- Warfarin takes several days before therapeutic INR is reached. Uses Lovenox, a LMWH, to bridge the gap.
- Normal INR: 0.75-1.25 secs, Therapeutic: 2-3
DVT- Prophylaxis

- Early mobilization after surgical procedures
- Bedrested pts should be moving positions often, dorsiflex their feet and rotate ankles every 2 to 4 hours
- Compression stockings on extremities to increase venous blood flow
Peripheral Vascular Disease (PVD)

- any disease or disorder of the circulatory system outside of the brain and heart
- It is caused by build-up of fatty material within the vessels, called atherosclerosis
- This is gradual process in which the artery gradually becomes blocked, narrowed, or weakened
Risk factors for PVD

- Older than 50 years
- Obesity
- Sedentary lifestyle
- Smoking
- Diabetes
- Hypertension
- High cholesterol
PVD – Signs/symptoms

- Pain in one or both calves, thighs or hips
- Pain occurs when walking or climbing stairs d/t increased oxygen demand
- Dull, cramping pain
- Sore foot or leg that will not heal
- One or both legs/feet that are cold, or change color
PVD - treatment

- Angioplasty with stents
- Meds to help lower BP, cholesterol, blood sugar and to quit smoking
- When the obstructive lesions are long and involve most of the vessel, surgery is the best alternative
Additional Source Information

for more information see: http://open.umich.edu/wiki/CitationPolicy
