AACN PCCN Webinar

Session 1
Cardiovascular

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I. INTRODUCTION

PCCN Test Plan

Cardiovascular: 33%

a. Acute Coronary Syndromes
   - Non-ST Segment Elevation MI
   - ST Segment Elevation MI
   - Unstable Angina
b. Acute Inflammatory Disease (e.g. myocarditis, endocarditis, pericarditis)
c. Aneurysm
   - Dissecting
   - Repair
d. Cardiac Surgery (e.g. open chest surgery) – more than 48 hrs post-op
e. Cardiac Tamponade
f. Cardiogenic Shock
g. Cardiomyopathies
   - Dilated (e.g. ischemic/non-ischemic)
   - Hypertrophic
   - Stress-Induced (e.g. Takotsubo)
h. Dysrhythmia
   - Bradydysrhythmias
   - Conduction Defects & Blocks
   - Device-related (e.g. ICD and Pacemaker)
   - Lethal Ventricular Dysrhythmias
   - Tachydysrhythmias
i. Genetic Cardiac Disease (e.g. long QT syndrome, Brugada syndrome)
j. Heart Failure
   - Acute Exacerbations (e.g., pulmonary edema)
   - Chronic
k. Hypertensive Crisis
l. Minimally-Invasive Cardiac Surgery (i.e., non-sternal approach)
m. Septal Defects (congenital and acquired)
n. Valvular Heart Disease
   • Aortic
   • Mitral

o. Vascular disease
   • carotid artery stenosis
   • minimally-invasive interventions (e.g., stents, endografts)
   • peripheral arterial occlusions
   • peripheral surgical interventions
   • peripheral venous thrombosis

**Cardiovascular Testable Nursing Actions**

a. Perform a comprehensive cardiovascular assessment

b. Identify, interpret, and monitor
   • Dysrhythmias
   • ST segments
   • QTc intervals

c. Select leads for cardiac monitoring for the indicated disease process

d. Recognize indications for and manage patients requiring hemodynamic monitoring using non-invasive hemodynamic monitoring

e. Monitor hemodynamic status and recognize signs and symptoms of hemodynamic instability
   • Pacemakers
   • Defibrillation
   • Arterial/venous sheaths
   • Transesophageal echocardiogram (TEE)

f. Monitor patients pre- and post-procedure
   • Cardioversion
   • Pericardiocentesis
   • Cardiac catheterization
   • Ablation
   • Arterial closure devices

g. Monitor normal and abnormal cardiovascular diagnostic test results

h. Administer cardiovascular medications and monitor response

i. Titrate vasoactive medications

j. Recognize signs and symptoms of cardiovascular emergencies, initiate standardized interventions, and seek assistance as needed

k. Monitor and manage patient following coronary intervention
II. ANATOMY & PHYSIOLOGY

III. CARDIAC ASSESSMENT

Cardiac Risk Factors

a. Nonmodifiable
   • Age
   • Gender
   • Family History
   • Race
b. Modifiable
   • Smoking
   • Hypertension
   • Diabetes
   • Obesity
   • Stress
   • Exercise
   • Hyperlipidemia
c. Medical & Surgical History
d. Social History
e. Medication History
f. Physical Exam
   • Color
   • Pulses
   • Rate & Rhythm
   • PMI Location
   • Extremity Temperature
   • Dyspnea
   • Fatigue Level
   • Fluid Retention
   • Palpitations
   • Dizziness
g. Chest Pain Exam
   • PQRST Assessment
     o P: Pain, Placement, Provocation
     o Q: Quality (sharp, stabbing, pressure) Quantity
     o R: Radiation, Relief
     o S: Severity, Systems (nausea, sweaty, dizziness)
     o T: Timing (when it started, how long did it last, what makes it better or worse)

h. Hemodynamic Stability
   • Vital Signs: supine, sitting and standing
   • Work of Breathing, Breath Sounds (congestion)
   • LOC
   • Noninvasive Cardiac Output Monitoring

Diagnostic Tests & Procedures

a. 12 Lead ECG
b. Echocardiography (Transthoracic and Transesophageal)
c. Stress Test
d. Cardiac Catheterization
e. Doppler Ultrasound
f. Blood Work
   • Acute Coronary Syndrome
     o Cardiac Enzymes: CK-MB
     o Amino Acids: Troponins
     o Heme Proteins: Myoglobin
   • Lipid Profile
     o Triglycerides
     o Cholesterol
     o Low Density Lipoproteins
     o High Density Lipoproteins
   • Coagulation Profile
     o PT/INR
     o aPTT
     o ACT
   • Miscellaneous
     o B Type Natriuretic Peptide (BNP)
     o C Reactive Protein
     o Homocysteine

Cardiac Assessment, Test Prep and Monitoring are listed under Nursing Actions. Remember to Review!
## Determinants of Cardiac Output

**CARDIAC OUTPUT**

**HEART RATE X STROKE VOLUME**

<table>
<thead>
<tr>
<th><strong>PRELOAD</strong></th>
<th><strong>AFTERLOAD</strong></th>
<th><strong>CONTRACTILITY</strong></th>
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<tbody>
<tr>
<td>• The volume of blood in the ventricle at end diastole</td>
<td>• The pressure or resistance the LV must contract against or overcome to eject the blood or create systole</td>
<td>• The ability of the myocardium to contract</td>
</tr>
<tr>
<td>• Total blood volume &amp; venous tone</td>
<td>• Arterial Tone</td>
<td>• Ventricular size, myocardial fiber stretch/shortening ability</td>
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<td></td>
<td>• Arterial constriction vs Arterial dilation</td>
<td>• Calcium availability</td>
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</table>

### Measurements

- **RV:** CVP 2-6mmHg
- **LV:** PAOP 4-12mmHg

**RV: PVR = (MAP-PAOP) X 80**

**LV: SVR = MAP-RAP x 80**

- **PVR normal 37-250dynes/sec/cm^5**
- **SVR normal 900-1400dynes/sec/cm^5**

**RV: RVSWI = SVI(PAM-CVP) X 0.0136**

**LV: LVSWI = SVI(MAP-PAOP) X 0.0136**

- **Normal 5-10g/beat/m^2**
- **Normal 45-65g/beat/m^2**

**LVEF = LVEDV X 100**

**RVEF = RVEDV X 100**

**CVP:** central venous pressure, **EF:** ejection fraction, **LV:** left ventricle, **LVEDV:** left ventricular end-diastolic volume, **LVEF:** left ventricular ejection fraction, **MAP:** mean arterial pressure, **MPAP:** mean pulmonary arterial pressure, **PAOP:** pulmonary artery occlusion pressure, **PVR:** pulmonary vascular resistance, **RAP:** right atrial pressure, **RV:** right ventricular, **RVEDV:** right ventricular end-diastolic volume, **RVEF:** right ventricular ejection fraction, **RVSWI:** right ventricular stroke work index, **SV:** stroke volume, **SVI:** stroke volume index, **SVR:** systemic vascular resistance

*Preload: The volume of blood creating a stretch on the muscle chamber at the end of diastole.*

**Decreases in Preload**

a. Hypovolemia  
b. Arrhythmia  
c. Loss of “Atrial Kick”  
d. Venous Vasodilatation
Increases in Preload
a. Left Heart
   • LV Failure/Dysfunction
   • Mitral Valve Disease
   • Aortic Valve Disease
   • Cardiac Tamponade/Effusion
   • Volume Overload
   • Decreased Compliance
b. Right Heart
   • RV Failure Due to Ischemia
   • Increased Pulmonary Vascular Resistance
   • Cardiac Tamponade/Effusion
   • Volume Overload
   • LV Failure

Afterload: The pressure or resistance the LV must contract against or overcome to eject the blood or create systole.

Decreases in Afterload
a. Vasodilation
b. Sepsis
c. Vasodilator Therapies

Increases in Afterload
a. Right Heart
   • Pulmonary Hypertension
   • Hypoxemia
   • Pulmonic Stenosis
b. Left Heart
   • Vasoconstriction
   • Vasopressors
   • Hypothermia
   • Aortic Stenosis

Contractility: The ability of the myocardium to contract.

Decreased Contractility
a. Parasympathetic Stimulation
b. Negative Inotropic Therapies
   • Beta Blockers
   • Calcium Channel Blockers
c. Metabolic States
   • Hyperkalemia
   • Myocardial Ischemia/Infarct
   • Acidosis
Increased Contractility

a. Sympathetic Stimulation

b. Inotropic Therapies
   - Epinephrine
   - Dopamine
   - Digoxin
   - Calcium

c. Metabolic States
   - Hypercalcemia

Heart Failure

I. PATHOPHYSIOLOGY OF HEART FAILURE

Definitions

a. Heart Failure is the inability of the heart to adequately supply blood to meet the metabolic demands of the tissues resulting in inadequate tissue perfusion and volume overload.
b. Acute Heart Failure occurs when the inability to meet the demands of the tissues takes place abruptly, frequently without time for compensatory mechanisms to be activated. If the failure is severe or rapid enough the result will be cardiogenic shock.

Cause

Heart failure is a potential complication of most cardiac conditions and many organic and systemic problems. Acute failure is frequently the result of a new event or progression of a preexisting heart failure state.

a. Cardiac Anatomical Causes
b. Cardiac Physiological Causes
c. Non-Cardiac Causes

Mechanism of Failure

a. Although failure may be caused by a variety of cardiac and non-cardiac pathologies, the outcome is the same - decline in cardiac function leads to a drop in cardiac output (CO). Low CO stimulates initial and progressive adaptation phases.
b. Initial Adaptation to Low CO
   - Drop in CO \( \rightarrow \) Drop in Ejection Fraction (EF)
   - Increase in End Diastolic Volume \( \rightarrow \) Myocardial Fiber Stretch
   - Increase contractility (augmented sarcomere sensitivity to Ca\(^{++}\))
• Activation of the Neurohormonal Systems
  o Adrenergic System
  o Renin-Angiotensin-Aldosterone System
  o Hypothalamic-Neurohypophyseal System
  o Endothelium Activated Mediators

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### Activation of the Neurohormonal Systems

**Adrenergic** + **Renin-Angiotensin** + **Hypothalamic-Neurohypophyseal** + **Endothelium**

- **Baroreceptors:**
  - Low BP/CO $\rightarrow$
  - Activation of Sympathetic Nervous System (SNS) $\rightarrow$
  - Norepinephrine & Epinephrine $\rightarrow$
  - Beta Stimulation
    - $\uparrow$ Heart Rate
    - $\uparrow$ Contractility
  - Alpha$_3$ Stimulation
  - Vasoconstriction

- **Low BP/CO $\rightarrow$**
  - $\downarrow$ GFR $\rightarrow$
  - Renin Release $\rightarrow$
  - Angiotensin I $\rightarrow$
  - Angiotensin II $\rightarrow$
  - Vasoconstriction
  - Aldosterone Release $\rightarrow$
  - $\text{Na}^+$ & $\text{H}_2\text{O}$ Retention

- **Low BP/CO $\rightarrow$**
  - Release of Vasopressin (ADH) from Posterior Pituitary $\rightarrow$
  - Vasoconstriction & $\text{Na}^+$ & $\text{H}_2\text{O}$ Retention

- **Low BP/CO $\rightarrow$**
  - Endothelin $\rightarrow$
  - Nitric Oxide, Endothelium-Derived Relaxing Factor (EDRF) $\rightarrow$ Vasodilation

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**Increased HR**
**Increased Contractility**
**Vasoconstriction**
**Sodium & $\text{H}_2\text{O}$ Retention**

**Increased CO & BP**

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c. Progression of Heart Failure

- Continued Activation of the Sympathetic System Causes Increased Afterload
- Release of Natriuretic Peptides: ANP & BNP
  - Atrial Natriuretic Peptide (ANP): produced by the stretched atria $\rightarrow$ promotes diuresis, vasodilation. Not strong enough to counteract the vasoconstricting mechanisms of the initial compensatory response
  - Brain Natriuretic Peptide (BNP): produced by the ventricles, is a marker for ventricular dysfunction and produces the same response as ANP
- Release of Cytokines
  - Tumor Necrosis Factor (TNF-α): produced secondary to hypervolemia, triggers both systemic and cardiac inflammatory responses
- Cardiac Hypertrophy & Remodeling: initially adaptive in nature, eventually leads to hypertrophy, ventricular dilation and increased $\text{O}_2$ demands leading to $\downarrow$CO & ischemia
- Reflex Response from the Baroreceptors, Stretch Receptors
- Increase Demand and Decrease Function $\rightarrow$ Progressive Failure
Classifications of Heart Failure

a. Systolic vs Diastolic
b. Right vs Left
c. High-Output vs Low-Output
d. Compensated vs Decompensated
e. New York Heart Association Classification of Congestive Heart Failure
   • Class I No Symptoms
   • Class II Symptoms on Maximal Exertion
   • Class III Symptoms on Minimal Exertion
   • Class IV Symptoms occur at Rest
f. ACC/AHA Evolution & Progression Classification System
   • Stage A At high risk for heart failure but without structural heart disease of symptoms of HF
   • Stage B Structural heart disease but without symptoms of HF
   • Stage C Structural heart disease with prior or current symptoms of HF
   • Stage D Refractory HF requiring specialized interventions

Signs & Symptoms of Heart Failure

a. Cardiac
   • Tachycardia
   • Weak Pulses
   • Low CO & BP
   • Jugular Venous Distention
   • S3 Diastolic Gallop
   • Displaced PMI
   • Chest X-ray: cardiomegaly and vascular prominence
   • 2D Echo: valvular abnormalities, cardiac enlargement
   • Peripheral Edema
   • Positive Hepatojugular Reflux
b. Pulmonary
   • Dyspnea
   • Bibasilar Rales
   • Paroxysmal Nocturnal Dyspnea
c. Neurological
   • Fatigue, Weakness &/or Dizziness
   • Change in LOC
   • Feeling of Impending Doom
II. HEART FAILURE MANAGEMENT

Goals of Therapy

a. Prevent and/or Reverse Cause of Failure
b. Decrease the Negative Spiral of the Compensatory Mechanisms
c. Decrease Demands on the Heart
d. Decrease Ectopy or Maintain Electrical Stability
e. Focus on Quality of Life

Prevent and/or Reverse Cause of Failure

a. If the primary cause is poor coronary perfusion the tx should be directed towards opening the arteries and revascularization of the myocardium.
   - Thrombolytics
   - Percutaneous Coronary Interventions (PCI)
   - Coronary Artery Bypass Graft Surgery (CABG)
b. Surgery to repair anatomical problem
c. Treat the physiological cause: CAD, HTN, Dysrhythmia

Decrease the Negative Spiral of the Compensatory Mechanisms.

The major treatment modalities in this category are pharmacologic

Vasodilators

Venodilators and arteriodilators are both helpful in the management of heart failure. They can decrease preload, decrease afterload, increase renal perfusion and improve symptoms of both failure and those related to the compensatory response.

a. Angiotensin-Converting Enzyme (ACE) Inhibitors: ACE inhibitors are now the flagship agent in drug management for heart failure. Utilized as a solo agent or in combination with other drugs.
   - Dilate Arterioles
   - Dilate Veins
   - Decrease Release of Aldosterone (decreasing the need for high doses of diuretics)
b. Angiotensin II Receptor Blockers (ARB): block the actions of Angiotensin II. Same pharmacological affects as ACE inhibitors without adverse effects of cough, hyperkalemia & angioedema. Used with ACE Inhibitors are not tolerated.
c. Hydralazine (Apresoline) a selective arteriole dilator and Isosorbide dinitrate (Isordil, sorbitrate) a nitrate that is a selective venous dilator, are commonly used together to create a similar effect as the ACE inhibiting agents. ACE inhibitors should be used first.
d. Nitroglycerin and the nitrate class drugs work directly on the vascular smooth muscle causing venodilation with only minimal arteriole dilation.
e. Calcium Channel Blockers: It would seem logical that Ca++ blockers because of their vasodilating effect would be useful in the tx of heart failure. Clinical trials have shown just the opposite. These agents have either shown not to be helpful or in some causes to actually be harmful to pts with heart failure. They are not recommended for use in the tx of heart failure.
f. **B-Type Natriuretic Peptide**: Nesiritide (Natrecor) simulates cGMP production and binds to the receptors in vasculature and kidneys. Increases cardiac output and GFR, decreases aldosterone levels, in order to promote diuresis. Main side effect is hypotension.

**Diuretics**  
One of the first string management agents for heart failure that will decrease both preload and afterload by reducing water retention. The therapeutic goal is to decrease the work of the failing heart muscle. Diuretics are recommended for use with all patients who have symptomatic heart failure.  
   a. Potassium Sparing Diuretics  
   b. Thiazide Diuretics  
   c. Loop Diuretics

**Inotropic Agents**  
Increase the force of myocardial contraction enhancing stroke volume → increasing cardiac output.  
   a. **Cardiac Glycosides**: Digoxin (Lanoxin) promotes the accumulation of Ca^{++} within the cardiac cell and therefore contractility by inhibiting Na^{+}/K^{+}ATPase. It also decreases heart rate by slowing conduction through the AV node.  
   b. **Sympathomimetics**: Dobutamine (Dobutrex) a synthetic catecholamine with selective beta-adrenergic agonist properties  
   c. **Phosphodiesterase Inhibitors**: Amrinone (Inocor) and Milrinone (Primacor) are non-catecholamine agents that increase contractility by increasing cyclic adenosine monophosphate (cAMP) → which enhances Ca^{++} entry into the cell. By blocking PDE III they block sympathetic vasoconstriction causing vasodilation.

**Beta Blockers**  
The normal response to beta blockage (in the non heart failure pt) is decreased heart rate, reduced forced of contraction, and decreased velocity of impulse conduction through the AV node. Blocking the activation of the sympathetic nervous system has an effect on the negative spiral of compensatory responses in heart failure.

**Decrease Demands on the Heart**  
   a. Intra-Aortic Balloon Pump (IABP)  
   b. Ventricular Assist Devices (VAD)
Decrease Ectopy and Maintain Electrical Stability

a. Approximately half of deaths from heart failure occur suddenly and are most likely the result of a dysrhythmia.
b. Treatments:
   • Oral Antidysrhythmic Agents: Amiodarone, Beta Blockers, Digoxin
   • Pacemakers:
     o Atrial(A) Pacing: electrode in right atria and spike before P wave
     o Ventricular(V) Pacing: electrode in right ventricle and spike before QRS Complex
     o Atrial/ Ventricular (AV) Pacing – Dual Chamber Pacing: electrode in both right sided chambers and spike before P and QRS complex
     o Biventricular or Cardiac Resynchronization Therapy: electrode in RA, RV and outside of LV
     o Troubleshooting Pacing: Failure to Capture, Failure to Sense, Failure to Fire
c. Implantable Cardioverter Defibrillators (ICD)
d. Cardiac Transplantation
e. Quality of Life Focus

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<th>Pacing Codes</th>
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<tbody>
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<td><strong>D</strong></td>
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<tr>
<td>Chamber Paced O=None A=Atrium V=Ventricle D=Dual (A+V)</td>
</tr>
</tbody>
</table>

III. CARDIOMYOPATHY

a. Dilated (congestive) Cardiomyopathy (DCM) Most Common Form
   • Ischemic
   • Non-Ischemic
   • Stress Induced
b. Hypertrophic Cardiomyopathy (HCM)
c. Restrictive Cardiomyopathy
d. Treatment
   • Treat the Causative Factors
   • Rest Heart
   • Relieve Pulmonary and Systemic Congestion
   • Prevent Thromboembolic Events (DCM)
   • Antidysrhythmic Agents/Pacer/ICD
   • Assist Devices
   • Consider Transplant
• Pharmacology
  o Digitalis *
  o Diuretics*
  o Beta-Blockers, Ace Inhibitors
  o Vasodilators
  o Inotropic Agents*
  o Antidysrhythmics
  o Anticoagulants
*caution with HCM
I. INTRODUCTION

The rupture or disruption of the plaque is caused from internal and/or external factors or triggers.

Definitions of chest pain syndromes

**Angina**
Myocardial Anoxia

**Exertional Angina**
Pain that is brought on during times of increased myocardial oxygen demand like exertion, eating, extreme emotions and exposure to cold temperatures, the four Es. These symptoms are typically caused by or a sign of atherosclerosis.

**Prinzmetal’s Angina or Variant Angina**
Pain that occurs at rest, during sleep or without evidence of provocation. Symptoms are thought to be caused by coronary vasospasm.

**Stable Angina**
Exertional angina with consistent symptoms which is typically relieved with rest or cessation of cause and possibly nitroglycerine administration.

**Unstable Angina**
Aka crescendo or pre-infarction angina. Angina that:
  a. Has a recent onset (within 2 months) and severely limits activity
  b. Newly occurs at rest
  c. Differs in characters or symptoms from the person’s ‘typical exertional angina’ (it occurs with less exertion, has a greater intensity or longer duration, requires more interventions before obtaining relief)

**Non-CAD Causes**
Non-ischemic causes of chest pain must be ruled out, such as:

**Cardiac Causes**
  a. Acute Pericarditis
  b. Cardiac Tamponade
  c. Acute Myocarditis
  d. Aortic Stenosis
  e. Myocardial Contusion
  f. Mitral Valve Prolapse
  g. Cardiomyopathies
Non Cardiac Causes
a. Panic Attack/Anxiety
b. Illicit Drug Use
c. Gastrointestinal Disorders
d. Spontaneous Pneumothorax
e. Pulmonary Embolism
f. Pulmonary Hypertension
g. Esophageal Rupture
h. Costochondritis
i. Hypovolemia

II. UNSTABLE ANGINA

Pathophysiology
Partially occluding thrombus

Assessment

History
a. Assessment of Angina: PQRST Assessment
   P: Pain, Placement, Provocation
   Q: Quality, Quantity
   R: Radiation, Relief
   S: Severity, Systems (nausea, sweaty, dizziness)
   T: Timing (when it started, how long did it last)
b. Medical History
c. Medications: Prescription, Over the Counter, Dietary Supplements
d. Social History
e. Family History
f. Major Risk Factors of Atherosclerosis

12-Lead Electrocardiogram (ECG)
Note: Patient can have a ST Segment Elevation Myocardial Infarction (STEMI) or a non-ST Segment Elevation Myocardial Infarction (non-STEMI). All situations are unique and the treatment team must look at entire presentation.

a. Ischemia: ST Segment Depression
b. Injury: ST Segment Elevation
c. Infarction: Q waves
d. Brugada Syndrome: Rare genetic cardiac rhythm disease. Intermittent ST segment elevation in V1-V3 (Brugada's sign). May lead to syncope, and even sudden cardiac death. More common to present during sleep. More common in males. Tx is AICD
Session 1: cardiovascular

<table>
<thead>
<tr>
<th>Location</th>
<th>Indicative Leads</th>
<th>Reciprocal Leads</th>
<th>Coronary Arteries</th>
<th>Major Complications</th>
</tr>
</thead>
</table>
| Anterior | • Leads V1, V2, V3, V4 | • Leads II, III, aVF | • LAD | • Cardiogenic Shock  
• Bundle Branch Blocks  
• Vent Dysrhythmias |
| Inferior | • Leads II, III, aVF | • Leads I, aVL | • RCA | • Bradycardia  
• Heart Blocks |
| Lateral | • Leads I, aVL, V5, V6 | • Leads II, III, aVF | • Circumflex | • Heart Blocks in some |
| Septal | • Leads V1, V2 | • Leads V5, V6 | • RCA | • Bundle Branch Blocks |
| Posterior | • Leads V7, V8, V9 | • Leads V1, V2 | • RCA or Circumflex | • Heart Blocks |

**Biochemical Cardiac Markers**

**Creatine Kinase (CK)**

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<tr>
<th>CK enzyme</th>
<th>Male</th>
<th>Female</th>
</tr>
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<tbody>
<tr>
<td>CK total</td>
<td>60 – 170 U/L</td>
<td>40 – 140 U/L</td>
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This enzyme is important in the breakdown of creatine to creatinine. It increases in the serum when muscle damage has occurred. Three CK isoenzymes have been identified; CK-I BB from the brain tissue and smooth muscle, CK-II MB from heart tissue and CK-III MM from muscle tissue. CK serum levels will begin to rise 3-6hr after chest pain, peak in 12-24 hrs and return to normal in 2-3 days.

<table>
<thead>
<tr>
<th>Isoenzymes</th>
<th>CK I</th>
<th>CK II</th>
<th>CK III</th>
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<tbody>
<tr>
<td>BB</td>
<td>0 – 1%</td>
<td>&lt; 3 - 6%</td>
<td>95 – 100%</td>
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**Troponin**

cTn T: < 0.1 mcg/L  
cTn I: < 3.1 mcg/L

The troponin complex is found on cardiac and skeletal muscle. Troponin C, T, & I are proteins that work in synchrony to regulate the force and speed of muscle contraction. These proteins modulate the interaction of actin and myosin. The amino acid structures of cardiac troponin T & I (cTn T & cTn I) are uniquely different than skeletal muscle. During periods of cardiac ischemia, intracellular troponin will leak out of the cell. Troponin levels can be detected within 3-5hrs after chest pain, cTnT will peak in 4-6hrs and cTnI 14-18hrs, and cTnT will return to normal 21 days and cTnI in 5-7 days.

**Myoglobin**

50 – 120 mcg/mL  
Carbonic Anhydrase III (CA-III): 13 – 29 mcg/L

Myoglobin is a heme protein located on cardiac and skeletal striated muscle. Due to its low molecular wt. it is released very rapidly from the muscle after an ischemic event (faster than
troponin or CK-MB). Serum levels will rise within 2 hrs of chest pain, peak in 3-15hrs and return to normal levels in 2 days. Because there is not a cardiac specific myoglobin, many non-cardiac events may cause an elevation. Carbonic Anydydrase III (CA-III) is another cytoplasmic protein found primarily in skeletal muscle. In skeletal muscle damage both CA III and myoglobin rise. In cardiac muscle damage there is only a rise in myoglobin. Therefore a rise in the myoglobin/CA III ratio is more indicative of an AMI than just an elevated myoglobin. A ratio of $\geq 3.21$ is considered abnormal and indicates for cardiac damage.

**Early Risk Stratification**

Early identification of the cause and severity of the pain is essential in determining triage and appropriate therapy. The five factors from the patient’s history that increase the likelihood that the ischemia is from CAD are:

a. Nature of Symptoms  
b. Prior History of CAD  
c. Gender & Age  
d. Number of CAD Risk Factors

**Treatment**

Treatment should be initiated as quickly as possible, while assessment is being completed. Immediate general-treatment includes:

a. Oxygen at 4L/min  
b. Aspirin 160-325mg (chewed)  
c. Nitroglycerin SL or spray  
d. Morphine IV (if pain not relieved by NTG)  
e. “MONA” meets the patient (Morphine, Oxygen, Nitroglycerin, Aspirin)  
f. Clopidogrel (Plavix) 600 mg, or prasugel (Effient) 60mg, or Ticagrelor (Brilinta) 180mg now part of AHA ACS guidelines (2013)

Once assessment is complete, patient is identified as having characteristics for one of four categories:

a. Non-Cardiac Diagnosis  
b. Chronic Stable Angina  
c. Possible Acute Coronary Syndrome (ACS)  
d. Definite ACS

**Possible ACS**

a. Give Aspirin – may have already done so  
b. Consider Primary Coronary Intervention in Cath Lab  
c. Consider Antithrombin Tx  
   - ASA  
   - Glycoprotein IIb/IIIa Inhibitor: Abciximab (ReoPro), Eptifibatide (Integrelin), Tirofiban (Aggrastat)  
   - Heparin  
d. Consider Beta Blocker
III. ACUTE MYOCARDIAL INFARCTION

Pathophysiology
Completely occlusive thrombus

Assessment

a. History
b. Physical Examination
c. 12-lead Electrocardiogram: ST elevation
d. Biochemical Cardiac Markers

Treatment

a. Give triple anti-thrombin tx
b. NTG if pain present
c. If ST-segment elevation - evaluate for reperfusion
   • Thrombolytics
   • Percutaneous Coronary Interventions
   • Coronary Artery Bypass Grafting

Thrombolytic Agents
Thrombolytic agents have been proven to decrease mortality and complications of acute MI.

Therapeutic Uses
Being given IV, directly into peripheral clot, intracoronary & intracerebral
a. Acute Coronary Thrombosis
b. DVT
c. Massive Pulmonary Emboli
d. Adjunct to PCI
e. Thrombotic Stroke
f. Combination tx of thrombolytic agents and GP IIb/IIIa, UFH, & LMWH have been shown to increase long term perfusion, mortality & morbidity

Absolute Contraindications
a. Active Bleeding
b. Aortic Dissection
c. Cerebral Neoplasm
d. History of Intracranial Hemorrhage
e. Recent (within 2 mo) intracranial or intraspinal surgery or trauma
f. Cerebral Vascular Disease (aneurysm, arteriovenous malformation)
g. Bleeding Diathesis
h. Severe Uncontrolled Hypertension (> 180/110)
Relative Contraindications
a. Recent (within 10 mo) Major Surgery
b. Recent (within 10 days) GI or GU bleeding
c. High likelihood of Left Heart Thrombus (mitral stenosis or A-fib)
d. Acute Pericarditis or Sub acute Bacterial endocarditis
e. Significant Liver Dysfunction
f. Pregnancy
g. Diabetic Hemorrhagic Retinopathy

Adverse Effects
a. Major Risk is for Bleeding
b. Should major bleeding occur
   • Stop infusion & other anticoagulants
   • Anticipate immediate head CT if ICH suspected
   • Administer cryoprecipitate, FFP, platelets
   • Aminocaproic acid (Amicar)

Interventional Cardiology
Percutaneous coronary interventions have increased in both number of procedures and success rates since the first balloon angioplasty was performed in 1977.

Percutaneous Coronary Interventions (PCI)
a. Diagnostic Coronary Angiography
b. Percutaneous Transluminal Coronary Angioplasty (PTCA)
c. Coronary Stents

Nursing Care Concerns
a. Pre Procedure
   • BUN/Creat Levels
   • Dye Allergy
   • Hydration Status
   • Anticoagulation and Antiplatelet Medications
   • Rate & Rhythm
   • Electrolyte Balance: Especially Potassium
   • Limb Circulation
b. Post Procedure/Potential Complications
   • Myocardial Ischemia
   • Stroke
   • Groin Site Bleeding
     o Arterial/venous sheaths
     o Arterial Closure Devices
   • Distal Circulation
   • Dysrhythmias
   • Coronary Artery Spasm
   • Abrupt Closure/Restenosis
Coronary Artery Dissection
• Peripheral Vascular Complication
• Discharge Education

**Coronary Artery Bypass Grafting**

**Purpose**
Revascularize the Heart.

**Procedure**
a. With the use of cardiopulmonary bypass, hypothermia and cardioplegia the heart is made motionless and bloodless.
b. Grafts are used to supply blood distal to occlusion.
c. Cannulation sites for bypass are typically the aorta & RA.
d. Minimally-invasive approach.
e. Smaller sternal incisions and non-sternal approaches. On and off bypass.

**Graft Option**
a. Saphenous Vein
b. Internal Mammary
c. Radial Artery
d. Gastric Artery

**Post Op Care: (for PCCN >48 hr post op care)**
a. Pain
b. Volume Overload
c. MI
d. Stroke
e. Dysrhythmias
f. Infection
g. Decrease CO
h. Impaired Gas Exchange
i. Impaired Work of Breathing
j. Hypoperfusion Complications

**Additional Nursing Concerns**
a. Pain
b. Immobility
c. Risk for Infection
d. Life Style Modification
e. Discharge Education
f. Nutrition

**Treatment Continued**
a. Give β-blocker
b. Once reperfused evaluate myocardial damage and provide post MI care
Complications of AMI
a. Cardiogenic Shock: infarction of ≥ 40% of the left ventricle
   • Hypotension: SPB < 100mmHg
   • Pulmonary Edema
   • Low Cardiac Output
   • Cardiogenic Pulmonary Edema
   • S&S of Poor Peripheral Perfusion
b. Arrhythmias Associated with Ischemia, Infarction & Reperfusion

Treatment Goals for Cardiogenic Shock
a. Assist Contractility
b. Alleviate Cause of Failure
c. Fluid
d. Pharmacological Agents
e. Coronary Reperfusion
f. Mechanical Assist