AACN ADULT CCRN Review

Cardiovascular System

Presenter: Bobbi Leeper MN, RN-BC, CNS M-S, CCRN, FAHA
Baylor University Medical Center
Dallas, Texas
BEHAVIORAL OBJECTIVES
At the end of this session, the participant will be able to:

1. State the normal values for direct and derived hemodynamic parameters.
2. Discuss factors that increase and decrease the direct and indirect hemodynamic parameters.
3. List the factors that affect:
   a. Preload
   b. Afterload
   c. Contractility
4. Discuss the hemodynamic variables and clinical presentation for:
   a. Cardiogenic shock
   b. Hypovolemic shock
5. Differentiate left and right ventricular failure.
6. State the indicative changes and appropriate leads associated with the primary sites of myocardial infarction.
7. Discuss important aspects of thoracic aneurysms, including clinical presentation and acute management.

CONTENT OUTLINE
I. Direct and Derived Hemodynamic Parameters
   A. Cardiac Output (CO):
      Normal CO: 4–8 L/min
      Cardiac Index = CO / BSA; 2.8 – 4.2 L/min/m²

      Formula: Stroke volume (SV) x heart rate (HR)
      1. Heart rate
         a. Bradyarrhythmias
         b. Tachyarrhythmias
      2. Stroke volume
         a. Determinants
            1) Preload (end diastolic volume)—how to assess:
               ⇒ RV: CVP / RA pressure (normal 2 – 6 mmHg)
               ⇒ LV: PAWP / LA pressure (normal 6 – 12 mmHg)
2) Afterload (pressure the ventricle must generate to open the semilunar valve and eject its contents)—how to assess:

- Systemic vascular resistance = LV afterload
  - Definition: reflects the overall resistance or impedance to systolic ejection into the entire systemic circulation. The greatest resistance to flow lies in the small arteries and arterioles
  - Formula: \( 80 \times (\text{MAP} - \text{RAP}) / \text{CO} \)
  - Normal: 800–1200 dynes/sec/cm\(^5\)
  - SVRI: 1970–2390 dynes/sec/cm\(^5\)/m\(^2\)
  - Causes of increased SVR
    - Volume infusions
    - Peripheral vasoconstriction
    - Low CO states
    - Hypothermia
    - Increased blood viscosity
  - Causes of decreased SVR
    - Diuretics
    - Vasodilators
    - Peripheral vasodilatation
    - Loss of vasomotor tone
    - Hyperdynamic phase of sepsis

- Pulmonary vascular resistance = RV afterload
  - Definition: Resistance or impedance to right ventricular ejection into the pulmonary vasculature
  - Formula: \( 80 \times (\text{MPAP} - \text{PAOP}) / \text{CO} \)
  - Normal: <250 dynes/sec/cm\(^5\)
  - PVRI: 255–285 dynes/sec/cm\(^5\)/m\(^2\)
  - Causes of increased PVR:
    - Hypoxia - PEEP
    - Pulmonary edema
    - ARDS
    - Pulmonary emboli
    - Congenital heart defects
  - Causes of decreased PVR
    - Vasodilator therapy - Prostaglandins
    - Correction of hypoxia

3) Contractility—how to assess:

- Stroke volume (SV) / stroke volume index (SVI)
- LV stroke work / index:
  - LVSWI: \( \text{SVI} \times (\text{MAP} - \text{PAOP}) \times 0.0136 \)
    - Normal: 50–62 gms – m/m\(^2\)/beat
  - RVSWI: \( \text{SVI} \times (\text{MPAP} - \text{RAP}) \times 0.0136 \)
    - Normal: 5–10 gms-m/m\(^2\)/beat
B. Pulmonary Artery Pressure
1. Normal range: 15–25/0–8 mmHg
2. Clinical significance
   a. High readings
      ⇒ Primary pulmonary hypertension
      ⇒ Valvular heart disease
   b. Low readings
      ⇒ Hypovolemia
      ⇒ Vasodilator therapy

C. Pulmonary Artery Occlusion Pressure
1. Normal range: 6–12 mmHg
2. Clinical significance
   a. High readings
      ⇒ Left ventricular failure
      ⇒ Mitral valve disease
      ⇒ Aortic valve disease
      ⇒ Cardiac tamponade
   b. Low readings
      ⇒ Hypovolemia

D. Mean Arterial Pressure
1. Definition: Average pressure in the circuit during systole and diastole
2. Formula: \[ \frac{\text{SBP} + (2 \times \text{DBP})}{3} \]
3. Normal: 70–105 mmHg
4. Causes of increased MAP
   a. Volume infusion
   b. Peripheral vasoconstriction
   c. Increased contractility
   d. Hypervolemia
   e. Vasopressors
5. Causes of decreased MAP
   a. Diuretics
   b. Peripheral vasodilatation
   c. Inotropic therapy
   d. Hypovolemia
   e. Vasodilators

E. Mean Pulmonary Artery Pressure
1. Definition: Average pressure in the pulmonary circuit during systole and diastole
2. Formula: \[ \frac{\text{SPAP} + (2 \times \text{DPAP})}{3} \]
3. Normal: 10–20 mmHg
4. Causes of increased MPAP
   a. Volume infusion
   b. Pulmonary vasoconstriction
   c. Decreased LV contractility
   d. Hypervolemia
   e. Hypoxia
   f. COPD
   g. Pulmonary hypertension
5. Causes of decreased MPAP
   a. Diuretics
   b. Pulmonary vasodilatation
   c. Inotropic therapy
   d. Hypovolemia
II. Manipulating Hemodynamics: Cardiovascular Drugs

A. Inotropes

1. Receptor-dependent vs. Phosphodiesterase inhibitors

B. Types of Receptors

1. Beta receptors
   a. $\beta_1$ receptors are found primarily in the heart. Stimulation produces ↑ heart rate, ↑ contractility
   a. $\beta_2$ receptors are found in the lungs and peripheral arterioles. Stimulation produces relaxation of the smooth muscle

2. Alpha receptors
   a. $\alpha_1$ receptors are found primarily in the lungs, peripheral arterioles. Stimulation produces constriction of the smooth muscle
   b. $\alpha_2$ receptors are found primarily in the brain

3. Dopaminergic receptors
   Found in the renal, mesenteric, and vascular beds. Stimulation produces vasodilation

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<tr>
<th>TISSUE</th>
<th>RECEPTOR</th>
<th>RESPONSE</th>
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<tbody>
<tr>
<td>Heart:</td>
<td>Beta</td>
<td>↑ rate</td>
</tr>
<tr>
<td>SA node</td>
<td>Beta</td>
<td>↑ conduction velocity</td>
</tr>
<tr>
<td>Atria</td>
<td>Beta</td>
<td>↑ rate</td>
</tr>
<tr>
<td>AV node</td>
<td>Beta</td>
<td>↑ conduction velocity</td>
</tr>
<tr>
<td>Ventricles</td>
<td>Beta</td>
<td>↑ contractility</td>
</tr>
<tr>
<td>Blood vessels:</td>
<td>Beta</td>
<td>Vasodilatation</td>
</tr>
<tr>
<td>Skeletal muscle</td>
<td>Alpha</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td>Skin, mucosa, GI tract &amp; kidney</td>
<td>Dopa, alpha</td>
<td>Vasodilatation</td>
</tr>
<tr>
<td>Renal</td>
<td>Dopa, alpha</td>
<td>Vasodilatation</td>
</tr>
<tr>
<td>Mesentery</td>
<td>Dopa, alpha</td>
<td>Vasodilatation</td>
</tr>
<tr>
<td>Bronchial smooth muscle</td>
<td>Beta</td>
<td>Relaxation</td>
</tr>
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### Receptor Dependent Inotropes

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<th>Specific Agents</th>
<th>Indications/Actions</th>
<th>Dosages</th>
<th>Side/Adverse Effects</th>
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</table>
| **Dopamine**    | • Shock states: cardiogenic, septic; post-cardiac surgery  
                    • Immediate precursor of norepinephrine  
                    • Neurotransmitter in the central and peripheral nervous system  
                    • Decreases aldosterone secretion in the adrenal cortex  
                    • Inhibits TSH and prolactin release  
                    • Inhibits insulin secretion  
                    • Titrate the IV infusion to achieve desired effects  
                    • 2–10 mcg/Kg/min = ↑ contractility (beta stimulation)  
                    • >10 mcg/Kg/min = vasoconstriction (alpha stimulation)  |
|                 |                     |         | Nausea, emesis  
                    Tachyarrhythmias (ventricular & supraventricular)  
                    Profound vasoconstriction |
| **Dobutamine**  | • Synthetic catecholamine; directly stimulates the β₁  
                    receptors, β₂ receptors, α receptors  
                    • Directly increases myocardial contractility and heart rate while modestly lowering peripheral vascular resistance  
                    • Will lose its effect during prolonged infusions due to downregulation of β receptors  
                    • Indications: congestive heart failure; shock states: cardiogenic, septic  
                    • Titrate the infusion to achieve desired effects  
                    • Usual dosage range: 2.5–20 mcg/Kg/min.  
                    • Half-life: 2.5–3 minutes  
                    • Do not administer in alkaline solutions  |
|                 |                     |         | Dysrhythmias |
| **Epinephrine** | • Cardiac effects are mediated through β receptors;  
                    • 0.005–0.02 mcg/Kg/min = ↑ heart rate; + inotropic effect, vasodilation → ↓ SVR  
                    • Vascular effects mediated through α receptors @ high doses: ↑ SVR, ↑ BP, renal artery vasoconstriction  
                    • β₂ stimulation → bronchodilation  
                    • Indications: low output states, cardiac arrest, shock states, asthma, anaphylaxis  
                    • 0.005–0.02 mcg/Kg/min = beta effects  
                    • Alpha effects: 1 mg IV push/via ET tube  
                    • Half-life = 2 minutes  |
|                 |                     |         | Restlessness, fear  
                    Tachyarrhythmias  
                    Severe hypertension → CVA, angina  
                    Hypokalemia  
                    Hypophosphatemia |
| **Norepinephrine** | • Naturally occurring catecholamine with effects that are dose dependent  
                        • low doses: β stimulation  
                        • higher doses: α stimulation  
                        • Indications: hypotensive states; cardiogenic shock (MI); GI bleeding  
                        • Titrate infusion via central line to achieve desired effect. Weigh cost/benefit ratio  
                        • Dosage/administration  
                        • Infusion rates 2–4mcg/min are suggested  
                        • Start at .05–0.1 mcg/Kg/min and titrate up  
                        • Half-life = 2.0–2.5 min  
                        • If infiltration occurs, the drug will cause sloughing of tissue; use phentolamine (Regitine) to block the intense vasoconstriction  |
|                 |                     |         | Contraindicated in mesenteric and renal thrombosis  
                    Side effects  
                    Tachyarrhythmias  
                    Headaches  
                    Tremors  
                    Restlessness  
                    Severe ↑ BP |
| **Phenylephrine** (Neosynephrine) | • Pure α stimulator; effects are primarily vascular, causing vasoconstriction resulting in ↑ SBP and ↑ DBP, ↑ PAP. Coronary and renal arteries constrict. If vasoconstriction is severe, blood flow to the vital organs could decrease  
                        • Initial dose: 100–180 mcg/min to achieve desired effect  
                        • Maintenance infusion: 40–60 mcg/min titrated to maintain BP  |
|                 |                     |         | Vasoconstriction  
                    Hypertension  
                    Bradycardia |
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| Epinephrine     | • Indirect effect; release of norepinephrine from storage sites<br>• At large doses, could stimulate $\beta_1$ receptors. A reflex bradycardia (from the ↑ BP) has been reported. This is mediated through vagal stimulation<br>  
  • Cardiac effects are mediated through $\beta$ receptors:<br>  0.005–0.02 mcg/Kg/min = ↑ heart rate;<br>  + inotropic effect, vasodilation → ↓ SVR<br>  • Vascular effects mediated through $\alpha$ receptors @ high doses: ↑ SVR, ↑ BP, renal artery vasoconstriction<br>  • $\beta_2$ stimulation → bronchodilatation<br>  • Indications: Low output states; cardiac arrest, shock states, asthma, anaphylaxis | • Pressor effects are immediate and will last 15–20 min<br>  • 0.005–0.02 mcg/Kg/min = beta effects<br>  • Alpha effects: 1 mg IV push/via ET tube<br>  • Half-life = 2 minutes | • Restlessness, fear<br>  • Tachyarrhythmias<br>  • Severe hypertension → CVA, angina<br>  • Hypokalemia<br>  • Hypophosphatemia |
<p>| Vasopressin     | • Antidiuretic hormone&lt;br&gt;  • Larger doses: $\alpha$ stimulator causing vasoconstriction. Note: does not have negative effects on myocardium such as those caused by epinephrine | • Initial dose (ACLS): 40 units&lt;br&gt;  • Infusion: 0.04 units/min | Vasoconstriction Hypertension                                                                                     |
| Phosphodiesterase Inhibitor |                                                                 |                                                                 |                                                                                                                   |
| Milrinone       | • Positive inotrope with less peripheral vasodilating effects than amrinone&lt;br&gt;  • Indications: low cardiac output states; acute CHF; cardiomyopathy | • Loading dose: 50 mcg/Kg—slowly over 10 minutes (undiluted)&lt;br&gt;  • Infusion: 50/250 cc start @ .5 mcg/Kg/min. Increase in increments of .37 mcg/Kg/min, max of .75 mcg/Kg/min | • Arrhythmogenic: SVT, VT&lt;br&gt;  • Headaches, tremors&lt;br&gt;  • Thrombocytopenia |
| Nitroglycerin   | Systemic and pulmonary venodilation&lt;br&gt;  • Decreased left and right ventricular filling pressures&lt;br&gt;  • Decreased left ventricular pressure volume relationship&lt;br&gt;  • Decreased aortic impedance&lt;br&gt;  • Decreased right and left ventricular afterload&lt;br&gt;  • Dilation of coronary arteries&lt;br&gt;  • Improvement of ischemic zone | • Indications&lt;br&gt;  • -Chest pain related to myocardial ischemia&lt;br&gt;  • -Preload reduction&lt;br&gt;  • -Afterload reduction&lt;br&gt;  • Dosage/administration: continuous infusion titrated to achieve desired effects. It is suggested that the infusion rate be started at 10 mcg/min and ↑ in 10 mcg/min increments until the desired effect is achieved | • Hypotension&lt;br&gt;  • Nitrate tolerance |</p>
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| Sodium nitroprusside | • Direct vasodilator with balanced effect on the arteriolar and venous systems.   | • Usual dosages are 0.25–10 mcg/Kg/min                                  | • CNS effects = nervousness, twitching, ataxia, headaches  
• Cardiac effects = hypotension, palpitations  
• Cyanide poisoning = impaired tissue oxygenation, confusion, hyper-reflexia, convulsions  
• Contraindications: use with caution in patients with hypothyroidism, hepatic or renal disease as well as those patients receiving other antihypertensive drugs |
| (Nipride)            | • In 10% of patients can increase pulmonary shunt; will see SpO₂ and PO₂ fall.    | • Duration of action: 1–5 minutes                                        |                                                                                                                                  |
|                      | • Can produce coronary steal syndrome                                               | • Long-term administration of the drug should be monitored with serum thiocyanate levels. |                                                                                                                                  |
|                      | • Indications:                                                                     | • Infusion rates of less than 3 mcg/Kg/min are not associated with toxicity |                                                                                                                                  |
|                      |   – Severe heart failure with ↑ SVR                                                | • Serum thiocyanate levels >10 mg/dL are considered to be toxic. (Lab costs: $100) |                                                                                                                                  |
|                      |   – Mitral regurgitation to ↓ afterload and improve                                 | • Poor renal function increases the risk for thiocyanate toxicity         |                                                                                                                                  |
|                      |   – Forward flow out of the ventricle                                              | antidote: sodium thiosulfate                                             |                                                                                                                                  |
|                      |   – Low CO syndrome with ↑ SVR                                                    |                                                                 |                                                                                                                                  |
|                      |   – Hypertensive crises                                                            |                                                                 |                                                                                                                                  |
| Nesiritide (Natrecor) | • Brain natriuretic peptide – identical to endogenous BNP                         | • Usual dosage:                                                          | • Side effect: Hypotension—monitor BP closely  
• Incompatible with:  
   – Enaliprilat  
   – Insulin  
   – Lasix  
   – Heparin  
   – Hydralazine  
   – Bumex |
|                      | • Effects                                                                          |   – Bolus: 2 mcg/Kg over 60 seconds                                        |                                                                                                                                  |
|                      |   – Vasodilation                                                                    |   – Infusion: 0.01 mcg/min                                                |                                                                                                                                  |
|                      |   – Natriuresis                                                                     | • Do not infuse though the same line with other medications               |                                                                                                                                  |
| Nicardipine (Cardene)| • Calcium channel blocker                                                           |                                                                 | • Side effect: Hypotension                                                                                                        |
|                      | • Indication: Hypertension                                                          |                                                                 |                                                                                                                                  |
D. Beta Blockers (the “lol’s”)
   1. General indications are for
      a. AMI: to prevent sudden death (may alter ventricular remodeling)
      b. Tachycardias (ventricular and supraventricular)
      c. Hypertension
   2. Side effects
      a. AV blocks
      b. Sinus bradycardia
      c. Use with caution in Raynaud’s syndrome, COPD, and IDDM

E. Calcium Channel Blockers (verapamil, diltiazem, nifedipine, etc.)
   1. Indications: hypertension, supraventricular arrhythmias
   2. Note that some (nifedipine) are stronger vasodilators; others (verapamil, diltiazem) are stronger AV blockers

F. ACE Inhibitors

Renin–Angiotensin–Aldosterone System

Endocrine RAS

\[
\begin{align*}
\downarrow \text{CO} & \rightarrow \downarrow \text{Renal perfusion} \\
\downarrow & \text{Juxtaglomerular cells release renin} \\
\downarrow & \text{Renin combines with angiotensinogen} \rightarrow \text{Angiotensin I} \\
\downarrow & \text{Angiotensin I + lung-converting enzyme} \rightarrow \text{Angiotensin II} \\
\downarrow & \text{Angiotensin II} \\
\downarrow & \text{Adrenal medulla} \quad \text{Cell growth} \quad \text{Peripheral arterioles} \\
\downarrow & \text{Aldosterone} \\
\downarrow & \text{Vasoconstriction} \\
\downarrow & \text{Na\textsuperscript{+} & H\textsubscript{2}O retention}
\end{align*}
\]
Tissue RAS
a. Exists in many systems, including the cardiac cells
b. Responsible for ventricular remodeling process that occurs following MI or with CHF

⇒ ACE INHIBITORS: Block the conversion of angiotensin I to angiotensin II
   End with “pril” (eg, captopril)
   Major side effects: cough, angioedema, renal insufficiency

⇒ ANGIOTENSIN RECEPTOR BLOCKERS:
   Directly block the All receptors on the cell membrane
   Effects equal to ACEIs

III. Shock
A. Definition: State that develops when there is inadequate tissue perfusion, causing the cells to be deprived of adequate oxygenation, converting to anaerobic metabolism resulting in the production of lactate and acidosis
B. Etiology/Types of Shock
   1. Hypovolemic—blood volume not sufficient to fill the vascular space
   2. Cardiogenic—myocardium unable to pump an adequate CO to maintain tissue perfusion
   3. Obstructive—physical obstruction to flow (eg, dissecting aortic aneurysm, pulmonary embolus)
   4. Distributive—abnormal distribution of intravascular volume; includes septic, anaphylactic, and neurogenic shock)

C. Hemodynamic profiles

<table>
<thead>
<tr>
<th>Type of Shock</th>
<th>Intravascular Volume</th>
<th>Preload</th>
<th>Afterload</th>
<th>Cardiac Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>-----</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Obstructive</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Distributive:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurogenic</td>
<td>-----</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
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<tr>
<td>Anaphylactic</td>
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<td>↓</td>
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<tr>
<td>Septic</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Early</td>
<td>-----</td>
<td>↓</td>
<td>↓</td>
<td>↑ or no change</td>
</tr>
<tr>
<td>Late</td>
<td>No change or ↓</td>
<td>No change, ↑ or ↓</td>
<td>↑</td>
<td>↓</td>
</tr>
</tbody>
</table>
1. Hypovolemic
   a. Impaired tissue perfusion resulting from severely diminished circulating blood volume
   b. Etiology
      ⇒ Hemorrhage (trauma, surgery, burns, severe dehydration)
      ⇒ Internal, extravascular fluid loss (3rd spacing)
      ⇒ Adrenal insufficiency
   c. Clinical picture
      ⇒ Anxious, irritable ⇒ ↓ LOC
      ⇒ Poor capillary refill ⇒ Skin pale and gray
      ⇒ Tachycardia ⇒ Hypotension
      ⇒ Collapsed neck veins ⇒ Tachypnea
      ⇒ ↓ urine output
   d. Labs
      ⇒ ↓ Hct ⇒ Abn electrolytes
      ⇒ Respiratory alkalosis, metabolic acidosis
   e. Management
      ⇒ Volume replacement
      ⇒ Identify and treat the cause

2. Cardiogenic
   a. Definition—myocardium unable to pump an adequate CO to maintain tissue perfusion
   b. Etiology
      ⇒ Most common is loss of >40%-50% viable myocardial tissue
      ⇒ Mechanical problems
         • Perforated intraventricular septum
         • Papillary muscle dysfunction/rupture
         • Myocardial rupture
         • Valvular heart disease
         • Post-op low CO syndrome
⇒ Cardiomyopathies
⇒ Others
  • Hypovolemia        • Metabolic dysfunction
  • Vasomotor dysfunction • Microcirculatory dysfunction
c. Pathophysiology
⇒ Marked decrease in CO: CI = <1.8 L/m/m²
⇒ Usual compensatory response is an increased peripheral vascular resistance
  If not, MAP will fall → ↓ coronary blood flow, worsening the ischemic process
⇒ If the compensatory mechanisms are working:
  ↑ SVR and ↑ catecholamine release
  ↓
  ↑ afterload  ↑ contractility (↑ β stimulation)
  ↓
  ↑ myocardial ischemia
⇒ LVEDV and LVEDP continue to increase → cavity distention → further ↑ing afterload
  • Limits filling of the endocardial vasculature → endocardial ischemia
  • ↑ LVEDP is reflected back into the pulmonary vasculature →
    ↑ pulmonary pressures → development of pulmonary edema →
    development of arterial hypoxemia, contributing to cellular acidosis.
    As pulmonary artery pressures rise, failure of the ischemia and right ventricular occurs.
⇒ Forrester Hemodynamic Subsets in Shock:

<table>
<thead>
<tr>
<th>CI ≥ 2.2 L</th>
<th>CI ≥ 2.2 L</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCWP ≤ 18 mmHg (warm and dry)</td>
<td>PCWP ≥ 18 mmHg (warm and wet)</td>
</tr>
<tr>
<td>CI ≤ 2.2 L</td>
<td>CI ≤ 2.2 L</td>
</tr>
<tr>
<td>PCWP ≤ 18 mmHg (cool and dry)</td>
<td>PCWP ≥ 18 mmHg (cold and wet)</td>
</tr>
</tbody>
</table>
d. Management

⇒ Goals
- Improve oxygen transport
  ⇒ Cardiac output
  ⇒ Oxygen content
    - Hemoglobin
    - Arterial oxygen saturation
- Maintain ventilation
- Maintain/improve nutrition
- Decreased oxygen demand
- Prevent complications

⇒ Pharmacological
- Inotropes
- Vasodilators

⇒ Mechanical support
- Intra-aortic balloon
- Intrapulmonary artery balloon
- Ventricular assist devices
- Extracorporeal membrane oxygenation (ECMO)

⇒ Surgical
- Revascularization
- Transplant

IV. Heart Failure
A. Definition
1. Failure of CO to meet metabolic demands of body
2. Systolic vs diastolic dysfunction
   a. Systolic: problem with contractility
   b. Diastolic: problem with filling

B. Cardiomyopathies
1. Dilated
   a. Causes
      - CAD
      - Viral

   Dilated cardiomyopathy.
• Chemotherapy
• Pregnancy
• Parasitic—Chaga’s disease
• Alcohol

2. Hypertrophic (HOCM)
   a. Causes
      • Aortic stenosis
      • Congenital—IHSS
   b. Management

3. Restrictive
   a. Causes
      • Infiltrative diseases
   b. Management

C. Signs & Symptoms

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<tr>
<th>Left Ventricular Failure:</th>
<th>Forward Failure: ↓ CO</th>
<th>Backward Failure: ↑ LVEDP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right Ventricular Failure:</td>
<td>↑ Venous Pressures</td>
<td></td>
</tr>
</tbody>
</table>
D. Management: Target Goals
1. Improve CO/Cl
   a. Rest
   b. Pharmacologic interventions
      ⇒ Inotropes
      ⇒ Vasodilators to reduce afterload / preload
      ⇒ Beta blockers to prevent sudden death
      ⇒ ACE inhibitors to block ventricular remodeling
      ⇒ Digitalis
      ⇒ Diuretics
2. ECG monitoring (sudden death common; therefore, many have ICDs implanted)
3. Mechanical assist
   a. IABP
   b. LVAD/RVAD
   c. ECMO
4. Prior to d/c: patient and family education

V. Acute Coronary Syndromes
A. Spectrum of Coronary Artery Disease (Atherosclerotic Process) That Includes:
   1. Unstable angina
   2. Non-ST elevation MI
   3. ST elevation MI

VI. Myocardial Infarction: EKG Interpretation: STEMI vs Non–STEMI
A. Current of Ischemia: Primary T-wave Inversion
   B. Current of Injury: ST–segment Elevation
   C. Current of Necrosis: Pathological “q” Wave
### Primary Sites

<table>
<thead>
<tr>
<th>PRIMARY SITE</th>
<th>INDICATIVE CHANGES</th>
<th>RECIPROCAL CHANGES</th>
<th>VESSEL INVOLVED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>Leads II, III, aVF</td>
<td>Leads I, aVL</td>
<td>Right coronary</td>
</tr>
<tr>
<td>Septal</td>
<td>Lead V1-2</td>
<td>Lead V5-6</td>
<td>Left anterior descending</td>
</tr>
<tr>
<td>Anterior</td>
<td>Leads V2, 3, 4</td>
<td>Leads II, III, aVF</td>
<td>Left anterior descending</td>
</tr>
<tr>
<td>Lateral</td>
<td>Leads I, aVL, V5, 6</td>
<td>Leads II, III, aVF</td>
<td>Circumflex</td>
</tr>
<tr>
<td>Posterior</td>
<td>Leads V8, 9</td>
<td>V1,2</td>
<td>Right coronary</td>
</tr>
</tbody>
</table>
Practice EKGs:

#1.
F. Non-STEMI: ST-segment Depression Over Involved Area
   1. Fibrinolytics not effective
   2. ASA and beta blocker should be started within 24 hours of presentation

G. Acute Management
   1. ASA
   2. Beta blocker
   3. Immediate reperfusion
      a. Fibrinolytic
      b. Primary PCI

H. Observe for Acute Closure or Extension
   1. ST-segment changes early indicator
   2. Silent ischemia

I. Discharge Medications
   1. Aspirin
   2. Beta blocker
   3. ACE inhibitor if EF <40%

J. Complications
   1. Heart failure
   2. Cardiogenic shock
   3. Arrhythmias
   4. Mechanical complications
      a. Papillary muscle dysfunction/rupture → acute onset mitral regurgitation
         ⇒ Loud systolic murmur
         ⇒ Falling BP and CO/CI
      b. Cardiac tamponade
         ⇒ Falling BP and CO/CI ⇒ Distended neck veins; ↑ CVP
         ⇒ Narrowing pulse pressure ⇒ Muffled heart tones
         ⇒ Sinus tachycardia ⇒ PEA
         ⇒ Paradoxical pulse ⇒ Equilibration of pressures
      c. Perforated ventricular septum
         ⇒ Falling BP, CO/CI
         ⇒ Loud holosystolic murmur
         ⇒ Insertion of PA catheter; look for oxygen step-up from RA to RV

VII. Valvular Heart Disease
   A. Aortic Valve
      1. Insufficiency: LV volume overload
      2. Stenosis
         a. Dev LVH
         b. Volume-dependent
         c. Onset of a fib can be catastrophic r/t loss of atrial kick
         d. PA pressures elevated
B. Mitral Valve
   1. Insufficiency
      a. Associated with large V wave in PAWP waveform
      b. PAP elevated
   2. Stenosis
      a. PAWP not helpful—falsely elevated
      b. PAP elevated

C. Surgery: Repair vs Replacement
   1. Repair: keep native valve
   2. Replacement: mechanical vs tissue valve
      a. Mechanical: issues r/t chronic anticoagulation Rx
      b. Tissue valves:
         - Porcine
         - Bovine pericardium
         - Homograft
         - Autograft

VIII. Coronary Bypass Surgery
   A. Approaches
      1. Minimally invasive
      2. Sternotomy
   
   B. Use of cardiopulmonary bypass
      1. On pump
      2. Off pump

   C. Postoperative Management
      1. Assess hemodynamic stability
      2. Titrating infusions
      3. Intra-aortic balloon pump
      4. Electrolyte status
         - Hypokalemia
         - Hypomagnesemia
      5. Cardiac arrhythmias
      6. Ventilatory status
         - ABGs
         - Early extubation protocol (if appropriate)
      7. Pain control can be challenging
         - Use of local anesthetics and delivery systems
         - Use of epidurals and PCA pumps
      8. Incisional care
      9. Activity progression
      10. ICU length of stay = 1 day

D. Early Complications
   1. Coagulopathies
   2. Excessive bleeding
   3. Cardiac tamponade
   4. Electrolytes—potassium, magnesium
   5. Respiratory failure/atelectasis
6. Renal insufficiency/acute tubular necrosis
7. Cardiogenic shock
8. Stroke

IX. **Peripheral Arterial Disease:**
   A. **Etiology:** Atherosclerosis – May Have History of Stroke, Coronary Artery Disease and/or hypertension
   
   B. **Signs and Symptoms**
   1. Pain—especially with elevation
   2. Pale, mottled with rubor with dependence of extremity
   3. Ulcers/gangrene
   4. Hair loss, skin is thin and shiny
   5. Weak or absent peripheral pulses
   6. Sluggish capillary refill

   C. Assessing the 6 “Ps”
   1. Pain
   2. Pallor
   3. Paresthesias
   4. Pulselessness
   5. Paralysis
   6. Poikilothermia

   D. **Diagnostic Studies—PAD**
   1. Doppler duplex
   2. Ankle-brachial index (ABI)
      - Used as a screening tool
      - Ankle systolic blood pressure divided by systolic blood pressure in the arm to derive an index
      - ABI scoring
        Normal: 0.9 – 1.3 (pressure normally higher in the ankle)
        ABI <0.9 positive for PAD
        ABI <0.4 indicates severe ischemia
   3. Peripheral angiography

   E. **Management**
   1. **Treatment**
      - Thrombolysis (t-PA)
      - Thrombectomy
      - Percutaneous angioplasty
      - Endovascular stent graft
   2. **Clinical Implications**
      - Assess for palpable pulses
      - Limbs are warm, pink and good capillary refill
      - No signs of bleeding
      - Pain is absent

X. **Aortic Aneurysms**
   A. **Etiologies**
   1. Various diseases
2. Iatrogenic injury—complication of aortic surgery
3. Trauma—severe blunt chest trauma
4. Congenital
   a. Marfan’s syndrome
   b. Coarctation

B. Thoracic Aortic Aneurysm
1. Less common than abdominal aneurysms
2. Types
   a. True: all layers involved
   b. False: partial or complete disruption of aortic wall with blood contained in the adventitial layer
3. Described in terms of shape and location
   a. Shape: fusiform vs saccular
   b. Location: ascending, transverse, or descending
4. Diagnosis
   a. Chest x-ray changes often before S&S
   b. CT scan
   c. Transesophageal echo
5. Signs and symptoms
   a. Ascending aorta: chest pain; AI; CHF
   b. Transverse aorta: dyspnea, stridor, hoarseness, cough, chest pain, JVD (less common)
   c. Descending aorta: back or chest pain
6. Significance: risk for rupture
7. Operative repair:
   a. When:
      - symptomatic
      - size exceeds twice of normal caliber segment -6cm
   b. Post-op assessment determined by site of aneurysm
      • Ascending aorta: often involves AVR
      • Aortic arch: involve flow to brachiocephalic vessels (head, neck and upper extremities)
      • Descending aorta
         - Adequacy of peripheral circulation: spinal cord, SMA, renals, etc.
         - Adequacy of peripheral neuro status
8. Medical management
   a. Focus on controlling and lowering BP
   b. Observe for onset of chest pain or other S&S as appropriate

REFERENCES
Self-assessment Questions

A patient in cardiogenic shock has the following hemodynamic profile:

BP  90/56  HR  110  CO/CI   1.4 / 0.8
PA  36/20  PAWP 18  SVR  3000
RA  10

The following medications are infusing: dobutamine at 10 mcg/Kg/min and epinephrine at 0.02 mcg/kg/min

1. You would be most concerned about:
   a. BP, CO/CI, PA
   b. CO/CI, SVR
   c. BP, SVR, CO/CI, CVP
   d. All of the above

2. Which of the following interventions would be appropriate?
   a. Afterload reduction with sodium nitroprusside
   b. Elevate blood pressure with epinephrine
   c. Reduce preload by giving a diuretic
   d. Improve renal blood flow with dopamine at 10 mcg/Kg/min

3. Indicative changes for acute myocardial infarction include:
   a. tall peaked T wave, ST-segment depression
   b. widened QRS duration > 0.12 sec
   c. T-wave inversion, ST-segment elevation and pathological q wave
   d. Prolonged PR interval

4. Indicative changes for an inferior MI can be found in:
   a. Leads II, III, aVF
   b. V1 – V2
   c. V2 – V3 – V4
   d. V5 – V6, I, aVL

5. Which of the following are signs of hypovolemic shock?
   a. ↓ intravascular volume; ↓ preload; ↑ afterload, ↑ CO
   b. ↓ intravascular volume, ↓ preload; ↓ afterload, ↓ CO
   c. ↓ intravascular volume; ↑ preload; ↑ afterload; ↓ CO
   d. ↓ intravascular volume; ↓ preload; ↑ afterload; ↓ CO

Answers: 1: b, 2: a, 3: c, 4: a, 5: d

Speaker Contact Information:
Barbara “Bobbi” Leeper, MN, RN, CCRN, FAHA
Clinical Nurse Specialist, Cardiovascular Services
Baylor University Medical Center
Dallas, Texas
e-mail: Bobbi.Leeper@baylorhealth.edu