AACN CCRN Review

Multisystem

Presenter: Carol Rauen, RN, MS, PCCN, CCRN, CCNS, CEN
Multisystem

I. Introduction
   A. AACN Blueprint 8%
      • Asphyxia
      • Distributive Shock (e.g., anaphylaxis)
      • Multi-Organ Dysfunction Syndrome (MODS)
      • Multisystem Trauma
      • Septic Shock/Septic Shock
      • System Inflammatory Response Syndrome (SIRS)
      • Toxic Ingestions/Inhalations (e.g., drug/alcohol overdose)
      • Toxin/Drug Exposure

II. Toxic Exposure (Ingestions/Inhalations)
   A. Pathophysiology
      • Absorption
      • Distribution
      • Metabolism
      • Elimination
   B. Assessment
      • ABCs…ALWAYS...ALWAYS...ALWAYS
      • DE and Poison Control
      • Secondary Survey (full assessment)
         • Vital Signs
            • LOC
            • Heart Rate and Rhythm
            • Temperature:
              Hyperthermia - Salicylates and Cocaine
              Hypothermia - Barbiturates and Opiates
            • Respiratory Rate
            • Blood Pressure
         • Full System Assessment
      • History
      • Environment/Bystanders
      • AMPLE: Allergies, Medications, Past Illnesses, Last Meal, Events
      • Diagnostic Work
         • Toxicology Screens: Blood, Urine, Gastric Aspirate
         • CBC, Chemistry, LFTs, Coags, ABG
         • Chest X-Ray, ECG
         • Abd X-Ray (body packing/stuffing)
         • Pregnancy Test
   A. Treatment Options
      • Rapid Response: Unknown Substance, Unconscious Victim
        • Ampule
        • D50 IV: Hypoglycemia
        • Thiamine 100mg IV: Prevent Wernicke-Korakoff’s Syndrome
        • Naloxone 2mg IV, IM or ET: Narcotic Antagonist
      • Antidote When Known and Available (see below)
- Prevent Absorption and Enhance Elimination
  - Oral gastric Lavage
  - Emetics (not recommended)
  - Activated Charcoal
  - Diuresis
  - Whole Bowel Irrigation
  - Hemodialysis

- Don’t Negate Psycho/Social and Family Indications

### B. Common Toxins (Review Complete Toxicology Table in Patho Book or AACN Core Curriculum)

<table>
<thead>
<tr>
<th>Toxin</th>
<th>Problem/Presentation</th>
<th>Antidote</th>
<th>Assessment and Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen</td>
<td>Liver Failure</td>
<td>N-Acetylcysteine (NAC)</td>
<td>N/V, Right UQ Pain, Bleeding Elevating LFTs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Mucomyst) PO, IV</td>
<td>NAC, Gastric Lavage, Charcoal</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Respiratory Depression</td>
<td>No Direct</td>
<td>Altered LOC, ETOH on Breath, Hx</td>
</tr>
<tr>
<td></td>
<td>Seizures</td>
<td>IV Fluids Helpful</td>
<td>Protect Airway, NTG – Lavage (within 1hr)</td>
</tr>
<tr>
<td></td>
<td>Liver Failure (chronic)</td>
<td></td>
<td>ABGs, IV Fluids, Seizure Precautions, Monitor and Tx Electrolyte Imbalance</td>
</tr>
<tr>
<td>Carbon Monoxide</td>
<td>Hypoxia</td>
<td>Removal From Exposure</td>
<td>Altered LOC, Headache, Seizures, Coma, Flu-Like Complaints</td>
</tr>
<tr>
<td></td>
<td>Replaces 0₂ on Hgb</td>
<td>Oxygen Admin</td>
<td>100% Oxygen</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hyperbaric Oxygen</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Stimulates Sympathetic System</td>
<td>None</td>
<td>OD Levels Look Like Hypoxia, Stroke, Head Injuries, MI, Hyperthermia</td>
</tr>
<tr>
<td></td>
<td>HTN, CP, ECG ∆s, Headache, Stroke,</td>
<td></td>
<td>Treat the Physical Presenting Problem (MI, Stroke etc.)</td>
</tr>
<tr>
<td></td>
<td>Seizures, Hyperthermia</td>
<td></td>
<td>Protect Airway – Admin O₂</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Benzodiazepines: Sedation</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Vasodilators: HTN</td>
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<td></td>
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<td></td>
<td>ACS Tx: see Cardiac Section</td>
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<td></td>
<td></td>
<td></td>
<td>Provide Cooling</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Seizure Tx and/or Prophylactic</td>
</tr>
<tr>
<td>Cyclic</td>
<td>CNS: Seizure, Coma</td>
<td>Sodium Bicarbonate</td>
<td>ECG ∆s: Tachy, Vent Dysrhythmias, Heart Blocks, Wide QRS</td>
</tr>
<tr>
<td>Antidepressants</td>
<td>CV: Rhythm Disturbance</td>
<td>Physostigmine (Antilirium)</td>
<td>Hypotension</td>
</tr>
<tr>
<td></td>
<td>Anticholinergic: Decreased Gastric</td>
<td>(Reverses CNS effects)</td>
<td>Altered LOC: Confusion, Agitation, Hallucinations, Seizures, Coma</td>
</tr>
<tr>
<td></td>
<td>Emptying, Urinary Retention</td>
<td></td>
<td>Admin NaBicarb, GI Evacuation: Lavage and Charcoal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Monitor ECG and Tx PRN</td>
</tr>
<tr>
<td>Opiates</td>
<td>Cardiac and Respiratory Depression</td>
<td>Naloxane (Narcan)</td>
<td>Decreased HR, BP, RR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Administer Naloxane</td>
</tr>
</tbody>
</table>
### III. Toxic Exposures (External)

**Chemical Exposure**
- Identify if Possible
- Antidote if Possible
- Remove Chemical
- Brush if Power
- Flush if Liquid- large volumes of NS or H2O
- Cover w Sterile Damp Dressing
- Never Rub Area

### IV. Asphyxia
- Severe oxygen deprivation (hypoxia) secondary to decreased air flow
- Drop in PaO\(_2\)
- Rise PaCO\(_2\)
- Decreased Level of Consciousness
- MODS \(\rightarrow\) Death

**Common Causes**
- Physical Suffocation/Hanging
- Foreign Body/Obstruction in Upper Airway
- Drowning
- Electrical Shock
- Gastric Aspiration
- Smoke or Toxic Gas Inhalation

**Treatment Priorities**
- Open Airway
- Oxygenate and Ventilate
- Monitor
- Lactate Level
- Consider Therapeutic Hypothermia
- Organ Support and Surgery if Required
V. Shock

A. Definitions

<table>
<thead>
<tr>
<th>Clinical Definition for Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>The inability of the circulatory system to supply oxygen and nutrients to the cells of the body.</td>
</tr>
<tr>
<td>The oxygen demands are greater than the supply.</td>
</tr>
</tbody>
</table>

B. Classifications of Shock

Hypovolemic Shock:

1. **Definition:**
   Hypovolemic Shock is the most common type of shock. It also is the easiest to treat if identified early. Shock develops when blood volume is insufficient to fill the intravascular space causing a preload deficit and ultimately a decreased cardiac output.

2. **Cause:**
   Absolute/Direct or Relative/Indirect Loss of Volume

<table>
<thead>
<tr>
<th>Absolute/Direct Losses</th>
</tr>
</thead>
<tbody>
<tr>
<td>External Hemorrhage</td>
</tr>
<tr>
<td>Gastrointestinal Volume Losses</td>
</tr>
<tr>
<td>- Diarrhea</td>
</tr>
<tr>
<td>- Vomited</td>
</tr>
<tr>
<td>- Gastric Suction</td>
</tr>
<tr>
<td>- Ostomies</td>
</tr>
<tr>
<td>Renal Volume Losses</td>
</tr>
<tr>
<td>- Massive Diuresis</td>
</tr>
<tr>
<td>- Hyperglycemic Osmotic Diuresis</td>
</tr>
<tr>
<td>- Diabetes Insipidus</td>
</tr>
<tr>
<td>Plasma Losses</td>
</tr>
<tr>
<td>- Burns</td>
</tr>
<tr>
<td>- Skin Lesions</td>
</tr>
<tr>
<td>- Fistulas</td>
</tr>
<tr>
<td>- Excessive Sweating</td>
</tr>
<tr>
<td>- High Fever</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Relative/Indirect Losses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sequestration of Fluid</td>
</tr>
<tr>
<td>- Cirrhosis</td>
</tr>
<tr>
<td>- Intestinal Obstruction</td>
</tr>
<tr>
<td>- Ileus</td>
</tr>
<tr>
<td>- Peritonitis</td>
</tr>
<tr>
<td>Internal Hemorrhage/Volume Losses</td>
</tr>
<tr>
<td>- Hemotherax</td>
</tr>
<tr>
<td>- Hemorrhagic Pancreatitis</td>
</tr>
<tr>
<td>- Ruptured Spleen</td>
</tr>
<tr>
<td>- Long Bone or Pelvic Fx</td>
</tr>
</tbody>
</table>
3. **Clinical Presentation:**
Patient presentation will depend
1. Percent volume loss
2. Duration of hypovolemia
3. Activation and response of compensatory mechanisms

4. **Therapeutic Goal:**
Restore adequate intravascular volume as quickly as possible and stop losses. The fluid options and crystalloid vs colloid controversy will be addressed in the management section of this seminar.

**Neurogenic Shock**

1. **Definition:**
A loss of vasomotor tone secondary to inhibition of neural output. The loss of sympathetic tone allows the parasympathetic nervous system to dominate, which causes a drop in systemic vascular resistance (massive vasodilation) and bradycardia. Cardiac output drops because of the lack of preload and slow heart rate.

2. **Causes:**
The most common cause of neurogenic shock is spinal cord injury at or above the T6 level. This injury can be complete or incomplete and the shock state typically occurs quickly after the injury and maybe self limiting or transient. The shock state may last up to three weeks.
   - Spinal Cord Injury
   - Deep General Anesthesia
   - Spinal Anesthesia
   - Damage to the Basal Regions of the Brain
   - Prolonged Medullary Ischemia
   - Central Nervous System Problems

3. **Clinical Presentation:**
Parasympathetic dominance is the hallmark of spinal shock. Vasodilation and bradycardia are the classic clinical presentation. During the shock state the patient will typically have no motor or sensory function below the level of the lesion. Long term disability/function cannot be determined until the shock state has subsided.

4. **Therapeutic Goal:**
Stop the initiating cause and stabilize the spine as soon as possible. During the shock state therapies revolve around administering volume (fill the tank), beta stimulation (increase heart rate), and alpha stimulation (vasoconstriction).
Anaphylactic Shock
1. **Definition:**
   Massive vasodilation occurs because of an antigen-antibody reaction which activates mast cells and basophils triggering the release of vasoactive mediators (histamine, serotonin, bradykinin, eosinophil chemotactic factor, prostaglandins, heparin, leukotrienes, platelet-activating factors, adenosine and various proteolytic enzymes) which stimulates a systemic response. This results in tremendous vasodilation and increased capillary permeability, with loss of fluid into the interstitial space and resultant hypotension from the relative hypovolemia.

2. **Cause:**
   The initial activating response can be immunoglobulin E (IgE) or non-IgE mediated. Anaphylaxis is IgE mediated and is typically the result of a specific antigen exposure. An anaphylatoid response is mediated by a non-IgE reaction. There is direct activation of the mediators listed above (not antigen-antibody) from a source. A wide range of agents can cause this response: anti-inflammatory drugs, contrast media, opiates, polysaccharide volume expanders and anesthetics.

### COMMON CAUSES

<table>
<thead>
<tr>
<th>Foods</th>
<th>Antibiotics</th>
<th>Chemotherapy</th>
<th>Other Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eggs</td>
<td>Penicillins</td>
<td>Cisplatin</td>
<td>Protamine</td>
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<tr>
<td>Milk</td>
<td>Cephalosporins</td>
<td>Cyclophosphamide</td>
<td>Thiazides</td>
</tr>
<tr>
<td>Nuts</td>
<td>Tetracyclines</td>
<td>Daunorubicin</td>
<td>Chloropropamid</td>
</tr>
<tr>
<td>Legumes</td>
<td>Erythromycin</td>
<td>Methotrexate</td>
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<tr>
<td>Venoms</td>
<td>NSADs</td>
<td>Insulin</td>
<td>Diagnostic Agents</td>
</tr>
<tr>
<td>Bees</td>
<td>Salicylates</td>
<td>Pork</td>
<td>Iodinated Radiocontrast</td>
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<tr>
<td>Wasps</td>
<td>Buprofen</td>
<td>Beef</td>
<td>Agents</td>
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<tr>
<td>Snakes</td>
<td>Indomethacin</td>
<td>Human</td>
<td></td>
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<td>Spiders</td>
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<tr>
<td>Blood Products</td>
<td>Local Anesthetics</td>
<td>General Anesthetics</td>
<td>Narcotics</td>
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</tbody>
</table>

3. **Clinical Presentation:**
The release of the vasoactive mediators causes an array of systemic effects which lead to decreased oxygen delivery and shock.
- Hypotension
- Generalized Edema (increased capillary permeability)
- Laryngeal Edema
- Severe Bronchoconstriction
- Difficulty Breathing
- Coronary Vasoconstriction
- Urticaria
- Angioedema
- Itching
- Fever
- Flushed or Warm Skin
- Anxiety

4. **Therapeutic Goal:**
Identify and stop the exposure to the causative agent. Block the effects of the vasoactive mediators. Treatment options are typically anti-histamines, vasoconstrictors, bronchodilators, and fluid resuscitation.
Septic Shock

1. **Definition:**
   
   **1992 ACCP/SCCM Definitions**  

   - **Sepsis:** the systemic response to infection, manifested by two or more of the following conditions as a result of infection:
     - Temperature > 38°C or < 36°C
     - Heart Rate > 90 beats per minute
     - Respiratory Rate > 20 bpm or PaCO2 < 32mmHg
     - WBC > 12,000 or < 4,000, or > 10% immature (bands) forms

   - **Systemic Inflammatory Response Syndrome (SIRS):** The systemic inflammatory response to a variety of severe clinical insults. The response is manifested by two or more of the following conditions:
     - Temperature > 38°C or < 36°C
     - Heart Rate > 90 beats per minute
     - Respiratory Rate > 20 bpm or PaCO2 < 32mmHg
     - WBC > 12,000 or < 4,000, or > 10% immature (bands) forms

   - **Severe Sepsis:** Sepsis associated with organ dysfunction, hypoperfusion, or hypotension. Hypoperfusion and perfusion abnormalities may include, but are not limited to:
     - Lactic acidosis
     - Oliguria
     - Acute alteration in mental status

   - **Septic Shock:** Sepsis-induced with hypotension despite adequate fluid resuscitation along with the presence of perfusion abnormalities that may include, but are not limited to:
     - Lactic Acidosis
     - Oliguria
     - Acute alteration in mental status
     
     Patient who is receiving inotropic or vaspressor agents may not be hypotensive at the time that perfusion abnormalities are measured.

   - **MODS:** Multiple Organ Dysfunction Syndrome: the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

2. **2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference**


   - Approved and supported 1992 definitions. Offered SandS for sepsis and staging system (lacks evidence at this time).

   - **General Variables:**
     - Fever (core >38.3C)
     - Hypothermia (core < 36C)
     - Heart Rate > 90min or > 2 SD above the normal value for age
     - Tachypnea
     - Altered mental status
     - Significant edema or positive fluid balance (20ml/kg over 24 hour)
     - Hyperglycemia (plasma glucose >120) in absence of DM
• Inflammatory Variables:
  • Leukocytosis (WBC > 12,000)
  • Leukopenia (WBC < 4,000)
  • Normal WBC with >10% immature forms (bands)
  • Plasma C-Reactive Protein > 2 SD above normal value
  • Plasma Procalcitonin > 2 SD above normal value (IL-6)

• Hemodynamic Variable:
  • Arterial Hypotension (SBP < 90mmHg, MAP < 70, or SBP decreased >40mmHg in adults or < 2SD below normal for age)
  • Sv02 > 70%
  • CI > 3.5L/min

• Organ Dysfunction Variables:
  • Arterial Hypoxemia (PaO2/FiO2 <300)
  • Acute Oliguria (UO < 0.5mL/kg/hr)
  • Creatinine Increase > 0.5mg/dL
  • Coagulation Abnormalities (INR > 1.5 or APT > 60sec)
  • Ileus (absent bowel sounds)
  • Thrombocytopenia (platelet count < 100,000)
  • Hyperbilirubinemia (plasma total bilirubin > 4mg/dL)

• Tissue Perfusion Variables:
  • Hyperlactatemia (>1mmol/L)
  • Decreased capillary refill or mottling

2. Causes:
   Infection is the cause of sepsis. The infective agent can be a bacteria (gram positive or negative), virus or fungi. Once the infection moves from a local to a systemic problem, sepsis and septic shock can result.

3. Clinical Presentation:
   Although initiated by a localized infection, once the patient is septic they present with a systemic inflammatory response. This response is a systemic reaction to the release of endotoxin and biochemical mediators stimulated by inflammation and inadequate oxygen delivery. The patient will present with a relative hypovolemia secondary to massive vasodilation.
   • Relative Hypovolemia and Hypoperfusion
   • Increased Capillary Permeability and Edema
   • Myocardial Depression
   • Lactic Acidosis
   • Pulmonary Capillary Leak Leading to ARDS
   • Activation of Complement System Leading to Microthrombi
   • Platelet Abnormalities
   • Gluconeogenesis and Insulin Resistance
4. **Therapeutic Goal:**
Identify and stop the causative agent. Block the effects of the inflammatory mediators. Treatment options typically include
1. Antibiotics
2. Fluid Resuscitation
3. Vasopressors
4. Ventilation and Oxygenation
5. Restore Hemopoietic Balance.

**Distributive Shock**
Neurogenic, anaphylactic and septic shock is also known as distributive shock because of the relative hypovolemia that occurs in each due to massive vasodilation.

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Septic</th>
<th>Neurogenic</th>
<th>Anaphylactic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Pressure</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>High</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Cardiac Output</td>
<td>High/Low</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Temperature</td>
<td>High</td>
<td>Normal</td>
<td>High</td>
</tr>
<tr>
<td>Skin</td>
<td>Warm/Cold</td>
<td>Warm</td>
<td>Warm</td>
</tr>
<tr>
<td>Respiratory Rate</td>
<td>High</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>WBC</td>
<td>High</td>
<td>Normal</td>
<td>High</td>
</tr>
<tr>
<td>Sympathetic NS</td>
<td>Stimulated</td>
<td>Blocked</td>
<td>Stimulated</td>
</tr>
<tr>
<td>Edema</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Bronchospasm</td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

### Initial Hemodynamic Parameters in Shock States

<table>
<thead>
<tr>
<th>Shock State</th>
<th>HR</th>
<th>BP</th>
<th>CO</th>
<th>PAPs</th>
<th>CVP</th>
<th>SVR</th>
<th>PAOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>High</td>
<td>Low</td>
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<tr>
<td>Cardiogenic</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Neurogenic</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
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<tr>
<td>Anaphylactic</td>
<td>High</td>
<td>Low</td>
<td>High/Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
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<tr>
<td>Septic</td>
<td>High</td>
<td>Low</td>
<td>High/Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
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</table>

### C. Stages of shock

All of the shock states cause hypoperfusion. There is inadequate oxygen supply to the tissue resulting from hypoperfusion, decreased blood pressure, and inadequate cardiac output. A supply/demand imbalance develops and the patient moves into anaerobic metabolism and lactic acidosis. Many physiologic mechanisms in the body delay this occurrence by compensating for the perfusion deficit.

*Rauen and Munro, 1998*

1. **Aerobic vs Anaerobic Metabolism**

2. **Stage 1 – Compensatory Stage**

   As inadequate perfusion persists and significant numbers of cells are affected, an imbalance of oxygen supply and demand occurs. Hypoxemia, hypotension, and acidosis activate the body’s compensatory mechanisms. The physiological goal of compensation is to supply or improve oxygenation and perfusion to the cells.

   - Neural Response
   - Hormonal Response
   - Chemical Response

   **Goal**

   Improve Cardiac Output and Oxygen Delivery

   **Mechanisms**

   - Activated Sympathetic Nervous System
   - Renin/Angiotensin/Aldosterone System
   - Chemoreceptor Stimulated Respiratory Alkalosis

3. **Stage 2 – Decompensatory Stage**

   As shock progresses, the compensatory mechanisms begin to fail. The progression of shock is evident at the cellular, organ, and system levels; and extensive physiological dysfunctions occur. The arteriolar and precapillary sphincters require sufficient energy in the form of adenosine triphosphate (ATP) to maintain a vasoconstrictive state. As energy dissipates with the progression of shock, the sphincters relax, allowing blood to flow into organs and sequester. Sludging of the blood in these capillary beds occurs, and the microcirculation becomes blocked. Metabolic waste products, microaggregates of platelets, white blood cells, and clots accumulate, further enhancing sludging and contributing to the development of metabolic acidosis. In response to these events chemical mediators are released that are harmful to the microcirculation and general system function. This will be reviewed in more detail in the cellular response to shock section.

4. **Stage 3 – Irreversible Stage**

   This is the final stage of shock. It is also referred to as the refractory phase because the body systems are no longer responsive to treatment. As each organ system decompensates and requires more and more support, they reach a point where therapeutic measures are no longer effective in maintaining function. The term irreversible is appropriate because it is at this point when several, if not all, of the systems cross the line from organ dysfunction to organ failure.
D. Cellular Response to Shock

By definition, shock is an imbalance between oxygen supply and demand. The resultant hypoxia and/or ischemia initiate a cascade of tissue, organ, and cellular responses/reactions. These reactions are intended to assist with shock compensation and healing but when left unregulated actually become the source of further chaos.

Organ Failure Mortality

<table>
<thead>
<tr>
<th># Failed Organs</th>
<th>Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single Organ Failure</td>
<td>10%-40%</td>
</tr>
<tr>
<td>Two Organ Failure</td>
<td>41%-67%</td>
</tr>
<tr>
<td>Three Organ Failure</td>
<td>60%-100%</td>
</tr>
<tr>
<td>Four Organ Failure</td>
<td>100%</td>
</tr>
</tbody>
</table>

VI. Summary
AACN CCRN Review

Hematology and Immunology

Presenter: Carol Rauen, RN, MS, PCCN, CCRN, CCNS, CEN
I. Introduction
AACN-CCRN Blueprint 2%
- Coagulopathies (e.g. ITP, DIC, HIT)

Physiology of Hematopoietic System
A. Purpose
- Circulate
- Provide Nutrition
- Provide Oxygen
- Remove Waste Products (carbon dioxide and metabolic wastes)
- Maintain Hemostasis

B. Location
- Veins and Venules 66%
- Pulmonary Loop 12%
- Arteries and Arterioles 11%
- Heart 6%
- Capillaries 5%

C. Composition
- 4-6 liters of blood
- Plasma 55%
- Cellular Components 45%
  - Erythrocytes (red blood cells)
  - Leukocytes (white blood cells)
  - Thrombocytes (platelets)

D. Function and Assessment (see review information at end of this section)
E. Transfusions (see review information at end of this section)

II. Bleeding Disorders
A. Causes for Bleeding
1. Vessel Integrity Disruption
   - Surgical
   - Trauma

2. Platelet Disorders
   - Quantitative
   - Qualitative

3. Coagulation Disorders
   - Acquired
   - Congenital

B. Coagulation Disorders
1. Acquired
   - Malnutrition
   - Liver Dysfunction (decrease synthesis of factors)
   - Vitamin K Deficiency
   - GI Dysfunction (unable to absorb Vit K)
   - Uremia
• Medications (heparin, Coumadin)
• Massive Transfusions
• Consumptive Coagulopathies (DIC)

2. Congenital
• Abnormal Structure or Function of Blood Vessels
  • Rendu-Osler-Weber Disease
• Platelet Coagulation Abnormality
  • Kasabach-Merrit Syndrome
  • vonWillebrand’s Disease
  • Hemophilia A or B
  • Afibrinogenemia
• Hyper-Coagulable Disorders
  • Protein C or S Deficiency

C. DIC - Disseminated Intravascular Coagulation
Definition - DIC is a secondary disorder resulting from a primary pathophysiologic state or disease. It is complex because it presents as an over stimulation of both bleeding and thrombosis. The victim has microvascular thrombi and bleeding occurring simultaneously. The disorder can be life-threatening, acute or chronic and has a mortality rate of 50%-80%. When DIC is a complication of sepsis or shock the mortality rate can be as high as 90%. It frequently is associated with MODS.

Risk Factors - There does not appear to be one common risk factor for this acquired coagulation disorder

<table>
<thead>
<tr>
<th>General Classifications</th>
<th>Primary Event/Disorder</th>
<th>Primary Event/Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue Damage</td>
<td>Major Surgery</td>
<td>Burns</td>
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<tr>
<td></td>
<td>Major Trauma</td>
<td>Transplant Rejection</td>
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<tr>
<td></td>
<td>Heat Stroke</td>
<td>Extracorporeal Circulation</td>
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<td></td>
<td>Head Injury</td>
<td>Snake Bites</td>
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<tr>
<td>Obstetric Complications</td>
<td>HELLP</td>
<td>NS Abortion</td>
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<td></td>
<td>Amniotic Emboli</td>
<td>Eclampsia</td>
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<td></td>
<td>Abruptio Placenta</td>
<td>Placenta Accreta</td>
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<td></td>
<td>Fetal Demise</td>
<td>Placenta Previa</td>
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<tr>
<td>Shock States</td>
<td>Cardiogenic Shock</td>
<td>Massive blood and volume</td>
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<tr>
<td></td>
<td>Septic Shock (severe infection or inflammation)</td>
<td>resuscitation</td>
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<tr>
<td></td>
<td>Hemorrhagic Shock</td>
<td>Drowning</td>
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<tr>
<td></td>
<td>Dissecting Aneurysm</td>
<td>Anaphylaxis</td>
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<tr>
<td>Neoplasms</td>
<td>Acute and Chronic Leukemia</td>
<td>Solid Tumors</td>
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<tr>
<td></td>
<td>Acute and Chronic Lymphoma</td>
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<tr>
<td>Hematologic Disorders</td>
<td>Thrombotic Thrombocytopenic Purpura (TTP)</td>
<td>Collagen Vascular Disorders</td>
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<tr>
<td></td>
<td></td>
<td>Thrombocythemia</td>
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<tr>
<td></td>
<td></td>
<td>Sickle Cell Crisis</td>
</tr>
<tr>
<td>Specific System Dysfunction</td>
<td>Acute and Chronic Renal Dis</td>
<td>Acute Pancreatitis</td>
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<tr>
<td></td>
<td>Ulcerative Colitis</td>
<td>Liver Dysfunction/Failure</td>
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<tr>
<td></td>
<td>DKA, Acid Ingestion</td>
<td>SIRS and MODS</td>
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<tr>
<td></td>
<td>HIV Disease</td>
<td>Pulmonary Embolism</td>
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<tr>
<td></td>
<td>Cirrhosis</td>
<td>Fat Embolism</td>
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</tbody>
</table>

Common Physiologic Response
• Tissue damage
• Platelet damage
• Endothelial damage

Pathophysiology
• Tissue Damage Occurs
• Healing is Stimulated (Clotting)
• Hemopoietic Chaos
• Fibrinolytic Mediators Released
• Initially Microvascular Thrombi
• Consumption Exceeds Synthesis
• Ability to Clot is Lost
• Fibrinolytic Mediators “Run a Muck”
• Lyse all Clots
• Bleeding State
• Consumption Coagulopathy

Physical Assessment and Findings: the primary problem and pre-existing condition certainly play a major role in the presentation. All systems are at risk for dysfunction. The most common problems occur in the pulmonary, renal and hematopoietic systems. Any bleeding patient who does not have a history of or “reason” for to bleed should be suspected of DIC.

<table>
<thead>
<tr>
<th>Laboratory Findings</th>
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<tbody>
<tr>
<td>Test</td>
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<tr>
<td>Hgb</td>
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<tr>
<td>HCT</td>
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<tr>
<td>Platelet Ct</td>
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<tr>
<td>PT</td>
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<tr>
<td>PTT</td>
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<tr>
<td>Fibrinogen</td>
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<tr>
<td>FDP/FSP</td>
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<tr>
<td>D-Dimer</td>
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</tbody>
</table>

Treatment: No definitive treatment exists for DIC. The major goal is to treat primary disorder – stopping the hemapoietic chaos. In addition patient and family emotional support is paramount for quality nursing care.
• Support/Treat the Primary Problem – Eradicate the Cause of DIC
• Early Recognition
• Decrease Bleeding Risk
• Treat Pain
• Transfusion Therapy – PRBC, FFP, Platelets, Cyro
• Vit K
• Anticoagulation Therapy – Heparin
• General Critical Care Management

D. HELLP Syndrome - Hemolysis, Elevated Liver enzymes and Low Platelets
Atypical variant of severe preeclampsia-eclampsia. Presenting with distinct physical and laboratory abnormalities.

Risk Factors:
• Second Trimester → Postpartum
• 70% between 27-37 Weeks Gestation
• Pregnancy-Induced Hypertension
• Older Multiparas
Pathophysiology:
- Preeclampsia: Vasoconstriction, Platelet Aggregation, Altered Thromboxane-to-Prostacyclin Ratio
- Microvascular Injury
- ? Inflammatory Condition of Hepatocytes
- The Physiological Response is Similar to Autoimmune Diseases

Treatment:
- Deliver the Baby
- Control Blood Pressure
  - Hydralazine, Labetalol, Nipride
  - Post Partum Nifedipine
- Hemotherapy
- Assess Liver
- Prevent Seizures: MgSO4
- Dexamethasome
  - Antapartum 10mg IV q12
  - Postpartum 10mg Q12 X2, 5mg q12 X2
- PP Monitor for SandS of MODS
- Future Pregnancies?

E. Heparin Induced Thrombocytopenia (HIT)
- Acquired Allergy to Heparin
- Antibodies are Produced to Heparin
- With Heparin Admin the Antibodies ‘attack’ Heparin and Thrombocytes
- Patient’s Platelet Count Drops: 50% drop from baseline typically between day 4-10 of Heparin Administration
- Treatment is to Stop all Heparin, Admin a Non-Heparin Anticoagulant and Admin Platelets Only if Needed

F. Thrombotic Thrombocytopenic Purpura (TTP)
- Drop in Platelet Ct
- Hemolytic Anemia
- Classically Presents with Neuro Symptoms or Renal Dysfunction and Fever
- Difficult Diagnosis
- Causes: Drugs or BMT, Autoimmune Dis, AIDS, Depressed Bone Marrow, DIC, HIT, Bleeding, Extracorporeal Cir., Medications, Artificial Heart Valve, Hemodilution
- Treatment
  - Stop Cause
  - Admin Platelets or Neumega
  - Plasmapheresis

H. Idiopathic Thrombocytopenic Purpura (ITP)
- Thrombocytopenia < 150,000
- Unable to Determine Cause

III. Summary