Multisystem

Presenter: Carol A. Rauen, RN, MS, CCNS, CCRN, PCCN, CEN
Independent Clinical Nurse Specialist & Education Consultant
rauen.carol104@gmail.com
Multisystem

I. INTRODUCTION

PCCN Test Plan

*Neurological, Multisystem & Behavioral: 15%*

a. Complex Wounds and Pressure Ulcers
b. Healthcare-Acquired Infections
   - Catheter-Associated Urinary Tract Infections (CAUTI)
   - Central-Line-Associated Blood Stream Infections (CLABSI)
c. End of Life (e.g., comfort care measures, hospice)
d. Infectious Diseases
   - Influenza
   - Multi-Drug Resistant Organisms (e.g., MRSA, VRE)
e. Pain
f. Palliative Care
g. Sepsis Continuum
   - Systemic Inflammatory Response Syndrome (SIRS)
   - Sepsis
   - Severe Sepsis
   - Septic Shock
h. Shock States (hypovolemic and anaphylactic)

II. HOLISTIC CARE

Pain

*Definitions*
“*A personal, private sensation of hurt. A harmful stimulus which signals current or impending tissue damage. A pattern of responses to protect the organism from harm.*”  -- *Sternback (1979)*

“*Pain is whatever the experiencing person says it is and exists whenever he/she says it does.*”  
-- *McCaffery (1979)*

*Physiologic Bases for Pain*

a. Somatic Pain
   - Cutaneous or deep skin, bone or muscles
b. Visceral Pain
   - Organs (poorly localized)
c. Referred Pain
   - Left arm pain perceived for chest pain, right shoulder pain perceived for gall bladder pain
d. Neuropathic Pain
   - Injury or damage to nerves (phantom limb pain)
e. Neurotransmitters
   - Acetylcholine
   - Norepinephrine
   - Epinephrine
   - Dopamine

*Types of Pain*

a. Acute
b. Chronic Malignant
c. Chronic Non-Malignant

*Pain Assessment*

*Subjective*

a. Precipitation Factors
b. Aggravating Factors
c. Localization of Pain
d. Character and Quality of Pain
e. Duration of Pain

*Physiological*

Acute Pain - Sympathetic Response

*Measurement Tools*

a. Visual Analog Scales VAS
b. Verbal Descriptors
c. Facial Expressions
d. Oucher Scale

*Principles of Pain Management*

**ABC Principle of Assessment and Planning**

a. Ask about pain regularly, Assess pain systematically
b. Believe the patient and family in their reports of pain and what relieves it.
c. Choose pain control options appropriate for the patient, family, and setting
d. Deliver interventions in a timely, logical and coordinated fashion.
e. Empower patients and their families. Enable them to control their course to the greatest extent possible.
Pharmacology
a. Narcotic
b. Anti-inflammatory
c. Non-Narcotic
d. Adjuvant

Physical Treatments
a. Massage
b. Heat or Cold
c. BioFeedback
d. TENS
e. Elevation

Alternative/Complimentary Therapies
a. Acupuncture
b. Aroma Therapy
c. Therapeutic Touch
d. Prayer
e. Mental Imaging

Palliative Care
A significant and legitimate part of acute care. This is more than just ‘comfort care.’ There are standards and guidelines. We, as the direct care provides are an important member of this team. Our patients and their loved ones depend on us.

End of Life Care

III. WOUNDS AND INFECTIONS

Major Functions of the Skin
a. Barrier From Environment
b. Absorption of Vitamins (Vit D)
c. Temperature Regulation
d. Sensory Perception
e. Shock Absorber
f. Appearance and Identity
g. Assists with Blood Pressure Regulation
Pressure Ulcers

a. Stage I: Skin intact with nonblanchable erythema
b. Stage II: Partial thickness epidermis open, superficial – not depth
c. Stage III: Full thickness through dermis
d. Stage IV: Full thickness through dermis with exposure of underlying structures (muscle, bone)
e. Unstageable: Unable to assess thickness (eschar or covered)
f. Prevention, Assessment and Appropriate Tx are KEY!

Infections

a. Bacterial
b. Viral: Warts, Herpes Zoster
c. Parasite: ex Scabies
d. Fungal: Candidiasis
e. Dermatitis: Contact

Wound Healing

Types

a. Primary Intention: surgical incisions
b. Secondary Intention: heal from inside out by granulation tissue
c. Tertiary Intention: heal from inside out by granulation tissue, typically delayed because of infection, inflammation and/or large open wound. May require skin grafting

Contributing Factors

a. General Health
b. DM
c. Infection
d. Nutrition
e. Activity
f. Age
g. Obesity

Drains

a. Hemovac
b. Jackson-Pratt - JP
c. Penrose
d. Vacuum Assisted Closure - VAC
**Dressings**

a. Dry  
b. Wet-to-Dry  
c. Moisture Retentive – hydrogel  
d. Debriding Agents: Granulex, Zymase, AccuZyme

**Hospital-Acquired Infections**

a. Catheter-Associated Urinary Tract Infections (CAUTI)  
b. Central-Line-Associated Blood Stream Infections (CLABSI)

**IV. SHOCK**

**Definition**

The inability of the circulatory system to supply oxygen and nutrients to the cells of the body. The oxygen demands are greater than the supply.

**Sepsis Continuum**


**Sepsis**

The systemic response to infection, manifested by two or more of the following conditions as a result of infection:

a. Temperature > 38°C or < 36°C  
b. Heart Rate > 90 beats per minute  
c. Respiratory Rate > 20 bpm or PaCO₂ < 32mmHg  
d. 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:

a. Temperature > 38°C or < 36°C  
b. Heart Rate > 90 beats per minute  
c. Respiratory Rate > 20 bpm or PaCO₂ < 32mmHg  
d. 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:

a. Lactic acidosis  
b. Oliguria  
c. 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
Pt who is receiving inotropic or vasopressor agents may not be hypotensive at the time that perfusion abnormalities are measured.

2001 SCCM/ESICM/ACCP/ATS/SIS
International Sepsis Definitions Conference

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

General Variables
- Fever (core >38.3°C)
- Hypothermia (core < 36°C)
- 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 hr)
- 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)
- SvO₂ > 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
Multisystem

d. Coagulation Abnormalities (INR > 1.5 or APT > 60sec)
e. Ileus (absent bowel sounds)
f. Thrombocytopenia (platelet count < 100,000)
g. Hyperbilirubinemia (plasma total bilirubin > 4mg/dL)

Tissue Perfusion Variables
a. Hyperlactatemia (>1mmol/L)
b. Decreased capillary refill or mottling

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.

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.
a. Relative Hypovolemia and Hypoperfusion
b. Increased Capillary Permeability and Edema
c. Myocardial Depression
d. Lactic Acidosis
e. Pulmonary Capillary Leak Leading to ARDS
f. Activation of Complement System Leading to Microthrombi
g. Platelet Abnormalities
h. Gluconeogenesis and Insulin Resistance

Therapeutic Goal
Identify and stop the causative agent. Block the effects of the inflammatory mediators. Treatment options typically include:
a. Antibiotics
b. Fluid Resuscitation
c. Vasopressors
d. Ventilation and Oxygenation
e. Restore Hemopoietic Balance


Hypovolemia Shock
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.
**Cause**

a. Absolute/Direct or Relative/Indirect Loss of Volume  
b. Relative/Indirect Losses  

**Clinical Presentation**

Patient presentation will depend  
a. Percent Volume Loss  
b. Duration of Hypovolemia  
c. Activation and Response of Compensatory Mechanisms  

**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.  

**Anaphylactic Shock**

**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.  

**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.  

**Clinical Presentation**

The release of the vasoactive mediators cause an array of systemic effects which lead to decreased oxygen delivery and shock.  
a. Hypotension  
b. Generalized Edema (increased capillary permeability)  
c. Laryngeal Edema  
d. Severe Bronchoconstriction  
e. Difficulty Breathing  
f. Coronary Vasoconstriction
Multisystem

g. Urticaria
h. Angioedema
i. Itching
j. Fever
k. Flushed or Warm Skin
l. Anxiety

**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.

**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 & Munro, 1998

**Aerobic vs Anaerobic Metabolism**

Aerobic Metabolism:
- \( \text{CO}_2 \)
- \( \text{H}_2\text{O} \)
- 38 ATP

Anaerobic Metabolism:
- 2 ATP
- Lactate
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.
   a. Neural Response
   b. Hormonal Response
   c. Chemical Response

Goal
Improve Cardiac Output and Oxygen Delivery

Mechanisms
   a. Activated Sympathetic Nervous System
   b. Renin/Angiotensin/Aldosterone System

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.

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.