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Shock and Initial Resuscitation

Kristen Sarna, RN, BSN

Critical outcomes

- Assess and identify the type and phase of shock in a presenting patient
- Manage the emergency nursing care of the patient with shock

Specific outcomes

- Predict differential diagnosis when presented with specific information regarding the history of a patient provided by the pre-hospital personnel
- Describe multiple organ dysfunction syndrome (MODS) as complication from shock
- Fluid and blood products:
  - Differentiate between colloids and crystalloids and explain the use for each within the emergency setting
  - Differentiate between the different blood products available and explain use for each within the emergency setting
  - Describe the advantages and disadvantages of the different fluids used during resuscitation

Specific outcomes con't

- Consider age-specific factors in the treatment of shock state
- Apply the medico-legal aspects pertaining to shock emergencies with regard to the emergency nurse
- Apply the above listed knowledge when analyzing a case scenario (paper based and real life scenarios)
- List and know the drugs used in your unit to manage shock
- Delineate the nursing process in the management of the patient with any of the above-mentioned conditions.

General strategy

- Assessment
- Analysis
- Planning and implementation/intervention
- Evaluation and ongoing monitoring
- Documentation of interventions and patient response
- Age-related considerations (pediatric/ geriatric)

What is shock?

- Inadequate tissue perfusion
- Multiple causes, but the pathophysiology is usually the same
- Life threatening
- Imbalance between the supply of and demand of oxygen and nutrients

Assessment of the shock patient

- Monitor vital signs closely
- Monitor mental status
- Monitor lab values
  - ABG with lactate
  - CBC – RBC remain normal, HCT- decreased and HGB- increased
  - Coagulation panel- PT and PTT are prolonged, INR and d dimer are also prolonged (watch for DIC)
  - Troponin, TCK, BUN, Creatinine are elevated
  - Glucose initially elevated, then decreases after glycogen is depleted

Assessment of the shock patient

- Tachypnea -> bradypnea
- Decreased urine output
- Pallor, cool, clammy skin
- Anxiety, confusion, agitation
- Absent bowel sounds

Diagnosing shock

- Based on history and physical assessment
- Elevated lactic and a base deficit
- 12 lead EKG
- Chest x-ray
- Continuous pulse ox

Planning and implementation/ interventions

- Fluid resuscitation
- Administration of blood products
- Monitor bleeding

Evaluation and ongoing monitoring

- Vital signs
- Lab values
- Mental status

Documentation

- Document any and all patient interventions and patient response
- Remember to document all vital signs, watching for subtle changes.
- Mental status
- Strict inputs and outputs

Age related considerations

- Pediatric
  - Increases cardiac output by increasing heart rate
  - Sustains arterial pressure despite significant volume loss
  - Loses 25% of circulating volume before signs of shock occur
- Geriatric
  - Shock progression is rapid
  - Reduced compensatory mechanisms
  - Preexisting disease states contribute to co-morbidities

Stages of shock

- 1. Compensated (nonprogressive) shock
- 2. Uncompensated (progressive) shock
- 3. Irreversible (refractory) shock

Compensated (nonprogressive) shock

- “Reversible stage during which compensatory mechanisms are effective and homeostasis is maintained”
- Clinical presentation begins to reflect the body’s response to the imbalance of oxygen supply and demand

Lewis, Heitkemper, Dirksen, O'Brien, Bucher(2007). Medical Surgical Nursing. St. Louis, MO: Mosby Elsevier

Compensated shock

- At first, blood pressure will decrease, which happens because of the decrease in cardiac output (CO) and a narrowing of the pulse pressure. The baroreceptors in the carotid and aortic bodies immediately respond by activating the sympathetic nervous system (SNS). The SNS stimulates vasoconstriction and release of epinephrine and norepinephrine (potent vasconstrictors)

Lewis, Heitkemper, Dirksen, O'Brien, Bucher(2007). Medical Surgical Nursing. St. Louis, MO: Mosby Elsevier

Compensated shock

- Blood flow to the vital organs, such as the heart and brain, are maintained, while blood flow to non-vital organs, the kidneys, liver, skin, GI tract and the lungs, is shunted.
- Decreased blood flow to the kidneys activates the renin-angiotensin system.
- Renin is released, which activates angiotensinogen to produce angiotensin I, which is then converted to antiotesnsin II.
- Angiotensin II causes vasoconstriction in both the arteries and venous system

Lewis, Heitkemper, Dirksen, O'Brien, Bucher(2007). Medical Surgical Nursing. St. Louis, MO: Mosby Elsevier

Compensated shock

- At this stage, the body is able to compensate for the changes in tissue perfusion. If the underlying cause is corrected, the patient will recover with little to no residual effects.
- If the body is unable to compensate the body will enter the progressive stage of shock

Lewis, Heitkemper, Dirksen, O'Brien, Bucher(2007). Medical Surgical Nursing. St. Louis, MO: Mosby Elsevier

Compensated Shock

- Neurologic
  - Alert and oriented to person, place and time
  - Restless, apprehensive, confused
  - Change in level of consciousness
- Cardiovascular
  - Release of epinephrine/norepinephrine which promotes vasoconstriction
  - ↑contractility
  - ↑heart rate
  - Coronary artery dilation
  - Narrow pulse pressure
  - BP remains adequate to perfuse vital organs

Compensated Shock

- Respiratory
  - ↓blood flow to the lungs
  - hyperventilation
- Gastrointestinal
  - ↓blood supply
  - Hypoactive bowel sounds
- Renal
  - ↓renal blood flow
  - ↑renin resulting in release of angiotensin (vasoconstrictor)
  - ↑aldosterone resulting in sodium and water re-absorption
  - ↑ antidiuretic hormone resulting in water re-absorption

Compensated Shock

- Hepatic
  - No changes at this stage
- Hematologic
  - No changes at this stage
- Temperature
  - Normal to abnormal
- Skin
  - Pale and cool
  - Warm and flushed (early septic shock)

Compensated Shock

- Key laboratory findings
  - ↑blood glucose
  - ↑pH
  - ↓PaO_2
  - ↓$PaCO_2$

Uncompensated (progressive) shock

- This stage of shock begins when the body's compensatory mechanisms fail
- Aggressive interventions are need to prevent the development of multiple organ dysfunction syndrome (MODS)
- Continued decreased cellular perfusion and resulting alerted capillary permeability are the distinguishing features of this stage

Uncompensated shock

- Altered capillary permiability allows leakage of fluid and protein out of the vascular space into the surrounding interstitial space causing a decrease in circulating volume and an increase in systemic interstitial edema.
- This fluid leak from the vascular space also affects the solid organs, liver, spleen, GI tract, lungs, and peripheral tissues by further decreasing oxygen perfusion

Lewis, Heitkemper, Dirksen, O'Brien, Bucher(2007). Medical Surgical Nursing. St. Louis, MO: Mosby Elsevier

Uncompensated Shock

- Neurologic
  - ↓cerebral perfusion pressure
  - ↓ cerebral blood flow
  - Listless or agitated
  - ↓responsiveness to stimuli

Uncompensated Shock

- Cardiovascular
  - ↑capillary permeability → systemic interstitial edema
  - ↓ cardiac output = ↓BP and ↑HR
  - MAP <60mmHG
  - ↓Peripheral perfusion
    - Ischemia of distal extremities
    - Diminished pulses
    - ↓capillary refill
  - ↓Coronary perfusion resulting in
    - Dysrhythmias
    - Myocardial ischemia
    - Myocardial infarction
    - Myocardial dysfunction → impaired cardiac output

Uncompensated Shock

- Respiratory
  - Acute respiratory distress syndrome (ARDS)
    - ↑capillary permeability
    - Pulmonary vasoconstriction
    - Pulmonary interstitial edema
    - Alveolar edema
    - Diffuse infiltrates
    - ↑ respiratory rate
    - ↓ compliance
  - Moist crackles

Uncompensated Shock

- Gastrointestinal
  - Vasoconstriction and decreased perfusion lead to ischemic gut (stomach, small and large intestines, gallbladder and pancreas)
  - Erosive ulcers
  - GI bleeding
  - Translocation of GI bacteria
  - Impaired absorption of nutrients

Uncompensated Shock

- Renal
  - Renal tubules become ischemic causing acute tubular necrosis
  - ↓urine output
  - ↑BUN/creatinine ratio
  - ↑urine sodium
  - ↓Urine osmolarity and specific gravity
  - ↓urine potassium
  - Metabolic acidosis

Uncompensated Shock

- Hepatic
  - Failure to metabolize drugs and waste products
  - Jaundice
  - Increase in lactate and ammonia
- Hematologic
  - DIC
    - Thrombin clots in microcirculation
    - Consumption of clots in microcirculation

Uncompensated Shock

- Temperature
  - Hypothermia
  - Sepsis: hyper or hypothermia
- Skin
  - Cold and clammy
- Key laboratory findings
  - ↑ liver enzymes: ALT, AST, GGT
  - ↑ bleeding times
  - thrombocytopenia

Irreversible (refractory) Shock

- Final stage of shock
- Decreased perfusion from peripheral vasoconstriction and decreased cardiac output exacerbate anaerobic metabolism
- Lactic acid accumulates and contributes to an increased capillary permeability and dilation of the capillaries
- Increased capillary permeability allows for fluid and plasma to leave the vascular space and move to the interstitial space

Lewis, Heitkemper, Dirksen, O'Brien, Bucher(2007). Medical Surgical Nursing. St. Louis, MO: Mosby Elsevier

Irreversible Shock

- Blood pools in the capillary beds secondary to constricted veins and dilated arteries
- Loss of intravascular volume leads to worsening of hypotension and tachycardia resulting in a decrease in coronary blood flow
- Decreased coronary blood flow results in decreased cardiac output
- Cerebral blood flow cannot be maintained and cerebral ischemia results

Lewis, Heitkemper, Dirksen, O'Brien, Bucher(2007). Medical Surgical Nursing. St. Louis, MO: Mosby Elsevier

Irreversible Shock

- Neurologic
  - Unresponsive
  - Arreflexia
  - Pupils nonreactive and dilated
- Cardiovascular
  - Profound hypotension
  - ↓ cardiac output
  - Bradycardia, irregular rhythm
  - Unable to perfuse vital organs
- Respiratory
  - Severe hypoxemia
  - Respiratory failure

Irreversible Shock

- Gastrointestinal
  - Ischemic gut
- Renal
  - anuria
- Hepatic
  - Metabolic changes from accumulation of waste products (ammonia, lactate, carbon dioxide)
- Hematologic
  - DIC

Irreversible Shock

- Temperature
  - hypothermic
- Skin
  - Mottled, cyanotic
- Key laboratory findings
  - ↓ blood glucose
  - ↑ ammonia, lactate and potassium
  - Metabolic acidosis

Fluid administration

- **Crystalloids:** increase intravascular volume through actual volume administered
- **Colloids:** pull fluid into the vascular space through osmosis

Fluid administration

- **Isotonic:** similar in composition to body fluid. Provides greater intravascular volume d/t more fluid staying in the vascular space

- **Hypotonic fluid**: shift fluid into intracellular spaces. Useful in preventing cellular dehydration. They deplete circulatory volume

- **Hypertonic:** move fluid from cells to extravascular space, may be used to replace electrolytes and promote diuresis

Fluid administration: crystalloids

- 0.9% Normal saline: Isotonic fluid
- 0.45% Normal Saline: hypotonic
- 5% Dextrose: hypotonic
- Lactated Ringer: Isotonic
- Hypertonic Saline (7.5%): hypertonic, pulls fluid from interstitial and intracelluar spaces into the vascular space

Fluid administration: Colloids

- Dextran →
- Hetastarch →

Rarely used. Used to expand vascular space.

- Fresh frozen plasma
- Albumin
- Whole blood
- Packed red blood cells

Blood products (natural colloids)

- Fresh frozen plasma: contains all clotting factors. Used as a blood volume expander
- Albumin: preferred as volume expander when risk from producing interstitial edema is great (pulmonary and heart disease)

Blood products (natural colloids)

- Packed Red blood cell's: Administer with normal saline
  - Increases oxygen affinity for hgb, and decrease oxygen delivery to the tissues
  - May cause: hypothermia, hyperkalemia, or hypocalcemia
- Whole blood: can be administered without normal saline, reduces donor exposure
  - May require greater amt than packed RBC's to increase oxygen-carrying capacity of blood
  - Not cost effective. Rarely used

Fluid administration

- Pediatric fluid guidelines
  - Up to 10 kg = 4ml/kg/hr
  - 11-20kg = 2ml/kg/hr plus 4ml/kg for first 10kg
  - >20kg = 1ml/kg/hr plus 2ml/kg for each kg 11 through 20 plus 4ml/kg for first 10 kg
- Volume replacement with crystalloids
  - Administer 2 ml for each ml lost
  - Pediatric: IV bolus of 20ml/kg of NS or LR
  - IV bolus of 200-300 ml NS in adults

Fluid administration: reassessment

- Monitor for fluid overload: continuous pulse ox, and other vital signs (HR, BP, RR)
- Monitor for electrolyte imbalances

Specific Shock Emergencies

- Hypovolemic Shock
- Cardiogenic Shock
- Distributive Shock
- Obstructive Shock

Hypovolemic Shock

- Loss or redistribution of blood, plasma, or other body fluids, which results in a decreased circulatory volume
- Inadequate fluid returning to the heart results in decreased cardiac output
- Third spacing occurs due to capillary permeability
- Example: hemorrhagic shock from trauma, intraabdominal bleeding, significant vaginal bleeding, GI bleeding or vomiting and diarrhea

Hypovolemic Shock

- Increased heart rate
- Decreased pulse pressure
- Decreased blood pressure

Hypovolemic Shock

- Cardiovascular
  - ↓ preload, stroke volume
  - ↓ capillary refill
- Pulmonary
  - Tachypnea → bradypnea (late sign)
- Renal
  - ↓ urine output
- Skin
  - Pallor
  - Cool, clammy

Hypovolemic Shock

- Neurologic
  - Anxiety
  - Confusion
  - agitation
- Gastrointestinal
  - Absent bowel sounds
- Diagnostic findings
  - ↓ hematocrit
  - ↓ hemaglobin
  - ↑ lactate
  - ↑ urine specific gravity
  - Changes in electrolytes

Hypovolemic Shock

- Treatment:
  - Correcting the underlying cause
  - Warm fluids
  - May need supportive therapy with vasopressors

Cardiogenic Shock

- Occurs when the heart fails as a pump resulting in significant reduction in ventricular effectiveness
- When pump failure occurs, the myocardium cannot forcibly eject blood
- Stroke volume decreases d/t decreased contractility, which decreases cardiac output and blood pressure, resulting in decreased tissue perfusion
- Decreased oxygenation to heart further complicates patient condition

Cardiogenic Shock

- Causes of Cardiogenic Shock include:
  - Myocardial infarction
  - Cardiomyopathy
  - Pericardial tamponade
  - Dysrhythmias
  - Trauma
  - Structural abnormalities
    - Valvular abnormality
    - Ventricular septal rupture
    - Tension pneumothorax

Cardiogenic Shock

- Increased heart rate
- Decreased pulse pressure
- Decreased blood pressure

Cardiogenic Shock

- Cardiovascular
  - Decreased capillary refill
  - May have chest pain
- Pulmonary
  - Tachypnea
  - Cyanosis
  - Crackles
  - rhonchi
- Skin
  - Pallor
  - Cool, clammy

Cardiogenic Shock

- Renal
  - ↑ sodium and water retention
  - ↓ renal blood flow
  - ↓ urine output
- Neurologic
  - ↓ cerebral perfusion
    - Anxiety
    - Confusion
    - agitation
- Gastrointestinal
  - ↓ bowel sounds
  - Nausea/vomiting

Cardiogenic Shock

- Diagnostic findings
  - ↑ cardiac markers
  - ↑ blood glucose
  - ↑ BUN
  - Dysrhythmias
  - Pulmonary infiltrates on chest x-ray
  - Left ventricular dysfunction on echocardiogram

Cardiogenic Shock

- Treatment:
  - Correct dysrhythmias
  - Drug Therapy:
    - Nitrates
    - Inotropes
    - Diuretics
    - Beta blockers

Distributive Shock

- Neurogenic Shock
- Anaphylactic Shock
- Septic Shock

Neurogenic Shock

- Results from spinal cord trauma (usually T5 or above) or spinal anesthesia
- Injury results in major vasodilation without compensation due to loss of sympathetic nervous system vasoconstrictor tone
- Major vasodilation leads to pooling of blood in the blood vessels, tissue hypoperfusion and ultimately impaired cellular metabolism

Neurogenic Shock

- Spinal anesthesia can block transmission of impulses from the SNS resulting in neurogenic shock
- Signs/symptoms
  - Hypotension
  - Bradycardia
  - Inability to regulate temperature

Neurogenic Shock

- Cardiovascular
  - ↑/↓ Temperature
  - Bradycardia
- Pulmonary
  - Dysfunction r/t level of injury
- Renal
  - Bladder dysfunction
- Skin
  - ↓ skin perfusion
  - Cool or warm
  - dry

Neurogenic Shock

- Neurologic
  - Flaccid paralysis below the level of the lesion/injury
  - Loss of reflex activity
- Gastrointestinal
  - Bowel dysfunction
- Diagnostic findings
  - history

Neurogenic Shock

- Treatment:
  - High dose steroids: to help decrease inflammation surrounding spinal cord
  - Treat the symptoms

Anaphylactic Shock

- Acute and life-threatening allergic reaction to a sensitizing substance
- Immediate response causing massive vasodilation, release of vasoactive mediators, and an increase in capillary permeablity
- Can lead to respiratory distress d/t laryngeal edema or severe bronchospasm, and circulatory failure d/t vasodilation

Anaphylactic Shock

- Sudden onset of symptoms
  - Chest pain
  - Dizziness
  - Incontinence
  - Swelling of lips and tongue
  - Wheezing and stridor
  - Flushing, pruritis, urticaria
  - Angioedema
  - Anxious and confused

Anaphylactic Shock

- Cardiovascular
  - Chest pain
  - Third spacing of fluid
- Pulmonary
  - Swelling to tongue and lips
  - Shortness of breath
  - Edema of larynx and epiglottis
  - Wheezing
  - Rhinitis
  - stridor

Anaphylactic Shock

- Renal
  - Decreased urine output
- Skin
  - Flushing
  - Pruritus
  - Urticaria
  - angioedema
- Neurologic
  - Anxiety
  - Decreased LOC

Anaphylactic Shock

- Gastrointestinal
  - Cramping
  - Abdominal pain
  - Nausea
  - Vomiting
  - Diarrhea
- Diagnostic findings
  - Sudden onset
  - History of allergens
  - Exposure to contrast media

Anaphylactic Shock

- Treatment:
  - Airway management
  - Epi 0.3mg SQ or IM to vastus lateralis
  - BLS/ACLS

Septic Shock

- Sepsis: systemic inflammatory response to a documented or suspected infection
- Septic Shock: presence of sepsis with hypotension despite fluid resuscitation along with the presence of tissue perfusion abnormalities.

Septic Shock

- The body responds through both hyper-inflammatory and anti-inflammatory means. Endotoxins released by the invading organisms prompt release of hydrolytic enzymes from weakened cell lysosomes, which causes cellular destruction of bacteria and normal cells
- When the body is unable to control the proinflammatory mediators, it produces a systemic inflammatory response

Septic Shock

- As a result, there is widespread cellular dysfunction to the endothelium, resulting in vasodilation, increased capillary permeability, and platelet aggregation and adhesions to the endothelium

Septic Shock

- Cardiovascular
  - ↑/↓ Temperature
  - Biventricular dilation
    - ↓ ejection fraction
- Pulmonary
  - Hyperventilation
  - Respiratory alkalosis then respiratory acidosis
  - Hypoxemia
  - Respiratory failure
  - ARDS
  - Pulmonary hypertension
  - crackles

Septic Shock

- Renal
  - Decreased urine output
- Skin
  - Warm and flushed then cool and mottled
- Neurologic
  - Alteration in mental status
  - Confusion
  - Agitation
  - coma
- Gastrointestinal
  - GI bleeding

Septic Shock

- Diagnostic findings
  - ↑/↓ WBC
  - ↓ Platelets
  - ↑ Lactate
  - ↑ Glucose
  - ↑ Urine specific gravity
  - ↓ Urine sodium
  - *positive blood cultures*

Septic Shock

- Treatment
  - Fluid administration
  - Monitor VS
  - Antibiotics: broad spectrum until source is identified
  - Vasopressors