Multi-Organ Dysfunction Syndrome
Lesson Description – Mitch Taylor

- At the completion of this lesson, the student will be able to:
  - Describe shock.
  - Classify shock into 3 major classifications.
    - Hypovolemic
    - Cardiogenic
    - Distributive
Multi-Organ Dysfunction Syndrome

- Identify 3 stages of shock and the physiologic changes and the nursing implications resulting from each stage.
- Apply nursing interventions to each type of shock.
- Discuss pharmacological treatment for shock.
- Describe Multiple Organ Dysfunction Syndrome.
- Distinguish how the priority nursing diagnosis of altered tissue perfusion relates to each system involved.
Multiple Organ Dysfunction Syndrome

- **SIRS** (Systemic Inflammatory Response Syndrome) can be diagnosed when two or more of the following are present:
  - Heart rate > 90 beats per minute
  - Body temperature < 36°C (96.8°F) or > 38°C (100.4°F)
  - Tachypnea (high respiratory rate) > 20 breaths per minute or, on blood gas, a PaCO2 < 32 mm Hg
  - White blood cell count < 4000 cells/mm³ or > 12000 cells/mm³, or the presence of greater than 10% immature neutrophils.

- **SIRS + proof of infection (positive blood cultures) = sepsis**

- It is this uncontrolled inflammatory response that is the problem. Remember the picture.
Multi-Organ Dysfunction Syndrome

The term Multiple System Failure or Multiple Organ Dysfunction Syndrome is the failure of two or more systems in the body. And shock is a term that describes the process the generally leads to MODS.

Shock Definition – as failure of the circulatory system to maintain adequate perfusion of vital organs.

Ineffective tissue perfusion is the main problem with all types of shock. The tissues are not being perfused, the body reacts in a crisis mode, and then tissues and organs begin to fail.

The cascade of these complex effects is what we label as shock.
Inadequate perfusion
  ↓
Cell hypoxia
  ↓
Energy deficit
  ↓
Lactic acid accumulation and fall in pH
  ↓
Anaerobic metabolism
  ←
Metabolic acidosis

Vasoconstriction
  ↓
Failure of pre-capillary sphincters
  ↓
Peripheral pooling of blood
  ↓
Cell membrane dysfunction and failure of ‘sodium pump’
  ↓
Intracellular lysosomes release digestive enzymes
  ↓
Toxic substances enter circulation
  ↓
Capillary endothelium damaged
  ↓
Further destruction, dysfunction and cell death

Efflux of potassium

Influx of sodium and water
Shock

- Three Types of Shock:
  - Hypovolemic – low blood volume
  - Cardiogenic - bad pump or can’t get the blood out
    - low cardiac output
    - obstruction of the vascular system
  - Distributive (Vasogenic) – changes in vessel tone
    - Anaphylactic
    - Neurogenic
    - Septic (Concept Map of Septic Shock).
Hypovolemic Shock – low blood volume

- The main problem with this is a large reduction in circulating blood volume so that the body’s metabolic needs cannot be met. Examples of causes include:
  - Burns/Large Traumas – fluid shifts out of the vascular space.
  - Dehydration – fluid loss or inadequate intake.
Cardiogenic Shock — the flow of blood is diminished to the tissues.

- Low Cardiac Output due to:
  - Myocardial Infarction (heart attack)
  - Blunt trauma to the chest
  - Open Heart Surgery
  - Heart Failure
  - Dysrhythmias

- The pump is not working well. The patient will have a low cardiac output, where the blood is not being pumped out well enough to perfuse the organs and tissues.
Cardiogenic Shock – the flow of blood is diminished to the tissues.

- Obstructive conditions include:
  - Pulmonary Embolism
  - Pericardial Tamponade
  - Tension Pneumothorax
  - Aneurysms and Valve Dysfunction

- These represent mechanical obstruction from the blood flow either to the heart or from the heart.

- The tissues do not get perfusion because something is blocking the way.
Distributive (Vasogenic) Shock – changes in vessel tone

- Distributive Shock results from inadequate vascular tone. Blood volume remains normal, but the size of the vascular space increases dramatically because of vasodilation.

- The blood is poorly distributed because the blood pressure drops and blood pools in the vessels because the vessels are dilated.
Distributive (Vasogenic) Shock – changes in vessel tone

- Anaphylactic Shock results from an acute allergic reaction from exposure to a substance that the patient has been sensitized.
  - PCN
  - Bee Stings
  - Peanuts, etc.
Distributive (Vasogenic) Shock – changes in vessel tone

- The body is exposed to a substance and then develops antibodies to that substance. When a person is re-exposed to the substance again, then it sets off a cascade of events.

- Manifestations include:
  - Vasodilation
  - Urticaria (hives)
  - Laryngeal edema
  - Bronchial constriction
Distributive (Vasogenic) Shock – changes in vessel tone

- Neurogenic Shock – Spinal Chord Injury
  - Sympathetic (gas system)
  - Parasympathetic (brake system)
  - Below the level of injury, the sympathetic nervous system is blocked, causing the parasympathetic system to go unopposed.

- Producing:
  - Vasodilation – drop in BP
  - Decreased Venous Return
  - Decreased Cardiac Output – drop in HR
  - Decreased Tissue Perfusion
Distributive (Vasogenic) Shock – changes in vessel tone

- **Septic Shock** (Concept Map of Septic Shock). Sepsis is the systemic response to infection.
- Microorganisms invade the blood stream and release endotoxins that produce systemic changes in the body.
  - Producing:
    - Capillary Vasodilation
    - Poor tissue perfusion – producing lactic acid
    - Low Cardiac Output
    - Vascular thrombi
    - Pulmonary Vasoconstriction
    - Peripheral Vasoconstriction
Three Stages of Shock

- **Nonprogressive Stage**
  - Decreased cardiac output due to low volume or relative low volume
  - The body responds and compensates by:
    - Sympathetic stimulation (releases epinephrine and norepinephrine)
    - The body then responds by vasoconstriction and compensation for loss of capillary blood flow
    - The body shunts fluid to the vascular space by increasing systemic vascular resistance (SVR-afterload).
    - Maintaining BP (with tachycardia)
Stages of Shock

- Progressive Stage
  - The persistent compensatory vasoconstriction continues.
    - Producing decreased venous return and the beginning of anaerobic metabolism because of inadequate tissue perfusion.
    - The anaerobic metabolism produces lactic acid.
    - The lactic acid tells the body to prepare for crisis mode, so the vessels become permeable to allow fluid to leak out of the vessels and into the tissues.
Progressive Stage (cont.)

- The capillaries where exchange takes place between the cells get the alarm and begin enlarging and pooling blood as a saving measure.
- But, this decreases venous return, decreases cardiac output, and the body can’t fill the vessels and the enlarged capillaries.
- The tissues become hypoxic and begin to die.
Stages of Shock

- **Irreversible Stage** – The cycle in the progressive stage continues and becomes worse leading to organ failure and death.

- **Effects of Shock on:**
  - The respiratory system – increase RR to keep up with tissue demand (compensatory mechanism) produces respiratory alkalosis as CO2 is blown off.
  - Acid-Base Balance – The lack of oxygen produces more anaerobic metabolism producing lactic acidosis.
  - Lyosomal enzymes released during cell death are activated in acid and then damages more cells.
Stages of Shock

Effects of Shock on:

- The cardiovascular system:
  - The effects of poor tissue perfusion releases a polypeptide known as Myocardial Depressant Factor causing a:
    - decrease in cardiac output,
    - low blood pressure,
    - tachycardia,
    - dysrhythmias (the cells do not fully repolarize)
Stages of Shock

- Effects of Shock on:
  - DIC (Disseminated Intravascular Coagulation):
    - Acidic blood is sluggish blood and when cell destruction releases clot initiating factors, then the body begins to form multiple clots in the vessels interrupting and damaging circulation.
    - The body attempts to reverse this by breaking down these clots, and it goes overboard affecting all clotting, producing bleeding.
Stages of Shock

Effects of Shock on:

GI tract:

- Blood is shunted away from the GI tract with sympathetic stimulation to provide more for the heart and the brain.
- With Shock this is a prolonged effect and the intestines are much more susceptible to poor perfusion and tissue necrosis.
- When this occurs the liver which normally detoxifies substances is overwhelmed from endotoxins from the intestinal tract.
Stages of Shock

Effects of Shock on:

Kidneys:

- Over time renal ischemia occurs and the body becomes unable to maintain the fluid-electrolyte balance and urine output decreases and renal failure follows.
Clinically Seen

- Low BP, decreased Cardiac Output
  - Tachycardia - to compensate

- Metabolic acidosis (may have resp. compensation)
  - Increased RR (compensatory mechanism)

- Decreased peripheral circulation
  - increased SVR (to compensate)

- Decreased circulation to GI tract, kidneys, and liver

- Potential for systemic clotting/bleeding

- Multi-organ collapse
2008 Guidelines for Management of Severe Sepsis and Septic Shock

Evidence of under resuscitation, i.e., hypotension, oliguria, or elevated serum lactate 4 mmol/L

- Aggressively volume resuscitate with crystalloids/colloids
- Insert Arterial line, CVP, Central Venous sat (SVO2) monitor

Goals:
- CVP = 8-12 mm Hg
- MAP ≥ 65 mm Hg
- Urine Output ≥ 0.5 mL/kg/hr
- SVO2 ≥ 70%

If on mechanical ventilation, then consider fluids until CVP 12-15 mmHg

Fluid boluses should be given 1000 mL of crystalloids or 300-500 mL of colloids over 30 minutes. May slow rate of administration if cardiac filling times are reduced or history of heart failure.

If MAP still < 65, then initial vasopressors can be added. Either dopamine 5-20 mcg/kg/min or norepinephrine 2-30 mcg/min (increase slowly) usually 2-12 mcg/min IV infusions to increase BP.

If SVO2 < 70%, then consider dobutamine infusion 5-20 mcg/kg/min

Begin IV antibiotics as soon as possible and within first hour of recognizing septic shock. Obtain 2 or more blood cultures and other sites if needed prior to starting antibiotics. Finding the site of infection is extremely important.

Hypovolemic Shock

- Treat the underlying cause: if bleeding-stop the bleeding, if a burn-stop the burning, if heat exhaustion-get the patient cooled off, if vomiting-get the vomiting to stop, if diabetic ketoacidosis-get the blood sugars under control.

- Fluid therapy

- Crystalloids – like NS or LRs. Colloids are controversial, now because Albumin or other hypertonic solutions may leak out of the vessels into the pulmonary interstitial spaces pulling fluid with it.

- Blood Products such as
  - Packed red cells – for blood replacement
  - Fresh frozen plasma for returning coagulation factors/proteins
Cardiogenic Shock

- **Treat the underlying cause:** if MI, stop the progression with MONA up to and including reperfusion strategies like TPA or PTCA to reopen the vessels, if aneurysm or valve disease or pulmonary embolism, may need to control symptoms until surgery can be performed.

- **Generally a Pulmonary Artery Catheter will need to be placed to monitor preload (PAWP), contractility (cardiac output), and afterload (systemic vascular resistance SVR).**
  - Treat with inotropics such as dopamine and dobutamine.
  - May have to treat with vasodilators such as Nitroprusside to reduce Systemic Vascular Resistance (unless BP too low).
  - Treat with pressors to increase BP such as Norepinephrine or Vasopressin.
  - Intra-Aortic Balloon Pump may be inserted to help relieve the workload on the heart, beta-blockers used to be used for this as well, but more evidence shows it to be a relative contraindication if the blood pressure is low.
  - Diuretics may be used if patient shows signs of pulmonary congestion (crackles)
Distributive Shock

- Treat the underlying cause: if anaphylactic shock stop the offending agent, then treat with a histamine antagonist like Benadryl (diphenhydromine), Epinephrine 1:1000 SQ or IM, Steroids, vasopressors, and IV fluid resuscitation, if neurogenic or vasovagal-stop the movement, lower the head, treat bradycardias with Atropine, low BP with IV fluids and vasopressors if needed.

- Septic Shock – need to treat the underlying cause, so blood, urine, sputum cultures all need to be done before antibiotics are given. Also if a patient has been on antibiotics, then Clostridium Difficile (C. Diff.) may have overgrown and be causing the problem.
Septic Shock

- Antibiotics
- Aggressive IV fluid resuscitation
- SVO2 monitoring (central venous monitoring of oxygen saturations) will change based on consumption within the body, so it is a good measure of tissue demand and thus an early predictor of sepsis. Want to keep > 70%.
- Xigris is the first FDA-approved therapy with a proven ability to increase survival in adult patients with high-risk severe sepsis.
Multiple Organ Dysfunction Syndrome

Medical Management involves scoring then treating:

- APACHE II was designed to measure the severity of disease for adult patients admitted to Intensive care units.
- 24 hours after admission to the ICU, the measurement has been completed and resulted in an integer point score between 0 and 71. No new score can be calculated during the stay. If a patient is discharged from the ICU and readmitted, a new APACHE II score can be calculated.
- Some procedures and some medicine is only given to patients with certain APACHE II score
- APACHE II score can be used to describe the morbidity of a patient when comparing the outcome with other patients.

Predicted mortalities are averaged for groups of patients in order to specify the group's morbidity.

Even though newer scoring systems, like SAPS II and III have replaced APACHE II in many places, APACHE II continues to be used extensively because so much documentation is based on it.

http://www.sfar.org/scores2/apache22.html - website
How Xigris works

- Xigris reduces microvascular dysfunction by reducing inflammation and coagulation, and increasing fibrinolysis.
- Run Xigris 24 mcg/kg/hr for 96 hours. Stop if have any signs of bleeding or if surgery is required.
- Xigris is very expensive, it is about $1700 per day to run for 96 hours = $6800 (www.xigris.com – cost listed by manufacturer-Lily)
Inadequate perfusion
  ↓
Cell hypoxia
  ↓
Energy deficit
  ↓
Lactic acid accumulation and fall in pH
  → Anaerobic metabolism
  ↓
Metabolic acidosis
  ←

Vasoconstriction
  ↓
Failure of pre-capillary sphincters
  ↓
Peripheral pooling of blood
  ↓

Cell membrane dysfunction and failure of ‘sodium pump’
  ↓
Intracellular lysosomes release digestive enzymes
  ↓
Toxic substances enter circulation
  ↓
Capillary endothelium damaged
  ↓
Further destruction, dysfunction and cell death

Efflux of potassium
  ↓
Influx of sodium and water
Nursing Management

- Involves treating the affected systems, monitoring the affected systems, and supporting those systems with an emphasis on airway, oxygenation, blood pressure, temperature regulation, maintaining nutrition, and urine output.
- Management of Ineffective Tissue Perfusion is primary for every system.
- Our identification of problems early on is the key to patient survival.
CRITICAL THINKING: CASE STUDY

As a new graduate has been asked to place a feeding tube (Dobhoff) and start tube feeding on a new trauma patient who is a busy patient. He/she complains to you because he/she has many other more critical things to do.

Case Study Questions

What is your rationale to explain to the new graduate why starting tube feeding is important?
Discussion Questions

- What are the different types of shock?
- If someone has a heart attack, which type of shock would that be?
- Position the patient flat on the bed if BP is low and start what drips to increase cardiac output?
- All types of shock start a process of anaerobic metabolism that because of poor what?
- Where does the body shunt the blood during this process? What systems are the body trying to save?
- When on drugs that cause vasoconstriction, monitor what systems and how?
- Why is a GI surgery patient at so much more risk for infection?