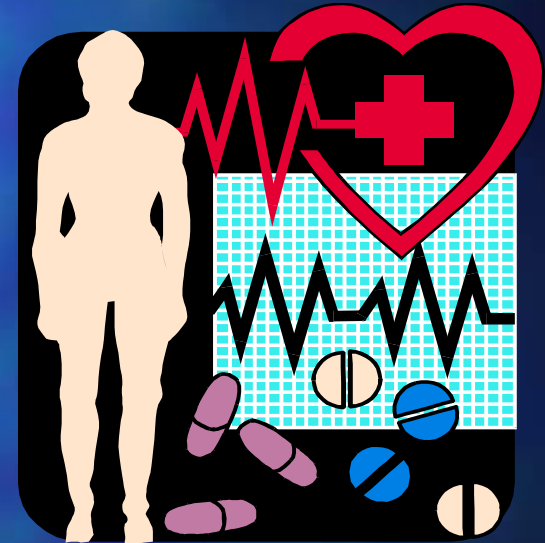


Pediatric Shock

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Introduction

- Shock is a syndrome that results from inadequate oxygen delivery to meet metabolic demands
- Shock = Demand > Supply
- Untreated this leads to metabolic acidosis, organ dysfunction and death



Oxygen Delivery

- Oxygen delivery = Cardiac Output x Arterial Oxygen Content
($DO_2 = CO \times CaO_2$)
- Cardiac Output = Heart Rate x Stroke Volume
($CO = HR \times SV$)
 - SV determined by preload, afterload and contractility
- Art Oxygen Content = Oxygen content of the RBC + the oxygen dissolved in plasma
($CaO_2 = Hb \times SaO_2 \times 1.34 + (.003 \times PaO_2)$)



Stages of Shock

■ **Compensated**

- Vital organ function maintained, BP remains normal.

■ **Uncompensated**

- Microvascular perfusion becomes marginal. Organ and cellular function deteriorate. Hypotension develops.

■ **Irreversible**

Shock

- Shock result from inadequate
 - **Blood flow**
 - **Oxygen delivery**
- Shock progresses over a continuum of severity
 - Compensated
 - Decompensate

Compensated shock

- Tachycardia
- Cool extremities
- Prolonged capillary refill
- Weak peripheral pulses compared with central pulses
- Normal blood pressure

Decompensate shock

- Depressed mental status
- Decreased urine output
- Metabolic acidosis
- Tachypnea
- Weak central pulses
- Hypotension

Clinical Presentation



- Early diagnosis requires a high index of suspicion
- Diagnosis is made through the physical examination focused on tissue perfusion
- Abject hypotension is a late and premonitory sign



Differential Diagnosis of Shock

■ Hypovolemic

- Hemorrhage
- Serum/Plasma loss

■ Distributive

- Anaphylactic
- Neurogenic
- Septic

■ Cardiogenic

- Myocardial
- Dysrhythmia
- CHD-(duct dependant)

■ Obstructive

- Pneumo,
Tamponade,
Dissection

■ Dissociative

- Heat, CO, Cyanide
- Endocrine

Neonate in Shock:

Include in differential:

- Congenital adrenal hyperplasia
- Inborn errors of metabolism
- Obstructive left sided cardiac lesions:
 - Aortic stenosis
 - Hypoplastic left heart syndrome
 - Coarctation of the aorta



Management-General

- Goal: increase oxygen delivery and decrease oxygen demand:
 - Oxygen
 - Fluid
 - Temperature control
 - Antibiotics
 - Correct metabolic abnormalities
 - Inotropes

Management-General (con't)

■ Airway

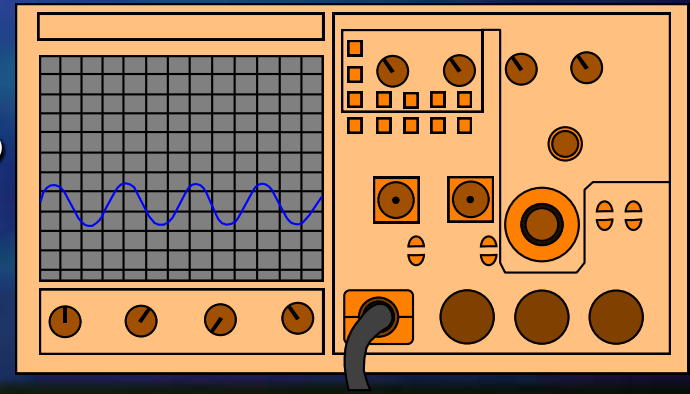
- If not protected or unable to be maintained, intubate.

■ Breathing

- Always give 100% oxygen to start
- Sat monitor

■ Circulation

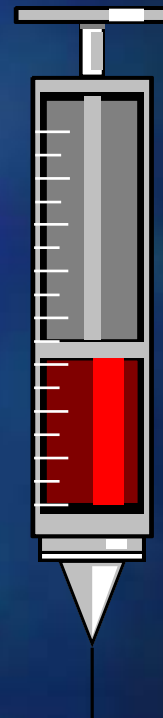
- Establish IV access rapidly
- CR monitor and frequent BP



Management-General (con't)

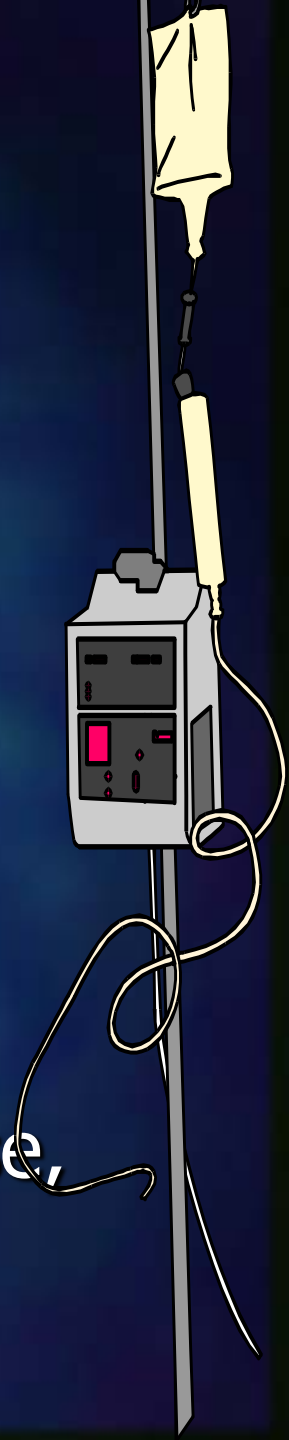
■ Laboratory studies:

- ABG
- Blood sugar
- Electrolytes
- CBC
- PT/PTT
- Type and cross
- Cultures



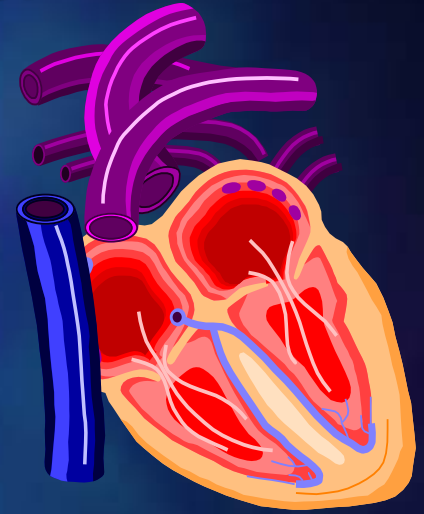
Management-Volume Expansion

- Optimize preload
- NS or RL
- Except for myocardial failure use 10-20cc/kg aliquots q 2-10 minutes
- At 40-60cc/kg reassess and consider: ongoing losses, adrenal, intestinal ischemia, obstructive shock. Get CXR. Consider colloid
- Further fluid therapy guided by response, labs, possibly CVP, CXR



Management - Cardiotonics I

- Lack of history of fluid losses, history of heart disease, hepatomegaly, rales, cardiomegaly and failure to improve perfusion with adequate oxygenation, ventilation, heart rate, and volume expansion suggests a cardiogenic or distributive component.



Management - Cardiotonics II

■ **Epinephrine**

- 0.05-1.5 ug/kg/min
- increase HR, SVR, contractility
- End point: adequate BP; acceptable tachycardia

■ **Norepinephrine**

- 0.05-1.0 ug/kg/min
- Increase SVR
- End point: adequate BP

■ **Dopamine**

- 2-20 ug/kg/min
- Lower doses, increases renal and splanchnic blood flow, & contractility. Higher doses increases HR and SVR
- End Point: Improved perfusion, BP, Urine

Management - Cardiotonics III

■ Dobutamine

- 1-20 ug/kg/min
- increases contractility, may reduce SVR, PVR
- End Point: Improved perfusion, may decrease BP

■ Prostaglandin E-1

- 0.05-0.1 ug/kg/min
- maintains patency of ductus



Hypovolemic Shock

- Most common form of shock world-wide
- Results in decreased circulating blood volume, decrease in preload, decreased stroke volume and resultant decrease in C.O.
- Etiology: Hemorrhage, renal and/or GI fluid losses, capillary leak syndromes

Hypovolemic Shock

- Clinically, history of vomiting/diarrhea or trauma/blood loss
- Signs of dehydration: mucous membranes, tears, skin turgor
- Hypotension, tachycardia without signs of congestive heart failure

Hypovolemic/Hemorrhagic Shock: Therapy

- Always begin with ABCs
- Replace circulating blood volume rapidly: start with crystalloid/colloid
- Blood products as soon as available for hemorrhagic shock (Type and Cross with first blood draw)
- Replace ongoing fluid/blood losses & treat the underlying cause

Septic Shock: “Warm Shock”

- Early, compensated, hyperdynamic state
- Clinical signs
 - Warm extremities with bounding pulses, tachycardia, tachypnea, confusion.
- Physiologic parameters
 - widened pulse pressure, increased cardiac output and mixed venous saturation, decreased systemic vascular resistance.
- Biochemical evidence:
 - Hypocarbia, elevated lactate, hyperglycemia

Septic Shock: "Cold Shock"

- Late, uncompensated stage with drop in cardiac output.
- Clinical signs
 - Cyanosis, cold and clammy skin, rapid, thready pulses, shallow respirations.
- Physiologic parameters
 - Decreased mixed venous sats, cardiac output and CVP, increased SVR, thrombocytopenia, oliguria, myocardial dysfunction, capillary leak
- Biochemical abnormalities
 - Metabolic acidosis, hypoxia, coagulopathy, hypoglycemia.

Septic Shock (con't)

- Cold Shock rapidly progresses to MOSF or death, if untreated
- Multi-Organ System Failure: Coma, ARDS, CHF, Renal Failure, Ileus or GI hemorrhage, DIC
- More organ systems involved, worse the prognosis
- Therapy: ABCs, fluid
- Appropriate antibiotics, treatment of underlying cause

Cardiogenic Shock

■ Etiology:

- Dysrhythmias
- Infection
- Metabolic
- Obstructive
- Drug intoxication
- Congenital heart disease
- Trauma

Cardiogenic Shock (con't)

- Differentiation from other types of shock:
 - History
 - PE:
 - Enlarged liver
 - Gallop rhythm
 - Murmur
 - Rales
 - CXR:
 - Enlarged heart, pulmonary venous congestion

Cardiogenic Shock (con't)

■ Management:

- Improve cardiac output::
 - Correct dysrhythmias
 - Optimize preload
 - Improve contractility
 - Reduce afterload
- Minimize cardiac work:
 - Maintain normal temperature
 - Sedation
 - Intubation and mechanical ventilation
 - Correct anemia

Distributive Shock

- Due to an abnormality in vascular tone leading to peripheral pooling of blood with a relative hypovolemia.
- Etiology
 - Anaphylaxis
 - Drug toxicity
 - Neurologic injury
 - Early sepsis
- Management
 - Fluid
 - Treat underlying cause

Obstructive Shock

- Mechanical obstruction to ventricular outflow
- Etiology: CHD, massive PE, tension pneumothorax, cardiac tamponade
- Inadequate C.O. in the face of adequate preload and contractility
- Tamponade: Narrow pulse pressure and/or EMD
- Treat underlying cause.

Dissociative Shock

- Inability of Hemoglobin molecule to give up the oxygen to tissues
- Etiology: Carbon Monoxide poisoning, methemoglobinemia, dyshemoglobinemias
- Tissue perfusion is adequate, but oxygen release to tissue is abnormal
- Early recognition and treatment of the cause is main therapy

Final Thoughts

- Recognize compensated shock quickly- have a high index of suspicion, remember tachycardia is first sign. Hypotension is late and ominous.
- Vein access quickly- if necessary use an IO line.
- Administer adequate amounts of fluid rapidly. Remember ongoing losses.
- Correct electrolytes and glucose problems quickly.
- If the patient is not responding the way you think he should, broaden your differential, think about different types of shock.