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SHOCK IN PEDIATRICS

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- *SHOCK* is an acute process characterized by the significant systemic reduction in tissue ***perfusion*** 
- body's inability to deliver adequate oxygen to meet the metabolic demands of vital organs and tissues
- normal aerobic cellular metabolism: shift to less efficient anaerobic metabolism

Shock stages

Compensated Shock

- complex set of responses attempt to preserve oxygenation of the vital organs (brain,heart, kidneys, liver) at the expense of other organs (skin, gastrointestinal tract, muscles)
- brain is especially sensitive to periods of poor oxygen supply given its lack of capacity for anaerobic metabolism
- **SBP** is maintained within the **normal** range
- HR is initially increased

Compensatory mechanisms

- increasing CO- SV- HR
- Increasing systemic vascular resistance (SVR)
- Respiratory : greater (CO_2) elimination in response to the metabolic acidosis and increased CO_2 production from poor tissue perfusion
- Renal : excretion of (H^+) and retention of (HCO_3^-)
- Maintenance of intravascular volume : sodium regulation through the renin-angiotensin-aldosterone and atrial natriuretic factor axes, cortisol - catecholamine synthesis and release, antidiuretic hormone secretion
- Increasing oxygen extraction
- Redistributing blood flow to the brain, heart, and kidneys

Decompensated shock

If treatment is not initiated or is inadequate **decompensated shock** develops with hypotension and tissue damage

Compensatory mechanisms are overwhelmed

HR is markedly elevated and hypotension develops

Signs of organ dysfunction (such as altered mental status) appear

SBP falls

- *Uncompensated* state requiring more aggressive therapies to achieve clinical recovery

Irreversible Shock

- Untreated shock causes irreversible tissue and organ injury (**irreversible shock**) and ultimately, death

Progressive end-organ dysfunction leads to irreversible organ damage and death. Tachycardia may be replaced by **bradycardia** and blood pressure becomes **very low.**

The process is often irreversible, despite resuscitative efforts.

- Of patients who do not survive, most do not die in the acute hypotensive phase of shock, but rather as a result of associated complications and **multiple-organ dysfunction syndrome (MODS)**

Types of Shock

- **HYPOVOLEMIC**
- Decreased preload secondary to internal or external losses
- **Blood loss**: hemorrhage
- **Plasma loss**: burns, nephrotic syndrome
- ***Water/electrolyte loss***: vomiting, diarrhea

- **CARDIOGENIC**
- ***Cardiac pump failure*** secondary to poor myocardial function
- Congenital heart disease
- Cardiomyopathies: infectious or acquired, dilated or restrictive
- Ischemia
- Arrhythmias

- **DISTRIBUTIVE**
- Abnormalities of ***vasomotor tone*** from loss of venous and arterial capacitance
- Neurologic: loss of sympathetic vascular tone secondary to spinal cord or brainstem injury
- Sepsis, hypoxia, poisoning, anaphylaxis, spinal cord injury, mitochondrial dysfunction

- **SEPTIC**
- Encompasses multiple forms of shock
- Hypovolemic: third spacing of fluids into the extracellular, interstitial space
- Distributive: early shock with decreased afterload
- Cardiogenic: depression of myocardial function by endotoxins
- Bacterial ,Viral , Fungal
- immunocompromised patients are at increased risk

- **OBSTRUCTIVE**
- Decreased cardiac output secondary to direct impediment to right- or left-sided heart outflow or restriction of all cardiac chambers tension
- pneumothorax
- Pericardial tamponade
- Pulmonary embolism
- Anterior mediastinal masses
- Critical coarctation of aorta

- **Dissociative**
- Inadequate oxygen releasing
- Profound anaemia
- Carbon monoxide poisoning
- methemoglobinemia

Pathophysiology of Shock

- Extracorporeal Fluid Loss
- Lowering Plasma Oncotic Forces
- Abnormal Vasodilation
- Increased Vascular Permeability
- Cardiac Dysfunction

Signs and Symptoms

- ***Central nervous system***:
- Restless, apathetic, anxious Agitated/confused, stuporous,coma
- ***Respiration*** : ↑ Ventilation
- ***Metabolism*** : Compensated metabolic Acidemia _Uncompensated metabolic acidemia

Signs and Symptoms

- ***Gut*** : ↓ Motility - Ileus
- ***Kidney*** : ↓ Urine volume- ↑ Urinary specific gravity
- Oliguria (<0.5 mL/kg/hr)
- Oliguria/anuria
- ***Skin*** : Delayed capillary refill - Cool extremities - Mottled, cyanotic, cold
- ***Cardiovascular system :***
- ↑ Heart rate
- ↑↑ Heart rate ↓ Peripheral pulses
- ↑↑ Heart rate ↓ Blood pressure- central pulses only

- *Shock may initially manifest as* **only tachycardia**, *with or without tachypnea.*

- Significant misconception : shock occures only with low BP

- hypotension is often a ***late*** finding and is not a criterion for the diagnosis of shock because of a complex set of compensatory mechanisms that attempt to preserve BP and peripheral perfusion
- ***Hypotension*** reflects an ***advanced state*** of decompensated shock and is associated with increased morbidity and mortality

- **Hypovolemic shock** :
- orthostatic hypotension
- dry mucous membranes
- dry axillae
- poor skin turgor
- decreased urine output
- cool distal Extremities
- pulses : normal, decreased, or absent
- SVR ↑ CO ↓ MAP ↓ CVP ↓

- **cardiogenic shock :**
- tachypnea
- cool extremities
- delayed capillary filling time
- poor peripheral and/or central pulses
- declining mental status
- decreased urine output
- decreased cardiac output
- peripheral vasoconstriction
- SVR ↑ CO ↓ MAP ↓ CVP ↑

- **Obstructive shock :**
- inadequate cardiac output because of a physical restriction of forward blood flow
- May quickly progress to cardiac arrest
- SVR ↑ CO ↓ MAP ↓ CVP ↑

- **Distributive shock :**
- peripheral vasodilation and increased but inadequate cardiac output
- SVR ↓ CO ↑ MAP ↓ CVP ↓

- ***Septic shock***
- Massive vasodilation , capillary leak , signs of reduced end organ perfusion
- DURING THE EARLY STAGE OF SEPTIC SHOCK: ***warm skin, tachycardia , widened pulse pressure : warm shock***
- Normal or high CO and decreased SVR
- Early : SVR ↓ CO ↑ MAP ↓ CVP ↓
- Late : SVR ↓ CO ↓ MAP ↓ CVP ↑

- **SIRS :** Two of 4 criteria : T>38.5°C or <36°C , Tachycardia , Tachypnea , Leukocyte count
- **Sepsis** : SIRS resulting from a suspected or proven infectios etiology
- **severe sepsis** : presence of sepsis combined with organ dysfunction : Cardiovascular / ARDA or sepsis plus >= 2 organ disfunctions

- **septic shock :** severe sepsis plus the persistence of hypoperfusion or hypotension despite adequate fluid resuscitation or a requirement for vasoactive agents

- **Multiple-Organ Dysfunction Syndrome (MODS) :** Presence of altered organ function such that homeostasis cannot be maintained without medical intervention.

- Additional clinical findings in shock :
- cutaneous lesions such as petechiae, diffuse erythema, ecchymoses, ecthyma gangrenosum,peripheral gangrene
- Jaundice can be present either as a sign of infection or as a result of MODS

Laboratory Findings

- ***hematologic*** :
- thrombocytopenia, prolonged PT, PTT , reduced fibrinogen, elevation of fibrin split products, anemia
- Elevated neutrophil, increased immature forms (bands,myelocytes, promyelocytes), vacuolation of neutrophils, toxic granulations, Döhle bodies, Neutropenia or leukopenia

- ***electrolyte*** disturbances :
- hyperglycemia or hypoglycemia , hypocalcemia, hypoalbuminemia, metabolic acidosis.

- Renal and/or hepatic function abnormalities
- **ARDS** or pneumonia
- impairment of **oxygenation** (decreased PaO2) (increased PaCO2)

- ***hallmark of uncompensated shock*** : imbalance between oxygen delivery (DO2) and oxygen consumption (VO2)
- Oxygen delivery normally exceeds oxygen consumption by threefold. The oxygen extraction ratio is approximately 25%, thus producing a normal mixed venous oxygen saturation (ScvO2) of approximately 75%

- lactic acid production ,high anion gap, metabolic acidosis

- *Elevated blood* **lactate** *levels reflect poor tissue oxygen delivery noted in all forms of shock.*

Treatment

- Early recognition and prompt intervention
- Stabilization of Airway
- Breathing
- Circulation

NEWBORNS

0 min

Recognize decreased perfusion, cyanosis, RDS.
Maintain airway and establish access according to NRP guidelines.

5 min

Push 10 mL/kg isotonic crystalloid or colloid boluses to 40 mL/kg until improved perfusion or unless hepatomegaly.
Correct hypoglycemia and hypocalcemia. Begin antibiotics.
Begin prostaglandin infusion until r/o ductal-dependent lesion.

15 min

Fluid refractory shock?

Infuse Dopamine (< 10 µg/kg/min) ± Dobutamine

Fluid refractory-dopamine resistant shock?

Titrate Epinephrine 0.05–0.3 µg/kg/min

60 min

Catecholamine-resistant shock?

ATTAIN
Normal MAP-CVP, $ScvO_2$ > 70%, SVC flow > 40 mL/kg/min or CI > 3.3 L/min/m^2

Cold Shock
Normal Blood Pressure
Poor LV function
$ScvO_2$ < 70%* /Hgb > 12g/dL
SVC flow < 40 mL/kg/min or CI < 3.3 L/min/m^2?

Add Nitrosovasodilator
Milrinone/Imrinone
With volume loading

Cold Shock
Poor LV function
PPHN
$ScvO_2$ < 70%*
SVC flow < 40 mL/kg/min or CI < 3.3 L/min/m^2?

Inhaled Nitric Oxide
Inhaled Iloprost/IV Adenosine
IV milrinone/amrinone

Low Blood Pressure
Warm Shock

Titrate Volume
Add Norepinephrine
?Vaso/Terlipressin
?Angiotensin
Keep $ScvO_2$ > 70%,
SVC flow > 40 mL/kg/min, or CI > 3.3 L/min/m^2 with inotropic support

Refractory shock?

Evacuate pneumothoraces and pericardial effusion. Give Hydrocortisone if Absolute Adrenal Insufficiency and T_3 if Hypothyroid. Begin Pentoxyfylline if VLBW newborn. Consider Closing PDA if hemodynamically significant

ECMO

CRITICAL CARE MEDICINE

0 min

INFANTS/CHILDREN

Recognize decreased mental status and perfusion.
Begin high flow O_2 and establish IO/IV access according to PALS.

5 min

If no hepatomegaly or rales/crackles then push 20 mL/kg isotonic saline boluses and reassess after each bolus up to 60 mL/kg until improved perfusion. Stop for rales, crackles or hepatomegaly. Correct hypoglycemia and hypocalcemia. Begin antibiotics.

15 min

Fluid refractory shock?

Begin peripheral IV/IO inotrope infusion, preferably Epinephrine 0.05–0.3 µg/kg/min
Use Atropine/Ketamine IV/IO/IM if needed for Central Vein or Airway Access

Titrate Epinephrine 0.05–0.3 µg/kg/min for Cold Shock.
(Titrate central Dopamine 5–9 µg/kg/min if Epinephrine not available)
Titrate central Norepinephrine from 0.05 µg/kg/min and upward to reverse Warm Shock.
(Titrate central Dopamine ≥ 10 µg/kg/min if Norepinephrine not available)

60 min

Catecholamine-resistant shock?

If at risk for Absolute Adrenal Insufficiency consider Hydrocortisone.
Use Doppler US, PICCO, FATD or PAC to Direct Fluid, Inotrope, Vasopressor, Vasodilators
Goal is normal MAP-CVP, $ScvO_2$ > 70* and CI 3.3–6.0 L/min/m^2

Normal Blood Pressure
Cold Shock
$ScvO_2$ < 70%* /Hgb > 10g/dL
on Epinephrine?

Low Blood Pressure
Cold Shock
$ScvO_2$ < 70%* /Hgb > 10g/dL
on Epinephrine?

Low Blood Pressure
Warm Shock
$ScvO_2$ < 70%*
on Norepinephrine?

Begin Milrinone infusion.
Add Nitroso-vasodilator if CI < 3.3 L/min/m^2 with High SVRI and/or poor skin perfusion.
Consider Levosimendan if unsuccessful.

Add Norepinephrine to Epinephrine to attain normal diastolic blood pressure. If CI < 3.3 L/min/m^2 add Dobutamine, Enoximone, Levosimendan, or Milrinone.

If euvolemic, add Vasopressin, Terlipressin, or Angiotensin. But, if CI decreases below 3.3 L/min/m^2 add Epinephrine, Dobutamine, Enoximone, Levosimendan.

Persistent Catecholamine-resistant shock?

Evaluate Pericardial Effusion or Pneumothorax,
Maintain IAP < 12 mmHg

Refractory shock?

ECMO

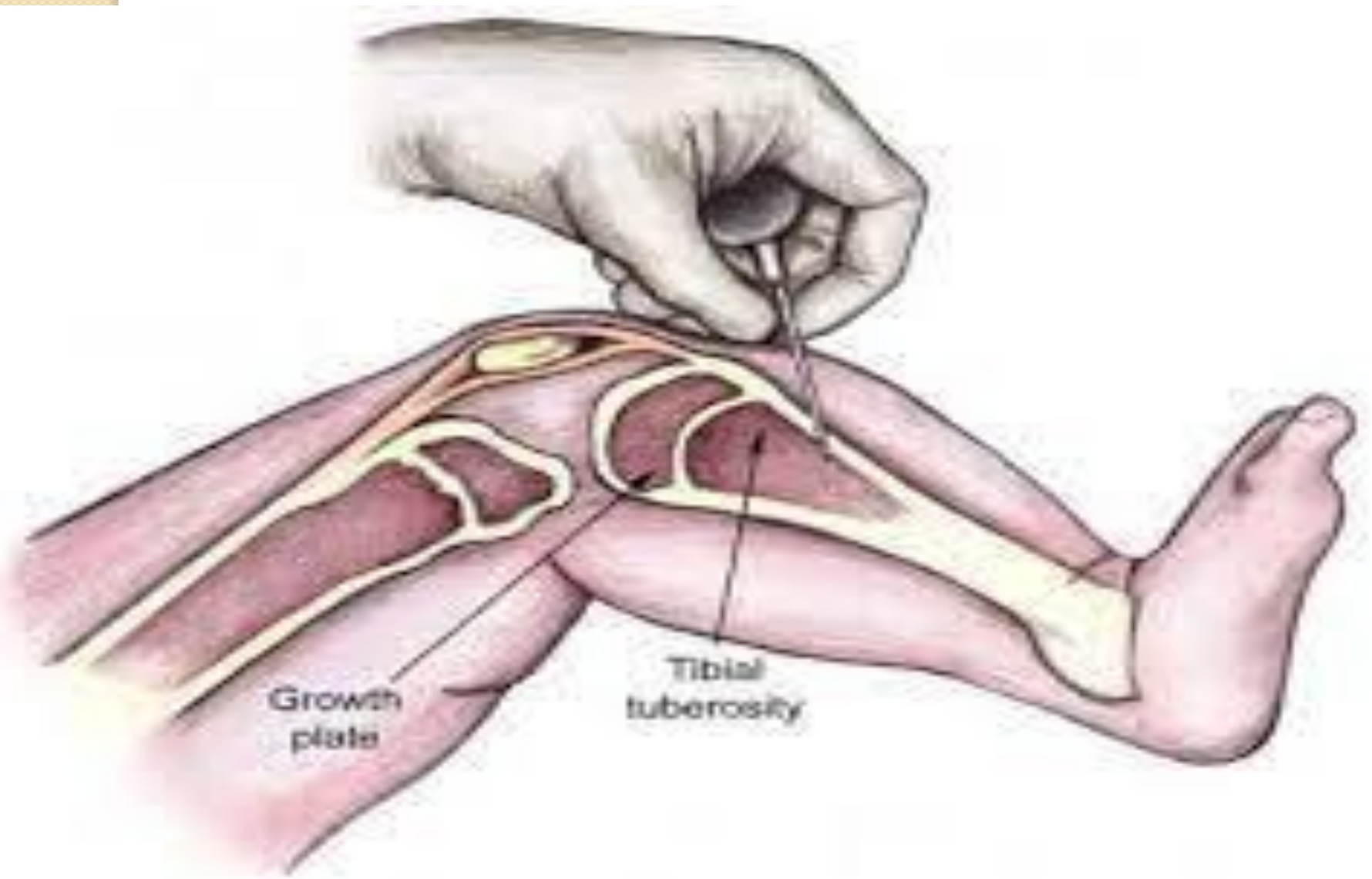
CRITICAL CARE MEDICINE

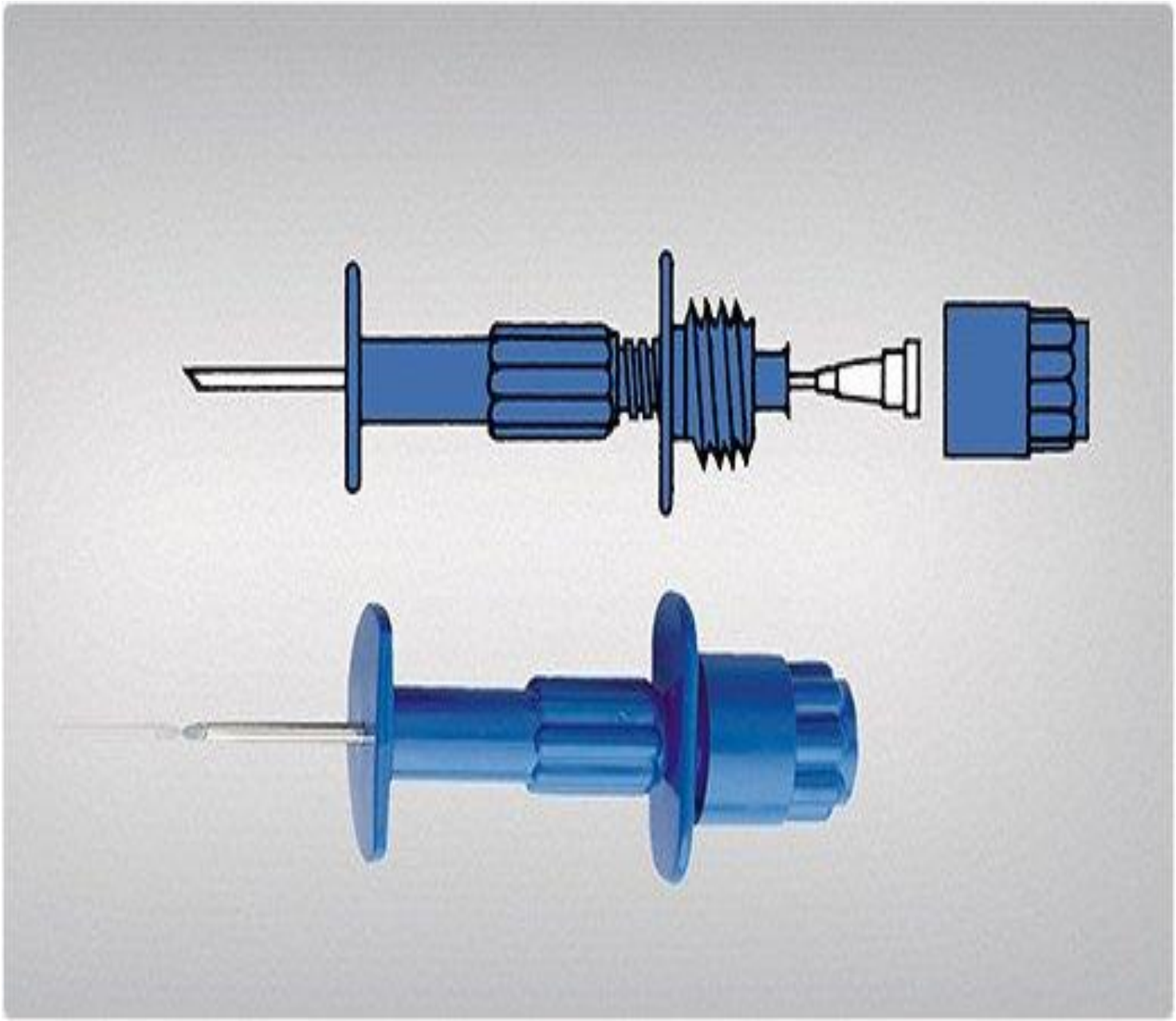
0 min

Recognize decreased mental status and perfusion

High flow O2

IO/ IV


Growth plate
Tibial tuberosity



5 min

- If no Hepatomegaly /rales
- Push 20 ml/kg isotonic saline
- Reassess after each bolus
- Up to 60 ml/kg until improved perfusion
- Correct hypoglycemia , hypocalcemia
- Antibiotics

15-60 min
Fluid refractory shock

- Inotrope
- Epinephrine 0.05_0.3 microg/kg/min
- Central vein
- Airway access

15-60 min
Fluid refractory shock

- Titrate Epinephrine 0.05 _ 0.3 microg/kg/min for cold shock(Dopamine 5_9)
- Titrate Norepinephrine from 0.05 and upward to reverse Warm shock (Dopamine >= 10)

Cold Shock vs. Warm Shock

Etiology: Decreased Stroke Volume
Impairment: Preload, Afterload, & Contractility

Etiology: Decreased Vascular Tone(SVR)
Pulse pressure: Wide

60 min <
Catecholamine resistant shock

- At risk for Adrenal insufficiency : ***Hydrocortisone***
- Goal :
- Normal MAP _ CVP
- ScvO2 > 70
- CI : 3.3 _ 6 L/min/m2

Cold shock with Normal BP

- ScvO2 < 70 / Hgb > 10 g/dL
- On Epinephrine
- ***Milrinone***
- If CI < 3.3 with high SVRI and poor perfusion
- Add vasodilator (Levosimendan)

Cold shock with low BP

- ScvO2< 70 / Hgb > 10 g/dL
- On Epinephrine
- Add ***Norepinephrine***
- Goal : normal DBP
- If CI < 3.3 add Dobutamine , levosimendan , milrinone

Warm shock with low BP

- ScvO2 < 70 on Norepinephrine
- If euvolemic add ***Vasopressin***
- If CI < 3.3 add
- Epinephrine , Dobutamine , Levosimendan

Persistent catecholamine resistant shock

- Pericardial effusion
- Pneumothorax
- Maintain IAP < 12 mmHg

- ***Refractory shock***
- ***ECMO***

Physiologic indicators should be targeted during therapy

Quality of central & peripheral pulses
Skin perfusion
Mental status
Urine output
HR
BP
Serum lactate
CVP & Scvo2
Noninvasive ultrasonic determination

respiratory

- Oxygen
- Noninvasive ventilation
- Early endotracheal intubation and mechanical ventilation
- Positive end-expiratory pressure (PEEP)
- Permissive hypercapnia
- High-frequency ventilation
- Extracorporeal membrane oxygenation (ECMO)

Hematologic

- Vitamin K
- Fresh-frozen plasma
- Platelets
- Heparinization

Endocrine

- Stress-dose steroids in patients previously given steroids
- Physiologic dose for presumed
- primary insufficiency in sepsis

Metabolic

- Treatment of hypovolemia (fluids),
- Improvement of renal acid excretion
- Low-dose (0.5-2.0 mEq/kg) ***sodiumbicarbonate*** if patient is not showing response, pH <7.1, and ventilation (CO_2 elimination) is adequate

Surviving Sepsis

- lactate level
- blood cultures
- broad-spectrum ***Antibiotics***
- 30 mL/kg crystalloid for hypotension or lactate ≥4 mmol/L
- ***vasopressors***

- Rapid IV 20 mL/kg isotonic fluid should be initiated to reverse the shock state
- repeat quickly up to 60-80 mL/kg or more
- to normalize HR according to age-based HRs
- urine output (to 1 mL/kg/hr)
- capillary refill time (to <2 sec)
- normal mental status
- If shock remains refractory : vasopressor therapy

- **Distributive shock** that is not secondary to sepsis
- abnormality in vascular tone
- Cardiac output is normal or supranormal
- volume resuscitation
- vasoconstrictive agent to increase SVR
- spinal cord injury and spinal shock:
- ***phenylephrine*** or ***vasopressin*** to increase SVR

- ***Anaphylactic*** shock :
- ***Epinephrine*** is the treatment of choice
- Epinephrine has peripheral α-adrenergic as well as inotropic effects that may improve the myocardial depression seen with anaphylaxis and its associated inflammatory response

cardiogenic shock

- may decompensate quickly when fluids are administered
- ***5-10 mL/kg***
- In any patient with shock whose clinical status deteriorates with fluid resuscitation, a cardiogenic etiology should be considered
- myocardial support with ***Epinephrine*** ,Dopamine , inodilator such as ***Milrinone*** , Dobutamine
- Norepinephrine and Vasopressin, should be avoided

obstructive shock

- fluid resuscitation may be briefly temporizing in maintaining CO
- ***pericardiocentesis*** for pericardial effusion
- pleurocentesis or ***chest tube*** placement for pneumothorax
- ***thrombectomy***/thrombolysis for pulmonary embolism
- ***prostaglandin*** infusion for ductus dependent cardiac lesions


Thank
you