IN THE NAME OF GOD

SHOCK

Dr. Marzie Fatemi Abhari

Assistant professor of Alborz University Of Medical Sciences

Definition

Shock is an acute process characterized ***by the body's inability to deliver adequate oxygen to meet the metabolic demands of vital organs and tissues.***

Insufficient oxygen at the tissue level is unable to support normal aerobic cellular metabolism, resulting in a shift to less efficient anaerobic metabolism.

EPIDEMIOLOGY

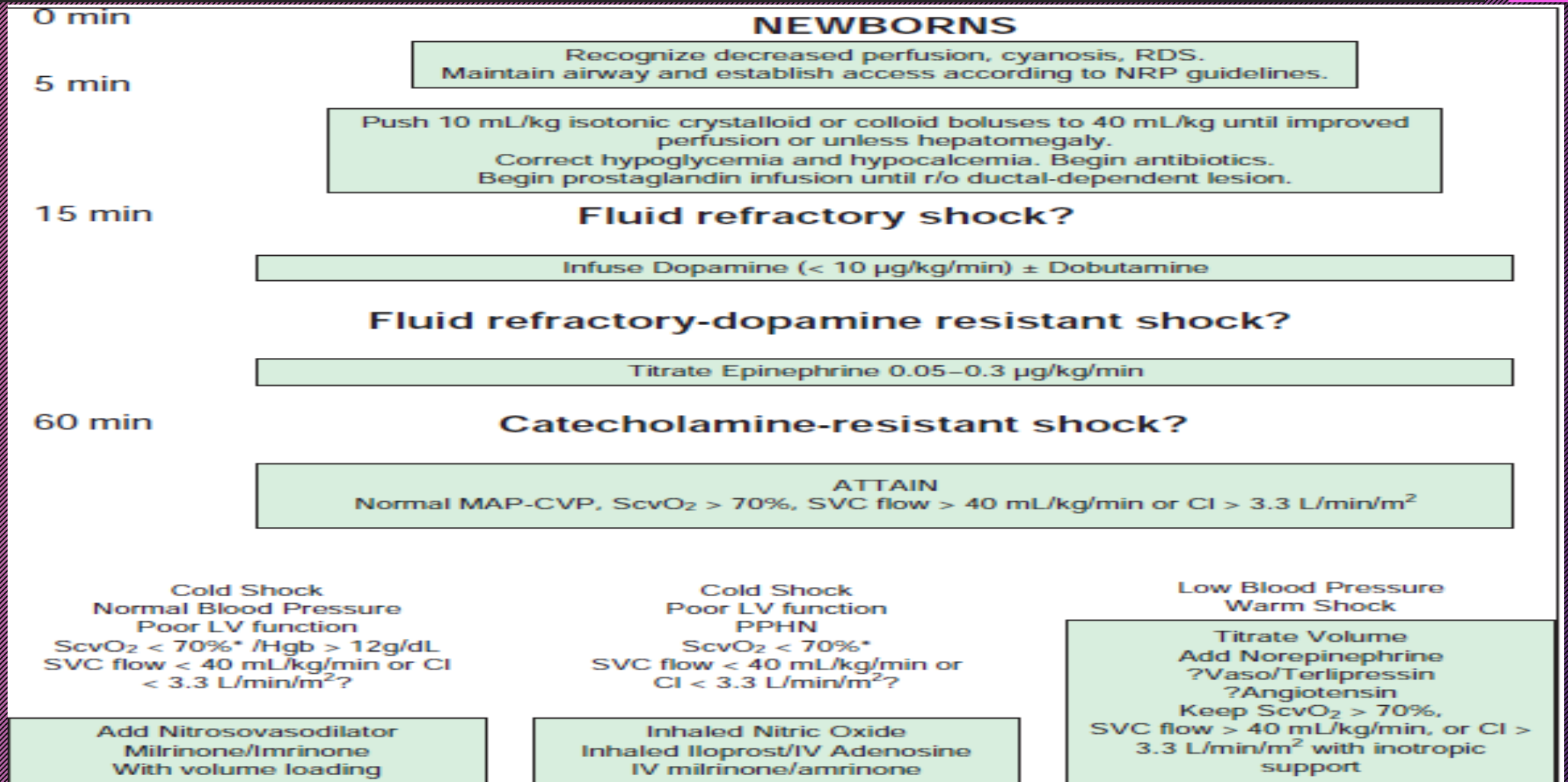
- Shock occurs in approximately 2% of all hospitalized infants, children, and adults in developed countries.
- The mortality rate varies substantially depending on the etiology and clinical circumstances.

TYPES OF SHOCK

- 5 major types of shock:

- hypovolemic,
- cardiogenic,
- distributive,
- obstructive,
- septic

American College of Critical Care Medicine algorithm for time-sensitive, goal-directed stepwise management of hemodynamic support in newborns.



American College of Critical Care Medicine algorithm for time-sensitive, goal-directed stepwise management of hemodynamic support in infants and children.

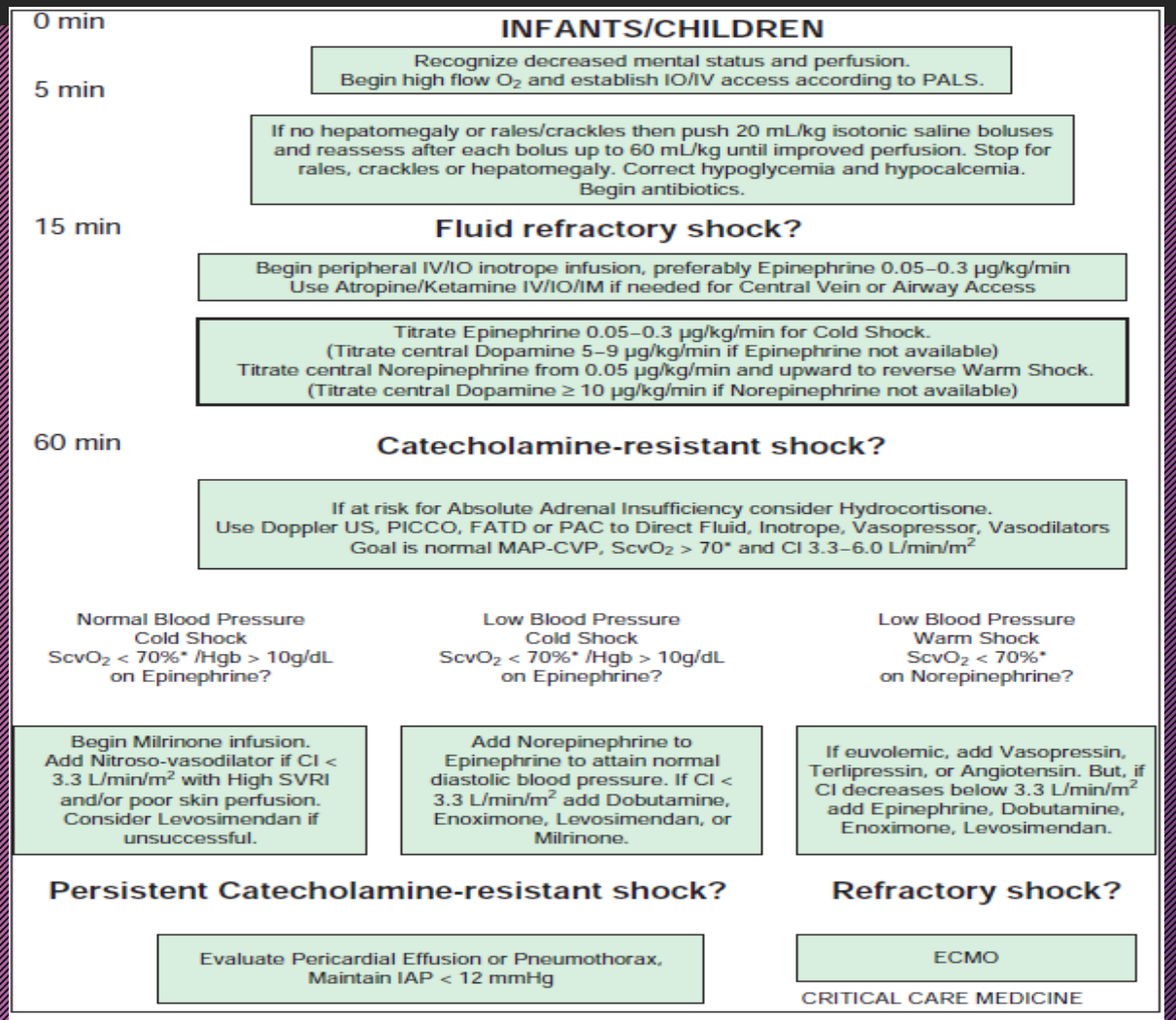


Table 88.1 Types of Shock

HYPOVOLEMIC	CARDIOGENIC	DISTRIBUTIVE	SEPTIC	OBSTRUCTIVE
Decreased preload secondary to internal or external losses	Cardiac pump failure secondary to poor myocardial function	Abnormalities of vasomotor tone from loss of venous and arterial capacitance	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	Decreased cardiac output secondary to direct impediment to right- or left-sided heart outflow or restriction of all cardiac chambers
POTENTIAL ETIOLOGIES				
Blood loss: hemorrhage Plasma loss: burns, nephrotic syndrome Water/electrolyte loss: vomiting, diarrhea	Congenital heart disease Cardiomyopathies: infectious or acquired, dilated or restrictive Ischemia Arrhythmias	Anaphylaxis Neurologic: loss of sympathetic vascular tone secondary to spinal cord or brainstem injury Drugs	Bacterial Viral Fungal (immunocompromised patients are at increased risk)	Tension pneumothorax Pericardial tamponade Pulmonary embolism Anterior mediastinal masses Critical coarctation of aorta

CLINICAL MANIFESTATIONS

- May initially as :
- only tachycardia, with or without tachypnea.
- Progression leads perfusion, respiratory distress or failure, alteration of mental status, and low BP;
- Hypotension is often a late finding, and reflects an advanced state of decompensated shock.

CLINICAL MANIFESTATIONS

- ***Hypovolemic shock:***
- Initially as orthostatic hypotension to decreased urine output, poor peripheral and is associated with dry mucous membranes, dry axillae, poor skin turgor, and decreased urine output.

CLINICAL MANIFESTATIONS

- ***Cardiogenic shock:***
- Tachypnea, cool extremities,
- delayed capillary filling time,
- poor peripheral and/or central pulses,
- declining mental status,
- and decreased urine output

CLINICAL MANIFESTATIONS

- ***Obstructive shock*** :

- Inadequate cardiac output because of a physical restriction of forward blood flow, and the acute presentation may quickly progress to cardiac arrest.

CLINICAL MANIFESTATIONS

- ***Distributive shock:***
- Initially as peripheral vasodilation and increased but inadequate cardiac output.

CLINICAL MANIFESTATIONS

- Regardless of etiology, ***uncompensated shock:***
- Hypotension,
- High SVR,
- Decreased cardiac output,
- Respiratory failure,
- Obtundation,
- Oliguria, occurs late in the progression of disease.

LABORATORY FINDINGS

- Thrombocytopenia,
- Prolonged PT,PTT
- Reduced serum fibrinogen level,
- Anemia.
- Neutrophilia and increased immature forms (i.e., bands, myelocytes, promyelocytes), vacuolation of neutrophils, toxic granulations, and Döhle bodies can be seen with infection. Neutropenia or leukopenia may be an ominous sign of overwhelming sepsis.

TREATMENT

- 1. INITIAL TREATMENT
- 2. Additional Early Considerations
- 3. Considerations for Continued Therapy

TREATMENT

- Initial Management:
- Early recognition and prompt intervention are extremely important in the management of all forms of shock .

- For **respiratory distress and hypoxemia**:
- start with face mask oxygen or,
- If needed and available, high-flow nasal cannula oxygen or nasopharyngeal CPAP (NP CPAP)

TREATMENT

- For **improved circulation**:
- IV or IO for fluid resuscitation and
- Inotrope infusion when a central line is not available.

TREATMENT

- *Initial therapeutic end-points of resuscitation of septic shock:*
- *CRT ≤2 sec, normal blood pressure for age, normal*
- *pulses with no differential between peripheral and central pulses,*
- *warm extremities, urine output >1 mL kg/hr,*
- *normal mental status.*
- *ScvO2 saturation ≥70% and cardiac index between 3.3 and*
- *6.0 L/min/m2 should be targeted thereafter.*

TREATMENT

- **FLUID RESUSCITATION**
  - 1. IN HYPOTENSIVE CASES Infusion of isotonic crystalloids or albumin with boluses of up to 20 mL/kg crystalloids (or albumin equivalent)over 5-10’
  - This bolus should be repeated quickly up to 60-80 mL/kg; it is not unusual for severely affected
  - patients to require this volume within the 1st 3 hr of treatment.

- Fluid resuscitation may sometimes require as much as 200 mL/kg
- or greater.
- If hepatomegaly or rales present, inotropic support should be
- implemented, not fluid resuscitation.
- In nonhypotensive children with severe hemolytic anemia (severe malaria or sickle cell crises), blood transfusion is considered superior to crystalloid or albumin bolus.

TREATMENT

- ***INOTROPES, VASOPRESSORS, AND VASODILATORS:***
- 1. Begin peripheral inotropic support until central venous access can be attained in children who are not responsive to fluid resuscitation.
- 2. Patients with low cardiac output and elevated systemic vascular
- resistance states with normal blood pressure should be given
- vasodilator therapies in addition to inotropes.

TREATMENT

- *Refractory pediatric septic shock and respiratory failure:*

ECMO

TREATMENT

- ***CORTICOSTEROIDS***
- Timely hydrocortisone therapy in children with fluid-refractory,
- catecholamine-resistant shock and suspected or proven absolute
- (classic) adrenal insufficiency.

TREATMENT

- ***MECHANICAL VENTILATION***
- Lung-protective strategies during mechanical ventilation.

TREATMENT

- ***SEDATION, ANALGESIA, AND DRUG TOXICITIES***
- 1. In critically ill, mechanically ventilated patients with sepsis.
- 2. Monitor drug toxicity lab results

TREATMENT

GLYCEMIC CONTROL

- . Control hyperglycemia ≤180 mg/dL.
- Glucose infusion should accompany insulin therapy

TREATMENT

- ***DIURETICS AND RENAL REPLACEMENT THERAPY***
- Use diuretics to reverse fluid overload when shock has resolved,
- and if unsuccessful, use continuous venovenous hemofiltration
- (CVVH) or intermittent dialysis to prevent >10% total body weight
- fluid overload.

TREATMENT

- ***NUTRITION***
- Enteral nutrition given to children who can be fed enterally,
- parenteral feeding in those who cannot.

TREATMENT

- ***ANTIBIOTICS AND SOURCE CONTROL***
- 1. within 1 hr of the identification of severe sepsis.
- 2. Blood culture
- The empirical drug choice should be changed as epidemic and endemic ecologies
- 3. Clindamycin and antitoxin therapies for toxic shock syndromes
- with refractory hypotension.
- 4. Clostridium difficile colitis should be treated with enteral antibiotics.
- Oral vancomycin is preferred for severe DISEASES

TREATMENT

- If ***shock remains:***
- vasopressor therapy (e.g., epinephrine, Dopamin)+ fluid

TREATMENT

- ***Distributive shock:***
- caused by a primary abnormality in vascular tone. Cardiac output in affected patients is usually maintained and may initially be supranormal.
- Volume resuscitation,
- Early initiation of a vasoconstrictive agent
- Patients with spinal cord injury and spinal shock:
- Either phenylephrine or vasopressin
- ***patients with anaphylaxis:***
- Epinephrine

TREATMENT

- ***cardiogenic shock:***
- poor response to fluid resuscitation and may decompensate quickly when fluids are administered.
- Smaller boluses of fluid (5-10 mL/kg)
- In any patient with shock whose clinical status deteriorates with fluid resuscitation, a cardiogenic etiology should be considered
- Early Initiation of myocardial support with ***epinephrine or dopamine.***
- An inodilator, such as ***milrinone.***
- ***Dobutamine*** or other vasodilating agents, such as ***nitroprusside***,

TREATMENT

- ***obstructive shock***,
- Fluid resuscitation may be briefly temporizing in maintaining cardiac output, but the primary insult must be immediately addressed.
- For Examples:
- ***pericardiocentesis*** for pericardial effusion,
- ***pleurocentesis***
- ***chest tube*** placement for pneumothorax,
- t***hrombectom***y
- ***thrombolysis*** for pulmonary embolism, and the initiation of a ***prostaglandin***
- infusion for ductus-dependent cardiac lesions.

TREATMENT

- ***Metabolic status*** should be meticulously maintained.
- Electrolyte levels should be monitored closely,
- ***Hypoglycemia*** is common and should be promptly treated. ***Hypocalcemia***, which may contribute to myocardial dysfunction, should be treated with a goal of normalizing the ionized calcium concentration.

TREATMENT

- ***Adrenal function*** is another important consideration in shock, and
- hydrocortisone replacement may be beneficial. Up to 50% of critically ill patients may have absolute or relative adrenal insufficiency.
- They should receive stress doses of hydrocortisone.
- In patients with shock that is unresponsive to fluid resuscitation and catecholamines HYDROCORTISON is helpful.

TREATMENT

- ***Considerations for Continued Therapy***
- After the 1st hr of therapy and attempts at early reversal of shock, focus
- on goal-directed end-points should continue in an intensive care setting

Clinical end-points serve as global markers for organ perfusion and oxygenation.

- Laboratory parameters such as SvO2 (or ScvO2),
- serum lactate concentration,
- Cardiac index,
- and hemoglobin.
- Hemoglobin should be generally maintained at 10 g/dL,
- SvO2 (or ScvO2) >70%,
- and cardiac index at 3.3-6.0 L/min/m2 to optimize
- oxygen delivery in the acute phase of shock.

TREATMENT

- Blood lactate level and calculation of base deficit from ABG values are very useful markers for the adequacy of oxygen delivery.
- There is increasing use of measures of local tissue oxygenation, including near-infrared spectroscopy of the cerebrum, flank, or abdomen.
- Respiratory support should be used as clinically appropriate.

TREATMENT

- When shock leads to ARDS requiring mechanical ventilation, lung-protective strategies to keep plateau pressure <30 cm H2O and maintain tidal volume at 6 mL/kg the initial shock state has been reversed, data demonstrate that judicious fluid administration, renal replacement therapy, and fluid removal may also be useful in children with anuria or oliguria and fluid overload
- Other interventions include correction of coagulopathy with FFP or cryoprecipitate and platelet transfusions as necessary, especially in the presence of active bleeding.

TREATMENT

- If shock remains refractory despite maximal therapeutic interventions,
- mechanical support with extracorporeal membrane oxygenation
- (ECMO) or a ventricular assist device (VAD) may be indicated. ECMO
- may be lifesaving in cases of refractory shock regardless of underlying
- etiology. Similarly, a VAD may be indicated for refractory cardiogenic
- shock in the setting of cardiomyopathy or recent cardiac surgery.

PROGNOSIS

- In septic shock, mortality rates are as low as 3% in previously healthy
- children and 6–9% in children with chronic illness (compared with
- 25–30% in adults).
- With early recognition and therapy, the mortality
- rate for pediatric shock continues to improve, but shock and MODS
- remain one of the leading causes of death in infants and children. The
- risk of death involves a complex interaction of factors, including the
- underlying etiology, presence of chronic illness, host immune response,
- and timing of recognition and therapy.

سپاس