

Shock

Definition - a state in which there is inadequate tissue perfusion to meet metabolic demands.

Types

Hypovolemic

dehydration
gastroenteritis
deprivation
heat stroke
hemorrhage
burns

Distributive

anaphylaxis
neurogenic
drug toxicity
septic shock*

Cardiogenic

congenital heart disease
ischemic heart disease
anoxia
Kawasaki's
traumatic
infectious cardiomyopathies
drug toxicity
tamponade

Miscellaneous

heat stroke
pulmonary embolus
clot, fat or air
pancreatitis
drug overdose (barbiturates, – agonists)

* Septic shock actually has components of several groups including distributive and cardiogenic.

Recognition:

- 1) hypotension is **NOT** a sign of shock, because of the compensatory mechanisms below. It can be present in shock
- 2) to catch early you must have a high index of suspicion, is the patient at risk for one of the above
- 3) History:
 - a) vomiting, diarrhea, decreased oral intake
 - b) lethargy, increased sleepiness
 - c) trauma
 - d) allergic symptoms/exposure (or rapid change in physiologic status)
 - e) congenital heart disease
- 4) Physical Findings:
 - a) decreased CNS activity
 - b) abnormal color pale => gray => mottled
 - c) decreased urine output (sign of overall tissue hypoperfusion)
 - d) tachypnea, tachycardia
 - e) delayed capillary refill
 - f) others depend on etiology of shock
- 5) Laboratory findings:
 - a) acidosis on ABG with a base deficit signifying tissue hypoperfusion
 - b) decreased mixed venous oxygen saturation on a VBG
 - c) electrolyte abnormalities (related to cause of shock)
- 6) hypotension is seen only late in shock states after compensatory mechanisms are exhausted

WHY is hypotension only seen as a late sign of shock?

<u>Compensatory mechanisms</u>	<u>Decompensatory effects</u>
Baroreceptors	Cardiac Failure
Chemoreceptors	acidosis
cerebral ischemia	CNS depression
reabsorption of tissue fluids	Disseminated Intravascular Coagulation
endogenous vasoconstrictors	depression of reticuloendothelial system
Renal conservation of water	

Compensatory Mechanisms

Baroreceptor reflexes (respond to small changes in vascular tone/pressure)

- located in the carotid sinus and aortic arch, stimulation causes
 - decreased vagal tone, which
 - increases heart rate
 - decreases coronary resistance (improves myocardial oxygen supply)
 - increased sympathetic tone, which causes
 - venoconstriction
 - constriction of blood reservoirs (increasing circulating blood volume)
 - spleen - dogs
 - skin and skeletal muscle - humans

Chemoreceptor

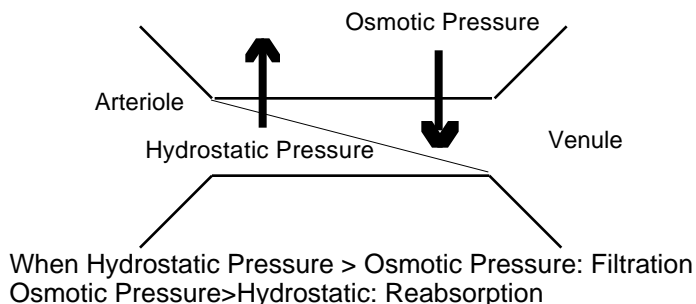
- located locally in tissue beds
- sense hypoxia (due to inadequate blood flow in peripheral tissues), and cause
 - further vasoconstriction
 - respiratory stimulation - improves venous return (pump model), also helps compensate for acidosis

Cerebral Ischemia (Cerebral Perfusion Pressure < 40 mmHg)

- activates sympathoadrenal system (more potent than Chemo or Baroreceptor mechanisms)
 - increased catecholamine release from both adrenal gland and sympathetic nerves
 - (can also get vagal stimulation which is counterproductive)

Reabsorption of tissue fluids

- decreased mean arterial pressure
 - arteriolar constriction
 - decreased venous pressure
- } All lead to decreased hydrostatic pressure in capillaries, leading to increased resorption of fluid
- 15ml/kg/hour, up to 1l/hour can be reabsorbed in adult sized patients



Compensatory Mechanisms (continued)

Endogenous Vasoconstrictors

- Epinephrine and Norepinephrine
 - released from adrenal medulla and sympathetic nerves
 - cause vasoconstriction and increased cardiac output
- Vasopressin (Antidiuretic hormone)
 - released from posterior pituitary
 - potent vasoconstrictor
- Renin (from decreased renal perfusion)
 - leads to angiotensinogen production eventually yielding angiotensin
 - a very potent vasoconstrictor

Renal Conservation of Water

- Aldosterone release
 - stimulated by vasopressin
 - Causes Na reabsorption in distal tubules
 - Water follows the sodium

Decompensatory Mechanisms

Cardiac Failure

- many potential etiologies (i.e. actual etiology is controversial)
- decreased circulating blood volume => decreased coronary blood flow => decreased cardiac function

Acidosis (from hypoperfusion, anaerobic metabolism => lactic acidosis)

- depressant of myocardial function
- decreased response to catecholamines both in myocardium and peripheral vasculature

CNS Depression

- opioid release (enkephalins, β -endorphin)
- Naloxone has been used as treatment in shock, with some success

Disseminated Intravascular Coagulation

- abnormalities of clotting system develop as a result of attempt to control hemorrhage but also dilution/loss of clotting factors
- GI hemorrhage seen as complication in dog model of acute hemorrhage, hours after the initial event

Reticuloendothelial System dysfunction

- function depressed in shock
- lose antibacterial function
- can get endotoxin release from native bacteria
- aggravates already compromised situation

Treatment

- 1) **Airway**
 - Needs will vary depending on etiology of shock, from no intervention to aggressive intervention (i.e. anaphylaxis)
- 2) **Breathing**
 - Patients need respiratory support and monitoring, consider O₂ to help with oxygen delivery even though sats may be OK, patients may need intubation, or other respiratory support, particularly to help compensate for a profound metabolic acidosis.
- 3) **Circulation**
 - Read on.

Treatment (after ABC's)

- 1) VOLUME, VOLUME, VOLUME
- 2) treat the underlying cause
- 3) correction of acidosis
- 4) Vasoactive substances

Volume

replacement must be made with isotonic and ideally isoosmotic fluids

Normal Saline is the solution of first choice because it is ubiquitously available in health care settings (including ambulances) and carries minimal risk

Initial Volume always at least 20ml/kg

in most cases of early shock it will take still more volume than this to correct deficits, don't be timid initially with fluid

For later fluid, try to replace with isotonic/isoosmotic fluid that is needed

pRBCs in hemorrhage

FFP also as needed

5% albumin also a good choice

Hespan if concerned about blood products

plasmanate also an option

Where ? Intravenous, which site:

Peripherally (saphenous, antecubital, other sites as visible/palpable)

largest bore IV obtainable, but any IV will work

Intraosseous

proximal tibia (can also go distal femur, iliac crest, sternum)

perpendicular to bone, two-three finger breadths below tibial tubercle

do not have to be able to draw out marrow for it to be in correct location

can put any IV fluid, med or colloid through IO

should be considered if rapid IV access is not obtainable

Central

If skilled person is available, will most useful, allow for blood draws as well

More than one lumen if possible.

sites include: Femoral (safest), Internal Jugular, Subclavian

Treat Underlying Cause

stop hemorrhage if bleeding

broad spectrum antibiotics if suspect septic

- specific antibiotics if known agent

cardiac support and close monitoring if cardiogenic

removal of offending agent if anaphylaxis, early use of epinephrine

Correct Acidosis

acidosis can cause significant problems with cellular function and should be aggressively treated

Bicarbonate replacement when deficit is >6mEq/l

Formula:

$0.3 \times (\text{weight in kg}) \times (\text{base deficit}) = \text{mEq NaHCO}_3$ to replace half of deficit

Should be given slowly in 1-2mEq/kg boluses

May need 10-20mEq/kg to correct acidosis

remember that this can be a large Na load leading to hyperosmolarity

Vasoactive Substances

select to optimize desired effect

consider the main effects as follows:

beta effects: increase inotropy and chronotropy - increasing cardiac output (beta-1)

also some pulmonary and peripheral vasodilation (beta-2)

alpha effects: increase systemic vascular resistance - maintaining blood pressure

vasodilators: decrease systemic vascular resistance - decrease afterload, potentially

improving cardiac function, but also dramatically reducing BP in hypovolemic pt

choices:

epinephrine (beta and alpha, stronger beta)

norepinephrine (alpha and beta, more alpha)

dobutamine (beta-1 alone)

dopamine (dopaminergic at low doses, beta at medium to high and alpha at high doses)

isoproterenol (pure beta effects, both beta-1 and beta-2)

nitroprusside (vasodilator, a nitric oxide donor)

nitroglycerin (vasodilator, a nitric oxide donor)

How to choose: depends on the etiology, and the patient's status

never use vasodilators on someone who is hypovolemic

norepinephrine has very potent vasoconstriction that can be very useful in some forms of distributive shock, but redundant and dangerous in other types of shock

epi and norepi both dramatically increase myocardial oxygen demand as well as cause other problems so should be used cautiously

isoproterenol's vasodilator effects often overwhelm the improved inotropy

Monitoring

Remember: Shock is not something that is broke that you fix and are done. It is an evolving process that is a symptom of something else going wrong with the patient, that left untreated can result in death.

Monitoring Keys

- a) electrolytes
- b) glucose
- c) blood gases (pH and oxygenation)
- d) Central venous pressure
- e) hemodynamics
- f) coagulation status
- g) urine output
- h) neurologic status

Electrolytes:

Na: can be markedly abnormal as a result of the underlying disease (hypo/hypernatremic dehydration), can get elevated during process of correcting base deficit. Goal should be to normalize Na to avoid abnormal fluid shifts, this should be done SLOWLY!

K: in acidotic state potassium can be elevated to the point of cardiac dysrhythmias, as correction of acidosis occurs K can be driven back into cells developing severe hypokalemia in some cases. It should be monitored and treated appropriately

Ca: in treatment of base deficit calcium can be chelated and dramatically decrease, leading to problems from seizures, hypotension and myocardial dysfunction, CaCl₂ can be used acutely to correct hypocalcemia.

Glucose

As part of response to compensatory mechanisms (epinephrine and corticosteroids) hyperglycemia is a common occurrence in stressed children. This can cause problems from osmotic diuresis and glucose intolerance. Some studies showed poor neurologic outcome could be best predicted by hyperglycemia in patients with shock. Care should be made not to overload the glucose management system in the body (i.e. no dextrose in flush solutions)

Monitoring (continued)*Blood Gases*

close monitoring is essential to evaluate correction of base deficit. In addition, in severe shock states respiratory compromise is common, and potential for respiratory failure is great. Patients may need intubation and mechanical ventilation. Failure to oxygenate is one sign of this. It is also important to maintain good oxygen delivery to help minimize anaerobic metabolism and worsening of acidosis.

Venous Blood Gases are also of benefit since mixed venous oxygen saturation is a measurement of tissue perfusion and cardiac output. Need to be from a central vein to be of use.

Central Venous Pressure

CVP monitoring allows close observation of hydration status and pre-load. This is essential in cardiogenic shock patients in whom too much fluid will worsen the shock state.

Hemodynamics

good, reliable blood pressure monitoring and EKG monitoring are important. Although blood pressure is typically normal in early shock, decreasing blood pressure is a sign of someone who is decompensating

Coagulation Status

as DIC is a common complication even early in shock, close monitoring of coagulation status will allow early correction of deficits

Urine Output

this is representative of organ perfusion, improving urine output can be a sign of improving volume status, while worsening UO suggests more aggressive therapy

Neurologic Status

this is also representative of organ perfusion, namely the brain.

Resources

Rogers, MC **Textbook of Pediatric Intensive Care**. Chapter 15, p483-524. Williams and Wilkins, Baltimore, MD 1987.

An excellent and concise overview of the etiology and management of Shock, both in general and for specific instances. There is a more recent addition than this, but the principles remain the same.

Berne RM and Levy MN. **Physiology, 3rd edition**. Chapter 32, pp 532-543. Mosby, St. Louis, 1993.

Basic Physiology of all the interactions that go on to normalize circulatory function.

De Bruin WJ et. al. "Fluid Resuscitation in Pediatrics." **Critical Care Clinics 8(2)**: 423-438. April 1992

A nice review of everything involved in fluid resuscitation of the Pediatric patient.

Griffel MI and Kaufman BS "Pharmacology of Colloids and Crystalloids." **Critical Care Clinics 8(2)**: 235-253. April 1992.

From the same Critical Care Clinics, another nice review of the fluids commonly used in resuscitation.

Textbook of Pediatric Advanced Life Support. American Heart Association.

Stresses the basics of early management and stabilization.