Pediatric Shock

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OBJECTIVES

Definition

Pathophysiology

Types

Management concepts

Definition

Inability to meet the cellular demand for oxygen

Oxygen delivery **<** Oxygen Consumption

Important concepts

CO= HR X SV

Know acceptable HR for age

Children can’t modify stroke volume well

SV determined by Preload, Contractility, Afterload

Important concepts

Oxygen Delivery $DO_2 = CO \times CaO_2$

Oxygen Consumption $= CO \times CvO_2 - CaO_2$

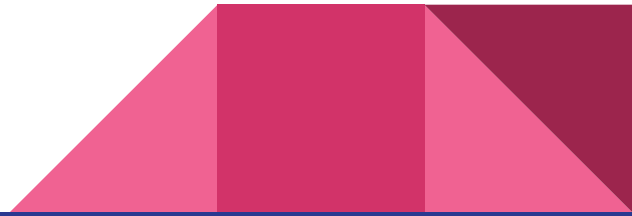
CaO_2

CaO_2 = Hb bound O_2+ Plasma Dissolved O_2

$$\underline{Hb} \times \underline{SaO_2} \times 1.34 + PaO_2 \times FiO_2 \times 0.003$$

$(12 \times 0.95 \times 1.34) + (80 \times 0.21 \times 0.003)$

$15.28 + 0.05 = 15.33$ ml/dl



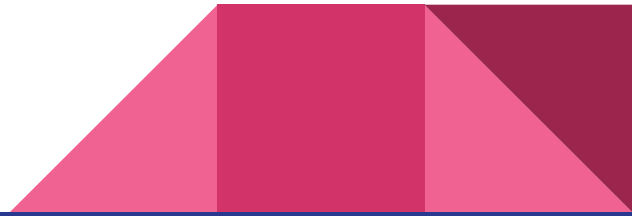
Critical Concepts

Normal CaO_2 :17-20 ml/dl

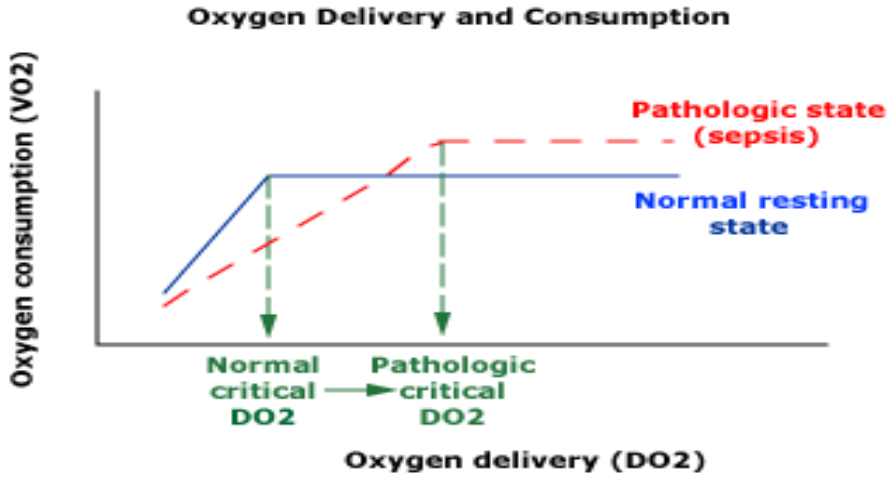
Normal DO_2: 500 ml/min/m2

Normal oxygen extraction : 25-30%

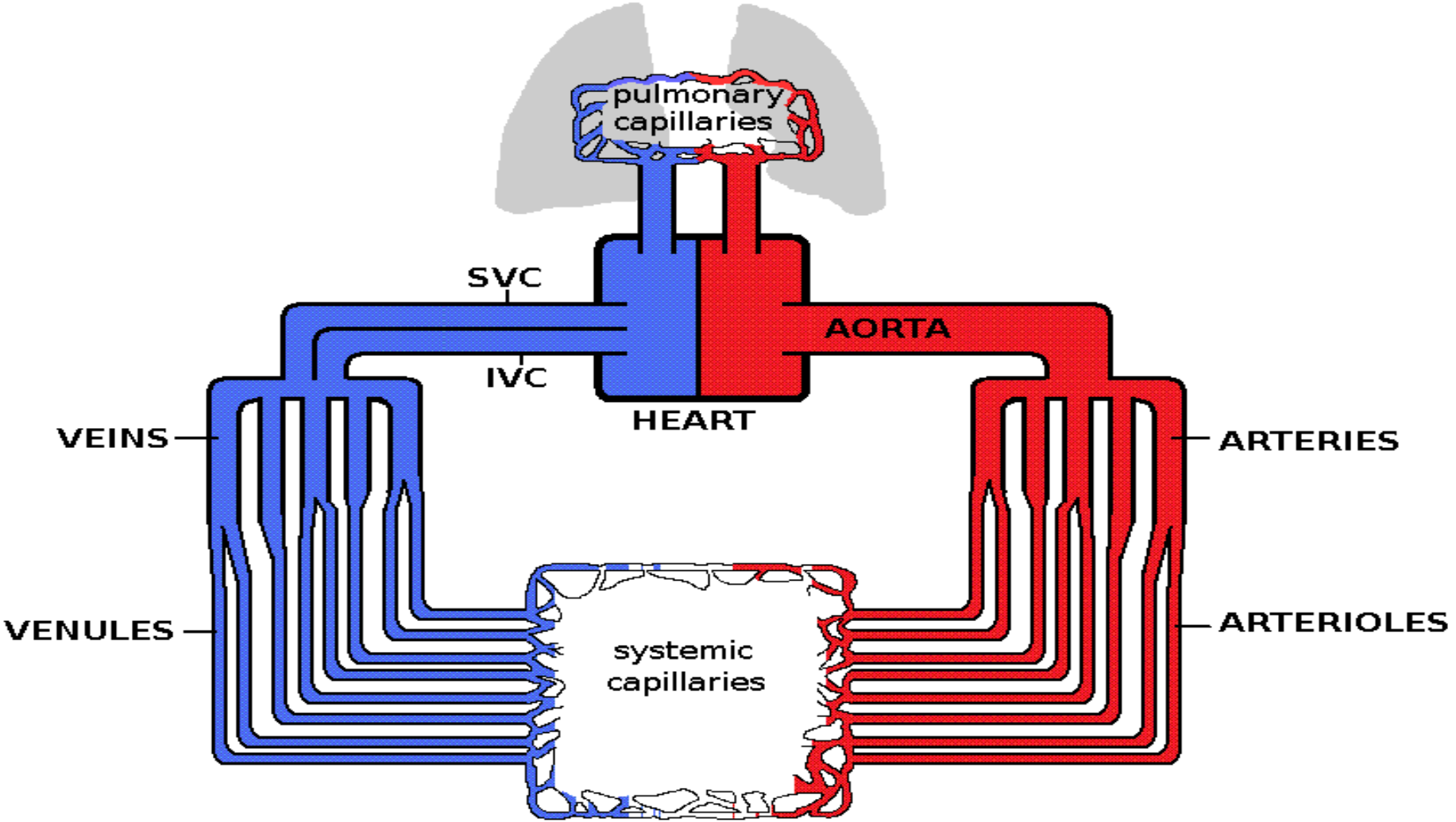
Normal oxygen consumption in an adult is 250 ml/min



Oxygen delivery (DO2) and consumption (VO2)



In the normal state (blue line), oxygen consumption is constant over a range of DO2, and decreases only when DO2 falls below a critical level (critical DO2). Pathologic changes caused by sepsis or systemic inflammatory responses (red line) cause increased VO2 and impaired peripheral oxygen utilization, resulting in an elevation in critical DO2.


pulmonary capillaries
SVC
IVC
AORTA
HEART
VEINS
ARTERIES
VENULES
ARTERIOLES
systemic capillaries

Types of Shock

Cardiogenic

Hypovolemic

Distributive

Obstructive

Dissociative

Cardiogenic

Congenital heart disease: Ductal dependent lesions such as HLHS, AS, Coarc or interrupted aortic arch

Cardiomyopathies : Dilated, restrictive , hypertrophic

Myocarditis: Infectious (Coxsackie B),Toxins(Cocaine), autoimmune

Abnormal rate or rythm: Extreme bradycardia, SVT, VT

Hypovolemic

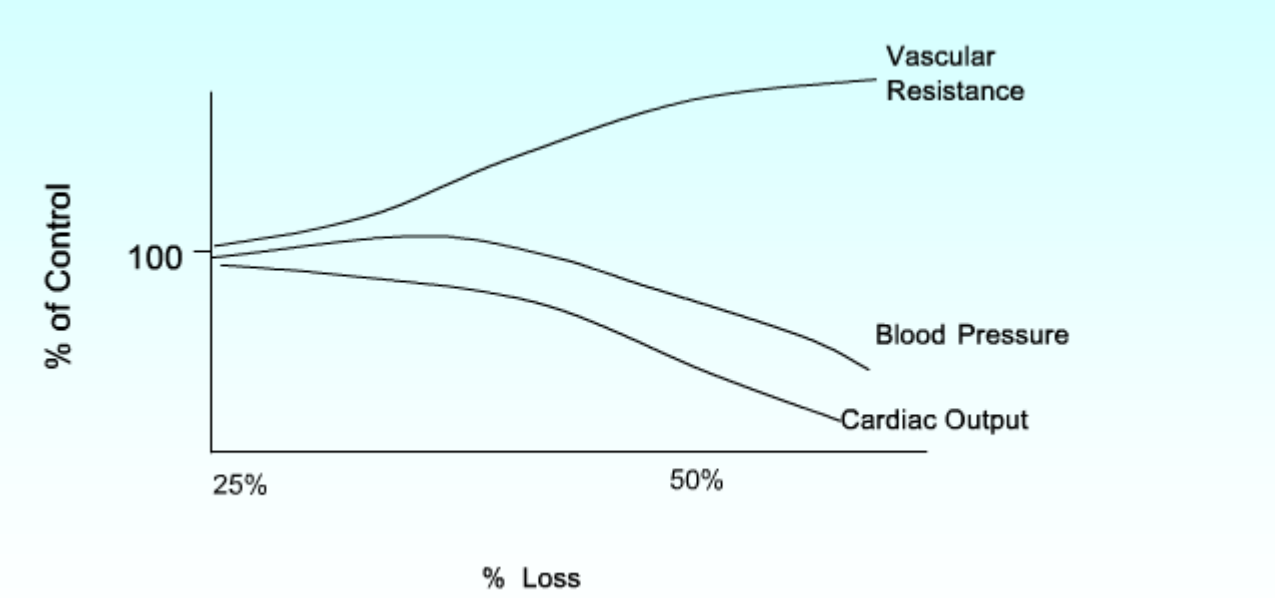
Hemorrhagic : (Think of hidden blood loss like femoral Fracture)

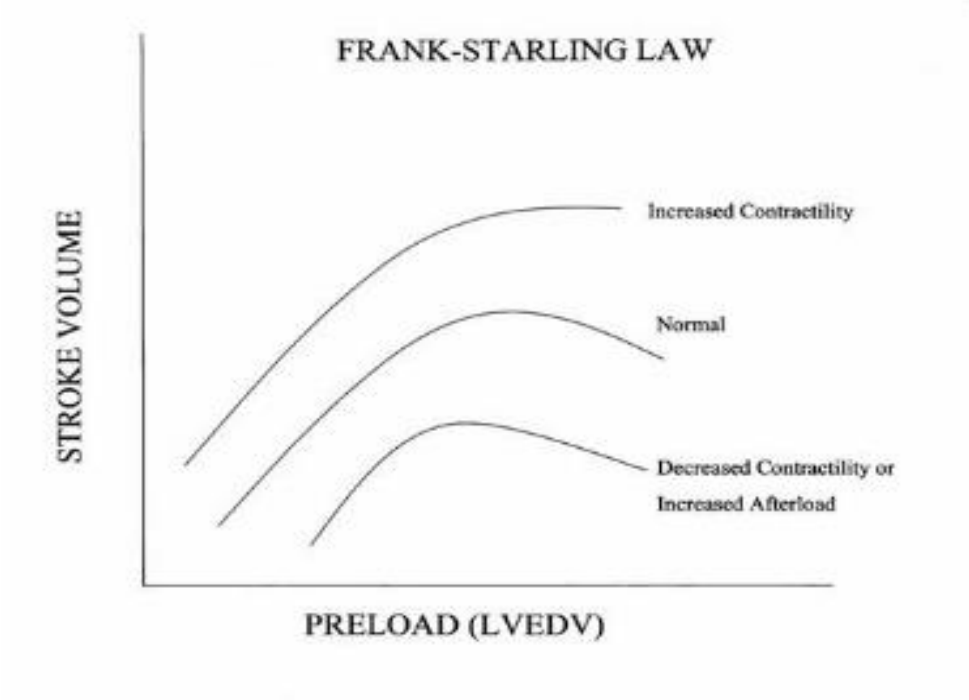
Plasma loss (Burns, Steven Johnson syndrome, Epidermolysis Bullosa)

Loss of water (Gastroenteritis, extreme diuresis in DKA, DI)

Relative hypovolemia (loss of fluid by third spacing)

Hemodynamic Response to Hemorrhage




FRANK-STARLING LAW
STROKE VOLUME
Increased Contractility
Normal
Decreased Contractility or
Increased Afterload
PRELOAD (LVEDV)

Distributive shock

Septic

SIRS

Anaphylactic

Neurogenic

Obstructive

Tension Pneumothorax

Cardiac Tamponade

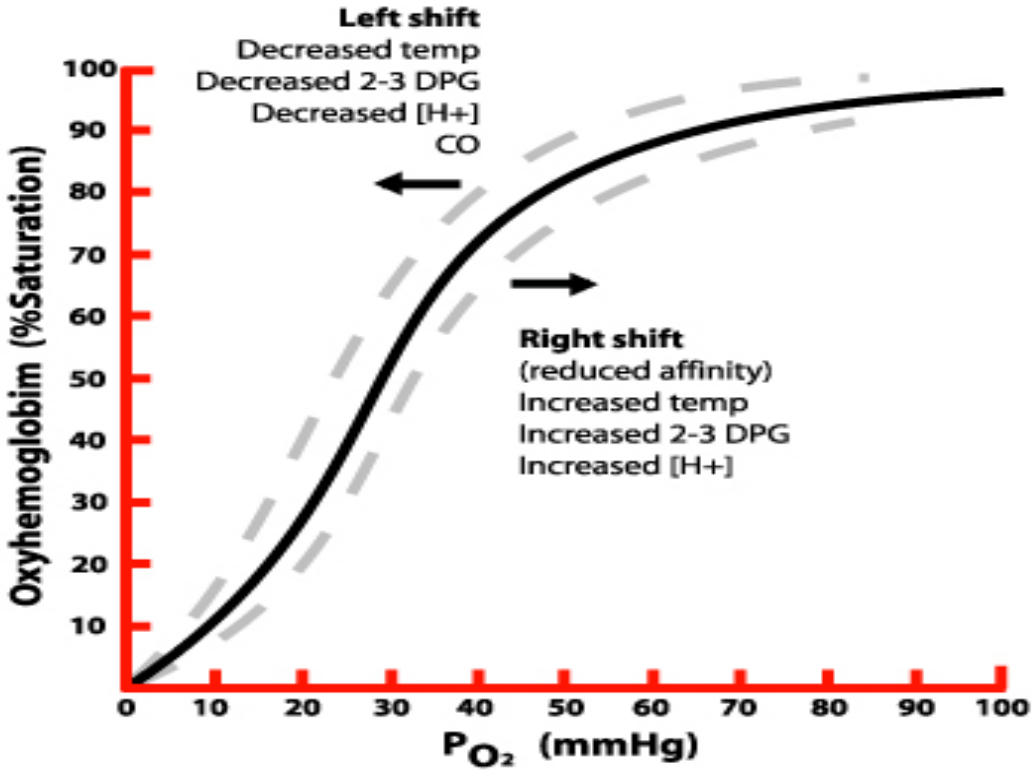
Pulmonary embolism

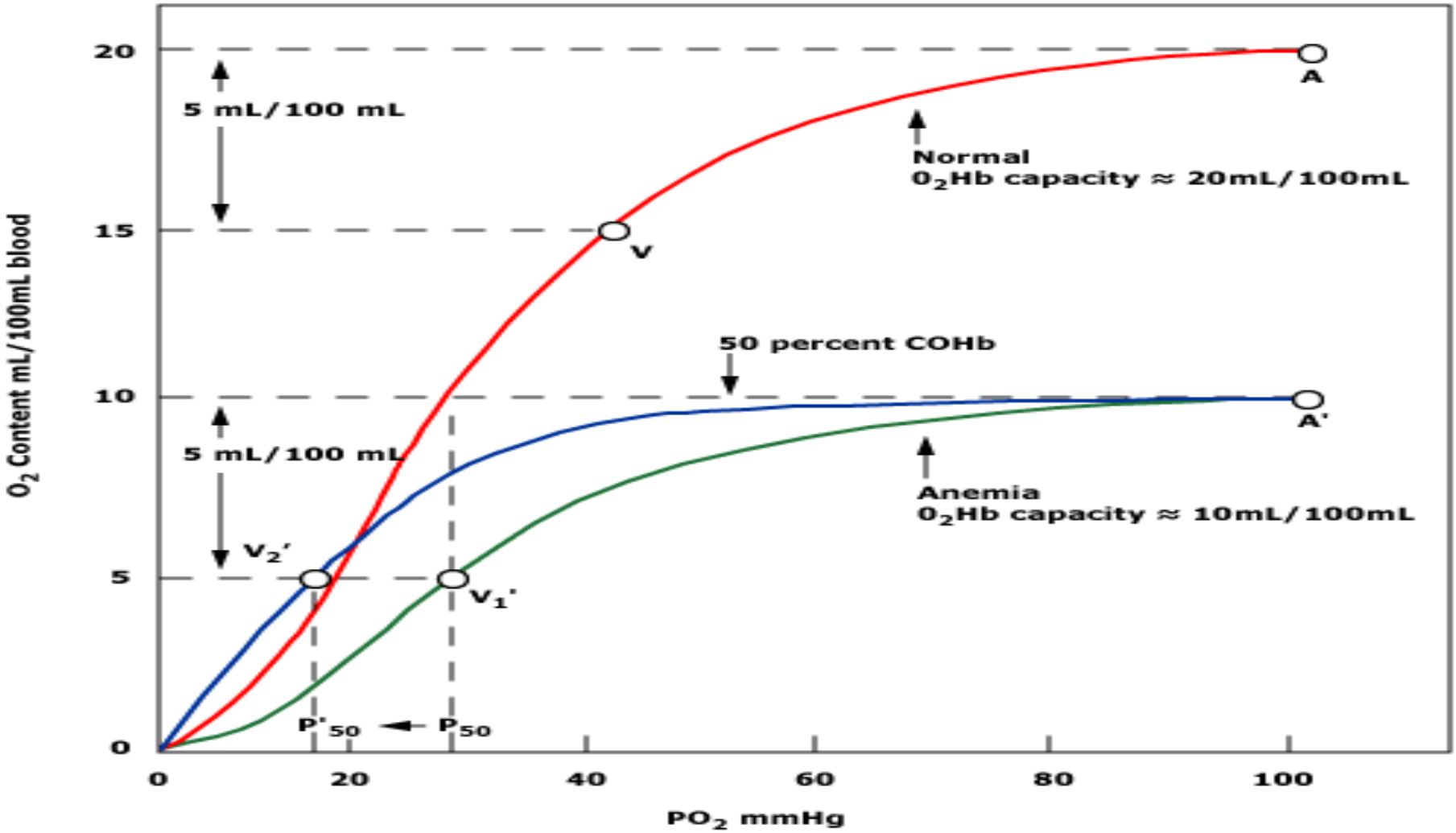
Dissociative

Cyanide poisoning: Blocks oxidative phosphorylation in the mitochondria with resulting switch to anaerobic metabolism

Carbon monoxide: Increased affinity of CO to Hb, perfusion is good but no O2 to deliver

Heat stroke


Left shift
Decreased temp
Decreased 2-3 DPG
Decreased [H+]
CO
Right shift
(reduced affinity)
Increased temp
Increased 2-3 DPG
Increased [H+]
Oxyhemoglobim (%Saturation)
100
90
80
70
60
50
40
30
20
10
0
10
20
30
40
50
60
70
80
90
100
P_{O_2} (mmHg)


20
15
10
5
0
5 mL/100 mL
5 mL/100 mL
A
V
Normal
O_2Hb capacity ≈ 20mL/100mL
50 percent COHb
A'
Anemia
O_2Hb capacity ≈ 10mL/100mL
V_2'
V_1'
P'_{50}
P_{50}
0
20
40
60
80
100
O_2 Content mL/100mL blood
PO_2 mmHg

Hemodynamic profiles of the types of shock in children

Physiologic variable	Preload	Pump function	Afterload	Tissue perfusion	Tissue perfusion
Clinical measurement	Clinical signs* or central venous pressure (if measured)	Cardiac output or index¶	Systemic vascular resistance	Capillary refill time$^{\Delta}$	Mixed venous oxygen saturation$^{\diamond}$
Hypovolemic	↓	↓	↑	↑	Low
Cardiogenic	↑	↓	↑	↑	Low
Distributive	↓ or ↔	↑	↓	↓ (initial)	High
Obstructive	↑	↓	↑	↑	Low

* Clinical signs of decreased preload include tachycardia, tachypnea, decreased or absent peripheral pulses; normal or weak central pulses; capillary refill time >2 seconds; skin that is pale, mottled, cold or diaphoretic; dusky or pale extremities, altered mental status, decreased urine output, and flat jugular veins. Clinical signs of increased preload include jugular venous distension, pulmonary edema, and hepatomegaly. These patients are also typically tachycardic and poorly perfused. Refer to topics on evaluation of shock in children.

¶ Cardiac index (cardiac output per body surface area) is typically what is measured during clinical care.

Δ In patients with shock, capillary refill time >2 seconds is associated with low mixed venous oxygen saturation while flash capillary refill suggests increased mixed venous oxygen saturation.

◇ A low mixed oxygen saturation is <70 percent when measured through a triple lumen catheter and <65 percent when measured through a pulmonary artery catheter.

	CO	SVR	MAP	Wedge	CVP
Hypovolemic	↑	↑	↔ Or ↓	↓↓↓	↓↓↓
Cardiogenic	↓↓	↑↑↑	↔ Or ↓	↑↑	↑↑
Obstructive	↓	↑	↔ Or ↓	↑↑	↑↑
Distributive	↑↑	↓↓↓	↔ Or ↓	↔ Or ↓	↔ Or ↓
Septic: Early	↑↑↑	↓↓↓	↔ Or ↓	↓	↓
Septic: Late	↓↓	↑↑	↓↓	↑	↑ or ↔

MANAGEMENT PRINCIPLES

Decrease O2 consumption

- Minimize work of breathing
- Treat Fever
- Treat pain and anxiety
- Treat Seizures
- Treat Infection

MANAGEMENT PRINCIPLES

Increase O2 Delivery

- Normalize Contractility
- Normalize Systemic Vascular Resistance
- Normalize Pulmonary Vascular Resistance
- Normalize Preload

Management

Administer FiO2

Intubate If Airway is compromised or patient in impending Respiratory Failure

Establish Vascular Access

Management

Start fluid resuscitation with 20 ml/kg of isotonic crystalloid as a push

In patients with suspected cardiogenic shock give only 10 ml/kg

Reasses following each bolus (HR,CRT, Pulses,BP)

May repeat if some improvement noted up to 4-5 boluses

Management

Check blood sugar and serum electrolytes including Ca & Mg

If anaphylaxis suspected give epinephrine, diphenhydramine and hydrocortisone. Conside H2 blocker.

Continnous monitoring of HR, RR, BP, SaO2 & Urine output

Management

If cardiovascular exam not back to normal consider starting Inotropic support

Start Emperic Antimicrobial Therapy for suspected Septic Shock

In Infants with suspected ductus dependant lessions start Prostaglandin E1 drip to reopen the ductus

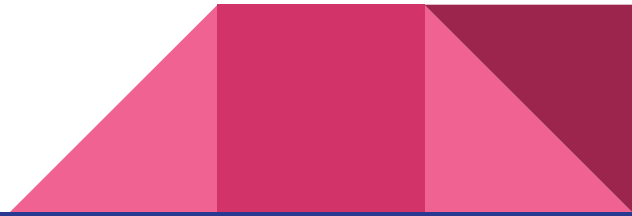
For Heamorhagic Shock give PRBC’s

Management

In Refractory shock consider Adrenal Insufficiency and possible dissociative shock

Asses End Point Organ perfussion

- Level of Concioussnes
- BP, Pulses
- Urine Output



Vasoactive medication receptor activity and clinical effects

Drug	Receptor activity				Predominant clinical effects
	Alpha-1	Beta-1	Beta-2	Dopaminergic	
Phenylephrine	+++	0	0	0	SVR ↑↑, CO ↔/↑
Norepinephrine	+++	++	0	0	SVR ↑↑, CO ↔/↑
Epinephrine	+++	+++	++	0	CO ↑↑, SVR ↓ (low dose) SVR/↑ (higher dose)
Dopamine (mcg/kg/min)*					
0.5 to 2.	0	+	0	++	CO
5. to 10.	+	++	0	++	CO ↑, SVR ↑
10. to 20.	++	++	0	++	SVR ↑↑
Dobutamine	0/+	+++	++	0	CO ↑, SVR ↓
Isoproterenol	0	+++	+++	0	CO ↑, SVR ↓

+++: Very strong effect; ++: Moderate effect; +: Weak effect; 0: No effect.

* Doses between 2. and 5. mcg/kg/min have variable effects.

Management

If shock not responding consider stat Echo to assess function and volume status

To Improve contractility consider Milrinone Or Dobutamine

If Contactility poor despite inotropic support consider afterlod reduction with vasodilators such as Nitroprusside

Management

Refractory Shock

In Refractory shock send cortisol levels and start stress dose Hydrocortisone

Vasopresin may be use if patient not responding to vasopressor therapy

Outcomes

Depends on cause

The sooner you make the diagnosis and start therapy the better the outcome