

Acute Respiratory Distress Syndrome	
Definition	<p>Acute respiratory failure not fully explained by cardiac etiology or fluid overload</p> <ul style="list-style-type: none"> ■ Excludes patients w/ perinatal pulmonary disease ■ CXR w/ pulmonary infiltrates (does not have to be bilateral) ■ Increased oxygenation index
Pathogenesis	<ul style="list-style-type: none"> • No unifying pathophysiology for ARDS - can be direct injury (pneumonia, traumatic contusion) or indirect (systemic inflammation from sepsis) • Overall, insult causes alveolar cell damage filling of airspaces w/ exudate. Over ~3 weeks, granulation tissue formation occurs which leads to remodeling and fibrosis • Alveolar collapse leads to V/Q mismatch
Clinical Presentation	<ul style="list-style-type: none"> • Respiratory distress out of proportion to underlying disease • Hypoxemia • Decreased lung compliance
Diagnostic Studies	<ul style="list-style-type: none"> • Chest XR: commonly see bilateral infiltrates, although not required for diagnosis • ABG: high A-a gradient • PaO₂ to FiO₂ ratio is < 300
Treatment	<p>Lung protective ventilatory strategies: reduce ventilator-induced lung injury</p> <ul style="list-style-type: none"> ■ Maintain TV 4-6cc/kg, use PEEP to improve oxygenation (continue increasing PEEP if FiO₂ above 0.6). Target SpO₂ 88-94% (wean if >98%), keep FiO₂ < 0.6 ■ Permissive hypercapnia (pH 7.15-7.30), PaCO₂ 60s

Shock				
Definition	<p>Metabolic demands of body > delivered oxygen to tissues</p> <ul style="list-style-type: none"> ■ Oxygen delivery (DO₂) = content of arterial oxygen (CaO₂) x cardiac output (CO) ■ CaO₂ = (1.34 x Hgb x % O₂ Sat) + (0.003 x PaO₂) ■ CO = SV x HR, SV determined by preload, afterload, and contractility. 			
Type of Shock	Causes	Physiology	Findings	Treatment
Hypovolemic	<p>Dehydration Hemorrhage Osmotic diuresis Third-spacing fluid Burns</p>	<p>Not enough fluid in vasculature → decreased <u>preload</u> & CVP → low CO → decr. O₂ delivery</p>	<p>Dry mucous membranes, oliguria, weak pulses w/ delayed capillary refill</p>	<p>Fluid resuscitation, stop fluid losses if possible (e.g. treat bleeding). Rapid transfusion protocol if hemorrhage Rapid infuser in ICUs, ED, OR</p>

Shock continued on next page →

Shock

Type of Shock	Causes	Physiology	Findings	Treatment
Distributive	Septic shock Anaphylactic shock (anaphylaxis & septic shock cause vasodilation & cap. permeability) Neurogenic shock (loss of sympathetic innervation to vascular tone)	Poor tone & leaking of vasculature → low SVR → relative hypovolemia/ preload, low DBP. Contractility may be depressed later in sepsis presentation, CVP will vary.	Pounding pulses & brisk capillary refill if capillaries are leaky → warm extremities (** not always true in pediatric septic shock) Low DBP (especially neurogenic) Widened pulse pressure.	Vasopressors (new guidelines are epinephrine for “cold” and norepinephrine for “warm,” may also see dopamine and vasopressin) *Anaphylactic: EPI *Neurogenic: NE
Cardiogenic	Arrhythmias; Myocarditis; CHF; Cardiomyopathy; Trauma; **Cardiac tamponade; **Pulmonary embolism	Poor contractility or ability to relax → Ineffective systolic output → Decreased cardiac output w/ initial low CVP and high SVR	Weak pulses w/ narrow pulse pressure due to low systolic blood pressure; Pallor; Cold extremities; Delayed capillary refill; Signs of heart failure (respiratory distress, hepatomegaly, JVD)	LIMIT fluid resuscitation (5-10cc/kg); Inotropic agents (low dose dopamine, or epinephrine, less commonly dobutamine); Can consider milrinone if BP normal to decrease afterload
	**Obstructive causes of shock that affect the heart's ability produced adequate cardiac output	Pulmonary embolism, cardiac tamponade	Tamponade - Pulsus paradoxus or electrical alternans, narrow pulse pressure w/ increased diastolic	Specific to underlying cause.

Labs	<p>VBG w/ Lactate</p> <ul style="list-style-type: none"> Assess pH and bicarb to determine degree of metabolic acidosis due to anaerobic metabolism - note, bicarb on blood gas is calculated based on the pH and pCO₂ - obtain chemistry to measure directly Increased lactate associated w/ inadequate tissue O₂ delivery in shock states (but can also be elevated if not cleared appropriately, for example in liver failure) <p>Mixed venous saturation (ScvO₂) / arterial-venous O₂ difference</p> <ul style="list-style-type: none"> Normal is 70-75%, low in earlier shock (inadequate delivery for utilization), high is concerning for organ dysfunction (impaired O₂ utilization by cells due to injury (usually a bad sign)) Only interpretable from central line terminating in distal SVC, preferably RA; not useful from peripheral VBG True pulmonary arterial saturation (SvO₂) no longer routinely utilized <p>CBC and Blood Culture</p> <ul style="list-style-type: none"> WBC count to assess infection Hemoglobin to assess adequacy of oxygen carrying <p>Chem 10 w/ LFTs</p> <ul style="list-style-type: none"> Chemistry to assess solutes (Na, K, Cl, gluc), bicarb, renal function (BUN/Cr), intravascular volume status (BUN:Cr ratio) LFTs to assess liver damage
-------------	--

Shock

Septic Shock Treatment Algorithm

0 min

Recognize decreased mental status and perfusion.
Begin high flow O₂ 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, ScvO₂ > 70%* and CI 3.3 – 6.0 L/min/m²

Normal Blood Pressure
Cold Shock
ScvO₂ < 70%* / Hgb > 10g/dL
on Epinephrine?

Low Blood Pressure
Cold Shock
ScvO₂ < 70%* / Hgb > 10g/dL
on Epinephrine?

Low Blood Pressure
Warm Shock
ScvO₂ > 70%*
on Norepinephrine?

Begin Milrinone infusion.
Add Nitroso-vasodilator if CI < 3.3L/min/m² 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/m² add Dobutamine, Enoximone, Levosimendan, or Milrinone.

If euvolemic, add Vasopressin, Terlipressin, or Angiotensin. But, if CI decreases below 3.3 L/min/m² add Epinephrine, Dobutamine, Enoximone, Levosimendan.

Persistent Catecholamine-resistant shock?**Refractory Shock?**

Evaluate Pericardial Effusion or Pneumothorax,
Maintain IAP < 12mmHg

ECMO

Davis AL, Carcillo JA, Aneja RK et al. American College of Critical Care Medicine clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock. Critical care medicine. 2017 Jun 1;45(6):1061-93.

Consideration: There are times when blood products may be indicated in acute resuscitation if there are abnormal hemoglobin/hematocrit values but generally crystalloid is used over colloid and there is no benefit to albumin over crystalloid.