Acute Respiratory Distress Syndrome	
Definition	Acute respiratory failure not fully explained by cardiac etiology or fluid overload ▪ Excludes patients w/ perinatal pulmonary disease ▪ CXR w/ pulmonary infiltrates (does not have to be bilateral) ▪ Increased oxygenation index
Pathogenesis	• 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	• Respiratory distress out of proportion to underlying disease • Hypoxemia • Decreased lung compliance
Diagnostic Studies	• Chest XR: commonly see bilateral infiltrates, although not required for diagnosis • ABG: high A-a gradient • PaO2 to FiO2 ratio is < 300
Treatment	Lung protective ventilatory strategies: reduce ventilator-induced lung injury ▪ 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	Metabolic demands of body>delivered oxygen to tissues ▪ 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	Dehydration Hemorrhage Osmotic diuresis Third-spacing fluid Burns	Not enough fluid in vasculature → decreased preload & CVP → low CO → decr. O_2 delivery	Dry mucous membranes, oliguria, weak pulses w/ delayed capillary refill	Fluid resuscitation, stop fluid losses if possible (e.g. treat bleeding). Rapid transfusion protocol if hemorrhage Rapid infuser in ICUs, ED, OR

Shock continued on next page →