Fundamentals of Pediatric Intensive Care
RD's in Practice
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Nick Anas, MD
Pediatrician-in-Chief
Director Pediatric Intensive Care Unit
CHOC Children's Hospital
Clinical Professor of Pediatrics
Department of Pediatrics
University of California, Irvine

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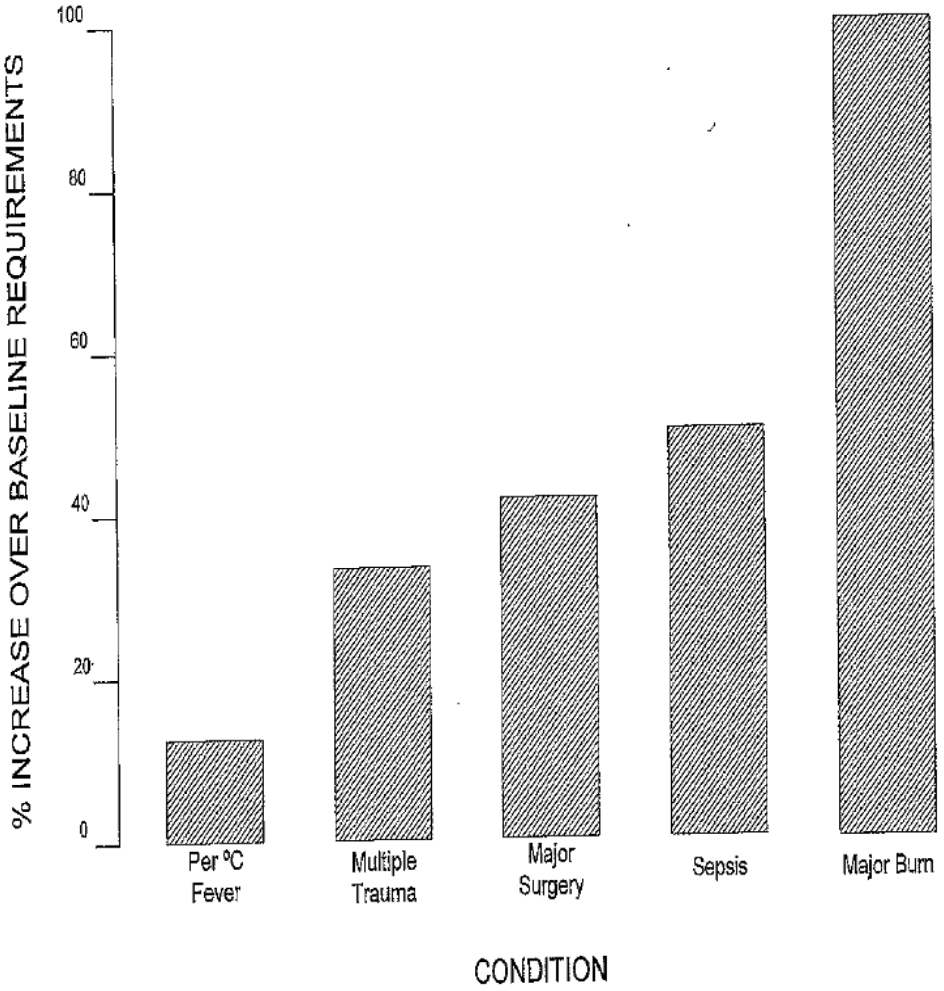
Critical Care – Nutritional Questions

1. Does nutritional support improve the outcome for children with critical illness?
2. What is the optimal nutritional support in a child with critical illness?
3. How do you monitor the adequacy of nutritional support in the child with critical illness?
4. Is there a benefit to enteral versus parenteral nutrition in the child with critical illness?
5. Should insulin be utilized to increase glucose administration in the child with critical illness?
6. Can you administer too much glucose, fat, or protein to the child with critical illness?

7. Is the triglyceride level the best test to monitor the adequacy of intralipid administration?
8. How do you determine the proper caloric input in the morbidly obese child?
9. How do you determine the proper caloric input in the severely malnourished critically ill child?
10. Is the metabolic demand in the critically ill child best monitored with the use of a metabolic cart?
11. In the management of a critically ill child, should metabolic demand be reduced rather than efforts made to increase oxygen delivery?

Caloric Requirement in Stress



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Shock

- **Hypovolemic**
- **Hemorrhagic**
- **Cardiogenic**
- **Distributive**
  - **Septic**
  - **Anaphylactic**

Hemodynamic Profiles of Shock

Type of Shock	Heart Rate	Cardiac Output	Ventricular Filling Pressures	Systemic Vascular Resistance	Pulse Pressure	$Sv_2/Scvo_2$
Cardiogenic	↑	↓	↑	↑	↓	↓
Hypovolemic	↑	↓	↓	↑	↓	↓
Distributive	↑	↑ or N[a]	↓	↓	↑	↑ or N[a]
Obstructive	↑	↓	↑ or N[a]	↑	↓	↓

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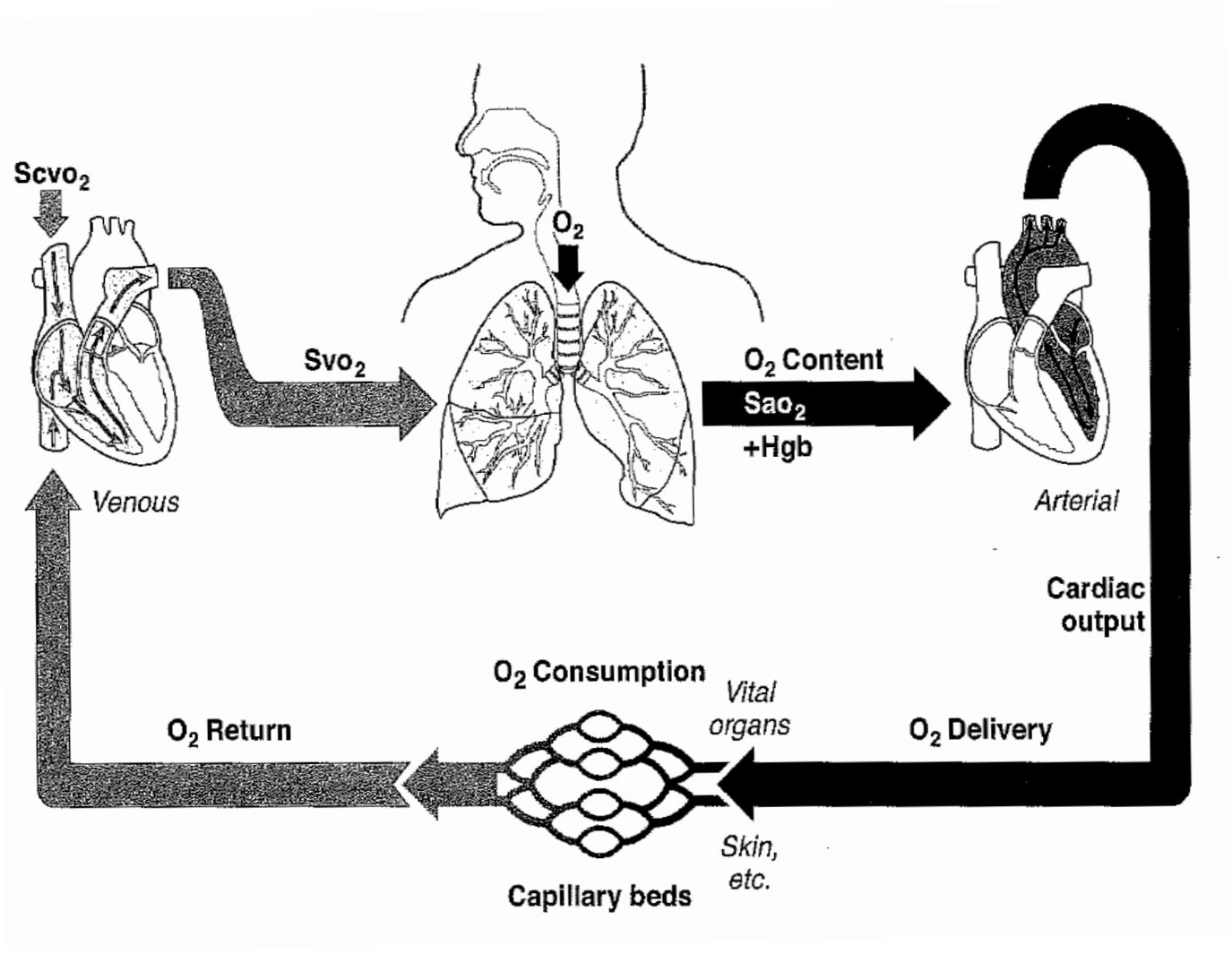
Oxygen Delivery

- **Arterial oxygen content x Cardiac output**

(CaO_2) x (CO)

Determinants of Oxygen Balance



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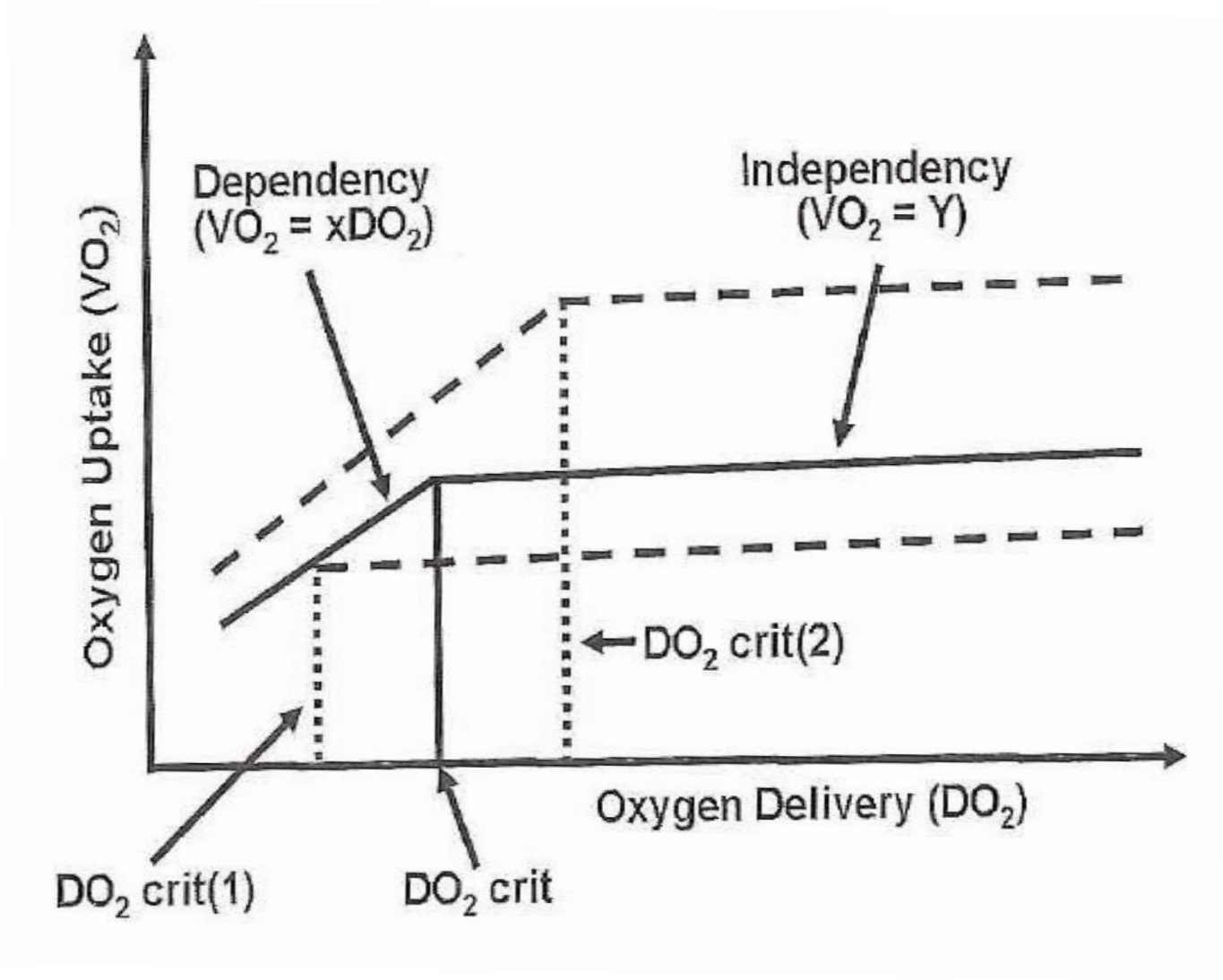
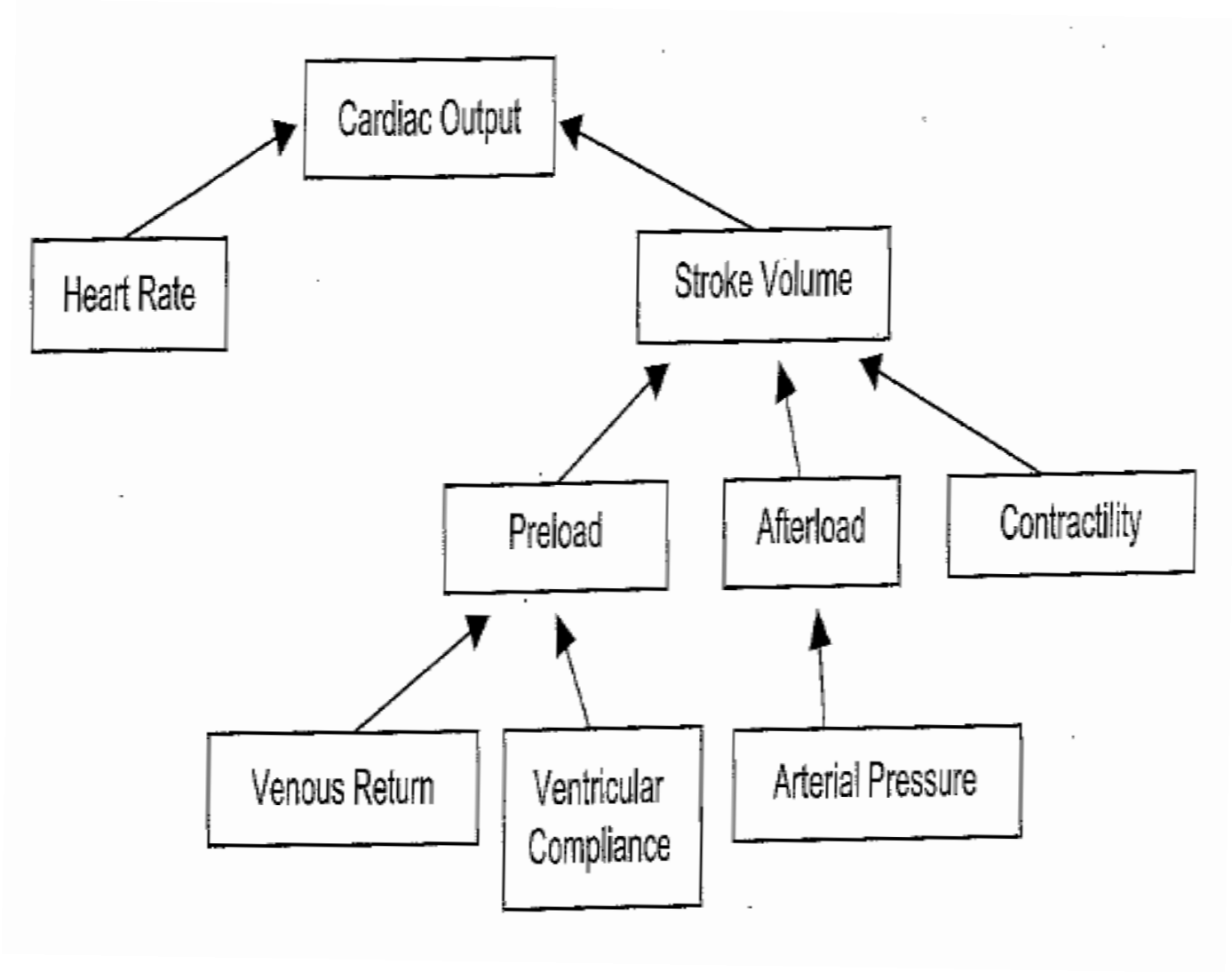
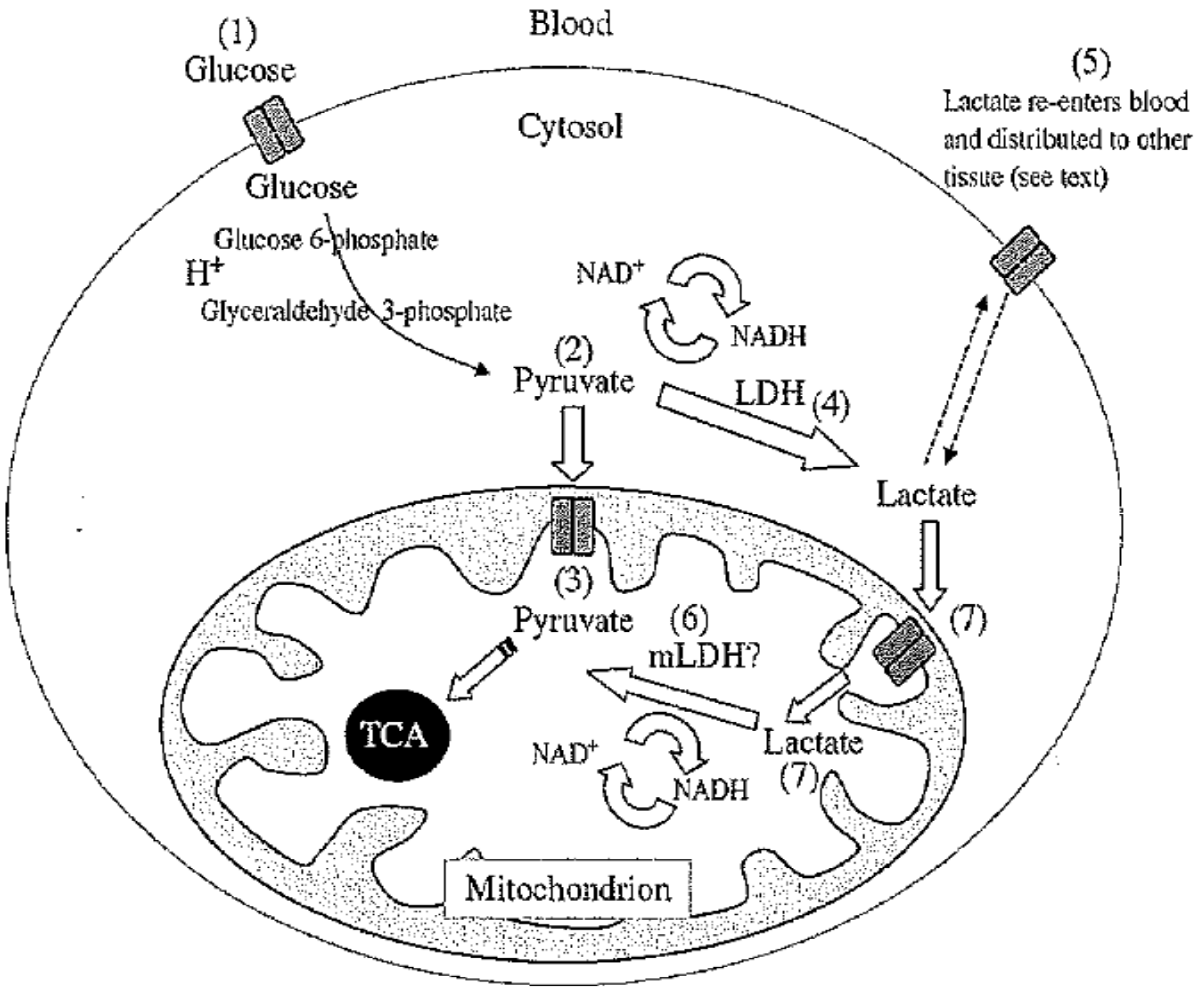
Oxygen Delivery and Extraction



Determinants of Cardiac Output



Lactate Metabolism and Prognosis



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Interventions for Managing Shock

Components	Intervention
Blood pressure	Fluids, vasopressor or vasodilator agents
Cardiac Output	
Preload Contractility Afterload	Fluids, vasodilator agents Inotropic agents Vasopressor or vasodilator agents
Oxygen Content	
Hemoglobin Hemoglobin saturation Oxygen demand	Blood transfusion Supplemental oxygen, mechanical ventilation Mechanical ventilation, sedation, analgesia, antipyretics

Sepsis Syndrome Criteria

I. Clinical evidence of infection (required)

II. Major criteria (two of four required)

Fever or hypothermia (temperature > 100.4 F or < 96 F)

Tachypnea or high minute ventilation (respiratory rate >20minute ventilation>10 L)

Tachycardia (pulse > 90 in absence of intrinsic heart disease or drug therapy inhibiting tachycardia)

Leukocytosis or leukopenia 9WBC>10,000/mm_3 or <4,000/mm_3) or >10% band forms on differential

III. Acute impairment of organ system function (one required)

Altered mental status (reduction in Glasgow come score >2 points)

Hypotension (SBP <90 mm Hg or fall in BP >40 mm Hg refractory to fluid challenge)

Impaired gas exchange or acute respiratory distress syndrome (PaO_2/FiO_2 ratio <300)

Metabolic acidosis/lactic acidosis

Oliguria or renal failure (urine output <0.5 mL/kg/h)

Hyperbilirubinemia

Coagulopathy (platelet count <80,000/mm_3 or a 50% decline within 48 h; INR >2.0:ptt >1.5 X control with elevated fibrin degradation products)

Respiratory Failure

- **Acute**
  - Hypoxemic
  - Hypercapnia
- **Chronic**
  - Broncho pulmonary dysplasia
  - Cystic fibrosis

Etiology Of Acute Respiratory Failure in Children

Location	Example
Upper airway obstruction	Infection (croup, epiglottitis, bacterial) Laryngotracheomalacia Foreign body Anaphylaxis
Lower airway obstruction	Asthma Bronchiolitis Cystic fibrosis
Respiratory lung disease	Acute respiratory distress syndrome Pleural effusion Pneumonia Pulmonary edema Abdominal compartment syndrome
Central nervous system disorder	Intracranial injury (hemorrhage, ischemia) Medication (sedatives) Metabolic encephalopathy
Peripheral nervous system and muscle disorders	Guillian Barre syndrome Muscular dystrophy Scoliosis Spinal cord injury Botulism Intoxications (i.e. Organophosphates)

ASSESSMENT OF GAS EXCHANGE

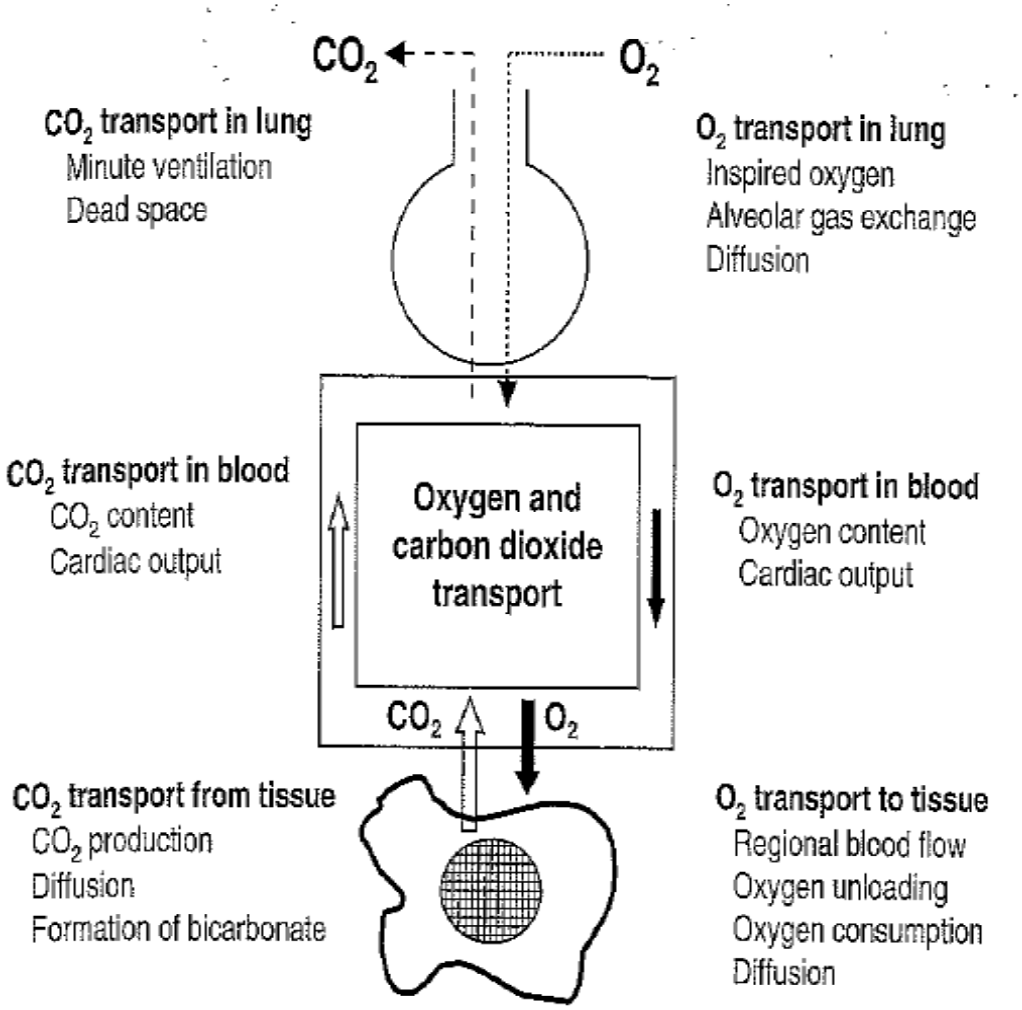
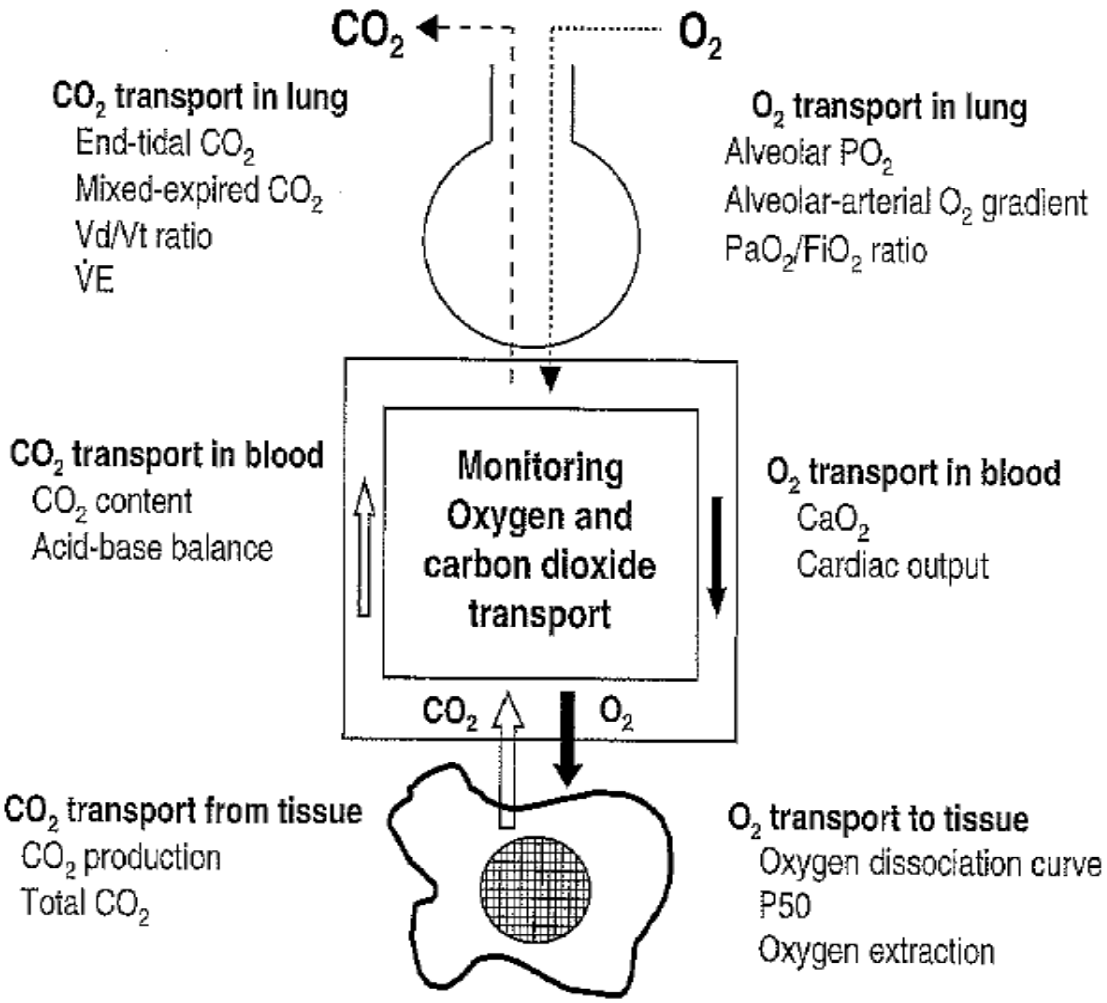


FIGURE 21.1. Physiology of oxygenation and ventilation. The primary functions of the cardiorespiratory system are to provide adequate oxygen to the tissues and to eliminate carbon dioxide via the lungs. This gas transfer process involves an elaborate interaction between the lungs, circulatory system, and the tissues throughout the body.

Cardiorespiratory Monitoring



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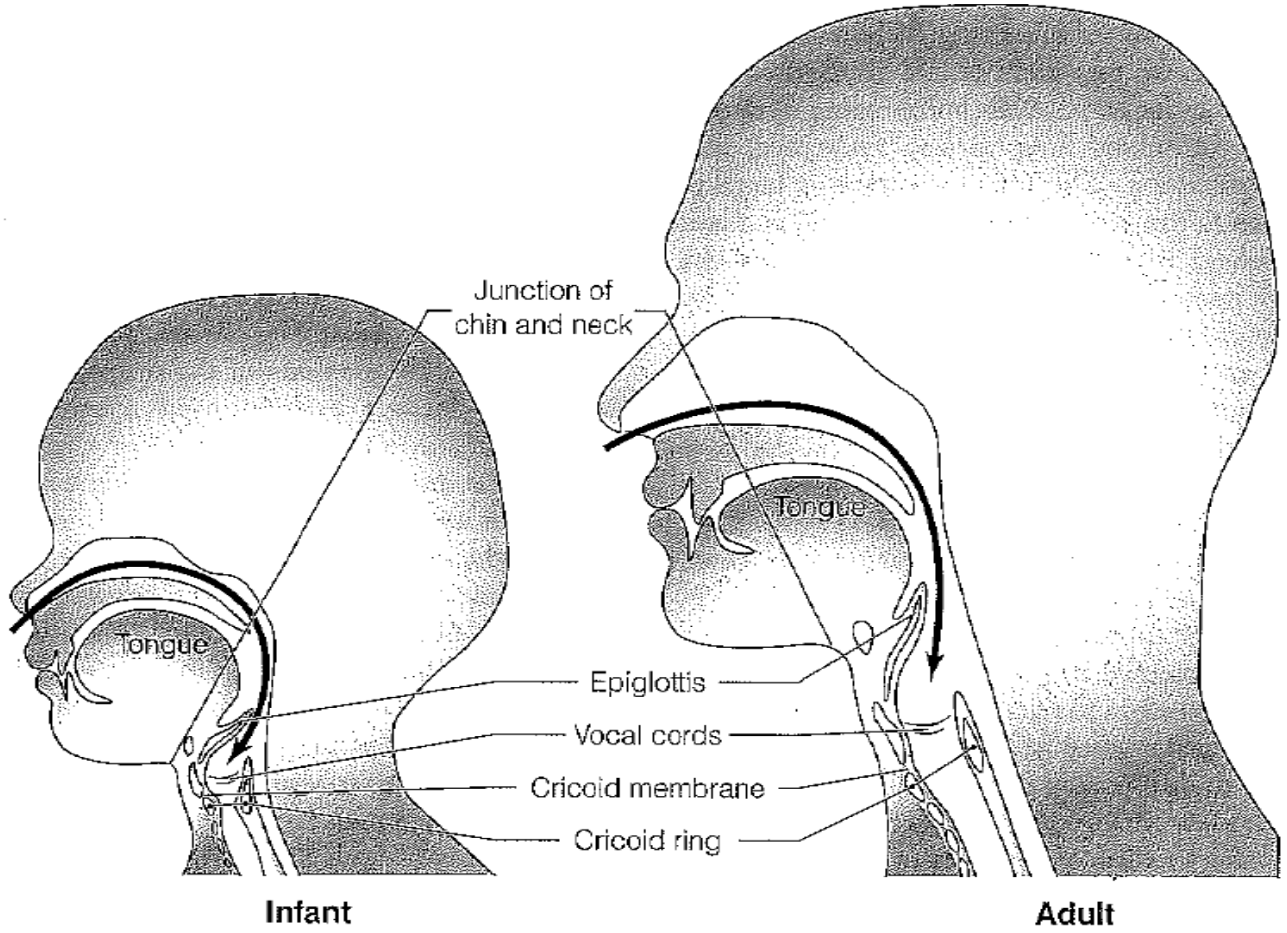
Anatomy of the Upper Airway



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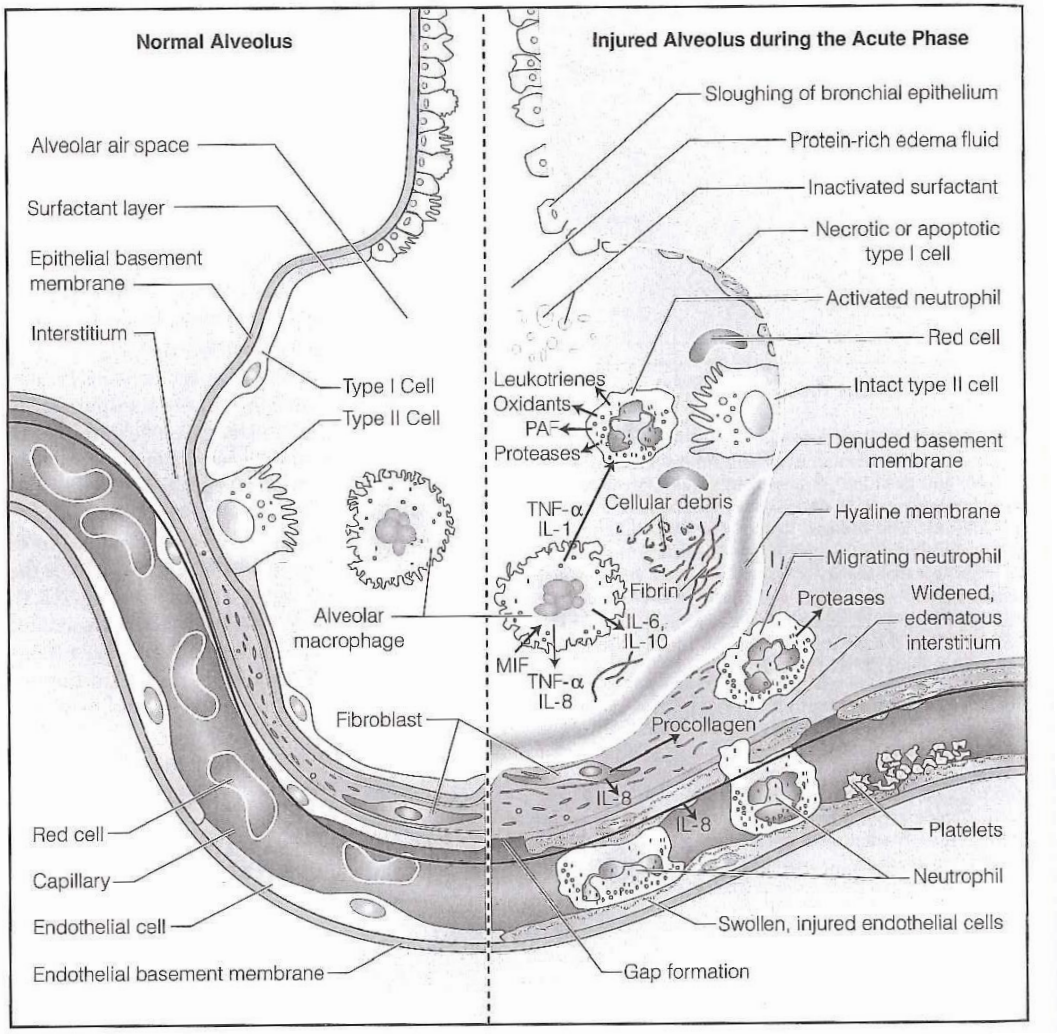
Acute Respiratory Distress Syndrome



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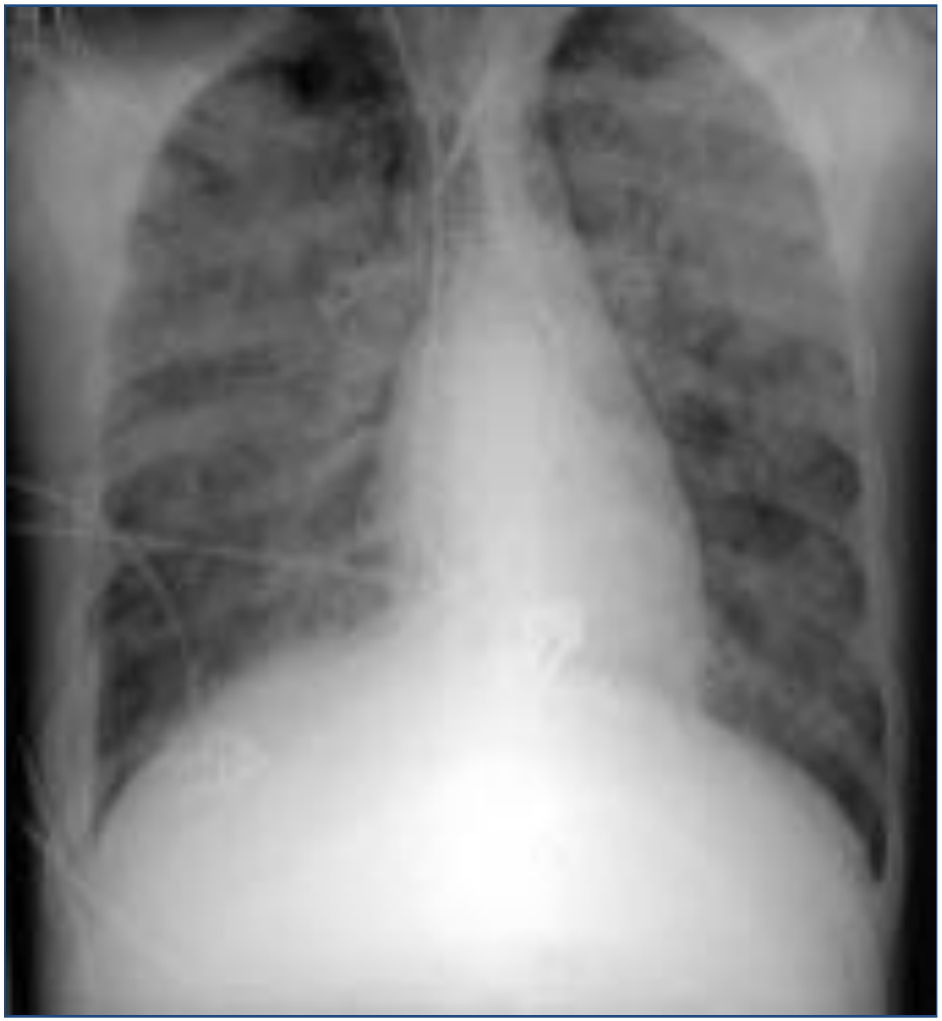
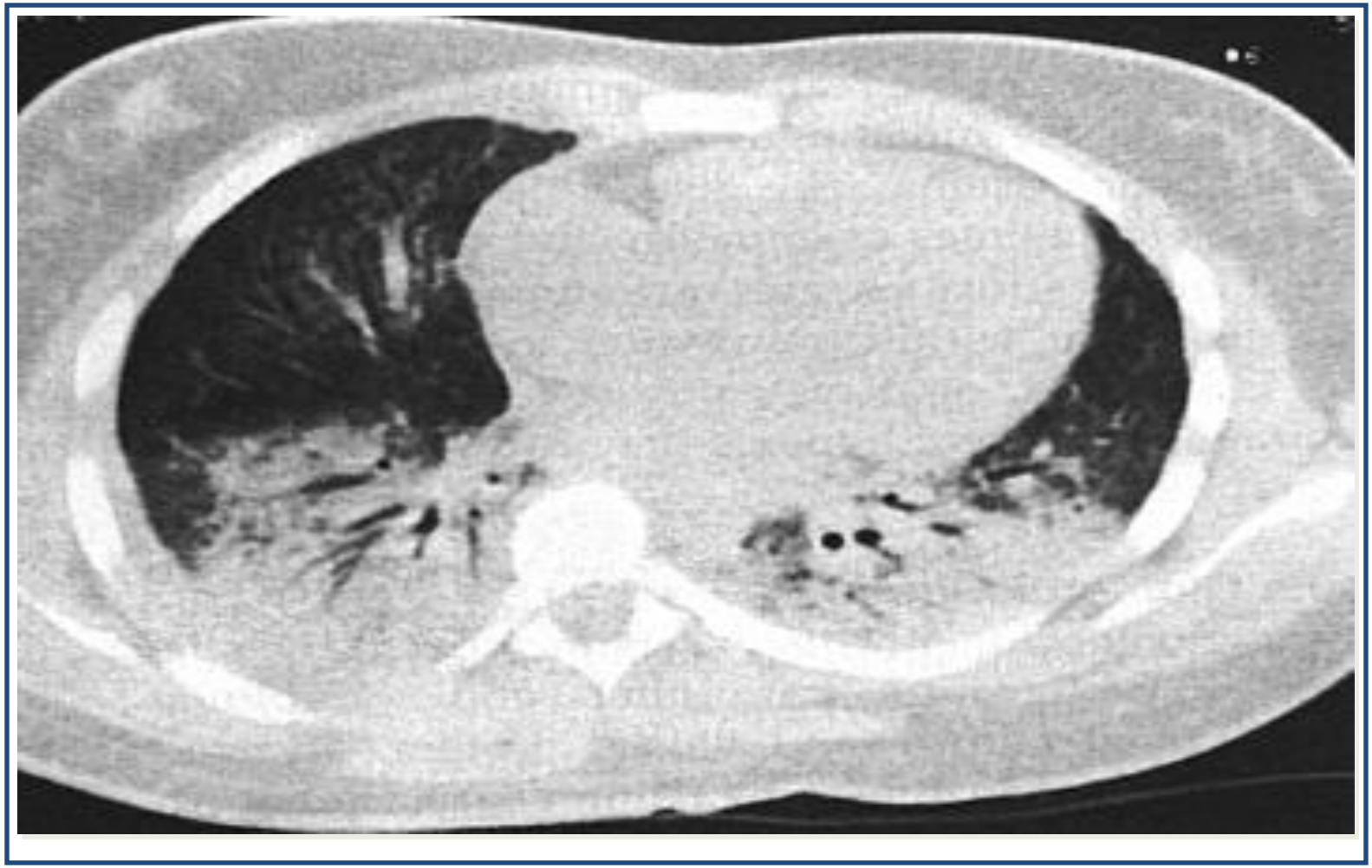
Pulmonary Edema Syndromes

- Cardiogenic
- Non-cardiogenic



ARDS: CT Scan of Chest



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Clinical Nutrition in Critically Ill Patients With Acute Respiratory Distress Syndrome (ARDS)

- Enteral nutrition, rather than parenteral nutrition, should be used in the vast majority of ARDS patients and should be started within 48 hours of ICU admission.
- Either full feeding or trophic feeding for the first few days of a patient's ICU stay is reasonable.
- After patients with shock are resuscitated and hemodynamically stable, they can safely receive enteral nutrition even if they are receiving stable lower doses of vasopressors.
- Consideration should be given to not monitor gastric residual volumes in most critically ill patients, because new evidence suggests this is safe and does not lead to worse outcomes.
- In reasonably well-nourished critically ill patients there is no role for parenteral nutrition either as a caloric supplement to enteral nutrition early during the ICU course or in patients who have a short-term relative contra-indication to enteral nutrition. The optimal role of parenteral nutrition in malnourished patients is currently being investigates.
- New data suggest that glutamine, antioxidants, and omega-3 fatty acids may not be beneficial in critically ill patients.

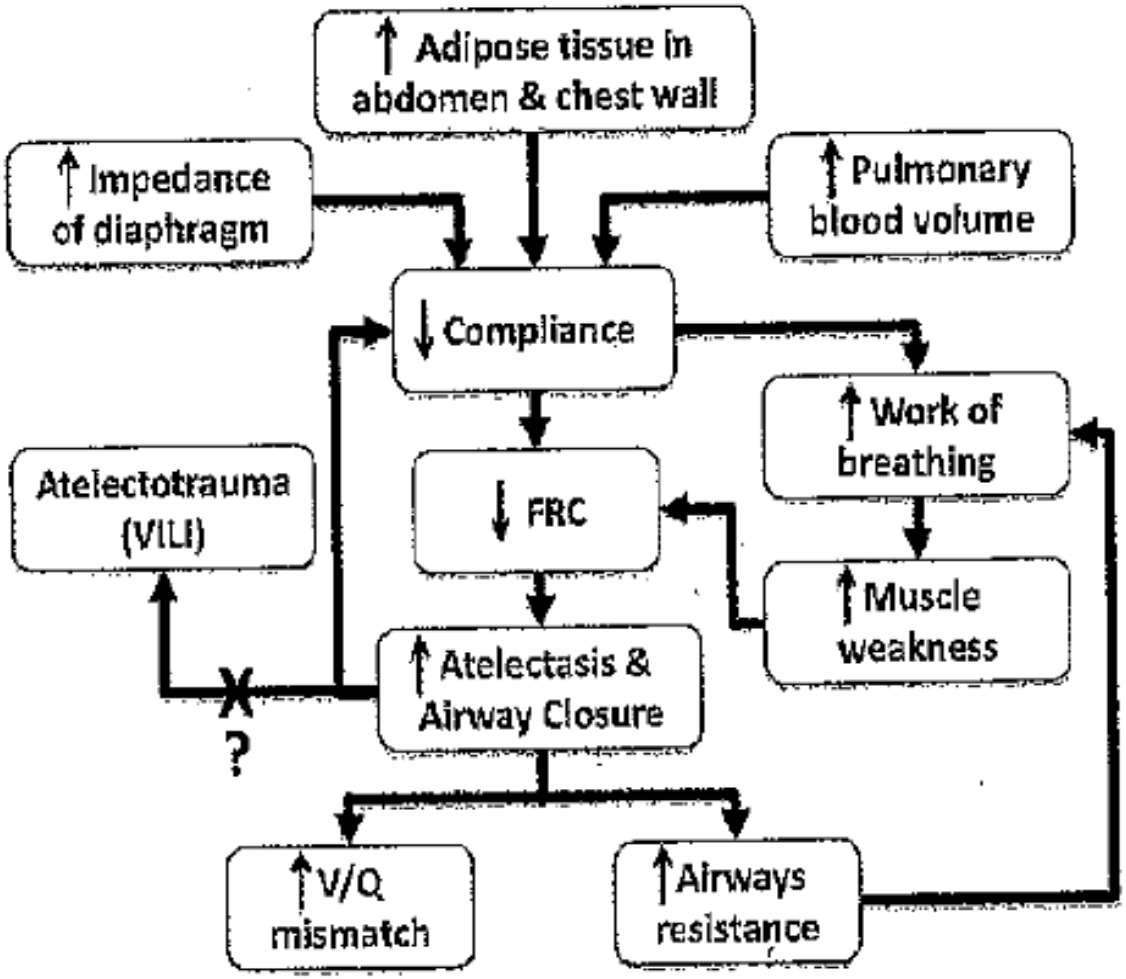
Nutrition in ARDS/critical illness: summary of the evidence

Intervention	Evidence
EN vs PN	In mechanically ventilated critically ill patients, EN is associated with fewer infectious complications but no change in survival
Early vs delayed EN	Among mechanically ventilated critically ill patients, early EN compared with delayed nutrient intake is associated with a trend toward mortality reduction and significant reduction in infectious complications, but no change in DMV or ICU LOS. Among 6 studies investigating EN vs no EN intravenous fluids, alone, trends in reduction of mortality and infectious complications hold.
Caloric prescription of EN	In patients with ARDS, there are no differences in VFDs, OFFDs, mortality, infectious complications, or long-term outcomes between patients receiving full or trophic enteral feeding for the first 6 d their ICU course.

Nutrition in ARDS/critical illness: summary of the evidence

Intervention	Evidence
Supplementation of calories with PN	Among general critically ill patients, early supplemental PN to meet calculated caloric goals does not change mortality or functional status at discharge, but does lead to longer ICU LOS, more infectious complications, longer DMV and renal replacement therapy, and higher costs. Among critically patients with a relative short-term contraindication to EN, early supplemental PN does not affect mortality, ICU LOS, or hospital LOS.
Glutamine	Controversial, but recent data suggest that glutamine should not be administered at higher doses in mechanically ventilated patients with multiple organ failure (increased 28-day and 6-month mortality).
Antioxidants	In mechanically ventilated critically ill patients, no clear benefit of antioxidants including selenium, as well as enteral zinc, B-carotene, and vitamins C and E.
Omega-3 fatty acids	Controversial, with studies of and enteral feeding formula containing omega -3s demonstrating benefits and studies of bolus enteral fish oil finding no benefit.

Obesity and its effects on pulmonary mechanics in ARDS



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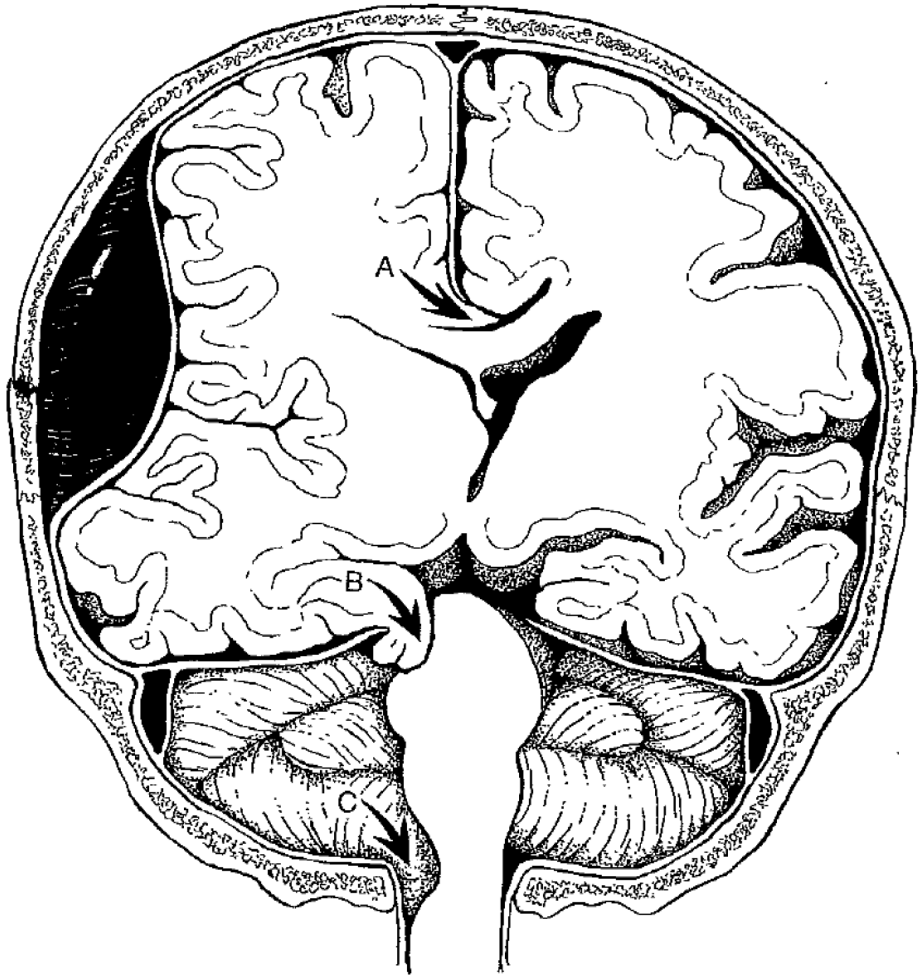
Central Nervous System Injuries

- **Trauma**
- **Infections**
- **Hypoxia-ischemia**
- **Malignancies**
- **Ingestions**

Intracranial Pressure

- **Brain**
- **Blood**
- **Cerebral spinal fluid (CSF)**
- **Extra**

Epidural Hematoma



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COMMON INJURY MECHANISMS AND CORRESPONDING INJURY PATTERNS IN CHILHOOD TRAUMA

Injury Mechanism	Details	Injury Pattern
Motor vehicle injury-occupant	Unrestrained Restrained	Head/neck injuries Scalp/facial lacerations Abdomen injuries Lower spine fractures
Motor vehicle injury-pedestrian	Single injury Multiple injuries	Lower extremity fractures Head/neck injuries Lower extremities fractures
Fall from height	Low Medium High	Upper extremity fractures Head/neck injuries Scalp/facial lacerations Upper extremity fractures Head/neck injuries Scalp/ facial lacerations Chest/abdomen injuries Extremity fractures

COMMON INJURY MECHANISMS AND CORRESPONDING INJURY PATTERNS IN CHILHOOD TRAUMA

Injury Mechanism	Details	Injury Pattern
Fall from bicycle	Unhelmeted Helmeted Handlebar impact	Head/neck injuries Scalp/facial lacerations Upper extremity fractures injuries Upper extremity fractures Abdomen injuries

Initial Assessment of Trauma

- **A**irway, maintenance with cervical spine precautions
- **B**reathing: ventilation and oxygenation
- **C**irculation: hemorrhage control
- **D**isability: brief neurologic examination
- **E**xposure/environment: clothing removed, avoiding hypothermia

General Principles of Managing Brain Injury

Promote Oxygen Delivery

1. Ensure systemic oxygen transport with adequate oxygenation, hemoglobin concentration, and cardiac output.
2. Ensures optimal blood pressure. Many primary insults are associated with hypertension that may be physiologic compensation or may be injurious. Elevated blood pressure may be undesirable in patients with unsecured aneurysms and recent intracranial hemorrhage. However, excessive lowering of blood pressure may results in secondary ischemia.
3. Avoid prophylactic or routine hyperventilation because an increase in extracellular brain pH constricts responsive vessels and may reduce cerebral blood flow to ischemic zones. Brief hyperventilation while instituting other methods to lower elevated intracranial pressure may be lifesaving in the patient with evidence of herniation.
4. Ensure euvolemia because hypoxemia may results in systemic hypotension and hypo perfusion of brain tissue.
5. Rapid-sequence intubation should be used for patients with increased intracranial pressure. Consider administration of intravenous lidocaine (1.5 mg/kg) or intravenous thiopental (5mg/kg) to blunt the rise n intracranial pressure. Consider administration of intravenous lidocaine (1.5 ng/kg) or intravenous thiopental (5 mg/kg) to blunt the rise in intracranial pressure associated with intubation.
6. Institute nimodipine immediately in patients with subarachnoid hemorrhage.

General Principles of Managing Brain Injury

Prevent Abnormal Oxygen Demands

1. Avoid fever. Fever increases metabolic demand, resulting in neuronal injury and elevating intracranial pressure.
2. Avoid seizures, Prophylactic anticonvulsant administration is indicated after moderate or severe traumatic brain injury to prevent seizures in the first week, but the available evidence does not support longer use in hear trauma or use in other neurologic injuries.
3. Avoid anxiety, agitation or pain. Neuronal oxygen consumption may be decreased by an antianxiety agent, sedation, and analgesia.
4. Avoid shivering
5. Minimize stimulation, particularly fro the first 72 hours.

GLASGOW COMA SCALE AND MODIFICATION FOR CHILDREN

Sign	Glasgow coma scale	Modification for children	Score
Eye opening	Spontaneous To command To pain None	Spontaneous To Sound To pain None	4 3 2 1
Verbal response	Oriented Confused Disoriented Incomprehensible sounds None	Age-appropriate verbalization, orients to sound, fixes and follows, social smile Cries, but consolable Irritable, persistent cries, inconsistently consolable Inconsolable crying , unaware of environment or parents, restless, agitated None	5 4 2 1

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GLASGOW COMA SCALE AND MODIFICATION FOR CHILDREN

Sign	Glasgow coma scale	Modification for children	Score
Motor response	Obeys commands	Obeys commands, spontaneous movement	6
	Localizes pain	Localizes pain	5
	Withdraws	Withdraws	4
	Abnormal flexion to pain	Abnormal flexion to pain	3
	Abnormal extension	Abnormal extension	2
Best total score			15

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ADVANTAGES OF ENTERAL NUTRITION

Maintains gut mucosal structure
Decreases bacteria and toxin translocation
Promotes enteric hormone secretion
Eliminates need for central catheter Reduced risk of sepsis and line related complications
Buffers gastric acid
Less likely to induce hyperglycemia
Provides unique and complex nutrients not available by TPN Glutamine Dietary fiber Medium-and short-chain fatty acids
Dramatically reduces cost

Gastrointestinal Complications of Enteral Nutrition

Complications	Probable cause	Prevention /treatment
Diarrhea	Overly fast infusion High-osmolarity formula Lactose intolerance Formula with high fat content Formula allergy/intolerance Medications (metoclopramide, Aminophylline, erythromycin, sorbitol, xylitol, magnesium, phosphorus)	Decrease Infusion speed Decrease dilution or formula type Use lactose-free formula Choose lower fat formula Use hydrolyzed protein formula Avoid enteral diarrhea causing medications, consider change to IV form
	Change in intestinal flora due to antibiotic therapy	Consider vancomycin or metronidazole orally (if c.diff)-?
	Potential bacterial and diet contamination	Choose closed system or limit hang time to 8 hours RTF or 4 hours with prepared/mixed formulas (aseptic technique)

Gastrointestinal Complications of Enteral Nutrition

Complications	Probable cause	Prevention /treatment
Abdominal distension	Use of antacids, antibiotics, rapid infusion, hypertonic or high fat formula, narcotic use, ileus	Consider suspending drugs Decrease flow or volume of infusion, consider formula change, review use of drugs causing gastric atonia
Nausea and Vomiting	Multifactorial	Decrease flow; consider change of formula, exclude infectious process.
Intestinal Obstipation	Low fiber formula, dehydration	Consider fiber-rich diet, maintain adequate hydration

Factors in the intensive care unit that may cause decreased gastric emptying and thus affect residual volumes

- Hyperglycemia
- Opiates
- Dopamine
- Increased intracranial pressure
- Electrolyte abnormalities
- Ischemia
- Hypoxia
- Sepsis
- Burns, trauma, surgery
- Hyperosmolar formulas

QUESTIONS ?

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THANK YOU

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