Hypoxemia

Workup	Get PaCO2 level, A-a gradient, and response to supplemental O2.
If PaCO2 is elevated	• **Hypoventilation is occurring**. • If the A-a gradient is normal, hypoventilation or low inspired PO2 is the only mechanism. • If the A-a gradient is increased, hypoventilation is not the lone mechanism of hypoxemia.
If PaCO2 is not elevated	• No hypoventilation. • If A-a gradient is not increased, low inspired PaO2 is the cause of hypoxemia.
If A-a gradient is increased	• With low to normal PaCO2. See if the lowered PaO2 is improved with supplemental O2. If so, there is V/Q mismatch. If it does not improve, shunting is present.
V/Q Mismatch	• Typically leads to **hypoxia without hypercapnia** (PaCO2 is usually low to normal). • Most common mechanism of hypoxemia (esp. in chronic lung disease). • **Responsive to supplemental oxygen**. • **Ventilation without perfusion**.

Hypoxemia (cont)

Intrapulmonary Shunting	• Little or **no ventilation in perfused areas** due to **collapsed or fluid-filled alveoli**. • Venous blood is shunted into the arterial circulation without being oxygenated. • **Perfusion without ventilation**. •Causes include atelectasis or fluid buildup in alveoli (pneumonia or edema) or direct right to left intracardiac blood flow in congenital heart disease. • **No responsive to supplemental oxygen**.
Hypoventilation	Leads to hypercapnia with secondary hypoxemia.
Increased CO2 production	In sepsis, DKA, hypertermia, etc. Leads to **hypercapnic respiratory failure**.
Diffusion Impairment	In ILD causes **hypoxemia without hypercapnia**.

Hypoxemic Failure

Hypoxia	**PaO2<60mmHg and PaCO2>50mmHg**. • Severe hypoxemia can result in irreversible damage to CNS and CVS, and must be corrected rapidly. • O2 saturation is <90% despite FiO2>0.6. • Causes include lung pathology like **ARDS**, severe pneumonia, and pulmonary edema. • **V/Q mismatch and intrapulmonary shunting** are the major pathophysiologic mechanisms.

Oxygen Delivery

Low Flow	• Nasal cannula has flow rate of 1-6L/min, FiO2 up to 0.40, is easy to use and comfortable. • Simple face masks have a flow rate of 1-10L/min, FiO2 of 0.40-0.60, and can deliver higher flow rates than nasal cannula.
High Flow	• Venturi masks have a flow rate of 4-10L/min, FiO2 up to 0.50 with precise measurements, and are preferred in CO2 retainers due to higher precision and control of oxygenation. • Nonrebreather masks have flow rates up to 15L/min, FiO2 up to 0.70-0.80, and can achieve higher FiO2 at lower flow rates.
NPPV	• BIPAP or CPAP via nasal mask or full-face mask. Indicated in patients in impending respiratory failure to avoid intubation or mechanical ventilation. • Success is highest in **hypercarbic respiratory failure (esp. COPD)**. Patient must be neurologically intact, awake, cooperative, and able to protect their airway.

Acute Respiratory Failure

Pathophysiology	Results when there is **inadequate oxygenate of blood or inadequate ventilation or both**.
Symptoms	**dyspnea is the first**. Cough may or may not be present, depending on the underlying cause.



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Acute Respiratory Failure (cont)

Signs	**Inability to speak in complete sentences**, use of **accessory muscles tachypnea**, tachycardia, cyanosis, impaired mentation (due to fatigue or hypercapnia, of if cause of respiratory failure is CNS depression).
Causes	• CNS Causes: depression or insult from drug overdose, stroke, or trauma. • Neuromuscular Causes: myasthenia gravis, polio, Guillain-Barre syndrome, amyotrophic lateral sclerosis. • Upper Airway Causes: obstruction due to stenoses, spasms, or paralysis. • Thorax and Pleural Causes: mechanical restriction due to kyphoscoliosis, flail chest or hemothroax. • CVS and Heme Causes: CHF, valvular diseases, PE, and anemia. • Lower Airway Causes: asthma, COPD, pneumonia, ARDS.
Diagnosis	ABG, CXR, CT, CBC, BMP, and **consider cardiac enzymes** if pulmonary edema is suspected.

Hypercarbic Failure

Treatment

• Treat underlying disorder with **bronchodilators, corticosteroids, antibiotics**, etc.
• Provide supplemental oxygen if hypoxemic. In hypoxemic failure, **use lowest concentration** of oxygen that provides sufficient oxygenation to avoid oxygen toxicity (due to free radicals).

• In hypercarbic failure, traditionally do not give high concentrations of O2, especially in patients with COPD to not depress respiratory drive. Apply NPPV only for conscious patients. Intubation and mechanical ventilation

Acute Respiratory Distress Syndrome

Pathophysiology	• A diffuse inflammatory process involving both lungs. **Neutrophils** are activated in the systemic or pulmonary circulations. • Not a primary disease but a disorder that arises from other conditions that cause a widespread inflammatory process. • There is **massive intrapulmonary shunting**, decreased pulmonary compliance (increased work), **increased dead space** (secondary to obstruction and destruction of pulmonary capillary bed), **low VC, and low FRC**.

Acute Respiratory Distress Syndrome (cont)

Intrapulmonary Shunting	• Severe hypoxemia with **no significant improvement on 100% oxygen**. • Shunting secondary to **widespread atelectasis**, collapse of alveoli, and surfactant dysfunction. • Interstitial edema and alveolar collapse are due to **increased lung fluid that leads to stiff lungs,** an increase in A-a gradient, and ineffective gas exchange. • Effects of the increase in pulmonary fluid are identical to those in cardiogenic pulmonary edema, but the cause is **increased alveolar capillary permeability**
Causes	• Sepsis is the most common (which can itself be secondary to pneumonia, urosepsis, wound infections, etc). Aspiration of gastric contents, severe trauma, fractures (femur, pelvis), **acute pancreatitis**, multiple or massive transfusions, near-drowning, drug overdose, toxic inhalation, intracranial HTN, and cardiopulmonary bypass.

Hyper capnia	• **PCO2>50mmHg**. • Severe hypercapnia can lead to dyspnea and vasodilation of cerebral vessels (with increased ICP and subsequent papilledema, HA, impaired consciousness, and finally coma). • A **failure of alveolar ventilation**. • Either a **decrease in minute ventilation** or an **increase in physiologic dead space** that leads to **CO2 retention** and eventually hypoxemia. • May be caused by underlying lung disease (COPD, asthma, CF, severe bronchitis) or without lung disease (patients with impaired ventilation due to neuromuscular diseases, CNS depression, mechanical restriction of the chest, or respiratory fatigue). Also increased CO2 production or diffusion impairment. • Hypercapnia can be **caused by hypoventilation** (secondary to a variety of causes). •**Respiratory acidosis occurs when hypercapnia** is present. • If chronic acidosis, renal compensation occurs and acidosis is less severe.

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Acute Respiratory Distress Syndrome (cont)

Signs	• Dyspnea, tachypnea, tachycardia due to increased work of breathing. • Progressive hypoxemia not responsive to supplemental oxygen **(ratio of PaO2 to FiO2<200**). • Patients are difficult to ventilate because of **high peak airway pressures due to stiff, noncompliant lungs**.
Diagnosis	• Hypoxemia that is **refractory to oxygen therapy** (ratio of PaO2/FiO2<200-300), bilateral diffuse pulmonary infiltrates on CXR, no evidence of CHF (PCWP <18mmHg).
CXR	• Shows **diffuse bilateral pulmonary infiltrates**. Improvement on CXR follows clinical improvement after 1-2 weeks.
Arterial Blood Gas	• **Hypoxemia (PaO2<-60mmHg**). • Initially, **respiratory alkalosis (PaCo2<40)** is present, which gives way to respiratory acidosis as the work of breathing increases and PaCO2 increases. • If patient is septic, metabolic acidosis may be present with or without respiratory compensation.

Acute Respiratory Distress Syndrome (cont)

Pulmonary Artery Catheter	• Enables a determination of PCWP, which **reflects left heart filling pressures** and is an **indirect marker of intravascular volume status**. • The most useful parameter in differentiating ARDS from cardiogenic pulmonary edema. • **If PCWP is <18mmHg, ARDS is more likely, whereas if PCWP is >18mmHg, cardiogenic is more likely**.
Bronchoscopy	**With bronchoalveolar lavage**. May be considered if patient is acutely ill and infection is considered. Fluid collected can be cultured and analyzed for cell differential, cytology, Gram stain, and silver stain.

Acute Respiratory Distress Syndrome (cont)

Treatment	• Oxygenation >90%. **Mechanical ventilation with PEEP** is usually required **(increases lung volume by opening collapsed alveoli and decreasing shunting**). • Avoid volume overload and maintain low-normal intravascular volume (PCWP 12-15mmHg). • Vasopressors may be needed to maintain BP. • Patients with sepsis have high fluid requirements, so this might be difficult. Treat underlying cause. Tube feedings are preferred over parenteral nutrition.

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