RESPIRATORY FAILURE

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OUTLINES

- **Definition**
- **Etiology and Types of RF**
- **Hypoxemic RF (ARDS)**
- **Vetilatory Failure (Hypercapnic)**
- **Other types of Respiratory Failure**
- **Diagnosis**
- **Management**

DEFINITION

- **Respiratory failure is a life-threatening condition either due to :**
- **1) impairment of oxygenation (Failure to oxygenate the body: defined as a PaO of < 60 mmHg)**
- **2)carbon dioxide elimination (defined as a PaCO of > 50 mmHg)**
- **3) or both.**

- **so we can define Respiratory failure: acute or chronic inability of the respiratory system to maintain gas exchange.**

ETIOLOGY AND TYPES

- **Types of respiratory failure**
- **Type I (hypoxemic respiratory failure) :Acute Hypoxemic Respiratory Failure (AHRF, ARDS)**
- **Type 2 (hypercapnic respiratory failure) :Ventilatory Failure**

❑ **Other Types of Respiratory Failure:**

- Perioperative respiratory failure

- Hypoperfusion

Types of respiratory failure		
	Type 1 (hypoxemic respiratory failure)	**Type 2 (hypercapnic respiratory failure)**
Definition	Respiratory failure characterized by hypoxemia and normocapnia or hypocapnia on arterial blood gas analysis	**Respiratory failure characterized hypercapnia and normoxemia or hypoxemia on arterial blood gas analysis**
PaO	**↓ (< 60 mmHg)**	**Normal or ↓ (< 80 mm Hg)**
PaCO	**Normal or ↓ (< 33 mm Hg)**	**↑ (> 50 mmHg)**

ACUTE HYPOXEMIC RESPIRATORY FAILURE (AHRF,ARDS)

- **Acute hypoxemic respiratory failure is defined as severe hypoxemia (PaO2 < 60 mmHg)**

- **It is caused by:**
- **-Impaired alveolar diffusion(e.g., due to pulmonary edema, severe pneumonia, pulmonary hemorrhage , idiopathic pulmonary fibrosis)**
- **Right-to-left shunts:**

  1)intrapulmonary shunting of blood with resulting in ventilation-perfusion (V/Q) mismatch due to airspace filling or collapse or possibly airway disease (., due to ARDS, pulmonary contusions/hemorrhage, lung collapse severe pneumonia, pulmonary edema, pulmonary embolism, atelectasis)

- **2)intracardiac shunting of blood from the right- to the left-sided circulation (e.g., due to atrial septal defect, VSD, PDA)**
- Deoxygenated venous blood bypasses the lungs and enters the systemic circulation. This phenomenon is termed Eisenmenger syndrome.

EXPLANATION :

- **Airspace filling in acute hypoxemic respiratory failure (AHRF) may result from:**

❖ Elevated alveolar capillary hydrostatic pressure, as occurs in left ventricular failure (causing pulmonary edema) or hypervolemia

❖ Increased alveolar capillary permeability, as occurs in any of the conditions predisposing to acute respiratory distress syndrome (ARDS)

❖ Blood (as occurs in diffuse alveolar hemorrhage) or inflammatory exudates (as occur in pneumonia or other inflammatory lung conditions)

TYPES OF HYPOXEMIA

Causes of hypoxemia			
	Example	**A-a gradient**	**Corrects with supplemental O_2?**
Reduced PiO_2	High altitude	Normal	Yes
Hypoventilation	CNS depression, neuromuscular weakness	Normal	Yes
V/Q mismatch	Pulmonary embolism, COPD	Increased	Yes
Diffusion limitation	Emphysema, ILD	Increased	Yes
Intrapulmonary shunt (V/Q = 0)	Pneumonia, pulmonary edema, atelectasis	Increased	No
Intracardiac shunt (right to left)	Tetralogy of Fallot, Eisenmenger syndrome	Increased	No

A-a gradient = alveolar-to-arterial oxygen gradient; **ILD** = interstitial lung disease; **PiO_2** = partial pressure of inspired oxygen; **V/Q** = ventilation/perfusion ratio.

PATHOPHYSIOLOGY

- The pathophysiologic mechanisms that account for the hypoxemia observed in a wide variety of diseases are V/Q mismatch and shunt.
- These 2 mechanisms lead to widening of the alveolar-arterial PO2 gradient, which normally is less than 15 mm Hg.
- They can be differentiated by assessing the response to oxygen supplementation or calculating the shunt fraction after inhalation of 100% oxygen.
- In most patients with hypoxemic respiratory failure, these 2 mechanisms coexist

1. V/Q mismatch

The most common cause of hypoxemia.

Alveolar units may vary from low-V/Q to high-V/Q in the presence of a disease process. The low-V/Q units contribute to hypoxemia and hypercapnia, whereas the high-V/Q units waste ventilation but do not affect gas exchange unless the abnormality is quite severe.

The low V/Q ratio may occur either from a decrease in ventilation secondary to airway or interstitial lung disease or from overperfusion in the presence of normal ventilation. The overperfusion may occur in case of pulmonary embolism, where the blood is diverted to normally ventilated units from regions of lungs that have blood flow obstruction secondary to embolism.

2. Shunt

Defined as the persistence of hypoxemia despite 100% oxygen inhalation.

The deoxygenated blood (mixed venous blood) bypasses the ventilated alveoli and mixes with oxygenated blood that has flowed through the ventilated alveoli, consequently leading to a reduction in arterial blood content.

Anatomic shunt exists in normal lungs because of the bronchial and thebesian circulations, which account for 2-3% of shunt. A normal right-to-left shunt may occur from atrial septal defect, ventricular septal defect, patent ductus arteriosus, or arteriovenous malformation in the lung.

Shunt as a cause of hypoxemia is observed primarily in pneumonia, atelectasis, and severe pulmonary edema of either cardiac or noncardiac origin. Hypercapnia generally does not develop unless the shunt is excessive (> 60%).

SYMPTOMS AND SIGNS OF AHRF

- Acute hypoxemia may cause :
- Dyspnea ,tachypnea
- Pleuritic chest pain
- Tachycardia and arrythmia
- Cynosis ,confusion ,coma ,restlessness and anxiety
- Inspiratory opening of closed airways causes crackles, detected during chest auscultation
- Jugular venous distention occurs with high levels of positive end-expiratory pressure (PEEP) or right ventricular failure.

VENTILATORY FAILURE

- **Ventilatory failure is a rise in PaCO2 (hypercapnia) that occurs when the respiratory load can no longer be supported by the strength or activity of the system**
- **It is caused by:**

- **Pulmonary causes :**
- **Airway obstruction (hypoventilation) and/or increased physiologic dead space**(e.g., due to exacerbation of COPD, acute severe bronchial asthma, bronchiolitis)

VENTILATORY FAILURE

- **Extrapulmonary causes**

1. **CNS depression (e.g., due to narcotic or sedative overdose, brain trauma/herniation, stroke)**

1. **Respiratory muscle weakness (e.g., due to myasthenia gravis, Guillain-Barre syndrome, myopathies, ALS, high cervical spinal cord injury, poliomyelitis)**

1. **Decreased chest wall compliance (e.g., due to rib fractures, tension pneumothorax, tetanus, seizures)**

1. **Increased O2 consumption and/or CO2 production (e.g., due to severe sepsis, toxic shock syndrome, cardiogenic shock, multiorgan dysfunction)**

1. **Electrolyte disturbances (e.g., anorexia nervosa)**

PATHOPHYSIOLOGY

- Arterial hypoxia with hypercapnia (type II respiratory failure) is seen in conditions that cause generalised, severe ventilation–perfusion mismatch, leaving insufcient normal lung to correct PaCO2, or any disease that reduces total ventilation.
- The latter includes not just diseases of the lung but also disorders affecting any part of the neuromuscular mechanism of ventilation. Acute type II respiratory failure is an emergency requiring immediate intervention. It is useful to distinguish between patients with high ventilatory drive (rapid respiratory rate and accessory muscle recruitment) who cannot move sufficient air, and those with reduced or inadequate respiratory effort.

PATHOPHYSIOLOGY OF VENTILATORY FAILURE

- Hypercapnia occurs when alveolar ventilation either **falls** or **fails** to rise adequately in response to increased carbon dioxide production.
- A fall in alveolar ventilation results from :
- a decrease in minute ventilation
- or an increase in dead space ventilation without appropriate compensation by increasing minute ventilation.
- Ventilatory failure can occur when there is excessive load on the respiratory system (eg, resistive loads or lung and chest wall elastic loads) versus neuromuscular competence for an effective inspiratory effort.
- When the minute ventilation load increases (eg, as occurs in sepsis), a compromised respiratory system may not be able to meet this increased demand (for causes, see figure The balance between load and neuromuscular competence).

PATHOPHYSIOLOGY OF VENTILATORY FAILURE

- **Physiologic dead space** is the part of the respiratory tree that does not participate in gas exchange. It includes:
  - Anatomic dead space (oropharynx, trachea, and airways)
  - Alveolar dead space (ie, alveoli that are ventilated but not perfused)
- Physiologic dead space can also result from shunt or low ventilation/perfusion (V/Q) if patients cannot increase their minute ventilation appropriately.
  The physiologic dead space normally is about 30 to 40% of tidal volume but increases to 50% in intubated patients and to >70% in massive pulmonary embolism, severe emphysema, and status asthmaticus.
  Thus, for any given minute ventilation, the greater the dead space, the poorer the carbon dioxide elimination.

PATHOPHYSIOLOGY OF VENTILATORY FAILURE

- Increased carbon dioxide production, as occurs with fever, sepsis, trauma, burns, hyperthyroidism, and malignant hyperthermia, is not a primary cause of ventilatory failure because patients should increase their ventilation to compensate.
- Ventilatory failure associated with these problems results only when the ability to compensate is compromised.
- Hypercapnia lowers arterial pH (respiratory acidosis).
- Severe acidemia (pH < 7.2) contributes to pulmonary arteriolar vasoconstriction, systemic vascular dilation, reduced myocardial contractility, hyperkalemia, hypotension, and cardiac irritability, with the potential for life-threatening arrhythmias.
- Acute hypercapnia also causes cerebral vasodilation and increased intracranial pressure, a major problem in patients with acute head injury.
- Over time, tissue buffering and renal compensation can largely correct the acidemia.
- However, sudden increases in $PaCO_2$ (partial pressure of carbon dioxide) can occur faster than compensatory changes ($PaCO_2$ rises 3 to 6 mm Hg/minute in a totally apneic patient).

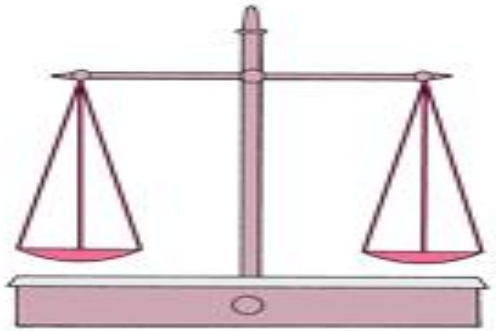
THE BALANCE BETWEEN LOAD (RESISTIVE, ELASTIC, AND MINUTE VENTILATION) AND NEUROMUSCULAR COMPETENCE (DRIVE, TRANSMISSION, AND MUSCLE STRENGTH) DETERMINES THE ABILITY TO SUSTAIN ALVEOLAR VENTILATION

Chest wall elastic loads
Abdominal distention
Ascites
Obesity
Pleural effusion
Pneumothorax
Rib fracture
Tumor

Lung elastic loads
Alveolar edema
Atelectasis
Infection
Intrinsic PEEP

Minute ventilation loads
Excess calories
Hypovolemia
Pulmonary embolus
Sepsis

Resistive loads
Bronchospasm (eg, asthma, bronchiolitis, COPD)
Edema, secretions, or scarring of airway
Obstructive sleep apnea
Upper airway obstruction (eg, croup, epiglottitis)



Impaired respiratory drive
Brain stem lesion
Drug overdose
Hypothyroidism
Sleep-disordered breathing

Impaired neurotransmission
Aminoglycosides
Amyotrophic lateral sclerosis
Botulism
Spinal cord lesion
Guillain-Barré syndrome
Myasthenia gravis
Neuromuscular blockers
Phrenic nerve injury

Muscle weakness
Electrolyte abnormalities
Fatigue
Hypoperfusion states
Hypoxemia
Myopathy
Undernutrition

SYMPTOMS AND SIGNS OF VENTILATORY FAILURE

- Clinical features of hypercapnia (ventilatory failure):
- Hypoventialtion ,daytime sleeping
- headache, anxiety,
- Warm extremities
- - vigorous use of accessory ventilatory muscles
  - tachypnea,
  - tachycardia,
  - diaphoresis,
  - irregular or gasping breathing patterns, and
  - paradoxical abdominal motion. (paralytic ileus)
- Ventilatory failure results in hypercapnia, causing central nervous system manifestations :
- ranging from subtle personality changes to marked confusion, obtundation, or coma.
- Chronic hypercapnia is better tolerated than acute and has fewer symptoms.

CLINICAL FEATURES

- Clinical features of the underlying condition

✓ Fever, e.g., due to sepsis, pneumonia

✓ Cough, e.g., due to pneumonia, COPD

✓ Chest pain, e.g., due to pneumonia, pulmonary embolism

✓ Signs of general muscle weakness, e.g., due to myasthenia gravis, Guillain-Barre syndrome, myopathies, ALS

✓ Pain on inspiration, e.g., due to rib fracture

✓ CNS depression, e.g., due to opioid use

PHYSICAL FINDINGS

- Hypotension usually with signs of poor perfusion suggest severe sepsis or pulmonary embolus
- Hypertension usually with signs of poor perfusion suggests cardiogenic pulmonary edema
- Wheeze & stridor suggest airway obstruction
- Tachycardia and arrhythmias may be the cause of cardiogenic pulmonary edema
- Elevated jugular venous pressure suggests right ventricular dysfunction
- Respiratory rate < 12b/m in spontaneously breathing patient with hypoxia or hypercarbia and acidemia suggest nervous system dysfunction
- Paradoxical respiratory motion suggest muscular dysfunction

HYPOXEMIC VS HYPERCAPNIC (VENTELATORY) RESPIRATORY FAILURE

Hypoxemic Respiratory Failure	Hypercapnic Respiratory Failure
Known as: Type I ARF, Lung Failure, Oxygenation Failure, Respiratory Insufficiency	***Known as:*** Type II ARF, Pump Failure, Ventilatory Failure
Definition: The failure of lungs and heart to provide adequate O_2 to meet metabolic needs	***Definition:*** The failure of the lungs to eliminate adequate CO_2
Criteria: $PaO_2 < 60$ mmHg on $F_IO_2 \geq .50$ or $PaO_2 < 40$ mmHg on any F_IO_2 $SaO_2 < 90$	***Criteria:*** Acute ↑ in $PaCO_2 > 50$ mmHg or Acutely above normal baseline in COPD with concurrent ↓ in pH < 7.30
Basic Causes: R-L shunt V/Q mismatch Alveolar hypoventilation Diffusion defect Inadequate FIO_2	***Basic Causes:*** Pump failure (drive, muscles, WOB) ↑ CO_2 production R-L shunt ↑ Deadspace

OTHER TYPES OF RESPIRATORY FAILURE

- **Perioperative respiratory failure** is usually caused by atelectasis (collapse of lung tissue with loss of volume). Effective means of preventing or treating atelectasis include:
  - ✓ Incentive spirometry
  - ✓ Ensuring adequate analgesia for chest and abdominal incisions
  - ✓ Upright positioning
  - ✓ Early mobilization
  - ✓ Atelectasis caused by abdominal distention should be alleviated according to the cause (eg, nasogastric suction for excessive intraluminal air, paracentesis to evacuate tense ascites).
- **Hypoperfusion,** regardless of cause, may result in respiratory failure through **inadequate delivery of oxygen to respiratory muscles** coupled with excess respiratory muscle load (eg, acidosis, sepsis).
- **Mechanical ventilation** is useful for diverting blood flow from overworked respiratory muscles to critical organs such as the brain, kidney, and gut.

DIAGNOSIS OF AHRF

- ❑ arterial blood gas (ABG) measurement
  The gold standard for the diagnosis of acute hypoxemic respiratory failure is **an arterial pO2 on room air less than 60 mmHg measured by arterial blood gases** (ABG).
- ❑ Chest x-ray

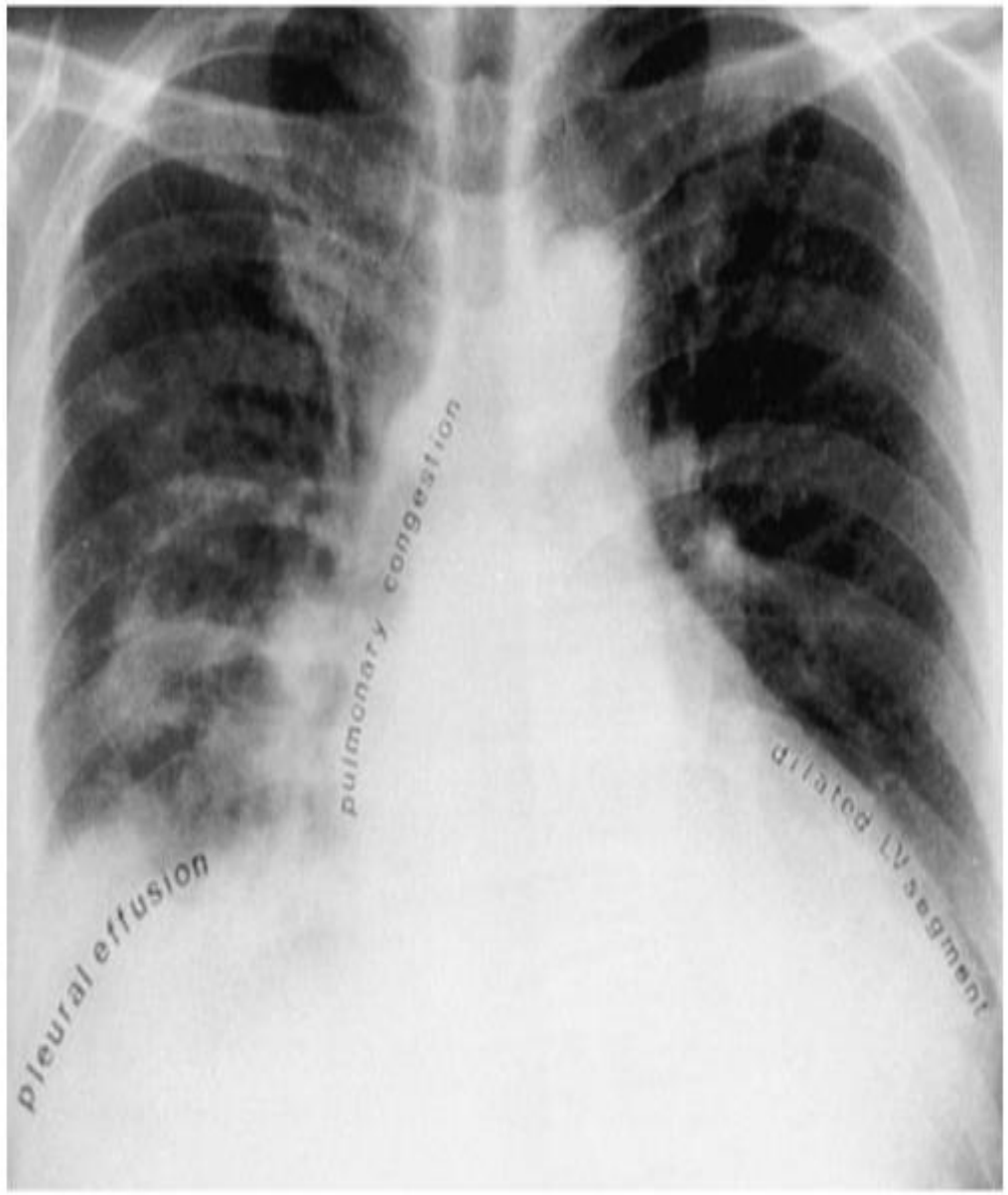
- Hypoxemia is usually first recognized using pulse oximetry. Patients with low oxygen saturation should have a chest x-ray and ABGs and be treated with supplemental oxygen while awaiting test results.
- If supplemental oxygen does not improve the oxygen saturation to > 90%, right-to-left shunting of blood should be suspected. An obvious alveolar infiltrate on chest x-ray implicates alveolar flooding as the cause, rather than an intracardiac shunt. However, at the onset of illness, hypoxemia can occur before changes are seen on x-ray.

DIAGNOSIS OF AHRF

- Once AHRF is diagnosed, the cause must be determined, considering both pulmonary and extrapulmonary causes.
- Sometimes a known ongoing disorder (eg, acute myocardial infarction, pancreatitis, sepsis) is an obvious cause.
- In other cases, **history** is suggestive; pneumonia is suspected in an immunocompromised patient, and alveolar hemorrhage is suspected after bone marrow transplantation or in a patient with a connective tissue disease.
- Frequently, however, critically ill patients have received a large volume of IV fluids for resuscitation, and high-pressure AHRF (eg, caused by ventricular failure or fluid overload) resulting from treatment must be distinguished from an underlying low-pressure AHRF (eg, caused by sepsis or pneumonia).

DIAGNOSIS OF AHRF

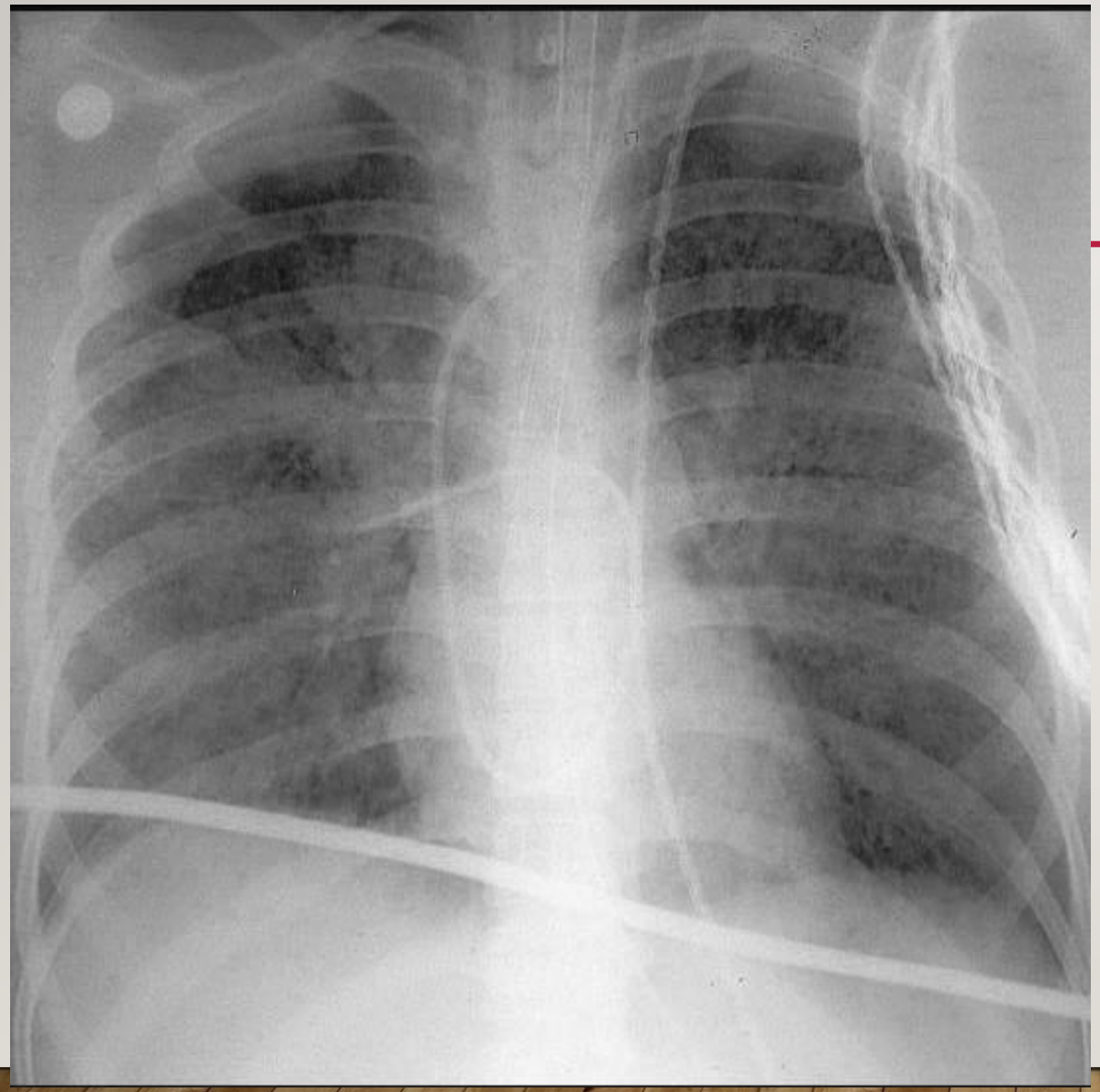
- High-pressure pulmonary edema due to left ventricular failure is suggested by:
  - a 3rd heart sound
  - jugular venous distention
  - peripheral edema on examination
- -the presence of diffuse central infiltrates
  - cardiomegaly
  - an abnormally wide vascular pedicle on chest x-ray.
- The diffuse, bilateral infiltrates of ARDS are generally more peripheral. Focal infiltrates are typically caused by lobar pneumonia, atelectasis, or lung contusion.
- Although echocardiography may show left ventricular dysfunction, implying a cardiac origin, this finding is not specific because sepsis can also reduce myocardial contractility.



Pulmonary Edema

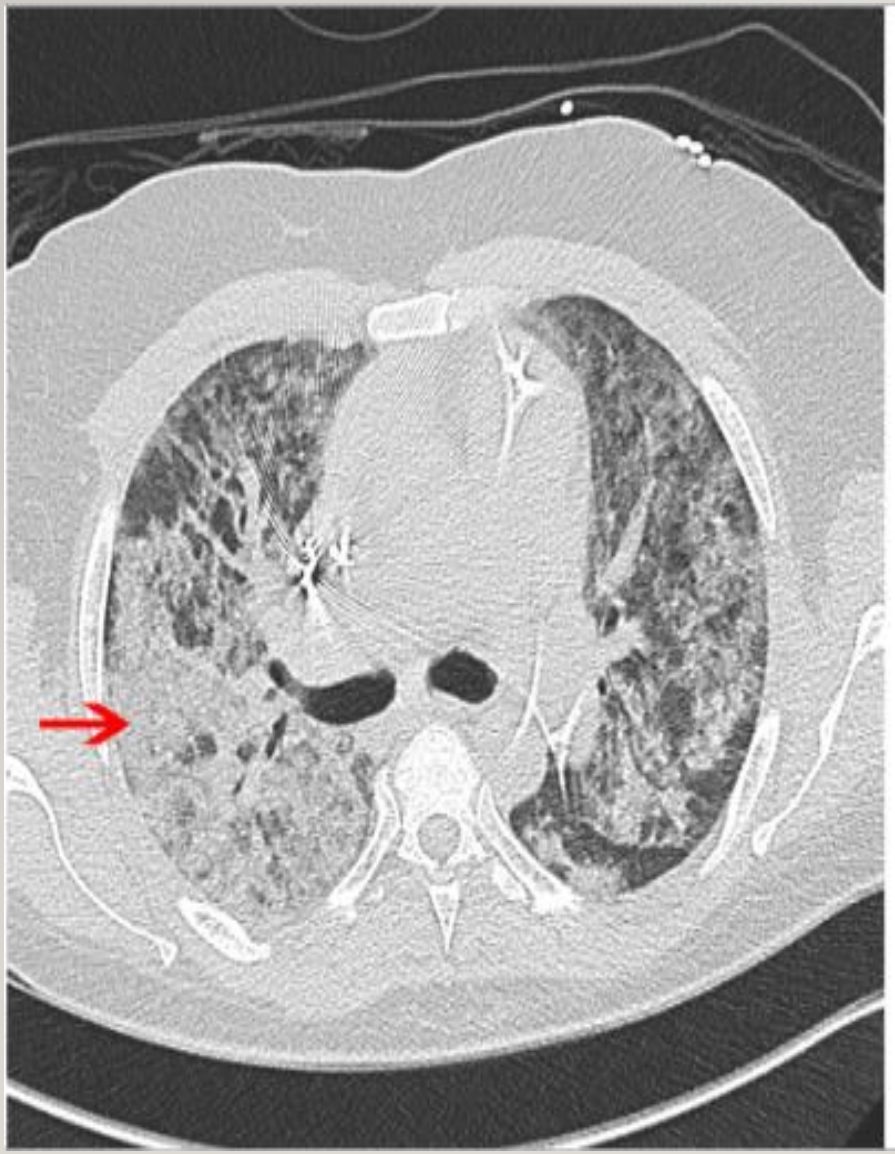
This upright chest x-ray demonstrates several of the characteristic features of acute pulmonary edema due to left ventricular failure, including cardiac dilatation, pulmonary congestion with interstitial edema, and vascular redistribution to the upper lobe vessels, as well as pleural effusions. LV = left ventricle.

Courtesy of Wada T: Basic and Advanced Visual Cardiology. Illustrated Case Report Multi-Media Approach. Philadelphia, Lea & Febiger, 1991; with permission.) By permission of the publisher. From O'Connor C, Tuman K. In Atlas of Anesthesia: Cardiothoracic Anesthesia. Edited by R Miller (series editor) and JG Reeves. Philadelphia, Current Medicine, 1999.



ACUTE RESPIRATORY DISTRESS SYNDROME

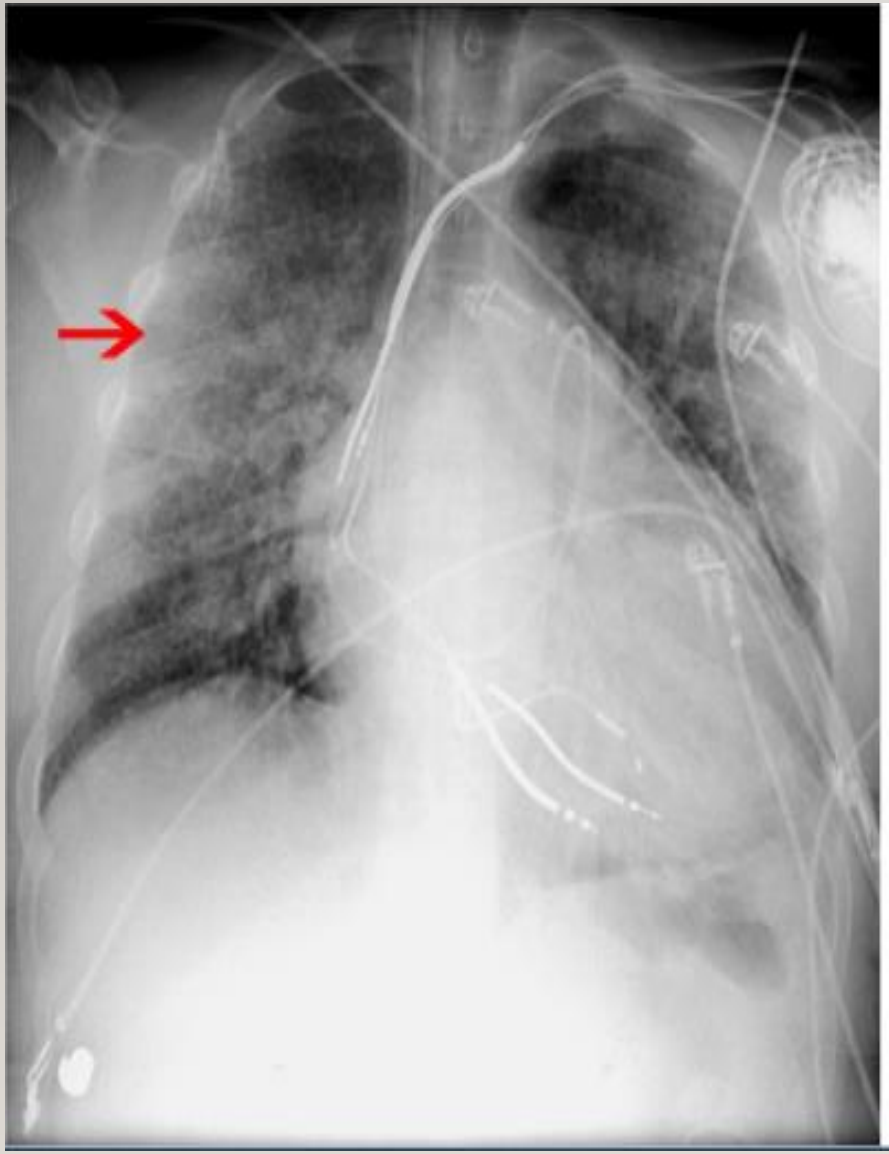
THIS UPRIGHT CHEST X-RAY SHOWS DIFFUSE BILATERAL OPACITIES CHARACTERISTIC OF ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS).



CT of a Patient with ARDS

2/3

The red arrow points to the diffuse alveolar opacities in a patient with ARDS (acute respiratory distress syndrome). The patient also has cardiomegaly, a triple lead automated implantable cardioverter defibrillator with tips in the right ventricle, and a Swan Ganz catheter with tip in the pulmonary artery.



X-ray of a Patient with ARDS

3/3

The arrow points to some of the diffuse alveolar opacities in a patient with ARDS (acute respiratory distress syndrome).

DIAGNOSTICS

- Arterial blood gas analysis(ABG): to confirm diagnosis

- Alveolar-arterial gradient:

✓ Normal Aa gradient:

- Hypoventilation or ↓ oxygen uptake

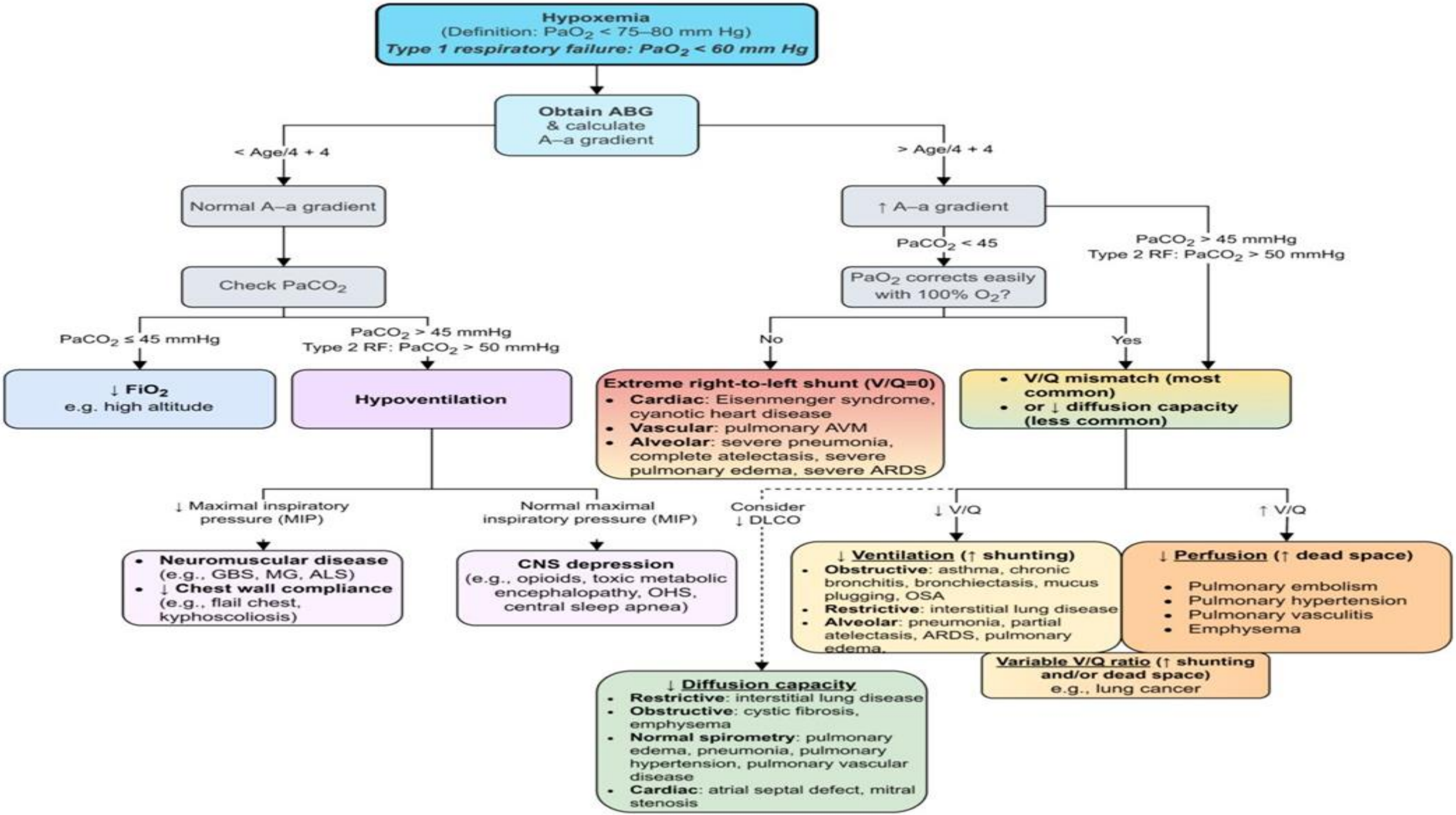
✓ ↑ Aa gradient:

- V/Q mismatch or shunting

CAUSES OF HYPOXEMIA

Causes of hypoxemia			
	Example	**A-a gradient**	**Corrects with supplemental O_2?**
Reduced PiO_2	High altitude	Normal	Yes
Hypoventilation	CNS depression, neuromuscular weakness	Normal	Yes
V/Q mismatch	Pulmonary embolism, COPD	Increased	Yes
Diffusion limitation	Emphysema, ILD	Increased	Yes
Intrapulmonary shunt (V/Q = 0)	Pneumonia, pulmonary edema, atelectasis	Increased	No
Intracardiac shunt (right to left)	Tetralogy of Fallot, Eisenmenger syndrome	Increased	No

A-a gradient = alveolar-to-arterial oxygen gradient; **ILD** = interstitial lung disease; **PiO_2** = partial pressure of inspired oxygen; **V/Q** = ventilation/perfusion ratio.


Hypoxemia
(Definition: PaO_2 < 75–80 mm Hg)
Type 1 respiratory failure: PaO_2 < 60 mm Hg
Obtain ABG
& calculate
A–a gradient
< Age/4 + 4
> Age/4 + 4
Normal A–a gradient
↑ A–a gradient
Check $PaCO_2$
$PaCO_2$ < 45
PaO_2 corrects easily with 100% O_2?
$PaCO_2$ > 45 mmHg
Type 2 RF: $PaCO_2$ > 50 mmHg
$PaCO_2$ ≤ 45 mmHg
$PaCO_2$ > 45 mmHg
Type 2 RF: $PaCO_2$ > 50 mmHg
No
Yes
↓ FiO_2
e.g. high altitude
Hypoventilation
Extreme right-to-left shunt (V/Q=0)
Cardiac: Eisenmenger syndrome, cyanotic heart disease
Vascular: pulmonary AVM
Alveolar: severe pneumonia, complete atelectasis, severe pulmonary edema, severe ARDS
V/Q mismatch (most common)
or ↓ diffusion capacity (less common)
↓ Maximal inspiratory pressure (MIP)
Normal maximal inspiratory pressure (MIP)
Consider ↓ DLCO
↓ V/Q
↑ V/Q
Neuromuscular disease (e.g., GBS, MG, ALS)
↓ Chest wall compliance (e.g., flail chest, kyphoscoliosis)
CNS depression
(e.g., opioids, toxic metabolic encephalopathy, OHS, central sleep apnea)
↓ Ventilation (↑ shunting)
Obstructive: asthma, chronic bronchitis, bronchiectasis, mucus plugging, OSA
Restrictive: interstitial lung disease
Alveolar: pneumonia, partial atelectasis, ARDS, pulmonary edema
↓ Perfusion (↑ dead space)
Pulmonary embolism
Pulmonary hypertension
Pulmonary vasculitis
Emphysema
Variable V/Q ratio (↑ shunting and/or dead space)
e.g., lung cancer
↓ Diffusion capacity
Restrictive: interstitial lung disease
Obstructive: cystic fibrosis, emphysema
Normal spirometry: pulmonary edema, pneumonia, pulmonary hypertension, pulmonary vascular disease
Cardiac: atrial septal defect, mitral stenosis

DIAGNOSTICS

- Assessment for underlying conditions

✓ **CBC** : Leukocytosis(and/or thrombocytopenia) in sepsis or pneumonia

✓ **pulmonary function tests** : COPD or asthma

✓ **ECG, echocardiography :** Heart defects (e.g., atrial septal defect, VSD, PDA)

DIAGNOSIS OF VENTILATORY FAILURE

- **Arterial blood gas (ABGs) measurement**
- **Chest x-ray**
- **Tests to determine etiology**

- Ventilatory failure should be suspected in patients with **respiratory distress**, visible ventilatory fatigue or cyanosis, or changes in sensorium and in those with disorders causing neuromuscular weakness. Tachypnea is also a concern; respiratory rates > 28 to 30/minute cannot be sustained for very long, particularly in older or weakened patients.

DIAGNOSIS OF VENTILATORY FAILURE

- If ventilatory failure is suspected,
  ABG analysis, continuous pulse oximetry, and a chest x-ray should be done.
- Respiratory acidosis revealed by the ABG measurement (eg, pH < 7.35 and PCO2 > 50) confirms the diagnosis.
- Patients with chronic ventilatory failure often have quite elevated PCO2 (eg, 60 to 90 mm Hg) at baseline, typically with a pH that is only slightly acidemic.
- In such patients, the degree of acidemia rather than the PCO2 must serve as the primary marker for acute hypoventilation.

DIAGNOSIS OF VENTILATORY FAILURE

- Because ABG measurements can be normal or show insufficient respiratory compensation:
  - in patients with metabolic acidosis and
  - incipient ventilatory failure,
- certain bedside pulmonary function tests can help predict ventilatory failure, particularly in patients with **neuromuscular weakness** who may succumb to ventilatory failure without exhibiting respiratory distress.
- Vital capacity < 10 to 15 mL/kg and an inability to generate a negative inspiratory force of 15 cm H2O suggest imminent ventilatory failure

DIAGNOSIS OF VENTILATORY FAILURE

- Once ventilatory failure is diagnosed, the cause must be identified.
- Sometimes a known ongoing disorder (eg, coma, acute asthma exacerbation, COPD exacerbation, severe hypothyroidism, myasthenia gravis, botulism) is an obvious cause.
- In other cases, **history** is suggestive;
  - sudden onset of tachypnea and hypotension after surgery suggests pulmonary embolism,
  - focal neurologic findings suggest a central nervous system or neuromuscular cause.
- Neuromuscular competence may be assessed through measurement of inspiratory muscle strength (negative inspiratory force and positive expiratory force), neuromuscular transmission (nerve conduction tests and electromyography), and investigations into causes of diminished drive (toxicology screens, brain imaging, and thyroid function tests).

MANAGEMENT

1. ABCDE
2. administration of supplemental oxygen through a patent airway
3. Treatment of the underlying causes
    - Airway obstruction (COPD, asthma): bronchodilators and/or inhaled corticosteroids
    - Infection (e.g., pneumonia, sepsis, bronchiolitis): antibiotic or antiviral treatment
    - Pulmonary embolism: empiric parenteral anticoagulation
    - Narcotic or sedative overdose: antidote
    - Brain trauma/herniation: surgical decompression
    - Ischemic stroke: reperfusion therapy
    - Pneumothorax: chest tube placement

Oxygen therapy
Methods of oxygen administration

A- Non invasive mechanical ventilation

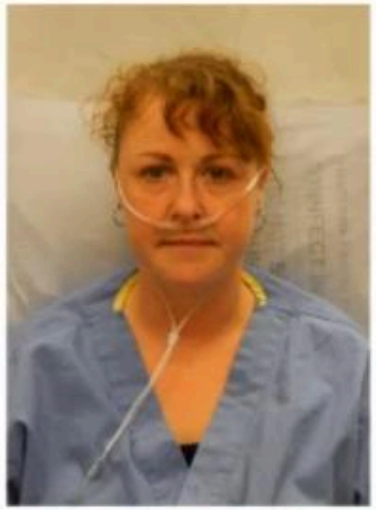
- face mask
- nasal cannula
- Mask with reservoir bag and non-rebreathing value

B- Invasive mechanical ventilation

- Endotracheal tube
- Tracheostomy

Oxygen Delivery Devices

Nasal Cannula



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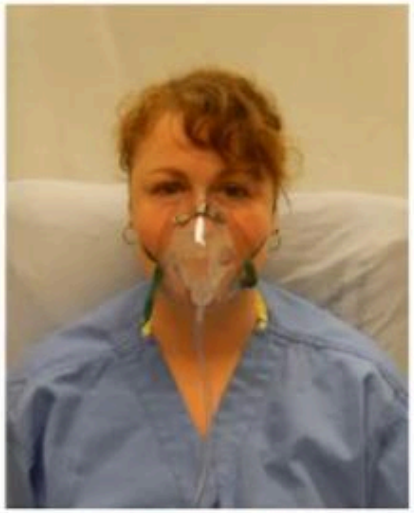
Face Mask



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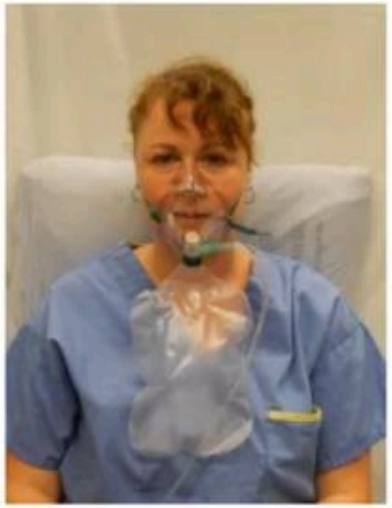
Venti Mask

LiveO2.com/Public Domain

Non-Rebreather



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High Flow Nasal Cannula

Medexsupply.com/Public Domain

Non invasive mechanical ventilation

1. negative-pressure ventilation (not commonly used like iron lung)
2. positive-pressure ventilation
    - continuous positive airway pressure (CPAP)
    - bilevel positive airway pressure (BiPAP)
    - volume-assured pressure support (VAPS)

Goal :

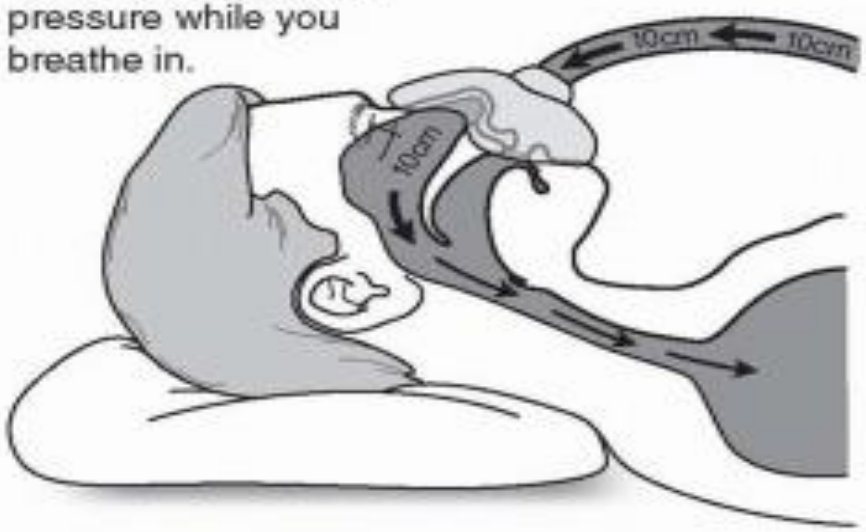
✓Correction of hypoxemia

✓Maintain adequate tissue oxygenation (PaO of 60 mmHg or SaO of > 90%)

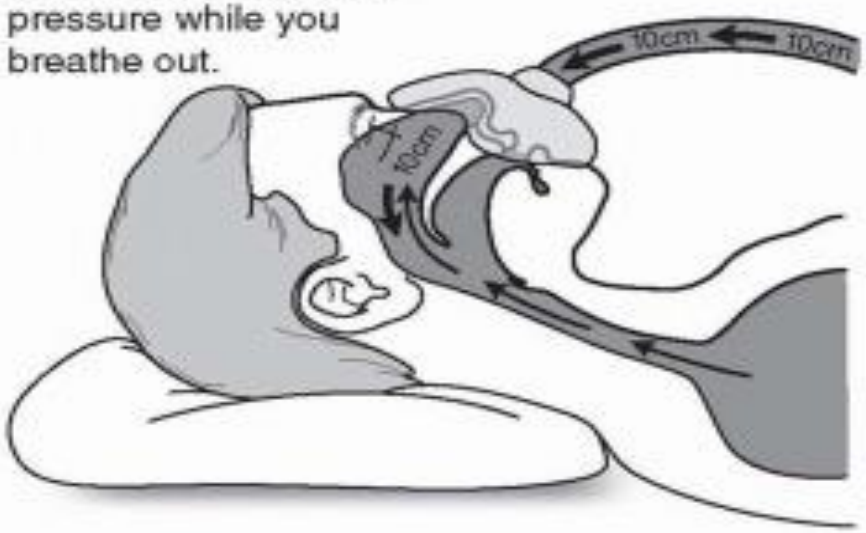

Continuous Positive Airway Pressure (CPAP)
Mask
Straps
Air hose
CPAP
machine

CPAP or APAP

Inhalation (breathing in)
CPAP blows constant pressure while you breathe in.

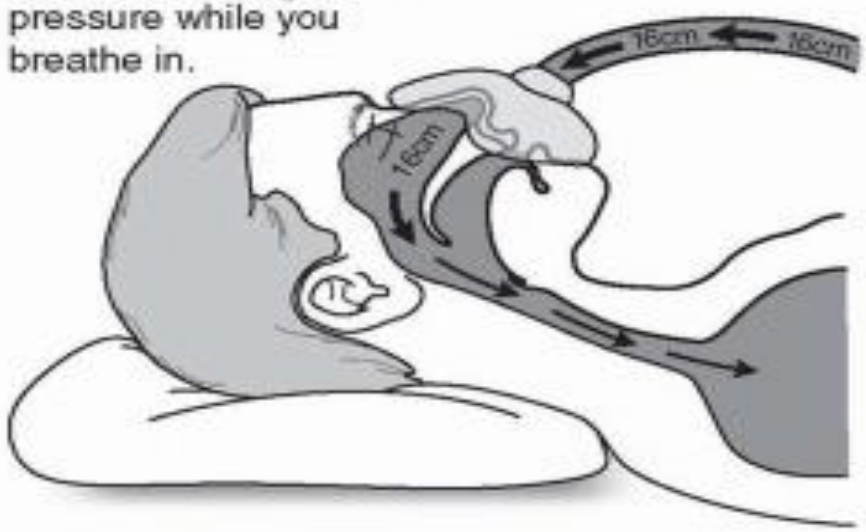


Exhalation (breathing out)
CPAP blows the same pressure while you breathe out.

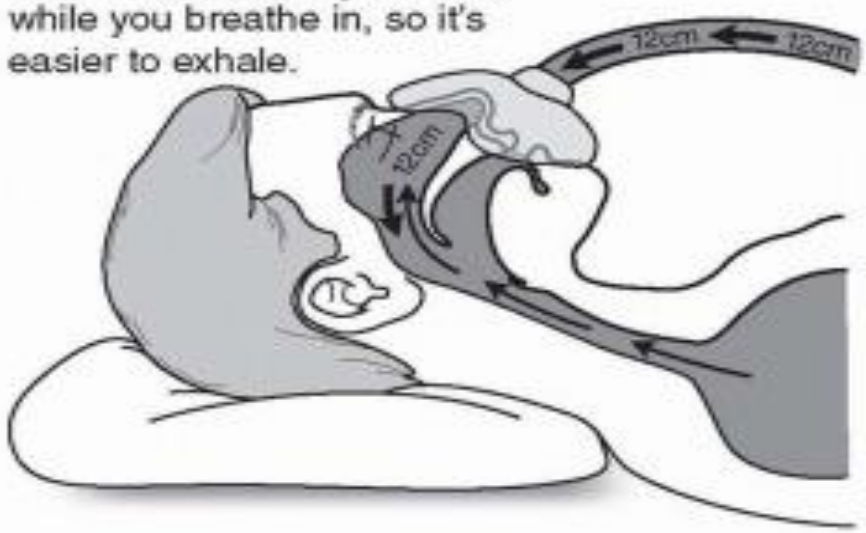


Bilevel PAP (BiPAP)

Inhalation (breathing in)
Bilevel blows higher pressure while you breathe in.



Exhalation (breathing out)
CPAP blows lower pressure while you breathe in, so it's easier to exhale.



Indication of non invasive mechanical ventilation

Box 16.5 Some indications for the use of non-invasive ventilation (NIV)

- Acute exacerbation of COPD (pH <7.35)
- Cardiogenic pulmonary oedema
- Chest wall deformity/neuromuscular disease (hypercapnic respiratory failure)
- Obstructive sleep apnoea
- Severe pneumonia (see Box 15.7)
- Asthma (occasionally)
- Weaning patients from invasive ventilation

Contraindications include facial or upper airway surgery, reduced conscious level, inability to protect the airway.

Modified from BTS guidelines after 1997, http://www.british thoracic society.co.uk.

Contraindications of non invasive mechanical ventilation

Box 3: Contraindications for the use of noninvasive positive-pressure ventilation

Absolute

- Substantially impaired level of consciousness
- Severe agitation
- Copious secretions
- Uncontrolled vomiting
- Inability to protect airway
- Repeated hemoptysis or hematemesis
- Recent esophagectomy
- Acute myocardial infarct
- Cardiac arrest
- Immediate endotracheal intubation necessary
- Apnea
- Upper airway obstruction
- Facial trauma
- Patient declines

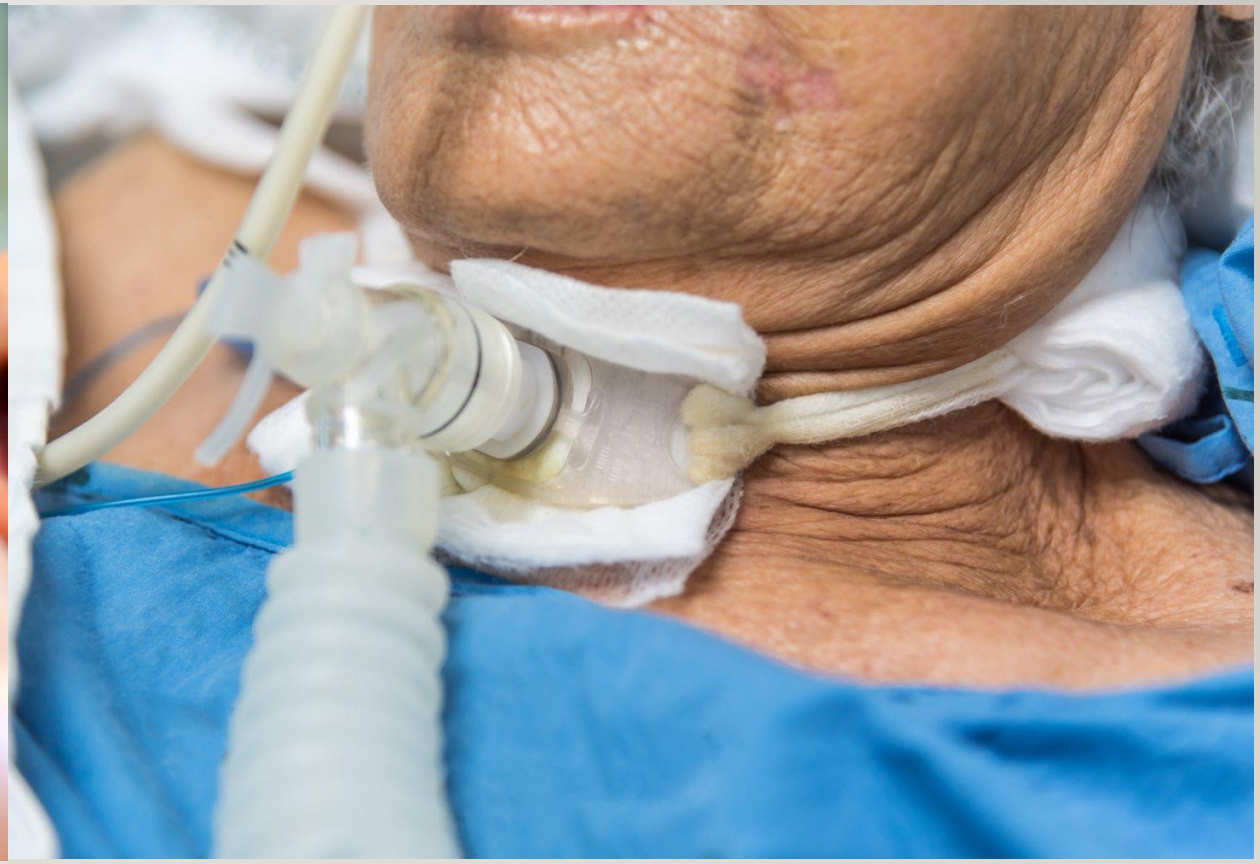
Relative

- Mildly decreased level of consciousness
- Progressive severe respiratory failure
- Uncooperative patient who can be calmed or comforted
- Suspected acute coronary ischemia
- Hemodynamic instability
- Pregnancy

Complication of noninvasive mechanical ventilation

- Air leaks
- Dry mucous membranes and thick secretion
- Mask discomfort
- Gastric distention
- Failure to ventilate

Invasive mechanical ventilation



Invasive mechanical ventilation

- Continuous ventilation
- Intermittent ventilation
- Pressure support ventilation
- Pressure control ventilation

Goal:

✓Correct hypercapnia and possible hypoxemia

✓Support weak/fatigued respiratory muscles

Indication of invasive mechanical ventilation

- Unable to tolerate NIV or NIV failure
- Respiratory or cardiac arrest
- Respiratory pauses with loss of consciousness or gasping for air
- Diminished consciousness, psychomotor agitation inadequately controlled by sedation
- Massive aspiration
- Persistent inability to remove respiratory secretions
- Heart rate <50 per min with loss of alertness
- Severe hemodynamic instability without response to fluids and vasoactive drugs
- Severe ventricular arrhythmias
- Life-threatening hypoxemia in patients unable to tolerate NIV

Contraindications of invasive mechanical ventilation

- Inability of patient to extend head
- Moderate to severe trauma to the cervical spine or anterior neck
- Infection in the epiglottal area or at skin site for tracheostomy
- Mandibular fracture or trismus
- Mild hypoxia
- Uncontrolled oropharyngeal hemorrhage
- Basilar skull fracture (during nasal intubation)
- Laryngeal cancer

Complication of endotracheal intubation

Immediate
Trauma to the upper airway
Tube in oesophagus
Tube in one or other (usually the right) main bronchus
Early
Migration of the tube out of the trachea
Leaks around the tube
Obstruction of tube because of kinking or secretions
Late
Sinusitis
Mucosal oedema and ulceration
Laryngeal injury
Tracheal narrowing and fibrosis
Tracheomalacia

Complication of tracheostomy

Table 16.9 **Complications of tracheostomy** *(As for tracheal intubation, see Table 16.8), plus:*

Early
- Death
- Pneumothorax
- Haemorrhage
- Hypoxia
- Hypotension
- Cardiac arrhythmias
- Tube misplaced in pretracheal subcutaneous tissues
- Subcutaneous emphysema

Intermediate
- Mucosal ulceration
- Erosion of tracheal cartilages (can cause tracheo-oesophageal fistula)
- Erosion of innominate artery (occasionally leads to fatal haemorrhage)
- Stomal infection
- Pneumonia

Late
- Failure of stoma to heal
- Tracheal granuloma
- Tracheal stenosis at level of stoma, cuff or tube tip
- Collapse of tracheal rings at level of stoma
- Cosmetic

Reference

- Kumar and Clark's clinical medicine
- MSD PRO
- Medstudy
- Vestbo J, Hurd SS, Agustí AG, et al. Global strategy for the diagnosis management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 201 3;187 (4):347-365.
- Medscape

Thank You