RESPIRATORY FAILURE

HINA VAISH

RESPIRATORY FAILURE

- "inability of the lung to meet the metabolic demands of the body. This can be from failure of tissue oxygenation and/or failure of CO_2 homeostasis."

RESPIRATORY FAILURE

- Definition
  Respiration is gas exchange between the organism and its environment. Function of respiratory system is to transfer O_2 from atmosphere to blood and remove CO_2 from blood.

- Clinically
  Respiratory failure is defined as PaO_2 <60 mmHg while breathing air, or a $PaCO_2$ >49 mmHg.

HYPOXEMIC RESPIRATORY FAILURE(TYPE 1)

- PaO_2 <60mmHg with normal or low $PaCO_2$ $\rightarrow$ normal or high pH
- common form of respiratory failure
- Lung disease is severe to interfere with pulmonary O_2 exchange, but over all ventilation is maintained
- Physiologic causes: V/Q mismatch and shunt

Causes of Hypoxemic Respiratory failure

- Caused by a disorder of heart, lung or blood.

- Etiology easier to assess by CXR abnormality:

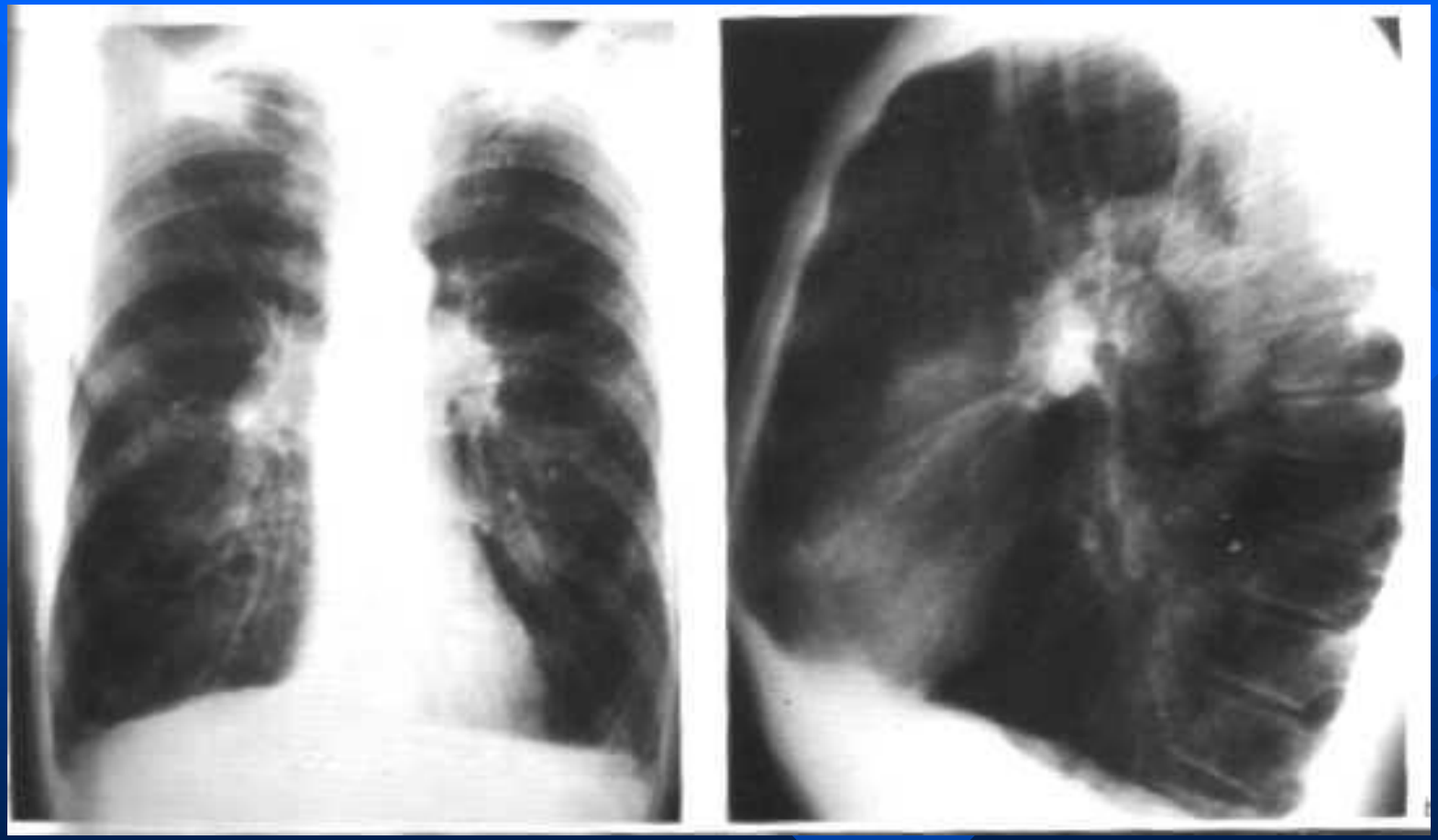
  - Normal Chest x-ray

    Cardiac shunt (right to left)

    Asthma, COPD

    Pulmonary embolism

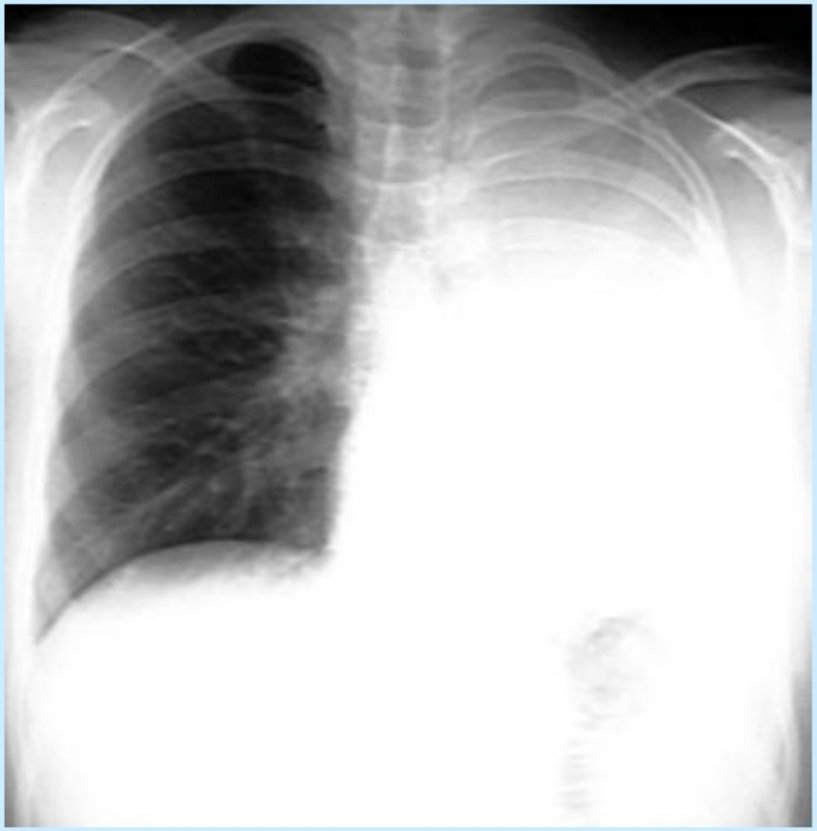
Hyperinflated Lungs : COPD



Causes of Hypoxemic Respiratory failure (cont'd.)

- Focal infiltrates on CXR
  - Atelectasis
  - Pneumonia

An example of intrapulmonary shunt

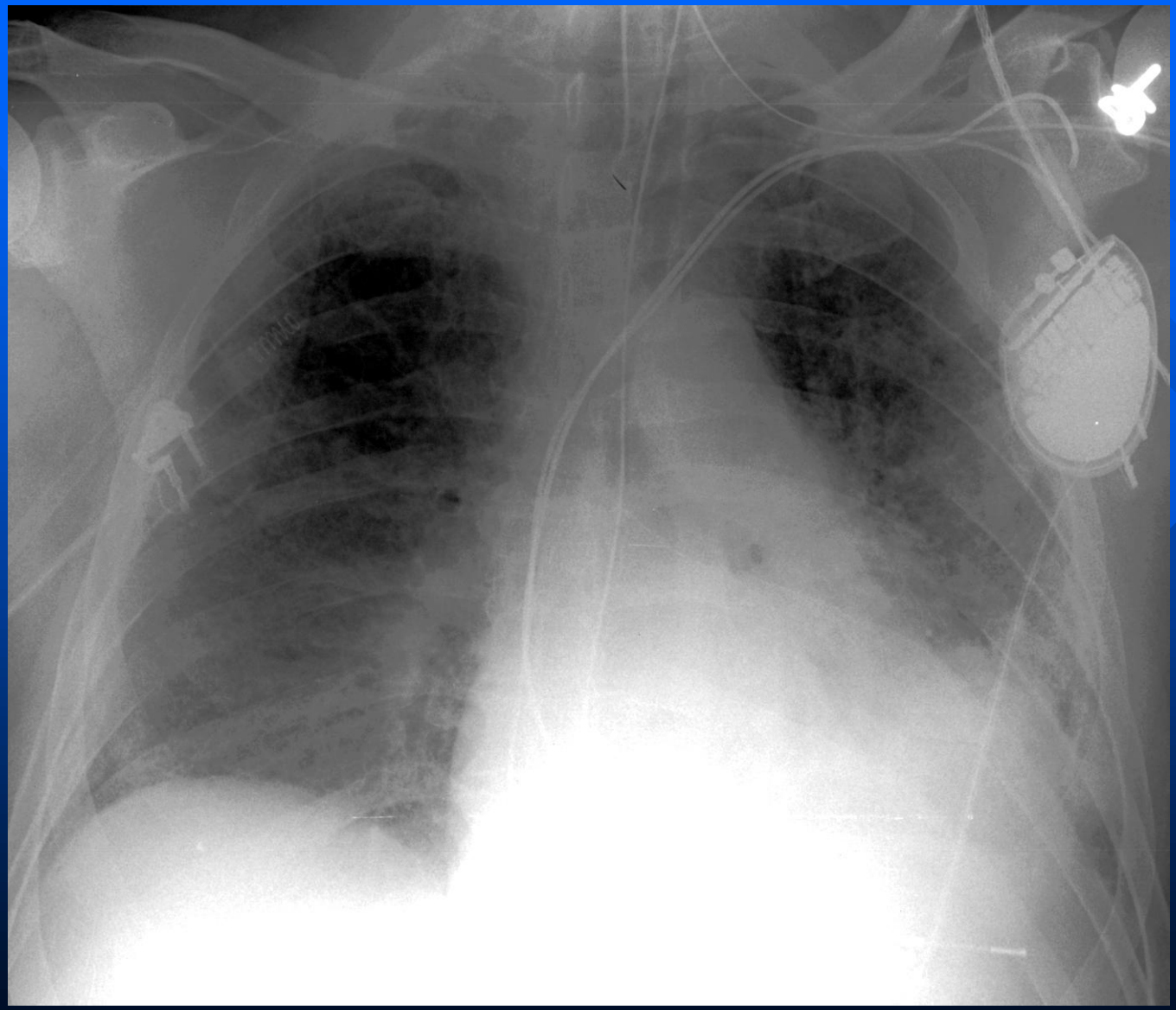


Causes of Hypoxemic Respiratory Failure (cont'd.)

Diffuse infiltrates on CXR

- Cardiogenic Pulmonary Edema
- Non cardiogenic pulmonary edema (ARDS)
- Interstitial pneumonitis or fibrosis
- Infections

Diffuse pulmonary infiltrates



Hypercapnic Respiratory Failure (Type II)

- $PaCO_2$ >49 mmHg
- Hypoxemia is always present
- pH depends on level of HCO_3
- HCO_3 depends on duration of hypercapnia
- Renal response occurs over days to weeks

Acute Hypercapnic Respiratory Failure (Type II)

- Acute
- Arterial pH is low
- Causes

  - sedative drug over dose
  - acute muscle weakness such as myasthenia gravis
  - severe lung disease:
    alveolar ventilation can not be maintained (i.e. Asthma or pneumonia)

- Acute on chronic:
- This occurs in patients with chronic CO_2 retention who worsen and have rising CO_2 and low pH.
- Mechanism: respiratory muscle fatigue

Causes of Hypercapnic Respiratory failure

- Respiratory centre (medulla) dysfunction
- Drug over dose, CVA, tumor, hypothyroidism,central hypoventilation
- Neuromuscular disease
  Guillain-Barre, Myasthenia Gravis, polio, spinal injuries
- Chest wall/Pleural diseases
  kyphoscoliosis, pneumothorax, massive pleural effusion
- Upper airways obstruction
  tumor, foreign body, laryngeal edema
- Peripheral airway disorder
  asthma, COPD

Clinical and Laboratory Manifestation

(non-specific and unreliable)

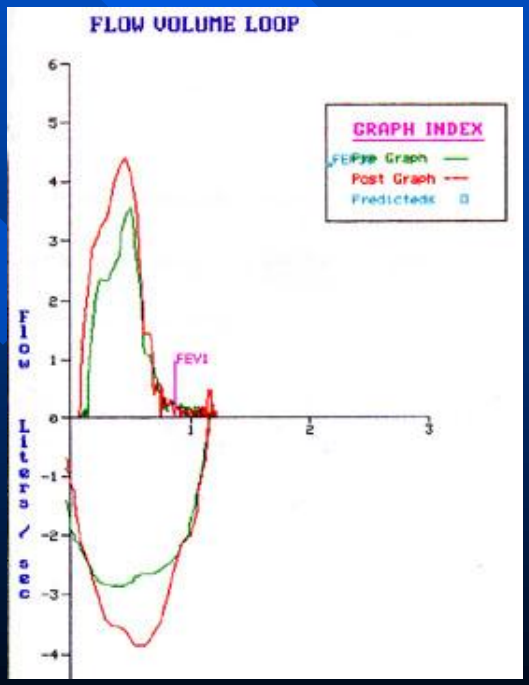
- Cyanosis
  - bluish color of mucous membranes/skin indicate hypoxemia
- - unoxygenated hemoglobin 50 mg/L
  - not a sensitive indicator
- Dyspnea
  - secondary to hypercapnia and hypoxemia
- Paradoxical breathing
- Confusion, somnolence and coma
- Convulsions

ASSESSMENT OF PATIENT

- Careful history
- Physical Examination
- ABG analysis
  -classify RF and help with cause

  Lung function

  Chest Radiograph
- EKG



Clinical & Laboratory Manifestations

- Circulatory changes
  - tachycardia, hypertension, hypotension
- Polycythemia
  - chronic hypoxemia - erythropoietin synthesis
- Pulmonary hypertension
- Cor-pulmonale or right ventricular failure

Management of Respiratory Failure Principles

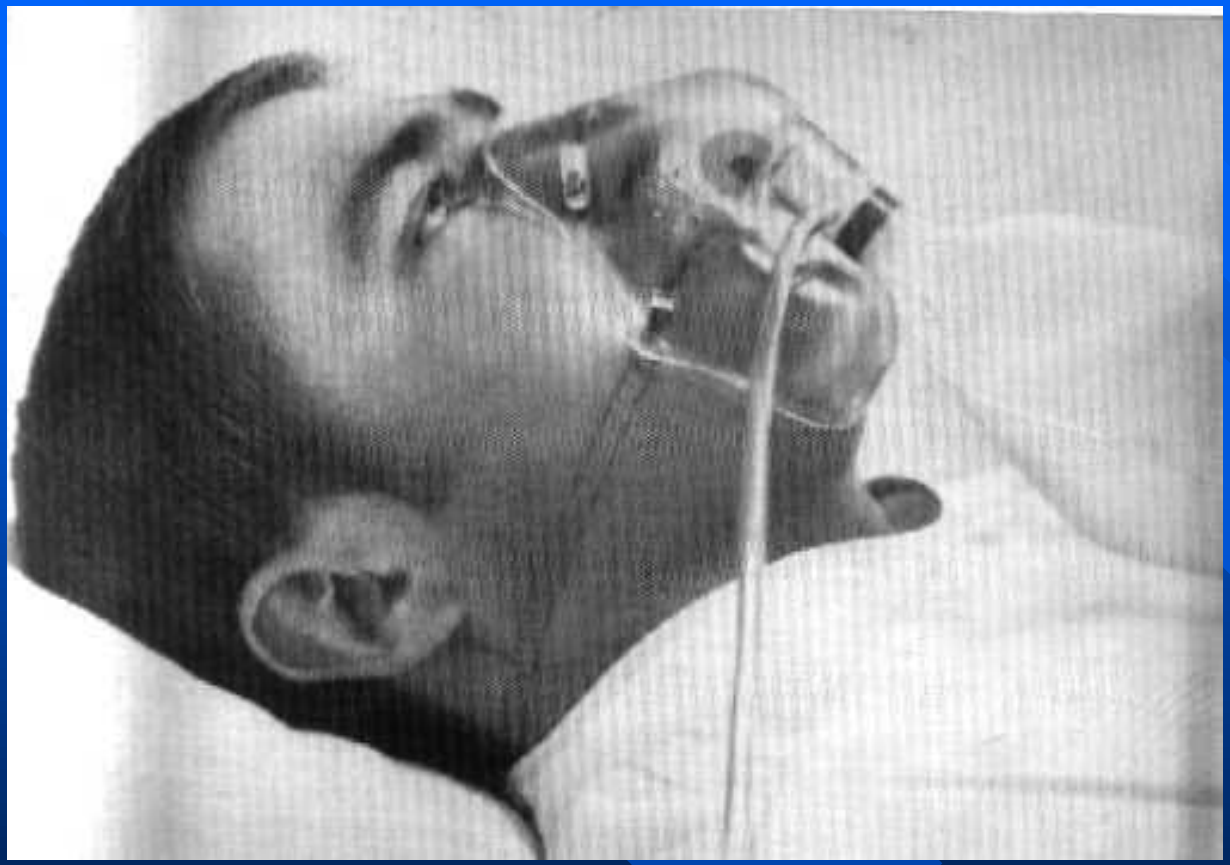
- Hypoxemia may cause death in RF
- Primary objective is to reverse and prevent hypoxemia
- Secondary objective is to control $PaCO_2$ and respiratory acidosis
- Treatment of underlying disease
- Patient's CNS and CVS must be monitored and treated

Oxygen Therapy

- Supplemental O_2 therapy essential
- titration based on SaO_2, PaO_2 levels and $PaCO_2$
- Goal is to prevent tissue hypoxia
- Tissue hypoxia occurs (normal Hb & C.O.)
  - venous $PaO_2 < 20$ mmHg or $SaO_2 < 40\%$
  - arterial $PaO_2 < 38$ mmHg or $SaO_2 < 70\%$
- Increase arterial $PaO_2 > 60$ mmHg($SaO_2 > 90\%$) or venous $SaO_2 > 60\%$
- O_2 dose either flow rate (L/min) or FiO_2 (%)

Risks of Oxygen Therapy

- **O_2 toxicity**:
  - very high levels(>1000 mmHg) CNS toxicity and seizures
  - lower levels (FiO_2 > 60%) and longer exposure: - capillary damage, leak and pulmonary fibrosis
  - PaO_2 >150 can cause retrolental fibroplasia
  - FiO_2 35 to 40% can be safely tolerated indefinitely
- **CO_2 narcosis:**
  - $PaCO_2$ may increase severely to cause respiratory acidosis, somnolence and coma
  - $PaCO_2$ increase secondary to combination of
    a) abolition of hypoxic drive to breathe
    b) increase in dead space



MECHANICAL VENTILATION

- Non invasive with a mask
- Invasive with an endobronchial tube
- MV can be volume or pressure cycled
  For hypercapnia:
  - MV increases alveolar ventilation and lowers $PaCO_2$, corrects pH
  - rests fatigues respiratory muscles
- For hypoxemia:
  - O_2 therapy alone does not correct hypoxemia caused by shunt
  - Most common cause of shunt is fluid filled or collapsed alveoli (Pulmonary edema)

POSITIVE END EXPIRATORY PRESSURE (PEEP)

- PEEP increases the end expiratory lung volume (FRC)
- PEEP recruits collapsed alveoli and prevents recollapse
- FRC increases, therefore lung becomes more compliant
- Reversal of atelectasis diminishes intrapulmonary shunt
- Excessive PEEP has adverse effects
  - decreased cardiac output
  - barotrauma (pneumothorax, pneumomediastinum)
  - increased physiologic dead space
  - increased work of breathing