Respiratory Failure

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Mechanisms

Definition

- **Definition**

  "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."

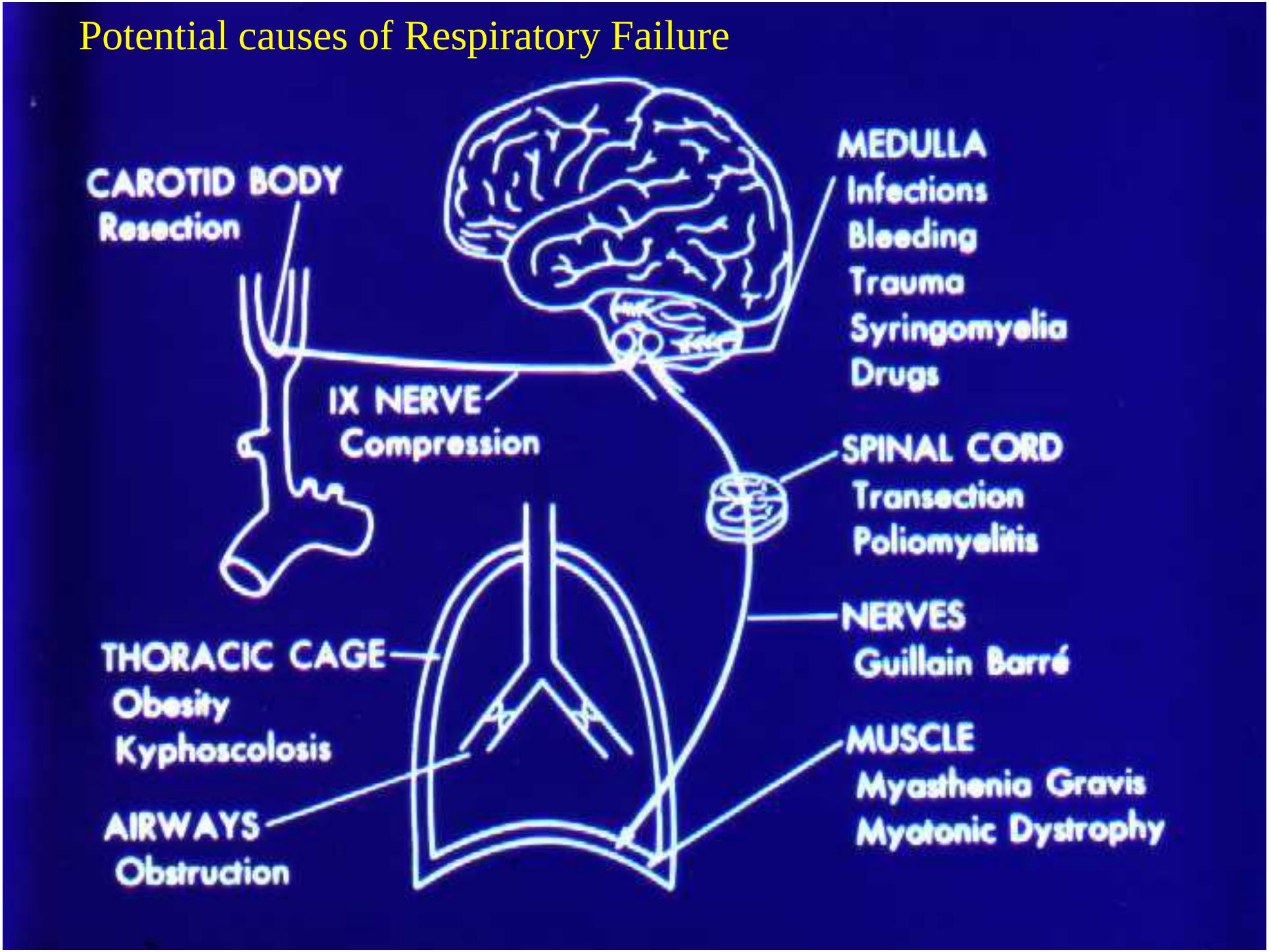
- **Clinically**

  Respiratory failure is defined as PaO_2 <60 mmHg while breathing air, or a $PaCO_2$ >50 mmHg.

Areas that may be included

CNS (medulla)
Peripheral nervous system (phrenic nerve)
Respiratory muscles - diaphragm
Chest wall - rib cage, spine
Lung - interstitium
Upper airways
Bronchial tree
Alveolar region – ducts, sacs, alveoli
Pulmonary vasculature (primarily, secondarily)

Potential causes of Respiratory Failure



HYPOXEMIC RESPIRATORY FAILURE (TYPE 1)

- PaO_2 <60mmHg with normal or low $PaCO_2$ → normal or high pH
- Most 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

HYPOXEMIC RESPIRATORY FAILURE
CAUSES OF ARTERIAL HYPOXEMIA

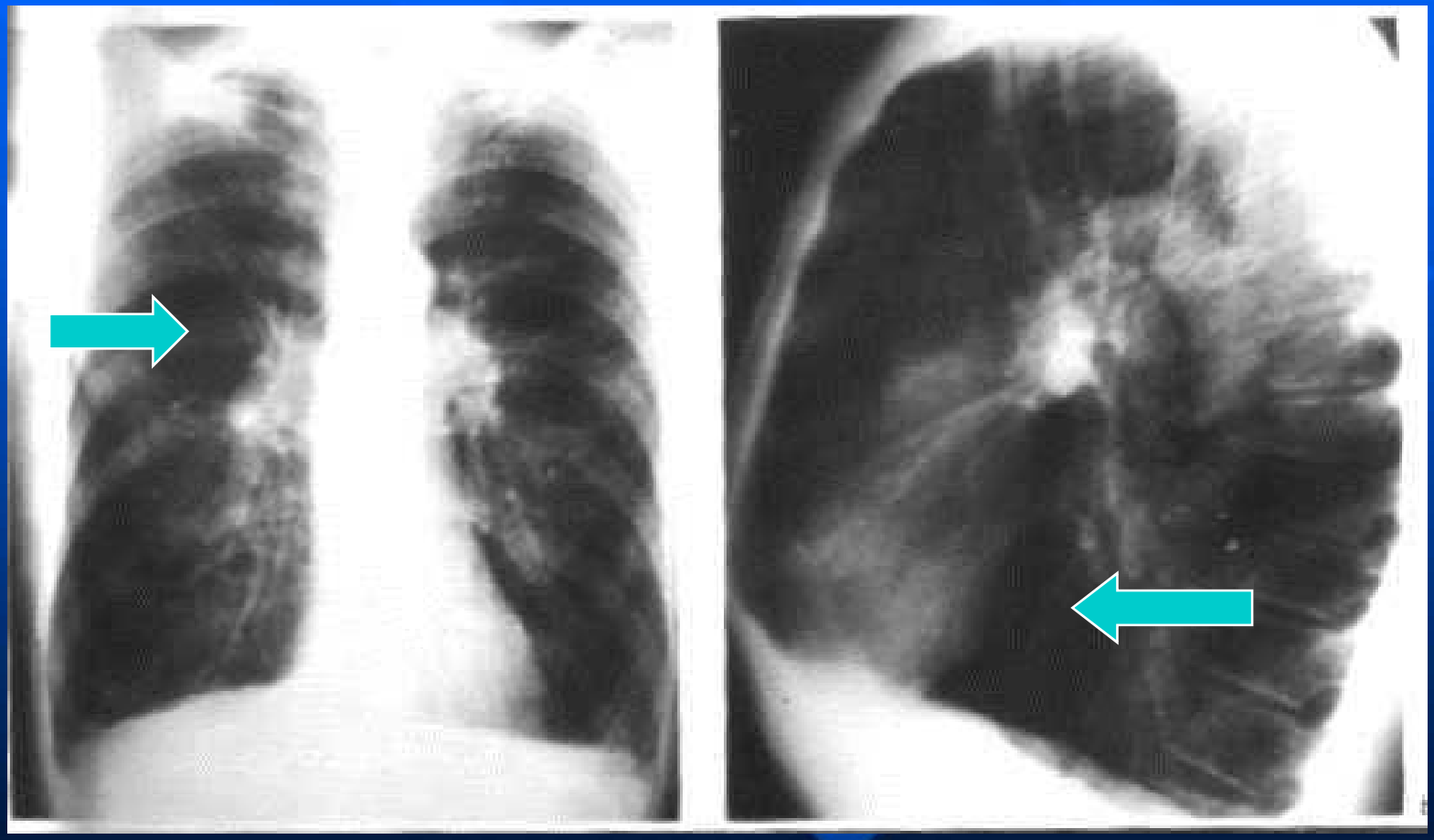
1. ↓FiO_2
2. Hypoventilation (↑ $PaCO_2$)
3. V/Q mismatch (eg.COPD)
4. Diffusion limitation ?
5. Intrapulmonary shunt
   - pneumonia
   - Atelectasis
   - CHF (high pressure pulmonary edema)
   - ARDS (low pressure pulmonary edema)

Hypercapnic Respiratory failure

Causes

- Disorder of heart, lung or blood.
- Finding chest X-ray (CRX) abnormality:
- Normal or hyperinflation on CXR:
  - Cardiac shunt (right to left)
  - Asthma, COPD
  - Pulmonary embolism
- Focal infiltrates on CXR:
  - Atelectasis
  - Pneumonia
- Diffuse infiltrates on CXR:
  - Cardiogenic pulmonary Edema
  - Non cardiogenic pulmonary edema (ARDS)
  - Interstitial pneumonitis or fibrosis
  - Infections

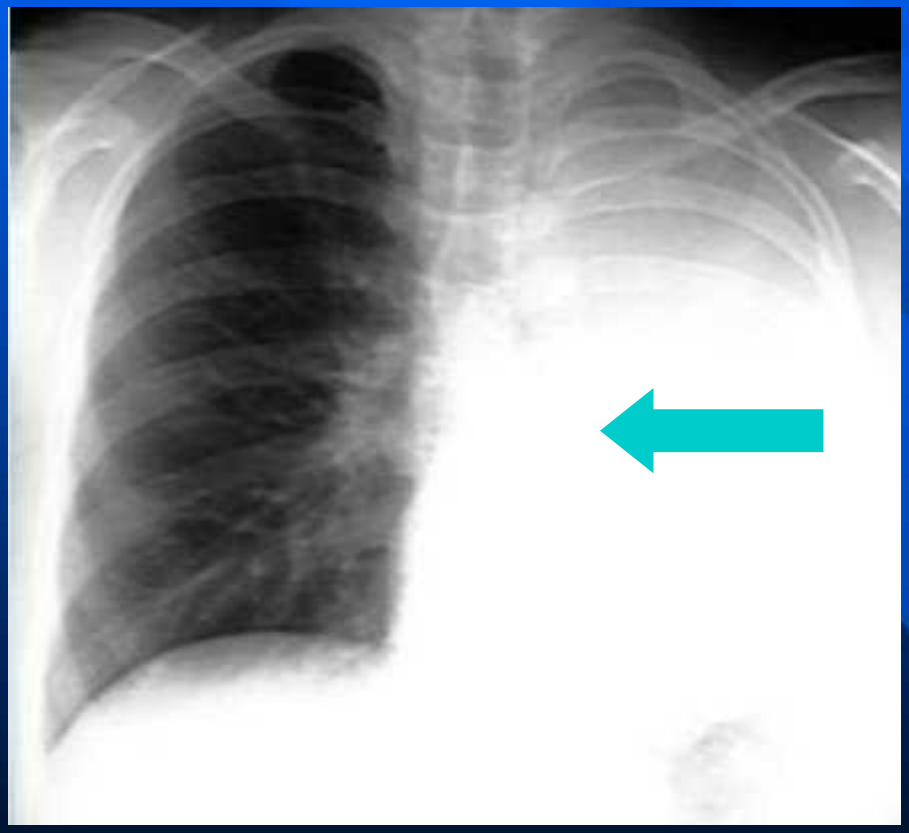
EXAMPLES



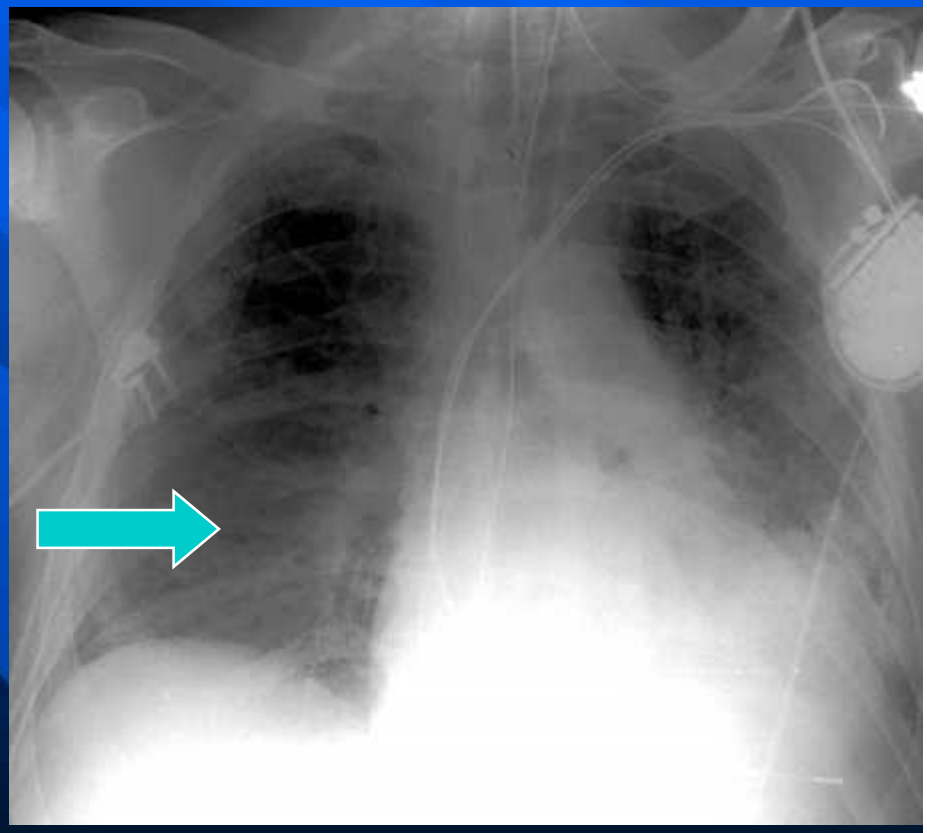
Hyperinflated Lungs COPD

EXAMPLES

Intrapulmonary shunt



Diffuse pulmonary infiltrates



Hypercapnic Respiratory Failure (Type II, global)

- Hypercapnia ($PaCO_2$ >50 mmHg)
- Hypoxemia (PaO_2 < 60 O_2 Sat < 90)
- Respiratory acidosis pH < 7.30

  Compensated by HCO_3^-:
  - » HCO_3 depends on duration of hypercapnia
  - » Renal response occurs over days to weeks

Causes

- **Acute**
  - Brain dysfunction: respiratory centre failure– sedative drug over dose, tumor, central hypoventilation
  - Hypothyroidism, Acute muscle weakness: myasthenia gravis, spinal injuries
  - Severe lung disease: asthma, pneumonia
  - Upper airways obstruction: foreign body, laryngeal edema

- **Chronic**
  - Muscle fatigue: Guillain-Barre, poliomyelitis
  - Chest wall/Pleural diseases: kyphoscoliosis, pneumothorax, massive pleural effusion
  - Airway obstruction: asthma, COPD, bronchiektasia, cystic fibrosis, tumor

Clinical Manifestations

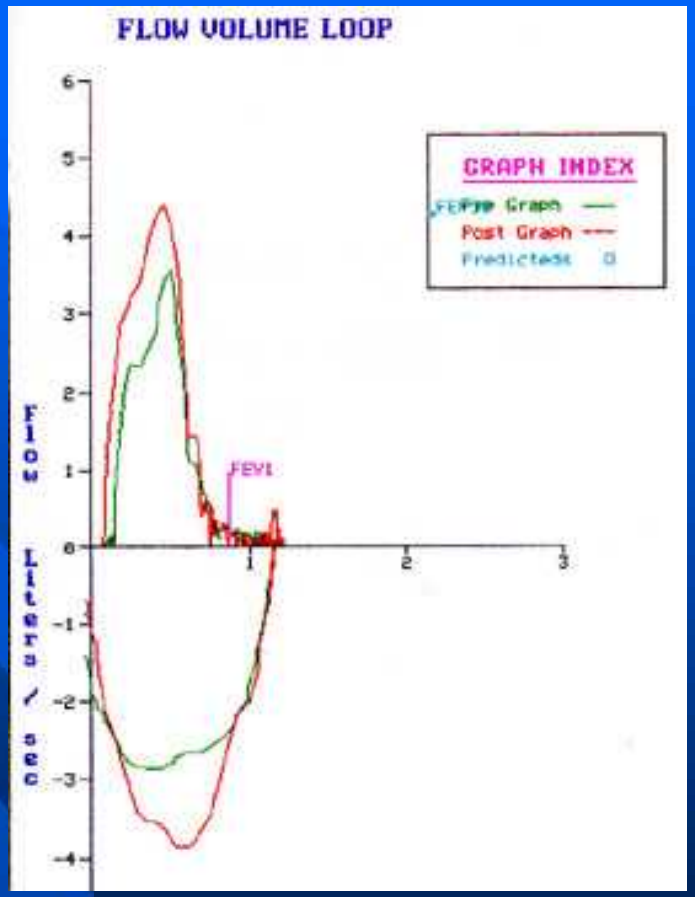
ASSESSMENT OF PATIENT

- Anamnesis - history
- Physical Examination - auscultation
- ABG analysis

  1) $PaCO_2 = \frac{VCO_2}{VA} \times 0.863$

  2) $P(A\text{-}a)0_2 = (PiO_2 - \frac{PaCO_2}{R}) - PaO_2$

- Lung function
- Chest radiography
- ECG



Clinical manifestations

- Signs of Hypoxemia
- Decreased PO2
  - Dyspnea, tachypnea
  - Cyanosis
  - Restlessness
  - Apprehension
  - Confusion
  - Tachycardia
  - Dysrhythmias
  - HTN
  - Metabolic acidosis

- Signs of Hypercapnia
- Increased PCO_2
  - Dyspnea $\rightarrow$ resp. depression
  - Headache
  - Papilledema
  - Tachycardia
  - HTN
  - Drowsiness, coma
  - Systemic vasodialation
  - Heart failure
  - Respiratory acidosis

Clinical manifestations (1.1)

- Cyanosis - unoxygenated hemoglobin 50 mg/l - not a sensitive indicator
- Dyspnea - secondary to hypercapnia and hypoxemia
- Paradoxical breathing
- Confusion, somnolence and coma
- Convulsions

Clinical manifestations (1.2)

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


Management

Goals

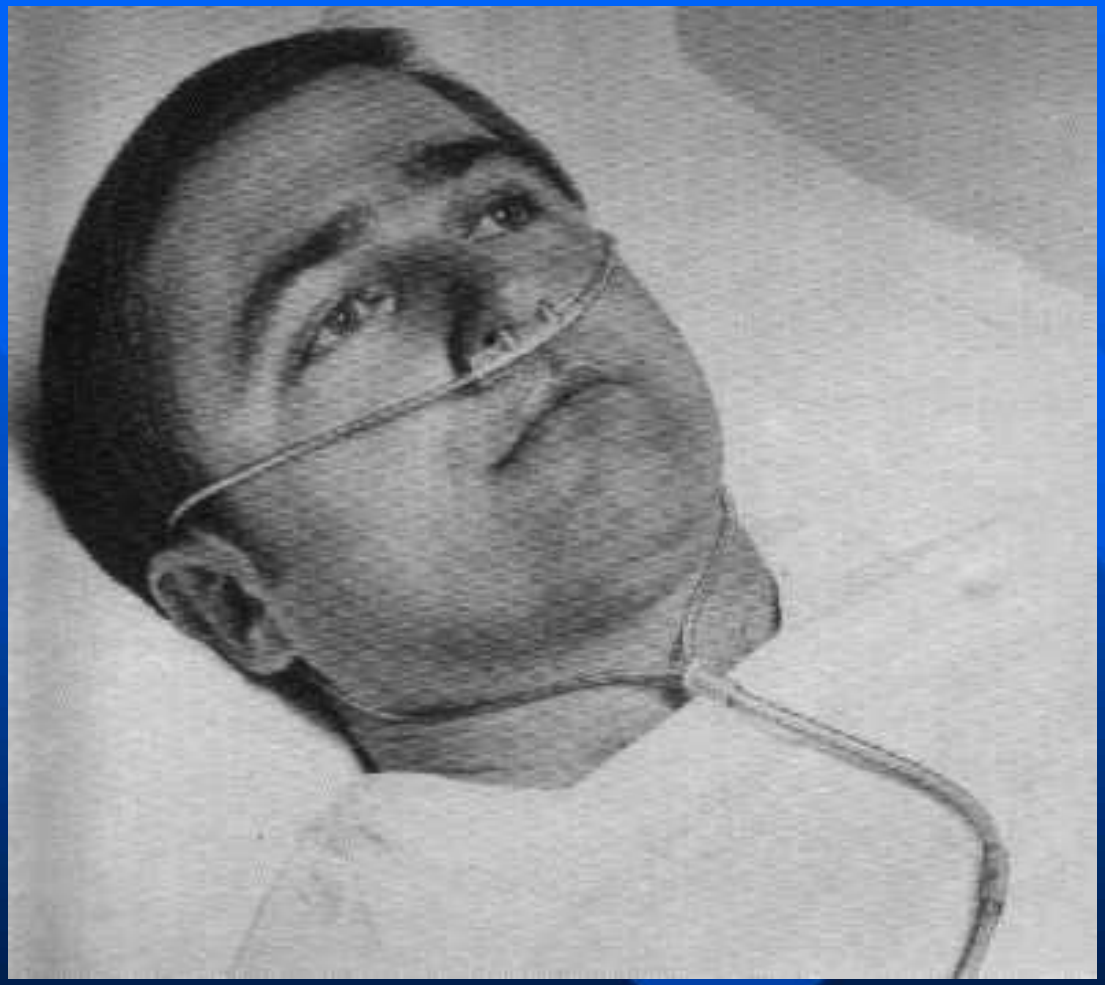
- Hypoxemia may cause death in RF
- Primary objective is to reverse and prevent hypoxemia
- Secondary objective is to control $PaCO_2$ and respiratory acidosis
- Treatment of underlying disease
- Patient's CNS and CVS must be monitored and treated

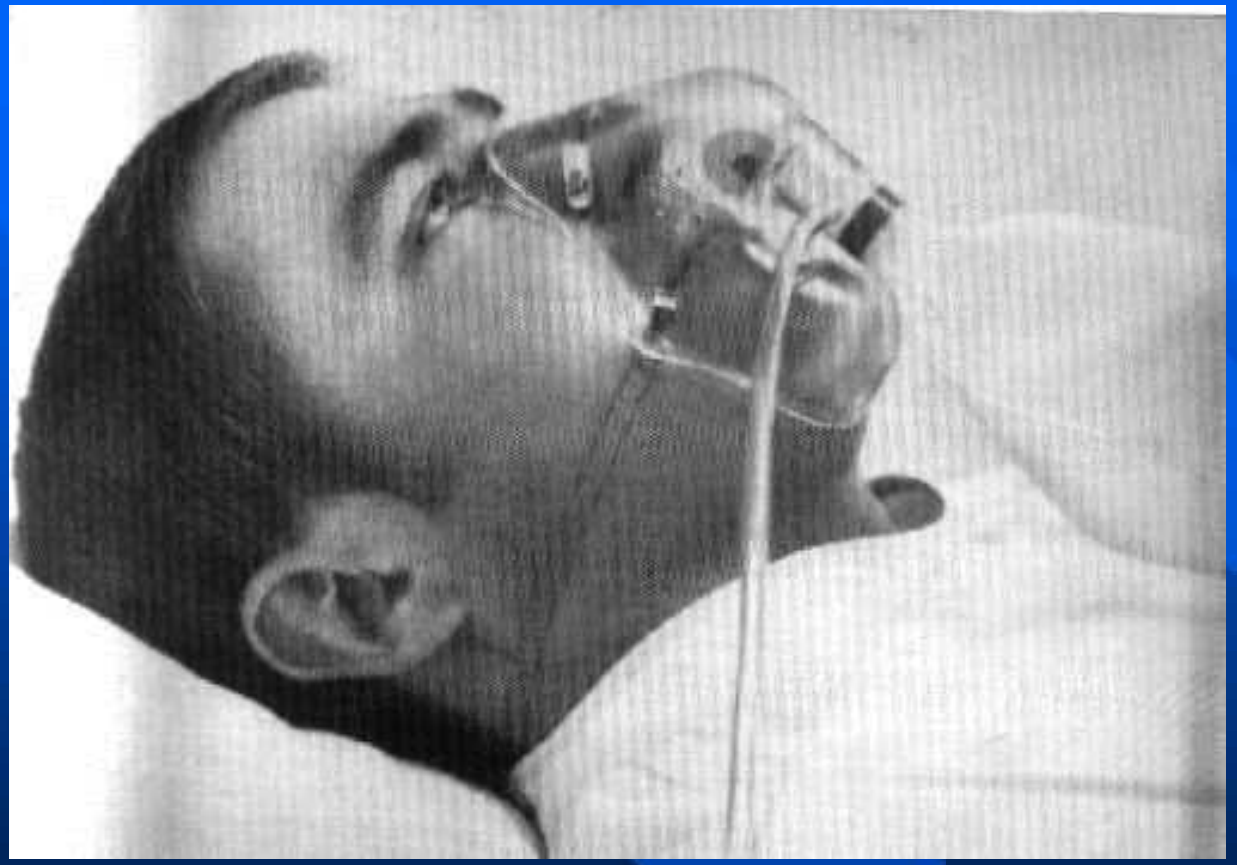
1. Oxygen Therapy

- 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

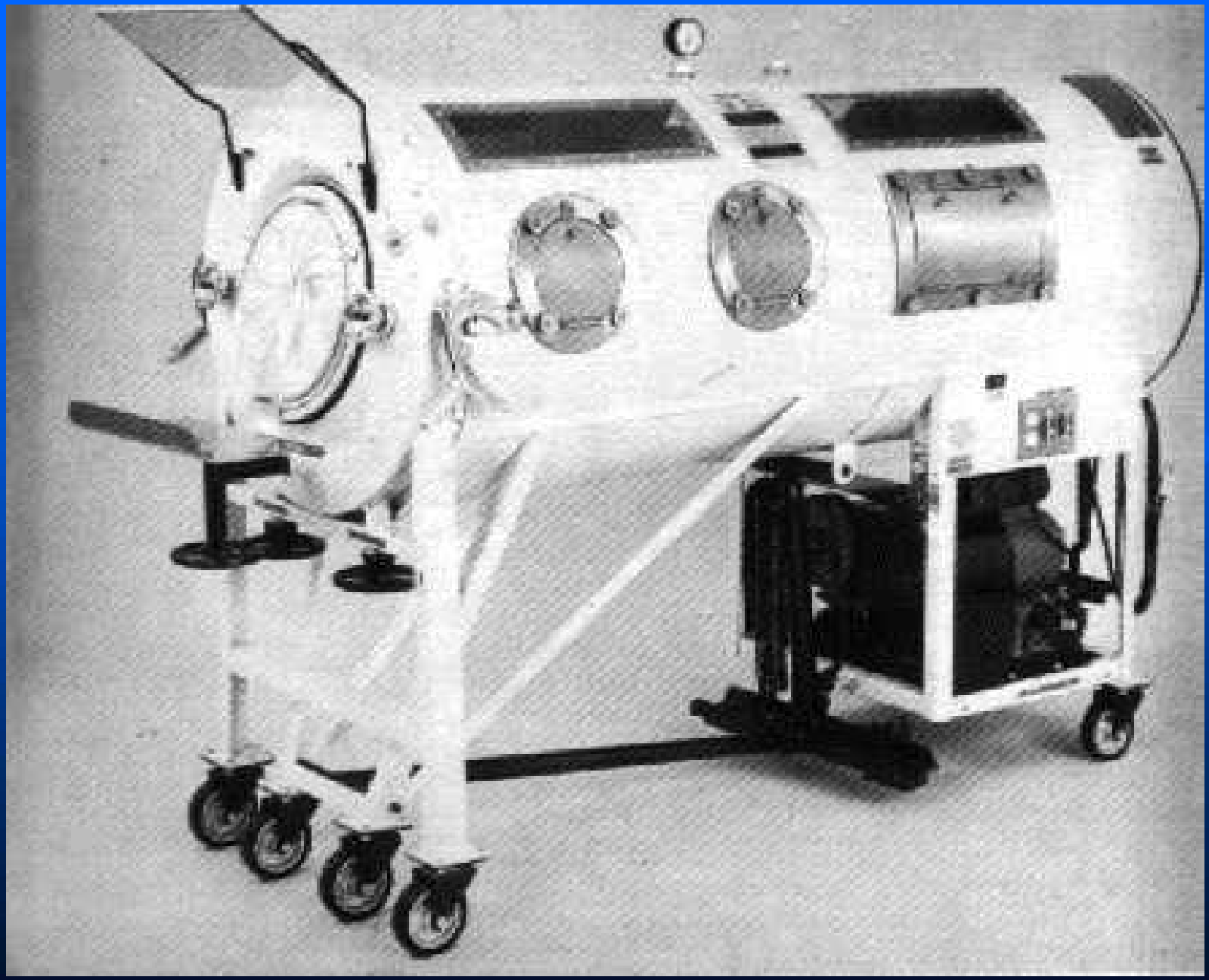
- **O_2 toxicity**:
  - very high levels(>1000 mmHg) CNS toxicity and seizures
  - lower levels (FiO_2 > 60%) and longer exposure: - capillary damage, leak and pulmonary fibrosis
  - PaO_2 >150 can cause retrolental fibroplasia
  - FiO_2 35 to 40% can be safely tolerated indefinitely
- **CO_2 narcosis:**
  - $PaCO_2$ 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





2. ARTIFITIAL 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

Sudden respiratory failure

- **PULMONARY EDEMA**
- **ACUTE RESPIRATORY DISSTRESS SYNDROME**

PULMONARY EDEMA

- Pulmonary edema is an increase in extravascular lung water
- Interstitial edema does not impair function
- Alveolar edema cause several gas exchange abnormalities
- Movement of fluid is governed by Starling's equation

$$QF = KF [(P_{IV} - P_{IS}) + \sigma (\pi_{IS} - \pi_{IV})]$$

QF = rate of fluid movement
KF = membrane permeability
P_{IV} & P_{IS} are intra vascular and interstitial hydrostatic pressures π_{IS} and π_{IV} are interstitial and intravascular oncotic pressures

σ reflection coefficient

- Lung edema is cleared by lymphatics

Adult Respiratory distress Syndrome (ARDS)

- Variety of unrelated massive insults injure gas exchanging surface of Lungs
- First described as clinical syndrome in 1967 by Ashbaugh & Petty
- Clinical terms synonymous with ARDS
  - Acute respiratory failure
  - Capillary leak syndrome
  - Da Nang Lung
  - Shock Lung
  - Traumatic wet Lung
  - Adult hyaline membrane disease

Risk Factors in ARDS

Sepsis	3.8%
Cardiopulmonary bypass	1.7%
Transfusion	5.0%
Severe pneumonia	12.0%
Burn	2.3%
Aspiration	35.6%
Fracture	5.3%
Intravascular coagulopathy	12.5%
Two or more of the above	24.6%

PATHOPHYSIOLOGY AND PATHOGENESIS

- Diffuse damage to gas-exchanging surface either alveolar or capillary side of membrane
- Increased vascular permeability causes pulmonary edema
- Pathology: fluid and RBC in interstitial space, hyaline membranes
- Loss of surfactant: alveolar collapse

CRITERIA FOR DIAGNOSIS OF ARDS

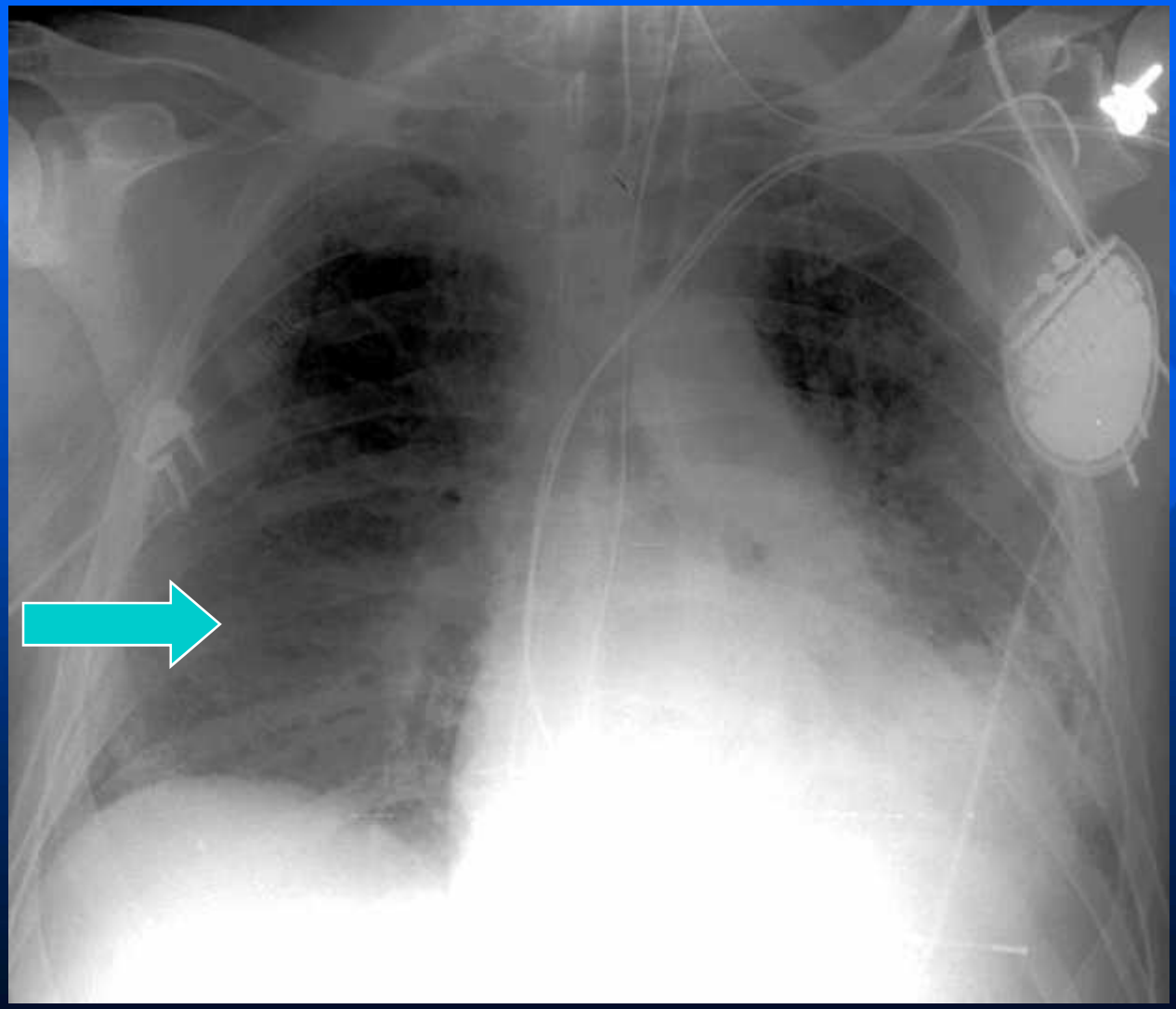
- Clinical history:

  Pulmonary or non pulmonary (shock, multi system trauma)
- Exclude:

  chronic pulmonary diseases
  left ventricular failure
- Typical in respiratory distress:

  tachypnea >20 breath/minute
  Labored breathing
  central cyanosis
  CXR- diffuse infiltrates
  PaO_2 <50mmHg FiO_2 >O.6
  Compliance <50 ml/cm H_2O increased shunt and dead space

ARDS



MANAGEMENT OF ARDS

- Mechanical ventilation
  corrects hypoxemia/respiratory acidosis
- Fluid management
  correction of anemia and hypovolemia
- Pharmacological intervention
  Dopamine to augment C.O.
  Diuretics
  Antibiotics
  Corticosteroids - no demonstrated benefit
  early disease, helpful 1 week later
- Mortality continues to be 50 to 60%