COPD Pathophysiology and Treatment Considerations

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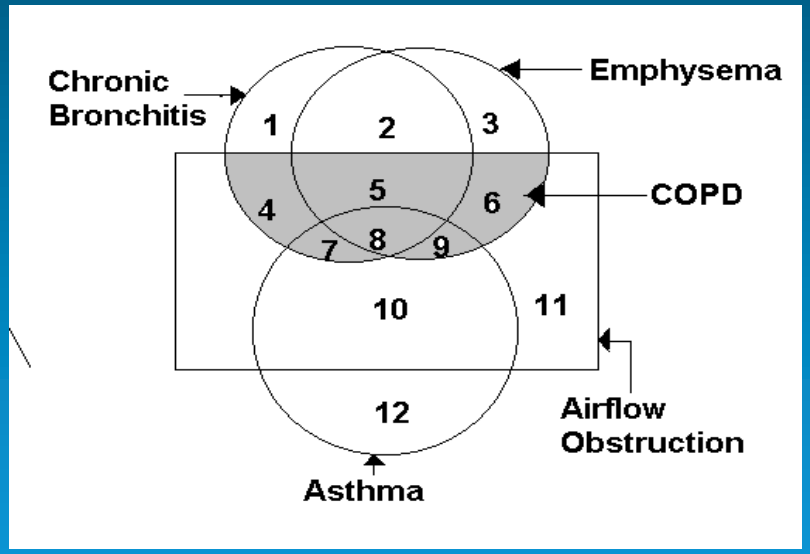
Chronic Obstructive Pulmonary Disease

- A disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema, with chronic respiratory symptoms (dyspnea, cough, sputum production and/or exacerbations).
- The diagnostic criteria is an FEV1/FVC of <70% obtained during a pulmonary function test.

COPD Disease Components

- Emphysema
- Chronic Bronchitis
- Asthmatic Bronchitis

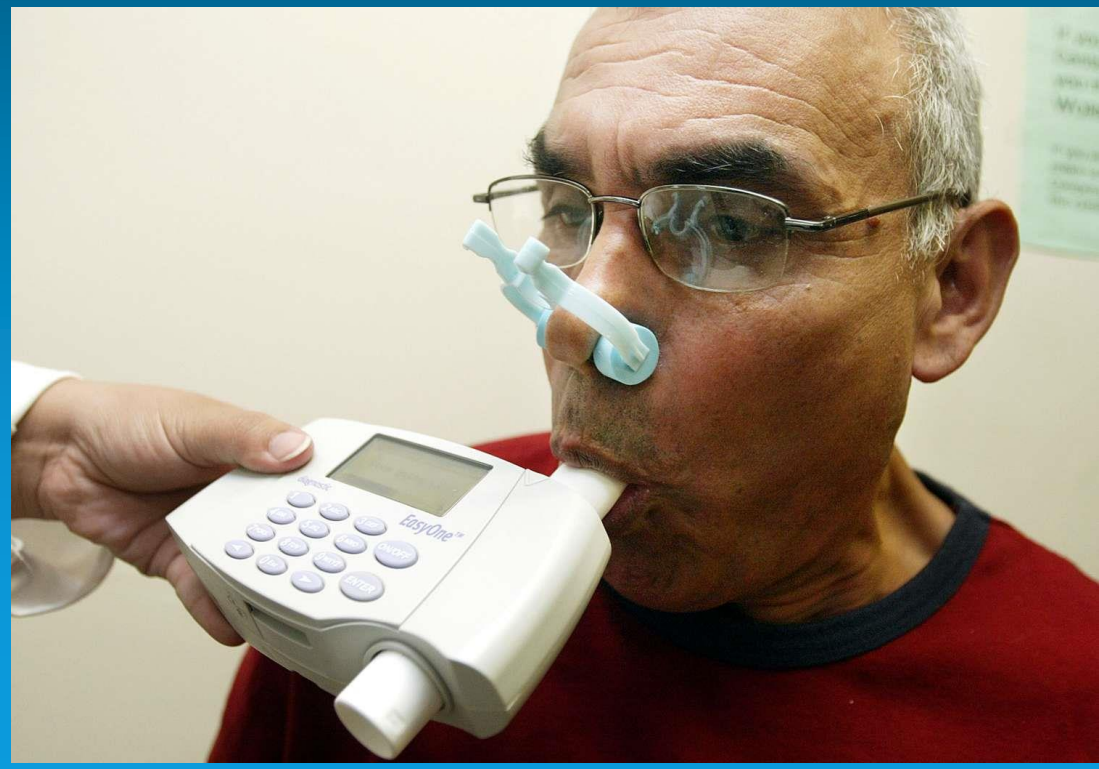


Quick facts about COPD

- 14 million persons in the US suffer from COPD
- 12.5 million from Chronic Bronchitis
- 1.65 from Emphysema
- About another 15 million undiagnosed
- Global prevalence 10.3% of population!!
- Third leading cause of death in the US
- Three million deaths worldwide per year
- Estimated 5.4 annual deaths by 2060.

COPD

- Reduced flowrates due to:
  - decreased elastic recoil
  - increased airway resistance
- Obstruction usually progressive
- May be partially reversible
- Generally accompanied by DOE



Knowledge check

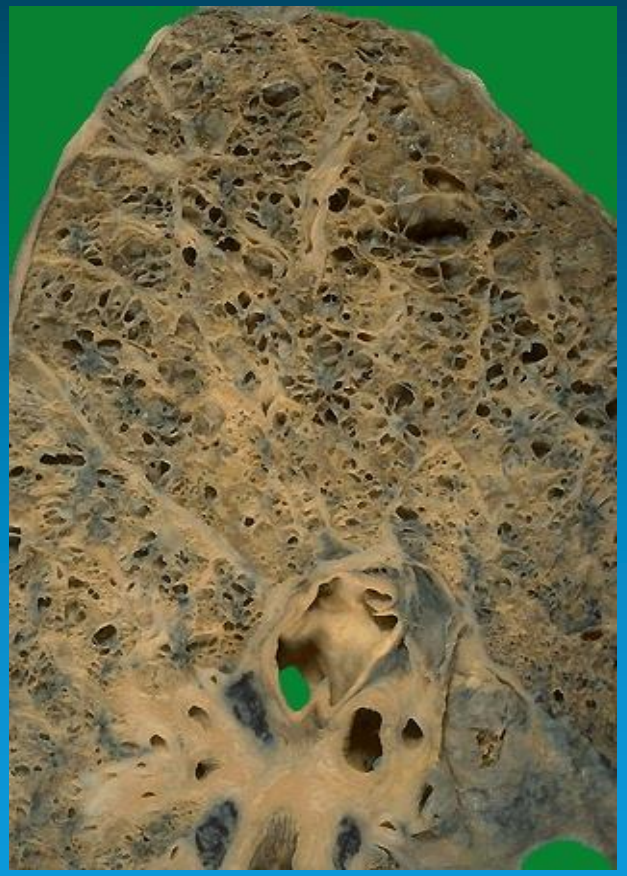
- Which two disease types constitute a diagnosis of COPD?
  - _____ or _____
- COPD is the _____ leading cause of death in the US.
- How many deaths each year are from COPD worldwide?
- Any questions or discussion?

Emphysema

A disease characterized by enlargement of the airspaces distal to the terminal bronchioles with loss of elastic fibers and destruction of alveolar septal walls.

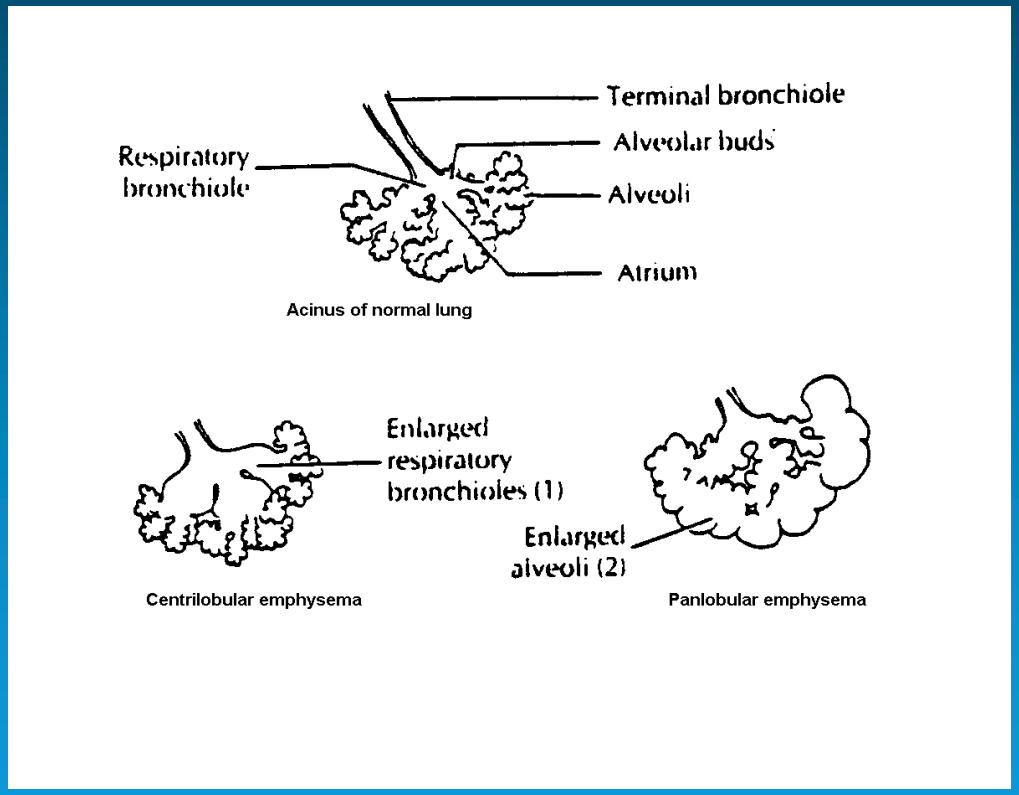
Lung Cross-Sections

Normal Specimen



Centrilobular emphysema

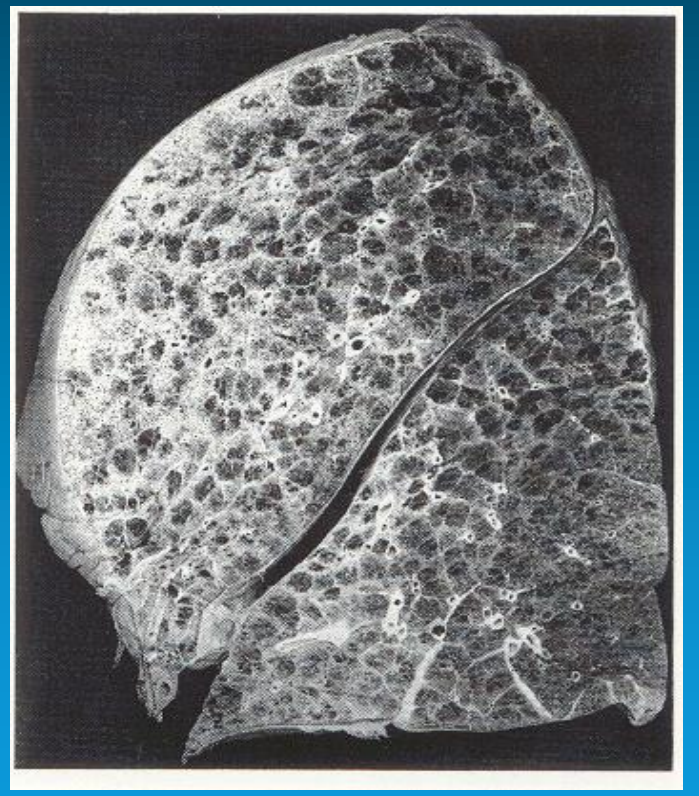
Types of emphysema



- Centrilobular
- Panlobular
- Bullous
- Subcutaneous
- Pulmonary Interstitial Emphysema (PIE)

Centrilobular emphysema

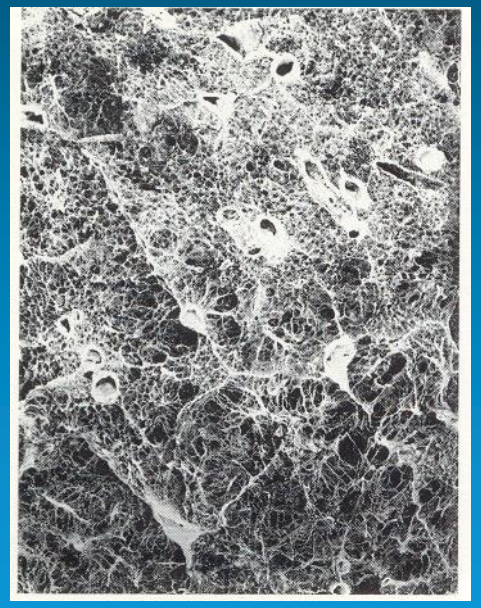
- Primarily respiratory bronchioles
- Predominates in upper lobes and upper segments of lower lobes



Centrilobular emphysema

- High correlation with smoking
- Associated with chronic bronchitis
- Male>female

Panlobular emphysema

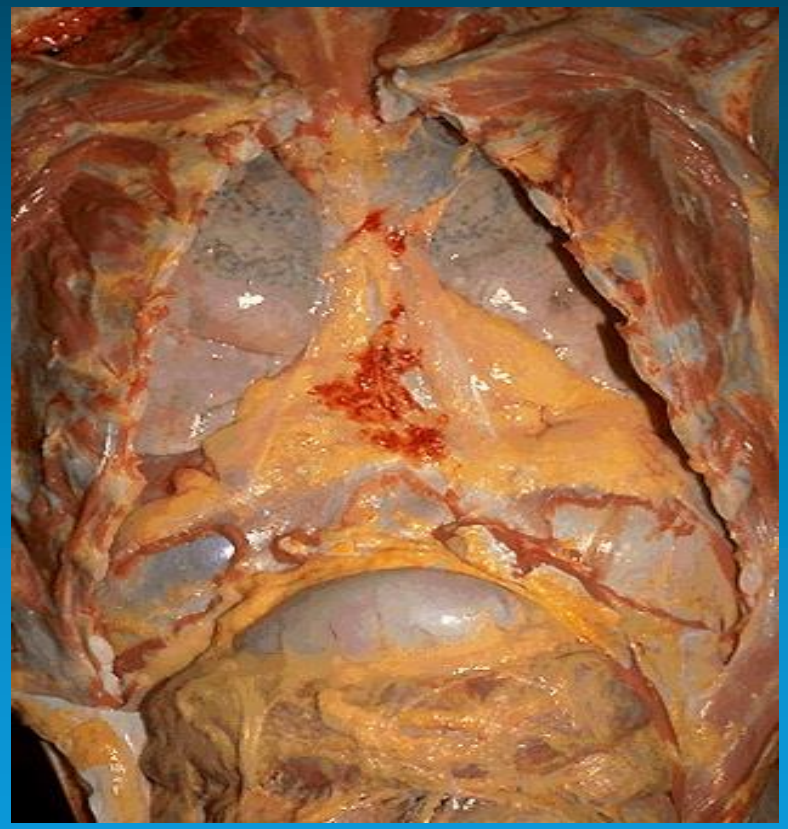


- Involves all alveoli
- Associated with α-1 antitrypsin deficiency
- Part of aging process
- male=female

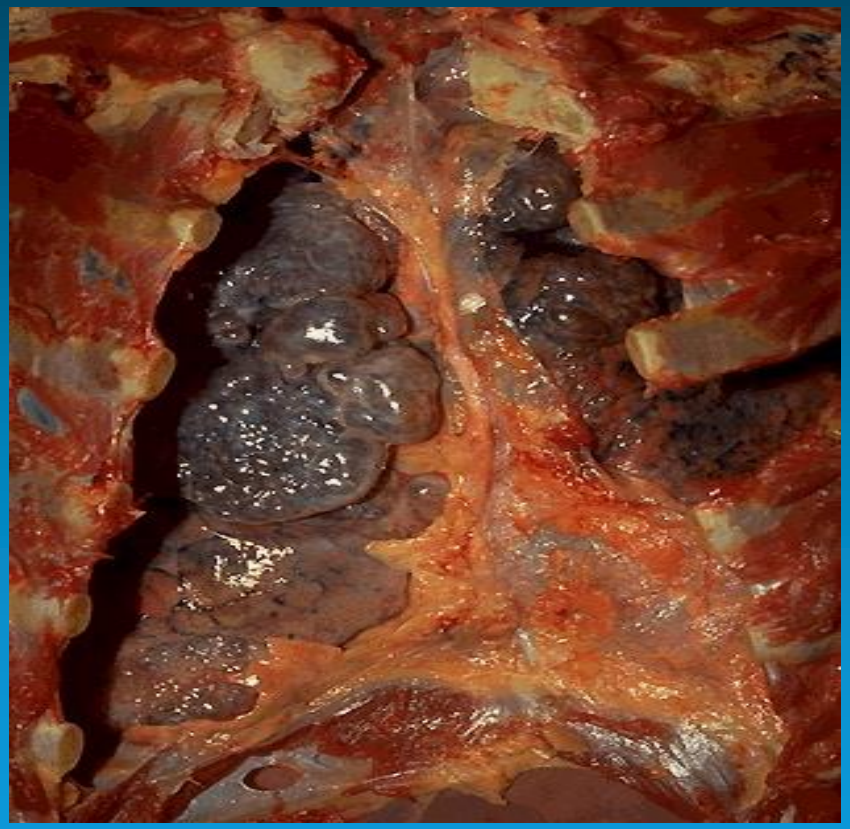
Bullous emphysema

- Thin walled, multiple cysts
- Bulla > 1 cm
- Bleb = superficial subpleural
- Cyst = large
- Pneumatoceles = huge
- Increased risk of pneumothorax

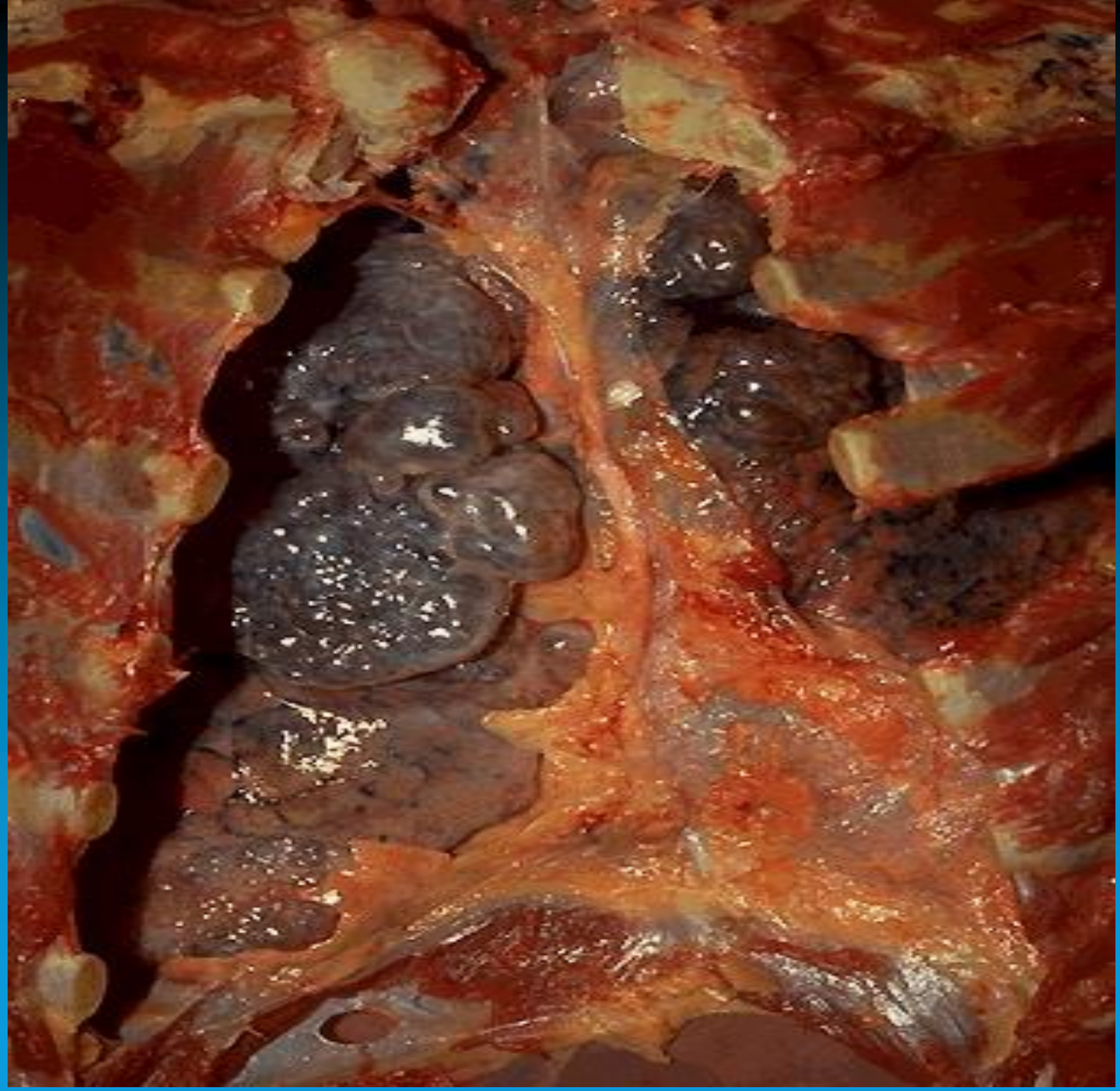
Gross pathology-Emphysema



Normal Specimen



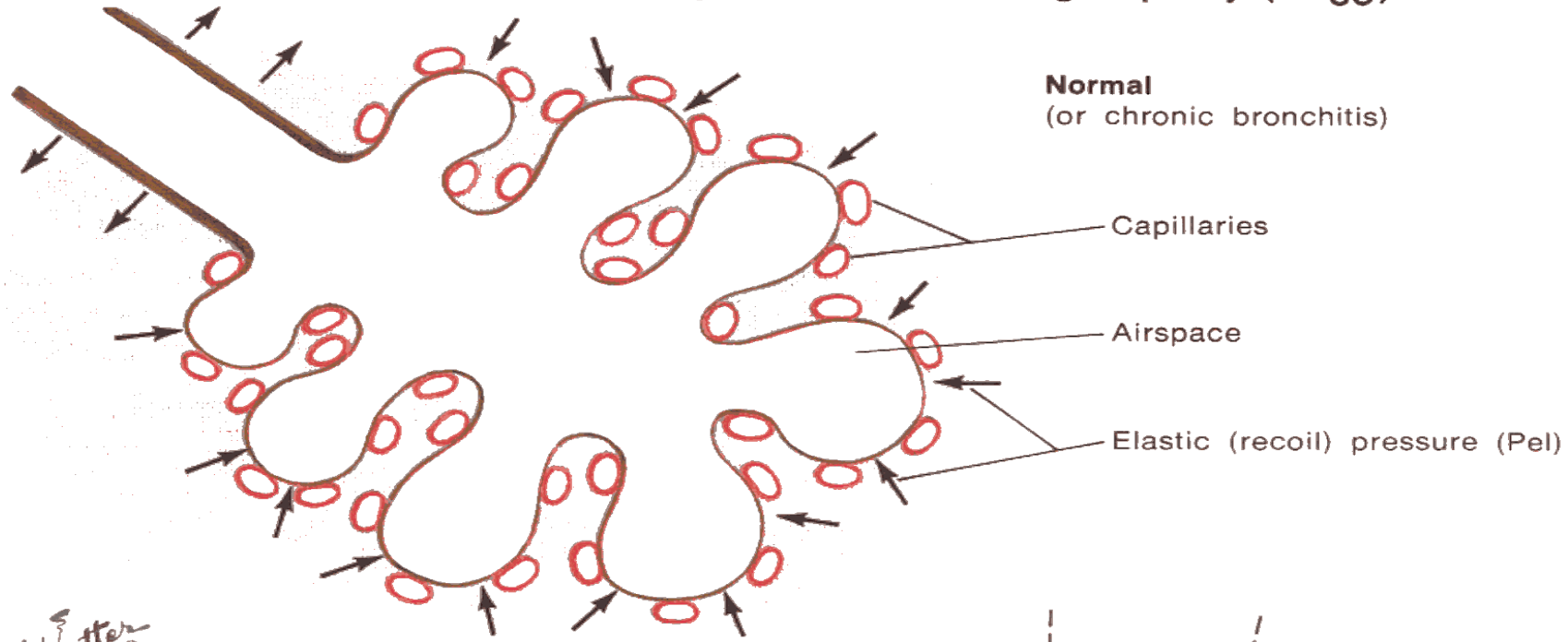
Bullous Emphysema



Summary of structural defects

- Enlargement of airspaces
- Loss of elastic fibers
- Destruction of alveolar septal walls
- Destruction of capillary bed
- Airway flabbiness (loss of support)

Effect of Emphysema on Compliance and Diffusing Capacity (DL_{CO})



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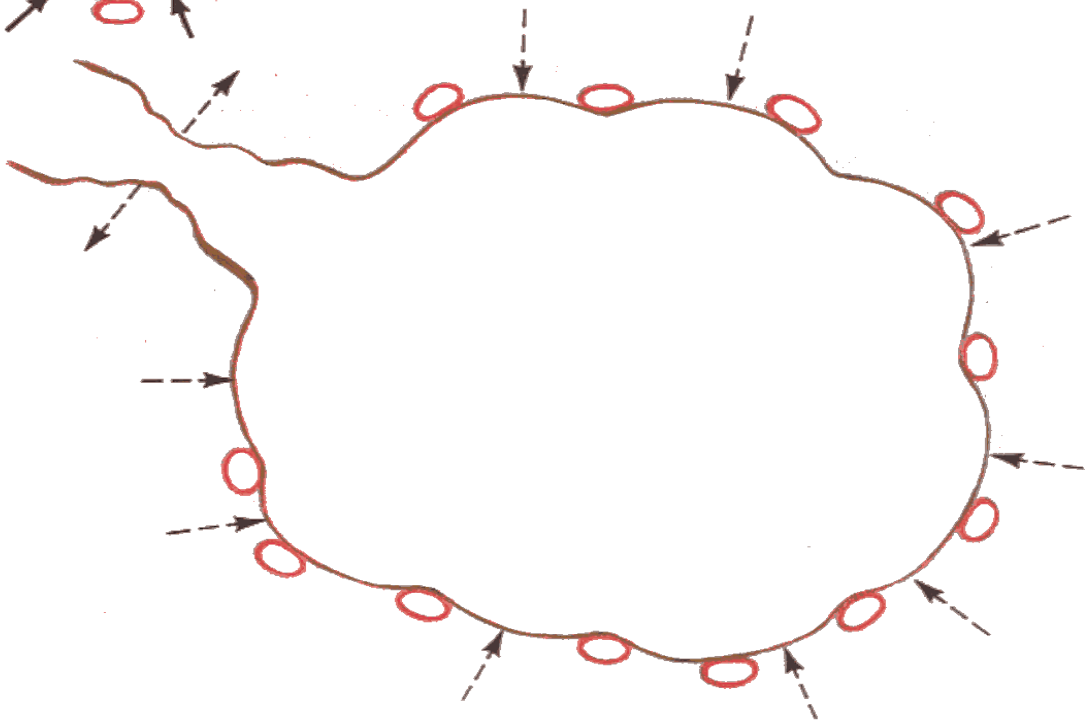


Emphysema

- Decreased alveolar surface area
- Decreased elastic recoil
- Increased alveolar gas volume
- Fewer capillaries

↓

- Increased compliance ($\Delta V / \Delta P$)
- Impaired diffusion
- Increased lung volume

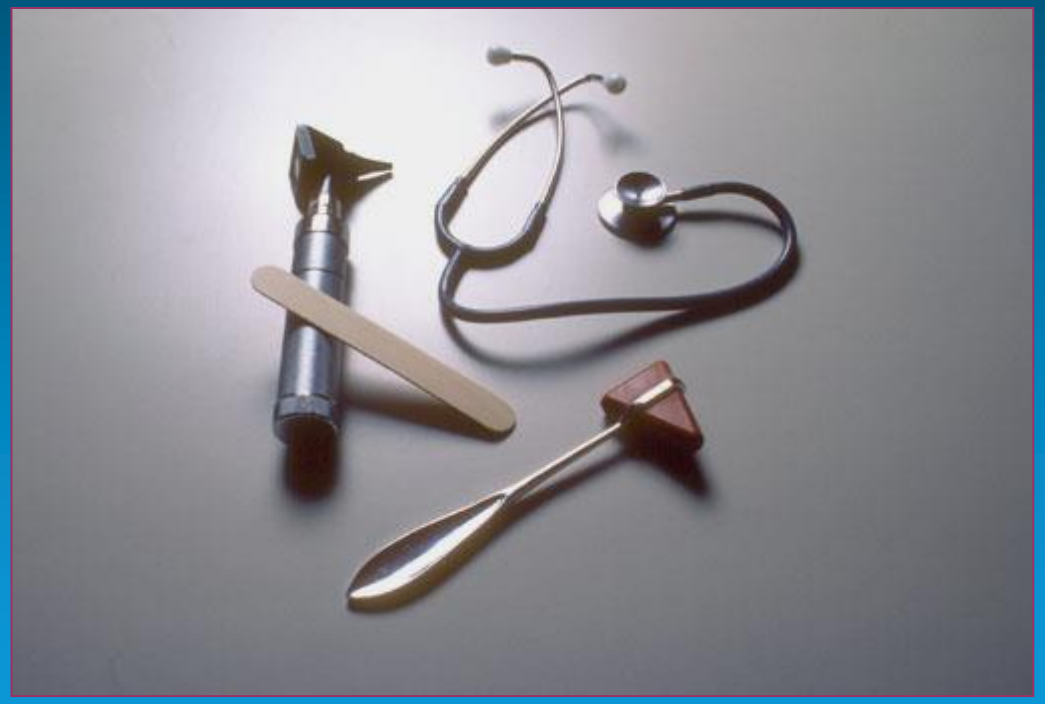
Emphysema
Physiologic defects

- Loss of elastic recoil
- Premature airway collapse on exhalation
- Increased TLC and RV
- Impaired oxygen diffusion
- Slight or no V/Q imbalance

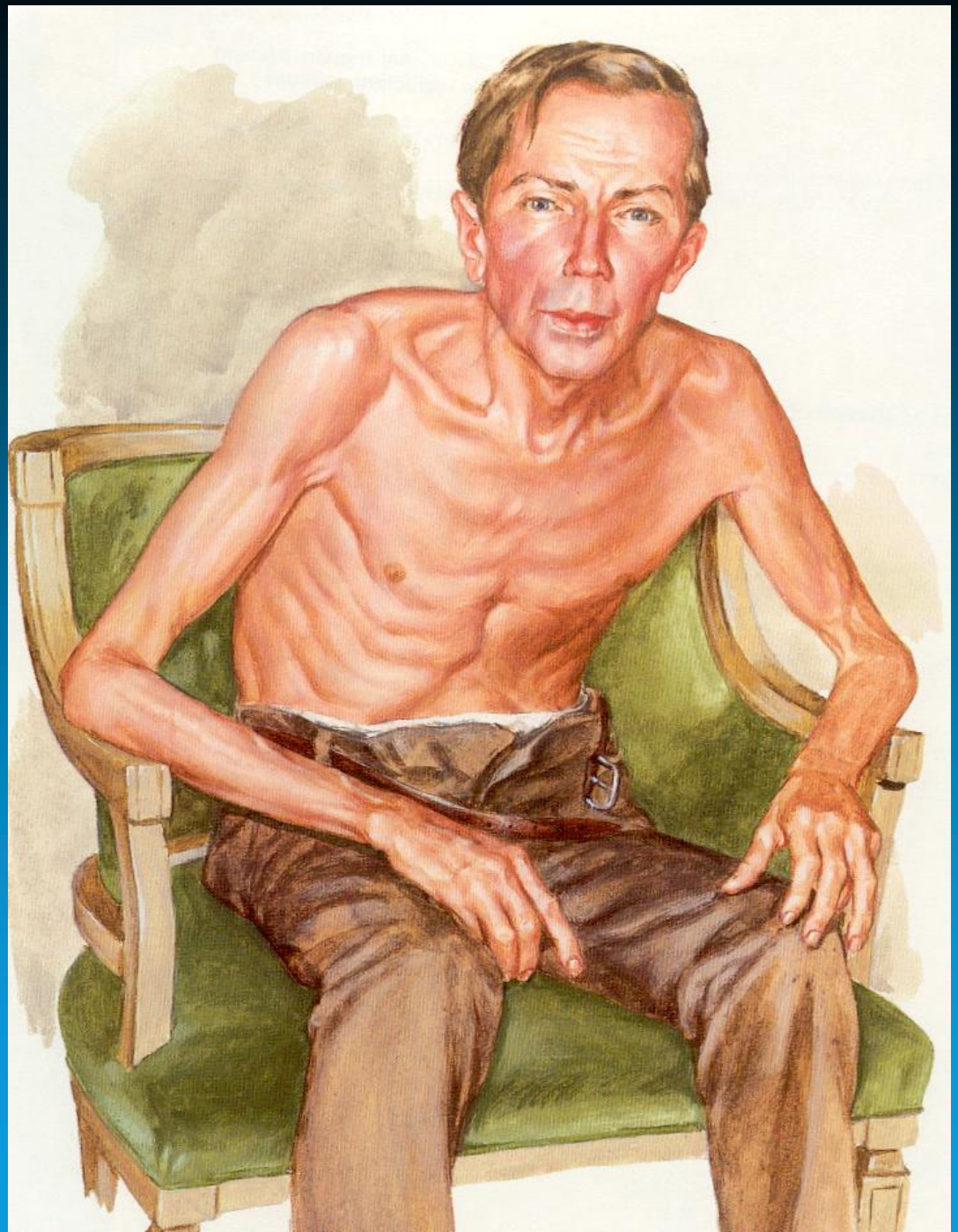
Etiology of Emphysema

- Cigarette Smoking
- Genetic predisposistion
  - Alpha 1 antitrypsin defiency
- Occupational exposure to chemical irritants
- Exposure to atmospheric pollutions

Clinical Presentation



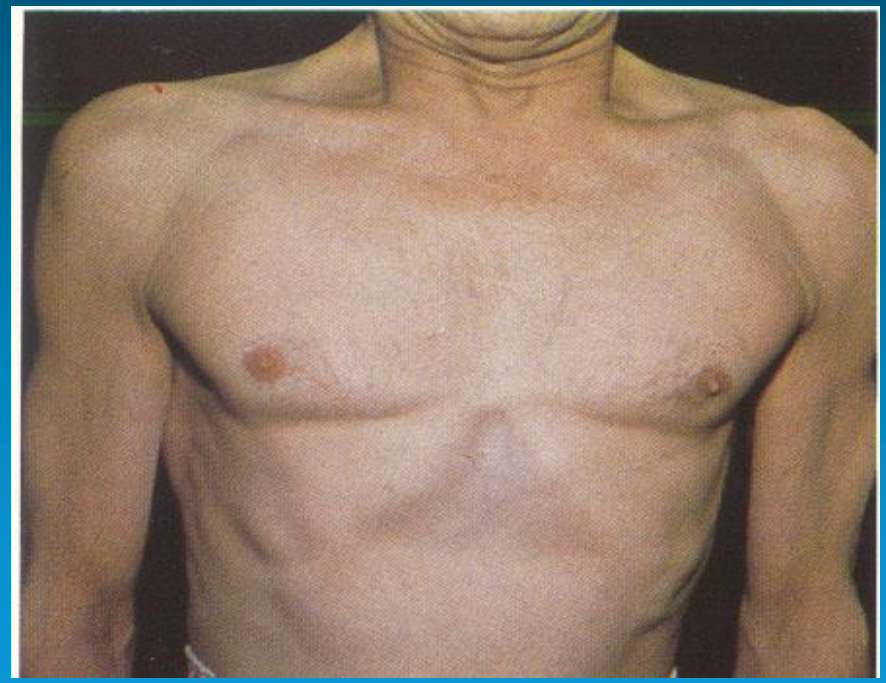
- 60 to 80 years old
- Accessory Muscle Use
- Scant sputum
- Severe dyspnea
- Pursed-lip breathing
- Thin body habitus

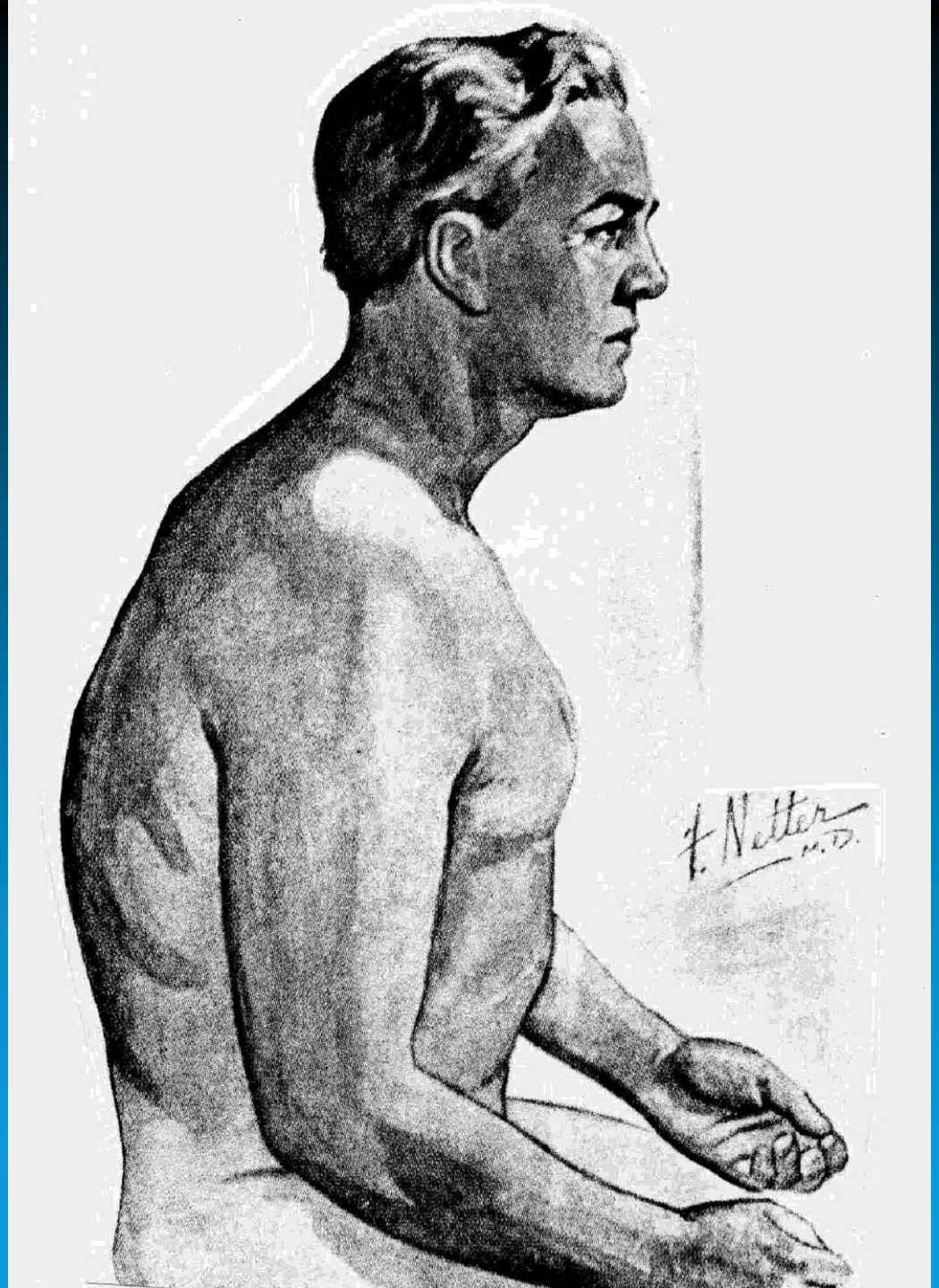


Pink
Puffer

Barrel Chest in Emphysema

- Horizontal ribs
- Prominent sternal angle
- Increased anteroposterior diameter

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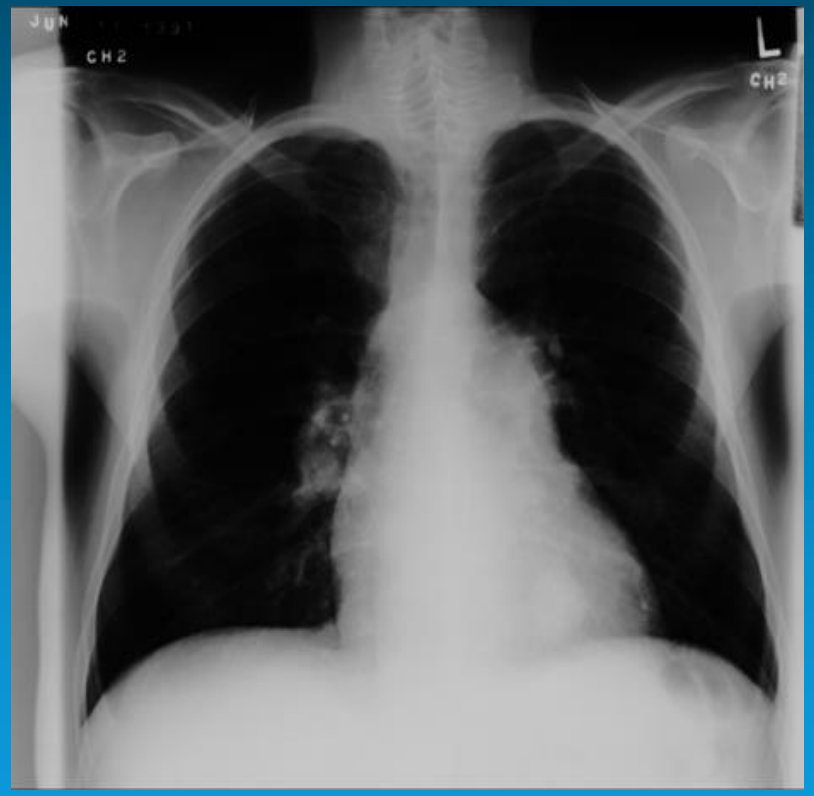
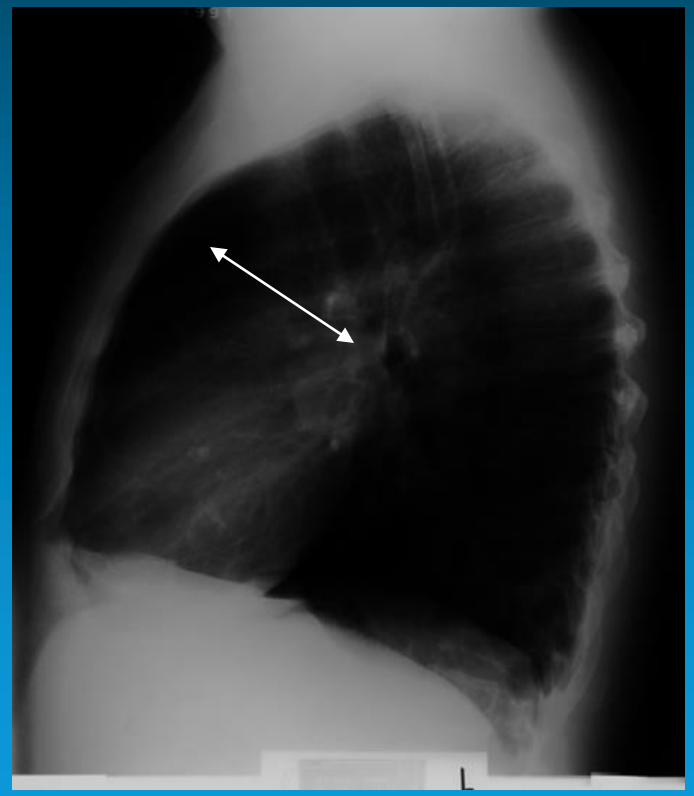
Chest x-ray

- Hyperlucency
- Narrow heart
- Flattened diaphragm

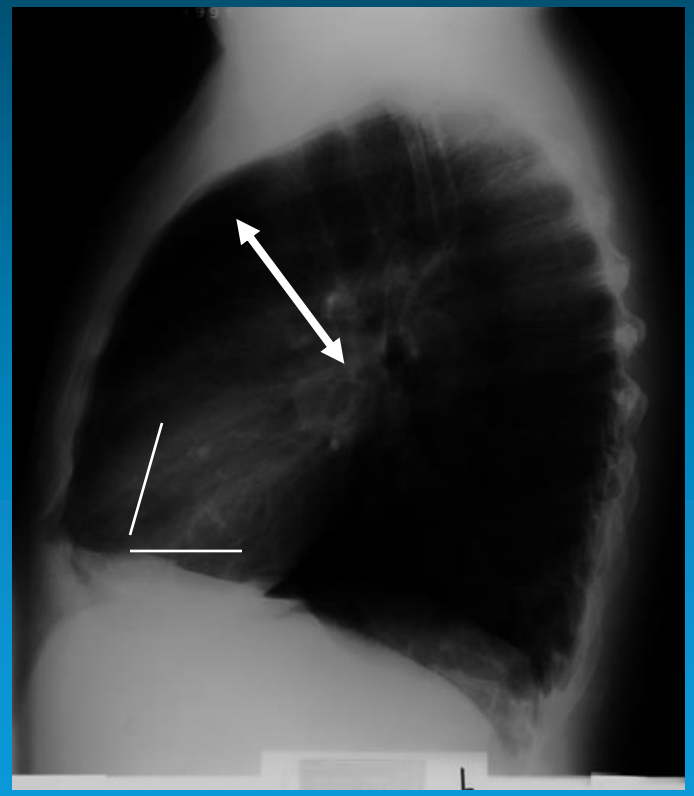


Lateral chest x-ray



- Increased retrosternal airspace

Lateral chest x-ray



- Increased retrosternal airspace
- Sternophrenic angle >90 degrees

Knowledge check

- As a result of emphysema, name two structural defects in the pulmonary system.
  - Airspace enlargement, loss of elasticity, destruction of alveolar walls, destruction of capillary beds, airway flabbiness.
- What are the primary reasons why emphysema develops (etiology)?
  - Cigarette smoking (vaping)?, genetics, exposure to chemical irritants, exposure to environmental irritants
- Any questions or discussion?

Chronic Bronchitis

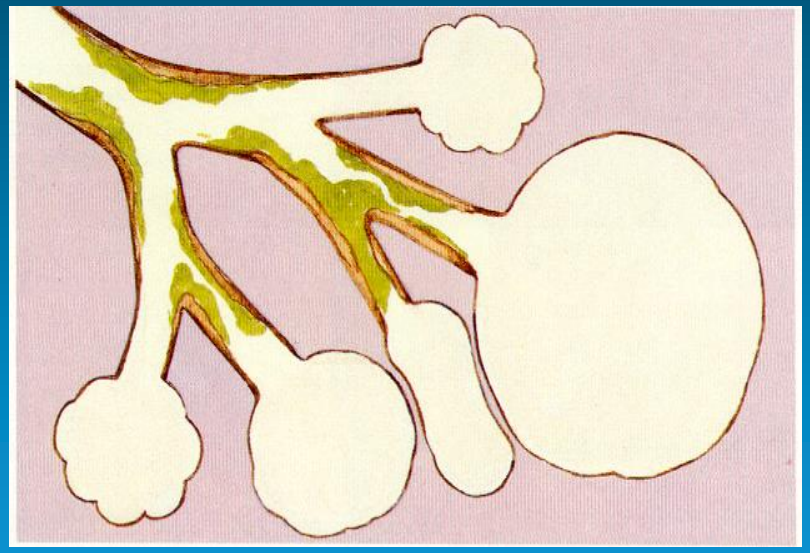
A disease characterized by cough and sputum production on most days for at least 3 months of the year for two or more consecutive years that is not due to another specific disease.

Summary of Structural Defects in Chronic Bronchitis

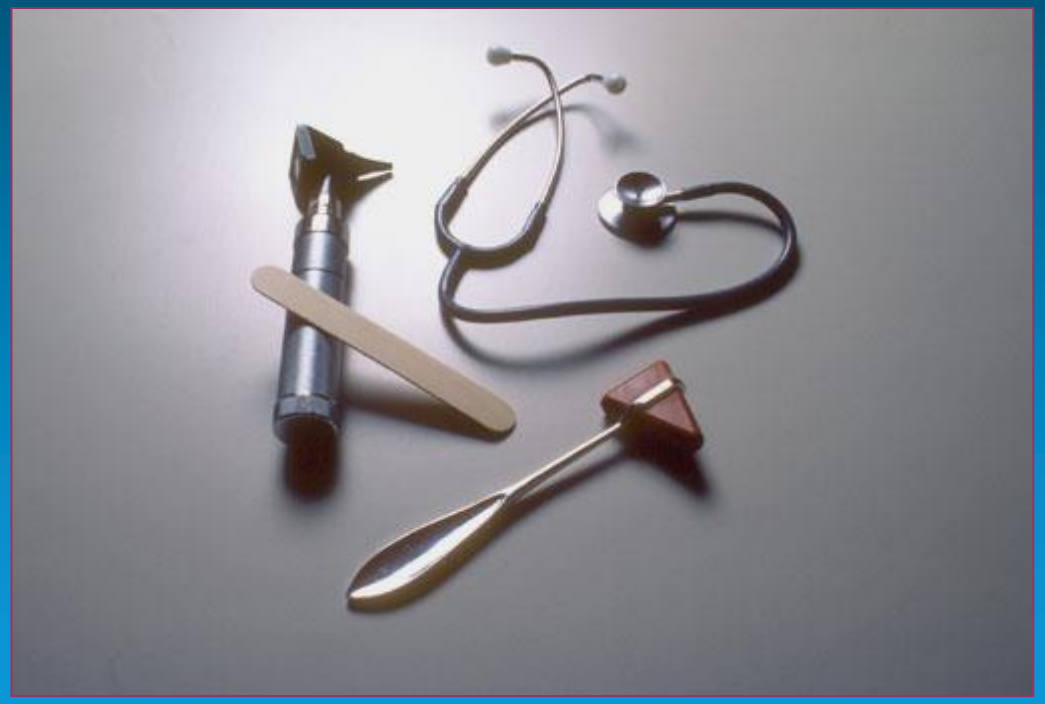
- Hypertrophy of mucous glands
- Increase number of goblet cells
- Loss of cilia
- Squamous metaplasia
- Chronic leukocytic and lymphocytic infiltration

Chronic Bronchitis Physiologic Defects

- Impaired mucociliary escalator
- Impaired ventilation
- Normal perfusion
- V/Q imbalance
- Hypoxemia



Clinical Presentation Chronic Bronchitis



- 40 to 60 years old
- Copious sputum
- Mild to severe dyspnea
- Cyanosis
- Tendency to obesity

Blue Bloater

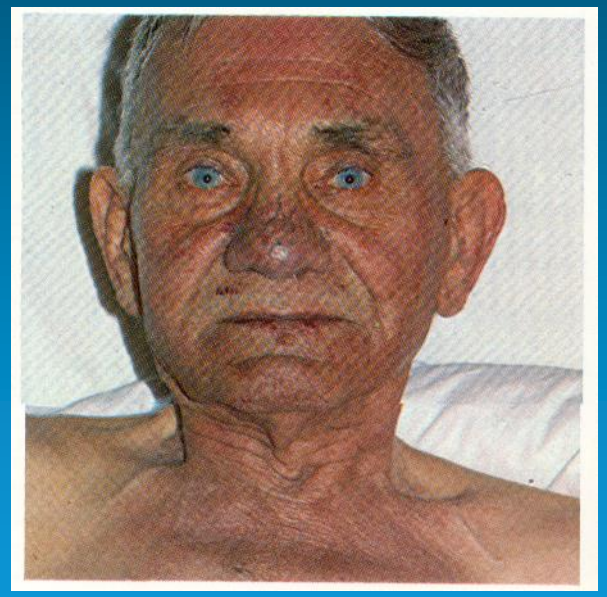
Cor Pulmonale

Right heart hypertrophy and failure secondary to chronic hypoxia

Cor pulmonle

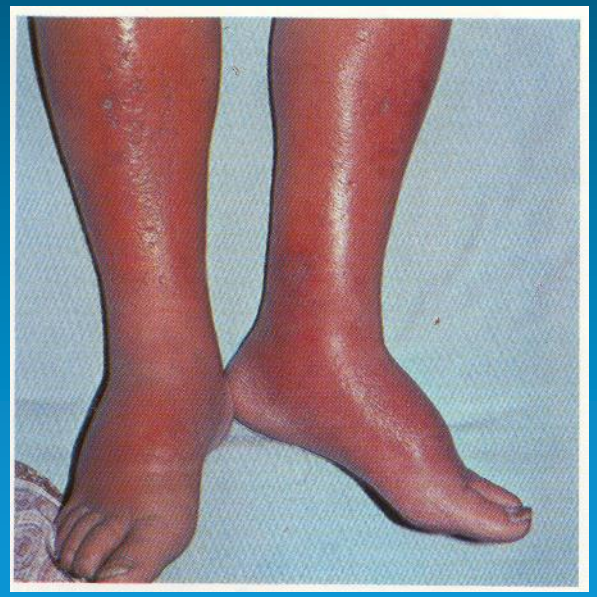
- Chronic hypoxia leads to:
- Pulmonary vasoconstriction (↑ PVR)
- Pulmonary hypertension
- Increased work of the right heart
- Ventricular hypertrophy
- Right heart failure

Cor pulmonale



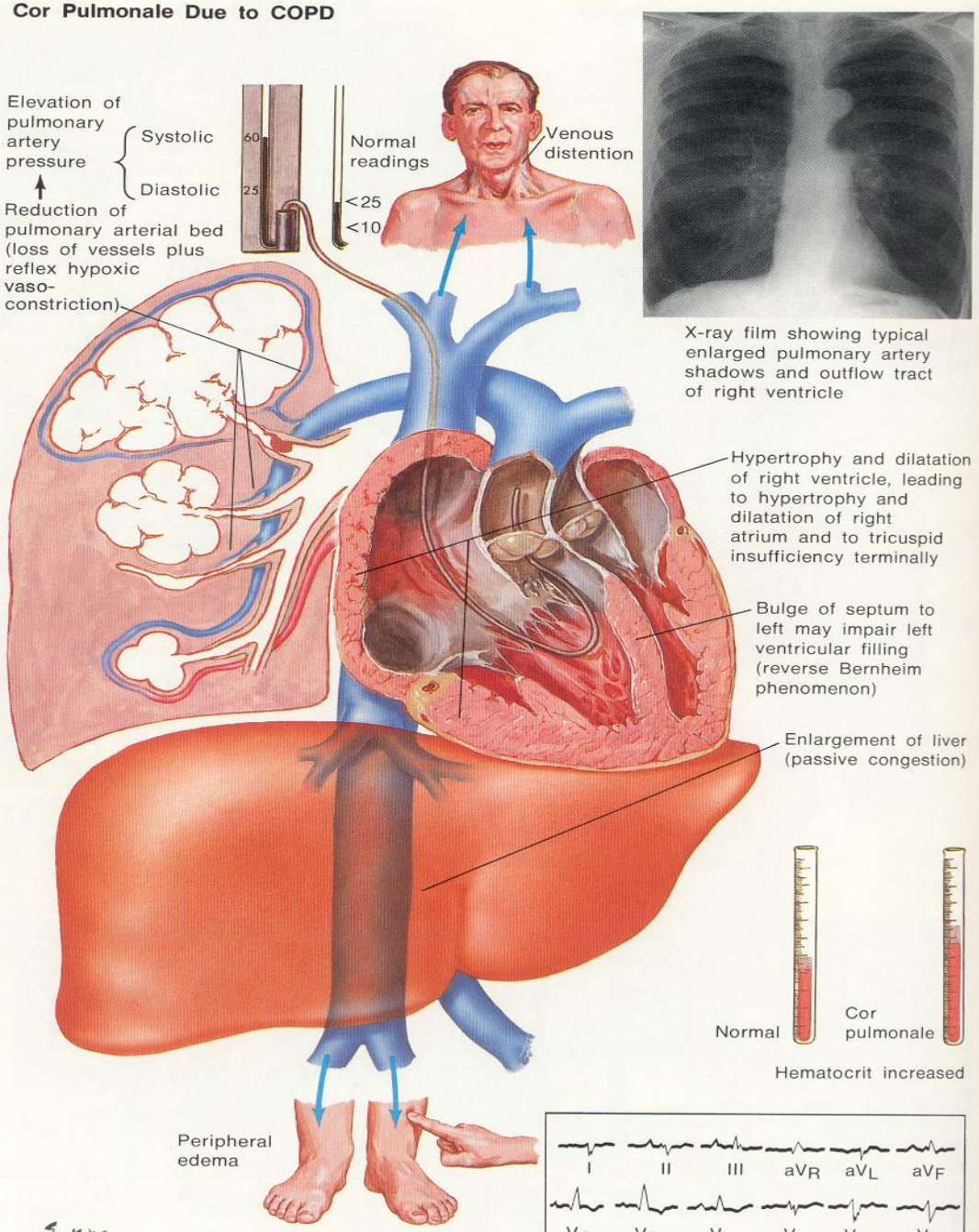
- Central cyanosis
- Engorged neck veins

Cor pulmonale



- Pedal edema

Cor Pulmonale Due to COPD



X-ray film showing typical enlarged pulmonary artery shadows and outflow tract of right ventricle

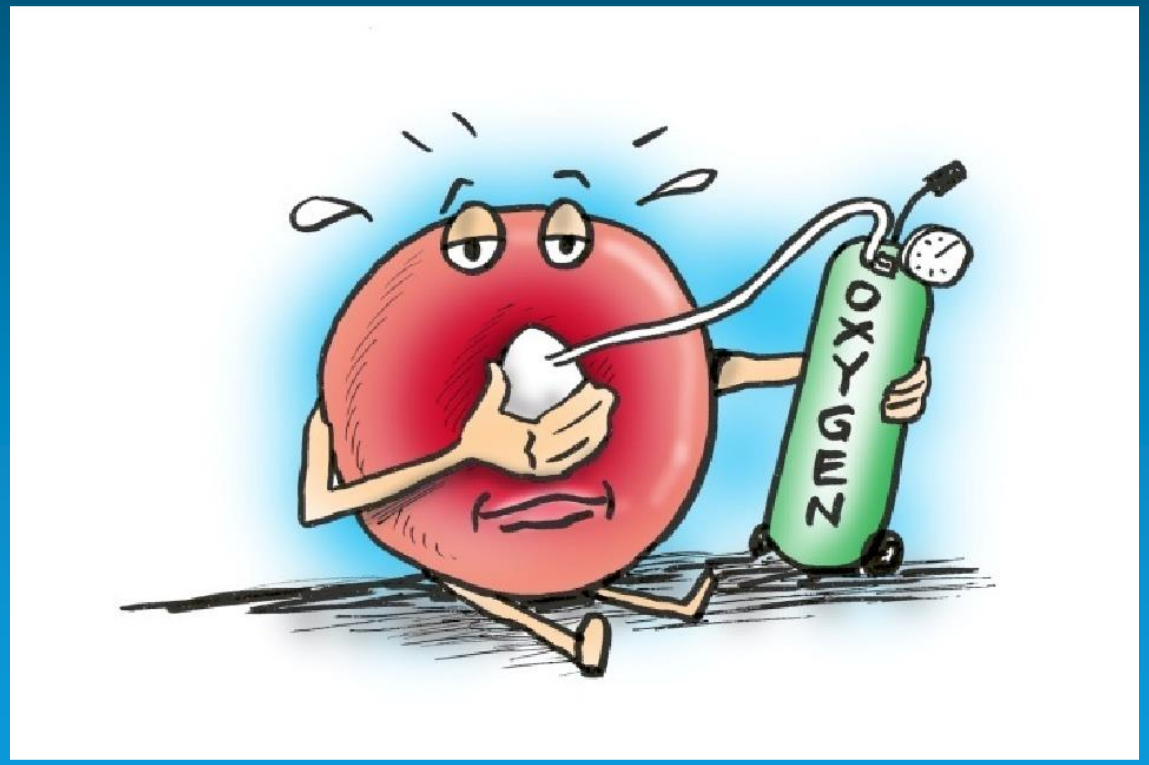
Controlled oxygen therapy

- Avoid PaO_2 levels above 65-70 mmHg
- SpO_2 88-92%
- Low flow/low FIO_2

Can You Give a COPD patient O2?

- Absolutely
- Long-term administration (>15 hours/day) has been shown to increase survival in patients with severe resting hypoxemia.
- PaO2 <55 mmHg

Knocking out the Hypoxic Drive



Thought of knocking out the Hypoxic Drive of a COPD patient

- Chronic CO_2 patients not as responsive to $PaCO_2$ changes due to CSF pH compensation and Arterial pH compensation from renal system
  - Note that not ALL COPD patients are CO_2 retainers, nor are all CO_2 retainers COPD patients

- All of this compensation blunts their ability to respond to $PaCO_2$ changes as rapidly, so PaO_2 becomes more important to their drive to breath.

West's Respiratory Physiology. The Essentials. 10th edition

Control of Breathing Refresher

- Central Chemoreceptors
  - Surrounded by brain extracellular fluid and respond to H+
  - CO_2 from the blood diffuses into CSF and then releases H+ due to hydrolysis
    - In normal states, blood brain barrier not permeable to H+
    - In severe acidosis it can become semipermeable to H+
  - Lower buffering capability in CSF due to lower protein levels, so pH changes more rapidly than blood
  - Long term pH changes in CSF will result in HCO_3 changes (just like blood)

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Control of Breathing Refresher

- <u>Peripheral Chemoreceptors</u>
  - Located in Carotid arteries and aortic arch
  - Respond to decreases in PaO_2, pH(Carotid Only), and $PaCO_2$
    - Little stimulation occurs until PaO_2 <100 mmHg
    - Responsible for all increases in Ve due to hypoxemia
    - Responsible for increased Ve during a metabolic acidosis

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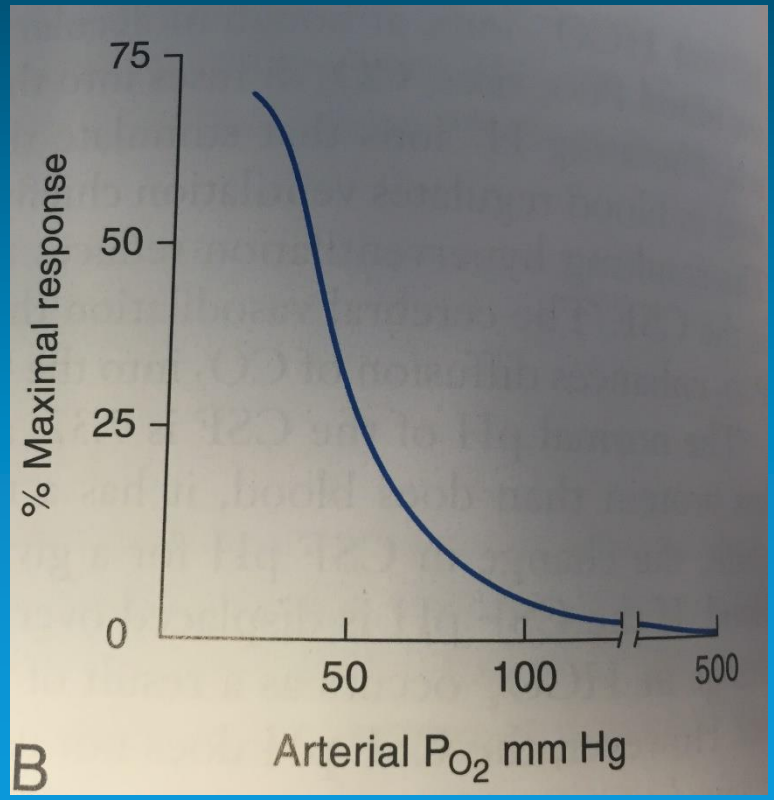


Control of Breathing Refresher

- Response to Oxygen
  - "When Alveolar PCO_2 is kept at about 36 mmHg, the alveolar PO_2 can be reduced to the vicinity of 50 mmHg before any appreciable increase in ventilation occurs. Raising the PCO_2 increases the ventilation at any PO_2. Note that when the PCO_2 is increased, a reduction in PO_2 below 100 mmHg causes some stimulation of ventilation, unlike the situation in which the PCO_2 is normal."

  - West's Respiratory Physiology. The Essentials. 10th edition

Can Oxygen Increase CO_2

- Yes, if PaO_2 is raised above 100 mmHg
- Yes, due to changes caused by vasodilation due to reversal of hypoxemia, which will lead to V/Q changes in poorly ventilated units
- Yes, due to the Haldane effect

- NOT DUE TO KNOCKING OUT DRIVE

Studies on this

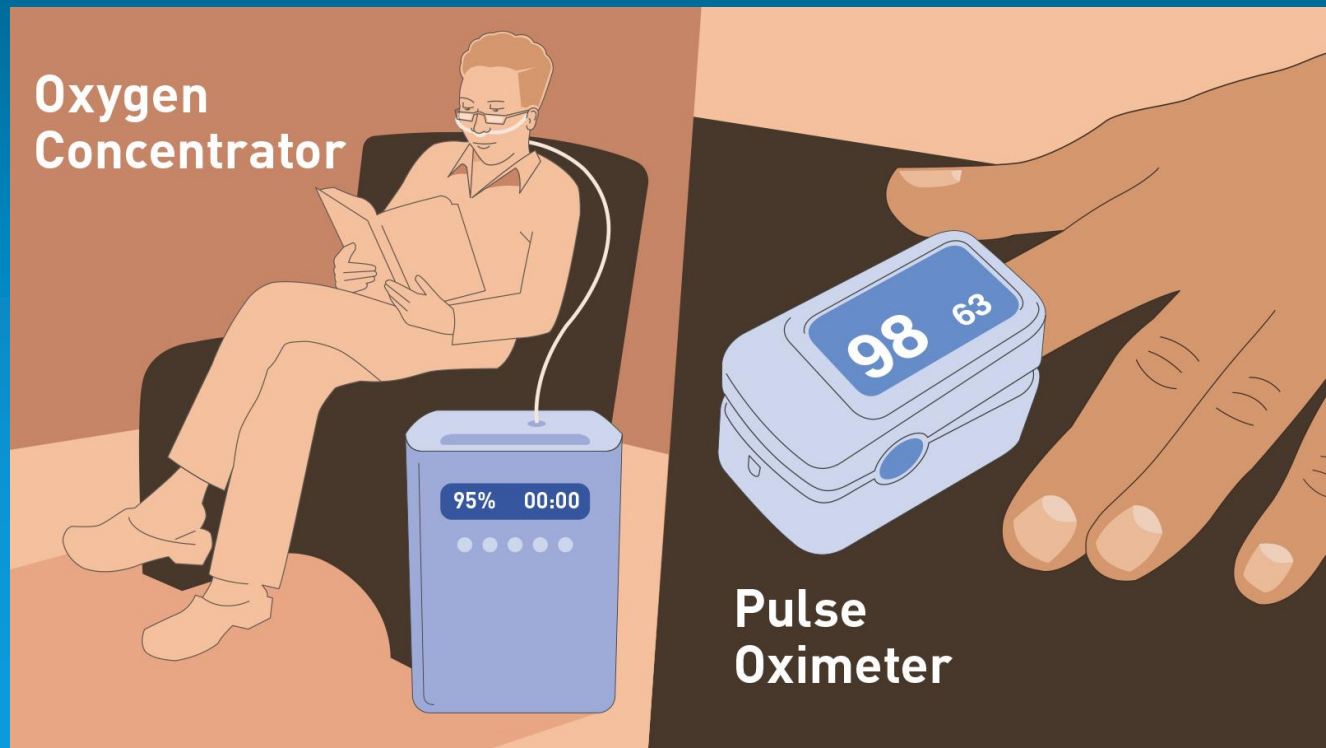
- Earliest study was back in the 60's and was just a few patients
- In the 80's – multiple studies demonstrated that CO_2 retaining COPD patients had NO changes in RR when PaO_2 and SpO_2 were increased
- Recent study, 2014, found that during NIV an increase in FiO_2 did NOT increase $PaCO_2$ in Co_2 retaining patients.

What you should know

- Cyanotic is cyanotic (blue is blue) – Give them O_2
- As patients move they require more O_2
- As patients move they increase their inspiratory flowrates, which decreases the delivered FiO_2 from any low flow device. So, an increase in flow to the NC might be needed just to maintain the same delivered FiO_2.

How are home O2 orders filled/completed

- Most commonly, DME company has a contract or relationship with hospital system.
- What does the patient insurance cover?
- Pay out of pocket



Let's talk about oxygen delivery

- Flow is the most important concept
- Flow = volume/time
- Flow = tidal volume/inspiratory time
- Flow demand increases exponentially with effort/shortness of breath
- Example: a normal breathing patient has a tidal volume of 500 mL and an inspiratory time of 1 second
  - That is a flow rate of 30 L/min
- If the same patient became short of breath, and reduced their inspiratory time to 0.5 seconds.
  - That is a flow rate of 60 L/min

Let's talk about oxygen delivery

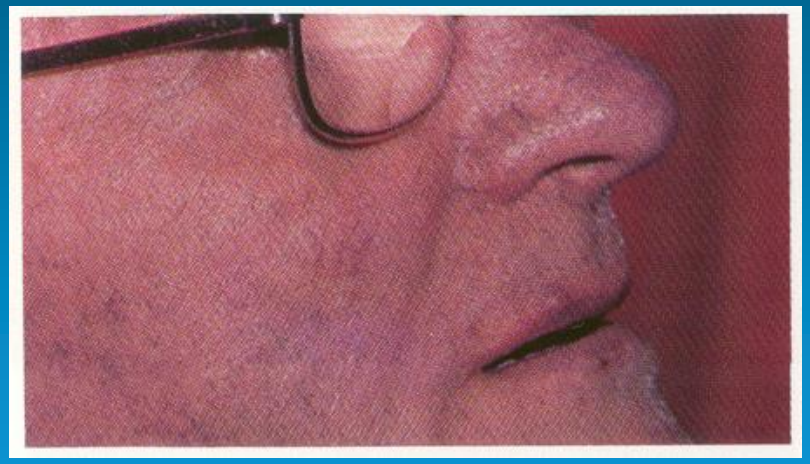
- If the patient was on continuous flow O2 at 2 L/min
- …and they changed from a flow rate of 30 to 60 L/min
- The oxygen being delivered to the lungs would be cut in half, if the O2 delivery was unchanged.
- How would we combat this problem?

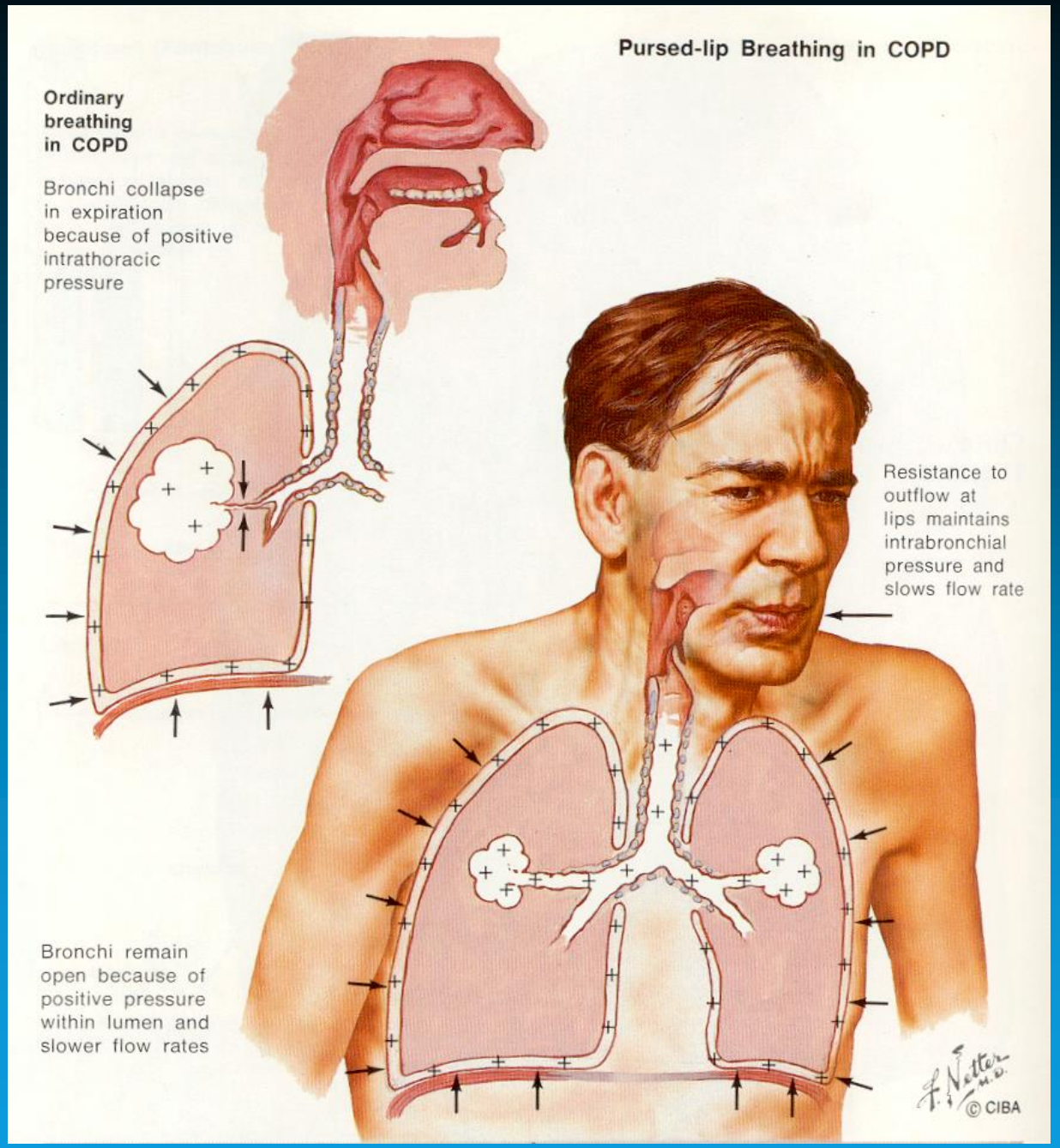
Oxygen delivery via POC

- Rate responsive therapy.
  - FiO2 output from device remains constant despite increased patient effort.
  - Makes it less likely that titration is needed when ambulating vs. at rest.
- Not the case with minute volume devices

Pursed-lip Breathing

- Spontaneous or taught
- Helps prevent premature airways collapse
- Can increase PaO_2




Pursed-lip Breathing in COPD
Ordinary breathing in COPD
Bronchi collapse in expiration because of positive intrathoracic pressure
Resistance to outflow at lips maintains intrabronchial pressure and slows flow rate
Bronchi remain open because of positive pressure within lumen and slower flow rates
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Treatments for COPD patients

- Bronchodilators
  - Beta agonist – albuterol
  - Anticholinergics – atrovent, spirivia
- Steroids
  - Inhaled
  - PO
  - IV

- COPD Patients - 430-720 kcal per day of WOB
  - 10 times more than normal individuals

Knowledge check

- What is the specific indication and duration for long-term oxygen therapy?
  - Resting PaO_2 of <55 mmHg (SpO_2 <88%), more than 15 hours/day
- Why do patients require more O_2 when ambulating, compared to at rest?
  - Increased inspiratory flow demand
- What are the primary characteristics of chronic bronchitis?
  - Cough and sputum production for 3 months of the year for 2 or more consecutive years.

Thank you!

- Questions?

- Contact info
  - burkb@otc.edu

Primary reference

- Global initiative for chronic obstructive pulmonary disease. 2023 Report. COPD Gold guidelines. Retrieved from https://goldcopd.org/2023-gold-report-2/