COPD

Epidemiology

- preventable, treatable, and true extra pulmonary effects
- airflow limitations not fully reversible
- progressive limitations with abnormal inflammatory response
- stimulated by fumes and dust
- Most common in places where tobacco is heavily used
- 3rd leading cause of death in the US
- 5th most common cause of death worldwide
- Asia and Africa with highest mortality

Risk factors

- tobacco
- occupational (coal, cotton fibers, miners, tunnels, concrete)
- genetics (a1ATD)
- Pollution, second hand
- biomass smoke in developing world (women more than men)
- asthma, TB are possible risk factors

Dx

- spirometry required
- post bronchodilator FEV1 / FVC < .70 (not FEV1 in isolation)
- after 4 puffs albuterol
- Clinical aside: fixed ratio may over diagnose COPD in elderly due to reduced flows due to normal aging
- watch for “combined pulmonary fibrosis and emphysema”
    - preserved airflow (ratio) but decreased DLCO
    - common among male smokers
    - frequent pHTN with suggests poor prognosis
    - Upper lobe emphysema and lower lobe fibrosis

COPD vs asthma

- **macrophages** vs mast cells
- **CD8** vs DC4
- **Neutrophils** vs Eosinophils
- **airway obstruction not completely reversible** vs fully reversible

Pathophysiology

- Increasing tissue in airway wall causes airflow obstruction
- Mucous hypersecretion
- Breakdown of alveolar attachments (emphysema)
- Inflammation and fibrosis (obliterative bronchiolitis)

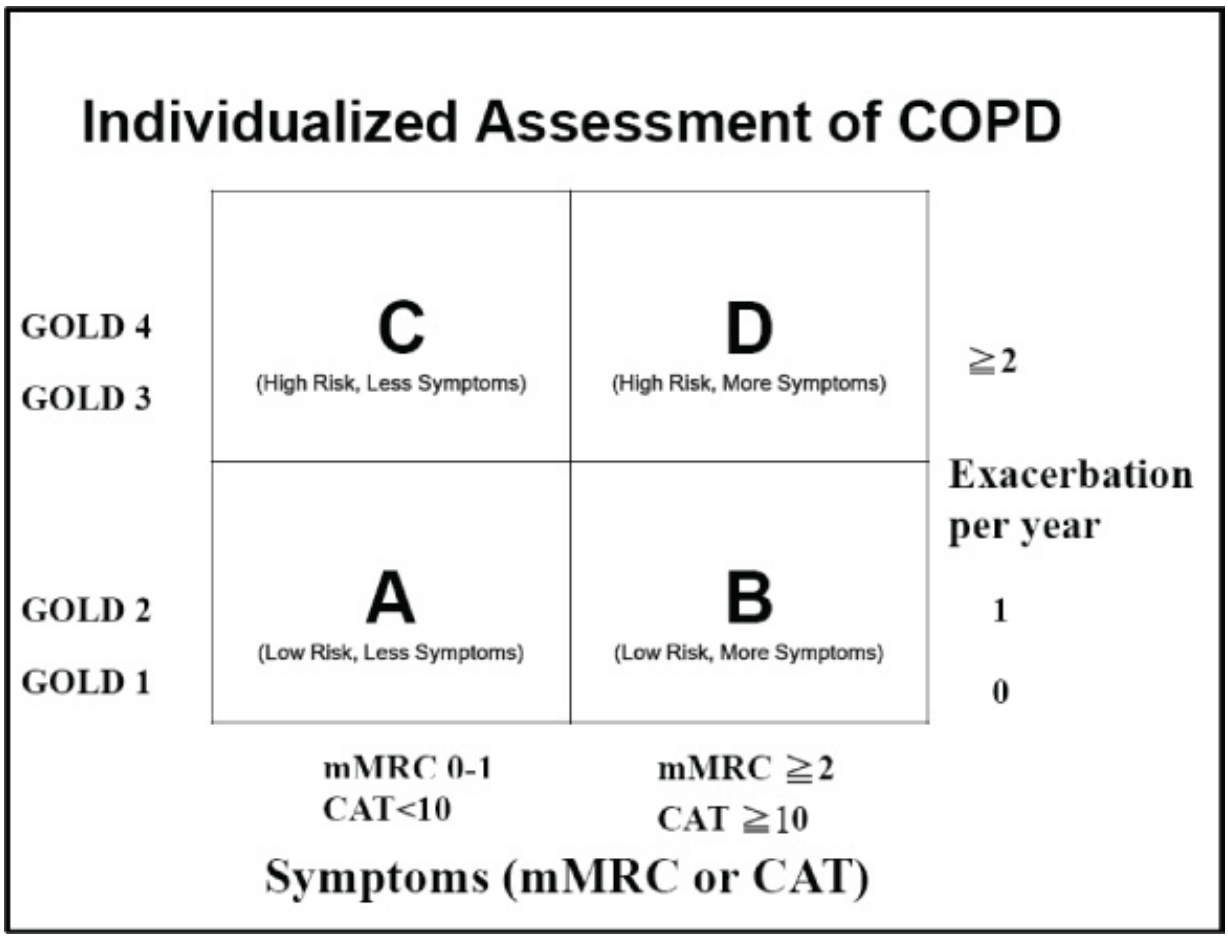
Autoimmune theory

- may explain delayed COPD

Physiology
- Exercise causes a decrease in inspiratory capacity
- TLC: Total lung capacity
- RV: Volume left after max exhalation
- EELV: End (normal) expiratory lung volume = TLC - RV
- FRC: Volume left after normal exhale
- IC: inspiratory volume available from normal exhalation
- With normal exercise: FRC doesn't change
- COPD
    - EELV at rest, FRC at rest is higher than normal
    - In COPD exercise, EELV progressively increases and IC decreases
        - Flow limitation causes air trapping and hyperinflation -> dyspnea

Clinical evaluation
- Most important to titrate medications to control symptoms
- FEV1 doesn't correlate well with symptoms
- Grade over stage
- Goals
    - Symptoms and functional limits
    - Severity of airflow limits
    - Risk of exacerbations (2 or more = frequent)
    - Co-morbidities

Grades
- Must have a ratio less than 70%
- 1 Mild FEV1 > 80%
- 2 Moderate 50-80%
- 3 Severe 30-50
- 4 Very severe <30%



Treatment

A (FEV1 > 50, low symptoms, < 2 exacerbations)
- short acting bronchodilators (can use long acting)

B (FEV1 > 50, increasing symptoms, < 2 exacerbations)
- long acting bronchodilators
- Pulmonary rehab

C (FEV1 < 50, low symptoms, 2 or more AE or 1 hospitalization)
- ICS + beta agonist or long active anticholingeric
- Pulmonary rehab

D (FEV1 <50, high symptoms, 2 or more AE or hospitalizations)
- Therapy of C or triple therapy
- Pulm rehab
- Consider roflumilast

Treatment Pearls
- Exercise for everybody. Rehab for all except group A
- No ICS mono therapy
- LABA+ICS option for C+D
- No routine abs per GOLD

- Consider roflumilast with FEV1 < 50%, chronic bronchitis, frequent AE

Acute Exacerbations (AEs)
- Requires change in therapy but may not require hospitalization
- Frequently can regain prior function
- Most important risk factor: Recent AE within last year
- All long acting medications decrease AEs
- Daily azithro 250mg decreases AE
    - Decreases hearing
    - Sudden cardiac death (tiny: 1 in 20k)
        - Check baseline Qtc and don't use if prolonged (no evidence)
        - Then repeat EKG at some point
- Treatment
    - oxygen, bronchodilators, systemic steroids
    - Abx even without evidence of pneumonia
    - NIV
        - mortality benefit!
        - best when used early
        - 25 (rr) - 35 (pH) - 45 (pCO_2)
    - Prednisolone 30-40mg daily 10-14 days traditional
        - oral preferred
        - 5 days vs 14 days probably no difference

Alpha-1-Antitrypsin Deficiency
- Single gene on chromosome 14
- Pi MM normal. Pi ZZ severe deficient. Anything that acts like a Z will causes deficiency
- Tobacco still plays a big role. Typically don't develop symptoms unless smoke.
- Pi MZ not at increased risk of COPD
- **Lower lobe emphysema**
- Screening for alpha should occur for all new diagnoses of A1AD (although rarely happens in clinical practice)
- Increased risk of cirrhosis and lung cancer
- treatment
    - stop smoking
    - COPD tx
    - Who gets augmentation?
        - A1AT levels < 11 umol/L (80 mg/dl)
        - and must have airflow obstruction (moderate or greater)
    - Consider transplant

Pulmonary Rehab
- Essential therapy in everybody except mild COPD (group A)
- Why?
    - increase 6mw
    - decrease Acute Exacerbations
    - increase QOL

Oxygen therapy

- PaO2 < 55 confirmed twice over a two week period
- PaO2 55-60 if pHTN, heart failure, polycythemia
- prolongs life in those with resting hypoxemia who use > 15hrs daily

Interventions

- lung volume reduction
  - NETT trial showed improve exercise and survival benefit in those with upper lobe disease and persistent low exercise tolerance
  - indications for
    - age < 75
    - upper lobe predominate emphysema
    - poor exercise tolerance despite rehab and pharmacy
    - FEV1 < 45% ; DLCO > 20%
    - RV > 150%, TLC > 100%, increased RV/TLC (severe air trapping)
    - No pHTN
- bronchoscopic valves
  - small increase FEV1
  - small increase 6 min walk
  - increases pneumonia, acute execrations, hemoptysis
- transplant
  - improves QOL but may not improve length of life
  - Who?
    - age < 70
    - smoke free for 6 months
    - poor functional status despite max medical therapy
    - Bode > 5-7
    - FEV1 < 20, DLCO < 20% or inhomogenous distribution (thus not lung volume reduction candidates)
    - Prednisone < 20mg/daily

Extra-pulmonary manifestations of COPD

- Don’t avoid using beta blockers (be careful with very high doses)