COPD Cheat Sheet

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COPD (Obstructive Disease)

COPD	Typical symptoms indicative of disease of large airways (**dyspnea, cough, and sputum** production) with evidence of **irreversible airflow obstruction** (FEV/FVC < 0.70).
Symptoms	Can be asymptomatic. Any combination of cough, sputum, and dyspnea (on exertion or at rest depending on severity). Depends on relative contributions of chronic bronchitis and emphysema. **Most patients have MIXED features of both**.
Risk Factors	Smoking (90% of cases), alpha1-antitrypsin deficiency (risk is even worse with smoking), environmental factors (second hand smoke), and chronic asthma.
Diagnosis	Get PFTs **(FEV/FVC < 0.70)**, CXR, A1A level in patients with a personal or family history of premature emphysema (<50yo), and ABG (shows chronic pCO2 retention and decreased PO2). **COPD leads to respiratory acidosis with metabolic alkalosis as compensation**.
CXR	Low sensitivity for diagnosing COPD. Only severe, advanced shows **hyperinflation, flattened diaphragm, enlarged retrosternal space, and diminished vascular markings**.

COPD (Obstructive Disease) (cont)

Screening	Measure peak expiratory flow rate using peak flow meter. If <350L/min, get PFTs to test for obstruction.
Pulmonary Function Tests	• Definitive diagnostic test. FEV1 (the amount of air that can be forced out of the lungs in 1s) is decreased. • **TLC, RV, and FRV are increased (air trapping)**. Although the TLC is increased, the air is not useful because it is RV (no gas exchange). Decreased vital capacity.
FEV1	• Best prognostic indicator for COPD • The best predictor of FEV1 is pack years of smoking • PaO2 falls when FEV1 is < 50% • When FEV1 is < 25% chronic retention of C02 occurs • Cor pulmonale occurs only after prolonged reduction in FEV1 (< 25%) with severe, chronic hypoxemia.

COPD (Obstructive Disease) (cont)

Signs	• Prolonged forced expiratory time. • **Timed full exhalation of VC >6sec.** • During auscultation, end-expiratory wheezes on forced expiration, decreased breath sounds, or inspiratory crackles. • Tachypnea, tachycardia, cyanosis, use of accessory respiratory muscles, hyperresonance on percussion, and signs of cor pulmonale.
Monitoring	**Serial FEV1 measurements** have high predictive value. Watch pulse oximetry and exercise tolerance too.
Complications	• acute exacerbations (most common causes are **infection, noncompliance and heart disease**), secondary polycythemia (Hct>55% in men or >47% in women) compensating to chronic hypoxemia, cor pulmonale, pulmonary HTN.

Emphysema

Define	• Permanent **enlargement of small airway spaces** distal to terminal bronchioles due to **destruction of alveolar walls**. • Decreased elastic recoil means **increased compliance, increased TLC, RV (air trapping)**, so the TC (or FVC) is decreased! • Air trapping leads to **dynamic hyperinflation** resulting in large auto-PEEP (intrinsic PEEP). • Thin patients with severe dyspnea, hyperinflated chests, decreased vascular markings, moderate oxygen desaturation.

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Emphysema (cont)

Pathophysiology of Emphysema	• Destruction of alveolar walls (impaires gas exchange) due to relative **excess in protease (elastase) activity or relative deficiency of antiprotenase** (alpha1-antitrypsin) activity in the lung. • **Elastase is released from PMNs and macrophages and digests human lung. This is inhibited by alpha1-antitrypsin.** • Tobacco smoke increases the number of activated PMNs and macrophages, inhibits A1A, and increases oxidative stress on lung by free radical production.
Pink Puffers	Tend to be thin due to increased energy expenditure during breathing. When sitting, they tend to lean forward, **barrel chest** (increased AP diameter). Tachypnea with **prolonged expiration through pursed lips**. Patient is distressed and uses accessory muscles (esp. strap muscles in the neck).

Emphysema (cont)

Centrilobular Emphysema	most common type, typically seen in **smokers**. Destruction is limited to respiratory bronchioles (proximal acini) with little changed distal acini. Predilection for **upper lung** zones.
Panlobular Emphysema	Panlobular Emphysema: seen in patients with **alpha1-antitrypsin deficiency**. Destruction involves both proximal and distal acini. Predilection for **lung bases**.

GOLD Criteria

• GOLD I =mild: FEV1 <80% predicted
• GOLD 2 =moderate: FEV1 <50--79% predicted
• GOLD 3 =severe: FEV1 3 <9% predicted
• GOLD 4=very severe: FEV1 < 30% predicted
Severity of symptoms
• A=fewer symptoms, low risk of exacerbations
• B = more symptoms, low risk
• C = fewer symptoms, high risk
• D =more symptoms, high risk

Treatment

• SABAs as needed in all patients.

• LABAs in moderate-to-very severe stages (reduce exacerbations and hospitalizations) when SABAs fail to control.

• **ICS is recommended in patients with GOLD 3-4 disease (FEV1< 50%)**. Reduce exacerbations, improved lung function, QoL. But increased risk for PNA. Should be combined with LABAs.

• Combination LABA + ICS is more e effective at reducing exacerbations associated with an increased risk of PNA.

• GOLD 3-4 patients may benefit from roflumilast, a phosphodiesterase-4 inhibitor for bronchitis, not emphysema.

Chronic Bronchitis

Define	• Chronic cough productive of sputum for at least 3 months per year for at least 2 consecutive years. • Due to **hypersecretion of mucus** and **structural changes** in the **large airway/tracheobronchial tree** • Bronchovascular markings, flattened diaphragm, and normal DLCO.
Pathophysiology of Chronic Bronchitis	Excess mucus production narrows the airways. Productive cough. **Inflammation and scarring in airways, enlargement in mucous glands, and smooth muscle hyperplasia** lead to obstruction.

Chronic Bronchitis (cont)

Blue Bloaters

- Predominantly **chronic bronchitis, overweight and cyanotic** (secondary to hypercapnia and hypoxemia).
- Chronic cough and sputum production.
- Signs of cor pulmonale may be present in severe or long-standing disease.
- Respiratory rate is normal or slightly increased, no apparent distress, no use of accessory muscles.

Acute Exacerbation

Define

- Persistent increase in **dyspnea not relieved with bronchodilators**. Increased sputum production and cough are common.
- Can lead to acute respiratory failure requiring hospitalization and possibly mechanical ventilation. •
- **Pulmonary infection** is one of the main precipitants.
- CXR shows **hyperinflation**. ABG shows **hypoxia, hypercarbia**, and **respiratory acidosis**.

Treatment of Acute Exacerbation

- bronchodilators (beta agonist and/or anticholinergics), **systemic corticosteroids (methylprednisolone)** when hospitalized, **antibiotics (azithromycin or levofloxacin)**, supplemental oxygen, noninvasive positive pressure ventilation (BIPAP or CPAP), and intubation if necessary (only severe CO2 retention). **NO INHALED CORTICOSTEROIDS**.

Oxygen Therapy

- Improves survival and quality of life in patients. Some need continuous oxygen, while others only require it during exertion or sleep. **Get ABG to determine need**.
- Criteria: **PaO2 55, SaO2<88%, or PaO2 55-59** plus **polycythemia or cor pulmonale**.
- Long standing hypoxemia may lead to **pulmonary HTN and cor pulmonale**
- Continuous oxygen **therapy for >18 hr/day** has been shown to reduce mortality
- Hypoxemia is due to **V/Q mismatching** therefore responsive to low flow oxygen (2-3L/min). If **not responsive to oxygen, consider shunt**.

Smoking Cessation

- **Most important intervention**.
- Disease progression is accelerated by continued smoking and can be greatly slowed by its cessation. Around age 35, FEV1 decreases approximate 25-30mL/yr. In smokers, the decline is faster (3-4x). If a smoker quits, the rate of decline slows to normal.
- Smoking does not completely reverse. Respiratory symptoms improve within 1 year of quitting.
- **Smoking cessation and oxygen therapy** are the only interventions shown to **reduce mortality**.

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