CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Airflow limitation that is not fully reversible ....

PATHOPHYSIOLOGY:

Two main pathologic entities:

EMPHYSEMA:
destruction of alveoli, decreased lung elasticity & closure of small airways due to loss of radial support.

- Progressively destroyed over time
- PROTEASE-ANTIPROTEASE IMBALANCE (exacerbated by smoking)

CHRONIC BRONCHITIS.
"presence of cough & sputum for at least 3 months in each of two consecutive years"

- Combination of airway obstruction and obliteration of the pulmonary vascular bed results in FAILURE OF GAS EXCHANGE.
- Effect of circulating inflammatory mediators - weight loss, wasting, depression, metabolic derangements

STAGING:

Table 73-1 Global Initiative for Chronic Obstructive Lung Disease (Gold) Classification of Chronic Obstructive Pulmonary Disease by Severity

Stage	Characteristics: For All Stages FEV_1/Forced Vital Capacity <0.7
I. Mild COPD	FEV_1 ≥80% predicted
	With or without chronic symptoms (cough, sputum production)
II. Moderate COPD	FEV_1 between 50%–79% predicted
III. Severe COPD	FEV_1 between 30%–49% predicted
	With or without chronic symptoms (cough, sputum production)
IV. Very severe COPD	FEV_1 <30% predicted
	or
	<50% predicted *plus* respiratory failure
	or
	<50% predicted *plus* clinical signs of right heart failure

ACUTE EXACERBATIONS:

- Not necessarily associated with major reductions in peak flow (c/w asthma)
- Characterised by CHANGE FROM BASELINE
- VIRAL INFECTIONS MOST COMMONLY IMPLICATED
- **NO CAUSE IDENTIFIED IN ONE THIRD CASES!**
- CONSIDER COMORBID CAUSES OF DETERIORATION

BOX 72-1 **CAUSES OF ACUTE DECOMPENSATION IN THE PATIENT WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE**

I. Acute exacerbations
   A. Infectious
      1. Viral
         Rhinovirus, respiratory syncytial virus, Coronavirus, influenza virus
      2. Bacterial
         Haemophilus influenza, Streptoccocus pneumoniae, Moraxella (Branhamella) catarrhalis, Pseudomonas aeruginosa
      3. Atypical bacteria
         Chlamydia pneumoniae, Legionella
   B. Air pollution
      1. NO_2
      2. Ozone
      3. Particulate particles
II. Other critical events
      1. Pneumothorax
      2. Pulmonary embolism
      3. Lobar atelectasis
      4. Congestive heart failure
      5. Pneumonia
      6. Pulmonary compression (e.g., obesity, ascites, gastric distention, pleural effusion)
      7. Trauma (e.g., rib fractures, pulmonary contusion)
      8. Neuromuscular and metabolic disorders
      9. Unrelated treatable chronic pulmonary disease (bronchiectasis, tuberculosis, sarcoidosis)
      10. Noncompliance with prescribed treatment regimens
      11. Iatrogenic
         a. inadequate therapy
         b. inappropriate therapy (e.g., deleterious drugs)

CLINICAL FEATURES:

- Progression is slow & insidious with increasingly frequent and debilitating exacerbations.
- *IF BRONCHITIS PREDOMINATES:*
  - Findings of respiratory failure/cor pulmonale (peripheral oedema)
  - Polycythaemia --> PLETHORIC APPEARANCE
  - If ventilatory failure present --> somnolence/asterixis
- *IF EMPHYSEMA PREDOMINATES:*
  - AUTO-PEEP --> purse lipped ventilation to increase intraluminal bronchial pressure, hunched forward, chronic overinflation
  - Hyper-resonance, diminished breath sounds, faint end-expiratory wheeze

DIAGNOSTIC STRATEGIES:

- CHANGE in pulse oximetry from BASELINE

CXR to determine if there is an ACUTE, TREATABLE cause for acute deterioration:

- Pneumothorax
- Consolidation
- Atelectasis
- Also, indicates alternate diagnoses eg. CHF, effusion, tumour.

ABG:

- Should NOT be used to determine need for intubation/NIPPV as this is guided by fatigue, comorbidities, response to treatment

PFT:

- Add little in decision making

SPUTUM --> no value

ECG:

- P-pulmonale (peaked P waves in II, III, aVF ~ 2.5mm)
- RVH suggests cor pulmonale, but its absence does not exclude diagnosis
- Atrial arrhythmias very common (AF, MFAT)

BLOOD TESTS:

- FBC: polycythaemia, ↑WCC (can be due to hyperadrenergic state, steroids, infection)
- Theophylline level (outdated)
- BNP --> good negative predictive value, poor specificity for CHF.

DIFFERENTIAL DIAGNOSES:

SUDDEN DETERIORATION:

- Acute pneumothorax
- PE (COPD patients are sedentary, and those with cor pulmonale have ↑viscosity, high peripheral venous pressure with venous stasis
- LOBAR ATELECTASIS
    - A result of sputum plugging.
- AMI

BROADER DIFFERENTIALS:

- CHF
- Asthma
- Pneumonia:
    - Devastating complication
    - Symptoms more non-specific, radiographic findings often less dramatic
- ARDS
- Metabolic acidosis/shock - ↑RR/ventilatory failure

MANAGEMENT:

- Only modalities that alter progression;
    - SMOKING CESSATION
    - OXYGEN THERAPY (severe disease)
    - VACCINES (influenza/pneumococcus)

VENTILATION CHOICES:

Table 72-2 Suggested Selection and Exclusion Criteria for the Use of NIVS

SELECTION CRITERIA (ONE OR MORE MAY BE PRESENT)	EXCLUSION CRITERIA (ANY MAY BE PRESENT)
Moderate to severe dyspnea with use of accessory muscles and paradoxical abdominal motion	Respiratory arrest
Respiratory rate > 25 breaths/minute	Cardiovascular instability
Moderate to severe acidosis (pH < 7.35) and hypercapnia ($Paco_2$ > 45 mm Hg)	Uncooperative patient (agitated or severely somnolent)
	Upper airway obstruction
	High aspiration risk
	Recent facial or gastroesophageal surgery
	Craniofacial trauma, fixed nasopharyngeal abnormalities
	Nonfitting mask

↑CO2 (≥ 45)
↓pH (<7.35)
RR ≥ 25
↑Work of breathing

HIGHLY EFFECTIVE AT AVOIDING INTUBATION, ↑pH, ↓CO2, reducing dyspnoea

Table 72-3 Proposed Indications for Mechanical Ventilation

Respiratory arrest
Worsening level of consciousness despite maximal therapy*
Cardiovascular instability (shock, heart failure)*
NIPPV failure or exclusion criteria (see Table 72-2)
Severe dyspnea with use of accessory muscles and paradoxical abdominal motion*
Severe tachypnea*
Life-threatening hypoxia
Severe acidosis and hypercapnea*
Other complications (metabolic abnormalities, sepsis, pneumonia, pulmonary embolism, barotraumas, massive pleural effusion)*

CLINICAL ACUMEN RATHER THAN ABG
Initial settings:
- TV 6-8mL/kg, RR 8-10

PERMISSIVE HYPERCAPNIA
(helps avoid barotrauma from iPEEP)
→ pH 7.15 -7.20.

Normalise over HOURS

OXYGEN ADMINISTRATION:
- Risks of hypoxaemia need to be weighted against the risk of reducing ventilation
- ***Titrate to maintain SaO2 ≥ 90%*** with Venturi mask
- Those breathing inappropriately SLOWLY are at highest risk of apnoea with oxygen therapy

DRUG THERAPY:

BRONCHODILATORS:
- First line agents
  (even though bronchospasm not inciting event in acute exacerbations)
  - β-agonists and anticholinergics in concert
- MDI if able, nebulised otherwise.
- Long-acting agents in chronic stable COPD (TIOTROPIUM, SALMETEROL), no use in ED setting

CORTICOSTEROIDS:
- Evidence points to modest decrease in relapse rate of acute exacerbations and improvement in dyspnoea
- Prednisone 30-50mg daily (dose recommendations vary widely), OR Hydrocortisone 100mg q6h and convert to oral as soon as possible
- Duration 7-14 days (no benefit with longer course)
- SIDE EFFECTS:
  - Myopathy
  - ↑BSLs
  - Immune suppression

ANTIBIOTICS:

- Recommended in patients with:
    - ↑ sputum purulence with either:
        - ↑ sputum volume
        - ↑ dyspnoea
- IV antibiotics generally required only if:
    - Impaired mental state
    - Unable to swallow safely
    - CXR confirming pneumonia --> follow CAP guidelines
- Use AMOXICILLIN 500mg q8h or DOXYCYCLINE 100mg bd for five days
- Use of newer fluoroquinolones controversial (data suggest improvement but limited by their use in less severely effected patients
  (use only if documented resistance).

DISPOSITION.

Significant deterioration from baseline is a general guideline for admission.

Consider response to ED treatment, comorbidities, ability to function at home.

Table 73-6 Indications for Hospital Admission for Acute Exacerbations of Chronic Obstructive Pulmonary Disease
Marked increase in intensity of symptoms, such as sudden development of resting dyspnea
Background of severe chronic obstructive pulmonary disease
Onset of new physical signs (e.g., cyanosis, peripheral edema)
Failure of exacerbation to respond to initial medical management
Significant comorbidities
Newly occurring arrhythmias
Diagnostic uncertainty
Older age
Insufficient home support