COPD

general
- the terms COPD or COAD are applied to patients with chronic bronchitis and / or emphysema

aetiology
- environmental factors:
(i) tobacco smoke
(ii) air pollution
- host factors:
(i) balance between circulating proteases and antiproteases (eg alpha 1 antitrypsin deficiency)
(ii) antioxidants (vitamins A, C & E)

differential diagnosis
1. chronic asthma
2. bronchiotis obliterans
3. bronchiectasis
4. CCF

causes of exacerbation

Infective (including aspiration)
Left ventricular failure (systolic and diastolic failure)
Sputum retention (post operative, traumatic)
Pulmonary embolism
Pneumothoraces and bullae
Uncontrolled oxygen
Sedation
Medication – non-compliance or side-effects
Nutritional (K, PO_4, Mg deficiency, CHO excess)
Sleep apnoea

- most common bacterial isolates are Strep pneumoniae and H. influenzae; Strep viridans, Moraxella catarrhalis, Mycoplasma pneumoniae & Pseudomonas may be found
- viruses can be isolated in 20-30% of exacerbations and include rhinovirus, influenza & parainfluenza, corona viruses & occasionally adenovirus and respiratory syncytial virus

clinical features

Normocapnic ($PaCO_2$ 35–45 mmHg)	Hypercapnic ($PaCO_2$ >45 mmHg)
(emphysema > chronic bronchitis)	(chronic bronchitis > emphysema)
thin	obese
pursed lip breathing	CNS depression consider the role of oxygen therapy
accessory muscle use	alcohol, sedatives, analgesics
hyperinflated	sleep related hypoventilation
right heart failure late	right heart failure early

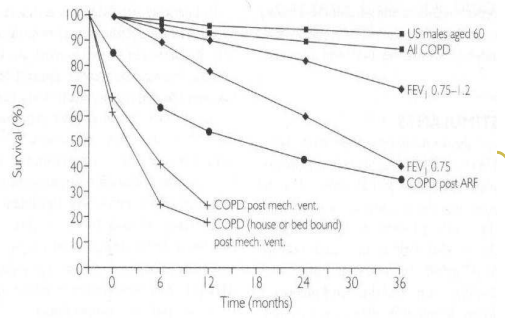
Mild disease (eg FEV1 50-70% predicted)
- an expiratory wheeze on forced expiration and mild exertional dyspnoea may be the only symptoms

Moderately severe COPD *(eg FEV1 30-50% predicted)
- modest to severe exertional dyspnoea is associated with clinical signs of hyperinflation & increased work of breathing

Severe COPD (eg FEV1 <30% predicted normal)
- marked accessory muscle use is associated with tachypnoea at rest, pursed lip breathing, hypoxaemia & signs of pulmonary hypertension and cor pulmonale
- in unstable COPD, there is marked tachypnoea at rest, hypoxaemia & tachycardia and signs of hypercarbia

investigations
1. spirometry
- allows confirmation of clinical diagnosis & determining severity of disease
2. flow-volume loops
- demonstrate reduced expiratory flow rates at various lung volumes & show characteristic 'concave' expiratory flow pattern
3. CXR:
- will commonly show hyperinflated lung fields, flattened diaphragms & a paucity of lung markings; pulmonary hypertension is manifest by enlarged proximal & attenuated distal lung markings
4. CT & HRCT:
- HRCT can demonstrate characteristic appearances; HRCT scans are less sensitive than standard CT scans for detecting pulmonary lesions such as neoplasms
5. ECG:
- commonly normal but may show features of right atrial or RV hypertrophy & RV strain, including P pulmonale, right axis deviation, RBBB & ST depression or inversion in V1-V3

prognosis



- when acute deterioration occurs, most precipitating factors are reversible justifying an aggressive approach to management

therapy

Uncontrolled oxygen administration:
- may precipitate acute hypercapnia in patients with more severe COPD due to:
(i) shunting blood to low VQ units and increasing dead space
(ii) loss of hypoxic drive
(iii) dissociation of CO2 from Hb molecule (Haldane effect)
(iv) anxiolysis and reduction in tachypnoea

Bronchodilators:
- bronchodilators are routinely given in all exacerbations of COPD because a small reversible component of airflow obstruction is common & bronchodilators improve mucociliary clearance
(i) anticholinergic agents:
- ipratropium bromide 500mcg neb initially 2 hourly & then 4-6 hourly
- long acting anticholinergics (eg tiotropium) offer potential of once daily dosing
- have fewer side effects and no tachyphylaxis than beta agonist
(ii) nebulised beta agonists:
- combination with ipratropium has been shown to be more effective than either agent alone
(iii) aminophylline:
- a weak bronchodilator in COPD
- some studies have shown no benefit and significant side effects while others have shown a small benefit in stable COPD

Steroids:
- in acute exacerbations of COPD, short-term steroids have been shown to improve airflow obstruction including in those patients requiring mechanical ventilation for COPD; they should be avoided if the exacerbation is clearlydue to pneumonia without bronchospasm

Antibiotics:
- antibiotics have an accepted role in the treatment of infection-induced exacerbations of COPD

Secretion clearance techniques:
(i) chest physio
(ii) nebulised mucolytic agents
(iii) oropharyngeal / nasopharyngeal suctioning
(iv) bronchoscopy (indicated for com

Non-invasive ventilation:
- there have been several randomised trials of NIPPV in patients with acute hypercapnic respiraotry failure which have demonstrated improved respiratory physiology, reduced mortality, reduced need for mechanical ventilation & reducedlength of hospital stay
- indications in a deterioration of COPD are:
(i) respiratory rate of >28 breaths / min
(ii) acute dyspnoea
(iii) PaCO2 >45mmHg with a pH <7.35 despite optimal medical treatment

Whether to institute invasive mechanical ventilation:
- invasive mechanical ventilation may be withheld in end stage lung disease when low survival, poor quality of life or ventilator dependence is likely; if end stage lung disease is suspected but there is insufficient information then a trial of aggressive therapy including invasive ventilation should be undertaken and subsequently withdrawn if unsuccessful
- indications for invasive mechanical support include:
(i) the clinical appearance of fatigue & impending respiratory collapse despite non-invasive ventilatory support
(ii) deteriorating conscious state due to fatigue, hypercapnia or both
(iii) hypoxia refractory to high levels of inspired oxygen
(iv) deterioration due to failure of secretion clearance
(v) respiratory arrest

Mechanical ventilation technique:
- goals of mechanical ventilation are to support ventilation while reversible components improve, to allow respiratory muscle to rest and recover whilst preventing wasting from total inactivity and to minimise dynamic hyperinflation
- excessive dynamic hyperinflation must be avoided by using a low minute ventilation & allowing time for expiration. Dynamic hyperinflation can be assessed by:
(i) clinical judgement
(ii) visualisation of the expiratory flow time curve
(iii) measurement of plateau pressure (end inspiratory pause of 0.5s) - if >25cmH20 then there is likely to be dynamic hyperinflation if chest wall compliance is low
(iv) measurement of intrinsic PEEP (prolonged expiratory pause) - provides a direct assessment of dynamic hyperinflation. In severe airflow limitation it may be necessary to accept low levels of PEEPi of 8-10cmH2O; however, when levels rise above this further prolongation of expiratory time must by considered