Clinical Practice Guidelines:
Respiratory/Chronic obstructive pulmonary disease

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<th>Date</th>
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<td>Purpose</td>
<td>To ensure consistent management of patients with Chronic obstruction pulmonary disease.</td>
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<td>Scope</td>
<td>Applies to all QAS clinical staff.</td>
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Chronic obstructive pulmonary disease (COPD) describes a number of pulmonary diseases that are characterised by chronic airflow limitation that is progressive and not fully reversible.

COPD includes:

Chronic bronchitis – is defined as daily sputum production for at least three months over two or more consecutive years.[1]

Classical presentation:
- Cyanosed
- Often overweight
- Oedematous
- Chronic cough
- Chronic sputum production
- Cor pulmonale (late sign)

Emphysema – characterised by dilatation and destruction of alveoli. The loss of elasticity and enlargement of these air spaces leads to hyperinflation of the lungs and increased work of breathing.[1]

Classical presentation:
- Thin
- Barrel chest
- Dyspnoea
- Tachypnoea
- Pursed lip breathing
- Intercostal or suprasternal recession
- Tripod posture

Both presentations can share symptoms of dyspnoea, cough and sputum production, with chest tightness, airway irritability and wheezes also common. The natural course of COPD is characterised by episodes of acute exacerbation where symptoms escalate.[2]

NOTE: COPD is a spectrum of disease and many patients have features of both chronic bronchitis and emphysema.

Clinical features

An ‘acute exacerbation’ of COPD usually follows infection, although in some cases no clear precipitant is apparent. Clinical features of an acute exacerbation include:

- History:
  - Upper Respiratory Tract Infection (URTI) symptoms
  - Increased dyspnoea, difficulty in speaking, reduced exercise tolerance, fatigue
  - Increased sputum volume and purulence
  - Chest tightness and wheeze
  - Increased cough
  - Anxiety
  - Increased medication use with minimal or no effect
**Risk assessment**

COPD exacerbation may mask other pathology, making diagnosis and management difficult. The following conditions are common differential diagnoses:[2]

- Cardiogenic APO/Congestive Cardiac Failure (CCF)/AMI
- Asthma
- Pneumonia/pleural effusion
- Upper airway obstruction
- Pulmonary embolism
- Pneumothorax
- Lung cancer

**Additional information**

- While COPD is characterised by irreversible airflow limitation, bronchodilators may act to improve clinical symptoms by the direct effect on bronchial smooth muscle and bronchomotor tone.

- **Hypoxic drive**
  - The aim of oxygen therapy is to prevent life-threatening hypoxia; attempts should be made to titrate supplemental oxygen to achieve SpO₂ readings between 88% and 92%.[2] Some COPD patients rely on hypoxia to drive respiration, rather than hypercapnia, due to chronically raised CO₂ levels. Thus uncontrolled oxygen therapy can result in suppression of respiratory drive, carbon dioxide narcosis and ultimately respiratory arrest.[3] The titration of oxygen to targets reduces mortality compared with high flow oxygen treatment secondary to reductions within hypercapnia and resultant acidosis.

  If the patient is hypoxic high dosages of oxygen therapy are indicated, with a view to de-escalate oxygen concentration where appropriate; the lowest dosage of O₂ possible should be used as soon as possible.

- Examination:
  - Respiratory distress
  - Intercostal or suprasternal recession
  - Accessory muscle use
  - Fever/sepsis
  - Cyanosis
  - Wheeze, crackle, reduced air entry on auscultation
  - Tachycardia
Consider:
- Oxygen
- Salbutamol Neb
- Ipratropium bromide Neb
- Salbutamol IV
- Hydrocortisone
- Adrenaline (epinephrine)
- IPPV

Severe respiratory distress?

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Consider:
- Maintain SpO₂ at 88 – 92%
- Salbutamol Neb
- Ipratropium bromide Neb
- Hydrocortisone

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Transport to hospital
Pre-notify as appropriate

Note: Officers are only to perform procedures for which they have received specific training and authorisation by the QAS.