



# Clinical Practice Guidelines: Respiratory/Chronic obstructive pulmonary disease

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<b>Date</b>	February, 2021
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<b>Scope</b>	Applies to Queensland Ambulance Service (QAS) clinical staff.
<b>Health care setting</b>	Pre-hospital assessment and treatment.
<b>Population</b>	Applies to all ages unless stated otherwise.
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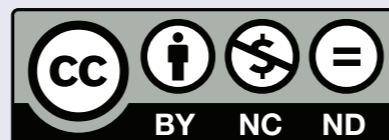
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# Chronic obstructive pulmonary disease

February, 2021

**Chronic obstructive pulmonary disease (COPD)** represents a significant burden of disease, both in Australia and worldwide. It is estimated to affect 9–14% of Australians over 40 years of age.

COPD is characterised by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.<sup>[1]</sup>

Diagnosis is based on:

- Symptoms of exertional breathlessness, cough and sputum
- A history of smoking (most important risk factor), or exposure to other noxious agents
- Spirometry – FEV<sub>1</sub>/FVC < 0.7 post-bronchodilator

The disease processes associated with COPD are:

- Chronic bronchitis – daily sputum production for at least three months, of two or more consecutive years
- Emphysema – alveolar dilation and destruction
- Chronic asthma

There is significant overlap in the disease processes associated with COPD. Determining distinct phenotypes (chronic bronchitis vs. emphysema) is no longer included in contemporary COPD definitions.

Patients with COPD experience a complex interplay between pathophysiology, pharmacology, risk factors and social factors. They are at significant risk of numerous comorbidities including:

- Cardiovascular disease (Ischemic heart disease, heart failure, pulmonary hypertension, stroke, cardiac dysrhythmias, pulmonary emboli)
- Aspiration, dysphagia and gastro-oesophageal reflux disease (GORD)
- Osteoporosis, fractures and falls
- Depression, sleep disorders and cognitive disorders
- Diabetes mellitus and metabolic disorders
- Cancer

## Clinical features



- Breathlessness on exertion
- Cough and sputum production
- Chest tightness
- Wheeze
- Both malnutrition and obesity are common
- Polypharmacy, often with limited effect
- Older age group

### Signs of advanced disease:

- Dynamic chest hyperinflation
- Soft breath sounds, prolonged expiratory phase
- Hypoxia and hypercapnia
- Inactivity, poor appetite and weight loss
- Many of these patients would have a history of long term O<sub>2</sub> therapy

### Acute Exacerbation of COPD (AECOPD):

- Change in baseline dyspnoea, cough or sputum – typically due to a respiratory infection.
- Difficulty in speaking, anxiety, tachypnoea, tachycardia, cyanosis
- Accessory muscle use, tracheal tug, intercostal recessions, paradoxical abdominal wall motion



## Risk assessment

Differential diagnoses and concomitant illness are common.

Consider:

- Heart failure and cardiogenic APO
- IHD and AMI
- Pulmonary embolism (found in 16% of COPD exacerbations)
- Pneumothorax (abrupt onset)
- Pleural effusion, pneumonia and lobar atelectasis
- Upper airway obstruction
- Anaphylaxis

**Note:** Beware an increased risk of adverse events associated with sedative and opiate drug administrations.



## 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.
- Hypercapnic respiratory failure secondary to excessive oxygen administration is an important concern in the management of AECOPD. All COPD patients with moderate to severe exacerbation are considered at risk. Proposed mechanisms include:
  - worsening of VQ mismatch (most important)
  - inhibition of ventilatory drive
  - the Haldane effect
  - absorption atelectasis
  - higher viscosity of O<sub>2</sub>

Hypercapnia and the resultant acidosis may have depressant effects on the CNS and CVS. COPD patients who receive excessive O<sub>2</sub> have been shown to have higher rates of mortality and adverse outcomes. The lowest dose of O<sub>2</sub> required to achieve a SpO<sub>2</sub> of 88–92% must be used in AECOPD.<sup>[2,3]</sup>

- AECOPD patients have little physical reserve and may deteriorate with minimal exertion. Clinicians must exercise extreme caution with their movement and have a low threshold for using aid devices for moving patients.
- Patient rehabilitation and education has been shown to improve outcomes in COPD.<sup>[1]</sup> In appropriate cases, patients should be encouraged to discuss the optimisation of their care with a physician. The Lung Foundation Australia also provides support ([www.lungfoundation.com.au](http://www.lungfoundation.com.au) or free-call [REDACTED]).

CPG: Clinician safety  
CPG: Standard cares

Severe respiratory distress?

N

**Consider:**

- Oxygen (maintain SpO<sub>2</sub> at 88–92%)
- Salbutamol
- Ipratropium bromide
- Hydrocortisone

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- Minimise patient exertion (e.g. walking)
- Patient reassurance

**Consider:**

- Oxygen (maintain SpO<sub>2</sub> at 88–92%)
- Salbutamol
- Ipratropium bromide
- Hydrocortisone
- Adrenaline (epinephrine)
- IPPV (+/- PEEP)

Transport to hospital  
Pre-notify as appropriate

*Note: Clinicians must only perform procedures for which they have received specific training and authorisation by the QAS.*