








Thoracic Society of Australia and New Zealand Position Statement on Acute Oxygen Use in Adults: ‘Swimming between the flags’

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Abstract

Oxygen is a life-saving therapy but, when given inappropriately, may also be hazardous. Therefore, in the acute medical setting, oxygen should only be given as treatment for hypoxaemia and requires appropriate prescription, monitoring and review. This update to the Thoracic Society of Australia and New Zealand (TSANZ) guidance on acute oxygen therapy is a brief and practical resource for all healthcare workers involved with administering oxygen therapy to adults in the acute medical setting. It does not apply to intubated or paediatric patients. Recommendations are made in the following six clinical areas: assessment of hypoxaemia (including use of arterial blood gases); prescription of oxygen; peripheral oxygen saturation targets; delivery, including non-invasive ventilation and humidified high-flow nasal cannulae; the significance of high oxygen requirements; and acute hypercapnic respiratory failure. There are three sections which provide (1) a brief summary, (2) recommendations in detail with practice points and (3) a detailed explanation of the reasoning and evidence behind the recommendations. It is anticipated that these recommendations will be disseminated widely in structured programmes across Australia and New Zealand.

KEYWORDS

acute oxygen therapy, oxygen prescription, position statement, target oxygen saturations, titrated oxygen

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A. INTRODUCTION

- Purpose:** The purpose of the Thoracic Society of Australia and New Zealand (TSANZ) Position Statement is to provide simple, practical evidence-based recommendations for the acute use of oxygen in adults in clinical practice. The intended users are all health professionals responsible for the administration, and/or monitoring of oxygen therapy in the acute management of patients in clinical settings (excluding intubated, mechanically ventilated patients), and those responsible for the training of such health professionals. The Position Statement represents a clinical practice and educational initiative of the TSANZ, which was established to improve the knowledge and understanding of lung disease, to prevent respiratory illness through research and health promotion and to improve health care for people with respiratory disorders (<http://www.thoracic.org.au/>).
- Organization of this Position Statement:** The Position Statement contains three sections. Section A contains the background to this document and provides the key recommendations in six headings in Table 1. The concepts behind each of the key recommendations are listed in Table 2. Section B covers those recommendations in detail, along with 'Practice points', organized by the same six headings. Finally, Section C summarizes the key evidence and reasoning behind those recommendations, organized similarly under those headings.
- Literature review:** Targeted literature reviews were conducted by working groups of this committee, to find relevant literature since publication of the original TSANZ Guideline document in 2015. Given the absence of a formal systematic review, this document is designated as a Position Statement. As with the 2015 TSANZ Guideline document, an extensive list of references is not provided, but rather reference is made to key reviews, studies and guidelines where appropriate. The readers are referred to the 2017 British Thoracic Society (BTS) guidelines¹ for a comprehensive review of the acute oxygen therapy literature.
- Grading:** Grades of recommendation are presented below and are related to the National Health and Medical Research Council grading system (Table 3), based on evidence base, consistency of evidence, clinical impact, generalizability and applicability.²
- Revision Group:** The Position Statement Revision Group was chaired by Gregory King, who initiated the formation of the group via the TSANZ. This group included respiratory physicians, respiratory nurses and intensive care physicians, and was formed following a general call for members by the TSANZ.
- Main updates compared with the 2015 document:** The evidence supporting the recommended SpO₂ (arterial oxygen saturation measured by pulse oximeter) targets has been strengthened by recent publications, and the use of oxygen prescription has also been further supported by local experience with the use of various modes of prescription. There has been more recent

TABLE 1 Key recommendations^a

- Assess oxygenation:
 - Pulse oximetry should be routinely recorded along with other vital signs (Grade C)
 - Measurement of SpO₂ and venous blood gases has significant limitations; the gold standard is ABG which should be measured when clinically appropriate (Grade C)
- Oxygen is a drug and thus requires prescription:
 - Oxygen prescription requires documentation of flow rate and delivery device, SpO₂ targets with specified upper and lower ranges and criteria that define deterioration and improvement (Grade D)
- Recommended SpO₂ targets:
 - 88%–92% is the recommended target range (i.e., administer O₂ when SpO₂ < 88%) in chronic respiratory diseases, where there is potential for hypercapnia (Grade B)
 - Otherwise, 92%–96% is the recommended target range in other clinical situations (i.e., administer O₂ when SpO₂ < 92%)^b (Grade B)
- Delivery:
 - Nasal cannulae are preferred in most situations due to ease and practicality
 - In COPD and other conditions associated with chronic respiratory failure, bronchodilators should be delivered by metered dose inhaler ± spacer (or if a nebulizer is necessary, then it should be air driven with oxygen supplementation continued using nasal cannulae) (Grade B)
 - hNHF-O₂ may be used in patients with acute, severe, hypoxaemic respiratory failure (Grade B)
- High FiO₂ to achieve target SpO₂ indicates serious illness:
 - An Early Warning Score (EWS) system should be used to detect deterioration and should combine FiO₂ and SpO₂ parameters as risk markers (Grade C)
 - Senior clinician^c review should occur if FiO₂ ≥ 0.40 or flow rate ≥ 6 L/min via simple face mask is required to maintain target SpO₂ (Grade D)
 - ICU review should occur if FiO₂ ≥ 0.50 or flow rate ≥ 8 L/min via simple face mask is required to achieve target SpO₂ (Grade D)
- Acute respiratory acidosis:
 - In patients with acute respiratory acidosis (arterial pH of <7.35 and PaCO₂ of >45 mm Hg), NIV or invasive ventilation should be considered. NIV is not usually indicated, however, in hypoxaemic respiratory failure (without acidosis) (Grade B)
 - Patients managed with NIV require close monitoring and regular assessment of improvement or deterioration, in which case intubation may be required (Grade C)

Abbreviations: ABG, arterial blood gases; COPD, chronic obstructive pulmonary disease; FiO₂, fraction of inspired oxygen; hNHF-O₂, humidified nasal high-flow oxygen; ICU, intensive care unit; NIV, non-invasive ventilation; PaCO₂, arterial partial pressure of carbon dioxide; SpO₂, arterial oxygen saturation measured by pulse oximeter.

^aThese recommendations apply to all patients receiving supplemental oxygen in the acute medical setting, but not to those receiving invasive mechanical ventilation.

^bExcept in sickle cell crisis, cluster headache, carbon monoxide and paraquat poisoning and previous bleomycin exposure.

^cClinician refers to medical, nursing, physiotherapy and other healthcare professionals responsible for patient care.

evidence, since the 2015 document, of the efficacy of humidified nasal high-flow oxygen (hNHF-O₂) therapy in hypoxaemic respiratory failure and so a substantial section now deals with its use to deliver oxygen. There is a large addition around non-invasive ventilation (NIV), given its greatly increased use in ventilatory failure in Australia and New Zealand. Recent evidence on the effectiveness of early warning systems that incorporate both oxygen flow rate and SpO₂ is included. Finally, the

TABLE 2 Concepts behind each of the key recommendations

- (1) Assess oxygenation:
- Hypoxaemia is an independent marker of risk and of poor outcomes, due to the severity of the underlying disease(s) that caused hypoxaemia
 - There are clinically significant limitations to the interpretation of pulse oximetry such that ABG should be taken in the appropriate clinical situations
- (2) Oxygen is a drug and thus requires prescription:
- Oxygen is a drug and should be prescribed for the relief of hypoxaemia, and not breathlessness in the absence of hypoxaemia
 - Oxygen can be initiated or the flow rate increased acutely when needed, but as applies to any emergency situation, this requires urgent medical review
- (3) Recommended SpO₂ targets:
- There are risks associated with both hypoxaemia and hyperoxaemia, which underlie the importance of prescribing oxygen, only if required, to a specific target oxygen saturation range ('swimming between the flags' concept)
 - The lower limits of safety for PaO₂ and SpO₂ are variable and depend on the clinical scenario. However, a PaO₂ of at least 50 mm Hg and SpO₂ of at least 80% is likely to prevent immediate death from hypoxaemia. There is now strong evidence supporting both lower and upper SpO₂ target ranges in acute respiratory and other medical conditions
 - Supplemental oxygen should not be used to achieve SpO₂ above the recommended target range, with the intent of protecting against future hypoxaemia in the setting of a clinical deterioration. This may mask any subsequent deterioration in SpO₂ delaying recognition of deterioration, senior clinician review and escalation of appropriate therapy
- (4) Delivery:
- Nasal cannulae are the preferred method of oxygen delivery for most patients because they can remain *in situ* during delivery of bronchodilators by inhaler and spacer, eating and other activities
 - hNHF-O₂ delivers high FiO₂ (up to 1.0) at high-flow rates (up to 70 L/min). It is therefore an effective oxygen delivery system for acute, severe hypoxaemic respiratory failure
- (5) High FiO₂ to achieve target SpO₂ indicates serious illness:
- Early warning systems are effective in picking up deterioration earlier, particularly when they combine oxygen delivery (FiO₂ or flow rate), with the resultant SpO₂
 - Increasing respiratory rate is also a highly sensitive marker of deterioration
 - Increasing FiO₂ requirements to maintain target saturations indicates deterioration. High FiO₂ to maintain adequate oxygen saturations means that should further deterioration occur, higher level respiratory support is then urgently required to avoid life-threatening hypoxaemia
- (6) Acute respiratory acidosis:
- This indicates severe physiological derangement (inadequate alveolar ventilation to clear CO₂) and has a number of potential causes. Augmentation of ventilatory support may be required through invasive or NIV
 - NIV is not usually indicated in the absence of acute respiratory acidosis

Abbreviations: ABG, arterial blood gases; FiO₂, fraction of inspired oxygen; hNHF-O₂, humidified nasal high-flow oxygen; NIV, non-invasive ventilation; PaO₂, arterial partial pressure of oxygen; SpO₂, arterial oxygen saturation measured by pulse oximeter.

Revision Group was of the opinion that there was declining use of arterial blood gases (ABG) across Australia and New Zealand, which is detrimental to optimal clinical practice. Therefore, the recommendation to use ABG in appropriate clinical settings is strengthened.

TABLE 3 Grades of recommendation

Grade of recommendation	Description
A	Body of evidence can be trusted to guide practice
B	Body of evidence can be trusted to guide practice in most situations
C	Body of evidence provides some support for recommendation(s), but care should be taken in its application
D	Body of evidence is weak and recommendation must be applied with caution

7. **Peer review:** This statement was sent to all relevant Australian professional societies and colleges for peer review. Comments were received from the Royal Australian College of General Practitioners, the Council of Ambulance Authorities Inc. and the College of Emergency Nursing Australasia. Their comments were reviewed by the Revision Group and incorporated as appropriate, before submission to *Respirology* for external peer review.
8. **Dissemination plan:** The revised document will be freely available as a published open-access document. The statement will be advertised widely via the TSANZ to relevant universities, area health services, professional bodies and societies.
9. **Implementation:** As implementation is key to practice change, recommendations and educational resources were also created and are available via the TSANZ website. Institutions should develop a long-term implementation plan so that recommendations are used in everyday clinical practice. The limited evidence available suggests that the key recommendations from the 2015 Guideline document are not in widespread use and suggests a widespread failure of implementation.^{3,4} We suggest that appropriate use of acute oxygen therapy should be taught at undergraduate and post-graduate levels, and to a range of healthcare staff at all institutions which administer acute oxygen therapy. Critical factors for implementation success include: local 'oxygen champions' (medical and/or nursing), supporting infrastructure and processes (e.g., paper or electronic medical record prescribing), decision assist prompts and regular education programmes for both new and longer-serving staff. It is important to ensure that knowledge and competence are updated and maintained.
10. **Expiry date:** 2027.

B. RECOMMENDATIONS IN DETAIL

1. Assess oxygenation:

- I. Pulse oximetry is a 'vital sign' to be considered together with other signs, including respiratory rate,

and is a predictor of potentially serious clinical events.⁵ Pulse oximetry should therefore be available in all clinical situations in which oxygen is used⁶ (Grade C).

Practice points:

- a. There is variable accuracy of pulse oximetry to predict SaO₂ (arterial oxygen saturation [measured by arterial blood gas]) in acutely ill patients, with SpO₂ measurements both over- and underestimating SaO₂, with wide limits of agreement.^{6–11} The accuracy of SpO₂ may worsen with factors including disease severity and patients' physical characteristics^{6–8,11–16} (see Section C.1.II). Clinicians need to be aware of, and take into account the variable accuracy of SpO₂ in using pulse oximetry in clinical practice and measure ABGs when appropriate.
 - b. An SpO₂ of ≥92% is a practically lower threshold to rule out hypoxaemia, defined as an SaO₂ < 90%⁸ or an arterial partial pressure of oxygen (PaO₂) < 60 mm Hg (8 kPa).⁷ However, SaO₂ may be underestimated by up to 8%¹² and an SaO₂ < 90% may be missed in up to 28%.⁸
 - c. There should always be clinical judgement when interpreting SpO₂ values, and to avoid over-reliance on pulse oximeters. In particular, when diagnosis, severity assessment and treatment are dependent on oxygen status, ABG may be necessary.
 - d. In the immediate assessment of an acutely unwell patient, oxygen saturations should be measured by oximetry, pending the availability of ABG if required (see Point 2).
- II. ABG measurement should be considered in the following situations (Grade C):
- Critically ill patients with cardiorespiratory or metabolic dysfunction.
 - In patients with an SpO₂ < 92% in whom hypoxaemia may be present.
 - Deteriorating SpO₂ requiring increased fraction of inspired oxygen (FiO₂).
 - Patients at risk of hypercapnia (see below).
 - Patients with symptoms or signs compatible with acute respiratory disease in whom a reliable oximetry signal cannot be obtained.

Practice points:

- a. Hypoxaemia requires investigation and treatment of the underlying cause(s), and consideration of the contribution of hypoventilation, including measurement of arterial partial pressure of carbon dioxide (PaCO₂) and pH.
- b. Peripheral venous blood gas (VBG) analysis is a less invasive test; however, it does not provide an accurate estimate of PaCO₂ or PaO₂.^{1,16,17} It does, however, provide rapid clinically important information to assess acutely unwell patients, including pH, lactate, glucose, haemoglobin,

sodium and potassium. A venous partial pressure of carbon dioxide (PCO₂) of <40 mm Hg makes hypercapnia unlikely, but does not rule it out. Therefore, exclusion of hypercapnia, when clinically relevant, requires ABG to be measured.^{16,17}

- c. Arterialized capillary earlobe or fingertip blood gas measurements represent an alternative if unable to obtain ABG, recognizing that whilst providing accurate information about PaCO₂ and pH, it variably underestimates PaO₂ measurements^{18,19} As a result, patient assessment can be based on pH and PCO₂ levels measured from earlobe or fingertip blood gases, together with SpO₂ by pulse oximetry.
- d. PaCO₂ may rise in susceptible individuals given oxygen therapy; therefore, a repeat ABG should be considered in those individuals.

2. Oxygen is a drug and thus requires prescription:

- I. A specific oxygen prescription should be documented in the patient medical record and the drug chart (Grade D).²⁰
- II. A target SpO₂ should be included as part of the prescription.

Practice points:

- a. The minimum requirement for an oxygen prescription is specification of delivery device, range of flow rates that may be administered and target SpO₂ range.
- b. In its most detailed form, the prescription could include (considerable space on the prescription form is needed to provide such detail; see Figure 1):
 - i. the delivery system and interface,
 - ii. the target oxygen saturation range,
 - iii. the range of flow rates that may be used for each delivery system,
 - iv. specification of SpO₂ and FiO₂ (or flow rate) at which clinical review should be sought and
 - v. if hNHF-O₂ therapy is utilized, the temperature setting, flow rate and FiO₂ of entrained oxygen.
- c. Oxygen therapy should be initiated or increased if clinically required, prior to prescription. Increasing oxygen requirement indicates underlying disease deterioration, and should result in clinical review.

3. Recommended SpO₂ targets (see Figure 2):

- I. An SpO₂ target of 88%–92% is recommended in exacerbations of chronic obstructive pulmonary disease (COPD) (Grade B),²¹ and other conditions associated with chronic respiratory failure (such as morbid obesity,²² obesity hypoventilation syndrome,²³ bronchiectasis, cystic fibrosis,²⁴ neuromuscular disease and chest wall deformities such as severe kyphoscoliosis) (Grade C). Where there is diagnostic uncertainty as to whether COPD is the primary cause of the exacerbation, it may be preferable to titrate oxygen therapy to the 88%–92% SpO₂ target range (Grade C).^{21,25,26}
- II. In the presence of hypoxaemia in other acute medical conditions, oxygen should be administered to achieve a target SpO₂ range of 92%–96% (Grade B),^{27,28} with

some evidence in support of a target range of 90%–94% (see Section C.3.IV) (Grade C).

- III. An SpO₂ target of around 85% is recommended in patients previously exposed to bleomycin or in paraquat poisoning (Grade D).^{29–32}
- IV. Patients with carbon monoxide poisoning, decompression sickness, cluster headaches and sickle cell crisis should receive normobaric hyperoxia. In the case of carbon monoxide poisoning, consideration should also be given to hyperbaric oxygen therapy (Grade B).^{33–35}

Details for Oxygen Therapy

Details | Order Comments | Diagnoses

Review Schedule Remaining Administrations: (Unknown) Stop: (Unknown)

Dose: 1 reminder

*Maximum L/min: 2-4 L/min

*Frequency: daily

*Oxygen Delivery Method: Nasal Prongs

FIO2:

*Target Oxygen Saturation: 88-92%

First Dose Date/Time: 17-11-2021 08:00

FIGURE 1 Example of oxygen prescription form (Westmead Hospital, NSW; courtesy: Jimmy Chien and Mary Roberts)

- V. Oxygen can be titrated using a closed-loop control system in which there is automated adjustment of the delivered oxygen concentration in response to continuous measurement of SpO₂. This results in a greater proportion of time within a prescribed target SpO₂ range compared to manual oxygen titration, in non-ventilated adult patients with acute illnesses using standard nasal cannulae, face mask^{36–38} or hNHF-O₂^{39,40} (Grade B).

Practice points:

- a. In the presence of COPD or conditions associated with chronic respiratory failure:
 - If SpO₂ ≥ 88%, oxygen therapy is not initially required.
 - If SpO₂ < 88%, oxygen can be administered via a 24% or 28% Venturi mask, at 1–2 L/min via nasal cannulae, or via a hNHF-O₂ device, and titrated to achieve the target SpO₂.
 - If an SpO₂ target of ≥92% is considered in such patients, ABGs are required to be measured to exclude hypercapnia, before adopting this target.
 - The avoidance of inappropriate high-concentration oxygen therapy may be facilitated by the provision

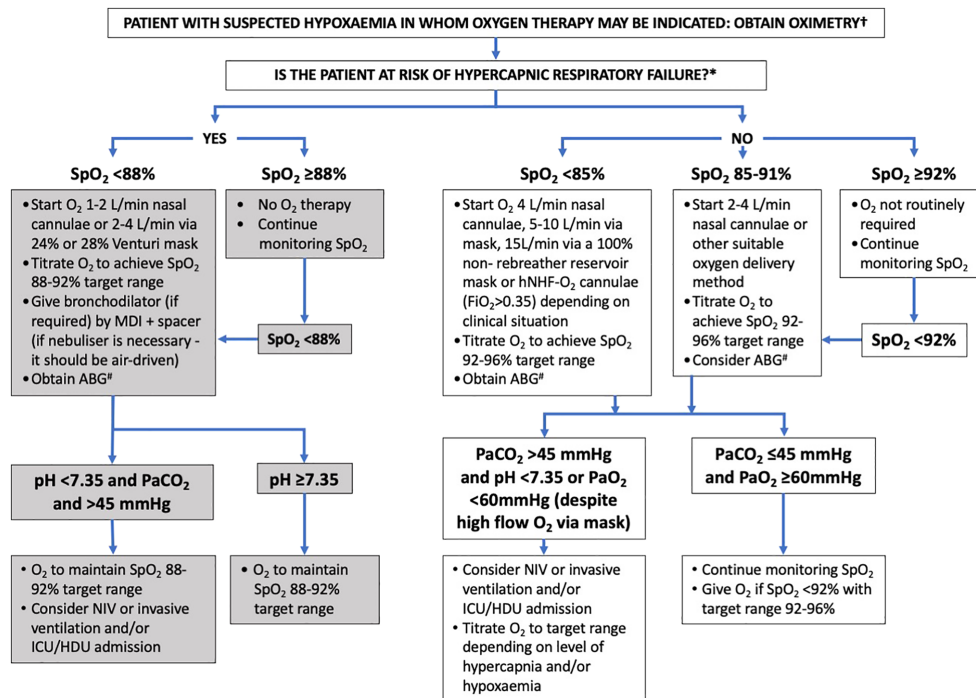


FIGURE 2 Treatment algorithm for oxygen therapy. †If oximetry is not available, or reliable oxygen saturations cannot be determined and hypoxaemia is suspected, oxygen can be delivered at: (1) 1–2 L/min via nasal cannulae or 2–4 L/min via 24% or 28% Venturi mask in patients with acute exacerbations of COPD or conditions known to be associated with chronic respiratory failure (*such as obesity hypoventilation syndrome, chest wall deformities, cystic fibrosis, bronchiectasis, neuromuscular disease and COPD); (2) 2–4 L/min via nasal cannulae in patients who are not critically ill and life-threatening hypoxaemia is not suspected; and (3) 5–10 L/min via simple face mask or 15 L/min through a reservoir mask in patients who are critically ill or in whom life-threatening hypoxaemia is suspected (e.g., post-cardiac arrest or resuscitation, shock, sepsis, near drowning, anaphylaxis, major head injury or in suspected carbon monoxide poisoning). NIV or invasive ventilation and transfer to HDU or ICU should also be considered in this situation. #When administering acute O₂ treatment in the community, the flow chart ends at the point of ABG, since this is not used in this setting. ABG, arterial blood gases; COPD, chronic obstructive pulmonary disease; HDU, high-dependency unit; hNHF-O₂, humidified nasal high-flow oxygen; ICU, intensive care unit; MDI, metered dose inhaler; NIV, non-invasive ventilation; O₂, oxygen; PaCO₂, arterial partial pressure of carbon dioxide; PaO₂, arterial partial pressure of oxygen; Sats, oxygen saturations; SpO₂, arterial oxygen saturation measured by pulse oximeter

of a COPD oxygen alert card,⁴¹ bracelet or warning in the medical record.

b. In the absence of COPD or known chronic respiratory failure:

- If SpO₂ ≥ 92%, oxygen therapy is not routinely required.
- If SpO₂ is 85%–91%, oxygen can be initially instituted at 2–4 L/min via nasal cannulae or other suitable oxygen delivery method, and titrated to achieve the target SpO₂. In many situations, this range of oxygen saturations is unlikely to be associated with risk, although oxygen is commonly administered.
- If SpO₂ < 85%, oxygen can be initiated at 4 L/min via nasal cannulae, through a simple face mask at 5–10 L/min, a 100% non-rebreather reservoir mask at 15 L/min or hNHF-O₂ device (FiO₂ > 0.35). The method of oxygen administration will depend on the SpO₂ level, with higher FiO₂ administered in response to increasingly more severe reductions in SpO₂. Oxygen is titrated to achieve the target SpO₂ as soon as practically possible.

c. If oximetry is not available or reliable SpO₂ cannot be determined and hypoxaemia is suspected, oxygen can be delivered at:

- 24% or 28% Venturi mask, or 1–2 L/min via nasal cannulae in patients with acute exacerbations of COPD or conditions known to be associated with chronic respiratory failure.
- 2–4 L/min oxygen via nasal cannulae in patients who are not critically ill and life-threatening hypoxaemia is not suspected.
- 5–10 L/min via simple face mask, 15 L/min through a 100% non-rebreather reservoir mask or high-flow nasal cannulae (FiO₂ > 0.35) in patients in whom life-threatening hypoxaemia is suspected (see Figure 2).

d. Supplemental oxygen should not be used in normoxic patients in an attempt to protect against subsequent hypoxaemia, in the event of deterioration such as worsening gas exchange and/or alveolar ventilation:

- A high SaO₂ due to supplemental oxygen obscures deterioration in SpO₂.
- In this situation, there is likely to be no major change in vital signs⁴² and no marked decrease in SpO₂ as assessed by pulse oximetry^{43,44} until a potentially life-threatening situation has developed. This may potentially delay recognition of the deterioration and thus provide a false reassurance that the patient is stable.^{45,46} At this late stage, further increasing oxygen therapy will have limited benefit while medical review and higher-level interventions, such as ventilation and transfer to an high-dependency unit (HDU) or intensive care unit (ICU), are undertaken.

4. Delivery:

I. For most patients, standard nasal cannulae are the preferred method of oxygen delivery, with the flow

rate varied to achieve the target oxygen saturation.

Practice point:

The FiO₂ levels delivered by the different delivery systems may vary considerably between patients and be influenced by a number of factors, including respiratory rate and whether the patient's mouth is open or closed.^{47–54} Approximate FiO₂ values delivered by different delivery systems are:

- Standard nasal cannulae can deliver an FiO₂ of 0.24–0.35 at an oxygen flow of 1–4 L/min.
- Venturi masks can deliver an FiO₂ of 0.24–0.60.
- hNHF-O₂ cannulae can deliver an FiO₂ of 0.21–1.0.
- A simple face mask can deliver an FiO₂ of 0.35–0.60 at an oxygen flow of 5–10 L/min.
- A 100% non-rebreather reservoir mask at 15 L/min can deliver an FiO₂ of >0.60.

II. For simple face masks, flow rates of <5 L/min should be avoided due to the potential risk of carbon dioxide rebreathing (Grade C).^{55,56}

III. hNHF-O₂ devices deliver heated, humidified oxygen via wide-bore nasal cannulae at delivered concentrations up to 100% and at high-flow rates up to 70 L/min. The recommendations in this document only apply when oxygen is being delivered via this device and not, for example, when it is used to deliver warm, humidified air. It should be noted that there are other names used for these types of devices which perform the same function (e.g. high-flow nasal oxygen, high-flow nasal prong and high-flow nasal cannula). hNHF-O₂ should be considered in selected patients with severe, hypoxaemic respiratory failure (PaO₂:FiO₂ < 300) (Grade B). Given the paucity of high-quality studies, current evidence does not support the routine use of hNHF-O₂ treatment in acute respiratory acidosis.⁵⁷

IV. Fully conscious hypoxaemic patients should be allowed to position themselves according to their preference (Grade D). In some, but not all, patients, upright posture may result in improved oxygenation.^{58,59}

V. In COPD and other conditions associated with chronic respiratory failure, if bronchodilator is required, the preferred method of administration is via metered dose inhaler (MDI) ± spacer (or via air-driven nebulizer if considered necessary), with supplementary nasal oxygen continued as required (usually via nasal cannulae) for both modes of bronchodilator delivery (Grade B).^{21,60}

Practice points:

- a. The administration of bronchodilator via an oxygen-driven nebulizer has the potential to cause an increase in PaCO₂.^{61–63} It has been recommended that if an oxygen-driven nebulizer is used, then its use is limited to 6 min.¹
- b. When hNHF-O₂ is used to treat serious illness with acute, hypoxaemic respiratory failure, ABG should be measured to exclude acute respiratory acidosis. It

also allows calculation of PaO₂:FiO₂ ratio which is a useful indicator of severity of illness. Because hNHF-O₂ use is indicated for serious illness, recommendations below in Section B.5: 'High FiO₂ to achieve target SpO₂ indicates serious illness' should be considered.

- VI. In asthma, if bronchodilator is required, the preferred method of delivery is by MDI ± spacer. If a nebulizer is required, then it should be air-driven (Grade C).⁶⁴ In both situations, if oxygen therapy is needed, it should continue via standard nasal cannulae during bronchodilator administration.
- VII. All aerosol-generating procedures, for example, hNHF-O₂ and non-invasive ventilatory support (non-invasive ventilation [NIV] and continuous positive airway pressure [CPAP]), pose a risk of transmission of viral infection to staff and patients. While these therapies offer significant benefits to some patients, there are often alternative approaches to management that have less risk of transmitting viral infection via aerosolization. This is of particular relevance during the 2019 SARS-CoV-2 pandemic where protection from aerosol spread remains standard practice.
- VIII. If hNHF-O₂ is used in a patient with suspected or known respiratory viral illness, including SARS-CoV-2, the patient must be fitted with an interface (e.g., face mask) to minimize leak and managed with the highest level of isolation available (class N-negative pressure room is optimal, single room with door closed is adequate) with five-piece personal protective equipment (PPE) (N95 or equivalent mask, gloves, goggles/glasses, gown, hat) precautions for healthcare personnel. Local facility infection control measures must be adopted (see <https://www.health.gov.au/committees-and-groups/infection-control-expert-group-iceg> and <https://covid19evidence.net.au/about-the-taskforce/>) (Grade D).
- IX. NIV and CPAP are delivered by a mask. Patients with suspected or proven respiratory viral illness requiring CPAP/NIV should receive this via a circuit using a non-vented mask with a filtered external expiratory port. Staff should wear five-piece PPE, and the patient should be in the highest available level of isolation. Local facility infection control measures must be adopted⁶⁵ (Grade D).

5. High FiO₂ to achieve target SpO₂ indicates serious illness:

- I. A high FiO₂ to maintain adequate SpO₂ is a clinical indicator of severe illness, and in this situation, there is limited capacity to increase FiO₂ to avoid life-threatening hypoxaemia should deterioration occur. Patients who need an estimated FiO₂ of ≥0.40, such as ≥6 L/min via a simple face mask, to maintain an adequate SpO₂, should receive senior clinician review and may require transfer to a facility such as HDU, where there are appropriate numbers of competent staff able

to provide more intensive monitoring and therapy (Grade D).

- II. Patients who need an estimated FiO₂ of ≥0.50, such as ≥8 L/min via a simple face mask, to maintain an adequate SpO₂, should receive ICU review and most will require a higher level of monitoring and supportive care which an ICU/HDU environment can provide (Grade D).

Practice points:

- a. A reduction in SpO₂ while the FiO₂ is maintained, or increasing FiO₂ requirements to maintain SpO₂, should lead to clinical review of the patient.
 - b. For patients whose oxygen saturations improve with oxygen therapy to above the target oxygen saturation range, oxygen therapy can be reduced or stopped. Oxygen saturation monitoring should continue to allow detection of any subsequent deterioration of the underlying condition and the requirement to increase or resume oxygen therapy.
- III. Early Warning Score (EWS) systems include a number of clinical parameters which together predict inpatient deterioration and subsequent life-threatening adverse patient events. EWS systems recommended for use in New Zealand and across Australia vary markedly.
 - It is recommended that EWS systems include scores that reflect (i) supplemental oxygen administration (i.e., scores increase as oxygen flow or FiO₂ delivery increases) and (ii) patient SpO₂ (i.e., scores increase as SpO₂ decreases) (Grade C).
 - Current evidence suggests that the Queensland Adult Deterioration Detection System (Q-ADDS) outperforms other systems.⁶⁶

Practice point:

- It is important that practitioners are familiar with the EWS system implemented in their healthcare organization. If no system is implemented, then the healthcare organization should consider using the Q-ADDS.

6. Acute respiratory acidosis:

- I. In patients with acute respiratory acidosis, in whom ABG show a pH < 7.35 and PaCO₂ > 45 mm Hg, NIV or invasive ventilation should be considered.⁶⁷⁻⁷⁰ Target SpO₂ during NIV for acute respiratory acidosis should be 88%–92% (Grade A). COPD patients managed with NIV require regular review to assess response to treatment or deterioration, in which case they may need intubation. Assessment should be based on clinical and biochemical parameters (e.g., O₂ requirements, pH, etc.) (Grade C).⁶⁹
- II. In patients in whom oxygen-induced hypercapnia is suspected, oxygen therapy should be titrated to maintain the 88%–92% target oxygen saturation range and not be abruptly stopped due to the risk of profound rebound hypoxaemia (Grade C).⁷¹⁻⁷³

Practice point:

- Reducing FiO₂ to appropriate levels may improve the acute respiratory acidosis. This should be determined by repeat ABG.
- III. In patients with severe cardiogenic pulmonary oedema, CPAP should be considered (Grade A).⁷⁴
- IV. While NIV is not routinely recommended in acute hypoxaemic respiratory failure, its use may be considered in certain groups such as immunosuppressed patients with pulmonary infiltrates requiring ventilatory support (Grade C).^{70,75–79}
- V. It is recommended that patients receiving ventilatory support are located in a ward area such as close observation unit (COU), HDU or ICU (and may include a general ward), where it is essential that there are adequate numbers of staff experienced in ventilatory support to provide an appropriate level of monitoring, clinical expertise and titration of therapy (Grade D).^{68,69,80,81}
- VI. An individualized NIV treatment plan which covers prescription of therapy, escalation and de-escalation of NIV/CPAP, goals of therapy and ‘ceiling of care’ should be documented (Grade D).^{68,80}

C. BACKGROUND EVIDENCE FOR RECOMMENDATIONS

1. Assess oxygenation:

- I. Hypoxaemia is both a marker of risk of a poor outcome due to the severity of the underlying disease(s) that has caused hypoxaemia, and an independent risk factor of poor outcome in its own right.^{82,83} No absolute safe lower limit of PaO₂ or SaO₂ can be set. The clinical effects of hypoxaemia depend on numerous factors including speed of onset and severity, duration, patient age, body temperature, disease chronicity, comorbidities such as anaemia, underlying acute conditions and their associated oxygen demand, physiological factors such as cardiovascular function which influence oxygen delivery to the tissues and individual variation in susceptibility to hypoxaemia.⁸⁴ For example, some patients with acute exacerbations of COPD and chronic respiratory failure may tolerate PaO₂ values of between 20 and 40 mm Hg, equivalent to an SaO₂ value of around 40%–70%,⁸⁵ and acclimatized elite mountaineers may tolerate an SaO₂ of between 35% and 70% descending Mt Everest.⁸⁶ In contrast, healthy adults in simulated high-altitude conditions may become confused at an SpO₂ around 65%, progressing to imminent unconsciousness around 55%.⁸⁷ It has been proposed that a PaO₂ of 50 mm Hg (6.6 kPa) can be considered as the safe lower limit of hypoxaemia in patients with COPD,¹⁸ and that oxygen therapy which achieves a PaO₂ of at least 50 mm Hg would prevent immediate death from hypoxaemia.⁸⁸

- II. Factors that may affect the accuracy of pulse oximetry include severe hypoxaemia and hypercapnia,^{11,12,16} sepsis,¹¹ carboxyhaemoglobin and methaemoglobin levels,⁸ anaemia, dark skin,¹⁴ low perfusion and low body temperature¹³ causing SpO₂ to overestimate SaO₂, while excessive ambient light and nail polish cause underestimation.^{6,15}
- III. A systematic review and meta-analysis comparing ABG and VBG measurements¹⁷ reported that the 95% prediction interval of bias for PaCO₂ was wide, from –10.7 to +2.4 mm Hg; PaO₂ was significantly higher than the venous partial pressure of oxygen (PO₂) by 36.9 mmHg (95% CI 27.2–46.6 mm Hg); the pH values were similar with the arterial pH 0.03 being higher than the venous pH (95% CI 0.029–0.038). A subsequent comparison of ABG and VBG in COPD found that venous PCO₂ was up to 21 mm Hg higher than arterial PCO₂ (95th percentile), but could also be lower than ABG by 11 mm Hg (5th percentile).¹⁶ Therefore, clinicians should guard against over-reliance on venous PCO₂, particularly as diagnosis of hypercapnic and metabolic acidosis has far-reaching implications. Thus, accurate diagnosis using ABG is required in those clinical situations but is likely being under-used in acute medical emergencies in Australia and NZ.

2. Oxygen is a drug and thus requires prescription:

- I. Oxygen is used to treat hypoxaemia, not breathlessness. Oxygen therapy does not relieve breathlessness in the absence of hypoxaemia. For example, there is no clinical benefit with short-burst oxygen therapy in COPD patients with breathlessness,^{89,90} or with the use of oxygen over room air via nasal cannulae for patients with COPD who do not have severe resting hypoxaemia. Similarly, there is no additional symptomatic benefit in the use of daily oxygen over room air via nasal cannulae for refractory breathlessness in the palliative setting.⁹¹ Thus, in the absence of hypoxaemia, oxygen therapy is not indicated except in carbon monoxide poisoning, cluster headaches or sickle cell crisis.
- II. The specification of a range of O₂ flow rates that can be administered with a particular delivery device is to avoid flow rates being increased to maintain SpO₂ within the specified target range, without considering potential underlying clinical deterioration and need for urgent clinical review. Hence, if SpO₂ cannot be maintained within the specified target SpO₂ range using the specified range of O₂ flow rates, then this requires clinical review.
- III. The potential risks due to hyperoxaemia with high-concentration oxygen therapy include respiratory (increased PaCO₂, absorption atelectasis and direct pulmonary toxicity), cardiovascular (increased systemic vascular resistance and blood pressure, reduced coronary artery blood flow, reduced cardiac output), cerebrovascular (reduced cerebral blood

flow) effects and increased reperfusion injury due to increased reactive oxygen species.^{91–96}

IV. The physiological response of an increase in PaCO₂ due to high-concentration oxygen therapy has been demonstrated not only in stable and acute exacerbations of COPD,⁸⁵ but also in severe asthma,^{27,97} community-acquired pneumonia,²⁸ morbid obesity²² and obesity hypoventilation syndrome.²³ Proposed mechanisms for oxygen-induced hypercapnia include increased ventilation perfusion mismatch due to reduced hypoxic pulmonary vasoconstriction, reduced ventilatory drive, atelectasis and the Haldane effect (increased PaCO₂ due to displacement from haemoglobin by O₂), with the contribution of each likely to depend on the clinical situation.¹

3. Recommended SpO₂ targets:

I. A target SpO₂ range of 88%–92% is recommended in the treatment of COPD and other conditions associated with chronic respiratory failure due to demonstration of:

- A greater than two-fold reduction in mortality with pre-hospital oxygen therapy titrated to this target, compared with high-concentration oxygen therapy in patients with an acute exacerbation of COPD.²¹
- In hospitalized COPD patients receiving supplemental oxygen during an exacerbation, the risk of mortality is greater in those within the 93%–96% oxygen saturation range, compared within the 88%–92% range, when data are adjusted for baseline mortality risk.⁹⁸
- A clinically significant increase in PaCO₂ results from several minutes of 100% oxygen therapy in patients with chronic respiratory failure due to obesity hypoventilation syndrome.²³

II. A general target SpO₂ range of 92%–96% in acute medical conditions, excluding invasively ventilated patients, has been recommended, based on the evidence from the systematic review and meta-analysis of all randomized controlled trials comparing liberal versus conservative oxygen therapy in critically ill adults.⁹⁹ A total of 25 randomized controlled trials enrolled 16,037 patients with sepsis, critical illness, stroke, trauma, myocardial infarction or cardiac arrest, and patients who had emergency surgery. Compared with a conservative oxygen strategy, a liberal oxygen strategy (median baseline SpO₂ across trials, 96% [range 94%–99%, IQR 96–98]) was associated with an increased risk of mortality in-hospital (relative risk [RR] 1.21, 95% CI 1.03–1.43). Morbidity outcomes were similar between groups. Findings were robust to trial sequential, subgroup and sensitivity analyses.

III. An international expert panel report, which used the Lancet review⁹⁹ to inform guidelines, made a strong recommendation for maintaining an SpO₂ of no more than 96% in acutely unwell patients (upper limit).¹⁰⁰ The panel suggested that patients with

acute stroke or myocardial infarction and an SpO₂ ≥ 90% not receive supplemental oxygen (a weak recommendation if SpO₂ is 90%–92% and a strong recommendation if SpO₂ is 93%–100%). The findings from two recent studies of mechanically ventilated patients suggest that there is no benefit of conservative versus liberal oxygen therapy in this setting,^{101,102} and thus there are potential differences in how oxygen should be used between mechanically ventilated and other acute medical patients.

IV. Physiological support for the suggested target range:

- An SpO₂ of 92% is a practical lower threshold to rule out hypoxaemia, defined as an SaO₂ < 90%⁸ or a PaO₂ < 60 mm Hg (8 kPa).⁷
- There is no known risk of hypoxic tissue injury at an SaO₂ of 90%.
- Older healthy subjects have SaO₂ levels to this lower level of 90%.^{103,104}
- Healthy subjects have a mean nadir SpO₂ of around 90% during sleep.¹⁰⁵
- Subjects with sleep-disordered breathing commonly tolerate SpO₂ levels between 70% and 90% for prolonged periods.¹⁰⁵
- Adults with comorbidities tolerate SpO₂ levels between 80% and 90% during long-distance travel.¹⁰⁶
- In adults with coronary artery disease, anaerobic metabolism indicative of myocardial ischaemia is observed in some patients with SaO₂ between 70% and 85%, suggesting a ‘safe’ lower limit of oxygen saturation of 90%.¹⁰⁷

V. There is a significant variability in SpO₂ targets recommended in national and international guidelines in acute cardiac and medical emergencies. For example, the National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand recommend supplemental oxygen therapy in patients suffering from acute coronary syndrome or heart failure when oxygen saturations are <94%, while recognizing that those with comorbid COPD should have target SpO₂ of between 88% and 92%.^{108,109} In comparison, the American College of Cardiology/American Heart Association recommend supplemental oxygen in patients suffering from non-ST elevation acute coronary syndrome when oxygen saturations are <90%, are in respiratory distress or have high-risk features of hypoxaemia.¹¹⁰ The European Society of Cardiology guidelines for the management of acute heart failure recommend giving supplemental oxygen to patients with oxygen saturations <90% or have a PaO₂ of <60 mm Hg.¹¹¹

VI. A recommended target SpO₂ range of 85% in patients with prior exposure to bleomycin or in paraquat poisoning is due to the demonstration of:

- Potentiation of lung injury by oxygen.^{29,30}
- Lack of harm from hypoxaemia with saturations around 85% in these clinical situations.³¹

VII. The evidence that oxygenation above target oxygen saturations delays deterioration of SpO₂ comes from physiological studies of hypoventilation^{43,44} and from modelling of rapidly increasing right to left shunt.⁴⁶ These studies show that there is clinically significant delay in SpO₂ falling, despite halving of ventilation and increasing alveolar partial pressure of CO₂. Although this has direct implications to patients receiving sedating drugs, SpO₂ may respond differently in other acute medical emergencies, where higher than the recommended target ranges for SpO₂ are achieved with supplemental oxygen.

4. Delivery:

I. The potential advantages of conventional nasal cannulae as an initial method of delivering oxygen therapy are:

- Ability to give bronchodilator by MDI ± spacer or air-driven nebulizer at the same time as oxygen is administered.
- Oxygen can be prescribed by variable flows to achieve a target saturation range rather than a fixed FiO₂, although oxygenation may be maintained better with Venturi mask.⁴⁷
- Comfort, ease of use and low cost.
- Less likely to be taken off to eat or speak, and less likely to fall off.
- No risk of rebreathing of carbon dioxide.
- However, skin damage from pressure areas and nasal drying may occur.

II. hNHF-O₂ delivers oxygen therapy at higher flow rates and FiO₂ than conventional oxygen therapy (simple face mask, nasal cannulae or Venturi mask) and, therefore, is a way to administer greater respiratory support in seriously ill patients. The higher flow rates (up to 70 L/min) can be titrated to provide sufficient flow according to the patients' minute ventilation, provide positive end-expiratory pressure (PEEP) > 5 cm H₂O depending on flow rate and other factors^{112,113} and reduce upper airway dead-space by washing out CO₂.

III. Potential advantages of hNHF-O₂ compared with standard O₂ therapy include:

- Reduced risk of endotracheal intubation in patients with hypoxaemic respiratory failure (although it is not associated with reduced mortality).^{57,79,114,115}
- Reduced respiratory rate and level of dyspnoea, and improved gas exchange (PaO₂:FiO₂ ratio) with reduced accessory muscle activation.
- Heated and humidified gas may facilitate greater comfort and airway secretion clearance.
- Preservation of upper airway function (e.g., speech, cough and swallowing).

IV. Potential disadvantages of hNHF-O₂ devices include:

- Risk of complacency if a high FiO₂ requirement is not recognized to represent life-threatening illness requiring more than correction of hypoxaemia.

- Role in severe exacerbations of COPD and asthma has not been clarified, with studies ongoing.

V. The efficacy of hNHF-O₂ has been studied in a variety of patients suffering from acute, severe, hypoxaemic respiratory failure.^{79,115,116} The inclusion criteria for patients varied between studies and mostly included patients with pneumonia and sepsis. In most studies, patients with reduced Glasgow Coma Scale (GCS) and/or a suspected inability to maintain the upper airway were excluded. Use of hNHF-O₂ in conditions such as asthma or COPD exacerbations with acute respiratory acidosis, and cardiogenic pulmonary oedema has not been established. Therefore, the use of hNHF-O₂ should be carefully considered to ensure that it is used in appropriate clinical settings.

VI. The risk of spread of infectious diseases from aerosol-generating procedures, which includes all delivery devices discussed in this document, remains uncertain. Although the oxygen delivery devices generate aerosols which potentially spread infectious agents, the absolute risk and RRs (compared with talking and coughing) are not currently able to be quantified. However, aerosol spread (hence the risk of spread of viral infection) may be considerably less than previously thought.^{65,117–120}

5. High FiO₂ to achieve target SpO₂ indicates serious illness:

I. A comparison of 33 different EWS systems¹²¹ showed those assigning scores to oxygen saturation (from pulse oximetry) and oxygen administration outperformed systems that did not include these parameters for predicting in-patient deterioration and subsequent adverse patient events. The best performing of these 33 systems was the National Early Warning Score (NEWS), introduced in the UK in 2012. Since then, numerous EWS systems have been developed. Those used in Australia and New Zealand vary markedly, including significant variation in how oxygen supplementation and pulse oximetry components are scored. The largest validation of Australian systems to date⁶⁶ compared the performance of state-wide systems in New South Wales (Between The Flags, BTF) and Queensland (Q-ADDS), and two other international systems (including NEWS). This showed Q-ADDS outperformed BTF in predicting adverse outcomes.

II. It is recommended that EWS systems include both scores that reflect supplemental oxygen administration (that increase with higher oxygen flow or percentage delivered) and patient oxygen saturation (that increase as saturation decreases). However, assigning scores for hyperoxia in EWS for patients at risk of type 2 respiratory failure receiving supplemental oxygen was not shown to improve system performance and therefore, the current evidence base does not support this approach.¹²²

- III. Increasing respiratory rate is a highly sensitive marker of clinical deterioration and high respiratory rates are strong predictors of serious events, such as cardiac arrest. Although respiratory rate measurement is common to all EWS (and thus does not contribute to differences in performance), it is nevertheless an important measure of the severity of underlying illness and predictor of poor outcome.⁵
- IV. Patients who fail hNHF-O₂ therapy and require intubation may be predicted with an index derived from FiO₂, SpO₂ and respiratory rate.¹²³ However, this score requires a more complex calculation to be performed and has only been validated in patients with respiratory failure from pneumonia or pneumonitis. EWS systems improve the detection of deterioration irrespective of the underlying pathology.¹²⁴
- 6. Acute respiratory acidosis:**
- V. There are local, national and international guidelines on the use of NIV and CPAP in acute medical emergencies, which provide concise and practical information on appropriate use. The reader is directed to those guidelines and statements for in-depth reviews that support those recommendations.^{67–69,80,125}
- VI. NIV and CPAP may be used to deliver mechanical respiratory support via a nasal or oro-nasal mask in clinically indicated scenarios, when oxygen therapy alone is insufficient. NIV provides higher inspiratory and lower expiratory pressures, thus providing ventilatory support, while CPAP maintains the same airway pressure during the entire respiratory cycle. Higher FiO₂ concentrations can also be delivered via nasal or oro-nasal masks compared to nasal cannulae or open face mask. There are variations in delivery circuits for NIV and CPAP to suit the clinical environments and applications (e.g., single limb filtered circuits used during the COVID-19 pandemic or dual limb circuits).
- VII. NIV and CPAP require specific expertise to administer and to monitor response. Therefore, patients should be clinically managed in environments which provide sufficient technical support for administration, continuous monitoring, sufficient nursing expertise and nursing ratios (1:2 or 1:3) and appropriate medical support. In most situations, this would be in an ICU, HDU, COU or a medical ward with high level of experienced staffing, continuous monitoring and support.^{68,69,80}
- VIII. NIV may be indicated for acute respiratory acidosis, in conditions such as COPD, non-cystic fibrosis and cystic fibrosis related bronchiectasis, neuromuscular and chest wall disease and obesity hypoventilation syndrome.^{68,69,80} Measurement of ABG is necessary prior to or, as soon as practical, after the initiation of NIV to confirm and measure the severity of respiratory acidosis and to monitor treatment response.
- IX. NIV is not routinely indicated in acute hypoxaemic respiratory failure without acidosis. Results from clinical trials and observational studies have provided mixed results for various patient groups,^{70,76,126–129} while weak evidence from a recent meta-analysis suggests that NIV may reduce mortality and the risk of intubation.⁷⁹ There may be a role for NIV, if clinically indicated in immunocompromised patients with pulmonary infiltrates.^{70,75,78,127,129} Should NIV be given in acute hypoxaemic respiratory failure without acidosis, it must be carefully considered as more severe illness (i.e., higher respiratory and heart rates, worse acidosis and hypoxaemia and impaired consciousness) predicts NIV failure. Delaying intubation and NIV failure are associated with increased mortality. Therefore, it must be delivered by suitably experienced clinicians and be regularly reviewed, ideally in ICU/HDU given the need for intubation should NIV fail.¹³⁰
- X. Helmet NIV is at least as efficacious as oro-nasal delivery in hypoxaemic and hypercapnic respiratory failure. There may be advantages in application (e.g., improved comfort and possibly improved outcomes) compared with oro-nasal administration or hNHF-O₂. However, there is little/no experience of its use in Australia and NZ and further studies to establish benefit are needed.^{79,130,131}
- XI. Asthma is highly heterogeneous; it may have a clinical appearance similar to COPD with long-standing, irreversible airways obstruction. In contrast, other patients may have acute, severe bronchospasm but have normal lung function at other times. There is little evidence to support the use of NIV in acute severe asthma, either with or without acute respiratory acidosis (there has only been a single trial of NIV in the former¹³²). As such, use of NIV in asthma should be carefully considered and should not delay intubation and mechanical ventilation which result in excellent outcomes when administered.⁶⁹
- XII. In acute pulmonary oedema and acutely decompensated chronic heart failure, NIV or CPAP may reduce intubation and mortality compared with O₂ therapy, with little evidence of benefit of NIV over CPAP. However, first-line treatment for these patients is pharmacological therapy, with escalation to non-invasive ventilatory support if clinically required. Caution should be exercised in acute cardiac failure with hypotension where NIV may reduce mean arterial blood pressure further.

CONFLICT OF INTEREST

Gregory King, Catherine Buchan, Jimmy Chien, Claude S. Farah, Christine F. McDonald, Belinda Miller, Maitri Munsif, Alex Psirides, Lynette Reid, Mary Roberts, Natasha Smallwood and Sheree Smith declared no conflicts of interest

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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