Managing acute on chronic respiratory failure: a guide to non-invasive ventilation

Acute-on-chronic respiratory failure is becoming an increasingly common medical emergency, mostly as a result of exacerbations of chronic obstructive pulmonary disease. It is associated with high mortality rates and so practising the best evidence-based management is vital.

This review article is designed to enable the reader to understand respiratory failure from first principles. Using this foundation, a pragmatic and up-to-date approach to diagnosing and treating patients with acute-on-chronic failure is fully explored.

Definition, pathophysiological mechanisms and causes of respiratory failure

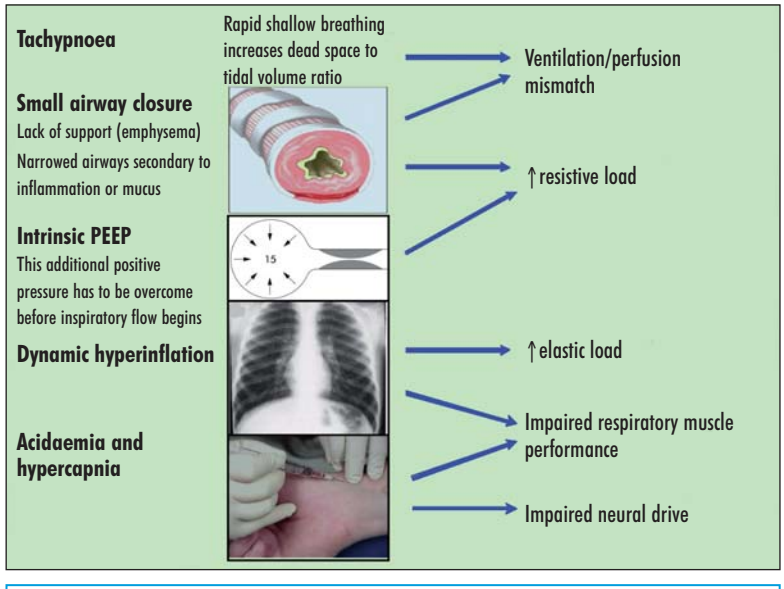
Impairment of either the lung or the respiratory pump which ventilates the lung or a combination of the two can lead to respiratory failure. In this situation, the respiratory system is unable to perform one or both of its gas exchange functions. Accordingly, the definition of respiratory failure is based on an arterial oxygen tension (PaO_2) of <8 kPa, an arterial carbon dioxide tension ($PaCO_2$) >6 kPa or both. Clearly, these parameters move along a continuum from normal to abnormal and the diagnosis of respiratory failure should take into account the patient's clinical status rather than relying on strict cut-off values (Roussos and Koutsoukou, 2003).

Lung failure usually results in hypoxaemic or type 1 respiratory failure (PaO_2 <8 kPa) and the commonest cause for this is ventilation/perfusion (V/Q) mismatch which can occur in airways diseases, pneumonia, pulmonary embolus and pulmonary oedema. Impaired diffusion (e.g. interstitial lung disease) and right-to-left shunting (e.g. arteriovenous malformations) also cause hypoxaemia.

Pump failure leads to alveolar hypoventilation and consequent hypercapnic or type 2 respiratory failure (PaO_2 <8 kPa with $PaCO_2$ >6 kPa). The respiratory pump comprises cortical and brainstem input, nerve and neuromuscular junction transmission and the chest wall, including the respiratory muscles, and so impairment at any of these sites can lead to pump failure. The commonest cause is respiratory muscle fatigue as a result of the excessive elastic load of a poorly compliant chest wall (e.g. kyphoscoliosis) or lungs (e.g. acute respiratory distress syndrome) or an excessive resistive load from obstructed airways.

The genesis of respiratory failure often involves multiple mechanisms (*Figure 1*) and one of the best ways to illustrate this is to observe how respiratory failure may evolve in an exacerbation of chronic obstructive pulmonary disease.

Figure 1. Acute-on-chronic respiratory failure in chronic obstructive pulmonary disease involves multiple mechanisms. PEEP = positive end expiratory pressure.



Chronic and acute-on-chronic respiratory failure

There are numerous underlying causes of chronic respiratory failure (*Table 1*), the commonest being chronic obstructive pulmonary disease. Patients with chronic respiratory failure have a normal pH but may have elevated bicarbonate levels to compensate for any hypercapnia.

Acute-on-chronic respiratory failure represents an acute deterioration in a patient with chronic respiratory failure and patients may present with worsening dyspnoea, altered mental state or respiratory arrest. The pH is less than 7.35 and in the presence of elevated bicarbonate levels, the $PaCO_2$ will be significantly higher than in patients with acute hypercapnic respiratory failure (Hart, 2008). The deterioration is most commonly the result of a respiratory tract infection which is enough to overwhelm a respiratory system that is functioning close to, or at its capacity. Other acute precipitants should also

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be borne in mind (*Table 2*) and the nature of the underlying cause often provides a clue. For example, patients with neuromuscular disorders are particularly prone to pneumonia either from retained secretions or aspiration which are both secondary to an ineffective cough.

The age distribution of acute-on-chronic respiratory failure is bimodal with the majority of patients being over the age of 60 years as exacerbations of chronic obstructive pulmonary disease are the commonest cause (*Figure 2*). Plant et al (2000) demonstrated that out of 972 patients admitted to hospital with acute exacerbations of chronic obstructive pulmonary disease, 454 (47%) had hypercapnia and 199 (20%) had a respiratory acidosis, with the latter group having worse outcomes. Indeed, acute-on-chronic respiratory failure tends to occur in patients at the more severe end of the disease spectrum and this is highlighted by 1-year mortality figures in the region of 20–40% for patients with chronic obstructive pulmonary disease depending on whether intensive care unit admission was necessary (Groenewegen et al, 2003; Ai-Ping et al, 2005) and 50% in patients with both cystic fibrosis (Ellafi et al, 2005) and non-cystic fibrosis bronchiectasis (Dupont et al, 2004). Global prevalence of acute-on-chronic respiratory failure is expected to increase as a result of the ageing population in the developed world, increased smoking rates in the developing world and improved therapy for patients with chronic lung disease.

Managing acute-on-chronic respiratory failure: general issues

Management is directed at reversing the acute precipitant while supporting ventilation and preventing or treating any other organ dysfunction. This latter point refers principally to circulatory collapse that may occur as a result of respiratory decompensation. Patients need to be managed by staff who have the necessary expertise and where there is rapid access to invasive ventilation and so the setting should be a specialized respiratory ward, high dependency unit or intensive care unit. Management will also depend on the underlying disease and this is outlined under the disease-specific heading below.

History taking

The underlying cause of the chronic respiratory failure will usually be known already. Occasionally the development of chronic respiratory failure is so insidious that symptoms only become apparent at decompensation. Obesity hypoventilation syndrome and chronic obstructive pulmonary disease–obstructive sleep apnoea overlap are two such examples and obesity hypoventilation syndrome may present de novo with coma (Mokhlesi and Tulaimat, 2007). This highlights the importance of taking both a respiratory history and a history of sleep-disordered breathing. Symptoms suggestive of sleep-disordered breathing include morning headache, daytime sleepiness and witnessed apnoeas.

Examination and investigation

The initial assessment is directed at finding reversible precipitants (*Table 2*). Primary investigations will include full blood count, C-reactive protein to assess inflammatory status, and urea and electrolytes and liver function tests to assess fluid status and to exclude other metabolic derangements. Arterial blood gases and a chest radiograph complete the mandatory tests.

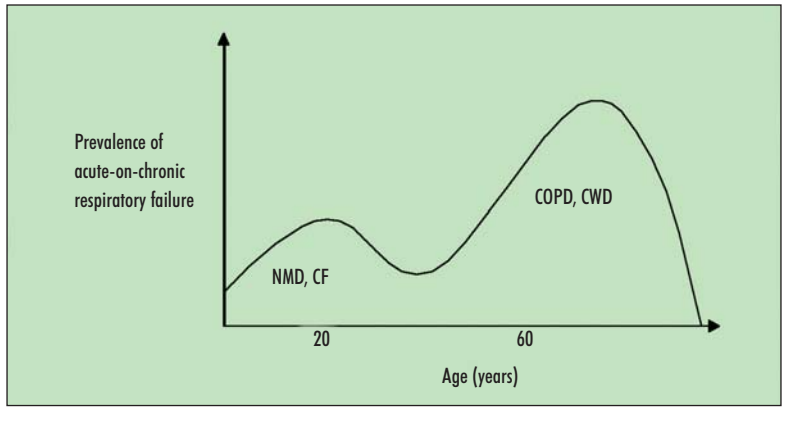
Table 1. Some causes of chronic respiratory failure

Obstructive lung diseases	Chronic obstructive pulmonary disease
	Cystic fibrosis and non-cystic fibrosis bronchiectasis
Chest wall disorders	Kyphoscoliosis
	Thoracoplasty or old tuberculosis procedures
	Obesity hypoventilation syndrome
CNS abnormalities	Brainstem cerebrovascular accident
	Congenital central hypoventilation syndrome
Neuromuscular disorders	Duchenne muscular dystrophy
	Old poliomyelitis
	Motor neurone disease
Miscellaneous	Severe obstructive sleep apnoea

Table 2. Common precipitants of acute-on-chronic respiratory failure

Viral and/or bacterial airway infection
Pneumonia
Pulmonary emboli
Pulmonary oedema
Pneumothorax
Uncontrolled oxygen therapy
Retained bronchopulmonary secretions
Sedatives
Postoperative

Figure 2. Schematic diagram of bimodal distribution of acute-on-chronic respiratory failure (not drawn to scale). CF = cystic fibrosis; COPD = chronic obstructive pulmonary disease; CWD = chest wall deformity; NMD = neuromuscular disease.



Oxygen therapy

Supplemental oxygen should be given in a controlled manner via a venturi mask and titrated to reach a target saturation of between 88 and 92% (O'Driscoll et al, 2008). This approach not only reduces the risk of hyperoxia-induced hypercapnia but also ensures the effects of oxygen toxicity, such as myocardial depression, are minimized.

Antibiotics

Broad spectrum antibiotics are often administered regardless of the underlying disease unless the precipitant is very clearly of non-infectious origin. Here the benefit of giving blind antibiotic treatment outweighs the potential risks of adverse drug reactions and emergence of resistant strains because of the seriousness of acute-on-chronic respiratory failure. Antibiotic prescribing should be based on local microbiological advice and stratified according to whether the pathogens are likely to have been community acquired or hospital acquired.

Ventilatory support

A proportion of patients will require ventilatory support, mostly non-invasively via a mask interface and occasionally invasively via an endotracheal tube, and these options will be detailed later. Continuous positive airway pressure is not usually indicated in acute-on-chronic respiratory failure as it does not augment the respiratory pump.

Doxapram

Before the advent of non-invasive ventilation, intravenous doxapram was often used in this scenario. It is the most well known and most effective of the respiratory stimulants although there is actually very little trial data pertaining to its use in ventilatory failure (Greenstone and Lasserson, 2003). Only one published randomized controlled trial has compared doxapram to non-invasive ventilation (Angus et al, 1996) and this demonstrated that non-invasive ventilation was superior in terms of blood gas improvement at 4 hours and survival at 24 hours. Perhaps the only role now for doxapram in hypercapnic respiratory failure is where there are no facilities to provide ventilatory assistance and in this instance the dose should be titrated to achieve maximum clinical benefit.

Ceiling of therapy and palliation

It is preferable to plan in advance with the patient and his/her carers how therapy should proceed in the event of an acute deterioration. This can help to define a point at which further treatment escalation is not desirable so that the ceiling of therapy can be ascertained. The decision-making process is likely to involve discussion of the types of ventilation and their likelihood of success and also discussion about the weaning process. The prognosis of a patient who has developed acute-on-chronic respiratory failure depends on many factors. Perhaps the most important of these are the nature and severity of the underlying disease process, associated co-morbidities, age and nutritional status.

It may be entirely appropriate to redirect care towards a palliative approach rather than aiming to recover the decompensation. This is particularly likely where the underlying disease is rapidly progressive (e.g. motor neurone disease). In terms of symptom control, dyspnoea may be attenuated by the use of opiates or benzodiazepines and non-invasive ventilation itself may also play a role by reducing the work of breathing. If non-invasive ventilation is being used for palliation then it is important to make sure that the back-up breaths option, which delivers mandatory breaths in the event of apnoeas, is deselected.

Managing acute-on-chronic respiratory failure: disease-specific issues

Chronic obstructive pulmonary disease

During the first hour, the main focus of therapy should be on instituting bronchodilators, systemic steroids and controlled oxygen therapy as a proportion of patients will improve without the need for ventilatory assistance (Elliot, 2007). There is no difference in outcome whether nebulized salbutamol or ipratropium bromide are used and, although commonly given, no evidence that the combination of the two is any more effective (McCrory and Brown, 2002). Nebulizers should be driven with air rather than oxygen. Systemic steroids marginally improve hospital outcomes (Davies et al, 1999) although their specific use for acute-on-chronic respiratory failure has not been evaluated and they are also implicated in the myopathy associated with critical illness (Hanson et al, 1997). Intravenous aminophylline is not recommended (Barr et al, 2003).

Lower respiratory tract infection is the commonest precipitating factor of respiratory failure with one study (Papi et al, 2006) demonstrating that 78% of patients admitted to hospital with severe exacerbation had evidence of viral and/or bacterial infection. Unfortunately there are no good data concerning the role of antiviral therapy in respiratory failure caused by chronic obstructive pulmonary disease. Indeed, there remains controversy over whether or not to give antibiotics but the available data support the use of antibiotics in those patients with purulent sputum (Anthonisen et al, 1987).

Neuromuscular disease

Two particular problems may occur in this group of patients: excessive bronchopulmonary secretions as a result of ineffective cough and worsened bulbar weakness secondary to acute illness (Simonds, 2007). Physiotherapy, particularly while the patient is using non-invasive ventilation which augments cough, is the key to secretion clearance and a cough insufflator/exsufflator may be of

additional use. This machine uses positive pressure to promote maximal lung inflation followed by an abrupt switch to negative pressure during which the patient coordinates his/her cough attempt with the result that the cough peak flow is increased (Chatwin et al, 2003).

Worsened bulbar weakness which impairs swallowing should alert the clinician to the need for nasogastric feeding as adequate nutrition and hydration is vital for recovery.

Cystic fibrosis

Pneumothorax is a relatively common complication in patients with cystic fibrosis and since there is an increased risk of pneumothorax with positive pressure ventilation, whether invasive or non-invasive, care has to be taken to use the lowest inspiratory pressures possible. There should also be a low threshold for repeating the chest X-ray if there is a further deterioration. As it can be difficult to differentiate pneumothorax from cystic bullae a computed tomography scan of the thorax may be required. If a pneumothorax was the initial precipitant then a chest drain must be inserted before ventilation is initiated.

Tenacious bronchopulmonary secretions are a hallmark of cystic fibrosis and mucolytic therapy, such as nebulized hypertonic saline and DNAase, along with physiotherapy should be intensified during an acute exacerbation.

First-line antibiotic therapy is a combination of a beta-lactam antibiotic and an aminoglycoside which are synergistic against Pseudomonas.

Non-invasive ventilation

Non-invasive ventilation assists the respiratory pump by expanding tidal volume as a result of increasing the pressure difference between inspiration and expiration. It can be delivered by either intermittent positive pressure to the upper airway or by intermittent negative pressure to the chest wall although the latter is seldom used. Many non-invasive ventilation machines in the UK are bi-level ventilators which provide both inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP or PEEP). Pressure-preset ventilators are favoured over volume-preset ventilators in the ward setting as they tend to compensate leaks more effectively and are simpler to use.

The main benefit of applying non-invasive ventilation is the avoidance of endotracheal intubation with its associated problems such as ventilator-acquired pneumonia. It often allows earlier ventilatory support and this can 'buy time' while pharmacological and physical therapies get to work. Furthermore, the non-invasive ventilation mask interface can be slipped on and off easily and so is ideal for weaning and it enables oral diet and verbal communication to continue. Patients with acute-on-chronic respiratory failure who are already established on home non-invasive ventilation may be ventilated for a longer duration and at higher pressures during acute decompensation.

When to use non-invasive ventilation

The rule of thumb is that the patient should have a pH<7.35, $PaCO_2$ >6 kPa and be tachypnoeic. Non-invasive ventilation can then either be used as a trial, with intubation if it fails, or as the ceiling of treatment. Most evidence for the use of non-invasive ventilation comes from patients with acute hypercapnic respiratory failure caused by chronic obstructive pulmonary disease and for this group of patients, it is considered the standard of care (Lightowler et al, 2003) because it reduces intubation rates, length of hospital stay and mortality (Brochard et al, 1995). However, non-invasive ventilation certainly works in hypercapnic respiratory failure from the conditions listed in *Table 1* as well.

In the past hypercapnic coma and/or severe acidosis in chronic obstructive pulmonary disease have been considered to be contraindications to non-invasive ventilation but evidence suggests that patients with these features can be treated successfully with non-invasive ventilation (Squadrone et al, 2004; Diaz et al, 2005).

When not to use non-invasive ventilation

The often cited contraindications to non-invasive ventilation were merely exclusion criteria in the early controlled trials rather than true contraindications. However, there are circumstances in which the use of non-invasive ventilation would be considered unwise (*Table 3*).

Practical guide to non-invasive ventilation

1. With acute decompensation, it is better to choose a face mask or nasal mask with chin straps as mouth breathing predominates in dyspnoeic patients and this causes leak. Make sure that the mask is the correct size by using the sizing chart that comes with the mask. Protect the nasal bridge with a dressing.
2. Spend time explaining to the patient what is going to happen as this simple step may mitigate against the claustrophobia that the patient is likely to feel. Patient compliance is a major determinant of success.
3. Entrain oxygen to attain target saturations of 88–92% – 1–2 litres/minute is usually sufficient.
4. The selection of ventilator and settings will depend on local protocol. For pressure preset bi-level ventilators set the initial IPAP at 12 cmH_2O and EPAP at

Table 3. Contraindications to non-invasive ventilation

Uncooperative patient
Imminent cardiorespiratory arrest
Undrained pneumothorax
Vomiting
Copious airway secretions
Inability to protect the airway
Orofacial abnormalities which interfere with the mask–face interface

4 cmH_2O. Use the spontaneous/timed mode and set the back up rate to 12 breaths per minute. If inspiratory time needs to be set then set between 2 and 3 seconds or less than 50% of the respiratory cycle. Set the IPAP trigger to -0.5 cmH_2O if prompted.

5. Titrate IPAP by up to 5 cmH_2O increments every 5–10 minutes according to patient comfort in order to get good chest expansion. The respiratory rate and heart rate are good guides to improvement and these should gradually come down.
6. Repeat arterial blood gases at 1 hour and titrate IPAP and EPAP settings according to patient comfort and arterial blood gas results (*Table 4*). Do not increase the EPAP above 10 cmH_2O as this provides no additional benefit and will reduce venous return.
7. Repeat arterial blood gases 1 hour after any subsequent change in settings.
8. Patients should improve within hours of starting non-invasive ventilation providing that ventilation has been optimized. However, up to 30% of patients will fail (British Thoracic Society Standards of Care Committee, 2002).

Weaning from non-invasive ventilation

Patients who benefit from non-invasive ventilation should use it as much as possible for the first 24 hours. Subsequent use should be focused at night time when patients naturally hypoventilate. Once successfully weaned from daytime non-invasive ventilation, most patients will require an additional night on non-invasive ventilation. So treatment should last 3–4 days in most cases although some patients will wish to self-wean before this (COPD Concise Guideline Group, 2008).

Invasive ventilation

The failure to improve on non-invasive ventilation usually signals the need for invasive ventilation, which allows precise manipulation of the breathing cycle and higher inspired oxygen and airway pressures if necessary. This, however, comes at the expense of possible ventilator-induced lung injury and an increased chance of sepsis and multi-organ failure.

Notably, patients with chronic respiratory failure are harder to wean from invasive ventilation and are more likely to fail post-extubation. A promising alternative to traditional weaning protocols is the use of non-invasive ventilation to facilitate early extubation, although there are conflicting results as to whether this reduces mortality and reintubation (Esteban et al, 2004; Burns et al, 2006).

A proportion of patients with acute-on-chronic respiratory failure will require chronic ventilation (Consensus Conference, 1999), either nocturnal plus or minus part of the day or full-time. Most often this can be provided non-invasively with a minority requiring tracheostomy ventilation. Patients requiring long-term ventilation from the intensive care unit setting can now be man-

Table 4. Troubleshooting

Persisting hypercapnia	Is chest expansion adequate?	Mask/circuit leak?
		Increase inspiratory positive airway pressure especially if the patient is obese or has a chest wall deformity. Consider decreasing expiratory positive airway pressure to increase tidal volume
	Is the ventilator synchronous with the patient's breathing cycle?	Mask/circuit leak?
		Increase trigger sensitivity if available
		Increase back-up rate to just below spontaneous breathing rate
		If spontaneous breathing rate is rapid then decrease rise/ramp time if available
		If patient has obstructive sleep apnoea then increase expiratory positive airway pressure and consider a nasopharyngeal airway
		In chronic obstructive pulmonary disease there may be intrinsic positive end expiratory pressure so increase expiratory positive airway pressure
	Rebreathing?	Check the exhalation valve is not blocked
	Is medical therapy optimal?	
	Have any complications developed?	
	Airway secretions?	Nose mask with chin strap may allow more effective cough
	Anxiety?	Rarely sedation may be required
	Do they just need longer on non-invasive ventilation?	
Persisting hypoxia	Ensure optimum ventilation	(see persistent hypercapnia)
	Increase expiratory positive airway pressure	Especially in presence of pulmonary oedema or atelectasis
	Increase entrained oxygen	

aged in regional weaning centres which circumvent the disadvantages of remaining in the intensive care unit environment.

Conclusions

Acute-on-chronic respiratory failure is a constellation of clinical and arterial blood gas features that occur when a chronically impaired respiratory muscle pump is pushed beyond its capacity by an acute precipitant. It is an important and increasingly common cause of morbidity and mortality which affects all age groups. The management of acute-on-chronic respiratory failure is often complicated by problems related to the severity and nature of the underlying disease. Ventilatory assistance is usually required and this has been revolutionized by the availability of non-invasive ventilation which offers considerable advantages over invasive ventilation. However, these advantages depend on staff understanding how to optimize non-invasive ventilation and how to recognize when it is failing. There has been a marked improvement in the provision of more comfortable mask interfaces and this development needs to continue so that non-invasive ventilation becomes more acceptable to a wider number of patients. BJHM

Conflict of interest: none.

Ai-Ping C, Lee KH, Lim TK (2005) In-hospital and 5-year mortality of patients treated in the ICU for acute exacerbation of COPD: a retrospective study. *Chest* **128**(2): 518–24

Angus RM, Ahmed AA, Fenwick LJ, Peacock AJ (1996) Comparison of the acute effects on gas exchange of nasal ventilation and doxapram in exacerbations of chronic obstructive pulmonary disease. *Thorax* **51**(10): 1048–50

Anthonisen NR, Manfreda J, Warren CP, Hershfield ES, Harding GK, Nelson NA (1987) Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. *Ann Intern Med* **106**(2): 196–204

Barr RG, Rowe BH, Camargo CA (2003) Methylxanthines for exacerbations of chronic obstructive pulmonary disease. *Cochrane Database Syst Rev* (2)

British Thoracic Society Standards of Care Committee (2002) BTS Guideline: Non-invasive ventilation in acute respiratory failure. *Thorax* **57:** 192–211

Brochard L, Mancebo J, Wysocki M et al (1995) Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. *N Engl J Med* **333**(13): 817–22

Burns KEA, Adhikari NKJ, Meade MO (2006) A meta-analysis of noninvasive weaning to facilitate liberation from mechanical ventilation. *Can J Anaesth* **53**(3): 305–15

Chatwin M, Ross E, Hart N, Nickol AH, Polkey MI, Simonds AK (2003) Cough augmentation with mechanical insufflation/exsufflation in patients with neuromuscular weakness. *Eur Respir J* **21**(3): 502–8

Consensus Conference (1999) Clinical indications for noninvasive positive pressure ventilation in chronic respiratory failure due to restrictive lung disease, COPD, and nocturnal hypoventilation- a concensus conference report. *Chest* **116**(2): 521–34

COPD Concise Guideline Group (2008) Non-invasive ventilation in the management of patients with chronic obstructive pulmonary disease admitted to hospital with acute type II respiratory failure. *Clin Med* (in press)

Davies L, Angus RM, Calverley PM (1999) Oral corticosteroids in patients admitted to hospital with exacerbations of chronic obstructive pulmonary disease: a prospective randomised controlled trial. *Lancet* **354**(9177): 456–60

Diaz GG, Alcaraz AC, Talavera JC et al (2005) Noninvasive positive pressure ventilation to treat hypercapnic coma secondary to respiratory failure. *Chest* **127**(3): 952–60

Dupont M, Gacouin A, Lena H et al (2004) Survival of patients with bronchiectasis after the first ICU stay for respiratory failure. *Chest* **125**(5): 1815–20

Ellafi M, Vinsonneau C, Coste J et al (2005) One year outcome after severe pulmonary exacerbation in adults with cystic fibrosis. *Am J Respir Crit Care Med* **171**(2): 158–64

Elliot MW (2007) Initiating NIV in acute exacerbations of COPD. In: Simonds AK, ed. *Non-invasive Respiratory Support: A Practical Handbook.* 3rd edn. Hodder Arnold, London: 39–56

Esteban A, Frutos-Vivar F, Ferguson ND (2004) Noninvasive positive-pressure ventilation for respiratory failure after extubation. *N Engl J Med* **350**(24): 2452–60

Greenstone M, Lasserson TJ (2003) Doxapram for ventilatory failure due to exacerbations of chronic obstructive pulmonary disease. *Cochrane Database Syst Rev*: (1)

Groenewegen KH, Shols AM, Wouters EF (2003) Mortality and mortality-related factors after hospitalization for acute exacerbation of COPD. *Chest* **124**(2): 459–67

Hanson P, Dive A, Brucher JM, Bisteau M, Dangoisse M, Deltombe T (1997) Acute corticosteroid myopathy in intensive care patients. *Muscle Nerve* **20**(11): 1371–80

Hart N (2008) Respiratory failure. *Medicine* **36**(5): 242–5

Lightowler JV, Wedzicha JA, Elliot MW, Ram FS (2003) Non-invasive positive pressure ventilation to treat respiratory failure resulting from exacerbations of chronic obstructive pulmonary disease: Cochrane systematic review and meta-analysis. *BMJ* **326**(7382): 185–90

McCrory DC, Brown CD (2002) Anticholinergic bronbchodilators versus beta2-sympathomimetic agents for acute exacerbations of chronic obstructive pulmonary disease *Cochrane Database Syst Rev*: (4)

Mokhlesi B, Tulaimat A (2007) Recent advances in obesity hypoventilation syndrome. *Chest* **132**(4): 1322–36

O'Driscoll BR, Howard L, Davison AM (2008) British Thoracic Society Guidelines for emergency oxygen use in adult patients. *Thorax* (in press)

Papi A, Bellettato CM, Braccioni F et al (2006) Infections and airway inflammation in chronic obstructive pulmonary disease severe exacerbations. *Am J Respir Crit Care Med* **173**(10): 1114–21

Plant PK, Owen JL, Elliot MW (2000) One year period prevalence study of respiratory acidosis in acute exacerbation of COPD; implications for the provision of non-invasive ventilation and oxygen administration. *Thorax* **55**(7): 550–4

Roussos C, Koutsoukou A (2003) Respiratory failure. *Eur Respir J* **22**(Suppl 47): 3s–14s

Simonds AK (2007) Acute non-invasive ventilation in neuromuscular disease. In: Simonds AK, ed. *Non-invasive Respiratory Support: A Practical Handbook.* 3rd edn. Hodder Arnold, London: 73–80

Squadrone E, Frigerio P, Fogliati C et al (2004) Noninvasive vs invasive ventilation in COPD patients with severe acute respiratory failure deemed to require ventilatory assistance. *Intensive Care Med* **30**(7): 1303–10

KEY POINTS

- Chronic obstructive pulmonary disease is the commonest cause of acute-on-chronic respiratory failure in the UK.
- Patients may present with worsening dyspnoea, altered mental state or respiratory arrest.
- Acute-on-chronic respiratory failure is most often caused by a respiratory tract infection.
- Management is directed at reversing the acute precipitant while supporting ventilation with either invasive or non-invasive ventilation.
- Supplemental oxygen therapy must be controlled.
- Decide before commencing non-invasive ventilation on how to proceed in the event of failure.