Non-invasive ventilation should be used as an adjunct to other therapies

Non-invasive ventilation in COPD exacerbations

In this article...
- An explanation of the physiology of respiratory failure
- How non-invasive ventilation can treat respiratory failure
- Treatment and monitoring considerations

Chronic obstructive pulmonary disease (COPD) is one of the most common respiratory diseases in the UK; it is most common in people over the age of 35, although most are not diagnosed until they are in their 50s (Department of Health, 2004). The term COPD is used for a collection of lung diseases including chronic bronchitis, emphysema and chronic obstructive airways disease (Plant et al, 2001). It is thought that over 64 million people have the disease worldwide; an estimated three million of these are in the UK, of whom only about 900,000 have been diagnosed (World Health Organization, 2012).

Acute onset of sustained deterioration in respiratory symptoms that are beyond normal day-to-day variations indicate an exacerbation episode (Box 1). This requires a multidisciplinary approach to care involving respiratory physicians, anaesthetists, nurses, critical care outreach and physiotherapists.

Respiratory failure
Respiratory failure (RF) is inadequate gas exchange by the respiratory system whereby levels of arterial oxygen (PaO₂), carbon dioxide (PaCO₂) or both cannot be maintained within the normal ranges. Normal ranges are as follows:
- Oxygen: PaO₂ >80mmHg (11kPa);
- Carbon dioxide: PaCO₂ <45mmHg (6kPa) (Woodrow, 2011).
RF can occur as a result of:
- Lung diseases and conditions affecting the flow of air and blood into/out of the lungs and gas exchange in the air sacs – examples are COPD, pneumonia, acute respiratory distress syndrome, pulmonary embolism and cystic fibrosis;
- Acute lung injuries such as smoke inhalation or pneumothorax;
- Conditions affecting the nerves and muscles that control breathing – these can include muscular dystrophy, spinal cord injuries, Guillain-Barré syndrome, and stroke;
- Spinal problems such as scoliosis (curvature), which can affect the bones and muscles used for breathing;
- Damage to the tissues and ribs located around the lungs, for example due to a chest injury;
- Drug or alcohol overdose – this can affect the area of the brain that controls respiration, leading to slow and shallow breathing.

The condition can be classified into type 1 or type 2 RF, relating to the absence or presence of hypercapnia (high levels of CO₂ in the blood) respectively:
- Type 1 is defined as PaO₂ <8kPa with a normal or low PaCO₂;
- Type 2 is defined as PaO₂ of <8kPa and a PaCO₂ of >6kPa (Woodrow, 2011).

Non-invasive ventilation can be extremely effective in treating acute type 2 respiratory failure in patients with exacerbations of chronic obstructive pulmonary disease.
Life-threatening ventilatory failure is characterised by the presence of respiratory acidosis, in which arterial pH falls to <7.35 due to either type 1 or type 2 RF.

Type 1 RF is caused by damage to lung tissue, which prevents adequate oxygenation of the blood (hypoxaemia); however, as CO₂ excretion requires less functioning lung tissue than is needed for oxygenation, the remaining normal lung is still sufficient to excrete the CO₂ produced by tissue metabolism.

Type 2 RF occurs when alveolar ventilation is insufficient to excrete the CO₂ being produced. Inadequate ventilation is due to reduced ventilatory effort, or inability to overcome increased resistance to ventilation – the condition affects the lung as a whole and causes CO₂ to build up. Complications include:

- Damage to vital organs due to hypoxaemia;
- Central nervous system depression due to increased CO₂ levels;
- Respiratory acidosis (CO₂ retention).

Type 2 RF is ultimately fatal unless treated; non-invasive ventilation (NIV) using bilevel positive airway pressure (BiPAP) is used for this purpose.

**Oxygen therapy in COPD**

Some clinicians are concerned that prescribing oxygen to patients with COPD who are retaining CO₂ will increase hypercapnia. However, any evident hypoxia must be treated with oxygen as a lifesaving intervention (O’Driscoll et al, 2008).

Patients who are, or are suspected of, retaining CO₂ should be given oxygen to achieve a target saturation of 88-92%. Those who do not retain CO₂ should be given oxygen to achieve target saturations of 94-98% (National Institute for Health and Clinical Excellence, 2010). Some patients with severe COPD may have lower oxygen saturation levels than those mentioned and should be assessed by a respiratory consultant.

**Arterial blood gases**

Optimising gaseous exchange in patients with an exacerbation of COPD requires careful monitoring of both oxygen and CO₂ levels using arterial blood gas (ABG) analysis as the gold standard. In order to achieve this it is important to understand normal blood gas values (Box 2) and how they relate to maintaining homeostasis.

Normal cellular metabolism requires a blood pH of 7.35-7.45 (Woodrow, 2011). A normal pH is regulated by respiratory and renal pathways and can respond within hours to changes in blood pH. The respiratory rate is the main determinant of CO₂ excretion by the lungs and, therefore, the amount in the blood.

Normal cellular metabolism results in continuous production of hydrogen ions (H⁺). The kidneys eliminate these ions and regenerate bicarbonate, providing a buffer to any acid production; they effectively “mop up” hydrogen ions. Renal regulation involves altering the bicarbonate reabsorption and is much slower than the respiratory pathway (over a period of days or weeks). Bicarbonate and base excess gives an indication of any metabolic (renal) compensation.

Up to 20% of patients admitted to hospital with an acute exacerbation of COPD will present with respiratory acidosis (elevated PaCO₂) (Plant et al, 2001). When CO₂ combines with water in the red blood cells it becomes carbonic acid (H₂CO₃); this causes the blood pH to fall, leading to rapid deterioration. In trying to compensate, respiratory chemoreceptors in the brain raise the respiratory rate to try to excrete more CO₂.

However, as the patient tires and the respiratory rate falls, the CO₂ level rises and the patient will develop type 2 RF. This is known as uncompensated respiratory failure.

The hallmark of life-threatening ventilatory failure in the uncompensated form is characterised by the presence of respiratory acidosis when arterial pH falls below 7.35 (Woodrow, 2011).

It is important to take a systematic approach to analysing ABGs in order to ensure appropriate intervention (Box 3).

**Pharmacological management of exacerbations**

It is crucial that the cause of RF is established and treated with maximal medical therapy. This should be undertaken for no more than one hour before NIV BiPAP is commenced (when indicated) (NICE, 2010). Pharmacological management may include:

- Controlled O₂ to maintain SaO₂ 88-92%;
- Nebulised salbutamol 2.5-5mg;
- Nebulised ipratropium 50ug;
- Prednisolone 30mg orally for 7-14 days;
- Antibiotics if infective cause suspected (NICE, 2010).

**Principles of acute NIV BiPAP therapy**

A number of studies have identified the effectiveness of NIV in patients with an acute exacerbation of COPD, complicated by type 2 RF (Quon et al, 2008; Royal College of Physicians et al, 2008; Lightowler et al, 2000; Plant et al 2001). This has been one of the major technological advances in respiratory management in the last decade. NICE recommends that NIV be made available in all hospitals admitting patients with COPD – over 90% of hospitals in the UK now offer this intervention (NICE, 2010).

**How does NIV work?**

Non-invasive positive pressure ventilation delivers intermittent positive airway pressure (PAP), which gives the patient ventilatory support using a nose or nasal mask. This is achieved through a pressure-cycled machine known as BiPAP. The higher level of pressure assists ventilation during...
**BOX 4. INCLUSION AND EXCLUSION CRITERIA FOR NIV BiPAP**

**Inclusion criteria**
- Primary diagnosis of COPD
- Ability to protect airway
- Conscious and cooperative
- Potential for recovery
- Complies with patient wishes

**Exclusion criteria**
- Severe comorbidity
- Severe cognitive impairment
- Facial burns/truma/surgery
- Vomiting
- Fixed upper airway obstruction
- Undrained pneumothorax
- Inability to protect airway
- Upper gastrointestinal surgery
- Copious respiratory secretions
- Patient moribund
- Bowel obstruction
- Life-threatening hypoxemia
- Haemodynamic instability

*NIV may still be appropriate if escalation to higher level of care is not in patient’s best interests and NIV is the “ceiling” of treatment*


**BOX 5. MONITORING PATIENTS**

The following should be undertaken when monitoring patients receiving NIV:
- Baseline observations – respiratory rate, heart rate, arterial blood gases (ABGs)
- Continuous pulse oximetry and electrocardiogram within the first 12 hours
- Repeat ABGs after one hour of starting NIV, then one hour after any change in settings, and then after four hours (or earlier if patients are not improving clinically)
- Hourly physiological observations including respiratory rate, heart rate, conscious level, chest-wall movement, accessory muscle usage, patient comfort, and compliance, mask fit and mask leak

*A multidisciplinary team should also decide whether invasive ventilation will be offered if NIV BiPAP fails and whether resuscitation should be attempted in the event of a cardio-respiratory arrest. This must be documented in the patient’s case notes.*

**Settings**

The patient should be in a sitting or semi-recumbent position before commencing NIV BiPAP. It is important the patient can tolerate initial settings to ensure concordance and positive response. Recommended initial settings are:
- 10cm H₂O for IPAP
- 4-5cm H₂O for EPAP

Titration of IPAP should be undertaken in 2.5cm increments at a rate of 5cm H₂O every 10 minutes – the usual maximum target pressure is 20cm H₂O.

If a patient remains hypoxaemic oxygen can be entrained into the NIV circuit and flow adjusted to maintain target saturations, usually 88-92%. EPAP can also be increased to a maximum of 6cm H₂O.

**Treatment failure**

Initial response to NIV and the severity of RF at presentation are key prognostic indicators: rapid improvements of pH, PaCO₂, PaO₂ and respiratory rate in the first 1-4 hours of treatment have been shown to be important indicators of success. Treatment failure is likely if there is:
- Worsening of the pH and/or respiratory rate after four hours of BiPAP therapy
- Failure to tolerate BiPAP
- Deterioration in Glasgow Coma Scale
- Hypotension
- Uncorrected hypoxaemia; or
- No change in both pH and respiratory rate at four hours.

Treatment failure occurs in 20-40% of patients, so it is vital to make a management plan of what to do in case of failure before treatment is initiated (Confalonieri et al, 2005).

Treatment is likely to be successful if there is improving pH, respiratory rate, PaCO₂ and PaO₂ at four hours of acute NIV BiPAP therapy.

**Duration of treatment and weaning**

The duration of BiPAP can vary depending on response to therapy, resuscitation...
status and tolerability:

- For patients who are deemed suitable for invasive ventilation but fail to respond to initial NIV BiPAP therapy, an urgent decision to proceed to invasive ventilation should be made by a senior medical officer after the first four hours.

- For patients who are not candidates for invasive ventilation, BiPAP is effectively the ceiling treatment; it can therefore be extended for as long as it is tolerated or discontinued if it is considered to be a futile measure.

- Patients can come off BiPAP temporarily – for example to receive drug therapy, physiotherapy, meals and fluids – but controlled oxygen therapy should be continued while they are not on the BiPAP machine. This is generally via a nasal cannula or a venturi device; the target saturation is >88%.

- If treatment is successful, the recommended duration is 48–72 hours, with the patient on the machine for as long as possible on day one, 16 hours on day two and 12 hours on day three. Treatment should be discontinued on day four.

Weaning from BiPAP is based on clinical observations, pulse oximetry and ABG results. No single set of parameters can predict weaning success but it is recommended that the following clinical parameters be taken into account:

- ABGs return to within normal parameters for the patient;
- Oxygen saturation >95% on air or 24–28% oxygen delivered by a venturi mask;
- Respiratory rate is <25 breaths per minute;
- No use of accessory muscles;
- Able to talk in full sentences.

There are several ways in which to wean from BiPAP, depending on the patient’s clinical condition. In some instances it can simply be discontinued, but in others it may be possible to discontinue it for only 2–3 hours at a time while monitoring the patient’s clinical condition. Some patients may need BiPAP overnight while remaining off ventilatory support during the day. If a patient being weaned begins to show signs of respiratory distress or the level of oxygen saturation is <88% on low concentrations of supplemental oxygen, BiPAP should be reinstated.

**Conclusion**

Non-invasive BiPAP can be lifesaving to patients with an acute exacerbation of COPD, leading to type 2 respiratory failure. However, an ongoing management plan must be in place before treatment starts. Regular physiological observations and ABG interpretation must be accompanied by timely intervention to maximise the benefits of this intervention. Thorough nursing assessment is vital to maintain the safety of these patients.

Studies have shown non-invasive ventilation also improves the quality of life of patients with chronic RF treated in the community [Ali and Kabir, 2007; Tuggey et al, 2003]. An understanding of NIV is therefore useful for practice and community nurses, both when reviewing patients after discharge from hospital and when considering NIV as a community-based treatment.

**References**


**BOX 6. CASE STUDY**

Donald Jones was admitted to a respiratory ward complaining of feeling more short of breath than usual and coughing up green phlegm. He was very drowsy and was diagnosed as having an infected exacerbation of chronic obstructive pulmonary disease (COPD). His arterial blood gas results were:

- pH 7.21
- PaCO2 12.7kPa
- PaO2 7.1kPa
- Base excess 2.1 mEq/L

These results suggest acute respiratory acidosis and can be defined as type 2 respiratory failure (pH low, PaO2 low with elevated PaCO2).

After treatment with nebulisers, steroids and controlled oxygen, it was felt Mr Jones would benefit from non-invasive ventilation using bilevel positive airway pressure (NIV BPAP). Mr Jones had had a number of COPD exacerbations and his quality of life was diminished. He used home oxygen 2L/16hrs per day. The multidisciplinary team felt if NIV was unsuccessful, invasive ventilation would be of no benefit. Following a discussion with Mr Jones and his family, it was decided that NIV was the ceiling of treatment and a “do not attempt cardiopulmonary resuscitation” was put in place.

NIV BPAP was commenced with an inspiratory positive airway pressure (IPAP) of 10cm H2O and an expiratory positive airway pressure (EPAP) of 4cm H2O. Mr Jones tolerated the mask well. Supplemental oxygen was administered at 2L to maintain his SaO2 at 88-92%. After one hour his arterial blood gases were repeated; they showed:

- pH 7.26
- PaCO2 10.3kPa
- PaO2 8.9kPa
- BE: 2.0mEq/L

While some improvement was noted (pH less acidic, PaCO2 reduced, PaO2 increased), Mr Jones remained in type 2 respiratory failure. The NIV was increased to IPAP 12 and EPAP 5 to help to remove harmful CO2 (IPAP) and to oxygenate (EPAP). Regular ABGs were taken and immediate action was taken on the results.

Mr Jones required NIV BPAP for two days; he was discharged home after eight days.

*The patient’s name has been changed*