Clinical guidelines
for non-invasive ventilation in
acute respiratory failure.

Summary:
Non-invasive ventilation (NIV) is increasingly being considered as a treatment option in acute respiratory failure. This guideline reviews some of the common clinical conditions where NIV may be considered. The evidence (or lack of) for the use of NIV under such circumstances is discussed. Some of the contraindications to NIV are discussed. A series of simple flow diagrams are introduced to guide clinical carers in the use of NIV. A similar series of flow diagrams are also introduced to help carers wean their patients from NIV.
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Introduction

Non-invasive ventilation (NIV) is an increasingly popular treatment option in varied clinical situations in patients presenting with acute respiratory failure (Caples & Gay 2005). NIV differentiates itself from other techniques which bypass the upper airway with a tracheal tube, laryngeal mask or tracheostomy – the invasive forms of ventilation. The advantages of NIV relate to the disadvantages of invasive ventilation – the potential for upper airway trauma, ventilator-associated pneumonia, impaired speech and swallowing and the relatively high costs and resource utilisation. NIV should complement the use of invasive ventilation and should not be regarded as its replacement. Careful patient selection and regular, well-timed bedside clinical assessments are keys to success. Failure of NIV should be recognised early as it can only delay more definitive therapy with invasive ventilation.

Definitions

Respiratory failure is defined as a failure to maintain adequate gas exchange. This manifest itself as abnormalities in arterial blood gas tensions. Type 1 failure is defined by a $P_{aO_2} < 8$ kPa and a normal or low $P_{aCO_2}$ (≤5 kPa). Type 2 failure is defined by a $P_{aO_2} < 8$ kPa and a $P_{aCO_2} > 6.5$ kPa (BTS 2016).

In acute hypercapnic respiratory failure, the arterial blood gas tensions will show a high $P_{aCO_2}$, low pH (pH < 7.35) and normal bicarbonate levels.

In chronic hypercapnic respiratory failure, the arterial blood gas tensions will show a high $P_{aCO_2}$, normal pH (pH 7.35 – 7.45) and high bicarbonate levels.

In acute-on-chronic hypercapnic respiratory failure, the arterial blood gas tensions will show a high $P_{aCO_2}$, low pH (pH < 7.35) and high bicarbonate levels.

Therefore, in clinical use, it is usually the low pH and not an elevated $P_{aCO_2}$ that determines the presence of acute respiratory failure and the need to consider NIV. An understanding of arterial blood gases is vital to the provision and management of patients with respect to NIV.
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NIV is defined as respiratory support delivered via a non-invasive interface – this is typically a face mask or nasal mask. However, other less common interfaces may be deployed e.g. nasal plugs/pillows, oral mouthpieces and full head helmets. Respiratory support may be delivered using continuous positive airway pressure (CPAP) devices or those that deliver bi-level positive airway pressure (BiPAP\(^\text{\textdegree}\)). For the purposes of this document, NIV includes both CPAP and BiPAP.

CPAP is often likened to breathing with your head stuck out of a moving car. It aims to deliver a continuous, single positive pressure throughout both the inspiratory and expiratory phases of breathing. It improves oxygenation by opening up collapsed airways, improving functional residual capacity (FRC) and improving preload and afterload in cardiogenic pulmonary oedema (Bersten et al 1991, Lin et al 1995). CPAP may also help by reducing the efforts required for breathing by improving lung compliance by preventing alveolar collapse (liken to the fact that to blow up a balloon that is already partially inflated is easier than a balloon that is collapsed) and by counteracting against the excessive intrinsic PEEP (positive end expiratory pressure) seen in obstructive lung conditions such as chronic obstructive pulmonary disease (COPD) (BTS 2016).

BiPAP (BTS 2016) aims to deliver two levels of positive airway pressure support. The lower level is similar to CPAP although it is more commonly called expiratory positive airway pressure (EPAP) as it is present only at the expiratory phase of breathing. The higher level of pressure is present at inspiration and is called the inspiratory positive airway pressure (IPAP). This higher level of pressure is triggered when the machine senses the patient’s inspiratory effort and aims to assist inspiration. The size of the breath (tidal volume) generated in a particular patient is dependent on the difference between EPAP and IPAP settings – the larger the difference between EPAP and IPAP settings, the larger the pressure difference between expiration and inspiration, resulting in a larger breath. In spontaneous mode (S Mode), the cycling from IPAP to EPAP and back to IPAP is under total patient control and is synchronised with the patient’s own inspiratory and expiratory cycles. In timed mode (T Mode), the cycling from…
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the different pressure levels is independent of the patient and is dependent on the number of breaths set. The two modes may be combined (S/T Mode) where the patient’s breathing is ventilator-assisted (S Mode) – if the patient does not breath a set minimum number of breaths per minute, the ventilator will supply those additional breaths (T Mode). It is envisaged that the S/T Mode will be the appropriate mode of ventilation under most clinical circumstances.

Indications

The indications for NIV depend upon the goals of therapy in the patients presenting with acute respiratory failure. These may be improving gas exchange resulting in enhanced oxygenation, better carbon dioxide elimination and normalising acidaemia; reducing cardiac workload and improving haemodynamics; unloading respiratory muscles, thereby decreasing respiratory rate and improving patient comfort and avoidance of invasive ventilation (BTS 2016). NIV may be used:

1. As an early intervention to assist ventilation in order to prevent the development of acute respiratory failure. This is usually at an earlier stage than that at which invasive ventilation would be considered.
2. As a trial with a view to tracheal intubation if NIV fails.
3. As the maximum ceiling of treatment in patients deemed inappropriate for tracheal intubation.

A decision about intubation must be made in all patients commenced on NIV. If at all possible, this must involve a discussion between the clinician and the patient. This should be done early, ideally prior to starting NIV, but may be delayed to allow for consultations with relatives, carers and other health professionals. In practice, the delay should be no longer than 4 hours in most cases (BTS 2016). The decision should be made by an appropriately experienced clinician and agreed upon by all the patient’s carers. The decision regarding intubation may be reviewed as necessary and changed if appropriate. The “do not attempt cardiopulmonary resuscitation” (DNACPR) status is a different issue altogether and a decision regarding the DNACPR status of a
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Patient must be made separate to that of the intubation status. A DNACPR order does not necessarily preclude a trial of NIV. Best practice is expected in terms of cardiovascular and fluid optimisation, management of any suspected infection, pain and distress management, prophylaxis against deep venous thrombosis and the provision of adequate nutrition.

Patient selection

A key to successful NIV is careful stratification, selection and matching of patients to the most appropriate treatment modality (BTS 2016, Caples 2005).

Acute exacerbations of chronic obstructive pulmonary disease (COPD)

This group of patients are the most frequently studied in trials of NIV. When they present with acute respiratory failure, arterial blood gas analysis shows an acute respiratory acidosis with a moderate degree of hypoxaemia. There is good evidence to support the use of BiPAP in this group, particularly in those whose blood pH is between 7.26 to 7.35, in terms of lowering the rates of tracheal intubation and subsequent mortality (Bott et al 1993, Brochard et al 1995, Plant et al 2000). Reduction in mortality rates up to 50% have been reported (Ram et al 2004), with a NNT of approximately 10. Guidelines for the management of acute hypercapnic respiratory failure during acute exacerbations of COPD endorsed by the British Thoracic Society, The Royal College of Physicians London and the Intensive Care Society were published in 2008 – these have recently been revised by the British Thoracic Society/Intensive Care Society and endorsed by the Royal College of Physicians, London, The Royal College of Emergency Medicine and The Royal College of Anaesthetists (BTS 2016). They suggested that BiPAP should be considered in all COPD patients with a persisting acidosis after standard medical therapy (30mg prednisolone, 2.5 – 5 mg nebulised salbutamol, 0.5 mg nebulised ipratropium, controlled oxygen targeted to maintain S\text{pO}_2 between 88 – 92%, antibiotics if indicated) given in the first one hour. Patients presenting with severe acidosis (pH < 7.25) should have NIV started as part of their initial
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treatment. Patients with COPD presenting with acute hypercapnic respiratory failure but with signs of consolidation on chest radiograph should be considered to be in pneumonia group (see below) – the benefits of NIV in these patients are considerably less (Honrubia et al 2005).

There is no good evidence that CPAP is beneficial in this group. The benefits of BiPAP may have rendered the use of CPAP in this group an irrelevance (BTS 2016). However, a trial of CPAP may be justified with some patients in this group when BiPAP is unavailable (Miro et al 1993).

**Hypoxaemic respiratory failure/pneumonia (non-COPD)**

Historically, this group of patients have been thought to be poorly responsive to NIV (Wysocki et al 1995, Honrubia et al 2005). However, current evidence suggests that BiPAP can reduce the rate of tracheal intubation, reduce ICU stay and increase survival rate (Ferrer et al 2003, Keenan et al 2004). CPAP is now increasingly being used to support immunocompromised patients with opportunistic chest infections (Hilbert et al 2001, Confalonieri et al 2002). In this situation, CPAP can improve oxygenation, reduce respiratory rate and lessen dyspnoea. Therefore, a trial of NIV may be warranted in the patient with single organ (respiratory) failure (Antonelli M et al 1998, Caples & Gay 2005). Possibly more than the others, this group of patients needs to be monitored closely for failure of treatment as continuing hypoxaemia can lead to rapid cardiovascular collapse and arrest. If the patient is considered appropriate for tracheal intubation in the event failure of NIV, then the ideal environment to care for such a patient should be one that can facilitate this treatment (Garpestad & Hill 2005).

Tracheal intubation should be considered in the face of deteriorating hypoxaemia despite NIV, the failure to achieve acceptable gas exchange indices within 2 hours or if there is instability in other organ systems. Delay in tracheal intubation can lead to an overall poorer outcome for the patient (Keenan et al 2004).
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**Cardiogenic pulmonary oedema**
CPAP has been associated with a lower intubation rate and a survival benefit in this group of patients (Bersten et al 1991, Lin et al 1995, Peter et al 2006). Some trials with BiPAP in patients presenting with acute pulmonary oedema had shown an excess of acute myocardial infarctions when compared to CPAP (Mehta et al 1997, Rusterholtz et al 1999) – criticisms have been made of a compromised randomisation procedure resulting in more patients with chest pain being allocated to the BiPAP group (Capes & Gay 2005). Furthermore, the use of BiPAP for the treatment of acute pulmonary oedema in the accident and emergency department appeared to be associated with higher hospital mortality when compared with CPAP (Crane et al 2004). A recent meta-analysis supports the use of both CPAP and BiPAP in reducing the need for invasive ventilation when compared with standard medical management. Furthermore, there was no evidence of mortality differences between CPAP and BiPAP (Peter et al 2006). The recent Three Interventions in Cardiogenic Pulmonary Oedema (3CPO) Trial (Gray et al 2008) showed no excess of acute myocardial infarctions in patients receiving BiPAP over CPAP. Both NIV modalities were equally effective and achieved better results at 1 hour in dyspnoea scores, heart rate, acidosis and hypercapnia when compared with standard oxygen therapy. However, there were no differences in intubation rates and 7-day mortality between the NIV and the standard oxygen groups. Therefore, in acute cardiogenic pulmonary oedema, NIV may be used as an adjunct with standard pharmacologic treatment especially in the presence of severe respiratory distress - CPAP should be the support of choice and BiPAP should only be used when CPAP fails or in the presence of acute hypercapnia (Peter et al 2006).

**Asthma**
Although BiPAP has been used successfully to support and reduce the rate of tracheal intubations in patients admitted with status asthmaticus and acute respiratory failure (Meduri et al 1996, Soroksky et al 2003), there is little justification in recommending its routine use, particularly as the mortality rates
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of intubation and invasive ventilation in this group is low (BTS 2016). Therefore, the use of NIV in acute severe asthma is not generally recommended. Some patients with chronic asthma with have symptoms of COPD – asthma COPD Overlap Syndrome (ACOS) – may present to hospital with signs of respiratory failure. This group responds well with NIV and should be managed as though they have an acute exacerbation of their COPD and along the AECOPD pathway (BTS 2016).

Chest wall deformity/neuromuscular disease
There are reports of good outcomes with the use of BiPAP in decompensated ventilatory failure in this group of patients (Elliott et al 1990). Without domiciliary NIV, the natural history of patients with neuromuscular disease (NMD) and chest wall deformity (CWD) is of progressive chronic hypercapnic failure leading to death. It is well recognised such individuals can survive long term on home NIV with a good quality of life, even if they present initially in severe respiratory failure. Thus, individuals with NMD and CWD who present with acute respiratory failure should not be denied acute NIV. The decision to use BiPAP will need to take into account pre-existing co-morbidities, the severity of ventilatory failure and the presence or absence of bulbar involvement. (BTS 2016). Patients in this group who are acutely unwell and present with hypercapnia but without acidosis should be considered for early BiPAP, as this may herald an impending crisis with rapid deterioration (Bourke et al 2006). Early involvement of senior staff and discussion with the patients and their families is essential. The presence of bulbar dysfunction may make the delivery of BiPAP more difficult due to the need to overcome the increased upper airway resistance – a higher EPAP may be needed. Experienced physiotherapy input is essential to ensure adequate sputum clearance. There is no evidence for the use of CPAP in this group.

Chest trauma
There is some evidence to support the use of CPAP in patients with isolated chest trauma and mild to moderate hypoxaemia (Hurst et al 1985, Bollinger &
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Van Eden 1990). Therefore, a trial of CPAP may be indicated in patients who remain hypoxic despite adequate analgesia and high flow oxygen. These patients must be adequately monitored for the possibility of developing a pneumothorax with CPAP (BTS 2016).

Postoperative respiratory failure
There is evidence that the early use of CPAP for the treatment of hypoxaemic respiratory failure following uncomplicated abdominal surgery can reduce the need for tracheal intubation (Squadrone et al 2005). BiPAP has been used with similar results following lung surgery (Auriant et al 2001).

Weaning from invasive ventilation
There is some evidence that patients intubated following an acute exacerbation of COPD may benefit from early extubation to NIV (Hilbert et al 1998, Nava et al 1998). A Cochrane review suggested that the use of NIV in the weaning process in this group of patients reduced mortality and incidence of pneumonia without the increasing the need for re-intubation (Burns et al 2014) and is to be recommended (BTS 2016). The evidence for the use of NIV as an adjunct to facilitate weaning from invasive mechanical ventilation in other groups is conflicting (Hilbert et al 1998, Nava et al 1998, Kilger et al 1999, Keenan et al 2002, Esteban et al 2004). Delays in reintubation may be responsible for some of the adverse results in some trials (Keenan et al 2002, Caples & Gay 2005).

Predictors of outcome of NIV
There is no one good objective predictor of NIV success available. Stratification of patients into the groups as outlined above and careful matching of patient to treatment modality can improve success rates (Caples & Gay 2005). Predictors of success include those with mild to moderate hypercapnia and acidaemia (pH 7.25 – 7.35) with mild hypoxaemia, a good level of consciousness and rapid improvements (<2 hours) to physiological parameters following the start of NIV (BTS 2002, Mehta & Hill 2001). Factors associated with a poorer response to NIV includes the more severely sick patient, the presence of pneumonia on
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chest radiograph, poor conscious level (Glasgow coma score <11), respiratory rate >30 breaths per minute, copious respiratory secretions, poor nutritional status and poor fitting of the face mask (Ambrosino et al 1995, BTS 2016, Confalonieri et al 2005). Defining the goals of NIV at the start can help target treatment and can also identify the patient in whom NIV is failing so that more definitive treatment such as invasive ventilation can be considered (Caples & Gay 2005).

Contraindications to NIV

The clinical conditions for which NIV is being used continue to increase. There are no absolute contraindications to the use of NIV (BTS 2016) but there are some conditions where the use of invasive ventilation may be preferable.

Invasive respiratory support may be preferable in:

- Recent facial trauma/burns including recent extensive facial surgery where the fitting of the interface may be compromised, painful or compromise the underlying surgery.
- Recent base of skull fracture with continuing CSF rhinorrhoea.

Although not contraindicated, careful consideration of the risk-benefit ratio must be taken in the following conditions prior to starting NIV. In patients for whom invasive intubation are deemed inappropriate, then NIV may be suitable in spite of the presence of these problems.

- Recent upper airway or upper gastrointestinal tract surgery.
- Fixed obstruction of the upper airway.
- Inability to protect the airway.
- Life threatening hypoxaemia.
- Haemodynamic instability.
- Severe co-morbidities.
- Impaired conscious levels.
- Confusion or agitation.
- Vomiting.
- Bowel obstruction.
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- Copious secretions.

In the patient with recent upper airway or upper gastrointestinal (particularly oesophageal) surgery, the use of NIV must be discussed with the surgical team (upper gastrointestinal, ENT, oro-maxillofacial or neurosurgery) involved prior to its commencement.

In patients with a severe respiratory acidosis (pH < 7.2) associated with type 2 respiratory failure, BiPAP may be started in conjunction with the appropriate medical therapies. If further escalation of support is deemed appropriate in the event of failure of NIV, then it is recommended that the Intensive Care Unit be contacted early. Close monitoring and frequent analyses of blood gases are recommended in the early stages as a failure to show rapid improvements may be an early sign that NIV may not be ultimately successful.

An undrained pneumothorax by itself is not a contraindication to NIV. However, under most circumstances, it would be expected that an intercostal pleural drain will be inserted before the start of NIV (BTS 2016). In the event that NIV is started without the prior insertion of an intercostal drain, the patient must be monitored closely for an expanding pneumothorax and personnel and equipment must be immediately available should it become necessary to do so.

Monitoring

Clinical monitoring (BTS 2016) is essential and is not replaced by physiological monitoring. Clinical monitoring should include looking at the coordination of the patient’s respiratory efforts with the ventilator, the degree of chest expansion, respiratory rate and its trend, heart rate and its trend, patient comfort and mental state and a clinical examination of the chest. The fitting of the chosen interface and the degree of air leak should also be noted.

All patients on NIV should have continuous ECG, automated BP set to appropriate intervals and continuous pulse oximetry monitoring as a minimum. The alarm limits for the monitors should be appropriately set. Arterial blood gas analysis should be performed an hour after the start of NIV – earlier, if clinically indicated. Arterialised capillary blood gas measurements (Pitkin et al 1994) may be used as a surrogate to arterial blood gas analysis, particularly if it has been
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referenced to an earlier arterial blood gas measurement. The use of an indwelling arterial cannula is not essential but may be more likely in the HDU/ICU setting.

Treatment failure

The conclusion that NIV has failed depends on the objectives set at the start of the treatment. Some factors to consider when arriving at such a conclusion may be a general deterioration in the patient’s condition, inability improve the respiratory failure and normalise oxygenation and carbon dioxide elimination, intolerance and inability to coordinate respiration with that of the ventilator, the development of new symptoms or complications and a wish to withdraw treatment. Care must be taken that all the NIV settings and the fitting of the interface are optimised (BTS 2016). A management plan in the event of NIV failure will usually have already been made and this should be commenced.

Withdrawal/weaning of NIV

One of the advantages of NIV is that breaks can be instituted for meals, physiotherapy and other activities. In the first 24 hours of treatment, the patient should be ventilated for as long as possible and for as long as tolerated (Kramer et al 1995). Thereafter, the decision to wean NIV will be made based upon the assessment of the general improvements and stability of the patient’s condition. A good sign is when a patient independently decides to stop the use of NIV. Generally, weaning of NIV should be in a stepwise fashion, reducing daytime ventilation before night-time ventilation (Brown et al 1998), with the rate of withdrawal based upon the preservation of favourable clinical and physiological parameters such as the patient’s general condition, respiratory rate, heart rate, mental state and indices of gas exchange.

In the event that NIV is being withdrawn due to failure to improve and the patient is deemed unsuitable for further escalation of treatment, then the stepwise method may be omitted. An assessment of palliative needs must be made prior to stopping NIV. The decision to start end-of-life care does not require the
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withdrawal of NIV – NIV may still be a reasonable component within the end-of-life care package (Shee & Green 2003). Under such circumstances, all relevant and involved parties must understand that the continuation of NIV may prolong the terminal illness. However, usual practice would be to withdraw NIV and to manage any symptoms of breathlessness with opioids and benzodiazepines.
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NIV initiation pathway

Respiratory failure despite optimisation of medical support?

Optimise/continue medical support.
- Controlled oxygen.
- Regular and frequent nebulised bronchodilators – prescribed and administered.
- Steroids if indicated.
- Antibiotics if indicated.
- Full treatment of cardiac failure (diuretics, nitrates).
- Effective analgesia if indicated.

Assessed by/discussed with experienced/senior doctor or ICU. No contraindications to NIV. Agreed management plan in case of NIV failure.

Acute type 2 respiratory failure or at risk of acute type 2 respiratory failure.
(pH < 7.35, $P_{aO_2} < 8$ kPa, $P_{aCO_2} > 6.5$ kPa)

BiPAP

Acute type 1 respiratory failure or at risk of acute type 1 respiratory failure.
(pH > 7.35, $P_{aO_2} < 8$ kPa, $P_{aCO_2} < 6.5$ kPa)

CPAP
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**CPAP pathway**

Start CPAP using body weight as a guide:
- < 60 Kg: 5 cm H₂O pressure/CPAP valve
- 60 – 90 Kg: 7.5 cm H₂O pressure/CPAP valve
- > 90 Kg: 10 cm H₂O pressure/CPAP valve

Appropriate oxygen to maintain SpO₂ > 92% (88% - 92% in patients known to be sensitive to oxygen).

Minimum monitoring:
- Continuous ECG and pulse oximetry
- Automated non-invasive BP
- Full EWS observations

Blood gas analysis in 1 hour or earlier if indicated.

Improvements in clinical parameters (e.g. respiratory rate, heart rate, mental state) and/or physiological parameters (blood gas analysis)?

- Yes
  - **Continue and repeat blood gas analysis in 2 hours or earlier if indicated.**

- No
  - Type 2 respiratory failure or at risk of Type 2 respiratory failure (PₐO₂ < 8 kPa, PₐCO₂ > 6 kPa) or patient tiring
    - No
      - Increase oxygen by 10% and/or CPAP by 2.5 cm H₂O increments.
        - Perform blood gas analysis 1 hour after each intervention or earlier if indicated.
        - Inform ICU if CPAP > 10 cm H₂O; maximum CPAP 15 cm H₂O.
        - Ask for ICU/experienced help early if requiring increasing levels of support without improvements.
        - Senior help must be sought when maximum support levels are reached.
    - Yes
      - Consider BiPAP.
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**BiPAP pathway**

- Commence BiPAP on S/T mode, IPAP 15 cm H₂O (20 cm H₂O if pH<7.25), EPAP 3 cm H₂O, 12 backup breaths, inspiratory time 1.6s, rise time 0.1s.
- *Increase IPAP by 2-5 cm H₂O every 5-10 minutes, until usual target pressure of 20 cm H₂O or a good response is achieved or to limits of patient tolerance.*
- Appropriate oxygen to maintain SpO₂ 88% - 92%.
- *Ventilators that supplement oxygen using a flowmeter CANNOT provide more than 50% inspired oxygen.*

**Minimum monitoring:**
- Continuous ECG and pulse-oximetry
- Automated non-invasive BP
- Full EWS observations
- Blood gas analysis in 1 hour or earlier if indicated.

**Improvements in clinical parameters (e.g. respiratory rate, heart rate, mental state) and/or physiological parameters (blood gas analysis)?**

- **Yes**
  - Continue and repeat blood gas analysis in 2 hours or earlier if indicated.

- **No**
  - Check:
    - Medical therapy optimum & has been given?
    - Complications (pneumothorax, gastric aspiration)?
    - Interface fitting well and circuit correctly set up?
    - Ventilation (chest expansion, minute/tidal volumes) adequate?
    - Good synchronisation with ventilator?

  - Correct and/or treat.

  - **Yes**
    - Improvements in clinical parameters (e.g. respiratory rate, heart rate, mental state) and/or physiological parameters (blood gas analysis)?

  - **No**
    - Continue and repeat blood gas analysis in 2 hours or earlier if indicated.

**PₐCO₂ high** – increase IPAP by 2 cm H₂O increments. Consider increasing respiratory rate.  
**PₐO₂ low** – increase oxygen by 10% or 4L/min increments. Consider increasing EPAP by 2 cm H₂O increments – *IPAP must be increased by a similar amount.*

Maximum levels of support are IPAP 30 cm H₂O, EPAP 10 cm H₂O, 90% oxygen, respiratory rate 20 bpm.

- **Ask for ICU/experienced help early if requiring increasing levels of support without improvements.**
- **Senior help must be sought when maximum support levels are reached.**
- **If appropriate, consider intubation early, especially if there are no improvements after 1-2 hours, continuing deterioration, or persistent hypoxaemia.**
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CPAP Weaning Pathway

Pre-conditions:
- Primary illness treated or under medical control.
- Targets in clinical and physiological parameters (respiratory rate, heart rate, mental state, blood gas parameters) achieved and stable for ≥ 12 hours.
- CPAP ≤ 10 cm H₂O pressure/CPAP valve.

Reduce CPAP by 2.5 cm H₂O every 4-6 hourly. Minimum CPAP = 5 cm H₂O pressure/CPAP valve.

Stable clinical and physiological signs/parameters?

Yes
- Control oxygen via face mask to maintain SpO₂ > 92%
  (88% - 92% in patients known to be sensitive to oxygen).

No
- Increase CPAP by 2.5 – 5 cm H₂O pressure/CPAP valve and review pre-conditions.

Yes
- CPAP = 5 cm H₂O pressure/CPAP valve for 4-6 hours?

No
- Stable clinical and physiological signs/parameters?

Yes
- Stable clinical and physiological signs/parameters for 4-6 hours?

No
- Restart NIV (see NIV pathway) and review pre-conditions.

Yes
- Continue with medical treatment and controlled oxygen.

Notes:
- Some patients may be suitable for weaning even when CPAP > 10 cm H₂O – discuss with experienced doctor/ICU.
- The rate of weaning in this pathway is a guide only – some patients can be weaned faster, others more slowly.
- The weaning process may be interrupted overnight in order to promote rest.
- Some patients (e.g. with post-operative lung collapse) may continue to require CPAP support overnight in order to overcome nocturnal hypoventilation.
- The PₐO₂ should not be used as the sole weaning parameter with the Boussignac system as the delivered oxygen is uncontrolled and typically in excess of 60%.
- This pathway is not suitable for patients for whom NIV is being withdrawn and palliative-only management started.
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BiPAP Weaning Pathway

Pre-conditions:
- Primary illness treated or under medical control.
- Targets in clinical and physiological parameters (respiratory rate, heart rate, mental state, blood gas parameters) achieved and stable for ≥ 12 hours.
- IPAP ≤ 18 cm H₂O, EPAP ≤ 8 cm H₂O, total respiratory rate ≤ 24 bpm.

Reduce IPAP by 2 cm H₂O and EPAP by 2 cm H₂O every 4–6 hourly. Reduce rate to 12 bpm. Minimum IPAP = 12 cm H₂O, EPAP = 4 cm H₂O.

Stable clinical and physiological signs/parameters?

Increase IPAP by 4 cm H₂O and/or EPAP by 2 cm H₂O and review pre-conditions. Consider CPAP.

IPAP = 12 cm H₂O and EPAP = 4 cm H₂O for 4-6 hours?

Stable clinical and physiological signs/parameters?

Controlled oxygen via face mask to maintain SpO₂ > 92% (88% - 92% in patients known to be sensitive to oxygen).

Restart NIV (see NIV pathway) and review pre-conditions.

Stable clinical and physiological signs/parameters for 4-6 hours?

Continue with medical treatment and controlled oxygen.

Notes:
- The rate of weaning in this pathway is a guide only – some patients can be weaned faster, others more slowly.
- The weaning process may be interrupted overnight in order to promote rest.
- Some patients may be weaned from BiPAP onto CPAP, before being weaned completely off NIV.
- Some patients (e.g. with post-operative lung collapse) may continue to require CPAP support overnight in order to overcome nocturnal hypoventilation.
- This pathway is not suitable for patients for whom NIV is being withdrawn and palliative-only management started.
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References.

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