NON-INVASIVE POSITIVE PRESSURE VENTILATION IN THE EMERGENCY DEPARTMENT

Reviewed and by K. Maddern 2010
**Introduction**

This learning package offers you the principles of non-invasive positive pressure ventilation (NPPV) and will assist you to provide appropriate and safe nursing care for patients who require NPPV.

This learning package consists of guidelines only and does not provide all the answers. Consequently you are expected to access additional resources and follow the emergency department protocols. Additionally, it is essential that when applying and monitoring a patient with NPPV that the nurse in charge is notified and the individual needs of the patient are taken into consideration.

**Assessment:**

Assessment of competency will include:

- Direct observation of correct setting up of a non-invasive ventilator
- Verbal response to direct questioning
- Satisfactory completion of a take home test

**Learning Outcomes:**

- Describes normal ventilation
- Lists the signs and symptoms of acute respiratory failure
- Explains the concept of non-invasive positive pressure ventilation
- Explains the benefits of NPPV
- Defines Continuous Positive Airway Pressure
- Defines Bi Level Positive Airway Pressure
- Identifies clinical indications that may require the use of NPPV
- Lists the contraindications of NPPV
- Demonstrates the ability to set-up BiPAP/CPAP
• Outlines the nursing responsibilities regarding the appropriate clinical assessment and physiological monitoring of a patient undergoing NPPV
• Identifies the principles of trouble shooting
• Maintains infection control principles
Background:

Non-invasive positive pressure ventilation has been used successfully for many years in patients with chronic hypoventilation syndromes who require respiratory support during sleep. However, NPPV has become increasingly successful when used to support ventilation, for Type 1 and Type 11 respiratory failure both acute and chronic, and for bronchial hygiene. Consequently, the use of NPPV is now commonplace in all clinical settings including general medical and surgical wards, intensive care units and in emergency departments.

Although NPPV has proven to be successful for many conditions and in numerous environments it is not without its dangers, and even when used appropriately by an experienced clinician, can pose a risk to the patient. Consequently, it is essential that nurse clinicians acquire and maintain a level of knowledge regarding the use of NPPV to ensure safe practice. Further, it is imperative that the clinician understands that the application of NPPV simply provides respiratory support for the patient while the underlying cause of the respiratory dysfunction is treated.
Physiology

In order to maintain homeostasis the body’s cells require oxygen for aerobic metabolism, conversely the body needs to rid itself of the by-products of metabolism, specifically carbon dioxide. In order for this to occur several processes need to follow;

1. The uptake of oxygen from the environment
2. Interface of ventilation and perfusion
3. The delivery of oxygen to the tissues
4. The utilization of oxygen at the cellular level (internal respiration/cellular respiration) and,
5. The exhalation of metabolism by-products to the external environment

To extract oxygen from the environment a functioning respiratory system is imperative. Stimulation of respiration involves both neural and chemical mechanisms. Chemical stimulation occurs when levels of hydrogen ions increase, which is directly related to the levels of carbon dioxide. Hydrogen ions stimulate chemical receptors located in the medulla oblongata, which instigates neural mechanisms by communication with respiratory centers in the brain to cause stimulation of the intercostals and phrenic nerves resulting in inhalation.

The diaphragm (the primary muscle of respiration) when stimulated contracts, increasing the volume in the thoracic cavity, therefore decreasing the pressure in the thoracic cavity. This increase in negative pressure results in air being drawn in from the atmosphere, from an area of higher pressure to an area of lower pressure, entering the lungs and diffusing to the alveoli.

When oxygen reaches the alveoli, it diffuses through the alveoli membrane through to the interstitial space and pulmonary capillary membrane where it is transported to the cells. As oxygen is being off loaded to the cells, a proportion of carbon dioxide is expelled from the cells and enters the venous supply, to return to the lungs to be exhaled. Exhalation, a passive process, occurs when stretch receptors are stimulated by inhalation, resulting in the passive relaxation of respiratory muscles. No matter how forced the expiration is, a person
is unable to exhale all of the air from their lungs, and in the ‘normal’ individual approximately 1200mls of air remains in the lungs; this is called the residual volume.

The functional residual capacity (FRC) is the amount of air in the lungs at the end of exhalation and is involved in gas exchange. A particular volume of air at the end of exhalation is required to keep the alveoli open and is known as the critical closing volume (CCV). When FRC is less than CCV atelectasis occurs resulting in incomplete expansion of the lungs.

A combination of three factors determine the work of ventilation:

1. Surface tension
2. Airway resistance, or the compliance of the lung and the thorax
3. The efforts of the inspiratory muscles

Any alteration to one or more of the above factors will alter normal ventilation and can result in a ventilation and perfusion mismatch. For ventilation (air) and perfusion (blood) to match, blood must perfuse ventilated regions of the lung and air must ventilate perfused regions of the lung.

A mismatch of ventilation and perfusion can result in wasted perfusion (shunt), as in atelectasis or pneumonia, which results in a reduced surface area for gas exchange, which can result in hypoxaemia and hypercapnia.

When hypoxia and hypercapnia occurs, the sympathetic nervous system (SNS) responds to assist normalization by increasing the respiratory and heart rates to promote the availability of oxygen to the tissues. However, an increase in the respiratory rate and heart rate are also associated with an elevation in the work of breathing (WOB). With an elevation in the WOB a simultaneous elevation in the bodies oxygen needs occurs. If the underlying cause for the increased need for oxygen is not corrected, oxygen demand exceeds oxygen supply; alveolar hypoventilation and respiratory muscle fatigue can ensue, while attempting to normalize gas exchange. Subsequently impending respiratory failure and respiratory arrest is imminent.
Respiratory Failure

Respiratory failure as defined by the British Thoracic Society is the, ‘...failure to maintain adequate gas exchange and is characterised by abnormalities of arterial blood gas tensions’ (British Thoracic Society Standards of Care Committee; 2000).

*Type 1 failure* is evident when the partial pressure of oxygen (Pao2) is <60mm Hg with a normal or low normal partial pressure of carbon dioxide (PaCO2). *Type 2 failure* is evident when the Pao2 is <60mm Hg and the PaCO2 is greater than 45 mm Hg.

Respiratory failure can be acute, acute on chronic or chronic.

**With acute hypercapnic respiratory failure** the patient presents with little evidence to suggest a pre-existing respiratory disease. Arterial blood gas analysis will indicate a high PaCO2, a low pH and normal bicarbonate levels.

**With chronic hypercapnic respiratory failure** the patient has a history of a respiratory disease. Arterial blood gas analysis indicates a high PaCO2 a normal pH and high bicarbonate levels.

**With acute-on-chronic hypercapnic respiratory failure** there is evidence of a pre-existing respiratory disease and pre-existing hypercapnic respiratory failure. Arterial blood gas analysis will indicate a high PaCO2, low pH and high bicarbonate levels.

<table>
<thead>
<tr>
<th>Chronic Respiratory Failure</th>
<th>Acute Respiratory Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory Muscle Dysfunction</td>
<td>Hypercapnic Respiratory Failure</td>
</tr>
<tr>
<td>- Poliomyelitis</td>
<td>- Acute exacerbations of COPD</td>
</tr>
<tr>
<td>- Muscular dystrophies</td>
<td>- Status asthmaticus</td>
</tr>
<tr>
<td>Neurological Disorders</td>
<td>- Cardiogenic pulmonary oedema</td>
</tr>
<tr>
<td>- Neuropathies</td>
<td>- Post extubation</td>
</tr>
<tr>
<td>- Spinal cord injuries</td>
<td>- Post surgical respiratory failure</td>
</tr>
<tr>
<td>- Brainstem lesions</td>
<td></td>
</tr>
<tr>
<td>- Primary alveolar hypoventilation</td>
<td></td>
</tr>
<tr>
<td>Chest Wall Disorders</td>
<td>- End stage lung disease awaiting transplantation</td>
</tr>
<tr>
<td>------------------------------------</td>
<td>----------------------------------------</td>
</tr>
<tr>
<td>- Scoliosis</td>
<td>- Patients who are not suitable for intubation</td>
</tr>
<tr>
<td>- Thoracoplasty</td>
<td>- Palliative care measure</td>
</tr>
<tr>
<td>Upper Airway Disorders</td>
<td>Hypoxemic Respiratory Failure</td>
</tr>
<tr>
<td>- Severe OSA</td>
<td>- Non-cardiogenic pulmonary oedema</td>
</tr>
<tr>
<td>- Obesity hypoventilation</td>
<td>- Post traumatic respiratory failure</td>
</tr>
<tr>
<td>Lung Disease</td>
<td>- AIDS with opportunistic pneumonia</td>
</tr>
<tr>
<td>- COPD</td>
<td>- Paediatric patients</td>
</tr>
<tr>
<td>- Cystic fibrosis</td>
<td>- Community acquired pneumonia</td>
</tr>
</tbody>
</table>
**Signs and symptoms of respiratory failure**

Signs and symptoms of respiratory failure can vary from each individual. Knowing your patient and being alert to the sometimes subtle changes in their condition will promote early recognition of respiratory dysfunction.

**Signs and symptoms include:**

- Restlessness
- Anxiety
- Confusion
- Increased respiratory rate
- Increased heart rate
- Pale, clammy skin
- Irritability
- Increased SOB
- Low oxygen saturations
- Fatigues easily
- Delerium
- Dementia

This list is not conclusive and it is important to recognise that some patients may exhibit few symptoms. Particularly those patients with a pre-existing lung disease or in those patients who have limited mobility and increasing breathlessness on exertion is not an issue.

In general many patients with acute respiratory failure are successfully treated with standard measures such as supplemental oxygen therapy and appropriate medical treatment. Nonetheless, many patients require the assistance of additional ventilatory support in the form of non-invasive positive pressure ventilation.
Non-Invasive Positive Pressure Ventilation

Non-invasive positive pressure ventilation (NPPV) is the application of positive pressure via the upper respiratory tract without the need for endotracheal intubation.

Positive pressure ventilation differs to that of spontaneous ventilation in that it induces a positive pressure in the lungs by forcing air into the lungs during inhalation, as opposed to the negative pressures that occur during normal spontaneous ventilation. Positive pressure ventilation assists the sustenance of gas exchange by promoting the interface of air and blood, reducing the work of breathing and preventing respiratory muscle fatigue. Additionally, NPPV avoids the need for more invasive ventilatory support while improving gas exchange.

Contraindications:

- Copious secretions
- Inability to clear secretions
- Respiratory arrest or need for immediate intubation
- Unconsciousness/unable to maintain own airway
- Hypotension
- Hypovolaemia, regardless of the cause
- Life threatening arrhythmias
- Upper airway obstruction
- Skull or facial trauma
- Inability to cooperate or fit mask
- Emphysematous bullae
- Pneumothorax
- Pneumomediastinum
- Massive epistaxis
- Acute sinusitis
- Vomiting
**Advantages of NPPV:**

- Airway defence remains intact
- Patient is able to communicate
- Decreases the need for invasive ventilation
- Improves patients comfort
- Decreases mortality
- Reduces ICU admissions
- Decreased need for sedation
- Reduces incidence of nosocomial infections
- If introduced in a prudent manner can reduce patient length of stay

  **Positive physiological effects** -  
  - Assists the alveoli to remain open
  - Increases functional residual volume
  - Enhances oxygenation and gas exchange
  - Reduces the work of breathing
  - Augments patients spontaneous respiratory effort

**Disadvantages of NPPV:**

- No direct access for suctioning
- In some models no alarms
- Respiratory deterioration can occur if patient dysynchronous with the device

  **Negative physiological effects** –

  *Decreased cardiac output* is the most common negative effect. As positive pressure inflates the lungs, pressure in the thorax builds decreasing the flow of blood to the vena cava (pre-load) and reducing the blood flow to the right atrium, consequently decreased filling of the left ventricle occurs resulting in a reduced cardiac output.

  Blood flow to the splanhnic area decreases as the diaphragm descends into the abdomen during the inspiratory phase, which can result in ischaemia of the gastric mucosa. Decreased blood flow to the kidneys and liver can also occur
Increase in intra-cerebral pressures can also occur, therefore has the potential to alter mentation, particularly if the patient is already neurologically compromised.

Rupture of the lung from increased intra-thoracic and intra-airway pressures (barotrauma), pneumothorax, subcutaneous emphysema and pneumomediastinum

The contraindications, advantages and disadvantages have to be evaluated on an individual basis and does not necessarily exclude or ensure the use of NPPV. It is essential to recognise that any NPPV system should not be used as a life support ventilator and should not be used if intubation is indicated.

**Modes of Ventilatory Support**

**CPAP (continuous positive airway pressure).**

The CPAP mode provides a single level of positive pressure to the airways throughout the respiratory cycle; that is, a constant pressure is applied throughout the inspiratory and expiratory cycle. In the CPAP mode the patient is in control of the respiratory cycle therefore must be breathing spontaneously.

During CPAP patients exhale against the prescribed amount of pressure. This back-pressure re-expands and stabilises alveoli assisting in maintaining patent upper and lower airways. The re-expansion of alveoli increases the surface area for gas exchange and improving end expiratory lung volumes, thereby promoting the interface of ventilation and perfusion.

**Indications:** CPAP is commonly used for individuals with obstructive sleep apnoea (OSA) and for those who require no tidal volume augmentation. If the primary problem is hypoxia or cardiogenic pulmonary oedema CPAP is usually indicated.
BiPAP (Bi level positive airway pressure).

The BiPAP mode provides two levels of positive pressure during the respiratory cycle. The inspiratory positive airway pressure (IPAP) and the expiratory positive airway pressure (EPAP)

IPAP is the pressure or support that is delivered to the patient during inspiration and is always higher than the EPAP setting.

- IPAP assists the patient to inhale more deeply thereby increasing the volume of air that is inspired.
- IPAP takes over some of the work of the inspiratory muscles and consequently reduces the work of breathing
- IPAP reduces PaCO2 by increasing alveolar washout

EPAP (expiratory positive airway pressure). EPAP is the pressure that remains in the circuit during the patient’s expiratory phase. EPAP is always lower than IPAP.

- EPAP will promote patency of the upper airway
- EPAP improves the end expiratory lung volumes
- EPAP splints open the alveoli

As the difference between IPAP and EPAP increases a concurrent increase in the tidal volume occurs. Health care personnel need to be aware that the patient is able to tolerate high tidal volumes. Inspiratory pressures that are too high can result in a worsening leak reducing the effectiveness of NPPV. These leaks are noticeably more evident in patients with weak facial muscles or in patients with stiff lungs or chest walls.

Indications: BiPAP is useful for those individuals who are hypoventilating and/or are retaining CO2. Typically this can include an exhausted patient with severe asthma, a patient with an opiate overdose and a patient with a pulmonary embolus (dead space ventilation). Further patients with COAD with CO2 retention and those with a restrictive lung disease can also benefit from BiPAP.

- It is important to remember that when IPAP equals EPAP that is equivalent to CPAP
- EPAP should never be greater than IPAP
**Spontaneous mode:** (S)
Triggering from EPAP to IPAP is reliant on patient effort therefore the patient must be spontaneously breathing and initiating inspirations.

**Spontaneous – Timed mode:** (S/T)
The device will still be initiated by the patient, however, a backup rate can be added if the respiratory rate falls below an ‘acceptable’ rate. Consequently, the device will deliver additional breaths to those spontaneously initiated.

**Timed mode:** (T)
This mode is utilised if the patient is not spontaneously breathing and should not be used on the wards. If this mode is required, endotracheal intubation is necessary. IPAP and EPAP pressures should be set with the respiratory rate.

**Additional settings:**
**IPAP Max** is the maximum time that the device remains in IPAP before switching to EPAP. The device usually switches from IPAP to EPAP when it senses an alteration in flow. If large mouth leaks are occurring the device may not be able to sense when the patient has begun expiration and as a result the device may stay in inspiration. As a consequence the work of breathing may increase.

**IPAP Min** ensures the minimum time that the device is set to stay in IPAP. A useful setting for patients with weak inspiratory muscles, however this setting is not usually required.

**The Equipment**
Numerous non-invasive positive pressure devices are available and although the principles of use are the same it is the clinician’s responsibility to ensure that they are familiar with the device they are using.

Essentially the equipment consists of three components:
1. The interface - incorporates the full-face mask, the nasal mask, the nasal pillow, the mouth piece and head gear (harness)
2. A flexible hose connecting the interface to the pump
3. The device (pump) that provides the necessary pressure support ventilation

NB: The CO2 washout valve must be either part of the mask, hose or added to the circuit. A bacteria filter and oxygen connector can be added.

**Mask:**
It is essential that the mask fit is appropriate and comfortable, as this will have an enormous impact on acceptance of therapy by the patient.

**Masks** can be quite difficult to put together so it is essential that you become familiar with mask types.

- Select mask size according to manufacturers guidelines
- The cushion should be the smallest that clears the nostrils
- The cushion membrane sits snugly around the nose
- If forehead arms are insitu ensure that the forehead pads touch the forehead when properly positioned

Know where your CO2 washout valve is situated. The CO2 washout valve may be part of the mask or you may have to add it to the mask.

**Flexible hose** - ideally the size of the hose should be standardised and when replacing, the clinician should ensure that the same length of hose is applied. The greater the length of the hose the greater the dead space will be, and if excessive lengths are used an increase in the work of breathing may occur.
What ventilatory support to use:

It is essential to determine the primary problem and the aims of therapy before deciding on what ventilatory mode and settings to commence on.

What is the primary problem? – Hypoxia, APO, hypoventilation, retaining CO2.

The choice should be determined by a physician and supported by the policy. However a general guideline is:

- **Hypoxia, APO**
  - CPAP

- **Hypoventilation/CO2 retention**
  - BiPAP

Nevertheless, if one mode does not work it is reasonable to attempt the other mode.

Pressure Settings:

The pressure settings should be determined by a physician, however as a general rule for particular client groups the following settings are suggestions only:

**Pneumonia** – BiPAP – 12cm IPAP and 6cm EPAP (MAX IPAP 24/EPAP 10)

IPAP can be increased by 4cm and EPAP increased by 2cm (IPAP should not be greater than EPAP by 14cm)
**Exacerbation of COPD** – IPAP is mostly recommended with low EPAP. Initial settings of 8cm IPAP and 4cm EPAP. IPAP increased gently by 2cm increments, IPAP should not exceed 16cm’s.

**Acute Pulmonary Oedema** – CPAP with setting to commence on 10cm. Increase by 2cm to maximum of 14cm.

Current practice indicates the APO should only be treated with CPAP, this is particularly for cardiogenic pulmonary oedema. However if the patient is not responding and/or has low tidal volumes changing to BiPAP with an IPAP of 16cm and an EPAP of 10cms is suggested, these alterations should be ordered by a physician.

**Atelectasis** – Is not an uncommon problem in the post-operative patient or a patient with abdominal conditions, for example pancreatitis. CPAP usually assists the reinflation of the lower lobes ranging from 5cms to 12 cm. The pressure settings will depend on the amount of abdominal distension, the type of surgery and x-ray findings.

**Fractured ribs** – With fractured ribs including a flail chest segment TV is frequently reduced which can lead to atelectasis and pneumonia. BiPAP can reduce pain by limiting mechanical work and preventing atelectasis if initiated early. An undrained pneumothorax should be excluded before treatment begins.
Setting should commence at IPAP 10cm and EPAP 4cm. IPAP can be increased by 2cm and should not exceed 18cm.

**These pressure settings are recommendations only. Pressure settings should be set and altered in consultation with a physician dependant on patient condition and response.**
Patient Monitoring and Care:
It is essential that the patient be closely monitored during NPPV. If a **full-face mask** is used the patient requires 1:1 nursing.

Providing education to the patient and family is essential especially before initiation of therapy. However, if time does not permit before initiation of treatment emotional support and education must be given throughout therapy.

**Observations:**
The clinical status of the patient should determine the level of observations performed.

**Before initiation of treatment baseline observations should be collected as well as constant reassessment throughout the therapy.** These observations include:

- Vital signs every 30 minutes
- Oxygen saturations
- ABG’s
- Respiratory assessment at least 1 hourly
- Glasgow coma score
- Current respiratory support
- Skin integrity

**Before applying NPPV**

- Be aware of hospital policy
- Verify the settings as prescribed by the physician
- Choose appropriate size mask
- Oxygen and suction is available
- Patient is close to the nurse’s station
- Turn the device on before applying mask to patient
- Patient should be in the upright position
During therapy:

For the first hour:

- **30 minutely** observations should be taken and additional observations when pressure support is altered.
- After the first hour, hourly observations are appropriate unless clinical condition suggests otherwise.
- Observations must include:
  - Assessing CO2 washout valve
  - Mask fit
  - Mask leaks – eye irritation
  - The integrity of the circuit
  - Patient comfort
  - Conscious state
  - Vital signs
  - Respiratory assessment
  - ABG’s are recommended one hour after initiation and then following alterations to therapy
  - Skin integrity
  - Settings are accurate
  - Complications of therapy

Troubleshooting

The application of positive pressure ventilation can cause discomfort or at times harm to the patient. The clinician must act appropriately when confronted with any problem. The following table is a guideline to assist the clinician.
If **PaCO₂ remains elevated** the clinician must assess for:
- The FiO₂
- Excessive leak
- Integrity of the circuit
- Patency of CO₂ washout valve
- Device not synchronizing with the patient
- Observes for chest expansion/chest auscultation
- In this instance IPAP may have to be increased, however a physician must be informed and preferably alter settings

If **PaCO₂ improves but PaO₂ remains low**:
- Consider increasing FiO₂
- Consider increasing EPAP following discussion with medical officer
- Check integrity of the system
- Consider different mode of ventilation

**When to stop NPPV**

An improvement may be evident as quickly as 15 minutes post initiation of treatment, however blood gases and other indicators of improvement may not be evident for at least one hour; the patient may be also be quite fatigued.

- If the patient can tolerate the treatment, positive pressure ventilation should be maintained for at least four hours or if possible for 24 hrs, with assessments according to hospital policy. However, if the patient is unable to tolerate treatment for a longer period a treatment regime should be developed according to the individual needs of the patient. Nocturnal support is usually necessary for all patient groups for the following 24-48 hrs.

- If the patient is reliant on NPPV while awake, attempts should be made to decrease the time that the patient is utilizing NPPV. This should be determined by clinical
indicators such as respiratory rate, use of accessory muscles, oxygen saturations and carbon dioxide levels.

- NPPV should not be continued if invasive ventilation is indicated

**Cleaning the equipment**

The principles of infection control should be maintained according to your institution's infection control policy.

- The device should be wiped over daily with a warm soapy cloth and between patients, manufacturers' instructions generally do not recommend cleaning device with alcohol solution.
- The mask should be wiped over daily with a soft damp cloth or changed every 24 hours.
- When the mask is no longer in use it should be wiped over and then sent to CSSD for cleaning.
- Tubing (hose) should be changed every seven days. If intermittent NPPV tubing and mask should be disconnected from the device and hung in a clean dry place
- Bacterial filters should be changed daily.
- Internal filters should be changed according to manufacturers' instructions as long as they follow hospital infection control policy.
- An individual circuit is required for each patient. The tubing is disposable, as is the mask (The mask can be sent to CSSD and is then cleaned and sent to impoverished countries).
Documentation

Accurate documentation cannot be over emphasized. Assessment of the patient before during and after treatment is essential to determine the efficacy of the treatment. Documenting the patient’s assessment, pressure settings and changes to settings also assists the overall assessment of the patient.

The maintenance of equipment and changes to equipment should also be documented.
Short Answer Questions

1. List the main components that ensures internal respiration.

2. Describe the difference in intrathoracic pressures between normal ventilation and positive pressure ventilation.

3. List ten signs and symptoms of acute respiratory failure.

4. What is the function of positive pressure ventilation
   During inspiration?
   During expiration?

5. What is Bi-Level positive airway pressure?

6. What is continuous positive airway pressure (CPAP)?

7. List the major contraindications for NIPPV.

8. What patient type can benefit from NIPPV

9. How does the application of NIPPV affect blood pressure?
10. How does the alteration in blood pressure affect urine output?

11. In what circumstances would you notify a medical officer?

12. What needs to occur if nursing a patient with a full-face mask?