Clinical practice review

Non-Invasive Ventilation for Adult Acute Respiratory Failure. Part I

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ABSTRACT

Objective: To detail the history, modes, physiological effects, and circuit geometry of non-invasive ventilation.

Data sources: A review of articles published in peer-reviewed journals from 1966 to 1998 and identified through a MEDLINE search on non-invasive ventilation.

Summary of review: Non-invasive ventilation (NIV) has been used for many years as an adjunct to standard therapy in patients with acute and chronic respiratory disorders. The newer modes of NIV which include continuous positive airway pressure (CPAP), pressure support ventilation (PSV), BiPAP (bi-level positive airway pressure) and controlled and assisted modes of intermittent non-invasive positive pressure ventilation (NIPPV) have additional advantages and are often used routinely in many respiratory diseases. These modes of ventilatory support have been found to improve arterial oxygenation, ventilation, work of breathing, and cardiac function, in patients with respiratory failure, although in normal subjects, respiration is often impaired.

Conclusions: Non-invasive ventilation using the modes of CPAP, PSV, BiPAP and NIPPV should be considered in patients with respiratory failure who are unresponsive to conventional therapy, before considering invasive mechanical ventilation. (Critical Care and Resuscitation 1999; 1: 187-198)

Key Words: Non-invasive ventilation, work of breathing, minute ventilation, ventilation circuits

Introduction

Management of critically ill patients often involves treatment of acute respiratory failure (ARF). Severe ARF may require tracheal intubation and mechanical ventilation (MV), both of which carry an associated morbidity. Efforts to reduce morbidity from MV have included early detection and prevention of respiratory failure, and new ventilatory modes which attempt to improve the safety of MV and expedite weaning. Despite progress in these areas, avoidance of MV where possible remains a desirable option.

Non-invasive ventilation (NIV) is a therapeutic option for respiratory failure and has been used for a variety of acute and chronic conditions, across all age groups. It provides respiratory support without the need for tracheal intubation, maintaining many of the physiological advantages of spontaneous respiration, and in some patient groups there is evidence to suggest it has lower morbidity compared to MV.

Currently published reviews of NIV1-4 are small in number and provide incomplete information regarding the physiology of NIV, its various modes, and their

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clinical role in separate models of ARF. This review is based on the premise that a physiological framework provides a clear guide to: 1) the potential benefits of NIV, 2) the differences between various modes of NIV, 3) the side effects of NIV and, 4) the clinical indications and limits of each mode. The aim of this two part review is to discuss the physiological and technical aspects of NIV (Part I), followed by the clinical use of NIV in adults with ARF (Part II).

**Disadvantages of mechanical ventilation**

Although clinical experience and research support the benefits of MV in ARF it may produce significant morbidity. These complications have been well described and are reviewed briefly in relation to the potential advantages of NIV.

Laryngoscopy and endotracheal intubation carries a significant risk of trauma to the upper airways. An endotracheal tube (ETT) bypasses the beneficial physiological effects of the upper airway, such as warming and humidification of inspired gas, addition of endogenous nitric oxide to the inspired gas, and protective reflexes. Depression of laryngo-tracheal reflexes increases the risk of pulmonary aspiration and, together with depression of mucociliary function, increases the risk of nosocomial infections.

Work of breathing is increased by the presence of an ETT and a breathing circuit. Problems of ventilator asynchrony and impaired weaning may result. Increased patient discomfort and the loss of verbal communication often result in increased sedative and analgesic requirements.

Commonly used modes of MV alter the distribution of ventilation and perfusion resulting in a fall in end-expiratory lung volume (EELV), lung compliance, and ventilation-perfusion (V/Q) mismatch. This may be partially overcome by using positive end-expiratory pressure (PEEP) and continuous positive airway pressure (CPAP). MV may cause lung injury through barotrauma, ventilator associated pneumonia, and oxygen toxicity.

The cardiovascular side-effects of MV relate chiefly to elevated transmural pressures and intra-thoracic lung volumes. These factors may induce a fall in venous return and cardiac output, and activation of compensatory sympathetic and hormonal responses. Many of these problems may be minimised or eliminated through the use of non-invasive ventilatory support modes.

**History of non-invasive ventilation**

Non-invasive respiratory support has been used in cardiopulmonary resuscitation for many years. Cuirass and other non-invasive negative-pressure generators were used for decades prior to the advent of positive pressure ventilators.

The use of mask-CPAP for the treatment of ARF was first advocated in the mid 1930’s. In 1936 Poulton reported a series of 22 patients with acute pulmonary oedema, asthma, and pneumonia. In the description of his CPAP circuit - the “pulmonary plus pressure machine” - he described many characteristics of an efficient circuit. It provided a high inspiratory flow using a household vacuum fan, an air-tight face mask, an expiratory pressure-relief valve using an Ambu valve which was placed close to the patient’s airway, and the option for warming the inspired gas using a hot water bottle in the dust bag chamber.

The early achievements of NIV in ARF were overshadowed by other developments. By the 1930’s there was widespread use of laryngoscopy and intubation for anaesthesia, and the early 1950’s saw the advent of simple and reliable positive pressure ventilators. There was also a conceptual shift away from the treatment of discrete pulmonary disorders (by the respiratory physician in the general ward setting), to the treatment of the generic pulmonary condition, ‘acute respiratory failure’, in a specialised ‘intensive care’ ward, by respiratory technicians and anaesthetists. NIV was relegated to a minor role as an adjunct to chest physiotherapy and to the delivery of bronchodilators.

In the 1960’s CPAP was being successfully utilised in paediatric practice for the management of post-cardiac surgery and hyaline membrane disease patients. Not long after, CPAP was described for prophylaxis for the (adult) acute respiratory distress syndrome (ARDS) and PEEP was demonstrated to be of benefit in mechanically ventilated ARDS patients.

The use of NIV in adults grew out of this early success and the simplicity of CPAP circuit construction. By the late 1970’s there were numerous anecdotal reports of the successful use of mask-CPAP for a variety of respiratory conditions.

Pressure support ventilation (PSV) as a NIV mode is of more recent origin. It arose out of the technological advances in pressure and flow sensors (which enabled mechanical ventilators to sense commencement and cessation of spontaneous inspiratory pressure or flow), and the demonstrable benefit of PSV in intubated patients.

**Modes of non-invasive ventilation**

There are five commonly described NIV modes:

1. Continuous positive airway pressure (CPAP)
2. Pressure support ventilation (PSV)
3. A combination of PSV + CPAP
4. BiPAP (bilevel positive airway pressure) or combined positive inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP).
5. Controlled and assisted modes of intermittent positive pressure ventilation (NIPPV).

There is a need for a consensus-based definition of “NIV” given the number of distinct modes, its widespread clinical use, and ongoing NIV research. All the above modes have three features in common: 1) the delivery of positive airway pressure (Paw) without airway intubation; 2) the respiratory cycle can be patient-controlled - with BiPAP and NIPPV also having the option of mechanical time-cycling, and; 3) the delivery of Paw via a nasal or naso-oral (“full face”) mask. Face-tents and head boxes have also been used for CPAP in paediatric practice.

There is also a need for clinical guidelines to clarify the indications for NIV in ARF. For some conditions (e.g., cardiogenic pulmonary oedema) there is strong evidence to support its routine use, but in others NIV appears to have little benefit. Although a detailed discussion of cardiopulmonary physiology is beyond the scope of this review, it is worthwhile considering some aspects of respiratory and cardiac function in relation to NIV and ARF.

**Physiology of non-invasive ventilation (Table 1)**

The physiological effects of NIV vary greatly and depend upon: 1) the nature of the subject investigated; 2) the pathophysiology of the acute respiratory disorder; 3) the severity of respiratory dysfunction; 4) the mode of NIV and level of Paw used, and; 5) the efficiency of the breathing circuit.

PEEP should be distinguished from CPAP. PEEP refers to the application of positive Paw to an intubated patient during the expiratory phase of a mechanical breath cycle, whereas CPAP refers to positive Paw during a spontaneous breath cycle. The distinction is important because the physiological effects of PEEP and CPAP are not necessarily equivalent. Furthermore, CPAP applied to an intubated patient is not necessarily identical to mask-CPPAP. In the former situation the subject must breath via an ETT and ventilator circuit both of which impose additional workload. These factors should be borne in mind when interpreting research data, and in determining the clinical applications of NIV.

The possible benefits of NIV on respiratory function are improved oxygenation and alveolar ventilation (V̇a), and reduced work of breathing (Wb). The potential effects on cardiac function are alterations in preload, afterload, chronotropy and lusitropy.

**Oxygenation**

The arterial oxygen content (CaO₂) is influenced by a number of factors some of which may be identified by rearranging the pulmonary shunt equation:

\[ CaO_2 = CcO_2 - Q_s/Q_t(CcO_2 - CvO_2) \]

Where
- CaO₂ = arterial oxygen content
- CcO₂ = end-pulmonary capillary oxygen content;
- CvO₂ = mixed venous oxygen content;
- Qₕ = effective pulmonary shunt flow; and
- Q₂ = cardiac output.

CcO₂ is itself dependent upon the alveolar partial pressure of oxygen (PₐO₂) and diffusion into the pulmonary capillary. Factors which influence PₐO₂ may be identified by considering the simplified ideal alveolar gas equation,

\[ P_AO_2 = [(P_B - P_H2O) \times F_IO_2] - PaCO_2/R \]

Where
- PₐO₂ = alveolar partial pressure of oxygen
- P_B = barometric pressure
- P_H2O = saturated water vapour pressure
- F_IO_2 = inspired oxygen concentration fraction
- PaCO₂ = arterial partial pressure of CO₂; and
- R = respiratory quotient.

Using these two equations it is possible to identify mechanisms through which NIV may improve oxygenation:

1. The majority of NIV circuits provide the ability to control F_IO₂ and deliver high inspired O₂ concentrations through a closed circuit. The increase in PₐO₂ is one of the most common mechanisms by which NIV improves PaO₂ and CaO₂.

2. In principle, diffusion may improve with the application of CPAP if redistribution of extravascular lung water decreases the water content of alveoli. At present there is no direct evidence for an increase in diffusing capacity independent of a change in lung volume or PₐO₂.

3. In hypercapnic patients breathing room air, a small rise in PₐO₂ may occur if NIV increases minute ventilation (VE) and reduces PaCO₂ (e.g., a chronic respiratory failure patient using domiciliary NIV without supplemental oxygen). A fall in PaCO₂ will also allow a rise in the CaO₂ via the Bohr effect.
4. $Q_t$ represents the combination of absolute pulmonary shunt and $V/Q$ mismatch. NIV may improve $V/Q$ relationships and $CaO_2$ through the recruitment of collapsed alveoli and end-expiratory lung volume (EELV).

5. Finally, oxygenation may also improve if cardiac output ($Q_t$) is increased. There are a number of studies which document an increase in cardiac output with mask-CPAP in particular patient subgroups (see below). CPAP has also been shown to increase oxygen delivery and $CvO_2$ in intubated patients with chronic cardiac disease, when compared to MV.  

Work of breathing ($W_B$)

$W_B$ is the pressure-volume work performed by the respiratory system. In healthy subjects it may be subdivided into the work required to overcome the impedance of the elastic ($W_{el}$) and the non-elastic properties of the lung and chest wall. Most of the non-elastic impedance comes from airway resistance to gas flow (producing a flow-resistance load, $W_{res}$), with a component from non-elastic tissue impedance (a non-elastic work load, $W_{n}$). 

In pathological states other forms of ventilatory work may be encountered. In severe airways obstruction an intrinsic end-expiratory alveolar pressure (PEEP) may exist which acts as an inspiratory threshold load (ITL) against which work ($W_{ITL}$) must be performed before gas flow can occur. In intubated subjects there is also the added circuit work ($W_{cir}$) required to overcome the impedance of the ETT, tubing, and valves. 

Therefore, $W_B = W_{el} + W_{res} + W_{n} + W_{ITL} + W_{cir}$

At any given lung volume, positive pressure applied to the airway ($P_{ao}$) will be distributed to the individual components; that is, $P_{ao} = P_{el} + P_{res} + P_{n} + P_{ITL} + P_{cir}$

Where

\begin{align*}
  P_{el} &= \text{pressure required to overcome elastic recoil;} \\
  P_{res} &= \text{pressure to overcome airway resistance;} \\
  P_{n} &= \text{pressure to overcome visco-elastic forces;} \\
  P_{ITL} &= \text{pressure to overcome an ITL (eg. PEEP);} \\
  P_{cir} &= \text{pressure to overcome circuit impedance.}
\end{align*}

It is also possible to mathematically redefine the components of the above equation as, 1) $P_{el} = \text{volume/dynamic compliance (V/C)}$; 2) $P_{res} = \text{flow resistance (V.R)}$; and, 3) $P_{ITL} = \text{total end expiratory alveolar pressure - extrinsic EEP}$. Therefore the equation above may be rewritten as,

$P_{ao} = V/C + V.R + EEP + P_{cir}$

so that the potential benefits of NIV on $W_B$ can be more easily identified by considering each component:

1. If the application of positive $P_{ao}$ results in a volume ($V$) of gas flowing into the lungs, then the amount of active patient work ($W_{pt}$) required may be reduced; that is, $W_{pt} = \text{WB} - (P_{ao} \times V)$. Many investigators have demonstrated that NIV reduces measured $W_{pt}$ and increases minute volume ($V_{E}$) or reduces respiratory rate and $PaCO_2$ implying an increase in $V_{E}$ (see below.)

2. The application of CPAP (and PEEP) may result in an upward shift of the pulmonary pressure-volume relationship. If the result is an increase in lung compliance ($C$) then $W_B$ will be decreased. An increase in $C$ may occur through redistribution of excess alveolar water, recruitment of alveoli, an increase in EELV, or a more homogeneous distribution of ventilation.

3. Inspiratory flow ($Vi$) will rise in proportion to the pressure gradient ($P_{ao} - P_{ab}$). For this to occur it is important that the circuit is capable of delivering a gas flow equivalent to peak $Vi$ (PIFR). Conversely the pressure gradient ($P_{ao} - P_{ab}$) may retard expiratory flow ($Ve$), except possibly in conditions where there is a tendency for small airways collapse. In this situation the application of expiratory positive $P_{ao}$ (PEEP or CPAP) could assist in minimising airway collapse and thus, paradoxically improve $Ve$.

4. There is evidence that CPAP reduces flow resistance ($R$) in both the upper airway and the lower (small) airways which is disproportional to the small rise in EELV.

5. An inspiratory threshold load (ITL) may occur in the presence of PEEP, particularly in patients with airflow obstruction. An elevated $P_{ao}$ may assist in decreasing this threshold work ($W_{ITL}$) by reducing the gradient (PEEP - $P_{ao}$) - the threshold load. This mechanism may explain why patients with PEEP, such as in asthma or COPD, have a reduction in spontaneous $W_B$ with mask-CPAP and that ‘optimal’ CPAP levels correlate with the measured threshold load.
6. Finally, \( W_B \) may also be reduced through a reduction in work \( (W_{\text{tot}}) \) required to overcome circuit impedance eg., circuit tubing, inspiratory and expiratory valves and ETT. Thus CPAP\(^{55} \) and PSV\(^{54,56} \) are advantageous in intubated patients weaning from MV. It should be remembered that NIV circuits will also add \( W_{\text{tot}} \) and therefore care must be taken in designing such circuits.\(^{57} \)

**Minute ventilation (\( V_E \)) and respiratory muscle fatigue**

If NIV reduces respiratory muscle effort \( (W_{\text{pt}}) \), this may allow an increase in \( V_E \) without increasing the risk of respiratory muscle insufficiency (i.e. fatigue). A reduction in \( W_{\text{tot}} \) together with an increase in oxygen supply-demand balance of the respiratory muscles, and may also allow an increase in \( V_E \).\(^{58,59} \) Persistent inspiratory muscle insufficiency and/or low \( V_E \) may benefit from an increase in the level of inspiratory \( P_{a0} \) (i.e. PSV).\(^{45,47} \)

**Cardiac function.**

The effect of NIV on the cardiovascular system is complex, although most of the changes are the result of a rise in mean intra-thoracic pressure and a fall in transmural pressure.

1. **Preload**
   A fall in transmural right atrial pressure will impede venous return. A subsequent reduction in right ventricular (RV) output will also reduce left ventricular (LV) preload or end-diastolic volume (LVEDV). Thus in the preload-sensitive heart, eg. healthy or hypovolaemic states, NIV may cause in a fall in cardiac output (CO).\(^{60,61} \)

2. **Right ventricular (RV) afterload**
   Pulmonary artery pressures rises proportionally with intra-thoracic pressure. An increase in RV afterload may cause a fall in ejection fraction.\(^{62,63} \) However, in the presence of reversible acute pulmonary hypertension - due to hypoxic pulmonary vasoconstriction - it is possible that the delivery of a high \( F_{\text{I}}O_2 \) with NIV may actually reduce RV afterload.

3. **Left ventricular afterload**
   LV afterload is increased by systemic vasoconstriction and negative intra-thoracic pressure.\(^{64} \) Even though positive intra-thoracic pressure increases RV afterload, it tends to reduce LV afterload because of a fall in transmural LV pressure.\(^{65,66} \) Thus in subjects with afterload-sensitive LV function, e.g. cardiomyopathy or congestive cardiac failure, a fall in afterload may result in an increase in CO.\(^{60,67} \)

4. **Chronotropy**
   Alteration in heart rate is often secondary to changes in stroke volume (SV), cardiac output (CO) and mean arterial blood pressure (MAP). If SV should fall then there may be a compensatory tachycardia to maintain CO and MAP. Conversely, in patients experiencing a rise in cardiac output with NIV there may be a compensatory reduction in heart rate.\(^{68,69} \) This may assist in decreasing LV work and the reduce the risk of myocardial ischaemia.

5. **Lusitropy**
   If NIV reverses an hypoxic state, reduces ventricular afterload, and slows the heart rate, then it may benefit myocardial oxygen supply-demand balance. Reversal of myocardial ischaemia will improve diastolic function - ventricular relaxation and compliance.\(^{70,71} \)

6. **Blood volume distribution**
   Positive intrathoracic pressure decreases thoracic blood volume.\(^{72} \) The distribution of extra-thoracic blood volume and flow are dependent upon the directional change in CO and MAP. If CO falls then there may be a reduction in renal,\(^{70,73} \) hepatic\(^{70,74} \) and splanchnic blood flows.\(^{73} \)

**Long-term benefits**

A detailed discussion of the long-term effects of NIV is beyond the scope of this review. However, it is worthwhile noting that there is evidence to suggest long-term benefits in cardiac and respiratory function with NIV.

Studies using mask-CPAP in patients with congestive cardiac failure have demonstrated that long-term use improves respiratory muscle strength\(^{75,76} \) and has short\(^{76} \) and long term benefits on cardiac function.\(^{77} \) Research into the long-term effects of NIV in COPD patients has also demonstrated improvements in respiratory muscle strength and improved exercise tolerance.\(^{78} \)

**Classification of modes and indications**

Based on the physiological effects of NIV, outlined above, it is possible to classify the potential benefits of various NIV modes (Table 1), and to identify specific clinical applications.

**Circuit geometry**

*Mask-CPAP*

CPAP is the most widely used and commonly described NIV mode, and implies the maintenance of
Table 1. Physiological effects of non-invasive ventilation modes in respiratory diseases

<table>
<thead>
<tr>
<th>Mode</th>
<th>CPAP</th>
<th>PSV</th>
<th>NIPPV</th>
<th>BiPAP</th>
</tr>
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<tbody>
<tr>
<td><strong>Respiratory Function</strong></td>
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<tr>
<td>P\textsubscript{A}O\textsubscript{2}</td>
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<td>++</td>
<td>++</td>
<td>+</td>
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<tr>
<td>V/Q</td>
<td>++</td>
<td>+</td>
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<td>+</td>
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<td>W\textsubscript{el}</td>
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<td>W\textsubscript{res}</td>
<td>++</td>
<td>+</td>
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<td>W\textsubscript{ITL}</td>
<td>++</td>
<td>0</td>
<td>0</td>
<td>+</td>
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<tr>
<td>W\textsubscript{cir}</td>
<td>++</td>
<td>++</td>
<td>+</td>
<td>+</td>
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<td><strong>Cardiac Function</strong></td>
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<td>Preload</td>
<td>0/-</td>
<td>0</td>
<td>0</td>
<td>0/-</td>
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<tr>
<td>Afterload</td>
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<td>+/-</td>
<td>+/0</td>
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<tr>
<td>Tachycardia</td>
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<td>+/0</td>
<td>+/-</td>
<td>+/0</td>
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<tr>
<td>Lusitropy</td>
<td>+/0</td>
<td>+/0</td>
<td>+/-</td>
<td>+/0</td>
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</tbody>
</table>

`++` = beneficial effect, `0` = no change, `+/-` = adverse effect. CPAP = continuous positive airway pressure, PSV = pressure support ventilation, NIPPV = non-invasive positive pressure ventilation, BiPAP = bilevel positive airway pressure. P\textsubscript{A}O\textsubscript{2} = Alveolar partial pressure of oxygen, V/Q = ventilation perfusion ratio, W\textsubscript{el} = work required to overcome elastic recoil, W\textsubscript{res} = work required to overcome airway resistance, W\textsubscript{ITL} = work required to overcome an inspiratory threshold load, W\textsubscript{cir} = work required to overcome circuit impedance, V\textsubscript{E} = minute ventilation.

an above-atmospheric P\textsubscript{ao}\textsubscript{2} throughout the respiratory cycle. Respiratory rate and tidal volume are determined by the subject. The circuit required is relatively simple.

To minimise W\textsubscript{el} and added W\textsubscript{cir} it is necessary to use a CPAP circuit which minimises fluctuations in the patient airway pressure (\Delta P\textsubscript{ao})\textsuperscript{14,79-81} and thus minimise large changes in intrapleural pressure.

There are a myriad of ‘home-made’ and commercial mask-CPAP circuits,\textsuperscript{14,81-84} they all have a number of common features, but not all perform efficiently. Although many mechanical ventilators are capable of delivering CPAP, their performance also varies widely.\textsuperscript{12} Some have a considerable imposed W\textsubscript{el} associated with inspiratory-flow triggering and expiratory valve opening,\textsuperscript{81,85} although most of the newer generation ventilators have improved considerably in this area. Data from lung models and from animal and human studies have helped to identify the important characteristics of an efficient CPAP circuit:

1. A high gas flow capable of matching spontaneous PIFR so that the desired P\textsubscript{ao}\textsubscript{2} level can be maintained and \Delta P\textsubscript{ao} minimised.\textsuperscript{12,86} Whilst quiet breathing may generate a PIFR of only 30L/min., in ARF this may be > 100L/min. High flows can be provided from a pressurised gas supply, a gas turbine,\textsuperscript{13} or jet venturi mechanism.\textsuperscript{15,88} In general, a continuous flow device is more efficient at reducing \Delta P\textsubscript{ao} than is a demand flow device.\textsuperscript{85}

2. An expiratory resistor capable of maintaining the desired P\textsubscript{ao}, yet offering a low resistance to expiratory flow.\textsuperscript{81} This may be a threshold resistor or flow resistor.\textsuperscript{13,42} Threshold resistors require a large valve area and a low opening pressure. The expiratory valve should be placed as close to the airway (i.e. mask) as possible.\textsuperscript{57}

3. Minimal length and wide bore tubing to reduce gas turbulence and flow resistance.

4. A comfortable and air-tight face or nasal mask.

5. Ability to accurately control F\textsubscript{I}O\textsubscript{2}.

6. Whilst a reservoir bag is unnecessary for a circuit capable of delivering high flows, its presence in low-flow (< 50L/min.) circuits will assist in minimising large \Delta P\textsubscript{ao}.\textsuperscript{57,89,90}

7. Other features such as humidification, pressure-relief safety valves, acoustic suppression and monitoring of volume and P\textsubscript{ao}\textsubscript{2} are desirable but not essential and have little influence on the circuit performance.

Both humidification\textsuperscript{92} and effective delivery of aerosol bronchodilators\textsuperscript{93} can be delivered through most CPAP circuits.

**Mask-PSV**

PSV involves the delivery of a patient-triggered inspiratory positive P\textsubscript{ao}\textsubscript{2} above baseline. Respiratory rate is determined by the subject, but the tidal volume is determined by a combination of the level of inspiratory P\textsubscript{ao} and patient factors including effort, W\textsubscript{el} and W\textsubscript{res}.\textsuperscript{57,92}
CPAP may be used in combination with PSV as the benefits of both are usually additive.\textsuperscript{44} Reports of PSV as a NIV mode have largely been confined to the management of hypercapnic ARF patients with chronic obstructive pulmonary disease (COPD).

Much of the early PSV research has been in intubated subjects, where it has been shown to reduce the $W_{c_{ex}}$ and assist weaning.\textsuperscript{12,42,56,95} Many of the principles of ‘invasive PSV’ also apply to its non-invasive use. The essential circuit features are:

1. A high gas flow capable of matching the PIFR so that the desired inspiratory $P_{aw}$ level can be sustained.
2. A pressure or flow sensing device to identify early spontaneous inspiratory effort and trigger inspiratory pressure. This can be achieved either by sensing a drop in circuit pressure (pressure-trigger) or a rise in circuit flow (flow-triggering).
3. A pressure or flow sensing device to identify the completion of spontaneous inspiration. This is usually achieved by sensing a fall in the circuit flow.
4. Minimal length and wide bore tubing to reduce gas turbulence and flow resistance.
5. A comfortable but air tight seal full-face or nasal mask.
6. Ability to control $F_{O_{2}}$.
7. Other desirable features include the ability to add CPAP, humidification, aerosol nebulisation, and monitoring of airway pressures and volumes.

Most current design mechanical ventilators fulfill these criteria.\textsuperscript{43} BiPAP is conceptually similar to PSV + CPAP as inspiratory (IPAP) and expiratory $P_{aw}$ (EPAP) levels may be adjusted separately. However the switch from IPAP to EPAP (and vice versa) depends upon the ventilator identifying a change in circuit flow.

**Mask-NIPPV**

NIPPV delivers controlled or assisted ventilation to the subject via a mask. Tidal volume and rate are determined by the type of ventilator (e.g. pressure- or volume-limited) and the cycling mode (patient initiated or time-cycled). This mode of NIV has been reported in the setting of chest physiotherapy, the delivery of nebulised drugs,\textsuperscript{96,97} in the treatment of ARF in COPD patients\textsuperscript{22,98,99} and domiciliary ventilation for chronic respiratory failure.\textsuperscript{99}

**Nasal or full-face mask**

Both nasal and full-face (oro-nasal) masks can be successfully used to deliver NIV.\textsuperscript{84} Desirable features of a NIV mask include: lightweight and transparent construction, a variety of sizes, a low pressure cuff (to maximise patient comfort and compliance and minimise pressure areas), and separate inspiratory and expiratory ports (to minimise gas turbulence). Low dead space is not necessary as the high gas flows tend to minimise rebreathing. A monitoring port for airway pressure and/or inspired $F_{O_{2}}$ may be advantageous with some circuits.

The full-face mask tends to produce more reliable and constant $P_{aw}$ because it is unaffected by mouth breathing. However, it impairs the patient’s ability to talk, eat and drink and expectorate. It has a greater tendency to produce pressure areas, especially on the nasal bridge. Patient tolerance of the full-face mask usually requires regular and more frequent ‘rest’ periods than the nasal mask.

Nasal masks are restricted to use with those circuits capable of rapidly delivering high gas flows sufficient to minimise the effect of an open mouth on $\Delta P_{aw}$.\textsuperscript{84} To overcome the drop in $P_{aw}$ with an open mouth it is necessary for the circuit to rapidly deliver high flows (100 - 200L minute).\textsuperscript{12,84,94} Few mask-CPAP or ventilator circuits are capable of this.

In general, the benefit of a nasal mask over a full face mask is one of greater patient comfort. They tend to have a smaller surface contact pressure area, and patients often feel less ‘claustrophobic’, they find that they can talk, cough more effectively, and even eat and drink. Hence nasal masks tend to be preferred for long-term domiciliary mask-CPAP and sleep studies.

Mask discomfort is a common cause of poor compliance with NIV. However many patients find the initial discomfort of a tight-fitting face-mask is outweighed by the symptomatic improvement resulting from reduction in $W_{b}$. Thus even sleep is possible. Some form of harness is required to seat the mask with an air-tight seal. Desirable features include: comfort, ease of application, adjustable, and reusable.

**Physiological effects of CPAP in healthy subjects**

By definition healthy subjects have no PEEP, have normal airways resistance and EELV, and have a low $W_{b}$. The primary effect of mask-CPAP on respiratory function is an increase in EELV.\textsuperscript{34,79,100-102} The pulmonary pressure-volume relationship is shifted upward\textsuperscript{40} and $W_{b}$ increased.\textsuperscript{79} In an attempt to minimise the mechanical disadvantage of the increased EELV there is heightened expiratory (abdominal) muscle activity\textsuperscript{103,104} together with an increase in tidal
In healthy animal and human subjects, CPAP reduces hepatic, renal, and muscle blood flow. There is conflicting evidence about the change in cerebral blood flow but a rise in cerebral blood flow or jugular venous pressure may increase intracranial pressure. Blood volume in the thorax is reduced and splanchnic volume is increased. Atrial natriuretic peptide levels, glomerular filtration rate and urine output decrease. The swallowing reflex is impaired during the application of mask-CPAP, although CPAP has also been shown to reduce nocturnal gastro-oesophageal reflux in patients with obstructive sleep apnoea.

In summary, the physiological effects of mask-CPAP in healthy subjects are of little benefit. This stands in contrast to the evidence of physiological benefit and clinical efficacy in critically ill patients with ARF.

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