

## Non-invasive Ventilation in Acute Respiratory Failure

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### ABSTRAK

*Salah satu dasar perawatan kritis adalah memberikan dukungan pada sistem respirasi yang memburuk. Terdapat 2 komponen utama dalam menangani gagal napas yaitu intervensi yang segera dan proses penyapihan. Banyak penelitian bertujuan untuk menentukan metode optimal ventilasi dan proses penyapihan dengan ventilasi noninvasif sebagai alternatif ventilasi invasif, pada berbagai penyebab gagal napas akut. Ventilasi noninvasif bertujuan untuk memberikan dukungan ventilasi pada paru, tanpa intubasi trakea. Ventilasi noninvasif telah menjadi alat yang penting pada penanganan gagal napas akut. Ventilasi noninvasif positif telah mengalami evolusi yang luar biasa selama beberapa dekade terakhir dan diharapkan akan memegang peranan penting dalam menangani gagal napas baik akut dan juga kronis. Terjadi perbaikan dalam pertukaran gas, mengembalikan otot pernapasan yang mengalami kelelahan, dan perbaikan klinis dengan mengurangi morbiditas dan mortalitas. Namun demikian, kontraindikasi dan kegagalan perlu diidentifikasi secara jelas, sebab penundaan intubasi endotrakeal berkaitan dengan peningkatan morbiditas dan mortalitas. Selanjutnya, meskipun pemasangan intubasi dan ventilasi mekanik merupakan hal yang umum dilakukan, komplikasi dapat disebabkan oleh proses intubasi (kerusakan jaringan lokal) dan selama ventilasi mekanik (pneumonia dan sinusitis yang berkaitan dengan ventilator) akan memperpanjang masa rawat di perawatan intensif, lama rawat di rumah sakit dan meningkatkan mortalitas pada pasien tertentu.*

**Kata kunci:** ventilasi noninvasif, ventilasi mekanik, gagal napas akut.

### ABSTRACT

*One of the cornerstones of critical care medicine is support of the failing respiratory system. The 2 major components of managing respiratory failure are the acute intervention and the weaning process. Many of the studies to determine the optimal methods of ventilation and weaning have focused on non-invasive positive-pressure ventilation as an alternative to invasive ventilation, with various causes of acute respiratory failure. Non-invasive ventilation refers to the provision of ventilatory support to the lungs, without the use of an endotracheal airway. It has emerged as an important tool in the treatment of acute respiratory failure. Non-invasive positive ventilation has undergone a remarkable evolution over the past decades and is assuming an important role in the management of both acute and chronic respiratory failure. There is improvement in gas exchange, relief of respiratory muscle fatigue, and clinical outcome with reduced morbidity and mortality. Nevertheless, contraindications and failures need to be identified early, as delaying endotracheal intubation is associated with increased morbidity and mortality. Furthermore, although it is common practice to give intubation and mechanical ventilation, complications can result from the intubation process (damage to local tissue) and during the course of ventilation (pneumonia and sinusitis associated with ventilators), prolonging stay in intensive care, length of hospital stay and mortality in selected patients.*

**Key words:** non invasive ventilation, mechanical ventilation, acute respiratory failure.

## INTRODUCTION

One of the first descriptions of a “pulmonary plus pressure machine” in 1936 describes varying success in the treatment of cardiac asthma and bronchial asthma.<sup>1</sup> Emergency physicians are often confronted with patients with acute respiratory failure. On occasion it is necessary to select therapeutic interventions, including a method of non-invasive ventilation (NIV), before a firm diagnosis is made.<sup>2</sup> Non-invasive ventilation refers to the provision of ventilatory support through the patient’s upper airway using a mask or similar device. This technique is distinguished from those which bypass the upper airway with a tracheal tube, laryngeal mask, or tracheostomy and are therefore considered invasive.<sup>3</sup> It has the advantage that it can be applied intermittently, avoids the need for sedation, and allows the patient to eat, drink and talk. The incidence of nosocomial pneumonia during NIV is lower than in intubated patients. Non-invasive ventilation has the additional advantage that it can be used with success outside the intensive care unit (ICU), thereby reducing the demand on ICU beds.<sup>4</sup>

However, NIV is not without its problems. The mask can be uncomfortable and claustrophobic for an acutely dyspnoeic patient. It can cause facial skin necrosis and, if poorly fitted, may be associated with large amounts of leakage which may compromise the efficiency of ventilation. Gastric distension is also recognised. Without the presence of an endotracheal/tracheostomy tube the lower airway cannot be easily accessed which makes bronchial toilet difficult. There has also been concern that NIV may delay intubation leading to a worse outcome. The ability to predict those likely to fail with NIV is important. Patients in whom there is a high likelihood of failure would be spared the discomfort of a trial of NIV and intubation would not be delayed. It would also be helpful in determining where NIV should take place; a patient with a high likelihood of failing, and for whom intubation would be considered appropriate, is best managed in the ICU, whereas the patient who is likely to be successfully treated with NIV can be managed on the ward.<sup>4</sup>

If ward based NIV is to be widely adopted

into clinical practice, it would be useful to identify individuals who are likely to fail to respond to NIV either before or shortly after a trial of therapy. This will allow such patients to be managed in a higher dependency area or an ICU with ready access to invasive mechanical ventilation. The failure to do this may lead to a delay in intubation and an increase in mortality.<sup>5</sup> Despite overwhelming evidence to support its use, non-invasive positive pressure ventilation (NPPV) is underused. Residents and hospitalists need to identify NPPV as a treatment option in acute respiratory failure.<sup>6</sup> This clinical review will address the use of non-invasive ventilation in acute respiratory failure, the evidence for its use in an emergency setting, and make some recommendations concerning its optimal use. In this document NIV refers to NPPV, and other less commonly used techniques such as external negative pressure or rocking beds will not be discussed.

## RESPIRATORY FAILURE

Respiratory failure is defined as a failure to maintain adequate gas exchange and is characterised by abnormalities of arterial blood gas tensions. Type 1 failure is defined by a  $\text{PaO}_2$  of  $<8$  kPa ( $<60$  mmHg) with a normal or low  $\text{PaCO}_2$ . Type 2 failure is defined by a  $\text{PaO}_2$  of  $<8$  kPa ( $<60$  mmHg) and a  $\text{PaCO}_2$  of  $>6$  kPa ( $>50$  mmHg). Respiratory failure can be acute, acute-on-chronic, or chronic. Although not always clearcut, this distinction is important in deciding on the location of patient treatment and the most appropriate treatment strategy, particularly in type 2 respiratory failure:<sup>3</sup> a). Acute hypercapnic respiratory failure: the patient will have no, or minor, evidence of pre-existing respiratory disease and arterial blood gas tensions will show a high  $\text{PaCO}_2$ , low pH, and normal bicarbonate. b). Chronic hypercapnic respiratory failure: evidence of chronic respiratory disease, high  $\text{PaCO}_2$ , normal pH, high bicarbonate. c). Acute-on-chronic hypercapnic respiratory failure: an acute deterioration in an individual with significant pre-existing hypercapnic respiratory failure, high  $\text{PaCO}_2$ , low pH, high bicarbonate.

Acute Respiratory Failure can result from a variety of etiologies. It can result from primary

pulmonary pathologies or can be initiated by extra-pulmonary pathology with high mortality rate. Causes are often multifactorial. Acute respiratory failure can be caused by abnormalities in:<sup>6</sup> a). Central Nervous System (drugs, metabolic encephalopathy, CNS infections, increased cranial pressure, obstructive sleep apnea, central alveolar hypoventilation); b). Spinal cord (trauma, transverse myelitis); c). Neuromuscular system (polio, tetanus, Myasthenia gravis, Guillain-Barre, critical care or steroid myopathy); d). Chest wall (Kyphoscoliosis, obesity); e). Upper airways (obstruction from tissue enlargement, infection, mass; vocal cord paralysis, tracheomalacia); f). Lower airways (bronchospasm, congestive heart failure, infection); g). Lung parenchyma (infection, interstitial lung disease); h). Cardiovascular system; i). Mediastinal mass, which Singh et al<sup>7</sup> reported has high mortality rate (100%).

Knowledge of arterial blood gases is essential before making a decision as to whether NIV is indicated. The patient should first be established on appropriate oxygen therapy and the arterial blood gases interpreted in light of the  $\text{FiO}_2$ . A proportion of patients who fulfil arterial blood gas criteria for NIV at the time of admission to hospital may improve rapidly with initial medical treatment with an appropriate  $\text{FiO}_2$ . It will usually then be necessary to repeat measurement of arterial blood gas tensions to see if NIV is still needed. Measurement of arterial blood gas tensions should be considered in all individuals with breathlessness of sufficient severity to warrant the use of NIV.<sup>3</sup>

### NON-INVASIVE VENTILATION

Non-invasive ventilation via mouthpiece or mask (**Figure 1**) has been used to treat acute respiratory failure of several etiologies. The main goals are to improve gas exchange and reduce the work of breathing. Non-invasive ventilation may also prevent intubation and its potentially severe complications, such as pulmonary and upper-airway infection, which are associated with a higher mortality risk.<sup>8</sup>

Non-invasive positive pressure ventilation should, however, be considered as an alternative to invasive mechanical ventilation rather than



**Figure 1.** Photograph of a patient treated with non-invasive ventilation.

its replacement. NIPPV cannot take over the use of invasive mechanical ventilation. Keys to the success of NPPV and to improving clinical outcomes of patients with acute respiratory failure are careful patient selection and a well designed clinical protocol because failure of NPPV only delays potentially more definitive therapy with invasive ventilation. Non-invasive positive pressure ventilation can be delivered using various types of ventilatory equipment and interfaces. Full-service ICU ventilators, portable bi-level pressure generators, and devices specifically designed to be used for NPPV are now available. The complete details of the application of NPPV are beyond the scope of this review, but virtually any ventilator, mode, and interface techniques may be used successfully. In **Figure 2**, Caples et al proposed a protocol used for CPAP and NIV used in acute respiratory failure.

Non-invasive positive pressure ventilation is used with patients with respiratory failure of various etiologies, but it should not be used with patients who have a low level of consciousness or noncompetent airway. A few studies have suggested that NPPV can be used for post-extubation ventilator weaning, and those studies found that NPPV reduces the need for re-intubation and sedation and reduces the occurrence of ventilator-associated pneumonia. NPPV remains controversial as a post-extubation technique, but NPPV trials have used only face mask or nasal mask, and some of the extubation failures in those trials might be attributable to

<b>Respiratory Therapy Driven Protocol For CPAP And NIV Use In Acute Respiratory Failure</b>	
I.	<u>Patient Evaluation</u> <ul style="list-style-type: none"> <li>• Review history, diagnosis, inclusion, exclusion criteria and discuss with MD</li> <li>• Ascertain whether new NIV use or an accustomed patient</li> <li>• Assess for component of chronic respiratory failure</li> </ul>
II.	<u>Indications and Goals</u> <ul style="list-style-type: none"> <li>• Choose as per patient diagnosis, level of distress, and whether ICU based</li> <li>• Decide options in event of early benefit or poor initial response</li> <li>• Reaffirm code status and patient preferences regarding alternative therapy</li> </ul>
III.	<u>Location</u> <ul style="list-style-type: none"> <li>• Determine observation and monitoring needs per patient condition and equipment used (a step-down unit may be appropriate for single organ failure or patients clearly recovering from multi-organ failure).</li> </ul>
IV.	<u>Equipment : Conditions, Initial Settings, and Targets</u> <ul style="list-style-type: none"> <li>• Airflow generator               <ol style="list-style-type: none"> <li>1. <b>CPAP.</b> Presumed obstructive sleep apnea or cardiogenic ARF for target of improved SpO<sub>2</sub> and PaCO<sub>2</sub></li> <li>2. <b>Portable Bi-Level Device.</b> Acute on chronic component for target of improved PaCO<sub>2</sub> and reduced dyspnea using visual analog scale. Use with portable battery attachment for transporting patients.</li> <li>3. <b>ICU Bi-Level Device.</b> Choose for patients in ICU, more severe distress in emergency department, or poor synchrony and need for waveform monitoring.</li> <li>4. <b>Full service ICU ventilator.</b> Severely distressed patients with poor synchrony in consideration of pressure or volume control modes</li> </ol> </li> <li>• Settings               <ol style="list-style-type: none"> <li>1. <b>CPAP.</b> Set at 10-12,5 cmH<sub>2</sub>O and titrate as needed for OSA or dyspnea</li> <li>2. <b>Portable Bi-Level Device.</b> Initial EPAP at minimum for neuromuscular disease patients. 5 cmH<sub>2</sub>O for others. May increase EPAP for OSA component or for hypoxemia. Attention to auto-PEEP, which may be counteracted by judicious use of EPAP. Initial IPAP at 8-10 cmH<sub>2</sub>O and target patient tolerance. Increase to 15-20 cmH<sub>2</sub>O as tolerance allows for relief of dyspnea and respiratory rate.</li> <li>3. <b>ICU Bi-Level Device.</b> Same EPAP and IPAP issues as for portable device but may need more aggressive titration as situation demands. Alter flow rate, sensitivity and inspiratory time to optimize synchrony.</li> <li>4. <b>Full Service ICU Ventilator.</b> Same issues as for ICU Bi-level device but may consider use of volume control and other modes.</li> <li>5. <b>Oxygen.</b> Guided by distress level and SpO<sub>2</sub>. In those at risk of worsening hypercapnia on CPAP, maintain between 88%-90%</li> </ol> </li> <li>• Masks               <ol style="list-style-type: none"> <li>1. <b>Nasal.</b> Utilize for less distressed patients and those with chronic component. Also consider nasal pillows for more claustrophobic patients</li> <li>2. <b>Full Face Mask.</b> Patients with severe distress or large oral air leaks</li> </ol> </li> </ul>
V.	<u>Monitoring</u> <ul style="list-style-type: none"> <li>• Oxymetry. All patients</li> <li>• Arterial Blood Gases. Baseline and discharge ABG's highly recommended. Useful for ICU patients needing frequent monitoring of PaCO<sub>2</sub>.</li> <li>• Ventilator with waveform monitoring for poor synchrony problems</li> </ul>
VI.	<u>Dismissal (Education and Communication)</u> <ul style="list-style-type: none"> <li>• Anticipate early for patients with chronic component</li> <li>• Review for diagnostic and reimbursement requirements</li> </ul>
VII.	<u>NNV registry/oxymetry (recommended but not obligatory)</u> <ul style="list-style-type: none"> <li>• Documentation. Log progress and outcome for future patient use and protocol quality assessment.</li> </ul>
<p>*Reproduced from Gay, PC, Hubmayr, RD; "Mechanical ventilation part II: noninvasive." In Irwin and Rippe's: Intensive Care Medicine 5th ed. 2003, p. 647-60</p>	

**Figure 2.** Respiratory therapy–driven protocol for continuous positive airway pressure (CPAP) and noninvasive ventilation (NIV) use in acute respiratory failure (ARF). ICU, intensive care unit; EPAP, expiratory positive airway pressure; PEEP, positive end-expiratory pressure; IPAP, inspiratory positive airway pressure; ABGs, arterial blood gases; OSA, obstructive sleep apnea; NNV, noninvasive nasal ventilation.<sup>10</sup>

patient dissatisfaction with the masks.<sup>11</sup>

## PATHOPHYSIOLOGICAL EFFECTS OF NON-INVASIVE VENTILATION

### Effects on the Respiratory System<sup>12</sup>

Extrinsically applied positive end expiratory pressure (ePEEP) increases alveolar size and recruitment. This expands the area available for

gas exchange, reduces intrapulmonary shunt, improves lung compliance, and decreases the work of breathing. Extrinsically applied PEEP (ePEEP) acts to negate the effects of intrinsic PEEP (iPEEP), which is the cause of dynamic airway compression and gas trapping. Ventilation is improved with beneficial effects on the alveolararterial gradient, hypercarbia and, to a



lesser extent, hypoxia. Pressure support (alone or as part of bilevel positive airway pressure, BiPAP) further augments alveolar ventilation and allows some respiratory muscle rest during the inspiratory phase.

#### **Effects on the Cardiovascular System<sup>12</sup>**

Positive end-expiratory pressure reduces venous return to the right side of the heart. Left ventricular preload, transmural pressure, and relative afterload are all decreased without altering myocardial contractility. Thus, the ejection fraction improves without an increase in myocardial oxygen consumption. It appears that those with worst ventricular dysfunction show the most significant improvement in stroke volume index with continuous positive airway pressure (CPAP). Overall, CPAP leads to a decrease in arterial pressure, heart rate, and rate-pressure product within 10 minutes, without exacerbation of hypotension.

#### **Effects on Other Systems<sup>12</sup>**

Intracranial pressure (ICP)—in patients with raised ICP there may be an increase in ICP by at least the same degree as the PEEP applied.

Renal—PEEP causes decreased sodium excretion and urine output, possibly due to raised vena caval pressure reducing cortical blood flow.

#### **PATIENT SELECTION**

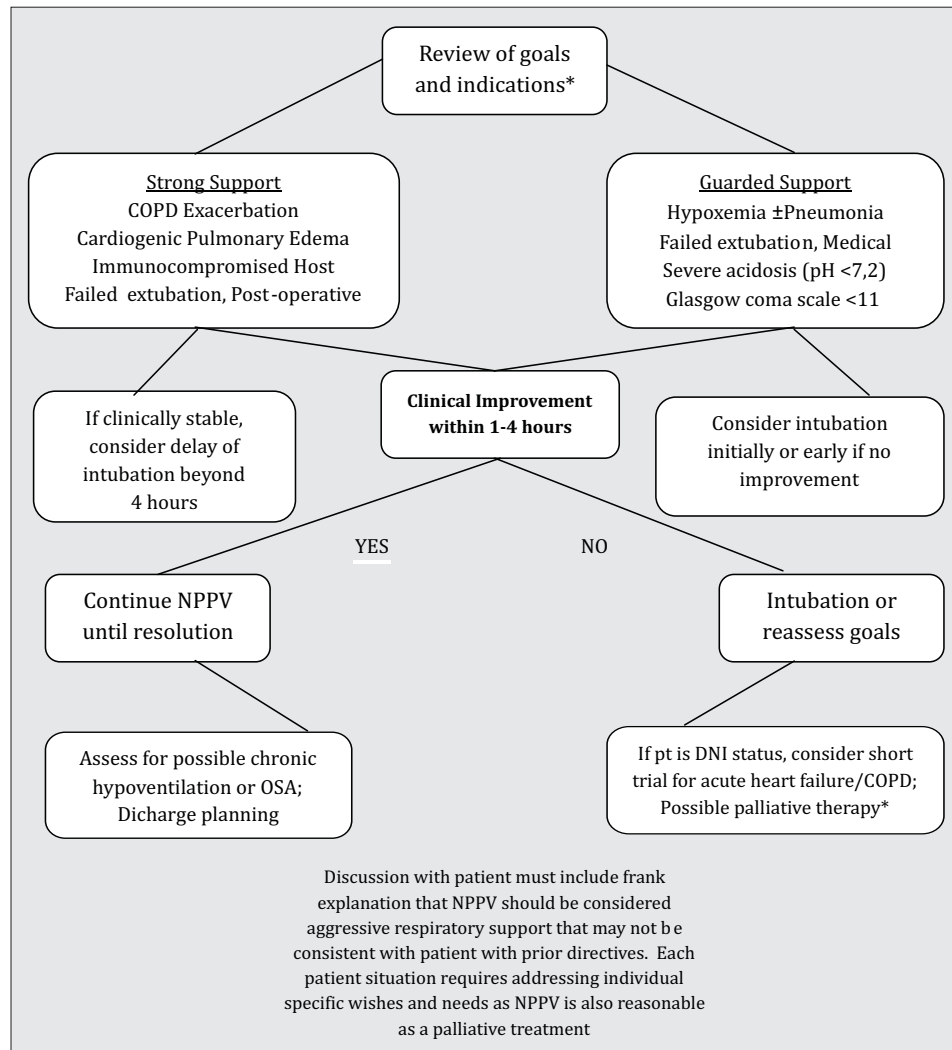
As stated previously, the success of NPPV is critically dependent on careful patient selection. There is strong evidence that patients with COPD respond well to NPPV, particularly under certain conditions. Predictors of success include younger age, unimpaired consciousness, moderate rather than severe hypercarbia and acidemia, and prompt physiologic response (improvement in heart and respiratory rates and gas exchange within 2 hrs). Confalonieri et al. recently published a logistic regression model to predict failure of NPPV in patients with acute exacerbations of COPD. Based on 1,033 consecutive COPD patients, of whom 236 (22.8%) failed NPPV, the model showed the highest risk for failure occurred in those with a Glasgow coma scale of <11, pH <7.25, and a respiratory rate of >30 breaths/min.<sup>10</sup> Based on the findings reported above, Caples and Gay<sup>10</sup>

proposed an algorithm for NPPV and urge the use of established institutional protocols that are subjected to frequent reassessment (**Figure 3**).

Studies often emphasize disease indications and the initiation process for NPPV but are perhaps lacking in statements regarding response to failure and less appropriate use of NPPV. For this reason, careful discussion of the goals and preferences of the patient and practitioners before initiating NPPV treatment should be discussed. However, a “do not resuscitate or intubate (DNR/DNI)” order should not itself preclude a trial of NPPV, which, as Levy et al. recently showed, can be associated with a favorable outcome in the setting of an acute event such as congestive heart failure or COPD exacerbation. In other settings of respiratory failure in DNR/DNI patients, NPPV may enhance patient comfort by reducing the work of breathing and may be appropriate for short-term use. However, fully informed consent should leave no ambiguity that NPPV could prolong a terminal event.<sup>10</sup>

#### **SHOULD NON-INVASIVE POSITIVE-PRESSURE VENTILATION BE USED IN ALL FORMS OF ACUTE RESPIRATORY FAILURE?**

Although NPPV has an established role as initial therapy in many forms of respiratory failure, it cannot be recommended for all patients in respiratory failure. In respiratory failure that develops soon after extubation, NPPV causes harm. But, if NPPV is used for patients who still at risk for respiratory failure after extubation, it can improve the outcomes of the patients. In the most severely ill patients with COPD or hypoxemic respiratory failure, NPPV is less likely to help. However, COPD or hypoxemic respiratory failure patients who have no contraindications to NPPV clearly shown improved outcome. In acute cardiogenic pulmonary edema, NPPV offers no advantage over simpler, less expensive interventions. In patients who require instantaneous ventilatory support or airway protection due to impaired consciousness, and for those who would interface poorly with the face mask, use of NPPV would be foolhardy. Clinical judgment, wisdom, and experience must still guide patient selection, but there probably remain many unrecognized



**Figure 3.** Noninvasive positive pressure ventilation (NPPV) for acute respiratory failure. COPD, chronic obstructive pulmonary disease; pt, patient; DNI, do not intubate; OSA, obstructive sleep apnea.<sup>10</sup>

or unrealized opportunities to improve patient care with NPPV.<sup>13</sup>

### PROBLEMS WITH NON-INVASIVE VENTILATION

Non-invasive ventilation appears to be probably free of serious complications and side effects. The primary disadvantage of non-invasive ventilation is its reliance on a spontaneously breathing patient who is able to protect their airway against the risk of aspiration. If either of these conditions are not met, endotracheal intubation and traditional ventilation is indicated. The most commonly reported complications of non-invasive ventilation are nasal bridge skin abrasions and patient intolerance of the treatment. Gastric distension and aspiration is a

frequently discussed side effect of non-invasive ventilation, but is relatively rarely described in clinical trial.

Non-invasive ventilatory systems are, generally, quite tolerant of leaks but mouth leaks with nasal masks can lead to therapeutic failure. Case reports exist of other complications of non-invasive ventilation. A variety of arrhythmias (bradycardia, ventricular tachycardia, and ventricular standstill) have been reported during CPAP but were all probably related to underlying myocardial infarction. Pneumothorax is reported to have complicated treatment of *Pneumocystis carinii* pneumonia, but these patients are at high risk of this when ventilated by traditional means. Pneumocephalus has been described in one patient receiving CPAP after weaning from

traditional ventilation who had an unrecognised base of skull fracture as a result of a motor vehicle accident. One case of bilateral tympanic rupture and otorrhagia is reported in an agitated patient coughing against CPAP. Subconjunctival emphysema and corneal abrasions have also been reported.<sup>12</sup>

## CONCLUSION

Endotracheal intubation and mechanical ventilation remain the preferred treatment in most cases of severe acute respiratory failure, especially if other systems are deranged at the same time. Continuous positive airway pressure provides relief of dyspnoea and an improvement in oxygenation in a proportion of patients, particularly those in whom hypoxaemia exists without hypercapnia. Non invasive positive pressure ventilation is feasible in patients with acute on chronic respiratory failure characterised by both hypoxaemia and hypercapnia in whom intubation is considered inappropriate, difficulties with weaning are anticipated, or long term ventilatory support might be needed. Admission to an intensive care unit can be avoided in some instances at least, and, if intubation does prove necessary, the technique may be useful as a means of helping the return of spontaneous breathing.<sup>14</sup>

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