Noninvasive positive pressure ventilation in acute hypercapnic respiratory failure

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NPPV is well established in the management of acute on chronic hypercapnic respiratory failure secondary to acute exacerbation of COPD (AECOPD), obesity hypoventilation syndrome, and restrictive thoracic disorders. Because of its design, success depends largely on patient cooperation and acceptance. The most commonly used interfaces in acute hypercapnic respiratory failure are oronasal masks. During noninvasive ventilation patients respiratory system is maintained throughout the whole respiratory cycle at a constant pressure higher than the atmospheric pressure, usually termed the positive end-expiratory pressure (PEEP). Pressure support ventilation (PSV), is the most famous mode of partial ventilatory support that during spontaneous inspiratory efforts imposes a set level of positive pressure in a patient with intact respiratory drive. Bilevel positive airway pressure (BiPAP) include PSV during inspiration and EPAP during expiration. The highest pressure reached during inspiration is called inspiratory positive airway pressure (IPAP) which equal PSV + EPAP.

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Acute hypercapnic respiratory failure of chronic obstructive pulmonary disease

Noninvasive positive pressure ventilation (NPPV) is a well-established technique used in the management of acute hypercapnic respiratory failure secondary to acute exacerbation of chronic obstructive pulmonary disease (AECOPD) [1], as shown in Fig. 1.

Physiological rationale of noninvasive positive pressure ventilation in acute exacerbation of chronic obstructive pulmonary disease

As shown in Fig. 2, the upward yellow arrow mentions the effect of continuous positive airway pressure or PEEP to counterbalance intrinsic positive end-expiratory pressure (PEEP) (decrease work of breathing with better trigger) [1]. The downward yellow arrow mentions the effect of pressure support to unload respiratory muscles and improving ventilation (decrease $PaCO_2$).

Indications of noninvasive positive pressure ventilation

NPPV should be started when pH is below 7.35 after standard medical treatment including oxygen therapy (Fig. 3) [2]. pH plays an important role for selection of location of admission and for decision of intubation as in the following:

(1) pH higher than 7.35: prophylactic use of NPPV and no benefit versus conventional therapy.
(2) pH from 7.25 to 7.35: mandatory use of NPPV in emergency department or respiratory ward but not ICU with excellent prognosis.
(3) pH below 7.25: mandatory use of NPPV and mandatory admission to ICU with good prognosis.
(4) pH below 7.20: expect the need for intubation and chance for successful NPPV is decreased.
(5) pH below 7.10: NPPV is contraindicated and intubation is mandatory.

Modes of noninvasive positive pressure ventilation in acute exacerbation of chronic obstructive pulmonary disease

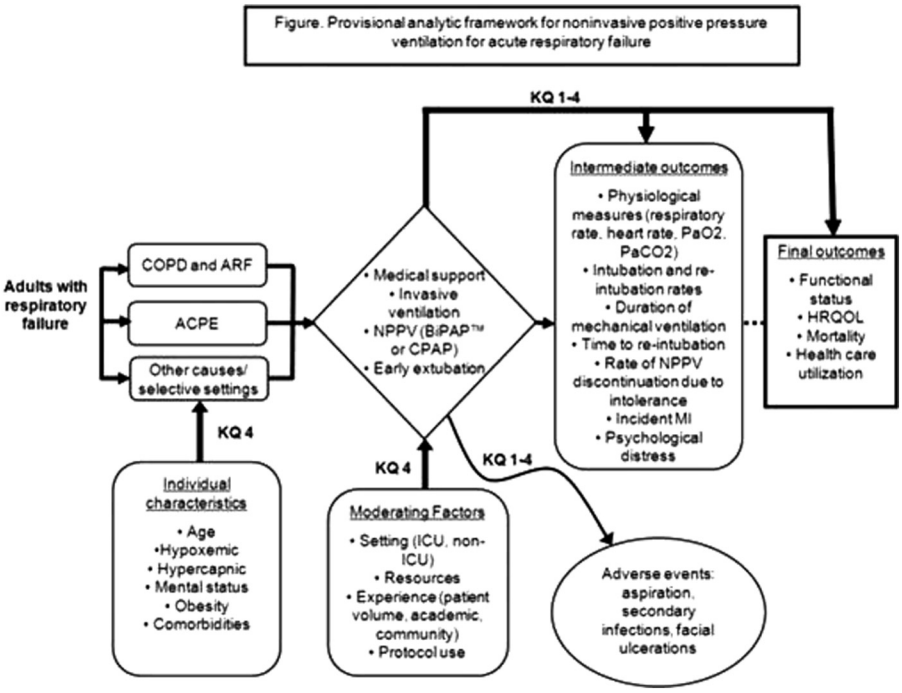
There are main and accessory modes of NPPV:

(1) The main mode acts during inspiration to augment spontaneous breathing such as pressure support (PSV), proportional assist, or volume assured pressure support (VAPS).
(2) Accessory mode uses either expiratory positive airway pressure (EPAP) or pressure control ventilation (timed mode or T).

Pressure support ventilation

This mode is a part of S or S/T modes, where S is the spontaneous mode and is called bilevel positive airway pressure and includes PSV during inspiration and EPAP during expiration (Fig. 4). The highest pressure reached during inspiration is called inspiratory positive airway pressure (IPAP), which equal PSV+EPAP [3].

Figure 1



Acute exacerbation of COPD with respiratory failure is the commonest indication for NPPV.

On the contrary, S/T (Fig. 5) is S mode plus T mode. T mode is delivered only if patient failed to initiate trigger within a time governed by backup rate.

Benefits of expiratory positive airway pressure

(1) Counterbalance auto PEEP.
(2) Maintain patency of upper airways at the end of expiration.
(3) Helps CO_2 wash from exhalation port and decrease its rebreathing.

How to set S/T mode

(1) EPAP: 5–7 cmH_2O.
(2) Set pressure support at least 8 cmH_2O and increased in increments of 2 cmH_2O every 30 min targeting tidal volume of 8 ml/kg and SPO_2 above 90%.
(3) IPAP is preferred not exceeding 20 cmH_2O to prevent gastric insufflation.

Proportional assist ventilation

The second common mode used in acute exacerbation of COPD (Fig. 6).

(1) It is proportional to patient effort by delivering volume assist and flow assist according to elastance and resistance [4]. Thus, pressure, volume, and flow are variable from breath to breath as shown in below figure.
(2) It is indicated if there is good triggering, otherwise S/T is indicated.

Volume assured pressure support

(1) A recent dual mode with target tidal volume (average VAPS) or target alveolar ventilation (intelligent VAPS).
(2) It is thought to augment alveolar ventilation with enhanced CO_2 wash and improved acidosis.

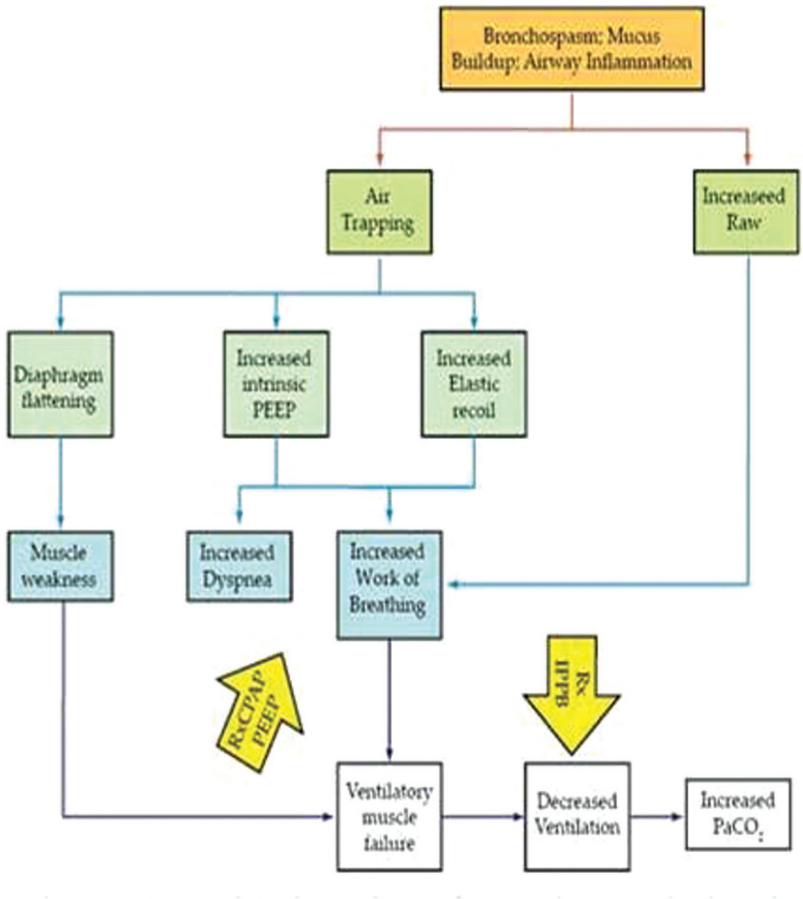
Monitoring during noninvasive positive pressure ventilation

(1) Continuous:
  (a) SpO_2.
  (b) ECG.
  (c) Respiratory rate.
(2) Intermittent:
  (a) Arterial blood gases.
  (b) Leaks.
  (c) Patient–ventilator synchrony.

Acute hypercapnic respiratory failure of obesity hypoventilation syndrome

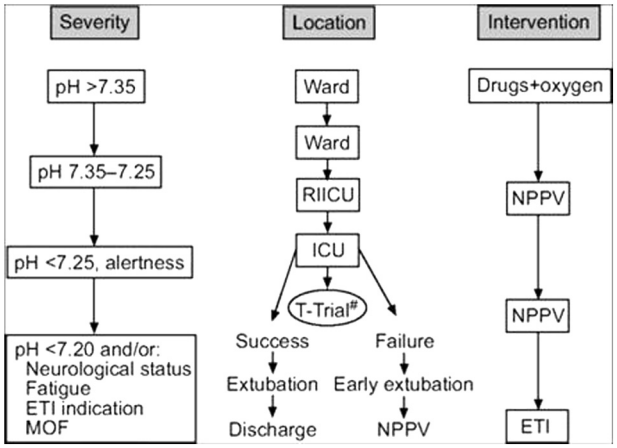
In the Western world, the prevalence of obese patients in ICUs is increasing. Additionally, morbid

Figure 2



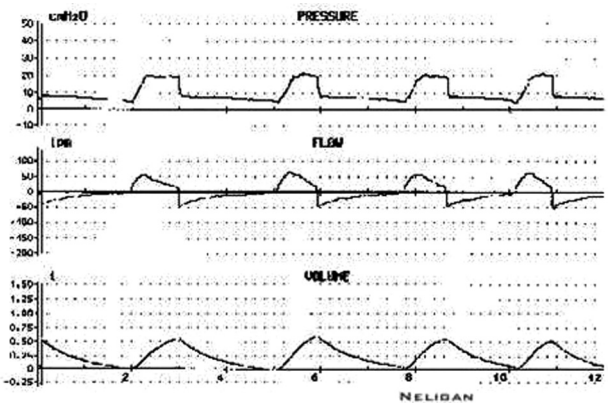
Flow chart for physiological rationale of application of NPPV in acute hypercapnic respiratory failure of COPD.

Figure 3



Flow chart for the application of NPPV in acute exacerbation of COPD, according to the severity of respiratory acidosis.

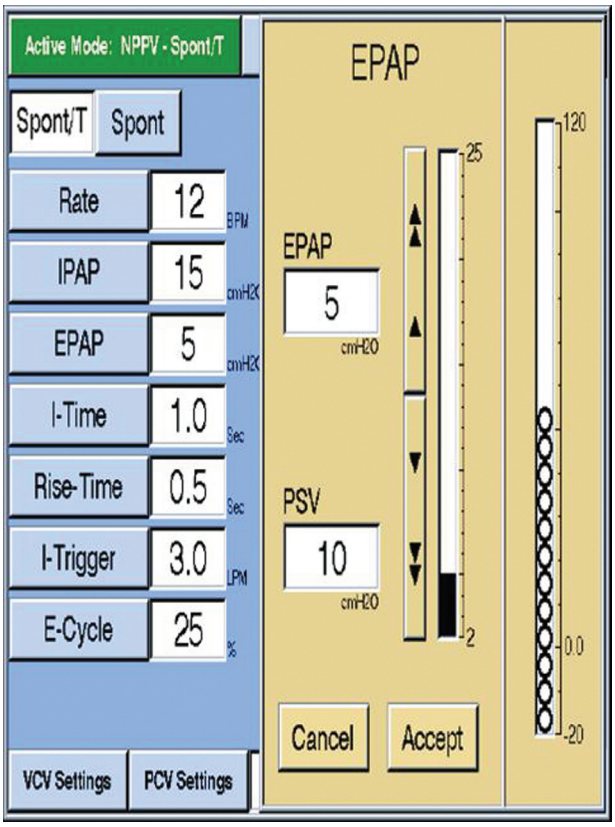
Figure 4



Pressure, flow, and volume tracers during pressure support.

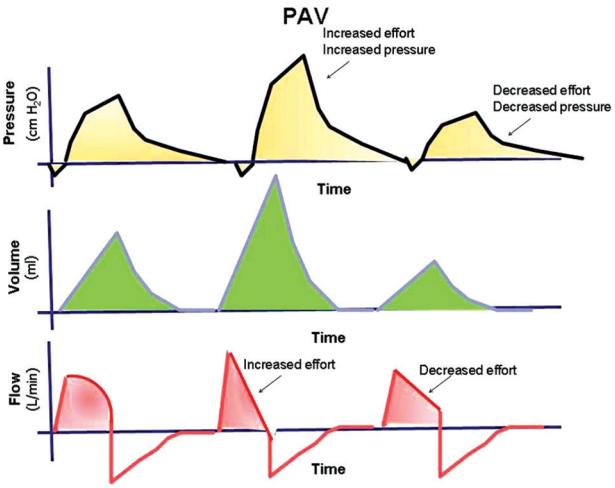
obesity has dramatic consequences on pulmonary function. Therefore, respiratory physicians and intensivistsare more likely to manage a larger number of acute hypercapnic respiratory failure (AHRF) episodes in patients with a BMI of at least 30 [5]. Cor pulmonale is a major cause of ICU admission, which requires mechanical ventilation with higher mortality in obese compared with nonobese patients. It is, therefore, surprising that experience of AHRF in obese patients has rarely been reported in the literature and, consequently, evidence-based guidelines remain to be established. There are cumulating reports that suggest that NPPV plays a key role in the treatment of obese patients with AHRF [6].

Figure 5



Different parameters for setting S/T mode.

Figure 6



Pressure, flow, and volume tracers during proportional assist ventilation.

Physiological rationale of noninvasive positive pressure ventilation in obesity hypoventilation syndrome

To control upper airways obstruction and overcome the respiratory muscle load, a high EPAP and high inspiratory driving pressure are required.

Specifically, the EPAP should set to optimise respiratory mechanics by modifying lung volume and the operating position on pressure volume curve as well as overcoming upper airways obstruction (reduce work of breathing) [7].

IPAP should be titrated to ensure satisfactory chest wall excursion with supplemental oxygen achieving target saturation from 90 to 92% [7].

Modes of noninvasive positive pressure ventilation in obesity hypoventilation syndrome

The optimal ventilatory mode is pressure support, using turbine-driven ventilators or ICU ventilators. A backup frequency rate is required in most cases, owing to the likelihood of sleep central apneas [8].

If a single circuit is used, it should be equipped with an expiratory valve or an intentional calibrated leak [7]. In this previous case, a slight expiratory positive pressure is required to limit CO_2 rebreathing. Clinical series show that obesity hypoventilation syndrome (OHS) patients with AHRF require high levels of inspiratory positive pressures (often 22 cmH_2O) owing to low thoracic compliance in this disease [8].

Another ventilatory mode in these patients is flow-preset mode that allows a better control of delivered volumes. AHRF in obese patients is one of the last medical situations where pressure support may be less effective than flow-preset modes [9]. After discharge, most OHS patients are managed with nocturnal bilevel NPPV without additional oxygen.

Noninvasive positive pressure ventilation settings in obesity hypoventilation syndrome

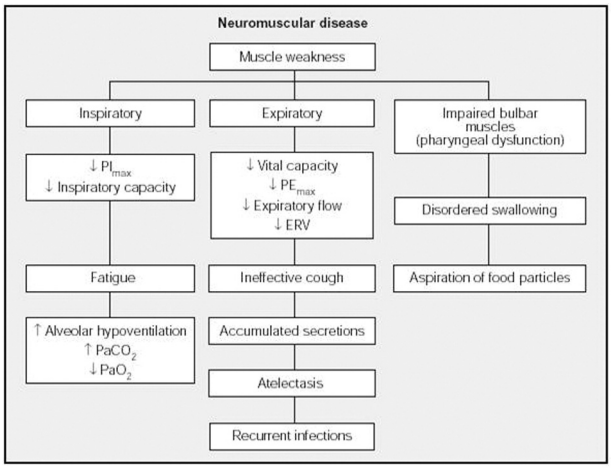
(1) EPAP set at 8–10 cmH_2O aiming $SpO_2 \geq 90\%$.
(2) IPAP set at 18–24 cH_2O aiming pH ≥7.35 and tidal volume 8–10 ml/kg of ideal body weight [10].
(3) Inspiratory time limit 1.4 s.

Acute hypercapnic respiratory failure of restrictive thoracic disorders

Patients with severe kyphoscoliosis and acute respiratory failure exhibit marked decrease of pulmonary compliance but, contrary to in COPD, increase in airway resistance, and intrinsic PEEP seem to play only a secondary role [11]. Because cough is not impaired like in neuromuscular diseases (NMD), secretion management is not so critical in this context, and management of ARF may be easier [11].

In neuromuscular disorders (NMD) (Fig. 7), the normality of the respiratory function requires the

Figure 7



Pathophysiology of muscle weakness in neuromuscular disease.

integrity of three main respiratory muscles: (i) inspiratory muscles, responsible for ventilation; (ii) expiratory muscles, involved in the ability to cough; and (iii) bulbar muscles, which protect against the risk of aspiration [12]. Acute NMD (such as Guillain–Barre's syndrome) together with the ICU acquired NMD are the most frequent NMD associated with ARF [13].

Concerning the mode of NPPV, it is preferable to use volume-cycled ventilators in assist control, with high tidal volumes, for sufficient lung expansion and to allow air-stacking for coughing. This can be performed during the day using a mouthpiece and at night with a nasal or oral interface [12].

When patients with amyotrophic lateral sclerosis under home mechanical ventilation need to be hospitalized because of ARF, there are some technical as well as ethical aspects that need to be considered. It needs to be confirmed if all noninvasive respiratory aids were optimized at home, if the patient has refused tracheostomy, if bulbar impairment is severe, and if the risk of aspiration is high. Lung function status, previous to the decompensation, can be helpful in the decision making [14].

Another issue is when nonbulbar patients with NMD are intubated because of failure of noninvasive ventilation or because of a more-conventional approach. In this context, it is a common attitude to gradually reduce the support of the ventilator until the patient is weaned [15]. Unfortunately patients with NMD with a pre-existing respiratory dysfunction often fail to wean off invasive ventilation and are almost invariably tracheostomized [14].

Noninvasive positive pressure ventilation in patients with an acute pneumothorax

For patients with NMD, or any other condition in which the risk of acute pneumothorax is high who require NPPV [16], the following strategies can be used:

(1) Insert chest drain.
(2) Use pressure preset, pressure limited ventilator to reduce peak airway pressure.
(3) Use the lowest IPAP and EPAP settings that are comparable to arterial blood gas tensions and symptoms.
(4) Temporarily withdraw NPPV.

Contraindications of noninvasive positive pressure ventilation

The boundaries for the use of NPPV continue to expand. NPPV is not appropriate in well-documented end-stage disease or when several co-morbidities are present.

There are no absolute contraindications, although a number of them have been suggested. In part, these 'contraindications' have been determined by the fact that they were exclusion criteria for the controlled trials [1]. It is therefore more correct to state that NPPV is not proven in these circumstances rather than that it is contraindicated.

Whether NPPV is contraindicated or not must depend on individual circumstances. For instance, if invasive ventilation is not considered appropriate but NPPV would be acceptable, there is nothing to be lost by a trial of NPPV and there are no contraindications [2].

The possible contraindications for NPPV can be summarized as follow:

(1) Facial trauma/burns.
(2) Recent facial, upper airway, or upper gastro-intestinal tract surgery.
(3) Fixed obstruction of the upper airway.
(4) Inability to protect airway.
(5) Cardiorespiratory arrest.
(6) Uncontrolled life-threatening arrhythmia.
(7) Hemodynamic instability.
(8) Severe co-morbidity.
(9) Coma.
(10) Vomiting and hematemesis.
(11) Bowel obstruction.
(12) Undrained pneumothorax

NPPV may be used, despite the presence of these contraindications (except for facial trauma/burn, and fixed obstruction of the upper airway), provided contingency plans for tracheal intubation have been made, or if it is to be the 'ceiling' of treatment in patients who are not candidates for intubation.

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Conflicts of interest
There are no conflicts of interest.

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