Noninvasive positive pressure ventilation

Positive-pressure ventilation delivered through a mask has become the predominant method of providing noninvasive ventilatory support and is the focus of this and subsequent sections. Early bedside physiologic studies in healthy patients and in patients with respiratory conditions document suocesfal ventilatory support.[1]

Ventilatory svpport can be achieved through a variety of interfaces (mouth piece or nasal, face, or helmet mask using a variety of ventilatory modes (eg, volume ventilation, pressure support, biIevel positive airway pressure [BiPAP], proportional-assist ventilation [PAV], continuous positive airway pressure [CPAP]) with either ventilators dedicated to noninvasive ventilation (NIV) or [those] capable of providing support through an endotracheal tube or mask. Older models of noninvasive ventilators required oxygen to be bled into the system, but current models incorporate oxygen blenders for precise delivery of the fraction of inspired oxygen (FI02).

Absolute contraindications of NIV

- Coma
- Cardiac arrest
- Respiratory arrest
- Any condition requiring immediate intubation
- Other contraindications (rare exceptions)
- Cardiac instability
- Shock and need for pressor support
- Ventricular dysrhythmias
- Complicated acute myocardial infarction
- GI bleeding - Intractable emesis and/or uncontrollable bleeding
- Inability to protect airway
- Impaired cough or swallowing
- Poor clearance of secretions

Patient inclusion criteria

- Patient cooperation (an essential component that excludes agitated, belligerent, or comatose patients)
- Dyspnea (moderate to severe, but short of respiratory failure)
- Tachypnea (>24 breaths/min)
- Increased work of breathing (accessory muscle use, pursed-lips breathing)
- Hypercapnic respiratory acidosis (pH range 7.10-7.3 5)
- Hypoxemia (Pa02/FIO2 <200 mm Hg, best in rapidly reversible causes of hypoxemia).[2]

Suitable clinical conditions for noninvasive ventilation (most patients)

- Chronic obstructive pulmonary disease
- Cardiogenic pulmonary edema
- Suitable clinical conditions for noninvasive ventilation (selected patients)
- After discontinuation of mechanical ventilation (COPD)
- Community-acquired pneumonia (and COPD)
- Asthma
- Immunocompromised state
- Postoperative respiratory distress and respiratory failure
- Do-not-intubate status
- Neuromuscular respiratory failure
- Decompensated obstructive sleep apnea/cor pulmonale
- Cystic fibrosis
- Acute respiratory distress syndrome
- Mild Pneumocystic carinii pneumonia

Application of Noninvasive Ventilation[3,4]

Several considerations can enhance the likelihood of successful noninvasive ventilation (htIV). In addition to these factors, the experience and expertise of front-line health care providers, specifically nursing and respiratory therapy staff, cannot be underestimated. This is not a concern in hospitals where noninvasive ventilation is well established, but it is an important factor in facilities where noninvasive ventilation has been infrequently administered or not used at all.

Location of application

- ICU (especially if possibility of intubation)
- Step-down unit (lower severity of illness)
- Moderately severe COPD (pH >7.30)
- Do-not-intubate status
- Intermittent or nocturnal ventilatory support
- Ward setting (not recommended if intubation is a consideration)
- Suitable **in** specialized units
- Same considerations as step-down unit

- Emergency department - Local considerations, expertise may mirror ICU or step-down unit

Patient interfaces

1. Masks 2. Ventilators.

Orofacial masks (general advantages)

- Best suited for less cooperative patients
- Better in patients with a higher severity of illness
- Better for patients with mouth-breathing or pursed-lips breathing
- Better in edentulous patients
- Generally more effective ventilation

Orofacial masks (cautions, disadvantages)

- Claustrophobic
- Hinder speaking and coughing
- Risk of aspiration with emesis

Nasal masks (general advantages)

- Best suited for more cooperative patients
- Better in patients with a lower severity of illness
- Not claustrophobic
- Allows speaking, drinking, coughing, and secretion clearance
- Less aspiration risk with emesis
- Generally better tolerated

Nasal masks (cautions, disadvantages)

- More leaks possible (eg, mouth-breathing or edentulous patients)
- Effectiveness limited in patients with nasal deformities or blocked nasal passages.

Ventilators

The choice of ventilators available to provide noninvasive ventilatory support has continued to expand. Early noninvasive ventilatory support was applied using either large bedside critical care volume ventilators or smaller volume or pressure specialty ventilators devoted to noninvasive ventilation. While the critical care ventilators had more options, they were also less tolerant of leaks. The specialty ventilators bad fewer options and range, but they were more leak tolerant[5,6,7].

Many critical care ventilators currently in use also have a noninvasive ventilation option, either as part of the original device or available as an upgrade option. The ideal device is dependent of a number of factors, including familiarity by staff and available options. The differences between the bedside critical care ventilator and specialty noninvasive ventilator continue to diminish as differences related to ventilator options, range of support, and leak tolerance are corrected in both devices. However, most hospitals continue to provide noninvasive support with the specialty ventilator.

Modes of ventilation

Choosing the initial mode of ventilation is based in part on past experience, in part on the capability of ventilators available to provide support, and in part on the condition being treated. Most patients who are provided noninvasive ventilation are provided support with pressure ventilation, with continuous positive airway pressure (CPAP), which is the most basic level of support. CPAP may be especially useful in patients with congestive heart failure or obstructive sleep apnea.

Bilevel positive airway pressure (BiPAP) is probably the most common mode of support and requires provision of inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). The difference between IPAP and EPAP is a reflection of the amount of pressure support ventilation provided to the patient, and EPAP is synonymous with positive end-

expiratory pressure (PEEP). Some noninvasive ventilation is provided using proportional-assist ventilation (PAV), which provides flow and volume assistance with each breath.

While volume ventilators can be used to provide noninvasive ventilatory support, the previously described modes are preferred because they provide better patient comfort and synchrony and are more tolerant of the leaks that accompany all noninvasive ventilatory interfaces.

Initial ventilator settings and adjustments

Adequate ventilation and oxygenation, correction of respiratory failure, and adequate patient tolerance and comfort are the primary goals of noninvasive ventilation, and adjustments are made to achieve these endpoints. Initial settings focus on achieving adequate tidal volumes, usually in the range of 5-7 mL/kg. Additional support is provided to reduce the respiratory rate to less than 25 breaths/minute. Oxygen is adjusted to achieve

adequate oxygenation, with a pulse oximetry goal of greater than 90%. Serial arterial blood gas measurements are essential to monitor the response to therapy and to guide further adjustments in the ventilator.

InitialIPA.P/EPAP settings

- Start at 10 cm water/5 cm water
- Pressures less than 8 cm water/4 cm water not advised as this may be inadequate
- Initial adjustments to achieve tidal volume of 5-7 mL/kg (PAP and/or EPAP)
- Subsequent adjustments based on arterial blood gas values
- Increase IPAP by 2 cm water if persistent hypercapnia
- Increase IPAP and EPAP by 2 cm water if persistent hypoxemia
- Maximal IPAP limited to 20-25 cm water (avoids gastric distension, improves patient comfort)
- Maximal EPAP limited to 10-15 cm water
- F102 at 1.0 and adjust to lowest level with an acceptable pulse oximetry value
- Back up respiratory rate 12-16 breaths/minute.[8]

Predictors of successful noninvasive ventilation

Predictors of success - Response to trial of NIV (1-2 h)

- Decrease in PaC02 greater than 8 mm Hg
- Improvement in pH greater than 0.06
- Correction of respiratory acidosis

Predictors of failure

Severity of illness

- Acidosis (pH <7.25)
- Hypercapnia (>80 and pH <7.25)
- Acute Physiology and Chronic Health Evaluation II (APACHE II) score higher than 20 *Level of consciousness*
- Neurologic score (>4 = stuporous, arousal only after vigorous stimulation; inconsistently follows commands)
- Encephalopathy score (>3 = major confusion, daytime sleepiness or agitation)
- Glasgow Coma Scale score lower than 8

Failure of improvement with 12-24 hours of noninvasive ventilation.[9,10]

Late failures (>48 h after initiation of noninvasive ventilation) Admission predictors offailure

- Lower functional status (Activity score < 2 = dyspnea light activity)
- Initial acidosis (pH <7.22)
- Hospital complications (pneumonia, shock, coma)

Noninvasive Ventilation in COPD

Patients with underlying chronic obstructive pulmonary disease (COPD) who present with an exacerbation of their COPD and hypercapnic respiratory distress or respiratory failure are the group most likely to be successfully treated with noninvasive ventilation (NIV). Exacerbations increase the respiratory load in these patients, exceeding their ability to adequately ventilate through a variety of mechanisms, including increasing hyperinflation with decreased diaphragmatic excursion and strength, increasing intrinsic positive end-expiratory pressure (PEEP), ineffective or inadequate tidal volume generation, respiratory patterns, and increased respiratory frequency. Noninvasive ventilation effectively unloads the respiratory muscles, increasing tidal volume, decreasing the respiratory rate, and decreasing the diaphragmatic work of breathing, which translates to an improvement in oxygenation, a reduction in hypercapnia, and an improvement in dyspnea.[11]

Non-invasive ventilation (NN), within both the intensive care unit (ICU) and the ward environment, has been shown in randomised controlled trials (RCTs) and systematic reviewsto reduce intubation rate and mortality in COPD patients with decompensated respiratory acidosis (pH <7.35 and PaC02 >6 kPa) following immediate medical therapy. NIV should therefore be considered within the first 60 minutes of hospital arrival in all patients with an acute exacerbation of COPD in whom a respiratory acidosis persists despite maximum standard medical treatment, which includes:

controlled oxygen to maintain Sa02 88-92%
nebulised salbutamol 2.5-5 mg
nebulised ipratroprium 500 µg
prednisolone 30 *mg*
antibiotic agent (when indicated).

Recommended inclusion and exclusion criteria for potential NIV are shown below.

Clinical inclusion and exclusion criteria for NIV.

Inclusion criteria

Primary diagnosis of COPD exacerbation (known diagnosis or history and examination consistent with diagnosis)

- Able to protect airway
- Conscious and cooperative
- Consider NN if unconscious and endo-tracheal intubation deemed inappropriate or NIV to be provided in a critical care setting.

Exclusion criteria

- Life-threatening hypoxaemia
- Severe co-morbidity
- Confusion/agitation/severe cognitive impairment
- Facial burns/trauma/recent facial or upper airway
- surgery
- Vomiting
- Fixed upper airway obstruction
- Undrained pneumothorax
- Upper gastrointestinal surgery
- Inability to protect the airway
- Copious respiratory secretions
- Haemodynamically unstable requiring
- inotrnpes/pressods (unless in a critical care unit)
- Patient moribund
- Bowel obstruction
- NIV is not the treatment of choice for patients whose
- primary diagnosis is heart failure or pneumonia but
- may be used in COPD patients with these
- complications if escalation to intubation and
- ventilation is deemed inappropriate[12,13].

Patient selection

NIV should be considered in. all patients with an acute exacerbation of COPD in whom a respiratory acidosis (pH <7.35 Pa.C02 >6 kPa), persists despite immediate maximum standard medical treatment on controlled oxygen therapy for no more than l hour. Set-up

The decision to commence NIV should be made by a doctor of specialty training (ST) level 2 or above who is competent to do so. A trained and competent healthcare professional should initiate NIV.

The patient should be in a sitting or semi-recumbent position in bed and the following are recommended:

- A full-face mask should be used for the first 24 hours, followed by switching to a nasal mask if preferred by the patient.
- An initial inspiratory positive airway pressure (IPAP) of 10 cm H20 and expiratory positive airway pressure (EPAP) of 4-5 cm H20 should be used.
- IPAP should be increased by 2-5 cm increments at a rate of approximately 5 cm H20 every 10 minutes, with a usual pressure target of 20 cm H20 or until a therapeutic response is achieved or patient tolerability has been reached.
- Oxygen, when required, should be entrained into the circuit and the flow adjusted to achieve the target saturation, usually 88-92%.
- Bronchodilators, although preferably administered off NIV, should as necessary be entrained between the expiration port and face mask.
- If a nasogastric tube is in place, a fine bore tube is preferred to minimise mask leakage.

Monitoring

Monitoring should include a mixture of physiological measures and clinical parameters.

These parameters should be used to assist in formulating a management plan and within the first 4 hours of NIV assist in the decision as to the need to escalate to intubation.

Staff involved in the care and monitoring of NN patients should be appropriately trained and experienced.

The following should be recorded and be used to formulate an iterative management plan:

- Baseline observations:
  - arterial blood gas (ABG)
  - respiratory rate
  - heart rate

- Continuous pulse oximetry and electrocardiogram (ECG) recording during the first 12 hours
- Repeat ABGs:
  - after 1 hour of NIV therapy and 1 hour after every subsequent change in settings
  - after 4 hours, or earlier in patients who are not improving clinically
- Frequent clinical monitoring of acutely ill patients:
  - every 15 minutes in the first hour
  - every 30 minutes in the I- to 4-hour period
  - hourly in the 4- to 12-hour period
- Observations including:
  - respiratory rate, heart rate
  - level of consciousness, patient comfort
  - chest wall movement, ventilator synchrony, accessory muscle use[13,14].

Duration of treatment

6.1 Patients who benefit from NIV during the first 4 hours of treatment should receive NIV for as long as possible (a minimum of 6 hours) during the first 24 hours.

- 6.2-Treatment should last until the acute cause has resolved, commonly after about 3 days.

6.3 In patients in whom NIV is successful (pH 7.35 achieved, resolution of underlying cause and symptoms, respiratory rate normalised) following the first 24 hours or longer, it is appropriate to start a weaning plan:

- gradual reduction of the duration of NIV should be determined by clinical improvement
- the use of a profoinia to chart physiological indices has been shown to improve successful weaning from NIV[14].

Weaning

Initially weaning should be during the day with extended periods off the ventilator for meals, physiotherapy, nebulised therapy etc. After successfully weaning during the day, many patients will require an additional night on NIV.

The weaning strategy should be documented in the medical and nursing records. The following is recommended:

- continue NIV for 16 hours on day 2 A
- continue NI:V for 12 hours on day 3 including 6-8 hours overnight use
- discontinue NIV on day 4, unless continuation is clinically indicated. Note that some patients may:
- show at an earlier stage that they no longer require NIV and self-wean
- improve rapidly, prompting a clinical decision to wean early
- require long-term nocturnal support, indicated following assessment by the respiratory team[15].

Palliation

Palliation of symptoms is appropriate in some patients, where standard medical treatment and NIV fails and a decision has been made and documented not to escalate to intubation and mechanical ventilation, or where a patient chooses not to have NIV or other interventionist treatment:

- If the patient gains symptom relief, continued NIV may be appropriate for palliation of breathlessness, but normally would be withdrawn.
- Opiates and benzodiazepines can be used to treat breathlessness in this situation.
- The palliative care team should be involved and a suitable care pathway followed after discussion with the patient and family.

Conclusion

The indications for NPPV are not as clear in patients with non-COPD causes of acute respiratory failure. For acute pulmonary edema, CPAP alone drastically reduces the need for intubation, although studies have not demonstrated reductions in morbidity or mortality rates. NPPV avoids intubation and reduces complication rates in patients with hypoxemic respiratory failure, but more controlled trials are needed to establish precise indications. In the meantime, NPPV administration to patients with non-COPD causes of acute respiratory failure appears to be safe as long as patients are selected carefully with particular attention to the exclusion of inappropriate candidates.

For chronic respiratory failure, a wide consensus now favors the use of NPPV as the ventilatory mode of first choice for patients with neuromuscular diseases and chest wall deformities, despite a lack of randomized controlled trials. Central hypoventilation and failure of obstructive sleep apnea to respond to CPAP are also considered acceptable indications, although evidence to support these latter applications is sparse. For patients with severe stable COPD, some evidence supports the use of NPPV in severely hypercapnic patients, particularly if there is associated nocturnal hypoventilation. However, the data are conflicting and do not permit the formulation of firm selection guidelines.

References:

1. Corrado A, Gorini M, Melej R, et al. Iron lung versus mask ventilation in acute exacerbation of COPD: a randomised crossover study. Intensive Care Med. 2009;35(4):648-55. [Medline].
2. Brochard L, Isabey D, Piquet J, et al. Reversal of acute exacerbations of chronic obstructive lung disease by inspiratory assistance with a face mask. N Engl J Med. 1990;323(22):1523-30. [Medline].
3. Brochard L, Mancebo J, Wysocki M, et al. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. N Engl J Med. Sep 28 1995;333(13):817-22. [Medline].
4. Plant PK, Owen JL, Parrott S, Elliott MW. Cost effectiveness of ward based non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease: economic analysis of randomised controlled trial. BMJ. 3 2003;326(7396):956. [Medline].
5. Keenan SP, Sinuff T, Cook DJ, Hill NS. Which patients with acute exacerbation of chronic obstructive pulmonary disease benefit from noninvasive positive-pressure ventilation? A systematic review of the literature. Ann Intern Med. 2003;138(11):8b1-70. [Medline].
6. [Best Evidence] Lightowler JV, Wedzicha JA, Elliott MW, Ram FS. Non-invasive positive pressure ventilation to treat respiratory failure resulting from exacerbations of chronic obstructive pulmonary disease: Cochrane systematic review and meta-analysis. BMJ. 2003;326(7382):185. [Medline].
7. Confalonieri M, Garuti G, Cattaruzza MS, et al. A chart of failure risk for noninvasive ventilation in patients with COPD exacerbation. Eur Respir J. 2005; 25(2): 348-55. [Medline].
8. Diaz GG, Alcaraz AC, Talavera JC, et al. Noninvasive positive-pressure ventilation to treat hypercapnic coma secondary to respiratory failure. Chest. Mar 2005;127(3):952-60. [Medline].
9. Scala R, Naldi M, Archinucci I, Coniglio G, Nava S. Noninvasive positive pressure ventilation in patients with acute exacerbations of COPD and varying levels of consciousness. Chest. 2005;128(3):1657-66. [Medline].
10. Girou E, Brun-Buisson C, Taille S, Lemaire F, Brochard L. Secular trends in nosocomial infections and mortality associated with noninvasive ventilation in patients with exacerbation of COPD and pulmonary edema. JAMA. 2003;290(22):2985-91. [Medline].
11. Girou E, Schortgen F, Delclaux C, et al. Association of noninvasive ventilation with nosocomial infections and survival in critically ill patients. JAMA. 2000;284(18):2361-7. [.Medline].-
12. [Guideline] Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. National Guideline Clearinghouse. 2008.
13. Masip J, Roque M, Sanchez B, Fernandez R, Subirana M, Exposito JA. Noninvasive ventilation in acute cardiogenic pulmonary edema: systematic review and meta-analysis. JAMA. 2005;294(24):3124-30. [Medline].
14. Gray A, Goodacre S, Newby DE, Masson M, Sampson F, Nicholl J. Noninvasive ventilation in acute cardiogenic pulmonary edema. N Engl J Med. 2008;359(2):142-51. [Medline].
15. British Thoracic Society: n chronic obstructive pulmonary disease: management of acute type 2 respiratory failure: National Guidelines: October 2008.