Noninvasive Positive Pressure Ventilation

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KEYWORDS

• Noninvasive positive pressure ventilation • Continuous positive airway pressure
• Bilevel positive airway pressure • Respiratory failure

HOSPITAL MEDICINE CLINICS CHECKLIST

1. Obtain arterial blood gas (ABG) to determine the acid-base disorder at baseline; this will aid in the selection of a noninvasive positive pressure ventilation (NIPPV) modality. In the case of hypercapnic respiratory failure, an ABG should be repeated within an hour of initiating NIPPV and compared with baseline.
2. Enlist the help of a respiratory therapist whenever possible.
3. Knowledge of the patient's admitting diagnosis, as well as medical history (namely any history of cardiac or pulmonary disorders), will allow for the prompt initiation of the appropriate intervention.
4. A focused physical examination may yield critical information in the development of a full clinical picture.
5. Consider the need for intubation (invasive mechanical ventilation) sooner rather than later.
6. Evaluate for any contraindication to NIPPV before initiation.
7. Consider transferring patients requiring NIPPV to a higher level of care (ie, specialized care unit or intensive care unit), as they need close ongoing monitoring of their respiratory status.

DEFINITIONS

1. What is noninvasive positive pressure ventilation?

Noninvasive positive pressure ventilation (NIPPV) is a form of ventilatory assistance delivered via a noninvasive interface (ie, full face mask, nasal mask/pillows), as opposed to invasive ventilation that is delivered through an endotracheal tube or tracheostomy.

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2. What differentiates continuous positive airway pressure from bilevel positive airway pressure?

Continuous positive airway pressure (CPAP) is a mode of noninvasive ventilation that supplies a constant positive pressure during inspiration and expiration, which opens obstructed upper airways to allow ventilation, raises functional residual capacity, and potentially opens deluged alveoli in patients with cardiogenic pulmonary edema. CPAP also decreases preload by reducing the volume of venous return to the right heart[1] and decreases the left ventricular transmural pressure, causing a reduction in afterload and an increase in cardiac output in those with acute congestive heart failure (CHF).[2]

Bilevel positive airway pressure (BPAP) is a mode that cycles between inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). This mode of noninvasive ventilation provides a combination of pressure support (via the higher IPAP) in addition to EPAP, and thereby reduces the work of breathing as it augments the patient's respiratory effort. By reducing the work of breathing and assisting in achieving higher tidal volumes, BPAP can improve hypercapnic respiratory failures during exacerbations of chronic obstructive lung disease (COPD), and assist in the management of CHF exacerbations.[3,4]

Potential benefits of NIPPV include improved gas exchange, decreased work of breathing, avoidance of intubation, facilitation of weaning/extubation, improved mortality, and decreased rate of nosocomial infections (in comparison with invasive ventilation). The magnitude of these benefits depends on the underlying cause of respiratory failure as well as patient variance.

INDICATIONS AND CONTRAINDICATIONS FOR NIPPV

1. What are the indications for NIPPV?

A trial of NIPPV is typically warranted in patients with respiratory failure who lack an indication for immediate intubation, without contraindications to NIPPV and especially if they have a condition responsive to NIPPV (ie, hypercapnic respiratory failure, acute exacerbations of COPD, acute cardiogenic pulmonary edema) (**Table 1**). Other

Table 1
Indications and contraindications for the use of NIPPV

Indications	Contraindications
No indication for immediate intubation	Indication for emergent intubation
No obvious contraindication to NIPPV	Inability to cooperate/agitation
Hypercapnic respiratory failure[a]	Severely impaired consciousness
COPD exacerbation[a]	Inability to protect airway or clear secretions
Cardiogenic pulmonary edema[a]	High aspiration risk/swallowing impairment
Hypoxemic respiratory failure due to causes other than cardiogenic pulmonary edema	Recent facial/upper airway/gastrointestinal surgery
Asthma exacerbations	Facial or upper airway trauma
Severe pneumonia	Upper gastrointestinal bleeding
Early weaning from mechanical ventilation	Copious airway secretions
Postextubation support	Anticipation of prolonged need for respiratory support

[a] These indications have the strongest evidence to support the use of NIPPV. Other indications have shown variable success in clinical trials.

conditions that warrant consideration of NIPPV are hypoxemic respiratory failure, asthma exacerbation, severe pneumonia, weaning from mechanical ventilation, and postextubation support.[5–9] Current evidence, however, suggests that the success of NIPPV in these patients is variable, and patients must be carefully monitored for evidence of NIPPV failure. In chronic states, CPAP remains the gold standard in the treatment of obstructive sleep apnea (OSA), whereas BPAP has limited benefit in selected COPD patients.

Once the decision has been made to trial NIPPV, it should be initiated immediately so as not to miss the therapeutic window and thus avoid clinical deterioration with delayed intervention (thereby increasing likelihood of NIPPV failure and transition to invasive ventilation). Patients who deteriorate or fail to improve should be expeditiously intubated.

2. What are the contraindications for NIPPV?

An indication for emergent intubation is an absolute contraindication to NIPPV, including cardiac or pulmonary arrest. Additional relative contraindications include inability to cooperate, inability to protect airway or clear secretions, severely impaired consciousness, high aspiration risk, facial surgery/trauma/deformity, recent esophageal anastomosis, or anticipation of prolonged need for respiratory support (see **Table 1**).

CLINICAL APPLICATION

1. COPD

COPD is a common respiratory condition related to smoking or inhalational exposure, which is associated with a high morbidity and mortality. Although less common, COPD can be related to α1-antitrypsin deficiency. COPD is characterized by progressive airflow limitation (measured by spirometry), which ultimately leads to the chronic retention of CO_2. As the disease progresses, these patients are often plagued with recurrent exacerbations of COPD. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines a COPD exacerbation as "an acute increase in symptoms beyond normal day-to-day variance."[10] This condition typically includes increased dyspnea, increase in sputum volume or change in appearance, and increase in the frequency and severity of cough.

Multiple studies have documented improved clinical outcomes of NIPPV in patients with an acute COPD exacerbation complicated by hypercapnic respiratory failure.[11–14] NIPPV decreased morality, intubation rate, treatment failure, complications related to treatment, and length of stay in hospital.[14] Patients with severe COPD exacerbations (as defined by pH <7.3 on admission) respond better to NIPPV than do patients with mild COPD exacerbations.[13] The benefit of NIPPV may be due to unloading of the respiratory muscles during an acute exacerbation, improved ventilation resulting from increased tidal volumes, and improvements of intrinsic positive end-expiratory pressure (PEEP) (ie, air trapping) via low levels of extrinsic PEEP, which optimizes airway mechanics.[15,16] NIPPV is also effective in early extubation of patients intubated for severe COPD exacerbations.[8] Whether the use of NIPPV portends improvement in chronic stable COPD patients is unclear. The use of NIPPV (typically BPAP) in these patients may result in an improvement in respiratory mechanics: decreased respiratory rate, increased tidal volume, increased minute ventilation, decreased partial pressure of arterial CO_2 ($Paco_2$), and increased partial pressure of blood oxygen (Pao_2).[17]

2. Acute decompensated heart failure

Patients with chronic heart failure (typically left ventricular systolic or diastolic dysfunction) are at risk for the development of a potentially life-threatening exacerbation of heart failure from several plausible causes, including severe hypertension, overload of iatrogenic fluid (ie, intravenous fluid bolus or blood transfusion), acute myocardial infarction, tachyarrhythmia, medication or dietary noncompliance, valvulopathies (eg, acute or progressive mitral regurgitation), and end-stage renal disease. A patient with acutely decompensated heart failure may complain of dyspnea related to the accumulation of fluid within the lungs, known as cardiogenic pulmonary edema. Some of these patients may present with flash pulmonary edema that causes acute respiratory distress, occurring when left ventricular diastolic pressure rises acutely and causes a rapid shift of fluid into the pulmonary interstitium. Patients with decompensated heart failure can also present with dyspnea and increased left ventricular filling pressures without pulmonary edema. Some patients, particularly the elderly, may present with hypercapnia during an exacerbation of acute heart failure.[18]

Initial stabilization of dyspneic patients with acute decompensated heart failure requires a rapid assessment of the airway and oxygenation status, followed by an evaluation for reversible causes of the acute decompensation. Although each clinical scenario is unique, the mainstay in the stabilization of these patients is administration of a diuretic and/or vasodilator. Maintenance of appropriate oxygenation often requires supplemental oxygen delivery and, in some cases, assisted ventilation. The use of NIPPV in patients with cardiogenic pulmonary edema has proven efficacy in relieving dyspnea, improving oxygenation and/or ventilation, and decreasing the need for intubation.[4,19] Physiologically, increased intrathoracic pressure with NIPPV reduces cardiac preload and afterload[2,20] that optimizes the function of the decompensated heart, and also reduces the work of breathing, which further assists in the management of respiratory failure. These patients require close monitoring of their respiratory status with early consideration for intubation, optimization of volume status with strict monitoring of intake and output, and prompt identification and management of the underlying abnormalities.

3. Obstructive sleep apnea

OSA is a condition whereby the upper airway repeatedly and episodically is obstructed during sleep. These patients usually present with complaints of daytime sleepiness and signs of disturbed sleep (ie, disruptive snoring, witnessed apneas, restlessness). OSA is seen more frequently in men than in women (although there is little gender difference after the sixth decade), and is more prevalent in African Americans who are younger than 35 years than in age-matched Caucasian counterparts (independent of body weight). Although OSA shows an obvious propensity for obese patients, it has been previously confirmed that Asia has a rate of OSA comparable with that of the United States, despite having a lower average body mass index (BMI). These findings suggest that race may be an independent risk factor for the development of OSA.[21] Craniofacial abnormalities such as tonsilloadenoid hypertrophy also confer an increased risk of developing OSA. Unfortunately, the elimination of these risk factors is not always curative. OSA is associated with multiple medical issues, including myocardial infarction,[22] stroke,[23] pulmonary hypertension,[24] hypoventilation syndrome,[25] and diabetes.[26] OSA is associated with an increase in all-cause mortality.[27]

Treatment of OSA, as with most sleep-related disorders, should begin with education aimed at improving sleep hygiene, as well as behavior modification to include weight loss,

alcohol avoidance, and medication selection aimed at avoiding agents that inhibit the central nervous system, when feasible.[28] CPAP therapy is an effective first-line therapy for patients with an apnea-hypopnea index (frequency of abnormal breathing episodes per hour of sleep) of more than 15 events per hour or between 5 and 14 events per hour with associated daytime sleepiness, insomnia, mood disorders, impaired neurocognitive function, cardiovascular disease, or a history of stroke.[28,29] Conventionally, patients begin with CPAP that is either titrated during a polysomnogram (PSG) or via the use of an autotitrating positive airway pressure (APAP) device. Because patients with OSA experience episodic airway collapse during sleep because of the loss of motor tone of the upper airways, NIPPV works by using air pressure to splint the airway open during sleep. This action allows regular breathing with elimination of associated arousals from sleep, and thereby allows a more restful sleep and elimination of sympathetic surges that occur during these respiratory events (which account for many of the associated comorbidities). If patients fail to respond or do not tolerate CPAP (despite trying different interfaces), BPAP may be offered. (There are other treatment options, but for the purposes of this article the discussion here is limited to the use of NIPPV.) Patients using positive airway pressure therapy to treat OSA experience improved sleep quality, decreased daytime sleepiness, improved quality of life, and reduction in morbidity and mortality.[30–32]

4. Central sleep apnea

Central sleep apnea (CSA) is a condition characterized by repetitive cessation (or decrease) of ventilatory effort, resulting in disrupted sleep and resultant daytime consequences such as sleepiness and poor concentration. CSA is further classified as either primary (ie, idiopathic CSA) or secondary. Some of the more common causes of secondary CSA are CSA associated with Cheyne-Stokes breathing (seen in patients with stroke or heart failure), central nervous system–suppressing drugs or substances, medical conditions (eg, central nervous system diseases or neuromuscular diseases), or high-altitude periodic breathing. CSA is more prevalent in patients who are elderly (>65 years old), male, or with comorbid conditions such as heart failure, stroke, renal failure, acromegaly, and use of chronic long-acting opioids (eg, methadone).

Positive airway pressure therapy is an effective treatment for CSA (along with treatment of the underlying cause such as optimizing heart failure or reducing narcotics).[33] Modality choices include CPAP, BPAP, and adaptive servoventilation (ASV). Because of intermittent pauses in respiratory effort, BPAP with a backup rate (which can deliver inspiratory pressure at a certain timed interval if the patient does not spontaneously initiate) is more effective than CPAP. A more advanced device using ASV is preferred for patients with Cheyne-Stokes respiration (CSR).[34] ASV provides the patient with a set level of EPAP as well as a variable amount of IPAP based on an analysis of individual inspiratory efforts, along with a backup rate. Therefore, when the patient is in a hypopneic phase of CSR, ASV will provide a higher IPAP, but during the hyperpneic phase it will provide a reduced IPAP.[34–36] Studies have consistently indicated that the use of ASV in these patients decreases the frequency of central apneas while improving sleep quality, left ventricular ejection fraction, and exercise capacity.[37]

5. Complex sleep apnea

Complex sleep apnea syndrome (CompSAS) is also referred to as treatment-emergent CSA. CompSAS is a condition diagnosed in patients with OSA whereby central apneas and hypopneas newly occur during CPAP treatment despite the resolution of obstructive events. This condition is noted during a PSG with CPAP titration

for OSA. It remains controversial whether CompSAS and OSA are 2 distinct disorders or a single condition along the same spectrum. The condition is more common among men than women, and more commonly diagnosed in patients with severe OSA, patients with heart failure, and patients with both obstructive and central events on their initial PSG.

The mainstay of treatment for CompSAS is positive airway pressure therapy. Treatment options include continuing CPAP versus a trial of ASV or BPAP with a backup respiratory rate. At least 50% will normalize their disordered breathing events if CPAP is continued for several months.[38] Many patients are started on ASV, which is more effective in treating CompSAS, but this option may be cost-prohibitive for some patients.[39]

6. Obesity hypoventilation syndrome

Obesity hypoventilation syndrome (OHS) is diagnosed in an obese patient (BMI >30 kg/m^2) with awake hypercapnia without an alternative explanation for hypoventilation (ie, no evidence of pulmonary disease, hypothyroidism, or neuromuscular weakness). Patients with OHS are thought to have obesity-related respiratory impairment of either decreased respiratory drive or excessive work of breathing, often resulting in daytime hypercapnia ($Paco_2$ >45 mm Hg), nocturnal hypoxemia, and excessive daytime sleepiness.[40] The prevalence is approximately 30% of individuals with BMI higher than 35 kg/m^2 and nearly 50% among individuals with BMI higher than 50 kg/m^2.[41] Approximately 90% of patients with OHS have coexisting OSA, implying that OSA alone is not sufficient to lead to OHS.[42] In fact, only 20% to 30% of patients with OSA have OHS.[25]

Untreated OHS is associated with a high mortality rate (typically from acute cardiopulmonary compromise), reduced quality of life, and a multitude of comorbidities (ie, insulin resistance, right heart failure, pulmonary hypertension), similarly to individuals with OSA.[42] OHS therapy with NIPPV and weight loss is aimed at normalizing nocturnal hypoxemia and hypercapnia so as to minimize the impact of the associated morbidities. Many patients with OHS do well with CPAP but may gain additional benefit with BPAP, particularly if they have more severe hypercapnia ($Paco_2$ >55 mm Hg) or nocturnal desaturation (<80% for >10 minutes).[41,43] Once started on NIPPV therapy, these patients should have careful follow-up to ensure normalization of their nocturnal oxygenation and ventilation, with adjustments of their device (either in changing pressures or transitioning to BPAP) or addition of supplemental oxygen as required.

7. Neuromuscular disorders

Patients with progressive neuromuscular disorders (eg, amyotrophic lateral sclerosis, multiple sclerosis, myasthenia gravis) eventually experience respiratory muscle weakness, which can progress to respiratory failure and death. Presenting signs of respiratory muscle weakness may include progressive dyspnea at rest, inability to lay supine, ineffective cough, or recurrent pneumonia caused by aspiration or poor clearance of respiratory secretions. Patients with neuromuscular weakness should have regular screening to assess for respiratory muscle weakness (via pulmonary function testing or overnight oximetry).[44] The American Academy of Neurology recommends monitoring vital capacity (VC) and to consider further evaluation for need of NIPPV if VC decreases to less than 50% predicted.[45] Medicare requires the presence of hypercapnia ($Paco_2 \geq$45 mm Hg), overnight oximetry demonstrating saturation 88% or less for at least 5 minutes on usual fraction of inspired oxygen (Fio_2), maximal inspiratory

pressure less than 60 cm H_2O, or forced vital capacity 50% predicted or less, to qualify for NIPPV therapy in these patients.[46]

Owing to weakness of respiratory muscles, these patients will not be able to tolerate any significant resistance to exhalation. Therefore, when BPAP is used EPAP must be kept as low as possible. Because these patients need help with the work of breathing and to augment tidal volumes, CPAP would not be effective; rather, BPAP with a backup rate (to help with respirations during rapid eye movement sleep when they can become profoundly bradypnic) is required. Invasive ventilation is not discussed here, but is indicated for patients with contraindications to NIPPV or who fail or progress despite NIPPV. Use of NIPPV in these patients may improve mortality, decrease the need for invasive ventilation, and decrease the length of stay in the intensive care unit (ICU).[47,48]

8. Weaning from invasive ventilation or prevention of reintubation

Once a patient has recovered from respiratory failure and is able to breathe spontaneously, but still needs some ventilatory support, early extubation and transition to NIPPV may be beneficial in terms of improved mortality, shorter duration of invasive ventilatory support, shorter ICU stay, and less need for tracheostomy.[5] This benefit may be most prominent in the COPD population.[8]

Similarly, NIPPV can be effective in reducing extubation failures. Ferrer and colleagues[49] showed that patients who were considered to be at risk for extubation failures (age >65 years, cardiac failure as the cause of respiratory failure, or severity of illness based on APACHE II score >12 on the day of extubation) had significantly lower reintubation rates and improved mortality when they were extubated to NIPPV rather than simple oxygen masks.[50] This benefit may be most notable in those who have hypercapnic respiratory failure.[49,51] When a patient fails extubation (lack of improvement in pH or P_{CO_2}, change in mental status, desaturation despite high-flow oxygen, signs of respiratory muscle fatigue, hypotension, or copious secretion that could not be adequately cleared), NIPPV does not reduce the need for reintubation but may increase the risk of death in comparison with standard therapy.[52]

9. Do-not-intubate situations

Each situation needs to be individualized with regard to the use of NIPPV in do-not-intubate (DNI) situations. Patients or their surrogate decision maker need to understand that NIPPV is a life support system but one that can be removed easily, should not require any sedation, and can be used simply to alleviate dyspnea. The use of NIPPV in DNI patients presenting with acute respiratory failure can result in reversal of their acute exacerbation and lead to survival in 43% to 63%.[53,54]

PRACTICAL APPLICATION

1. How to start

Once a patient has been determined appropriate for a trial of NIPPV, the respiratory therapist should be notified immediately to avoid delay in treatment. The patient may need to be transferred to a higher level of care (ie, ICU, progressive care unit, or respiratory care unit) depending on the clinical stability of the patient. Some facilities may require initiation of NIPPV, such as BPAP, to be done only in specialized care units. The plan of care should be discussed with the patient or surrogate decision maker, including the possibility of advancing to intubation if indicated and reaffirming the goals of care.

2. Interface options

NIPPV interfaces include a full-face mask, full-face shield, nasal mask, and nasal pillows. According to a randomized study evaluating the 3 types of interfaces in patients with chronic hypercapnic respiratory failure,[55] patients treated with a full-face mask showed greater physiologic improvement. However, the nasal mask was better tolerated and just as effective if the patient is able to keep the mouth closed. A full-face mask is preferred for the initiation of NIPPV in most cases. If the patient is unable to tolerate a full-face mask, other interfaces should be trialed.

3. Initial pressure settings

There is no established protocol for determining the initial CPAP/BPAP settings. If the patient is already on home NIPPV therapy, the same setting can be resumed and adjusted as necessary. If CPAP is to be tried for presumed OSA, an APAP can be used with the range of pressure of 5 to 15 cm H_2O. Depending on the technology available on the APAP, some devices may not be able to distinguish between OSA and CSA. In such cases the device may erroneously increase CPAP pressure for CSA, which worsens the frequency of CSA. If this occurs, the upper range of APAP pressures will need to be reduced. In an effort to maintain patient comfort as well as avoiding overshooting (in the case of hypercapnia), BPAP should begin at a low-pressure setting (ie, 8–10 cm H_2O IPAP and 4–5 cm H_2O EPAP). The minimum difference between IPAP and EPAP must be 4 cm H_2O to provide some pressure support in the patient's respiratory effort. IPAP can be increased gradually (to 10–20 cm H_2O) as needed to improve dyspnea, work of breathing, synchronization between the patient and the NIPPV, and hypercapnia. If hypoxia persists despite maximizing supplemental oxygen, EPAP can be increased to improve oxygenation. However, this should be done cautiously, especially in COPD patients, as excessive EPAP will result in worsening hyperinflation as well as discomfort for many patients. It is essential to assess early for patient synchrony with BPAP and evidence of reduced work of breathing. For some patients, use of NIPPV may only worsen their work of breathing. If this occurs, early invasive ventilatory support needs to be considered.

In COPD patients, careful adjustment of EPAP is required to reduce the impact of auto-PEEP or intrinsic PEEP. Because of their prolonged expiratory time, these patients may not have completely exhaled before they take their next breath. With each subsequent breath they will trap a certain amount of air, resulting in a progressive increase in intrathoracic pressure. This process results in increasing the work of breathing and may cause difficulty in triggering the BPAP. To minimize this effect, use of a bronchodilator and very judicious use of EPAP may be required. However, as mentioned earlier, excessive EPAP should be avoided, as this will also result in hyperinflation.

4. What kind of follow-up is required now that the patient is on NIPPV? Is it necessary to repeat an arterial blood gas analysis after initiation of NIPPV for acute hypoxic/hypercapnic respiratory failure?

Patients must be very closely monitored in the first 30 minutes after initiation of NIPPV. Both subjective and clinically apparent improvements should be evident within the first hour of NIPPV initiation.

In the case of hypoxic respiratory failure, it is unnecessary to repeat an arterial blood gas analysis (APG). Instead, a noninvasive pulse oximetry can be used to guide

titration of the pressures and oxygen to maintain a target oxygen saturation of greater than 90%.

Hypercapnic respiratory failure necessitates frequent monitoring of arterial pH and CO_2 ($Paco_2$). Within 1 hour of initiating BPAP, a repeat ABG should be obtained to ensure that the arterial pH and $Paco_2$ are moving in the desired direction. Values obtained from the repeat ABG are used to guide decisions regarding pressure settings. Some centers may use end-tidal CO_2 monitors to make necessary changes to the BPAP settings. A patient with acute hypoxic/hypercapnic respiratory failure who is poorly responsive to NIPPV should be promptly intubated. Other indications of impending failure are hemodynamic instability, persistent hypoxia, increased agitation or encephalopathy, inability to clear secretions, or inability to tolerate any of the interfaces. Failure to recognize clinical deterioration and the need for emergent intubation may lead to poor outcome for the patient.

In some patients with overlapping OSA and acute respiratory failure, the initial setting may need to be further adjusted once they fall asleep, as they may require higher EPAP to overcome upper airway obstruction that is due to their OSA.

5. What is to be done if patients are intolerant of NIPPV?

Many patients with acute respiratory failure may have difficulty tolerating NIPPV because of claustrophobia, or simply anxiety attributable to severe dyspnea. It takes much patience and encouragement to help a patient become accustomed to NIPPV. Typically, starting at lower pressures and initially holding the mask by hand rather than strapping it on to the head may help. It is important to encourage patients to take slow deep breaths and determine what may be bothering them. Some may express that they are not getting enough air, in which case the pressure will need to be slowly increased. Some may indicate that they are too claustrophobic, in which case a trial of a nasal interface may be warranted. Occasionally a sedative may need to be considered, but this needs to be done with extreme caution because sedatives may reduce respiratory drive and result in worsening hypercapnic respiratory failure. If a sedative is to be used, the lowest dose of a short-acting agent should be considered, with careful monitoring of the patient's mental and respiratory status.

PERFORMANCE IMPROVEMENT

1. Increased use of NIPPV

Recent data suggest that NIPPV is underutilized, as only 12% of patients with COPD and acute respiratory failure are initiated on NIPPV.[56] This situation may derive from a lack of familiarity with NIPPV or limitations in staff and/or equipment. With improved knowledge and experience with NIPPV, it is hoped that its use will increase. As the benefits of NIPPV are not clearly seen in all forms of respiratory failure, careful patient selection and close follow-up early in the course of NIPPV use is critical.

2. Increased collaboration with a specialist

Depending on an individual's level of comfort with NIPPV and its adjustments, a close collaboration with a specialist may be in the best interests of patients. This approach may allow for increased use of NIPPV and avoidance of invasive ventilation, but also may expeditiously escalate care if patients are showing signs of NIPPV failure. Collaboration may also improve outcomes and allow for close outpatient follow-up in some patients who may require long-term NIPPV use.

CLINICAL GUIDELINES

At present, there are very few guidelines available on the use of NIPPV. The British Thoracic Society (BTS) developed a clinical guideline in 2002 that is still relevant today.[57] The BTS later provided an update specifically for COPD patients with acute hypoxic and hypercapnic respiratory failure.[58] Most recently, the Canadian Medical Association provided an update on the use of NIPPV in 2011.[59]

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