CME CREDIT *EDUCATIONAL OBJECTIVE:* Readers will identify acute care patients who may be candidates for noninvasive positive pressure ventilation

LOUTFI S. ABOUSSOUAN, MD
Respiratory Institute, Cleveland Clinic

BASMA RICAURTE, MD
Pulmonary and Critical Care, Fairview Hospital, Cleveland, OH

Noninvasive positive pressure ventilation: Increasing use in acute care

ABSTRACT

In the past 2 decades, noninvasive positive pressure ventilation (NIPPV) has been increasingly used in acute respiratory failure to avoid the risks associated with intubation. It is now considered standard first-line therapy in several situations. In this review, we summarize how NIPPV has evolved, the current level of evidence that supports its use in various clinical situations, its potential contraindications, and its limitations in acute respiratory failure.

KEY POINTS

The advantages of NIPPV over invasive ventilation are that it preserves normal physiologic functions such as coughing, swallowing, feeding, and speech and avoids the risks of tracheal and laryngeal injury and respiratory tract infections.

The best level of evidence for the efficacy of NIPPV is in acute hypercarbic or hypoxemic respiratory failure during exacerbations of chronic obstructive pulmonary disease, in cardiogenic pulmonary edema, and in immunocompromised patients.

NIPPV should not be applied indiscriminately for less-established indications (such as in unconscious patients, respiratory failure after extubation, acute lung injury, or acute respiratory distress syndrome), in severe hypoxemia or acidemia, or after failure to improve dyspnea or gas exchange. The use of NIPPV in these situations may delay a necessary intubation and increase the risks of such a delay, including death.

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Noninvasive positive pressure ventilation (NIPPV)—delivered via a tight-fitting mask rather than via an endotracheal tube or tracheostomy—is one of the most important advances in the management of acute respiratory failure to emerge in the past 2 decades. It is now recommended as the first choice for ventilatory support in selected patients, such as those with exacerbations of chronic obstructive pulmonary disease (COPD) or with cardiogenic pulmonary edema.[1–3] In fact, some authors suggest that using NIPPV in more than 20% of COPD patients is a characteristic of respiratory care departments that are "avid for change"[4]—change being a good thing.

However, NIPPV has not been universally accepted, with wide variations in its utilization. In a 2006 survey, it was being used in only 33% of patients with COPD or congestive heart failure, for which it might be indicated.[5] Some potential reasons for the low rate are that physicians do not know about it, respiratory therapists are not sufficiently trained in it, and hospitals lack the equipment to do it.[5]

Our goal in this review is to familiarize the reader with how NIPPV has evolved and with its indications and contraindications in specific acute care conditions.

FROM A VACUUM CLEANER TO THE INTENSIVE CARE UNIT

NIPPV appears to have been first tried in 1870 by Chaussier, who used a bag and face mask to resuscitate neonates.[6]

TABLE 1
Types of noninvasive ventilation with proposed settings

MODE	DESCRIPTION	SETTINGS
CPAP	Provides a constant pressure, but no ventilatory support. More effective in hypoxemic than in hypercapnic states. Improves alveolar edema and increases functional residual capacity.	Slowly increase up to 5–12 cm H_2O to improve hypoxemia.
Pressure-limited	Cycles between higher inspiratory and lower expiratory pressures. Breath trigger includes spontaneous patient effort (with pressure support) or a time instruction such as backup rate (for pressure control).	IPAP 8–20 cm H_2O for respiratory rate < 25 breaths per minute, EPAP/PEEP of 0–10 cm H_2O to improve oxygenation. Adjust settings to goals. For instance, a pressure support of 15 cm H_2O with PEEP of 5 cm H_2O can decrease dyspnea, respiratory rate, and $Paco_2$ more than a pressure support of 10 cm H_2O with PEEP of 10 cm H_2O. However, the latter setting can be associated with better oxygenation.
Volume-limited	Provides a constant volume. Triggers to the breaths include patient effort (with assisted breaths) or a time instruction such as backup rate (for controlled breaths).	250–500 mL (4–8 mL/kg) volumes to obtain a respiratory rate < 25 breaths per minute. Adjust settings to goals: increase volumes for ventilatory support and hypercapnia, adjust PEEP upwards to improve oxygenation.

CPAP = continuous positive airway pressure; IPAP = inspiratory positive airway pressure; EPAP = expiratory positive airway pressure; PEEP = positive end-expiratory pressure; $Paco_2$ = partial pressure of arterial carbon dioxide

BASED ON INFORMATION IN ANTONELLI M, CONTI G. NONINVASIVE POSITIVE PRESSURE VENTILATION AS TREATMENT FOR ACUTE RESPIRATORY FAILURE IN CRITICALLY ILL PATIENTS. CRIT CARE 2000; 4:15–22; AND HILLBERG RE, JOHNSON DC. NONINVASIVE VENTILATION. N ENGL J MED 1997; 337:1746–1752.

Reasons for using NIPPV in acute respiratory failure: to improve oxygenation and ventilation without use of invasive ventilation

In 1936, Poulton and Oxon[7] described their "pulmonary plus pressure machine," which used a vacuum cleaner blower and a mask to increase the alveolar pressure and thus counteract the increased intrapulmonary pressure in patients with heart failure, pulmonary edema, Cheyne-Stokes breathing, and asthma.

In the 1940s, intermittent positive pressure breathing devices were developed for use in high-altitude aviation. Motley, Werko, and Cournand[8,9] subsequently used these devices to treat acute respiratory failure in pneumonia, pulmonary edema, near-drowning, Guillain-Barré syndrome, and acute severe asthma.

Although NIPPV was shown to be effective for acute conditions, invasive ventilation became preferred, particularly as blood gas analysis and ventilator technologies simultaneously matured, spurred at least in part by the polio epidemics of the 1950s.[10]

NIPPV reemerged in the 1980s for use in chronic conditions. First, continuous positive airway pressure (CPAP) came into use for obstructive sleep apnea,[11] followed by noninvasive positive-pressure volume ventilation in neuromuscular diseases.[12] Bilevel positive pressure devices (ie, with separate inspiratory and expiratory pressures) soon followed, again initially for obstructive sleep apnea[13] and then for diverse neuromuscular diseases.[14]

NIPPV is now a mainstream therapy for diverse conditions in acute and chronic care.[3] One reason we now use it in acute conditions is to avoid the complications associated with intubation.

Some clinicians initially resisted using NIPPV, concerned that it demanded too much of the nurses' time[15] and was costly.[16] However, in a 1997 study in patients with COPD and acute respiratory failure, Nava et al[17] found that NIPPV was no more expensive and no more demanding of staff resources than invasive mechanical ventilation in the first 48 hours of ventilation. Further, after the first few days of ventilation, NIPPV put fewer time demands on physicians and nurses than did invasive mechanical ventilation.

THREE MODES: CPAP, PRESSURE-LIMITED, VOLUME-LIMITED

The term "noninvasive ventilation" generally encompasses various forms of positive pressure ventilation. However, negative pressure ventilation, in the form of diaphragm pacing, may regain a foothold in the devices used for respiratory support.[18] We therefore favor the term "NIPPV" in this review.

The different modes of NIPPV—ie, CPAP, pressure-limited, and volume-limited—are compared in TABLE 1. Of these, the pressure-limited mode is most commonly used.[2,19–21] Though there are several NIPPV-only devices, machines for invasive ventilation can also provide NIPPV.

NIPPV IN ACUTE RESPIRATORY FAILURE

The main reasons to use NIPPV instead of invasive ventilation in acute care are to avoid the complications of invasive ventilation, to improve outcomes (eg, reduce mortality rates, decrease hospital length of stay), and to decrease the cost of care.

The decision whether to initiate noninvasive support and where to provide it (ie, in a regular hospital ward, intensive care unit, or respiratory care unit) is best made by following the indications for and contraindications to NIPPV (TABLE 2), considering the specific disease, the strength of the recommendation (TABLE 3), and the expertise and skill of the staff.[1,2,19] In general, NIPPV is more likely to fail in patients with more severe disease and lower arterial pH.[3] It should not be applied indiscriminately, as it may simply delay a necessary intubation and raise the concomitant risks of such a delay, including death.[22]

NIPPV is the standard of care for acute exacerbations of COPD

NIPPV is currently considered the standard of care for patients who have acute exacerbations of COPD.[23–26]

In a meta-analysis of eight randomized controlled trials,[24] the specific advantages of NIPPV compared with usual care in acute exacerbations of COPD included:

- A lower risk of treatment failure, defined as death, need for intubation, or inabil-

TABLE 2

NIPPV: Indications and contraindications in acute care

Indications
Subjective dyspnea with respiratory rate > 25 breaths per minute
Use of accessory muscles
$Paco_2$ > 45 mm Hg with pH ≤ 7.35
Pao_2/Fio_2 < 200 mm Hg
Conscious and cooperative (with possible exception of COPD: see discussion in text)
Proper mask fit
Contraindications (any of the following)
Severe hypoxemia (Pao_2/Fio_2 < 75)
Severe acidemia
Multiorgan failure or slowly reversible disease (in short term)
Upper airway obstruction
Anatomic abnormalities that interfere with gas delivery (eg, facial burn, trauma)
Respiratory arrest, apnea
Cardiac arrest and hemodynamic or cardiac instability
Uncooperative patient
Encephalopathy with inability to protect airways and a high risk of aspiration
Increased risk of aspiration: copious secretions, vomiting, or severe gastrointestinal bleeding
Recent airway or gastrointestinal surgery
Inability to fit mask
Criteria for discontinuation of NIPPV and intubation
Mask intolerance and poor adherence
Failure to improve dyspnea, gas exchange: eg, Pao_2/Fio_2 ≤ 146, or ≤ 175 for ARDS, after 1 hour of NIPPV
Failure to improve mental status within 30 minutes
Hemodynamic instability, cardiac ischemia, arrhythmias
Difficulties with managing secretions

ARDS = acute respiratory distress syndrome; Fio_2 = fraction of inspired oxygen; NIPPV = noninvasive positive pressure ventilation; $Paco_2$ = partial pressure of arterial carbon dioxide; Pao_2 = partial pressure of arterial oxygen

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TABLE 3

Specific conditions of acute respiratory failure in which NIPPV has been used, categorized by level of evidence

LEVEL OF EVIDENCE	STRENGTH OF RECOMMENDATION [a]	LOCATION
A: Multiple randomized controlled trials and meta-analyses		
COPD exacerbation [b]	Recommended	ICU, RCU, ward
To facilitate weaning in COPD	Guideline	ICU, RCU
Cardiogenic pulmonary edema [c]	Recommended	ICU, RCU
Immunocompromised with hypoxemic failure	Recommended	ICU, RCU
B: More than one randomized, controlled trial, case-control series, or cohort study		
Postoperative respiratory failure	Guideline	ICU
To improve oxygenation before intubation	Option	ICU
To facilitate bronchoscopy	Guideline	ICU, RCU
To prevent respiratory failure after extubation (chronic lung disease, $Paco_2$ > 45 mm Hg)	Option	ICU
C: Case series or conflicting data		
Asthma, status asthmaticus	Option	ICU, RCU
Palliative	Guideline	Ward, RCU
Pneumonia	Option	ICU, RCU
Acute lung injury, acute respiratory distress syndrome	Option	ICU
Extubation failure	Guideline	ICU

COPD = chronic obstructive pulmonary disease; ICU = intensive care unit; NIPPV= noninvasive positive pressure ventilation; $Paco_2$ = partial pressure of arterial carbon dioxide; RCU = respiratory care unit

[a] Recommended: first choice for ventilatory support in selected patients. Guideline: can be used in appropriate patients, but careful monitoring is advised. Option: suitable for a very carefully selected and monitored minority of patients.

[b] Best evidence for severe COPD with pH < 7.35.[26]

[c] In most recent review, no evidence of survival benefit.[28]

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ity to tolerate the treatment (relative risk [RR] 0.51, number needed to treat [NNT] to prevent one treatment failure = 5)
- A lower risk of intubation (RR 0.43, NNT = 5)
- A lower mortality rate (RR 0.41, NNT = 8)
- A lower risk of complications (RR 0.32, NNT = 3)
- A shorter hospital length of stay (by about 3 days).

Mechanisms by which NIPPV may impart these benefits include reducing the work of breathing, unloading the respiratory muscles, lessening diaphragmatic pressure swings, reducing the respiratory rate, eliminating diaphragmatic work, and counteracting the threshold loading effects of auto-positive end-expiratory pressure (auto-PEEP).[24–26]

Also, if a patient with COPD is intubated, NIPPV seems to help after the tube is removed, preventing postextubation respiratory

failure and facilitating weaning from invasive ventilation.[27] These topics are discussed below.

A Cochrane systematic review[24] concluded that NIPPV should be tried early in the course of respiratory failure, before severe acidosis develops. The patients in the studies in this review all had partial pressure of arterial carbon dioxide ($Paco_2$) levels greater than 45 mm Hg.

In patients with severe respiratory acidosis (pH < 7.25), NIPPV failure rates are greater than 50%. However, trying NIPPV may still be justified, even in the presence of hypercapnic encephalopathy, as long as no other indications for invasive support and facilities for prompt endotracheal intubation are available.[1]

However, in another systematic review,[26] in patients with mild COPD exacerbations (pH > 7.35), NIPPV was no more effective than standard medical therapy in preventing acute respiratory failure, preventing death, or reducing length of hospitalization. Moreover, nearly 50% of the patients could not tolerate NIPPV.

Rapid improvement in cardiogenic pulmonary edema, but possibly no lower mortality rate

The Three Interventions in Cardiogenic Pulmonary Oedema (3CPO) trial,[28] with 1,156 patients, was the largest randomized trial to compare NIPPV and standard oxygen therapy for acute pulmonary edema. It found that NIPPV (either CPAP or noninvasive intermittent positive pressure ventilation) was significantly better than standard oxygen therapy (through a variable-delivery oxygen mask with a reservoir) in the first hour of treatment in terms of the dyspnea score, heart rate, acidosis, and hypercapnia. However, there were no significant differences between groups in the 7- or 30-day mortality rates, the rates of intubation, rates of admission to the critical care unit, or in the mean length of hospital stay.

In contrast, several smaller randomized trials and meta-analyses showed lower intubation and mortality rates with NIPPV.[29,30] Factors that may account for those differences include a much lower intubation rate in the 3CPO trial (2.9% overall, compared with 20% with conventional therapy in other trials), a higher mortality rate in the 3CPO trial, and methodologic differences (eg, patients for whom standard therapy failed in the 3CPO trial received rescue NIPPV).

If NIPPV is beneficial in cardiogenic pulmonary edema, the mechanisms are probably its favorable hemodynamic effects and its positive end-expiratory pressure (PEEP) effect on flooded alveoli. Specifically, positive intrathoracic pressure can be expected to reduce both preload and afterload, with improvement in the cardiac index and reduced work of breathing.[31,32]

Notwithstanding the possible lack of impact of NIPPV on death or intubation rates in this setting, the intervention rapidly improves dyspnea and respiratory and metabolic abnormalities and should be considered for treatment of cardiogenic pulmonary edema associated with severe respiratory distress. A subgroup in which the NIPPV may reduce intubation rates is those with hypercapnia.[33] A concern that NIPPV may increase the rate of myocardial infarction[34] was not confirmed in the 3CPO trial.[28] Interestingly, there were no differences in outcomes between CPAP and noninvasive intermittent positive pressure ventilation in this setting.[28,34,35]

NIPPV is more likely to fail in patients with more hypoxic respiratory failure and lower arterial pH

Immunocompromised patients with acute respiratory failure

A particular challenge of NIPPV in immunocompromised patients, particularly compared with its use in COPD exacerbation or cardiogenic pulmonary edema, is that the underlying pathophysiology of respiratory dysfunction in immunocompromised patients may not be readily reversible. Therefore, its application in this group may need to follow clearly defined indications.

In one trial,[20] inclusion criteria were:

- Immune suppression (due to neutropenia after chemotherapy or bone marrow transplantation, immunosuppressive drugs for organ transplantation, corticosteroids, cytotoxic therapy for nonmalignant conditions, or the acquired immunodeficiency syndrome)
- Persistent pulmonary infiltrates
- Fever (temperature > 38.3°C; 100.9°F)

- A respiratory rate greater than 30 breaths per minute
- Severe dyspnea at rest
- Early hypoxemic acute respiratory failure, defined as a ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen (Pao_2/Fio_2 ratio) less than 200 while on oxygen.

Compared with patients who received conventional treatment, fewer of those randomized to additional intermittent noninvasive ventilation had to be intubated (46% vs 77%, $P = .03$), suffered serious complications (50% vs 81%, $P = .02$), or died in the intensive care unit (38% vs 69%, $P = .03$) or in the hospital (50% vs 81%, $P = .02$).

Similarly, in a randomized trial in 40 patients with acute respiratory failure after solid organ transplantation, more patients in the NIPPV group than in the control group had an improvement in the Pao_2/Fio_2 ratio within the first hour (70% vs 25%, $P = .004$) or a sustained improvement in the Pao_2/Fio_2 ratio (60% vs 25%, $P = .03$); fewer of them needed endotracheal intubation (20% vs 70%, $P = .002$); fewer of them died of complications (20% vs 50%, $P = .05$); they had a shorter length of stay in the intensive care unit (mean 5.5 vs 9 days, $P = .03$); and fewer of them died in the intensive care unit (20% vs 50%, $P = .05$). There was, however, no difference in the overall hospital mortality rate.[36]

NIPPV should be tried early in the course of respiratory failure in COPD, before severe acidosis develops

MAY NOT HELP AFTER EXTUBATION, EXCEPT IN SPECIFIC CASES

NIPPV has been used to treat respiratory failure after extubation,[22,37] to prevent acute respiratory failure after failure of weaning,[38–41] and to support breathing in patients who failed a trial of spontaneous breathing.[42–45]

Unfortunately, the evidence for using NIPPV in respiratory failure after extubation, including unplanned extubation, appears to be unfavorable, except possibly in patients with chronic pulmonary disease (particularly COPD and possibly obesity) and hypercapnia. An international consensus report stated that NIPPV should be considered in patients with hypercapnic respiratory insufficiency, especially those with COPD, to shorten the duration of intubation, but that it should not be routinely used in extubation respiratory failure.[46]

Treatment of respiratory failure after extubation

Two recent randomized controlled trials compared NIPPV and standard care in patients who met the criteria for readiness for extubation but who developed respiratory failure after mechanical ventilation was discontinued.[22,37] Those two studies showed a longer time to reintubation for patients randomized to NIPPV but no differences in the rate of reintubation between the two groups and no difference in the lengths of stay in the intensive care unit.

Of greater concern, one study showed a higher rate of death in the intensive care unit in the NIPPV group than in the standard therapy group (25% vs 14%, respectively).[22] This finding suggests that NIPPV delayed necessary reintubation in patients developing respiratory failure after extubation, with a consequent risk of fatal complications.

Prevention of respiratory failure after extubation

Other studies used NIPPV to prevent respiratory failure after extubation rather than wait to apply it after respiratory failure developed.[38–41]

Nava et al,[40] in a trial in patients successfully weaned but considered to be at risk of reintubation, found that fewer of those randomized to NIPPV had to be reintubated than those who received standard care (8% vs 24%), and 10% fewer of them died in the intensive care unit. Risk factors for reintubation (and therefore eligibility criteria for this trial) included a $Paco_2$ higher than 45 mm Hg, more than one consecutive failure of weaning, chronic heart failure, other comorbidity, weak cough, or stridor.

Extubated patients are a heterogeneous group, so if some subgroups benefit from a transition to NIPPV after extubation, it will be important to identify them. For instance, a subgroup analysis of a study by Ferrer et al[38] indicated the survival benefit of NIPPV after extubation was limited to patients with chronic respiratory disorders and hypercapnia during a trial of spontaneous breathing.

In a subsequent successful test of this hy-

pothesis, a randomized trial showed that the early use of noninvasive ventilation in patients with hypercapnia after a trial of spontaneous breathing and with chronic respiratory disorders (COPD, chronic bronchitis, bronchiectasis, obesity-hypoventilation, sequelae of tuberculosis, chest wall deformity, or chronic persistent asthma) reduced the risk of respiratory failure after extubation and the risk of death within the first 90 days.[39]

Others in which this approach may be helpful are obese patients who have high $Paco_2$ levels. Compared with historical controls, 62 patients with a body mass index greater than 35 kg/m^2 who received NIPPV in the 48 hours after extubation had a lower rate of respiratory failure, shorter lengths of stay in the intensive care unit and hospital, and, in the subgroup with hypercapnia, a lower hospital mortality rate.[41]

NIPPV to facilitate weaning

In several studies, mechanically ventilated patients who had failed a trial of spontaneous breathing were randomized to undergo either accelerated weaning, extubation, and NIPPV or conventional weaning with pressure support via mechanical ventilation.[42–46] Most patients developed hypercapnia during the spontaneous breathing trials, and most of the patients had COPD.

A meta-analysis[47] of the randomized trials of this approach concluded that, compared with continued invasive ventilation, NIPPV decreased the risk of death (relative risk 0.41) and of ventilator-associated pneumonia (relative risk 0.28) and reduced the total duration of mechanical ventilation by a weighted mean difference of 7.33 days. The benefits appeared to be most significant in patients with COPD.

NIPPV IN ASTHMA AND STATUS ASTHMATICUS

Noninvasive ventilation is an attractive alternative to intubation for patients with status asthmaticus, given the challenges and conflicting demands of maintaining ventilation despite severe airway obstruction.

In a 1996 prospective study of 17 episodes of asthma associated with acute respiratory failure, Meduri et al[48] showed that NIPPV could progressively improve the pH and the $Paco_2$ over 12 to 24 hours and reduce the respiratory rate.

In a subsequent controlled trial, Soroksky et al[49] randomized 30 patients presenting to an emergency room with a severe asthma attack to NIPPV with conventional therapy vs conventional therapy only. The study group had a significantly greater increase in the forced expiratory volume in 1 second compared with the control group (54% vs 29%, respectively) and a lower hospitalization rate (18% vs 63%).

Another randomized trial of NIPPV, in patients with status asthmaticus presenting to an emergency room, was prematurely terminated due to a physician treatment bias that favored NIPPV.[50] The preliminary results of that study showed a 7.3% higher intubation rate in the control group than in the NIPPV group, along with trends toward a lower intubation rate, a shorter length of hospital stay, and lower hospital charges in the NIPPV group.

Despite these initial favorable results, a Cochrane review concluded that the use of NIPPV in patients with status asthmaticus is controversial.[51] NIPPV can be tried in selected patients such as those with mild to moderate respiratory distress (respiratory rate greater than 25 breaths per minute, use of accessory muscles to breathe, difficulty speaking), an arterial pH of 7.25 to 7.35, and a $Paco_2$ of 45 to 55 mm Hg.[52] Patients with impending respiratory failure or the inability to protect the airway should probably not be considered for NIPPV.[52]

In immunocompromised patients, respiratory dysfunction may not be readily reversible

IN ACUTE LUNG INJURY AND ACUTE RESPIRATORY DISTRESS SYNDROME

The most challenging application of NIPPV may be in patients with acute lung injury and the acute respiratory distress syndrome.

Initial trials of NIPPV in this setting have been disappointing, and a meta-analysis of the topic concluded that NIPPV was unlikely to have any significant benefit.[53] An earlier study that used CPAP in patients with acute respiratory failure predominantly due to acute lung injury showed early physiologic improvements but no reduction in the need for intubation, no improvement in outcomes, and a higher

rate of adverse events, including cardiac arrest, in those randomized to CPAP.[54]

A subsequent observational cohort specifically identified shock, metabolic acidosis, and severe hypoxemia as predictors of NIPPV failure.[55]

A more recent prospective study demonstrated that NIPPV improved gas exchange and obviated intubation in 54% of patients, with a consequent reduction in ventilator-associated pneumonia and a lower rate of death in the intensive care unit.[56] A Simplified Acute Physiology Score (SAPS) II greater than 34 and a Pao_2/Fio_2 ratio less than 175 after 1 hour of NIPPV were identified as predicting that NIPPV would fail.[56]

MISCELLANEOUS APPLICATIONS

The more widespread use of NIPPV has encouraged its use in other acute situations, including during procedures such as percutaneous endoscopic gastrostomy (PEG)[57,58] or bronchoscopy,[59,60] for palliative use in patients listed as "do-not-intubate,"[61–63] and for oxygenation before intubation.[64]

NIPPV should not be routinely used in respiratory failure after extubation

NIPPV during PEG tube insertion

NIPPV during PEG tube placement is particularly useful for patients with neuromuscular diseases who are at a combined risk of aspiration, poor oral intake, and respiratory failure during procedures. The experience with patients with amyotrophic lateral sclerosis[58] and Duchenne muscular dystrophy[57] indicates that even patients at high risk of respiratory failure during procedures can be successfully managed with NIPPV. The most recent practice parameters for patients with amyotrophic lateral sclerosis propose that patients with dysphagia may be exposed to less risk if the PEG procedure is performed when the forced vital capacity is greater than 50% of predicted.[65]

In randomized trials of CPAP[59] or pressure-support NIPPV[60] in high-risk hypoxemic patients who needed diagnostic bronchoscopy, patients in the intervention groups fared better than those who received oxygen alone, with better oxygenation during and after the procedure and a lower risk of postprocedure respiratory failure. Improved hemodynamics with a lower mean heart rate and a stable mean arterial pressure were also reported in one of those studies.[60]

Palliative use in 'do-not-intubate' patients

In patients who decline intubation, NIPPV appears to be most effective in reversing acute respiratory failure and improving mortality rates in those with COPD or with cardiogenic pulmonary edema.[61,62] Controversy surrounding the use of NIPPV in "do-not-intubate" patients, particularly as a potentially uncomfortable life support technique, has been addressed by a task force of the Society of Critical Care Medicine, which recommends that it be applied only after careful discussion of goals of care and parameters of treatment with patients and their families.[63]

Oxygenation before intubation

In a prospective randomized study of oxygenation before rapid-sequence intubation via either a nonrebreather bag-valve mask or NIPPV, the NIPPV group had a higher oxygen saturation rate before, during, and after the intubation procedure.[64]

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***ADDRESS**: Loutfi Aboussouan, MD, Respiratory Institute, Cleveland Clinic Beachwood, 26900 Cedar Road, Suite 325-S, Beachwood, OH 44122; e-mail: aboussl@ccf.org.*