



PULMONARY AND CRITICAL CARE BULLETIN

Vol. X, No. 1, January 15, 2004
Website : indiachest.org
(pp. 1-8)

IN THIS ISSUE :

1. Non-Invasive Ventilation - Overview and Its Application In Acute Hypoxemic Respiratory Failure

Balamugesh T.
M.D., D.M.

3. Rhinitis and Asthma - "One Airway, One Disease"

R.S. Bedi and U.S. Bedi

Published under the auspices of

Pulmonary C.M.E. Programme of
The CHEST
(Chest Health Care, Education & Research Trust)

Editorial Board

Dr. S.K. Jindal, Chief Editor

Dr. D. Behera

Dr. D. Gupta

Dr. A.N. Aggarwal

Department of Pulmonary Medicine,
Postgraduate Institute of Medical
Education & Research, Chandigarh

Subscription

Annual : Rs. 100

Life Subscription : Rs. 700

Subscription should be paid through a draft drawn in favour of "The CHEST, PGI, Chandigarh" Add bank charges (Rs. 30) for outstation cheques.

Address all correspondence to the Chief Editor

NON-INVASIVE VENTILATION - OVERVIEW AND ITS APPLICATION IN ACUTE HYPOXEMIC RESPIRATORY FAILURE

Traditionally, an endotracheal tube is inserted into the trachea to deliver positive pressure to the lungs of patients with respiratory failure. Potential complications and discomfort associated with placing this artificial airway have confined the use of mechanical ventilation to the most severe forms of acute respiratory failure. Injury to the upper airways can occur at the point of contact between the mucosa and the endotracheal tube or cuff and can result in ulceration, edema, and hemorrhage, with potential stenosis. Most importantly, however, the endotracheal tube directly places patients at significant risk for developing life - threatening nosocomial infections-mainly ventilator - associated pneumonias and sinusitis. Non - invasive methods, which avoid these complications, include external negative pressure, chest wall oscillation, and positive-pressure ventilation administered through a mask.

The origin of positive - pressure ventilation dates back to the "creation of man" (Genesis 2:7) : *And the Lord God formed man of the dust of the ground, and breathed into his nostrils and breath of Life, and man become a living soul.*

The first report of positive pressure applied by facemask was by Bunnell in 1912, to maintain lung expansion during thoracic surgery¹. Its first use outside operating room was described by Poul-ton and Oxon I in 1936: they used a vacuum cleaner to generate gas flow and a spring-loaded valve to oppose expiration². In 1935-38, Barach reported a series of studies involving a powered mechanical ventilator to deliver continuous positive airway pressure (CPAP) through a face mask^{3,4}. In the last decade there has been renewed interest in non invasive positive-pressure ventilation and it has become a standard treatment modality for a number of respiratory problems.

Patient Selection

1. Alert and cooperative patient is critical for initiating NPPV (Non invasive positive pressure ventilation) or CPAP. Patients with COPD and CO₂ narcosis, however, are an exception.
2. Hemodynamic stability
3. No need for endotracheal intubation to:
a) Protect the airways (mental obtundation, impaired swallowing, or active upper gastrointestinal bleeding)
or, b) Remove excessive secretions.
4. No acute facial trauma
5. Properly fitted mask

Interface : Mask CPAP studies almost exclusively have used a facial mask whereas a nasal mask was used more often in NPPV studies. Nasal masks add less dead space (105 ml)⁵, cause less claustrophobia, minimize potential complications if vomiting occurs, and allow for expectoration and oral intake without mask removal. With a nasal mask, patients can vocalize more clearly and can discontinue ventilation voluntarily by opening the mouth. But mouth breathing with nasal mask decreases effectiveness and dyspnoeic patients are usually mouth breathers. Although dead-space volume of a facial mask is larger (250 ml)⁵ one group reported greater success with a face mask NPPV versus the institutional historical control with a nasal mask⁶. Also improvement in ABG appears slower in nasal mask as compared to facemask. So it can be concluded that face mask is best suited for patients with severe ARF (Acute Respiratory Failure) and dyspnea. In mild forms of ARF, a nasal mask could be tried first, switching to a facial mask if necessary.

A mask with a transparent dome is preferred because it allows visual monitoring of the oral airway for secretions. The mask should be light-weight to aid in its application and have a soft, pliable, adjustable seal to reduce trauma and leakage. To prevent drying of the nasal passages and oropharynx, a humidifier

should be connected but the heater should be turned off. because the upper airways that naturally warm inspired gas are not bypassed with NIV.

Comfort : Because patient tolerance is essential to the success of NPPV, a tight, uncomfortable fit should be avoided. A small degree of air leakage is well tolerated if the returned tidal volume is adequate (>7mL/kg). When securing the mask, there should be enough space to pass two fingers beneath the head straps. When necessary, a skin patch can be used to plug air leaks. Properly fitting the mask is difficult in edentulous patients and in those with a beard.

Methodology : The initial ventilator settings for NPPV are usually CPAP, 0 cm H₂O, and PSV 10 cm H₂O; the mask then is held gently on the patient's face until the patient is comfortable and in full synchrony with the ventilator. FiO₂ is titrated to achieve an O₂ saturation greater than 90%. After the mask is secured, CPAP is increased to 3 to 5 cm H₂O, and PSV is increased to achieve large (>7mL/kg) exhaled VT, a respiratory rate under 25 breaths/mt, and patient comfort. To avoid gastric distention, peak mask pressure should be kept at or below 30 cm H₂O. It has been reported that most patients have best comfort at CPAP of 5.3±2.8 cm H₂O and VT 7.1 mL/kg^{7,8}. The head of the bed should be elevated at or above a 45° angle. This facilitates manipulation of the mask and to minimize the risk of aspiration in patients without airway protection. It also provides increased development of inspiratory muscles and decreased abdominal compliance that increase rib cage expansion⁹. NIV is delivered continuously until ARF is resolved. Intermittent 5 to 15 min periods off NPPV are provided for oral intake or expectoration.

Monitoring

A. Response : 1. Physiological
a) Continuous oximetry
b) exhaled tidal volume
c) ABG should be obtained with 1 hour and, as necessary, at 2 to 6 hours intervals.

2. Objective: a) Respiratory rate
b) blood pressure
c) Rate

3. Subjective : a) dyspnea b) comfort c) mental alertness

B.) Mask : Fit, Comfort, Air leak, Secretions, Skin necrosis

C.) Respiratory muscle unloading : Accessory muscle activity, paradoxical abdominal motion

D.) Abdomen : Gastric distension, activation with inspiration.

First 30 minutes of NPPV are labor intensive. Bedside presence of a respiratory therapist or nurse familiar with this mode is essential. Providing reassurance and adequate explanation to the patient about what to expect is of the utmost importance.

Criteria to discontinue NPV are :

1. Inability to tolerate the mask because of discomfort or pain
2. Inability to improve gas exchange or dyspnea
3. Need for endotracheal intubation to manage secretions or protect airway
4. Hemodynamic instability
5. ECG-ischemia/arrhythmia
6. Failure to improve mental status in those with CO₂ narcosis.

Predictors of Success : In patients with hypoxemic ARF, predictors of response to mask CPAP therapy have included degree of hypoxea at initiation of therapy¹⁰, improvement in gas exchange, and respiratory rate shortly after applying CPAP¹¹.

Weaning : Following improvement in respiratory failure, patients are weaned from mechanical ventilation by lowering the amount of delivered pressure or by titrating periods off mechanical ventilation to patient's tolerance and objective

findings. At a low level of pressure support (5-8 cm H₂O), the patient is disconnected from the ventilator while receiving supplemental oxygen by nasal cannula or facemask.

Advantages of NPPV :

1. Noninvasiveness
 - a. Application (compared with endotracheal intubation)
 - b. Easy to implement
 - c. Easy to remove
 - d. Allows intermittent application
 - e. Improves patient comfort
 - f. Reduces the need for sedation
 - g. Oral patency (preserves speech, swallowing, and cough, reduces the need for nasogastric tubes)
2. Avoids the resistive work imposed by the endotracheal tube
3. Avoids the complications of endotracheal intubation, which are :
 - Early (local trauma, aspiration)
 - Late (injury to the hypopharynx, larynx, and trachea, nosocomial infections)

Disadvantages

1. **Systemic** : Slower correction of gas exchange abnormalities, Increased initial time commitment, Gastric distension (occurs in <2% patients)
2. **Mask** : Air leakage, Transient hypoxemia from accidental removal, Eye irritation

Facial skin necrosis - This is most common

complication with overall incidence of about 10%¹². Rapid healing occurs spontaneously within 2 to 7 days of discontinuing mask use. A patch or adhesive dressing can be used over pressure points to prevent this. Development of facial skin necrosis is not influenced by the duration of NPPV, age, type of respiratory failure, level of pressure applied or serum albumin level¹³.

3. Lack of airway access and protection

- Suctioning of secretions
- Aspiration

Cost effectiveness : NPPV decreases the cost of medical care by decreasing the duration of mechanical ventilation; decreased need for diagnostic studies or empiric antibiotic therapy for ventilator associated pneumonia and decreased need for intubation and mechanical ventilation. NIV decreases the intubation rate in COPD and ARF from 62% to 20%¹⁴.

Use of NIV in Hypoxemic Respiratory Failure : In hypoxemic patients, PEEP and CPAP are used for recruiting underventilated alveoli by increasing lung volume at end-expiration, which results in improved gas exchange. This can be achieved by mask. Increments of CPAP levels (5, 10 and 15 cm H₂O) cause proportional increases in minute ventilation, primarily caused by an increase in VT at 5 cm H₂O and respiratory rate at 15 cm H₂O. Studies have shown that addition of PSV to CPAP after extubation results in no change in hemodynamic parameters and increase in PaO₂/FiO₂ ratio and decrease in respiratory rate^{15, 16}.

Cardiogenic pulmonary edema : Intermittent positive pressure ventilation with or without PEEP, reduces venous return to the heart and thereby

reduces the preload. Also a portion of the airway pressure is transmitted to the left ventricle and thoracic aorta, thereby reducing the tension the heart has to develop (transmural pressure) to eject blood (decreased after load)

Application of mask CPAP in cardiogenic pulmonary edema patients results in improvement in cardiac index, significant drop in heart rate, reduction in plasma norepinephrine levels and reduction in airway resistance and work of breathing. Rasanen et al randomly assigned 40 patients with cardiogenic pulmonary edema (19 with acute myocardial infarction) to ambient airway pressure or 10 cm H₂O CPAP while FiO₂ was kept constant at 28% to 30%¹⁷. In contrast to controls, CPAP rapidly improved Pa O₂ and simultaneously decreased respiratory rate and heart rate with improvement in blood pressure. In chronic heart failure, nasal CPAP decreases recurrent apneas in association with Cheyne-Stocks respiration⁸, rises Left ventricular ejective fraction, and improvement in functional class from NYHA classes III and IV to class II. NPPV increases stroke volume and is also associated with rapid and sustained improvement in gas exchange.

In conclusion, the results of randomized clinical studies clearly support the continuous (not intermittent) application of mask CPAP in patients with CPE. Improvements in hemodynamic parameters, respiratory rate, dyspnea, and gas exchange are rapid. In patients failing to improve during mask CPAP, adding IPPV should be considered.

ARDS : In 1982, Covelli et al¹⁹ evaluated the use of mask CPAP in 33 patients of severe ARDS (PaO₂/FiO₂ of 133±7 mmHg). Significant improvement in PaO₂/FiO₂ was seen within 1 hour of therapy, shunt fraction decreased from 44±4% to 28±4%. Only 5 required intubation. But sufficient data

are not available to evaluate the clinical usefulness of NIV in ARDS and its use in clinical practice is discouraged, unless the patient is not a candidate for intubation.

Postoperative respiratory failure : Following upper abdominal surgery, applying mask CPAP improves FVC, FRC, and gas exchange²⁰. CPAP is superior to incentive spirometry or a regimen of coughing and deep breathing because it requires less effort from the patient, and less pain. To be most effective, CPAP treatment should be initiated soon after extubation and applied (intermittently) for several days.

Severe community-acquired pneumonia : In 1981, Suter et al applied CPAP by face mask in 19 patients with pneumonia and ARF¹¹. Intubation was avoided in 9(47%) patients, those with thick bronchial secretions had a high rate of failure (66%). In another study, NPPV improved gas exchange in more than 75% of patients and avoided intubation in 62% of patients with severe pneumonia with or without COPD¹³.

Postextubation respiratory failure and difficult weaning : Metaanalysis of seven studies (90 patients) with postextubation respiratory failure caused by respiratory muscle fatigue, reversible upper airway obstruction, or severe hypoxemia showed that applying NPPV avoided reintubation in 79% patients²¹. No patient developed nosocomial pneumonia.

Udwadia et al used NPPV as a weaning modality in difficult to extubate patients²². Patients with prolonged respiratory failure (median 31 days) with at least one failed extubation were switched to NPPV via nasal mask after extubation. In those with tracheostomy, the cuff was deflated and the tube occluded. If NPPV was well tolerated, decannulation was performed within 36 hours. Transfer to NPPV failed in only 2 patients, both had pulmonary fibrosis.

FOB & BAL in Hypoxemic Patients : NPPV delivered by face mask has been applied usefully to patients with severe hypoxemia ($\text{PaO}_2:\text{FiO}_2 < 100$) undergoing diagnostic fiberoptic bronchoscopy (FOB) with bronchoalveolar lavage²³ (BAL).

Balamugesh T.

M.D., D.M. (Pulm & Critical Care)

Lecturer, Christian Medical College, Vellore

References :

1. Bunnell S: The use of nitrous oxide and oxygen to maintain anesthesia and positive pressure for thoracic surgery. *JAMA* 58:835-838, 1912.
2. Poulton EP, Oxon DM: Left-sided heart failure with pulmonary edema-its treatment with the "pulmonary plus pressure machine." *Lancet* 231:981-983, 1936.
3. Barach AL, Martin J, Eckman M: Positive -pressure respiration and its application to the treatment of acute pulmonary edema. *Ann Intern Med* 12:754-795, 1938.
4. Barach AL, Martin J, Eckman M: Positive -pressure respiration and its application for the treatment of acute pulmonary edema and respiratory obstruction. *Proc Am Soc Clin Invest* 16:664-680, 1937.
5. Criner GJ, Travaline JM, Brennan KJ, et al : Efficacy of a new full-face mask for noninvasive positive pressure ventilation. *Chest* 106:1109-1115, 1994.
6. Vitacca M, Rubini F, Foglio K, et al: Noninvasive modalities of positive pressure Ventilation improve the outcome of acute exacerbations in GOLD patients. *Intensive Care Med* 19:450-455, 1993.
7. Marantz S, Webster K, Patrick W, et al: Respiratory responses to different levels of proportional assist (PAV) in ventilator-dependent patients. *Am Rev Respir Dis* 145:A525, 1992.
8. Shivaram U, Donath J, Khan FA, et al: Effects of continuous positive airway pressure in acute asthma. *Respiration* 52:157-162, 1987.
9. Druz WZ, Sharp JT: Activity of respiratory muscles in upright and recumbent humans. *J. Appl Physiol* 51:1552-1561, 1981.

-
-
10. Covelli HD, Weled BJ, Beekman JF: Efficacy of continuous positive airway pressure administered by face mask. *Chest* 81:147-150, 1982.
 11. Suter PM, Kobel N: Treatment of acute pulmonary failure by CPAP via face mask: When can intubation be avoided? *Klin Wochenschr* 59:613-616, 1981.
 12. Smurthwaite GJ, Ford P: Skin necrosis following continuous positive airway pressure with a face mask. *Anaesthesia* 48:147-148, 1993.
 13. Meduri GU, Turner RE, Abou-Shala N, et al: Non invasive positive-pressure ventilation via face mask: First-line intervention in patients with acute hypercapnic and hypoxemic respiratory failure. *Chest* 109:179-193, 1996.
 14. Brochard L, Mancebo J, Wysocki M, et al: Noninvasive ventilation for exacerbations of chronic obstructive pulmonary disease. *N Engl J Med* 333:335-361, 1988.
 15. Gregoretti C, Burbi L, Berardino M, et al: Noninvasive mask ventilation (NIMV) in trauma and major burn patients. *Am Rev Respir Dis* 145:A75, 1992.
 16. Jousela I: Endotracheal tube versus face mask with and without continuous positive airway pressure (CPAP). *Anaesthesiol Scand* 37:381, 1993.
 17. Rasanen J, Heikkila J, Downs J, et al: Continuous positive airway pressure by face mask in acute cardiogenic pulmonary edema. *Am J Cardiol* 55:296-300, 1985.
 18. Takasaki Y, Orr D, Popokin J, et al: Effect of nasal continuous positive airway pressure on sleep apnea in congestive heart failure. *Am Rev Respir Dis* 140:1578-1584, 1989.
 19. Covelli HD, Weled BJ, Beekman JF: Efficacy of continuous positive airway pressure administered by face mask. *Chest* 81:147-150, 1982.
 20. Linder KH, Lotz P, Ahnefeld FW: Continuous positive airway pressure effect on functional residual capacity, vital capacity and its subdivision. *Chest* 92:66-70, 1987.
 21. Meduri GU. Noninvasive positive-pressure ventilation in patients with acute respiratory failure. *Chest* 109, 1996.
 22. Udawadia ZF, Santis GK, StevenMH, et al: Nasal ventilation to facilitate weaning in patients with chronic respiratory insufficiency. *Thorax* 47:715-718, 1992.
 23. Antonelli M, Conti G, Riccioni L, et al: Pressure support ventilation administered with facial mask allows bronchoalveolar lavage in severely hypoxemic patients. *Intensive Care Med* 21:S42, 1995.

Rhinitis and Asthma - "One Airway, One Disease"

It has been a common clinical practice to treat allergic rhinitis (AR) and bronchial asthma (BA) as two separate diseases. The recent publication of the "Allergic Rhinitis and its Impact on Asthma" guidelines has definitely made this idea an outdated concept. The evidence is accumulating to look upon airways as a single entity from the tip of the nose to the alveolus. The evidence from epidemiologic, pathophysiologic and clinical data is so compelling, that the concept of "one

airway, one disease" (united airway disease) is becoming generally acceptable, although many questions still remain unanswered.

(A) Epidemiological Data : Epidemiological studies have consistently shown that asthma and rhinitis often coexist, 50-80% of patients with BA have AR and 20-30% of patients with rhinitis have associated BA. Prevalence rates of both BA and AR are increasing all over the world. Patients with AR have a three times greater chance than healthy subjects to

develop asthma, regardless of the atopic status of the patient. It has also been shown that AR usually precedes the onset of BA. In a 23 - year survey, about 2/3 of subjects with AR appeared more likely to develop asthma than the controls.

(B) Patho - Physiological Data : There are number of anatomical and physiological similarities between the nose and the lungs. Anatomically the upper and the lower airways share a common respiratory mucosa. Pathophysiological studies have suggested a strong relationship between AR and BA. Patients with concomitant AR and BA have higher number of eosinophils in both their nasal mucosa and in the lower bronchial mucosa. Eosinophilic inflammation has been found in the lower airways of AR patients without BA and in the upper airways of BA patients without AR. Also, segmental bronchial provocation in AR patients is shown to result in mast cell degranulation and influx of increased numbers of basophils in nasal mucosa. Nasal provocation with methacholine in asthmatic patients with rhinitis resulted in an increase in lower airway resistance.

(C) Clinical Data : A number of drugs like steroids, anti-leukotrienes and anti-cholinergics are effective in both AR and BA. The effect on asthma symptoms by treating rhinitis or sinusitis has been documented.

A number of studies have examined the effects of topical nasal steroids in patients with AR and coexistent BA. Such studies have shown improvement in both nasal and asthma symptoms and also in decreasing the level of bronchial hyperresponsiveness (BHR).

Further, effective management of AR with nasal steroids and oral antihistamines has shown to reduce the utilization of emergency health services and inpatients hospitalizations due to asthma among patients with co-existent asthma. Early treatment of AR may help to prevent the occurrence of asthma and sensitization to new allergens in children. Another report demonstrated a beneficial effect of bronchial treatment with inhaled corticosteroids on nasal symptoms. High-dose allergen immunotherapy for AR has also shown to decrease chest symptoms and BHR and the risk of developing asthma (in children). Data is now available to support the anti-inflammatory properties of a number of novel antihistamines and thus these agents may have significant effects in lower airway inflammation in patients who have both AR and BA. Anti-IgE antibodies are also promising agents in BA and AR.

Cardinal Differences : There are however some differences in the upper and lower airways. The nose has no airway smooth muscle. Upper airways contain prominent venous sinusoids and submucosal glands where as none of the former and very few of the latter are found in the lower airway. Nose and sinuses are rigid cavities, whereas the lower airways have an elastic parenchyma, rich in peribronchial smooth muscles. Similarly, the epithelium is fragile and more easily disrupted in patients with BA, whereas in rhinitis, epithelial integrity is more or less maintained. In the sub-basement zone, observable abnormalities highlighting the importance of remodeling process, are found in BA patients.

There are some differences in the management of the two conditions. Beta-2 agonist have a major role in BA but not in AR. Anti-histamines and decongestants are very effective in AR but not in asthma.

Possible Mechanisms for The Link :

Several mechanisms have been postulated for the close association between nasal and airway inflammation, including aspiration of infected or inflamed secretions, a nasobronchial reflex (controversial role), and the mechanical effect of nasal blockage that leads to mouth breathing and exposure to cold or dry air and an increase in allergens in the lower respiratory tract. Another mechanism is inflammation of the airways via systemic absorption of inflammatory mediators. Absorption of mediators generated within the nasal mucosa into the systemic circulation can result in an inflammatory response within the entire respiratory tract and not just in the nasal mucosa. According to this concept of disease, controlling the overall

inflammatory load with effective treatment of nasal tract inflammation serves to reduce inflammation in the lung and hence to control asthma. Another recently highlighted mechanism of the non-specific reactivity is the so-called neurogenic inflammation, in which neuropeptides (substance P, neurokinin A) are involved.

Conclusion : Airway allergy is not a disease confined to a specific target organ, but rather a disorder of the whole respiratory tract. To achieve optimal treatment for patients it is recommended that patients with persistent AR should be evaluated for asthma, and that patients with asthma should be evaluated for rhinitis. A strategy combining the treatment of both upper and lower airway disease to achieve better control of both should be our target.

R.S. Bedi and U.S. Bedi
Chest Consultants
Patiala