Noninvasive Ventilation and Lung Volume Reduction

Patrick Brian Murphy, MRCP, PhD[a,1],
Zaid Zoumot, MBBS, MRCP, MSc[b,1],
Michael Iain Polkey, PhD, FRCP[b,*]

KEYWORDS

• Lung volume reduction surgery • Noninvasive ventilation • Emphysema • Collateral ventilation

KEY POINTS

- Noninvasive ventilation given to patients with acute hypercapnic exacerbation of chronic obstructive pulmonary disease reduces mortality and morbidity.
- Lung volume reduction surgery is effective in patients with heterogeneous upper zone emphysema and reduced exercise tolerance, and is probably underused.
- Rapid progress is being made in nonsurgical approaches to lung volume reduction, but use outside specialized centers cannot be recommended presently.

INTRODUCTION

As parenchymal lung disease in chronic obstructive pulmonary disease (COPD) becomes increasingly severe there is a diminishing prospect of drug therapies conferring clinically useful benefit. As a result, when the load on the respiratory muscle pump exceeds the capacity, respiratory failure and/or breathlessness ensue.[1] Noninvasive ventilation (NIV) and lung volume reduction (LVR) surgery (LVRS) both address this in different ways; NIV is intended to offload the work of breathing by augmenting airflow into the lung and offsetting intrinsic positive end-expiratory pressure (PEEP), whereas LVR is intended to restore elastic recoil and improve VQ matching.

NIV

NIV provides ventilatory support without the need for endotracheal intubation and thus supports the respiratory muscle pump both by reducing the work of breathing and by improving tidal volume.[2] The ability to provide ventilatory support without the need for invasive mechanical ventilation has been one of the major advances in the management of acute respiratory failure complicating exacerbations of COPD. The use of NIV during decompensated hypercapnic respiratory failure secondary to acute exacerbations of COPD has become standard practice and forms part of national[3] and international[4] guidelines, whereas the use of NIV in patients with stable chronic

This work was supported in part by the NIHR Respiratory Biomedical Research Unit, which part funds the salary of M.I. Polkey and wholly funds the salary of Z. Zoumot. Z. Zoumot's institution has received reimbursement for trial expenses from PneumRx, Spiration, and Broncus. P. Murphy was previously supported by a grant for a study cofounded by ResMed and Respironics. M.I. Polkey receives fees for consulting from Respironics, Broncus, and PortAero, and his institution held on his behalf an award from PortAero for clinical research.

[a] Lane Fox Clinical Respiratory Physiology Group, Guy's & St Thomas' NHS Foundation Trust, London, UK; [b] NIHR Respiratory Biomedical Research Unit, Royal Brompton and Harefield NHS Foundation Trust, Imperial College London, London, UK
[1] Drs P. Murphy and Z. Zoumot contributed equally to this article.
* Corresponding author. NIHR Respiratory Biomedical Research Unit, Royal Brompton Hospital, Fulham Road, London, UK.
E-mail address: m.polkey@rbht.nhs.uk

Clin Chest Med 35 (2014) 251–269
http://dx.doi.org/10.1016/j.ccm.2013.10.011
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hypercapnic respiratory failure remains controversial and its use lacks a clear consensus.[5]

NIV DURING ACUTE HYPERCAPNIC RESPIRATORY FAILURE

Indications

Acute exacerbations of COPD represent a dynamic shift in the load-capacity-drive relationship of the respiratory system caused by an increased load exerted on it.[6] The increased load is mediated principally via changes in both inspiratory resistance and dynamic chest wall elastance, as well as a threshold load exerted by intrinsic PEEP.[7] This acute increase in load on the respiratory system causes a significant increase in the work of breathing, with transdiaphragmatic pressure changes in patients with acute exacerbations of COPD being double those of stable counterparts.[8,9] These effects can be further exacerbated by dynamic hyperinflation resulting from severe airflow limitation impairing expiratory flow time and causing an increase in operating lung volumes to a less advantageous part of the compliance curve.[10] In severe cases this load overcomes the ability of the respiratory muscle pumps to maintain carbon dioxide homeostasis. Increasing arterial partial pressure of carbon dioxide ($PaCO_2$) results in a decrease in arterial pH (<7.35); it is this respiratory acidosis that is the hallmark of acute decompensated respiratory failure.

Acute exacerbations of COPD complicated by respiratory failure are associated with high rates of inpatient mortality[11] and so offer the opportunity for therapeutic intervention. It is in this patient group that NIV was initially used as a means of preventing intubation. Initial promising data from small studies indicated a physiologic response with improvements in arterial pH, $PaCO_2$, and subjective dyspnea and a trend toward improvement in 30-day mortality in comparisons of the addition of NIV with standard care.[12] These encouraging results led to larger trials confirming the physiologic improvement that could be obtained with the application of NIV for acute hypercapnic respiratory failure during acute exacerbations of COPD, as well as indicating reduction in rates of intubation and mortality.[13,14] These initial intensive care–based studies have been supplemented by those conducted in respiratory specialist units confirming the reduction in length of hospital stay and mortality that is attributable to the use of NIV in this patient group.[15] These studies all enrolled patients with acute decompensated respiratory failure secondary to acute exacerbations of COPD and defined respiratory acidosis as a pH less than 7.35. The studies all excluded those deemed to need immediate intubation on either specific (pH<7.30) or general (clinician discretion) criteria. None of the studies used sham ventilation as a control but used standard care comparisons. Notwithstanding these limitations, the consensus from these studies is that NIV should be initiated in those patients with mild to moderate respiratory acidosis during exacerbations.

Predictors of Treatment Success and Treatment Failure

The use of NIV is now considered gold standard for the management of respiratory failure during COPD exacerbations but the exact characteristics of the patients who benefit remain debated (**Box 1**). The factors that predict treatment failure can be divided into patient-associated and therapy-associated factors.

Patients who, at the onset of therapy, have a poor nutritional state as indicated by lower body weight and lower percentage of ideal body weight, but not by lower serum albumin levels, are more likely to fail a trial of NIV and require invasive mechanical ventilation than patients with better nutritional status.[16] The presence of pneumonic consolidation indicates a higher mortality during unselected admissions caused by acute exacerbations of COPD.[17] In the subgroup of patients requiring NIV, those patients with pneumonic consolidation on chest radiograph are similarly

Box 1
Predictors of NIV success in acute hypercapnic exacerbation of COPD

Factors present at initiation:

- Poor nutritional status
- Lower Glasgow Coma Score
- More severe physiologic derangement
  - Higher APACHE II score
  - Lower pH
  - Higher $PaCO_2$
- Excessive secretions

Factors following initial trial of NIV:

- Failure to improve in the first hour
  - Respiratory rate
  - pH
  - $PaCO_2$
  - Glasgow Coma Score
- Poor patient-ventilator interaction
- Excessive mask leak

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