Thorax 1996;51:1069–1070

THORAX

Editorials

Negative pressure ventilation in acute hypercapnic chronic obstructive pulmonary disease

A K Simonds

Any technique that resurfaces for different indications over a century warrants re-evaluation. Negative pressure ventilation (nPV) is often associated with long term domiciliary respiratory support in chronic neuromuscular disorders, but was originally developed as a resuscitation aid in acute ventilatory failure. An early exponent was the Scottish inventor Alexander Graham Bell who devised a negative pressure vacuum jacket for the resuscitation of infants shortly after the death of his day old son in 1881.[1] The original iron lung or tank ventilator, devised by Drinker, was first manufactured in the UK in 1934 and pressed into action during the acute poliomyelitis outbreak in 1938 and subsequent epidemics in the 1940s and 1950s. Plum and Wolff[2] described the lifesaving impact of nPV in a severely ill group of patients with rapidly progressive ventilatory failure due to poliomyelitis (including bulbar cases), though it was evident that the iron lung was more effective than the smaller negative pressure devices such as cuirass in this acute situation.

Experience with nPV in exacerbations of chronic obstructive pulmonary disease (COPD) has been more mixed. Successful resuscitation and the long term survival of an emphysematous patient with decompensated hypercapnic respiratory failure treated in the iron lung was reported by Bourteline-Young and Whittenberger in 1951,[3] the authors attributing recovery to resetting of hypercapnic ventilatory drive. nPV was largely supplanted by intubation and intermittent positive pressure ventilation (IPPV) for acute respiratory failure in the 1950s, but recently the work of Rochester, Braun and others[4] has prompted the re-exploration of nPV during sleep and for short periods during the day to improve respiratory muscle function in ventilatory failure. Despite a promising outcome in restrictive disorders, the use of long term nPV in chronic ventilatory failure due to COPD has produced poor results.[5–7] Furthermore, the emergence of nasal intermittent positive pressure ventilation (NIPPV) in the last decade has focused attention on the outcome of this newer technique in acute exacerbations of COPD.[8 9]

A few centres have continued to use nPV as first line therapy for acute respiratory failure, gaining extensive experience in the process. On pp 1077–82 of this issue of *Thorax* Corrado *et al* present the results of iron lung ventilation in a large series of COPD patients with acute exacerbations treated over a 10 year period in a single institution.[10] Striking features of the study include the considerable level of cerebral impairment on admission and the severe degree of hypercapnia (mean Pco_2 14.9 kPa) and acidosis (mean pH 7.13). Although this is a retrospective uncontrolled series, it is instructive to compare the outcome of nPV with NIPPV in acute exacerbations of COPD. Both nPV and NIPPV produce a reduction in mortality (9% NIPPV,[9] 24% nPV) compared with some groups given standard treatment. A fall in Pco_2 and rise in pH after one hour of ventilation are good early prognostic signs using both techniques. However, careful interpretation of the results is required as the studies differ in several important respects. Whereas all the patients in the study by Corrado *et al* had a Glasgow coma score (GCS) of 8 or less, encephalopathy was less severe and unconsciousness an indication for intubation in the largest controlled study of NIPPV in acute COPD,[9] suggesting that nPV may be superior to NIPPV in some cases. At first sight it is surprising that comatose patients in the iron lung survive at all without succumbing to complications such as aspiration. The authors minimised this risk by using a nasogastric tube in all patients and placing a nasopharyngeal airway until consciousness was regained.

A plausible explanation for the favourable effects of nPV in this study may be that it has a more beneficial effect on cardiopulmonary haemodynamics in patients with acute COPD than positive pressure ventilation. Negative pressure applied to the chest wall increases venous return and can improve the performance of a right ventricle overloaded by an acute rise in pulmonary artery pressure caused by hypoxic pulmonary vasoconstriction, together with the effects of hyperinflation and intrinsic positive end expiratory pressure. However, relative to atmospheric pressure, left ventricular pressures are decreased by nPV which means that a greater pressure output is required from the left ventricle to maintain constant systemic blood pressure – that is, left ventricular ejection is impeded.[11] An increase in right ventricular size as a consequence of enhanced venous return may also decrease left ventricular diastolic compliance by displacing the interventricular septum. In contrast, positive pressure ventilation tends to reduce venous return and may augment left ventricular ejection. Independent of these changes, an increase in lung volume may reduce ventricular filling and alter pulmonary vascular resistance and capacitance.[11] As a result of these complex interactions, the effects of nPV and IPPV will differ significantly according to the underlying cardiopulmonary status of the patient. Predictably, the few studies that have examined the haemodynamic effects of positive and negative pressure ventilation have indicated no major advantage to either,[11 12] and it should be remembered that these circulatory effects are at their most profound at the initiation of ventilatory support and during weaning. Nevertheless, it is conceivable that nPV may be of particular benefit in patients subject to severe right heart overload.[13]

Considering other possible mechanisms of action, there is little to support the contention that nPV is superior to NIPPV in facilitating respiratory muscle rest,[14] and the relative importance of offloading the respiratory muscles in acute COPD is still debated. Improvements in arterial pH and blood gas tensions occur over a similar time course during nPV and NIPPV. Although the iron lung imposes a controlled pattern of ventilation and NIPPV equipment is usually operated in assist/control mode, it seems likely that the high negative pressures used by Corrado *et al* (mean -48 cm H_2O) and the greater flexibility of the new generation of iron lungs (which offer variable I:E ratio) are responsible for the better results seen in this series than with older versions of the iron lung.

On the debit side, iron lungs are expensive and experience in operating them – which is a major and underestimated factor – is restricted to a few centres. Patients are inaccessible and immobilised in the supine position during treatment, and the management of arterial and venous lines, chest drains, and urinary catheters is more of a practical challenge than during IPPV. Obstructive sleep apnoea may be provoked in predisposed individuals, but did not appear to be a problem in the study by Corrado *et al.* A possible benefit is that the confused hypercapnic patient may be more easily kept in the iron lung than persuaded to wear an NIPPV mask during the first critical hours of treatment. There is no information on whether patients prefer NIPPV to nPV, or find it less claustrophobic.

Several conclusions can be drawn. The authors provide persuasive evidence that, in experienced hands, nPV using the iron lung can be efficacious in patients with severe acute hypercapnic exacerbations. These results should not be extrapolated to other negative pressure devices such as the cuirass or Hayek oscillator without further studies. The outcome in individuals with a Glasgow coma score of less than 5 is dismal and intubation and IPPV seems preferable in this situation. As the authors suggest, a randomised comparison of nPV and NIPPV in patients with acute COPD would provide important physiological insights. However, notwithstanding the outcome of such a study, the effectiveness, efficiency and utility of any intervention such as nPV requires the translation of trial results into general clinical practice. With limited nPV facilities and expertise in most countries, it seems likely that NIPPV will remain the most widely applied non-invasive ventilatory method, with nPV continuing as a viable option in some centres into the next century.

Respiratory Support Service,
Royal Brompton Hospital,
London SW3 6NP, UK

A K SIMONDS

1 Woollam CHM. The development of apparatus for intermittent negative pressure respiration (1) 1832–1918. *Anaesthesia* 1976;**31**:537–47.
2 Plum F, Wolff HG. Observations on acute poliomyelitis with respiratory insufficiency. *JAMA* 1951;**146**:442–6.
3 Bourteline-Young HG, Whittenberger JL. The use of artificial respiration in pulmonary emphysema accompanied by high carbon dioxide levels. *J Clin Invest* 1951;**30**:838–46.
4 Rochester DF, Braun NM, Laine S. Diaphragmatic energy expenditure in chronic respiratory failure. *Am J Med* 1977;**63**:223–31.
5 Celli B, Lee H, Criner G, *et al.* Controlled trial of external negative pressure ventilation in patients with severe chronic airflow limitation. *Am Rev Respir Dis* 1989;**140**:1251–6.
6 Zibrak JD, Hill NS, Federman EC, Kwa SL, O'Donnell C. Evaluation of intermittent long term negative-pressure ventilation in patients with severe COPD. *Am Rev Respir Dis* 1988;**138**:1515–8.
7 Shapiro SH, Ernst P, Gray-Donald K, *et al.* Effect of negative pressure ventilation in severe chronic obstructive pulmonary disease. *Lancet* 1992; **340**:1425–9.
8 Bott J, Carroll MP, Conway JH, Keilty SEJ, Ward EM, Brown AM, *et al.* Randomised controlled trial of nasal ventilation in acute ventilatory failure due to chronic obstructive airways disease. *Lancet* 1993;**341**:1555–7.
9 Brochard L, Mancebo J, Wysocki M, Lofaso F, Conti G, Rauss A, *et al.* Noninvasive ventilation for acute exacerbations of chronic pulmonary disease. *N Engl J Med* 1995;**333**:817–22.
10 Corrado A, De Paola E, Gorini M, Messori A, Bruscoli G, Nutini S, *et al.* Intermittent negative pressure ventilation in the treatment of hypoxic hypercapnic coma in chronic respiratory insufficiency. *Thorax* 1996;**51**: 1077–82.
11 Pinsky MR. Cardiopulmonary interaction – the effects of negative and positive pleural pressure changes on cardiac output. In: Dantzker DR, ed. *Cardiopulmonary critical care.* Orlando: Grune and Stratton, 1986: 89–121.
12 Ambrosino N, Corbelli F, Torbicki A, *et al.* Haemodynamic effects of negative pressure ventilation in patients with COPD. *Chest* 1990;**97**: 850–6.
13 Penny DJ, Hayek Z, Redington AN. The effects of positive and negative extrathoracic pressure on pulmonary blood flow after the total cavopulmonary shunt procedure. *Int J Cardiol* 1991;**30**:128–30.
14 Belman MJ, Soo Hoo GW, Kuei JH, Shadmehr R. Efficacy of positive vs negative pressure ventilation in unloading the respiratory muscles. *Chest* 1990;**98**:850–6.