Biphasic positive pressure ventilation in acute cardiogenic pulmonary oedema

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Abstract

Non-invasive positive pressure ventilation (NIPPV) may be used in the treatment of acute cardiogenic pulmonary oedema. It has been shown to reduce the need for intubation and to improve left ventricular function. Patients do not need to be admitted to intensive care but can be managed in a coronary care unit. Two cases are described in this article. The indications, contraindications and complications of NIPPV are described and a practical guide to its use is given.

Key words: NIPPV, BiPAP, acute cardiogenic pulmonary oedema, left ventricular failure, intubation.

Introduction

In the 1980s non-invasive positive pressure ventilation (NIPPV) was developed for the management of obstructive sleep apnoea and respiratory failure associated with neuromuscular disease and chest wall deformity. More recently the usefulness of NIPPV has been recognised in the management of acute exacerbations of chronic obstructive pulmonary disease, where it has been shown to reduce mortality and intubation rates and to lead to shorter hospital stays when compared with controls.[1-4] NIPPV has many applications, but in the UK it is perhaps least often used in the treatment of acute cardiogenic pulmonary oedema; here it has been shown to reduce the need for intubation and to improve left ventricular function, and it can be used easily outside Intensive Care.

This article presents two cases in which biphasic positive pressure ventilation (BiPAP), was effective in severe acute cardiogenic pulmonary oedema: the patients were managed on the Coronary Care Unit. We also review the evidence, pathophysiology, contraindications, indications and complications of BiPAP use and offer a practical guide to the use of BiPAP in this setting. A glossary of terms used in this article is given in table 1.

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Table 1. Glossary of terms

- NIPPV: non-invasive positive pressure ventilation. By means of a tight-fitting mask, the system gives positive pressure support to the patient's own ventilation. Sometimes NIPPV is written as 'nasal intermittent positive pressure ventilation'
- CPAP: continuous positive airway pressure. A continuous pressure is delivered throughout the patient's respiratory cycle
- BiPAP: CPAP plus added higher pressure on inspiration. In non-invasive BiPAP, this is triggered by the patient's own respiration and is said to be more comfortable than CPAP
- PEEP: positive end-expiratory pressure, applied in both CPAP and BiPAP
- Strictly speaking CPAP is not ventilation although many people refer to CPAP and BiPAP as types of 'non-invasive ventilation'-using face masks to deliver positive pressure. In this article NIPPV can mean CPAP or BiPAP

Case 1

An 86 year old man attended Accident and Emergency by ambulance, having had sudden breathlessness for two hours. His past medical history was unknown at the time but he was a bilateral above-knee amputee. His skin was clammy, he had a respiratory rate of 35 per minute, pulse of 130 per minute, blood pressure (BP) of 234/130 mmHg and oxygen saturations of 83% on air. He was unable to speak, was sweating profusely and had a reduced level of consciousness. Examination revealed a gallop rhythm and widespread coarse inspiratory crepitations consistent with severe acute left ventricular failure. His chest X-ray showed florid pulmonary oedema. The ECG showed significant ST segment elevation in leads V1-V3 with ST-segment depression in leads V5 and V6. Blood gases on 10L oxygen showed pH 7.06, PCO_2 10.76, PO_2 19.3 and bicarbonate 23.1.

He was treated initially with intravenous frusemide 100 mg, diamorphine 2 mg and nebulised salbutamol. An isosorbide dinitrate 0.05% infusion was commenced at 0.3 mg/hr. A further bolus of frusemide 200 mg was given and the isosorbide dinitrate infusion was increased to 0.75 mg/hr. Despite these measures his condition did not improve.

NIPPV using nasal BiPAP was started once the patient was admitted to the Coronary Care Unit. The inspiratory pressure (IPAP) was set at 10 cm H_20 and the expiratory pressure (EPAP) at 6 cm H_20. 3L supplemental oxygen was added. The patient improved, showing less respiratory effort as evidenced by reduced intercostal recession and a slower respiratory rate. Blood

gases taken half an hour later showed pH 7.31, PCO_2 6.41, PO_2 11.25 and bicarbonate 24.1. He remained on BiPAP overnight. On review five hours later he was not breathless and he had a clear chest on examination.

Case 2

A 66 year old man arrived in Accident and Emergency having had acute shortness of breath for three hours. Three days previously he had been discharged home from the Coronary Care Unit after an anterior myocardial infarction, for which he was given thrombolysis. When the patient arrived he was clammy, mottled, agitated and restless, with a respiratory rate of 40 per minute, pulse of 130 per minute and BP 150/80 mmHg. On examination he had a gallop rhythm and widespread coarse inspiratory crepitations in the chest. The chest X-ray was consistent with severe pulmonary oedema. The ECG showed significant ST segment elevation in leads V1-V6, which was a new finding. Blood gases on arrival on 60% oxygen showed pH 7.1, PCO_2 9.02, PO_2 7.4 and bicarbonate 21.7.

Initial treatment in Accident and Emergency consisted of intravenous frusemide 200 mg, diamorphine 5 mg, nebulised salbutamol and an isosorbide dinitrate 0.05% infusion at 0.3 mg/hr. Although the isosorbide dinitrate infusion was increased to 0.75 mg/hr his condition did not improve.

He was admitted to the Coronary Care Unit, where thrombolysis was given and NIPPV using nasal BiPAP was commenced with an inspiratory pressure (IPAP) of 10 cm H_20 and an expiratory pressure (EPAP) of 6 cm H_20. Blood gases taken one and a half hours later showed pH 7.32, PCO_2 5.41, PO_2 9.54 and bicarbonate 20.7. An hour later BiPAP was discontinued and the patient was noted to be alert, with a respiratory rate of 18 per minute, heart rate of 90 per minute and BP of 125/70 mmHg. Blood gases on 35% oxygen showed a pH of 7.38, PCO_2 5.0, PO_2 10.2 and bicarbonate 22.1. He was discharged home a week later.

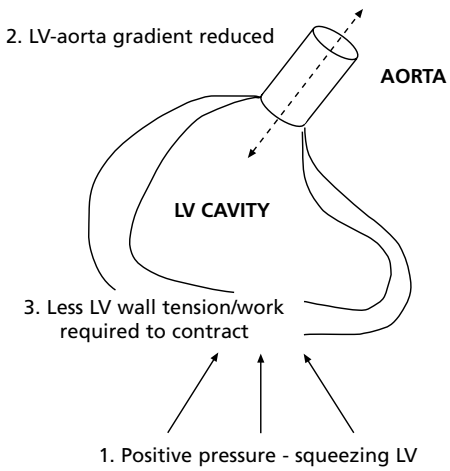
Discussion

NIPPV in the form of continuous positive airway pressure ventilation (CPAP) reduces the need for intubation in patients with acute cardiogenic pulmonary oedema and there is no significant difference in mortality.[5,6] Although the benefits of CPAP in this setting are well documented, the trials involving BiPAP are not well enough designed to be conclusive; in theory similar results could be expected. Hughes commented that BiPAP has overtaken CPAP in many institutions because clinical experience suggests that it is better tolerated by patients.[5]

Randomised trials show that the use of NIPPV on general wards makes no more demands on nursing staff than standard care, but these trials were done in centres where staff were experienced in its use.[2] Only four of the 15 coronary care units in our region use NIPPV as a treatment for severe acute left ventricular failure; usually patients are referred to the intensive care unit.

Our cases illustrate that non-invasive BiPAP is effective in acute cardiogenic pulmonary oedema in the kind of patients who would otherwise be referred for an urgent anaesthetic assessment and intubation – and that such patients can be managed easily on a coronary care unit. No differences in mortality rates between intubation and CPAP in acute cardiogenic pulmonary oedema have been found so far,[7-9] which indicates that NIPPV could provide an effective low-cost early intervention in such cases. Referral to the intensive care unit could be reserved for those patients who fail to improve despite use of NIPPV.

Figure 1. How NIPPV reduces afterload and improves left ventricular (LV) function



Afterload is defined as work required during contraction or LV wall tension
Intra-ventricular pressure = 2hT/r, where h = ventricular wall thickness,
T = LV wall tension and r = radius of ventricular cavity (Laplace's law)

Pathophysiology

The use of NIPPV in the acute setting is recommended earlier rather than later because the greater the degree of acidosis, the smaller the chances of improvement with time.[1] It should be used as an adjunct to full medical treatment of the underlying cause of acute respiratory failure.

Patients with severe acute cardiogenic pulmonary oedema may require intubation and ventilation for respiratory failure. Intubation and ventilation may have detrimental effects on the cardiovascular system because the sedation required may produce hypotension and the high intrathoracic pressures generated may reduce cardiac output. NIPPV, by contrast, has beneficial effects on the cardiovascular system by reducing left ventricular afterload (leading to an increase in stroke volume), improving arterial blood oxygenation and reducing the work of breathing.[7,8,10-13] Existing hypotension is not aggravated by NIPPV and several studies of CPAP in acute severe left ventricular failure show improvement in left ventricular function.[11,14,15] Because respiratory effort is maintained there is no reduction in venous return or cardiac output.[16] In addition, relief of respiratory distress leads to haemodynamic improvement and reversal of hypertension and tachycardia, probably through reduced sympathoadrenergic stimulation.[7]

Figure 2. Summary of the mechanism of NIPPV in acute cardiogenic pulmonary oedema

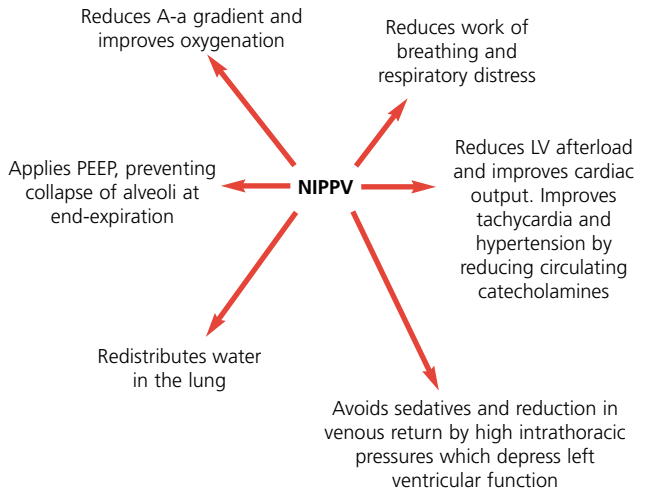


Table 2. Relative contraindications to the use of NIPPV

- Bullous lung disease
- Significant confusion
- Reduced conscious level
- Uncooperative patient
- High risk of aspiration
- Life-threatening hypoxaemia

NIPPV reduces afterload because the increased intrathoracic pressure has a squeezing effect on the left ventricle (LV). There is a subsequent reduction in the pressure gradient between the LV and the aorta, which has the effect of reducing the work required during contraction (see figure 1).

Patients with acute pulmonary oedema have increased lung water, reduced lung volumes and reduced lung compliance. In the respiratory system NIPPV causes a decline in the level of shunt because of redistribution of lung water from the alveolar space to the perivascular cuffs.[8] Positive end-expiratory pressure (PEEP) has been shown to reduce extravascular lung water and to increase lymphatic flow through the thoracic duct in animal studies.[17] NIPPV also leads to an increase in functional residual capacity and reduced work of breathing. PEEP increases the end-expiratory volume by distending lung units that are already open, preventing collapse of alveoli at the end of expiration, recruiting collapsed lung units, improving oxygenation and redistributing water within the lung.[18] Rapid improvement in respiratory rate, acidaemia and oxygenation and a reduction in intubation rates are reported in cardiogenic pulmonary oedema when CPAP is used.[9] Other randomised trials show marked

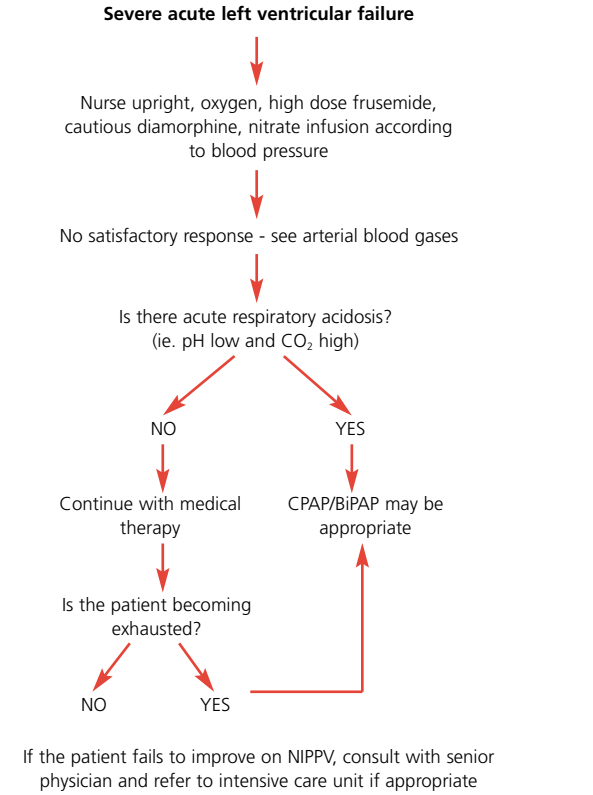
Table 3. Indications for NIPPV

- Alveolar hypoventilation ie. respiratory acidosis
- Unacceptable fatigue
- Hypoxaemia despite supplemental oxygen

Table 4. Complications of NIPPV versus intubation

NIPPV	Endotracheal intubation
• Oxygen toxicity	• Oxygen toxicity
• Necrosis of skin	• Laryngeal injury
• Barotrauma (smaller risk)	• Tracheal stenosis/tracheomalacia
• Aspiration	• Sinusitis
	• Barotrauma (greater risk)
	• Pneumonia
	• Complications of sedation/paralysis

Figure 3. A practical guide to the use of NIPPV in acute severe LVF



Key messages

- Non-invasive ventilation in severe acute cardiogenic pulmonary oedema reduces the need for intubation and there is no significant difference in mortality. It works by supporting ventilation and by improving left ventricular function
- Randomised trials show that the use of non-invasive ventilation on general wards makes no more demands on nursing staff than standard care
- Few coronary care units use non-invasive ventilation for patients with severe acute cardiogenic pulmonary oedema despite its obvious cost-effectiveness, the relative disadvantages of intubation and the lack of intensive care beds in the UK

physiological improvement in patients on NIPPV when compared with those on standard oxygen therapy alone.[8,9] A summary of the cardiovascular and respiratory effects of NIPPV in acute cardiogenic pulmonary oedema is given in figure 2.

Contraindications, indications and complications of NIPPV

Caution in the use of NIPPV is required in patients with bullous lung disease, who are susceptible to pneumothorax. Confusion, a significantly reduced conscious level, an uncooperative patient, high risk of aspiration and life-threatening hypoxaemia are also considered relative contraindications to the use of NIPPV (table 2).

The main indications for NIPPV in the acute setting are patients who exhibit clinically unacceptable or worsening alveolar hypoventilation, as evidenced by respiratory acidosis or clinically unacceptable or worsening fatigue. It may also be used in patients with unacceptable hypoxaemia despite high concentrations of supplemental oxygen[19] (table 3).

NIPPV is delivered via a tight-fitting face mask or nasal mask. Nasal masks may be preferable as they have a smaller risk of aspiration of gastric contents and will not lead to rebreathing of carbon dioxide in the event of circuit disconnection. However, patients may find it difficult to remember to breathe through the nose; chin straps may be used in this situation and, if there is no improvement, a full face mask should be used. Necrosis of the skin over the bridge of the nose can occur with prolonged use. Complications of NIPPV versus intubation and IPPV are shown in table 4. Patients require continuous observation and regular blood gas measurements.

A flow chart for the use of NIPPV in acute severe left ventricular failure is given in figure 3.

Conclusion

NIPPV in acute cardiogenic pulmonary oedema was first described as long as 50 years ago[20] yet, despite its well documented benefits, it is not a routine early treatment in many centres. Patients can be managed on a specialist ward such as a coronary care unit. In a country in which the UK Emergency Bed Service frequently reports a shortage of intensive care beds, why aren't we using this more?

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