Critical care in the emergency department: acute respiratory failure

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Accepted 17 October 2009
Published Online First 26 November 2010

ABSTRACT

Acute respiratory failure (ARF) is a frequent emergency department (ED) presentation. The definition and common causes of ARF are discussed in this article and ED management of the condition is discussed, using an illustrative case report. The paper considers the role of B-type natriuretic peptide in diagnosis and non-invasive ventilation (NIV) in treatment. Intensive care unit admission denial is common in ARF, although the evidence base is lacking. Finally, the decision-making process is described when a patient with ARF fails to improve with NIV.

Respiratory failure is a condition caused by failure of oxygenation, carbon dioxide clearance or both. Type I respiratory failure occurs when the partial pressure of oxygen in arterial blood would be less than 8 kPa when breathing air, and type II respiratory failure, ventilatory failure, when the partial pressure of carbon dioxide in arterial blood exceeds 6.5 kPa. When the latter condition occurs acutely there is an associated increase in hydrogen ion concentration in the plasma (pH <7.30). The annual incidence of severe acute respiratory failure (ARF) in the UK emergency department (ED) setting is approximately 150 per 10^5, leading to 50 000 admissions to intensive care. Common causes of ARF are listed in table 1.[1] The mortality of ARF depends upon the aetiology; an acute exacerbation of chronic obstructive pulmonary disease (COPD) carries an average mortality rate of 30% and causes 30 000 deaths each year in the UK.[2] ED management of ARF is directed at improving hypoxia and hypercarbia, reducing the work of breathing and reversing the cause of the acute deterioration. Further management may involve the selection of appropriate patients for mechanical ventilation or decisions on treatment limitation.

ILLUSTRATIVE CASE REPORT

A 68-year-old 90 kg woman is brought to the ED with a 24-h history of severe breathlessness. An ex-smoker, she has ischaemic heart disease and COPD and has had a number of previous admissions with similar complaints. She is usually independent in activities of daily living but does not often get out of her ground floor apartment due to severely limited exercise tolerance, estimated at 100 m. She is known to be on a variety of cardiac medications and inhaled bronchodilators. She does not receive home oxygen therapy.

On initial assessment she is distressed, mildly centrally cyanosed and clammy. Respiratory rate is 28 breaths per minute with the use of accessory muscles and prolonged expiratory phase. There are scattered rhonchi and bi-basal crackles on auscultation. Oxygen saturation is 86% after 15 min of oxygen therapy, initiated by paramedics. The heart rate is approximately 130 beats per minute, atrial fibrillation and blood pressure is 185/110 mm Hg. The jugular venous pressure cannot be determined clinically; however, there is mild pedal oedema. Arterial blood gases (ABG) on high flow oxygen via reservoir mask are as follows: pH 7.22, partial pressure carbon dioxide (pCO_2) 7.61 kPa, partial pressure oxygen (pO_2) 7.48 kPa, bicarbonate 28 mmol/l, base deficit 12.0 mmol/l, lactate 3.9 mmol/l. The working diagnosis is exacerbation of COPD and the patient is considered for non-invasive ventilation (NIV).

Questions (1)

i. What are the indications for NIV in the ED?
ii. Distinguish continuous positive airway pressure (CPAP), BiPAP and bi-level continuous positive airway pressure (BL-PAP).
iii. Describe how a patient is established on NIV. What are the most appropriate initial settings?

Discussion (1)

i. The term 'non-invasive ventilation' (NIV) conventionally includes both CPAP and BL-PAP. NIV enables the delivery of ventilatory support without the need for an endotracheal tube or tracheostomy. The interface is most commonly a well-fitting face mask, although nasal masks and hoods are used in other settings. The use of these techniques in ED patients has led to survival benefit, as well as fewer intubations and reduced length of stay in hospital.[3 4] The British Thoracic Society has recommended that NIV should be available 24 h a day in every hospital likely to admit patients with ARF.[5] NIV is frequently used in exacerbations of COPD,[4 6 7] but has also been used in other causes of ARF, including cardiogenic pulmonary oedema (CPO), pneumonia and severe acute asthma.[8–13] Eligible patients are those with acute hypoxia (with or without hypercarbia) and clinical evidence of acute respiratory distress. Factors predictive of success include younger age, less severe clinical disease and moderate respiratory acidosis (pH 7.10–7.35). NIV is contraindicated in respiratory arrest and when there is a high risk of pulmonary aspiration, haemodynamic instability or life-threatening arrhythmia. The treatment is relatively contraindicated in uncooperative patients and those who are unable to cough and clear tracheobronchial secretions.[5 7]

Table 1 Some causes of acute respiratory failure

Type I (hypoxaemic) respiratory failure	Type II (hypercarbic) respiratory failure
Asthma	COPD
COPD	Severe asthma
Pneumonia	Acute or acute on chronic hypoventilation:
Pulmonary oedema	Poisoning
Pneumothorax	Chest wall injury
Pulmonary embolism	Brain injury or disorder
Adult respiratory distress syndrome	Spinal cord injury or disorder
Acute alveolitis	Neuromuscular disorders
Pulmonary fibrosis	Obesity

Modified with permission from Chakrabati B, Angus RM. Ventilatory failure on acute take. Clin Med 2005;5:630–4.
COPD, chronic obstructive pulmonary disease.

ii. Positive end-expiratory pressure maintains airway pressure above atmospheric pressure throughout the breathing cycle. This is termed 'continuous positive airway pressure' (CPAP) in spontaneously breathing patients and 'expiratory positive airway pressure' (EPAP) in the context of non-invasive ventilation. CPAP preserves functional residual capacity, preventing the collapse of smaller airways at the end of expiration. This improves oxygenation and reduces the work of breathing. It also increases the transalveolar capillary gradient, aiding the reabsorption of alveolar oedema. CPAP is the mechanical treatment of choice in patients with type I ARF. However, it may increase intrathoracic pressure and impair venous return, leading to hypotension, particularly in hypovolaemic patients.

iii. BiPAP is a proprietary device for a system more correctly described as 'bi-level continuous airway pressure' (BL-PAP). In addition to EPAP a pressure-controlled ventilator delivers a predetermined assist with each spontaneous breath (inspiratory positive airway pressure; IPAP). Many ventilators allow adjustment of the inspiratory trigger sensitivity. BL-PAP improves both oxygenation and carbon dioxide clearance and is the device of choice in patients with type II respiratory failure, provided there is no established indication for endotracheal intubation.

The patient should sit upright or at least at 45° head-up tilt. The correct size mask easily covers the mouth and nose and at the outset is held against the patient's face (eg, by the patient herself), delivering 100% oxygen. Initial settings are an EPAP of 4 cm water and (for BL-PAP) an inspiratory support (IPAP) of 12 cm water. A back-up respiratory rate of 12–15 breaths per minute is set in case of apnoea. The mask is ultimately secured with a harness, adjusted to a tight fit. A wound dressing (eg, Granuflex) is applied to the nasal bridge and any other pressure areas to aid patient comfort and compliance. Some degree of air leak is inevitable at the interface. If excessive, this can interfere with triggering, preventing the assisted breath and promoting asynchrony. Air leak is minimised by use of the pressure support mode. Tachypnoea is common in ARF and the ventilator must be able to support a spontaneous rate of at least 40 breaths per minute and an inspiratory flow of 60 l/min.[5]

Baseline investigations include ABG and chest radiograph. Continuous monitoring of vital signs and cardiorespiratory parameters is established and findings recorded. Minimum standards for monitoring include ECG, peripheral oxygen saturation (SpO_2) and non-invasive blood pressure. It is usual to insert an arterial line for blood pressure monitoring and repeat ABG analysis. The fractional inspired oxygen is set to achieve a target SpO_2 of 88–92% in COPD exacerbations.

CASE PROGRESSION

The patient is established on BL-PAP. The anterior–posterior chest radiograph shows probable cardiomegaly, overinflated lungs with bi-basal shadowing and prominent bronchovascular markings (figure 1). There is no convincing evidence of consolidation or pneumothorax. Repeat ABG at 30 min are as follows: pH 7.29, pCO_2 5.83 kPa, pO_2 8.28 kPa, bicarbonate 26 mmol/l, base deficit 10.0 mmol/l, lactate 3.2 mmol/l.

The plasma N-terminal Pro-B-Type natriuretic peptide (BNP) is 970 pg/ml. The diagnosis is amended to ARF due to an exacerbation of COPD, with at least some contribution from CPO.

Questions (2)

i. What is the role of B-type natriuretic peptides in the assessment of acute dyspnoea in the emergency setting?
ii. Discuss the efficacy and safety of BL-PAP versus CPAP in CPO.

Discussion (2)

i. B-type natriuretic peptides are synthesised by the ventricular myocardium and released into the blood stream in response to myocardial wall stress due to stretch or ischaemia. BNP is synthesised as a pre-prohormone polypeptide (pre-proBNP) comprising 134 amino acids. This molecule is cleaved to produce proBNP (108 amino acids) and an N-terminal signal

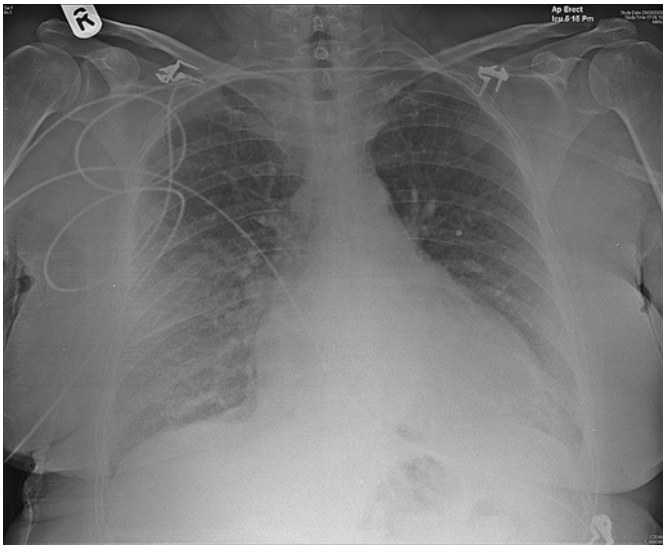

Figure 1 Portable anteroposterior chest radiograph showing cardiomegaly and overinflated lungs consistent with pulmonary emphysema. There is no pneumothorax and no evidence of consolidation.

peptide (26 amino acids). Just before its release into the circulation, proBNP is cleaved in equal proportions into the biologically active 32 amino acid C-terminal BNP and the biologically inert 76 amino acid N-terminal fragment (NT-proBNP).

BNP exerts potent diuretic and natriuretic effects, promotes vasodilatation and cardiovascular response to ischaemia, and downregulates the renin–angiotensin–aldosterone system. BNP has a half-life of 20 min in the circulation and is cleared via neural endopeptidase and endocytosis, whereas NT-proBNP has a half-life of 120 min and is renally excreted.[14 15] Several studies have evaluated the clinical utility of these peptides in the assessment of acute dyspnoea. Both BNP and NT-proBNP are sensitive and specific biochemical markers that can be used to distinguish pulmonary from cardiac causes of acute dyspnoea, in conjunction with clinical assessment.[16–18]

When the plasma NT-proBNP level is below the appropriate cut-off (taking account of the patient's age and gender and the clinical situation) it is unlikely that heart failure is the cause of dyspnoea. An elevated plasma NT-proBNP level indicates the need for further cardiac assessment. The higher the NT-proBNP level the higher the positive predictive value for the presence of heart failure. In the present case the NT-proBNP level exceeds 900 pg/ml, considered to be diagnostic of acute heart failure in this age group.[16]

ii. There has been much debate on the comparative safety and effectiveness of CPAP and BL-PAP in the early management of ED patients with acute CPO. In the context of pulmonary oedema and hypercapnia, BL-PAP may be superior because of the presumed additional benefit of increased minute volume and the reduction in the work of breathing. However, a much-cited paper by Mehta and colleagues[19] suggested that the use of BL-PAP increased the risk of myocardial infarction (MI) in these patients. This effectively stopped trials for a period of almost a decade.

Two meta-analyses[20 21] suggested that there is no difference between CPAP and BL-PAP for any outcome parameter. A subsequent re-analysis of the data of Mehta *et al*[19] showed that more patients with ischaemic heart dsease were recruited to the BL-PAP arm, confounding the trial outcomes. A more recent study from Italy excluded patients with cardiac ischaemia and infarction. They found no difference in the subsequent MI rate between the two modalities.[22]

A recent large pragmatic trial in the UK ED setting, the 3CPO trial,[8] investigated the effectiveness of non-invasive ventilation (CPAP or BL-PAP) compared with standard oxygen therapy, and the comparative effectiveness of CPAP and BL-PAP. There was no difference between either modality in terms of efficacy, safety and hospital length of stay. Investigators specifically recruited CPO patients with respiratory acidosis to maximise the likelihood of benefit of BL-PAP over CPAP.

CPAP and BL-PAP are thus similarly effective and safe in ED patients with acute pulmonary oedema.

FURTHER CASE PROGRESSION

The patient has been on BL-PAP for an hour and is becoming alternatively drowsy and agitated. She is pulling at the mask and appears to be becoming exhausted. She is tachycardic and hypertensive. Ventilator settings have been increased to EPAP 10 cm water and IPAP 25 cm water. The SpO_2 is 92%. Repeat ABG show the following: pH 6.91, pCO_2 12.67, pO_2 10.94, bicarbonate 21.2, base deficit 11.25.

Questions (3)

i. What are the potential causes of failure to improve on NIV and what treatments are available?
ii. Should this patient be considered for endotracheal ventilation, or for palliative care?

Discussion (3)

i. Possible causes of failure include progression of the underlying condition, development of complications such as MI or pneumothorax, failure of concomitant treatment and lack of efficacy or poor compliance with the ventilator. Poor compliance may result from asynchrony due to undetected inspiratory effort, delay in response to inspiration or failure to detect the end of inspiration (breath stacking).

Failure to improve oxygenation should prompt an increase in fractional inspired oxygen and EPAP, while failure to improve the hypercarbia should lead to an increase in IPAP. A range of ventilators is available for ED use with variable degrees of sophistication. The practitioner should be familiar with the ventilator and able to adjust the mode, mandated breathing rate and trigger sensitivities to maximise efficacy, patient comfort and compliance.

The patient has developed hypercarbic narcosis and is delirious. Respiratory arrest is imminent. A respiratory stimulant such a doxapram, combined with NIV, may be considered as an alternative to intubation.

ii. The principal diagnosis is an acute exacerbation of COPD. If repeat ABG show no significant improvement after 2–4 h, sooner if clinical deterioration, then endotracheal ventilation (ETV) should be considered.[4] ETV is not appropriate in every patient and a decision on whether or not to escalate treatment often has to be made.

The range of intensive care unit (ICU) mortality in COPD patients requiring ETV is 11–45%, reflecting a variable threshold for intubation in different institutions. The range for hospital mortality is 14–82%.[23] Factors associated with non-survival include previous mechanical ventilation, severe obstructive lung disease (on pulmonary function tests), anaemia, malnutrition, high sickness scores and active malignancy. Long-term oxygen therapy may be associated with a 5-year survival of approximately 50% depending on the nutritional status of the patient, disease severity and the absence of anaemia.[24] The effect on prognosis of hypercarbia at presentation is unknown.

Patients with COPD and respiratory failure are often denied ICU admission due to supposed poor prognosis: 'He/she cannot be weaned.' Despite evidence to the contrary a variety of myths persist.

Kaelin *et al*[25] examined the prognostic value of simple indices in determining 6-month survival in 35 COPD patients requiring ETV. The authors concluded that survival cannot be predicted from simple data available to the physician at the time of intubation.

Pearlman[26] studied 205 physician estimates of survival for a hypothetical patient with ARF complicating COPD. Estimates of survival ranged from 1 month to 5 years. There was no correlation between predictive accuracy and physicians' level of training. More recently, Wildman and colleagues[27] demonstrated significant differences between actual and predicted survival to 6 months. The difference between predicted and observed was greatest in those patients who had the poorest outlook.

Menzies *et al*[28] concluded that premorbid activity was the strongest indicator of outcome and this highly correlated with several clinical and physiological measurements, especially forced

expiratory volume in 1 s (FEV_1). The derangement in FEV_1needs to render the patient completely dependent upon carers for activities of daily living to have relevance.

Connors *et al*[29] conducted a multicentre, prospective cohort study of 1016 patients admitted with exacerbation of COPD and partial pressure of carbon dioxide in arterial blood greater than 50 mm Hg. Twenty-six per cent of patients reported good, very good or excellent quality of life at 6 months and 50% of these patients were still alive at 2 years.

ETV and ICU admission, like all decisions to adopt extraordinary medical intervention, depend upon the answers to a series of questions concerning the principles of beneficence, non-maleficence and distributive justice. Patients have a right to expect clinicians to preserve life and ameliorate symptoms, but futile treatments are unethical because they may be burdensome on the individual and wasteful of resources. When possible the options should be discussed with the patient. The clinician must be clear on the diagnosis and the prognosis, and be aware that the latter is determined by the chronic health evaluation, functional status before admission and the acute sickness severity. Early treatments also influence the acute physiology, and it may be prudent to defer a decision on ICU admission until resuscitative measures are underway. Given the choice, most patients will request access to potentially life-prolonging interventions, but a minority may opt for comfort measures only.[30]

CASE CONCLUSION

The patient is pre-oxygenated in the sitting up position for 5 min, using a self-inflating bag–valve–mask device attached to wall oxygen at 15 l/min, and synchronised to the patient's spontaneous breathing. Intravenous cannulae are sited in both antecubital fossae and an infusion of Ringer's lactate solution is in progress. No pretreatment drugs are given. Anaesthesia and paralysis are induced with etomidate 18 mg and suxamethonium 100 mg while the patient is sitting upright. The patient is laid flat after induction and a size 8 endotracheal tube is passed into the trachea without difficulty. The patient receives an infusion of propofol and is attached to a portable ventilator delivering intermittent mandatory ventilation with the following settings: rate 14; inspiratory:expiratory ratio 1:3; minute volume 7.5 l/min; positive end-expiratory pressure 5 cm water. A nasogastric tube and urinary catheter are placed and invasive blood pressure monitoring is in progress. The patient is transferred to the ICU.

SUMMARY AND CONCLUSIONS

NIV is a useful modality in ARF, whatever the cause. BL-PAP is as safe and effective as CPAP in patients who have heart failure. Elderly patients with ARF due to exacerbation of COPD are frequently denied ICU admission on inadequate grounds. In most cases, ETV should be offered in patients who are ineligible for, or fail on, NIV. There are few evidence-based indications for withholding treatment, particularly in the ED setting when information may be incomplete. The severity of the acute physiological derangement, response to early treatment and the previous functional status are relevant. When possible, the patient's views should be taken into account.

Competing interests None.

Provenance and peer review Commissioned; not externally peer reviewed.

REFERENCES

1. **Chakrabati B,** Angus RM. Ventilatory failure on acute take. *Clin Med* 2005;**5**: 630–4.
2. **Britton M.** The burden of COPD in the U.K.: results from the Confronting COPD survey. *Respir Med* 2003;**97**(Suppl C):S71–96.
3. **Ram FSF,** Picot J, Lightowler J, *et al*. Non-invasive positive pressure ventilation for treatment of respiratory failure due to exacerbations of chronic obstructive pulmonary disease. [update in Cochrane Database Syst Rev 2004;(3):CD004104; PMID: 15266518] [update of Cochrane Database Syst Rev 2003;(1):CD004104; PMID: 12535509]. *Cochrane Database Syst Rev* 2004(1):CD004104.
4. **Brochard L,** Mancebo J, Wysocki M, *et al*. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. *N Engl J Med* 1995;**333**:817–22.
5. **British Thoracic Society.** Non-invasive ventilation in acute respiratory failure. *Thorax* 2002;**57**:192–211.
6. **Plant PK,** Owen JL, Elliott MW. Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial. *Lancet* 2000;**355**:1931–5.
7. **Keenan SP,** Sinuff T, Cook DJ, *et al*. Which patients with acute exacerbation of chronic obstructive pulmonary disease benefit from noninvasive positive-pressure ventilation? A systematic review of the literature [summary for patients in Ann Intern Med 2003 Jun 3;138(11):I27; PMID: 12779309]. *Ann Intern Med* 2003;**138**:861–70.
8. **Gray A,** Goodacre S, Newby DE, *et al*. Noninvasive ventilation in acute cardiogenic pulmonary edema. *N Engl J Med* 2008;**359**:142–51.
9. **Keenan SP,** Sinuff T, Cook DJ, *et al*. Does noninvasive positive pressure ventilation improve outcome in acute hypoxemic respiratory failure? A systematic review. *Crit Care Med* 2004;**32**:2516–23.
10. **Antonelli M,** Conti G, Rocco M, *et al*. A comparison of noninvasive positive-pressure ventilation and conventional mechanical ventilation in patients with acute respiratory failure. *N Engl J Med* 1998;**339**:429–35.
11. **Meduri GU,** Turner RE, Abou-Shala N, *et al*. Noninvasive positive pressure ventilation via face mask: first-line intervention in patients with acute hypercapnic and hypoxemic respiratory failure. *Chest* 1996;**109**,179–93.
12. **Meduri GU,** Cook TR, Turner RE, *et al*. Noninvasive positive pressure ventilation in status asthmaticus. *Chest* 1996;**110**,767–74.
13. **Fernandez MM,** Villagra A, Blanch L, *et al*. Non-invasive mechanical ventilation in status asthmaticus. *Intensive Care Med* 2001;**27**,486–92.
14. **Elin RJ,** Winter WE. Laboratory and clinical aspects of B-type natriuretic peptides. *Arch Pathol Lab Med* 2004;**128**:697–9.
15. 5Rawlins ML**,** Owen WE, Roberts WL. Performance characteristics of four automated natriuretic peptide assays. *Am J Clin Pathol* 2005;**123**:439–45.
16. **Januzzi JL,** Camargo CA, Anwaruddin S, *et al*. The N-terminal Pro-BNP Investigation of Dyspnoea in the Emergency Department (PRIDE) Study. *Am J Cardiol* 2005;**95**: 948–54.
17. **Dao Q,** Krishnaswamy P, Kazanegra R, *et al*. Utility of B-type natriuretic peptide in the diagnosis of congestive heart failure in an urgent-care setting. *Am J Cardiol* 2001;**37**:379–85.
18. **Moe GW,** Howlett J, Januzzi JL, *et al*. N-terminal pro-B-type natriuretic paptide testing improves the management of patients with suspected acute heart failure. Primary results of the Canadian Prospective Randomised Multicenter IMPROVE-CHF Study. *Circulation* 2007;**106**:3103–10.
19. **Mehta S,** Jay GD, Woolard RH, *et al*. Randomised, prospective trial of bilevel versus continuous positive airway pressure in acute pulmonary edema. *Crit Care Med* 1997;**25**:620–8.
20. **Masip J,** Roque M, Sanchez B, *et al*. Noninvasive ventilation in acute cardiogenic pulmonary edema: systematic review and meta-analysis. *JAMA* 2005;**294**:3124–30.
21. **Peter JV,** Moran JL, Phillips-Hughes J, *et al*. Effect of non-invasive positive pressure ventilation (NIPPV) on mortality in patients with acute cardiogenic pulmonary oedema: a meta-analysis. *Lancet* 2006;**367**:1155–63.
22. **Bellone A,** Monari A, Cortellaro F, *et al*. Myocardial infarction rate in acute pulmonary edema: noninvasive pressure support ventilation versus continuous positive airway pressure. *Crit Care Med* 2004;**32**:1860–5.
23. **Nevins ML,** Epstein SK. Predictors of outcome in patiets with COPD requiring invasive mechanical ventilation. *Chest* 2001;**119**:1840–9.
24. **Chambellen A,** Chailleux E, Similowski T. Prognostic value of the hematocrit in patients with severe COPD receiving long-term oxygen therapy. *Chest* 2005;**128**: 1201–8.
25. **Kaelin RM,** Assimacopoulos A, Chevrolet JC. Failure to predict six-month survival of patients with COPD requiring mechanical ventilation by analysis of simple indices. *Chest* 1987;**92**:971–8.
26. **Pearlman RA.** Variability in physician estimates of survival for acute respiratory failure in chronic obstructive pulmonary disease. *Chest* 1987;**91**:515–21.
27. **Wildman MJ,** Sanderson C, Groves J, *et al*. Implications of prognostic pessimism in patients with chronic obstructive pulmonary disease (COPD) or asthma admitted to intensive care in the UK within the COPD and asthma outcome study (CAOS): multicentre observational cohort study. *BMJ* 2007;**335**:1132.
28. **Menzies R,** Gibbons W, Goldberg P. Determinants of weaning and survival among patients with COPD who require mechanical ventilation for acute respiratory failure. *Chest* 1989;**95**:398–405.
29. **Connors AF,** Dawson NV, Thomas C, *et al*. Outcomes following acute exacerbation of severe chronic obstructive lung disease. The SUPPORT investigators (Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments). *Am J Respir Crit Care Med* 1996;**154**:959–67.
30. **General Medical Council (GMC).** *Witholding and withdrawing - guidance for doctors*. http://www.gmc-uk.org/guidance/ethical_guidance/witholding_lifeprolonging_guidance.asp (accessed Mar 2010).

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Emerg Med J 2011 28: 94-97 originally published online November 26, 2010
doi: 10.1136/emj.2005.030643