Acute cardiogenic pulmonary oedema: reflecting on the management of an intensive care unit patient

Jaime Skinner and Aidín McKinney

ABSTRACT

Aim: The aim of this paper is to reflect upon the management interventions of non-invasive ventilation (NIV) and diuretic therapy that were implemented for a patient admitted to an intensive care unit (ICU) with acute cardiogenic pulmonary oedema.

Background: Acute pulmonary oedema is a serious and life-threatening complication of acute heart failure, particularly if this results from an ischaemic event. Statistics highlight that of those patients treated for acute cardiogenic pulmonary oedema, approximately only one third were alive after 1 year. Many of these patients will require intensive care management in order to restore homeostasis. It is therefore imperative that nurses understand the condition and the relevant management of it in order to maximize the already poor prognosis.

Results: Using Driscoll's (2000) reflective model to guide critical thinking, this paper reflects on the management of one patient who was admitted to ICU with acute cardiogenic pulmonary oedema as a result of heart failure. Although there are many aspects of patient management that can be explored, specific care interventions that this patient received in relation to NIV and diuretic therapy will be considered. The evidence base for their use, together with the relevant nursing management issues, and patient implications will be critically analysed and outlined.

Conclusions: This paper identifies that standard therapy for acute cardiogenic pulmonary oedema is largely supportive and aimed at promoting gaseous exchange. It also highlights that nurses have a key role in ensuring that these essential treatments are as efficacious as possible.

Relevance to clinical practice: By using a reflective analysis approach, this paper highlights how reflecting on practice improves knowledge and understanding of the use of NIV and diuretic therapy interventions and should facilitate nurses working in ICU to become more competent in ensuring that the treatment provided for acute cardiogenic pulmonary oedema is as successful as possible.

Key words: Effectiveness of health care intervention • Intensive care

INTRODUCTION

The prevalence of heart failure is rapidly increasing due to the age of the population (Nicholas, 2004) and according to Collins *et al*. (2008), it is a worldwide problem of epidemic proportions. The prognosis of this condition is poor with an estimated 40% mortality rate within a year of diagnosis (Lakasing and Francis, 2006). Furthermore, National Institute for Health and Clinical Excellence (2003) indicates that it accounts for 5% of all emergency admissions to hospital resulting in 1 million inpatient bed days/year, and therefore makes heart failure a phenomenally important issue for health care. Although the mortality rate is high for congestive heart failure, the risk is actually greater for those who present with acute cardiogenic pulmonary oedema as a symptom (Shamagian *et al*., 2007). Acute pulmonary oedema (APO) is a serious and life-threatening complication of congestive heart failure and although heart failure has many causes, including hypertension, myocarditis and arrhythmias to name a few (Lip, 2000; cited in Nicholas, 2004), the most frequent cause is myocardial infarction (Lakasing and Francis, 2006; Ekman *et al*., 2007; Fiutowski *et al*., 2008). A recent study by Shamagian *et al*. (2007) showed

Authors: J Skinner, BSc Nursing Sciences, RN, Staff Nurse, Intensive Care Unit, Ulster Hospital, Dundonald, Belfast, BT16 1RH, UK; A McKinney, MSc, BSc (Hons) Nursing, PGCHET, RN, Teaching Fellow, School of Nursing and Midwifery, Queens University Belfast, Medical Biology Centre, Belfast, BT9 7BL, UK

Address for correspondence: A McKinney, School of Nursing and Midwifery, Queens University Belfast, Medical Biology Centre, 97 Lisburn Road, Belfast, BT9 7BL, UK

E-mail: a.mckinney@qub.ac.uk

BACCN Nursing in Critical Care

that patients with congestive heart failure and APO resulting from an ischaemic event have less chance of survival. These poor statistics are supported by Ekman *et al*. (2007) who found that, of those patients treated for acute cardiogenic pulmonary oedema, only 35% were alive after 1 year. Given the poor prognosis associated with this condition, it is evident that many of these patients will require admission to intensive care units (ICU). It is important therefore that nurses understand the issues and key interventions involved in the treatment to maximize patient care and provide the best possible outcome for these patients. By reflecting on a patient cared for in practice, this paper will outline the pathophysiology of cardiac failure with resulting pulmonary oedema and will explore the use of non-invasive ventilation (NIV) and diuretic therapy as management interventions that were used in the treatment of this condition.

REFLECTIVE APPROACH

Reflective practice is a process of examination that practitioners can use to give careful consideration to past experiences, with the aim of learning from them in order to improve practice and understanding (Sice, 2006). It is an approach to learning which is patient centred and which acknowledges the variation and at times confusing world of the practice environment (Price, 2004). As such, reflection may be described as a means whereby nurses can bring their theoretical knowledge and the practice of nursing into closer alignment (O'Regan and Fawcett, 2006). By doing so, it is advocated that clinical practice should be enhanced (Driscoll, 2000).

To structure any process of reflection, there are several reflective models available that give guidance, such as Kolb's (1984) experiential learning model and Gibb's (1988) six-step process. However, the following reflective account will use Driscoll's (2000) model of 'what?, so what? and now what?' to guide the process. As a novice to reflection, this three-stage process was appealing as it is clear, simple and straightforward to use.

The what?

In this reflective account, the patient reflected on is a 69-year-old lady who was admitted to the ICU with APO secondary to heart failure. The patient's name will be changed for confidentiality reasons in keeping with Nursing and Midwifery Council (2008) guidelines and will be referred to as Mrs Smyth. In Mrs Smyth's case, her heart failure was caused by a myocardial infarction she had unwittingly suffered 2 days previously when she had experienced severe pain between her shoulder blades lasting approximately 20 min. She had mistakenly assumed the pain was muscular, hence the delayed admission to hospital. On arrival to the unit, she was clearly distressed and extremely short of breath. Within a few hours of admission, various treatment interventions had been commenced, including the use of NIV and diuretic therapy.

As a junior nurse in intensive care, I had not previously encountered an admission of a patient with APO secondary to heart failure. As such, my experience of caring for this patient was very much based on guidance and support from more senior colleagues, rather than that gleaned from evidence-based guidelines. It was therefore felt necessary to reflect upon this practice encounter. In particular, it prompted me to investigate the causes and pathophysiology of heart failure. It also encouraged an analysis into the interventions of NIV and diuretic therapy in managing this patients' care, as the rationale for the use and management of these was not fully understood.

So what?

Overview of preload, afterload and contractility

Before describing the pathophysiology of heart failure, it is useful to review the key determinants of cardiac function. Cardiac output (CO) is a measurement of the heart's overall performance and it measures blood flow to the peripheries. It is defined as the volume of blood that is pumped out of the heart each minute and is expressed in litres per minute. It can be calculated using a simple equation, i.e. CO = heart rate (HR) × stroke volume (SV). HR is simply the amount of times the heart will beat each minute and SV is the amount of blood ejected from the ventricle with each heart beat (Martini and Bartholomew, 2000). In the resting adult, SV averages approximately 70 mL/beat and HR is about 75 beats/min. This gives an average CO of 5·25 L/min (McCance and Huether, 2006). There are several factors that determine the SV and therefore heart function. They include preload, afterload and contractility. Preload is the volume and pressure of blood that stretches the heart muscle before contraction. The more a muscle fibre is stretched, the greater the force of contraction, which is referred to as 'Starling's Law'. However, if the muscle is overstretched then the force of contraction is actually reduced. Afterload is the force of resistance against which the heart has to pump in order to eject blood during contraction, e.g. the diameter of the blood vessel into which the blood is being pumped. A narrow vessel will increase the force, thereby increasing the

afterload. Contractility is simply the force generated by the myocardium (Martini and Bartholomew, 2000).

Pathophysiology of heart failure

Heart failure is a complex syndrome that can result from any structural cardiac disorder that impairs the heart's ability to function effectively as a pump to support physiological circulation. The causes of heart failure vary ranging from heart damage of unknown cause, such as dilated cardiomyopathy, to heart damage that is associated to a history of hypertension. However, the most common cause of heart failure in the UK is due to coronary artery disease, with a large number of patients having had a myocardial infarction in the past (National Institute for Health and Clinical Excellence, 2003). An initial insult to the heart, such as that from a myocardial infarction, causes dysfunction or a loss of myocytes and therefore can result in contractile failure of the heart muscle (Bales and Sorrentino, 1997). In Mrs Smith's case, this insult was indeed as a consequence of a myocardial infarction. The pain of a myocardial infarction can often present as an atypical chest pain, such as pain radiating to the back. This is not uncommon but can lead to misdiagnosis or a delay in the patient seeking medical attention as occurred in this case (Hand, 2001). The resulting cardiac dysfunction prevents adequate tissue perfusion because of a reduced volume of blood being pumped from the heart (Hand, 2001). This leads to the stimulation of certain compensatory mechanisms that attempt to improve functioning, but instead become responsible for many of the clinical manifestations of heart failure (Bales and Sorrentino, 1997). The compensatory mechanisms are mainly neurohormonal and cardiovascular, which work together and will affect the preload and afterload of the heart. Combined effects cause vasoconstriction and sodium and water retention, which in the short term will increase blood pressure (BP) and improve circulation (McCance and Heuther, 2006). However, in the longer term, the increased arterial tone from the vasoconstriction results in an increased afterload, which simply puts pressure on the workload of an already failing heart that is struggling to pump efficiently (Lakasing and Francis, 2006). This increase in afterload impedes blood flow from the left ventricle. Consequently, this can lead to impaired CO resulting in a reduced BP and thus poor tissue and cellular perfusion. Furthermore, the increased volume and pressure in the left ventricle will result in a backflow into the pulmonary venous system, where the preload or pressure also increases leading to an accumulation of fluid in the lungs (Lakasing and Francis, 2006). The sodium and water retention increases the volume of fluid in an attempt to raise the BP, but the increased preload simply exacerbates the problem of fluid retention (Lakasing and Francis, 2006).

Fluid is kept to a minimum in the lungs, mostly by a balance between hydrostatic pressures and capillary/osmotic pressures (Bucher and Melander, 1999). However, eventually when there is a backflow of blood into the pulmonary circulation, the hydrostatic pressures rise and eventually exceed the osmotic pressure causing fluid to move into the interstitial spaces leading to pulmonary oedema (Bucher and Melander, 1999). As fluid accumulates in the alveoli, some collapse, some are compressed by surrounding oedematous alveoli and others cannot function as the airways are full of fluid (Kelly, 1999). Blood that flows past oedematous alveoli cannot pick up as much oxygen as normal and hypoxaemia occurs (Kelly, 1999). The decreased lung compliance and the resistance within the airways makes it very difficult for the lungs to open and the patient has to work incredibly hard to initiate inspiration and get air into the lungs (McCance and Heuther, 2006). In this situation, death is a foregone conclusion unless prompt treatment is initiated.

It is therefore evident that patients with heart failure may have a number of symptoms and these vary greatly depending on the severity of the syndrome, the aetiology, precipitating and exacerbating factors and identification of concomitant disease. The most common symptoms, however, are breathlessness, fatigue, exercise intolerance and fluid retention. Out of these breathlessness is perhaps acknowledged as one of the main primary symptoms of heart failure. However, again the degree of breathlessness amongst patients vary and thus is often used to grade the severity of symptoms into one of four functional classes (National Institute for Health and Clinical Excellence, 2003) (Table 1).

Non-invasive ventilation – indications for use

On admission to ICU, Mrs Smyth was conscious but clearly in respiratory distress with symptoms of breathlessness, tachypnoea, wheeze, cyanosis, tachycardia and she was sweaty, pale, cold and clearly anxious. All these symptoms are common for patients in heart failure with APO (Kelly, 1999; Ekman *et al.*, 2007; Collins *et al.*, 2008). The first priority was to support Mrs Smyth's airway and breathing. Her respiratory distress required immediate intervention, which was in the form of NIV. NIV refers to the provision of ventilator support through the patient's upper airway using a mask or similar device, and thus is distinguished from those which bypass the upper airway with a tracheal tube (British Thoracic

Table 1 New York Heart Association classification of heart failure symptoms (National Institute for Health and Clinical Excellence, 2003)

Class	Symptoms
I	No limitations. Ordinary physical activity does not cause fatigue, breathlessness or palpitation.
II	Slight limitation of physical activity. Such patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, breathlessness or angina pectoris (symptomatically 'mild' heart failure).
III	Marked limitation of physical activity. Although patients are comfortable at rest, less than ordinary physical activity will lead to symptoms (symptomatically 'moderate' heart failure).
IV	Inability to carry on any physical activity without discomfort. Symptoms of congestive cardiac failure are present even at rest. With any physical activity increased discomfort is experienced (symptomatically 'severe' heart failure).

Society, 2002). One of the first uses of NIV was for the treatment of hypoventilation at night in patients with neuromuscular disease. This lead to NIV being used widely in patients with chronic hypercapnic respiratory failure caused by chest wall deformity, neuromuscular disease or impaired central respiratory drive. However, NIV also started to be used in patients with acute hypercapnic respiratory failure, particularly in chronic obstructive pulmonary disease. Even more recently, the value of NIV has been indicated in the case of APO (Masip *et al.*, 2000; Kaye *et al.*, 2001; Masip *et al.*, 2005; Nehyba, 2007) and in particular in the case of cardiogenic pulmonary oedema by the British Thoracic Society (2002). The form of NIV used for Mrs Smyth was 'continuous positive airway pressure' otherwise known as CPAP. Confusion commonly arises between NIV and CPAP in clinical practice and whether or not CPAP is a type of NIV. However, CPAP can refer to the non-invasive application of positive airway pressure using a face mask, as occurred in the case of Mrs Smyth, rather than in conjunction with invasive techniques. Masip *et al.* (2005) describe it as oxygen being delivered via a tight fitting mask or helmet with an expiratory valve that maintains a constant positive intrathoracic pressure. There are, however, certain contraindications to NIV set out by the British Thoracic Society (2002) (Table 2), which include impaired consciousness, inability to protect the airway, pneumothorax, vomiting or raised intracranial pressure. Elliott (2004) argues that these 'contraindications' have been mostly identified by the fact that they were part of exclusion criteria in controlled trials; therefore, it is more correct to state that NIV is simply not proven in these areas rather than contraindicated. Nevertheless, it seems appropriate to take these points into consideration as it would be unsafe to commence NIV on a patient who was actively vomiting or had an unresolved pneumothorax. Refusal by the patient should also preclude the use of NIV; however, refusal might come from an acutely hypoxic and confused patient so obtaining consent could be problematic (Woodrow, 2003a). In Mrs Smyth's case, there were no contraindications and she gave consent for CPAP to be used (Table 2).

Table 2 Contraindications to non-invasive ventilation (NIV)

Facial trauma/burns
Recent facial, upper airway, or upper gastrointestinal tract surgery*
Fixed obstruction of the upper airway
Inability to protect airway*
Life-threatening hypoxaemia*
Haemodynamic instability*
Severe comorbidity*
Impaired consciousness*
Confusion/agitation*
Vomiting
Bowel obstruction*
Copious respiratory secretions*
Focal consolidation on chest X-ray*
Undrained pneumothorax*

*NIV may be used, despite the presence of these contraindications, with extreme caution, provided contingency plans for tracheal intubation have been made, or if a decision has been made not to proceed to invasive ventilation (British Thoracic Society, 2002).

NIV has benefits for both respiratory and cardiac function in a patient with acute cardiogenic pulmonary oedema. The positive pressure applied to the lungs and the volume of air between breaths reverses atelectasis, recruits alveoli, improves oxygenation, reduces the work of breathing, decreases pulmonary oedema and improves cardiac function (Pang *et al.*, 1998). The positive pressure helps to keep alveoli partially inflated and for those that have collapsed it will reinflate them (Woodrow, 2003a). It will also force the interstitial fluid back into the pulmonary circulation, while preventing further fluid moving back into the alveoli (Mehta *et al.*, 1997; Masip *et al.*, 2000; Nehyba, 2007). These combined effects result in reduced pulmonary oedema, which then improves the work of breathing and reverses hypoxaemia (Woodrow, 2003a; Nehyba, 2007). Cardiac function is improved as the increased intrathoracic positive pressure decreases the left ventricular afterload, thereby improving CO (Kaye *et al.*, 2001).

NIV – the nurses' role

NIV is a medical treatment but it is nurses who monitor its effectiveness and care for the patients. Elliot (2004, p. 288) stresses the importance of the expertise of the nursing staff, stating 'nurses must be familiar

with it because they are the only care providers who are with the patient 24 h/day. They must be both confident about the technique and recognize when there are problems'. The British Thoracic Society (2002) also supports the role of the nurse in NIV and actually recommends that it should be nursing staff that initiate it and run it. The benefits of NIV will alleviate most of the symptoms associated with APO, but there are potential problems that can hinder the success of the treatment. Continuous monitoring and assessment of the patient is necessary including hourly measurement of all observations, such as HR, BP, respiratory rate, depth and rhythm, pulse oximetry and arterial blood gases. Changes in any of these observations could indicate a problem with the treatment (Sawkins, 2001).

There are other physiological or psychological problems associated with NIV that need to be considered. Patients such as Mrs Smyth who present with APO will be tired, breathless, agitated, anxious and quite possibly confused (Collins *et al*., 2008). These symptoms will limit the patients' ability to carry out certain tasks. They will need assistance with positioning to prevent pressure sores developing and a pressure sore assessment tool should be used such as the Braden Scale (Woodrow, 2003b).

Mouth care is particularly important for these patients as NIV, sodium retention and breathlessness will make their mouth very dry. Humidifying the oxygen will help reduce this but humidification is not without problems (Lellouche, 2002). The temperature needs to be regulated to prevent airway burns and the heated water within the reservoir provides a perfect medium for bacterial growth, although sterile water within a closed system should help reduce this risk (Woodrow, 2003b).

Gastric distension is another problem with NIV. It occurs as the oesophagus and trachea lie so close together that patients will often swallow the air whilst attempting to get used to the high pressure of oxygen (Preston, 2001). It is estimated that this will occur in approximately half the patients who receive NIV (Parsons, 2000). A nasogastric tube will help expel the excess air. Nausea and vomiting is common but can be dangerous if the face mask is *in situ* as the vomit can be inhaled and aspirated into the lungs causing aspiration pneumonia. Antiemetics will help reduce the risk of this but it is important for nurses to communicate with their patients and be aware of problems such as nausea (Woodrow, 2003a).

Patients with severe heart failure are more at risk of developing malnutrition according to Gibbs (2000), therefore nutrition is very important. However, Sawkins (2001) highlighted that nurses may be reluctant to remove the face mask, possibly because they may experience reluctance from the patient to put the mask back on, or it may simply be because of a fear of oxygen levels dropping. Whatever the reason, it is important for nurses to become more confident in this area and should gain input from the dietician (Nicholas, 2004).

Complications of NIV

The principal problem with NIV and non-compliance involves the tight fitting mask (Jarvis, 2006). NIV masks are larger then normal oxygen masks, they are tight fitting and usually secured with straps around the patients head. A common complication due to the tightness is pressure ulcers on the bridge of the nose (Jarvis, 2006). Removing the mask periodically or using a dressing on the nose can help prevent this. Jarvis (2006) suggests using a full face mask or total face mask to reduce skin necrosis. She also explains how the process of mask fitting is difficult and there must be an adequate seal to achieve the continuous positive pressure to ensure success of the treatment. NIV will usually exacerbate feelings of distress and agitation in patients especially those who are confused and this may result in them trying to remove the mask continuously, which will reduce its effectiveness. Elliott (2004) comments that NIV needs 20 min continuous treatment before it will be of any benefit. Anxiety can be reduced and confidence increased if the nurse lets the patient hold the mask whilst becoming accustomed to it (Woodrow, 2003a). Opiates such as morphine can also help settle the patient (Masip *et al*., 2000). Indeed in this case, Mrs Smyth had received a 2 mg bolus of morphine before commencement of NIV, which helped her to relax and tolerate the treatment well. However, it is important to highlight that opiates should be used with caution in order not to further depress an already compromised respiratory system and should also be used sparingly in conjunction with good ongoing assessment.

Despite these problems, a study by Ekman *et al*. (2007), which investigated how patients with acute cardiogenic pulmonary oedema perceived their condition and treatment, showed that out of all the patients who received NIV which was the majority of patients in the study, none of them mentioned the treatment which suggests that it was well tolerated. This supported another study by Kelly *et al*. (2002) who concluded that NIV is a routine treatment for these patients and was normally well tolerated with only minor discomfort. Mrs Smyth supported this view and complied with her treatment very well and accepted it as a welcome relief to her breathing difficulties.

Alternative forms of NIV

CPAP, as one form of NIV, may have its disadvantages but there are few alternatives to this treatment. Bilevel NIV is another form of NIV that requires a ventilator and provides two levels of pressure, one to assist patients with inspiratory positive airway pressure and the other, such as CPAP, maintains an expiratory positive pressure (Woodrow, 2003b). There are many studies comparing CPAP and bilevel NIV but with conflicting results. An early study by Mehta *et al*. (1997) was terminated prematurely as 4 out of 10 patients on bilevel NIV suffered myocardial infarctions. This was a small study and therefore these events could have been coincidental. Indeed, later studies by Masip *et al*. (2000) and Chadda *et al*. (2002) found no association between bilevel NIV and myocardial infarctions. In fact, their results favoured bilevel to CPAP. Other authors would also support this view (Somauroo, 2000; Woodrow, 2003b; Nadar *et al*., 2005). Despite these studies and reviews, the British Thoracic Society (2002) stated cautiously that bilevel NIV should only be used when CPAP is unsuccessful.

Crane *et al*. (2004) also concluded that patients with acute cardiogenic pulmonary oedema are more likely to survive to hospital discharge, if treated early with CPAP and conventional therapy than with bilevel and conventional therapy. This supports studies by Lin *et al*. (1995); Kelly *et al*. (2001) and Kelly *et al*. (2002). With such conflicting evidence, it is possible that further studies are needed to compare the two forms of NIV, but at present it would seem that CPAP should be the ventilatory mode of choice in patients with acute cardiogenic pulmonary oedema (British Thoracic Society, 2002; Elliott, 2004).

Regardless of treatment, the ultimate aim is to improve a patient's condition and prevent them from deteriorating further. NIV will reduce the need for intubation (Nehyba, 2007) and avoiding invasive ventilation will usually be in the patient's best interests, as this brings more risks such as ventilator acquired pneumonia. However, for patients who need this additional support, NIV should not be inappropriately prolonged thereby delaying intubation and ventilation. This will only lead to unnecessary deterioration and possible death (Woodrow, 2003b). Woodrow (2003b) suggests that if NIV fails to work after approximately 6 h then intubation and ventilation should follow, although individual assessment of patients is important here. In Mrs Smyth's case, she tolerated NIV for a period of 14 h and the mask was removed as her condition and breathing had greatly improved.

Use of diuretic therapy

In addition to NIV, Mrs Smyth also received diuretics to remove the excess fluid that had accumulated in her lungs. In congested patients, diuretics will reduce preload and intracardiac pressures and improve cardiac performance (Khatib, 2008; Sorrentino, 1997). There are different categories of diuretics that include loop diuretics, thiazides and potassium sparing diuretics. Within the kidney, loop diuretics such as furosemide increase the secretion of sodium and water in the loop of Henle and increase potassium secretion in the distal tubule of the nephron (Nicholas, 2004). Loop diuretics are the most commonly used diuretic in heart failure (Nicholas, 2004; Wang and Gottlieb, 2008). Thiazides inhibit sodium and chloride reabsorption in the distal tubule of the nephron, which then leads to sodium and water excretion (Nicholas, 2004; Wang and Gottlieb, 2008). Potassium sparing diuretics, e.g. spironolactone, increase sodium and chloride excretion but cause potassium retention (Nicholas, 2004). They are a relatively weak group and are usually prescribed in conjunction with loop and thiazide diuretics because of their ability to retain potassium (Nicholas, 2004). With the other groups, a common side-effect is hypokalaemia, and regular monitoring of urea and electrolytes is important to detect this so that it can be treated (Davies, 2000). If untreated, hypokalaemia can cause severe and life-threatening cardiac arrhythmias (Gupta and Neyses, 2005). Other side-effects common to all diuretics include hypotension, dehydration and renal dysfunction; therefore, medical staff need to be careful not to be over aggressive with their use of diuretics and nurses need to be aware of what symptoms may precipitate with these side-effects (Nicholas, 2004).

Diuretics remain the first-line treatment for patients with heart failure; however, surprisingly there is very little evidence supporting them (Khatib, 2008). There is no conclusive evidence that one type of diuretic is better than another; therefore, any effective one will be appropriate whether it is one drug or a combination of different ones. There is also a lack of evidence on mortality or morbidity benefits from diuretics. It is simply accepted that they are the most efficacious drugs available to relieve symptoms of shortness of breath and oedema rapidly (Gupta and Neyes, 2005). Khatib (2008) discusses how this lack of evidence is partly, because diuretics were introduced, before the advent of large clinical trials with mortality end points and as time has progressed, placebo-controlled trials of diuretics in heart failure would now be considered unethical because of their now well-established benefits in improving the symptoms of congestion in heart

failure. Furthermore, the lack of a suitable alternative for control of symptoms of congestion means that diuretics will continue to be used as first-line therapy in the management of heart failure. In Mrs Smyth's case, she received bolus doses of furosemide 20 mg with very good effect. Blood results were monitored every few hours to monitor her potassium levels. Mrs Smyth's acute symptoms were relieved within 24 h and she was discharged from ICU and admitted to the cardiology ward for further investigations, so that appropriate treatment could be initiated to prevent further events occurring in the future.

Now what?

The prognosis of patients with acute cardiogenic pulmonary oedema remains grave, despite the increased knowledge base and developments in heart failure care that have occurred in recent years (National Institute for Health and Clinical Excellence, 2003; Riley, 2007). These patients often have several heart-related conditions and their chance of survival is poor (Ekman *et al*., 2007). Surprisingly, heart failure has a worse prognosis than most cancers (Nicholas, 2004). Standard therapy is largely supportive and treatment is aimed at promoting gaseous exchange, reducing the heart's oxygen demand and reducing fluid overload (Kelly, 1999). With the prognosis already so poor for these patients, it is essential therefore that the treatment they do receive is as efficacious as possible so that they are given every chance possible to survive. As outlined in this paper, it is important therefore for nurses to be able to identify heart failure and understand and reflect on the treatment and management interventions provided. In particular, they must have an understanding of the evidence-based rationales for any treatment interventions used. Furthermore, it is essential to have an awareness of when it is most appropriate to introduce treatment interventions and how to maximize their effectiveness. Using Driscoll's (2000) model to reflect on this particular patient's care, it has promoted an enhanced appreciation of the value of NIV and when it should or should not be used in practice. It also reinforces the view that the individual management of treatment interventions such as these will differ slightly depending on the patient's clinical condition and their psychological adjustment to care.

CONCLUSION

This paper shows how the integration of reflective theory, evidence base and nursing practice can lead to the development of personal and professional knowledge (Marks, 2001). By doing so, it is hoped that this process will facilitate and encourage nurses to provide the best possible care to patients and where necessary to adjust their practice accordingly. Reflective analysis, such as this, should also help to ensure that treatment interventions, such as NIV and diuretic therapy, are as successful as possible and go some way towards improving the prognosis of cardiogenic pulmonary oedema.

WHAT IS KNOWN ABOUT THIS TOPIC

- The mortality rate for those with acute cardiogenic pulmonary oedema is very high.
- It is not, however, a condition that is commonly reviewed in the ICU nursing literature.

WHAT THIS PAPER ADDS

- This paper provides a critical reflection on the key interventions used to restore homeostasis, namely NIV and diuretic therapy.
- It draws attention to the relevant knowledge and practice required by nurses in order to manage the care of these patients as successfully as possible.

REFERENCES

Bales AC, Sorrentino MJ. (1997). Causes of congestive heart failure. Prompt diagnosis may affect prognosis. *Postgraduate Medicine*; **101:** 44–45.

British Thoracic Society. (2002). Non-invasive ventilation in acute respiratory failure. *Thorax*; **57:** 192–211.

Bucher L, Melander S. (1999). *Critical Care Nursing*. Philadelphia: Saunders.

Chadda K, Annane D, Hart N, Gajdes P, Raphael JC, Lofaso F. (2002). Cardiac and respiratory effects of continuous positive airway pressure and non-invasive ventilation in acute cardiac pulmonary edema. *Critical Care Medicine*; **30:** 2457–2461.

Collins S, Storrow AB, Kirk JD, Pang PS, Diercks DB, Gheorghiade M. (2008). Beyond pulmonary edema: diagnostic, risk stratification and treatment challenges of acute heart failure management in the emergency department. *Annals of Emergency Medicin*; **51:** 45–57.

Crane SD, Elliott MW, Gilligan P, Richards K, Gray AJ. (2004). Randomised controlled comparison of continuous positive airways pressure, bilevel non-invasive ventilation and standard treatment in emergency departments patients with acute cardiogenic pulmonary oedema. *Emergency Medicine Journal*; **21:** 155–161.

Davies M. (2000). ABC of heart failure. Management: diuretics, ACE inhibitors and nitrates. *British Medical Journal*; **320:** 428–431.

Driscoll J. (2000). *Practising Clinical Supervision: A Reflective Approach*. London: Bailliere Tindall.

Ekman I, Ekstrand L, Schaufelberger M. (2007). Pulmonary oedema – a life threatening disease. *European Journal of Cardiovascular Nursing*; **6:** 259–264.

Elliott M. (2004). Non-invasive mechanical ventilation. *Medicine*; **32:** 99–101.

Fiutowski M, Waszyrowski T, Krzeminska-Pakula M, Kasprzak JD. (2008). Pulmonary edema prognostic score predicts in-hospital mortality risk in patients with acute cardiogenic pulmonary edema. *Heart and Lung*; **37:** 46–53.

Gibbs G. (1988). *Learning by Doing: A Guide to Teaching and Learning Methods*. Oxford: Further Education Unit.

Gibbs C. (2000). ABC of heart failure. Non–drug management. *British Medical Journal*; **320:** 366–369.

Gupta S, Neyses L. (2005). Diuretic usage in heart failure: a continuing conundrum in 2005. *European Heart Journal*; **26:** 644–649.

Hand H. (2001). Myocardial infarction: part 1. *Nursing Standard*; **15:** 45–53.

Jarvis H. (2006). Exploring the evidence base of the use of non-invasive ventilation. *British Journal of Nursing*; **15:** 756–759.

Kaye DM, Mansfield D, Aggarwal A, Naughton MT, Esler MD. (2001). Acute effects of continuous positive airway pressure on cardiac sympathetic tone in congestive heart failure. *Circulation*; **103:** 2336–2338.

Kelly AL. (1999). Left ventricular systolic heart failure resulting in acute pulmonary oedema; pathophysiology and nursing management in the emergency department. *Australasian Emergency Nursing Journal*; **2:** 5.

Kelly C, Newby DE, Boon NA. (2001). Support ventilation versus conventional oxygen. *Lancet*; **357:** 1126.

Kelly CA, Newby DE, McDonagh TA. (2002). Randomised controlled trial of continuous positive airway pressure and standard oxygen therapy in acute pulmonary oedema; effects on plasma brain natriuretic peptide concentrations. *European Heart Journal*; **23:** 1379–1386.

Khatib R. (2008). Diuretics in heart failure part 1: pharmacology and mechanisms. *British Journal of Cardiac Nursing*; **3:** 310–314.

Kolb D. (1984). *Experiential Learning as the Science of Learning and Development*. New Jersey: Prentice Hall.

Lakasing E, Francis H. (2006). Diagnosis and management of heart failure. *Primary Health Care*; **16:** 36–39.

Lellouche F. (2002). Effect of the humidification device on the work of breathing during non-invasive ventilation. *Intensive Care Medicine*; **28:** 1558–1589.

Lin M, Yang YF, Chiang HT. (1995). Reappraisal of continuous positive airway pressure therapy in acute pulmonary oedema. *Chest*; **107:** 1379–1386.

Lip G. (2000). ABC of heart failure: aetiology. *British Medical Journal*; **320:** 104–107. Cited in: Nicholas M. (2004).

Marks C. (2001). Reflective practice in thermoregulatory nursing care. *Nursing Standard*; **15:** 38–41.

Martini FH, Bartholomew EF. (2000). *Essentials of Anatomy and Physiology*, 2nd edn. New Jersey: Prentice-Hall Inc.

Masip J, Betbese AJ, Paez J, Vecilla F, Canizares R, Padro J, Paz MA, Otero J, Ballus J. (2000). Non-invasive pressure support ventilation versus conventional oxygen therapy in acute cardiogenic pulmonary oedema: a randomised trial. *Lancet*; **356:** 2126–2132.

Masip J, Roque M, Sanchez B, Fernandez R, Subirana M, Exposito JO. (2005). Non-invasive ventilation in acute cardiogenic pulmonary edema: systematic review and meta-analysis. *JAMA*; **294:** 3124–3130.

McCance KL, Huether SE. (2006). *Pathophysiology: The Biologic Basis for Disease in Adults and Children*, 5th edn. St. Louis: Mosby Inc.

Mehta S, Jay GD, Woolard RH. (1997). Randomised, prospective trial of bi-level vs. continuous positive pressure in acute pulmonary oedema. *Critical Care Medicine*; **25:** 620–628.

Nadar S, Prasad N, Taylor RS, Lip GYH. (2005). Positive pressure ventilation in the management of acute and chronic cardiac failure: a systematic review and meta-analysis. *International Journal of Cardiology*; **99:** 171–185.

National Institute for Health and Clinical Excellence. (2003). *Chronic Heart Failure: Management of Chronic Heart Failure in Adults in Primary and Secondary Care*. London: NICE.

Nehyba K. (2007). Continuous positive airway pressure ventilation. Part two: indications and contra-indications. *British Journal of Cardiac Nursing*; **2:** 18–24.

Nicholas M. (2004). Heart failure: pathophysiology, treatment and nursing care. *Nursing Standard*; **2:** 46–51.

Nursing and Midwifery Council. (2008). *The NMC Code of Professional Conduct: Standards for Conduct, Performance and Ethics*. London: NMC.

O'Regan H, Fawcett T. (2006). Learning to nurse: reflections on bathing a patient. *Nursing Standard*; **20:** 60–64.

Pang D, Keenan SP, Cook DJ, Sibbald WJ. (1998). The effect of positive pressure airway support on mortality and the need for intubation in cardiogenic pulmonary edema: a systematic review. *Chest*; **114:** 1185–1192.

Parsons C. (2000). Non-invasive positive pressure ventilation: averting intubation of the heart failure patient. *Dimensions of Critical Care Nursing;* **19:** 18–24.

Preston R. (2001). Introducing non-invasive positive pressure ventilation. *Nursing Standard*; **15:** 42–45.

Price A. (2004). Encouraging reflection and critical thinking in practice. *Nursing Standard*; **18:** 46–52.

Riley J. (2007). Developments in heart failure care. *British Journal of Cardiac Nursing*; **2:** 317.

Sawkins D. (2001). Non-invasive positive pressure ventilation. *Nursing Times*; **97:** 52–54.

Shamagian LG, Roman AV, Rames PM, Acuna JMG, Veloso PR, Gonalez-Juanatey JR. (2007). Acute pulmonary edema in patients with decompensated heart failure. Role of underlying cardiopathy on the prognosis. *International Journal of Cardiology*; **121:** 302–305.

Sice A. (2006). Intra-aortic balloon counterpulsation complicated by limb ischaemia: a reflective commentary. *Nursing in Critical Care*; **11:** 297–304.

Somauroo J. (2000). Effect of nasal bi-level positive airway pressure (BIPAP) and continuous positive airway pressure (CPAP) ventilation on cardiac haemodynamics in patients with congestive heart failure. *Heart*; **83** (Suppl.): A20.

Sorrentino MJ. (1997). Drug therapy for congestive heart failure; appropriate choices can prolong life. *Postgraduate Medicine*; **101:** 83–93.

Wang DJ, Gottlieb SS. (2008). Diuretics: still the mainstay of treatment. *Critical Care Medicine*; **36** (Suppl.): s89–s94.

Woodrow P. (2003a). Using non-invasive ventilation in acute wards: part 1. *Nursing Standard*; **18:** 39–44.

Woodrow P. (2003b). Using non-invasive ventilation in acute wards: part 2. *Nursing Standard*; **18:** 41–44.