Title: Physiological Effects of Positive Pressure Ventilation

Author: Dennis L. Oakes, Capt, USAF, NC

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ABSTRACT

Respiratory insufficiency is caused by numerous physiological aberrations, some of which can be treated by mechanical ventilatory support.. This paper presents the more common forms of mechanical ventilatory support with the adjuncts of PEEP and CPAP. Physiological effects of positive pressure ventilation are explained with emphasis on major organ systems. This paper concludes with a discussion of the clinical nurse specialist's role in providing care for the patient who is mechanically ventilated.

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Pressure Ventilation

Dennis L. Oakes, RN, BSN

Seminar Paper submitted to the Faculty of the

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Candidate: Dennis L. Oakes, RN
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Seminar Paper Approved: _____
                         Marguerite Littleton, RN, DNSc
                         Assistant Professor
                         Department of Psychophysiological
                         Nursing
                         Graduate Program
                         Trauma/Critical Care

Date Approved: _____

Seminar Paper Approved: _____
                         Dorrie Fontaine, RN, DNSc, CCRN
                         Assistant Professor
                         Department of Psychophysiological
                         Nursing
                         Graduate Program
                         Trauma/Critical Care

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Introduction

Mechanical ventilation is not a recent invention in medicine. True positive pressure ventilation in one form or another has been utilized since the beginning of the twentieth century (Mörch, 1985). The devices used were crude, but they provided the groundwork for the development of today's sophisticated ventilators.

Admission to a critical care unit is sometimes necessary because the patient requires mechanical ventilatory support. This ventilatory support usually is needed due to the inability of the individual to sufficiently ventilate the lung or oxygenate his/her tissues (Karpel & Aldrich, 1986). Respiratory insufficiency is caused by numerous physiological aberrations, some of which can be reversed or improved by mechanical respiratory support, thus allowing the patient to regain eventual control of his own ventilation and/or oxygenation.

The modes for ventilatory support are many. Often, decisions to use one over the other depend on the availability of the type of ventilator, the patient's condition, and the experience of the

clinician. Each method of ventilation interacts with the patient in some physiological manner. The physiological consequences, however may both be beneficial and detrimental for the patient.

Today's ventilators have improved efficiency over those in the past, primarily due to the use of advanced microelectronics (Mörch, 1985). Microelectronics also have given information on the physiological effects of positive pressure ventilation through invention of new monitoring tools. These physiological effects are at times detrimental to the critically ill patient. The advanced practicing nurse must recognize and understand the physiological effects of mecahnical ventilation to provide expert and quality care.

Chapter One

Controlled Mechanical Ventilation

Controlled mechanical ventilation (CMV) delivers a preselected ventilatory rate, tidal volume (V_T), and inspiratory flow rate independent of spontaneous effort on the part of the patient (Banner, Blanch, & Desutels, 1990). A major indication for CMV includes any condition in which apnea is present, such as injury to the central nervous system or spinal cord, drug overdose, or neuromuscular paralysis. CMV can be achieved by chemical neuromuscular blockade or delivering a ventilator respiratory rate that produces hypocarbnia (Spearman & Sanders, 1985).

Intermittent Mandatory Ventilation

Intermittent mandatory ventilation (IMV) was originally developed as a means to ventilate infants (Downs, 1973). It was thought that IMV more closely mimicked the natural cycle of ventilation. Today, IMV is the most widely used and effective ventilatory technique for patients with ventilatory failure (Venus, Smith, & Mathru, 1987).

Designed to deliver positive pressure, IMV

delivers gas at a fixed rate while allowing unrestricted spontaneous breathing to occur between mechanical cycles (Karpel & Aldrich, 1986). This permits the patient to augment and increase the minute ventilation supplied by the ventilator. This increasing reliance on spontaneous respiration is believed to more closely approximate the actual breathing pattern of the patient (Downs, 1973).

IMV is suggested as a means to maintain normal alveolar ventilation (Douglas & Downs, 1980). The ventilator rate can be adjusted to deliver just the mechanical augmentation needed to produce normal alveolar ventilation, with alveolar ventilation measured by arterial carbon dioxide partial pressure (PCO_2) and pH. Thus, respiratory acidemia and alkalemia can be minimized with the combination of spontaneous breaths taken by the patient and the ventilator delivered breaths (Douglas & Downs, 1980).

IMV is thought to be more beneficial than CMV on physiological dead space. Physiological dead space is the fraction of each inhaled tidal volume that does not participate in gas exchange. Downs and Mitchell (1976)

found that dead space ventilation increases as the rate of mechanical ventilation increases. When the patient was allowed to breathe spontaneously, as with IMV, physiological dead space returned to normal.

Synchronized Intermittent Mandatory Ventilation

Synchronized intermittent mandatory ventilation (SIMV) is similar to IMV in that it allows the patient to breathe spontaneously between delivered breaths. The difference is in the timing of the ventilator breath. SIMV was introduced because of the concern that during IMV, a mechanical breath might be superimposed on a spontaneous breath (Banner, et al., 1990). This "stacking" of mechanical on spontaneous breaths could increase the peak inflation, airway, and intrapleural pressures.

Studies have been conducted comparing cardiovascular effects of SIMV with IMV. Hasten, Downs, and Heenen (1980) found that cardiovascular effects were not significantly different, although peak inspiratory pressures were higher in IMV than SIMV. This is supported by another study in which cardiovascular effects, plural pressure, and

intrapulmonary shunting were comparable in the two
ventilatory methods (Heenan, Downs, & Douglas, 1980).
Again, peak inspiratory pressure and mean airway
pressures were higher in IMV than SIMV but appeared to
cause no significant ill effects.

Pressure Support Ventilation

Pressure support ventilation (PSV), a recent
innovation in mechanical ventilation, augments
spontaneous breaths with a set amount of inspiratory
pressure (MacIntyre & Leatherman, 1990). When the
patient initiates a breath, the ventilator delivers
flow proportional to that patient's inspiratory effort,
while maintaining a preset inspiratory pressure
(Ashworth, 1990). The major benefits of maintaining
constant pressure on inspiration are twofold.

The first benefit of PSV is to minimize the effort
of breathing. PSV changes the characteristic of the
work of breathing by reducing the ventilatory muscle
requirement (McIntyre, 1986). Once the breath is
initiated, the ventilatory pressure delivered controls
the inspiratory phase, relieving the inspiratory muscle
requirement. With PSV, respiratory frequency decreases

and tidal volumes increase resulting in a more stable minute ventilation (Ershowsky & Krieger, 1987). The outcome is a decrease in the oxygen requirement for the work of breathing (Tokioka, Sarto, & Kosaka, 1989; Kanak, Fahey, & Vanderwart, 1985). This aspect of PSV is important for the chronic lung patient and asthmatic who tires easily or the trauma patient in an oxygen debt state because the muscles of respiration are not consuming oxygen.

It is believed that PSV increases the patient's comfort of breathing and lowers the spontaneous ventilatory rate as compared to IMV (McIntyre, 1986). Research with mechanical models to simulate compromised pulmonary function found that the ventilatory frequency and tidal volume pattern with PSV minimize expenditure of respiratory energy (MacIntyre, 1986). As the level of inspiratory work is relieved, the optimal ventilatory pattern was one of slow deep breaths.

Overcoming airway resistance can be accomplished with PSV. Airway resistance may be increased due to pathologic processes as asthma or chronic bronchitis, which increase the work of breathing (Ashwoth, 1990).

Resistance also increases as endotracheal tube diameter decreases (Shapiro, Wilson, Casar, Bloom, & Teague, 1986). PSV has also been successfully used with low lung compliances where inflation resistance is increased (Banner, Kirby, & MacIntyre, 1991).

The amount of pressure required depends on the clinician's judgement of how much effort is beneficial and the comfort expressed by the patient. If resistance must be overcome, then small amount of pressure will generally suffice to provide adequate flow and pressure to overcome resistance (Ashworth, 1990). If the reduction of the work of breathing is the purpose, then higher pressures may be required to reduce the work of inspiration (MacIntyre, 1986). The greater the pressure given, the more the ventilator assumes the patient's inspiratory work.

Positive End Expiratory Pressure

Positive end-expiratory pressure (PEEP) is not a mode of ventilatory support, but actually an adjunct employed to achieve greater residual lung volume (Spearman, 1988). The result is an increase in the functional residual capacity (FRC) through recruitment

of greater numbers of alveoli. PEEP is similar to continuous positive pressure ventilation (CPAP) except that PEEP pressure is maintained at end-expiration only and CPAP is maintained throughout the ventilatory cycle. Positive pressure ventilation often produces lung areas with a mismatch in ventilation to perfusion ratios (V_A/Q). PEEP is employed in an attempt to improve alveolar ventilation to lung areas where ventilation is less than perfusion (low V_A/Q) or to areas with complete alveolar collapse. Increasing the fractional inspired oxygen (FiO2) may improve hypoxia, but does not correct the pathophysiologic effects leading to poor V_A/Q matching (Norwood, 1990). By increasing alveolar ventilation, PEEP is thought to lower oxygen requirements, thereby reducing the risk associated with oxygen toxicity (Downs & Douglas, 1980).

Continuous Positive Airway Pressure

Continuous positive airway pressure can be defined as positive airway pressure maintained throughout the entire respiratory cycle (Ayers, Schlichtig, & Sterling, 1988). Often, CPAP is employed for the same

reasons as PEEP. The goal of therapy is the recruitment of alveoli for gas exchange.

SUMMARY

The clinician has numerous modes of mechanical ventilation to choose based upon the patient's physiological status and illness. CMV is indicated in any condition where apnea is present or total control of ventilation is warranted. IMV and SIMV are choices for the spontaneous breathing patient who can assist in the ventilation process. The use of IMV is purported to a decrease the amount of physiological dead space when compared to CMV. Pressure support ventilation is gaining acceptance and is an excellent choice for the patient who requires a reduction in the work of breathing.

Adjuncts employed with mechanical ventilation are PEEP and CPAP. PEEP is effective at end-expiration where CPAP maintains pressure throughout the entire ventilatory cycle. Both adjuncts serve to increase FRC and offset the effect of V_A/Q mismatch.

Chapter Two

Pulmonary

To appreciate the effects of positive pressure
ventilation, one must understand the physiology of
spontaneous respirations. Inspiration is accomplished
through the creation of a pressure differential between
the upper airway and the alveoli (Douglas & Downs,
1979). The difference in pressure is created by
diaphragmic contraction and chest wall expansion
causing movement of air. Artificial ventilation
creates this pressure difference by raising the airway
pressure above the pleural pressure (Ppl). The two
methods work on the same principle that movement of air
occurs from an area of high pressure to one of low
pressure (Douglas & Downs, 1980).

This difference in Paw and Ppl can be expressed as
a function of work. Work of breathing (WOB) may
increase for two reasons (Douglas & Downs, 1980). The
first is the resistance of the airway system to
deformation or movement, which is the compliance of the
lungs and thorax. The second is the resistance to the
flow of gases through the airway. The greater the

resistance to ventilation, the greater the work required to create a pressure differential between the Paw and Ppl.

The use of positive pressure ventilation can increase or decrease the WOB. Increases in WOB may be attributed to the ventilator circuit (Marini, 1987). Circuit resistance increases as the endotracheal tube (ETT) diameter decreases (Shapiro, Wilson, Casar, Bloom & Teague, 1986). Also, resistance increases proportionally with the endotracheal tube length (Norwood, 1990). Secretions, bronchoconstriction, and tube patency will increase the WOB (Norton & Neureuter, 1990). A 50 percent reduction in the radius will increase resistance 16-fold (Norwood, 1990). Finally, decreases in lung or thorax compliance will increase the WOB (Douglas & Downs, 1980).

Decreases in the WOB is attributed to the mode of ventilation used. Highest during spontaneous ventilation, WOB is somewhat lower in assisted ventilation as IMV or SIMV, and is the lowest in CMV (Snyder, Carroll, Schuster, Culpepper, & Klain, 1984). The greatest fear is that prolonged ventilator support

leads to respiratory muscle detraining, weakness, fatigue, and even atrophy (Shikora, Bistrian, Burlase, Blackburn, Stone, & Benotti, 1990).

As the proportion of ventilatory control is transferred from the patient to the ventilator, changes appear in the distribution of air flow in the lungs. At one extreme is the paralyzed patient in which CMV is employed. FRC decreases and dead space ventilation (V_D) to tidal volume (V_T) ratios increase (Norwood, 1990). With increased V_D, total ventilation must increase in order to maintain oxygenation, often resulting in delivered V_T of 12-15 ml/Kg (Snyder et al., 1984). The need for increased V_T can be further explained by examining airflow to dependent and non-dependent airway structures.

When the diaphragm is paralyzed or movement is inhibited by abdominal contents of the supine patient, the dependent lung portion moves little (Froese & Bryan, 1974). Movement is greatest in the nondependent lung region, thus receiving the greatest amount of delivered tidal volume. The ventilator creates an increased V_A/Q ratio in the nondependent areas and a

decreased V_A/Q ratio in the dependent areas (Banner et al., 1990). Since V_D increases with mechanical ventilation, larger V_T is required to maintain efficient alveolar ventilation (Snyder et al., 1984).

The maldistribution of delivered V_T causes normal alveoli to become overdistended. The overdistention can damage the alveoli unit and increase the risk for pulmonary edema (Snyder & Froese, 1987). It has been demonstrated that high volume/low pressure ventilation contributes to the development of pulmonary edema more than low volume/high pressure ventilation in animal models (Dreyfuss, Solar, Bassett, & Saumon, 1988). The authors speculated that high volumes attributed to greater leakage of fluid from the interstitial space by impairing epithelial tightness (Dreyfuss et al., 1988).

The efficiency of alveolar ventilation refers to the effectiveness of blood oxygenation and the elimination of carbon dioxide (CO_2). Venous admixture, or intrapulmanary shunt, determines oxygenation efficiency (Snyder et al., 1984). Venous admixture is the amount of deoxygenated venous blood not exposed to alveolar gas and occurs when blood circulates through

capillaries adjacent to airless alveoli (Von Rueden, 1989). The efficiency of CO_2 elimination is represented in the V_D/V_T ratio (Snyder et al., 1984). As this ratio increases, FRC decreases compromising CO_2 exchange, hypoventilation then ensues causing hypercarbia and a decrease in arterial oxygenation (Von Rueden, 1989).

To compensate for the above effects, restoration of FRC is thought to be beneficial. The restoration intervention does not try to normalize the FRC, but to increase it by recruiting lung alveoli who have a decreased V_A/Q (Snyder et al., 1984). The treatment aimed at restoration of FRC consist of the application of CPAP or PEEP.

The effect of PEEP on FRC has been extensively studied. Researchers believe that with PEEP, an increase in FRC will inevitably occur because the lung is not allowed to return to its normal resting end-expiratory position (Berend, Christopher, & Voelkel, 1982). The added pressure maintains open alveoli and improves the V_A/Q ratio and oxygenation due to increased alveoli recruitment.

Clinicians have documented a phenomena where PEEP occurs in ventilatory patients without producing it mechanically. This form of intrinsic PEEP represents auto-PEEP. This is defined as the spontaneous development of PEEP at the alveolar level due to insufficient expiratory time (Benson & Pierson, 1988). Auto-PEEP occurs in patients with airflow obstruction. The patient's airway pressure does not return to zero before the next ventilator breath is given. Auto-PEEP may have the same, if not more, deleterious hemodynamic effects as PEEP (Pepe & Marini, 1982).

Researchers have suggested another variable working with PEEP to increase FRC. Berend and colleagues (1982) examined the effects of prostacyclin production in 12 beagle dogs. The researchers wanted to determine if prostaglandins, particularly prostacyclin I2 (PGI2), played a role in determining FRC during PEEP ventilation. FRC was measured before, during, and after PEEP was employed. Then, indomethacin, a known prostaglandin inhibitor, was administered and the measurement of FRC was repeated.

Results suggested that FRC was significantly

reduced after the administration of indomethacin, even when PEEP was used. The authors concluded that endogenous PGI2 may be partially responsible for the elevation in FRC with PEEP (Berend et al., 1982). Later research has shown that prostaglandins, particularly PGI2 have both vasodilator and bronchodilator properties. Dilation of the alveolar ducts and respiratory bronchioles can increase lung volumes leading to increases in FRC (Parker, Hernandez, Lonenecker, Peevy, & Johnson, 1990).

Lindner, Lotz, and Ahnefield (1987) studied the effect of CPAP on FRC in 34 adults who underwent major abdominal surgery. The authors regarded the FRC as a measure of the alveolar volume available for pulmonary gas exchange. CPAP was administered to 17 patients daily for five days and their lung volume parameters were compared to the control group of 17 patients who received no CPAP. There was a significant increase in the FRC in the experimental group receiving CPAP compared to the control group on each day of the study. The researchers concluded that CPAP proved effective in increasing FRC and should be employed soon after

surgery to improve pulmanary gas exchange.

Cardiovascular

Preload, afterload, and contractility influence cardiac output. Preload is determined by the end diastolic volume, which is proportional to the end diastolic filling pressures (Bore, Gravenstein, & Kirby, 1990). Afterload is the impedance to ejection of blood from the ventricle. Contractility is the intrinsic muscle power that allows the heart to contract at a constant preload and afterload (Von Rueden, 1989). Hemodynamic consequences of mechanical ventilation result from Paw transmitted to the pleural space, the heart, and great vessels within the thorax (Florete & Gammage, 1990).

Transmission of Paw to the pleural space is thought to depend on lung and thorax compliance (Chapin, Downs, Douglas, Murphy, & Ruiz, 1979). In patients with lower thorax compliance and normal lung compliance, greater airway pressure was transmitted. The transmission of positive Paw to the intrapleural space may be of such degree that cardiac output is compromised. Whereas, when thoracic compliance was

increased and lung compliance was decreased, less transmission of pressure to the intrapleural space was observed (Chapin et al., 1979).

Recent studies have questioned the protective effects of low compliant lungs on cardiovascular functioning. Venus, Cohen and Smith (1988) examined the effect of CMV with PEEP in five swine with normal lung compliance and seven swine with decreased lung compliance. They concluded that transmission of Paw to the intrapleural space was less in the low compliant lung group than the normal compliant group. However, the end-expiration tracheal pressure was significantly higher in the low compliant group. Cardiac output (CO) was depressed equally in both groups despite mean Paw reduction in the low compliant group. The authors concluded low compliant lungs may not offer as much protection against the effects of positive Paw as one thought (Venus et al., 1988).

Transmission of positive Paw has many effects on the right and left heart. Pressure transmission decreases the pressure gradient in the right heart causing a decrease in venous return (Abel, Salerno,

Panos, Greyson, Rice, Teoh, Lichten, 1987). The greater the pressure transmission, the greater the effect.

Douglas and Downs (1980) illustrate how increased pleural pressure can decrease venous return. If in the spontaneous breathing individual, the right atrial pressure (RAP) is 5 mmHg and -2 mmHg is generated by the muscles of inspiration, the transmural filling pressure is 5 - (-2) for a total of 7 mmHg. In the mechanically ventilated patient, the pleural pressure is now positive. The resulting filling pressure is now decreased from 7 mmHg to 5 - (+3) or 2 mmHg, thus a gradient for venous return has been established.

The most important hemodynamic effect that occurs is that positive pressure ventilation will decrease CO by decreasing the pressure gradient for venous return, for venous return is the major component for determining CO (Guyton, 1986). The decrease in CO can be compensated for by intravenous fluid administration, since the primary cause of the decrease in CO is a decreased pressure gradient for venous return (DeMaria, Burchard, Carlson, & Gann, 1990).

Myocardial blood flow (MBF) decreases in the presence of increased intrapleural pressures. This results from the transmission of pressure to the pericardium and in turn to the coronary artery, especially when high levels of PEEP are employed (Fessler, Brower, Wise, & Permutt, 1990; Ben-Haim, Amar, Shotty, & Dinnar, 1991). Concurrent with decreased MBF is the reduction of myocardial oxygen consumption (Ben-Haim et al., 1991; Hevrøy, Grundnes, Bjertanaes, & Mjøs, 1989). This decrease in myocardial oxygen consumption is not clearly understood. It is suggested that the determinants of left ventricular tension, pressure and volume, are markedly reduced, thereby, reducing left ventricular workload demand (Ben-Haim et al., 1991). Another possible explanation may be a result of the heart's intrinsic ability to regulate blood flow in relation to oxygen requirements (Guyton, 1986b).

The third cardiovascular variable affected by positive pressure ventilation is afterload. Positive pressure has been found to decrease afterload through transmission of pleural pressure to the great vessels,

including the aorta (Fessler, Brower, Wise, & Permutt,
1988; Mathru, Rao, El-Etr, & Pifarre, 1982). The
pressure transmitted to the aorta displaces a greater
proportion of blood and lowers the mean aortic
pressure. This lower pressure seems to be beneficial.
In fact, it is, particularly when patients with altered
left ventricular function are mechanically ventilated
(Mathru et al., 1982). CO is actually increased in
these patients. However, once dependence on artificial
ventilation is decreased, instability in maintaining
cardiac output and oxygen delivery are compromised
(Fessler et al. 1988; Mathru et al., 1982).

Renal/Fluid Balance

Critical care patients often exhibit changes in
fluid and electrolyte balances. Illness and injury
with ventilatory support may aggravate an already
compromised system, such as pulmonary edema, which may
impair gas exchange (Knebel, 1991). The reasons for
water and electrolyte imbalances are many.

Increased Ppl pressure caused by positive pressure
decreases venous return and reduces CO (DeMaria,
Burchard, Carlson, & Gann, 1990). This decrease in

output may activate the sympathetic nervous system (Kennedy, Weintraub, & Skillman, 1977). Catecholamines constrict the afferent renal arterioles, thereby reducing glomerular filtration rate. The decreased renal blood flow, with increased sympathetic activity, stimulate the renin-angiotensin system. This activation decreases blood flow through further constriction.

Another mechanism urinary output decreases is through the left atrial stretch receptor activation due to decreased thoracic blood volume (Florete & Gammage, 1990). The stretch receptors release plasma arginine vasopressin (AVP), which acts as a vasoconstrictor and antidiuretic. This agent, associated with positive presssure ventilation, promotes distal tubular reabsorption of water and decreases free water clearance (Hammer, Viquerat, Suter, & Vallotton, 1980).

Increased water and sodium retention is thought to be a product of atrial natriuretic factor (ANF) (Andrivet, Adnot, Brun-Buisson, Chabrier, Braquet, & Lemaire, 1988). ANF, a peptide synthesized by granules and located in atrial cardiocytes, promotes diuresis

and naturesis through hormonal and renal mechanisms in response to atrial distention or elevation in atrial pressure (Norwood, 1990). It has been suggested that it exerts inhibitory effects on renin and aldosterone secretion (Laragh, 1985). The exact relationship ANF and positive pressure ventilation hold has not been established.

Andrivet et al. (1988) examined circulating plasma levels of ANF during mechanical ventilation using zero PEEP. The patients studied were in lower body pneumatic trousers and mechanical ventilated with and without 12 cm H_2O. PEEP ventilation resulted in a significant reduction in diuresis and natruresis that was proportional to decrease in plasma ANF levels and right atrial pressure. When the lower body pneumatic trousers were inflated without fluid volume loading during PEEP, diuresis and plasma ANF levels were restored to near zero PEEP values. The authors concluded that intrathoracic pressure may contribute to alterations in renal excretory function during PEEP (Andrivet et al., 1988). This suggests that the use of PEEP could reduce atrial stretch and reduce ANF

secretion, thereby inhibiting diuresis and naturesis.

Other investigators attempted to clarify the effect of ANF on sodium and water excretion when PEEP was used (Andrivet, et al., 1991). Exogenous ANF was injected during zero PEEP and with the application of 10cm H20 of PEEP in eight patients. When PEEP was used, urine sodium and water excretion were significantly less and plasma renin levels were more when compared with control values obtained with zero PEEP alone. When ANF was injected during zero PEEP, urine sodium and water excretion markedly increased. Then, ANF was injected during PEEP, urine sodium and water excretion was noted to increase while serum levels of renin decreased, but not as significantly as with zero PEEP. It was suggested that possibly the renin-angiotensin-aldosterone and sympathetic nerve activity may be an important variable role in explaining the renal response to PEEP since urine sodium and water excretion during peep may occur independently of any reduction in ANF levels. (Andrivet et al., 1991)

Other investigations into the role of ANF can be

found. One group of researchers reported a negative correlation between the CVP and circulating ANF (Schütten, et al., 1990). As the CVP increased in the healthy subjects, the circulating volume of ANF decreased instead of increasing as reported previously. Still, there are studies that support no correlation between ANF, CVP, and the use of PEEP up to 15cm H2O (Teba, Dedhia, Schiebel, Blehschmidt, & Linder, 1990).

Infection

Nosocomial infections of the respiratory tract are a frequent occurance in the mechanically ventilated. Several factors contribute to the susceptibility of the critically ill patient. Major sources of organisms include oropharynx contamination with hospital organisms, inhalation of organisms from contaminated equipment, direct introduction into the ETT or tracheostomy, and aspiration of gastric contents (Florete & Gammage, 1990). Immobility due to injury or illness promotes stasis of secretions and provides a nutrient rich medium for bacterial growth (Thompson, 1990). Medications, poor fluid intake, and decreased cilial action also contribute to secretion stasis.

The development of pneumonia is correlated with the length of mechanical ventilation. One study reported that of 724 ventilated patients, 5.5% of the patients ventilated less than 24 hours developed pneumonia, while 26.6% of the patients ventilated for greater than 24 hours developed pneumonia (Langer, Mosconi, Cigada, & Mancelli, 1989). Others have reported similar rates of 25.7% and 24% for patients ventilated for greater than 24 hours (Rello, et al., 1991; Torres, et al., 1990).

Langer and his associates reported that pneumonia occurred most frequently the first eight to ten days of mechanical ventilation, with gram-negative bacteria the major organism found (Langer et al., 1989). Gram-negative bacteria proved to be the major pathogen in other studies as well (Rello et al., 1991; Torres et al., 1990).

Numerous mechanisms for the colonization of the respiratory tract have been suggested. When several variables are considered, reintubation was seen as the greatest contributor to pneumonia followed by gastric aspiration (Torres et al., 1990). Reintubation was

implicated for introducing bacteria to the respiratory membranes and gastric aspirate implicated particularly when the pH is high.

Torres et al. (1990) reported twice the rate of pneumonia in patients with high gastric pH than those with low gastric pH. Often, antacids and histamine 2 (H2) antagonists are given to raise gastric pH stem the development of stress ulcers and gastric bleeding. This decreased acidity of the stomach creates an environment favorable to colonization by gram-negative organisms (Donowitz, Page, Mileur, & Geuthner, 1986).

The frequency of enteral feedings has been suggested as a risk for pneumonia development in ventilated patients. Twenty-four patients, 12 in each group, underwent either continuous enteral feeding or intermittent feeding. The authors failed to describe the time interval for the intermittent feedings. The continuously fed group had higher 7 AM gastric pH and all but one of the twelve developed pneumonia. Whereas, the pH of the intermittently fed group was lower and had only one incidence of pneumonia. This suggest that intermittent enteral feedings resulted in

higher gastric pH and leads to lower infection rates.

Central Nervous System

Pressures generated by positive pressure ventilation and its resulting increase in intrathoracic pressure, lung distention, and increased RAP with decreased venous return from the head contribute to increased intracranial pressures (ICP) (Epstein, Ward, & Becker, 1987). In patients with a head injury, the use of high tidal volumes or the use of PEEP may further exacerbate the rise in ICP.

Cerebral blood flow and its relation to ICP is regulated by a "Starling Resistor" between the sagittal sinus and the cerebral veins (Luce, Husby, Kirk, & Butler, 1982). As intrathoracic pressure increases, venous return decreases. There develops an increased pressure in the superior vena cava and sagittal venous pressure up to a "waterfall pressure" without adversely increasing ICP. Research conducted in dogs has shown that once this "waterfall pressure" is reached, there develops a direct relationship between the superior vena cava, sagittal sinus, and CVP pressures (Luce et al., 1982). Thus, increases in CVP will be reflected

in increases in ICP.

Furthermore, positive pressure ventilation affects cerebral blood flow and ICP by altering $PaCO_2$ levels. Cerebrovasodilation is associated with hypercarbnia (Gildengerg & Frost, 1985). Consequently, this vasodilation increases ICP by increasing cerebral blood volume. The other extreme, hypocarbnia, also affects cerebral blood flow and oxygen extraction. Hypocarbnia produces vasoconstriction decreasing cerebral blood volume. Hypocarbnia also induces a shift of the oxygen dissociation curve to the left, making less oxygen available to the tissues. This reportedly has been implicated in lowering the seizure threshold (Gildenberg & Frost, 1985).

Mechanical ventilated patients undergo many manipulations of the ETT to maintain pulmonary hygiene. Suctioning and its preparatory maneuvers increases ICP (Tsementzis, Harris, & Loisou, 1982; Parsons & Shogan, 1984). Manual hyperinflation has been shown to increase ICP in head injured patients, probably as a result in increasing lung volumes, but is not near as significant as with suctioning (Parsons & Shogan,

1982). Suctioning stimulates the cough reflex and reflex vagal inhibition, which may cause straining maneuvers causing an increase in both the intrathoracic and intrabdominal pressures (Tsementzis et al., 1982).

Barotrauma

Any intervention that the medical team employs to support bodily functions of a critically ill patient has risk. Complications of mechanical ventilation manifest themselves in varying ways. One of the most common is barotrauma.

Pulmonary barotrauma involves a pressure injury to the lungs resulting in extra-alveolar air. This occurs when alveoli are subjected to high ventilatory pressures causing alveolar overdistention. Gas escapes from in the alveoli base and transverses along the perivascular sheath to the helium (Powner, 1988). The migratory process of the gas may force it into several anatomic areas, thus producing the sequelae listed in table 1.

Table 1.

<u>Consequences of Extra-Alveolar Gas</u>

Interstitial emphysema

Lung cyst

Pneumomediastinum

Subcutaneous emphysema

Pneumopericardium

Pneumothorax

Pneumoretroperitoneum

Pneumoperitoneum

Bronco-pleural fistula

Air entry into the vascular space

<u>Note</u>. From "Lung Barotrauma in the Icu" by D. J.

Powner, 1988, <u>Indiana Medicine</u>, <u>81</u>, p. 614.

High peak inspiratory pressure (PIP) is most

commonly cited as the main risk for barotrauma (Kaake,

Shlichtig, Ulstad, & Henschen, 1987). Peterson and

Baier (1987) reported a 43 percent incidence of

barotrauma in patients with PIP greater than 70 cm H_2O,

whereas patients ventilated with PIP between 50-70 cm

H_2O was eight percent. Another researcher reported

barotrauma in 12 of 13 patients who had PIP above 40 cm H_2O (Woodring, 1985). No exact PIP can guarantee absence of barotrauma, but it is reasonable to focus on minimizing PIP as much as possible (Haake et al., 1987).

If reduced PIP is associated with a decreased incidence in barotrauma, then the mode of ventilation may play an important determinate. Mathru and Venus (1983) examined the incidence of barotrauma in patients ventilated with IMV with CPAP and CMV. The IMV-CPAP group and an incidence rate of seven percent, whereas the CMV group was 22 percent. They suggested that the employment of IMV-CPAP reduced the development of barotrauma and should be considered for use instead of CMV (Mathru & Venus, 1983). McIntyre (1986) demonstrated that PSV produces lower PIP than SIMV. PSV may reduce the incidence of barotrauma, but no research comparing barotrauma incidence with another ventilatory mode could be located.

Nutrition

Caloric intake of the ventilated critically ill patient may be variable owing to the condition of the

patient, the number and frequency of surgical procedures, and other complications that would prohibit enteral intake (Brown & Heizer, 1984). The ventilated patient often cannot express hunger due to the ETT, fatigue, or level of consciousness. These contribute to inadequate nutritional intake.

Nutrition affects the respiratory system in different ways. The first impact is upon the muscles of respiration. Poor nutritional intake may impair the supply of adequate amounts of energy substrate required for metabolism (Brown & Heizer, 1984). Muscle wasting may occur, particularly when protein intake is insufficient. Driver and LeBrun (1980) retrospectively reviewed the records of 26 medical patients who required ventilatory support for at least six days. Protein intake was found inadequate in 21 of the 26 patients. The authors concluded that adequate intake was often neglected in patients requiring ventilatory support.

Furthermore, malnutrition has been suspect in decreased surfactant production. Sahebjami, Vassalo, and Wirman (1978) showed that nutritional deprivation

in rats caused high alveolar surface tension and decreased lung elasticity. This was attributed to decreasing stores of surfactant. The state of decreased surfactant increases surface tension and could cause alveolar collapse.

Ventilatory drive is yet another component where nutrition interfaces. Hypermetabolism is common in illness and is associated with increases in oxygen consumption (Elwyn, Kinney, & Arkanazi, 1981). To understand the role of oxygen consumption and its byproduct, CO_2, one must understand the meaning of respiratory quotient (RQ). The RQ is calculated as the CO_2 production divided by the oxygen consumption (Barrett, Robin, Armstrong, & Bone, 1987). Each substrate is associated with its own RQ: Carbohydrate, 1.00; fat, 0.70; protein 0.82; and lipogenesis or the synthesis of fat from glucose, 8.0. Therefore, an RQ greater than one implies a shift towards lipogenesis, or synthesis of fat from glucose. This occurs during states of carbohydrate excess and causes an increase in carbon dioxide production, therefore affecting the patient's ventilatory drive (Brown & Heizer, 1984).

Researchers have reported that carbon dioxide levels decrease in ventilated patients receiving total parenteral nutrition (TPN) when the portion of non-protein calories in TPN fluid was changed from glucose to fat (Askanezi, et al., 1981). CO_2 production decreased eighteen percent in eight patients who were changed from 100% glucose to a combination of 67% glucose and 33% fat. CO2 decreased 24% in six patients changed from 100% glucose to a combination of 50% glucose and 50% fat. Concurrent with CO_2 decreases, minute ventilation was noted to decrease.

This same group of researchers also examined the effect of switching from 50% fat to 100% glucose in 12 injured patients (Nordenström, et al., 1982). Again, increases in CO_2 and minute ventilation were noted in the 100% glucose group. The authors concluded that moderate quantities of fat emulsion with TPN in place of glucose substantially reduced CO_2 production.

Hepatic

Positive pressure ventilation increases RAP. Consequently, it effects the pressure gradient of blood returning from the venous system. One study examined

how the interaction of positive pressure ventilation with PEEP impacts hepatic blood flow (HBF) in humans (Bonnet, Richard, Glaser, Lafay, & Guesde, 1982). HBF was found to correlate negatively with increases in PEEP. HBF decreases were not statistically different in the group exposed zero PEEP and a control group of normal subjects, but HBF did decrease approximately 32% in the experimental group with PEEP of 20 cm H_2O compared to the control group. The authors speculated that decreases in HBF was due to decreased CO, increased hepatic resistance due to liver compression by the diaphragm, and increased pressure between the portal vein and the inferior vena cava. These physiological variables were contibuted to increased Ppl. These results are supported by another study that found HBF to decrease as Paw increased when CPAP was used (Perkins, Dastra, DeHaven, Halpern, & Downs, 1989).

Summary

The physiological effects of positive pressure ventilation manifest themselves in many organ systems. Artificial ventilation creates a pressure difference

between the upper airway and the pleural space creating a change in the WOB. The physiological effects that result are from transmission of generated ventilator pressure in the airway to that of the great vessels and structures within the thorax.

Changes are seen in airflow patterns when positive pressure is employed. There develops a mismatch in the V_A/Q and a decrease in FRC. CPAP and PEEP are useful adjuncts to correct these problems, but these interventions increase the chance for barotrauma and attenuate the ill effects on the cardiovascular status.

The greatest effect positive pressure ventilation has on the cardiovascular system is in decreasing venous return, which in turn decreases CO. The reduced venous return and CO sets into motion compensatory mechanisms. These include the retention of water and sodium through hormonal mediators as AVP and ANF. Another mechanism includes the stimulation of the sympathetic nervous system in an attempt to increase CO and conserve renal perfusion through vasoconstriction.

Infections are common in the ventilated patient and increase proportionally with the length of time

mechanical ventilation is employed. Major infection sources are contributed to repeated breaks in the ventilator circuit and aspiration of gastric contents. There appears to be a positive correlation with enteral feeding frequency and the use of antacids and H2 blockers.

Blood flow to the brain and liver are impaired or decreased, owing to a decrease in venous return. Decreases in veous return are reflected in elevations in the ICP and decreased HBF. Perfusion of these two organs is of concern if impairment exists concomitant with the requirement for artificial ventilation.

Finally, nutritional status and the choice of nutritional substrates effect metabolism and its byproducts. Malnutrition is a common problem. Carbohydrates, when given as the sole substrate in TPN, increase oxygen demand and increase CO_2 production. Nutritional support in the form of carbohydrates with lipid solutions is recommended.

Chapter 3

The clinical nurse specialist (CNS) occupies an essential position in the care of artificially ventilated patients. The literature delineates the role of the CNS into five subroles. The subroles include advanced practitioner, researcher, consultant, educator, and manager. What follows is a description of the subroles and their relationship with the care of patients who are artificially ventilated.

Advanced Practitioner

The subrole of advanced practitioner lies in the perceived and expected competence of a specialized body of knowledge. Koetters (1989) viewed the foremost responsibility of the CNS as one of an expert practitioner who focuses on patient care. Therefore, the CNS possesses the knowledge and ability to anticipate potential health problems in a given patient population. This role requires the CNS to use complex intellectual processes of decision-making to improve the care and achieve positive patient outcomes.

The expert practice role involves both direct and indirect patient care activities. Providing direct

care places the CNS at the bedside providing care to the ventilated patient. The CNS utilizes skilled assessment techniques and anticipates physiological changes resulting from the use of artificial ventilation. Maintaining and updating knowledge of recent technological advances, such as positive pressure ventilation, can only occur by actively caring for patients. Putting into practice what has been read confirms and increases understanding allowing the CNS greater opportunities to impact patient outcomes and function as a role model for other nursing staff.

The indirect advanced practice role offers the CNS the chance to formulate expanded nursing diagnoses. The skilled CNS assesses, questions, and understands the physiology and pathophysiology of respiratory failure. Realizing the potential effects of positive pressure ventilation, the CNS can anticipate potential nutritional, cardiovascular, or neurological problems where nursing care can make a difference. This area is especially important for the CNS who makes rounds on artificially ventilated unit patients. The CNS, together with the staff nurse, utilize critical

thinking to formulate personalized care plans.

The advanced practice role is not limited to patient care. The CNS realizes the importance of the family and strives to incorporate them into the total picture. Knowledge of stress and its resulting disequilibrium requires skillful use of crisis intervention techniques when respiratory failure suddenly occurs in a family member. Assessment of family and support system functioning patterns allows for education and incorporation of care plans that include an often overlooked patient care component.

Researcher

The role of research is well suited for the CNS who participates in the care of ventilated patients. Involvement in the research process depends greatly on the position held within the organization and personal interest. Educational preparation allows the CNS the opportunity to participate in the research process in a number of ways, from interpreting research findings to actually conducting research independently.

McQuire and Harwood (1989) suggest CNS involvement with research be conducted within three levels. The

first level requires the CNS to identify nursing practice problems relevant to patient care. Responsibilities include continually examining the practice environment for research ideas, exploring the literature for recent topics, and disseminating research results to staff members and other health professionals. Issues the CNS may want explore are the effects of positioning on oxygenation, cardiovascular effects of suctioning, and ways to increase the comfort in artificially ventilated patients.

The second level of research necessitates the CNS to become actively involved in the research process. Collaboration with other health care professionals, be they fellow staff nurses or physicians, encourages personal and professional growth. With a focus on patient outcomes, the CNS can conduct research into the timing of enteral feedings and its relationship with the development pneumonia in the ventilated patient. The CNS evaluates research for the purpose of integrating the results into present practice models, as in the recommendation limiting the number of suction passes to two per procedure in adult head injury

patients (Rudy, Turner, Baun, Stone, & Brucia, 1991).
Also, replication of research assists in establishing
validity and reliability to others' findings.

As the CNS becomes more skilled in the research
endeavors of the previous two levels, independent
research may be performed. This constitutes the third
level of research involvement. Again, the CNS may
conduct research as an individual or it may entail
collaboration with other nurses. The increase in
patient acuity, particularly the increase in the
elderly population, mandates changes in ventilator care
and use. The CNS is the perfect individual to
determine research topics exploring the relationships
between the physiological effects of aging with the
physiological effects of positive pressure ventilation.

Research into the effects nursing procedures have
with newer forms of ventilatory support add to the ever
expanding body of knowledge. A research study could be
performed examining when, in the ventilation cycle of
PSV, should the injectate for CO be administered to
obtain accurate results. Additionally, research
examining the role of active upper and lower body

exercise and their effect on lung volumes may be
independently performed.

Consultant

The care of ventilated patients can be extremely
challenging for critical care nurses. Sometimes, the
nursing staff is faced with complex patient care
scenarios that push patient care abilities to the
limit. The CNS is cognizant of the needs of the
clinical setting, these include staff as well as
patients and families. Such cases may include the
ventilated chronic obstructive pulmonary disease
patient who cannot be weaned from ventilatory support.
It is times such as these that the CNS is called upon
to function in a consultative manner.

Caring for a ventilated patient requires the
interaction of many disciplines. The CNS understands
the interactions among departments and institutes means
to assist collaborative practice. Prescribing
interventions must be avoided. The purpose of the
consultation process is not to give answers, but to
improve problem solving skills, as in the cardiac
patient who experiences decreased CO when placed on

ventilatory support. By improving problem solving
skills, the next time the same problem is faced, the
health care staff will be better prepared to handle the
situation.

The CNS expertly utilizes and understands the
nursing process. Clarifying the nature of the problem,
as in a patient who fights a ventilator, and the
variables contributing to it, constitutes the initial
step. Objectivity cannot be overemphasized in this
role, for blaming others for the patient problem
commonly occurs. Assessment is accomplished by the CNS
alone or in collaboration with other health care
members.

The CNS stimulates the formulation of
interventions. One method available to the CNS is to
hold a patient care conference. During the conference,
the sharing of information provides staff the
opportunity to problem solve for themselves.
Interventions are the result of collective thought, for
the goal of consultation process is to increase problem
solving skills.

Furthermore, the CNS may be asked to assist in the

development of policies and procedures pertaining to
the removal of ventilatory support. The recent passage
of federal and state laws prescribing increased patient
rights impact the delivery of care. The CNS assists in
the identification of resources and gives insight into
the implications these laws have in policy development
as they relate to the artificially ventilated patient.

Educator

The role of educator covers a multitude of arenas.
Not only is the CNS charged with education of staff,
but the education role should encompass the patient and
family. The CNS also plays an important role in the
formal and informal education of other health
professionals, such as respiratory therapy,
nutritionist, medical staff, and social workers.

Education encompasses several levels, the first of
which is the intensive care unit (ICU) staff. The
enthusiasm of staff, particularly those inexperienced,
are an asset the CNS can build upon. As an educator,
the CNS is knowledgeable of adult learning theories and
adjust the educational experience to the appropriate
level of the participants. This can be accomplished

through patient care conferences for those nurses new
to the ICU. Patient care conferences offer the
opportunity for nurses to correlate classroom theories
of pulmonary physiology and pathophysiology with
clinical practice. The CNS then teaches, coaches, and
supports appropriate problem solving abilities in
nursing staff members.

The CNS is important for the education of family
members. A patient may require home ventilation, thus
the family members must be taught the required care.
The CNS possesses skills that allow for proper
assessment of family functioning and tailors the
educational experience to the family's needs.
Instruction would include ventilator operation,
respiratory care, and methods to reduce the development
of infection. The CNS then works with the social
worker to coordinate in-home care based upon the
knowledge level of family members.

Education of other health professionals is another
expected responsibility of the CNS. The care of the
artificially ventilated patient is seen through a
holistic approach, with interventions affecting the

patient's and family's psychosocial functioning. The CNS expands the knowledge of other health professionals to the psychological, social, and ethical components of care. Thus, education strives to effect a positive patient outcome by assuming the role of patient and family advocate.

Manager

The CNS who occupies a line position impacts all areas of patient care. For the ventilated patient, the CNS has the responsibility in making unit policies and procedures, these may include matching ventilator modes with disease processes, management of barotrauma, or the use of sedatives. Policies are written to effectively deliver patient care and are based upon sound principles derived from research. The CNS also is charged with monitoring patient outcomes based upon the nursing care delivered.

One of the goals of the CNS manager is to create an environment conducive to professional nursing practice. The CNS is a prime candidate, as he/she possesses the characteristics of a knowledgeable practitioner, skilled communicator, and expert problem

solver. The combination of each of these attributes gives the CNS strengths that other managers may not possess. The CNS not only manages the unit, the CNS leads through demonstrating expert patient care, with incorporation of current researcher recommendations.

The CNS is a catalyst for change. Acquiring knowledge about newer generation ventilators and research, the CNS can better stimulate the staff's receptiveness to new procedures and equipment. The CNS understands the change process and puts into place an environment which is conducive for change.

The greatest threat for the CNS who occupies a management position is the sharing of subroles. The CNS needs to perform a self assessment to determine the extent the managing position affects the subrole implementation. Time management becomes a must. The percentage of subrole delivery shifts and the CNS must be comfortable in handling this arrangement. The CNS must have proper support from the administrative side of the house as well as from staff members to effectively deliver this subrole.

This arrangement may benefit the functioning of a

critical care unit. Too many mangers of units do just that, they manage. The CNS becomes involved with patient care and interacts with the ICU staff. This provides this new breed of manager the opportunity to observe, assess, and determine the staff's ability to deliver high levels of patient care and attain positive patient outcomes.

Summary

The activities of the CNS involved in the care of artificially ventilated patients revolve around five subroles. The subrole of advanced practitioner lies in the expert knowledge of the physiological effects of positive pressure ventilation. As a researcher, the CNS contributes to the body of knowledge that helps shape patient care. Consulting with staff nurses and other disciplines increases the delivery of holistic patient care and stimulates problem solving abilities. Education of patient, family, and staff is a continual process used to achieve positive patient outcomes. As a manager, the CNS has the responsibility for developing unit policies and procedures assuring safe patient care.

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