CARDIOVASCULAR DYNAMICS IN THE THERMALLY INJURED PATIENT FOLLOWING INSTITUTION OF POSITIVE END-EXPIRATORY PRESSURE

A THESIS

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We hereby recommend that the thesis prepared under our supervision by Margaret S. Peters entitled Cardiovascular Dynamics in the Thermally Injured Patient Following Institution of Positive End-Expiratory Pressure

be accepted as fulfilling this part of the requirements for the Degree of Master of Science.

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CHAPTER I

INTRODUCTION

Thermal injury, a form of massive trauma, is frequently complicated by lethal pulmonary problems. Acute respiratory failure has become one of the major pulmonary complications seen in the severely traumatized. Studies in recent years have been directed toward early recognition and treatment of this life-threatening complication. The ventilatory management generally accepted for acute respiratory failure is volume ventilation utilizing positive end-expiratory pressure (PEEP).

Although PEEP is used in treating patients with acute respiratory failure, many variable effects can occur within the cardiovascular system as a result. These effects have been described in clinical studies of numerous types of trauma patients, but not specifically thermally injured patients.

Knowledge and understanding of PEEP in the thermally injured is essential for nurses. The nurse at the bedside is continually making thorough assessments of the patient, including his response to all treatment regimes. The data collected provide the basis for appropriate nursing interventions; and evaluation of these actions provides a means for delivering a high standard of nursing care.

Without the necessary background of information about PEEP, the nurse is left with physiologic data that cannot be accurately synthesized. In particular, the expected cardiovascular effects of PEEP must

6. Compare the values of cardiac output taken before and after institution of positive end-expiratory pressure

Background and Significance

Respiratory failure "is today one of the most challenging therapeutic problems in acute medicine" (Seriff et al. 1973). It is an important complication in 30-50 percent of the patients who die in critical care units "following an initial episode of trauma, hemorrhage, major operation, burn or shock" (Moore et al. 1969). For the burned patient pulmonary complications are common not only in the immediate post burn period, but also at later times (Baxter 1967). The exact cause of respiratory failure in all these patients is still controversial. (Alcott et al. 1971). Many eponyms for this syndrome exist, several are respiratory distress syndrome (R.D.S.), adult respiratory insufficiency syndrome, stiff lung syndrome, wet lung, white lung syndrome, progressive respiratory distress, and post traumatic pulmonary insufficiency (Blaisdell and Schlobohm 1973). There has been a marked increase during recent years in the incidence of respiratory insufficiency (Blaisdell and Schlobohm 1973). These authors further stated that this rise in incidence may be due to the increased ability to recognize this problem. It is concluded by both Powers et al. (1973) and Giordano and Harken (1975) that the post traumatic form of the respiratory distress syndrome is characterized by a low arterial oxygen tension ($Pa0_2$) which generally does not respond to increases in inspired oxygen ($Fi0_2$).

It is now well established that positive end-expiratory pressure is the treatment of choice for acute respiratory failure. PEEP acts to improve the arterial oxygen tension of patients with acute respiratory failure and probably does so "by increasing the functional residual capacity and decreasing intrapulmonary right to left shunting" (Ellertson et al. 1974). However, according to Brown et al. (1974) the clinical improvement by use of PEEP "may be limited by the adverse circulatory effects of PEEP impairing venous return and reducing cardiac output." This is a significant point because systemic oxygen delivery is dependent on cardiac output (CO) as well as an arterial oxygen content (Giordano and Harken 1975).

Since clinical reports regarding the effect of PEEP on cardiac output are inconclusive, Horton and Cheney (1975) conducted a study to determine the effects PEEP had on oxygenation and cardiac output in patients with respiratory failure. They studied PEEP at different pressures and observed the changes in PaO_2 and cardiac output. Their results were variable and showed that all patients on at least one occasion exhibited an improved PaO_2; however, on about one-third of the occasions the PaO_2 fell with increasing levels of PEEP. Depression of cardiac output resulted in significant hypotension in almost one-half of the occasions. Therefore, the authors warned that the application of PEEP should be closely monitored.

Suter et al. (1975) also conducted a study on PEEP at different pressures. Their study involved fifteen normovolemic patients with acute pulmonary failure. Application of PEEP increased oxygen transport in thirteen of the fifteen patients studied. There was a progressive

increase in oxygen transport up to a certain level of PEEP and this level varied from patient to patient. At higher levels of PEEP the oxygen transport decreased. The authors state that the decrease in cardiac output caused the decrease in oxygen transport at higher levels of PEEP. Therefore it was concluded that both cardiac output and mixed venous oxygen tension are useful measurements to identify the level of PEEP resulting in maximum oxygen transport.

Powers et al. (1973) studied thirty-three trauma and major surgery patients with an average of a 26 percent shunt. PEEP was used in all the patients to decrease the level of the shunt. The shunt decreased after addition of five to ten centimeters of water PEEP in about three-fourths of the instances, and in each of these instances there was an increase in functional residual capacity (FRC). In the much smaller group increased PEEP increased the shunt, and the FRC decreased in approximately one-third. Cardiac output fell as a result of PEEP in approximately one-half of the instances. In about one-third of all these instances the decrease in cardiac output outweighed the improved arterial oxygen tension with a decrease in oxygen delivery and a fall in tissue oxygen consumption. Powers et al. further state that in healthy man on PEEP the reduction of cardiac output is ascribed to a decrease in the filling pressure of the right side of the heart. Because of the pressure PEEP exerts, it probably obstructs venous return which therefore decreases cardiac output (Suter et al. 1975). According to Olcott et al. (1971) "the presence of an adequate blood volume has been shown to be necessary to prevent decreased cardiac output and blood pressure in response to increased airway pressure." Shires et al. (1973) make similar

statements regarding cardiac output and therefore in their practice attempt to assure adequate intravascular volume before introducing respiratory assistance.

Criteria utilized to initiate PEEP vary from institution to institution and physician to physician. Powers (1974) stated that when a patient is on 50 percent inspired oxygen and his arterial PO_2 is less than 80 mm Hg, then PEEP should be instituted rather than raise the inspired oxygen greater than 50 percent. Different criteria have been suggested recently (Department of Surgery 1976). The indications are that for a patient who is on continuous ventilatory therapy and has optimal cardiovascular dynamics, PEEP might be initiated when any one of the following abnormalities are present: a compliance less than 40 ml/cm water, "a PaO_2 of less than 65 mm. Hg, an alveolar-arterial oxygen difference of greater than 400 mm. Hg on FiO_2 of 1.0, and venoarterial shunt greater than 20%."

According to Blaisdell and Schlobohm (1973), the respiratory distress syndrome gradually begins developing in the first twenty-four hours after initial injury of trauma and reaches a peak after twenty-four to forty-eight hours. It is also concluded by Giordano and Harken (1975) that "the onset of the deterioration of pulmonary parameters begins 24 hours after the initial insult". Therefore it is suggested that prophylactic or early institution of PEEP be used in high risk groups because it prevents the progression of the ventilatory changes of acute respiratory insufficiency.

Since there are no known clinical studies on institution of PEEP in the thermally injured, the intent of this study was to identify the

effects of PEEP on cardiovascular dynamics. This knowledge is essential for nurses because they constantly monitor the thermally injured patient receiving PEEP. Only when they have this information can accurate nursing assessments and appropriate interventions be carried out when PEEP is in use.

It has been shown in this discussion that acute respiratory failure is a detrimental complication and that PEEP is used in ventilatory management of the problem. The hope is to continue to study the effects of PEEP in order to acquire knowledge which may be helpful in more accurately assessing the patient whose management includes the institution of PEEP. A part of this research will be accomplished when conducting this study.

Hypotheses

For purposes of this study the hypotheses were:

1. There will be no difference in the values of arterial systolic blood pressure before and after institution of PEEP
2. There will be no difference in the values of heart rate before and after institution of PEEP
3. There will be no difference in the values of central venous pressure before and after institution of PEEP
4. There will be no difference in the values of mean pulmonary artery pressure before and after institution of PEEP
5. There will be no difference in the values of pulmonary capillary wedge pressure before and after institution of PEEP
6. There will be no difference in the values of cardiac output before and after the institution of PEEP

Definition of Terms

For purposes of this study, the following definitions were used:

1. Positive end-expiratory pressure (PEEP) - positive pressure exerted at the end of expiration by use of a ventilator
2. Cardiac output (CO) - volume of blood which the heart pumps per minute
3. Compliance - expression of the distensibility or stretchability of the lung
4. Thermal burns - destruction of tissue due to hot liquids, flame, flash or chemicals
5. Pulmonary artery pressures (PAP) - the pressure measured by Swan-Ganz catheter and composed of systolic, diastolic and mean readings
6. Pulmonary capillary wedge pressure (PCWP) - the hydrostatic pressure in the capillary and closely correlated with mean left atrial pressure, under normal circumstances
7. Functional residual capacity - the volume of gas in the lung at the end of normal expiration
8. Institution of PEEP - therapy initiated when one or more of the following conditions exist (Hunt and Baxter 1976):
   a. PaO_2 less than 70 mm. Hg with an FiO_2 of 40 percent
   b. Pulmonary veno-arterial shunt greater than 25 percent
   c. Compliance less than 50 ml/cm water
   d. Alveolar-arterial oxygen difference of greater than 400 with an FiO_2 of 100 percent

Limitations

The limitations of this study were the following:

1. Parameters may be measured and/or recorded inaccurately
2. The optimal time for institution of PEEP is not yet defined
3. Selected cardiovascular parameters may not be available prior to the institution of PEEP
4. Individual physiological responses may vary

Delimitations

The delimitations of this study were the following:

1. Males or females between the ages of eighteen and sixty-five
2. The subjects will have sustained thermal injury of second and/or third degree
3. The subjects will have no history of pre-existing cardiopulmonary disease
4. The subjects will be receiving ventilatory assistance by use of the volume ventilator and this will be their first exposure to PEEP
5. The subjects will have PEEP initiated according to set criteria

Summary

The significance of pulmonary problems in thermally injured patients has been presented. Treatment using positive end-expiratory pressure (PEEP) in the thermally injured and its effects on the cardiovascular system have not been documented in the literature. Therefore a need existed to conduct an investigation to determine the effects in this group of patients.

Chapter II presents additional review of the pulmonary complications in the thermally injured and more specifically the adult respiratory distress syndrome (ARDS) which is often encountered in patients with major burns. PEEP has become the major treatment in patients with ARDS and an indepth discussion of what is known about it thus far is presented. The literature review concludes with a presentation of the assessment of patients receiving PEEP. Chapter III presents the procedure used to collect the data for this clinical study. The results of this study are presented in Chapter IV. Analysis was performed by computing the percent change from control level to post-PEEP levels in the parameters. Chapter V includes a summary of the conclusions based on the data results. Implications and recommendations for future nursing studies are set forth.

CHAPTER II

REVIEW OF LITERATURE

Introduction

Thermally injured victims face innumerable crises throughout their hospitalization. Pulmonary complications pose a major threat and are a common cause of mortality in burned patients. The stages of pulmonary complications will be discussed in this chapter with emphasis on the adult respiratory distress syndrome (ARDS). The major treatment of this lethal complication is positive end-expiratory pressure (PEEP) and this form of respiratory therapy is discussed in detail. PEEP has the potential of making both beneficial and detrimental changes in a patient's condition, and therefore the latter part of this chapter explains all the assessment parameters vital for properly monitoring patients being treated with PEEP.

Pulmonary Complications of the Thermally Injured

Pulmonary complications are significant in the early mortality statistics of thermally injured patients "and a major contributing cause of late mortality" (Ashbaugh and Petty 1972). More specifically, respiratory failure is the most common cause of death in burned patients (Parks 1976). The incidence of pulmonary complications is greater today because successful resuscitation measures, improved systemic antibiotics and topical antimicrobial therapy have decreased the early deaths from

burn shock and sepsis, thus allowing the time for the lethal pulmonary complications to intervene (Pruitt et al. 1975; Shook et al. 1968; and Foley et al. 1968).

The pulmonary complications of thermal injury can be explained best if classified in stages according to their chronological onset and termed the immediate, intermediate and late complications (Achauer et al. 1973). The acute or immediate stage is seen within minutes to hours post burn. The second stage of progressive or intermediate problems is noted twenty-four hours to five days post burn (Pierson 1976, Achauer et al. 1973). The third stage or late pulmonary complications following thermal injury commonly occurs after the fifth post burn day (Achauer et al. 1973).

Problems in the immediate stage can be the result of one or a combination of the following occurrences after a burn incident and these "are the most immediate threats to life in burn patients" (Baxter 1977). According to several authors (Cohn 1973; Achauer et al. 1973; Parks 1976; and Pierson 1976), heat and irritating chemicals may cause edema and bronchospasm which can lead to large upper airway obstruction. Lower airway damage may result from deep inhalation of toxic particles and vapors into the small airways and alveoli (Parks 1976, Achauer et al. 1973). Additionally, inhalation of carbon monoxide, which binds with hemoglobin reducing oxygen transport, can lead to confusion, collapse, and even death if severe enough (Mellins and Park 1975).

After the first twenty-four hours another major problem, the intermediate stage, becomes evident in a number of thermally injured patients. It has also been named progressive pulmonary insufficiency

(PPI) and "is a particularly lethal complication of massive cutaneous burns" (Bredenberg 1974). Haeringen et al. (1975) have suggested that PPI is associated with diffuse alveolar collapse.

In thermally injured patients PPI can result from large amounts of fluid during fluid resuscitation; effects of the massive burn itself (Murray 1970, Blaisdell 1974); inhalation injury, shock, and/or sepsis (Pruitt et al. 1975, Hessel 1975, Rosen 1975). The adult respiratory distress syndrome (ARDS) is a synonym to PPI.

Pulmonary complications during the delayed stage include pneumonia, pulmonary emboli (Achauer et al. 1973) and ulcerative tracheobronchitis in patients who have tracheostomies (Pruitt et al. 1975). Harrison (1968) stated that bronchopneumonia had "become a major cause of death among burned patients, and that it was the cause of "much of the pulmonary embarrassment which occurs in the second to sixth weeks post burn". Presently, bacterial pneumonia is still the most common complication in the delayed stage (Parks 1976) and is the "principal late cause of death" (Pierson 1976).

The adult respiratory distress syndrome has been a significant problem and there has been no effective treatment method. This has resulted in high mortality rates in all types of patients. A major breakthrough in the treatment of ARDS occurred in the last ten years and mortality rates, more noticeably early mortality, have finally been reduced (Ashbaugh and Petty 1972). One form of treatment is positive end-expiratory pressure (PEEP) which is given in addition to the ventilatory management of critically ill patients. Before a thorough description of the use of PEEP is presented in this paper, an explanation concerning ARDS will be presented.

Adult Respiratory Distress Syndrome

In 1969, Moore et al. described and classified ARDS into four phases. This classification with some modification is currently utilized because "it provides a reasonable clinical description for understanding the pathophysiology" (Bryan-Brown and Shoemaker 1973).

Phase one consists of the initiating factor which may be injury, surgery, infection, or hemorrhage followed by resuscitation from one of these low flow states. Patients exhibit hyperventilation with hypocarbia even though there is still elevated lactic acid from the low flow state. Pulmonary compliance may be decreased but arterial oxygen tension is normal (Golden et al. 1974). Recovery often occurs following phase one, but if the process is to continue, phase two is designated as the circulatory stabilization phase, and is accompanied by apparent respiratory difficulties (Moore et al. 1969). Cardiac output is often elevated and tissue perfusion restored. Tachypnea and borderline PaO_2 on room air seen at this time are the danger signs of continuing problems. This phase may last for hours or from three to five days. Recovery does occur following this phase.

Phase three is called progressive pulmonary insufficiency, and is manifested by dyspnea, hypoxemia refractory to high oxygen concentrations, hypocarbia and increasing infiltration noted on chest x-rays. Ventilatory assistance with an artificial airway and mechanical ventilation become necessary for survival for these fatigued hypoxic patients. Recovery can still occur but if continued treatment is unsuccessful, the final phase of terminal hypoxia and hypercarbia with cardiac failure ensues. Phase four is usually a short phase lasting only hours with

few survivors (Moore et al. 1969). The lungs of patients with ARDS at autopsy have been found to be wet and edematous with liver-like consistency (Golden et al. 1974).

Etiologies of ARDS

There are other etiologies of ARDS, including use of banked blood, aspiration, fat embolism, renal transplantation and oxygen toxicity (Bredenberg 1974). The clinical setting in ARDS usually involves a combination of etiologic factors "making clear delineation of the specific etiologic factors impossible" (Bowen and Miller 1975).

For the thermally injured, it is apparent that several combinations of these etiologic factors can be involved in the development of ARDS. The early onset of ARDS occurring within the first day is more likely associated with the severe injury, fluid resuscitation, aspiration and/or burn shock. A later onset of ARDS, occurring at three to five days post injury, is most frequently caused by sepsis. This relationship was reported recently by Fulton and Jones (1975) in a clinical study including a variety of patients with major illness or injury. Their results support the clinical experience reported by Walker and Eiseman (1975).

ARDS is compounded by certain factors in the thermally injured which potentiate the process. These factors include painful burns, constricting eschar from burns of the thorax, and cerebral depression from medications. These conditions depress the mechanism for sighing and deep breathing and potentiate alveolar collapse (Achauer et al. 1973).

Pathophysiology of ARDS

As a result of the pathophysiologic mechanisms at the cellular level, damage to the pulmonary capillary endothelium produces a "capillary leak phenomenon" (Gracey 1975), followed by the formation of interstitial and intra-alveolar edema and hemorrhage (Pruitt et al. 1975, Murray 1970, Haeringen 1975). The accumulation of protein, fibrin, fluid, and/or cellular elements of blood in the alveoli and interstitial tissue may lead to alveolar collapse (Webb 1976, Haeringen et al. 1975). According to Clowes et al. (1975) diffuse alveolar collapse is present in patients with ARDS. Fluid-filled alveoli and alveolar collapse lead to many pathophysiologic abnormalities. These include decreased functional residual capacity, decreased lung compliance, increased pulmonary right-to-left shunting, and decreased PaO_2 (Pruitt et al. 1975; Pontoppidan et al. 1972; Haeringen et al. 1975; Blaisdell 1974; Petty 1974; Powers et al. 1972) Functional residual capacity (FRC) is the quantity of air remaining in the lung at the end of expiration. Solliday (1976) states the decrease in FRC may be due to the fluid within the alveoli, and may also be the result of a defect in surfactant which can subsequently enhance alveolar collapse. Surfactant is a phospholipid present in the alveolar lining fluid thought to be produced by Type II alveolar cells (Schonell 1974, Wade 1973). It is capable of lowering surface tension as the surface of the alveoli become small (Bushnell 1974, Schonell 1974, Moore et al. 1969). Several authors speculate that surfactant production is altered or inactive in ARDS because of damage to the surfactant producing cells (Blaisdell 1973; Rosen 1975; Solliday 1976). According to Rosen (1975) theoretically the damage occurs as a result of "low

flow perfusion" and similarly Blaisdell (1974) concludes that "impaired nutritional flow" may cause the damage. Another factor of importance is sepsis which Rubin et al. (1972) stated decreased the synthesis of surfactant. The consequence of the disruption of surfactant is that surface tension at low lung volumes is no longer reduced, thus eliminating the stabilization of the alveoli and terminal airways and enhancing alveolar collapse (Solliday 1976; Clowes et al. 1975; Golden et al. 1974; and Pontoppidan et al. 1972).

Another common feature that occurs in ARDS is the loss of compliance. Compliance is the measure of the distensibility of the lung and is described as "the amount of gas which fills the lung for each centimeter of water inflating pressure" (Rosen 1975). Thus the measurement involves a pressure/volume relationship (Bushnell 1974). The decrease in compliance is likely caused by damage to the surfactant producing cells (Blaisdell 1974). Collapsed alveoli then require higher pressures for reinflation (Asbaugh et al. 1967, Pontoppidan et al. 1972). The relationship between decreased FRC and compliance has been observed in various research studies of trauma and surgery patients with ARDS. One such study with twelve trauma patients exhibiting ARDS conducted by Powers et al. (1972) found that compliance values were decreased and were "invariably associated with a drastic reduction in functional residual capacity". Also pulmonary edema contributes to a decrease in compliance because "liquid is less compliant than gas" (Solliday 1976).

One of the most important consequences of fluid filled or collapsed alveoli is increased pulmonary right-to-left shunting associated with a drastic reduction in FRC (Powers et al. 1972, Hessel 1975). Pulmonary shunting occurs as blood flows through areas of atelectasis

and arrives back in the pulmonary venous system undersaturated (Boyd 1972, Murray 1970). The result is a decrease in PaO_2 as measured in arterial blood gases (Hessel 1975). It is essential to understand that the magnitude of the shunt is dependent on the percent reduction in FRC and the variations in perfusion of the collapsed alveoli (Powers et al. 1972). A close relationship exists among decreased FRC, decreased compliance, increased right-to-left pulmonary shunting and hypoxemia (Solliday 1976).

As the pulmonary abnormalities occur there are associated alterations in the cardiovascular system. Patients with ARDS often exhibit high cardiac outputs and tachycardia (Moore et al. 1969, Petty 1974). Moore et al. (1969) stated that in phase two, cardiac output elevations twice the normal sometimes occurred. Powers et al. (1972) reported striking increases in cardiac outputs in their study of trauma patients with ARDS. Frequently, cardiac outputs were greater than 10L/min and although not proven statistically in their study, some of these increases seemed to be associated with hypoxemia. Kontos et al. (1967) also suggested that hypoxemia was the cause of increased cardiac output and tachycardia. It was emphasized by Doty et al. (1969) that the increase in cardiac output is a compensation for intrapulmonary shunting.

Clinical Indications of ARDS

According to Moore et al. (1969) the early warning signs of impending pulmonary insufficiency are characteristically respiratory and exemplify the phase of circulatory stability. Initially, tachypnea and an increased work of breathing are noted (Brendenburg 1974; Petty 1974; Blaisdell 1974; Ashbaugh et al. 1969; Matsumoto and Hayes 1973). Some

patients exhibit intercostal and suprasternal retractions (Webb 1976, Haeringen et al. 1975). These signs of increased work of breathing result from increasingly less compliant lungs (Gracey 1974, Moore et al. 1969, Rosen 1975).

If clinical signs are not marked, the progressive decrease in PaO_2 may be noticed first (Brendenburg 1974), and often in the usual clinical setting, it is the first parameter to become abnormal (Matsumoto and Hayes 1973). As ARDS progresses, hypoxemia becomes refractory to increases in FiO_2 and hypocarbia is present due to hyperventilation.

Assessment of the lungs reveal normal breath sounds initially, minimal secretions and usually a normal chest x-ray (Matsumoto and Hayes 1973; Ashbaugh et al. 1969; Blaisdell 1974; Haeringen et al. 1975). Gradually, the chest x-ray shows evidence of bilateral fluffy alveolar infiltrates, and at this time rales, rhonchi, and diminished breath sounds can be heard on auscultation. (Ashbaugh et al. 1969). On other occasions chest x-rays may become suddenly opacified rather than take a gradual course (Cook 1974).

It is now well established that there are several pulmonary abnormalities associated with ARDS. With the thorough description of this lethal complication completed, a discussion of the major breakthrough in its treatment, positive end-expiratory pressure, will follow.

Positive End-Expiratory Pressure

History

Ten years have passed since Ashbaugh et al. (1967) revived the concept of positive end-expiratory pressure (PEEP). In 1938, Barach et al. used this concept in treating hypoxia associated with acute pulmonary edema. These authors designated this treatment as constant positive pressure breathing (CPPB), a nonventilatory technique in which the patient breathes against a fixed pressure. More effective means of treating this type of edema were found, and it was discontinued in later years (Fratto 1973).

The revival of PEEP began as a result of a study by Ashbaugh et al. published in 1967. Included were twelve patients with the respiratory distress syndrome who had not responded to the ordinary methods of respiratory therapy. Several measures to improve these patients' conditions were utilized, but the most striking therapeutic treatment was the use of PEEP. It was initiated in five of the patients, utilizing either five or ten centimeters (cm) water pressure. Each patient manifested an increase in PaO_2. Central venous pressures and arterial blood pressures either remained stable or improved. The authors hypothesized that PEEP would improve oxygenation by preventing the collapse of alveoli.

After this initial study using the application of PEEP in treating the respiratory distress syndrome, Ashbaugh et al. continued to utilize PEEP and in 1969 published another paper including their total experience with PEEP, which at this time they called continuous positive pressure breathing (CPPB). The authors again reported positive results and suggested that CPPB may also reduce congestion and edema besides

opening and preventing the collapse of alveoli as the mechanisms of improving alveolar ventilation.

Since the reports by Ashbaugh et al. (1967, 1969), many studies have appeared in the literature describing the effects of PEEP. After review of the literature, it is apparent that several terms have been used interchangeably with PEEP. A few of these are end-expiratory pressure (EEP), continuous positive pressure breathing (CPPB), (Ashbaugh et al. 1969), continuous positive pressure ventilation (CPPV) (Kumar et al. 1970, Chusid and Bryan 1973, McIntyre et al. 1969), positive pressure end pressure plateau (PEPP) (Petty et al. 1971), and continuous positive pressure (CPP) (Lutch and Murray 1972). Since the multitude of terms led to confusion, Petty and Ashbaugh in 1971 began using the word PEEP as a more preferable term for ventilatory maneuvers rather than CPPB. In 1972, Petty suggested that CPPB be reserved exclusively for the non-ventilatory technique first described by Barach et al. (1938). He further stated that the word PEEP is the most appropriate term to use for ventilatory manuevers "because it is an accurate commentary upon the expiratory pressure wave form with positive end-expiratory pressure throughout the exhalation phase, and it is easy to say". (Petty 1972).

Pulmonary Effects of PEEP

Initially, the thought was that PEEP prevents alveolar collapse (Ashbaugh et al. 1969) because the air remaining in the lung at end-expiration is increased, thus keeping many alveoli open that would tend to collapse (Ashbaugh 1970, Webb 1976). Bowen and Miller (1975) state that PEEP "seems to 'splint the lung'" thereby preventing alveolar collapse.

Other authors describe PEEP as opening closed alveolar units (Powers 1974; Hammon et al. 1976; Matsumoto and Hayes 1973). According to Solliday et al. (1976), increased intra-airway pressure should theoretically "stabilize, expand, and re-open alveoli, thereby increasing FRC".

An increase in FRC as a result of opening and/or maintaining the patency of alveoli has been found by several researchers after conducting some of the initial PEEP studies (Petty and Ashbaugh 1971; Powers et al. 1972; Monaco et al. 1972). A consequence of increased numbers of alveoli participating in ventilation, is an improvement in the ventilation to perfusion relationship which leads to several other related beneficial effects (Brendenberg 1974). First there is a decrease in intrapulmonary shunting and subsequently an increase in the PaO_2 (Powers 1974; Kirby et al. 1975; Nicotra et al. 1973; Downs et al. 1973; Gracey 1975; Petty and Ashbaugh 1971). Additional research studies on PEEP have also reported increased arterial oxygenation in their patients after PEEP (Ashbaugh et al. 1967 and 1969; Kumar et al. 1970; Sugerman et al. 1972; Falke et al. 1972; Leftwich et al. 1973; Haeringen et al. 1975; and Hobelman et al. 1975).

The degree of the increase in PaO_2 with PEEP is then "related to the resulting increase in lung volume" or FRC (Pontoppidan et al. 1972). While evaluating increases in PaO_2, some authors have found marked increases in PaO_2 within minutes up to an hour after initiation of PEEP (Matsumoto and Hayes 1973, Leftwich et al. 1973). The other related benefits from increased FRC are an expected increase in compliance (Powers 1974) and consequently, as compliance improves, a decrease in the work of breathing occurs (Solliday 1976).

In contrast to the preceding discussion, there are instances when PEEP does not exert such improvements in PaO_2. McMahon et al. (1973) stated that PEEP is effective when shunting and hypoxemia are caused by perfusion of collapsed alveoli, but it is much less beneficial when shunting is caused by pneumonitis or obstructed airways. McMahon et al. (1973) conducted a research study of twenty-seven patients treated with PEEP. Significant increases in PaO_2 occurred in the patients with congestion or pulmonary edema, atelectasis, and fat emboli. However, the authors found no significant response in those patients with chronic obstructive airway disease, pneumonia, or other respiratory disorders. Several patients in the latter group responded adversely to PEEP and others had no change in PaO_2.

In 1975, Barat and Asuero suggested that PEEP may not be beneficial in patients with chronic obstructive airway disease, unless the hypoxemia is believed to be due to a large intrapulmonary shunt. They studied patients with chronic obstructive airway disease in acute respiratory failure. Increases in PaO_2 were minimal in their patients suggesting that in obstructive disease, PEEP may be of little clinical benefit; however, in a few isolated cases it can create clear improvements. Barat and Asuero (1975) suggested that PEEP "not be used routinely in patients with chronic obstructive airway disease who require controlled ventilation". These patients usually have less intrapulmonary shunting, less elastic recoil of their lungs, reduced tendency for alveolar collapse and their FRC is not as depressed (Barat and Asuero 1975).

Additionally, PEEP may not be beneficial in unilateral lung conditions according to Kanarek and Shannon (1975). The case report they

presented was a patient with pulmonary infiltration of the right lung and severe hypoxemia, but with a clear left lung. During the care of the patient, they found that PEEP had produced quite detrimental effects and when discontinued, the PaO_2 increased significantly and perfusion shifted from the diffusely infiltrated lung to the normal lung. Therefore although PEEP increases FRC and improves ventilation to perfusion ratios in areas of pathology, these advantages may be offset by disadvantageous effects on normal alveoli (Kanarek and Shannon 1975).

Increases in PaO_2 after PEEP often lead to the reduction in FiO_2 from high levels, thus avoiding oxygen toxicity (Ashbaugh et al. 1969; Chusid and Bryan 1973; Petty 1972; Kumar et al. 1970; and Gracey 1975). High oxygen concentrations cause damage to the respiratory tract by endothelial cell damage and interstitial edema (Nash 1967). Barber et al. (1970) noted in a group of patients ventilated with 100 percent O_2 that definite respiratory damage occurred after thirty hours. However, no agreement is found in the literature on the length of time or the level of oxygen producing a toxic state. According to Gracey (1975) "prolonged exposure to greater than 60 percent oxygen in the inspired air is toxic to the lung". Rosen (1975) stated that many experts feel that FiO_2 greater than 40 to 50 percent should not be given for long periods because of the potentially damaging effect. Thus by utilizing PEEP, lower, more safe levels of oxygen can be used in order to maintain acceptable arterial oxygenation.

Effects on Lung Water

In 1969, Ashbaugh et al. presented another theory regarding potential benefits of PEEP. Because infiltrates on chest x-ray cleared

early after PEEP in patients with ARDS, the authors suggested that edema fluid was cleared and congestion decreased. The next year, Kumar et al. (1970) similarly postulated that PEEP might cause a "shift in interstitial pulmonary water". This same idea was suggested again in 1972 by Sugerman et al. who stated that although there was no documentation, pulmonary extravascular lung water was decreased with PEEP. Lutch and Murray (1972) stated this decrease occurred because of the "increase in interstitial pressure which provides a 'barrier to the extravasation of fluid". On the other hand Pontoppidan et al. (1972) stated there was still no evidence "that PEEP reduces the total water content" and Alexander et al. (1973) stated PEEP does not actually push water out of the alveoli.

Recently additional research has been conducted to evaluate the question of reduction in lung water and variability has been found in the literature. In a laboratory study by Demling et al. (1975), it was found that increased accumulation of interstitial lung water did occur after an increase from zero to ten cm water pressure. In contrast Dunegan et al. (1975) concluded from their laboratory study that PEEP did effect a small reduction in lung water. Following a study in 1975, Caldini et al. stated that even though improvement of PaO_2 occurs in pulmonary edema, it does not prevent extravasation of fluid in the lungs when perfused at a constant blood flow. Further research will hopefully determine whether PEEP actually decreases lung water or if it increases with PEEP.

Hemodynamic Effects of PEEP

Even though PEEP has proved to be a major breakthrough in the treatment of ARDS a controversy remains concerning the hemodynamic

effects of PEEP. Several well-known researchers have monitored hemodynamic parameters as well as pulmonary parameters in the hope to clearly and completely describe PEEP and its effects.

Blood Pressure and Heart Rate

In the studies which brought renewed interest in PEEP by Ashbaugh et al. in 1967 and 1969, it was found that arterial blood pressure (BP) either remained stable or improved when their patients were placed on PEEP. However, these authors used PEEP only up to 10 cm water pressure in their research.

In the following two studies, data were collected on both heart rate and blood pressure. Levine et al. (1972) compared different ventilatory methods on ten patients with respiratory failure requiring volume ventilation. PEEP was used at 7 cm water pressure or greater. Systolic blood pressure and heart rate were not affected by PEEP. Diastolic blood pressure, however, was increased by PEEP nearly four and one-half millimeters of mercury. The authors summarized that this increase in diastolic blood pressure was probably of questionable biological significance. No patient in the group was in marginal borderline cardiovascular compensation or shock and this may have accounted for the stability of systolic blood pressure and heart rate. They theorized from these results that the cardiac output had not changed in a major way.

Leftwich et al. (1973) studied fifteen patients with severe arterial hypoxemia who also required volume ventilation. PEEP was initiated from 5 to 15 cm water pressure and no significant changes occurred in either blood pressure or heart rate. This group of researchers did not mention the adequacy of the vascular volume in their

patients, however, they do state that inadequate vascular volume should be restored in patients on PEEP. It may be assumed then that vascular volume was adequate. This factor may have prevented any significant changes in blood pressure and heart rate.

Blood Pressure, Cardiac Output/Cardiac Index

Lutch and Murray (1972) evaluated blood pressure and cardiac index in their study of nineteen patients. After application of 5 and 10 cm water pressure, the patients with stiff thorax syndrome (those with decreased compliance) had significant decreases in cardiac index at 10 cm water pressure without a change in systemic blood pressure. They found no decrease in cardiac index at 5 cm water PEEP. The authors accounted for the stable blood pressure by stating that there was an intact baroreceptor mechanism sufficient enough to increase systemic vascular resistance. Research by King et al. (1973) showed similar results to those of Lutch and Murray. PEEP was used at 5, 10, 15, and 20 cm water pressure in hemodynamically stable patients with ARDS. Cardiac output declined significantly at 10 cm water pressure or greater. Systemic mean arterial pressure was not significantly altered until 20 cm water pressure was applied. This lack of change in blood pressure until 20 cm water PEEP suggests "that the vascular reflexes were intact and that effective circulatory fluid volume was adequate in most patients" (King et al. 1973).

Hemodynamic parameters were evaluated by Sugerman et al. (1972) in a study of eight patients with severe arterial hypoxemia. Data were collected while the patients were on 5, 10, and 15 cm water pressure. Neither blood pressure not cardiac output changed significantly at any level of PEEP. Vascular volume was restored when it was low

before or immediately after PEEP was initiated, and this is believed to be an important factor in their study. Sugerman et al. (1972) suggested that no significant decrease in cardiac output occurred "because only a small fraction of the positive end-expiratory pressure was transmitted to the venous system", an amount not sufficient to decrease venous return and thus lower cardiac output.

Another study of patients in acute respiratory failure was done by Falke et al. (1972),and PEEP was utilized at 5, 10, and 15 cm water pressure. The majority of their patients exhibited a fall in cardiac index from baseline of zero PEEP, although at 5 cm water pressure there were little or no changes. This finding at 5 cm water pressure is consistent with data reported by McIntyre et al. (1969). Vascular volume was not mentioned in Falke et al's. study. Theoretically, this factor may have affected cardiac index detrimentally.

One of the more recent PEEP studies was conducted by Hobelmann et al. in 1975. Their research was initiated to evaluate the effects of PEEP in the hope of identifying factors which lead to decreases in cardiac output in some patients. Twelve major surgery patients, hemodynamically stable, requiring volume ventilation were included in the research. Parameters were measured prior to PEEP and at 5, 10, 15 and 20 cm water pressure. At the two lower levels of PEEP, cardiac index remained stable and in a few cases made slight increases. In contrast to these results, cardiac index decreased at PEEP of 15 and 20 centimeters water. Another interesting factor is that only half of the patients were studied at 20 centimeters water PEEP because there were significant decreases in cardiac index at 15 centimeters water PEEP. Systemic arterial

pressure remained essentially unchanged through the increases in PEEP. The only patient who sustained a significant decrease in mean blood pressure did so at 20 cm water pressure. Hobelman et al. (1975) suggested that blood pressure probably did not decrease as a "result of circulatory reflex activity in response to a fall in cardiac output." In view of their data, the authors felt that in a variety of patients, PEEP is safe to 10 cm water pressure if the patient has a cardiac index of three or greater. It was also suggested by them that although other studies have not shown declines in cardiac index, variations are likely to occur because of "differences in patient populations with regard to age, weight, or degree of pulmonary disease" (Hobelman et al. 1975).

The data from these studies have revealed that blood pressure may predictably remain stable up to 20 cm water pressure. However, the cardiac index may only remain stable at 5 cm water pressure and variable results may occur at higher levels of PEEP. There have been several theoretical reasons why cardiovascular parameters at times remain stable, improve or deteriorate. It seems at this point that the state of vascular volume, systemic vascular reflexes, and/or the degree of pulmonary disease may affect these changes.

Two other interesting research studies with quite different protocols were conducted to evaluate cardiac output. To help identify hemodynamic effects of PEEP, in these two studies it was compared with IPPB. The first study, done by Kumar et al. (1970) found a decline in mean cardiac index from 4.5 to 3.6 liters when an average of 13 of PEEP was applied after patients were stable on IPPB. This level of cardiac index was normal or above normal in most of the patients and rose even further when patients were changed back over to IPPB.

In Colgan and Morocco's 1972 study, cardiac output made a small decline following the conversion from IPPB to 5 cm water PEEP. Kumar et al. (1970) suggested the reason there were no detrimental effects on cardiac index in their study was because of initial blood volume augmentation. Similarly Colgan and Morocco (1972) concluded that noticeable depressions could likely occur "particularly in the hypovolemic or marginally compensated cardiac patient". Again, variable results occurred in cardiac output for reasons only theorized. In 1974 contrasting statements to these theories about cardiac patients and hypovolemia were made by Harken et al. as a result of their research study. These authors stated that the hypovolemic patient with compromised cardiac function after cardiac surgery may be quite responsive to increased airway pressure. They included in their study twenty-seven post-operative cardiac surgery patients who all had uniformly low blood volumes. One group of patients exhibited a decrease in cardiac output in response to a PEEP of 10 cm water and a mean left ventricular end-diastolic pressure (LVEDP) of seven and six-tenths. Another group of patients who had a mean LVEDP of 12.85 before surgery, exhibited increases in cardiac output in response to 10 cm water PEEP. Harken et al. (1974) concluded that PEEP does not always reduce CO, but may "boost a failing left ventricle and augment CO". Therefore, impaired peripheral venous tone and hypovolemia are only relative and should not be considered absolute contraindications to PEEP. Similar results were reported by Colgan et al. (1974) who also conducted a study with postoperative heart patients and found that those patients with a cardiac index below normal experienced increases in cardiac index after PEEP. They therefore suggested "that the low-output state following

cardiac surgery need not be a deterrant to the use of PEEP". Harken et al. (1974) state that in cases when PEEP increases cardiac output, it "appears that left ventricular function is limiting cardiac output prior to the institution of PEEP". As a result of the positive results with PEEP in low-output cardiac patients the authors suggested that in order to determine the hemodynamic response of patients who need this therapy, a trial of PEEP would be the best determiner.

Another recent study conducted to clarify the relationship between PEEP and vascular volume was done by Qvist et al. (1975). This laboratory study, conducted on normovolemic dogs, evaluated the effects of PEEP with and without blood volume augmentation. Results of their research indicated an abrupt fall in cardiac index with the application of 12 cm water PEEP, and after discontinuation of PEEP, the cardiac index returned to control values. Blood volume was increased with blood transfusion in one group while on PEEP and their cardiac index rose significantly returning to near control value that existed prior to PEEP. In the period of eight hours prior to augmentation, the authors found some evidence of compensation as heart rate did increase 23 percent in the first hour after PEEP, but systemic vascular resistance remained unaffected. This data suggests that an adequate vascular volume is necessary during PEEP and may need to be augmented further to prevent detrimental changes because PEEP lowers ventricular filling pressures which can lower cardiac output.

Central Venous Pressure, Pulmonary Artery Pressures and Pulmonary Capillary Wedge Pressures

Three other physiologic parameters frequently monitored in the critically ill patient are central venous pressure (CVP), pulmonary

artery pressure (PAP), and pulmonary capillary wedge pressure (PCWP). Collectively, these parameters are used in assessing fluid balance as well as left and right heart function. Because PEEP potentially affects these parameters, an evaluation has been conducted in several of the PEEP studies.

It was found that CVP remained stable in response to 10 cm water PEEP in the studies done by Ashbaugh et al. (1967 and 1969). Other research has reported small increases in CVP with increases in PEEP (Falke et al. 1972; Sugerman et al. 1972; Leftwich et al. 1973; DeJesus 1974; and Hobelmann et al. 1975). No mean change in CVP was found by Nicotra et al. (1973), however, a few patients did demonstrate significant increases in CVP. Increases in CVP, according to Hobelman et al. (1975), are expected as a result of PEEP because it increases intrathoracic pressure (ITP) and when ITP rises, all pressures measured within the cavity should reflect increases. The changes in ITP are related to the compliance of the lungs. "The less compliant the lungs, the less pressure is transmitted to the pleural space" (Giordano and Harken 1975).

Pulmonary artery pressures have had more varied responses. Sugerman et al. (1972) and Hobelmann et al. (1975) both found that PAP and PCWP rose with each increase in PEEP. In the latter study these increases were greater and more significant at 15 and 20 cm water PEEP than at both 5 and 10 cm water PEEP. In contrast to these results, but at levels only up to 12 cm water PEEP, Nicotra et al. (1973) reported no changes in mean PAP and PCW pressures. Likewise, Hayes et al. (1974) found no changes in PCWP in response to PEEP as high as 22 cm water pressure.

Correlation of these parameters was studied by Lozman et al. (1974) in order to make predictions about effectiveness of PEEP. The authors were concerned that left arterial pressure (LAP) might not be accurately reflected by PCWP following PEEP. Five surgical patients were studied using varying levels of PEEP and direct measurements of LAP and PCWP were made. The authors found that at 5 cm water PEEP the readings correlated significantly statistically. However, at 10 and 15 cm water PEEP, they found no significant correlation. Reasons were theorized, and differences between the two may have resulted because alveolar pressure exceeded left atrial pressure after high PEEP, and in these instances PCWP reflected the higher alveolar pressure.

Lozman et al. (1974) summarized their study by stating that "in any case, a rise in pulmonary wedge pressure in association with a fall in cardiac output or blood pressure is an ominous sign and should signal an immediate reduction in the level of PEEP". The next year Hobelman et al. (1975) found this relationship to be true. Decreases in CI were significantly correlated to increase in PCWP. In many of their patients, CI made its major fall when PCWP made striking increases.

Criteria for Instituting PEEP

Criteria utilized before instituting PEEP are not formally established. It is well agreed that PEEP is indicated in ARDS in patients with reduced FRC, but the point at which it is instituted must still be defined.

One of the earlier sets of criteria was suggested in 1972. Pontoppidan et al. (1972) stated that before PEEP is instituted, the

patient must be unable to (1) maintain a PaO_2 of 70 mm Hg with a FiO_2 of 50 percent or more while on intermittent positive pressure breathing, (2) other therapeutic techniques to lower the intrapulmonary shunt must have failed, and (3) the patient must have "adequate blood volume as indicated by the circulatory response to PEEP". The criteria for arterial oxygen is similar to that stated by Powers (1974) who suggested when a patient had a PaO_2 less than 80 mm Hg with a FiO_2 of 50 percent, it was preferable to institute PEEP rather than increase FiO_2. Another author (Rosen 1975) suggested institution of PEEP when a patient could not maintain a PaO_2 of 60 mm Hg with a FiO_2 of 60 percent. Yet another group, Wilson and Rie (1975) mention that it is difficult to establish criteria for use of PEEP; however, they stated a valid indication for PEEP is "the inability to maintain PaO_2 above 60 torr with an inspired oxygen concentration of 50 percent or greater".

Alveolar - arterial oxygen difference is another parameter used by Estafanous (1975) before the application of PEEP. He recommended the institution of PEEP when the A - a difference was greater than 300 mm Hg and when a FiO_2 over 60 percent was needed.

Complications of PEEP

Positive end-expiratory pressure is not without the potential detrimental effects of barotrauma. Several factors lead to the lung's increased susceptability, especially after PEEP. Infection and hypoxia both harm the integrity of the alveolar endothelium (Estafanous 1975). Also the post-burn pulmonary injury through its evolution, damages and weakens pulmonary parenchyma (Jelenko et al. 1975). A third reason is

that overdistended alveoli, which likely surround areas of atelectasis, are more easily ruptured (Estafanous 1975).

Increased airway resistance resulting from low compliance creates the necessity for large tidal volumes, high ventilatory pressures and PEEP to maintain oxygenation. These pressures are transmitted to the vulnerable areas already damaged and/or overdistended and lead to rupture and pneumothorax, the most frequently mentioned complication. Another form of barotrauma often preceeding or accompanying pneumothorax is subcutaneous emphysema. Finally pneumomediastinum, an infrequently mentioned complication, is one which has been found in more recent research.

Incidences of pneumothorax have been reported by many of the researchers previously mentioned that have utilized PEEP. Steir et al. (1974) reported that 17 percent of the patients, a mean of 12, developed pneumothoraces, and in all these cases subcutaneous emphysema was present before the pneumothorax. Another group (McMahon et al. 1973) reported 14 percent of 27 patients developed pneumothorax after PEEP and half of these exhibited subcutaneous emphysema. The complications in this group occurred after the patients had been on PEEP for two days or more. A 14 percent pneumothorax incidence was reported by Nicotra et al. (1973), after clinically managing their thirty-six patients with up to 20 cm water PEEP in order to effect optimal oxygenation. Leftwich et al. (1973) reported only 6 percent incidence of pneumothorax while using PEEP up to nine days in some of their patients. Levels of PEEP ranged from 5 to 15 cm water pressure. Interestingly, two studies which utilized PEEP for short intervals to assess its effects, reported no

complications after using PEEP. In the first one by Colgan and Morocco (1972), PEEP was used at 5 cm water pressure, the second by King et al. (1973) applied PEEP from 5 to 20 cm water pressure.

Length of time PEEP is used may well be a major factor affecting the rate of pneumothorax as noted in the different incidence findings of pneumothorax. Another reason has been speculated by Haeringen et al. (1975). In a study of patients with respiratory failure, the incidence of pneumothorax after PEEP occurred significantly more frequently in those patients with direct pulmonary injury than in those with non-pulmonary injury. The ratios were 11 out of 16 patients with direct pulmonary injury and only 4 out of 20 with non-direct injury.. It was felt by these researchers that pneumothorax "was more closely linked to the primary lesion, i.e. chest injury, than to the method of treatment, i.e. PEEP ventilation". Pontoppidan et al. (1972) made similar assumptions regarding incidences of subcutaneous or mediastinal emphysema and pneumothorax by stating "it is likely that they are related more to the type and degree of pulmonary disease than to the ventilatory pattern".

Bronchopleural fistula has been found in a small number of patients following mechanical ventilation and PEEP (Downs and Chapman 1976). These authors have shown that bronchopleural fistula can successfully be treated and resolved.

More recently, another potential complication has been mentioned, pulmonary hyperinflation associated with mechanical ventilation and enhanced with PEEP. Baeza et al. (1975) state it occurs in the presence of pathophysiologic mechanisms. The first is ball-valve airway obstruction in which air can go in by an incompletely occluded airway, but is

partially obstructed on expiration. Secondly, the circumstance of differential compliance between the two lungs seems to cause this complication. When either of these abnormalities is present, areas become hyperinflated. In the ball valve obstruction the area behind the obstruction continues to hyperinflate, and the area of normal compliance also hyperinflates. The latter occurs as a result of high airway pressures used for the area of low compliance in an attempt to open closed ventilatory units. Mediastinal shifts can be the result of this hyperinflation if a whole lobe or lung is hyperinflated enough to compress these structures. According to Baeza et al. "the hyperinflated lobe or lung alters little in volume between inspiration and expiration," and thus alveolar ventilation is compromised. The end result of these derangements can be a further decrease in arterial oxygenation. In these cases, PEEP must be discontinued since it is creating detrimental situations.

Assessment of Patients on PEEP

Efficient management of critically ill patients requires a continuous nursing assessment. Clinical observations, physiologic monitoring, and laboratory data are all necessary parameters that must be included in the process of making appropriate decisions in patient care. Each variable added to a treatment regime brings its own potential for making physiologic changes both beneficial and detrimental. Nurses often have the major responsibility of obtaining the data for the initial patient assessment. This includes procuring laboratory samples, performing hemodynamic monitoring and making clinical observations. It is

essential for nurses because of their close association with their patients to be keen observers. As Bryan-Brown and Shoemaker (1973) stated "the observer at the patient's bedside provides the best warning system".

Each variable must also be assessed with precision in order for optimal management to be a continuous process. Consequently when PEEP is utilized in patient management, specific related parameters must be watched closely. In this section, the data necessary for effective utilization of PEEP are discussed.

Clinical Parameters

Comprehensive clinical assessments of critically ill patients are an indispensable part of the complete assessment. Observation of the patient comes before attention is focused on the numerous lines, tubes, and/or machines which are attached to or surround the patient. The nurse is the central member of the health team and by the nature of professional responsibility is in continuous close contact with the patient, and therefore performs the major part of all assessments.

After institution of PEEP, it is anticipated that the patient's clinical condition will improve. Signs and symptoms that indicate an optimal clinical response may include some of the following: a change in skin color from cyanotic to pink; a change from cool skin to warm skin; a return to more stable blood pressure; and a return to more normal heart rate. In patients who are assisting in their respiratory cycle, there may indeed be a decrease in the number of respirations and a return to synchronization with the ventilator. All these occur because of improvement in oxygenation without cardiovascular compromise.

However, attention is also focused on indications that signify a detrimental change and pose a major threat to the patient. All the signs and symptoms post-PEEP are compared to the pre-PEEP assessments to aid in evaluating the response to PEEP therapy. The specific observations will be discussed separately according to body system.

Respiratory System

During the process of this assessment, the techniques utilized include inspection, palpation, percussion, and auscultation. Certain instances preclude the use of some techniques. An example in the care of the thermally injured wound include a patient with a partially or totally burned thorax who has either edema, thick eschar, skin coverage and/or surgical dressings. The degree to which a patient is sedated or paralyzed with medication may also alter the observable changes.

Inspection includes general observations such as color of skin or mucous membranes; and the rate, rhythm, symmetry and amount of chest movement during chest excursion (Schonell 1974). The presence of pain associated with respiration must be noted along with the relative ease or difficulty the patient experiences during the respiratory cycle. Additionally the patient's general behavior and emotional status are noted including presence of restlessness, agitation, or irritability.

Determining chest expansion, position of the trachea and the apex beat are of particular importance in palpation of the chest. Any areas of underlying tenderness and the presence of subcutaneous emphysema must also be noted. As percussion of the chest wall is performed "the quality of percussed sounds is described in relation to their intensity, pitch, and duration" (Beyers and Dudas 1977). Percussion of abnormal sounds

helps to detect the sites of the underlying abnormality as well as other pulmonary complications.

The fourth technique is auscultation of the lungs. Determining the intensity and quality of breath sounds along with the presence or disappearance of adventitious sounds is of great benefit to the clinician (Schonell 1974). During this clinical respiratory assessment after PEEP, signs and symptoms indicative of further problems may be associated with PEEP. It is therefore essential to become more familiar with these signs and symptoms of complications not only in the respiratory system but also the cardiovascular system.

The most frequent complication of PEEP is pneumothorax and signs and symptoms which indicate its occurrence in the non-sedated patient include increasing hypoxia, restlessness, irritability, chest pain and intolerance to the ventilator (Estafanous et al. 1975). Observable signs and symptoms of pneumothorax in both the sedated and unsedated patient include cyanosis, distended neck veins, asymmetry and limited chest movement on the affected side (Maykoski and Fabre 1975, Jelenko et al. 1975). Abnormalities during palpation include a shift of the trachea and the apex beat to the unaffected side after mediastinal shift which can be caused by a large tension pneumothorax (Green 1968). Hyperresonance on the affected side along with possible diminished or absent breath sounds are usually present after pneumothorax (Cherniack et al. 1972, Steir et al. 1974).

Subcutaneous emphysema may be apparent along with and often prior to pneumothorax (Steir et al. 1974). It is recognized by increasing puffiness under the soft tissues of the neck, face, and chest wall.

During palpation "a peculiar crackling sensation of bubbles of air underneath the skin" can be noted (Green 1968).

Significant pulmonary hyperinflation, another complication of mechanical ventilation and PEEP, may be indicated by restlessness, intolerance to the ventilator and respiratory distress. If untreated, "signs of obstructive respiratory insufficiency become apparent: agitation, cyanosis, wheezing, prolonged and difficult expiration, diminished breath sounds, and tympanism of the affected lung" (Baeza et al. 1975).

Cardiovascular System

Assessment of this system also includes the techniques of inspection, palpation, percussion, and auscultation. An overlap does exist in the observations of the cardiovascular system and the respiratory system since their functions are so closely interrelated.

During inspection, observation of the general appearance of the skin should include noting the presence of cyanosis, flushing or pallor (Beyers and Dudas 1977). The presence of cyanosis can also be assessed from observing the mucous membranes, lips, ear lobes, tip of the nose, feet and hands.

Palpation includes assessing the skin temperature and moisture by noting particularly whether coolness and clamminess or warmth and dryness exist. The radial pulse is first felt for quality, rate, and rhythm, and then the other peripheral pulses are assessed. Auscultation of the heart will indicate presence of normal heart sounds and existence of arrhythmias.

The cardiovascular system also is affected by tension pneumothorax often manifested by hypertension and electrocardiographic signs of ischemia (Estafanous et al. 1975). Pneumomediastinum, mentioned previously in this section as a complication of PEEP, causes "pressure on the mediastinum and irritation of the receptors around the large vessels" (Estafanous et al. 1975). Arrhythmias and/or hypertension may then appear as the cardiovascular signs present with pneumomediastinum.

The cardiovascular system is disrupted when pulmonary hyperinflation is uncorrected. Signs manifested include systemic hypotension, bradycardia or tachycardia, and eventually profound circulatory collapse (Baeza et al. 1975).

Clinical indications of a decreased cardiac output, (a detrimental change resulting from PEEP) may be evidenced by a decrease in the quality of peripheral pulses. The pulses may become thready or absent if the decrease is significant. Skin temperature and color can also show signs of a decrease in cardiac output. A change from warm, dry skin to cool, moist skin and/or cyanosis which has not been present previously may indicate compromised cardiac output (Brunner and Suddarth 1975). Decreased urinary output may also represent a decline in cardiac output.

Hemodynamic Parameters

Serial assessment of hemodynamic parameters along with pulmonary parameters adds to the precision so essential in optimal patient management. "Precision alone has undoubtedly been a major factor in the improvement of patient survival from acute respiratory distress syndromes" (Bredenberg 1974). Assessment and evaluation of these hemodynamic

parameters directly aid in the evaluation of the effects of PEEP. On the other hand hemodynamic monitoring "exposes the patient to a variety of catheterizations" (Bryan-Brown and Shoemaker 1973) and all may not be available. Because of the potential detrimental effects of PEEP, as well as the beneficial effects exerted by PEEP, it is obvious that the hemodynamics must be carefully assessed.

Heart Rate and Blood Pressure

Two parameters readily available for rapid assessments are heart rate and blood pressure. Together these give an indirect assessment of changes in cardiac output. The frequency of heart rate "is very important in rapid adjustments of cardiac output" (Sodeman and Sodeman 1974). One example of how these may be altered by PEEP is as follows: if the thoracic pressure changes with PEEP reduce venous return and consequently cardiac output, the changes can be assessed by noting decreases in systemic blood pressure and compensatory increases in heart rate. Before blood pressure declined, it may have remained normal for a time because peripheral vascular resistance increased; however, it falls when "adaptive homeostatic mechanisms can no longer compensate" for the reduced cardiac output (Shires et al. 1973). Measurement of blood pressure can be performed directly by means of an intra-arterial catheter connected to a Mercury manometer or a pressure transducer. Indirectly it can be determined by use of the sphygnomanometer and a stethescope, ultrasonic doppler flow meter, or by digital palpation. In comparison of the two methods, Sodeman states that the cuff readings "are on the average 5 mm Hg too low for systolic" and about the same for diastolic pressure (when utilizing the point at which sound disappears as the diastolic in

the cuff reading) (Sodeman and Sodeman 1974). When blood pressure is taken in the leg, systolic is usually ten to forty mm Hg higher than in the arm and diastolic is about the same (Sodeman and Sodeman 1974).

Cardiac Output

Cardiac output, the amount of blood pumped by the left ventricle per minute, helps to "define the extremes of cardiac function" (Weisel et al. 1975) and is a component of the complete physiologic data utilized in assessing critically ill patients. Serial cardiac output determinations will benefit the clinician in difficult clinical situations as follows: evaluating high risk patients for surgery, guiding fluid administration, evaluating responses to cardiotonic drugs and guiding PEEP therapy (Tietjen et al. 1974). These determinations are necessary during PEEP therapy because of the variable effects illicited. This parameter is so important that in 1975 Clowes stated that without monitoring cardiac output in conjunction with pulmonary hemodynamics, "PEEP in excess of 15 cm H_2O should not be employed" (Clowes et al. 1975).

There are several methods that can be used to determine cardiac output, however, not all are practical in the clinical setting. The two methods most frequently used at the bedside for direct measurement are the indicator dilution and the thermodilution. In the first method, the indicator most often used is indocyanine green. The dye is injected into the venous circulation and arterial blood is drawn through a densitometer in order to determine the amount of dye that has circulated to the arterial blood. A computer is then utilized to determine the cardiac output. This method is time consuming, technically complex and relatively inaccurate "at high and low flows" (Weisel et al. 1975). Measurement by

thermodilution is probably the method of choice in clinical practice today and is rapid, simple, and accurate (Swan and Ganz 1975, Weisel et al. 1975). A pulmonary artery catheter with a thermister at the tip is required for thermodilution. The technique is performed by linking the catheter to a small computer and then injecting a designated amount of iced saline or dextrose through the CVP port. The thermister senses the temperature change in the blood and in less than a minute cardiac output is computed and displayed digitally (Weisel et al. 1975, Teitjen et al. 1975). The simplicity of this method brings to nurses the unique advantage of easily performing this major part of the total assessment quickly and as frequently as necessary. With such immediate accurate access to this cardiovascular parameter, the precision necessary in patient assessments is greatly enhanced.

In the adult, normal cardiac output averages five liters per minute (Burrell and Burrell 1973, Boyd 1972). Cardiac output can be expressed in terms of body size in square meters and is referred to as cardiac index. "The resting cardiac index in normal man is approximately 3.3 L. per min. per $M.^2$, with a low value of about 2.8 L. per min. per $M.^2$" (Sodeman and Sodeman 1974).

Central Venous Pressure

Central venous pressure measurements are simple and have for years been used in assessing both cardiac function and fluid balance. In patients without, or with only minimal cardiopulmonary disease, it still may suffice as a guide for both assessments (Toussaint et al. 1974, Blaisdell and Schlobohm 1973, Berk 1975). However, in recent years the accuracy of CVP readings as a measurement of cardiac function in

critically ill patients has been questioned and thoroughly studied. Subsequently it has been found that CVP does not accurately reflect left ventricular function in patients with multiple trauma, hepatic failure, advanced peritonitis, preexisting heart disease, and in thermally injured patients with massive injuries, preexisting cardiopulmonary disease, renal disease, smoke inhalation and associated injuries (Civetta and Gabel 1972; Sharefkin and MacArthur 1972, German et al. 1973). German et al. (1973) state that many thermally injured patients may be managed satisfactorily by "using CVP as a guide to the upper limits of fluid infusion".

Therefore, the results of these studies and others have lead to the use of CVP in the assessment of right ventricular function (Bowen and Miller 1975, German et al. 1973, Berk 1975). It is used to "detect early changes from thoracic pressure changes" following mechanical ventilation (Chusid and Bryan 1973). Consequently, CVP measurements after PEEP are important in assessing the effects increased airway pressure has made on pleural pressure. Increases in pleural pressure will cause increases in right atrial pressure. The amount transmitted to the pleural space is dependent on the compliance of the lung. Stiff lungs with low compliance will transmit less pressure than normal lungs (Wilson and Rie 1975, Giordano and Harken 1975). The adverse effects of increased airway pressure result when right atrial pressure rises causing a decrease in venous return. In turn, this can cause a decrease in cardiac output which "is directly dependent on venous return" (Giordano and Harken 1975). The chain of events can be altered if there is an immediate change in mean systemic pressure (MSP) since "the rate of venous return is regulated by

the relationship between right atrial pressure" and mean systemic pressure (Giordano and Harken 1975). The needed change in this circumstance is an increase in MSP accomplished by either venous constriction or volume expansion which in turn can cause a rise in venous return and prevent detrimental effects on cardiac output (Giordano and Harken 1975).

For CVP readings it is necessary to have either a catheter threaded into the superior vena cava or use of a pulmonary artery catheter which has a CVP port. Measurement can be done by connecting the catheter to a water manometer or a transducer for electronic monitoring. Zero point, the level of the right atrium, is extremely important and the manometer or transducer must be at that point for each measurement. Relative changes in response to therapy are more important than isolated readings (Berk 1975). Measurement done with the patient off the ventilator (and PEEP) at the end of expiration will avoid pressure artifacts caused by increased intrathoracic pressure (Sharefkin and MacArthur 1972, Murray and Smallwood 1977). Normal CVP readings vary slightly according to different sources. German et al. (1973) use from five to ten cm water as normal while others use five to twelve cm water (Walker 1975, Brunner and Suddarth 1975).

Pulmonary Artery Pressure and Pulmonary Capillary Wedge Pressure

Flow directed balloon-tipped pulmonary artery catheters (Swan-Ganz catheters) have gained wide acceptance for use in the clinical area since their introduction in 1970 by Swan and colleagues (Swan et al. 1970, Pace and Horton 1975). At the bedside, the catheter can be introduced in a peripheral vein and advanced into a patient's pulmonary artery without fluoroscopy. The Swan-Ganz catheter thus makes available not only

pulmonary artery and pulmonary wedge pressures, but also cardiac output by thermodilution and mixed venous blood sampling. Having such easy access to these parameters, the physican is now capable of managing "diverse types of circulatory and respiratory failure" (Pace and Horton 1975).

The hemodynamic status and left ventricular function of patients can be assessed quite accurately by monitoring both pulmonary artery and pulmonary capillary wedge pressures. Utilization of the catheter for these pulmonary pressures has been cited as particularly beneficial in the management of the following types of situations: patients with cardiac or pulmonary disease during fluid replacement, in managing patients with acute myocardial infarction, shock, critically ill surgical patients and the thermally injured with extensive injury and/or smoke inhalation. (German et al. 1973; Archer and Cobb 1974; Pace and Horton 1975; Ellertson et al. 1974; Swan and Ganz 1975; and Sharefkin and MacArthur 1972). Pulmonary artery pressure monitoring is also indicated in the "evaluation of therapeutic interventions with mechanical ventilation, vasoactive drugs, hemodialysis, and assisted circulation" (Pace and Horton 1975). One last important indication for use of the Swan-Ganz catheter is its use during PEEP, and according to Powers (1974) proper care of patients on PEEP demands the insertion of this catheter prior to institution of PEEP. Pulmonary artery pressure readings are essential after PEEP in order to obtain the necessary balance between re-expansion of alveoli without compromising pulmonary circulation. Therefore, a satisfactory response to PEEP is one where the magnitude of the intrapulmonary shunt decreases without causing a rise in the pulmonary wedge pressure (Powers 1974).

In order to obtain readings, the Swan-Ganz catheter is connected to a pressure transducer and the pressure tracings observed on a monitor and on the digital readout. The balloon on the catheter is inflated with air to occlude a capillary and the wedge tracing and reading are obtained. To avoid pressure artifacts, the readings should be obtained at end-expiration (Sharefkin and MacArthur 1972). According to Archer and Cobb (1974) pulmonary capillary wedge pressure is elevated by positive pressure ventilation from 5 to 10 mm Hg and at times even more. The authors also stated that "the quantitative effect of positive pressure elevation was variable, not only from patient to patient, but also in a given patient over a period of time". Therefore, in order to obtain a true wedge reading, patients should be momentarily taken off the ventilator at end expiration whenever possible. In evaluating the effects of PEEP on the wedge, readings with the patient on the ventilator also should be taken before and after institution of PEEP. This will give an indication of directional changes in the wedge responding to PEEP.

Pulmonary capillary wedge pressures closely approximate left atrial pressure (Ellertson et al. 1974, Archer and Cobb 1974). Normal pulmonary capillary wedge pressure is from 5 to 12 mm Hg (Robin et al. 1977). Pulmonary artery systolic pressure correlates with pulmonary vascular resistance and normal is from 15 to 30 mm Hg (Sodeman and Sodeman 1974). Pulmonary artery diastolic pressure within normal limits of 6-12 closely reflects pulmonary capillary wedge pressure and subsequently left atrial pressure. However, in many critically ill patients when pulmonary vascular resistance is increased, abnormal

left ventricular function, or tachycardia greater than 120 are present, this relationship between PCWP and pulmonary diastolic pressure correlates poorly (Archer and Cobb 1974; Ellertson et al. 1974; and German et al. 1973). Safer upper limit of normal for pulmonary diastolic pressure is 20 mm Hg (Sharefkin and MacArthur 1972).

Pulmonary Parameters

Arterial Blood Gases

Laboratory methods, along with other assessment data are necessary to adequately monitor the pulmonary system in critically ill patients. Arterial blood gas (ABG) measurements are one of the most frequently used sets of data utilized in assessing the adequacy of ventilation and are an invaluable guide to therapy. Sequential measurements are necessary for meaningful evaluations with the frequency dependent on individual patient status. When specific changes in a patient's therapy are undertaken such as addition of a ventilator or institution of PEEP, ABG samples are taken more frequently. Any sudden deterioration in ABG's as the result of complications of PEEP (i.e. pneumothorax) or from the lesion itself indicate the need for more frequent ABG sampling.

Procurement of arterial blood for ABG's is often the nurse's responsibility and an awareness of proper technique cannot be overemphasized. As Boyd (1972) stated it, "incorrect or unreliable measurements are as dangerous as no measurements at all". Therefore, it is of utmost importance that all arterial samples be placed immediately in an ice water slush and expedited to the laboratory for measurement. This is necessary because the loss of oxygen tension "through the syringe may be

as high as 50 mm Hg per 5 minutes" in blood with an oxygen tension greater than 160 mm Hg (Boyd 1972).

When PEEP is being utilized, it is highly important to record the level of PEEP as well as the FiO_2 at the time each sample is drawn. Obviously this data is necessary to make accurate assessments regarding a patient's oxygenation in relation to his therapy.

Arterial oxygen tension should be maintained between 60 and 80 mm Hg with the lowest PEEP level and FiO_2 less than 50 percent to maintain that level. Carbon dioxide levels should be between 35 and 45 mm Hg or possibly a little below at 32 to 36 mm Hg. (Blaisdell and Schlobohm 1973). By closely observing and managing CO_2 levels, the pH of the blood can be maintained between 7.35 and 7.45.

Intrapulmonary Shunting

The degree of pulmonary shunting is an index of pulmonary function and another guide to PEEP therapy. According to Powers (1974) the determination of the degree of pulmonary shunting should be done repeatedly while managing patients with severe respiratory distress. "Its correlation to other parameters and its response to different modes of mechanical ventilation gives a meaningful trend that can be diagnosed and treated" (Monaco et al. 1972).

For an accurate calculation of the shunt using the Berrgren formula $\frac{(Qs)}{(Qt)}$, mixed venous blood should be used from a pulmonary artery catheter rather than central venous blood (Bendixen et al. 1965, Horovitz et al. 1971). After the patient has been breathing 100 percent oxygen for twenty to thirty minutes, mixed venous and peripheral arterial blood

samples are drawn simultaneously. The results are then placed in the shunt equation which is as follows:

$$\frac{\text{Arterial oxygen deficit}}{\text{Arteriovenous oxygen difference}} = \frac{\text{Oxygen Content of Pulmonary capillary} - \text{Oxygen Content of Arterial blood}}{\text{Oxygen content of Pulmonary capillary} - \text{Oxygen content of Mixed venous blood}}$$

where oxygen content of pulmonary capillary is assumed to be the same as alveolar partial pressure of oxygen (Peters 1977). The results can also be placed in a nomogram for quick estimation of the shunt (Powers 1974). Normal intrapulmonary shunting is less than 5 percent of cardiac output (Olcott et al. 1971) and is attributed to the blood flow through bronchial or coronary pathways which empty directly into the left heart (Moore et al. 1969). Shunting of 12 to 15 percent is seen in almost all patients with major trauma (Boyd 1972). When a shunt is greater than 15 percent and other pulmonary parameters have deteriorated, ventilatory support may be indicated (Blaisdell and Schlobohm 1973, Peters 1977). This deterioration becomes apparent in Phase II according to Moore et al. (1969), since shunting at that time may increase from 15 to 30 percent.

Alveolar-Arterial Oxygen Difference

Another useful pulmonary parameter which aids in assessing oxygenation is the alveolar-arterial oxygen difference (A-a DO_2). "It represents the maximal quantity of oxygen that can be made available to perfusing capillaries after complete displacement of all nitrogen from every alveolus" (Boyd 1972). A-a DO_2 is employed as an indirect assessment of the magnitude of intrapulmonary shunting. A widened A-a DO_2 indicates increased intrapulmonary shunting when cardiac output is

without significant abnormality (Luce et al. 1976). According to Rosen (1975) the A-a DO_2 value often prognosticates "further difficulties even when the patient appears to look well clinically". Its use in evaluating PEEP therapy is based on the knowledge that with PEEP, shunting should decrease as alveoli are opened. Therefore serial measurements are valuable while following clinical changes occurring as a result of PEEP therapy.

For determination of the A-a DO_2, peripheral arterial blood is drawn after the patient has inspired 100 percent oxygen for twenty to thirty minutes. This blood is also expedited to the laboratory so accurate results can be obtained. When results are returned the A-a DO_2 can be calculated utilizing the formula "$P_AO_2 - PaO_2 + P_ACO_2 + P_AH_2O$, where P_AO_2 is the alveolar oxygen tension and PaO_2 is measured directly and P_ACO_2 is assumed the same as $PaCO_2$ and P_AH_2O is 47 mm Hg at 37° C" (Boyd 1972). Normal range for A-a DO_2 is from twenty-five to sixty-five (Wilson and Rie 1975). A gradient greater than 300 to 350 mm Hg, usually is indicative of the need for ventilatory support (Blaisdell and Schlobohm 1973, Peters 1977).

Compliance

Compliance is a measurement which aids in determining the adequacy of ventilation and according to Rosen (1975) is a "prognosticator of the degree of respiratory impairment". It is defined as a relationship between volume and pressure $\frac{(V)}{(P)}$. Knowledge of serial compliance measurements is an important indication of the effects of PEEP therapy (Wilson and Rie 1975). Measurement of compliance is simple, rapid, non-invasive (Fleming et al. 1972), and can be performed by respiratory

therapists, nurses, or physicians. The value of obtaining compliance measurements exists only when the measurements are performed serially and evaluations of the serial readings are made.

Determination of compliance can be made in two ways. The first is dynamic or total lung compliance and changes in its values can be "caused by stiffness of the lung, thoracic wall damage, or abdominal distension" (Boyd 1972). It is determined "by noting the airway pressure and tidal volume at end of inspiration when airflow is transiently zero" (Bredenberg 1974). The value for tidal volume is then divided by the peak inspiratory pressure $= \frac{\text{(Tidal Volume)}}{\text{(Peak Inspiratory Pressure)}}$ to yield the compliance (Boyd 1972). While PEEP is in use, its level in centimeters of water is subtracted from peak pressure before the dividion is carried out (Kumar et al. 1970). Normally dynamic effective compliance ranges from 35 to 45 milliliters per centimeter of water for adult females and 40 to 50 for adult males (Bendexin 1965).

A second compliance measurement is static compliance and is indicated "when overall elastic properties of the lung are studied" (Cherniack et al. 1972). Before calculating static compliance the "inflation hold" knob on the volume ventilator is turned "on" in order to hold inflation for one to two seconds. While observing the manometer during inspiration, the indicator will reach peak pressure and then move down several centimeters of water and hold momentarily. The holding point is called plateau pressure. After noting the plateau pressure and tidal volume for same breath, inflation hold knob is turned off. Static compliance is calculated by dividing tidal volume by plateau pressure. When PEEP is in use, its level in centimeters of water

pressure is subtracted from plateau pressure before the computation is made. Normal static compliance values are between sixty-five and seventy-five centimeters of water.

Optimal use of PEEP also requires the performance of compliance measurements and evaluation of the results. The usefulness of compliance measurements and their value in determining the optimal level of PEEP have been recently studied by Suter et al. (1975). They studied fifteen patients with acute respiratory failure and found there was a certain level of PEEP in each individual patient which produced both pulmonary and cardiovascular benefits. The level which coincided with maximum oxygen transport was termed "best PEEP". Higher levels caused a decrease in static compliance and oxygen transport (cardiac output x arterial oxygen content). Interestingly though, while those parameters deteriorated, both intrapulmonary shunt and PaO_2 improved. The negative effects on compliance and oxygen transport indicated overdistended alveoli and decreased cardiac output. Optimal cardiopulmonary effects occurred when compliance was highest. The authors felt that determination of static compliance provides a "means of finding the degree of lung distention that provides the best gas exchange with the least risk of alveolar overdistention and lung rupture" (Suter et al. 1975). Reliance can now be placed on static compliance as an indicator of optimal PEEP levels. Additionally, compliance is of particular benefit to the clinician when cardiac output and mixed venous blood are not readily available.

Comprehensive assessments of patients on PEEP provide a wealth of knowledge about the patient, his respiratory status and his response

to PEEP. It is imperative throughout the clinical course of PEEP as well as before and after its initiation, to employ vigorous pulmonary toilet. This includes transtracheal suctioning as well as proper positioning and turning. The latter two aid in allowing "passive redistribution of blood flow to different areas of the lung and thus tend to minimize atelectasis" (Matsumoto and Hayes 1973).

Summary

Pulmonary complications have high mortality rates in the thermally injured. These complications may begin immediately, after several days, or later in the patient's hospitalization. Complications arising after forty-eight hours are in the intermediate stage and one of the most frequent and detrimental in this group is the adult respiratory distress syndrome. Mortality from this entity was nearly 100 percent until the revival and successful use of positive end-expiratory pressure in 1967. This mode of respiratory therapy has been the major advance in the treatment of ARDS.

Positive end-expiratory pressure exerts its effects by opening closed alveoli and/or preventing the closure of alveoli. Thus the beneficial effects it produces include increasing functional capacity, decreasing intrapulmonary shunts and increasing PaO_2. It can also produce such detrimental changes as a decrease in PaO_2 in patients with chronic obstructive airway problems. Many varied results from PEEP are seen in parameters such as blood pressure, heart rate, cardiac output, central venous pressure and pulmonary artery and wedge pressures.

Criteria for institution of PEEP have not been formally established. Several parameters are utilized to assess the severity of the

problem and physicians tend to initiate therapy based on their own institution's belief. PEEP is not without its complications, and the most frequent is pneumothorax. Presence of complications does not in all cases demand discontinuance of PEEP, but does necessitate appropriate treatment.

Assessment of patients on PEEP requires continuous thorough nursing assessments. Clinical, hemodynamic and pulmonary parameters are monitored frequently. The nurse is responsible for much of the data collection which includes clinical observations, procurement of blood samples, and physiologic monitoring. Throughout the use of PEEP, continued meticulous care of the pulmonary tree by suctioning, turning and positioning is mandatory.

CHAPTER III

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

Introduction

The purposes of this study were to describe the cardiovascular effects after institution of positive end-expiratory pressure in the thermally injured. To make these determinations, a descriptive study of nonexperimental design was performed (Abdellah 1965). It is the purpose of this chapter to present the procedure used to collect the data and the statistical analysis utilized in its analysis.

Setting

The population in this study included patients from the Adult Burn Unit of Parkland Memorial Hospital, Dallas, Texas. Agency permission was obtained before data collection began (Appendix A). This hospital is the city-county hospital in a large metropolitan area and has an eight hundred bed capacity. The Adult Burn Unit has a six bed intensive care unit and a fourteen bed intermediate care unit. Patients fourteen years of age and older with burn injuries are admitted to the adult unit.

Population

The method of convenience sampling was utilized to obtain the sample for this project (Abdellah 1965). In June 1976, the study

began and continued through October 1977 and all patients receiving PEEP during that time were evaluated as potential candidates for this study. Prior to instituting data collection approval from the Human Research Committee of Texas Woman's University and University of Texas Health Science Center at Dallas was received (Appendix B).

Patients to be included in the population had to meet two sets of criteria. When patients met the following set of criteria, they could be considered potential candidates for the study:

1. Males or females between the ages of eighteen and sixty-five
2. Subjects with thermal injury of second and/or third degree
3. Subjects with no pre-existing cardiopulmonary disease
4. Subjects receiving ventilatory assistance by use of a volume ventilator and had not previously been receiving PEEP

If a potential candidate's respiratory status deteriorated and one or more of the following criteria were present, PEEP was ordered by the physician in charge. The respiratory criteria were:

1. PaO_2 less than 70 mm. Hg with an FiO_2 of 40 percent
2. Pulmonary veno-arterial shunt greater than 25 percent
3. Compliance less than 50 ml/cm water
4. Alveolar-arterial oxygen difference greater than 400 with a FiO_2 of 100 percent

When PEEP was instituted according to these criteria and the patient met the first set of criteria, then the patient was eligible to be included in this study. Because each patient was too critically ill to sign a consent, each patient's legal representative was given a verbal explanation of the study, its purposes and the use of data in

lay terminology. After the explanation was made, each representative was asked to sign appropriate permission for the study. Only patients whose representatives consented to the study were included in the population.

Six patients met the criteria and were included in this research study (Appendix C). There were five men and one woman with ages ranging from twenty-one to sixty. The burn size range was 50 percent to 87 percent, and all the patients sustained second and/or third degree burns. All six patients were studied for a period of forty-eight hours. At some point after the study was completed, each of the six patients expired.

Tool

The form utilized for data recording was designed by the researcher to include all necessary demographic data and physiologic data (Appendix D). Standardized equipment was utilized in obtaining the six cardiovascular parameters designated for data collection. The sphygmomanometer and stethoscope were used to obtain blood pressure readings. Either an apical pulse by stethoscope or a radial pulse by palpation was obtained for heart rate. Central venous pressure readings were obtained by connecting a water manometer to a subclavian intravenous line. Either the Hewlett Packard (78201B) or the Tektronix 414 monitor was utilized with a thermodilution pulmonary artery catheter (IL441667F) to receive a digital readout and pressure waveform of the pulmonary artery pressures and the pulmonary capillary wedge pressures. The IL 601 cardiac output computer connected to the thermodilution catheter was used to obtain the cardiac output values.

Data Collection

Data collection began by obtaining the demographic data from each patient's medical record. The six cardiovascular parameters monitored in the study were blood pressure, heart rate, cardiac output, pulmonary artery pressure, pulmonary capillary wedge pressure, and central venous pressure. These parameters were then obtained from the medical record and recorded from six hours prior to initiation of PEEP every hour to within the hour that PEEP was started. For the next forty-eight hours each patient's six parameters were obtained either personally by the researcher, or recorded from the nursing flow sheet. Recording was done according to the following schedule: systolic blood pressure and heart rate every hour, central venous pressure, mean pulmonary artery pressure, and pulmonary capillary wedge pressure every four hours, and cardiac output once daily. In several instances this data was unobtainable because of technical problems with the monitoring equipment.

Treatment of Data

All data was hand computed and the percent change from control was determined for all parameters at intervals of one hour, eight hours, sixteen hours, twenty-four hours and forty-eight hours after institution of PEEP. The last value for each parameter which was within an hour prior to PEEP was termed the control value. Uncontrollable clinical circumstances prevented the collection of data at some intervals leading to missing data points in a few instances. Percent change as the method of reporting results was chosen because of the small sample size and the

lack of any significant trends which prevented the use of other statistical analyses.

Summary

Six thermally injured adult patients in the Adult Burn Unit at Parkland Memorial Hospital were included in this study. The legal representative for each patient gave written consent for the patient's inclusion in the study after verbal explanation of the study was given by the researcher. The method of convenience sampling was utilized in obtaining the population for this study.

After meeting the criteria for the study, six cardiovascular parameters were monitored in each patient for forty-eight hours. Systolic blood pressure, heart rate, central venous pressure, cardiac output, mean pulmonary artery pressure and pulmonary capillary wedge pressures were the parameters recorded.

Data at intervals of one hour, eight hours, sixteen hours, twenty-four hours, and forty-eight hours post-PEEP were chosen for comparison to the control value of each parameter. The percent change for these data points from control levels was computed. The following chapter presents the analysis of this data with reference to the hypotheses and purposes of this study.

CHAPTER IV

ANALYSIS OF DATA

Introduction

This chapter presents the findings derived from analysis of the data describing the cardiovascular effects after institution of PEEP in thermally injured patients with respiratory complications. The percent change from the pre-PEEP control level to five post-PEEP levels in all six cardiovascular parameters was computed. The post-PEEP levels analyzed were at one hour, eight hours, sixteen hours, twenty-four hours and forty-eight hours after institution of PEEP. Each parameter will be discussed separately in the following sections.

Systolic Blood Pressure

Systolic blood pressure readings for the entire group ranged between 110 mm Hg and 170 mm Hg pre-PEEP. After all patients were placed on PEEP, both increases and decreases in systolic blood pressure were noted. The percent of change from pre-PEEP or control levels to the various post-PEEP intervals was used to clearly describe the changes that occurred. The maximum increase after PEEP was 18 percent and the maximum fall was 35 percent for the total group. In two of the six patients, there was a transient rise in systolic blood pressure one hour after PEEP, however at forty-eight hours the systolic blood pressure readings were lower than control levels. The three patients who had

decreases in systolic blood pressure at one hour post-PEEP, remained below control level the remainder of the study period. One patient experienced no change in systolic blood pressure at one hour, had slight increases and decreases from eight to twenty-four and by forty-eight hours was again at the pre-PEEP level (Table 1).

TABLE 1

SYSTOLIC BLOOD PRESSURE
PRE AND POST-PEEP

Patients	Pre-PEEP	Post-PEEP									
	Control	1 Hour		8 Hours		16 Hours		24 Hours		48 Hours	
	mm Hg	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change
1	110	130	↑ 18	102	↓ 7	120	↑ 9	102	↓ 7	90	↓ 18
2	170	190	↑ 11	134	↓ 21	130	↓ 23	148	↓ 12	158	↓ 7
3	130	100	↓ 23	110	↓ 15	110	↓ 15	90	↓ 30	84	↓ 35
4	160	138	↓ 13	154	↓ 3	142	↓ 11	148	↓ 7	140	↓ 12
5	145	98	↓ 32	128	↓ 11	130	↓ 10	134	↓ 7	142	↓ 2
6	120	120	0	124	↑ 3	98	↓ 18	128	↑ 6	120	0

The first hypothesis which stated "there will be no difference in the values of arterial systolic blood pressure before and after institution of PEEP" has been accepted since a significant difference was not found in the data.

Heart Rate

Heart rate both increased and decreased after institution of PEEP in this group of patients. The range of control heart rates in beats per minute (BPM) was from 91 to 132. In the entire group, the maximum rise

was 20 percent and the maximum fall was 19 percent from the control value and each occurred at the forty-eighth hour. Two of the patients had no deviation from control at one hour post-PEEP, however each of these exhibited a decrease at eight hours. At sixteen hours post-PEEP, both of these patients again experienced increases in heart rate. This trend continued, and at twenty-four and forty-eight hours both patient's heart rates were above control levels. The other four patients initially had rises at one hour between 2 percent and 9 percent. By the end of the study two of these four patients were 9 percent above control value. The other two patients of this group of four were below control level from 3 percent to 19 percent at forty-eight hours (Table 2).

TABLE 2

HEART RATE
PRE AND POST-PEEP

Patients	Pre-PEEP	Post-PEEP									
	Control	1 Hour		8 Hours		16 Hours		24 Hours		48 Hours	
	BPM*	BPM	% Change	BPM	% Change	BPM	% Change	BPM	% Change	BPM	% Change
1	100	100	0	90	↓ 10	104	↑ 4	106	↑ 6	120	↑ 20
2	100	100	0	96	↓ 4	100	0	104	↑ 4	108	↑ 8
3	91	93	↑ 2	74	↓ 18	94	↑ 3	104	↑ 14	100	↑ 9
4	108	114	↑ 5	114	↑ 5	104	↓ 3	100	↓ 7	118	↑ 9
5	132	144	↑ 9	148	↑ 12	128	↓ 3	130	↓ 1	128	↓ 3
6	112	116	↑ 3	96	↓ 14	96	↓ 14	118	↑ 5	90	↓ 19

*BPM Beats per minute

The second hypothesis which stated "there will be no difference in the values of heart rate before and after institution of PEEP" has been accepted because no significant difference in the values was found in this group of patients.

Central Venous Pressure

Only three patients out of the six studied had central venous lines available for monitoring central venous pressures both before and after PEEP. The observed changes in CVP were increases after PEEP in all three patients with only one exception in one patient which occurred eight hours post-PEEP and was a 25 percent decline. The maximum rise in CVP in this group of three was 183 percent and occurred at both twenty-four and forty-eight hours in one of the patients. One general trend was noted at sixteen hours post-PEEP when all three patients exhibited increases in CVP. The range of this increase from control was from 50 percent to 133 percent. At the end of twenty-four hours, all three patients continued to be above control levels. At the close of the study, only two patients had CVP readings and both were also above control levels. As a result of the analysis of the data, the third hypothesis which stated "there will be no difference in the values of central venous pressure before and after institution of PEEP" has been accepted because no significant difference in the values of central venous pressure was found in this group of thermally injured patients (Table 3).

TABLE 3

CENTRAL VENOUS PRESSURE
PRE AND POST-PEEP

Patients	Pre-PEEP	Post-PEEP									
	Control	1 Hour		8 Hours		16 Hours		24 Hours		48 Hours	
	Cm H_2O	Cm H_2O	% Change	Cm H_2O	% Change	Cm H_2O	% Change	Cm H_2O	Change	Cm H_2O	% Change
1	7	8	↑ 14	5.2	↓ 25	12	↑ 71	12	↑ 71	10	↑ 42
2	16	-	-	-	-	24	↑ 50	20	↑ 25	-	-
6	6	8	↑ 33	12	↑ 100	14	↑ 133	17	↑ 183	17	↑ 183

Mean Pulmonary Artery Pressure

Three patients had a pulmonary artery catheter placed before institution of PEEP and had mean pulmonary artery pressures available both pre and post-PEEP. The pre-PEEP readings in these three patients ranged from 11 mm Hg to 27 mm Hg. Both rises and falls in mean pulmonary artery pressure occurred after PEEP. The maximum increase was 81 percent of control level and the maximum decrease was 18 percent and both were in the same patient. In two of the patients there was a decrease up to 10 percent of control at one hour. Both of these then had increases to above control at eight hours and by forty-eight hours each patient's mean pulmonary artery pressure was up from 3 percent to 15 percent above control level. The third patient had a 9 percent increase at one hour and remained stable at eight hours. At sixteen hours there was a 45 percent increase above control and an 81 percent increase at twenty-four hours. The only decrease this patient experienced was at forty-eight hours with an 18 percent fall (Table 4).

TABLE 4

MEAN PULMONARY ARTERY PRESSURE
PRE AND POST-PEEP

Patients	Pre-PEEP	Post-Peep									
	Control	1 Hour		8 Hours		16 Hours		24 Hours		48 Hours	
	mm Hg	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change
2	27	25	↓ 7	35	↑ 29	30	↑ 11	30	↑ 11	28	↑ 3
3	19	17	↓ 10	21	↑ 10	-	-	19	0	22	↑ 15
4	11	12	↑ 9	12	↑ 9	16	↑ 45	20	↑ 81	9	↓ 18

Results of the above data indicated there was no significant difference found and therefore the fourth hypothesis which stated "there will be no difference in the values of mean pulmonary artery pressure before and after institution of PEEP" has been accepted.

Pulmonary Capillary Wedge Pressure

Two of the six patients in the study had a pulmonary artery catheter with a functioning balloon-tip to permit capillary wedge pressures to be measured before and after PEEP. There were both increases and decreases in PCWP after PEEP in these two patients. The maximum increase was 40 percent and the maximum decrease was 15 percent. At one hour post-PEEP, both patients experienced an increase in PCWP, however, at eight hours one had another increase and the other patient's PCWP decreased below control level. By forty-eight hours, both patients had values which were above control level (Table 5).

TABLE 5

PULMONARY CAPILLARY WEDGE PRESSURE
PRE AND POST-PEEP

Patients	Pre-PEEP	Post-PEEP									
	Control	1 Hour		8 Hours		16 Hours		24 Hours		48 Hours	
	mm Hg	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change	mm Hg	% Change
2	15	16	↑ 6	21	↑ 40	20	↑ 33	21	↑ 40	18	↑ 20
3	13	14	↑ 7	11	↓ 15	-	-	11	↓ 15	14	↑ 7

In the fifth hypothesis which stated "there will be no difference in the values of pulmonary capillary wedge pressure before and after institution of PEEP" has been accepted because no significant difference was found.

Cardiac Output

Cardiac output readings were taken less frequently and only three patients had both pre-PEEP and post-PEEP cardiac output determinations. Most of the readings decreased, however there was one increase above control level in one patient. The increase which occurred was 16 percent above control and the maximum decrease was 29 percent. Two of the patient's first post-PEEP cardiac output readings were at sixteen hours and both of these experienced a decrease between 17 percent and 29 percent. The third patient who initially had a very high cardiac output at twenty-four hours had only a 1 percent decrease in cardiac output. At forty-eight hours the other two patients had values in opposite directions of control. One was 16 percent greater than control and the second patient was 6 percent lower than control level (Table 6).

TABLE 6

CARDIAC OUTPUT
PRE AND POST-PEEP

Patients	Pre-PEEP	Post-PEEP					
	Control	16 Hours		24 Hours		48 Hours	
	L/Min*	L/Min	Change	L/Min	Change	L/Min	Change
2	9.32	7.68	↓ 17	-	-	10.83	↑ 16
3	17.00	-	-	16.8	↓ 1	-	-
4	6.60	4.66	↓ 29	-	-	6.2	↓ 6

* Liters per minute

The sixth hypothesis which stated "there will be no difference in the values of cardiac output before and after institution of PEEP" has also been accepted because of the lack of significance in the above data.

Summary

Data was analyzed on the six adult burned patients included in this study. Six cardiovascular parameters were observed before and after institution of PEEP to determine if a difference existed between the values of these parameters as a result of PEEP. Analysis indicated that there was no difference in the values before and after PEEP in all six parameters and all null hypotheses were accepted. In the following chapter, a summary of this study with its conclusions and implications are given along with recommendations for future study in this area.

CHAPTER V

SUMMARY, CONCLUSIONS, IMPLICATIONS AND RECOMMENDATIONS

Summary

Pulmonary complications occur frequently and are potentially detrimental to the thermally injured patient. Positive end-expiratory pressure is a useful modality in treatment of patients with certain pulmonary problems, however the effects of its use in thermally injured patients has not been available. Therefore, this study was undertaken in an effort to describe the cardiovascular effects after institution of PEEP in the thermally injured.

The literature review revealed that there are three stages of pulmonary complications for which the thermally injured are at risk. All the problems in each stage are detrimental and continue to cause a high rate of mortality. One problem which occurs in the second stage of complications is the adult respiratory distress syndrome (ARDS). The successful treatment of this form of respiratory failure did not come about until positive end-expiratory pressure (PEEP) was introduced in 1967. PEEP has since received much attention and intense research. The mechanisms and the numerous effects of PEEP, including the pulmonary and hemodynamic changes are described.

Assessment of each patient is an integral part of the management involved when PEEP is being utilized and must include specific clinical, hemodynamic and pulmonary data which has been documented.

Patients included in this study were chosen by the method of convenience sampling. Each patient was observed immediately pre-PEEP and for forty-eight hours post-PEEP. During the study time the six cardiovascular parameters (when available) were monitored and recorded on the data sheet. Determination of the percent change from pre-PEEP to five different times post-PEEP up through forty-eight hours was the statistical analysis computed. Results revealed there was no significant difference before and after PEEP in the six cardiovascular parameters studied.

Conclusions

The findings in Chapter IV provide the basis for the following conclusions. There is high variability in all of the data, however certain trends did occur and are described in subsequent paragraphs.

Three patients who had transient decreases in systolic blood pressure at one hour post-PEEP (5 cm water pressure), also showed compensatory increases in heart rate in the same hour. This response could have been anticipated because the vascular volume in these three patients was lower than normal. Such declines at 5 cm water PEEP are different from the findings in a study by King et al. (1973) who noted no systemic arterial pressure changes at this level of PEEP; however, the patients in his study were hemodynamically stable. Another study in contrast to these trends is one by Levine et al. (1972) who found no effect on heart rate or systolic blood pressure at PEEP of 7 cm water pressure or greater in hemodynamically stable patients. Leftwich et al. (1973) reported no significant differences in pulse rate and blood pressure at PEEP from 5 to 15 cm water pressure in their patient study. Sugerman et al. (1972)

also found in patients with adequate vascular volume that blood pressure did not change up to 15 cm water PEEP. It may be hypothesized from these results that, had vascular volume been adequate in these patients, systolic blood pressure may have remained stable initially.

The two patients who initially had increases in systolic blood pressure at one hour post-PEEP (5 cm water pressure) had no change in heart rate in the same hour. These two patients also had normal to above normal vascular volumes. The improvement in blood pressure may have been caused by an increase in cardiac output resulting from increased oxygenation; the same rationale DeJesus et al. (1974) proposed when their patients experienced increased blood pressure post-PEEP.

Generally, in this study, declines in cardiac output were noted after institution of PEEP or after increased levels of PEEP. One patient who experienced a decline had a lower than normal vascular volume. This is in contrast to Falke et al's. (1972) study which found little or no change in cardiac output at 5 cm water PEEP. Another patient whose cardiac output dropped 17 percent one hour after an increase in PEEP from 6 to 8 cm water pressure also experienced a decline in systolic blood pressure the same hour. At the same time, however in this patient, heart rate remained unchanged, until the next hour when it rose along with systolic blood pressure. Vascular volume at this stage of the patient's disease was unknown, and therefore it is only assumed that it was lower than normal because of the noticeable declines in cardiac output and systolic blood pressure.

Central venous pressure tended to increase after PEEP. These findings are congruent with those of Falke et al. (1972); Sugerman et al.

(1972); Leftwich et al. (1973); DeJesus et al. (1974); and Hobelman et al. (1975).

Variability in mean pulmonary artery pressures and pulmonary capillary wedge pressures was high. In this study, two patients with an increase in PEEP from the initial 5 cm water pressure showed an increase in mean pulmonary artery pressure. Pulmonary wedge pressures were available in only one of these two patients and also increased with changes in PEEP from 5 to 6 to 8 cm water pressure. The findings are in accord with the research of Sugerman et al. (1972) and Hobelmann et al. (1975), who stated that pulmonary artery pressure and pulmonary capillary wedge pressure are expected to increase with rises in PEEP, the changes being greater and more significant at 15 and 20 cm water pressure than at lower levels. These increases in pulmonary pressures at lower levels of PEEP are in contrast to the results reported by Nicotra et al. (1973) whose patients had no changes in pulmonary artery pressure up to 12 cm water PEEP. It is also contrary to the research of Hayes et al. (1974) whose patients had no change in pulmonary capillary wedge pressure in response to PEEP as high as 22 cm water pressure.

Implications

Implications for the nursing profession derived from this study are described. Nursing responsibilities have increased tremendously since the role of the nurse has expanded. In depth nursing assessments, including not only clinical parameters but physiologic monitoring techniques bring a wealth of knowledge to the nurse and provide the basis for appropriate nursing interventions.

In recent years a great amount of knowledge has been gained concerning the thermally injured patient. Nursing the burned patient has made significant strides and this advance must be intensified. Nursing education programs must contain within their curriculum the problem of the thermally injured. Continuing education programs and staff education programs must also make this content available. Within their framework, the detrimental pulmonary complications this group may encounter from time of injury until total recovery, must be taught.

New modes of treating pulmonary complications are being sought and utilized to decrease mortality. Positive end-expiratory pressure is one major treatment method. Its mechanisms of actions and expected outcomes must be included in educational nursing programs. PEEP is complex and nurses must know there are numerous specific clinical, hemodynamic and pulmonary assessment parameters which must be frequently monitored in order to intervene appropriately. Nursing care of these patients must continue to include vigorous pulmonary toilet. Just by the addition of this one mode of therapy, many changes either detrimental or beneficial can occur. Nurses have to be ready with a thorough understanding of these changes so appropriate nursing actions can continue to be a reality in the nursing profession.

Recommendations

Based on the findings of this study, the following recommendations are made:

1. The study be repeated to include a larger number of patients with the following modifications: the study time be

shortened to one and one-half hours utilizing PEEP levels of 5, 10, and 15 cm water pressure for 15 minutes each and collect the same data as PEEP is decreased by 5 cm water pressure for 15 minute intervals until PEEP is zero

2. A PEEP study be conducted on burned patients with a normal to above normal intravascular volume and observe cardiac output, blood pressure and heart rate
3. A study be conducted to determine the effects of suctioning on arterial blood gases when patients are receiving PEEP
4. A study be conducted to determine the relationship among static compliance, cardiac output and arterial oxygenation at different levels of PEEP

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APPENDIX A

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING
DENTON, TEXAS

DALLAS CENTER
1810 Inwood Road
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HOUSTON CENTER
1130 M.D. Anderson Blvd.
Houston, Texas 77025

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE PARKLAND MEMORIAL HOSPITAL

GRANTS TO MARGARET S. PETERS

a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem:

The problem of this study will be to delineate the effects that institution of positive end-expiratory pressure has on cardiovascular dynamics of thermally injured patients.

The conditions mutually agreed upon are as follows:

1. The agency (may) ([redacted]) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) ([redacted]) be identified in the final report.
3. The agency (wants) ([redacted]) a conference with the student when the report is completed.
4. The agency is (willing) ([redacted]) to allow the completed report to be circulated through interlibrary loan.
5. Other: _____

Date June 25, 1976

Signature of Agency Personnel

Margaret S. Peters
Signature of student

Geraldine M. Brown
Signature of Faculty Advisor

*Fill out and sign three copies to be distributed as follows: Original -- Student; first copy -- agency; second copy -- T.W.U. College of Nursing.

GMIS - 110

TEXAS WOMAN'S UNIVERSITY
DALLAS, TEXAS 75235

COLLEGE OF NURSING

July 19, 1976

Margaret S. Peters
1813 Redbud
Carrollton, Texas 75006

Dear Ms. Peters:

As a recently appointed member of the Human Research Review Committee, Dallas Center, I have read and approved your proposed protocol for research entitled, "Cardiovascular Dynamics in the Thermally Injured Patient Following Institution of Positive End-Expiratory Pressure".

Please continue with your plans for collection of data.

Sincerely,

Tommie R. Wallace

Tommie R. Wallace, R.N., M.S.
Assistant Professor and Coordinator
Graduate Maternal-Child Health Nursing

TRW:rw

OFFICE OF THE ASSOCIATE DEAN
TEXAS WOMAN'S UNIVERSITY
DALLAS CENTER
1810 INWOOD ROAD
DALLAS, TEXAS 75235

OFFICE OF THE DEAN
TEXAS WOMAN'S UNIVERSITY
BOX 23026, TWU STATION
DENTON, TEXAS 76204

OFFICE OF THE ASSOCIATE DEAN
TEXAS WOMAN'S UNIVERSITY
1130 M. D. ANDERSON BLVD.
HOUSTON, TEXAS 77025

APPENDIX B-2

THE UNIVERSITY OF TEXAS
HEALTH SCIENCE CENTER AT DALLAS

Southwestern Medical School

ARTMENT OF INTERNAL MEDICINE

5323 HARRY HINES BOULEVARD
DALLAS, TEXAS 75235
TELEPHONE (214) 688-3466

July 12, 1976

John L. Hunt, M.D.
Department of Surgery

Dear Dr. Hunt:

The Human Research Review Committee has approved your request for a study entitled "Cardiovascular Dynamics In The Thermally Injured Patient Following Institution Of Positive End-Expiratory Pressure".

The Committee asked me to remind you that both the University and the Department of Health, Education and Welfare regulations require that written consents must be obtained from all human subjects in your studies. These forms must be kept on file for a period of three years past completion or discontinuation of the study and will no doubt be subject to inspection in the future.

Furthermore, we have been directed to review any change in research precedure that you might find necessary. In other words, should your project change, another review by the Committee is required, according to DHEW regulations.

Sincerely,

J. Donald Smiley, M.D.

J. Donald Smiley, M.D.
Chairman
Human Research Review Committee

jw

APPENDIX C

DEMOGRAPHIC DATA

Patients	Age	% Burn	Race*	Sex**
1	31	87	B	M
2	60	56	W	M
3	21	85	W	M
4	57	74	W	M
5.	60	62	B	F
6	57	50	W	M

* W - White

B - Black

** F - Female

M - Male

APPENDIX D

DATE__________ DATE OF BURN__________

PATIENT INITIALS________ UNIT NUMBER________ AGE____ SEX____ RACE________

TYPE BURN__________ PERCENT BURN______ INHALATION INJURY- yes or no DATE DIAGNOSED________

CARDIOVASCULAR PARAMETERS PULMONARY PARAMETERS

ne	cm PEEP	H.R.	B.P.	Time	cm PEEP	H.R.	B.P.	Time	PAP Sys.	PAP DYST.	PAP mean	PCW.	Time	C.O.		Time	FiO_2	pH	Hgb	PCO_2	PO_2	O_2 sat	HCO_3	Δ Base	V-a Shunt	FRC	A-a O_2 diff	Compliance	