EFFECT OF POSITIVE PRESSURE VENTILATION AND STATIC EFFECTIVE LUNG COMPLIANCE UPON PULMONARY ARTERY AND WEDGE PRESSURES

by

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STATEMENT BY AUTHOR

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This thesis is dedicated with love to my Mom and Dad.
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ABSTRACT

This descriptive study determined if differences in pulmonary artery and wedge pressures, as measured by a balloon tipped flow directed pulmonary artery catheter, existed between mechanical and spontaneous ventilation. This study also attempted to establish if a relationship existed between the subjects' static effective compliance and differences in pulmonary artery and wedge pressures on and off mechanical ventilation.

Measurements of pulmonary artery systolic, diastolic, and wedge pressures were taken on 14 acutely ill patients during mechanical and spontaneous ventilation. Static effective compliance measurements were made at approximate tidal volumes of 8, 10, 12, and 14 cc per kilogram of body weight.

The data showed there was a significant increase in pulmonary artery systolic, diastolic, and wedge measurements during spontaneous compared to mechanical ventilation. No correlation was found between static effective compliance and changes in the hemodynamic pressures.

It was proposed that the alterations in pulmonary artery and wedge pressures occurred due to the inability of the left ventricle to handle the increased right ventricular output which occurred as a result of the conversion from mechanical to spontaneous ventilation in 12 subjects with myocardial disease.
CHAPTER 1

INTRODUCTION

The normal lung with an acute insult or the abnormal lung with an exacerbation unresponsive to conventional therapy often requires the use of mechanical ventilation. Patients ventilated mechanically with positive pressure receive several nursing and medical measures aimed at relief, if possible, of the underlying pathology or support until the pathology resolves. Arterial blood gases, chest roentgenogram, vital signs, clinical appearance of the patient, and hemodynamic monitoring of pulmonary artery pressures are all valuable devices which may be utilized to evaluate a patient's pulmonary status. Pulmonary artery pressures are measured via a balloon tipped flow directed pulmonary artery catheter (FDPAC) which resides inside the lung vessels. Flow directed pulmonary artery catheters provide direct measurements of pulmonary artery pressures and pulmonary capillary wedge pressure which, in most cases, directly reflects left ventricular end-diastolic pressure. The catheter also enables a means of obtaining blood for mixed venous oxygen measurements which reflect tissue oxygenation and it provides a means to determine cardiac output.

The pulmonary and systemic circulations are based upon pressure relationships which enable blood to move from regions of higher to lower pressure. Mechanical ventilation may disturb these pressure relationships by producing a positive mean airway pressure which may
be transmitted to the pleural space, thus replacing the normal negative or sub-atmospheric pressure therein. A fall in cardiac output is not uncommon when mechanical ventilation is initiated. Also high ventilator generated peak airway pressures may cause the pressure in the alveolus to be dissipated across the pulmonary microcirculation. Measurements obtained from the FDPAC may be affected by ventilator induced alveolar and pleural pressure changes.

The degree of transmission of the ventilator generated positive pressure to the vasculature is believed to depend upon the state of lung compliance. Compliant lungs transmit most of the pressure to the vasculature while non-compliant or stiff lungs transmit least. This study examines the factors of lung compliance and ventilator generated peak airway pressures and their effect upon the measurements obtained from the FDPAC.

**Statement of the Problem**

Patients who are mechanically ventilated may experience the effects of positive pressure on their cardiovascular and pulmonary systems. For patients with a FDPAC in place and who are mechanically ventilated, the procedure for obtaining pulmonary artery measurement varies. In some instances measurements are taken with the patient off the ventilator to delete the effects of positive pressure on the vascular system. In some cases measurements are taken on the ventilator since they may more accurately reflect the patient's present hemodynamic status. In still other instances pressures may be taken on and off the ventilator and compared.
This study attempts to determine the effect of positive pressure ventilation upon pulmonary artery and pulmonary capillary wedge pressures in acutely ill patients requiring mechanical ventilation. The study also seeks to determine if there is a relationship between the state of lung compliance and FDPAC measurements on and off the ventilator.

**Significance of the Problem**

Flow directed pulmonary artery catheters are used extensively in critically ill patients because in most cases they provide an accurate estimation of left atrial pressure and left ventricular end diastolic pressure. In the setting of positive pressure ventilation the FDPAC measurements are potentially susceptible to distortion due to the increased intrapleural pressure. Positive end expiratory pressure (PEEP) causes an even further increase in mean airway pressures which, if transmitted to the pulmonary capillaries, may result in their compression. In the latter case pulmonary capillary wedge pressure (PCWP), instead of reflecting left atrial pressure would more accurately reflect alveolar pressure.

The measurements obtained from the FDPAC are used as a guideline to fluid replacement and left ventricular function, therefore it is important to sort out the effect of positive peak airway pressure and lung compliance before interpreting these measurements. Pulmonary artery and pulmonary capillary wedge pressures that had been markedly higher on the ventilator compared to spontaneous ventilation would most likely indicate that the thoracic vessels had been influenced by the
transmission of positive pleural pressure. In such cases, measurements should be examined closely on and off the ventilator for future reference in the particular patient.

**Research Questions**

1. What will be the changes in the pulmonary artery and pulmonary capillary wedge pressure measurements measured during spontaneous and during mechanical ventilation?

2. Will the state of lung compliance be correlated with differences of pulmonary artery and pulmonary capillary wedge pressures measured during spontaneous ventilation and during mechanical ventilation?

**Theoretical Framework**

Flow through the pulmonary circulation is gravity dependent and affected by differences in pulmonary vascular pressure and resistance. The pressures in the pulmonary arteries, veins, and the alveoli determine blood flow gradients in the lung. Unlike the systemic circulation, the pulmonary circulation is surrounded by gas and so is very much affected by the distending and compressive forces of this gas. Likewise a negative or subatmospheric pleural space pressure facilitates blood flow into and through the lungs. Consequently a change in the normal pressure characteristics of the lung or thoracic cavity will alter pulmonary blood flow.

The FDPAC floated into a pulmonary vessel, directly measures pulmonary vascular pressures. Therefore any event which alters thoracic pressures will be reflected in a change in the measurements
of the FDPAC. Positive pressure ventilation and the state of lung compliance each may alter thoracic pressure relationships. The following discussion focuses upon the pulmonary circulation, positive pressure ventilation, lung compliance, and the interrelationships therein.

Pulmonary Circulation

There are two types of intrapulmonary vessels, namely alveolar and extralveolar. These vessels may be differentiated by their location in the lung and the pressures to which they are exposed. Alveolar vessels are as large as 30 μm (Murray, 1976). It is unclear exactly which vessels are alveolar, but it is believed that these include not only the capillaries but small arterioles and venules as well (West, 1974). Each of these alveolar vessels are suspended in a gas and the pressure surrounding them is believed to be even less than alveolar pressure, approximately -3 to -5 centimeters (cm) of water (Murray, 1976). The major effect on the caliber of alveolar vessels is due to alveolar pressure (Palv). Alveolar pressure is the pressure in the alveolus and under static conditions is equal to the sum of transpulmonary pressure (Pstl) and pleural pressure (Ppl), therefore Palv = Pstl + Ppl (Murray, 1976). The tendency of the lung to recoil inward establishes the transpulmonary pressure when the airways are open and there is no airflow. Because alvolar vessels are thin walled and lacking in support they are liable to be compressed or distended by Palv. When Palv rises above the pressure inside the alveolar vessels, these vessels are compressed and their resistance increases. An
increase in Palv, such as increased lung volume, and/or a decrease in capillary pressure (Pcap), such as decreased flow, can cause Palv to exceed Pcap causing capillary compression. An additional factor is that the caliber of the capillaries is reduced at larger lung volumes due to stretching. Thus, even if transmural pressure of the capillary is not changed with large lung inflations, their vascular resistance increases.

The second type of intrapulmonary vessels are extralveolar vessels. These are usually greater than 100 µm, and include all of the arteries and veins in the lung parenchyma (West, 1974). Their caliber is greatly affected by lung volume since this determines the expanding pull of the parenchyma on their walls. Under conditions of normal spontaneous breathing, extralveolar vessels are exposed to transpulmonary pressure, which is equal to the elastic recoil of the lung. Because Pstl is equal and opposite to Ppl, the pressure surrounding the extralveolar vessels is the same as Ppl. Lung volume therefore has a major effect on these vessels. As lung volume increases to total lung capacity (TLC), the extralveolar vessels are maximally dilated and lengthened and their resistance decreases; approaching residual volume (RV) vessel size decreases. This response to volume change is completely opposite to the resistance changes in the alveolar vessels.

Very large pulmonary vessels near the hilum and the other great vessels are also exposed to Ppl. Systemic venous return is influenced by Ppl or intrathoracic pressure and as a result right and left ventricular output varies with the rate and depth of respiration.
During normal inspiration Ppl falls. This negative or sub-atmospheric pressure facilitates blood flow into the right heart and lungs; however, as Ppl approaches atmospheric pressure blood flow decreases to these areas. Although right ventricular and thoracic blood volume is increased during inspiration, left ventricular filling and cardiac output is decreased due to extralveolar vessel dilatation and alveolar vessel compression.

During normal expiration, intrathoracic pressures increase, and blood flow to the right heart, right ventricular output, and total thoracic blood volume decrease. Extralveolar vessels are compressed and Palv decreases facilitating thoracic blood volume to flow into the distended alveolar vessels. Blood continues to flow from pulmonary veins and capillaries into the left heart secondary to a slightly higher pulmonary capillary pressure as compared to left atrial pressure.

Blood flow through the pulmonary circulation is not uniform but rather gravity dependent and related to differences in pulmonary vascular pressure and resistance. West (1977) has classified blood flow through the lung into three zones. Alveolar pressure is assumed to be consistent throughout the lung whereas Pcap and pulmonary artery pressure (PAP) exhibit hydrostatic gradients down the lung. Inflow pressure to any lung region is determined by the maximum pressure within the pulmonary artery. In the upper zones (I and II), Palv tends to exceed Pcap thus limiting flow. In zone III, the driving pressure for flow is the difference between PAP and Pcap, the normal circulatory gradient. In this zone PAP exceeds Pcap which exceeds Palv.
A flow directed pulmonary artery catheter floated from the right heart tends to go to zone III since the catheter follows flow. This catheter records pulmonary artery systolic (PAS), diastolic (PAD), and pulmonary capillary wedge (PCWP) or wedge pressures. In the absence of mitral valve disease or pulmonary vascular disease the PAD and the PCWP are fairly equal and consistent and these in turn reflect the end diastolic pressure in the left ventricle (Swan et al., 1970). This information about the left ventricle is possible because during diastole the mitral valve is open making the left atrium and left ventricle one chamber. Since there are no valves between the pulmonary veins and the left atrium, PCWP approximates left ventricular and diastolic pressure. The measurements obtained from the FDPAC provide information regarding blood flow through the lungs, left and right heart pumping ability, condition of the mitral valve, and it enables diagnosis and treatment of several cardio-pulmonary disorders.

Pulmonary Vascular Resistance

The major site of resistance in the normal pulmonary bed is probably in the capillaries. Recruitment and distension of pulmonary capillaries are the two mechanisms which are chiefly involved in lowering pulmonary vascular resistance. Normally as blood flow increases through the right heart, pulmonary vascular resistance decreases to accommodate the increased flow. Pulmonary artery pressure usually does not increase until cardiac output increases approximately three to four times. However as the pulmonary bed is attenuated by
disease it becomes increasingly unable to undergo the normal processes of distension and recruitment.

Changes in the caliber of the pulmonary vessels can be determined by the interaction of active and passive influences (Murray, 1976). Passive changes in pulmonary blood vessels occur secondary to changing mechanical conditions in the lung or to hemodynamic events in the systemic circulation. Mitral stenosis is an example of a hemodynamic event initiating alterations in pulmonary vascular resistance. In mitral stenosis, as blood flow backs up into the left atrium, left atrial pressure increases. Consequently pulmonary capillary wedge, PAD, and PAS pressures must increase. Subsequently pulmonary vascular resistance also rises. In contrast, active changes in the pulmonary bed are caused by contraction or relaxation of the smooth muscles of blood vessel walls. Neural, humoral, and chemical stimuli can induce these active changes. Hypoxemia, especially in the presence of acidemia, may lead to severe pulmonary hypertension even in the absence of anatomic changes in the vascular bed (Burrows, Knuudson, and Kettel, 1975).

Positive Pressure Ventilation

In normal spontaneous breathing, maintenance of a negative or subatmospheric pleural pressure facilitates blood flow through the lungs and enhances systemic circulation. Normally during inspiration $P_{pl}$ becomes more negative or subatmospheric to facilitate air entry into the lungs. At the same time the caliber of the great vessels increase, thus promoting blood flow into the right heart and expanding pulmonary
blood volume. During expiration Ppl is less negative and Palv becomes positive with respect to atmospheric pressure to facilitate air flow out of the lung. The increase in Ppl at expiration causes the extralveolar vessels to decrease in size thus facilitating blood flow from the capillaries to the left heart.

Certain circumstances, however, necessitate the use of mechanical ventilation to force air or an oxygen enriched mixture into the lungs. Ventilation is therefore performed under positive pressure. There is much terminology, used clinically and in the literature, describing the methods of mechanical ventilation. The following terminology will be utilized in this thesis. The term positive pressure ventilation describes all types of ventilation employing positive airway pressure during inspiration, expiration, or both. Intermittent positive pressure ventilation (IPPV) is the addition of positive airway pressure only during mechanical inspiration. Positive and expiratory pressure (PEEP) is a ventilatory maneuver in which positive airway pressure is maintained during exhalation. Continuous positive pressure ventilation (CPPV) is the addition of varying positive airway pressure during both inspiration and exhalation with positive pressures being higher during inspiration than expiration (IPPV + PEEP). Continuous positive airway pressure (CPAP) is the maintenance of a constant positive airway pressure during both inspiration and exhalation under conditions of spontaneous breathing. Intermittent mandatory ventilation (IMV) is a mode of ventilation in which the patient is allowed to breathe spontaneously in between a preset number of ventilator delivered breaths.
Intermittent Positive Pressure Ventilation

The circulatory effects of IPPV depend largely on the degree to which airway pressures are transmitted to intrathoracic and alveolar vessels. During IPPV, Palv is positive during inspiration. Intrathoracic pressure (Ppl) can also become positive at peak inspiration. These pressure changes which occur in the lung are exactly opposite to the changes previously discussed which occur during normal spontaneous breathing. During inspiration intrathoracic pressure rises and Palv increases as gas flows into the lungs under positive pressure. The great vessels can be compressed secondary to the intrathoracic pressure increase, delaying right ventricular filling; thus right ventricular stroke volume and output are diminished (Cournand et al., 1948; Sheldon, 1963). Secondary to the decreased venous return, peripheral venous pressure may rise. Because during inspiration extralveolar vessels are compressed some pulmonary blood may be expelled from the lungs and pulmonary capillaries leading to a fall in thoracic blood volume. As a result left ventricular filling and output rise for a few beats corresponding to the time required for the decrease in pulmonary blood flow to occur. During expiration, intrathoracic pressure falls back to sub-atmospheric. The extralveolar and great vessels have enlarged in size. Venous return is unimpeded and right ventricular filling rises if exhalation is longer than inspiration. Right ventricular output and total thoracic blood volume increase. Since less blood is going to the left heart its output momentarily falls (Cournand et al., 1948; Sheldon, 1963). A decrease in cardiac output
during IPPV may occur but is usually compensated by an increased peripheral vascular resistance, in the absence of hypovolemia and central nervous system dysfunction (Price, Conner, and Dripps, 1954). Some reports state that IPPV can also aid in increasing cardiac output, presumably by decreasing the work of breathing and correcting myocardial hypoxia and ischemia.

Several factors determine the overall effects of IPPV on pulmonary circulation and cardiac output. Lung compliance, airway resistance, ventilatory pattern, and overall mean airway or peak inspiratory ventilator induced pressures are pulmonary factors. Circulating blood volume, heart contractility, vasomotor reflexes, and the presence or absence of other complicating disease are also involved in determining whether or not positive pressure effects will ensue.

Positive End Expiratory Pressure (PEEP)

The major physiologic effect of PEEP is an increase in functional residual capacity which in turn causes a decrease in shunting of blood. Alveolar and small airway closure is prevented by the increased functional residual capacity and lung units which were previously collapsed may be opened. Maintenance of lung units in an open position may allow production of normal surfactant which may be altered in the diseased lung.

Positive end expiratory pressure (PEEP) like IPPV may cause a decrease in cardiac output. When CPPV is used, the peak and mean airway pressure can increase to very high levels. Inspiratory airway pressures may be higher than with IPPV alone and expiratory airway
pressures, instead of returning to atmospheric levels as in IPPV, remain supraatmospheric or positive. It is generally felt that the increased Ppl which is generated can be of sufficient magnitude to cause compression of the extrathoracic or great vessels causing decreased right heart filling pressures. Also, it is generally felt that the addition of 5 centimeters (cm) of water PEEP is "physiologic" and it has been proven in at least one study by McIntyre, Laws, and Ranachandran (1969) that 5 cm PEEP does not cause a decrease in cardiac output. Higher levels of PEEP are associated with more problems of decreased cardiac output. The same factors which determine the effect of positive Ppl with IPPV are operative with PEEP and CPPV.

If the lungs are overdistended, a potential adverse effect of PEEP is compression of alveolar vessels by higher alveolar pressures. The result of this compression is increased pulmonary vascular resistance. Potential compression of extralveolar vessels due to the positive Ppl generated by CPPV may further contribute to increasing pulmonary vascular resistance as well as decreasing cardiac output.

Compression of alveolar vessels during CPPV makes it particularly important to carefully assess FDPAC measurements. If Palv exceeds Pcap and one attempts to obtain a wedge measurement, the reading obtained will reflect Palv rather than left ventricular and diastolic pressure. Correspondingly, PAP will read higher if extralveolar vessels are compressed, correctly reflecting pulmonary vascular pressure under positive pressure.
Lung Compliance

Compliance is an expression of the distensibility of the elastic respiratory system. This property of compliance depends upon the elastic characteristics of the lung and chest wall. The latter consists not only of the skeleton and tissues of the thoracic cage but of the diaphragm, abdominal contents, and abdominal wall (Burrows et al., 1975). The elastic recoil properties of the lung originate from the lung tissue itself and pulmonary surfactant, a substance which functions to lower surface tension and decrease pressure required to distend alveoli. Stated more precisely, compliance is the volume change per unit of pressure change measured under static conditions and is expressed in liters or milliliters per centimeter of water. Static conditions of no flow must exist during compliance measurements to ensure that the pressure applied to the system is opposed only by elastic forces and not by flow resistive forces of the airways. Pressure and volume changes are plotted. The slope of this plotted curve (P/V or pressure/volume curve) is the compliance. As the lung is inflated to total lung capacity each increase in volume is accompanied by a pressure change. As the lung approaches total lung capacity, pressure changes result in smaller volume changes than near functional residual capacity. Any further increase in pressure at total lung capacity may rupture the lung. At total lung capacity the relation of volume to pressure change is low and the tendency of the lung to recoil is high. Therefore compliance is low at total lung capacity. The reverse is true at low lung volumes. Lung recoil pressure (Pstl) is low and the volume change for a unit of pressure
change is large. Therefore compliance is high at the steep portion of the normal P/V curve.

Destruction of alveolar walls and formation of large parenchymal air spaces as occurs in emphysema renders the lung highly compliant with the loss of lung recoil. The P/V curve for this lung type is usually shifted to the left and steep.

Surfactant deficiency, interstitial fibrosis, diffuse parenchymal disease, or decrease in the number of functioning lung units renders the lung stiff with low compliance and high recoil. The P/V curve for this stiff lung type is usually shifted to the right and flattened.

The chest wall tissues also have elastic recoil and compliance. The elastic tissues of the thorax, if not acted on by any force (i.e., lungs), enlarge the thorax from resting volume to about 600 cc above it. Thoracic cage compliance may be decreased in all types of kyphoscoliosis, scleroderma, skeletal muscle disease, marked obesity, and in abdominal disorders with marked elevation of the diaphragm. When chest wall compliance is fairly consistent, static compliance measurements of the lung enables an accurate estimation of respiratory system compliance.

Positive Pressure Ventilation Effect on Ppl

The normal lung inflated mechanically has its muscle force replaced by positive airway pressure. Instead of Ppl being equal and opposite to Pstl as during static conditions of spontaneous breathing, Ppl is now equal to chest wall recoil (Burrows, 1978). Normally the
chest wall wants to recoil outward. However when lung volume exceeds
60-70% of total lung capacity, the chest wall wants to get smaller and
therefore chest recoil becomes positive. Thus Ppl is positive and peak
positive ventilator generated airway pressures may possibly be trans-
mitted to thoracic vessels causing all of the positive pressure effects
on the circulation previously discussed.

The non-compliant lung has a very high Pstl. The lung has the
tendency to always return to its small resting volume. When positive
pressure is applied, it is difficult for the lung to expand. Lung
recoil pressure may even be greater than PAlv or airway pressure. The
chest wall expands since its volume is below 70% of total lung capacity
making chest recoil pressure negative. Pleural pressure therefore
remains negative as in the normal lung during spontaneous breathing.
It is unlikely that the positive airway pressures will be conveyed to
the pleural space and the thoracic vessels, since Ppl is negative.

Decreased elastic forces make the compliant lung easily
distensible. It takes only a small amount of positive pressure to
further distend this already hyperinflated, highly compliant lung.
Because lung volume is high, chest wall volume is greater than 70% of
total lung capacity. The chest wall tends to recoil producing
positive chest wall and Ppl pressures. Compliant lungs are therefore
very susceptible to transmission of positive airway pressures.

Unfortunately patients' lungs are rarely homogeneously compliant
or non-compliant. Consequently positive pressure may be transmitted
unevenly throughout lung tissue. One could assume that a patient with
fairly homogeneous stiff lungs would not transmit positive pressure
to his circulatory system and thus FDPAC measurements taken on and off positive pressure ventilation should be congruous. Likewise a patient with fairly compliant lungs with positive pressure effects on the circulatory system should most likely show incongruous FDPAC measurements on and off positive pressure ventilation. Patients with non-homogeneous lung compliance changes should vary in response to positive pressure ventilation. However, those with a tendency to a low lung compliance may show no considerable measurement changes while those with a tendency toward high compliance may show considerable measurement changes.
CHAPTER 2

REVIEW OF LITERATURE

A summary of the literature pertinent to (1) the effects of positive pressure ventilation on specific hemodynamic measurements, (2) measurement of static effective lung compliance and the effects of lung compliance on the transmission of positive pressure to the intrapleural space, and (3) the clinical measurement of pulmonary artery and pulmonary capillary wedge pressure is presented.

Effect of Positive Pressure Ventilation on Hemodynamic Measurements

Grenvik (1966) studied 22 thoracic surgical patients during spontaneous and intermittent positive pressure ventilation (IPPV). He found with both an air-oxygen mixture and air alone that the transmural right atrial pressure decreased with IPPV as compared to spontaneous ventilation (SV). In nine of the 22 patients, a left atrial catheter was in place and measurements demonstrated that the transmural left atrial pressure remained essentially unchanged between IPPV and SV. Transmural PAP decreased during IPPV as compared to SV. There was no change in pulmonary vascular resistance (PVR) between IPPV and SV with an air/oxygen mixture. However, PVR decreased with IPPV and air alone and increased with spontaneous air ventilation. A significant increase in cardiac index of 23% occurred during spontaneous ventilation as compared to IPPV. The researchers attributed the decrease in cardiac
output and right atrial pressure during IPPV to increased intrathoracic pressure and decreased oxygen consumption. Oxygen consumption increased by 8% during SV. The researchers offered no explanation for the constant left atrial pressure during IPPV and SV.

Davison, Parker, and Harrison (1978) measured wedge pressures on and off the ventilator in 29 patients with a variety of medical and surgical conditions (postoperative complications, pulmonary, neurologic, and cardiovascular disease). The patients were maintained on a volume ventilator with spontaneous ventilatory efforts absent or suppressed by sedatives or relaxants. Thirteen of the patients received positive end expiratory pressure (PEEP); seven at a level of 10 cm, one at 9 cm, two at 8 cm, and the remaining subjects at 5 cm. For the total sample wedge pressures were the same on and off PEEP. In 16 observations performed on the patients not receiving PEEP, mean wedge pressures were the same on and off IPPV. Measurements were taken during end-exhalation from a strip chart recorder. In 6 (21%) of the 29 patients, the researchers observed a delayed progressive elevation of wedge pressure when off the ventilator. This latter phenomenon was observed in the setting of underlying heart disease. The higher pressure was presumed to reflect the higher filling pressures required by the failing left ventricle in order to accommodate the increased venous return that results from a restoration of intrathoracic pressures to atmospheric levels. The authors concluded that PEEP did not result in the occlusion of pulmonary capillaries, so that wedge pressures measured under the conditions of 10 cm of PEEP did not more closely reflect alveolar pressure than left atrial pressure. Also, the authors felt that it is
unjustified to purposely disconnect positive pressure ventilation apparatus in order to measure wedge pressures.

Shinn, Woods, and Huseby (1979) studied the effect of IPPV upon pulmonary artery and wedge pressures in 18 acutely ill medical-surgical patients. They found that there was a small but significant decrease in pulmonary artery pressures when the patients breathed spontaneously compared to those pressures measured during IPPV. However, the authors felt that the differences were small and probably not clinically significant. There was no significant difference between wedge pressures measured off and on the ventilator. The authors used a mean of all pressures displayed over one respiratory cycle to obtain the pulmonary artery and wedge pressures.

Askitopoulou, Sykes, and Young (1978) studies the effect of IPPV, CPAP at a level of 10 cm and spontaneous ventilation in 11 patients after open heart surgery. All subjects received each mode of ventilation. The authors found that there were no significant differences in cardiac output or mean PAP during the three modes of ventilation. However, the mean PAP in five patients and mean left atrial pressure in three patients were consistently greater during CPAP and spontaneous breathing than during IPPV; the differences were not significant because relatively few measurements were made. No explanation was offered for this latter finding. The authors also found a significant increase in mean arterial blood pressure and oxygen transport when IPPV was discontinued. Even though cardiac output did not increase during CPAP as compared to IPPV, the authors stated that they expected this to occur. Their hypothesis was that the use of CPAP would bring
the tidal breathing range to the steep part of the pressure/volume curve. Thus tidal exchange could be accomplished with minimal respiratory work. Consequently, mean intrapleural pressure would be maintained at minimal values and not compromise cardiac output.

Civetta, Brons, and Gabel (1972) demonstrated the hemodynamic measurement differences which occurred during SV, CPAP at a level of 10 cm, IPPV, and CPPV with a level of 10 cm PEEP in two intubated patients. Cardiac output increased dramatically and mean PAP increased slightly during SV and CPAP as compared to IPPV and CPPV. The researchers attributed the increased cardiac output during SV and CPAP to the avoidance of high peak airway pressures.

Downs et al. (1977) compared the hemodynamic effects of four different positive pressure ventilatory modes on ten postoperative revascularization patients. Measurements were obtained while patients received the following ventilatory modes: (1) IMV of two breaths per minute with zero PEEP, (2) IMV of two breaths per minute with 5 cm PEEP, (3) IMV of two breaths per minute with 10 cm PEEP, and (4) IPPV of 12 breaths per minute. Each patient received the same treatment order, and was ventilated for 10 minutes during each mode. Mean transmural right atrial pressure did not change significantly with the addition of 5 cm PEEP, but decreased significantly when the ventilatory rate was increased to 12 with IPPV. Cardiac output and mean PAP decreased significantly when IPPV was initiated compared to the other three modes. The fall in PAP paralleled the decrease in cardiac output during IPPV. Pulmonary arterial resistance was unchanged during the entire study. The researchers hypothesized that the spontaneous
ventilatory activity, which occurred between the mandatory breaths or IMV, maintained thoracic venous inflow of blood, thereby enabling cardiac output to remain at pre-PEEP levels. It was not clear at which point during the ventilatory cycle the measurements were taken.

Kennedy, Weintraub, and Skillman (1977) studied the cardiorespiratory and sympathoadrenal responses during weaning from controlled ventilation in 20 acutely ill patients with a diversity of clinical problems. Patients were placed on 100% oxygen via a mechanical ventilator for 15 minutes, after which baseline measurements were taken. The subjects then breathed spontaneously on 100% oxygen via a T-piece and measurements were taken at several intervals during the initial one hour. Ten patients failed to wean successfully and had to be placed back on the ventilator. The authors found that in both groups (weaners and non-weaners) the partial pressure of carbon dioxide (pCO$_2$) rose quickly from control levels and remained at a significantly elevated level throughout the one hour period of weaning. The most striking characteristic of patients who failed to wean successfully was an abrupt, persistent increase in the alveolar-arterial oxygen difference (AaDO$_2$) within five to 10 minutes after controlled ventilation was withdrawn. The mean cardiac index increased significantly during weaning in comparison to control (IPPV) in all groups. Successful weaners had a slightly higher mean cardiac index in comparison to non-weaners. The authors' data did not allow a clear interpretation of whether withdrawal of positive pressure ventilation or an increased pCO$_2$ was the stimulus for the increased cardiac index. Mean left atrial pressure or wedge pressure was measured in seven patients, three
of whom weaned successfully and four of whom did not. There were no significant changes in mean left atrial or wedge pressure. Mean left atrial pressure values were greater in each period (except one) among non-weaners than the corresponding value for weaners. The authors concluded that the increased AaDO₂ in non-weaners was due to the increased venous admixture (Qs/Qt), since cardiac output had increased. Since the rise in venous admixture was not associated with a concomitant rise in left atrial pressure or wedge pressure, the sudden increase in venous admixture was thought by the authors to be caused by the acute development of atelectasis, rather than left ventricular heart failure and pulmonary edema. Withdrawal of mechanical ventilation increased the stress on the heart in both groups. Left ventricular work increased significantly during weaning with non-weaners having a greater increase.

Beach, Miller, and Grenvik (1973) studied hemodynamic responses to the discontinuance of mechanical ventilation in 37 patients. The patients were arbitrarily grouped according to their cardiac output response to discontinuance of mechanical ventilation. Group A had an increase in cardiac output; group B had a decrease in cardiac output when changed from IPPV to SV. Central venous pressure did not change significantly in either group. Mean PAP increased during spontaneous ventilation in all of the four patients of group B in whom it was measured but left atrial pressure did not. There were no data mentioned in the study regarding group A and mean PAP and left atrial pressure. Pulmonary vascular resistance, which was measured only in two patients of group A and four patients of group B, increased in both groups.
Several of the cardiac patients in group B, those with decreased cardiac output during SV, showed clinical signs of early shock with decreased oxygen consumption, dyspnea, restlessness, apprehension, diaphoresis, and increased respiratory frequency and heart rate. The patients with these symptoms did not show the decreased partial pressure of oxygen (pO₂) which is consistent with pulmonary shunting. Therefore it was believed that the reduction in oxygen availability at the tissue level was the cause. Fifteen of the 18 patients who responded with a decreased cardiac output had undergone cardiac surgery. There was a somewhat higher baseline central venous pressure in group B that was probably a sign of heart failure. Also the patients in group B showed decreased oxygen consumption that may have been an early sign of shock. The authors commented that observation of chest roentgenograms of patients demonstrated that the heart appears to be larger during SV than during IPPV. The authors suggested that additional dilatation of an already large heart may result in a decreased cardiac output when venous return and ventricular filling pressure increased following the switch from IPPV to SV. The study concluded with the statement by the authors that discontinuance of mechanical ventilation may sometimes cause an unexpected decrease in cardiac output in absence of significant deterioration of arterial blood gas values. This change is more likely in patients with limited myocardial function.

Wolff and Gradel (1975) studied hemodynamic responses to discontinuance of IPPV, in 38 post-open heart surgery patients. One hundred per cent oxygen was utilized during both IPPV and spontaneous ventilation. Conversion to spontaneous ventilation resulted in an
increase in right atrial, left atrial, PAP, and aortic pressures and systemic and pulmonary vascular resistance. Cardiac output decreased as well as right and left ventricular work indices. Pulmonary vascular resistance increased substantially more than systemic resistance. Also the increase in pulmonary vascular resistance was associated with little or no change in right ventricular stroke work index while the relatively small increase in systemic vascular resistance was followed by a marked increase in left ventricular stroke work index. The authors suggest two possible mechanisms for their findings. One, that reduction or removal of the increased airway pressure in patients made hypovolemic following open heart surgery, resulted in a marked shift of blood volume from the systemic to the pulmonary circulation. During mechanical ventilation 48 hours preextubation; left atrial pressure was 22.9 torr, right atrial pressure 13.4 torr. Since the pulmonary vasculature was probably functioning on the top portion of its compliance curve, addition of blood to the central blood volume resulted in a decreased compliance, rise in ventricular stroke work, and increased right ventricular oxygen consumption. On the other hand, a small increase in systemic vascular resistance resulted in decreased left ventricular stroke work index and a rise in left atrial pressure suggestive of decreased left ventricular contractility and decreased ventricular oxygen consumption. The authors proposed that under the conditions studied, i.e., following open heart surgery with some endocardial ischemia, small changes in coronary blood flow are critical for appropriate ventricular function. It is likely that with increased right ventricular afterload, right ventricular stroke work index and
right ventricular oxygen consumption, the increased blood flow to the right ventricle may compromise flow to the left, particularly if global flow is unchanged. This decreased flow to the left ventricle may result in decreased contractility, rise of left atrial pressure and decreased stroke volume. Thus, the magnitude of the blood volume readjustment attendant upon a reduction in impedance to venous return magnifies the discrepancy in right versus left ventricular function. Therefore myocardial failure becomes more evident and weaning from mechanical ventilation is not possible.

Downs and Douglas (1980) measured transmural cardiac filling pressures on 10 mongrel dogs during SV, IPPV, and CPPV with a level of 20 cm PEEP before and after near drowning. Room air was utilized during all modes of ventilation. Left ventricular end diastolic pressure decreased significantly on conversion from SV to IPPV; CPPV at 20 cm PEEP increased left ventricular end diastolic pressure to control (SV) values. Left ventricular filling pressures decreased significantly during IPPV compared to SV and further decreased during CPPV before and during near drowning. There was no change in cardiac output, pulmonary vascular resistance, or wedge pressure on conversion from SV to IPPV. However, cardiac output decreased with CPPV before and after near drowning; PVR increased during CPPV before and increased even more after near drowning, and wedge pressure doubled during CPPV and increased further after near drowning CPPV. Wedge pressure and left ventricular end diastolic pressure did not differ during SV. However a change in wedge pressure in a direction opposite to the change in left ventricular end diastolic pressure occurred after institution
of IPPV and CPPV before and during near drowning. Expiratory pleural pressure did not change between SV and IPPV but increased significantly during CPPV before and during near drowning. Mechanical ventilation and near drowning injury caused a significant discrepancy between left ventricular end diastolic pressure and wedge pressure. The researchers suggested that this occurred since more airway pressure was transmitted to the pulmonary artery than the intrapleural space. With momentary interruption of CPPV during near drowning, pleural pressure, right atrial pressure, and PVR decreased suddenly. With increased flow to the left heart left ventricular end diastolic pressure decreased and then increased with each heart beat. Symptoms of pulmonary edema occurred during this temporary disconnection. The authors suggested that after removal of CPPV, the increase in pulmonary blood flow through the damaged pulmonary vasculature caused pulmonary edema fluid to accumulate rapidly in every animal with pulmonary damage.

Trichet and associates (1975) studied the effects of mechanical ventilation with and without PEEP on hemodynamic performance in 10 patients following open heart surgery. Five patients had an aortic valve replacement and the absence of pulmonary vascular disease; the remaining five patients were post-mitral valve replacement and had pulmonary vascular disease. Ten cm PEEP caused a decrease in cardiac index in both groups, but less in the mitral valve group. Cardiac index returned to control levels one hour after removal of PEEP in the aortic valve patients; however, the patients with mitral valve replacement had persistently low cardiac indices. The authors concluded that with increasing severity of pulmonary vascular disease, changes in
airway pressure will have an unpredictable effect on cardiac index unless the level of myocardial competence is taken into account. The authors made several predictions from their study. In patients with acute respiratory failure if the pulmonary vascular resistance is high, right ventricular performance is likely to be impaired. Also, the directional change in cardiac output during PEEP mechanical ventilation will depend on the patient's blood volume, state of myocardial contractility, and the effect of the altered ventilatory pattern on right and left ventricle pre- and arterload. If airway pressure is reduced or if weaning from the ventilator is attempted in the presence of right ventricular failure, combined with a state of hypervolumia, the extent of right ventricular failure may be accentuated and a paradoxical decrease in cardiac output will be in evidence.

McIntyre and associates (1969) studied five patients in severe respiratory failure on 0 and 5 cm PEEP. They did not find a change in cardiac output when the PEEP was discontinued. Three of these patients were thought to have reduced cardiac reserves. The authors offered no explanation for these findings.

Static Effective Compliance

Bone (1976) measured static effective compliance on 25 intubated subjects being ventilated via volume ventilators. He measured compliance by occluding the expiratory line of the ventilator for greater than 2.5 seconds to obtain a plateau pressure. He recorded exhaled volume minus compressible volume and divided this value by the plateau pressure minus end-expiratory pressure to obtain compliance.
Two types of error may result in compliance measurements of patients on ventilators if the respiratory muscles are not relaxed. If the subject is actively inspiring, the pressures developed by the ventilator to inflate the respiratory system will be less than the total pressure required. If the subject is resisting mechanical ventilation, the total pressure developed by the ventilator will be in excess of that required to inflate the relaxed system. Bone also states that most subjects receiving continuous mechanical ventilation become accustomed to the ventilator rapidly and do not actively control respiration.

In a later communication Bone (1978) stated that he made preliminary observations of the correlation between pulmonary compliance and static effective compliance and that changes in effective compliance correlated well with changes in pulmonary compliance.

Grimby, Hedenstierna, and Lofstrom (1975) calculated lung mechanics during mechanical ventilation via volume ventilator on six anesthetized intubated supine subjects considered free of respiratory disease on the basis of clinical examination and chest radiograph. They measured total thoracic compliance at three tidal volumes: 386 cc, 722 cc, and 1126 cc. Total thoracic compliance (Ctot) results in milliliters per centimeters of water were 44.8 ± 4.4, 56.2 ± 6.3, and 56.5 ± 5.7 respectively.

Ayres, Kozam, and Lukas (1963) measured total thoracic compliance on six normal subjects, six subjects with emphysema and/or asthma, and five subjects with fibrotic lung disease. Subjects sat in a chair and breathed through a rubber mouthpiece attached to an intermittent positive pressure breathing machine. Total thoracic
compliance measurements were taken when complete relaxation of the subjects occurred at tidal volumes of 0.5 to 1 liter. Total thoracic compliance for normals ranged from 72 to 110 ml per cm, for subjects with obstructive lung disease the range went from 47 to 77 ml per cm, and for subjects with fibrotic lung disease the range was from 33 to 64 ml per cm.

Suter, Fairley, and Isenberg (1978) measured total effective static compliance on nine subjects with acute pulmonary parenchymal disease and who were without abdominal distension. All subjects were intubated and being ventilated with a volume controlled ventilator. Total effective static compliance measurements were the following: 25 ± 3 ml per cm at 300 cc, 29 ± 3. ml per cm at 500 cc, 33 ± 4 ml per cm at 700 cc, 33 ± 5 ml per cm at 850 cc, and 30 ± 4 ml per cm at 1000 cc.

Chapin and associates (1979) quantified the effect of compliance changes on the transmission of airway pressure to the intrapleural space on 10 anesthetized swine. The swine were intubated and placed on a constant volume ventilator. Control measurements of static lung compliance, thoracic compliance, total respiratory compliance, and airway and intrapleural pressures were made while the swine were being ventilated. The measurements were studied during the following five experimental conditions: (1) pneumatic binders were placed around the abdomen and thorax to decrease thoracic compliance, (2) hydrochloric acid was injected into the lungs to decrease lung compliance, (3) a combination of (1) and (2) to decrease thoracic and lung compliance, (4) sternotomy and multiple parasternal rib fractures to increase thoracic
compliance, and (5) combination of (4) and (2) to increase thoracic and
decrease lung compliance. During control conditions (normal lung and
thoracic compliance) approximately half (52 ± 9%) of the applied airway
pressure was transmitted to the intrapleural space. This latter event
was an expected normal outcome. Transmission of airway pressure to the
intrapleural space increased to 65 ± 9% (P < .02) when lung compliance
was normal and thoracic compliance decreased. When thoracic compliance
was increased and lung compliance decreased there was less transmission
of airway pressure to the intrapleural space (P < .001). Transmission
of airway pressure was reduced to 11 ± 6% when lung compliance decreased
and thoracic compliance increased. The reduction of lung compliance
with normal thoracic compliance resulted in a significant decrease in
the fractional transmission of airway pressure to the intrapleural
space. The authors concluded that lung conditions which are associated
with a decrease in lung compliance or increase in thoracic compliance
will tend to minimize the transmission of airway pressure to the intrapleural
space. Conversely, factors that may decrease thoracic compliance
or increase lung compliance will increase airway pressure transmission
to the intrapleural space. The authors also warn that the interpretation
of intravascular pressures could be influenced by the conditions
of airway pressure, lung compliance, and thoracic compliance.

In 1972, Hutch and Murray studied the effects of continuous
positive pressure ventilation at 0, 5, and 10 cm PEEP on systemic
oxygen and peripheral tissue oxygenation. They studied 19 patients who
were divided into three groups based on clinical criteria (history,
physical exam, chest X-ray), arterial blood gases, and mechanical
properties of the lung (peak airway pressure required to produce a
tidal volume of 10-16 ml per kg). The three groups were normal, stiff
thorax syndrome, and chronic obstructive pulmonary disease. The authors
found that cardiac index fell by nearly the same percentage in every
group of patients as PEEP was added. At 10 cm PEEP the decrease in
cardiac index was statistically significant in all three groups. At
5 cm PEEP it was significant in only the normal group. The authors
related the fall in cardiac output to the change in mean intrapleural
pressure. The authors postulated that two conditions may cause a
decrease in cardiac output due to impairment of venous return: (1)
with normal or stiff lungs ventilated with large tidal volumes from an
initially low functional residual capacity and (2) a patient whose
functional residual capacity is increased by the application of
continuous positive pressure ventilation.

Clinical Measurement of Pulmonary Artery
and Wedge Pressures

Berryhill, Benumoff, and Rauscher (1978) simultaneously recorded
airway and pulmonary vascular pressures on 10 critically ill patients
receiving mechanical ventilation with IMV and PEEP, 3 to 10 cm.
Simultaneous measurements of airway and vascular pressures were made
in order to establish the end-exhalation point on the vascular pressure
tracing. It is especially difficult to identify the end-exhalation point
when patients have rapid wide swings in pleural and airway pressures.
The authors recommend that measurements be taken at end-exhalation.
Because there is no airflow at end-exhalation, the intrapleural pressure
is considered a static influence upon pulmonary hemodynamics. The
authors also pointed out in their article the unreliability of digital panel meter printout. The digital printout is derived from data selected randomly every two seconds from the pressure pattern on the oscilloscope and thus is influenced by both the rate and mode of ventilation, whether positive pressure or spontaneous. There is also a time delay in the mean or venous mode (6.9 ± 0.1 seconds, Hewlett Packard 78205B) necessary for the integration process. This delay makes it impossible to visually synchronize the appropriate digital value readout of the pressure with the patient's ventilatory pattern.

Maran (1980) concurred with Berryhill et al. (1978) that digital output systems have no advantage over graphic system. Maran advocates monitoring of pulmonary vascular pressures with an analog (strip chart) recording system, since it provides a more accurate graphic measurement of pulmonary artery pressures correlated to intrathoracic pressures at end-exhalation.

Gooding and Laws (1977) demonstrated the differences in wedge pressure during various modes of positive pressure ventilation and SV. The authors advocated measurement of wedge pressure during end-exhalation at ambient pressures even when the patient is ventilated with PEEP. Gooding and Laws also state that a more accurate reflection of true heart filling pressures is obtained when the difference between recorded wedge and intrapleural pressure (transmural wedge pressure) is calculated.

Hoyt et al. (1980) compared end-exhalation values of pulmonary artery systolic, diastolic, and wedge pressures taken from the bedside monitor display using Hewlett Packard modules 78205B and C to values
derived from a strip chart recorder. The authors found clinically significant errors in digital readout of PAS pressures for patients during mechanical ventilation (IMV or IPPV) and in PAD pressure for patients during IMV and SV.
CHAPTER 3

METHODOLOGY

In this chapter the research design, sample selection, measurement description, methodology, and methods for analyzing data are presented.

Research Design

This was a descriptive study designed to identify the effects of positive pressure ventilation and static effective lung compliance on the measurements obtained from a balloon tipped flow directed pulmonary artery catheter (FDPAC).

Sample

Fourteen subjects from the intensive care units of a Southwestern university hospital participated in the study. Criteria for subject selection were that the subjects:

1. Were tracheally intubated and receiving mechanical ventilation via a volume ventilator.
2. Were hemodynamically stable having maintained their normal blood pressure and heart rate for at least three hours prior to participation in the study.
3. Had a FDPAC in place.
4. Were able to tolerate lying in the flat supine position.
5. Could breathe spontaneously off the ventilator for one to two minutes.

Persons who met the above criteria had the purpose and nature of the study explained to them or to an immediate family member and were asked if they were willing to participate. Signed consents were obtained from subjects or their legal guardian and from their physicians (Appendices A and B). Assurance was given to subjects that they were free to withdraw from the study at any time without affecting either their relationship with any doctor or nurse, or the quality of their treatment or care. Confidentiality was assured by assigning a number to each subject.

The research proposal, Subjects Consent Form (Appendix A), and Physician's Consent Form (Appendix B) was submitted to the University of Arizona Human Subjects Committee for approval. A letter of their approval can be seen in Appendix C.

**Measurements**

Flow Directed Pulmonary Artery Catheter (FDPAC) Measurements

Pulmonary artery systolic, diastolic, and wedge pressures were obtained using a pressure transducer (Statham #6034) connected to a waveform (Hewlett-Packard #7803), digital monitor (Hewlett-Packard #78205B, 78201B, 78901A) and waveform strip chart (Heath EU-205-11). Initially the pressure transducer was calibrated, zeroed, and positioned at the level of the phlebostatic axis (Winsor and Burch, 1945) on the
patient's flat supine chest. The phlebostatic axis is approximately located at the fourth intercostal space, mid-axillary line.

Pulmonary artery systolic (PAS) and PAD pressures were recorded at the end-exhalation portion of the respiratory cycle. The balloon of the FDPAC was then inflated slowly with 1 to 1.5 cc of air until the PAP waveform dampened and the characteristic pulmonary capillary wedge pressure (PCWP) oscillations were recorded at end-exhalation. After the PCWP was recorded the balloon was deflated and the pressure tracing observed until return to the PAP waveform. The pressure for each hemodynamic measurement (PAS, PAD, and PCWP) was recorded as the average of three consecutive end-expiratory points taken from the mid portion of the total recording period.

Static Effective Compliance

Static measurements of pressure and volume were recorded at consecutive ventilator volumes of approximately 8, 10, 12, and 14 cc per kg. A spirometer was calibrated before use with a standard volume syringe. The patients' exhaled volume was measured via the spirometer connected to the ventilator exhalation port to measure the exact volume exhaled by the patient. A ventilator-tubing compliance factor of 3 cc of volume per cm of water of system pressure was subtracted from the exhaled volume value. The ventilator was set to the desired tidal volume, a breath was given, and the expiratory flow vent occluded by clamping the exhalation valve tubing for three to five seconds to validate the airway pressure reading as a true static pressure. After three to five seconds the exhaled volume and plateau
pressure was recorded and the exhalation tubing unclamped to allow the patient to exhale. The airway plateau pressure for each volume was recorded in the same manner. When PEEP was used the level of the expiratory pressure plateau was subtracted from the airway plateau pressure at end-inspiration to obtain the change in pressure that produced the volume change.

A static pressure/volume curve was constructed for each subject. The effective static compliance for each volume was calculated by dividing the exhaled volume minus compressible volume by plateau pressure minus the level of end-expiratory pressure.

Ventilatory Parameters

The investigator recorded the subject's exhaled tidal volume, ventilatory rate, ventilatory mode, and peak airway pressure during mechanical ventilator breaths. The level of PEEP and oxygen concentration was recorded.

Other Clinical Parameters

The investigator also collected additional data concerning the subjects' weight, diagnosis, presence of previously diagnosed lung disease, heart disease, current medications, last chest roentegram, most current arterial blood gases, blood pressure, heart rate and rhythm, and number of hours on mechanical ventilation and number of hours with FDPAC in place. This information was recorded on the Data Collection sheet in Appendix D.
Protocol

Subjects were randomly assigned to one of two measurement sequences. The two measurement sequences were:

A

1. FDPAC measurements on the ventilator
2. Static effective compliance
3. Five minutes of prescribed rate, mode, and volume
4. FDPAC measurements off the ventilator

B

1. FDPAC measurements off the ventilator
2. Five minutes of prescribed rate, mode, and volume
3. FDPAC measurements on the ventilator
4. Static effective compliance

Subjects' respiratory rates were controlled on the ventilator for two to three minutes while FDPAC measurements were taken. Immediately after FDPAC measurements were taken on the ventilator, the subjects remained on controlled ventilation approximately two to three more minutes while static effective compliance measurements were taken. Subjects breathed spontaneously off the ventilator for one to two minutes while FDPAC measurements were taken.

FDPAC Measurements On the Ventilator

Initially the transducer was calibrated to atmospheric pressure at the level of the phlebostatic axis in the flat supine patient. The fraction of inspired oxygen (FIO₂) remained constant at the prescribed concentration. If the subject was breathing spontaneously on an IMV ventilatory mode, the mode was changed to control for eight to 20
ventilator breaths. When the patient was totally controlled by the ventilator without his/her own spontaneous respirations FDPAC measurements were recorded from the tracing. The PAS pressure was recorded as the average of three consecutive end-expiratory points taken from the mid-portion of the total recording period. The average PAS was recorded on the data collection sheet. The same procedure was performed for PAD. To obtain PCWP the FDPAC balloon was inflated with 1 to 1.5 cc of air until the waveform showed a wedge configuration. The PCWP was recorded as the average of three consecutive end-expiratory points taken from the mid-portion of the total recording period. The average PCWP was recorded on the data collection sheet.

If at any time during the measurements, the subject experienced ventricular arrythmias in excess of those previously present or shortness of breath, the ventilator was to be returned to the prescribed rate until the subject was asymptomatic. No subject experienced any of the latter effects.

FDPAC Measurements Off the Ventilator

The ventilator rate was decreased to zero and the patient's ventilatory pattern observed. The fraction of inspired oxygen remained constant at the prescribed concentration and in the patients receiving PEEP, the same expiratory pressure plateau was maintained. Flow directed pulmonary artery catheter (FDPAC) measurements were recorded as previously done on the ventilator. Immediately after the measurements were taken the ventilator rate was increased to the prescribed rate.
If at any time during this part of the study the subject experienced ventricular arrhythmias in excess of those previously present or shortness of breath, the ventilator was to be returned to the prescribed rate until the subject was asymptomatic. No subject experienced any of the latter effects.

Static Effective Compliance

The fraction of inspired oxygen remained at the prescribed concentration during this procedure. Immediately after FDPAC measurements were taken on the ventilator, the effective static compliance was measured at approximate tidal volumes of 8, 10, 12, and 14 cc per kg for one to three breaths at each volume on the ventilator. Immediately after measurements were taken the patient was placed upon the prescribed ventilatory rate and mode. If at any time during this part of the study the ventilator airway pressure increased greater than 15 cm for any volume change the volume would have been lowered to the prescribed volume. Also if during any time the subject experienced any shortness of breath or ventricular arrhythmias in excess of those previously present, the ventilator tidal volume would have been returned to the prescribed rate and the subject allowed to rest till she/he was asymptomatic. No subject experienced any of the latter effects.

Analysis of Data

Analysis of the data was accomplished utilizing the following methods. Paried two-tailed t tests (Colton, 1974) were performed on PCWP, PAS, and PAD pressures measured off and on the ventilator to determine if significant differences were present. Pearsons correlation
coefficients (Colton, 1974) were calculated between PCWP and PAD pressures on and off the ventilator to determine if a significant relationship existed between these two measurements.

One way analysis of variance (Colton, 1974) was performed to determine if there was a significant relationship between the amount of change in pressures (PCWP, PAS, and PAD) and each of three variables. The variables were: smoking history, sequence of measurement order, and the presence of PEEP. Pearson's correlation coefficients (Colton, 1974) were calculated to determine any relationship between static effective compliance measurements and each of the following variables: changes in PAS pressure, PAD pressure and PCWP on and off the ventilator, and the level of peak airway pressure.
CHAPTER 4

PRESENTATION AND ANALYSIS OF DATA

The purpose of this chapter is to present the characteristics of the sample and to present and analyze the data obtained with hemodynamic monitoring and static effective compliance determinations.

Characteristics of the Sample

The sample was selected from patients in the medical-surgical intensive care units of a Southwestern university hospital. Fourteen adults, four females and 10 males, ranging in age from 44 to 76 years with a mean age of 62.4 years, who met the criteria of the study established in Chapter 3 were studied. Seven subjects had undergone open heart surgery, two subjects had a myocardial infarction, and five subjects had undergone major abdominal surgery. The subjects received a variety of medications (Dopamine, Nitroprusside, Lasix, Aminophylline, etc.) at the time of the study. Table 1 lists the subjects' age, sex, major diagnoses, and drugs being administered at the time of the study. All fourteen subjects had a balloon tipped flow directed pulmonary artery catheter (FDPAC) in acceptable position at the time of the study.

Nine subjects had a positive smoking history ranging from 26 pack years to 100 pack years with a mean of 62 pack years. Two of these same subjects had a history of chronic obstructive pulmonary disease. The chest radiographs of three subjects (#3, #6, and #8) showed no
Table 1. Characteristics of the Sample Including Age, Sex, Major Diagnoses, and Medications

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Age</th>
<th>Sex</th>
<th>Major Diagnoses</th>
<th>Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44</td>
<td>M</td>
<td>Choledocojejunostomy and drainage of abcess</td>
<td>Cephadyl, Gentamycin, Dopamine</td>
</tr>
<tr>
<td>2</td>
<td>72</td>
<td>M</td>
<td>Myocardial infarction status post cardiac arrest</td>
<td>Nitroprusside, Aminophylline</td>
</tr>
<tr>
<td>3</td>
<td>57</td>
<td>F</td>
<td>Mitral Valve Replacement</td>
<td>Dopamine, Lidocaine, Cephapirim</td>
</tr>
<tr>
<td>4</td>
<td>70</td>
<td>M</td>
<td>Aortic Commissurotomy, Mitral Valve Replacement</td>
<td>Epinephrine, Nitroprusside, Dopamine, Digoxin</td>
</tr>
<tr>
<td>5</td>
<td>69</td>
<td>M</td>
<td>Aortic Valve Replacement, Mitral Valve Replacement, Single Coronary Artery Bypass</td>
<td>Nitroprusside, Dopamine, Lasix, Cephapirim</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>F</td>
<td>Mitral Valve Replacement</td>
<td>Nitroprusside, Digoxin, Lasix, Cephapirim</td>
</tr>
<tr>
<td>7</td>
<td>70</td>
<td>M</td>
<td>Aortic and Mitral Valve Replacement, Left Ventricle Aneuresectomy, Single Coronary Artery Bypass</td>
<td>Nitroprusside, Dopamine, Digoxin, Lasix</td>
</tr>
<tr>
<td>8</td>
<td>55</td>
<td>M</td>
<td>Myocardial Infarction (anterior-septal)</td>
<td>Lidocaine, Digoxin</td>
</tr>
<tr>
<td>9</td>
<td>62</td>
<td>M</td>
<td>Right leg amputation Sepsis</td>
<td>Penicillin, Cleocin, Cimetidine, Digoxin, Gentamycin, Terbutaline, Solu-Cortef.</td>
</tr>
<tr>
<td>10</td>
<td>48</td>
<td>M</td>
<td>Exploratory laporotomy, Small bowel resection, ileostomy, Bacterial pericarditis</td>
<td>Nafcillin</td>
</tr>
<tr>
<td>Subject #</td>
<td>Age</td>
<td>Sex</td>
<td>Major Diagnoses</td>
<td>Medications</td>
</tr>
<tr>
<td>----------</td>
<td>-----</td>
<td>-----</td>
<td>-----------------------------------------------------</td>
<td>----------------------</td>
</tr>
<tr>
<td>11</td>
<td>64</td>
<td>F</td>
<td>Abdominal Aortic Aneurysm Repair, Chronic Renal Failure</td>
<td>Cimetidine, Amphofel, Vancomycin</td>
</tr>
<tr>
<td>12</td>
<td>66</td>
<td>M</td>
<td>Triple Coronary Artery Bypass Left Ventrical Aneurysm repair</td>
<td>Cephapirin</td>
</tr>
<tr>
<td>13</td>
<td>61</td>
<td>M</td>
<td>Triple Coronary Artery Bypass</td>
<td>Dopamine, Lidocaine, Nitroprusside, Cephapirin</td>
</tr>
<tr>
<td>14</td>
<td>76</td>
<td>F</td>
<td>Abdominal Aortic Aneurysm repair, Intraoperative myocardial infarction</td>
<td>Nitroprusside, Digoxin, Cephapirin</td>
</tr>
</tbody>
</table>

Table 1.—Continued
evidence of active disease at the time of the study, all other subjects had some type of abnormality (effusion, infiltrate, etc.) at the time of the study. Table 2 lists the pulmonary history and chest radiographic data for each subject.

Two subjects had a negative preoperative cardiac history (#1 and #9). Although subject 9 had a negative preoperative history, postoperatively he had an acute episode of supraventricular tachycardia which responded to digitalization (digitalis was subsequently discontinued). Two subjects (#2 and #11) had a long history of systemic hypertension. Five subjects had severe valvular disease and two of these had severe coronary artery disease as well; two other patients had severe coronary artery disease and one other had bacterial pericarditis. One patient (#14) had an intraoperative inferior myocardial infarction and two patients (#2 and #8) had myocardial infarctions.

Three subjects (#2, #7, and #10) expired prior to leaving the intensive care unit, two days to three weeks poststudy. Of the remaining 11 subjects, #12 had to be reintubated approximately 12 hours after being studied, secondary to acute respiratory distress probably due to pulmonary edema. All other subjects proceeded through weaning and extubation without additional problems.

All subjects were ventilated with volume set ventilators. The ventilator delivered tidal volumes ranged from 800 to 1000 cc with a mean of 932 cc, or 9 to 17 cc per kg. All subjects were on IMV. The IMV rates ranged from two to eight breaths per minute with a mean of five breaths per minute. The fraction of inspired oxygen (FIO₂) for
<table>
<thead>
<tr>
<th>Subject #</th>
<th>Pulmonary History</th>
<th>Chest Radiograph</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26 pack years</td>
<td>Bilateral pleural effusions</td>
</tr>
<tr>
<td>2</td>
<td>Chronic obstructive pulmonary disease</td>
<td>Clear of bilateral alveolar infiltrate</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>Clear</td>
</tr>
<tr>
<td>4</td>
<td>Chronic obstructive pulmonary disease</td>
<td>Left sided density</td>
</tr>
<tr>
<td>5</td>
<td>None</td>
<td>Bilateral infiltrates</td>
</tr>
<tr>
<td>6</td>
<td>None</td>
<td>Clear</td>
</tr>
<tr>
<td>7</td>
<td>100 pack years</td>
<td>Minimal discoid atelectasis both bases</td>
</tr>
<tr>
<td>8</td>
<td>60 pack years</td>
<td>Clear</td>
</tr>
<tr>
<td>9</td>
<td>40 pack years</td>
<td>Diffuse infiltrate on left with pleural effusion</td>
</tr>
<tr>
<td>10</td>
<td>90 pack years</td>
<td>Increased density left lung field, bilateral pleural effusions or pulmonary edema</td>
</tr>
<tr>
<td>11</td>
<td>30 pack years</td>
<td>Bilateral pleural parenchymal changes, Atelectatic changes and infiltrates in mid and lower lungs on right and left</td>
</tr>
<tr>
<td>12</td>
<td>100 pack years</td>
<td>Interstitial pulmonary edema, with some alveolar component of the disease in bases</td>
</tr>
<tr>
<td>13</td>
<td>80 pack years</td>
<td>Minimal atelectasis left base</td>
</tr>
<tr>
<td>14</td>
<td>40 pack years</td>
<td>Marked pulmonary edema</td>
</tr>
</tbody>
</table>
all subjects ranged from 0.35 to 0.6 with a mean of 0.4. The FIO$_2$ for each subject remained constant throughout the study. Eleven of the 14 subjects were ventilated with 5 cm PEEP during IMV and during spontaneous breaths between IMV (CPAP). Three of the 14 subjects (#2, #8, and #10) were ventilated without PEEP. When measurements were taken on the ventilator the patients' respirations were totally controlled by the ventilator. The ventilator delivered tidal volume, tidal volume expressed in cc per kg, level of PEEP, FIO$_2$, peak airway pressure, ventilator rate, and patient's respiratory rate while measurements were taken on and off the ventilator are listed in Table 3.

Prior to the study the subjects had been ventilated mechanically from 15 to 96 hours with a mean time of 45.5 hours. The subjects had a FDPAC in place from 15 to 108 hours with a mean time of 47.5 hours.

**Statistical Analysis**

**Pulmonary Artery Systolic Pressure**

For all 14 subjects pulmonary artery systolic (PAS) pressures measured on the ventilator ranged from 22 to 50.5 torr with a mean of 34.6 torr. Pulmonary artery systolic pressures measured off the ventilator ranged from 24 to 57 torr with a mean of 39.7 torr. The differences in pulmonary artery systolic pressures, pressures measured off minus pressures measured on the ventilator, ranged from +.5 to +10.5 with a mean difference of +5.5 torr. Individual PAS pressures on and off the ventilator for all subjects are presented in Appendix E. Individual PAS differences on and off the ventilator for all subjects are listed in Table 4.
Table 3. Ventilatory Status of Each Subject — Fraction of Inspired Oxygen (FI02), Ventilator Rate (VR), Subject's respiratory rate (SR), Tidal volume (TV) in cubic centimeters, cubic centimeters per kilogram of body weight, PEEP in centimeters of water, and level of peak airway pressure (AWP) in cubic centimeters of water.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Ventilatory Status on Ventilator</th>
<th>Off Ventilator</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FIO2  VR TV/cc per kg PEEP AWP</td>
<td>FIO2 SR PEEP</td>
</tr>
<tr>
<td>1</td>
<td>.35  14 1000/13.8 5 28</td>
<td>.35 20 5</td>
</tr>
<tr>
<td>2</td>
<td>.40  20 900/10.9 0 28</td>
<td>.40 40 0</td>
</tr>
<tr>
<td>3</td>
<td>.40  8 1000/14.9 5 38</td>
<td>.40 8 5</td>
</tr>
<tr>
<td>4</td>
<td>.50  20 1000/13.2 5 28</td>
<td>.50 32 5</td>
</tr>
<tr>
<td>5</td>
<td>.50  12 900/13.8 5 25</td>
<td>.50 21 5</td>
</tr>
<tr>
<td>6</td>
<td>.40  14 900/12.3 5 34</td>
<td>.40 16 5</td>
</tr>
<tr>
<td>7</td>
<td>.40  10 900/13.6 5 38</td>
<td>.40 24 5</td>
</tr>
<tr>
<td>8</td>
<td>.40  12 900/10.3 0 30</td>
<td>.40 32 0</td>
</tr>
<tr>
<td>9</td>
<td>.40  12 1000/17.2 5 42</td>
<td>.40 18 5</td>
</tr>
<tr>
<td>10</td>
<td>.40  14 900/9 0 24</td>
<td>.40 16 0</td>
</tr>
<tr>
<td>11</td>
<td>.40  14 850/12.3 5 28</td>
<td>.40 22 5</td>
</tr>
<tr>
<td>12</td>
<td>.60  17 900/918 5 30</td>
<td>.60 24 5</td>
</tr>
<tr>
<td>13</td>
<td>.50  12 1000/14.7 5 28</td>
<td>.50 16 5</td>
</tr>
<tr>
<td>14</td>
<td>.40  16 800/11.9 5 26</td>
<td>.40 22 5</td>
</tr>
</tbody>
</table>
### Table 4. Individual Differences, Spontaneous Breathing Minus Mechanical Ventilation, for Pulmonary Artery Systolic, Diastolic, and Wedge Pressures (torr)

<table>
<thead>
<tr>
<th>Subject</th>
<th>PAS</th>
<th>PAD</th>
<th>PCWP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+1</td>
<td>+1.5</td>
<td>+2</td>
</tr>
<tr>
<td>2</td>
<td>+5.5</td>
<td>+3.5</td>
<td>+8.5</td>
</tr>
<tr>
<td>3</td>
<td>+4</td>
<td>+1.5</td>
<td>not obtained</td>
</tr>
<tr>
<td>4</td>
<td>+7</td>
<td>+3</td>
<td>+5</td>
</tr>
<tr>
<td>5</td>
<td>+4.5</td>
<td>+3</td>
<td>-5</td>
</tr>
<tr>
<td>6</td>
<td>+10.5</td>
<td>+6.5</td>
<td>+3.5</td>
</tr>
<tr>
<td>7</td>
<td>+8</td>
<td>+4</td>
<td>+5</td>
</tr>
<tr>
<td>8</td>
<td>+7</td>
<td>+2</td>
<td>not obtained</td>
</tr>
<tr>
<td>9</td>
<td>+4</td>
<td>+3.5</td>
<td>-1</td>
</tr>
<tr>
<td>10</td>
<td>+0.5</td>
<td>+1.5</td>
<td>+13.5</td>
</tr>
<tr>
<td>11</td>
<td>+3.5</td>
<td>+2</td>
<td>+8.5</td>
</tr>
<tr>
<td>12</td>
<td>+9.5</td>
<td>+5.5</td>
<td>+4</td>
</tr>
<tr>
<td>13</td>
<td>+6</td>
<td>+4</td>
<td>+0.5</td>
</tr>
<tr>
<td>14</td>
<td>+6</td>
<td>+4</td>
<td>+12</td>
</tr>
</tbody>
</table>
For subjects receiving 5 cm PEEP (n = 11), PAS pressures on the ventilator ranged from 23 to 50.0 torr with a mean of 35.5 torr. Pulmonary artery systolic pressure measured on 5 cm CPAP ranged from 24 to 57 torr with a mean of 41.3 torr. The differences in PAS pressures on 5 cm CPAP minus pressures on the ventilator with 5 cm PEEP ranged from +1 to +10.5 torr, with a mean difference of +5.7 torr. For those not receiving PEEP (n = 3), PAS pressures measured on the ventilator were 22, 30, and 41.5 torr with a mean of 31.1 torr. Pulmonary artery systolic pressures measured off the ventilator during spontaneous ventilation (SV) were 27.5, 37, and 42 torr with a mean of 35.5 torr. The differences in PAS pressures off minus on the ventilator without PEEP were +.5, +5.5, and +7 torr with a mean difference of +4.3 torr.

A paired two-tailed t test (Colton, 1974) performed on PAS measurements off and on the ventilator showed a significant increase (P < .001) in PAS pressures off versus on the ventilator. A one way analysis of variance (Colton, 1974) performed between the changes in PAS off and on the ventilator and the variables of sequence, presence of PEEP, level of peak airway pressure generated by the ventilator during measurements, and lung disease or smoking history respectively, demonstrated no significant relationships.

Pulmonary Artery Diastolic Pressure

For all 14 subjects, PAD pressures measured on the ventilator ranged from 10 to 30 torr with a mean of 18.9 torr. Pulmonary artery diastolic pressures measured off the ventilator ranged from 13.5 to
33 torr with a mean of 22.2 torr. The differences in PAD pressures, pressures measured off minus pressures measured on the ventilator, ranged from +1.5 to +6.5 torr with a mean difference of +3.3 torr. Individual PAD pressures on and off the ventilator for all subjects are presented in Appendix E. Individual PAD differences on and off the ventilator for all subjects are listed in Table 4.

For subjects receiving 5 cm PEEP (n = 11), PAD pressures on the ventilator ranged from 12 to 27 torr with a mean of 19.5 torr. Pulmonary artery diastolic pressures measured on 5 cm CPAP ranged from 17 to 33 torr with a mean of 23.1 torr. The differences in PAD pressures on 5 cm CPAP minus pressures on the ventilator with 5 cm PEEP ranged from +1.5 to +6.5 torr, with a mean difference of +3.5 torr. For those not receiving PEEP (n = 3), PAD pressures measured on the ventilator were 10, 17, and 22.5 torr with a mean of 16.5 torr. Pulmonary artery diastolic pressures measured off the ventilator without CPAP were 13.5, 19, and 24 torr with a mean of 18.8 torr. The differences in PAD pressures off the ventilator and during spontaneous ventilation were +1.5, +2, and +3.5 torr with a mean difference of +2.3 torr.

A paired two-tailed t test (Colton, 1974) performed on PAD measurements off and on the ventilator showed a significant increase (P < .001) in PAD pressures off versus on the ventilator. A one way analysis of variance (Colton, 1974) performed between the changes in PAD off and on the ventilator and the variables of sequence, presence of PEEP, level of peak airway pressure generated by the ventilator
during measurements, and lung disease or smoking history respectively, demonstrated no significant relationships.

Pulmonary Capillary Wedge Pressure

For 12 subjects, the pulmonary capillary wedge pressure (PCWP) was measured on the ventilator. For two subjects the FDPAC could not be floated into wedge position for the study. Pulmonary capillary wedge pressures for 12 subjects on the ventilator ranged from 6 to 22.5 torr with a mean of 13.6 torr. Pulmonary capillary wedge pressures measured off the ventilator ranged from 12.5 to 28 torr with a mean of 18.1 torr. The differences in PCWP, pressures measured off minus pressures measured on the ventilator, ranged from -5 to +13.5 torr with a mean difference of +4.2 torr. Individual PCWP on and off the ventilator for all subjects are presented in Appendix E. Individual PCWP differences on and off the ventilator for all subjects are listed in Table 4.

For subjects receiving PEEP 5 cm (n = 10), PCWP on the ventilator ranged from 8 to 22.5 torr with a mean of 14.3 torr. Pulmonary capillary wedge pressures measured off the ventilator with 5 cm CPAP, ranged from 12.5 to 23 torr with a mean of 18.6 torr. The differences in PCWP, pressures measured off minus pressures measured on the ventilator, ranged from -5 to +12 torr with a mean difference of +2.9 torr. For those subjects not receiving PEEP (n = 2), PCWP measured on the ventilator were 6 and 14.5 torr, with a mean of 10.2 torr. Pulmonary capillary wedge pressures measured off the ventilator without CPAP were 14.5 and 28 torr with a mean of 21.2 torr. The differences in PCWP measured off the ventilator without PEEP minus
on the ventilator without PEEP were +8.5 and +13.5 torr with a mean difference of +11 torr.

A paired two-tailed t test (Colton, 1974) performed on PCWP off and on the ventilator showed a significant increase ($P < .05$) in PCWP off versus on the ventilator. A one way analysis of variance (Colton, 1974) performed between the changes in PCWP on and off the ventilator and the following variables: sequence, presence of PEEP, level of peak airway pressure generated by the ventilator during measurements, and lung disease or smoking history respectively, demonstrated no significant relationships. Pearsons correlation coefficients (Colton, 1974) calculated between the changes in PCWP and the changes in PAD pressures on and off the ventilator demonstrated that no statistically significant relationship ($R = .4$) existed.

Static Effective Compliance

Effective static lung compliance measurements were performed on all 14 subjects at approximate tidal volumes of 8, 10, 12, and 14 cc per kg immediately after PAP and PCWP were taken on the ventilator. It was impossible to ventilate each subject with an exact tidal volume according to the criteria set (8, 10, 12, and 14 cc per kg). The range of compliance measurements in ml per cm of water at each level of ventilation (cc per kg) are listed in Table 5. Appendix F lists all static effective compliances obtained.

Pressure volume curves, utilizing the ventilator generated end-inspiratory plateau pressure and exhaled volume, were plotted for each subject. Three subjects (#3, #7, and #9) clearly had curves which were
Table 5. Range of Static Effective Compliance (SEC) — Measurements in milliliters per cubic centimeter of water; Ideal Tidal Volume (Vt Ideal) in cubic centimeters of water per kilogram of body weight at which SEC measurements were to be taken; and Tidal Volume (Vt Real) in cubic centimeters of water per kilogram of body weight at which the actual SEC measurements were taken.

<table>
<thead>
<tr>
<th>Vt Ideal</th>
<th>Vt Real</th>
<th>SEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 cc/kg</td>
<td>6.4–8.6 cc/kg</td>
<td>28.66–64.5</td>
</tr>
<tr>
<td>10 cc/kg</td>
<td>8.0–10.1 cc/kg</td>
<td>27.0–73.11</td>
</tr>
<tr>
<td>12 cc/kg</td>
<td>9.9–12.2 cc/kg</td>
<td>23.78–56.61</td>
</tr>
<tr>
<td>14 cc/kg</td>
<td>12.7–14.7 cc/kg</td>
<td>29.75–59.94</td>
</tr>
</tbody>
</table>

shifted to the right. These three subjects also had static effective compliances (23.78 to 36.33 ml per cm of water) which were lower than the other 11 subjects. The remaining 11 subjects had curves which somewhat resembled normal P/V curves. The static effective compliances of these 11 subjects ranged from 33.66 to 73.11 ml per cm of water.

The numerical values for static effective lung compliance at approximately 10 cc per kg were selected for statistical correlation. This part of the curve was selected for statistical analysis because shifts in the curve detected at larger values of tidal volume were usually reflected at this tidal volume. Also this tidal volume was near the midpoint of the determined curves and thus more accurately reflected shifts in the curve on the horizontal axis. One exception was subject
#13. For this subject the third point on the curve (11.1 cc per kg) was a more accurate representation for his particular curve.

Pearson's correlation coefficients (Colton, 1974) calculated between static effective compliance measurements and each of the following variables, changes in PCWP, PAS, and PAD pressures, demonstrated no significant relationship. However, Pearson's correlation coefficients (Colton, 1974) calculated between static effective compliance measurements and the level of peak airway pressure on the ventilator during the measurements demonstrated a significant (R = -.8) negative relationship.
CHAPTER 5

DISCUSSION AND CONCLUSIONS

In this chapter the findings of this study are compared and contrasted with those of other investigators. The study results are discussed and the mechanisms which may have contributed to the findings are suggested. The limitations of the study and implications for health professionals directly responsible for the hemodynamic monitoring of critically ill patients are discussed. In conclusion, recommendations are made for further study.

Pulmonary Artery Systolic and Diastolic Pressures

In the present study PAS pressure during SV with 0 or 5 cm CPAP demonstrated a statistically significant increase ($P < .001$) compared to controlled positive pressure ventilation with 0 or 5 cm PEEP. There was a mean increase of 5.1 torr between pressures during spontaneous as opposed to mechanical ventilation. Pulmonary artery diastolic pressure during SV with 0 or 5 cm PEEP also demonstrated a statistically significant increase ($P < .001$) compared to controlled positive pressure ventilation with 0 or 5 cm PEEP. There was a mean increase of 3.3 torr during SV in contrast to mechanical ventilation.

The findings of this study were in contrast to the study performed by Shinn et al. (1979), where there was a small but significant decrease in PAP when the patients were breathing spontaneously compared to the pressure measured during IPPV. Shinn et al.'s study,
however, did not measure PAP at end-exhalation, but rather averaged all PAS and PAD pressures during one respiratory cycle. This method of measurement would also average in the higher PAP during positive pressure inspiration and perhaps explain why the pressures were higher during mechanical ventilation.

Askitopoulou et al. (1978) found in their study of 11 patients post-cardiac valve repair and coronary artery bypass surgery, that mean PAP was consistently greater during CPAP 10 cm and SV than during IPPV. However, the differences were not significant because relatively few measurements were made. Civetta et al. (1972) also demonstrated that mean PAP increased slightly during SV and CPAP 10 cm as compared to mechanical ventilation with zero and 10 cm PEEP. The investigators presented data on only two patients with adult respiratory distress syndrome secondary to acute trauma. Downs et al. (1977) found that mean transmural PAP decreased significantly in 10 postoperative coronary artery bypass patients when IPPV was initiated compared to IMV (rate of two) with zero PEEP, IMV (two) with 5 cm PEEP and IMV (two) with 10 PEEP. The authors measured pleural pressure from an intra-pleural catheter and subtracted expiratory Ppl from mean PAP to obtain the measurement. Beach et al. (1973) measured mean PAP in four patients whose cardiac output decreased when they were changed from IPPV to SV. The researchers found an increase in PAP during SV in all four patients. Wolff and Gradel (1975) studied 38 post-open heart surgery patients and also found an increase in mean PAP on conversion from IPPV to SV. Mean PAP increased an average of 5 to 6 torr. This increase in mean PAP is in accord with the findings of the present study.
which showed an average increase of 5 torr in PAS pressure and 3.3 torr increase in PAD pressure during conversion from mechanical to spontaneous ventilation.

**Pulmonary Capillary Wedge Pressure**

In the present study PCWP during SV with 0 or 5 cm CPAP demonstrated a statistically significant increase ($P < .05$) compared to controlled positive pressure ventilation with 0 or 5 cm PEEP. There was a mean increase of 4.5 torr between PCWP during SV compared to mechanical ventilation. Only one subject (#5) in the present study showed a decrease in wedge pressure on change from CPPV to CPAP.

These findings are in contrast to the studies of Grenvik (1966) who found no change in mean left atrial pressure (LAP) on nine patients on change from IPPV to SV. Davison et al. (1978) also found no significant difference in wedge pressures on and off CPPV (PEEP up to 10 cm); however, patients were sedated without spontaneous ventilatory activity during the measurements. Davison et al. also found a delayed progressive increase in wedge pressure in six patients with the change from mechanical to spontaneous ventilation; these patients were those who had underlying heart disease. Shinn et al. (1979) found no significant change in wedge pressure on conversion from IPPV to SV in 18 patients. Again, Shinn et al. used a mean of all pressures during one respiratory cycle to determine this measurement. Kennedy et al. (1977) also found no significant change in mean LAP or wedge pressure in seven patients during weaning from IPPV to SV. Downs and Douglas (1980) found no change in cardiac output, pulmonary
vascular resistance, or transmural wedge pressure in 10 healthy mongrel dogs ventilated on IPPV and breathing spontaneously. Transmural left ventricular end diastolic pressure decreased significantly on conversion from SV to IPPV. However on 20 cm PEEP CPPV before and during near drowning cardiac output decreased and pulmonary vascular resistance and wedge pressure increased significantly. Wedge and left ventricular end diastolic pressure did not differ during SV. A change in wedge pressure in a direction opposite to the change in left ventricular pressure occurred after institution of IPPV and CPPV before and during near drowning.

The findings of this study are in accord with the findings of Wolff and Gradel (1975) who found increased LAP of an average of 2 to 2.5 torr on conversion from IPPV to SV and Askitopoulou et al. (1978) also demonstrated consistently greater LAP in three patients during CPAP and SV as compared to IPPV. Also in accord with this study, Shinn et al. (1979) found statistically significant differences between the changes in PCWP and PAD pressures both on and off IPPV.

**Physiological Interpretation**

A simple explanation for the findings of the present study is not possible. Rather, each individual case should be viewed individually incorporating all the physiological data for each subject. However, a general pattern of elevated PAP and PCWP is apparent. The following suggestions are offered for the pattern which occurred in this study. Because, in this study, cardiac output (CO) was not measured it is difficult to evaluate the hemodynamic measurement
alterations which occurred. Therefore two interpretations, one based on the assumption that CO increased during SV and the other that CO decreased during SV, will be discussed.

Four studies (Civetta et al., 1972; Downs et al., 1977; Kennedy et al., 1977; Grenvik, 1966) demonstrated an increase in CO on change from mechanical to spontaneous ventilation. Three of these same studies (Civetta et al., 1972; Downs et al., 1977; Grenvik, 1966) also reported an increase in mean PAP during SV as compared to mechanical ventilation. Kennedy et al. (1977) and Grenvik (1966) also reported no significant change in LAP on change from mechanical to spontaneous ventilation.

If CO did indeed increase in the majority of subjects in the present study the hemodynamic alterations may be explained as follows. When intrathoracic pressure decreases with conversion to SV, venous return to the right heart and blood flow into the lungs would also increase. Normally, PAP should increase only if CO increases four times normal CO, since a decrease in pulmonary vascular resistance should accommodate this increased flow. The majority of the subjects in the present study either have some type of myocardial disease (n = 12) and/or pulmonary dysfunction (n = 12) as evidenced by chest roentgenogram and/or smoking history. Since there is a close relationship between the heart and lungs, disease of one commonly affects the other, evidenced by the alterations in the pulmonary vasculature. The pulmonary vasculature is affected by hypoxia or passive pulmonary hypertension secondary to mitral stenosis, mitral regurgitation or left ventricular hypertrophy and may be unable to respond normally to
increased blood flow by decreasing pulmonary vascular resistance. The presence of pulmonary vascular disease and mitral stenosis may account for the inconsistencies in PAD and PCWP. Increased PAP secondary to acute hypoxia caused by ventilation perfusion abnormalities as a result of the change in ventilation mode (mechanical to spontaneous ventilation) is also a possible explanation for increased PAP. This latter hypothesis may explain the elevated PAP in those subjects without primary pulmonary vascular disease, or in those subjects status post-mitral valve replacement who may have reversed their pulmonary vascular dysfunction postoperatively. Beach et al. (1973) and Wolff and Gradel (1975) demonstrated an increase in pulmonary vascular resistance on conversion from mechanical to spontaneous ventilation. Subjects who had an increase in PAS pressure of 7 torr or greater (#6, #7, #8, and #12) may have had more severe pulmonary vascular disease secondary to mitral stenosis (#4, #6, and #7) or pulmonary disease secondary to smoking (#8 and #12).

Pulmonary capillary wedge pressure may have increased in this population because the stroke volume of the left ventricle must increase to pump the increased blood flow from the lungs. Since 12 of 14 patients had some type of myocardial dysfunction an increase in wedge pressure is expected with increased blood flow. Subjects #1 and #9 had clinically insignificant changes in wedge pressure, both of these subjects had no known left ventricular disease. Eight of the 12 subjects in whom wedge pressure was measured increased their PCWP in excess of 3 torr; all of these subjects had some type of myocardial disease. The increase in PCWP suggests that these eight subjects had
difficulty pumping the increased blood flow and compensated by increased left ventricular end-diastolic pressure. Four of these same subjects showed an increase in wedge pressure in excess of 8 torr (#2, #10, #11, and #14). Subject #2 had a cardiac arrest with a failing left ventricle, #10 had bacterial pericarditis and possibly pulmonary edema. Subjects #2 and #10 eventually died. Subject #11 had chronic renal failure and was on hemodialysis, and was three days postoperative abdominal aneurysm repair. This subject had not been dialized since before surgery so one can assume that her intravascular volume was large. Thus, on conversion to SV an even greater blood flow would occur through the lungs to the left heart. Subject #14 was postoperative abdominal aortic aneurysm repair with an intraoperative myocardial infarction. Subject #5 also had myocardial disease but decreased PCWP 5 torr on conversion from mechanical to spontaneous ventilation.

Beach et al. (1973) demonstrated a decrease in CO of 17% in 15 of 18 postoperative open heart patients on conversion from IPPV to SV. Wolff and Gradel (1975) also demonstrated a decreased CO in their study of open heart patients. Beach et al. (1973) demonstrated an increase in mean PAP without change in LAP, while Wolff and Gradel (1975) demonstrated an increased PAP and LAP. If CO did decrease in this study on changing from mechanical to spontaneous ventilation, none of the subjects showed clinical signs of early shock such as dyspnea, restlessness, apprehension, and diaphoresis attributed to decreased tissue oxygen in Beach et al.'s (1973) study. Three mechanisms have been proposed for the decrease in CO with change from mechanical to
spontaneous ventilation. One proposed by Beach et al. (1973) was that with withdrawal of positive pressure to the thorax the already dilated heart of patients with myocardial damage may have additional dilatation and therefore be unable to handle the increased blood flow. Another theory (Wolff and Gradel, 1975) is that with release of positive intrathoracic pressure there is a marked shift of blood from the systemic to pulmonary circulation, thus requiring the right heart to pump harder. The patient postoperative open heart surgery is often hypervolemic. The left ventricle because it is damaged has difficulty handling the increased blood flow from the pulmonary circulation so CO is decreased. Wolff and Gradel (1975) also propose that following open heart surgery there is some endocardial ischemia with perhaps greater coronary flow to the right versus left heart thus decreasing left ventricular contractility. With decreased intrathoracic pressure venous return is increased to the right heart, and overall pulmonary blood volume is increased. A heart which is damaged will have difficulty handling the increased volume of blood both from the systemic and pulmonary circulation. It is understandable that CO might decrease. Pulmonary artery pressure may initially rise to accommodate the increased blood flows (in the presence of elevated pulmonary vascular resistance) and subsequently wedge pressures rise because of the increased left ventricular end diastolic pressure needed to contract the damaged left ventricle. Despite the initial increase in PAP, PAP may eventually decrease secondary to the decreased CO and blood return to the right heart. However with an increase in PCWP, PAP may subsequently increase also. The amount of increase in PCWP would
depend upon the degree of left ventricular disease and the ability of the left ventricle to handle the increased blood flow.

**Static Effective Compliance**

The values for static effective compliance obtained during this study are first compared with values obtained by other investigators and then analyzed in relation to the hemodynamic results of the study. Static effective compliance measurements obtained in this study can be found in Table 5 and in Appendix F.

The static effective compliance measurements obtained during this study are in accord with the values of total thoracic compliance obtained by Grimby et al. (1975) on six intubated normal subjects. Subjects #3, #7, and #9 demonstrated lower static effective compliance measurements than the normals in Grimby et al.'s study; all others fell in the normal range. Suter et al. (1978) measured static effective compliance in nine subjects with acute parenchymal disease, the values obtained in these nine subjects are in accord with the values obtained for subjects #3, #7, and #9 in the present study. Ayres et al. (1968) measured total thoracic compliance on normals, emphysematous, and fibrotic lung disease subjects who breathed via a mouthpiece connected to an intermittent positive pressure breathing machine. Measurements obtained in the present study for static effective compliance fell into the obstructive and fibrotic subject values.

The findings of this study indicate that the state of lung compliance as measured by static effective compliance did not have an effect upon the transmission of positive pressure to intrathoracic
vessels. Subjects #3, #7, and #9 all had low values for static effective compliance, each of these had increased PAS pressures of four or greater torr, and an increase of two or greater in PAD pressures on conversion from mechanical to spontaneous ventilation. Pulmonary capillary wedge pressure, which was measured in only two of these subjects, increased 5 torr in one subject and decreased 1 torr in the other on conversion from spontaneous to mechanical ventilation. These changes are consistent with the other subjects with "normal" static effective compliance.

The significant negative correlation \( R = -.8 \) between static effective compliance values at approximately 10 cc per kg and the level of ventilator generated peak airway pressure generated during hemodynamic measurements indicates that the state of lung compliance can be predicted from the ventilator generated peak airway pressure. As ventilator generated peak airway pressures increase, lung compliance decreases and vice versa.

**Limitations of the Study**

Several factors may have contributed to the outcome of this study. First, there was no measurement of cardiac output during mechanical and spontaneous ventilation. This variable could have been used to calculate pulmonary vascular resistance, and in turn a more exact explanation of hemodynamic phenomenon would be possible. Also, the sample should have included more subjects with very low and high static effective compliances. This would allow for more evidence to establish the fact that the transmission of positive airway pressure to
the pleural space is not related to the state of lung compliance. It would have been helpful to include more subjects who received mechanical ventilation without PEEP. There were only three of these subjects in the study and a larger number would be better to establish if differences do exist between PEEP and zero end expiratory pressure ventilation.

Clinical Implications

The two major implications of this study are that patients on controlled mechanical ventilation should have FDPAC measurements taken at end-exhalation without being momentarily disconnected from the ventilator. This study has demonstrated significant increases in PAP and PCWP when controlled mechanical ventilation is momentarily interrupted, especially in patients with myocardial disease. It may be physiologically jeopardizing to disconnect a patient from controlled mechanical ventilation to obtain FDPAC measurements. Patients with severe heart disease may develop overt symptoms of left heart failure upon disconnection from the ventilator. If it becomes necessary to establish the differences in FDPAC measurements on and off the ventilator, temporary disconnection of the ventilator should be done cautiously. Once a baseline of measurement differences on and off the ventilator is assessed, all further readings should be taken while the patient is on the ventilator.

The study also emphasizes the importance of measuring end exhalation FDPAC pressures, preferably, with a strip chart recorder or, if such equipment is not available, from the oscilloscope tracing.
When utilizing either strip chart or oscilloscope tracing, the critical care professionals must be familiar with intrathoracic pressure influences upon FDPAC measurements. Practice and attention to the details of the FDPAC waveforms are necessary to determine end exhalation points. During this study the simultaneous recording of digital and waveform (strip chart) pressures revealed inconsistencies. Due to the averaging of inspiratory elevations of pressure during mechanical ventilation and inspiratory decrease of pressure during spontaneous ventilation, digital PAS and PAD pressures were consistently higher than waveform end-exhalation values.

Observation of the alterations in PAP and PCWP on conversion from mechanical to spontaneous ventilation may be utilized to assess the weaning progress in individual patients. Wolff and Gradel (1975) have recommended that to ensure optimal weaning of the post-open heart patient, during spontaneous ventilation, changes in PAP should not exceed a 7 torr increase and changes in PCWP or left atrial pressure should not exceed a 5 torr increase. Pulmonary artery systolic, diastolic, and wedge pressure increased to a greater degree in this study without clinical symptoms of heart failure or decreased cardiac output; it is recognized that not all of the patients in this study were in the process of weaning during the time the measurements were taken.

Critical care personnel may utilize peak airway ventilator pressures as an indication of the state of lung compliance and may follow changes in these pressures as an indication of response to therapeutic interventions. Data from this study indicate that absolute
peak airway pressures or PEEP up to 5 cm do not appear to affect intravascular monitoring.

**Suggestions for Further Study**

Suggestions for further study include the following. A replication of this study using a larger and more diversified sample including subjects with lower and higher static effective compliances, and subjects being ventilated with and without PEEP, would clarify and give more credence to the study.

Measurement of cardiac output, central venous pressure, and calculation of pulmonary vascular resistance during mechanical and spontaneous ventilation would help to establish evidence for the mechanism of alterations in PAP and PCWP on and off the ventilator.

Measurement of PAP, PCWP, central venous pressure, pulmonary vascular resistance, and cardiac output during successive time periods while subjects breathe spontaneously up to periods of 30 minutes would help to determine if subjects compensate and adjust further hemodynamically to the change in ventilation.

A descriptive study demonstrating the changes in cardiac output, central venous pressure, pulmonary vascular resistance, PAP, and PCWP between patients with and without myocardial disease would help to establish if the changes in intravascular pressure are unique to cardiac patients.

A study to determine if there are differences in transmural and intravascular pressures on and off the ventilator may help to further sort out the mechanisms for intravascular pressure changes.
A study to determine if differences exist in PAP, PCWP, and cardiac output between IMV with and without PEEP, and spontaneous ventilation and CPAP would help to determine if the lower mean intrathoracic and airway pressures during IMV alter FDPAC measurements as much as controlled mechanical ventilation.
CHAPTER 6

SUMMARY

Measurements obtained from the balloon tipped flow directed pulmonary artery catheter (FDPAC) are used extensively in critically ill patients to monitor left ventricular function, assess pulmonary status, and to guide fluid therapy. Since the FDPAC is floated from the right heart into the lungs, the measurements obtained may be influenced by pleural and intrathoracic pressure changes. Positive pressure ventilation, because it disrupts the normal inspiratory and expiratory pleural pressure changes, may alter the measurements obtained by the FDPAC. There has also been some scientific evidence that patients with stiff or non-compliant lungs convey very little of the positive airway pressure generated by the ventilator to the pleural space and intrathoracic vessels. Conversely, patients with highly compliant lungs may convey larger increments of the positive airway pressure generated by the ventilator to their pleural space and intrathoracic vessels. Thus patients with non-compliant lungs would be expected to show little or no change in PAP and PCWP on and off mechanical ventilation while patients with very compliant lungs may show changes in pressures on and off mechanical ventilation.

The purpose of this study was to determine if differences existed in PAP and PCWP during mechanical and spontaneous ventilation. The study also sought to determine if differences in FDPAC pressures
on and off the ventilator related to the state of the patient's lung compliance.

In the present investigation, a total of 14 subjects were studied. Pulmonary capillary wedge, PAS, and PAD pressures were obtained from a strip chart recorder at end-exhalation during both controlled mechanical and spontaneous ventilation. Eleven subjects received 5 cm PEEP during both mechanical and spontaneous ventilation. The remaining three subjects received IPPV and breathed spontaneously without PEEP. Static effective lung compliance measurements were obtained at approximate tidal volumes of 8, 10, 12, and 14 cc per kg immediately after the hemodynamic measurements were taken during controlled mechanical ventilation.

Results of this study showed that there was a significant increase (P < .001) in PAS and PAD pressures and a significant increase in PCWP (P < .05) off as opposed to on mechanical ventilation. Static effective compliance measurements obtained demonstrated that only three subjects had low compliance. The remaining 11 subjects had compliances within normal limits or normal for those on ventilators. Pressure/Volume curves calculated for each subject demonstrated that the three subjects with low compliance had P/V curves which were shifted to the right. All other curves (n = 11) somewhat resembled normal P/V curves. There was no significant relationship (P > .05) between changes in PAP and PCWP and static effective lung compliance calculated at approximate values of 10 cc per kg. There was however a significant negative correlation (R = -.8) between static effective compliance and ventilator generated peak airway pressure.
Since 12 of the 14 subjects had some type of myocardial disease, it was proposed that the changes in hemodynamic pressures on and off the ventilator were due to the difficulty of the left ventricle in handling the increased cardiac output which occurred upon conversion from mechanical to spontaneous ventilation. Two subjects without evidence of myocardial disease demonstrated no significant changes in wedge pressures on and off the ventilator.

The results of this study indicate therefore that higher PAP and PCWP can be expected in patients with myocardial disease when they are changed from mechanical to spontaneous ventilation. This study also demonstrated that in this particular sample, lung compliance did not have a significant effect upon hemodynamic measurements.
APPENDIX A

SUBJECT'S CONSENT

Project Title: Effect of Positive Pressure Ventilation and Static Lung Compliance Upon Pulmonary Artery and Wedge Pressures

I, Patricia Van Sciver, R.N., am conducting an investigation to determine if a patient's lung condition and breathing pattern on a mechanical ventilator (breathing machine) will have an effect on the measurements taken in the lung by a Flow Directed Pulmonary Artery Catheter (FDPAC). The Flow Directed Pulmonary Artery Catheter is a tube inserted through your vein into your lung to record pressures in your blood vessels. In order to take part in this study you must be a patient in the Intensive Care Unit (ICU) who has a FDPAC in place and a tube in your windpipe to breathe on a mechanical ventilator. Neither the tube in your windpipe nor the catheter in your blood vessel will be inserted solely for this study. The information gained from this study will enable nurses and doctors in intensive care units to set up procedures for the most accurate FDPAC measurements.

In order to obtain measurements from the FDPAC, patients are sometimes temporarily disconnected from the ventilator. Measurements can also be taken when the patient is on the ventilator. These Flow Directed Pulmonary Artery Catheter measurements are routinely taken as a normal part of your vital sign monitoring (blood pressure, heart and respiratory rate) by the nurses in the ICU.

When it is time for your FDPAC measurements to be taken I will take the measurements once while you are receiving breaths from the ventilator and the other time when you are receiving oxygen but no breaths from the ventilator. Whether or not the measurements will be taken first when you are receiving ventilator breaths or first when you are just receiving oxygen will be explained to you at that time. Each set of measurements should take no longer than 2 minutes. After I have taken the measurements of the FDPAC with you receiving only oxygen from the ventilator, I will immediately set the ventilator to give you your normal (previous set rate) breaths for 5 minutes, just in case you need rest. If at any time when I am taking the FDPAC measurements, you feel shortness of breath which is bothersome or any other ill effect, I will immediately give you ventilator breaths at your prescribed amount.

The third measurement I will be taking is a measurement of the ability of your lungs to stretch (Effective Static Compliance). While you are breathing on the ventilator I will set the ventilator to give you four different volumes of air to breathe. After each breath I will
have you hold your breath for 3-5 seconds while I record the pressure reading on the ventilator. This should take no longer than 3 minutes. I will be taking this part of the study immediately after you have been receiving ventilator breaths for the FDPAC measurements. If at any time during this part of the study you feel shortness of breath which is bothersome or any other ill effects I will return your ventilator to its prescribed volume and rate until you feel better.

The entire study will take approximately 12-15 minutes including one 5 minute rest period.

Because FDPAC measurements are routine in your care and because occasionally the ability of your lungs to stretch is also measured, there will be no significant added risk to you by this study.

There is no cost to you for your participation in this study.

I will answer any questions regarding this study that you may ask. You are free to withdraw or to refuse to participate in this study at any time; it will not affect the quality of your treatment or care. Your identity will remain confidential and the information obtained from this study will be recorded and analyzed by computer. The data obtained from this study may be combined with data in subsequent studies.

If you understand what is involved and you consent to participate in this study, please sign your name below.

The nature, demands, risks and benefits of this study have been fully explained to me and I fully understand what my participation involves. I also understand that I may ask questions and I am free to withdraw from the study at any time without ill will (or affecting my medical care).

I understand that in the event of physical injury resulting from the research procedures, financial compensation for wages and time lost and the costs of medical care and hospitalization is not available and must be borne by the subject.

I also understand that this consent form will be filed in an area designated by the Human Subjects Committee with access restricted to the principal investigator or authorized representatives of the particular department. A copy of this consent form is available to me upon request.

Subject or Legal Guardian's Signature __________________________

Date ______________________

Witness' Signature __________________________

Date ______________________
APPENDIX B

PHYSICIAN'S CONSENT

Project Title: Effect of Positive Pressure Ventilation and Static Lung Compliance Upon Pulmonary Artery and Wedge Pressures

Permission has been given to Patricia Van Sciver, R.N., to utilize my patient, __________________________, in the collection of data for a research study conducted through The University of Arizona, College of Nursing, Graduate Division. I understand that the study involves measurements of Pulmonary Artery and Wedge Pressures on and off the ventilator, and a measurement of effective static compliance. In signing this form I agree that my patient, __________________________, is able to breathe spontaneously off the ventilator for one to two minutes and that he/she should also be able to tolerate receiving tidal volumes of 8, 10, 12, and 14 cc/kg for one to three breaths each. I understand that the peak pressure generated by the ventilator should not increase greater than 15 cm water during any of the above volume changes, or if the patient should show any signs of respiratory distress during these maneuvers the data collection will be terminated. This consent is given with the permission that the researcher also obtains the consent of the individual patient or his/her's immediate family member and the approval of the University Hospital, Tucson, Arizona.

I also understand that this consent form will be filed in an area designated by the Human Subjects Committee with access restricted to the principal investigator or authorized representatives of the particular department.

Signature:_____________________________________

Date:______________________________________
APPENDIX C

HUMAN SUBJECTS COMMITTEE LETTER OF APPROVAL

THE UNIVERSITY OF ARIZONA
TUCSON, ARIZONA 85724
HUMAN SUBJECTS COMMITTEE
ARIZONA HEALTH SCIENCES CENTER

14 February 1980

Patricia T. Van Sciver, R.N., B.S.N.
College of Nursing
Arizona Health Sciences Center

Dear Ms. Van Sciver:

We are in receipt of your project entitled, "Effect of Positive Pressure Ventilation and Lung Compliance upon Pulmonary Artery and Wedge Pressure Measurements", which was submitted to the Human Subjects Committee and concur with the opinion of the Departmental Review Committee's examination and recommendation of this minimal risk project. Therefore, approval is granted effective 14 February 1980, under the condition that you add the following statement to the last paragraph of the subject's consent form: "A copy of this consent form is available upon request".

Approval is granted with the understanding that no changes will be made in the procedures followed or in the consent form used (copies of which we have on file) without the knowledge and approval of the Human Subjects Committee and the Departmental Review Committee. Any physical or psychological harm to any subject must also be reported to each committee.

The Committee has reviewed the current practice of filing signed consent forms on the patient's chart. A summary of the clinical research protocol is now being filed on the chart for the information of patient care personnel. In order to insure that all signed consent forms are available in one location in the event that university officials or investigators require the information and the principal investigator is no longer on the staff or unavailable for some other reason and to eliminate duplication, you are instructed to forward all signed consent forms to the Department (or College) Head for permanent filing in an area under his direct supervision. Do not file them on the patient's chart. This requirement will apply to all projects which are approved from this date forward.
Patricia T. Van Sciver, R.N., B.S.N.
Re: "Effect of Positive Pressure Ventilation and Lung Compliance upon Pulmonary Artery and Wedge Pressure Measurements"
14 February 1980

Page Two

In keeping with Professional Standards Review Committee requirements concerning the filing of clinical research protocol summaries on the individual's patient chart, mentioned above, we are enclosing a memorandum from Dr. William Scott, Chief of Staff, Arizona Health Sciences Center.

Sincerely yours,

Milan Novak, M.D., Ph.D.
Chairman
Human Subjects Committee

MN/jm
Enclosure

cc: Ada Sue Hinshaw
Departmental Review Committee
APPENDIX D

DATA COLLECTION SHEET

Patient Profile Data

Subject number_____ Age_____ Sex____
Measurement Order A B Height_____ Weight____

Diagnosis

Previously diagnosed Lung Disease

FEV1/FVC (date)__________
ABG's (date)__________
Other (pulm. meds., activity tolerance)_____________________________

Smoking History_____________________________

Current History

Most Current ABG's (date/time)

PVO2__________________ B/P________________
COP__________________ Heart Rate & Rhythm________________

Current Medications_____________________________

Last CXR (date)____________________________________

Ventilation Parameters

FIO2________ IMV______ Peak Airway Pressure________
Vt (exhaled)________ PEEP______ Pt's respiratory rate______
Number of hours on mechanical ventilation____________________
Number of hours with FDPAC__________________________
| Presence of ABD. distension____, Scoliosis________ |
| Initial PAS____, PAD____, PCWP____ (on/off vent) |

<table>
<thead>
<tr>
<th>Spontaneous rate____</th>
<th>PAS</th>
<th>PAD</th>
<th>PCWP</th>
<th>Airway Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEEP____</td>
<td></td>
<td></td>
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</tbody>
</table>

| Controlled rate____  |     |     |      |                 |

<table>
<thead>
<tr>
<th>Exhaled Vol.</th>
<th>Plateau Pressure</th>
<th>PEEP</th>
<th>CV</th>
<th>Compliance</th>
</tr>
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<tbody>
<tr>
<td>1. 8 cc/kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. 10 cc/kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. 12 cc/kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. 14 cc/kg</td>
<td></td>
<td></td>
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</table>
APPENDIX E

INDIVIDUAL PULMONARY ARTERY SYSTOLIC, DIASTOLIC, AND PULMONARY CAPILLARY WEDGE PRESSURES, ON AND OFF THE VENTILATOR (torr)

<table>
<thead>
<tr>
<th>Subject</th>
<th>PAS On</th>
<th>PAS Off</th>
<th>PAD On</th>
<th>PAD Off</th>
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<th>PCWP Off</th>
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<td>22</td>
<td>27.5</td>
<td>10</td>
<td>13.5</td>
<td>6</td>
<td>14.5</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>34</td>
<td>20</td>
<td>22</td>
<td>--</td>
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</tr>
<tr>
<td>4</td>
<td>50</td>
<td>57</td>
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<td>13.5</td>
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<td>5</td>
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<td>12</td>
<td>18.5</td>
<td>9.5</td>
<td>13</td>
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<tr>
<td>7</td>
<td>38</td>
<td>46</td>
<td>18</td>
<td>22</td>
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<td>9</td>
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<td>10</td>
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<td>42</td>
<td>22.5</td>
<td>24</td>
<td>14.5</td>
<td>28</td>
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<tr>
<td>11</td>
<td>35.5</td>
<td>34</td>
<td>18</td>
<td>20</td>
<td>8</td>
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<td>39.5</td>
<td>49</td>
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<td>19</td>
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<td>56.5</td>
<td>22</td>
<td>26</td>
<td>22.5</td>
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</tr>
<tr>
<td>14</td>
<td>24.5</td>
<td>30.5</td>
<td>17</td>
<td>21</td>
<td>9.5</td>
<td>21.5</td>
</tr>
</tbody>
</table>

Mean  34.6  39.7  18.9  22.2  13.6  18.1

SD    10.0  10.5  5.4   5.2   5.0   4.8

Significance  P < .001  P < .001  P < .05

\(^a\) No PEEP

\(^b\) Standard Deviation
APPENDIX F

INDIVIDUAL SUBJECT DATA DETAILING TIDAL VOLUME, TIDAL VOLUME EXPRESSED IN CUBIC CENTIMETERS PER KILOGRAM, CHANGE IN PRESSURE, AND STATIC EFFECTIVE LUNG COMPLIANCE IN MILLILITERS PER CUBIC CENTIMETER OF WATER

<table>
<thead>
<tr>
<th>Subject</th>
<th>Tidal Volume/ cc per kilogram</th>
<th>Change in Pressure</th>
<th>Static Effective Compliance</th>
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<tbody>
<tr>
<td>1</td>
<td>592 cc/ 8.2</td>
<td>11</td>
<td>53.81</td>
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<td>668 cc/ 9.2</td>
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<td>47.71</td>
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<tr>
<td></td>
<td>884 cc/12.2</td>
<td>17</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>1060 cc/14.7</td>
<td>20</td>
<td>53</td>
</tr>
<tr>
<td>2</td>
<td>645 cc/ 7.8</td>
<td>10</td>
<td>64.5</td>
</tr>
<tr>
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<td>1056 cc/12.8</td>
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<td>58.66</td>
</tr>
<tr>
<td>3</td>
<td>430 cc/ 6.4</td>
<td>15</td>
<td>28.66</td>
</tr>
<tr>
<td></td>
<td>540 cc/ 8</td>
<td>20</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>666 cc/ 9.9</td>
<td>28</td>
<td>23.78</td>
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<tr>
<td></td>
<td>883 cc/13.1</td>
<td>29</td>
<td>30.44</td>
</tr>
<tr>
<td>4</td>
<td>561 cc/ 7.4</td>
<td>13</td>
<td>43.15</td>
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<td>736 cc/11.3</td>
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