THE EFFECT OF LATERAL POSITIONING ON PULMONARY ARTERY AND PULMONARY CAPILLARY WEDGE PRESSURES IN CRITICALLY ILL PATIENTS

BY

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CHAPTER ONE

INTRODUCTION

The measurement of pulmonary artery (PA) and/or pulmonary capillary wedge (PCW) pressures is a procedure frequently carried out by nurses in the intensive care unit (ICU). Since the introduction of the flow directed balloon-tipped catheter, Swan-Ganz Catheter (Swan, 1970), fluoroscopy is no longer essential for safe and rapid catheter placement. Nursing care of the patient in whom a Swan-Ganz catheter is placed is described in the literature (Gernert, 1973; Bolognini, 1974; Woods, 1976).

Statement of the Problem

Pulmonary artery and PCW pressures have traditionally been measured with the patient in the supine (0° backrest) position for every reading. Recently the need for this practice has been questioned (Woods and Mansfield, 1976; Hansen, 1976). Sleep deprivation resulting from the frequent repositioning of the critically ill patient for monitoring purposes only is not desirable. If these measurements could be taken with the patient in the lateral position, the frequency with which patients are awakened and repositioned could be decreased. Thus, the critically

ill patient could benefit from longer intervals of uninterrupted sleep.

Review of the Literature

The literature reviewed considered three concepts: 1) sleep deprivation, its occurrence, cause and effect in the critically ill patient; 2) the evaluation, physiologic basis and clinical measurement of hemodynamic monitoring; and 3) patient positioning as it affects hemodynamic measurements and reference levels.

Sleep Deprivation

According to Kleitman's (1963) evolutionary theory of sleep and wakefulness, a human being's day has a natural rhythm. A short term periodicity characterizes this rhythm in which cycles of 80 to 90 minutes duration occur. Five of these cycles occur during normal sleep in most people. Each sleep cycle consists of four stages and is punctuated by one period of rapid eye movement or REM I sleep. The stages are sequentially experienced in a descending fashion; after reaching stage IV, the stage of deepest sleep, the stages are retraced and culminate in REM I sleep. REM sleep is an indication of eventful dreaming. Dement and Kleitman (1957) found an 80% incidence of vivid detailed dream recall in 191 awakenings of subjects during REM I sleep.

Sleep deprivation has been discussed as the inability to perform the number of sleep cycles to which one is acclimated (McFadden, 1971). Total deprivation and deprivation of certain stages has been studied in both animals and in human beings. Koella (1967) reported some of the adverse effects of sleep deprivation: irritability, inattention, loss of memory, illusions, hallucinations and hyperirritability. Recovery sleep, or that sleep occurring after deprivation, is characterized by greater proportions of stage IV and REM I sleep.

McFadden and Giblin (1971) and Walker (1972) documented sleep deprivation experienced by patients in an ICU. Woods (1972) measured frequency of interruption of rest and sleep by nursing care activities. Direct monitoring (Wood, 1972) defined as obtaining vital signs, weight and urinary output was the most frequently occurring interruption.

A patient's physical and psychosocial response to a life threatening illness may be complicated by the adverse effects of sleep deprivation (Koella, 1967). Nursing care of the critically ill patient is a primary source of interruption of rest and sleep. Direct monitoring has been identified as the category of nursing intervention that most frequently interrupts (Woods, 1972). One element of this category is hemodynamic monitoring.

Hemodynamic Monitoring

Rapid advances have occurred in the treatment of the critically ill patient. With these advances additional techniques of hemodynamic assessment and monitoring have emerged. The number of deaths due to acute arrhythmias in patients with myocardial infarction has declined as pre-hospital and hospital coronary care has advanced. Since the major threat of death due to arrhythmias has been reduced, the support and assessment of the mechanical function of the heart has become a major focus of treatment and monitoring. Techniques for monitoring left ventricular function are being performed more frequently.

Right atrial or central venous pressures, pulmonary capillary wedge-pressures and left ventricular pressure monitoring are used to assess left ventricular function, to monitor effects of therapy, to determine prognosis and to classify severity of disease (Rackley, 1972). Additionally, cardiac output measurements supplement data derived from history, physical examination, laboratory work, x-ray studies and communication with the patient in the evaluation of hemodynamic status (Forrester, 1976).

Left ventricular function can be represented as a relationship between cardiac output and the pressure in the left ventricle at end diastole (LVEDP). The force of ventricular contraction is dependent upon the myocardial

fiber length just prior to ventricular systole, which is related to the end diastolic volume and/or pressure. Normal cardiac muscle will increase the force of contraction in response to an increase in left ventricular volume, up to a point. After this point, force of contraction will not increase. Both end diastolic volume and tension are difficult to measure. Therefore, the end diastolic pressure is used (Scheinman, M., Evans, T., Weiss, A., Rapaport, E., 1973). Likewise cardiac output is used as a measure of force of ventricular contraction.

The relationship between cardiac output and left ventricular end diastolic pressure is expressed in graphic form in Figure 1. Curve A represents normal left ventricular function and contractility. With an increase in the amount of blood in the left ventricle at end diastole, the muscle fiber length, i.e., preload, increases producing a corresponding increase in force of contraction, stroke volume and cardiac output. An example of increased preload occurs with changes in venous return to the left ventricle in the respiratory cycle.

Curve B describes left ventricular function associated with increased contractility as seen in the epinephrine stimulated heart. A rise in LVEDP produces an increase in cardiac output.

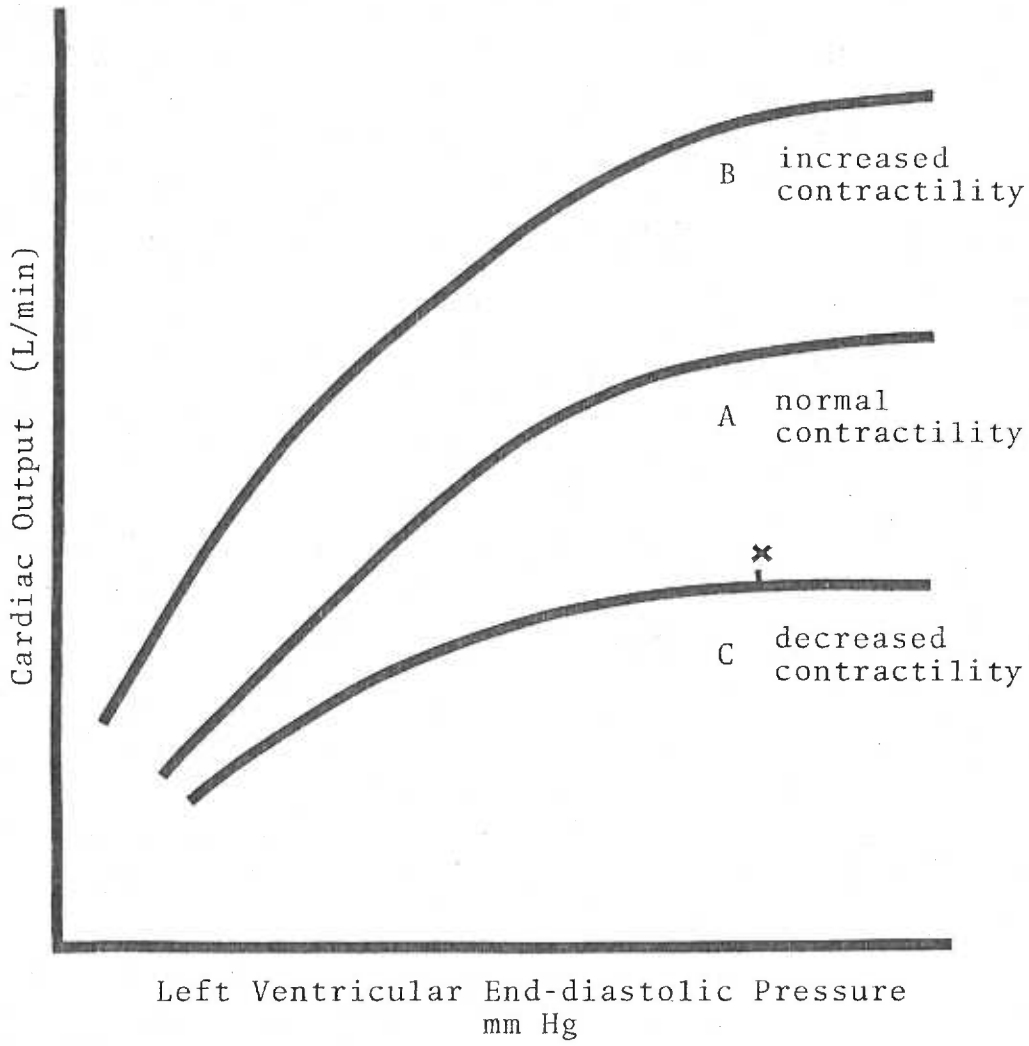

Figure 1

Left Ventricular Function Curves

(adapted from Hurst, 1974)

Left ventricular function associated with reduced contractility is represented by curve C. There is reduced contractility and therefore cardiac output at any given LVEDP in curve C relative to the same LVEDP in curves A and B.

In acute congestive heart failure associated with decreased contractility, an increase in LVEDP does not result in a sufficient increase in cardiac output. Point X on curve C depicts this situation. At this point an increased left ventricular volume does not result in an increased stroke volume. Excessive residual blood in the left ventricle is augmented by diastolic filling. End diastolic pressure in the left ventricle rises. The pressure in the left atrium and pulmonary venous system also increase. As hydrostatic pressure in the pulmonary circulation increases, transudation of fluid occurs first into the interstitial space and then into the alveoli. If left ventricular function remains decompensated, right ventricular and right atrial pressures ultimately increase. Pressure measurements in the right side of the heart are therefore of limited value in the early detection, diagnosis and treatment of left ventricular failure.

Measurement of the LVEDP is used clinically to estimate left ventricular function and to guide therapy. The use of LVEDP as an indicator of left ventricular function has some limitations in the clinical setting (Braunwald,

1963; Rahimtoola, 1973).

For example, Braunwald (1963) cautioned against the inflexible interpretation of LVEDP as a measure of left ventricular function. He cited hypervolemia, valvular abnormalities, circulatory shunts, and pericardial constriction as situations in which LVEDP rises. In these cases increased LVEDP is not necessarily an indication of impaired myocardial contractility. Braunwald also reasoned that patients with normal LVEDP may have diminished cardiac function.

Rahimtoola (1973) discussed two left ventricular diastolic pressures: the first (LVDP pre a) is measured prior to left atrial contraction; the second (LVEDP) is measured just after left atrial contraction. Because of the decreased compliance of a failing ventricle, the effect of left atrial contraction may be exaggerated. He suggested using LVEDP as the most accurate indicator of left ventricular function.

Measurement of LVEDP may be accomplished by direct or indirect methods. Direct cannulation of the left ventricle is difficult to perform and is of considerable risk to the patient. Indirect measurement is safer and is based on the premise that the pressure in the left atrium is equal to the left ventricular pressure at the end of diastole, given a normal mitral valve and left atrium. Pulmonary capillary wedge pressure (PCWP), pulmonary artery

end diastolic pressure (PAEDP) are likewise thought to be correlated to LVEDP. There is controversy about the accuracy of this correlation in certain situations (Lappas, Lell, Gabel, Ciretta and Lowenstein, 1973; Rahimtoola, Loeb, and Ehsani, 1972; Falicov and Resnick, 1970; Fischer, DeFelici and Parisi, 1975).

Falicov, et al. (1970) found a good correlation between PAEDP and PCWP in subjects with left ventricular dysfunction and whose pulmonary vascular resistance was less than 400 dynes sec cm^{-5}/m^2 BSA. Neither of these two measurements was found to accurately reflect LVEDP. These investigators found, however, that in the failing ventricle an "a wave" representing retrograde conduction of the left atrial contraction wave was seen in the pulmonary artery pressure and pulmonary wedge pressure tracings. The pulmonary artery "a wave" pressure was found to more accurately represent the LVEDP in patients with ventricular dysfunction in the absence of pulmonary vascular obstruction.

Additionally, Rahimtoola, et al. (1972) reported that PAEDP did not reliably indicate left atrial pressure (LAP) in patients with acute myocardial infarction. They attributed this to an increased pulmonary vascular resistance. Further, they stated that in the failing ventricle, mean LAP was equal to the left ventricular diastolic pressure prior to contraction of the left atrium (LVDP pre a). Thus

the authors held that the indirect methods of measuring LVEDP were not accurate.

In a population not controlled for altered pulmonary vascular resistance Lappas et al. (1973) found the PCWP to reliably indicate LAP. These investigators attributed a discrepancy between PAEDP and PCWP to the effects of tachycardia and/or abnormal pulmonary vascular resistance.

Fischer, et al. (1975) described pulmonary capillary "a wave" pressures (PCa) as the most representative of LVEDP. In the absence of tachycardia and marked pulmonary vascular disease, PCWP was found to reliably reflect LVDP pre a as did PAEDP. The investigators cited difficulties in identifying "a waves" in pulmonary artery tracings in the clinical setting. While PCa pressures were identifiable in all patients, interpretation required an additional effort impractical for monitoring purposes. The authors suggested using PCa only in experimental settings where exact correlation with LVEDP was important and recommended the clinical use of PCWP as a reliable indicator of LVDP pre a. Humphrey (1976) also supported the use of PCWP as a clinical tool for estimation of LAP in patients with cardiac disease. Recently, Gorlin (1977) stated that the use of PCWP in diagnosing left ventricular failure was imperative.

Patient Position and External Reference Point Determination

Measurement of pulmonary artery (PA) and pulmonary capillary wedge (PCW) pressures have traditionally been done with the patient in the supine (0° backrest) position. The reference point is determined by the principles of the phlebostatic axis and the phlebostatic level, and represents the level of the left atrium. The effect of change in the angle of the backrest of the supine patient on PA and PCW pressures has been studied (Prakash, Parmley, Dikshit, Forrester and Lowenstein, 1973; Woods and Mansfield, 1976; Hansen, 1976).

Hemodynamic indicators were measured by Prakash et al. (1973) in 21 patients with acute myocardial infarction who were positioned in the supine (0° backrest) position and moved to the semierect (70° backrest) position. The investigators reported a mean rise in PCW pressure of 3.4 mm Hg which was significant on paired t-test analysis ($p \leq 0.01$). Nine subjects showed a difference of 4 mm Hg or greater. Additionally, nursing researchers have studied positional effects on the measurement of PA and PCW pressures.

Woods and Mansfield (1976) using phlebostatic level as the reference point varied the angle of the backrest in 10 noncritically ill patients. In the 0°, 20°, and 45° positions, they found no significant difference in the

pulmonary artery mean (PAM), pulmonary artery diastolic (PAD), and pulmonary capillary wedge (PCW) pressures. In the 90° and dangle position, however, significant differences were found ($p \leq 0.05$).

Hansen (1976) studied the same relationship in 18 critically ill patients. She found a significant difference of four mm Hg or greater in the PA and PCW pressures in 10 of 18 subjects with position change. Data were collected over a 60 minute period. The findings were thought to reflect a change in the status of the critically ill subjects and/or a true effect of position change.

These findings suggest that a change in the angle of the backrest to 45° or less in the supine patient may not affect the PA and PCW pressures. Maintaining a constant reference point and taking readings quickly enough so that patient status does not change are two challenges.

Patients are also placed in the lateral recumbent position. In this position, the frontal plane of the patient is at a 90° angle with the surface of the bed. The effect of the lateral position on central venous pressure was studied by Jereos (1971). She used an external reference point to determine the level of the right atrium. For the lateral position she selected a reference point on the chest wall in the fourth intercostal space to the right of the sternum. For the supine position, she selected the

point representing half the anterior-posterior diameter of the chest in the plane of the fourth intercostal space. In the data analysis this point correlated with a midaxillary point. There was a significant difference between the pressures measured in the supine and lateral positions.

Wright (1974), investigated the reference points used by Jereos. She determined that the reference point in the supine position that approximated the level of the right atrium, should be in the anterior axillary line. This determination was based on the location of the right atrium in cadavers. Thus, the reference level must be constant when comparing pressures taken with the patient in various positions.

The phlebostatic axis and level were determined by Burch and Windsor (1945) to assist identification of the level of the heart in any person of any build in the supine (backrest at any angle) position. In identifying the level of the heart in most patients they recommended that the phlebostatic axis should be identified. This line is formed by the intersection of the transverse plane through the fourth intercostal spaces at the sternum and the frontal plane passing through a point one half the anterior-posterior diameter of the chest measured at the fourth intercostal space. Wright (1974) found a midaxillary point to be consistently posterior to the level of the mid-right

atrium by about three centimeters. The point half the anterior-posterior diameter in the plane of the fourth intercostal space as approximated at the midaxillary line is the recommended reference point for use in measuring PA and PCW pressures in the supine patient. This external reference point is thought to represent the level of the left atrium.

The left atrium is located in the midline and is superior and posterior to the other chambers of the heart (Hurst, 1974). It is described as being at the approximate level of the eighth thoracic vertebra posteriorly; anteriorly it is at the level of the intersection of the fourth intercostal space with the sternum. A point at the sternum where it intersects with the fourth intercostal space is a reference level for the measurement of the PA and PCW pressures in the patient in the lateral position.

Purpose

The purpose of this study was to assess the effect of lateral patient positioning on the pulmonary arterial and pulmonary capillary wedge pressures in critically ill subjects.

Hypotheses

1. Pulmonary artery pressures and pulmonary capillary wedge pressures measured in the supine and lateral positions will not differ significantly.

2. Pulmonary artery pressures and pulmonary capillary wedge pressures measured at one minute after position change will not be significantly different from those measured five minutes after position change.

3. Pulmonary artery diastolic pressures will not differ from pulmonary capillary wedge pressures for patients in the supine and in the lateral position.

CHAPTER TWO

METHODOLOGY

Design

The study was a modified time-series field experiment, a quasi-experimental design described by Campbell and Stanley (1963). Patients were assigned to a group according to catheter insertion site, after which they were randomly assigned to subgroups. Measurements were taken in the initial position at one and five minutes. Patients were turned to an alternate position and measurements were again taken at one and five minutes. Finally, the patient was returned to the original position and measurements were repeated at one and five minutes. This procedure was repeated three times with a two hour interval between trials. Comparison of five minute pulmonary capillary wedge (PCW) pressures in the first and third positions were used to assess patient stability through each trial.

Subjects and Setting

Subjects were the first eight patients who met the selection criteria in either the University Hospital Medical Intensive Care Unit or Veterans Administration Hospital

Coronary Care Unit during the data collection period. Criteria for the selection of persons for inclusion in the study were:

1. Permission from the patient and/or relative in compliance with one's human rights. (See Appendix A, page 45)

2. Presence of a functioning Swan-Ganz catheter.

3. Identification of the patient as critically/ acutely ill as determined by admission to the intensive care unit.

4. Ability of the patient to tolerate the lateral position as determined by physician in charge at the time of measurements.

5. Absence of diagnosed mitral insufficiency.

Procedure

Prerequisite to the data collection was a consistent method for determining external reference points in the supine and lateral positions. The procedures for reference point determination are as follows.

Reference point for the supine patient. The point is located on the lateral chest wall of a patient derived in compliance with the concepts of the phlebostatic axis and approximates the level of the left atrium.

1. The transverse plane through the fourth intercostal (between the fourth and fifth ribs) space at the lateral margin of the sternum is identified.

2. The anterior-posterior diameter of the patient's chest is measured in that plane using a Picker x-ray calipers.

3. The mid-point of that measurement is marked on the lateral chest wall of the patient and is called the reference point.

4. Using a yardstick and level the port of the stopcock on the transducer which is open to atmospheric pressure during calibration is aligned with the external reference point.

5. The level of the reference point represents relative zero pressure to which the pulmonary artery and pulmonary capillary wedge pressures refer.

Reference point in the laterial position. It is a point on the anterior or posterior chest wall in the midline which approximates the level of the left atrium.

1. The midline of the subject's thorax is identified in the plane of the fourth intercostal space by the mid-sternum and/or the eighth vertebra.

2. A point in this line is marked on the anterior or posterior chest wall and is called the reference point for the lateral position.

3. The port of the stopcock on the transducer which is opened to atmospheric pressure is aligned with the external reference point.

4. The level of the reference point represents relative zero pressure to which the pulmonary artery and pulmonary capillary wedge pressure refer when taken in the lateral position.

Each patient was placed into one of four groups. Group A included those patients whose Swan-Ganz catheter had been inserted through a vein in their right arm. They were positioned on their left side. Group B included those patients whose Swan-Ganz catheter had been inserted through a vein in the left arm. They were positioned on their right side. Group C included patients whose Swan-Ganz catheter had been inserted in a left subclavian vein. They were positioned on their right side. This procedure was used to prevent catheter twisting or compression as a cause of variation in pressure which could have occurred if patients were positioned on the side of catheter insertion.

Patients were then randomly assigned to subgroups 1 and 2 in the groups A, B and C. Those patients in the subgroup designated by number 1 were moved from the supine position, to the respective lateral position and returned to the supine position. Those subjects in the subgroup 2

were turned from the lateral position to the supine position, and returned to the lateral position. Alternate sequencing was used to prevent systematic bias in the results.

The sequence and/or side of positioning differed for each subgroup. Patients in A1 started in the supine position, were turned to the left lateral position and returned to the supine position. Patients in group A2 started in the left lateral position, were turned to the supine position and returned to the left lateral position. Patients in group B1 and C1 started in the supine position, were turned to the right lateral position and returned to the supine position. Patients in groups B2 and C2 started in the right lateral position, were turned to the supine position and returned to the right lateral position.

Each subject was placed in the first of the three positions. After one and five minutes pulmonary artery (PA) and pulmonary capillary wedge (PCW) pressures were recorded and marked with position, time and trial number. The subject was then turned to the second position. After one and five minutes the measurements were repeated. Finally, the subject was returned to the original position and measurements were repeated again after one and five minutes. Measurements made in the fifteen minute period constituted one trial. Three additional trials were made at approxi-

mately two hour intervals. After each position change the transducer was moved to align the airport of the stopcock with the appropriate external reference point.

The recorder was calibrated using a mercury manometer. After each movement of the transducer the recorder was recalibrated to a zero baseline. The pulmonary artery diastolic (PAD) and pulmonary artery systolic (PAS) pressures were recorded first, followed by pulmonary artery mean (PAM) and pulmonary capillary wedge (PCW) pressures. In each case a Statham transducer, an Electronics for Medicine recorder (model VR-6 or DR-12), and rapid photographic write out were used. Mean pressures were obtained electronically. At least eight seconds were allowed for stabilization of the mean recordings. Measurements were recorded at 25 mm/sec paper speed.

A portable anterior-posterior chest x-ray was examined on the morning of pressure measurements to locate the tip of the catheter. The side of catheter tip placement in the pulmonary artery was recorded as either right or left. The distance between the catheter tip and the midsternal line was recorded. When available a second chest x-ray was examined. This was done to determine if the catheter tip had moved.

Pressure readings were interpreted by the investigator and by a staff cardiologist. An average of the two numbers was recorded.

CHAPTER THREE

RESULTS AND DISCUSSION

Eight male patients, admitted to the intensive care units of the two hospitals during the data collection period, met the selection criteria and were subjects of this study. The patients ranged in age from 45 to 84 years. Individual patient data describing the sample are presented in Table 1. The sample represented a wide range of diagnoses, age, mean PCW pressures and pharmacologic interventions. Each side and sequencing of positioning were equally represented.

Initially, analysis was done across patients to determine trends in the sample. Where appropriate, further trends of variance were sought within individual patient data. The results and discussion are presented simultaneously. Hypothesis one and two are discussed first, followed by hypothesis three.

To ascertain differences across patients between pressures measured in the supine and lateral position, hypothesis one, a two factor analysis of variance with one repeated measure was done. This manipulation also tested the difference between pressures measured at one minute and at five minutes after position change, hypothesis two. F

TABLE 1

Individual Patient Data

Patient	Group	Age	$\overline{PCW}$[a]	Diagnosis	Medication
1	A1	84	25.38	Post Cardiopulmonary Arrest	None
2	A1	63	9.00	Gastro-intestinal Bleeding, Chronic CHF, COPD	Aminophylline Pitressin
3	A2	66	9.50	Antero-lateral Myocardial Infaction	None
4	A2	56	18.00	Angina, S/P ASD Repair	None
5	B1	69	6.13	Gastro-intestinal Bleeding	None
6	C2	58	34.25	Myocardial Infarction	Furosemide Nitroprusside
7	B2	45	16.75	Sepsis, Hepatic Failure	Gentamicin Chloramphenicol
8	C1	61	20.38	Post Cardiopulmonary Arrest	Dopamine

[a] $\overline{PCW}$ is the mean five minute supine PCW pressure across trials measured in the supine position.

scores were calculated to reveal any interaction between effect of position change, factor A, and time after position change, factor B, on pressures. Each pressure in each trial was tested separately. The findings are summarized in Appendix B, p. 50 and the raw data are included in Appendix C, p. 52.

Hypothesis one, the major hypothesis, addressed the effect of position change on pulmonary artery (PA) and pulmonary capillary wedge (PCW) pressures. Statistical analysis showed no significant difference between pressures in the supine and lateral position. Clinical significance, however, does not always correspond to statistical significance. Therefore, individual pressure differences were inspected for clinical significance.

While it is customary to monitor all pressures in critically ill patients, PCW pressures are used as a guide to therapy. A change in PCW pressure of 4 mm Hg or greater in either direction typically requires some type of intervention and has been considered a change of a clinically significant magnitude by one nursing researcher (Hansen, 1976). Thus, a change of 4 mm Hg was used in grouping differences in the discussion of clinical significance.

Table 2 summarizes changes in PCW pressures seen in the 32 trials with position change. In 66 percent of the trials, no clinically significant change in PCW pressure

with lateral positioning was seen. In eleven trials, however, differences of a clinically significant magnitude were seen. These differences occurred in Patients 1, 2, 5, 6, and 8.

TABLE 2

Summary of Change in Pulmonary Capillary Wedge Pressure with Position Change for Eight Patients

Change	# Trials	% Trials
<4mm Hg	21	66%
4mm Hg	5	16%
5mm Hg	3	9%
5-10mm Hg	3	9%

Table 3 illustrates the frequency with which changes of a clinically significant magnitude occurred within four trials for each patient. Patient data is presented with mean PCW pressures ($\overline{PCW}$) in descending rank order. A mean difference between first and third position five minute PCW pressures (ΔPCW) indicates degree of patient stability through the trials. The three patients with the highest mean PCW pressures, all above 20.00 mm Hg showed the highest frequency of clinically significant change in PCW pressure (#PCW) with position change. Two subjects, Patients 6 and 8, showed the highest mean difference between

TABLE 3

Summary of Frequency of Changes in Pulmonary Capillary Wedge Pressure by Patient

Patient	$\overline{PCW}$[a]	# PCW[b]	Δ PCW[c]	Medication
6	34.25	3	5.5	Nitroprusside Furosemide
1	25.38	3	1.2	Digoxin
8	20.38	3	4.7	Dopamine
4	18.00	0	1.0	None
7	16.75	0	2.2	Gentamicin Chloramphenicol
3	9.50	0	3.5	None
2	9.00	1	1.0	Aminophylline Pitressin
5	6.13	1	1.2	None

[a] $\overline{PCW}$ represents the mean five minute PCW pressure, in mm Hg, across trials measured in the supine position.

[b] # PCW represents the number of trials in which the differences between supine and lateral PCW pressures was 4 mm Hg or greater.

[c] ΔPCW represents the mean differences between five minute PCW pressures in mm Hg measured in the first and third position.

first and third position five minute PCW pressures (ΔPCW) indicating the least stability. Patients 4, 7, and 3 whose PCW pressures were in the middle range showed no clinically significant effect of position change on PCW pressure. Patients 2 and 5 who had the lowest mean PCW pressures each showed a 4 mm Hg difference with position change in one trial. In summary 82 percent of the clinically significant differences occurred in the three patients with the highest PCW pressures.

While not statistically significant, differences between PCW pressures measured in the supine and lateral positions were clinically significant in 11 trials. These changes occurred most frequently in patients with the highest PCW pressures.

One explanation might be that lateral positioning causes an increase in left ventricular end diastolic volume. The effect on LVEDP would depend on the compliance of the left ventricle. In a relatively compliant heart characterized by a low or middle range LVEDP, a small change in left ventricular end diastolic volume might produce a small change in LVEDP. However, the same change in left ventricular volume in a relatively non-compliant left ventricle, as evidenced by a high range LVEDP, would produce a larger change in LVEDP.

A second explanation might be the length of equilibration period. That is, five minutes may not be sufficient in patients whose left ventricular function is severely impaired. Finally, the medications the patients were receiving may have affected equilibration time and therefore account for the results.

The second hypothesis addressed the difference between pressures measured at one and five minutes after position change. In the two factor analysis of variance, Factor B represented time after position change. Two F scores were significant for Factor B. The significant findings occurred in PCW pressures in trials one and three. Figure 2 illustrates these results. Mean population PCW pressures in trials one and three were lower in five minute readings than in one minute readings.

To identify further trends in differences between PCW pressures measured at one and five minutes after position change, data within individual patient trials were reviewed. Differences between one and five minute pressures were grouped into supine and lateral categories and were inspected for clinical significance. Differences of 4mm Hg or greater were considered to be clinically significant. Frequencies of clinically significant differences are presented in Table 4.

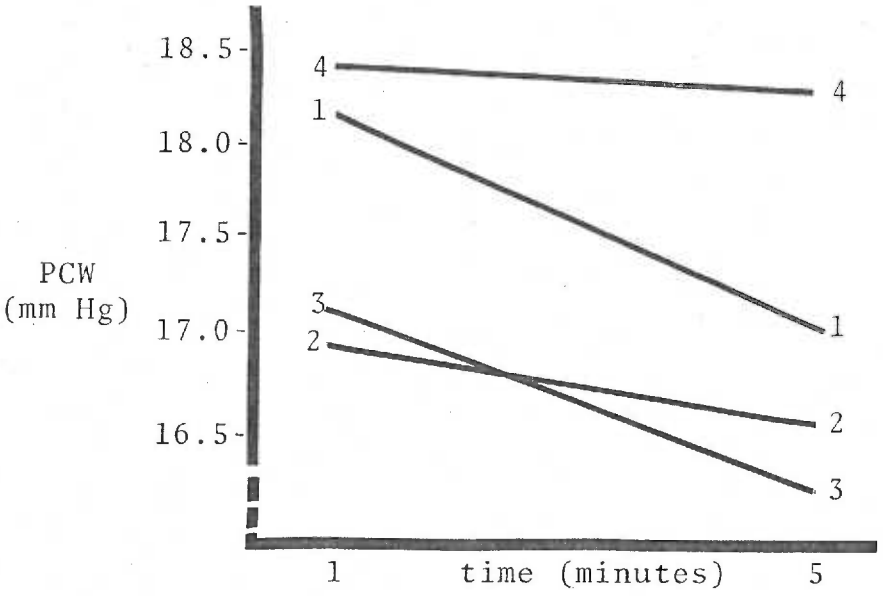

Figure 2. Mean Population Pulmonary Capillary Wedge Pressures at One and Five Minutes After Position Change by Trial.

The only clinically significant differences between one and five minute PCW pressures were seen in patients 6, 1, and 8 who had the highest mean PCW pressures. It is interesting that lateral pressure differences were more frequently clinically significant than their supine counterparts. The supine differences represent 4 of 20 (20%) pairs of PCW pressures while 8 of 16 (50%) of the lateral pairs showed a clinically significant difference.

To maximize the validity of PCW pressure readings, measurements should be made under consistent conditions. One such condition is the length of time allowed after position change for hemodynamic equilibrium to be

TABLE 4

Summary of Frequency of Differences Between One and Five Minute Pulmonary Capillary Wedge Pressures

Patient	$\overline{PCW}$[a]	Group	#dPCW-S[b]	#dPCW-L[c]	Total
6	34.25	C2	1	4	5
1	25.38	A1	1	1	2
8	20.38	C1	2	3	5
4	19.00	A2	0	0	0
7	16.75	B2	0	0	0
3	9.50	A2	0	0	0
2	9.00	A1	0	0	0
5	6.13	B1	0	0	0
Total			4	8	12

[a] $\overline{PCW}$ represents the mean five minute PCW pressure in mm Hg across trials measured in the supine position.

[b] #dPCW-S represents the number of clinically significant differences (i.e., 4mm Hg or greater) between one and five minute supine PCW pressures for each patient across trials.

[c] #dPCW-L represents the number of clinically significant differences (i.e., 4mm Hg or greater) between one and five minute lateral PCW pressures for each patient across trials.

established. In research protocols, in the study of critically ill patients, it is advantageous to measure pressures as quickly as possible after position change. Change in patient status is less likely, then, to confound the results. However, appropriate time must be allowed to insure hemodynamic equilibration.

A five minute equilibration period has been allowed after supine position change in a study of patients with acute myocardial infarction (Prakash, et al., 1973). In a sample of non-critically ill subjects, Woods and Mansfield (1976) did not allow five minutes for equilibration between supine position changes and pressure measurements. The results of the present study suggest that a five minute period of equilibration after supine-lateral position change is desirable in patients with high PCW pressures. In patients whose PCW pressure is in the medium or low range, findings indicate that valid measurements can be made one minute after position change.

One statistically significant time-position interaction ($p = 0.05$) occurred. The interaction was noted for pulmonary artery mean (PAM) pressures in trial two. This finding indicates that a unique effect of the combined time-position factors may exist. Since this finding did not directly affect the evaluation of a hypothesis no further analysis was made.

Hypothesis three predicted that there would be no difference between pulmonary artery diastolic (PAD) and pulmonary capillary wedge (PCW) pressures in this sample. No statistically significant correlation between five minute first position PAD and PCW pressures was seen in trial one. To further describe the sample, additional analysis was done.

Using five minute readings, mean individual PAD and PCW pressures in the supine and lateral positions were obtained (see Appendix D, p. 55). Patient 2 shows a 17.88 mm Hg difference between mean supine PAD and PCW pressures and a 16.50 mm Hg difference between pressures measured in the lateral position. In the remaining seven subjects differences between mean supine pressures range from 1.12 to 4 mm Hg. Mean lateral pressure differences ranged from 0.25 to 3.75 mm Hg. Figure 3 illustrates the individual patient differences.

When first position five minute PAD and PCW pressures were compared excluding data from Patient 2, a high correlation was seen ($r=0.87$, $p\leq 0.01$, $N=7$). Pairs of five minute trial one PAD and PCW in these seven patients were analyzed by position. Similar correlations between PAD and PCW pressures were seen for supine pressures ($r=0.94$, $p\leq 0.01$, $N=7$) and for lateral pressures ($r=0.90$, $p\leq 0.01$, $N=7$).

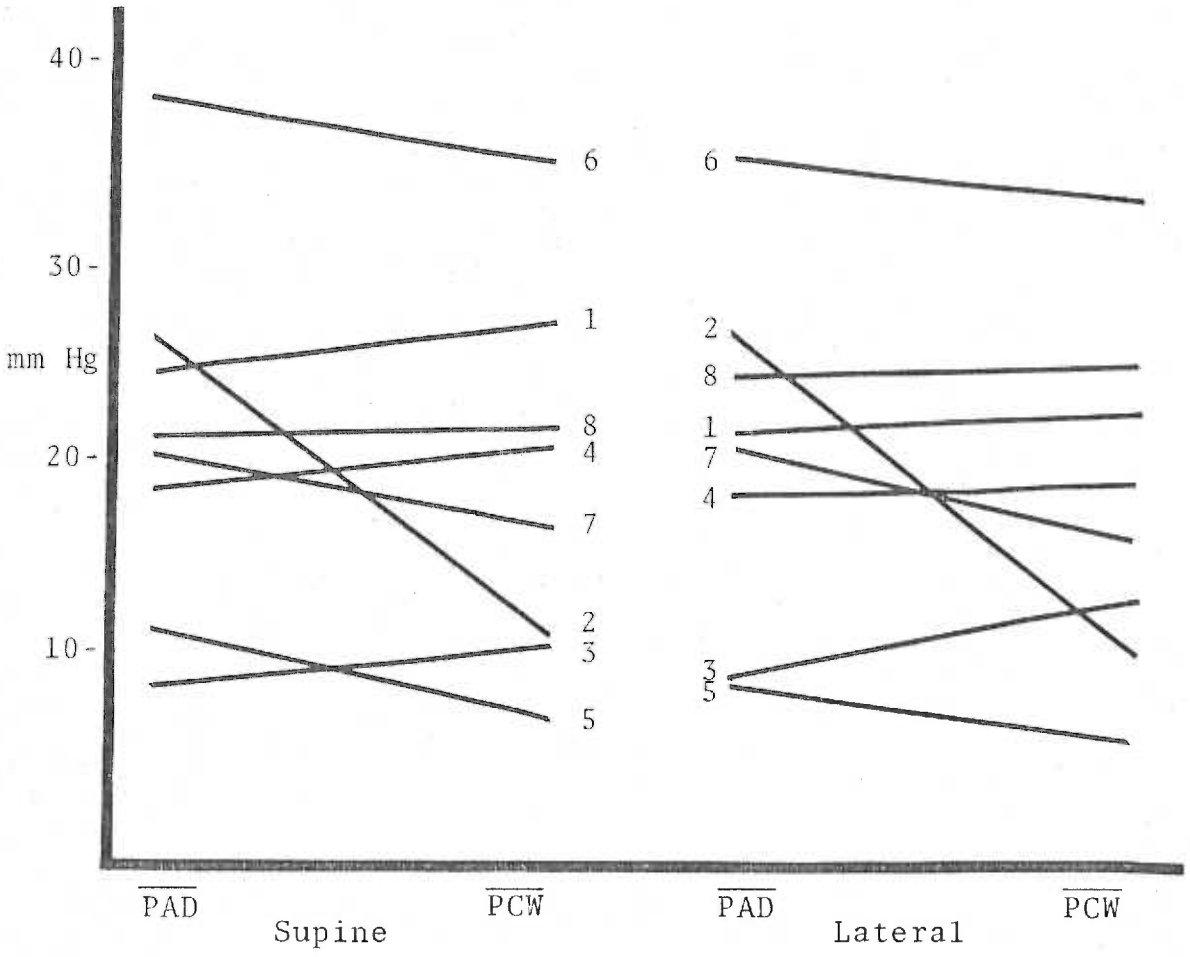

Figure 3. Comparison of Mean Five Minute Pulmonary Artery Diastolic and Pulmonary Capillary Wedge Pressures Measured in Supine and Lateral Positions in Eight Patients.

Interestingly, Patient 2 was receiving an intravenous infusion of Pitressin throughout all four trials. The pressor effect of Pitressin on smooth muscles (Goodman and Gilman, 1971) of the pulmonary vasculature can cause an increase in pulmonary vascular resistance and is one explanation for the discrepancy between PAD and PCW pressures in Patient 2.

The PAD pressure is sometimes used in lieu of PCW pressures to assess left ventricular function in the clinical setting. Falicov, et al. (1970) found a high correlation between the two pressures in 71 subjects with a wide range of left ventricular function in the absence of severe pulmonary vascular obstruction. In ten non-critically ill patients no clinically significant differences between PAD and PCW pressures were seen at 0, 20 and 45 degree supine backrest position (Woods and Mansfield, 1976).

The results of the present study of critically ill patients are in agreement with these findings. A good correlation between PAD and PCW pressures was found. Position did not alter the relationship; the correlation between pressures was similar in the supine ($r=0.94$, $p \leq 0.01$) and lateral ($r=0.90$, $p \leq 0.01$) position.

Although the findings of this study support previous research done in the area of position change and PA and PCW measurement, they differed from the literature with regard to time necessary for hemodynamic equilibration in patients with high PCW pressures. Generalizations drawn from the data of the study must be viewed tentatively because of the small sample size. Also, a multivariate analysis might reveal additional relationships.

CHAPTER FOUR

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

Position variation and sleep promotion are goals of nursing practice in the intensive care unit. Alternate positioning of critically ill patients promotes ventilation, circulation and comfort. At the same time provision for rest and sleep of a patient supports the natural maintenance and restorative processes of the body. These goals are often difficult to achieve because a patient's rest is frequently interrupted by repositioning for monitoring purposes.

Pulmonary artery (PA) and pulmonary capillary wedge (PCW) pressures have traditionally been made with the patient in the supine 0° backrest position. Because this position is infrequently maintained for any length of time by a patient in the intensive care unit, awakening and repositioning is frequently necessary for PA and PCW pressure measurement. Valid measurements made with the patient in a therapeutic position of comfort would yield information for monitoring hemodynamic status with minimal disturbance of the patient.

This study explored one such position, the lateral recumbent position. The effect of lateral positioning on PA and PCW pressures was assessed. Subjects were eight male patients admitted to a coronary care or medical intensive care unit. Control for site of catheter placement and sequence of positioning was built into the quasi-experimental modified time-series design.

The first hypothesis predicted the PA and PCW pressures measured in the supine and lateral position would not differ in the sample. To test the major hypothesis, data were subjected to two factor analyses of variance with one repeated measure. No statistically significant differences were found between pressures measured in the supine and lateral positions. However, clinically significant differences of 4mm Hg or greater were seen in some patients. The three patients with PCW pressures greater than 20mm Hg showed clinically significant variation in three of four trials.

The second hypothesis anticipated no difference between one and five minute PA and PCW pressures. The two factor analyses of variance indicated statistically significant differences between one and five minute PCW pressures in two of four trials. When one and five minute pressures were inspected for clinical significance, differences of 4mm Hg or greater were seen only in the three patients with the highest PCW pressures.

Hypothesis three predicted that pulmonary artery diastolic (PAD) pressures would not differ from PCW pressure in the supine and in the lateral position. No statistically significant correlation was found when data from the entire sample in both positions were analyzed. Inspection of individual differences revealed an approximate 17mm Hg difference between PAD and PCW pressures in one patient receiving an intravenous infusion of Pitressin. When data from this patient was excluded, a high correlation was seen between PAD and PCW pressures in the supine position ($r=0.94$, $p\leq 0.01$, $N=7$) in the lateral position ($r=0.90$, $p\leq 0.01$, $N=7$) both positions ($r=0.87$, $p\leq 0.01$, $N=7$).

Conclusions

The results of this study indicate that for patients whose PCW pressure is in the middle and low range, PA and PCW pressures can reliably be measured in the lateral position. Middle range PCW pressures in this sample were from 9.5 to 18.0mm Hg. Low range pressures were 9mm Hg or less. Variations of a clinically significant magnitude may occur in patients whose PCW pressure is in the high range. In this sample high range included pressures of 20 to 35mm Hg.

The findings suggest that in patients with PCW pressures in the medium or low range, immediate measurements of PA and PCW pressures after supine-lateral position change

will be valid. However, at least a five minute period of equilibration after position change was indicated for patients with PCW pressures in the high range.

Pulmonary artery diastolic pressures are sometimes used clinically instead of PCW pressures as an estimate of left ventricular end-diastolic pressure (LVEDP). The results of this study support this practice when patients are in the lateral or supine position. However, the small sample size and the analysis of the first two hypotheses must be taken into consideration if the conclusions are generalized.

Recommendations

Position change in a critically ill patient may affect the left ventricular end diastolic volume. The degree of this effect on left ventricular end diastolic pressure and therefore pulmonary capillary wedge pressure would then depend on the compliance of the left ventricle. It is therefore recommended that:

1. The range of pulmonary capillary wedge pressure be considered in further investigation of the effect of change in patient position on pulmonary artery and pulmonary capillary wedge pressure.

Time allowed for hemodynamic equilibration after lateral positioning may vary with degree of left ventricu-

lar dysfunction. To further investigate the findings of this study, it is recommended that:

2. Further investigation be done into the length of equilibration period necessary after position change in critically ill patients whose pulmonary capillary wedge pressures are 20mm Hg or greater.

The lateral ninety degree position is difficult to rigidly maintain in lethargic or obtunded patients. A more frequently used position clinically is the oblique position where the frontal plane of the patient is at a forty-five degree angle with the surface of the bed. For these reasons it is further recommended that:

3. The reference point for measuring pulmonary artery and pulmonary capillary wedge pressures in the oblique position be determined.

4. A study be done assessing the effects of oblique positioning on pulmonary artery and pulmonary capillary wedge pressure measurements using multivariate analysis.

REFERENCE LIST

Bendixen, H. H. Editorial Comment. Archives of Surgery, 1974, 109, 541.

Bolognini, V. The Swan-Ganz pulmonary artery catheter; Implications for nursing. Heart and Lung, 1974, 3, 976.

Braunwald, E., Ross, J. The ventricular end-diastolic pressure: Appraisal of its value in the recognition of ventricular failure in man. The American Journal of Medicine, 1963, 34, 147.

Burch, G. E. and Winsor, T. Phlebostatic axis and phlebostatic level; Reference levels for venous pressure measurements in man. Proceedings of the Society of Experimental Biologic Medicine, 1945, 58, 165.

Campbell, Donald T., Stanley, Julian C. Experimental and Quasi-experimental Designs for Research. Chicago, Rand McNally College Publishing, 1963.

Comroe, J. H. Physiology of Respiration (Second Edition), Chicago: Yearbook Medical Publishers, 1974.

Dement, W. and Kleitman, H. The relation of eye movement during sleep to dream activity: An objective method for the study of dreaming. Journal of Experimental Psychology, 1957, 53, 339.

Falicov, R.E. and Resnekov, L. Relationship of the pulmonary artery end-diastolic pressure to the left ventricular end-diastolic and mean filling pressures in patients with and without left ventricular dysfunction. Circulation, 1970, 42, 65.

Fisher, M. L., DeFelice, C. E., and Parisi, A. F. Assessing left ventricular filling pressure with flow-directed (Swan-Ganz) catheters; detection of sudden changes in patients with left ventricular dysfunction. Chest, 1975, 68, 542.

Forrester, J. S., Diamond, G., Chatterjee, K., Swan, H.J.C. Hemodynamic therapy of myocardial infarction. Part one. New England Journal of Medicine, 1976, 295, 1356.

Gernert, S. and Schwartz, S. Pulmonary artery catheterization. American Journal of Nursing, 1973, 73, 1182.

Goodman, L.S. and Gilman, A. The Pharmacological Basis of Therapeutics. (Fourth Edition), New York: MacMillan Co., 1971.

Gorlin, R. Current concepts: Practical cardiac hemodynamics. The New England Journal of Medicine, 1977, 296, 203.

Hansen, W.J. Effect of Body Position Upon Pulmonary Artery and Pulmonary Capillary Wedge Pressure in Acutely Ill Patients. Unpublished masters thesis, University of Washington, 1976.

Humphrey, C. B. An analysis of direct and indirect measurements of left atrial filling pressures. The Journal of Thoracic and Cardiovascular Surgery, 1976, 71, 643.

Hurst, J. W. The Heart. (Third Edition), New York: McGraw Hill, 1974.

Jeroes, R. Effect of lateral positioning on central venous pressure. Unpublished masters thesis, Loma Linda University, 1971.

Kleitman, N. Sleep and Wakefulness. (Third Edition), Chicago: University of Chicago Press, 1963.

Koella, W. P. Sleep: Its Nature and Physiologic Organization. Springfield: Charles C. Thomas, 1967.

Lappas, D., Lell, W. A., Gabel, C. G., Civetta, J. M. and Lowenstein, E. Indirect measurement of left-atrial pressure in surgical patients; pulmonary-capillary wedge and pulmonary-artery diastolic pressures compared with left-atrial pressures. Anesthesiology, 1973, 38, 394.

McFadden, E. H., and Giblin, E. C. Sleep deprivation in patients having open heart surgery. Nursing Research, 1971, 20, 249.

Prakash, R., Parmley, W., Dikshit, K., Forrester, J., and Swan, H. J. C. Hemodynamic effects of postural changes in patients with acute myocardial infarction. Chest, 1973, 64, 7.

Rackley, C., Russell, R. Left ventricular function in acute myocardial infarction and its clinical significance. Circulation, 1972, 45, 231.

Rahimtoola, S. H., Loeb, H. S., and Ehsani, A. Relationship of pulmonary artery to left ventricular pressures in acute myocardial infarction. Circulation, 1972, 46, 283.

Rahimtoola, S. H. Left ventricular end-diastolic and filling pressures in assessment of ventricular function (editorial). Chest, 1973, 63, 858.

Scheinman, M., Evans, T., Weiss, A., Rapaport, E. Relationship between pulmonary artery end diastolic pressure and left ventricular filling pressure in patients in shock. Circulation, 1973, 47, 3, 7.

Swan, H. J. C., Ganz, W., Forrester, J., Marcus, H., Diamond, G., Chonette, D. Catheterization of the heart in man with use of flow directed balloon-tipped catheter. New England Journal of Medicine, 1970, 283, 447.

Walker, B. B. The post surgical patient: Amount of uninterrupted time for sleep and rest during the first, second and third post operative days in a teaching hospital. Nursing Research, 1972, 21, 164.

Woods, N. F. Patterns of sleep in post-cardiotomy patients. Nursing Research, 1972, 21, 347.

Woods, S. L. and Mansfield, L. W. Effect of body position upon pulmonary artery and pulmonary capillary wedge pressures in noncritically ill patients. Heart and Lung, 1976, 5, 83.

Woods, S. L. Monitoring pulmonary artery pressures. American Journal of Nursing, 1976, 76, 1765.

Wright, J. Central Venous Pressure Extrathoracic Reference Point Measured on Postmortem Subjects. Unpublished masters thesis. Loma Linda University, 1974.

APPENDICES

APPENDIX A

INFORMED CONSENT FORMS

I, _____ , herewith agree to serve
(first name) (middle Name) (last name)
as a subject in the investigation named, "The effect of lateral positioning on pulmonary artery and pulmonary capillary wedge pressures" by Cathleen MurphyR.N., B.S. under the supervision of Barbara Gaines R.N., DEd. The investigation aims at improving the quality of nursing care in the intensive care unit.

The procedures to which I will be subjected are currently necessary for my nursing care. For the purpose of this study they will be done more frequently for an eight hour period. These procedures include being turned to a side lying position for an additional number of reading of blood vessel pressure measured through the catheter now in place.

Although I may not benefit directly form these extra measurements, I understand that they may help improve health care for other patients in intensive care units. The risks for me are as follows. I may experience some discomfort from the position. Additionally I may experience some shortness of breath. I understand that if this occurs I will immediately be repositioned.

The information obtained will be used for statistical analysis. My name will not appear on the study records and anonymity will be insured by the use of code numbers. Cathleen Murphy has offered to answer any questions that I might have about my participation in this study. I understand that I am free to refuse to participate or to withdraw from participattion in the study at any time without effect on my relationship with or treatment at the Portland Veterans Administration Hospital.

I have read the forehoing.

_____ _____
(date) (subjects signature)

_____
(witness's signature)

I, _____, herewith agree to allow my relative
(first name) (middle name) (last name)
_____ to serve as a subject in the investigation named "The effect of lateral positioning on pulmonsry artery and pulmonary capillary wedge pressures" by Cathleen Murphy R.N., B.S. under the supervision of Barbara Gaines R.N., DEd. The investigation aims at improving the quality of nursing care in the intensive care unit.

The procedures to which my relative will be subjected are currently necessary for his/her nusing care. For the purposes of this study they will be done more frequently for an eight hour period. These procedures include being turned to a side lying position for an additional number of readings of a blood vessel pressure measured through the catheter now in place.

Although my relative may not benefit directly from these extra measurements, I understand that they may help improve health care for other patients in intensive care units. The risks for my relative are as follows. He/She may experience some shortness of breath. Additionally he/she may experience some discomfort from the position. I understand that if this occurs my relative will immediately be repositioned.

The information obtained will be used for statistical analysis. My relative's name will not appear on the study records and anonymity will be insured by the use of code numbers. Cathleen Murphy has offered to answer any questions I might have about my relative's participation in this study. I understand that I am free to refuse to allow my relative to participate in or to withdraw from participation in the study at any time without effect on my relative's relationship with or treatment at the Portland Veterans Administration Hospital.

I have read the foregoing.

_____ _____
(date) (relative's signature)

_____
(witness's signature)

I, _____ , herewith agree to
(first name) (middle name) (last name)
serve as a subject in the investigation named, "The effect of lateral positioning on pulmonary artery and pulmonary capillary wedge pressures" by Cathleen Murphy R.N., B.S. under the supervision of Barbara Gaines R.N.,DEd. The investigation aims at improving the quality of nursing care in the intensive care unit.

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I have read the foregoing.

_____ _____
(date) (subject's signature)

_____
(witness' signature)

I, _____ , herewith agree to
(first name) (middle name)(last name)

allow my relative_____ to serve as a subject in the investigation named "The effect of lateral positioning on pulmonary artery and pulmonary capillary wedge pressures" by Cathleen Murphy R.N., B.S. under the supervision of Barbara Gaines R.N., DEd. The investigation aims at improving the quality of nursing care in the intensive care unit.

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The information obtained will be used for statistical analysis. My relative's name will not appear on the study records and anonymity will be in insured by the use of code numbers. Cathleen Murphy has offered to answer any questions I might have about my relative's participation in this study. I understand that I am free to refuse to allow my relative to participate in or to withdraw from participation in the study at any time without effect on my relative's relationship with or treatment at the University of Oregon Health Sciences Center.

I have read the foregoing.

_____
(relative's signature

_____
(date

_____
(witness's signature

APPENDIX B

SUMMARY OF F SCORES OF TWO FACTOR ANALYSIS OF VARIANCE

Summary of F Scores of Two Factor Analysis of Variance.

Trial	Pressure	SOURCE		
		A (position)	B (time)	AB (interaction)
1	PAS	0.00069	0.153	4.415
	PAD	0.011	2.441	1.793
	PAM	0.00088	0.115	2.886
	PCW	0.001	6.066*	o.123
2	PAS	0.049	2.539	0.036
	PAD	0.013	2.519	0.565
	PAM	0.001	0.623	4.718**
	PCW	0.021	0.22	3.767
3	PAS	0.014	1.423	0.777
	PAD	0.003	0.072	0.008
	PAM	0.006	0.169	1.523
	PCW	0.021	4.906***	0.127
4	PAS	0.005	3.33	1.267
	PAD	0.0004	0.219	0.457
	PAM	0.0001	0.021	0.085
	PCW	0.008	0.092	1.88

* p=0.05
** p=0.05
*** p=0.05

APPENDIX C

RAW DATA

Raw Data For Trials One and Two

Patient	Group	Position	trial															
			pressure	PAS		PAD		PAM		PCW		PAS		PAD		PAM		PCW	
			time	1	5	1	5	1	5	1	5	1	5	1	5	1	5	1	5
1	A-1	S		56	56	22	24	35	35	27	25	66	65	24	27	37	39	29	26
		L		57	51	21	18	36	31	25	20	60	56	18	19	31	30	--	20
		S		61	56	27	25	37	34	27	26	55	57	21	23	34	36	23	23
2	A-1	S		62	75	22	25	33	41	8	7	64	62	29	28	35	37	9	10
		L		74	70	27	26	42	38	13	11	61	65	21	24	34	36	8	9
		S		63	62	19	21	34	33	8	8	58	58	26	26	36	34	8	7
3	A-2	S		33	31	9	7	17	19	11	13	32	28	9	5	17	13	7	9
		L		31	33	11	8	20	19	13	11	30	28	8	9	13	13	8	8
		S		26	28	9	8	17	15	10	8	34	35	9	10	18	19	12	11
4	A-2	S		32	33	14	14	22	22	14	16	37	38	16	16	23	23	17	16
		L		36	31	18	13	23	21	17	15	41	40	18	18	26	25	18	18
		S		33	34	15	16	21	22	16	16	37	37	16	16	23	23	17	17
5	B-1	L		35	32	12	12	18	18	8	8	30	30	10	10	15	17	7	7
		S		34	32	9	9	15	14	4	4	36	35	8	8	16	15	10	7
		L		34	35	10	12	18	19	9	9	28	28	7	8	13	13	4	4
6	D-2	L		69	65	35	32	44	43	33	36	73	75	37	37	48	48	40	33
		S		66	74	37	40	45	51	35	32	73	73	33	37	48	47	35	41
		L		70	67	36	31	48	44	30	31	74	71	36	37	48	45	37	30
7	B-2	L		36	38	21	22	27	28	18	18	31	29	20	18	24	22	16	14
		S		34	36	21	20	25	24	17	16	31	28	19	18	23	23	18	17
		L		36	35	20	19	26	25	17	16	31	31	20	19	25	24	18	17
8	D-1	L		43	46	26	27	33	32	22	21	34	36	17	18	26	26	15	15
		S		44	40	31	28	35	36	28	25	46	43	24	25	33	31	26	25
		L		39	41	20	23	24	30	21	22	40	40	20	20	26	30	31	25

Raw Data For Trials Three and Four

Patient	Group	Position	trial	1								2							
			pressure	PAS		PAD		PAM		PCW		PAS		PAD		PAM		PCW	
			time	1	5	1	5	1	5	1	5	1	5	1	5	1	5	1	5
1	A-1	S		68	69	28	26	39	39	27	28	55	56	21	21	5	33	24	23
		L		56	60	22	24	34	33	20	23	58	53	21	20	33	--	23	22
		S		65	61	29	25	40	37	31	28	62	58	27	23	39	35	28	24
2	A-1	S		58	59	25	29	35	35	10	10	65	62	31	29	38	37	9	10
		L		62	63	29	28	37	38	11	11	69	65	31	29	38	38	11	10
		S		60	60	29	28	37	37	11	10	60	63	29	29	36	38	10	10
3	A-2	S		23	25	8	7	14	14	10	9	28	29	9	9	14	16	9	9
		L		22	29	7	7	12	16	10	8	30	33	8	9	16	17	11	11
		S		32	33	9	10	19	20	13	13	34	39	11	9	19	18	12	12
4	A-2	S		38	37	17	16	22	22	15	15	44	40	23	23	30	28	21	21
		L		42	40	20	19	28	26	19	18	44	41	23	20	30	28	22	21
		S		39	--	16	--	22	--	18	17	40	38	20	23	28	29	20	20
5	B-1	L		28	27	7	9	13	11	5	4	31	32	10	10	15	16	6	6
		S		30	30	6	6	11	12	2	2	33	25	9	8	16	13	3	3
		L		28	28	9	9	14	13	5	4	30	31	9	11	15	15	6	7
6	D-2	L		70	68	35	35	46	43	30	29	73	72	40	39	52	50	36	40
		S		68	68	34	35	43	44	32	29	70	64	32	39	49	49	37	35
		L		70	68	35	37	45	45	37	33	65	71	32	34	42	44	30	30
7	B-2	L		33	34	20	20	24	22	16	15	34	35	22	23	26	26	19	19
		S		31	31	20	19	23	22	16	16	34	33	22	22	26	24	18	18
		L		33	32	21	20	26	24	18	17	34	32	20	19	26	25	19	17
8	D-1	L		30	35	18	20	24	26	23	18	38	35	22	24	28	29	23	24
		S		37	34	22	20	29	25	23	18	40	41	22	24	29	32	23	28
		L		29	31	17	19	22	24	16	20	31	40	19	22	26	32	21	18

APPENDIX D

SUMMARY OF INDIVIDUAL MEAN PULMONARY ARTERY DIASTOLIC PRESSURE AND MEAN PULMONARY CAPILLARY WEDGE PRESSURE

Summary of Individual Mean PAD Pressures in Supine and Lateral Positions at One and Five Minutes.

Position	Supine		Lateral	
Subject	1 minute	5 minute	1 minute	5 minute
1				
mean	24.88	24.25	20.50	20.25
SD	3.27	1.91	1.73	2.63
2				
mean	26.25	26.88	27.00	26.75
SD	4.10	2.80	4.32	2.22
3				
mean	8.50	8.25	9.13	8.13
SD	1.73	0.96	0.84	1.73
4				
mean	19.75	17.50	17.29	17.71
SD	2.36	3.11	3.15	3.68
5				
mean	9.25	10.13	8.00	7.75
SD	1.67	1.46	1.41	1.26
6				
mean	34.00	37.75	35.75	35.25
SD	2.16	2.22	2.25	2.77
7				
mean	20.50	19.75	20.50	20.00
SD	1.29	1.71	0.76	1.69
8				
mean	19.88	21.50	24.75	24.25
SD	3.00	3.07	4.27	3.30

Summary of Individual Mean PCW Pressures in Supine and Lateral Positions at One and Five Minutes.

Position	Supine		Lateral	
Subject	1 minute	5 minute	1 minute	5 minute
1				
mean	27.00	25.38	22.67	21.67
SD	2.56	1.10	2.52	1.53
2				
mean	9.13	9.00	10.75	10.25
SD	1.05	1.32	2.06	0.96
3				
mean	10.50	9.50	10.50	10.50
SD	2.08	1.73	1.93	2.00
4				
mean	19.00	18.00	17.25	17.25
SD	2.23	2.45	2.38	2.12
5				
mean	6.25	6.13	4.75	4.00
SD	1.67	1.96	3.60	2.16
6				
mean	34.75	34.25	34.13	32.75
SD	2.03	5.12	3.91	3.69
7				
mean	17.25	16.75	17.63	16.63
SD	0.48	0.96	1.19	1.60
8				
mean	20.25	20.38	25.00	24.00
SD	2.86	3.34	2.44	4.24

AN ABSTRACT OF THE THESIS OF

CATHLEEN E. MURPHY

for the Master of Nursing

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Title: THE EFFECT OF LATERAL POSITIONING ON PULMONARY ARTERY AND PULMONARY CAPILLARY WEDGE PRESSURES IN CRITICALLY ILL PATIENTS

Approved [redacted]
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Valid measurement of pulmonary artery and pulmonary capillary wedge pressures made with the patient in a therapeutic position of comfort yields information for monitoring hemodynamic status with minimal disturbance of the patient. This study explored the effect of one such position, the lateral recumbent, on pulmonary artery and pulmonary capillary wedge pressures. Additionally, the effect of time after position change on pressures was assessed. Finally, the correlation of pulmonary artery diastolic pressure with pulmonary capillary wedge pressure was investigated.

Subjects were eight male patients admitted to a coronary care or medical intensive care unit and selected by criteria. Control for sequence of positioning and site of catheter placement was built into the quasi-experimental modified time series design. Each trial included pressures taken at one and five minutes after three position manipulations. Four trials per patient were made.

No statistically significant differences were found between pressures measured in the supine position and those measured in the lateral position when data were subjected to two factor analyses of variance. However, clinically significant differences of 4mm Hg or greater were seen between supine and lateral pulmonary capillary wedge pressures. Eighty-two percent of the differences were found in the three patients whose pulmonary capillary wedge pressures were greater than 20mm Hg. These results suggested that in most cases pulmonary artery and pulmonary capillary wedge pressures can be measured accurately in the lateral position. However, findings indicated that the hemodynamic response to lateral position change was dependent on left ventricular function. It was concluded that in patients with severely impaired left ventricular function, as indicated by a pulmonary capillary wedge pressure of 20mm Hg or greater, clinically significant differences between pulmonary capillary wedge pressures measured in the supine

position and those measured in the lateral position may be seen. Differential effect of altered left ventricular volume on pulmonary capillary wedge pressure and/or increased time needed for hemodynamic equilibration after position change were suggested mechanisms for differences seen in this segment of the critically ill patient population.

The two factor analyses of variance indicated significant differences between pulmonary capillary wedge pressures measured at one and five minutes after position change. Upon inspection of the data these differences were seen only in the three patients with pulmonary capillary wedge pressures greater than 20mm Hg. It was concluded that pulmonary artery systole, pulmonary artery diastolic, pulmonary artery mean and pulmonary capillary wedge pressures can reliably be measured at one and/or five minutes after position change in patients whose pulmonary capillary wedge pressure is less than 20mm Hg.

A high correlation was seen between pulmonary artery diastolic and pulmonary capillary wedge pressure ($r=0.87$, $p \leq 0.01$). These findings supported the interchangeable use of the two indicators in assessing left ventricular function. Recommendations for further study were made.