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Effect of the 30-Degree Lateral Recumbent Position on Pulmonary Artery and Pulmonary
Artery Wedge Pressures in Critically Ill Adults

by

Elizabeth Joan Bridges

A dissertation submitted in partial fulfillment
of the requirements for the degree of

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Abstract

Effect of the 30-Degree Lateral Recumbent Position on Pulmonary Artery and Pulmonary
Artery Wedge Pressures in Critically Ill Adults

by Elizabeth Joan Bridges

Chairperson of the Supervisory Committee: Professor Susan L Woods

Department of Biobehavioral Nursing and Health Systems

A common therapeutic nursing intervention is patient positioning. Despite demonstrated benefits of lateral positioning, critically ill patients may require prolonged supine positioning in order to obtain reproducible hemodynamic measurements. The rationale for supine positioning is based on research that suggests that pulmonary artery (PA) pressure measurements in the lateral and supine positions are different. However, this research was limited by the lack of an angle-specific left atrial (LA) reference, which may have introduced systematic measurement error. It was unknown if PA pressure measurements in the 30-degree lateral position, using an angle-specific LA reference, were similar to supine measurements.

To determine the effect of 30-degree right and left lateral positions on PA and PAW pressures in critically ill adults, 35 post-cardiac surgery patients, aged 27 to 89 (mean 63.6 ± 11 years) were positioned in supine, right- and left-lateral positions. The patients served as their own controls. PA and PAW pressures were measured in each position.

Data analysis included analysis of variance for repeated measures, post-hoc paired t-tests, and comparison to baseline pressure fluctuations. On average, PAS, PAED and PAM pressures in the left-lateral position were significantly different ($p < .05$) from supine pressures. The largest position-related pressure difference occurred in the PAS (2.0 ± 2.1 mm Hg), while the maximum PAED and PAW pressure differences were 1.4 ± 2.7 mm Hg and 1.6 ± 2.4 mm Hg, respectively. Clinically significant position-related pressure changes occurred in 12 of 581 pressure-pairs (2.1%). The changes occurred in six patients. Two patients had significant PAED pressure changes (five pressure-pairs, four attributed to one patient). Only one patient had a clinically significant change in PAW pressure. There were no demographic or clinical characteristics that differentiated between patients with and without significant pressure changes.

This was the first large study of the effect of 30-degree lateral position with no backrest elevation on PA and PAW pressures, using a validated LA reference point. Results indicate that in hemodynamically stable cardiac surgery patients during the first 24 postoperative hours, PA and PAW pressures measured in the 30-degree lateral and supine positions are interchangeable.

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DEDICATION

This dissertation is dedicated to Mom and Dad and all my family; for your unending support.

“It is good to have an end to journey toward but it’s the journey that matters in the end.”

-Ursula LeGuin

CHAPTER I

INTRODUCTION AND PROBLEM STATEMENT

Since its introduction in 1970 (Swan et al., 1970), invasive hemodynamic monitoring with a pulmonary artery (PA) catheter has become one of the most commonly used diagnostic tools available in critical care (Ginosar & Sprung, 1996). Although there is some controversy regarding this widespread use, management of patients based on information obtained from the PA catheter has been shown to improve outcomes in patients with complicated myocardial infarction, refractory congestive heart failure, and high-risk vascular surgery (Hollenberg & Hoyt, 1997; Pulmonary Artery Consensus Conference Participants [PACCP], 1997). In addition, data obtained from the PA catheter improves diagnostic accuracy in patients with respiratory failure (Connors, McCaffree, & Gray, 1983; Eisenberg, Jaffe, & Schuster, 1984). In light of the frequent use of the PA catheter, and the potential for improved diagnostic accuracy and outcomes, the American Association of Critical Care Nurses (AACN) Consensus Conference on Research gave high priority to research related to the effect of nursing interventions on hemodynamic indices (Lindquist et al., 1993).

A common nursing intervention performed in the care of a critically ill patient is patient positioning. Lateral positioning is performed to prevent the negative effects of prolonged bedrest, such as decubitus ulcer formation. Lateral positioning has also been shown to improve oxygenation in patients with unilateral lung disease. Finally, therapeutic positioning has been shown to decrease complications (fever, atelectasis) and length of stay in cardiac surgery patients. Despite adequate literature to support

therapeutic lateral positioning, many critically ill patients are either left in a supine position or are frequently disturbed for placement into a supine position in order to obtain reproducible hemodynamic measurements.

The rationale for supine positioning during the performance of hemodynamic measurements is the need to reference and level the PA catheter system to the left atrium. Previously, reference points were available only for the supine and 90-degree lateral positions. A catheter system referenced below the left atrium reports a measured pressure that includes a hydrostatic component; thus, the reported pressure overestimates the actual intracardiac pressure. Conversely, a catheter system referenced above the left atrium results in a reported pressure that underestimates actual intracardiac pressure (O'Quin & Marini, 1983).

In a study of the 90-degree lateral position on PA pressures using an angle-specific left atrial (LA) reference, Bryant and Kennedy (Bryant & Kennedy, 1982; Kennedy, Bryant, & Crawford, 1984), found no clinically or statistically significant ($p > .05$) differences. Twelve other studies of the effect of 20- to 60-degree lateral position, which used a variety of non-angle specific LA references, on PA pressures were found (Aitken, 1995; Briones, Dickenson, & Bieberitz, 1991; Cason, Lambert, Holland, & Huntsman, 1990; Groom, Frisch, & Elliott, 1990; Guenther, Kay, Cheng, & Lauer, 1987; Keating, Boylard, Eichler, & Reed, 1986; Lange, Katz, McBride, Moore, & Hillis, 1988; Murphy, 1977; Osika, 1989; Ross & Jones, 1995; Whitman, 1982; Wild, 1983). In all cases with rotation greater than 20-degrees, there were clinically and statistically significant differences in

PA pressures ($p < .05$). There may be physiologic changes that occur in the lateral position that cause these results. However, it is impossible to rule-out the introduction of systematic error into the measured pressures as a result of the various reference points above or below the left atrium (midsternum, phlebostatic axis) that were used. Therefore, it was imperative to control for this potential source of error before further study of the physiologic effects of lateral position on PA pressure measurements was undertaken.

Recently, VanEtta and colleagues (1993) identified the LA reference point for the 30-degree lateral position. Only one study, which was conducted by Duke and colleagues (1994), could be found that used this reference point. Five of the six critically ill patients studied in this pilot study did not demonstrate clinically significant PA pressure differences in the 30-degree lateral versus supine position. However, one subject had dramatic pressure differences in the lateral position relative to the supine measurements. Unfortunately, analysis of these results was limited by the small sample size.

Therefore, it was not known if PA pressure measurements obtained from a patient in a 30-degree lateral position, using an angle-specific LA reference point, were similar to those obtained in the supine position. If there were no differences in PA pressures measured in the two different positions, then critically ill patients could be monitored, while left undisturbed to enjoy the benefits of therapeutic lateral positioning.

CHAPTER II

CONCEPTUAL FRAMEWORK AND SIGNIFICANCE

This chapter presents an overview of the intrinsic physiologic factors that affect cardiac output (CO) and the basic principles related to the measurement of these factors using a PA catheter. To provide an understanding of the relationship between PA pressure monitoring and lateral position, the following are discussed: (1) The therapeutic benefits of lateral positioning; (2) the effect of lateral positioning on PA pressure measurements, and the need to identify an angle-specific LA reference point; and (3) a discussion of the previous research related to position-induced changes in PA pressure. The chapter concludes with a summary and a statement of purpose of the study.

Cardiac Output

Cardiac output is a key determinant of arterial blood pressure (ABP) and the delivery of oxygen to the tissues. Cardiac output, which is defined as the volume of blood ejected from the heart per minute, is the product of stroke volume (SV) and heart rate (HR). Stroke volume is the volume of blood ejected from the heart with each beat. Three factors: preload, afterload, and contractility are the major intrinsic variables that affect SV. The relationship between CO, HR, SV, and the factors that affect SV are demonstrated graphically in Figure 1.

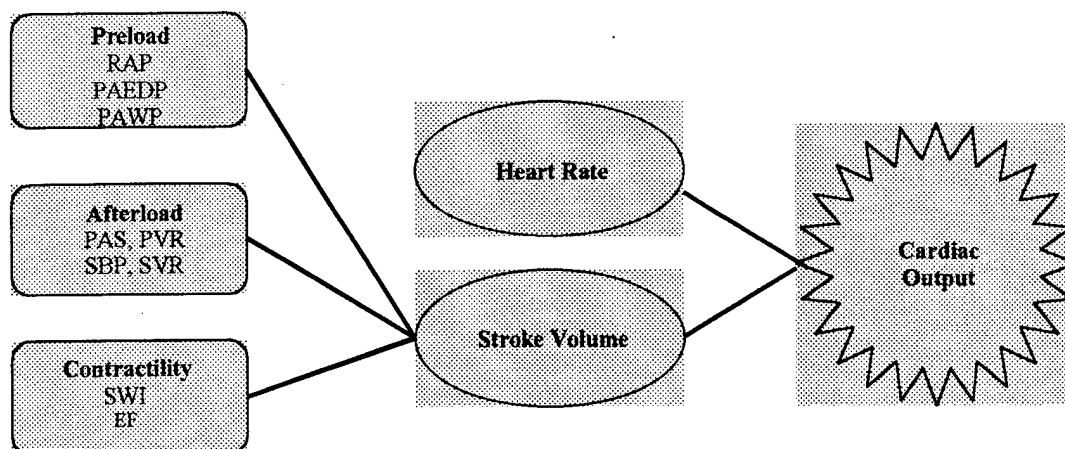


FIGURE 1. Graphic representation of the relationship between CO, HR, and SV, the factors that affect SV (preload, afterload, and contractility), and the most common indices of these factors. RAP = right atrial pressure, PAEDP = pulmonary artery end-diastolic pressure, PAWP = pulmonary artery wedge pressure, PAS = pulmonary artery systolic pressure, PVR = pulmonary vascular resistance, SVR = systemic vascular resistance, SWI = stroke work index, EF = ejection fraction.

Preload

At the level of the muscle fiber, preload is defined as the force acting to stretch the ventricular fibers at end-diastole (Sonnenblick, 1962). Preload is related to CO by the Frank-Starling Law of the Heart, which states that an increase in myocardial muscle length is associated with an increase in the force of contraction (Sonnenblick, 1962; Starling, 1918). The increased force of contraction results in an increase in SV and CO (Hedges, 1983). This relationship can be visualized using a ventricular function curve (Figure 2), which demonstrates that within limits, for any given afterload, an increase in preload is associated with an increase in SV or CO (Sarnoff, 1955; Weber, Janicki, Reeves, Hefner, & Reeves, 1974).

Afterload

In muscle-fiber experiments, preload is the tension in the muscle prior to contraction, and afterload is the additional tension that develops in the muscle during contraction before shortening occurs (Hedges, 1983; Sonnenblick, 1962). At the level of ventricle, afterload is defined as ventricular-wall tension during the shortening phase of contraction, and reflects the sum of the forces against which the ventricle must act to eject blood. However, given the varied angles of the contractile fibers and the torsion of the ventricle during systole, a single measure of ventricular wall tension is inadequate to define afterload (Huntsman & Feigl, 1989). In the intact system in vivo, afterload is defined as the pressure in the aorta during systole. Aortic compliance, SVR, CO, and reflection of the pressure wave from the periphery influence aortic systolic pressure.

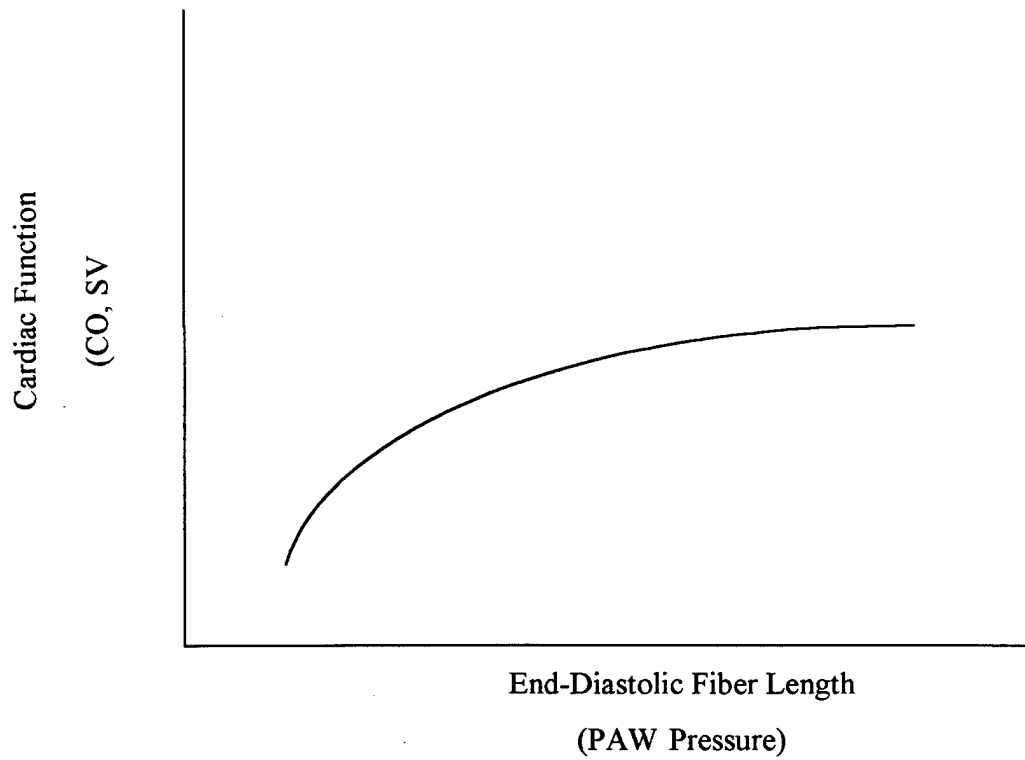


FIGURE 2. Ventricular function curve demonstrating the effect of increased end-diastolic fiber length (as indicated by increased PAW pressure) on cardiac function (as indicated by CO or SV).

The last concept, pressure wave reflection occurs as the arterial pressure pulse travels out into the body and reaches the level of the arterioles, where a portion of it is reflected upstream in a retrograde manner. This retrograde wave collides with the anterograde pressure waves. The collision results in an augmentation of the peak pressure (O'Rourke, 1992; O'Rourke & Kelly, 1993). In hypertension, the pressure pulse is rapidly reflected from the periphery and results in augmentation of pressure during systole; thus it increases afterload (Kelly et al., 1990; O'Rourke, 1993; Simkus & Fitchett, 1990; Westerhof & O'Rourke, 1995). In younger individuals, arterial pressure wave reflection occurs more slowly and pressure augmentation occurs during diastole, and is thus advantageous for coronary perfusion. Knowledge and understanding of pressure wave reflection and the other factors that affect afterload is important in the differentiation of various effects these factors may have on afterload and thus cardiac function.

As described by the force-velocity relation, for any given preload there is an inverse relationship between afterload and muscle shortening, and thus SV (Huntsman & Feigl, 1989; Ross, 1976; Ross, Covell, Sonnenblick, & Braunwald, 1966; Weber et al., 1974). Although this relationship is observed in an isolated muscle fiber, it is not clinically apparent in vivo in individuals with normal cardiac function. In individuals with normal cardiac function, an increase in afterload results in an acute decrease in SV, and a subsequent increase in end-diastolic volume (preload). As described by the length-tension relationship, the increased preload results in an increase in SV; thus, a new steady state is reached, characterized by an increase in preload and afterload, without a change

in SV (Ross, 1976). However, in individuals with a chronically depressed inotropic state (e.g., heart failure, cardiomyopathy), a steady state with altered ventricular dimensions (hypertrophy, dilatation) and maximal employment of the length-tension relation occurs. Thus, in these individuals in the face of an increase in afterload, the reserve provided by the length-tension relationship is exhausted, and SV decreases acutely (Ross, 1976). These findings help to explain the use of afterload-reducing agents in patients with heart failure.

Of importance to this study is whether the post-cardiopulmonary bypass/cardiac surgery heart functions in a manner similar to a failing heart relative to a change in afterload, and if so, for how long after surgery. In patients with normal preoperative ventricular function, ventricular dysfunction occurred within the first four hours post-cardiopulmonary bypass, with a return to preoperative levels occurring over 4 to 48 hours postoperative (Breisblatt et al., 1990; Mangano, 1985; Phillips et al., 1983; Royster, 1993; Vinten-Johansen & Nakanishi, 1993). In patients with abnormal ventricular function, defined as preoperative ejection fraction less than 45% or contractile dyssynergy, the depressed function lasted for at least 24 postoperative hours, and the return to baseline was more prolonged (Mangano, 1985). Thus, patients during the postoperative period, particularly those with pre-existing ventricular dysfunction, may be sensitive to acute changes in afterload.

Contractility

Contractility refers to the intrinsic properties of cardiac myocytes that reflect the activation, formation, and cycling of cross-bridges between actin and myosin filaments (Grossman, 1996). At a constant preload and afterload, an increase in contractility results in greater extent and velocity of shortening (Opie, 1997; Sonnenblick, 1962). In addition, at any given muscle length, an increase in the frequency of contraction or changing the chemical environment (calcium, catecholamines) alters contractility (Sonnenblick, 1962). In the intact heart, a change in contractility is defined as an alteration in cardiac performance that is independent of preload and afterload.

Heart Rate

The effect of HR on CO is defined by the equation:

$$\text{Heart Rate} \times \text{Stroke Volume} = \text{Cardiac Output}$$

Depending on the level of analysis (i.e., myocardial fiber versus intact cardiovascular system), the effect of HR on cardiac function is variable. At the level of the myocardial fiber an increase in HR is associated with an increase in contractility (treppe effect), independent of preload or afterload (Huntsman & Feigl, 1989; Lendrum, Feinberg, Boyd, & Katz, 1960; Opie, 1997; Sonnenblick, 1962). However, in the intact system, the effect of a HR induced increase in contractility on SV and CO is offset by decrease in ventricular filling time (Bevegård, Jonsson, Karlof, Lagergren, & Sowton, 1967; Janicki, Sheriff, Robotham, & Wise, 1996), and the translocation of blood into the peripheral vasculature (Rowell, 1993; Sheriff, Zhou, Scher, & Rowell, 1993). The translocation of

blood results in a decrease in RA pressure and thus SV and CO (Janicki et al., 1996; Rowell, O'Leary, & Kellogg, 1996; Sheriff et al., 1993). For example, a pacemaker induced increase in HR from an idioventricular rate of 30 beats per minute up to approximately 70 beats per minute was associated with an increase in CO. However, a further increase in HR up to 160 beats per minute was associated with a 70% decrease in LV filling time, and the CO remained the same or decreased (Bevegård et al., 1967). This inverse relationship between HR and RA pressure is of practical importance when attempting to increase CO and ABP by increasing HR with a pacemaker, particularly in a patient who is preload dependent (Lancon, Pillet, Gabrielle, Fayollle, & Tatou, 1994; Schaefer et al., 1988).

Ventricular Function Curves

The interaction between preload, afterload, and contractility and the effect of various disease processes (heart failure, hemorrhage) and therapeutic actions (vasodilator or inotropic drug therapy) can be visualized in a family of ventricular function curves (Figure 3) (Sarnoff, 1955). These curves demonstrate three primary effects: (1) A change in preload is represented by movement up and down a single curve. For example, an increase in preload related to volume infusion results in movement from point A to point B. It can also be seen that above a certain filling pressure there is no further increase in SV or CO. However, the curves do not demonstrate a descending limb (decrease in SV for further increases in preload). (2) A change in contractility is represented by an upward or downward shift of the curve. For example, administration of an inotropic

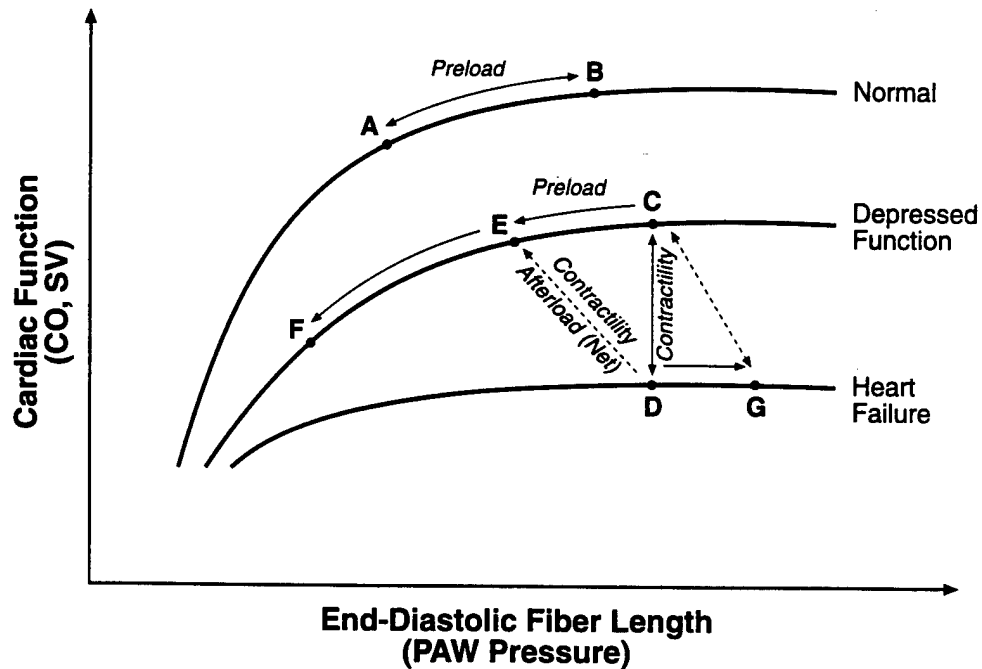


FIGURE 3. Family of ventricular function curves. Idealized ventricular function curves representing normal, depressed, and severely depressed function. Point A to Point B and Point B to A reflect an increase and decrease, respectively, in preload. Point D to E reflects the net effect of a decrease in afterload on a failing heart. This upward and lateral shift reflects two actions: Point D to C an increase in force of contraction and Point C to E a decrease in preload due to increased systolic ejection. This shift appears similar to an increase in contractility. A downward shift in the entire curve reflects a decrease in contractility, that is, for any given preload and afterload, the CO is decreased. In a failing heart, an additional effect of decreased contractility is an increase in preload due to decreased systolic ejection, thus the net effect of a decrease in contractility is to shift the curve down and to the left (Point C to G).

agent results in an upward shift of the entire curve; while depression of cardiac function is represented by a downward shift. (3) A change in afterload results in a shift in the curve that appears similar to that caused by a change in contractility. For example, in a patient with depressed cardiac function, a reduction in afterload will have two results. First, as defined by the force-velocity relationship, a decrease in afterload will result in an increase in the velocity of contraction (increased SV) (Point D to Point C). In addition, as a result of the increased SV, the end-diastolic volume will decrease (Point C to Point E). The net effect of the increased force of contraction and decreased afterload is reflected by shift of the curve up and to the left (Point D to Point E). The effect of afterload reduction on SV (Point F) is dependent upon the filling pressures. Afterload reduction in a patient with increased filling volumes and decreased cardiac function will result in a shift in the curve up and to the left (Point D to Point B). However, afterload reduction in a patient with decreased filling pressures will result in either no change or a decrease in SV, because as noted the reduction in afterload is also associated with a decrease in preload. Thus, the patient would move down the ascending limb of the curve, as defined by the Frank-Starling mechanism.

Measurement of Hemodynamic Indices

Principles of PA Pressure Measurement

The flow-directed PA catheter, which was introduced into clinical practice in 1970 (Swan et al., 1970), provides a method for the clinical measurement or derivation of indices of preload, afterload, and contractility. The PA catheter (Figure 4), which is a

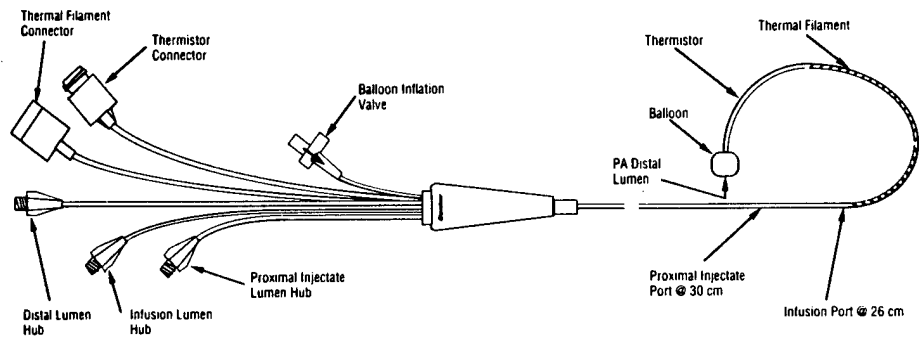


FIGURE 4. Continuous cardiac output pulmonary artery catheter. (Courtesy of Baxter Healthcare Corporation, Edwards Critical-Care Division).

multilumen catheter, is inserted percutaneously through a central vein. Once the catheter reaches the right atrium, the 1.5 ml balloon on the distal end of the catheter is inflated, and the catheter is "floated" through the right atrium and ventricle, and out into a branch of the PA where it wedges (Swan & Ganz, 1975; Wiedemann, Matthay, & Matthay, 1984). Correct positioning of the catheter is determined by evaluation of the waveforms associated with the right atrium and ventricle, and PA (Figure 5), and chest radiograph interpretation.

The RA pressure is obtained by interpretation of a pressure waveform transmitted through the proximal port of the PA catheter. The PA systolic, diastolic, and mean pressures are transmitted through the distal port with the balloon deflated. The PAW pressure is transmitted through the distal port when the balloon is inflated, and the catheter is wedged in a small branch of the PA. Wedging of the PA catheter results in a static, continuous vascular segment between the catheter tip and the left atrium. The catheter records the pressure at the junction (J point) of the static column and flowing venous channels. Because there are no valvular structures or obstructions between the J-point and the left atrium, at end-diastole when the mitral valve is open there is essentially an uninterrupted vascular segment between the tip of the catheter located in the PA and the left ventricle (Figure 6) (Leatherman & Marini, 1993; O'Quin & Marini, 1983). It is the presence of this continuous column of blood that allows the PAW pressure to be used as an indirect indicator of LV preload (Leatherman & Marini, 1993; O'Quin & Marini, 1983).

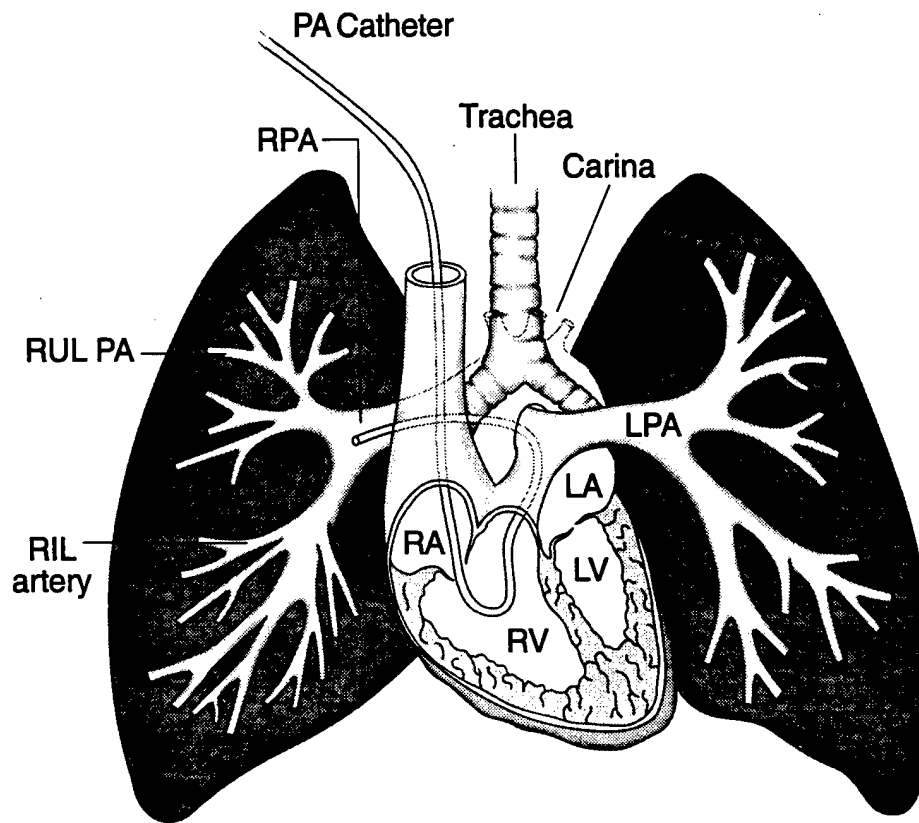


FIGURE 5. Schema of the cardiopulmonary structures demonstrating the relationship between the left atrium, pulmonary artery (PA) and pulmonary vasculature, with a correctly positioned PA catheter. RUL PA: Right upper lobe pulmonary artery, RIL Right interlobar pulmonary artery, RPA: Right pulmonary artery, LPA: Left pulmonary artery, RA: right atrium, LA Left atrium, RV: Right ventricle, LV: Left ventricle.

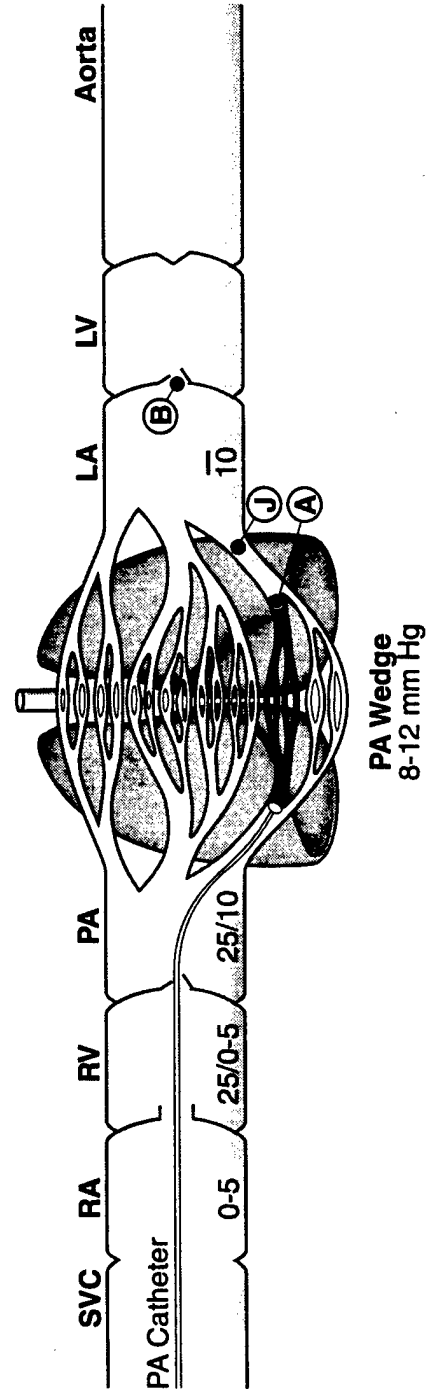


FIGURE 6. Schema of the principle underlying the use of PAW pressure as an indicator of LV preload. When the inflated balloon on the PA catheter obstructs arterial flow, the catheter will record the pressure at the junction of the static column of fluid and the flowing venous channels (J point). The PAW pressure underestimates the pulmonary capillary pressure (P_{cap}) when there is increased resistance in the post-capillary vessels proximal to the J-point (Point A). The PAW overestimates LVED pressure in the presence of obstruction distal to the J-point (Point B) (e.g., mitral stenosis, LA myxoma), while the PAW underestimates the LVED pressure in the presence of premature closure of the mitral valve as a result of aortic insufficiency (Leatherman & Marini, 1993; O'Quin & Marini, 1983).

Indices of Preload

Clinically, the RA pressure measures right heart preload. The left-ventricular end-diastolic (LVED) pressure is used as an indicator of LVED volume. At end-diastole in the absence of mitral valve stenosis, venous obstruction, or premature closure of the mitral valve due to aortic insufficiency, there is no pressure gradient between the left atrium and left ventricle. Therefore, the LVED and LA pressures equalize, and the LA pressure may be used as an indicator of LVED pressure (Falicov & Resnekov, 1970; Fitzpatrick, Hampson, & Burgess, 1972; Little & Braunwald, 1997). In patients without venous obstruction or increased pulmonary vascular resistance (PVR), the pressure gradient between the pulmonary vasculature and left atrium and left ventricle is small (3 mm Hg), and at end-diastole pressures equalize across the left heart chambers and pulmonary vasculature. Therefore, the PAED and PAW pressures provide adequate approximations of LA and LVED pressures (Bouchard, Gault, & Ross, 1971; Falicov & Resnekov, 1970; Fitzpatrick et al., 1972; Forsberg, 1971; Jenkins, Bradley, & Branthwaite, 1970; Kaltman, Herbert, Conroy, & Kossmann, 1966; Lappas, Lell, Gabel, Civetta, & Lowenstein, 1973; Scheinman, Evans, Weiss, & Rapaport, 1973; Swan, 1975; Swan, Forrester, Diamond, Chatterjee, & Parmley, 1972; Swan & Ganz, 1975; Walston & Kendall, 1973).

Limitations of Pulmonary Artery Wedge Pressure Indices

Clinically, the PAW pressure is the primary indirect indicator of preload. However, when evaluating the implications of an absolute PAW pressure value, two factors, ventricular compliance and juxtacardiac pressure must be considered (Raper & Sibbald,

1986). First, an increased PAW pressure is assumed to indicate an increase in ventricular volume (preload). The key assumption using PAED and PAW pressures as indices of ventricular end-diastolic volume or preload is that there is a linear relationship between pressure and volume. However, this relationship is more appropriately described a curvilinear (Figure 7). As demonstrated by the normal curve (Figure 7), given the curvilinear pressure- volume relationship, compliance is greater at a lower filling volume compared with a higher volume (Leatherman & Marini, 1993). Unfortunately, it is not known for any patient at what volume the change in compliance will occur. In clinical practice, assessment of the P-V relationship is performed by the administration of small volumes of fluid. A large change in pressure for a small change in volume (all other factors being stable) is assumed to indicate a move to the steeper portion of the P-V curve. Identification of this point is important as a further increase in volume may place the patient at high risk for the development of pulmonary congestion without a further increase in CO (demonstrated by the flattening of the ventricular function curve at higher filling volumes).

A change in pressure may reflect a change in volume (Point A to Point B) or it may reflect a change in ventricular compliance in isolation. This change is exemplified by the left-shift of the P-V curve, where for any given end-diastolic volume the measured pressure is increased (Point A to Point C). Examples of conditions that cause an acute decrease in compliance are myocardial ischemia or infarction, while chronic conditions

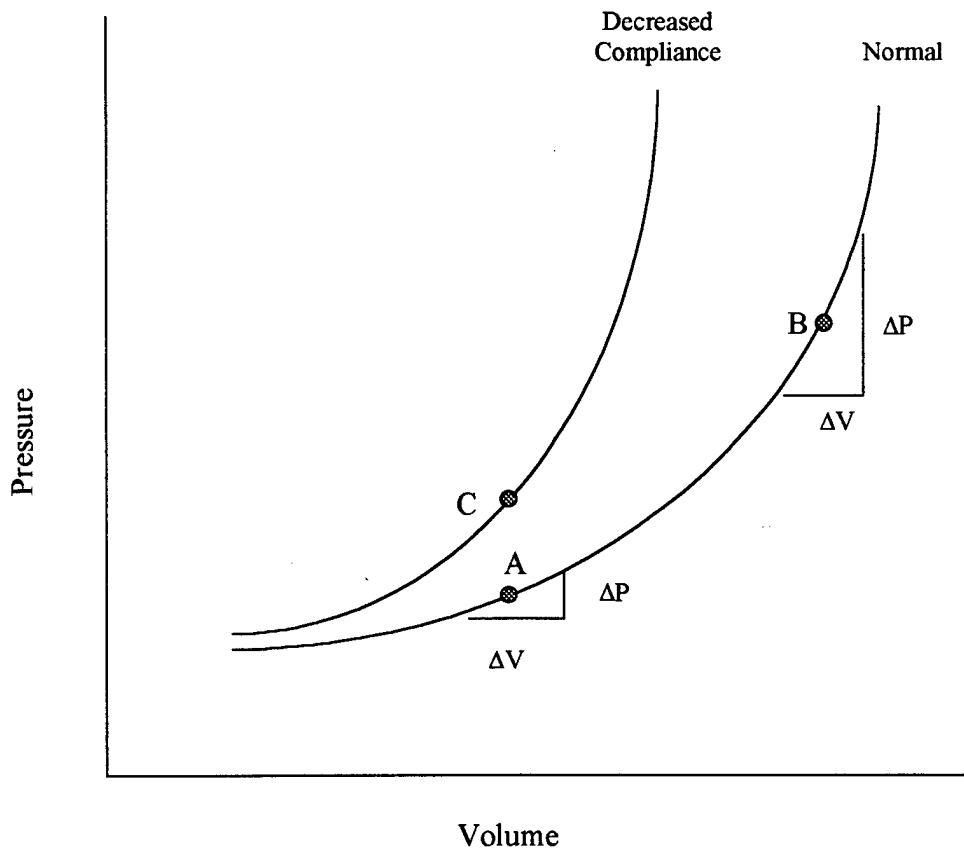


FIGURE 7. Pressure volume curves. The result of the curvilinear pressure-volume relationship is that compliance is greater at a lower filling volume (Point A) compared with a higher volume (Point B). In addition, a decrease in compliance without a change in filling volume is manifested by a left shift of the curve. Thus for any given filling volume the measured pressure is increased (Point A to Point C). Therefore a change in the measured pressure may indicate an increase in filling volume or an alteration in compliance (Leatherman & Marini, 1993).

such as ventricular hypertrophy also cause similar findings. In clinical practice there is no ideal method for determination of an acute change in compliance; thus, evaluation of the patient based on clinical assessment and history is necessary to provide an accurate interpretation of any given PAW pressure value.

The second factor that affects interpretation of the PAW pressure is juxtacardiac pressure. The pressure of clinical interest is the end-diastolic distending pressure (transmural pressure), which is equal to intracavitary pressure minus juxtacardiac or pleural pressure. The use of intracavitary pressure as an indicator of end-diastolic distending pressure is based on the assumption that juxtacardiac pressure is zero. In most cases, at end-expiration this assumption is correct. However, in cases of increased intrathoracic pressure (positive-end expiratory pressure (PEEP), increased intraabdominal pressure, or active expiration) or pericardial tamponade the true distending pressure may be decreased (Cheatham, Safcsak, Zoma, Block, & Nelson, 1998; Schuster & Seeman, 1983). Thus, the measured intracavitary pressure underestimates preload (Janicki et al., 1996; Lange et al., 1988).

In addition, Lange (1988) demonstrated that in the 90-degree lateral position there was an increase in intracavitary pressure without a change in end-diastolic volume. The most probable explanation for this finding is a position-induced increase in transmural compression. This finding may, in part, explain the findings related to the effect of lateral position on PA pressures.

Pulmonary Artery Wedge Pressure as an Indicator of Pulmonary Capillary Pressure

The PAW pressure is also used as an indirect indicator of pulmonary capillary pressure (P_{cap}). The P_{cap} is a major determinant of fluid flux across the pulmonary capillary endothelium into the pulmonary interstitium (Fang, Krahmer, Rypins, & Law, 1996; Guyton & Lindsey, 1959; Starling, 1896). The Starling equation (Starling, 1896) reflects the interaction between hydrostatic and interstitial pressures:

$$Q = k(P_{cap} - P_{int}) - \sigma(\pi_{cap} - \pi_{int})$$

Q = flow rate

k = filtration coefficient of plasma

P_{cap} = pressure within the pulmonary capillaries at the site of filtration

P_{int} = pulmonary interstitial pressures

σ = reflection coefficient of the capillary endothelium to molecules within the plasma

π_{cap} = oncotic pressure in the capillary

π_{int} = oncotic pressure in the interstitial space

In a patient with an intact pulmonary vascular bed and no increase in pulmonary venous resistance, the PAW pressure is normally 1-2 mm Hg less than P_{cap} , and may be followed as an indicator of the risk for development of pulmonary edema (Cope, Allison, Dumond, & Taylor, 1988; Cope, Allison, Parmentier, Miller, & Taylor, 1986; Leatherman & Marini, 1993). For example, in post-myocardial infarction patients the onset of pulmonary congestion (suggestive of fluid filtration out of the pulmonary capillaries) occurs at a PAW pressure of 18 mm Hg (Forrester, Diamond, McHugh, &

Swan, 1971; Forrester, Diamond, & Swan, 1977; McHugh, Forrester, Adler, Zion, & Swan, 1972). However, an alteration in pulmonary vascular permeability, such as occurs in some patients after cardiopulmonary bypass (Sladen & Berkowitz, 1993), results in an increased rate of fluid flux out of the pulmonary vascular bed at a lower intravascular (PAW) pressure. Therefore, initiation of therapeutic activities based on absolute hemodynamic values (e.g., diuretic therapy whenever PAW = 18 mm Hg) should be avoided.

In addition, if there is an increase in resistance in the post-capillary vessels proximal to the junction of the static and flow channels (J-point, Figure 6), the PAW pressure will underestimate P_{cap} when flow is resumed (balloon deflated) [Cope, 1987 #704](Cope et al., 1986; Leatherman & Marini, 1993). This condition has been demonstrated status post-mitral valve replacement, but has not been found to occur post-coronary artery bypass graft (CABG) surgery or aortic valve replacement (AVR) (Cope et al., 1988) (Cope et al., 1986). If there is an obstruction distal to junction of the static and flow channels (Figure 6), for example mitral stenosis, the PAW pressure is an accurate estimate of the P_{cap} , but overestimates the LV pressure (Leatherman & Marini, 1993).

Indices of Afterload

Aortic pressure during systole is the most useful clinical measure of LV afterload. In addition, SVR, mean ABP, and CO are factors that affect aortic pressure and thus afterload. The PA systolic is an indicator of right ventricular (RV) afterload, along with PVR and CO.

In analyzing the effects of each of these factors on afterload, it is useful to define afterload as it applies to the level of the muscle fiber. Use of this definition of afterload allows for a more clear definition and analysis of the factors that affect and alter afterload. For example, an increase in SVR is associated with an increase in mean ABP, the pressure about which the arterial systolic and diastolic pressures fluctuate. An increase in mean ABP results in increased aortic pressure during systole, with a resultant increase in intraventricular pressure, an increase in ventricular wall tension, and finally increased myocardial fiber tension (increased afterload). On the next contraction (as defined by the force-velocity relationship), the increase in afterload is associated with decreased length of shortening and thus a decrease in SV. The subsequent increase in ventricular volume results in increased ventricular wall tension as described by the Law of Laplace (tension = pressure x radius/thickness), and afterload is further increased. This example demonstrates that SVR is not afterload, but merely one factor that affects afterload.

Indices of Contractility

There are no optimal clinical measures of contractility. In clinical practice, indices of systolic function such as ejection fraction, LV and RV stroke work index (SWI) are used. These indices do not reflect the pure definition of contractility, as preload and afterload influence them. For example, ejection fraction, which is defined as end-systolic volume divided by end-diastolic volume ($EF = ESV/EDV$) is sensitive to afterload. In a case characterized by an increase in afterload the SV may acutely decrease with a resultant

decrease in EF. However, in this case contractility may be normal, even though EF is decreased. An awareness of the sensitivity of these derived indices to preload and afterload is crucial in the implementation of therapeutic activities to optimize cardiac function (e.g., vasodilator versus inotropic therapy). These indices of contractility can be used to characterize contractility as long as these limitations are kept in mind. For example, in conditions of unchanging preload and afterload, an increase in SV or CO is assumed to indicate an increase in contractility.

Lateral Position

Clinical Importance of Lateral Positioning

In critically ill patients, therapeutic positioning is undertaken to minimize the negative effects of prolonged bedrest, such as decubitus ulcer formation; and to promote optimal pulmonary function (Fontaine & McQuillan, 1989; Goodrich & March, 1992; Maklebust, 1987; Ross & Dean, 1989). Research has demonstrated that the 30-degree lateral position is adequate to relieve pressure on sacral pressure points without an increase in pressure on the trochanter; while more severe degrees of lateral position (90-degrees) are associated with increased pressure on the trochanter (Colin, Abraham, Preault, Bregeon, & Saumet, 1996; Seiler, Allen, & Stahelin, 1986).

Placement into a supine position is associated with a marked decrease (1100 ml) in functional residual capacity (FRC), which in the face of an unchanging closing volume, may result in partial or complete airway closure with resultant atelectasis (Blair & Hickam, 1955; Harper & Lyles, 1988). The physiologic effect of a closing volume that is

greater than FRC is an increase in ventilation-perfusion mismatching and a subsequent decrease in the partial pressure of arterial oxygen (PaO_2) (Tyler, 1984). The position-induced increase in risk of atelectasis related to decreased FRC typically occurs in supine subjects over the age of 44 years (LeBlanc, Ruff, & Milic-Emili, 1970). Placement in the lateral position has been shown to increase FRC by 400 ml over the supine position, with an improvement in oxygenation in patients with unilateral lung disease (Blair & Hickam, 1955; Zack, Pontoppidan, & Kazemia, 1974).

In patients with unilateral pulmonary atelectasis, positioning the patient such that the unaffected lung is in the dependent position may improve oxygenation through optimization of ventilation-perfusion matching (Dhainaut, Bons, C, & Monsallier, 1980; Gillespie & Rehder, 1987; Hess, Agarwal, & Myers, 1992; Remolina, Kahn, Santiago, & Edelman, 1981; Seaton, Lapp, & Morgan, 1979; Sonnenblick, Melzer, & Rosin, 1983). In unilateral pulmonary interstitial emphysema or lung abscess the patient is positioned with the affected lung down in order to minimize the effects of the disease process (Demers, 1987). Finally, in patients with central airway obstruction due to tumor compression, placement of the affected lung in the dependent position improves oxygenation (Chang, Chang, Shiao, & Perng, 1993). Therefore, placement of a patient into a lateral position is often both prophylactic and an essential part of therapy for certain pulmonary diseases.

Patients undergoing cardiothoracic procedures are at high risk for postoperative pulmonary complications. Of these patients, 20% to 74% develop multicausal left lower

lobe atelectasis (Banasik, Bruya, Steadman, & Demand, 1987; Chulay, Brown, & Summer, 1982; Gavigan, Kline-O'Sullivan, & Klumpp-Lybrand, 1990) (Sladen & Berkowitz, 1993; Wilcox et al., 1988). Intraoperative variables associated with more severe atelectasis include an increased number of grafts, longer operative and bypass time, entering the pleural space, and a lower body temperature (Wilcox et al., 1988). General anesthesia and paralysis have been shown to cause a preferential distribution of gas flow to the non-dependent regions of the lung and to decrease FRC by 20% as a result of the cephalad movement of the diaphragm (Froese & Bryan, 1974; Matthay & Wiener-Kronish, 1989; Sladen & Berkowitz, 1993). In addition, the increased risk of atelectasis in older patients (LeBlanc et al., 1970) is of particular concern given that 98% of CABG procedures are performed on individuals older than 45 years of age (Graves, 1994a; Graves, 1994b).

Turning every two hours to a 45-degree angle has been shown to significantly ($p < .05$) decrease the incidence (Gavigan et al., 1990) and length (Chulay et al., 1982) of postoperative fever. This finding is of importance because every 1° C increase in temperature is associated in an approximate 10 % to 13% increase in resting oxygen consumption (Dubois, 1936; Manthous et al., 1995), which places undue stress on the critically ill patient. Animal studies have demonstrated a decreased incidence of pulmonary complications and morbidity with 30-, 60- and 90-minute turning protocols (Ray et al., 1974; Wahrenbrock, Carrico, Schroeder, & Trummer, 1970). In the two human studies cited there were no significant differences in atelectasis or pleural

effusions in the CABG patients who were or were not turned every two hours (Chulay et al., 1982; Gavigan et al., 1990). However, in these two studies, patients who were turned had a shorter length of intensive care unit (ICU) stay compared with controls (39.7 ± 14.2 versus 58.3 ± 27.7 hours, $p < .025$), and a shorter, but nonsignificant ($.10 > p > .05$) decrease in length of endotracheal intubation (14.4 ± 5.4 versus 19.2 ± 8.7 hours)(Chulay et al., 1982). Gavigan and colleagues did not replicate these latter results.

In CABG patients receiving mechanical ventilation, with an average tidal volume ranging from 600 to 1200 ml, the right lateral and supine positions were found to be associated with higher arterial oxygen partial pressure (PaO_2) and saturation (SaO_2) than the left lateral position (Banasik et al., 1987). These findings are consistent with work related to the effect of unilateral lung disease or pleural effusion on PaO_2 or SaO_2 . In these studies positioning with the "good" lung down was associated with improved gas-exchange (Gillespie & Rehder, 1987) and oxygenation (Chang, Shiao, & Rerng, 1989; Dhainaut et al., 1980; Remolina et al., 1981; Seaton et al., 1979; Sonnenblick et al., 1983). However, these results were not replicated in CABG patients receiving higher tidal volumes (15 ml/kg) (Chan & Jensen, 1992), which may reflect increased alveolar recruitment in the dependent lung.

The process of turning, while associated with a transient change in systemic oxygenation variables, has not been found to be associated with significant ($p > .05$) changes in PaO_2 , SaO_2 , or mixed venous oxygen saturation ($\text{S}\bar{\text{v}}\text{O}_2$) at 5 or 10 minutes (Banasik et al., 1987; Shively, 1988; Tidwell, Ryan, Osguthorpe, Paull, & Smith, 1990;

Winslow, Clark, White, & Tyler, 1990). Therefore, positioning is a well-tolerated, safe, and beneficial therapeutic activity for postoperative CABG patients.

Finally, in terms of patient comfort, in a pilot study of the effect of lateral position on PA pressures, Wild (1983) initially placed the critically ill patients in a 45-degree lateral position. All three patients complained of discomfort associated with the position. However, after revision of the degree of turn to the 30-degree position, there were no further reports of position-related discomfort.

Although there is adequate literature to support the need for repositioning, in many cases critically ill patients are left in the supine position. One reason is the need for frequent recording of hemodynamic measurements. The current standard of practice for performing PA pressure monitoring requires that the patient be placed in the flat, supine position with backrest elevation between 0- and 60-degrees (Bridges & Woods, 1993; Gardner & Bridges, 1995). Patients who require frequent measurements are either left in the supine position or are frequently disturbed due to the need to return them to the supine position. Therefore, research was needed to determine if PA pressures obtained in the lateral position were similar to those obtained in the supine position. If the PA pressures were similar, the patient could enjoy the benefits of the lateral position without being disturbed for pressure measurements.

Effect of Lateral Position on Hemodynamic Indices

There is a growing body of literature regarding the effect of lateral position on CO, HR, ABP, and indices of systemic oxygenation in critically ill patients. However, there is

limited data regarding the effect of lateral position on the factors (preload, afterload, contractility) that affect SV, or on the indices (PAS, PAED, PAW pressures) of these factors. Prior to this study, it was unknown if PA pressures measured in lateral positions, other than the 90-degree lateral position (Bryant & Kennedy, 1982), were comparable to those obtained in the supine, flat position. The key factor limiting this research was the identification of angle-specific LA reference points. A study conducted by Doering and Dracup (1988) in post-cardiac surgery patients placed in the 45-degree lateral position highlights how this lack of knowledge of the effect of lateral position on the factors that affect SV impairs our ability to identify possible mechanisms for an observed response to a position. Results of the study indicated that HR was not significantly ($p > .05$) affected by position; however, there was a wide degree of variability in SV (1.5 to 35 ml, mean 6.6 ml) and CO (variation greater than 10% in 45% of subjects). While the authors were able to state that there were changes in SV, they were unable to hypothesize about possible mechanisms. If it were known that hemodynamic indices obtained in a lateral position were comparable to those obtained in the supine position, possible mechanisms for the observed changes, for example an increase in preload, could be postulated.

Reference Level for PA Pressure Measurement

There are numerous technical factors that affect the accuracy and reliability of PA pressure measurements. Of particular importance to this research is the accurate referencing of the PA catheter system to the left atrium. Referencing of the PA catheter system is accomplished by placing the air-fluid interface of the system at a specified

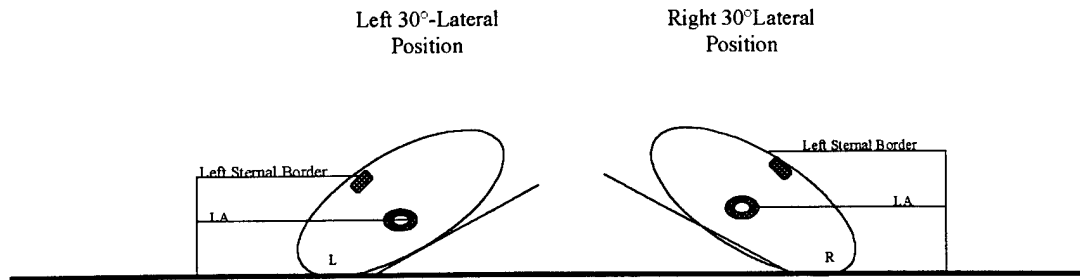
reference point to negate the weight effect of the fluid in the catheter tubing. The standard reference level for PA pressure measurements is the left atrium. Failure to correctly reference the pressure system results in an inaccurate estimate of LA pressure, as a result of the inclusion of hydrostatic pressure. For example, referencing the catheter system at a point 1-cm below the horizontal plane of the physiologic reference (the left atrium) will result in a 0.73 mm Hg increase in measured pressure. Conversely, a catheter system referenced 1 cm above the physiologic reference point will result in a 0.73 mm Hg decrease in measured pressure (Nave & Nave, 1985). The effect of inclusion of the hydrostatic pressure is of great clinical importance considering a normal PAW pressure is only 6 to 12 mm Hg.

With lateral rotation of the body, the horizontal plane that passes through the left atrium shifts due to the displacement of the intrathoracic structures relative to external anatomic references. Using chest radiography, the mean displacement of the left atrium relative to its supine position was studied in 10 patients with pleural effusions. The mean displacement was 3.4 ± 1.6 mm in the right lateral decubitus position, and 2.8 ± 1.6 mm in the left lateral decubitus position (Kennedy et al., 1984). Based on this study, the fourth intercostal space at the midsternum was specified as the horizontal level for the left atrium in both right and left lateral decubitus positions. Using a computerized axial tomography (CAT) scan, Paoletta and colleagues (1984) confirmed these results, but recommended a slightly modified reference point for the right (fourth intercostal space, midsternum) and left (left parasternal border) lateral decubitus positions. The difference

between Kennedy's and Paoletta's reference points is clinically insignificant. While identification of a reference point in the lateral decubitus position is important, it is of limited clinical utility. Patients are more often positioned in lesser degrees of lateral rotation than 90-degrees; therefore, identification of reference points for lesser degrees of turn was needed.

Using echocardiography and a trigonometric model based on the work of Kee and colleagues (1993), VanEtta and colleagues (1993) identified the location of the left atrium in the right and left 30-degree lateral positions. The level of the left atrium was approximately one-half the vertical distance between the surface of the bed and the left sternal border in both the 30-degree left and right lateral positions (Figure 8). The mean distance of the left atrium above the bed to the left sternal border was $47.7\% \pm 7.23\%$ of in the 30-degree left and $53.05\% \pm 9.79\%$ in the 30-degree right lateral positions.

The clinical utility of this estimated reference point was validated by determining the approximate error in pressure measurements associated with its use, relative to the actual LA position (as defined by echocardiography). The approximate error in pressure measurements was -0.58 mm Hg (SEM 0.33) and 0.78 mm Hg (SEM 0.22) in the 30-degree right and left lateral positions, respectively. These potential errors are clinically insignificant. Therefore, this clinically validated reference level was used as the reference level for the left atrium in this study.



PA = Phlebostatic Axis; LA = left atrium

FIGURE 8. Left atrial reference point in 30-degree lateral position. The reference point is located one-half the vertical distance between the bed surface and the left sternal border in both right and left 30-degree lateral positions. From DJ VanEtta (1992). Location of the left atrium in thirty-degree right- and thirty-degree left lateral recumbency in adults. Unpublished master's thesis. University of Washington, Seattle, Seattle Washington. Adapted with permission

Effect of Lateral Position on Pulmonary Artery and Pulmonary Artery Wedge Pressures

Nine studies (Appendix A) have been conducted to evaluate the effect of a 20- (Whitman, 1982), 30- (Cason et al., 1990; Duke, 1994; Ross & Jones, 1995; Wild, 1983), 45- (Briones et al., 1991; Groom et al., 1990; Keating et al., 1986) or 60-degree (Aitken, 1995) lateral position with various degrees of backrest elevation on PA pressures. In studies by Whitman (1982) and Ross and Jones (1995) the changes in PA pressures in the 20-degree lateral position were clinically insignificant. In all of the studies with lateral position of 30-degrees or greater, there were clinically and statistically significant differences ($p < .05$) in PA and PAW pressures relative to supine, flat measurements. In many cases, the mean differences were not clinically significant, but there were large individual variations. In five of the six studies that used the midsternum as the reference level, there were clinically and statistically significant mean differences between supine and lateral measurements. Pressure differences ranged from -8.8 mm Hg to 6.1 mm Hg in the right and 4.9 to 9.2 mm Hg in the left lateral positions.

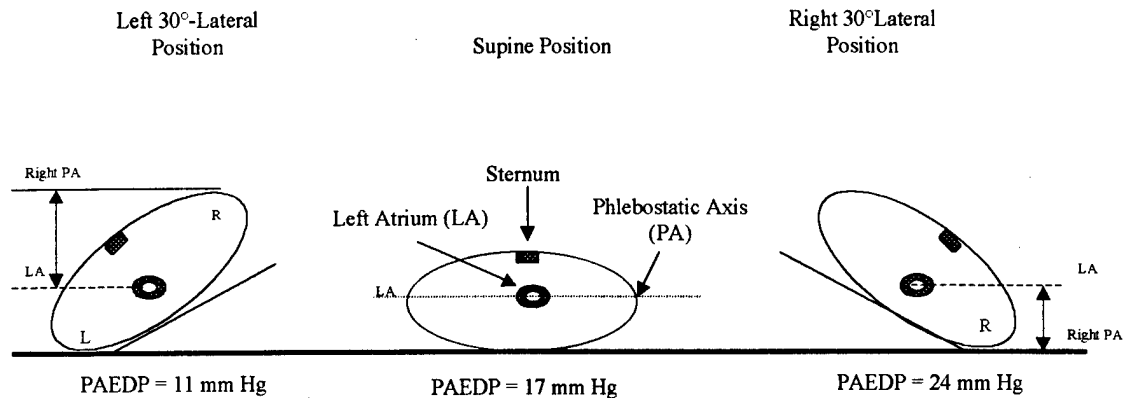
Only one study has been conducted using the reference level specified by VanEtta (1993). Duke (1994) studied the effect of the 30-degree lateral position on PA pressure measurements in six critically ill patients. While the mean pressure differences in the supine versus 30-degree right and left lateral positions were small, there was a large amount of variance. For example, the mean PAW pressure differences between the supine and 30-degree right positions was 0.83 mm Hg (SD 3.06); while the mean difference in the left lateral position was 1.16 mm Hg (SD 2.04). However, there was a

large amount of variation in the pressures over the period of the study. For example, in one subject the PAED pressure decreased 14 mm Hg over the period of the study. It is important to note that the large amount of variance in the different pressures was reflective, in all cases, of the effect of one subject. These results highlighted the need to repeat this study with a larger number of subjects.

Evaluation of the results of the nine studies cited is complicated by methodological limitations such as the use of digital data, non-standardized degree of position, measurement of pressures over an entire respiratory cycle, lack of interrater reliability, non-standardized period between pressure measurements, and varying levels of backrest within a study. However, the most important limitation of all the studies, with the exception of Duke (1994), was the use of variable reference points that introduce pressure measurement error due to the inclusion of hydrostatic pressure. The inclusion of a hydrostatic pressure component could explain, in part, the statistically and clinically significant results of these studies. For example, using data from VanEtta's study, the mean difference between the validated reference point for the left atrium and the left sternal border was 10 cm. Referencing the catheter system at the left sternal border would introduce a 7.36 mm Hg pressure difference. It is important to note that the current standard for defining a clinically significant change in PA pressures is 4 mm Hg (Nemens & Woods, 1982); thus the introduction of systematic measurement error related to inaccurate referencing could lead to erroneous results.

Further support for the assumption that the results of the above studies reflect the introduction of systematic error resulting in erroneous measurements, and thus the need to reevaluate the effect of 30-degree lateral position on PA pressures using a validated LA reference level, is provided by research using various reference points within one study (Ross & Jones, 1995) (Figure 9). In a patient placed in the 30-degree right lateral position, with the right phlebostatic axis as the reference point, the PAED pressure was significantly ($p < .05$) higher than supine values (17 mm Hg versus 24 mm Hg). In this case, the level of the right phlebostatic axis was below the level of the left atrium, with a hydrostatically-induced increase in measured pressure. Conversely, when the patient was placed in the 30-degree left lateral position, the reference point was above the left atrium, and the opposite effect on pressure was noted (17 mm Hg versus 11 mm Hg). These data highlight the effect of using an inaccurate reference level on measured pressures.

Finally, additional support for the assumption that lateral position has minimal effect on PA pressures, as long as a reference point consistent with the level of the left atrium is used, is provided by four studies of the effect of the 90-degree lateral position on PA pressures. In the three studies using a fluid-filled PA catheter, there were no clinically or statistically significant ($p > .05$) changes in PA or PAW pressures in 43 of the 45 subjects studied (Guenther et al., 1987; Kennedy et al., 1984; Murphy, 1977). However, Lange (1988), using a micromanometer-tipped catheter placed in the left ventricle detected a 4 to 7 mm Hg pressure difference between LVED pressure in the left lateral versus supine and right lateral position. This increase in pressure was not associated with a significant



PA = Phlebostatic Axis; LA = left atrium

FIGURE 9. Effect of use of non-angle specific reference points on measured PA pressures. Demonstration of the effect of variable reference points on the recorded PA pressures. In the supine position, using the phlebostatic axis as the reference for the left atrium, the measured pressure was 17 mm Hg. In the 30-degree left lateral position, if the right phlebostatic axis is used as the reference, the measured pressure is 11 mm Hg, which is decreased due to erroneous referencing above the left atrium. In the 30-degree right lateral position when the right phlebostatic axis is used as the reference, the measured pressure is 24 mm Hg. This increase in pressure is due to the inclusion of hydrostatic pressure secondary to referencing below the level of the left atrium. Based on data from Ross and Jones (1995).

($p > .05$) change in end-diastolic volume ($62 \pm 15 \text{ ml/m}^2$ versus $66 \pm 13 \text{ ml/m}^2$ in the left and supine positions, respectively), which is most likely indicative of position-induced transmural compression. However, conclusions from these latter results were limited by the small sample size ($N = 7$).

Summary

The determination of the effect of lateral position on hemodynamic indices has been hampered by the lack of angle-specific reference points. Results of previous research related to the effect of lateral position on hemodynamic indices may be erroneous due to the inclusion of hydrostatic pressure related to inaccurate referencing. Research has demonstrated that there are no statistically ($p > .05$) or clinically significant changes in PA or PAW pressures in the 90-degree lateral position, as long as an angle-specific reference level is used. It was anticipated that PA and PAW pressure measurements obtained in a less-extreme degree of position, using an angle-specific reference level, would yield similar results. If the PA pressures were similar in the supine and 30-degree lateral positions, the patient could enjoy the benefits of the lateral position without being frequently disturbed for pressure monitoring.

Purpose

The purpose of this study was to determine the effect of right and left 30-degree lateral positions on PA and PAW pressures in critically ill, adult postoperative cardiac surgery patients.

CHAPTER III

METHODS OF PROCEDURE

This chapter presents the design of the study, sample, data collection instruments and procedures, and information related to the use of human subjects. The chapter concludes with a description of the data analysis procedures.

Design

An experimental repeated-measures design was used to determine the effect of right and left 30-degree lateral positions on PA and PAW pressures in critically ill, postoperative cardiac surgery patients. Following determination of baseline PA and PAW pressure fluctuation, the patients who served as their own control, were randomly assigned to a sequence of position changes.

Sample

A convenience sample of 42 men and women, age 18 years and older, who were undergoing a cardiac surgical procedure and required PA pressure monitoring, were recruited from the Cardiothoracic Surgery Service at a medical center in Seattle. The study took place on the Cardiothoracic Critical Care Unit at the Medical Center.

The sample size was based on an a priori power analysis of data from Duke's (1994) study, which demonstrated a moderate effect size of 0.5. A sample of 40 provided a power of 0.88 ($p = .05$) for a 1 mm Hg pressure difference, and a power of 1.0 for a pressure difference of 1.5 mm Hg. The rationale for selection of a very high level of statistical power (0.95 or higher) was that low statistical power increases the probability

of concluding that there were no significant differences between pressure measurements in the supine versus lateral positions, when a difference actually existed. With regard to this study, a finding of no difference between PA pressure measurements in the supine and 30-degree lateral position would lead to a recommendation that patients could have their PA and PAW pressures measured in the 30-degree lateral position and that the patients do not need to be repositioned for pressure measurements. However, if a power of 0.80 were selected, there would be a 20% chance of reporting no difference in pressures, when a difference existed. Thus, the recommendation for a change in clinical practice would be erroneous. Because these hemodynamic indices are used to guide therapy in critically ill patients, an erroneous recommendation has very serious implications.

All adult patients undergoing a cardiac surgical procedure were eligible for the study. Inclusion criteria included: (1) placement of a PA catheter, (2) ability to read and speak English; (3) hemodynamic stability, which was defined as a systolic ABP greater than 90 mm Hg, baseline HR less than 130 beats per minute, a mixed-venous oxygen saturation ($\text{S}\bar{\text{v}}\text{O}_2$) greater than 60% or arterial oxygen saturation (SaO_2) greater than 90% over the previous hour; (4) chest tube drainage of less than 100 ml/hr over the previous three hours; (5) more than four hours postoperative; (6) a PA temperature greater than 36.0°C ; and (7) no fluid boluses exceeding 250 ml/hr, alterations in inotropic or vasoactive infusions, or intravenous diuretics or morphine within the hour before the study or during

the study. Patients requiring postoperative cardiac pacing were included, unless clinically unstable.

Exclusion criteria included: (1) inability to tolerate a 0-degree flat backrest position (increased intracranial pressure, pulmonary edema, gastroesophageal reflux); (2) clinical instability, which was defined as systolic ABP less than 90 mm Hg, SaO_2 less than 90% or an SvO_2 less than 60%, or baseline HR greater than 130 beats per minute; (4) mitral stenosis or LA myxoma; (5) receiving hemodialysis, (6) requiring an intra-aortic balloon pump, (7) chest wall deformity, for example, kyphoscoliosis, and (8) per the request of the patient or health-care provider.

Data Collection Instruments

The data collection forms included the "Data Collection Sheet" (Appendix B), and analog recordings of the PA and PA wedge pressures for each position. An example of the analog data is located in Appendix C. The data collection sheet included demographic and clinical data including age, gender, height, weight, body surface area, race, medical history and diagnosis, surgical information, days of bedrest prior to surgery, midsleep (based on usual time of sleep and awakening), vital signs and baseline hemodynamic data, ventilator settings and oxygen administration, intravenous fluids, medications, position of the PA catheter as determined from the postoperative chest radiograph, and technical characteristics of the pressure monitoring system. In addition, outcome data (i.e., PA and PAW pressures) associated with baseline fluctuation and each

position, and the distance the LA reference level was from the surface of the bed in each position were recorded.

Data on the following individual characteristics that have been shown to affect response to position and position change were collected to analyze for potential confounding or effect modification: age greater than 70 years, PA temperature ($< 36.5^{\circ}\text{C}$), time since surgery, type of surgery, autonomic dysfunction, medications (beta-adrenergic blockers, calcium channel blockers, nitrates, diuretics, (e.g., furosemide), and analgesics, (e.g., morphine sulfate), and length of bedrest before surgery (Dikshit et al., 1973; Doering & Dracup, 1988; Harper & Lyles, 1988; Vismara, Mason, & Amsterdam, 1974; Ward, McGrath, & Weil, 1972; Zelis, Mansour, Capone, & Mason, 1974). In addition, time of day may be associated with variability in cardiovascular reactivity (Saito et al., 1993). Time of day of the experimental procedure was analyzed relative to usual, pre-hospital midsleep.

Pulmonary Artery and PAW Pressure Measurement

Technical Information

Pulmonary artery systolic, end-diastolic, mean, and wedge pressures were measured with various PA catheters (Table 1) attached to a Spacelabs pressure monitoring system and monitor (Model 90303 Bedside and Model 90402 Dual Blood Pressure Module). The pressure module is accurate to 1% of range (-50 to +300 mm Hg), with a zero drift of $< 0.1 \text{ mm Hg}/^{\circ}\text{C}$. The monitors are fixed calibration pressure monitoring systems, which require routine calibration every six months (Gardner, 1996; Spacelabs Product Manual).

The natural frequency (Fn) and damping characteristics of the catheters used in the study are outlined in Table 1. Despite manufacturer reports of a high Fn, in vivo analysis of an Edwards catheter (93A-831-7.5-Fr) with a reported Fn of 17 Hz demonstrated a maximum Fn of 11.9 ± 2.5 Hz, an amplitude ratio of 1.93 ± 0.33 , and a damping coefficient of 0.28 ± 0.66 (Rutten, Nancarrow, Ilsley, & Runciman, 1987). In addition, the in vivo dynamic response characteristics of the pressure monitoring systems (transducer, pressure tubing, catheter) are generally less than that reported by manufacturers (Gore, Middleton, & Bridges, 1995). Given the discrepancy between the dynamic response characteristics reported by the manufacturer and in vivo performance, the dynamic response characteristics of each catheter system was assessed using the technique outlined by Gardner (1981) and Bridges and Middleton (1997) (Appendix D).

The disposable transducers that were used in this study were fixed calibration transducers that did not require additional calibration at the bedside (Gardner, 1996). Specifications for the Truwave™ disposable pressure transducer included a zero drift of ± 1 mm Hg per 8 hours in the pressure range of -50 to $+300$ mm Hg, a sensitivity thermal drift of $\leq \pm 0.1\%$ per centigrade, and a natural frequency of 40 Hz nominal for a standard kit (48"/12") and > 200 Hz for the transducer alone (Baxter, Product Insert). Analog recordings were obtained from a Spacelabs 90323 PC Two-Channel System printer, with paper speed at 25 mm/sec and a bandwidth of DC to 40 Hz.

TABLE 1. Specifications of Baxter-Edwards Pulmonary Artery Catheters

Catheter Name	Brand Number	Length (cm)	Size (Fr)	Lumen	Fn (Hz)	Amplitude Ratio
Swan-Ganz VIP™ (Baxter)	831HVF75 831VF75	110	7.5	Distal Injectate VIP	34 43 47	2.6:1 3.1:1 3.1:1
Intellicath CCO/VIP™ (Baxter)	139-7.5F 139H-7.5F	110	7.5	Distal Injectate Infusion	25 33 41	2.0:1 2.5:1 2.9:1
CCO/SVO ₂ ™ (Baxter)	744H7.5F 744F75	110	7.5	Distal Injectate	25 45	2.1:1 2.7:1

PA Waveform Interpretation

All PA waveforms were measured at end-expiration (Berryhill, Benumof, & LA, 1978; Wild & Woods, 1985), from an analog recording (Cengiz, Crapo, & Gardner, 1983; Dobbin, Wallace, Ahlberg, & Chulay, 1992; Ellis, 1985; Johnson & Schumann, 1995; Levine, 1985; Lundstedt, 1997; Maran, 1980; Silverman, Eppler, Pitman, & Patz, 1984), and relative to a simultaneous electrocardiogram (ECG) tracing (Gardner & Bridges, 1995).

The PA systolic pressure, which is represented by a steep rise during RV ejection and usually occurs after the QRS or near the T wave of the ECG, was measured at the peak of the PA waveform (Figure 10A). The PAED pressure was measured 0.08 second after the onset of the QRS complex (Lipp-Ziff & Kawanishi, 1991), and the PA mean was determined by bisecting the end-expiratory PA waveform (Gardner & Bridges, 1995). In the presence of LV dysfunction, the presystolic “a” wave, if present, was used as the indicator of PAED pressure (Falicov & Resnekov, 1970; Rahimtoola, Loeb, & Ehsani, 1972). The PAW pressure is similar to the LA pressure, except that it is slightly damped and phase delayed (50-70 milliseconds) (Lange, Moore, & Cigarroa, 1989). Two waveforms were identified in the PAW pressure tracing: the “a” wave, which is located after the P-R interval on the ECG, and the “v” wave, the second positive deflection located near the T-P interval. The PAW pressure is a mean pressure and was determined by bisecting the “a” wave and “v” wave, so there was an equal area above and below the bisection (Figure 10B).

B. Pulmonary artery wedge pressure. Two primary waveforms (“a” and “v”) were identified. The “a” wave is located after the “P” wave of the electrocardiogram (ECG). The “v” wave occurs during the T-P segment of the ECG. The PAW pressure is a mean pressure, and is measured by bisecting the waveform so there is an equal area above and below the bisection. From Cardiac Nursing, (p. 441), by S.L Woods, E.S. Sivarajan Froelicher, C.J. Halpenny, S. Underhill Motzer, 1995, Philadelphia: J.B. Lippincott. Reprinted with permission.

Lung Zone Determination

Of particular concern in this study was the ability to assess for maintenance of a continuous vascular segment (Zone 3 segment) between the catheter tip and the left atrium. Three methods were used to assess for probable Zone 3 catheter placement.

First, anatomically, approximately 50 % of the lung volume is below the left atrium (Friedman, Peters, C, Brimm, & Meltvedt, 1986; West, Dollery, & Naimark, 1964), and vascular beds below the level of the left atrium have an increased propensity for Zone 3 characteristics (Glenny & Robertson, 1990; Glenny, Lamm, Albert, & Robertson, 1991; Glenny, McKinney, & Robertson, 1997; Glenny, Polissar, & Robertson, 1991; Walther, Domino, Glenny, & Polissar, 1997). In the anteroposterior (AP) chest radiograph, the level of the left atrium is located approximately 3 cm below the level of the carina, and the carina is vertically close to the bifurcation of the PA (Benumof, Saidman, Arkin, & Diamant, 1977). Therefore, the patient's most recent AP chest radiograph was reviewed for correct PA catheter placement (Figure 5) (Hasan, Malanga, Braman, Corrao, & Most, 1984; Kane et al., 1978; Malanga, Hasan, Bramon, Corrao, & Most, 1983; Roy, Powers, Feustel, & Butler, 1977; Shasby et al., 1981; Tooker, Huseby, & Butler, 1978). Second, during wedging of the catheter the PA waveform was analyzed for the following changes:

- (1) The PA waveform should flatten into a characteristic atrial waveform, although in some cases distinct "a" and "v" waves may not be discernable.
- (2) The waveform should return immediately to a PA configuration with deflation of the balloon. A partial wedge is characterized as a waveform different from the phasic PA waveform, but intermediate

between the phasic PA waveform and the atrial waveform. Partial PAW pressures do not accurately reflect left heart pressures, and were not used (Morris & Chapman, 1985; Morris, Chapman, & Gardner, 1985). (3) The PAW pressure should be lower than the mean PA pressure in the absence of a large "V" wave (Leatherman & Marini, 1993). The third method involved the evaluation of the respiratory artifact induced by mechanical ventilation (inspiratory peak value minus expiratory peak value) on the PA and PAW pressure tracings (Teboul et al., 1988). In patient's with Zone 2 placement where there is increased respiratory artifact reflecting changes in alveolar pressure, the change in the ratio of the PAW pressure change relative to the change in PA pressure ($\Delta\text{PAWP}/\Delta\text{PAP}$) is greater than 2 at all levels of PEEP. A value of one for the ratio of $\Delta\text{PAWP}/\Delta\text{PAP}$ indicates no respiratory artifact due to Zone 2 alveolar compression.

Cardiac Output Measurement

Ideally, in order to provide a complete physiologic picture of the changes in the hemodynamic variables, measurement of CO should be completed. However, for purposes of this study, performance of thermodilution CO measurements would require the injection of 20, 5- to 10-ml iced or room temperature dextrose or saline boluses into the right atrium. The injection of the fluid boluses represents a physiologic stressor, and is has some degree of risk. For example, iced injectate has been associated with a transient decrease in HR (Harris, Miller, Beattie, Rosenfeld, & Rogers, 1985). In a study of the effect of technique on the variability of thermodilution CO measurements, 31% of 1,982 injections were classified as unacceptable with a resultant increase in variability:

6.15 ± 0.68 L/min (unacceptable) versus 8.36 ± 0.49 L/min (acceptable) ($p < .005$) (Stites, Barnes, Overman, & O'Boynick, 1998). Of particular importance, the technical errors were not apparent from the routine monitoring of the distal injectate curve. Given that Whitman's (1982) study indicated that the lateral position-induced change in CO was less than 200 ml, the variability introduced by technical errors may negate accurate assessment. In addition, the variability of CO measurements ranged from 6.4% to 9.9% depending on the stability of other cardiopulmonary variables such as HR, RR, and PAM pressure (Sasse, Chen, Berry, Sassoon, & Mahutte, 1994). Therefore, given the risks and the utility of the information that would be provided, thermodilution CO measurement was not appropriate for this study.

New technology, which employs a stochastic system identification technique and a pseudorandom binary sequence of heat pulses is available for continuous CO (CCO) measurement (Gillman, 1992; Siegel, Hennessey, & Pearl, 1996; Yelderman, 1993; Yelderman et al., 1992). Continuous CO is comparable to thermodilution CO (bias $0.02 - 0.31$ L/min, 95% CI = -1.34 to 1.18 L/min) in a wide variety of hemodynamically stable critically ill patients (Auger et al., 1994; Boldt, Menges, Wollburck, Hammerman, & Hempelmann, 1994; Burchell, Yu, Takiguchi, & Myers, 1996; Davis & Sakuma, 1992; Ditmyer, Shively, Burns, & Reichman, 1995; Haller, Zollner, Briegel, & Forst, 1995; Lichtenthal & Wade, 1993; Medin, Brown, Onibene, & Cunnion, 1997; Yelderman et al., 1992). However, a key assumption of CCO measurement is that the CO is stable during the interval analyzed by the cross-correlation algorithm (Siegel et al., 1996). In human

(Haller et al., 1995; Lazor, Stanley, Cass, & Pierce, 1996) and animal models (Decruyenaere, DeDeyne, Hoste, Troisi, & Colardyn, 1997; Ryan, Far, Lee, & Bongard, 1993; Siegel et al., 1996), while CCO accurately detects an acute change in CO (Ryan et al., 1993), the time interval for reporting 90% of an acute change in CO was 12 to 14 minutes. Given the five-minute stabilization period used in this research, this time lag was too long. Therefore, no method currently in use in clinical practice was able meet the requirements of this study.

Procedures

Preoperative Procedures

Subjects were identified through the Cardiothoracic Surgery services. The nurse caring for the patient asked the patient if they would be willing to talk with a research nurse about a study. If the patient agreed, the primary investigator explained the study to the patient and obtained their consent. The consent was obtained either at the preoperative clinic visit or during hospitalization in preparation for surgery.

Study Protocol

The study consisted of two parts, the first related to the description of baseline PA pressure fluctuations. The second portion of the study was related the effect of the 30-degree lateral position on PA and PAW pressures. The study protocol is outlined in Appendix E.

Pulmonary Artery Pressure Fluctuation

Hemodynamic variables fluctuate minute by minute and over a 24-hour period. The clinician must have knowledge of this fluctuation in order to determine if a pressure change represents a clinically significant event or simply an expected fluctuation. Based on a study of pressure fluctuations in 26 ICU patients, the following pressure changes are frequently cited as indicative of clinically significant changes: PAS 5 mm Hg, PAM 4 mm Hg, PAED 4 mm Hg, and PAW 4 mm Hg (Nemens & Woods, 1982). However, in patients with increased PAW pressures (greater than 18 mm Hg), pressure fluctuations generally exceed these accepted clinical standards (Cason et al., 1990; Moser & Woo, 1996; Murphy, 1977). In Nemens and Woods (1982) study of pressure fluctuation, seven measurements were taken over a period of 30 minutes. Data from Nemens' study were reanalyzed, comparing pressure fluctuation over the first 15 minutes with fluctuations over the entire 30-minute period. There were no statistical ($p > .05$) or clinically significant differences (pressure fluctuation difference less than 1 mm Hg) in pressure fluctuations over the first 15-minute period compared with the entire 30-minute period. Therefore, for purposes of this study, pressure fluctuations were measured over a 15-minute period. The fluctuations in PA and PAW pressures over a period of 15 minutes were described for each subject in order to determine if observed changes in PA or PAW pressures in the lateral position were reflective of the effect of position or the expected fluctuation. The sequence of measurements is outlined in Figure 11.

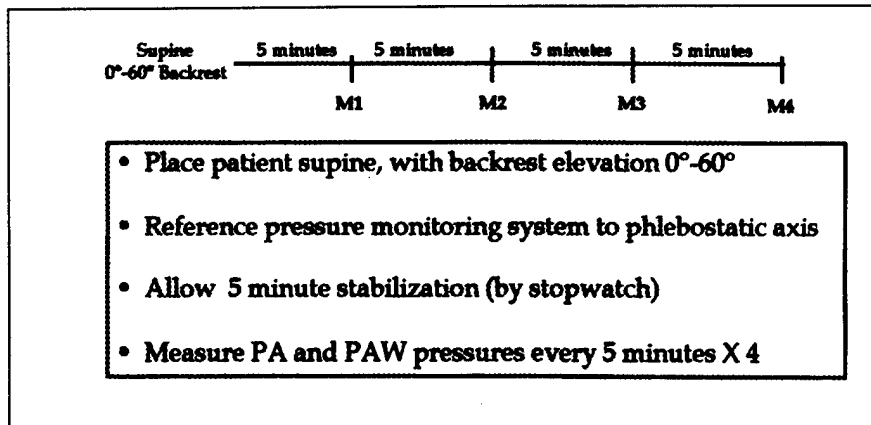


FIGURE 11. Protocol for measurement of PA pressure fluctuations.

Patients were placed in the supine position, with the backrest at whatever degree of elevation the patient was found (0- to 60-degrees). Four patients were found in a lateral position with a backrest elevation ranging from 5- to 22-degrees (14 ± 7 -degrees), while the remain 38 patients were in supine position with backrest elevation ranging from 0- to 47-degrees (24 ± 12 -degrees). The patients found in the lateral position were placed in a supine position, and a five-minute stabilization period was observed before initiation of data collection. The pressure monitoring system was referenced to the phlebostatic axis. The phlebostatic axis was defined as the intersection of an axis that transects the body at the junction of the fourth intercostal space and the sternal margin, and a frontal plane passing midway between the anterior and posterior surfaces of the chest (Winsor & Burch, 1945). Referencing was accomplished by placing the air-fluid interface of the stopcock on the top of the transducer at the level of the phlebostatic axis (Figure 12A and 12B). Exact leveling was ensured with the aid of a carpenter's level.

The focus of this study was on the effect of position on PA and PAW pressures and not the response to position change; therefore, the patients were stabilized for five minutes, as measured by a stopwatch, before each pressure measurement. This stabilization period was based on research that indicated that in patients with a PA wedge pressure of 18 mm Hg or less, cardiovascular stabilization following lateral positioning occurred within in five minutes or less Aitken, 1995; Banasik & Emerson, 1996; Briones et al., 1991; Carroll, 1992; Kennedy et al., 1984; Murphy, 1977; Noll, Duncan, & Fountain, 1991; Pena, 1989; Shinnors & Pease, 1993; Shively, 1988; Tidwell et al., 1990;

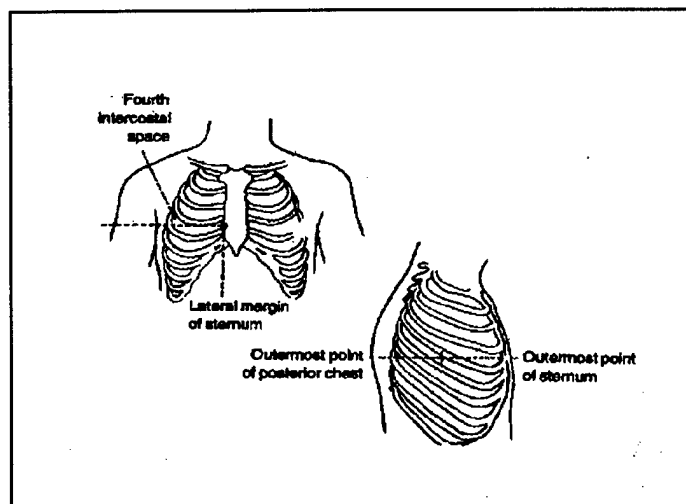


FIGURE 12A. Identification of the phlebostatic axis. See text for details. Shinn, J.A., Woods, S.L., & Huseby, J.S. (1979). Effect of intermittent positive pressure ventilation upon pulmonary capillary wedge pressures in acutely ill patients. *Heart Lung*, 8(2), 324. Reprinted with permission.

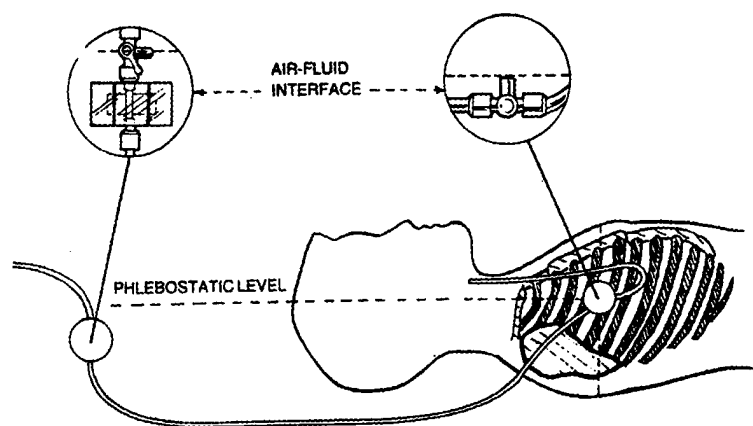


Figure 12B. Schematic demonstrating correct referencing of the pressure monitoring system to the phlebostatic axis. From Bridges, E.J., & Woods, S.L. (1993). Pulmonary artery pressure monitoring: state of the art. *Heart Lung*, 22(2), 101. Reprinted with permission.

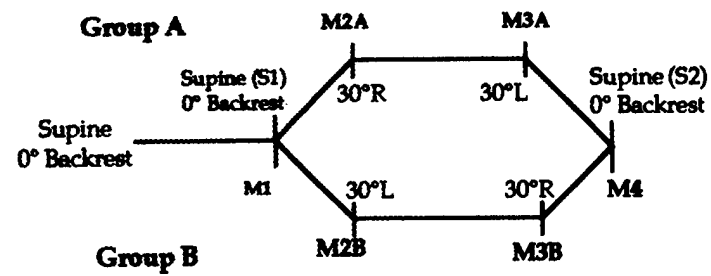
Winslow et al., 1990). Pulmonary artery and PAW pressures were obtained every 5 minutes for 15 minutes, for a total of four sets of measurements.

Pulmonary artery systolic, end-diastolic, and mean pressures were recorded for up to 30 seconds to obtain an analog tracing free from artifact (coughing, movement). To obtain PAW pressures the balloon on the catheter was inflated for a maximum of 15 seconds; thus, the PAW pressures were recorded for 15 seconds.

Position Effect

The patients were randomized into one of two treatment sequence groups (Group A: flat, supine; 30-degree left lateral recumbent, 30-degree right lateral recumbent, supine; Group B: flat, supine, 30-degree right lateral recumbent, 30-degree left lateral recumbent, flat, supine) (Figure 13). Randomization was performed to control for latency (effect of previous position on subsequent measurement) and sequence effect. The first subject was assigned by a coin toss, and subsequent subjects were assigned to a sequence by odd or even number. Following sequence assignment the subject was placed in the supine position with 0-degrees backrest elevation. In all positions, one pillow was placed under the subject's head for comfort. The phlebostatic axis was located and marked with a pen, and the air-fluid interface of the PA catheter system was referenced and zeroed at this point.

When supine measurements were completed, the subject was passively positioned in either the 30-degree right or 30-degree left lateral position (depending on the assigned sequence). Standardization of right and left lateral positions was accomplished by the use



***5 minutes stabilization before each measurement**

FIGURE 13. Protocol for determination of effect of lateral position on PA and PAW pressures.

of a 30-degree hard-foam wedge placed behind the subject's back with the edge at the intersection of the patient's chest wall and the bed surface. In the lateral positions, the LA reference point was identified with a caliper and a laser carpenter's level, and marked on the chest with a pen. The catheter system was referenced and zeroed, and the PA pressures were obtained as previously described. The subject was then turned to the opposite lateral position, and the procedure for identification of the reference level, positioning, and pressure measurements were repeated. Finally, the patient was returned to the supine position and the procedure repeated again, using the previously identified phlebostatic axis as the reference level.

Human Subjects

The University of Washington's Human Subjects Review Committee, and the Nursing Services Investigation Review Board approved study procedures at the University of Washington Medical Center (UWMC) (Appendix F). Written, informed consent was obtained preoperatively from the patient. The study posed no additional risk to the patient as the intervention reflected treatment the subject would already receive (turning and PA pressure measurements). There was a potential for discomfort related to positioning, however, all actions were taken to minimize the discomfort. The study provided no direct benefit to the patient. However, the PA pressure measurements obtained during the study were shared with the nurse caring for the patient; therefore, additional measurements were not required. The patient's name did not appear on any of the data collection forms and all data were kept confidential by the investigators. The

patient was free to withdraw from the study at any time. The study data will be retained indefinitely, and may be used in future related research.

Data Analysis

Baseline Data

Demographic and baseline physiologic data were analyzed using descriptive and inferential statistics. Baseline data were analyzed to determine if there were differences between the two study groups.

Outcome Variables

Pulmonary artery pressure fluctuations were analyzed using descriptive statistics. The mean and maximum fluctuation and variance were described for each individual. The data were also analyzed to determine if there was increasing heteroscedasticity with increasing absolute pressure.

A two-way analysis of variance for repeated measures was used to determine the occurrence of any statistically significant differences in PA and PAW pressures due to position, position sequence, or interaction between position and sequence. If comparisons reached significance, a paired t-test was performed to identify specific group differences. An alpha of .05 was used.

Interrater reliability (IRR) was obtained by independent interpretation of a random sample of 10% of the analog data strips by the primary investigator and an experienced critical care nurse. Reliability was assessed by calculation of a correlation coefficient. In

addition, both investigators reanalyzed any measurements that differed by greater than 2 mm Hg, and the method for interpretation was clarified

The pressure fluctuation observed during the position portion of the study was compared with the baseline fluctuation. This analysis was performed to determine if characterization of fluctuation described during the baseline period was applicable to the position portion of the study, as there was a small increase in the absolute pressures in the supine position with 0-degree backrest elevation relative to the baseline pressures. Prior to comparison the experimental (position-related) effects were eliminated by excluding position-related data that were significantly different from the supine measurements. For example, the PAS pressures measured in the left-lateral position were significantly higher ($p = .00$) than PAS pressures in all other positions; therefore, these pressures were excluded and the pressure fluctuations in the remainder of the data were analyzed.

Agreement analysis (Bland & Altman, 1986; Bland & Altman, 1995; Szaflarski & Slaughter, 1996) was used to determine the extent of agreement between pressures in the supine and lateral positions, and to answer the question: Are the measurements obtained in the 30-degree lateral position interchangeable with those obtained in the supine position? This method of analysis depends on the description of the following: (1) The bias (mean difference between the measurements), which reflects the systematic error between the measurements. A bias of 0 indicates perfect agreement between the two measurements. (2) The limits of agreement ($LOA = \text{bias} \pm 2 \text{ SD}$), which for normally distributed (Gaussian) differences includes 95% of the differences. (3) The precision of

the bias, which is equal to the bias $\pm$ standard error of the bias. The limits of agreement are only estimates of the values that apply to the whole population (Bland & Altman, 1986); thus, second sample would give different limits. Therefore, the precision of the LOA describes the precision of the estimates if the differences followed a normal distribution. The precision of the limits of agreement allows for comparison across samples.

The mean of the measures (e.g., mean of Supine-1, Supine-2, Left) on the X-axis and the difference between the measures on the Y-axis. On the opposing Y-axis the bias and limits of agreement were plotted. In addition, the pressures indicative of clinical significance were plotted on the Y-axis to allow for graphical comparison of these points relative to the limits of agreement. If the differences within the limits of agreement were not considered clinically significant (i.e., the limits of agreement did not exceed the level of clinical significance), the recommendation was made that measurements obtained in the supine and the specified lateral position were interchangeable. If the limits of agreement exceeded the level of clinical significance it was interpreted that while most patients would not demonstrate clinically significant position-related pressure changes, individual responses needed to be assessed.

Finally for individual data, position-related pressure changes were graphically compared with baseline pressure fluctuation. Two questions were asked relative to the individual data: (1) What was the frequency of position-related pressure changes relative to baseline frequency? That is, how many subjects demonstrated position-related

pressure changes that were above or below baseline fluctuation? (2) Did individuals who demonstrated increased changes in baseline fluctuation also demonstrate increased position-related pressure changes? That is, did the presence of increased baseline fluctuation allow for the prediction of which individuals would demonstrate increased position-related pressure changes?

CHAPTER IV

RESULTS

To determine the effect of the 30-degree lateral recumbent position on PA and PAW pressures, 42 critically ill adults, who were status post-cardiac surgery, were studied. A repeated measures experimental design was used. This chapter begins with a description of the sample characteristics and the baseline fluctuation in PA and PAW pressures over time. Based on the baseline fluctuation data, pressure changes deemed to be clinically significant were specified. Findings related to the effect of the 30-degree lateral recumbent position are then presented, followed by a description of the individuals who demonstrated clinically significant pressure changes. The chapter concludes with a summary of the findings.

Sample Characteristics

Forty-two patients who underwent cardiac surgery were included in the study. Seven of these subjects were excluded from the study of the effect of lateral position on PA pressures. Subject #39 was excluded after the third measurement of the baseline fluctuation due to a vasovagal bradycardia, which was unrelated to the study. Subject #41 was excluded due to nausea after the fourth baseline measurement. Four subjects were excluded after supine-1 for a variety of reasons including coughing with probable airway obstruction (Subject # 13- in lateral position), nausea (Subjects # 21 and # 42), and increased blood pressure (Subject # 32 – in lateral position and family members in to visit). Finally, Subject # 29, who completed the study, was excluded because of the

effect of suctioning after Supine-1 on the measured pressures. There were no significant differences ($p > .05$) in baseline demographics, cardiopulmonary variables, or surgical information between those included and excluded. Therefore, 42 subjects were included in the description of baseline fluctuation of PA pressures over a 15 minute period, and 35 subjects were included in the statistical analysis related to the effect of lateral position on PA pressure measurements.

The sample consisted of 31 men and 11 women, with ages ranging from 27 to 89, with a mean (± 1 SD) of 63.6 ± 11.2 years. Table 2 provides a summary of the patient's demographics and surgical information, while Table 3 provides the patient's baseline cardiopulmonary indices. There were no significant differences between groups (Group A: Supine-Right-Left-Supine; Group B: Supine-Left-Right-Supine) in demographic or surgical information or baseline cardiopulmonary indices. Individual data including baseline cardiopulmonary indices, medical history, surgical information, and medications administered before or during the study are located in Appendix G.

Interrater Reliability

Prior to data analysis, a random selection of 10% of the analog data strips were interpreted by the primary investigator and an experienced critical care nurse. The pressure waveforms were interpreted as specified in the study protocol (Appendix E). Interrater reliability was very high for all measures: PAS: $r = 0.996$, $p = .01$; PAED: $r = .988$, $p = .01$; PAM: $r = .994$, $p = .01$; PAW: $r = .991$, $p = .01$. In addition the mean difference between the measurements from each rater was small: PAS: -0.23 ± 0.97

TABLE 2. Baseline Characteristics of Sample (N = 42) by Group

Variable	Group A (n = 23) Mean $\pm$ SD	Group B (n = 19) Mean $\pm$ SD
Age (years)	64 $\pm$ 11	66 $\pm$ 15
Sex		
-Male (n)	17	14
-Female (n)	6	5
Height (cm)	175 $\pm$ 8	172 $\pm$ 6
Weight (kg)	81 $\pm$ 13	87 $\pm$ 19
Body Surface Area (m ²)	1.95 $\pm$.17	2.00 $\pm$.22
Body Mass Index (Quetelet)	28.69 $\pm$ 6.32	31.28 $\pm$ 8.35
% Ideal Body Weight (% Quetelet) ⁺	119 $\pm$ 20	129 $\pm$ 27
Bypass Time (min)	143 $\pm$ 51	153 $\pm$ 50
Ischemic Time (min)	111 $\pm$ 42	116 $\pm$ 41
Surgery		
-Coronary Artery Bypass Graft	10	12
-Aortic Valve Replacement	4	1
-Mitral Valve Replacement	3	1
-CABG & Valve Replacement	4	3
-Ross Procedure	1	1
-Atrial Septal Defect Repair	1	0
Grafts (n)	2.2 $\pm$ 2.1	2.9 $\pm$ 1.6
Hours after surgery	16.5 $\pm$ 3.5	18.5 $\pm$ 4.6
Chest Tube Output (ml/3°)	69 $\pm$ 61	92 $\pm$ 64
Pre-Study Backrest Elevation (degrees)	23 $\pm$ 12	23 $\pm$ 11
Midsleep (hour)	0220 $\pm$ 130	0311 $\pm$ 115
Number of hours study performed after midsleep	7.4 $\pm$ 3.2	7.0 $\pm$ 1.9
Days on Bedrest Before Surgery	0.5 $\pm$ 1	0.22 $\pm$ 0.94
Position of PA Catheter on Chest Radiograph		
-Main PA	6	8
-Right PA	8	9
-Left PA	1	0
-RV Outflow	8	2

+ Based on medium body mass

TABLE 3. Baseline Cardiopulmonary Indices

Variable	Group A (n =23) Mean $\pm$ SD	Group B (n = 19) Mean $\pm$ SD
Cardiac Output (L/min)	6.70 $\pm$ 2.15	6.43 $\pm$ 1.91
Cardiac Index (L/min/m ²)	3.39 $\pm$ 1.01	3.37 $\pm$.78
Stroke Volume (ml/beat)	76 $\pm$ 26	75 $\pm$ 18
Heart rate (beats/minute)	90 $\pm$ 11	87 $\pm$ 14
Normal sinus rhythm	14	12
Sinus Tachycardia	1	2
Atrial Flutter/Fibrillation	5	1
Sinus Rhythm with Heart Block	1	0
Sinus Rhythm with Ectopy	0	1
Paced	2	3
Systemic Vascular Resistance (dynes/sec/cm ⁻⁵)	739 $\pm$ 194	849 $\pm$ 236
Systolic blood pressure (mm Hg)	114 $\pm$ 13	113 $\pm$ 19
Diastolic blood pressure (mm Hg)	52 $\pm$ 10	57 $\pm$ 10
Mean blood pressure (mm Hg)	72 $\pm$ 10	76 $\pm$ 12
Right atrial pressure (mm Hg)	10 $\pm$ 3	10 $\pm$ 3
Pulmonary Artery Temperature (°C)	37.3 $\pm$.60	37.2 $\pm$.43
Respiratory rate (breaths/minute)	18 $\pm$ 4	19 $\pm$ 4
FiO ₂	.37 $\pm$.07	.38 $\pm$.01
SaO ₂ (%)	97 $\pm$ 2	95 $\pm$ 3
Spontaneous Ventilation	20	17
Mechanical Ventilation	3	2

mm Hg; PAED: -0.02 ± 1.15 mm Hg; PAM: -0.27 ± 0.86 mm Hg; PAW: -0.31 ± 0.85 mm Hg. Therefore, data interpretation was considered reliable.

Baseline Fluctuation in PA and PAW Pressures

Baseline pressure fluctuations were determined to provide a means of comparison for the pressure changes observed during the positioning portion of the study. Forty-two post-cardiac surgery patients were included in this portion of the study. Subjects were studied in the supine position at the backrest elevation in which they were found (mean 23.7 ± 11.6 - degrees, ranging from 0- to 47- degrees). The frequency of subjects in the various degrees of backrest elevation is summarized as follows:

	0-	1- to 10-	11 to 20-	21 to 30-	31 to 40-	41+
	Degrees	Degrees	Degrees	Degrees	Degrees	Degrees
Number	1	5	13	12	6	4
Percentage	2.4	12.2	31.7	29.3	14.6	9.8
Cumulative percentage	2.4	14.6	46.3	75.6	90.2	100

Four subjects were repositioned from the lateral position, while the remaining 38 were found in a supine position.

Reference Level

The supine LA reference level was identified at the phlebostatic axis (Winsor & Burch, 1945). The LA reference level (the point one-half the distance from the left sternal border at the fourth intercostal space to the surface of the bed) as specified by VanEtta

(VanEtta, Gibbons, & Woods, 1993; VanEtta, 1992) was used to identify the level of the left atrium for the 30-degree right- and left-lateral positions. In all cases the patients were on Baxter Maxifloat™ mattresses. One subject also had an egg-crate mattress; however, a flat plate caliper was used to ensure that the chest wall at the surface of the bed was correctly identified. The reference points (distance from the surface of the bed) were as follows: Supine 9.1 ± 1.8 cm; Right Lateral 10.3 ± 1.5 cm, and Left Lateral 9.2 ± 1.5 cm (Figure 14). There were statistically significant differences between the Supine and Right-Lateral (Figure 15A) (mean difference = -1.3 ± 1.3 cm; 95% CI = -1.7, 0.8, $p = .000$) and Right- and Left-Lateral reference points (Figure 15C) (mean difference = 1.1 ± 1.1 cm; 95% CI = 0.8, 1.6, $p = .000$). There was no difference between the Supine and Left-lateral reference levels (Figure 15B) (mean difference = -0.08 ± 1.4 cm; 95% CI = -0.6, 0.4, $p > .05$). The maximum difference between the Supine-Right and Right-Left reference levels was 3.5 cm, which is equivalent to a potential 2.6 mm Hg pressure difference; while one subject had a 5 cm difference (3.7 mm Hg) between the Supine-Left reference levels.

Definition of Clinically Significant Pressure Fluctuations

Prior to specification of the absolute pressure indicative of clinical significance, (i.e., a physiologic change not directly attributable to the expected pressure fluctuation), the data were analyzed for a normal distribution. If the data were normally distributed the absolute pressure fluctuation indicative of clinical significance was the mean ± 2 SD, which would include 95% of all cases. If the data were not normally distributed, as

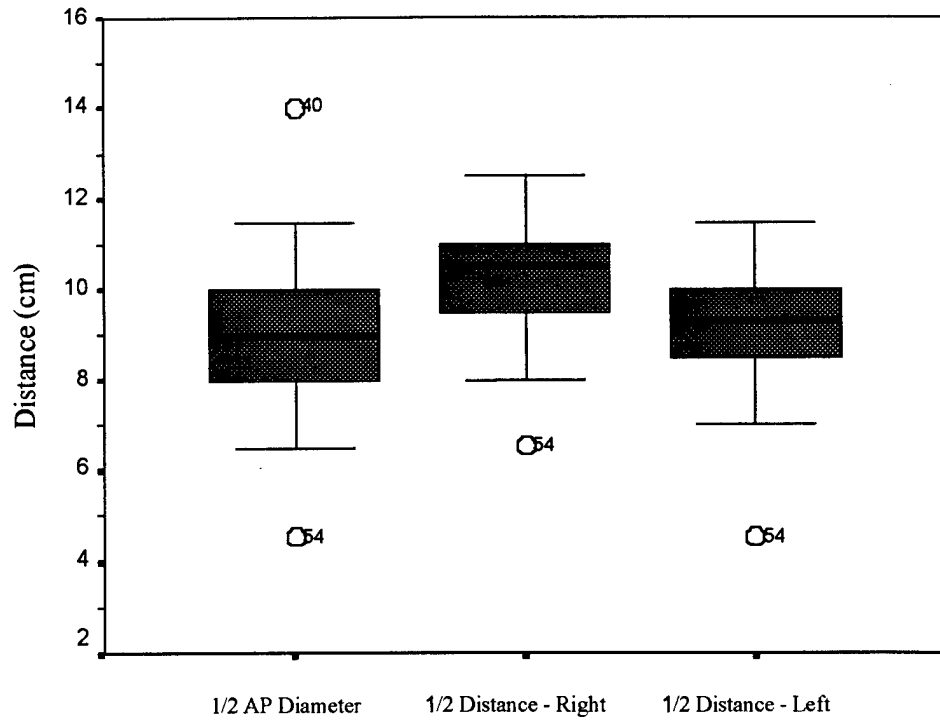
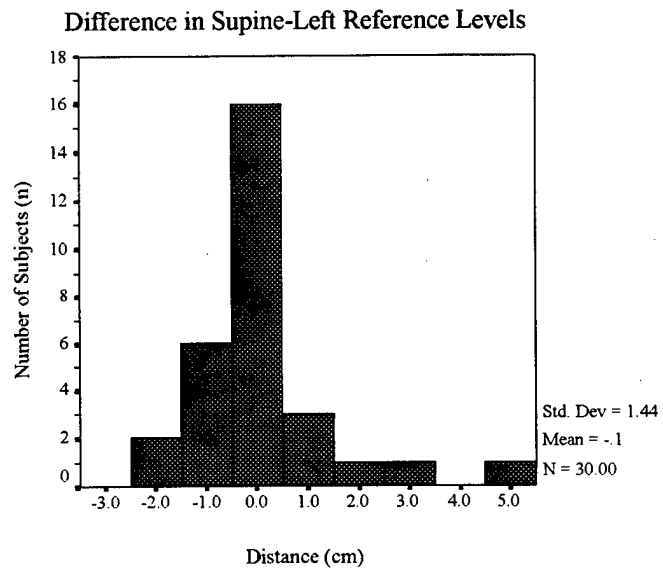


FIGURE 14. Height of LA Reference Levels (Supine and Lateral Positions). AP = Anteroposterior ; Right = 30-degree right lateral position; Left = 30-degree left lateral position

A.



B.

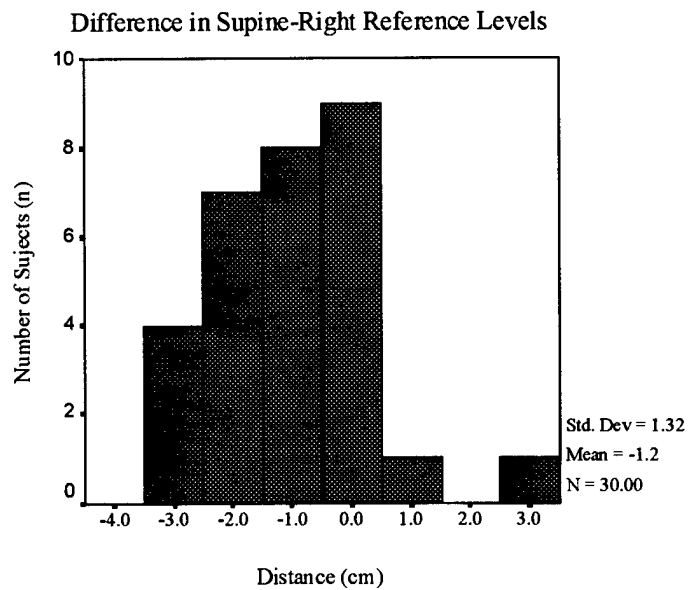


FIGURE 15. Difference in LA Reference Levels in Supine and Lateral Positions

C.

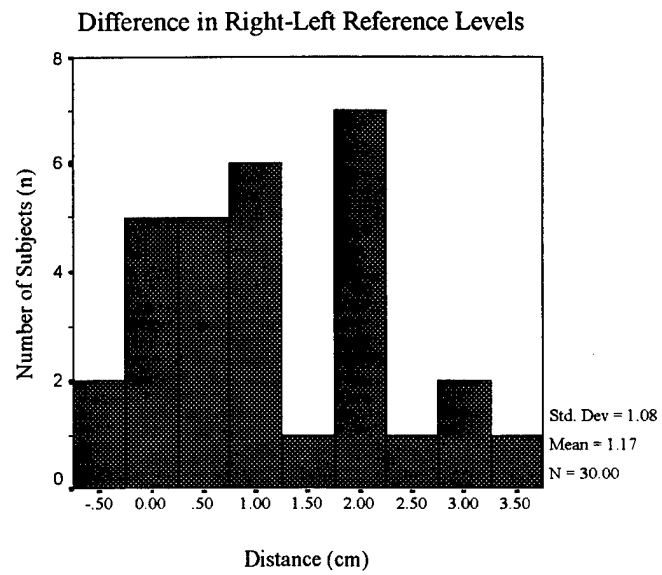


FIGURE 15. Difference in LA Reference Levels in Supine and Lateral Positions (continued)

determined by (1) examination of a histogram of the data, (2) mean approximately equal to median, and (3) Pearson's Skewness Coefficient (0 = perfect symmetry; skewness > 0.2 or < -0.2), the cumulative percentage on a frequency table of absolute pressure fluctuation was evaluated for the pressure that included 95% of the cases. The latter method was appropriate as it was unaffected by the presence of extreme values. The distributions of the PAED and PAS pressures were normally distributed, while the PAM and PAW pressures were not.

Pulmonary Artery Systolic Pressure

Over the 15-minute study period a total of 165 PAS pressure measurements were obtained from the 42 subjects. The PAS pressures ranged from 17 to 50 mm Hg, with a mean (± 1 SD) of 30.6 ± 8.7 mm Hg. Individual data are summarized in Appendix H.

Pulmonary artery systolic pressure fluctuation over the 15-minute study period ranged from 0 to 6 mm Hg, with a mean of 2.7 ± 1.5 mm Hg (95% CI = 2.3, 3.2), and a median of 3.0 mm Hg. The data were normally distributed; therefore, the cutoff point for clinical significance was defined as the pressure included within ± 2 SD of the mean.

The frequencies of the pressure fluctuations are summarized in Table 4. A 4.3 mm Hg pressure fluctuation reflected inclusion of a sample that was within 1 SD of the mean, while a 5.8 mm Hg pressure reflected inclusion of a sample that was within 2 SD of the mean. Analysis of the frequency table indicated that a pressure fluctuation of 5 mm Hg included 98% of the sample, therefore, a pressure fluctuation greater than 5 mm Hg was defined as a clinically significant fluctuation for PAS pressure.

TABLE 4. Frequency of PAS Pressure Fluctuations (N = 42)

Fluctuation (mm Hg)	Frequency	Percentage	Cumulative Percentage
0	1	2.4	2.4
1	10	23.8	26.2
2	8	19.0	45.2
3	7	16.7	61.9
4	11	26.2	88.1
5	4	9.5	97.6
6	1	2.4	100

Pulmonary Artery End-Diastolic Pressure

Over the 15-minute study period a total of 161 PAED pressure measurements were obtained from the 42 subjects. The PAED pressures ranged from 7 to 34 mm Hg, with a mean (± 1 SD) of 17.1 ± 5.0 mm Hg (95% CI = 15.5, 18.6). Individual data are summarized in Appendix H.

Pulmonary artery end-diastolic pressure fluctuation over the 15 minute study period ranged from 0 to 7 mm Hg, with a mean of 2.2 ± 1.5 mm Hg (95% CI = 1.8, 2.7), and a median of 2.0 mm Hg. The data were normally distributed; therefore, clinical significance was defined as the pressure included within ± 2 SD of the mean.

The frequencies of the pressure fluctuations are summarized in Table 5. A 3.7 mm Hg pressure fluctuation reflected inclusion of a sample that was within 1 SD of the mean, while a 5.1 mm Hg reflected inclusion of a sample that was within 2 SD of the mean. Therefore, a pressure fluctuation greater than 5 mm Hg was defined as clinically significant for PAED pressure. This pressure fluctuation was confirmed by analysis of the frequency table that indicated a 4 mm Hg pressure fluctuation included 93% of the sample, while a 5 mm Hg included 98% of the sample.

Pulmonary Artery Mean Pressure

Over the 15-minute study period a total of 164 PAM pressure measurements were obtained from the 42 subjects. The PAM pressures ranged from 10 to 41 mm Hg, with a mean (± 1 SD) of 22.2 ± 5.7 mm Hg (95% CI = 20.4, 24.0). Individual data are summarized in Appendix H.

TABLE 5. Frequency of PAED Pressure Fluctuations

Fluctuation (mm Hg)	Frequency	Percentage	Cumulative Percentage
0	3	7.1	7.1
1	12	28.6	35.7
2	11	26.2	61.9
3	10	23.8	85.7
4	3	7.1	92.9
5	2	4.8	97.6
6	1	2.4	100.0

Pulmonary artery mean pressure fluctuation over the 15-minute study period ranged from 0 to 9 mm Hg, with a mean of 2.6 ± 1.8 mm Hg (95% CI = 2.1, 3.2), and a median of 2.0 mm Hg. The distribution of the PAM pressure was positively skewed as indicated by the difference between the mean and the median. Because the data were skewed, the frequency table was evaluated to identify the absolute pressure that included 95% of the cases. The frequencies of the pressure fluctuations are summarized in Table 6. In this case, a fluctuation of 5 mm Hg included 95.2% of the cases; thus a pressure fluctuation of greater than 5 mm Hg would be considered clinically significant for PAM pressure.

Pulmonary Artery Wedge Pressure

Over the 15-minute study period a total of 94 PAW pressure measurements were obtained from the 42 subjects. There were fewer PAW pressure measurements obtained due to the intraoperative withdrawal and positioning of the PA catheter in the main PA, which in many cases, did not permit wedging of the catheter. The PAW pressures ranged from 4 to 26 mm Hg, with a mean (± 1 SD) of 13.6 ± 5.3 mm Hg. Individual data are summarized in Appendix H.

Pulmonary artery wedge pressure fluctuation over the 15-minute study period ranged from 0 to 6 mm Hg, with a mean of 1.7 ± 1.4 mm Hg (95% CI = 1.1, 2.3), and a median of 2.0 mm Hg. The distribution of the PAW pressure was negatively skewed as indicated by the difference between the mean and the median

The frequencies of the pressure fluctuations are summarized in Table 7. A review of the frequency table indicates that a 4 mm Hg pressure fluctuation includes 96% of the

TABLE 6. Frequency of PAM Pressure Fluctuations

Fluctuation (mm Hg)	Frequency	Percentage	Cumulative Percentage
0	3	7.1	7.1
1	7	16.7	23.8
2	14	33.3	57.1
3	5	11.9	69.0
4	9	21.4	90.5
5	2	4.8	95.2
6	1	2.4	97.6
9	1	2.4	100.0

TABLE 7. Frequency of PAW Pressure Fluctuations

Fluctuation (mm Hg)	Frequency	Percentage	Cumulative Percentage
0	5	20.0	20.0
1	7	28.0	48.0
2	8	32.0	80.0
3	3	12.0	92.0
4	1	4.0	96.0
5	0	0.0	96.0
6	1	4.0	100.0

sample; therefore, a pressure fluctuation greater than 4 mm Hg was defined as clinically significant for PAW pressure.

Effect of Position on PA and PAW Pressures

A repeated-measures analysis of variance (RANOVA) was performed to examine the effect of supine, and right and left 30-degree lateral positions on PA pressure measurements. The three null hypothesis addressed for each PA pressure (systolic, end-diastolic, mean, and wedge) were:

- 1) There were no significant differences in PA pressure measurements dependent on the sequence of positioning
- 2) There were no significant differences in PA pressure measurements in the supine versus right and left 30-degree lateral positions
- 3) There was no interaction between sequence of positioning and position on PA pressure measurements

Subject Characteristics

Individual PA systolic, end-diastolic, mean, and wedge pressures in each of four positions (supine-1, right, left, and supine-2) are summarized in Appendix I. Thirty-six subjects were included in the position portion of the study. There were 26 men (72%) and 10 women (28%), aged 63.3 ± 13.3 years. Coronary artery bypass graft (CABG) surgery was performed in 18 subjects (50%), while seven subjects (19%) had a combination CABG surgery and valve repair. Five subjects (14%) had an aortic valve repair, three subjects (8%) had mitral valve repair, two (6%) underwent a Ross procedure,

and one subject (3%) had an atrial-septal defect repair. Thirty-two of the subjects (89%) were breathing spontaneously, while four of the subjects were mechanically ventilated. Subject #29, who was excluded after the study, was mechanically ventilated. Thirty-five of the 36 (97%) subjects were receiving intravenous vasoactive medications during the study; however, the medications were not adjusted during the study period. Twenty-one subjects received nitroglycerin, (mean dose = $1.45 \pm 4.7 \mu\text{g/kg/min}$; 14 subjects received dopamine (mean dose = $2.03 \pm 0.8 \mu\text{g/kg/min}$), nine subjects received sodium nitroprusside (mean dose = $0.79 \pm 0.60 \mu\text{g/kg/min}$); three subjects received dobutamine (mean dose = $1.50 \pm 0.50 \mu\text{g/kg/min}$), three subjects received epinephrine (mean dose = $0.11 \pm 0.08 \mu\text{g/kg/min}$); one subject received milrinone (dose = $0.14 \mu\text{g/kg/min}$) and one subject received diltiazem (dose = 2.5 mg/hr). Twenty-two subjects (61%) received a single drug, 10 subjects (28%) received a combination of two drugs, and three subjects (8%) received three vasoactive medications.

Four subjects received continuous infusions of a sedative or analgesic agent during the study. Three subjects received propofol with a mean dose of $172 \pm 169 \mu\text{g/hr}$, and two subjects received fentanyl with a mean dose of $40 \pm 30 \mu\text{g/hr}$. None of the subjects received intravenous morphine or furosemide in the hour before the study. As noted, there were no significant differences ($p > .05$) between those included or excluded from this portion of the study, thus the sample characteristics outlined in Tables 2 and 3 are representative of this group of subjects.

The stabilization periods and total time for the study were on average 5 and 48.44 ± 7.02 minutes respectively. The average study time for the fluctuation portion of the study was 16.03 ± 1.80 minutes, while the position effect portion was 24.6 ± 7.44 minutes (ranging from 7 to 43 minutes). Seven subjects required a stabilization period of greater than 5 minutes; however, in only two of the subjects was the delay due to a failure of the vital signs to return to baseline. Physician visits, nausea, and suctioning were the other factors associated with delays.

Interaction Effect of Sequence and Position on PA Pressures

There was no significant interaction effect between sequence and position for PAS ($F = 1.32$, $p = .327$), PAED ($F = 0.9$, $p = .494$), PAM ($F = 0.7$, $p = .460$) or PAW ($F = 0.07$, $p = .974$) pressures (Table 8). Because there were no interaction effects, the data from Sequence Group A and B were combined for analysis for all pressures.

Main Effect of Sequence on PA Pressures

There were no significant differences between any of the PA pressures between subjects assigned to Sequence A (S1-R-L-S2) and Sequence B (S1-L-R-S2) for PAS ($F = .097$, $p = .768$), PAED ($F = .598$, $p = .503$), PAM ($F = .340$, $p = .599$) or PAW ($F = .089$, $p = .815$) pressures (Table 8). Therefore, the null hypothesis of no difference in PA pressures based on the sequence of position failed to be rejected.

TABLE 8. Analysis of Variance for Pulmonary Artery Pressures

Source of Variation	Sum of Squares	df	Mean Square	F	p value
<u>PAS</u>					
Sequence	9.776	1	9.776	.097	.060
Position	75.960	3	25.320	8.380	.000
Sequence*Position	11.832	3	3.944	1.323	.274
<u>PAED</u>					
Sequence	20.546	1	20.546	.498	.447
Position	30.270	3	10.090	3.173	< .05
Sequence*Position	8.638	3	2.876	.901	> .05
<u>PAM</u>					
Sequence	16.823	1	16.823	.340	.565
Position	36.910	3	12.303	3.709	.015
Sequence*Position	7.301	3	2.434	.725	.540
<u>PAW</u>					
Sequence	1.056	1	1.056	.059	.815
Position	14.875	3	4.958	1.615	.171
Sequence*Position	.675	3	.225	.073	.974

Main Effect of Position on PA Pressures

There was a significant position effect for PAS ($F = 8.38$, $p = .000$), PAED ($F = 3.17$, $p < .05$), PAM ($F = 3.7$, $p < .05$). The position effect was not significant ($F = 1.61$, $p = .171$) for PAW pressure (Table 8). Because the RANOVA omnibus F test was significant, post hoc analysis was performed for each pressure (PAS, PAM, PAED) and pressure-pair using a paired t-test to determine which of the specific position-pairs were significantly different. Analysis for each pressure (PAS, PAED, PAM, and PAW) follows.

Pulmonary Artery Systolic Pressure

As indicated by the RANOVA results there was a significant position effect ($F_{3,72} = 8.38$, $p = .000$) with respect to the PAS pressure. As summarized in Figure 16, the post-hoc paired t-test (Table 9) revealed that the PAS pressure in the 30-degree Left lateral position was significantly greater than PAS pressures measured in the Supine-1 ($p = .000$), Right 30-degree lateral ($p = .03$), and Supine-2 positions ($p = .000$). In all three cases, the PAS in the Left-lateral position was greater than the PAS in the Supine-1 (mean difference = 2.0 ± 2.1 mm Hg; 95% CI = 1.2, 2.8 mm Hg), Right-lateral (mean difference = 1.2 ± 2.8 mm Hg; 95% CI = 0.1, 2.2 mm Hg) and Supine-2 positions (mean difference = 1.9 ± 2.4 mm Hg; 95% CI = 1.0, 2.8 mm Hg). Individual data are presented in Appendix J, and are graphically summarized in Figure 17.

As demonstrated by the 95% CIs, the pressure differences were small. For example, the 95% CI for the pressure difference between Left-lateral and Supine-1 positions was

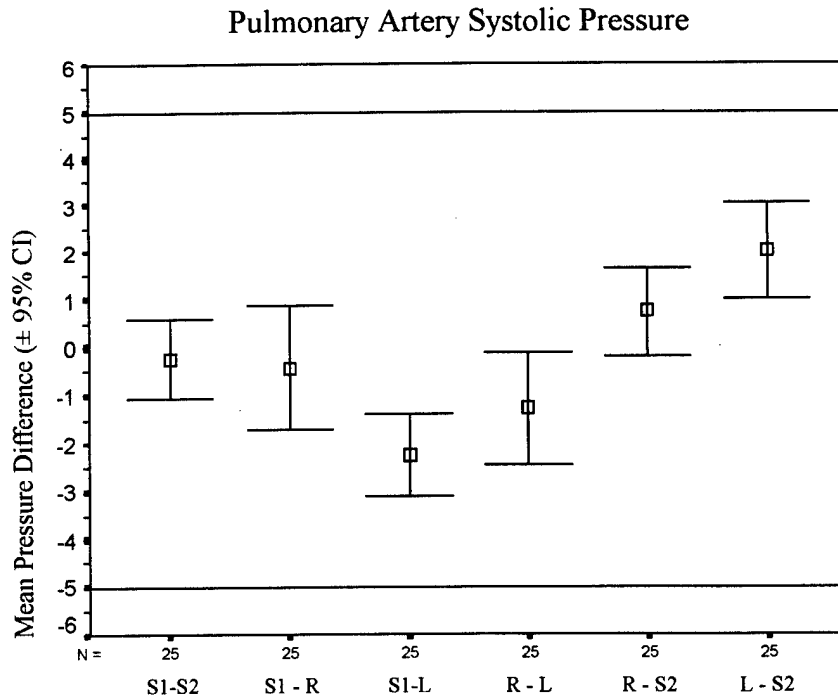


FIGURE 16. Mean Differences ($\pm$ 95% CI) in PAS Pressure-Position-Pairs (***) $p < .001$

TABLE 9. Paired Samples Test for PAS Pressure Pairs

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	SD	SE	95% CI of Difference				
					Lower	Upper			
1	S1-S2	-0.3	2.0	0.4	-1.1	0.5	-0.697	25	.493
2	S1-Right	-1.0	2.8	0.5	-2.1	0.1	-1.817	27	.080
3	S1-Left	-2.0	2.1	0.4	-2.8	-1.2	-4.979	27	.000***
4	Right-Left	-1.2	2.8	0.5	-2.2	0.12	-2.283	28	.030*
5	Right- S2	0.9	2.3	0.4	-0.03	1.8	1.983	28	.057
6	Left – S2	1.9	2.4	0.4	1.0	2.8	4.367	29	.000***

* $p < .05$; ** $p < .01$; *** $p < .001$

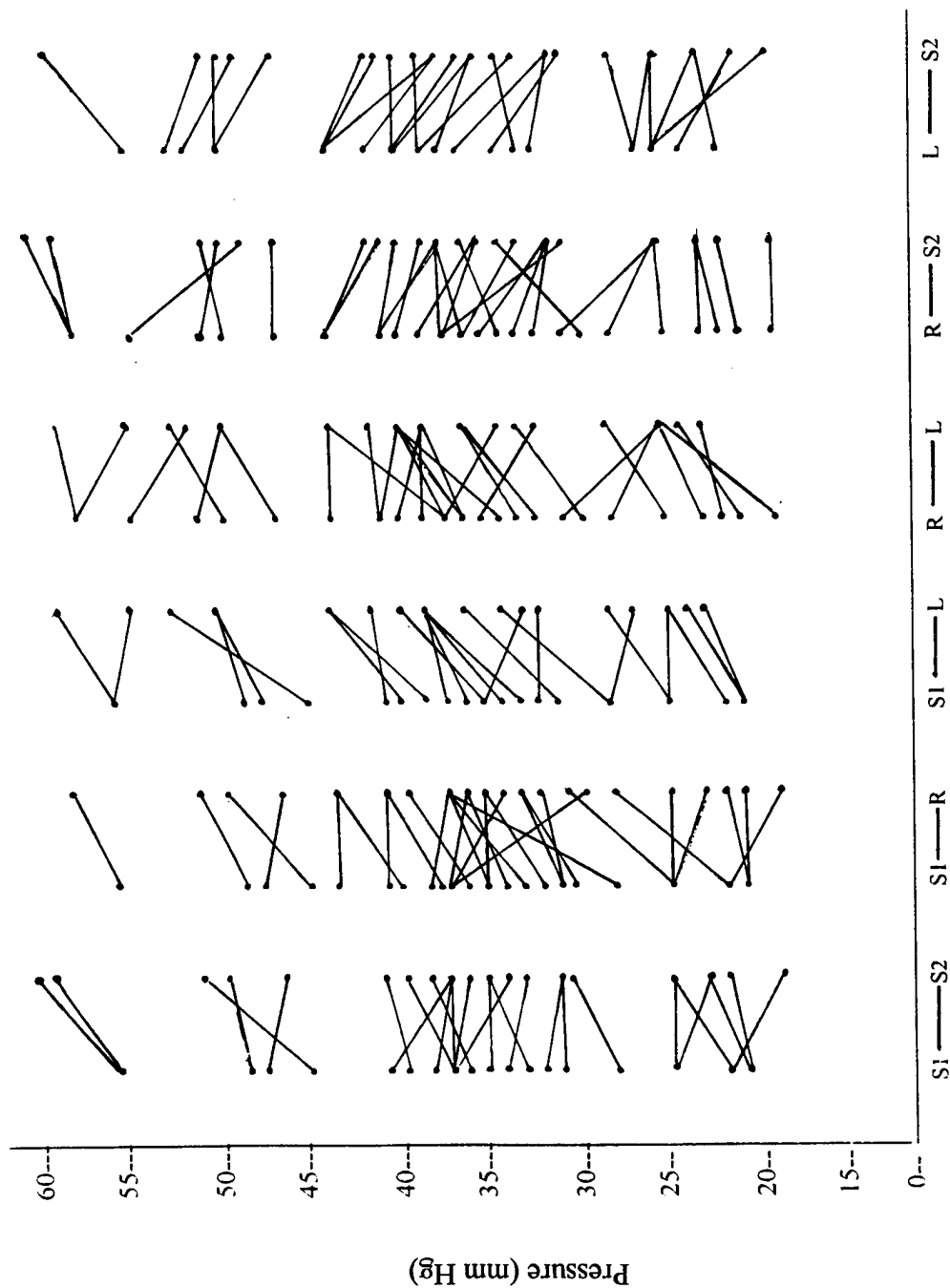


FIGURE 17. Summary of Individual PAS Pressure-Pair Differences. On average, the PAS pressures in the left-lateral position were significantly larger than Supine-1 ($p = .000$), Right ($p = .03$) and Supine-2 ($p = .000$) positions.

1.2 to 2.8 mm Hg, which was interpreted as indicating that with 95% confidence, we can state that on average the PAS pressure in the Left-lateral position was between 1.2 to 2.8 mm Hg greater than the PAS pressure in the Supine-1 position.

Pulmonary Artery End-Diastolic Pressure

Prior to interpretation of the RANOVA statistics for PAED pressure, correction of the critical F value was required because the assumption of sphericity (equal variance across measures) was not met (Mauchly's Test of Sphericity). The robustness of the RANOVA does not withstand violation of this assumption: therefore, adjustment of the degrees of freedom using an epsilon statistic was required (Munro, 1997). The degrees of freedom were corrected using the Huynh-Feldt epsilon statistic ($\epsilon = .868$) (Girden, 1992; Munro, 1997). Using this adjustment, the critical value for $F_{(2,63,.05)}$ was 2.36. The calculated F statistic ($F = 3.173$) was greater than the critical value, thus the omnibus F test for the RANOVA was significant ($p < .05$), and post-hoc paired t-tests were performed. Individual data are summarized in Figure 18 and are presented in Appendix I. The data from the paired sample statistics are presented in Appendix J.

The post-hoc paired t-test (Table 10 and Figure 19) revealed that the PAED pressures in the 30-degree left lateral position were significantly greater than PAED pressures measured in the Supine-2 position (mean difference = 1.4 ± 2.7 mm Hg; 95% CI = 0.4, 2.4 mm Hg, $p = .008$).

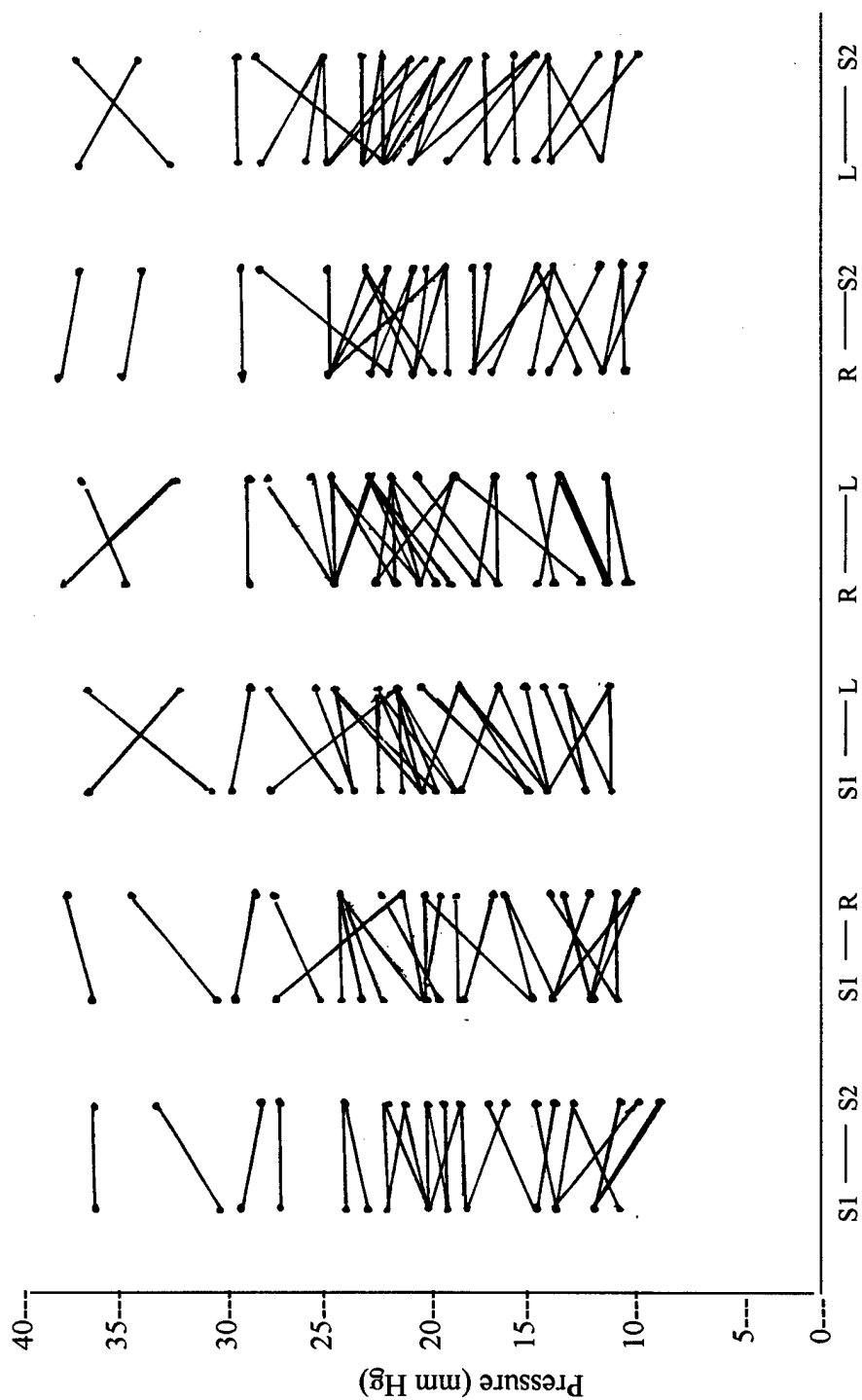


FIGURE 18. Summary of Individual PAED Pressure-Pair Differences. On average, the PAED pressures in the left-lateral position were significantly larger than pressures in the Supine-2 position ($p = .008$)

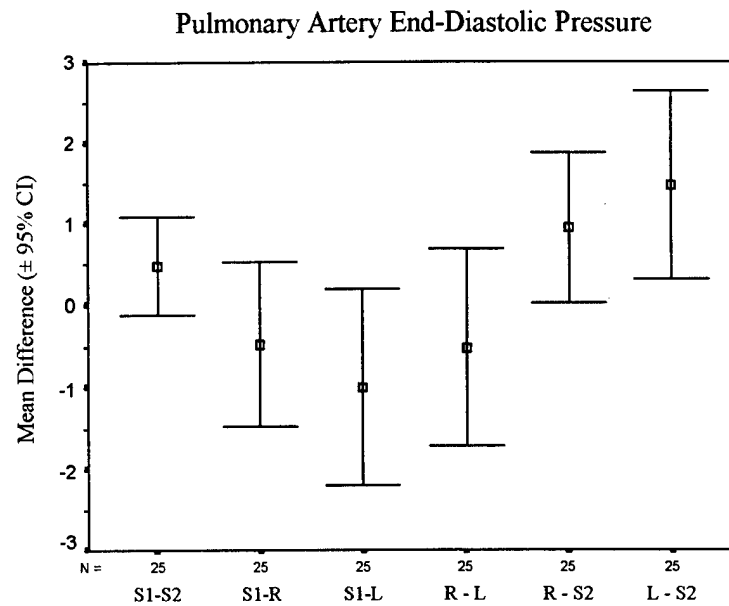


FIGURE 19. Mean Differences ($\pm$ 95% CI) in PAED Pressure Position-Pair
 (***) $p < .001$

TABLE 10. Paired Samples Test for Pulmonary Artery End-Diastolic Pressure

Pair		Paired Differences					t	df	Sig. (2-tailed)
		Mean	SD	SEM	95% CI of Difference				
					Lower	Upper			
1	S1-S2	0.5	1.4	0.28	-0.1	1.1	1.625	25	.117
2	S1-Right	-0.5	2.3	0.44	-1.4	0.4	-1.053	27	.302
3	S1-Left	-1.0	2.7	0.44	-2.0	0.1	-1.861	27	.074
4	Right-Left	-0.6	2.8	0.51	-1.6	0.5	-1.139	28	.264
5	Right- S2	0.8	2.2	0.41	-0.1	1.6	1.933	28	.063
6	Left – S2	1.4	2.7	0.48	0.4	2.4	2.830	29	.008**

*** $p < .001$

As demonstrated by the 95% CI, the pressure difference was small. The 95% CI was interpreted as indicating that with 95% confidence, we can state that on average the PAS pressure in the left-lateral position was between 0.4 to 2.4 mm Hg greater than the PAED pressure in the Supine-2 position. There was also a trend towards significance for the pairs Supine-1 - Left (mean difference = 1.0 ± 2.7 mm Hg, $p = 0.74$, 95% CI = -2.0, 0.1 mm Hg) and Right-Supine-2 (mean difference = 0.8 ± 2.2 mm Hg, $p = 0.63$, 95% CI = -0.5, 1.6 mm Hg). However, the differences as reflected by the 95% CIs were small.

Pulmonary Artery Mean Pressure

The RANOVA data indicated that there was a significant position effect ($F_{3,72} = 8.38$, $p = .015$) with respect to the PAM pressure (Table 8). Individual data are graphically summarized in Figure 20 and are presented in Appendix I. The data from the paired sample statistics are presented in Appendix J. The post-hoc paired t-tests (Table 11 and Figure 21) revealed that the PAM pressure in the 30-degree left lateral position was significantly greater than PAM pressure in the Supine-1 ($p = .014$) and Supine-2 positions ($p = .015$), and the PAM pressure in the 30-degree right lateral position was significantly greater than the PAM pressure in the Supine-2 position ($p = .024$). In all three cases the PAM pressure in the lateral position was greater than the PAM pressure in the supine positions: Supine-1 - Left (mean difference = -1.5 ± 2.9 mm Hg; 95% CI = 0.3, 2.6 mm Hg), Right-Supine 2 (mean difference = 0.8 ± 1.9 mm Hg; 95% CI = 0.1, 1.5 mm Hg) and Left-Supine-2 (mean difference = 1.2 ± 2.6 mm Hg; 95% CI = 0.3, 2.2).

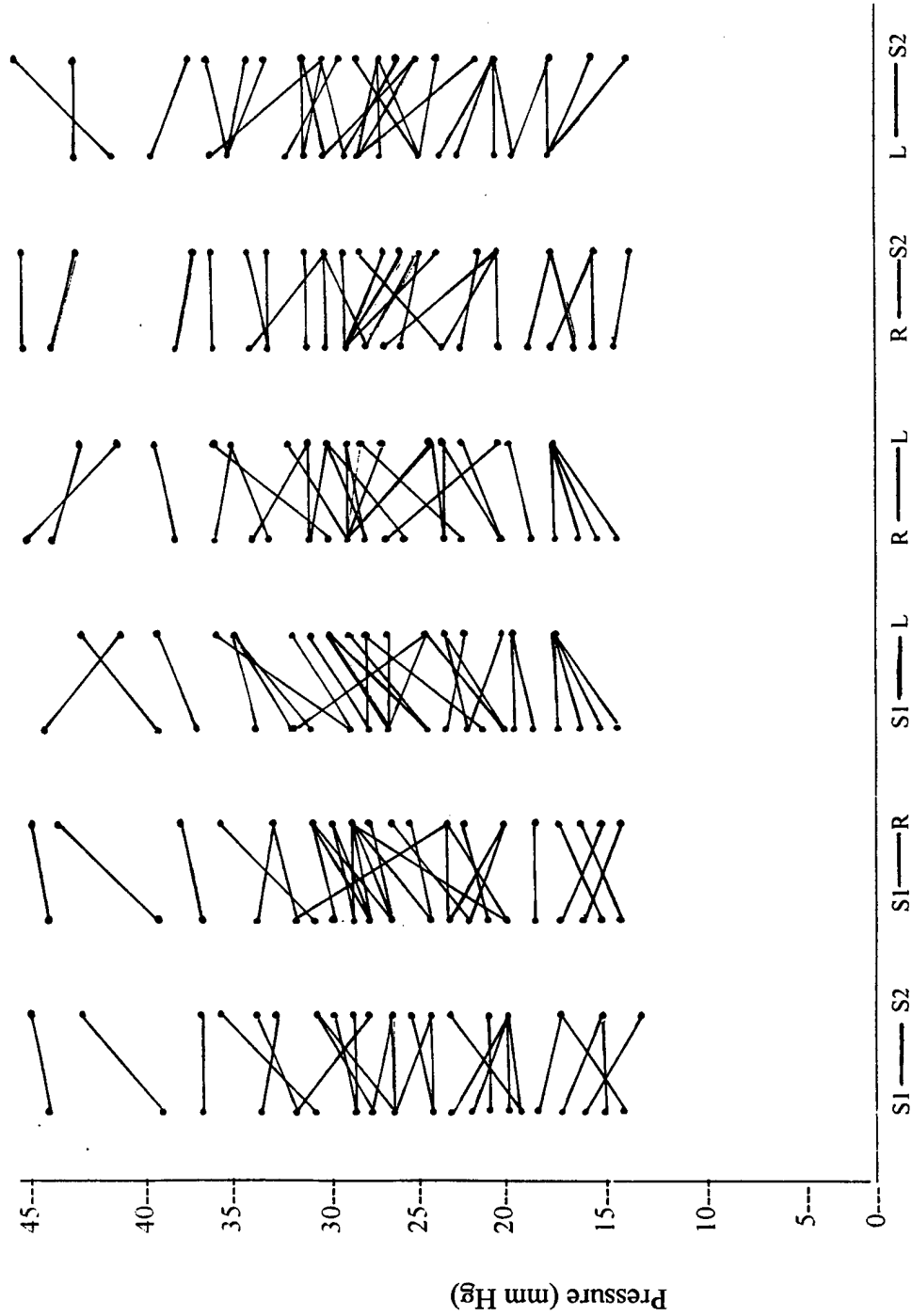


FIGURE 20. Summary of Individual PAM Pressure-Pair Differences. On average, the PAM pressures in the left-lateral position were significantly larger than pressures in the Supine-1 ($p = .014$), Supine-2 ($p = .015$), while the pressures in the right-lateral position were greater than the pressures in the Supine-2 position ($p = .024$).

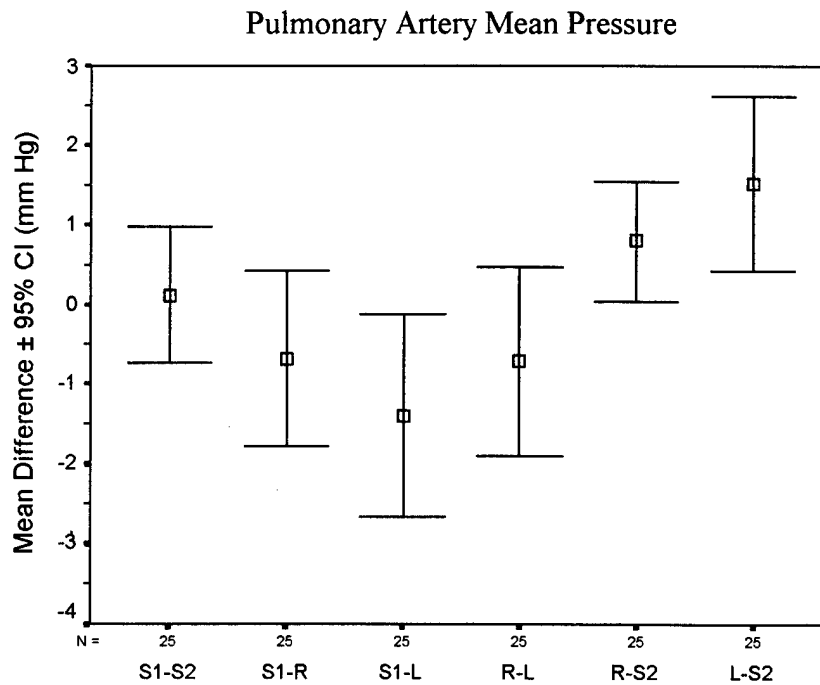


FIGURE 21. Mean Differences ($\pm$ 95% CI) in PAM Pressure Position-Pairs (* $p < .05$)

TABLE 11. Paired Samples Test for PAM Pressure

Pair		Paired Differences					t	df	Sig. (2-tailed)
		Mean	SD	SEM	95% CI of Difference				
					Lower	Upper			
1	S1-S2	-0.04	2.1	0.41	-0.8	0.9	0.095	25	.925
2	S1-Right	0.6	2.5	0.48	-1.6	0.3	-1.338	27	.192
3	S1 - Left	-1.5	2.9	0.56	-2.6	-0.3	-2.638	27	.014*
4	Right-Left	-0.5	2.8	0.52	-1.5	0.6	-0.855	28	.400
5	Right- S2	0.8	1.9	0.35	0.1	1.5	2.381	28	.024*
6	Left – S2	1.2	2.6	0.47	0.3	2.2	2.576	29	.015*

$p < .05$

As demonstrated by the 95% CIs, the pressure differences were small; with the largest difference being 2.6 mm Hg.

Pulmonary Artery Wedge Pressure

Analysis of the RANOVA data demonstrated that there was no significant position effect ($F_{3,27} = 1.800$, $p = .17$) with respect to the PAW pressure (Table 8); therefore, the performance of the post-hoc paired t-tests was not necessary. The failure to reject the null hypotheses of no sequence or position effect may reflect insufficient power due to the small sample size (10 to 16 pairs were analyzed). Therefore, the mean differences and the 95% confidence intervals of the difference were analyzed (Table 12 and Figure 22). Individual data are presented in Appendix I and are graphically summarized in Figure 23.

The absolute mean differences ranged from 0.1 to 1.6 mm Hg, while the 95% CIs ranged between 3.3 mm Hg less than to 3.5 mm Hg greater than the accompanying pressure in the pair. The smallest mean difference between a supine and lateral position pressure was between the Supine-1 and Left positions (mean difference -0.9 ± 1.7 mm Hg, 95% CI $-1.8, 3.5$). The 95% CI indicates that with 95% confidence, we can state that, on average the PAW pressure in the Left-Lateral position was between 1.8 mm Hg less and 3.5 mm Hg greater than the PAW pressure in the Supine-1 position. The largest mean difference was between the Supine-1 and Right-Lateral positions (mean -1.6 ± 2.4 mm Hg, 95% CI $= -3.3, 0.1$ mm Hg). The 95% was interpreted as indicating that with 95% confidence we can say that on average the PAW pressure in the Right-Lateral

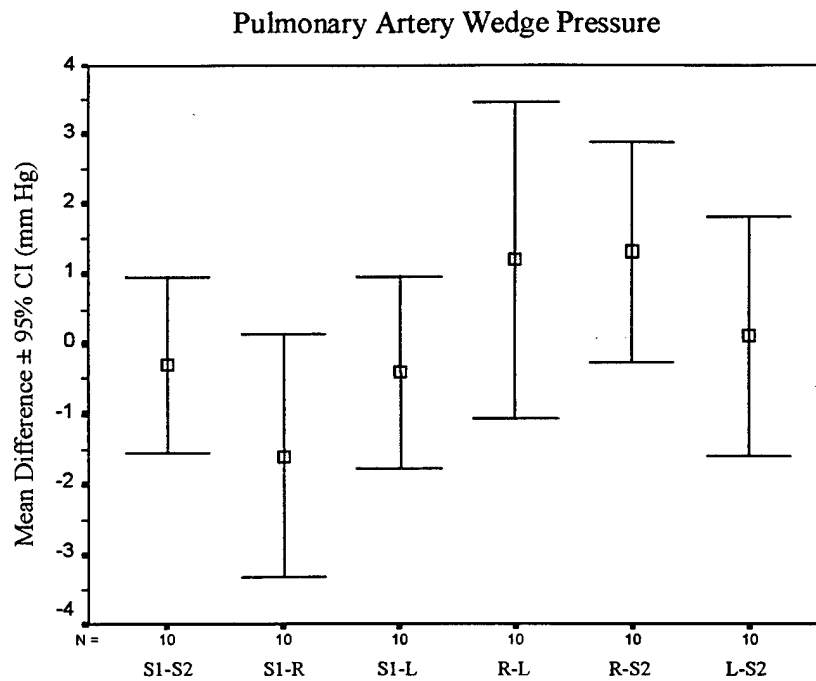


FIGURE 22. Mean Differences ($\pm$ 95% CI) in PAW Pressure Position-Pairs

TABLE 12. Paired Samples Test for Pulmonary Artery Wedge Pressure

Pair		Paired Differences					t	df	Sig. (2-tailed)
		Mean	SD	SEM	95% CI of Difference				
					Lower	Upper			
1	S1-S2	-0.8	1.6	0.4	-1.7	0.06	-1.979	15	.066
2	S1-Right	-1.6	2.4	0.8	-3.3	0.1	-2.097	9	.065
3	S1-Left	-0.9	1.7	0.4	-1.8	0.1	-2.049	15	.058
4	Right-Left	1.2	3.2	1.0	-1.1	3.5	1.203	9	.260
5	Right- S2	1.3	2.2	0.7	-2.9	0.3	1.857	9	.096
6	Left – S2	0.1	2.1	0.5	-1.2	1.1	0.126	14	.902

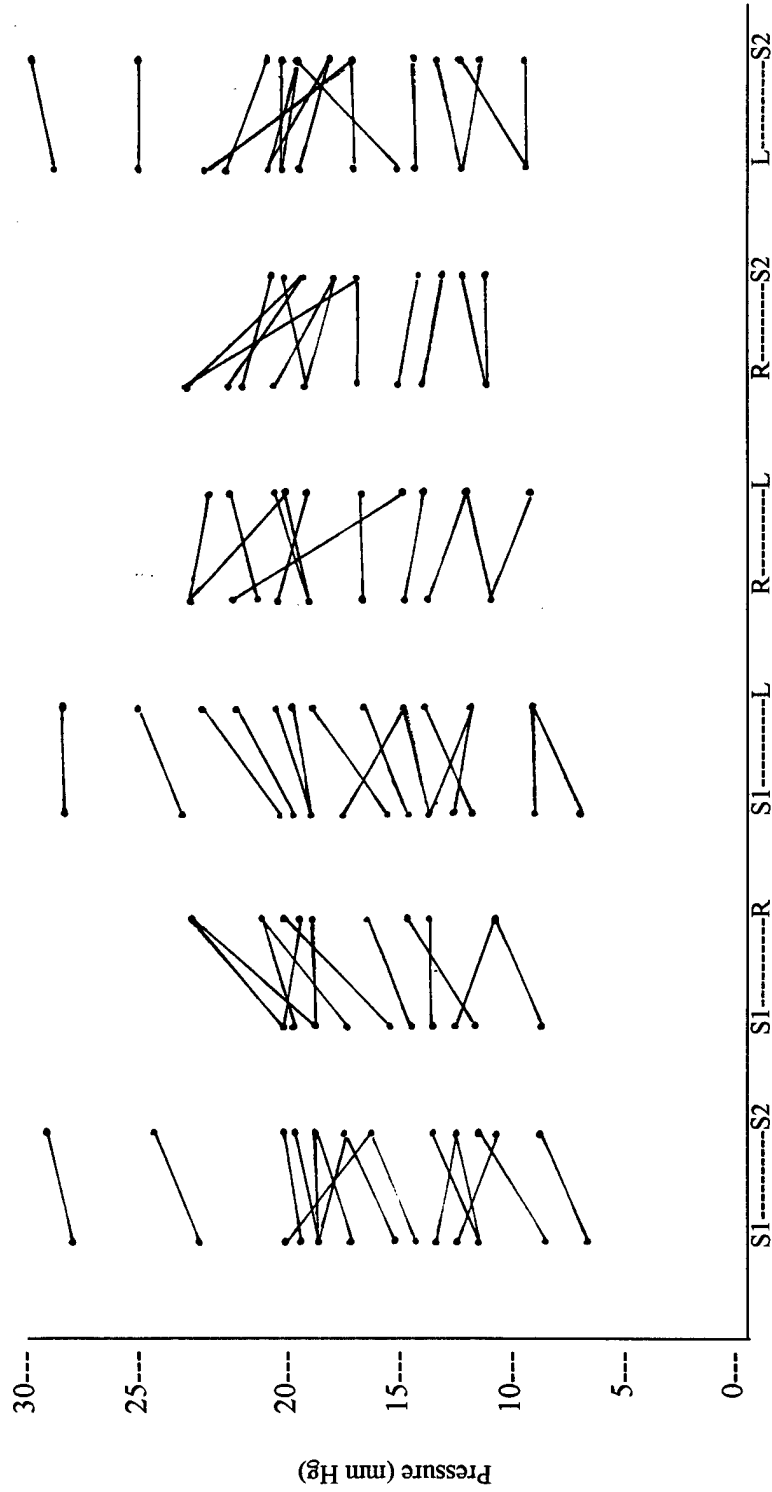


FIGURE 23. Summary of Individual PAW Pressure-Pair Differences. There were no significant ($p > .05$) differences between the pressure pairs.

position was between 3.3 mm Hg less than and 0.1 mm Hg greater than the PAW pressure in the Supine-1 position. In all cases, as will be discussed, the pressure differences reflected by the maximum absolute values in the 95% CIs were not clinically significant.

Individual Patient Data

Data regarding baseline fluctuation were collected to aid in the interpretation of position-related changes, that is, were the observed changes in pressure associated with the various positions greater than those that can be explained by expected pressure fluctuation? Of concern was that if the degree of fluctuation was dependent on absolute pressure values, would the increase in pressure observed between the baseline and Supine-1 positions affect the degree of fluctuation.

For all pressures (PAS, PAED, PAM, and PAW) there was an increase in the measured pressure between the baseline fluctuation period and the mean of the supine pressures. In order to determine if there was a difference in fluctuation between the two periods of the study, the pressure fluctuation observed during the first portion of the study was compared to the fluctuation observed during the positioning portion of the study using a paired t-test. For the PAS, PAED, and PAM pressures the data from the left-lateral position were omitted, as they reflected the experimental effect and not baseline fluctuation. In order to equalize the groups for comparison, the data from the M3 measurement (time = 10 minutes) were omitted from the baseline fluctuation. There were no statistically significant differences ($p > 0.5$) in the fluctuation observed during

the baseline period compared with the positioning portion of the study for PAS, PAED, and PAW pressures (Table 13). For example, the PAS pressure fluctuation observed during the position portion of the study after control for the experimental effect (i.e., removal of data related to 30-degree left lateral position) was not significantly different from the fluctuation observed during the baseline period ($p > .05$, 95% CI -0.6, 0.8). Based on the 95% confidence interval, on average the mean PAS pressure fluctuation in the position portion of the study was between -0.6 mm Hg less and 0.8 mm Hg greater than fluctuation during the baseline period.

For the PAM pressures, data from both the right and left lateral positions were omitted as they reflected the experimental effect. There was a statistically significant difference for the PAM pressure ($p = .018$). However, the mean difference of 0.6 ± 1.6 mm Hg (95% CI = 0.1, 1.2), while indicating that on average the PAM pressure changes during the position portion of the study were greater than those that occurred during the baseline period, the differences were not clinically significant. Therefore, the baseline fluctuation data and indices of clinical significance were applicable to the position portion of the study.

Agreement Analysis

The individual data were analyzed using agreement analysis (Bland & Altman, 1986; Bland & Altman, 1995). Three pressure pairs were analyzed: Supine – Left, Supine – Right, and Right-Left. Because the pre and post measurements (Supine-1 and Supine-2) were not significantly different ($p > .05$) a mean of the supine pressures was used in the

TABLE 13. Difference in Position versus Baseline Pressure Fluctuation

Pressure (Position- Baseline)	Mean Difference	SD	95% Confidence Interval		t	df	Sig (2-tailed)
			Lower	Upper			
PAS	0.1	2.2	-0.6	0.8	.372	38	.712
PAED	0.1	2.1	-0.6	0.8	.309	37	.759
PAM	0.6	1.6	0.1	1.2	2.480	38	.018*
PAW	-0.7	2.3	-1.4	0.1	-1.821	38	.077

*p < .05

analysis. For each pressure pair the normality of distribution of the differences and any potential relation between the difference and the mean (i.e., increased pressure difference with increased mean pressure) were analyzed.

Pulmonary Artery Systolic Pressure

Pulmonary artery systolic supine – left.

The differences between PAS Supine and Left are summarized in Figure 24. The differences were normally distributed and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The mean difference was -1.9 mm Hg, and the limits of agreement for the PAS Supine-Left were $(-5.9, 2.0)$; that is, we can say with 95% assurance, that on average the pressure differences would be expected to lie between -5.9 mm Hg and 2.0 mm Hg. A clinically significant fluctuation for PAS was defined as a pressure change of greater than 5 mm Hg, and as demonstrated in Figure 24 only 3 of 32 subjects (9%) exceeded 5 mm Hg. Because, with the exception of the three subjects, the position-related pressure differences were not considered clinically important, the PAS measurements obtained in the supine and 30-degree left lateral position could be used interchangeably. However, as the limits of agreement exceeded the values specified as clinically significant, individual response to position needed to be assessed.

The precision of the bias between the two measurements was described by the mean difference (bias = -1.9 mm Hg) and the 95% confidence interval of the bias (95% CI =

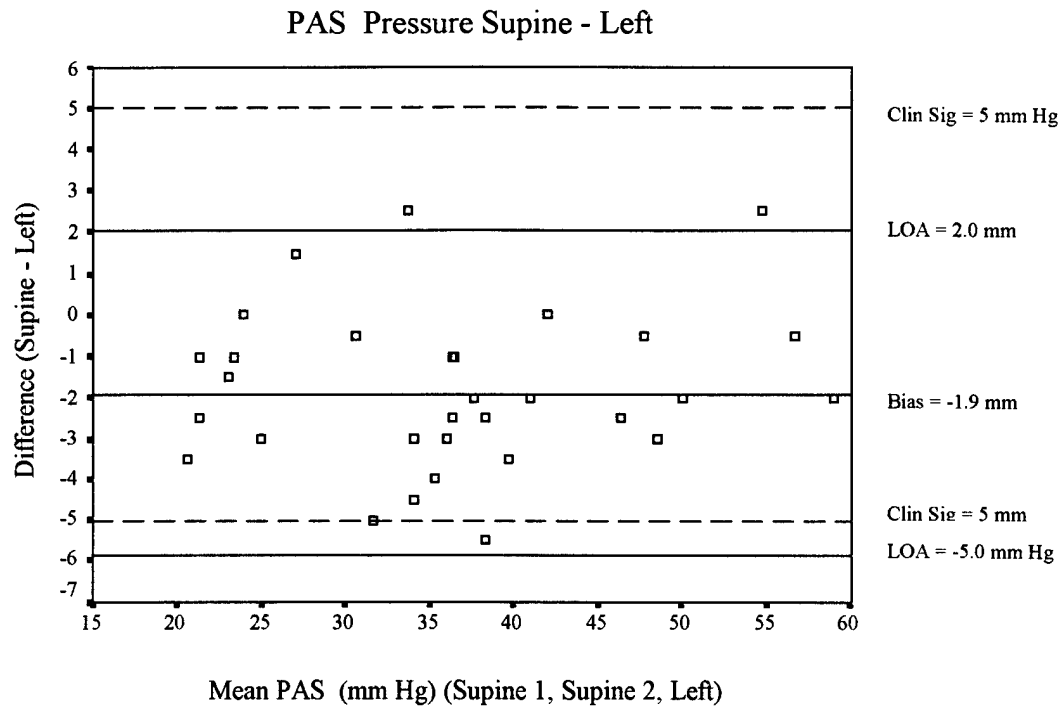


FIGURE 24. Agreement Analysis: PAS Supine – PAS Left. The bias was -1.9 mm Hg and the precision was -1.9 ± 0.7 mm Hg (-2.63 mm Hg, -1.2 mm Hg). The LOA (mean ± 2 SD) were 2.0 mm Hg and -5.8 mm Hg. However, the LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus the measurements are interchangeable; however, individual response to position must be assessed.

-2.6, -1.2). Thus, we can say with 95% confidence, on average, the Left PAS pressures were greater than the Supine PAS pressures by 1.2 to 2.6 mm Hg, a difference that was not clinically significant. The latter findings further support the interchangeable nature of the supine and left-lateral measurements.

Pulmonary artery systolic supine – right.

The differences between PAS Supine and Right are summarized in Figure 25. The differences were normally distributed and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -1.0 mm Hg and the limits of agreement for the PAS Supine-Right were $(-5.7, 3.8)$; that is, we can say with 95% assurance that on average the pressure differences would be expected to lie between -5.7 mm Hg and 3.8 mm Hg. The expected fluctuation for PAS was 5 mm Hg, and as demonstrated in Figure 25 only 2 of 32 subjects (6%) exceeded 5 mm Hg. Because, with the exception of the two subjects, the position-related pressure differences were not considered clinically important, the PAS measurements obtained in the supine and 30-degree left lateral position could be used interchangeably. However, as the limits of agreement exceeded the values specified as clinically significant, individual response to position needed to be assessed.

The precision of the bias was -1.0 mm Hg ± 0.9 mm Hg, (95% CI = $-1.8, -0.1$). Thus, with regard to the individual measurements, we can say with 95% confidence that for

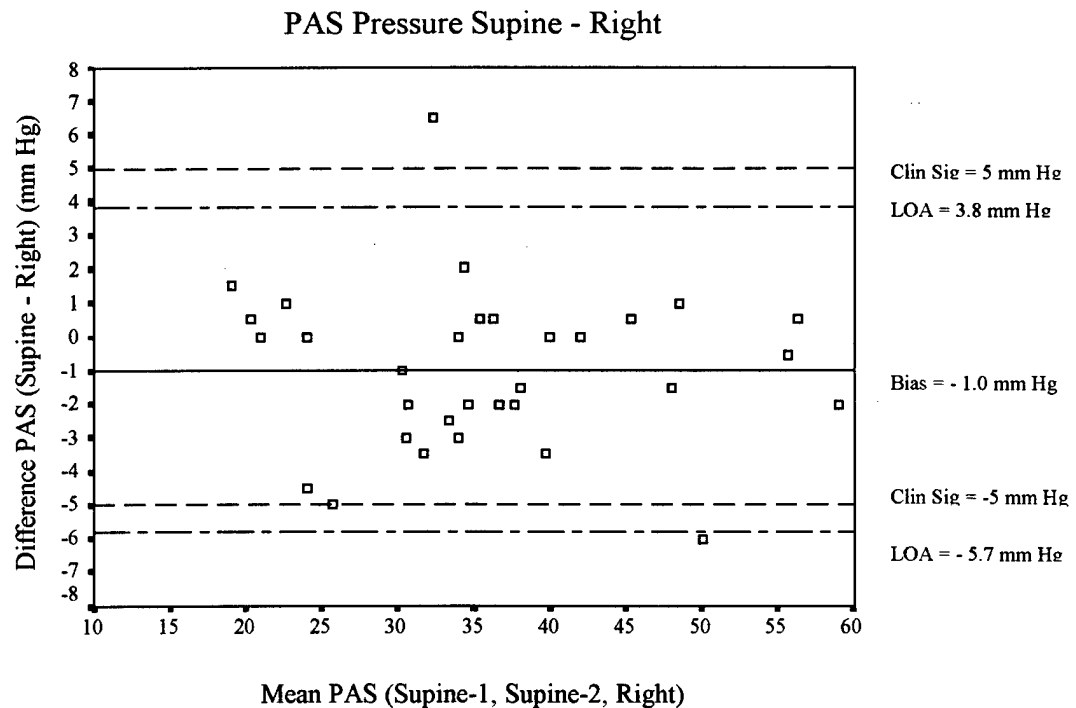


FIGURE 25. Agreement Analysis: PAS Supine – PAS Right. The bias was -1.0 mm Hg and the precision was -1.0 ± 0.9 mm Hg (-1.8 mm Hg, -0.1 mm Hg). The LOA (mean ± 2 SD) were 3.8 mm Hg and -5.7 mm Hg. However, the LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus, the measurements are interchangeable; however, individual response to position must be assessed.

individual measurements, on average, the Right PAS pressure was 0.1 to 1.8 mm Hg greater than the Supine PAS, which was not a clinically significant difference.

Pulmonary artery systolic pressure: right – left.

The differences between PAS Right and Left are summarized in Figure 26. The differences were normally distributed and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -1.17 mm Hg, and the limits of agreement for the PAS Right - Left were (-6.7, 4.4); that is, we can say with 95% assurance that, on average, the pressure differences would be expected to lie between -6.7 mm Hg and 4.4 mm Hg. The expected fluctuation for PAS was 5 mm Hg, and as demonstrated in Figure 26 only 1 of 32 subjects (3%) exceeded 5 mm Hg. Because, with the exception of the one subject, the position-related pressure differences were not considered clinically important, the PAS measurements obtained in the right and left 30-degree lateral position could be used interchangeably. However, as the LOA exceeded the values specified as clinically significant, individual response to position needs to be assessed.

The precision of the bias was -1.2 mm Hg $\pm$ 1.0 mm Hg (95% CI = -2.2, -0.1). Thus, we can say with 95% confidence that for the individual data, on average, Left PAS measurements were greater than the Right PAS measurements by 0.1 to 2.2 mm Hg, which was not a clinically significant difference.

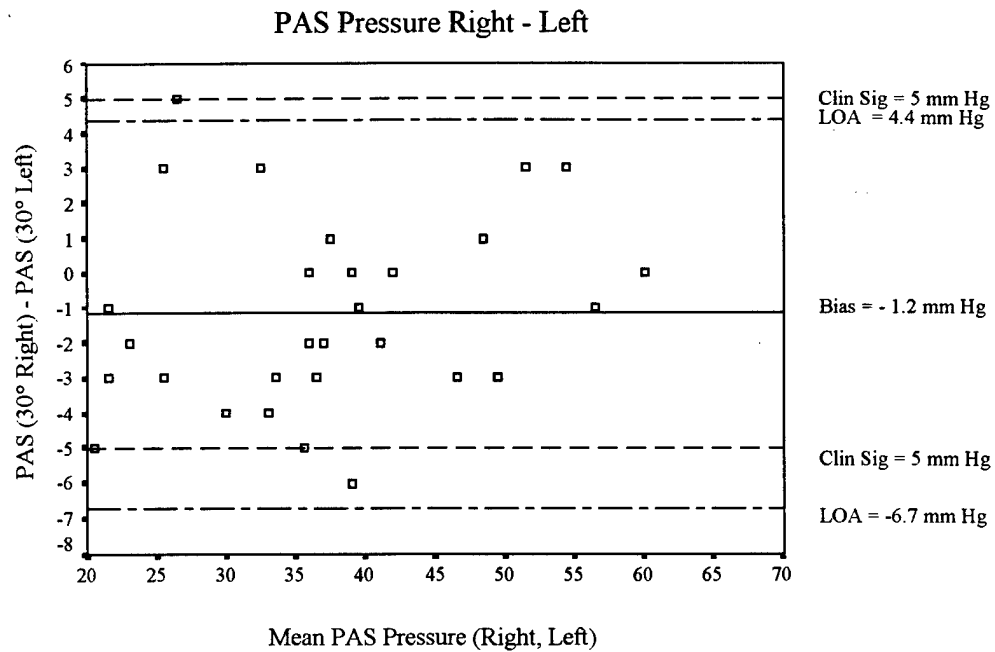


FIGURE 26. Agreement Analysis: PAS Right – PAS Left. The bias was -1.2 mm Hg and the precision was -1.2 ± 1.9 mm Hg (-2.2 mm Hg, -0.1 mm Hg). The LOA (mean ± 2 SD) were 4.4 mm Hg and -6.7 mm Hg. The LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus, the measurements are interchangeable. However, individual response to position must be assessed.

Individual Position-Related PAS Pressure Changes Versus Individual Baseline

Fluctuation

For each individual, the change in pressure observed during the position portion of the study was compared to the baseline fluctuation for the individual. These data are summarized in Figure 27. As can be seen in Figure 27, 16 of 34 subjects (47%) exceeded baseline fluctuation, but only four of these individuals (#5, #6, #8, #24), who are described below, had pressure changes that were clinically significant.

As can be seen in Figure 27, there was no relationship between the subjects whose pressure changes during position exceeded baseline and the occurrence of a clinically significant pressure change. Of the subjects who demonstrated clinically significant changes, none of them had baseline fluctuations that were considered clinically significant. In contrast, only one subject (#1) demonstrated a clinically significant baseline fluctuation (baseline fluctuation = 6 mm Hg). This subject had a maximum position-related change of 5 mm Hg, however, this change was the result of mixed effect (Supine-1-Right decreased 2 mm Hg, Supine-1-Left increased 3 mm Hg, only be invoked if the pre-post measurements are dissimilar.

Pulmonary Artery End Diastolic Pressure

Pulmonary artery end-diastolic pressure: supine – left.

The differences between PAED Supine and Left are summarized in Figure 28. The differences were normally distributed and there was no relation between the difference

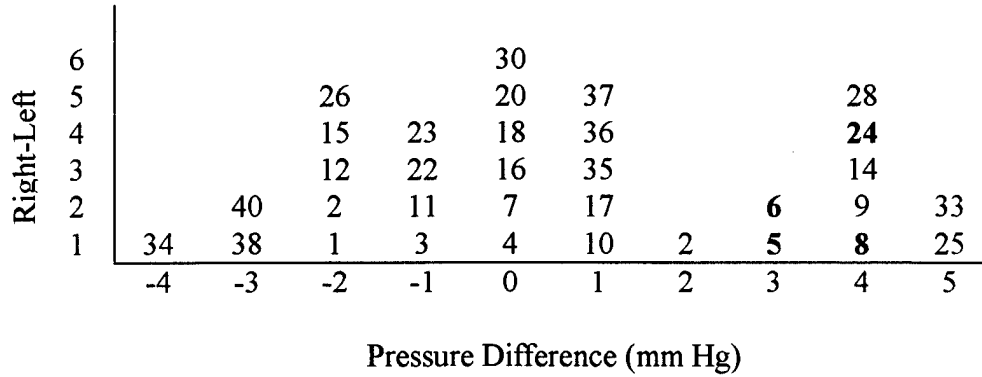


FIGURE 27. Pulmonary Artery Systolic Pressure: Difference between maximum position-related pressure change minus baseline pressure fluctuation for each subject. The pressure difference is plotted on the X-axis. The number of subjects demonstrating a particular pressure change relative to baseline fluctuation is on the Y-axis. The numbers on the graph are the subject code numbers. The bold numbers reflect subjects who demonstrated a clinically significant pressure change. For example, subject # 5 had a 5 mm Hg baseline pressure fluctuation and an 8 mm Hg position-related pressure change. In contrast, Subject #25 had no baseline fluctuation and a 5 mm Hg position-related pressure change.

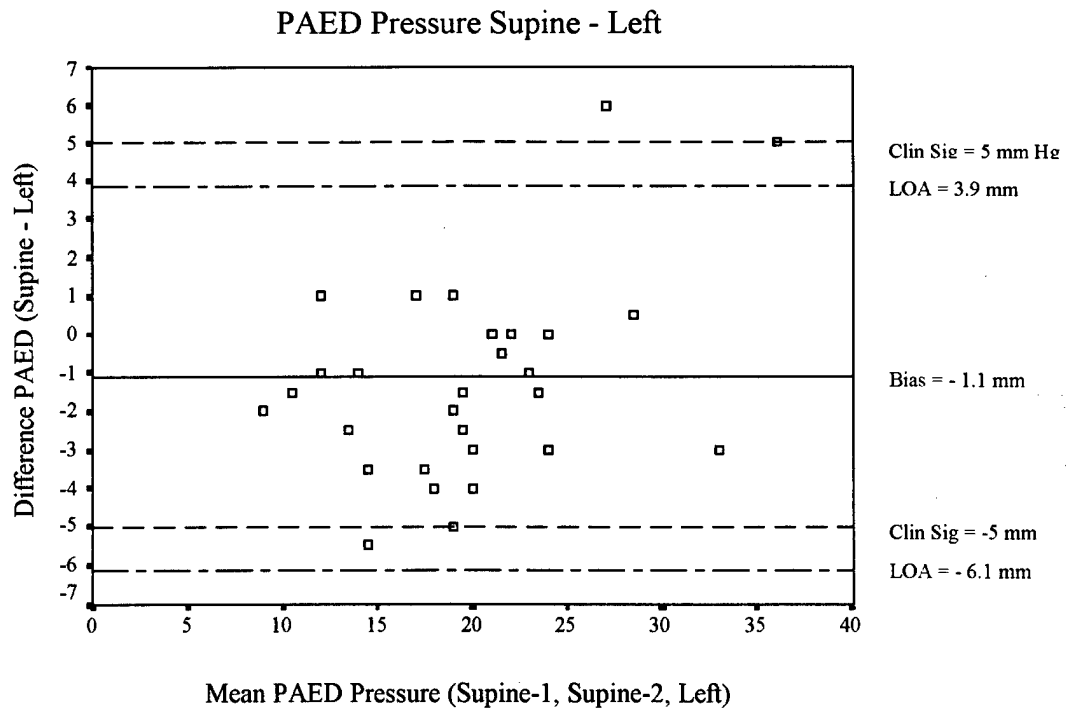


FIGURE 28. Agreement Analysis: PAED Pressure Supine – PAED Pressure Left. The bias was -1.1 mm Hg and the precision was -1.1 ± 0.6 mm Hg (-1.7 mm Hg, -0.6 mm Hg). The LOA (mean ± 2 SD) were 3.9 mm Hg and -6.1 mm Hg. The LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus, the measurements are interchangeable; however, individual response to position must be assessed.

and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -1.1 mm Hg and the limits of agreement for the PAED Supine-Left were $(-6.1, 3.9)$; that is, we can say with 95% assurance that on average the pressure differences would be expected to lie between -6.1 mm Hg and 3.9 mm Hg. The expected fluctuation for PAED was 5 mm Hg, and as demonstrated in Figure 28 only 3 of 32 subjects (9%) exceeded 5 mm Hg. With the exception of the three subjects (who are described below), the position-related pressure differences were not considered clinically important; thus, the PAED pressure measurements obtained in the supine and 30-degree left lateral position could be used interchangeably. However, as the 95% limits of agreement exceeded the values specified as clinically significant, individual response to position needs to be assessed.

The precision of the bias was -1.1 mm Hg $\pm$ 0.6 mm Hg (95% CI = $-1.7, -0.5$). Thus, we can say with 95% confidence that on average the Left PAS was greater than Supine PAED pressure measurements by 0.5 to 1.7 mm Hg, which was not a clinically significant difference.

Pulmonary artery end-diastolic pressure supine – right.

The differences between PAED Supine and Right are summarized in Figure 29. The differences were normally distributed and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie

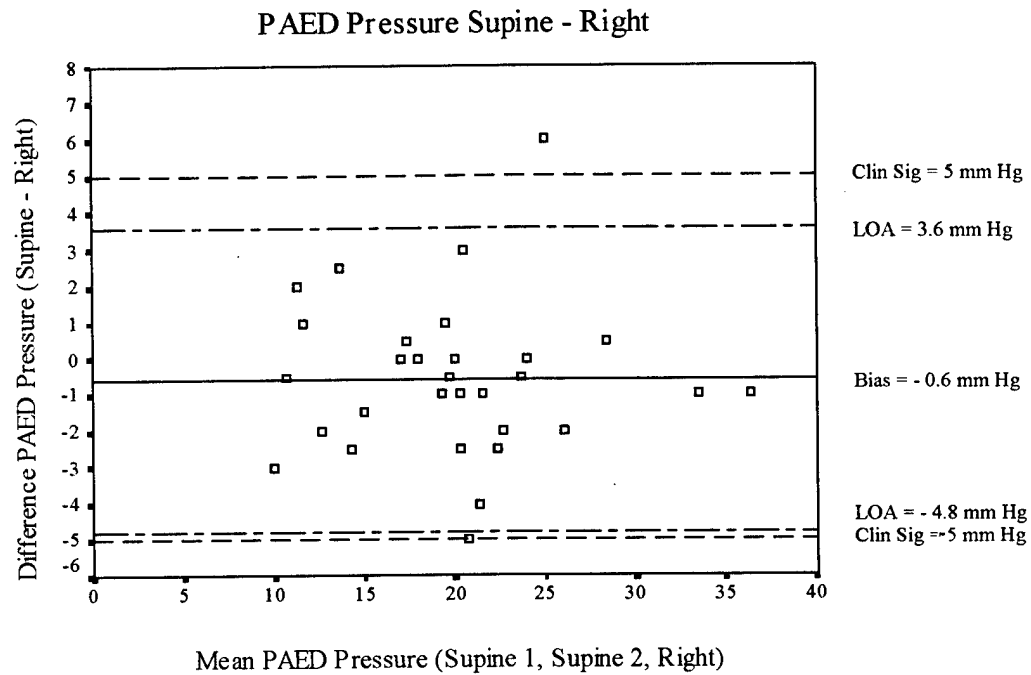


FIGURE 29. Agreement Analysis: PAED Pressure Supine – PAED Pressure Right. The bias was -0.6 mm Hg and the precision was -0.6 ± 0.8 mm Hg (-1.3 mm Hg, 0.2 mm Hg). The LOA (mean ± 2 SD) were 3.6 mm Hg and -4.8 mm Hg. The LOA did not exceed the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change; thus, the measurements are interchangeable.

within two standard deviations of the mean. The bias was -0.6 mm Hg and the limits of agreement for the PAED Supine-Right were $(-4.8, 3.6)$; that is, we can say with 95% assurance that on average the pressure differences would be expected to lie between -4.8 mm Hg and 3.6 mm Hg. The expected fluctuation for PAED was 5 mm Hg, and as demonstrated in Figure 29 only 2 of 32 subjects (6%) exceeded 5 mm Hg. With the exception of the two subjects (who are described below), the position-related pressure differences were not clinically significant, thus, the PAED measurements obtained in the supine and 30-degree right lateral position could be used interchangeably. It is important to note that the limits of agreement did not exceed the values specified as clinically significant, thus providing additional support for the interchangeable nature of the measurements obtained in the supine and right-lateral positions.

The precision of the bias was -0.6 mm Hg ± 0.8 mm Hg (95% CI = $-1.3, 0.2$). Thus, we can say with 95% confidence that for the individual data, on average, the Right PAS was between 1.3 mm Hg less than and 0.2 mm Hg greater than Supine PAED measurements, which was not a clinically significant difference.

Pulmonary artery end-diastolic pressure: right – left.

The differences between PAED 30-degree right and left pressures are summarized in Figure 30. The differences were normally distributed with the exception of one outlier, and there was no relation between the difference and the mean. The bias was -0.6 mm Hg and the limits of agreement for the PAS Supine-Left were $(-6.1, 5.0)$ mm Hg; that is, we can say with 95% confidence that on average the pressure differences would be

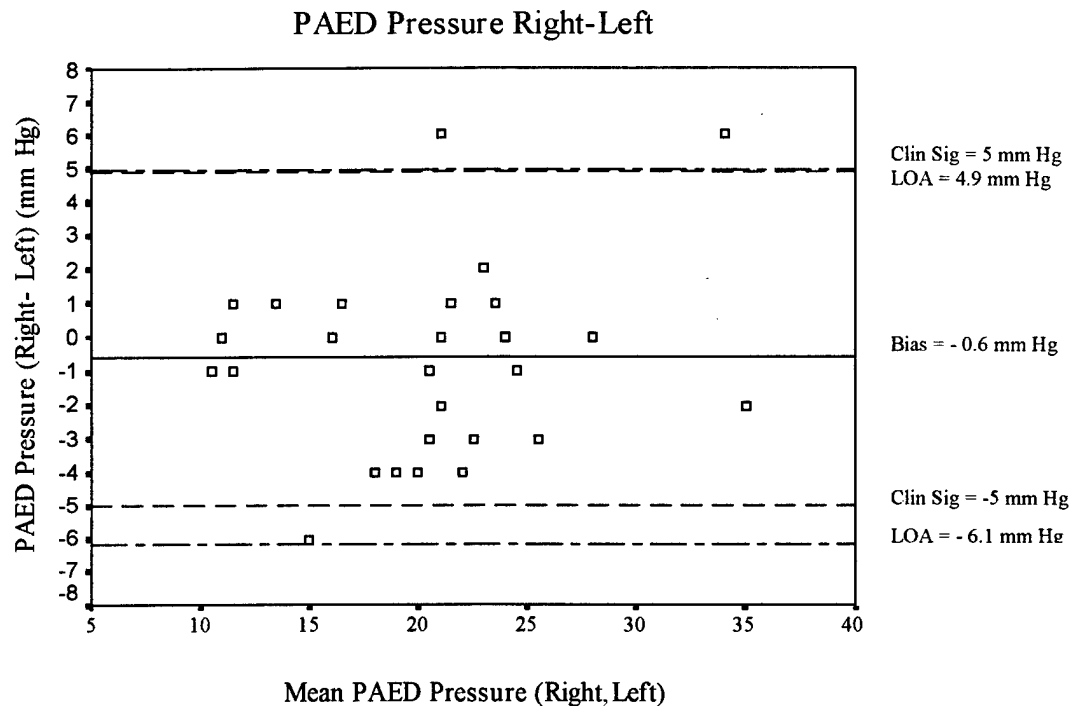


FIGURE 30. Agreement Analysis: PAED Pressure Right – PAED Pressure Left. The bias was -0.6 mm Hg and the precision was -0.6 ± 1.0 mm Hg (-1.6 mm Hg, -0.5 mm Hg). The LOA (mean ± 2 SD) were 4.9 mm Hg and -6.1 mm Hg. The LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus, the measurements are interchangeable. However, individual response to position must be assessed.

expected to lie between -6.1 mm Hg and 5.0 mm Hg. A PAED pressure change of greater than 5 mm Hg was described as clinically significant, and as demonstrated in Figure 30, 3 of 29 subjects (10%) exceeded 5 mm Hg. With the exception of the three subjects (who are described below), the position-related pressure differences were not considered clinically important; therefore, the PAED measurements obtained in the supine and 30-degree left lateral position can be used interchangeably, although individual responses need to be assessed.

The precision of the bias was $-0.6 \text{ mm Hg} \pm 1.0 \text{ mm Hg}$, 95% CI = -1.6, 0.5. Thus, we can say with 95% confidence, that for the individual data, on average, the Left PAED was 0.5 mm Hg less than to 1.6 mm Hg greater than Right PAS measurements. This difference was clinically insignificant, and provides further support for the interchangeability of the PAED pressure measurements in the supine and 30-degree left lateral position.

Individual Position-Related PAED Pressure Changes Versus Individual Baseline Fluctuation

For each individual, the change in pressure observed during the position portion of the study was compared to the baseline fluctuation for the individual. These data are summarized in Figure 31. As can be seen in Figure 31, 19 of 32 subjects (58%) exceeded baseline fluctuation, but only five of these individuals (#4, #5, #11, #17, #28), who are described below, had pressure changes that were clinically significant. With the exception of subject #28, who demonstrated a pressure change that was 6 mm Hg greater

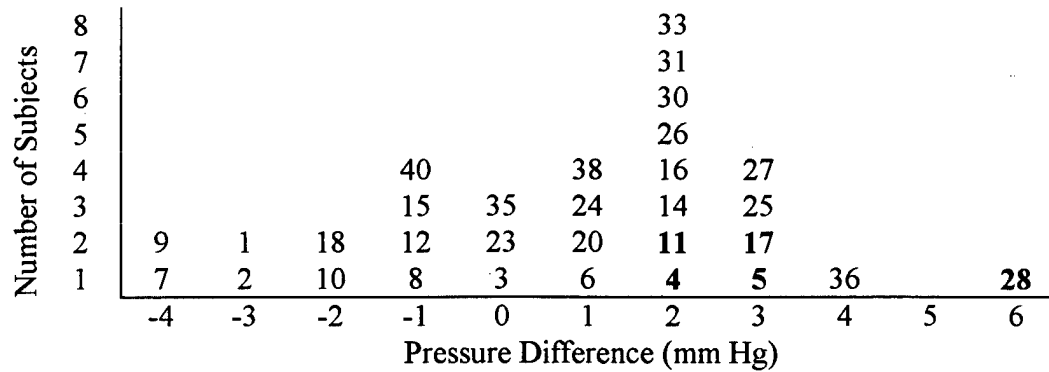


FIGURE 31. Pulmonary Artery End-Diastolic Pressure: Maximum Position-Related Pressure Change versus Baseline Pressure Fluctuation For Each Subject. The difference between the baseline fluctuation and the maximal pressure fluctuation observed during the position portion of the study are plotted on the Y-axis. The numbers are the subject code numbers. The bold numbers reflect subjects who demonstrated a clinically significant pressure change of greater than 5 mm Hg.

than baseline fluctuation, the pressure changes during the position portion of the study were only 2 to 3 mm Hg greater than baseline pressure fluctuation.

As can be seen in Figure 31, there was no relationship between the subjects whose pressure changes during position exceeded baseline and the occurrence of a clinically significant pressure change. Therefore, the degree of baseline fluctuation cannot be used as a predictor of which patients will demonstrate increased pressure changes in response to a specific position. In addition, citing the expected fluctuation as a cause of the position-related pressure change may only be invoked if the pre-post measurements are dissimilar.

Pulmonary Artery Mean Pressure

Pulmonary artery mean pressure: supine – left.

The differences between PAM Supine and Left are summarized in Figure 32. The differences were normally distributed, with the exception of one outlier, and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -1.3 mm Hg and the limits of agreement for the PAM Supine-Left were $(-1.3, 95\% \text{ CI} = -6.3, 3.7)$; that is, we can say with 95% assurance that on average the pressure differences would be expected to lie between -6.3 mm Hg and 3.7 mm Hg. The expected fluctuation for PAM was 5 mm Hg, and as demonstrated in Figure 32, 4 of 32 subjects

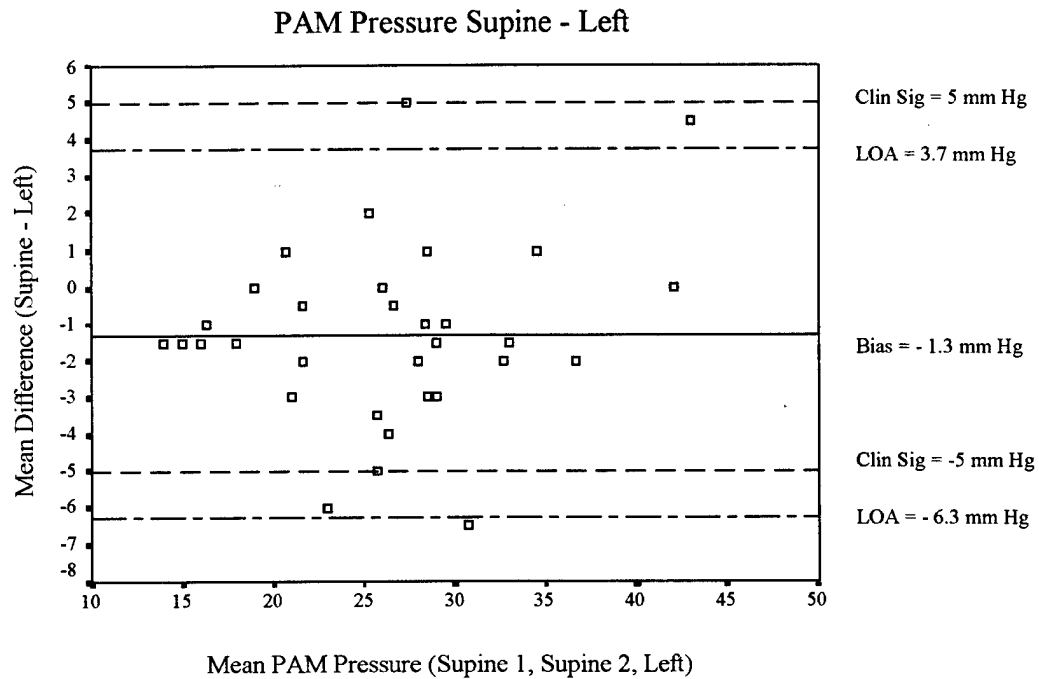


FIGURE 32. Agreement Analysis: PAM Pressure Supine – PAM Pressure Left. The bias was -1.3 mm Hg and the precision was -1.3 ± 0.9 mm Hg (-2.1 mm Hg, -0.4 mm Hg). The LOA (mean ± 2 SD) were 3.7 mm Hg and -6.3 mm Hg. The LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus, the measurements are interchangeable. However, individual response to position must be assessed.

(12.5%) exceeded 5 mm Hg. With the exception of the four subjects (who are described below), the position-related pressure differences were not considered clinically important, therefore, the PAM measurements obtained in the supine and 30-degree left lateral position could be used interchangeably. However, as the 95% limits of agreement exceeded the values specified as clinically significant, individual response to position needed to be assessed.

The precision of the bias was $-1.3 \text{ mm Hg} \pm 0.9 \text{ mm Hg}$ (95% CI = -2.2, -0.4). Thus, we can say with 95% confidence that on average the Left PAM was greater than Supine PAM measurements by 0.4 to 2.2 mm Hg, which was not a clinically significant difference. The small bias provides further support for the interchangeable nature of the measurements obtained in the supine and left lateral positions.

Pulmonary artery mean pressure: supine – right.

The differences between PAM Supine and Right are summarized in Figure 33. The differences were normally distributed, with the exception of one outlier, and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -0.7 mm Hg and the limits of agreement for the PAM Supine-Right were $(-0.7, 95\% \text{ CI} = -4.7, 3.2)$; that is, we can say with 95% assurance that on average the pressure differences would be expected to lie between -4.7 mm Hg and 3.2 mm Hg . A change in

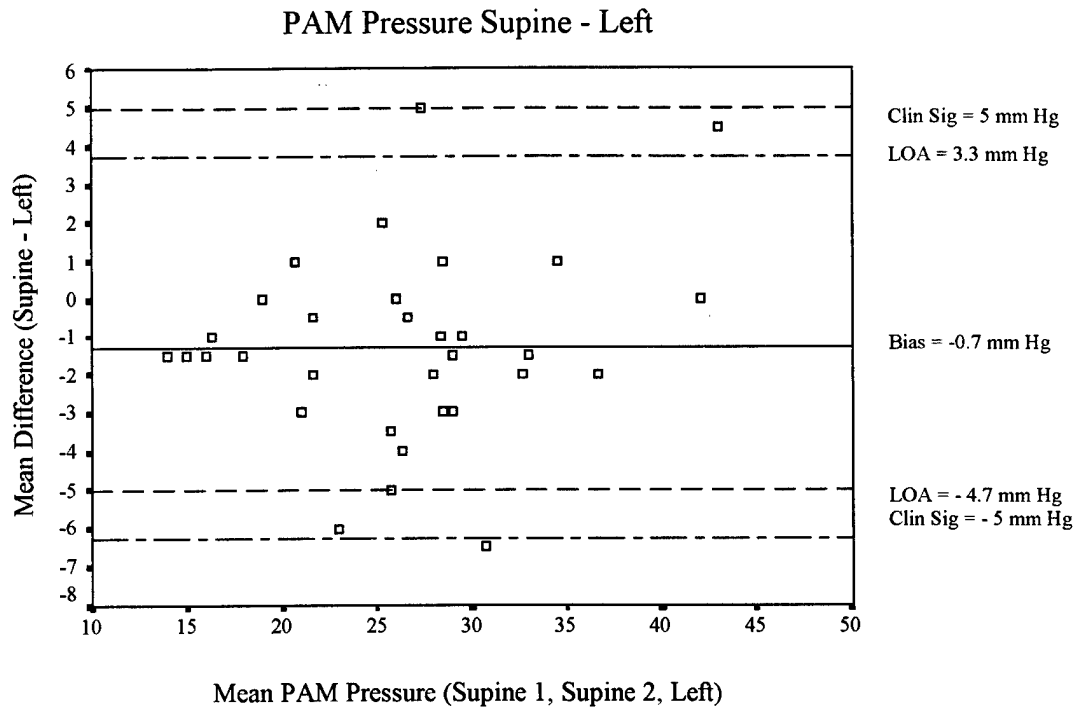


FIGURE 33. Agreement Analysis: PAM Pressure Supine – PAM Right. The bias was -0.7 mm Hg and the precision was -0.7 ± 0.7 mm Hg (-1.4 mm Hg, 0 mm Hg). The LOA (mean ± 2 SD) were 3.3 mm Hg and -4.7 mm Hg. The LOA did not exceed the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change; thus, the measurements are interchangeable.

PAM of greater than 5 mm Hg was defined as clinically significant, and as demonstrated in Figure 33, only 1 of 32 subjects (3%) exceeded 5 mm Hg. With the exception of the one subject (who is described below), the position-related pressure differences were not considered clinically important; thus, the PAM measurements obtained in the supine and 30-degree right lateral position may be used interchangeably. It is important to note that the limits of agreement did not exceed the values specified as clinically significant; providing additional support for the interchangeable nature of the measurements from the supine and right-lateral positions.

The precision of the bias was $-0.7 \text{ mm Hg} \pm 0.7 \text{ mm Hg}$ (95% CI = -1.4, -0.02). Thus, we can say with 95% confidence that for the individual data, on average, the Right PAM was from 1.4 mm Hg to 0.02 mm Hg less than the Supine PAS measurements, which was not a clinically significant difference. The extremely small difference between the two measurements further supports the interchangeable nature of the measurements from the supine and right-lateral positions.

Pulmonary artery mean pressure right – left.

The differences between the PAM pressures in the 30-degree right and left positions are summarized in Figure 34. The differences were normally distributed, with the exception of one outlier, and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -0.5 mm Hg and the limits of agreement

for the PAM Pressure Supine-Left were (-6.1, 5.2 mm Hg); that is, we can say with 95% confidence that on average the pressure differences would be expected to lie between -6.1 mm Hg and 5.2 mm Hg. The expected fluctuation for PAM was 5 mm Hg, and as demonstrated in Figure 34, 2 of 29 subjects (7%) exceeded 5 mm Hg. With the exception of the two subjects (who are described below), the position-related pressure differences were not clinically significant, thus the PAM pressure measurements obtained in the supine and 30-degree left lateral position may be used interchangeable. However, as indicated by the limits of agreement, which exceeded the values specified as clinically significant, individual responses needed to be assessed.

The precision of the bias was $-0.5 \text{ mm Hg} \pm 1.1 \text{ mm Hg}$ (95% CI = -1.5, 0.6). Thus, we can say with 95% confidence that for the individual data, on average, the Left PAM was 0.5mm Hg less than to 1.5 mm Hg greater than Right PAM measurements, which was not a clinically significant difference. The small bias provides further support for the interchangeable nature of the measurements obtained in supine and left-lateral positions.

Individual Position-Related PAM Pressure Changes Versus Individual Baseline Fluctuation

For each individual, the change in pressure observed during the position portion of the study was compared to the baseline fluctuation for the individual. These data are summarized in Figure 35. As can be seen in Figure 35, 19 of 33 subjects (58%) exceeded baseline fluctuation, but only four (12%) of these subjects (#4, #5, #24, #28), who are

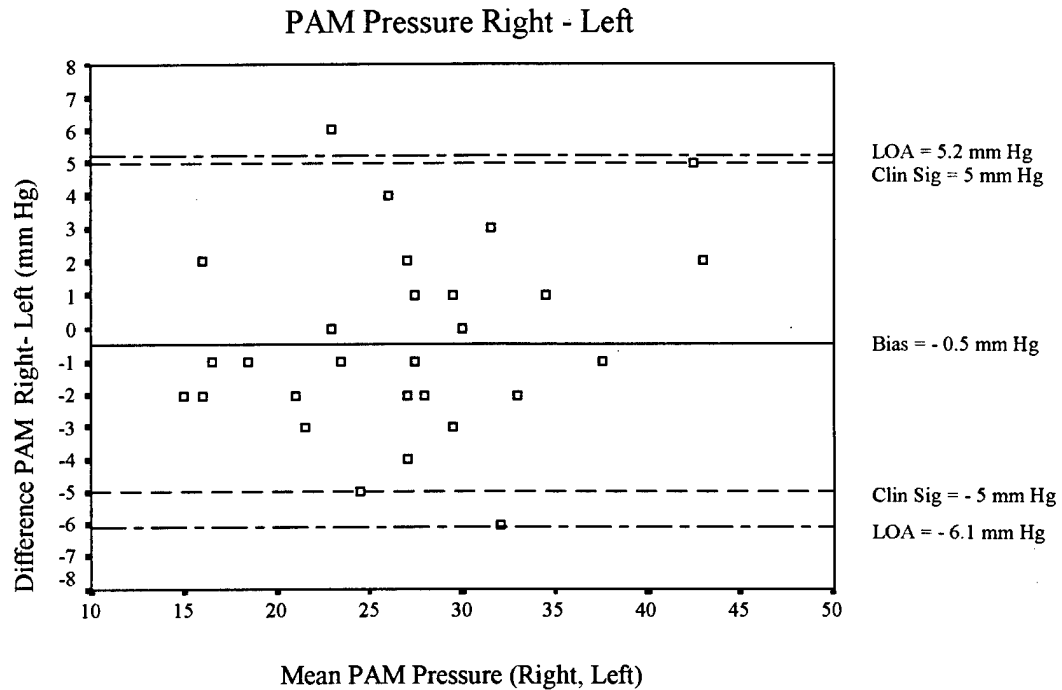


FIGURE 34. Agreement Analysis: PAM Pressure Right – PAM Pressure Left. The bias was -0.5 mm Hg and the precision was -0.5 ± 1.1 mm Hg (-1.5 mm Hg, -0.6 mm Hg). The LOA (mean ± 2 SD) were 5.2 mm Hg and -6.1 mm Hg. The LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus, the measurements are interchangeable. However, individual response to position must be assessed.

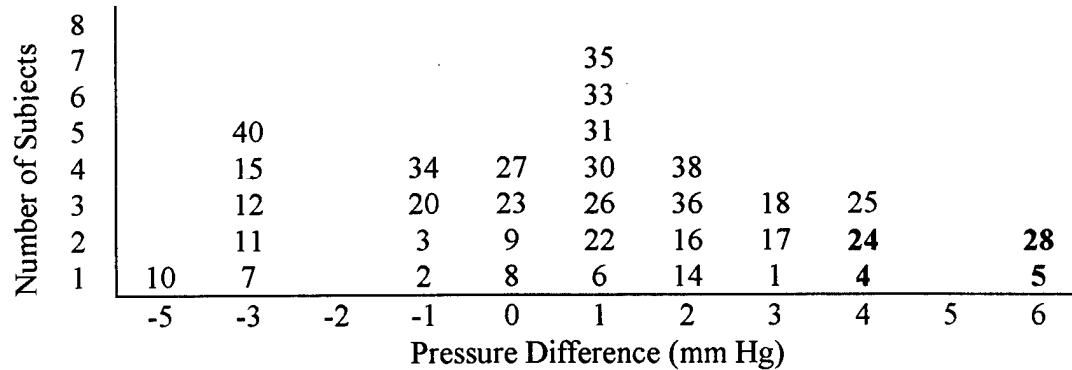


FIGURE 35. Pulmonary Artery Mean Pressure: Difference between the maximum position-related pressure change and baseline fluctuation for each individual. The difference between the baseline fluctuation and the maximal pressure fluctuation observed during the position portion of the study is plotted on the X-axis. The numbers on the graph are the subject code numbers. The bold numbers reflect subjects who demonstrated a clinically significant pressure change. Subject #10 had a 5 mm Hg difference between the baseline fluctuation (7 mm Hg) and the maximum position-related change (2 mm Hg); thus, the difference was not associated with a clinically significant position-related pressure change. In contrast, Subject #24 demonstrated a 3 mm Hg baseline fluctuation and a 7 mm Hg position-related pressure change. described below, had pressure changes that were clinically significant.

As can be seen in Figure 35, there was a trend toward an increased number of subjects with pressure changes greater than baseline who also exhibited clinically significant pressure changes during the position portion of the study. However, none of these subjects who had clinically significant changes during the position portion had clinically significant changes during baseline. Therefore, the degree of baseline fluctuation was not a useful predictor of which patients would demonstrate increased position-related pressure changes. In addition, citing the expected fluctuation as a cause of the position-related pressure change was only invoked if the pre-post measurements were dissimilar.

Pulmonary Artery Wedge Pressure

Pulmonary artery wedge pressure: supine – left.

The differences between PAW Pressure Supine and Left are summarized in Figure 36. The differences were normally distributed, and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -0.5 mm Hg and the limits of agreement for the PAW Pressure Supine-Left were $(-3.8, 3.3)$; that is, we can say with 95% assurance that on average the pressure differences would be expected to lie between -3.8 mm Hg and 3.3 mm Hg. A PAW pressure change of greater than 4 mm Hg was indicative of a clinically significant change, and as demonstrated in Figure 36 only one subject exceeded 4 mm Hg. Therefore, the PAW pressure

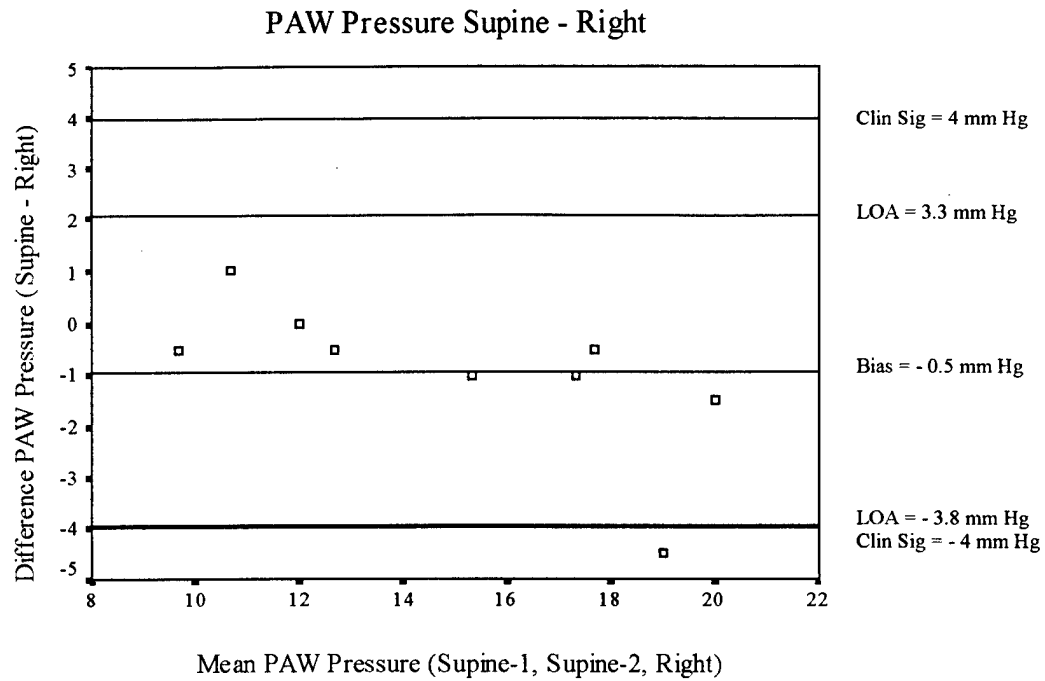


FIGURE 36. Agreement Analysis: PAW Pressure Supine – PAW Pressure Left. The bias was -0.5 mm Hg and the precision was -0.5 ± 0.9 mm Hg (-1.4 mm Hg, -0.4 mm Hg). The LOA (mean ± 2 SD) were 3.3 mm Hg and -3.8 mm Hg. The LOA did not exceed the 4 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change; thus, the measurements are interchangeable.

measurements obtained in the supine and 30-degree left lateral position are interchangeable.

The precision of the bias was $-0.5 \text{ mm Hg} \pm 0.9$ (95% CI = -1.4, -0.4). Thus, we can say with 95% confidence that on average the Left PAW pressure was greater than Supine PAW pressure by 0.4 to 1.4 mm Hg. The small difference provides further support for the interchangeable nature of the measurements obtained in the supine and 30-degree left-lateral position.

Pulmonary artery wedge pressure: supine – right.

The differences between PAW Supine and Right are summarized in Figure 37. Subject # 27 was excluded from this analysis, as pressures in the right position were affected by suctioning. The differences were normally distributed and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -0.94 mm Hg and the limits of agreement for the PAW Supine-Right were $(-4.0, 2.1)$; that is, we can say with 95% assurance that on average the pressure differences would be expected to lie between -4.0 mm Hg and 2.1 mm Hg . The expected fluctuation for PAW was 4 mm Hg , and as demonstrated in Figure 37, only one of nine subjects (11%) exceeded 4 mm Hg . Because, with the exception of the one subject (who is described below), the position-related pressure differences were not considered clinically

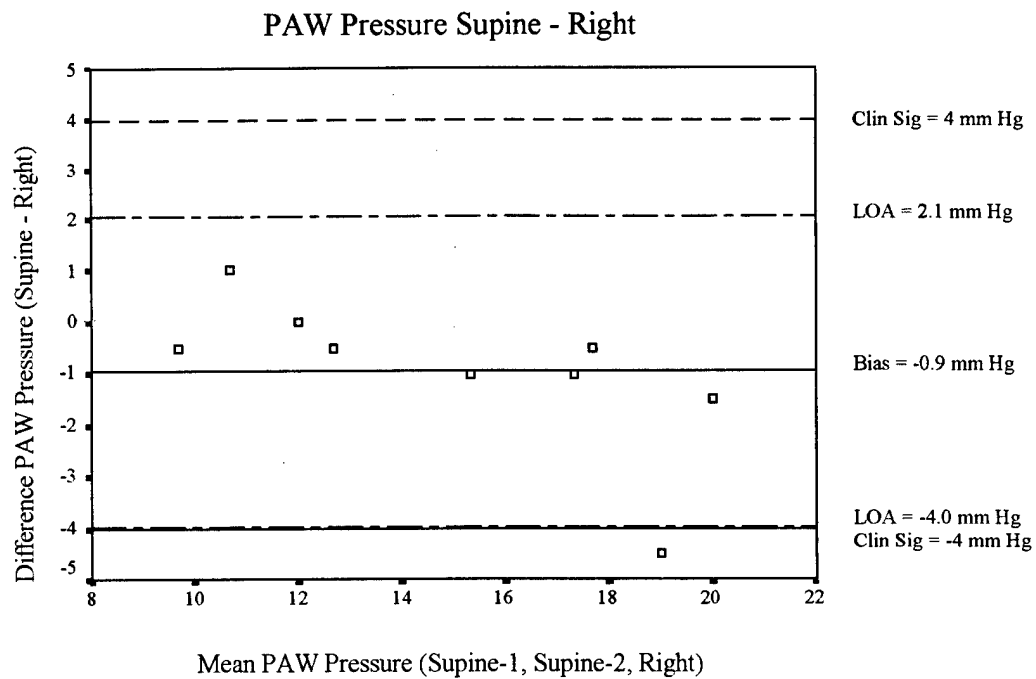


FIGURE 37. Agreement Analysis: PAW Pressure Supine – PAW Pressure Right. The bias was -0.94 mm Hg and the precision was -0.9 ± 1.2 mm Hg (0.21 mm Hg, -2.1 mm Hg). The LOA (mean $\pm$ 2 SD) were from 2.1 mm Hg to -4.0 mm Hg. We can say with 95% assurance, that on average the mean differences would lie between the LOA. The LOA are narrow, and do not exceed the 4mm Hg pressure change that was specified as the level of a clinically significant (clin sig) change; thus, the two measurements are in agreement and may be used interchangeably.

important, the PAED measurements obtained in the supine and 30-degree right lateral position could be used interchangeably.

The precision of the bias was $-0.94 \text{ mm Hg} \pm 1.2 \text{ mm Hg}$ (95% CI = -2.1, 0.21). Thus, we can say with 95% confidence that for the individual data, on average, the Right PAW was between 0.21 mm Hg less than and 2.1 mm Hg greater than Supine PAW measurements, which was not a clinically significant difference.

Pulmonary artery wedge pressure: right – left.

The differences between PAW 30-degree right and left pressures are summarized in Figure 38. Subject # 27 was eliminated from the analysis as the pressure changes observed in the right position were related to suctioning. The differences were normally distributed, with one outlier, and there was no relation between the difference and the mean. As the differences were normally distributed and were independent of the magnitude of the measurement, it was expected that 95% of the differences would lie within two standard deviations of the mean. The bias was -0.78 mm Hg and limits of agreement for the PAW Supine-Left were $(-5.3, 6.8 \text{ mm Hg})$; that is, we can say with 95% confidence that on average the pressure differences would be expected to lie between -5.3 mm Hg and 6.8 mm Hg . The expected fluctuation for PAW was 4 mm Hg , and as demonstrated in Figure 38 one of nine subjects (11%) exceeded 4 mm Hg .

The precision of the bias was $0.78 \text{ mm Hg} \pm 2.3 \text{ mm Hg}$ (95% CI = -1.6, 3.2). Thus, we can say with 95% confidence that for the individual data, on average, the Left PAW

PAW Pressure Right-Left

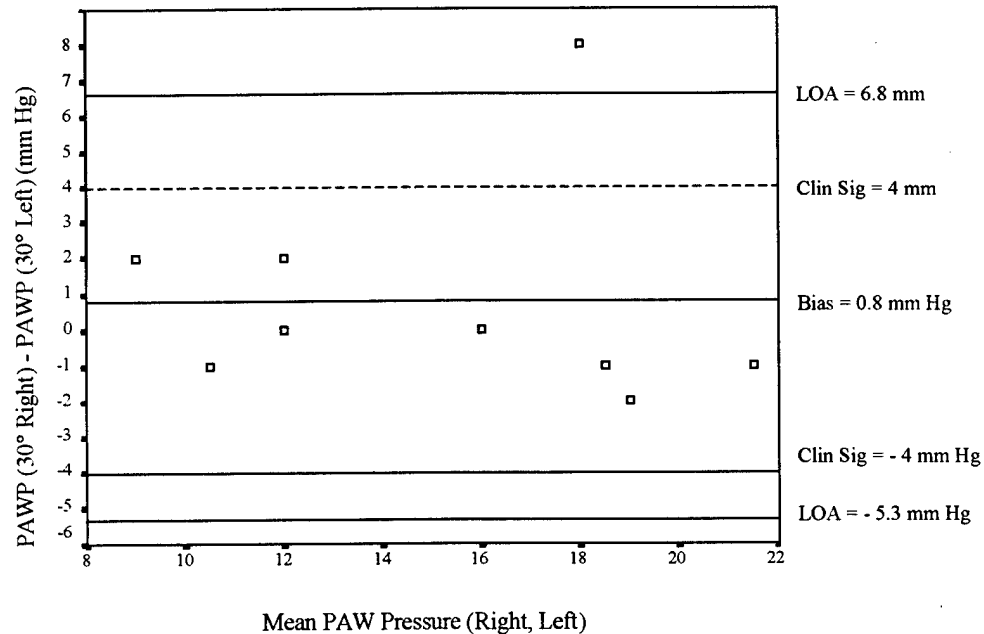


FIGURE 38. Agreement Analysis: PAW Pressure Right – PAW Pressure Left. The bias was 0.8 mm Hg and the precision was 0.8 ± 2.3 mm Hg (-1.6 mm Hg, 3.2 mm Hg). The LOA (mean ± 2 SD) were 6.8 mm Hg and -5.3 mm Hg. The wide LOA was reflective of the small sample size and the effect of an outlier. The LOA exceeded the 5 mm Hg pressure change that was specified as indicative of a clinically significant (clin sig) change. We can say with 95% assurance that on average the mean differences would lie between the LOA; thus, the measurements are interchangeable. However, individual response to position must be assessed.

was 1.6 mm Hg less than to 3.2 mm Hg greater than Right PAW measurements, which is a clinically insignificant difference. The results of this analysis must be viewed with caution due to the small sample size ($n = 9$) and the marked effect of the outlier on the variability of the measurements (Subjects #28: 8 mm Hg pressure difference)

As demonstrated in Figure 38 and supported by the analysis of bias, the pressure difference between right and left measurements was small for approximately 90 % of the sample. Therefore, the measurements obtained in either position are interchangeable. However as demonstrated by the outlier and the limits of agreement that were greater than the pressure differences specified as clinically significant, individual variations in response to position must be assessed.

Individual Position-Related PAW Pressure Changes Versus Individual Baseline Fluctuation

For each individual, the change in pressure observed during the position portion of the study was compared to the baseline fluctuation for the individual. These data are summarized in Figure 39. As can be seen in Figure 39, 8 of 18 subjects (44%) exceeded baseline fluctuation, but only two of these individuals (#27 and #28), who are described below, had pressure changes that were clinically significant. Although subjects #27 and #28 had the largest difference between baseline fluctuation and position-related pressure changes, neither subject demonstrated a clinically significant fluctuation in baseline PAW pressure (#27: fluctuation = 2 mm Hg; #28: fluctuation = 3 mm Hg). Therefore, the degree of baseline fluctuation was not useful as a predictor of which patients would

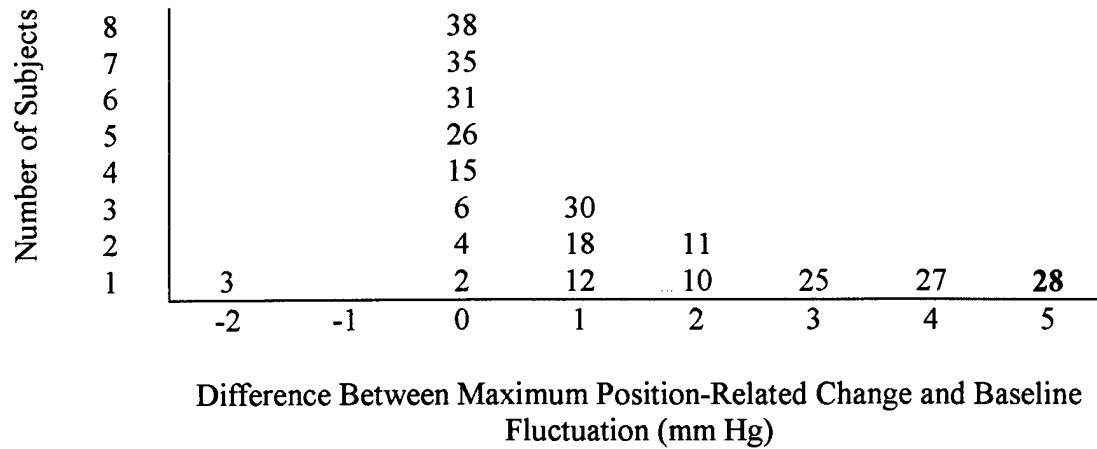


FIGURE 39. Pulmonary Artery Wedge Pressure: Difference between the maximum position-related pressure change and baseline fluctuation for each subject. The difference between the baseline fluctuation and the maximal pressure fluctuation observed during the position portion of the study are plotted on the X-axis. A negative number indicates the position-related pressure change was less than baseline fluctuation for the individual, while a positive number indicates that the position-related change was greater than baseline fluctuation. The numbers on the graph are the subject code numbers. The bold number reflects the one subject (#28) who demonstrated a clinically significant position-related pressure change.

demonstrate clinically significant pressure changes in response to a specific position. In addition, citing the expected fluctuation as a cause of the position-related pressure change was only appropriate if the pre-post measurements were dissimilar.

Individual Data for Subjects Who Exceeded Expected Pressure Fluctuation

Nine subjects demonstrated clinically significant differences in one or more pressures (e.g., PAS, PAED, PAM, PAW). Of these, clinically significant pressure changes in one pressure were noted in five subjects (#6, #8, #11, #17, #27), in two pressures in two subjects (#4, #24), and in three pressures in two subjects (#5, #28). Clinically significant pressure differences were previously described (PAS > 5 mm Hg, PAED > 5 mm Hg, PAM > 5 mm Hg, PAW > 4 mm Hg). A summary of the subjects with clinically significant differences is located in Table 14, and the individual data are located in Appendix K.

Subjects Who Demonstrated Clinically Significant Pressure Differences in Three Pressures

Subject #5

Subject #5, a 50-year old man who was 20 hours status-post a four-vessel CABG, demonstrated clinically significant differences in three pressures (PAS, PAED, and PAM). The patient had clinically significant pressure changes in one PAS pressure pair (Supine-1 – Right), four PAED pressure pairs (Supine-1–Right, Supine-1–Left, Right–Supine-2, and Left–Supine-2), and two PAM pressure pairs (Supine-1–Right, Supine-1–Left).

TABLE 14. Summary of Subjects with Clinically Significant* Pressure Differences

Position Pair (n)	Number (n)	Valid Percent	Subject Number	Pressure Change	Possible Etiology of Observed Change
S1 – S2					
PAS (26)	0	0%	---	---	---
PAED (26)	0	0%	---	---	---
PAM (26)	0	0%	---	---	---
PAW (16)	0	0%	---	---	---
S1 – Right					
PAS (28)	2	6.2%	#5 #6	↓8 mm Hg ↑6 mm Hg	Mixed Effect: S1-S2 ↓ 3 mm Hg; S1-R ↓ 5 mm Hg Mixed Effect: S1-S2 ↑ 3 mm Hg; S1-R ↑ 6 mm Hg
PAED (28)	1	3.6%	#5	↓6 mm Hg	Position Effect: S1-S2 ↑ 2 mm Hg; S1-R ↓ 5 mm Hg
PAM (28)	1	3.6%	#5	↓8 mm Hg	Mixed Effect: S1-S2 ↓ 4 mm Hg; S1-R ↓ 8 mm Hg
PAW (10)	2	20.0%	#27 #28	↑6 mm Hg ↑5 mm Hg	Position Effect: S1-S2 0 mm Hg; S1-R ↑ 6 mm Hg Mixed Effect: S1-S2 ↑ 1 mm Hg; S1-R ↑ 4 mm Hg
S1 – Left					
PAS (28)	0	0%	---	---	---
PAED (28)	1	3.6%	#6	↓6 mm Hg	Position Effect: S1-S2 ↑ 2 mm Hg; S1-L ↓ 6 mm Hg
PAM (28)	3	10.7%	#4 #5 #24	↑6 mm Hg ↓7 mm Hg ↑7 mm Hg	Position Effect: S1-S2 = 0; S1-L ↑ 6 mm Hg Mixed Effect: S1-S2 ↓ 4 mm Hg; S1-L ↓ 7 mm Hg Position Effect: S1-S2 ↑ 1 mm Hg; S1-L ↑ 7 mm Hg
PAW (16)	0	0	---	---	---
Right – Left					
PAS (29)	1	3.8%	#24	↑6 mm Hg	Position Effect: S1 – L ↑ 5 mm Hg/S1-R ↓ 1 mm hg
PAED (29)	3	10.3%	#11 #17 #28	↑6 mm Hg ↓6 mm Hg ↓6 mm Hg	Position Effect: S1-R ↓ 3 mm Hg/S1-L ↑ 3 mm Hg Position Effect: S1-L ↓ 5 mm Hg/S1-R ↑ 1 mm Hg Position Effect: S1-L ↓ 2 mm Hg/ S1-R ↑ 4 mm Hg
PAM (29)	2	6.9%	#24 #28	↑6 mm Hg ↓6 mm Hg	Position Effect: S1-L ↑ 7 mm Hg/ S1-R ↓ 1 mm Hg Mixed Effect: S1-S2 ↓ 2 mm Hg; S1-R ↑ 4 mm Hg/S1-L ↓ 2 mm Hg
PAW (10)	2	20.0%	#27 #28	↓5 mm Hg ↓8 mm Hg	Position Effect: S1-R ↑ 6 mm Hg; S1-L ↑ 1 mm Hg Position Effect: S1-R ↑ 5 mm Hg/S1-L ↓ 3 mm Hg, S1-S2 ↑ 1 mm Hg

TABLE 14. Summary of Subjects with Clinically Significant* Pressure Differences
(continued)

Position Pair	Number (n)	Valid Percent	Subject Number	Pressure Change	Possible Etiology of Observed Change
Right – S2					
PAS (29)	1	3.5%	#8	↓6 mm Hg	Equivocal: No pre-post measure
PAED (29)	2	6.8%	#5	↓6 mm Hg	Position Effect: S1-S2 = 0; S1-R ↓ 6 mm Hg
			#28	↓6 mm Hg	Mixed Effect: S1-S2 ↓ 2 mm Hg/S1-R ↑ 4 mm Hg
PAM (29)	1	3.4%	#28	↓6 mm Hg	Mixed Effect: S1-S2 ↓ 2 mm Hg/ S1-R ↑ 4 mm Hg
PAW (10)	1	10.0%	#24	↓6 mm Hg	Position Effect: S1 – R ↑ 6 mm Hg
Left – S2					
PAS (30)	1	3.3%	#24	↓6 mm Hg	Position Effect: S1-S2 ↓ 1 mm Hg; S1-L ↑ 5 mm Hg
PAED (30)	2	6.6%	#5	↓6 mm Hg	Position Effect: Position Effect: S1-L ↓ 6 mm Hg
			#4	↑ 6 mm Hg	Mixed Effect: L ↑ 5 mm Hg/S1-S2 ↓ 1 mm Hg
PAM (30)	2	6.7%	#4	↓6 mm Hg	Position Effect: S1-L ↑ 6 mm Hg
			#24	↓6 mm Hg	Position Effect: S1-S2 ↑ 2 mm Hg; S1-L ↑ 7 mm Hg
PAW (15)	0	0	---	---	---

S1 = Supine-1 position; S2 = Supine-2 position, Right = 30-degree right lateral position; Left = 30-degree left lateral position.

*Clinically significant pressure change is defined as: Δ PAS > 5 mm Hg; Δ PAED > 5 mm Hg; Δ PAM > 5 mm Hg, Δ PAW > 4 mm Hg

The difference in the PAS Supine-1–Right pressure pair was a mixed effect, that is, the clinically significant pressure change was related to a 3 mm Hg pressure decrease between the Supine-1 and Supine-2 measurements and a 5 mm Hg pressure decrease between Supine-1 and Right. Because this pressure difference was a combination of pressure changes, it was interpreted as not clinically significant. However, these data demonstrate the potential for a clinically significant difference, simply as a result of the expected pressure fluctuation (e.g., pressure difference Supine-1-Supine-2).

The difference in both PAED pressure pairs were related to 6 mm Hg pressure decreases in PAED pressures in both right and left-lateral positions. There was no pressure difference between the Supine-1 and Supine-2 measurements. These pressure changes were classified as position-related, that is, an expected pressure fluctuation could not be used to explain these changes.

The differences in the PAM pressure pairs were related to 6 mm Hg pressure decreases in PAM pressures in both right and left-lateral positions. However, there was also a 4 mm Hg decrease in pressure between the Supine-1 and Supine-2 measures; thus, a portion of this PAM pressure decrease could be explained by the expected fluctuation in pressures. Therefore, neither the 8 mm Hg pressure decrease between Supine-1 and Right nor the 7 mm Hg decrease between Supine-1 and Left were classified as position induced clinically significant pressure changes. These pressure changes again demonstrate the potential for a clinically significant difference simply as a result of the expected pressure fluctuation (e.g., pressure difference between Supine-1 and Supine-2).

Possible factors that may have contributed to these changes included extreme damping of the pressure waveforms in all positions. However, there were clear decreases in pressures in both the right and left lateral positions. The subject who was receiving oxygen at 48% FiO_2 and had a SaO_2 of 96%, had a rapid respiratory rate that varied in each position (Supine-1 = 30 breaths/minute; Right = 40 breaths/minute; Left = 30 breaths/minute; Supine-2 = 35 breaths/minute). If the rapid respiratory rate were contributory, as a result of active expiration, the observed pressure changes would be expected to increase not decrease. A respiratory-induced decrease in measured pressures cannot be used as an explanation, as this was controlled for by measuring pressures at end-expiration. However, as can be seen in Figure 40, which is an example of the PA waveforms from this subject, the amplitude of the waveforms was highly variable and may reflect a respiratory effect that was amplified in the lateral position. The PA catheter was positioned in the right PA, but wedge pressures were not obtainable. Thus the catheter may have been in a Zone 2 vascular bed and more susceptible to alveolar pressure changes. However, as previously noted, measuring pressures at end-expiration controlled for the effects of alveolar pressure. Other factors that may have contributed include the patient's hyperdynamic status, which was demonstrated by sinus tachycardia (120 beats/minute), $\text{CO} = 13.4 \text{ L/min}$, $\text{CI} = 5.5 \text{ L/min/m}^2$, $\text{SVR} = 400 \text{ dynes/sec/cm}^5$; however, there were no changes in the patient's cardiovascular indices during the study. There were also no changes in vasoactive medications during the study, although the

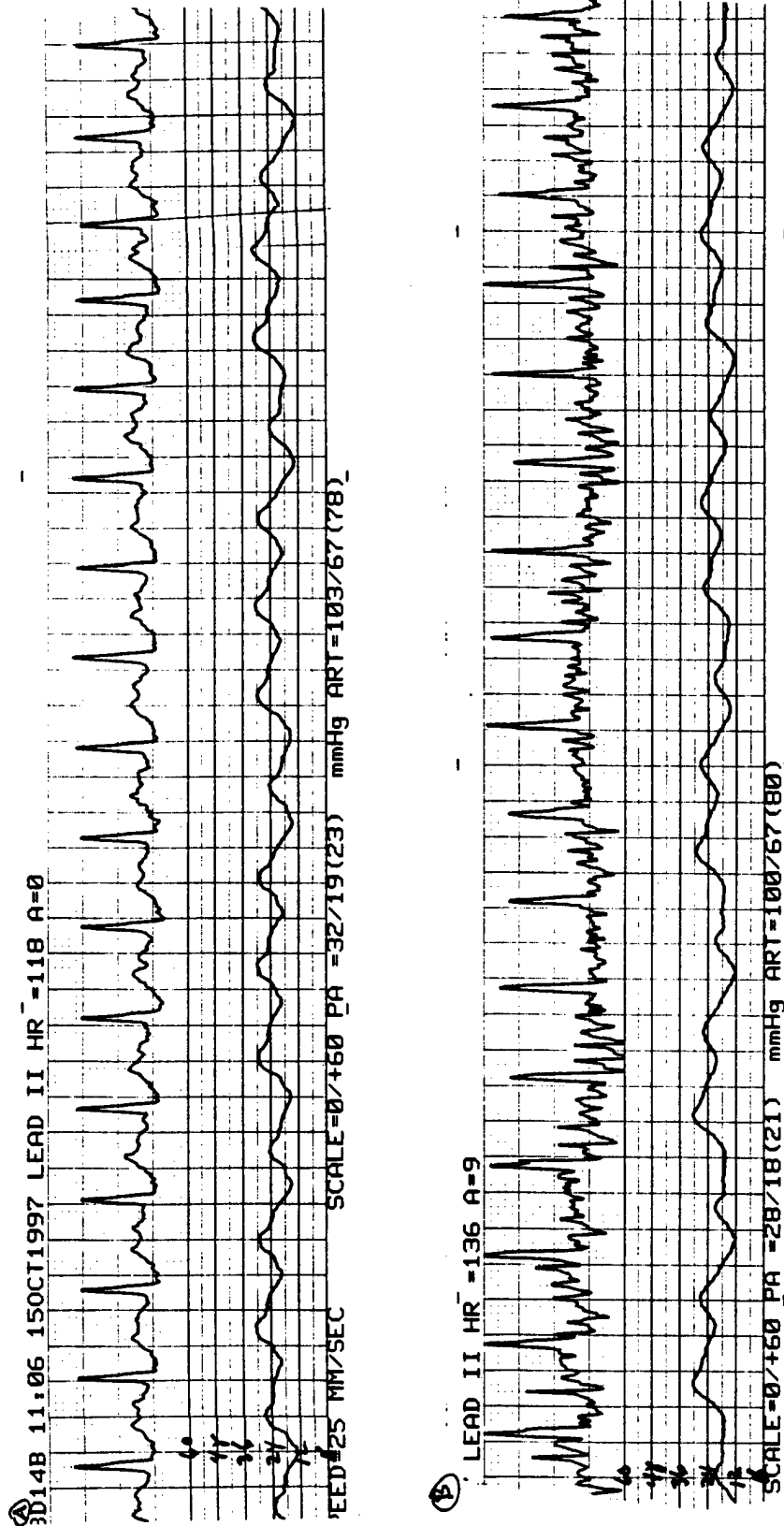


FIGURE 40. PA Waveforms: Subject #5. Note the increased waveform variability in the right-lateral (B) versus supine (A) position. The increased variability of the amplitude of the waveforms may reflect a respiratory effect that was amplified in the lateral position. However, the $\Delta\text{PAWP}/\Delta\text{PA}$ ratio was less than 2; thus, the changes were not indicative of a Zone 2 effect.

patient did receive intravenous morphine 75 minutes before the study and metoprolol, a beta adrenergic blocking agent, 45 minutes before the study. These medications do not appear to be contributory as the pre (Supine-1) and post (Supine-2) measurements were similar. Medical history that may have been contributory included diabetes mellitus, gastro-esophageal reflux disease, obesity (165% of ideal body weight), and anxiety.

Subject #28

Subject #28, a 72 year old woman who was 16 hours status post AVR and a three vessel CABG surgery, demonstrated clinically significant pressure differences in three pressures (PAED, PAM, and PAW). The patient had clinically significant pressure changes in two PAED pressure pairs (Right-Left and Right – Supine-2), two PAM pressure pairs (Right-Left, Right–Supine-2), and two PAW pressure pairs (Supine-1–Right, Right-Left).

The differences in the PAED pressure pairs were the result of a combination of pressure changes. The Right-Left difference of 6 mm Hg was attributable to a 2 mm Hg decrease in the PAED in the Left position and a 4 mm Hg increase in the PAED in the Right position. The Right –Supine-2 pressure difference of 6 mm Hg was attributable to a 2 mm Hg pressure decrease between the pre (Supine-1) and post (Supine-2) measures, and a 4 mm Hg increase in the PAED in the Right. Because the pressure differences were a combination of pressure changes, they were interpreted as clinically insignificant for any single measurement. However, these data demonstrate the potential for a clinically significant difference simply as a result of the expected pressure fluctuation

(e.g., pressure difference Right–Left), or the combination of expected fluctuation (Supine-1-Supine-2) in addition to a position-related effect (Right).

The differences in the PAM pressure pairs were a combination of a 4 mm Hg pressure increase in PAM pressures in the Right position and a 2 mm Hg decrease in pressure between Supine-1–Left and Supine-1–Supine 2. Although there was an increase in pressure in the Right lateral position, this pressure change was characterized as mixed as a result of the change in the pre-post measures. That is, the pressure difference (Right-Left and Left–Supine-2) may have increased as a result of expected fluctuation (2 mm Hg decrease between Supine-1 and Supine-2). However, as demonstrated by increases in other pressures in the right lateral position, there was a consistent position-related component to the pressure change.

The differences in the PAW pressure pairs were a combination of a 5 mm Hg pressure increase in PAW pressures in the right position, a 3 mm Hg pressure decrease in the left position, and a 1 mm Hg increase in pressure between Supine-1 – Supine 2. The 5 mm Hg pressure increase between the Supine-1–Right was clinically significant, but the expected pressure fluctuation (Supine-1–Supine-2) may have contributed slightly. The 8 mm Hg pressure difference between the Right and Left lateral positions was also characterized as position-related, as the Right position change exceeded clinical significance. These particular findings are extremely important as they demonstrate the potential for marked difference between pressures measured in different positions as the result of a combination of position effect and expected fluctuation.

The subject's PA waveforms were highly variable, with a gradual pressure decline from the onset of expiration to end-expiration, as well as an 18 to 24 mm Hg decrease in pressure during inspiration (Figure 41). The waveforms were not excessively damped. The subject's blood pressure was labile during the study (Supine-1: 82/44/62; Right: 104/52/70; Left: 109/57/70; Supine-2: 84/53/64). However, the highest PA pressures (Right) did not occur at the same time as the highest blood pressure. The lability in blood pressure may have been related to the patient's underlying cardiovascular disease, which included LV hypertrophy with an akinetic and thinned inferoposterior wall, mitral regurgitation (which was not apparent on the PA or PAW pressure waveforms), LA enlargement, LV dilatation, moderate tricuspid regurgitation. In addition, the patient required an intra-aortic balloon pump (IABP) for weaning from cardiopulmonary bypass and for the first four hours after surgery, at which time the IABP was removed. It is important to note that the increases in blood pressure occurred only in the lateral position, and may have reflected the physiologic stress of positioning or the lateral position. Finally, the patient received oral doses of isosorbide dinitrate (Isordil) (nitrate; onset = 45-60 minutes), amlodipine (Norvasc) (calcium-channel blocker; onset = 30-50 minutes), and furosemide (Lasix) (diuretic; onset = 30-60 minutes). However, these medications do not appear to have affected the PA pressures or the ABP, as both the Supine-1 and Supine-2 PA pressure and ABP were similar.

FIGURE 41. PA Waveforms: Subject #28. Patient demonstrated clinically significant changes in PA pressures. Despite the large respiratory variation, the $\Delta\text{PAWP}/\Delta\text{PA}$ ratio was less than 2, which indicates that the pressure changes were not related to a Zone 2 effect. The decrease in pressures throughout expiration may represent the normal decrease in SV that occurs during the expiratory period.

Subjects Who Demonstrated Clinically Significant Pressure Differences in Two PressuresSubject #4

Subject # 4, a 60 year-old man who was 14 hours status post AVR, demonstrated clinically significant pressure differences in two pressures (PAED and PAM). The patient had clinically significant pressure changes in one PAED pressure pair (Left–Supine-2) and two PAM pressure-pairs (Supine-1-Left and Left–Supine-2).

The 6 mm Hg pressure decrease between Left – Supine-2 was the result of a 5 mm Hg increase in the PAED pressure in the left lateral position and a 1 mm Hg decrease in pressure between the pre (Supine-1) and post (Supine-2) measurements. While the 6 mm Hg pressure difference was significant, it was the result of a nonsignificant 5 mm Hg pressure increase between the Supine-1 – Left and a 1 mm Hg pressure change that may have been the result of baseline fluctuation. Therefore, this position-related pressure change was characterized as clinically nonsignificant, although there was a strong position component to the pressure change.

The 6 mm Hg pressure increase in the Supine-1-Left was the result of a 6 mm Hg increase in the PAM pressure in the left lateral position, and the 6 mm Hg decrease between Left – Supine-2 was the result of a return to baseline. There was no difference in the Supine-1 and Supine-2 pressure measurements. Therefore, this pressure change was characterized as position-related.

One clinical factor that was of particular interest, and perhaps contributory to these findings, was the 3.5 cm difference between the distance from the bed surface to the LA

reference level (Right = 12.0 cm; Left = 8.5 cm; Supine = 11.5 cm). If one measurement were considered to be erroneous, this difference in the height of the reference levels would result in a 2.5 mm Hg measurement error, which would contribute to, but not entirely explain the position-related pressure change. However, the effect of the variable reference level cannot be entirely discounted for either pressure. The blood pressure was also variable during the study (Supine-1: 111/54/73; Right: 125/54/74; Left: 127/58/76; Supine-2: 142/73/88); however, there was no difference in the pre (Supine-1) and post (Supine-2) pressure measurements, despite the increase in blood pressure. Finally, while the PAM position change in the left lateral position was characterized as position-related, it was possible that the PAED pressure change in the left lateral position may have simply been a reflection of an expected pressure fluctuation.

Subject #24

Subject #24, an 89 year-old man who was 22 hours status post AVR, demonstrated clinically significant differences in two pressures (PAS, PAM). The patient had clinically significant pressure changes in three PAS pressure pairs (Supine-1 – Left, Right – Left and Left – Supine-2), and three PAM pressure pairs (Supine-1 – Left, Right – Left, and Left – Supine-2).

The differences in all three PAS pressure pairs were related to a 5 mm Hg pressure increase in the PAS pressure in the left-lateral position. There was a 1 mm Hg pressure decrease between the Supine-1 and Supine-2 measurements and the Supine-1 – Right measurements. These pressure changes were classified as non-position-related, as the

position-related change (Supine-1 – Left) did not exceed clinical significance. Although the pressure change was not classified as position-related, there was a strong position component to the pressure change. Further support for a position-related component of this pressure change was the increase in both the PAM and ABP in the Left position.

The differences in all three PAM pressure pairs were related to a 7 mm Hg pressure increase in the PAM pressure in the left-lateral position. There was a 1 mm Hg pressure increase between the Supine-1 and Supine-2 measurements and the Supine-1 – Right measurements. These pressure changes were classified as position-related, that is, an expected pressure fluctuation could not be used to explain these changes, although the 1 mm Hg pressure increase between Supine-1-Supine-2 contributed slightly to the difference.

One factor that may have contributed include the marked variability in blood pressure throughout the study (Supine-1 = 148/63/90, Right = 133/65/81, Left = 151/70/96, Supine-2 = 137/62/87), while the HR was continuously paced at 96. The highest PA pressure measurements occurred in the left-lateral position (Sequence B = second position). Although there was marked catheter whip, there were increases in all the PA pressures (PAS, PAED, and PAM) in the left-lateral position compared with other positions, and the waveform configuration was unchanged; therefore, these changes were characterized as position-related. The increased blood pressure was interpreted as an effect of the left-lateral position and a possible contributory factor to the increased PA pressures, thus, position was still characterized as the primary causal factor.

Subjects Who Demonstrated Clinically Significant Pressure Differences in One PressureSubject #6

Subject #6, a 73 year-old man who was 14 hours status-post five vessel CABG surgery, demonstrated a clinically significant pressure difference in one PAS pressure-pair (Supine-1-Right). The 6 mm Hg pressure increase between Supine-1 and Right was the result of a 6 mm Hg increase in the PAS pressure in the Right-lateral position and a 3 mm Hg increase in pressure between the pre (Supine-1) and post (Supine-2) measurements. Because this pressure change was a combination of a change in the pre-post measurements and a position-related change, it was classified as clinically nonsignificant position-related change. Further support for the non-position-related classification included: (1) the maximum increase for any other pressure in the Right position was a 2 mm Hg increase in the PAED, (2) the blood pressure was stable throughout the position portion of the study, and (3) the difference between the Right and Left reference levels was 0.5 cm.

Subject #8

Subject #8, a 76 year old man who was 15 hours status post patch closure of an atrial septal defect with pericardium and a superior vena cava augmentation patch, had a clinically significant difference in one PAS pressure pair (Right – Supine-2). Classification of the pressure change was not possible, as the Supine-1 data were not available. However, several events during data collection, which required delays in the protocol (repositioning of the PA catheter, nausea, and an MD visit) all introduced

possible external factors that could have affected the measurements. In addition, possible clinical factors that could have affected the measurements included atrial fibrillation and variability of blood pressure during the study (Right: 110/50/71; Left: 117/50/73; Supine-2: 130/51/72). However there was no relationship between and increase in blood pressure and the PA pressures. Due to the lack of a pre-post comparison pressures, characterization was not possible. However, as described there were numerous external factors that may have affected these measurements.

Subject #11

Subject #11, a 73 year-old man who was 15 hours status post AVR, had a clinically significant difference in one PAED pressure pair (Right – Left). The 6 mm Hg pressure difference was attributable to a 3 mm Hg decrease in the PAED in the right lateral position and a 3 mm Hg increase in the PAED in the left lateral position. There were no changes in hemodynamic indices or medications during the study. The PA catheter was located in the right PA; however, wedge pressures were not attainable. The other finding on chest radiograph was a left pleural effusion. Of note, there was a 3.0 cm difference between the distance from the surface of the bed to the LA reference point for the two lateral positions: Right = 11.5 cm; Left = 8.5 cm. If it was assumed that one measurement was correct and the other incorrect, these different heights would result in an approximate 2 mm Hg pressure measurement error for one reading, which would not explain the observed difference in its entirety.

Subject #17

Subject #17, a 72 year-old man who was 10 hours status-post four vessel CABG surgery, demonstrated a clinically significant pressure difference in one PAED pressure-pair (Right – Left). This pressure change was a result of a 5 mm Hg decrease in the PAED in the left lateral position and a 1 mm Hg pressure increase in the right lateral position.

Medical history that may have been contributory included ischemic cardiomyopathy (ejection fraction 15-20%), congestive heart failure, non-insulin dependent diabetes mellitus (NIDDM), pulmonary hypertension, and atrial fibrillation. The subject also had a postoperative bleed, and received fresh frozen plasma (FFP) three hours before the study. At the time of the study chest tube output was 20 ml/3 hours. The most likely contributory factors were atrial fibrillation and the variability in blood pressure during the study (S1: 90/44/58; Right: 90/49/61; Left 79/49/56; S2: 86/45/58), which was probably related to the atrial fibrillation. It is important to note that the waveforms that were selected for pressure measurement were end-expiratory with a similar diastolic filling period (R-R interval) relative to the preceding complex (Braunwald, Frye, Aygen, & Gilbert, 1960; Iwase, Aoki, Maeda, Yokota, & Hayashi, 1989). The decreased ABP in the left lateral position, while within 10% of baseline, may have contributed to the decrease in the PAED pressure in the left-lateral position. However, it is impossible to specify a temporal relation between the decreased blood pressure and the decreased PAED, that is, did the decreased blood pressure cause the decreased PAED, or did the

left-lateral position cause a decrease in one or both hemodynamic indices (blood pressure and PAED). Therefore, this pressure change was considered to be the effect of position.

Subject #27

Subject #27, was a 74 year-old man who was 19 hours status post AVR and a four-vessel CABG surgery, demonstrated clinically significant pressure changes in three PAW pressure-pairs (Supine-Right, Right-Left, Right-Supine-2). The pressure changes were all related to a 6 mm Hg increase in the PAW pressure in the Right-Lateral position.

There was no change in pressure between the pre (Supine-1) and post (Supine-2) measures. Of importance, the patient, who was intubated, required suctioning after the Left-position. Therefore, this pressure increase and the concurrent increase in blood pressure and HR in the Right -position (Supine-1: 109/55/70, HR = 95; Right: 130/61/85, HR = 104; Left: 114/58/74, HR = 96; Supine-2: 108/55/70; HR = 101), most likely reflected the effect of suctioning. It is of interest to note that despite a delay of approximately 10 to 12 minutes the patient's vital signs and PA pressures remained elevated; however, the pressure did return to baseline at the time of the Supine-2 measurements (approximately 25 minutes after suctioning). Therefore, these pressure changes were characterized as related to a non-position-related external factor.

Lung Zone Effect

Of particular concern in this study was the ability to assess for maintenance of a continuous vascular segment (Zone 3 segment) between the PA catheter tip and the left atrium. Therefore, three methods were used to assess for probable Zone 3 placement.

First, the patient's most recent AP chest radiograph was reviewed. One hundred percent of the sample had the tip of the catheter positioned at a point that was at or below the level of the left atrium. Second, during wedging the pressure waveform was analyzed for the following: (1) A flattening of the waveform into a characteristic atrial waveform. (2) Upon deflation of the balloon, an immediate return to a PA waveform configuration. The presence of a partial wedge, which was characterized as a waveform intermediate to the phasic PA waveform and the atrial waveform, resulted in the exclusion of the waveform from analysis. (3) The evaluation of the respiratory artifact induced by mechanical ventilation (inspiratory peak minus expiratory peak value) on the PA and PAW pressure tracings (Teboul et al., 1988). A change in the ratio of the PAW pressure change relative to the change in PA pressure ($\Delta PAWP/\Delta PAP$) greater than 2 was indicative of Zone 2 alveolar compression; while a value of 1 for the ratio indicated no respiratory artifact due to Zone 2 alveolar compression.

Two groups of patients were of concern with regard to the potential for conversion to a non-Zone 3 vascular bed when placed in lateral position: those requiring mechanical ventilation with PEEP and those with decreased intravascular pressures. It was anticipated that if Zone-2 effects were to occur, they would occur when the patient was positioned such that the PA catheter tip was above the left atrium.

There were only four subjects receiving mechanical ventilation, three of who had PEEP of 5 cm H₂O, and one subject with PEEP at 10 cm H₂O. All subjects had increased intravascular pressures, ranging from 12 to 21 mm Hg for the PAED pressure. One

subject had the PA catheter in the right PA, two had the catheter positioned in the main PA, and one subject had the catheter positioned in the RV outflow tract. With the exception of Subject #27, who had an increase in pressure in the right lateral position that was related to endotracheal suction, none of these individuals demonstrated any clinically significant position-related pressure changes.

The pressure changes associated with mechanical ventilation were assessed according to the criteria established by Teboul in four of the five subjects (#25, #27, #29, #35) who completed the position-portion of the study. In subject #36 it was not possible to wedge the catheter. In Subject #29 in the right-lateral position, the pressure ratio was 2.0, which may indicate that there was alveolar compression contributing to the increased pressures observed in this position. In addition, the PAW pressure waveform was slightly more damped in this position relative to other positions. However, the PAM remained greater than the PAW pressure. Therefore, the specification of the pressure changes as indicative of alveolar compression is equivocal. In the other subjects none of the ratios approached 2. Therefore, with the exception of Subject #29 in the right-lateral position, it does not appear that a Zone-2 effect was present.

There were four subjects in the study with decreased PA pressures, two of whom also demonstrated clinically significant pressure changes during the position portion of the study. Subject #4 had a mean PAW pressure of 7 mm Hg and subject # 6 had a mean PAW pressure of 4 mm Hg. The PA catheters for both subjects were positioned in the main PA. In subject #4 all four pressures increased in the left lateral position: (PAS +4

mm Hg, PAED +5 mm Hg, PAM +6 mm Hg, and PAW +1mm Hg). There were no changes in waveform configurations or respiratory variation, and the PAM remained greater than the PAW in all positions. In addition, there was no change in the PAED-PAW pressure gradient in the left lateral position. Therefore, the 1 mm Hg increase in the PAW pressure was interpreted as a position-related change, but not the result of a conversion to a Zone-2 vascular bed. In subject #6, the pressures increased in both right and left position relative to the Supine-1 measurements: (PAS-R: 6 mm Hg, PAS-L: 3 mm Hg; PAED-R: 4 mm Hg, PAED-L: 3 mm Hg; PAM-R: 5 mm Hg, PAM-L: 3 mm Hg; PAW-R: unable to wedge, PAW-L: 2 mm Hg). As with subject #4 these data, particularly the small increase in PAW pressure and no increase in the PAED-PAW pressure gradient, do not support a Zone-2 effect as a causative factor for the observed changes.

The other two subjects (#15 and #38) both had a mean PAW pressure of 7 mm Hg. Subject # 15 had the PA catheter positioned in the main PA, while subject # 38 had the catheter in the left PA. Subject #15 demonstrated no change or a decrease in all PA pressures in all positions, with the largest change a 4 mm Hg decrease in the PAED pressure in the right-lateral position. The PAW pressure decreased 2 mm Hg in the right and 1 mm Hg in the left, Subject #38 had an increase in all pressures in all positions, with the maximum change a 3 mm Hg increase in the right lateral position. The PAW pressure increased 1 mm Hg in both the right and left lateral positions. There were no

changes in waveform configurations and minimal respiratory variation for either subject. Therefore, these changes do not appear to be related to a Zone-2 effect.

Summary

Baseline Fluctuation and Specification of Clinical Significance

Descriptive statistics were used to describe the fluctuation in PA pressures over a 15-minute period. The patient's were not disturbed during the period (no change in medications, suctioning, or position). The fluctuation data are summarized in Table 15. As demonstrated in the scatter plots (Figure 42) there was no relationship between the degree of fluctuation and the absolute pressure.

The mean PAS pressure was 30.6 ± 8.7 (ranged from 17 to 50 mm Hg). The mean fluctuation over the 15-minute study period was 2.8 ± 1.4 mm. The data were normally distributed; therefore, clinical significance was defined as the maximum absolute pressure represented by the mean $\pm 2SD$, which was equal to 5.8 mm Hg. Analysis of the frequency table indicated that an absolute pressure fluctuation of 5 mm Hg included 98% of the sample, therefore an absolute pressure change greater than 5 mm Hg was described as clinically significant.

The mean PAED pressure was 17.1 ± 5.0 mm Hg (ranging from 7 to 34 mm Hg). The mean fluctuation over the 15-minute study period was 2.2 ± 1.4 mm Hg. The data were normally distributed; therefore, clinical significance was defined as the maximum absolute pressure represented by mean $\pm 2SD$, which was equal to 5.1 mm Hg. Analysis of the frequency table indicated that an absolute pressure fluctuation of 5 mm Hg

TABLE 15. Summary of Baseline Fluctuation Data

	Mean (mm Hg)	SD	95% CI of Mean		Median	Minimum	Maximum	Range
			Lower	Upper				
PAS	2.8	1.4	2.3	3.4	3.0	1	5	4
PAED	2.2	1.4	1.7	2.8	2.0	0	5	5
PAM	2.8	2.0	2.0	3.6	2.0	0	9	9
PAW	1.7	1.4	1.1	2.3	2.0	0	6	6

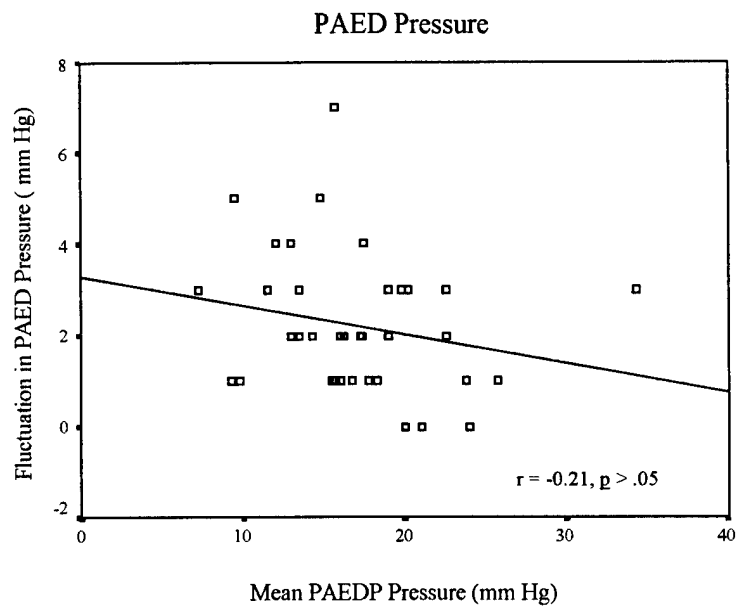
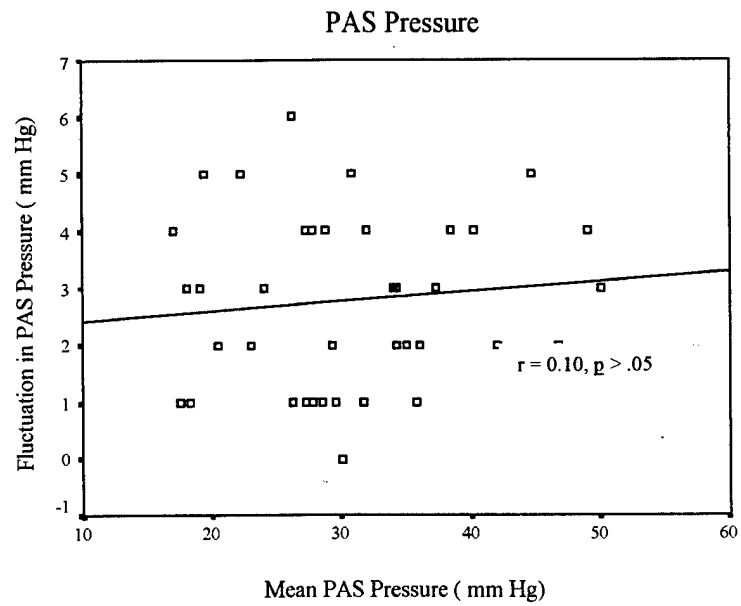


FIGURE 42. Relationship between the Pressure Fluctuation and Absolute Pressure for PAS (A) and PAED Pressures (B).

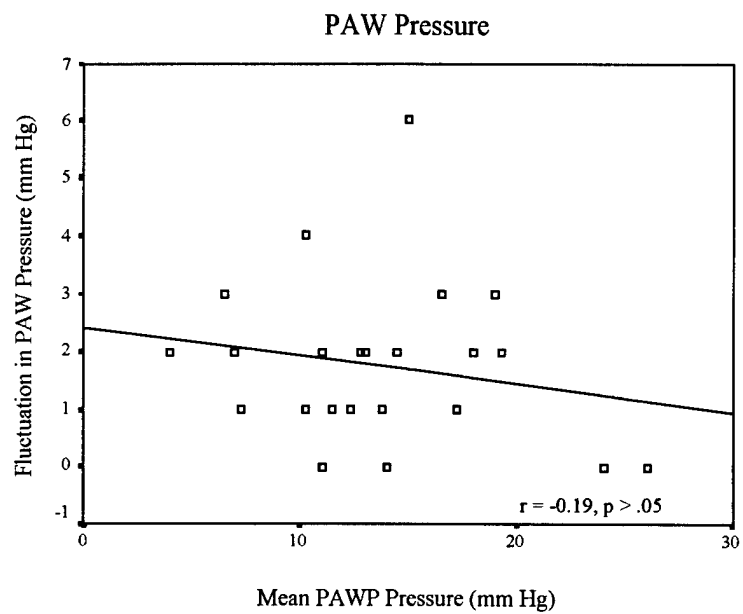
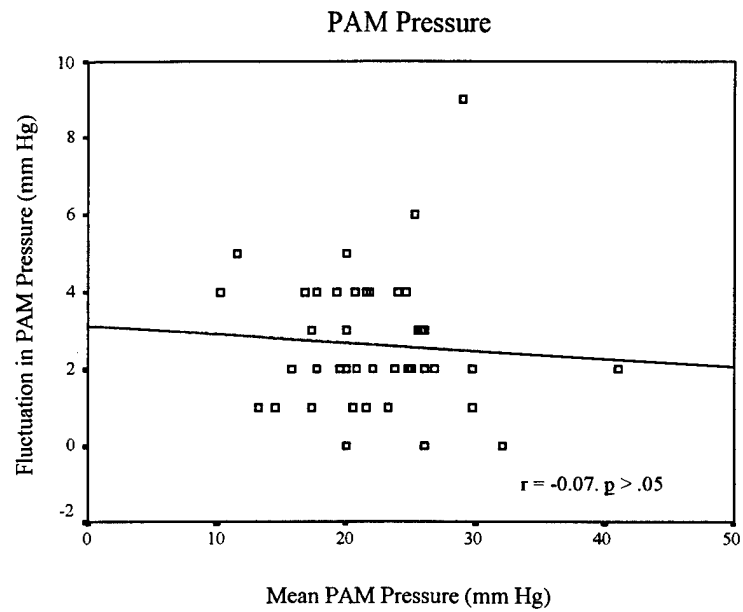


FIGURE 42 (continued). Relationship between the Pressure Fluctuation and Absolute Pressure for PAM (C) and PAW (D) Pressures

included 98% of the sample, therefore an absolute pressure change greater than 5 mm Hg was described as clinically significant.

The mean PAM pressure was 22.2 ± 5.7 mm Hg (ranged from 7 to 34 mm Hg). The mean fluctuation over the 15-minute study period was 2.8 ± 2.0 mm Hg. The data were positively skewed; therefore, clinical significance was defined as the maximum absolute pressure that included 95% of the sample as indicated on a frequency table. An absolute pressure change of 5 mm Hg included 95.2% of the sample. Therefore an absolute pressure change greater than 5 mm Hg was described as clinically significant.

The mean PAW pressure was 13.6 ± 5.3 mm Hg (ranged from 4 to 26 mm Hg). The mean fluctuation over the 15-minute study period was 1.7 ± 1.4 mm Hg. The data were negatively skewed; therefore, clinical significance was defined as the maximum absolute pressure that included 95% of the sample as indicated on a frequency table. An absolute pressure change of 4 mm Hg included 96% of the sample. Therefore an absolute pressure change greater than 4 mm Hg was described as clinically significant.

Effect of Position on PA and PAW Pressures

Repeated-measures analysis of variance and paired t-tests revealed significant differences ($p < .05$) in 7 of 24 pressure pairs (Table 16). In addition, there was a trend towards significance ($.05 < p < .10$) in five additional pairs, including three of six PAW pressure-pairs. However, the small sample size may have limited the power to detect statistically significant differences in the PAW pressures. In all cases, where the difference between pressure pairs was statistically significant, the pressure measured in

TABLE 16. Summary of Statistically Significant Position-Related Pressure Differences

	Mean Difference (± SD)	95% Confidence Interval		Significance
		Low	High	
PAS				
Left > Supine-1	2.0 ± 2.1	1.2	2.8	.000***
Left > Right	1.2 ± 2.8	0.1	2.2	.030*
Left > Supine-2	1.9 ± 2.4	1.0	2.8	.000***
PAED				
Left > Supine-2	1.4 ± 2.7	0.4	2.4	.008**
Left > Supine-1	1.0 ± 2.7	0.1	2.0	.074
Right > Supine-2	0.8 ± 2.2	-0.1	1.6	.063
PAM				
Left > Supine-1	1.5 ± 2.9	0.3	2.6	.014*
Left > Supine-2	1.2 ± 2.6	0.3	2.3	.015*
Right > Supine-2	0.8 ± 1.9	0.1	1.5	.024*
PAW				
Supine-2 > Supine-1	0.8 ± 1.6	-0.06	1.7	.066
Right > Supine-1	1.6 ± 2.4	-0.1	3.3	.065
Left > Supine-1	0.9 ± 1.7	-0.1	1.8	.058

* p < .05; ** p < .01; *** p < .001

the 30-degree left lateral position was greater than the pressures measured in either the Supine-1 or Supine-2 positions. At no time were the mean pressures in either Supine-1 or Supine-2 greater than the pressures obtained in the right or left lateral positions. With the exception of the PAS pressure, the differences between pressure measured in the right and left lateral positions were not statistically significant. In addition, the pressures in the right lateral position were never greater than the Supine-1 pressure measurements, but were statistically significantly ($p < .05$) larger than the Supine-2 measurements for both the PAED and PAM pressures. Despite the statistically significant differences in the seven pairs and the trend toward significance in another five pressure-pairs, the largest mean difference for any pressure-pair, as described by the 95% CI of the mean, was 3.3 mm Hg, which was not a clinically significant difference.

Agreement Analysis

The data were graphically analyzed using the agreement analysis technique described by Bland and Altman (Bland & Altman, 1986; Bland & Altman, 1995). This graphical method of analysis, which allowed for a comparison of the mean difference and variance of data relative to the indicators of clinical significance, provided a means of answering the question: Are the PA pressure measurements obtained in the supine and lateral positions interchangeable?

As summarized in Table 17, with the exception of a few outliers, the pressure differences observed for the supine and lateral pressure pairs did not exceed the values specified as clinically significant; thus the measurements may be considered

TABLE 17. Summary of Agreement Analysis

	Mean Difference (mm Hg) ($\pm$ SD)	95% Limits of Agreement		Precision 95% CI Mean Difference		# Subjects with Clinically Significant Changes (%)
		Low	High	Low	High	
PAS						
Supine - Left	-1.9 $\pm$ 1.9	-5.9	2.0	-2.6	-1.2	3 (9%)
Supine - Right	-1.0 $\pm$ 2.4	-5.7	7.8	-1.8	-0.1	2 (6%)
Right - Left	-1.2 $\pm$ 2.8	-6.7	4.4	-2.2	-0.1	1 (3%)
PAED						
Supine - Left	-1.1 $\pm$ 2.5	-6.1	3.9	-1.7	-0.5	3 (9%)
Supine - Right	-0.6 $\pm$ 2.1	-4.8	3.6	-1.3	0.2	2 (6%)
Right - Left	-0.6 $\pm$ 2.8	-6.1	5.0	-1.6	0.5	3 (10%)
PAM						
Supine - Left	-1.3 $\pm$ 2.5	-6.3	3.7	-2.2	-0.4	4 (13%)
Supine - Right	-0.7 $\pm$ 2.0	-4.7	3.2	-1.4	-0.02	1 (3%)
Right - Left	-0.5 $\pm$ 2.8	-6.1	5.2	-1.5	0.6	2 (7%)
PAW						
Supine - Left	-0.5 $\pm$ 1.7	-3.8	3.3	-1.4	-0.4	3 (9%)
Supine - Right	-0.9 $\pm$ 1.5	-4.0	2.1	-2.1	0.21	1 (11%)
Right - Left	0.78 $\pm$ 3.0	-5.3	6.8	-1.6	3.2	1 (11%)

interchangeable. As summarized below, only a very small percentage of the clinically significant pressure changes were directly attributable to position. However, for some pressure-pairs, the limits of agreement exceeded the pressure changes specified as clinically significant; thus, there was a potential that a small group of patients could demonstrate either a random or a physiologically induced clinically significant pressure change in response to the lateral position. Therefore, the pressures measured in the lateral and supine positions were considered interchangeable; however, individual response to a given position needed to be assessed.

Characteristics of Individuals with Clinically Significant Changes

Of the 581 pressure-pairs analyzed, 28 were initially characterized as clinically significant. The three pressure-pairs from Subject # 27 were excluded, as they were related to the effect of suctioning. Of the remaining 25 pressure-pairs, 12 (2.1%) were directly attributed to position, while 13 (2.2%) were characterized as mixed, that is a pressure change that consisted of a combination of an expected fluctuation plus in some cases a position-related effect. Although the pressure changes observed Subject #8 were indeterminate due to a lack of pre-post measures, they were included in the position-related changes for purposes of analysis.

The clinical characteristics of the eight subjects who demonstrated either clinically significant position-related pressure changes or mixed effects for any position-pair are summarized in Table 18. The characteristics of these subjects were compared with the subjects who did not demonstrate clinically significant changes (Appendix L). There

Table 18. Characteristics of Individuals with Clinically Significant Pressure Changes

Subject #	5	28	4	24	6	8	11	17
Gender	Male	Female	Male	Male	Male	Male	Male	Male
Age	50	72	60	89	73	76	73	73
% IBW	165	139	128	112	129	102	94	128
Diagnosis	CAD	AS	AS, AR	AS, CAD	CAD	ASD	AS	CAD
Diabetes ?	Yes	No	No	No	Yes	No	No	Yes
Surgery	CABG	AVR	AVR	AVR	CABG	ASD Repair	AVR	CABG
CPB	140	176	62	127	200	112	118	122
IT	118	137	--	71	179	74	95	87
HR	125	99	87	97	91	70	76	110
Rhythm	ST	Sinus	Sinus	Paced (VOO)	Sinus	AFib	Sinus	AFib
BP	98/64/77	103/54/68	134/78/97	136/61/85	150/68/94	122/54/77	112/58/76	110/40/60
CI	5.5	2.6	4.4	2.7	4.7	2.9	3.2	3.0
EF	--	40%	72%	--	--	--	--	15-20%
Chest Radiograph	--	Minimal Effusion	--	Bilateral Effusions	Left Pleural Effusion	Left pleural effusion	Left pleural effusion	Left pleural effusion
PA Catheter Position	Right PA	Right PA	Main PA	RV outflow tract	Main PA	Right PA	Right PA	Main PA
# Clinically Significant Changes	PAS (1) PAED (4) PAM (2)	PAED (2) PAM (1) PAW (2)	PAED (1) PAM (2)	PAS (3) PAM (3)	PAS (1)	PAS (1) ?	PAED (1)	PAED (1)
# Position Related Changes	PAS (0) PAED (4) PAM (0)	PAED (0) PAM (0) PAW (1)	PAED (0) PAM (2)	PAS (0) PAM (3)	PAS (0)	PAS (1) ?	PAED (0)	PAED (1)

CAD = coronary artery disease; AS = aortic stenosis; AR = aortic regurgitation; ASD = atrial septal defect; CABG = coronary artery bypass graft; AVR = aortic valve replacement; ST = sinus tachycardia; AFib = atrial fibrillation; PA = pulmonary artery; RV = right ventricular

were no statistically significant ($p > .05$) differences in any characteristics. However, in comparison with the group without clinically significant differences the group with clinically significant differences tended to be older (70 ± 13.5 years versus 64.5 ± 12.7 years) and have undergone a greater number of aortic valve replacement (AVR) procedures than expected (Actual = 3, Expected = 1.1). The age difference was entirely related to the AVR group, although in this sub-sample the difference was not statistically significant (74 ± 14.5 versus 54.5 ± 17.7 years, $p = .27$). There was no difference in age among the patients who underwent CABG surgery (65.3 ± 13.3 versus 64 ± 8.7 years, $p > .05$). Of interest, the cardiopulmonary bypass (123 ± 37 minutes versus 155 ± 56 minutes) and ischemic times (97 ± 29 minutes versus 120 ± 49 minutes) of the clinically significant group were less than those of the group without clinically significant changes.

CHAPTER V

DISCUSSION

The purpose of this study was to determine the effect of 30-degree right and left lateral positions on PA and PAW pressures in critically ill adults. This chapter discusses the results, including a description of the baseline fluctuation in pressures, the position-related changes in PA and PAW pressures, and a description of the subjects who demonstrated a clinically significant change in pressure. Also included are the limitations of the study, recommendations for further study, conclusions, and implications for nursing practice and research.

Discussion of Results

Baseline Pressure Fluctuation

The fluctuation in PA pressures was similar over the baseline and position portions of the study. The data from the baseline period are summarized in Table 19. In comparison with three other studies related to PA pressure fluctuation (Cason et al., 1990; Moser & Woo, 1996; Nemens & Woods, 1982), the mean and variation of the fluctuation for each pressure was less than that observed in the different patient populations (medical-surgical ICU and end-stage heart disease).

The differences in results may simply reflect the differences in the characteristics of the sample. In this study, the inclusion criteria resulted in very homogenous group of patients, while subjects from the studies by both Nemens and Woods (1979) and Cason and colleagues (1990) were more heterogeneous. In addition, methods to improve

TABLE 19. Comparison of Baseline Fluctuation Across Different Studies

	Nemens (1979)	Retailiau (1985)	Cason (1990)	Moser (1996)	Bridges (1998)
Sample	26 ICU patients	32 post-cardiac surgery patients	16 ICU patients	35 end-stage heart failure patients	42 post-cardiac surgery patients
Study Period	q5 X 7 (30 minutes)	q3 x 10 (30 minutes)	Q5 X 6 (25 minutes)	q15 X 7 (90 minutes) (EF = 21 ± 5%)	q5 X 4 (15 minutes)
Mean CO	6.27	--	--		6.57
PAS Pressure Mean ± SD	32.2 ± 11.7	--	32.6 ± 9.0	---	30.6 ± 8.7
PAED Pressure Mean ± SD	18.4 ± 7.1	--	12.6 ± 5.9	---	17.1 ± 5.0
PAM Pressure Mean ± SD	25.0 ± 8.2	--	20.44 ± 7.2	---	22.2 ± 5.7
PAW Pressure Mean ± SD	12.6 ± 3.6	LAP 11.6	---	---	13.6 ± 5.3
Fluctuation PAS Mean ± SD	3.9 ± 1.4	--	6.6 ± 4.1	7	2.8 ± 1.5
Median	4.0	--	5	--	3.0
Min - Max	2-7	--	2-16	1-20	0-6
Fluctuation PAED Mean ± SD	3.4 ± 1.5	--	4.3 ± 3.1	7	2.2 ± 1.4
Median	3.0	--	4	--	2.0
Min - Max	1-6	--	0-11	1-16	0-7
Fluctuation PAM Mean ± SD	2.8 ± 1.6	--	4.9 ± 8.8	--	2.6 ± 1.8
Median	3.0	--	4	--	2.0
Min - Max	1-5	--	0-13	--	0-9
Fluctuation PAW Mean ± SD	3.4 ± 1.7	1.4 (LAP)	--	4	1.7 ± 1.4
Median	3.0		--	--	2.0
Min - Max	0-7	0-3	--	1-12	0-6

Min = minimum fluctuation; Max = maximum fluctuation; LAP = left atrial pressure

reliability of PA pressure measurements have changed over time. For example, Nemens measured pressures over an entire respiratory cycle, while measurements in this study were obtained at end-expiration, which would decrease the pressure variability.

The differences in the degree of fluctuation may be disease or population (post-surgical) specific. For example, in the study by Retailliau and colleagues (1985) of LA pressure fluctuation over a 30-minute period, the mean LA pressure fluctuation was 1.4 mm Hg. One hundred percent of the fluctuations ranged from 0 to 3 mm Hg. These results are similar to the findings in this study, where 92% of the subjects had PAW pressure fluctuations less than or equal to 3 mm Hg. In contrast, in the study by Nemens and Woods, in a heterogeneous group of ICU patients, only 60% of the subjects had PAW pressure fluctuations less than or equal to 3 mm Hg (Nemens & Woods, 1982).

The relative decrease in variability observed in this study may reflect the depression and desynchronization of the rhythmic fluctuations in hemodynamic indices (HR, SV, CO) that have been reported in post-cardiac surgery patients, specifically, (Lanuza, 1995; Woods, 1991; Woods, Felver, & Hoeskel, 1993), and postoperative patients, in general (Farr, Campbell-Grossman, & Mack, 1988; Farr, Keene, Samson, & Michael-Jacoby, 1986). The decreased pressure fluctuations may also be a manifestation of the general cardiac depression that occurs in the first 24-hours after cardiopulmonary bypass (CPB) (Breisblatt et al., 1990; Mangano, 1985; Phillips et al., 1983; Royster, 1993). The CO is one of the factors that affects PAS pressure: thus, a decrease in CO variability may be manifested by a decrease in PAS pressure variability.

Another possible explanation for the differences in the degree of fluctuation between groups is that there is a direct relationship between the degree of variation and the absolute pressure, that is, an increase in absolute pressure is associated with an increase in pressure fluctuation. One possible mechanism for the relationship between variation and absolute is the significant relationship ($r = 0.69$, $p = .002$) between PA pressure and CO variability that has been observed in patients with pulmonary hypertension (Rich, D'Alonzo, Dantzker, & Levy, 1985). This relationship has also been observed in some medical ICU (MICU) patients (Sasse et al., 1994). In the MICU patients with stable ($\pm 5\%$ of the mean) covariables (HR, RR, mean arterial pressure, PAM pressure), the mean coefficient of variation for CO was 6.4%; whereas, "covariable unstable" patients (covariable change greater than $\pm 5\%$) had a mean coefficient of variation for CO of 9.9% ($p < .05$). That is, in subjects with increased CO variability, there was also increased variability in the cardiopulmonary covariables, including the PAM pressure; although a temporal relationship could not be specified.

This relationship between CO and PA pressure variability may help to explain the increased pressure fluctuations observed in the ICU patients in the studies by Nemens and Woods (1979) and Cason and colleagues (1990). In addition, the increased variability in CO by Moser and Woo (1996) may explain the large pressure fluctuations in the patients with end-stage heart failure. In contrast, depression of the rhythmic changes in CO observed in cardiac surgery patients may be manifested by a decrease in

PA pressure fluctuation. It is of interest to note that, for this study, there was no relationship between the absolute pressure and the degree of pressure fluctuation.

Use of Baseline Fluctuation as a Guide to Interpretation of Response to Position Change

In a recent summary of the literature related to position, Gawlinski (1997) recommended comparing baseline supine fluctuation and the difference between supine and lateral position pressures. If the fluctuations were similar, Gawlinski suggested it would be appropriate to perform the PA pressure measurements in the lateral position, even if these pressure changes were greater than the standard definition of clinical significance (Nemens & Woods, 1982).

As noted in this study there was no predictive relationship between the degree of baseline fluctuation and the response to a given position. Therefore, the recommendation by Gawlinski for the use of baseline fluctuation as a point of comparison may not be appropriate, unless the definition of clinical significance is further refined to: "any change greater than baseline fluctuation". This definition would seem to have limited utility as many of the patients in this study had baseline fluctuation changes of 2 mm Hg or less.

Position Effect

There are several possible explanations for the observed increase in the mean pressures in both the left and right lateral positions. This section addresses four questions related to these findings: (1) What effect would an error in the reference level have on the differences observed in this study? (2) Could these findings be the result of compression

of the heart in the lateral position, and if so, what are the possible sources of this compression? (3) Could a change in the relative relationship between cardiac structures, specifically the vena cava and right ventricle, account for the observed changes? (4) Could a position-induced conversion from a functional Zone 3 to a Zone 2 pulmonary vascular bed account for the changes observed in the study?

Reference Level

The first question is: Could a systematic error in the reference level account for the differences observed in the study?

The reference level for the 30-degree right and left lateral positions was a point one-half the distance from the surface of the bed to the left sternal border (VanEtta et al., 1993; VanEtta, 1992). This 50% value, which was slightly different from the actual height of the left atrium, represents a clinical compromise (i.e., it would be difficult in a clinical setting to readily identify a point 48% of the distance from the surface of the bed to the left-sternal border). In the 30-degree left lateral position the left atrium was at a point $47.7\% \pm 7.23\%$ (95% CI = 43.8%, 51.6%) of distance from surface of the bed to the left-sternal border at the fourth intercostal space. In the 30-degree right lateral position the left atrium was at a point $53\% \pm 9.8\%$ (95% CI = 47.9%, 58.3%) of the distance from the surface of the bed to the left sternal border at the fourth intercostal space.

There were no significant differences ($p < .05$) between the data from this study and VanEtta's study with regard to the age, sex, height, weight, body surface area, or percent ideal body weight of the subjects. The only difference was the patient's diagnosis, with

the subjects from VanEtta's study predominantly having medical diagnosis (cardiac: 62.5%, gastrointestinal bleed: 18.75%; surgery – nonthoracic: 6.25%). However, there were no indications of differences in chest wall configuration between samples (e.g., number of subjects with underlying pulmonary disease). Therefore, the data from VanEtta's study, regarding the location of the left atrium, were thought to be generalizable to this study.

Using data from this study regarding the average distance from the surface of the bed to the reference level (Right 10.3 ± 1.5 cm, Left 9.2 ± 1.5 cm, Supine 9.1 ± 1.8 cm) and applying VanEtta's data regarding the exact LA reference level relative to the 50% point used in this study would result in the following pressure measurement errors (Figure 43):

Supine: Mean pressure difference = - 0.73 mm Hg (underestimate the actual pressure by 0.62 mm Hg), and on average the measurement error would be between 0.96 and 0.34 mm Hg less than the actual pressure;

30-degree right-lateral: Mean pressure difference = 0.44 mm Hg (overestimate), and on average the potential pressure measurement error would be between 1.0 mm Hg less than to 0.37 mm Hg greater the actual pressure;

30-degree left-lateral position: Mean pressure difference = - 0.31 mm Hg (underestimate), and on average the potential measurement error would be between 1.28 mm Hg less than to 0.61 mm Hg greater than the actual pressure.

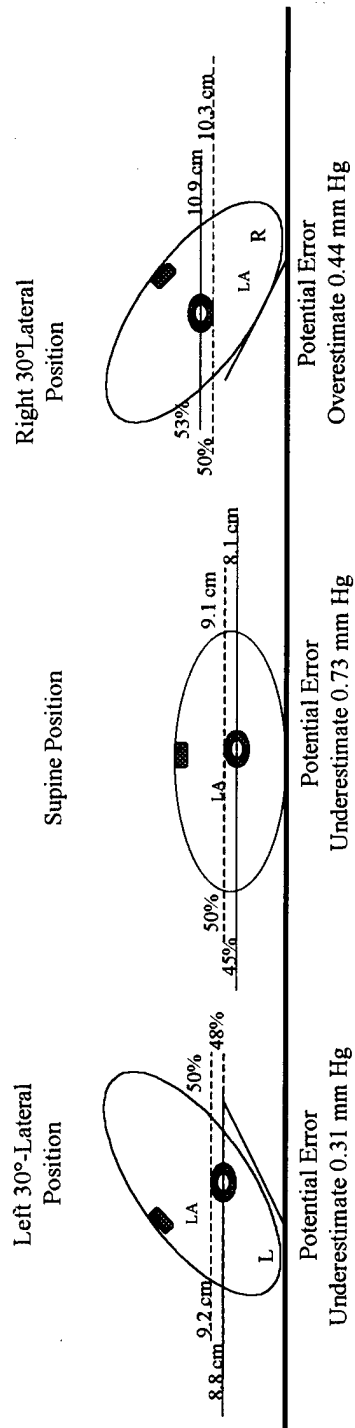


FIGURE 43. Variation in Reference Level. Using data from VanEtta's (1992) study regarding the exact location of the left atrium in comparison to the 50% reference point used in this study would result in the following pressure measurement errors: Supine: underestimate by 0.73 mm Hg, Right: overestimate pressure by 0.44 mm Hg, and Left: underestimate by 0.31 mm Hg.

For the left-lateral position, as summarized in Table 16, were on average 1.28 mm Hg greater than those measured in the supine position. Both the supine and left-lateral pressures were underestimated; thus, the actual pressures may have been higher than reported. Despite the increase in the absolute pressures, the average difference between the two pressures actually decreased to approximately 1.0 mm Hg. For example, a correction for the measurement error related to the different reference levels would result in a relative Supine pressure of 0.73 mm Hg ($0 \text{ mm Hg} + 0.73 \text{ mm Hg}$) and a relative average pressure in the Left-lateral position of 1.6 mm Hg ($1.28 \text{ mm Hg} + 0.32 \text{ mm Hg}$). Thus, the difference between the pressures measured in the left-lateral and supine positions would decrease to 0.87 mm Hg ($1.6 \text{ mm Hg} - 0.73 \text{ mm Hg}$) (Figure 44). Similar results were observed between the relative pressures in the Supine and Right-lateral positions (uncorrected difference = 0.69 mm Hg; corrected difference = -0.48 mm Hg), that is the supine pressures were greater than the right lateral pressure by 0.48 mm Hg.

Although the pressure difference related to the small inaccuracies in the reference levels may have contributed to the observed differences in the Left-lateral position it did not entirely explain the pressure change. However, the reference error could have accounted for the entire pressure difference observed in the Right-lateral position.

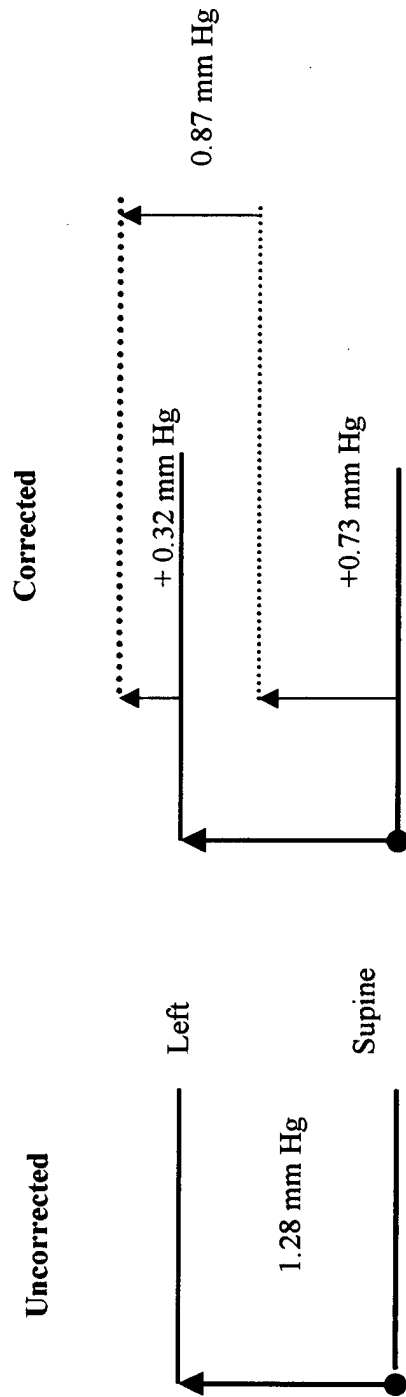


FIGURE 44. Potential Error due to Variation in Reference Level. Example of correction for measurement error introduced by using 50% reference point versus exact location of left atrium. In the left-lateral position, the uncorrected average difference between pressures the Supine and Left-Lateral position was 1.28 mm Hg. As demonstrated, if the Supine pressure is corrected by 0.73 mm Hg and the Left-lateral position by 0.32 mm Hg, results in a corrected pressure difference of 0.87 mm Hg. Thus measurement error contributed to the small pressure difference between Supine and Left-lateral measurements.

Transmural Compression

One mechanism that has been suggested as an explanation for the increase in PA pressures in the left lateral position is transmural compression of the left ventricle (Lange et al., 1988). The use of intracardiac pressure as an indicator of end-diastolic distending pressure is based on the assumption that, at end-expiration, the juxtacardiac pressure is zero. This assumption has been confirmed in animal models (Cabrera, Nakamura, Montague, & Cole, 1989; Santamore, Constantinescu, & Little, 1987) and post-cardiac surgery patients in the supine position (Guyton et al., 1987; Smiseth, Thompson, Ling, Robinson, & Miyagishima, 1996; Tyberg et al., 1986). However, in the 90-degree lateral position intracavitary pressures, particularly LV pressures, have been shown to increase without a change in end-diastolic volume (Beppu, Naito, Matsuhisa, Miyatake, & Nimura, 1990; Lange et al., 1988). The most probable explanation for these findings is a position-induced increase in juxtacardiac pressure. Thus, the measured intracavitary pressure would overestimate the true ventricular distending pressure (preload) (Janicki et al., 1996; Lange et al., 1988). Possible sources for a position-induced increase in juxtacardiac pressure include direct pressure from the lungs, increased intrathoracic pressure secondary to external chest-wall compression, decreased chest-wall movement as the result of splinting, active expiration or increased intraabdominal pressure (Cheatham et al., 1998; Schuster & Seeman, 1983). Any or all of these factors may apply to the subjects in this study. It is important to note that the pressure changes observed in

this study were not as large as those observed by Lange (4 to 7 mm Hg), with the most likely explanation being the lesser degree of turn (90- versus 30-degrees).

However, studies of the effect of PEEP on pericardial or mediastinal pressures would challenge direct cardiac compression as a causative mechanism. In both dogs and non-obese humans, mediastinal pressures measured in supine and lateral decubitus positions were similar (Marini, O'Quin, Culver, & Butler, 1982). Thus, as indicated by the direct mediastinal pressure measurements there does not appear to be an increase in juxtacardiac pressure in the lateral position. (It is important to note that the majority of subjects in this study exceeded ideal body weight).

Another issue that would challenge the transmural compression mechanism into question is that if the pressure changes were related to a more global factor such as increased intrathoracic pressure, the question remains: why would this global factor, have a differential effect in the left position? Based on individual results from this study, citing direct transmural compression as the explanation for the increase in pressures in the lateral position cannot be supported. For example, Subject # 5 demonstrated a 5 mm Hg decrease in PAED pressure in both right and left lateral positions compared with supine measurements. In addition, Subject # 28 demonstrated a 4 mm Hg pressure increase in the PAED pressure in the right-lateral position and a 2 mm Hg pressure decrease in the left lateral position relative to the Supine-1 measurement. Therefore, other possible mechanisms need to be considered.

Position-Induced Change in the Relationship of Intrathoracic Structures

Another possible explanation is a position-induced change in the relationship between the vena cava (RV filling reservoir) and the right ventricle (Figure 45). In animal models (dogs and pigs) in the supine position the inferior vena cava (IVC) is approximately 8 cm below the right ventricle. In the left-lateral position the IVC was above the right ventricle by 0.4 to 1.6 cm, while in the right lateral position the IVC was between 1.5 cm below to 3.2 cm above the right ventricle. In the left lateral position, there was an 8.4 cm difference (Supine: 8 cm below, Left: 0.4 cm above) in the height of the IVC relative to right in comparison to the supine relationship. This 8.4 cm difference is equivalent to a gravitational gradient of 6.2 mm Hg ($8.4 \text{ cm} \times 0.73 \text{ mm Hg/cm}$). In the right lateral position the height difference would result in an 8.2 mm Hg gravitational gradient. This gravitational gradient would facilitate RA and RV filling, with a resultant increase in RVED filling. The increased RV volume would explain the increase SV, CO and filling pressures observed in the lateral positions (Whitman & Verga, 1982).

A relatively similar relationship between the chambers of the heart and the major cardiac vessels exists in humans (Beppu et al., 1988; Beppu et al., 1990; El-Khoury, Bergman, & Montgomery, 1995). The lateral relationship between the right atrium and ventricle can be visualized on an anteroposterior (AP) chest radiograph by observing the path a PA catheter (Figure 5) or a transvenous pacing wire takes as it passes from the vena cava to the right atrium and into the right ventricle. In the AP view the right ventricle is medial to the right atrium and vena cava; thus, in a left lateral position the

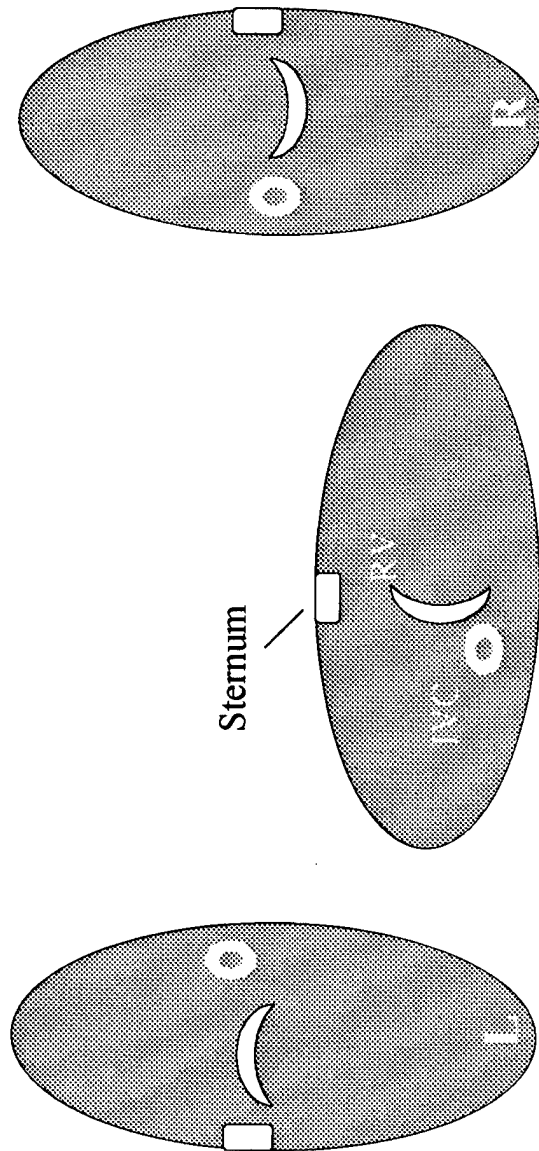


FIGURE 45. Schematic of the Change in the Relationship of Cardiac Structures in the Lateral Position. Based on a dog model (Nakao, et al., 1986), the change in the relative relationship of the inferior vena cava (IVC) and right ventricle (RV) in the left (L) and right (R) lateral decubitus positions may create a gravitational gradient that facilitates right ventricular filling. The position-induced increase in right ventricular filling may explain the increased right heart pressures and cardiac output associated with the lateral position.

right ventricle would be inferior to these structures. A lateral chest radiograph demonstrates that the vena cava is posterior to the right ventricle; thus, in both left and right lateral positions the vena cava would be at or above the level of the right ventricle (Shasby et al., 1981). Evaluation of the absolute relationship and distance between these structures may provide a means to measure the potential increase in the gravitational gradient between the vena cava and right ventricle, as there is limited displacement of cardiac structures in the lateral position (Kennedy et al., 1984; Paoletta, Dortman, Cronan, & Hasan, 1988). Thus, based on anatomy alone the mechanism of increased CO in the lateral position described by Nakao and colleagues is possible.

However, the mechanism proposed by Nakao has not been studied in humans, and results in the study by Lange (1988) would seem to challenge this interpretation. Lange and colleagues (1988) noted a significant ($p < .05$) increase in RV peak systolic (Supine: 29 ± 9 mm Hg; Left lateral: 36 ± 9 ; Right-lateral: 34 ± 9 mm Hg) and end-diastolic pressures (Supine: 8 ± 3 mm Hg; Left-lateral: 11 ± 3 mm Hg; Right-lateral: 11 ± 3 mm Hg) in both right and left lateral decubitus positions relative to supine pressures. While the LV end-diastolic pressure was also significantly increased ($p < .05$) in the left-lateral decubitus position relative to the supine pressure (Supine: 20 ± 7 mm Hg; Left-lateral: 24 ± 8 mm Hg; Right-lateral: 20 ± 7 mm Hg), the CO, SV, and HR were similar in all positions. Therefore, the increased end-diastolic pressure was not translated into an increase in SV, as would be predicted by the Frank-Starling Law of the Heart. One

possible explanation, as already discussed, is that the pressure increase was reflective of increased juxtacardiac pressure, and not increased volume.

Another possible explanation, which is a combination of the mechanisms identified by Nakao and Lange, is based on the coupling of the ventricles by the intraventricular septum and the pericardium. As a result of this coupling, the ventricles are interdependent; thus, any factor that affects the volume of one ventricle affects the pressure-volume curve of the other ventricle (Gilbert & Glantz, 1989; Raper & Sibbald, 1986; Smiseth, Kingma, Refsum, Smith, & Tyberg, 1985; Weber, Janicki, Shroff, & Fishman, 1981). Therefore, an increase in RV volume, as described by Nakao, may result in a decrease in the compliance of the left ventricle. The change in compliance may be mediated through a shift in or stiffening of the intraventricular septum or a change in pericardial pressure, with a resultant increase in LV pressure for any volume (Figure 7) (O'Quin & Marini, 1983; Raper & Sibbald, 1986).

Support for this mechanism comes from studies of the effect of lateral position on chamber pressures and volume in normal controls and individuals with a congenital absence of a left pericardium (Beppu et al., 1988; Beppu et al., 1990). While the data from each study are incomplete, it is possible to demonstrate that in both controls and subjects without a left pericardium, the major volume change occurs between the right and the supine position, with the largest volume change occurring in the right ventricle. For example, rotation from the supine to the 90-degree left lateral position resulted in increase of 3 ± 8 ml in the left ventricle and a 3 ± 3 ml increase in the right ventricle.

However, the LV pressure in the 90-degree left-lateral position was 7 ± 2 mm Hg higher than in the 90-degree right lateral position, while the RV pressure only increased 1 ± 2 mm Hg. In a second study, in normal controls in a "semilateral decubitus position" the RVED dimension increased approximately 10 mm when the subject was in the left (21 ± 2 mm) versus right lateral position (12 ± 3 mm) (Beppu et al., 1988). Combining the results of these two studies, there was an increase in RV volume without a change in RV pressure, and an increase in LV pressure without an increase in volume. The most likely explanation was an increase in RV chamber dimensions, which resulted in stiffening of the intraventricular septum, and subsequently decreased the LV compliance. The increase in RV dimensions may reflect chamber distortion due to lateral positioning, or as described by Nakao a position-induced increase in RV volume due to the change in the relative relationship between the IVC and the right ventricle.

It is interesting to note that in studies of the effect of lateral position on cardiac pressures and volume, the normal controls did not demonstrate any shift in intraventricular septum (Beppu et al., 1988; Beppu et al., 1990). Thus, the small volume change that occurs in the lateral position may be insufficient to cause a shift in the septum; however, stiffening of the septum cannot be ruled-out.

The effects of a change in intraventricular volume have been observed in patients even when the pericardium has been removed, although the effects on LV compliance would be greater if the pericardium was undisturbed (Janicki & Weber, 1980). Of interest to this study is the finding that pericardial constraint increases when the pericardium has

been cut and then reapproximated (Hammond, White, Bhargava, & Shabetai, 1992; Stokland, Miller, Lekven, & Ilebekk, 1980). It is difficult to determine the effect of pericardial constraint on the subjects in this study as the surgical practice is to reapproximate the “upper” portion of the pericardium to provide structural stability, and to leave the “lower” portion of the pericardium open to decrease the incidence of tamponade.

The other mechanism to consider is a position-induced increase in afterload. The increased RV peak systolic pressure may reflect a position-induced increase in pulmonary vascular resistance (change in lung volume, hypoxic vasoconstriction) and right heart afterload without a change in CO. The increased right heart pressures would indirectly affect left heart compliance and the pressure-volume relationship.

Effect of Lung Zones on PA Pressure Measurements

Previous research in animals and humans has demonstrated that in the presence of either increased alveolar pressure (PEEP) or decreased intravascular pressure (hemorrhage, diuresis) there is an increased conversion to non-Zone 3 conditions in the lung (alveolar pressure exceeds pulmonary arterial and/or pulmonary venous pressures) (Rajacich, Burchard, Hasan, & Singh, 1989; Shasby et al., 1981; Tooker et al., 1978). If a PA catheter is positioned such that pressures are obtained from a non-Zone 3 vascular bed, the PA pressure measurements no longer reflect left heart pressures, but instead reflect alveolar pressure. While Zone-1 conditions seldom occur in humans, Zone-2 conditions are more likely. In a Zone-2 condition when the balloon on the PA catheter is

deflated the pulmonary arterial pressure is greater than the alveolar pressure and a continuous column of fluid is maintained. However, when the balloon is inflated flow ceases through the vascular bed, resulting in collapse of the vascular bed. In this case the PAW pressure reflects alveolar pressure and may be artificially increased (O'Quin & Marini, 1983).

In the setting of decreased intravascular pressure or increased alveolar pressure the height of the PA catheter tip relative to the left atrium is important. When the PA catheter tip is at or below the level of the left atrium, the PAW pressure is an accurate indirect indicator of LA pressure, even in the presence of increased levels of PEEP and/or decreased intravascular pressures. However, when the catheter tip is above the left atrium, the PAW pressure may not accurately reflect LA pressure, particularly at PEEP greater than 15 cm H₂O (Neville et al., 1975; Rajacich et al., 1989; Shasby et al., 1981; Tooker et al., 1978).

It was not surprising that an alteration in the relationship between alveolar and intravascular pressures (as indicated by clinical criteria consistent with non-Zone 3 conditions) did not change during the lateral position portion of the study. A majority of the patients were spontaneously breathing and had increased intravascular pressures (mean PAW pressure = 13.6 ± 5.3 mm Hg). Therefore, the combination of increased airway pressure and decreased intravascular pressure did not occur in any of the subjects. The only patients of concern were those who demonstrated active-exhalation as determined by a progressive rise in PA pressures during exhalation.

End-Diastolic Pressure-Stroke Volume Relationship

Previous research has reported a small increase (250 to 500 ml) in CO in the left lateral position, which was maintained for as long as 15-minutes (Doering & Dracup, 1988; Whitman & Verga, 1982) to 30 minutes (Nakao et al., 1986) after position change. However, Lange (1988) observed no difference in CO in the 90-degree position. If there is an increase in CO, what is the mechanism of this change, and could the relationship between the change in preload and CO be quantified?

This study sought to evaluate one intrinsic component of CO: preload. Two of the other three intrinsic factors (afterload and HR) were controlled for, by implementing a stabilization period, such that the subject's HR and ABP returned to baseline before initiation of the PA pressure measurements. The one factor that could not be controlled was a change in contractility.

A possible mechanism for an increase in contractility is that position or position change, as a physiologic stressor, may increase systemic oxygen consumption ($\dot{V}O_2$) and sympathetic nervous system activation, and subsequently increase contractility. One indicator of the oxygen supply and demand balance is mixed venous oxygen saturation ($\bar{S}VO_2$). In cardiac surgery patients placed in a lateral position, $\bar{S}VO_2$ initially decreased, but returned to baseline within 5 minutes (Banasik, 1990; Banasik et al., 1987; Banasik & Emerson, 1996; Shively, 1988; Tidwell et al., 1990; Winslow et al., 1990).

As described by a derivation of the Fick equation:

$$\bar{S}VO_2 = SaO_2 - (\dot{V}O_2 \times Hb \times 1.38)$$

a decrease in $\bar{SvO}_2$ that is concurrent with an increase in CO ($\dot{Q}$) (assuming an unchanged SaO_2 or hemoglobin (Hb)) is indicative of an increase in oxygen consumption ($\dot{V}O_2$), or a supply/demand imbalance caused by an increased $\dot{V}O_2$ and an inadequate increase in CO. (Ledingham & Naguib, 1993) Although a physiologic stress-induced increase in contractility may explain the increase in CO in the lateral position, it remains unclear why this mechanism would result in the selective increase in CO and PA pressures in the left lateral position.

If the CO did increase in the left lateral position, and the increase in preload was the primary factor responsible for this change, would it be possible to quantify this relationship? Specific to this study, what effect would a 1 to 2 mm Hg increase in end-diastolic pressure have on SV and CO?

There is limited research regarding quantification of the relationship between a change end-diastolic pressure and changes in SV and CO. In dogs with normal cardiovascular function, the effect of a 1 mm Hg pressure change depends on the EDP. For example, when the PAED pressure was 3 to 8 mm Hg, a 1 mm Hg increase in pressure resulted in a 6.8% increase in ejection fraction. However, when the PAED pressure was 8 to 13 mm Hg, a 1 mm Hg pressure increase only resulted in a 1.4% increase in ejection fraction. It is important to recall that the change in ejection fraction or stroke volume can only be quantified if the EDV is known (Palacios, Powers, & Powell, 1985).

Raphael and colleagues (1977) quantified the relationship between a change in the PAED pressure and a change in the CI in patients with unstable angina or an acute

myocardial infarction (MI). The slope for the MI patients was $0.04 \text{ L/min/m}^2/\text{mm Hg}$, while the slope was $0.23 \text{ L/min/m}^2/\text{mm Hg}$ for the patients with unstable angina. Thus, a 1 mm Hg pressure change would result in a 40 to 230 ml/min/m^2 change in CI. Given the average body surface area of 2.0 m^2 observed in this study, this would be equivalent to an 80 to 460 ml/min change in CO, which is consistent with the findings of Whitman (1982) and Doering and Dracup (1988).

The findings from Raphael's study are more applicable to subjects in this study, in contrast to the data from Palacios in normal hearts, as there is consistent decrease in cardiac function in the post-cardiopulmonary bypass period (Breisblatt et al., 1990; Mangano, 1985; Phillips et al., 1983; Royster, 1993; Vinten-Johansen & Nakanishi, 1993). Although the changes reported by Raphael are smaller than those observed in normal hearts, the changes may reflect an extremely important mechanism for maintaining or increasing the SV in the failing heart (Holubarsch et al., 1996).

It is important to note that the changes in CO as a result of a change in EDP are only estimates, as there is an inconsistent relationship between changes in EDP and CO in post-cardiac surgery patients (i.e., a change in EDP may have little effect on SV or CO) (Ellis, Mangano, & Van Dyke, 1979; Gaasch, Levine, Quinones, & Alexander, 1976; Hansen et al., 1986). This poor relationship may reflect the post-cardiopulmonary bypass decrease in cardiac function manifested by a depression and flattening of the cardiac function curve.

Individual Characteristics

Previous research suggests that demographic and clinical variables, such as age, body temperature, cardiac index, cardiopulmonary bypass time, time elapsed since surgery, vasoactive medications, and mechanical ventilation, may effect a patient's hemodynamic response to position change (Doering & Dracup, 1988). In this study, there were no significant differences ($p > .05$) between those subjects who did and did not demonstrate clinically significant position-related pressure changes. However, very few subjects had prolonged cardiopulmonary bypass time (greater than 250 minutes), and all subjects were normothermic, at least four hours postoperative, and perhaps most importantly, hemodynamically stable. In addition, a majority of the subjects were receiving stable, relatively low doses of vasoactive medications, and only five subjects were receiving mechanical ventilation. Of the patients requiring mechanical ventilation, four of five had only 5 cm H₂O of PEEP and were in the process of being weaned for extubation. Thus the patients in this study were stable and were relatively homogeneous from a clinical perspective.

The only trends (non-significant) noted were that there were a greater number of older subjects who were status-post aortic valve replacement (AVR), who demonstrated clinically significant pressure changes. Four of the ten patients who underwent AVR demonstrated clinically significant pressure changes, which was greater, but not significantly different ($p > .05$) from that which would occur by chance. Other than age, there were no demographic or clinical characteristics, including baseline

cardiopulmonary indices, heart failure, vasoactive medications, or mechanical ventilation, that differentiated between the two groups.

It is not surprising that the AVR patients had an increased frequency of clinically significant changes, as the primary hemodynamic consequence of aortic stenosis is an increase in LVED pressure, which results in LV hypertrophy and decreased LV compliance. As demonstrated on the pressure-volume curve (Figure 5), a decrease in compliance increases the slope of the curve and shifts it left. Thus, any change in volume would be associated with a larger pressure change in comparison with a heart with normal compliance. The decrease in compliance may be particularly important if there is a position-related change in intraventricular volume or ventricular interaction resulting in a further decrease in LV compliance. It remains unclear why only certain patients in this subgroup demonstrated an increased response to position, as all of these patients had increased PA pressures, and would likely be on the steep portion of the pressure-volume curve.

The importance of age, in relation to the increased frequency of clinically significant changes in the older subgroup of patients who underwent AVR surgery, may simply reflect the long-term effects of aortic valve disease with more severe hypertrophy. If age were an isolated characteristic that affected the response to lateral position, it would be expected that all older subjects, regardless of surgical procedure, would demonstrate an altered response to position, which was not the case in this study.

Limitations of Study

The study of the effect of lateral position on the PAW pressure was limited by the small sample size. Only 17 of the 35 subjects (49%) had the PA catheter positioned such that a wedge could be obtained. These findings were not unexpected in a cardiac surgery population, given the practice of withdrawing the PA catheter into the main PA prior to the initiation of cardiopulmonary bypass. However, if this study were to be replicated in other patient populations the positioning of the PA catheter tip in the main PA could potentially increase the number of subjects who had catheters in a Zone 2 vascular bed; thus, negating the accuracy of the pressure measurements.

In addition, the study was performed on hemodynamically stable patients, the majority of whom were breathing spontaneously. In addition, while a majority of the patients had some degree of pleural effusion, none of the patients in the subject had clinically significant lung disease. Therefore, generalization of these results to other patient populations, particularly those receiving increased levels of PEEP or with decreased intravascular pressure, which would increase the likelihood of Zone 2 conditions, are limited.

The study was, on average, performed within 14 to 18 hours of surgery, and as such most often took place between 0800 and 1200. It was therefore impossible to assess for possible effect of circadian-based change in cardiovascular reactivity, or to determine if the pressure fluctuations or response to position change varied throughout a 24-hour

period. It is possible that these results reflect a response that typically occurs during this specific period of recovery; therefore, generalization to other time periods is not possible.

Finally, the interpretation of the analog data was accomplished by hand, which increased the probability of measurement error. Although there was high interrater reliability for all pressure measurements ($r = 0.99$), the data were read relative to a scale where misinterpretation by a single line on the scale would potentially result in a 1 mm Hg error (see Appendix C for an example of the data and the scale). If a 1 or 2 mm Hg error was made on one waveform and an error in the opposite direction made on another waveform, identification of a clinically significant change could occur simply as the result of measurement error. This was most problematic in the interpretation of the PAM pressure. The PAS and PAED pressures were identified in relation to the ECG; thus, a clear point could be identified, and interpretation was simplified. In the future, use of a method for computerized analysis will be imperative.

Areas for Further Study

This study was performed in a homogenous group of post-cardiac surgery patients. The subjects in this study were without significant lung disease, and had normal or increased intravascular pressures. Subjects with lung disease or severe cardiac dysfunction (cardiomyopathy) may respond differently to the lateral position. In addition, in patients with lung disease requiring ventilatory support the potential for Zone 2 effects on PA pressure measurement would increase. From a pragmatic perspective, given the rapid recovery of cardiac surgery patients most of these patients require

invasive monitoring for only a short period of time, and progress rapidly from bedrest to increased activity. Therefore, the patients that would benefit most from this research are those patients who require prolonged invasive monitoring along with therapeutic positioning, for example, patients with septic shock and acute respiratory distress syndrome or end-stage cardiomyopathy. It is in this more heterogeneous population where this study should be replicated.

Specification of Clinical Significance

There is no standard method for determining what absolute pressure change is indicative of clinical significance. Nemens and Woods (1979) used a combination of the mean pressure fluctuation and analysis of the accompanying frequency table of the absolute pressure changes to specify clinical significance. In the studies by Cason and colleagues (1990) and Moser and Woo (1996) mean pressures were reported, but the method used to determine clinical significance was not specified (Table 19). The method used for this study was the mean fluctuation plus two standard deviations for normally distributed data (PAS, PAED), or for data that were skewed (PAM, PAW) the absolute pressure fluctuation that included 95% of the sample. In studies of the fluctuation of $\bar{SvO}_2$, the highest and lowest absolute values were divided by a mean value in order to describe fluctuation as a percentage change (Noll et al., 1992). Finally, in studies where there was a relationship between an absolute value and the degree of variance (e.g., CO) a coefficient of variation was used to quantify the degree of fluctuation (Sasse et al., 1994).

In the latter two cases, clinical significance was defined as a change in $\text{S}\bar{\text{V}}\text{O}_2$ or CO that exceeded the described fluctuation.

The function of specification of a certain pressure change as indicative of clinically significant change is to serve as a screening tool. The tool is used to correctly identify those patients who demonstrate clinically significant changes and in fact have clinically significant changes (sensitivity), and to correctly identify those patients whose pressure changes, are not clinically significant (specificity). However, there is an inverse relationship between sensitivity and specificity. Therefore, a decision must be made whether it is more important to avoid missing patients who have clinically significant changes or to avoid misdiagnosing and potentially treating patients whose changes are not actually clinically significant (Hennekens & Buring, 1987).

The most frequently cited standard for describing clinical significance is based on the work of Nemens and Woods (1982). As noted, this standard, which is conservative, is based on the mean fluctuation; thus, any patient exceeding this "average" would be considered to have a clinically significant pressure change. In this case, the sensitivity of the standard is high (probability of being classified as "clinically significant" when a clinically significant change actually occurs); however, a large number of patients who do not have clinically significant changes will be incorrectly identified (lower specificity). In this case patients who need to be treated will be treated, but a number of patients who do not require treatment may also receive treatment.

In contrast, in this study, only the patients whose absolute pressure fluctuations exceed the mean ± 2 standard deviations were characterized as having clinically significant changes. In this case a greater proportion of patients whose pressure changes were not clinically significant would be correctly identified (higher specificity); however, an increased number of patients who did have clinically significant changes would be missed (lower sensitivity). Thus, the patients who did not require a response were correctly identified, but the number of patients who may have required, but did not receive treatment, would increase.

It is important to note that the actual pressure difference between the "less stringent" criterion specified by Nemens and Woods (1982) and the "more stringent" criterion in this study is only 1 mm Hg. Therefore, correction of the pressure change indicative of clinical significance to some halfway point between these two standards (e.g., mean ± 1 SD) would be clinically irrelevant.

The purpose of the specification of clinically significant changes can be addressed as follows: (1) The level of significance serves as a cue that there is a clinical condition that is potentially abnormal (increased sensitivity) and further assessment of the changes is warranted. (2) The level of significance provides support for the interpretation of change as indicative of a "true" clinically significant change, and further action may be required. For the purposes of this study it was important to be able to correctly characterize the clinically significant pressure changes; thus, the more stringent criteria with fewer false positives was appropriate.

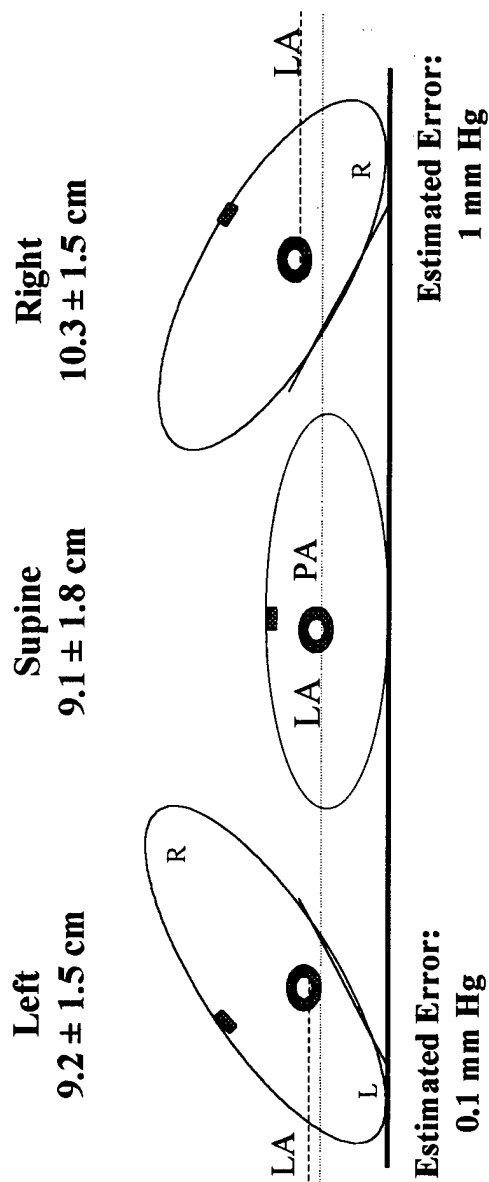
Perhaps a more important point is that in clinical practice an isolated pressure change would rarely be used as the sole indicator for a change in therapy. Characterization of a pressure change as indicative of clinical significance is only useful if this characterization is used in concert with other clinical data. That is, are there other signs and symptoms (e.g., increased chest tube output) or external events (e.g., suctioning) that would lead the clinician to believe that the observed changes were or were not clinically significant?

Reference Level

Is it possible to leave the transducer at a fixed position for measurement in both the supine and 30-degree lateral positions? In studies conducted by Whitman (1982) and Ross and Jones (1995) in post-cardiac surgery patients the transducer was not moved from the level of the supine phlebostatic axis when the patients were positioned in the 20- and 30-degree lateral positions, respectively. In both cases, although the pressures were slightly higher in the lateral position relative to the supine, the pressure differences were not clinically significant. In this study, if the transducer was not moved from the level of the supine phlebostatic axis the estimated measurement error would be: Supine-Right: 1 mm Hg and Supine-Left: 0.1 mm Hg. Therefore, from a clinically pragmatic perspective, it may be possible to leave the transducer at the level of the phlebostatic axis.

Combined Effect of Lateral Position and Backrest Elevation on PA Pressures

The next logical step for research related to the lateral position would be to study the combined effects of lateral position and backrest elevation on PA pressures. Positioning a patient in the lateral position with the backrest elevated would theoretically have less



PA = Phlebostatic Axis; LA = left atrium

FIGURE 46. Can the Supine Reference Be Used in the Lateral Position? The measurements at the top are the average distance the reference point was above the surface of the bed. The error associated with leaving the transducer at the height of the supine reference point would introduce a 0.1 to 1 mm Hg error in measurement. Therefore, from a clinically pragmatic perspective it may be possible to leave the transducer at the level of the supine-phlebostatic axis.

effect on intrathoracic pressure as the diaphragm would not be elevated by abdominal pressure. Given the research related to the decrease in functional residual capacity, and the potential intolerance to the supine position in some patients (e.g., increased intracranial pressure, pulmonary edema, gastro-esophageal reflux) studies related to the effect of the lateral position with this backrest elevation would have important clinical implications.

The study by Ross and Jones (1995), which is summarized in Appendix A, provides support for the measurement of PA pressures in the lateral position with the backrest elevated. The study evaluated the combined effects of a 30-degree lateral rotation with 20-degree backrest elevation. Further support for the stability of PA pressures measured with the backrest elevated comes from the findings of numerous studies of the effect of backrest elevation on PA pressures. In these studies there were no clinically significant differences in PA pressure measurements with a backrest elevation up to 60-degrees, as long as the phlebostatic axis was used as the LA reference point (Chulay & Miller, 1984; Clochesy, Hinshaw, & Otto, 1984; Laulive, 1982; Miller & Chulay, 1982; Prakash, Parmley, Dikshit, Forrester, & Swan, 1973; Retalliau, McGregor-Leding, & Woods, 1985; Woods, Grose, & Laurent-Bopp, 1982; Woods & Mansfield, 1976).

Steps to Clarify Mechanism of Action

The measurement of RA pressure in the supine and lateral positions would provide possible insight into the mechanism of the increase in pressure observed in the 30-degree left lateral position. In studies of the effect of PEEP on transmural pressure, several

studies (Smiseth et al., 1996; Tyberg et al., 1986) have supported the use of a change in the RA pressure as an indicator of a change in pericardial pressure. If the RA pressure is insensitive to the cause of increase in the pericardial pressure (PEEP versus position-induced compression) an increase in the RA relative to the supine position may provide some insight into the position-induced change in transmural pressure. However, as described by Nakao (1986) there may also be a position-induced increase in filling due to a shift in the relative relationship between the vena cava and the right ventricle. In addition, it is unclear if the relationship between pericardial and RA pressure is maintained in patients with right atrial or ventricular hypertrophy, or ischemic changes (Santamore et al., 1987).

Suctioning

Two subjects (#27, #29) required suctioning during the study. Despite delays of approximately 15 to 17 minutes, both subjects demonstrated continued elevation of all PA pressures. For Subject #27 PA and ABP returned to baseline approximately 25 minutes following suctioning; while the PA pressures in Subject #29 did not return to baseline during the study period (approximately 27 minutes). It is often the case in clinical practice that on-the-hour patients are repositioned and vital signs and hemodynamic pressure measurements are obtained. However, as a result of repositioning, patients who are intubated often require suctioning. If the observations in subject #27 and #29 are reflective of the prolonged effects of suctioning on PA pressures, even after HR and ABP have returned to baseline, further research is needed to describe

the temporal course in PA pressure changes after suctioning. In addition, description or identification of clinical indicators other than HR and ABP that may provide insight into when the patient has returned to baseline status is necessary.

Identification of PAEDP in Patients with RBBB Pattern

An incidental finding that requires further exploration is the identification of an appropriate ECG reference point for PAED pressure in the presence of a right bundle branch (RBBB) ECG pattern. Normally, PAED pressures are measured 0.08 second after the onset of the QRS complex (Lipp-Ziff & DT, 1991). However, in patients with a RBBB ECG pattern the QRS complex is widened. In addition there is a delay in RV systole that results in the traditionally measured PAED pressure underestimating left heart pressures (Kern, Donohue, Bach, & Aguirre, 1993).

Significance and Implications for Nursing Practice and Research

This study was the first large study to evaluate the effect of 30-degree lateral position on PA and PAW pressures using a validated LA reference point. As hypothesized, the use of this validated reference point negated the measurement error that contributed to statistically and clinically significant findings in previous studies of the effect of lateral position on PA and PAW pressures. Although there were statistically significant differences between PA pressures in the supine and 30-degree right and left lateral positions, only 25 (4.3%) of the 581 pairs analyzed were initially classified as clinically significant, and of those, only 12 (2.1%) were deemed directly attributable to position. Therefore, during the first 24 postoperative hours, in hemodynamically stable cardiac

surgery patients, PA and PAW pressures measured in the right and left 30-degree lateral positions may be considered equivalent to those obtained in the supine position.

However, individual response to position needs to be assessed.

This study validates the importance of using an angle-specific reference point to negate measurement error due to the inclusion of hydrostatic pressure. In addition, the results of this study are consistent with those related to the effect of 90-degree lateral position on PA pressure measurements, when an angle specific reference point was used (Guenther et al., 1987; Kennedy et al., 1984). As predicted, on average, in a less-severe degree of lateral rotation (30-degrees) using an angle-specific reference, there were, no clinically significant position-related PA pressure changes.

Based on the fluctuation of the PA pressures over a 15-minute period, pressure changes characterized as clinically significant were specified. In determining the clinical significance of a pressure change it is prudent to use the patient as his or her own control. However, as demonstrated by the potential variation in pressure fluctuation across patient populations and the numerous methods used to describe clinical significance, the first step necessary to comparing pressure fluctuation across populations is to standardize the terminology and methods used to define clinical significance. In addition use of similar sampling techniques is needed in order to control for the effect of measurement technique on the observed variance of the pressures. Performance of measurements on patients with a wide range of pressures would also be useful in order to determine if there is a relationship between absolute PA pressures and the degree of fluctuation. If the latter

case occurs, use of a coefficient of variation, which reports the standard deviation as a percentage of the absolute pressure, may be appropriate.

An interesting finding that requires further study is whether the amplitude of the rhythmic fluctuations is depressed in the cardiac surgery population. Further research using cosinor analysis to evaluate longer periods of time (Chen, Sharkey, Cornelissen, Holte, & Halberg, 1994; Cugini et al., 1993; Gibbs et al., 1989; Gibbs, Keegan, Wright, Fox, & Poole-Wilson, 1990; Ikram, Richards, Hamilton, & Nicholls, 1984; Levy et al., 1996; Richards et al., 1986; Richards, Ikram, Crozier, Nicholls, & Jans, 1990; Shasby et al., 1981), as well as measurement over 15- to 60-minute periods (the time period most often used in clinical practice between measurements) needs to be undertaken. This study will help to clarify if the decreased pressure fluctuation observed in this study was the result of a smaller sampling period or if it reflected a characteristic specific to this patient population.

Determination of the Effect of Therapeutic Positioning on Hemodynamic Indices

The American Association of Critical Care Nurses Consensus Conference on Research (Lindquist, et al, 1993) gave high priority to research related to the effect of therapeutic activities (such as positioning) on hemodynamic indices. From a research perspective, the results of this study may be useful in the reanalysis or reinterpretation of data. For example, in Doering and Dracup's (1988) study of the effect of the 45-degree lateral position on HR, SV and CO there was a wide degree of variability in SV and CO in response to the lateral position. However, the authors were unable to hypothesize about

possible mechanisms (e.g., change in preload or afterload) underlying the variability, as the effect of lateral position on the indices of preload (PA and PAW pressures) and afterload (ABP) were not known. The results of this study may provide insight into this mechanism, given that large PA pressure changes are not expected in the lateral position. If there are large PA position related-pressure changes, a change in preload may explain the changes in SV and CO. This analysis may subsequently lead to identification of the patients who demonstrated varying responses to lateral position.

Clinically results of this study may be useful in assessing the effect of therapeutic positioning on a patient, or in identifying those patients at risk for intolerance to lateral positioning. It is important to note that replication of this study is necessary before generalization can be made to a wide variety of critically ill patients. For example, in response to the placement of a patient into a 30-degree left-lateral position, a PAW pressure increase of 2 mm Hg would not be unexpected. However, a pressure change of 5 mm Hg, which is not an expected position-related pressure change, may be an indicator of intolerance to the therapeutic activity; necessitating a more in-depth assessment of the patients status.

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APPENDIX A

Effect of Lateral Position on PA Pressures

INVESTIGATOR, VARIABLES, SAMPLE	METHODS	FINDINGS	LIMITATIONS/IMPLICATIONS																																			
Murphy (1977) IV: Position change: supine, 90° lateral; stabilization period 1 and 5 minutes DV: PAD, PAWP SAMPLE: N = 8 critically ill patients	Reference Level: -Supine: 4th ICS/½ AP diameter -Lateral: 4th ICS/Midline Protocol repeated X 4 at 2 hour intervals Pressures measured at 1 and 5 minutes in supine position, and 1 and 5 minutes after position change	NS diff in PAD and PAWP between supine and lateral positions Percent of trials with clinically significant PAWP change (supine to lateral) < 4 mm Hg = 61%; 4 mm Hg = 16%; 5 mm Hg = 9%; 5-10 mm Hg = 9% -Differences between PAW (supine - lateral) clinically significant in 11 trials; mostly in patients with higher (> 20 mm Hg) PAW pressure. Stabilization Period Five minute period of stabilization after supine-lateral position change is required in patients with PAWP > 20 mm Hg. In patients with PAWP < 20 mm Hg, valid measurements can be made within 1 minute of position change. PAD-PAW pressure correlation: supine $r = 0.95$, $p < .01$ ($n = 7$); lateral: $r = 0.90$, $p < .01$ ($n = 7$). Correlation not affected by position (excluded one data set with large difference)	Small sample size Implications Stabilization at 1 minute sufficient if PAWP < 20 mm Hg; if PAWP > 20 mm Hg may require 5 minutes or more Increased fluctuation in pressures over time in patients with PAWP > 20 mm Hg																																			
Bryant, Kennedy (1982) IV: Position change: Supine with 0° backrest elevation; 90° right and left lateral decubitus DV: PAS, PAD, PAM, PAWP SAMPLE: N = 25 men	Reference Level: -Supine: 4th ICS/MAL; Lateral: 4th ICS/Midsternum	<table><thead><tr><th></th><th>Supine</th><th>Right</th><th>Left</th></tr></thead><tbody><tr><td>PAS</td><td>31 ± 5.6</td><td>31 ± 5.6</td><td>32 ± 5.6</td></tr><tr><td>PAD</td><td>15 ± 3.9</td><td>15 ± 3.6</td><td>15 ± 3.9</td></tr><tr><td>PAM</td><td>21 ± 4.5</td><td>20 ± 4.5</td><td>21 ± 4.5</td></tr><tr><td>PAWP</td><td>13 ± 3.6</td><td>13 ± 3.7</td><td>13 ± 3.6</td></tr></tbody></table> Maximum change PAM/PAWP 2 to 3 mm Hg		Supine	Right	Left	PAS	31 ± 5.6	31 ± 5.6	32 ± 5.6	PAD	15 ± 3.9	15 ± 3.6	15 ± 3.9	PAM	21 ± 4.5	20 ± 4.5	21 ± 4.5	PAWP	13 ± 3.6	13 ± 3.7	13 ± 3.6	On versus off ventilator? Stabilization time? How position ensured? Reading of waveforms?															
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Whitman (1982) IV: Position change: Supine with 20-degree backrest elevation; 20-degree right and left lateral DV: PAS, PAD, PAM, PAWP SAMPLE: N = 50 adult cardiac surgical patients	Reference Level: Supine: Phlebostatic axis (same for all positions) Random position: 6 sequences Stabilization: 15 minutes; Total collection time: 60-75 minutes Data analysis: End-expiration; pressures repeated X 2, with the mean of the two readings used	Group means PAS/PAWP: NS difference in any position ($p > .05$) PAD/RAP: Statistically significant difference ($p < .05$); Newman-Keuls: difference present in supine position - lower than right/left lateral ($p < .05$) Maximum difference: PAD < 1.34 mm Hg; RAP < 1.06 mm Hg	No comparison to baseline values How was position ensured? Sequence effect? Baseline values?																																			
Wild (1983) IV: Position change: Flat, supine, 30° right/left lateral DV: PAS, PAD, PAM, PAWP SAMPLE: N = 30 critically ill patients. Age 21-88, mean 54.5; 16 men, 14 women	Reference Level: -Supine: Phlebostatic axis -Lateral: 4th ICS/ Midsternum Stabilization: 3 minutes; analog data. Total time: 30 minutes Pressures read over one respiratory cycle Randomized position change (S-L-R-S; S-R-L-S)	Mean Pressures Differences Between Position Combinations (mm Hg) <table><thead><tr><th></th><th>S1-L</th><th>S1-R</th><th>R-S2</th><th>L-S2</th><th>L-R</th><th>S1-S2</th></tr></thead><tbody><tr><td>PAS</td><td>3.8</td><td>3.8</td><td>3.6</td><td>3.6</td><td>0</td><td>0.2</td></tr><tr><td>PAM</td><td>3.7</td><td>4.4</td><td>4.3</td><td>3.6</td><td>0.7</td><td>0.1</td></tr><tr><td>PAD</td><td>3.5</td><td>4.4</td><td>4.1</td><td>3.2</td><td>0.9</td><td>0.3</td></tr><tr><td>PAW</td><td>4.9</td><td>4.9</td><td>4.3</td><td>4.3</td><td>0</td><td>0.6</td></tr></tbody></table> # Subjects Exceeding Expected Range of Fluctuation (PAWP > 7 mm Hg): S1-L: 12 (40%); S1-R: 8 (27%); R-S2: 6 (20%); L-S2: 9 (30%); L-R: 4 (13%); S1-S2: 4 (13%)		S1-L	S1-R	R-S2	L-S2	L-R	S1-S2	PAS	3.8	3.8	3.6	3.6	0	0.2	PAM	3.7	4.4	4.3	3.6	0.7	0.1	PAD	3.5	4.4	4.1	3.2	0.9	0.3	PAW	4.9	4.9	4.3	4.3	0	0.6	Limitations: Pressures read over one respiratory cycle Implications: Clinically significant changes PA and PAW pressures noted in 93% of the patients. Mean differences within were all within expected fluctuations; however, wide individual variation existed.
	S1-L	S1-R	R-S2	L-S2	L-R	S1-S2																																
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Kennedy, Bryant, Crawford (1984) IV: Position change: Supine, Left/Right lateral decubitus DV: PAS, PAD, PAM, PAWP SAMPLE: N = 25 patients with cardiac disease. Age: 31-74, mean 54.	Reference Level: -Supine: 4th ICS/MAL -Lateral: 4th ICS/Midsternum Elapsed time after position change: 3 minutes No external support Analog data read over one respiratory cycle	<table><tr><td>Supine</td><td>PAS ($\pm$ SD)</td><td>PAD ($\pm$ SD)</td><td>PAM ($\pm$ SD)</td><td>PAWP ($\pm$ SD)</td></tr><tr><td>Right</td><td>34.6 $\pm$ 13</td><td>17.7 $\pm$ 9</td><td>23.7 $\pm$ 10</td><td>15.7 $\pm$ 9</td></tr><tr><td>Left</td><td>34.8 $\pm$ 14</td><td>17.8 $\pm$ 9</td><td>23.5 $\pm$ 10</td><td>16.0 $\pm$ 9</td></tr><tr><td></td><td>34.9 $\pm$ 14</td><td>17.6 $\pm$ 10</td><td>23.8 $\pm$ 10</td><td>16.4 $\pm$ 9</td></tr><tr><td>Range</td><td></td><td>Supine (mean)</td><td>Left (mean)</td><td>Right (mean)</td></tr><tr><td>PAS</td><td>12-70</td><td>34.6 $\pm$ 13</td><td>34.9 $\pm$ 14</td><td>34.8 $\pm$ 14</td></tr><tr><td>PAD</td><td>6-37</td><td>17.7 $\pm$ 9</td><td>17.6 $\pm$ 10</td><td>17.8 $\pm$ 9</td></tr><tr><td>PAM</td><td>8-42</td><td>23.7 $\pm$ 10</td><td>23.8 $\pm$ 10</td><td>23.5 $\pm$ 10</td></tr><tr><td>PAWP</td><td>4-35</td><td>15.7 $\pm$ 9</td><td>16.4 $\pm$ 9</td><td>16.0 $\pm$ 9</td></tr><tr><td>Positio</td><td>PAD</td><td>PAWP</td><td>Mean Diff</td><td></td></tr><tr><td>n</td><td>17.7</td><td>15.7</td><td>2.0</td><td></td></tr><tr><td>Supine</td><td>17.8</td><td>16.0</td><td>1.8</td><td></td></tr><tr><td>Right</td><td>17.6</td><td>16.4</td><td>1.2</td><td></td></tr><tr><td>Left</td><td></td><td></td><td></td><td></td></tr></table>	Supine	PAS ($\pm$ SD)	PAD ($\pm$ SD)	PAM ($\pm$ SD)	PAWP ($\pm$ SD)	Right	34.6 $\pm$ 13	17.7 $\pm$ 9	23.7 $\pm$ 10	15.7 $\pm$ 9	Left	34.8 $\pm$ 14	17.8 $\pm$ 9	23.5 $\pm$ 10	16.0 $\pm$ 9		34.9 $\pm$ 14	17.6 $\pm$ 10	23.8 $\pm$ 10	16.4 $\pm$ 9	Range		Supine (mean)	Left (mean)	Right (mean)	PAS	12-70	34.6 $\pm$ 13	34.9 $\pm$ 14	34.8 $\pm$ 14	PAD	6-37	17.7 $\pm$ 9	17.6 $\pm$ 10	17.8 $\pm$ 9	PAM	8-42	23.7 $\pm$ 10	23.8 $\pm$ 10	23.5 $\pm$ 10	PAWP	4-35	15.7 $\pm$ 9	16.4 $\pm$ 9	16.0 $\pm$ 9	Positio	PAD	PAWP	Mean Diff		n	17.7	15.7	2.0		Supine	17.8	16.0	1.8		Right	17.6	16.4	1.2		Left					Implication: Accurate measurements of PA and PAWP can be obtained in the lateral decubitus position as long as the transducer is leveled with the left atrium No external support
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Keating, Boylard, Eichler, Reed (1986) IV: Position change: Supine with 0° backrest; 45° right/left lateral DV: PA/PAWP SAMPLE: N = 20 critically ill patients with stable HR and BP	Reference Level: Supine: Phlebostatic axis Lateral: 4th ICS/Midsternal line Stabilization: 5 minutes Pressures averaged over one respiratory cycle	<table><tr><td>Supine-1</td><td>PAS</td><td>PAD</td><td>PAWP</td></tr><tr><td>Right</td><td>39.70</td><td>21.25</td><td>17.16</td></tr><tr><td></td><td>33.00</td><td>13.85</td><td>11/10</td></tr><tr><td>Supine-2</td><td>39.90</td><td>20.65</td><td>17/16</td></tr><tr><td>Left</td><td>34.80</td><td>17.16</td><td>10.79</td></tr></table> No significant change S1 to S2 or S-Lateral PAS (p = 0.785), Significant change S1-Lateral PAD (p = .0007); S1-Lateral PAWP (p = .0012). Individual differences: PAS: 3-17 (mean 10.3), PAD: 3-20 (mean 9.45), PAWP: 5-14 (mean 9.84)	Supine-1	PAS	PAD	PAWP	Right	39.70	21.25	17.16		33.00	13.85	11/10	Supine-2	39.90	20.65	17/16	Left	34.80	17.16	10.79	No standardization of position (positioned with pillows) Pressures read over one respiratory cycle																																																		
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Guenther, Kay, Cheng, Lauer (1987) IV: Position change: Supine and prone with 0-degree backrest, 90-degree lateral DV: HR, MAP, CVP, PAWP, CI, SVR, PVR Group 1: n = 12 s/p laminectomy patients Group 2: n = 12 critically ill adults	Reference Level: Supine/Prone: 4 th ICS/MAL Lateral: 4 th ICS, midsternum	<table><tr><td></td><td>Group 1</td><td></td><td>Group 2</td><td></td></tr><tr><td></td><td>Supine</td><td>Prone</td><td>Supine</td><td>Right</td><td>Left</td></tr><tr><td>HR</td><td>90 $\pm$ 5</td><td>76 $\pm$ 5</td><td>73 $\pm$ 6</td><td>84 $\pm$ 4</td><td>90 $\pm$ 3</td></tr><tr><td>MAP</td><td>90 $\pm$ 6</td><td>109 $\pm$ 6</td><td>103 $\pm$ 5</td><td>90 $\pm$ 6</td><td>80 $\pm$ 6</td></tr><tr><td>CVP</td><td>7 $\pm$ 1</td><td>8 $\pm$ 1</td><td>6 $\pm$ 1</td><td>6 $\pm$ 1</td><td>5 $\pm$ 1</td></tr><tr><td>PAWP</td><td>10 $\pm$ 2</td><td>16 $\pm$ 1</td><td>10 $\pm$ 1</td><td>11 $\pm$ 2</td><td>10 $\pm$ 2</td></tr><tr><td>CI</td><td>4 $\pm$ 0.5</td><td>3.2 $\pm$ 0.3</td><td>3.0 $\pm$ 0.2</td><td>3.9 $\pm$ 0.3</td><td>4.0 $\pm$ 0.5</td></tr><tr><td>SVR</td><td>990 $\pm$ 130</td><td>1489 $\pm$ 186</td><td>1499 $\pm$ 177</td><td>994 $\pm$ 139</td><td>976 $\pm$ 143</td></tr><tr><td>PVR</td><td>123 $\pm$ 17</td><td>147 $\pm$ 25</td><td>134 $\pm$ 24</td><td>134 $\pm$ 21</td><td>109 $\pm$ 20</td></tr></table>		Group 1		Group 2			Supine	Prone	Supine	Right	Left	HR	90 $\pm$ 5	76 $\pm$ 5	73 $\pm$ 6	84 $\pm$ 4	90 $\pm$ 3	MAP	90 $\pm$ 6	109 $\pm$ 6	103 $\pm$ 5	90 $\pm$ 6	80 $\pm$ 6	CVP	7 $\pm$ 1	8 $\pm$ 1	6 $\pm$ 1	6 $\pm$ 1	5 $\pm$ 1	PAWP	10 $\pm$ 2	16 $\pm$ 1	10 $\pm$ 1	11 $\pm$ 2	10 $\pm$ 2	CI	4 $\pm$ 0.5	3.2 $\pm$ 0.3	3.0 $\pm$ 0.2	3.9 $\pm$ 0.3	4.0 $\pm$ 0.5	SVR	990 $\pm$ 130	1489 $\pm$ 186	1499 $\pm$ 177	994 $\pm$ 139	976 $\pm$ 143	PVR	123 $\pm$ 17	147 $\pm$ 25	134 $\pm$ 24	134 $\pm$ 21	109 $\pm$ 20	Stabilization period? When read in respiratory cycle? BP - No discussion of reference No significant effects noted; however, CI with 900-1000 ml change from supine to lateral BP/HR varied between positions; hemodynamically unstable																	
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Lange, Katz, McBride, Moore, Hillis, (1988) IV: Position change: Supine, Left/Right lateral decubitus DV: HR, CO, SV, LV Peak Systolic Pressure, LVEDP, RV peak systolic pressure, RVEDP SAMPLE: N = 24 cardiac patients undergoing elective cardiac catheterization. Age 25-71. Group I (n = 17): 11 men, 6 women. Age 48 ± 12 years Group II (n = 7): 4 men, 3 women. Age 54 ± 12	Micromanometer tipped catheters Group I: PA Cath, micromanometer tipped catheters in right/left ventricles. Group II: Micromanometer tipped catheter in left ventricle; ventriculography.	Group I (n = 17) Left Lateral (mean ± 1 SD) 77 ± 15 5 ± 1.01 67 ± 18 140 ± 20 24 ± 8* 36 ± 9* 11 ± 3 Supine (mean ± 1 SD) 79 ± 16 5.06 ± 1.10 67 ± 18 136 ± 17 20 ± 7 29 ± 9* 8 ± 3 RV Peak Systolic LVEDP RV Peak Systolic RVEDP * p < .05 Group 2 (n = 7) Left Lateral 27 ± 5 62 ± ml/m² Supine 20 ± 3 66 ± 13 ml/m² LVEDV	Right Lateral (mean ± 1 SD) 79 ± 15 5.23 ± 1.37 67 ± 18 136 ± 17 20 ± 7 34 ± 9* 11 ± 3	Small sample size per group Limited data. Mean data only
Osika (1989) IV: Position Change: Supine, lateral position with backrest elevation of 20°, 30, or 45-degrees backrest elevation DV: PA pressures SAMPLE: N = 21 critically ill MICU patients	Reference level: Not specified Degree of lateral rotation: Not specified Protocol: Supine, lateral with backrest at 20°, 30°, 45-degrees (3 data sets) Clinically significant: PAS/PAD/PAM > 5 mm Hg; PAWP > 4 mm Hg	Clinically significant differences 71% at lateral/20-degrees backrest elevation 57% at lateral/30-degrees backrest elevation 76% at lateral/45-degrees backrest elevation		Limitations: Minimal discussion related to methods
Cason, Lambert, Holland, & Huntsman (1990) IV: Position change: Supine, 30-degree right/left lateral with 0-degree backrest elevation; supine, 30-degree right/left lateral with 30-degree backrest elevation DV: PAS, PAD, PAM SAMPLE: N = 16 critically ill patients. Age 62.5 (SD 10.64).	Reference Level -Supine: Phlebostatic Axis -Lateral: 4th ICS/ Midsternum Stabilization period: 5 minutes Protocol: Supine-Lateral-Lateral-Supine-Supine-30-degrees-Lateral 30-degrees-Lateral 30-degrees Pressures read from bedside monitor Compared each subjects normal fluctuation with pressure change associated with position change	Pressure PAS Position Supine Right Lateral Left Lateral Supine Right Lateral Left Lateral Supine Right Lateral Left Lateral Pressure PAS/PAD	0° Backrest 32.6 (9.02) 30.06 (9.79) 28.56 (11.80) 12.56 (5.85) 9.37 (6.55) 7.81 (7.20) 20.44 (7.2) 16.99 (8.59) 16.25 (9.55) Backrest Elevation 0° 30° 0° 30° % Fluctuation ≥ Baseline 56.25% / 62.5% 25% / 56.3% 50% / 56.3% 31.5% / 43.7%	Limitations: Digital pressures Small sample size Not specified when read during respiratory cycle Implications: Statistically and clinically significant changes in pressures in the lateral position with backrest at 0- and 30-degrees

Groom, Frisch, Elliott (1990) IV: Position change: Supine, 45° right/left lateral. Lateral reference point. DV: PAS, PAD, PAWP, PAM SAMPLE: N = 60 stable ICU patients. SICU patients (n = 30); MICU patients (n = 30).	Reference Level: -Supine: 4th ICS/Midaxillary line -Lateral: SICU - 4th ICS/Dependent midaxillary line; MICU: 4th ICS/Sternum Measurements obtained every 2 hours Cross over of reference points (SICU n = 10; MICU, n = 10).	SICU: NS difference in PAS, PAD, PAM, PAWP in right/left lateral position. MICU: Statistically significant changes in PAS, PAD, PAM, PAWP when turned to either side (S-L, $p < .001$; S-R, $p < .01$). Lateral pressures systematically lower than supine. Clinically significant changes (based on Nemens & Woods) -MICU - 72%; SICU 7% Cross-over of reference point. SICU: NS difference in pressures; decrease correlation: Left Lateral: $r = .888$ to $r = .77$; Right Lateral: $r = .877$ to $r = .64$ MICU: Significant difference in pressures; increase correlation Left lateral: $r = .598$ to $r = .66$; Right lateral: $r = .530$ to $r = .705$	Limitations: No interrater reliability. No standardization of position. Pressures obtained 2 hours apart. Variable backrest elevation. Implications: Author's recommend use of 4th ICS/MAL as reference for 45-degree lateral position. Variance in sidelying positions consistently greater in MICU patients (other pathology?)
Briones, Dickenson, Biebertz (1991) IV: Position change: Supine, 30° backrest, 45° left/right lateral with 30° backrest DV: PAS, PAD, PAM, PAWP, SvO₂ SAMPLE: N = 80 critically ill patients (SBP > 90 mm Hg)	Reference Level -Supine: Phlebostatic axis -Lateral: 4th ICS/midsternum Stabilization: 15 minutes	NS differences ($p > .05$) in all hemodynamic measurements and SvO₂	Limitations: Abstract - no individual or group data provided. Limited methods Implications: PA/PAW pressures may be obtained in 45° lateral position with 30° backrest if 4th ICS/midsternum used as reference. Recommendations limited by availability of data.

Duke (1994) IV: Position change: Supine, 30° right/left lateral DV: PAS, PAD, PAM, PAWP SAMPLE: N = 6 critically ill adults (trauma)	Reference Level: -Supine: 4th ICS/ ½ anterior-posterior diameter; Lateral: ½ distance from left sternal border to surface of bed End-expiration, analog waveform, 3 minute stabilization Protocol: S-Lat-S-Lat-S (5 measurements)	PAS S-R S-L S1-S5 PAD S-R S-L S1-S5 PAM S-R S-L S1-S5 PAWP S-R S-L S1-S5	Mean Difference 0.33 2.33 -2.33 1.00 0.83 -2.50 -0.66 2.16 0.66 -0.83 1.16 -0.16	SD Mean Difference 4.59 3.26 7.11 3.52 4.16 6.47 3.07 4.91 3.67 3.06 2.04 1.72	SE Mean Difference 1.87 1.33 2.90 1.43 1.70 2.64 1.25 2.00 1.49 1.24 0.83 0.70	Small sample size Questionable data interpretation (PAWP > PAD) Implications -Trend toward a greater increase in pressures in the 30° left lateral position -No consistent trends in pressures for each individual, e.g., an increase in PAD was not always accompanied by an increased PAWP
Ross & Jones (1995) IV: Position: Supine, 20° backrest, 30° right/left lateral with 20° backrest DV: PAS, PAD SAMPLE: N = 40 hemodynamically stable post-cardiac surgery patients (31 men, 9 women). Age 38-71. Ventilator (n = 21), CPAP (n = 4), nebulizer (n = 14), oxygen via nasal cannula (n = 1)	Reference Level: Supine: Phlebostatic axis Lateral: Level of phlebostatic axis, right phlebostatic axis, midsternum PA catheter position verified by CXR, PA waveform Stabilization: 5 minutes	PAS Transducer Reference Supine Phlebostatic Axis Right Phlebostatic Axis Midsternum PAD Transducer Reference Supine Phlebostatic Axis Right Phlebostatic Axis Midsternum Clinically Significant Changes Supine Phlebostatic Axis Right Phlebostatic Axis Midsternum	mm Hg (SD) 30° R Lateral 33.31 (9.78)* 38.31 (9.92)*+ 24.47 (9.85)*+ 30° R Lateral 18.87 (4.57)* 24.04 (4.88)*+ 10.08 (4.60)*+ PAS (> 5 mm Hg) 0% 69% 74%	mm Hg (SD) 30° Left Lateral 34.16 (9.50)* 25.96 (9.77)*+ 25.16 (9.64)*+ 30° Left Lateral 19.46 (4.54)* 10.99 (4.62)*+ 10.28 (4.57)*+ PAD (> 4 mm Hg) 0% 91% 98%	Limitations: Individual data? Individual fluctuations PEEP? Normal PAD Implications Study highlights effect of various reference points on measured pressures.	

Aitken (1995) IV: Position change: Supine-60° lateral with 45° backrest on/off mechanical ventilation DV: PAS, PAD, PAM, PAWP at 2 and 10 minutes after position change SAMPLE: N = 28 Cardiothoracic ICU patients	Sequence: Supine-Lateral-Lateral Data Analysis (A) = Spontaneously breathing; (B) = mechanical ventilation, Digital data except with respiratory swing Reference: Supine - Phlebostatic axis, Lateral: Mathematical midpoint (based on pressure) of thorax	Pressure (A)	Lateral Position	Supine	2 Minutes (mm Hg)	10 Minutes (mm Hg)	Limitations
		PAS	Left	34.36	38.75†	35.57*	-Lateral reference point not validated -Position maintained with pillows No data related to pressure change over period of study (supine-supine) Implications Spontaneously Breathing Patients: Significant change in all PA pressures at 2 minutes after position change. Resolved by 10 minutes. Mechanical Ventilation: Statistically significant differences between supine-lateral at 10 minutes for PAS, PAD, PAM. PAWP stable after 2 minutes. Improvement in accuracy (decreased difference) of pressures from catheter in right lung in right lateral position (catheter in dependent lung). Results remained significantly different (p < .05). Supine-Lateral pressure changes may reflect effect of variable reference level.
		PAD	Left	16.39	14.57†	14.57*	
		PAM	Left	23.04	23.32†	23.32*	
		PAWP	Left	16.25	14.57†	14.57*	
		PAS	Right	38.86	36.86†	34.75*	
		PAD	Right	16.07	16.07	15.11	
		PAM	Right	24.54	24.54†	23.21*	
		PAWP	Right	16.82	16.82†	14.93*	
		Pressure (B)	Lateral Position	Supine	2 Minutes (mm Hg)	10 Minutes (mm Hg)	
		PAS	Left	29.64	32.29†	29.75*	
		PAD	Left	14.57	14.57	14.14	
		PAM	Left	21.64	22.43†	20.93*	
		PAWP	Left	11.07	10.61†	9.75*	
		PAS	Right	30.61	32.14	29.18**	
		PAD	Right	15.00	15.29	13.93**†	
		PAM	Right	21.29	22.54†	20.36**†	
		PAWP	Right	10.43	10.79	9.71	

* p < .05 Supine 2-10 minutes; † Supine-2 minutes; ‡ Supine-10 minutes

Abbreviations Used in Appendix A

BP.	Blood pressure	PAM.	Pulmonary artery mean
CI.	Cardiac index	PAS	Pulmonary artery systolic
CPAP	Continuous positive airway pressure	PAWP	Pulmonary artery wedge pressure
CVP.	Central venous pressure	PEEP	Positive end-expiratory pressure
DV.	Dependent variable	PVR	Pulmonary vascular resistance
HR.	Heart rate	S-2	Supine-2 position
ICS.	Intercostal Space	R.	Right position
IV.	Independent variable	RAP.	Right atrial pressure
L	Left position	RV	Right ventricular
LV.	Left ventricular	RVEDP	Right ventricular end-diastolic pressure
LVEDP.	Left ventricular end-diastolic pressure	S1.	Supine-1 position
MAL.	Midaxillary line	S-2	Supine-2 position
MAP.	Mean arterial pressure	SBP.	Systolic blood pressure
MICU.	Medical intensive care unit	SICU.	Surgical intensive care unit
NS.	Nonsignificant	SV.	Stroke volume
PAD.	Pulmonary artery diastolic	SvO₂.	Mixed venous oxygen saturation
		SVR	Systemic vascular resistance

APPENDIX B

Data Collection Sheet

CODE NUMBER □□-□□

Date ____/____/19__

PATIENT DEMOGRAPHICS

1. Age _____	2. Sex ____ (Female = 1; Male = 2)
3. Height _____ (inches)	4. Weight _____ (kg/lbs)
5. BSA _____	
6. Race _____ (1 = American Indian/Alaskan Native; 2 = Asian/Pacific Islander; 3 = Black; 4 = Caucasian; 5 = Hispanic)	

INCLUSION/EXCLUSION CRITERIA

Inclusion Criteria	Exclusion Criteria
☐ PA Catheter ☐ Read/speak English ☐ Hemodynamically stable (SBP > 90 mm Hg, HR < 130 beats per minute, $\text{S}\bar{\text{V}}\text{O}_2 > 60\%$, $\text{SaO}_2 > 90\%$) ☐ Chest tube drainage < 100 ml/hr over previous 3 hrs ☐ More than four-hours postoperative ☐ No fluid boluses > 250 ml/hr within previous hour ☐ No alteration in inotropic or vasoactive infusions within previous hour ☐ No IV diuretics or morphine within previous hour ☐ PA Temperature $\geq 36.0^\circ\text{C}$	☐ No PA Catheter ☐ Inability to tolerate 0°-flat backrest position ☐ Clinically unstable (systolic BP < 90 mm Hg; $\text{SaO}_2 < 90\%$; $\text{S}\bar{\text{V}}\text{O}_2 < 60\%$; baseline HR > 130 bpm) ☐ Mitral stenosis or left atrial myxoma ☐ Receiving hemodialysis ☐ Chest wall deformity (kyphoscoliosis) ☐ Kinetic therapy (Roto-Rest) ☐ Intra-aortic balloon pump ☐ Subject request: Reason _____ ☐ PI/RN decision: Reason _____

DIAGNOSES

 Unilateral Lung Disease: Yes ____ No ____ Side _____
 Autonomic Neuropathy: Yes ____ No ____
 Surgery Yes ____ No ____
 Type _____
 #Hours since Surgery _____
 #Hours on bypass _____
 EBL _____
 # Days on bedrest _____

VITAL SIGNS

Usual Bedtime _____ Usual Awakening _____ (Midsleep = _____)

Temperature _____ (C/F)

Heart Rate _____ (bpm)

Rhythm

Blood Pressure _____ (S/D/M)

Source _____

Cardiac Output _____ (L/min)

Cardiac Index _____ (L/min/m²)

Right Atrial Pressure _____ SVR/SVRI _____

RR _____ (bpm)

Fio2 _____ Mode _____

 SaO_2 _____
$$S\bar{v}O_2 \underline{\hspace{1cm}}$$

Chest Tube Output X 3° _____ (exclusion > 100 ml/hr X 3°)

Mechanical Ventilation

Mode _____

Rate

Vt _____

PEEP/CPAP

PIP _____

IV FLUIDS/RATES

VASOACTIVE DRUGS/DOSE/RATE

TOTAL IVF RATE _____

MEDICATIONS

Route

Dose

Last Dose

PA CATHETER

Catheter Size _____

Brand/Type _____

Insertion Site _____

Tip Location (CXR) _____ (Right vs Left PA)

_____ (Vertical distance from carina/mediastinum)

PRESSURE SYSTEM

Brand _____ Monitor Brand _____

Length _____

Stopcocks _____

DYNAMIC RESPONSE CHARACTERISTICS

Fn _____ (Hz) Heart Rate (Hz) _____

A2/A1 _____

Characterization _____

(Include analog record)

Corrective Actions _____

PRESSURE FLUCTUATIONS

Position Found In _____

Backrest Elevation _____ (degrees)

Start Time _____

Stop Time _____

	M1	M2	M3	M4
PAS				
PAM				
PAEDP				
PAWP				

POSITION EFFECT

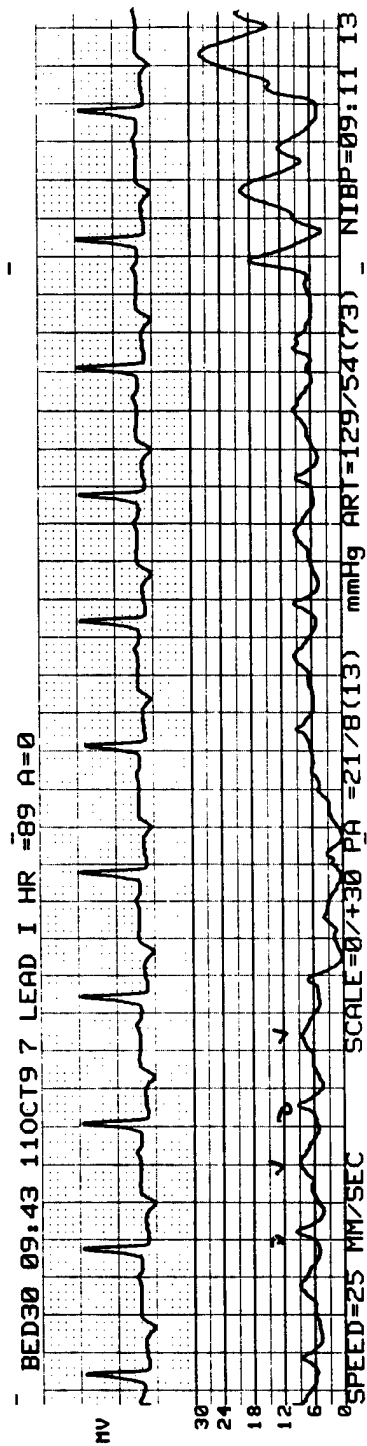
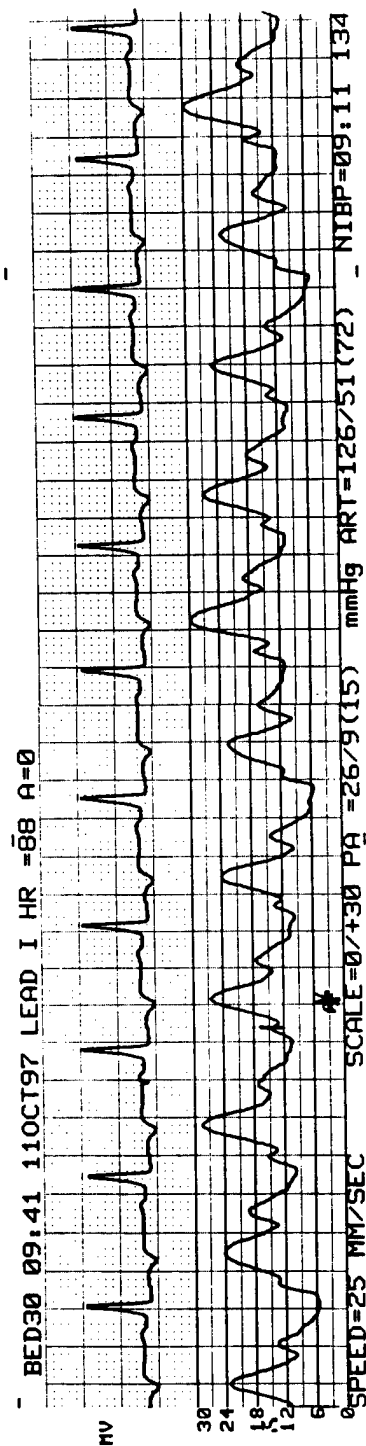
Start Time _____

Completion Time _____

Group A					Group B				
	S-1	Right	Left	S-2		S-1	Left	Right	S-2
PAS					PAS				
PAM					PAM				
PAED					PAED				
PAWP					PAWP				
Balloon Volume					Balloon Volume				
PAWP Damped ?					PAWP Damped ?				
Δ PAWP/ Δ PA Ratio					Δ PAWP/ Δ PA Ratio				
Stabilization Time					Stabilization Time				
Reference Level (distance from surface of bed)					Reference Level (distance from surface of bed)				

APPENDIX C

Examples of Analog Data (PA and PAW Pressures)



APPENDIX D

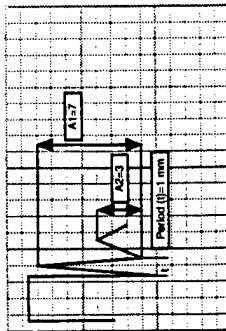
Dynamic Response Characteristics

From: Bridges, E., & Middleton, R. (1997). Direct arterial vs oscillometric monitoring of blood pressure: Stop comparing and pick one (a decision making algorithm). Critical Care Nurse, 17(3), 58-72. With permission.

Decision Making Algorithm

How to Assess Dynamic Response Characteristics

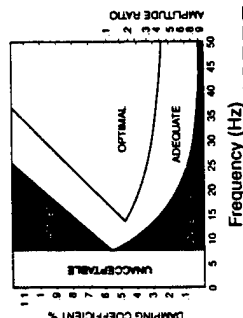
- Determine Natural Frequency of System (F_n)
 - Fast Flush System and Record Strip
 - Measure period (t) of once cycle
 - $F_n = \text{Paper speed (mm/sec) / one cycle (mm)}$
- Determine Amplitude Ratio
 - Compare the amplitude of two successive peaks (A_2/A_1)
- Plot Amplitude Ratio Against Natural Frequency
 - Apply algorithm if system other than OPTIMAL or ADEQUATE



Example:

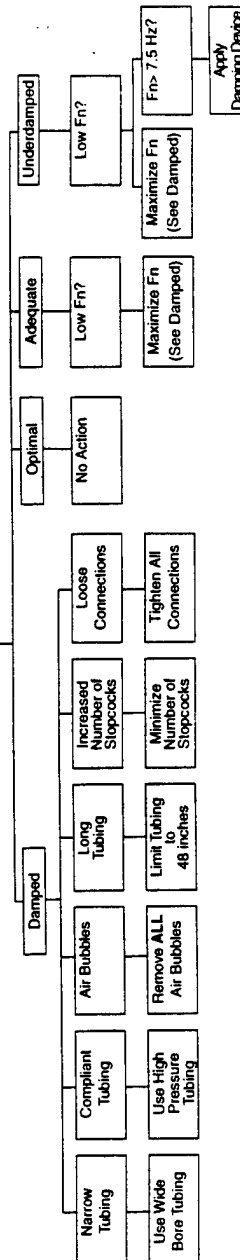
- Determine F_n
Paper Speed=25 mm/sec
 $t=1$ mm
 $F_n=25/1=25$ cycles/sec
- Determine Amplitude
 $A_2/A_1=3/7=0.43$
- Plot on graph=ADEQUATE

Frequency versus Amplitude Ratio Plot



Frequency versus damping coefficient plot that illustrates the five areas into which the catheter, tubing, and transducer systems fall. Systems in the optimal area reproduce even the most demanding (fast heart rate and rapid systolic upstroke) waveforms without distortion. Systems in the adequate area reproduce the most typical waveforms with little or no distortion. All other areas cause serious wave distortion. (Gardner, RM Hollingsworth, KW: Optimizing the electrocardiogram and pressure monitoring. Crit Care Med 14: 651-658m 1986. With permission).

Dynamic Response Characteristics



APPENDIX E

Study Protocol

BEFORE INITIATION OF STUDY PROTOCOL

1. Evaluate patient for inclusion/exclusion criteria

Inclusion Criteria	Exclusion Criteria
☐ PA Catheter ☐ Read/speak English ☐ Hemodynamically stable (SBP > 90 mm Hg; HR < 130 bpm; $\text{s}\bar{\text{v}}\text{O}_2$ > 60%, SaO_2 > 90%) over previous hour ☐ Chest tube drainage < 100 ml/hr over previous 3 hrs ☐ More than four-hours postoperative ☐ No fluid boluses > 250 ml/hr within previous hour ☐ No alteration in inotropic or vasoactive infusions within previous hour ☐ No IV diuretics or morphine within previous hour ☐ PA temperature $\geq 36.0^\circ\text{C}$	☐ No PA catheter ☐ Inability to tolerate 0° -flat backrest position ☐ Clinically unstable (systolic BP < 90 mm Hg; SaO_2 < 90%; $\text{s}\bar{\text{v}}\text{O}_2$ < 60%; baseline HR > 130 bpm) ☐ Mitral stenosis or left atrial myxoma ☐ Receiving hemodialysis ☐ Chest wall deformity (kyphoscoliosis) ☐ Kinetic therapy (Roto-Rest) ☐ Intra-aortic balloon pump ☐ Patient request (specify) ☐ PI/RN Decision

2. Evaluate placement of PA catheter

- Review anteroposterior chest radiograph.
- Determine if PA catheter tip is approximately 3 cm below the level of the left atrium.
- Document position of catheter tip, and note whether positioned in right versus left PA.

3. Evaluate dynamic response characteristics of PA catheter system (Appendix A). Document on data collection record.

- Troubleshoot system if the dynamic response is underdamped or overdamped. (Discuss possible actions with nurse).

Pulmonary Artery Pressure Fluctuation

- Place patient in supine position at whatever degree of backrest elevation (0° to 60°).
- Initiate 5 minute stabilization period. Measure period with stopwatch.
- Identify the phlebostatic axis (intersection of an axis that transects the body at the junction of the fourth intercostal space and the sternal margin, and a frontal plane passing midway between the anteroposterior surface of the chest (Figure 1). Mark point on chest with marker.

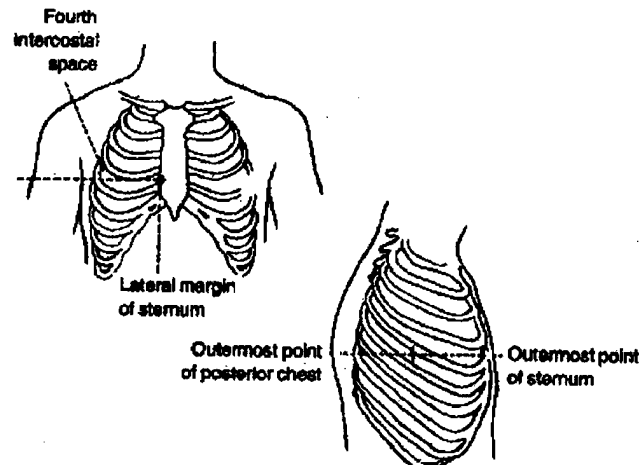


FIGURE 1: Identification of the phlebostatic axis. See text for details. Shinn, J.A., Woods, S.L., & Huseby, J.S. (1979). Effect of intermittent positive pressure ventilation upon pulmonary capillary wedge pressures in acutely ill patients. *Heart Lung*, 8(2), 324. Reprinted with permission.

4. Reference and zero PA catheter system to phlebostatic axis.

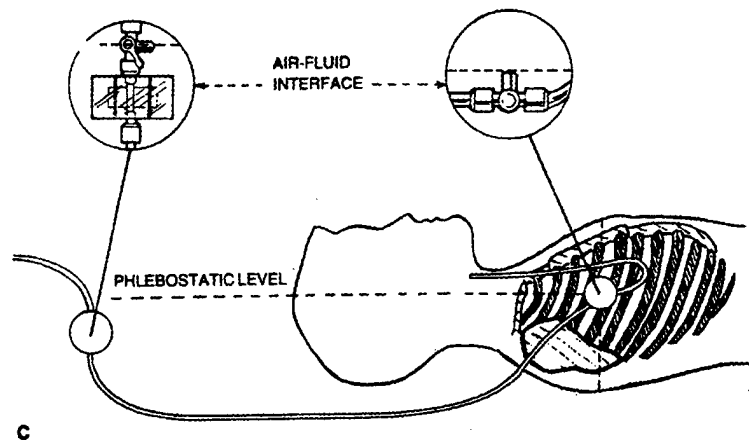


FIGURE 2. Schematic of referencing the pressure monitoring line to the phlebostatic axis. From Bridges, E.J., & Woods, S.L. (1993). Pulmonary artery pressure monitoring: state of the art. *Heart Lung*, 22(2), 101. Reprinted with permission

- a. Place the air-fluid interface of the distal stopcock of the PA catheter at the phlebostatic axis.
- b. Using aseptic technique, open the system to air.
- c. Activate the "Zero" button on the monitor.
- d. Close the system.

(If no in-line stopcock is available, the air-fluid interface of the stopcock on top of the transducer will be referenced to the phlebostatic axis with the aid of a carpenter's level).

5. Following the five minute stabilization period, determine if patient's heart rate has returned to baseline. If not, continue the stabilization period.
 - a. If HR at baseline initiate data collection.
 - b. Record length of stabilization period on data collection record.
6. Measure PA systolic, end-diastolic, and mean, relative to a simultaneous ECG tracing, at end-expiration from an analog recording (Figure 3).
 - a. PA systolic: Represented by the steep rise during right ventricular ejection and usually occurs after the QRS or near the T wave of the ECG.
 - b. PA end-diastolic: Measure 0.08 seconds after the onset of the QRS complex. If left ventricular dysfunction is present, the presystolic "a" wave, if present, will be used as the indicator of PAED pressure.
 - c. PA mean: Bisect the end-expiratory PA waveform.

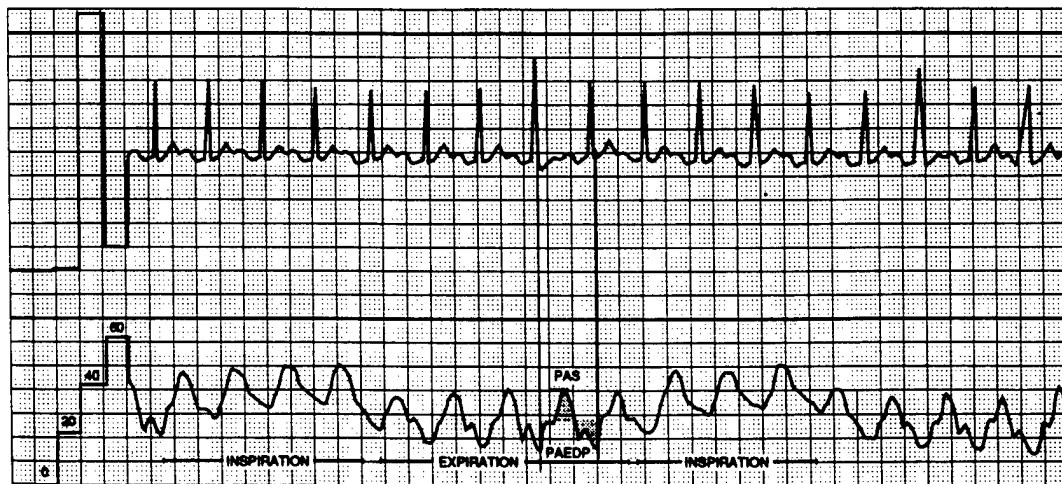


FIGURE 3. Measurement of PA pressure waveform in mechanically ventilated patient. The PA systolic is measured at the peak pressure, the PAED pressure is measured 0.08 second after the QRS complex, and the PA mean is measured by bisecting the waveform. Pressures are read at end-expiration. Example: PA systolic = 44 mm Hg; PA mean = 33 mm Hg; PAED pressure = 22 mm Hg. From *Cardiac Nursing*, (p. 441), by S.L Woods, E.S. Sivarajan Froelicher, C.J. Halpenny, S. Underhill Motzer, 1995, Philadelphia: J.B. Lippincott. Reprinted with permission

7. Measure PA wedge pressure

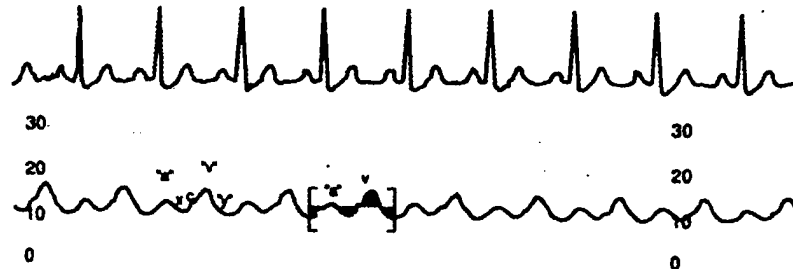


FIGURE 4. Pulmonary artery wedge pressure. Two primary waveforms ("a" and "v") will be identified. The "a" wave is located after the "P" wave of the electrocardiogram (ECG). The "v" wave occurs during the T-P segment of the ECG. The PAW pressure is a mean pressure, and will be measured by bisecting the waveform so there is an equal area above and below the bisection. From *Cardiac Nursing*, (p. 429), by S.L. Woods, E.S. Sivarajan Froelicher, C.J. Halpenny, S. Underhill Motzer, 1995, Philadelphia: J.B. Lippincott. Reprinted with permission.

- a. Inflate the balloon slowly while observing the PA waveform. Maximum inflation volume is 1.5 ml.
 - b. Observe PA waveform flatten into characteristic atrial waveform ("a" and "v" waves).
 - c. Observe for "partial wedge" pattern: A waveform different from the phasic PA waveform, but intermediate between the phasic PA waveform and the atrial waveform. If partial waveform observed, deflate balloon and reinflate. If unable to achieve "wedge" pattern, notify nurse.
 - d. Maintain wedge for a maximum of 15 seconds.
 - e. Deflate balloon by removing syringe from injection port, and observe waveform for return to characteristic PA pattern.
 - f. Record amount of air required to wedge the balloon.
 - g. If inflation volume less than 1 ml, or "overwedge" pattern appears with balloon inflation, immediately deflate balloon by removing the syringe, and notify the nurse. Discontinue protocol.
8. Evaluate waveforms for indication of non-Zone 3 vascular bed.
- a. In mechanically ventilated patients, evaluate the respiratory artifact induced by the mechanical ventilation (inspiratory peak minus expiratory peak value) on the PA and PAW pressure tracings. If the ratio of the PAW pressure change relative to the PA pressure change ($\Delta\text{PAWP}/\Delta\text{PAP}$) is greater than 2 at 0 cm H₂O PEEP, the catheter is in a non-Zone 3 vascular bed. A ratio of 1 for the $\Delta\text{PAWP}/\Delta\text{PAP}$ indicates no respiratory artifact due to Zone 2 alveolar compression.
 - b. Verify that PAW pressure is less than mean PA pressure in the absence of a large "V" wave. If large "V" wave is present ("v" wave 10 mm Hg greater than the peak of the "a" wave, read the PAW pressure at the nadir of the "x" descent.

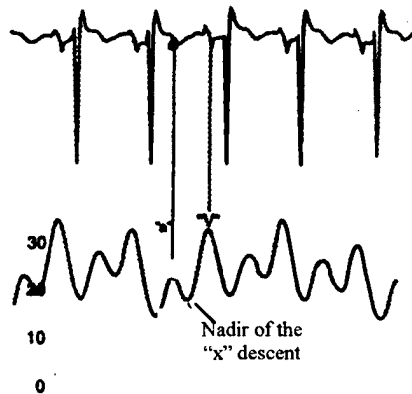


FIGURE 5: PAW pressure waveform with large "V" wave. The PAW pressure is measured at the nadir of the "x" descent. From *Cardiac Nursing*, (p. 429), by S.L. Woods, E.S. Sivarajan Froelicher, C.J. Halpenny, S. Underhill Motzer, 1995, Philadelphia: J.B. Lippincott. Reprinted with permission.

9. If the catheter is determined to be in a non-zone 3 vascular segment or "overwedges" notify the nurse. If the catheter can be repositioned, re-enter the patient into the protocol, otherwise exclude the patient from the study.

POSITION EFFECT

1. Randomize subject into treatment group (Group A: flat, supine; 30-degree left lateral recumbent position, 30-degree right lateral recumbent position, supine; Group B: flat, supine, 30-degree right lateral recumbent, 30-degree left lateral recumbent, flat, supine). The first subject will be assigned by a coin toss, and subsequent subjects will be assigned to a sequence by odd or even number.
2. Place subject into supine position, with 0-degree backrest elevation. (One pillow may be placed under the subject's head for comfort).
3. Initiate five minute stabilization period.
4. Zero and reference the PA catheter system to the phlebostatic axis as previously described.
5. At five minutes assess subject for return to baseline heart rate. If heart rate at baseline, and less than 130 beats per minute, continue protocol.
6. Measure PA systolic, diastolic, mean, and wedge pressures as previously described.
7. Passively position subject into right or left 30-degree lateral position (depending on assigned sequence).
8. Place 30-degree hard wedge behind the subject's back with the bed at the intersection of the chest wall and bed surface.
9. Initiate the five minute stabilization period.

10. Identify the left atrial reference.
 - a. Using flat plate calipers, place horizontal blades parallel to the bed surface and left sternal border.
 - b. Identify point one-half the vertical distance between the left sternal border and bed surface.
 - c. With the aid of a carpenter's level, identify the left atrial reference level (mark on chest).
 11. Reference and zero the catheter to the left atrial level. (Record difference (cm) from supine reference level).
 12. If HR has returned to baseline, measure PA pressures as specified. (Monitor closely for decreased balloon volume or overwedge associated with distal migration of catheter).
 13. Passively rotate subject to opposite lateral position and repeat steps 8 through 12.
 14. Place subject in supine, 0-degree backrest elevation position. Repeat steps 2 through 6.
 15. Place patient in position of comfort, and thank them for their participation.
 16. Provide nurse caring for patient, PA pressure measurements obtained with patient in the supine position. Notify the nurse of any unusual findings noted during the course of the study.
 17. Period of study
 - a. Determine total study time and stabilization periods using a stopwatch.
 - b. Document stabilization periods and study time on data collection record.
-

APPENDIX F

Consent Form

University of Washington

Consent Form

Accuracy of Pulmonary Artery Pressures in the Side-Lying Position

Elizabeth Bridges, MN, RN, CCRN, Doctoral Candidate, School of Nursing,

Telephone: (206) 616-8407

Susan L. Woods, PhD, RN, Professor, Biobehavioral Health Systems Nursing and Associate Dean for Academic Programs, School of Nursing. Telephone: (206) 616-8407

Investigator's Statement:

Purpose and Benefits

As part of your care, a small catheter will be positioned in your heart. It is used to measure the pressure in your heart. These pressure measurements are usually obtained while you are lying on your back, sometimes every hour. This study is designed to determine if the pressure measurements obtained, with the catheter that is in your heart, are accurate if taken while you are lying on your side. This information is important to know, so that future patients could have measurements taken while they sleep on their side without being awakened to turn on their back for the pressure measurements. There is no benefit to you for participating.

Procedures

To determine how your heart pressures change over time, routine pressure measurements will be obtained over a 15-minute period while you are resting quietly in bed. A total of four measurements will be obtained. Following this 15-minute period you will be assisted into a flat position in bed and then you will be turned to both the right and left sides, and the measurements will again be taken with you in each of those positions. There will be a pause of 5 minutes on each side before the measurements are taken. A total of eight measurements will be taken. The total time will be about 45 minutes (15 minutes to determine baseline fluctuation, and 30 minutes for the positioning portion of the protocol). During your routine care, the measurement of one set of these pressures takes about 5 minutes. Information from your

chart will be obtained about your medical history, blood pressure, pulse, height, weight, and medications you are taking.

Risks, Stress, or Discomfort

Both pressure measurement and turning side to side are routine procedures in a critical care unit. There are no risks expected. There may be some discomfort from turning and the side-lying position. All efforts will be taken to minimize the discomfort of turning. If any of the additional measurements or positions should make you uncomfortable, the measurements will be stopped immediately and you will be placed in a position that is most comfortable for you. In the event of an injury during the time that the readings are being taken from your catheter, you will be cared for in the critical care unit.

Other Information

The alternative to participation in this study is to not participate. Your name will not be used and it will not be possible to identify you by any information about you. The data will not be anonymous, but will be kept confidential with only the principal investigator and co-investigator having access to the data. The data will be retained for an indefinite amount of time. The information will be used for the purpose of a doctoral dissertation, which will be available in the University of Washington library. You may refuse to participate or may withdraw from the study at any time without penalty or loss of benefits to which you are otherwise entitled. There is no compensation for participation in this study. The costs of treatment will be the responsibility of you and your insurance company, the same as for the other treatment you are receiving.

Signature of Investigator Date

Subject's Statement

This study has been explained to me, and I voluntarily consent to participate in this activity. I have had the opportunity to ask questions. I understand that future questions I may have about the research or about my rights as a subject will be answered by one of the investigators listed above.

Signature of Subject Date

Copies to: Subject
Investigator's File

APPENDIX G

Individual Demographic Data

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time – Hrs before study)
1B	72 yo, female; BSA = 2.11 (194% IBW), <u>Midsleep</u> = 0130; <u>Bedrest</u> = 0 days; <u>Position</u> = Supine, HOB @ 18° <u>Vital Signs</u> : HR = 90 (Sinus), BP 128/56; Temp: 36.7 RR 16 (Spont), SaO2 96% (FiO2 = 0.35); CO/CI: 6.2/3.0; RAP: 16; SVR = 853; CT: 70 ml/3°; CXR: Catheter: Main PA	Inferior MI, DM, HTN, GERD EF: MD	CABG X 5 CPB = 131 min IT = MD Hours since surgery: 23 Time of study: 1115-1228 Hours after midsleep: 11	ASA (2°), Levodroxine (2°), Lotensin (2°), Furosemide IV (3°) Infusion: Insulin, magnesium Total IVF Rate: 24 ml/hr
2A	63 yo, female; BSA = 1.94 (158% IBW), <u>Midsleep</u> = 0045; <u>Bedrest</u> = 0 days; <u>Position</u> = Supine, HOB @ 20° <u>Vital Signs</u> : HR = 100 (Sinus), BP 110/58/78; Temp: 36.5 RR 20 (Spont), SaO2 96% (FiO2 = MD); CO/CI: 5.2/2.5; RAP: 12; SVR = 904; CT: 120 ml/3°; CXR: Catheter: Main PA; Bilateral pleural effusion (L > R)	CAD, NIDDM, HTN, Bells Palsy, Hyperlipidemia EF: "normal"	CABG X 5 CPB = 131 min IT = 95 Hours since surgery: 18 Time of study: 1437-1531 Hours after midsleep: 14	Morphine IV (90 min), cefazolin (5°), Infusion: Insulin, magnesium, potassium phosphate, dopamine @ 2 ug/kg/min, nitroglycerin @ 5 ug/min Total IVF Rate: 78.3 ml/hr
3B	82 yo, male; BSA = 1.90 (110% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0 days; <u>Position</u> = Supine, HOB @ 26° <u>Vital Signs</u> : HR = 88 (Sinus), BP 110/60; Temp: 37 RR 20 (Spont), SaO2 99% (FiO2 = 0.40); CO/CI: 5.3/2.9; RAP: 6; SVR = 1054; CT: 250 ml/3°; CXR: Catheter: Main PA	HTN, asthma, dysphagia, dyspnea on exertion, ataxia, vertigo, CVA, depression	CABG X 2 CPB = 102 min IT = 64 Hours since surgery: 20 Time of study: 1006 – 1101 Hours after midsleep: 8	Infusion: Magnesium, nitroglycerin @ 0.22 ug/kg/min, dopamine @ 3 ug/kg/min Total IVF Rate: 24.9 ml/hr
4A	60 yo, male; BSA = 2.04 (128% IBW), <u>Midsleep</u> = 0230; <u>Bedrest</u> = 1 days; <u>Position</u> = Supine, HOB @ 26° <u>Vital Signs</u> : HR = 87 (Sinus), BP 134/78/97; Temp: 37.4 RR = 20 (Spont), SaO2 98% (FiO2 = 0.28); CO/CI: 8.8/4.4; RAP: MD; SVR = 780; CT: 20 ml/3°; CXR: Catheter: Main PA	Aortic valve stenosis & regurgitation, HTN, LVH EF: 72%	Aortic valve replacement CPB: 62 IT: MD Hours since surgery: 14 Time of study: 0935-1031 Hours after midsleep: 7	Morphine-IV (1°), Multivitamin (1°), potassium IV (90 min), hydralazine-PO (during study), percoet (during study) Infusion: Magnesium, nifedipine @ 0.22 ug/kg/min Total IVF Rate: 20 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time - Hrs before study)
5B	50 yo, male, BSA = 2.42 (165% IBW), <u>Midsleep</u> = 0145; <u>Bedrest</u> = 0 days; <u>Position</u> = Supine, HOB @ 19° <u>Vital Signs</u> : HR = 125 (Sinus Tach), BP 98/64/77; Temp: 37.3, RR = 25 (Spont), SaO2 96% (FiO2 = 48), CO/CI 13.4/5/5; RAP: 10; SVR = 400; CT: 90 ml/3°; CXR: Catheter: Right PA	CAD, PTCA, recurrent angina, sleep apnea, HTN, IDDM, GERD, obesity, anxiety	CABG X 4 CPB: 140 min IT: 118 min Hours since surgery: 20 Time of study: 1027-1122 Hours after midsleep: 8.5	Isordil- (PO - 3°), Lasix (IV - 3°), Percocet (PO - 3°), Morphine (IV - 75 min), Metoprolol (IV - 45 min), Infusion: Magnesium; Dopamine 2 ug/kg/min Total IVF Rate: 16.7 ml/hr
6A	73 yo, male, BSA = 2.17 (129% IBW), <u>Midsleep</u> = 0130; <u>Bedrest</u> = 0 days; <u>Position</u> = Supine, HOB @ 24° <u>Vital Signs</u> : HR = 91 (Sinus), BP: 150/68/94; Temp: 36.7, RR 14 (Spont), SaO2 98% (FiO2 = 0.50); CO/CI 10.1/4.7; RAP: 9; SVR: 634; CT: 10 ml/3°; CXR: Catheter: Main PA, left pleural effusion, bilateral fluffy infiltrates	CAD, stable angina, MI (inferior), NIDDM, migraine, arthritis	CABG X 5 CPB: 200 IT: 179 Hours since surgery: 14 Time of study: 1651-1740 Hours after midsleep: 15.5	Morphine-IV (70 min), Blood (4°) Infusion: Magnesium, nitroglycerin @ 0.25 ug/kg/min, dopamine @ 2 ug/kg/min, nifedipine @ 1.8 ug/kg/min Total IVF Rate:
7A	52 yo, male, BSA = 1.83 (91% IBW), <u>Midsleep</u> = erratic; <u>Bedrest</u> = 4 days; <u>Position</u> = Supine, HOB @ 42° <u>Vital Signs</u> : HR = 93 (Sinus), BP: 115/39/55; Temp: 36.6, RR: 20 (Spont), SaO2 97% (FiO2 = 0.48); CO/CI: 8.4/4.6; RAP: 10; SVR = 603; CT: 250 ml/3°; CXR: Catheter: RV outflow tract	Aortic insufficiency with LVE, aortic stenosis, mild mitral stenosis/insufficiency, acute renal failure, anemia, endocarditis, EF = 54%	Aortic root replacement with homograft, CABG X 1 CPB: 176 IT: 128 Hours since surgery: 18 Time of study: 1736-1831 Hours after midsleep: N/A	Cefazolin (90 min), sucralate (5°), percocet (3°), morphine (2°) Infusion: Milrinone @ 0.14 ug/kg/min, epinephrine @ 0.02 ug/kg/min, nitroglycerin @ 0.25 mcg/kg/min Total IVF Rate: MD
8A	76 yo, male, BSA = 1.88 (102% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = MD; <u>Position</u> = Supine, HOB @ 45° <u>Vital Signs</u> : HR = 70 (AFib), BP: 122/54/77; Temp: 36.1, RR: 16 (Spont), SaO2 94% (FiO2 = 0.24); CO/CI: 5.4/2.9; RAP: 6; SVR = 823; CT: 60 ml/3°; CXR: Catheter: Right PA, marked left pleural effusion	ASD with 4:1 L→R shunt, chronic AFib with RBBB, lower extremity edema, SOB, DOE	Patch closure of ASD with pericardium and SVC augmentation patch CPB: 112 IT: 74 Hours since surgery: 15 Time of study: 0930-1025 Hours after midsleep: 7.5	Furosemide- PO (30 min), vancomycin (during study) Infusion: Magnesium, nitroglycerin @ 1.25 ug/kg/min, dopamine @ 2 ug/kg/min Total IVF Rate: 67.9 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time – Hrs before study)
9A	73 yo, male, BSA = 1.98 (118% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 0° <u>Vital Signs</u> : HR = 76 (Sinus), BP: 116/58/76; Temp: 37.5, RR: MD (Spont), SaO2 96% (FiO2 = MD); CO/CI: 9.5/5.5; RAP: 16; SVR = 489; CT: 80 ml/3°; <u>CXR</u> : Catheter: RV outflow (looping)	Unstable angina, inferolateral ischemia, HTN, hx brain tumor with left sided hemiparesis	CABG X 5 CPB: 228 IT: 200 Hours since surgery: 12 Time of study: 0854-0950 Hours after midsleep: 7	Infusion: Magnesium, nitroglycerin @ 0.5 ug/kg/min, dopamine @ 1.5 ug/kg/min, lidocaine @ 1.0 mg/min Total IVF Rate: 43.5 ml/hr
10B	82 yo, female, BSA = 1.98 (151% IBW), <u>Midsleep</u> = 0245; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 12° <u>Vital Signs</u> : HR = 60 (Junctional), BP: 93/34/54; Temp: 36.6, RR: 12 (Spont), SaO2 96% (FiO2 = 0.28); CO/CI: 5.4/2.7; RAP: 14; SVR: 593; CT: 50 ml/3°; <u>CXR</u> : Catheter: Right PA – periphery – pulled back before study	Aortic stenosis, coronary artery insufficiency, EF = 53%	AVR, CABG X 2 CPB: 154 IT: 109 Hours since surgery: 19 Time of study: 0835-0922 Hours after midsleep: 6	Furosemide-IV (90 min) Infusion: Dopamine @ 1.0 ug/kg/min Total IVF Rate: 3.4 ml/hr
11A	73 yo, male, BSA = 1.78 (94 % IBW), <u>Midsleep</u> = 0300; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 25° <u>Vital Signs</u> : HR = 76 (Sinus), BP: 112/58/76; Temp: 37.4, RR: 20 (Spont), SaO2 94% (FiO2 = 0.36); CO/CI: 6.1/3.2; RAP: 7; SVR: 905; CT: 120 ml/3°; <u>CXR</u> : Catheter: Right PA; left pleural effusion	Aortic stenosis, hiatal hernia	AVR CPB: 118 IT: 95 Hours since surgery: 15 Time of study: 0819-0910 Hours after midsleep: 8.5	Morphine-IV (2°), cefazolin (6°) Infusion: Nitroglycerin @ 0.5 ug/kg/min Total IVF Rate: 10.2 ml/min
12B	61 yo, male, BSA = 2.04 (131% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 9° <u>Vital Signs</u> : HR = 87 (Sinus), BP: 98/62/74; Temp: 37.3, RR: 22 (Spont), SaO2 91% (FiO2 = 0.40); CO/CI: 6.1/3.0; RAP: 12; SVR: 786; CT: 130 ml/3°; <u>CXR</u> : Catheter: Right PA; left pleural effusion	CAD, PTCA, Stent	CABG X 4 CPB: 146 IT: 120 Hours since surgery: 18 Time of study: 0837-0924 Hours after midsleep: 6.5	Infusion: Magnesium, nitroglycerin @ 0.25 ug/kg/min Total IVF Rate: 18.8 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time – Hrs before study)
13B	71 yo, male, BSA = 2.2 (160% IBW), <u>Midsleep</u> = 0330; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 22° <u>Vital Signs</u> : HR = 82 (Sinus), BP: 100/56/80; Temp: 37.0, RR: 12 (Spont), SaO ₂ 94% (FiO ₂ = 0.48); CO/CI: 6.5/2.9; RAP: 10; SVR: 800; <u>CT</u> : 25 ml/3°; <u>CXR</u> : Catheter: Main PA; left pleural effusion	Unstable angina, MI (inferior), right carotid endarterectomy, hiatal hernia, snoring	CABG X 3 CPB: 107 IT: 79 Hours since surgery: 16 Time of study: 0801-0824 Hours after midsleep: 4.5	Insulin-SQ (3°) Infusion: Magnesium, nitroglycerin @ 0.3 ug/kg/min Total IVF Rate:
14A	66 yo, male, BSA = 1.87 (103% IBW), <u>Midsleep</u> = 0015; <u>Bedrest</u> = 1; <u>Position</u> = Supine, HOB @ 10° <u>Vital Signs</u> : HR = 92 (Sinus), BP: 112/50/71; Temp: 37.2, RR: 25 (Spont), SaO ₂ 99% (FiO ₂ = 0.24); CO/CI: 4.4/2.2; RAP: 8; SVR: 484; <u>CT</u> : 25 ml/3°; <u>CXR</u> : Catheter: Right PA; minimal pleural effusion	1 year history of V/VI SEM, increased DOE & SOB	AVR, CABG X 3 CPB: 114 IT: 95 Hours since surgery: 15 Time of study: 0818-0906 Hours after midsleep: 8	Furosemide-IV (75 min), Isordil-PO (15 min) Infusion: Nitroglycerin @ 0.24 ug/kg/min Total IVF Rate: 5 ml/hr
15B	60 yo, male, BSA = 1.92 (123% IBW), <u>Midsleep</u> = 0500; <u>Bedrest</u> = MD; <u>Position</u> = Supine, HOB @ 33° <u>Vital Signs</u> : HR = 93 (Sinus), BP: 114/52/65; Temp: 36.7, RR: 12 (Spont), SaO ₂ 96% (FiO ₂ = 0.22); CO/CI: 5.5/2.9; RAP: 4; SVR: 975; <u>CT</u> : 50 ml/3°; <u>CXR</u> : Catheter: Main PA/RV outflow; minimal pleural effusion	MI, colon CA (pre-op), IDDM with triopathy	CABG X 3 CPB: 130 IT: 101 Hours since surgery: 17 Time of study: 0922-1011 Hours after midsleep: 4.5	Isordil -PO (30 min), percocet (3°) Infusion: Magnesium, insulin, dopamine @ 2 ug/kg/min Total IVF Rate: 20 ml/hr
16B	60 yo, male, BSA = 2.03 (131% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 24° <u>Vital Signs</u> : HR = 95 (Sinus), BP: 95/52/66; Temp: 37.5, RR: 20 (Spont), SaO ₂ 96% (FiO ₂ = 0.36); CO/CI: 4.7/2.3; RAP: 10; SVR: 953; <u>CT</u> : 100 ml/3°; <u>CXR</u> : Catheter: Right PA; left lung hazy, left and right pleural effusions, poor inspiratory film	CAD – Redo CABG, HTN, AFib (treated with cardioversion), DM	CABG X 4 CPB: 154 IT: 98 Hours since surgery: 32 Time of study: 0803-0857 Hours after midsleep: 6	Infusion: Dopamine @ 4 ug/kg/min Total IVF Rate: 13.4

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time – Hrs before study)
17A	73 yo, male, BSA = 2.3 (128% IBW), <u>Midsleep</u> = 0330; <u>Bedrest</u> = 2; <u>Position</u> = Lateral/Supine, HOB @ 17° <u>Vital Signs</u> : HR = 110 (Afib/Flutter), BP: 100/40/60; Temp: 38.4, RR: 21 (Spont), SaO ₂ 95% (FiO ₂ = 0.40); SvO ₂ : 62%; CO/CI: 6.4/3.0; RAP: 14; SVR: 554; <u>CT</u> : 20 ml/3°; <u>CXR</u> : Catheter: Main PA; underdeveloped film, large cardiac silhouette, left pleural effusion	CAD, Ischemic cardiomyopathy, CHF, MI, Afib, NIDDM, TIA, pulmonary HTN EF: 15-20%	CABG X 4 CPB: 122 IT: 87 Hours since surgery: 10 Time of study: 0840-0928 Hours after midsleep: 5	Furosemide-IV (70 min), isordil (70 min), digoxin-PO (30 min), albuterol nebulizer (80 min) Infusion: Fentanyl, magnesium, insulin, lidocaine @ 1 mg/min, epinephrine @ 0.06 ug/kg/min Total IVF Rate: 51.4 ml/hr
18A	72 yo, male, BSA = 2.06 (145% IBW), <u>Midsleep</u> = 0045; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 14° <u>Vital Signs</u> : HR = 81 (Sinus), BP: 94/48/63; Temp: 37.5, RR: 20 (Spont), SaO ₂ 94% (FiO ₂ = 0.36); CO/CI: 5.2/2.5; RAP: 9; SVR: 831; <u>CT</u> : 60 ml/3°; <u>CXR</u> : Catheter: Right PA; small left pleural effusion, left lung hazy	Dyspnea, CAD, new onset reflux X 6 months	CABG X 4 CPB: 89 IT: 52 Hours since surgery: 21 Time of study: 0938-1024 Hours after midsleep: 9	Furosemide-IV (90 min), atenolol-PO (30 min) Infusion: Magnesium Total IVF Rate: 11.5 ml/hr
19A	57 yo, male, BSA = 1.87 (90% IBW), <u>Midsleep</u> = 0415 (sleeps in 2° periods due to pain); <u>Bedrest</u> = bedridden; <u>Position</u> = Supine, HOB @ 60° - lowered to 45° for study <u>Vital Signs</u> : HR = 63-77 (Afib), BP: 105/38/58; Temp: 36.9, RR: 20 (Spont), SaO ₂ 95% (FiO ₂ = 0.36); CO/CI: 5.4/2.0; RAP: 13; SVR: 667; <u>CT</u> : 30 ml/3°; <u>CXR</u> : Catheter: RV outflow; bilateral haziness up ½	Rheumatic fever, s/p porcine valve (1986), multiple sclerosis	MVR CPB: 105 IT: 73 Hours since surgery: 16 Time of study: 0805-0837 Hours after midsleep: 4	Cefazolin-IV (1°), gentamycin-IV (1°), ciprofloxacin-IV (1°) Infusion: Dopamine @ 2 ug/kg/min Total IVF Rate: 5.1 ml/hr
20B	59 yo, male, BSA = 2.00 (122% IBW), <u>Midsleep</u> = 0430; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 38° <u>Vital Signs</u> : HR = 74 (Sinus), BP: 108/60/78; Temp: 37.2, RR: 20 (Spont), SaO ₂ 91% (FiO ₂ = 0.50); CO/CI: 6.9/3.4; RAP: 10; SVR: 791; <u>CT</u> : 160 ml/3°; <u>CXR</u> : Catheter: Main PA; haziness right lower lobe, left pleural effusion	Redo CABG, MI X 2, HTN, angina, reflux	CABG X 4 CPB: 87 min IT: 70 min Hours since surgery: 19 Time of study: 0822-0917 Hours after midsleep: 4	Infusion: Magnesium, nitroglycerin @ 20 ug/min, dopamine @ 20 ug/kg/min, diltiazem @ 2.5 mg/hr Total IVF Rate: 27.3 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time - Hrs before study)
21B	73 yo, male, BSA = 1.93 (105% IBW), <u>Midsleep</u> = 0330; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 47° <u>Vital Signs</u> : HR = 89 (100% AAI-Paced), BP: 113/71/85; Temp: 37.0, RR: 20 (Spont), SaO2 92% (FiO2 = 0.28); CO/CI: 5.5/4.6; RAP: 6; SVR: 781; CT: 210 ml/3°; CXR: Catheter: RV outflow; left pleural effusion, right upper lobe hazy	Stable angina, 3-vessel CAD	Surgery: CABG CPB: 167 min IT: 146 min Hours since surgery: 17 Time of study: 0851-0907 Hours after midsleep: 5.5	KCL-IV (2°) Infusion: Magnesium, nitroglycerin 0.2 ug/kg/min, dopamine 5.0 mg/kg/min Total IVF: 30.5 ml
22B	72 yo, male, BSA = XX (130% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 9° and 4°reverse trendelenburg <u>Vital Signs</u> : HR = 105 (Sinus Tach), BP: 164/80/110; Temp: 37.6, RR: 26 (Spont), SaO2 94% (FiO2 = 0.40); CO/CI: 5.8/3.0; RAP: 11; SVR: 1416; CT: 40 ml/3°; CXR: Catheter: Right PA; hazy bilaterally, no pleural effusion	CAD (MI/PTCA), depression, HTN, DM, PUD	Surgery: CABG X 4 CPB: 138 min IT: 126 min Hours since surgery: 16 Time of study: 0855-0927 Hours after midsleep: 7	Metoclopramide-IV (2°), calcium gluconate-IV (3°), furosemide-IV (3.5°) Whole blood (4.5°), hespan (6°) Infusion: Magnesium, nitroglycerin @ 1.0 ug/kg/min, nifedipine @ 0.3 ug/kg/min Total IVF Rate: 42.5 ml/hr
23B	53 yo, male, BSA = 2.09 (133% IBW), <u>Midsleep</u> = 0245; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 38° <u>Vital Signs</u> : HR = 81 (Sinus with RBBB), BP: 90/45/60; Temp: 38.1, RR: 20 (Spont), SaO2 97% (FiO2 = 0.24); CO/CI: 5.5/2.9; RAP: 8; SVR: 840; CT: 70 ml/3°; CXR: Catheter: Right PA; poor inspiratory film, left lung field hazy, ? bilat pleural effusion	CABG/AVR, hx CABG X 4, esophageal reflux	Surgery: CABG X 3 CPB: 167 min IT: 130 min Hours since surgery: 13 Time of study: 0952-1038 Hours after midsleep: 7.5	Furosemide-PO (1°), metolazone-PO (2°), morphine-IV (3°), percofet (4°) Patient with poor pain relief with percofet X 4; no increased discomfort associated with study procedure Total IVF Rate: 0 ml/hr
24B	89 yo, male, BSA = 1.90 (112% IBW), <u>Midsleep</u> = 0245; <u>Bedrest</u> = MD; <u>Position</u> = Lateral - Supine, HOB @ 22° <u>Vital Signs</u> : HR = 97 (100% Paced - VOO), BP: 136/61/85; Temp: 36.8, RR: 18 (Spont), SaO2 93% (FiO2 = 0.50); CO/CI: 4.2/2.7; RAP: 13; SVR: 1123; CT: 90 ml/3°; CXR: Catheter: RV outflow; bilateral pleural effusion, permanent pacemaker	AVR/CABG, rheumatic fever, bradycardia treated with pacemaker, GERD, aortic stenosis, HTN, CHF	Surgery: Aortic valve replacement CPB: 127 min IT: 71 min Hours since surgery: 22 Time of study: 1116-1201 Hours after midsleep: 9	Multivitamin (2°), digoxin-PO (2°), vancomycin-IV (5°), amlodipine-PO (2°), furosemide-IV (3°), calcium gluconate-IV (2°) Infusion: Dopamine @ 1.0 ug/kg/min, nifedipine @ 0.42 ug/kg/min, magnesium Total IVF Rate: 25 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time - Hrs before study)
25A	55 yo, male, BSA = 1.98 (134% IBW), <u>Midsleep</u> = MD; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 14° <u>Vital Signs</u> : HR = 95 (Sinus), BP: 106/58/76; Temp: 38.0, RR: 12/18 (Vent), PEEP: 5, Vt: 900, SaO2 98% (FiO2 = 0.40); CO/CI: 7.8/3.8; RAP: 12; SVR: 543; <u>CT</u> : 30 ml/3°; <u>CXR</u> : Catheter: Right PA	Recurrent chest pain, MI (anteroseptal), reflux, CHF, chronic low back pain	Surgery: CABG X 3 CPB: 110 min IT: 73min Hours since surgery: 13 Time of study: 0835-0927 Hours after midsleep: MD	Infusion: Magnesium, propafol @ 150 mg/hr, fentanyl @ 7.5 ug/hr, epinephrine @ 0.8 ug/kg/min, nitroglycerin @ 0.2 ug/kg/min, dopamine @ 4 ug/kg/min Total IVF Rate: 67.5 ml/hr
26A	75 yo, female, BSA = 1.78 (86% IBW), <u>Midsleep</u> = 0130; <u>Bedrest</u> = 1; <u>Position</u> = Supine, HOB @ 30° <u>Vital Signs</u> : HR = 95 (AFib), BP: 113/63/83; Temp: 37.8, RR: 26 (Spont), SaO2 97% (FiO2 = 0.36); CO/CI: 6.1/3/5; RAP: 10; SVR: 1145; <u>CT</u> : 10 ml/3°; <u>CXR</u> : Catheter: Main PA; blunting right costophrenic margin, left pleural effusion with haziness in all fields on left, and right lower lobe	Rheumatic fever, mitral regurgitation, CHF, atypical chest pain, TIA, atrial fibrillation, multiple cerebral infarcts, orthopnea, 3- year hx SOB	Surgery: MVR, CABG X 3, endarterectomy CPB: 244 min IT: 195min Hours since surgery: 19 Time of study: 1032-1133 Hours after midsleep: 9	Furosemide-IV (90 min), imdur (isosorbide mononitrate - PO (90 min) Infusion: Magnesium, nifedipine @ 1.4 ug/kg/min Total IV Fluid Rate: 26.0 ml/hr
27B	74 yo, male, BSA = 2.14 (144% IBW), <u>Midsleep</u> = MD; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 14° <u>Vital Signs</u> : HR = 94 (Sinus), BP: 111/53/68; Temp: 37.8, RR: 10/23 (Vent), PEEP: 10, Vt: 1000, SaO2 96% (FiO2 = 0.60); CO/CI: 7.3/3.3; RAP: 13; SVR: 513; <u>CT</u> : 120 ml/3°; <u>CXR</u> : Catheter: RV outflow, bilateral haziness, small right pleural effusion	Angina with increasing SOB, DM, CHF, cerebellar stroke, severe GERD, gout, spinal stenosis, EF < 50% with apical, inferior, posterior hypokinesis	Surgery: AVR, CABG X 4 CPB: 321 min IT: 244min Hours since surgery: 19 Time of study: 1320-1421 Hours after midsleep: MD	Sucralfate (4°), vancomycin-IV (5°), acetaminophen (5°), furosemide-IV (90min/3°) Infusion: Magnesium, sodium phosphate, insulin, fentanyl @ 30 mcg/hr, dopamine @ 2.0 ug/kg/min, epinephrine @ 0.06 ug/kg/min, nitroglycerin @ 0.25 ug/kg/min Total IVF Rate: 65.6 ml

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time – Hrs before study)
28A	72 yo, female, BSA = 1.84 (139% IBW), <u>Midsleep</u> = 0330 (naps); <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 22° <u>Vital Signs</u> : HR = 99 (Sinus), BP: 103/54/68; Temp: 36.3, RR: 14 (Spont), SaO2 94% (FiO2 = 0.40); CO/CI: 4.8/2/6; RAP: 10; SVR: 815; CT: 30 ml/3°; <u>CXR</u> : Right PA, haziness right and left lower lobes, minimal effusion	Severe aortic stenosis, LVH with akinetic and thinned inferior and posterior walls, moderate mitral regurgitation, LAE, LV dilation, moderate tricuspid regurgitation EF: 40%	Surgery: AVR, CABG X 3 CPB: 176 min IT: 137 min Hours since surgery: 16 Time of study: 0827-0915 Hours after midsleep: 5	Vancomycin – IV (30 min), norvasc-PO (30 min), isordil (30 min), furosemide-PO (30 min), potassium elixer (30 min) Infusion: Magnesium, nitroglycerin @ 0.5 ug/kg/min Total IVF Rate: 24 ml/hr
29A	62 yo, female, BSA = 1.68 (110% IBW), <u>Midsleep</u> = naps at night; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 0° <u>Vital Signs</u> : HR = 89 (Paced - AAI), BP: 111/53/72; Temp: 37.4, RR: 12/12 (Vent), PEEP: 5, Vt: 700, PIP: 36, SaO2 100% (FiO2 = 0.40); CO/CI: 4.0/2.4; RAP: 10; SVR: 917; CT: 180 ml/3°; <u>CXR</u> : RV outflow, hazy on left, minimal pleural effusion, pacemaker	CP (10-20 SL NTG QD), IDDM, TIA, hypothyroidism, chronic low back pain, hypercholesterolemia, HTN, GERD, claudication EF 60%, inferobasilar hypokinesia	Surgery: CABG X 2 CPB: 121 min IT: 69 min Hours since surgery: 13 Time of study: 0838-0926 Hours after midsleep: N/A	Infusion: Propafol @ 150 ug/hr, fentanyl @ 25 mcg/hr, magnesium, insulin, dopamine @ 2.0 ug/kg/min, nitroglycerin @ 0.25 ug/kg/min Total IVF Rate: 43.3 ml/hr
30B	47 yo, Male, BSA = 1.63 (83% IBW), <u>Midsleep</u> = 0215; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 15° <u>Vital Signs</u> : HR = 89 (Paced - AAI), BP: 97/60/78; Temp: 37.0, RR: 16, SaO2: 95% (FiO2 = 0.32); CO/CI: 6.9/4.2; RAP: 11; SVR: 608; CT: 60 ml/3°; <u>CXR</u> : Right PA; small left pleural effusion	Mitral valve regurgitation	Surgery: MVR CPB: 127 min IT: 107 min Hours since surgery: 19 Time of study: 0811-0857 Hours after midsleep: 6	Magnesium Sulfate -IV (1°), calcium gluconate - IV (1°) Infusion: Dopamine @ 3 ug/kg/min Total IVF Rate: 6.2 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time ~ Hrs before study)
31A	56 yo, Female, BSA = 1.90 (126% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 21° <u>Vital Signs</u> : HR = 79 (Paced - AAI), BP: 112/59/77; Temp: 36.7, RR: 14, SaO2: 97% (FiO2 = 0.28); CO/CI: 4.9/2.6; RAP: 14; SVR: 938; <u>CT</u> : 60 ml/3°; <u>CXR</u> : Right PA; left hazy without pleural effusion	Mitral stenosis, hyperthyroidism, GERD EF = 40%	Surgery: MVR CPB: 143 min IT: 103 min Hours since surgery: 18 Time of study: 0811-0856 Hours after midsleep: 6	Vancomycin IV (1°), percocet (2°) Infusion: Magnesium, NTG @ 0.25 ug/kg/min Total IVF Rate: 17 ml/hr
32A	80 yo, Female, BSA = 1.61 (109% IBW), <u>Midsleep</u> = 0300; <u>Bedrest</u> = 0; <u>Position</u> = Lateral 20°, HOB @ 5° <u>Vital Signs</u> : HR = 94 (AFib), BP: 133/49/77 (variable); Temp: 37.2, RR: 8/12 (Vent), PEEP: 5, Vt: 750, PIP: 44, SaO2: 96% (FiO2 = 0.40); CO/CI: 4.1/2.5; RAP: 12; SVR: 1268; <u>CT</u> : 20 ml/3°; <u>CXR</u> : RV outflow, marked haziness on left	Mitral valve regurgitation, S/P MVR, CVA, HTN, AFib, RBBB	Surgery: Reop MVR CPB: 133 min IT: 113 min Hours since surgery: 22 Time of study: 1134-1155 Hours after midsleep: 8.5	Sucralfate (3°), meperidine IV (7°) Infusion: Fentanyl, furosemide @ 5 mg/hr, nifedipine @ 1.25 ug/kg/min, NTG @ 1 ug/kg/min, dobutamine @ 1.25 ug/kg/min Total IVF Rate: 91.5 ml/hr
33A	35 yo, Male, BSA = 1.90 (98% IBW), <u>Midsleep</u> = 0500; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 10° <u>Vital Signs</u> : HR = 94 (Sinus), BP: 136/52/77; Temp: 37.3, RR: 20 (Spont), SaO2 98% (FiO2 = 0.28); CO/CI: 6.54/3.37; RAP: 13; SVR: 815; <u>CT</u> : 30 ml/3°; <u>CXR</u> : Catheter: RV outflow; right pleural effusion/haziness	Coarctation of the aorta (repair 1976), endocarditis with CHF, severe aortic insufficiency	Surgery: Ross CPB: 139 min IT: 104 min Hours since surgery: 20 Time of study: 0809-0857 Hours after midsleep: 3	Infusion: Magnesium, dopamine @ 0.99 ug/kg/min, lidocaine @ 2 mg/min, nifedipine @ 0.22 ug/kg/min Total IVF Rate: 52.6 ml/hr
34B	75 yo, Female, BSA = 2.12 (129% IBW), <u>Midsleep</u> = 0230; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 36° <u>Vital Signs</u> : HR = 70 (Intermittent VVI Pacer), BP: 124/43/61; Temp: 36.6, RR: 18 (Spont), SaO2 91% (FiO2 = 0.36); CO/CI: 6.7/3.2; RAP: 8; SVR: 749; <u>CT</u> : 25 ml/3°; <u>CXR</u> : Right PA, left lung field hazy	Aortic stenosis, IDDM, MI X 2	Surgery: AVR, CABG X 2 CPB: 205 min IT: 158 min Hours since surgery: 20 Time of study: 0926-1002 Hours after midsleep: 7	Atenolol-PO (30 min), docusate (30 min), levothyroxine (30 min), magnesium sulfate-IV (3°), isordil (30 min), metoclopramide (45 min) Infusion: Insulin, sodium phosphate, dopamine @ 2.0 ug/kg/min, nifedipine @ 1.0 ug/kg/min (titrated during study), nitroglycerin @ 0.25 ug/kg/min Total IVF Rate: 55.9 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time – Hrs before study)
35B	27 yo, Female, BSA = 1.43 (76% IBW), <u>Midsleep</u> = MD; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 12°, Tendelenburg 12° <u>Vital Signs</u> : HR = 94 (Sinus), BP: 108/67/81; Temp: 37.7, RR: 9/21(Vent), PEEP: 5, Vt: 560, PIP: 29, SaO ₂ : 100% (FiO ₂ = 0.30); CO/CI: 5.6/4.2; RAP: 4; SVR: 1100; <u>CT</u> : 100 ml/3°; <u>CXR</u> : Main PA; slight haziness left lower lobe	Severe AS, congenital tricuspid atresia	Surgery: Ross CPB: 156 min IT: 129 min Hours since surgery: 8 Time of study: 0812-0858 Hours after midsleep: MD	Vancomycin IV (4°) Infusion: Magnesium, fentanyl Total IVF Rate: 12 ml/hr
36A	55 yo, Male, BSA = 2.07 (119% IBW), <u>Midsleep</u> = 0300; <u>Bedrest</u> = MD; <u>Position</u> = Supine, HOB @ 15°, <u>Vital Signs</u> : HR = 97 (Sinus), BP: 103/49/67; Temp: 38.2, RR: 10/13(Vent), PEEP: 5, Vt: 900, PIP: 36, SaO ₂ : 94% (FiO ₂ = 0.50); CO/CI: 5.6/2.7; RAP: 15; SVR: 743; <u>CT</u> : 150 ml/3°; <u>CXR</u> : Main PA	CAD, angina, GERD, HTN	Surgery: CABG X 5 CPB: 102 min IT: 87 min Hours since surgery: 14 Time of study: 0818-0912 Hours after midsleep: 5	Furosemide IV (2°), cefazolin (15 min) Infusion: Fentanyl, potassium chloride, dopamine @ 2.0 ug/kg/min, NTG @ 0.25 ug/kg/min Total IVF Rate: 72.8 ml/jr
37A	59 yo, Male, BSA = 2.20 (142% IBW), <u>Midsleep</u> = 0030; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 31° <u>Vital Signs</u> : HR = 115 (Sinus Tach), BP: 108/52/70; Temp: 37.6, RR: 20, SaO ₂ : 95% (FiO ₂ = 0.36); CO/CI: 11.5/5.2; RAP: 12; SVR: 435; <u>CT</u> : 60 ml/3°; <u>CXR</u> : RV outflow; left lower lobe hazy without effusion, ? elevated right hemidiaphragm	Angina (new onset), DM, HTN, TIA, "back surgery", asbestosis	Surgery: CABG X 3 CPB: 93 min IT: 73 min Hours since surgery: 17 Time of study: 0820-0856 Hours after midsleep: 5.5	Cefazolin IV (2°), percocet (30 min), morphine IV (90 min) Infusion: Magnesium, dopamine @ 1 ug/kg/min, NTG 0.25 ug/kg/min Total IVF Rate: 23.4 ml/hr
38A	67 yo, Male, BSA = 2.04 (108% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 23° <u>Vital Signs</u> : HR = 87 (Sinus), BP: 126/42/70; Temp: 37.8, RR: 16, SaO ₂ : 99% (FiO ₂ = 0.40); CO/CI: 10.2/5.0; RAP: 3; SVR: 525; <u>CT</u> : 20 ml/3°; <u>CXR</u> : Left PA; bilateral pleural effusions	Aortic stenosis, CHF, Mitral regurgitation, aortic insufficiency, pulmonary HTN, COPD EF 25-34%	Surgery: AVR CPB: 180 min IT: 143 min Hours since surgery: 21 Time of study: 1021-1106 Hours after midsleep: 8.5	Furosemide – IV (2.5°), calcium gluconate IV (20 min), sucralfate (4.5°), vancomycin (1.5°), metoclopramide IV (during study) Infusion: Magnesium, nitroglycerin @ 0.5 ug/kg/min Total IVF Rate: 24 ml/hr

Subj Seq	Demographics/Vital Signs/Physiologic Data	Diagnosis	Procedure	Medications (Time - Hrs before study)
39B	84 yo, Male, BSA = 1.94 (131% IBW), <u>Midsleep</u> = 0500; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 16° <u>Vital Signs</u> : HR = 75 (Sinus), BP: 138/58/85; Temp: 37.1, RR: 20 (grunting), SaO ₂ : 94% (FiO ₂ = MD); CO/CI: 7.3/3.8; RAP: 10; SVR: 965; <u>CT</u> : 10 ml/3°; <u>CXR</u> : Main PA; poor inspiratory film - ? pleural effusion	Crescendo angina, HTN, exertional dyspnea, NIDDM, PUD, Inferior MI EF = 68%	Surgery: CABG X 4 CPB: 140 min IT: 93 min Hours since surgery: 18 Time of study: 0813-0821 Hours after midsleep: 3	Infusion: Insulin @ 3 u/hr, magnesium, NTG @ 0.5 ug/kg/min, dopamine @ 2 ug/kg/min Total IVF Rate: 32.2 ml/hr
40A	43 yo, Male, BSA = 2.08 (135% IBW), <u>Midsleep</u> = 0200; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 25° <u>Vital Signs</u> : HR = 94 (Sinus), BP: 113/51/69; Temp: 36.9, RR: 16, SaO ₂ : 97% (FiO ₂ = 0.32); CO/CI: 6.5/3.1; RAP: 12; SVR: 702; <u>CT</u> : 40 ml/3°; <u>CXR</u> : RV outflow; left pleural effusion	Aortic stenosis, GERD	Surgery: AVR CPB: MD min IT: MD min Hours since surgery: 18 Time of study: 0849-0930 Hours after midsleep: 6.5	Kefzol IV (30 min), furosemide IV (3°), percoet (10 min), metoclopramide IV (2.5°) Infusion: Magnesium, NTG 0.35 ug/kg/min Total IVF Rate: 22.1 ml/hr
41B	67 yo, Male, BSA = 2.08 (123% IBW), <u>Midsleep</u> = 0230 (naps); <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 19° <u>Vital Signs</u> : HR = 74 (Sinus), BP: 110/52/79; Temp: 36.8, RR: 20, SaO ₂ : 93% (FiO ₂ = 0.50); CO/CI: 7.4/3.6; RAP: 8; SVR: 822; <u>CT</u> : MD; <u>CXR</u> : Right PA; marked left pleural effusion, blunting right costophrenic border	CAD, CABG (1989), exertional angina, cerebrovascular disease, PTCA (1988, 89)	Surgery: Redo CABG X 4 CPB: 186 min IT: 126 min Hours since surgery: 14 Time of study: 0826-0842 Hours after midsleep: 6	Metoclopramide (start of study) Infusion: Magnesium, NTG @ 0.2 ug/kg/min, dopamine @ 1 ug/kg/min Total IVF Rate: 21.9 ml/hr
42A	67 yo, Male, BSA = 2.32 (142% IBW), <u>Midsleep</u> = 0300; <u>Bedrest</u> = 0; <u>Position</u> = Supine, HOB @ 38° <u>Vital Signs</u> : HR = 92 (Sinus with 1° heart block), BP: 106/41/61; Temp: 37.5, RR: 16, SaO ₂ : 100% (FiO ₂ = 0.50); CO/CI: 6.6/2.8; RAP: 8; SVR: 642; <u>CT</u> : 30 ml/3°; <u>CXR</u> : Right PA; blunting left costophrenic border, bilateral haziness	CAD, HTN, SOB, DOE, bilateral internal carotid stenosis, non-Q wave MI, CHF, GERD, s/p laminectomy, hypercholesterolemia	Surgery: CABG X 5 CPB: 208 min IT: 147 min Hours since surgery: MD Time of study: 0826-0852 Hours after midsleep: 5.5	Metoclopramide IV (2°), morphine (3.5°) Infusion: Magnesium, nitroglycerin @ 0.25 ug/kg/min, dopamine @ 1 ug/kg/min Total IVF Rate: 24 ml/hr

Abbreviations Used in Appendix G

AFib. Atrial fibrillation	IDDM. Insulin dependent diabetes mellitus
ASD. Atrial septal defect	IT. Ischemic time
AVR. Aortic valve replacement	LAE. Left atrial enlargement
BP. Blood pressure	LV. Left ventricular
BSA. Body surface area	LVE. Left ventricular enlargement
CA. Cancer	LVH. Left ventricular hypertrophy
CABG. Coronary artery bypass graft	MD. Missing data
CAD. Coronary artery disease	MI. Myocardial infarction
CHF. Congestive heart failure	MVR. Mitral valve replacement
CI. Cardiac index	NIDDM. Non-insulin dependent diabetes mellitus
CO. Cardiac Output	PA. Pulmonary artery
CP. Chest pain	PaO₂. Partial pressure arterial oxygen
CPB. Cardiopulmonary bypass time	PEEP. Positive end-expiratory pressure
CT. Chest tube output	PIP. Peak inspiratory pressure
CXR. Chest radiograph	PTCA. Percutaneous transluminal coronary angioplasty
DM. Diabetes mellitus	PUD. Peptic ulcer disease
DOE. Dyspnea on exertion	RAP. Right atrial pressure
EF. Ejection fraction	RBBB. Right bundle branch block
FiO₂. Fraction inspired oxygen	RR. Respiratory rate
GERD. Gastroesophageal reflux disease	RV. Right ventricular
HOB. Head of bed (degrees backrest elevation)	SaO₂. Arterial oxygen saturation
HR. Heart rate	SEM. Systolic ejection murmur
HTN. Hypertension	SOB. Shortness of breath
IBW. Ideal body weight (based on Quetelet Index)	Spont. Spontaneous ventilation

SvO₂. Mixed venous oxygen saturation

SVR. Systemic vascular resistance

Temp. Temperature (°C)

Vt. Tidal volume

VOO. Asynchronous pacemaker code

TIA. Transient ischemic attack

VOO. Asynchronous pacemaker code

APPENDIX H

Baseline Pressure Fluctuation

Pulmonary Artery and Pulmonary Artery Wedge Pressures in Individual Patients

(Summary)

Sequence A (S-R-L-S)					Sequence B (S-L-R-L)				
Subject	PAS (mm Hg)	PAED (mm Hg)	PAM (mm Hg)	PAW (mm Hg)	Subject	PAS (mm Hg)	PAED (mm Hg)	PAM (mm Hg)	PAW (mm Hg)
2	4	1	3	1	1	4	0	1	MD
4	4	4	2	1	3	4	2	5	4
6	3	3	4	2	5	5	3	2	MD
7	3	4	4	MD	10	4	3	9	0
8	2	2	2	1	12	4	3	6	2
9	1	7	3	MD	13	2	2	4	MD
11	6	4	6	MD	15	4	5	5	2
14	1	1	1	MD	16	2	1	0	MD
17	3	3	2	0	20	3	3	4	2
18	4	5	2	1	21	4	2	4	1
19	3	2	4	MD	22	2	3	2	MD
25	0	1	1	0	23	4	2	2	MD
26	5	1	1	3	24	2	3	3	MD
28	1	0	0	3	27	2	0	2	2
29	1	1	2	0	30	3	2	3	2
31	1	1	2	2	33	0	1	2	MD
32	1	1	4	MD	34	4	2	3	MD
36	3	1	2	MD	35	2	3	1	2
37	2	1	0	MD	39	1	2	2	1
38	5	1	1	3	41	1	2	1	1
40	3	3	4	MD					
42	1	2	2	MD					

Pulmonary Artery Systolic Pressure

Sequence A (S-R-L-S)					Sequence B (S-L-R-L)				
Subject	M1	M2	M3	M4	Subject	M1	M2	M3	M4
2	30	28	27	26	1	38	42	41	40
4	26	27	26	30	3	30	30	29	26
6	18	20	17	17	5	34	30	29	30
7	37	39	37	36	10	48	52	48	48
8	42	43	41	42	12	36	39	40	39
9	28	27	MD	28	13	MD	36	37	35
11	28	28	22	27	15	18	19	15	16
14	17	18	17	18	16	46	47	46	48
17	51	48	50	51	20	18	18	21	19
18	30	32	32	34	21	23	25	20	21
19	35	34	35	32	22	35	34	36	35
25	27	26	26	26	23	27	26	30	26
28	29	29	28	28	24	35	34	35	33
29	28	27	27	27	33	42	41	38	40
31	36	35	36	36	27	28	30	29	30
26	46	45	48	45	30	33	33	35	36
36	30	33	34	31	35	22	20	20	20
37	23	24	23	22	39	18	18	19	18
38	23	19	18	18	41	30	30	29	29
40	24	25	25	22					
42	32	32	31	MD					

Pulmonary Artery Mean Pressure

	Sequence A (S-R-L-S)					Sequence B (S-L-R-L)			
Subject	M1	M2	M3	M4	Subject	M1	M2	M3	M4
2	22	19	19	20	1	23	23	23	24
4	18	17	17	19	3	22	22	19	17
6	12	12	8	9	5	24	25	23	23
7	23	25	26	22	10	28	24	33	31
8	31	29	30	29	12	24	22	27	28
9	23	19	MD	20	13	MD	26	26	22
11	17	17	16	19	15	13	14	10	9
14	13	13	13	14	16	32	32	32	32
17	41	40	41	42	20	15	16	19	17
18	18	20	20	20	21	17	20	18	16
19	19	17	21	20	22	26	26	28	27
25	20	20	21	21	23	20	21	22	20
28	26	26	26	26	24	27	25	26	24
29	21	20	20	19	33	27	26	25	26
31	26	24	25	25	27	24	26	24	25
26	30	30	29	30	30	26	24	26	27
36	23	24	25	23	35	17	17	18	17
37	20	20	20	20	39	16	15	17	15
38	14	14	15	15	41	21	22	21	21
40	22	23	23	19					
42	23	21	22	MD					

Pulmonary Artery-End Diastolic Pressure

Sequence A (S-R-L-S)					Sequence B (S-L-R-L)				
Subject	M1	M2	M3	M4	Subject	M1	M2	M3	M4
2	16	16	15	15	1	20	20	20	20
4	14	12	11	15	3	14	13	12	13
6	8	9	6	6	5	20	21	20	18
7	19	19	18	15	10	21	21	24	24
8	21	23	23	23	12	18	18	19	21
9	20	13	MD	14	13	16	17	15	20
11	11	11	11	15	15	9	10	7	12
14	10	10	9	10	16	25	26	26	26
17	36	33	34	34	20	12	12	15	15
18	12	15	15	17	21	15	14	15	13
19	14	14	12	14	22	20	20	21	18
25	15	15	16	16	23	18	18	17	16
28	24	24	24	24	24	22	20	20	19
31	18	18	19	18	27	21	21	21	21
26	24	24	23	24	30	18	18	20	20
29	16	16	16	15	33	19	18	18	18
36	18	18	18	17	35	13	10	13	10
37	16	16	16	15	39	15	14	15	13
38	9	10	9	9	41	16	17	17	15
40	19	20	20	17					
42	18	16	18	MD					

Pulmonary Artery Wedge Pressure

Sequence A (S-R-L-S)					Sequence B (S-L-R-L)				
Subject	M1	M2	M3	M4	Subject	M1	M2	M3	M4
2	11	12	11	12	1	UTW	UTW	UTW	UTW
4	7	7	7	8	3	11	12	10	8
6	3	4	5	4	5	UTW	UTW	UTW	UTW
7	18	15	15	12	10	UTW	UTW	24	24
8	18	17	17	17	12	14	14	14	16
9	UTW	UTW	UTW	UTW	13	UTW	UTW	UTW	UTW
11	UTW	UTW	UTW	UTW	15	7	8	6	7
14	UTW	UTW	UTW	UTW	16	UTW	UTW	UTW	UTW
17	26	26	26	26	20	13	12	13	14
18	13	14	14	14	21	13	12	12	UTW
19	UTW	UTW	UTW	UTW	22	UTW	UTW	UTW	UTW
25	14	14	14	14	23	UTW	UTW	UTW	UTW
28	19	17	20	20	24	UTW	UTW	UTW	UTW
29	11	11	11	11	33	UTW	UTW	UTW	UTW
31	14	12	13	12	27	18	19	17	18
26	18	17	15	16	30	19	18	20	20
36	UTW	UTW	UTW	UTW	35	11	10	11	12
37	UTW	UTW	UTW	UTW	39	10	10	10	11
38	5	8	7	6	41	14	14	14	13
40	UTW	UTW	UTW	UTW					
42	17	UTW	UTW	UTW					

APPENDIX I

Individual Data: Pulmonary Artery and Pulmonary Artery Wedge Pressures

		PA Pressures (mm Hg)					
Subj #	Variable	S-1	Right	Left	S-2	Baseline Fluctuation	Comments
1B	PAS	MD	42	42	40	4	7 minute delay between M1 - M2 d/t sternal discomfort (not position induced)
	PAM	MD	33	30	29	1	
	PAED	MD	19	22	22	0	
	PAW	MD	MD	MD	MD	MD	
	ABP	MD	107/52/67	117/49/69	117/50/69		
	HR	MD	92	101	101		
	REF (cm)	14	11.5	9	14		
2A	PAS	27	MD	26	29	4	
	PAM	19	MD	19	20	3	
	PAED	14	MD	15	15	1	
	PAWP	11	MD	MD	12	1	
	ABP	85/45/59	85/54/66	89/52/65	88/46/60		
	HR	91	95	94	94		
	REF (cm)	9	9	7	9		
3B	PAS	36	39	38	38	4	Catheter-whip
	PAM	26	28	24	26	5	
	PAED	18	17	16	16	2	
	PAW	13	13	11	12	4	
	ABP	133/61/84	138/65/86	144/64/88	131/59/84		
	HR	86	90	88	85		
	REF (cm)	8.5	11	9	8.5		
4A	PAS	34	36	38	34	4	? Effect of variable reference height on position-left.
	PAM	21	22	27	21	2	
	PAED	15	16	20	14	4	
	PAWP	13	MD	14	MD	1	
	ABP	111/54/73	125/54/74	127/58/76	142/73/88		
	HR	86	86	87	83		
	REF (cm)	11.5	12.0	8.5	11.5		
5B	PAS	38	28	32	36	5	
	PAM	31	23	24	27	2	
	PAED	27	21	21	27	3	
	PAW	MD	MD	MD	MD	MD	
	ABP	102/68/80	100/67/80	103/67/78	109/70/82		
	HR	120	136	118	119		
	REF (cm)	9.5	12.5	11.5	9.5		
6A	PAS	24	27	24	23	3	Epinephrine and NTG titrated during study; milrinone off immediately before study (t ½ = 3°); PAS Δ may reflect Δ BP
	PAM	15	17	17	15	4	
	PAED	12	13	14	11	3	
	PAW	6	MD	8	8	2	
	ABP	139/58/86	132/61/87	146/64/90	139/62/88		
	HR	99	96	93	94		
	REF (cm)	11	11.5	11	11		
7A	PAS	43	48	51	49	3	PAS Δ possibly related to atrial fibrillation and irregular diastolic filling period and beat-to-beat variations in blood pressure
	PAM	30	35	34	35	4	
	PAED	21	24	24	24	4	
	PAW	MD	MD	MD	MD	MD	
	ABP	94/54/67	110/62/77	105/55/72	109/56/73		
	HR	89	92	96	97		
	REF (cm)	8.5	8.5	9.0	8.5		

8A	PAS	MD	53	50	47	2	
	PAM	MD	27	28	29	2	
	PAED	MD	22	21	21	2	
	PAW	MD	MD	18	MD	1	
	ABP	MD	110/50/71	117/50/73	130/51/72		
	HR	MD	80	74	70		
	REF (cm)	9	9.5	9.5	9.0		
9A	PAS	33	35	37	32	1	
	PAM	20	23	23	20	3	
	PAED	14	16	16	13	7	
	PAW	MD	MD	MD	MD	MD	
	ABP	Damped	Damped	Damped	Damped		
	HR	80	78	82	77		
	REF (cm)	8.0	10.	9.5	8.0		
10B	PAS	54	56	57	59	4	HR/BP changed in S2 (check BP measures)
	PAM	36	37	38	36	9	
	PAED	29	28	28	28	3	
	PAW	30	MD	30	32	0	
	ABP	104/44/60	99/44/56	99/40/55	136/47/69		
	HR	59	60	59	78		
	REF (cm)	9	11	9	9		
11A	PAS	36	33	38	35	6	Note difference in reference levels. Reference induced error?
	PAM	23	20	22	20	6	
	PAED	15	12	18	14	4	
	PAW	MD	MD	MD	MD	MD	
	ABP	116/65/82	120/70/89	126/69/90	121/66/84		
	HR	83	86	83	85		
	REF (cm)	9	11.5	8.5	9		
12B	PAS	47	49	48	48	4	
	PAM	31	32	34	33	6	
	PAED	23	24	25	24	3	
	PAW	19	21	22	20	2	
	ABP	87/50/64	85/58/69	92/56/67	90/58/70		
	HR	85	87	84	87		
	REF (cm)	9.5	10.5	9.5	9.5		
13B	PAS	39	MD	MD	MD	2	STUDY STOPPED after M5. Patient in left lateral position – started coughing with ? airway obstruction with tongue (continued with this problem throughout day in all positions). No sequelae.
	PAM	28	MD	MD	MD	4	
	PAED	20	MD	MD	MD	2	
	PAW	MD	MD	MD	MD	MD	
	ABP	109/54/72	MD	MD	MD		
	HR	82	MD	MD	MD		
	REF (cm)	12	MD	11.5	MD		
14A	PAS	21	18	24	18	1	Catheter whip
	PAM	16	14	17	13	1	
	PAED	12	11	13	9	1	
	PAW	MD	MD	MD	MD	MD	
	ABP	MD	MD	MD	MD		
	HR	90	90	93	88		
	REF (cm)	7	9.5	8	7		
15B	PAS	24	22	24	22	4	Catheter whip
	PAM	17	15	17	15	5	
	PAED	14	10	11	10	5	
	PAW	12	10	11	10	2	
	ABP	113/52/69	109/56/73	112/55/77	113/55/76		
	HR	79	85	82	85		
	REF (cm)	8	9	8	9		

16B	PAS PAM PAED PAW ABP HR REF (cm)	51 38 30 MD 94/55/70 96 11	60 44 34 MD 112/61/79 99 11	60 42 36 MD 103/61/76 99 11	58 42 33 MD MD 98 11	2 0 1 MD	OMITTED S1 DATA due to delay and demonstrated change in baseline (BP). Post-op bleed/hemodynamic instability. Delay after S1 (awaiting RN assistance). Increased BP may explain change in pressures S1-L/S1-R. Stabilization delay position-R (6 min) due to increased RR to 26; patient denied discomfort/SOB
17A	PAS PAM PAED PAW ABP HR REF (cm)	54 44 36 24 90/44/58 102 8.5	56 45 37 MD 90/49/61 105 12	53 40 31 26 79/49/56 105 10	57 45 36 26 86/45/58 105 8.5	3 2 3 0	Atrial fibrillation resulting in highly variable BP measurements. Postoperative bleed. FFP (3° before study).
18A	PAS PAM PAED PAW ABP HR REF (cm)	39 26 20 18 Damped 78 9	39 27 20 MD Damped 77 11	40 29 21 20 Damped 76 9	36 24 18 18 Damped 79 9	4 2 5 1	Variable PA baseline
19A	PAS PAM PAED PAW ABP HR REF (cm)	42 23 20 MD 106/47/69 62 7.5	42 23 20 MD 115/52/72 63 10.5	MD MD MD MD MD MD MD	MD MD MD MD MD MD MD	3 4 2 MD	STUDY STOPPED after M6 due to nausea (patient drinking fluids before study). Atrial fibrillation. Catheter whip - RV outflow tract
20B	PAS PAM PAED PAW ABP HR REF (cm)	24 22 18 MD 104/59/75 71 9	24 20 17 MD 96/54/72 72 10.5	27 23 21 MD 122/65/83 74 10	24 20 17 MD 105/56/74 72 9	3 4 3 2	↑ ABP-L may explain ↑ PAS-R. Damped waveform with marked variation in waveform configuration
21B	PAS PAM PAED PAW ABP HR REF (cm)	MD MD MD MD MD MD 8.0	MD MD MD MD MD MD MD	MD MD MD MD MD MD MD	MD MD MD MD MD MD MD	4 4 2 1	STUDY STOPPED after M5 due to nausea after 5 minutes supine. EXCLUDED (Patient drinking liquids before study). Post-op bleed treated with amicar (CT output = 1000 ml/8°). Bleeding controlled in 3° before study.
22B	PAS PAM PAED PAW ABP HR REF (cm)	36 27 23 MD 153/75/10 5 106 7.5	MD MD MD MD MD MD MD	37 30 24 MD 165/83/113 105 8.5	MD MD MD MD MD MD MD	2 2 3 MD	STUDY STOPPED after M6 due to discomfort. Postoperative bleed treated with platelets, FFP, whole blood and hespan. CT output post-op > 150 ml/hr.

23B	PAS PAM PAED PAW ABP HR REF (cm)	36 27 22 MD 90/50/64 78 11	35 28 24 MD 92/53/65 76 10.5	38 27 22 MD 102/57/73 78 11	36 26 22 MD 88/50/63 76 11	4 2 2 MD	Right bundle branch block Marked variability in waveform height
24B	PAS PAM PAED PAW ABP HR REF (cm)	37 28 20 MD 148/63/90 96 (paced) 9.5	36 29 21 MD 133/65/81 96 (paced) MD	42 35 24 MD 151/70/96 96 (paced) MD	36 29 20 MD 137/62/87 96 (paced) 9.5	2 3 3 MD	Variable ABP throughout study. Proximal port located in RV (detected by waveform; distal located in PA but unable to wedge. Catheter whip
25A	PAS PAM PAED PAW ABP HR REF (cm)	30 24 18 18 113/63/78 94 9	32 25 18 18 115/65/81 97 12.5	35 29 22 19 116/67/84 98 9	30 24 18 19 109/61/77 97 9	0 1 1 0	Measurement – left and right delayed 6+ minutes d/t increased BP. Postoperative bleed. (FFP – 500 ml – 6° before study). After study 2 units – PRBCs. 3.5 cm difference in reference height. Catheter whip
26A	PAS PAM PAED PAW ABP HR REF (cm)	46 33 24 18 Damped 94 7.5	45 32 24 18 106/60/77 86 9	48 34 27 20 112/57/77 92 9	45 32 24 17 88/49/63 86 7.5	5 1 1 3	Stopped after S-1 d/t nausea. HOB elevated to 10°. Variable recorded HR and BP throughout study d/t atrial fibrillation
27B	PAS PAM PAED PAW ABP HR REF (cm)	31 26 22 18 109/55/70 95 10.5	34 28 24 24 130/61/85 104 10	31 26 22 19 114/58/74 96 11.0	30 26 21 18 108/55/70 101 10.5	2 2 0 2	Suctioned before Right ↑ Pressures – most likely r/t suctioning. 12 minute delay after suctioning. Post-op bleed (2 units PRBC's completed 2 before study), decreased UOP – r/o renal failure
28A	PAS PAM PAED PAW ABP HR REF (cm)	24 22 20 17 82/44/62 99 10	29 26 24 22 104/52/70 97 10	24 20 18 14 109/57/70 99 10	24 20 18 18 84/53/64 97 10	1 0 0 3	IABP – removed 4° post-op BP labile PA waveforms highly variable, with marked change in amplitude throughout expiration
29A	PAS PAM PAED PAW ABP HR REF (cm)	27 20 15 15 116/57/75 89 7	36 28 20 20 117/60/78 89 8	33 24 18 18 112/56/74 89 7	29 23 17 17 105/54/69 89 7	1 2 1 0	DATA OMITTED Postoperative bleed: CT 860ml/12°; treated with 2 units PRBCs Suctioned after S1, probable cause of ↑ PA pressures. BP at baseline during all positions.
30B	PAS PAM PAED PAW ABP HR REF (cm)	35 26 20 20 105/63/79 89 6.5	38 30 24 24 101/65/80 89 8	37 29 22 23 96/61/79 89 7.5	37 30 21 16 98/62/78 89 6.5	3 3 2 2	PAW ≥ PAED

31A	PAS PAM PAED PAW ABP HR REF (cm)	38 27 19 14 108/58/74 79 10	42 30 22 16 112/64/80 79 11	42 30 21 16 115/64/82 79 10	39 30 20 16 109/61/77 79 10	1 2 1 2	Atrial Fibrillation, Pacemaker
32	PAS PAM PAED PAW ABP HR REF (cm)	28 20 15 MD 138/49/71 110 7.5	MD MD MD MD MD MD MD	MD MD MD MD MD MD MD	MD MD MD MD MD MD MD	1 4 1 MD	Study stopped d/t increased BP. (Family arrived for visit/discomfort in lateral position)
33B	PAS PAM PAED PAW ABP HR REF (cm)	30 28 20 MD 115/55/72 94 8.5	31 28 20 MD 121/63/77 97 9.5	35 31 22 MD 128/65/81 97 7.5	30 28 22 MD 123/63/78 98 8.5	0 2 1 MD	Catheter Whip
34B	PAS PAM PAED PAW ABP HR REF (cm)	42 27 21 MD 126/49/67 63 12	MD MD MD MD MD MD MD	42 29 21 MD 131/59/90 62 12	MD MD MD MD MD MD MD	4 3 2 MD	STUDY STOPPED after M6 (left) due to increased blood pressure requiring treatment with nipride. Patient received atenolol – PO 30 minutes before study, with a decrease in HR and CI (1.9) requiring pacing @ 70. Nipride off with ↓ BP secondary to ↓ HR. Nipride off after S-1 and restarted after position – left.
35B	PAS PAM PAED PAW ABP HR REF (cm)	20 18 11 11 100/63/77 96 4.5	20 18 14 14 102/67/79 95 6.5	23 19 13 13 103/62/80 96 4.4	21 17 13 13 116/71/86 95 4.5	2 1 3 2	
36A	PAS PAM PAED PAW ABP HR REF (cm)	32 24 19 MD 102/53/68 96 10	36 28 20 MD 95/59/69 99 10	36 28 24 MD 91/58/70 95 10	34 25 19 MD 101/56/69 96 10	3 2 1 MD	No external factors
37A	PAS PAM PAED PAW ABP HR REF (cm)	29 23 20 MD 104/60/72 96 11.5	32 23 19 MD 96/60/71 98 12.5	MD MD MD MD MD MD MD	MD MD MD MD MD MD MD	2 0 1 MD	STUDY STOPPED after M6 d/t discomfort. (Patient with history of chronic back pain treated with narcotics/anxiolytics).
38A	PAS PAM PAED PAW ABP HR REF (cm)	20 14 11 8 138/49/70 89 7.5	21 16 11 10 100/44/59 89 10.5	22 17 11 8 110/46/65 84 10	22 17 13 11 129/46/67 88 7.5	5 1 1 3	Arterial line markedly damped

39B	PAS	MD	MD	MD	MD	1	STUDY STOPPED after M3 due to vasovagal bradycardia (unrelated to study).
	PAM	MD	MD	MD	MD	2	
	PAED	MD	MD	MD	MD	2	
	PAW	MD	MD	MD	MD	1	
	ABP	MD	MD	MD	MD		
	HR	MD	MD	MD	MD		
	REF (cm)	10	MD	MD	MD		
40A	PAS	34	34	MD	MD	3	STUDY STOPPED after M6 due to discomfort. (Patient underwent emergent resternotomy in ICU 2 hours postoperative).
	PAM	29	30	MD	MD	4	
	PAED	25	27	MD	MD	3	
	PAW	MD	MD	MD	MD	MD	
	ABP	115/52/69	113/55/73	MD	MD		
	HR	95	94	MD	MD		
	REF (cm)	11.5	12.5	MD	MD		
41B	PAS	MD	MD	MD	MD	1	STUDY STOPPED after M4
	PAM	MD	MD	MD	MD	1	
	PAED	MD	MD	MD	MD	2	
	PAW	MD	MD	MD	MD	1	
	ABP	MD	MD	MD	MD		
	HR	MD	MD	MD	MD		
	REF (cm)	10.5	MD	MD	MD		
42	PAS	19	MD	MD	MD	1	STUDY STOPPED after M5 due to nausea (patient with history of GERD)
	PAM	17	MD	MD	MD	2	
	PAED	15	MD	MD	MD	2	
	PAW	12	MD	MD	MD	MD	
	ABP	108/43/64	MD	MD	MD		
	HR	92	MD	MD	MD		
	REF (cm)	12	MD	MD	MD		

APPENDIX J

PAIRED SAMPLE STATISTICS

Pulmonary Artery Systolic Pressure

Pair		Mean	N	SD	SEM
1	S1	33.23	26	9.56	1.88
	S2	33.50	26	10.25	2.01
2	Right	34.61	28	9.63	1.82
	S1	33.64	28	9.31	1.73
3	Left	35.64	28	9.33	1.76
	S1	33.64	28	9.36	1.77
4	Left	37.79	29	10.62	1.97
	Right	36.62	29	11.21	2.08
5	Right	36.62	29	11.21	2.08
	S2	35.76	29	11.24	2.08
6	Left	37.40	30	10.66	1.95
	S2	35.50	30	11.10	2.03

S1 = Supine-1 position, Right = 30-degree right lateral position, Left = 30-degree left lateral position, S2 = Supine-2 position.

Pulmonary Artery End-Diastolic Pressure

Pair		Mean	N	SD	SEM
1	S1	18.81	26	6.08	1.19
	S2	18.35	26	6.16	1.21
2	Right	19.75	28	6.03	1.14
	S1	19.29	28	5.89	1.11
3	Left	20.00	28	5.30	1.00
	S1	19.04	28	5.92	1.12
4	Left	20.79	29	5.90	1.10
	Right	20.21	29	6.41	1.19
5	Right	20.21	29	6.41	1.19
	S2	19.41	29	6.45	1.20
6	Left	20.60	30	5.89	1.08
	S2	19.23	30	6.41	1.17

S1 = Supine-1 position, Right = 30-degree right lateral position, Left = 30-degree left lateral position,
 S2 = Supine-2 position.

Pulmonary Artery Mean Pressure

Pair		Mean	N	SD	SEM
1	S1	24.62	26	7.05	1.38
	S2	24.58	26	7.28	1.43
2	Right	25.54	28	6.82	1.29
	S1	24.89	28	6.72	1.27
3	Left	26.25	28	6.68	1.26
	S1	24.79	28	6.81	1.29
4	Left	27.28	29	7.14	1.33
	Right	26.83	29	7.71	1.43
5	Right	26.83	29	7.71	1.43
	S2	26.00	29	7.79	1.45
6	Left	27.00	30	7.17	1.31
	S2	25.80	30	7.73	1.41

S1 = Supine-1 position, Right = 30-degree right lateral position, Left = 30-degree left lateral position, S2 = Supine-2 position.

Pulmonary Artery Wedge Pressure

Pair		Mean	N	SD	SEM
1	S1	16.06	16	6.03	1.51
	S2	16.88	16	6.26	1.56
2	Right	16.40	10	5.04	1.59
	S1	14.80	10	3.74	1.18
3	Left	17.06	16	6.40	1.60
	S1	16.19	16	5.94	1.48
4	Left	15.20	10	4.69	1.48
	Right	16.40	10	5.04	1.59
5	Right	16.40	10	5.04	1.59
	S2	15.10	10	3.38	1.07
6	Left	17.27	15	6.57	1.70
	S2	17.20	15	6.34	1.64

S1 = Supine-1 position, Right = 30-degree right lateral position, Left = 30-degree left lateral position,
S2 = Supine-2 position.

APPENDIX K

Position-Related Pressure Changes

PAS Pressure

Subject #	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2	Subject #	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2
1	MD	MD	MD	↑2	0	↓2	22	MD	MD	↑1	MD	MD	MD
2	↑1	MD	↓1	MD	MD	↑2	23	↓1	↓1	↑2	↑3	0	↓3
3	↑2	↑3	↑3	0	↓1	↓1	24	↓1	↓1	↑5	↑6	0	↓6
4	0	↑2	↑4	↑2	↓2	↓4	25	0	↑2	↑5	↑3	↓2	↓5
5	↓3	↓8	↓4	↑4	↑5	↑1	26	↓1	↓1	↑2	↑3	0	↓3
6	↑3	↑6	↓3	↓3	↓3	0	27	↓1	↑3	0	↓3	↓4	↓1
7	MD	MD	MD	↑3	↑1	↓2	28	0	↑5	0	↓5	↓5	0
8	MD	MD	MD	↓3	↓6	↓3	29	MD	MD	MD	MD	MD	MD
9	↓1	↑2	↑4	↑2	↓3	↓5	30	↑2	↑3	↑2	↓1	↓1	0
10	↑5	↑2	↑3	↑1	↑3	↑2	31	↑1	↑4	↑4	0	↓3	↓3
11	0	↓2	↑3	↑5	↑2	↓3	32	MD	MD	MD	MD	MD	MD
12	↓1	↑2	↑1	↓1	↓1	0	33	0	↑1	↑5	↑4	↓1	↓5
13	MD	MD	MD	MD	MD	MD	34	MD	MD	0	MD	MD	MD
14	↓3	↓3	↑2	↑5	0	↓5	35	↑1	0	↑3	↑3	↑1	↓2
15	↓2	↓2	0	↑2	0	↓2	36	↑2	↑4	↑4	0	↓2	↓2
16	MD	MD	MD	0	↓2	↓2	37	MD	↑3	MD	MD	MD	MD
17	↑3	↑2	↓1	↓3	↑1	↑4	38	↑2	↑1	↑2	↑1	↑1	0
18	↓3	0	↑1	↑1	↓3	↓4	39	MD	MD	MD	MD	MD	MD
19	MD	0	MD	MD	MD	MD	40	MD	0	MD	MD	MD	MD
20	0	0	↑3	↑3	0	↓3	41	MD	MD	MD	MD	MD	MD
21	MD	MD	MD	MD	MD	MD	42	MD	MD	MD	MD	MD	MD

S1 = Supine 1; S2 = Supine 2; **Bold** = clinically significant change; MD = Missing data

↓ = First pressure greater than second pressure; ↑ First pressure less than second pressure

PAED Pressure

Subject #	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2	Subject #	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2
1	MD	MD	MD	↑3	↑3	0	22	MD	MD	↑1	MD	MD	MD
2	0	MD	↑1	MD	MD	↓1	23	0	↑2	0	↓2	↓2	0
3	↓2	↓1	↓2	↓1	↓1	0	24	0	↑1	↑4	↑3	↑1	↓4
4	↓1	↑1	↑5	↑4	↓2	↓6	25	0	0	↑4	↑4	0	↓4
5	0	↓6	↓6	0	↑6	↑6	26	0	0	↑3	↑3	0	↓3
6	↑2	↑4	↑3	↓1	↓2	↓1	27	↓1	↑2	0	↓2	↓3	↓1
7	MD	MD	MD	0	0	0	28	↓2	↑4	↓2	↓6	↓6	0
8	MD	MD	MD	↓1	0	↓1	29	MD	MD	MD	MD	MD	MD
9	↓1	↑2	↑2	0	↓3	↓3	30	0	↑4	↑3	↓1	↓4	↓3
10	↓1	↓1	↓1	0	0	0	31	↑1	↑3	↑2	↓1	↓1	↓2
11	↓1	↓3	↑3	↑6	↑2	↓4	32	MD	MD	MD	MD	MD	MD
12	↑1	↑1	↑2	↑1	0	↓1	33	↓1	0	↑2	↑2	↓1	↓3
13	MD	MD	MD	MD	MD	MD	34	MD	MD	0	MD	MD	MD
14	↓3	↓1	0	↑1	↓2	↓3	35	↑2	↑3	↑2	↓1	↓1	0
15	↓4	↓4	↓3	↑1	0	↓1	36	0	↑1	↑5	↑4	↓1	↓5
16	MD	MD	MD	↑2	↓1	↓3	37	MD	MD	MD	MD	MD	MD
17	0	↑1	↓5	↓6	↓1	↑5	38	↑2	0	0	0	↑2	↑2
18	↓2	0	↑1	↑1	↓2	↓3	39	MD	MD	MD	MD	MD	MD
19	MD	0	MD	MD	MD	MD	40	MD	↑2	MD	MD	MD	MD
20	↓1	↓1	↑3	↑4	0	↓4	41	MD	MD	MD	MD	MD	MD
21	MD	MD	MD	MD	MD	MD	42	MD	MD	MD	MD	MD	MD

S1 = Supine 1; S2 = Supine 2; **Bold** = clinically significant change; MD = Missing data

↓ = First pressure greater than second pressure; ↑ First pressure less than second pressure

PAM Pressure

Subject #	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2	Subject	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2
1	MD	MD	MD	↓3	↓4	↓1	22	MD	MD	↑3	MD	MD	MD
2	↑2	MD	↑1	MD	MD	↑1	23	↓2	↓1	↑1	0	↓2	↓1
3	0	↑2	↓2	↓4	↓2	↑2	24	↑1	↑1	↑7	↑6	0	↓6
4	0	↑1	↑6	↑5	↑1	↓6	25	0	↑1	↑5	↑4	↓1	↓5
5	↓4	↓8	↓7	↑1	↑4	↑3	26	↓1	↓1	↑1	↑2	0	↓2
6	↑3	↑5	↑3	↓2	↓2	0	27	0	↑2	0	↓2	↓2	0
7	MD	MD	MD	↓1	0	↑1	28	↓2	↑4	↓2	↓6	↓6	0
8	MD	MD	MD	↑1	↑2	↑1	29	MD	MD	MD	MD	MD	MD
9	0	↑3	↑3	0	↓3	↓3	30	↑4	↑4	↑3	↓1	0	↑1
10	0	↑1	↑2	↑1	↓1	↓2	31	↑3	↑3	↑3	0	0	0
11	↓3	↓3	↓1	↑2	0	↓2	32	MD	MD	MD	MD	MD	MD
12	↑2	↑1	↑3	↑2	↑1	↓1	33	0	0	↑3	↑3	0	↓3
13	MD	MD	MD	MD	MD	MD	34	MD	MD	↑2	MD	MD	MD
14	↓3	↓2	0	↑2	↓1	↓3	35	↓1	0	↑1	↑1	↓1	↓2
15	↓2	↓2	0	↑2	0	↓2	36	↑1	↑2	↑4	↑2	↓1	↓3
16	MD	MD	MD	↓2	↓2	0	37	MD	0	MD	MD	MD	MD
17	↑1	↑1	↓4	↓5	0	↑5	38	↑3	↑2	↑3	↑1	↑1	0
18	↓2	↑1	↑3	↑2	↓3	↓5	39	MD	MD	MD	MD	MD	MD
19	MD	0	MD	MD	MD	MD	40	MD	MD	MD	MD	MD	MD
20	↓2	↓2	↑1	↑3	0	↓3	41	MD	MD	MD	MD	MD	MD
21	MD	MD	MD	MD	MD	MD	42	MD	MD	MD	MD	MD	MD

S1 = Supine 1; S2 = Supine 2; **Bold** = clinically significant change; MD = Missing data

↓ = First pressure greater than second pressure; ↑ First pressure less than second pressure

PAW Pressure

Subject #	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2	Subject #	S1-S2	S1-Right	S1-Left	Right-Left	Right-S2	Left - S2
1	MD	MD	MD	MD	MD	MD	22	MD	MD	MD	MD	MD	MD
2	↑ 1	MD	MD	MD	MD	MD	23	MD	MD	MD	MD	MD	MD
3	↓ 1	0	↓ 2	↓ 2	↓ 1	↑ 1	24	MD	MD	MD	MD	MD	MD
4	MD	MD	MD	MD	MD	MD	25	↓ 2	0	↑ 1	↑ 1	↓ 2	↓ 3
5	MD	MD	MD	MD	MD	MD	26	↓ 1	0	↑ 2	↑ 2	↓ 1	↓ 3
6	↑ 2	MD	↑ 2	MD	MD	0	27	0	↑ 6	↑ 1	↓ 5	↓ 6	↓ 1
7	MD	MD	MD	MD	MD	MD	28	↑ 1	↑ 5	↓ 3	↓ 8	↓ 4	↑ 4
8	MD	MD	MD	MD	MD	MD	29	MD	MD	MD	MD	MD	MD
9	MD	MD	MD	MD	MD	MD	30	↑ 3	MD	↑ 3	MD	MD	0
10	↑ 2	MD	0	MD	MD	↑ 2	31	↑ 2	↑ 2	↑ 2	0	0	0
11	MD	MD	MD	MD	MD	MD	32	MD	MD	MD	MD	MD	MD
12	↑ 1	↑ 2	↑ 3	↑ 1	↓ 1	↓ 2	33	MD	MD	MD	MD	MD	MD
13	MD	MD	MD	MD	MD	MD	34	MD	MD	MD	MD	MD	MD
14	MD	MD	MD	MD	MD	MD	35	↑ 2	↑ 1	↑ 1	0	↑ 1	↑ 1
15	↓ 2	↓ 2	↓ 1	↑ 1	0	↓ 1	36	MD	MD	MD	MD	MD	MD
16	MD	MD	MD	MD	MD	MD	37	MD	MD	MD	MD	MD	MD
17	↑ 2	MD	↑ 2	MD	MD	0	38	↑ 3	↑ 2	0	↓ 2	↑ 1	↑ 3
18	0	MD	↑ 2	MD	MD	↓ 2	39	MD	MD	MD	MD	MD	MD
19	MD	MD	MD	MD	MD	MD	40	MD	MD	MD	MD	MD	MD
20	MD	MD	MD	MD	MD	MD	41	MD	MD	MD	MD	MD	MD
21	MD	MD	MD	MD	MD	MD	42	MD	MD	MD	MD	MD	MD

S1 = Supine 1; S2 = Supine 2; **Bold** = clinically significant change; MD = Missing data

↓ = First pressure greater than second pressure; ↑ First pressure less than second pressure

APPENDIX L

Comparison of Characteristics of Patients with Clinically Significant Pressure Changes versus Patients without Clinically Significant Pressure Changes

Variables	Clinically Significant Changes (n = 8) Mean $\pm$ SD	No Clinically Significant Changes (n = 27) Mean $\pm$ SD
Age (years)	70.0 $\pm$ 13.5	64.5 $\pm$ 13
Sex		
-Male (n)	7	19
-Female (n)	1	9
% Ideal Body Weight (% Quetelet) ⁺	129 $\pm$ 22	122 $\pm$ 27
Bypass Time (min)	123 $\pm$ 37	154 $\pm$ 56
Ischemic Time (min)	97 $\pm$ 29	120 $\pm$ 49
Surgery		
-Coronary Artery Bypass Graft	3	15
-Aortic Valve Replacement	3	2
-Mitral Valve Replacement	0	3
-CABG & Valve Replacement	1	6
-Ross Procedure	0	2
-Atrial Septal Defect Repair	1	0
Grafts (n)	1.8 $\pm$ 2.0	2.9 $\pm$ 1.7
Hours after surgery	16.2 $\pm$ 4.3	17.7 $\pm$ 4.5
Chest Tube Output (ml/3°)	52 $\pm$ 33	88 $\pm$ 65
Pre-Study Backrest Elevation (degrees)	25 $\pm$ 10	23 $\pm$ 12
Midsleep (hour)	0246 $\pm$ 73	0214 $\pm$ 129
Days on Bedrest Before Surgery	0.75 $\pm$ 1.0	0.50 $\pm$ 1.2
Position of PA Catheter on Chest Radiograph		
Main PA	4	8
Right PA	4	11
Left PA	0	1
RV Outflow	0	8
Study Time		
Fluctuation	16 $\pm$ 1.5	16 $\pm$ 1.5
Position	25 $\pm$ 5	25 $\pm$ 8
Total	51 $\pm$ 5	49 $\pm$ 8

+ Based on medium body mass

Baseline Cardiopulmonary Indices

Variables	Clinically Significant Changes (n = 8) Mean $\pm$ SD	No Clinically Significant Changes (n = 27) Mean $\pm$ SD
Cardiac Output (L/min)	7.17 $\pm$ 3.45	6.37 $\pm$ 1.49
Cardiac Index (L/min/m ²)	3.52 $\pm$ 1.18	3.32 $\pm$ 0.83
Stroke Volume (ml/beat)	72 $\pm$ 27	75 $\pm$ 20
Heart rate (beats/minute)	98 $\pm$ 18	87 $\pm$ 11
Normal sinus rhythm	4	20
Sinus Tachycardia	1	2
Atrial Flutter/Fibrillation	2	2
Sinus Rhythm with Heart Block	0	0
Sinus Rhythm with Ectopy	0	0
Paced	1	4
Systemic Vascular Resistance (dynes/sec/cm ⁻⁵)	749 $\pm$ 249	802 $\pm$ 238
Systolic blood pressure (mm Hg)	119 $\pm$ 19	111 $\pm$ 15
Diastolic blood pressure (mm Hg)	59 $\pm$ 12	55 $\pm$ 11
Mean blood pressure (mm Hg)	78 $\pm$ 13	73 $\pm$ 13
Right atrial pressure (mm Hg)	11 $\pm$ 3	10 $\pm$ 3
Pulmonary Artery Temperature (°C)	37.1 $\pm$ 0.8	37.2 $\pm$ 0.5
Respiratory rate (breaths/minute)	19.0 $\pm$ 4	20 $\pm$ 4
FiO ₂	0.36 $\pm$ 0.1	0.37 $\pm$ 0.1
SaO ₂ (%)	95 $\pm$ 2	96 $\pm$ 3
Spontaneous Ventilation	8	24
Mechanical Ventilation	0	4
Pleural Effusion		
Yes	5	18
No	3	9