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University of Alberta

Timing of Hemodynamic Pressure Measurements and Thermodilutional Cardiac Outputs on Derived Hemodynamic Parameters

by

Gayle Urquhart 

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Nursing

Faculty of Nursing

Edmonton, Alberta

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled **Timing of Hemodynamic Pressure Measurements and Thermodilutional Cardiac Outputs on Derived Hemodynamic Parameters** by **Gayle Urquhart** in partial fulfillment of the requirements for the degree of **Master of Nursing**.

Dr. L. Jensen
Supervisor

Dr. K. Peters
Committee member

Dr. D. Humen
Committee member

DEDICATION

I would like to dedicate this work to my mother, Edna and to the memory of my father, Robert Urquhart. My parents have continuously provided me with support and have instilled the values of questioning and to strive for excellence.

ABSTRACT

The critical care nurse makes clinical judgements, based on thermodilution (TD) cardiac output (CO), hemodynamic pressures, and derived hemodynamic parameters. The relationship between hemodynamic pressures, derived hemodynamic parameters, and volume of TD CO injectate has not been studied. A repeated measures design was conducted with 21 cardiac surgical patients. Three hemodynamic pressures and derived hemodynamic parameters were recorded pre and post TD CO measurements (Set 1) and then repeated 30 minutes later (Set 2). Then the entire sequence was duplicated in 4 hours (Set 3 & 4). Of the derived hemodynamic parameters, pulmonary vascular resistance index at Set 1, and systemic vascular resistance index at Set 3 were significantly lower pre TD CO compared to post TD CO. Hemodynamic pressures and hence, derived hemodynamic parameters had minimal alterations over time. Thus the volume of TD CO injectate had minor effects on the hemodynamic pressures and hence, derived hemodynamic parameters.

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The staff of the cardiothoracic surgical unit I feel privileged to have worked with during the data collection process. The staff made me feel welcome at the bedside, even during times of extreme patient acuity. The staff also enabled me to wear two hats: a graduate student and a clinical nurse educator, which at time I believe they found frustrating.

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CHAPTER ONE

Introduction

The critical care nurse frequently makes clinical judgements based on the patient's hemodynamic pressures, consisting of blood pressure, right atrial pressure (RAP), pulmonary artery pressures (PAP), pulmonary artery wedge pressures (PAWP), and thermodilution (TD) cardiac output (CO). Derived hemodynamic parameters are calculated incorporating the hemodynamic pressures and TD CO measurements. Many authors have examined the techniques of obtaining TD CO and hemodynamic pressures, yet none have examined the relationship of the timing of the hemodynamic pressures and TD CO measurements to the derived hemodynamic parameters. Derived hemodynamic parameters consist of systemic and pulmonary vascular resistance index, stroke volume (SV), stroke volume index (SVI), and left and right ventricular stroke work index. An assumption is made that the measurements of hemodynamic pressures are valid and reliable indicators, which enables the nurse to make sound clinical judgements (Ahrens, 1998; Nelson & Houtchens, 1982; Pinsky, 1990). If the hemodynamic pressures obtained prior to the TD CO measurements reflect the steady state of the patient, these hemodynamic pressures would increase the accuracy of the hemodynamic parameters and hence treatment.

There is a proliferation of literature detailing the mechanisms to obtain accurate and reproducible hemodynamic pressures, and TD CO, which are then utilized to calculate the derived hemodynamic parameters. However, is it important whether the hemodynamic pressures are obtained before or after the TD CO measurements when calculating the

hemodynamic parameters? Should the hemodynamic pressures be obtained at a certain time to ensure accurate and reliable hemodynamic parameters? These questions have not previously been studied, which is of concern, as one must "do the right thing, as well as how to do the thing right" (Bryan-Brown & Dracup, 1993 p. 270). Also recently there has been an increased concern that critical care physicians and nurses lack a sufficient knowledge base about pulmonary artery catheters and the derived pressure measurements (Ahrens, & Taylor, 1998; Brandstetter, Grant, & Gitler, 1998; Brandstetter et al., 1998; Burns, Burns, & Shively, 1996; Dalen & Bone, 1996; Gnaegi, Feihl, & Perret, 1997; Grap, Pettrey, & Thornby, 1997). There is an impetus to ensure evidence-based practice as the basis of pulmonary artery pressure monitoring (Brandstetter, Grant, & Gitler, 1998; Sandham, Hull, & Brant, 1998; Vincent, Dhainaut, Perret, & Suter, 1998).

In current clinical practice, the nurse makes an independent decision as to when to obtain the required hemodynamic pressures. The effect of obtaining the hemodynamic pressures before or after TD CO measurements upon the calculated hemodynamic parameters requires exploration. If there is an alteration in the pressure volume loops due to injection of the solution required for TD CO measurements, the hemodynamic parameters may be different if calculated prior to rather than after TD CO measurements. Valid measurements are crucial, as the calculated hemodynamic parameters are used to direct medical and nursing care, which in turn impacts patient outcomes.

Purpose of the Study

The purpose of this study was to determine the effect of the timing of hemodynamic pressure measurements with TD CO measurements on the derived

hemodynamic parameters in cardiac surgical patients. The hemodynamic parameters of systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), right ventricular stroke work index (RVSWI), left ventricular stroke work index (LVSWI), SV, and SVI were calculated based on the hemodynamic pressures of mean arterial pressure (MAP), RAP, PAP, mean pulmonary artery pressure (MPAP), PAWP, and heart rate obtained prior to and after the completion of TD CO measurements. The following hypotheses were tested:

1. Hemodynamic pressures obtained prior to TD CO measurements in cardiac surgical patients will be lower than the hemodynamic pressures obtained after TD CO measurements.

2. Hemodynamic parameters calculated using the hemodynamic pressures obtained prior to the TD CO measurements in cardiac surgical patients will be lower when compared to the hemodynamic parameters calculated using the hemodynamic pressures obtained after TD CO measurements.

Significance of Study

The critical care nurse frequently makes clinical judgments based on the cardiovascular stability of the patient. The nurse determines the appropriate dosages of vasodilators, vasopressors, and inotropic agents based on the CO, cardiac index, and the derived hemodynamic parameters (Ramsey & Tisdale, 1995; Urban, 1986; Wilson, Bermingham-Mitchell, Wells, & Zachary, 1996). To date no other investigators have examined the relationship between hemodynamic pressures, derived hemodynamic parameters, and TD CO measurements, including the volume of injectate required for the

CO procedure. As well, in current practice there is no consistency as to when hemodynamic pressure measurements are recorded in relation to TD CO measurement. However, many investigators have studied the accuracy and reliability for the procedures of obtaining TD CO measurements, hemodynamic pressures, and derived hemodynamic parameters.

The findings of this study may provide support for the rationale of the timing of the hemodynamic pressures and TD CO measurements, increasing the accuracy and reliability of the derived hemodynamic parameters. The volume of injectate alters the pressure volume loops and vascular function curves, which then affects the hemodynamic pressures and derived hemodynamic parameters. The hemodynamic pressures obtained prior to the TD CO measurements reflect the physiological steady state of the patient. These hemodynamic pressures increase the accuracy of the hemodynamic parameters and may improve treatment. Consequently, the patients may require a decreased dosage of inotropes, vasodilators, and vasopressors, shortening their critical care unit length of stay.

CHAPTER TWO

Review of the Literature

The value of accurate and reliable hemodynamic monitoring, including hemodynamic pressure measurements, cardiac output (CO), and derived hemodynamic parameters, is required for several patient outcomes. These include the ability to: (a) assess perfusion, (b) detect inadequate perfusion early, (c) provide care based on the hemodynamic values, and (d) differentiate types of organ dysfunction (Brandstetter et al., 1998; Nelson, 1996). The following review of the literature will include hemodynamic pressures, CO, hemodynamic parameters, and the determinants of derived hemodynamic parameters.

Hemodynamic Pressures

Hemodynamic pressures are measurements of the dynamic moving forces of the cardiovascular system, which are then incorporated into the determinants of cardiac output and the derived hemodynamic parameters. The hemodynamic pressures consist of right atrial pressure (RAP), pulmonary artery pressures (PAP), pulmonary artery wedge pressure (PAWP), pulmonary artery systolic (PAS), pulmonary artery diastolic (PAD), mean pulmonary artery pressure (MPAP), mean arterial pressure (MAP), arterial systolic blood pressure (SBP), and arterial diastolic blood pressure (DBP).

Monitoring Hemodynamic Pressures

Within the critical care unit (CCU) milieu, the hemodynamic pressures may be obtained via the invasive technique and electrically recorded. A transducer converts the intravascular pulsatile flow into an electrical signal, which is displayed on a monitor as

millimetres of mercury (mm Hg). The monitor may display the hemodynamic pressures digitally or graphically, although it is highly recommended that intracardiac pressures be recorded graphically to eliminate respiratory influences and instrumentation issues (Cathelyn, 1997; Dolter, 1989; Gengiz, Crapo, & Garnder, 1983; Keckeisen, 1998; Lipp-Ziff & Kawanishi, 1991; Wilson et al., 1996). Cenziz, Crapo, and Garnder (1983) compared automated digital to graphic recordings of PAP and PAWP in spontaneous breathing and mechanically ventilated subjects. The digital PAP and PAWP were statistically lower in the mechanically ventilated group. Subjects on assist control did not have a statistical difference between the graphic and digital recordings. Subjects supported with an intermittent mechanical ventilator had statistically lower digital values, however, not as great as the spontaneously breathing subjects. Limitations of the study were: (a) unequal group size, (b) limited pressure determinations to less than four measurements, (c) transducers leveled to the mid-axilla line versus the mid-anterior-posterior of the thoracic cavity, and (d) no provision of sample inclusion and exclusion criteria.

Dobbin, Wallace, Ahlberg, and Chulay (1992) compared digital to graphic recordings of PAP in mechanically ventilated and spontaneous breathing patients. The authors determined that the digital and graphic recordings were not statistically significant different in mechanically ventilated patients. However, the PAD and PAWP were significant higher in the spontaneous breathing patients with the graphic recordings.The limitations of the study were: (a) lack of randomization to backrest elevation and graphic versus digital recordings, (b) no analysis of extraneous variables, which included airway pressure, positive end expiratory pressure, and hypoxia, and (c) not all subjects were

studied during spontenous breathing. Lundstedt (1997) compared repeated measures of digital, graphic, and stop cursor methods for determination of PAP, yet PAWP was not examined. Difference scores were calculated between the three methods of determination with clinical significance defined as greater than 2 mm Hg. Digital minus the cursor method was acceptable in 85% of systolic pressures and 74% of diastolic pressures for subjects receiving mechanical ventilation. The digital minus the graphic method was clinically significant in subjects mechanically ventilated for the systolic and diastolic pressures in 57% and 40% of the pressure determinations. In comparison subjects breathing spontaneously had lower rates of clinical significance. The digital minus the cursor method was clinically significant in 53% of the systolic pressure measurements and 37% of the diastolic pressure determinations. The digital minus the graphic method was clinically significant in 38% of the systolic pressure determinations and 12% of the diastolic pressure measurements. The recommendation was to utilize the graphic or stop cursor method to determine accuracy of any digital values. The limitations of the study were: (a) no reference to sample inclusion or exclusion criteria and demographic data, (b) no statistical analysis beyond difference scores, (c) sample size presented in the data table was different than the values presented in the discussion, (d) a significant clinical difference of $\pm$2 mm Hg was used instead of the standard difference of 5 mm Hg or PAS and 4 mm Hg for PAD (Nemens & Woods, 1982). Therefore, the recommendation remains to obtain intracardiac pressures using a graphic recording. Athough with the newer bedside monitors ability to ignore artifact, such as respiratory influence, the graphic versus digital recording issue needs to be revisited (Bridges & Woods, 1993; Dobbin et

al., 1992).

Instrumentation may affect the hemodynamic pressure measurement, as the monitor algorithm analyzes the highest and lowest point. However, the electronic equipment algorithm does not have the capability to differentiate artifact from a true value. Any pressure that is electronically obtained is exposed to this risk. The normal waveform of each hemodynamic pressure must be present prior to recording any measurement if one wants to ensure accurate values. There must be an analysis of the electronic system, and any variables that may affect the hemodynamic pressures, to ensure valid and reliable values. Catheter whip, zero-balancing, calibration, and the dynamic characteristics of resonant frequency and dampening coefficient may affect the accuracy of the system to reproduce the hemodynamic pressures (Biga & Bethel, 1991; Bridges & Middleton, 1977; Bridges & Woods, 1993; Cengiz, Crapo, & Garnder, 1983; Dolter, 1989;Gardner & Hollingworth, 1986; Gibbs & Gardner, 1988; Kiess-Daily & Schroeder, 1994; Mark, 1998; Quaal, 1988; Wiedemann, Matthay, & Matthay, 1984).

Zero-balancing and referencing are required to ensure that the hemodynamic pressures are relative to atmospheric pressures (Ahrens, Penick, & Tucker, 1995; Bridges & Woods, 1993; DeGroot & Damato, 1986; Dolter, 1989; Gawlinski, 1997). The recommendations are for zero-balancing to be performed: (a) a minimum of once a shift, (b) if the monitor is turned off or disconnected from the patient, or (c) if the hemodynamic pressures have altered drastically (DeGroot & Damato, 1986; Dolter, 1989; Keckeisen, 1998). Referencing is required with each patient position change. Zero-balancing and referencing require that the air-fluid interface of the transducer must be level with the right

atrium, which is defined as the phlebostatic level, as the effect of hydrostatic pressure is negated (Biga & Bethel, 1991; Bridges & Middleton, 1997; Bridges & Woods, 1993; Campbell, 1997; DeGroot & Damato, 1986; Dolter, 1989; Garnder & Hollingsworth, 1986; Gawlinski, 1997; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Klinger, 1996). For each one centimeter the air-fluid interface is below the phlebostatic level the hemodynamic pressures are increased by .74 mm Hg. The visa versus occurs if the air-fluid interface is above the phlebostatic level (Bridets, Bond, Ahrens, Daly, & Woods, 1997; Dolter, 1989; Keckeisen, 1998; Mark, 1998). All hemodynamic pressure measurements must be referenced to the phlebostatic level, since it is the central transmural pressure which is important (Bridges et al., 1997; Chulay, 1997; Mark, 1998).

Ahrens, Penick, and Tucker (1995) recently recommended that zero-balancing could be performed upon the initial set of readings, at 24 hours, or once a day. The recommendation was based on the discovery that transducers zero drift within the first initial 24 hours and that the zero drift rate is 1%. However, the limitations of the study were: (a) no indications as to whether the transducers were disposable or non-disposable, (b) no interrater and intrarater reliability reported, (c) a misunderstanding of the zero-balancing and levelling procedures, (d) no clear definition of the air-fluid interface for levelling of the transducer, (e) no statistical analysis beyond percentages, and (f) no rationale provided for defining zero drift as ± 2 mm Hg. Based on the limitations of the study, it is more feasible to perform zero-balancing a minimum of once a shift until there are further replication studies (Ahrens, Penick, & Tucker, 1995). Furthermore, electrical calibration of the system is required to prevent spurious measurements caused by ambient

temperature, variations in power voltage, alterations in atmospheric pressure, and damage to the electronic system, and should be performed once a shift (DeGroot & Damato, 1986; Dolter, 1989; Kiess-Daily & Schroeder, 1994). Precalibration of the transducer is performed by the manufacturers of disposable transducer, although manual calibration is required for non-disposable transducers (Kiess-Daily & Schroeder, 1994).

The dynamic characteristics of a physiological monitoring system are determined by the resonant or natural frequency and dampening coefficient (Bridges & Middleton, 1997; Bridges & Woods, 1993; Garnder & Hollingsworth, 1986; Gibbs & Gardner, 1988; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Mark, 1998; Quaal, 1995). The recommendations are for the dynamic characteristes to be assessed: (a) a minimum of once a shift, (b) with distorted or dampened pressure waveforms, and (c) after any opening of the system (Bridges & Middleton, 1997; Garnder & Hollingsworth, 1986; Gibbs & Gardner, 1988; Keckeisen, 1998; Quaal, 1995). The resonant frequency, the rate at which oscillations occur, is measured as hertz (HZ) and may be determined by the distance between the peaks with fast flush oscillations divided into the speed of the strip recorder (Ahrens, & Taylor, 1992; Bridges & Middleton, 1997; Bridges & Woods, 1993; Campbell, 1997; Gibbs & Gardner, 1988; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Mark, 1998; Quaal, 1995). As the resonant frequency decreases, so does the fidelity of the monitoring system. The resonant frequency should be at least 15 HZ for optimal fidelity and a value of less than 7.5 HZ is unacceptable (Gardner & Hollingsworth, 1986; Gibbs & Gardner, 1988; Mark, 1998).

The dampening coefficient, which is the time period required for a system to return

to baseline after stimulation, may be calculated by a complex formula, the determination of the amplitude ratio, or assessed visually (Bridges & Middleton, 1997; Gardner & Hollingsworth, 1986; Kiess-Daily & Schroeder, 1994; Mark, 1998). After the square wave test or the application of pressure using the flush device, there should be a small undershoot and then a petite overshoot, followed by rapid recovery to the hemodynamic pressure waveform. An underdampened waveform will exhibit greater than two oscillations, while an overdampened system will lack an overshoot (Gardner & Hollingsworth, 1986; Kiess-Daily & Schroeder, 1994). One may also visually assess for the occurrence of two to four transient oscillations, followed by rapid recovery to the hemodynamic pressure waveform after the application of the flush device (Dolter, 1989; Gibbs & Gardner, 1988). The amplitude ratio is determinded by the ratio of two oscillation. The ratio is then plotted on a graph with the natural frequency to determine the dampening coefficient (Bridges & Middleton, 1997; Bridges & Woods, 1993; Keckeisen, 1998; Mark, 1998). Dampening occurs when there is air, fluid, or mechanical obstruction present, loose connections, or alteration of the compliance of the system tubing inhibiting the normal system response (Biga & Bethel, 1991; Campbell, 1997; DeGroot & Damato, 1986; Dolter, 1989; Garnder & Hollingsworth, 1986; Kiess-Daily & Schroeder, 1994; Quaal, 1995). Loss of the dicrotic notch, elevated diastolic pressure, decreased systolic pressure, a poor waveform, and sloping of the square wave test are considered to be signs of overdampening (DeGroot & Damato, 1986; Dolter, 1989; Kiess-Daily & Schroeder, 1994; Keckeisen, 1998; Mark, 1998). Underdampening is characterized by rapid oscillations after the square wave test, elevated systolic pressure, decreased diastolic

pressure, and the presence of artificial wave components (Garnder, & Hollingsworth, 1986; Gibbs, & Garnder, 1988; Keckeisen, 1998; Mark, 1998). Frequency response and dampening coefficient should be available from all transducer manufacturers. Finally, catheter whip may be suspected with an unrealistic high hemodynamic pressure (DeGroot & Damato, 1986).

Arterial Blood Pressure

Arterial blood pressure reflects the blood flow multiplied by the resistance or compliance of that flow. The flow is reflected by CO and the compliance is determined by the systemic vascular resistance (Berne & Levy, 1992; DeGroot & Damato, 1986; Kiess-Daily & Schroeder, 1994). The SBP reflects the maximal pressure of ejection of the stroke volume (SV) during ventricular contraction, while the DBP mirrors the elastic recoil of the vascular system and the peripheral run-off pressure during ventricular relaxation. The SBP may be used to indirectly monitor afterload (Berne & Levy, 1992; DeGroot & Damato, 1986; Kiess-Daily & Schroeder, 1994; Meehan, 1986). The normal SBP is 90 to 140 mm Hg, while the typical DBP is 60 to 90 mm Hg (DeGroot & Damato, 1986; Kiess-Daily & Schroeder, 1994; Meehan, 1986).

The pressure of the arterial system averaged over the entire cardiac cycle is referred to as the MAP with a normal range of 70 to 90 mm Hg. The MAP is dependent on blood volume and arterial compliance, which are determined by CO and peripheral resistance (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Meehan, 1986). The calculation for MAP is [SBP + (2 x DBP)/3] (Kiess-Daily & Schroeder, 1994). As blood flow progresses down the arterial tree, the SBP increases and the DBP decreases, with no

overall alteration in the MAP. This difference in pressure is augmented with increased compliance and decreases with a reduction in compliance (Berne & Levy, 1992; Bridges & Middleton, 1997; Bryan-Brown & Dracup, 1993; Kiess-Daily & Schroeder, 1994).

Blood pressure may be measured via the direct and indirect techniques, each with unique factors that may affect the comparability between these arterial pressure measurements. Within the CCU milieu, blood pressure is typically measured by the invasive method, which is approximately 5 to 20 mm Hg greater than the indirect technique (DeGroot & Damato, 1986; Kiess-Daily & Schroeder, 1994). The indirect technique is based on flow, while the direct procedure is based on pressure (Bridges & Middleton, 1997; Campbell, 1997; DeGroot & Damato, 1986: Kiess-Daily & Schroeder, 1994). In comparing the invasive radial and the indirect brachial measurement, the SBP will be elevated and the DBP decreased in the former technique (Chyun, 1985; DeGroot & Damato, 1986). This discrepancy increases with an elevation in systemic vascular resistance index (SVRI), hypertension, occurrence of arrhythmias, valvular dysfunction, and hemodynamic instability, as there is a diminution of Korotkoff's sounds (Bryan-Brown & Dracup, 1993; Campbell, 1997; Chyun, 1985; DeGroot & Damato, 1986; Kiess-Daily & Schroeder, 1994). When comparing indirect and invasive measurements, the instrumentation of the pressure monitor must be evaluated, as the electronic equipment algorithm analyzes the highest and lowest values. It is recommended that the invasive and indirect blood pressure determinations should occur in the same extremity, taking into consideration that palpation correlates higher than auscultation with invasive blood pressure values (Berne & Levy, 1992; Chyun, 1985; DeGroot & Damato, 1986;

Kiess-Daily & Schroeder, 1994)

Cardiac Pressures

Frank-Starling's law explains the principle of preload, which determines the degree of contractility during systole based on end-diastolic volume (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Wiedemann et al., 1984). Preload is the degree of stretch of the myocardial fibers at end-diastole and is referred to as left ventricular end-diastolic volume (LVEDV) and right ventricular end-diastolic volume (RVEDV) (Calvin, Driedger, & Sibbald, 1981; Durham, Neunaber, Vogler, Shapiro & Muzuski, 1995; Ramsey & Tisdale, 1995; Urban, 1986). The larger the preload, the greater the stretch of the myocardial fibers and force of contractility. If the force of contractility increases, SV and CO should increase, although there is a limit to the relationship. At a certain point the contractility decreases with concommiant reduction in CO, even though preload is elevated (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Urban, 1986).

Preload volume is dependent upon: (a) diastolic filling pressure, (b) total blood volume, (c) distribution of blood volume, and (d) atrial systole. Diastolic filling time is affected by heart rate, atrial pressure, venous return, and blood volume. Distribution of blood volume is affected by venous return, body position, intrathoracic pressure, venous tone, and intrapericardial pressure (Kiess-Daily & Schroeder, 1994). The indicators of RAP, PAD, and PAWP are very sensitive to alterations in preload volume (Ramsey & Tisdale, 1995; Urban, 1986). Diuresis, vomiting, diarrhea, venoconstriction, venodilation, decreased diastolic filling time, hypervolemia, and hemorrhage are examples of clinical conditions that may alter the preload status quickly (Urban, 1986; Wiedemann et al.,

1984).

Within the CCU, the only mechanism to provide preload volume is with a special pulmonary artery catheter (PAC), which provides right ventricular ejection fraction, right ventricular end-diastolic and end-systolic volumes, and thermodilutional (TD) CO measurements (Beique & Ramsay, 1994; Diebel, Myers, & Dulchavsky, 1997; Durham et al., 1995; Kraut, Owings, Anderson, Hanowell, & Moorre, 1997; Nelson, 1996; Safcsak & Nelson, 1994; Wilson et al., 1996). Therefore, left ventricular end-diastolic pressure (LVEDP) and right ventricular end-diastolic pressure (RVEDP) are more commony used as preload indicators, as no special PAC is required. The RAP reflects RVEDP, while the PAD and PAWP mirror the LVEDP (Dolter, 1989; Durham et al., 1995; Kiess-Daily & Schroeder, 1994; Lipp-Ziff & Kawanishi, 1991; Meehan, 1986; Ramsey & Tisdale, 1995; Wiedemann et al., 1984). An assumption made is that the preload pressures approximate preload volume (Brandstetter et al., 1998; Calvin et al., 1981; Dolter, 1989; Mark, 1998; Nelson, 1996; Ramsey & Tisdale, 1995). It is important to realize that the preload volume is determined by the preload pressures of transmural ventricular distention and ventricular compliance (Beique & Ramsay, 1994; Cengiz, Crapo, & Garnder, 1983; Diebel et al., 1997; Dorinsky & Whitcomb, 1983; Durham et al., 1995; Kiess-Daily & Schroeder, 1994; Kraut et al., 1997; Wiedemann et al., 1984).

Transmural pressure is the difference in strain across a vessel or between internal and external forces. The transmural pressure is considered the true pressure of a vessel (Ahrens & Taylor, 1992; Klinger, 1996). Therefore, the transmural ventricular distention pressure is the intracardiac minus the extracardiac pressures, such as pleural or pericardial

pressure. As the pleural pressure increases the true transmural ventricular distention pressure will decrease in relation to the PAP, RAP, and PAWP. However, a transducer does not reflect the transmural distention pressure, as extracardiac pressues are not measured (Ahrens, 1995; Ahrens & Taylor, 1992; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Wiedemann et al., 1984).

Ventricular compliance is the pressure-volume relationship and reflects the change in pressure for any given alteration in volume (Beique & Ramsay, 1994; Calvin et al., 1981; Dolter, 1989; Dorinsky & Whitcomb, 1983; Mark, 1998). Ventricular compliance may decrease with pericardial disease, hypothermia, cardiopulmonary bypass, and myocardial ischemia and infarction, and may increase with an enlarged heart, valvular insufficiency, or intracardiac shunts (Beique & Ramsay, 1994; Calvin et al., 1981; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Mark, 1998; Ramsey & Tisdale, 1995; Wiedemann et al., 1984). Therefore, one should not automatically equate preload pressures and volume, without examining the components of ventricular compliance and transmural ventricular distention pressure (Calvin et al., 1981; Dolter, 1989; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Nelson, 1996; Wiedemann et al., 1984).

The PAC enables PAP, MPAP, PAS, PAD, PAWP, RAP, and CO measurements. The PAP reflects the pressure of the right and left ventricles (Klinger, 1996; Meehan, 1986). The PAS mirrors the pressure generated within the right ventricle, pulmonary bed, and left atrium, as there is an open channel with a normal value of 15 to 30 mm Hg

(Meehan, 1986; Nelson, 1996). The MPAP reflects the average pressure of the pulmonary bed and is calculated via the MAP formula [PAS + (2 x PAD) /3]. The PAD reflects the pressure of the pulmonary bed, left atria, and left ventricle and is normally 1 to 4 mm Hg greater than the PAWP. Therefore, the normal PAD is 8 to 15 mm Hg with a typical PAWP of 5 to 12 mm Hg (Biga & Bethel, 1991; Kiess-Daily & Schroeder, 1994; Meehan, 1986; Wiedemann et al., 1984). The PAD reflects the PAWP and LVEDP, unless the heart rate (HR) is greater than 120 beats per minute, pulmonary hypertension is present, or the PAC is placed within zone I or II of the lungs (Dolter, 1989; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Lipp-Ziff & Kawanishi, 1991; Wiedemann et al., 1984). With inflation of the PAC balloon, the resulting PAWP provides an indirect measurement of the LVEDP and the pulmonary capillary hydrostatic pressure (Biga & Bethel, 1991; Kiess-Daily & Schroeder, 1994; Wiedemann et al, 1984). An accurate PAWP requires that the balloon be inflated with .80 to 1.5 cubic centimetres (cc) of air to prevent eccentric balloon inflation. With eccentric balloon inflation the PAWP will be artificially elevated as their will be a reflection of the inflated balloon against the vessel wall versus the LVEDP (Beique & Ramsay, 1994; Dolter, 1989; Kiess-Daily & Schroeder, 1994; Wiedemann et al, 1984).

The vascular pressure equilibrates between the PAC and the left ventricle if there is an uninterrupted column of blood flow. During end-diastole the mitral valve is open and the PAWP, pulmonary venous pressure, left atrial pressure, and LVEDP are all equal (Beique & Ramsay, 1994; Biga & Bethel, 1991; Dolter, 1989; Kiess-Daily & Schroeder, 1994; Lipp-Ziff & Kawanishi, 1991; Wiedemann et al., 1984); however, there are variables

that may affect this relationship. When there is an obstruction between the PAC balloon and the left atria, there will be no equalization of pressures (Beique & Ramsay, 1994; Dolter, 1989; Wiedemann et al., 1984). The pulmonary venous pressure will be greater than the left atrial pressure. Thoracic tumours, atrial myxoma, positive end expiratory pressure (PEEP), and mediastinal fibrosis are sources of obstruction (Beique & Ramsay, 1994; Brandstetter et al., 1998; Dolter, 1989; Klinger, 1996; Wiedemann et al., 1984).

If left ventricular compliance is decreased, left atrial contraction will cause a large increase in LVEDP compared to the left atrial diastolic pressure (Beique & Ramsay, 1994). The LVEDP may be 5 mm Hg or more greater than the measured PAWP (Wiedemann et al, 1984). Aortic regurgitation, myocardial ischemia, and infarction may reflect a PAWP less than the true LVEDP. Mitral stenosis and regurgitation may reflect a PAWP greater than the true LVEDP, as the left atrial diastolic pressure is elevated (Beique & Ramsay, 1994; Dolter, 1989; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Lipp-Ziff & Kawanishi, 1991; Wiedemann et al., 1984).

Correct and accurate placement of the PAC requires the catheter to be placed within zone III of the lungs, as the PAP are greater than alveolar and venous pressures (Beique & Ramsay, 1994; Berne & Levy, 1992; Brandstetter et al., 1998; Bridges & Woods, 1993; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Wiedemann et al., 1984). With the inflation of the PAC balloon, there is an uninterrupted flow of blood between the PAC and the pulmonary veins. Consequently, a true PAWP and LVEDP should be reflected. If the PAC is within zone I or II of the lungs, the PAP and venous pressures are not equal. There is compression of one or both vessels because of the aveolar pressure

(Beique & Ramsay, 1994; Berne & Levy, 1992; Bridges & Woods, 1993; Kiess-Daily & Schroeder, 1994; Wiedemann et al., 1984). The PAWP will not reflect the true pressure of the left atria, as there is an interruption of blood flow between the PAC and the left atrium. Instead the PAWP will reflect the alveolar or PAP and not the left atrial pressure. This is will be reflected by the PAWP being greater than the PAD (Dolter, 1989; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). Zone III may be recruited into either zone I or II if the normal pulmonary artery-venous-alveolar relationship is altered. If PEEP is greater than 10 centimetres of water (cmH_20), the pulmonary capillaries are compressed (Dolter, 1989; Klinger, 1996; Wiedemann et al., 1984). Hypovolemia decreases the pulmonary venous pressure and the upright patient position increases the pulmonary venous pressure within the dependent regions of the lungs (Beique & Ramsay, 1994; Berne & Levy, 1992; Brandstetter et al., 1998; Dolter, 1989; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Paolella, Dorfman, Cronan, & Hasan, 1988; Quaal, 1988; Wiedemann et al., 1984).

Frequently the PAWP is employed to reflect the capillary pressure, which has led to the misnomer of pulmonary capillary wedge pressure (Beique & Ramsay, 1994). It is assumed that the PAWP has a fixed relationship with the pulmonary capillary hydrostatic and left atrial pressures, although this is not accurate. For example, hypoxia, sympathetic stimulation, and norepinephrine each have a different effect on the pulmonary capillary hydrostatic pressure (Wiedemann et al., 1984).

The development of pulmonary edema is dependent on pulmonary capillary permeability and the balance between the serum colloid oncotic and hydrostatic filtration

pressures (Wiedemann et al., 1984). The PAWP may provide an estimate of the capillary hydrostatic filtration pressure, while the serum colloid oncotic pressure may be directly measured; however, it is difficult to discern the level of pulmonary capillary permeability. An assumption is made that the PAWP will reflect the degree of capillary hydrostatic filtration pressure if the serum colloid oncotic pressure and capillary permeability are normal (Wiedemann et al., 1984). However, increased pulmonary capillary permeability is present with pulmonary disease, such as adult respiratory distress syndrome, which negates the PAWP and capillary hydrostatic pressure relationship. On average the PAWP may be employed to determine capillary hydrostatic pressure, although the nurse must be aware of the factors that may affect this relationship (Wiedemann et al., 1984).

Influence of Intrathoracic Pressure. There is a variation in the PAC pressures between spontaneous and mechanical breaths because of the alteration in the transmural distention pressure. During spontaneous inspiration the thoracic pressure is negative, reflected in the low PAP and RAP; however, there is a increase in the venous return and preload volume (Ahrens, 1995; Ahrens & Taylor, 1992; Berne & Levy, 1992; Cathelyn, 1997; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Shinn, Woods, & Huseby, 1979; Wilson et al., 1996). During mechanical inspiration the thoracic pressure is positive, which is reflected in the increased PAP, RAP, and pulmonary vascular resistance index (PVRI); however, there is a decrease in the venous return, preload volume, and transmural ventricular pressure (Ahrens, 1995; Berne & Levy, 1992; Cathelyn, 1997; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Shinn et al., 1979; Wiedemann et al., 1984; Wilson et al., 1996). The extent of the

elevation varies during an individual respiratory cycle. It is recommended that the PAP readings are averaged over one respiratory cycle rather than a single cardiac period (Kennedy, Bryant, & Crawford, 1984; Shinn et al., 1979; Quaal, 1988).

The transducer of the PAC is zero-balanced to atmosphere pressure to ensure accurate results (Ahrens & Taylor, 1992; DeGroot & Damato, 1986; Dolter, 1989; Gawlinski, 1997). This requires that the cardiac measurements occur when pleural and atmospheric pressures are equal and near zero, which is during end-exhalation. Cardiac measurements during end-expiration will reflect vascular and transmural pressure (Ahrens, 1995; Ahrens & Taylor, 1992; Beique & Ramsay, 1994; Biga & Bethel, 1991; Cathelyn, 1997; Keckeisen, 1998; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Lipp-Ziff & Kawanishi, 1991; Quaal, 1988; Wiedemann et al., 1984; Wilson et al., 1996). Obtaining a pulmonary artery pressure measurement at any other part of the respiratory cycle is equated with incorrect transducer placement (Cengiz, Crapo, & Garnder, 1983). End-exhalation has minimal alteration in venous return, least respiratory artifact, and is the period where intrapleural pressures should have least effect, therefore the RAP, PAP, and PAWP should reflect a steady state of the pressure volume loops (Ahrens, 1995; Ahrens & Taylor, 1992; Berne & Levy, 1992; Biga & Bethel, 1991; Brandstetter et al., 1998; Bridges & Woods, 1993; Cathelyn, 1997; Cengiz, & Crapo, & Garnder, 1983; Kiess-Daily & Schroeder, 1994; Wiedemann et al., 1984).

Positive end expiratory pressure alters the atmospheric and intrapleural relationship, as the latter pressure cannot be equilibrated with the former. The alteration in the pleural pressure may or may not equal the amount of PEEP, as this depends on the

amount of airway pressure reflected to non lung structures (Klinger, 1996). Positive end expiratory pressure may affect the RAP, PAP, and CO measurements because of decreased venous return and SV and an increased PVRI. Right ventricular dilation results and there is a shifting of the ventricular septum to the left (Diebel et al., 1997; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Nelson, 1996; Schuster et al., 1990). If the PEEP is greater than 8 cmH_20, the hemodynamic pressures of patients with heart failure will be affected and the cardiac index (CI) decreases significantly with PEEP at 16 cmH_20 (Kiess-Daily & Schroeder, 1994; Schuster et al., 1990). At the same time PEEP may not affect the cardiac measurements if the airway pressures are not transmitted to the heart and pleural space. The compliance of the lungs and thoracic cavity determine the effect of PEEP on the heart and pleural space (Klinger, 1996; Mark, 1998).

Influence of Patient Position. The pressures from the PAC should be obtained with the air-fluid interface of the transducer levelled, zero-balanced, and calibrated to the phlebostatic level, as this is the position of the right atrium when the patient is supine (Bartz, Maroun & Underhill, 1988; Biga & Bethel, 1991; Bridges & Middleton, 1997; Bridges & Woods, 1993; Dolter, 1989; Gawlinski, 1997; Winsor & Burch, 1945). It is interesting to note that the phlebostatic axis was originally defined as the plane located at the fourth intercostal space that transects with half the distance from the dorsal surface of the thorax and the xiphoid process, that is, mid anterior-posterior diameter (Winsor & Burch, 1945). Succeeding authors have defined the phlebostatic axis as either the mid anterior-posterior or mid-axilla diameter of the thorax, which makes it difficult to interpret

and compare study results (Bridges & Woods, 1993; Dolter, 1989; Groom, Frisch, & Elliott, 1990; Kennedy et al., 1984; Kraut et al., 1997; Laulive, 1982; Lipp-Ziff & Kawanishi, 1991; Lundstedt, 1997; Schuster et al., 1990; Shinn et al., 1979; Shinners & Pease, 1993). Secondly, the phlebostatic axis was originally defined for the right atrium (Winsor & Burch, 1945). Very few succeeding authors have limited their definition of the phlebostatic axis to only the right atrium (Biga & Bethel, 1991; Cline & Gurka, 1991; DeGroot & Damato, 1986; Dobbin et al., 1992; Gawlinski, 1997; Lipp-Ziff & Kawanishi, 1991; Quaal, 1988). There have been a limited number of authors that have tried to define the phlebostatic axis for the left atrium (Kee, Simonson, Stotts, Skov, & Schiller, 1993; Kennedy et al., 1985; Paolella et al., 1988). Therefore, with calibration, zero-balancing, and leveling the PAC to the right atrium there is the chance that the measured values do not represent true LVEDP.

Various patient positions and the influence on the PAC pressures have been examined. The flat supine position was compared to the supine and head of bed (HOB) elevated 20, 45, or 60 degrees by Laulive (1982). The largest change in PAP was from the 45 to the 60 degree angle. Laulive indicates that no clinical differences, defined as a change in pressures of 2 mm Hg, were noted. Subsequent authors examined statistical significance of positioning and determined that the supine position with the HOB less than 45 degrees provides accurate PAP (Chulay & Miller, 1984; Cline & Gurka, 1991; Dobbin et al., 1992; Groom et al., 1990; Lambert & Cason, 1990; Wilson et al., 1996). Various lateral positions and HOB elevations have been compared to supine, however, the phlebostatic axis was utilized for the supine transducer placement, and the fourth

intercostal space mid-sternum, left parasternal border, right sternal border, tricuspid area, mid-spine, or the dependent phlebostatic axis at mid-axilla for the lateral positions. Consequently, there are conflicting results reported for PAP and the lateral positions (Cason & Lambert, 1990; Groom et al., 1990; Keating, Bolyard, Eichler, & Reed, 1986; Kee et al., 1993; Kennedy et al., 1984; Lambert & Cason, 1990; Paolella et al., 1988; Ross & Jones, 1995; Shinners & Pease, 1993). The current recommendation is for the patient to be supine with the HOB elevated 0 to 45 degrees to ensure minimal influence of venous return and to enable accurate location of the phlebostatic axis until an appropriate transducer placement can be determined for lateral positions (Cason & Lambert, 1990; Chulay & Miller, 1984; Cline & Gurka, 1991; Groom et al., 1990; Keating et al., 1986; Keckeisen, 1998; Kee et al., 1993; Lambert & Cason, 1990; Laulive, 1982; Potger & Elliott, 1994; Ross & Jones, 1995; Shinners & Pease, 1993).

It is important to recognize that the normal fluctuation of the PAWP and PAD is less than 4 mm Hg, and for the PAS is less than 5 mm Hg (Nemens & Woods, 1982). The accepted normal fluctuation of the PAP will influence the recording of these pressures in relationship to patient position, PEEP, and mechanical ventilation. However, one could argue that each patient's normal fluctuation of hemodynamic pressures should guide the recording of the cardiac values (Lambert & Cason, 1990).

Cardiac Output

Cardiac output, the product of SV and HR, measured in liters/minute (l/min.), is determined by the: (a) preload pressures of RAP and PAWP, (b) afterload calculations of SVRI and PVRI, and (c) contractility values of stroke volume index (SVI), left ventricular

stroke work index (LVSWI), and right ventricular stroke work index (RVSWI) (Berne & Levy, 1992; Biga & Bethel, 1991; Bowdle, Freund, & Rooke, 1991; Hewlett Packard, 1985; Kiess-Daily & Schroeder, 1994; Meehan, 1986; Urban, 1986). The normal resting CO is 4 to 8 l/min. (Bowdle et al., 1991; Kiess-Daily & Schroeder, 1994; Meehan, 1986). Another parameter to assess CO is caridac index (CI), which is based on the CO value divided by the patient's body surface area (BSA). The CI is considered more accurate, as the CO is adjusted for BSA with a normal resting CI of 2.5 to 4 litres/minute/meter squared (l/min./m^2) (Biga & Bethel, 1991; Kiess-Daily & Schroeder, 1994; Sommers, Woods, & Courtade, 1993; Urban, 1986). Both CO and CI are only a sample of the factors that determine the amount of oxygen delivered to the tissues, consequently evaluation of CO and CI are often important in examining the effectiveness of patient interventions (Berne & Levy, 1992; Bowdle et al., 1991; Kiely, Byers, Greenwood, Carroll, & Carroll, 1998; Kiess-Daily & Schroeder, 1994; Meehan, 1986; Sommers et al., 1993).

Cardiac output is dependent on HR and SV, and SV is determined by preload, afterload, and contractility (Berne & Levy, 1992; Biga & Bethel, 1991; Bowdle et al., 1991; Kiess-Daily & Schroeder, 1994; Meehan, 1986; Urban, 1986). Consequently, any biological or technical event that affects any of the four determinants may alter the CO. It must therefore be recognized that any single CO measurement represents the net relationship between many variables, requiring the nurse to analyze all four determinants of CO. It is very rare that only one determinant of CO is altered without a modification of another factor (Berne & Levy, 1992; Bowdle et al., 1991).

Heart rate is the most effective and quickest way to alter the CO. An increase in HR can double or triple the CO in a healthy individual (Kiess-Daily & Schroeder, 1994; Meehan, 1986). Tachycardia may decrease CO because of decreased diastolic filling time and preload volume. There is a decrease in the end-diastolic stretch of the myocardial fibers with a reduction in the SV and CO (Berne & Levy, 1992; Biga & Bethel, 1991; Meehan, 1986). The preload volume may be dependent upon atrial and ventricular synchrony, as atrial contraction may increase the end-diastolic volume of the ventricles. During bradycardia, atrial contraction augments ventricular filling to a small degree; however, during tachycardia atrial contribution is very important to the preload volume of the ventricles. If there is no synchronization between the atriums and ventricles the end-diastolic volume will decrease, with a reduction in the myocardial fiber length, SV, and CO (Berne & Levy, 1992; Biga & Bethel, 1991; Meehan, 1986). Factors mentioned previously which affect preload are: (a) diastolic filling pressure, (b) total blood volume, (c) distribution of blood volume, (d) atrial systole, (e) transmural ventricular distention, (f) ventricular compliance, (g) intrathoracic pressure, and (h) patient position. Factors affecting afterload are: (a) blood viscosity, (b) radius and length of the vessels, (c) the condition of the cardiac valves, and (d) ventricular radius and wall tension. The sympathetic nervous system, electrolyte and acid-base imbalances, oxygen level, end-diastolic volume, degree of resistance and ventricular wall tension, and positive or negative inotropic agents are factors that affect contractility.

Measurement of Cardiac Output

Within the CCU, the gold standard for CO measurement is the intermittent TD

technique, which measures the CO of the right ventricle. It is assumed that the left and right ventricular CO's are similar, although the left ventricular CO curve lies below and to the right of the right ventricle (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994). When the right and left atrial pressures are equal, the right ventricle has a greater CO than the left ventricle, increasing the preload volume of the latter ventricle. There is increased myocardial fiber length of the left ventricle, with an augmentation of left ventricular CO until it matches the right ventricular CO. However, under stable conditions the left atrial pressure is greater than the RAP enabling equilibration of CO. It is the Frank-Starling mechanism that ensures the right and left ventricular COs are equal (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994). Consequently, with an alteration in preload volume of the right ventricle, TD CO may not immediately and accurately reflect the left ventricular CO.

Recently a continuous form of TD CO has been introduced to the CCU mileau (Beique & Ramsey, 1994; Garnder, 1998; Nelson, 1996). Although, it might be more accurate to refer to the measurement as semi-continuous, as the measurements are averaged over several seconds to minutes (Sequin et al., 1998). In comparisons of continuous and intermittent TD CO, the correlations range from r=.89 to .94 (Bottger et al., 1996; Dimyer, Shively, Burns, & Reichman, 1995; Jacquet, Hanique, Glorieux, Matter, & Goenen, 1996; Yelderman et al., 1992). However, the special PAC and computer used for continuous TD CO cost more than the PAC for intermittent TD CO, thus continuous TD CO is used infrequently (Bottger et al., 1996; Ditmyer et al., 1995; Guilbeau & Applegate, 1996). Therefore, TD CO will subsequently be used when

referring to intermittent TD CO and when required continuous TD CO is specified.

Thermodilution CO measurement, based on the Fick and indicator dilution principle and the Stewart-Hamilton equation, involves injection of an accurate known volume and temperature of an indicator solution and the formation of a time-temperature curve (Berne & Levy, 1992; Bowdle et al., 1991; Conway & Lund-Johansen, 1990; Kiess-Daily & Schroeder, 1994; Kadota, 1985; Levett, & Replogle, 1979). Thermodilution CO replaced dye indicator and Fick CO determination within the CCU due to having several advantages: (a) it can be performed quickly, (b) minimal equipment is required, (c) the PAC is utilized frequently within the CCU, and (d) the PAC provides TD CO, RAP, and PAP measurements (Berne & Levy, 1992; Bowdle et al, 1991; Weil, 1977). Although TD CO is the gold standard within the CCU, there are factors variables that affect the accuracy and reproducibility of TD CO. The factors that have to be considered include: (a) injectate temperature, volume, and solution, (b) technique of injection, (c) loss of solution indicator, (d) influence of intrathoracic pressures, and (e) patient position.

Injectate Temperature, Volume, and Solution. A variety of injectate temperatures and volumes have been examined extensively within a broad spectrum of critical illnesses. Initially, iced temperature injectate (ITI) was utilized, as the decreased temperature of the solution increased the signal-noise ratio and accuracy of TD CO values. Eventually, room temperature injectate (RTI) replaced ITI, as ITI: (a) required 45 to 60 minutes for equilibration; (b) needed a special ice bath; (c) involved awkward temperature measurement of the injectate solution; (d) presented a risk of thermal loss due to heat gain

from the operator's hands with handling of the syringe, as an one degree celsius ([c]) change leads to a 2.68% error in CO; (e) required injection within 30 seconds of removing from the ice bath, and (f) had increased risk of infection and bradycardia (Bourdillon & Fineberg, 1989; Kadota, 1985; Kiely et al., 1998; Levett, & Replogle, 1979; Nelson & Anderson, 1985; Nishikawa & Dohi, 1990; Riedinger & Shellock, 1984; Safcsak & Nelson, 1994). In comparisons of ITI and RTI, the correlations range from r=.90 to .989, providing support for the utilization of RTI (Barcelona et al., 1985; Bourdillon & Fineberg, 1989; Lyons & Dalbow, 1986, Nelson & Anderson, 1985; Nishikawa & Dohi, 1990; Price & Fowlow, 1993; Shellock, Riedinger, Bateman, & Gray, 1983; Vennix, Nelson & Pierpont, 1984; Wallace & Winslow, 1993). Recently authors (Kiely et al., 1998) compared RTI to ITI. However, the ITI was defined incorrectly, as 6 to 12 degrees Celsius.

Initially, 10 millimetres (ml.) of injectate was utilized, as larger volumes increased the signal-noise ratio (Barcelona et al., 1985; Kadota, 1985, 1986; Lyons & Dalbow, 1986; Pearl, Rosenthal, Nieslon, Ashton, & Brown, 1986; Shellock et al., 1983; Sommers et al., 1993). Succeeding authors examined 3, 5, and 10 ml. of injectate and determined that volumes of 5 and 10 ml. are considered accurate. Five ml. of ITI has greater accuracy than 5 ml. of RTI (r=.96 versus .89), therefore 10 ml. of solution should be utilized with RTI (Pearl et al., 1986). Three ml. should only be used in extreme cases and always with ITI, as the signal-noise ratio is lower with RTI (Elkayam et al., 1983; Pearl et al., 1986; Shellock et al., 1983).

Thermodilution CO is reported to have an inherent biological and technical error

of 5 to 20%, and as RTI has a greater variability than ITI, it is vitally important that there is accuracy of the injection technique (Burchell, Yu, Takiguchi, Ohta & Myers, 1997; Ditmyer et al., 1995; Elkayam et al., 1983; Levett & Replogle, 1979; Sasse, Chen, Berry, Sassoon, & Mahutte, 1994; Vennix et al., 1984). Thermodilution CO requires more than a single injection and an average of at least 3 values to ensure valid measurements (Ditmyer et al., 1995; Elkayam et al., 1983; Kadota, 1985, 1986; Levett & Replogle, 1979; Stevens, Raffin, Mihm, Rosenthal, & Stetz, 1985; Vennix et al., 1984).

The Stewart-Hamilton equation is based on the specific gravity and heat of dextrose 5% in water (D5W), resulting in the recommendation to utilize D5W as the injectate solution (Bowdle et al., 1991; Kadota, 1985; Kiess-Daily & Schroeder, 1994; Levett & Replogle, 1979). An interesting note is the number of investigators that have utilized normal saline as the injectate solution (Boerboom, Kinney, Olinger, & Hoffman, 1993; Bottger et al., 1996; Burchell et al., 1997; Calvin et al., 1981; Ditmyer et al., 1995; Doering & Dracup, 1988; Garnder, Monat, & Woods, 1987; Kiess-Daily & Mersch, 1987; Kraut et al., 1997; Lynch & Kaemmerer, 1990; Nelson & Houtchens, 1982; Pearl et al., 1986; Snyder & Powner, 1982; Stevens et al., 1990). Normal saline injectate alters the specific gravity and heat of the Stewart-Hamilton equation, which limits the ability to generalize the findings of the studies (Boerboom, Kinney, Olinger, & Hoffman, 1993; Calvin et al., 1981; Ditmyer et al., 1995; Doering & Dracup, 1988; Garnder, Monat, & Woods, 1987; Kiess-Daily & Mersch, 1987; Lynch & Kaemmerer, 1990; Nelson & Houtchens, 1982; Pearl et al., 1986; Snyder & Powner, 1982; Stevens et al., 1990).

Technique of Injection. The time interval between subsequent TD injections is

infrequently mentioned in the literature. There are reports of waiting 1 minute between injections when using 10 ml. of RTI, though the reason behind the delay is not indicated, nor if a stopwatch was used to time the delay (Biga & Bethel, 1991; Kiess-Daily & Mersch, 1987; Nelson & Anderson, 1985). The possibility exists that a 1 minute delay is required to stabilize the degree of thermal loss. The time interval to account for biological variation should be 20-180 seconds or when the CO computer displays a ready signal (Doering & Dracup, 1988; Kadota, 1985, 1986).

The indicator solution should be injected smoothly and quickly, resulting in a curve that exhibits a smooth, rapid upstroke and an even downslope that gradually returns to baseline (Bowdle et al., 1991; Conway & Lund-Johansen, 1990; Kiess-Daily & Mersch, 1987; Kiess-Daily & Schroeder, 1994; Levett & Replogle, 1979; Riedinger & Shellock, 1984; Sommers et al., 1993). Even though the recommendation is for the analysis of a CO curve to ensure reliable and valid values, very few investigators have reported if a CO curve was utilized (Boerboom et al., 1993; Bottger et al., 1996; Cline & Gurka, 1991; Davies, Jebson, Glasgow, & Hess, 1986; Driscoll, Shanaham, Crommy, & Gleeson, 1995; Gardner et al., 1987; Jacquet et al., 1996; Kadota, 1986; Kiely et al., 1998; Kiess-Daily & Mersch, 1987; Lyons & Dalbow, 1986; Price & Fowlow, 1993; Safcsak & Nelson, 1994; Sasse et al., 1994; Shellock et al., 1983; Snyder & Powner, 1982; Stevens et al., 1985; Wallace & Winslow, 1993). The CO injection should occur in less than 4 seconds, as there is an increased risk of underestimation of CO when greater than 8 seconds occurs (Biga & Bethel, 1991; Cline & Gurka, 1991; Doering & Dracup, 1988; Kadota, 1985; Kiess-Daily & Mersch, 1987; Okamoto et al., 1986; Price & Fowlow, 1993; Wallace & Winslow,

1993). Only two studies indicated that a stopwatch was employed to time the injection (Cline & Gurka, 1991; Driscoll et al., 1995). Manual versus the automatic technique of injection has also been examined. There has been no difference found between the two techniques if the manual procedure is uniform, steady, and consistent (Manifold, 1984; Nelson & Houtchens, 1982). There are only a few investigators that utilize the automatic technique of injection (Davies et al., 1986; Grose, Woods, & Laurent, 1981; Jansen, Schreuder, Settels, Kloek, & Versprille, 1990).

Loss of Indicator. The Fick principle is based on constant flow, complete mixing of injectate solution and blood and no loss of indicator solution temperature or volume during the CO process (Berne & Levy, 1992; Bowdle et al., 1991; Conway & Lund-Johansen, 1990; Kadota, 1985; Wiedemann et al., 1984). However, with TD CO, heat may be transferred from the PAC to the blood altering the indicator solution temperature. This loss of indicator temperature may overestimate CO by 3-12% (Kadota, 1985). It is possible that the first injectate has the greatest thermal loss and should be discarded from the calculation of the CO. The first injectate has been discarded in both RTI and ITI studies, yet the clinically accepted 4-10% variance was not employed (Lynch & Kaemmerer, 1990; Nelson & Anderson, 1985; Price & Fowlow, 1993; Wallace & Winslow, 1993). The reproducibility of CO using 10 ml. of ITI, comparing the first, second, and third injectate has been examined (Kadota, 1986). The first injectate was significantly higher than the second and third injectate, yet was within the accepted 10% range of variance. If the first injectate is significantly higher than the second and third, one should analyze the CO measurements and determine if more injections are required. If the

injections differ by less than 10% of each other or the median value, no measurement should be discarded (Kadota, 1986; Safcsak & Nelson, 1994). The assumption of TD CO is that any temperature change is because of the CO injectate solution. Therefore, any sources that affect baseline temperature may cause indicator loss. Sources of indicator loss are intracardiac shunts, tricuspid regurgitation, and rapid volume administration (Boerboom et al., 1993; Conway & Lund-Johansen, 1990; Heerdt, Pond, Blessios, & Rosenbloom, 1992; Wetzel & Latson, 1985).

Influence of Intrathoracic Pressure. The cyclic change in intrathoracic pressure may decrease the pulmonary artery temperature, due to the return of systemic blood. The increased venous return may decrease the baseline pulmonary artery temperature up to .086 degrees [c], thus lessening the TD CO signal-noise ratio (Bottger et al., 1996; Kiess-Daily & Mersch, 1987; Levett & Replogle, 1979; Shellock et al., 1983). Mechanical ventilation may increase the effect upon the TD CO signal-noise ratio and CO due to alterations in venous return, preload, and pulmonary afterload (Guilbeau & Applegate, 1996; Okamoto et al., 1986; Snyder & Powner, 1982; Stevens et al., 1985; Tajiri, Katsuya, Okamoto, Urata, & Sato, 1984). Injection at end-expiration in mechanically ventilated patients will increase reliability, but may overestimate CO by 1 to 1.5 times (Kiess-Daily & Schroeder, 1994; Sommers et al., 1993). The majority of authors examining TD CO performed injections at end-expiration because of the ease of timing and minimal cyclic changes on venous return, RAP, PAP, and pulmonary artery temperature, all of which may affect CO (Bottger et al., 1996; Bowdle et al., 1994; Burchell et al., 1997; Driscoll et al., 1995; Kadota, 1985; Kiely et al., 1998; Nelson &

Anderson, 1985; Pinsky, 1990; Quaal, 1988; Shinners & Pease, 1993; Sommers et al., 1993; Stevens et al., 1985).

The accuracy of CO measurement during mechanical ventilation may be increased if the injections are made at specific points along the entire respiratory cycle, as a true CO mean would be provided (Bottger et al., 1996; Bourdillon et al., 1989; Guilbeau & Applegate, 1996; Jansen et al., 1990; Kiess-Daily & Schroeder, 1994; Okamoto et al., 1986; Riedinger & Shellock, 1984; Sasse et al., 1994; Sommers et al., 1993; Snyder & Powner, 1982; Stevens et al., 1985). Examples of injection points are: (a) even spaced intervals during the entire respiratory cycle, (b) mid-inspiration and mid-exhalation, (c) peak-inspiration and end-exhalation, and (d) mid or end-inspiration and mid-exhalation. There are conflicting suggestions for the appropriate respiratory point of TD CO injection, as both mid and end inhalation and exhalation are cited as the points of highest CO (Daper, Parquier, Preiser, Contempre, & Vincent, 1986; Okamoto et al, 1986; Snyder & Powner, 1982; Stevens et al., 1985; Tajiri et al., 1982). Two specific point measurements obtained one-half a ventilator cycle apart, have been found to be as accurate as four random measurements (Jansen et al., 1990; Stevens et al., 1985). Therefore, there are conflicting results regarding the injection point, with no clinical studies incorporating any of these recommendations to increase accuracy of TD CO.

Influence of Patient Position. Early studies determined that TD CO was accurate with the patient supine and the HOB elevated 20 degrees (Grose et al., 1981; Sommers et al., 1993). The flat supine position was compared to the HOB elevated 45 degrees by Driscoll et al (1995). The 45 degree position caused statically lower CO values and 40%

of these cases decreased by greater than the accepted clinical variation of 10%. It is interesting to note that Driscoll et al (1995) believe the alteration in CO measurements is a true value and not a measurement error. This is based on the non significant statistical alteration in RAP and PAWP.

Subsequently, lateral and supine positions have been compared with the HOB elevated 20 to 30 degrees. There is no consistency of results, other than the left lateral position tends to yield the highest CO (Cline & Gurka, 1991; Doering & Dracup, 1988; Gawlinski, 1997; Whitman, Howaniak, & Verga, 1982). Based on the findings of the above studies, TD CO should be performed with the patient supine and the HOB elevated less than 30 degrees. Only a few TD CO or hemodynamic pressure measurement studies have reported the patient's position (Driscoll et al., 1995; Gardner et al., 1987; Grose et al., 1981; Kiess-Daily & Mersch, 1987; Lyons & Dalbow, 1986; Price & Fowlow, 1993; Shellock et al., 1983; Wallace & Winslow, 1993; Whitman et al., 1982).

The majority of authors examine the statistical significance of TD CO results, although they rarely interpret the results in regards to the accepted clinical 4-10% variation (Driscoll et al., 1995; Kadota, 1985; Lynch & Kaemmerer, 1990; Sasse et al., 1994; Vincent, 1994). Clinical, as well as statistically significant results should be interpreted and compared, to ultimately guide patient outcomes.

Hemodynamic Parameters

Hemodynamic parameters are calculations of the dynamic moving force of the cardiovascular system and incorporate the hemodynamic pressures, CO, and CI measurements. The hemodynamic parameters are indicators of the afterload and

contractility determinants of CO and consist of SVRI, PVRI, and LVSWI, RVSWI, and SVI, respectively.

Afterload Indicators

Afterload is the impedance or resistance to blood flow from the right and left ventricles during systole and is measured in absolute resistance units indexed (dynes/sec/cm^{-5}/m^2). As afterload increases the SV decreases, and if the resistance lessens there is a elevation in the SV (Beique & Ramsay, 1994; Hewlett Packard, 1985; Kiess-Daily & Schroeder, 1994; Meehan, 1986; Ramsey & Tisdale, 1995; Urban, 1986). The resistance to ejection of the LVEDV into the aorta is referred to as SVRI and is based on the formula of (MAP - RAP) x 79.96 / CI or systemic vascular resistance x BSA. Systemic vascular resistance is based on the formula of (MAP - RAP) x 79.96 / CO. The normal SVRI is 1970 to 2390 (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994). The PVRI reflects the resistance of blood flow into the lungs and is calculated as (MPAP - PAWP) x 79.96 / CI or pulmonary vascular resistance x BSA. Pulmonary vascular resistance is based on the formula of (MPAP - PAWP) x 79.96 / CO. The normal PVRI is 225 to 315 (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994).

An indirect measurement of afterload is SBP, although the variables of CO, arterial compliance, and the dynamics of the electronic system affect the accuracy. The degree of afterload is affected by blood viscosity, radius and length of vessels, condition of the cardiac valves, and ventricular radius and wall tension (Berne & Levy, 1992; Biga & Bethel, 1986; Klinger, 1996). Therefore, hypoxia, arterial and venous dilation and constriction, valvular stenosis and regurgitation, hemoconcentration, PEEP, and shock

may affect afterload (Biga & Bethel, 1991; Meehan, 1986; Klinger, 1996; Urban, 1986).

Contractility Indicators

Contractility is the inherent ability of the myocardium to alter the force and length of the myocardial fibers during systole independent of preload and afterload (Berne & Levy, 1992; Bridges & Woods, 1998; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995). Contracitlity is difficult to measure within clinical practice, although it may be calculated. Stroke volume, which is the volume of blood ejected with each heart beat may provide an non-specific indication of contractility. Stroke volume is determined by CO / HR x 1000 with a normal value of 60 to 120 ml./beat (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995; Urban, 1986). The SV may be adjusted for BSA by the calculation of SVI. The formula is SV / BSA with a normal value of 30 to 60 ml./beat/m^2 (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995; Urban, 1986). The amount of exertional work performed with each heart beat against aortic or pulmonic impedance adjusted for BSA is referred to as stroke work (Ramsey & Tisdale, 1995). The specific indicators of contractility are RVSWI and LVSWI, and are measured as grams (gr)-m/m^2/beat (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995). The exertional work performed by the right ventricle is referred to as RVSWI and the formula is SVI x (MPAP - RAP) x .0136. The normal RVSWI is 4 to 8 gr-m/m^2/beat (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995; Urban, 1986). A similar value is obtained for the left ventricle referred to as LVSWI, and calculated by SVI x (MAP - PAWP) x .0136. The typical LVSWI is 40 to 75 gr-m/m^2/beat (Hewlett Packard,

1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995; Urban, 1986).

As the RVSWI and LVSWI incorporate SVI and change in pressure, an assessment can be made of the relationship between cardiac performance and vasculature (Beique & Ramsay, 1994). Yet it is important to realize that preload and afterload parameters are incoporated within the calculation of LVSWI and RVSWI, therefore, the latter are not true reflections of contractility (Bridges & Woods, 1998). As the RVSWI and LVSWI values decrease, there is myocardial dysfunction (Calvin et al., 1981). Variables that affect contractility are: (a) the sympathetic nervous system, (b) positive or negative inotropic agents, (c) electrolyte and acid-base imbalances, (d) oxygenation status, (e) end-diastolic volume or preload, and (f) the degree of resistance and ventricular wall tension (Berne & Levy, 1992; Biga & Bethel, 1991; Calvin et al., 1981; Halfmann-Franey, 1988; Kiess-Daily & Schroeder, 1994; Urban, 1986).

Determinants of Derived Hemodynamic Parameters

Pressure Volume Loops

Interdependent pressure volume loops provide an explanation for the variability in end-diastolic and end-systolic pressures and volumes based on preload, afterload, and contractility (Berne & Levy, 1992; Klinger, 1996; Mark, 1998; Meehan, 1986). As the preload volume of the ventricles increase, the loops are shifted upwards and to the right. There is an increase in the end-diastolic volume, with a resulting augmentation of SV, enabling a constant end-systolic volume. The peak systolic pressure should not be altered, as afterload has not been affected. If the afterload increases, the loop shifts upwards, as there is an increase in peak systolic pressure. Because of the increased resistance to

outflow, the SV decreases and the end-systolic volume increases. An increase in contractility shifts the loops upwards and to the left. There is an increase in the SV and peak systolic pressure, and decrease in end-systolic volume (Berne & Levy, 1992; Klinger, 1996).

Thermodilution CO measurements, with the injection of 30 to 50 ml. of fluid, may increase the RAP and PAWP preload measurements due to an alteration of intracardiac blood volume. There may be an increase in the end-diastolic volume shifting the pressure volume loop to the right, increasing the SV and CO (Berne & Levy, 1992; Klinger, 1996). Secondly, due to the increased volume from the TD CO measurements, the HR may increase or decrease from the initiation of the Bainbridge reflex (Berne & Levy, 1992). When bradycardia exists, an increase in the preload volume may increase HR. When tachycardia exists an increase in the preload volume may decrease HR. The net effect may be an alteration in the SV and CO (Berne & Levy, 1992). Ventricular compliance is a change in pressure for any given alteration in volume. Thermodilution CO, with the injection of 30 to 50 ml. of fluid, may increase the cardiac pressures for a given volume to a greater degree for a non-compliant ventricle (Dolter, 1989; Laulive, 1982). Thus, due to the Frank-Starling law, the end-diastolic volume, SV, and CO may increase due to the alteration in preload from the TD CO solution (Berne & Levy, 1992). It must be stressed that TD CO measures the CO of the right ventricle, although it is assumed that the left ventricular CO is similar too the right ventricle (Berne & Levy, 1992).

Vascular Function Curve

The vascular function curve defines the alteration in preload due to the change in

CO. As the CO increases, there is a greater fraction of the total blood volume within the arterial system compared to the venous vessels. This is reflected in a decreased central venous pressure. When venous pressure falls below the required opening level of venous vessels CO will fall because of decreased venous return. The vascular function curve is dependent upon peripheral resistance, blood volume, and arterial and venous compliance (Berne & Levy, 1992). The CO increases with an elevated preload, which may occur with TD CO measurements. The intersection of the pressure volume loops and vascular function curve determines the steady state of CO and RAP (Berne & Levy, 1992). Thermodilution CO measurements may disrupt this relationship, as the heart is trying to equilibrate due to the increased volume that occurs during the TD CO procedure.

Therefore, due to the possible increase in RAP and PAWP from the injection of the TD CO solution, the hemodynamic parameters of SVRI, PVRI, LVSWI, RVSWI, and SVI may be lower than the parameters calculated using the RAP and PAWP obtained prior to the performance of TD CO. Hemodynamic pressures obtained prior to the TD CO measurements may enable calculated hemodynamic data to be based on a steady clinical state, as compared to the pressures obtained after the TD CO is measured. No studies to date have examined this relationship.

Influence of Intrathoracic Pressure

As the heart and lungs are enclosed in a common compartment, pleural pressure may also affect the pressure volume loops. This is particularly noteworthy with mechanically ventilated patients. Mechanical ventilation and the alteration in peak airway pressure, respiratory rate, and inspiratory-expiratory ratio may alter pulmonary blood

flow, preload, right ventricular afterload, and ventricular geometry. The increased pleural pressure may: (a) decrease venous return, RAP, and PAWP, (b) increase cardiac pressures if transmitted to the intrathoracic vascular system, (c) increase PVRI due the elevated pulmonary pressures, and (d) decrease the size and compliance of the left ventricle (Dorinsky & Whitcomb, 1983; Okamoto et al., 1986; Shinn et al., 1979; Stevens et al., 1985). Consequently, pleural pressure might also be a factor affecting the relationship of hemodynamic pressure measurements and the derived hemodynamic parameters.

Summary

Hemodynamic pressure measurements, TD CO, and derived hemodynamic parameters are utilized frequently by the critical care nurse to guide therapy and achieve the endpoints of hemodynamic monitoring. The derived hemodynamic parameters along with analysis of oxygen suppply, demand, and consumption frequently determine the type of therapy the critically ill patient requires. Therefore, it is vitally important to determine the relationship of the timing of the hemodynamic pressure measurements to TD CO and the derived hemodynamic parameters. Thermodilution CO may be performed with either ITI or RTI, if the injections occur over 4 seconds and are timed at end-expiration, with a minimum level of PEEP. The patient should be supine with the HOB elevated less than 30 degrees. The accuracy and reliability of the hemodynamic parameters are dependent upon the recording of the pressures with the air-fluid interface of the transducer level to the mid-anterior-posterior phlebostatic level. Events such as patient position and intrathoracic pressure that affect the accuracy of TD CO values will affect the hemodynamic pressures to a similar degree, as CO is based on the determinants of preload, afterload, contractility,

and HR. The hemodynamic pressures should be obtained under the same conditions as TD CO, minimizing the affects on the pressure volume loops.

CHAPTER THREE

Method

The purpose of this study was to determine the effect of the timing of hemodynamic pressure measurements with thermodilutional (TD) cardiac output (CO) measurements on the derived hemodynamic parameters in cardiac surgical patients. The hemodynamic parameters of systemic vascular resistance index, pulmonary vascular resistance index, right ventricular stroke work index, left ventricular stroke work index, stroke volume (SV), and stroke volume index (SVI) were calculated based on the hemodynamic pressures of mean arterial pressure (MAP), right atrial pressure (RAP), pulmonary artery pressures (PAP), mean pulmonary artery pressure (MPAP), pulmonary artery wedge pressure (PAWP), and heart rate (HR) obtained prior to and after the completion of TD CO measurements.

Design

A repeated measures design was used to explore the relationship of hemodynamic pressure measurements, TD CO, and derived hemodynamic parameters. Each subject had two sets of serial measurements performed and then repeated in fours hours (Figure 1). Each set of serial measuremtns consisted of three hemodynamic pressure measurements pre TD CO measurements and three hemodynamic pressure measurements post TD CO measurements. The TD CO measurements were performed 30 minutes apart between sets and consisted of a minimum of three room temperature injectate (RTI) dextrose 5% in water (D5W) 10 millimetres (ml.) injections. Derived hemodynamic parameters were calculated from each pre and post hemodynamic pressure measurements on all subjects.

Hemodynamic pressures consisted of HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), MAP, RAP, PAWP, MPAP, pulmonary artery systolic (PAS), and pulmonary artery diastolic (PAD). Derived hemodynamic parameters consisted of systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), SV, SVI, left ventricular stroke work index (LVSWI), and right ventricular stroke work index (RVSWI).

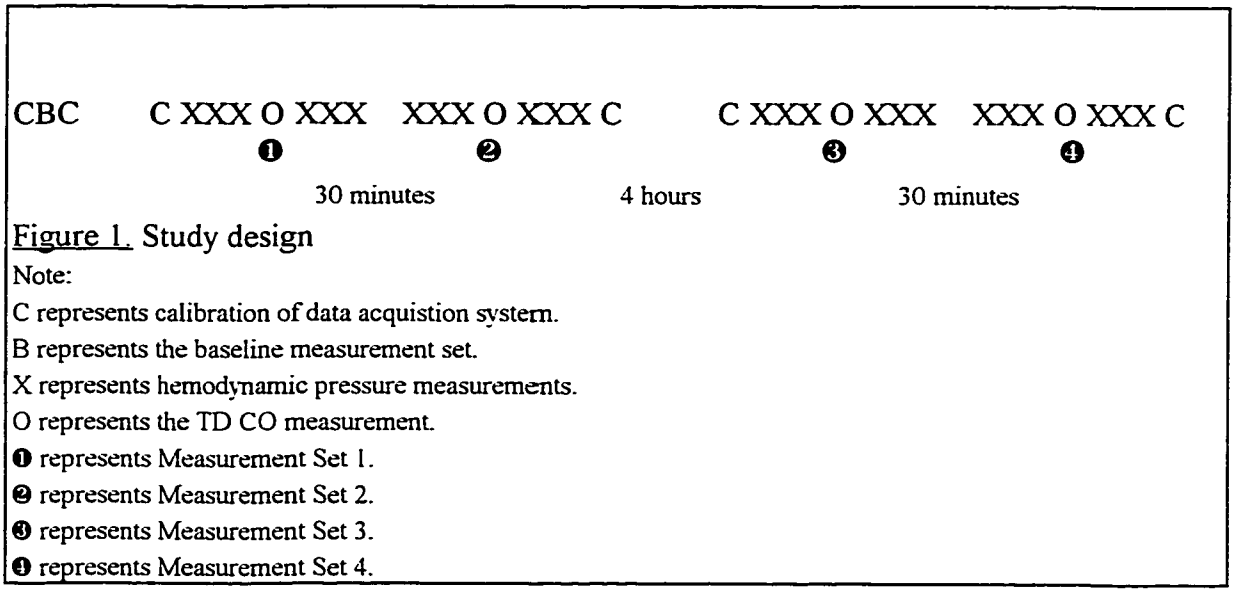

Figure 1. Study design

Note:
C represents calibration of data acquistion system.
B represents the baseline measurement set.
X represents hemodynamic pressure measurements.
O represents the TD CO measurement.
❶ represents Measurement Set 1.
❷ represents Measurement Set 2.
❸ represents Measurement Set 3.
❹ represents Measurement Set 4.

Setting

The study was conducted in the cardiothoracic surgical critical care unit (CCU) of the University of Alberta Hospital, where there are approximately 1200 cardiac surgeries per year. There were approximately 20 to 25 adult cardiac surgeries per week.

Sample

A convenience sample of 30 cardiac surgical subjects was used to yield 120 TD CO measurements and 720 hemodynamic pressure measurements, and 720 derived

hemodynamic parameters. This would provide a power of .80, with an alpha of .05 and an effect size of 3 millimetres of mercury (mm Hg) between the pre and post hemodynamic pressure measurements. The effect size was chosen based on the accepted 4 mm Hg clinical variation for PAWP, PAD, and RAP, and 5 mm Hg for PAS (Nemens & Woods, 1982). An effect size of less than 3 mm Hg was considered not clinically relevant. Cardiac surgery included aortic or mitral valvular repair or replacement and coronary artery bypass grafts, with either an internal mammary or radial artery or saphenous vein graft. Inclusion criteria included: (a) men and women older than 18 years who had elective cardiac surgery; (b) wrote, read, and understood English; (c) pulmonary artery catheter (PAC) placed within an internal jugular or subclavian vein; (d) PAC position confirmed by x-ray and waveform analysis; (e) minimum of 1 cubic centimeters (cc) of air required to obtain a PAWP; (f) presence of RAP waveform from the proximal port; (g) radial arterial line; (h) required continuous monitoring of HR, MAP, RAP, and PAP; (i) orally intubated with either synchronous intermittent mechanical ventilation (SIMV), assist control (AC), or pressure suppport (PS), or non-ventilated with spontaneous respirations; (j) hemodynamic pressures fluctuating less than 10% and requiring no manipulation of ventilator settings, patient position, inotropes, vasodilators, vasopressors, or antiarrthymics within 10 minutes prior to initiation of the study protocol; (k) PAWP less than 20 mm Hg; (l) HR less than 120 to enable blood to circulate from the lungs to the left atrum; (m) absence of cardiac tamponade; and (n) within 8 hours post cardiac surgery.

Exclusion criteria included: (a) known or suspected intracardiac shunts, valvular stenosis or insufficiency, thoracic tumour, atrial myxoma, mediastinal fibrosis, or adult

respiratory distress syndrome; (b) inability to tolerate fluid requirements or D5W for TD CO; (c) presence of open sternum or ventricular assist devices; (d) requirement for mechanical or medication administration for dialysis; (e) chest tube drainage greater than 2 ml./kilogram/hour within four hours prior to the study; (f) ambient room temperature greater than 25 degrees celcius ([c]); (g) chronic pulmonary disease or hypertension; (h) postoperative shivering which increases the carbon dioxide level; or hypoxia, as defined as arterial saturation less than 90%, as there is pulmonary vasoconstriction; (i) postive end expirartory pressure (PEEP) greater than 10 centimetres of water (cmH_2O); (j) peak inspiratory airway pressure greater than 35 cmH_2O; (k) core body temperature less than 34.5 degrees [c]; (l) cardiac arrest within four hours prior to the study; (m) uncontrolled dysrhythmias; (n) received rapid volume administration of greater than 100 ml./hour; (o) CO less than 2 or greater than 15 litres/minute (l/min.), due to increased risk of measurement error; (p) inability to obtain a PAWP or if the PAWP was greater than the PAD; (q) continuous infusions via the proximal port of the PAC, as TD CO could not be performed; and (q) myocardial ischemia or infarction within the last 14 days.

Definition of Terms

Supine: trunk of the body maintained on the horizontal axis, with the head of the bed (HOB) elevated 15 degrees. The elevation was determined with a carpenter's level and protractor, which measured degrees of an angle. Prior to implementation of the study and usage with each subject the protractor and level were placed on a flat horizontal stationary object to ensure accuracy. Each patient position was checked by a staff nurse of the unit.

Heart Rate: number of heart beats per minute, continuously updated by the Hewlett-

Packard (HP) electrocardiograph (ECG) module (Model #M1002A) and displayed on the HP bedside monitor (Model #M1094B). The bedside monitors had standard preventive maintenance once a year by the Clinical Engineering Department (University of Alberta Hospital Site). The accuracy of the ECG module was ±1% within the range of 15 to 300 beats per minute with a 2 second nominal display rate update (Hewlett Packard, 1996b). The HR that was recorded was based on a mean of 3 displayed ranges within ±10%.

Cardiac Output: volume of blood ejected by the ventricles, with a normal value of 4 to 8 l/min.. The CO measurements were performed using the thermodilution method with all of measurements obtained by the researcher.

Cardiac Index: volume of blood ejected by the ventricles based on body surface area (BSA) and the CO, expresses as litres/minute/meter squared (l/min./m^2). The cardiac index (CI) was determined manually with a calculator and electronically with the HP CO module and the hemodynamic calculation software. All CI values were manually and electronically calculated. The CI formula for manual and electronic determination was the same, therefore the calculations were identical. The electronic determination were utilized in the data analysis.

Thermodilution Cardiac Output: injection of a known volume and temperature of solution into the right atrium via a PAC. All CO measurements were performed with the patient supine and during end-expiration. An average of 3 to 7 measurements within 10% of each other constituted the mean CO. The mean CO was determined manually with a calculator and electronically with the HP CO module. All mean CO measurements were manually and electronically calculated. The formula for manual and electronic determination was the

same, therefore the calculations were identical. The electronic determinations were utilized in the data analysis. During each TD CO injection, a curve was obtained to ensure a rapid, smooth upstroke and slow, smooth downstroke of the CO curve (Kiess-Daily & Schroeder, 1994; Levett & Replogle, 1979). Any abnormal curves were eliminated and replaced with another injection. The initial set of TD CO measurement curves were checked with a staff nurse of the unit.

Dye dilution and TD CO have correlations of r=.88 to .98 (Conway & Lund-Johansen, 1990; Kadota, 1985; Runciman, Ilsley, & Roberts, 1981), while TD CO and Fick CO have correlations of r=.63 to .98. Also 10 ml. of room temperature and iced injectate injectate have a correlation of r=.90 to .97 (Kiess-Daily & Mersch, 1987; Davies et al., 1986; Kadota, 1985; Lynch & Kaemmerer, 1990; Sommers et al., 1993).

The same HP CO module (HP model #M1012A) and injectate probe were used for all subjects. All of the equipment had preventive maintenance performed at the initiation of the study by the Clinical Engineering Department (University of Alberta Hospital Site). The CO module calculated the CO and CI, based on the area under the resulting time-temperature curve. The accuracy of the CO module was ±3% within a range of .1 to 20 l/min. The blood temperature range was 17 to 43 degrees [c] with an accuracy of ±.1 degree [c]. The injectate temperature range was -1 to 27 degrees [c] with an accuracy of ±.1 degrees [c] (Hewlett Packard, 1996b).

Room Temperature Injectate: 10 ml. of D5W stored at a room temperature of 19-25 degrees [c]. The temperature was measured using the HP injectate probe placed inside a 250 ml. D5W intravenous solution located directly beside the injectate solution. If the

injectate temperature probe is not beside the injectate intravenous solution, there may be an 1 to 2 degree temperature difference between the measured and injectate solution, which will invalidate the Stewart-Hamilton equation (Vennix et al., 1984). The injectate solution was D5W, as the Stewart-Hamilton equation is based on the specific gravity and heat of D5W (Bowdle et al., 1991; Kadota, 1985; Kiess-Daily & Schroeder, 1994; Levett & Replogle, 1979).

Hemodynamic Pressures: measurement of the dynamic moving forces of the cardiovascular system. The hemodynamic pressures consist of SBP, DBP, MAP, RAP, PAS, PAD, MPAP, and PAWP. The researcher obtained all of the hemodynamic pressures. For each subject a staff nurse of the unit, checked every tenth set of digital hemodynamic pressure measurements to determine reliability of measurements. Also, the data acquisition system (DAS) values were randomly checked by a thesis committee member or the clinical nurse educator for the Cardiac Care Intensive Care Unit to determine reliability of measurements. The MAP, DBP, and SBP were obtained via the digital method. The PAS, PAD, MPAP, PAWP, and RAP were obtained via the digital method and with a DAS prior to and after each set of 12 hemodynamaic pressure measurements (Figure 1) DAS samples of data for determination of baseline were obtained. The DAS baseline data were utilizied to adjust the obtained hemodynamic pressure measurements to aquire calibrated data. The calibrated data were used for the determination of the DAS derived hemodynamic parameters. The pre and post DAS baseline data were averaged to obtain the offset numerical value. When the difference between the pre and post baseline data was greater than 2 mm Hg the hemodynamic

measurement pressures were repeated, which were then utilized in the data analysis. The DAS incorporates Labtech Notebook (Version 6.3.0, 1991). The DAS has a resolution of ± 0.25 mm Hg with a sampling rate that is configurable, which was set at 20 hertz (HZ) or 300 data points over 15 seconds (personal communication, A. Sackiw, Clinical Engineering, University of Alberta Hospital Site, 1997).

The bedside monitors used were the HP Model #M1094B. The same pressure module (HP Model #M1006B) was employed for all subjects. The specifications for the bedside and pressure modules were automatic zero with a range of ±200 mm Hg, accuracy of ±l mm Hg, zero drift of less than .10 mm Hg/degree of [c] and calibration drift of less than .05%/degree of [c] (Hewlett Packard, 1996b). The Cobe disposable transducers were zero balanced and the monitor calibrated at the beginning of each study protocol. Zero-balance was indicated with a square sign and a value of zero displayed. Calibration of the monitor was present when a value of 200 was displayed.

The disposable transducer was the Cobe CDXpress with the specifications of: (a) pressure range -50 to +300 mm Hg; (b) dynamic response of 100 HZ; (c) zero temperature coefficient ±.4 mm Hg/degree of [c], which was the drift from zero baseline with a change in temperature; (d) eight hour zero drift of less than 1 mm Hg; and (e) the combined sensitivity, linearity, and hysteresis did not exceed ±2% or 1 mm Hg, whichever is greater (Cobe, 1990).

The dynamic characteristics of resonant frequency and damping coefficient were not included even though there is support within the literature. The rationale was that the determination of dynamic characteristics had been required when using old technology

pressure transducers and graphic recorders. Current technological design eliminates the need for on-going calibrations and testing of the dynamic characteristics. These devices have published specifications and dynamic characteristics, which are guaranteed by the manufacturer (personal communication, A. Sackiw, Clinical Engineering, University of Alberta Hospital, 1997). Calibration runs were performed to verify these characteristics and found to be acceptable. The Code CDXpress has a dynamic response of 100 HZ and the HP pressure model #M1006B filter frequencies above 40 HZ (Cobe, 1990).

Mean Arterial Pressure: pressure of the arteries averaged over the entire cardiac cycle. The MAP is dependent on the blood volume and the compliance of the vessels. The calculation for MAP was one SBP + two DBP / 3 with a normal value of 70 to 90 mm Hg. The HP module incorporated beat averaging to determine the MAP (HP Medical Response Center, personal communication, December 2, 1994).

Preload: end-diastolic volume and pressure of the ventricles that influences the myocardial fiber length and force of contraction. The preload values obtained were the RAP, PAD, and PAWP. The type of pulmonary artery catheter (Baxter Model #7F 13157) remained consistent during the study.

Right Atrial Pressure: reflects the preload of the right ventricle. The RAP was obtained via the proximal port of a PAC and was displayed on the HP monitor. The normal RAP is -1 to 7 mm Hg.

Pulmonary Artery Pressure: reflects the pressure and volume of the right and left ventricle, measured via the distal port of a PAC, which was displayed on the HP bedside monitor and consisted of a systolic, diastolic, and mean pressure. The normal values

respectively are 15 to 30 mm Hg, 8 to 15 mm Hg, and 10 to 12 mm Hg. The formula for MPAP was 1 systolic pressure + 2 diastolic pressure / 3. The HP module incorporated beat averaging to determine the MPAP (HP Medical Response Center, personal communication, December 2, 1994).

Pulmonary Artery Wedge Pressure: reflects the preload of the left ventricle when the balloon of the PAC is inflated. The PAWP was displayed on the HP monitor. The normal PAWP is 5 to 12 mm Hg.

Hemodynamic Parameters: calculation of the dynamic moving forces of the cardiovascular system, based on the hemodynamic pressures. The derived hemodynamic parameters were SVRI, PVRI, LVSWI, RVSWI, and SVI. The researcher obtained all of the derived hemodynamic parameters. The derived hemodynamic parameters were calculated electronically utilizing the hemodynamic calculation software. The electronic calculations have a hard copy print-out. Every tenth set of hemodynamic parameters were calculated electronically and manually with a calculator. However, as the formulas are identical only the electronic determinations were utilized in data analysis.

Afterload: resistance to flow from the ventricles and is influenced by radius, length, and viscosity. The calculated afterload values of PVRI and SVRI are measured in absolute resistance units indexed (dynes/sec/cm^{-5}/m^2).

Systemic Vascular Resistance Index: resistance the left ventricle has to overcome to eject the LVEDV into the aorta. The calculation for SVRI was systemic vascular resistance x BSA with a normal value of 1970 to 2390. Systemic vascular resistance was calculated as (MAP - RAP) x 79.96 / CO.

Pulmonary Vascular Resistance Index: resistance the right ventricle has to overcome to eject the RVEDV into the pulmonary artery. The calculation for PVRI was pulmonary vascular resistance x BSA with a normal value of 225 to 315. Pulmonary vascular resistance was calculated as (MPAP - PAWP) x 79.96 / CO.

Contractility: inherent capability of the myocardium to increase or decrease the myocardial fiber length independent of preload and afterload. Contractility was indirectly determined as SVI, LVSWI, and RVSWI and measured as grams (gr)-m/m^2/beat.

Stroke Volume: amount of blood ejected with each heart beat. The calculation was CO/HR x 1000 with a normal value of 60 to 120 ml./beat. The SV was determined electronically and manually. Each set of TD CO measurements was utilized to manually calculate the SV. The SV formula for manual and electronic determinations was the same, therefore the calculations were identical.

Stroke Volume Index: amount of blood ejected with each heart beat adjusted for BSA. The formula for SVI was SV / BSA. The normal SVI is 30 to 60 ml./beat/m^2. The SVI was determined electronically and manually. Each set of TD CO measurements was utilized to manually calculate the SVI. The SVI formula for manual and electronic determinations were the same, therefore the calculations were identical.

Left Ventricular Stroke Work Index: amount of work performed by the left ventricle adjusted for BSA. The calculation for LVSWI was SVI x (MAP - PAWP) x .0136 with a normal value of 40 to 75 gr-m/m^2/beat.

Right Ventricular Stroke Work Index: amount of work performed by the right ventricle adjusted for BSA. The calculation for RVSWI was SVI x (MPAP - RAP) x .0136 with a

normal value of 4 to 8 gr-m/m^2/beat.

Data Collection Procedure

Demographic data, which may have influenced measurements, were collected for each subject and included: (a) age; (b) cardiac surgical procedure; (c) gender; (d) height; (e) weight the day prior to surgery; (f) BSA; (g) relevant medical history including myocardial infarction and ischemia, pericardial disease, and smoking; (h) ventilator settings including fraction of inspired oxygen, PEEP, pressure support, peak inspriatoy airway pressure, exhaled tidal volume, and respiratory rate; (i) presence of supplementary oxygen; (j) arterial blood gases; (k) electrocardiograph rate and rhythm; (l) size and site of PAC and arterial vascular catheter; (m) cross-clamp and cardiopulmonary bypass time and number of minutes since surgery; (n) complications during and post surgery; (o) most recent hematocrit, hemoglobin, potassium, and magnesium; (p) core body temperatures upon completion of surgery, upon admission to the CCU, and at baseline and all CO measurements; (q) dosage of inotropes, vasodilators, vasopressors, antiarrthymics, sedation and analgesia; and (r) fluid balance including type and volume of fluids, chest tube loss, and urine output per hour. All data were recorded on the data collection record (Appendix A).

The study protocol was as follows:

Baseline Measures

1. Potential subjects were approached based on the cardiac surgical operating room slate the day prior to surgery. Informed consent was obtained after an explanation of the study protocol.
2. Upon the day of surgery the charge nurse and bedside nurse within the CCU were

informed that the patient had agreed to be a participant in the study. This same information was recorded on the CCU assignment record.

3. After the subject returned from the operating room the researcher determined if all of the inclusion and exclusion criteria for continuation in the study were met. The respiratory management for the patient followed the standard protocol of the CCU. Pain management followed the protocol of the unit, and just prior to initiation of the data collection procedure, analgesia was provided and recorded. When sedation or analgesia was required during the data collection period, the type, time, route, and dosage of the agent was recorded. During data collection any required alteration in the ventilator settings, dosage of medications, patient position, or suctioning, data collection was interrupted until a steady state was reassessed, as the actions may have affected the hemodynamic pressure measurements and TD CO values. The form of treatment was recorded. When aggressive treatment was required the subject was removed from the study. The data collection period was commenced within 2 to 4 hours after surgery. Complete data collection occured within 12 hours of surgery as the patient required frequent TD CO measurements.

4. All hemodynamic pressures were analyzed visually to ensure normal waveforms, as inaccurate patterns may affect the hemodynamic pressures and derived hemodynamic parameters. Troubleshooting procedures were implemented to eliminate abnormal waveforms (Hewlett Packard, 1982; Kern, 1993; Kiess-Daily & Schroeder, 1994). The study protocol did not commence until all hemodynamic pressure waveforms were normal.

5. Next, the subject was placed in a supine position with the HOB elevated 15 degrees. The elevation was determined with a carpenter's level and a protractor. Only one pillow

was placed beneath the subject's head. A staff nurse of the unit checked the patient position.

6. The air-fluid interface of the transducers were levelled to the phlebostatic level, which is the intersection of the fourth intercostal space that transects with half the distance from the dorsal surface of the thorax and the xiphoid process. This distance was determined with thorax calipers and then marked with a black felt pen on the subject's thorax cage. The air-fluid interface of the transducers were levelled, using a carpenter's level, with the black felt mark on the thorax cage. An experienced staff nurse of the unit checked the transducers positions. The air-fluid interface of the transducers were then zero-balanced and the monitor calibrated (Cobe, 1990; Hewlett Packard, 1982). When zero-balancing or calibration were not present the appropriate troubleshooting procedures were commenced (Hewlett Packard, 1982; Kern, 1993; Kiess-Daily & Schroeder, 1994). The patient was removed from the study if zero-balancing and calibration was not possible.

7. The injectate temperature probe required for the TD CO measurements was placed inside a 250 ml. solution of D5W, located directly beside the injectate solution. The injectate and core body temperature were obtained and recorded. The correct CO computer constant was determined and entered into the CO module and recorded (Hewlett Packard, 1996b). A staff nurse of the unit checked the computer constant. There must be a minimum of 10 to 12 degrees [c] difference between the injectate and core body temperature to ensure accurate TD CO measurements (Gardner & Woods, 1987; Halfman-Franey, 1988; Kiess-Daily & Schroeder, 1994; Levett & Replogle, 1979). When there was not a minimum of 10 degrees [c] difference, a new solution of D5W was

obtained. When there still was not a minimum of 10 degrees [c] difference, a new injectate probe was obtained. When there was still a problem with the temperature difference, the subject was removed from the study.

8. The correct height and weight were entered into the HP bedside monitor (Hewlett Packard, 1996a). This was checked with a staff nurse of the unit. The TD CO 10 ml. syringe was attached in a linear line to the proximal port stopcock. This was to ensure that no right angle existed between the stopcock and the syringe, which may decrease the speed and ease of injection of the D5W solution.

9. Baseline hemodynamic pressures were obtained one hour prior to commencement of data collection. A sample of DAS baseline data was obtained. Digital and DAS recordings were obtained for the RAP, PAS, PAD, MPAP, and PAWP, while the SBP, DBP, and MAP were determined only by the digital method. The digital value was the lowest observed measurement. Each digital value was recorded simaltaneously with the DAS recording for each of the required pressure measurements. The hemodynamic pressures were recorded in the sequence of HR, SBP, DBP, MAP, PAS, PAD, MPAP, PAWP, and RAP. The PAS, PAD, PAWP, and RAP were determined and recorded at end-expiration, as the pleural pressure was static. For a mechanical intiated breath end-expriation was determined as the lowest numerical value prior to an elevation in the hemodynamic pressure measurement. During spontaneous ventilation end-expriation was determined as the highest numerical value prior to a decrease in the hemodynamic pressure measurement (Kern, 1993; Kiess-Daily & Schroeder, 1994). The PAWP, PAS, PAD, and RAP values were determined as the mean over a minimum of one respiratory cycle. The MPAP was

obtained via the digital method and by mathematical calculation from the mean DAS values of the PAS and PAD.

The PAWP was obtained with the balloon inflated for less than 15 seconds, as timed by a stopwatch and the DAS, with a minimum of 1 cc of air. The digital PAWP was obtained by the stop cursor method of the bedside monitor. If the PAWP was greater than the PAD, the PAWP was repeated, and if the PAWP remained elevated, the physician was notified to ensure correct placement of the PAC. When necessary the subject was removed from the study at this point, as an obstruction might have existed between the pulmonary artery and the left ventricle.

10. Upon completion of the acquisition of the hemodynamic pressure measurements DAS baseline data was obtained. The pre and post DAS baseline data was averaged to obtain the offset value. When the difference between the pre and post DAS baseline data was greater than 2 mmHG the baseline measurement of hemodynamic pressures was repeated.

Measurement Set 1

1. Arterial blood gases were obtained 5 minutes prior to obtaining the first set of measurements. The time was 55 minutes after obtaining the baseline hemodynamic pressures. When any medications were infusing via the proximal port of the PAC, the data collection period did not commence until the completion of the infusion of the medications.

2. The subject was placed supine with the HOB elevated 15 degrees per standard protocol for the unit. The elevation was determined with a carpenter's level and protractor. Only one pillow was placed beneath the subjects head. A staff nurse of the unit

checked the patient position.

3. It was ensured that the transducers were levelled to the phlebostatic level following the same protocol as in the baseline measurements. An experienced staff nurse of the unit checked the transducer's positions.

4. A sample of DAS baseline data was obtained. Then three serial hemodynamic pressures were obtained within two to five minutes of each other, timed with a stopwatch and the exact time was recorded. The first series of hemodynamic pressures had to within $\pm$10% of the baseline hemodynamic pressures. When the hemodynamic pressures obtained were greater than the $\pm$10% variance of the baseline measurements, the data collection was delayed until the subject was in a steady state. Digital and DAS recordings were obtained for the RAP, PAS, PAD, MPAP, and PAWP, while the SBP, DBP, and MAP were determined only by the digital method. Each digital value was recorded simaltaneously with the DAS recording for each of the required pressure measurements. Each series of hemodynamic pressures was obtained, as conducted for the baseline measurement set.

5. Upon completion of obtaining the third set of hemodynamic pressure measurements, the TD CO measurements were performed utilizing the CO module and recorded (Hewlett Packard, 1996b; Kern, 1993; Kiess-Daily & Schroeder, 1994). The injectate and core body temperature and computer constant were determined, following the same protocol as in the baseline measurement. When the computer constant had to be altered from the initial set-up, the new computer constant was be entered into the CO module (Hewlett Packard, 1996b). The injectate and core body temperature, and computer constant were recorded. The core body temperature was utilized in data analysis. The TD CO 10 ml.

syringe was checked to ensure a linear relationship with the proximal port. When the syringe was at right angles to the proximal port, the correct linear position was obtained.

The manual injection technique was employed for the TD CO measurements. The injections occured at end-expiration and in less than 4 seconds, as timed by a stopwatch. A CO curve was obtained for each measurement with the initial curve checked by a staff nurse of the unit. Succeeding injections occured when the CO module indicated ready. The syringe was filled with 10 ml. of D5W prior to each injection. The barrel of the syringe was not handled, because if the injectate solution temperature varies during the procedure the Stewart-Hamilton equation is invalidated. The total volume required for TD CO measurements was recorded.

During the determination of CO, no medications were infused via the proximal port of the PAC, nor were any medications infusing through the introducer discontinued, as per protocol of the unit. This was to prevent the risk of inadvertent bolus of a medication or an alteration in the serum drug level, and the resulting change in the hemodynamic pressures and CO measurements. During the TD CO procedure if any medications had to be infused via the proximal port of the PAC, the data collection period was restarted.

Any individual CO measurements were discarded and repeated injections occured under the following conditions: (a) the first TD varied by greater than 10% from the succeeding injections, (b) any single measure was greater than 10% from the other injections, (c) the upstroke of the CO curve was uneven, (d) the downslope of the CO curve was not smooth, (e) the CO module indicated an unstable curve, and (f) oscillations

of the baseline, as represents respiratory influence and hemodynamic instability. The injections were repeated to a maximum of 7 to prevent fluid overload. The CI value was determined based on the mean CO for each TD CO measurements. The mean CO from each set of TD CO measurements was employed to determine manual and electronic calculations of the CI.

6. Upon completion of the TD CO measurements, the next three serial hemodynamic pressure measurements commenced, following the same protocol as in the baseline measurement set.

Measurement Set 2

1. When 30 minutes had lapsed since the last hemodynamic pressure measurement, as timed by a stopwatch, the next set of hemodynamic pressures and TD CO measurements commenced. It was ensured that the subject was supine with the HOB elevated 15 degrees, following the same protocol as in the baseline measurements. An experienced staff nurse of the unit checked the patient position.

2. It was ensured that the transducers were levelled to the phlebostatic level, following the same protocol as in the baseline and first measurement groups. A staff nurse of the unit checked the transducers positions.

3. Three serial hemodynamic pressure measurements were obtained within two to five minutes of each other timed with a stopwatch and the exact time was recorded. Digital and DAS recordings were obtained for the RAP, PAS, PAD, MPAP, and PAWP, while the SBP, DBP, and MAP were determined only by the digital method. Each digital value was recorded simaltaneously with the DAS recording for each of the required pressure

measurements.

4. Upon completion of obtaining the third series of hemodynamic pressure measurements the TD CO measurements were performed, following the same protocol, as in Measurement Set 1.

5. Upon completion of the TD CO measurements, the next three serial hemodynamic pressure measurements were commenced. Three serial hemodynamic pressure measurements were obtained two to five minutes apart timed with a stopwatch and the exact time was recorded. Digital and DAS recordings were obtained for the RAP, PAS, PAD, MPAP, and PAWP, while the SBP, DBP, and MAP were determined only by the digital method. Each digital value was recorded simaltaneously with the DAS recording for each of the required pressure measurements. At the completion of the acquistion of the hemodynamaic pressure measurements, DAS baseline data was obtained. The pre and post DAS baseline data was averaged to obtain the offset value. When the difference between the pre and post DAS data was greater than 2 mm Hg Measurement Sets 1 and 2 hemodynamic pressures were repeated.

Measurement Sets 3 and 4

Four hours after the second set of hemodynamic pressures were obtained, Measurement Set 3 commenced. Arterial blood gases were repeated prior to obtaining Measurement Set 3 to ensure cardiorespiratory stability. Thereafter, a fourth measurement set was obtained from which derived hemodynamic parameters were calculated.

Derived Hemodynamic Parameters

1. Upon completion of the TD CO measurements and the hemodynamic pressure

measurements, the derived hemodynamic parameters were calculated. The derived hemodynamic parameters were determined based on each of the sets of hemodynamic pressures. Therefore, there were 6 sets of derived hemodynamic parameters per measurement. Additionally, the mean of the 3 hemodynamic pressure measurements pre and post TD CO measurements, which included HR were used to determine derived hemodynamic parameters.

2. The derived hemodynamic parameters were calculated every tenth series manually and coincided with the hemodynamic pressures that were obtained with the assistance of the staff nurse of the unit. For the remainder of the derived hemodynamic parameters, calculations were provided by the hemodynamic calculations software. The hemodynamic calculations software was utilized for the electronic determinations; however, the values were not automatically retrieved from the HP bedside monitor. The hemodynamic calculations software incorporates all the appropriate formula variables; however it was difficult to discern the timing of the required formula variables in relation to the TD CO measurements and the study protocol. The derived hemodynamic parameters incorporated the digital and DAS recordings of the PAWP, RAP, PAS, MPAP, and PAD, and the digital values for the SBP, DBP, and MAP. The baseline DAS sample data were used to adjust the actual DAS hemodynamic pressure measurements to acquire calibrated data. All of the digital and DAS calibrated hemodynamic pressure measurements were entered manually into the hemodynamic calculations software, and then the electronic calculations occured.

3. Systemic vascular resistance index was determined based on the digital value of MAP

and the DAS and digital measurements of RAP. Therefore, there were two RAP measurements and a singular MAP value. There were two SVRI determinations electronically calculated based on the digital values of the MAP and RAP, and the DAS value of RAP and digital measurement of MAP. Also, every tenth hemodynamic pressure measurement had two SVRI determinations manually calculated based on the DAS value of RAP and the digital measurement of MAP, and the digital values of MAP and RAP.

4. Pulmonary vascular resistance index was determined based on the digital and DAS measurements of the MPAP and PAWP. Therefore, there were two PAWP and MPAP values. There were two PVRI determinations electronically calculated based on the digital values of MPAP and PAWP, and the DAS values of PAWP and MPAP. Also, every tenth hemodynamic pressure measurement had two PVRI determinations manually calculated based on the DAS and digital values of the PAWP and MPAP.

5. Stroke volume was calculated based on the mean CO. The SV was determined electronically. Also, every tenth hemodynamic pressure measurement coincided with the manual determination of SV and CO.

6. Stroke volume index was calculated based on the SV value. The SVI was determined electronically. The manual calculation of SVI coincided with the manual determination of SV.

7. Left ventricular stroke work index was determined based on the digital value of the MAP, the DAS and digital measurements of PAWP, and the SVI. Therefore, there were two PAWP measurements, and singular MAP and SVI values. There were two LVSWI determinations electronically calculated based on the DAS value of PAWP and digital

measurement of MAP, and the digital measurements of PAWP and MAP. Also, every tenth hemodynamic pressure measurement had two LVSWI determinations manually calculated based on the DAS value of PAWP and the digital measurement of MAP, and the digital values of PAWP and MAP.

8. Right ventricular stroke work index was determined based on the digital values of MPAP and RAP, the DAS measurements of RAP and MPAP, and the SVI. Therefore, there were two MPAP and RAP measurements and a singular SVI value. There were two RVSWI determinations electronically calculated based on the DAS values of MPAP and RAP, and the digital measurements of MPAP and RAP. Also, every tenth hemodynamic pressure measurement had two RVSWI determinations manually calculated based on the DAS values of MPAP and RAP, and the digital values of MPAP and RAP.

Data Analysis

Demographic data were described by employing frequency distributions and measures of central tendency and variability. Frequency distributions consisted of grouped frequency distributions, percentiles, and frequency and percentage polygons. Measures of central tendency consisted of median and mean. Measures of variability consisted of range, variance, and standard deviation.

Frequency distributions and the measures of central tendency and variability were employed for a description of the hemodynamic pressures, derived hemodynamic parameters, TD CO and CI measurements, and the volume of TD injectate required. Mean and median were the measures of central tendency conducted. Range, variance, and standard deviation were the measures of variability that were employed. The measures of

central tendency and variability were performed for each subject and the entire sample examining: (a) each hemodynamic pressure and derived hemodynamic parameter within every set of measures before and after the TD CO measurement, (b) TD CO and CI measurements, and (c) the volume of TD CO injectate required.

Regression analysis was conducted to examine the correlation between the digital and DAS recordings of the hemodynamic pressures.To test the hypothesis that hemodynamic pressures obtained prior to TD CO measurements are lower than the hemodynamic pressures obtained after the TD CO measurements, repeated measures analysis of variance (ANOVA) was conducted. Repeated measures analysis of variance was also conducted to test the hypothesis that derived hemodynamic parameters calculated using the hemodynamic pressures obtained prior to TD CO measurement was lower than compared to the derived hemodynamic parameters calculated using the hemodynamic pressures obtained after the TD CO measurements. Analysis of covariance (ANCOVA) was then conducted to control for the variables of: (a) level of PEEP and pressure support; (b) airway pressure; (c) core body temperature; (d) aortic cross clamp and cardiopulmonary bypass time; (e) fluid balance; (f) hypoxia; (g) base excess level; (h) dosage of inotropes, vasodilators, vasopressors, sedatives, and analgesia; and (i) volume of TD CO injectate required. Two-tailed tests with a significance level set at $p = .05$ were employed.

Clinical significance was also considered, as a manipulation of any treatment on human subjects requires more than statistical analysis. A treatment may have statistical significance, yet may not affect clinical outcomes. Bryan-Brown and Dracup (1996) have

indicated that the endpoints or clinical data must support the appropriate clincial outcomes. The normal fluctuation of the PAWP, PAD, and RAP is less than 4 mm Hg. Therefore, the clinical significance was considered greater than 4 mm Hg of fluctuation. The normal fluctuation of PAS is less than 5 mm Hg. Therefore, a clinical significance was considered greater than 5 mm Hg of fluctuation (Nemens & Woods, 1982). The hemodynamic pressures, derived hemodynamic parameters, and TD CO and CI had a clinical significance level greater than 10% variance, as the accepted CO biological variability is less than 10% (Kadota, 1985; Lynch & Kaemmerer, 1990; Sasse, Chen, Berry, Sassoon, & Mahutte, 1994; Vincent, 1994).

Ethical Considerations

Prior to implementation of the study, approval was obtained from the Faculty of Nursing, and the Special Services and Research Committee, University of Alberta Hospital. Support for the study was sought from the patient care manager, cardiac surgeons, and intensivists. During the study protocol when a clinical significance of greater than 10% was evident with a particular relationship of hemodynamic pressures, parameters, and TD CO, the patient care manager, cardiac surgeons, intensivists, and thesis committee members were notified.

Potential subjects were approached by the researcher to determine eligibility. The researcher provided a verbal and written explanation of the purpose of the study to potential subjects, including the risks and benefits. It was stressed that all of the hemodynamic pressures, hemodynamic parameters, and TD CO were part of the normal care postoperatively. However, the difference was in the number of measurements and the

sequence of these values. The benefits discussed included the advantage of TD CO and PAWP and the importance of determining the relationship between the hemodynamic pressure measurements and parameters, and TD CO. An explanation was provided to indicate that the hemodynamic pressure measurements and the TD CO procedure were of no additional risk to the patient. The inclusion and exclusion criteria were discussed and information provided regarding the possible necessity for removal from the study. Explanation was provided regarding the patient's right to withdraw at any time without consequences to the nursing care provided. An informed signed consent (Appendix B) was obtained. Each subject received a code number, which was located on the data collection record. The corresponding names were kept in a seperate locked drawer maintaining confidentially during the study.

CHAPTER FOUR

Findings

A repeated measures within subject design was used to explore the relationship of hemodynamic pressure measurements, thermodilution (TD) cardiac output (CO), and derived hemodynamic parameters. There were four sets of serial measurements repeated for each subject. Three hemodynamic pressure measurements were performed prior to and three after the TD CO measurement, providing for a pre and post TD CO comparison of derived hemodynamic parameters on all subjects. Thirty minutes lapsed between Measurement Set 1 and Measurement Set 2; 4 hours between Measurement Set 2 and Measurement Set 3; 30 minutes between Measurement Set 3 and Measurement Set 4. A set of hemodynamic pressures consisted of heart rate (HR); systolic, diastolic, and mean arterial pressure; pulmonary artery systolic, diastolic and mean pressure; right atrial pressure (RAP); and pulmonary artery wedge pressure (PAWP). A set of derived hemodynamic parameters consisted of systemic and pulmonary vascular resistance index; stroke volume (SV); stroke volume index (SVI); and left and right ventricular stroke work index.

Characteristics of the Subjects

A total of 98 subjects were enrolled in the study with 50 subjects returning from surgery with a pulmonary artery catheter (PAC) in situ. Of these 50 subjects, 21 met the inclusion criteria for the study. The final sample ($n = 21$) consisted of 17 males and 4 females, ranging in age from 35 to 84 years ($M = 62.57$ years, $SD = 12.11$) (Table 1). The subjects ranged in height from 155 to 183 centimetres (cm) and 42.9 to 108 kilograms

(kg) in weight. Body surface area varied from 1.38 to 2.17 metres^2 . Serum hematocrit, hemoglobin, potassium, and magnesium levels upon admission to the critical care unit (CCU) are provided in Table 1. Serum hemoglobin was not obtained for 6 subjects. Based on the type of cardiac surgery, the cardiopulmonary bypass (CPB) time ranged from 46 to 230 minutes, with an aortic cross clamp (ACC) time of 24 to 173 minutes. One subject was not placed on cardiopulmonary bypass. The subjects were enrolled in the study between 54 and 240 minutes after admission to the CCU (M = 109.43 minutes, SD = 48.37) (Table 1).

Table 1

Subject Characteristics

Characteristic	Range	M	SD
Age (years)	35.00 - 84.00	62.57	12.11
Height (cm)	155.00 - 183.00	170.52	7.52
Weight (kg)	42.90 - 108.00	84.52	15.70
Body surface area (m^2)	1.38 - 2.17	1.96	0.20
Serum hematocrit (l/l)	0.22 - 0.38	0.30	0.05
Serum hemoglobin (gr/l)	77.00 - 141.00	106.27	20.54
Serum potassium (mmol/l)	3.50 - 5.00	4.34	0.41
Serum magnesium (mmol/l)	0.68 - 1.48	1.07	0.17
Cardiopulmonary bypass time (minutes)	46.00 - 230.00	119.80	45.24
Aortic cross clamp time (minutes)	24.00 - 173.00	76.50	38.12
Time since surgery (minutes)	54.00 - 240.00	109.43	48.37

A cardiac history was obtained upon admission to the CCU and cardiac risk factors were recorded. Twelve subjects (57%) had a history of hypertension, 7 subjects (33%) had a history of myocardial infarction or diabetes mellitus, 5 subjects (24%) had hyperlipidemia, and 3 subjects (15%) had coronary artery disease. Finally, 4 subjects reported a history of smoking.

Coronary artery bypass graft was the surgical procedure for 14 subjects (67 %).

The remaining 7 subjects, underwent valvular replacement or repair (Table 2). Only 2 subjects had a reported complication during surgery, which included shock requiring the insertion of an intra-aortic balloon catherter, and bleeding defined as greater than two millimeters (ml) per kg.

Table 2

Types of Surgical Procedures

Surgical Procedure	f	%
Coronary artery bypass graft	14	66.70
Aortic valve replacement	3	14.30
Aortic and Mitral valve replacement	1	4.80
Mitral valve repair	1	4.80
Mitral valve replacment	1	4.80
Bentall's Procedure	1	4.80

All subjects ($n = 21$) had a size 7 french PAC in situ (Baxter Model #7F 13157). Nineteen (90%) subjects had the PAC inserted via the right internal jugular vein and for the remaining 2 subjects via the left subclavian vein and the left internal jugular vein. A radial arterial line was in situ for all subjects; the right radial artery in 19 subjects and the left radial artery in 2 subjects. As reported by the anaesthetist, the arterial cannual was size 20 french in 13 (62 %) subjects, with no recorded cannula size for the remaining 8 subjects. Respiratory Status

Arterial Blood Gases. At Measurement Set 1, the oxygenation status as reflected by the partial pressure of oxygen (PaO_2), varied between 80 and 210 millimeteres of mercury (mm Hg) ($M = 124.71 \pm 33.82$ mm Hg). The partial pressure of carbon dioxide ($PaCO_2$) reflected normal ventilation status ($M = 38.0 \pm 4.71$ mm Hg), with a range from 28.0 to 48.0 mm Hg. However, at Measurement Set 3, mean PaO_2 and $PaCO_2$ had

decreased to 104.38 ± 28.53 mm Hg and 38.29 ± 5.19 mm Hg, respectively. At Measurement Set 1 there was no acid-base imbalance, with the hydrogen ion concentration ranging from 32.0 and 54.0 nanograms (nM) (M = 41.33 ± 5.83 nM). Base excess (BE) ranged from -7.0 to 3.0 milliequivilent per litre (meq/l) (M = -1.67 ± 2.71 meq/l) (Table 3).

Ventilation Parameters. At Measurement Set 1, all subjects (n =21) were intubated and received mechanical ventilation. Twenty subjects received the synchronized intermittent ventilation mode, while the remaining subject received pressure support (PS) ventilation. The ventilator rate ranged from 6 to 15 breaths per minute (M = 8.43 ± 2.28) with a patient rate of 6 to16 breaths per minute (M = 9.29 ± 2.55). All 21 subjects received positive end expiratory pressure (PEEP). The majority of subjects (n = 20) received 5 cmH_20 of PEEP, while one subject received 8 cmH_20 of PEEP. Twenty subjects also received PS, which varied between 3 and 12 cmH_20 (M = 3.38 ± 2.13 cmH_20). The airway pressure (AWP) recorded at the time of the ABG ranged from 12.7 to 38 cmH_20 (M = 31.28 ± 4.99), yet upon commencement of data collection the AWP in all subjects was less than 35 cmH_20.

At Measurement Set 3, there were 7 subjects (33%) who were extubated and received supplementary oxygen via nasal prongs. The 14 remaining mechanically ventilated subjects received synchronized intermittent mechanical ventilation (n = 13) and PS (n = 1). The majority of subjects (n = 13) received PEEP of 5 cmH_20 and one subject continued to receive 8 cmH_20 of PEEP. In comparison to Measurement Set 1 the amount of PS had decreased. Thirteen subjects (62%) received 3 cmH_20 of PS, while one subject

continued to receive 12 cmH_20 of PS. The AWP at Measurement Set 3 remained similar to Measurement Set 1 (Table 3).

Table 3

Ventilation Parameters and Arterial Blood Gases

	Measurement Set 1			Measurement Set 3		
Parameter	N	M	SD	N	M	SD
PaO_2 (mm HG)	21	124.71	33.82	21	104.38	28.53
$PaCO_2$(mm HG)	21	38.00	4.71	21	38.29	5.19
Hydrogen (nM)	21	41.33	5.83	21	41.57	5.85
Saturation (%)	21	98.19	1.03	21	97.24	1.34
Base Excess (meq/l)	21	-1.67	2.71	21	-2.0	3.33
FIO_2	21	.48	.19	14	.36	.09
PEEP (cmH_20)	21	5.14	.66	14	5.21	.80
PS (cmH_20)	21	3.38	2.13	14	3.64	2.41
Rate (ventilator)	20	8.43	2.38	13	8.31	2.81
Rate (patient)	21	9.29	2.55	21	13.33	.41
Exhaled TV (ml)	21	1062.19	221.69	14	990.14	245.99
Airway pressure (cmH_20)	21	31.28	4.99	13	31.33	6.52

Body Temperature

Core temperature was obtained via the PAC. At the end of surgery, core temperature was the lowest ($\underline{M} = 36.17 \pm .75^0$ C). Upon admission to the CCU, the temperature remained low ($\underline{M} = 35.51 \pm .60^0$ C) and rose from $37.16 + .49^0$ C at baseline to $37.54 \pm .69^0$ C at Measurement Set 4 (Table 4).

Table 4

Core Body Temperature Via the Pulmonary Artery Catheter

	Range	M	SD
End of surgery	34.00 - 37.50 ^{0} C	36.17	0.75
Admission to CCU	34.10 - 36.70 ^{0} C	35.51	0.60
Baseline	35.40 - 38.00 ^{0} C	36.61	0.66
Measurement Set 1	36.00 - 38.70 ^{0} C	37.16	0.69
Measurement Set 2	36.30 - 38.40 ^{0} C	37.27	0.62
Measurement Set 3	36.60 - 38.20 ^{0} C	37.56	0.49
Measurement Set 4	36.70 - 38.20 ^{0} C	37.54	0.50

Fluid Balance

Fluid balance at baseline was -217.76 $\pm$ 331.68 millilitres per hour (ml/hr). A positive fluid balance occured at Measurement Sets 2 and 3. At Measurement Set 4, there was a small negative fluid balance (Table 5).

Table 5

Fluid Balance

	Range	M	SD
Baseline (ml/hr)	-930 - 343	-217.76	331.68
Measurement Set 1 (ml/hr)	-375 - 563	-19.28	241.63
Measurement Set 2 (ml/hr)	-513 - 555	75.33	273.04
Measurement Set 3 (ml/hr)	-335 - 568	17.10	217.37
Measurement Set 4 (ml/hr)	-225 - 481	-2.86	134.34

Medications

Inotropes. At baseline, 15 subjects (71%) were on a continuous infusion of intropes (Table 6). Progressively between baseline and Measurement Set 4, the number of subjects who required inotropes decreased. At Measurement Set 4, 52% of the subjects (n = 11) required a continuous infusion of an inotropic agent. The agent utilized to the

greatest extent was Epinephrine at a dose less than (<) .049 micrograms per kg per minute (ug/kg/min). Epinephrine < .049 mg/kg/min was used in 48% of the subjects (n = 10) at baseline and 28 % of the subjects (n = 6) at Measurement Set 4. Epinephrine was never used at the higher dose range of greater than (>) .20 ug/kg/min.

The next most frequently used inotropic agent was Milrinone at the dose range of .33 to .67 ug/kg/min, with the highest frequency of use being at Measurement Sets 3 and 4. Milrinone > .68 ug//kg/min was utilized for 5% of the subjects (n = 1) at at baseline only. Inocor > 10.0 ug/kg/min was administered at Measurement Set 1. The only dosage of Dopamine used was the renal dose of < 2.49 ug/kg/min for one subject throughout the four measurement sets.

Table 6

Inotropes Dosage at Measurement Sets

	Baseline	Measurement Set 1	Measurement Set 2	Measurement Set 3	Measurement Set 4
Medication	f	f	f	f	f
Dopamine					
< 2.49 ug/kg/min	1	1	1	1	1
> 2.5 ug/kg/min	0	0	0	0	0
Epinephrine					
<.049 ug/kg/min	10	7	8	8	6
.05 - .19 ug/kg/min	2	4	2	1	2
> .20 ug/kg/min	0	0	0	0	0
Inocor					
0 - 9.99 ug/kg/min	0	0	0	0	0
> 10.0 ug/kg/min	1	1	0	0	0
Milrinone					
< .32 ug/kg/min	0	0	0	0	1
.33 - .67 ug/kg/min	0	1	2	2	1
>. 68 ug/kg/min	1	0	0	0	0

Vasoldilators, Vasopressors, and Venodilators. Venodilator-vasodilator combinations were utilized to the greatest extent for subjects at baseline (n = 11) (Table 7). The agent employed was Nitroglycerine at a dose range of 1.0 to 5.99 ug/kg/min. Progressively between baseline and Measurement Set 4, the number of subjects who required Nitroglycerine decreased. The second most utilized agent was Sodium Nitroprusside. At all four measurement sets, Sodium Nitroprusside was infused at a dose range of < .99 ug/kg/min in 13% of the subjects (n = 3). The dose range of 1.0 to 4.99 ug/kg/min was required by one subject at baseline and Measurement Set 1. Levophed was only utilized at the lower dose range of < .029 ug/kg/min for one subject continuously throughout all four measurement sets.

Table 7

Vasodilators, Vasopressor, and Venodilators Dosage at Meaurement Sets

	Baseline	Measurement Set 1	Measurement Set 2	Measurement Set 3	Measurement Set 4
Medication	f	f	f	f	f
Sodium Nitroprusside					
< .99 ug/kg/min	3	3	3	3	3
1.0 - 4.99 ug/kg/min	1	1	0	0	0
5.0 - 10.0 ug/kg/min	0	0	0	0	0
Nitroglycerine					
< .99 ug/kg/min	4	3	0	0	0
1.0 - 5.99 ug/kg/min	11	6	5	2	2
> 6.0 ug/kg/min	0	0	0	0	0
Levophed					
< .029 ug/kg/min	1	1	1	1	1
> .03 ug/kg/min	0	0	0	0	0

Analgesics, Sedatives, and Antiarrhythmics. The most frequently employed analgesic/sedative agent was a continuous infusion of Diprivan (Table 8). Fifty-three

percent of the subjects (n = 11) required one of three dose ranges of Diprivan at baseline measurement. Forty-three percent (n = 9) of the subjects received an infusion at 1.0 to 2.99 mg/kg/hr. The dose range of > 3.0 mg/kg/hr was utilized for 10% of the subjects (n = 2) at Measurement Sets 1 and 2. There was one subject who recieved a bolus of Valium at Measurement Set 1.

Morphine Sulphate was provided as a 4.0 to 5.99 mg intravenous bolus to 62% (n = 13) of the subjects at baseline. There were 3 subjects who recieved a bolus of Demerol instead of the standard analegesic Morphine Sulphate. The Demerol was required for shivering. Five subjects did not receive an analgesic or sedative at baseline, as the bedside nurse had done so within one hour of commencement of the study. Morphine Sulphate bolus of 4.0 to 5.99 mg was also given during the four measurement sets (Table 8).

The only antiarrhythmic agent utilized was a continuous infusion of Xylocaine. One subject required Xylocaine at 2.0 to 2.99 milligrams per minute at all four measurement sets.

Table 8

Analgesic, Sedative, and Antiarrhytmic Dosages at Measurement Sets

	Baseline	Measurement Set 1	Measurement Set 2	Measurement Set 3	Measurement Set 4
Medication	f	f	f	f	f
Demerol	3	2	0	0	0
Diprivan					
< .999 mg/kg/hr	1	1	1	0	0
1.0 - 2.99 mg/kg/hr	9	6	6	3	2
> 3.0 mg/kg/hr	1	2	2	1	1
Morphine Sulphate					
2.5 - 3.99 mg	0	0	0	1	0
4.0 - 5.99 mg	13	4	1	3	4
Xylocaine					
< 1.99 mg/min	0	0	0	0	0
2.0 -2.99 mg/min	1	1	1	1	1
3.0 - 4.0 mg/min	0	0	0	0	0

Miscellaneous Agents. Insulin, as a continuous infusion, was the other most frequently used medication (Table 9). Four subjects (19%) required an Insulin infusion at baseline and Measurement Sets 1 and 2. At Measurement Set 4 there was one subject who also required a bolus of sodium bicarbonate because of acidosis and another subject required an Amicar continuous infusion at all four measurement sets.

Table 9

Miscellanous Medications Dosage at Measurement Sets

	Baseline	Measurement Set 1	Measurement Set 2	Measurement Set 3	Measurement Set 4
Medication	f	f	f	f	f
Amicar	1	1	1	1	1
Sodium Bicarbonate	0	0	0	0	1
Calcium Chloride	1	0	0	0	1
Magnesium Sulphate	1	0	0	0	0
Insulin	4	4	4	5	6

Electrocardiographic Rhythm

The electrocardiographic (ECG) rhythm was determined at the commencement of each measurement set. Normal sinus rhythm (NSR) occurred with the greatest frequency (Table 10). Ten subjects (48%) exhibited NSR at baseline and Measurement Set 1, which then increased to 57% (n = 12) at Measurement Sets 3 and 4.

The second most common ECG rhythm was pacemaker related. Pacemaker rhythms consisted of atrial, ventricular, or atrial-ventricular. The temporary pacemakers employed did not have the capability to sense atrial activity. When the atrial pacemaker was employed there was no underlying atrial activity, however, there was normal intrinsic ventricular response. When the requirement for atrial activity was not a necessity ventricular pacemakers were utilized. If atrial-ventricular synchrony was required, an atrial-ventricular pacemaker was used. The atrial pacemaker was the most frequently used, with the largest occurence (n = 4) at baseline. At Measurement Sets 1 and 2, the atrial pacemaker was utilized in 3 subjects (14%). For Measurement Sets 3 and 4, atrial and atrial-ventricular pacing decreased to 2 subjects (10%). One subject had a ventricular pacemaker at Measurement Set 4.

Table 10

Electrocardiographic Rhythm at Measurement Sets

	Baseline	Measurement Set 1	Measurement Set 2	Measurement Set 3	Measurement Set 4
Rhythm	f	f	f	f	f
Sinus rhtymn	10	10	11	12	12
Sinus rhthym with 1° HB	2	0	0	1	1
Sinus rhthym with PVC	0	0	0	1	0
Sinus bradycardia	0	0	0	1	1
Sinus tachycardia	3	4	3	2	2
Sinus tach. with 1° HB	0	0	1	0	0
Atrial paceemaker	4	3	3	2	2
Ventricular pacemaker	0	0	0	0	1
A-V pacemaker	1	3	3	2	2
Other	1	1	0	0	0

Thermodilutional Cardiac Output Measurements

The volume of injectate used to perform TD CO varied between 30.00 ml. to 70.00 ml. among the four measurement sets. The largest amount of TD CO injectate occurred at Measurement Sets 1 and 4 ($\underline{M} = 38.10 \pm 11.67$ ml; 37.14 ± 11.46 ml, respectively). The time required to perform the TD CO varied from 3.19 ± 1.14 minutes to 3.95 ± 1.76 minutes over the four measurement sets. The longest time period occured at Measurement Set 3 ($\underline{M} = 3.95 \pm 1.47$ minutes), with the shortest at Measurement Set 4 ($\underline{M} = 3.19 \pm 1.72$ minutes). However, at Measurement Set 4 the greatest range of 8 minutes occured due to one subject who snored during the procedure, which increased the time required to perform the TD CO measurements to 10 minutes. However, if this subject is omitted, the maximum time at Measurement Set 4 is 4 minutes, although at Measurement Set 3 the maximum time required remains at 6 minutes (Table 11).

Table 11

Cardiac Ouput and Cardiac Index at Meaurement Sets

	Range	M	SD
Measurement Set 1			
CO (l/min)	3.26 - 8.89	5.84	1.46
CI (l/min/m2)	1.96 - 4.47	2.97	0.66
Time (mintues)	1.00 - 7.00	3.76	1.76
Volume (ml)	30.00 - 70.00	38.10	11.67
Measurement Set 2			
CO (l/min)	3.04 - 8.77	6.18	1.64
CI (l/min/m2)	2.16 - 4.74	3.14	0.71
Time (mintues)	2.00 - 6.00	3.60	1.14
Volume (ml)	30.00 - 60.00	38.10	9.28
Measurement Set 3			
CO (l/min)	4.38 - 9.76	6.63	1.36
CI (l/min/m2)	2.46 - 4.58	3.38	0.56
Time (minutes)	2.00 - 6.00	3.95	1.47
Volume (ml)	30.00 - 60.00	40.95	12.21
Measurement Set 4			
CO (l/min)	4.22 - 8.98	6.55	1.35
CI (l/min/m2)	2.19 - 4.56	3.35	0.61
Time (minutes)	2.00 - 10.00	3.19	1.72
Volume (ml)	30.00 - 70.00	37.14	11.46

To determine whether the CO, and hence CI, changed over time, repeated measures of variance (ANOVA) was conducted. There was no overall time effect found for CO or CI ($\underline{F} = 2.32$, $\underline{p} = .11$; $\underline{F} = 2.93$, $\underline{p} = .06$; respectively). The lowest CO and CI were obtained at Measurement Set 1 ($\underline{M} = 5.84 \pm 1.46$ l/min; $\underline{M} = 2.97 \pm .66$ l/min/m^2, respectively). At Measurement Set 3 the highest CO and CI were obtained ($\underline{M} = 6.63 \pm 1.36$ l/min, $\underline{M} = 3.38 \pm .56$ l/min./m^2, respectively) (Table 13). The smallest mean CO and CI difference of .08 ml/min and .03 ml/min/m^2, respectively occured between Measurements Sets 3 and 4 (Table 12).

Table 12

Analysis of Variance of Cardiac Output and Cardiac Index

	Diff.	SS	MS	DF	F	p
		Cardiac Output				
Measurement Set 1 vs Measurement Set 2	-.34	2.40	2.40	1/20	1.98	.18
Measurement Set 2 vs Measurement Set 3	-.45	4.27	4.27	1/20	2.12	.16
Measurement Set 3 vs Measurement Set 4	.08	.15	.15	1/20	.17	.69
		Cardiac Index				
Measurement Set 1 vs Measurement Set 2	-.17	.60	.60	1/20	2.02	.17
Measurement Set 2 vs Measurement Set 3	-.24	1.23	1.23	1/20	2.27	.15
Measurement Set 3 vs Measurement Set 4	.03	.02	.02	1/20	.11	.74

Correlation of Digital and Data Acquisition Pressure Measurements

Regression analysis was conducted to examine the accuracy between the digital and data acquisition system (DAS) hemodynamic pressure measurements at baseline and during the four measurement sets. The DAS hemodynamic pressure measurements were considered the gold standard, as graphic records of 300 data points were obtained over 15 seconds upon which to compare the digital recordings. The pulmonary artery systolic pressure (PAS), pulmonary artery diastolic pressure (PAD), mean pulmonary artery pressure (MPAP), and PAWP DAS and digital recordings were found to be significantly correlated at all four measurement sets (Table 13). For the RAP correlations there were modest or no significant correlations found between digital and DAS recordings (Table 14). The regression lines are provided in Appendix C.

Table 13

Correlations Between Data Acquisiton System and Digital Pulmonary Artery Systolic, Diastolic, and Mean Pressure Recordings

	Pre TD CO DAS	Pre TD CO DAS	Pre TD CO DAS	Post TD CO DAS	Post TD CO DAS	Post TD CO DAS
			Pulmonary Artery Systolic Pressure			
Measurement Set 1						
Digital	.97	.99	.99	.98	.99	.96
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 2						
Digital	.92	.98	.98	.99	.90	.97
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 3						
Digital	.98	.91	.97	.91	.90	.92
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 4						
Digital	.91	.89	.90	.95	.95	.95
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
			Pulmonary Artery Diastolic Pressure			
Measurement Set 1						
Digital	.94	.94	.98	.97	.91	.93
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 2						
Digital	.92	.93	.95	.93	.94	.95
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 3						
Digital	.88	.74	.77	.76	.81	.75
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 4						
Digital	.76	.73	.81	.80	.76	.78
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
			Mean Pulmonary Artery Pressure			
Measurement Set 1						
Digital	.96	.95	.98	.96	.93	.95
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 2						
Digital	.97	.96	.97	.98	.92	.95
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 3						
Digital	.90	.79	.87	.86	.82	.84
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 4						
Digital	.80	.80	.83	.78	.79	.89
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)

Note. DAS = data acquistion system, Pre TD CO = pressure measurement obtained before thermodilution cardiac output, Post TD CO = pressure measurement obtained after thermodilution cardiac output. $p \leq .05$.

Table 14

Correlations Between Data Acqusition System and Digital Pulmonary Artery Wedge Pressure and Right Atrial Pressure Recordings

	Pre TD CO DAS	Pre TD CO DAS	Pre TD CO DAS	Post TD CO DAS	Post TD CO DAS	Post TD CO DAS
			Pulmonary Artery Wedge Pressure			
Measurement Set 1						
Digital	.93	.92	.89	.93	.93	.89
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 2						
Digital	.90	.88	.92	.94	.97	.92
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 3						
Digital	.89	.76	.89	.75	.82	.79
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 4						
Digital	.82	.80	.89	.70	.80	.80
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
			Right Atrial Pressure			
Measurement Set 1						
Digital	.93	.89	.87	.79	.75	.90
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 2						
Digital	.86	.80	.90	.83	.83	.87
	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)	(p = .000)
Measurement Set 3						
Digital	.66	.46	.37	.42	.61	.39
	(p = .001)	(p = .036)	(p = .103)	(p = .059)	(p = .003)	(p = .081)
Measurement Set 4						
Digital	.34	.41	.59	.35	.35	.55
	(p = .126	(p = .065)	(p = .005)	(p = .119)	(p = .117)	(p = .009)

Note. DAS = data acquistion system, Pre TD CO = pressure measurement obtained before thermodilution cardiac output, Post TD CO = pressure measurement obtained after thermodilution cardiac output. $p \leq .05$.

Heart Rate and Hemodynamic Pressures Pre-Post Thermodilution Cardiac Output

To test the hypothesis that HR and hemodynamic pressures obtained prior to TD CO measurements are lower than compared to the HR and hemodynamic pressures acquired post TD CO measurements, repeated measures ANOVA was conducted. Since the DAS and digital pressures were generally correlated, the digital values were used for

analysis, except for systemic vascular resistance (SVRI) and right ventricular stroke work index (RVSWI). Means were calculated for the three HR and hemodynamic pressure measurements obtained before and then after TD CO measusrement within the four measurement sets. Therefore, there was a total of eight mean HR and hemodynamic pressure measurements based on 24 measurements. Hemodynamic pressure measurements consisted of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), PAS, PAD, MPAP, PAWP, and RAP. When the multivariate ANOVA tests were statistically significant, univariate tests are provided. Unless otherwise indicated, compound symmetry was met, as Mauchly's test of sphericity was statistically non significant. When Mauchly's test of sphericity was significant the Greenhouse-Geisser results are provided.

Heart Rate

There was no overall time effect found for HR obtained pre and post TD CO measurements ($F = .50$, $p = .74$). Between measurement sets, the mean HR ranged from 88.33 ± 13.42 bpm to 89.96 ± 12.13 bpm. Although not statistically significant, the pre TD CO measurement HR was lower than the post TD CO measurement at Measurement Set 3, with the mean HR being higher pre TD CO measurement in the other measurement sets (Table 15).

Table 15

Analysis of Variance of Heart Rate Means

	Pre TD CO		Post TD CO							
	M	SD	M	SD	Diff.	SS	MS	DF	F	p
Measurement Set 1	89.71	12.42	89.48	12.48	.24	.60	.60	1/20	.63	.44
Measurement Set 2	88.62	12.99	88.33	13.42	.29	.86	.86	1/20	1.06	.32
Measurement Set 3	88.76	13.08	89.14	12.26	-.38	1.52	1.52	1/20	.81	.38
Measurement Set 4	89.86	12.13	89.43	12.58	.43	1.93	1.93	1/20	.73	.40

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output, Diff = mean difference between pre and post TD CO measurement. $p = < .05$.

Arterial Blood Pressure

Systolic Blood Pressure. There was a statistically significant overall time effect found for mean SBP ($F = 4.17$, $p = .02$), with mean SBP obtained prior to TD CO measurements being lower than post TD CO measurements (Figure 2).The mean SBP ranged from 119.05 ± 16.65 mm Hg to 125.24 ± 19.75 mm Hg, with the lowest recorded value ($M = 119.05 \pm 16.65$ mm Hg) occurring pre TD CO measurement at Measurement Set 3. Measurement Set 3 had the largest mean pre-post TD CO difference of -5.48 mm Hg ($F = 13.61$, $p = .00$) (Table 16).

Diastolic Blood Pressure. There was also a statistically significant overall time effect found for mean DBP obtained prior to compared to after TD CO measurement ($F = 3.11$, $p = .04$) (Figure 3). The mean DBP means ranged from 58.10 ± 9.20 mm Hg to 60.67 ± 6.14 mm Hg over the four measurement sets. Within the measurement sets the mean DBP ranged from 40.00 ± 9.11 mm Hg to 78.00 ± 7.91 mm Hg. Similar to mean SBP, the lowest mean DBP was obtained prior to TD CO measurement at Measurement Set 3 ($M = 58.10 \pm 9.20$ mm Hg), giving the largest mean difference of -1.86 mm Hg

($F = 10.61$, $p = .00$). Similar to mean HR, at Measurement Set 2 the mean DBP pre TD CO was higher than the mean DBP post TD CO, with a mean difference of .10 mm Hg ($F = .05$, $p = .83$) (Table 16).

Mean Arterial Pressure. The MAP obtained prior to TD CO compared to after TD CO measurement was significantly lower over all four measurement sets ($F = 3.22$, $p = .04$) (Figure 4). The mean MAP ranged from 77.19 ± 9.90 mm Hg to 79.81 ± 9.92. The MAP pre TD CO (77.19 ± 9.90 mm HG) was lowest and the post TD CO MAP (79.81 ± 9.92 mm Hg) was the highest at Measurement Set 3 (Table 16). Similar to the mean SBP and mean DBP, the difference between pre and post TD CO measurements of the MAP was found to be statistically significant at Measurement Set 3 ($F = 8.98$, $p = .01$).

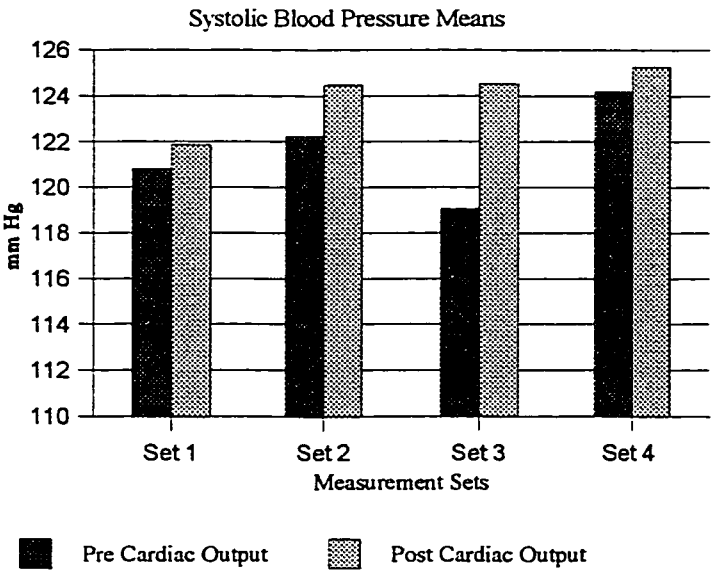

Figure 2. Systolic blood pressure pre-post TD CO.

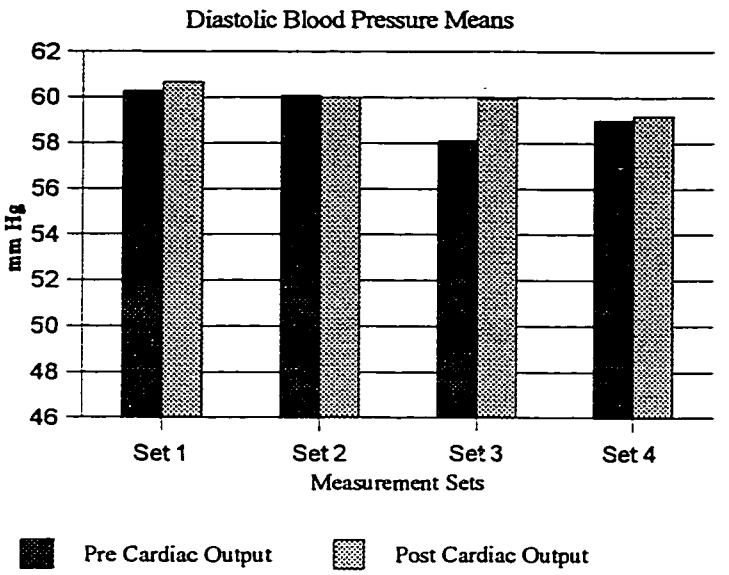

Figure 3. Diastolic blood pressure pre-post TD CO.

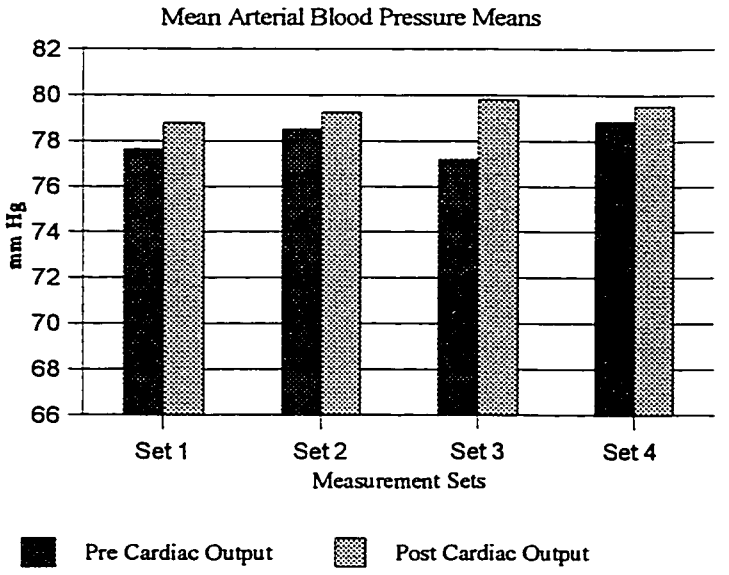

Figure 4. Mean arterial blood pressure pre-post TD CO.

Table 16

Anlaysis of Variance of Systolic, Diastolic, and Mean Arterial Blood Pressure Means

	Pre TD CO M	Pre TD CO SD	Post TD CO M	Post TD CO SD	Diff.	SS	MS	DF	F	p
Systolic Blood Pressure										
Measurement Set 1	120.81	13.75	121.86	13.84	-1.05	11.52	11.52	1/20	.42	.52
Measurement Set 2	122.24	19.15	124.48	18.65	-2.24	52.60	52.60	1/20	4.10	.06
Measurement Set 3	119.05	16.65	124.52	16.60	-5.48*	314.88	314.88	1/20	13.61	.00*
Measurement Set 4	124.19	16.55	125.24	19.75	-1.05	11.52	11.52	1/20	.42	.53
Diastolic Blood Pressure										
Measurement Set 1	60.29	6.91	60.67	6.14	-.38	1.52	1.52	1/20	.21	.65
Measurement Set 2	60.10	7.91	60.00	7.81	.10	.10	.10	1/20	.05	.83
Measurement Set 3	58.10	9.20	59.95	9.68	-1.86*	36.21	36.21	1/20	10.61	.00*
Measurement Set 4	59.00	8.87	59.19	9.11	-.19	.38	.38	1/20	.06	.81
Mean Arterial Blood Pressure										
Measurement Set 1	77.62	8.42	78.81	7.99	-1.19	14.88	14.88	1/20	1.41	.25
Measurement Set 2	78.52	9.84	79.24	9.09	-.71	5.36	5.36	1/20	1.62	.22
Measurement Set 3	77.19	9.90	79.81	9.92	-2.62*	72.02	72.02	1/20	8.98	.01*
Measurement Set 4	78.86	9.46	79.52	11.29	-.67	4.67	4.67	1/20	.43	.52

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output, Diff = mean difference between pre and post TD CO measurement. * $p \leq .05$.

Cardiac Pressures

Pulmonary Artery Systolic Pressure. There was a statistically significant overall time effect found for PAS obtained prior to compared to after TD CO measurement, with pre TD CO lower than post TD CO measurements ($F = 5.47, p = .01$). The mean PAS ranged from 31.14 ± 8.06 mm Hg to 34.19 ± 7.11 mm Hg over the four measurement sets (Figure 5). Similar to mean SBP, mean DBP, and mean MAP, during Measurement Set 3 the difference between the pre and post TD CO mean PAS (-.86 mm Hg) was found to be statistically significant ($F = 6.63$, $p = .02$). The lowest mean PAS pre TD CO measurement was obtained at Measurement Set 1 with the second largest mean difference

of -.57 mm Hg (F = 10.44, p = .00) (Table 17).

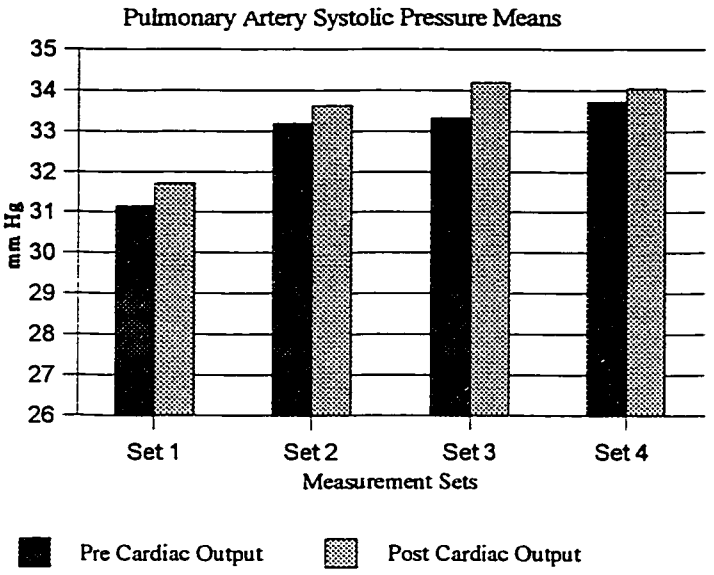

Figure 5. Pulmonary artery systolic pressures pre-post TD CO.

Pulmonary Artery Diastolic Pressure. There was no overall time effect found for PAD obtained prior to TD CO measurement compared to post TD CO measurement (F = .58, p = .68). The mean PAD ranged from 16.86 ± 2.87 mm Hg to 17.57 ± 3.54 mm Hg over the four measurement sets. At Measurement Set 4, the lowest mean PAD occured prior to TD CO measurement (M = 16.86 ± 2.87 mm Hg). The highest mean PAD was found at Measurement Set 2 post TD CO measurement (M = 17.57 ± 3.54 mm Hg). Although not statistically significant the PAD prior to TD CO measurement was higher than the post TD CO measurement at Measurement Set 1 (Table 17).

Mean Pulmonary Artery Pressure. There was no overall time effect found for MPAP obtained prior to TD CO measurement compared to after TD CO measurement with pre TD CO measurement tending to be lower than post TD CO measurements

($F = 1.61$, $p = .22$). Between the measurement sets the mean MPAP ranged from 22.52 ± 4.79 mm Hg and 24.05 ± 4.04 mm Hg. The lowest MPAP was found at Measurement Set 1 prior to TD CO measurement ($M = 22.52 \pm 4.79$ mm Hg). During Measurement Set 4 the highest mean MPAP was obtained post TD CO measurement ($M = 24.05 \pm 4.04$ mm Hg) (Table 17).

Pulmonary Artery Wedge Pressure. There was no overall time effect found for PAWP obtained prior to TD CO compared to post TD CO measurement ($F = 1.05$, $p = .41$). Between the measurement sets the mean PAWP ranged from 11.76 ± 3.49 mm Hg to 13.29 ± 2.55 mm Hg. At Measurement Set 1, the lowest men PAWP was obtained post TD CO measurement ($M = 11.76 \pm 3.49$ mm Hg). The highest mean PAWP was found post TD CO measurement at Measurement Set 3 ($M = 13.29 \pm 2.55$ mm Hg). The PAWP obtained pre TD CO measurement was higher than the post TD CO measurement within Measurement Set , although it was not significantly different (Table 17).

Right Atrial Pressure. There was no statistically significant overall time effect found for RAP obtained pre TD CO measurement compared to post TD CO measurement ($F = .53$, $p = .71$). Between the measurement sets the mean RAP ranged from 11.00 ± 1.92 mm Hg to 12.10 ± 3.08 mm Hg. The lowest mean RAP was found at Measurement Set 4 pre TD CO measurement ($M = 11.00 \pm 1.92$ mm Hg). At Measurement Set 2, the highest mean RAP post TD CO measurement ($M = 12.10 \pm 3.08$ mm HG) was recorded. At Measurement Set 1, the RAP obtained prior to TD CO measurement was higher than the post TD CO measurement, although not significantly different (Table 17).

Repeated measures ANOVA was also performed on the RAP DAS values, as the

RAP digital and DAS pressure measurements within Measurement Sets 3 and 4 were not found to be significantly correlated (Table 14). There was no statistically significant overall time effect found for the RAP DAS obtained prior to TD CO measurement compared to post TD CO measurement ($F = 2.12$, $p = .12$). Similar to the digital mean RAP, mean HR, mean PAWP, and mean PAD, the RAP obtained prior to TD CO was higher than the post TD CO at Measurement Set 1, although the difference was not statistically significant.

Table 17

Analysis of Variance of Pulmonary Artery Pressures and Right Atrial Pressure Means

	Pre TD CO M	Pre TD CO SD	Post TD CO M	Post TD CO SD	Diff.	SS	MS	DF	F	p
Pulmonary Artery Systolic Pressure										
Measurement Set 1	31.14	8.06	31.71	7.96	-.57*	3.43	3.43	1/20	10.44	.00*
Measurement Set 2	33.19	7.32	33.62	7.88	-.43	1.93	1.93	1/20	2.65	.12
Measurement Set 3	33.33	7.05	34.19	7.11	-.86*	7.71	7.71	1/20	6.63	.02*
Measurement Set 4	33.71	6.19	34.05	7.24	-.33	1.17	1.17	1/20	.36	.55
Pulmonary Artery Diastolic Pressure										
Measurement Set 1	17.24	3.32	17.19	3.36	.05	.02	.02	1/20	.03	.87
Measurement Set 2	17.43	3.19	17.57	3.54	-.14	.21	.21	1/20	.26	.61
Measurement Set 3	17.33	3.31	17.48	3.11	-.14	.21	.21	1/20	.38	.55
Measurement Set 4	16.86	2.87	17.33	3.23	-.48	2.38	2.38	1/20	2.20	.15
Mean Pulmonary Artery Pressure										
Measurement Set 1	22.52	4.79	23.00	4.70	-.48	2.38	2.38	1/20	2.87	.11
Measurement Set 2	23.33	4.29	23.76	4.60	-.43	1.93	1.93	1/20	3.33	.08
Measurement Set 3	23.57	4.51	23.90	4.05	-.33	1.17	1.17	1/20	1.63	.22
Measurement Set 4	23.67	3.54	24.05	4.04	-.38	1.52	1.52	1/20	.92	.35
Pulmonary Artery Wedge Pressure										
Measurement Set 1	12.05	3.35	11.76	3.49	.29	.86	.86	1/20	.95	.34
Measurement Set 2	12.19	2.86	12.38	2.82	-.19	.38	.38	1/20	.88	.36
Measurement Set 3	12.86	3.05	13.29	2.55	-.43	1.93	1.93	1/20	2.20	.15
Measurement Set 4	13.14	2.56	13.24	2.57	.10	.10	.10	1/20	.09	.77
Right Atrial Pressure										
Measurement Set 1	11.95	3.26	11.81	2.06	.14	.21	.21	1/20	.30	.60
Measurement Set 2	12.00	2.93	12.10	3.08	-.10	.01	.01	1/20	.13	.72
Measurement Set 3	11.33	2.33	11.57	2.20	-.24	.60	.60	1/20	.80	.39
Measurement Set 4	11.00	1.92	11.38	2.16	-.38	1.52	1.52	1/20	.97	.34

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output, Diff = mean difference between pre and post TD CO measurement. * $p \leq .05$.

Derived Hemodynamic Parameters Pre- Post Thermodilution Cardiac Output

To test the hypothesis that derived hemodynamic parameters calculated using the hemodynamic pressures means obtained prior to TD CO measurements were lower than compared to the derived hemodynamic parameters calculated using the hemodynamic

pressures means obtained post TD CO measurements, repeated measures ANOVA was conducted using the digital hemodynamic parameters. Analysis of covariance was then conducted to control for the variables of : (a) level of PEEP and PS; (b) AWP; (c) core body temperature; (d) CPB anad ACC time; (e) fluid balance; (f) hypoxia (saturation $\{SaO_2\}$ and PaO_2); (g) BE level; (h) medications (inotropes; vasodilators and vasopressors; sedatives and analgesia); and (i) volume of TD CO injectate required (Appendix D).

The means were calculated for the three derived hemodynamic parameters obtained before and after TD CO measurement, within the four measurement sets. Therefore, there were a total of eight means for the derived hemodynamic parameters based on 24 measurements. Derived hemodynamic parameters consisted of SV, SVI, left ventricular stroke work index (LVSWI), right ventricular stroke work index (RVSWI), systemic vascular resistance index (SVRI), and pulmonary vascular resistance index (PVRI).

Afterload Indicators

Systemic Vascular Resistance Index. There was a statistically significant overall time effect found for SVRI obtained prior to TD CO measurement compared to post TD CO measurement, with pre TD CO measurement being lower than post TD CO measurements ($F = 144.22$, $p = .01$) (Figure 6). The mean SVRI ranged from 1600.81 ± 362.42 dynes/sec/cm^{-5}/m^2 to 1875.43 ± 440.02 dynes/sec/cm^{-5}/m^2. Similar to mean SBP, mean DBP, mean MAP, and mean PAS, the difference between pre and post TD CO measurement of SVRI was found to be statistically significant at Measurement Set 3 ($F = 6.87$, $p = .02$). Measurement Set 3 had the lowest mean SVRI pre TD CO

measurement ($\underline{M}$ = 1600.81 ± 362. 42 dynes/sec/cm^{-5}/m^2) with the largest pre-post mean difference of -60.38 dynes/sec/cm^{-5}/m^2 ($\underline{p}$ = .02) (Table 18).

Repeated measures ANOVA was also conducted on the SVRI determinations calculated from DAS pressure measurements, as there were nonsignificant correlations between the digital and DAS RAP pressure measurements within Measurement Sets 3 and 4 (Table 14). There was a similar overall time effect found for DAS SVRI obtained pre TD CO measurement compared to post TD CO measurement ($\underline{F}$ = 3.97, $\underline{p}$ = .02). As well, the DAS SVRI obtained prior to TD CO measurements at Measurement Set 3 was found to be significantly lower than the post TD CO measurement, with the largest mean difference of -54.19 dynes/sec/cm^{-5}/m^2 ($\underline{F}$= 6.46, $\underline{p}$ = .02).

In the determination of mean SVRI pre TD CO measurement compared to post TD CO measurement, several covariates were examined. Aortic cross clamp time and CPB time were not found to influence SVRI . There was also no effect found for SaO_2, BE, Epinephrine, and Sodium Nitroprusside (SNP). However, when adjusting for the effect of PaO_2 obtained at Measurement Set 1 the mean SVRI pre TD CO at Measurement Set 2 became significantly lower than the post TD CO measurement ($\underline{F}$ = 7.57, $\underline{p}$ = .01), with a mean difference of -26.95 dynes/sec/cm^{-5}/m^2 ($\underline{p}$ = .04). When PEEP and PS were independently controlled, the mean SVRI difference of -41.21 dynes/sec/cm^{-5}/m^2 at Measurement Set 3 became nonsignificant ($\underline{p}$ = .11; $\underline{p}$ = .12, respectively). Similar to PEEP and PS, when controlling for AWP, no significant difference was found between the pre and post TD CO determination of SVRI at Measurement Set 3 (Mean difference = -41.46 dynes/sec/cm^{-5}/m^2, $\underline{p}$ = .19). At Measurement Set 4, when controlling for the

effect of CO injectate volume, mean SVRI pre TD CO became significantly lower compared to post TD CO ($F = 8.25$, $p = .01$), although the difference was not significant, similarly when controlling for the effect of the covariate of CBT at baseline ($F = 2.07$, $p = .02$). The mean SVRI at Measurement Set 4 became significantly higher post TD CO when controlling for the negative fluid balance at Measurement Set 1 ($F = 4.55$, $p = .05$) and the positive fluid balance at Measurement Set 2 ($F = 8.91$, $p = .01$). When controlling for the effect of Nitroglycerine at Measurement Set 1, mean SVRI at Measurement Set 2 became significantly lower pre TD CO compared to post TD CO ($F = 4.47$, $p = .05$), giving a mean difference of -26.95 dynes/sec/cm^{-5}/m^2 ($p = .02$). When removing the effect of being on Demerol, Morphine, and Diprivan at Measurement Set 2 mean SVRI became significantly lower pre TD CO compared to post TD CO at Measurement Set 2 ($F = 6.51$, $p = .02$) and at Measurement Set 3 ($F = 6.00$, $p = .03$), although the differences did not reach statistical significance. .

Pulmonary Artery Vascular Resistance Index. There was a statistically significant overall time effect found for the PVRI determined prior to TD CO compared to post TD CO ($F = 6.45$, $p = .00$). The mean PVRI ranged from 236. 76 $\pm$ 80.90 dynes/sec/cm^{-5}/m^2 to 314.67 $\pm$ 113.34 dynes/sec/cm^{-5}/m^2 (Figure 7). Similar to mean PAS, the largest mean difference of -22.57 dynes/sec/cm^{-5}/m^2 occured at Measurement Set 1($F = 10.45$, $p = .00$) (Table 18).

Mean PVRI was not influenced by the ACC and CPB time, SaO_2, BE, PEEP, PS, AWP, volume of CO injectate, fluid balance, Epinephrine, SNP, Nitroglycerine, Demerol, Morphine, and Diprivan. However, at Measurement Set 3 mean PVRI became

significantly higher pre TD CO compared to post when controlling for the effect of PaO_2 at Measurement Set 1 ($F = 7.97$, $p = .01$), although the mean difference did not reach statistical significance ($p = .80$). Also, at Measurement Set 4 mean PVRI became significantly lower pre TD CO compared to post TD CO when adjusting for PaO_2 at Measurement Set 3 ($F = 7.73$, $p = .01$), although the mean difference was not significant ($p = .70$). At Measurement Set 3, when controlling for the effect of core temperature at the end of surgery, mean PVRI pre TD CO was statistically higher than post TD CO ($F = 10.70$, $p = .01$), although the mean difference did not reach statistical significance ($p = .79$). Also, at Measurement Set 4, when controlling for the effect of core temperature at baseline, mean PVRI pre TD CO became significantly lower than post TD CO ($F = 4.64$, $p = .05$), yet the mean difference was not significant ($p = .74$).

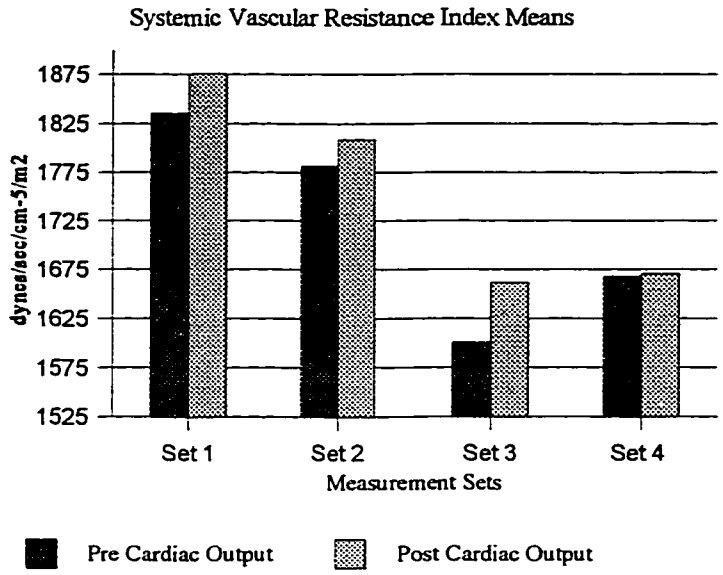

Figure 6. Systemic vascular resistance index pre-post TD CO.

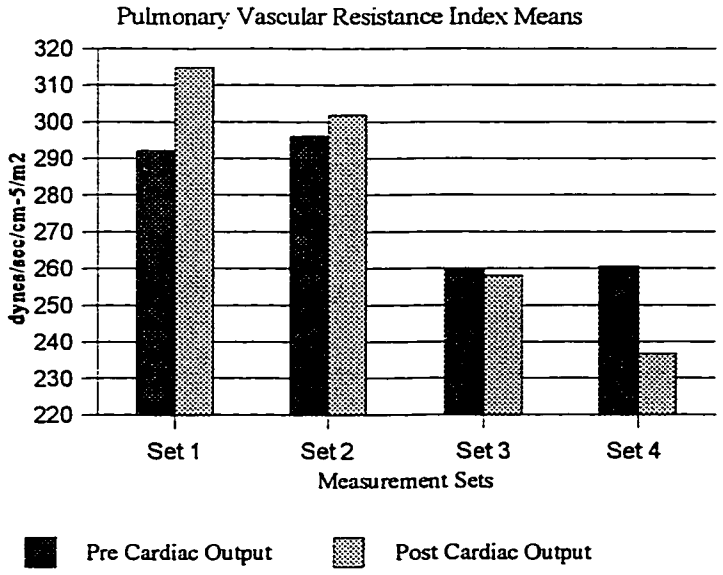

Figure 7. Pulmonary vascular resistance index pre-post TD CO.

Table 18

Analysis of Variance of Systemic and Pulmonary Vascular Resistance Index Means

	Pre TD CO M	Pre TD CO SD	Post TD CO M	Post TD CO SD	Diff.	SS	MS	DF	F	p
Systemic Vascular Resistance Index										
Measurement Set 1	1835.19	412.69	1875.43	440.02	-40.24	17000.60	17000.60	1/20	1.60	.22
Measurement Set 2	1781.24	472.09	1808.19	469.85	-26.95	7627.52	7627.52	1/20	3.09	.09
Measurement Set 3	1600.81	362.42	1661.19	373.92	-60.38*	38281.52	38281.52	1/20	6.87	.02*
Measurement Set 4	1667.67	351.27	1670.52	359.83	-2.86	85.71	85.71	1/20	.01	.92
Pulmonary Vascular Resistance Index										
Measurement Set 1	292.10	108.00	314.67	113.34	-22.57*	5349.43	5349.43	1/20	10.45	.00*
Measurement Set 2	296.10	87.34	301.81	95.75	-5.71	342.86	342.86	1/20	.97	.34
Measurement Set 3	260.10	79.73	258.14	80.11	1.95	40.02	40.02	1/20	.05	.83
Measurement Set 4	260.57	75.80	263.76	80.09	-3.19	106.88	106.88	1/20	.11	.74

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output, Diff = mean difference between pre and post TD CO measurement. * $p \leq .05$.

Contractility Indicators

Stroke Volume. There was no overall time effect found for the determination of SV prior to TD CO compared to post TD CO ($F = .51$, $p = .73$). The SV ranged from 66.23 $\pm$ 16.81 ml/beat to 77.40 $\pm$ 23.41 ml/beat (Figure 8). Similar to mean PVRI, at Measurement Set 3 mean SV was higher pre TD CO compared to post TD CO, although the difference was not statistically significant (Table 19).

When adjusting for the effects of PaO_2, SaO_2, BE, CPB and ACC time, fluid balance, volume of CO injectate, SNP, Demerol, Morphine, and Diprivan, there was no alteration in the mean SV pre TD CO compared to post TD CO. When PEEP and PS were independently controlled at Measurement Set 4, the mean differences of -.66 ml/beat between pre and post TD CO became statistically significant ($p = .01$; $p = .01$, respectively). At Measurement Set 4, when controlling for the effect of AWP at

Measurement Set 1, mean SV became significantly lower pre TD CO compared to post TD ($F = 4.77$, $p = .05$), although the mean difference was small (Mean difference = -.72 ml/beat, $p = .00$). At Measurement Set 3, when controlling for the effect of core temperature at Measurement Sets 1 and 2, mean SV became significantly higher pre TD CO compared to post TD CO ($F = 16.97$, $p = .00$; $F = 16.84$, $p = .00$, respectively). Also, when adjusting for the effect of core temperature at baseline and Measurement Set 1 mean SV became significantly lower pre TD CO compared to post TD CO at Measurement Set 4 ($F = 13.17$, $p = .00$; $F = 8.61$, $p = .01$, respectively). When controlling for the effect of Epinephrine at Measurement Set 3, it was found that mean SV at Measurement Set 4 became significantly higher pre TD CO compared to post TD CO ($F = 5.14$, $p = .04$), although the mean difference did not reach statistical significance ($p = .38$). At Measurement Set 2, when controlling for the effect of Nitroglycerine, mean SV became significantly higher pre TD CO compared to post TD CO ($F = 6.97$, $p = .02$), although the mean difference was not significant ($p = .18$).

Stroke Volume Index. Similar to SV, there was no overall time effect found for SVI obtained prior to TD CO measurement compared to post TD CO measurement ($F = .60$, $p = .67$). This was to be expected as the former is determined by SV and body surface area. The mean SVI ranged from 33.71 ± 7.99 m/beat/m^2 to 39.39 ± 10.90 m/beat/m^2 (Figure 9). Similar to mean PVRI and mean SV, at Measurement Set 3 the mean SVI was higher pre TD CO compared to post TD CO, although this difference was not statisticaily significant (Table 19).

When controlling for the effects of PaO_2, BE, CPB and ACC time, fluid balance,

volume of CO injectate, SNP, Demerol, Morphine, and Diprivan, there was no alteration in the statistical significance of mean SVI pre TD CO compared to post TD CO measurement. At Measurement Set 3, when controlling for the effect of SaO_2, the mean SVI became significantly higher pre TD CO compared to post TD CO ($F = 4.84$, $p = .04$), although the mean difference was not significant ($p = .29$). When PEEP and PS were independently controlled the pre-post TD CO SVI mean differences (-.36 ml/beat/m^2) became statistically significant at Measurement Set 4 ($p = .00$; $p = .00$, respectively). Similar to SV, at Measurement Set 4 when controlling for the effect of AWP at Measurement Set 1, mean SVI pre TD CO became significantly lower compared to post TD CO ($F = 5.45$, $p = .04$), giving a mean difference of -.39 ml/beat/m^2 ($p = .00$). Similar to SV, at Measurement Set 3 when controlling for the effect of core temperature at Measurement Sets 1 and 2, mean SVI became significantly higher pre TD CO compared to post TD CO ($F = 18.37$, $p = .00$; $F = 18.48$, $p = .00$, respectively). Also similar to SV, when adjusting for the effect of core temperature at baseline and Measurement Set 1, mean SVI at Measurement Set 4 became significantly lower pre TD CO compared to post TD CO ($F = 12.83$, $p = .00$; $F = 8.06$, $p = .01$). However, like SV, the mean differences were not statistically significant ($p = .19$; $p = .33$, respectively). Similar to SV, when controlling for the effect of Epinephrine at Measurement Set 3, mean SVI at Measurement Set 4 pre TD CO became significantly lower than post TD CO ($F = 5.44$, $p = .03$), although the mean difference was not significant ($p = .32$). Similar to SV, at Measurement Set 2 when controlling for the effect of Nitroglycerine, mean SVI pre TD CO became significantly lower compared to post TD CO ($F = 5.96$, $p = .03$), although the mean

difference was not significant ($p = .16$).

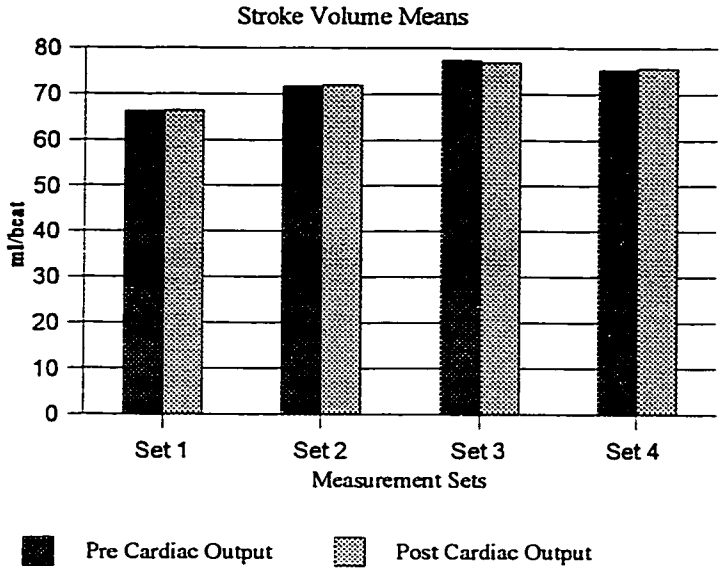

Figure 8. Stroke volume pre-post TD CO.

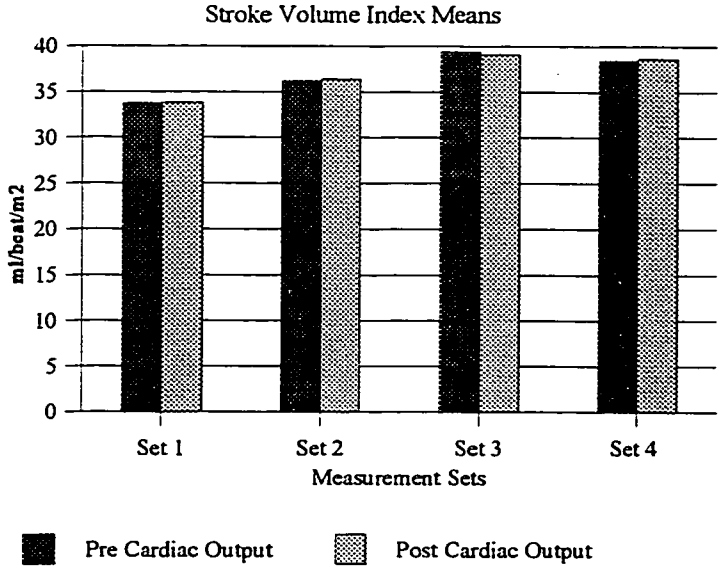

Figure 9. Stroke volume index pre-post TD CO.

Left Ventricular Stroke Work Index. The LVSWI obtained prior to TD CO compared to after TD CO measurements was not significantly different over time ($F = 2.02$, $p = .14$). The mean LVSWI ranged from 30.16 ± 8.34 gr-m/m^2/beat to 35.30 ± 10.41 gr-m/m^2/beat (Figure 10). Measurement Set 3 had the largest mean LVSWI post TD CO measurement ($M = 35.30 \pm 10.41$ gr-m/m^2/beat), giving the largest pre-post TD O mean difference of -.87 gr-m/m^2/beat ($F = 3.38$, $p = .08$) (Table 19).

When adjusting for the effects of SaO_2, BE, AWP, CPB and ACC time, SNP, Demerol, Morphine, and Diprivan, there were no changes in the mean LVSWI pre TD CO compared to post TD CO. Similar to PVRI, an interaction effect was found between PaO_2 at Measurement Set 1 with mean LVSWI at Measurement Set 2 ($F = 6.42$, $p = .02$), and between PaO_2 and mean LVSWI at Measurement Set 3 ($F = 4.63$, $p = .05$). The mean LVSWI obtained pre TD CO became statistically lower than post TD CO at Measurement Sets 2 and 3, giving mean differences of -.53 gr-m/m^2/beat ($p = .05$) and -.87 gr-m/m^2/beat ($p = .05$), respectively. When PEEP and PS were independently controlled the mean differences of -1.02 gr-m/m^2/beat became significant at Measurement Set 1 ($p = .04$; $p = .04$, respectively). When controlling for the effect of core temperature at baseline and Measurement Set 1, at Measurement Set 3 mean LVSWI became significantly lower pre TD CO compared to post TD CO ($F = 4.48$, $p = .05$; $F = 6.14$, $p = .03$, respectively), with a mean difference of -.87 gr-m/m^2/beat ($p = .05$). At Measurement Set 4, when controlling for the effect of fluid balance at Measurement Set 2, mean LVSWI became found to be significantly lower pre TD CO compared to post TD CO ($F = 5.20$, $p = .04$), although the mean difference was not significant ($p = .19$). Similar to SVRI, at

Measurement Set 4, when controlling for the effect of volume of CO injectate mean LSWI became significantly lower pre TD CO compared to post TD CO ($F = 5.25$, $p = .04$), although the mean difference was not significant ($p = .21$). When controlling for the effect of Nitroglycerine at Measurement Set 2 mean LVSWI at Measurement Set 3 became significantly higher post TD CO ($F = 5.87$, $p = .03$) with a mean difference of -.87 gr-m/m2/beat ($p = .03$). At Measurement Set 3, when removing the effect of Epinephrine, mean LVSWI pre TD CO became significantly higher post TD CO compared to pre TD CO ($F = 5.71$, $p = .03$), although the mean difference was not significant ($p = .07$).

Right Ventricular Stroke Work Index. There was no overall time effect found for RVSWI determination pre TD CO compared to post TD CO ($F = 1.57$, $p = .23$). The mean RVSWI varied from 4.76 ± 1.53 gr-m/m^2/beat to 6.71 ± 2.99 gr-m/m^2/beat (Figure 11). At Measurement Set 1 the lowest mean RVSWI was obtained prior to TD CO measurement ($M = 4.76 \pm 1.53$ gr-m/m^2/beat) with the largest mean difference of -.35 gr-m/m^2/beat ($F = 3.80$, $p = .07$). Similar to mean SV, mean SVI, and mean PVRI, at Measurement Set 3 the pre TD CO determination of RVSWI was greater than post TD CO determination of RVSWI, although this difference was not statistically significant (Table 19).

Repeated measures ANOVA was conducted on the RVSWI determinations calculated from DAS pressure measurements, as there were statistically non significant correlations of the digital and DAS RAP pressure measurements within Measurement Sets 3 and 4 (Table 14). In contrast to the digital results, there was an overall time effect found for RVSWI DAS obtained pre TD compared to post TD CO, with the RVSWI obtained

prior to TD CO being lower than RVSWI post TD CO ($F = 3.27$, $p = .04$). The mean DAS RVSWI ranged from 4.61 ± 1.81 gr-m/m^2/beat to 7.14 ± 2.82 gr-m/m^2/beat. Measurement Set 4 had the highest mean DAS RVSWI pre TD CO measurement ($M = 7.14 \pm 2.82$ gr-m/m^2/beat), giving the largest mean difference of -.45 gr-m/m^2/beat ($F = 6.49$, $p = .02$). Measurement Set 1 had the lowest mean RVSWI pre TD CO measurement ($M = 4.91 + 1.81$ gr-m/m^2/beat), with a mean difference of -.40 gr-m/m^2/beat ($F = 6.25$, $p = .02$) (Table 19).

When controlling for the influence of PaO_2, SaO_2, PEEP, PS, AWP, CPB and ACC time, CBT, volume of CO injectate, Epinephrine and Nitroglycerine, mean digital RVSWI pre TD CO remained similar to post TD CO. At Measurement Set 1 an interaction effect was found between BE and mean RVSWI ($F = 4.32$, $p = .05$). Mean RVSWI became significantly lower pre TD CO compared to post TD CO, giving a mean difference of -.35 gr-m/m^2/beat ($p = .05$). Also at Measurement Set 1, when controlling for the effect of fluid balance at baseline, the mean RVSWI became significantly lower pre TD CO compared to post TD CO ($F = 5.34$, $p = .04$), although the mean difference was very small (Mean difference = -.35 gr-m/m^2/beat, $p = .05$). Similar to BE and fluid balance, at Measurement Set 1 when adjusting for the effect of SNP, the mean RVSWI pre TD CO became significantly lower than post TD CO ($F = 6.36$, $p = .02$), with a small mean difference of -.35 gr-m/m^2/beat ($p = .03$). Also, similar to BE, fluid balance, and SNP, at Measurement Set 1 when controlling for the effect of Demerol, Diprivan, and Morphine the mean RVSWI was found to be significantly lower pre TD CO compared to post TD CO ($F = 6.23$, $p = .02$), although the mean difference was small (Mean difference = -.35

gr-m/m^2/beat, p = .01).

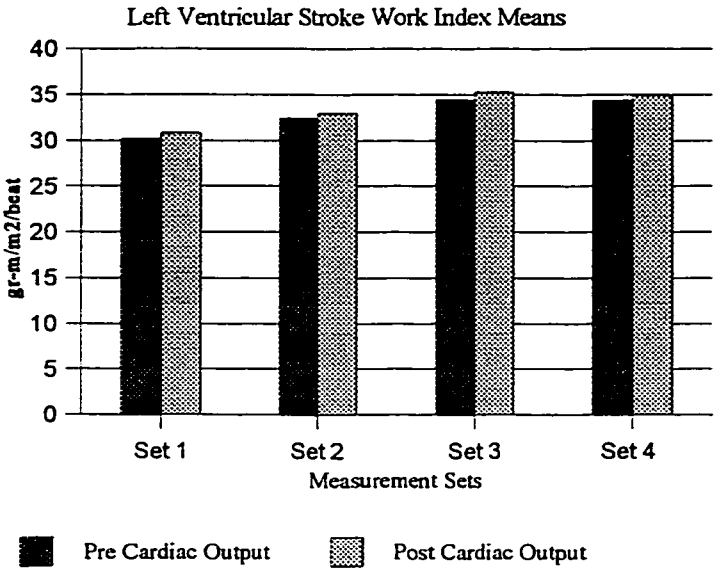

Figure 10. Left ventricular stroke work index pre-post TD CO.

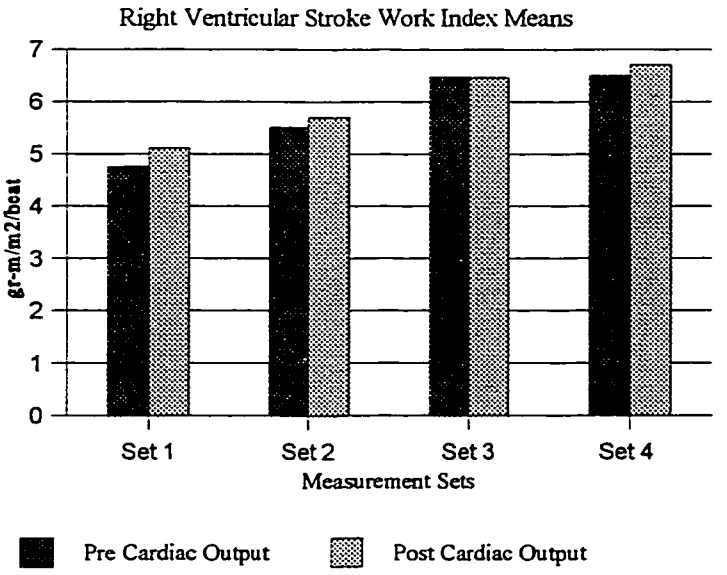

Figure 11. Right ventricular stroke work index pre-post TD CO.

Table 19

Analysis of Variance of Stroke Volume, Stroke Volume Index, and Left and Right Stroke Work Index Means

	Pre TD CO		Post TD CO							
	M	SD	M	SD	Diff.	SS	MS	DF	F	p
Stroke Volume										
Measurement Set 1	66.23	16.81	66.42	17.05	-.11	.38	.38	1/20	.54	.47
Measurement Set 2	71.58	21.92	71.94	22.32	-.36	1.34	1.34	1/20	1.59	.23
Measurement Set 3	77.40	23.41	76.86	22.94	.53	2.99	2.99	1/20	.95	.34
Measurement Set 4	75.27	22.09	75.57	22.17	-.30	.95	.95	1/20	.55	.47
Stroke Volume Index										
Measurement Set 1	33.71	7.99	33.83	8.13	-.11	.14	.14	1/20	.78	.39
Measurement Set 2	36.20	9.89	36.37	10.06	-.18	.33	.33	1/20	1.78	.20
Measurement Set 3	39.39	10.90	39.12	10.68	.27	.75	.75	1/20	1.06	.32
Measurement Set 4	38.42	10.61	38.59	10.70	-.17	.31	.31	1/20	.72	.41
Left Ventricular Stroke Work Index										
Measurement Set 1	30.16	8.34	30.79	8.21	-.63	4.15	4.15	1/20	2.94	.10
Measurement Set 2	32.41	9.09	32.95	9.03	-.53	2.99	2.99	1/20	3.21	.09
Measurement Set 3	34.44	10.55	35.30	10.41	-.87	7.89	7.89	1/20	3.38	.08
Measurement Set 4	34.38	10.79	34.91	11.29	-.53	2.93	2.93	1/20	1.57	.23
Right Ventricular Stroke Work Index (Digital)										
Measurement Set 1	4.76	1.53	5.11	1.77	-.35	1.23	1.23	1/20	3.80	.07
Measurement Set 2	5.51	2.14	5.70	2.07	-.19	.38	.38	1/20	2.40	.14
Measurement Set 3	6.48	2.39	6.46	2.18	.03	.01	.01	1/20	.02	.90
Measurement Set 4	6.50	2.09	6.71	2.99	-.21	.48	.48	1/20	.57	.46
Right Ventricular Stroke Work Index (DAS)										
Measurement Set 1	4.91	1.81	5.31	2.22	-.40*	1.65	1.65	1/20	6.25	.02*
Measurement Set 2	5.97	2.86	6.10	2.88	-.13	.18	.18	1/20	.53	.48
Measurement Set 3	7.03	2.48	6.83	2.52	.19	.39	.39	1/20	1.29	.27
Measurement Set 4	6.69	2.43	7.14	2.82	-.45*	.48	2.15	1/20	6.49	.02*

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output, Diff = mean difference between pre and post TD CO measurement. * $p = < .05$.

CHAPTER FIVE

Discussion of Findings

The purpose of this study was to determine the effect of the timing of hemodynamic pressure measurements with thermodilutional (TD) cardiac output (CO) on the derived hemodynamic parameters. There were four sets of measurements, with three hemodynamic pressures obtained prior to and three after determination of the TD CO measurements, providing for a pre and post TD CO comparison of derived hemodynamic parameters. Thirty minutes lapsed between Measurements Set 1 and 2; 4 hours between Measurement Sets 2 and 3; and 30 minutes between Measurement Sets 3 and 4. A set of hemodynamic pressures consisted of heart rate (HR); systolic, diastolic, and mean arterial blood pressure; pulmonary artery systolic, diastolic, and mean pressure; pulmonary artery wedge pressure (PAWP); and right atrial pressure (RAP). A set of derived hemodynamic parameters consisted of stroke volume (SV), stroke volume index (SVI), systemic and pulmonary vascular resistance, and right and left ventricular stroke work index.

Twenty-one subjects met the inclusion criteria for the study. Seventeen subjects were male with coronary artery bypass graft surgery (67%) being the most prevalent surgical procedure. Throughout this study all of the subjects remained stable, requiring minimal intervention. Serum hematocrit, hemoglobin, potassium, and magnesium values were within intended ranges for the cardiac surgical population. The mean serum hematocrit level was $.30 \pm .05$ mmol/l suggesting hemodilution. Hemodilution may decrease cardiac workload, as afterload is decreased, with a concomitant increase in

contractility, SV, and CO (Biga & Bethel, 1991; Meehan, 1986; Klinger, 1996; Urban, 1986). However, hypovolemia was not present, as there was only a small negative fluid balance. During all measurement sets, partial pressure of oxygen (PaO_2), oxygen saturation (SaO_2), hydrogen ion concentation, base excess (BE), and partial pressure of carbon dioxide ($PaCO_2$) reflected normal oxygenation and ventilation status. Positive end expiratory pressure, (PEEP), pressure support (PS), airway pressure (AWP), core body temperature, fluid balance, and use of medications varied minimally over the four measurement sets.

Repeated measures analysis of variance (ANOVA) was conducted to test the hypothesis that hemodynamic pressure measurements and derived hemodynamic parameters obtained pre TD CO were lower than post TD CO. Analysis of covariance was also conducted on derived hemodynamic parameters to control for: (a) level of PEEP and PS; (b) airway pressure; (c) core body temperature (CBT); (d) aortic cross clamp (ACC) and cardiopulmonary bypass (CBP) time; (e) fluid balance; (f) hypoxia (SaO_2 and PaO_2 levels); (g) base excess ; (h) dosage of inotropes, vasodilators, vasopressors, sedatives, and analgesia; and (i) volume of TD CO injectate required.

Clinical significance was also considered. The normal fluctuation of PAWP, pulmonary artery diastolic pressure (PAD), and RAP is less than 4 millimeters of mercury (mm Hg). Therefore, clinical significance was considered greater than 4 mm Hg of variation. The normal fluctuation of pulmonary artery systolic pressure (PAS) is less than 5 mm Hg, therefore, clinical significance was considered greater than $\pm$ 5 mm Hg (Nemens & Woods, 1982). The hemodynamic pressures, derived hemodynamic parameters, and TD

CO and cardiac index (CI) were considered clinical significant if greater than 10% variance occured. The accepted CO biological variability is less than 10% (Kadota, 1985; Lynch & Kaemmerer, 1990; Sasse, Chen, Berry, Sassoon, & Mahutte, 1994; Vincent, 1994).

Pulmonary artery systolic pressure, PAD, mean pulmonary artery pressure (MPAP), PAWP, and RAP were recorded digitally and via the data acquisition system (DAS). It is highly recommended that intracardiac pressures be recorded graphically to eliminate respiratory influences and instrumentation issues (Cathley, 1997; Dolter, 1989; Gengiz, Crapo, & Garnder, 1983; Keckeisen, 1998; Lipp-Ziff & Kawanishi, 1991; Wilson et al., 1996). Therefore, the DAS hemodynamic pressure measurements were considered the gold standard, as graphic records of 300 data points were obtained over 15 seconds upon which to compare the digital recordings. The difference between the pre and post DAS baseline data was never greater than 2 mm Hg. Therefore, the digital determinations were used in data analysis, since within the clinical setting there is no access to the DAS. The PAS, PAD, MPAP, and PAWP digital and DAS recordings were found to be significantly correlated. However, at Measurement Sets 3 and 4, the DAS RAP was signficantly less than the digital recordings. Therefore, upon interpretation of any results that included the RAP at Measurement Sets 3 and 4, measurement error was considered.

Derived Hemodynamic Parameters

The majority of derived hemodynamic parameters had no differences between pre TD CO compared to post TD CO. Only systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), and DAS right ventricular stroke work index

(RVSWI) were found to be significantly different. At Measurement Set 1, mean PVRI and mean DAS RVSWI were found to be significantly lower pre TD CO compared to post TD CO. At Measurement Set 3, mean SVRI was found to be significantly lower pre TD compared to post TD CO. Similarly, the majority of hemodynamic pressures were similar pre TD CO compared to post TD CO. Only systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) at Measurement Set 3, and PAS at Measurement Sets 1 and 3; were significantly lower pre TD CO compared to post TD CO.

Afterload Indicators

Afterload is the impedance to blood flow from the right and left ventricles during systole. As resistance increases, SV decreases (Beique & Ramsey, 1994; Hewlett Packard, 1985; Kiess-Daily & Schroeder, 1994; Meehan, 1986; Ramsey & Tisdale, 1995; Urban, 1986). Afterload is affected by blood viscosity, radius and length of vessels, condition of cardiac valves, and ventricular radius and wall tension (Berne & Levy, 1992; Biga & Bethel, 1986; Klinger, 1996).

Systemic Vascular Resistance Index. The resistance to ejection of blood flow into the aorta adjusted for body surface area (BSA) is referred to as SVRI. Mean arterial pressure, and CI are the determinants of SVRI (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994) and these determinants must be examined when evaluating left ventricular afterload. Therefore, the interrelationship of pressure difference across vessels and flow will determine left ventricular workload. Overall, between Measurement Sets 1 and 3, SVRI decreased by more than 10%, as well as between Measurement Sets 1 and 4. However, SVRI did not differ by greater than 10% pre-post TD CO at any of the four

measurement sets. As well SBP, DBP, MAP, and RAP did not vary by greater than 10% among the four measurement sets or between pre-post TD CO recordings. Cardiac index and CO also increased by more than 10% between Measurement Sets 1 and 3, and between Measurement Sets 1 and 4. The concomitant improvement in CI and the reduction in left ventricular workload would be expected, as patient acuity decreased over time with an improvement in their clinical status. Because of the interrelationship between pressure difference and flow, blood pressure remained steady.

Mean SVRI pre-post TD CO was consistently found to be below the normal physiological range of 1970 to 2390 dynes/sec/cm^{-5}/m^2, and over time mean SVRI continued to decrease. In assessing the determinants of SVRI, CI and RAP were not found to vary. Cardiac index was within the normal physiological range, although RAP was higher than the normal range. The elevated RAP was an expected finding within this patient cohort with preexisting cardiac dysfunction, as there may be an alteration in ventricular compliance (Beique & Ramsey, 1994; Calvin et al., 1981; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Mark, 19998; Ramsey & Tisdale, 1995; Wiedemann et al., 1984). Systolic blood pressure, DBP, and MAP changed significantly over time. Diastolic blood pressure progressively decreased, while SBP and MAP gradually increased. Since SBP and DBP are the determinants of MAP, it would be expected that an inverse relationship between SBP and DBP would lead to an increase in MAP. Therefore, this increased arterial compliance may have lead to the continuous decrease found in SVRI, since CI and RAP did not vary. The continual improvement in blood pressure and decrease in afterload was anticipated, since over time patient acuity

decreased with a concomitant clinical improvement of cardiovascular stability.

At Measurement Set 3, mean SVRI was found to be significantly lower pre TD CO compared to post TD CO. In fact the lowest mean SVRI was found at Measurement Set 3, which coincided with the lowest SBP, DBP, and MAP and highest CI. Mean arterial pressure, SBP, and DBP were also found to be significantly lower pre TD CO compared to post TD CO at this time. Upon further examination of SBP within Measurement Set 3, it was found that the first SBP recording post TD CO was statistically higher than the last SBP recording prior to TD CO. Concomitantly, the first MAP recording post TD CO was found to be significantly higher than the last MAP recording pre TD CO. Also, the first DBP post TD CO was found to be significantly higher than the last DBP recording pre TD CO. Thus, as arterial pressures were lower pre TD CO versus post TD CO, similarly SVRI would be lower pre TD CO compared to post TD CO.

Furthermore, within Measurement Set 3 were outliers for BE, $PaCO_2$, PaO_2, and hydrogen ion concentration. Therefore, local vasodilation and decreased contractility may have been present (Berne & Levy, 1992). However, the degree of vasodilation compared to contractility affects SV, and hence blood pressure. Therefore, if vasodilation is not greater than the decreased contractility, blood pressure will be maintained. Cardiac index did not decrease at Measurement Set 3, and since SBP and MAP gradually increased over time, SV was not affected by BE, $PaCO_2$, and PaO_2. An increased blood volume within the arterial system compared to the venous system may have occurred because of vasodilation and increased arterial compliance, with a concomitant decrease in preload volume. The pressure volume loop may have shifted downwards and to the left from either

the decrease in afterload or preload volume (Berne & Levy, 1992). With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres, which would increase SV, CI, arterial blood pressure, and hence, SVRI post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984).

When AWP, PEEP, and PS were controlled, mean SVRI pre TD CO became the same as TD CO at Measurement Set 3. Measured cardiac pressures do not reflect the true transmural distention pressure of a vessel, as the transducers do not measure extracardiac pressures. Extracardiac pressures which may affect transmural distention pressure are PEEP and PS. However, cardiac pressure measurements may not be affected if AWP is not transmitted to the heart and pleural space. It would be anticipated that PEEP and PS may decrease the transmural distention pressure in relation to the measured cardiac pressures (Ahrens, 1995; Ahrens, & Taylor, 1992; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Wiedemann et al., 1984). Also, PS and PEEP may increase right ventricular afterload with a concomitant decrease in SV (Diebel, et al., 1997; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Nelson, 1996; Schuster et al., 1990). Therefore, being on PEEP and PS would lead to a decrease in contractility and hence, SV, with a compensatory response of increased resistance as measured by SVRI. These subjects had low levels of AWP, PEEP, and PS which did not vary over time. Also, only the lowest recording of SVRI, which occurred at Measurement Set 3, was affected by the removal of PEEP and PS. Furthermore, PEEP and PS, when combined with drug-induced increased

arterial and venous compliance contributed to the lower mean SVRI pre TD CO compared to post TD CO at Measurement Set 3. Drug-induced vasodilation may have increased blood volume within the arterial system and venous dilation may have decreased venous return and preload volume (Berne & Levy, 1992). Decreased venous return and preload volume may have occurred because of increased intrathoracic pressure from the PEEP and PS (Ahrens, 1995; Berne & levy, 1992; Cathelyn, 1997; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Shinn, Woods, & Huseby, 1979; Wilson et al., 1996). The pressure volume loop may have shifted downwards and to the left. With the provision of TD CO fluid it would be anticipated that there would be increased stretch of myocardial fibres, which would further shift the pressure volume loop to the right and increase SV, CI, arterial pressure, and hence, SVRI post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984).

When PaO_2 at Measurement Set 1 was controlled, mean SVRI at Measurement Set 2 pre TD CO became signficantly lower compared to post TD CO. When lowering PaO_2 systemic vasodilation should occur (Berne & Levy, 1992) and combined with drug-induced increased arterial and venous compliance, SVRI pre TD CO became lower compared to post TD CO. Vasodilation may have increased blood volume within the arterial system and venodilation may have decreased venous return and preload volume (Berne & Levy, 1992). Again, the pressure volume loop may have shifted downwards and to the left from either the decrease in afterload or preload. With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres and a shift of the pressure volume loop to the right, which would increase SV, CI, arterial

pressure, and hence, SVRI post TD CO (Berne & Levy, 1992; Kiers-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). Also, at Measurement Set 2 when the effects of analgesia and sedation were removed, SVRI pre TD CO became significantly lower compared to post TD CO. Analgesics and sedatives may decrease contractility and increase arterial and venous compliance. Therefore, as the dosage of these agents decrease contractility should increase with a decrease in arterial and venous compliance. The pressure volume loop may shift to the left because of a decrease in preload volume. Also, if systemic resistance is increased the pressure volume loop may shift upwards and to the right. Therefore, the relationship between alterations in contractility and resistance will affect the pressure volume loop (Berne & Levy, 1992). With the provision of TD CO fluid myocardial fibres would be stretched, which would in turn increase SV, CI, arterial pressure, and hence SVRI post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). Finally, controlling for being on Nitroglycerine at Measurement Set 1 significantly lowered the pre TD CO SVRI compared to post TD CO at Measurement Set 2. Decreased arterial and venous compliance may have reduced SV and increased venous return with the result of an increase in preload volume. The pressure volume loop shifted upwards and to the right (Berne & Levy, 1992; Dolter, 1989; Laulive, 1982). With addition of the TD CO fluid the pressure volume loop may have shifted further upwards and to the right, increasing cardiac volume, and hence SV, arterial pressure, and SVRI post TD CO.

Volume of TD CO injectate was found to significantly affect SVRI post TD CO compared to pre TD CO at Measurement Set 4. Within Measurement Set 4 there was one

subject with whom it took 10 minutes to perform TD CO. Measurement Set 4 coincided with the lowest use of Nitroglycerine and Epinephrine and the highest recordings of SBP, DBP, and MAP. Decreased arterial and venous compliance and a shift of the pressure volume loop upwards and to the right may have occurred (Berne & Levy, 1992). With the provision of TD CO injectate, the pressure volume loop curve may have shifted further to the right with an additional increase in arterial pressure, and hence SVRI post TD CO (Berne & Levy, 1992). Also, removing the effects of a positive fluid balance at Measurement Set 2 significantly lowered SVRI pre TD CO compared to post TD CO at Measurement Set 4. This may be due to a decreased preload volume with a shift of the pressure volume loop downwards and to the left (Berne & Levy, 1992). Also, decreased total blood volume may increase peripheral resistance, which then may decrease or increase preload volume. Thermodilution CO injecate will increase the preload volume and shift the pressure volume loop upwards and to the right. Stroke volume, CO, arterial pressure, and hence SVRI would increase post TD CO. Furthermore, when removing the effects of a negative fluid balance at Measurement Set 1, mean SVRI pre TD CO became significantly lower compared to post TD CO at Measurement Set 4. This may be due to the increased preload volume and shift of the pressure volume loop upwards and to the right (Berne & Levy, 1992). Myocardial stretch would increase with a concomitant improvement in contractility (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). Also, increased total blood volume may decrease peripheral resistance. Thermodilution CO injectate will increase preload volume and shift the pressure volume loop further to the right with an increase in SV, CO, and arterial

pressure and hence SVRI post TD CO. Finally, when removing the effects of CBT at baseline, mean SVRI pre TD CO became significantly lower compared to post TD CO at Measurement Set 4. Temperature is an important determinant of afterload. Hyperthermia decreases vasoconstriction and increases ventricular compliance (Biga & Bethel, 1991; Dolter, 1989; Laulive, 1983; Meehan, 1986; Klinger, 1996; Urban, 1986). Warming of CBT when combined with drug- induced increased arterial and venous compliance at Measurement Set 4 increased SVRI pre TD CO compared to post TD CO. Drug-and temperature-induced vasodilation may have increased blood volume within the arterial system compared to the venous system (Berne & Levy, 1992). The pressure volume loop may have shifted downwards and to the left. With the provision of TD CO fluid it would be anticipated that there would be an increased stretch of the myocardial fibres, which would increase SV, arterial pressure, and hence SVRI post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984).

Pulmonary Vascular Resistance Index. The resistance to ejection of blood flow into the lungs adjusted for BSA is referred to as pulmonary vascular resistance index (PVRI). Mean pulmonary artery pressure, PAWP, and CI are the determinants of PVRI (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994), and these determinants must be examined when evaluating right ventricular afterload. Therefore, the interrelation of pressure difference across vessels and flow will determine right ventricular workload. Overall, between Measurement Sets 1 and 3, PVRI decreased by more than 10%, as well as between Measurement Sets 1 and 4. However, PVRI did not differ by greater than 10% pre-post TD CO at any of the four measurement sets. Pulmonary artery systolic pressure,

pulmonary artery diastolic pressure (PAD), MPAP, and PAWP did not vary by greater than 10% among the four measurement sets or between pre-post TD CO recordings. Cardiac index and CO increased by more than 10% between Measurement Sets 1 and 3, and between Measurement Sets 1 and 4. The concomitant improvement in CI and the reduction in right ventricular workload would be expected, as patient acuity decreased over time with an improvement in their clinical status. Because of the interrelationship between flow and pressure difference, pulmonary artery pressures remained steady.

Mean PVRI pre-post TD CO was consistently found to be within the normal physiological range yet steadily decreased over the four measurement sets. In examining the determinants of PVRI, CI, MPAP, and PAWP were not found to vary over time. Cardiac index was within the normal physiological range, although PAWP was slightly higher than the normal range. The elevation of PAWP was an expected finding within this patient cohort with preexisting cardiac dysfunction, as there may be an alteration of ventricular compliance (Beique & Ramsey, 1994; Calvin et al., 1981; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Mark, 19998; Ramsey & Tisdale, 1995; Wiedemann et al., 1984). Although MPAP and PAD did not change, over time PAS progressively increased, with PAS pre TD CO being significantly lower than post TD CO at Measurement Sets 1 and 3. However, PAS did not affect MPAP, as the latter did not vary significantly.

At Measurement Set 1, mean PVRI was found to be significantly lower pre TD CO compared to post TD CO. The highest PVRI was found at Measurement Set 1 and coincided with the lowest PAS asnd CI. Pulmonary artery systolic pressure was also found

to be significantly lower pre TD CO compared to post TD CO at this time, although PAD, MPAP and PAWP did not vary. Therefore, PAS did not affect PVRI pre-post TD CO at Measurement Set 1. Furthermore, PS and PEEP may increase right ventricular afterload, with a concomitant decrease in SV and increase in PAS (Diebel, et al., 1997; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Nelson, 1996; Schuster et al., 1990), although this was not found. These subjects had low AWP, PEEP, and PS, which did not vary over time. Drug induced vasodilation and venodilation may have increased blood volume within the arterial system and decreased venous return (Berne & Levy, 1992). Decreased venous return and preload volume may have occurred because of increased intrathoracic pressure induced by PEEP and PS (Ahrens, 1995; Berne & levy, 1992; Cathelyn, 1997; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Shinn, Woods, & Huseby, 1979; Wilson et al., 1996). The pressure volume loop may have shifted downwards and to the left. With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres, which would increase SV and hence PAS post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). Additionaly, within Measurement Set 1 were outliers for $PaCO_2$ and fluid balance. Therefore, pulmonary vasoconstriction may have been present due to hypercapnia and decrease preload volume (Berne & Levy, 1992). However, the degree of vasoconstriction to contractility will affect SV and hence pulmonary artery pressure. Therefore, if vasconstriction is not greater than contractility, pulmonary artery pressures and PVRI will be maintained. Since CI and pulmonary artery pressures were within normal ranges, fluid

balance and $PaCO_2$ did not appear to affect PVRI and PAS at this time.

Although there was no pre-post TD CO difference in PVRI at Measurement Set 3, PAS was significantly lower pre TD CO compared to post TD CO. Similar, to the rationale provided with the discussion of PVRI and PAS at Measurement Set 1, PEEP and PS may have lowered PAS pre TD CO compared to post TD CO at Measurement Set 3. Also, at Measurement Set 3 Nitroglycerine and Epinephrine were not used frequently. Both of these agents will decrease preload and afterload status because of venodilation and vasodilation. Therefore, increased preload volume may occur with decreased use of Nitroglycerine and Epinephrine. The pressure volume loop may be shifted upwards and to the right. With addition of the TD CO injectate there would be increased myocardial stretch and SV, which may increase PAS post TD CO, although not PVRI.

When controlling for oxygenation status at Measurement Set 1, mean PVRI became significantly higher pre TD CO compared to post TD CO at Measurement Set 3. As PaO_2 decreases, pulmonary vessels constrict with an increase in pulmonary resistance to maintain normal ventilation and oxygenation (Berne & Levy, 1992). Also, Measurement Set 3 coincided with decreased utilization of PEEP and PS. Positive pressure within the intrathoracic cavity may increase right ventricular afterload and pulmonary resistance (Diebel, et al., 1997; Dorinsky & Whitcomb, 1983; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Nelson, 1996; Schuster et al., 1990). Decreased PaO_2 when combined with increased pulmonary compliance because of drug induced increased arterial and venous compliance and decreased right ventricular afterload, increased PVRI pre TD CO compared to post TD CO. Vasodilation may have increased blood volume

within the arterial system and venodilation may have decreased venous return (Berne & Levy, 1992). The decreased right ventricular afterload because of decreased use of PEEP and PS may have decreased preload volume. However, as PaO_2 decreased the associated increase in pulmonary resistance shifted the pressure volume loop upwards and to the right with an increase in peak systolic presssure. With the provision of TD CO fluid preload volume would increase further shifting the pressure volume loop upwards and to the right. Stroke volume would increase and a concomitant decrease in resistance with a decrease in pulmonary pressure and hence PVRI post TD CO. Finally, when removing the effects of CBT at surgery completion, mean PVRI pre TD CO became significantly higher compared to post TD CO at Measurement Set 3. Temperature is an important determinant of afterload. Warming of CBT decreases vasoconstriction and increases ventricular compliance (Biga & Bethel, 1991; Dolter, 1989; Laulive, 1983; Meehan, 1986; Klinger, 1996; Urban, 1986). When combined with increased pulmonary compliance because of drug induced increased arterial and venous compliance, increased PVRI pre TD CO compared to post TD CO. Drug and temperature induced vasodilation may have increased blood volume within the arterial system compared to the venous system (Berne & Levy, 1992). The pressure volume loop may have shifted downwards and to the left. With the provision of TD CO fluid it would be anticipated that there would be an increased stretch of the myocardial fibres, which would increase right ventricular SV with a concomitant decrease in resistance and pulmonary pressure and hence PVRI post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984).

When controlling for the effects of oxygenation status at Measurement Set 3, mean

PVRI at Measurement Set 4 became lower pre TD CO compared to post TD CO. Decreasing PaO_2 would decrease pulmonary compliance with an increase in preload volume. The pressure volume loop shifted upwards and to the right. With the provision of TD CO fluid preload volume would increase with a further shift of the pressure volume loop to the right and an increase in pulmonary pressure and hence PVRI post TD CO. Finally, when removing the effect of CBT at baseline, mean PVRI pre TD CO became significantly lower pre TD CO compared to post TD CO at Measurement Set 4. Hyperthermia increases pulmonary and systemic compliance. Preload volume would decrease with the pressure volume loop shifted downwards and to the left (Berne & Levy, 1992). The provision of TD CO fluid increased preload volume and shifted the pressure volume loop upwards and to the right increasing pulmonary pressure and hence PVRI post TD CO.

Contractility Indicators

Contractility is the inherent ability of the myocardium to alter force and length of the myocardial fibres during systole independent of preload and afterload (Berne & Levy, 1992; Bridges & Woods, 1998; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995). Variables that affect contractility are: (a) the sympathetic nervous system, (b) positive or negative inotropic agents, (c) electrolyte and acid-base imbalances, (d) oxygen level, (e) end-diastolic volume, and (f) the degree of resistance and ventricular wall tension (Berne & Levy, 1992; Biga & Bethel, 1991; Calvin, et al., 1981; Halfmann-Franey, 1988; Kiess-Daily & Schroeder, 1994; Urban, 1986).

Stroke Volume and Stroke Volume Index. Stroke volume, the volume of blood

ejected with each heart beat is determined by CO and heart rate (HR). Stroke volume index is SV adjusted for BSA. Stroke volume and SVI are nonspecific contractility indicators (Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995; Urban, 1986). Therefore, evaluation of SV and SVI must include the assessment of HR and CO. Discussion will focus on SV and unless otherwise specified the explanation applies to SVI. Overall, between Measurement Sets 1 and 3, SV increased by more than 10%, as well as between Measurement Sets 1 and 4. However, SV did not differ by greater than 10% pre-post TD CO at any of the four measurement sets. Heart rate did not vary by greater than 10% among the four measurement sets or between pre-post TD CO recordings. Cardiac output increased by more than 10% between Measurement Sets 1 and 3, and between Measurement Sets 1 and 4. The concomitant improvement in CO and SV would be expected, since SV is determined by CO. Also, as patient acuity decreased over time with an improvement in their clinical status, the contractility indicators should improve. Because of the interrelationship between SV and CO, HR remained steady.

Mean SV pre-post TD CO was within normal physiological range and did not vary significantly over time. In assessing the determinants of SV, CO and HR also did not fluctuate significantly. Cardiac output and HR were within normal physiological range. Within Measurement Sets 1 and 3 were outliers for negative fluid balance. Negative fluid balance, with the resultant decreased preload volume, may shift the pressure volume loop downwards and to the right (Berne & Levy, 1992). Myocardial stretch would decrease with a concomitant decrease in SV and CO (Berne & Levy, 1992; Kiess-Daily &

Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). However, negative fluid balance did not affect SV, as SV did not significantly decrease at Measurement Sets 1 and 3.

When removing the effect of being on Nitroglycerine, SV pre TD CO was found to become significantly lower compared to post TD CO at Measurement Set 2. Decreased arterial and venous compliance may have lessened the blood volume within the arterial system compared to the venous system and increased venous return and preload volume. The pressure volume loop may have shifted upwards and to the right (Berne & Levy, 1992; Dolter, 1989; Laulive, 1982). With addition of the TD CO fluid, the pressure volume loop may have shifted further upwards and to the right, increasing cardiac volume, and hence SV post TD CO.

As CBT increased, SV pre TD CO became significantly higher compared to post TD CO at Measurement Set 3, which coincided with the highest recording of SV and CO. Temperature is an important determinant of afterload and contractility. Normothermia should coincide with normal peripheral vascular resistance and hence adequate myocardial contractility. Increasing CBT when combined with drug-induced increased contractility increased SV pre TD CO compared to post TD CO because of vasodilation and increased ventricular compliance and contractility (Biga & Bethel, 1991; Dolter, 1989; Laulive, 1983; Meehan, 1986; Klinger, 1996; Urban, 1986). Drug-and temperature-induced vasodilation may have decreased preload volume with a shift of the pressure volume loop downward and to the left. Also, increased contractility will present with concomitant decreased resistance and preload volume and increased blood volume in the arterial system

(Berne & Levy, 1992). With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres, which would increase SV post TD CO (Berne & Levy, 1992). However, SV decreased post TD CO, therefore, based on Frank-Starling's law the myocardial fibres must have been overstretched with a decrease in contractility and hence SV post TD CO (Berne & Levy, 1992). Finally, at Measurement Set 3, when Sa02 was controlled SVI became higher pre TD CO compared to post TD CO. Since SVI is dependent on SV and BSA, the statistically significant results do not seem plausible. Body surface area might affect oxygenation, however, it seems more appropriate to wonder if measurement error was present.

Lessening amounts of AWP, PEEP, and PS were found to lower SV pre TD CO compared to post TD CO at Measurement Set 4. It would be anticipated that a decrease in PEEP and PS would lead to an increase in contractility and hence, SV, with a compensatory response of decreased HR and SVRI. Increased venous return and preload volume may have occurred because of decreased intrathoracic pressure and resistance induced by decreased amounts of PEEP and PS (Ahrens, 1995; Berne & levy, 1992; Cathelyn, 1997; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Shinn, Woods, Huseby, 1979; Wilson et al., 1996). The pressure volume loop may have shifted to the right (Berne & Levy, 1992). With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres, which would further shift the pressure volume loop to the right with an increase in SV post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). As the beta and alpha affects of Epinephrine at Measurement Set 3 decreased,

SV pre TD CO became significantly lower compared to post TD CO at Measurement Set 4. Decreased arterial compliance will lead to decreased blood volume within the arterial system compared to the venous system. Decreased contractility would increase preload volume. The pressure volume loop may shift upwards and to the right (Berne & Levy, 1992). With the provision of TD CO injectate the pressure volume loop may shift further to the right, increasing SV post TD CO. Finally, when removing the effects of CBT at baseline and at Measurement Set 1, SV pre TD CO became significantly lower pre TD CO compared to post TD CO at Measurement Set 4. Warming of CBT increases systemic and pulmonary vasodilation with a concomitant increase in SV and decrease in preload volume. The pressure volume loop shifted downwards and to the left. With provision of TD CO fluid myocardial fibres would stretch with the pressure volume loop shifted upwards and to the right, which would increase SV post TD CO.

Left Ventricular Stroke Work Index. The amount of exertional work performed with each heart beat against aortic impendence adjusted for BSA is referred to as left ventricle stroke work index (LVSWI) (Ramsey & Tisdale, 1995). Stroke volume index, MAP and PAWP are the determinants of LVSWI, therefore, these factors must be examined upon interpretation of LVSWI results (Beique & Ramsey, 1994; Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995; Urban, 1986). It is important to realize that LVSWI incorporates preload, afterload, and contractility parameters. The LVSWI may reflect significant alterations, however, the individual parameters may not mirror the change. Also, significant alterations may occur with the individual parameters and not be reflected within LVSWI. Overall, between

Measurement Sets 1 and 3, LVSWI increased by more than 10%, as well as between Measurement Sets 1 and 4. However, LVSWI did not differ by greater than 10% pre-post TD CO at any of the four measurement sets. Stroke volume varied by greater than 10% between Measurement Sets 1 and 3 and between Measurement Sets 1 and 4. Stroke volume did not differ by greater than 10% pre-post TD CO at any of the four measurement sets. Mean arterial pressure and PAWP did not vary by greater than 10% at any of the four measurement sets. The concomitant improvement in SV and LVSWI would be expected, as patient acuity decreased over time with an improvement in myocardial function. Because of the interrelationship between the determinants of LVSWI, arterial pressure remained steady. Mean LVSWI pre-post TD CO did not significantly vary over time, although during all of the four measurement sets LVSWI was below the normal physiological range of 40 to 75 gr-m/m^2/beat. The decreased LVSWI may reflect myocardial dysfunction (Calvin et al., 1981).

Controlling for PS and PEEP was found to lower LVSWI pre TD CO compared to post TD CO at Measurement Set 1. Less PS and PEEP may have shifted the pressure volume loop upwards and to the right because of increased venous return (Ahrens, 1995; Berne & levy, 1992; Cathelyn, 1997; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Mark, 1998; Nelson, 1996; Shinn, Woods, & Huseby, 1979; Wilson et al., 1996). With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres, which would further shift the pressure volume loop to the right, which would increase SV, CI, arterial pressure, and hence, LVSWI post TD CO (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984).

Controlling for oxygenation status at Measurement Set 1 was found to significantly lower LVSWI pre TD CO compared to post TD CO at Measurement Set 2. Similar effects were found between oxygenation status and LVSWI at Measurement Set 3. As PaO_2 decreases, systemic vasodilation should occur (Berne & Levy, 1992), and when combined with drug-induced arterial and venous compliance, lowered LVSWI pre TD CO compared to post TD CO. Vasodilation may have increased blood volume within the arterial system and venodilation decreased venous return (Berne & Levy, 1992). The pressure volume loop may have shifted downwards and to the left. With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres, which would shift the pressure volume loop to the right and increase SV, CI, arterial pressure, and hence, LVSWI post TD CO (Berne & Levy, 1992; Kiers-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984).

When removing the beta and alpha effects of Epinephrine at Measurement Set 3, LVSWI was found to become significantly lower LVSWI pre TD CO compared to post TD CO. Decreased arterial compliance will lead to decreased blood volume within the arterial system. Also, decreased contractility increased preload volume. The pressure volume loop shifted upwards and to the right (Berne & Levy, 1992). With the provision of TD CO injectate the pressure volume loop may shift further upwards and to the right, increasing SV, blood pressure, and hence LVSWI post TD CO. Also, removing the effects of CBT at baseline and at Measurement Set 1 were found to significantly lower LVSWI pre TD CO compared to post TD CO at Measurement Set 3. Temperature is an important determinant of afterload and contractility. Warming of CBT increases vasodilation,

ventricular compliance and contractility (Biga & Bethel, 1991; Dolter, 1989; Laulive, 1983; Meehan, 1986; Klinger, 1996; Urban, 1986). Increased CBT when combined with drug-induced increased contractility, venodilation and vasodilation, lowered LVSWI pre TD CO compared to post TD CO. Drug and temperature induced improvment in contractility may have decreased preload volume with a shift of the pressure volume loop downwards and to the left. Also, drug and temperature mediated vasodilation and venodilation may increase blood volume in the arterial system and decrease venous return (Berne & Levy, 1992). With the provision of TD CO fluid it would be anticipated that there would be increased stretch of the myocardial fibres and a shift of the pressure volume loop upwards and to the right, which would increase SV, blood pressure, and hence LVSWI post TD CO (Berne & Levy, 1992). Finally, controlling for being on Nitroglycerine at Measurement Set 2 significantly lowered the mean LVSWI pre TD CO compared to post TD CO at Measurement Set 3. A decrease in arterial and venous compliance may have decreased SV and increased venous return and preload volume. The pressure volume loop shifted upwards and to the right (Berne & Levy, 1992; Dolter, 1989; Laulive, 1982). With addition of the TD CO fluid the pressure volume loop may have shifted further upwards and to the right, increasing cardiac volume, and hence SV and LVSWI post TD CO.

Similar to SVRI, cardiac output volume was found to significantly affect LVSWI post TD CO compared to pre TD CO at Measurement Set 4. Within Measurement Set 4 there was one subject who took 10 minutes to perform TD CO. Measurement Set 4 coincided with the lowest use of Nitroglycerine and Epinephrine and the highest

recordings of SBP, DBP, and MAP. Decreased arterial and venous compliance and a shift of the pressure volume loop upwards and to the right may have occurred (Berne & Levy, 1992). With the provision of TD CO injectate, the pressure volume loop curve may have shifted further to the right with an additional increase in SV, blood pressure, and hence LVSWI post TD CO (Berne & Levy, 1992). Finally at Measurement Set 4, controlling for a positive fluid balance significantly lowered LVSWI pre TD CO compared to post TD CO. Measurement Set 4 coincided with the highest recordings of SBP, DBP, and MAP. A decrease in positive fluid balance may have decreased preload volume with a shift of the pressure volume loop downwards and to the left (Berne & Levy, 1992). Also, decreased total blood volume may increase peripheral resistance, which then may decrease or increase preload volume. Therefore, the relationship of the vascular and cardiac function curve will dictate if preload volume is increased, decreased, or normal. Thermodilution CO injecate will increase the preload volume and shift the pressure volume loop upwards and to the right. Stroke volume, CO, arterial pressure, and hence LVSWI would increase post TD CO.

Right Ventricular Stroke Work Index. The amount of exertional work performed with each heart beat against pulmonic impendence adjusted for BSA is referred to as right ventricular stroke work index (RVSWI) (Ramsey & Tisdale, 1995). Stroke volume index, MPAP, and RAP are the determinants of RVSWI (Beique & Ramsey, 1994; Hewlett Packard, 1985, 1996a; Kiess-Daily & Schroeder, 1994; Ramsey & Tisdale, 1995; Urban, 1986). The RVSWI may reflect significant alterations, however, the individual determinants may not mirror the change. Also, significant alterations may occur with the

individual determinants and not be reflected within RVSWI. Similar to SV, SVI, CO, CI, SVRI, PVRI, and LVSWI, RVSWI increased by more than 10% between Measurement Sets 1 and 2, Measurement Sets 1 and 3, and Measurement Sets 1 and 4. However, RVSWI did not differ by greater than 10% pre-post TD CO at any of the four measurement sets. Stroke volume index increased by more than 10% between Measurement Sets 1 and 3, and between Measurement Sets 1 and 4, although SV did not vary by greater than 10% pre-post TD CO. Right atrial pressure and MPAP did not vary by greater than 10% among the four measurement sets or between pre-post TD CO recordings. The continuous improvement in SV and RVSWI would be expected, as over time patient acuity decreased and myocardial function improved. Cardiac pressures remained steady because of an improvement in contractility.

Mean digital RVSWI did not vary significantly over time or between pre-post TD CO determinations. In assessing the determinants of digital RVSWI, SVI, MPAP, and RAP were not found to vary. Stroke volume index was within normal physiological range, although digital RAP was higher than the normal range. The elevation of RAP was an expected finding within this patient cohort with preexisting cardiac dysfunction. However DAS RVSWI over time progressively increased. At Measurement Sets 1 and 4, DAS RVSWI was significantly lower pre TD CO compared to post TD CO. However, DAS determinants of RAP did not vary. The DAS RAP was slightly higher than the normal physiological range, although DAS RAP was less than digital RAP. Since, DAS RAP, MPAP, and SVI did not vary, the combination of DAS RVSWI determinants was an important factor that lead to the significant change of DAS RVSWI over time.

When removing the effects of a negative fluid balance at baseline, RVSWI pre TD CO became significantly lower compared to post TD CO at Measurement Set 1. Measurement Set 1 coincided with the lowest PAS, PAD, MPAP, SVI, and RVSWI, and highest PVRI and SVRI. An increase in fluid balance would increase preload volume and shift the pressure volume loop upwards and to the right (Berne & Levy, 1992). Myocardial stretch would increase with a concomitant improvement in contractility (Berne & Levy, 1992; Kiess-Daily & Schroeder, 1994; Klinger, 1996; Wiedemann et al., 1984). Also, increased total blood volume may decrease peripheral resistance. Thermodilution CO injectate will increase preload volume and shift the pressure volume loop further to the right. Furthermore when removing the effects of BE, RVSWI pre TD CO became significantly lower compared to post TD CO at Measurement Set 1. An increase in base excess would increase myocardial contractility. The improvement in contractility may have lead to decreased preload volume with the pressure volume loop shifted downwards and to the left. The addition of TD CO injecate may have increased preload volume and shifted the pressure volume loop to the right and upwards, which would increase SV and hence RVSWI post TD CO. Also when removing the effects of SNP, RVSWI pre TD CO became significantly lower compared to post TD CO at Measurement Set 1. Decreased compliance of the pulmonary and systemic may have occurred. The pulmonary system is a low pressure system, therefore, small changes in resistance may have affected the pulmonary vessels to a greater extent than the systemic circulation (Berne & Levy, 1992). Decreased pulmonary compliance increased right ventricular workload and decreased right ventricular contractility. Preload volume may increase, which may have shifted the

pressure volume loop upwards and the right. Also, decreased arterial compliance would decrease the blood volume within the arterial system (Berne & Levy, 1992). Thermodilution CO injectate may increase preload volume, which would further shift the pressure volume loop upwards and to the right with an increase in SV and pulmonary blood volume, and hence RVSWI post TD CO. Finally, at Measurement Set 1 when the effects of analgesia and sedation were removed, mean RVSWI pre TD CO became significantly lower compared to post TD CO. These agents may decrease contractility and increase arterial and venous compliance. Therefore, as the dosage of these agents decrease, contractility should increase, with a resultant decrease in arterial and venous compliance. Preload volume may decrease with a shift of the pressure volume loop downwards and to the left. Also, if resistance is increased the pressure volume loop may shift upwards and to the right, therefore, the relationship between alteration in contractility and resistance will affect the pressure volume loop (Berne & Levy, 1992). With addition of fluid from TD CO the pressure volume loop may shift upwards and to the right and a resultant increase in SV, pulmonary volume and hence RVSWI post TD CO.

In summary, the pressure volume loop provides an explanation of the minimal effects of the volume of TD CO injecate on hemodynamic pressures and hence derived hemodynamic parameters. Also, the pressure volume loop provides an explanation for the clinical physiological improvement found in contractility and afterload between Measurement Sets 1 and 4. The pressure volume loop is not dependent on time, per se, but reflects changes in volume and pressure of the ventricles during the cardiac cycle. Alterations in preload, afterload, or contractility can alter the shape of the pressure volume

loop. A decrease in preload volume narrows the loop, while an increase in afterload tapers and increases the height of the pressure volume loop. An increase in contractility widens and inceases the height of the pressure volume loop. Also, changes in ventricular compliance alters the pressure volume loop, as there may not be a linear relationship between pressure and volume. In this cardiac surgical sample, decreased ventricular compliance was present at Measurement Set 1 due to hypothermia, acidosis, and use of cardioplegia solution during surgery, thus narrowing the pressure volume loop. Also, immediately post cardiac surgery volume depletion was present, which decreased preload volume, further contributing to narrowing of the pressure volume loop. Over time ventricular compliance increased due to an improvement in CBT, acid-base balance, and dispersion of the cardioplegia effect. Furthermore, over time the negative fluid balance decreased, thus the pressure volume loop widened (Berne & Levy, 1992). This was supported by the clinical increase found in SV, SVI, CO, CI, LVSWI, and RVSWI between Measurement Sets 1 and 4. Also between Measurement Sets 1 and 4, SVRI and PVRI decreased. Therefore, over time cardiovascular stability may change, which in turn affects the relationship of TD CO measurements to hemodynamic pressures and, thus, derived hemodynamic parameters.

Limitations of the Study

The procedure for data collection included the analysis of hemodynamic pressures to ensure normal waveforms, as inaccurate patterns may affect the hemodynamic pressures and hence derived hemodynamic parameters (Hewlett Packard, 1982; Kern, 1993; Kiess-Daily & Schroeder, 1994). In August of 1998, it was determined that there were a large

number of abnormal hemodynamic pressure waveforms, especially pulmonary artery pressures and RAP. After troubleshooting procedures were implemented, including discussion with clinical engineering and vendors for the bedside monitor, pulmonary artery catheter, and transducer system, it was determined severe underdampening and a presystolic wave were present. The vendor for the transducer system had altered a manufacturing procedure, which appears to have caused underdampening. After discussion with clinical engineering and thesis committee members it was determined that the issue could not be resolved, therefore, the study continued. Since underdampening and a presystolic wave were present, interpretation of the results must include the consideration of measurement error.

Also, the response time of the TD CO module and the DAS may have affected the results. It is possible during the required wait time of the TD CO module and DAS, that post TD CO the pressure volume loop and vascular curve had returned to the pre TD CO measurement value. Therefore, when post TD CO hemodynamic pressures were ready to be obtained, the TD CO injectate alteration in the pressure volume loop and vascular curve would no longer have been present.

Another limitation is that baseline measurements did not commence for a mean of 109.43 ± 48.37 minutes from time of surgery. During baseline measurement TD CO and derived hemodynamic parameters were not obtained. Therefore, it is possible that during the first 2 hours upon admission to the critical care unit, the subject's pressure volume loop and vascular curve may differ, as there may be a carry over effect of hypothermia from the surgical procedure. Finally, the small sample size contributed to the lack of

statistical power. As well this study did not include unstable subjects, therefore, the results cannot be generalized beyong the present sample.

Implications of the Findings

The critical care nurse frequently makes clinical judgements based on the patient's hemodynamic pressures, which include SBP, DBP, MAP, PAS, PAD, and MPAP. Also, the hemodynamic pressures and TD CO measurements are also used for the determination of derived hemodynamic parameters, which include SV, SVI, SVRI, PVRI, LVSWI, and RVSWI. However, the critical care nurse decides which hemodynamic pressure to use for the derived hemodynamic parameters, which may not be referenced to the timing of TD CO measurements.

From the results of this study, when the cardiac surgical patient is stable with minimal alteration in hemodynamic pressures, the hemodynamic pressures values may be obtained pre or post TD CO in determining derived hemodynamic parameters. However, there are exceptions. Immediately after cardiac surgery it seems most prudent to obtain the PVRI pre TD CO compared to post TD CO. Also, whenever vasodilation is present, the SBP, DBP, MAP, and SVRI should be obtained pre TD CO compared to post TD CO. The critical care nurse must also understand the factors that affect the pulmonary and cardiovascular system. Examples include PEEP, PS, and utilization of inotropic agents. When various treatment modalities effects were controlled, the hemodynamic presssures and derived hemodynamic parameters became lower pre TD CO compared to post TD, except for SV, SVI, and PVRI at Measurement Set 3 when the hemodynamic pressures and derived hemodynamic parameters were higher pre TD CO compared to post TD CO.

The hemodynamic pressures and derived hemodynamic parameters post TD CO would not reflect a steady physiological state, as the TD CO injectate alters preload volume and hence SV. Thus, when a patient is receiving mulitple treatments in combination, they may have opposite effects on the pressure volume loop and vascular curve. Consequently, the recommendation is to obtain all hemodynamic pressures and derived hemodynamic parameters pre TD CO. This is because of the interactions found between various variables and the hemodynamic pressures and derived hemodynamic parameters. Hemodynamic pressures and derived hemodynamic parameters should reflect the steady state of a patient, which may not be represented by hemodynamic pressures and derived hemodynamic parameters obtained post TD CO.

Since this was the first reported study examining the hemodynamic pressure measurements and derived hemodynamic parameters obtained pre TD CO compared to post TD CO, there are many avenues that require further research. Cardiac surgical patients immediately after surgery require further investigation, as this patient cohort may have a different pressure volume loop and vascular curve. Also, unstable patients need to be investigated, as the patient cohort of this study were stable. Examples of unstable subjects are those with: (a) ventricular assist devices; (b) concomitant utilization of large dosages of vasopressors, venodilators, vasodilators, and inotropic agents; (c) CO less than 2 l/min or greater than 15 l/min; (d) uncontrolled dysrrhythmias; (e) PEEP greater than 10 cmH_20; (f) presence of valvular stenosis or regurgitation; and (g) presence of hypovolemia. Also, with the recent use of non cardiopulmonary bypass cardiac surgery these patients require investigation, as they may have an increased risk of myocardial

infarction with the concomitant alteration in ventricular compliance. Ventricular compliance may affect the pressure volume loop and vascular function curve. Other critically ill patient populations should be also examined, which include: (a) recent myocardial infarction; (b) distributive shock; (c) neurogenic shock; and (d) trauma. Finally, with the introduction of new catheters, such as the right ventricular ejection fraction pulmonary artery catheter and the semi-continuous pulmonary artery catheter, these catheters might have a faster response time than the intermittent TD CO catheter and require futher investigation.

Conclusion

Hemodynamic monitoring is a mainstay of critical care units, although the forms of hemodynamic monitoring may vary from institution to institution. Recently within the critical care nursing literature the increased concern has arisen tha health care professionals lack a sufficient knowledge base about hemodynamic monitoring. One aspect of hemodynamic monitoring is obtaining derived hemodynamic parameters. The derived hemodynamic parameters (SVRI, PVRI, SV, SVI, LVSWI) are determined from hemodynamic pressures and TD CO. The literature abounds with the proper "techniques" required to obtain valid and reliable obtain hemodynamic pressures and TD CO. However, there are no criteria for determing hemodynamic parameters. It is as if once the hemodynamic parameters and TD CO are obtained, the derived hemodynamic parameter must be accurate. However, when measuring TD CO fluid is provided to the heart which may alter the pressure volume loop and vascular function curve. Therefore, one must question whether hemodynamic pressures obtained before TD CO measurement are

different than those obtained post TD CO.

The subjects in this study were clincially stable as HR, blood pressure (SBP, DBP, MAP), pulmonary artery pressures (PAS, PAD, MPAP), PAWP, and RAP did not vary by greater than 10% during the data collection period. Over time, patient acuity decreased, as supported by decreased requirement for mechanical ventilation and use of vasopressors, vasodilators, venodilators, and inotropic agents. As well, a clinical signifcant increase of greater than 10% for CO, CI, LVSWI, and RVSWI occured over time as subjects recovered physiologically.

Within the first hours post cardiac surgery PVRI pre TD CO was significantly lower than post TD CO. Also, when pulmonary resistance was decreased with adequate CO and the effects of subnormal CBT and hyperoxygenation were removed PVRI pre TD CO became significantly higher at Measurement Set 3 and significantly lower compared to post TD CO at Measurement Set 4. Also, SVRI pre TD CO was found to be significantly lower compared to post TD CO approximately 6 hours post cardiac surgery. When removing the the volume of TD CO injectate, Nitroglycerine, and analgesics/sedatives, SVRI pre TD CO became significantly lower compared to post TD CO. When controlling for the effects of PEEP, PS, AWP, fluid balance, Epinephrine, SNP, Nitroglycerine, CBT, oxygenation, and volume of TD CO injectate, other derived hemodynamic parameters became statisitically significant at different measurement times. Left ventricular stroke work index and RVSWI became significantly lower pre TD CO compared to post TD CO. Stroke volume and SVI became significantly lower pre TD CO compared to post TD CO, except for Measurement Set 3 where SV and SVI became significantly higher pre TD CO

compared to post TD CO. Therefore, the derived hemodynamic parameters which incorporate hemodynamic pressures should be obtained pre TD CO when more than one treatment modality is being used.

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APPENDIX A

Inclusion and Exclusion Criteria.

PHN	code#	date	time
INCLUSION CRITERIA	MET	EXCLUSION CRITERIA	MET
> 18 years of age		ASD/VSD	
elective surgery		valvular stenosis or regurgitation	
ability for english		thoracic tumour	
PAC (int. jugular/subclavian)		ARDS	
PAC--normal waveform		MI/ischemia within last 14 days	
PAC--x-ray confirmed		mediastinal fibrosis	
PAWP <20 mm HG		chronic pulm. disease/hypertension	
PAWP >1 cc air		open sternum/VAD	
RAP via proximal port		Chest Tube loss >2 ml./kg/hr within 4 hours prior to study	
radial arterial line		dialysis	
HR <120/min.		room temperature >25 degrees [c]	
continuous pressures		shivering	
extubated or intubated\SIMV\AC\PS		core temperature <34.5 degrees [c]	
within 8 hours of surgery		PEEP >10 cmh_20	
<10% variance of pressures		airway pressure >35 cmh_20	
no ventilator, inotropes, vasodilators/pressors, antiarrthymics, or position change within 10 min		arterial saturation < 90% (hypoxia)	

PHN	code#	date	time
INCLUSION CRITERIA	MET	EXCLUSION CRITERIA	MET
		fluid restriction	
		volume intake >100 ml./hr	
		CO <2 or >15 l/min.	
		cardiac arrest within 4 hours of study	
		PAWP>PAD	
		no PAWP	
		continuous infusion via proximal port	
		uncontrolled dysrrhythmias	

Demographic Data

PHN_____ code #_____ date_____ time_____

age_____ gender_____ height (cm)_____ weight(kg)_____ BSA_____

medical history _____

_____

PAC: size_____ site_____ **Art. line**: size_____ site_____

OR: CPB time____ clamp time_____surgical procedure_____

# of minutes since OR_____ complications_____

Temperature (T/time): end of surgery_____ admission to unit_____

ECG: Baseline: time_____ rhythm_____

First Set: time_____ rhythm_____

Second Set: time_____ rhythm_____

Third Set: time_____ rhythm_____

Fourth Set: time_____ rhythm_____

Laboratory Data: time_____ HCT_____ Hgb_____ K+_____ Mg+_____

ABG's #1: time_____ PaO_2_____ $PaCO_2$____ H_____ sats._____ BE_____

Ventilator settings: time_____ FiO_2_____ PEEP_____ PS_____ SIMV\AC\PS

RR____ Exhaled TV_____ RR____ peak inspiratory airway pressure_____

ABG's #2: time_____ PaO_2_____ $PaCO_2$_____ H_____ sats._____ BE_____

Ventilator settings: time_____ FiO_2_____ PEEP_____ PS_____ SIMV\AC\PS

RR_____ Exhaled TV_____ RR____ peak inspiratory airway pressure_____

Fluid Balance: **Baseline Set:** time_____ IV intake (hr)_____

blood products (type/volume/time)_____

UO_____ (hr)

CT loss _____ (hr)

Balance +/-_____ ml.

First Set: time_____ IV intake (hr)_____

blood products (type/volume/time)_____

UO_____ (hr)

CT loss _____ (hr)

Balance +/-_____ ml.

Second Set: time_____ IV intake (hr)_____

blood products (type/volume/time)_____

UO_____ (hr)

CT loss _____ (hr)

Balance +/-_____ ml.

Third Set: time_____ IV intake (hr)_____

blood products (type/volume/time)_____

UO_____ (hr)

CT loss _____ (hr)

Balance +/-_____ ml.

Fourth Set: time_____ IV intake (hr)_____

blood products (type/volume/time)_____

UO_____ (hr)

CT loss _____ (hr)

Balance +/-_____ ml.

Inotropes: **Baseline Set:** time/drug/dosage/ml/hr _____

_____

_____

_____

First Set: time/drug/dosage/ml/hr _____

_____

_____

_____

_____

Second Set: time/drug/dosage/ml/hr _____

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Third Set: time/drug/dosage/ml/hr _____

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Fourth Set: time/drug/dosage/ml/hr _____

Vasodilators: **Baseline Set:** time/drug/dosage/ml/hr _____

First Set: time/drug/dosage/ml/hr _____

Second Set: time/drug/dosage/ml/hr _____

Third Set: time/drug/dosage/ml/hr _____

Fourth Set: time/drug/dosage/ml/hr _____

Vasopressors: **Baseline Set**: time/drug/dosage/ml/hr

First Set: time/drug/dosage/ml/hr

Second Set: time/drug/dosage/ml/hr

Third Set: time/drug/dosage/ml/hr

Fourth Set: time/drug/dosage/ml/hr

Antiarrhythimics: **Baseline Set:** time/drug/dosage/ml/hr _____

_____

_____

_____

First Set: time/drug/dosage/ml/hr _____

_____

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Second Set: time/drug/dosage/ml/hr _____

_____

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Third Set: time/drug/dosage/ml/hr _____

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_____

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Fourth Set: time/drug/dosage/ml/hr _____

_____

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_____

Analgesia/sedation: **Baseline Set**: time/drug/dosage/ml/hr _____

_____

First Set: time/drug/dosage/ml/hr _____

Second Set: time/drug/dosage/ml/hr _____

Third Set: time/drug/dosage/ml/hr _____

Fourth Set: time/drug/dosage/ml/hr _____

Analgesia provided at baseline measurement: yes/no drug_____dose_____

Misc:

Hemodynamic Pressures

Code #_____

Time								
Phase Code	B	X1	X2	X3	O	X4	X5	X6
Core Temp.								
Injectate Temp.								
CO constant								
HR								
SBP								
DBP								
MAP								
PAS	X	X	X	X	X	X	X	X
digital								
DAS								
PAD	X	X	X	X	X	X	X	X
digital								
DAS								
MPAP	X	X	X	X	X	X	X	X
digital								
DAS								
PAWP	X	X	X	X	X	X	X	X
digital								
DAS								
RAP	X	X	X	X	X	X	X	X
digital								
DAS								

CODE #								
Time								
Phase Code	B	X1	X2	X3	0	X4	X5	X6
CO	X	X	X	X	X	X	X	X
1.								
2.								
3.								
4.								
5.								
6.								
7.								
Volume of injectate								
Time to perform CO								
Mean CO electronic								
Manual								
CI electronic								
manual								

DAS Baseline Data

Pre DAS baseline data mean (mmHg)	
Post DAS baseline data mean (mmHg)	
Pre - Post baseline difference (mmHg)	
Offset mean value (mmHg)	

Mean Hemodynamic Pressure Measurements and Heart Rate

	X1, X2, X3	X4, X5, X6		X1, X2, X3	X4, X5, X6
HR			PAD DAS		
SBP			MPAP digital		
DBP			MPAP DAS		
MAP			PAWP digital		
PAS digital			PAWP DAS		
PAS DAS			RAP digital		
PAD digital			RAP DAS		

Derived Hemodynamic Parameters

code #_____

Time								
Phase Code	X1	X2	X3	MEAN	X4	X5	X6	MEAN
SV	X	X	X	X	X	X	X	X
electronic								
manual								
SVI	X	X	X	X	X	X	X	X
electronic								
manual								
SVRI	X	X	X	X	X	X	X	X
electronic digital								
DAS								
manual digital								
DAS								
PVRI	X	X	X	X	X	X	X	X
electronic digital								
DAS								
manual digital								
DAS								

Code #								
Time								
Phase Code	X1	X2	X3	MEAN	X4	X5	X6	MEAN
LVSWI	X	X	X	X	X	X	X	X
electronic digital								
DAS								
manual digital								
DAS								
RVSWI	X	X	X	X	X	X	X	X
electronic digital								
DAS								
manual digital								
DAS								

APPENDIX B

Consent Form

PROJECT TITLE: Timing of Hemodynamic Pressure Measurements and Thermodilutional Cardiac Output on Derived Hemodynamic Parameters.

INVESTIGATORS:

Gayle Urquhart	Dr. Louise Jensen
M.N. Candidate	Thesis Supervisor
Faculty of Nursing	Faculty of Nursing
Phone: 492-6846	492-6795

BACKGROUND: It is important after heart surgery to measure how much blood your heart pumps and the blood pressures of your heart. This is normally done by using small catheters. One is put into a vein in your neck and placed into your heart during surgery. Another small catheter is placed in the artery in your wrist. Measurements are normally taken every four hours while you are in the intensive care unit after surgery.

PURPOSE: The purpose of this study is to examine if the time when nurses measure the pressures of the heart affects their computed values of heart and blood pressure functioning.

PROCEDURE: The study will take place in the intensive care unit on the day of your surgery. The researcher will measure the blood pressures of your heart and how much blood your heart pumps. Your participation in this study involves having one more set of readings taken than you would normally have after surgery.

1. First, you will be placed flat in your bed and the blood pressures of your heart will be recorded from the bedside monitor.
2. Next, the researcher will measure the amount of blood your heart pumps in the same way that it would normally be measured after your surgery using the catheter in your neck. This is called cardiac output.
3. Next, there will be another recording of the blood pressure of your heart from your bedside monitor.This is how the blood pressures of your heart are measured after surgery.
4. Thirty minutes later, the same recordings will be repeated. This set of recordings is different than from your normal care.
5. Four hours later, the recordings of the blood pressure of your heart and cardiac output will be repeated. The recorded blood pressures of the heart and cardiac output will be used to determine the work of your heart and blood vessels.

PARTICIPATION: There will be no harm or benefits for being in this study. Results from

this study may help nurses to understand when to measure the blood pressures of the heart. This may help to improve the care that nurses give to patients. You do not have to be in this study if you do not wish to be. You can drop out of the study at any time by notifying the researcher. Taking part in this study or dropping out will not affect your care after surgery. Your name will not appear in this study. Only a code number will be used on the recordings. All records will be kept in a locked cabinet. Data may be used for another study in the future, if the researcher receives further ethical approval. The findings of this study may be published or presented at conferences, however, your name or any material than may identify you will not be used.
If you have any questions about the study at any time, you can call the researchers at the numbers above. If you have concerns about any aspect of this study, you can contact the patient representative of the Capital Health Authority at 492-6961, who has no association with the researchers.

CONSENT: I have had the procedures for this study explained to me. I am satisfied with the answers to my questions. I understand that I may contact the persons named above, if I have further questions either now or in the future. I understand that there are no risks or immediate benefits from taking part in this study. I have been assured that my records relating to this study will be kept confidential. I understand that I am free to drop out of the study at any time. My nursing care will not be affected. I understand that if any information becomes available that could influence my decision to continue in this study, I will be informed promptly. I have been given a copy of this form to keep.

_____ _____

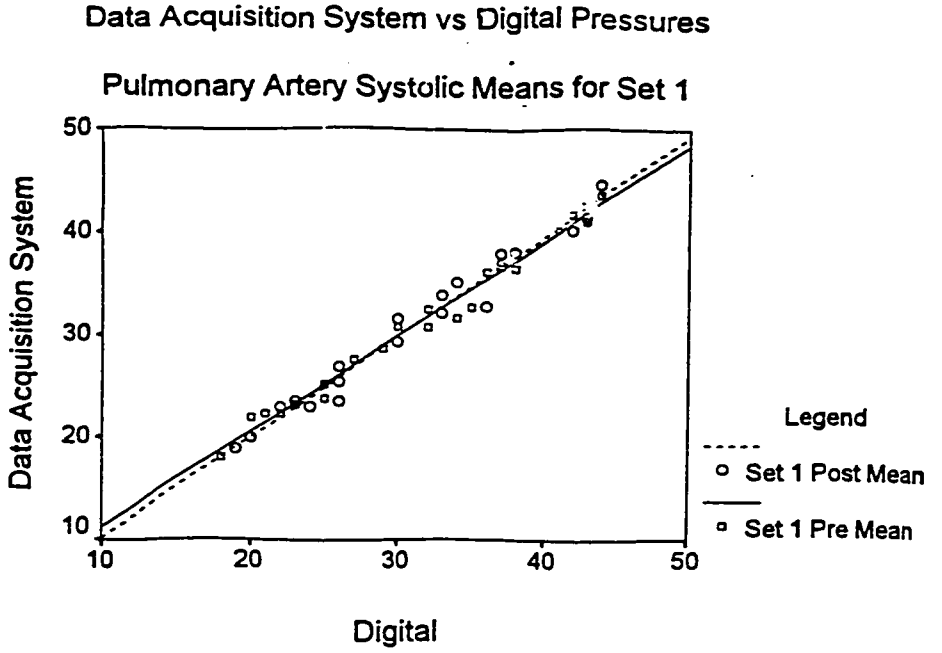
Signature of Participant Date

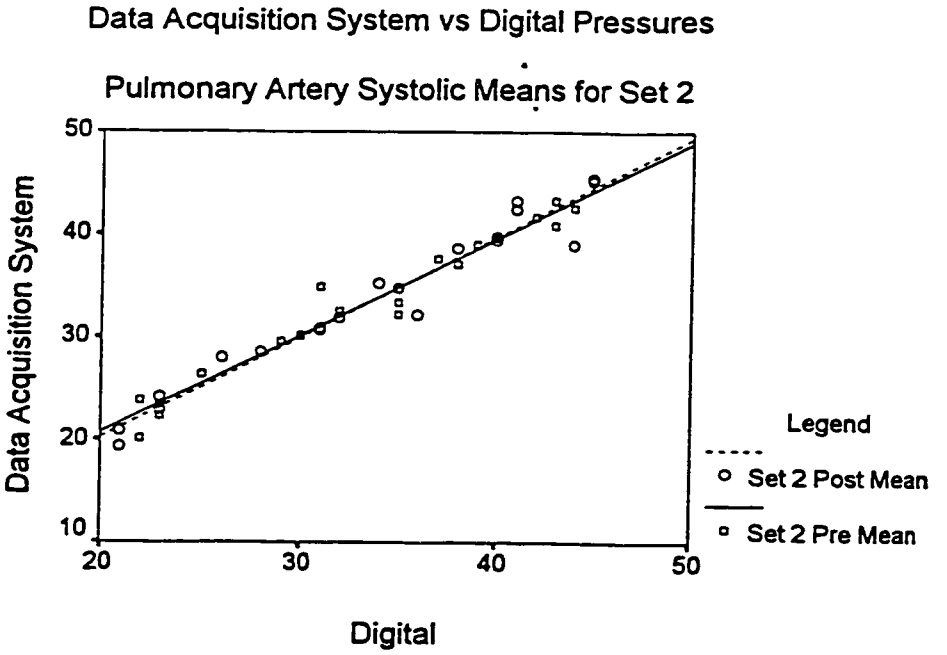
_____ _____

Signature of Researcher Date

APPENDIX C

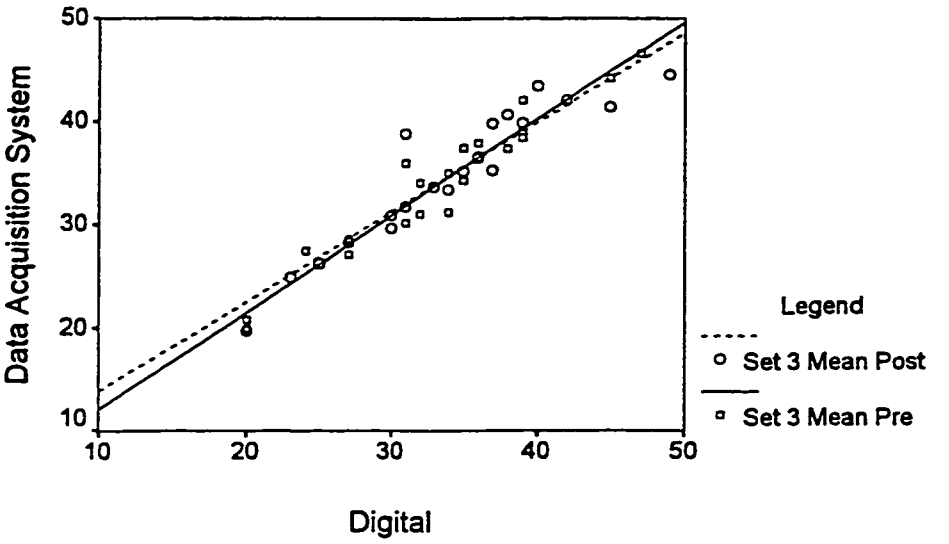
Correlations Between Data Acqusition System and Digital Hemodynamic Pressures





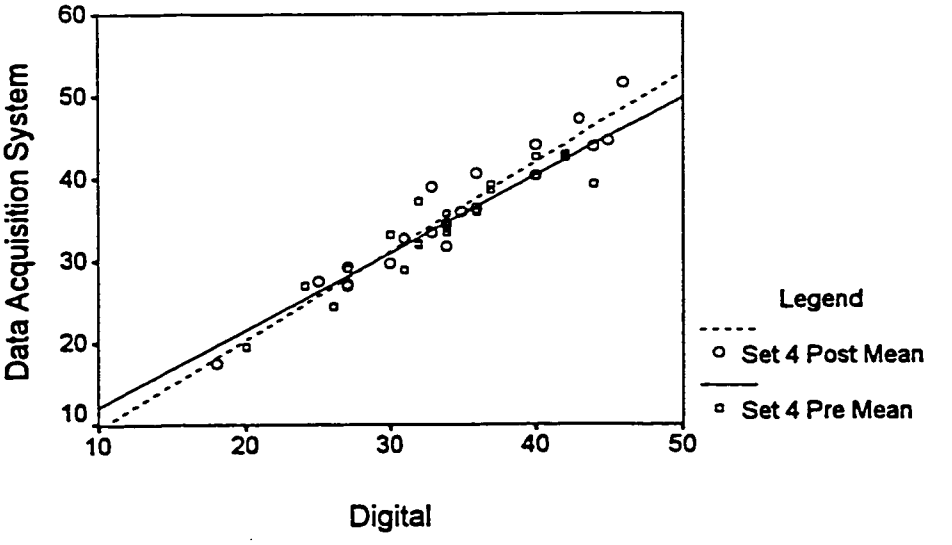
Data Acquisition System vs Digital Pressures

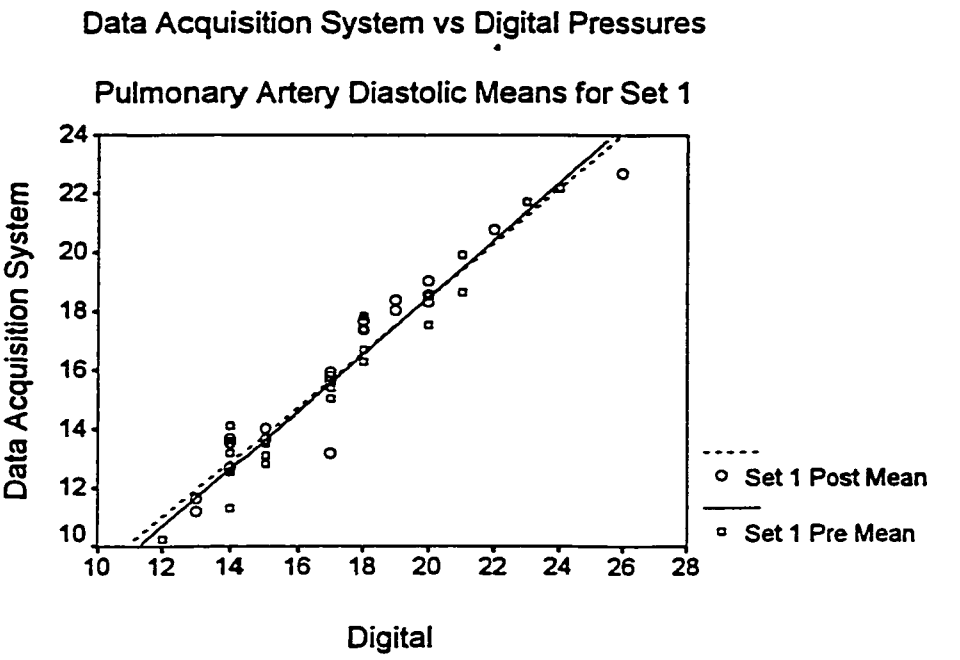
Pulmonary Artery Systolic Means for Set 3

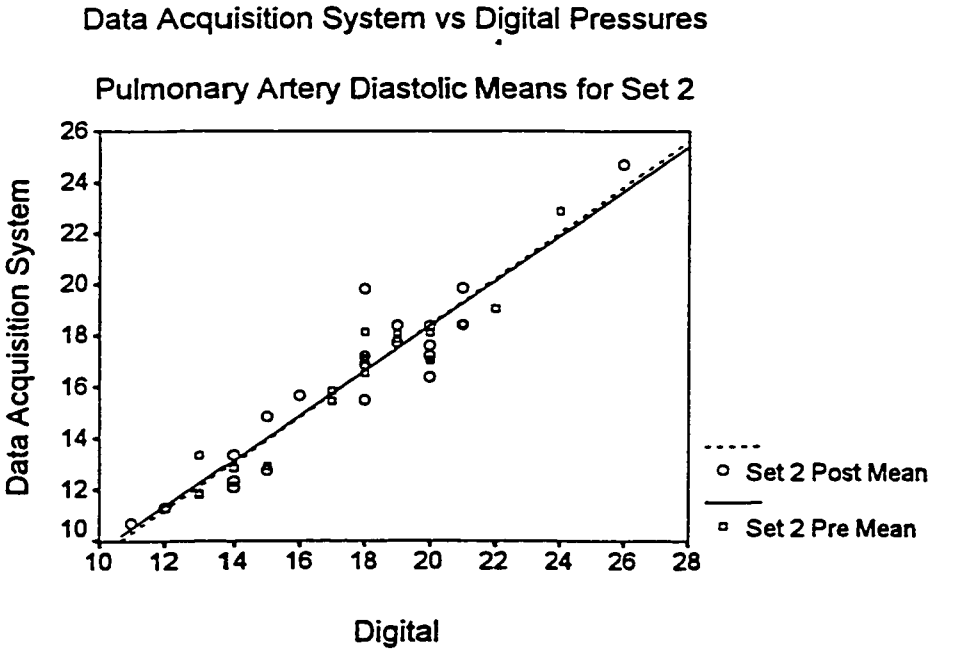


Data Acquisition System vs Digital Pressures

Pulmonary Artery Systolic Means for Set 4

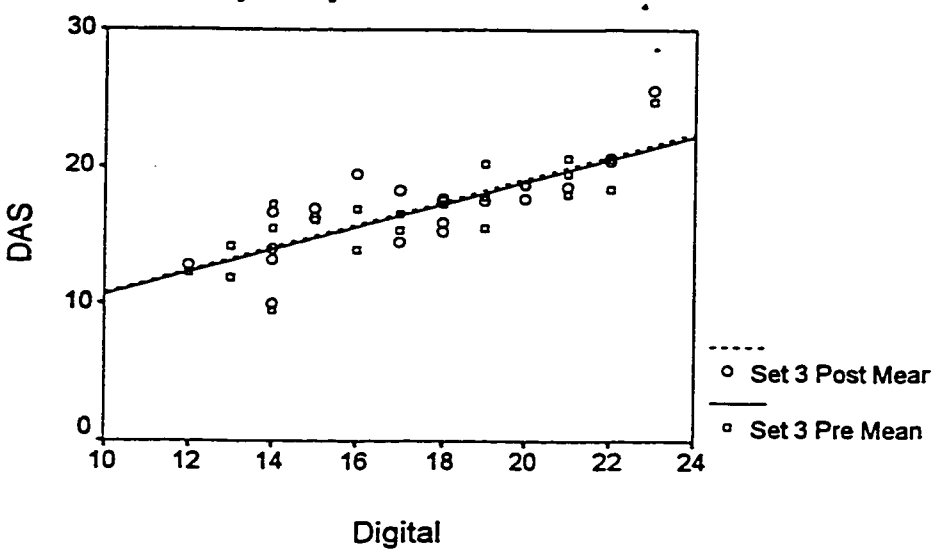



Data Acquisition System vs Digital Pressures
Pulmonary Artery Diastolic Means for Set 1
Data Acquisition System
24
22
20
18
16
14
12
10
10 12 14 16 18 20 22 24 26 28
Digital
Set 1 Post Mean
Set 1 Pre Mean


Data Acquisition System vs Digital Pressures
Pulmonary Artery Diastolic Means for Set 2
Data Acquisition System
26
24
22
20
18
16
14
12
10
10 12 14 16 18 20 22 24 26 28
Digital
Set 2 Post Mean
Set 2 Pre Mean

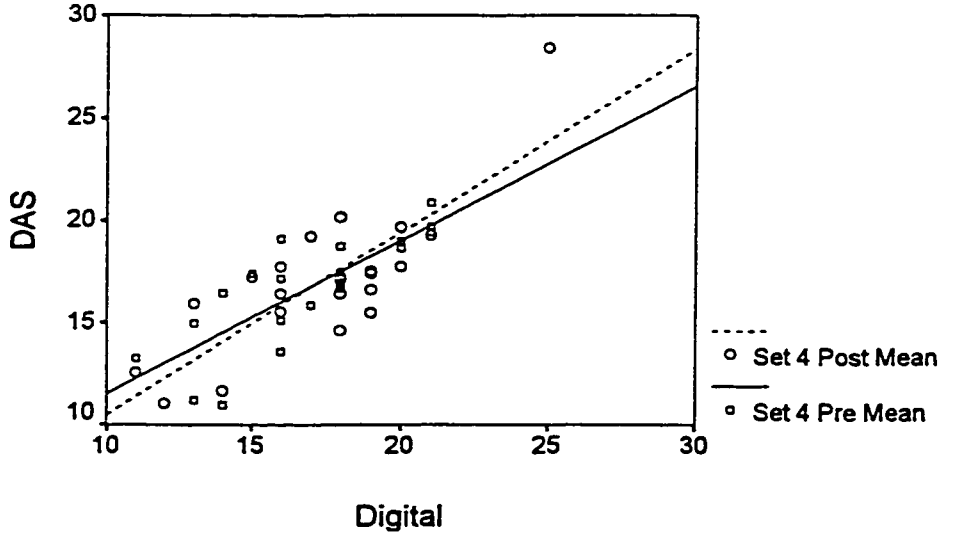
Data Acquisition System vs Digital Pressures

Pulmonary Artery Diastolic Means for Set 3



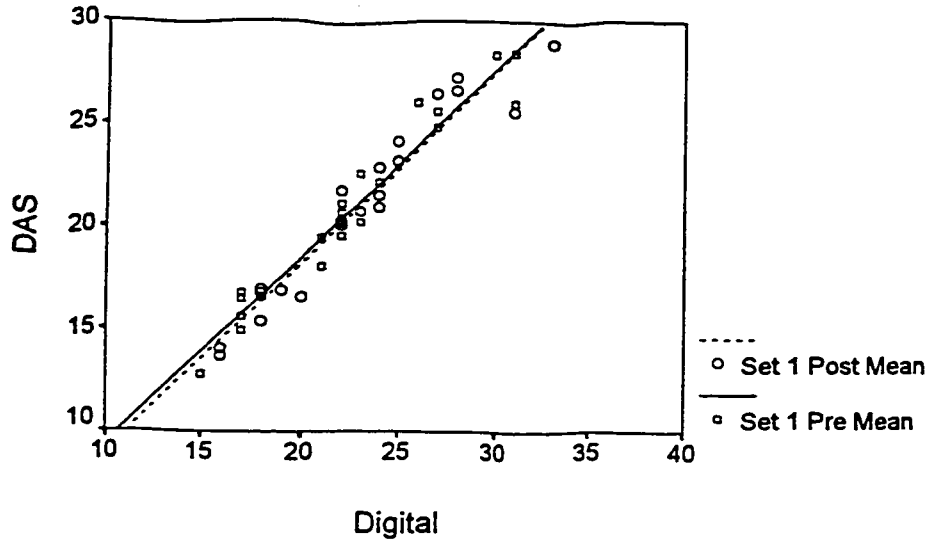
Data Acquisition System vs Digital Pressures

Pulmonary Artery Diastolic Means for Set 4



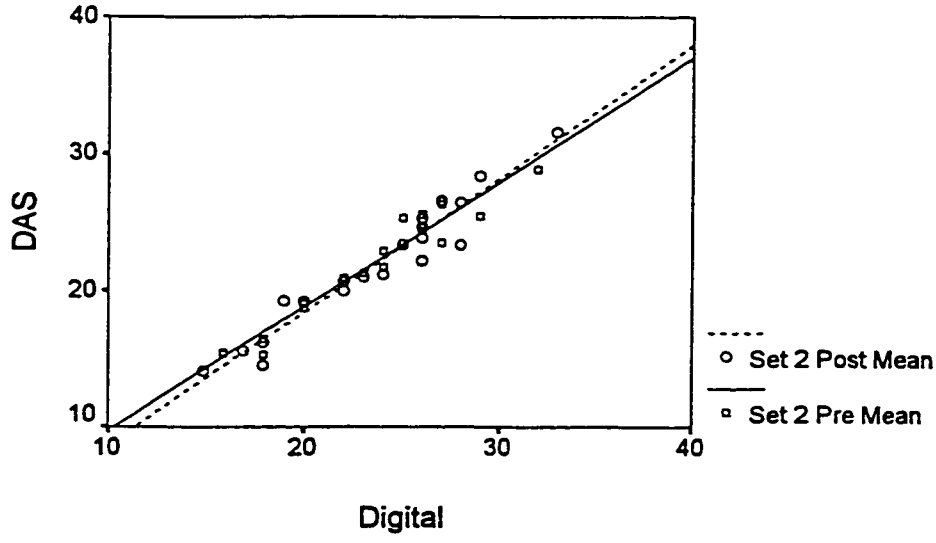
Data Acquisition System vs Digital Pressures

Mean Pulmonary Artery Pressure Means For Set 1



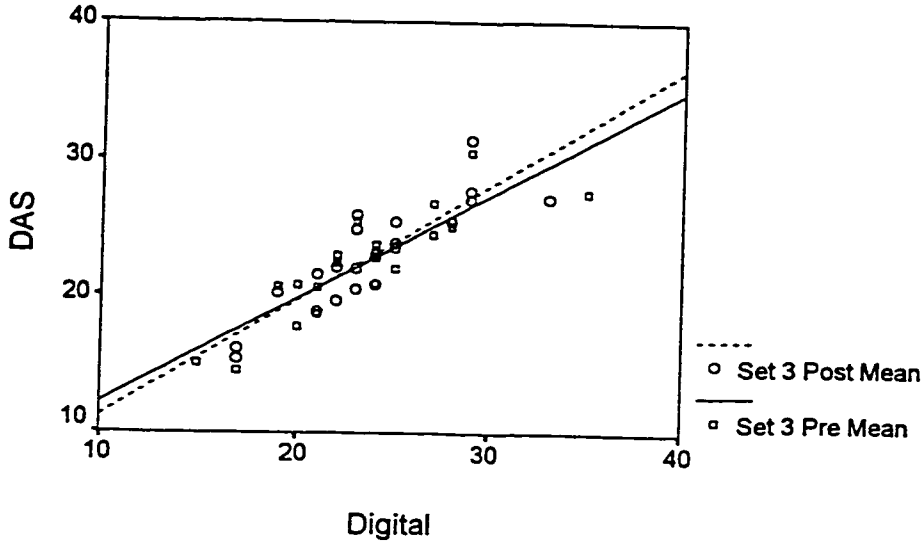
Data Acquisition System vs Digital Pressures

Mean Pulmonary Artery Pressure Means For Set 2



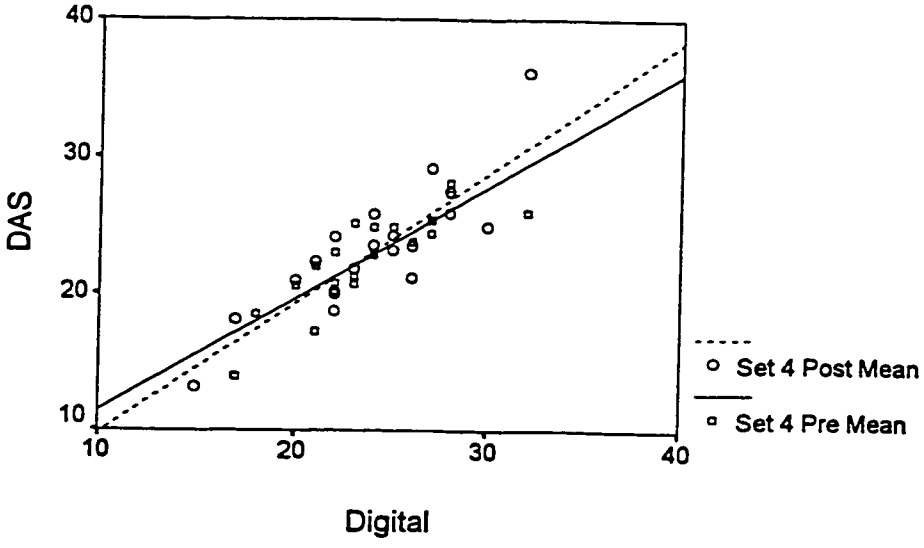
Data Acquisition System vs Digital Pressures

Mean Pulmonary Artery Pressure Means For Set 3



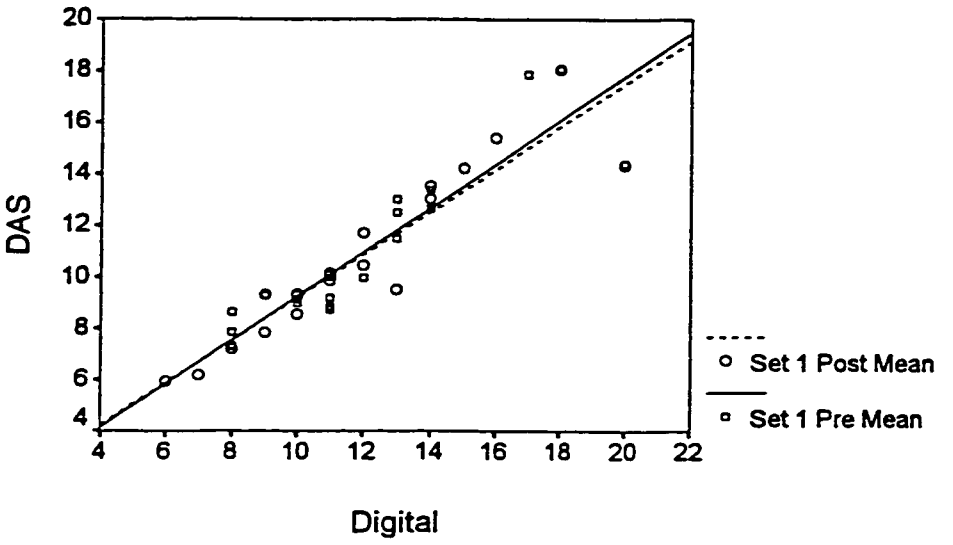
Data Acquisition System vs Digital Pressures

Mean Pulmonary Artery Pressure Means For Set 4



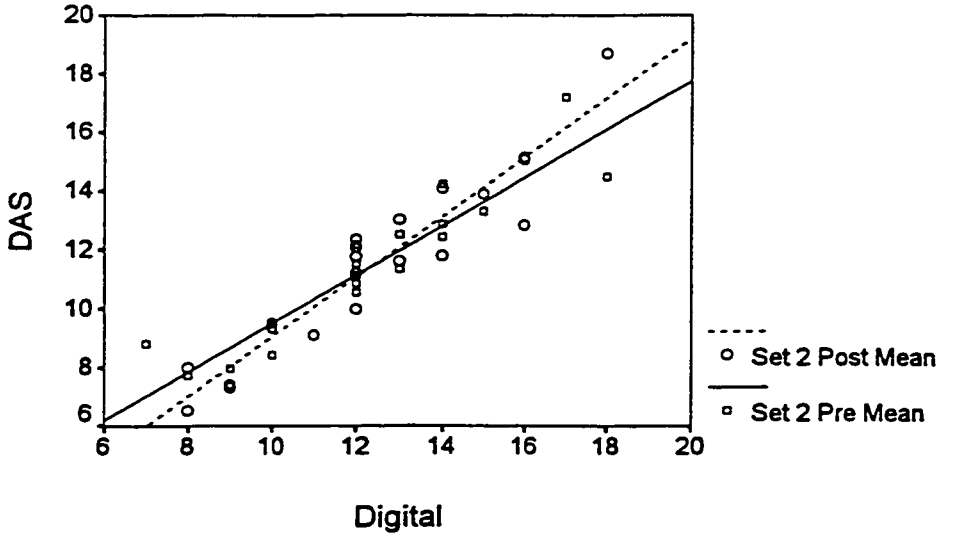
Data Acquisition System vs Digital Pressures

Pulmonary Artery Wedge Pressure Means for Set 1



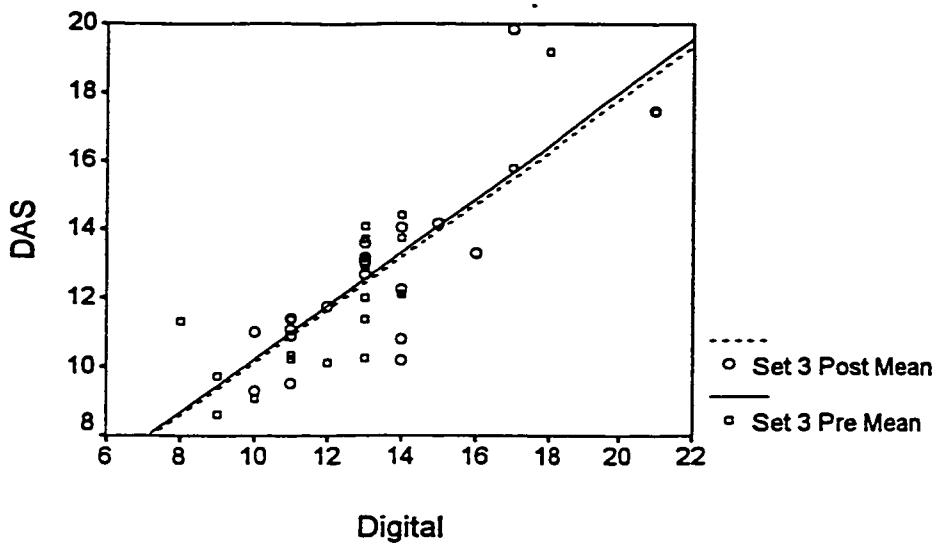
Data Acquisition System vs Digital Pressures

Pulmonary Artery Wedge Pressure Means for Set 2



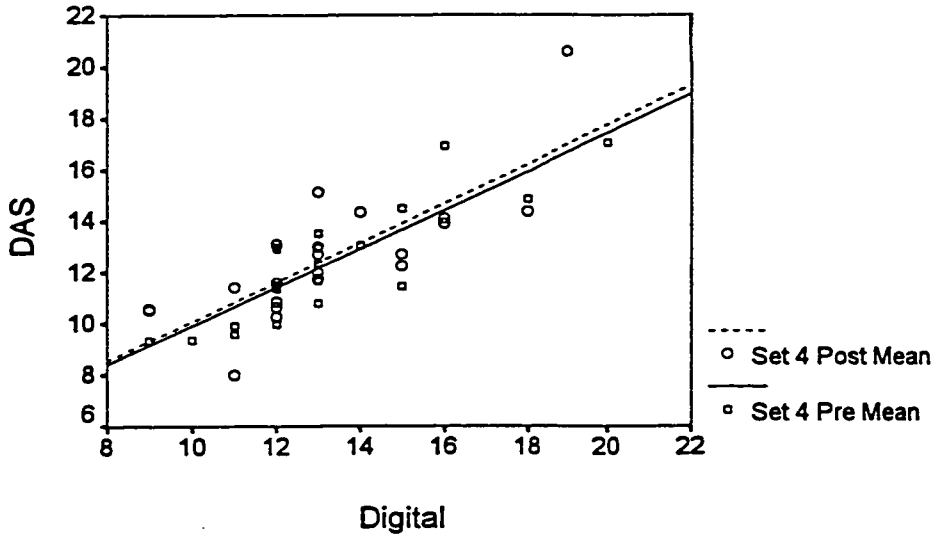
Data Acquisition System vs Digital Pressures

Pulmonary Artery Wedge Pressure Means for Set 3



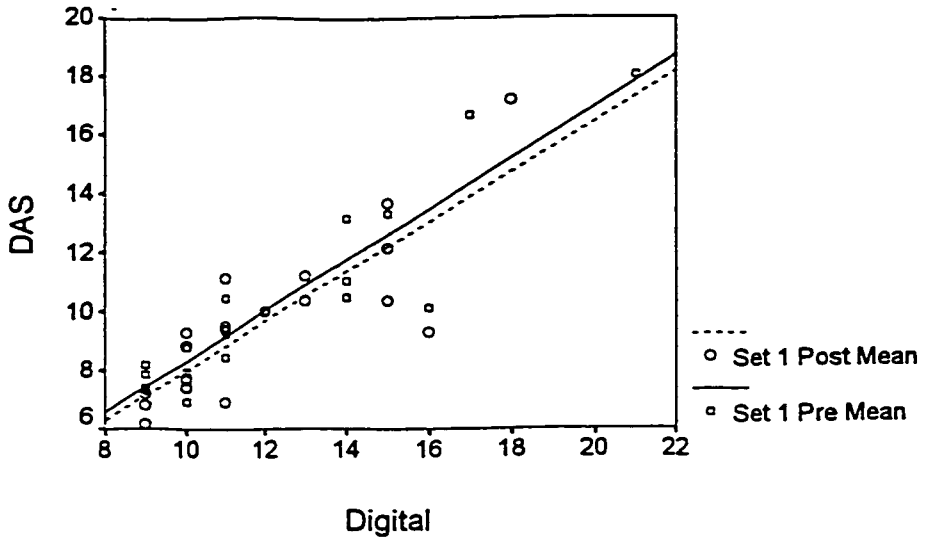
Data Acquisition System vs Digital Pressures

Pulmonary Artery Wedge Pressure Means for Set 4



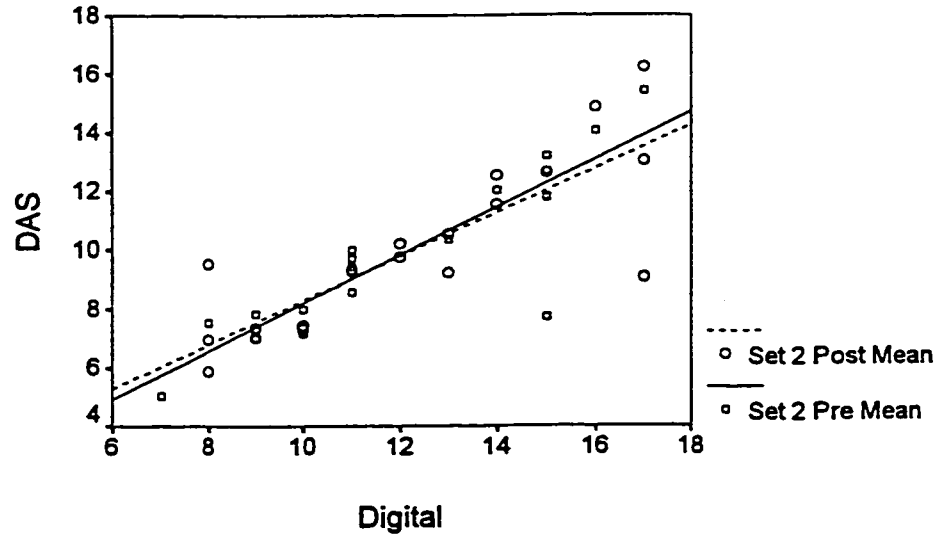
Data Acquisition System vs Digital Pressures

Right Atrial Pressure Means for Set 1



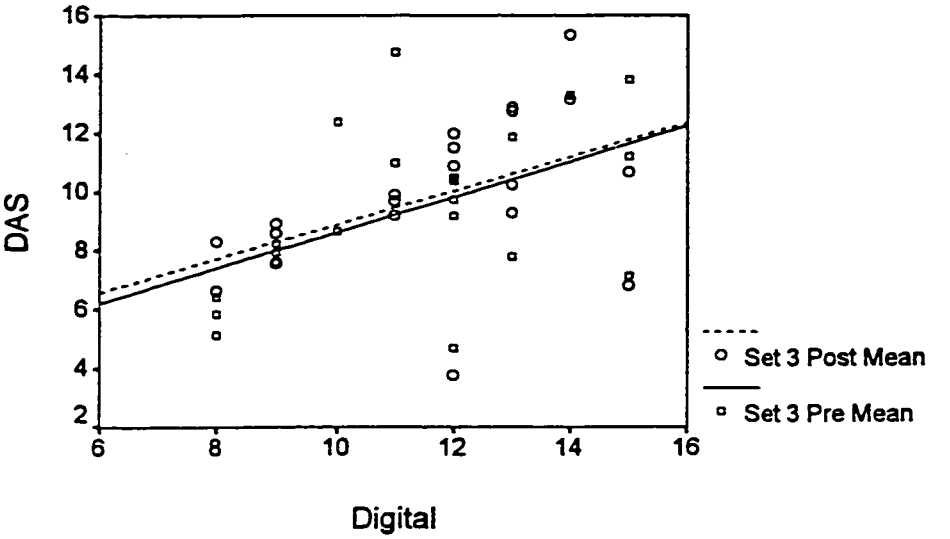
Data Acquisition System vs Digital Pressures

Right Atrial Pressure Means for Set 2



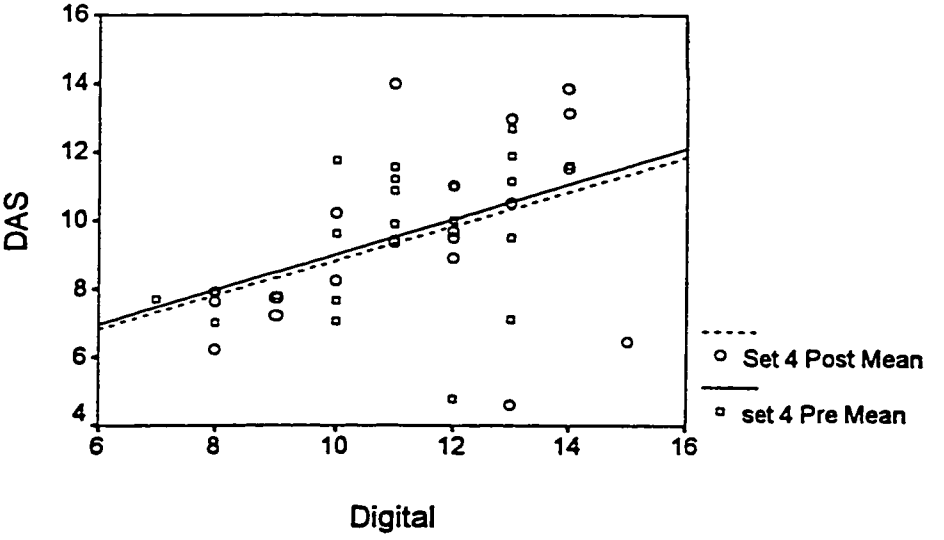
Data Acquisition System vs Digital Pressures

Right Atrial Pressure Means for Set 3



Data Acquisition System vs Digital Pressures

Right Atrial Pressure Means for Set 4



APPENDIX D

ANCOVA Results

Analysis of Covariance of Mean Stroke Volume

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Partial Pressure of Oxygen at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.11	.11	1/18	.17	.69
Measurement Set 2	71.58	21.92	71.94	22.32	.51	.51	1/18	.56	.46
Measurement Set 3	77.40	23.41	76.86	22.94	.00	.00	1/18	.00	.99
Measurement Set 4	75.27	22.09	75.57	22.17	.03	.03	1/18	.02	.90
Partial Pressure Oxygen at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	3.97	3.97	1/18	1.21	.29
Measurement Set 4	75.27	22.09	75.57	22.17	2.20	2.20	1/18	1.24	.28
Oxygen Saturation at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.90	.90	1/18	1.71	.21
Measurement Set 2	71.58	21.92	71.94	22.32	.04	.04	1/18	.04	.85
Measurement Set 3	77.40	23.41	76.86	22.94	1.59	1.59	1/18	.56	.46
Measurement Set 4	75.27	22.09	75.57	22.17	1.46	1.46	1/18	.88	.36
Oxygen Saturation at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	11.93	11.93	1/18	4.19	.06
Measurement Set 4	75.27	22.09	75.57	22.17	1.11	1.11	1/18	.66	.43
Base Excess at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.05	.05	1/18	.07	.80
Measurement Set 2	71.58	21.92	71.94	22.32	.00	.00	1/18	.00	.97
Measurement Set 3	77.40	23.41	76.86	22.94	1.17	1.17	1/18	.35	.56
Measurement Set 4	75.27	22.09	75.57	22.17	1.37	1.37	1/18	.79	.39
Base Excess at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	.16	.16	1/18	.05	.83
Measurement Set 4	75.27	22.09	75.57	22.17	2.66	2.66	1/18	1.52	.23
Airway Pressure at Measurement Set 1									
Measurement Set 1	60.20	16.71	60.41	16.78	.00	.00	1/10	.02	.91
Measurement Set 2	66.42	23.80	66.62	24.18	.16	.16	1/10	.23	.64
Measurement Set 3	75.59	27.19	75.75	27.47	.73	.73	1/10	.63	.45
Measurement Set 4	73.93	27.18	73.93	27.03	.94	.94	1/10	4.77	.05*
Airway Pressure at Measurement Set 3									
Measurement Set 3	75.59	27.19	75.75	27.47	3.69	3.69	1/10	3.19	.10
Measurement Set 4	73.93	27.18	73.93	27.03	.04	.04	1/10	.20	.67

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Stroke Volume

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Cardiopulmonary Bypass Time									
Measurement Set 1	67.18	16.67	67.38	16.91	.04	.04	1/18	.05	.82
Measurement Set 2	73.00	21.48	73.38	21.88	2.37	2.37	1/18	2.96	.10
Measurement Set 3	78.07	23.81	77.48	23.36	.14	.14	1/18	.04	.84
Measurement Set 4	76.24	22.20	76.53	22.30	3.53	3.53	1/18	2.06	.17
Aortic Cross Clamp Time									
Measurement Set 1	67.18	16.67	67.38	16.91	.16	.16	1/18	.21	.65
Measurement Set 2	73.00	21.48	73.38	21.88	.96	.96	1/18	1.09	.31
Measurement Set 3	78.07	23.81	77.48	23.36	.06	.06	1/18	.02	.90
Measurement Set 4	76.24	22.20	76.53	22.30	3.81	3.81	1/18	2.25	.15
Core Temperature End of Surgery									
Measurement Set 1	66.23	16.81	66.42	17.05	.03	.03	1/13	.04	.84
Measurement Set 2	71.58	21.92	71.94	22.32	.04	.04	1/13	.04	.85
Measurement Set 3	77.40	23.41	76.86	22.94	.98	.98	1/13	.54	.49
Measurement Set 4	75.27	22.09	75.57	22.17	.51	.51	1/13	.43	.53
Core Temperature Upon Admission									
Measurement Set 1	66.23	16.81	66.42	17.05	.00	.00	1/13	.00	.96
Measurement Set 2	71.58	21.92	71.94	22.32	.02	.02	1/13	.02	.89
Measurement Set 3	77.40	23.41	76.86	22.94	1.72	1.72	1/13	.94	.35
Measurement Set 4	75.27	22.09	75.57	22.17	.57	.57	1/13	.48	.50
Core Temperature At Baseline									
Measurement Set 1	66.23	16.81	66.42	17.05	.27	.27	1/13	.34	.57
Measurement Set 2	71.58	21.92	71.94	22.32	.18	.18	1/13	.16	.70
Measurement Set 3	77.40	23.41	76.86	22.94	3.44	3.44	1/13	1.89	.19
Measurement Set 4	75.27	22.09	75.57	22.17	15.77	15.77	1/13	13.17	.00*
Core Temperature at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	2.29	2.29	1/13	2.88	.11
Measurement Set 2	71.58	21.92	71.94	22.32	.70	.70	1/13	.62	.45
Measurement Set 3	77.40	23.41	76.86	22.94	31.02	31.02	1/13	16.97	.00*
Measurement Set 4	75.27	22.09	75.57	22.17	10.32	10.32	1/13	8.61	.01*
Core Temperature at Measurement Set 2									
Measurement Set 2	71.58	21.92	71.94	22.32	1.53	1.53	1/13	1.36	.27
Measurement Set 3	77.40	23.41	76.86	22.94	30.78	30.78	1/13	16.84	.00*
Measurement Set 4	75.27	22.09	75.57	22.17	4.39	4.39	1/13	3.77	.08
Core Temperature at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	1.60	1.60	1/13	.87	.37
Measurement Set 4	75.27	22.09	75.57	22.17	.49	.49	1/13	.41	.53
Core Temperatuare at Measurement Set 4									
Measurement Set 4	75.27	22.09	75.57	22.17	3.41	3.41	1/13	2.84	.12

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Stroke Volume

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Volume of Cardiac Output Injectate at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	1.43	1.43	1/16	1.97	.18
Measurement Set 2	71.58	21.92	71.94	22.32	.02	.02	1/16	.03	.87
Measurement Set 3	77.40	23.41	76.86	22.94	.00	.00	1/16	.00	.97
Measurement Set 4	75.27	22.09	75.57	22.17	4.12	4.12	1/16	2.40	.14
Volume of Cardiac Output Injectate at Measurement Set 2									
Measurement Set 2	71.58	21.92	71.94	22.32	.50	.50	1/16	.60	.45
Measurement Set 3	77.40	23.41	76.86	22.94	2.79	2.79	1/16	1.48	.24
Measurement Set 4	75.27	22.09	75.57	22.17	6.35	6.35	1/16	3.70	.07
Volume of Cardiac Output Injectate at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	6.15	6.15	1/16	3.26	.09
Measurement Set 4	75.27	22.09	75.57	22.17	.31	.31	1/16	.18	.68
Volume of Cardiac Output Injectate at Measurement Set 4									
Measurement Set 4	75.27	22.09	75.57	22.17	.94	.94	1/16	.55	.47
Fluid Balance at Baseline									
Measurement Set 1	66.23	16.81	66.42	17.05	.50	.50	1/15	.60	.45
Measurement Set 2	71.58	21.92	71.94	22.32	.62	.62	1/15	.62	.45
Measurement Set 3	77.40	23.41	76.86	22.94	3.73	3.73	1/15	1.01	.33
Measurement Set 4	75.27	22.09	75.57	22.17	.39	.39	1/15	.18	.68
Fluid Balance at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.09	.09	1/15	.10	.75
Measurement Set 2	71.58	21.92	71.94	22.32	.12	.12	1/15	.12	.73
Measurement Set 3	77.40	23.41	76.86	22.94	1.37	1.37	1/15	.37	.55
Measurement Set 4	75.27	22.09	75.57	22.17	.39	.39	1/15	.18	.68
Fluid Balance at Measurement Set 2									
Measurement Set 2	71.58	21.92	71.94	22.32	1.31	1.31	1/15	1.32	.27
Measurement Set 3	77.40	23.41	76.86	22.94	.99	.99	1/15	.27	.61
Measurement Set 4	75.27	22.09	75.57	22.17	1.27	1.27	1/15	.60	.45
Fluid Balance at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	.39	.39	1/15	.11	.75
Measurement Set 4	75.27	22.09	75.57	22.17	.67	.67	1/15	.32	.58
Fluid Balance at Measurement Set 4									
Measurement Set 4	75.27	22.09	75.57	22.17	.43	.43	1/15	.20	.66

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. $*p \leq .05$.

Analysis of Covariance of Mean Stroke Volume

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Sodium Nitroprusside at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.49	.49	1/17	.68	.42
Measurement Set 2	71.58	21.92	71.94	22.32	.35	.35	1/17	.39	.54
Measurement Set 3	77.40	23.41	76.86	22.94	.31	.31	1/17	.09	.77
Measurement Set 4	75.27	22.09	75.57	22.17	4.22	4.22	1/17	3.02	.10
Sodium Nitroprusside at Measurement Set 2									
Measurement Set 2	71.58	21.92	71.94	22.32	.00	.00	1/17	.00	.99
Measurement Set 3	77.40	23.41	76.86	22.94	.10	.10	1/17	.03	.87
Measurement Set 4	75.27	22.09	75.57	22.17	2.47	2.47	1/17	1.76	.20
Nitroglycerine at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.81	.81	1/17	1.03	.32
Measurement Set 2	71.58	21.92	71.94	22.32	2.41	2.41	1/17	3.54	.08
Measurement Set 3	77.40	23.41	76.86	22.94	.02	.02	1/17	.01	.94
Measurement Set 4	75.27	22.09	75.57	22.17	1.71	1.71	1/17	.91	.35
Nitroglycerine at Measurement Set 2									
Measurement Set 2	71.58	21.92	71.94	22.32	4.73	4.73	1/17	6.97	.02*
Measurement Set 3	77.40	23.41	76.86	22.94	10.01	10.01	1/17	3.56	.08
Measurement Set 4	75.27	22.09	75.57	22.17	.22	.22	1/17	.12	.74
Epinephrine at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.00	.00	1/16	.00	.96
Measurement Set 2	71.58	21.92	71.94	22.32	.01	.01	1/16	.01	.92
Measurement Set 3	77.40	23.41	76.86	22.94	1.50	1.50	1/16	.41	.53
Measurement Set 4	75.27	22.09	75.57	22.17	1.10	1.10	1/16	.92	.35
Epinephrine at Measurement Set 2									
Measurement Set 2	71.58	21.92	71.94	22.32	.04	.04	1/16	.04	.84
Measurement Set 3	77.40	23.41	76.86	22.94	.07	.07	1/16	.02	.89
Measurement Set 4	75.27	22.09	75.57	22.17	2.92	2.92	1/16	2.47	.14
Epinephrine at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	.62	.62	1/16	.17	.69
Measurement Set 4	75.27	22.09	75.57	22.17	6.07	6.07	1/16	5.14	.04*
Epinephrine at Measurement Set 4									
Measurement Set 4	75.27	22.09	75.57	22.17	.14	.14	1/16	.12	.74
Demerol, Morphine, and Diprivan at Measurement Set 1									
Measurement Set 1	66.23	16.81	66.42	17.05	.01	.01	1/16	.01	.93
Measurement Set 2	71.58	21.92	71.94	22.32	1.22	1.22	1/16	1.37	.26
Measurement Set 3	77.40	23.41	76.86	22.94	8.83	8.83	1/16	2.78	.12
Measurement Set 4	75.27	22.09	75.57	22.17	.05	.05	1/16	.02	.89
Demerol, Morphine, and Diprivan at Measurement Set 2									
Measurement Set 2	71.58	21.92	71.94	22.32	1.42	1.42	1/16	1.59	.23
Measurement Set 3	77.40	23.41	76.86	22.94	4.31	4.31	1/16	1.36	.26
Measurement Set 4	75.27	22.09	75.57	22.17	.15	.15	1/16	.08	.79
Demerol, Morphine, and Diprivan at Measurement Set 3									
Measurement Set 3	77.40	23.41	76.86	22.94	.58	.58	1/16	.18	.68
Measurement Set 4	75.27	22.09	75.57	22.17	.50	.50	1/16	.24	.63
Demerol, Morphine, and Diprivan at Measurement Set 4									
Measurement Set 4	75.27	22.09	75.57	22.17	.32	.32	1/16	.16	.70

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. * $p \leq .05$.

Analysis of Covariance of Mean Stroke Volume Index

	Pre TD CO M	SD	Post TD CO M	SD	SS	MS	DF	F	p
Partial Pressure of Oxygen at Measurement Set 1									
Measurement Set 1	33.71	7.98	33.83	8.13	.02	.02	1/18	.12	.74
Measurement Set 2	36.20	9.88	36.37	10.06	.15	.15	1/18	.79	.39
Measurement Set 3	39.39	10.90	39.12	10.68	.00	.00	1/18	.01	.95
Measurement Set 4	38.42	10.61	38.59	10.70	.01	.01	1/18	.01	.91
Partial Pressure Oxygen at Measurement Set 3									
Measurement Set 3	39.39	10.90	39.12	10.68	1.12	1.12	1/18	1.56	.23
Measurement Set 4	38.42	10.61	38.59	10.70	.57	.57	1/18	1.29	.27
Oxygen Saturation at Measurement Set 1									
Measurement Set 1	33.71	7.98	33.83	8.13	.22	.22	1/18	1.69	.21
Measurement Set 2	36.20	9.88	36.37	10.06	.02	.02	1/18	.12	.74
Measurement Set 3	39.39	10.90	39.12	10.68	.45	.45	1/18	.74	.40
Measurement Set 4	38.42	10.61	38.59	10.70	.36	.36	1/18	.87	.36
Oxygen Saturation at Measurement Set 3									
Measurement Set 3	39.39	10.90	39.12	10.68	2.99	2.99	1/18	4.84	.04*
Measurement Set 4	38.42	10.61	38.59	10.70	.30	.30	1/18	.71	.41
Base Excess at Measurement Set 1									
Measurement Set 1	33.71	7.98	33.83	8.13	.00	.00	1/18	.02	.89
Measurement Set 2	36.20	9.88	36.37	10.06	.00	.00	1/18	.00	.98
Measurement Set 3	39.39	10.90	39.12	10.68	.26	.26	1/18	.35	.56
Measurement Set 4	38.42	10.61	38.59	10.70	.39	.39	1/18	.90	.36
Base Excess at Measurement Set 3									
Measurement Set 3	39.39	10.90	39.12	10.68	.05	.05	1/18	.07	.80
Measurement Set 4	38.42	10.61	38.59	10.70	.64	.64	1/18	1.46	.24
Airway Pressure at Measurement Set 1									
Measurement Set 1	31.00	7.55	31.11	7.54	.00	.00	1/10	.10	.76
Measurement Set 2	33.83	10.02	33.93	10.14	.03	.03	1/10	.19	.68
Measurement Set 3	38.85	12.50	38.91	12.55	.20	.20	1/10	.76	.40
Measurement Set 4	37.72	12.64	38.11	12.59	.28	.28	1/10	5.45	.04*
Airway Pressure at Measurement Set 3									
Measurement Set 3	75.59	27.19	75.75	27.47	.96	.96	1/10	3.71	.08
Measurement Set 4	73.93	27.18	73.93	27.03	.01	.01	1/10	.24	.63

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. * $p \leq .05$.

Analysis of Covariance of Mean Stroke Volume Index

	Pre TD CO M	Pre TD CO SD	Post TD CO M	Post TD CO SD	SS	MS	DF	F	p
Cardiopulmonary Bypass Time									
Measurement Set 1	34.13	7.95	34.25	8.11	.01	.01	1/18	.03	.87
Measurement Set 2	36.85	9.67	37.03	9.85	.44	.44	1/18	2.48	.13
Measurement Set 3	39.65	11.11	39.35	10.91	.03	.03	1/18	.04	.85
Measurement Set 4	38.84	10.70	39.01	10.80	.86	.86	1/18	2.02	.17
Aortic Cross Clamp Time									
Measurement Set 1	34.13	7.95	34.25	8.11	.05	.05	1/18	.25	.63
Measurement Set 2	36.85	9.67	37.03	9.85	.15	.15	1/18	.80	.38
Measurement Set 3	39.65	11.11	39.35	10.91	.01	.01	1/18	.02	.90
Measurement Set 4	38.84	10.70	39.01	10.80	.94	.94	1/18	2.21	.16
Core Temperature End of Surgery									
Measurement Set 1	33.71	7.98	33.83	8.13	.02	.02	1/13	.10	.76
Measurement Set 2	36.20	9.89	36.37	10.06	.02	.02	1/13	.06	.81
Measurement Set 3	39.40	10.90	39.12	10.68	.19	.19	1/13	.50	.49
Measurement Set 4	38.42	10.61	38.59	10.70	.12	.12	1/13	.39	.54
Core Temperature Upon Admission									
Measurement Set 1	33.71	7.98	33.83	8.13	.00	.00	1/13	.00	.99
Measurement Set 2	36.20	9.89	36.37	10.06	.00	.00	1/13	.00	.98
Measurement Set 3	39.40	10.90	39.12	10.68	.37	.37	1/13	.97	.34
Measurement Set 4	38.42	10.61	38.59	10.70	.17	.17	1/13	.57	.47
Core Temperature At Baseline									
Measurement Set 1	33.71	7.98	33.83	8.13	.04	.04	1/13	.20	.66
Measurement Set 2	36.20	9.89	36.37	10.06	.06	.06	1/13	.25	.62
Measurement Set 3	39.40	10.90	39.12	10.68	.76	.76	1/13	2.00	.18
Measurement Set 4	38.42	10.61	38.59	10.70	3.90	3.90	1/13	12.83	.00*
Core Temperature at Measurement Set 1									
Measurement Set 1	33.71	7.98	33.83	8.13	.49	.49	1/13	2.42	.14
Measurement Set 2	36.20	9.89	36.37	10.06	.11	.11	1/13	.44	.52
Measurement Set 3	39.40	10.90	39.12	10.68	7.02	7.02	1/13	18.37	.00*
Measurement Set 4	38.42	10.61	38.59	10.70	2.45	2.45	1/13	8.06	.02*
Core Temperature at Measurement Set 2									
Measurement Set 2	36.20	9.89	36.37	10.06	.27	.27	1/13	1.07	.32
Measurement Set 3	39.40	10.90	39.12	10.68	7.06	7.06	1/13	18.48	.00*
Measurement Set 4	38.42	10.61	38.59	10.70	1.02	1.02	1/13	3.34	.09
Core Temperature at Measurement Set 3									
Measurement Set 3	39.40	10.90	39.12	10.68	.47	.47	1/13	1.22	.30
Measurement Set 4	38.42	10.61	38.59	10.70	.10	.10	1/13	.34	.57
Core Temperatuare at Measurement Set 4									
Measurement Set 4	38.42	10.61	38.59	10.70	.80	.80	1/13	2.63	.13

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Stroke Volume Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Volume of Cardiac Output Injectate at Measurement Set 1									
Measurement Set 1	33.71	7.98	33.83	8.13	.27	.27	1/16	1.46	.25
Measurement Set 2	36.20	9.88	36.37	10.06	.02	.02	1/16	.09	.77
Measurement Set 3	39.39	10.90	39.12	10.68	.01	.01	1/16	.01	.92
Measurement Set 4	38.42	10.61	38.59	10.70	1.00	1.00	1/16	2.35	.15
Volume of Cardiac Output Injectate at Measurement Set 2									
Measurement Set 2	36.20	9.88	36.37	10.06	.09	.09	1/16	.51	.49
Measurement Set 3	39.39	10.90	39.12	10.68	.54	.54	1/16	1.24	.28
Measurement Set 4	38.42	10.61	38.59	10.70	1.54	1.54	1/16	3.58	.08
Volume of Cardiac Output Injectate at Measurement Set 3									
Measurement Set 3	39.39	10.90	39.12	10.68	1.44	1.44	1/16	3.29	.09
Measurement Set 4	38.42	10.61	38.59	10.70	.08	.08	1/16	.18	.68
Volume of Cardiac Output Injectate at Measurement Set 4									
Measurement Set 4	38.42	10.61	38.59	10.70	.25	.25	1/16	.59	.45
Fluid Balance at Baseline									
Measurement Set 1	33.71	7.98	33.83	8.13	.10	.10	1/15	.49	.49
Measurement Set 2	36.20	9.88	36.37	10.06	.14	.14	1/15	.63	.44
Measurement Set 3	39.39	10.90	39.12	10.68	.83	.83	1/15	1.01	.33
Measurement Set 4	38.42	10.61	38.59	10.70	.08	.08	1/15	.15	.71
Fluid Balance at Measurement Set 1									
Measurement Set 1	33.71	7.98	33.83	8.13	.04	.04	1/15	.18	.68
Measurement Set 2	36.20	9.88	36.37	10.06	.01	.01	1/15	.05	.83
Measurement Set 3	39.39	10.90	39.12	10.68	.31	.31	1/15	.38	.55
Measurement Set 4	38.42	10.61	38.59	10.70	.11	.11	1/15	.21	.65
Fluid Balance at Measurement Set 2									
Measurement Set 2	36.20	9.88	36.37	10.06	.25	.25	1/15	1.12	.31
Measurement Set 3	39.39	10.90	39.12	10.68	.28	.28	1/15	.34	.57
Measurement Set 4	38.42	10.61	38.59	10.70	.35	.35	1/15	.66	.43
Fluid Balance at Measurement Set 3									
Measurement Set 3	39.39	10.90	39.12	10.68	.17	.17	1/15	.21	.66
Measurement Set 4	38.42	10.61	38.59	10.70	.20	.20	1/15	.37	.55
Fluid Balance at Measurement Set 4									
Measurement Set 4	38.42	10.61	38.59	10.70	.11	.11	1/15	.20	.66

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Stroke Volume Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Sodium Nitroprusside at Measurement Set 1									
Measurement Set 1	33.71	7.98	33.83	8.13	.11	.11	1/17	.61	.45
Measurement Set 2	36.20	9.88	36.37	10.06	.08	.08	1/17	.42	.52
Measurement Set 3	39.40	10.90	39.12	10.69	.07	.07	1/17	.10	.76
Measurement Set 4	38.42	10.61	38.59	10.70	.95	.95	1/17	2.70	.12
Sodium Nitroprusside at Measurement Set 2									
Measurement Set 2	36.20	9.88	36.37	10.06	.00	.00	1/17	.00	.99
Measurement Set 3	39.40	10.90	39.12	10.69	.04	.04	1/17	.05	.83
Measurement Set 4	38.42	10.61	38.59	10.70	.56	.56	1/17	1.61	.22
Nitroglycerine at Measurement Set 1									
Measurement Set 1	38.42	10.61	38.59	10.70	.18	.18	1/17	.90	.36
Measurement Set 2	36.20	9.88	36.37	10.06	.45	.45	1/17	2.96	.10
Measurement Set 3	39.40	10.90	39.12	10.69	.02	.02	1/17	.02	.88
Measurement Set 4	38.42	10.61	38.59	10.70	.45	.45	1/17	.97	.35
Nitroglycerine at Measurement Set 2									
Measurement Set 2	36.20	9.88	36.37	10.06	.92	.92	1/17	5.96	.03*
Measurement Set 3	39.40	10.90	39.12	10.69	2.08	2.08	1/17	3.31	.09
Measurement Set 4	38.42	10.61	38.59	10.70	.07	.07	1/17	.16	.70
Epinephrine at Measurement Set 1									
Measurement Set 1	38.42	10.61	38.59	10.70	.00	.00	1/16	.02	.89
Measurement Set 2	36.20	9.88	36.37	10.06	.01	.01	1/16	.04	.84
Measurement Set 3	39.40	10.90	39.12	10.69	.31	.31	1/16	.37	.55
Measurement Set 4	38.42	10.61	38.59	10.70	.22	.22	1/16	.77	.39
Epinephrine at Measurement Set 2									
Measurement Set 2	36.20	9.88	36.37	10.06	.03	.03	1/16	.16	.69
Measurement Set 3	39.40	10.90	39.12	10.69	.01	.01	1/16	.02	.90
Measurement Set 4	38.42	10.61	38.59	10.70	.81	.81	1/16	2.80	.11
Epinephrine at Measurement Set 3									
Measurement Set 3	39.40	10.90	39.12	10.69	.14	.14	1/16	.17	.69
Measurement Set 4	38.42	10.61	38.59	10.70	1.58	1.58	1/16	5.44	.03*
Epinephrine at Measurement Set 4									
Measurement Set 4	38.42	10.61	38.59	10.70	.04	.04	1/16	.14	.71
Demerol, Morphine, and Diprivan at Measurement Set 1									
Measurement Set 1	36.20	9.88	36.37	10.06	.02	.02	1/16	.09	.77
Measurement Set 2	36.20	9.88	36.37	10.06	.21	.21	1/16	1.04	.32
Measurement Set 3	39.40	10.90	39.12	10.69	1.88	1.88	1/16	2.67	.12
Measurement Set 4	38.42	10.61	38.59	10.70	.01	.01	1/16	.02	.89
Demerol, Morphine, and Diprivan at Measurement Set 2									
Measurement Set 2	36.20	9.88	36.37	10.06	.24	.24	1/16	1.18	.29
Measurement Set 3	39.40	10.90	39.12	10.69	.90	.90	1/16	1.27	.28
Measurement Set 4	38.42	10.61	38.59	10.70	.05	.05	1/16	.11	.75
Demerol, Morphine, and Diprivan at Measurement Set 3									
Measurement Set 3	39.40	10.90	39.12	10.69	.17	.17	1/16	.24	.63
Measurement Set 4	38.42	10.61	38.59	10.70	.11	.11	1/16	.22	.65
Demerol, Morphine, and Diprivan at Measurement Set 4									
Measurement Set 4	38.42	10.61	38.59	10.70	.08	.08	1/16	.16	.70

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Systemic Vascular Resistance Index

	Pre TD CO M	SD	Post TD CO M	SD	SS	MS	DF	F	p
Partial Pressure of Oxygen at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	426.03	426.03	1/18	.05	.83
Measurement Set 2	1781.24	472.09	1808.19	469.85	11585.29	11585.29	1/18	7.57	.01*
Measurement Set 3	1600.81	362.42	1661.19	373.92	2533.25	2533.25	1/18	.47	.50
Measurement Set 4	1667.67	351.27	1670.52	359.83	602.65	602.65	1/18	.07	.79
Partial Pressure Oxygen at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	11104.17	11104.17	1/18	2.08	.17
Measurement Set 4	1667.67	351.27	1670.52	359.83	3738.58	3738.58	1/18	.45	.51
Oxygen Saturation at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	386.26	386.26	1/18	.03	.86
Measurement Set 2	1781.24	472.09	1808.19	469.85	3940.41	3940.41	1/18	1.93	.18
Measurement Set 3	1600.81	362.42	1661.19	373.92	443.78	443.78	1/18	.07	.79
Measurement Set 4	1667.67	351.27	1670.52	359.83	946.90	946.90	1/18	.11	.74
Oxygen Saturation at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	409.73	409.73	1/18	.07	.80
Measurement Set 4	1667.67	351.27	1670.52	359.83	1343.71	1343.71	1/18	.16	.70
Base Excess at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	1102.04	1104.04	1/18	.09	.76
Measurement Set 2	1781.24	472.09	1808.19	469.85	814.93	814.93	1/18	.32	.58
Measurement Set 3	1600.81	362.42	1661.19	373.92	13407.01	13407.01	1/18	2.55	.13
Measurement Set 4	1667.67	351.27	1670.52	359.83	2746.03	2746.03	1/18	.33	.57
Base Excess at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	11926.77	11926.77	1/18	2.27	.15
Measurement Set 4	1667.67	351.27	1670.52	359.83	1642.97	1642.97	1/18	.20	.66
Airway Pressure at Measurement Set 1									
Measurement Set 1	1938.00	441.16	2108.38	464.28	41346.35	41346.35	1/10	3.74	.08
Measurement Set 2	1867.69	536.59	1884.31	523.16	2047.49	2047.49	1/10	.69	.43
Measurement Set 3	1558.62	401.37	1600.08	420.38	10067.30	10067.30	1/10	1.80	.21
Measurement Set 4	1686.69	423.81	1654.23	384.67	3020.75	3020.75	1/10	.67	.43
Airway Pressure at Measurement Set 3									
Measurement Set 3	1558.62	401.37	1600.08	420.38	3565.65	3565.65	1/10	.64	.44
Measurement Set 4	1686.69	423.81	1654.23	384.67	6.16	6.16	1/10	.00	.97

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. * $p \leq .05$.

Analysis of Covariance of Mean Systemic Vascular Resistance Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Cardiopulmonary Bypass Time									
Measurement Set 1	1799.00	387.72	1849.85	435.14	34.98	34.98	1/18	.00	.95
Measurement Set 2	1750.05	461.62	1774.65	455.53	1154.82	1154.82	1/18	.44	.52
Measurement Set 3	1610.75	368.89	1668.20	382.21	5187.80	5187.02	1/18	.89	.36
Measurement Set 4	1658.90	358.03	1663.20	367.57	7792.95	7792.95	1/18	.96	.34
Aortic Cross Clamp Time									
Measurement Set 1	1799.00	387.72	1849.85	435.14	1231.15	1231.05	1/18	.12	.74
Measurement Set 2	1750.05	461.62	1774.65	455.53	8.20	8.20	1/18	.00	.96
Measurement Set 3	1610.75	368.89	1668.20	382.21	11531.04	11531.04	1/18	2.11	.16
Measurement Set 4	1658.90	358.03	1663.20	367.57	14188.16	14188.16	1/18	1.83	.19
Core Temperature End of Surgery									
Measurement Set 1	1835.19	412.69	1875.43	440.02	2331.69	2331.69	1/13	.20	.66
Measurement Set 2	1781.24	472.09	1808.19	469.85	7874.71	7874.71	1/13	2.82	.12
Measurement Set 3	1600.81	362.42	1661.19	373.92	11578.91	11578.91	1/13	1.87	.20
Measurement Set 4	1667.67	351.27	1670.52	359.83	2207.84	2207.84	1/13	.44	.52
Core Temperature Upon Admission									
Measurement Set 1	1835.19	412.69	1875.43	440.02	13.30	13.30	1/13	.00	.97
Measurement Set 2	1781.24	472.09	1808.19	469.85	5305.80	5305.80	1/13	1.90	.19
Measurement Set 3	1600.81	362.42	1661.19	373.92	5295.64	5295.64	1/13	.86	.37
Measurement Set 4	1667.67	351.27	1670.52	359.83	80.32	80.32	1/13	.02	.90
Core Temperature At Baseline									
Measurement Set 1	1835.19	412.69	1875.43	440.02	20.48	20.48	1/13	.00	.97
Measurement Set 2	1781.24	472.09	1808.19	469.85	134.60	134.60	1/13	.05	.83
Measurement Set 3	1600.81	362.42	1661.19	373.92	587.75	587.75	1/13	.01	.76
Measurement Set 4	1667.67	351.27	1670.52	359.83	33978.18	33978.18	1/13	6.83	.02*
Core Temperature at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	24079.48	24079.48	1/13	2.07	.17
Measurement Set 2	1781.24	472.09	1808.19	469.85	908.75	908.75	1/13	.33	.59
Measurement Set 3	1600.81	362.42	1661.19	373.92	629.49	629.49	1/13	.10	.76
Measurement Set 4	1667.67	351.27	1670.52	359.83	263.78	263.78	1/13	.05	.82
Core Temperature at Measurement Set 2									
Measurement Set 2	1781.24	472.09	1808.19	469.85	478.31	478.31	1/13	.17	.69
Measurement Set 3	1600.81	362.42	1661.19	373.92	21.19	21.19	1/13	.00	.95
Measurement Set 4	1667.67	351.27	1670.52	359.83	832.83	832.83	1/13	.17	.69
Core Temperature at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	2776.15	2776.15	1/13	.45	.52
Measurement Set 4	1667.67	351.27	1670.52	359.83	42.56	42.56	1/13	.01	.93
Core Temperatuare at Measurement Set 4									
Measurement Set 4	1667.67	351.27	1670.52	359.83	11784.08	11784.08	1/13	2.37	.15

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. * $p \leq .05$.

Analysis of Covariance of Mean Systemic Vascular Resistance Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Volume of Cardiac Output Injectate at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	1162.90	1162.90	1/16	.11	.75
Measurement Set 2	1781.24	472.09	1808.19	469.85	283.39	283.39	1/16	.10	.76
Measurement Set 3	1600.81	362.42	1661.19	373.92	5581.57	5581.57	1/16	.94	.35
Measurement Set 4	1667.67	351.27	1670.52	359.83	68.52	68.52	1/01	.01	.92
Volume of Cardiac Output Injectate at Measurement Set 2									
Measurement Set 2	1781.24	472.09	1808.19	469.85	156.43	156.43	1/16	.05	.82
Measurement Set 3	1600.81	362.42	1661.19	373.92	9948.05	9948.05	1/16	1.68	.21
Measurement Set 4	1667.67	351.27	1670.52	359.83	6329.27	6329.27	1/16	1.00	.33
Volume of Cardiac Output Injectate at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	966.28	966.28	1/16	.16	.69
Measurement Set 4	1667.67	351.27	1670.52	359.83	5551.65	5551.65	1/16	.88	.36
Volume of Cardiac Output Injectate at Measurement Set 4									
Measurement Set 4	1667.67	351.27	1670.52	359.83	52317.94	52317.94	1/16	8.25	.01*
Fluid Balance at Baseline									
Measurement Set 1	1835.19	412.69	1875.43	440.02	16192.13	16192.13	1/15	1.51	.24
Measurement Set 2	1781.24	472.09	1808.19	469.85	808.33	808.33	1/15	.46	.51
Measurement Set 3	1600.81	362.42	1661.19	373.92	8045.68	8045.68	1/15	1.25	.28
Measurement Set 4	1667.67	351.27	1670.52	359.83	3.43	3.43	1/15	.00	.98
Fluid Balance at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	1500.49	1500.49	1/15	.14	.71
Measurement Set 2	1781.24	472.09	1808.19	469.85	2822.13	2822.13	1/15	1.60	.23
Measurement Set 3	1600.81	362.42	1661.19	373.92	6365.93	6365.93	1/15	.99	.34
Measurement Set 4	1667.67	351.27	1670.52	359.83	27548.35	27548.35	1/15	4.55	.05*
Fluid Balance at Measurement Set 2									
Measurement Set 2	1781.24	472.09	1808.19	469.85	5902.72	5902.72	1/15	3.35	.09
Measurement Set 3	1600.81	362.42	1661.19	373.92	411.76	411.76	1/15	.06	.80
Measurement Set 4	1667.67	351.27	1670.52	359.83	53903.24	53903.24	1/15	8.91	.01*
Fluid Balance at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	2395.62	2395.62	1/15	.37	.55
Measurement Set 4	1667.67	351.27	1670.52	359.83	218.89	218.89	1/15	.04	.85
Fluid Balance at Measurement Set 4									
Measurement Set 4	1667.67	351.27	1670.52	359.83	8810.98	8810.98	1/15	1.46	.25

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Systemic Vascular Resistance Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Sodium Nitroprusside at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	9086.14	9086.14	1/17	1.14	.30
Measurement Set 2	1781.24	472.09	1808.19	469.85	115.73	115.73	1/17	.05	.83
Measurement Set 3	1600.81	362.42	1661.19	373.92	1546.24	1546.24	1/17	.28	.60
Measurement Set 4	1667.67	351.27	1670.52	359.83	5.0	5.0	1/17	.00	.98
Sodium Nitroprusside at Measurement Set 2									
Measurement Set 2	1781.24	472.09	1808.19	469.85	29.42	29.42	1/17	.01	.91
Measurement Set 3	1600.81	362.42	1661.19	373.92	3523.78	3523.78	1/17	.64	.44
Measurement Set 4	1667.67	351.27	1670.52	359.83	228.07	228.07	1/17	.04	.85
Nitroglycerine at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	3480.04	3480.04	1/17	.47	.50
Measurement Set 2	1781.24	472.09	1808.19	469.85	5060.51	5060.51	1/17	4.47	.05*
Measurement Set 3	1600.81	362.42	1661.19	373.92	4280.01	4280.01	1/17	1.12	.30
Measurement Set 4	1667.67	351.27	1670.52	359.83	5922.04	5922.04	1/17	.70	.42
Nitroglycerine at Measurement Set 2									
Measurement Set 2	1781.24	472.09	1808.19	469.85	20.72	20.72	1/17	.02	.89
Measurement Set 3	1600.81	362.42	1661.19	373.92	11204.68	11204.68	1/17	2.94	.11
Measurement Set 4	1667.67	351.27	1670.52	359.83	864.05	864.05	1/17	.10	.75
Epinephrine at Measurement Set 1									
Measurement Set 1	1835.19	412.69	1875.43	440.02	6402.14	6402.14	1/16	.58	.46
Measurement Set 2	1781.24	472.09	1808.19	469.85	5047.24	5047.24	1/16	2.26	.15
Measurement Set 3	1600.81	362.42	1661.19	373.92	1150.12	1150.12	1/16	.19	.67
Measurement Set 4	1667.67	351.27	1670.52	359.83	1258.01	1258.01	1/16	.20	.66
Epinephrine at Measurement Set 2									
Measurement Set 2	1781.24	472.09	1808.19	469.85	280.59	280.59	1/16	.13	.73
Measurement Set 3	1600.81	362.42	1661.19	373.92	2429.54	2429.54	1/16	.41	.53
Measurement Set 4	1667.67	351.27	1670.52	359.83	21717.56	21717.56	1/16	3.44	.08
Epinephrine at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	12711.84	12711.84	1/16	2.14	.16
Measurement Set 4	1667.67	351.27	1670.52	359.83	19305.55	19305.55	1/16	3.06	.10
Epinephrine at Measurement Set 4									
Measurement Set 4	1667.67	351.27	1670.52	359.83	316.70	316.70	1/16	.05	.83
Demerol, Morphine, and Diprivan at Measurement Set 1									
Measurement Sset 1	1835.19	412.69	1875.43	440.02	4212.93	4212.93	1/16	.46	.51
Measurement Set 2	1781.24	472.09	1808.19	469.85	8490.36	8490.36	1/16	4.27	.06
Measurement Set 3	1600.81	362.42	1661.19	373.92	4156.89	4156.89	1/16	.92	.35
Measurement Set 4	1667.67	351.27	1670.52	359.83	.01	.01	1/16	.00	.99
Demerol, Morphine, and Diprivan at Measurement Set 2									
Measurement Set 2	1781.24	472.09	1808.19	469.85	12948.64	12948.64	1/16	6.51	.02*
Measurement Set 3	1600.81	362.42	1661.19	373.92	27051.12	27051.12	1/16	6.00	.03*
Measurement Set 4	1667.67	351.27	1670.52	359.83	71.87	71.87	1/16	.01	.92
Demerol, Morphine, and Diprivan at Measurement Set 3									
Measurement Set 3	1600.81	362.42	1661.19	373.92	10632.28	10632.28	1/16	2.36	.14
Measurement Set 4	1667.67	351.27	1670.52	359.83	6700.79	6700.79	1/16	.96	.34
Demerol, Morphine, and Diprivan at Measurement Set 4									
Measurement Set 4	1667.67	351.27	1670.52	359.83	29034.94	29034.94	1/16	4.14	.06

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Pulmonary Vascular Resistance Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Partial Pressure of Oxygen at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	100.89	100.89	1/18	.19	.67
Measurement Set 2	296.10	87.34	301.81	95.75	266.38	266.38	1/18	.71	.41
Measurement Set 3	260.10	79.73	258.14	80.11	4799.58	4799.58	1/18	7.97	.01*
Measurement Set 4	260.57	75.80	263.76	80.09	297.72	297.72	1/18	.42	.53
Partial Pressure Oxygen at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	627.99	627.99	1/18	1.04	.32
Measurement Set 4	260.57	75.80	263.76	80.09	5474.00	5474.00	1/18	7.73	.01*
Oxygen Saturation at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	327.03	327.03	1/18	.60	.45
Measurement Set 2	296.10	87.34	301.81	95.75	37.97	37.97	1/18	.10	.75
Measurement Set 3	260.10	79.73	258.14	80.11	1979.45	1979.45	1/18	2.55	.13
Measurement Set 4	260.57	75.80	263.76	80.09	2.39	2.39	1/18	.00	.96
Oxygen Saturation at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	87.95	87.95	1/18	.11	.74
Measurement Set 4	260.57	75.80	263.76	80.09	1766.84	1766.84	1/18	1.89	.19
Base Excess at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	1069.00	1069.00	1/18	2.62	.12
Measurement Set 2	296.10	87.34	301.81	95.75	168.94	168.94	1/18	.46	.51
Measurement Set 3	260.10	79.73	258.14	80.11	224.72	224.72	1/18	.26	.62
Measurement Set 4	260.57	75.80	263.76	80.09	1774.56	1774.56	1/18	1.86	.19
Base Excess at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	377.30	377.30	1/18	.43	.52
Measurement Set 4	260.57	75.80	263.76	80.09	379.82	379.82	1/18	.40	.54
Airway Pressure at Measurement Set 1									
Measurement Set 1	320.15	109.22	346.77	118.47	1149.81	1149.81	1/10	2.47	.15
Measurement Set 2	325.77	95.14	335.62	104.48	141.60	141.60	1/10	.33	.58
Measurement Set 3	289.15	77.54	286.08	78.99	19.19	19.19	1/10	.01	.91
Measurement Set 4	283.92	82.69	285.15	12.59	3084.00	3084.00	1/10	2.95	.18
Airway Pressure at Measurement Set 3									
Measurement Set 3	263.76	80.09	38.59	10.70	896.39	896.39	1/10	.59	.46
Measurement Set 4	283.92	82.69	285.15	12.59	168.90	168.90	1/10	.16	.70

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Pulmonary Vascular Resistance Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Cardiopulmonary Bypass Time									
Measurement Set 1	284.20	104.41	306.20	109.26	49.24	49.24	1/18	.09	.77
Measurement Set 2	286.85	78.35	291.00	84.08	37.27	37.27	1/18	.10	.75
Measurement Set 3	255.30	78.63	254.40	80.28	.01	.01	1/18	.00	.99
Measurement Set 4	255.70	74.32	260.30	80.55	2421.97	2421.97	1/18	2.70	.12
Aortic Cross Clamp Time									
Measurement Set 1	284.20	104.41	306.20	109.26	2.28	2.28	1/18	.00	.95
Measurement Set 2	286.85	78.35	291.00	84.08	9.59	9.59	1/18	.03	.87
Measurement Set 3	255.30	78.63	254.40	80.28	329.94	329.94	1/18	.37	.55
Measurement Set 4	255.70	74.32	260.30	80.55	3365.90	3365.90	1/18	3.99	.06
Core Temperature End of Surgery									
Measurement Set 1	292.10	108.00	314.67	113.34	753.20	753.20	1/13	1.48	.25
Measurement Set 2	296.10	87.34	301.81	95.75	1007.89	1007.89	1/13	3.42	.09
Measurement Set 3	260.10	79.73	258.14	80.11	5889.65	5889.65	1/13	10.70	.01*
Measurement Set 4	260.57	75.80	263.76	80.09	728.87	728.87	1/13	.80	.39
Core Temperature Upon Admission									
Measurement Set 1	292.10	108.00	314.67	113.34	11.01	11.01	1/13	.02	.89
Measurement Set 2	296.10	87.34	301.81	95.75	185.46	185.46	1/13	.63	.44
Measurement Set 3	260.10	79.73	258.14	80.11	1431.81	1431.81	1/13	2.60	.13
Measurement Set 4	260.57	75.80	263.76	80.09	264.10	264.10	1/13	.29	.60
Core Temperature At Baseline									
Measurement Set 1	292.10	108.00	314.67	113.34	1959.42	1959.42	1/13	3.85	.07
Measurement Set 2	296.10	87.34	301.81	95.75	236.51	236.51	1/13	.80	.39
Measurement Set 3	260.10	79.73	258.14	80.11	222.05	222.05	1/13	.40	.54
Measurement Set 4	260.57	75.80	263.76	80.09	4223.88	4223.88	1/13	4.64	.05*
Core Temperature at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	836.69	836.69	1/13	1.64	.22
Measurement Set 2	296.10	87.34	301.81	95.75	222.87	222.87	1/13	.76	.40
Measurement Set 3	260.10	79.73	258.14	80.11	64.12	64.12	1/13	.12	.74
Measurement Set 4	260.57	75.80	263.76	80.09	1374.68	1374.68	1/13	1.51	.24
Core Temperature at Measurement Set 2									
Measurement Set 2	296.10	87.34	301.81	95.75	2.35	2.35	1/13	.01	.93
Measurement Set 3	260.10	79.73	258.14	80.11	113.14	113.14	1/13	.21	.66
Measurement Set 4	260.57	75.80	263.76	80.09	724.41	724.41	1/13	.80	.39
Core Temperature at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	103.05	103.05	1/13	.19	.67
Measurement Set 4	260.57	75.80	263.76	80.09	62.23	62.23	1/13	.07	.80
Core Temperatuare at Measurement Set 4									
Measurement Set 4	260.57	75.80	263.76	80.09	110.19	110.19	1/13	.12	.73

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Pulmonary Vascular Resistance Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Volume of Cardiac Output Injectate at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	1549.74	1549.74	1/16	3.32	.09
Measurement Set 2	296.10	87.34	301.81	95.75	3.55	3.55	1/16	.01	.93
Measurement Set 3	260.10	79.73	258.14	80.11	44.45	44.45	1/16	.05	.84
Measurement Set 4	260.57	75.80	263.76	80.09	1180.86	1180.86	1/01	1.31	.27
Volume of Cardiac Output Injectate at Measurement Set 2									
Measurement Set 2	296.10	87.34	301.81	95.75	123.88	123.88	1/16	.30	.59
Measurement Set 3	260.10	79.73	258.14	80.11	409.40	409.40	1/16	.42	.53
Measurement Set 4	260.57	75.80	263.76	80.09	3193.89	3193.89	1/16	3.53	.08
Volume of Cardiac Output Injectate at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	199.34	199.34	1/16	.20	.66
Measurement Set 4	260.57	75.80	263.76	80.09	733.39	733.39	1/16	.81	.38
Volume of Cardiac Output Injectate at Measurement Set 4									
Measurement Set 4	260.57	75.80	263.76	80.09	286.31	286.31	1/16	.32	.58
Fluid Balance at Baseline									
Measurement Set 1	292.10	108.00	314.67	113.34	7.59	7.59	1/15	.01	.92
Measurement Set 2	296.10	87.34	301.81	95.75	91.34	91.34	1/15	.26	.62
Measurement Set 3	260.10	79.73	258.14	80.11	220.87	220.87	1/15	.20	.66
Measurement Set 4	260.57	75.80	263.76	80.09	1037.05	1037.05	1/15	.99	.33
Fluid Balance at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	8.18	8.18	1/15	.01	.91
Measurement Set 2	296.10	87.34	301.81	95.75	635.32	635.32	1/15	1.78	.20
Measurement Set 3	260.10	79.73	258.14	80.11	.01	.01	1/15	.00	.99
Measurement Set 4	260.57	75.80	263.76	80.09	40.34	40.34	1/15	.04	.85
Fluid Balance at Measurement Set 2									
Measurement Set 2	296.10	87.34	301.81	95.75	49.78	49.78	1/15	.14	.71
Measurement Set 3	260.10	79.73	258.14	80.11	67.96	67.96	1/15	.06	.81
Measurement Set 4	260.57	75.80	263.76	80.09	.08	.08	1/15	.00	.99
Fluid Balance at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	27.01	27.01	1/15	.03	.88
Measurement Set 4	260.57	75.80	263.76	80.09	1857.18	1857.18	1/15	1.79	.20
Fluid Balance at Measurement Set 4									
Measurement Set 4	260.57	75.80	263.76	80.09	10.63	10.63	1/15	.01	.92

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Pulmonary Vascular Resistance Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Sodium Nitroprusside at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	1280.88	1280.88	1/17	2.75	.12
Measurement Set 2	296.10	87.34	301.81	95.75	25.23	25.23	1/17	.06	.81
Measurement Set 3	260.10	79.73	258.14	80.11	2.45	2.45	1/17	.00	.96
Measurement Set 4	260.57	75.80	263.76	80.09	2244.48	2244.48	1/17	2.52	.13
Sodium Nitroprusside at Measurement Set 2									
Measurement Set 2	296.10	87.34	301.81	95.75	2.66	2.66	1/17	.01	.94
Measurement Set 3	260.10	79.73	258.14	80.11	94.99	94.99	1/17	.10	.76
Measurement Set 4	260.57	75.80	263.76	80.09	3346.92	3346.92	1/17	3.75	.07
Nitroglycerine at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	260.04	260.04	1/17	.51	.49
Measurement Set 2	296.10	87.34	301.81	95.75	82.51	82.51	1/17	.21	.65
Measurement Set 3	260.10	79.73	258.14	80.11	950.04	950.04	1/17	1.22	.29
Measurement Set 4	260.57	75.80	263.76	80.09	1.04	1.04	1/17	.00	.97
Nitroglycerine at Measurement Set 2									
Measurement Set 2	296.10	87.34	301.81	95.75	421.17	421.17	1/17	1.09	.31
Measurement Set 3	260.10	79.73	258.14	80.11	1309.29	1309.29	1/17	1.68	.21
Measurement Set 4	260.57	75.80	263.76	80.09	952.38	952.38	1/17	1.70	.21
Epinephrine at Measurement Set 1									
Measurement Set 1	292.10	108.00	314.67	113.34	840.05	840.05	1/16	1.78	.20
Measurement Set 2	296.10	87.34	301.81	95.75	.23	.23	1/16	.00	.98
Measurement Set 3	260.10	79.73	258.14	80.11	240.14	240.14	1/16	.26	.62
Measurement Set 4	260.57	75.80	263.76	80.09	40.80	40.80	1/16	.04	.85
Epinephrine at Measurement Set 2									
Measurement Set 2	296.10	87.34	301.81	95.75	130.97	130.97	1/16	.32	.58
Measurement Set 3	260.10	79.73	258.14	80.11	1391.82	1391.82	1/16	1.50	.24
Measurement Set 4	260.57	75.80	263.76	80.09	538.25	538.25	1/16	.47	.50
Epinephrine at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	302.16	302.16	1/16	.33	.58
Measurement Set 4	260.57	75.80	263.76	80.09	17.54	17.54	1/16	.02	.90
Epinephrine at Measurement Set 4									
Measurement Set 4	260.57	75.80	263.76	80.09	118.88	118.88	1/16	.11	.75
Demerol, Morphine, and Diprivan at Measurement Set 1									
Measurement Sset 1	292.10	108.00	314.67	113.34	133.85	133.85	1/16	.25	.63
Measurement Set 2	296.10	87.34	301.81	95.75	.08	.08	1/16	.00	.99
Measurement Set 3	260.10	79.73	258.14	80.11	196.77	196.77	1/16	.24	.63
Measurement Set 4	260.57	75.80	263.76	80.09	678.64	678.64	1/16	.75	.40
Demerol, Morphine, and Diprivan at Measurement Set 2									
Measurement Set 2	296.10	87.34	301.81	95.75	252.28	252.28	1/16	.65	.43
Measurement Set 3	260.10	79.73	258.14	80.11	2019.77	2019.77	1/16	2.50	.13
Measurement Set 4	260.57	75.80	263.76	80.09	3090.63	3090.63	1/16	3.39	.08
Demerol, Morphine, and Diprivan at Measurement Set 3									
Measurement Set 3	260.10	79.73	258.14	80.11	36.40	36.40	1/16	.05	.84
Measurement Set 4	260.57	75.80	263.76	80.09	199.53	199.53	1/16	.22	.65
Demerol, Morphine, and Diprivan at Measurement Set 4									
Measurement Set 4	260.57	75.80	263.76	80.09	1975.39	1975.39	1/16	2.17	.16

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Left Ventricular Stroke Work Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Partial Pressure of Oxygen at Measurement Set 1									
Measurement Set 1	30.16	8.35	30.79	8.21	.11	.11	1/18	.08	.78
Measurement Set 2	32.43	9.09	32.95	9.03	4.45	4.45	1/18	6.42	.03*
Measurement Set 3	34.44	10.55	35.30	10.41	4.13	4.13	1/18	2.30	.15
Measurement Set 4	34.38	10.79	34.91	11.29	.77	.77	1/18	.39	.54
Partial Pressure Oxygen at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	8.32	8.32	1/18	4.63	.05*
Measurement Set 4	34.38	10.79	34.91	11.29	.28	.28	1/18	.14	.71
Oxygen Saturation at Measurement Set 1									
Measurement Set 1	30.16	8.35	30.79	8.21	1.25	1.25	1/18	.83	.37
Measurement Set 2	32.43	9.09	32.95	9.03	1.37	1.37	1/18	1.60	.22
Measurement Set 3	34.44	10.55	35.30	10.41	.45	.45	1/18	.20	.66
Measurement Set 4	34.38	10.79	34.91	11.29	.99	.99	1/18	.51	.49
Oxygen Saturation at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	3.57	3.57	1/18	1.58	.23
Measurement Set 4	34.38	10.79	34.91	11.29	1.76	1.76	1/18	.90	.36
Base Excess at Measurement Set 1									
Measurement Set 1	30.16	8.35	30.79	8.21	.01	.01	1/18	.01	.93
Measurement Set 2	32.43	9.09	32.95	9.03	.35	.35	1/18	.38	.54
Measurement Set 3	34.44	10.55	35.30	10.41	8.66	8.66	1/18	4.10	.06
Measurement Set 4	34.38	10.79	34.91	11.29	1.22	1.22	1/18	.61	.45
Base Excess at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	2.01	2.01	1/18	.95	.34
Measurement Set 4	34.38	10.79	34.91	11.29	.02	.02	1/18	.01	.93
Airway Pressure at Measurement Set 1									
Measurement Set 1	27.80	7.58	28.88	7.20	1.44	1.44	1/10	.95	.35
Measurement Set 2	30.54	8.41	31.05	8.74	1.55	1.55	1/10	2.10	.18
Measurement Set 3	32.91	11.00	33.44	10.82	4.03	4.03	1/10	2.36	.16
Measurement Set 4	33.29	12.30	33.58	12.62	1.73	1.73	1/10	.87	.37
Airway Pressure at Measurement Set 3									
Measurement Set 3	32.91	11.00	33.44	10.82	7.60	7.60	1/10	4.45	.06
Measurement Set 4	33.29	12.30	33.58	12.62	.38	.38	1/10	.19	.67

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Left Ventricular Stroke Work Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Cardiopulmonary Bypass Time									
Measurement Set 1	30.89	8.49	31.10	8.29	.41	.41	1/18	.28	.61
Measurement Set 2	32.98	8.94	33.52	8.86	2.20	2.20	1/18	2.42	.14
Measurement Set 3	34.74	10.74	35.59	10.59	.98	.98	1/18	.39	.54
Measurement Set 4	34.62	11.01	35.21	11.49	4.52	4.52	1/18	2.52	.13
Aortic Cross Clamp Time									
Measurement Set 1	30.89	8.49	31.10	8.29	.28	.28	1/18	.15	.70
Measurement Set 2	32.98	8.94	33.52	8.86	.92	.92	1/18	.94	.35
Measurement Set 3	34.74	10.74	35.59	10.59	2.53	2.53	1/18	1.03	.32
Measurement Set 4	34.62	11.01	35.21	11.49	5.57	5.57	1/18	3.21	.09
Core Temperature End of Surgery									
Measurement Set 1	30.16	8.35	30.79	8.21	.03	.03	1/13	.02	.88
Measurement Set 2	32.43	9.09	32.95	9.03	3.06	3.06	1/13	2.91	.11
Measurement Set 3	34.44	10.55	35.30	10.41	2.83	2.83	1/13	1.74	.21
Measurement Set 4	34.38	10.79	34.91	11.29	2.14	2.14	1/13	1.69	.22
Core Temperature Upon Admission									
Measurement Set 1	30.16	8.35	30.79	8.21	.03	.03	1/13	.02	.88
Measurement Set 2	32.43	9.09	32.95	9.03	.49	.49	1/13	.46	.51
Measurement Set 3	34.44	10.55	35.30	10.41	3.47	3.47	1/13	2.14	.17
Measurement Set 4	34.38	10.79	34.91	11.29	.04	.04	1/13	.03	.87
Core Temperature At Baseline									
Measurement Set 1	30.16	8.35	30.79	8.21	.19	.19	1/13	.13	.73
Measurement Set 2	32.43	9.09	32.95	9.03	.23	.23	1/13	.20	.65
Measurement Set 3	34.44	10.55	35.30	10.41	7.27	7.27	1/13	4.48	.05*
Measurement Set 4	34.38	10.79	34.91	11.29	4.29	4.29	1/13	3.37	.09
Core Temperature at Measurement Set 1									
Measurement Set 1	30.16	8.35	30.79	8.21	2.12	2.12	1/13	1.43	.25
Measurement Set 2	32.43	9.09	32.95	9.03	2.04	2.04	1/13	1.94	.19
Measurement Set 3	34.44	10.55	35.30	10.41	9.96	9.96	1/13	6.14	.03*
Measurement Set 4	34.38	10.79	34.91	11.29	1.09	1.09	1/13	.85	.37
Core Temperature at Measurement Set 2									
Measurement Set 2	32.43	9.09	32.95	9.03	2.59	2.59	1/13	2.47	.14
Measurement Set 3	34.44	10.55	35.30	10.41	5.90	5.90	1/13	3.64	.08
Measurement Set 4	34.38	10.79	34.91	11.29	2.76	2.76	1/13	2.17	.16
Core Temperature at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	.04	.04	1/13	.03	.88
Measurement Set 4	34.38	10.79	34.91	11.29	.23	.23	1/13	.18	.68
Core Temperatuare at Measurement Set 4									
Measurement Set 4	34.38	10.79	34.91	11.29	2.31	2.31	1/13	1.81	.20

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. * $p \leq .05$.

Analysis of Covariance of Mean Left Ventricular Stroke Work Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Volume of Cardiac Output Injectate at Measurement Set 1									
Measurement Set 1	30.16	8.35	30.79	8.21	.44	.44	1/16	.30	.60
Measurement Set 2	32.43	9.09	32.95	9.03	.01	.01	1/16	.01	.93
Measurement Set 3	34.44	10.55	35.30	10.41	3.82	3.82	1/16	1.63	.22
Measurement Set 4	34.38	10.79	34.91	11.29	.00	.00	1/01	.00	.97
Volume of Cardiac Output Injectate at Measurement Set 2									
Measurement Set 2	32.43	9.09	32.95	9.03	.17	.17	1/16	.17	.69
Measurement Set 3	34.44	10.55	35.30	10.41	5.83	5.83	1/16	2.49	.13
Measurement Set 4	34.38	10.79	34.91	11.29	.40	.40	1/16	.23	.64
Volume of Cardiac Output Injectate at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	1.26	1.26	1/16	.54	.47
Measurement Set 4	34.38	10.79	34.91	11.29	.30	.30	1/16	.17	.68
Volume of Cardiac Output Injectate at Measurement Set 4									
Measurement Set 4	34.38	10.79	34.91	11.29	9.08	9.08	1/16	5.25	.04*
Fluid Balance at Baseline									
Measurement Set 1	30.16	8.35	30.79	8.21	1.52	1.52	1/15	.92	.35
Measurement Set 2	32.43	9.09	32.95	9.03	.34	.34	1/15	.36	.56
Measurement Set 3	34.44	10.55	35.30	10.41	.24	.24	1/15	.08	.78
Measurement Set 4	34.38	10.79	34.91	11.29	.65	.65	1/15	.42	.53
Fluid Balance at Measurement Set 1									
Measurement Set 1	30.16	8.35	30.79	8.21	.01	.01	1/15	.01	.94
Measurement Set 2	32.43	9.09	32.95	9.03	1.48	1.48	1/15	1.59	.23
Measurement Set 3	34.44	10.55	35.30	10.41	2.61	2.61	1/15	.91	.35
Measurement Set 4	34.38	10.79	34.91	11.29	2.42	2.42	1/15	1.55	.23
Fluid Balance at Measurement Set 2									
Measurement Set 2	32.43	9.09	32.95	9.03	.24	.24	1/15	.26	.62
Measurement Set 3	34.44	10.55	35.30	10.41	.02	.02	1/15	.01	.94
Measurement Set 4	34.38	10.79	34.91	11.29	8.13	8.13	1/15	5.20	.04*
Fluid Balance at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	.40	.40	1/15	.14	.71
Measurement Set 4	34.38	10.79	34.91	11.29	.76	.76	1/15	.50	.50
Fluid Balance at Measurement Set 4									
Measurement Set 4	34.38	10.79	34.91	11.29	4.76	4.76	1/15	3.05	.10

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. * $p \leq .05$.

Analysis of Covariance of Mean Left Ventricular Stroke Work Index

	Pre TD CO M	Pre TD CO SD	Post TD CO M	Post TD CO SD	SS	MS	DF	F	p
Sodium Nitroprusside at Measurement Set 1									
Measurement Set 1	30.16	8.34	30.79	8.21	1.24	1.24	1/17	1.42	.25
Measurement Set 2	32.41	9.09	32.95	9.03	.04	.04	1/17	.04	.85
Measurement Set 3	34.44	10.55	35.30	10.41	.07	.07	1/17	.03	.88
Measurement Set 4	35.38	10.79	34.91	11.29	.03	.03	1/17	.01	.91
Sodium Nitroprusside at Measurement Set 2									
Measurement Set 2	32.41	9.09	32.95	9.03	.00	.00	1/17	.00	.99
Measurement Set 3	34.44	10.55	35.30	10.41	.50	.50	1/17	.19	.67
Measurement Set 4	35.38	10.79	34.91	11.29	.13	.13	1/17	.06	.80
Nitroglycerine at Measurement Set 1									
Measurement Set 1	30.16	8.34	30.79	8.21	.06	.06	1/17	.04	.85
Measurement Set 2	32.41	9.09	32.95	9.03	.02	.02	1/17	.04	.86
Measurement Set 3	34.44	10.55	35.30	10.41	2.67	2.67	1/17	1.85	.19
Measurement Set 4	35.38	10.79	34.91	11.29	1.58	1.58	1/17	.84	.37
Nitroglycerine at Measurement Set 2									
Measurement Set 2	32.41	9.09	32.95	9.03	1.20	1.20	1/17	1.77	.20
Measurement Set 3	34.44	10.55	35.30	10.41	8.46	8.46	1/17	5.87	.03*
Measurement Set 4	35.38	10.79	34.91	11.29	.38	.38	1/17	.20	.66
Epinephrine at Measurement Set 1									
Measurement Set 1	30.16	8.34	30.79	8.21	.21	.21	1/16	.14	.72
Measurement Set 2	32.41	9.09	32.95	9.03	1.73	1.73	1/16	1.89	.19
Measurement Set 3	34.44	10.55	35.30	10.41	.02	.02	1/16	.01	.93
Measurement Set 4	35.38	10.79	34.91	11.29	.21	.21	1/16	.11	.75
Epinephrine at Measurement Set 2									
Measurement Set 2	32.41	9.09	32.95	9.03	.03	.03	1/16	.03	.87
Measurement Set 3	34.44	10.55	35.30	10.41	1.57	1.57	1/16	.75	.10
Measurement Set 4	35.38	10.79	34.91	11.29	3.07	3.07	1/16	1.54	.23
Epinephrine at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	11.93	11.93	1/16	5.71	.03*
Measurement Set 4	35.38	10.79	34.91	11.29	.97	.97	1/16	.48	.50
Epinephrine at Measurement Set 4									
Measurement Set 4	35.38	10.79	34.91	11.29	.00	.00	1/16	.00	.98
Demerol, Morphine, and Diprivan at Measurement Set 1									
Measurement Set 1	32.41	9.09	32.95	9.03	.99	.99	1/16	.70	.42
Measurement Set 2	32.41	9.09	32.95	9.03	.01	.01	1/16	.01	.93
Measurement Set 3	34.44	10.55	35.30	10.41	.01	.01	1/16	.01	.94
Measurement Set 4	35.38	10.79	34.91	11.29	.05	.05	1/16	.03	.86
Demerol, Morphine, and Diprivan at Measurement Set 2									
Measurement Set 2	32.41	9.09	32.95	9.03	.17	.17	1/16	.16	.70
Measurement Set 3	34.44	10.55	35.30	10.41	2.36	2.36	1/16	.96	.34
Measurement Set 4	35.38	10.79	34.91	11.29	1.06	1.06	1/16	.66	.43
Demerol, Morphine, and Diprivan at Measurement Set 3									
Measurement Set 3	34.44	10.55	35.30	10.41	1.55	1.55	1/16	.63	.44
Measurement Set 4	35.38	10.79	34.91	11.29	6.03	6.03	1/16	3.75	.08
Demerol, Morphine, and Diprivan at Measurement Set 4									
Measurement Set 4	35.38	10.79	34.91	11.29	5.69	5.69	1/16	3.53	.08

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. * $p \leq .05$.

Analysis of Covariance of Mean Right Ventricular Stroke Work Index

	Pre TD CO M	SD	Post TD CO M	SD	SS	MS	DF	F	p
Partial Pressure of Oxygen at Measurement Set 1									
Measurement Set 1	4.76	1.53	5.11	1.77	.65	.65	1/18	1.95	.18
Measurement Set 2	5.52	2.14	5.70	2.07	.00	.00	1/18	.02	.89
Measurement Set 3	6.48	2.39	6.46	2.18	.67	.67	1/18	1.52	.23
Measurement Set 4	6.50	2.10	6.71	2.99	.05	.05	1/18	.05	.82
Partial Pressure Oxygen at Measurement Set 3									
Measurement Set 3	6.48	2.39	6.46	2.18	.02	.02	1/18	.05	.83
Measurement Set 4	6.50	2.10	6.71	2.99	.00	.00	1/18	.01	.94
Oxygen Saturation at Measurement Set 1									
Measurement Set 1	4.76	1.53	5.11	1.77	.29	.29	1/18	.82	.38
Measurement Set 2	5.52	2.14	5.70	2.07	.12	.12	1/18	.72	.41
Measurement Set 3	6.48	2.39	6.46	2.18	.44	.44	1/18	.99	.33
Measurement Set 4	6.50	2.10	6.71	2.99	.36	.36	1/18	.40	.53
Oxygen Saturation at Measurement Set 3									
Measurement Set 3	6.48	2.39	6.46	2.18	.00	.00	1/18	.00	.96
Measurement Set 4	6.50	2.10	6.71	2.99	.03	.03	1/18	.04	.85
Base Excess at Measurement Set 1									
Measurement Set 1	4.76	1.53	5.11	1.77	1.25	1.25	1/18	4.32	.05*
Measurement Set 2	5.52	2.14	5.70	2.07	.06	.06	1/18	.37	.55
Measurement Set 3	6.48	2.39	6.46	2.18	.05	.05	1/18	.12	.73
Measurement Set 4	6.50	2.10	6.71	2.99	.96	.96	1/18	1.09	.31
Base Excess at Measurement Set 3									
Measurement Set 3	6.48	2.39	6.46	2.18	1.05	1.05	1/18	2.53	.13
Measurement Set 4	6.50	2.10	6.71	2.99	.18	.18	1/18	.20	.66
Airway Pressure at Measurement Set 1									
Measurement Set 1	4.66	1.31	5.04	1.58	.22	.22	1/10	.57	.47
Measurement Set 2	5.57	2.31	5.66	2.22	.08	.08	1/10	.31	.59
Measurement Set 3	6.87	2.73	6.99	2.55	.08	.08	1/10	.18	.68
Measurement Set 4	6.70	2.48	6.94	3.49	.97	.97	1/10	1.08	.32
Airway Pressure at Measurement Set 3									
Measurement Set 3	6.87	2.73	6.99	2.55	.05	.05	1/10	.11	.75
Measurement Set 4	6.70	2.48	6.94	3.49	1.14	1.14	1/10	1.27	.29

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Right Ventricular Stroke Work Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Cardiopulmonary Bypass Time									
Measurement Set 1	4.77	1.57	5.16	1.80	.77	.77	1/18	2.43	.14
Measurement Set 2	5.61	2.14	5.78	2.09	.05	.05	1/18	.29	.60
Measurement Set 3	6.50	2.45	6.43	2.24	.06	.06	1/18	.14	.71
Measurement Set 4	6.53	2.14	6.76	3.06	.01	.01	1/18	.01	.93
Aortic Cross Clamp Time									
Measurement Set 1	4.77	1.57	5.16	1.80	.75	.75	1/18	2.36	.14
Measurement Set 2	5.61	2.14	5.78	2.09	.07	.07	1/18	.40	.53
Measurement Set 3	6.50	2.45	6.43	2.24	.02	.02	1/18	.05	.83
Measurement Set 4	6.53	2.14	6.76	3.06	.09	.09	1/18	.09	.76
Core Temperature End of Surgery									
Measurement Set 1	4.76	1.53	5.11	1.77	.03	.03	1/13	.13	.72
Measurement Set 2	5.51	2.14	5.70	2.07	.02	.02	1/13	.11	.75
Measurement Set 3	6.48	2.39	6.46	2.18	.01	.01	1/13	.03	.88
Measurement Set 4	6.50	2.09	6.71	2.99	.01	.01	1/13	.01	.94
Core Temperature Upon Admission									
Measurement Set 1	4.76	1.53	5.11	1.77	.30	.30	1/13	1.28	.28
Measurement Set 2	5.51	2.14	5.70	2.07	.01	.01	1/13	.07	.79
Measurement Set 3	6.48	2.39	6.46	2.18	.21	.21	1/13	.37	.55
Measurement Set 4	6.50	2.09	6.71	2.99	.31	.31	1/13	.30	.60
Core Temperature At Baseline									
Measurement Set 1	4.76	1.53	5.11	1.77	.07	.07	1/13	.28	.61
Measurement Set 2	5.51	2.14	5.70	2.07	.08	.08	1/13	.48	.50
Measurement Set 3	6.48	2.39	6.46	2.18	.21	.21	1/13	.38	.55
Measurement Set 4	6.50	2.09	6.71	2.99	.47	.47	1/13	.45	.52
Core Temperature at Measurement Set 1									
Measurement Set 1	4.76	1.53	5.11	1.77	.34	.34	1/13	1.43	.25
Measurement Set 2	5.51	2.14	5.70	2.07	.01	.01	1/13	.08	.78
Measurement Set 3	6.48	2.39	6.46	2.18	.08	.08	1/13	.14	.71
Measurement Set 4	6.50	2.09	6.71	2.99	.14	.14	1/13	.13	.72
Core Temperature at Measurement Set 2									
Measurement Set 2	5.51	2.14	5.70	2.07	.08	.08	1/13	.34	.57
Measurement Set 3	6.48	2.39	6.46	2.18	.05	.05	1/13	.13	.72
Measurement Set 4	6.50	2.09	6.71	2.99	.07	.07	1/13	.06	.81
Core Temperature at Measurement Set 3									
Measurement Set 3	6.48	2.39	6.46	2.18	.66	.66	1/13	1.19	.30
Measurement Set 4	6.50	2.09	6.71	2.99	.31	.31	1/13	.30	.59
Core Temperatuare at Measurement Set 4									
Measurement Set 4	6.50	2.09	6.71	2.99	.02	.02	1/13	.02	.89

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Right Ventricular Stroke Work Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
Volume of Cardiac Output Injectate at Measurement Set 1									
Measurement Set 1	4.76	1.52	5.11	1.77	.01	.01	1/16	.04	.85
Measurement Set 2	5.51	2.14	5.70	2.07	.05	.51	1/16	.30	.59
Measurement Set 3	6.48	2.39	6.46	2.18	.07	.07	1/16	.15	.71
Measurement Set 4	6.50	2.09	6.71	2.99	1.63	1.63	1/01	1.88	.19
Volume of Cardiac Output Injectate at Measurement Set 2									
Measurement Set 2	5.51	2.14	5.70	2.07	.00	.00	1/16	.00	.97
Measurement Set 3	6.48	2.39	6.46	2.18	.00	.00	1/16	.01	.92
Measurement Set 4	6.50	2.09	6.71	2.99	.28	.28	1/16	.32	.58
Volume of Cardiac Output Injectate at Measurement Set 3									
Measurement Set 3	6.48	2.39	6.46	2.18	.61	.61	1/16	1.23	.28
Measurement Set 4	6.50	2.09	6.71	2.99	.36	.36	1/16	.42	.53
Volume of Cardiac Output Injectate at Measurement Set 4									
Measurement Set 4	6.50	2.09	6.71	2.99	.64	.64	1/16	.74	.40
Fluid Balance at Baseline									
Measurement Set 1	4.76	1.52	5.11	1.77	1.50	1.50	1/15	5.34	.04*
Measurement Set 2	5.51	2.14	5.70	2.07	.10	.10	1/15	.79	.39
Measurement Set 3	6.48	2.39	6.46	2.18	.15	.15	1/15	.31	.59
Measurement Set 4	6.50	2.09	6.71	2.99	.03	.03	1/15	.04	.84
Fluid Balance at Measurement Set 1									
Measurement Set 1	4.76	1.52	5.11	1.77	.13	.13	1/15	.46	.51
Measurement Set 2	5.51	2.14	5.70	2.07	.36	.36	1/15	2.89	.11
Measurement Set 3	6.48	2.39	6.46	2.18	.16	.16	1/15	.33	.57
Measurement Set 4	6.50	2.09	6.71	2.99	2.10	2.10	1/15	3.08	.10
Fluid Balance at Measurement Set 2									
Measurement Set 2	5.51	2.14	5.70	2.07	.02	.02	1/15	.20	.67
Measurement Set 3	6.48	2.39	6.46	2.18	.35	.35	1/15	.74	.40
Measurement Set 4	6.50	2.09	6.71	2.99	2.52	2.52	1/15	3.69	.07
Fluid Balance at Measurement Set 3									
Measurement Set 3	6.48	2.39	6.46	2.18	.23	.23	1/15	.48	.50
Measurement Set 4	6.50	2.09	6.71	2.99	2.17	2.17	1/15	3.18	.10
Fluid Balance at Measurement Set 4									
Measurement Set 4	6.50	2.09	6.71	2.99	1.03	1.03	1/15	1.51	.24

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.

Analysis of Covariance of Mean Right Stroke Work Index

	Pre TD CO		Post TD CO						
	M	SD	M	SD	SS	MS	DF	F	p
				Sodium Nitroprusside at Measurement Set 1					
Measurement Set 1	4.76	1.53	5.11	1.77	1.37	1.37	1/17	6.36	.02*
Measurement Set 2	5.51	2.14	5.70	2.07	.01	.01	1/17	.06	.80
Measurement Set 3	6.48	2.39	6.46	2.18	.38	.38	1/17	.78	.39
Measurement Set 4	6.50	2.09	6.71	2.99	.29	.29	1/17	.32	.48
				Sodium Nitroprusside at Measurement Set 2					
Measurement Set 2	5.51	2.14	5.70	2.07	.02	.02	1/17	.01	.92
Measurement Set 3	6.48	2.39	6.46	2.18	.18	.18	1/17	.37	.55
Measurement Set 4	6.50	2.09	6.71	2.99	.48	.48	1/17	.52	.48
				Nitroglycerine at Measurement Set 1					
Measurement Set 1	4.76	1.53	5.11	1.77	.05	.05	1/17	.13	.72
Measurement Set 2	5.51	2.14	5.70	2.07	.52	.52	1/17	3.40	.08
Measurement Set 3	6.48	2.39	6.46	2.18	.18	.18	1/17	.48	.50
Measurement Set 4	6.50	2.09	6.71	2.99	.24	.24	1/17	.25	.62
				Nitroglycerine at Measurement Set 2					
Measurement Set 2	5.51	2.14	5.70	2.07	.27	.27	1/17	1.81	.20
Measurement Set 3	6.48	2.39	6.46	2.18	.96	.96	1/17	2.62	.12
Measurement Set 4	6.50	2.09	6.71	2.99	.01	.01	1/17	.01	.91
				Epinephrine at Measurement Set 1					
Measurement Set 1	4.76	1.53	5.11	1.77	.09	.09	1/16	.27	.61
Measurement Set 2	5.51	2.14	5.70	2.07	.37	.37	1/16	2.59	.13
Measurement Set 3	6.48	2.39	6.46	2.18	.55	.55	1/16	1.17	.30
Measurement Set 4	6.50	2.09	6.71	2.99	.06	.06	1/16	.01	.94
				Epinephrine at Measurement Set 2					
Measurement Set 2	5.51	2.14	5.70	2.07	.41	.41	1/16	2.86	.11
Measurement Set 3	6.48	2.39	6.46	2.18	.57	.57	1/16	1.22	.29
Measurement Set 4	6.50	2.09	6.71	2.99	1.09	1.09	1/16	1.18	.29
				Epinephrine at Measurement Set 3					
Measurement Set 3	6.48	2.39	6.46	2.18	.31	.31	1/16	.65	.43
Measurement Set 4	6.50	2.09	6.71	2.99	.85	.85	1/16	.93	.35
				Epinephrine at Measurement Set 4					
Measurement Set 4	6.50	2.09	6.71	2.99	.65	.65	1/16	.71	.41
				Demerol, Morphine, and Diprivan at Measurement Set 1					
Measurement Set 1	4.76	1.53	5.11	1.77	.77	.77	1/16	6.23	.02*
Measurement Set 2	32.41	9.09	32.95	9.03	.32	.32	1/16	1.95	.18
Measurement Set 3	6.48	2.39	6.46	2.18	1.06	1.06	1/16	2.93	.11
Measurement Set 4	6.50	2.09	6.71	2.99	.46	.46	1/16	.55	.47
				Demerol, Morphine, and Diprivan at Measurement Set 2					
Measurement Set 2	5.51	2.14	5.70	2.07	.22	.22	1/16	1.38	.26
Measurement Set 3	6.48	2.39	6.46	2.18	.00	.00	1/16	.00	.98
Measurement Set 4	6.50	2.09	6.71	2.99	.80	.80	1/16	.95	.34
				Demerol, Morphine, and Diprivan at Measurement Set 3					
Measurement Set 3	6.48	2.39	6.46	2.18	1.12	1.12	1/16	3.07	.10
Measurement Set 4	6.50	2.09	6.71	2.99	.02	.02	1/16	.03	.88
				Demerol, Morphine, and Diprivan at Measurement Set 4					
Measurement Set 4	6.50	2.09	6.71	2.99	3.18	3.18	1/16	3.81	.07

Note. Pre TD CO = measurement pre cardiac output, Post TD CO = measurement post cardiac output. *$p \leq .05$.