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The Effect of Head of Bed Elevation on Cerebrovascular Dynamics in Mild or Moderate Cerebral Vasospasm Following Aneurysmal Subarachnoid Hemorrhage

Patricia A. Blissitt

Doctor of Philosophy

University of Washington

2002

Program Authorized to Confer Degree:

School of Nursing

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Abstract

The Effect of Head of Bed Elevation on Cerebrovascular Dynamics in Mild or Moderate Cerebral Vasospasm Following Aneurysmal Subarachnoid Hemorrhage

Patricia A. Blissitt

Chair of the Supervisory Committee:
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Stroke is the leading cause of disability and the third leading cause of death. One type of stroke, aneurysmal subarachnoid hemorrhage accounts for approximately 7% of all strokes. One of the complications associated with aneurysmal subarachnoid hemorrhage is cerebral vasospasm. The sustained constriction of one or more intracranial blood vessels may result in additional cerebral ischemia or infarction. Vasospasm may begin from 3 to 5 days after the subarachnoid hemorrhage and continue up to 21 to 28 days after the bleed.

In the patient with aneurysmal subarachnoid hemorrhage, a delicate balance exists between preventive measures to decrease the consequences of immobility and the need to support optimal cerebral perfusion to prevent secondary brain injury. Based on tradition and consensus in individual institutions, the degree of head elevation during vasospasm has been limited by some clinicians in an attempt to minimize vasospasm and/or its sequelae. As a result, some individuals have remained on bedrest for weeks. However, prior to this study, no scientific investigation has been conducted to demonstrate the effect of head of bed elevation on cerebrovascular dynamics in vasospasm following aneurysmal subarachnoid hemorrhage.

This study is descriptive and quasi-experimental. Twenty subjects, between days 3 and 14 post aneurysmal subarachnoid hemorrhage in mild or moderate vasospasm, underwent head of bed elevations in a 0-20-45-0 degree sequence. Middle cerebral artery flow velocities and middle cerebral artery to internal carotid artery ratios, the determinants of cerebral vasospasm, were recorded in each position using transcranial Doppler technology. Descriptive and inferential statistics were computed. No patterns or trends were found that indicate head of bed elevation increases vasospasm. As a group, there were no statistically significant differences within subjects at the different head positions ($p \leq 0.05$). In addition, no individuals increased to severe vasospasm with the head of bed elevations at 20 and 45 degrees. In general, increasing the head of the bed did not cause harmful changes in cerebral blood flow. The fact that one individual did experience an increase from mild to moderate vasospasm indicates the value of being able to monitor vasospasm during head of bed elevation. Additional studies with a larger sample size, longer duration of head of bed elevations, and/or increased head of bed elevations are warranted.

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The author wishes to express sincere appreciation to the School of Nursing for their extended long-term support and especially to Professor Pamela H. Mitchell for her vast reserve of patience and knowledge. This thesis would never have been completed without the encouragement of family, friends, and colleagues.

Dedication

To my parents who selflessly devoted much of their life to me.

CHAPTER 1

INTRODUCTION and PROBLEM STATEMENT

Treatment of critically ill and injured individuals requires bedrest and thus relative immobilization. Espoused benefits of immobilization have included decreased oxygen consumption, decreased trauma to an affected body part, and conservation of energy resources needed for healing (Szaflarski, 1996). Although traditionally considered a cornerstone in the therapeutic management of critically ill and injured patients, immobilization may paradoxically result in secondary injury.

One patient population at risk for the consequences of immobility and impaired cerebral perfusion are critically ill persons with a diagnosis of acute stroke. An extremely vulnerable subpopulation of these patients are those who experience subarachnoid hemorrhage. Seven percent of all strokes are subarachnoid hemorrhage (American Heart Association, 2002). Stroke is the leading cause of disability and the third leading cause of death in the United States (American Heart Association, 1997; American Heart Association, 2002). Six hundred thousand strokes occur annually in the United States (American Heart Association, 2002). The annual cost for acute and long-term stroke care is approximately $30.8 billion (American Heart Association, 2002).

In the patient with acute stroke, including subarachnoid hemorrhage, a delicate balance exists between preventive measures to decrease the consequences of immobilization and the need to support optimal cerebral perfusion to prevent secondary brain injury. The nursing intervention of changing the bed position to a full sitting position illustrates this therapeutic dilemma. Positioning regimens are typically based on tradition and consensus at individual institutions. Few are actually based on physiologic responses to position change, specifically cerebral blood flow. Also few regimens identify individuals at risk for impaired cerebral perfusion when the head is raised to sitting position.

Goals of critical care nursing include not only facilitating critical care therapeutics but also protecting or at least minimizing the incidence of 1) secondary injury related to the primary illness or injury, and 2) consequences of immobility in this vulnerable population. Two specific consequences of immobility that may result in or contribute to secondary injury include 1) the loss of postural reflexes or orthostatic capacity, which may impair cerebral blood flow, and 2) pulmonary sequelae of atelectasis, pneumonia, aspiration, and pulmonary embolus.

Like other victims of stroke, aneurysmal subarachnoid hemorrhage patients are at risk for the consequences of immobility. Their care is further complicated by cerebral vasospasm that may occur from day 3 to day 28 after their hemorrhage (Mayberg, Batjer, & Dacey, 1994; Ullman & Bederson, 1996). Cerebral vasospasm may impair cerebral perfusion resulting in secondary neuronal ischemia and infarction.

In an effort to gain greater understanding toward optimal care of the patient with subarachnoid hemorrhage, the overall goal of this study was to explore the effect of the head of bed elevation on cerebral blood flow in aneurysmal subarachnoid hemorrhage vasospasm. The specific aims were to:

1) determine the effect of head of bed elevations of 20 and 45 degrees on cerebrovascular dynamics in mild or moderate cerebral vasospasm following aneurysmal subarachnoid hemorrhage in adults; and

2) describe mild or moderate vasospasm response to head of bed elevations of 20 and 45 degrees with respect to such demographics as grade of subarachnoid hemorrhage, autoregulation, and degree of vasospasm in adults.

CHAPTER 2

BACKGROUND AND SIGNIFICANCE

A number of factors must be taken into consideration when investigating the effect of the head position in vasospasm following aneurysmal subarachnoid hemorrhage. The systemic effects of the imposed bedrest restriction is one possible influence in regard to intracranial dynamics. The intracranial dynamics themselves are a complex interplay of factors, including intracranial pressure, cerebral perfusion pressure, autoregulation, cerebral metabolism, and vasospasm. Each and/or all of these together of these may impact the response to head of bed elevation following aneurysmal subarachnoid hemorrhage.

The Effects of Bedrest

The deconditioning effects of bedrest are well documented. Potentially detrimental physiologic changes begin within the first one to two days of bedrest. Several studies have been conducted with the head of the bed down in regard to the effects of immobilization on postural reflexes. Studies investigating the physiologic effects of immobilization, specifically bedrest, on healthy subjects date back to 1948 and are as recent as 1994. The period of bedrest ranged from three days to seven weeks with sample sizes of 4-14, predominantly men. The results of this research studies consistently showed a decreased tolerance to head-up tilt.

In 1948, Deitrick, Whedon, and Shorr, immobilized four healthy men for 6-7 weeks in plaster casts. The subjects were placed on a tilt table horizontally at 0 degrees, allowed to stabilize for 20 minutes and then tilted at 65 degrees. Heart rate, blood pressure, and clinical presentation were monitored every 1-2 minutes for 20 minutes. Results of the tilt table study showed clinical signs and symptoms of orthostatic hypotension, including fainting, within a week of immobilization. In addition, heart rate increased, blood pressure decreased and pulse pressure decreased by 10-12 mm Hg. In 1963, a similar study with comparable results was conducted by Birkhead and Blizzard (Valbona, Vogt, Cardus, et al., 1965).

Also in the mid-1960s in conjunction with the National Aeronautics Space Administration, a number of studies on the effect of bedrest were conducted by Valbona, Vogt, Cardus, et al. (1965). Prolonged bedrest was used as an analogue of weightlessness. At 3 and 14 days of bedrest, Valbona, Cardus, Vogt, and others (1965) demonstrated a decreased ability to tolerate passive tilt. A concurrent decrease in blood pressure and increase in heart rate was noted with worsening at day 14 compared to day 3. In a 1966 report of their work to date, Valbona and others implicated venous pooling and extracellular fluid shifts as one etiology of orthostatic intolerance (Vogt, Spencer, Cardus, et al., 1965).

Following the work of Valbona, and others, the use of more sophisticated technology in the 1990s demonstrated an impaired baroreceptor-cardiac reflex response in six subjects at days 3, 6, and 10 on bedrest with a head-down tilt of 6 degrees. Neck chambers were worn to stimulate receptors. As the subjects' heads were raised, prolongation of the R to R interval with continue bedrest reflected impaired baroreceptor response (Eckberg & Fritsch, 1992). Again in 1994, Takenaka, Suzuki, Kawakubo, and others demonstrated after 20 days of bedrest increased heart rate, decreased blood pressure and cardiac output. In addition, using neck chambers, this study demonstrated that the carotid vagal baroreceptor reflex was decreased in the flat position but unchanged in the sitting position.

Thus, decreased tolerance to head-up tilt is noted with prolonged bedrest in healthy individuals, with presumed intact autoregulation. In addition, as demonstrated by Takenaka, and others (1994), the postural reflexes may be more adversely affected by flat positioning than sitting.

Greenleaf and Kozlowski (1993) developed a time line for the physiologic changes that occur with bedrest in healthy individuals. From days 0-3, urinary diuresis and calcium loss; decreased plasma, interstitial and extracellular fluid volumes; decreased gastric secretions; decreased blood flow in the lower extremities; increased venous compliance; glucose intolerance; and loss of postural reflexes occur. On days 4-7, urinary loss of creatinine and phosphate; negative nitrogen balance, increased blood fibrinogen, clotting, and fibrinolytic activity; increased auditory thresholds; decreased visual acuity; and tilt-table intolerance were demonstrated. Days 8-14 included decreased red cell mass, decreased white cell phagocytosis, increased perspiration; and decreased heat conductance. Beyond 15 days, urinary loss of calcium; a secondary increase in auditory threshold; a change in heat sensitivity; and continued loss of postural reflexes occur. The head of bed down position also places the patient with neurological impairment at risk for aspiration from oral and/or gastric secretions and enteral feedings.

One patient population particularly at risk for the consequences of immobility and bedrest as described above are critically ill patients with a diagnosis of acute stroke. An extremely vulnerable subpopulation of these patients are those who experience subarachnoid hemorrhage. Subarachnoid hemorrhage accounts for 7% of all strokes (American Heart Association, 2002). Stroke is the leading cause of disability and the third leading cause of death in the United States (American Heart Association, 1997; American Heart Association, 2002). Six hundred thousand strokes occur annually in the United States (American Heart Association, 2002). The annual cost for acute and long-term stroke care is approximately $30.8 billion (American Heart Association, 2002). Depending on the reference and the study population, the incidence of subarachnoid hemorrhage ranges from 6 to 28 per 100,000 persons annually (Broderick, Brott, & Tomsick, 1993; Miller & Diringer, 1995).

In the patient with acute stroke, including subarachnoid hemorrhage, a delicate balance exists between preventive measures to decrease the consequences of immobilization and the need to support optimal cerebral perfusion to prevent secondary brain injury. The nursing intervention of changing the bed position to head elevation at various degrees illustrates this therapeutic dilemma. The patient with aneurysmal subarachnoid hemorrhage is at risk for secondary neurological injury, additional ischemia or irreversible infarction, related to impaired cerebral autoregulation, inadequate cerebral perfusion pressure, impaired cerebral metabolism and/or cerebral vasospasm. Cerebral autoregulation is the ability of the cerebrovasculature to maintain an adequate cerebral perfusion, within limits, despite variation in systemic blood pressure (Ursino, 1991). Cerebral perfusion pressure is the pressure gradient at which brain cells are perfused. Cerebral perfusion pressure is a function of the blood pressure and the intracranial pressure, specifically, mean arterial blood pressure minus intracranial pressure (Powers, 1992). Cerebral vasospasm is sustained arterial constriction (Findlay, Macdonald, & Weir, 1991). From day 3 to day 21 following intracranial aneurysm rupture, up to 46-70% of all individuals who survive the initial subarachnoid hemorrhage are at risk for cerebral vasospasm (Biller, Godersky, & Adams, 1988; Solenski, Haley, Kassel, et al., 1995). Cerebral vasospasm is typically most severe days 10-14 after the aneurysmal subarachnoid hemorrhage. The severity of vasospasm frequently follows a crescendo-decrescendo pattern with an intermediate plateau phase.

The effect of head of bed elevation in patients with vasospasm following aneurysmal subarachnoid hemorrhage has not been scientifically studied. Positioning regimens are typically based on tradition and consensus at individual institutions. Few are actually based on physiologic responses to position change, specifically cerebral blood flow. Some clinicians have adopted a regimen of bedrest with the head of the bed no higher than 30 degrees while other clinicians advocate increased mobilization to the sitting position with attention to blood pressure, cerebral perfusion pressure, and clinical presentation as indicators of cerebral perfusion. The rationale given for limiting the head of bed elevation to 30 degrees is the increased risk of decreased cerebral perfusion pressure and subsequent worsening of cerebral ischemia in the presence of vasospasm.

Previous research in regard to head position and cerebral perfusion is not supportive of a head of bed down, less than 20 degrees, position in cerebral vasospasm. Upon review of 29 studies conducted over the past 30 years in regard to head position and cerebral perfusion, this investigator found that most have involved head injury patients with cerebral perfusion pressure measurement being the single most frequently used indicator of cerebral blood flow (Blissitt, 2000). In the studies that included subarachnoid hemorrhage, the presence or absence of vasospasm was not noted. Position sequence and time from position change to measurement were variable. Furthermore, correlation between cerebral blood flow indicators and intravascular volume status were not noted. Timing of position change in regard to administration of medications was noted in only one study (March, Mitchell, Grady et al., 1990). Also, while head of bed down, flat and/or Trendelenburg, are frequently used in patients who are hypotensive, hemodynamically unstable, and/or with low cardiac output, at least three studies have found no association between blood pressure and head of bed elevation in critically ill patients (Kirchoff, Rebenson-Piano, & Patel, 1984; Quaglietti, Stotts, & Lovejoy, 1988; Grap, Cantley, Munro, et al., 1999).

Varying degrees of head of bed elevation is not full mobilization of the patient. However, head of bed elevation in the presence of mild or moderate aneurysmal subarachnoid hemorrhage vasospasm utilized before and after the time period when vasospasm is likely to be maximal, days 5 through 14, may rebuild any losses sustained from the initial immobilization associated with the intracranial and systemic effects of the aneurysmal subarachnoid hemorrhage, early surgical intervention, and head of bed down position with severe vasospasm. Thus, the head of bed elevation during mild or moderate vasospasm would allow a recovery period before and after vasospasm is its most severe, and prior to even greater mobilization.

Therefore, since no previous research specifically supported the head of bed down position nor disputed the head of bed up position in aneurysmal subarachnoid hemorrhage vasospasm, and head of bed elevation provided positive physiologic and psychosocial benefits, research regarding the effect of the head of bed elevation in mild or moderate vasospasm following aneurysmal subarachnoid was long overdue. Because patient positioning is in large part facilitated by nursing, this area of research is particularly suited to nursing. In addition to predicting the effects of earlier head of bed elevation on cerebral blood flow in the presence of vasospasm, the overall results of such a study may yield information which will support earlier mobilization of individuals with aneurysmal subarachnoid hemorrhage.

The intracranial dynamics of aneurysmal subarachnoid hemorrhage are complex. Multiple intracranial factors impact cerebral perfusion in aneurysmal subarachnoid hemorrhage. This chapter reviews each of these factors with particular attention to vasospasm. The physiology and physiologic measurement of vasospasm is described. Also, a review of previous research conducted on the relationship between head position and cerebral perfusion in critically ill patients with intracranial pathology is summarized.

Increased Intracranial Pressure/Decreased Perfusion Pressure

At the time of the aneurysmal rupture, arterial blood forcefully floods the subarachnoid space, including the basal cisterns. In less than a minute, the intracranial pressure approaches the diastolic or the mean arterial blood pressure. As a result, the cerebral perfusion pressure is drastically reduced and is woefully inadequate (Grote & Hassler, 1988). The cerebral perfusion pressure is the pressure gradient at which brain cells are perfused and is calculated clinically as the mean arterial blood pressure minus the intracranial pressure. Normal cerebral perfusion pressure is 70-100 mm Hg (Germon, Ladd, & Newton, 1996). In addition to increased volume in the subarachnoid space, extravasated blood/blood clots in the ventricles may block the outflow of cerebrospinal fluid and further contribute to increased intracranial pressure.

Although incompletely understood, two other mechanisms are thought to contribute to the initial rise in intracranial pressure. Transcranial Doppler recordings have demonstrated impaired autoregulation within the first few minutes after subarachnoid hemorrhage. Autoregulation, the ability of the cerebral blood vessels to maintain constant blood flow despite varying cerebral perfusion pressure, is only operational at a systemic mean arterial blood pressure of 50-170 mm Hg and is impaired at low cerebral perfusion pressures (Lassen, 1964; Strandgaard & Paulson, 1992). The initial increase in intracranial pressure may also be accompanied by a compensatory rise in systemic blood pressure. In the presence of impaired autoregulation, also referred to as vasomotor paralysis, an elevation in systemic blood pressure may markedly increase intracranial intravascular volume since the cerebral blood flow is passively dependent on the systemic blood pressure (Nornes & Magnes, 1972; Nornes, 1973; Grote & Hassler, 1988). The increased intracranial pressure that results from the increased intracranial volume may be sufficient to impair if not temporarily "arrest" cerebral blood flow resulting in primary neuronal injury and infarction. The mechanism of increased intracranial pressure in rebleeding is similar.

Cerebral edema increases over the first 24-72 hours after the bleed and may result in additional episodes of increased intracranial pressure. Hydrocephalus, communicating or noncommunicating, may also result in increased intracranial pressure for an indefinite time period (Milhorat, 1987; Heros, 1989; King & Martin, 1994). Other causes of increased intracranial pressure include cerebral edema resulting from surgical manipulation or infarction, and increased intravascular volume during hypervolemic hemodilution therapy for vasospasm (Orpello, Weiner, & Benjamin, 1996). A number of intracranial pressure monitoring systems are available. The fiberoptic-tipped catheter is one of the more commonly used methods. The catheter is zeroed prior to insertion. The catheter may be placed intraparenchymally, intraventricularly, epidurally, subdurally, or in the subarachnoid space. The mean intracranial pressure and waveform may be continuously monitored. The cerebral perfusion pressure is most easily obtained when the patient has a systemic arterial catheter in addition to the intracranial pressure monitor. Mean arterial pressure is used. While, the more accurate reference level for the measuring cerebral arterial pressure may be the ventricles, commonly referenced as the external auditory meatus, the arterial catheter is most frequently leveled at the phlebostatic axis to reflect systemic arterial pressure.

Autoregulation and Carbon Dioxide Reactivity (Vasoreactivity)

The impaired autoregulation that is postulated to occur as early as the first few minutes after aneurysmal subarachnoid hemorrhage may continue well beyond the time of the initial bleed. The upper and lower limits of autoregulation have been found to be shifted toward higher systemic blood pressures particularly during vasospasm (Yamamoto, Nishizawa, Tsukada, et al., 1998). With impaired cerebral vasomotor reactivity to systemic arterial blood pressure, passive decreases in cerebral blood flow as a result of systemic blood pressure below the lower limits of autoregulation may result in ischemia while passive increases in cerebral blood flow as a result of systemic blood pressure above the upper limits of autoregulation may result in edema and/or hemorrhage (Volby, Enevoldsen, & Jensen, 1985; Tenjin, Hirakawa, Mizukawa, et al., 1988; Dernbach, Little, Jones, et al., 1988).

In addition to dysautoregulation, reactivity to carbon dioxide may be impaired as well following aneurysmal subarachnoid hemorrhage. Carbon dioxide is a potent cerebral vasodilator and cerebral blood flow has been found to have a linear correlation ($r = +0.86$) with changes in arterial carbon dioxide (Grubb, Raichle, & Eichling, et al., 1974; Edvinsson, MacKenzie, & McCulloch, 1993). In the presence of impaired carbon dioxide reactivity, cerebral vessels may not constrict in response to controlled hyperventilation in an attempt to decrease intracranial pressure (Dernbach, Little, Jones, et al., 1988; Schmeider, Jarus-Dziedzic, Wronski, et al., 1997). An autoregulatory index may be obtained with transcranial Doppler technology and continuous arterial blood pressure monitoring.

Cerebral Blood Flow and Metabolism

A continued reduction in cerebral blood flow has been demonstrated following the initial precipitous drop associated with the time of aneurysmal rupture. Normal cerebral blood flow is approximately 50 milliliters per 100 grams of brain tissue per minute (Carpenter, 1991). Following aneurysmal subarachnoid hemorrhage, cerebral blood flow may be as low as 26-36 milliliters per 100 grams of brain tissue per minute (Fazl, Houlden, & Weaver, 1991). While the cerebral blood flow is generally at its lowest by two weeks after hemorrhage, it typically remains below normal for at least three weeks post bleed (Meyer, Lowe, Meyer, et al., 1983). Factors that contribute to the lower cerebral blood flow include systemic hypotension, increased intracranial pressure, cerebral vasospasm, and decreased cerebral metabolism. Cerebral blood flow is typically coupled to cerebral metabolic rate. Like cerebral blood flow, the rate of cerebral oxygen metabolism is typically decreased following aneurysmal subarachnoid hemorrhage and continues during vasospasm. The subarachnoid blood may act as a toxin on the cerebral metabolism. Later vasospasm may secondarily depress cerebral metabolism by decreasing cerebral blood flow (Martin, Baker, Grubb, et al., 1984; Carpenter, Grubb, Tempel, et al., 1991). One technology that allows continuous monitoring of the jugular oxygen saturation and calculation of the intracranial arteriovenous oxygen difference, an indirect measure of the cerebral metabolic rate of oxygen, is the jugular bulb catheter.

Cerebral Vasospasm

Overview

Cerebral vasospasm is defined as sustained arterial constriction, and is sometimes referred to as the "second stroke" following aneurysmal subarachnoid hemorrhage (Findlay, Macdonald, & Weir, 1991, p. 336). Sustained arterial constriction may result in cerebral ischemia and infarction. However, cerebral vasospasm is frequently differentiated as: 1) clinical, 2) radiographic, or 3) both. Cerebral vasospasm may be evident on angiography yet not present on clinical exam. Fewer than half of the individuals with angiographic vasospasm will show clinical signs of cerebral ischemia. Clinical cerebral vasospasm is sometimes termed "delayed cerebral ischemia" (Miller & Diringer, 1995, p. 464) or "delayed ischemic deficit (DID)" (Ullman & Bederson, 1996, p. 698). Vasospasm is not restricted to the cerebral vessel that ruptured but commonly includes other vessels throughout the cerebral circulation (Ullman & Bederson, 1996). The onset of cerebral vasospasm typically occurs between days 3 and 5 after aneurysmal subarachnoid hemorrhage, is maximal between days 5 and 14, and gradually resolves over days 14 to 28 (Mayberg, Batjer, & Dacey, 1994; Ullman & Bederson, 1996).

According to Miller and Diringer (1995), both genders are equally at risk for vasospasm as are those between 30 and 60 years old. Of the 15 studies reported in regard to the occurrence of subarachnoid hemorrhage and circadian rhythmicity, none describe a circadian rhythmicity specifically in regard to vasospasm (Folgelholm, Turjanmaa, Nuutila, et al., 1995; Kleinpeter, Schatzer, & Bock, 1995; Gallerani, Portaluppi, & Maida, et al., 1996). Individuals who are hypotensive, hypovolemic and/or critically ill are considered at greatest risk for cerebral vasospasm (Adams, Kassell, & Torner, 1987; Adams, 1992). According to several investigators, the greatest predictor for cerebral vasospasm is the amount of blood in the subarachnoid space/basal cisterns. Clots greater than 3 millimeters by 5 millimeters in the basal cisterns or layers of blood in the subarachnoid space of 1 millimeter or thicker are strongly associated with vasospasm nearly 100% of the time (Fisher, Kistler, & Davis, 1980; Miller & Diringer, 1995).

Pathogenesis and Biochemical Alterations

The pathogenesis of cerebral aneurysms, aneurysmal subarachnoid hemorrhage, and vasospasm is not entirely known. The question as to whether cerebral aneurysms are congenital, or acquired/degenerative, or both remains unanswered. Evidence for the congenital defect hypothesis, specifically, a defect in the medial layer of the arterial wall, includes: 1) the 20% incidence of multiple intracranial aneurysms; 2) an increased occurrence among family members; and 3) the occurrence of aneurysms in association with arteriovenous malformations and other systemic inherited diseases such as polycystic kidney, Marfan's syndrome, Ehlers-Danlos syndrome, and coarctation of the aorta (Wilkins, 1981; Weaver & Fisher, 1994). Support for an acquired/degenerative defect in the arterial wall includes the increased frequency of cerebral aneurysms with age, systemic hypertension, smoking, and atherosclerosis (Knekt, Reunanen, Aho, et al., 1991; Weaver & Fisher, 1994).

Following subarachnoid hemorrhage, a number of biochemical alterations occur at the cellular level, including the development of lactic acidosis with increased lactate concentrations and lactate/pyruvate ratios; generation of free radicals and lipid peroxidation; the release of excitatory amino acids, including glutamate and aspartate; changes in glial function; and apoptosis. Recent bedside neurochemical monitoring using intracerebral microdialysis catheters in the neurosurgical intensive care unit has shown increased lactate/pyruvate ratios and increased concentrations of lactate, glutamate and aspartate in poor-grade/poor outcome aneurysmal subarachnoid hemorrhage patients (Saveland, Nilsson, & Boris-Moller, 1996; Persson, Valtysson, Enblad, et al., 1996; Unterberg, Sakowitz, Sarrafzedeh, et al., 2001). These multiple biochemical alterations are thought to be responsible for much of the ischemia and secondary neuronal injury associated with subarachnoid hemorrhage.

In addition to biochemical changes described above, a number of substances have been investigated as potential mediators of cerebral vasospasm as well, including catecholamines, serotonin, bradykinin, bilirubin, angiotensin, fibrin degradation products, oxyhemoglobin, endothelin, oxygen free radicals, eicosanoids (prostaglandins), and calcium. Since vasospasm follows the release of blood into the subarachnoid space, many investigators have logically focused on these agents because of their presence in the blood vessel wall, the blood itself, and/or cerebrospinal fluid. Those currently under most intense scrutiny are oxyhemoglobin, endothelin, oxygen free radicals, eicosanoids, and calcium (Cook, 1995; Miller & Diringer, 1995). The release of oxyhemoglobin from the hemolysis of erythrocytes in the subarachnoid clot is generally believed to be a major factor in the development of cerebral vasospasm (Pasqualin, 1998). The oxidized hemoglobin and iron released from the breakdown of erythrocytes contribute to the production of free radicals and lipid peroxidation. The formation of free radicals and lipid peroxidation are injurious to the endothelium of the blood vessels. Although oxyhemoglobin itself is considered a weak vasoconstrictor, the free radicals directly constrict the vascular smooth muscle (Cook, 1995). As a result of endothelial injury, protein kinase C (PKC) is activated which results in additional constriction. Protein kinase C also contributes to the endothelium injury and the production of vasoconstricting prostaglandins. Following subarachnoid hemorrhage, the synthesis of vasoconstricting prostaglandins and thromboxane is increased, and the decreased production of the vasodilatory prostaglandin, prostacyclin may or may not contribute to vasospasm (Pasqualin, 1998; Findlay, Macdonald, & Weir, 1991).

Physiologic Measurement

Cerebral angiography remains the gold standard for the diagnosis of cerebral vasospasm. On angiogram, vasospastic cerebral vessels appear narrowed, "string-effect" or completely obliterated since the movement of contrast material through the narrowed lumen is dramatically decreased or halted. However, within the past 15 years, transcranial Doppler ultrasonography has been increasingly used for bedside diagnosis of cerebral vasospasm. It is noninvasive and does not expose the patient to radiation or potentially nephrotoxic contrast material. Cerebral vasospasm following subarachnoid hemorrhage is associated with increased middle cerebral artery flow velocity and increased middle cerebral artery (MCA) flow velocity to internal carotid artery (ICA) flow velocity ratio (Table 1).

Table 1. Criteria for Determination of Middle Cerebral Artery Vasospasm

Severity	Velocity	MCA: ICA Ratio
Mild	120-149 cm/sec	3.0-5.9
Moderate	150-199 cm/sec	3.0-5.9
Severe	200+ cm/sec	6.0+

The Effect of Head of Bed Elevation on Cerebral Blood Flow in Critically Ill Patients with Intracranial Pathology

As stated in Chapter 1, no research has been conducted specifically in regard to the effect of head of bed elevation on cerebral blood flow in vasospasm following aneurysmal subarachnoid hemorrhage. However, in the past 17 years, eight studies have investigated the effect of head of bed elevation on cerebral blood flow in critically ill patients with intracranial pathology. In general, these studies were of small sample size, ranging from 4 to 37 patients. Samples consisted primarily of individuals with varied traumatic brain injury but some studies included such diagnoses as brain tumors, hydrocephalus, and near-drowning. A diagnosis of subarachnoid hemorrhage was included in five studies: March, Mitchell, Grady, et al., (1990); Feldman, Kanter, Robertson, et al., (1992); Schneider, vonHelden, Franke, et al., (1993); Meixensberger, Baunach, Amschler, et al., (1997); and Moraine, Berre, & Melot (2000). However, in each of these studies, the etiology of the subarachnoid hemorrhage was either traumatic or not stated. None of the studies included information about the presence or absence of vasospasm.

In the earliest two studies involving critically ill patients with intracranial pathology, Durward, Amacher, and Del Maestro (1983), and Rosner and Coley (1986), measured cerebral perfusion pressure as an indirect indicator of cerebral blood flow. Cerebral perfusion pressure was obtained by subtracting intracranial pressure from the mean arterial pressure, using arterial catheter pressures with transducers leveled at both the heart and head.

The six later studies, March, Mitchell, and Grady, (1990), Feldman, Kanter, and Robertson (1992), Schneider, vonHelden, and Franke (1993) Gopinath, Robertson, and Narayan (1994), Meixensberger, Baunach, and Amschler (1997), and Moraine, Berre, and Melot (2000) measured other indicators of cerebral blood flow in addition to cerebral perfusion pressure, including: cerebral blood flow velocity per transcranial Doppler (March, Mitchell, Grady, et al., 1990); cerebral arteriovenous oxygen difference and oxygen saturation per jugular bulb (Schneider, vonHelden, & Franke, 1993); metabolic rate of oxygen (CMRO2), and cerebral vascular resistance per Kety-Schmidt nitrous oxide technique and oxygen saturation per jugular bulb (SjO_2) (Feldman, Kanter, Robertson, et al., 1992); cortical cerebral blood flow and blood volume per thermal diffusion cerebral blood flow probe and near-infrared spectroscopy, respectively (Gopinath, Robertson, & Narayan, 1994); regional partial pressure of oxygen per tissue-pO2 microcatheter (Meixensberger, Baunach, & Amschler, 1997); and cerebral blood flow velocity, arteriovenous pressure gradient and metabolic calculations derived from jugular catheter data, and cerebral autoregulation testing (Moraine, Berre, & Melot, 2000).

Variations in the method of head of bed elevation and time from measurement were noted. Durward, Amacher, and Del Maestro elevated the head of the bed at 15-30-60 and 0-30-60 degree sequences and began measurements five minutes after position change. Rosner and Coley elevated the head of the bed from 0-50 degrees in 10 degree increments. March, Mitchell, and Grady elevated the head of the bed from 0 to 30 degrees and obtained cerebral perfusion pressure and cerebral blood flow velocity at 2 and 15 minutes after position change. Study reports by Feldman, Kanter, and Robertson; Schneider, vonHelden, and Franke; and Gopinath, Robertson, and Narayan did not include time from position change to measurement. Feldman, Kanter, and Robertson elevated the head of the bed from 0 to 30 degrees and Gopinath, Robertson, and Narayan elevated the head of the bed from 0 to 30 and 0 to 60 degrees. Schneider, vonHelden, and Franke began data collection with the head of the bed at 45 degrees and then decreased it to 30, 15, and 0 degrees. Meixensberger, Baunach, and Amschler elevated the head of the bed from 0 to 30 degrees and obtained measurements 10-15 minutes later. Moraine, Berre, and Melot randomly assigned patients to only one of four positions, 0, 15, 30, or 45 degrees head of bed elevation and allowed 10 minutes stabilization before data collection.

Results from these eight studies have been varied. Durward, Amacher, and Del Maestro demonstrated that elevating the head of the bed to 60 degrees decreased the cerebral perfusion pressure significantly. Rosner and Coley showed that cerebral perfusion pressure was maximal at 0 degrees, when compared with various degrees of elevation. March, Mitchell, and Grady observed variable results in regard to cerebral perfusion pressure and cerebral blood flow with head elevation to 30 degrees. Feldman, Kanter, and Robertson showed that head elevation to 30 degrees maintained cerebral perfusion pressure and cerebral blood flow in 17 of 22 patients. The remaining 5 patients demonstrated 5% decreases in cerebral blood flow that were not considered significant (p = 0.119). Schneider, vonHelden, and Franke demonstrated that head elevations of 0, 15, 30, and 45 degrees did not markedly decrease cerebral perfusion pressure or jugular venous oxygenation. Gopinath, Robertson, and Narayan, observed an 11-20% decrease in blood volume at 30 and 60 degrees respectively but no other parameter changes were considered. Meixensberger, Baunach, and Amschler showed that head of bed elevation to 30 degrees did not impair regional cerebral microcirculation. Finally, Moraine, Berre, and Melot found that arteriovenous pressure, not cerebral perfusion pressure, was the major determinant of cerebral blood flow.

Conclusions

Intracranial dynamics impacting cerebral perfusion in aneurysmal subarachnoid hemorrhage include increased intracranial pressure/decreased cerebral perfusion pressure, impaired autoregulation, impaired cerebral metabolism, and vasospasm. Numerous studies have been conducted in regard to head of bed elevation and cerebral perfusion. Methods and results have varied. However, no one has specifically studied the effect of head of bed elevation on vasospasm following aneurysmal subarachnoid hemorrhage.

CHAPTER III

METHODS OF PROCEDURES

A discussion of the research design, sample, measures and instruments, data collection procedure and information related to human subjects is included in this chapter. A description of the data analysis procedures is provided as well.

Design

This study was a repeated measures design that was prospective and quasi-experimental. Subjects served as their own controls. Physiologic response to head of bed elevations of 20 and 45 degrees on cerebrovascular dynamics in the presence of mild or moderate cerebral vasospasm following aneurysmal subarachnoid hemorrhage was observed. Each subject underwent a sequence of 0-20-45-0 degrees head of bed position change.

Sample

Data were collected on a convenience sample of 24 patients in the intensive care units at a West Coast university medical center. Criteria for selection include adults, ages 18 to 99 years with mild or moderate cerebral vasospasm, defined as a middle cerebral artery flow velocity of 120-149 cm/sec and 150-200 cm/sec respectively and a middle cerebral artery to internal carotid artery ratio of 3.0-5.9, within 3-14 days following aneurysmal subarachnoid hemorrhage. The presence and degree of vasospasm were determined by routine transcranial Doppler (TCD) studies that were administered routinely to all patients who were post-aneurysmal subarachnoid hemorrhage. All patients were postoperative or post-placement of coils in the aneurysmal sac, since surgical or interventional neuroradiologic intervention within the first 72 hours of the aneurysmal bleed is the usual treatment.

Criteria for exclusion from the study included subarachnoid hemorrhage due to traumatic brain injury and/or arteriovenous malformation rupture; the absence of vasospasm as determined by transcranial Doppler; severe cerebral vasospasm as determined by transcranial Doppler (200+ cm/sec middle cerebral artery flow velocity and $\geq$ 6.0 middle cerebral artery to internal carotid artery ratio); the presence of ruptured aneurysm without surgical or interventional neuroradiographic intervention; and a baseline cerebral perfusion pressure less than 70 mm Hg.

Measures and Instruments

Middle cerebral artery (MCA) blood flow velocity, middle cerebral artery to internal carotid artery (MCA:ICA) ratio, heart rate, oxygen saturation, and mean arterial blood pressure (MAP) were measured in each position. When available, autoregulatory index (ARI), intracranial pressure (ICP), cerebral perfusion pressure (CPP), pulmonary artery pressure, pulmonary artery wedge pressure (PAWP), and/or central venous pressure (CVP) were obtained as well. Intracranial pressure, cerebral perfusion pressure, vital signs, and hemodynamic parameters per Camino, Spacelab, and Baxter-Edwards monitors were part of the standard care of the patient with subarachnoid hemorrhage. Cerebral perfusion pressure was obtained based on calculations using data obtained from Camino intracranial (ICP) monitors and radial arterial catheters or continuous noninvasive blood pressure monitors.

When supine and flat, baseline 0 degrees and the second 0 degree, and at each head of bed elevation, 20 and 45 degrees, radial arterial catheter transducers were leveled with the heart (at the phlebostatic axis) and with the head (at the external auditory meatus) and differences noted. When radial arterial catheters were not present, continuous noninvasive blood pressures were obtained using the Nellcor N-CAT continuous noninvasive blood pressure monitor. The Nellcor N-CAT continuous noninvasive blood pressure monitor combines tonometry in combination with the standard oscillometric measurement to provide continuous blood pressure waveforms, systolic, diastolic, and mean blood pressures, and heart rate with a digital display. A sensor was placed on the wrist in addition to a cuff around the upper arm. The cuff was leveled at the phlebostatic axis for accuracy (Nellcor, 1991).

The middle cerebral artery flow velocity was derived from the transcranial Doppler, Multi-Dop X4, using a headgear for continuous monitoring. The Multi-Dop X4 Doppler unit provides continuous graphic and digital display with a continuous recording feature that includes simultaneous measurement of bilateral middle cerebral artery flow velocity, mean arterial pressure and intracranial pressure. The autoregulatory index may be obtained as well with rapid and marked decreases in blood pressure. The autoregulatory index ranges from 0 to 9, with 0 indicating the absence of autoregulation and 9 signifying maximal autoregulation. Approximately 70% of healthy subjects have an autoregulatory index of 5 (Multi-Dop X4, 1996). The bilateral internal carotid artery blood flow velocity was determined with the Neuroguard Transcranial Doppler. The right and left middle cerebral arteries were insonated separately and simultaneously during the study procedure. The right and left internal carotid arteries were insonated separately with a handheld probe. Criteria for the determination of vasospasm (specific to the middle cerebral artery and its severity is included in Table 1 (Douville, Newell, & Trimble, 1990; Seiler & Newell, 1992).

Table 1. Criteria for Determination of Middle Cerebral Artery Vasospasm

Severity	**Velocity**	**MCA:ICA Ratio**
Mild	120-149 cm/sec	3.0-5.9
Moderate	150-199 cm/sec	3.0-5.9
Severe	200+ cm/sec	6.0+

Prior to each data collection, proper positioning of the transcranial Doppler headgear for accurate insonation and clear signal was verified by the investigator and an experienced sonographer. All hemodynamic monitoring, arterial catheter blood pressure, central venous pressure and/or pulmonary artery wedge pressure were zeroed and placed at phlebostatic axis with the exception of the arterial catheter transducer that was also placed at the external auditory meatus for the arterial blood pressure at the subject's head. Phlebostatic axis and external auditory meatus were verified by two experienced critical care nurses, the researcher and a staff nurse. Transducers were re-zeroed at each position change. Pulmonary artery pressure/pulmonary artery wedge pressure and central venous pressures were obtained at end-expiration and determined by waveform interpretation with paper recording. Again the two experienced critical care nurses, the researcher and a staff nurse, verified waveform interpretation. Physiologic measures are summarized in Table 2.

Each change in head of bed elevation was verified with a goniometer. Head of bed elevation was verified with an experienced critical care nurse and/or a transcranial Doppler sonographer. Baseline vital signs were obtained 15 minutes prior to beginning the study. Data including grade of aneurismal subarachnoid hemorrhage per Hunt-Hess Scale, days since subarachnoid hemorrhage, and current medications and intravenous fluids (and their last administration) were obtained from the clinical record as well.

Procedures

Potential subjects who were able to give consent or the legal next-of -kin of potential subjects who were unable to give consent were approached in regard to consent within the first two or three days of admission or first one or two days post-operative or post coiling. For those subjects who met the inclusion criteria and who provided informed consent was obtained, the study procedure was added on to the routine transcranial Doppler study once the routine study was completed, after the subject was found to be in mild or moderate vasospasm. In most instances, the study data were collected a few hours after the daily transcranial Doppler. Each subject underwent four head of bed positions: 0, 20, 45, and 0 degrees, pausing approximately 8-10 minutes at each position; allowing measurement of intracranial pressure, cerebral perfusion pressure, middle cerebral artery blood flow velocity, internal carotid artery blood flow velocity, autoregulatory index, and hemodynamic parameters (heart rate, arterial blood pressure, and central venous pressure or pulmonary artery wedge

Table 2. Summary of Physiologic Measures

Physiologic Measure	**Instrument**	**Measurement**
Heart rate (beats per minute)	Single Lead EKG/Spacelab Bedside Monitor	Continuous
SaO2 (%)	Pulse Oximeter/Spacelab Bedside Monitor	Continuous
Arterial Blood Pressure (mm Hg)	Arterial Catheter/Transducer or Nellcor Continuous Noninvasive Blood Pressure Monitor	Continuous
Intracranial Pressure (mm Hg)	Camino Fiberoptic Transducer-tip Catheter	Continuous
Cerebral Perfusion Pressure MAP-ICP (mm Hg)	Camino Fiberoptic transducer tip Catheter/Arterial Catheter/transducer or Nellcor Continous Noninvasive Blood Pressure Monitor	Continuous
Pulmonary Artery Pressure/Pulmonary Artery Wedge Pressure (mm Hg) at end expiration	Baxter-Edwards Swan Ganz (Pulmonary Artery) Catheter/Transducer; Space Lab Bedside Monitor	Continuous Intermittent
Central Venous Pressure (mm Hg) at end expiration	Cordis introducer or Arrow Triple Lumen Catheter/Transducer/Space Lab	Intermittent
MCA Flow Velocity (cm/sec), MCA/ICA Ratio, and ARI	Multi-Dop X4 Transcranial Doppler with headgear	Continuous
ICA Flow Velocity (cm/sec), MCA/ICA Ratio	Neuroguard Transcranial Doppler	Intermittent

pressure) and SaO2. Zero degrees was considered baseline. The order of elevation was fixed at 0, 20, 45, and 0 degrees since this sequence was a clinically feasible order and the return to 0 degrees may differentiate changes associated with time versus data changed associated with position changes. Therefore counterbalancing was not necessary. Each subject was only studied once at 0, 20, 45, and 0 degrees. No sign of neurologic deterioration occurred with any subject. Two potential subjects were determined to be in severe vasospasm at baseline, through moderate vasospasm earlier in the day. These individuals were not studied for at least another day and then only following a routine transcranial Doppler study indicating mild or moderate vasospasm.

Continuous recordings were obtained in each position for 2 to 5 minutes after allowing approximately 2 minutes for the subject to stabilize in each position and verification of correct placement and interrater agreement of head of bed position, transducers and TCD probes. A stopwatch was used. The study procedure took an additional 30-40 minutes, including positioning and data collection. Visitors were typically not present during the study procedure. Any routine nursing procedures occurred during the study procedure were included in the data collection, including medications (e.g., analgesics, nimodipine) or intravenous fluids (e.g. normal saline or albumin) that might have altered blood pressure, systemic hemodynamics, and/or intracranial dynamics.

Data Analysis

Descriptive and inferential statistics were computed. The level of significance was set at 0.05. Data from each position change were used to address both aims of the study. In regard to Aim 1, determine the effect of head of bed elevations of 20 and 45 degrees in mild or moderate cerebral vasospasm following aneurysmal subarachnoid hemorrhage, intracranial pressure, cerebral perfusion pressure, middle cerebral artery flow velocity, and middle cerebral artery to internal carotid artery blood flow velocity change were determined at each of the four head of bed positions, 0, 20, 45, and 0 degrees. Mean, median, standard deviation, and range (minimum to maximum) right and left middle cerebral artery blood flow velocities and middle cerebral artery to internal carotid artery ratios for subjects at each of the four levels were compared. Ninety-five percent confidence intervals were constructed to determine if there were statistically significant differences in middle cerebral artery flow velocity and middle cerebral artery to internal carotid artery ratio between 0, 20, 45, and 0 degrees. A repeated measures analysis of variance was conducted to determine statistically significant within-subject changes in middle cerebral artery flow velocities and middle cerebral artery to internal carotid artery ratios as a group at each head position. Continuous data were recorded for 2 to 5 minutes. Hard copy trend recordings were used for statistical interpretation in regard to heart rate, SaO2, central venous pressure, and/or pulmonary artery wedge pressure, and internal carotid artery flow velocity. Data regarding middle cerebral artery flow velocity, intracranial pressure, arterial blood pressure, and autoregulation were available on floppy diskette (zip disk) from the Multi-Dop X4 transcranial Doppler. Internal carotid artery flow velocity was obtained from Neuroguard TCD hard copy recordings. Continuous and simultaneous data recording of middle cerebral arteries and internal carotid arteries was not possible due to interference in signal intensity and quality.

For Aim 2, describe mild or moderate vasospasm response to 20 and 45 degrees head of bed elevations with respect to such demographics as grade of subarachnoid hemorrhage, autoregulation status/index, and degree of vasospasm, subjects were grouped according to changes in severity of vasospasm, i.e., mild to moderate, mild to severe, moderate to severe. Data indicating increasing severity of vasospasm, increasing MCA flow velocity and/or MCA to ICA ratio, were analyzed for any grouping variables that might predict its incidence and presence.

Human Subjects

The University of Washington Human Subjects Committee approved this study in 1999 and yearly renewals were obtained in 2000 and 2001. Twenty-four subjects were recruited and enrolled in the study after providing written consent. Twenty eight additional subjects were recruited and they or their legal next of kin provided written consent but were not studied because they did not meet inclusion criteria or data collection was not possible during the time they met inclusion criteria. The data used for this study were collected from transcranial Doppler ultrasonography, bedside monitors, and medical records generated as part of the ongoing clinical monitoring and care. All data were maintained in coded form, and identified by code number generated for this study. Medical record numbers were maintained until all data were verified and destroyed, thus providing anonymity.

CHAPTER IV

RESULTS

Data were analyzed in regard to subjects, middle cerebral artery flow velocities and middle cerebral artery to internal carotid artery ratios, intracranial pressure and cerebral perfusion pressure, and other physiologic parameters. Individual and group responses to head of bed elevation were determined. Descriptive and inferential statistics were used.

Subjects

Twenty subjects were included in the study. Fifteen were women and five were men. Seventeen were Caucasian; two were Native American and one was Filipino. Thirteen were post-hemorrhage day 3 through 7 and seven were day 8 through 14. The age ranges of the subjects included 36 years through 67 years with one between 30 to 39; 10 between 40 to 49; six between 50 to 59; and three between 60-69 years. Thirteen subjects were Hunt-Hess grades 1 through 2; one was grade 3; four were grade 4 through 5; and two were not classified.

Statistical Analysis

Data were collected on 24 subjects. However four subjects were not included in the final statistical analysis. Two of the four excluded had data sets that were incomplete. The other two subjects exhibited mild vasospasm on their routine daily exam. However, later that day when pre-study transcranial Doppler studies were obtained, the subjects were found to be without vasospasm. Since these two individuals were thought to be particularly at risk for an increase in vasospasm with head of bed elevation, these two subjects were studied but vasospasm was not reproduced, and therefore not included in the statistical analysis. Middle cerebral artery (MCA) flow velocity and middle cerebral artery to internal carotid artery (MCA: ICA) ratio data were analyzed employing two different strategies. In both instances, right and left were analyzed separately. However, the first data analysis included all 20 right MCA flow velocities and all 20 right MCA: ICA ratios, and all 20 left MCA flow velocities and all 20 left MCA: ICA ratios. The second data analysis included only those vessels meeting vasospasm criteria, right MCA flow velocities 120 cm/sec or greater or right MCA: ICA ratios 3.0 or greater and only those left MCA flow velocities 120 cm/sec or greater or left MCA: ICA ratios 3.0 or greater.

Descriptive Data

Descriptive statistics, including mean, median, standard deviation, range (minimum to maximum), and confidence intervals were obtained for MCA flow velocities and MCA:ICA ratios as well as other physiologic parameters including ICP, CPP, heart rate, SaO2, MAP, CVP, and PAWP. A summary of the mean, median, standard deviation, range (minimum to maximum), and confidence intervals of MCA flow velocities and MCA:ICA ratios at each position are included in Tables 3 and 4.

Right and Left MCA Flow Velocities

The means for all right and all left MCA flow velocities were stable at each position, varying only ± 1-3 cm/sec. The mean for left MCA flow velocities equal to or greater than 120 cm/sec was also relatively stable at all four positions within ± 1-2 cm/sec. However, the mean for the elevated right MCA flow velocities varied as much as 7 cm/sec. Though small, the mean for 1) all right MCA flow velocities as a group and 2) all elevated right MCA flow velocities as a group decreased from the baseline 0 degrees to the second 0 degrees position. In contrast, the mean for all left MCA flow velocities and all elevated left MCA flow velocities increased from the baseline 0 to the second 0.

Table 3. Descriptive Statistics for Right and Left MCA Flow Velocities

Parameter	**Mean at 0, 20, 45, 0 degrees**	**Median at 0, 20, 45, 0 degrees**	**Standard Deviation at 0, 20, 45, 0 degrees**	**Range (Minimum to Maximum) at 0, 20, 45, 0 degrees**	**Confidence Intervals at 0, 20, 45 and 0 degrees**
Right MCA flow velocities (all)	120, 118, 118, 117	129, 125, 118, 124	34, 30, 34, 30	137 (33-170), 125 (41-166), 130 (40-170), 130 (38-168)	104-136, 104-132, 102-134, 102-131
Right MCA flow velocities ≥ 120 (cm/sec)	141, 136, 137, 134	140, 136, 140, 126	14, 14, 21, 15	45 (125-170), 48 (118-166), 65 (105-170), 54 (114-168)	133-149, 128-144, 124-149, 124-143
Left MCA flow velocities (all)	129, 130, 131, 131	133, 145, 138, 134	27, 27, 29, 31	107 (75-182), 85 (77-162), 93 (77-170) 111 (78-189)	116-142, 117-142, 117-144, 115-147
Left MCA flow velocities ≥ 120 cm/sec	140, 141, 142, 142	143, 146, 143, 138	17, 16, 17, 21	65 (117-182), 54 (108-162), 57 (113-170), 75 (114-189)	131-149, 132-149, 133-152, 131-153

The median for the right and left MCA flow velocities, total and those equal to or greater than 120 cm/sec, was less stable. The direction of the median, increase versus decrease, for all right and left MCA flow velocities varied through the four positions as much as 11-12 cm/sec. The direction for the median for right and left MCA flow velocities equal to or greater than 120 cm/sec varied slightly more through the four positions at 8-14 cm/sec. The left MCA flow velocity median was elevated at 20 degrees but then decreased at 45 degrees and the second 0 degrees. The right MCA flow velocity median was decreased at 20 and 45 degrees but increased with the second 0 degrees. The elevated left MCA flow velocity median was increased at 20 degrees, decreased at 45 degrees, and decreased at the second 0 degrees. The elevated right MCA flow velocity median was decreased at 20 degrees, increased at 45 degrees, and decreased at the second 0.

Neither the means nor medians for the right and left MCA flow velocities in any position exceeded 146 cm/sec, which is consistent with mild vasospasm. Individually, four subjects' right MCA flow velocities exceeded 150 cm/sec, moderate vasospasm criteria; however each of these four subjects, numbers 3, 10, 13, and 17, exceeded 150 cm/sec initially at the baseline 0 degrees. In contrast eight subjects, numbers 4, 6, 9, 12, 13, 14, 16, and 19, exhibited left MCA flow velocities equal to or exceeding 150 cm/sec but only four subjects, numbers 6, 9, 13, and 16, met moderate vasospasm criteria at baseline 0 degrees.

The standard deviation for right and left MCA flow velocities, all and those equal or exceeding 120 cm/sec, stabilized for two to three positions, especially 0 degrees and

20 degrees. The standard deviation at the second 0 degrees did not agree with the standard deviation at the baseline 0 degrees. The difference in the range of flow velocity varied up to 7 cm/sec in all right MCA vessels but up to 20 cm/sec in right MCA vessels equal or exceeding 120 cm/sec; up to 26 cm/sec in all left MCA vessels; and up to 21 cm/sec in left MCA vessels equal or exceeding 120 cm/sec.

As expected, the 95 % confidence intervals for right and left MCA flow velocities were greatest when only right and left middle cerebral artery flow velocities equal to or exceeding 120 cm/sec were considered. The upper limits for the 95% confidence level for elevated left MCA flow velocities did exceed 149 cm/sec twice, at 152 and 153 cm/sec, which is consistent with moderate vasospasm. The highest upper limits for confidence intervals were obtained at the baseline 0 in all right MCA flow velocities and at the second 0 in all left MCA flow velocities. The upper limits of the confidence intervals for the right MCA flow velocities equal to or exceeding 120 cm/sec were highest at the baseline 0 degrees and 45 degrees. The upper limits of the confidence intervals for elevated left MCA flow velocities exceeding 120 cm/sec were highest at 45 degrees and the second 0. All right and left MCA flow velocities, elevated right and left MCA flow velocities and elevated right and left MCA flow velocities with MCA:ICA ratios of 3.00 or greater are illustrated graphically (Figures 1-6).

Right and Left MCA to ICA Ratios

The mean for all right and all left MCA to ICA ratios varied from 3.00 to 3.82. This ratio never met severe criteria, equal to or greater than 6.00. The greatest mean

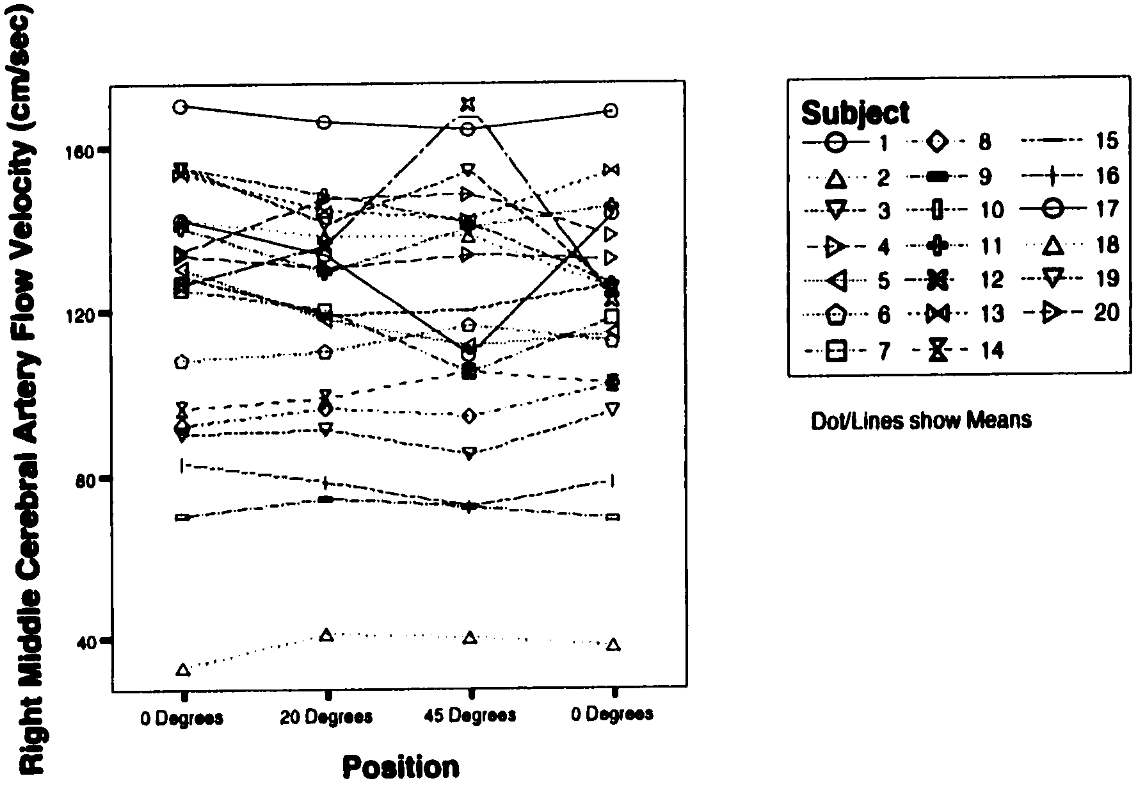


Figure 1. All Right Middle Cerebral Artery Flow Velocities at 0, 20, 45 and 0 Degrees

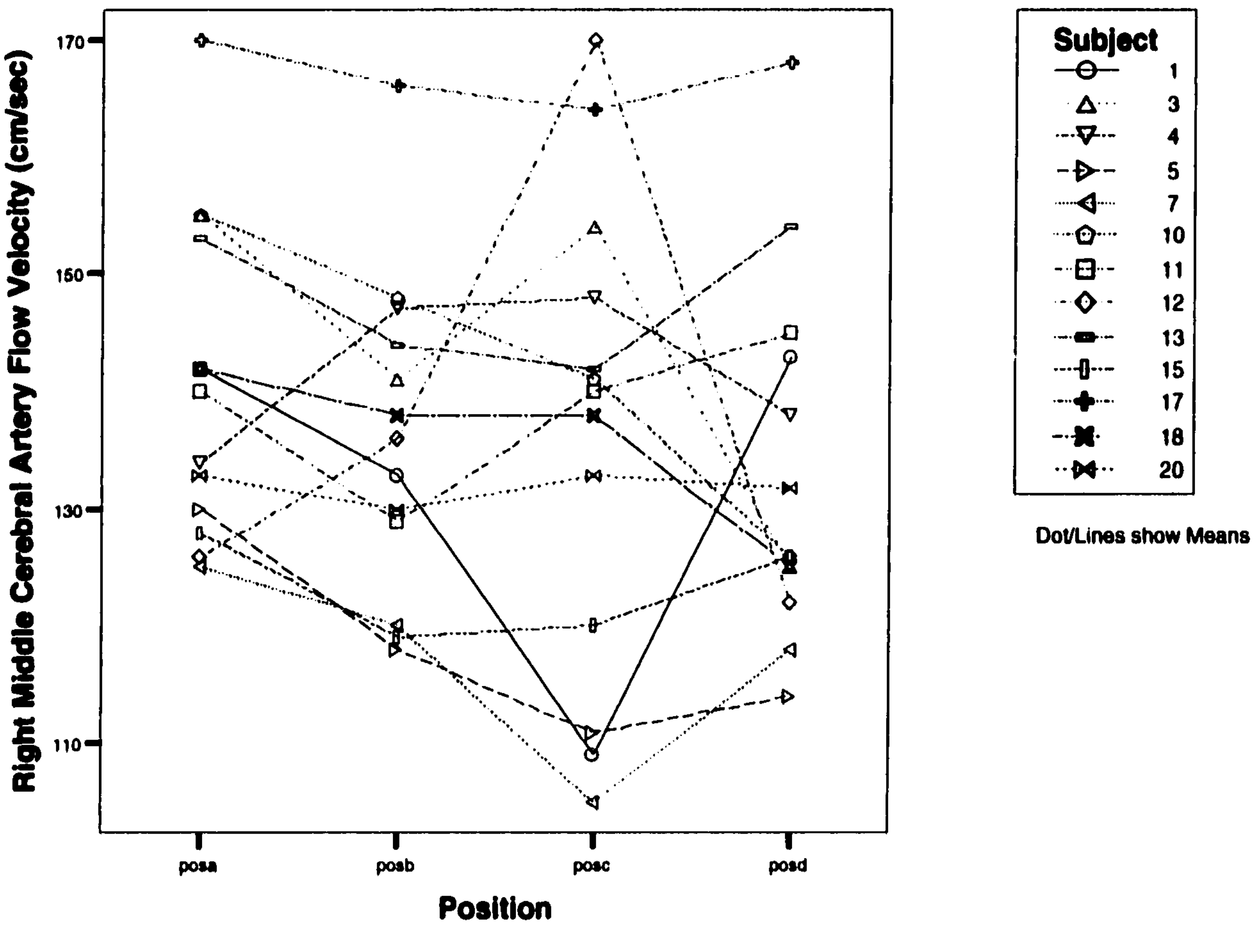


Figure 2. Elevated Right Middle Cerebral Artery Flow Velocities at 0, 20, 45, and 0 Degrees

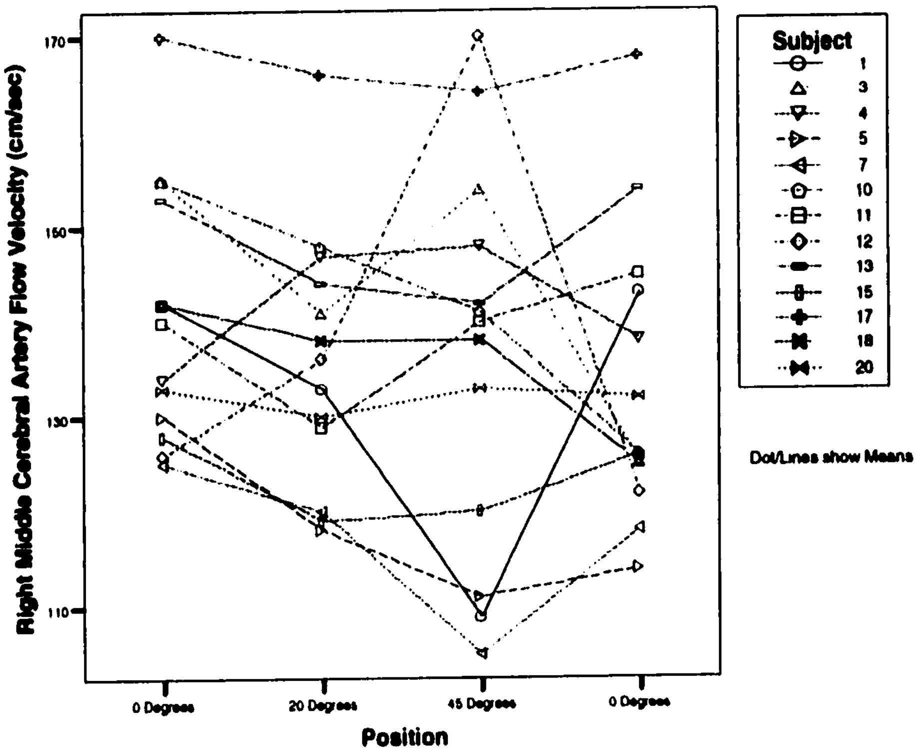


Figure 3. Elevated Right Middle Cerebral Artery Flow Velocities with Right Middle Cerebral Artery to Right Internal Carotid Artery Ratios of 3.00 or Greater at 0, 20, 45, and 0 Degrees

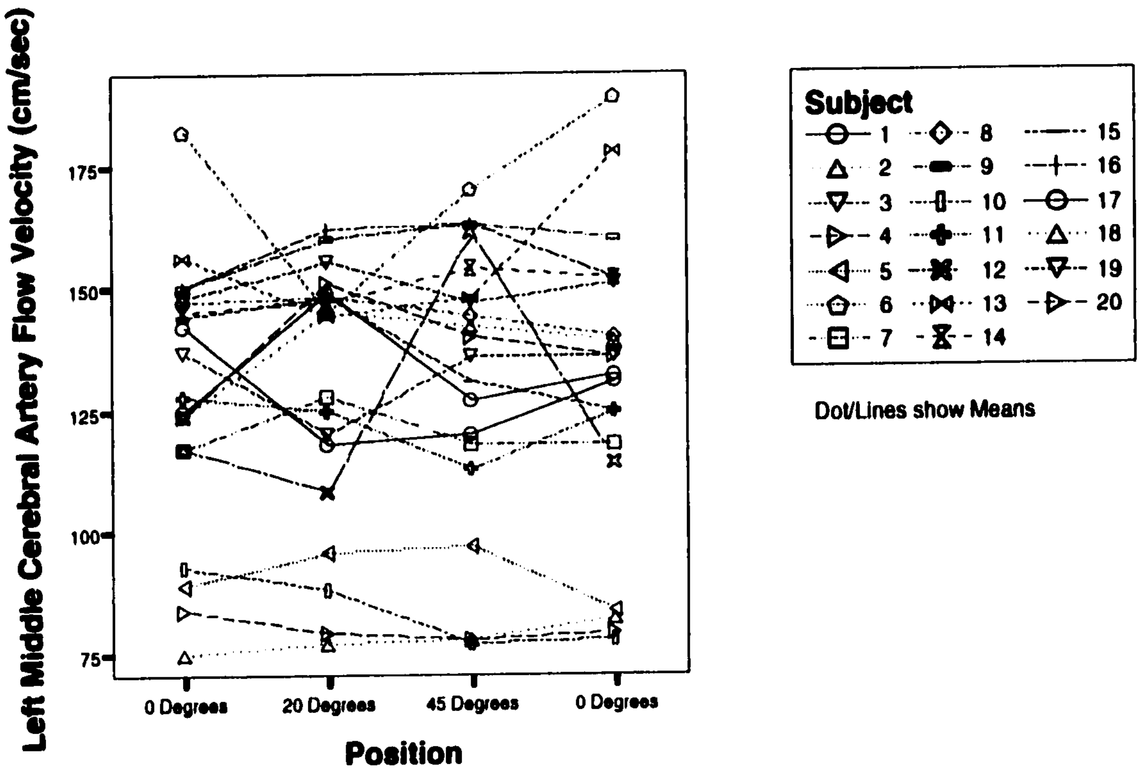


Figure 4. All Left Middle Cerebral Artery Flow Velocities at 0, 20, 45 and 0 Degrees

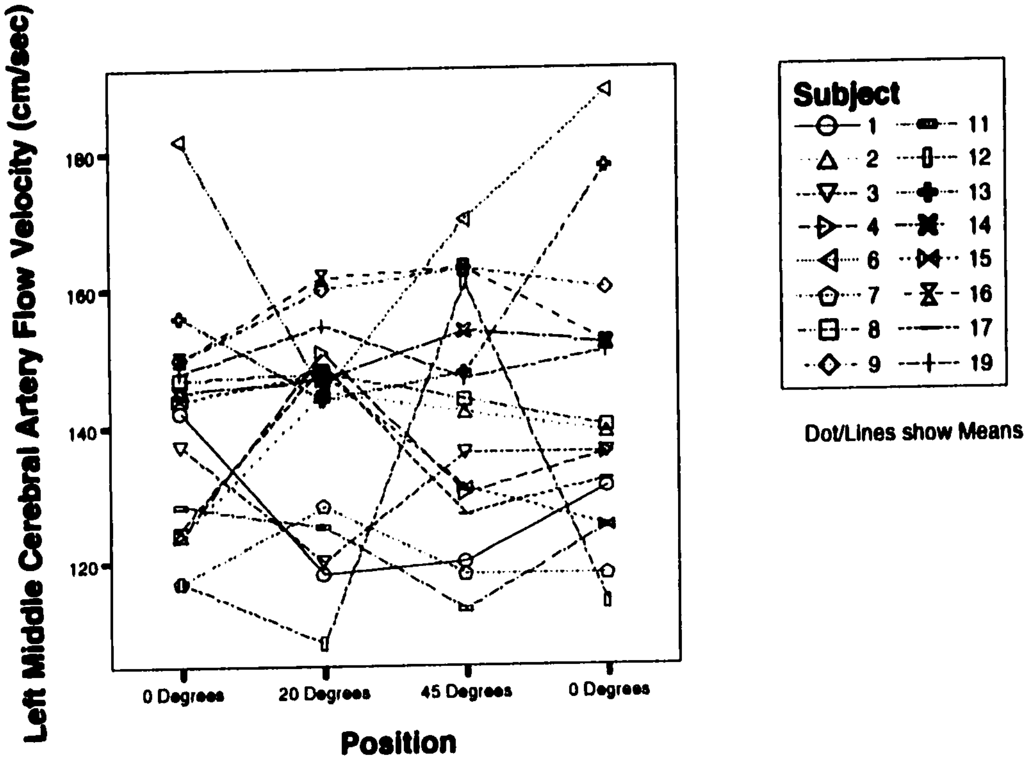


Figure 5. Elevated Left Middle Cerebral Artery Flow Velocities at 0, 20, 45 and 0 Degrees

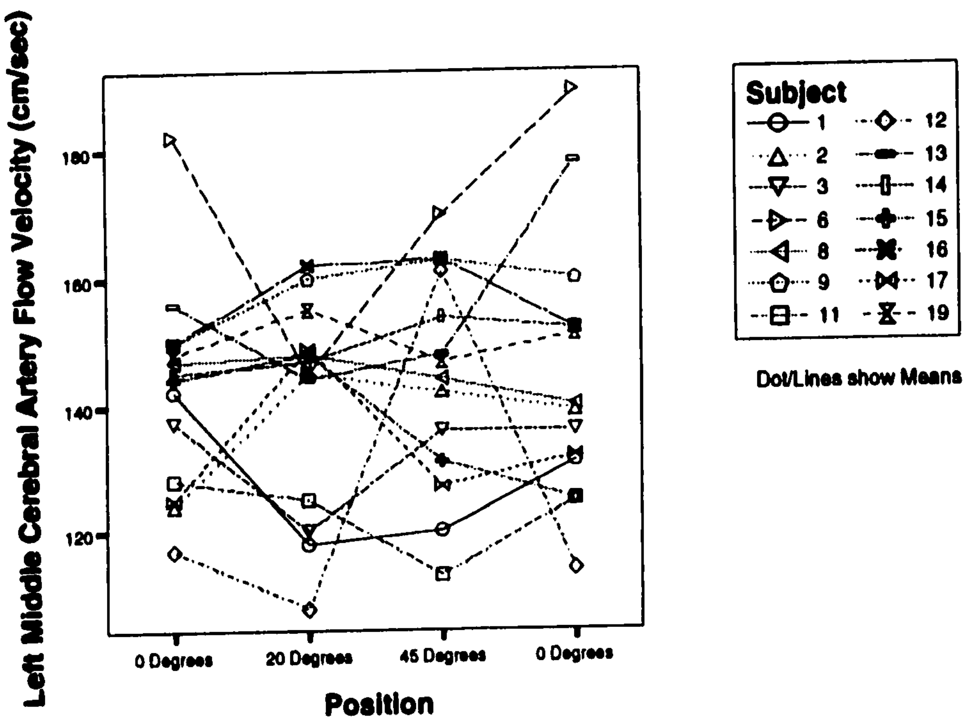


Figure 6. Elevated Left Middle Cerebral Artery Flow Velocities with Left Middle Cerebral Artery to Left Internal Carotid Artery Ratios of 3.00 or Greater at 0, 20, 45, and 0 Degrees

was noted at 20 degrees for all right MCA to ICA ratios; at 20 degrees for right MCA to ICA ratios equal to or exceeding 3.00; and at 45 degrees for all left MCA to ICA ratio; and at 45 degrees for left MCA to ICA ratios equal to or exceeding 3.00. The greatest difference for the mean of all right MCA to ICA ratios at all four positions was 0.09; however for all left MCA to ICA ratios the greatest difference was 0.15. For right and left ratios equal to or greater than 3.00, the greatest difference was 0.15-0.16.An alternating increase and decrease in ratio was noted in the ratios' means as subjects were studied at the 0, 20, 45 and 0 sequence.

In contrast, the median ratio was as low as 2.83 and 2.99 in all right and all left MCA to ICA ratios respectively, which is less than mild vasospasm criteria. No trend was noted in regard to increases or decreases in the median in regard to ratios. Individually, only one subject, number 12, reached a ratio of equal to or greater than 5.00 (5.15) on the right. However, he was at an even slightly higher ratio of 5.33 at baseline 0 degrees.

In regards to measures of variability, the ratios' differences in standard deviation in all four positions fluctuated as much as 0.05 and 0.21, all right and all left MCA: ICA ratios respectively. The difference in the standard deviation at all four positions for the right MCA to ICA ratios equal to or exceeding 3.00; all left MCA to ICA ratio; and all left MCA to ICA ratios equal to or greater than 3.00 ranged from 0.20 to 0.50. The range for the ratios was maximal at 45 degrees when all right and left MCA to ICA ratios and left MCA to ICA ratios equal to or exceeding 3.00 were considered. The range was maximal for elevated right MCA:ICA ratios at the second 0 degrees.

As expected, not unlike the confidence intervals for MCA flow velocities, the upper limits for 95% confidence intervals were greatest when only right and left ratios meeting vasospasm criteria were considered at upper boundaries of 4.21 and 4.09, right and left respectively. All right MCA:ICA ratios and all elevated right MCA:ICA ratio confidence interval upper boundaries were similar at the baseline and the second zero. The upper limits of the MCA to ICA ratio confidence intervals for all right and the elevated right MCA:ICA ratios were greatest at 20 degrees. The upper limits of all left MCA:ICA ratio and elevated left MCA:ICA confidence intervals were greatest at 45 degrees (Table 4). All right and left and the elevated right and left MCA: ICA ratios are illustrated in Figures 7-10.

Percentage Change from Baseline

The percentage change from baseline was calculated for 0 to 20 degrees, 0 to 45 degrees, and 0 to 0 degrees for all right and left MCA flow velocities and all right and left MCA:ICA ratios. The results are summarized in Tables 5 and 6. The most frequent increase from baseline for both right and left flow velocities and right and left MCA:ICA ratios was greater than 1 percent but less than 10 percent. Forty-one to 55% of all subjects' right and left MCA flow velocities at each position were elevated but only three subjects, numbers 2, 4, and 12, were elevated 20.00% of more. The second subject's right MCA flow velocities were elevated at a 24.24% and 21.21% increase over baseline 0 at 20 and 45 degrees; the fourth subject's left MCA flow velocity was elevated 21.77% from baseline 0 to 20 degrees; and the twelfth subject's right and left MCA flow velocities were elevated at a 26.98% and 37.61% increase

Table 4. Descriptive Statistics for Right and Left MCA to ICA Ratios

Parameter	**Mean at 0, 20, 45, 0 degrees**	**Median at 0, 20, 45, 0 degrees**	**Standard Deviation at 0, 20, 45, 0 degrees**	**Range (minimum to maximum) at 0, 20, 45, 0 degrees**	**Confidence Intervals at 0, 20, 45, 0 degrees**
Right MCA:ICA Ratios (all)	3.08, 3.14, 3.05, 3.10	3.18, 3.16, 3.01, 2.83	1.13, 1.11, 1.10, 1.08	4.43 (.90-5.33), 3.76 (.95-4.71), 4.56 (.62-5.15), 3.47 (1.62-5.09)	2.55-3.61, 2.62-3.66, 2.54-3.56, 2.59-3.61
Right MCA:ICA Ratios ≥ 3.0	3.75, 3.82, 3.66, 3.69	3.52, 3.92, 3.76, 3.67	0.74, 0.64, 0.75, 0.84	2.17 (3.16-5.33), 1.81 (2.90-4.71), 2.59 (2.56-5.15), 2.67 (2.42-5.09)	3.31-4.20, 3.44-4.21, 3.21-4.11, 3.18-4.20
Left MCA:ICA Ratios (all)	3.13, 3.00, 3.15, 3.01	3.38, 2.99, 3.24, 3.18	0.82, 0.91, 1.03, 0.90	2.73 (1.68-4.41), 3.34 (1.43-4.77), 3.39 (1.35-4.74), 2.87 (1.44-4.31)	2.74-3.51, 2.58-3.43, 2.66-3.63, 2.59-3.43
Left MCA:ICA Ratios ≥ 3.0	3.58, 3.56, 3.65, 3.49	3.60, 3.32, 3.76, 3.47	0.46, 0.63, 0.76 0.26	1.71 (2.70-4.41), 2.16 (2.61-4.77), 2.69 (2.05-4.74), 1.58 (2.73-4.31)	3.31-3.84, 3.09-3.82, 3.21-4.09, 3.20-3.78

respectively over baseline at 45 degrees.

Similarly, an increase in left and right MCA:ICA ratios occurred 40 to 52% of the time, respectively. Nine different subjects, numbers 2, 3, 4, 12, and 16 (left), and subjects, numbers 1, 3, 4, 5, 6, and 7 (right), exhibited 20% or greater increases in ratios. Four subjects, numbers 1, 3, 4, and 16, experienced greater than 20% increase at varying degrees but less than 30%; five subjects, numbers 2, 4, 5, 6, and 7, experienced greater than 30% increase but less than 40% at varying degrees; and two subjects, numbers 5 and 12, exhibited greater than 40% but less than 50% increases at

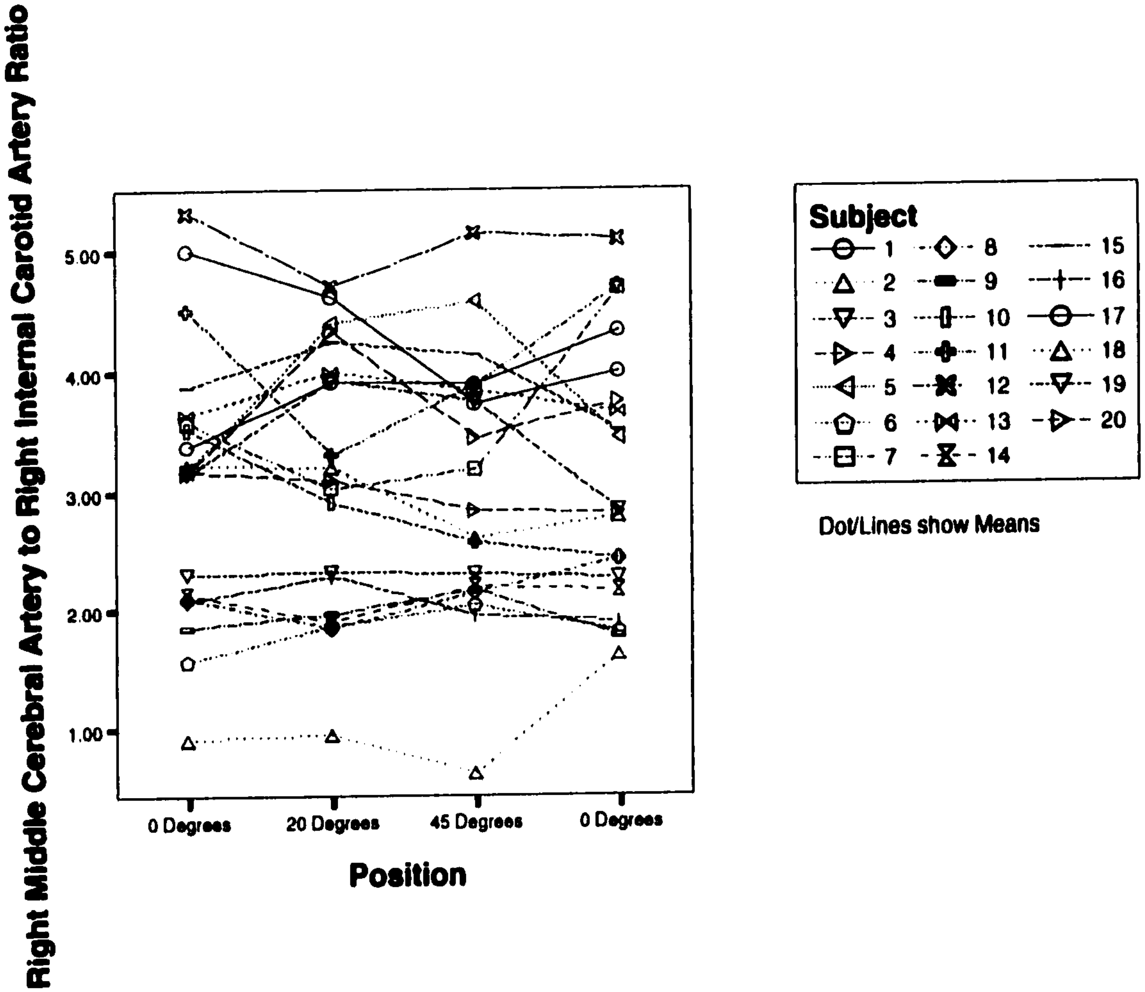


Figure 7. All Right Middle Cerebral Artery to Right Internal Carotid Artery Ratios at 0, 20, 45 and 0 Degrees

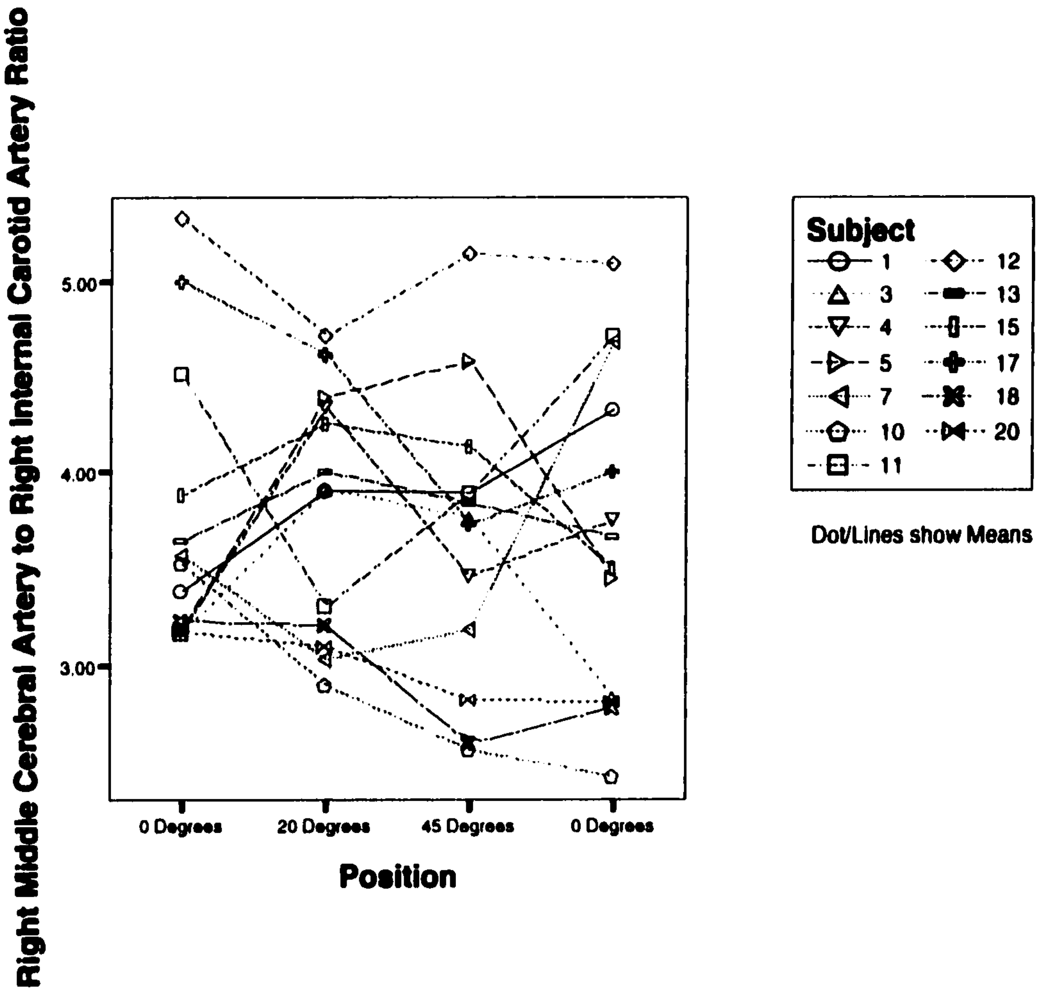


Figure 8. Elevated Right Middle Cerebral Artery to Right Internal Carotid Artery Ratios at 0, 20, 45 and 0 Degrees

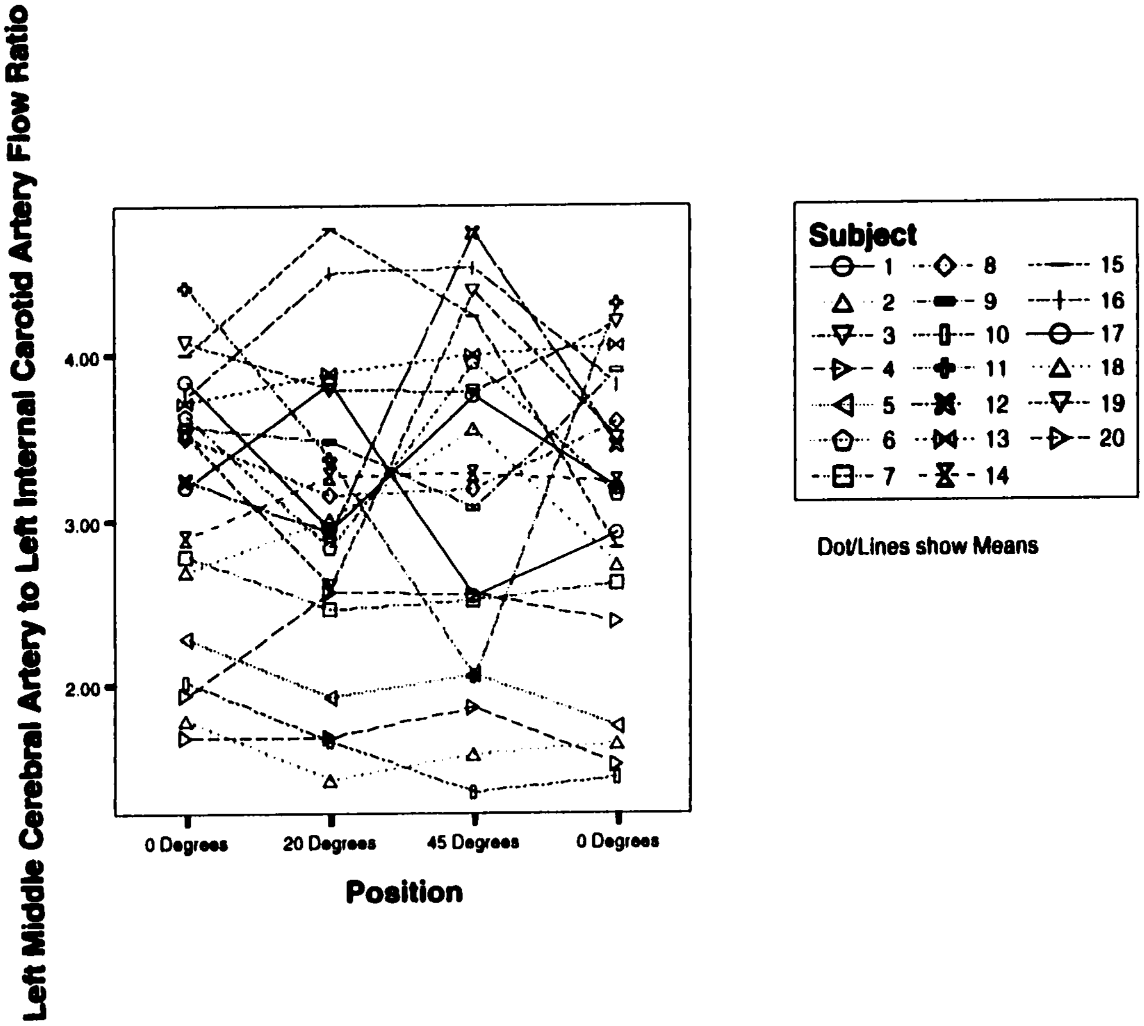


Figure 9. All Left Middle Cerebral Artery to Left Internal Carotid Artery Ratios at 0, 20, 45 and 0 Degrees

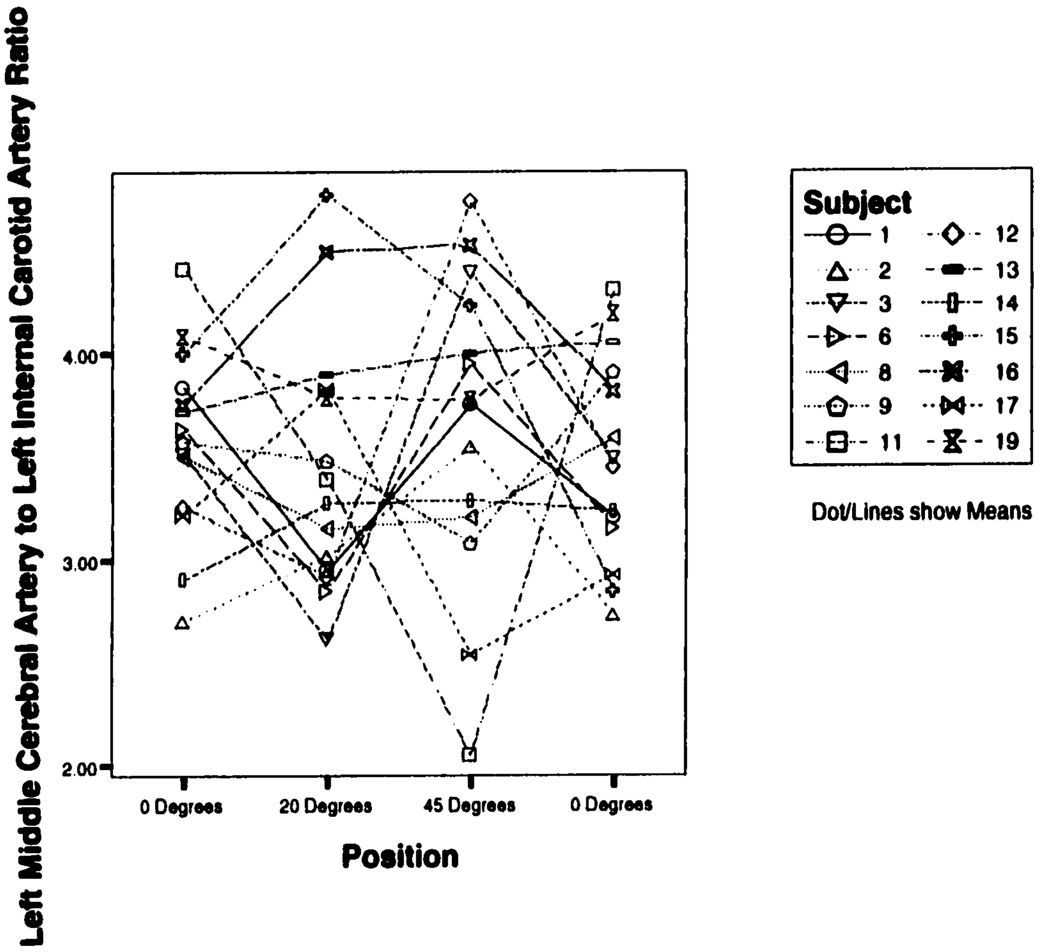


Figure 10. Elevated Left Middle Cerebral Artery to Left Internal Carotid Artery Ratios at 0, 20, 45 and 0 Degrees

Table 5. Percentage Change (Increase or Decrease) from Baseline 0 for Right and Left MCA Flow Velocities

Percentage Change	**Number of subjects from 0 to 20 degrees**		**Number of subjects from 0 to 45 degrees**		**Number of subjects from 0 to Second 0**	
	Right	**Left**	**Right**	**Left**	**Right**	**Left**
No change	0	0	2	0	0	0
Increase < 1%	0	1	0	1	2	1
Decrease < 1%	0	0	1	2	1	1
Increase > 1 to 10%	7	8	5	6	5	7
Decrease >1 to 10%	12	5	6	5	7	6
Increase > 10 to 20%	0	2	1	2	2	2
Decrease > 10 to 20%	0	2	3	3	3	2
Increase > 20 to 30%	1	1	2	0	0	0
Decrease > 20 to 30%	0	1	0	0	0	1
Increase > 30 to 40%	0	0	0	1	0	0
Decrease > 30 to 40%	0	0	0	0	0	0
Increase > 40 to 50%	0	0	0	0	0	0
Decrease > 40 to 50%	0	0	0	0	0	0

Table 6. Percentage Change from Baseline 0 for Right and Left

MCA to ICA Ratios

Percentage Change	**Number of subjects from 0 to 20 degrees**		**Number of subjects from 0 to 45 degrees**		**Number of subjects from 0 to Second 0**	
	Right	**Left**	**Right**	**Left**	**Right**	**Left**
No change	0	1	0	0	0	0
Increase < 1%	1	1	0	0	1	0
Decrease < 1%	1	0	1	0	0	1
Increase > 1 to 10%	4	1	5	3	3	7
Decrease >1 to 10%	2	1	3	4	6	5
Increase > 10 to 20%	3	4	3	2	3	2
Decrease > 10 to 20%	5	4	4	3	1	1
Increase > 20 to 30%	1	1	0	2	1	1
Decrease > 20 to 30%	1	5	1	1	0	3
Increase > 30 to 40%	2	1	1	2	1	0
Decrease > 30 to 40%	0	0	1	1	1	0
Increase > 40 to 50%	0	0	1	1	0	0
Decrease > 40 to 50%	0	0	0	0	0	0
Increase > 50 to 60%	0	0	0	0	0	0
Decrease > 50 to 60%	0	0	0	1	0	0
Increase > 60%					1 (80%)	0

45 degrees. The 80% increase from the first to the second 0 in regard to the right MCA to ICA ratios in subject number 2 was not important clinically, with the actual numbers being .90 to 1.62.

Inferential Data

MCA Flow Velocities and MCA to ICA Ratios

Repeated measures analysis of variance was conducted in regard to MCA flow velocities and MCA: ICA ratios. As a group, neither the right and left MCA flow velocities, nor the right and left MCA:ICA ratios through the four positions of 0-20-45-0 yielded statistically significant within-subjects findings ($p \leq 0.05$). Furthermore, as a group, neither the elevated right and left MCA flow velocities nor the elevated right and left MCA:ICA ratios through the four positions of 0-20-45-0 yielded statistically significant within-subjects findings ($p \leq 0.05$). Table 7 summarizes the results of the repeated measures analysis of variance within-subjects effects for all right and left flow velocities; all right and left MCA to ICA ratios; elevated right and left flow velocities; and elevated right and left MCA:ICA ratios.

Intracranial Pressure and Cerebral Perfusion Pressure

Descriptive and Inferential Data

Following analysis of the MCA flow velocities and the MCA:ICA ratios, the intracranial pressure and cerebral perfusion pressure data were examined.
As expected, the intracranial pressure consistently decreased with head of bed elevation and then increased when returned to the second zero. The mean ICP for each

of the four positions consecutively were: 17.92; 11.25; 9.08, and 16.50 with the medians being 15.50, 9.50, 8,00, and 15.50 (Table 8). As a group, the repeated measures analysis of variance of the intracranial pressure at 0-20-45-0 degrees yielded statistically significant results within-subjects effects at $p = 0.000$ (Table 9).

Table 7. Repeated Measures Analysis of Variance for Right and Left MCA Flow Velocities and Left MCA to ICA Ratios (Within-Subjects Effects) ($p \leq 0.05$)

Parameter	**F** **(df)**	**Significance**
All Right MCA Flow Velocities	.589 (3, 57)	.625
Right MCA Flow Velocities ≥ 120 cm/sec	1.149 (3, 36)	.343
All Left MCA Flow Velocities	.075 (3, 57)	.973
Left MCA Flow Velocities ≥ 120 cm/sec	.193 (3, 45)	.901
All Right MCA to ICA Ratios	.165 (3, 57)	.919
Right MCA to ICA Ratios ≥ 3.0	.261 (3, 36)	.853
All Left MCA to ICA Ratios	.482 (3, 57)	.696
Left MCA to ICA Ratios ≥ 3.0	.318 (3, 39)	.812

A function of the mean arterial pressure and the intracranial pressure, MAP-ICP, the cerebral perfusion pressure (CPP) was statistically variable depending on the method of measurement. The mean arterial pressure was obtained in one of three ways: arterial line with the transducer at the level of the heart, phlebostatic axis level; arterial line with the transducer at the head, at the level of the external auditory

Table 8. Descriptive Statistics for Intracranial Pressure and Cerebral Perfusion Pressure

Parameter	**Mean at 0, 20, 45, 0 degrees**	**Median at 0, 20, 45, 0 degrees**	**Standard Deviation at 0, 20, 45, 0 degrees**	**Range (Minimum to Maximum) at 0, 20, 45, 0 degrees**	**Confidence Intervals at 0, 20, 45 and 0 degrees**
Intracranial Pressure (mm Hg)	18, 11, 9, 17	16, 10, 8, 16	7, 7, 38, 31	24 (11-35), 24 (5-29), 19 (2-21), 18 (10-28)	14-22, 7-17, 5-13, 13-20
Cerebral Perfusion Pressure at Heart (Phlebostatic Axis) (mm Hg)	84, 91 99, 88	83, 90, 97, 85	12, 16, 18, 16	43 (66-109), 50 (65-115), 57 (68-125), 49 (64-113)	74-92, 78-104, 84-114, 75-101
Cerebral Perfusion Pressure at Head (External Auditory Meatus) (mm Hg)	82, 85, 91, 85	81, 85, 86, 85	11, 16, 25. 15	40 (61-101), 49 (57-106) 78 (60-138), 51 (58-109)	73-91. 72-99, 70-112. 70-112
Cerebral Perfusion Pressure with Continuous Noninvasive Blood Pressure Monitor (Cuff) (mm Hg)	83, 92, 93, 90	83, 94, 92, 89	8, 12, 10, 12	18 (74-92), 29 (76-105), 24 (82-106), 28 (77-105)	71-95, 73-111, 76-110 71-109

meatus; or with a continuous noninvasive tonometric cuff with the arm at heart level. In individuals with an arterial line, mean arterial pressure at the phlebostatic axis level was consistently obtained before determining the mean arterial pressure at the level of the external auditory meatus. For each mean arterial pressure determination and at each head position, 0, 20, 45, and 0 degrees, the transducer was zeroed and leveled with interrater reliability by verbal agreement with experienced critical care nurses when present and/or the sonographers.

Repeated measures analysis of variance were again employed for inferential analysis. When the mean arterial pressure was measured at the phlebostatic axis, as a group, the cerebral perfusion pressure within-subjects effects were statistically significant with $p= 0.00$. However, when mean arterial pressure was measured at the head at the external auditory meatus , the cerebral perfusion pressure within-subjects effects were not significant with $p = 0.282$ on repeated measures. When the mean arterial pressure was measured with a cuff, at heart level, the cerebral perfusion pressure was also not statistically significant yielding a $p = 0.169$. Figures 11-14 illustrate the change in ICP and CPP at 0, 20, 45, and 0 degrees.

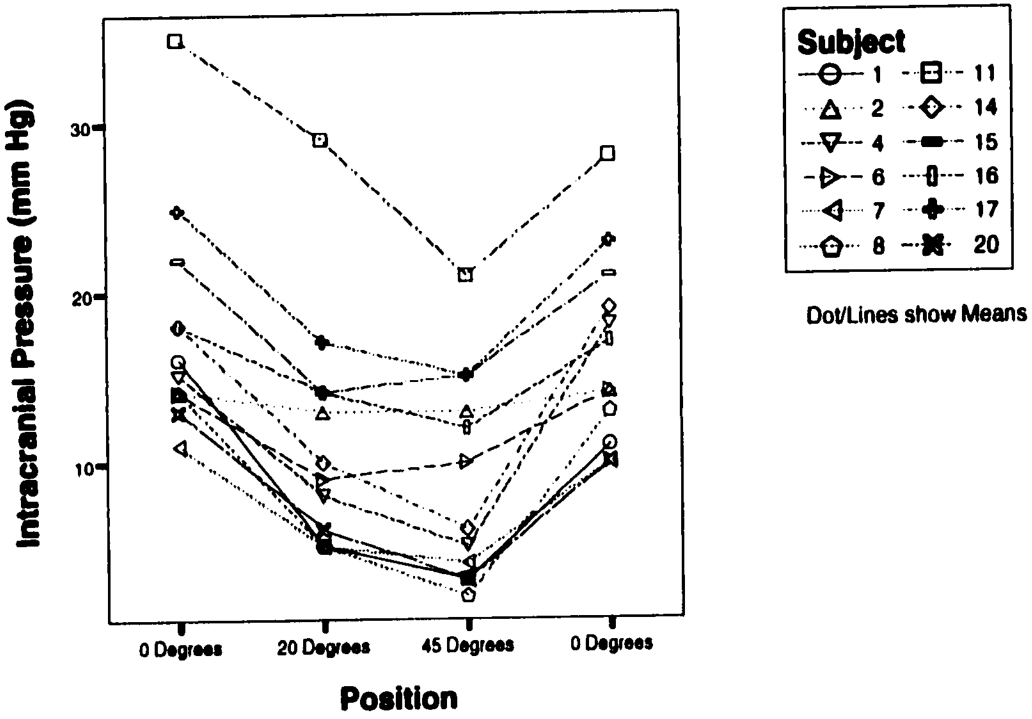


Figure 11. Intracranial Pressure at 0, 20, 45 and 0 Degrees

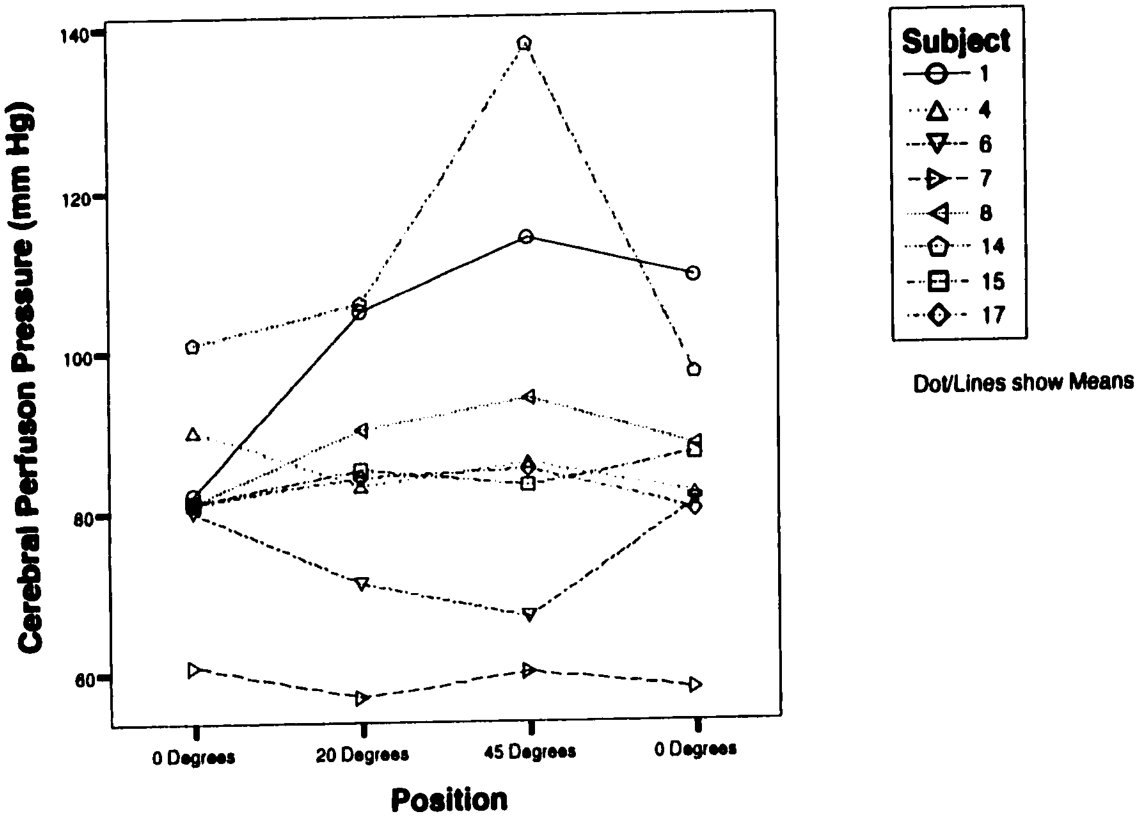
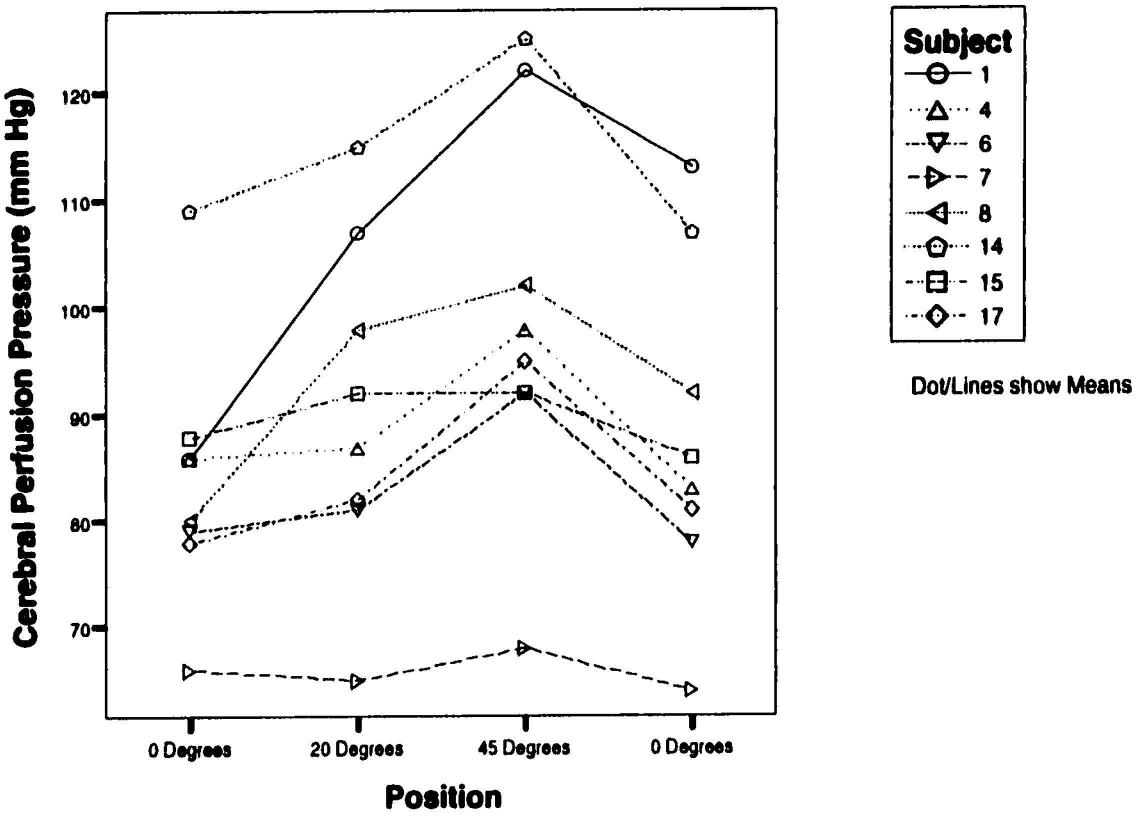


Figure 12. Cerebral Perfusion Pressure with Transducer at Head (External Auditory Meatus) at 0, 20, 45 and 0 Degrees



Dot/Lines show Means

Figure 13. Cerebral Perfusion Pressure with Transducer at Heart (Phlebostatic Axis) at 0, 20, 45 and 0 Degrees

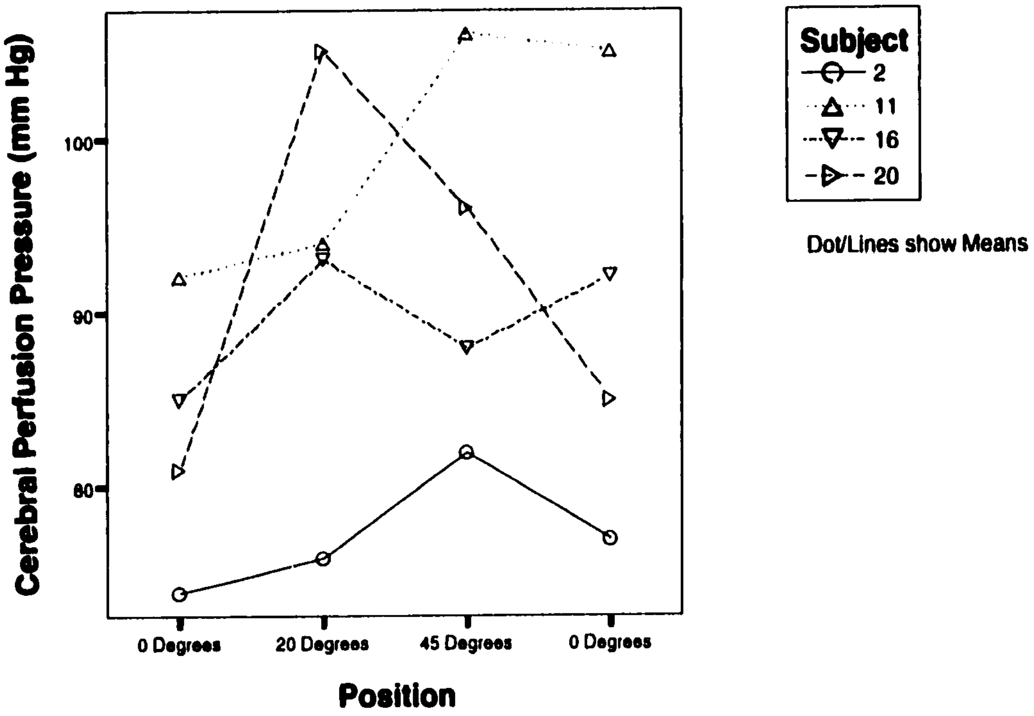


Figure 14. Cerebral Perfusion Pressure with Continuous Noninvasive Blood Pressure Monitor at 0, 20, 45 and 0 Degrees

Table 9. Repeated Measures Analysis of Variance for Intracranial Pressure and Cerebral Perfusion Pressure (Within-Subjects Effects)

($p \leq .05$)

Parameter	**F** **(df)**	**Significance**
Intracranial Pressure	44.080 (3, 33)	.000
Cerebral Perfusion Pressure at Heart (Phlebostatic Axis)	11.091 (3, 21)	.000
Cerebral Perfusion Pressure at Head (External Auditory Meatus)	1.362 (3, 21)	.282
Cerebral Perfusion Pressure with Continuous Noninvasive Blood Pressure Monitor (Cuff)	2.108 (3, 9)	.169

Hemodynamic Parameters

Descriptive and Inferential Data

Descriptive and inferential statistics were also used to analyze all other physiologic parameters. The means, medians, standard deviations, ranges (minimum to maximum), and confidence intervals were all relatively stable at all four positions (Table 10).

On repeated measures analysis of variance, heart rate, SaO2, MAP at head and MAP with cuff, CVP, and PWP as a group were not statistically significant. However, the within-subjects effect for the MAP measured at the phleobstatic axis (with an arterial catheter) was statistically significant at 0.01. As stated previously, the cerebral perfusion

Table 10. Descriptive Statistics for Hemodynamic Parameters

Parameter	**Mean at 0, 20, 45, 0 degrees**	**Median at 0, 20, 45, 0 degrees**	**Standard Deviation at 0, 20, 45, 0 degrees**	**Range (Minimum to Maximum) at 0, 20, 45, 0 degrees**	**Confidence Intervals at 0, 20, 45 and 0 degrees**
Heart Rate (beats per minute)	88, 87, 89, 88,	88, 87, 90, 87	16, 19, 22, 18	61 (59-120), 71 (55-126), 69 (56-125), 67 (57-124)	81-96, 78-96, 79-99, 79-96
Oxygen Saturation (%)	97, 97, 97, 97	97, 98, 98, 97	2, 2, 2, 2	7 (93-100), 7 (93-100), 8 (92-100), 8 (920100)	96-98, 96-98, 96-98, 96-98
Mean Arterial Pressure at Heart (mm Hg)	101, 101, 107, 104	102, 101, 106, 105,	14, 16 17, 16,	50 (77-127), 55 (70-125), 59 (72-131), 52 (74-126)	91-110, 89-112, 94-119, 92-115
Mean Arterial Pressure at Head (mm Hg)	100, 97, 101, 103	101, 99, 100, 103	14, 17, 23, 16	47 (72-119), 54 (62-116), 80 (64-144), 52 (68-120)	90-111, 84-110, 83-118, 91-115
Mean Arterial Pressure with Cuff (mm Hg)	110, 114, 115, 114	109, 115, 114, 111	16, 13, 19, 17	52 (88-140), 42 (89-131), 53 (94-147), 53 (91-144)	98-121, 104-124, 102-129, 102-126
Central Venous Pressure (mm Hg)	9, 9, 9, 9	10, 9, 8, 10	3, 3, 3, 3	12 (4-16), 11 (5-16), 11 (5-16), 10 (5-15)	6-11, 6-11, 7-11, 7-11
Pulmonary Artery Wedge Pressure (mm Hg)	16, 16, 17, 16	18, 16, 18, 18	2, 2, 2, 2	6 (12-18), 5 (13-18), 6 (14-20), 4 (14-18)	14-19, 14-17, 15-19, 15-18

pressure when MAP was measured at the heart was also statistically significant and cerebral perfusion pressure is a function of MAP-ICP (Table 11). (See Appendix A for hemodynamic parameters.)

Table 11. Repeated Measures Analysis of Variance for Hemodynamic Parameters

(Within-Subjects Effects)

($p \leq 0.05$)

Parameter	**F** **(df)**	**Significance**
Heart Rate	.324 (3, 57)	.808
Oxygen Saturation	.583 (3, 57)	.629
Mean Arterial Pressure at Heart (Phlebostatic Axis)	4.603 (3, 27)	.010
Mean Arterial Pressure at Head (External Auditory Meatus)	1.337 (3, 24)	.286
Continuous Noninvasive Mean Arterial Pressure with Cuff	1.941 (3, 27)	.147
Central Venous Pressure	.442 (3, 30)	.725
Pulmonary Artery Wedge Pressure	1.477 (3, 18)	.254

CHAPTER V

DISCUSSION

Changes in head of bed position from 0, 20, 45, and 0 degrees in aneurysmal subarachnoid hemorrhages did not result in statistically significant within-subjects increases or decreases in cerebral vasospasm in this sample as a group. However, other information obtained from this study warrants further discussion in regard to the subjects, physiologic effects of head of bed elevation, factors affecting cerebral blood flow, the specific aims of the study, and technology. Limitations, recommendations, and implications for nursing practice and future nursing research will be discussed as well.

Subjects

Of the 20 subjects included in the study, 15 were women and 5 were men. As past and more recent literature reports, more women than men experience aneurysmal subarachnoid hemorrhage. In 1993, Broderick, Brott, Tomsick, and others reported a female to male ratio of 1.7 to 1. In 1996, Kongable, Lanzino, Germanson, and others reported a ratio approaching 2 to 1 in a sample of 906 subjects with aneurysmal subarachnoid hemorrhage. This sample's female to male ratio was 3 to 1. The mean age was 50 years which is slightly younger than the mean age, 57 years, noted by Brown, Whisnant, Sicks, and others in 1996. Sixty-five percent were post-aneurysmal subarachnoid hemorrhage day 3 through 7 while 35 percent were day 8 through 14. While vasospasm typically starts after day 3 post aneurysmal subarachnoid hemorrhage an increase in severity is reported around days 7 through 10.

Based on positive intake and output balances and CVP or PAWP measurements, all patients were euvolemic to hypervolemic at the time of the study. All were receiving hourly fluid rates of at least 150 to 200 ml/hr; five were receiving fluid boluses based on CVP or PAWP and three were receiving phenylephrine infusions. While the effectiveness of hypervolemic hemodilution with or without controlled hypertension is controversial in the management of vasospasm (Oropello, Weiner, & Benjamin, 1996; Ullman & Bederson, 1996), this treatment regimen may have altered, i.e. blunted, the vasospastic response to head elevation. All study subjects also received routine nimodipine beginning at admission. This cerebroselective pharmacologic agent may have enhanced circulation (Hauerberg, Rasmussen, Juhler, et al., 1995; Feigin, Rinkel, Algra, et al., 2000). Four subjects, numbers 5, 14, 15, and 19, were post-angioplasty. Two received MCA angioplasty for previously clinically significant and/or severe vasospasm and two received MCA angioplasty prophylactically as part of a multi-center study. The previously angioplastied vessel with or without intra-arterial injection of papaverine may have responded differently to head of bed elevation in the presence of recurrent vasospasm. Angioplasty for aneurysmal subarachnoid hemorrhage vasospasm is associated with morphological changes in cerebral arteries (Honma, Fujiwara, Irie, et al., 1995). However, when compared to the other study subjects, no differences were noted in the angioplastied subjects' responses to the head of bed elevation at 20 and 45 degrees. Study subjects on bedrest greater than 7 days did not respond differently from those on a less lengthy period of bedrest.

Physiologic Effects of Head of Bed Elevation

None of the 20 subjects developed flow velocities and ratios consistent with severe vasospasm during the study. One subject, number 12, increased his right MCA flow velocity consistent with an elevation from mild to moderate vasospasm (125 to 170 cm/sec) at 45 degrees and his left MCA flow velocity consistent with an elevation from normal to moderate vasospasm (117 to 161 cm/sec) also at 45 degrees. However, his right MCA to ICA ratio remained consistent with mild and moderate vasospasm and his left MCA to ICA ratio fluctuated from mild-moderate to normal to back to the mild-moderate ratio category. Subject number 12 was a 48 years old man, 11 days post-aneurysmal subarachnoid hemorrhage, Hunt-Hess grade I, with an anterior communicating artery aneurysm. He had bilateral MCA vasospasm on the day of the study and was not receiving boluses or vasopressors. However, he was not the only subject with bilateral MCA vasospasm, not receiving fluid boluses or vasopressors (Figure 15). Subjects numbers 5, 10, 13, and 15 were also in bilateral MCA vasospasm and not receiving vasospasm or vasopressors. Yet subject numbers 5, 10, 13, and 15 did not increase their flow velocities to the same extent. Subjects numbers 4 and 11 maintained mild right MCA vasospasm throughout the 0-20-45-0 degree sequence; and subject 17 maintained moderate right MCA vasospasm throughout the four positions. In regard to left MCA vasospasm, subjects number 8 and 15 maintained flow velocities and MCA:ICA ratios consistent with mild vasospasm; and subjects, numbers 9 and 16, maintained moderate vasospasm throughout all four positions.

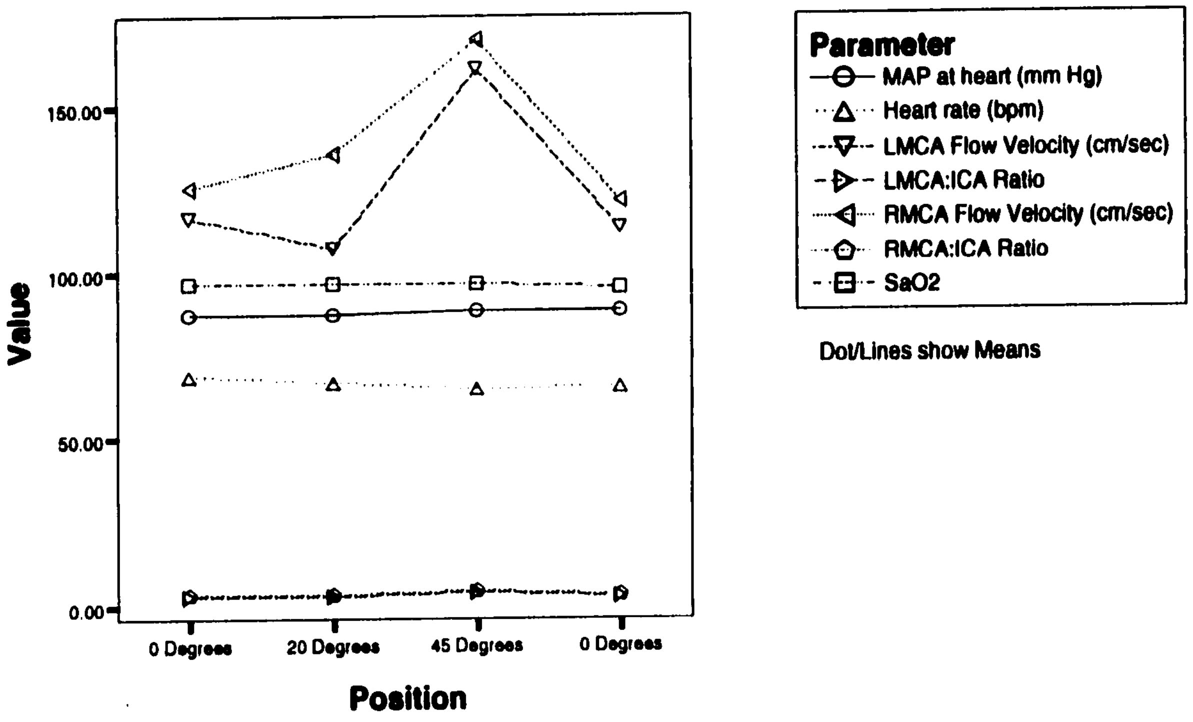


Figure 15. Subject 12 at 0, 20, 45, and 0 Degrees

Subject number 19 increased from mild to moderate left MCA vasospasm during the 0-20-45-0 sequence but maintained a left MCA flow velocity within 10 cm/sec, ranging from 148 to155 cm/sec throughout, and a left MCA: ICA ratio within 0.42, ranging from 3.77 to 4.19. (See Appendix A for graphs of individual responses.)

As expected, intracranial pressure significantly and consistently decreased with head of bed elevation and increased with return to the second 0 degrees. The changes in cerebral perfusion pressure, a function of mean arterial pressure minus intracranial pressure, with the transducer at the phlebostatic axis was statistically significant; however changes in cerebral perfusion pressure with the transducer leveled at the head (external auditory meatus) was not statistically significant. While it is well established that aneurysmal subarachnoid hemorrhage, and chronic hypertension, a risk factor for intracranial aneurysm, impair cerebral autoregulation (Yamamoto, Nishizawa, Tsukada, et al., 1998; Volby, Enevoldesen, & Jensen, 1985; Tenjin, Hirakawa, Mizukawa, et al., 1988; Dernbach, Little, Jones, et al., 1988), an autoregulatory index could not be calculated with the TCD software at either the phlebostatic axis or head due to small and gradual mean arterial blood pressure changes during the study. Perhaps some degree of cerebral autoregulation occurred since changes in cerebral perfusion pressure measured with the transducer at the external auditory meatus were not statistically significant as a group at 20 and 45 degrees head elevation. (March, Mitchell, & Grady, 1990).

Cerebral blood flow is influenced by a number of physiologic factors including intracranial pressure, cerebral perfusion pressure, autoregulation, and vasospasm. This study has focused primarily on vasospasm with some attention to intracranial pressure, cerebral perfusion pressure, and autoregulation.

Specific Aims

Each of the specific aims of this study has been addressed. The effect of the head of bed elevations of 20 and 45 degrees on mild and moderate cerebral vasospasm following aneurysmal subarachnoid hemorrhage has been addressed. Right and left MCA flow velocities and ratios were calculated for each of the 20 subjects. The overall response to head of bed elevation and individual responses to head of bed elevation were both noted. While individual differences were noted, as a group, the response to head elevation was not statistically or clinically significant. However, two individuals did exhibit clinically significant changes. While no individual developed severe vasospasm during the study procedure, some increased their flow velocities and ratios from normal to mild or mild to moderate and/or maintained mild or moderate vasospasm. No specific trends/ pattern(s) were identified in regard to specific patient characteristics or demographics, which consistently influenced vasospasm response to head of bed elevation. Perhaps increasing bolus parameters for individuals who experience clinically significant increases with head elevation prior to or during head of bed elevation is warranted.

Technology

While the technology utilized during this investigation indirectly measured cerebral blood flow, MCA flow velocities were measured continuously and the ICA flow velocity used to determine the MCA to ICA ratio was measured intermittently during the study. Signal interference between the two Dopplers prohibited simultaneous recording of the two parameters. In addition, the headband holding the ultrasound probes, would only allow access to the middle cerebral arteries. Information regarding other cerebral vasculature was not possible. The number and type of physiologic parameters measured also varied from patient to patient, depending on existing invasive devices at the time of the study. Some of the study participants did not have arterial lines and/or intracranial pressure monitors. Other measurements of cerebral blood flow and/or oxygenation were also not available, including jugular bulb oxygen saturation and catheters measuring cerebral oxygenation. Though considered a late sign by some, cerebral vasospasm has been associated with increased partial pressure of carbon dioxide and lower pH at the cellular level (Charbel, Du, Hoffman et al., 2000).

Limitations

A number of limitations are associated with this study. The sample size was limited to 20, which limited statistical power. In addition, a few patients who exhibited recurrent vasospasm or remained in vasospasm underwent angioplasty prior to the study. Angioplasty may or may not have affected the vasospastic vessels response to head elevation. In addition, only the MCA and ICA vessels were examined due to limitations of the technology. Data collection on the MCA vessels was continuous; ICA data collection was intermittent. While right and left MCA and MCA to ICA ratios were obtained on all 20 subjects at 0, 20, 45, and 0 degrees, the amount of data collection on other physiologic parameters varied based on the presence or absence of invasive monitoring devices at the time of the study. Each subject was given two minutes in each position to stabilize and then data was collected for approximately two to four minutes. Temporary compensatory mechanisms may not have been thoroughly exhausted at each level. Additional time at 20 and 45 degrees may have yielded different findings. Finally, this study took place in an environment where aggressive medical management of the aneurysmal subarachnoid hemorrhage patient at risk for vasospasm is the standard of care may limit the usefulness of this study in environments where less aggressive medical management, is the standard.

Implications for Nursing Practice

This study elucidates at least two implications for nursing practice. While, the effect of head of bed elevation in mild or moderate vasospasm following aneurysmal subarachnoid hemorrhage was not statistically significant as a group, one individual did show clinically significant changes. If head of bed elevation is undertaken, close monitoring of the individual response is warranted. Secondly, prophylactic management for increased vasospasm prior to or during head of bed elevation may be indicated for those individuals who are at risk or show evidence of clinically significant changes. Prophylactic management would include increasing blood pressure and/or fluid status parameters beyond those already in effect for the patient.

Implications for Future Nursing Research

A number of recommendations may be made in regard to future studies on the effect of head of bed elevation in vasospasm following aneurysmal subarachnoid hemorrhage. A larger sample size would increase the statistical power of the study. If possible, the study would consist of a more homogeneous sample in regards to no previous angioplasty. Multi-modality monitoring, such as jugular bulb oxygen saturation and brain oxygenation might also enhance our understanding of the effect of head of bed position on vasospasm. Alterations in the study design itself include increasing the degree of elevation to 60 degrees or increasing the time in each position to determine if tolerance is a function of time. Improvements in the existing transcranial Doppler technology would also be beneficial, including a single multi-channel transcranial Doppler for simultaneously and continuously recording middle cerebral arteries and internal carotid arteries; a different style of headband and/or sensor that allows multi-channel recording of different vessels simultaneously (e.g., middle cerebral artery, anterior cerebral artery, posterior cerebral artery); and inclusion of continuous transcranial Doppler monitoring as a parameter available with bedside critical care monitoring systems.

Conclusions

Twenty subjects 3-14 days post-aneurysmal subarachnoid hemorrhage in mild or moderate vasospasm who were post-operative or post interventional neuroradiologic coiling, underwent consecutive head positions of 0, 20, 45, and 0 degrees. Mean differences in MCA flow velocities and MCA: ICA ratios within- subjects as a group were not statistically significant. None of the subjects experienced flow velocities or ratios consistent with severe vasospasm. However, the degree of response varied among individual subjects without obvious explanation and was clinically significant in one of the twenty subjects. Additional studies are warranted. If patients with aneurysmal subarachnoid hemorrhage who are post operative or post interventional neuroradiology undergo head of bed elevations beyond 20-30 degrees, individual responses must be closely monitored.

LIST OF REFERENCES

_____ (2002). "2002 Heart and Stroke Statistical Update." Dallas: American Heart Association. p. 14, 33.

_____ (1997). Acute stroke in R.O. Cummins (ed.) Advanced cardiac life support. Dallas: American Heart Association. p. 10-11.

Adams, H.P. (1992). Prevention of brain ischemia after aneurysmal subarachnoid hemorrhage. Neurologic Clinics of North America, 10(1), 251-268.

Adams, H.P., Kassell, N., & Torner, J., et al. (1987). Predicting cerebral ischemia after aneurysmal subarachnoid hemorrhage: Influences of clinical conditions, CT results, and antifibrinolytic therapy. Neurology, 37(10), 1586-1591.

Biller, J., Godersky, J.C., & Adams, H.P. (1988). Management of aneurysmal subarachnoid hemorrhage. Stroke, 19(10), 1300-1305.

Blissitt, P.A. (2000). Positioning in the management of cerebral perfusion: A research review. University of Washington, Seattle, WA. (Unpublished paper).

Brian, J.E., Faraci, F.M., & Heistad, D.D. (1996). Recent insights into the regulation of cerebral circulation. Clinical and Experimental Pharmacology and Physiology, 23(6-7), 449-457.

Broderick, J.P., Brott, T., Tomsick, T., et al. (1993). Intracerebral hemorrhage more than twice as common as subarachnoid hemorrhage. Journal of Neurosurgery, 78(2), 188-191.

Brown, R.D., Whisnant, J.P., Sicks, J.D., et al. (1996) Stroke incidence, prevalence and survival. Secular trends in Rochester, Minnesota, through 1989. Stroke 27(3), 373-380.

Carpenter, D.A., Grubb, R.L., Tempel, L.W., et al. (1991). Cerebral oxygen metabolism after aneurysmal subarachnoid hemorrhage. Journal of Cerebral Blood Flow and Metabolism. 11(5), 837-844.

Carpenter, M.B. (1991). Core text of neuroanatomy. Williams and Wilkins: Baltimore. pp. 434.

Charbel, F.T., Du, X., Hoffman, W.E., et al. (2000). Brain tissue pO2, pCO2, and pH during cerebral vasospasm. Surgical Neurology. 54(6), 432-437.

Cook, D.A. (1995). Mechanisms of cerebral vasospasm in subarachnoid hemorrhage. Pharmacology and Therapeutics, 66, 259-284.

Cook, D.A. & Vollrath, B. (1995). Free radicals and intracellular events associated with cerebrovascular spasm. Cardiovascular Research, 30(4), 493-500.

Deitrick, J.E., Whedon, G.D., & Shorr, E. (1948). Effects of immobilization upon various metabolic and physiologic functions of normal men. American Journal of Medicine, 4, 3-36.

Dernbach, P.D., Little, J.R., Jones, S.C., et al. (1988). Altered cerebral autoregulation and carbon dioxide reactivity after aneurysmal subarachnoid hemorrhage. Neurosurgery, 22(5), 822-826.

Douville, C.M., Newell, D.W., Trimble, B.A., et al. (1990). Detection of vasospasm following subarachnoid hemorrhage using transcranial Doppler. The Journal of Vascular Technology, 14(3), 111-115.

Durward, Q.J., Amacher, A.L., Del Maestro, R.F.D., et al. (1983). Cerebral and cardiovascular responses to change in head elevation in patients with intracranial hypertension. Journal of Neurosurgery, 59(6), 938-944.

Eckberg, D.L, & Fritsch, J.M. (1992). Influence of ten day head-down bedrest on human carotid baroreceptor-cardiac reflex function. Acta Physiological Scandinavica, 144, (S604), 69-76.

Edvinsson, L., MacKenzie, E.T., & McCulloch, J. (1993). Cerebral blood flow and metabolism. Raven Press: New York.

Ehrenreich, H. & Schilling, L. (1995). New developments in the understanding of cerebral vasoregulation and vasospasm: The endothelin-nitric oxide network. Cleveland Clinic Journal of Medicine, 62(2), 105-116.

Fazl, M., Houlden, D.A., & Weaver, K. (1991). Correlation between cerebral blood flow, somatosensory evoked potentials, CT scan, grade, and neurological grade in patients with subarachnoid hemorrhage. The Canadian Journal of Neurological Sciences, 18(4), 453-457.

Feigin, V.L., Rinkel, G.J., Algra, A, et al.(2000). Calcium antagonists for aneurysmal subarachnoid hemorrhage. The Cochrane Library. Issue 2. Oxford: Update Software.

Feldman, Z., Kanter, M.J., Robertson, C.S., et al. (1992). Effect of head elevation on intracranial pressure, cerebral perfusion pressure, and cerebral blood flow in head-injured patients. Journal of Neurosurgery, 76(2), 207-211.

Findlay, J.M. (1997). Current management of aneurysmal subarachnoid hemorrhage. Guidelines from the Canadian Neurosurgical Society. The Canadian Journal of Neurological Sciences, 24(2), 161-170.

Findlay, J.M., Macdonald, R.L., & Weir, B.K. (1991). Current concepts of pathophysiology and management of cerebral vasospasm following aneurysmal subarachnoid hemorrhage. Cerebrovascular and Brain Metabolism Reviews, 3(4), 336-361.

Fisher, C.M., Kistler, J.P., & Davis, J.M. (1980). Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. Neurosurgery, 6(1), 1-9.

Fogelholm, R.R., Turjanmaa, V.M.H. , Nuutila, M.T., et al. (1995). Diurnal blood pressure variations and onset of subarachnoid haemorrhage: A population-based study. Journal of Hypertension, 13(5), 495-498.

Gaetani, P., Tartara, F., Pignatti, P., et al. (1998). Cisternal CSF levels of cytokines after subarachnoid hemorrhage. Neurological Research, 20(4), 337-342.

Gallerani, M., Portaluppi, F., Maida, G., et al. (1996). Circadian and circannual rhythmicity in the occurrence of subarachnoid hemorrhage. Stroke, 27(10), 1793-1797.

Germon, K., Ladd, M., & Newton, C. (1996). "Increased intracranial pressure" in Core Curriculum in Neuroscience Nursing. Chicago. American Association of Neuroscience Nurses.

Gopinath, S.P., Robertson, C.S., Narayan, R.K., et al. (1994). The effect of changes in head position on cerebral hemodynamics in H. Nagal, K. Kamiya, S. Ishii (eds) Intracranial pressure IX. Tokyo: Springer-Verlag; 87-90.

Grap, M.J., Cantley, M., Munro, C.L., et al. (1999). Use of backrest elevation in critical care: A pilot study. American Journal of Critical Care, 8(1), 475-480.

Greenleaf, J.E. & Kozlowski, S. (1993). Physiological consequences of reduced physical activity during bedrest. Washington, DC: National Aeronautics and Space Administration.

Grote, E. & Hassler, W. (1988). The critical first minutes in subarachnoid hemorrhage. Neurosurgery , 22(4), 654-661.

Grubb, R.L., Raichle, M.E., Eichling, J.O., et al. (1974). The effects of changes in PaCO2 on cerebral blood volume, blood flow, and vascular mean transit time. Stroke, 5, 630-639.

Hauerberg, J. Rasmussen, G. Juhler, M., et al. (1995). The effect of nimodipine on autoregulation of cerebral blood flow after subarachnoid hemorrhage in rats. Acta Neurochirurgica, 132(1-3), 98-103.

Heros, R.C. (1989). Acute hydrocephalus after subarachnoid hemorrhage. Stroke, 20(6), 715-717.

Honma, Y., Fujiwara, T., Irie, K., et al. (1995). Morphological changes in human cerebral arteries after percutaneous transluminal angioplasty for vasospasm caused by subarachnoid hemorrhage. Neurosurgery, 36(6), 1073-1081.

Kazuki, S., Ohta, T., Ogawa, R., et al. (1997). Effect of intraluminal or extraluminal endothelin on perfused rabbit basilar arteries. Journal of Neurosurgery, 86(5), 859-865.

King, W.A. & Martin, N.A. (1994). Critical care of patients with subarachnoid hemorrhage. Neurosurgery Clinics of North America, 5(4), 767-787.

Kirchhoff, K.T., Rebenson-Piano, M., & Patel, M.K. (1984). Mean arterial pressure reading: Variations with positions and transducer level. Nursing Research, 33, 343-345.

Kleinpeter, G., Schatzer, R., & Bock. F. (1995). Is blood pressure really a trigger for the circadian rhythm of subarachnoid hemorrhage. Stroke, 26(10) 1805-1810.

Knekt, P.A., Reunanen, K., Aho, M., et al. (1991). Risk factors for subarachnoid hemorrhage in a longitudinal population study. Journal of Clinical Epidemiology, 44(9), 933-939.

Kongable, G.L., Lanzino, G., & Germanson, T.P. (1996). Gender-related differences in aneurysmal subarachnoid hemorrhage. Journal of Neurosurgery, 84(1), 43-48.

Lassen, N.A. (1994). Autoregulation of cerebral blood flow. Circulation Research, 14(SI), I201-I204.

March, K., Mitchell, P., Grady, S., et al. (1990). Effect of backrest position on intracranial and cerebral perfusion pressure. Journal of Neuroscience Nursing, 22(6), 375-381.

Martin, W.R.W., Baker, R.P., Grubb, R.L., et al. (1984). Cerebral blood volume, blood flow, and oxygen metabolism in cerebral ischemia and subarachnoid hemorrhage: A vivo study using positron emission tomography. Acta Neurochirurgica, 70(1-2), 3-9.

Mayberg, M.R., Batjer, H.H., Dacey, R., et al. (1994). Guidelines for the management of aneurysmal subarachnoid hemorrhage. Circulation, 90(5), 2592-2605.

Meyer, C.H., Lowe, D., Meyer, M., et al. (1983). Progressive change in cerebral blood flow during the first three weeks after subarachnoid hemorrhage. Neurosurgery, 12(1), 58-76.

Meixensberger, J., Baunach, S., Amschler, J., et al. (1997) Influence of body position on tissue-pO2, cerebral perfusion pressure and intracranial pressure on patients with acute brain injury. Neurology Resident, 19(3), 249-255.

Milhorat, T.H. (1987). Acute hydrocephalus after aneurysmal subarachnoid hemorrhage. Neurosurgery, 20(1), 15-20.

Miller, J. & Diringer, M. (1995). Management of aneurysmal subarachnoid hemorrhage. Neurologic Clinics of North America, 13(3), 451-478.

Moraine, J.J., Berre, J., & Mellot, C. (2000). Is cerebral perfusion pressure a major determinant of cerebral blood flow during head elevation in comatose patients with severe intracranial lesions? Journal of Neurosurgery, 92(4), 606-614.

_______ (1996). Multi-Dop X4 Operating Instructions. DWI.

_______ (1991). N-CAT Continous Noninvasive Blood Pressure Monitor Model N- 500 Operator's Manual. Hayward CA: Nellcor.

_______ (1995). Neuroguard and CDS Transcranial Doppler Systems Instruction Manual. Fremont, CA: Neurogard, Inc.

Nornes, H. (1973). The role of intracranial pressure in the arrest of hemorrhage in patients with ruptured intracranial aneurysm. Journal of Neurosurgery, 39, 226-234.

Nornes, H. & Magnes, B. (1972). Intracranial pressure in patients with ruptured saccular aneurysm. Journal of Neurosurgery, 36, 537-547.

Oropello, J.M., Weiner, L., & Benjamin, E. (1996). Hypertensive hypervolemic hemodilutional therapy for aneurysmal subarachnoid hemorrhage: Is it efficacious? No. Critical Care Clinics of North America, 12(3), 709-730.

Pasqualin, A. (1998). Epidemiology and pathophysiology of cerebral vasospasm following subarachnoid hemorrhage. Journal of Neurological Sciences, 42(1), Supplement 1, 15-21.

Persson, L., Valtysson, J., Enbald, P., et al. (1996). Neurochemical monitoring using intracerebral microdialysis in patients with subarachnoid hemorrhage. Journal of Neurosurgery, 84(4), 606-616.

Powers, W.J. (1992). Hemodynamics and metabolism in ischemic cerebrovascular disease. Neurologic Clinics, 10(1), 31-48.

Quaglietti, S.E., Stotts, N.A., & Lovejoy, N.C. (1988). The effect of selected positions on rate pressure product for the myocardial infarction patient. Journal of Cardiovascular Nursing, 2(4), 77-85.

Rosner, MJ, & Coley, IB. (1986). Cerebral perfusion pressure, intracranial pressure, and head elevation. Journal of Neurosurgery, 65(5), 636-641.

Salom, J.B. Torregrosa, G., & Alborch, E. (1995). Endothelins and the cerebral circulation. Cerebrovascular and Brain Metabolism Reviews, 7(2), 131-152.

Saveland, H., Nilsson, O.G., Boris-Moller, F., et al. (1996). Intracerebral microdialysis of glutamate and aspartate in two vascular territories after aneurysmal subarachnoid hemorrhage. Neurosurgery, 38(2), 12-19.

Schmieder, K., Jarus-Dziedzic, K., Wronski, J., et al. (1997). Continuous monitoring of jugular bulb saturation in comatose patients: Therapeutic implications. Acta Neurochirurgica, 134(1-2), 71-75.

Schnieder, G.H., vonHelden, A, Franke, R., et al. (1993). Influence of body position on jugular venous oxygen saturation, intracranial pressure and cerebral perfusion pressure. Acta Neurochirurgica, 59(Supplement), 107-112.

Seifert, J., Loeffler, B.M., Zimmermann, M., et al. (1995). Endothelin concentrations in patients with aneurysmal subarachnoid hemorrhage. Correlation with cerebral vasospasm, delayed ischemic neurological deficits, and volume of hematoma. Journal of Neurosurgery, 82(1), 55-62.

Seiler, R.W. & Newell, D.W. (1992). Subarachnoid hemorrhage and vasospasm in D.W. Newell & R. Aaslid (eds.) Transcranial Doppler. New York: Raven Press. pp. 101-107.

Solenski, N.J., Haley, E.C., Kassel, N.F., et al. (1995). Medical complications of aneurysmal subarachnoid hemorrhage: A report of the multicenter, cooperative aneurysm study. Critical Care Medicine, 23(6), 1007-1017.

Strandgaard, S. & Paulson, O.B. (1992). Regulation of cerebral blood flow in health and disease. Journal of Cardiovascular Pharmacology, 19(S6), S89-S93.

Szaflarski, N.L. (1996). Immobility phenomena in critically ill adults in J.M. Clochesy, C. Breu, S. Cardin, et al. (eds) Critical Care Nursing. Philadelphia: W.B. Saunders. pp. 1313-1334.

Takenaka, K., Suzuki, Y., Kawakubo, et al. (1994). Cardiovascular effects of 20 days bed rest in healthy young subjects. Acta Physiologica Scandinavica, 150(S616), 59-63.

Tenjin, H., Hirakawa, K., Mizukawa, N., et al. (1988). Dysautoregulation in patients with ruptured aneurysms: Cerebral blood flow measurements obtained during surgery by a temperature-controlled thermoelectrical method. Neurosurgery, 23(6), 705-709.

Ullman, J.S. & Bederson, J.B. (1996). Hypertensive hypervolemic, hemodilutional therapy for aneurysmal subarachnoid hemorrhage: Is it efficacious? Yes. Critical Care Clinics of North America, 12(3), 697-707.

Unterberg, A.W., Sakowitz, O.W., Sarrafzadeh, A.S., et al. (2001). Role of bedside microdialysis in the diagnosis of cerebral vasospasm following aneurysmal subarachnoid hemorrhage. Journal of Neurosurgery, 94(5), 740-749.

Ursino, M. (1991). Mechanisms of cerebral blood flow regulation. Critical Reviews in Biomedical Engineering, 18(4), 255-288.

Valbona, C., Cardus, D., Vogt, F.B., et al. (1965). The effect of bedrest on various parameters of physiological function: Part VIII. The effect on the cardiovascular tolerance to passive tilt. Washington DC: National Aeronautics and Space Administration.

Valbona, C., Vogt, F.B., Cardus, D., et al. (1965). The effect of bedrest on various parameters of physiological function: Part I. Review of the literature on the physiological effects of immobilization. Washington, DC: National Aeronautics and Space Administration.

Vogt, F.B., Spencer, W.A., Cardus, D., et al. (1995). The effect of bedrest on various parameters of physiological function: Part XIII. A review of possible mechanisms of orthostatic intolerance to passive tilt. Washington, DC: National Aeronautics and Space Administration.

Volby, B., Enevoldsen, E.M., & Jensen, F.T. (1985). Cerebrovascular reactivity in patients with ruptured intracranial aneurysms. Journal of Neurosurgery, 62, 59-67.

Weaver, J.P. & Fisher, M. (1994). Subarachnoid hemorrhage: An update of pathogenesis, diagnosis, and management. Journal of the Neurological Sciences, 125(2), 119-131.

Wilkins, R.H. (1981). Update: Subarachnoid hemorrhage and saccular intracranial aneurysm. Surgical Neurology, 15(2), 92-101.

Yamamoto, S., Nishizawa, S., Tsukada, H., et al. (1998). Cerebral blood flow autoregulation following subarachnoid hemorrhage rates: Chronic vasospasm shifts the upper and lower limits of autoregulation range toward higher blood pressures. Brain Research, 782(1-2), 194-201.

Appendix A

Graphic Representation of Physiologic Data

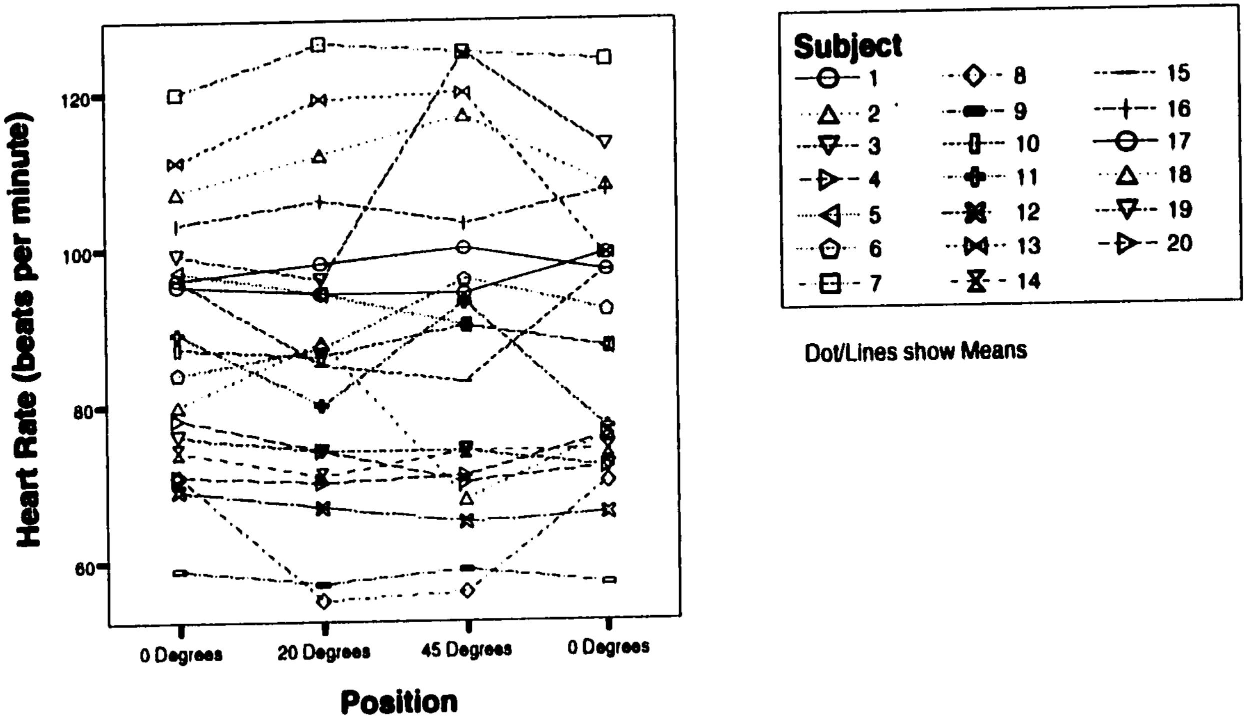
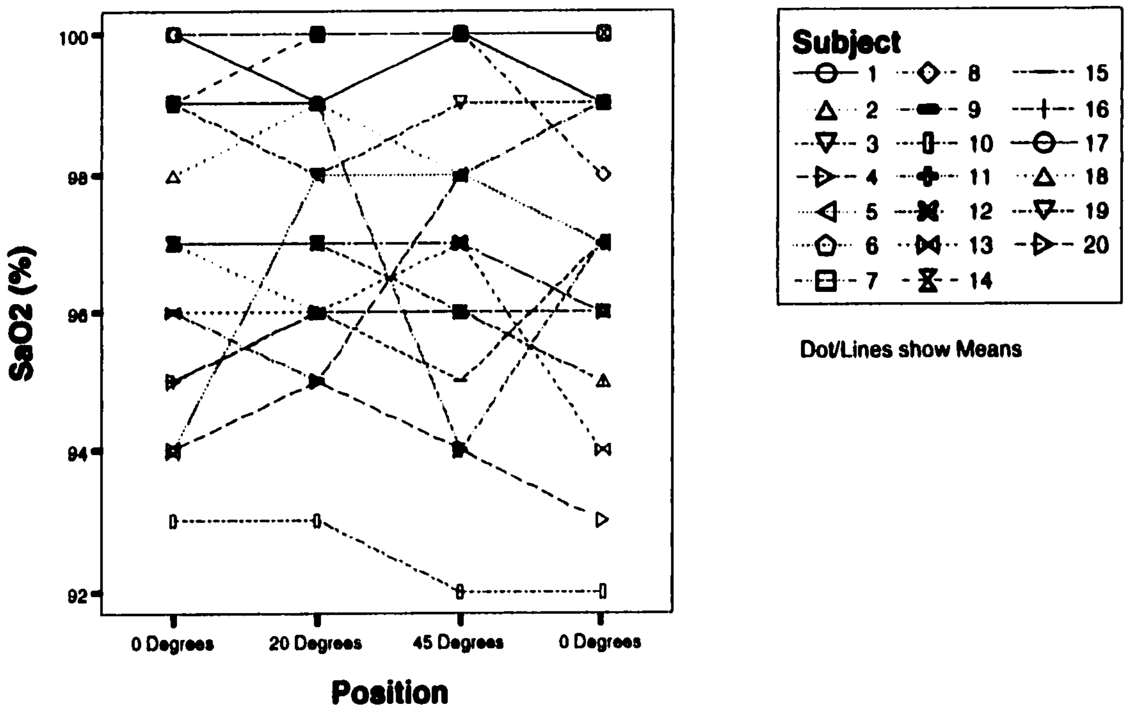


Figure 16. Heart Rate at 0, 20, 45 and 0 Degrees



Dot/Lines show Means

Figure 17. Oxygen Saturation at 0, 20, 45 and 0 Degrees

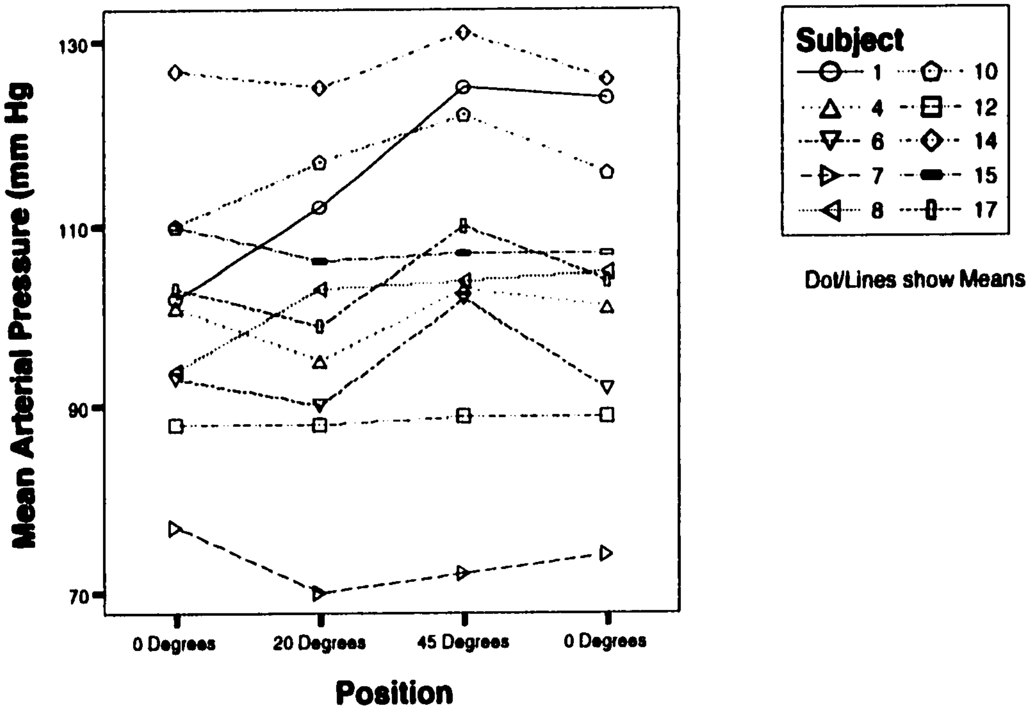


Figure 18. Mean Arterial Pressure Measured at Heart (Phlebostatic Axis) at 0, 20,45, and 0 Degrees

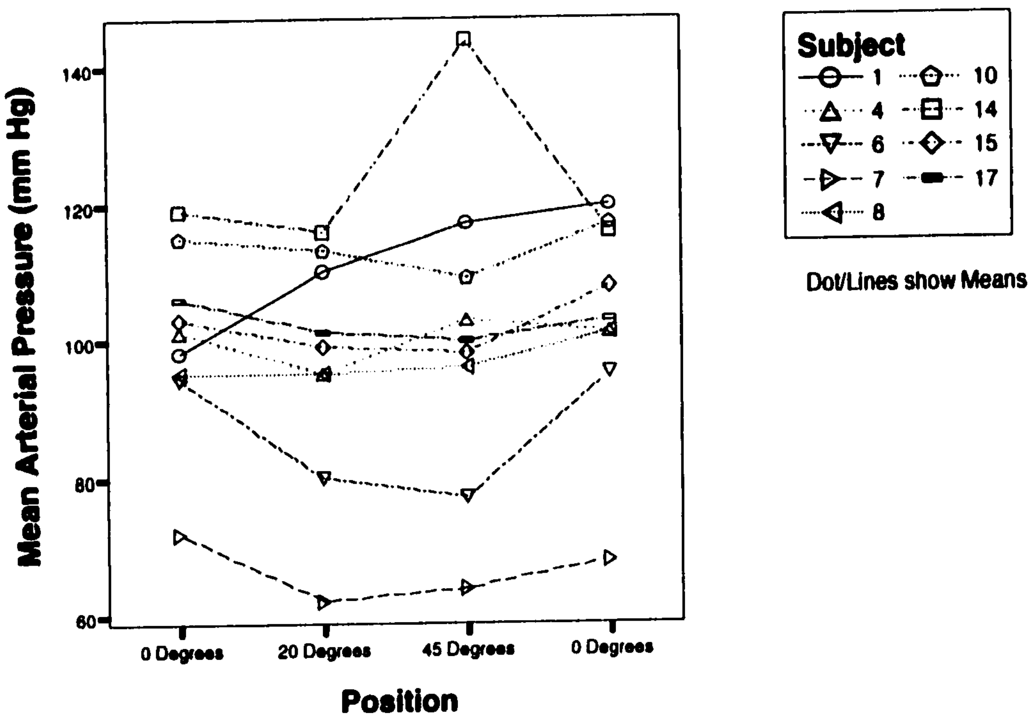


Figure 19. Mean Arterial Pressure at Head (External Auditory Meatus) at 0, 20, 45 and 0 Degrees

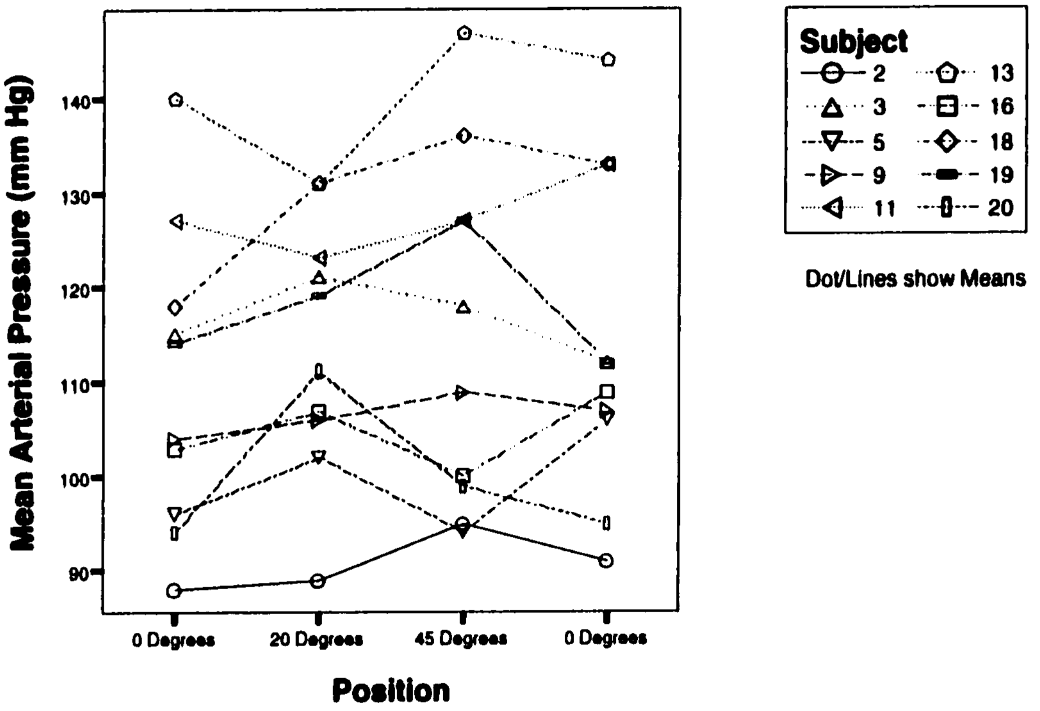


Figure 20. Mean Arterial Pressure Measured with Continuous Noninvasive Blood Pressure Monitor at 0, 20, 45 and 0 Degrees

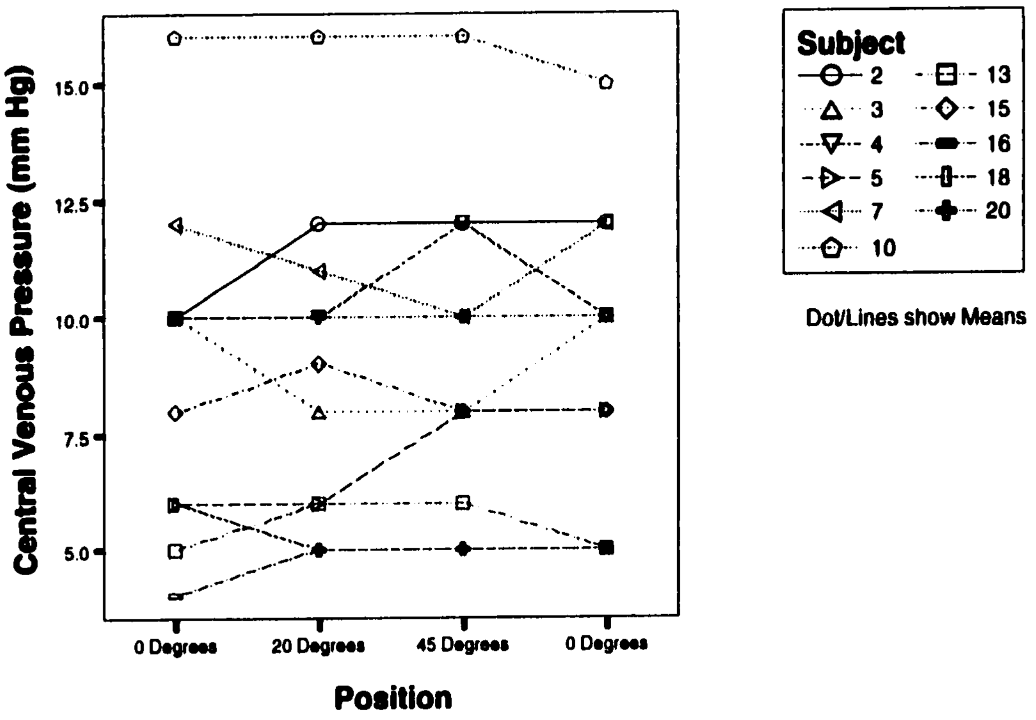
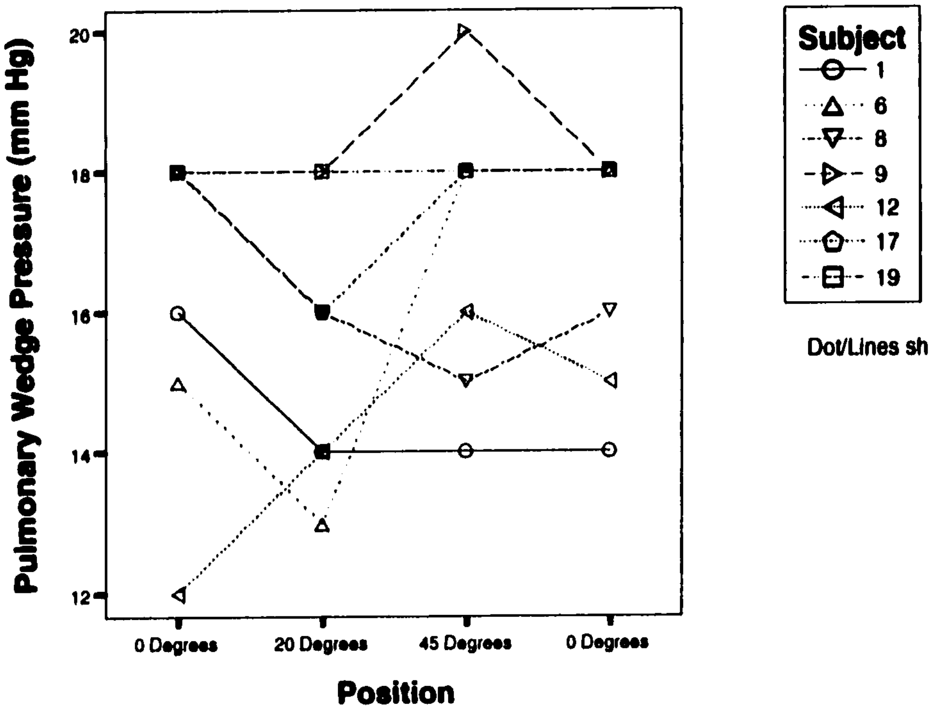
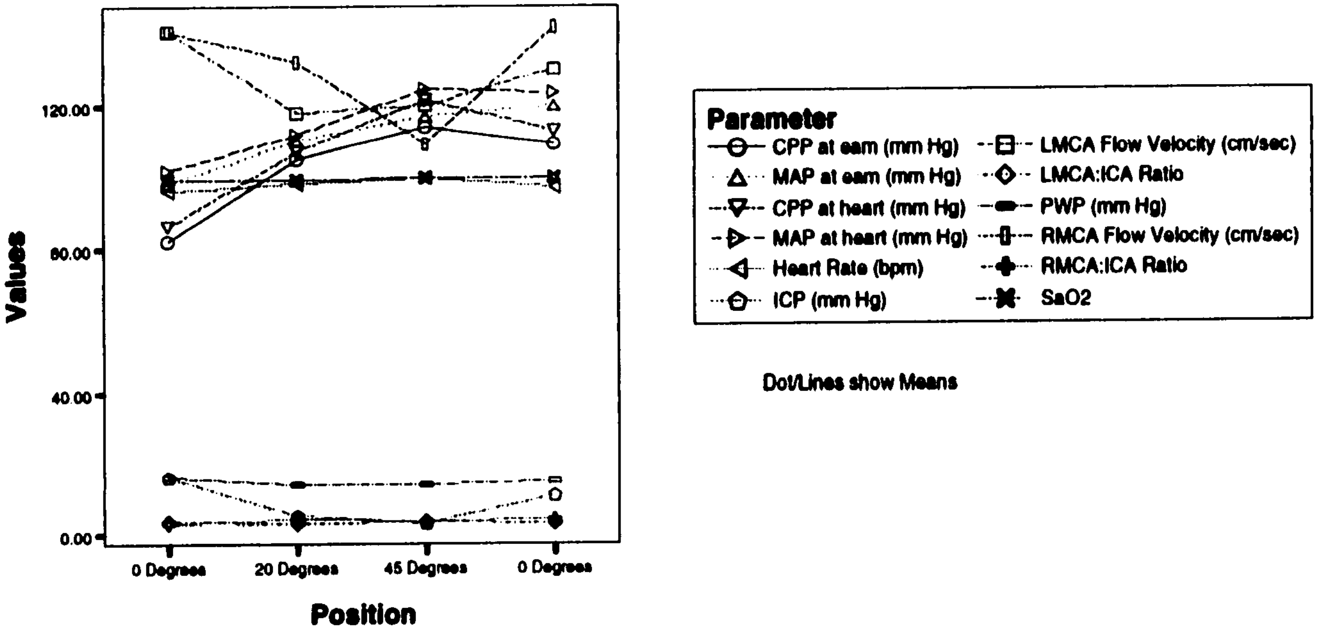


Figure 21. Central Venous Pressure at 0, 20, 45 and 0 Degrees



Dot/Lines show Means

Figure 22. Pulmonary Wedge Pressure Pressure at 0, 20, 45 and 0 Degrees



Dot/Lines show Means

Figure 23. Subject 1 at 0, 20, 45, and 0 Degrees

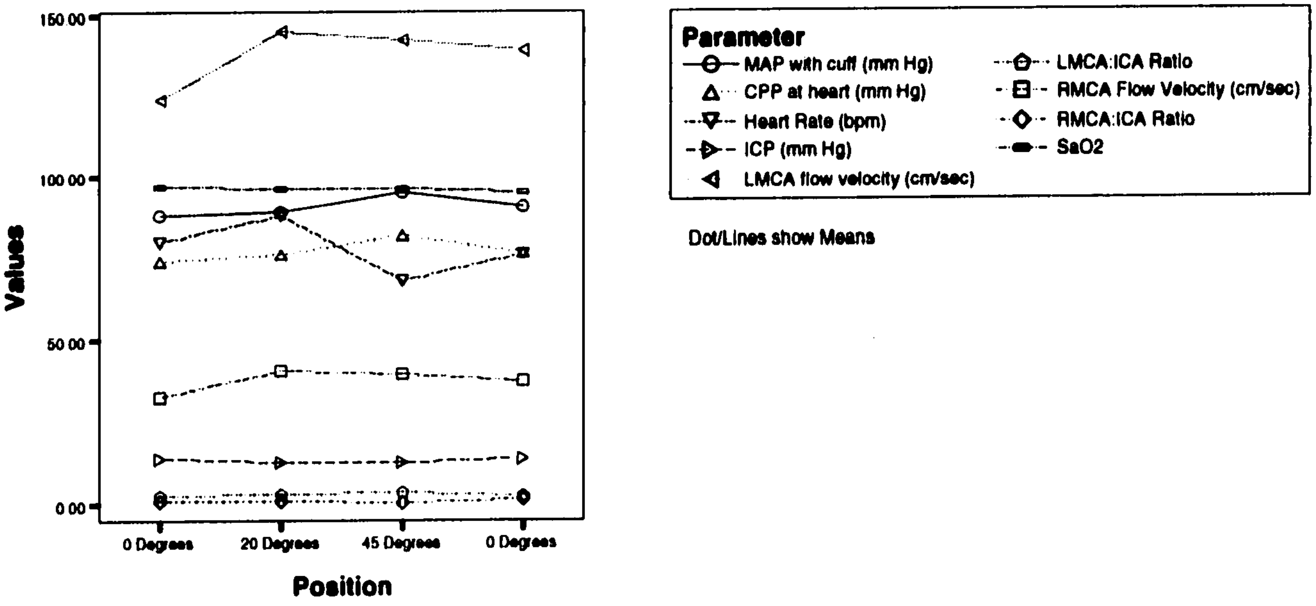


Figure 24. Subject 2 at 0, 20, 45, and 0 Degrees

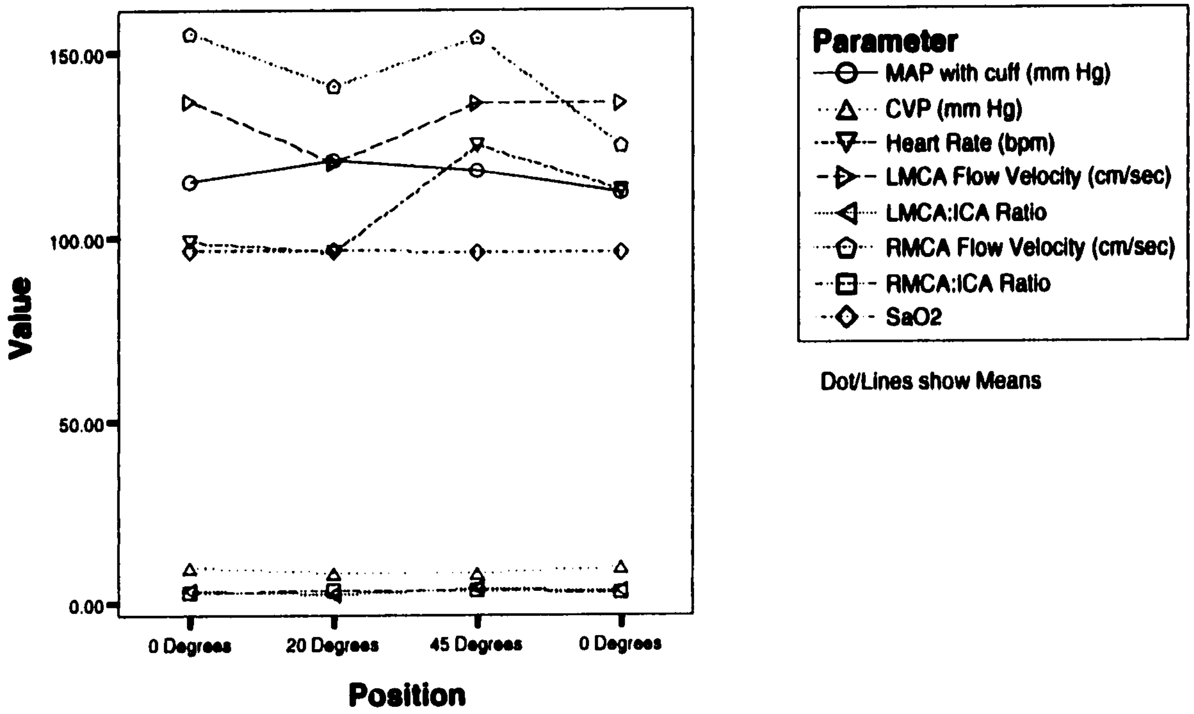


Figure 25. Subject 3 at 0, 20, 45, and 0 Degrees

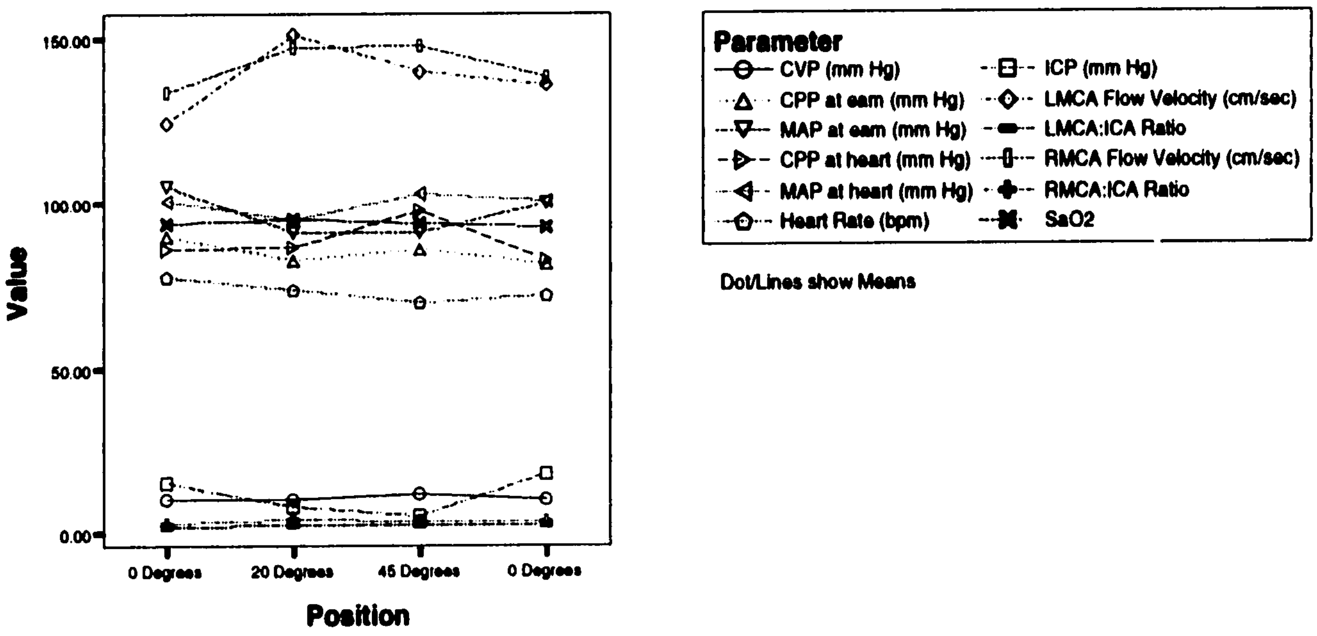


Figure 26. Subject 4 at 0, 20, 45, and 0 Degrees

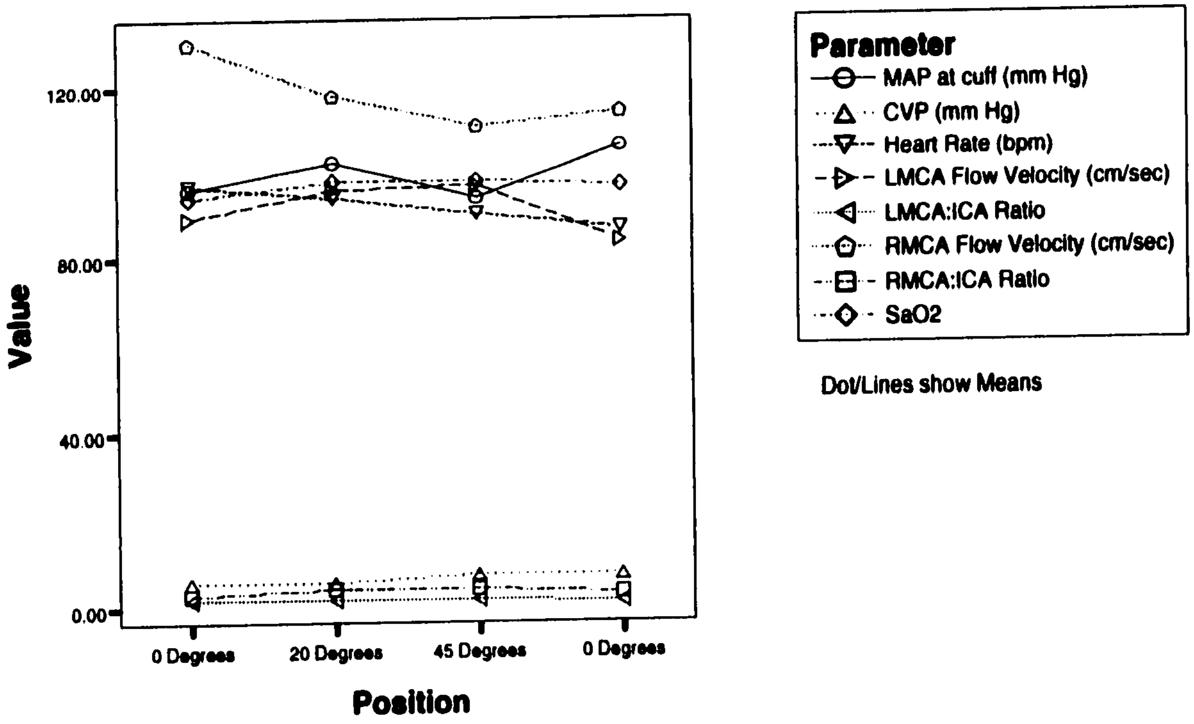


Figure 27. Subject 5 at 0, 20, 45, and 0 Degrees

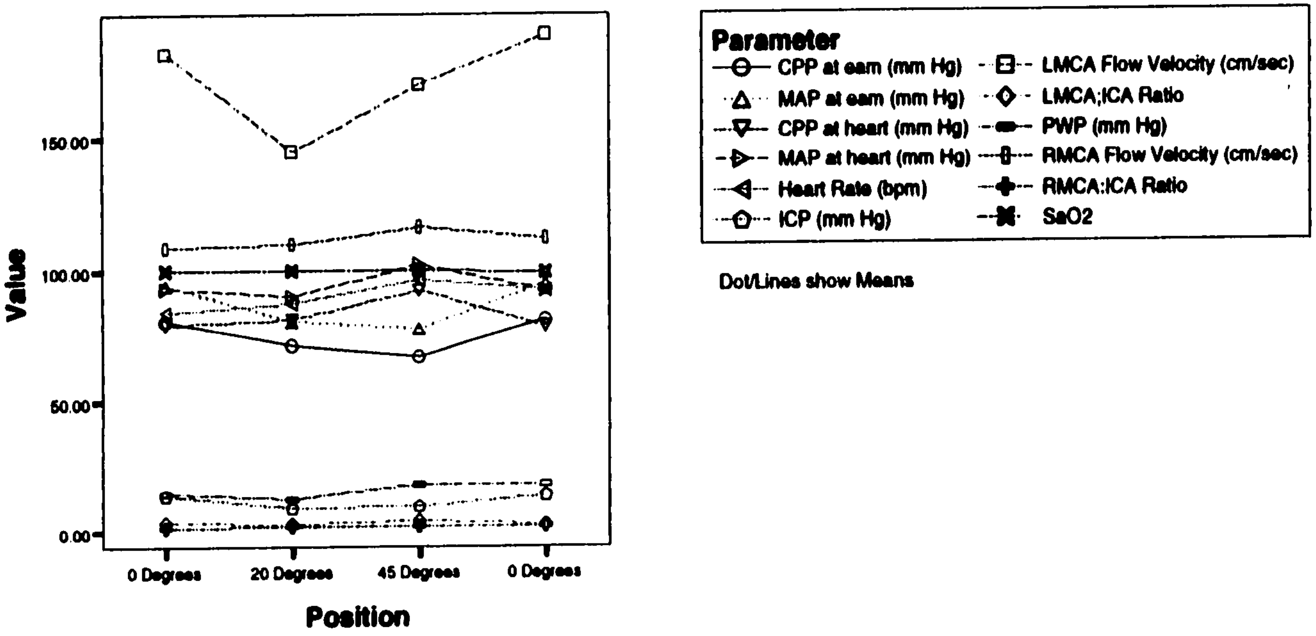


Figure 28. Subject 6 at 0, 20, 45, and 0 Degrees

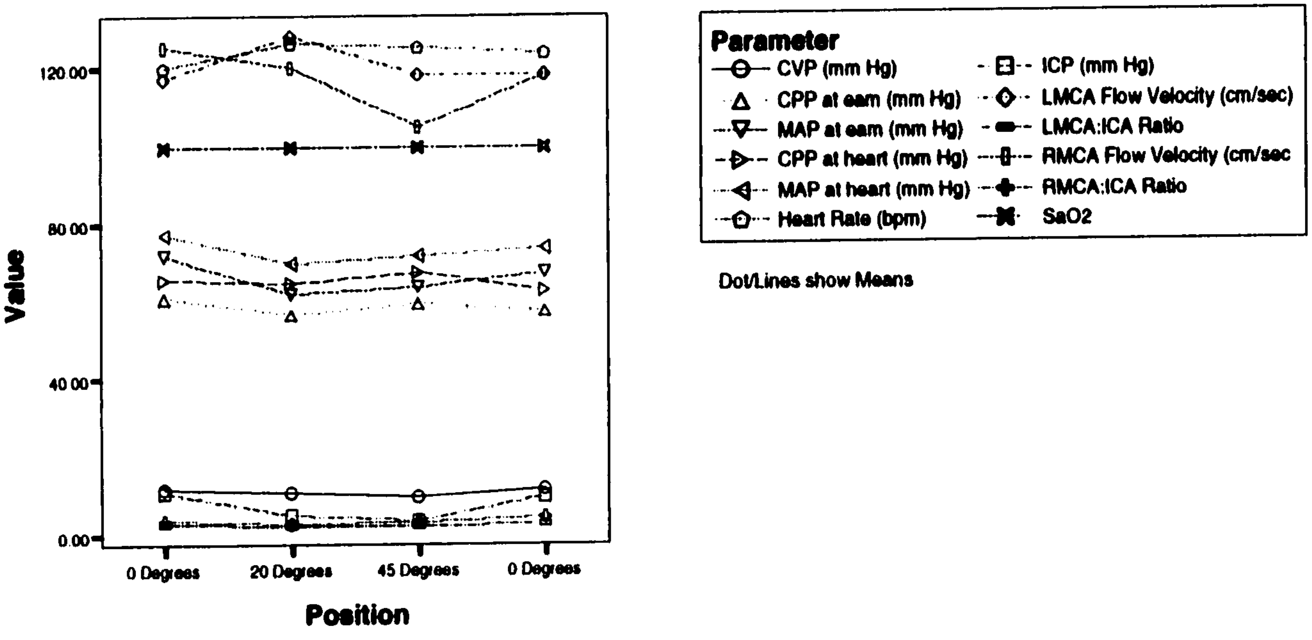


Figure 29. Subject 7 at 0, 20, 45, and 0 Degrees

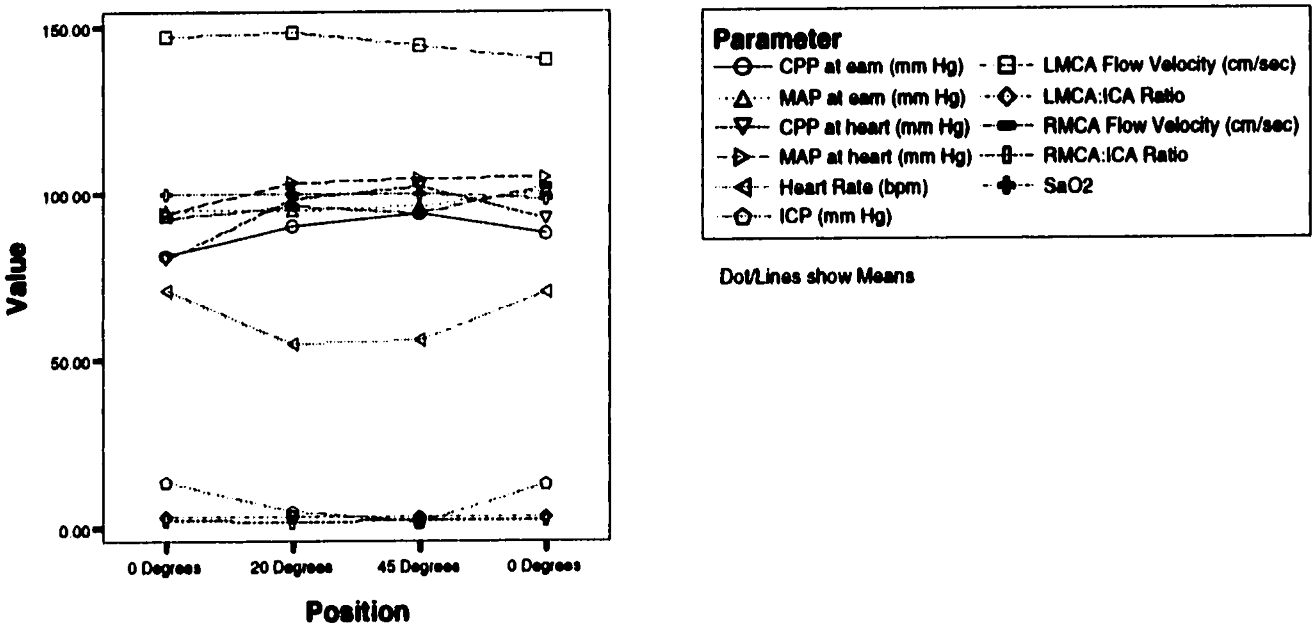


Figure 30. Subject 8 at 0, 20, 45, and 0 Degrees

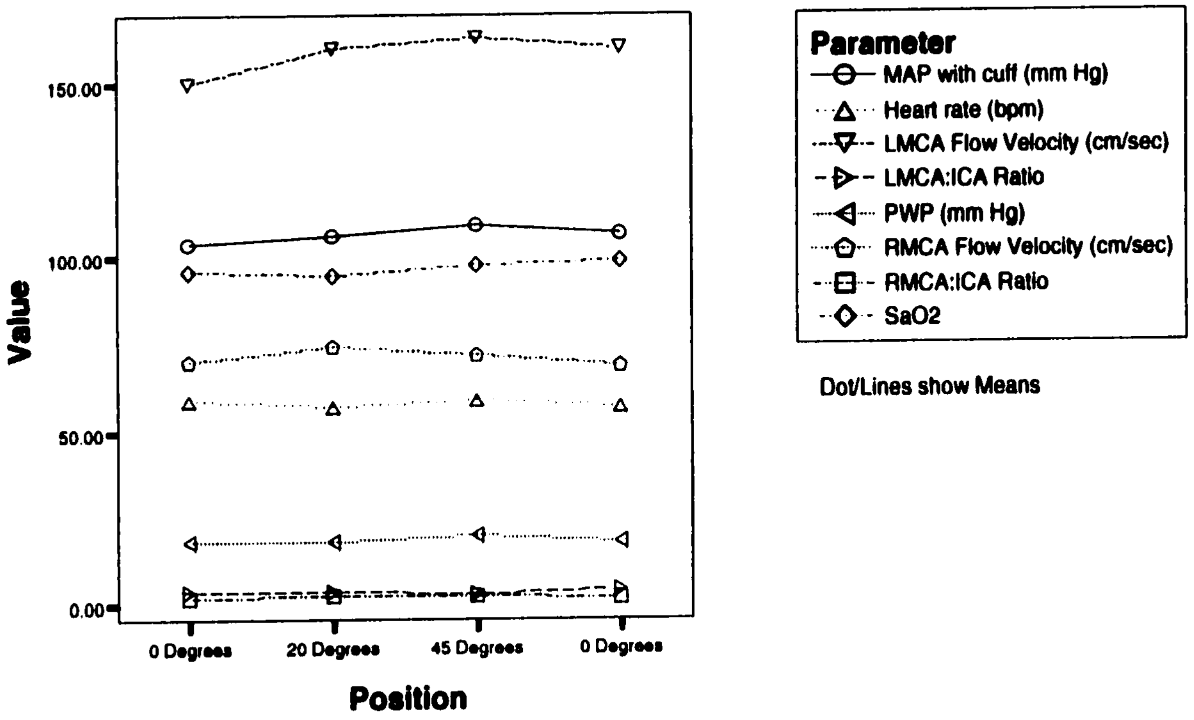
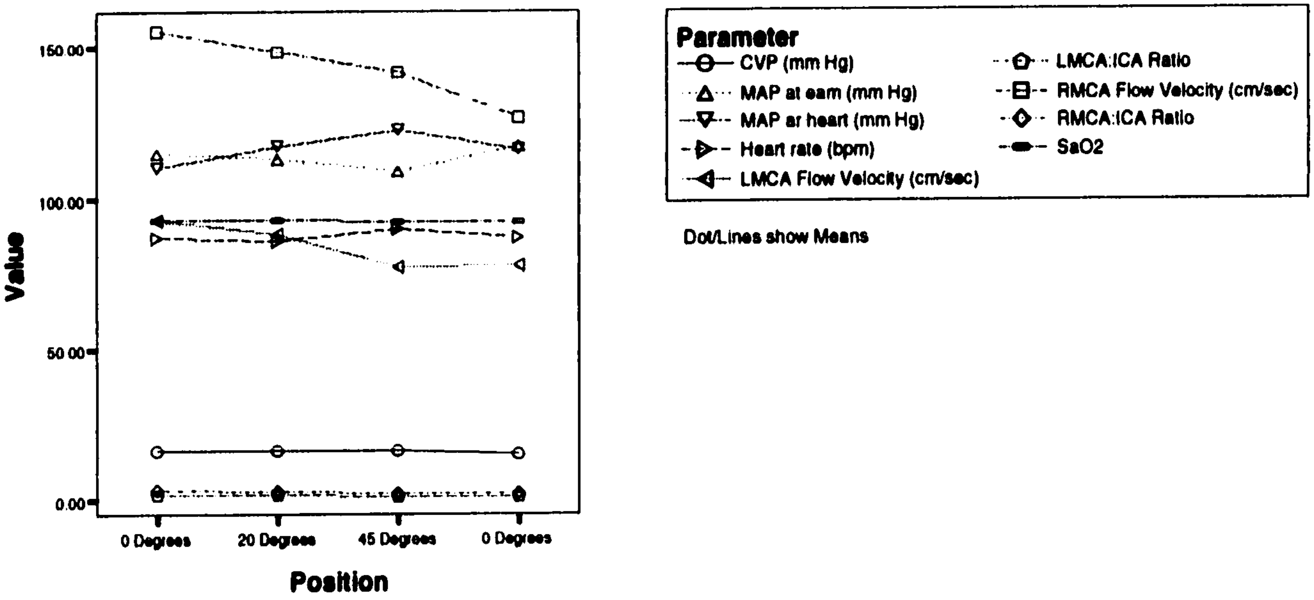


Figure 31. Subject 9 at 0, 20, 45, and 0 Degrees



Dot/Lines show Means

Figure 32. Subject 10 at 0, 20, 45, and 0 Degrees

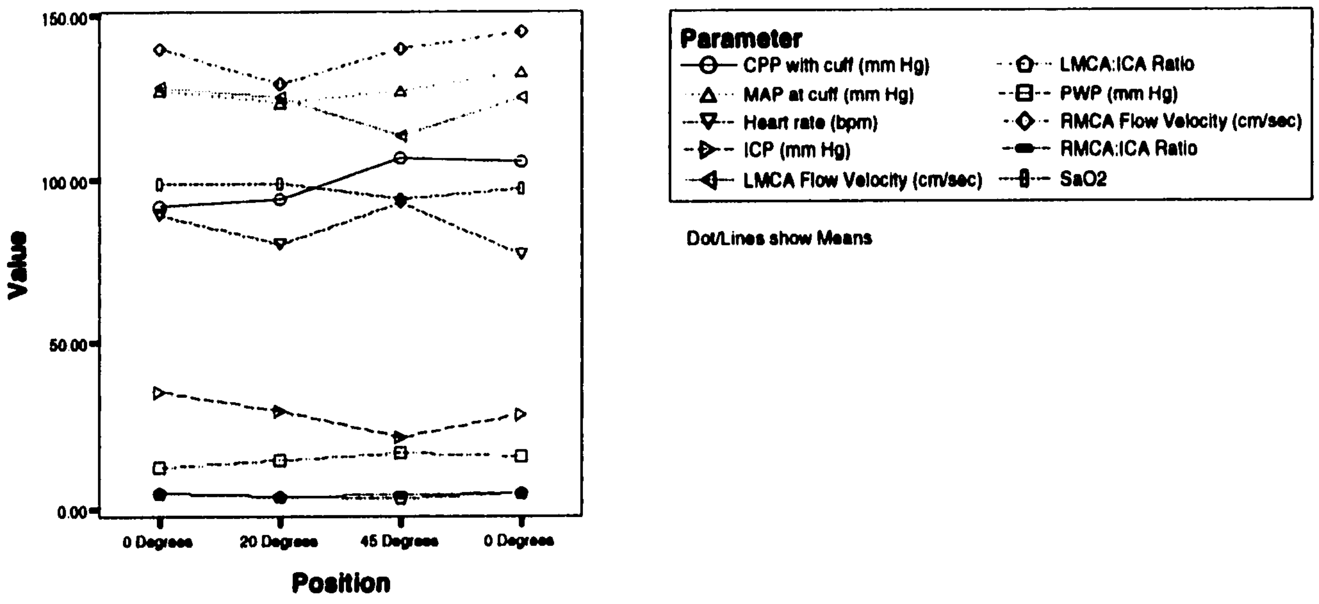
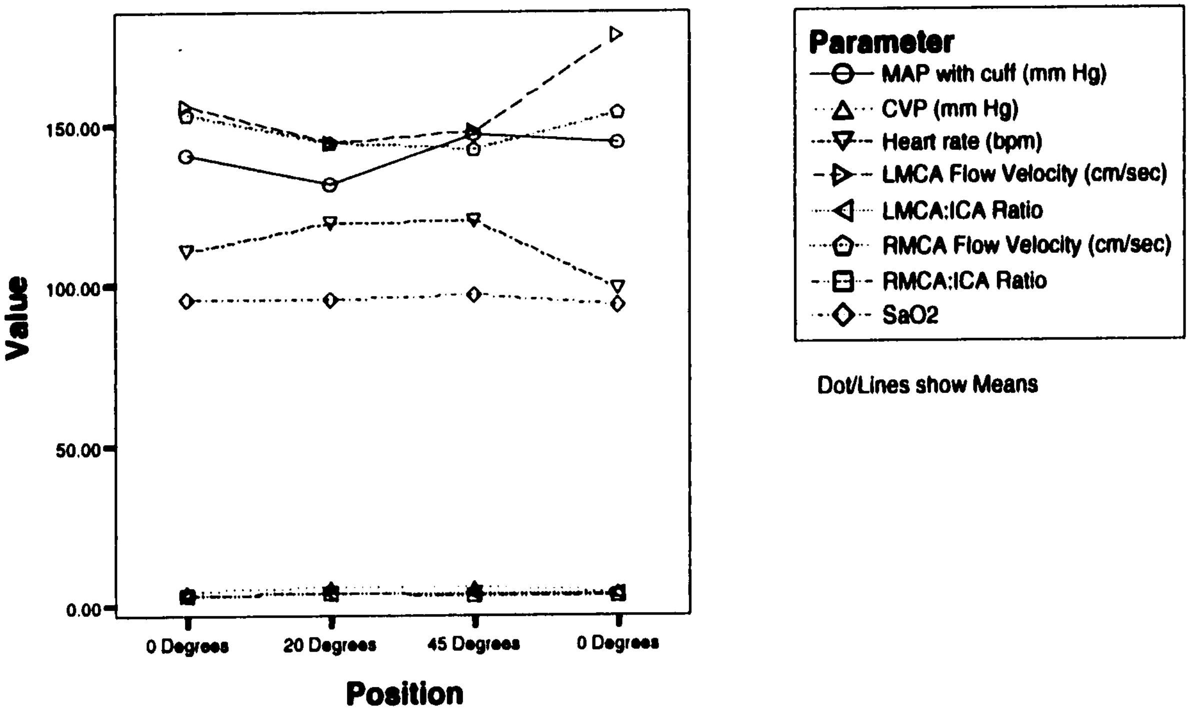
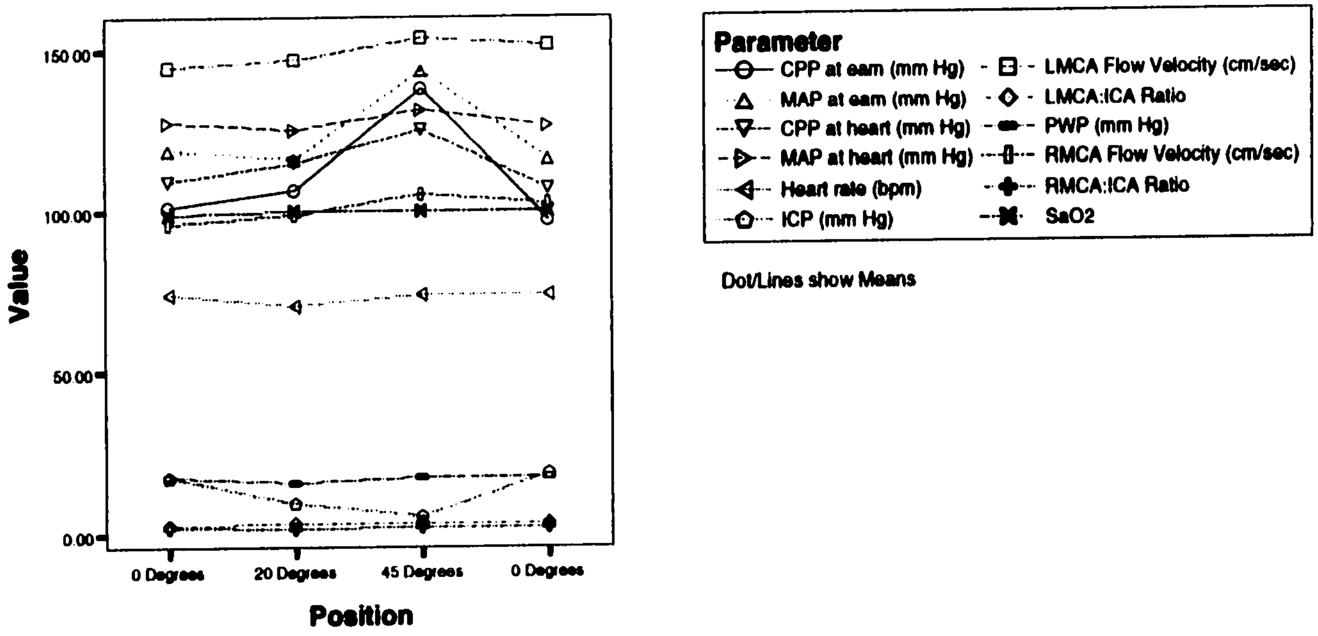


Figure 33. Subject 11 at 0, 20, 45, and 0 Degrees



Dot/Lines show Means

Figure 34. Subject 13 at 0, 20, 45, and 0 Degrees



Dot/Lines show Means

Figure 35. Subject 14 at 0, 20, 45, and 0 Degrees

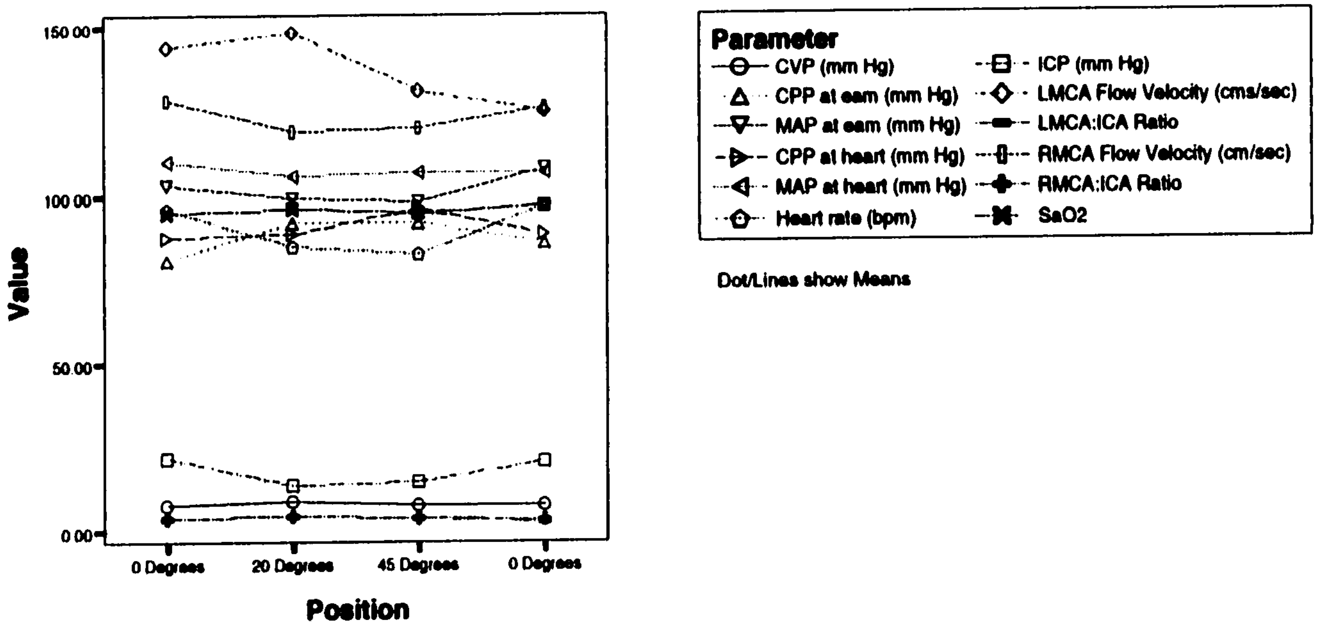


Figure 36. Subject 15 at 0, 20, 45, and 0 Degrees

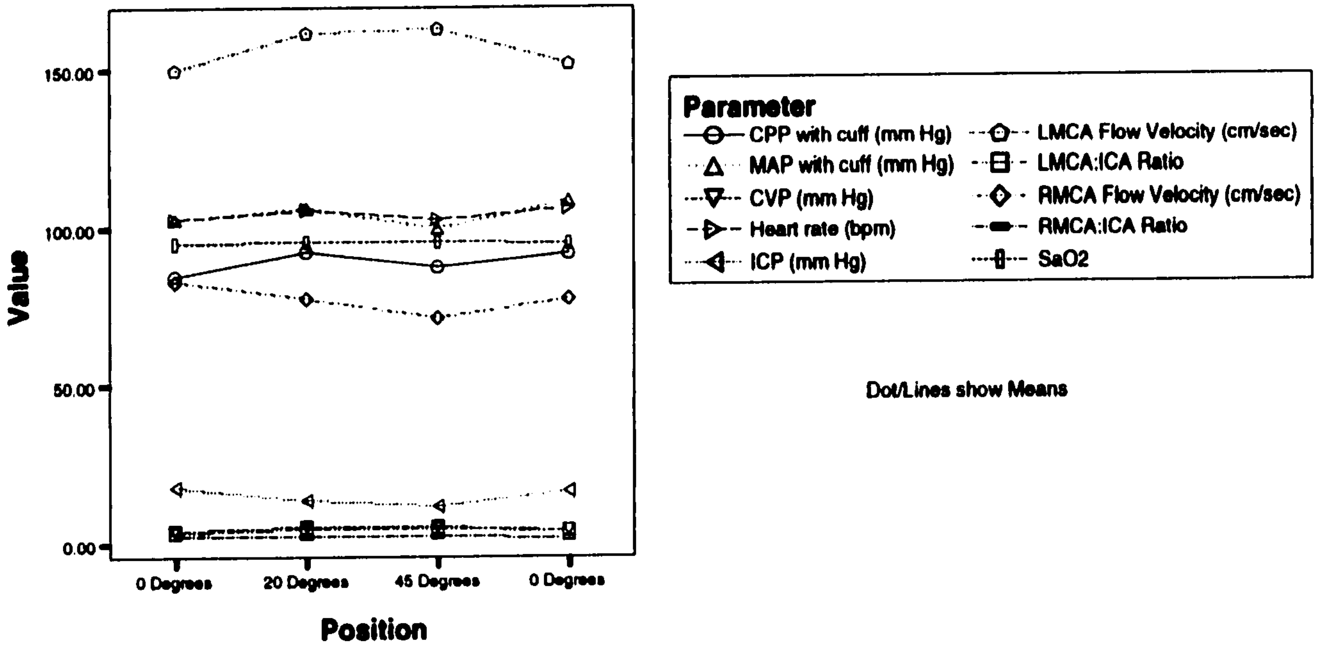


Figure 37. Subject 16 at 0, 20, 45, and 0 Degrees

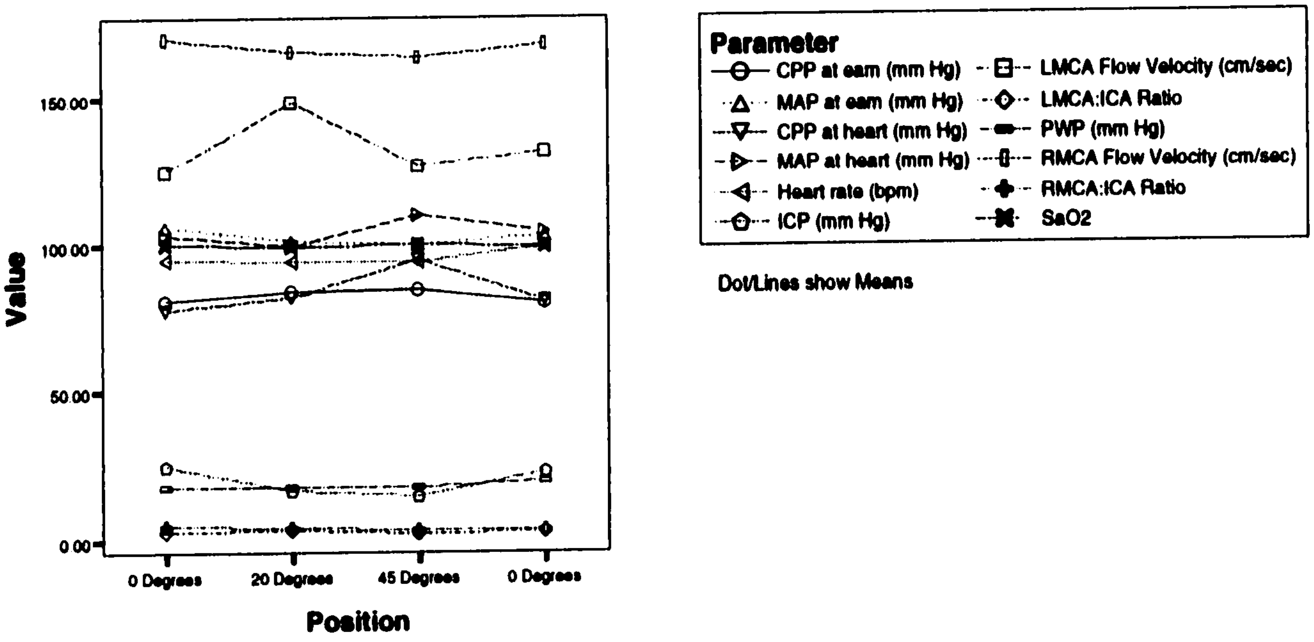


Figure 38. Subject 17 at 0, 20, 45, and 0 Degrees

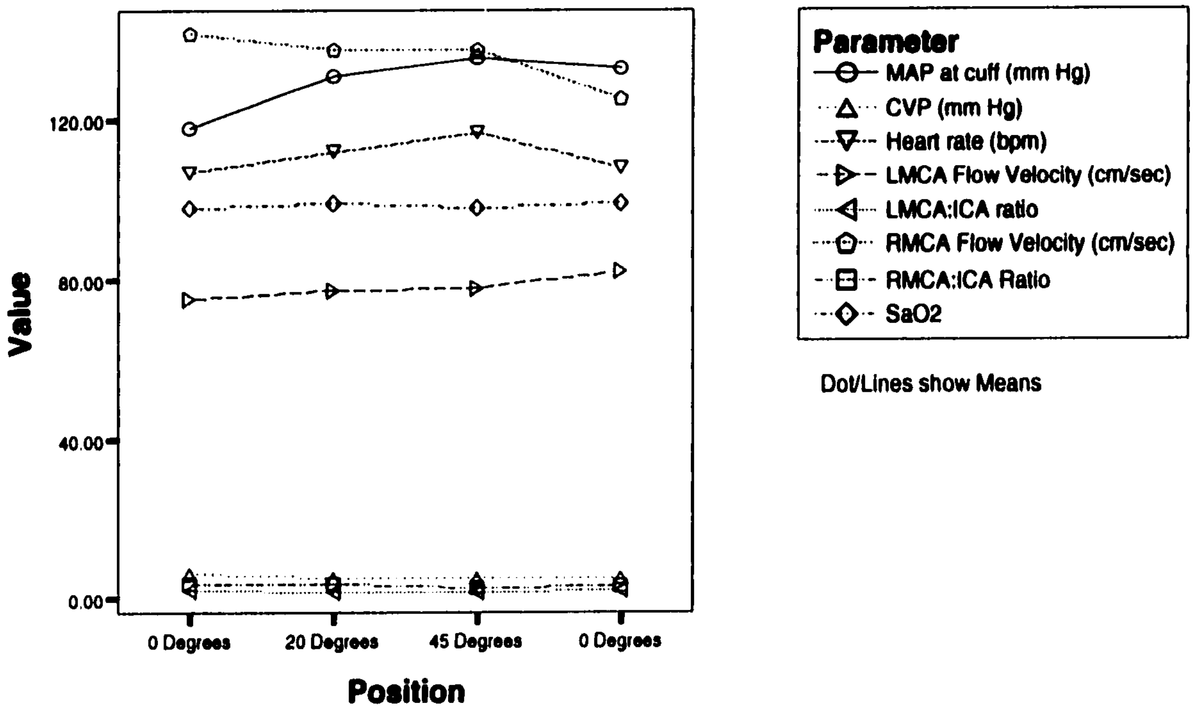


Figure 39. Subject 18 at 0, 20, 45, and 0 Degrees

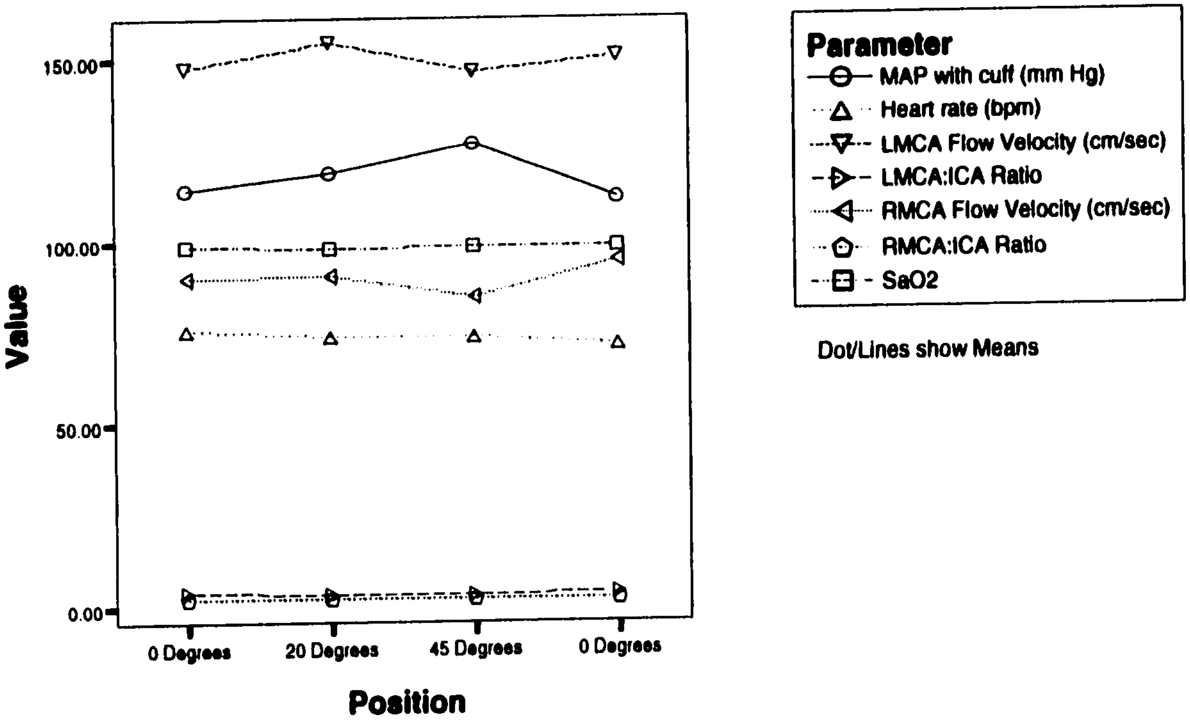
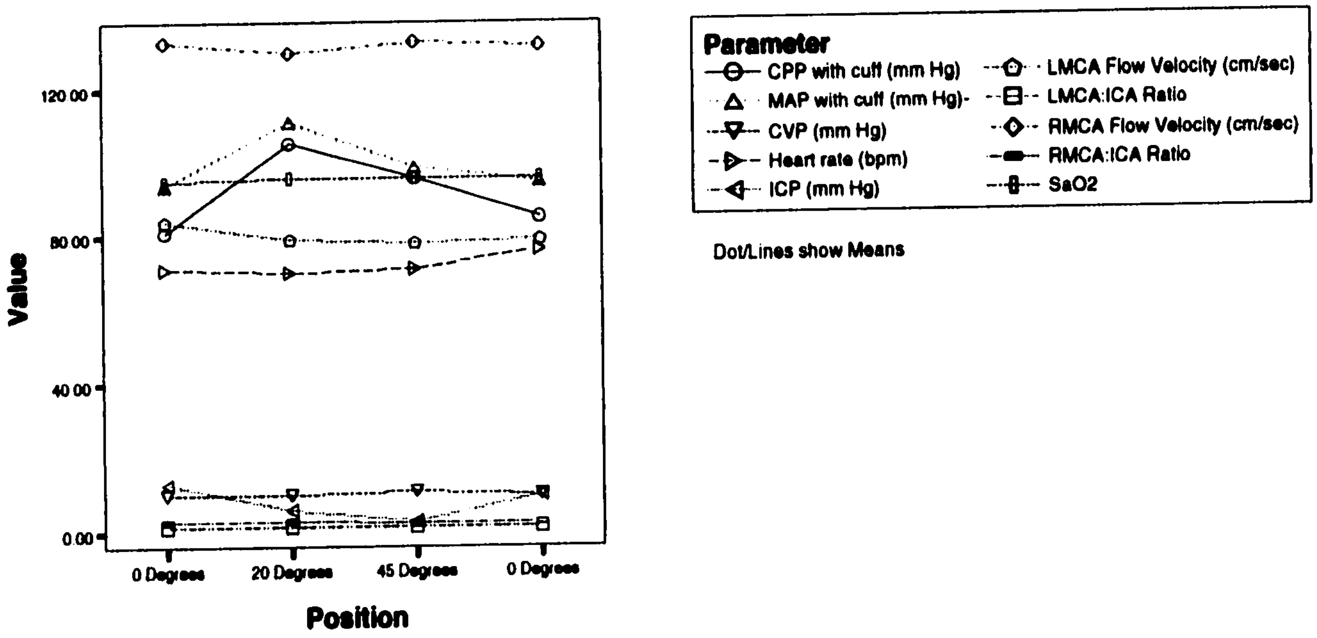


Figure 40. Subject 19 at 0, 20, 45, and 0 Degrees



Dot/Lines show Means

Figure 41. Subject 20 at 0, 20, 45, and 0 Degrees

Appendix B

Consent Form

University of Washington

The Effect of the Head-up Position on Cerebral Blood Flow in Mild or Moderate Cerebral Vasospasm Following Aneurysmal Subarachnoid Hemorrhage

Patricia A. Blissitt, R.N., M.S.N., CCRN, CNRN, CCM, CS, Doctoral Candidate, School of Nursing

24-Hour Emergency Telephone Number: (206) 447-9873 (if no answer, leave message and phone number on answer machine)
Beeper: (206) 405-5792 (9:00 a.m. to 5:00 p.m., Monday through Friday)

Pamela H. Mitchell, R.N., Ph.D., CNRN, FAAN, Professor, Biobehavioral and Health Systems Nursing and Associate Dean for Nursing Research, School of Nursing

Telephone (206) 685-1525

Purpose and Benefits

Patients recovering from subarachnoid hemorrhage as a result of an aneurysm rupture are bedridden for days or weeks following surgery. Unfortunately, prolonged bedrest is known to pose risks to the patient including, fainting with head elevation, pneumonia, the formation of blood clots, and constipation. Elevation of the head while in bed is thought to decrease the negative effects of prolonged bedrest. In addition, patients whose heads are elevated are able to get out of bed earlier and may make a faster recovery. However, the benefits of raising the head must be weighed against the risk of lowering blood flow in the brain.

In this research study we will be measuring blood flow in the brain when your head is elevated at various angles. We wish to determine what elevation angle is safe for patients who are recovering from subarachnoid hemorrhage.

You will not personally benefit from this study. However, future patients recovering from subarachnoid hemorrhage may benefit if this study demonstrates when and to what degree it is safe to raise the head during recovery.

Procedures

As part of your routine care, transcranial Doppler ultrasound tests are conducted nearly everyday while you are a patient in the intensive care unit. Transcranial Doppler tests indirectly measure brain blood flow and detect brain blood vessel spasm (vasospasm). After routine transcranial Doppler tests are conducted and if you are found to have mild or moderate blood vessel spasm (vasospasm), after surgery or after placement of coils in the aneurysm, and 3 to 14 days after the subarachnoid hemorrhage, the head of your bed will be positioned at 0, 20, 45, and 0 degrees for approximately 8-10 minutes at each position and brain blood flow measurements and routine vital signs and measurements will be recorded for 2-5 minutes at each position. The study will only be conducted once.

During the test the probe that was held in the technician's hand for the routine Doppler ultrasound test will be held by a headband that will be carefully placed on your head. The test will take an additional 40-60 minutes. Information will also be obtained from your chart and the computerized nurses' notes including history, diagnosis, previous diagnostic studies, nursing and medical treatments, vital sign flow sheets, nursing assessments, and progress notes. The Doppler information and vital signs taken from the bedside monitors will be recorded on computer disks.

If you are one of the first few patients who agrees to participate in this study, you may be part of a pilot study. The pilot study procedure is the same as the research study. However pilot study data may or may not be used in determining the final results of the study.

Risks, Stress, or Discomfort

Both the transcranial Doppler tests and head position changes up to 20 to 30 degrees are routine procedures for patients following aneurysmal subarachnoid hemorrhage in the intensive care unit. The additional one-time 40-60 minutes of ultrasound monitoring and the increase in the head elevation to 45 degrees are the only two changes from routine care. There is no evidence that the ultrasound used in the transcranial Doppler is harmful. Significant decreases in brain blood flow may occur with the head elevation; however brain injury is considered unlikely. If any signs of significant decreased brain blood flow occur during the study procedure, the study will be stopped immediately, your head will be lowered to the usual 20 to 30 degree position and you will continue to receive care in the intensive care unit. You will not participate in the study again.

Other Information

You may choose to participate or to not participate in the study. Participation is voluntary. Your care will not be affected by your decision. You will receive standard care regardless of your choice. Your name will not be used and no one other than the principal investigator and co-investigator will be able to identify you with your data. The data will be kept confidential and will be kept in a locked file for up to a year after the completion of the principal investigator's dissertation. The information will be used for the purpose of a doctoral dissertation and related professional publications which will be available at the University of Washington library. Data that are identified with you may be provided to qualified investigators for further analyses. You may refuse to participate or may withdraw from the study at any time. You will receive no compensation for participation in this study. In the event of a physical injury as the direct result of study procedures, you will be referred for treatment at no cost within the limits of the University of Washington Compensation Plan.

____

Signature of Investigator Date

Subject's Statement

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

____

Signature of Subject Date

Or

____

Signature of subject's legally Date
authorized representative

____

Relationship of representative to subject

Copies to: Subject
Investigator's File

Appendix C

Patient Information Sheet

Questions You May Have About Your Participation in the Nursing Research Study

The Effect of the Head-up Position on Cerebral Blood Flow in Mild or Moderate Vasospasm After Aneurysmal Subarachnoid Hemorrhage

Why is this study being conducted?

Patients recovering from subarachnoid hemorrhage as a result of an aneurysm rupture are bedridden for days or weeks following surgery. Unfortunately, prolonged bedrest is known to pose risks to the patient including, fainting with head elevation, pneumonia, the formation of blood clots, and constipation. Elevation of the head while in bed is thought to decrease the negative effects of prolonged bedrest. In addition, patients whose heads are elevated are able to get out of bed earlier and may make a faster recovery. However, the benefits of raising the head must be weighed against the risk of lowering blood flow in the brain.

In this research study, we will be measuring blood flow in the brain when your head is elevated at various angles. We wish to determine what elevation angle is safe for patients who are recovering from subarachnoid hemorrhage. You will not personally benefit from this study. However, future patients recovering from subarachnoid hemorrhage may benefit if this study demonstrates when and to what degree it is safe to raise the head during recovery

What will I experience during the study?

As part of your routine care, transcranial Doppler ultrasound tests are conducted nearly everyday while you are a patient in the intensive care unit. Transcranial Doppler tests indirectly measure brain blood flow and detect brain blood vessel spasm (vasospasm). After routine transcranial Doppler tests are conducted and if you are found to have mild or moderate blood vessel spasm, after surgery or placement of coils in the aneurysm, and 3 through 14 days after the subarachnoid hemorrhage, the head of your bed will be positioned at 0, 20, 45, and 0 degrees for approximately 8-10 minutes at each position and brain blood flow measurements and routine vital signs and measurements will be recorded for 2-5 minutes at each position. The study will only be conducted once.

During the test the probe that was held in the technician's hand for the routine Doppler ultrasound test will be held by a headband that will be carefully placed on your head. The test will take an additional 40-60 minutes. Information will also be obtained from your chart and the computerized nurses' notes including history, diagnosis, previous diagnostic studies, nursing and medical treatments, vital sign flow sheets, nursing assessments, and progress notes. The Doppler information and vital signs taken from the bedside monitors will be recorded on computer disks.

If you are one of the first few patients who agrees to participate in this study, you may be part of a pilot study. The pilot study procedure is the same as the research study. However pilot study data may or may not be used in determining the final results of the study.

Will I be harmed by participating in this study?

If at anytime you experience significant discomfort or indicate that you wish to stop the study, the study will be stopped. In addition, although significant harm is unlikely, the nurse researcher and ultrasound technician will stop the study immediately if any signs or symptoms indicate possible harm to you.

Who will know about my study results?

Your name will not be used on data collection sheets; you will be identified with a code number that links the data collection sheets with your medical records. You will not be identified by name in the publication or presentation of the results of this study. The results of this publication will be available at the University of Washington Health Sciences Library. Other qualified researchers may use the data for additional research to learn after the effect of head-up position on cerebral blood flow in blood vessel spasm after the entire study is completed.

Will there be any costs to me as a participant in the study?

No, there will be no costs to you as a participant in the study. In the event of a physical injury as the direct result of study procedures, you will be referred for treatment at no cost within the limits of the University of Washington Compensation Plan.

What if I have more questions?

If you have any other questions about this study, do not hesitate to let your nurse know. She will have a copy of your consent form on your chart and will know how to reach the nurse researcher 24 hours a day.

Thank you for participating in the study.

Appendix D

Critical Care Nurses' Information Sheet

To: ICU Nurses and Support Staff

From: Pat Blissitt, R.N., M.S.N., Ph.C., CCRN, CNRN, CCM, CS
Doctoral Candidate, University of Washington
School of Nursing

Re: Nursing Intervention Study: "The Effect of Head-up Position on
in Mild or Moderate Cerebral Vasospasm Following Aneurysmal
Subarachnoid Hemorrhage"

Date: January 25, 2000

In accordance with the requirements for completion of a Ph.D. in Nursing at the University of Washington School of Nursing, I will be conducting research for my dissertation in the near future. As you may or may not be aware. a scientific inquiry in regard to the effect of the head-up position on cerebral blood flow or cerebral blood flow velocity in cerebral vasospasm has never been published. As the above title indicates, I have chosen to study the effect of the head-up position on cerebral blood flow velocity in mild or moderate cerebral vasospasm following aneurysmal subarachnoid hemorrhage. Over the next 6 months to 1 year (or until my proposed sample size has been met) I will be collecting data in the Intensive Care Units Monday through Friday, most likely during the day shift.

Data collection will follow routine transcranial Doppler studies. Once informed consent has been obtained and the patient has met inclusion criteria, including mild or moderate vasospasm, post-operative or post-coiling and within 14 days following the aneurysmal subarachnoid hemorrhage, and without evidence of rebleed, data collection will add an additional 40-60 minutes to the routine TCD study. Each subject will undergo a 0-20-45-0 degree position sequence. Visitors will be held during data collection. Blood pressure lowering agents will also be held until data is collected, as appropriate. Each subject will only be studied once.

In addition to middle cerebral artery flow velocity, MCA to ICA ratio, and Autoregulatory Index, a number of other physiologic parameters will be monitored, including intracranial pressure, cerebral perfusion pressure, heart rate, oxygen saturation, arterial blood pressure, and pulmonary artery wedge pressure or central venous pressure. In accordance with Human Subjects Guidelines at the University of Washington, the patient or legal representative of the patient must initially be approached about the study by someone who has had previous contact with the patient or family. As a result, I will be asking the staff nurse taking care of the patient or the charge nurse to ask the patient or the family if I may discuss the study with them. I will also request your assistance, as needed, in positioning the patient and leveling and zeroing transducers as needed.

I will make every attempt to insure that any participation you may have in this study is as positive as possible. A copy of my research proposal is available upon request. In addition to my home phone, I will also be available by beeper, as indicated by the consent form which will be placed in the patient's medical record. The patient and/or their family will also receive a copy of an information sheet regarding this study. Thank you in advance for your assistance.

Appendix E

Inclusion Criteria Checklist

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

(To be completed during routine TCD monitoring, prior to data collection)

Patient ID number: ________________________

1. Consent form signed and on chart? Yes: __________ No: _________
2. Aneurysmal Subarachnoid Hemorrhage (vessel)
   ____
3. Mild or moderate vasospasm?
   Mild: __________
   Cerebral blood flow velocity (cm/sec): __________
   MCA:ICA ratio: __________
   Moderate: _____________
   Cerebral blood flow velocity (cm/sec): __________
   MCA:ICA ratio: __________
4. Date of Bleed? ____________________________________
5. Date of Clipping? ________________ Date of Coiling?______________
6. Any other Intervention (Sacrifice of blood vessel, wrapping) ?
   Yes: __________ No: _________ Type of intervention: _________________
7. Any other unclipped ruptured aneurysm ? Yes: _________ No: ___________
   Location (vessel(s)): _________________________________

Inclusion Criteria Checklist

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

(To be completed during routine TCD monitoring, prior to data collection)

Patient ID number: ______________________

8. History of rebleed? Yes: ________ No: _________ Date: _________

9. Name, dose and time of last medication(s) that may decrease blood pressure?

____

____

____

____

____

10. Current CPP? ____

Comments: ____

____

____

Appendix F

Pilot Study Data Collection Form

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

(To Determine Time from Position Change to Completion of Data Collection)

Patient ID number: ______________________

1 Get Consent Form Signed (if not previously signed).

2. Review Medical Record for Inclusion and Exclusion Criteria.

3. Routine TCD monitoring.

If subject is in mild (120-149 cm/sec and MCA:ICA ratio 3-5.9) or moderate (150-199 cm/sec and MCA:ICA ratio 3-5.9) vasospasm:

4. Attach elongated arterial transducer pressure tubing (for referencing at ear) or no arterial line, attach continuous noninvasive BP sensor to forearm;

5. Start stopwatch;

6. Position patient; verify with goniometer and staff nurse;

7. Position TCD headgear; insonate MCAs; verify with sonographer, record cerebral flow velocities (begin continuous recording); insonate extracranial ICAs;

8. Position arterial line transducers (external auditory meatus, phlebostatic axis); Zero, verify waveform;

9. Position PAP/CVP transducer (phlebostatic axis); Zero, verify waveform;

10. Zero Camino waveform, verify with staff nurse, record measurement (verify jack in correct data port);

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

Pilot Study Data Collection Form

(To Determine Time from Position Change to Completion of Data Collection)

Patient ID Number: ______________________

11. "Mark" continuous recording of cerebral blood flow velocity and mean cerebral blood flow. Record for 2-5 minutes. Record heart rate, SaO2, BP, ICP, CPP, PAP/PWP, CVP; verify with staff nurse;

12. Start stopwatch (record time) ______________________________.

To be completed at 4 positions: 0-20-45-0 degrees.

Comments:
____
____
____
____

Appendix G

Supplemental Data Collection Form

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

(To be used with HMC/UW Transcranial Doppler Report Form)

Patient ID number: ______________________

Date/Time: Date: __________ Start Time: __________ End Time: __________

Age: ____________________

Ethnicity: _______________

Gender: _________________

Date of Subarachnoid Hemorrhage: ____________________________

Location of Ruptured Aneurysm (vessel) ________________________________

Date of Aneurysm Clipping: ____________________________________

Does patient have other unclipped aneurysms? ____________________________

Sonographer (initials): ________________

Transcranial Dopplers used: Right and Left MCAs Serial Number __________

Right and Left ICAs Serial Number __________

Supplemental Data Collection Form

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

(To be used with HMC/UW Transcranial Doppler Report Form)

Patient ID number: ________________________

Intracranial Hemodynamics, Systemic Hemodynamics, and Vital Signs within 15 minutes prior to beginning study:

Parameter	Time	Value
Temperature (degrees centigrade)		
Heart rate (beats per minute)		
Respirations (breaths per minute)		
SaO2 (%)		
Arterial BP (include MAP) (mm Hg)		
ICP (mm Hg)		
CPP (mm Hg)		
PAP (mm Hg)		
CVP (mm Hg)		
PWP (mm Hg)		

Current Medications:

Medication	Dose (strength and frequency)	Time of last administration

Supplemental Data Collection Form (continued)

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

(To be used with HMC/UW Transcranial Doppler Report Form)

Patient ID number: ______________________

Record time and values where indicated

Right MCA	0 degrees	20 degrees	45 degrees	0 degrees
Verification of head position with goniometer				
ICP zeroed (jack in correct data port)				
Arterial catheter transducer #1 zeroed at phlebostatic axis or continuous cuff (wrist) at heart level				
Arterial catheter transducer #2 zeroed at external auditory meatus (cerebral blood pressure approximation)				
Pulmonary artery or CVP catheter zeroed at phlebostatic axis				
Heart rate (beats per minute)				
Respirations (breaths per minute)				
Arterial BP (include MAP) (mm Hg)				
ICP (mm Hg)				
CPP (mm Hg)				
PAP (mm Hg)				
CVP (mm Hg)				
PWP (mm Hg)				
SaO2 (%)				
Cuff BP (include (MAP) (mm Hg)				
Right MCA flow velocity (cm/sec)				
Right MCA:ICA ratio				
Right Autoregulatory Index				

Supplemental Data Collection Form

Head-Up Position and Aneurysmal Subarachnoid Hemorrhage Vasospasm

(To be used with HMC/UW Transcranial Doppler Report Form)

Patient ID Number: ____________________

Left MCA	0 degrees	20 degrees	45 degrees	0 degrees
Verification of head position with goniometer				
ICP zeroed (jack in correct data port)				
Arterial catheter transducer #1 zeroed at phlebostatic axis or continuous cuff (wrist) at heart level				
Arterial catheter transducer #2 zeroed at external auditory meatus (cerebral blood pressure approximation)				
Pulmonary artery or CVP catheter zeroed at phlebostatic axis				
Respirations (breaths per minute)				
Arterial BP (include MAP) (mm Hg)				
ICP (mm Hg)				
CPP (mm Hg)				
PAP (mm Hg)				
CVP (mm Hg)				
PWP (mm Hg)				
SaO2 (breaths per minute)				
Cuff BP (include mean) mm Hg				
Left MCA flow velocity (cm/sec)				
Left MCA:ICA ratio				
Left MCA Autoregulatory Index				

Comments:

____

____

VITA

Patricia A. Blissitt, R.N., M.S.N., CCRN, CNRN, CCM, CS

Career Summary

Patricia A. Blissitt has practiced neuroscience nursing for the past 26 years in a number of different positions, including acute care and critical care staff nurse, nurse manager, neurosurgeon's nurse in the outpatient/inpatient setting and first surgical assistant, clinical nurse specialist, trauma nurse coordinator, case manager, and adjunct faculty at three schools of nursing.

Education

1976: Bachelor of Science in Nursing, University of Tennessee, College of Nursing, Memphis, Tennessee (graduated with high honors)

1985: Master's of Science in Nursing, University of Tennessee, College of Nursing, Memphis, Tennessee (medical-surgical clinical nurse specialist track)

Specialty Certification

1986 - Currently: Certified Critical Care Nurse, American Association of Critical Care Nurses/American Association of Critical Care Nurses Certification Corporation (CCRN)

1987 - Currently: Certified Neuroscience Nurse, American Association Neuroscience Nurses/American Board of Neuroscience Nurses (CNRN)

1993 - Currently: Certified Case Manager. Rehabilitation Specialist Commission for Case Manager Certification (CCM)

1999 - Currently: Certified Clinical Specialist in Medical-Surgical Nursing, American Nurses' Credentialing Center Commission on Certification/American Nurses Association

Selected Publications

August 2001: Sleep, Memory, and Learning. <u>Journal of Neuroscience Nursing,</u> <u>33</u>(4), 208-215.

February 2001: Case Management and Outcomes Management in Acute Traumatic Brain Injury. CareManagement, 7(1), 26-33.

December 1996: Physiologic Response to Generalized Convulsive Status Epilepticus (abstract). Journal of Neuroscience Nursing, 28(6), 396.

1995: “Cerebrovascular Accident” in N. Urban, K. Greenlee, J. Krumberger, et al. (eds) Guidelines for Critical Care Nursing. St. Louis: C.V. Mosby. 66-77.

October 1992: Ticlopidine Hydrochloride. Journal of Neuroscience Nursing. 24(5), 296-300.

June 1992: Pituitary tumor, hypophysectomy, and diabetes inspidus (abstract). Journal of Post Anesthesia Nursing, 7(3), 209.

September 1990: Nutritional management in acute spinal cord injury” in J. Sullivan (ed.) Spinal Cord Injury in Critical Care Nursing Clinics of North America, 2(3), 375-384.

April 1986: Nursing management of diabetic peripheral neuropathies. Journal of Neuroscience Nursing, 18(2), 81-85.