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Cerebrovascular Dynamics With Head-of-Bed Elevation in Patients With Mild or Moderate Vasospasm After Aneurysmal Subarachnoid Hemorrhage

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• Background *In patients with aneurysmal subarachnoid hemorrhage, elevation of the head of the bed during vasospasm has been limited in an attempt to minimize vasospasm or its sequelae or both. Consequently, some patients have remained on bed rest for weeks.*
• Objectives *To determine how elevations of the head of the bed of 20º and 45º affect cerebrovascular dynamics in adult patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage and to describe the response of mild or moderate vasospasm to head-of-bed elevations of 20º and 45º with respect to variables such as grade of subarachnoid hemorrhage and degree of vasospasm.*
• Methods *A within-patient repeated-measures design was used. The head of the bed was positioned in the sequence of 0º-20º-45º-0º in 20 patients with mild or moderate vasospasm between days 3 and 14 after aneurysmal subarachnoid hemorrhage. Continuous transcranial Doppler recordings were obtained for 2 to 5 minutes after allowing approximately 2 minutes for stabilization in each position.*
• Results *No patterns or trends indicated that having the head of the bed elevated increases vasospasm. As a group, there were no significant differences within patients at the different positions of the head of the bed. Utilizing repeated-measures analysis of variance,* P *values ranged from .34 to .97, well beyond .05. No neurological deterioration occurred.*
• Conclusions *In general, elevation of the head of the bed did not cause harmful changes in cerebral blood flow related to vasospasm. (American Journal of Critical Care. 2006;15:206-216)*

Stroke is the leading cause of disability and the third leading cause of death in the United States.[1] Although only 3% to 7% of all strokes are caused by subarachnoid hemorrhage (SAH),[1,2] the loss of potential life years as a result of SAH before the age of 65 years is comparable to the loss associated with ischemic stroke.[3] Furthermore, more than 50% of patients who survive strokes caused by SAH have a permanent disability.[4]

In patients with acute stroke, including those with SAH, a delicate balance exists between preventive measures to decrease the consequences of immobilization and the need to support cerebral perfusion to prevent secondary brain injury. The nursing intervention of changing the position of the head of the bed to a full sitting position illustrates this therapeutic dilemma. Positioning regimens are typically based on tradition and consensus at individual institutions. Few regimens are actually based on physiological responses, specifically changes in cerebral blood flow (CBF), to changes in position. Also, few regimens include determining

which patients are at risk for impaired cerebral perfusion when the bed is raised to a sitting position.

Like other patients with stroke, patients with aneurysmal SAH are at risk for the consequences of immobility. Care of SAH patients is further complicated by cerebral vasospasm that may occur from day 3 to day 21 after the hemorrhage and has been reported to occur up to 28 days after the hemorrhage.[5,6] Cerebral vasospasm may impair cerebral perfusion, resulting in secondary neuronal ischemia and infarction.

Background

A number of factors must be considered when the effect of head-of-bed position in vasospasm after aneurysmal SAH is investigated. The systemic effects of imposed bed rest are one possible influence in regard to intracranial dynamics. The intracranial dynamics themselves are a complex interplay of factors, including intracranial pressure (ICP), cerebral perfusion pressure (CPP), autoregulation, CBF, and vasospasm. Each or all of these together may affect a patient's response to head-of-bed elevation after aneurysmal SAH.

The deconditioning effects of bed rest are well documented. Potentially detrimental physiological changes begin in the first 1 to 2 days of bed rest. Virtually every body system is affected.[7-12] Regardless of the duration of bed rest, having the head of the bed in the down position places patients with neurological impairment at substantial risk for aspiration of oral and/or gastric secretions and enteral feedings. Intubation and mechanical ventilation alone increase the risk of pneumonia 10% to 25%.[13-18]

At the time of the aneurysmal rupture, the CPP is drastically reduced and is inadequate to perfuse brain cells.[19] The CPP is calculated clinically as the mean arterial blood pressure (MAP) minus the ICP. According to traumatic brain injury guidelines,[20-22] normal CPP is 60 to 100 mm Hg. No guidelines for normal CPP after aneurysmal SAH exist. The guidelines of the American Stroke Association for treatment of patients with aneurysmal SAH are currently under revision. In addition to increased volume in the subarachnoid space, extravasated blood and blood clots in the ventricles may block the outflow and reabsorption of the cerebrospinal fluid and further contribute to increased ICP.[23-26]

Although incompletely understood, 2 other mechanisms are thought to contribute to the initial increase in ICP: impaired autoregulation and a compensatory increase in systemic blood pressure. Autoregulation, the ability of the cerebral blood vessels to maintain constant adequate blood flow despite variations in CPP, is only operational at a systemic MAP of approximately 50 to 170 mm Hg and is impaired at low CPPs.[26-28] The initial increase in ICP may also be accompanied by a compensatory increase in systemic blood pressure.[18,28,29]

The initial clinical features of patients with SAH correlate strongly with the patients' outcome. The Hunt and Hess Scale[30] is a universally accepted and widely used instrument. With this instrument, a patient's clinical condition is scored on a scale of I (asymptomatic or mild headache) to V (comatose). Grades I and II are associated with the best outcomes; grades IV and V, with the worst outcomes.

Cerebral edema increases during the first 24 to 72 hours after the hemorrhage and may result in additional episodes of increased ICP for an indefinite period.[22,23,31] Other causes of increased ICP include cerebral edema due to surgical manipulation or infarction and increased intravascular volume due to hypervolemic hemodilution therapy with or without controlled hypertension for vasospasm.[32,33]

The impairment in autoregulation may continue well beyond the time of the initial hemorrhage. The upper and lower limits of autoregulation are shifted toward higher systemic blood pressures, particularly during vasospasm.[34] A continued reduction in CBF can occur after the initial precipitous decrease associated with increased ICP at the time of aneurysmal rupture.[20-22] Although CBF is generally at its lowest 2 weeks after hemorrhage, it typically remains lower than normal for at least 3 weeks after the hemorrhage.[35] Factors that contribute to the lower CBF include systemic hypotension, increased ICP, cerebral vasospasm, and decreased cerebral metabolism.[36-40]

Cerebral vasospasm is defined as sustained arterial constriction and sometimes results in a delayed ischemic neurological deficit or a "second stroke" after aneurysmal SAH.[33,41] Sustained arterial constriction may result in cerebral ischemia and infarction. Vasospasm is not restricted to the cerebral vessel that ruptured; it commonly includes other vessels throughout the cerebral circulation.[6] The onset of cerebral vasospasm typically occurs between days 3 and 5 after aneurysmal SAH, is maximal between days 5 and 14, and gradually resolves during days 14 to 28, with up to day 21 being the most reported duration.[5,6] Cerebral vasospasm is often differentiated as clinical, radiographic, or both. Clinical vasospasm is discernable on clinical assessment and correlates with the areas perfused by the vasospastic vessels. Radiographic vasospasm is evident on angiograms but may or may not be clinically apparent. Patients who are hypotensive, hypovolemic, and/or critically ill are considered at greatest risk for cerebral vasospasm.[42,43]

No research has been published specifically on the effect of head-of-bed elevation on CBF in vasospasm

after aneurysmal SAH. However, since 1977, the effect of elevating the head of the bed on cerebral perfusion in critically ill patients with intracranial abnormalities has been investigated in 16 studies.[44-59] In general, these studies had small sample sizes, from 4 to 38 patients. Samples consisted primarily of patients with various traumatic brain injuries, but some studies included patients with brain tumors, ischemic stroke, or hydrocephalus or patients who had experienced near-drowning. Patients with SAH were included in 6 studies.[48,50-52,54,55] However, much of the SAH was due to trauma, the presence or absence of vasospasm was not reported, and data were aggregated and analyzed with data on patients with other diagnoses. The systematic reviews of the effect of body position on cerebral perfusion have largely excluded patients with SAH.[60,61]

Purpose

The overall goal of this study was to explore how elevating the head of the bed affects cerebral blood flow in patients with vasospasm after aneurysmal SAH. The specific aims were to

- determine the effect of elevations of 20º and 45º on cerebrovascular dynamics in patients with mild or moderate cerebral vasospasm after aneurysmal SAH and
- describe the response of mild or moderate vasospasm to head-of-bed elevations of 20º and 45º with respect to variables such as grade of subarachnoid hemorrhage and degree of vasospasm.

Methods

The investigation was a prospective within-patient, repeated-measures study. The head of the bed was positioned in the sequence of 0º-20º-45º-0º in a convenience sample of 20 adult patients who were 18 to 99 years old and had mild or moderate vasospasm between days 3 and 14 after aneurysmal subarachnoid hemorrhage. Multiple cerebrovascular and hemodynamic variables were measured before and after each change in position. The study was approved for the protection of human subjects by the University of Washington Institutional Review Board and adhered to the ethical standards set forth in the Helsinki Declaration of 1975. Informed consent was obtained from eligible participants or their legal next of kin or both.

Mild vasospasm was defined as a mean flow velocity (MFV) of 120 to 149 cm/s in the middle cerebral artery (MCA) and an MCA to internal carotid artery (ICA) MFV ratio (MCA/ICA MFV) of 3.0 to 5.9 within 3 to 14 days after aneurysmal SAH. Moderate vasospasm was defined as an MFV of 150 to 199 cm/s in the MCA and an MCA/ICA MFV ratio of 3.0 to 5.9 within 3 to 14 days after aneurysmal SAH.[62-65]

All participating patients had had surgery or placement of coils in the aneurysmal sac, because surgical or interventional neuroradiological procedures within the first 72 hours of the aneurysmal hemorrhage are the usual treatment at Harborview Medical Center in Seattle, where the data were collected. The presence and degree of vasospasm were determined via routine transcranial Doppler studies that were done on all patients who had had aneurysmal SAH.

Criteria for exclusion from the study included SAH due to traumatic brain injury or rupture of an arteriovenous malformation or both; the absence of vasospasm as determined by transcranial Doppler imaging; severe cerebral vasospasm as determined by transcranial Doppler imaging (MCA MFV ≥ 200 cm/s and MCA/ICA MFV ratio ≥ 6.0); the presence of ruptured aneurysms without surgical or interventional neuroradiological intervention; and a baseline CPP less than 70 mm Hg.

MCA MFV, MCA/ICA MFV ratio, heart rate, oxygen saturation, and MAP were measured in each position. When available, measurements of ICP, CPP, pulmonary artery pressure, pulmonary artery wedge pressure (PAWP), and/or central venous pressure (CVP) were obtained as well. ICP, CPP, vital signs, and hemodynamic parameters per Camino (Integra NeuroSciences, Plainsboro, NJ) ICP, Spacelabs (Spacelabs Medical, Inc, Issaquah, Wash) cardiac, and Baxter-Edwards (Edwards Lifesciences, Irvine, Calif) hemodynamic monitors were part of the standard care of patients with SAH. CPP was calculated by using data obtained from Camino ICP monitors and radial arterial catheters or a Nellcor (Nellcor, Pleasanton, Calif) continuous noninvasive blood pressure monitor.

When patients were lying supine and flat (ie, baseline 0º position and the second 0º position) and at each elevation of the head of the bed (20º and 45º), radial arterial catheter transducers were leveled with the heart (at the phlebostatic axis) and the head (at the external auditory meatus), and differences in the systolic, diastolic, and mean arterial pressures were noted. Arterial catheter transducers have been leveled with the head (at the external auditory meatus) in scientific investigations as a estimate of a patient's intracranial blood pressure.[49-51] When patients did not have radial arterial catheters in place, continuous noninvasive blood pressures were obtained by using the Nellcor N-Cat continuous noninvasive blood pressure monitor. The cuff at the brachial artery was leveled at the phlebostatic axis for accuracy.[66]

The MCA MFV was derived by using the Multi-Dop X4 transcranial Doppler unit (DWL Elektronische Systems GmbH, Sipplingen, Germany) with headgear

for continuous monitoring. The MultiDop X4 unit provides continuous graphic and digital display and has a continuous recording feature that includes simultaneous measurement of bilateral MCA flow velocities, MAP, and ICP. The bilateral ICA blood flow velocities were determined by using a Neuroguard transcranial Doppler unit (Medasonics, Fremont, Calif). The right and left MCAs were insonated separately and simultaneously during the study procedure. The right and left ICAs were insonated separately with a handheld probe.

Before each data collection, proper positioning of the transcranial Doppler imaging headgear for accurate insonation and clear signal was verified by the investigator (P.A.B.) and an experienced sonographer. All hemodynamic monitoring transducers (ie, those used to measure arterial blood pressure, CVP, and pulmonary artery pressure) were placed at the phlebostatic axis and zeroed. In addition, the arterial catheter transducer was placed at the external auditory meatus for measurements of arterial blood pressure at the patient's head. The phlebostatic axis and external auditory meatus were verified by 2 experienced critical care nurses, the researcher (P.A.B.), and a staff nurse. Transducers were rezeroed at each change in position of the head of the bed. Pulmonary artery pressure, PAWP, and CVP were measured at end-expiration and were determined by interpreting waveforms on paper recordings. The 2 experienced critical care nurses, the researcher, and the staff nurse verified waveform interpretation.

Each change in position of the head of the bed was verified with a goniometer. The angle of the head of the bed was verified by an experienced critical care nurse and the researcher or by a transcranial Doppler sonographer and the researcher. For each patient, baseline vital signs were obtained within 15 minutes before the study procedure was started. Data including grade of aneurysmal SAH per the Hunt and Hess Scale, days since SAH, current medications and intravenous fluids, and fluid balance were obtained from the clinical records.

Data were collected on each patient at 4 positions of the head of the bed in the sequence given, 0º, 20º, 45º, and 0º, with a pause of approximately 5 to 10 minutes at each position. The first 2 minutes of each pause was for stabilization of hemodynamic and intracranial parameters and leveling and zeroing of hemodynamic and intracranial monitors. Then, ICP, CPP, MCA flow velocity, ICA flow velocity, hemodynamic parameters (heart rate, arterial blood pressure, and CVP or PAWP), and oxygen saturation were determined for 2 to 5 minutes. The position of 0º was considered baseline. The order of head-of-bed positions was fixed at 0º, 20º, 45º, and 0º because this sequence was a clinically feasible order and the return to 0º might differentiate changes with time from data changes associated with head-of-bed elevations. Each patient was studied only once.

Continuous recordings were obtained in each position for 2 to 5 minutes. Hard-copy recordings showing trends were used for statistical interpretation for heart rate, oxygen saturation, CVP or PAWP, and ICA flow velocity. Data on MCA flow velocity, ICP, and arterial blood pressure were available on diskette from the MultiDop X4 transcranial Doppler unit. ICA flow velocity was obtained from hard copies of recordings obtained with the Neuroguard transcranial Doppler unit.

The study procedure took 30 to 40 minutes. Any routine nursing procedures done during the study procedure were noted, including administration of medications or intravenous fluids that might have altered blood pressure, systemic hemodynamic parameters, or intracranial dynamics. Each patient had a neurological assessment before and immediately after the study procedure to determine any detectable change in neurological status. The formal stopping rule for change in status was never invoked.

Data Analysis

Descriptive and inferential statistics were computed. The level of significance was set at $P < .05$. Data from each position change were used to address both aims of the study. To determine the effect of elevations of 20º and 45º in patients with mild or moderate cerebral vasospasm after aneurysmal SAH, ICP, CPP, MCA MFVs, and MCA/ICA MFV ratios were determined at each of the 4 positions: 0º, 20º, 45º, and 0º. Means, medians, SDs, and ranges (minimum to maximum) of right and left MCA MFVs and of MCA/ICA MFV ratios for patients at each of the 4 positions were compared. Confidence intervals of 95% were constructed to determine if differences in MCA MFVs and MCA/ICA MFV ratios between 0º, 20º, 45º, and 0º were significant. A 1-way repeated-measures analysis of variance was used to determine if significant effects existed for position in MCA MFVs and MCA/ICA MFV ratios across head-of-the-bed positions.

MCA MFVs and MCA/ICA MFV ratios were analyzed by using 2 different strategies. In both instances, right and left MCA MFVs and right and left MCA/ICA ratios were analyzed separately. However, the first data analysis included all 20 right MCA MFVs and all 20 right MCA/ICA MFV ratios and all 20 left MCA MFVs and all 20 left MCA/ICA MFV ratios. The second data analysis included only those vessels that met the criteria for vasospasm: right MCA MFVs 120 cm/s or greater or right MCA/ICA MFV

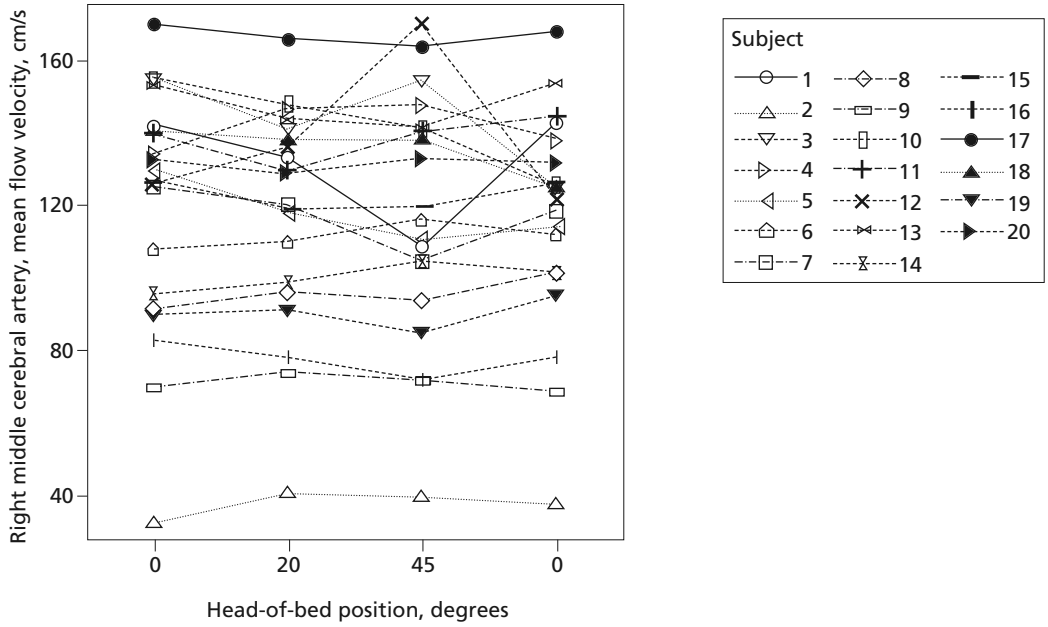

Figure 1 Mean flow velocities in the right middle cerebral artery at 0°, 20°, 45°, and 0° head-of-bed positions in 20 patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage.

ratios 3.0 or greater and left MCA MFVs 120 cm/s or greater or left MCA/ICA MFV ratios 3.0 or greater.

Results

Characteristics of Patients

The sample consisted of 15 women and 5 men. Of these, 17 were white, 2 were Native American, and 1 was Filipino. Thirteen were studied on day 3 through 7 after SAH, and 7 were studied on day 8 through 14 after SAH. The patients were 36 to 67 years old: 1 patient was between 30 and 39 years old, 10 were between 40 and 49 years old, 6 were between 50 and 59 years old, and 3 were between 60 and 69 years old. The mean age was 50.25 years (SD 7.97). Hunt and Hess scores were grades I or II for 13 patients, grade III for 1 patient, and grades IV or V for 4 patients. Scores were not documented for 2 patients.

Effects of Position

All MCA MFVs and MCA/ICA MFV ratios are illustrated in Figures 1 through 4.

Descriptive statistics, measures of central tendency and variability, 95% CIs, and percent change from baseline did not reveal any patterns or trends toward impaired CBF velocity with elevation in the head of the bed (Table 1). Repeated-measures analysis of variance did not indicate any significant effect of position for right or left MCA MFVs or right or left MCA/ICA MFV ratios. Furthermore, in the repeated-measures analysis of variance for the total sample, neither the elevated right and left MCA MFVs (≥120 cm/s) nor the elevated right and left MCA/ICA MFV ratios (≥3.0) through the 4 positions of 0º, 20º, 45º, and 0º yielded statistically significant within-patient findings. All *P* values were greater than .05 (Table 2). In 1 patient, at 45º, consistent with elevation, the right MCA MFV increased from a value indicative of mild vasospasm (125 cm/s) to a value indicative of moderate vasospasm (170 cm/s), and the left MCA MFV increased from normal (117 cm/s) to a value indicative of moderate vasospasm (161 cm/s). However, the right MCA/CA MFV ratio remained consistent with mild-to-moderate vasospasm, and the left MCA/ICA MFV ratio fluctuated from mild-to-moderate to normal and then back to the mild-to-moderate category.

In order to describe mild or moderate vasospasm at head-of-bed elevations of 20º and 45º with respect to variables such as grade of SAH and degree of vasospasm, patients were grouped according to changes in severity of vasospasm, specifically mild to moderate, mild to severe, and moderate to severe. Data including increasing severity of vasospasm, increasing MCA MFV, and/or MCA/ICA MFV ratio were analyzed for any grouping variables that might be predictive of its incidence and presence. No trends or patterns were found.

After the MCA MFVs and MCA/ICA MFV ratios were analyzed, the ICP and CPP data were examined. As expected, ICP consistently decreased when the head of

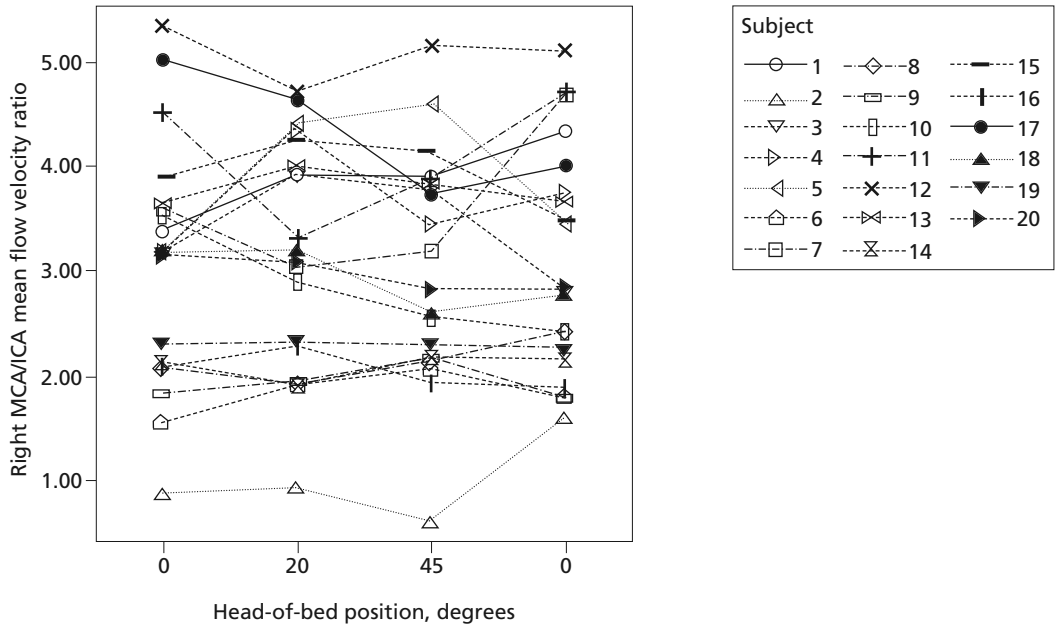

Figure 2 Ratio of mean flow velocity in right middle cerebral artery (MCA) to mean flow velocity in right internal carotid artery (ICA) at 0°, 20°, 45°, and 0° head-of-bed positions in 20 patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage.

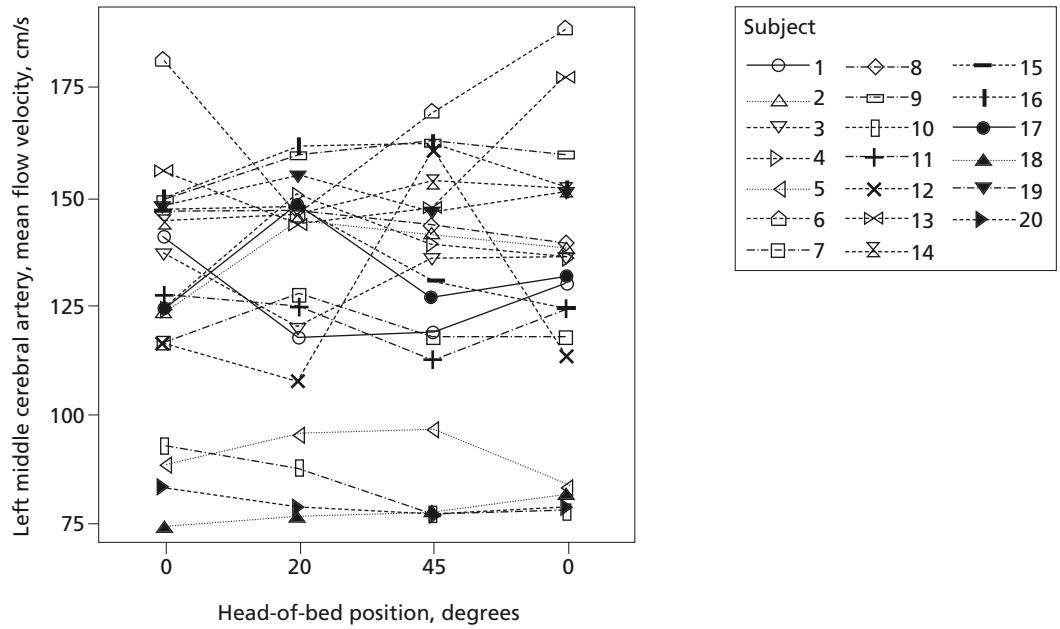

Figure 3 Mean flow velocities in the left middle cerebral artery at 0°, 20°, 45°, and 0° head-of-bed positions in 20 patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage.

the bed was elevated and then increased when the head of the bed was returned to 0º. For patients as a group, the repeated-measures analysis of variance of the ICP at 0º, 20º, 45º, and 0º indicated a significant effect for the position of the head of the bed ($F_{3,33}=44.08$, $P<.001$).

A function of the MAP and the ICP, the CPP was statistically variable depending on the method of measurement: the arterial catheter with the transducer at the phlebostatic axis, the arterial catheter with the transducer at the level of the external auditory meatus, or a continu-

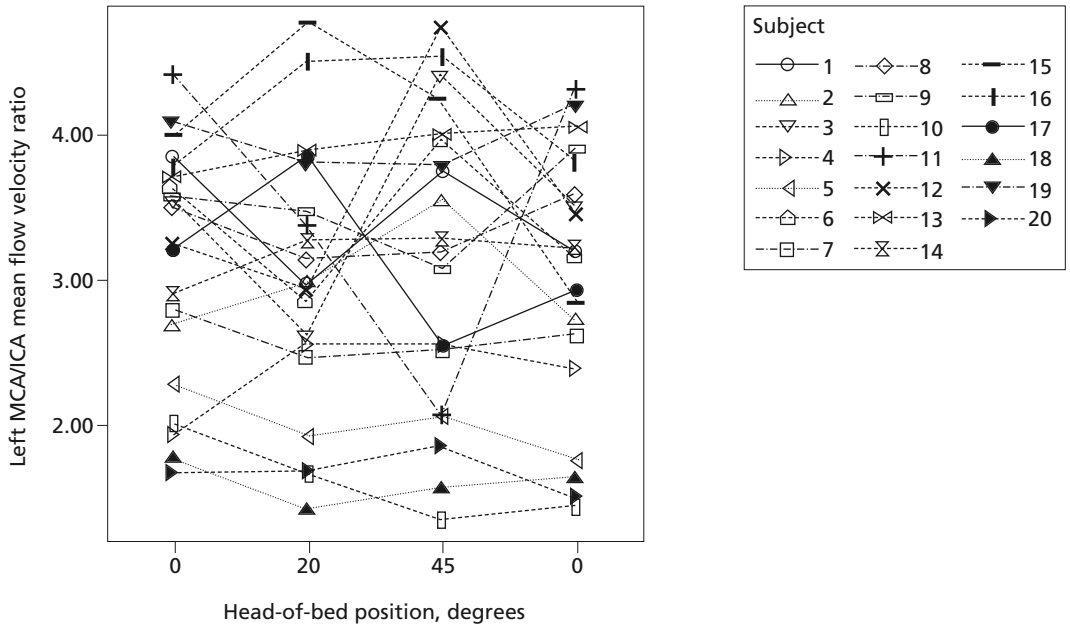

Figure 4 Ratio of mean flow velocity in left middle cerebral artery (MCA) to mean flow velocity in left internal carotid artery (ICA) at 0°, 20°, 45°, and 0° head-of-bed positions in 20 patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage.

ous noninvasive tonometric cuff. A significant effect for position on CPP was found when the MAP was measured at the phlebostatic axis ($F_{3,21} = 11.091$, $P < .001$). However, when the MAP was measured at the external auditory meatus or with the cuff at the level of the heart, the effects of head-of-bed position on CPP were not significant by repeated-measures analysis of variance.

Discussion

Changes in the position of the head of the bed in the sequence of 0º, 20º, 45º, and 0º after aneurysmal SAH did not result in statistically significant increases or decreases in cerebral vasospasm in the patients as a group in this study. However, other information obtained in the study warrants further discussion of the patients, the physiological effects of elevating the head of the bed, technology, implications for nursing practice, and future nursing research.

Patients

More women than men experience aneurysmal SAH.[67] In our study, the ratio was 3:1. The mean age of the patients in our sample was 50.25 years old (SD 7.97), slightly younger than the mean ages of 51.4 and 57 years in other investigations.[67,68] In our investigation, 65% of the patients were studied on days 3 to 7 after the aneurysmal SAH had occurred and 35% were studied on days 8 through 14 after aneurysmal SAH had occurred. Although vasospasm typically starts 3 days after aneurysmal SAH, an increase in severity occurs about 5 to 14 days after the event.

As indicated by positive intake and output fluid balances and CVP and PAWP measurements, all the patients in our sample were euvolemic to hypervolemic at the time of the study. All were receiving hourly intravenous fluid at rates of at least 150 to 200 mL/h, 5 were receiving fluid boluses on the basis of CVP or PAWP values, and 3 were receiving phenylephrine (Neo-Synephrine) infusions. Although the effectiveness of hypervolemic hemodilution with or without controlled hypertension is controversial in the management of patients with vasospasm,[6,32,69] this treatment may have blunted the vasospastic response to elevating the head of the bed. All the patients also routinely received nimodipine (Nimotop) beginning at the time of admission. The exact mechanism of nimodipine is unknown; however, this cerebroselective pharmacological agent may have enhanced collateral circulation.[25,70-72] A total of 4 patients had had MCA angioplasty, 2 for previously clinically significant or severe vasospasm or both and 2 prophylactically as part of a multicenter study. The vessel involved in the angioplasty, with or without intra-arterial injection of papaverine, may have responded differently to elevation of the head of the bed in patients with recurrent vasospasm.[73] However, compared with the responses of the other patients to elevations of 20º

Table 1 Descriptive statistics for right and left MCA MFVs and right and left MCA/ICA MFV ratios

Parameter	Position of head of bed, degrees	Mean	Median	SD	Range (minimum to maximum)	95% CI
Right MCA MFVs, all, cm/s	0	120	129	34	137 (33-170)	104-136
	20	118	125	30	125 (41-166)	104-132
	45	118	118	34	130 (40-170)	102-134
	0	117	124	30	130 (38-168)	102-131
Right MCA MFVs ≥120, cm/s	0	141	140	14	45 (125-170)	133-149
	20	136	136	14	48 (118-166)	128-144
	45	137	140	21	65 (105-170)	124-149
	0	134	126	15	54 (114-168)	124-143
Right MCA/ICA MFV ratios, all	0	3.08	3.18	1.13	4.43 (.90-5.33)	2.55-3.61
	20	3.14	3.16	1.11	3.76 (.95-4.71)	2.62-3.66
	45	3.05	3.01	1.10	4.56 (.62-5.15)	2.54-3.56
	0	3.10	2.83	1.08	3.47 (1.62-5.09)	2.59-3.61
Right MCA/ICA MFV ratios ≥3.0	0	3.75	3.52	0.74	2.17 (3.16-5.33)	3.31-4.20
	20	3.82	3.92	0.64	1.81 (2.90-4.71)	3.44-4.21
	45	3.66	3.76	0.75	2.59 (2.56-5.15)	3.21-4.11
	0	3.69	3.67	0.84	2.67 (2.42-5.09)	3.18-4.20
Left MCA MFVs, all, cm/s	0	129	133	27	107 (75-182)	116-142
	20	130	145	27	85(77-162)	117-142
	45	131	138	29	93 (77-170)	117-144
	0	131	134	31	111 (78-189)	115-147
Left MCA MFVs ≥120, cm/s	0	140	143	17	65 (117-182)	131-149
	20	141	146	16	54 (108-162)	132-149
	45	142	143	17	57 (113-170)	133-152
	0	142	138	21	75 (114-189)	131-153
Left MCA/ICA MFV ratios, all	0	3.13	3.38	0.82	2.73 (1.68-4.41)	2.74-3.51
	20	3.00	2.99	0.91	3.34 (1.43-4.77)	2.58-3.43
	45	3.15	3.24	1.03	3.39 (1.35-4.74)	2.66-3.63
	0	3.01	3.18	0.90	2.87 (1.44-4.31)	2.59-3.43
Left MCA/ICA MFV ratios ≥3.0	0	3.58	3.60	0.46	1.71 (2.70-4.41)	3.31-3.84
	20	3.56	3.32	0.63	2.16 (2.61-4.77)	3.09-3.82
	45	3.65	3.76	0.76	2.69 (2.05-4.74)	3.21-4.09
	0	3.49	3.47	0.26	1.58 (2.73-4.31)	3.20-3.78

Abbreviations: ICA, internal carotid artery; MCA, middle cerebral artery, MFV, mean flow velocity.

and 45º, the responses of the patients who had angioplasty did not differ. Patients who had been on bed rest longer than 7 days did not respond differently from those on a less lengthy period of bed rest.

Physiological Effects of Elevation of the Head of the Bed

MFVs and MFV ratios consistent with severe vasospasm did not occur in any of the 20 patients during the study. None of the patients had deterioration in neurological status during or after the study procedure. As noted earlier, 1 patient did have a change in vasospasm status from mild to moderate, but without neurological changes.

As expected, ICP significantly and consistently decreased when the head of the bed was elevated and increased when the head of the bed was returned to 0º. The changes in CPP measured with the transducer at the phlebostatic axis were statistically significant; however, changes in CPP measured with the transducer leveled at the head (external auditory meatus) were not statistically significant. CBF is influenced by a number of physiological factors, including ICP, CPP, vasospasm, and autoregulatory status. In our study, we focused primarily on vasospasm, with some attention to ICP and CPP.

The overall response to elevation of the head of the bed and individual responses to the elevation were both noted. Although severe vasospasm did not develop in any patient during the study procedure, some patients had increases in MCA MFVs and MCA/ICA MFV ratios that indicated changes from normal to mild or moderate vasospasm or maintenance of mild or moderate vasospasm or both. Perhaps, for patients who experience increases in MFVs when the head of the

Table 2 Repeated-measures analysis of variance for right and left MCA MFVs and right and left MCA/ICA MFV ratios (within-patients effects)

Parameter	F (df)	P*
All right MCA MFVs	0.589 (3, 57)	.62
Right MCA MFVs ≥120 cm/s	1.149 (3, 36)	.34
All right MCA/ICA MFV ratios	0.165 (3, 57)	.92
Right MCA/ICA MFV ratios ≥3.0	0.261 (3, 36)	.85
All left MCA MFVs	0.075 (3, 57)	.97
Left MCA MFVs ≥120 cm/s	0.193 (3, 45)	.90
All left MCA/ICA MFV ratios	0.482 (3, 57)	.70
Left MCA/ICA MFV ratios ≥3.0	0.318 (3, 39)	.81

Abbreviations: ICA, internal carotid artery; MCA, middle cerebral artery; MFV, mean flow velocity.
* $P < .05$ significant.

bed is elevated, increasing the volume or frequency of fluid boluses to maintain higher CVP or PAWP before or during head-of-bed elevation is warranted.

Technology

In previous trials,[74,75] the sensitivity of transcranial Doppler imaging for detecting vasospasm in the MCA ranged from 38% to 86%, and the specificity ranged from 94% to 100%. In our study, the MCA MFVs were measured continuously, and the ICA MFV, used to determine the MCA/ICA MFV ratio, was measured intermittently. Signal interference between the 2 Doppler probes prohibited simultaneous recording of the 2 MFVs. In addition, the headband holding the ultrasound probes allows insonation access to the MCAs only. Obtaining information on other cerebral vessels was not possible. The number and type of physiological parameters measured also varied from patient to patient, depending on the invasive devices in place at the time of the study. Some patients did not have an arterial catheter or an ICP monitor or either. Other measures of CBF or oxygenation were not available, including measurement of jugular bulb oxygen saturation and regional brain tissue oxygenation.

Each participant was given 2 minutes in each position for stabilization in status, and data were collected for approximately 2 to 5 minutes. Temporary compensatory mechanisms may not have been thoroughly exhausted at each level. Additional time at 20º and 45º might have yielded different findings. This study took place in an environment in which aggressive medical management of patients with aneurysmal SAH at risk for vasospasm is the standard of care; thus the usefulness of our findings in environments in which less aggressive medical management is used is unknown.

Implications for Nursing Practice

Our results elucidate at least 2 implications for nursing practice. Although the effect of elevation of the head of the bed in patients with mild or moderate vasospasm after aneurysmal SAH was not statistically significant within patients as a group and none of the patients experienced a deterioration in neurological status, 1 patient did have an increase in MCA MFVs and MCA/ICA MFV ratios from values indicative of mild vasospasm to values indicative of moderate vasospasm. If the head of the bed is elevated, monitoring of patients' responses is warranted. Second, prophylactic hypervolemic hemodilution with or without controlled hypertension for increased vasospasm before or during the time the head of the bed is elevated may be indicated for patients who are also at risk for neurological deterioration related to vasospasm or have increases in MFV and MCA/ICA MFV ratios.[69,76-79] At a minimum, a normal intravascular volume must be maintained.[25,80]

Implications for Future Nursing Research

A number of recommendations for future research can be made. A large sample size would increase the statistical power of the findings. If possible, the sample should be more homogeneous than our sample in regard to previous angioplasty. Multimodality monitoring such as measurements of jugular bulb oxygen saturation or regional brain tissue oxygenation might also enhance understanding of the effect of the position of the head of the bed on vasospasm.[81] Alterations in the study design could include increasing the degree of elevation to 60º or increasing the time in each position to determine if tolerance is a function of time. Improvements in the existing transcranial Doppler imaging technology would also be beneficial, including a single multichannel transcranial Doppler imaging system for simultaneously and continuously recording MCA MFVs and ICA MFVs, a different style headband and/or a sensor that allows simultaneous recording of multiple vessels, and inclusion of continuous transcranial Doppler monitoring as a method available with bedside critical care monitoring systems.

Conclusions

Our results provide preliminary information that supports earlier mobilization of patients with aneurys-

mal SAH. Mean differences in MCA MFVs and MCA/ICA MFV ratios within the 20 patients as a group when the head of the bed was elevated were not statistically significant. None of the patients experienced MCA MFVs or MCA/ICA MFV ratios consistent with severe vasospasm, and none had deterioration in neurological status. However, the degree of changes in MFVs and MCA/ICA MFV ratios varied among individual patients without obvious explanation and resulted in an increase in vasospasm from mild to moderate for 1 of the 20 subjects. Additional research is warranted. Meanwhile, the responses of individual patients must be closely assessed and monitored.

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