Effects of Body Position on Intracranial Pressure and Cerebral Perfusion in Patients With Large Hemispheric Stroke

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Background and Purpose—The purpose of this study was to prospectively evaluate the effects of body position in patients with large supratentorial stroke.

Methods—We performed 43 monitoring sessions in 18 patients with acute complete or subtotal middle cerebral artery (MCA) territory stroke. Intracranial pressure (ICP) was monitored with a parenchymal probe. Mean arterial blood pressure, ICP, and MCA peak mean flow velocity (V_mMCA) were continuously recorded. Patients with acute ICP crises were excluded. After baseline values at a 0° supine position were attained, the backrest was elevated in 2 steps of 5 minutes each to 15° and 30° and then returned to 0°.

Results—Baseline mean arterial pressure was 90.0±1.6 mm Hg and fell to 82.7±1.7 mm Hg at 15° and 76.1±1.6 mm Hg at 30° backrest elevation (P<0.0001). ICP decreased from 13.0±0.9 to 12.0±0.9 mm Hg at 15° and 11.4±0.9 mm Hg at 30° backrest elevation (P<0.0001). As a result, cerebral perfusion pressure decreased from a baseline value of 77.0±1.8 to 70.0±1.8 mm Hg at 15° and 64.7±1.7 mm Hg at 30° backrest elevation (P<0.0001). V_mMCA was already higher on the affected side during baseline measurements. V_mMCA decreased from 72.8±11.3 cm/s at 0° to 67.2±9.7 cm/s at 15° and 61.2±8.9 cm/s at 30° on the affected and from 49.9±3.7 cm/s at 0° to 47.7±3.6 cm/s at 15° and 46.2±2.2 cm/s at 30° on the contralateral side (P<0.0001).

Conclusions—in patients with large hemispheric stroke without an acute ICP crisis, cerebral perfusion pressure was maximal in the horizontal position although ICP was usually at its highest point. If adequate cerebral perfusion pressure is considered more desirable than the absolute level of ICP, the horizontal position is optimal for these patients. (Stroke. 2002;33:497-501.)

Key Words: blood flow velocity ■ body position ■ cerebral perfusion pressure ■ intracranial pressure ■ middle cerebral artery ■ stroke

Although there are no data from standardized studies, moderate elevation of the head is standard practice in the management of intracranial pressure (ICP); thus, patients with massive hemispheric stroke are traditionally nursed with the head elevated in most institutions. For patients with head trauma, it is generally agreed that moderate (15° to 45°) head elevation significantly reduces ICP, whereas head elevation >45° may be dangerous because of a critical decrease in cerebral perfusion pressure (CPP).1-6 However, in these patients, even moderate head elevation may compromise CPP.3,4 Until now, for patients with ischemic stroke, the question of optimal body position has not been addressed in systematic studies.

We evaluated the effects of moderate backrest elevation on ICP, CPP, and middle cerebral artery (MCA) peak mean flow velocities (V_mMCA) as an estimate of cerebral blood flow (CBF) in patients with large hemispheric stroke.

Patients and Methods
From January to July 2001, 18 consecutive patients with acute complete or subtotal (more than two thirds) MCA territory stroke were studied. All patients were treated in the neurocritical care unit according to institutional protocol for patients with large MCA territory stroke. All patients were intubated, ventilated, and anesthe-tized with fentanyl and midazolam. The patients were routinely nursed in a 30° upright position. Ventilation parameters were adjusted to achieve normocapnia and a PaO_2 >90 mm Hg. ICP was continuously monitored with an intraparenchymatous device (Spiegelberg) inserted into the affected hemisphere in all patients. ICP, pulse oxygenation, heart rate, and mean arterial blood pressure (MAP), which was measured via a catheter in the radial or femoral artery, were continuously monitored. The arterial blood pressure transducer was kept at the level of the foramen of Monro. Crystalline fluids and hydroxyethyl starch solutions were administered to achieve a central venous pressure of between 12 and 16 cm H_2 O. If volume substitution was not sufficient to reach a CPP of ≥70 mm Hg, norepinephrine as a continuous infusion was adminis-
sive surgery or hypothermia (33°C over 72 hours) as previously described. Patients who underwent hypothermia were treated with atracurium for muscular relaxation. Other specific therapeutic measures such as osmotherapy or hyperventilation were not used until the ICP reached 20 mm Hg or clinical signs of markedly increased ICP such as papillary abnormalities had developed.

During a single monitoring session, ventilation parameters, concomitant medication, and volume replacement remained unchanged, and nursing procedures such as turning or endotracheal suction were restricted to a minimum. Monitoring was initiated ≥2 hours after specific therapy for increased ICP. Patients in whom the ventilation parameters, fluid therapy, or medication had to be modified during the observation period were excluded from further analysis. Because induced arterial hypertension could, in theory, critically increase an already raised ICP, we excluded any patients with an acute ICP crisis (ICP >20 mm Hg or papillary abnormalities).

Patients without a transtemporal bone window adequate (at least unilaterally) for transcranial ultrasound were not included in this study. The MCAs were identified at a depth of 50 to 58 mm and continuously insonated with the 2-MHz transducers of a pulsed-wave ultrasound machine (Multi-Dop-X4, DWL), which were fixed on the skull in a custom-designed frame. In some patients, only unilateral monitoring was feasible because of permanent MCA occlusion on the affected side. VmMCA was registered online. MAP, ECG, and ICP data were exported to the ultrasound machine as analog data. The CPP was calculated as the difference between MAP and ICP. Repeated measurements could be performed in the same patient on a daily basis with an interval of ≥18 hours between 2 single measurements.

A single monitoring session consisted of 4 steps, each lasting 5 minutes. The patient’s position was changed by manipulating the backrest of the bed. At the beginning of the protocol, baseline values were obtained with the patient lying flat and the head in a horizontal midline position (0°). The backrest was then elevated to 15° and 30°, and at the end of the protocol, the backrest was lowered to the horizontal (0°) body position again. The degree of backrest elevation was measured by flexion at the hips. Great care was taken to keep the head in a neutral midline position because rotatory and flexion-extension movements of the neck could substantially alter the ICP. For each step, the arterial blood pressure transducer was recalibrated and positioned at the level of the foramen of Monro. A single monitoring session is depicted in Figure 1.

A monitoring session was evaluated further only if the blood pressure values acquired at the beginning and end of the protocol did not differ by >10%. It was otherwise assumed that the changes observed were not exclusively related to the changes in position. All parameters monitored were calculated by averaging the values recorded over the last minute before the next step. Thus, only 1 value for each parameter and level was used for further statistical analysis.

The patient outcome was assessed at discharge with the Glasgow outcome scale.10

This study was conducted according to the local ethics committee standards. Informed consent was obtained from the patients’ relatives. All data were analyzed without identification of the patient.

Statistical analysis was performed with the Wilcoxon signed-rank test to detect differences between each time point and baseline values. Differences were considered significant at values of P<0.05. Data are presented as mean±SEM.

Results

In all, 43 monitoring sessions were performed in 18 patients (7 women, 11 men; mean age, 61.2±2.2 years). Infarct location was the left hemisphere in 9 and the right hemisphere in the remaining 9 patients. The cause of stroke was dissection of the internal carotid artery (n=3), cardioembolic resulting from atrial fibrillation (n=7) or other cardiac disease (n=3), and embolic of unclear origin (n=5). Seven patients with right MCA territory stroke underwent decompressive hemicraniectomy, and 11 patients with left MCA territory stroke were treated with therapeutic hypothermia (33°C over 72 hours). Fourteen measurements were performed in patients after decompressive surgery. In 7 patients (22 episodes), only unilateral monitoring of the VmMCA could be performed because of permanent occlusion of the vessel on the affected side. All measurements were performed in the first 6 days after stroke. In 29 episodes, the patients were treated with vasopressor drugs (norepinephrine or dobutamine as a continuous infusion) for arterial hypotension. At discharge, 4 patients had died of uncontrollable intracranial hypertension. Three of those patients were treated medically. The remaining patients remained severely disabled (Glasgow outcome scale, 3). Concomitant medication and ventilation parameters could be kept stable during all monitoring sessions.

MAP, ICP, CPP, and VmMCA values for each step are depicted in Figure 2. MAP fell from a mean baseline of 90.2±1.8 to 82.7±1.7 mm Hg at 15° backrest elevation and further to 76.1±1.6 mm Hg at 30° backrest elevation (P<0.0001) and returned to baseline values of 90.3±1.8 mm Hg after the backrest was again lowered to the flat position. ICP decreased from 13.0±0.9 to 12.0±0.9 mm Hg at 15° backrest elevation and further to 11.4±0.9 mm Hg (P<0.0001) at 30° backrest elevation and returned to baseline levels again (13.0±0.9 mm Hg) in the horizontal position. However, the change in ICP in absolute numbers was rather small (1.0±0.2 mm Hg at 15° and 1.6±0.3 mm Hg at 30° backrest elevation). In 8 episodes, there was no ICP decrease at all from 0° to 30° backrest elevation, and in 34 episodes, ICP decreased between 1 and 4 mm Hg. In 1 episode, there was a marked decrease in ICP of 9 mm Hg at 30° backrest elevation. An increase in ICP with backrest elevation was never noted.
V_mMCA values were recorded on the side contralateral to the lesion in all and on the affected side in 21 episodes. VmMCA was more pronounced on the affected side, demonstrating impaired autoregulatory mechanisms (P < 0.001). CPP returned to baseline values (77.2 ± 1.8 mm Hg) at 0° at the end of the protocol.

Maximal CPP always occurred with the body in a horizontal position.

As a result of the changes in MAP and ICP, CPP was highest in the flat position (77.0 ± 1.8 mm Hg) and decreased to 70.0 ± 1.8 mm Hg at 15° and further to 64.7 ± 1.7 mm Hg at 30° (P < 0.0001). CPP returned to baseline values (77.2 ± 1.8 mm Hg) at 0° during 21 monitoring sessions as a result of permanent surgery with the medically treated patient group.

Comparison of the patients who underwent decompressive surgery with the medically treated patient group revealed a higher V_mMCA on the affected side (91.8 ± 15.7 versus 40.1 ± 6.8 cm/s at baseline) and on the contralateral side (67.6 ± 7.2 versus 41.1 ± 3.2 cm/s at baseline). This difference was significant (P < 0.001) throughout all steps on both sides, whereas the ICP and CPP were not different between the 2 groups.

Discussion

In this study, we systematically examined for the first time the effects of body position on physiological variables in patients with ischemic stroke. The use of head elevation in the literal sense is not useful because it may lead to compression of the jugular veins with subsequent increases in ICP. Therefore, we prefer the term “backrest elevation” to “head elevation,” which could be misleading, although the 2 terms are used synonymously in most studies.

The recommendations for optimal body position in stroke patients are derived from pathophysiological considerations and the results from neurosurgical or mixed patient collectives. Because of the differences in pathophysiology, the results from head trauma patients can hardly be used to guide the management of stroke patients. Most studies in neurosurgical patient collectives agree that moderate head elevation decreases ICP in patients with head trauma.1–3,5,6,9,11 However, in head trauma patients, head elevation also decreases CPP because of a marked decrease in MAP.3,4

Some authors have examined CBF in relation to body position. Feldman et al11 used the Kety-Schmidt technique to measure CBF during 0° and 30° head elevation in head-injured patients. They found no statistically significant changes in either CPP or CBF. In contrast, in a recent publication, Moraine et al12 showed a marked, gradual decrease in CBF between 0° and 45° while the CPP remained stable between 0° and 30°. The authors concluded from measurements of the jugular bulb pressure that the arteriovenous pressure gradient, not the CPP, was the major determinant of CBF in these patients. In a small study of 10 patients suffering from various forms of intracranial pathology, Cunitz and Schregel13 described a decrease in MCA flow velocities after head elevation from 0° to 30° and 60°, which is in agreement with the findings of Moraine et al12 and the results of our study.

It has to be underscored, however, that V_mMCA is not identical to CBF. Transcranial Doppler provides only indirect information about CBF, measuring the flow velocity and not the volume flow; therefore, changes in blood flow velocity correspond to CBF only if the vessel diameter is constant, which cannot be assumed a priori. Moreover, even very small changes in the position of the transcranial Doppler probe can alter the blood flow velocity readings. However, transcranial Doppler monitoring is the only available method of noninvasively monitoring CBF changes continuously, and it is generally accepted that the dynamic changes in CBF are closely reflected in the transcranial Doppler readings.14–20 Unfortunately, we were not able to estimate CBF on the affected side if the vessel was permanently occluded. Therefore, all information on the perfusion of the affected side is derived from those patients in whom the MCA had recanalized. Still, it can be assumed that an optimized perfusion is more important in patients with a permanently occluded vessel...
than in patients with an already hyperemic infarction after recanalization.

We measured the MAP via a radial artery catheter and calibrated the pressure transducer at the level of the foramen of Monro. This technique may provide only an inaccurate estimate of the actual cerebral blood pressure. In particular, in older patients, there may be significant differences between MAP in the carotid arteries and in the peripheral arteries, where the MAP is usually measured. However, this effect presumably does not influence the relative changes in MAP and CPP during head elevation, although it may lead to overestimation of the absolute value.

There are various pathophysiological mechanisms by which changes in body position may affect systemic and intracerebral pressures and cerebral perfusion. Among them are alterations in blood pressure, chest wall compliance, ventilation mode, venous outflow resistance, and displacement of cerebrospinal fluid. All would be expected to promote a reduction in ICP with the head elevated. With progressive elevation of the head above the heart, the hydrostatic pressure at the cranial levels declines. The most important mechanism appears to be related to the increase in venous outflow through the valveless jugular veins and vertebral venous plexuses. Cerebral venous and jugular venous pressures decline with head elevation, leading to a reduction in the cerebral venous blood volume and a subsequent decrease in ICP. Kenning et al suggested that rapid hydrostatic displacement of cerebrospinal fluid into the spinal subarachnoid space from the intracranial compartment might lower cerebrospinal fluid pressure. It seems possible that these compensatory mechanisms are already exhausted when the ICP is elevated over a longer period of time and therefore that the impact of head elevation on ICP could decrease.

In this study, we examined only patients without an acute ICP crisis to prevent the possibility of acute decompression of an already critically raised ICP when the backrest was decreased to 0°. According to the present findings, the study of the effects of a horizontal position even in those patients with elevated ICP seems to be important. The study of the effects of a horizontal position for patients in whom ICP rises substantially in a flat position, our data give little support for the idea that the horizontal position should be established individually rather than routinely nursing these patients with the backrest elevated. For patients with large hemispheric stroke without acute ICP crises, our data give little support for the routine use of a 30° backrest elevation, because the increase in ICP in the horizontal position is probably not clinically significant but the decrease in CPP and MAP may be hazardous for patients with a massive ischemic infarct. If a sufficient CPP is the main criterion for guiding the management of these patients, the horizontal position is probably preferable as standard procedure. In those few patients in whom ICP rises substantially in a flat position, moderate backrest elevation may be the better choice.

References


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Stroke. 2002;33:497-501
doi: 10.1161/hs0202.102376

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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