Effects of the Sitting Position on Blood Flow in the Internal Carotid Artery of Man During General Anesthesia*

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Placing a patient in a sitting position during an operation on the cervical spine or the posterior fossa offers at least two technical advantages to the neurosurgeon: it is easier to maintain a given position, and there is less bleeding. However, two well-recognized potential hazards are associated with this surgical position, namely, air embolism and inadequate cerebral blood flow.

When a patient is in the sitting position, venous pressure at the cervical level can fall below atmospheric pressure during inspiration; thus, an opening in a vein could allow a fatal amount of air to enter the heart.3–5 Although clinical experience with patients who have undergone surgery in the sitting position has shown that cerebral perfusion is usually sufficient to preserve normal cerebral function, the degree to which cerebral flow is depressed during these procedures has not been previously measured. Since anesthesia accompanied by hypocarbia is known to cause a marked reduction in cerebral blood flow,14 any further decrease resulting from the sitting position could lower cerebral blood flow to a dangerous level.

The following experiments were designed to determine the effects of the sitting position on blood flow and pressure in an internal carotid artery in anesthetized patients. In addition, the effects of general anesthesia combined with hyperventilation on internal carotid arterial flow and pressure were evaluated.

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Method

Continuous blood-flow measurements were made in nine male patients who were undergoing surgical exposure of the carotid vessels so that an anti-tumor drug‡ could be infused directly into the internal carotid artery. In each patient, craniotomy and subtotal resection of a supratentorial brain tumor had been performed 10 to 20 days before the study. The histologic diagnosis was glioblastoma multiforme in seven patients and astrocytoma in two. Pre-anesthetic medication consisted of 0.6 mg atropine sulfate and 100 mg sodium secobarbital. The patients were initially anesthetized with 10.0 cm² of a 2.0% solution of sodium thiopental administered intravenously. They were then given 40.0 mg succinylcholine chloride and 3.0 mg decamethonium bromide intravenously and intubated. Anesthesia was continued using a 50–50 mixture of nitrous oxide and oxygen, with sufficient Halothane (0.1–0.5%) to prevent coughing or gross movements. The patients were manually hyperventilated, and the level of anesthesia was maintained as constant as possible throughout the study. These anesthetic techniques are identical with those ordinarily employed at Duke University Medical Center for neurosurgical procedures done in the sitting position.

A neck dissection was performed, and the common carotid artery and proximal portions of the internal and external carotid arteries were exposed. To monitor arterial pressure, a 16-gauge Rochester catheter was inserted into the common carotid artery and connected to a pressure transducer.§ The probe of a Kolin-Kado type electromagnetic

‡ S-112 (a-chlorothythioacetamide), given in a dosage of 0.08 mg/kg body weight.

§ Model P23db, Statham Instruments, Los Angeles, California.
flowmeter (EMF)** was placed about the common carotid artery approximately 4 cm proximal to its bifurcation.\(^5\) A Crutchfield clamp was used to occlude the external carotid artery throughout the experimental study so that blood in the common carotid artery was distributed only to the internal carotid system. A second Crutchfield clamp was placed around the internal carotid artery and left open. This clamp was used briefly to occlude the internal carotid artery in order to establish the zero flow reference for the EMF. The EMF probe leads and the arterial catheter were brought out through the incision, which was temporarily closed with silk sutures. Continuous measurements of blood flow and arterial pressure were obtained as described in previous reports.\(^2,12\)

The EMF was calibrated following the surgical procedure by passing known quantities of normal saline solution through the probes in a given period of time. The calibration factor for the probes (the flow per unit EMF signal) remained within a SD \(\pm 6.8\%\) during the period of study. Right atrial level was used as zero reference for the arterial pressure. Mean values for both pressure and flow were obtained by electrical integration.

Brachial arterial samples were drawn for measurement of pCO\(_2\), pO\(_2\), and pH* during control period and at the end of a 10-minute period of sitting. In addition, cardiac output was obtained during each period using the indicator dilution technique: Indocyanine green dye was injected into the median basilic vein and sampled from the brachial artery. All data were recorded on a direct-writing oscillograph.*

With the patient in a horizontal position, continuous measurements of blood flow and arterial pressure were obtained during a 5-minute control period. At the end of this time, the operating table was adjusted so that the patient was in a sitting position with the head supported by a Craig chair attachment and the cervical spine in moderate flexion and perpendicular to the floor. The legs were extended at the knees and the thighs were flexed at the hips. Each leg had been previously wrapped securely to mid-thigh level with Ace bandages. Positioning the patient required approximately 2 minutes; the position was then maintained for 10 minutes while recordings of blood flow and arterial pressure were made.

With the patients still in the sitting position, five of them were stimulated by pinching the skin of the neck for 2 to 3 minutes to simulate the effects of surgical dissection. Both blood pressure and flow in the internal carotid artery were recorded during this period. The patients were then returned to a horizontal position and the procedure concluded.

To obtain control data of internal carotid flow and arterial pressure, these measurements were recorded approximately 3 hours after anesthesia was discontinued in four of these nine patients. In addition, similar measurements were obtained in six other patients who were not placed in the sitting position. Arterial pCO\(_2\), pO\(_2\), and pH were determined in both the awake state and during general anesthesia.

Although continuous measurements of both phasic and mean arterial pressures and flow were obtained, only mean values will be presented. The data given for the control period and for the awake study represent the average for measurements made each minute during a 5-minute period. During the period of sitting, data recorded at 2, 4, 5, 8, and 9 minutes were analyzed and an index of cerebral vascular resistance (CVR) was obtained from

\[
\text{CVR} = \frac{P}{Q} \cdot 1
\]

where P (mm Hg) is the mean arterial pressure and Q (cm\(^3\)/min) is the simultaneous mean flow of the internal carotid artery. Standard statistical techniques were used to evaluate the data.\(^11\)

**Results**

The data obtained on 10 patients illustrating the effects of general anesthesia combined with hyperventilation are listed in Table 1. The mean value for internal carotid flow in these subjects while awake was 201 cm\(^3\)/min, SD \(\pm 38\). During anesthesia, flow decreased significantly (\(p<0.01\)) by an average of 34\% (range: 4 to 50\%) to a mean

**Model K-2000 Statham Instruments, Los Angeles, California.**

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TABLE 2

<table>
<thead>
<tr>
<th>Effect of Sitting Position</th>
<th>Supine</th>
<th>Sitting (9 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Int. Car. Artery Flow (cm³/min)</td>
<td>141 ± 59</td>
<td>121 ± 56</td>
</tr>
<tr>
<td>Mean Arterial Pressure (mm Hg)</td>
<td>90 ± 17</td>
<td>91 ± 17</td>
</tr>
<tr>
<td>pO₂ (mm Hg)</td>
<td>194 ± 64</td>
<td>205 ± 41</td>
</tr>
<tr>
<td>pH</td>
<td>7.59 ± .02</td>
<td>7.59 ± .06</td>
</tr>
<tr>
<td>pCO₂ (mm Hg)</td>
<td>92 ± 4</td>
<td>92 ± 4</td>
</tr>
<tr>
<td>Cardiac Output (cm³/min)</td>
<td>4145 ± 1474</td>
<td>3401 ± 1258</td>
</tr>
</tbody>
</table>

TABLE 1

<table>
<thead>
<tr>
<th>Effects of general anesthesia combined with hyperventilation</th>
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<tbody>
<tr>
<td>Awake</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>Int. Car. Artery Flow (cm³/min)</td>
</tr>
<tr>
<td>Mean Arterial Pressure (mm Hg)</td>
</tr>
<tr>
<td>pO₂ (mm Hg)</td>
</tr>
<tr>
<td>pH</td>
</tr>
<tr>
<td>pCO₂ (mm Hg)</td>
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</table>

value of 134 cm³/min, SD ± 52. At the same time, average values for mean arterial pressure were 102 mm Hg, SD ± 14, and 87 mm Hg, SD ± 17, during the awake and anesthetized states respectively. This difference is also statistically significant (p<0.01). CVR calculated from these data revealed a mean value of 0.51 mm Hg/cm³/min for the control period. During anesthesia, the CVR in all patients increased significantly (p<0.01) to an average value of 0.65 mm Hg/cm³/min. The arterial pCO₂ was 40 mm Hg, SD ± 5, in the awake state and 21 mm Hg, SD ± 3, during general anesthesia, representing a significant decrease (p<0.01). There were significant increases in arterial pO₂ (p<0.01) and pH (p<0.01) in association with general anesthesia.

The data recorded from nine anesthetized patients in the sitting position are shown in Table 2 and illustrated in Fig. 1. The values used to construct Fig. 1 were normalized prior to averaging by first dividing each parameter by that patient's control values, that is, flow by control flow. Normalized values were then averaged at the time periods shown in Fig. 1. The crossed bars in Fig. 1 represent the standard error. The mean value for internal carotid flow in the supine position was 141 cm³/min, SD ± 59 (Table 2, column 1). After 2 minutes in the sitting position, there was an average decrease of 18% in the internal carotid artery flow. After 9 minutes, flow increased somewhat but remained 14% below control values. At this time the mean internal carotid flow was 121 cm³/min, SD ± 56 (Table 2, column 2), representing a statistically significant reduction (p<0.05) from the control.

The magnitude of the changes in blood flow during the sitting position varied considerably among the nine subjects. In six patients the decrease ranged from 9 to 27% below control values, in two patients there was no change, and in one subject there was a 10% increase in flow. Although the mean arterial pressure for the group did not change during sitting (see Table 2), there were changes in the individual subjects which roughly paralleled the changes in internal carotid blood flow. The heart rate increased by approximately 5% throughout the period of observation in the sitting position. In four subjects, data were recorded when the patients were awake in the horizontal position and anesthetized in the sitting position; the average decrease in flow was 36% (range: 4 to 48%) of control flow.

The mean value for cardiac output in the horizontal position was 4145 cm³/min, SD ± 1474; at the end of the 9-minute sitting period, the mean value was 3401 cm³/min, SD ± 1258. This difference is statistically significant (p<0.05). There were no significant changes in either the arterial pCO₂, pO₂, or pH associated with the sitting position.

At the conclusion of the 10-minute period of observation in the sitting position, five patients were stimulated by pinching. In each
Fig. 1. Effects of the sitting position on (from top down) mean arterial pressure, mean internal carotid flow, and heart rate. The vertical bars represent the standard error. Data were normalized prior to averaging by dividing each parameter by that subject’s control value; for example, arterial pressure was divided by the subject’s control (patient horizontal) arterial pressure. The values shown were obtained at 2, 4, 5, 8, and 9 min after placing the anesthetized patient in the sitting position. Note that flow falls to 18% below control value at 4 min, and at the end of 9 min is still 14% below control value. Mean arterial pressure (zero pressure reference at the right atrium) rises slightly at ~2 and 4 min but returns to control value by the end of 5 min. Heart rate remains slightly above control level throughout the period of observation.

Discussion

The 34% average decrease in internal carotid artery blood flow that occurred in our patients during general anesthesia associated with hyperventilation is consistent with data previously recorded by Wollman and associates. These investigators used similar anesthetic techniques and noted that cerebral blood flow decreased from a control value of 44.4 cm³/100 gm brain/min to 27.2 cm³/100 gm brain/min in normal male volunteers. The two factors primarily responsible for the reduction in internal carotid flow noted in our subjects during anesthesia were the fall in arterial pressure and the marked decrease in arterial pCO₂. If the cerebral vascular resistance was the same in both the awake and anesthetized states, an average reduction in flow of 8.5% would occur secondary to the decrease in perfusion pressure. Thus, the major factor responsible for the fall in blood flow was that cerebral vascular resistance increased due to the marked hypocarbia present during general anesthesia. In fact, Wollman and co-workers have shown that cerebral blood flow is actually higher than control levels in normocarbic patients anesthetized with Halothane. For the purpose of this discussion, the significant finding in both case there was a transient increase in mean arterial pressure leading to an increase in internal carotid flow (Fig. 2) that persisted for 3 to 5 minutes after stimulation ceased.
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Effects of stimulation during the sitting position on (from top down) blood pressure, internal carotid pulsatile flow, and internal carotid mean flow. Control measurements recorded with the patient horizontal during anesthesia are shown in Panel A. Panel B shows the measurements made after the anesthetized patient had been in the sitting position for 10 minutes. There is a significant reduction in flow associated with the sitting position. At the point indicated by the arrow (~), the patient was stimulated by repetitive pinching of the cervical region. Note the immediate increase in flow and blood pressure.

The present study and the one by Wollman, et al., is that a marked reduction in cerebral blood flow is associated with anesthesia in which hyperventilation techniques (hypocarbia) are used.

Internal carotid artery blood flow was further reduced when these patients were placed in the sitting position. After 2 minutes, flow had fallen in all subjects by an average of 18%. Thereafter, the individual responses were somewhat variable, but mean flow was 14% below control at the end of 9 minutes. These findings are consistent with the effects of postural changes upon cerebral blood flow recorded by several investigators in awake human subjects. Scheinberg and Stead used a nitrous oxide technique to measure flow and noted a 21% reduction in cerebral blood flow in 20 normal subjects who were tilted head up 65° from the horizontal.

Although there is disagreement concerning the magnitude of the reduction in cerebral perfusion pressure when the upright position is assumed, the fall in blood flow noted in our subjects can be explained by the reduction in effective arterial pressure. If it is assumed that mean arterial pressure at the level of the mid-cerebrum is the true cerebral perfusion pressure in the upright position, the perfusion pressure decreased in our subjects by an average of 24 mm Hg. Thus, if the cerebral vascular resistance remained constant while the subjects were placed in a sitting position, it would be predicted that the initial response to sitting would be a 20%
fall in blood flow. This value closely approximates the 18% decrease in internal carotid artery blood flow observed at the end of 2 minutes. As the patient remains in the sitting position, cerebral vascular resistance gradually decreases from a value of 0.65 mm Hg/cm³/min at 2 minutes to 0.55 mm Hg/cm³/min at 9 minutes. This reduction is quantitatively similar to the 16% decrease in cerebral vascular resistance following 65° head-up tilt in normal subjects measured by Scheinberg and Stead.¹⁰

In the actual operating room situation the magnitude of the fall in cerebral blood flow may be less than in our patients, due to stimulation of the sympathetic nervous system afforded by the dissection. In subjects who were stimulated by pinching, both the arterial pressure and internal carotid blood flow increased. It should be noted that the arterial pCO₂ was constant in our subjects during this entire period and therefore can be eliminated as a significant factor in the regulation of cerebral flow.

The volume of blood necessary to perfuse the brain adequately during general anesthesia in man obviously has not been precisely defined. Multiple factors such as age, existing vascular disease, or anesthetic drugs will markedly affect this value. In addition, any figure for total cerebral blood flow cannot take into account variations in regional perfusion. In studying awake subjects, Finnerty, et al.,¹ showed that signs and symptoms of cerebral ischemia invariably developed when cerebral blood flow was reduced 42% from a control value of 55 cc/100 gm brain/min to a value of 31.5 cc. The level to which cerebral blood flow can be reduced during general anesthesia without incurring brain damage is probably greater than during the awake state, due to lower metabolic requirements. Wollman, et al., found a 17% decrease in cerebral oxygen consumption from control awake values in human subjects who were anesthetized with Halothane and made hypocarbie.¹³ However, any attempt to extrapolate these figures to give an exact value for a critical level of cerebral blood flow during anesthesia would not be justified.

In the present study, flow was reduced by approximately 36% (maximum of 50%) in patients placed in the sitting position and undergoing anesthesia with hyperventilation. The cerebral blood flow is reduced markedly with the anesthetic techniques usually employed in neurosurgical procedures done in the sitting position. Therefore, these patients will be quite susceptible to any further reduction in cerebral perfusion and should be closely observed during surgery, with particular attention to maintenance of normal blood-pressure levels.

Summary

1. The effect of the sitting position on flow in the internal carotid artery and on arterial pressure has been determined in 9 patients during general anesthesia. In addition, measurements of internal carotid arterial flow and pressure were made in 10 patients while awake and during anesthesia using hyperventilation techniques.

2. Mean values for internal carotid flow and arterial pressure in the 10 awake patients were 201 cm³/min, SD ± 38, and 102 mm Hg, SD ± 14, respectively. During anesthesia mean values were 134 cm³/min, SD ± 52, for flow and 87 mm Hg, SD ± 17, for arterial pressure. Mean values for arterial pCO₂ were 40 mm Hg, SD ± 5, and 21 mm Hg, SD ± 3, while awake and during anesthesia respectively. It is felt that the decrease in arterial pCO₂ is the principal cause for the significant reduction (34%) in flow in the internal carotid artery.

3. The mean value for internal carotid flow in 9 anesthetized patients in the horizontal position was 141 cm³/min, SD ± 59. Nine minutes after placing these patients in a sitting position, the mean value for internal carotid flow was 121 cm³/min, SD ± 56. This difference represents a 14% decrease in flow. There was a significant decrease in cardiac output (17%) and an increase in heart rate associated with the sitting position, but no change in pCO₂, pO₂, or mean arterial pressure measured at the level of the right atrium.

4. There was a transient rise in mean arterial pressure and internal carotid flow associated with painful stimuli (pinching) applied to the patient while in the sitting position.

5. The results of the study indicate that, prior to placing an anesthetized patient in a sitting position, there has already been a significant reduction in cerebral blood flow if
hyperventilation anesthetic techniques are being employed. The additional reduction in cerebral blood flow associated with the sitting position is potentially hazardous in a situation where the intracranial vasculature is partially compromised by vascular disease.

Acknowledgments
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References