Continuous measurement of jugular venous oxygen saturation in response to transient elevations of blood pressure in head-injured patients

JOHN B. FORTUNE, M.D., PAUL J. FEUSTEL, PH.D., CARL G. M. WEIGLE, M.D., AND A. JOHN POPP, M.D.

Section of Trauma Surgery and Division of Neurosurgery, Department of Surgery, and Section of Pediatric Intensive Care, Department of Pediatrics, Albany Medical Center, Albany, New York

Following traumatic brain injury, continuous jugular venous oxygen saturation (SjvO2) measurements have been made and used to assess cerebral oxygenation. Transients of SjvO2 may reflect cerebral blood flow (CBF) changes if measurements are made over a short period of time during which cerebral metabolic rate for oxygen is assumed unchanged. In response to alterations in perfusion pressure, transients of SjvO2 may indicate the extent to which autoregulation has been preserved after injury. The effect of arterial pressure changes on SjvO2 was measured in 14 severely head-injured patients (Glasgow Coma Scale score < 8) within 36 hours of injury. Mean arterial blood pressure (MABP), arterial oxygen saturation, and intracranial pressure (ICP) data were also continuously recorded by a computer at the patients' bedside. The reliability of the SjvO2 oximetry measurements varied among patients, and an average 38% of SjvO2 measurements were off by more than 6% saturation, necessitating recalibration. During periods of satisfactory catheter performance, 120 instances were found in which MABP was elevated more than 8 torr (mean ± standard deviation: 32 ± 13 torr) due to endotracheal suctioning. In 94 of these measurements, there was an associated increase in the ICP of 5 torr or more, averaging 16.6 ± 10.2 torr. The SjvO2 was 0.62 ± 0.10 before the increase in MABP and rose to a peak of 0.77 ± 0.10 during the maximum MABP elevation, suggesting increased CBF during the transient hypertension. In 34 of 37 instances of persistent blood pressure elevations lasting for more than 10 minutes (mean 16.0 ± 8.0 minutes), the SjvO2 elevation persisted (average duration 15.0 ± 12.4 minutes), suggesting impaired or lost autoregulatory vasoconstriction. The presence or absence of hyperemia was unrelated to the extent of the autoregulation response. Results indicate that SjvO2 rises with increasing perfusion pressure during and after endotracheal suctioning, suggesting a feeble or absent autoregulatory response following traumatic brain injury.

KEY WORDS - traumatic brain injury - cerebral blood flow - autoregulation - cerebral perfusion pressure - jugular venous oxygen saturation - oximetry

After traumatic brain injury in the human, histopathological and metabolic evidence suggests the existence of areas of ischemia; yet, 12 hours to several days after injury, normal or elevated global cerebral blood flow (CBF) is a more common finding. If the primary injury, however, leads to altered CBF regulation, then provoked or unprovoked transient episodes of low blood flow or significant regional maldistribution of flow may exist without discovery by intermittent global measurements. It has been demonstrated in humans and animals that flow regulation abnormalities in response to alterations in perfusion pressure, blood gas levels, and metabolic demand occur after traumatic brain injury. The traumatized brain is known to be especially susceptible to transient episodes of hypoxia or ischemia after significant injury. If flow regulation is disturbed, then ordinarily unimportant transient changes in perfusion pressure, PaCO2, or PaO2 may result in exaggerated or diminished flow changes. Since monitoring techniques examine only intracranial pressure (ICP) with infrequent intermittent global CBF measurements, transient tissue ischemia secondary to low flow in the presence of altered autoregulation may contribute substantially to morbidity and mortality.
Recent studies have suggested the use of continuous jugular venous oxygen saturation (SjvO$_2$) measurement via oximetric techniques as a means of monitoring therapeutic interventions following traumatic brain injury.$^{2-3,12,20}$ Assuming that the cerebral metabolic rate of oxygen (CMRO$_2$) and hemoglobin concentration remain relatively constant and neglecting the small contribution of dissolved oxygen, the difference between arterial oxygen saturation (SaO$_2$) and SjvO$_2$ will reflect CBF by the Fick principle. Continuous fiberoptic oximetry of SjvO$_2$ combined with simultaneous continuous recordings of ICP, arterial pressure, and SaO$_2$ may be helpful in assessing the relationship between perfusion pressure and blood flow as the clinical course of severely head-injured patients unfolds. We have used continuous jugular venous oximetry to assess the effects of transient changes in arterial pressure on ICP and SjvO$_2$. These data were used to quantitate the extent of autoregulation after head injury and to determine the effect on CBF of endotracheal suctioning, which typically elevates arterial blood pressure and ICP.

**Clinical Material and Methods**

**Patient Population**

Patients who suffered closed head injury with subsequent Glasgow Coma Scale scores of less than 8 were candidates for this study. Initial resuscitation was directed at maintaining the airway and arterial oxygenation and treating any shock states. Once the patient's status was stabilized, early cranial computerized tomography (CT) was performed. Appropriate neurosurgical intervention was then undertaken to evacuate any intracranial collections of blood. Any other operative intervention necessary for life or limb salvage was performed under the same general anesthetist. All patients were then transferred to the surgical intensive care unit for follow-up care. After full disclosure, permission to proceed with the investigation as approved by the Institutional Review Board was then obtained from the next of kin.

**Management Protocol**

The protocol for the management of these patients involved prevention of secondary injuries. Epidural or intraventricular fiberoptic monitors were used for continuous measurement of ICP.$^*$ Swan-Ganz and arterial catheters were placed in all patients to monitor central and peripheral hemodynamics. All patients were intubated and placed on volume-controlled ventilators for hyperventilation to a PaCO$_2$ of 25 to 30 torr. A fraction of inspired oxygen was chosen that would maintain PaO$_2$ at 100 torr or greater. All patients were sedated with continuous infusion of morphine sulfate; midazolam and vecuronium bromide were used in most cases to induce paralysis. The ICP was maintained below 20 torr, if possible, by head of bed elevation, sedation, and hyperventilation. Intermittent boluses of mannitol were given if the other maneuvers were ineffective at controlling ICP.

Between 6 and 36 hours after injury, SjvO$_2$ monitoring was initiated. A No. 4 French pediatric fiberoptic umbilical artery catheter with its computerized monitoring system was brought to the patient's bedside. After appropriate antiseptic preparation of the right side of the neck and infiltration of local anesthesia, the Seldinger technique was used to place a No. 5 French catheter introducer retrogradely into the jugular vein via the anterior approach. The calibrated oximetry catheter was inserted into the vein through the introducer until it could advance no further, then was withdrawn approximately 1 cm. The usual distance from the skin to the tip of the catheter was between 10 and 15 cm. The catheter was sutured into place and covered with a transparent dressing. An anteroposterior roentgenogram of the neck confirmed that the catheter tip was at the level of the C-2 vertebral body. Catheter calibration was checked in vivo every 4 to 8 hours; this involved slowly aspirating a sample of jugular venous blood and comparing the value obtained from the oximetry catheter and the O$_2$ saturation measured in the blood sample.$^+$ If the values differed by more than 5% saturation, the oximetry catheter was recalibrated to the actual O$_2$ saturation. Arterial O$_2$ saturation was determined continuously by pulse oximetry from a peripheral digit.$^8$

After appropriate filtering, analog signals of SjvO$_2$, ICP, mean arterial blood pressure (MABP), and SaO$_2$ were digitized at 1 Hz, displayed, and stored. Data acquisition and storage utilized an IBM XT microcomputer contained on a bedside cabinet and programmed with the ASYST language.

Both internal carotid arteries (ICA's) were subjected to duplex scanning, which quantifies flow in blood vessels,$^\dagger$ on a daily basis and the results were recorded with the SjvO$_2$, SaO$_2$, ICP, and MABP. This technique and its validity for these measurements have been described previously.$^{17,20}$ Briefly, duplex scanning obtains a vessel diameter measurement of the ICA from a B-mode ultrasound study and a time-averaged velocity of blood flow from the simultaneously obtained Doppler ultrasound signal. Flow in each ICA is quantified by multiplying the vessel area by the time-averaged velocity to determine flow in milliliters per minute. Hyperemic blood flow after head injury was defined as bilateral ICA blood flow (ICBF) greater than two standard deviations above the PaCO$_2$ corrected normal value. This value was determined in a previous study of 20 normal individuals in whom bilateral ICBF was measured under conditions of normoxia or hypoxia and altered CO$_2$. For this part of the study, the normal ICBF cor-

---

*$^*$ Fiberoptic monitors manufactured by Camino Laboratories, San Diego, California.


$^\dagger$ Co-oximeter, Model IL 482, manufactured by Instrumentation Laboratories, Watertown, Massachusetts.

$^8$ Pulse oximeter manufactured by Nelcor, Inc., Hayward, California.

$^\dagger$ Duplex scanner, Model DRF400, manufactured by Diasonics, Inc., Milpitas, California.
Jugular venous oximetry following head injury

TABLE 1
Characteristics of 14 head-injured patients entered into the study

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>GCS Score</th>
<th>Time From Injury to Study (hrs)</th>
<th>ICBF (ml/min)</th>
<th>Clinical Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23, M</td>
<td>4</td>
<td>yes</td>
<td>48</td>
<td>532 Lt pupil fixed; posterior occipital craniectomy, evacuation of epidural hemorrhage</td>
</tr>
<tr>
<td>2</td>
<td>20, M</td>
<td>6</td>
<td>no</td>
<td>35</td>
<td>423 Dilated Lt pupil; CT: edema without evidence of intracranial bleed; Lt frontal depression</td>
</tr>
<tr>
<td>3</td>
<td>20, M</td>
<td>4</td>
<td>no</td>
<td>48</td>
<td>920 Seizures; CT: edema with scattered SAH; Lt subdural hemorrhage, 1-cm midline shift</td>
</tr>
<tr>
<td>4</td>
<td>22, M</td>
<td>5</td>
<td>yes</td>
<td>24</td>
<td>1149 CT: diffuse swelling, blood in ventricles, fractured temporal orbit, Lt ventricular hemorrhage</td>
</tr>
<tr>
<td>5</td>
<td>16, F</td>
<td>4</td>
<td>yes</td>
<td>48</td>
<td>— Frontal contusion; multiple extremity fractures</td>
</tr>
<tr>
<td>6</td>
<td>36, F</td>
<td>6</td>
<td>yes</td>
<td>48</td>
<td>1004 Lt temporal/frontal contusion, Lt pupil dilated</td>
</tr>
<tr>
<td>7</td>
<td>17, M</td>
<td>5</td>
<td>yes</td>
<td>24</td>
<td>702 Multiple fractures; closed head injury; CT: diffuse edema</td>
</tr>
<tr>
<td>8</td>
<td>18, M</td>
<td>5</td>
<td>yes</td>
<td>18</td>
<td>883 Dilated rt pup; CT: blood in ventricles, diffuse swelling, rt skull fracture</td>
</tr>
<tr>
<td>9</td>
<td>25, F</td>
<td>7</td>
<td>yes</td>
<td>63</td>
<td>1017 CT: multiple punctate contusions, rt frontal swelling</td>
</tr>
<tr>
<td>10</td>
<td>18, F</td>
<td>7</td>
<td>yes</td>
<td>36</td>
<td>585 CT: negative</td>
</tr>
<tr>
<td>11</td>
<td>37, M</td>
<td>6</td>
<td>yes</td>
<td>24</td>
<td>958 Pneumothorax; CT: bifrontal &amp; rt temporal contusions</td>
</tr>
<tr>
<td>12</td>
<td>22, M</td>
<td>4</td>
<td>yes</td>
<td>16</td>
<td>665 CT: diffuse cerebral edema, SAH; blood in interhemispheric fissure, rt temporal contusion</td>
</tr>
<tr>
<td>13</td>
<td>20, M</td>
<td>4</td>
<td>yes</td>
<td>27</td>
<td>989 CT: hemorrhagic contusion, edema in posterior temporal/occipital lobe, compression of Lt lateral ventricle</td>
</tr>
<tr>
<td>14</td>
<td>18, F</td>
<td>4</td>
<td>yes</td>
<td>41</td>
<td>838 CT: Lt temporal contusion, slitlike ventricles, SAH</td>
</tr>
</tbody>
</table>

* MVA = motor vehicle accident; GCS = Glasgow Coma Scale; ICBF = bilateral internal carotid artery blood flow, obtained by duplex scanning; CT = computerized tomography; SAH = subarachnoid hemorrhage.

Rected to the patients' PaCO2 was: ICBF (ml/min) = 742 + 18.4 (PaCO2 − 40). The standard deviation for ICBF was 54 ml/min.

Statistical Analysis

Calibration data were analyzed by linear regression, and comparison between groups at different time points was made by analysis of variance. Significance was defined as a p value of less than 0.05.

Results

Fourteen patients, with a mean age of 22 years, were entered into the study within 24 hours of injury. Table 1 outlines the patient population in terms of age, severity of injury, time and value of initial ICBF measurement, CT scan report, and outcome. The survival rate for this group was 86%.

The reliability of SjvO2 determination by oximetry varied among patients. In each case in which calibration was checked, recalibration of the catheter output signal was required 7% to 78% of the time, with a mean of 38%. Figure 1 shows the results of all calibration analyses, comparing the measured oxygen saturation in the blood sample of oximetry data. The scatter of points around the line of identity demonstrates some problems with the stability of the oximetry signal, but there appears to be no consistent error. The r value for this regression is 0.58. Figure 2 shows the distribution of the percentage of recalibrations required for each patient. Most patients required recalibration of the oximetry catheter during about one-half of all calibration measurements. In five patients, SjvO2 readings were noted to be variable depending on the head position; in these patients, maintenance of a head position strictly perpendicular to the bed improved the readings. Because signal intensity and reflectance would frequently change quite rapidly, in many instances it was believed that the problem with the oximetry signal was related to the catheter lying against the vessel wall. Slight withdrawal of the catheter and the use of a con-

J. Neurosurg. / Volume 80 / March, 1994 463
tinuous slow heparin infusion to maintain the patency of the catheter tip often helped to stabilize the signal and reduce the number of recalibrations necessary. There were no complications directly attributed to the indwelling catheter.

A typical tracing of the simultaneous continuous measurements of $\text{SjvO}_2$, $\text{SaO}_2$, ICP, and MABP, displayed at one reading/sec in one patient over about a 70-minute period, is demonstrated in Fig. 3 left and center. At three times during this period (10, 36, and 49 minutes), endotracheal suction was carried out to clear the airway of this severely congested patient. The simultaneous increases in MABP and ICP can easily be seen in conjunction with these suctioning maneuvers. With each elevation in blood pressure, there is a concomitant increase in $\text{SjvO}_2$, suggesting an increase in CBF at these times. Additionally, the magnitude of the response of $\text{SjvO}_2$ appears to be proportionally related to the degree of blood pressure elevation, although the return to baseline for $\text{SjvO}_2$ is somewhat delayed when compared to that for ICP and MABP. Figure 3 right shows the tracing of the calculated cerebral perfusion pressure (CPP) and cerebral extraction of oxygen (CEO$_2$) defined as the difference between $\text{SaO}_2$ and $\text{SjvO}_2$ during the same 70-minute period. As anticipated, increases in CPP resulted in marked decreases in CEO$_2$, suggesting that the driving force for this increased CBF may be the increased CPP. Elevations in ICP for these short periods also appear to be related to increased blood flow, most likely via vascular distention.

In the group of patients studied, there were 120 instances of acute MABP elevations of more than 8 torr (mean ± standard deviation: 32 ± 13 torr) due to endotracheal suctioning. In these cases the calibration of the oximetry catheter before and after the event was accurate to within 5% saturation. In 96 of these measurements, there was an associated increase in the ICP of 5 torr or more, with a mean increase of 16.6 ± 10.2 torr. The mean CPP increased from 80.7 ± 11.9 to 97.4 ± 18.0 torr during the suctioning episodes. Mean $\text{SjvO}_2$ was 0.62 ± 0.10 before the CPP elevation and rose to a peak of 0.77 ± 0.10 at the point of peak CPP elevation. Increases in MABP, ICP, CPP, and $\text{SjvO}_2$ were all statistically significant ($p < 0.05$).

To examine steady-state pressure autoregulation, only those instances where blood pressure elevations persisted for more than 5 minutes were analyzed; 37 such occurrences were found. In these cases, the MABP elevations lasted for a mean of 16.0 ± 8.4 minutes. In 34 of these intervals, the $\text{SjvO}_2$ elevations also persisted for more than 5 minutes, with an average duration of 15.0 ± 12.4 minutes, suggesting a loss of autoregulatory vasoconstriction. The duration of the associated ICP elevations averaged 11.7 ± 6.1 minutes.

To determine whether hyperemia contributed to the dysfunction of autoregulation, ICBF data for each patient were classified as being normal or hyperemic. Figure 4 shows the peak and steady-state responses to elevations of arterial pressure in five patients (15 observations) with normal flow and in six patients (15 observations) with definite hyperemia. In one patient, one of the three observations was determined to have been taken while the patient was hyperemic. The CPP elevations between the two groups do not appear to be different. For CEO$_2$, the hyperemic patients had a statistically significantly lower value at baseline, but the difference became insignificant as the CPP increased and then returned toward normal levels.

Figure 5 examines the relationship between the persistent changes in CEO$_2$ and CPP in patients with normal and hyperemic flow. Intact autoregulation suggests that no change would occur in CEO$_2$ as the CPP was altered, indicated by a horizontal line emanating from the zero point of the y-axis. However, the autoregulatory index of Obrist, et al. ($\Delta$% CPP/$\Delta$% cerebral vascular resistance (CVR) > 2, where CVR = CPP/ICBF) is demonstrated as a line slanting downward from the zero point on the y-axis. Of the 26 observations, 18 were below this line, suggesting a lack of autoregulation after head injury in 60% of observations in these patients. No difference between the hyperemia and the normal flow groups was found, with the $\Delta$% CEO$_2$/$\Delta$% CPP being 0.37 ± 0.19 in the hyperemia group and 0.49 ± 0.22 in the normal flow group.

**Discussion**

**Continuous Jugular Venous Oximetry**

The ability to continuously measure $\text{SjvO}_2$ and, via the Fick principle, make inferences about CBF appeals to clinicians treating head-injured patients. Presently, the availability of only intermittent CBF measurements with technology found in large centers ($^{133}$Xe washout techniques) limits our understanding of cerebral hemodynamics and metabolism after injury. For the individual patient, merely monitoring ICP and from this inferring CBF via the Monroe-Kellie principle ignores much of the available information on the influence of cerebral venous blood volume on ICP as well as the altered autoregulation after injury. While continuous jugular venous oximetry may seem like a simple method for the continuous measurement of the ratio...
Jugular venous oximetry following head injury

![Graph showing jugular venous oxygen saturation (SjvO₂) and arterial oxygen saturation (SaO₂) over time](image)

**Fig. 3.** *Left and Center:* Representative tracing of the measurements of jugular bulb venous oxygen saturation (SjvO₂) and arterial oxygen saturation (SaO₂, left), and intracranial pressure (ICP) and mean arterial blood pressure (MABP, center) in a single patient. Elevations of MABP in response to endotracheal suctioning at 10, 36, and 49 minutes can easily be seen, and are associated with simultaneous elevations of ICP and SjvO₂. The elevated SjvO₂ for short intervals is consistent with a higher cerebral blood flow. *Right:* A tracing of the calculated cerebral perfusion pressure (CPP) and cerebral extraction of oxygen (CEO₂) during the same 70-minute period. It appears that elevations in CPP resulted in a reduction in CEO₂, suggesting that increased CPP resulted in increased cerebral blood flow.

![Graphs showing cerebral perfusion pressure (CPP) and cerebral extraction of oxygen (CEO₂)](image)

**Fig. 4.** Graphs showing the temporal relationship of cerebral perfusion pressure (CPP, *left*) and cerebral extraction of oxygen (CEO₂, *right*) changes to elevations of arterial pressure in six patients with hyperemia (triangles) and in five patients with normal cerebral blood flow (squares). In both groups the increase in CPP resulted in a similar fall in CEO₂, suggesting no influence of hyperemia on this response.

...of metabolism to CBF, its successful use as an effective monitoring tool will be determined by the availability and accuracy of the oximetry technique.

Because of their popularity in the continuous measurements of pulmonary SjvO₂ for cardiovascular monitoring in critically ill patients, oximetry monitors have found their way into most intensive care units. The fiberoptic catheters presently being used for jugular venous oximetry in this and other studies were primarily developed for neonatal umbilical arteries. The size of these devices (No. 4 French) makes them particularly easy to insert retrogradely into the jugular vein and the cost (approximately $150) is comparable with other specialty medical devices. Monitoring can be performed at the bedside without any interference in standard clinical care. However, since these catheters are thin-walled, their pliability and position retrograde to blood flow make them especially susceptible to angulation against the wall of the vein with subsequent distortion of the “light-intensity” signal. In this study, when calibration checks were performed every 4 to 8 hours, the mean recalibration rate was 38% per catheter. Many of the recalibrations were necessary after the monitor screen displayed prolonged periods of unsatisfactory light-intensity signals from the catheter. Repositioning of the head and neck, slight withdrawal of the catheter, and a continuous flush of heparinized saline appear to improve the reliability of the catheter.

Our experience is similar to that of others using this catheter. Andrews and Dearden² reported a direct comparison of SjvO₂ measurement from oximetry in simultaneous blood samples and found substantial variation between the values at the time of insertion. After in vivo calibration, the values correlated closely but after 12 hours the values again became disparate. Steinberg, et al.,²⁰ found that, when SjvO₂ fell below 50%, only about one-half of the head-injured patients had the oximetry value confirmed as accurate. Usually, a low light-intensity reading from the catheter tip was the cause of the inaccurate reading. In their study, the correlation between oximetry and blood samples was significant, but the r value was 0.6, similar to our data.

Clearly, if this monitoring technique is to be useful, a new catheter needs to be developed. Characteristics of an optimal catheter include an increased stiffness to prevent change in conformation as it lies facing blood...
flow, a more central location of the light fiber at the tip of the catheter, and a mechanism to center the catheter in the vessel. Our initial experience with a catheter having the above characteristics is encouraging.

Inferences on CBF alterations based on SjvO₂ and SaO₂ changes involve several assumptions regarding the physiology and pathophysiology of the injured brain: 1) that CMRO₂ remains constant over the period of measurement; 2) that hemoglobin concentration remains constant; 3) that dissolved oxygen can be ignored; and 4) that cerebral tissue and blood volume stores of oxygen contribute little to transients in arteriovenous saturation differences.

The inability to verify that CMRO₂ remains constant is the principal difficulty with this technique. The rate can vary widely with neural activity, and reports of intermittent measures in brain injury indicate changes in CMRO₂. Decreases in the SaO₂ to SjvO₂ difference, if not due to an increase in blood flow, could be the result of a decrease in CMRO₂ during suctioning. Since coughing and agitation often accompany this procedure, it is more likely that CMRO₂ increases and, therefore, estimates of blood flow changes are underestimated. Similarly, it is unlikely that hemoglobin concentration changes are occurring over the short time period of these studies. For longer-term studies, changes in both CMRO₂ and hemoglobin concentrations must be accounted for in order to infer changes in CBF. The percentage of oxygen carried in physical solution should be relatively small if PO₂ is less than 200 torr and hemoglobin is not at a low level. Inferences of cardiac output and, in this case, CBF are little influenced by ignoring this contribution.¹⁶

Assuming that CMRO₂ remains constant, changes in blood flow reflected by the SaO₂ to SjvO₂ difference will be delayed from the actual CBF change because of the time required to reach a new steady state. Cerebral blood volume is estimated to be 5% to 10% of cranial volume, but may be more after traumatic brain injury. Oxygen solubility in brain tissue is small relative to that in blood; a conservative estimate would equate the oxygen capacity of 100 g of brain to 20 ml of blood. Average blood flow in the brain is 40 ml/min/100 g and the mean transit time for oxygen will therefore be 30 seconds. Thus, changes in SjvO₂ will be 63% of the way to the new steady state within 30 seconds and 89% of the way in 1 minute.

**Autoregulation After Head Injury**

Data to determine the preservation of autoregulation after head injury were obtained only during periods of catheter reliability evidenced by accurate in vivo calibration determinations before and after the observation. The arterial hypertension induced by endotracheal suction was studied for several reasons. First, in a nonparalyzed patient this maneuver results reliably and reproducibly in a transient elevation in MABP that usually returns to baseline within 5 to 10 minutes. Second, this is a necessary intermittent bedside treatment to clear lung secretions, which means that no special vasoconstrictors or ionotropic agents were needed to induce elevation of blood pressure. Finally, bedside interventions such as endotracheal suction usually tend to increase ICP transiently, which may be of some concern to the clinician. For these reasons, the decision was made to determine the effect of the transient and sustained rises in ICP on CBF as measured by changes in SjvO₂.

Using SjvO₂ data during periods of arterial hypertension in all patients, we found a concomitant increase in both mean ICP and mean SjvO₂. The SjvO₂ data demonstrate that a close correlation between rapid rises in CPP and CBF is present. This associated increase in ICP must be secondary to the increased blood flow and associated increased volume. Early changes in CBF in response to increased MABP are not unexpected since autoregulation in response to sudden increases in intravascular pressure requires some time to become apparent. In pial vessels, the onset of the response to reduced arterial pressure occurs within 3 to 7 seconds, with a steady state achieved within approximately 1 minute.¹⁹ In most of our cases of arterial hypertension, the SjvO₂ did not return to baseline more rapidly than the arterial pressure, suggesting some lack of autoregulation for these transient events. Since SjvO₂ is somewhat dependent on “oxygen washout” from the brain, rapid responses are not expected; however, because CBF is high relative to the oxygen storage capacity of the brain and to brain blood volume, equilibration should be relatively rapid.

Other studies of autoregulation were carried out in situations in which persistent blood pressure elevation occurred. Since pressure autoregulation for CBF should be apparent within 5 minutes, we determined

---

466

---

J. B. Fortune, et al.

**Fig. 5.** Graph demonstrating the relationship between changes in cerebral extraction of oxygen (CMRO₂) and changes in cerebral perfusion pressure (CPP) in head-injured patients with hyperemia (triangles) and normal cerebral blood flow (squares). Intact autoregulation suggests that no change would occur in CMRO₂ as the CPP was altered (horizontal dotted line). The autoregulatory index of Obst等,⁷ is indicated by downward slanted dotted line originating from the zero point on the y-axis. Data points lying above this line would indicate intact autoregulation and those below the line defective autoregulation. Autoregulation appears to be defective in 60% of observations and is not related to the presence of hyperemic blood flow.
Jugular venous oximetry following head injury

the number of observations in which SjvO₂ remained elevated for longer than 5 minutes in the presence of arterial hypertension. In 65% of the cases, we found persistently elevated SjvO₂, suggestive of defective autoregulation. This percentage is greater than that reported by other authors. Bouma and Muizelaar, who altered blood pressure with administration of vasoconstrictors and vasodilators, found autoregulation to be intact in 59% of the cases when blood pressure was elevated and in 78% of the cases in which blood pressure was reduced. In a study of children, Muizelaar, et al., found autoregulation to be intact in 59% of the measurements.

The difference between our data and the findings of others may relate to the mechanism by which hypertension was induced or to the time when the response was noted. Typically, arterial hypertension in our patients was induced quite rapidly with gradual reduction over the ensuing 15 to 20 minutes, while in other studies the hypertension is induced gradually and maintained over a period of 30 minutes. The sudden rise in blood pressure in response to endotracheal suction followed by a gradual decline may result in a different autoregulatory response because of sudden vessel distention. Additionally, since our CBF measurements were made at 5 minutes after the onset of hypertension rather than at 30 minutes (as in other studies), we may be making the measurement prior to the full autoregulatory response, that is, autoregulation in some patients may proceed slowly. On the other hand, individual observations show that CEO₂ changes back to baseline tend to occur more slowly than the decrease in ICP and MABP.

Hyperemia, as detected by the ICBF measurements, did not appear to influence the presence or absence of autoregulation in our patients. As expected, the CEO₂ level was less in the hyperemic patients. As the arterial pressure was increased, these differences were no longer significant. The lack of effect of hyperemia on autoregulation is consistent with the findings of Bouma and Muizelaar, who showed no influence of hyperemia on autoregulation, which was intact in 63% of hyperemic patients. On the other hand, Muizelaar, et al., showed that, in children, absolute hyperemia resulted in a statistically significant increase in the number of patients with impaired autoregulation.

Clinical Implications

An easily obtainable continuous measure of CBF would be an excellent addition to the monitoring profile presently available for head-injured patients. In most head-injury treatment centers, measurements of ICP, arterial pressure, and intermittent blood gas levels provide the clinical input into the decisions necessary to prevent secondary brain injury. Intracranial pressure monitoring is certainly helpful as a guide for prevention of increased swelling, which may compress the brain stem and lead to herniation. However, the concept that ICP is an inverse measurement of CBF, utilizing the Monroe-Kellie principle, has many limitations. The standard use of hyperventilation to reduce ICP may lead to even further reductions of CBF, resulting in ischemia at the extreme. The presence of low blood flow may be undetectable by present monitoring techniques; the use of hyperventilation may actually be detrimental. The use of continuous jugular venous oximetry may be of some benefit in determining the need for and effectiveness of hyperventilation, as suggested by several authors.

Jugular venous oximetry may also be helpful for monitoring other treatment modalities, such as arterial hypertension or mannitol administration for reducing ICP via an intact autoregulatory mechanism. For this method to be effective, autoregulation must be intact. If autoregulation were defective, then increasing the arterial pressure might result in undesirable increases in ICP secondary to vascular distention. Jugular venous oximetry may be a useful technique for assessing autoregulatory status.

In this study, it has been shown that transient increases in ICP secondary to blood flow increases may not adversely affect cerebral perfusion. Frequently, bedside nursing staff are told to avoid procedures that might lead to these transient ICP increases. It was feared that manipulations such as endotracheal suctioning, repositioning of the patient, and routine bedside care would result in increasing ICP and reducing CBF. However, these transient episodes of arterial hypertension are more likely to result in increased CBF despite elevated ICP, and in fact it is probably the increased CBF that is causing the raised ICP. While these transient ICP increases may have a detrimental effect on brain-stem compression, CBF does not appear to be adversely affected.

Continuous measurement of SjvO₂ appears to have a role in the routine monitoring of severely head-injured patients in conjunction with ICP monitoring and optimization of the hemodynamics, and can offer significant independent information on CBF changes.

References


Manuscript received January 5, 1993.
Accepted in final form July 16, 1993.
This work was supported by National Institutes of Health, National Institute of Neurological Disorders and Stroke Grant 30303.
Address reprint requests to: John B. Fortune, M.D., Section of Trauma Surgery, Department of Surgery, A-61 GE, Albany Medical Center, Albany, New York 12208.