Continuous monitoring of jugular venous oxygen saturation in head-injured patients

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The continuous measurement of jugular venous oxygen saturation (SjvO₂) with a fiberoptic catheter is evaluated as a method of detecting cerebral ischemia after head injury. Forty-five patients admitted to the hospital in coma after severe head injury had continuous and simultaneous monitoring of SjvO₂, intracranial pressure, arterial oxygen saturation, and end-tidal CO₂. Cerebral blood flow, cerebral metabolic rates of oxygen and lactate, arterial and jugular venous blood gas levels, and hemoglobin concentration were measured every 8 hours for 1 to 11 days. Whenever SjvO₂ dropped to less than 50%, a standardized protocol was followed to confirm the validity of the desaturation and to establish its cause. Correlation of SjvO₂ values obtained by catheter and with direct measurement of O₂ saturation by a co-oximeter on venous blood withdrawn through the catheter was excellent after in vivo calibration when there was adequate light intensity at the catheter tip (176 measurements: $r = 0.87, p < 0.01$). A total of 60 episodes of jugular venous oxygen desaturation occurred in 45 patients. In 20 patients the desaturation value was confirmed by the co-oximeter. There were 33 episodes of desaturation in these 20 patients, due to the following causes: intracranial hypertension in 12 episodes, hypocardia in 10, arterial hypoxia in six, combinations of the above in three, systemic hypotension in one, and cerebral vasospasm in one. The incidence of jugular venous oxygen desaturations found in this study suggests that continuous monitoring of SjvO₂ may be of clinical value in patients with head injury.

**Key Words** · jugular venous oxygen saturation · cerebral blood flow · head injury · cerebral ischemia

The adverse effects of cerebral ischemia and hypoxia superimposed on severe head injury have been well established in laboratory and clinical studies. Hypoxia and hypotension after fluid-percussion injury in rats was shown to cause brain damage and a decrease in high-energy phosphates that was greater than when either insult occurred alone. Clinical studies have demonstrated that one-third of head-injured patients have sustained episodes of hypoxia and hypotension which in some cases adversely affected outcome. Ischemic brain damage has been found on neuropathological examination in 88% of head-injured patients. The incidence of ischemic damage and its severity was found to be unchanged in two series of patients studied at an interval of 10 years.

A clinically useful method for detecting cerebral ischemia should be sensitive enough to identify a reduction in cerebral oxygen delivery prior to neurological injury. The purpose of this study was to evaluate continuous measurement of jugular venous oxygen saturation (SjvO₂) by a fiberoptic catheter as a method of detecting cerebral ischemia. The reliability of the fiberoptic catheter and the incidence and causes of ischemia identified by jugular venous desaturation were investigated.

**Clinical Material and Methods**

**Patient Population and Management**

Between July 15, 1989, and November 30, 1990, 45 comatose patients (Glasgow Coma Scale ≤ 8) who had been admitted to Ben Taub General Hospital with severe head injury had measurements of SjvO₂. Ninety-one percent of the patients studied were male, with an average age of 31 ± 14 years (± standard deviation). Ninety-one percent of the patients studied had closed head injuries, while the remaining 9% had suffered gunshot wounds to the brain. On admission to the hospital, 82% of the patients were comatose. The initial GCS score was 4 or 5 in 24% of the patients and 6 to 8 in 58%. Eighteen percent of the patients had an initial GCS score greater than 8, but later deteriorated to coma. The mortality rate at 3 months after injury was 36%.
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All patients were managed with a protocol that emphasized prompt evacuation of intracranial hematomas and prevention of secondary injury to the brain. Patients were intubated and ventilated to maintain a PaO$_2$ greater than 100 mm Hg and a PaCO$_2$ of approximately 35 mm Hg. Intracranial pressure (ICP) was monitored, usually by ventriculostomy, and pressures greater than 20 mm Hg were treated. Medications included morphine for sedation, pancuronium, phenytoin, and antibiotic drugs. Steroids were not administered.

Intracranial hypertension was treated with cerebrospinal fluid drainage, hyperventilation (PaCO$_2$ 25 to 30 mm Hg), sedation, paralysis, and mannitol. Barbiturate coma was used only if intracranial hypertension was refractory to the above regimen.

**Technique of SjvO$_2$ Measurement**

To monitor SjvO$_2$, a No. 4 French fiberoptic oxygen saturation catheter* was inserted percutaneously into the internal jugular vein through a No. 4.5 French peel-away introducer.† The tip of the catheter was positioned in the jugular bulb, which was verified by x-ray film. The catheter was placed on the side of the most severe injury or on the right side if the injury was diffuse and was maintained as a central venous catheter with a continuous heparin flush device. No medications were given through the catheter.

The catheter was calibrated in vitro prior to insertion according to the colorimetric method supplied by the manufacturer. Immediately after insertion, and every 8 hours thereafter, in vivo verification of catheter calibration was performed by drawing a blood sample from the catheter and measuring the oxygen saturation on a co-oximeter.‡ If the catheter- and co-oximeter-derived values differed by more than 4%, the catheter was recalibrated to reflect the co-oximeter saturation.

The catheter display provided a value for reflected light intensity at 2-minute intervals. The adequacy of light intensity was an indicator of the correct position of the catheter tip in relation to the flow of blood in the jugular bulb. The value for SjvO$_2$ in normal individuals is 65%; values greater than 50% were considered to be acceptable.†

**Continuous Physiological Measurements**

The following physiological parameters were continuously monitored for as long as ICP monitoring was required, which ranged from 1 to 11 days after injury: arterial blood pressure via a radial artery cannula, arterial oxygen saturation (SaO$_2$), end-tidal CO$_2$, and SjvO$_2$. All parameters were recorded at approximately 1-minute intervals, stored in a computerized data base.

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* Fiberoptic oxygen saturation catheter manufactured by Abbott Laboratories, North Chicago, Illinois.
† Peel-away introducer manufactured by Cook Critical Care, Bloomington, Indiana.
‡ Co-oximeter, Model IL-282, manufactured by Instrumentation Laboratories, Lexington, Massachusetts.

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$p$ Corning 170 blood gas analyzer manufactured by Ciba Corning Diagnostics Corp., Medfield, Massachusetts.
∥ Lactate analyzer, Model YSI-236, manufactured by Yellow Springs Instruments, Yellow Springs, Ohio.
portions were tested with the chi-squared statistic for contingency tables.

Results

Reliability of Fiberoptic Catheter SjvO2 Measurements

During the course of monitoring in 45 patients, 361 simultaneous measurements were made of SjvO2 with the fiberoptic catheter and with the co-oximeter. The correlation between the values obtained with the fiberoptic catheter and the reference co-oximeter was significant ($r = 0.60$, $p < 0.01$). However, there were many outlying points that could be explained by one of two circumstances. The first cause was a low light-intensity reading from the catheter tip, indicating low blood flow and/or abutment of the catheter tip against the vessel wall. Thirty-three of the 45 patients had at least one referenced measurement at a time of poor catheter positioning. The second circumstance was the initial measurement of SjvO2 made at the time of the first in vivo calibration after placement of the catheter. When these points were not included in the analysis, the correlation between the fiberoptic catheter and the reference co-oximeter was excellent (176 measurements: $r = 0.87$, $p < 0.01$) (Fig. 2).

Very few technical difficulties occurred while the catheters were in place. On three occasions catheters were found on x-ray film to be looped in the internal jugular vein and the tip did not reach the jugular bulb. Blood in the lumen of two catheters clotted due to a lack of adequate flushing. The fiberoptic cable of one catheter broke, which required its removal. There were no infections or other complications in patient care referable to the use of the internal jugular catheters.

Episodes of Jugular Venous Desaturation

Sixty episodes of jugular venous oxygen desaturation, defined as an SjvO2 of less than 50% for more than 15 minutes, were identified in 45 patients. In 20 patients, the catheter measurement of desaturation was confirmed as being accurate and not due to artifact. In all but one of the artifactual decreases in SjvO2, the catheter light intensity was inadequate, indicating poor catheter positioning as the likely cause for the invalid reading. In these 20 patients there were 33 episodes of desaturation: 11 patients had one episode, five had two episodes, and four had three episodes of desaturation. The duration of the venous desaturation ranged from 15 minutes to 11 hours, with a median time of 35 minutes. Causes for the jugular venous desaturation included: intracranial hypertension (12 episodes); hypocarbia, defined as a PaCO2 less than 28 mm Hg (10 episodes); arterial hypoxia, defined as an SaO2 less than 90% (six episodes); combinations of the above (three episodes); and one episode each of systemic hypotension and cerebral vasospasm.

Decreases in SaO2 to between 85% and 90% were common, but these usually were not reflected by a decrease in the SjvO2. In a few patients, however, decreases in SaO2 were paralleled by decreases in the SjvO2 (Fig. 3). In the latter circumstance, CBF apparently did not increase sufficiently to compensate for the drop in
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Fig. 3. Graphs showing arterial oxygen saturation (SaO₂) and jugular venous oxygen saturation (SjvO₂) in a patient with jugular venous desaturation due to arterial hypoxia.

Fig. 5. Graphs showing mean arterial pressure (MAP) and jugular venous oxygen saturation (SjvO₂) in a patient with jugular venous desaturation due to systemic hypotension.

Fig. 6. Graphs showing cerebral perfusion pressure (CPP) and jugular venous oxygen saturation (SjvO₂) in a patient with jugular venous desaturation during a transient decrease in CPP secondary to elevated intracranial pressure.

Fig. 7. Graphs showing cerebral perfusion pressure (CPP) and jugular venous oxygen saturation (SjvO₂) in a patient with jugular venous desaturation due to refractory intracranial hypertension following evacuation of a subdural hematoma. Cerebral blood flow (CBF) ranged from 0.67 to 0.75 ml/gm/min during the first 4 days after the injury. On Day 5, the intracranial pressure was unresponsive to maximum medical therapy including pentobarbital. As CPP fell to 25 mm Hg, both pupils became fixed and dilated. With further decreases in CPP, the SjvO₂ fell to a low of 20%. CMRO₂ = cerebral metabolic rate of oxygen; CMRL = cerebral metabolic rate of lactate; CT = computerized tomography.

the arterial oxygen content. Hypocarbia regularly resulted in a reversible drop in the SjvO₂ (Fig. 4).

A decreased cerebral perfusion pressure can be caused by either systemic hypotension or intracranial hypertension. Systemic hypotension was identified reliably by jugular venous oxygen desaturation (Fig. 5); however, this desaturation was not found to be an early indicator of intracranial hypertension. Occasionally, decreases in SjvO₂ to less than 50% were seen with transient elevations of ICP (Fig. 6). More commonly, SjvO₂ increased or remained unchanged during brief ICP elevations. While the SjvO₂ dropped to less than 50% in all patients who died of intracranial hypertension, the venous desaturation typically occurred after clinical signs of herniation had developed (Fig. 7). When cerebral perfusion pressure dropped to very low levels, SjvO₂ returned to normal or elevated levels. In this situation, the jugular venous blood was probably extracerebral in origin.

CMRO₂ = cerebral metabolic rate of oxygen; CMRL = cerebral metabolic rate of lactate; CT = computerized tomography.
Association Between CBF, Jugular Venous Oxygen Desaturation, and Outcome

The 45 patients studied received a total of 363 CBF measurements, with an average of eight measurements per patient. Sixteen (36%) of the 45 patients had a reduced CBF, nine (20%) had a normal CBF, and 20 (44%) had an elevated CBF. There was no significant difference in age, initial GCS score, or type of injury among the three CBF categories. Among the 16 patients with reduced CBF, nine (56%) died, compared with three (33%) of the nine patients with normal CBF and four (20%) of the 20 patients with elevated CBF.

The incidence of jugular venous oxygen desaturation tended to be related to the CBF and to outcome, although the numbers of patients are small for this analysis. Of the 33 confirmed desaturations, 54% occurred in the reduced CBF group, while only 21% were in the normal CBF group and 24% were in the elevated CBF group. Of the 16 patients with a reduced CBF, 10 (63%) had at least one episode of desaturation, compared to three (33%) of the nine with normal CBF and seven (35%) of the 20 with elevated CBF (p = 0.19). Of the 25 patients without jugular desaturation, six (24%) died compared to five (45%) of the 11 patients with one confirmed episode of desaturation and five (55%) of the nine patients with more than one confirmed episode of jugular desaturation (p = 0.17).

Discussion

The present data indicate the episodes of jugular venous oxygen desaturation are common in patients with severe head injury, even when they are receiving intensive care with advanced cardiovascular and intracranial monitoring. These episodes of desaturation would not have been detected in most patients without continuous monitoring of the SjvO2. The potential utility of these measurements is underscored by the observation that patients with confirmed episodes of oxygen desaturation had a higher mortality rate than those without such episodes.

There have been few other studies assessing jugular bulb fiberoptic oximetry in patients with severe head injury. In one study of 10 head-injured patients, the authors noted rapid fluctuations in SjvO2 caused both by procedures performed on patients and by physiological phenomena. The authors noted that these fluctuations limited the usefulness of any single measurement.3 We also noted brief desaturation episodes in some patients that were related to routine procedures performed in the intensive care unit, most commonly suctioning of the endotracheal tube. However, those desaturations that were secondary to the pathophysiological sequelae of head injury tended to persist for longer periods of time.

In another study of 12 patients with continuous monitoring of SjvO2 and SaO2, Cruz4 found a significant difference in outcome between patients with episodes of systemic hypoxemia and those without these episodes. The author concluded that continuous monitoring was useful because it gave an outline assessment of oxygen delivery, established causal relationships for desaturation, and allowed for shorter response times to remedy hypoxemia once it was detected.

We have found two major limitations in monitoring cerebral ischemia with the fiberoptic catheter. While systemic causes of ischemia were readily identified with SjvO2 monitoring, the SjvO2 did not usually decrease with intracranial hypertension until after neurological signs of tentorial herniation were already observed. It may be that global CBF is preserved when the brain stem is selectively compressed. Alternatively, as CBF drops to very low levels, extracerebral contamination of jugular venous blood may increase proportionally and obscure the true saturation of cerebral venous blood.

Cerebral ischemia due to intracranial hypertension was commonly seen and knowledge of the SjvO2 provided information that was useful in managing intracranial hypertension. For example, while many patients could be hyperventilated to PaCO2 levels of 25 mm Hg in attempts to lower ICP, some patients had a significant drop in CBF in response to this maneuver, as illustrated in Fig. 4. Hypoperfusion due to hypocarbia was readily identified by jugular desaturation and was remedied by allowing the PaCO2 to rise. This observation may provide a basis for understanding the poorer outcomes reported in patients with severe head injury who were “prophylactically” hyperventilated.15

The second limitation of the fiberoptic catheter is the occurrence of artifacts in the oxygen saturation measurement caused by movement of the catheter within the jugular bulb. Almost one-half of the decreases in SjvO2 below 50% that were identified during monitoring were found to be incorrect. Usually, the light-intensity value for the catheter suggested interaction of the catheter with the vessel wall as the cause of the inaccurate measurements of SjvO2.

The algorithm shown in Fig. 1 provides a systematic approach to determining the cause of jugular desaturation and to treating it. Ideally, it would be best to monitor multiple physiological parameters continuously and to search methodically for the cause of desaturation when it occurs. However, in most instances the cause for desaturation can be easily deduced with routine laboratory determinations. While the methodology of measurement of SjvO2 has significant potential, the design of the fiberoptic oxygen saturation catheter needs to be improved to reduce artifactual readings before it can be introduced into clinical practice.

References

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Manuscript received June 10, 1991. This study was supported by National Institutes of Health Grant P01-NS27616.

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