Marked Regional Heterogeneity in Venous Oxygen Saturation in Severe Head Injury Studied by Superselective Intracranial Venous Sampling: Case Report

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OBJECTIVE: Continuous monitoring of jugular venous oxygen saturation ($SjvO_2$) is useful in the management of severe head injury. Abnormally high $SjvO_2$ values can be caused by increased cerebral blood flow, decreased cerebral metabolism, brain death, contamination from extracerebral venous blood, or traumatic arteriovenous fistula.

CLINICAL PRESENTATION: A 20-year-old man with severe head injury was diagnosed to have a traumatic dural carotid-cavernous sinus fistula on the day of trauma. Continuous left $SjvO_2$ monitoring from Days 4 to 12 revealed oxygen saturation ranging between 85 and 98%.

INTERVENTION: Superselective intracranial and extracranial venous sampling on Day 5 demonstrated marked regional heterogeneity in venous oxygen saturation as follows: superior sagittal sinus, 95 to 97%; straight sinus, 88%; right transverse sinus, 94%; left transverse sinus, 74%; right $SjvO_2$, 95%; left $SjvO_2$, 89%; the basilar plexus, 99%; right internal jugular vein, 98%; the left internal jugular vein, 94%. Extremely high oxygen saturation in the superior sagittal sinus and basilar plexus was attributed to severe brain damage and carotid-cavernous sinus fistula, respectively.

CONCLUSION: Although jugular bulb oximetry is useful in the management of severe head injury, high oxygen saturation values should be interpreted with caution because they cannot show the intracranial heterogeneity of venous oxygen saturation. (Neurosurgery 45:1469–1473, 1999)

Key words: Head injury, Hyperemia, Jugular bulb oximetry, Traumatic carotid-cavernous sinus fistula

In the last decade, continuous jugular bulb oximetry, pioneered by Kim et al. (9) in 1976, has become an established method of monitoring to detect cerebral ischemia/hypoxia in patients with severe head injury (5, 7, 11, 15–17). Abnormal balance between oxygen supply and demand for the brain can be monitored continuously by jugular venous oxygen saturation ($SjvO_2$). Thus, abnormally high or low $SjvO_2$ can be caused by an imbalance of oxygenation in the brain.

We report a patient with severe head injury with a traumatic dural carotid-cavernous sinus fistula (CCF) whose $SjvO_2$ level was abnormally high. Superselective intracranial venous sampling, however, revealed marked regional heterogeneity of venous oxygen saturation. We discuss heterogeneous oxygen saturation and the usefulness of continuous $SjvO_2$ monitoring in severe head injury.

CASE PRESENTATION

A 20-year-old man with a severe head injury was transferred to us in a state of cardiopulmonary arrest as a result of massive oronasal bleeding. His blood pressure was not measurable, and his heart rate was 30 to 40 beats/minute at admission. No respiration was observed. Glasgow Coma Scale (19) score was 3. His pupils were anisocoric and unreactive to light (right, 6.0 mm; left, 5.0 mm). A computed tomographic scan of the brain revealed marked pneumocephalus in the subarachnoid and subdural spaces (*Fig. 1A*). After successful cardiopulmonary resuscitation and massive blood transfusion, diagnostic and therapeutic angiography was performed 80 minutes after trauma. Active bleeding from the bilateral facial and sphenopalatine arteries was controlled by superselective embolization.

Carotid angiograms at the time of embolization revealed a small dural CCF of Barrow Type B (1) in the left cavernous sinus caused by laceration of the meningohypophyseal trunk of the left internal carotid artery, but there was no CCF in the right cavernous sinus. From Day 4 to Day 12, $SjvO_2$ was monitored with a 5.5-French fiberoptic catheter in the left jugular bulb and the Oximetrix oximetry system (Abbott Laboratories, North Chicago, IL). $SjvO_2$ values ranged between 85 and 98% from Day 4 to Day 12, which was confirmed by in vitro oximetry. Low-dose barbiturate therapy with pentobarbital sodium at a dose of 2.0 mg/kg/h was required to control increased intracranial pressure from Day 3 to Day 10, but high-dose therapy was not applied to avoid hypotension. A computed tomographic scan on Day 6 revealed diffuse brain swelling and bitemporal thin epidural hematomas (*Fig. 1B*). $SjvO_2$ values re-

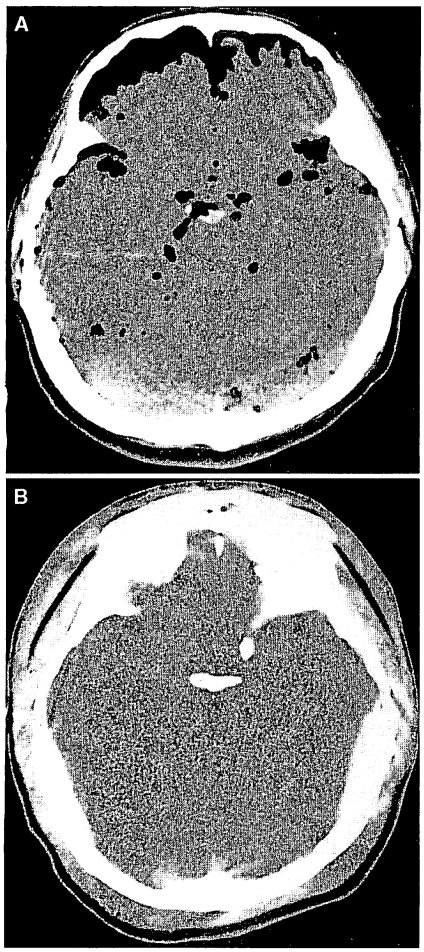

FIGURE 1. *A*, computed tomographic scan at admission showing marked pneumocephalus in the subarachnoid and subdural spaces, but no brain swelling. *B*, computed tomographic scan on Day 6 showing diffuse brain swelling and bitemporal thin epidural hematomas.

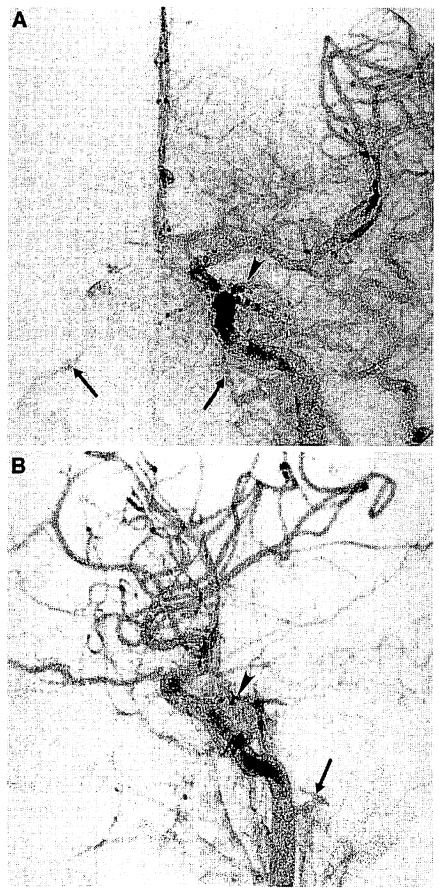

FIGURE 2. Left carotid angiograms (*A*, frontal view; *B*, lateral view) showing traumatic dural CCF (*arrowheads*), which drains to the bilateral inferior petrosal sinuses (*arrows*) and then to the bilateral internal jugular veins.

mained between 90 and 93% on Days 11 and 12, even after discontinuation of barbiturate therapy.

After informed consent was obtained from the patient's family, follow-up angiography and superselective intracranial venous sampling were performed on Day 5 to investigate the cause of the abnormally high $SjvO_2$ level. Carotid angiograms revealed bilateral small dural CCFs, which drained to the bilateral inferior petrosal sinuses. There was no cortical drainage (*Fig. 2*). At that time, intracranial pressure was approximately 25 to 30 mm Hg, even under barbiturate therapy, whereas cerebral perfusion pressure was approximately 65 to 70 mm Hg. Superselective venous sampling was performed at various intracranial locations, bilateral jugular bulbs, and internal jugular veins through a microcatheter navigated by a transfemoral approach. Oxygen tension (mm Hg) and saturation (%) were measured using the drawn blood samples at each location (*Table 1*). Hemoglobin concentration was 13.1 g/dl. In the basilar plexus, oxygen tension was 260 mm Hg, and saturation was 99%, as a result of arteriovenous fistula in the cavernous sinuses, whereas in the right femoral artery, oxygen tension was 471 mm Hg, and saturation was 100%. In the superior sagittal sinus, oxygen tension was 80 mm Hg, and saturation was 97%; whereas in the right and left transverse sinuses, oxygen tensions were 60 and 33 mm Hg, and saturation values were 94 and 74%, respectively. Right and left $SjvO_2$ saturation levels were 95 and 89%, respectively. Higher saturation in the right than in the left jugular bulb was attributed to the larger arteriovenous fistula draining to the right jugular bulb. The data on the venous oxygen saturation are shown in *Figure 3*.

Follow-up cerebral angiography at 2 months after trauma showed a right dural CCF (Barrow Type B) and no CCF in the left cavernous sinus. The right dural CCF was finally occluded at 3 months by transarterial embolization with interlocking detachable coils (Target Therapeutics, Fremont, CA). The patient remained in a persistent vegetative state at the final follow-up 6 months after trauma.

DISCUSSION

Jugular bulb oximetry

Jugular bulb oximetry measures the balance between cerebral oxygen delivery and consumption and has been reported to be a useful method for monitoring patients with severe head injury (5, 7, 11, 15–17). The monitoring techniques involve unilateral placement of the fiberoptic catheter in the jugular bulb and continuous monitoring of $SjvO_2$. Intermittent in vitro calibration is required to correct the values of the fiberoptic catheter.

Normal $SjvO_2$ values range between 55 and 71%, with a mean of 61.8% (8). Jugular bulb desaturation is usually defined as a value below 50 or 55% for more than 10 or 15 minutes (11, 16, 17). Desaturation is caused by either a cerebral cause (increased intracranial pressure), a systemic cause (hypocapnia, hypoxia, hypotension, anemia), or a combination of the two (5, 11, 15–17). Conversely, high $SjvO_2$ values (>75%) indicate that cerebral oxygen delivery exceeds cerebral consumption, as a result of uncoupling between cerebral blood flow and metabolism (impaired autoregulation to blood pressure changes and/or decreased cerebral metabolism), increased arterial oxygen content, brain death, or contamination

TABLE 1. Oxygen Saturation and Tension in the Intracranial and Extracranial Venous System[a]

Location	Location in *Figure 3*	Saturation (%)	Tension (mm Hg)
Superior sagittal sinus	A	97	80
Superior sagittal sinus	B	95	69
Torcular herophili	C	90	49
Straight sinus	D	88	44
Right transverse sinus	E	94	60
Left transverse sinus	F	74	33
Right sigmoid sinus	G	94	59
Left sigmoid sinus	H	80	38
Right jugular bulb	I	95	64
Left jugular bulb	J	89	49
Basilar plexus	K	99	260
Right jugular vein	L	98	99
Left jugular vein	M	94	58
Right femoral artery	—	100	471

[a] **Locations from A to M correspond with those in *Figure 3*.**

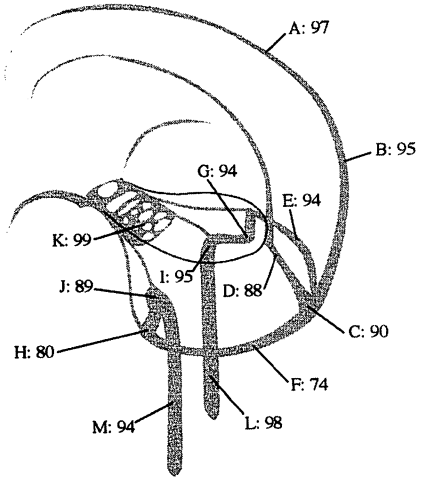

FIGURE 3. Superselective venous sampling in the intracranial and extracranial venous system. Data at each location indicate oxygen saturation (%). Oxygen saturation in the right femoral artery is 100%. Locations from *A* to *M* correspond with those in *Table 1*.

from extracerebral venous blood (6, 13, 14). However, increased arterial oxygen content alone does not result in such an abnormally high $SjvO_2$ value as occurred in our patient. There was also no chance of contamination from the extracranial venous blood (except for samples from the jugular bulbs and internal jugular veins) because venous sampling was performed purely intracranially through a microcatheter.

High oxygen saturation values and $SjvO_2$

Intracranial venous sampling data in our patient showed higher oxygen saturation in the superior sagittal sinus (97%) than in the straight sinus, torcular herophili, and transverse and sigmoid sinuses. These data could be interpreted to mean that extremely high oxygen saturation was chiefly caused by severe parenchymal damage for the following reasons: decreased metabolism induced by barbiturate therapy did not contribute extensively to high oxygen saturation in our patient because oxygen saturation in the other venous sinuses did not show such high oxygen saturation; a dose of barbiturate was not so high as to avoid hypotension; and discontinuation of barbiturate therapy did not decrease $SjvO_2$. CCF did not contribute to high oxygen saturation in the superior sagittal sinus because the arteriovenous fistula in the cavernous sinus was small, and there was no cortical drainage.

Traumatic CCF and $SjvO_2$

Five cases of traumatic CCFs were reported to be detected by jugular bulb oximetry, which disclosed abnormally high $SjvO_2$ levels (2–4, 6). Direct venous sampling in our patient revealed extremely high oxygen saturation when sampled from the basilar plexus. However, continuous jugular venous oximetry did not show such a high saturation because of a mixture of shunted blood and intracranial venous return. In the early stage of severe head injury, especially in cases with concomitant cranial base fracture, not only direct CCFs, but also dural (indirect) CCFs, are occasionally observed (3). Komiyama et al. (10) reported four of eight patients (50%) with severe craniofacial injury in whom angiographic study on the day of trauma revealed traumatic dural CCFs. In one patient, traumatic middle meningeal arteriovenous fistula also was discovered. This implies that a large number of patients with severe head injury may develop high $SjvO_2$ levels as a result of concomitant traumatic arteriovenous fistulae. Although a discrepancy in $SjvO_2$ values between the bilateral jugular bulbs in severe head injury has been reported (12, 18), we think that it could be attributed, in some cases, to the presence of undetected traumatic arteriovenous fistulae.

Heterogeneity of regional oxygen saturation

Superselective venous sampling through the microcatheter disclosed heterogeneity of regional oxygen saturation in our patient. The oxygen saturation values in the superficial venous drainage system collected in the superior sagittal sinus (95–97%) were much higher than in the deep venous drainage system collected in the straight sinus (88%). This indicates that the majority of the cerebral cortex was more severely damaged than the subcortical structures. This heterogeneity of regional oxygen saturation in severe head injury may be overlooked by $SjvO_2$ monitoring alone.

CONCLUSION

In the case of abnormally high $SjvO_2$ levels, increased cerebral blood flow, decreased cerebral metabolism, a traumatic arteriovenous fistula, brain death, contamination from extracerebral venous blood, or a combination of these factors should be suspected. Although jugular bulb oximetry is useful in the management of severe head injury, high oxygen saturation values should be interpreted with caution because they

cannot show the intracranial heterogeneity of venous oxygen saturation.

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COMMENTS

In a surprisingly aggressive clinical investigation of a severely injured patient, Komiyama et al. have studied the venous oxygen saturation ($SjvO_2$) in multiple intracranial and juxtacranial locations and related it to jugular venous oxygen saturation. Perhaps their most interesting finding was that sagittal sinus saturation was high compared with the saturation of blood in the straight sinus and left transverse sinus (97% versus 88% and 74%, respectively), demonstrating a regional difference. The authors imply that this difference is a result of regional heterogeneity in brain injury and, subsequently, metabolism. In other words, the data reflect a relative preservation of metabolism of the deeper structures of the brain compared with the cortex. Considering the patient's Glasgow Coma Scale score (3) and his outcome (persistent vegetative state), this proved to be a reasonable conclusion.

The difficulty in interpreting this study, and in interpreting venous oxygen saturation in general, is that venous saturation is the product of blood flow and metabolism; therefore, to make conclusions about one of the variables, the other must be known. The authors conclude that the data reflect regional differences in brain "damage." However, the reader should be aware of other explanations, such as regional differences in blood flow or differences in metabolism that are not caused by injury per se, which may also contribute to their findings. For instance, the authors acknowledge that the presence of an arteriovenous fistula will contaminate their data, as metabolism of the brain is bypassed. However, they disregard the contribution of the fistula to the venous sampling data of the intracranial circulation, in which it may heterogeneously alter the pressure dynamics of the system. Furthermore, the authors trivialize the contribution of barbiturate administration, although there are no electroencephalographic data to measure the extent of metabolic suppression. Barbiturates may affect the metabolism of the brain differentially. Temperature is also an important determinant of metabolism and hemoglobin-oxygen binding, but it is unlikely that there were significant regional differences in temperature that could account for the differences in the data. Despite these caveats, I think that this case report contributes to our understanding and interpretation of $SjvO_2$ data and, as such, is an important addition to the literature on this subject.

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Continuous jugular bulb oximetry has become an accepted technique to monitor global cerebral ischemia/hyperoxia in patients with severe head injury. Jugular bulb desaturation (<50%) is observed during episodes of ischemia/hypoxia/intracranial pressure increase, whereas high values (>75%) are suggestive of metabolic uncoupling, near brain death situations, and, although rare, as shown in this case, arteriovenous fistulae.

In this case study, Komiyama et al. describe continuous left jugular bulb oxygen saturation in a complex and

difficult-to-manage patient with severe traumatic brain injury and a small basal traumatic dural carotid-cavernous sinus fistula (CCF). The patient received intracranial pressure-directed management, which included low-dose barbiturates. At admission, a Barrow Type B, left-sided CCF was found and was treated with embolization. On retrograde venous cannulation, a marked regional heterogeneity in cerebral venous oxygen saturation was found, with the highest oxygen saturation in the superior sagittal sinus and in the basilar plexus (>95%). The authors demonstrated the heterogeneity of venous cerebral oxygen saturation after traumatic brain injury. This suggests a need for caution in interpretation of high oxygen saturation.

In our opinion, the strongest message of this study is the heterogeneity of the cerebral oxygen saturation at different venous locations. We agree with the authors that the very high saturation in the superior sagittal sinus is most likely caused by the severe parenchymal injuries and intracerebral shunting in these regions. Unfortunately, no xenon cerebral blood flow studies were performed to confirm this speculation.

This report shows the problems and difficulties in interpreting and monitoring brain oxygenation and metabolism after traumatic brain injury. The regional heterogeneity of cerebral venous oxygen saturation is clearly documented here and needs to be taken into consideration when treating these patients. A progressive increase in jugular oxygen saturation in a patient who is clearly not brain dead or hyperemic may rarely be the result of an arteriovenous fistula.

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In this patient with severe craniofacial trauma and a CCF, persistently elevated $SjvO_2$ values prompted repeat cerebral angiography and superselective venous sampling on postinjury Day 5. This study revealed global elevations in venous oxygen saturation but with considerable heterogeneity in the degree of increased saturation. Most impressive were the high saturation values in the superior sagittal sinus and the right transverse sinus. Given that there was no drainage of the CCF into the cortical venous system, these high values are most consistent with a severely hypometabolic state, resulting from the initial brain injury and compounded by prolonged hypotension and hypoxia. As the authors state, it is unlikely that barbiturate therapy played a significant role in this metabolic suppression because $SjvO_2$ values did not change appreciably after barbiturates were stopped.

This report demonstrates how jugular bulb oximetry can be used in the diagnosis and treatment of patients with suspected CCF. In this patient and in others previously reported, traumatic CCFs are typically associated with cranial base fractures. In a patient with the combination of markedly elevated jugular oxygen saturation values and a cranial base fracture observed on computed tomography, cerebral angiography should be performed. This case also clearly demonstrates the major limitation of jugular bulb oximetry, namely in its inability to detect regional heterogeneity in cerebral oxygen saturation.

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