Cerebral Monitoring: Jugular Venous Oximetry

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xygenation of cerebral venous outflow has been investigated as a neuromonitor for more than 50 years (1–3). Currently, jugular venous oxygen saturation (SjVO₂) provides an indirect assessment of cerebral oxygen use and is used to guide physiologic management decisions in a variety of clinical paradigms (4,5). An overview of SjVO₂ monitoring and an update on its clinical applications follow.

Placement of SjVO₂ Catheters

Anatomy

The internal jugular vein exits the skull and continues its course, within the carotid sheath, beneath the sternocleidomastoid muscle in a posterolateral approximation to the carotid artery. The jugular bulb is the dilated portion of the jugular vein just below the base of the skull and is the preferred site for blood sampling (see Figure 1).

Although blood in the jugular bulb is derived from both cerebral hemispheres (approximately 70% ipsilateral and 30% contralateral) (6–8), it is generally accepted that most patients have a dominant side of venous drainage, usually the right (9,10). The two lateral sinuses that drain to the jugular bulbs differ in size in 88% of patients (11), and mixing of cerebral venous blood within the sinuses is incomplete (1,12)

Jugular Venous Sampling

The jugular bulb may be punctured directly by a needle inserted 1 cm below and 1 cm anterior to the mastoid process (1). Alternatively, an intravascular catheter, similar to those used for central venous pressure monitoring, may be placed retrograde, via the internal jugular vein, into the jugular bulb (13).

More recently, reflectance oximetry, by using a fiberoptic catheter (see Figure 1) in a manner analogous to monitoring of mixed venous oxygen saturation in the pulmonary artery, has allowed for continuous SiVO₂ monitoring (14–16). Fiberoptic oximetry is based on the unique light absorption spectrum of oxyhemoglobin. The Baxter-Edwards system (Edslab Sat II, Baxter Edwards Critical Care Division, Irvine, CA) uses two wavelengths of light for reflectance spectrophotometry and is calibrated against a sample of the patient's blood. Conversely, Abbott's system (Opticath Oximetrix, Abbott Critical Care System, Abbott Park, IL) uses three wavelengths of light and may be calibrated *in vivo* (against the patient's blood) or *in* vitro (built-in calibration). A SjVO₂ catheter contains two optical fibers. Light is directed into the blood by one of the fibers, reflected back to the second fiber, and transmitted to a photosensor. The photosensor measures the absorption of the reflected light at the various wavelengths with SiVO2 displayed as a percentage of oxygenated hemoglobin to total hemoglobin. For SjVO₂ catheters using two wavelengths of light, the patient's hemoglobin concentration must be manually entered, with each SiVO₂ value dependent on the validity of the entered value to current conditions. For catheters using three wavelengths, the hemoglobin concentration is calculated from the absorption spectrum, allowing continuous, real-time monitoring of $SjVO_2$.

Which Side Should be Monitored?

In patients with bilateral brain injury, the catheter is usually placed in the internal jugular vein on the side of dominant drainage, usually the right (10,17). In the presence of a focal brain injury, it is controversial if the catheter should be placed on the side ipsilateral to brain injury or on the dominant side, if different. Stochetti et al. (18) noted that the proportion of headinjured patients with relevant discrepancies of SjVO₂ between the jugular veins is quite high. Fifteen of 32 patients showed oxygen saturation differences >15% between the two jugular veins, with only 8 patients having consistent differences of <5%. Beards et al. (9) proposed that asymmetry of >10%, in SjVO₂ values, occurs 65% of the time.

The dominant side may be determined by comparing the intracranial pressure (ICP) increase caused by manual compression of each internal jugular vein (19), by computerized tomographic assessment of jugular

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560 MEDICAL INTELLIGENCE ANESTH ANALG 2000:90:559-66

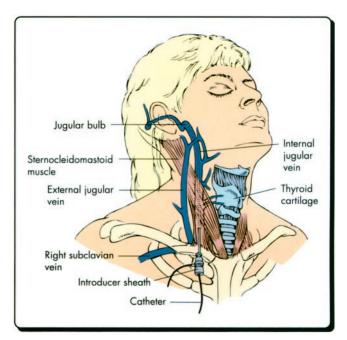


Figure 1. The jugular bulb is the dilated portion of the jugular vein just below the base of the skull that contains blood with little extracerebral contamination. Analysis may be performed by intermittent blood sampling via standard intravascular catheters, continuous oxyhemoglobin saturation monitoring via fiberoptic oximetry catheters, or semicontinuous jugular Po₂ monitoring via fiberoptic probes placed retrograde via the internal jugular vein in the jugular bulb. Reprinted with permission from *Atlas of Anesthesia*, Vol. III.

foramen size (18), or by ultrasonography to compare internal jugular vein size. The assumption of the compression technique is that a greater ICP increase on one side is the result of occlusion of a larger portion of the cerebral outflow, and therefore more reflective of global conditions. Likewise, the assumption with imaging studies is that there is a correlation between the larger jugular foramen or vein and the dominant side (18).

Catheter Placement

The central approach, with a puncture sight similar to that used for central venous catheterization, to the internal jugular vein is often used (4,5,20–22). However, contrary to central venous catheterization, the needle, guidewire, and catheter are advanced in a cephalad direction. Because of concern for vascular injury to the jugular bulb, the Seldinger guidewire should be J-shaped and only advanced 2–3 cm beyond the needle insertion site, at which point the catheter is advanced until resistance is met at the jugular bulb, usually about 15 cm. At this time, an awake patient may note a sensation in the jaw or ear as the catheter abuts the base of the skull, indicating that the tip of the catheter is in the jugular bulb. The catheter is then pulled back 0.5–1.0 cm so that the catheter does not

continue to abut the roof of the jugular bulb and to minimize the cephalad vascular impact with head movement, thereby reducing the risk of vascular injury. Alternatively, an oximetry catheter may be inserted via an introducer to a distance equal to that measured from the point of insertion to the level of the mastoid process (approximately the level of the jugular bulb) or until resistance is met.

Relative contraindications to SjVO₂ monitoring include a cervical spine injury or the presence of a tracheostomy or a coagulopathy. It is important to note that routine maneuvers which aid placement of a catheter inserted via the internal jugular vein, such as Trendelenberg's position or head rotation, may be ill advised in patients with increased ICP or cervical spine injury (21,23). Moreover, the internal jugular vein may be absent, occluded, or in an unusual configuration in approximately 10% of patients (24,25). Therefore, some clinicians advocate using 2-dimensional ultrasound or a percutaneous needle with Doppler probe (21) to facilitate localization of the internal jugular vein with the patient's head and neck maintained in a neutral position. Complications of SiVO₂ monitoring are uncommon and related to catheter insertion. Complications include carotid artery puncture, pneumothorax, nerve injury, infection, and thrombosis. The concern that a jugular catheter might obstruct venous return and increase ICP appears to be unfounded (26,27).

Skull roentgenography can be used to confirm placement (28). On a lateral neck radiograph, the catheter tip should be at the level of, and just medial to, the mastoid process. Radiography may also detect kinks and confirm that the catheter tip is above the lower border of C1, which reduces the chance of extracranial contamination.

Physiology of SjVO₂ Monitoring

Jugular venous oxygen is an indirect assessment of cerebral oxygen use (see Figure 2). Simplistically, when demand exceeds supply, the brain extracts greater oxygen, resulting in decreased jugular bulb oxygen saturation. If cerebral blood flow (CBF) decreases, a point is eventually reached at which the brain can no longer completely compensate for decreased CBF by a further increase in oxygen extraction. At this point, oxygen consumption decreases and anaerobic metabolism with lactate production ensues. When cerebral oxygen supply exceeds demand, oxygen saturation of jugular bulb blood is increased.

Cerebral oxygen delivery (DO₂) is described by the following equation (CaO_2 = arterial oxygen content):

$$DO_2 = CBF \times CaO_2$$

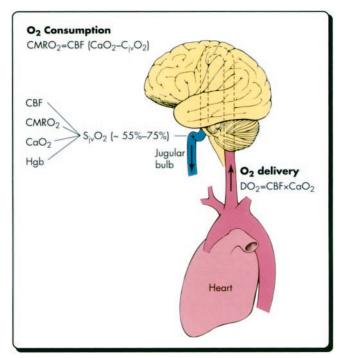


Figure 2. Physiology of jugular venous oxygenation. Clinical measurements of SjVO₂ reflect the balance of oxygen supply and consumption of the brain. Factors affecting SjVO₂ include CBF, cerebral oxygen consumption, arterial oxygen content, and hemoglobin concentration. DO_2 = delivery of oxygen, CBF = cerebral blood flow, CaO_2 = arterial oxygen content, CMRO₂ = cerebral metabolic rate of oxygen, CjVO₂ = jugular venous oxygen content, Hgb = hemoglobin, SjVO₂ = jugular venous oxygen saturation. Reprinted with permission from *Atlas of Anesthesia*, Vol III.

While cerebral oxygen consumption (CMRO₂) is described by the equation ($CjvO_2 = oxygen$ content of jugular venous blood):

$$CMRO_2 = CBF \times (CaO_2 - CjvO_2)$$

The difference in oxygen content between arterial and jugular venous blood is expressed by the term ($CaO_2 - CjvO_2$) or $AjvDO_2$. By rearranging the above equation it is apparent that:

$$AjvDO_2 = CMRO_2/CBF$$

Normally, AjvDO₂ is stable at 4–8 mL O₂/100 mL blood (29,30). If CMRO₂ remains constant, changes in AjvDO₂ should reflect changes in CBF. If AjvDO₂ is <4 mL O₂/100 mL blood, it is assumed that oxygen supply is greater than demand (i.e., luxuriant). An AjvO₂ >8 mL O₂/100 mL blood suggests that demand is in excess of supply (i.e., ischemia).

If CMRO₂ increases without an increase in CBF, the brain extracts more oxygen from the blood, and there is a decrease in oxygen content or saturation of the venous effluent from the brain (widened AjvDO₂). Jugular venous oxygen saturation is normally approximately 55%–75% (1), which is lower than systemic mixed venous oxygen saturation.

If the hemoglobin concentration is stable, the arterial oxygen saturation is approximately 100% and the amount of oxygen dissolved in plasma is physiologic, SjVO₂ is an appropriate correlate of AjvDO₂. As SjVO₂ is a global measure, SjVO₂ monitoring has high specificity but low sensitivity for ischemia, i.e., a normal saturation may not reflect focal areas of ischemia, but a low saturation is indicative of low flow.

Experimental studies suggest thresholds for jugular venous oxygen values and neurologic change which are summarized in Table 1. If the SjVO $_2$ is < 50%, therapy(s) directed at increasing cerebral oxygen supply and/or decreasing demand should be initiated as detailed in Figure 3.

Technical/Methodological Limitations

A limiting factor in the acceptance of $\rm SjVO_2$ monitoring has been a history of relatively poor correlation of concomitant values obtained from cerebral oximetry catheters to an internal jugular venous sample analyzed by a co-oximeter for oxygen saturation (see Table 2). The original oximetry catheters were designed for use in the umbilical arteries of neonates. When used in the venous system with nonpulsatile, retrograde flow and a vessel wall that is susceptible to catheter abutment, there have been limitations in the correlation of the online saturation value to oxygen saturation measured by a cooximeter. These concerns, although still present, have been addressed by recent technologic advances (37–39).

A second limiting factor concerns the possibility of extracerebral contamination. If blood is sampled at a site within 2 cm of the jugular bulb and at a rate of < 2 mL/min there is negligible (approximately 3%) extracerebral contamination (6,40,41). A related concern is the observation that, as CBF decreases, the relative extracerebral contribution to a SjVO₂ reading increases. A final technologic issue is the concern of catheter migration and abutment against the vessel wall. When this occurs, a change in the light intensity signal results with a triggering of the light intensity alarm. Distinguishing a "desaturation reading" as a result of a change in position of the catheter tip from a pathologic desaturation can be problematic. Slight repositioning of the catheter or of the patient's head may be all that is needed to achieve an acceptable light signal. It is recommended that because of the above concerns, cerebral oximetry catheters be routinely calibrated to a cooximeter control.

It should be emphasized that SjVO₂ is a measure of global cerebral oxygenation and is not particularly sensitive to small areas of focal ischemia. Accordingly, the likelihood of a false negative value during an episode of focal ischemia is dependent on the area of ischemia and the capacity of the surrounding "normal" brain tissue to mask or average-out the ischemic tissue. In addition, the

562 MEDICAL INTELLIGENCE ANESTH ANALG 2000;90:559-66

Table 1. Summary of Data Suggesting	a Correlation Between	a Threshold Jugular	Venous Oxygen	Value and Neurologic
Change			, ,	0

Species (Ref.)	Pathologic/Physiologic Insult	Jugular Venous Oxygen Value	Neurologic Change
Cat (31)	Increased intracranial pressure, hypocarbia	Oxygen content of 1.4 ± 0.6 mL/100 mL	Metabolic failure (³¹ P-magnetic resonance spectroscopy)
Human (2)	Nitrogen breathing	$S_{\rm j}VO_2 < 33\%$	Confusion
Human (2)	Nitrogen breathing	$S_1^2VO_2^2 \approx 26\%$	Lost consciousness
Human (32)	Nitrogen breathing	$\dot{SiVO_2} < 40\%$	Electroencephalographic slowing
Human (33)	Carotid endarterectomy	\hat{S} i $VO_2 < 50\%$	Neurologic deficit
Human (34)	Rewarming after hypothermic cardiopulmonary bypass	$\dot{\text{SjVO}}_2^2 < 50\%$	Cerebral anaerobic metabolism
Human (35)	Surgery for evacuation of traumatic intracranial hematomas	$\mathrm{SjVO_2} < 50\%$	Cerebral anaerobic metabolism
Human (36)	Acute, severe, closed-brain trauma	$\mathrm{SjVO_2} < 30\%$ for > 10 min	Decreased Glasgow Coma Scale score

SjVO₂ = jugular venous oxygen saturation.

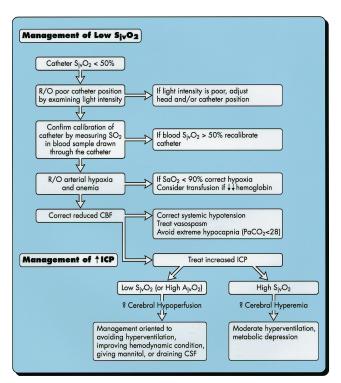


Figure 3. A strategy for management of low jugular venous oxygen saturation. SjVO $_2$ = jugular venous oxygen saturation, R/O = rule out, SaO $_2$ = arterial oxygen saturation, CBF = cerebral blood flow, Paco $_2$ = arterial partial pressure carbon dioxide, AjvDO $_2$ = the difference in oxygen content between simultaneously drawn samples of arterial and jugular venous blood. Modified and reprinted with permission from *Atlas of Anesthesia*, Vol III.

recommendation regarding which jugular vein to cannulate is not clear. As 70% of the cerebral venous blood drains via the ipsilateral jugular veins, some clinicians advocate cannulating at the side of injury. However, in the case of diffuse cerebral injury, most clinicians would monitor the right side, as it is commonly dominant, whereas some clinicians would advocate monitoring the side of dominant flow in all situations (see Which Side

Should be Monitored?). More study is needed to definitively determine if there is an optimal side for SjVO₂ monitoring.

Clinical Factors Altering SjVO₂

Many factors affect the relationship of CMRO₂ and oxygen delivery (see Figure 4). CBF can be decreased by head injury, thromboembolism, intracranial hypertension, hypotension, hyperventilation, or vasospasm. If CMRO₂ remains constant or increases under these conditions, SjVO₂ will decrease. Arterial hypoxia and increased CMRO₂ (e.g., febrile illness, seizures) can also result in SjVO₂ desaturation.

Causes of increased SjVO₂ and luxuriant oxygenation are described in Figure 4. Correct interpretation of increased SjVO₂ requires confirmation that the catheter tip is at the jugular bulb. Reduced CMRO₂ (e.g., hypothermia, sedatives), increased CBF, pathologic arterial-venous communications, and brain death may result in increased SjVO₂.

Clinical Utility of SjVO₂ Monitoring-Overview

Jugular venous oximetry is most often used in patients with head injuries for neurosurgical procedures and for cardiovascular procedures.

Head Injury

In the setting of head injury, $\rm SjVO_2$ monitoring provides an early diagnosis of ischemia resulting from either intracranial or systemic causes (42–49). Moreover, $\rm SjVO_2$ monitoring may be useful to guide decisions for optimizing hyperventilation therapy (43,44,48,50,51), guiding fluid management and oxygenation (43,51,52), optimizing perfusion pressure (53–55), and detecting

Table 2. Limitations of Jugular Venous Oximetry

Limitation	Rationale	Management
Incomplete mixing	Venous sample may not be representative of the entire brain if asymmetric venous drainage.	Cannulate dominant internal jugular vein (usually right), or consider placing on side of the most severe focal injury.
Extracerebral contamination	≈3% of jugular blood is contaminated by blood from scalp, meninges, and skull.	Radiograph confirmation. Location of catheter tip above lower border of C1 and withdraw sample slowly (<2 mL/min).
Bohr effect	Falsely high SjVO ₂ values may occur from a leftward shift of the oxyhemoglobin dissociation curve during alkaline conditions.	Detect by measuring low jugular bulb Po ₂ (<27 mm Hg)
Global measure	With focal cerebral injuries, SjVO ₂ may not provide information about regional injury.	Measurement of arteriovenous lactate may be helpful as an indicator of anaerobic metabolism.
Insensitive to infratentorial flow	Brain stem and cerebellum contribute little to the venous outflow from the brain.	Is of limited value for monitoring patients with brainstem injuries.
Monitoring errors	Catheter may be against wall of vein, coiled back on itself, or have fibrin formation at tip.	May need catheter repositioning, recalibration of continuous fiberoptic catheter, or heparinized saline flush (≈3 mL/h).

 $SjVO_2$ = jugular venous oxygen saturation. Modified and reprinted with permission from Atlas of Anesthesia, Vol III.

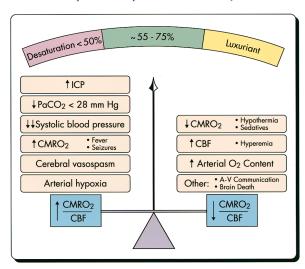


Figure 4. Jugular venous oxygen saturation (%). Many factors alter the relationship between cerebral oxygen consumption (CMRO₂) and supply. Simplistically, when cerebral oxygen demand exceeds supply, the brain extracts oxygen from hemoglobin, resulting in a decreased oxygen saturation of the blood in the jugular bulb. Although somewhat arbitrary, a jugular venous oxygen saturation <50% is considered low, and therapy directed at improving cerebral oxygen supply or decreasing demand should be instituted. ICP = intracranial pressure, Paco₂ = arterial partial pressure carbon dioxide, CMRO₂ = cerebral metabolic rate of oxygen, CBF = cerebral blood flow, O₂ = oxygen. Modified and reprinted with permission from *Atlas of Anesthesia*, Vol III.

arterial-venous fistulas (56,57). Used with a transcranial Doppler monitor, $SjVO_2$ can help discern between hyperemia and vasospasm. With high flow velocity detected by transcranial Doppler, $SjVO_2$ is increased during hyperemia and normal or low if cerebral vasospasm is present.

Barbiturate-induced cerebral metabolic suppression and induced hyperventilation are examples of two therapies for head injury that may be guided by SjVO₂ monitoring. Cruz (58) identified a group of headinjured patients who responded to pentobarbital with a decrease in SjVO₂. It was hypothesized that the vasoconstrictive effect of barbiturates resulted in increased cerebrovascular resistance and oligemic cerebral hypoxia in these patients.

Routine hyperventilation after traumatic brain injury is not currently recommended (45). Rather, contemporary guidelines recommend "optimal hyperventilation" guided by SjVO₂ monitoring, thus identifying those head-injured patients with the potential for an ischemic response to hypocarbia (45). Moreover, SjVO₂ monitoring is also useful in evaluating prognosis for head-injured patients (59). A strategy for management of low SjVO₂ occurring after head injury is detailed in Figure 3.

Cardiovascular and Neurologic Surgery

Neurologic dysfunction is not uncommon after cardiac surgery with cardiopulmonary bypass and is attributed to the adverse effects of nonphysiologic modes of perfusion (60). A particularly critical period concerns rewarming after hypothermic cardiopulmonary bypass. Rewarming has been related to frequent SjVO₂ desaturations which are associated with increased postoperative neurocognitive deficits (61–63). It has been suggested that SjVO₂ monitoring may have

564 MEDICAL INTELLIGENCE ANESTH ANALG 2000;90:559-66

a role in cerebral monitoring during adult (64) and pediatric (65) cardiac surgery.

The potential applications of SjVO₂ monitoring during neurosurgery have been studied by Matta et al. (66). They demonstrated that the SjVO₂ catheter could be placed quickly and detect frequent critical episodes of SjVO₂ desaturation that would otherwise have been untreated. During intracranial aneurysm surgery, SjVO₂ monitoring has been used to determine the minimal blood pressure that should be maintained to avoid hypoperfusion (67).

Systemic versus Jugular versus Cerebral Oxygen Saturation

The possibility of using systemic mixed venous oxygenation as a surrogate of SjVO₂ to assess cerebral oxygenation has been studied. Given that the brain receives approximately 15% of the total cardiac output and that it extracts a greater fraction of oxygen from arterial blood than do other organs, it does not seem logical that systemic mixed venous oxygenation would be a reliable marker of cerebral oxygenation. Indeed, the lack of an association between systemic mixed venous oxygen and SjVO₂ has been validated with limited studies (34,68–70).

A concept with some promise is the potential for near-infrared spectroscopy (NIRS), a continuous, non-invasive monitor of brain oxygenation at the tissue level, to correlate with SjVO₂ as a measure of cerebral oxygen use. Presently, NIRS methods in adults are suspect primarily because of the inadequate predictability of extracerebral tissue to affect the NIRS signal, changes in arteriovenous partitioning, algorithm limitations, and variations in optical path length. It is likely that future advances in NIRS technology will result in a better understanding of these effects, with application of real-time correction factors. At this time, the data do not support a correlation between SjVO₂ and NIRS when assessing cerebral oxygen saturation in adults (71,72).

Despite the limitations of $\mathrm{SjVO_2}$ monitoring, there is no better, commercially available, continuous, relatively low-cost, bedside monitor to assess the adequacy of cerebral oxygenation. Jugular venous oxygenation provides information on global brain oxygenation and is recommended in the treatment of patients with head injury, especially those receiving hyperventilation therapy.

Jugular venous oxygen monitoring is often performed in conjunction with other monitoring and imaging techniques and provides early detection of cerebral ischemia that might otherwise go unrecognized. However, the question should be asked: Do clinical decisions impacted by $SjVO_2$ data effect patient outcome? Although there appears to be a correlation between episodes of $SjVO_2$ desaturation and poor neurologic outcome, the impact of decisions influenced by $SjVO_2$ data and their effect on long-term cerebral function has yet to be proven.

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MEDICAL INTELLIGENCE

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