Jugular venous oximetry

Avanish Bhardwaj, Hemant Bhagat, Vinod K. Grover

Abstract

The measurement of saturation of venous blood as it drains out of brain by sampling it from the jugular bulb provides us with an estimate of cerebral oxygenation, cerebral blood flow and cerebral metabolic requirement. Arterio-jugular venous difference of the oxygen content (AVDO$_2$) and jugular venous oxygen saturation (SjVO$_2$) values per se helps clinicians in identifying the impairment of cerebral oxygenation due to various factors thereby prompting implementation of corrective measures and the prevention of secondary injury to the brain due to ischaemia. SjVO$_2$ values are also used for prognostication of patients after traumatic brain injury and in other clinical situations. Sampling and measuring SjVO$_2$ intermittently or continuously using fibreoptic oximetry requires the tip of the catheter to be placed in the jugular bulb, which is a relatively simple bedside procedure. In the review below we have discussed the relevant anatomy, physiology, techniques, clinical applications and pitfalls of performing jugular venous oximetry as a tool for measurement of cerebral oxygenation.

Key words: Cerebral oxygenation, jugular bulb, jugular venous oximetry

INTRODUCTION

Minimising secondary injury to the brain after a primary insult forms the backbone of intervention in neuroanaesthesia and neurointensive care. However, in order to achieve this it is of absolute importance to identify the insult as promptly as possible so that appropriate intervention can be instituted. The salvageable portion of the brain, that is, the penumbra requires a constant supply of oxygen so that it does not undergo irreversible brain damage. Jugular bulb oximetry is one such monitor, which provides information about the status of cerebral oxygenation and helps in guiding the therapy to prevent secondary injury due to various factors.

Department of Anaesthesia and Intensive Care, PGIMER, Chandigarh, India

Address for correspondence:
Dr. Hemant Bhagat, Department of Anaesthesia and Intensive Care, PGIMER, Chandigarh, India.
E-mail: hembhagat@rediffmail.com

Percutaneous sampling and analysis of human cerebral venous blood from the jugular bulb was first described by Myerson et al. in 1927.[1] Subsequently Gibbs et al. in 1942 studied the arteriovenous difference between oxygen, glucose and lactate content and proposed them as measures of the balance between oxygen supply and demand.[2] It is more than six decades now that the measurement of oxygenation of the jugular venous blood is being used to monitor the cerebral physiology.[3] As of now, jugular venous oxygen saturation (SjVO$_2$) provides us information about the balance between cerebral oxygen supply and demand, and is used to guide physiologically based management of various clinical situations including traumatic brain injury, during neurosurgery and cardiopulmonary bypass.[4-6] Oxygen that is not utilised by the brain is carried into the systemic circulation via internal jugular veins (IJVs), and hence, the measurement of SjVO$_2$ helps to determine the balance between cerebral blood flow (CBF) and cerebral metabolic requirement of oxygen (CMRO$_2$).

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Bhardwaj A, Bhagat H, Grover VK. Jugular venous oximetry. J Neuroanaesthesiol Crit Care 2015;2:225-31.
**CLINICAL ANATOMY**

Blood from the brain drains mainly through the venous sinuses. Almost whole of the blood comes into the right and left sigmoid sinus, which continue downwards as respective IJVs after passing through the jugular foramen. The jugular bulb is the dilatation of the jugular vein as it exits from the base of the skull. The tip of the catheter is placed in the jugular bulb for withdrawal of a sample or continuous monitoring of SjVO₂ [Figure 1]. The jugular bulb contains the blood, which is being drained out from both sides of the brain, out of which around 70% is from the same hemisphere and 30% is from the opposite hemisphere.[7,8] Most of the patients will have a dominant side of the venous drainage and in the majority of them it will usually be right.[9,10] The side with major venous drainage, as seen on the cerebral angiogram, gives an idea about the side of dominance.

The comparison of jugular foramen size using computed tomography scan can also be used to predict the side of the predominant drainage, with the larger size of foramen corresponding to the side of the major drainage.[11] Ultrasonography can also be used to compare the size of the veins with the vein having larger diameter being the one with dominant drainage. The patients in whom the intracranial pressure (ICP) is being monitored, the dominant vein can be determined by the one with a higher rise in ICP following manual compression of each side. It is assumed that compressing the side with more drainage will cause a greater increase in ICP as a larger portion of cerebral outflow will be occluded.[12]

**MONITORING SITE**

Patients sustaining an injury to both the hemispheres of the brain should have a catheter placed in the jugular bulb on the side of major drainage, which in most of the cases is generally right.[10] However, if the patient has a focal injury, then there are no clear cut guidelines regarding the side on which the catheter should be placed. As more than two-third of the blood from the brain is drained through the ipsilateral IJV, it may be reasonable to cannulate the IJV on the side of the lesion, even if it is not the side with dominant drainage.[5]

**TECHNIQUES OF MEASUREMENT**

A catheter that is used to monitor the SjVO₂ is almost same as the one, which is used to monitor the central venous pressure. The catheter is inserted in the IJV in a retrograde direction and the tip is advanced up to the jugular bulb. Intermittent samples may be drawn and SjVO₂ measured.[13] It is important to note that the rate of aspiration of blood for measurement should be <2 ml/min to avoid contamination from extracranial vessels. Fibre optic technology has allowed the development of in vivo catheters wherein reflectance oximetry using such catheters allows continuous SjVO₂ monitoring, thus obviating the need for repeated sampling and measurement.[14,15] Oxyhaemoglobin has a unique light absorption spectrum and this forms the basis of fibreoptic oximetry. Catheters that are used to measure SjVO₂ contains two optical fibres, one of which is used to direct the light into the blood and the other one is used to transmit the reflected light to a sensor. The sensor measures the absorbed light which is reflected at different wavelengths. The SjVO₂ values are therefore a percentage of oxygenated haemoglobin to the total haemoglobin. These catheters using a dual wavelength of light (Edslab, Baxter-Edwards system) require that the haemoglobin concentration of the patient to be manually entered. The SjVO₂ values are thus dependent on the values entered. Alternatively, the catheters which use three wavelengths (Opticath Oximetrix, Abbott Critical Care System) calculate the haemoglobin concentration from the absorption spectrum itself and hence can be used for a real time, continuous monitoring of SjVO₂.

**CATHETER INSERTION**

Puncture site for insertion is similar to that used for IJV cannulation. However, the needle followed by guidewire and subsequently the catheter are all advanced in a direction towards the skull [Figure 1]. To prevent injury, it is important that the tip of the guidewire is J-shaped and is advanced only 3–4 cm beyond the site of needle insertion. The catheter is advanced over the guide wire in a cephalad direction till there is a feeling of resistance at the jugular bulb. The catheter is subsequently withdrawn for about 0.5–1 cm so that that the tip does not lodge against the roof of the jugular bulb. This also reduces the risk of injury.

Figure 1: Jugular bulb anatomy
to the bulb and prevents occlusion of the catheter tip. Markings on the guidewire may also be used to help us in determining the distance to which the catheter should be inserted [Figure 2]. It should be 1 cm lesser than the length as measured from the point at which the catheter enters the neck up to the mastoid process. Skull and neck radiography can be used to confirm the correct placement of the catheter.[16] On a lateral radiograph of the neck the tip of the catheter should be at the level of mastoid process and just medial to it and also above the lower border of C1 vertebrae, as the aspiration of blood from this position will reduce the chance of contamination from extracranial vessels [Figure 3]. Alternatively fluoroscopy can also be used intraoperatively for quick confirmation of the position of the catheter tip.

**PRINCIPLE OF JUGULAR VENOUS OXIMETRY**

Jugular venous saturation indirectly gives us an idea of the use of oxygen by the brain. To put it in simpler terms when the demand for oxygen is more, the brain extracts a greater amount of oxygen, resulting in decreased jugular bulb oxygen saturation. When cerebral oxygen supply exceeds demand, the extraction is less and saturation of oxygen in the venous blood increases. If the CBF reduces too much, then a point is reached when the neurons of the brain cannot tolerate the reduction of blood flow. After this stage is reached, the consumption of oxygen reduces leading to anaerobic metabolism and production of lactate. The oxygen saturation in jugular bulb is related to the CMRO$_2$ and CBF as represented by the Ficks equation:

$$\text{CMRO}_2 = \text{CBF} \times (\text{CaO}_2 - \text{CjO}_2)$$

$$\text{CaO}_2 \ (\text{Arterial oxygen content}) = \text{SaO}_2 \times 1.34 \times \text{Hb} + 0.0031 \times \text{PaO}_2$$

$$\text{CjO}_2 \ (\text{Jugular venous oxygen content}) = \text{SjO}_2 \times 1.34 \times \text{Hb} + 0.0031 \times \text{PjO}_2$$

The contribution from dissolved oxygen, as we know, is very small and can safely be neglected. The difference between arterial oxygen content and oxygen content of venous blood is represented as (CaO$_2$ - CjO$_2$) or AVDO$_2$ (arterio-venous oxygen difference). If the above equation is rearranged then:

$$\text{AVDO}_2 = \text{CMRO}_2 / \text{CBF}$$

Normal AVDO$_2$ values are between 4 ml/dl and 8 ml/dl.[17,18] Presuming that if CMRO$_2$ does not change then any change in AVDO$_2$ should be because of the change in CBF. If AVDO$_2$ is <4 ml/dl of blood, it is probably because the oxygen supply is in excess of the demand (i.e. luxury perfusion or infarction). An AVDO$_2$ value more than 8 ml/dl of blood points towards the demand being more than the supply (i.e., ischaemia). If CMRO$_2$ increases but the CBF does not increase concomitantly, then there will be enhanced extraction of oxygen from the blood. There is a proportional decrease in oxygen content, as well as oxygen saturation, of the venous blood coming out of the brain with eventual widening of AVDO$_2$. In a healthy brain flow-metabolism coupling is preserved and SjVO$_2$ ranges between 55% and 75%.[2] However in pathological conditions, the coupling between CMRO$_2$ and CBF is lost and in circumstances where hypoperfusion is not accompanied by proportional reduction in CMRO$_2$, the brain will extract a greater proportion of arterial oxygen and SjVO$_2$ will be observed to fall. Values below 54% imply that oxygen supply may be critically low for the metabolic demand and the brain is at risk for ischaemic injury.[19] Value more than 75% is representative of cerebral hyperaemia or decreased oxygen extraction as in cerebral infarction.
DETERMINANTS OF JUGULAR VENOUS OXYGEN SATURATION

A reduction in $S_{jVO_2}$ may be due to fall in supply of oxygen or an increase in usage of oxygen by the brain whereas an increase in $S_{jVO_2}$ values may be seen due to increase in oxygen delivery or a reduction in oxygen consumption:

Decreased jugular venous oxygen saturation (<50%)
- Due to reduced supply of oxygen: Cerebral vasoconstriction, hypocapnia, vasospasm, hypotension, anaemia and sepsis
- Due to increased oxygen requirement: Increased cerebral metabolism, which include causes such as fever, agitation, inadequate sedation, pain and seizures.

Increased jugular venous oxygen saturation (>75%)
- Increased supply of oxygen: Cerebral vasodilation, hypercapnia, hypertension
- Reduced oxygen requirement: Deep sedation, coma, hypothermia, cerebral infarction, brain death.

CLINICAL APPLICATIONS

Traumatic brain injury
In patients who have sustained a traumatic brain injury, $S_{jVO_2}$ monitoring provides a means to identify ischaemia, which may be due to either systemic or intracranial causes.[20,21] The goal is to maintain a $S_{jVO_2}$ value above 50% as we know that the values lesser than this suggest a relative failure of oxygen supply compared with demand and episodes of such desaturation have demonstrated to predict poor outcome after head injury.[10] Whenever hyperventilation is to be instituted, $S_{jVO_2}$ monitoring is a valuable aid in guiding its optimal usage without causing cerebral hypoperfusion.[22‑25] As per the brain trauma foundation guidelines for the management of traumatic brain injury, prophylactic hyperventilation (<25 mm Hg PaCO$_2$) is not recommended, more so in the first 24 h after injury. If other measures to reduce ICP have failed, then hyperventilation is to be used only as a temporary measure. It is vital to monitor $S_{jVO_2}$ or brain tissue oxygen tension (PBrO$_2$) while hyperventilating this group of patients.[26] $S_{jVO_2}$ monitoring can also be used to guide administration of fluids and level of oxygenation[22,25] and optimise the cerebral perfusion pressure.[21,27,28] A scheme for management of low $S_{jVO_2}$ (<50%) in neurosurgical patients is outlined in Figure 4. Though low $S_{jVO_2}$ predicts poor outcome but it is also important to emphasize that even $S_{jVO_2}$ values >85% may be associated with a poor outcome as high values suggest a state of hyperaemia or failing oxygen utilisation either due to a state of cellular dysfunction and death or due to shunting of arterial blood.[29] The current clinical use of jugular venous oximetry in patients with traumatic brain injury is limited to prognosticate the outcome. There is no evidence to suggest that clinical management based on jugular venous oximetry values have influenced the outcome of the patient.

Lactate oxygen index (LOI) can also be used to predict outcome in traumatic brain injury patients.[10] Its normal values are <0.03 and values >0.08 are seen in patients with failing oxygen extraction, leading to failure of aerobic metabolism and increased production of lactic acid in the brain especially in the first 24 h after head injury.[30] It is calculated by the formula mentioned below:

$$LOI = \left( -AVDL \right) \times 2.24 / AVDO_2$$

$AVDL = $Arterial to jugular difference for lactate

$AVDO_2 = $Arterial to jugular difference in oxygen content.

Neurosurgery
$S_{jVO_2}$ monitoring has been extensively used by Matta et al., in neurosurgical patients.[5] They demonstrated that the $S_{jVO_2}$ catheter could be placed quickly and can detect frequent critical episodes of $S_{jVO_2}$ desaturation that would otherwise have been undetected and untreated. During intracranial aneurysm surgery, $S_{jVO_2}$ monitoring has been used to determine the minimal blood pressure that should be maintained to avoid hypoperfusion and also to measure LOI. Intraoperative LOI >0.08 during surgery for aneurysm clipping has been associated with a poor outcome.[31] $S_{jVO_2}$ monitoring can also be used in any neurosurgical procedure to help in deciding the appropriate mean arterial pressure and PaCO$_2$ values to ensure adequate cerebral oxygenation. Our own experience suggests that measurement of $S_{jVO_2}$...
in patients undergoing clipping of ruptured cerebral aneurysm is feasible and can help in guiding the perioperative anaesthetic management.[32]

**Cardiac surgery**

Neurologic dysfunction is seen quite commonly after cardiac surgery especially in those patients who undergo cardiopulmonary bypass.[33] Most of the episodes of SjVO₂ desaturation are seen during the period of rewarming after hypothermic cardiopulmonary bypass. SjVO₂ desaturation can result in increased incidence of post-operative cognitive dysfunction.[34-36] It would be prudent to monitor SjVO₂ during cardiac surgery to prevent these episodes of desaturation and improve the cognitive outcome.[37]

**Cardiac arrest**

SjVO₂ values are higher in the non-survivors than the survivors of cardiac arrest (values of 80% vs. 67%) probably due to the inability of dead neurons to utilise the supplied oxygen.[38] Moreover, SjVO₂ values more than the mixed venous oxygen saturation are also associated with poor outcome.[39] However, these findings are still controversial and have been countered by studies, which did not find any difference in the SjVO₂ values in between survivors and non-survivors of cardiac arrest.[40]

**Research applications**

Jugular venous oximetry can be used to guide research to evaluate the effect of various anaesthetic agents on CBF. In a recently concluded study comparing propofol and desflurane in patients undergoing clipping of aneurysmal neck after subarachnoid haemorrhage, it was observed that use of desflurane was associated with increase in SjVO₂ values indicating hyperaemia whereas SjVO₂ values were maintained within physiological range with the use of propofol indicating better flow-metabolism coupling.[41]

**DRAWBACKS AND LIMITATIONS**

- It is an invasive procedure requiring the insertion of the catheter retrograde into the jugular vein. Cannulating the vein may fail at times due to anatomical variations and abnormalities. Carotid puncture and subsequent haematoma formation can also occur
- Malpositioning of the catheter tip can occur which may give erroneous readings if the position of the tip is not confirmed prior to the measurement of SjVO₂ values
- Mechanical complications of injury to the vessel wall or jugular bulb may occur. Infectious complications have also been reported though the incidence of such complications is very less[41]
- Subclinical jugular vein thrombosis may occur if catheter is kept in situ for more than 6 days but the presence of such catheters has not shown to increase the ICP any further[42]
- No consensus exists as to which side jugular bulb should be cannulated. As 70% of the cerebral venous blood drains via the ipsilateral jugular veins, some clinicians advocate cannulating 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
- The catheter may impinge against the wall of the jugular bulb and may give false low readings in case of a continuous monitoring catheter or make aspiration of blood difficult for measurement of SjVO₂ values for intermittent monitoring
- The catheter may also get kinked or there may be deposition of fibrin at the tip which may affect the quality of the signal. However, the current generation of catheters are stiff and less prone to kinking. They may also have an antithrombotic coating to prevent fibrin deposition. However, continuous monitoring catheters should be frequently calibrated against a co-oximeter control before instituting any therapy based on SjVO₂ values
- If the rate of aspiration is >2 ml/min, then considerable contamination can be there from the extracranial vessels. Hence, it is important that for the catheters used for intermittent sampling the rate of aspiration to withdraw the sample should be <2 ml/min[43]
- SjVO₂ gives an idea of global or at best hemispheric oxygenation and may have poor sensitivity for focal ischaemia, which may not be reliably detected. As a matter of fact, regions of infarcted tissue which do not utilise any oxygen due to failure of cellular metabolism and function like physiological shunts may mask the effect of surrounding ischaemic areas leading to falsely reassuring values of SjVO₂
- It is of limited value in monitoring patients with infratentorial injuries or lesions as the brain stem and cerebellum contributes very little to the venous outflow from the brain
- Insertion of the catheter may be difficult in patients with associated cervical spine injuries, trauma to the neck, presence of a tracheostomy tube and is contraindicated in patients with coagulopathy.

**CONCLUSIONS**

Jugular venous oximetry is the oldest technology for the measurement of cerebral oxygenation and hence becomes a natural benchmark against which newer modalities of monitoring are evaluated. Despite its limitations, it is
a relatively low-cost monitor and the fact that it can be inserted at the bedside to assess the adequacy of cerebral oxygenation makes it quite attractive. Its role becomes even more important when hyperventilation or blood pressure directed management has to be instituted for patients with traumatic brain injury and other intracranial pathologies. As a part of multimodality monitoring which includes ICP monitoring, monitoring of CBF by transcranial Doppler and assessing the adequacy of cerebral oxygen delivery at the target area by brain tissue oxygen measurement (PBrO2) and cerebral microdialysis catheters, SjVO2 monitor functions as a tool for global cerebral oxygenation and helps to guide various goal-directed therapies in the management of patients with various intracranial pathologies. It subserves as an important tool for research related to various interventions, which can influence CBF and cerebral oxygenation. Hence in the absence of other viable alternatives to measure the adequacy of cerebral oxygenation, Jugular venous oximetry will continue to thrive as a useful monitoring modality for this purpose.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**REFERENCES**