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REVIEW

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# Cerebrovenous oxygen saturation monitoring: practical considerations and clinical relevance

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**Abstract** Measurement of the saturation of brain effluent blood gives a global estimate of cerebral oxygenation. It may provide clinicians with information to assist in reducing secondary insults to the brain with potential benefits to a range of patients with actual or potential acute brain injury such as trauma and cardiac bypass procedures. The technology to continuously measure this variable is simple to use but requires attention to detail; it is limited in its ability to detect discrete regions of ischaemia or hyperaemia unless these are of sufficient magnitude to influence the saturation of brain effluent blood. There are few complications that result from this invasive technique and they are usually of a minor nature. The technique also enables research opportunities from the ability to sample blood as it leaves the cranium. Poor outcomes are seen in patients with traumatic brain injury who exhibit either reduced or increased cerebrovenous oxygen saturation and it remains to be seen if detection and correction of these anomalies will produce patient benefits.

**Key words** Cerebrovenous oxyhaemoglobin saturation · Monitoring · Cerebrovascular circulation

## Introduction

Intensive care patients with acute brain injury suffer further brain damage (secondary injury) from physiological insults such as hypotension, hypoxia and raised intracranial pressure, and from destructive inflammatory processes initiated as a consequence. Improved long-term outcome is the aim of a multidisciplinary approach including emergency care teams, neurosurgeons, intensive care physicians, physiotherapists, speech therapists, and psychologists. Minimising secondary insults is the most important aim for intensive care doctors. However, in order to do this, the insults must first be detected. Older technologies permitted continuous monitoring of systemic variables, including blood pressure and oxygen saturation, which were then applied to brain physiology. Newer techniques and equipment allow the brain to be directly monitored (including intracranial pressure and cerebrovenous oxygen saturation) with almost instant recognition of physiological challenge, thus allowing prompt corrective measures, as necessary. Monitoring for, and reducing the burden of, secondary insults – including the use of cerebrovenous (jugular bulb) oxygen saturation – may allow interventions that will reduce adverse long-term consequences of secondary brain injury.

This review will discuss the theory behind jugular bulb monitoring, the practicalities of the technique and equipment, and its clinical application. The discussion will also examine other uses for the technique, such as during cardiac surgery and some research possibilities.

## Anatomy

The internal jugular veins drain virtually all of the blood from the brain. Blood courses from the brain into sinuses (superior sagittal sinus, inferior sagittal sinus, straight sinus, right and left transverse sinus, and occipital sinus) which terminate in the right and left sigmoid sinuses. These two sinuses pass through their respective jugular foramina at the base of the skull and dilate to form the jugular bulb – the dilated cephalad part of the internal jugular vein. A small proportion of cerebral effluent blood may drain via the vertebral venous plexus which is most pronounced when in the erect position [1].

## Physiology

Saturation of blood in the jugular bulb is related to cerebral blood flow (CBF), arterial blood saturation, and brain oxygen extraction – the cerebral metabolic rate for oxygen ($CMRO_2$). The Fick equation relates these variables:

$CMRO_2 = CBF \times$ (arterio-venous $O_2$ difference)

The arterio-venous oxygen difference is the difference in content of oxygen ($CaO_2$) where:

$CaO_2 = (\%$ saturation $\times 1.34 \times Hb) +$ (arterial or venous $O_2$ tension $\times 0.003$)

Since the haemoglobin (Hb) level is practically constant between arterial and venous circulations, and dissolved oxygen is negligible, it can be seen that $CaO_2$ varies with saturation. In practical terms if the arterial saturation is constant:

$SjO_2 \alpha CBF/CMRO_2$

Thus $SjO_2$ is a function of arterial saturation, $CMRO_2$, and CBF. There is debate as to what the normal range of $SjO_2$ is, but most authorities assume 50–54 % to be the lower limit of normal and 75 % the upper limit. Different methods for defining normality has caused controversy as to the exact lower limit of normal $SjO_2$ with some authors assuming 50 % [2, 3] and others 54 % [4, 5, 6, 7]. In fact, neither are wrong as shown in a recent paper comparing $SjO_2$ monitoring with brain tissue oxygenation ($PtiO_2$) [8] where $PtiO_2$ values of 8.5–11.0 mmHg (albeit with a large variability) corresponded with $SjO_2$ values of 50–54 %.

## Cannulation

Which side?

The choice of which jugular bulb to cannulate can potentially influence results. It cannot be assumed that the jugular bulb contains exclusively cerebral effluent blood as it may be contaminated by extracranial drainage. Early experimentation by Shenkin et al. [9] on eight patients without cerebrovascular lesions, in whom dye was injected into the *external* carotid artery, demonstrated that 0–6.6 % (mean 2.7 %) of blood in the jugular bulb was derived from extracranial sources. This is due to the emissary and frontal veins draining into the sagittal sinus, and the cavernous sinus connection to the sigmoid sinus and the jugular bulb through the petrosal sinuses [10]. The largest source of potential contamination is the facial vein that joins the internal jugular vein a few centimetres below the jugular bulb. Recent experiments show that the rate of withdrawal of blood from the jugular bulb affects the composition of the sample; a rate of 1–2 ml per min is optimum [10].

Somewhat surprisingly, the composition of the two jugular bulbs is not equal. Cortical tissue drains into the sagittal sinus while subcortical tissue drains into the straight sinus. These join to form the torcular Herophili that divides into the two lateral or transverse sinuses which ultimately drain into the jugular bulb via the sigmoid sinuses. Mixing of blood from the cortex and deeper regions of the brain is incomplete; the lateral sinus is larger on the right in 62 %, the left in 26 %, and equal in 12 % of subjects [11]. In an autopsy study, Gibbs [12] found that blood from subcortical areas draining via the straight sinus tended to flow into the left lateral sinus, while blood from cortical tissue (draining via the sagittal sinus) flowed mostly into the right lateral sinus. Shenkin et al. [9] suggested that two-thirds of the content of the internal jugular vein is from the ipsilateral hemisphere and one-third from the contralateral hemisphere. Simultaneous sampling of both right and left jugular bulbs in normal individuals has shown that oxygen saturation at the level of the internal jugular vein is equal on both sides [13]. More recently, in a study of 32 head-injured patients, Stocchetti [14] cannulated both jugular bulbs and took 171 paired blood samples. The differences in saturation of the paired samples was $5.32 \pm 5.15$ % (mean ± SD). It is probably the case that in normal individuals there is no difference in saturation between the right and left sides, but there are differences in head-injured patients. It is good clinical practice to test intracranial compliance and identify the dominant side by compressing each jugular vein in turn and observing the rise in intracranial pressure [15, 16]. The side with the greatest increase in ICP drains most blood and therefore allows $SjO_2$ monitoring of the largest vascular territory of brain. If there is no difference between

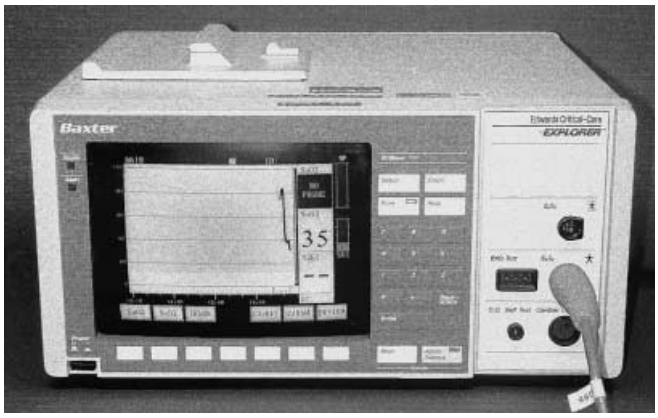


**Fig. 1** Edslab oximeter

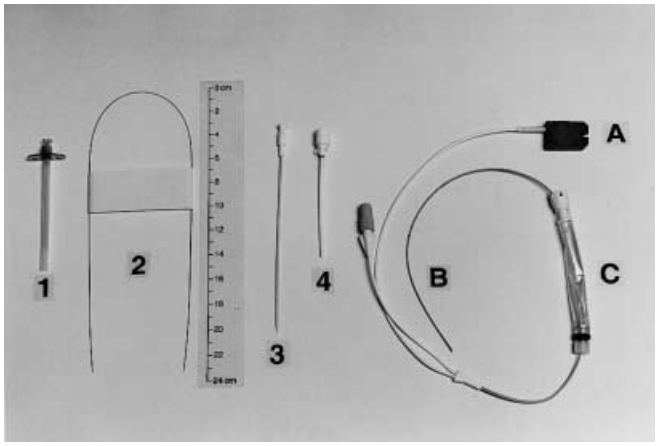


**Fig. 2** 4-French gauge reflection spectrophotometric catheter and insertion equipment. *1*: Cook 18 F gauge 7 cm needle; *2–4*: Daig Fast-Cath 7-F gauge haemostasis introducer kit (*2* guide wire, *3* dilator, *4* introducer). *A* Catheter connection to optics module *B* catheter tip (patient end) with graduated markings, *C* contamination sheath

the two sides, the right side is commonly used in diffuse injury as it is more likely to be the dominant anatomical side [17]. Metz et al., however, recommended CT assessment for the larger jugular foramen in diffuse injury [18].

## Cannulation technique

Retrograde cannulation of the internal jugular vein is a simple and safe procedure in experienced hands [19]. It may be cannulated distally where it courses close to the skin between the two heads of the sternocleidomastoid muscle [20, 21], or more proximally at the level of the cricoid ring [15]. The patient is positioned head down (without allowing ICP to exceed 20 mmHg), or as close

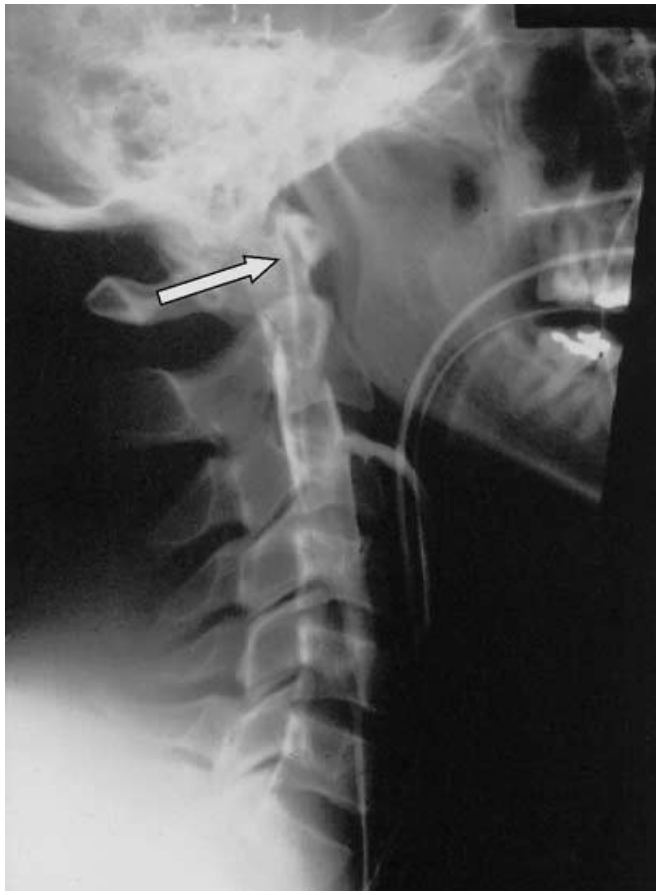

**Fig. 3** Lateral neck X-ray demonstrating catheter tip position above the disc of $C_1/C_2$

to horizontal as possible. The appropriate landmarks are identified and, using a Seldinger technique, the vein is cannulated and the fibreoptic catheter advanced into the bulb (at about 12–15 cm) (Figs. 1, 2, 3). Segal has described cannulation in the head up position with the aid of a needle with an in situ Doppler probe [22]. The catheter tip must be above the disc of $C_1/C_2$ to minimise facial vein contamination, thus position of the catheter tip must be ascertained by X-ray to ensure accurate measurement and reduce complications. An overpenetrated lateral radiograph is the simplest and most reliable type of X-ray [23]; alternatively, anteroposterior radiographs are preferred by some clinicians [24].

## Complications

As with any invasive procedure iatrogenic injury may occur, the incidence of which can be minimised by following standard techniques. Complications may arise as a consequence of catheter insertion or be associated

with the catheter remaining in the vein for long periods. These include carotid artery puncture (incidence 1–4.5 % [25, 26]), haematoma formation, thrombosis [26, 27] and, rarely, raised ICP. In a study of paediatric patients, Goetting measured intracranial pressure in 28 patients with jugular bulb catheters and found that neither cannulation nor the presence of catheters in situ raised the pressure further [28]. In an observational study of 44 patients with jugular bulb catheters, Coplin et al. [26] concluded that complications related to catheter insertion were rare and clinically insignificant, and that the risk of bacteraemia related to the catheter was negligible. However, on ultrasound, the incidence of *subclinical* internal jugular vein thrombosis after jugular bulb catheter monitoring was as high as 40 % with catheters in situ for up to 6 days. Patients with a proven thrombus did not develop any symptoms. On a balance of risks, jugular bulb monitoring is safe and can provide useful information for the clinician.

## Equipment

The early pioneers of jugular bulb monitoring used intermittent sampling with offline blood gas analysis. Fibreoptic technology has allowed the development of in vivo reflection spectrophotometric catheters. Two systems are in common use – the Oximetrix 3 System (Abbott Laboratories, North Chicago, Ill., USA) and the Edslab system (Baxter Healthcare, Irvine, Calif., USA) (Figs. 1, 2). The Oximetrix 3 system uses three light wavelengths in the red/infrared spectrum, allowing the measurement of haemoglobin concentration and calculation of saturation. The Edslab uses two light wavelengths, hence the haemoglobin value must be entered manually. In clinical practice Souter and Andrews found the Edslab to be reliable if calibrated every 24 h [29], compared to 12 hourly with the Oximetrix 3 system [15]. Gunn et al. [30] found the Edslab to be comparable to blood gas analysis during neurosurgical procedures lasting an average of 7 h. Others have found both the Edslab [31] and Oximetrix 3 [6] inaccurate after the first calibration, but with satisfactory results thereafter. In a volunteer study [32] the Oximetrix fared reasonably well compared to blood gas analysis with a mean difference ($d$) in paired samples of 0.54 % with limits of agreement ranging from –9.5 to 8.4 %. A clinical study of the Edslab revealed a higher degree of accuracy ($d = 0.28$ %) with limits of agreement ranging from –4.88 to 4.32 % [29]. This accuracy was verified recently in a clinical study of patients with traumatic brain injury [33]. Another catheter system, the Paratrend 7 (Biomedical Sensors, High Wycombe, Bucks, UK), which was designed for intra-arterial use, may also be placed in the jugular bulb. It incorporates a miniaturised Clark electrode for measuring $P_{O_2}$, two optical fibres for measuring $P_{CO_2}$ and pH, and a thermistor for temperature [34].

A criticism of the jugular bulb saturation method for detecting brain ischaemia is that it is a global measure of brain venous blood that may not detect an abnormality affecting small focal areas of brain. However, brain tissue oxygenation measured by polarographic intraparenchymal microcatheters correlated well with $SjO_2$ monitoring in 13 patients with severe head injury [35]. Difficulties with stability and consistency exist with the non-invasive near infrared spectrophotometer technique (NIRS) [36]. In clinical comparisons of $SjO_2$ versus NIRS, where global cerebral blood flow was altered by clamping of the internal carotid artery [37], circulatory arrest [38], or altering $PaCO_2$ levels [39], correlation of the two methods was found to be clinically acceptable. However, in a small qualitative study of head-injured patients, Kirkpatrick et al. [40] found NIRS to have a greater specificity than $SjO_2$ monitoring for detecting desaturation events. These two techniques measure different physical properties [41]; NIRS represents all blood (arterial, capillary and venous) within a small area, and may therefore be complimentary in alerting the clinician to secondary insults.

Many neuro-intensive care units now routinely use jugular bulb saturation as part of their multimodality monitoring [4, 42, 43, 44, 45]. The pathophysiology detected by monitoring of jugular bulb saturation is not only recorded but also used to implement treatment regimens aimed at reducing and preventing secondary insults.

## $SjO_2$-guided treatment regimens

### Physiological insult detection

As part of multimodality monitoring for acute brain injury e. g., pulse oximetry, ECG, ICP, arterial pressure, and CPP, jugular bulb saturation is an integral component which can make important contributions to clinical management by directing effective therapeutic strategies. Monitoring of $SjO_2$ has applications in neurosurgery, neuro-intensive care, and in cardiac surgery with cardiopulmonary bypass and hypothermic techniques [46]. Normally, if cerebral oxygen delivery decreases, the oxygen extraction ratio increases and $SjO_2$ will decrease acutely. CBF is then increased by compensatory regulatory mechanisms which are often absent or ineffective in acute brain injury. The main objective is to detect and treat cerebral hypoperfusion to minimise secondary insults [47] (Fig. 4). Much of the secondary brain injury is caused by ischaemia [48], perhaps as a consequence of inflammatory processes within the brain [49, 50, 51]. As mentioned above, normal values for $SjO_2$ are approximately 54–75 %. In acute brain injury low

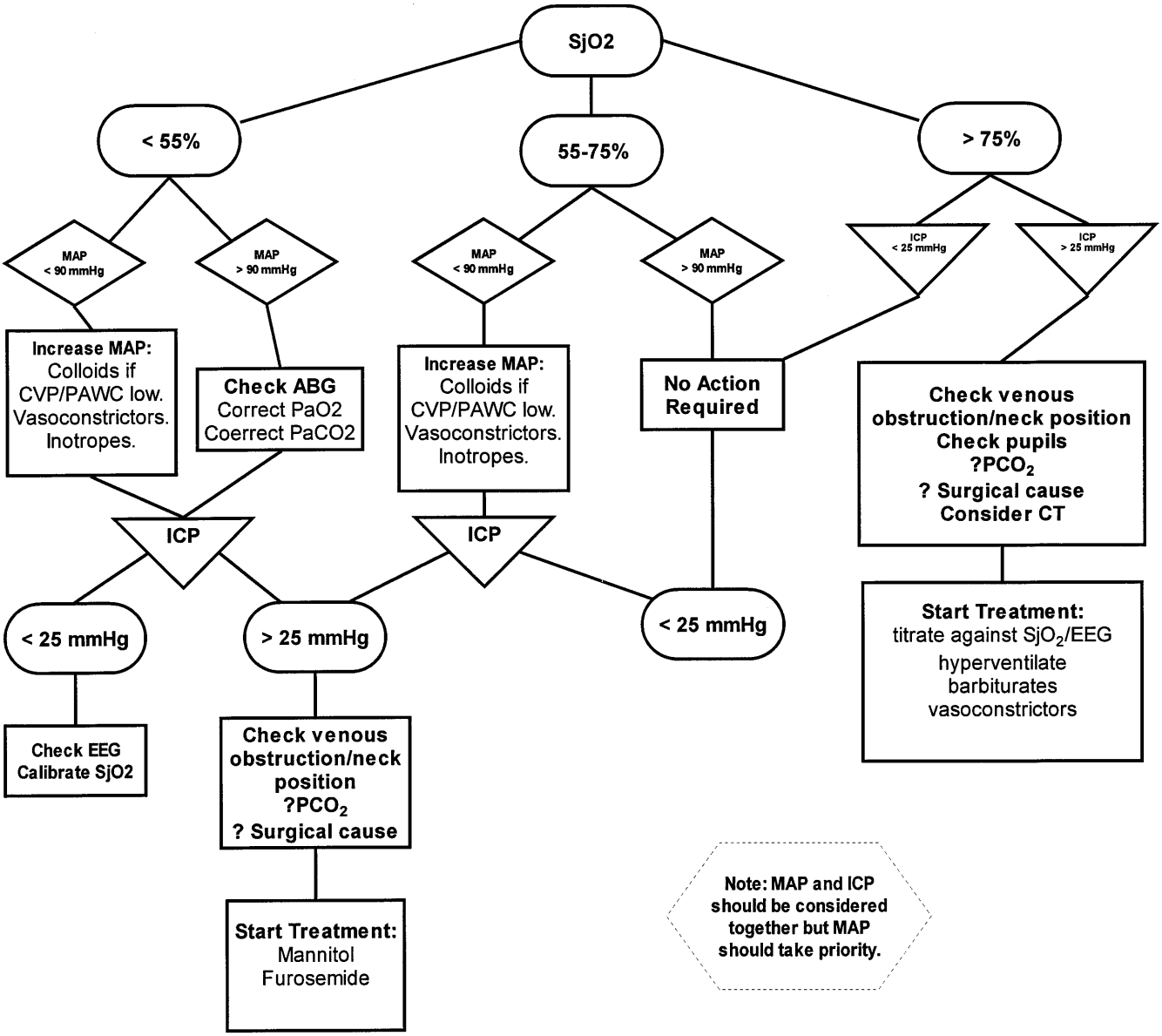


**Fig. 4** Jugular bulb saturation clinical guidelines

values are indicative of increased brain extraction of oxygen as a result of systemic arterial hypoxia, low CBF from hypotension or vasospasm, or a high ICP with a low CPP. Chan et al. [52] demonstrated a reduction in $SjO_2$ when CPP fell below 70 mmHg. Pyrexia and seizures (which may not be obvious if the patient is paralysed) also result in low $SjO_2$ due to increased brain metabolic requirements.

Conversely, an increased $SjO_2$ can be the product of either hyperaemia [53] or a failure of oxygen extraction (similar to the 'sick cell syndrome' manifest in septic patients). Another possibility exists in patients with a high $SjO_2$ who may have a very low CPP due to a high ICP as a pre-terminal event with shunting of arterial blood. A cause for hyperaemia should be sought and treated, e.g., hypercapnia [54]. There is currently no treatment option for failure of oxygen extraction.

## Acute brain injury

The most common cause of acute brain injury is trauma with an incidence of up to 400 per 100 000 in Europe and North America [55]. Subarachnoid haemorrhage is the other main reason for admission to neuro-intensive care units; both conditions justify $SjO_2$ monitoring as

further brain injury results in a worse outcome [47, 56, 57]. Monitoring from the earliest possible time following primary injury is important as many secondary events occur during this early period [4]. The most unstable period is often in the first few days when many episodes of desaturation in the jugular bulb have been demonstrated [54, 58, 59]. CBF is at its lowest during the first 12 h post-injury [60]. Many researchers now believe CPP is more critical than ICP and that CPP should be maintained above 70 mmHg to avoid ischaemic brain metabolism implied by a low $SjO_2$ [52]. However, as cerebral autoregulatory mechanisms may not be functioning, $SjO_2$ monitoring becomes an even more important tool to guide therapy. It correlates well with brain tissue oxygenation [35] and is less invasive. There is some evidence that mannitol may initially reduce oxygenation of the brain; this can be detected by $SjO_2$ monitoring and treated, e.g., by increasing $PaCO_2$.

In addition to confirming the deleterious effects of a low CPP or systemic desaturation, $SjO_2$ can be used to guide interventional therapies. The initial active treatment for a high ICP is to 'bag' the patient to lower $PaCO_2$ levels, and thus lower ICP via cerebral vasoconstriction. It is impossible to know the threshold $PaCO_2$ for each patient before cerebral ischaemia occurs unless $SjO_2$ is monitored [59]. This is because an individual's $PaCO_2$ threshold is dependent upon their CPP, the steady-state $PaCO_2$, and comorbid factors such as atherosclerosis. $SjO_2$ monitoring permits therapeutic hypocapnia until the $SjO_2$ is nearing the lower limit of normal. Extreme hyperventilation (to $PaCO_2$ 3.3 kPa) has been shown to be deleterious in severe head injury [61]. Cruz et al. [62] showed a reduction in $SjO_2$ when hypocapnia was induced for control of ICP. Hence, ventilation as a therapeutic manoeuvre is a strong indication for the monitoring of $SjO_2$ being recommended by the American Association of Neurosurgeons [63]; in its absence, current guidelines recommend ventilation to a minimum $PaCO_2$ of 4 kPa [64]. Hyperoxia is another therapeutic manoeuvre described by Matta et al. [65] to allow the lowest possible $PaCO_2$ while preserving global oxygenation as monitored by $SjO_2$.

With regard to subarachnoid haemorrhage, a major cause of morbidity and mortality is delayed neurological deficit [66], a syndrome including arterial vessel narrowing and neurological deficit. Systemic and local treatments for this vessel narrowing involves triple 'H' therapy (hypertension, haemodilution, and hypervolaemia) [67], and localised injection of the arterial vasodilator drug papaverine. Using jugular bulb saturation to monitor papaverine treatment, immediate improvements were noted by Fandino et al., in a small series of ten patients [68].

## $SjO_2$ measurement and outcome data

There are several clinical features associated with a poor outcome following traumatic brain injury including hypoxia, hypotension, and pyrexia [4, 47, 56]. Abnormalities in $SjO_2$ have also recently been associated with a poor outcome compared to patients who do not demonstrate such physiological derangement. In a study of more than 100 patients admitted to intensive care after traumatic brain injury, Gopinath et al. [2] demonstrated that multiple episodes of low $SjO_2$ were more often associated with poorer outcomes than those patients who had only one or no desaturation events. Conversely, high $SjO_2$ values have also been associated with poor outcomes [7, 69].

## Other clinical uses for $SjO_2$ monitoring

### Cardiac bypass

A new application for $SjO_2$ techniques is the intra- and post-operative monitoring of patients undergoing cardiopulmonary bypass. There is no relationship between mixed venous saturation and $SjO_2$ during stable hypothermia or following re-warming [70], hence the importance of direct $SjO_2$ monitoring. A particularly vulnerable time for cerebral ischaemia, as assessed using a jugular bulb catheter, is during normothermic bypass, [71] especially during the re-warming phase [46, 72, 73]. In children, where very low temperatures are used during bypass, different cooling regimens have been investigated using jugular bulb sampling to determine the degree of brain protection [74]. However, it is controversial whether continuous saturation catheters can be used with confidence under hypothermic conditions [75, 76].

### Cardiac arrest

The global acute brain injury from hypoxia during cardiac arrest has been studied by numerous techniques including $SjO_2$. An interesting finding from a small study was that non-survivors had mean $SjO_2$ values of 80%, significantly higher than survivors (67%) [77]. This could be explained by hyperaemia or an inability of damaged neurones to utilise delivered oxygen. In another small study patients with the poorest outcomes had $SjO_2$ values higher than mixed venous values, while the opposite was seen in those who fared better [78]. These findings are controversial as other studies have found no difference in $SjO_2$ values [79].

## Research applications for jugular bulb monitoring

Access to blood draining from the brain provides the opportunity for a range of research possibilities. In early studies sampling was intermittent and mainly used for oxygen saturation/content measurement. The development of continuous saturation monitoring by fibreoptic methods means that therapeutic interventions may be monitored and evaluated, and enable observational studies relating physiological derangement to outcome parameters.

In addition to oxygen measurement, analysis of blood sampled from the jugular bulb has implicated the brain in inflammatory processes. Studies have shown the brain to be contributing to the inflammatory response after primary injury from trauma or subarachnoid haemorrhage with production of cytokines such as interleukin-6 [49]; high concentrations of sICAM-1 have been associated with poor outcome after traumatic brain injury.

Post-mortem examinations of brains of patients with traumatic brain injury have demonstrated ischaemic areas not related to the primary injury [48]. This led to measurement of blood lactate in the jugular bulb to identify cerebral ischaemia [80]. The lactate–oxygen index (LOI) was derived: the negative ratio of cerebral arterio-venous difference in lactate to the difference in oxygen ($LOI = -AVDL/AVDO_2$) [81]. The LOI is normally less than 0.03; higher values ( $> 0.08$) may indicate ischaemia. It has been used widely to study brain metabolism during cardiac bypass and liver transplantation, after brain trauma, and during drug trials.

## Conclusion

Jugular bulb monitoring has evolved over recent years into a versatile and useful addition to multimodality monitoring for acutely brain injured patients in intensive care. There is abundant evidence to support the fact that secondary physiological insults to the injured brain result in further brain damage; clinicians have a duty to minimise these. Jugular bulb saturation monitoring provides early warning of brain ischaemia from systemic disturbances such as hypotension and hypoxia, and permits therapeutic manoeuvres to be undertaken safely without inducing brain ischaemia. The risks associated with the technique are far outweighed by the benefits. $SjO_2$ monitoring should be considered part of the optimal modality monitoring in every patient with acute brain injury requiring ventilation and intensive care.

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