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Detection of Cerebral Venous Desaturation by Continuous Jugular Bulb Oximetry Following Acute Neurotrauma

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SUMMARY

A prospective observational study was performed to assess the reliability of fibreoptic oximetric catheters and to identify the incidence and causes of jugular bulb oxygen desaturation in patients with acute closed head injury. There were twenty-five patients (30±16 years) with GCS ≤8 in this study.

Jugular bulb oximetry, mean arterial pressure, intracranial pressure, end-tidal CO_2 and pulse oximetry were monitored continuously. Catheter calibration against a laboratory oximeter was performed post insertion and thereafter eight-hourly. Cerebral venous desaturation was defined as a jugular bulb oxygen saturation <55% of >10 minutes duration. There was a poor correlation for the first in vivo calibration ($r^2=0.602$, $P<0.001$, $n=25$). Thereafter a close correlation between jugular bulb catheter and oximetry values was demonstrated ($r^2=0.868$, $P<0.001$, $n=205$). Forty-two episodes of jugular bulb oxygen desaturation of 88 minutes mean duration (range 10 to 555) were observed. 83% occurred within 48 hours following injury. Hypocapnia was associated in 45% of episodes; hypoperfusion in 22%; raised ICP in 9% and a combination of the above in 24%.

Validation with a laboratory oximeter is essential prior to continuous jugular bulb oximetry. Sustained episodes of cerebral venous desaturation are frequent within the first 48 hours following acute head injury. Factors such as hypocapnia and cerebral hypoperfusion that primarily reduce cerebral blood flow are predominant.

Key Words: MEASUREMENT TECHNIQUES: continuous jugular bulb oximetry, intracranial pressure, cerebral perfusion pressure, capnography; BRAIN: venous desaturation, trauma.

Following neurotrauma, autoregulatory mechanisms for maintaining cerebral perfusion and oxygenation may be compromised, rendering cerebral tissue more vulnerable to secondary ischaemic/hypoxic insults[1].

Pathological studies have shown secondary ischaemic brain damage in 92% of fatal head injuries. The same group of investigators reported a similar incidence 10 years later despite improvements in intensive care[2,3]

Clinical studies utilizing a computerized system for monitoring physiological variables have shown that 91% of 124 patients with severe head injury suffered at least one significant secondary insult. Further subset analysis showed that hypoxia, hypotension and pyrexia were significant predictors of mortality[4].

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Measurement of intracranial pressure and cerebral perfusion pressure does not reflect adequacy of cerebral oxygen utilization. Furthermore, there is no readily available continuous clinical measurement of cerebral blood flow or cerebral metabolic rate.

Jugular bulb oximetry (SjO_2) continuously measures cerebral venous oxygen saturation. Normally, cerebral blood flow and metabolism are coupled, so that the arterio-jugular oxygen saturation difference remains constant. In the majority of head-injured patients, cerebral blood flow and metabolic rate are uncoupled due to loss of cerebral autoregulation[5-7]. Cerebral blood flow can increase or decrease independently of the metabolic rate. Arterio-jugular oxygen saturation difference will vary and may reflect the adequacy of cerebral blood flow for a given cerebral metabolic rate.

The aims of this prospective observational study were twofold. Firstly, fibreoptic oximetric catheters were tested for reliability in vivo. Secondly, the incidence and causes of jugular bulb oxygen desaturation in patients with acute closed head injury were evaluated.

This study was performed within the guidelines set out by the National Health and Medical Research

Council and was approved by the Research Ethics Committee of the Royal Adelaide Hospital.

PATIENTS AND METHODS

Patients admitted to the Royal Adelaide Hospital Intensive Care Unit with a closed head injury and a Glasgow Coma Score equal to or less than 8 were studied. All patients were mechanically ventilated to achieve modest hypocapnia (35-40 mmHg) and to maintain arterial oxygen tension (P_aO_2) greater than 100 mmHg. All patients were sedated with combined opioid and benzodiazepine infusion and nursed at 30 degrees head elevation.

MONITORING

Routine haemodynamic monitoring included systemic arterial pressure via arterial cannulation (Sorenson Transpac disposable transducer, Abbott Critical Care System, North Chicago, Illinois, USA), central venous pressure and continuous heart rate via electrocardiography. Respiratory monitoring included continuous pulse oximetry (Criticare Systems Inc, 504 Pulse Oximeter), end-tidal capnography (Datex Normocap, Instrumentation Corp, Helsinki, Finland), minute ventilation and lung compliance (Servo 900C, Siemens Systems, Helsinki, Finland). Intracranial pressure (ICP) was monitored by a frontal intraparenchymal fibreoptic pressure tip transducer (Model 110-4B Camino Laboratories San Diego, California, USA).

Jugular bulb oximetry was measured as follows:

1. Equipment

Jugular bulb oxygen saturation was monitored by a 4-French fibreoptic oxygen saturation catheter (40 cm Shaw Opticath. Abbott Laboratories, North Chicago, Illinois, USA).

2. Procedure

The internal jugular vein with the greater drainage was determined by compressing each internal jugular vein for 20 seconds[8]. The side producing the greater rise in intracranial pressure was selected for cannulation. If the intracranial pressure rise was equal, then the side of greater trauma as determined by CT was chosen. If the CT indicated a diffuse injury, then the right side, which is usually the side of dominant drainage, was cannulated.

The oximetric catheter was introduced percutaneously by retrograde cannulation using the Seldinger technique. The catheter tip was verified radiologically to be above the lower border of C1, thereby avoiding extracranial venous contamination. Patency of the catheter sampling port was maintained by a continuous infusion of heparinized saline (10 U/ml) at 3 ml/hour.

Patients were nursed at 30 degrees head up with the head maintained in the neutral position.

3. Calibration

Prior to insertion, the catheter was calibrated using a reference cuvette supplied with the catheter. Calibration against a laboratory Co-oximeter (IL-482, Instrumentation Laboratories, North Chicago, Illinois, USA) was performed immediately after insertion and every eight hours by withdrawing a blood sample through the catheter. If the catheter or laboratory oximeter values differed by more than 3%, the catheter was recalibrated in accordance with the laboratory oximeter saturation.

4. Jugular Bulb Oxygen Desaturation

Jugular bulb oxygen desaturation was defined as an oxygen saturation less than 55% lasting more than ten minutes under conditions of normal catheter light intensity. Poor catheter light intensity has been associated with artifactual saturation values[9]. The following physiological parameters were recorded during an episode of desaturation: end-tidal carbon dioxide ($ETCO_2$), cerebral perfusion pressure (CPP), ICP and pulse oximetry (SpO_2). Abnormal variation of these measures were defined as follows: (1) hypocapnia ($ETCO_2 < 30$ mmHg); (2) cerebral hypoperfusion due to arterial hypotension in the absence of raised intracranial pressure where CPP is less than 60 mmHg; (3) intracranial hypertension (ICP > 20 mmHg); (4) hypoxia ($SpO_2 < 90\%$). An arterial blood gas sample taken at this time confirmed the end-tidal and pulse oximetry values. Duration of these perturbations for ten or more minutes during an episode of desaturation was considered an association.

Statistics

Values for oxygen saturation determined by the two methods were compared using linear regression analysis.

RESULTS

Twenty-five consecutive severely head-injured patients (17 male, 8 female) with a mean age of 30 years were studied. The mean delay from the time of head injury to the commencement of jugular bulb oximetry was six hours, while the mean delay in time from arrival at hospital to monitoring was seven hours. There was no incidence of infection, thrombosis or any other problems directly related to the catheters for the entire period of insertion, totalling 1973 hours.

TABLE 1

Case nos	Age (years)	Sex	GCS	Time from injury to admission (hours)	Time from admission to monitoring (hours)	Episodes of jugular bulb desaturation	Clinical condition
1	65	M	7	0.5	3	1	CT: Bilateral frontal focal haemorrhages.
2	15	M	8	2	8	2	Left pupil dilated. CT: left extradural haematoma (evacuated).
3	16	M	3	9	2	1	CT: Small left frontal intracerebral haemorrhage, small contusions left sylvian fissure.
4	21	M	7	14	6	2	CT: Bilateral temporal contusions, thin left subdural.
5	21	M	7	24	12	2	CT: Right intracerebellar haematoma (evacuated), left fronto-temporal contusion, thin left subtemporal subdural. Right pneumothorax, severe abrasions.
6	30	M	6	4	7	8	CT: Thin right frontal subdural, bilateral frontal contusions.
7	49	M	5	38	6	6	CT: Left internal capsule intracerebral haemorrhage. Multiple limb fractures.
8	18	F	8	1	4	1	CT: Diffuse cerebral swelling, left frontal subdural haematoma (evacuated), basal skull fracture.
9	15	F	5	2.5	2.5	2	CT: Diffuse cerebral swelling.
10	17	M	5	3	13	2	CT: Left post-temporal contusion. Multiple limb fractures.
11	27	M	3	4	10	1	CT: Right sylvian fissure contusions with focal cerebral oedema.
12	55	M	7	1	18	1	CT: Left frontal subdural haemorrhage (evacuated), swollen left cerebral hemisphere.
13	33	F	4	1	3	5	CT: Left cerebral hemisphere swelling.
14	14	F	3	1	13	0	CT: Multiple cerebral contusions.
15	26	F	3	4	4	1	CT: Left frontal subdural (evacuated).
16	26	M	3	2	13.5	1	CT: Right fronto-temporal subdural (evacuated), right temporal contusion. Pelvic and multiple limb fractures.
17	19	F	4	4	4	0	CT: Multiple cerebral contusions, intraventricular and subarachnoid haemorrhage.
18	19	M	6	1	6	3	CT: Multiple haemorrhagic contusions.
19	66	F	4	2	12	1	CT: Bilateral temporal contusions, basal skull fracture. Multiple limb fractures.
20	20	F	5	2	5	0	CT: Left subdural haematoma (evacuated), basal skull fracture. Facial fractures. Fractured pelvis.
21	30	M	6	5	4	2	CT: Shallow right extradural, left parietal contusion, basal skull fracture. Left ulnar fracture.
22	17	M	3	6	5	0	CT: Left subdural haematoma (evacuated), multiple contusions, basal skull fracture. Facial fractures.
23	19	M	3	5	6	0	CT: Bilateral frontal contusions, small right fronto-temporal subdural, basal skull fracture. Facial fractures.
24	56	M	8	6	7	0	CT: Small left posterior, parietal extradural haematoma, left temporal intracerebral haemorrhage, left parieto-occipital skull fracture.
25	22	M	4	2	9	0	CT: Right fronto-temporal contusion, basal skull fracture. Facial fractures. Rib fractures.

There was a poor correlation between the catheter and laboratory oximeter at the first in vivo calibration ($r^2 = 0.602$, $P < 0.001$, $n = 25$).

Thereafter, during routine eight-hourly calibration there was a significant correlation ($r^2 = 0.868$, $P < 0.001$, $n = 205$) between the laboratory oximeter and catheter saturation values.

These data suggest that once the initial in vivo calibration is performed this catheter system is accurate over the clinical ranges observed.

Episodes of jugular bulb desaturation occurring under conditions of normal catheter light intensity were examined. There were 42 episodes of jugular bulb oxygen desaturation in 18 patients, ranging in duration from 10 minutes to 9 hours with a median duration of 40 minutes. Of these desaturations, 83% occurred within 48 hours of the head trauma.

Jugular bulb oxygen desaturation was most frequently associated with hypocapnia ($ETCO_2 < 30$ mmHg), occurring as the only association in 45% (19/42) of episodes. The mean P_aCO_2 measured at the time of these episodes was 28.5 mmHg (range 22 to 34 mmHg). Hyperventilation during periods of raised intracranial pressure was associated with reversible jugular bulb oxygen desaturation in two patients. Cerebral hypoperfusion ($CPP < 60$ mmHg) due to arterial hypotension in the absence of intracranial hypertension was the only association in 22% (9/42). Intracranial hypertension ($ICP > 20$ mmHg) was the only association in only 9% (4/42). A further 5%

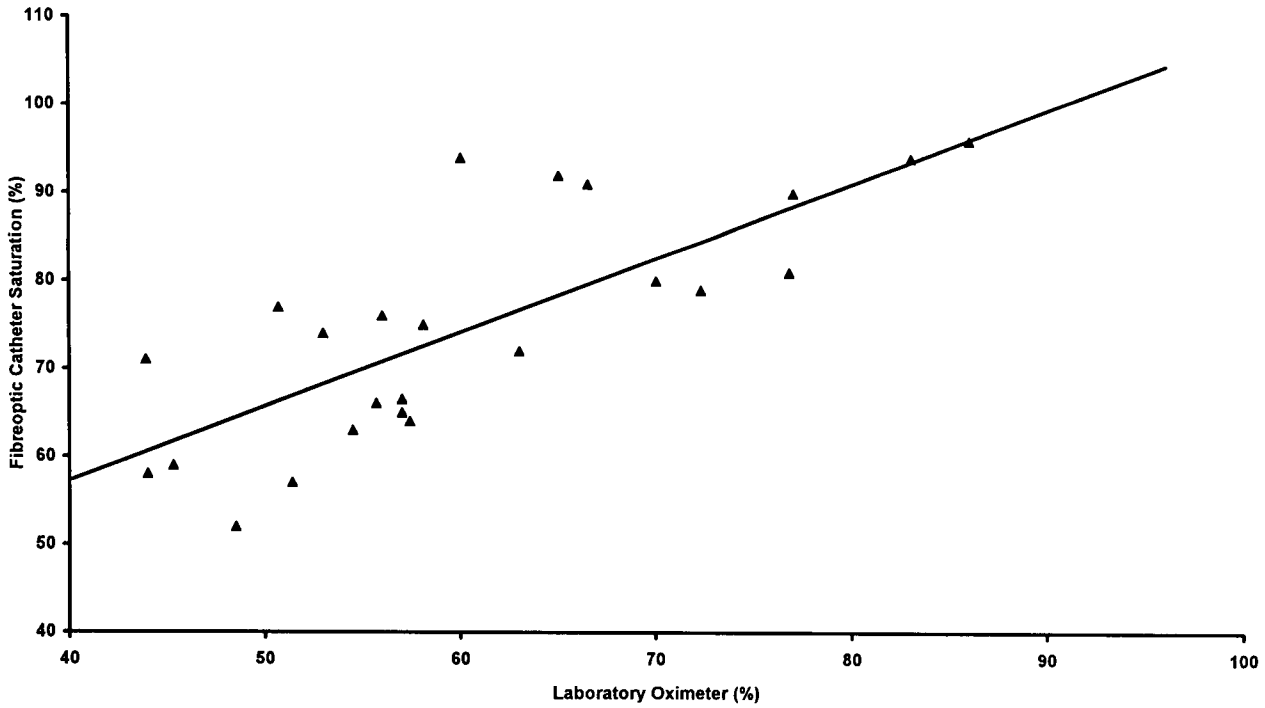

FIGURE 1: Scatter plot of jugular bulb oxygen saturation by laboratory oximeter vs fibreoptic catheter for the first in vivo calibration ($r^2=0.602$, $P<0.001$, n=25).

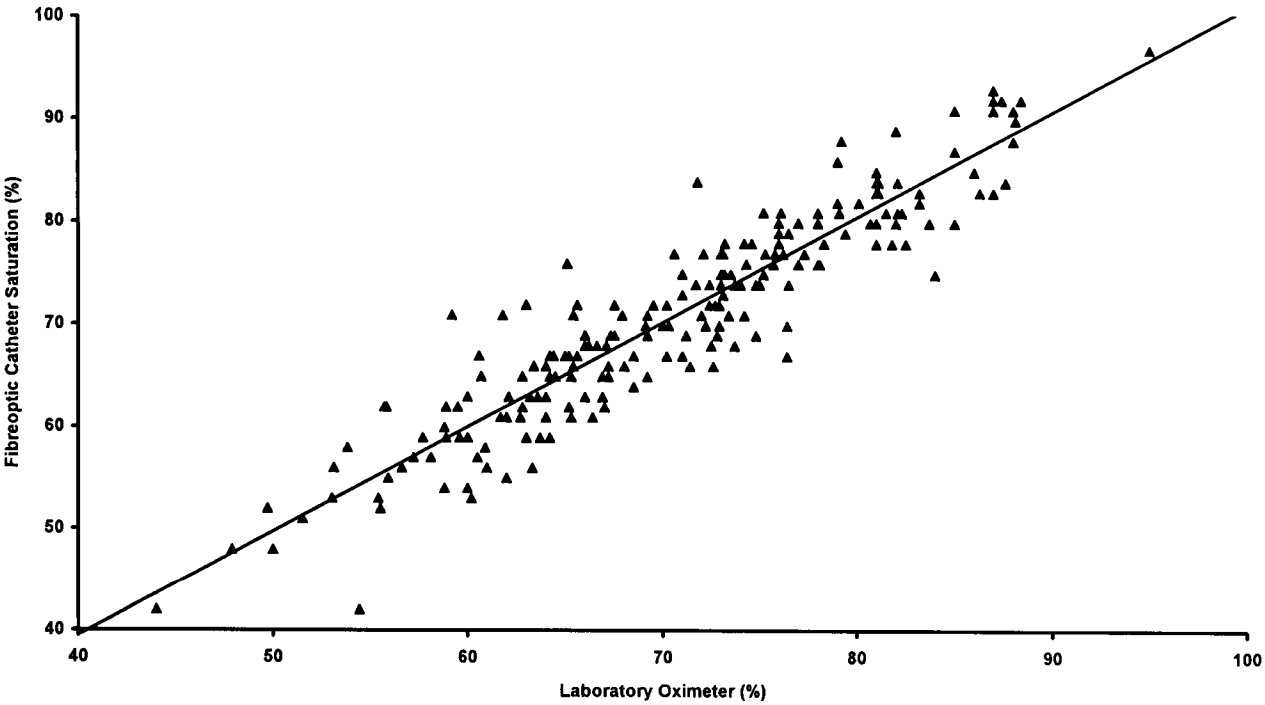

FIGURE 2: Correlation between jugular bulb oxygen saturations determined by laboratory oximeter and fibreoptic catheter on samples taken every 8 hours following the first in vivo calibration ($r^2=0,868$, $P<0.001$, n=205).

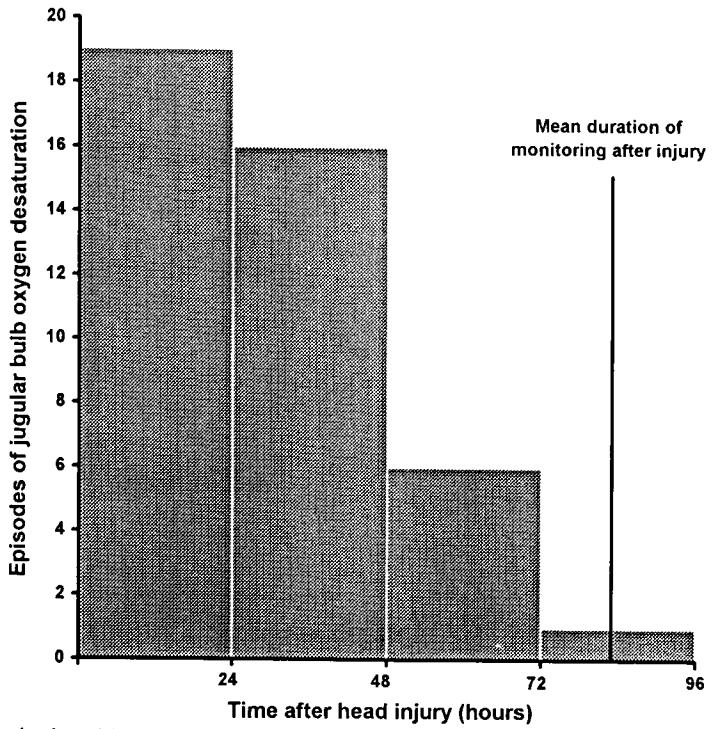

FIGURE 3: Distribution of episodes of jugular bulb oxygen desaturation in relationship to the time elapsed since head injury.

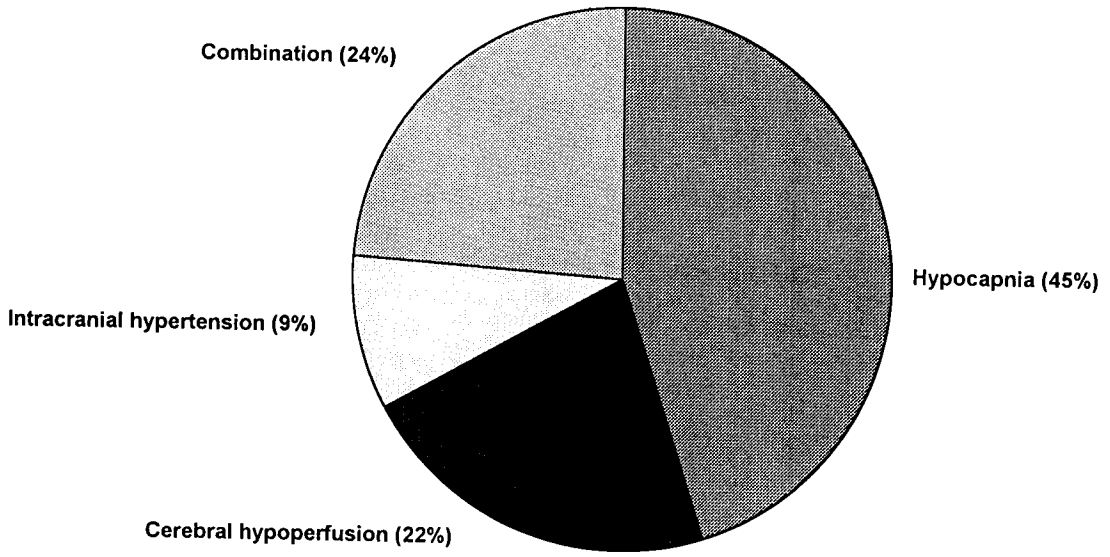

FIGURE 4: The association between jugular bulb oxygen desaturation and hypocapnia, cerebral hypoperfusion, intracranial hypertension and a combination of these.

(2/42) occurred with raised ICP associated with cerebral hypoperfusion. It was more common to see episodic rises in ICP due to suctioning or other patient care manoeuvres resulting in transient increases in jugular venous oxygen saturation. Combinations of the above insults were present in 24% (10/42) of episodes.

DISCUSSION

One of the principal reasons for monitoring patients with severe head injury is the early detection of secondary ischaemic/hypoxic insults which have been clearly shown to adversely affect outcome[10]. The complex relationship between cerebral blood flow and

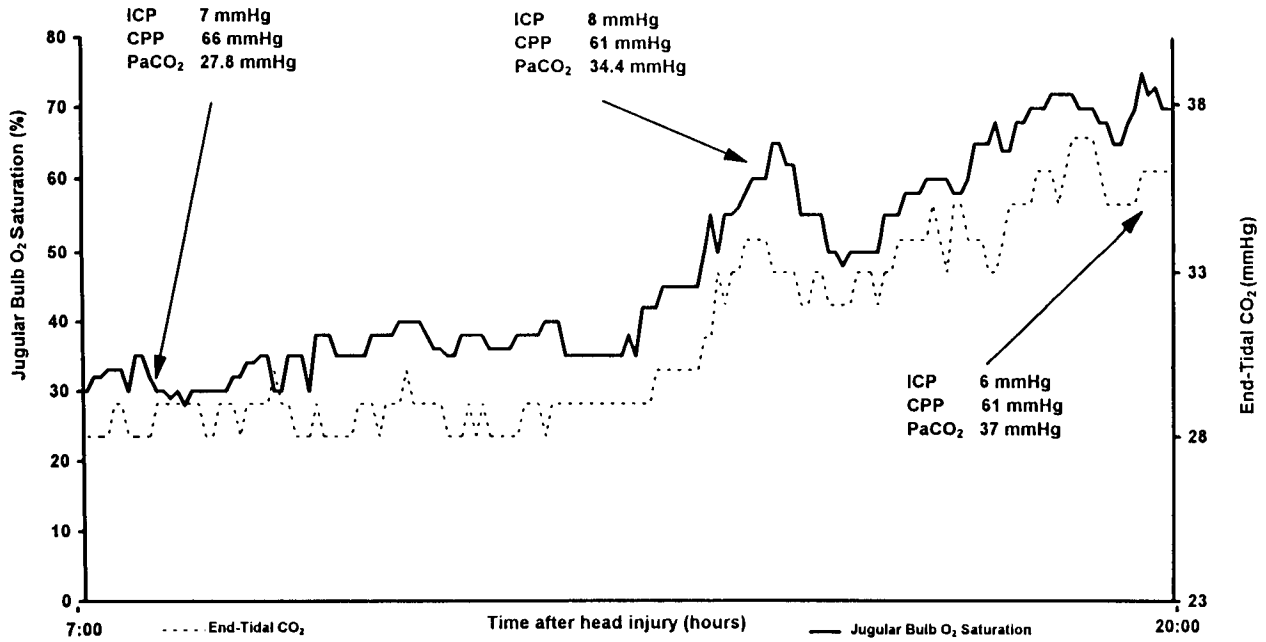

FIGURE5: Jugular bulb oxygen desaturation associated with hypocapnia. Hyperventilation has reduced the end-tidal CO_2 to 28 mmHg confirmed on arterial sample. The jugular bulb oxygen saturation was recorded at 32% indicating cerebral hypoperfusion. As P_aCO_2 increased, the jugular bulb oxygen saturation followed reaching a value indicating adequate cerebral perfusion at a P_aCO_2 of 37 mmHg.

cerebral metabolism after brain injury remains difficult to measure directly; jugular bulb oximetry measures some aspect of this relationship. This study shows that jugular bulb catheterization is a safe procedure. There were no infections, thrombosis or any other problems directly related to the catheters. Similar low morbidities have been reported from other centres with accidental carotid puncture being the most frequent insertion complication (3 to 4%) and catheter infection observed rarely (0 to 2%)[11,12].

In vivo calibration of the oximetric catheter against laboratory co-oximetry is essential. The disparity between in vitro calibration using the reference cuvette and in vitro calibration against a laboratory oximeter is consistent with the findings of others[9,13]. It has been suggested that the discrepancy may be due to catheter design. The catheters were originally developed for placement in the pulmonary artery where they are subjected to pulsatile flow away from the catheter tip rather than non-pulsatile blood flowing toward the catheter as occurs in the internal jugular vein[13]. In vitro calibration using the supplied reference cuvette simply checks the integrity of the fibre optic system prior to insertion and not the accuracy.

This study demonstrated that following insertion and in vivo calibration the catheter is accurate over the clinical range of measurement and remains so provided calibration is performed every eight hours.

The second aim of the study was to determine the clinical utility of jugular bulb oximetry.

In line with other studies, we found that episodes of cerebral circulatory compromise occurred frequently, and would not have been detected without jugular bulb oxygen measurement[9,13,14].

The majority of jugular bulb oxygen desaturations occurred within the first 48 hours of injury. Recent work suggests that the frequency of episodes of jugular bulb oxygen desaturation is closely correlated with outcome[15]. Jugular bulb desaturation occurs even in the closely supervised conditions of ICU. It is reasonable to predict that periods of undetected desaturation will occur during intrahospital transport from the emergency department to radiology to intensive care, and may be a significant factor in the known poor outcome in patients with head and extracranial injuries. These observations emphasize the need to institute appropriate intracranial monitoring as soon as possible once resuscitation is complete. In this study the commonest cause of jugular bulb oxygen desaturation was hypocapnia due to hyperventilation, commonly used as standard therapy of acute head injury. The cerebral vasoconstrictive effects of hypocapnia are illustrated in Figure 5.

These episodes of cerebral oligaemia would not have been detected by monitoring intracranial pressure and cerebral perfusion pressure alone. Recent studies have reported the adverse effects of routine hyperventilation on outcome in severely head-injured patients[16]. Jugular bulb oximetry may allow more selective and appropriate application of vasoconstrictive manoeuvres such

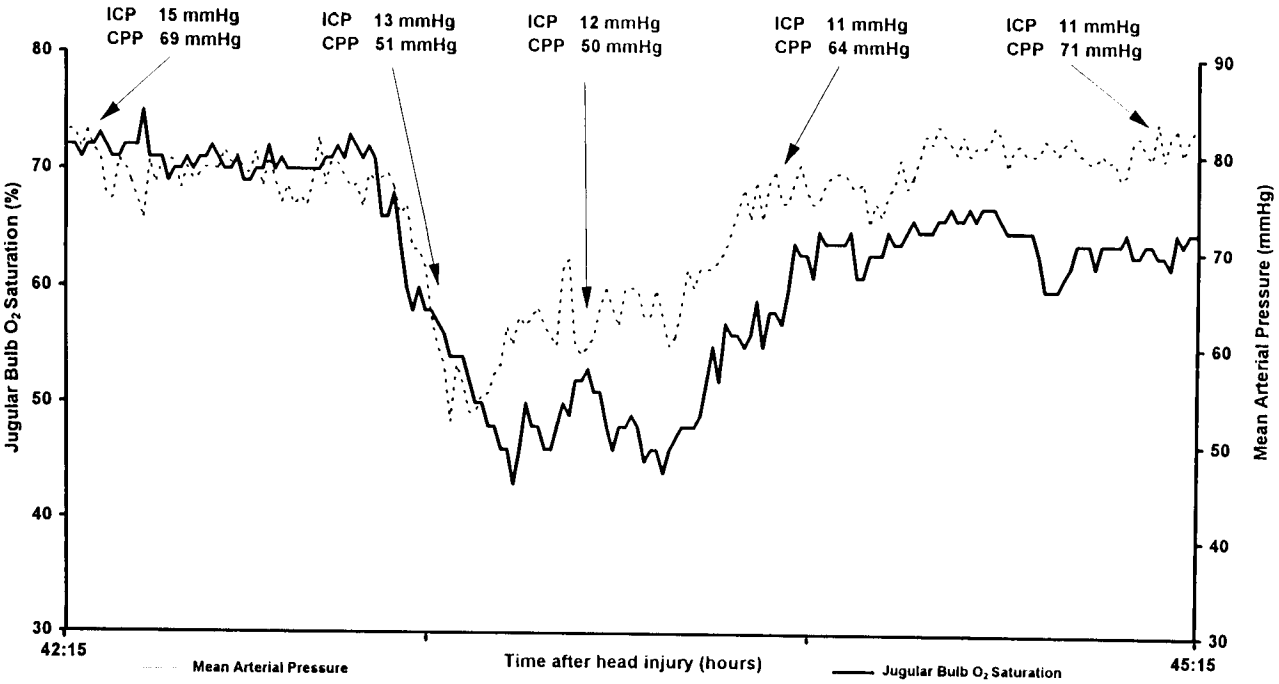

FIGURE 6: Cerebral hypoperfusion due to arterial hypotension associated with a decrease in jugular bulb oxygen saturation to a critical level. No change in ICP was noted.

as hyperventilation[17].

ICP monitoring alone does not reflect cerebral oxygen utilization. Increases in ICP occurred late or not at all following jugular bulb oxygen desaturation due to cerebral hypoperfusion (Figure 6).

Artifactual desaturation due to poor light intensity was not specifically examined by this study. However approximately 30% of "desaturations" observed in the first eight patients were attributed to poor catheter light intensity, most often due to poor catheter position. Others have reported that as many as 50% of "desaturations" are artifactual[18]. With increased awareness of the importance of head and neck positioning[14], ensuring correct catheter placement and firmly securing the catheter to patient, this incidence in our experience declined dramatically, and in the remaining patients, the incidence of false "desaturation" was less than 5%.

The place of this monitoring method in the routine clinical monitoring of head-injured patients is yet to be determined. However it is interesting to record that over the course of this study there was greater attention to maintaining P_aCO_2 at normocarbic levels and CPP>70 mmHg and this was associated with a reduced incidence of desaturation. Indeed no episodes were detected for the last four patients (Table 1). The major limitation of jugular bulb oximetry is the global nature of the measurement. It is probable that focal areas of cerebral oligaemia will not be detected by this method. In addition, when cerebral blood flow is low, extracranial contamination may increase the jugular bulb saturation value. It has also been reported recently that the mean venous oxygen saturation difference between jugular bulbs may be as high as 15%. Careful evaluation of the dominant side of jugular venous drainage by CT or jugular compression test may reduce this error[8,11]. However, transient episodes of global cerebral oligaemia can be detected by this monitoring method and provide clinically useful and relevant information regarding the complex relationship between cerebral metabolic rate and cerebral blood flow hitherto undetected by conventional ICP and CPP monitoring[9,14,19]. This may allow the titration of therapies applied to head injured patients[18,20,21]. Further prospective studies examining the utility of jugular bulb oximetry in guiding therapy and the effect on outcome are now required.

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