Elevated jugular venous oxygen saturation after severe head injury

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Object. The aim of this study was to investigate the incidence of elevated ($\geq 75\%$) jugular venous oxygen saturation (SjvO₂) and its relationship to cerebral hemodynamic and metabolic parameters and to outcome after severe head injury.

Methods. Data from 450 severely head injured patients admitted to the Neurosurgical Intensive Care Unit of Ben Taub General Hospital were analyzed retrospectively. The SjvO₂ was measured in blood obtained from indwelling jugular bulb catheters. Patients were classified into the following categories: high (Group I), normal (Group II), or low SjvO₂ (Group III) if their mean SjvO₂ over the duration of monitoring was 75% or higher, 74 to 56%, or 55% or lower, respectively.

A high SjvO₂ occurred in 19.1% of patients. There was no consistent relationship between SjvO₂ and simultaneous cerebral blood flow (CBF) or cerebral perfusion pressure measurements. Compared with Groups II and III, the patients in Group I had a significantly higher CBF and lower cerebral metabolic rate of oxygen (CMRO₂). In Group I, the outcomes were death or persistent vegetative state in 48.8% of patients and severe disability in 25.6%. These outcomes were significantly worse than for patients in Group II. Within Group I, the patients with a poor neurological outcome were older and more likely to have suffered a focal head injury; they demonstrated a lower CMRO₂ and a greater rate of cerebral lactate production than the patients who attained a favorable outcome.

Conclusions. Posttraumatic elevation of SjvO₂ is common but cannot be automatically equated with hyperemia. Instead, elevated SjvO₂ is a heterogeneous condition that is associated with poor outcome after head injury and may carry important implications for the management of comatose patients.

KEY WORDS • cerebral blood flow • cerebral metabolic rate of oxygen • cerebral perfusion pressure • dysoxia • head injury • ischemia • jugular venous oxygen saturation

JUGULAR venous oxygen saturation (SjvO₂) reflects the balance between cerebral oxygen delivery and the cerebral metabolic rate of oxygen (CMRO₂), if arterial oxyhemoglobin saturation, hemoglobin concentration, and the hemoglobin dissociation curve remain constant. Any disturbance that increases CMRO₂ or decreases oxygen delivery may decrease SjvO₂. Conversely, a disorder that decreases CMRO₂ or increases oxygen delivery may increase SjvO₂.

Low SjvO₂ reliably indicates cerebral hypoperfusion or ischemia. In contrast, there are several possible causes of elevated SjvO₂, the elucidation of which may be difficult without concomitant measurements of regional and/or global cerebral blood flow (CBF).¹ Focal cerebral ischemia may occur in the presence of a normal or elevated SjvO₂ if the involved area of the brain is small or if the reduced oxygen saturation from the ischemic brain is offset by a high oxygen saturation from surrounding hyperemic brain. Dysoxia (defined as oxygen-limited cytochrome turnover)^{7,23} and/or restricted oxygen diffusion from the erythrocytes to the mitochondria can also result in a normal or elevated SjvO₂. In some cases, measured

values of cerebral oxygen extraction and CMRO₂ do not accurately reflect neural metabolic demands, and cerebral ischemic damage could occur despite a superabundant supply of oxygen to the brain. Although oxygen delivery and oxygen consumption are measurable, the true metabolic requirements of the cells, the "oxygen demand," cannot be determined.

The anatomy of the jugular venous system may also play a role in generating high SjvO₂ values. The internal jugular vein chosen for monitoring (right or left) may affect the values obtained for SjvO₂.²⁷ More important, when CBF is globally low, extracerebral contamination of blood in the jugular bulb may be significant and may contribute to an increase in the value obtained for SjvO₂. Rapid drawing of the blood sample through the catheter (rates > 2 ml/minute) has been another source of artifactual increases in SjvO₂.¹⁶

The purpose of this study was to investigate the incidence of SjvO₂ values above the upper limit of normal ($\geq 75\%$) in severely head injured patients and to relate these elevated values to measurements of global CBF and metabolism and also to clinical outcome.

Clinical Material and Methods

Patient Characteristics

This study was approved by the Baylor Affiliates Review Board for Human Subject Research, and informed consent for participation was obtained from the patients' family members. Data on CBF and cerebral metabolic rates were gathered from 465 severely head injured patients admitted to the Neurosurgical Intensive Care Unit of Ben Taub General Hospital between 1986 and 1997. Their levels of consciousness were seriously impaired: these patients were either not obeying commands on admission or deteriorated to this level after admission. All patients were managed by a standard protocol that emphasized prompt evacuation of intracranial hematomas and prevention of secondary insults to the brain. Intracranial pressure (ICP) was continuously monitored using a ventriculostomy, a parenchymal microtransducer, or a fiberoptic monitor. Data from periods when the patients were treated with pentobarbital coma for refractory intracranial hypertension were excluded from the analysis. This resulted in total exclusion of 15 patients from the study because no measurements of CBF prior to receiving barbiturates were available for them and also resulted in exclusion of segments of data from 67 of the remaining 450 patients.

Physiological Parameters

By using an oximeter (IL-284 CO-Oximeter; Instrumentation Laboratory, Lexington, MA), SjvO_2 was determined from blood samples drawn through an indwelling catheter positioned in the jugular bulb. In the first 102 patients, the catheter was placed on the side of the worst injury or on the right side if the injury was diffuse. In the remaining patients, the catheter was placed on the side of the larger internal jugular vein. Blood samples were obtained at least every 8 to 24 hours to calibrate a concomitantly placed fiberoptic oxygen saturation catheter (No. 4 French catheter; Abbott Laboratories, North Chicago, IL) that measured SjvO_2 continuously. Samples were also obtained in the event of jugular venous desaturation. The SjvO_2 data analyzed in this report were all obtained from the oximeter and not the catheter.

Glucose and lactate concentrations were measured using a stat-Plus analyzer (YSI 2300; Yellow Springs Instruments, Yellow Springs, OH). Global CBF was estimated using the Kety-Schmidt method with N_2O as a tracer. The CMRO_2 and cerebral metabolic rates of glucose (CMRG) and lactate (CMRL) were calculated from the product of the CBF and the arteriovenous difference of oxygen (AVDO_2), glucose (AVDG), and lactate (AVDL), respectively. The lactate/glucose index (LGI) was calculated using the following formula: $(\text{AVDL}) \times 100\% / (\text{AVDG} \times 2)$. The LGI is used to estimate the percentage of total glucose that is metabolized anaerobically to the end-product lactate and is normally less than 5%.⁵ Cerebrovascular resistance (CVR) values were obtained by dividing cerebral perfusion pressure (CPP) by CBF.

Classification of Groups

High SjvO_2 (Group I) was defined as 75% or higher, normal SjvO_2 (Group II) as 56 to 74%, and low SjvO_2 (Group III) as 55% or lower. These categories refer to the

mean SjvO_2 for the entire period of monitoring for each patient.

Outcome at 6 months was assessed using the Glasgow Outcome Scale (GOS).¹¹ Outcomes were classified into three groups: 1) dead or persistent vegetative state; 2) severe disability; and 3) moderate disability or good recovery. When a 6-month outcome was not available, the outcome at the last known contact (usually 3 months) was used.

Statistical Analysis

All summary data are expressed as the mean $\pm$ standard deviation or median (25th and 75th percentiles). For the summary data, the average of variables from the entire period of monitoring was calculated for each patient.

The distributions of the cerebral hemodynamic/metabolic variables are shown in the figures as box plots. The horizontal line across the box marks the median, and the lower and upper ends of box indicate the 25th and 75th percentiles. The error bars mark the 10th and 90th percentiles.

The median values of the physiological measurements were compared by analysis of variance on ranks, followed by Dunn's test when multiple comparisons were made. Differences in proportions were analyzed using the chi-square test.

Results

Demographic Characteristics

The 450 patients studied had a median age (25th and 75th percentiles) of 30 years (range 23–41 years) and a median Glasgow Coma Scale²⁸ (GCS) score of 7 (range 4–8) on the 1st day postinjury. Eighty-six percent of the patients were men. Eighty-nine percent of the patients presented with a closed head injury (diffuse brain injury 24.6%, contusion or intracerebral hematoma 29.8%, subdural hematoma 28%, and epidural hematoma 6.7%). A penetrating injury was present in the other 11%. At 6 months postinjury, a good recovery or moderate disability had been achieved in 42% of patients, severe disability was present in 24.4%, and 33.6% remained in a vegetative state or were dead.

Distribution of SjvO_2 Measurements

In the 450 patients 2977 individual measurements of CBF and cerebral metabolism were made (average of 6.6 measurements per patient) during Days 1 to 10 postinjury. The average SjvO_2 for each patient was calculated from these individual values. Figure 1 *upper* illustrates the distribution of these average SjvO_2 values for the 450 patients, and the limits that were used to divide the patients into Groups I, II, or III. Elevated mean values for SjvO_2 were seen in 86 (19.1%) of 450 patients, 342 (76%) had a normal SjvO_2 , and 22 (4.9%) had a low SjvO_2 .

The distribution of the individual measurements of SjvO_2 was similar (Fig. 1 *lower*). Of the 2977 individual measurements of SjvO_2 , 830 (28%) were 75% or higher, 1875 (63%) were between 56% and 74%, and 272 (9%) were 55% or lower.

Figure 2 shows the change in the distribution of the

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FIG. 1. Bar graphs showing the distribution of the SjvO₂ values among the 450 patients (upper) and distribution of the 2799 individual measurements of SjvO₂ (lower), with division into three groups with low, normal, and high SjvO₂ values.

SjvO₂ measurements during the first 5 days postinjury. Most of the low SjvO₂ values occurred on Day 1 postinjury. In contrast, the number of high SjvO₂ values was lowest on Day 1 and increased steadily from Days 1 to 5. The percentage of the total SjvO₂ measurements that were 55% or less were 20.2%, 7%, 6.2%, 7.1%, and 6% on Days 1 to 5, respectively, whereas the percentages of the total SjvO₂ measurements that were 75% or higher were 16.8%, 27.2%, 27.9%, 28.4%, and 30.3% on Days 1 to 5, respectively.

The individual SjvO₂ values were compared with the simultaneous measurements of CBF and CPP, and when all samples were examined, SjvO₂ was not significantly correlated with either of these measures. The high SjvO₂ values ($\geq 75\%$) were distributed over a wide range of CBF values (6.7–195 ml/100 g/minute). Of importance, in the low CBF range, in which a compensatory increase in oxygen extraction might be expected to cause lower SjvO₂ readings, many high SjvO₂ values were observed. A wide spectrum of CPP values (20–119 mm Hg) was associated with elevated SjvO₂ values. More than half (432 [53.7%] of 805) of all high SjvO₂ readings occurred when CPP was 70 mm Hg or higher, which is often considered to be an important threshold for severely head injured patients.^{4,25}

Characteristics of the Three Groups

The demographic characteristics of the three SjvO₂ groups are summarized in Fig. 3 left. Age, admission GCS score, and type of injury were similar in the three groups of patients. There were no significant differences in the ICP, mean arterial pressure, or CPP (Fig. 4 left), and hemoglobin concentration and PaCO₂ did not differ significantly among the groups.

The neurological outcome, however, was strikingly dif-

FIG. 2. Bar graph showing the distribution of the SjvO₂ values during the first 5 days postinjury.

ferent in the three groups. Patients in both Groups I and III had significantly higher mortality rates, and there were smaller numbers of patients with favorable outcomes than in Group II (Table 1). The outcome was not significantly different between Groups I and III ($p = 0.103$).

The most important additional differences among the three groups were in the cerebral hemodynamic and metabolic variables (Figs. 5 and 6 left). The hemodynamic picture of patients in Group II was typical of severely head injured patients, with a CMRO₂, CMRG, and GMRL that were lower than normal, a normal CBF and CVR, and a lower than normal AVDO₂, AVDG, and AVDL. Values in Groups I and III varied significantly from this typical head injury pattern.

In Group I we found a higher CBF, CMRL, and LGI, and a lower CVR, CMRO₂, and AVDO₂ than in Group II. Patients in this group also tended to have a lower CMRG and a higher AVDL than those in Group II, but the differences were not significant when adjusted for multiple comparisons. The patients in Group III, in contrast, had a lower CBF and a higher AVDO₂ and AVDG than those in

FIG. 3. Boxplots depicting comparisons of demographic characteristics in the three SjvO₂ (left) and the three outcome groups (right). * = different from patients who died or became vegetative; † = different from patients who had a severe disability ($p < 0.05$). DBI = diffuse brain injury.

FIG. 4. Boxplots depicting comparisons of pressure variables in the three SjvO₂ (left) and the three outcome groups (right). * = different from patients who died or became vegetative; † = different from patients who had a severe disability ($p < 0.05$).

Group II. The CMRO₂, CMRG, and CMRL values were not significantly different in Groups II and III.

Relationship of CBF and Cerebral Metabolic Parameters to Outcome

Outcome in All Patients. The relationship of outcome to the demographic characteristics and the CBF and cerebral metabolic variables are shown in Figs. 3 to 6 right. The patients who recovered to a GOS score of good recovery or moderate disability were younger and had a higher admission GCS score than the patients who died or became vegetative. Gender distribution and type of injury were not significantly different among the outcome groups. The patients who attained a favorable outcome also demonstrated a higher CMRO₂ than those who were severely disabled and those who died or became vegetative. They had a lower ICP than the patients who died or became vegetative, and a lower mean arterial blood pressure and CVR than the patients who recovered with a severe disability. The CBF tended to be higher in the patients who had a good outcome, but the difference was not quite statistically significant ($p = 0.085$).

Outcome in Group I. Twenty-two (25.6%) of the patients in Group I had a favorable outcome, defined as a good recovery or moderate disability. Twenty-two (25.6%) re-

TABLE 1

Glasgow Outcome Scale scores in 450 severely head injured patients categorized into three SjvO₂ groups*

Parameter	Group I	Group II	Group III
no. of patients	86	342	22
GOS score†			
good recovery/moderate disability	22 (25.6)	161 (47.1)	7 (31.8)
severe disability	22 (25.6)	85 (24.8)	3 (13.6)
persistent vegetative state/dead	42 (48.8)	96 (28.1)	12 (54.6)

* Numbers in parentheses are percentages. See text for definition of groups.

† $p < 0.001$ according to the chi-square test.

FIG. 5. Boxplots depicting comparisons of blood flow variables in the three SjvO₂ (left) and the three outcome groups (right). Left: * = different from patients who had a normal SjvO₂ ($p < 0.05$). Right: * = different from patients who died or became vegetative ($p < 0.05$). The highlighted area shows the normal range for CBF.

covered with a severe disability. Forty-two (48.8%) died or entered a persistent vegetative state. As illustrated in Figs. 3 to 6 right, there were significant differences in the demographic and cerebral hemodynamic/metabolic characteristics among these outcome groups.

The patients in Group I who had a favorable outcome were younger than the patients who were severely disabled and those who died or became vegetative. The patients in Group I with a favorable outcome had more diffuse injuries, and more intracranial hematomas were found in patients with a severe disability and those who died or became vegetative. The patients with a favorable outcome had a lower ICP than those who died or became vegetative. The patients in Group I who had a favorable outcome tended to have a higher CBF and a lower CVR than the patients who had an unfavorable outcome. The patients in whom a favorable outcome was achieved had

FIG. 6. Boxplots depicting comparisons of cerebral metabolism variables in the three SjvO₂ (left) and the three outcome groups (right). Left: * = different from patients who had normal SjvO₂ ($p < 0.05$). Right: * = different from patients who died or became vegetative; † = different from patients who had a severe disability ($p < 0.05$). The highlighted areas mark the normal ranges.

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a significantly higher CMRO₂ and a significantly greater cerebral lactate production than the patients in Group I who became vegetative or died.

Discussion

Monitoring of SjvO₂ is commonly used to judge the adequacy of cerebral oxygenation because it provides an assessment of the overall balance between cerebral metabolism and CBF. A greater than normal SjvO₂ is often interpreted as indicating that the CBF is more than adequate to satisfy cerebral metabolic requirements. This study raises the possibility that, in many cases, this interpretation may be incorrect and that an elevated SjvO₂ should not be automatically equated with hyperemia. Instead, as diagrammed in Fig. 7, an elevated SjvO₂ appears to be a heterogeneous condition. If the cause of the high SjvO₂ is primarily an increase in CBF and not a major decrease in CMRO₂, the outcome is generally favorable. Patients with these symptoms are often young and have a less severe head injury with mild, if any, intracranial hypertension. However, if the cause of the high SjvO₂ is a decrease in the CMRO₂, especially to < 1.5 ml/100 g/minute, the outcome is usually poor. The cause of the reduced CMRO₂ is either an alteration in cerebral oxygen extraction capabilities or the presence of ischemic regions in the brain. Patients with such symptoms are often older and have severe intracranial hypertension.

Comparison With Other Studies of SjvO₂ and CBF

On the surface, the data reported here may seem to conflict with previously published studies. Most of these apparent conflicts arise because an elevated SjvO₂ cannot always be equated with an elevated CBF.

Our data demonstrated a significantly worse outcome in patients who had an elevated SjvO₂, whereas many previous studies have emphasized a worse outcome in patients in whom a reduced CBF and episodes of reduced SjvO₂ were found.^{9,13,20} This difference probably stems from the fact that a high SjvO₂ did not always indicate an elevated CBF. A reduced CMRO₂ was more commonly the cause of the high SjvO₂. In the previous studies, CMRO₂ was an important covariate in the relationship between CBF and outcome, and within each CBF level (low, normal, elevated), there was a demonstrable impairment in outcome following decreases in CMRO₂.²¹

We observed no correlation between CBF and SjvO₂. In another study a significant nonlinear relationship has been shown between CBF and AVDO₂, and between CBF and SjvO₂.²⁰ However, this relationship was only present when patients with increased cerebral lactate production were excluded. When all patients were examined, the same poor relationship between CBF and AVDO₂ and between CBF and SjvO₂ was observed.

Our data indicated that, as a group, patients with a high SjvO₂ have a lower CMRO₂ than patients with a normal or low SjvO₂. In other studies, a high CBF has been associated with a relatively higher CMRO₂.²⁰ This difference is probably seen because SjvO₂ can be elevated because of a high CBF or because of a low CMRO₂, and in most of the cases in the present series a high SjvO₂ was associated with a low CMRO₂.

FIG. 7. Diagram outlining the interpretation of SjvO₂ data suggested by the present study.

We found that there was no difference in ICP among the three SjvO₂ groups; within Group I, ICP was significantly higher only in the patients who died. This same pattern was observed in the entire group of 450 patients. Some investigators have reported a higher incidence of intracranial hypertension in patients who have an elevated CBF.^{12,19} Other investigators have not found a significant relationship between intracranial hypertension and the level of CBF.^{18,20}

In many other respects, our data are entirely consistent with previous reports. We found that a reduced CBF and SjvO₂ were most common during the first 24 hours postinjury. An elevated SjvO₂ was more common after the 1st day. These data are consistent with previous work showing that hyperemia commonly occurs in the days following head injury,¹⁹ and they are also consistent with those reported in other studies of CBF early after injury.^{2,3} Martin, et al.,¹⁵ also described a characteristic CBF pattern of initial hypoperfusion followed by a period of hyperemia and then a period of vasospasm after head injury.

An SjvO₂ of 75% or higher was not always related to an adequate cerebral perfusion. In fact, elevated SjvO₂ was associated with a very wide range of CBF and CPP values. The CPP and/or CBF values that were normal or even elevated were by themselves insufficient to guarantee the adequacy of cerebral oxygen metabolism. It has been shown that cerebral hemodynamic and metabolic variables in traumatic brain injury do not necessarily correlate with CPP. In a porcine model of cryogenic brain injury, Zhuang, et al.,³⁰ found persistent posttraumatic ischemia despite normalization of CPP. They proposed a significant increase in CVR as the cause of the ischemia.

A relationship between clinical condition, CMRO₂, and outcome after traumatic coma has been noted by several authors.^{10,17-19,26} Shalit, et al.,²⁶ found that CMRO₂ values below 1.4 ml/100 g/minute during coma were incompatible with recovery of consciousness. In the series of Roquefeuil, et al.,²⁴ the lowest CMRO₂ value consistent with recovery was 1.5 ml/100 g/minute. Jaggi, et al.,¹⁰ reported that AVDO₂ values obtained during the 1st week postinjury were indicative of hyperemia significantly more often in patients who died or remained vegetative than in the group with a 6-month outcome of severe disability, moderate disability, or good recovery. Yukota, et al.,²⁹ demonstrated that very high SjvO₂ readings (> 90%) were associated with the poorest outcome. Muizelaar, et al.,¹⁸ demonstrated a greater degree of hyperemia at deep-

er levels of coma (except for patients with a GCS score of 3), indicating uncoupling of CBF and metabolism beginning on the 1st day postinjury. In a recent study, Cruz⁸ found higher mortality rates in patients who had an initial decrease in cerebral oxygen extraction and suggested that an initial increase in oxygen extraction was indicative of neuronal viability in patients with traumatic diffuse swelling. Robertson, et al.,²² measured CMRO₂ and CBF in 44 comatose head-injured patients to determine if metabolic changes could be used to identify the patients who would develop cerebral infarction. A CMRO₂ of less than 1.3 ml/100 g/minute was rare in patients who had no ischemic injury but occurred at some time in all patients who developed an infarction.

Implications for Management

Determining the adequacy of cerebral perfusion in individual comatose patients remains difficult. Defining ischemia only in terms of CBF and oxygen supply overlooks the importance of the functional state of the tissue. These results may have some important implications for the management of head-injured patients. Posttraumatic alterations in CBF are often heterogeneous. Regional CBF may be simultaneously increased in some parts of the brain and decreased in others. It is therefore possible that attempts to control elevated ICP by maneuvers that globally reduce CBF might actually produce focal areas of relative ischemia. Unless patients are adequately monitored, significant potential hazards may accompany such therapies as vasoconstrictor administration and profound hyperventilation.^{6,14}

In some cases, the SjvO₂ may appear normal although brain parenchyma are actually suffering from hypoperfusion, in which case augmentation of an already supranormal CBF may be required. Factors underlying such a phenomenon may include the need to overcome cytochrome inhibition, to provide an increased oxygen diffusion gradient to meet cerebral metabolic demands, and/or to ensure adequate CBF to unaffected brain regions or to injured brain in those cases in which no excessive perfusion occurs.

Unfortunately there is no hemodynamic or oxygen transport-related variable that directly reflects the adequacy of the oxygen supply at the tissue and cellular level. Macrocirculatory oxygen transport can be measured, but it is the microcirculation that is the major determinant of tissue oxygen delivery. Only at this level can one assess whether therapeutic interventions actually improve the cellular oxygen supply. Further studies of elevated SjvO₂ as an index of impaired oxygen extraction should help clarify these issues.

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