

Cerebral Oxygen Desaturation with Normal ICP and CPP in Severe TBI

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Abstract: *Introduction:* Standard monitoring of severe traumatic brain injury patients (TBI) by intracranial pressure (ICP) and cerebral perfusion pressure (CPP) monitoring fails to recognize episodes of cerebral oxygen desaturation. We found and characterized frequent episodes of desaturation of jugular venous oxygen (SjO₂) in the face of normal ICP and CPP.

Methods: Fifty six patients with severe TBI had SjO₂ and ICP monitors placed. The charts were retrospectively reviewed and all episodes of desaturation were recorded and characterized.

Results: Nineteen patients had episodes of desaturation with normal ICP and CPP. The average GCS score was 5.8. 63% of desaturations occurred in the first 24 hours, 17% of desaturations occurred in 24-48 hours, and 20% occurred in 48-72 hours. The depth of desaturation was 50-54% in 50% of instances, 45-49% in 37% of instances, and 40-44% in 13% of instances. The duration of the desaturation episodes was less than 10 minutes in 47%, 10-30 minutes in 17%, 30-60 minutes in 23%, and greater than 50 minutes in 13%. Treatment of the desaturation was elevation of FIO₂ in all patients, elevation of pCO₂ in 15 patients, volume expansion in 9 patients, pressors in 9 patients, and Propofol in 5 patients.

Conclusions: The monitoring of severe TBI patients with ICP and CPP alone is insufficient to recognize cerebral oxygen desaturation episodes in 34% of patients. The monitoring of SjO₂ facilitates the recognition and treatment of these episodes.

Keywords: Traumatic brain injury, intracranial pressure monitoring, cerebral perfusion monitoring, jugular venous oxygen saturation, cerebral ischemia, cerebral oxygen monitoring.

INTRODUCTION

The treatment of traumatic brain injury (TBI) has advanced considerably over the last decade. The publication of "The Guidelines for the Treatment of Severe Head Injury", published by the AANS/BTF in 1995 and last revised in 2007 established the scientific basis for modern TBI care [1]. In the Guidelines it was noted that hyperventilation should not be used without monitoring brain oxygenation because of the risk of cerebral hypoxia. The regular use of jugular bulb catheters in our patients to monitor the jugular oxygen saturation (SjO₂) led to a better understanding of the occurrence and incidence of cerebral desaturation. In particular, desaturation episodes were seen independent of elevations in intracranial pressure (ICP) with normal cerebral perfusion pressure (CPP). We reviewed all of our monitored patients to further characterize this phenomenon.

MATERIALS AND METHODS

All patients were cared for at Mission Hospital and Regional Medical Center, an ACS Level II trauma center. Inclusion criteria were: Glasgow Coma Scores (GCS) of 3-8, non-penetrating head injuries, and placement of ICP and SjO₂ monitors. There were a total of 56 patients with severe TBI who received ICP and SjO₂ monitors. A retrospective review of the prospectively collected data was undertaken for the first three days of hospitalization. This revealed that 19 patients had episodes of desaturation with normal ICP

and CPP. There were 18 males and one female. The age ranged from 15-74 years with an average age of 40. The Glasgow Coma Score (GCS) ranged from 3 to 14 with two patients, with GCS of 10 and 14, deteriorating to lower levels in the first 24 hours. The average GCS was 5.8 with a median GCS of 6. The injury severity score ranged from 16 to 50 with an average score of 30 and a median score of 26.

All patients were admitted on the trauma service. The patients went to surgery within 24 hours of admission for placement of ventriculostomies, sometimes in conjunction with other decompressive procedures. The ventriculostomies were either Camino bolts (Integra NeuroSciences, Plainsville, NJ) or Codman ventricular catheters (Codman, Raynam, MA). Jugular bulb catheters (Abbott Laboratories, Chicago, IL) were placed by the trauma surgeons in the operating room or in the intensive care unit (ICU) utilizing fluoroscopic and ultrasound guidance. All patients were treated using a Severe TBI protocol based on the Guidelines integrated with SjO₂ monitoring. Craniectomies were performed when clinically indicated in 12 patients: 7 day one and 6 day 2-3 (one patient receiving an early and a late craniectomy). ICP and SjO₂ were monitored for 3-22 days (average 11, median 10). Functionality of the SjO₂ system was monitored continuously to assess catheter occlusion or malposition, and accuracy was confirmed at least once every 12 hours by sending a venous sample to the laboratory. Ventilator days ranged from 3-58 days (average 22, median 20). ICU length of stay was 3-60 days (average 24, median 22). Hospital length of stay was 3-78 days (average 28, median 20). Hospital charges ranged from \$66,104 to \$743,046. Mechanism of injury was: motor vehicle accident 37%, fall

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37%, bike vs auto 16%, skateboard 5%, pedestrian vs auto 5%. Radiological findings were: cerebral edema 13, contusion 10, subdural hematoma 9, skull fracture 2, traumatic subarachnoid hemorrhage 1.

RESULTS

There were 56 patients who fit the initial selection criteria. Normal SjO₂ levels (55%-75%) were found in 30 patients (54%). In this group 13 patients (23%) had low ICP and 17(31%) had high ICP. Low SjO₂ and high ICP (>20) were found in 7 patients (13%). Low SjO₂ and low ICP and normal CPP were found in 19 patients (34%). It is this group of 19 patients with low SjO₂ in the face of normal ICP and CPP that we further characterized. One patient example is noted in Fig. (1).

The timing of the occurrences of desaturations was tabulated (Fig. 2). 63% of desaturations occurred in the first 24 hours. 17% of desaturations occurred in 24-48 hours and 20% occurred in 48-72 hours. The severity of desaturations below 55% was tabulated (Fig. 3). The depth of desaturation was 50-54% in 50% of instances, 45-49% in 37% of instances, and 40-44% in 13% of instances. The duration of desaturations was tabulated (Fig. 4). The duration of the desaturation episodes was less than 10 minutes in 47%, 10-30 minutes in 17%, 30-60 minutes in 23%, and greater than 50 minutes in 13%. The interventions most commonly utilized for treatment of the desaturation were elevation of FIO₂ in all patients, elevation of pCO₂ in 15 patients, volume expansion in 9 patients, pressors in 9 patients, and Propofol in 5 patients. GOS scores were: GOS 4&5-68.4%, GOS 2&3-15.8%, and GOS 1-15.8%.

Craniectomies had been utilized for the control of ICP in 12 of the 19 patients (63%). 7 patients had early craniectomies associated with the removal of an acute subdural hematoma. Six patients required a delayed craniectomy for delayed elevations in ICP. One patient required both an early and a late craniectomy.

DISCUSSION

Measures for decreasing the impact of TBI have been aimed at prevention of primary injury as well as prevention of secondary injury [1-4]. An extensive body of literature supports the importance of adequate monitoring and treatment to avoid secondary injury related to hypotension, hypoxia, and elevated ICP [2, 5-10]. Marion, *et al.*, in 1991, showed that cerebral blood flow was reduced after TBI in the first 12 -24 hours [8]. Bouma, *et al.*, in 1992, showed that there was a 31.4% incidence of ischemia in the first 3.1h +/- 2.1h after injury [11]. Chestnut, *et al.*, in 1993, demonstrated that hypotension (BP_{sys} < 90 mmHg) was associated with an increase in morbidity and mortality [3]. Robertson, *et al.*, saw an increase in mortality in patients suffering episodes of cerebral oxygen desaturation [12].

In adopting the Guidelines into our clinical practice we established clinical protocols for the bedside management of the severe TBI patient. These have been previously published [13]. They incorporated The Guidelines and cerebral oxygen monitoring utilizing jugular bulb catheters to measure oxygen saturation (SjO₂). The initial results of this work showed an improved outcome with decrease in mortality (GOS 1) from 43% to 27%, a decrease in severe disability and vegetative state (GOS 2 & 3) from 30% to 11%, and an

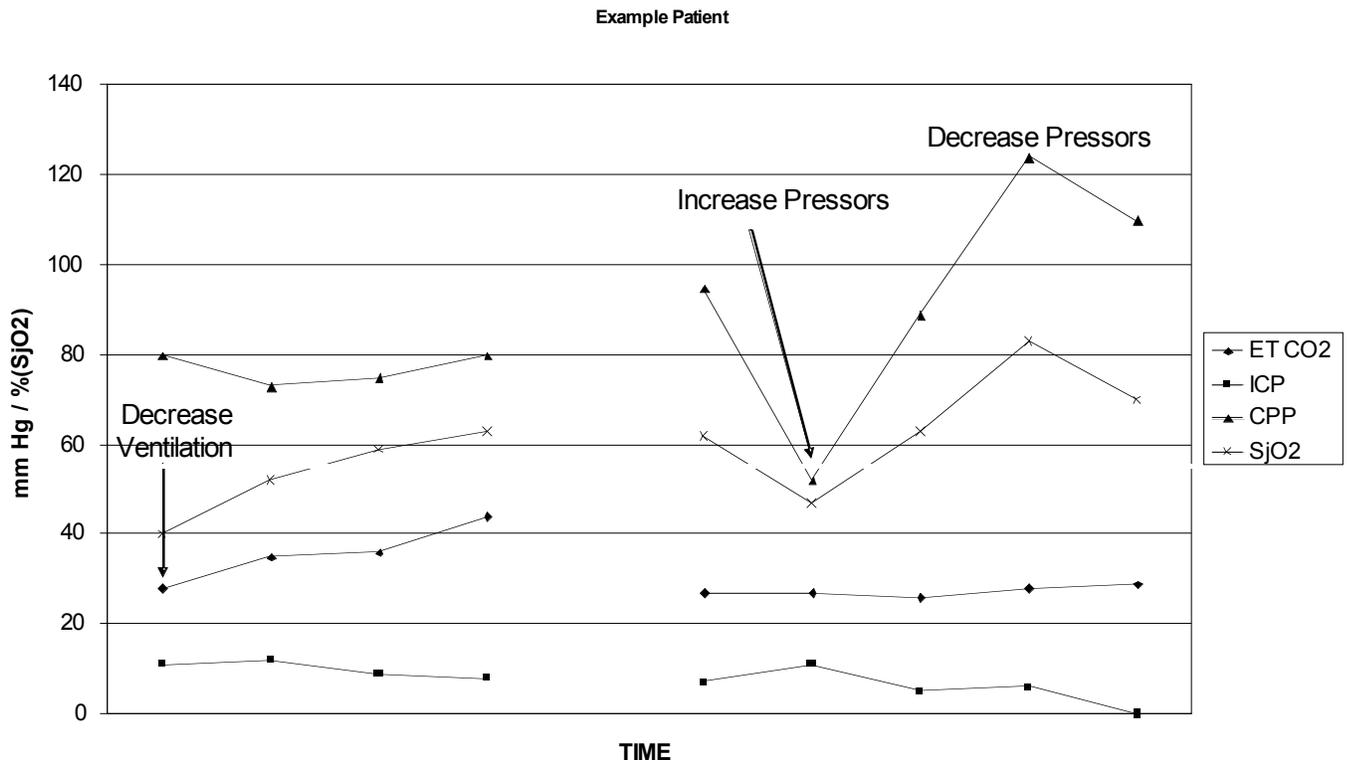


Fig. (1).

Timing of Desaturation

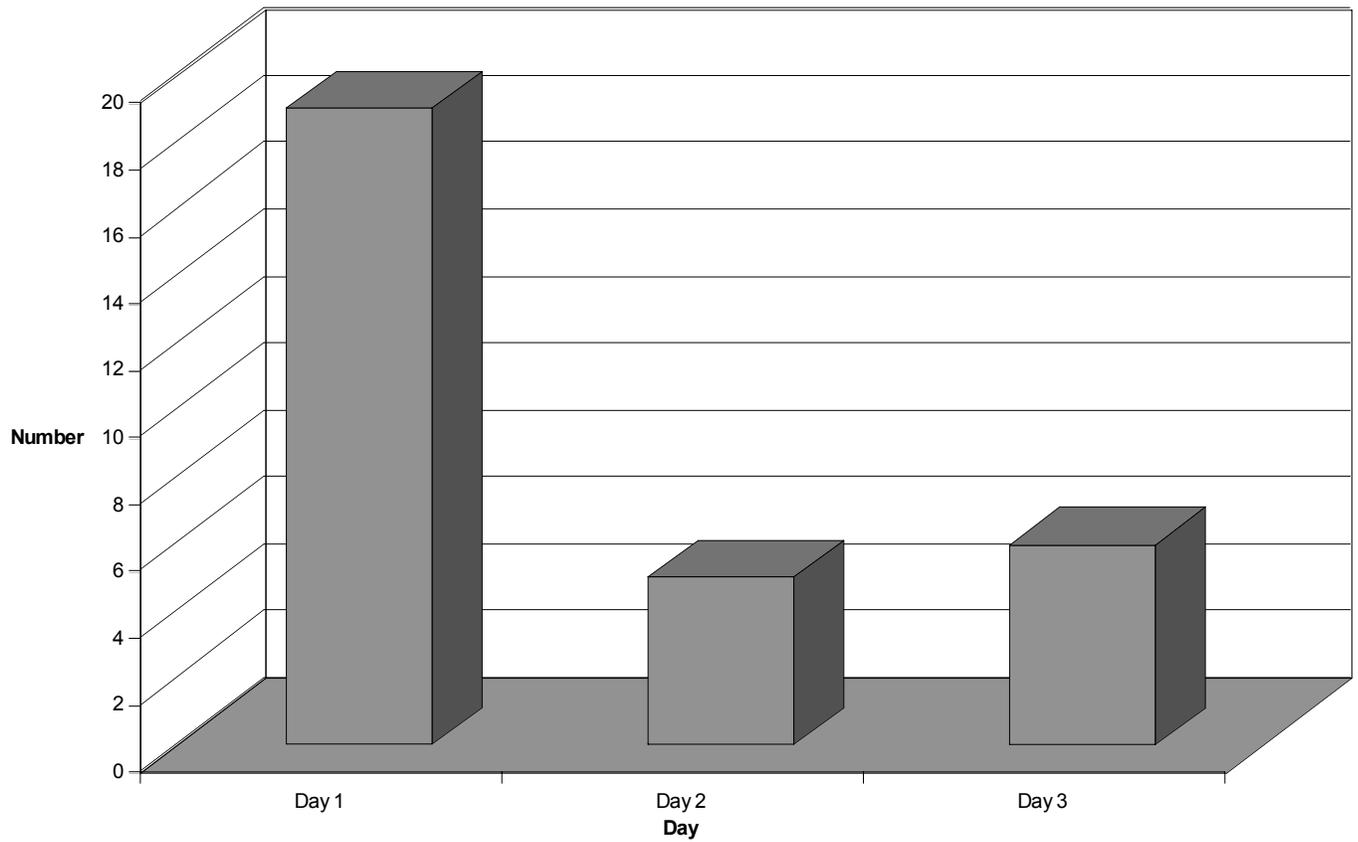


Fig. (2).

Depth of Desaturation

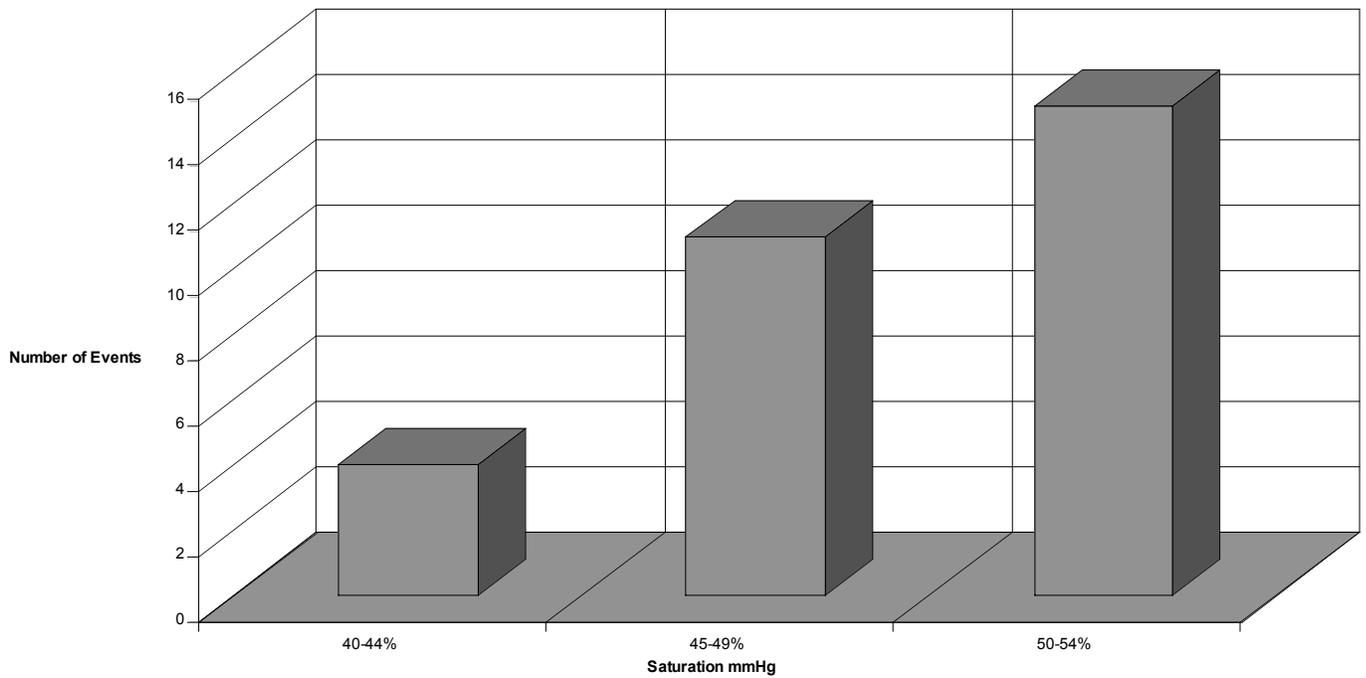


Fig. (3).

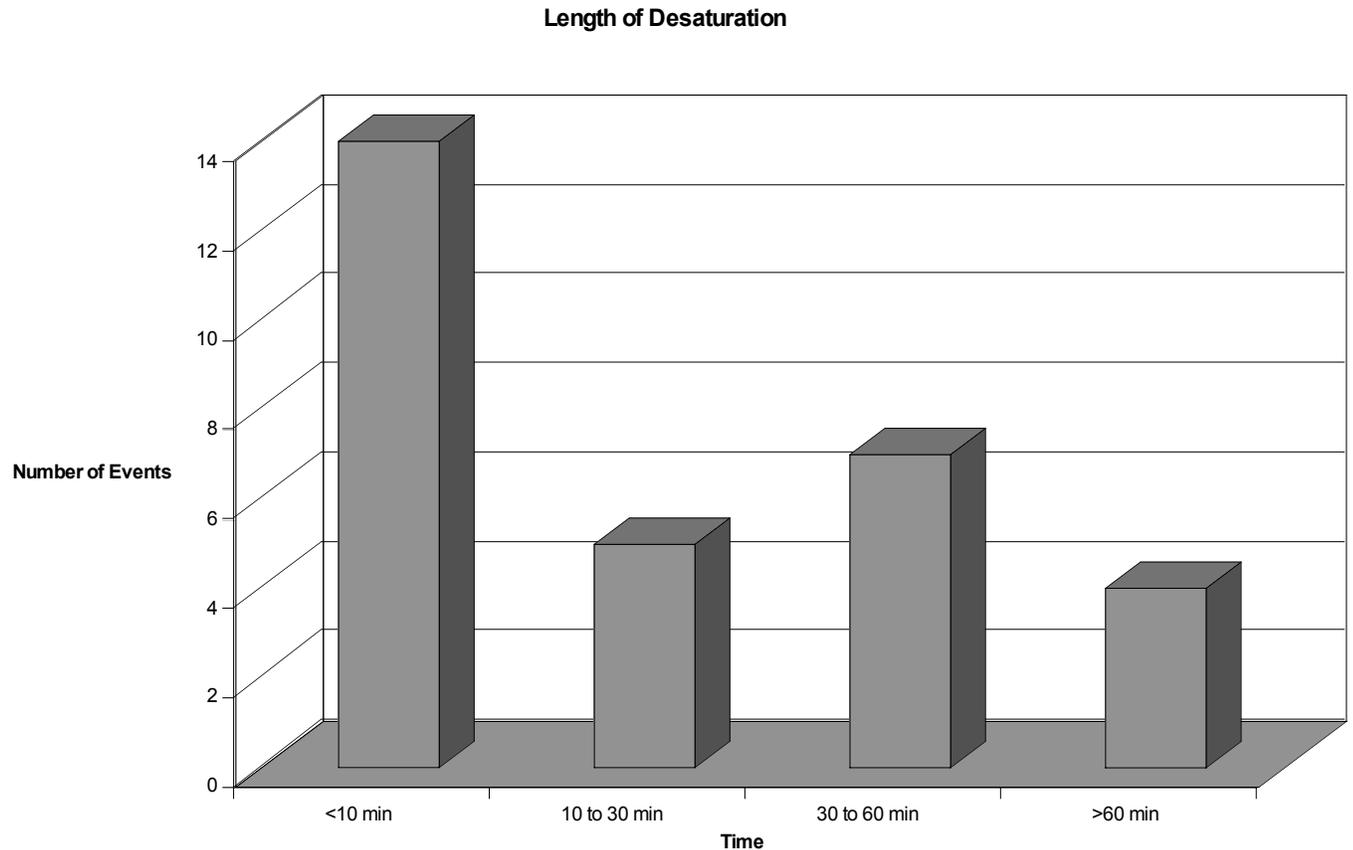


Fig. (4).

increase in good and moderate disability (GOS 4 & 5) from 27% to 43% [13]. These numbers have been stable at last evaluation December 2006, $n = 193$, with GOS 1-13%, GOS 2 & 3-14.5%, and GOS 4 & 5-72.5% (recent experience has included the use of intraparenchymal oxygen monitoring).

The adequacy of using ICP and CPP monitoring alone to direct clinical decision making has been brought into question in several studies. Graham, *et al.*, in 1989, wrote that ischemic cell changes were found in 70% of brains following fatal severe TBI without clinical elevations in ICP [14]. van Santbrink, *et al.*, in 1996, followed cerebral oxygenation in 22 severe TBI patients [15]. They looked at the correlation with multiple factors including ICP, CPP, SjO₂, PbtO₂, among others. They found a weak correlation between cerebral oxygenation and either ICP or CPP. Using parenchymal oxygen monitoring, Harl, Artue, van den Brink, and Stochetti explored the causes of decreased cerebral oxygenation. Harl, *et al.*, in 1997, found that mannitol could control elevated ICP but had no effect on cerebral oxygenation in eleven patients [4]. They concluded that cerebral oxygenation should be followed separately from ICP. Artu, *et al.*, in 1998, demonstrated that despite CPP in the therapeutic range that cerebral hypoxia was seen [16]. Increasing the CPP to supra-therapeutic levels could restore adequate oxygenation. This suggested that CPP monitoring alone was inadequate in avoiding cerebral hypoxia. Van den Brink, in 2000, demonstrated that early inadequate oxygen metabolism was seen in spite of aggressive treatment of ICP and CPP [17]. They found that cerebral oxygenation was an independent predictor of outcome. Stochetti *et al.* found in 9 patients with focal cerebral contusions that several had ab-

normal PbtO₂ in spite of the ICP and the CPP being in the normal range [18].

SjO₂ monitoring has proven useful in better understanding the consequences of severe TBI. Shoon, *et al.*, in 2002, looked at 116 severe TBI patients without elevated ICP on admission and found that the incidence of development of elevated ICP was significantly higher in patients with abnormal SjO₂ [19]. The common causes were hyperventilation (40.7%), hypovolemia 28.4%, and anemia (21%). They postulated that, "Early detection of disturbances in oxygen supply-demand relationship and prevention or resolution of the secondary insults which produce these disturbances, might lead to a reduction in the incidence of intracranial hypertension." In 2001, Cuza, *et al.*, stated that, "Episodes of desaturation are frequent in neurocritical patients and are associated with increased mortality" [20].

Low cerebral oxygen is significantly correlated with poor outcome as shown by several authors utilizing parenchymal oxygen monitoring, including: van den Brink, Bardt, and Dings. van den Brink, in 2000, looked at 101 patients with severe TBI and found that hypoxia was an independent predictor of unfavorable outcome and death [17]. Barth *et al.*, in 1998, reported that severe hypoxia for greater than 30 minutes resulted in substantially worse outcome than hypoxia limited to under 30 minutes [21]. Dings *et al.*, in 1998, reported that patients with lower cerebral oxygenation showed lower Glasgow Outcome Scores (GOS) at 6 months [22]. Stiefel *et al.*, showed significant improvement in patient outcomes when they added oxygen monitoring to their TBI treatment protocol [23].

In our study of 56 patients, 19 patients (34%) had episodes of cerebral desaturation in spite of normal ICP. CPP was also above 60mmHg in all but one patient where it fell briefly to 50 (Fig. 1). The fact that ICP and CPP measurements failed to effectively predict cerebral oxygen desaturation left 34% of patients at risk for secondary hypoxic brain injury. This demonstrates the need for multimodality monitoring of severe TBI patients to include some form of cerebral oxygen monitoring. Latronico, *et al.*, found that in 34 of 319 observations that SjO₂ was abnormal in the face of normal ICP and CPP [24]. They did not modify treatment based on SjO₂ abnormalities alone, but did treat if a low SjO₂ was associated with hypovolemia or hypocapnia. Contrary to our conclusions, they concluded that intermittent SjO₂ monitoring was not clinically useful.

An analysis of the timing, depth, and duration of desaturation episodes demonstrates several importation points of clinical relevance. The episodes occur early, are variable in depth, and are of short duration. Real time monitoring is of critical importance to allow the timely institution of treatments to correct the desaturations before secondary injury can occur. The use of critical thinking algorithms allows the caregiver at the bedside to institute appropriate corrective actions in real time without delays, which could result by the need to contact other medical care providers not at the bedside [25]. This empowerment of nursing and other bedside personnel accelerates the treatment of the patient while relieving the Neurosurgeon/Intensivist of frequent calls to handle issues adequately covered by protocol and competent bedside caregivers.

CONCLUSION

The monitoring of severe TBI patients with ICP and CPP alone is insufficient to recognize cerebral oxygen desaturation episodes in 34% of patients. The monitoring of SjO₂ facilitates the recognition and treatment of these episodes. Critical thinking algorithms are invaluable in assisting in the real time management of these patients.

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