The Impact on Outcomes in a Community Hospital Setting of Using the AANS Traumatic Brain Injury Guidelines

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Background: **Traumatic brain injury poses a serious public health challenge. Treatment paradigms have dramatically shifted with the introduction of the American Association of Neurologic Surgeons (AANS) *Guidelines for the Management of Severe Head Injury*. Implementation of the AANS guidelines positively affects patient outcomes and can be successfully introduced in a community hospital setting.**

Methods: **Data were collected both retrospectively and prospectively from the records of all trauma patients between 1994 and 1999. A cohort of 93 patients was selected. Thirty-seven patients were treated before the implementation of the AANS guidelines, and these were statistically compared with 56 patients treated after the implementation of the guidelines.**

Results: **Implementation of the recommendations in the AANS guidelines in a standardized protocol resulted in a 9.13 times higher odds ratio of a good outcome relative to the odds of a poor outcome or death compared with a group managed before the practice change. A Glasgow Coma Scale (GCS) admission score > 8 was associated with a 6.58 times higher odds ratio of a good outcome compared with a GCS admission score ≤ 8. Odds ratio of a good outcome decreased by a factor of 0.92 for each year increase in age of patients starting at age 9. A dedicated neurotrauma team and comprehensive treatment algorithms are critical elements to this success. Hospital charges increased by more than $97,000 per patient, but are justifiable in the face of significantly improved outcomes.**

Conclusion: **Implementation of a traumatic brain injury protocol in a community hospital setting is practical and efficacious. Appropriate invasive monitoring of systemic and cerebral parameters guides care decisions. The protocol results in an increase in resource usage, but it also results in statistically improved outcomes justifying the increase in expenditures.**

Key Words: **Traumatic brain injury, Intracranial pressure, Neurotrauma, Sjo_2 monitoring.**

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Traumatic brain injury (TBI) continues to pose a serious public health challenge: 50,000 deaths occur each year and 80,000 to 90,000 individuals sustain long-term disability.[1] Approximately 500,000 hospital admissions to acute care hospitals occur each year from head injury, and 10% of these cases are severe (Glasgow Coma Scale [GCS] score of 3–8). The mortality rate in this group remains high at approximately 25%, with poor outcomes, Glasgow Outcome Scale scores of 1 to 3, approximating 50%.[2–4]

For decades, the traditional therapy for severe TBI has been directed toward intracranial pressure (ICP) management. Elevations in ICP, related to severe TBI, have been correlated with poor outcome.[3,4] The mainstays of this therapy included intubation with hyperventilation, decompression of mass lesions, mannitol, dehydration, ventriculostomy with cerebrospinal fluid (CSF) drainage, and possibly barbiturate coma. Recent laboratory and clinical research has called into question this longstanding treatment paradigm.[5–7] Therapies focusing primarily on ICP reduction may have placed the brain at risk for secondary injury because of a failure to account for the effects of this therapy on cerebral blood flow (CBF) and cerebral oxygenation.

Failure to implement the TBI guidelines and wide variation in management schema hampered rational treatment of patients with TBI.[6,8] TBI management protocols help achieve "best practice" goals. A large number of TBI patients are treated outside of university and research centers. It is imperative that community-based treatment of TBI, to be effective, must be rational and scientifically based. In a multidisciplinary effort at our community hospital trauma center, these guidelines and other relevant literature were adapted into a management protocol in June 1997 (Fig. 1). We believe that, with a dedicated trauma response team, this is eminently possible and that excellent results can be achieved.

The purpose of this article is to report the impact of implementing the recommendations made in the American Association of Neurologic Surgeons (AANS) *Guidelines for the Management of Severe Head Injury*[9] and related research regarding cerebral oxygenation and cerebral perfusion to clinical practice in a community trauma center. Data were collected retrospectively and prospectively. The data comparing the outcomes of severe TBI patients before and after implementation of protocols focused on a combined cerebral perfusion pressure (CPP), ICP, and cerebral oxygenation therapy paradigm. The results of the study have implications for practitioners in community hospital trauma centers.

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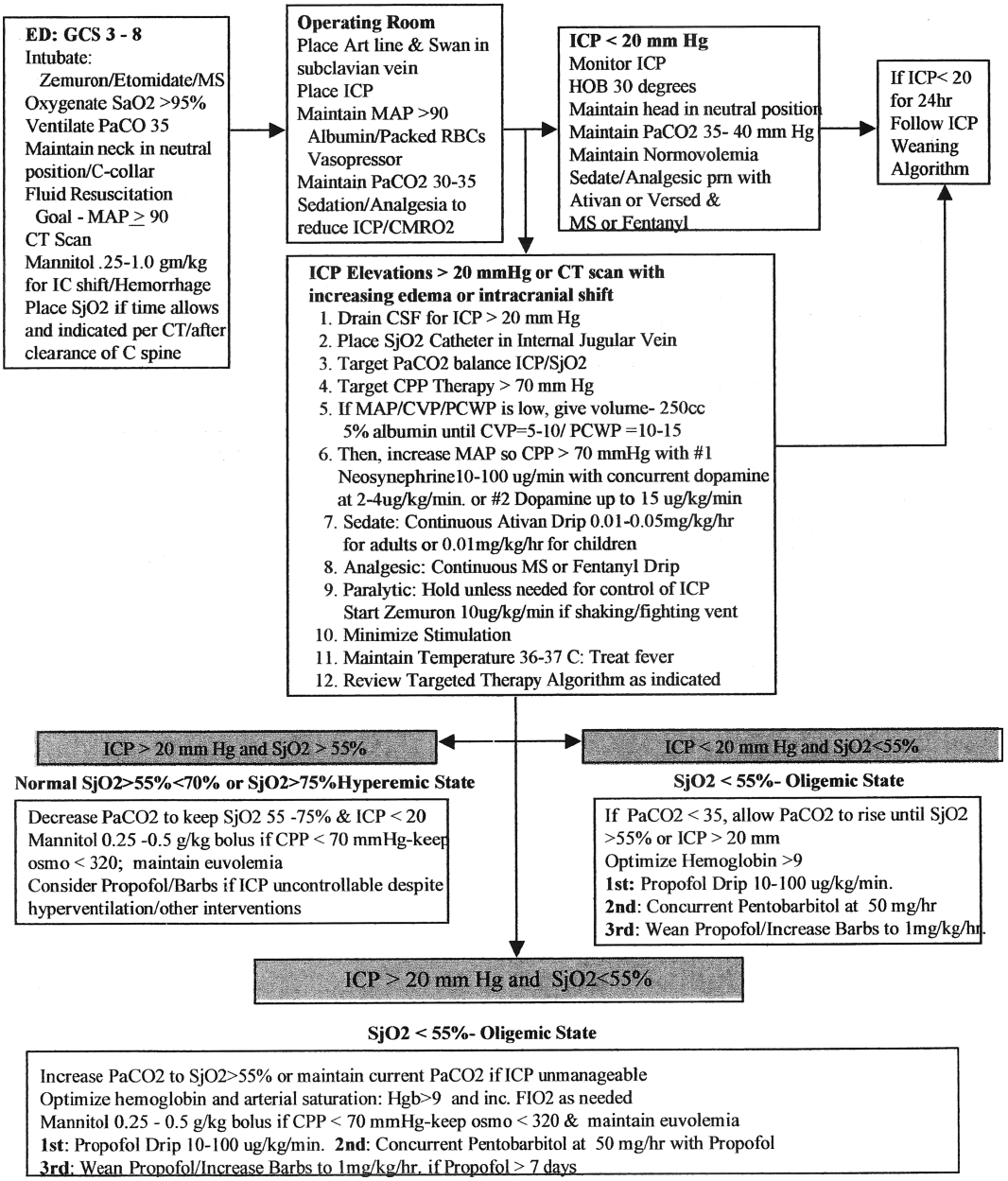

Fig. 1. *Management of severe head injury. (© Mission Hospital Regional Medical Center, Mission Viejo, California. Used with permission.)*

PATIENTS AND METHODS

Selection of Patients and Data Collection

Data were collected both retrospectively and prospectively from the records of trauma patients admitted to the Mission Hospital Regional Medical Center between 1994 and 1999. The data from patients, admitted from January 1994 until June 1997, before the implementation of the AANS TBI guidelines, and a clinical protocol for TBI was compared with the data from patients admitted from July 1997 until December 1999, after implementation of the protocol.

Inclusion criteria were as follows. Patients had to have an admission GCS score of 3 to 8 or deteriorate from a higher level in the first 48 hours. Computed tomographic (CT) scan had to demonstrate findings indicative of brain injury. Patients had to be ≥8 years of age. Age 8 was chosen because of the difficulty of placing Sjo_2 monitors in younger children. Patients had to have a closed head injury. Patients had to have an intracranial pressure monitor. The monitors were either Codman ventriculostomy catheters (Johnson & Johnson, Piscataway, NJ) attached to a manometric system or Camino fiberoptic systems (Integra Neurosciences, Plainsboro, NJ), at the discretion of the treating surgeon.

Exclusion criteria for patients were as follows: age < 8 years of age; penetrating head trauma; pronounced dead within 24 hours of admission; and GCS scores of 3 to 8 but without evidence on CT scan or clinical examination of brain

injury (i.e., systemic causes, positive drug screen, postictal state). The selection criteria resulted in the selection of a cohort of 93 patients. Thirty-seven patients treated before the implementation of the guidelines (pre-TBI guidelines group) and 56 patients treated after the implementation (post-TBI guidelines group). Six-month outcomes were divided into three groups using the Glasgow Outcome Scale: 1 (expired), 2 and 3 (severe disability/vegetative), and 4 and 5 (good outcome/moderate disability).

Treatment Protocols

Two groups of patients were studied. Group I, the pre-TBI guidelines group consisting of 37 patients admitted between January 1994 and June 1997, was managed with an emphasis on ICP reduction. Group II, the post-TBI guidelines group consisting of 56 patients admitted between June 1997 and December 1999, was managed with an emphasis on concurrent ICP reduction, CPP enhancement, and maximization of cerebral oxygenation. A collaborative practice team met and developed clinical guidelines for managing the severe TBI patients on the basis of the recommendations in the AANS TBI guidelines and supportive scientific literature, particularly as related to jugular O_2 saturation (Sjo_2) monitoring. The care was divided into phases and included individual algorithms for the emergency department (ED)/resuscitation/operative phase and the intensive care unit (ICU) phase (Fig. 1). Agreement was obtained among the trauma surgeons, neurosurgeons, anesthesiologists, intensivists, and ancillary personnel on implementing the recommended interventions identified in each phase. The group met at regular intervals to clarify any protocol issues. Patient goals were established for each phase optimizing $Paco_2$, Pao_2, mean arterial pressure (MAP), central venous pressure (CVP)/pulmonary capillary wedge pressure (PCWP), ICP, CPP, and Sjo_2.

Group I Management (Pre-TBI Guidelines Group)

On arrival to the ED, severe TBI patients were intubated with medications such as morphine sulfate, midazolam, and/or a paralytic. Patients were hyperventilated to a $Paco_2$ between 25 and 30 mm Hg. Systolic blood pressure (BP) was maintained $>$ 90 mm Hg. Fluids were kept at a minimum unless systemic bleeding was present. Mannitol was given to reduce cerebral edema. Radiographs of the chest and cervical spine were obtained, as was a CT scan of the brain. In the presence of a mass lesion or cerebral edema, the patient was rapidly transported to the operating room (OR) for surgery.

Patients were admitted to the ICU, where management priorities included intermittent sedation with morphine sulfate and midazolam; drainage of CSF for ICP $>$ 20 mm Hg; fluid restriction and use of mannitol (0.25–1.0 g/kg) for ICP $>$ 20 mm Hg uncontrolled with hyperventilation or CSF drainage; mechanical ventilation with support of arterial oxygenation ($Pao_2 > 90$ mm Hg) and hyperventilation ($Paco_2$ of 25–30 mm Hg); and barbiturate therapy for uncontrolled ICP. A minimum systolic BP of 90 mm Hg was maintained with the use of vasopressors but not fluids, and no minimum CPP was enforced. All patients received anticonvulsants. Nutritional support was supplied to all patients but not always early on in their ICU course.

Group II Management (Post-TBI Guidelines Group)

On arrival to the ED, patients were emergently intubated using a rapid-sequence intubation protocol including medications such as lidocaine, etomidate, rocuronium, morphine sulfate, and midazolam (Fig. 1). The $Paco_2$ was targeted at 35 mm Hg with an $Sao_2 > 95\%$. Circulatory status was assessed and enhanced with intravenous fluids such as normal saline and 5% albumin to maintain a MAP $>$ 90 mm Hg, and CVP 5 to 10 mm Hg/PCWP 10 to 15 mm Hg, to maintain euvolemia. Intravenous mannitol (1.0 g/kg) was administered in the presence of extensor/flexor posturing and/or change in pupils consistent with tentorial herniation. Fluid replacement was accomplished concurrently with diuresis from the mannitol, maintaining euvolemia. Chest and cervical spine radiographs and a rapid CT scan were accomplished within 30 minutes of admission. Rapid operative intervention followed the CT scan. Hemodynamic monitoring lines were placed in the OR, including placement of invasive arterial catheter, continuous cardiac output pulmonary artery catheter, and jugular venous oximetric catheter (Sjo_2) (Abbott Laboratories, Abbott Park, IL). OR phase goals were maintained (Fig. 1). Administration of intravenous propofol by anesthesia provided ICP reduction and reduction in cerebral oxygen use as well as anesthesia. A ventriculostomy ICP monitor was placed and other operative interventions were performed when indicated.

Once admitted to the ICU, the multidisciplinary team collaborated on interventions to maintain established goals. These goals included CPP $>$ 70 mm Hg; ICP $<$ 20 mm Hg; CVP 5 to 10 mm Hg/PCWP 10 to 15 mm Hg, Sjo_2 55% to 75%; and $Sao_2 > 95\%$. Interventions were prioritized on the basis of patient responses (Fig. 1). Because of the number of parameters to monitor and titrate, a group of ICU nurses in collaboration with the neuro clinical nurse specialist and neurosurgeons developed three critical thinking algorithms to stratify primary, secondary, and tertiary interventions (Fig. 1). The collaborative team strictly followed the algorithms and evaluated individual patient responses to each intervention. All patients received anticonvulsants. All patients received early aggressive nutritional support.

Statistical Methods

Student's *t* test was used to compare the mean values of various demographic and other variables across the two groups. A p value < 0.05 was the criterion chosen for ascertaining statistical significance. A χ^2 test was conducted to assess the significance of the association between outcomes and group membership. An ordinal logistic regression model was used to compute the odds of a good outcome versus the

Table 1 Descriptive Statistics for Demographic and Other Relevant Variables

Variable	Group	No. of Patients	Mean	SE
Age	Pre-TBI	37	41.35	±3.65
	Post-TBI	56	38.10	±2.50
ISS score	Pre-TBI	34 (3 missing)	32.82	±2.35
	Post-TBI	56	28.32	±1.20
GCS admission	Pre-TBI	37	6.43	±0.67
	Post-TBI	56	6.88	±0.50
ICP days	Pre-TBI	37	9.97	±0.88
	Post-TBI	56	10.96	±0.56
Ventilator days	Pre-TBI	37	17.46	±2.21
	Post-TBI	56	19.20	±1.60
ICU LOS	Pre-TBI	37	21.03	±2.70
	Post-TBI	56	22.00	±1.60
Total LOS	Pre-TBI	37	24.35	±3.21
	Post-TBI	56	25.40	±1.90
Charges	Pre-TBI	37	$196,128	±23,166
	Post-TBI	56	$293,065	±19,959

LOS, length of stay.

cumulative odds of death or a poor outcome for the two groups of patients. The model also included age, gender, Injury Severity Score, GCS scores at admission (GCS scores at admission were dichotomized into two categories GCS score ≤ 8 and GCS score > 8), and the change in GCS scores 48 hours after admission. For significant risk factors, 95% confidence intervals were computed for the estimated odds ratios to assess the precision of the estimates. We also conducted an approximate likelihood ratio test to check the assumption of proportionality of odds and a test derived from the deviance statistic to assess the "goodness-of-fit" of the model.

RESULTS

The study sample showed a male predominance that was in keeping with other studies:[2,3,10–13] 78% or 84% of the selected patients were males subjects. The patient demographics and other relevant statistics are shown in Table 1. Except for mean charges, none of the other mean values for variables shown in Table 1 were significantly different from each other between the two groups. There were a total of 22 patients who deteriorated from higher GCS scores to below 8 in less than 24 hours (pre-TBI guidelines = 9, post-TBI guidelines = 13). Mean charges differed significantly between the pre-TBI guidelines and the post-TBI guidelines groups. The post-TBI guidelines group mean charges were more than $97,000 higher per patient. Offsetting the increase in mean charges was the substantial improvement in post-TBI guidelines outcomes. The proportion of deaths fell by more than half, whereas the proportion of good outcomes more than doubled in the post-TBI guidelines group of patients, as shown in Table 2.

Table 2 Glasgow Outcome Scale (GOS) by Study Group Membership

Group	GOS = 1, n (%)	GOS = 2 and 3, n (%)	GOS = 4 and 5, n (%)
Pre-TBI	16 (43.24)	11 (29.73)	10 (27.03)
Post-TBI	9 (16.07)	8 (14.29)	39 (69.64)
Total	25 (26.88)	19 (20.43)	49 (52.69)

Table 3 shows the results obtained from the ordinal regression model mentioned above. Odds ratios given in Table 3 should be interpreted as follows: for patients in the post-TBI guidelines group, odds of a good outcome (relative to the odds of a poor outcome or death) are nine times higher compared with the patients in the pre-TBI guidelines group; for patients with GCS scores > 8 at admission, odds of a good outcome (relative to the odds of a poor outcome or death) are almost 6.6 times higher compared with the patients with GCS scores ≤ 8 at admission; odds of a good outcome (relative to the odds of a poor outcome or death) decreased by a factor of 0.92 for each 1-year increase in the age of patients starting from a base age of 9 years. Note that all these odds ratios are independent of the effect of the other model variables on the outcome.

Perusal of the 95% confidence intervals shows fairly wide bounds because of the relatively small sample size. Adopting a conservative approach, we can assert with 95% confidence that the odds of a good outcome (relative to the odds of a poor outcome or death) are, at least, three times higher for patients in the post-TBI guidelines group compared with their counterparts in the pre-TBI guidelines group. Similar conservative statements apply to the other significant variables shown in Table 3.

Results obtained from the goodness-of-fit test for the model show no evidence for lack of fit (deviance $\chi^2 = 104$, $df = 135$, $p = 0.022$). Similarly, results obtained from the approximate likelihood ratio test for proportionality of odds show no evidence for rejecting the assumption (likelihood χ^2 1.34, $df = 3$, $p = 0.7202$). Given these results, a fair amount of confidence can be placed in the accuracy of the estimated odds ratios reported in Table 3.

Table 3 Glasgow Outcomes Scale: Odds Ratios for the Significant Variables (n = 93)

Variable	Odds Ratio[a]	95% Confidence Interval		p Value
		Lower Bound	Upper Bound	
Post TBI	9.13	3.25	25.67	<0.005
GCS score > 8	6.58	1.87	23.14	0.003
Age, y	0.92	0.90	0.95	<0.005

[a] Each odds ratio is adjusted for the effect of all other variables in the model.

DISCUSSION

Research exploring changes in CBF and cerebral ischemia after TBI reported that alterations in the cerebral vasculature and disruption of blood flow occur after the insult to the brain. This altered state places the brain at risk for secondary injury because of the brain's inability to increase CBF in response to hypotension, hypoxemia, and anemia.[14–16] Hypotension sustained after injury was found to adversely affect outcome.[17,18] Hyperventilation ($Paco_2 < 30$ mm Hg) reduces cerebral blood flow to the brain and was found to negatively impact outcomes of patients.[19] Hypotension, hypocapnia, anemia, and hypoxia impact cerebral oxygenation and result in episodes of cerebral oxygen desaturations.[20] Robertson et al. reported an increase in mortality in patients sustaining episodes of cerebral oxygen desaturations.[20] The need to use technologies to monitor ICP, BP, cerebral oxygenation, and overall hemodynamics has been identified in the literature as providing essential information to prioritize interventions and minimize increases in ICP, detect decreases in cerebral oxygenation, and optimize hemodynamics.

Because of this increased body of scientific knowledge questioning traditional therapies for ICP management in severe TBI, a rigorous review of the literature was undertaken from 1993 to 1995 by a select group of expert clinicians. The critical analysis and grading of each article using a scientific method rather than expert opinion culminated in the publishing of the *Guidelines for the Management of Severe Head Injury* in 1995 by the American Association of Neurologic Surgeons and the Brain Trauma Foundation.[9] The guidelines contain recommendations on managing key issues related to severe TBI and represent a major paradigm shift in the management of this critical patient population. The challenge for practitioners in centers managing this patient population is to critically review the guidelines and recent clinical research and integrate them into practice in the clinical setting. In addition, there must be consensus among trauma, neurosurgical, and intensive care physicians with regard to primary, secondary, and tertiary interventions. This consensus must be shared with the nursing, respiratory, pharmacy, and ancillary practitioners caring for the patient.

Guidelines derived from scientific data as presented in the AANS severe head injury guidelines[9] would be expected to improve or at least standardize delivery of care to patients with severe TBI. However, the suggestions need to be incorporated into a multidisciplinary clinical protocol to be effective. This protocol can then serve as a reference for the multidisciplinary team caring for the patient. Variations are thereby minimized between different physicians, nurses, and support personnel. We have demonstrated that such a clinical protocol can be successfully implemented in a community hospital setting. Variance from protocol can be an issue, but diligence on the part of the team will minimize this problem. In a community hospital, even with centralization of trauma care, numbers of patients remain small and a reference protocol keeps the team on track.

We believe that there are critical elements to the TBI team. A dedicated trauma team should include a limited number of general surgeons who are familiar with and who accept the protocol. This is particularly true for the initial evaluation and resuscitation effort. In a similar vein, a select group of neurosurgeons is also important to minimize protocol variance. An advanced practice nurse is critical to the success of the process. He or she serves as a resource and interface between the doctors, nurses, families, and support personnel. His or her involvement in clinical support, education, data collection, and interpretation are important in the implementation and the continued quality improvement of the program.

Success in implementation of the protocol must begin in the resuscitative phase. In the post-TBI guidelines era, intubation proceeded in patients who were agitated or unable to speak or maintain their airway.[21] Choosing medications that would blunt the noxious stimulation of intubation was imperative. The anesthesia team recommended medications such as etomidate, lidocaine (Xylocaine), fast-acting paralytics, and subsequent analgesia/sedative agents to minimize any untoward effects on ICP. A rapid sequence intubation protocol was developed and placed on the wall of the trauma room. The patient's $Paco_2$ was maintained at 35 to 40 mm Hg. Mannitol was only administered for specific signs of herniation such as a dilated nonreactive pupil and/or flexor/extensor posturing. Rehydration was commenced immediately with electrolyte and colloid solutions and blood products where indicated, to maintain euvolemia. Specific resuscitation phase goals were set including maintaining a minimum MAP > 90 mm Hg, CVP 5 to 10 mm Hg/PCWP 10 to 15 mm Hg, $Paco_2$ 35 mm Hg, and $Pao_2 > 100$ mm Hg. Appropriate monitoring lines were started and the patients were transferred to the CT scanner for rapid identification of intracranial lesions. After CT, the patients were taken to the operating room for ICP and Sjo_2 monitor placements.

Involvement of the anesthesia team in the protocol is critical. The patients can spend a significant part of the first 24 hours in the operating room depending on the extent of their associated injuries. The maintenance of the CPP and the Sjo_2 in the zone is critical during this early stage when the brain is most vulnerable to secondary injury. Goals for the OR phase placed emphasis on maintaining the $Sjo_2 > 55\%$, MAP > 90 mm Hg, ICP < 20 mm Hg, and titration of $Paco_2$ to meet the Sjo_2, ICP, and MAP goals. The use of sedative-hypnotic agents, such as thiopental or propofol, is helpful to reduced cerebral metabolic usage of oxygen.[21,22] A coordinated effort by the team is enhanced at our facility with the presence of the trauma nurse who stays with the patient from the initial resuscitation until admission to the ICU.

The establishment of the protocol with goals for the ICU phase was imperative for team management on a shift-to-shift basis. As seen in Figure 1, the priorities focused on keeping

ICP < 20 mm Hg and Sjo_2 between 55% and 70%. Interventions were prioritized on the basis of ICP and Sjo_2. The team's consistent selection of the most appropriate interventions was assisted by critical thinking algorithms (Fig. 1).

In clinical practice, we found that the protocol did improve clinical outcomes as predicted.[7,8] Even after controlling for differences in age and admission severity, outcomes were improved significantly, with the odds of a good outcome (relative to a poor outcome or death) at least three times higher in the post-TBI guidelines patients compared with their pre-TBI guidelines counterparts.

Other independently significant variables were also found. GCS score > 8 was independently associated with a greater odds of a good outcome. This is in keeping with many other studies.[12,23] The other independently significant variable was patient age, with younger patients having a greater odds of a good outcome. This is also not surprising, being well supported in the literature.[12,23]

Clearly, this report suffers from the problems that beset all studies that use historic controls. Changes in ICU management, improved imaging, and other advances in technology and treatment can influence outcome. In a comparable study by Rosner et al.,[7] there was an 80% improvement in survival, with a mortality rate of 29%. These results are broadly comparable with our results.

An intriguing group of patients were those who initially arrived with GCS scores > 8 and then deteriorated. In the pre-TBI guidelines group, there were nine such patients: six of these patients had good outcomes and two died. In the post-TBI guidelines group, there were 13 such patients: 9 of these patients had good outcomes and 1 died. The group that deteriorates from a less severely injured score selects for patients with less severe initial injuries who then probably develop secondary deterioration. As a group, they should have a better chance of recovery than the patients with lower admission GCS scores. This phenomenon is seen in the pre-TBI guidelines group. In the post-TBI guidelines group, outcomes for patients with or without deterioration are similar. The entire post-TBI guidelines group has been improved to the level of the best pre-TBI guidelines group by limiting, we believe, secondary injury in the more severely injured patients.

Secondary head injuries are physiologic insults that occur after the initial injury that further contribute to brain dysfunction and injury.[21] The brain is especially sensitive to decreases in blood pressure and oxygenation.[15] Analysis of data from the Traumatic Coma Data Bank found that those patients sustaining episodes of hypotension and/or hypoxia had a worse outcome than those with no episodes. The data showed that hypotension had a more profound negative impact on survival and outcome than any other factor.[15] DeWitt et al.[16] suggest that the injured brain has an enhanced vulnerability to ischemia because of the injured brain's inability to increase cerebral blood flow when challenged with hypotension, hypoxemia, or anemia. These factors are within the team's control if recognized early and treated aggressively. The TBI clinical guidelines algorithm was established to systematize the control of these parameters and thereby limit secondary brain injury.

To control the parameters that may result in secondary brain injury, invasive monitoring is essential. Besides the standard complement of arterial pressure monitor, central venous pressure monitor, and end-expiratory Pco_2 monitor, specific cerebral monitors are very helpful. A ventricular ICP monitor gives accurate assessment of intracranial pressure while also allowing drainage of CSF when necessary to control elevations in ICP. We have used both a simple fluid transducer system as well as a fiberoptic system. The monitors were always placed in the operating room to decrease the risk of infection, as suggested by Bader et al.[24] CPP was maintained above 70 mm Hg as recommended by Kelly et al.[21]

A direct or indirect measure of cerebral oxygenation is also critical to our treatment algorithm. Although direct monitoring catheters are available, we elected to use measurement of Sjo_2.[25–27] Woodman and Robertson assert that this is a useful tool for detection of cerebral ischemia because it "reflects the balance between oxygen delivery to the brain and oxygen consumption by the brain" (p. 519).[28] Situations that increase oxygen consumption or decrease oxygen delivery may be reflected in decreases in the Sjo_2. Conversely, situations that decrease the oxygen consumption or increase oxygen delivery may result in an increase in Sjo_2. The goal was an optimal range for Sjo_2 of 55% to 75%.[28–30] Desaturation requiring modification of treatment was regularly seen even with ICPs less than 20, especially during the first 24 hours after injury. Optimization of blood pressure and ventilation, and the use of propofol, resulted in optimization of Sjo_2. We strive to place the Sjo_2 monitor as soon after admission as clinically feasible.

We regularly used craniectomy in the treatment of severe TBI. Craniectomy has been useful in the control of intractable elevations in ICP.[31–33] We used craniectomy extensively both preemptively and in response to uncontrollable ICP. Ten patients in the pre-TBI guidelines group and 18 patients in the post-TBI guidelines group received craniectomies. These patients are the subject of a publication in preparation.

The implementation of the guidelines has resulted in no change in the apparent length of ICU stays or days on the ventilator (Table 1). This is, however, deceptive. The intensity of the intervention and the complexity of the care are dramatically increased. Figure 1 presents the algorithms for intervention on the basis of changes in CPP, Sjo_2, or ICP. The algorithms are a logical portrayal of the AANS guidelines interwoven with critical Sjo_2 data. A major difference from the ICP only earlier treatment paradigm is the fact that patients are actively hydrated instead of dehydrated. This has resulted in medical complications including congestive heart failure, pulmonary edema, and pneumonia. A concomitant of the hydration and pressors has been an increased incidence of

acute respiratory distress syndrome. This is in keeping with the findings of Robertson et al. of a fivefold increase in the frequency of acute respiratory distress syndrome.[34] This increase can be mitigated with intensive pulmonary management, including frequent turning, proning, and therapeutic bronchoscopies.

We analyzed our financial data to find out the effect on charges of implementing this new guideline. Spain et al.[10] evaluated the use of resources in 133 patients before and after the institution of a clinical pathway for severe traumatic brain injury (prepathway, n = 49; postpathway, n = 84) and found a significant reduction in ventilator days and intensive care unit days.[10] An improvement in resource use was also found by Simons et al., after introducing a trauma program with a clinical trauma service, revised trauma protocols, and a dedicated trauma unit.[35] We did not confirm the results reported in these studies. In our study, our two groups were comparable in mean total ventilator days and mean total ICU days. In fact, because of the increased complexity of care, the charges increased significantly in the post-TBI guidelines group, by more than $97,000 per patient. This was negatively influenced by the use of expensive drugs such as propofol and increased technological complexity with the use of SjO_2 monitors and fiberoptic ICP monitors. Since this increase in charges led to improved outcomes, we believe that the increased expenditures are justified. Furthermore, improved survival leads to the added benefit of increased societal productivity and less dependence on costly long-term care.

CONCLUSION

Implementation of a TBI clinical protocol in a community hospital setting is practical and efficacious. The protocol should be derived from current scientific literature. A multidisciplinary team is essential to coordinate care during all phases of treatment. Appropriate invasive monitoring of systemic and cerebral parameters guide care decisions. The protocol results in an increase in resource use but it also results in statistically improved outcomes justifying the increase in expenditures.

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