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Management of brain and spine injuries

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Over the past 30 years, remarkable progress has been made in improving outcome from traumatic brain injury (TBI). Overall mortalities from severe TBI have dropped from in the range of 36% in the late 1980s into the low-to-middle teens at specialized TBI centers [1,2]. Although many etiologies for such improvement have been suggested, it is unclear what changes actually affected the improvement in outcome. Despite the fervor for laboratory investigations of TBI models, it is sobering to recognize that no treatments have come from bench to bedside. As such, there are no magic bullets in the treatment of central nervous system (CNS) trauma in general, and TBI in particular. Instead, it appears to be a combination of many improvements in brain injury care, trauma systems, and critical care that has produced the decrease in mortality.

Secondary insults

It is critical to differentiate between primary and secondary insults in understanding trauma to the nervous system. The primary insult is the physical damage that occurs during the traumatic event. It may manifest as shearing or direct damage to the parenchyma or as injury to tissue or vessels that results in hemorrhage and compression of the surrounding brain.

Secondary insults are those processes occurring following the injury. They may be induced directly by the traumatic event or result from processes (sometimes iatrogenic) that follow later, or they may be caused by associated, extracerebral events. Cerebral edema, metabolic derangements, calcium toxicity, excitotoxic injury, or apoptosis are examples of processes that are initiated as a result of the trauma but evolve over time. Other secondary insults, the best known being hypoxia and hypotension, may result from the multi-system trauma or from difficulties with management issues involving other systems.

In general, the damage occurring at the time of trauma is not amenable to alteration. Secondary insults, however, are often amenable to prevention or

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reversal. Most efforts at managing TBI, from early intubation and resuscitation through intracranial pressure (ICP) and cerebral perfusion pressure (CPP) management, are focused at avoiding and minimizing secondary injuries. When the data are analyzed objectively, it is clear that the progress that has been made over the last 20 years in minimizing mortality and optimizing recovery from TBI have been because of abilities to mitigate the influence of secondary insults to the injured brain. No longer is surgery the mainstay of TBI management; indeed, probably 95% of successful management occurs in the ICU.

The management of TBI is still a young and evolving field. Because much is understood poorly or studied inadequately, there is no one way to manage TBI. On the other hand, surveys of management practice generally reveal an undo degree of variation in treatment, often involving inappropriate or incorrect use of various agents or practices [3,4]. In response to this, a set of evidence-based guidelines for the management of TBI was drawn up and distributed in 1996 by the American Association of Neurologic Surgeons and endorsed by the World Health Organization in 1997 [5]. These guidelines have been subjected to periodic revision [6]. These guidelines classified the peer-reviewed literature as to rigor and validity using a three-point scale. They then produced guidelines based on the scientific value of the supporting literature. As such, standards were based on class I literature, guidelines on class II literature, and options on class III literature. Although most of guidelines were at the option level (reflecting the rather dismal state of the literature), these guidelines have been received well, and there is a growing body of evidence that their incorporation into practice is associated with marked improvements in outcome, both on the international scene and in the United States [1]. Subsequent to the formulation of the Guidelines for the Management of Severe Head Injury, a set of evidence-based Guidelines for the Prehospital Management of Severe Head Injury have been written, funded the National Highway Traffic Safety Administration [7]. Guidelines also have been published on the management of penetrating brain injury and on the surgical management of TBI and the management of pediatric TBI [8]. As such, wherever possible, the discussions are based on evidence-based guidelines.

Resuscitation of traumatic brain injury

As in any trauma patient, the resuscitation of the TBI victim revolves around the ABCs of airway, breathing, and circulation. To this has been added D, standing for disability, representing the importance of early recognition and stabilization of CNS injury.

Airway management (A & B)

Airway control and assisted ventilation should be accomplished in all patients with a Glasgow Coma Scale (GCS) of at least 8 or any TBI patient where facial or

other injuries, aspiration, agitation, or other factors conspire against adequate ventilation, oxygenation, or airway control. In the field, endotracheal intubation is preferred in adults [7,9]. In children, no advantage has been shown for endotracheal intubation, as long as adequate ventilation and oxygenation can be delivered. In the hospital, endotracheal intubation should be accomplished using a rapid sequence intubation (RSI) protocol. In the field, although it has been reported that RSI can be used advantageously in extremely selective systems [10], there is insufficient evidence to suggest any one particular protocol for airway management. In all cases, the endotracheal position of the airway should be confirmed by return of carbon dioxide.

Even though these patients may be comatose, they still will elevate their ICP to the stimulus of endotracheal stimulation, sometimes to the point of herniation. As such, adequate analgesia and sedation are required during intubation and subsequently.

Initially, oxygen should be delivered with an FiO_2 (inspired oxygen fraction) of 1. Ventilation should be targeted at maintaining a P_aCO_2 of approximately 35 mm Hg (ie, the lower end of eucapnia). Hyperventilation should be delivered to patients only where intracranial hypertension is strongly suspected because of the presence of abnormal papillary size or reactivity (eg, anisocoria or bilaterally dilating or blown pupils), motor posturing, or progressive neurologic deterioration not attributable to extracranial circumstances (eg, hypotension, drugs, or other causes) [6,7]. It is suggested that capnography be used whenever possible to prevent inadvertent hypo- or hyperventilation. Unfortunately, despite its notable efficacy in lowering ICP and its long history of widespread use in TBI, hyperventilation induces cerebral vasoconstriction and reduces cerebral blood flow. This allows for inadvertent and undetectable iatrogenic ischemic damage. Routine use of hyperventilation in TBI patients (versus selective use targeted specifically at proven intracranial hypertension) has been shown to be associated with poorer recovery [11]. Therefore, routine hyperventilation of actual or suspected TBI patients is no longer supported in the absence of specific signs of intracranial hypertension.

For decades, it was a widely accepted tenet of TBI management to keep the patient dry by restricting fluids. This practice was based primarily on anecdote and very poor evidence (mostly extrapolated from research on tissues without a blood–brain barrier). Now it is accepted that fluid restriction in TBI is extremely hazardous, and this practice has been condemned [5,7].

Fluid resuscitation (C)

The primary early predictors of outcome from TBI are GCS score, papillary exam, age, intracranial CT diagnosis, and the presence of an episode of hypotension (defined as a systolic blood pressure no more than 90 mm Hg) [6]. Of these, hypotension is most amenable to prevention or minimization. Data from the Traumatic Coma Data Bank revealed that the presence of a single measurement of a systolic blood pressure less than or equal to 90 mm Hg from the

accident scene through resuscitation doubled the mortality and decreased the likelihood of satisfactory recovery in survivors [12]. Such episodes occurred in 35% of patients, despite relatively sophisticated urban prehospital care systems at the contributing centers. These data, supported by other studies [13,14], clearly support volume resuscitation (versus fluid restriction) as a stalwart component of all TBI resuscitation.

The tonicity of the resuscitation fluid seems to be important. Because free water distributes freely across the blood brain barrier, even when completely intact, it is highly recommended that isotonic fluids be used for resuscitation. This has led to the preference for physiologic (0.9%) normal saline over solutions such as Ringer's Lactate, a practice somewhat different that the general course of trauma resuscitation. Indeed, there is a growing body of evidence suggesting that hypertonic solutions should play a role, particularly in early resuscitation. Posthoc subgroup analysis of prospective randomized controlled trials of 7% sodium chloride solutions used as the first 250 ccs of resuscitation fluid in hypotensive patients has suggested that TBI patients have less hypotension, require less fluid to reach acceptable resuscitation values, and have survival rates superior to that predicted by Major Trauma Outcome Score (MTOS) norms [15–17]. A meta-analysis of hypertonic fluid resuscitation of TBI patients by Wade suggested that its use improved the odds ratio for survival to discharge following severe TBI by 2.12 [18]. Although 7% saline solution is not generally available, and the practice of administering it as the first 250 cc' of resuscitation fluid has not been incorporated widely, these data clearly support the critical importance of tonicity in the choice of resuscitation fluids.

In general, isotonic resuscitation of the TBI patient should be every bit as vigorous as for any trauma patient, perhaps more so, since the injured brain is so extraordinarily sensitive to hypoperfusion. Although studies have been performed using a systolic blood pressure less than or equal to 90 mm Hg as the threshold, it is obvious that the mean arterial pressure (MAP) is a more desirable value. In addition, because the ICP is also a critical variable in determining cerebral perfusion (CPP = MAP – ICP), even a normal MAP may be accompanied by cerebral ischemia in some cases. Overall, assuming a minimal acceptable CPP value of 50 mm Hg and using an ICP of 20 mm Hg in the equation, a MAP of 70 mm Hg is suggested at the lower treatment threshold during resuscitation. A MAP threshold of 90 mm Hg has been suggested as a treatment option [6].

The primary method of maintaining such pressures is full volume resuscitation. Because of the extreme sensitivity of the injured brain to hypoperfusion, however, pressors also may be needed during resuscitation. They may be needed temporarily, when fluid resuscitation is being accomplished, but the MAP remains unacceptably low. Alternatively, pressors may be needed supplementally, when full volume resuscitation does not establish an adequate MAP. In general, alpha agonists are preferred (such as phenylephrine), although, in young patients, some selective beta activity also can be useful (ie, with noradrenaline). In any instance, once pressors have been initiated, a major goal should be to wean them off while maintaining adequate perfusion using other, less toxic means. When pressors are

used, central venous pressure monitoring is necessary, and pulmonary artery catheterization should be considered if they are continued for any length of time.

Disability (D)

It is important to recognize the presence of CNS injury from the earliest contact with the patient. One of the most important tools for this purpose is the GCS score [19]. This score is simple to perform and reasonably reliable within and between observers. Indeed, when facile with the score, it can be performed intercurrent to other early resuscitative efforts without interruption.

Decreases in the GCS score represent neurologic deterioration and mandate treatment. Also, many triage and treatment protocols are based on the initial GCS. Severe brain injury is defined as GCS score of 3 to 8; moderate injury is defined as a GCS score of 9 to 12, and mild injury is defined as a GCS score of 13 to 15. Given the widespread use and general acceptance of this scale, it should remain the standard and be incorporated and documented in any trauma contact. Alternate scales, such as the AVPU (alert, verbal, painful, unresponsive) are not acceptable [7].

Authors have presented data suggesting that the GCS routinely is performed poorly and is often inaccurate when performed in the prehospital setting [20]. The general consensus, however, is that this is not the fault of the scale, but, rather, reflects inadequate training in its use, poor recognition of its value to the resuscitative effort, and lack of quality assurance feedback on the part of prehospital care systems.

The GCS is not a neurologic exam. It does not include a pupil examination. Also, the motor score represents the best limb, such that a patient with a hemi-, para- or triplegia might achieve a GCS motor score of 6 despite a significant neurologic deficit. As such, it is important to not only score the GCS but also to observe the pupils and the symmetry of motor activity as a ''forme fruste'' neurologic exam during the resuscitation.

Algorithms

Fig. 1 represents an algorithm for prehospital management of the TBI patient, adapted based on the evidence-based Guidelines for the Prehospital Management of Severe Brain Injury [7]. The ABC(D)s lead off the effort, which then includes decision points depending on the presence or absence of signs of intracranial hypertension and on whether the patient is euvolemic. If signs of intracranial hypertension are seen, hyperventilation should be initiated (targeting a P_aCO_2 of 30 mm Hg), and consideration should be given to mannitol administration if the patient is euvolemic and mannitol is available. Triage to a center with specialty expertise in neurotrauma care must be facilitated.

Fig. 2 represents an algorithm for management of the TBI patient upon arrival at hospital [6]. ABC(D) issues not addressed in the field must be managed upon initial contact, and methods of airway control other than endotracheal

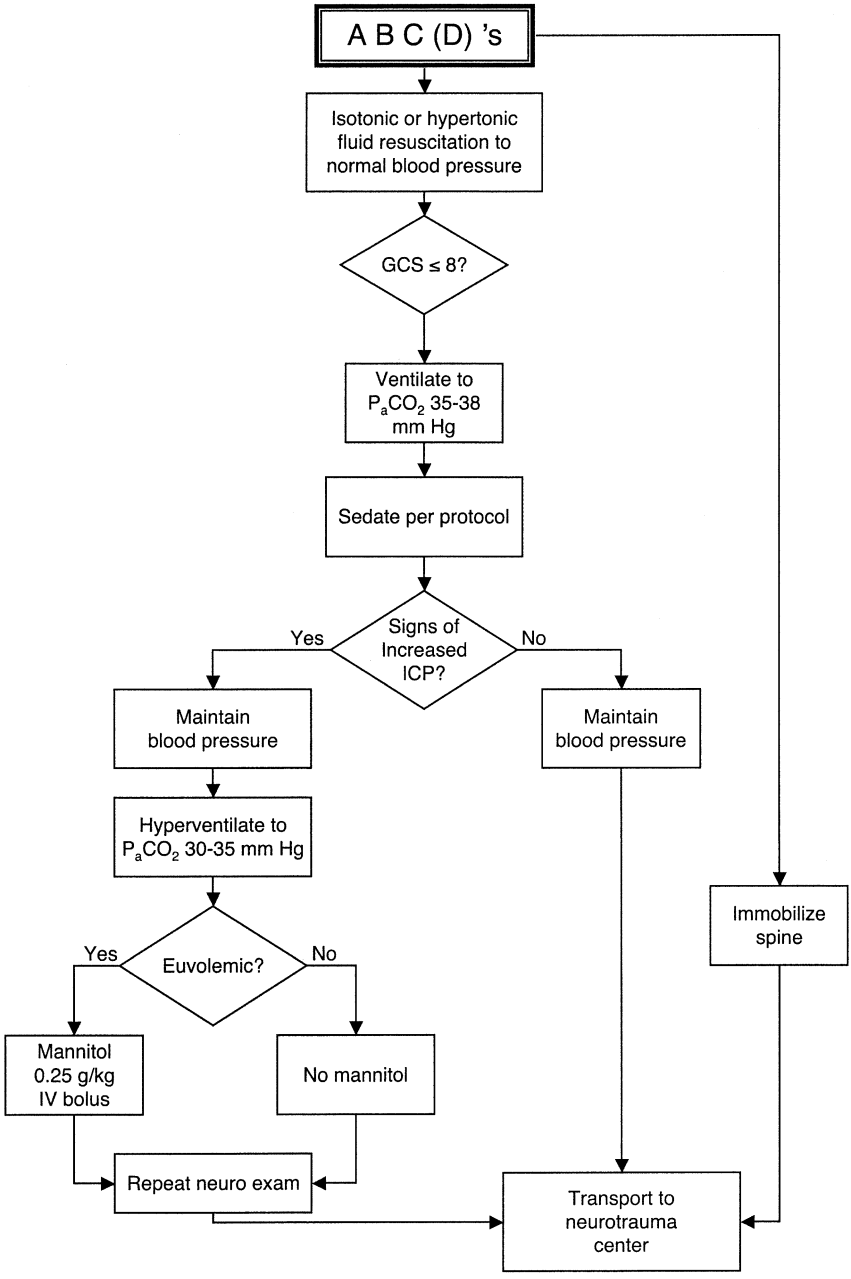


Fig. 1. Algorithm for prehospital management of patients with traumatic brain injury.

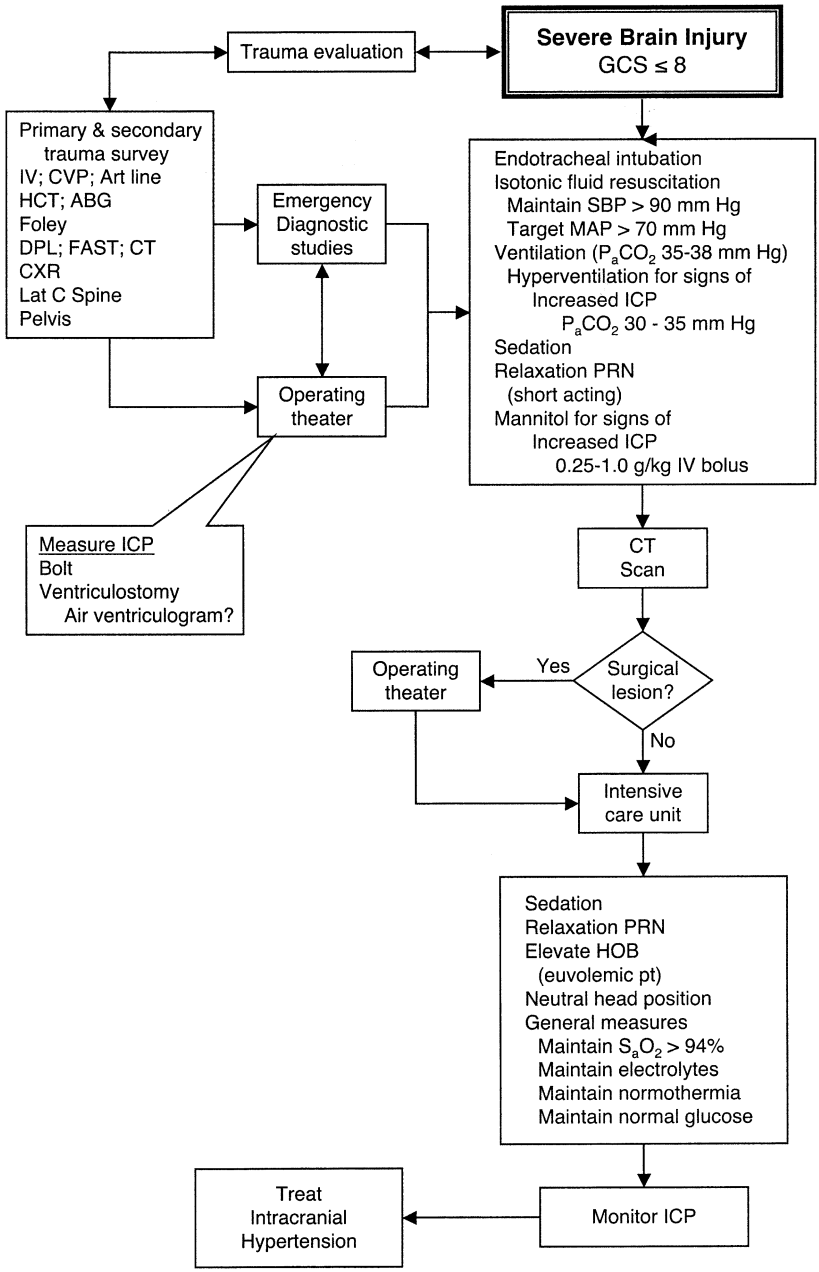


Fig. 2. Algorithm for managing patients with traumatic brain injury upon arrival at the hospital, prior to placement of an ICP monitor. *Abbreviations:* Art line, arterial pressure line; CVP, central venous pressure catheter; CXR, chest radiograph; DPL, diagnostic peritoneal lavage; FAST, abdominal ultrasound; HOB, head of bed; IV, intravenous catheter; Lat C spine, lateral cervical spine radiograph; SBP, systolic blood pressure; SaO2, peripheral oxygen saturation.

intubation should be converted. Laboratories appropriate to the setting should be evaluated. With respect to the resuscitation of the injured brain, P_aCO_2, hemoglobin/hematocrit, and serum signs of under-resuscitation are paramount. End-tidal carbon dioxide monitoring, calibrated against arterial blood gas values, is recommended. Volume resuscitation lines are mandatory. The insertion of central venous and arterial catheters also is recommended. Whenever possible, all of these should be accomplished by the resuscitation team at the earliest possible point such that they are available during transport to CT imaging and other procedures.

When sufficiently stable, the patient should be transported to the CT scanner for brain imaging. This is a critical point in the patient's resuscitation, as it often determines the feasibility and safety of other diagnostic and therapeutic maneuvers, and a positive CT finding may mandate neurosurgical consultation or operative intervention immediately. Unfortunately, the CT suite is not an ICU, so it is critical that personnel, monitoring systems, and equipment and medications requisite to any reasonably probable medical situations be constantly with the patient throughout the imaging period. Given the importance of the brain CT, it is also critical that the interpretation be immediate and correct. This mandates the involvement of a staff radiologist with appropriate familiarity with neurologic imaging, or a neurosurgeon, in the immediate interpretation of the films.

From the CT scanning suite, the patient should go to ICU unless surgery is necessary. When immediate operation is indicated, the trauma surgeon should be amenable to altering the workup in a manner facilitating craniotomy. This may mean using a diagnostic peritoneal lavage or an ultrasound evaluation in lieu of a CT scan of the abdomen, as these can be performed in the operating theater while the head is being opened. When simultaneous operative management of the head and the thorax or abdomen is necessary, the two teams should work simultaneously with two separate scrub teams.

In those instances where hemodynamic instability of the patient obviates CT imaging of the head in a patient suspected of TBI, ICP monitoring should be started in the operating theater while the extracranial surgery is being initiated. Intraparenchymal monitors are extremely useful in these instances, as they are placed very rapidly and simply (they can be done even without any skin incision), and the presence or absence of intracranial hypertension at any time during the case can drive therapy or initiate further diagnostic maneuvers. In those instances where intracranial hypertension is found, the presence of mass effect can be studied by placing a ventriculostomy and injecting a small amount of air to determine midline shift. Alternatively, burr hole exploration may be initiated immediately, frequently with beneficial diagnostic and therapeutic results [21]. There has been discussion in the literature about which patients may be taken to theater for celiotomy or thoracotomy without CT imaging of the brain [22]. Because this assumes that CT scanning has no diagnostic substitute, however, a situation countered by the previous discussion, it becomes clear that emergent extracranial surgery implies altering rather than delaying TBI diagnostics. At the least, ICP monitoring should not delayed by extracranial surgery.

An evidence report regarding the surgical management of TBI is in press. It deals with operative techniques, decompressive craniectomy, and timing of surgery, which are not discussed here. One important point, however, is that surgical management of intracranial mass lesions is most frequently not definitive, and meticulous ICU care following surgery is critical to good outcome. As such, it should be the exception rather than the rule that other, non-life-threatening procedures be performed in theater following craniotomy. Instead, the patient should be transported to ICU with a plan for return to theater after the patient has proven stable over time. In any case when there is a delay in transport from theater following craniotomy, ICP monitoring should be initiated and closely followed by the anesthesia team until arrival in ICU.

Intensive care room management

In those cases where the patient arrives directly in ICU before initiation of ICP monitoring, care is divided into what to do before and what to do after formal monitoring of ICP. The insertion of the ICP monitor should be performed as soon as possible so that definitive treatment can be initiated.

Preintensive care unit monitoring of the traumatic brain injury patient

The major goal of pre-ICP monitoring ICU management is to accomplish a normal milieu internale for the injured brain. Normal arterial oxygen tension must be maintained. Ventilation should be adjusted to keep the P_aCO_2 within the lower range of normal (35 mm Hg), unless the previously discussed clinical signs of intracranial hypertension necessitate hyperventilation to a P_aCO_2 of 30 mm Hg. Normal electrolyte values should be maintained, albeit iatrogenic elevation of the serum sodium (up to approximately 150 mg/dL) may be acceptable when hypertonic saline is being used for treating intracranial hypertension.

Normothermia is desired following TBI. Fever will increase the cerebral metabolism and raise the ICP. As such, aggressive management of the patient's temperature is supported, particularly when ICP control is an issue. If acetaminophen does not produce adequate temperature control, indomethacin is generally quite effective.

The recent National Institutes of Health-funded prospective, randomized, controlled trial on the efficacy of induced hypothermia in improving outcome from TBI did not support its use [23]. This study was focused on all patients with TBI, not simply those with intracranial hypertension. As such, iatrogenically lowering the body temperature into the range of 32° to 34°C is not supported. This study found that there was an advantage to not aggressively warming patients who come in mildly hypothermic, and it now is suggested that such patients be allowed to warm up on their own (rather than with warming blankets or other means) unless problems such as coagulopathy mandate more aggressive warming.

Sedation lowers ICP and facilitates patient management. The difference between sedation and analgesia must be remembered. Short-acting agents are preferred so as to facilitate repeated neurologic examination. The most common methods are a fentanyl infusion for continuous analgesia and airway reflex depression and an infusion of either midazolam or propofol for sedation. The ability of propofol to lower cerebral metabolism, and, therefore, to improve the ischemia threshold makes it generally the preferred agent. Unfortunately, propofol cannot be used in the pediatric population.

Neuromuscular blockade will augment ICP control by preventing ventilator asynchrony and lowering muscular tension. The routine use of prophylactic neuromuscular blockade (ie, for the first 24 hours following admission) has been shown to be associated with prolonged ICU stay and increased complications without benefiting outcome [24]. Therefore, neuromuscular blockade should be used only in the treatment of intracranial hypertension and discontinued as soon as possible. Again, short-acting agents are preferred.

Raising the head of the bed will lower ICP. Its effects on CPP, however, are variable. Careful analysis of the most frequently quoted study elevating the head of the bed reveals that most patients were under-resuscitated according to their central venous pressure values [25]. Most subsequent studies have suggested that elevating the head of the bed in fully resuscitated patients improves ICP and CPP. In the individual patient, close observation of the effects of this maneuver on ICP and CPP should be used to determine the proper nursing position. Elevation of the head of the bed, when performed, initially should be in reverse Trendelenberg position. This subsequently should be changed to the Semi-Fowler position when the thoracic and lumbar spines have been cleared.

The Guidelines for the Management of Severe Head Injury found that the literature does not support the early administration of antiseizure medications for the purpose of preventing the development of post-traumatic epilepsy [6]. The available evidence does support the efficacy of early anticonvulsants in preventing early seizures. There has, however, been no demonstrated correlation between early seizures and outcome. As such, early administration of anticonvulsants is suggested only in patients who have manifested early seizures or in whom early epilepsy might go undiagnosed (ie, patients under neuromuscular blockade) or might be particularly hazardous (ie, in patients with marginal control of ICP).

Based on class I literature, the Guidelines for the Management of Severe Head Injury promoted a standard that the use of glucocorticoids is not recommended for improving outcome or reducing intracranial pressure in patients with severe head injury [6].

Postintracranial pressure monitoring of the traumatic brain injury patient

Following the rapid insertion of an ICP monitor, the phase of ICP management begins. The classical management approach is a staircase-type method, wherein treatments are added sequentially when present therapies are inadequate. The Guidelines for the Management of Severe Head Injury has adopted such an

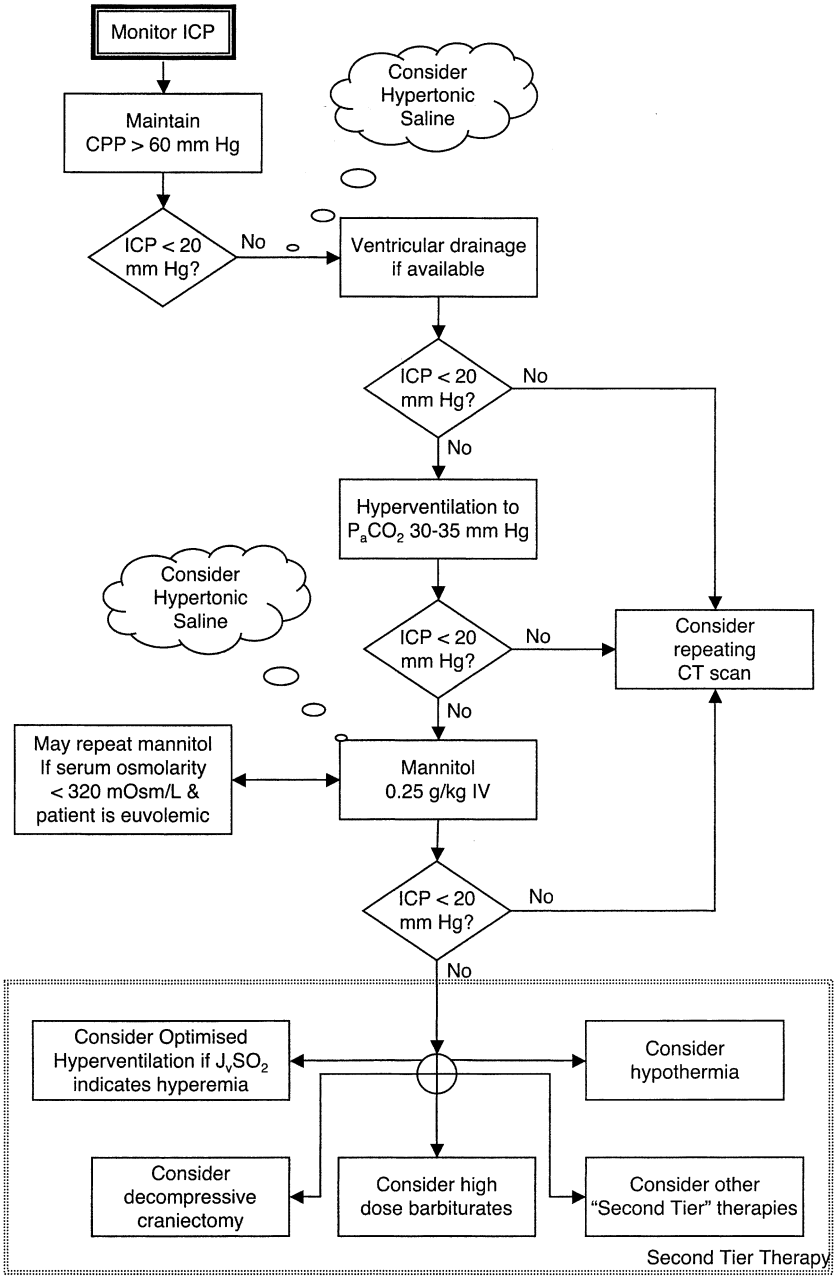


Fig. 3. Evidence-based algorithm for managing patients with traumatic brain injury following placement of an ICP monitor, including management of intracranial hypertension. The patient enters the second tier when control of ICP does not respond to conventional treatment. *Abbreviation:* J_vSO_2, jugular venous oxygen saturation.

approach, ordering the sequence of therapeutic escalation based on the risk:benefit ratio of individual treatments [6]. The resultant algorithm is shown in Fig. 3.

The conditio sine qua non of ICP management is maintaining an adequate CPP. Although some authors have argued that it is necessary to maintain the CPP at a markedly supraphysiologic level (ie, 70 to 90 mm Hg) [26,27], the general consensus is trending more toward levels of 50 to 60 mm Hg [28–30]. Indeed, there is a growing argument that the elevation of CPP serves mainly as an extra wide margin of safety against hypotensive episodes, which, unfortunately, may occur rather frequently in the ICU with devastating effect [31–33]. The consequences of protracted use of hypovolemia and pressors appear to obviate the benefits of CPP elevation [30], and, in children, the detrimental effect of hypotension appears to be a distinct threshold effect with no apparent added benefit of CPP elevation [34]. Therefore, it is advised to maintain a CPP of 50 to 60 mm Hg such that there is absolutely no tolerance for values below such a threshold.

When intracranial hypertension occurs, the treatment algorithm is activated. The adequacy of the general points made previously is assessed quickly (ie, adequate ventilation, absence of fever, adequate sedation) and corrected, if necessary. If a ventricular catheter is being used for ICP monitoring, drainage is initiated. If an intraparenchymal system is being employed, placing a separate ventricular drain for CSF diversion should be considered. If CSF drainage is not effective, then hyperventilation to a P_aCO_2 of 30 mm Hg is initiated. If this is not effective, mannitol may be given and repeated as needed. During this entire cascade, the possibility that a surgical mass lesion might be contributing to the intracranial hypertension must be considered, and repeating the CT of the brain should be entertained. Should none of these measures be effective, then one must decide whether to enter the second tier of ICP management (vide infra).

Ventricular drainage is safe and effective in lowering ICP [35]. When carefully inserted in noncoagulopathic patients, the risk of hemorrhage is low. The reported risk of infection varies widely but probably lies in the range of 1% to 2% when colonization is differentiated from infection [36]. When being drained, there should be a slightly positive gradient to the drainage siphon (eg, 5 cm water). Drainage should be intermittent, as ICP monitoring is inaccurate as long as the system is open to drain. One drainage algorithm is to drain for 2 minutes, then close the drain and measure the ICP. Under such an algorithm, a change in management is indicated when drainage is needed five or more times in an hour. In general, surveillance cultures are taken every 24 to 48 hours, and the catheter is removed or replaced if there is a rising white count, hypoglycorrhachia, or evidence of bacteria on either Gram's stain or culture. It appears that the major risk for infection in ventriculostomies maintained as closed systems occurs at the time of insertion [37]. As such, catheters should be implanted under sterile conditions, and opening of the system, such as for samples or compliance testing, should be minimized.

There is strong evidence that overuse of hyperventilation impedes recovery [11]. As such, this treatment modality must be used with discretion and limited in duration and magnitude. Additionally, monitoring of cerebral blood flow or the

balance between perfusion and metabolism should be considered. When initiated, the initial target of hyperventilation should be 30 mm Hg. Following end tidal carbon dioxide may be useful in maintaining ventilation at the proper volume. Further depression of the P_aCO_2 is considered second tier therapy and should not be performed at this stage.

Although long a mainstay of ICP management, there is little literature on the relative efficacy of mannitol for managing intracranial hypertension [6]. It is believed to work by a combination of osmotic dehydration decreasing the tissue volume and viscosity-autoregulation-induced vasoconstriction, reducing the cerebral blood volume [38,39]. It is generally effective in lowering ICP for a variable period. There is some evidence that lower doses (eg, 0.25 g/kg) have ICP lowering effects similar to higher doses (eg, 1 g/kg) [40]. As such, the lowest effective individual dose is recommended to maintain the repeatability of this treatment. Somewhat arbitrarily, a serum osmolarity of 320 mOsm/L generally has been set. Beyond this, further doses are felt to place the kidneys at risk. As mannitol is a diuretic, volume contraction (with its attendant risk of hypotension) must be avoided [41]. Mannitol is generally more effective when given as a bolus than as a slower infusion.

Second tier therapy

When intracranial hypertension obtains despite the above therapies, classical ICP treatment enters the second tier. At this point, in patients considered salvageable, treatments such as optimized hyperventilation, barbiturate coma, and decompressive craniectomy are considered.

Optimized hyperventilation consists of increasing the minute ventilation to bring the P_aCO_2 below 30 mm Hg while monitoring the brain for evidence of iatrogenic ischemia. The concept is that, in a certain percentage of patients, the cerebral blood flow is not coupled properly to the metabolic needs of the brain, such that a state of hyperperfusion exists. Because hyperventilation produces vasoconstriction and lowers the cerebral blood volume, and since lowering the cerebral blood volume lowers ICP, the idea is to use this flow:metabolic uncoupling to the physician's advantage. By monitoring for evidence of global ischemia, generally through the use of jugular venous saturation monitoring, hyperventilation is increased slowly until ICP control is obtained, or the jugular oxygen saturation nears the lower threshold of acceptability (eg, 60% to 70%) [42]. In some cases, particularly in patients with diffuse swelling of the brain without evidence of widespread contusion or primary parenchymal injury, this can produce adequate ICP control without evidence of global ischemia [42]. Unfortunately, there are concerns about missing cerebral ischemia with jugular oxygen saturation monitoring, which continue to limit the acceptance of optimized hyperventilation (vide infra).

The best studied second tier therapy for intracranial hypertension is barbiturate coma [43]. Pentobarbital sodium is loaded at 10 mg/kg intravenously over 30 minutes, followed by an infusion of 5 mg/kg per hour for three hours then

maintained at 1–3 mg/kg per hour, titrated to burst suppression on continuous bedside electroencephalogram (EEG). Serum levels are followed, but clinical and electrophysiological endpoints direct dosage. This profoundly depresses the cerebral metabolism, which, by virtue of cerebral metabolic vascular autoregulation, decreases cerebral blood flow, and, therefore, cerebral blood volume without inducing ischemia. As a result, ICP drops. The infusion is maintained until ICP control is satisfactory for 24 hours, then backed off by approximately 50% per day. Such a treatment has been shown in a prospective randomized controlled trial (PRCT) to be more effective than conventional therapy in reducing intractable intracranial hypertension in a fashion that appears to be associated with improved outcome [43].

Unfortunately, barbiturate coma for refractory intracranial hypertension is hazardous and arduous. High-dose pentobarbital has a strong tendency to produce hypotension, the ischemic consequences of which obviate the benefits of its ICP control [43–45]. Very careful attention to volume status, frequently supplemented by a need for invasive monitoring and pressor use, is necessary to avoid such complications. In addition, barbiturate coma produces a somewhat anergic state, wherein infections such as pneumonia are common. Early signs of sepsis such as fever may be blunted, and decreased blood pressure may be attributed directly to the drug.

Extreme vigilance in an excellent ICU experienced in barbiturate coma and prevention of its complications generally is required to reap its benefits. As a result, this treatment modality has lost a great deal of its popularity. Attempts have been made to replace barbiturates with propofol, titrating propofol to a state similar to burst suppression [46]. Although such management may be successful in producing ICP control, the possibility of potentially fatal cardiac toxicity at the required doses of propofol has dampened enthusiasm for this course markedly [47].

Of note, PRCTs have found that barbiturate coma is not useful as prophylaxis for intracranial hypertension [45] or as a substitute for mannitol at earlier stages of ICP management [44]. The hypotensive complications eliminated any beneficial effects of high-dose barbiturates in both such instances.

A third second tier therapy is decompressive craniectomy, which also can be somewhat separately considered as an alternative treatment strategy at earlier stages of treatment in some patients (vide infra). The history of decompressive craniectomy as a last ditch effort is controversial, with a seminal report of success being tempered by a follow-up article suggesting that it resulted in unsatisfactory neurologic recovery [48,49]. Subsequent articles have revealed that the success of decompressive craniectomy is related directly to the number of secondary insults suffered by the injured brain before decompression [50–52]. It is generally felt that the relatively unsophisticated prehospital, diagnostic, and ICU systems in place during patient collection for the early reports played strong roles in the poor recovery in a manner that should not obtain at present. Nevertheless, decompressive craniectomy probably is viewed much more appropriately as an alternative treatment to be considered much earlier in the ICP course rather than as a salvage procedure.

Alternative treatment methods

Alternative treatment methods are newer, less well studied, or somewhat controversial issues lie outside of evidence-based guideline efforts. Therefore, they hold somewhat unclear places in management. Examples of alternative treatment methods are hypertonic saline, lumbar drainage, and decompressive craniectomy.

Hypertonic saline induces osmotic dehydration and viscosity-related vasoconstriction in like manner to mannitol. The use of hypertonic saline to reduce intracranial hypertension has been best studied in children. In this population, administration of hypertonic saline (generally 3% NaCl at approximately 0.5–1.0 mg/kg per hour) seems associated with fewer ICP spikes, a lesser need for interventions, and improved short-term survival with few complications [53–55]. Serum sodium levels were raised into the range of 150 to 160 mEq/L, and serum osmolarities went up to around 360 mOsm/L. Unfortunately, there is no class II or greater evidence of improved long-term outcome. Hypertonic saline therapy, therefore, appears to be a relatively safe method of managing intracranial hypertension, but it has not been studied satisfactorily in terms of long-term efficacy.

Although CSF diversion by way of ventriculostomy is a mainstay in the management of intracranial hypertension, lumbar drainage generally has not been used based on concerns of inducing downward herniation in patients with increased ICP. In children, however, reports have suggested that it may be an effective supplement to ventricular drainage in very selected cases, because it lowers intracranial hypertension refractory to more classical management [56]. Unfortunately, there are no controlled outcome data using this technique. As such, at least in the pediatric population, lumbar drainage might be considered for refractory intracranial hypertension with a functioning ventriculostomy, open basal cisterns, and no evidence of a major mass lesion or shift on imaging studies.

Decompressive craniectomy has been discussed previously as a second tier therapy. It is known, however, that its efficacy is related directly to its being performed in patients who have not suffered significant secondary insults (ie, episodes of significant intracranial hypertension, cerebral hypoperfusion, herniation syndromes) [50–52]. Instead, the optimal patient for decompressive craniectomy would be a young patient with reactive pupils and a GCS score greater than 3 whose CT scan reveals uni- or bilateral hemispheric swelling with relatively little CT evidence of direct parenchymal damage or clinical evidence of brainstem injury [50]. Such patients' outcomes are dependent on avoiding postinjury complications rather than the reversal of primary injuries. Surgical decompression may be considered in such instances earlier rather than later, when medical management of ICP monitoring is failing. Optimally, it is considered before frank failure and is performed in advance of refractory intracranial hypertension and prolonged drops in CPP.

The concept behind decompressive craniectomy is to allow the brain to swell in a fashion that is not harmful to it. The craniectomy needs to be sufficiently large, so that venous outflow from the bulging tissue is not embarrassed at the margins. In

general, this means taking off as much bone as possible. In cases of global swelling, bilateral fronto–temporo–parietal craniectomies are performed. When unilateral hemispheric swelling is at fault, a very large lateral fronto–temporo–parietal flap is removed. Also, the brain must not be restricted in its swelling, so a generous duraplasty must be performed. It is not necessary, and, indeed, not advisable to perform a watertight dural closure. Instead, a large dural patch (xenograft, allograft, or synthetic) may be laid on the brain, the edges tucked under the margins of the defect, and the cruciate dural flaps simply centrally oriented with a single stitch placed through the tips. When the flap is replaced at 3months, a thick pseudodura will be encountered.

The goal is to obviate further ICP therapy completely. This is particularly useful when ICP therapy is proving toxic (eg, pressors in acidosis), poorly tolerated (eg, hyperventilation in acute respiratory distress syndrome [ARDS]), or contraindicated (eg, barbiturate coma in a patient with marginal blood pressure or an ongoing pneumonia). Cerebral perfusion tends to be voluminous following such treatment. Indeed, the elevation of CPP following decompression is probably a bad idea, as it appears to induce further cerebral hyperemia because of an iatrogenic loss of pressure autoregulation (R.M.C., personal observation).

Monitoring

As with any organ system, the necessity and efficacy of treatment must be determined through monitoring of physiologic processes. On the other hand, the addition of a monitor to a treatment process should reflect an underlying thought process. The main goals of TBI management are to avoid pressure-related tissue damage and cerebral ischemia. Pressure is monitored with an ICP monitor. The ICP also plays a role in managing perfusion, as it is part of the cerebral perfusion pressure equation (CPP = MAP – ICP). As such, ICP monitoring is a mainstay in almost all TBI care. Cerebral blood flow (CBF) monitoring may be accomplished locally (by way of thermistors or laser Doppler), regionally (by way of transcranial Doppler), or globally (by way of xenon or contrast-mediated CT CBF measurement or time-of-flight MRI). As flow is only part of the spectrum of variables determining the adequacy of perfusion, however, CBF cannot be used to detect ischemia directly. This requires data on oxygen carrying capacity (ie, serum hemoglobin), oxygen saturation, and cerebral metabolism. Because cerebral metabolism (CMRO2) is very difficult to measure clinically (eg, positron emission tomographic [PET] scanning), the general approach is to monitor the balance between supply (perfusion) and demand (metabolism). This cannot be measured clinically on a truly global basis. Jugular venous saturation is the most global measurement strategy; methods such as tissue oxygen saturation provide only regional data.

Jugular oxygen saturation produces an average value for a large (but not total) volume of the brain. Therefore, by virtue of admixture, it will average out small areas of ischemia in brains with heterogeneous metabolism or flow distribution.

Although monitoring for cerebral lactate production (ie, measuring the jugular arteriovenous difference in lactate) or following the cerebral lactate–oxygen index can be used to attempt to avoid such an error, there is significant concern about missing regional ischemia using jugular monitoring [57]. As such, it should be used with caution, and supplementation with monitoring of cerebral blood flow or tissue oxygen saturation should be considered.

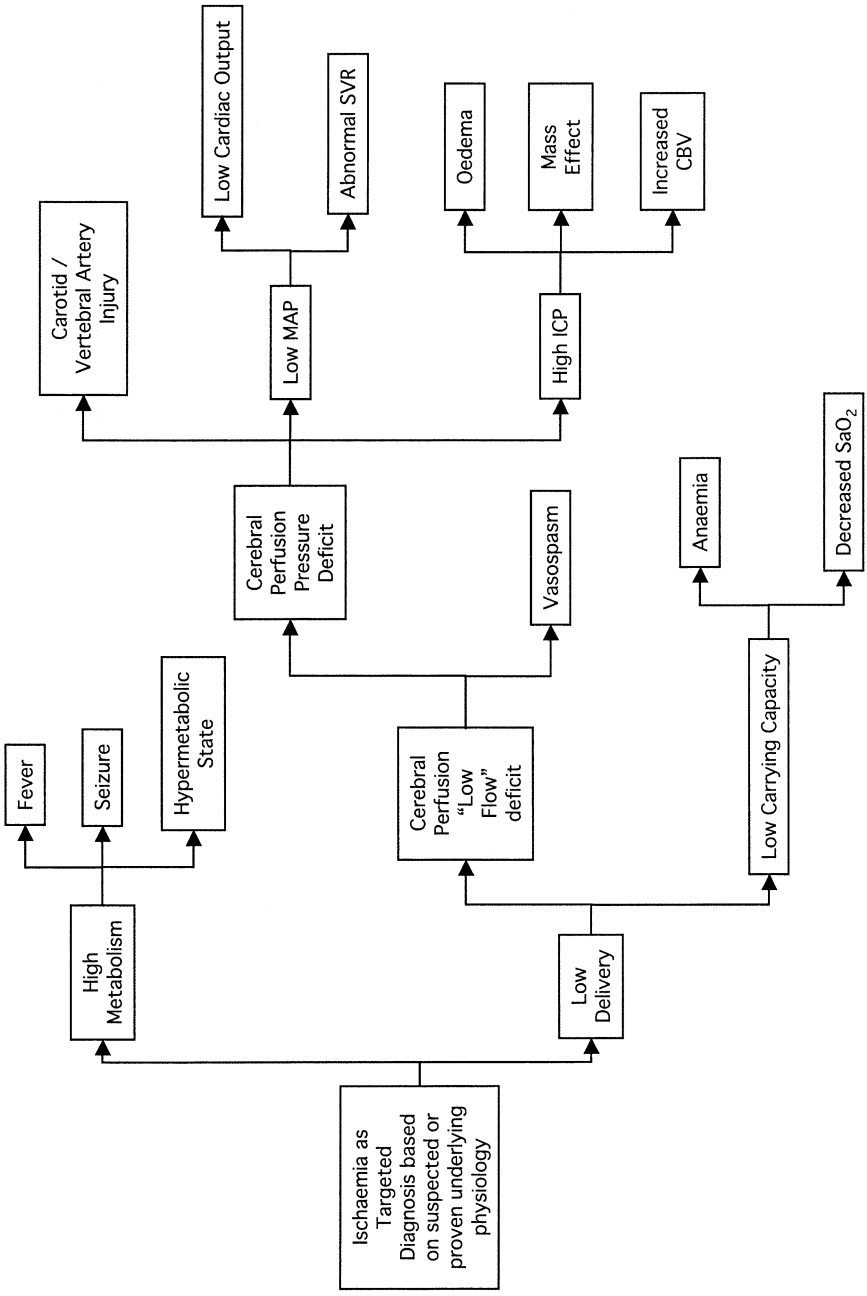
Electrophysiologic studies can be useful in the ICU. Somatosensory evoked potential (SSEP) and brainstem auditory evoked potentials (BAERS) may be used to search for injuries to deep brain structures for prognostic reasons. EEG may be used to monitor induced depression of CMRO2 for therapeutic reasons (eg, barbiturate or propofol coma) or to look for the presence of seizure behavior in patients with suggestive motor behavior, unstable vital signs, or unexplained intracranial hypertension. Unfortunately, despite various suggestive articles, EEG has yet to prove itself very useful in general prognosis.

Targeted therapy

As understanding of the physiological processes underlying TBI and abilities to monitor these processes improve, there is a growing tendency to alter the fundamental approach to managing ICP and cerebral perfusion. As noted previously, the classical approach has resembled a staircase, wherein there is escalation up a set cascade of treatments as ICP proves increasingly intractable. As such, in general, mild hyperventilation is used before mannitol, and strategies such as decompressive craniectomy or barbiturate coma are reserved for severe cases. Although this strategy makes sense in terms of risk:benefit ratios for individual treatments, it risks a mismatch between the pathophysiologic processes underlying a given patient's injury and the treatment delivered. For instance, in a situation wherein ICP is driven by cerebral hyperemia recruited by high metabolic needs, mannitol decreasing tissue edema would not address the problem, and hyperventilation risks ischemia. More properly, this situation would be detected by finding increased cerebral blood flow, an ICP waveform consistent with cerebral hyperemia, and low jugular venous oxygen saturation and tissue oxygen tension. Such a spectrum of findings would lead to increasing the carrying capacity and oxygen saturation of the delivered blood, and, perhaps, to decreasing the cerebral metabolism with a sedative–hypnotic such as propofol. Such a physiology-based, monitor-driven therapeutic approach, common in other disease entities, has been termed targeted therapy [58,59]. An example of a physiologic reasoning tree useful in guiding targeted treatment of intracranial hypertension is illustrated in Fig. 4.

Spine and spinal cord trauma

Despite significant efforts in this area, there is no treatment effective in promoting recovery or repairing the damaged spinal cord. As with TBI, minimi-


Ischaemia as Targeted Diagnosis based on suspected or proven underlying physiology
High Metabolism
Low Delivery
Fever
Seizure
Hypermetabolic State
Cerebral Perfusion "Low Flow" deficit
Low Carrying Capacity
Cerebral Perfusion Pressure Deficit
Vasospasm
Anaemia
Decreased SaO_2
Carotid / Vertebral Artery Injury
Low MAP
High ICP
Low Cardiac Output
Abnormal SVR
Oedema
Mass Effect
Increased CBV

zation of secondary insults remains critical in minimizing deficits. It is in this area that progress with these injuries lies.

In truth, credit for the improved outcomes from spinal cord injury (SCI) in general and for the shift from plegia toward paresis in SCI patients arriving at receiving institutions must be attributed primarily to vigilance on the part of prehospital care providers. It is critical to assume that all trauma patients have spine injuries (cord, cauda equina, or column) until proven otherwise. This leads to rigid immobilization at all stages, from initial contact through clearing of the spine. Use of the backboard to immobilize the thoracic and lumbosacral regions plus tape and sandbags (or equivalent) with or without cervical orthoses allows life threatening resuscitation efforts to be performed while any spinal injury is placed on hold. This is a conditio sine qua non of all trauma care, and any lapses in its rigor will lead to disastrous errors.

Clearing the spine

A corollary of this tenet is that no area of the spine may be mobilized until specifically cleared. Clearance may range from clinically clearing the patient based on an interview and examination through clearance based on static and dynamic imaging using various modalities. An evidence-based analysis of the literature on clearing the cervical spine has been produced by the Eastern Association for the Surgery of Trauma, and the following recommendations are based on their work and subsequent validation studies [60–62]. Patients who are awake, alert, sober (of all recreational and therapeutic agents) and who do not have injuries to another system that will distract them from a spinal injury (eg, an extremity fracture) are candidates for clearance on clinical grounds. Such patients may be mobilized if they deny axial skeleton pain, a careful neurological examination reveals no deficits, and the examination of their spine uncovers no areas of suspicion. The region in question then may be mobilized and, if pain-free under dynamic conditions, immobilization may be ended. Should they have pain on mobilization that seems to be spinal in origin, they should be immobilized again, and imaging should be obtained. An algorithm illustrating such a pathway is shown in Fig. 5.

In the thoracic and lumbosacral regions, negative, good quality images under these conditions generally allow mobilization. In the cervical spine, neck pain on flexion and extension despite good quality images that do not show evidence of injury is not as clear an indicator of the absence of instability. Case reports of delayed dynamic instability have led to suggestions ranging from clearance based on MRI imaging (including STIR sequences) to discharge in a rigid cervical orthosis with follow-up dynamic films at 7 to 14 days. The general point is that patients with significant neck pain will splint unstable motion segments early after their injuries. As such, imaging their soft tissues or re-examining the patient

Fig. 4. Algorithm illustrating the physiologic processes to be considered when evaluating the traumatic brain injury patient for treatment based on targeted therapy. *Abbreviations:* CBV, cerebral blood volume; S_aO_2, peripheral oxygen saturation; SVR, systemic vascular resistance.

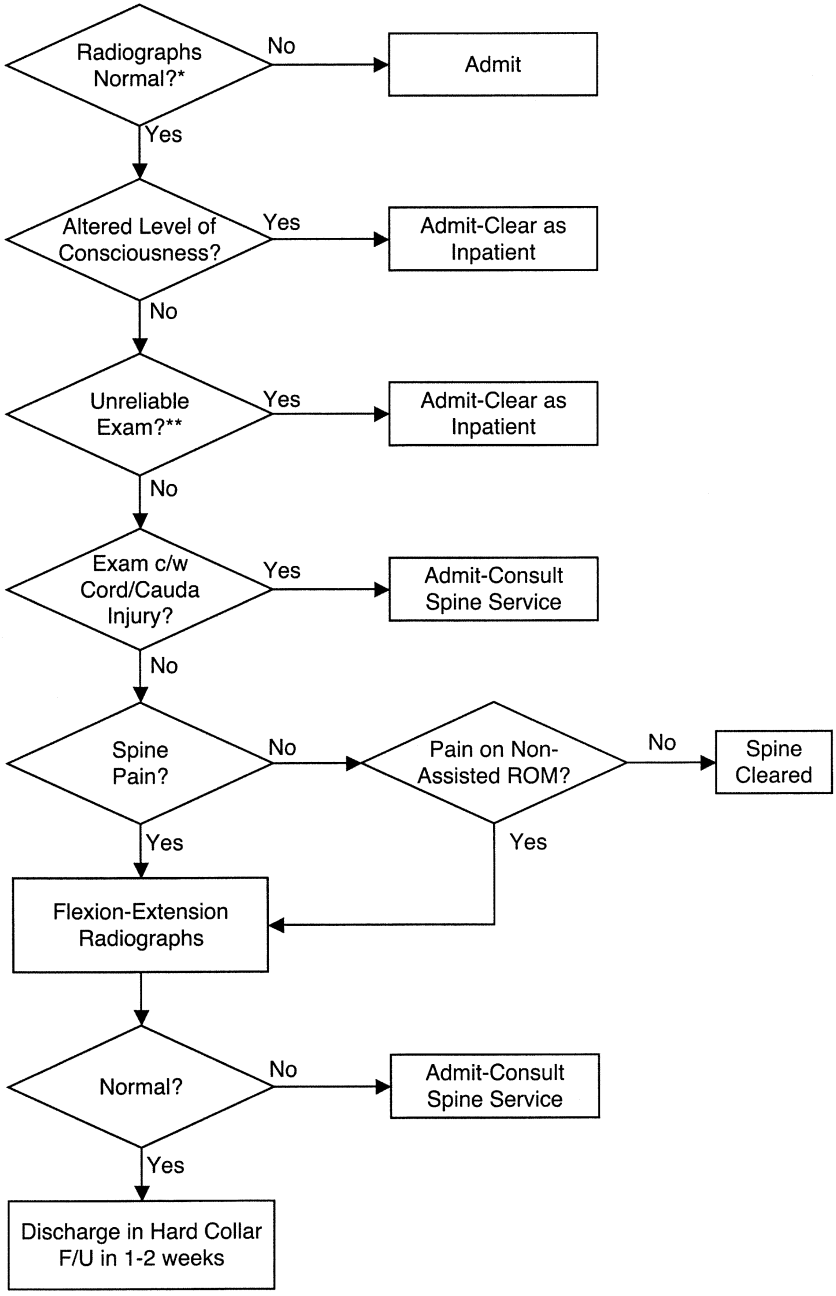


Fig. 5. Algorithm for clearing cervical spine in a conscious patient. *Abbreviations:* c/w, consistent with; F/U, follow-up; ROM, range of motion.

and repeating dynamic films after some of the acute spasm has dissipated generally is recommended.

In patients where the previous conditions do not apply, imaging is mandated. In cases where neurologic injury is not in question, imaging is focused on finding evidence of fracture or ligamentous instability. In cases where neurologic injury is suspected, imaging of the neural structures also is required. Clearing the spine in cases of TBI is particularly problematic, and evidence-based protocols generally lie only at the option level. Further discussion in this area is beyond the scope of this article; the reader is referred to the evidence report performed by the Eastern Association for the Surgery of Trauma at www.east.org [61]. The algorithm used at the author's institution is shown in Fig. 6. In this algorithm, radiographic clearance indicates that a plain lateral image and thin cut CT from the occiput through T1 are read as normal by a staff radiologist. This reading must be corroborated with a reading by the clearing service (neurosurgery, orthopedic spine, or trauma). The patient's exam also must be sufficient to rule out gross mono-, hemi-, para-, or quadra-pareses attributable to a possible spinal injury. This obviates clearing the spine in patients on neuromuscular blockade. Should the films not be consistent with a fracture or misalignment and no gross deficit detected, cervical immobilization is removed.

ABCs

Airway management (A & B)

Although much less well studied than in TBI, the proper management of the airway and maintenance of proper oxygenation and ventilation are no less important in SCI. Airway management is complicated by the necessity of immobilization and the tenuous nature of the spinal cord in the patient with cervical instability. Aspiration is a particularly onerous problem in this population. As such, early, pre-emptive airway control is desirable but complicated by the difficulty of intubating a patient with an unclear cervical spine. As such, the necessity of endotracheal intubation should be evaluated early and repeatedly. When indicated, the optimal manner of intubation is using a fiber optic system in a controlled environment. When this is not available or possible (eg, in the field), endotracheal intubation should be accomplished with absolute attention to spine immobilization. It is critical to avoid axial traction when immobilizing the cervical spine, as marked distraction of injuries has been demonstrated to occur with even minimal traction [63]. The old concept of axial traction is to be avoided; the head must be held still, but no axial force should be applied. The Sellick maneuver may be useful to prevent vomiting during intubation. There has been no definite advantage shown for the nasotracheal or the orotracheal route.

When endotracheal intubation cannot be performed easily, the acceptability of alternate methods of airway control (such as laryngeal masks) should be

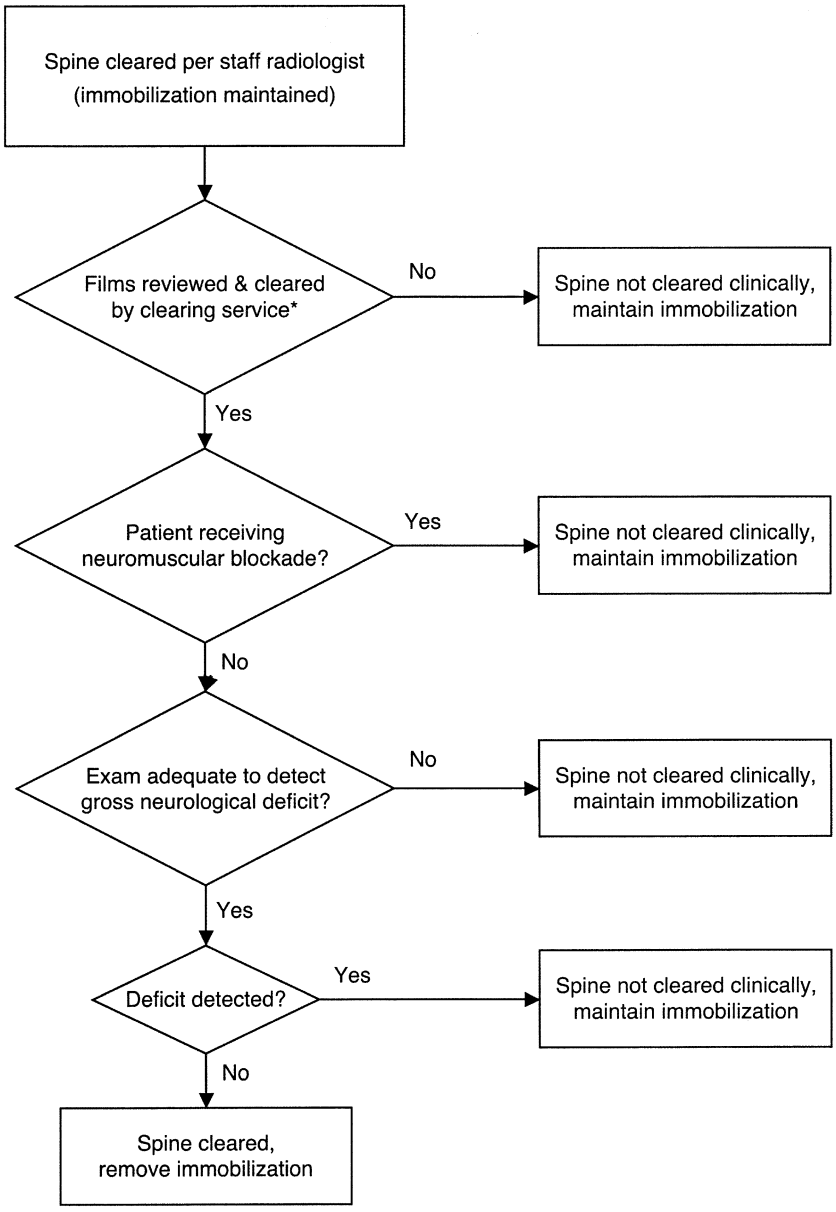


Fig. 6. Algorithm for clearing cervical spine in a patient with an altered level of consciousness.

considered rather than placing the patient at undue risk. When airway control is mandated, and endotracheal intubation is not successful, a surgical airway should be established.

Extubating patients with severe mid- to high cervical spinal cord injuries is problematic, as they are diaphragmatic breathers, and it takes little to precipitate decompensation. As such, many centers perform tracheostomies in all such patients and maintain them throughout the acute care period, quoting benefits in terms of fewer complications and decreased hospital costs [64]. Although this may result in some patients receiving a surgical airway that they might not need, it avoids the frequently disastrous instances of aspiration or hypoxia that may occur in extubated high cervical cord injury patients after they have been moved from the ICU to a less well-observed venue.

Circulation (C)

Although much less clearly studied, the injured spinal cord appears to mimic the injured brain in its extreme sensitivity to hypoperfusion. Early hypotension is associated with increased mortality and decreased neurologic recovery [65]. As such, vigorous volume resuscitation to normal perfusion values is necessary. Unfortunately, SCI can complicate this process when sympathetic tone is lost in regions distal to the injury (producing peripheral pooling of volume), or sympathetic drive to the heart is lost, and unopposed parasympathetic drive prevents reflex tachycardia or produces bradycardia. In such instances, volume resuscitation is complicated, and central pressure monitoring to some extent is required. Volume redistribution also may be useful, such as by elevating the legs or preventing venous pooling with compression stockings. Pressors may be useful early on to reverse the drop in peripheral vascular resistance. In general, alpha agonists (eg, phenylephrine) are the most useful. In instances of bradycardia, anticholinergic agents such as atropine or glycopyrrolate may be used.

Despite the absence of much support in the human clinical literature, it is a frequent practice to artificially elevate the blood pressure for some time following SCI. Although it is not clear whether this improves perfusion to the area of injury, MAP targets of 90 to 100 mm Hg are discussed. This generally requires pressors in addition to strict maintenance of volume loading. One possible advantage of such a policy is that it adds a buffer that serves against transient hypotension, the effects of which are damaging (vide supra). In the absence of proven benefit, however, artificial hypertension should not be used in the presence of any contraindications or complications of such therapy. Finally, as a result of the SCI, blood pressure often will fall to somewhat low levels when pressors are stopped, complicating the issue of stopping hypertensive therapy. There is really no good source of guidelines to assist in directing such therapy. As such, a reasonable practice is to maintain MAPs of 90 to 100 mm Hg for approximately 72 hours, thereafter backing off and letting the blood pressure drop to its new baseline under close clinical observation.

Timing of surgery

The proper timing of surgery following SCI remains controversial. Experimental studies in animals have suggested that there is a window that appears to range up to 4 to 6, perhaps 8 hours following injury, wherein decompression of the injured spinal cord improves recovery [66]. Clinically, seeing suspected compression of an injured cord on imaging studies is very disturbing and leads one to desire to perform a surgical decompression immediately. Unfortunately, the experimental situation is markedly different from the laboratory setting, and predictors of outcome are understood poorly at the time of presentation. The laboratory SCI is controlled and isolated. Clinically, however, what is seen on arrival at the hospital does not represent the often dramatic occurrences at the cord level that took place during the moments of trauma. The degree of compression on a backboard may not reflect the maximal compression seen by the cord. Unfortunately, in the absence of the rare visualization of partial or complete transection, the imaging modalities available give very little indication of the severity of the neural injury. It is distressingly true that two injuries that look precisely the same on arrival can make markedly different recoveries even with identical care. Finally, the one factor over which physicians have control that can alter recovery is hypotension. Taking a poly-trauma patient to theater for a major spine operation on an emergent basis risks unexpected deterioration during surgery because of missed or incompletely controlled hemorrhage sources, even if the resuscitation and initial evaluation have been perfect. It is probable that a significant episode of hypotension would obviate and even reverse any desired benefit from early surgery.

Given the marked difference between the laboratory and the clinical situation, it is important to base practice on clinical studies. Two recent studies have provided the best, albeit incomplete, evidence on this. Vaccaro et al performed PRCT of 64 patients with cervical SCI and documented cord compression who were randomized to surgery within 72 hours or surgery at greater than 5 days after injury [67]. They found no statistically significant differences between groups in motor recovery or Frankel scores. They did report increased cost for the late surgery group, presumably because of the associated increased acute hospital stay. Unfortunately, this study lost 32% of patients to long-term follow-up, did not stratify patients by injury or neurologic status, and did not use multivariate statistics. Additionally, a study of only 64 patients allows a very high probability of a Type 2 error, so little confidence should be placed on their negative finding.

Mirza et al performed a retrospective analysis of 30 patients from two institutions, one of which followed a policy of observation for 10 to 14 days. The other performed surgery within 3 days [68]. They reported that, although both groups showed statistically significant improvement between admission and discharge, those undergoing surgery within 3 days did significantly better than those whose surgery was delayed. They interpreted this as support for early surgery. Unfortunately, in addition to being a retrospective study with no long-term follow-up, this study neither controlled treatment practices at the two institutions nor adjusted for such differences through multivariate analysis. The 30 patients were

taken from a pool of 43 without control of selection. Also, there was no control for differences between institutions in examination, management, or surgical technique. Therefore, the difference between groups is better considered an interinstitutional difference than attributed specifically to surgical timing.

Finally, both studies, as well as others [69–72], have been forced to set their definition of early versus late surgery based on pre-existing practice or feasibility rather than on the extremely brief laboratory window of approximately 4 to 8 hours. In actuality, then, there are almost no clinical data on which to base the decision to perform emergent surgery. As such, it is recommended that emergent surgery be considered only in patients with no or minimal complicating injuries to other systems, where decompression can be expected to be accomplished within a very short period, when the risk of iatrogenic hypotension is negligible, and where conditions are favorable to the performance of such surgery in a safe, coordinated, and expedient fashion. Where such constraints do not hold, it is suggested that surgery be considered urgent rather than emergent and be performed at the earliest possible point following full medical stabilization, when the optimal operating conditions can be arranged. Such an approach should shorten hospital stay, minimize surgical and medical complications, facilitate care of other organ systems, and decrease acute care costs without jeopardizing the patient.

Steroids

Despite the fervor that occurred on initial publication and the enthusiasm that continues in popular and legal circles, the role of steroids in promoting recovery from SCI has become clouded. The initial North American SCI Study II (NASCIS II) report suffered from a number of important weaknesses [73,74]. Steroids were not beneficial in the total group, but, rather, the beneficial effect was found on posthoc analysis of the group treated within 8 hours. Some have argued that the effects were caused more by differences between placebo groups than treatment groups. There was no functional scale used to assess the utility of the reported recovery to the patients. A full discussion of this topic is beyond the scope of this article. The reader is referred to a recent evidence report that classified the use of steroids in SCI at the option level [75]. A widespread opinion at this time is that there is not sufficient evidence to establish steroid treatment in SCI as a standard of practice and that it would not be used in many cases if not for the legal ramifications in the United States.

A follow-up study on duration of treatment also suffers from methodologic problems. The NASCIS III study investigated whether there is an advantage to treating for 24 versus 48 hours [76,77]. They reported that groups where treatment was initiated between 3 and 8 hours following SCI did better when steroids were continued for 48 hours. Careful analysis of the 1-year follow-up report, using intent-to-treat analysis to make up for the attrition rate, suggests that there was no significant difference between groups in motor scores or functional scale scores (using the Functional Impairment Measure [FIM]) [74]. There were, however,

statistically more cases of severe pneumonia in those treated for 48 hours and trends towards more sepsis and a higher mortality in the same group. It is suggested that the clinician keep these caveats in mind when considering the duration of steroid treatment following SCI.

General care

The need for initial strict immobilization and the injury-induced alterations in motor, sensory, and autonomic function conspire to add complexity to the general care of the SCI patient. Particular areas of focus include pulmonary care, skin care, prophylaxis and treatment of deep venous thrombosis (DVT) or pulmonary embolism (PE), and management of the urinary system.

Pulmonary care

Pulmonary complications are the major cause of morbidity in SCI patients. Atelectasis and pneumonia may result from difficulties with clearing secretions and small spontaneous tidal volumes. Respiratory dynamics are hampered by paralysis of the chest or abdominal wall musculature. As such, these patients require meticulous pulmonary toilet and close surveillance for the development of infection. The development of amounts of pulmonary compromise that would be tolerated easily by others may prompt respiratory failure and require intubation in the SCI patient. For this reason, use of kinetic therapy tables during the period of mandatory immobilization is recommended. Following daily extubation parameters also may be useful in detecting early compromise of respiratory dynamics. As discussed previously, the necessity of tracheostomy should be considered during the early course of patients with mid- of high cervical SCIs.

Skin care

The combination of immobility and decreased or absent sensation greatly increases the risk of skin breakdown. The rapidity with which serious decubiti can occur is startling. As such, avoidance of pressure areas, close surveillance of all contact areas, and rapid management of any early signs of integument compromise is important. Patients should be mobilized off of the backboard at the earliest safe point. Orthoses should be well fit. Kinetic therapy tables can be very useful in preventing skin breakdown in immobilized patients. With or without such devices, however, the patient's back should be inspected routinely for signs of pressure. Early delivery of adequate nutrition should be pursued vigorously as both therapeutic and prophylactic.

Deep vein thrombosis/pulmonary embolism

The risk of DVT in trauma patients is raised by a factor of two in the presence of spinal fractures and a factor of three in patients with SCI [78,79]. The incidence of

PE is more difficult to estimate but may occur in approximately 10% of SCI patients [80]. Prevention of both DVT and PE is the primary goal. Sequential compression stockings are used commonly but, alone, they do not appear to be sufficient in SCI patients [81]. Low molecular weight heparin and adjusted-dose heparin titrated to raise the partial thromboplastin time to 1.5 times control are both effective, but they spawn concerns about bleeding at the injury site of during required surgery [82,83]. Because the risk of DVT is quite low during the first 3 days and does not peak until the end of the first week, one approach is to use sequential compression stockings for the first 7 days, not initiating anticoagulation until that point. Although many centers place inferior vena cava filters in patients with para- or quadra-paresis, there is a fair amount of controversy in this area [78,84,85]. It appears that the incidence of PE is lower in SCI patients with such filters than in historical controls. Such treatment can be considered in situations where the use of anticoagulants in contraindicated. The availability of temporary, removable filters can be useful in such circumstances.

Urinary system

In the past, urinary infections were the primary source of morbidity in SCI patients. Although this is no longer the case, management of the genitourinary system remains important. SCI patients generally have problems with retention, so an indwelling catheter is indicated during the acute period. After 1 to 2 weeks, this should be changed to an intermittent catheterization program. Vigilance for the development of urinary tract infection should be a continuous aspect of SCI care.

Summary

For both SCI and TBI, physicians are unable to affect reversal of the cellular injuries suffered at the time of trauma directly. Unfortunately, understanding such processes is just on the horizon. Physicians do, however, have significant influence on recovery through the avoidance of secondary insults to the injured nervous system. In keeping with trauma in general, the mechanism for this is focused and coordinated multi-disciplinary care originating at the earliest contact and continuing through acute care. Aggressive and pre-emptive attention to the ABC(D)s with attention to the needs of the injured nervous system, appropriate monitoring in all patients, meticulous medical management, and prompt surgical intervention when indicated have made marked improvements in outcome, particularly in TBI. Focusing on the basics and strict attention to detail appear to be the major roles played in the care of CNS trauma.

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