

Nursing Management of Adults with Severe Traumatic Brain Injury

AANN Clinical Practice Guideline Series



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Preface

In 1997, the American Association of Neuroscience Nurses (AANN) created a series of patient care guidelines, the *AANN Reference Series for Clinical Practice*, to meet its members' needs for educational tools. To better reflect the nature of the guidelines and the organization's commitment to developing each guideline based on current literature and evidence-based practice, the name of the series was changed in 2007 to the *AANN Clinical Practice Guideline Series*.

Traumatic brain injury (TBI) is a leading cause of disability worldwide. It is caused by a bump or blow to the head that affects how the brain normally works (National Center for Injury Prevention and Control, 2008). Because nurses frequently are the professionals who see the full impact of TBI and have the skills that can alter the course of a patient's recovery, it is important for nurses to have a valuable resource to help them achieve the best possible outcomes. This clinical practice guideline was developed in response to the 2006 AANN member needs survey, in which respondents identified information on the nursing management of patients with TBI as a priority for the organization. This guideline helps translate the latest research into an easy-to-use reference. The purpose of this document is to provide recommendations based on

current evidence that will help registered nurses, intensive care unit personnel, and institutions provide safe and effective care to adults with severe TBI. We recognize the developmental differences in the pediatric TBI population, and we will adapt these guidelines for these young patients at a future date.

As a result of the high profile of TBI, particularly injuries that are blast-related, new medical, nursing, and rehabilitation treatments are frequently emerging. Resources and recommendations must describe the best practices that can enable neuroscience nurses to provide optimal care for adults with severe TBI. Accordingly, adherence to these guidelines is voluntary, and the ultimate determination regarding their application must be made by practitioners in light of each patient's individual circumstances. This reference is an essential resource for nurses providing care to adults with severe TBI. It is not intended to replace formal learning, but rather to augment clinicians' knowledge base and provide a readily accessible reference tool. Nursing and AANN are indebted to the volunteers who have devoted their time and expertise to this valuable resource, which was created for those who are committed to excellence in the care of brain-injured patients.

I. Introduction

A. Purpose

The purpose of this document is to provide recommendations based on current evidence that will help registered nurses, intensive care unit personnel, and institutions provide safe and effective care to severely injured patients with traumatic brain injury (TBI). For the purposes of this guideline, *severe TBI* is defined as a brain injury incurred by a traumatic mechanism of injury with a resultant level of consciousness categorized by a Glasgow Coma Scale (GCS) score of 8 or lower. The goal of these guidelines is to offer evidence-based recommendations on nursing activities that have the potential to maximize outcomes for severe TBI. These recommendations are not inclusive of all activities that might improve outcomes, but reflect interventions commonly found in the literature that have been scientifically examined within the last decade. Not all recommendations concern activities independently performed by nurses, but nurses are responsible for implementing and monitoring the outcomes of these activities. The evidence presented here may help neuroscience nurses make appropriate choices when caring for patients with severe TBI.

B. Statement of the Problem

Severe TBI kills more than 50,000 people yearly and can result in lifelong functional, behavioral, and cognitive disabilities (Novack, 2000). Falls (28%), motor vehicle crashes (20%), being struck by/against impact (19%), and assaults (11%) are the leading causes of TBI (Langlois, Rutland-Brown, & Thomas, 2006). TBI rates are highest among people age 15–24 years and those older than age 65 years (National Center for Injury Prevention and Control, 1999) and occurs 1.5 times more often in men than in women (Novack). The lifetime cost of caring for a person with a severe TBI is estimated at more than \$3 million (Novack).

TBI has become the signature injury of military personnel involved in conflicts in the Middle East. In contrast to the civilian population, TBI in the military most often results from blast mechanism of injury. Of the 22,600 soldiers evacuated from Operation Enduring Freedom and Operation Iraqi Freedom to Walter Reed Army Medical Center, 28% were found to have at least a mild TBI (Warden, 2006). Among this group, 12% sustained penetrating injury of variable severity. Mild TBI affected fewer than 50% of patients in the sample.

TBI severity generally is categorized with a GCS score. Data compiled by the Traumatic Coma Data Bank established that a GCS score of 3–8 identified a severe injury. Additional criteria for a severe injury include loss of consciousness for longer than 6 hours and posttraumatic amnesia lasting longer than one week (Greenwald, Burnett, & Miller, 2003).

C. Search Strategy

A computerized search of Medline and the Cumulative Index to Nursing and Allied Health Literature (CINAHL) was performed using the names of specific nursing interventions with and without ICP or CPP as keywords. The search was primarily restricted to works in English published in 2000–2009 in which all or part of the sample included adults with severe TBI. Few studies were found on certain nursing interventions. In these instances, the search was expanded to include the years 1997–1999 for relevant works. The reference lists of identified articles also were searched for additional studies. Two neurotrauma nursing experts determined the level of evidence for each study included under every recommendation, summarizing the level of evidence for each recommendation.

D. Levels of Evidence Supporting the Recommendations

- Class I: Randomized controlled trial without significant limitations or metaanalysis
 - Class II: Randomized controlled trial with important limitations (e.g., methodological flaws or inconsistent results), observational studies (e.g., cohort or case-control)
 - Class III: Qualitative studies, case study, or series
 - Class IV: Evidence from reports of expert committees and/or expert opinion of the guideline panel, standards of care, and clinical protocols
- The Clinical Practice Guidelines and recommendations for practice are established based upon the evaluation of the available evidence (AANN, 2005, adapted from Guyatt & Rennie, 2002; Melnyk, 2004):
- Level 1 recommendations are supported by class I evidence.
 - Level 2 recommendations are supported by class II evidence.
 - Level 3 recommendations are supported by class III and IV evidence.

II. TBI Pathophysiology

TBI initially produces skull fractures, brain tissue disruption, and torn cerebral vessels that are managed by specific medical and surgical strategies. Within minutes, hours, or days of the primary injury, a secondary brain injury can occur.

A. Secondary Injury

This secondary injury involves multiple metabolic mechanisms that result from interruption of blood flow and oxygen to undamaged cells, producing anaerobic metabolism, inadequate synthesis of adenosine triphosphate, or cellular acidosis. The subsequent loss of energy-dependent ion transportation that controls cellular sodium, chloride, calcium, and water movement produces cytotoxic edema (Bayir, Clark, & Kochanek, 2003). These injuries initiate

an inflammatory response that, combined with the release of excessive neuronal calcium, precipitates a biochemical cascade of mediators that in turn precipitate the dangerous metabolic mechanisms of excitotoxicity and neuronal death (Hutchinson et al., 2007; Osteen, Moore, Prins, & Hovda, 2001; Tymianski & Tator, 1996; Wong, 2000). These processes are thought to extend the neuronal damage responsible for the severe physical and cognitive disabilities found in severely brain-injured patients. Preventing and/or interrupting metabolic mechanisms responsible for secondary brain injury may lessen the impact of the brain injury and improve short- and long-term outcomes.

Secondary injury also occurs as a result of altered cerebral blood flow (CBF). Within the first few hours after TBI, a decrease in CBF leaves the brain vulnerable to hypoperfusion due to hypotension or inadvertent hyperventilation. CBF increases 12–24 hours after the injury, leading to a mismatch between cerebral demand and supply. Often, intracranial pressure (ICP) increases as a result of the increased CBF. This increased flow lasts for 1–3 days and is followed by a decrease in CBF 4–15 days after the injury (Bouma & Muizelaar, 1992; Martin et al., 1997). The brain is especially vulnerable to secondary injury during this phase because CBF often is reduced. If CBF and cerebral oxygenation are not monitored, the decreased CBF may be missed.

Worsening secondary cellular injury also can manifest as intracranial hypertension and insufficient cerebral perfusion pressure (CPP). In addition, elevated ICP may intensify the processes involved in secondary brain injury, negatively affecting outcomes. It is well established that intracranial hypertension negatively affects morbidity and mortality. Over the past decades, preserving CPP via application of evidence-based protocols has resulted in significant reductions in mortality (Brain Trauma Foundation, American Association of Neurological Surgeons, & Congress of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care, AANS/CNS, 2007). ICP and CPP are known to be adversely affected by hyperthermia, hypotension, hypoxia, hypocarbia, and hypercarbia. It is the bedside nurse who intervenes to maintain ICP, manage CPP, and prevent situations that adversely affect these pressures.

Using evidence to guide practice, neuroscience nurses also can intervene to prevent complications commonly associated with TBI, such as deep vein thrombosis (DVT), hyperglycemia, and excessive protein loss. These guidelines are intended to provide neuroscience nurses with recommended interventions to promote optimal ICP and CPP and prevent DVT complications, inadequate nutrition, detrimental hyperglycemia, and seizures in adults with severe TBI.

B. Research Questions

These guidelines address the following research questions:

- What nursing interventions maintain or decrease ICP in patients with severe TBI?
- What nursing interventions maintain adequate CPP or increase CPP in patients with severe TBI?
- What monitoring modalities can successfully guide nursing interventions in severe TBI?
- What nursing interventions prevent DVT in patients with severe TBI?
- What nursing interventions promote adequate nutrition in patients with severe TBI?
- What nursing interventions prevent hyperglycemia in patients with severe TBI?
- What nursing interventions prevent seizures in patients with severe TBI?

III. Recommendations (Figure 1)

A. Maintaining or Decreasing ICP

1. Maintaining ICP at Less than 20 mm Hg Improves Outcomes (Level 1)

Uncontrolled intracranial hypertension leads to an absence of cerebral perfusion and results in brain death. Numerous studies have identified the ICP treatment threshold to be higher than 20 mm Hg. The original *Guidelines for the Management of Severe Head Injury* recommended that treatment be initiated for ICP thresholds above 20 mm Hg (Bullock, Chestnut, & Clifton, 1995).

2. Draining Cerebrospinal Fluid (CSF) Decreases ICP (Level 2)

The cranial compartment is a rigid box containing three components: the brain, blood, and CSF. The Monro-Kellie hypothesis states that a normal ICP can be maintained as one component increases as long as there is a corresponding decrease of another component. Therefore, decreasing one of the three components decreases ICP. As early as 1960, Lund demonstrated that removal of CSF via ventriculostomy temporarily decreased ICP (Lund, 1960). “The Critical Pathway for the Treatment of Intracranial Hypertension,” published in the *Guidelines for the Management of Severe Head Injury* lists ventricular drainage as the first step to take to reduce intracranial hypertension (Brain Trauma Foundation, American Association of Neurological Surgeons, & The Joint Section on Neurotrauma and Critical Care, 2000). Draining as little as 3 ml of CSF was found to decrease ICP by 10.1% relative to the baseline value for 10 minutes in 58 patients with severe TBI (Kerr, Weber, Sereika, Wilberger, & Marion, 2001). Protocols for CSF diversion range from time-dependent

(leave the drain open for 5 minutes, then close), CSF-volume-dependent (drain 5 cc then close), to continuous drainage (open all the time, closed at intervals to obtain an accurate ICP reading).

3. Do Not Induce Hyperventilation to Decrease ICP (Level 2)

Until the last decade, hyperventilation was routinely used to manage severe TBI. It initially was hypothesized that because hyperventilation induced vasoconstriction and decreased the blood component within the cranial vault, the resultant lowering of ICP was beneficial. Studies done in the 1990s demonstrated the vasoconstriction associated with hyperventilation also resulted in decreased cerebral blood flow and precipitated further cerebral ischemia (Bouma et al., 1992; Marion, Darby, & Yonas, 1991; Sioutos et al., 1995). One randomized study found that 3- and 6-month Glasgow Outcome Scale scores were significantly lower in the hyperventilation group compared to the normocapnia group (Muizelaar et al., 1991). After synthesizing the results from numerous early studies, *Guidelines for the Management of Severe Traumatic Brain Injury, 3rd Edition* recommended maintaining normocapnia in most patients with severe TBI (Brain Trauma Foundation et al., 2007).

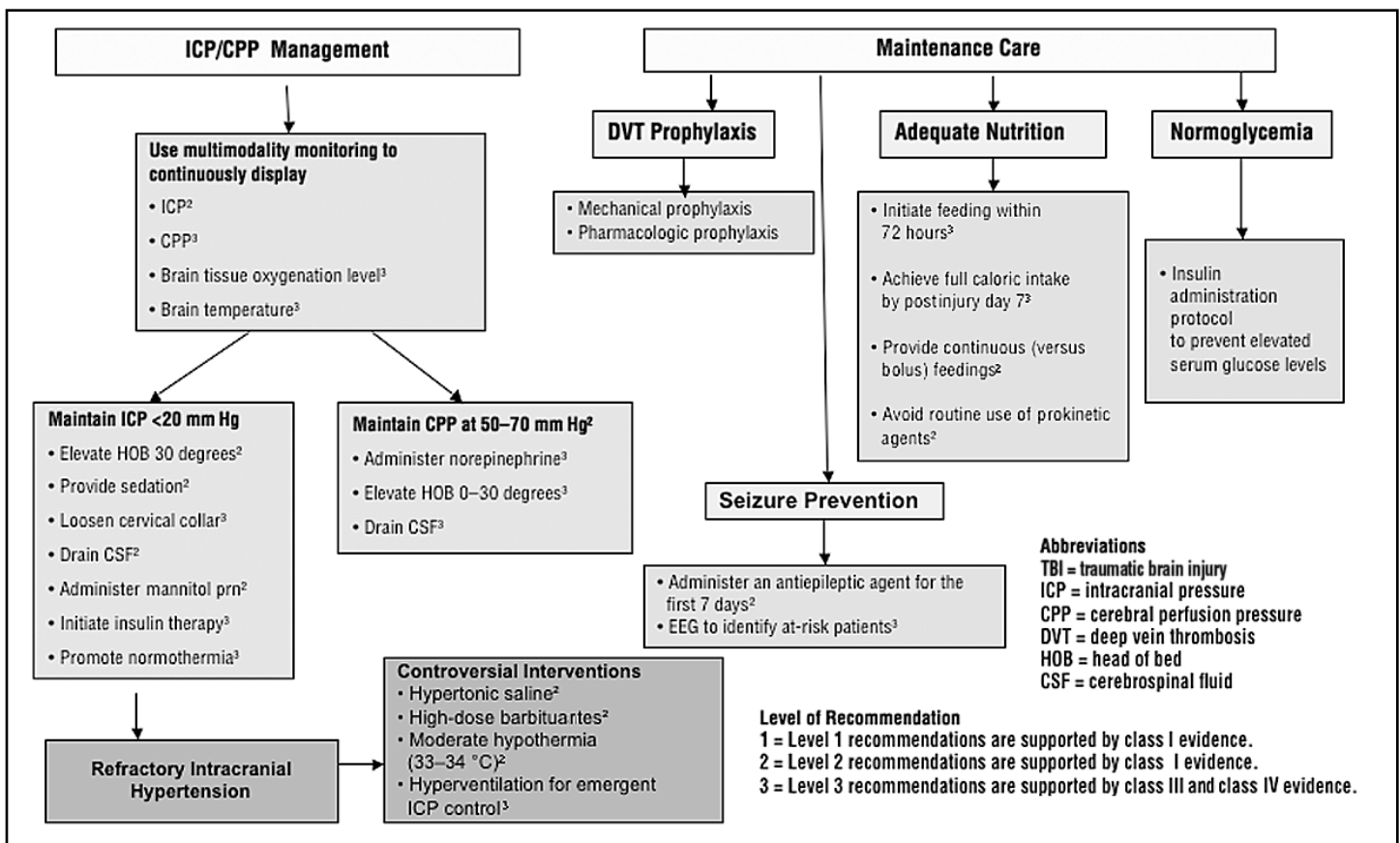
4. Administering Sedation Prevents ICP Increases (Level 2)

Both agitation and coughing in patients with severe TBI increase the cerebral metabolic rate for oxygen consumption, which could negatively affect secondary cellular brain injury. Endotracheal suctioning is a necessary intervention in severe TBI, but it may cause deleterious increases in ICP. A study of 17 patients with severe TBI found ICP was significantly higher and there was a significant decrease in CPP with endotracheal suctioning among patients who were inadequately sedated compared to patients who were well-sedated with propofol (Gemma et al., 2002). Inadequate sedation occurred when patients moved or coughed during suction. A randomized controlled trial of 42 patients with TBI found the use of propofol (rather than morphine) resulted in significantly lower ICPs by postinjury day 3, with less use of neuromuscular blockers, benzodiazepines, and barbiturates and less CSF drainage was required (Kelly et al., 1999).

5. Administering Mannitol Is Effective in Decreasing ICP (Level 2)

Infusions of mannitol immediately increase vascular volume and produce an osmotic effect within 15–30 minutes (Barry & Berman, 1961).

Figure 1. Treatment Algorithm: Clinical Practice Guidelines for the Nursing Management of Adults with Severe TBI



The diuretic effect of mannitol can cause increased sodium and serum osmolarity levels, and should be monitored at regular intervals. *Guidelines for the Management of Severe TBI, 3rd Edition* states “mannitol is effective for control of raised ICP at doses of 0.25 gms/kg to 1.0 gm/kg body weight” (Brain Trauma Foundation et al., 2007). The dose usually is held or limited by a serum osmolarity level >320 mOsm/L (Brain Trauma Foundation et al., 2000). A recent Cochrane Review comparing mannitol to other ICP-lowering agents found mannitol more beneficial than pentobarbital, but less beneficial than hypertonic saline in terms of impact on mortality (Wakai, Roberts, & Schierhout, 2007).

Mannitol is infused via intravenous bolus through a filter. Mannitol 20% contains 20 g of mannitol in 100 cc. Eighty percent of a 100 g dose appears in the urine within 3 hours of infusion.

6. Elevate the Head of the Bed (HOB) 30 Degrees to Maintain or Decrease ICP (Level 2)

Elevating the HOB is thought to promote intracranial venous return and increase CSF drainage from the head, resulting in decreased ICP (Fan, 2004). Four controlled studies with sample sizes ranging from 5 to 38 patients with severe TBI found significant decreases in ICP with HOB elevations of 30 degrees (Moraine, Berré, & Mélot, 2000; Ng, Lim, & Wong, 2004; Schulz-Stubner & Thiex, 2006; Winkleman, 2000). Increases to 45 degrees caused ICP to rise from the level found at 30 degrees in one study (Moraine et al.). A systematic review of 11 studies found that 9 studies demonstrated significantly decreased ICP at elevations of 30 degrees (Fan). All 9 studies included patients with severe TBI, with sample sizes ranging from 11 to 25. The effect size (to determine how effectively HOB elevation decreases ICP) was calculated to be moderate-to-large in the 5 studies for which such data were available.

7. Removing or Loosening Rigid Cervical Collars May Decrease ICP (Level 3)

Rigid cervical collars are used in severe TBI until spinal stability is confirmed. These collars may impede venous outflow and cause pain and discomfort, elevating ICP. Two controlled studies of patients with severe TBI ($N = 30$, $N = 10$) demonstrated significantly higher ICPs with the application of rigid cervical collars (Hunt, Hallworth, & Smith, 2001; Mobbs, Stoodley, & Fuller, 2002). The increases in ICP were greater in patients with a baseline ICP higher than 15 mm Hg (Hunt et al.).

8. Administering Intensive Insulin Therapy May Reduce ICP (Level 3)

Hyperglycemia is common in severe TBI and has a negative effect on outcome. Isolated patients

with severe TBI ($N = 33$) treated with intensive insulin therapy to maintain glucose levels lower than 110 mg/dl had lower mean and maximal ICPs than subjects in a randomized control group ($N = 30$) treated with insulin only when their glucose levels exceeded 220 mg/dl. The intensive insulin therapy group did not experience more hypoglycemic episodes and required less vasopressors to achieve the same CPP as the control group (Van Beek, Schoonheydt, Becx, Bruyninckx, & Wouters, 2005).

9. Maintaining Normothermia May Prevent ICP Increases (Level 2)

Hyperthermia is prevalent in the TBI population, with reported rates as high as 68% within 72 hours of injury (Rumana, Gopinath, Uzura, Valadka, & Robertson, 1998) and 79% in the first week postinjury (Thompson, Kirkness & Mitchell, 2007). In the stroke population, hyperthermia within the first 24 hours correlates with a mortality rate of 78%, compared with 2% in normothermic patients (Castillo et al., 1994). There have been no long-term outcome studies on the effects of normothermia in a TBI population. One descriptive study of 20 patients, 10 of whom sustained acute TBI, found an increase in brain temperature was associated with a significant rise in ICP; as fever ebbed, there was a significant decrease in ICP (Rossi, Zanier, Mauri, Columbo, & Stocchetti, 2001). Though an unpublished retrospective study of 26 patients with TBI found hyperthermic brain ICP was significantly higher than normothermic brain ICP, a subsequent retrospective study of 31 subjects (57.5% with a diagnosis of TBI) found no significant difference in the incidence of hyperthermic ICP and normothermic ICP (measured by brain and core temperature; Mcilvoy, 2001, 2007). Frequent-to-constant CSF drainage was thought to lower closed ICP readings, possibly affecting the difference between the incidence of hyperthermic and normothermic ICP.

B. Controversial Treatments for Refractory Intracranial Hypertension

It is estimated that 10%–15% of patients with severe TBI will develop elevated ICP that is resistant to standard forms of treatment (Allison, Domonoske, & Nates, 2000). The following modalities have demonstrated their efficacy in treating elevated ICP that is refractory to customary treatment, but a lack of Level-1 evidence, conflicting evidence, and possible complications make these treatments controversial. Given the potential for death and severe disability when intracranial hypertension is uncontrolled, some authors advocate use of these intensive therapies when other conventional measures have failed.

1. Inducing Moderate Hypothermia May Decrease ICP in Refractory Intracranial Hypertension (Level 2)

Animal studies have shown that hypothermia provides extensive neuroprotection against indirect cerebral ischemia (Bramlett, Dietrich, Green & Busto, 1997; van der Worp, Sena, Donnan, Howells, & Macleod, 2007). Multiple human studies have demonstrated decreased ICP with the induction of moderate hypothermia (33 °C–36 °C) in patients with severe TBI (Clifton, Miller, et al., 2001; Marion, Obrist, Carlier, Penrod, & Darby, 1993; Polderman, Tjong Tjin, Peerdeman, Vandertop, & Girbes, 2002b; Tokutomi, Miyagi, Morimoto, Karukaya, & Shigemori, 2004; Tokutomi et al., 2003). However, while ICP was decreased, induced hypothermia did not improve patient outcomes at 6 months postinjury in the National Acute Brain Injury Study on Hypothermia (Clifton, Miller, et al., 2001). This study has been criticized for design flaws, and several subsequent studies have reported significant improvement in neurological outcome and survival in TBI with induced hypothermia (Clifton, Choi, et al., 2001; Polderman, et al., 2002b; Polderman, Tjong Tjin, Peerdeman, Vandertop, & Girbes, 2002b; Polderman, van Zanten, Nipshagen, & Girbes, 2004; Zhi, Zhang, & Lin, 2003). *Guidelines for the Management of Severe TBI, 3rd Edition* found improved outcomes from studies conducted in single centers versus studies conducted in multiple centers (Brain Trauma Foundation et al., 2007).

In January 2008, the Brain Trauma Foundation issued a new Level 3 recommendation for optimal and cautious use of induced hypothermia for adults with TBI. This recommendation resulted from the findings of a new metaanalysis of hypothermia treatment for TBI that examined eight trials ($N = 781$) of comparable groups (Peterson, Carson, & Carney, 2008). The analysis suggests that hypothermia maintained for more than 48 hours reduces mortality and results in favorable neurological outcomes when they are measured 1–2 years postinjury. Of interest was the finding that hypothermia was of significant benefit only to patients who did not receive barbiturates.

Induced hypothermia is associated with complications. Pneumonia rates as high as 40%–45% have been reported in hypothermia trials; a Cochrane Library review cited the odds of patients developing pneumonia with hypothermia were nearly double than the odds for normothermic patients. The 2008 metaanalysis warns the increased risk of pneumonia may offset the benefits of hypothermia (Alderson, Gadkary,

& Signorini, 2004; Peterson et al., 2008). Electrolyte disturbances, cardiac arrhythmias, shivering, hiccups, and increased intensive care unit length of stays have been reported in patients receiving induced hypothermia (McIlvoy, 2005).

2. Administering Hypertonic Saline May Decrease ICP in Refractory Intracranial Hypertension (Level 3)

The exact mechanisms of the ways in which hypertonic saline reduces ICP are unknown. When used to resuscitate trauma patients, the vascular expansion of the infusion increases mean arterial pressure (MAP; Vassar et al., 1993). Numerous experimental studies have shown hypertonic saline reduces brain water through dehydration of regions of uninjured brain tissue with intact blood brain barrier (Bayir et al., 2003; Qureshi & Suarez, 2000). In studies of hypertonic saline, researchers have used concentrations of 2%–3%, 7.5%, and 23.4%, though there is no evidence that one concentration is more effective than others in reducing brain water volume (Qureshi & Suarez).

Studies conducted during the 1990s were plagued by small sample sizes and design flaws, yet two studies found hypertonic saline decreased ICP in severe TBI (Hartl, Ghajar, Hochleuthner, & Mauritz, 1997; Qureshi, Suarez, Castro, & Bhardwaj, 1999; Shackford et al., 1998). More recent studies examined not only the effect of hypertonic saline on ICP, but also its efficacy against mannitol. A 15-minute infusion of 7.2% hypertonic saline decreased ICP to 30% of baseline throughout the study period in 14 patients with moderate-to-severe TBI, producing a significant positive correlation between ICP and serum osmolality at 5 minutes postinfusion, and increasing CPP during the first hour postinfusion (Munar et al., 2000). A study of 20 patients with TBI randomly assigned to receive 2 ml/kg of 7.5% hypertonic saline or 20% mannitol infusions found the mean number of intracranial hypertension episodes per day and the duration of the episodes were significantly lower in patients receiving the hypertonic saline (Violet et al., 2003). Though the patients received equal infusion volumes, the hypertonic saline group received a higher osmolar dose. A greater decrease in ICP was found with infusions of 100 ml of 7.5% saline/6% dextran solution over 100 ml of 20% mannitol in a pilot randomized controlled trial of 9 patients with severe TBI (Battison, Andrews, Graham, & Petty, 2005).

Guidelines for the Management of Severe TBI, 3rd Edition suggests hypertonic saline may

effectively treat intracranial hypertension; however, the guidelines state there is not enough evidence to recommend its use at this time (Brain Trauma Foundation et al., 2007). There still are unanswered questions regarding the mechanism of action by which hypertonic saline decreases brain water, which concentration is most beneficial, and how the solution should be delivered (bolus versus constant infusion). Most protocols for the infusion of hypertonic saline include a bolus followed by a continuous infusion, titrated to maintain the minimum necessary sodium level to maintain ICP at <20 mm Hg (Levine, 2006). In addition, sodium levels and urine output require close monitoring.

3. Administering High-Dose Barbiturates May Decrease ICP in Refractory Intracranial Hypertension (Level 3)

High-dose barbiturates are thought to suppress cerebral metabolism, reducing cerebral metabolic demand and cerebral blood volume. No studies conducted in the last two decades have examined the effect barbiturates have on ICP or outcomes in adults with severe TBI. The Cochrane database summarized several older studies in which ICP was lowered in some patients with refractory ICP (Roberts, 2006). In these studies, mortality was the primary outcome and was reduced in patients treated with barbiturates. Complications including cardiac depression/hypotension occurred in 25% of patients, however. As a result of these older studies, the Cochrane Database of Systematic Reviews stated “There is no evidence that barbiturate therapy improves outcome. . . .The hypotensive effect of barbiturate therapy will offset any ICP-lowering effect on cerebral perfusion pressure.” When using this treatment paradigm, continuous electroencephalogram (EEG) monitoring or a bispectral index monitor should be used to guide this dose-dependent therapy (Bader & Arbour, 2005). After administration of a loading dose (typically 40 mg/kg), infusion rates of 4–8 mg/kg/hr usually are required to maintain a 50% burst suppression pattern on an EEG (Urwin & Menon, 2004). Hemodynamic stability must be achieved before instituting high-dose barbiturates.

4. Hyperventilation Rapidly Decreases ICP in Emergent Intracranial Hypertension (Level 3)

Despite the negative effects of hyperventilation, it is an effective intervention for rapidly reducing ICP (Stocchetti, Maas, Chieregato, & van der Plas, 2005). Level 3 evidence dating back to the 1950s supports the use of hyperventilation as a temporizing measure to reduce ICP (Brain Trauma Foundation et al., 2007). In the event this ICP therapy technique is used, advanced monitoring

techniques such as jugular venous oxygen saturation or brain tissue oxygenation should be considered to ensure adequate substrate delivery to the vulnerable brain (Brain Trauma Foundation et al.; Imberti, Bellinzona, & Langer, 2002; Oertel et al., 2002).

C. Maintaining Adequate CPP or Increasing CPP

1. Maintaining CPP Between 50–70 mm Hg Optimizes Cerebral Perfusion (Level 2)

CPP is defined as the MAP minus the ICP ($CPP = MAP - ICP$). This pressure gradient drives cerebral blood flow, improving the likelihood of adequate oxygen and metabolite delivery. Since the early 1990s, CPP management has been widely practiced, but consensus remains elusive on the optimal CPP level for severe TBI. A CPP low enough to promote ischemia will initiate secondary cellular injury cascades. Using fluids and vasopressors to aggressively raise CPP may cause pulmonary complications. *Guidelines for the Management of Severe TBI, 3rd Edition* cautions a CPP above 70 mm Hg and below 50 mm Hg should be avoided (Brain Trauma Foundation et al., 2007). The guidelines further state a threshold of 10 mm Hg above the target threshold may be important to avoid dips below a critical level. A general threshold of 60 mm Hg may be appropriate with further fine-tuning based on multimodality monitoring.

2. Administering Norepinephrine May Maintain Adequate CPP or Increase CPP (Level 3)

Vasopressors cause vasoconstriction and are routinely used to maintain or increase MAP for both systemic and cerebral perfusion in patients with severe TBI. The catecholamine vasopressors dopamine and norepinephrine are the principal vasopressors used with critically ill patients and the only vasopressors that have been investigated in patients with TBI. Norepinephrine, a potent alpha agonist and moderate beta 1 agonist, produces vasoconstriction while reflexively reducing heart rate. Dopamine’s predominant effects are dose-related; lower doses activate dopamine receptors in renal, mesenteric, coronary, and intracerebral vascular beds causing vasodilation, while higher doses activate alpha and beta 1 receptors resulting in vasoconstriction and increased heart rate (Zaritsky, 1994).

When CPP was increased to 65 mm Hg, 75 mm Hg, and 85 mm Hg first using either dopamine or norepinephrine in 10 patients with severe TBI, norepinephrine led to predictable and significant increases in cerebral flow velocity for each step of CPP increase, but the CPP increases with dopamine were variable and inconsistent (Steiner et al., 2004). Using a similar

design with 11 patients with severe TBI, raising CPP to 65 mm Hg and then 85 mm Hg using both norepinephrine and dopamine in a randomized order, the same investigators found that norepinephrine—not dopamine—resulted in a significant reduction in arterio-venous oxygen differences and a significant increase in brain tissue oxygen without deleteriously affecting ICP (Johnston et al., 2004).

A study of 16 patients with severe TBI with CPP maintained at 70 mm Hg with noradrenalin (norepinephrine), dopamine, and methoxamine found norepinephrine was safe and effective at doses of 0.5 mg–5 mg/hr, while dopamine was not as effective at doses greater than 10 mcg/kg/min (Biestro et al., 1998). One study used both norepinephrine and low-dose dopamine in 20 patients with severe TBI; use of both drugs to keep CPP higher than 60 mm Hg increased urine output and natriuresis (Benmalek et al., 1999).

These catecholamines can cause negative side effects such as skin ulcers and decreased blood flow to renal and mesenteric circulations, especially with prolonged use at high doses. There are many noncatecholamine vasopressors (i.e., phenylephrine) that also are used to raise CPP; however, no studies of their efficacy or advantages over catecholamines exist.

3. Elevating the HOB 0–30 Degrees May Maintain Adequate CPP or Increase CPP (Level 3)

While head elevation increases venous drainage from the head, it also can decrease perfusion. In a systematic review of the impact of HOB elevation on ICP and CPP, five of nine studies found no significant change in CPP when the HOB was elevated between 0 and 30 degrees (Fan, 2004). A study of 8 patients with TBI found CPP clinically improved with HOB elevations of 30 degrees (Winkleman, 2000). Studies in the ischemic stroke population have found that 0 degrees of elevation promotes a higher cerebral blood flow velocity (Wojner-Alexander, Garami, Chernyshev, & Alexandrov, 2005).

4. CSF Drainage May Be an Effective Treatment for Low CPP (Level 3)

Decreasing the volume of CSF by drainage decreases the total intracranial volume. In the 1950s, Ryder and colleagues (1953) hypothesized that draining CSF in patients with TBI would decrease the size of the ventricles, allowing for cerebral vessel dilation and improved cerebral perfusion. Kerr and colleagues (2001) demonstrated a 3 mm withdrawal of CSF in 58 patients with severe TBI resulted in a sustained 10.1% decrease in ICP and a 2.2% increase in CPP relative to baseline

CPP for 10 minutes. Though the 2.2% increase is of little clinical significance, it does support the thought that cerebral perfusion can be augmented after CSF drainage. Kinoshita and colleagues (2006) found that CSF drainage as a treatment for low CPP was as effective as mannitol administration in 26 patients with severe TBI, and those who received CSF drainage received less crystalloid infusion.

D. Monitoring Modalities

1. Continuous ICP Monitoring and Display Successfully Guide Nursing Interventions (Level 2)

ICP cannot be measured by CT scan. *Guidelines for the Management of Severe TBI, 3rd Edition* recommends monitoring ICP in all salvageable patients with an abnormal CT and a GCS score of 3–8 after resuscitation and in patients with a normal CT scan who have two or more of the following features: age over 40 years, motor posturing, or systolic blood pressure lower than 90 mm Hg (Brain Trauma Foundation et al., 2007).

2. Continuous CPP Monitoring and Display May Successfully Guide Nursing Interventions (Level 3)

CPP is determined by MAP minus ICP, and traditionally is computed by nurses at specified intervals and recorded on a flow sheet (not displayed continuously on a bedside monitor). Declining CPP may not be readily noticed until it drops below a specified low level. When patients with severe TBI were randomized to beds with prominent continuous CPP displays ($N = 79$) and compared to patients without CPP displays ($N = 78$), the odds of survival at hospital discharge were significantly better in the group with the continuous CPP display (Kirkness, Burr, Cain, Newell, & Mitchell, 2006).

3. Continuous Brain Tissue Oxygen (PbtO₂) Monitoring and Display May Successfully Guide Nursing Interventions (Level 3)

Secondary cerebral ischemia worsens outcomes for patients with severe TBI (Bouma, Muizelaar, Choi, Newlong, & Young, 1991; Chesnut et al., 1993). By directly measuring the partial pressure of oxygen in a region of the brain, changes in cerebral oxygenation can be detected and used to guide interventions to increase or maintain oxygen levels. Low-brain PbtO₂ has been significantly correlated with poor outcomes and increased mortality in patients with severe TBI (Bardt et al., 1998; Dings, Jager, Meixensberger, & Roosen, 1998; Ruwaida et al., 2003; Stiefel et al., 2005; Valadka, Gopinath, Contant, Uzura, & Robertson, 1998; van der Brink et al., 2000).

Two technologies make continuous measurement of brain tissue oxygenation possible: the LICOX System (Integra Neurosciences, Plainsboro, NJ) and the Neurotrend System (Codman & Shurtleff, Raynham, MA). No presently available brain oxygen probe concurrently measures ICP. There is debate over placing the monitors in injured or uninjured brain tissue. When placed in contused brain tissue, one study found P_{btO_2} was always below the hypoxic threshold of 10 mm Hg (Sarrafzadeh et al., 1998).

Cerebral tissue partial pressure of oxygen (PO_2) levels below 15–20 mm Hg may cause tissue infarction (Vespa, 2006; Zauner, Daugherty, Bullock, & Warner, 2002). *Guidelines for the Management of Severe TBI, 3rd Edition* recommends a treatment threshold for P_{btO_2} of less than 15 mm Hg (Brain Trauma Foundation et al., 2007). Studies have demonstrated that increasing fraction of inspired oxygen (FiO_2) increases P_{btO_2} (McLeod, Igielman, Elwell, Cope, & Smith, 2003; Reinert et al., 2003) and hyperventilation decreases P_{btO_2} (Sarrafzadeh, Kiening, Callsen, & Unterberg, 2003; Schneider et al., 1998).

4. Monitoring and Displaying Brain Temperature May Successfully Guide Nursing Interventions (Level 3)

There is clear evidence that hyperthermia is prevalent in patients with acute brain injury (Albrecht, Wass, & Lanier, 1998; Kilpatrick, Lowry, Firlik, Yonas, & Marion, 2000). Elevated core temperatures contribute to increased lengths of stay and have been strongly associated with poor outcomes in severe TBI (Diringer, Reaven, Funk, & Uman, 2004; Geffroy et al., 2004; Jiang, Gao, Li, Yu, & Zhu, 2002). Though the majority of animal hyperthermia studies used brain temperature as the primary measure of temperature, almost none of the human TBI studies did so. Brain temperature has been found higher than all measures of core temperature in all published studies that statistically compared the two measurements and did not involve cooling therapies (Mcilvoy, 2004, 2007). In the absence of brain temperature monitoring, the likelihood of detecting a brain fever is limited.

The level of hyperthermia that constitutes a fever differs widely in the literature. The Society of Critical Care Medicine defines fever in the intensive care unit as a temperature of 38.3 °C (100.9 °F; O'Grady et al., 1998), while the *AANN Core Curriculum for Neuroscience Nursing* defines a fever as a temperature greater than 38.0 °C (100.4 °F; March et al., 2004). Monitoring brain temperature and maintaining brain normothermia may potentially reduce TBI morbidity and mortality. Reducing brain temperature

remains problematic, however. Antipyretic therapy alone or combined with traditional physical cooling blankets has been shown to be effective in decreasing temperature in 40%–50% of patients with neurotrauma (Mayer et al., 2001; Mcilvoy, 2001; Stocchetti et al., 2002). Newer cooling systems that incorporate body pads more successfully reduce fever, but they can cause shivering (Carhuapoma, Gupta, Coplin, Muddasir, & Meratee, 2003; Mayer et al., 2004).

E. Preventing DVT

1. Pharmacologic Treatment May Be Safe for DVT Prophylaxis (Level 3)

As its number one national patient safety practice, the Agency for Healthcare Research and Quality (AHRQ) recommends the use of prophylaxis to prevent venous thromboembolism for at-risk patients. A traumatic mechanism of injury initiates inflammation and coagulation cascades that disturb the fibrinolytic process, increasing the likelihood pathologic thrombi will occur (Kudsk et al., 1989). An examination of the American College of Surgeons' National Trauma Data Bank found that severe TBI increases DVT risk by 1.24 times above the risk for trauma patients who do not have head injuries, and that being on a ventilator for more than 3 days (which is typical for patients with severe TBI) increases DVT risk more than eightfold (Knudson, Ikossi, Khaw, Morabidto, & Speetzen, 2004). DVT rates as high as 25% have been reported in isolated TBI cases (Denson et al., 2007).

Two major pharmacologic agents are used for DVT prophylaxis: low-dose heparin (LDH) and low-molecular-weight heparin (LMWH). An AHRQ metaanalysis examined all randomized controlled and nonrandomized studies to examine the effectiveness of LDH in trauma patients; no difference was found in DVT incidence when LDH, LMWH, and no prophylaxis were used (Velmahos et al., 2000). The majority of studies examining pharmacologic prophylaxis exclude TBI because of the risk of causing or increasing intracranial bleeding.

Norwood and colleagues (2002) examined the use of LMWH within 24 hours of traumatic intracranial hemorrhage in 150 patients. LMWH administration continued until discharge. Six patients' (4%) CT scans worsened after LMWH was initiated, but all survived hospitalization. Kim and colleagues (2002) administered LDH to 47 patients with severe TBI within 72 hours of injury, and to 17 patients with severe TBI later than 72 hours postinjury. No patient in the early group had an increase in intracranial bleeding on CT scan

or deterioration on neurological examination, and there was no statistical difference between DVT rates between the two groups.

2. Applying Mechanical Prophylaxis on Admission May Prevent DVT in Patients Who Cannot Receive Immediate Pharmacologic Prophylaxis Due to Risk of Bleeding (Level 3)

Graduated compression stockings (GPS) used alone have been found to effectively diminish DVT risk in hospitalized patients, but they are more effective when combined with another method of prophylaxis (Amaragiri & Lees, 2003). Knee-high stockings are as effective as thigh-length stockings, and they reduce costs and are easier to apply (Sajid et al., 2006). The use of mechanical prophylaxis such as GPS and/or intermittent pneumatic compression (IPC) devices are recommended for patients with severe TBI who have a high risk of bleeding, according to recommendations established during the Seventh American College of Chest Physicians' Conference on Antithrombotic and Thrombolytic Therapy and *Guidelines for the Management of Severe TBI, 3rd Edition* (Geerts et al., 2004; Brain Trauma Foundation et al., 2007). However, in a study of 32 patients with severe TBI that compared no prophylaxis ($N = 18$) with use of IPC devices ($N = 14$), 28% of the IPC group developed a pulmonary embolism (Gersin et al., 1994). Plantar venous intermittent compression devices (A-V foot pumps) increase venous blood flow in the popliteal vein by 250% and have been found effective in preventing DVT in patients with blunt lower extremity skeletal trauma when compared with IPC devices or combined with delayed enoxaparin (Elliott et al., 1999; Spain, Bergamini, Hoffman, Carillo, & Richardson, 1998; Stannard et al., 2006).

F. Adequate Nutrition

1. Initiating Adequate Nutrition Within 72 Hours of Injury May Improve Outcomes (Level 3)

The metabolic expenditure in isolated comatose patients with TBI is at least 100%–180% higher than what would be expected in noninjured people (Clifton, Robertson, & Grossman, 1989). Though studies on adequate nutrition have not demonstrated decreases in acute care length of stay, a study of the effect of malnutrition on rehabilitation length of stay found that patients with malnutrition had lengths of stay that were 28 days longer than patients with adequate nutrition (Denes, 2004).

Two systematic reviews, one examining 11 studies and the other 30 studies, found a trend toward improved mortality and less disability with early feeding in patients with severe TBI (Krakau,

Omne-Ponten, Karlsson, & Borg, 2006; Perel et al., 2006). *Guidelines for the Management of Severe TBI, 3rd Edition* recommends patients be fed so that full caloric requirements are met by postinjury day 7 (Brain Trauma Foundation et al., 2007).

2. Providing Continuous Intra-gastric Feeding May Improve Tolerance (Level 3)

Continuous feeding was better tolerated and achieved 75% of nutritional goals faster than bolus feeding in 152 consecutive patients admitted to a neurosurgical intensive care unit (20% of whom had sustained a severe TBI; Rhoney, Parker, Formean, Yap, & Coplin, 2002). Feedings via percutaneous endoscopic gastrostomy in 118 patients with moderate-to-severe TBI was well-tolerated without complication in 97% of patients (Klodell, Carroll, Carrillo, & Spain, 2000).

3. Prokinetic Agents Have Shown No Effect on Feeding Tolerance (Level 2)

Gastric feeding tolerance is variable in patients with TBI. Prokinetic agents are commonly used to facilitate gastric feeding; however, published papers lack scientific support for this practice. A prospective randomized double-blind study of 19 patients with severe TBI that compared metoclopramide with normal saline found no difference in feeding intolerance or complication rates between the groups (Nursal et al., 2007). Prokinetic agents demonstrated no improvement in feeding tolerance in 57 patients in barbiturate-induced comas for refractory intracranial hypertension (Bochicchio et al., 2006). The amount of time it took to achieve nutritional goals was not reduced with the use of prokinetic agents in a neurosurgical ICU in which 20% of patients had severe TBI (Rhoney et al., 2002). While not evaluated in patients with TBI, there is some evidence that erythromycin is more effective than metoclopramide for critically ill patients (Berne et al., 2002; Boivin & Levy, 2001; Chapman, 2000; Nguyen, Chapman, Fraser, Bryant, & Holloway, 2007). Its use is limited secondary to concerns of cardiac toxicity and development of bacterial resistance.

G. Glycemic Control

1. Administering intensive insulin therapy for serum glucose greater than 110 mg/dL improves outcomes (Level 2)

Glucose levels exceeding 170 mg/dl during the first 5 days post-severe TBI correlate with prolonged hospital length of stay and increased mortality (Jeremitsky, Omert, Dunham, Wilberger, & Rodriguez, 2005). Administering intensive insulin therapy for elevated serum glucose can improve outcomes (a Level 2 recommendation). A glucose level higher than 200 mg/dl that goes

untreated during the first 24 hours post-severe TBI has been associated with worse outcomes and is related to increased ICP and impaired pupillary reaction (Rovlias & Kotsou, 2000). Admission serum glucose values higher than 150 mg/dl were associated with higher mortality but were not associated with extended Glasgow Outcome Scale scores in severe TBI 6 months postinjury (Vespa et al., 2006). The IMPACT study examining the prognostic value of admission laboratory parameters in TBI found the strongest effect in increasing levels of glucose to poorer outcome (Van Beek et al., 2007).

Intensive insulin therapy for patients with glucose levels higher than 110 mg/dl ($N = 33$) resulted in fewer seizures, less diabetes insipidus, and improved independent functioning at 1 year post-TBI, compared to outcomes experienced by patients who received insulin for glucose levels higher than 220 mg/dl ($N = 30$; Van Beek et al., 2005). When comparing glucose reduction to 120–150 mg/dl in 33 patients with TBI to patients with a glucose reduction to 90–120 mg/dl (in 14 patients with TBI), a reduction in microdialysis glucose by 70% of baseline was found in the 14 patients with tighter glucose control, compared to a 15% reduction in the 33 patients with the looser insulin protocol (Vespa et al., 2006). A significantly increased incidence of microdialysis markers of cellular distress (such as glutamate and lactate/pyruvate ratios) and an increase in the global oxygen extraction fraction was noted in patients with tighter glucose control. Because the brain increases the need for glucose to supply restorative pathways, this study's authors believed a reduction in the supply of glucose along with increased signs of metabolic distress may not be advantageous in an injured brain (Vespa et al.). Further research is needed to determine the level of glycemic control that achieves optimal outcomes for patients with TBI.

Subcutaneous insulin administration has been shown to be unsafe and less effective than intravenous insulin administration in critically ill patients (Brown & Dodek, 2001; Digman, Borto, & Nasraway, 2005). Several nurse-driven insulin infusion protocols demonstrate successful control of hyperglycemia in critical care units (Collier et al., 2005; Dilkhush, Lannigan, Pedcroff, Riddle, & Tittle, 2005; Goldberg et al., 2004; Osbourne et al., 2006).

H. Preventing Seizures

1. Administering Antiepileptic Drugs Decreases the Incidence of Early Posttraumatic Seizures (Level 2)

Seizure activity is a known cause of secondary brain injury, causing increased metabolic demand and neurotransmitter release. Certain pathology types (subdural hematoma, contusion) carry higher risk for the development of early seizures. Other mechanical factors such as removal of intracerebral hematoma or dural penetration by injury also have been correlated with increased risk of seizure (Temkin, 2003; Wiedemayer, Triesch, Schafer, & Stolke, 2002). The timing of seizure activity after trauma is categorized as acute/immediate (within 24 hours of injury), subacute/early (within the first 2–7 days postinjury) and late (after 7 days; Brain Injury Special Interest Group of the American Academy of Physical Medicine and Rehabilitation, 1998). Phenytoin and valproate administration after TBI has been shown to decrease the risk of early posttraumatic seizures without effect on the development of late seizure disorders (Chang & Lowenstein, 2003; Schierhout & Roberts, 2001). *Guidelines for the Management of Severe TBI, 3rd Edition* recommends the use of anticonvulsants to decrease the incidence of posttraumatic seizure within the first 7 days of injury when the brain is particularly vulnerable to secondary injury (Brain Trauma Foundation et al., 2007). Chronic prophylaxis should be avoided, as the current body of literature fails to demonstrate an impact on the development of late seizures and there is a high side-effect profile (Chang & Lowenstein).

2. EEG Technology May Help to Identify Patients at Risk for Seizures (Level 3)

Not all seizure activity is accompanied by convulsive activity, and not all early seizures occur immediately posttrauma. Continuous EEG monitoring among 70 patients with TBI found a 33% seizure incidence that occurred 74 ± 47 hours after trauma (Ronne-Engstrom & Winkler, 2006). Continuous EEG monitoring has been used to identify a 20% seizure incidence with 50% of patients identified as nonconvulsive (Vespa & Nuwer, 2000).

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