

## Prehospital management of traumatic brain injury

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The aim of this study was to review the current protocols of prehospital practice and their impact on outcome in the management of traumatic brain injury. A literature review of the National Library of Medicine encompassing the years 1980 to May 2008 was performed. The primary impact of a head injury sets in motion a cascade of secondary events that can worsen neurological injury and outcome. The goals of care during prehospital triage, stabilization, and transport are to recognize life-threatening raised intracranial pressure and to circumvent cerebral herniation. In that process, prevention of secondary injury and secondary insults is a major determinant of both short- and long-term outcome. Management of brain oxygenation, blood pressure, cerebral perfusion pressure, and raised intracranial pressure in the prehospital setting are discussed. Patient outcomes are dependent upon an organized trauma response system. Dispatch and transport timing, field stabilization, modes of transport, and destination levels of care are addressed. In addition, special considerations for mass casualty and disaster planning are outlined and recommendations are made regarding early response efforts and the ethical impact of aggressive prehospital resuscitation. The most sophisticated of emergency, operative, or intensive care units cannot reverse damage that has been set in motion by suboptimal protocols of triage and resuscitation, either at the injury scene or en route to the hospital. The quality of prehospital care is a major determinant of long-term outcome for patients with traumatic brain injury.

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**KEY WORDS** • hypotension • hypoxia • intracranial hypertension • outcome assessment • traumatic brain injury

**T**REATMENT of patients with TBI begins at the time of impact. By its nature, trauma demands focused attention on the most paramount life-threatening problems. Less urgent problems are relegated to a lesser priority. However, less urgent does not necessarily equate with less important. We performed a literature search of the National Library of Medicine encompassing the years 1980 to May 2008 using search terms pertinent to prehospital care, emergency triage, trauma system organization, and brain injury. The goal of this review was to evaluate the critical role that prehospital management plays in the pathophysiology and outcome of TBI. By the time the patient is in the emergency department, the damage of primary injury has made its mark and the processes of secondary injury have been set in motion. The quality of the care rendered in the prehospital setting is as important, if not more important, than the time taken to

reach definitive treatment. The key goals of the emergency response team are to stabilize and treat damage from the primary injury, to abrogate or minimize secondary injury, and to prevent secondary insults at all costs. With comparatively few available resources, care in the field (prehospital setting) is a critical determinant of outcome for patients with TBI.

### Epidemiology

In the US every year, 1.4 million people sustain a TBI. Trauma is the leading cause of death and disability in children and young adults, and TBI accounts for approximately 52,000 (or one-third) of all deaths.<sup>148</sup> Roughly twice this number suffer permanent neurological deficits. The severity of TBI is categorized based on the GCS score. Severe TBI (GCS Scores from 3 to 8) comprises approximately 10% of TBI, moderate head injury (GCS scores from 9 to 12) comprise approximately another 10%, and the vast majority of TBI (~ 80%) is classified as mild head injury, presenting with a GCS Score ranging from 13 to 15.<sup>78</sup> In the last 30 years, dedicated trauma programs have demonstrated that aggressive prehospital programs reduce morbidity and death from TBI.<sup>1,7,9,61,82</sup>

*Abbreviations used in this paper:* CBF = cerebral blood flow; CPP = cerebral perfusion pressure; GCS = Glasgow Coma Scale; ICP = intracranial pressure; OR = odds ratio; RSI = rapid sequence intubation; TBI = traumatic brain injury.

### Mechanisms of Injury

An understanding of the pathophysiological mechanisms of primary and secondary injury are crucial to setting optimal protocols of care to enable further advances in the outcome of patients with TBI. Primary injury occurs at the time of impact and is not reversible. Direct damage involves contact energy transfer and inertia energy transfer. In addition to these mechanical forces to the brain itself, primary injury also occurs to the cerebral vasculature leading to vessel shear and disruption. Secondary injury evolves as a cascade of cellular events that are set in motion at the time of the primary impact. Within the first hours after injury a complex array of inflammatory, excitotoxic, oxidative stress, metabolic, vascular, and mitochondrial mechanisms have been activated and each has progressed to initiate further injury. The various components of secondary injury interact with each other in a multiplicative rather than additive fashion. Secondary injury is a direct consequence of the primary impact, whereas secondary insults are discrete processes, often iatrogenic, that occur independently of the primary impact. Improved outcomes following aggressive treatment of mass lesions, hypoxia, hypotension, and fluid and electrolyte abnormalities have shown that secondary events following TBI are preventable and treatable.<sup>160</sup>

### Prevention of Secondary Injury in the Prehospital and Emergency Setting

Increased ICP, cerebral edema, cerebral dysautoregulation, and alterations in brain metabolism are inherent sequelae of the primary brain injury. Exogenous or iatrogenic events exacerbate these secondary injury processes. Patients with multiple traumas frequently incur injuries that compromise cardiopulmonary status, and they are therefore particularly vulnerable to secondary injury. Secondary insults are common and are independent predictors of poor outcome in patients with TBI.<sup>28,47,102</sup> Depth and duration of hypotension, but not hypoxia, has been shown to trend in a dose-response manner with the 3-month functional Glasgow Outcome Scale score.<sup>6</sup> In contrast, hypoxia and hypotension do not have nearly as profound an effect on outcome in patients with extracranial trauma. The prehospital guidelines were developed by the Brain Trauma Foundation ([www.braintrauma.org](http://www.braintrauma.org)) to standardize acute TBI care and prevent secondary injuries and insults.<sup>19,59,146</sup> Implementation of these guidelines with prevention and treatment of secondary injury in the early phases of care significantly improves patient outcomes following severe TBI.<sup>160</sup>

#### Brain Oxygenation

Airway obstruction and aspiration are major causes of death in patients who die of treatable head injuries.<sup>149</sup> Under normal physiological circumstances, the brain is dependent on aerobic metabolism to maintain the high adenosine triphosphate requirements of neuronal function, and energy failure occurs after a few minutes of anaerobic metabolism.<sup>104,138</sup> Apnea, even for just a brief period, accompanies most head injuries.<sup>47,103</sup> In response

to hypoxia, a compensatory increase in CBF occurs, but not until the partial pressure of O<sub>2</sub> drops to < 50 mm Hg.<sup>50,86,101</sup> Results from the Trauma Coma Data Bank showed that a low PaO<sub>2</sub> ( $\leq$  60 mm Hg) occurred in 46% of 717 admissions to the emergency department.<sup>26</sup> Arterial desaturations to this degree, even for a short time, were associated with a 50% mortality rate and 50% severe disability among survivors.<sup>143</sup> Multiple studies have confirmed that a low PaO<sub>2</sub> (< 60 mm Hg) correlates with worsened patient outcomes.<sup>23,28,104,156</sup>

Although airway control is intuitively important, a few studies have unexpectedly reported worse outcomes for patients with TBI who were intubated in the field.<sup>14,33,37,41,53,110,158</sup> In the field, hypoxia is defined as apnea, cyanosis, an O<sub>2</sub> saturation < 90%, or a PaO<sub>2</sub> < 60 mm Hg. Marked degrees of hypoxia to saturations < 70% are common during intubation with 57% of patients experiencing transient hypoxia lasting a mean of 2.3 minutes.<sup>51</sup> The risk of desaturation associated with intubation is dependent on the starting O<sub>2</sub> saturation and occurs 100% of the time if the O<sub>2</sub> saturation is  $\leq$  93%, versus 6% if it is > 93% ( $p < 0.01$ ).<sup>38</sup> A comparison of 1797 patients with severe TBI who were intubated in the prehospital setting with 2301 patients who were intubated in the emergency department demonstrated a 4-fold increase in death and a significantly higher risk of poor neurological and functional outcome in the group of patients who were intubated in the field.<sup>158</sup>

Endotracheal intubation is associated with risks of increased ICP, aspiration, and hypoxia. The detrimental effects of aspiration or hypoxia before arrival of the emergency response team may not be reversible.<sup>38</sup> Positive pressure ventilation can increase intrathoracic pressure, which may decrease venous return, and in a hypovolemic patient this can impair CPP. Furthermore, sedative agents used during RSI can cause hypotension.<sup>39</sup> Prehospital intubation may also delay transport. Importantly, endotracheal intubation in the field predisposes the patient to an increased incidence of overly aggressive hyperventilation that has been shown to adversely affect outcome.<sup>34,36,110</sup> A series of 851 patients with TBI, prehospital intubation, and a range of PCO<sub>2</sub> levels on arrival to the emergency department (17%, PCO<sub>2</sub> < 30; 47%, PCO<sub>2</sub> 30–39; and 26%, PCO<sub>2</sub> > 40) were divided into 2 groups. Those within the target PCO<sub>2</sub> range of 30–39 had a mortality rate of 21%, whereas those whose PCO<sub>2</sub> persistently remained outside the target range experienced a mortality rate of 34%.<sup>159</sup> With increasing head severity, there was an increased survival benefit for patients who attained their goal PCO<sub>2</sub> level.

Airway management and the prevention of hypoxia is a priority.<sup>20</sup> All patients should receive supplemental O<sub>2</sub> to maintain saturations > 90%. In the prehospital setting, intubation has been a mainstay procedure in the treatment of patients with severe TBI and GCS scores  $\leq$  8, both for maintenance of good oxygenation and prevention of aspiration. The prehospital guidelines of the Brain Trauma Foundation recommend that unconscious or unresponsive patients with GCS scores  $\leq$  8 or those unable to maintain an adequate airway, and those with hypoxemia (arterial O<sub>2</sub> saturation < 90%) despite supplemental

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O<sub>2</sub>, should be intubated. In the prehospital setting of a comatose patient with a head injury, orotracheal intubation is preferred over nasotracheal intubation because the status of possible basal skull fractures is unknown and noxious stimulation of the nares can elevate ICP. Intubation not only enables adequate oxygenation, but can also alleviate hypercarbia that itself can worsen ICP. In the absence of signs of raised ICP, patients should not undergo hyperventilation prophylactically.

A consensus panel has addressed the question of poorer outcomes in prehospital patients with TBI who have been intubated. This panel found no prospective controlled trials to adequately address the efficacy of paramedic RSI for severe TBI.<sup>35</sup> Concerns were raised regarding the use of a GCS score alone to identify patients who warrant intubation.<sup>43</sup> The GCS score provides no information as to patient oxygenation status and there is poor interobserver reliability for this score.<sup>8,65</sup> Furthermore, the force of the impact is often sufficient to induce a ventilatory pause, the duration of which is proportional to the force, and early assessments of GCS scores immediately after injury may be inaccurate. The consensus panel suggested that other factors and methods such as pulse oximetry and transport time be incorporated, in addition to the GCS score, to define candidates for intubation.<sup>35</sup> Pulse oximetry and capnometry were recommended for monitoring depth of sedation during conscious sedation. Importantly, success of intubation with RSI does not alone improve outcome.<sup>37,42</sup> The continued treatment of the ventilated patient with careful monitoring of end-tidal CO<sub>2</sub> and prevention of hyperventilation is paramount to achieving improved outcomes in the field.<sup>36,122</sup>

### *Hypotension*

Numerous studies have shown a significant association between hypotension and poor outcome in patients with head injuries.<sup>16,26,27,56,92,95,111,132,143,156</sup> In a study of 613 patients from the Traumatic Coma Data Bank, a single episode of hypotension (systolic blood pressure < 90 mm Hg) in the field doubled the mortality rate.<sup>27,120</sup> Estimates suggest that 8–13% of patients with severe head injuries are hypotensive at the injury scene or in the emergency department.<sup>26</sup> Isolated head injury does not cause hypotension. The brain's ability to extract O<sub>2</sub> protects it from hypoxia as long as cerebral perfusion is maintained.<sup>26</sup> In accordance with this concept, hypotension has been reported to be a stronger predictor of poor outcome than hypoxia.<sup>6,26</sup> Cerebral ischemia is evident in the vast majority of patients who die of head injury.<sup>66</sup> Secondary ischemia is a manifestation of the loss of autoregulation and is more common and more severe with increasing severity of brain injury.<sup>25,95,99</sup> In the absence of intact autoregulation, brain perfusion becomes passively dependent on the systemic blood pressure and hypotension leads to hypoperfusion and brain ischemia. Furthermore, in experimental animal models, mechanical injury decreases the threshold for ischemic damage and neuronal loss.<sup>77</sup> Low CBFs are frequent in the early hours following head injury, and even in the absence of blood loss a brief hypotensive episode can initiate irreversible cell death mechanisms in injured neurons.<sup>116</sup>

Despite the strong association between poor outcome and hypotension in patients with head injuries, the general trauma literature has questioned the value of time spent securing intravenous access and administering fluids in the field.<sup>87,131</sup> It has been argued that obtaining intravenous access is time-consuming and the small volumes infused during short transports may not significantly affect outcome.<sup>139</sup> Arguments in favor of delayed resuscitation with “permissive hypotension” include reports that administration of intravenous fluids to actively bleeding patients, before definitive surgical control, may increase blood loss because of hemodilution, higher blood pressures, impaired thrombus formation, and clot disruption.<sup>12,117</sup> For patients with severe head injuries, delayed fluid resuscitation risks significant secondary injury that occurs when CPP falls.<sup>17,124,125</sup> Patients with head injuries treated with delayed resuscitation have been shown to experience progressive intracerebral swelling and increased ICP, as well as higher lactate/pyruvate ratios as a result of delayed restoration of CBF.<sup>2</sup>

Management of hypotension in the field improves outcome for patients with severe TBI.<sup>64,150</sup> In children, systolic pressure goals are lowered in an age-dependent manner.<sup>83</sup> Intravenous fluids should be administered to avoid hypotension or to minimize the duration and extent of hypotension. The scalp has a rich blood supply and scalp lacerations should be addressed as a treatable cause of hypotension. Excessive bleeding consumes coagulation factors and platelets. Administration of intravenous fluids in the setting of excessive bleeding may further worsen coagulopathy through mechanisms of dilution of clotting factors and platelets, hyperchloremia leading to acidosis, hypocalcemia, and hypothermia.<sup>58</sup>

The optimal fluid for resuscitation has not been clearly determined. Intravenous fluids should be isotonic to reduce brain swelling and cerebral edema. Normal saline is preferred over lactated Ringer solution, and solutions containing 5% dextrose (D5W) should be rigorously avoided. Hypertonic saline expands intravascular volume by 4- to 10-fold that of the volume infused.<sup>49</sup> Hypertonic saline provides the potential benefit of enabling blood pressure stabilization with smaller volumes. It supports CPP without aggravating leakage and extravascular fluid accumulation that leads to cerebral edema and increased ICP, which occurs following infusion of high volumes of isotonic fluids.<sup>48</sup> Hypertonic saline also acts in a fashion similar to mannitol and induces an osmotic diuresis that assists treatment of raised ICP. Resuscitation with hypertonic saline doubles the survival rate of patients with TBI who present with hemorrhagic shock.<sup>151,155</sup> In subgroup analyses, patients with TBI and GCS scores ≤ 8 are most likely to benefit from hypertonic saline.<sup>150,152</sup> However, studies have yet to demonstrate an improvement in long-term neurological outcomes for patients with head injuries resuscitated using hypertonic saline.<sup>31</sup> A prospective intervention trial is planned to compare hypertonic saline, hypertonic saline with dextran 6%, and normal saline in a subgroup of patients with TBI and GCS scores ≤ 8, both with and without hypovolemic shock (systolic blood pressure ≤ 90 mm Hg).<sup>18</sup>



*Management of Suspected Raised ICP*

The motor component of the GCS score is the most informative measure of the score for long-term outcome.<sup>29,47,97</sup> Hypoxemia and hypotension can lower a patient's GCS score, and thus assessments made following resuscitation are more reliable.<sup>94</sup> If possible, a GCS score prior to administration of sedative or paralytic agents serves as a valuable baseline. Serial examinations are important. A drop of 2 or more points in the GCS score is considered significant and should raise an index of suspicion for an expanding intracranial mass lesion.<sup>135</sup> In a series of 81 patients with initial field GCS scores of 13 or 14 who subsequently deteriorated and required prehospital intubation, 31% had an abnormal CT scan and 21% had evidence of an intracranial hemorrhage.<sup>54</sup> The development of an oval or irregular pupil is the first sign of uncal herniation.<sup>98</sup> Signs of cerebral herniation also include asymmetric pupils with a difference > 1 mm in size, unilateral or bilateral dilated and fixed pupils, extensor (decerebrate) posturing, or a progressive loss of 2 points in GCS score starting from an initial GCS score ≤ 8.

In the setting of signs of cerebral herniation, urgent measures are needed to lower ICP. These measures should include acute hyperventilation and mannitol administration.<sup>154</sup> In addition, good outcomes for patients with traumatic intracerebral mass lesions correlate strongly with prompt surgical evacuation.<sup>72,121,133,144</sup> In the face of a declining GCS score, the prime goal should be definitive neurosurgical care. In these situations, timing becomes critical as outcome directly correlates with the duration of the mass effect.

**Mannitol Administration.** The mechanisms underlying the therapeutic benefits of mannitol are not thoroughly understood. By increasing the osmotic gradient between blood and the brain, water is drawn from normal and edematous brain into the vascular compartment, leading to prompt osmotic diuresis and a reduction in ICP.<sup>76,161</sup> The onset of action is within 15–30 minutes with a peak response at 1 hour, lasting for 6–8 hours.<sup>5</sup> Mannitol is more effective when given in a high dose (1.4 gm/kg) as a bolus rather than by continuous infusion.<sup>21,32,142</sup> Mannitol also reduces blood viscosity and improves the rheology of blood flow.<sup>24</sup> There is evidence to suggest that mannitol also acts through vasoconstriction in response to these changes in blood viscosity.<sup>106,108</sup>

**Hypertonic Saline.** Hypertonic saline has been shown to be as effective as mannitol in treating raised ICP in patients with head trauma.<sup>48,57,112</sup> Concentrations ranging from 7.5% (2 mg/Kg) to 23% (1 ampule, 30 ml) are effective.<sup>71,145,154,162</sup> Hypertonic saline acts through osmotic, vasodilatory, hemodynamic, antiinflammatory, and neurochemical mechanisms.<sup>48</sup> Through osmotic effects, hypertonic saline draws fluid into the intravascular compartment, reducing brain water and improving perfusion. In both human and animal studies, hypertonic saline has been shown to increase mean arterial pressure, likely as a result of plasma volume expansion.<sup>48</sup> Problems of volume depletion and hypotension are not as profound with hypertonic saline as they are with mannitol.<sup>71,109,114,153</sup>

**Hyperventilation Therapy.** Moderate hyperventilation therapy is indicated as a temporizing, life-saving intervention for the comatose patient with impending cerebral herniation. Carbon dioxide is a potent vasodilator of the cerebral microcirculation. For every 1 mm Hg drop in PCO<sub>2</sub> there is a concomitant 3% decrease in CBF.<sup>15</sup> Hyperventilation quickly decreases cerebral arteriolar diameter and can dangerously lower CBF.<sup>96,111</sup> Microdialysis and brain tissue O<sub>2</sub> measurements have demonstrated that even brief periods of hyperventilation can lead to hypoxia and clinically significant changes in metabolites, together, indicative of cerebral ischemia.<sup>93,96</sup> The potential for hyperventilation to compromise CBF is especially true in the early phases of severe brain injury when autoregulation is impaired and CBF is reduced from the injury itself.<sup>95,107,137</sup> Ventilatory interventions to hyperventilate the patient frequently also alter thoracic pressures and may have deleterious effects by decreasing venous return and raising ICP. For these reasons, hyperventilation should be reserved for situations of acutely increased ICP with impending cerebral herniation.<sup>22</sup>

Without clear evidence of cerebral herniation, pCO<sub>2</sub> goal values in the prehospital setting should be in the range of 35 and 40 mm Hg, typically achieved with tidal volumes of 10 ml/kg and ventilatory rates of 10 breaths/minute.<sup>74</sup> Herniating patients should be hyperventilated to PCO<sub>2</sub> levels not < 30 mm Hg, with a goal of 30–35 mm Hg. End-tidal PCO<sub>2</sub> may not be a reliable measure of PaCO<sub>2</sub>, particularly in patients with multiple traumas, chest injuries, hypovolemia, and hypotension secondary to massive blood loss.<sup>11,74,134</sup> In practice, studies continue to show a relative disparity between the guidelines and clinical practice.<sup>147,159</sup> Manual ventilation, with high ventilatory assist rates, appears to predispose the patient to low end-tidal CO<sub>2</sub> levels.<sup>147</sup>

*Temperature*

Hypothermia is a predictor of death in trauma patients.<sup>80,89</sup> A retrospective analysis of 38,520 trauma patients, aged ≥ 16 years with an admission body temperature ≤ 35°C, demonstrated an increased risk of death in all patients (OR 3.03, 95% confidence interval 2.6–3.5) as well as in a subgroup of patients with isolated, severe TBI (OR 2.21, 95% confidence interval 1.6–3.0).<sup>157</sup> In experimental models, induced hypothermia has several physiological actions beneficial to TBI. Hypothermia acts on the central nervous system to decrease metabolism, and for every 1°C drop in temperature there is a 6–7% decrease in the cerebral metabolic rate for O<sub>2</sub>.<sup>55,126</sup> The results of several good quality meta-analyses showed a benefit to induced therapeutic hypothermia for severe TBI, but failed to demonstrate statistical improvements in long-term clinical outcome.<sup>119</sup> During induced therapeutic hypothermia, shivering and catecholamine responses are controlled and the pathophysiological mechanisms may differ from those occurring in spontaneous exposure hypothermia, wherein stress responses proceed unabated. Further, the duration of induced hypothermia is usually 24–48 hours or longer as compared with the relatively short periods for field hypothermia. Rebound increases in ICP may also occur during rewarming.<sup>13</sup> However, early

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spontaneous hypothermia, such as that which occurs with field hypothermia, may be effective in minimizing brain injury if patients are not rewarmed. Young patients < 45 years of age who present with hypothermia (< 35°C) experienced a statistically higher incidence of better ICP control and a lower incidence of poor outcome (76%) if they were kept hypothermic (33°C) for 48 hours, as compared with those who were warmed on admission.<sup>30,90</sup>

### Organized Emergency Trauma Systems

#### *Dispatch and Triage Planning*

Optimal treatment of patients with TBI begins with the 911 call and efforts to acquire information regarding the possibility of TBI, to thereby dispatch the most appropriate team of providers to the scene. The level of skill of the response team and the time to reach the hospital strongly influence the efforts to stabilize the patient in the field. There appears to be a threshold set of skills necessary to prevent secondary injury, above which further abilities appear to be less important. Technical facility with airway management, securing intravenous access, and the ability to administer mannitol would appear advantageous to modern aggressive response efforts to prevent secondary injury from hypoxia, hypotension, and cerebral herniation in the prehospital setting. The response care team needs to integrate the mechanism of injury, the location of and duration to the hospital, the patient's neurological examination, the involvement of other life-threatening extracranial injuries, and the potential for further deterioration. A rapid decision is made, balancing further efforts in the field against direct transport of the patient. The degree to which the patient is stable and/or can be stabilized in the field guides whether the patient should be transported to a Level I or regional trauma center as compared with the closest medical center with emergency services.

In the field, an assessment of the severity of the head injury determines the need for field intervention and stabilization. The emergency response team needs to perform repeated neurological examinations. Patients with TBI require special attention to signs of cerebral herniation that indicate a mass lesion. A unilateral dilated pupil in a comatose patient is the hallmark sign, a warning signal of a possible expanding hematoma. This sign should prompt the highest priority for urgent transport to a center capable of providing definitive neurosurgical care. Secondary injury is more commonly associated with severe as compared with mild or moderate TBI. A degree of hypoxia or hypotension may be tolerated without sequelae in a patient with mild head injury. The same insult in a patient with severe TBI can result in irreversible neuronal injury and permanent deficit. Difficult extractions threaten survival and dramatically increase the risk of secondary injury following TBI. In a similar fashion, associated extracranial injuries may increase the risk of subsequent secondary events from profound or prolonged hypotension. In preparation for air flight, assessment of thoracic and cranial injuries is critical. Open depressed fractures, fractures into the air sinuses, and signs of basal

skull fracture may be indicators of underlying intracranial pneumocephalus and should increase awareness for possible complications from expansion of the air collection and/or development of tension pneumocephalus during transport at elevated altitudes. During triage in the field, the response team must identify the patients with a potential risk for deterioration and integrate this risk into field stabilization efforts, mode of transport, and transport destination decisions.

#### *Timing of Treatment*

The merits of prehospital stabilization versus the need for prompt transport to definitive care need to be tailored to each individual patient.<sup>87</sup> The "golden hour" principle guides prehospital response teams to get the patient to their destination center so that they are able to receive surgery within the 1st hour of the injury.<sup>85</sup> In the general trauma literature, studies comparing advanced life support versus basic life support have supported a "scoop and run" approach and this philosophy has been widely adopted.<sup>113</sup> However, whereas insults such as hypotension or hypoxia may be tolerated for a period of time in a trauma patient with a mild head injury, these same insults can be highly detrimental to a patient with a severely injured brain. For patients with severe TBI, better survival rates and long-term outcomes are achieved with on-scene stabilization in the field.<sup>44,63</sup>

Organized trauma systems improve patient outcomes.<sup>67,127,136,140</sup> Level of education and organization of trauma response teams may influence adverse events more so than geography and speed. Several studies have documented that critical care teams composed of physicians and trained nurses achieve better outcomes for patients with severe TBI.<sup>1,7,61,62,115</sup> Even though critical care response teams incur longer median prehospital times than typical response teams (113 vs 45 minutes, respectively;  $p < 0.001$ ), these increases were not an independent predictor of outcome.<sup>61</sup> Urgent transport following stabilization is, however, a priority for patients with mass lesions and impending cerebral herniation. For acute subdural hematomas, the mortality rate increases to 90% if surgical evacuation is delayed 4 hours or more after injury, as compared with a mortality rate of 30% if evacuation occurs sooner.<sup>133</sup> There is evidence from early studies to suggest that within a 4-hour time window, duration of transport has a relatively small influence on outcome.<sup>46,88</sup> Together these data suggest that on-scene stabilization and the quality of care in the field is as important as speed in improving outcomes following severe TBI.<sup>7,44</sup>

#### *Mode of Transport*

Most dispatch decisions focus on estimated time or distance for ground transport to reach the target receiving hospital, and a threshold leads to the decision to request aeromedical transport. Odds of survival (1.6–2.25) are better following helicopter as compared with ground transport and it is the patients with severe TBI who benefit the most from air transport.<sup>40</sup> Reasons for this benefit may include the presence of ancillary nurse or physician members on the response team, careful attention to postin-

tubation ventilatory parameters with close monitoring of end-tidal CO<sub>2</sub>, and a transport destination to a definitive Level I or regionalized trauma center.<sup>35,122</sup> Furthermore, at the receiving hospital 46% of patients brought in by the aeromedical team had CT performed immediately upon arrival, as compared with only 3% of those transported by land emergency medical services.

There is an expected but unpredictable drop in barometric pressure with rapid altitude ascent. An average drop in O<sub>2</sub> saturation of 4% has been found in healthy passengers seated in air cabins at altitudes of 8000 feet.<sup>105</sup> Stable oxygenation can be achieved during air flight transport and mechanical ventilation. In studies from Iraq, 22 patients requiring mechanical ventilation were continuously monitored during transport from Balad to Germany, with an average flight time of 6–8 hours at altitudes of 37,000 feet.<sup>4</sup> There were 3 desaturation events (saturation of peripheral O<sub>2</sub> < 90%), the longest lasting 280 seconds, and each resolved spontaneously without intervention.

#### *Transport Destination*

The choice of final destination for transport of patients with head injuries is an important component in prehospital management of TBI. Rapid transport to a center with round-the-clock neurosurgical expertise is probably the single most important determinant of outcome in patients with mass lesions.<sup>72,133</sup> Lower mortality rates for patients with TBI occur in those transferred to a Level I trauma facility as compared with those treated in a Level II center ( $p = 0.017$ ).<sup>100</sup> Significant delay and worse outcome generally occurs if a patient with head injury is transported first to a local hospital for hemodynamic stabilization and then requires transfer to another hospital for neurosurgical management of their intracranial injury.<sup>45,70,121,128,130,163</sup> Even after stabilization at the original hospital, Dunn et al.<sup>52</sup> found a 6% incidence of hypoxia (saturation < 95%) and a 16% incidence of hypotension (systolic blood pressure < 90 mm Hg) upon arrival at the transfer receiving hospital. Treatment volume of the center also influences outcomes for patients with severe TBI. A higher risk of death occurs in low-volume trauma centers,<sup>141</sup> and advanced trauma centers report a one-third reduction of deaths due to head injuries, as compared with hospitals without a dedicated trauma center.<sup>68</sup>

All regions should have an organized trauma system and protocols to guide field stabilization, timing of transport, and choice of destination. In general, patients with a GCS score of 14 or 15 should be transported to the nearest emergency department. Patients with GCS scores of 9–13 have a potential need for neurosurgical intervention and should be transported to a trauma center. Patients with severe head injuries (GCS Scores from 3 to 8) who can be stabilized at the scene or en route should be transported to a center with neurosurgical expertise and specialized TBI capabilities. Proponents of regionalized trauma centers exalt the benefits of high quality, standardized care for all patients with TBI in these centers.<sup>69,75,91</sup> Hannan et al.<sup>69</sup> reported an OR of 0.53–0.85 for death in patients with TBI treated at a regionalized trauma center as compared with care at other hospitals.

Special transport considerations pertain to the pedi-

atric population. In general, pediatric patients with severe TBI are best treated in a pediatric trauma center or in an adult trauma center with qualifications for managing children. Direct transport, rather than transport to a temporizing facility and delayed transfer to a definitive pediatric trauma center, also leads to improved survival and better outcomes in pediatric patients with severe TBI.<sup>79</sup> Pediatric patients with severe TBI have poorer survival rates if they are treated at Level II adult trauma centers.<sup>123</sup>

#### *Disaster and Mass Casualty Events*

Recent world events, political and natural, have heightened the awareness and need for prehospital care of the highest standards for mass casualty events. The levels of trauma care in the Iraq war are a model for mass casualty and disaster trauma programs.<sup>3</sup> In Iraq there are 5 levels of care—3 in the combat zone, 2 outside the combat zone. In the combat zone, levels of care progress from first aid in battle (Level I), to mobile resuscitation surgical teams supporting combatant units in the field (Level II), to hospitals that provide advanced care similar to the level of a civilian trauma center (Level III). Definitive neurosurgical care and CT scanners are available at Level III centers, which are typically 15–45 minutes away by air transport. Level IV care is provided outside the combat zone and Level V care is provided at a center in the US.

High-energy mechanisms are involved in many disaster and mass casualty situations. Blast injuries have been the signature injury of the Iraq war.<sup>60</sup> Hypotension and profound near-exsanguination levels of hemorrhage are common. Operation Iraqi Freedom and evaluation of casualties from previous wars have shown that exsanguinations from extremity trauma can be prevented with prehospital tourniquets with little risk to limb survival if tourniquet times are < 6 hours.<sup>10</sup> With the potential for serious lung injury, massive transfusions, and higher incidences of adult respiratory distress syndrome, the availability of adequate O<sub>2</sub> supplies may be a limiting resource and mass casualty planning needs to prepare for this eventuality.<sup>4</sup> Critical care air transport teams need to be able to mobilize quickly. Drugs typically used in the prehospital setting need special storage as they may be detrimentally affected by the extremes in temperature that frequently prevail in these situations.<sup>73</sup> In addition, increased awareness of the importance of preventative injury for the emergency response team needs to be a priority. Of 282 patients hospitalized following the 911 World Trade Center terrorist attack, 35 cases of TBI were identified, one-third of these occurring in the rescue workers.<sup>129</sup>

Secondary injury and insults are often underappreciated in crises, and this risk is even more likely during mass casualty care. In a mass casualty crisis, nonneurosurgeon caregivers may be faced with management of severe TBI including penetrating injuries to the cranium. Inexperienced response persons, while trying their best, may inadvertently cause injuries that lower the normally accepted standards of care. The sensitivity of the injured neuron to secondary insults makes patients with TBI more vulnerable in the setting of mass casualty. Closed-loop control systems that use feedback mechanisms to deter-



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mine the dynamic output and maintain parameters within preset goals are being introduced in many technological aspects of trauma and critical care.<sup>118</sup> These new strategies may help maintain the high standards of prehospital care needed for TBI casualties.

### Ethical Considerations

Studies have demonstrated that it is very difficult to accurately predict prognosis within the first 24 hours of TBI.<sup>81</sup> Aggressive resuscitation efforts in the acute phases of TBI decrease mortality rates. More controversial is the question of whether these resuscitation efforts improve the number of patients with good outcome or whether they increase the number of survivors with vegetative and disabled outcomes. Patients with very severe head injuries generally die regardless of the speed, intensity, and quality of prehospital care.<sup>84,113</sup> In a study of accidental fatalities, approximately one-half of all patients died within the first minutes after injury.<sup>149</sup> In one-third of casualties, death occurred in the first few hours following injury, and brain injury and hemorrhage were found to be the principal causes of death.<sup>149</sup> With the intervention of air medical and rapid-response teams, patients with minimal residual neurological functions (GCS scores of 4) have shown the greatest increase in survival.<sup>40</sup> In contrast, patients with a GCS score of 3 did not fare better with aeromedical response teams, suggesting that these patients have suffered irreversible damage from their injuries that is not salvageable, no matter how quickly emergency personnel arrive on the scene or how quickly the injured are transported to a hospital.<sup>7</sup> Longer transport times allow for severely injured patients with devastating primary injury to declare themselves as casualties. Unresponsiveness from the moment of impact, accompanying cardiac arrest in the field with hypoxia and hypotension, together with short transport times to the hospital are risk factors for persistent vegetative outcomes. It is important for emergency response teams to critically evaluate the best neurological examination of the patient in the field. This examination serves as a marker for the degree of primary injury and aids resuscitation efforts in these difficult situations. In the acute setting, the goals of the prehospital care team should be to do everything possible to stabilize the patient and prevent secondary injury. In this time frame, it is too early, even in the most dismal of situations, to prognosticate patient outcome. Assessments of medical futility and possible organ donation are best delayed until sufficient time has allowed the results of resuscitative efforts to be carefully evaluated.

### Conclusions

The goals of prehospital care for TBI are to stabilize patients for transport, to triage those with mass lesions and impending cerebral herniation, and to prevent secondary insults and injury. Organized trauma systems with defined protocols for field resuscitation, modes of transport, and trauma facility destination ensure reproducible high-quality prehospital care. Given the proximity to the time of impact, advances in prehospital practice

are a key target for further improvements in long-term functional outcomes following TBI.

### Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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