

## PREHOSPITAL CARE

### Prehospital Management of the Injured Brain

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Prehospital and emergency medical care of patients with traumatic brain injury should focus on minimizing the effects of secondary insults to the brain. In the field, the primary objectives are to prevent hypoxemia and hypotension and to monitor vital signs and neurologic status, including clinical assessments of intracranial pressure (ICP). Current training standards for field care providers in the United States follow guidelines developed by the Department of Transportation, National Highway Traffic Safety Administration, Paramedic National Standard Curriculum (Table 1).

There is little information in the literature that evaluates the abilities of emergency medical services (EMS) systems to meet the primary objectives of prevention of hypoxemia and hypotension. In addition, there are few published reports of prehospital interventions to reduce secondary insults to the brain from the time of injury to hospital admission. The purpose of this paper is 1) to review studies that examine the efficacy of prehospital EMS systems to prevent hypoxemia and hypotension in patients with traumatic brain injury; 2) to summarize the literature on published studies that identify interventions to minimize secondary brain injury during transportation from the field; and 3) to examine how early interventions change long-term outcome.

The interval from brain injury to arrival at a medical facility is an important time for the brain because many patients with severe head injury have hypoxemia, with hospital admission arterial oxygen tensions ( $\text{PaO}_2$ ) values  $<60$  mmHg or pulse oximeter  $\text{O}_2$  saturations ( $\text{SpO}_2$ ) of about 90%. This may be caused by associated traumatic injuries to the chest or hemorrhage from vascular injury or organ rupture. Clinical studies show that more than 20% of patients with severe head injury had  $\text{SpO}_2 <90\%$  on hospital admission, indicating a major reduction in oxygen transport.<sup>1</sup> In the Trauma Coma Data Bank, hypoxemia was identified in 46% of head-injured patients and was associated with an 85% increase in mortality.<sup>2</sup>

The Trauma Coma Data Bank prospective data collected on 717 patients demonstrate that a single observation of field systemic blood pressure  $<90$  mmHg or hypoxia (apnea/cyanosis or  $\text{SpO}_2 <90\%$  or  $\text{PaO}_2 <60$  mmHg by arterial blood gas) was the most powerful predictor of outcome independent of the other major predictors such as age, admission Glasgow

Coma Scale (GCS) score or intracranial diagnosis, and pupillary status.<sup>2</sup>

Among the 717 patients, one third had a combination of hypotension and hypoxia. A finding of the Trauma Coma Data Bank analysis that has since been confirmed by other studies is that a single episode of hypotension results in a doubling of mortality.<sup>3</sup> Although the question of reversal of field hypotension in brain-injured patients has not been addressed directly, a post-hoc analysis of a randomized placebo-controlled multicenter trial that had a subgroup of patients who were given either 7.5% saline or normal saline showed that the group given hypertonic saline had improved blood pressure responses, decreased overall fluid requirements, and improved survival.<sup>4</sup> However, the critical values for the duration and magnitude of hypotensive or hypoxemia episodes have not been determined.

We examined whether additional factors in prehospital management predicted long-term outcome, by comparing outcomes in patients with traumatic brain injury who underwent tracheal intubation in the field with outcomes of those who did not and outcomes of those who had positive blood alcohol concentrations compared with those who were sober.<sup>5</sup> Five of the 31 patients were intubated in the field and 12 had positive blood alcohol concentrations. The Injury Severity Score (ISS) of the field-intubated patients was significantly higher. The change in the GCS score from admission to discharge from the trauma center after an almost identical length of stay of 30 to 35 days was no different, despite the fact that the admitting GCS score of intubated patients averaged only 3.6. The change in Rancho Los Amigos score (RAS) was also not different. Admission  $\text{PaO}_2$  in patients requiring tracheal intubation was 99 to 205 mmHg, which appeared adequate even though four fifths of these patients had chest injuries. Systolic blood pressure on admission was above 100 mmHg in all groups of patients. Our conclusion was that, despite significantly greater ISS and lower admission GCS and  $\text{PaO}_2$ , brain-injured patients who were intubated in the field had no difference in GCS and RAS on discharge after the same length of stay, suggesting that optimal care to minimize secondary brain damage was provided in the prehospital phase during field management in Maryland.<sup>5</sup>

Oxygenation by face mask, Combitube® placement, and tracheal intubation have been advocated to optimize oxygen saturation in the field. Training of field care providers in airway management techniques that minimize neck movement is required because cervical spine injury is four times more likely in association with head injury than in the overall blunt trauma patient population.<sup>6</sup> The incidence of cervical spine injury in association with blunt trauma is about 2.6% versus a reported incidence as high as 10% in head-injury patients with GCS scores of 8 or less.<sup>7</sup> Neck stabilization should be used during transportation and airway management in all head-injured patients.

The Maryland Institute for Emergency Medical Services Systems (MIEMSS) is the body charged with developing prehospital management protocols and providing training and continuing education to paramedics in Maryland. The training

Based on a presentation at TraumaCare 2001, the 14th Annual Trauma Anesthesia and Critical Care Symposium, San Diego, California, May 17-19, 2001.

**Table 1.** Training for Field Management of Head Injury

<p><b>Assessment in the Field</b></p> <ul style="list-style-type: none"> <li>a. Altered level of consciousness, amnesia of event, confusion, disorientation, lethargy or combativeness, focal deficit or weakness: due to effect on cerebral cortices and/or reticular activating system</li> <li>b. Vomiting - hypothalamic effect</li> <li>c. Brain stem effects causing <ul style="list-style-type: none"> <li>i. elevated blood pressure to maintain cerebral perfusion</li> <li>ii. bradycardia due to vagal effect</li> <li>iii. irregular respiration or tachypnea</li> <li>iv. decerebrate/decorticate posturing</li> </ul> </li> <li>d. Seizures</li> </ul>	<p><b>Field Management of Head-Injured Patients</b></p> <p><b>Airway/Ventilation</b></p> <ul style="list-style-type: none"> <li>a. Suspect cervical spine injury</li> <li>b. Oxgenate to 95%–100% O<sub>2</sub> saturation</li> <li>c. Hyperventilate with signs and symptoms of elevated intracranial pressure (see above)</li> <li>d. Do not exceed respiratory rate 30/min assisted ventilation</li> <li>e. Avoid nasal intubation</li> </ul>
<p><b>Clinical Evaluation of Levels of Increasing Intracranial Pressure</b></p> <ul style="list-style-type: none"> <li>a. Involvement cerebral cortex and upper brain stem <ul style="list-style-type: none"> <li>i. Blood pressure starts to rise and heart rate begins to slow</li> <li>ii. Pupils still reactive</li> <li>iii. Cheyne-Stokes respiration</li> <li>iv. Patient still localizes to painful stimuli</li> <li>v. Withdraws with flexion</li> </ul> </li> <li>b. Mild brain stem involvement <ul style="list-style-type: none"> <li>i. Wide pulse pressure and bradycardia</li> <li>ii. Pupils non-reactive or sluggish</li> <li>iii. Central neurogenic hyperventilation</li> <li>iv. Extension</li> </ul> </li> <li>c. Lower brain stem, medullary involvement <ul style="list-style-type: none"> <li>i. Dilated pupil on same side</li> <li>ii. Respiration erratic or apnea</li> <li>iii. Facial paralysis</li> <li>iv. Irregular pulse with rate changes</li> <li>v. QRS, S-T, and T wave changes on ECG</li> <li>vi. Low and labile blood pressure</li> </ul> </li> </ul>	<p><b>Circulation</b></p> <ul style="list-style-type: none"> <li>a. Start infusion isotonic fluid (normal saline or Ringer's lactate) and titrate to maintain blood pressure and prevent hypotension.</li> <li>b. If hypotension present, look for evidence of internal bleeding.</li> <li>c. Stop external bleeding.</li> </ul>
<p><b>Field Data Collection</b></p> <ul style="list-style-type: none"> <li>a. Determine Glasgow Coma Scale score to assess level of consciousness</li> <li>b. Vital signs</li> <li>c. Pupil size and reaction</li> <li>d. Presence of focal deficits</li> <li>e. History of unconsciousness or amnesia of event</li> </ul>	<p><b>Disability</b> Repeat assessments of intracranial pressure, Glasgow Coma Scale score, focal deficits, and vital signs.</p> <p><b>Pharmacology</b></p> <ul style="list-style-type: none"> <li>a. Diuretics: mannitol or furosemide</li> <li>b. Paralysis/sedation after securing the airway</li> <li>c. Avoid glucose unless hypoglycemia is confirmed</li> </ul> <p><b>Positioning</b></p> <ul style="list-style-type: none"> <li>a. Place in cervical collar on backboard</li> <li>b. Elevate head end of back board 30°</li> <li>c. Minimize stimulation</li> </ul>
<p>Modified from the Paramedic National Standard Curriculum (US Department of Transportation: National Highway Traffic Safety Administration)</p>	<p><b>Transport</b> Moderate to severe head injury (Glasgow Coma Scale score &lt; 12)</p> <ul style="list-style-type: none"> <li>a. Transport to trauma center</li> <li>b. Helicopter versus ground</li> <li>c. Use lights/siren</li> </ul>

of MIEMSS field care providers in management of patients with head injury is summarized in Table 1. The general patient care management protocol developed by MIEMSS for field management includes initial assessment, guidelines for airway, breathing, oxygenation, and ventilation of the head-injured patient. A rapid-sequence tracheal intubation protocol (including Combitube® and cricothyroidotomy) and the field management of seizures and management protocols for altered mental status are advocated by the MIEMSS.

### Field Interventions to Minimize Secondary Brain Injury

Potential interventions in the field include an immediate reduction in intracranial pressure (ICP) by administration of mannitol. This is thought to occur because of reduced viscosity, resulting in increased cerebral blood flow and oxygen delivery.<sup>8</sup> These rheologic effects probably explain why mannitol reduces ICP within a few minutes of a bolus, with an effect that lasts 90 minutes to 6 hours or more after administration, yet its diuretic effect takes 15 to 30 minutes to establish osmotic gradients between plasma and cells. Mannitol has been used instead of 7.5% hypertonic saline as a small-volume resuscitation fluid. Mannitol was superior to thiopental (Pentothal) in improving cerebral perfusion pressure, intracranial pressure, and outcome in a randomized trial by the Toronto Group.<sup>9</sup>

Brain swelling and elevated ICP develop in 40% of patients with severe TBI.<sup>10</sup> Since hyperventilation can cause a rapid reduction in ICP, most clinicians had assumed that hyperventilation benefits the patient. However, hyperventilation reduces ICP by causing cerebral vasoconstriction and subsequent reduction in CBF. There is a risk of causing cerebral ischemia with aggressive hyperventilation, especially considering that in the first day after injury, cerebral blood flow is less than half that of normal individuals. In a study in which patients with head injury and no elevated ICP were treated with hyperventilation, at autopsy there was histologic evidence of cerebral ischemia, possibly caused by the hyperventilation. In a prospective, randomized study, 77 patients with severe TBI were randomized to a group with PaCO<sub>2</sub> 25 ± 2 mmHg compared with 35 ± 2 mmHg for 5 days. At 3 and 6 months after injury, the patients with an initial GCS score of 4 or 5 who were not hyperventilated had a significantly better outcome.<sup>11</sup> This suggests hyperventilation should not be used in the field unless there are obvious signs of elevated ICP (Table 1).

Small-volume resuscitation for traumatic injury has been studied extensively in Europe. A meta-analysis of controlled clinical studies in trauma patients shows that among six reported studies of 604 patients, hypertonic saline dextran (HSD; 7.5% saline in 6% dextran) significantly increased systolic blood pressure (n = 301), compared with standard of care interventions (SOC) (n = 303).<sup>12</sup> Trauma and Injury Severity Scores (TRISS) were the same for both groups (mean ± SD): 0.82 ± 0.32 and 0.81 ± 0.33 for HSD versus SOC, respectively. The significant increase in blood pressure in the HSD versus SOC group of patients persisted throughout admission to the hospital and emergency department care. Survival was improved with HSD (odds ratio, 1.46; 95% confidence interval, 0.93–2.23). Discharge survival was also significantly improved with HSD (odds ratio, 1.5; 95% confidence interval, 1.01–2.23).

Overall, there was a 27% reduction in mortality in patients treated with HSD. Small volume (250 ml HSD) improved

survival in patients with penetrating injuries who subsequently required surgery (odds ratio, 1.97; 95% confidence interval, 1.07–3.16).<sup>13</sup> When hypertonic saline/hydroxethyl starch solution (HSS) was used to treat raised ICP after stroke, it reduced ICP faster and more effectively than mannitol. HSS patients received 100 ml, whereas 200 ml of 20% mannitol was infused in both instances over 15 minutes. HSS patients reached their lowest ICP within 25 minutes and mannitol patients within 40 minutes.<sup>14</sup>

Barbiturates have been used as a means of reducing cerebral metabolic rate for oxygen (CMRO<sub>2</sub>) so that cerebral blood flow decreases and ICP falls.<sup>15</sup> Barbiturates, either pentobarbital or pentothal, are free radical scavengers, so they may be useful in cerebral ischemia. There are practical issues in the use of barbiturates, in that to reduce CMRO<sub>2</sub>, doses are required that reduce blood pressure, so inotropic and respiratory support may be necessary. This makes the use of barbiturates to control ICP and CMRO<sub>2</sub> impractical in the field. It is still uncertain whether barbiturate coma can result in ICP control and whether absolute control of intracranial pressure improves outcome. Other neuroprotective drugs with fewer side effects than barbiturates may be given with advantage in the field.

Hypothermia is beneficial in reducing ICP in patients with severe head injury.<sup>16,17</sup> However, in a recent multicenter prospective randomized clinical trial, reducing temperature to 33°C within 8 hours of injury did not improve outcome status 6 months after injury.<sup>17</sup> Nonetheless, among patients 45 years of age or younger who had head injury and hypothermia on admission to a hospital, there was a significantly lower incidence of intracranial pressure of more than 30 mmHg in patients assigned to the hypothermia group. Field hypothermia was more frequent in brain-injured patients with higher ISS, prehospital hypotension, positive blood alcohol admission in winter, and greater prehospital fluid volume.<sup>17</sup> The implications for field management are that older patients (>45 years) do not benefit from hypothermia and actually do worse than normothermic patients. If young brain-injured patients are hypothermic on admission to a hospital, as were 102/392 patients in this study who had an average bladder temperature of 33°C, it is not advisable to actively warm them. These data suggest that early cooling may be effective in minimizing brain injury, but maintaining hypothermia for 48 hours was not effective in improving clinical outcome.

Safar's group in Pittsburgh used early cooling to produce cerebral hypothermia by means of rapid aortic cannulation. Using the percutaneous approach with a Sellinger technique, a large-bore aortic cannula was placed and cold flush administered to produce rapid cerebral cooling. They achieved normal cerebral outcomes after up to 45 minutes of no cerebral flow.<sup>18</sup> In a model of a 20-minute exsanguination cardiac arrest in dogs, the addition of Tempol to the 24°C aortic flush improved outcome but did not avoid histologic damage to neurons.<sup>19</sup> It is clear that suspended animation by rapid brain cooling improved outcome in these animal experiments. It also suggests that such techniques may be a future intervention for brain-injured humans.


Interventions are now possible in the prehospital phase using enhanced telecommunication through a Web-based system that enables four multi-plexed phones to send images from the field.<sup>20</sup> Using this system, a neurologic exam can be conducted in about 8 minutes with the help of the paramedic.

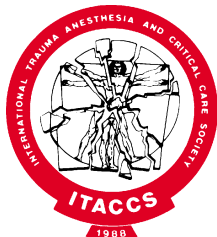
Transport time can now be used as diagnostic time. The first patient was treated after diagnosis of stroke made via telecommunication links in 2000.<sup>21</sup> These communications may be used for giving neuroprotective agents, which may have great benefit for patients with head injury when given before hospital arrival, nearer the time of injury.

### Summary

The most important management goals in the field are as follows: avoid hypoxemia; avoid hypotension; give hypertonic saline; administer neuroprotective agents when advanced field telecommunications are available; and consider hypothermia, which seems to be promising initially but may not be so helpful later. Lastly, a futuristic approach is suspended animation—protecting the brain by cold aortic flush and neuroprotective agents to prevent further secondary damage.

### References

1. Fearnside MR, Cook RJ, McDougall P, et al. The Westmead Head Injury Project outcome in severe head injury: a comparative analysis of prehospital, clinical, and CT variables. *Br J Neurosurg* 1993; 7:267–79.
2. Chestnut RM, Marshall LF, Klauber MR, et al. The role of secondary brain injury in determining outcome from severe head injury. *J Trauma* 1993; 34:216–22.
3. Miller JD, Becker DP. Secondary insults to the injured brain. *J R Coll Surg (Edinb)* 1982; 27:292–8.
4. Vassar MJ, Fischer RP, O'Brien PE, et al. A multicenter trial for resuscitation of injured patients with 7.5% sodium chloride: the effect of added dextran. The Multicenter Group for the Study of Hypertonic Saline in Trauma Patients. *Arch Surg* 1993; 128:1003–11.
5. Mackenzie CF, Ho SJ, Jaweed N, et al. Influence of early factors on long-term head injury outcome. *Anesthesiology* 2000; 93:A366.
6. Kirschenbaum KJ, Nadimpalli SR, Fanhy R, et al. Unsuspected upper cervical spine fractures associated with significant head trauma: role of CT. *J Emerg Med* 1990; 8:183–98.
7. Hills DW, Deane SA. Head injury and facial injury: is there an increase risk of cervical spine injury? *J Trauma* 1993; 34:549–93.
8. McGraw CP, Howard G. The effect of mannitol on increased intracranial pressure. *Neurosurgery* 1983; 13:269–71.
9. Schwartz M, Tator C, Towed D, et al. The University of Toronto Head Injury Treatment Study: a prospective, randomized comparison of pentobarbital and mannitol. *Can J Neuro Sci* 1984; 11:434–40.
10. Brain Trauma Foundation. [www.Braintrauma.org/index.nsf](http://www.Braintrauma.org/index.nsf)
11. Muizelaar JP, Marmarow A, Ward JD, et al. Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. *J Neurosurg* 1991; 75:731–9.
12. Wade CE. Small volume resuscitation: a meta analysis of controlled clinical studies [abstract]. *TraumaCare* 2001; 11:23.
13. Wade CE, Kramer GC, Grady JJ, et al. Efficacy of hypertonic 7.5% saline and 6% dextran-70 in treating trauma: a meta analysis of controlled clinical studies. *Surgery* 1997; 122:609–16.
14. Schwartz S, Schwab S, Bertram M, et al. Effects of hypertonic saline hydroxyethyl starch solution and mannitol in patients with increased intracranial pressure after stroke. *Stroke* 1998; 29:1550–5.
15. Eisenberg HM, Frankowski RF, Contant CF, et al. High dose barbiturates control elevated intracranial pressure in patients with severe head injury. *J Neurosurg* 1988; 69:15–23.
16. Shiozaki T, Sugimoto H, Taneda H, et al. Selection of severely head injury patients for mild hypothermia therapy. *J Neurosurg* 1998; 89:206–11.
17. Clifton GL, Miller ER, Choi SC, et al. Lack of effect of induction of hypothermia after acute brain injury. *N Engl J Med* 2001; 344:556–63.
18. Behringer W, Prueckner S, Radotsky A, et al. Suspended animation (SA). I: Hypothermic aortic flush during prolonged exsanguination cardiac arrest (EXCA) in dogs. *Anesthesiology* 2000; 93:A473.
19. Behringer W, Wu X, Radorsky A, et al. Tempol by aortic arch flush (AAF) for cerebral preservation during prolonged exsanguination cardiac arrest (CA) in dogs: exploratory experiments. *Anesthesiology* 2000; 93:A475.
20. Xiao Y, Gagliano D, LaMonte M, et al. Design and evaluation of real time mobile telemedicine system for ambulance transport. *J High Speed Networks* 2000; 9:47–56.
21. LaMonte MP, Xiao Y, Mackenzie CF, et al. Tele-BAT: Mobile telemedicine for the Brain Attack Team. *J Stroke Cerebrovasc Dis* 2000; 9:128–35. 



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