GUIDELINES FOR PREHOSPITAL MANAGEMENT OF TRAUMATIC BRAIN INJURY

Brain Trauma Foundation, New York

U.S. Department
of Transportation

**National Highway
Traffic Safety
Administration**

Supported by a grant from the United States Department of Transportation, National Highway Traffic Saftey Administration.

CONTENTS

GUIDELINES FOR PREHOSPITAL MANAGEMENT OF TRAUMATIC BRAIN INJURY

Authors

Edward J. Gabriel, MPA, AEMT/P
Chief, Division Commander
Bureau of Operations—EMS Command
Fire Department, The City of New York

Jamshid Ghajar, MD, PhD, FACS
President, Brain Trauma Foundation
Clinical Associate Professor of Neurosurgery
Weill Medical College of Cornell University

Andrew Jagoda, MD, FACEP
Associate Professor of Emergency Medicine
Mount Sinai School of Medicine
(Represented American College of Emergency Physicians on Review Team)

Peter T. Pons, MD, FACEP
Senior Physician
Department of Emergency Medicine
Denver Health Medical Center

Thomas Scalea, MD, FACS
Physician in Chief, Shock Trauma Center
University of Maryland Medical Center

Beverly C. Walters, MD, MSc, FRCSC, FACS
Chief of Neurosurgery, The Miriam Hospital, Associate Professor
Clinical Neurosciences (Neurosurgery) Brown University School of Medicine

Participants

James R. Bedell, MD	American Academy of Pediatrics
John Bruns, Jr., MD	Mount Sinai Department of Emergency Medicine
Randall M. Chesnut, MD, FCCM	Oregon Health Services University
Captain Garry B. Criddle, RN, CDR	U.S. Department of Transportation—NHTSA
Lorraine M. Giordano, MD, FACEP	Fire Department, The City of New York
Peter C. Quinn	Brain Trauma Foundation
David Thurman, MD	Centers for Disease Control
Pamela D. Walker	Brain Trauma Foundation

Review Team

Robert Bass, MD	National Association of State EMS Directors
Dianne Danis, RN, MS, CEN	Emergency Nurses Association
Laura Lee Demmons	Association of Air Medical Services
Peter W. Glaeser, MD	American Academy of Pediatrics
Bryna Helfer, MA, CTRS	Traumatic Brain Injury Technical Assistant Center
Mary Irving, RCP	National Association of Emergency Medical Technicians
Carden Johnson, MD, FAAP, FACEP, FRCP	American Academy of Pediatrics
Jon Krohmer, MD, FACEP	National Association of EMS Physicians
Bill Myers, Chief	International Association of Fire Chiefs
Karen Oldham, MD	American Ambulance Association
Maryellen Smith	National Association of EMS Educators
Ken Threet	National Council of State EMS Training Coordinators
Jack Wilberger, MD	American College of Surgeons, Committee on Trauma
Christopher Wuerker, MD, FACEP	Association of Air Medical Services

INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of death and disability in children and adults in their most productive years. An estimated 1.6 million head injuries occur every year in the United States. Approximately 800,000 of the injured receive emergency department or other outpatient care, and approximately 270,000 are admitted to the hospital.[1, 2] Every year, approximately 52,000 deaths occur from TBI, and an estimated 70,000 to 90,000 people are left with permanent neurological disabilities.[3, 4] TBI has a devastating effect on the lives of the injured individuals and their families because disability results in a significant loss of productivity and income potential.[5] The cost to society is more than $30 billion annually.[6] Thus, neurotrauma is a serious public health problem that mandates continuing efforts in the areas of prevention and treatment.[6]

During the past two decades, understanding of the pathophysiology of TBI has increased remarkably. One central concept is now known: All neurological damage does not occur at the moment of impact (primary injury), but rather evolves over the ensuing minutes, hours, and days. This secondary brain injury can result in increased mortality and more disabling injuries. The *Guidelines for the Management of Severe Head Injury* was developed in 1995 using a scientific, evidence-based methodology, aimed at improving in-hospital care and outcome for the patient with this potentially treatable injury.[5] However, no group has systematically compiled evidence to support guidelines for the early assessment, treatment, and transport to appropriate facilities for severe head injury patients in a prehospital setting. It is hoped that the application of these new guidelines will improve future patient outcome by bringing more accurate scientific assessment and treatment to the care of patients.

Emergency Medical Services (EMS) providers are often the first health care providers for patients with TBI. TBI treatment often begins in the field by EMS providers who have varied skills, backgrounds, and qualifications. They continue this care en route to the hospital. Thus, prehospital assessment and treatment is the first critical link in providing appropriate care for individuals with severe brain injury.[7] Over the past thirty years, EMS providers have developed sophisticated systems for delivering emergency medical care to patients. The initial impetus for this development was the need to deliver such life-saving interventions as cardiac defibrillation.[8] Treating trauma patients in the field, especially the head-injured patient, has lagged behind prehospital advancements in medical and general trauma management. Only in the past five to ten years has any attempt been made to evaluate rigorously the care provided to trauma victims in the field.

The National Highway Traffic Safety Administration (NHTSA) estimates that the United States has 70,000 paramedics and 500,000 other EMS providers. More than forty levels of Emergency Medical Technician (EMT) certifications exist in the United States.[9] The NHTSA and the *National EMS Education and Practice Blueprint* have established standard knowledge and practice expectations for four categories that encompass the forty levels of EMS providers: First Responder, EMT-Basic, EMT-Intermediate, and EMT-Paramedic. Physicians, nurses, nurse practitioners, physician assistants, respiratory therapists, and others continue to be actively involved in EMS systems providing emergency care, planning, research, administrative review, and education. In addition, the Emergency Nurses Association has developed a prehospital nursing curriculum.

The EMS system is designed to provide medical intervention at first contact with the injured person. First contact occurs when someone recognizes a need for medical assistance and calls the local emergency number to access help. The caller will usually be routed to a call-taker, who obtains the information needed to initiate an appropriate EMS response to the scene. In many systems, the call-taker also assesses the medical situation before EMS personnel arrive, using established Emergency Medical Dispatch (EMD) procedures. The call-taker may provide prearrival instructions as simple as putting pressure on a bleeding site or as complex as opening an airway or performing CPR.[10] To determine the appropriate level of EMS response for the situation, the call-taker carefully questions the caller to determine the severity of the injury. The caller's responses may reveal that the injured person is unresponsive because of severe head injury, prompting the call-taker to send the highest level EMS responder available.

Once the EMS providers arrive on the scene, they assess and intervene. Emergency medical care in the field is provided by a wide variety of personnel.[11] The First Responder and EMT-Basic provide patient assessment and noninvasive intervention. However, a growing trend to offer additional training to the EMT-Basic now permits some of them to perform some invasive procedures, such as intravenous (I.V.) line placement and advanced airway skills. EMT-Intermediate training includes invasive interventions, such as I.V. line placement, endotracheal intubation, and the administration of a limited list of resuscitation drugs. The EMT-Paramedic, who has the highest level of EMT training, is allowed to perform advanced patient assessment as well as endotracheal intubation, EKG recognition, I.V. line placement, needle thoracostomy, and the administration of a comprehensive list of medications. In many countries, physicians in ambulances or helicopters respond to the call and care for the patient in the prehospital setting.[8]

The care of the head-injured patient should be provided as rapidly as possible to establish an airway and administer oxygen. Any level of responder can perform these procedures. Advanced interventions, such as endotracheal intubation, may be required, necessitating a higher level of responder.

Equally important to the assessment and care provided to the patient is the selection of the hospital destination. Whenever possible, the choice of a hospital should be predicated on selecting the most appropriate place for the patient to receive the care needed. In the case of the critically injured trauma victim, the highest-level trauma center available should be selected, consistent with local policy. In the case of the patient with severe TBI, a facility, usually a trauma center, with immediate diagnosing and intervention capabilities is the preferred direct transport destination. This facility must have appropriate medical personnel, a CT scanner, an operating room, intracranial pressure monitoring, and an intensive care unit.[5]

The 1995 *Guidelines for the Management of Severe Head Injury* is composed of fourteen chapters of evidence-based recommendations for the patient with head injury. Three address

prehospital issues and the remaining eleven discuss the hospitalized patient. To expand the chapters on prehospital care for these guidelines, an EMS task force was formed. Its assignment was to develop recommendations for managing prehospital patients with severe head injuries. Following the scientific methodology of the 1995 guidelines, the EMS task force avoided the subjectivity associated with managing these cases.[5]

The task force developing the prehospital guidelines used a meticulous methodology of scientific evidence rather than expert opinion. In addition, the task force actively involved representatives of national and international medical societies, EMS nonphysician practitioners and experts, and individuals with expertise, interest, and experience in the prehospital care of patients with severe head injury and in evidence-based methodology.

These guidelines focus primarily on the prehospital management of severe TBI in adult and pediatric patients with a Glasgow Coma Scale (GCS) score of 3 to 8. They also address the standard prehospital approach to patients, including the basic Airway, Breathing, Circulation (ABCs) of prehospital assessment.[5, 7] Believing that these guidelines should be "realistic" and "user-friendly" for all EMS providers was key to their development. Understanding EMS issues, such as patient access and dispatch criteria, was also a significant factor in developing these guidelines.

EMS providers must be familiar with the complex presentation of severe TBI patients. Early recognition of the initial signs and symptoms of TBI has a significant impact on the outcome of these patients. In addition to initial treatment, EMS providers must know about interventions aimed at minimizing secondary injury. Specifically, EMS providers must aggressively assess and treat hypoxemia and hypotension.[7, 12, 13] All recommendations in these guidelines are supported by the best available scientific evidence. The EMS task force recognized the unique and often difficult environments and circumstances that EMS professionals encounter. The skills and approaches they use must be realistic and flexible enough to adapt to those environments and circumstances. The guidelines' authors, with the assistance of EMS experts, have made every attempt to address these concerns.

Finally, these guidelines are intended to clearly delineate the current scientific basis for EMS prehospital practice in managing the patient with severe TBI.[14] As with most clinical practice parameters, scientific evidence is often insufficient to support standards of care. Upgrading these EMS clinical practice parameters from option to guideline and then to a standard of care requires focused, well-designed, and carefully implemented clinical research in the prehospital setting.[14]

In these guidelines, as in most guidelines, scientific evidence is insufficient to support a standard of care. A standard of care is a term meaning the highest-grade recommendation that a guideline can make and it must be supported by focused, well-designed, and carefully implemented clinical research. This kind of research will be needed in the future to upgrade and strengthen recommendations about prehospital care of the patient with TBI.

References

1. Sosin DM, Sniezek JE, Thurman DJ: Incidence of mild and moderate brain injury in the United States, 1991. Brain Inj 10(1):47-54, 1996.
2. Centers for Disease Control and Prevention: Traumatic brain injury—Colorado, Missouri, Oklahoma, and Utah, 1990-1993. MMWR 46(1):8-11, 1997.
3. Sosin DM, Sniezek JE, Waxweiler RJ: Trends in death associated with traumatic brain injury, 1979 through 1992: success and failure. JAMA 273:1778-1780, 1995.
4. U.S. Department of Health and Human Services: Interagency Head Injury Task Force Report. Washington, DC: U.S. Department of Health and Human Services, 1989.
5. Guidelines for the Management of Severe Head Injury. New York: Aitken Brain Trauma Foundation, 1995.
6. National Institutes of Health: A report of the task force on trauma research, 1994. Bethesda Maryland: 67.
7. Baxt WG, Moody P: The impact of advanced prehospital care on the mortality of severely brain-injured patients. J Trauma 27:365-369, 1987.
8. Pantridge JF, Geddes JS: A mobile intensive care unit in the management of myocardial infarction. Lancet 2:271-273, 1967.
9. U.S. Department of Transportation—National Highway Traffic and Safety Administration (NHTSA), 1998.
10. Clawson J: Telephone treatment protocols. J Emerg Med Serv 11:43-47, 1986.
11. The Journal of Emergency Care, Rescue and Transportation: State and Province Survey 1997. Volume 26, Number 12, December 1997.
12. Eichorn J: Prevention of intraoperative anesthesia and related severe injury through safety monitoring. Anesthesiology 70:573-577, 1989.
13. Klauber, MR et al.: The epidemiology of head injury: a prospective study of an entire community—San Diego County, California, 1978. Am J Epidemiol 113(5):500-509, 1981.
14. American Medical Association: Office of Quality Insurance & Health Care Organizations' Attributes to Guideline Development of Practice Parameters. Chicago: AMA, 1990.

Disclaimer of Liability

The information contained in the *Guidelines for Prehospital Management of Traumatic Brain Injury*, which reflects the current state of knowledge at the time of completion (February 2000), is intended to provide accurate and authoritative information about the subject matter covered. Because there will be future developments in scientific information and technology, it is anticipated that there will be periodic review and updating of these guidelines. These guidelines are distributed with the understanding that the Brain Trauma Foundation, the National Highway Traffic Safety Administration, and the other organizations that have collaborated in the development of these guidelines are not engaged in rendering professional medical services. If medical advice or assistance is required, the services of a competent physician should be sought. The recommendations contained in these guidelines may not be appropriate for use in all circumstances. The decision to adopt a particular recommendation contained in these guidelines must be based on the judgment of medical personnel, who take into consideration the facts and circumstances in each case, and on the available resources.

METHODOLOGY:

GUIDELINE DEVELOPMENT RATIONALE AND PROCESS

The stimulus for clinical practice guideline development comes from three sources: 1) health care providers who want a cohesive approach to the care of their patients; 2) patients who want the best, most up-to-date care for their health problems; and 3) payers, including the government, that want the best care for the lowest cost. However, because the best approach to care is not always obvious, many medical practitioners have attempted over the years to develop guidelines to help direct patient-care decisions. In developing such guidelines, they choose between two methodologies: consensus-based or evidence-based. Evidence-based guidelines have an advantage over consensus-based guidelines in that they are objective and less susceptible to personal or professional bias. Thus, the methodology chosen for these guidelines is evidence-based, and the guidelines follow the recommendations of the Institute of Medicine (IOM) Committee to Advise the Public Health Service on Clinical Practice Guidelines outlined below:[1]

1. There should be a link between the available evidence and the recommendations.
2. Empirical evidence should take precedence over expert judgment in the development of guidelines.
3. The available scientific literature should be searched using appropriate and comprehensive search terminology.
4. A thorough review of the scientific literature should precede guideline development.
5. The evidence should be evaluated and weighted, depending on the scientific validity of the methodology used to generate the evidence.
6. The strength of the evidence should be reflected in the strength of the recommendations, reflecting scientific certainty (or lack thereof).
7. Expert judgment should be used to evaluate the quality of the literature and to formulate guidelines when the evidence is weak or nonexistent.
8. Guideline development should be a multidisciplinary process, involving key groups affected by the recommendations.

The American Medical Association (AMA) and many specialty societies, including the American Association of Neurological Surgeons (AANS) and the American Academy of Neurology (AAN), have further formalized this process,[2,3,4] by designating specific relationships between the strength of evidence and the strength of recommendations, a task the IOM committee declined to undertake. Evidence is indexed into several classes. **Class I evidence** is derived from the strongest studies of therapeutic interventions (randomized

controlled trials) in humans. In this paradigm, Class I evidence is used to support treatment recommendations of the highest order, called practice **standards. Class II evidence** consists of comparative studies with less strength (nonrandomized cohort studies, randomized controlled trials with significant design flaws, and case-control studies) that are used to support recommendations called **guidelines**. **Class III** evidence consists of other sources of information, including case series and expert opinion, that support practice **options**. Standards, guidelines, and options reflect a high, moderate, or unclear clinical certainty, respectively, as indicated by the scientific evidence available. The overall term for all of the recommendations is **practice parameters**. Because so few practice standards exist, the most common term used to describe the whole body of recommendations is practice guidelines. Thus, we have entitled this document *Guidelines for the Prehospital Management of Traumatic Brain Injury.*

The IOM committee responsible for articulating the foundation of evidence-based guidelines recommended caution to those using the paradigm outlined above to avoid having a poorly designed randomized controlled trial take precedence over a well-designed case-control or nonrandomized cohort study. However, the authors of this guideline attempted to avoid such pitfalls by following the example of the authors of the *Guidelines for the Management of Severe Head Injury*, who carefully evaluated the quality and type of each study before classifying it. In this way, a Class II study that did not provide adequate (at least six months) follow-up information was reclassified as a Class III study. Similarly, a randomized controlled trial that had inappropriate outcome measures was reclassified as a Class II study. All of these criteria apply to practice parameters that pertain to *treatment.* For an assessment of the literature that pertains to *prognosis, diagnosis,* and *clinical assessment*, completely different criteria must be used. These are described below.

For clinical assessments, such as measuring pupillary response, Glasgow Coma Scale, or hypotension, we must be assured that the measure is reliable. Reliability means that different people with different backgrounds make an observation and see the same thing most of the time. Fortunately, good studies of the reliability of pupillary response, Glasgow Coma Scale, and hypotension have been carried out and will be discussed in the sections on assessment.

If we use clinical assessments, such as diagnostic tests, particularly as predictors of poor outcome, we must be able to determine whether the diagnostic test has sensitivity, specificity, and positive or negative predictive value. In this paradigm, the most important aspect of diagnostic assessment is positive predictive value, which represents the number of patients who had the clinical sign or prognostic indicator and had a poor outcome. For this statistic to be meaningful and useful, the guideline task force required a positive predictive value of 70% or greater to make a strong recommendation. To then relate clinical assessment to outcome requires different criteria for evaluation using studies of prognosis.

As with studies of therapeutic effectiveness, prognosis studies (including prognosis with treatment) can be strong or weak. In the strongest studies, the patients should:

- be seen at a uniform time in their diseases (e.g., within 24 hours of injury),
- be followed over time (e.g., for at least six months after injury),
- have their outcomes measured definitively and reliably (e.g., mortality or Glasgow Outcome Score),
- be part of a continuous or defined cohort of patients (e.g., an ongoing, prospectively collectable database), and

- be examined for extraneous prognostic variables, such as underlying disease or age (e.g., use of appropriate statistics, such as multivariate analysis)

To use the same designations (Class I, II, and III) as those used for therapeutic effectiveness, the guideline task force developed the following paradigm: Class I included studies with all of the five characteristics listed above; Class II included studies exhibiting four of the five characteristics (including prospectively collected data); and Class III included studies exhibiting three or fewer of the five characteristics. Using this classification scheme, significant papers were evaluated and listed in the evidence tables within each section. It should be noted that a study, such as a case series, that might be designated as Class I by the above criteria would only be a Class III if it is included as a study on therapeutic effectiveness. Unlike therapeutic effectiveness, studies on prognosis cannot be transposed directly from classification to recommendation. In the guidelines' sections on assessment, which include prognosis studies, therefore, the guideline task force summarized the evidence rather than made recommendations.

These guidelines used a multidisciplinary approach by involving surgeons, other physicians, paramedics, and other Emergency Medical Services (EMS) personnel in retrieving, reviewing, and evaluating the literature. These members of the guideline task force then cooperated in formulating the guidelines during several work sessions aimed at completing understandable and applicable recommendations based on the best evidence available.

These guidelines, entitled *Guidelines for Prehospital Management of Traumatic Brain Injury,* cover three main areas: Assessment Triage, Prehospital Treatment, and Hospital Transport Decisions. A consensus assessment and treatment algorithm is included to provide an overview of all these aspects of management. The members of the guideline task force were selected for their academic expertise in traumatic brain injury (TBI), their knowledge of EMS systems, and/or their experience in guideline development in TBI. Each member of the guideline task force was assigned a topic, for which the member conducted a MEDLINE search, reviewed and graded clinical articles pertinent to the topic, and then wrote a report. Their reports were critically reviewed by the entire guideline task force in subsequent meetings, resulting in a draft version of the guidelines. At several points during the development process, a review team comprised of the major national associations that focus on trauma or EMS systems evaluated the document. Several draft documents were produced and evaluated before the final document was agreed on.

The Brain Trauma Foundation (BTF) of New York City managed the guideline project under a grant from the U.S. Department of Transportation, National Highway Traffic Safety Administration (NHTSA). BTF is a not-for-profit organization dedicated to restoring the injured brain through medical personnel education and clinical research. During the past six years, BTF funded and coordinated efforts to compile scientific evidence and educational material to improve patient care through physician education. In 1995, BTF developed and published the *Guidelines for the Management of Severe Head Injury*. This evidence-based document is for physicians addressing the care of the brain-injured patient. The *Guidelines*, updated this year, were approved by the American Association of Neurological Surgeons and endorsed by the World Health Organization's Committee on Neurotraumatology. BTF has promoted the *Guidelines* nationally and internationally through lectures, presentations, and the hands-on training of physicians. The *Guidelines* form the core of BTF's effort in the area of

clinical care and provided the template for this document, *Guidelines for Prehospital Management of Traumatic Brain Injury.*

Recognizing that correct identification of brain injury and prompt resuscitation and treatment can significantly improve the chances of a good outcome even in severe traumatic brain injury, NHTSA awarded a grant to BTF in 1998 to extend its efforts in TBI guideline development to prehospital care. The goal of the grant is to carry out that task to assist EMS providers in assessing and treating patients with TBI.

References

1. Clinical Practice Guidelines: Directions for a new program. Committee to Advise the Public Health Service on Clinical Practice Guidelines (Institute of Medicine). Washington, D.C.: National Academy Press, 1990.
2. Eddy DM: Designing a practice policy: standards, guidelines, and options. JAMA 26(3):3077-3084, 1990.
3. Walters, BC: Clinical Practice Parameter Development in Neurosurgery, in Bean J (ed): Neurosurgery in Transition Baltimore, 1998, pp. 99-111.
4. Rosenberg J, Greenberg MK: Practice Parameters: strategies for survival into the nineties. Neurology 42:1110-1115, 1992.

ASSESSMENT:

OXYGENATION AND BLOOD PRESSURE

I. Conclusions

A. Hypoxemia (< 90% arterial hemoglobin oxygen saturation) or hypotension (< 90 mm Hg systolic blood pressure)* are significant parameters associated with a poor outcome in patients with severe head injury in the prehospital setting.

B. Measuring hypoxemia and hypotension:
   1. How to measure:
      a) Blood oxygenation: Percentage of blood oxygen saturation should be measured with a pulse oximeter.
      b) Blood pressure: Systolic (SBP) and diastolic blood pressure (DBP) should be measured using the most accurate method available under the circumstances.
   2. When to measure: Oxygenation and blood pressure should be measured as often as possible and should be monitored continuously if possible.
   3. Who should measure: Trained medical personnel should measure oxygenation and blood pressure.

* In children, Class II evidence indicates that SBP is linked to poor outcome according to the following age groups: SBP < 65 mm Hg (0-1 year of age); < 75 (2-5 years); < 80 (6-12 years); < 90 (13-16 years). Therefore, the above values should be considered hypotension for the corresponding age groups with severe TBI.

II. Overview

A growing body of evidence indicates that in severe head injury secondary insults occur frequently and exert a profound influence on outcome. This influence appears to differ markedly from that of hypoxemic or hypotensive episodes of similar magnitude occurring in trauma patients who do not have neurologic involvement. Therefore, we attempted to determine whether any strong evidence exists to support threshold values for oxygenation and blood pressure.

III. Search Process

MEDLINE was searched from 1966 to 1998 using the following search terms: 1) head injury and (hypoxemia or hypotension) and human subject; 2) head injury and (field or

prehospital) and (treatment or management or resuscitation). This search produced 238 references. Of these, 94 references were directly relevant to outcome analysis and clinical orientation; these were individually reviewed for design and content. Some studies of in-hospital patients with severe head injury and hypotension were used to corroborate prehospital hypotension studies. The analysis presented here is based on this literature review.

IV. Scientific Foundation

The deleterious influence of hypotension and hypoxemia on the outcome of patients with severe head injury was analyzed from a large (717 patients), prospectively collectable dataset from the Traumatic Coma Data Bank (TCDB).[1,2] The TCDB study demonstrated that prehospital hypotension (defined as a single observation of < 90 mm Hg SBP) and hypoxemia (defined as apnea, cyanosis, or a hemoglobin oxygen saturation < 90% in the field or as a PaO_2 < 60 mm Hg by arterial blood gas analysis) were among the five most powerful predictors of outcome. These clinical findings were statistically independent of other major predictors, such as age, admission Glasgow Coma Scale (GCS) score, admission GCS motor score, intracranial diagnosis, and pupillary status. A single episode of hypotension was associated with a doubling of mortality and an increased morbidity when compared with a matched group of patients without hypotension (Table A). Notably, the TCDB study defined hypotension and hypoxemia as a single reported incidence that meets the definition of each and does not require a protracted duration for secondary insult.

A smaller Class II study from Australia corroborated the above findings; particularly with respect to the effects of hypotension on outcome.[3] The clinical predictors of mortality derived from this study were identical. Notably, in both studies, the two predictors with the potential for being altered through clinical manipulations are hypotension and hypoxemia.

A retrospective review analyzing outcome in children (ages 3 months to 14 years) with GCS scores 6 to 8 found that patients with poor outcome had significantly more hypotensive episodes (hypotension levels with respect to age, defined above in "Conclusions"*) in the prehospital setting.[4]

Another retrospective review of prospectively collectable data in children younger than 17 years of age corroborated these results.[5] In that study, hypotension markedly increased morbidity and mortality independent of other predictors of outcome, eliminating the improvement in survival generally afforded by youth. These data are similar to those in other retrospectively analyzed Class II and III in-hospital reports in adults.[6-15]

No Class I study has directly addressed the efficacy of preventing or correcting early hypotension to improve outcome. However, a subgroup of severe head injury patients was subjected to post hoc analysis in a recent prospective, randomized, placebo-controlled, multicenter trial that compared the efficacy of administering 250 ml of hypertonic saline versus normal saline as the initial resuscitation fluid in hypotensive trauma patients. In that trial, the hypertonic saline group had improved blood pressure responses, decreased overall fluid requirements, and associated improvements in survival. The investigators retrospectively reviewed the records of the subgroup of patients with severe head injuries and found that this group had statistically significant improvement in survival to discharge.[16] Although this was a post hoc analysis of Class I data, it strongly suggests that elevating the blood pressure in hypotensive severe head injury patients improves outcome.

The value of 90 mm Hg systolic pressure to delineate the threshold for hypotension has arisen arbitrarily and is more a statistical than a physiologic parameter. In considering the evidence concerning the influence of cerebral perfusion pressure (CPP) on outcome, it is possible that systolic pressures significantly > 90 mm Hg would be desirable during the prehospital and resuscitation phase, but no studies have been performed to corroborate this. The importance of mean arterial pressure (MAP), as opposed to systolic pressure, should also be stressed, not only because of its role in calculating (CPP) = [MAP – intracranial pressure (ICP)], but because the lack of a consistent relationship between the systolic and mean pressures makes calculations based on systolic values unreliable. It may be valuable to maintain MAPs considerably above those represented by systolic pressures of 90 mm Hg throughout the patient's course.

A study from Italy of 50 patients with head trauma transported by helicopter revealed that 55% had an oxygen saturation < 90% measured at the scene prior to intubation.[17] Both hypoxemia and hypotension had significant negative impacts on outcome. Of the 28 patients who were hypoxemic, 13 did not have associated hypotension. There was a significant ($p < 0.005$) association between arterial desaturation and poor outcome.

Oxygen Saturation	Mortality	Severe Disability
> 90%	14.3% (3/21)	4.8% (1/21)
60-90%	27.3% (6/22)	27.3% (6/22)
< 60%	50% (3/6)	50% (3/6)

The incidence of hypoxemia and hypercarbia has been reported in several studies. A recent retrospective chart review of 72 pediatric patients with a GCS score between 6 and 8 who were admitted to a single center reported that 13% of patients had a documented hypoxic episode and 6% had hypercarbia. However, that study could not demonstrate a relationship to outcome.[4]

A small study from England evaluating the use of pulse oximetry in the field and in a moving ambulance for trauma patients documented that 16% of patients had an SaO2 < 75%, and an additional 28% had oxygen saturations between 75% and 90%. The major cause of low oxygen saturation was severe head injury.[18]

V. Summary

Early postinjury episodes of hypotension or hypoxemia greatly increase morbidity and mortality from severe head injury. At present, the precise definitions of hypotension and hypoxemia are unclear in these patients. However, ample Class II evidence exists regarding hypotension, defined in these studies as a single observation of an SBP < 90 mm Hg (see previous note in "Conclusions"* for blood pressure levels for children), or hypoxemia, defined in these studies as apnea or cyanosis in the field or an arterial oxygen saturation < 90%. The evidence indicates that these values must be avoided, if possible, or rapidly corrected in severe head injury patients. Strong Class II evidence suggests that raising the blood pressure in hypotensive, severe head injury patients improves outcome in proportion to the efficacy of the resuscitation.[18]

VI. Key Issues for Future Investigation

Clinical trials are needed in the following areas:

1. Do prospective data correlate magnitude and duration of hypotensive and hypoxic episodes to outcome?
2. Is mean arterial pressure (MABP = [DBP + 1/3 (SBP - DBP)]) a more accurate indicator of hypotension than systolic blood pressure?
3. How accurate are devices that measure systolic, diastolic, and mean blood pressures during transportation?
4. A similar assessment to that outlined in (#2) above is needed for arterial oxygen saturation.
5. Prospective studies on the above four points are needed for the pediatric population.

The two major areas needing investigation are 1) the critical values for duration and magnitude of hypotensive and hypoxemic episodes and how they affect neurological outcome, and 2) the optimal resuscitation protocol (fluid type, route of administration, etc.) for resuscitating the patient with severe head injury. The former question is not a subject for a controlled trial for ethical reasons and, therefore, is best undertaken using a prospective data collection study with the precise collection of prehospital blood pressure and oxygenation data, which is then correlated with outcome. The latter question can be studied in prospective, randomized investigations, several of which are presently underway.

Table A.
Outcome by Secondary Insult at Time of Arrival at Traumatic Coma Data Bank Hospital for Mutually Exclusive Insults[a]

			Outcome (%)		
Secondary Insults	**Number of Patients**	**% Total Patients**	**Good or Moderately Disabled**	**Severely Disabled or Vegetative**	**Dead**
Total cases	699	100%	43	20	37
Hypoxemia[b]	78	11	45	22	33
Hypotension[b]	113	16	26	14	60
Neither	456	65	51	22	27
Both	52	8	6	19	75

[a]Adapted from Chesnut RM, Marshall LF, Klauber MR, et al: The role of secondary brain injury in determining outcome from severe head injury. J Trauma 34:216-222, 1993.
[b]Hypoxemia PaO_2 < 60 mm Hg; hypotension: SBP < 90 mm Hg.

VII. Evidence Tables

Chesnut,[1] 1993

Description of Study: A prospective study of 717 severe head injury patients admitted consecutively to four centers investigated the effect on outcome of hypotension (SBP < 90 mm Hg) occurring from injury through resuscitation.

Classification: II

Conclusions: Hypotension was a statistically independent predictor of outcome. A single episode of hypotension during this period doubled mortality and also increased morbidity. Patients whose hypotension was not corrected in the field had a worse outcome than those whose hypotension was corrected by time of emergency department arrival.

Fearnside,[3] 1993

Description of Study: A prospective study of 315 severe head injury patients admitted consecutively to a single center investigated prehospital and in-hospital predictors of outcome.

Classification: II

Conclusions: Hypotension (SBP < 90 mm Hg) occurring at any time during a patient's course independently predicts worse outcome.

Gentleman,[6] 1992

Description of Study: A retrospective study of 600 severe head injury patients in three cohorts evaluated regarding the influence of hypotension on outcome and the effect of improved prehospital care in decreasing its incidence and negative impact.

Classification: III

Conclusions: Improving prehospital management decreased the incidence of hypotension, but its impact on outcome in patients suffering hypotensive insults maintained as a statistically significant, independent predictor of poor outcome. Management strategies that prevent or minimize hypotension in the prehospital phase improve outcome from severe head injury.

Hill,[7] 1993

Description of Study: Retrospective study of the prehospital and ED resuscitative management of 40 consecutive multitrauma patients. Hypotension (SBP ≤ 80 mm Hg) correlated strongly with fatal outcomes. Hemorrhagic hypovolemia was the major etiology of hypotension.

Classification: III

Conclusions: Improving the management of hypovolemic hypotension is a major potential mechanism for improving the outcome from severe head injury.

Jeffreys,[8] 1981

Description of Study: A retrospective review of hospital records of 190 head injury patients who died after admission. Hypotension was one of the four most common avoidable factors correlated with death.

Classification: III

Conclusions: Early hypotension appears to be a common and avoidable cause of death in severe head injury patients.

Kohi,[9] 1984

Description of Study: A retrospective evaluation of 67 severe head injury patients seen over a 6-month period was correlated with 6-month outcome. For a given level of consciousness, the presence of hypotension resulted in a worse outcome than would have been predicted.

Classification: III

Conclusions: Early hypotension increases the mortality and worsens the prognosis of survivors in severe head injury.

Kokoska,[4] 1998

Description of Study: A retrospective review of 72 pediatric patients (ages 3 months–14 years) with regard to hypotensive episodes and outcome. Hypotensive episode was defined as a blood pressure reading of less than the fifth percentile for age that lasted longer than 5 minutes.

Classification: II

Conclusions: Prehospital, ED, and ICU hypotensive episodes were significantly associated with a poor outcome.

VII. Evidence Tables (continued)

Marmarou,[2] 1991

Description of Study: From a prospectively collected database of 1,030 severe head injury patients, all 428 patients who met ICU monitoring criteria were analyzed for monitoring parameters that determined outcome and their threshold values. The two most critical values were the proportion of hourly ICP readings > 20 mm Hg and the proportion of hourly SBP readings < 80 mm Hg.

Classification: II

Conclusions: The incidence of morbidity and mortality resulting from severe head injury is strongly related to ICP and hypotension measured during the course of ICP management.

Miller,[10] 1982

Description of Study: 225 severe head injury patients were prospectively studied with respect to the influence of secondary insults on outcome. Hypotension (SBP < 95 mm Hg) was significantly associated with increased morbidity and mortality. The predictive independence of hypotension in comparison with other associated factors, however, was not investigated.

Classification: II

Conclusions: Strong statistical relationship between early hypotension and increased morbidity and mortality from severe head injury.

Miller,[11] 1978

Description of Study: 100 consecutive severe head injury patients were prospectively studied with respect to the influence of secondary insults on outcome (report of first 100 patients in subsequent report of 225 patients [vide supra]). Hypotension (SBP < 95 mm Hg) associated with a trend (not statistically significant) toward worse outcome in entire cohort; trend met statistical significance for patients without mass legions. Seminal report relating early hypotension to increased morbidity and mortality. Influence of hypotension on outcome not analyzed independently from other associated factors.

Classification: II

Conclusions: First prospective report implicating early hypotension as a major predictor of increased morbidity and mortality from severe head injury.

Narayan,[12] 1982

Description of Study: Retrospective analysis of the courses of 207 consecutively admitted severe head injury patients. Management included aggressive attempts to control ICP using a threshold of 20 mm Hg. Outcome was significantly correlated with the ability to control ICP.

Classification: III

Conclusions: ICP control using a threshold of 20 mm Hg as part of an overall aggressive treatment approach to severe head injury may be associated with improved outcome.

Pietropaoli,[13] 1992

Description of Study: Retrospective review of the impact of intraoperative hypotension (SBP < 90 mm Hg) on 53 otherwise normotensive severe head injury patients who required early surgery (within 72 hours of injury). The mortality rate was 82% in the normotensive group ($p < 0.001$). The duration of intraoperative hypotension was inversely correlated with Glasglow Outcome Scale score using linear regression ($R = -0.30$; $p = 0.02$).

Classification: III

Conclusions: Early hypotension is correlated with significantly increased mortality from severe head injury in a duration-dependent fashion.

Pigula,[5] 1993

Description of Study: 58 children (< 17 years old) with severe head injuries were prospectively studied for the effect of hypotension (SBP < 90 mm Hg) on outcome. An episode of hypotension decreased survival fourfold. This finding was confirmed in a concomitant analysis of the effect of hypotension on outcome in 509 patients in the National Pediatric Trauma Registry. Hypotension appeared to eliminate any neuro-protective mechanisms normally afforded by age.

Classification: II

Conclusions: The detrimental effects of hypotension (SBP < 90 mm Hg) on outcome appear to extend to children.

Rose,[14] 1977

Description of Study: Retrospective review of hospital records and necropsy material of 116 head injury patients who talked before dying. Hypotension was a major avoidable factor related to the increased mortality in this group.

Classification: III

Conclusions: Hypotension is a major avoidable cause of increased mortality in patients with moderate head injury.

VII. Evidence Tables (continued)

Stocchetti,[17] 1996

Description of Study: A prospective study of data collected at the accident scene from 50 severely head-injured patients rescued by helicopter. Instead of classifying blood pressure or oxygen saturation measurements as above or below a certain threshold, SBP was classified as < 60 mm Hg, 60-80 mm Hg, 81-99 mm Hg, or > 99 mm Hg; arterial oxygen saturation measured via pulse oximeter was classified as < 60%, 60-80%, 81-90%, or > 90%. Patients with lower blood pressure or oxygen saturation fared worse than those with higher values.

Classification: II

Conclusions: Low prehospital blood pressures or oxygen saturations are associated with worse outcomes. Arterial oxygen saturation of 80% or lower was associated with a 47% mortality compared with 15% mortality when oxygen saturation was > 80%.

Vassar,[16] 1993

Description of Study: Prospective, randomized, controlled, multicenter trial comparing the efficacy of administering 250 ml of hypertonic saline vs normal saline as the initial resuscitation fluid in facilitating the resuscitation and improving the outcome of hypotensive trauma patients. In this trial, the hypertonic saline group had significantly improved blood pressure responses and decreased overall fluid requirements. Although there was an associated improvement in survival for the overall group, it did not reach statistical significance. Post hoc analysis of the severe head injury group (Class II analysis), however, revealed that the hypertonic saline group had a statistically significant improvement in survival to discharge.

Classification: II

Conclusions: Raising the blood pressure in hypotensive severe head injury patients improves outcome in proportion to the efficacy of the resuscitation.

Winchell,[15] 1996

Description of Study: From a trauma registry of 1,013 patients, 157 patients with severe anatomic head injury (i.e., Head and Neck Abbreviated Injury Scale score of 4 or 5) were identified. These included 88 patients with a GCS > 8. The 157 patients had a total of 831 episodes of systolic hypotension (< 100 mm Hg) while in the ICU. The total number and the average daily number of hypotensive events were independent predictors of death in the ICU.

Classification: III

Conclusions: Transient hypotensive (SBP < 100 mm Hg) episodes in the ICU are associated with a significantly worse outcome. Mortality rose from 9-25% in patients who had 1-10 hypotensive episodes, and to 35% in patients with >10 episodes.

VIII. References

1. Chesnut RM, Marshall LF, Klauber MR, et al.: The role of secondary brain injury in determining outcome from severe head injury. J Trauma 34:216-222, 1993.
2. Marmarou A, Anderson RL, Ward JD, et al.: Impact of ICP instability and hypotension on outcome in patients with severe head trauma. J Neurosurg 75:S159-S166, 1991.
3. Fearnside MR, Cook RJ, McDougall P, et al.: The Westmead Head Injury Project outcome in severe head injury. A comparative analysis of pre-hospital, clinical, and CT variables. Br J Neurosurg 7:267-279, 1993.
4. Kokoska ER, Smith GS, Pittman T, et al.: Early hypotension worsens neurological outcome in pediatric patients with moderately severe head trauma. J Pediatr Surg 33:333-338, 1998.
5. Pigula FA, Wald SL, Shackford SR, et al: The effect of hypotension and hypoxemia on children with severe head injuries. J Pediatr Surg 28:310-314; discussion 315-316, 1993.
6. Gentleman D: Causes and effects of systemic complications among severely head injured patients transferred to a neurosurgical unit. Int Surg 77:297-302, 1992.
7. Hill DA, Abraham KJ, West RH: Factors affecting outcome in the resuscitation of severely injured patients. Aust N Z J Surg 63:604-609, 1993.
8. Jeffreys RV, Jones JJ: Avoidable factors contributing to the death of head injury patients in general hospitals in Mersey Region. Lancet 2:459-461, 1981.
9. Kohi YM, Mendelow AD, Teasdale GM, et al.: Extracranial insults and outcome in patients with acute head injury—relationship to the Glasgow Coma Scale. Injury 16:25-29, 1984.
10. Miller JD, Becker DP: Secondary insults to the injured brain. J Royal Coll Surg (Edinburgh) 27:292-298, 1982.
11. Miller JD, Sweet RC, Narayan R, et al.: Early insults to the injured brain. JAMA 240:439-442, 1978.
12. Narayan R, Kishore P, Becker D, et al.: Intracranial pressure: to monitor or not to monitor? A review of our experience with head injury. J Neurosurg 56:650-659, 1982.
13. Pietropaoli JA, Rogers FB, Shackford SR, et al.: The deleterious effects of intraoperative hypotension on outcome in patients with severe head injuries. J Trauma 33:403-407, 1992.
14. Rose J, Valtonen S, Jennett B: Avoidable factors contributing to death after head injury. Br Med J 2:615-618, 1977.
15. Winchell RJ, Simons RK, Hoyt DB: Transient systolic hypotension. Arch Surg 131:533-539, 1996.
16. Vassar MJ, Perry CA, Holcroft JW: Prehospital resuscitation of hypotensive trauma patients with 7.5% NaCl versus 7.5% NaCl with added dextran: A controlled trial. J Trauma 34:622-632, 1993.
17. Stocchetti N, Furlan A, Volta F: Hypoxemia and arterial hypotension at the accident scene in head injury. J Trauma 40: 764-767, 1996.
18. Silverston P: Pulse oximetry at the roadside: a study of pulse oximetry in immediate case. Br Med J 298:711-713, 1989.

ASSESSMENT:

GLASGOW COMA SCALE SCORE

I. Conclusions:

A. No Class I evidence is available on which to base conclusions for this parameter. Studies performed or initiated in the prehospital setting were reviewed in order to determine this.

B. Class II data indicate that the prehospital measurement of the Glasgow Coma Scale (GCS) score (Table A) is a significant and reliable indicator of the severity of head injury, particularly in association with repeated scoring and improvement or deterioration of the score over time. A single field measurement of the GCS score cannot predict outcome; however, a decrease of two points with a GCS score of nine or lower indicates serious injury. Prehospital- and hospital-based study data indicate that a GCS score of 3 to 5 has at least a 70% positive predictive value for poor outcome.

C. Obtaining the GCS score:

1. How to measure the GCS score:
   a. The GCS score must be obtained through interaction with the patient (e.g., by giving verbal directions or, for patients unable to follow commands, by applying a painful stimulus such as nail bed pressure or axillary pinch).
2. When to measure the GCS score:
   a) The GCS score should be measured after the initial assessment, after a clear airway is established, and after necessary ventilatory or circulatory resuscitation has been performed.
   b) The GCS score should be measured preferably prior to administering sedative or paralytic agents, or after these drugs have been metabolized.
3. Who should measure the GCS score?
   a) The GCS score can be measured fairly reliably by trained Emergency Medical Services personnel.

II. Overview

Teasdale and Jennett developed the Glasgow Coma Scale in 1974 as an objective measure of the level of consciousness after head trauma.[1] It has since become the most widely used clinical measure of the severity of traumatic brain injury (TBI). The GCS permits a repetitive and moderately reliable standardized method of reporting and recording ongoing neurologic

evaluations even when performed by a variety of health care providers. The GCS evaluates three independent responses: eye opening, motor response, and verbal response.

Teasdale and Jennett stated that for patients unable to follow commands, the motor response is scored on the best-observed response to a standardized stimulus. The stimulus can be blunt pressure applied to the nail bed using a pencil or a pinch of the patient's anterior axillary skin.

The GCS score, however, can be affected by post-traumatic complications that may impair neurologic response that field providers can recognize and treat immediately. Hypoxemia and hypotension, which are common complications after trauma, have been shown to negatively affect GCS scoring. Therefore, these complications should be corrected and the patient resuscitated as fully as possible prior to measuring the GCS score.

Another GCS scoring difficulty involves preverbal children. The American College of Emergency Physicians and the American Academy of Pediatrics in its 1998 publication *APLS—The Pediatric Emergency Medicine Course*[2] agreed that for children under the age of two, a modified GCS that assigns a full verbal score (5) for spontaneous cooing, is appropriate.

A number of studies confirmed a moderate degree of inter- and intrarater reliability in scoring the GCS, including GCS scores that prehospital EMS providers obtain. [3-5]

On the other hand, some educational courses for Emergency Medical Care providers utilize the AVPU system described below to assess the level of consciousness during the initial assessment.[6] This system classifies the patient into one of four categories: 1) **A**lert; 2) responds to **V**erbal stimuli; 3) responds to **P**ainful stimuli; or 4) **U**nresponsive. Although the system is useful for rapid assessment, it provides only a gross evaluation of the neurologic status of the patient and does not quantify the degree of motor dysfunction. Quantifying motor dysfunction in the GCS score from 1 to 6 allows a finer distinction of neurologic status (e.g., extensor verses flexion posturing) that has been demonstrated to be prognostic and guide therapy, such as hyperventilation for extensor posturing.

III. Search Process

A MEDLINE search was conducted from 1970 to 1998 using the key words "head injury" with "GCS" or "level of consciousness," using "emergency medical services" or "prehospital care" or "field care" as search parameters. The search resulted in a list of 54 journal articles. The abstracts of all articles were reviewed, and those that appeared to test the strength of prehospital GCS scoring as a marker of head injury severity were selected for review of the complete article. Manual searches of the reference lists from these articles, as well as prehospital journals not listed in MEDLINE, were also reviewed for additional relevant citations. This process resulted in five articles dealing with the prehospital measurement of the GCS score, four of which related the prehospital GCS score to outcome.

IV. Scientific Foundation

Modern prehospital treatment of general trauma patients and, in particular, head-injured patients, includes such interventions as endotracheal intubation, often accompanied by pharmacological sedation or paralysis. Recognizing and treating airway or ventilatory compromise constitutes the first steps in the field management of the trauma victim.[6,7] These interventions have the undesirable side effect of complicating the prehospital and early hospital determination of valid GCS scores. Two surveys of major trauma centers in the United States found that practice varies substantially in assessing the initial GCS score when patients are

admitted following such prehospital treatment.[8,9] Finally, many emergency medical systems often do not record the GCS score in head-injured patients.[10] These factors may explain the dearth of prehospital studies on the use of the GCS score in the field setting and its correlation to patient outcome.

Change in GCS Score from the Field to the Emergency Department

Winkler evaluated 33 consecutive head-injured patients, comparing the field GCS score to the GCS score obtained on arrival in the emergency department (ED).[11] Patients were grouped according to their final outcome (no deficits, minor deficits, major deficits, or dead). All four groups had similar GCS scores in the field. However, those who ultimately were discharged with no or minor deficits had significant improvements (> 2 points) in the GCS score when they had been assessed in the ED. In contrast, those who had significant deficits or who died showed little or no improvement in the GCS score when assessed in the ED.

Servadei, et al. used change between the prehospital setting GCS score and the ED score as one criterion to determine the need for operative evacuation of post-traumatic subdural hematomas.[12] For example, a patient whose GCS score was unchanged or improved was often a candidate for nonoperative management. On the other hand, if the GCS score deteriorated from the field to the ED, there was a significant likelihood of the need for surgical intervention. Other criteria involved in the decision to operate included the size of the hematoma and the amount of midline shift. Patients treated surgically in that study had an average two-point decrease in the GCS score, whereas those treated expectantly did not change significantly.

Prehospital GCS Score and Patient Outcome

Another retrospective study evaluated the GCS score measured at various times, including the prehospital setting, and related the score to early (at the time of discharge) and late (5-7 years postinjury) outcome, as measured by the Glasgow Outcome Score.[13] The field GCS score related well to early outcome, that is, patients having a GCS score of 6 to 15 were thirty times more likely to have a good outcome than those with a GCS score of 3 to 5. This relationship did not hold up for late outcome. It should be noted that 24% of patients included in the study did not have a field GCS score recorded.

Baxt and Moody compared advanced prehospital care provided by aeromedical scene responders to ground transport providers using less sophisticated medical interventions.[14] The mortality rates for GCS scores performed in the field by the flight team were obtained (Table B). The predictive value for mortality of a GCS = 3 to 5 is 50% and 61% for helicopter and ground transported patients, respectively. The predictive value for a GCS = 6 to 8 is 14.5% and 15.3% respectively. In addition, the predictive value for a bad outcome (dead, vegetative, or severely disabled) for GCS = 3 to 5 is 81.6% and 84% for helicopter and ground transported patients, respectively; for a GCS = 6 to 8, it is 34.5% and 40.7%, respectively. The study is limited by the fact that the GCS score for ground transported patients was not calculated in the field by the paramedics, but rather on arrival in the ED.

The data from the two preceding sections indicate that the initial prehospital GCS score, particularly deterioration in GCS score from the field to the ED, predicts outcome after severe TBI.

In-hospital GCS score and Patient Outcome

Despite the paucity of prehospital data, the GCS score measured in the hospital has been shown to have a significant correlation with patient outcome following severe TBI, either as a sum score or just as the motor component. In a prospective study by Narayan, et al. a positive predictive value of 77% for a poor outcome (dead, vegetative, or severely disabled) was measured for patients with a GCS score of 3 to 5 and 26% for those with a GCS score of 6 to 8.[15] In a study from Australia, a significant inverse correlation was found between the initial GCS score in the hospital (obtained 6 to 48 hours after injury) and mortality (Table C).[16]

In another series of patients with closed head injury entered into the U.S. Traumatic Coma Data Bank, mortality rates for patients with initial GCS scores of 3, 4, or 5 were 78.4%, 55.9%, and 40.2%, respectively.[17] Of note, however, is that 4.1%, 6.3%, and 12.2% of the three groups, respectively, had good outcome.

The relationship of outcome to GCS score has also been demonstrated in children in hospital-based studies. Using only the motor component of the GCS and a dichotomized outcome of good (moderate, no disability) versus bad (dead, vegetative, or severely disabled), a study of 109 children who sustained TBI revealed that the GCS motor component alone was indicative of outcome (Table D).[18]

Most studies that focused on the GCS score and patient outcome have been in patients with GCS scores between 3 and 8. A number of studies[19-23] (Table E) evaluated the need for diagnostic evaluation and the incidence of intracranial pathology in patients with GCS scores of 9 to 15, although most did not address outcome.

Schynoll, et al. conducted a study that attempted to define high-yield criteria that would predict abnormal intracranial findings on brain CTs of patients who sustained head trauma. He reported that 7% of patients with GCS scores of 15 had an abnormal scan, whereas 29% of patients with GCS scores of 13 or 14 and 25% of patients with GCS scores of 9 to 12 had abnormal CT scans.[24] Finally, in a study of multiple trauma patients with head injury who had GCS scores obtained approximately six hours postinjury and outcome evaluated at one month postinjury, good recovery was reported in 99% of patients with GCS scores of 13 to 15, 71% of patients with GCS scores of 9 to 12, and 35% of patients with GCS scores of 3 to 8.[25] Mortality for these groups was 1.15%, 12.5%, and 40.7%, respectively. Thus, it is apparent from these studies that patients with GCS scores of 9 to 13 are at risk for significant intracranial pathology and, based on hospital data, patients with GCS scores of 9 to 12 have the worse outcomes.

Reliability of Prehospital GCS Scoring

One additional question relates to the ability of Emergency Medical Care providers to obtain the GCS score reliably. Menegazzi, et al. used videotaped scenarios of patients with severe, moderate, and mild/no alteration of level of consciousness in a classroom setting to evaluate the inter- and intrarater GCS scoring reliability of paramedics and emergency physicians.[5] He demonstrated inter-rater kappa (agreement beyond chance) values of 0.48, 0.34, and 0.85 for the three groups, respectively, and intrarater values of 0.66 for the physicians and 0.63 for the paramedics, demonstrating moderate reliability in obtaining the GCS score.

V. Summary

Data documenting the prehospital GCS and its relationship to patient outcome after traumatic brain injury is scarce. Limited prehospital GCS data indicate that initial field GCS scores between 3 and 5, as well as lack of improvement or deterioration of GCS score of two points or more from the field to the emergency department, have significant value as predictors of poor patient outcome. GCS scoring should occur after hypoxemia and hypotension are corrected and the patient has been resuscitated. Hospital data indicate that when an initial GCS score is obtained reliably, 20% or more of patients with the worst GCS score (3-5) will survive, and 8% to 10% will have a functional survival.

VI. Key Issues for Further Investigation

A number of issues require study to evaluate the role of the GCS score in the prehospital setting:

1. What is the ability of the initial field GCS score to predict outcome compared with the score obtained postresuscitation or compared with any improvement or deterioration in score during the prehospital phase?
2. How does the presence of central nervous system depressants affect the field measurement of the GCS score and its predictive value? Numerous issues, such as the presence or absence of intoxicants, complicate the field measurement of GCS score. The prehospital measurement of the GCS score in these patients may give a false low score, unrelated to any head injury directly. In addition, sedatives or chemical paralytic agents used to obtain airway control or patient restraint is known to impair the ability to obtain a reliable GCS score in the hospital.
3. Is the motor score alone, obtained in the prehospital setting, a reliable indicator and predictor of outcome? Several studies have suggested that the motor score alone can be used as a reliable measure of the severity of injury after TBI.[10,19] If this is shown to be the case, the difficulties associated with obtaining a full GCS score in patients with facial trauma and orbital swelling or those who have undergone endotracheal intubation can be obviated.
4. Is GCS score a stronger indicator of severity of injury when patients have documented oxygen saturations > 90% and systolic blood pressure > 90 mm Hg?
5. Can any mechanisms, such as training or educational programs, be identified to improve the reliability of GCS scoring?

VII. Evidence Tables

Winkler,[11] 1984

Description of Study: Prospective study of field vs ED GCS score in 33 patients with field GCS < 8 and head trauma, grouped by outcome (I = no deficit, II = minor deficit, III = major deficit, IV = died).

Classification: III

Conclusions:

Mean Field GCS	ED GCS	Outcome
4.14	9.43	I
4.67	7.33	II
4.45	66.27	III
4.33	5.17	IV

Baxt,[14] 1987

Description of Study: Review of 128 patients treated and transported by ground ambulance and 104 patients treated and transported by rotorcraft air ambulance.

Classification: III

Conclusions:

Field GCS	Mortality	
	Ground	Air
3	75%	68%
4	60	23
5	35	36
6	8	13
7	9	14
8	45	18

Massagli, [13] 1996

Description of Study: Retrospective review of 33 children admitted to level I trauma center after severe head injury comparing early and late outcomes to various injury indices.

Classification: III

Conclusions:

Field GCS	Good Outcome	
	Early	Late
3-5	6%	12%
6-15	67	33

VII. Evidence Tables (continued)

Servadei,[12] 1998

Description of Study: Prospective study of 65 patients with acute post-traumatic subdural hematoma comparing the need for surgical evacuation with GCS change from the field to the ED, as well as CT scan findings including size of hematoma and amount of midline shift.

Classification: III

Conclusions:

	GCS		
Evacuation	Field	ED	Mortality
Yes	8.4	6.7	56%
No	7.2	7.2	20

VIIa. Description of the Studies According to the Classification Criteria

First Author	Number of Patients	Prospective	Time	When Was Indicator Measured	What Method
Baxt[14]	232 (128 land, 104 air)	NR	NR	ED for land patients, Field for air patients.	GCS
Massagli[13]	33	N	1985-1986	Field, ED, and in-patient	GCS
Servadei[12]	65	NR	1994-1996	*Prehosp.* & on admission	GCS
Winkler[11]	33	Y	NR	*Prehosp.* & in ED	GCS

Who Did It	Outcome Measure	When	Blinded Observer	Multivariate Statistics	What Statistics
NR	GOS	NR	NR	NR	Chi Square
					Mantzel-Haenszel Test
NR	GOS	Hosp. discharge and 5-7 years Postadmission	NR	NR	Mann-Whitney U test, Spearman Correlation
Emergency Physician, Intensivist, Neurosurgeon	GOS	6 months Postinjury	NR	NR	Chi Square, Fischer's Exact test, Unpaired T-test
Paramedic, Emergency Physician	GOS (modified)	8-12 weeks postinjury	NR	NR	Student's T-test

Table A.
Glasgow Coma Scale (Teasdale[1])

Eye Opening	Motor Response	Verbal Response
Spontaneous - 4	Obeys - 6	Oriented - 5
To speech - 3	Localizes - 5	Confused - 4
To pain - 2	Withdraws - 4	Inappropriate - 3
No Response - 1	Abnormal flexion - 3	Incomprehensible - 2
	Extensor response - 2	None - 1
	None - 1	

Table B.
Aeromedical vs ground transportation death rates in TBI patients (Baxt[14])

GCS Score	Mortality Flight Team	Mortality Ground Transport
3	68%	75%
4	23	60
5	36	35
6	13	8
7	14	9
8	18	45

Table C.
GCS score 6-48 hours after injury and mortality in TBI patients (Fearnside[16])

GCS Score	Mortality
3	65%
4	45
5	35
6	24
7-13	10-15

Table D.
GCS motor score vs good (moderate/no disability) outcome in pediatric TBI (Beca[18])

GCS Motor Score	Good Outcome
1	0%
2	47
3	41
4	78
5	75

Table E.
Incidence of intracranial pathology and surgical intervention in mild TBI patients (Schynoll [24])

GCS Score		13	14	15
Shackford[20]	Abnormal CT scan	33%	17.5%	14.8%
	Craniotomy	10.8	3.8	3.2
Borczuk[21]	Abnormal CT scan	27.5	18.2	5.9
	Craniotomy	7.5	3.6	0.08
Stein[22]	Abnormal CT scan	37.5	24.2	13.2
(Although there was no breakdown by GCS score, 58 of the 265 patients (22%) with abnormal CT findings required surgery.)				
Harad[23]	Abnormal CT scan	17	27	23
(Eleven of the 55 (20%) patients with abnormal CT scans required craniotomy.)				

VIII. References

1. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale. Lancet 2:81-84, 1974.
2. APLS—The Pediatric Emergency Medicine Course. American College of Emergency Physicians and American Academy of Pediatrics, 1998.
3. Braakman R, Avezaat CJ, Maas AI, et al.: Interobserver agreement in the assessment of the motor response of the Glasgow "Coma" Scale. Clin Neurol Neurosurg 80:100-106, 1977.
4. Fielding K, Rowley G: Reliability of assessments by skilled observers using the Glasgow Coma Scale. Aust J Adv Nurs 7:13-21, 1990.
5. Menegazzi JJ, Davis EA, Sucov AN, et al.: Reliability of the Glasgow Coma Scale when used by emergency physicians and paramedics. J Trauma 34:46-48, 1993.
6. Patient assessment and management in PHTLS Basic and Advanced. Prehospital Trauma Life Support. 3rd ed. St. Louis: Mosby-Yearbook, 1994, pp. 50-71.
7. Silvestri S, Aronson S: Severe head injury: prehospital and emergency department management. Mt Sinai J Med 64:329-338, 1997.
8. Marion DW, Carlier PM: Problems with initial Glasgow Coma Scale score assessment caused by the prehospital treatment of head-injured patients: results of a national survey. J Trauma 36:89-95, 1994.
9. Buechler CM, Blostein PA, Koestner A, et al: Variation among trauma centers' calculation of Glasgow Coma Scale score: results of a national survey. J Trauma 45:429-432, 1998.
10. Ross SE, Leipold C, Terregino C, et al.: Efficacy of the motor component of the Glasgow Coma Scale in trauma triage. J Trauma 45:42-44, 1998.
11. Winkler JV, Rosen P, Alfrey EJ: Prehospital use of the Glasgow Coma Scale in severe head injury. J Emerg Med 2:1-6, 1984.
12. Servadei F, Nasi MT, Cremonini AM, et al.: Importance of a reliable admission Glasgow Coma Scale score for determining the need for evacuation of post-traumatic subdural hematomas: a prospective study of 65 patients. J Trauma 44:868-873, 1998.
13. Massagli TL, Michaud LJ, Rivara FP: Association between injury indices and outcome after severe traumatic brain injury in children. Arch Phys Med Rehabil 77:1125-1132, 1996.

14. Baxt WG, Moody P: The impact of advanced prehospital emergency care on the mortality of severely brain-injured patients. J Trauma 27:365-369, 1987.
15. Narayan RK, Greenberg RP, Miller JD, et al.: Improved confidence of outcome prediction in severe head injury. J Neurosurg 54:751-762, 1981.
16. Fearnside MR, Cook RJ, McDougall P, et al.: The Westmead Head Injury Project outcome in severe head injury. A comparative analysis of pre-hospital, clinical and CT variables. Br J Neurosurg 7:267-279, 1993.
17. Marshall LF, Gautille T, Klauber MR, et al.: The outcome of severe closed head injury. J Neurosurg 75:S28-S36, 1991.
18. Beca J, Cox PN, Taylor MJ, et al.: Somatosensory evoked potentials for prediction of outcome in severe brain injury. J Pediatr 126:44-49, 1995.
19. Baxt WG, Jones G, Fortlage P: The trauma triage rule: a new resource based approach to the prehospital identification of major trauma victims. Ann Emerg Med 19:1401-1406, 1990.
20. Shackford SR, Wald SL, Ross SE, et al.: The clinical utility of computed tomographic scanning and neurologic examination in the management of patients with minor head injuries. J Trauma 33:385-394, 1992.
21. Borczuk P: Predictors of intracranial injury in patients with mild head trauma. Ann Emerg Med 25:731-736, 1995.
22. Stein SC, Ross SE: Mild head injury: a plea for routine early CT scanning. J Trauma 33:11-13, 1992.
23. Harad FT, Kerstein MD: Inadequacy of bedside clinical indicators in identifying significant intracranial injury in trauma patients. J Trauma 32:359-363, 1992.
24. Schynoll W, Overton D, Krome R, et al.: A prospective study to identify high-yield criteria associated with acute intracranial computed tomography findings in head-injured patients. Am J Emerg Med 11:321-326, 1993.
25. Pal J, Brown R, Fleiszer D: The value of the Glasgow Coma Scale and Injury Severity Score: predicting outcome in multiple trauma patients with head injury. J Trauma 29:746-748, 1989.

ASSESSMENT:

PUPILS

I. Conclusions

A. Data are insufficient to support conclusions on the diagnostic and prognostic value of an examination of the pupils performed in the prehospital environment.

B. Parameter measurement:

1. How should the pupils be examined?
   - Asymmetry is defined as a 1 mm (or more) difference in the size of one pupil.
   - A fixed pupil is defined as no response (< 1 mm) to bright light.
   - Note evidence of orbital trauma.
   - Note left and right distinction and duration of the following:
     - Unilateral or bilateral fixed pupil(s)
     - Unilateral or bilateral dilated pupil(s)
     - Fixed and dilated pupil(s)
2. When should the pupils be examined?
   - After the patient has been resuscitated and stabilized
3. Who should examine the pupils?
   - Trained prehospital care providers

II. Overview

Examining the pupils is a standard component of the neurologic examination that is particularly important in evaluating patients with traumatic brain injury (TBI). The examination consists of assessing pupil size, symmetry, and reactivity to light. The initial pupil examination, along with the Glasgow Coma Scale (GCS) score, establishes the baseline against which all subsequent neurologic evaluations are compared. Abnormalities found in the pupil examination are helpful in generating a differential diagnosis and in directing diagnostic testing and therapeutic interventions.[1] Specifically, an unconscious TBI patient with a unilaterally dilated pupil or with bilaterally fixed and dilated pupils is presumed to have cerebral herniation and requires emergent interventions to lower the intracranial pressure.

Pupillary constriction to light is mediated through the parasympathetic nervous system. A bright light shined into one eye results in pupillary constriction in both the ipsilateral eye (direct response) and contralateral eye (consensual response). This light reflex depends on an intact afferent system that carries light from the retina to the midbrain, and an efferent system

that carries the parasympathetic fibers on the outside of the third cranial nerve from the midbrain to the pupil. Anatomically, the third cranial nerve nuclei in the midbrain are adjacent to the brainstem areas that control consciousness. Thus, assessing pupil response is critical in patients with altered mental status. The third cranial nerve exits the midbrain under the uncus, a portion of the temporal lobe, and is susceptible to compression that may result from edema, intracerebral hemorrhage, and epidural or subdural hematomas. Unilateral third nerve compression compromises the efferent pathway of the pupillary light reflex, thus blocking the direct light response while preserving the consensual response.

III. Search Process

A MEDLINE search was conducted from 1976 to 1998 using the key words "ambulance," "prehospital care," "EMS," "out-of-hospital care," and "pupils" or "eye exam," or "light reflex." No articles were found.

IV. Scientific Foundation

Pupils are generally round and roughly equal in diameter. Inequality in pupil size of less than 1 mm is common and has no pathologic significance.[1] The pupillary light reflex pathways are adjacent to the brain structures that are essential for cognitive function, and to the temporal lobe. Increased intracranial pressure resulting in uncal herniation compresses the third cranial nerve. This reduces parasympathetic tone to the pupillary constrictor fibers that produce a dilated pupil. Destruction of the third nerve parasympathetic brainstem pathway also results in a dilated pupil that is fixed to light. Therefore, the pupillary light reflex is an indirect measure of herniation or brainstem injury. Dilation and fixation of one pupil signifies herniation, whereas bilaterally dilated and fixed pupils are consistent with brainstem injury. However, hypoxemia, hypotension, and hypothermia are also associated with dilated pupil size and abnormal reactivity, making it necessary to resuscitate and stabilize the patient before assessing pupillary function.[2,3] Direct trauma to the third nerve in the absence of significant intracranial injury or herniation can occur causing pupillary abnormalities, although this is usually associated with motor deficits.

No literature exists regarding the ability of prehospital care providers to accurately assess pupils in the field in patients with TBI, nor is there prehospital literature regarding the relationship of pupil findings in the field to patient outcome. Literature does exist regarding the in-hospital evaluation of pupils by physicians, and the relationship of pupil findings to outcome.[4-6] However, most of this literature fails to rigorously measure size and reaction of the pupils. The in-hospital literature generally fails to document the length of time the pupil was fixed and dilated and the actual degree of dilation. Caution must be exercised in extrapolating in-hospital outcomes to the prehospital setting.

In one prospective study of 133 in-patients with severe TBI, bilaterally absent pupillary light reflex was noted in 35% of the cases. A poor outcome occurred in 70% of these patients.[5] In a larger study of 305 in-hospital patients, bilaterally absent pupillary light reflex was associated with 91% mortality.[6] One fixed pupil has been associated with good recovery in up to 54% of patients.[6] A limitation in terms of prognosis is related to conditions that affect pupil size and reactivity other than the result of brainstem or third cranial nerve trauma such as hypoxemia, orbital trauma, drugs, or hypothermia.[2,3] These entities must be considered to avoid the inappropriate management of intracranial pressure.

V. Summary

The pupil examination is an important component of the prehospital evaluation of patients with head trauma. However, the ability of prehospital providers to perform an accurate examination and the diagnostic and management implications of their findings has never been systematically investigated.

Two recommendations for prehospital care can be proposed based on the in-hospital literature:

1. The pupil size and light reflex should be assessed and documented for each eye.
2. The duration of pupillary dilation and fixation should be documented.

VI. Key Questions for Future Investigation

The prehospital environment is significantly different from that of the hospital. Numerous factors, such as lighting, temperature, and movement, impact patient assessment. Prehospital providers are increasingly involved in decision making with regard to therapeutic interventions and transport destination. Given that many of these decisions are based on physical findings, it is particularly important that the prehospital provider be able to accurately perform a physical examination. Significant implications are assigned to pupil size and reactivity in the severe brain-injured patient, making this a fertile area for future investigation. The following key questions need to be addressed:

1. Can prehospital providers accurately assess pupil size and light reactivity in the prehospital environment?
2. Is there acceptable interobserver reliability in the prehospital pupil examination?
3. Are field pupil findings predictive of patient outcome?

VII. References

1. Meyer B: Incidence of anisocoria and difference in size of palpebral fissures in five hundred normal subjects. Arch Neurol Psychiatry 57:464-470, 1947.
2. Meyer S, Gibb T, Jurkovich G: Evaluation and significance of the pupillary light reflex in trauma patients. Ann Emerg Med 22:1052-1057, 1993.
3. Plum F, Posner J: The Diagnosis of Stupor and Coma, 3d Ed. Philadelphia: F.A. Davis, 1982, p. 47.
4. Rivas J, Lobato R, Sarabia R, et al.: Extradural hematoma: Analysis of factors influencing the courses of 161 patients. Neurosurg 23:44-51, 1988.
5. Narayan R, Greenberg R, et al.: Improved confidence of outcome prediction in severe head injury. J Neurosurg 54:751-762, 1981.
6. Braakman R, Gelpke G, Habbema J, et al.: Systemic selection of prognostic features in patients with severe head injury. Neurosurg 6:362-370, 1980.

TREATMENT:

AIRWAY, VENTILATION, AND OXYGENATION

I. Recommendations

A. Standards

Insufficient data about airway, ventilation, and oxygenation in the prehospital setting have been published to support a treatment standard on this topic.

B. Guidelines

1. Hypoxemia (apnea, cyanosis, or arterial hemoglobin oxygen saturation [SaO_2] < 90%) must be avoided, if possible, or corrected immediately. When equipment is available, oxygen saturation should be monitored on all patients with severe traumatic brain injury (TBI) as frequently as possible or continuously. Hypoxemia should be corrected by administering supplemental oxygen.

C. Options

1. The airway should be secured in patients who have severe head injury (Glasgow Coma Scale [GCS] < 9), the inability to maintain an adequate airway, or hypoxemia not corrected by supplemental oxygen. Endotracheal intubation, if available, is the most effective procedure to maintain the airway.
2. Routine prophylactic hyperventilation should be avoided. Hyperventilation in the field is indicated only when signs of cerebral herniation, such as extensor posturing or pupillary abnormalities (asymmetric or unreactive), are present after correcting hypotension or hypoxemia.
3. Normal ventilation is defined as approximately 10 breaths per minute (bpm) for adults, 20 bpm for children, and 30 bpm for infants. Hyperventilation is defined as approximately 20 breaths per minute (bpm) for adults, 30 bpm for children, and 35–40 bpm for infants. (Hyperventilation is discussed in greater detail in the following section on brain-targeted therapy.)

II. Overview

Prospective randomized controlled studies have never been conducted in the prehospital setting on the effects of hypoxemia and hyperventilation and the benefits of endotracheal intubation or other airway management techniques for the patient with head injury. However, case-control studies that include some prehospital data indicate that secondary insults from hypoxemia and hypotension have a significant detrimental effect on the outcome of these patients.[1,2] In addition, hypoxemia and hypotension profoundly affect the ability to accurately obtain the GCS score.[3]

Hyperventilation has been recommended as the primary field treatment for patients with TBI because it is easy to perform and has a rapid effect. Hyperventilation produces a rapid decrease in the arterial partial pressure of carbon dioxide ($PaCO_2$), which causes vasoconstriction, decreased cerebral blood flow (CBF), and lower intracranial pressure (ICP).

Recent studies raise questions about the value of routine hyperventilation. After TBI, CBF is reduced by as much as two-thirds of normal. Hyperventilation can further decrease the CBF, potentially to the point of cerebral ischemia. Although field studies do not exist, evidence from in-hospital studies and data clearly indicates that prophylactic early hyperventilation can seriously compromise cerebral perfusion and patient outcome.[4,5] Therefore, routine hyperventilation of patients with TBI is no longer recommended.

III. Search Process

A MEDLINE search was conducted from 1970 to 1998 using the key words "head injury" and "emergency medical services," or "field" or "prehospital" and "airway management," "intubation," "oxygenation," "hyperventilation," or "hypoxia" as well as "head injury" and "intubation" and "lidocaine." This search resulted in a list of 163 references. Of these, 47 articles that appeared relevant to the prehospital setting were individually reviewed for design and content. All studies reviewed were Class II and III studies. The most informative of these references are discussed and summarized here.

IV. Scientific Foundation

The detrimental effect of hypoxemia on the outcome of patients with TBI was demonstrated in several studies.[1,2] The largest study, involving 717 patients admitted to four centers, showed that hypoxemia (defined as an apneic or cyanotic episode in patients in the field, and as $PaO_2 < 60$ mm Hg on arterial blood gas in patients in the Emergency Department [ED]) has a detrimental effect on patient outcome, particularly when associated with hypotension.[1] Mortality was 26.9% if neither hypoxemia nor hypotension occurred, 28% for hypoxemia alone ($p = 0.013$), and 57.2% if both were noted.

A study from Italy of 50 patients with head trauma who were transported by helicopter revealed that 55% had oxygen saturation less than 90% measured at the scene prior to intubation.[6] That study indicated that both hypoxemia and hypotension had a negative impact on outcome. Of the 28 patients who were hypoxemic, 13 had no associated hypotension (Table A). There was a significant ($p < 0.005$) association between arterial desaturation and poor outcome.

The incidence of hypoxemia and hypercarbia was reported in several studies. A recent retrospective chart review of 72 pediatric patients admitted to a single center with a GCS score between 6 and 8 reported that 13% had a documented hypoxemic episode and 6% had hypercarbia.[2] However, this review did not demonstrate a relationship between these clinical findings and outcome. A small study from England evaluating pulse oximetry in trauma patients in the field and in a moving ambulance documented that 16% had an oxygen saturation < 75%, and an additional 28% had an oxygen saturation between 75% and 90%.[7] The significant cause of low oxygen saturation was severe TBI. Another study from Ireland that prospectively audited early treatment of 131 patients with severe head injury reported that 27% were hypoxemic on arrival at the nearest emergency department.[8]

No studies have evaluated in the field, active, airway management or endotracheal intubation in patients with severe TBI. One retrospective case-control study[9] of 1092 patients with severe head injury (defined as a scene GCS < 9 and a head or neck [H/N] Abbreviated Injury Score [AIS] 4) c ompared patients who underwent prehospital endotracheal intubation with those who did not. Paramedics were permitted to intubate only if patients were apneic, unconscious with ineffective ventilation, and had no gag reflex. The study protocol required that no pharmacologic agents be used for intubation and a maximum of three intubation attempts were permitted. Patients were characterized as having isolated head injury if they had no AIS score greater than 3. The findings are summarized in Table B. A summary of findings appears in Table B. In that study, prehospital endotracheal intubation was associated with significantly improved survival. The study did not compare the incidence of secondary insult (hypoxemia) between the two groups, and because hypoxemia may influence patient outcome, the value of the study is limited.

In another study of prehospital patients, Hsiao correlated the GCS score to the need for intubation in the field or within 30 minutes of ED arrival, and correlated the need for intubation to CT scan findings.[10] This retrospective evaluation included patients with a GCS score equal to or less than 13 as measured in the ED, as well as patients who were intubated in the field and had a GCS scored by the field medical providers. Of note, the lowest reported field GCS score was used for this study. GCS scores grouped patients as follows: 3 to 5, 6 to 7, 8 to 9, 10 to 13. Patients with the lowest GCS scores had the highest need for emergent intubation (in the field or ED) and had the highest number of positive CT scans (as displayed in Table C). Hsiao concluded that patients with a GCS score less than or equal to 9 are candidates for aggressive airway management, including intubation and use of pharmacologic agents, if needed.

An important factor in the field management of all trauma patients is that a large percentage of prehospital care provided in the United States is performed by EMT-Basics. Efforts have been made to add endotracheal intubation to basic EMT training. However, several recent studies report disappointing results as to the ability of EMTs with a basic level of training to successfully intubate patients.[11, 12] Two studies report successful intubation rates of 49% and 51%, respectively. Despite the low success rates reported, intubation remains a skill to be encouraged, taught, and maintained.

Several studies indicate that neuromuscular blockade, a procedure used mostly in helicopter ambulance programs, facilitates endotracheal intubation in the field, and is a feasible and successful technique for a wide variety of medical and trauma patients.[13-16] However, ground ambulance services have not widely implemented this technique because of concerns about failure to intubate and potential complications.[16] In addition, using neuromuscular blocking agents and sedatives to control the patient complicates the ability to score the GCS after arrival at the hospital until these drugs have been metabolized or reversed.[3]

No prehospital studies have evaluated the role of prehospital hyperventilation and the outcome of patients with head injury who were hyperventilated in the field. However, several in-hospital studies demonstrate the detrimental effect of long-term prophylactic hyperventilation on CBF and outcome, particularly when used prophylactically.[4, 5]

V. Summary

Class III evidence indicates that all severe TBI patients treated in the field should receive supplemental oxygen to maximize arterial oxygen saturation. If pulse oximetry is available, the oxygen saturation should be maintained at a minimum of 90% or greater. A patent airway should be ensured and endotracheal intubation performed for patients with a GCS score less than 9 or those who are unable to maintain or protect their airway. The evidence indicates that routine hyperventilation should not be performed. If ventilatory assistance after endotracheal intubation is provided, a respiratory rate of 10 breaths per minute (bpm) for adults, 15–20 bpm for children, and 20 bpm for infants should be maintained. After correction of hypoxemia or hypotension, if the patient shows obvious signs of cerebral herniation, such as extensor posturing or pupillary asymmetry or nonreactivity, the health care provider should hyperventilate the patient at a rate of 20 bpm for adults, 30 bpm for children, and 35–40 bpm for infants. This hyperventilation may be performed as a temporizing measure until the patient arrives at the hospital when blood gas analysis will guide the ventilation rate.

VI. Key Issues for Future Investigation

Some areas for future investigation include the following:

1. How do other airway management devices available for field use by EMTs with basic training compare with each other and with endotracheal intubation? In particular, studies could focus on the ability to maintain adequate oxygen saturation and how that affects outcome of patients with TBI.
2. What is the effect on patient outcome of early short-term hyperventilation after TBI, beginning in the prehospital setting? These studies need to consider extremely variable prehospital times, for example, the short prehospital time in urban areas that permit only a brief period of hyperventilation. In addition, objective measures of the degree and effectiveness of hyperventilation in the prehospital setting need to be developed.

VII. Evidence Tables

Chesnut,[1] 1993

Description of Study: A prospective study of 717 consecutive severe head injury patients admitted to four centers investigated the effect on outcome of hypotension (SBP < 90 mm Hg) occurring from injury through resuscitation.

Classification: II

Conclusions: Hypotension was a statistically independent predictor of outcome. A single episode of hypotension during this period doubled mortality and also increased morbidity. Patients whose hypotension was not corrected in the field had a worse outcome than those whose hypotension was corrected by time of emergency department arrival.

VII. Evidence Tables (continued)

Cooke,[8] 1995

Description of Study: A prospective audit of 131 patients with severe head injury that evaluated the early management of these patients in Northern Ireland.

Classification: III

Conclusions: Twenty-seven percent of patients were hypoxemic on arrival to the ED.

Hsiao,[10] 1993

Description of Study: A retrospective trauma registry–based study of 120 patients with a GCS < 14, which evaluated the need for emergency intubation in the field or ED and evaluated CT scan findings.

Classification: III

Conclusions: The patients in GCS group 3-5 were all intubated and 73% had abnormal CT scans; 73% of patients with GCS 6-7 were intubated and 36% had CT scan abnormalities; 62% of patients with GCS 8-9 were intubated and 62% had CT scan abnormalities; 20% of patients with GCS 10-13 required intubation and 23% had abnormal CT scans.

Kokoska,[2] 1998

Description of Study: A retrospective chart review of 72 pediatric patients admitted to a single center with GCS 6-8, which primarily evaluated morbidity from hypotension, brief mention of hypoxemia.

Classification: III

Conclusions: Thirteen percent of patients had a documented hypoxemic episode and 6% had hypercarbia; the exact location (prehospital, ED, OR, PICU) of these episodes was not given. Neither hypoxemia nor hypercarbia could be related to outcome.

Silverston,[7] 1989

Description of Study: A study of 25 consecutive trauma patients, including head injury; evaluated the use of noninvasive pulse oximetry in the field and in a moving ambulance.

Classification: III

Conclusions: Sixteen percent of patients had SaO_2 < 75%, and an additional 28% were between 75% and 90%. There were no demonstrated difficulties using the pulse oximeter in the field or ambulance.

Stocchetti,[6] 1996

Description of Study: A cohort study of 50 trauma patients transported from the scene by helicopter, which evaluated the incidence and effect of hypoxemia and hypotension and effect on outcome.

Classification: II

Conclusions: Fifty-five percent of patients were hypoxemic (SaO_2 < 90%) and 24% had hypotension. Both hypoxemia and hypotension negatively affected outcome; however, the degree to which each independently affected the outcome was not studied.

Winchell,[9] 1997

Description of Study: A retrospective case-control study of patients with severe head injury with field GCS < 9 and head or neck AIS > 4. This study compared those patients who underwent prehospital endotracheal intubation with those who did not.

Classification: II

Conclusions: Prehospital endotracheal intubation was associated with a statistically significant improved survival.

Table A.
Oxygen saturation prior to intubation vs mortality and severe disability outcomes in Italian TBI patients transported by helicopter (Stocchetti)[6].

Oxygen Saturation	Mortality	Severe Disability
> 90%	14.3% (3/21)	4.8% (1/21)
60-90%	27.3 (6/22)	27.3 (6/22)
< 60%	50 (3/6)	50 (3/6)

Table B.
Prehospital endotracheal intubation and outcome in severe head injury patients (Winchell)[9].

	Intubated	Not Intubated
All Patients - Mortality	26%	36.2%
Isolated TBI - Mortality	22.8	49.6

Table C.
Field GCS score and the need for prehospital endotracheal intubation in TBI patients (Hsiao)[10].

	GCS Score			
	3-5	6-7	8-9	10-13
Field intubation	27%	27%	8%	2%
ED intubation	73	45	53	18
CT scan positive	73	36	62	23

VIII. References

1. Chesnut RM, Marshall LF, Klauber MR, et al.: The role of secondary brain injury in determining outcome from severe head injury. J Trauma 34:216-222, 1993.
2. Kokoska ER, Smith GS, Pittman T, et al.: Early hypotension worsens neurological outcome in pediatric patients with moderately severe head trauma. J Pediatr Surg 33:333-338, 1998.
3. Marion DW, Carlier PM: Problems with initial Glasgow Coma Scale score assessment caused by the prehospital treatment of head-injured patients: results of a national survey. J Trauma 36:89-95, 1994.
4. Muizelaar JP, Marmarou A, Ward JD, et al.: Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. J Neurosurg 75:731-739, 1991.
5. Sheinberg M, Kanter MJ, Robertson CS, et al.: Continuous monitoring of jugular venous oxygen saturation in head-injured patients. J Neurosurg 76:212-217, 1992.
6. Stocchetti N, Furlan A, Volta F: Hypoxemia and arterial hypotension at the accident scene in head injury. J Trauma 40:764-767, 1996.
7. Silverston P: Pulse oximetry at the roadside: a study of pulse oximetry in immediate care. Br Med J 298:711-713, 1989.
8. Cooke RS, McNicholl BP, Byrnes DP: Early management of severe head injury in Northern Ireland. Injury 26:395-397, 1995.
9. Winchell RJ, Hoyt DB: Endotracheal intubation in the field improves survival in patients with severe head injury. Arch Surg 132:592-597, 1997.
10. Hsiao AK, Michelson SP, Hedges JR: Emergency intubation and CT scan pathology of blunt trauma patients with Glasgow Coma Scale scores of 3-13. Prehosp Disast Med 8:229-236, 1993.
11. Bradley JS, Billows GL, Olinger ML, et al.: Prehospital oral endotracheal intubation by rural basic emergency medical technicians. Ann Emerg Med 32:26-32, 1998.
12. Sayre MR, Sackles JC, Mistler AF, et al.: Field trial of endotracheal intubation by basic EMTs. Ann Emerg Med 31:228-233, 1998.
13. Ma OJ, Atchley RB, Hatley T, et al.: Intubation success rates improve for an air medical program after implementing the use of neuromuscular blocking agents. Am J Emerg Med 16:125-127, 1998.
14. Murphy-Macabobby M, Marshall WJ, Schneider C, et al.: Neuromuscular blockade in aeromedical airway management. Ann Emerg Med 21:664-668, 1992.
15. Rhee KJ, O'Malley RJ: Neuromuscular blockade-assisted oral intubation versus nasotracheal intubation in the prehospital care of injured patients. Ann Emerg Med 23:37-42, 1994.
16. Syverud SA, Borron SW, Storer DL, et al.: Prehospital use of neuromuscular agents in a helicopter ambulance program. Ann Emerg Med 17:236-242, 1998.

TREATMENT:

FLUID RESUSCITATION

I. Recommendations

A. Standards

Data are insufficient to support a treatment standard for fluid resuscitation in the patient with severe traumatic brain injury.

B. Guidelines

Fluid resuscitation in patients with TBI should be administered to avoid hypotension and/or limit hypotension to the shortest duration possible. In the adult trauma literature, hypotension is usually defined as a systolic blood pressure (SBP) ≤ 90 mm Hg. In children, hypotension is usually defined as SBP less than the fifth percentile for the age. In children with severe TBI, Class II data link blood pressure and outcome. Hypotension can, therefore, be defined as a systolic blood pressure < 65 mm Hg (age 0-1 year), < 75 mm Hg (age 2-5 years), < 80 mm Hg (age 6-12 years), and < 90 mm Hg (age 13-16 years) in pediatric severe TBI patients.[1]

C. Options

Based on Class III evidence, fluid therapy is utilized to support cardiovascular performance in an effort to maintain adequate cerebral perfusion pressure and limit secondary brain injury. The most commonly used resuscitation fluid for trauma patients in the prehospital setting is isotonic crystalloid solution. It is administered in quantities necessary to support blood pressure in the normal range, although there are little data to support a specific target blood pressure. Inadequate fluid volumes or under-resuscitation can precipitate sudden hypotension and should be avoided. Hypertonic resuscitation, generally utilizing hypertonic saline with or without dextran, has been used with some encouraging results. No studies prove the efficacy of mannitol in the prehospital setting.

II. Overview

The principal issue concerning prehospital fluid resuscitation in patients with TBI centers around preventing and/or rapidly treating shock. Hypotension produces a significant secondary brain injury that substantially worsens outcome. Even so, the exact parameters for fluid resuscitation remain unclear for several reasons, including: 1) The number of patients presenting to hospitals in shock is relatively low. Even in high-volume trauma centers, only approximately 5% of patients present in severe hemorrhagic shock. 2) Prehospital patient care is

perhaps the least controllable care. 3) Data collection is often necessarily relegated to the least trained person. 4) Accurate measurement of vital signs may be problematic at the scene or in transport. 5) Trauma patients often present to the hospital during off-hours, at night, or on weekends, when the fewest resources are available for research. Thus, the quality of the data collected on TBI patients is suspect, and the ability to collect accurate data is poor. Thus, it is not surprising that we have been unable to clearly delineate parameters for field administration of fluid.

Preventing shock and/or promptly treating hypotension are important components of TBI patient care.[2-5] A single episode of hypotension has been shown to double mortality.[5] Perhaps even more important is maintaining cerebral perfusion pressure (CPP) and delivering oxygen to the injured brain. In most Emergency Medical Services (EMS) systems, CPP (CPP = MAP – ICP) is not calculated because mean arterial pressure (MAP) and intracranial pressure (ICP) are not measured in the prehospital setting.

In general, vital signs, such as heart rate and blood pressure, are used as indirect measures of oxygen delivery in the prehospital phase as well as during the initial Emergency Department (ED) evaluation.[6] While these are crude measurements that often do not correlate well with blood loss, there is no other readily available means of accurately quantifying blood loss. Clearly, there is a continuum of response to blood loss. Autoregulation often fails following head injury, placing the brain at increased risk from decreases in preload. As cardiac output falls, so does oxygen delivery. Ideally, resuscitative interventions should begin early enough to prevent a subsequent drop in blood pressure. However, identification of the TBI patient with significant blood loss can be difficult until hypotension is evident. Unfortunately, hypotension is not clearly defined. For example, while a systolic blood pressure of 90 mm Hg is most often used to define hypotension in adults, some of the studies we analyzed for this guideline used 80 mm Hg and others 100 mm Hg.[2, 5, 7]

Crystalloid fluid is utilized to augment cardiac preload, maintaining cardiac output, blood pressure, and peripheral oxygen delivery. General recommendations involve the rapid infusion of 2 liters of isotonic fluid, generally Ringer's lactate or normal saline, as the initial fluid bolus in adults.[6] Because under-resuscitation can lead to hypotension, EMS providers should be careful to avoid it. In patients without head injury, however, there is the concern that resuscitation without surgical hemostasis will lead to secondary blood loss by displacement of potentially hemostatic clots. In addition, crystalloid resuscitation can lead to hemodilution. In one randomized series, patients with penetrating torso trauma and hypotension who were treated with fluid before transfer to the operating room had increased mortality. This issue was raised in an animal model of head injury and uncontrolled hemorrhagic shock.[9] In these animals, delaying resuscitation until the time of hemostasis increased cerebral oxygen delivery. Animals treated with standard resuscitation techniques showed a trend toward higher ICPs, although the data were not statistically significant.

The goal of prehospital fluid resuscitation is to support oxygen delivery and avoid hypotension, if possible. If hypotension occurs, blood pressure and oxygen delivery should be promptly restored to avoid secondary brain injury. Ideally, this should be done in a way that does not cause secondary blood loss or hemodilution. To date, few studies have scientifically investigated prehospital fluid resuscitation in patients with traumatic head injuries.

III. Search Process

A MEDLINE search was conducted from 1978 to 1999 using the key words "head injury," "field or prehospital," and "fluid resuscitation." The search turned up 150 references, of which the 40 were relevant to fluid therapy for the patient with severe head injury. These were individually reviewed for content. The results were collated, and the analysis is presented here.

IV. Scientific Foundation

Crystalloid resuscitation is the therapy most often used in the prehospital treatment of patients with TBI. While little scientific evidence regarding the use of crystalloids has been published, the Advanced Trauma Life Support[6] course, as well as most textbooks, advises judicious use of fluids, also termed "keeping the patient dry." The concern is that any fluid may worsen cerebral edema and increase ICP, thereby ultimately worsening the outcome. This issue has not been examined in the prehospital environment. However, Scalea demonstrated in a study of critical care units that the amount of fluid or blood infused and ICP are not related.[10] In addition, they demonstrated no relationship between central venous pressure, pulmonary capillary wedge pressure, and ICP. The only statistically significant relationship was between ICP and serum lactate. Lactate is an accurate measurement of the adequacy of resuscitation.[11] As lactate rose, so did ICP.

The use of hypertonic saline has been investigated in many clinical situations. Hartl demonstrated that hypertonic saline reliably reduces ICP in patients with TBI and intracranial hypertension.[12] No direct study compares hypertonic saline and standard crystalloid in the prehospital resuscitation of patients with TBI. However, several randomized, prospective trials investigated hypertonic saline in groups of patients with hypotension (Table A). In a multicenter trial, Mattox demonstrated a higher systolic blood pressure in patients treated with hypertonic saline versus crystalloid resuscitation.[13] Survival was significantly statistically better in patients who required surgery, and the hypertonic saline group had fewer complications compared with the group receiving the standard isotonic crystalloid treatment. That trial did not mention head injuries specifically. Wade identified all the published controlled studies of hypertonic saline/dextran, then abstracted the data on patients who had TBI (defined by an abbreviated injury score [AIS] for the head of 4 or greater) and performed a meta-analysis.[14] Hypotension was defined as a systolic blood pressure <90 mm Hg. Survival to discharge was 37.9% for patients treated with hypertonic saline and 26.9% for standard therapy. These findings failed to reach statistical significance (p=0.08). When logistic regression analysis was performed, the odds ratio was 1.92 for 24-hour survival and 2.12 for survival to discharge when hypertonic saline was compared with standard therapy. Thus, the authors of that study concluded that patients who have TBI and receive hypertonic saline dextran are about twice as likely to survive as those who receive standard therapy. This was a statistically significant difference (p=0.048).

Vassar and her colleagues published four prospective, randomized, double-blind trials between 1990 and 1994 concerning the use of hypertonic saline.[15-18] In 1990, they compared two groups of head injury patients, one group receiving hypertonic saline, the other receiving normal saline.[15] Twenty-six percent of the head injury patients were found to have intracranial pathology with bleeding. No difference in outcome was found between the two groups. However, importantly, intracranial bleeding did not increase with either therapy. In 1991, Vassar compared hypertonic saline with Ringer's lactate in 166 patients, 32% of whom had severe TBI

(defined as an AIS of 4 or higher).[16] Crude mortality measurement was the same. When logistic regression analysis was used, hypertonic saline/dextran was associated with a statistically significant higher survival rate than isotonic crystalloid. In 1993, Vassar, et al. published a trial of hypertonic saline versus hypertonic saline dextran in 258 patients.[18] Only 10% had severe TBI. However, in patients with a Glasgow Coma Scale (GCS) score lower than 8 and in patients with severe anatomic cerebral damage, survival with either agent was statistically significantly greater than what would be predicted with the Trauma Related Injury Severity Score (TRISS). The addition of dextran did little to improve survival. In 1993, Vassar, et al. also published a multicenter trial of 194 patients of whom 74% had severe TBI.[17] There was no statistically significant increase in the survival in the overall patient population with the use of hypertonic saline. However, the survival rate in the hypertonic saline group was higher than in the Ringer's lactate group for patients with an initial GCS score of 8.

Pentastarch, another hyperosmolar solution, was tested in 1992 by Younes in a Phase 2 clinical trial of 23 hemorrhage patients.[19] Although that study did not state the number of patients with severe TBI, some of them almost certainly had head injuries because the average GCS score was 11 ± 5. Both Pentastarch and saline increased blood pressure equally, although the volume requirements with Pentastarch were less. No differences were found in complication rates in the two patient groups.

Mannitol is another therapy that has been proven to reduce ICP in hospital patients with intracranial hypertension. Animal data suggest that hypertonic saline and mannitol have similar properties, and that hypertonic saline is equally effective in treating elevated ICP when compared with mannitol.[20] One concern is that mannitol may produce hypotension from volume deficits secondary to its osmotic diuresis. This could potentially produce secondary brain injury. Israel examined the issue of volume depletion in animals with shock and intracranial hypertension.[7] Animals resuscitated with mannitol had better cerebral perfusion and cardiac performance, as well as lower ICPs, than did animals resuscitated with saline. One prospective, randomized, double-blind, controlled trial investigated the prehospital administration of mannitol in head-injured patients, comparing mannitol with standard crystalloid resuscitation.[21] The demographics in that study did not differ, nor did the overall head injury severity between the two groups. Mortality was the same in both groups. Importantly, systolic blood pressure did not change significantly in the mannitol group at the time of ED presentation. However, two hours after hospital arrival, systolic blood pressure was statistically significantly lower in the mannitol group when compared with the placebo group. When all time periods were compared, no differences were found. Very few of these patients were hypotensive. Thus, no statement could be made about the use of mannitol with hypotension.

V. Summary

The deleterious effects of hypotension in patients with TBI have been clearly documented in the literature. In fact, even a brief period of hypotension is associated with increased morbidity and mortality.[5] Because the underlying cause of hypotension in these patients is almost always secondary to bleeding or other fluid losses, intravascular volume seems to be the most efficacious way of restoring blood pressure. In contrast, data indicate that early restoration of blood pressure in patients with penetrating torso trauma worsens outcome. The relationship between this data and outcome in patients with TBI is unknown. Isotonic crystalloid solution is

the fluid most often used in the prehospital resuscitation of head injury patients. However, little data have been published to support its use. Some data indicate that hypertonic saline offers distinct survival advantages in patients with TBI. In the prehospital period, mannitol is probably not deleterious, although only one study was done on this subject, and it included only 41 patients.[21] That work is too preliminary to be conclusive.

VI. Key Areas for Future Investigation

The area of fluid resuscitation has been underinvestigated, consequently, every aspect needs investigation. There are little data to guide endpoints of therapy. One target blood pressure may be better than another, and MAP may be a better guide to therapy than systolic pressure, but these questions require investigation. In addition, the current concern that raising blood pressure may increase secondary blood loss, thus worsening cerebral hemodynamics, needs to be validated in humans. Finally, more work must be done to elucidate the most effective fluid for resuscitation.

The following specific questions need should be studied in the future:

1. What is the optimal target blood pressure for resuscitation in both isolated TBI and the patient with multiple injuries?
2. Is mean arterial blood pressure a better endpoint than systolic blood pressure?
3. Is there a subgroup of patients in whom a lower volume of resuscitation fluid should be used?
4. What is the ideal resuscitation fluid in the prehospital setting?
5. Is there a role for large particle colloids in the prehospital setting?

VII. Evidence Tables

Wade,[14] 1997

Description of Study: Cohort analysis of individual patient data from previously published prospective, randomized, double-blind trials of hypertonic saline/dextran in patients with TBI and hypotension. TBI was defined as AIS for the head of 4 or greater. Hypotension was defined as a systolic blood pressure of 90 mm Hg or less. 1,395 data records were analyzed from six separate studies. 233 patients were then included in this review. 80 patients were treated in the ED and 143 were treated in the prehospital phase.

Classification: I

Conclusions: There was no statistically significant difference in overall survival when hypertonic saline was compared with normal saline. Logistic regression analysis was performed on patients with TBI showing an odds ratio of 1.92 for 24-hour survival and 2.12 for survival until discharge. Thus, patients with TBI in the presence of hypotension who received hypertonic saline/dextran were approximately twice as likely to survive as those who received saline. This was statistically significant with $p = 0.048$.

Chestnut,[5] 1993

Description of Study: A prospective study of 717 consecutive severe head injury patients admitted to four centers investigated the effect on outcome of hypotension (SBP < 90 mm Hg) occurring from injury through resuscitation.

Classification: II

Conclusions: Hypotension was a statistically independent predictor of outcome. A single episode of hypotension during this period doubled mortality and also increased morbidity. Patients whose hypotension was not corrected in the field had a worse outcome than those whose hypotension was corrected by time of ED arrival.

Vassar,[17] 1993

Description of Study: A prospective, randomized, double-blind, multicenter trial comparing the efficacy of administering 250 ml of hypertonic saline vs normal saline as the initial resuscitation fluid in 194 hypotensive trauma patients over a 15-month period. 144 of these patients (74%) had a severe brain injury (defined as an abbreviated injury score AIS for the head of 4, 5, or 6). Here, hypertonic saline significantly increased blood pressure and decreased overall fluid requirements. Post-hoc analysis of the severe head injury group (Class II analysis) revealed that the hypertonic saline group had a statistically significant improvement in survival to discharge. However, the improvement in overall survival was not statistically significant.

Classification: II

Conclusions: Raising the blood pressure in the hypotensive, severe head injury patient improves outcome in proportion to the efficacy of the resuscitation. Prehospital administration of 7.5% sodium chloride to hypotensive trauma patients was associated with a significant increase in blood pressure compared with infusion of Ringer's lactate (RL) solution. The survivors in the RL and hypertonic saline (HS) groups had significantly higher blood pressures than the nonsurvivors. There was no significant increase in the overall survival of patients with severe brain injuries, however, the survival rate in the HS group was higher than that in the LR group for the cohort with baseline GCS scores 5 of 8 or less.

Vassar,[15] 1990

Description of Study: A prospective, randomized, double-blind, clinical trial of 106 patients over an 8-month period. Intracranial hemorrhage was present in 28 patients (26%) .

Classification: II

Conclusions: No adverse effects of rapid infusion of 7.5% NaCl or 7.5% NaCl/6% dextran 70 were noted. Nor were any beneficial effects noted. There was no evidence of potentiating intracranial bleeding. There were no cases of central pontine myelinolysis; however, patients with severe preexisting disease were excluded from the study.

VII. Evidence Tables (continued)

Sayre,[21] 1996

Description of Study: A prospective, randomized, double-blind, placebo-controlled clinical trial of 41 patients over a one-year period at a university-based Level I trauma center. All patients were endotracheally intubated head trauma victims with GCS < 12 and evaluated within 6 hours of injury.

Classification: II

Conclusions: Out-of-hospital administration of mannitol did not significantly change systolic blood pressure in this group of head-injured, multiple trauma patients. There are an insufficient number of patients in this pilot study to assess whether out-of-hospital administration of mannitol to head-injured patients is beneficial overall.

Vassar,[16] 1991

Description of Study: Prospective, randomized, double-blind, multicenter clinical trial of 166 hypotensive patients over a 44-month period. Fifty-three of these patients (32%) had a severe head injury (defined as an AIS for the head of 4, 5, or 6)

Classification: II

Conclusions: Survival was not significantly different in the total patient group. The rate of survival to hospital discharge in patients with severe head injuries was significantly higher in those patients who received hypertonic saline/dextran (HSD) (32% of patients with HSD vs 16% in patients with LR) when using logistic regression analysis. Patients with severe head injury could benefit from HSD administration both because the solution can reduce brain swelling and because, by increasing cardiac output, it can increase O_2 supply to injured cerebral parenchyma.

Vassar,[18] 1993

Description of Study: Prospective, randomized, double-blind, controlled clinical trial of 258 hypotensive patients over 31 months at a university-based trauma center. Twenty-seven of these patients (10%) had a severe head injury (defined as an abbreviated injury score for the head of 4, 5, or 6 only for anatomic lesions).

Classification: I

Conclusions: The administration of 7.5% NaCl (HS) and 7.5% NaCl/6% dextran 70 (HSD) caused no neurologic abnormalities. On the contrary, their use was associated with improvement in survival (as compared with predicted survival) in the patients with low initial GCS score (< 8) and in patients with anatomic confirmation of severe cerebral damage. It appeared that the dextran added little to improvement in survival when compared with hypertonic saline alone. Hypertonic saline solutions did increase the blood pressure response in all patients.

Younes,[19] 1992

Description of Study: Prospective, randomized, Phase II clinical trial of 23 patients. The number of patients with severe head injury was not indicated. Average GCS score was 11 ± 5.

Classification: III

Conclusions: Following initial treatment with isotonic saline or Pentastarch (PS) the MAP increased at a similar rate, although the volumes necessary for hemodynamic recovery (MAP > 100 mm Hg) was significantly greater than those given isotonic saline. By the end of the treatment period, MAP increased to the same level, although the rate of increase in the PS group was higher. The patients who received Pentastarch had a survival rate similar to that of the patients treated with isotonic saline solution. The authors hypothesize that Pentastarch is more efficient, volume for volume, than isotonic saline. There were no differences in the complication rates of the two groups.

Table A. Summary of resuscitation fluid outcome data.

Study	Total Patient (n)	Severe Head Injury GCS ≤8 (%)	Severe Head Injury AIS brain ≥4 (%)	Definition of Hypotension	Hypotension (%)	Severe Head Injury and Hypotension (%)	Total Cohort Mortality (%)	Glasgow Outcome Score	Isotonic Fluid (#)	Hypertonic Saline (#)	Hypertonic Saline/ Dextran (#)
Wade,[14] 1997	223	72%	100%	SBP <90 mm Hg	100%	100%	68%	Good Disabled Vegetative	N/A		
Vassar,[15] 1990	106	N/A	N/A	SBP <90 mm Hg SBP <80 mm Hg	100	N/A	49	Good Disabled Vegetative	N/A		
Vassar,[16] 1991	166	N/A	32	SBP <100 mm Hg	100	32	39	Good Disabled Vegetative	N/A		
Vassar,[18] 1993	258	22	10	SBP <90 mm Hg	100	22	18	Good Disabled Vegetative	1 2	12 2	10 12 1
Vassar,[17] 1993	194	57	74	SBP ≤90 mm Hg	100	74	48	Good Disabled Vegetative	1	1 1	1 2 1
Sayre,[21] 1996	41	All GCS < 12	N/A	SBP ≤90 mm Hg	22	N/A	20	Good Disabled Vegetative	N/A		

TABLE B. (continued) **Summary of resuscitation fluid data.**

Study	Total Patient (n)	GCS ≤8 AIS ≥4	% of Total	Hypotensive	% of Total	% in Severe TBI & Hypotension	Overall Mortality	Mortality by Treatment: Isotonic Fluid	Mortality by Treatment: Hypertonic Saline	Mortality by Treatment: Hypertonic Saline Dextran	Glasgow Outcome Scale in GCS ≤ 8 in Survivors: Isotonic Fluid	Glasgow Outcome Scale in GCS ≤ 8 in Survivors: Hypertonic Saline	Glasgow Outcome Scale in GCS ≤ 8 in Survivors: Hypertonic Saline & Dextran
Wade,[14] 1997	223	167 223	72% 100	223 < 90 mm Hg	100%	100%	68%	#15 (17%)	14%	22%	1 Good	1 Good 1 Moderate	1 Good 2 Moderate 1 Vegetative
Vassar,[15] 1990	106	N/A	N/A	59 ≤ 90 mm Hg 47 ≤ 80 mm Hg	100	N/A	49	#14 (49%)	40%	50%	1 Good 2 Moderate	12 Good 2 Moderate	10 Good 12 Moderate 1 Vegetative
Vassar,[16] 1991	166	N/A 53	N/A 32	166 < 100mm Hg	100	N/A 3	39	#13 (41%)	N/A	36%	N/A		
Vassar,[18] 1993	258	57 27	22 10	258 < 90 mm Hg	100	22 10	18	#9 (14%)			Mannitol 25%		
Vassar,[17] 1993	194	110 144	57 74	194 < 90 mm Hg	100	57 74	48	#12 (45%)			N/A		
Sayre,[21] 1996	41	All GCS < 12	100	9 < 90 mm Hg	22	N/A	20	#16 (73%)			N/A		

VIIa. Description of the Studies

First Author	Number of Points	Prospective	Time	When Indicator was Measured	What Method
Chesnut,[1] 1993	717	Y	1984-1987	Injury through resusc†	dir ms; exam*
Fearnside,[2] 1993	315	Y	NR	Injury through resusc†	dir ms
Kokoska,[7] 1998	72	Y	1990-1995	Prehospital	dir ms
Miller,[9] 1982	225	Y	NR	Admission†	dir ms
Miller,[10] 1978	100	Y	NR	Admission†	dir ms
Pigula,[13] 1993	58	Y	1985-1993	Admission†	dir ms
Stochetti,[16] 1996	50	Y	1992-1994	Injury scene	dir ms
Vassar,[17] 1993	258	Y	1988-1991	Injury scene	dir ms

Who Did It	Outcome Measure	When	Blinded Observer?	Multivariate Statistics	Statistical Method
EMS or MD	GOS	6 mos	N	Y	Log reg
NR	GOS	6 mos	NR	Y	Log reg
NR	GOS	3 mos	NR	N	ANOVA
NR	GOS	6 mos	NR	N	NR
NR	GOS	NR	NR	N	Chi Square
NR	Survival	NR	NR	N	Chi Square, ANOVA
Nurse	GOS	6 mos	Y	N	Chi Square
EMS	Dead or alive	Hosp discharge		N	Y Lin/Log Reg Cox Prop Haz

*Hypoxemia prior to admission defined as apnea or cyanosis.
† Hypoxemia also recorded in this report.

VIII. References

1. Kokoska ER, Smith GS, Pittman T, Weber TR: Early hypotension worsens neurological outcome in pediatric patients with moderately severe head trauma. J Pediatr Surg 33:333-338, 1990.
2. Feldman JA, Fish S: Resuscitation fluid for a patient with head trauma and hypovolemic shock. J Emerg Med 9:465-468, 1991.
3. Gruen P, Liu C: Current trends in the management of head injury. Emerg Med Cl NA16: 63-83, 1998.
4. Silvestri S, Aronson S: Severe head injury: prehospital and emergency department management. Mt Sinai J Med 64:329-338, 1997.
5. Chestnut RM, Marshall LF, Klauber MR: The role of secondary brain injury in determining outcome from severe head injury. J Trauma 34:216-222, 1993.
6. American College of Surgeons: Advanced Trauma Life Support Instructor's Manual. Chicago, 1996.

7. Israel RS, Marx JA, Moore EE, Lowenstein SR: Hemodynamic effect of mannitol in a canine model of concomitant increased intracranial pressure and hemorrhagic shock. Ann of Emerg Med 17:560-566, 1998.
8. Bickell WH, Wall Jr MJ, Pepe PE et al.: Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. N Engl J Med331:1105, 1994.
9. Bourguignon PR, Shackford SR, Shiffer C, et al.: Delayed fluid resuscitation of head injury and uncontrolled hemorrhagic shock. Arch Surg 133:390-398, 1998.
10. Scalea TM, Maltz S, Yelon J, et al.: Resuscitation of multiple trauma and head injury: role of crystalloid fluids and inotropes. Crit Care Med22:1610-1615, 1994.
11. Abramson D, Scalea TM, Hitchcock D, et al.: Lactate clearance and survival following injury. J Trauma 35:584-589, 1993.
12. Hartl R, Ghajar J, Hochleuthner H, Mauritz W: Hypertonic/hyperoncotic saline reliably reduces ICP in severely head-injured patients with intracranial hypertension. Acta Neurochirugia 70:126-129, 1997.
13. Mattox KL, Maningas PA, Moore EE, et al.: Prehospital hypertonic saline/dextran infusion for post-traumatic hypotension. Ann Surg 213:482-491, 1991.
14. Wade CE, Grady JJ, Kramer GC, et al.: Individual patient cohort analysis of the efficacy of hypertonic saline/dextran in patients with traumatic brain injury and hypotension. J Trauma 42:561-565, 1997.
15. Vassar MJ, Perry CA, Holcroft JW: Analysis of potential risks associated with 7.5% sodium chloride resuscitation of traumatic shock. Arch Surg 125:1309-1315, 1990.
16. Vassar MJ, Perry CA, Gannaway WL, Holcroft JW: 7.5% sodium chloride/dextran for resuscitation of trauma patients undergoing helicopter transport. Arch Surg 126:1065-1072, 1991.
17. Vassar MJ, Fischer RP, O'Brien PE, et al.: A multicenter trial for resuscitation of injured patients with 7.5% sodium chloride. Arch Surg 128:1003-1011, 1993.
18. Vassar MJ, Perry CA, Holcroft JW: Prehospital resuscitation of hypotensive trauma patients with 7.5% NaCl versus 7.5% NaCl with added dextran: A controlled trial. J Trauma 34:622-632, 1993.
19. Younes RN, Yin KC, Amino CJ, et al.: Use of Pentastarch solution in the treatment of patients with hemorrhagic hypovolemia: randomized phase II study in the emergency room. World J Surg 22:2-5, 1998.
20. Freshman SH, Battistella FD, Matteucci M, Wisher DH: Hypertonic saline versus mannitol: a comparison for treatment of acute head injuries. J Trauma 35:344-348, 1993.
21. Sayre MR, Daily SW, Stern SA, et al.: Out-of-hospital administration of mannitol to head-injured patients does not change systolic blood pressure. Acad Emerg Med 3(9):840-848, 1996.

BRAIN TARGETED THERAPY

I. Conclusions

A. Standards
Class I data are insufficient to support prehospital brain-specific treatment standards, including hyperventilation, mannitol, lidocaine, sedation, analgesics, paralytics, and glucose administration for the patient with traumatic brain injury (TBI).

B. Guidelines
Class II data are insufficient to support the creation of prehospital brain-specific treatment guidelines.

C. Options
Class III data support the following treatment options:

Treatment of cerebral herniation: The signs of cerebral herniation in an unconscious and unresponsive patient include extensor posturing, asymmetric, dilated or unreactive pupils or progressive neurologic deterioration (decrease in the GCS score of more than two points from the patients's prior best score in patients with an initial GCS < 9). Hyperventilation (20 bpm in a adult, 30 bpm in a child, and 35–40 bpm in an infant less than one year old) is the first line of intervention in the patient with suspected impending cerebral herniation. Neurologic status requires frequent reevaluation and, in the subsequent absence of clinical signs of herniation, hyperventilation should not be continued.

The prehospital use of mannitol currently cannot be recommended.

Treatments to optimize patient transport: Sedation, analgesia, and neuromuscular blockade can be useful to optimize transport of the head-injured patient. Because no outcome studies provide guidance on the use of these adjuncts, the timing and choice of agents are best left to local Emergency Medical Services (EMS) protocols.

Treating other causes of altered mental status: Hypoglycemia has been reported as the cause of traumatic events. As with brain injury, hypoglycemia may present with altered mental status with or without focal neurologic deficits. From Class III data, this guideline recommends that patients with altered mental status of undetermined etiology have a rapid glucose determination or be given glucose empirically.

II. Overview

Neuronal injury may result from the initial trauma (primary injury) or as the result of indirect mechanisms (secondary injury), such as hypoxemia, hypotension, and cerebral edema. Injury may also occur as the result of associated conditions that caused the trauma, such as hypoglycemia or drug toxicity. The goal of resuscitation in TBI is to preserve cerebral perfusion and to minimize neuronal injury. As discussed in other sections of these guidelines, hypotension and hypoxemia are associated with poor outcomes in patients with TBI, thus systemic resuscitation is the highest priority in prehospital management.

Management of patients with TBI is directed at maintaining cerebral perfusion. Signs of cerebral herniation include fixed and dilated pupil(s), asymmetric pupils, extensor posturing, or neurologic deterioration (decrease in the GCS score of more than two points from the patient's prior best score in patients with an initial GCS score less than 9). Hyperventilation, as discussed in the previous section, is beneficial in the immediate management of patients demonstrating signs of cerebral herniation, but it is not recommended as a prophylactic measure.[1] Mannitol is effective in reducing intracranial pressure (ICP) and is recommended for control of increased ICP. However, no data support its use in patients without signs of cerebral herniation and without ICP monitoring. A number of pharmacologic agents have been investigated in an attempt to prevent the secondary injury associated with TBI, but none have proven efficacious.[2]

III. Search Process

A MEDLINE search was conducted from 1976 to 1998 using the key words "ambulance," "prehospital," "EMS," "out of hospital," and "head injury," and "mannitol" or "glucose" or "paralytic agents" or "sedation" or "analgesic" or "lidocaine" ("hyperventilation" was searched for another section of these guidelines). One article on glucose,[3] one article on mannitol,[4] and one article on sedation[5] were identified. Only the article on glucose, which was a case series and, therefore, Class III evidence, had clinical relevance to the outcome.

IV. Scientific Foundation

No scientific literature is available on the efficacy of brain-specific treatment in managing patients in the prehospital setting. Some conclusions can be drawn by extrapolation of the in-hospital literature; however, caution must be exercised when comparing treatments in two such different environments.

Treatments for Cerebral Herniation

Hyperventilation

Hyperventilation in the acute setting reduces ICP by causing cerebral vasoconstriction with a subsequent reduction in cerebral blood flow.[6] The long-term benefit of hyperventilation when used in the acute setting has not been determined. The preceding section discussed the growing concern regarding the exacerbation of postinjury cerebral ischemia by vasoconstriction. Consequently, hyperventilation is reserved as a temporizing measure for those patients with severe brain injury who show signs of cerebral herniation, such as extensor posturing, asymmetric or unreactive pupils, or progressive neurologic deterioration (decrease in the GCS score of more than two points from the patient's prior best score in patients with an initial GCS score less than 9).

Mannitol

Although the mechanism is controversial, mannitol's benefit in managing severe TBI is linked to its immediate plasma-expanding properties and to its delayed osmotic/diuretic action. Mannitol has an immediate plasma-expanding effect that reduces the hematocrit and blood viscosity.[7-10] These effects result in increased cerebral blood flow and increased cerebral oxygen delivery, which are fundamental to central nervous system resuscitation. Because mannitol is a plasma expander, it has been recommended as a resuscitation fluid in patients with coexistent shock due to systemic injuries or TBI[9-12]; however, an evidence-based recommendation must await well-designed studies to validate its effectiveness.

Mannitol's osmotic effect creates a fluid gradient out of cells, with consequent diuresis. This osmotic effect initially decreases intracellular edema, thus decreasing ICP. The onset of action is delayed 15 to 30 minutes, but then persists for 90 minutes to 6 hours.[7, 13, 14] Over time, mannitol may accumulate in the brain and result in a "reverse osmotic shift," potentially increasing ICP.[13, 15] To minimize this complication, mannitol when given in the in-hospital setting is dosed in repeated boluses rather than by continuous infusion.[1] A potential complication of mannitol includes acute renal failure, which has been reported in patients taking nephrotoxic drugs and in patients with sepsis or preexisting renal disease.[16, 17]

Concern exists that mannitol's diuretic properties may cause hypotension despite its demonstrated, immediate, plasma-expanding properties. One study investigated the prehospital use of mannitol and its effect on systolic blood pressure.[4] This was a prospective, randomized, double-blind, placebo-controlled pilot study that compared 20 patients receiving mannitol (1 gm/kg) with 21 patients treated with placebo. All patients were intubated, had GCS < 12, and had pulse and systolic blood pressure measured every 15 minutes for 2 hours. The study did not measure outcome, diastolic pressures, or late sequelae. The results indicate that systolic blood pressure did not change significantly in either group.

Lidocaine

Intravenous lidocaine prevents the increases in ICP that occur with endotracheal intubation.[18] No study has demonstrated that transient increases in ICP during airway manipulation have an impact on patient outcome. Regardless, many rapid-sequence protocols recommend that lidocaine, 1.5 mg/kg, be given several minutes before laryngoscopy; these protocols also recommend that other drugs that protect the central nervous system, such as fentanyl or thiopental, be given as part of the intubation protocol.[19-22] No literature could be found to support the use of lidocaine as a single agent prior to intubation.

Treatments to Optimize Patient Transport

Sedation and Analgesia

Sedation and analgesia are key components of comprehensive patient care and are important considerations in prehospital management: This is particularly true when long transport times are involved. The first step in managing the agitated or combative TBI patient is assessing and correcting hypotension, hypoxemia, hypoglycemia, and patient discomfort. Mechanical restraints for the severely agitated patient are generally not recommended and have been associated with placing patients at risk for physical harm.[23] Because patient cooperation is

critical for a safe transport, there are times when pharmacologic interventions, including neuromuscular blockade, are clearly indicated.

Benzodiazepines and phenothiazines are the two categories of drugs commonly used to manage combative patients in the emergency department.[20] No studies have specifically investigated the use of benzodiazepines in the prehospital setting to manage the agitated patient. Rosen, et al. performed a randomized, prospective, double-blind study to evaluate the efficacy of intravenous droperidol to manage the combative patient in the prehospital setting.[24] Twenty-three patients who received 5 mg of intravenous droperidol were compared with 23 patients who received saline. Five patients in the droperidol group and seven in the saline group were diagnosed with TBI in the emergency department. Droperidol was found to be significantly effective within five minutes of administration in sedating the combative patient. Forty-eight percent of the saline group and only 13% of the droperidol group required further sedation on arrival at the emergency department. None of the patients receiving droperidol developed hypotension or seizures; one patient developed restlessness that responded to diphenhydramine. This study is limited by its small sample size, but it supports the use of droperidol in managing combative patients during transport.

Studies have demonstrated the safety of using short-acting neuromuscular blockade in the field to facilitate intubation performed by prehospital care providers.[21-28] No studies could be identified that specifically examined the impact of long-acting neuromuscular blockade or analgesia in the field on outcome from TBI.

Painful stimuli may increase ICP, and the use of sedation, analgesia, and neuromuscular blockade under certain conditions appears intuitively appropriate, despite the lack of outcome studies. These agents are not without risks, and their use may interfere with determining the GCS score.[29] Consequently, each EMS system must carefully weigh and monitor a risk/benefit analysis of the prehospital use of sedation, analgesia, and neuromuscular blockade.

Managing Hypoglycemia

Glucose is the primary fuel for neuronal function. Blood levels less than 80 mg/dl may result in symptoms, although there is no clear correlation of symptoms with levels.[30] Mild hypoglycemia classically presents with diaphoresis, headache, and weakness. However, in one study, these findings were absent in 25% of prehospital patients with altered mental status caused by hypoglycemia.[31] Focal neurologic deficits and seizures can occur.[32,33] Blood glucose levels of 30 mg/dl are associated with confusion and delirium, and levels less than 10 mg/dl usually produce deep coma that may be irreversible.[34]

One case series in the literature reported on four patients with presumed severe TBI where life-threatening hypoglycemia went undiagnosed for a significant period of time.[3] Delay in diagnosis resulted in unnecessary diagnostic testing and interventions. This case series illustrates the difficulty that may occur in obtaining important historical information in patients with head injury who are unresponsive. It also emphasizes the importance of comprehensive management in patients with altered mental status not biased by the trauma setting in which the patient is found.

Controversies regarding prehospital diagnosis and management of hypoglycemia include: 1) the accuracy of glucose reagent test strips in the prehospital setting; 2) the effect of poor peripheral perfusion on the accuracy of glucose reagent test strips; and 3) the potential harm of empiric glucose administration to patients with brain injury.

Lavery, et al. performed a prospective evaluation of glucose reagent test strips in the prehospital setting.[35] They studied 181 patient samples using a test strip reading of 90 mg/dl or less as a measure of hypoglycemia. Test strips were found to be 100% sensitive and 57% specific.

In another prospective study, Sylvain, et al. evaluated the accuracy of finger-stick glucose values in shock patients with poor peripheral perfusion.[36] Significant differences were found between finger-stick measurements and venous blood laboratory glucose values. This introduces important field diagnostic issues in the hypotensive patient with multiple injuries and altered mental status.

Hoffman and Goldfrank performed a critical review of the literature regarding the risks and benefits of empiric dextrose in patients with altered mental status.[37] The literature is controversial, but some evidence exists that patients with ischemic brain injury and hyperglycemia have worse outcomes than their normoglycemic counterparts. The authors recommended field testing of glucose levels rather than empiric dextrose treatment. However, they emphasized the importance of using clinical suspicion in decision making, and they did recommend empiric dextrose in cases where a glucose level was unavailable and the patient had altered mental status without focal deficits. It should be noted that the available literature primarily involves nontraumatic conditions, making extrapolation to patients with TBI difficult.

V. Summary

Preservation of cerebral perfusion and oxygenation are the first priorities in managing patients with TBI. Patients who exhibit signs of cerebral herniation during field management and transport should be treated initially with hyperventilation. The role of mannitol in treating cerebral herniation is yet to be determined, and currently it is not recommended in these guidelines. Sedation, analgesia, and neuromuscular blockade are important considerations during the transport of patients with TBI to minimize changes in intracranial pressure and to maximize safety during transport. Hypoglycemia may mimic TBI and should be considered in all patients with altered mental status regardless of the suspected etiology.

VI. Key Questions for Future Investigation

EMS systems vary significantly; and levels of care available and transport times have a potentially large impact on patient outcome. The prehospital use of brain-specific therapies clearly needs definition and further study. Key questions include the following:

1. What treatment protocols are EMS systems currently using to manage suspected cerebral herniation?
2. How are EMS systems presently managing combative or agitated patients with TBI?
3. Is mannitol safe to administer in the prehospital environment?
4. Does the use of mannitol during transport in patients with cerebral herniation offer any advantage over hyperventilation alone?
5. Is there a role for the prehospital use of mannitol in the "prophylactic" treatment of patients with severe TBI who are not suspected of cerebral herniation?

VII. Evidence Tables

Luber,[3] 1996

Description of Study: Case series of 4 patients with altered mental status due to hypoglycemia. In each case, the patient was initially misdiagnosed and treated as if their altered mental status was due to head trauma.

Classification: III

Conclusions: In patients with altered mental status of undetermined etiology, a serum blood sugar determination should be performed or the patient should be given empiric glucose. Study emphasizes the importance of not presuming that altered mental status is due to a head injury in all trauma patients.

Sayre,[4] 1996

Description of Study: Prospective, randomized, double-blind, placebo-controlled clinical trial of 41 patients. All patients were intubated head trauma patients with GCS score < 12 evaluated within 6 hours of injury. Patients received 1 gm/kg mannitol and had pulse and blood pressure measured every 15 minutes for 2 hours.

Classification: —

Conclusions: This small pilot study did not report on outcome past two hours, or late sequelae; therefore, no conclusions could be drawn regarding the role of mannitol in managing ICP in the field (explaining why this study is not classified despite its scientific study design).

Chillero,[5] 1992

Description of Study: Review article primarily dealing with in-hospital use of sedatives.

Classification: —

Conclusions: No conclusions could be could be drawn regarding prehospital management.

Rosen, [24] 1997

Description of Study: Randomized, prospective, double-blind prehospital study comparing 23 agitated patients treated with IV droperidol to 23 patients treated with saline. Only 5 droperidol patients had a TBI.

Classification: II

Conclusions: Droperidol, 5 mg IV, was found to be safe and effective. The small number of TBI patients downgraded the classification of this study despite the scientific study design.

VIII. References

1. Brain Trauma Foundation: Guidelines for the Management of Severe Head Injury. New York: Brain Trauma Foundation, 1995, section 10.
2. McIntosh T, Garde E, Saatman K, et al.: Central nervous system resuscitation. Emerg Med Clin No Am 15:527-550, 1997.
3. Luber S, Brady W, Brand A, et al.: Acute hypoglycemia masquerading as head trauma: a report of four cases. Am J Emerg Med 14:543-547, 1996.
4. Sayre M, Daily S, Stern S, et al.: Out-of-hospital administration of mannitol to head-injured patients does not change systolic blood pressure. Acad Emerg Med 3:840-848, 1996.
5. Chiolero R: Sedatives and antagonists in the management of severely head-injured patients. [Review] Acta Neurochir Suppl 55:43-46, 1992.
6. Raichle, M, Plum F: Hyperventilation and cerebral blood flow. Stroke 3:566-575, 1972.
7. Barry K, Berman A: Mannitol infusion. Part III. The acute effect of the intravenous infusion of mannitol on blood and plasma volume. N Engl J Med 264:1085-1088, 1961.
8. Brown F, Johns L, Jafer J, et al.: Detailed monitoring of the effects of mannitol following experimental head injury. J Neurosurg 50:423-432, 1979.
9. Israel R, Marx J, Moore E, et al.: Hemodynamic effect of mannitol in a canine model of concomitant increased intracranial pressure and hemorrhagic shock. Ann Emerg Med 17:560-566, 1988.
10. Muizelaar J, Lutz H, Becker D: Effect of mannitol on ICP and CBF and correlation with pressure autoregulation in severely head-injured patients. J Neurosurg 61:700-706, 1984.
11. Freshman S, Battistella F, Matteucci M, et al.: Hypertonic saline (7.5%) versus mannitol: a comparison for treatment of acute head injuries. J Trauma 35:344-348, 1993.
12. Wisnerd H, Busche F, Sturm J: Traumatic shock and head injury: effects of fluid resuscitation on the brain. J Surg Res 46:49-52, 1989.
13. Becker D, Vries J: The alleviation of increased intracranial pressure by the chronic administration of osmotic agents. In Intracranial Pressure, Brock M, Deitz H (eds). New York: Springer, 1972, pp. 309-315.
14. Cruz J, Miner M, Allen S, et al.: Continuous monitoring of cerebral oxygenation in acute brain injury: injection of mannitol during hyperventilation. J Neurosurg73:725-730, 1990.
15. Kaufman A, Cardozo E: Aggravation of vasogenic cerebral edema by multiple dose mannitol. J Neurosurg 77:584-589, 1992.
16. Oken D: Renal and extrarenal considerations in high-dose mannitol therapy. Renal Failure 16:147-159, 1994.
17. Feig P, McCurdy D: The hypertonic state. N Engl J Med 297:1449-1451, 1977.
18. Hamill J, Bedford R, Weaver D, et al.: Lidocaine before endotracheal intubation: intravenous or laryngotracheal. Anesthesiology; 55:578-581, 1981.
19. Walls R: Rapid-sequence intubation in head trauma. Ann Emerg Med; 22:1008-1013, 1993.
20. Walls R, Luten R, Murphy M, Schneider R: Manual of Emergency Airway Management, 3rd ed. Wellesley, MA: Airway Management Education Center, 1999.
21. Sing R, Reilly P, Rotondo M, et al.: Out-of-hospital rapid-sequence induction for intubation of the pediatric patient. Acad Emerg Med 3:41-45, 1996.
22. Syverud S, Borron S, Storer D, et al.: Prehospital use of neuromuscular blocking agents in a helicopter ambulance program. Ann Emerg Med 17:236-242, 1988.

23. Stratton S, Rogers C, Green K: Sudden death in individuals in hobble restraints during paramedic transport. Ann Emerg Med 25:710-712, 1995.
24. Rosen C, Ratliff A, Wolfe R, et al.: The efficacy of intravenous droperidol in the prehospital setting. J Emerg Med 15:13-17, 1997.
25. Hedges J, Dronen S, Feero S, et al.: Succinylcholine-assisted intubations in prehospital care. Ann Emerg Med 17:469-472, 1988.
26. Brownstein D, Shugerman R, Cummings P, et al.: Prehospital endotracheal intubation of children by paramedics. Ann Emerg Med 28:34-39, 1996.
27. Murphy-Macabobby M, Marshall W, et al.: Neuromuscular blockade in aeromedical airway management. Ann Emerg Med 21:664-668, 1992.
28. Rhee K, O'Mally R: Neuromuscular blockade-assisted oral intubation versus nasotracheal intubation in the prehospital care of injured patients. Ann Emerg Med 23:37-42, 1994.
29. Marion D, Carlier P: Problems with initial Glasgow Coma Scale assessment caused by prehospital treatment of patients with head injuries: results of a national survey. J Trauma 36:89-95, 1994.
30. Malouf R, Brust J: Hypoglycemia: causes, neurological manifestations, and outcome. Ann Neurol 17:421-430, 1985.
31. Hoffman J, Schriger D, Votey S, Luo J: The empiric use of hypertonic dextrose in patients with altered mental status: a reappraisal. Ann Emerg Med 21:20-24, 1992.
32. Wallis W, Donaldson I, Scott R Wilson J: Hypoglycemia masquerading as cerebrovascular disease (hypoglycemia hemiplegia). Ann Neurol 18:510-512, 1985.
33. Foster J, Hart R: Hypoglycemia hemiplegia: two cases and a clinical review. Stroke 18:944-946, 1987.
34. Ferrendelli J: Cerebral utilization of nonglucose substrates and their effect in hypoglycemia. In Brain Dysfunction in Metabolic Disorders, Plum F (ed). Res Publ Assoc Nerv Ment Dis 53; 113-130, 1974.
35. Lavery R, Allegra J, Cody R, et al.: A prospective evaluation of glucose reagent test strips in the prehospital setting. Am J Emerg Med 9:304-308, 1991.
36. Sylvain H, Pokorny M, English S, et al.: Accuracy of fingerstick glucose values in shock patients. Am J Crit Care 4:44-48, 1995.
37. Hoffman R, Goldfrank L: The poisoned patient with altered consciousness: controversies in the use of a "coma cocktail." JAMA 274:562-569, 1995.

HOSPITAL TRANSPORT DECISIONS

I. Recommendations

A. Standards
Class I data are insufficient to support a treatment standard for this topic.

B. Guidelines
Class II data support the recommendation that all regions have an organized trauma care system that develops protocols to direct Emergency Medical Services (EMS) personnel regarding transport decisions for trauma victims. Recognizing at the scene or in the ambulance that a patient has sustained severe traumatic brain injury (TBI) guides hospital destination.

Class II data support the recommendation that patients with severe TBI with a Glasgow Coma Scale (GCS) score less than 9, be transported directly to a facility identified as having the following capabilities: immediately available CT scanning, prompt neurosurgical care, and the ability to monitor intracranial pressure and treat intracranial hypertension, as delineated in *Guidelines for the Management of Severe Head Injury.*[1]

C. Options
Class III data support the recommendation that all EMS systems develop transport protocols to help line EMS personnel make specific decisions regarding trauma center destination for head injury patients. Patients with GCS scores of 9 to 13 have potential for intracranial injury and neurosurgical intervention, and should therefore be transported to a trauma center for evaluation.

II. Overview

Injury causes approximately 150,000 deaths in the United States each year, approximately one-third as a result of fatal head injuries.[2] Trauma, including neurotrauma, is a serious public health problem requiring continuing improvement in the prehospital and hospital care of injured patients. Trauma system development and organization and better injury prevention appear to lower the incidence of death and disability because of intentional and unintentional injury and should be available to all people worldwide.

The EMS provider's assessment and recognition of head injury as brain specific and the subsequent response are paramount to the patient's recovery. Prehospital hypoxemia and hypotension are significant contributors to poor outcome in these patients. Appropriate

prehospital emergency medical care can help minimize the impact of secondary injury in patients with severe TBI.

The EMS field transport choice of hospital destination for patients with severe TBI is also one of the most important decisions affecting patient outcome. The commitment of the receiving institution to be prepared to receive these patients and to have the necessary staff and equipment available is paramount to the patient's outcome. Based on general studies of trauma centers and their effectiveness, these centers clearly play a crucial role in the successful outcomes of general trauma patients.

A number of steps and decisions that can impact patient outcome are involved in the prehospital management of patients with severe TBI. These include the following four:

1. Detailed information gathering by EMS call-takers and dispatchers can help guide decisions about the type of personnel dispatched to the scene of a trauma incident. Directed questioning by the call-taker of the person calling for assistance can provide specific information about the potential for significant brain injury. Asking if the patient is awake, able to talk, open the eyes, or move the extremities can help determine the likelihood of brain injury. If the answers indicate potentially serious brain injury, the highest available level of care and EMS provider should be dispatched to the scene.
2. The prehospital evaluation of the mechanism of the injury (e.g., vehicular deformation severity, windshield violation, the use or nonuse of seat belts or other safety devices), the scene, and especially the patient examination are crucial components in assessing the overall neurologic situation. Vital signs, including pulse oximetry when available, will aid in recognizing hypotension and hypoxemia. The GCS score and state of the pupils will provide information about the severity of the brain injury.
3. Based on the patient assessment, prehospital interventions are initiated to prevent or correct hypotension or hypoxemia and to address other potential threats to life or limb. At this step, the decision regarding the level of responder dispatched to the scene impacts on patient care. The higher the level of training of the responder sent to the scene, the greater the likelihood of providing interventions, such as fluid resuscitation and definitive airway management, to correct hypotension and hypoxemia, thus minimizing secondary injury.
4. Finally, the hospital selected by the EMS provider can also have a profound impact on outcome. As mentioned above, an organized trauma system improves outcome in patients with multisystem trauma because the receiving institutions have committed to having the necessary equipment and personnel available to immediately assess and treat the trauma victim. Therefore, all EMS systems should identify the institutions that are appropriate destinations for trauma patients.

A number of factors may affect the EMS system and impact its ability to consistently provide optimal care to each patient. In urban settings, more specific demands on EMS systems exist. For example, short response times, an increase in the number of hospitals, and short transport times suggest that care is quicker and closer for the severe head injury victim. However, urban EMS systems often have a large volume of calls, they struggle with responses and transports through congested streets, and their protocols may not allow them to bypass one institution in favor of another, such as a trauma center.

In rural communities without a nearby trauma center, a field response system should be in place for EMS personnel to use faster means of transport (e.g., helicopter). As mentioned above,

direct transport to a trauma center when possible improves patient outcome. If such a center is not available, transport to the closest facility for initial stabilization is appropriate, with subsequent transfer based on patient need. All EMS providers involved in extended transport times, whether the initial transport to a hospital or the transfer to a referral center, should be trained to perform continuing neurological assessments to evaluate and recognize any change in the patient's condition and neurologic status.

III. Search Process

A MEDLINE search conducted from 1970 to 1999 using the key words "trauma systems," "trauma centers," "emergency medical services," "prehospital care," and "ambulance transports" identified 147 articles. Careful review and analysis of all 147 articles permitted an assessment of trauma systems and the role of EMS in managing patients with severe TBI.

IV. Scientific Foundation

Since the late 1970s, several investigators have tried to demonstrate the efficacy of EMS systems and trauma systems. Studies performed in the late 1970s and early 1980s attempted to show that excessive "preventable" trauma deaths occurred in regions without organized EMS or trauma care.[3] The investigators' methodology relied on physician panels who reviewed patient care case by case and then used various consensus methods to determine the appropriateness of the treatment. This technique has been criticized as being too subjective because blinding of the panel participants to the treatment setting is often extremely difficult and the various means used to reach consensus produce different results.[4] Later studies relied on series of patients treated at one or more trauma centers and compared them with those patients treated in a nontrauma center within a region[5] or across the United States,[6] using prospectively collected, standardized data on severity and outcome. In all comparisons between organized and nonorganized EMS and trauma systems, patient outcome was worse without organization.[5, 7] A number of studies and their methodologies have been summarized in publications.[6, 8] To deliver the best possible trauma care, it is crucial that trauma victims first receive competent on-scene prehospital EMS care before being removed directly to a hospital. In addition, because victims of severe trauma usually have a life-threatening condition, the receiving hospital must be sufficiently equipped and qualified to take care of their injuries.

Recent literature suggests that the outcomes of trauma patients clearly improve when prehospital care, triage, and admission to designated trauma centers are coordinated within regional trauma systems. It should be noted, however, that nearly all of these studies refer to the general trauma patient, and only a few primarily address the patient with TBI. There are no published data suggesting that the lack of a trauma care system is superior to organized systems. A retrospective study that compared head trauma outcome before and after the implementation of a trauma system in Oregon reported an odds ratio of 0.80 for mortality after system implementation.[9]

A report of preventable deaths in San Diego County compared non-TBI and TBI deaths before and after instituting a regional trauma care system.[10] Reviewers were blinded to the facility where care was rendered. Preventable deaths for non-TBI cases decreased from 16/83 (20%) to 2/211 (1%) ($p<0.005$), and for TBI cases, preventable deaths decreased from 4/94 (5%) to 1/149 (0.7%) ($p<0.10$), respectively, before and after the trauma system was put in place.

Another before and after study compared outcome of injured patients in a rural hospital before it chose to meet American College of Surgeons Committee on Trauma guidelines for a level II trauma center with outcome after it became a level II trauma center.[11] Survival for all patients who had a calculated probability of survival of 25% was 13% before and 30% after meeting trauma center criteria. For patients with closed head trauma, the survival was 15.4% before and 32% after meeting the criteria.

Several articles studied the EMS system's impact within the overall trauma system. One study of two centers in New Delhi, India, and in Charlottesville, Virginia, compared mortality rates after head injury using the motor score portion of the GCS to stratify patients.[12] While outcome was not statistically different in those patients with the lowest motor scores, mortality in patients with a motor score of five was notably different. Patients in Charlottesville had a mortality of 4.8%, whereas those in New Delhi had a mortality of 12.5% (p=0.001). The authors postulated that one reason for this difference may be that only 0.5% of patients in New Delhi arrived to the hospital by ambulance, versus 84% in Charlottesville. In addition, only 7% of patients in New Delhi arrived at the hospital within one hour and an additional 33% in two to three hours, compared with 50% within one hour and an additional 39% within three hours in Virginia. Thus, the lack of an EMS system and delay in presentation were thought to be important factors that account for the difference in outcome between the two cities.

The second study compared trauma patients with an injury severity score (ISS) of 9 or more in Seattle and Monterrey, Mexico.[13] Patients were taken to an urban hospital in Monterrey and to a level I trauma center in Seattle. Overall mortality was 55% in Monterrey and 34% in Seattle (p=0.001). Deaths in Monterrey occurred in the field (40%) and in the Emergency Department (ED) (11%) compared with Seattle where 21% died in the field and 6% in the ED (p=0.001 and 0.003, respectively). In addition, at hospital arrival, 39% of patients in Monterrey had a systolic blood pressure less than or equal to 80 mm Hg compared with 18% (p=0.001) in Seattle. Of those patients who were hypotensive, 5% in Monterrey and 79% in Seattle underwent endotracheal intubation in the field (p=0.001), and 70% in Monterrey and 99% in Seattle had fluid resuscitation en route (p=0.001).

The need for the in-house presence of the trauma surgeon 24 hours a day versus the ability of the trauma surgeon to respond quickly to the hospital has generated significant controversy. A report from one level II trauma center in Oklahoma concluded that level II trauma centers with attending trauma surgeons who are available but not "in-house" have outcomes as good as those with surgeons present in the hospital at all times.[14] This study was performed internally comparing daytime hours when the attending trauma surgeon was in-hospital versus evening and night hours when call was taken from outside. Using survival as predicted by the Major Trauma Outcome Study, this study evaluated 3,689 patients with major trauma. Overall survival was 97% with a predicted survival rate of 96%. Subgroup analysis revealed that, for patients with a trauma score less than 12, predicted survival and actual survival was 84%. In comparing whether the trauma surgeon was present, patients with severe thoracoabdominal trauma had a predicted survival of 79% and actual survival of 77% when the surgeon was in-house, and a predicted and actual survival of 74% and 81%, respectively, when the surgeon was called in from outside. In addition, patients with head trauma had predicted survival of 61% and actual of 63% when the surgeon was immediately available, and 57% predicted and 63% actual when the surgeon came in from home. All p-values were described as nonsignificant. Whether or not the trauma surgeon takes call from home, the important point in delivering trauma care to the patient is the physical presence of an appropriate team at the time of patient arrival in the ED.

Another issue that has also resulted in significant controversy relates to experience and patient volume criteria. Using data collected by trauma nurse coordinators, a retrospective study evaluating volume measurements on patient outcome compared trauma centers in Chicago. The trauma centers treating larger volumes of trauma patients were found to have better patient outcomes than those with fewer admissions. Patients transported to low-volume centers had a 30% greater chance of death when compared with high-volume centers.[15] However, a recent report questions the impact of case volume on patient outcome. Richardson, et al. evaluated mortality and morbidity outcomes, such as length of stay of trauma patients by case volume per attending surgeon.[16] They found no difference based on annual case volume or years of experience. While the optimal number of cases per trauma center and per trauma surgeon may be debated, the individual physicians on the treating team must have adequate experience to make the complex decisions often required when caring for a patient with severe multisystem or brain injury.

Another study that evaluated 1,332 patients with femoral fractures who underwent operative repair compared outcome in terms of morbidity and mortality between trauma centers and non-trauma centers.[4] Morbidity was 21% in the trauma centers and 33% in the nontrauma centers (p=0.001), and mortality was 1.0% versus 2.2%, respectively.

Several studies from Quebec demonstrated similar results. Mortality for all trauma patients before implementation of a trauma system was 20%, but only 10% after the system was put in place.[17] A subsequent review of trauma care in Quebec compared the outcome of 2,756 trauma patients transported directly to a trauma center with 1,608 patients who first were treated at a local hospital and subsequently transferred to the trauma center.[18] Mortality was 4.8% for patients taken directly to the trauma center and 8.9% if transfer occurred later (p=0.001).

These findings apply to both adults and children transported by EMS systems directly from the scene to trauma centers. For example, in a study of 1,320 children of whom 98 sustained severe head injuries, mortality for the children brought directly from the accident scene to a pediatric trauma center was 27%. However, children transported first to the nearest available hospital and subsequently transferred to the trauma center had a mortality of 50%. [19]

A number of studies attempted to evaluate the differences and difficulties associated with providing trauma care in rural settings compared with urban settings that have integrated trauma systems. Rogers, et al. reviewed trauma deaths in an organized urban trauma system compared with a rural state without a formal trauma system.[20] The results are summarized in Table A. The authors suggest that the higher incidence of prehospital deaths may be related to delays in discovering the patient and the longer response and transport times required in the rural setting, particularly for interhospital transfers.

Young, et al. compared the outcome of patients with ISS >15 who were transported directly to their level I trauma center with those who were first taken to another rural hospital and subsequently transferred.[21] Outcome measures included mortality, total hospital days, and intensive care unit (ICU) days. When all patients were included the two groups did not differ. However, when patients who died within the first 24 hours were excluded, length of stay, both in the hospital and in the ICU, was significantly longer (p<0.05) in the group transferred from another hospital, although there was no difference in mortality. The GCS score of the patients who died within the first 24 hours should, however, be noted. The GCS score for the patients taken directly to the trauma center was 5, compared with 10 for those patients transferred from an outside hospital (p<0.05). In addition, of patients who died in the first 24 hours (probability of survival > 0.50), the observed mortality for the direct transport group was 28% (7/25) compared with 75% (12/16) in the transferred group (p<0.05). The authors stated that although

these differences were noted between the groups, the study did not identify specific subgroups that would clearly benefit from direct transport to the trauma center. However, they did recommend that whenever possible patients with major trauma should be transported from the scene directly to a trauma center.

As noted in the chapter Assessment: Glasgow Coma Scale Score, a significant percentage of patients with hospital GCS scores of 9 to 13 have serious intracranial injury requiring neurosurgical intervention, and poor outcome, but no studies were found that compared outcomes based on choice of destination.

Severe TBI patients transported to trauma centers without prompt neurosurgical care or intracranial pressure monitoring are at risk for a poor outcome. Acute subdural hematomas in severe TBI patients are associated with a 90% mortality rate if evaluated more than four hours after injury and only a 30% mortality rate if evaluated earlier.[22] If subdural evaluation is done in less than two hours after injury, one study reported a 70% decrease in mortality.[23] To achieve this surgical timing , 24-hour availability of CT scanning is necessary. Intracranial pressure monitoring guides specific treatment to maintain cerebral perfusion and is recommended based on supporting scientific evidence for improved patient outcome given in the *Guidelines for the Management of Severe Head Injury*.[1]

V. Summary

The management by EMS personnel of the head-injured patient prior to arrival at the hospital is influenced by a number of factors, including the mechanism of injury, the type and severity of injury, and the decision regarding choice of destination. When an integrated EMS and trauma system is in place and EMS agencies transport a patient directly from the scene of the accident to an appropriate receiving facility (trauma center), the patient is entered into a system of care that has been shown to improve overall patient outcome. Interhospital transfers of these head injury patients are known to delay the time until neurosurgical consultation and intervention. This delay puts the patient at great risk for secondary insult to the brain.

VI. Key Issues for Future Investigation

Prospective, controlled class I and II studies are needed to answer the following questions:

1. What effect do prehospital assessment, treatment, transport, and destination decisions have on the outcome of the patient with severe TBI?
2. How is outcome affected when patients are treated by organized EMS systems within a trauma system versus EMS systems without a trauma system? These studies should evaluate the various levels of EMS provider training and hospital preparation, and they must include patients with different degrees of severity of injury.
3. What is the role of EMS call-takers and dispatchers in assessing the potential severity of a head injury and in determining the appropriate level of responder to dispatch? How does this decision affect patient outcome? It has been suggested that the motor component of the GCS score may be a potential discriminator. The caller would ask the patient to follow simple commands, and if the patient is unable to do so, the call-taker should assume the worst.[24]

4. What is the impact of transport time on the outcome of patients with severe TBI, and under what conditions should a closer hospital be bypassed in order to bring a patient to a trauma center versus the added time in transport?
5. What are the minimum requirements for a facility that treats patients with severe TBI?
6. What is the optimum destination for patients with mild-to-moderate TBI based on patient outcome?

The outcomes of these studies will assist in the future analysis and development of EMS and trauma systems.

VII. Evidence Tables

Smith,[5] 1990

Description of Study: Analysis of data abstracted from computerized discharge information about patients with femoral shaft fractures requiring operation over a one-year period (n=1332) comparing morbidity and mortality between patients treated at trauma centers and those treated at nontrauma centers.

Classification: II

Conclusions: Patients treated in trauma care centers had significantly fewer deaths and complications than in nontrauma centers

Smith,[15] 1990

Description of Study: A cohort analysis was performed on data from severely injured patients using three statistical methods to determine the relationship between trauma center volume and mortality (n=1643).

Classification: II

Conclusions: Low-volume trauma centers (fewer than 140 patients annually) had significantly higher mortality, when adjusted for head injury, than did high-volume trauma centers (more than 200 patients annually) ($p< 0.04$).

Shackford,[16] 1987

Description of Study: Analysis of patients admitted after traumatic injury, of whom 283 were severely injured (trauma score 8). Of those who had sufficient data (n=189) to compare with a national cohort study that provided a model for predicting survival in patients, actual survival was 29% whereas predicted survival (PS) was 18%. In patients with penetrating injury, PS was 8% and actual survival was 20% (n=3393).

Classification: II

Conclusions: The improved survival was attributed to the integration of prehospital and hospital care and expeditious surgery.

Sampalis,[17] 1995

Description of Study: This study evaluated the impact of trauma center development and designation on mortality in Quebec, Canada, comparing mortality before and after the trauma system was implemented.

Classification: III

Conclusions: There was a significant reduction in trauma-related mortality after implementing a trauma system.

Mullins,[9] 1996

Description of Study: This study evaluated the influence of implementing the Oregon State-wide trauma system on admission distribution and risk of death using a before and after comparison.

Classification: III

Conclusions: The Oregon trauma system resulted in reduction in risk of trauma-related death.

Sampalis,[18] 1997

Description of Study: This study compared outcome of severely injured patients (including head trauma) who were transported directly to trauma centers with those who were transferred after first being transported to a less-specialized, local facility (n=1608).

Classification: III

Conclusions: This study showed that transport of severely injured patients from the scene to level 1 trauma centers is associated with a significant reduction in mortality.

Arreola-Risa,[13] 1995

Description of Study: This study compared patients with ISS > 8 in Seattle and Monterrey, Mexico.

Classification: III

Conclusions: There was significantly greater mortality in Monterrey compared to Seattle. EMS differences included fewer patients undergoing endotracheal intubation or fluid resuscitation in Mexico.

VII. Evidence Tables (continued)

Colohan,[12] 1989

Description of Study: This study compared outcome after head injury between New Delhi, India, and Charlottesville, Virginia, using the motor score of the GCS to group patients.

Classification: III

Conclusions: Outcome in New Delhi was significantly worse in patients with a GCSM of 5 compared to Charlottesville, suggesting that differences in EMS were significant factors.

Guss,[10] 1989

Description of Study: The authors compared non-central nervous system (CNS) and CNS preventable deaths before and after a trauma system was implemented.

Classification: III

Conclusions: Preventable deaths for both non-CNS and CNS patients decreased after placement of a trauma system.

Johnson,[19] 1995

Description of Study: This study compared the mortality of 98 children who sustained severe head injury and were transported directly to a pediatric trauma center with those that were first taken to the closest hospital and later transferred.

Classification: III

Conclusions: Mortality for children taken directly to the pediatric trauma center was 27% and for those taken to the closest hospital first was 50%.

Rogers,[20] 1997

Description of Study: Trauma deaths in an urban trauma system were compared with those in a rural state without a trauma system.

Classification: III

Conclusions: Rural patients were more likely to die at the scene and were found to have lower ISS scores. The authors suggest long discovery and transfer times as possible causes of the increased mortality and suggest focusing on improving the EMS system in rural areas.

Young,[21] 1998

Description of Study: Trauma patients with ISS >15 who were taken directly to a trauma center were compared with those who were first taken to a rural hospital and later transferred.

Classification: III

Conclusions: Patients taken directly to the trauma center had shorter ICU and total hospital stays although mortality was not different.

Thompson,[14] 1992

Description of Study: Cohort analysis of trauma admissions at a level II trauma center showed no difference between survival in that center and survival among patients in the Major Trauma Outcome Study (n >15,000). Whether the trauma surgeon was on call out of the hospital did not adversely affect survival in patients with severe thoracoabdominal injury, compared with the trauma surgeon available in-house (n=3689).

Classification: II

Classification: Level II trauma centers can achieve mortality rates equal to that shown in a large multicenter trauma study, and trauma surgeons promptly available from outside a hospital can produce mortality rates equal to in-house trauma surgeons.

Table A
Trauma mortality comparison between an urban (trauma system) setting and a rural (non-trauma system) setting.

		Urban	Rural
Scene Deaths		41%	72%
ISS*		54	39
Hospital Deaths	First 24 hours	40%	16%
	ISS	52	33
	After 24 hours	15%	12%
	ISS	37	21

*ISS — Injury Severity Score (ISS) is a calculated score derived from the Abbreviated Injury Scale (AIS) that scores the magnitude of an injury by anatomic region. The ISS is the sum of the squares of the AIS score for the three most severely injured body regions.

VIII. References

1. Guidelines for the Management of Severe Head Injury: Brain Trauma Foundation, NY 1995.
2. Sosin DM, Sniezek JE, Waxweiler RJ: Trends in death associated with traumatic brain injury 1979 through 1992. JAMA 273:1778-1780, 1995.
3. West J, Trunkey D, Lim R: Systems of trauma care. A study of two counties. Arch Surg 114:455-460, 1979.
4. Wilson DS, McElligott J, Fielding LP: Identification of preventable trauma deaths: confounded inquiries? J Trauma 32:45-51, 1992.
5. Smith J, Martin L, Young W, et al.: Do trauma centers improve outcome over nontrauma centers? The evaluation of regional trauma care using discharge abstract data and patient management categories. J Trauma 30:1533-1538, 1990.
6. Shackford SR, Mackersie RC, Hoyt DB, et al.: Impact of a trauma system on outcome of severely injured patients. Arch Surg 122:523-527, 1987.
7. Roy P: The value of trauma centres: a methodologic review. Can J Surg 30:7-22, 1987.
8. Pantridge JF, Geddes JS: A mobile intensive care unit in the management of myocardial infarction. Lancet 2:271-273, 1967.
9. Mullins R, Venum-Stone J, Hedges JR, et al.: Influence of a statewide trauma system on the location of hospitalization and outcome of injured patients. J Trauma 40:536-545, 1996.
10. Guss DA, Meyer FT, Neuman TS, et al.: The impact of a regionalized trauma system of trauma care in San Diego County. Ann Emerg Med 18:1141-1145, 1989.
11. Norwood S, Fernandez L, England J: The early effects of implementing American College of Surgeons Level II criteria on transfer and survival rates at a rurally-based community hospital. J Trauma 39:240-245, 1995.
12. Colohan ART, Alves WM, Gross CR, et al.: Head injury mortality in two centers with different emergency medical services and intensive care. J Neurosurg 71:202-207, 1989.
13. Arreola-Risa C, Mock CN, Cavazos L, et al.: Trauma care systems in urban Latin America: the priorities should be prehospital and emergency room management. J Trauma 39:457-462, 1995.
14. Thompson C, Bickell W, Siemens R, et al.: Community hospital Level II trauma center outcome. J Trauma 32:336-343, 1992.
15. Smith R, Frateschi L, Sloan E, et al.: The impact of volume on outcome in seriously injured trauma patients: two years' experience of the Chicago Trauma System. J Trauma 30:1066-1076, 1990.
16. Richardson JD, Schmieg R, Boaz P, et al.: Impact of trauma attending surgeon case volume on outcome: is more better? J Trauma 44:266-272, 1998.
17. Sampalis JS, Lavoie A, Boukas S, et al.: Trauma center designation: initial impact on trauma-related mortality. J Trauma 39:232-239, 1995.
18. Sampalis JS, Denis R, Frechette P, et al.: Direct transport to tertiary trauma centers versus transfer from lower level facilities: impact on mortality and morbidity among patients with major trauma. J Trauma 43:288-296, 1997.
19. Johnson DL, Krishnamurthy S: Send severely head-injured children to a pediatric trauma center. Pediatr Neurosurg 25:309-314, 1996.

20. Rogers FB, Shackford SR, Hoyt DB, et al.: Trauma deaths in a mature urban vs. rural trauma system. Arch Surg 132:376-382, 1997.
21. Young JS, Bassam D, Cephas GA, et al.: Interhospital versus direct scene transfer of major trauma patients in a rural trauma system. Am Surg 64:88-92, 1998.
22. Seeling JM, Becker DP, Miller JD, et al.: Traumatic acute subdural hemotoma: major mortality reduction in comatose patients treated within four hours. N Engl J Med 304: 1511-1518, 1981.
23. Haselberger K, Pucher R, Auer LM: Prognosis after acute subdural or epidural hemorrhage. Acta Neurochir 90: 111-116, 1988.
24. Meredith JW, Rutledge R, Hansen AR, et al.: Field triage of trauma patients based upon ability to follow commands: a study in 29,573 injured patients. J Trauma 38:129-135, 1995.

EXPLANATION

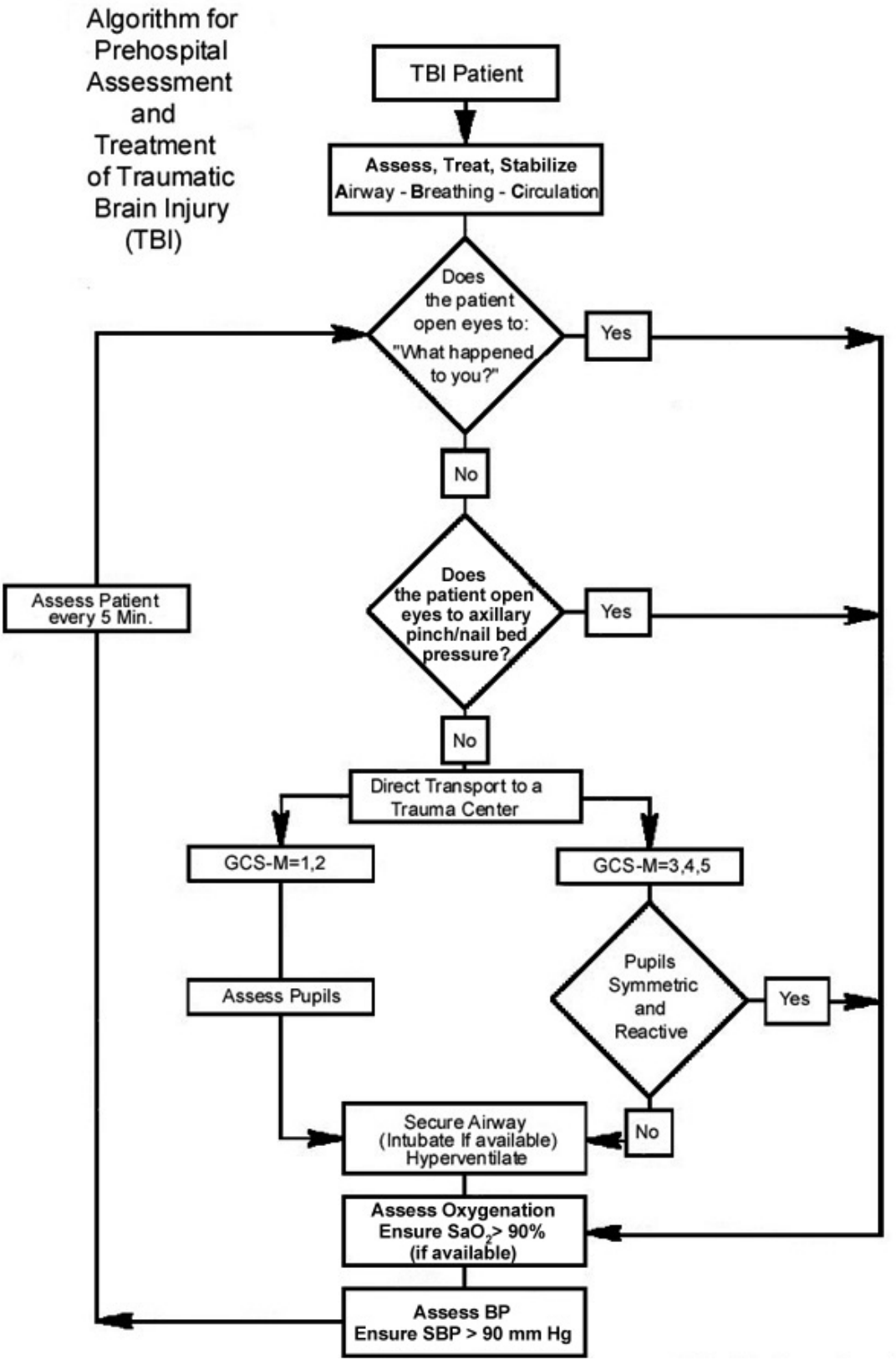
OF THE ALGORITHM FOR PREHOSPITAL ASSESSMENT AND TREATMENT OF TRAUMATIC BRAIN INJURY

The Emergency Medical Services (EMS) task force used a consensus method to develop an algorithm based on the scientific evidence contained in *Guidelines for Prehospital Management of Traumatic Brain Injury.* The algorithm can be used as a framework to assess, treat, and transport the patient with traumatic brain injury (TBI). Individual and regional circumstances may require prehospital health care providers to modify the algorithm, because it may not be appropriate for all patients and locations. The following points provide more detail for summaries of the steps in the graphic algorithm:

- The health care provider's first priority in assessing, stabilizing, and treating a TBI patient is to follow basic resuscitation protocols that prioritize airway, breathing, and circulation assessment and treatment.
- Following stabilization of airway, breathing, and circulation, the health care provider assesses the patient by first asking him or her, "What happened to you?"
- If the patient opens his or her eyes, the provider then asks the questions in the verbal and motor sections of the Glasgow Coma Scale (GCS) to determine the total score. Patients with a GCS score of 9 to13 (moderate TBI) and patients with a GCS score of 3 to 8 (severe TBI) should be transported to a trauma center.
- If the patient does not open his or her eyes, the health care provider applies blunt pressure to the nail bed or pinches the anterior axillary skin to elicit eye opening.
- If the patient opens his or her eyes with nail bed pressure or axillary pinch, the health care provider assesses the verbal and motor sections of the GCS to determine the total score.
- Patients who are unresponsive with a GCS score of 3 to 8 should be transported to a trauma center with the following TBI capabilities:
  1. 24-hour CT scanning capability
  2. 24-hour available operating room and prompt neurosurgical care
  3. The ability to monitor intracranial pressure and treat intracranial hypertension as delineated in the *Guidelines for the Management of Severe Head Injury.*
- Patients with a GCS score of 14 to15 can be transported to a nontrauma center hospital, which has the basic emergency department capabilities for immediate resuscitation of the critically injured.
- If the patient does not open his or her eyes with nail bed pressure or axillary pinch, he or she should be transported directly to a trauma center described above.

- For unresponsive patients who respond to nail bed pressure with extensor posturing or who are flaccid, the health care provider should secure the airway (intubate, if available) and hyperventilate (20 bpm in an adult, 30 bpm a child, and 35–40 bpm in an infant).
- For unresponsive patients who respond to nail bed pressure or axillary pinch with abnormal flexion or a higher GCS motor response, but have asymmetric and/or dilated and fixed pupil(s), the prehospital health care provider should hyperventilate at the rates described above.
- All TBI patients should have their oxygenation assessed at least every five minutes and their O_2 saturation maintained at >90%. Systolic blood pressure should also be measured, and maintained greater than 90 mm Hg in adults and for ages 12 to 16; 80 mm Hg for ages 5 to 12; 75 mm Hg for ages 1 to 5; and 65 mm Hg for infants less than one year of age.

Because the patient's neurological status may change, the health care provider should fully assess the patient every five minutes and treat or modify treatment as appropriate.


Algorithm for Prehospital Assessment and Treatment of Traumatic Brain Injury (TBI)
TBI Patient
Assess, Treat, Stabilize
Airway - Breathing - Circulation
Does the patient open eyes to: "What happened to you?"
Yes
No
Does the patient open eyes to axillary pinch/nail bed pressure?
Yes
No
Assess Patient every 5 Min.
Direct Transport to a Trauma Center
GCS-M=1,2
GCS-M=3,4,5
Assess Pupils
Pupils Symmetric and Reactive
Yes
No
Secure Airway (Intubate If available) Hyperventilate
Assess Oxygenation Ensure SaO_2 > 90% (if available)
Assess BP Ensure SBP > 90 mm Hg