

Practice Management Guidelines for the Management of Mild Traumatic Brain Injury: The EAST Practice Management Guidelines Work Group

James G. Cushman, MD, Nikhilesh Agarwal, MD, Timothy C. Fabian, MD, Victor Garcia, MD, Kimberly K. Nagy, MD, Michael D. Pasquale, MD, and Arnold G. Salotto, MD

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I. STATEMENT OF THE PROBLEM

Mild traumatic brain injury (MTBI), or concussion, is a common cause for admission at trauma centers, particularly those centers admitting primarily blunt trauma victims. Represented by ICD-9-CM codes 850.0–850.9, MTBI may be generally defined as an injury caused by blunt acceleration/deceleration forces which produce a period of unconsciousness for 20 minutes or less and/or brief retrograde amnesia, a Glasgow Coma Scale (GCS) score of 13 to 15, no focal neurologic deficit, no intracranial complications (e.g., seizure activity), and normal computed tomography (CT) findings.^{1–3} This brief loss of consciousness and/or retrograde amnesia has to be referred to as a transient disturbance of neurologic function and is a sine qua non to the diagnosis of MTBI. Focal neurologic deficits as well as seizure activity fall outside the definition of MTBI in this guideline. Despite the frequency of MTBI, there is no uniform agreement regarding the nature of the illness, the role of a variety of diagnostic tests, or the necessity of acute hospitalization.

Neurotrauma textbooks and a large number of review articles have addressed the definition, epidemiology, and clinical characteristics of MTBI.^{1–8} Similarly, a number of studies have examined the role of CT^{9–31} and neuropsychological testing^{32–46} in the diagnosis and management of MTBI. Several studies, mostly retrospective, suggest which patients might be best served by hospital admission versus evaluation and discharge to home.^{9,47–53} Additional studies exist regarding management strategies in MTBI from the neurosurgeon's perspective.^{17,28,31,54–64} Finally, the complicated and poorly understood issues surrounding posttraumatic and emotional symptoms in patients with MTBI are discussed

in several publications.^{65–69} From this core of knowledge, recommendations can be made to facilitate a safe, more uniform, and cost-effective approach to the understanding and management of MTBI.^{9,15,70–72}

II. PROCESS

A computerized search of MEDLINE and Cochrane databases was performed. Key words included brain injury, concussion, closed head injury, and/or brain trauma, including modifiers such as mild and minor. English language references between 1975 and 1998 were listed.

Primary exclusions involved studies or reviews not relevant to acute MTBI. Approximately 100 remaining citations were supplemented by reference sections from selected articles and texts. For the purposes of developing an institutional protocol, secondary exclusions included eliminating poor quality studies or reviews felt noncontributory or redundant. Subcommittee members for this EAST document followed a similar process, which yielded a total of 75 citations (see Evidentiary Table on page 1038).

III. RECOMMENDATIONS

A. Level I

There are insufficient data to support a recommendation at this level.

B. Level II

1. CT of the brain is the gold standard diagnostic imaging study for MTBI patients and should be performed on all patients sustaining a transient disturbance of neurologic function secondary to trauma.
2. MTBI patients perform less well on complicated tasks requiring prolonged attention and rapid response times when compared with controls, and this deficit resolves in the majority of patients by 1 month postinjury. Patients may be advised and reassured of this prognosis during outpatient follow-up.
3. A subset of patients sustaining MTBI will develop persistent symptoms in the absence of anatomic findings. Patients who continue to experience symptoms more than 6 weeks after MTBI should undergo formal neuropsychological testing. A variety of tests can be performed,

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From Lehigh Valley Hospital (J.G.C., M.D.P., A.G.S.), Allentown, Pennsylvania, Wyntrebroke S.A. (N.A.), York, Pennsylvania, University of Tennessee (T.C.F.), Memphis, Tennessee, Children's Hospital Medical Center (V.G.), Cincinnati, Ohio, and Cook County Hospital and Rush University (K.K.N.), Chicago, Illinois.

Address for correspondence: Michael D. Pasquale, MD, Department of Surgery, Division of Trauma/Surgical Critical Care, Lehigh Valley Hospital, P.O. Box 689, Cedar Crest and I-78, Allentown, PA 18105-1556; email: sally.lutz@lvh.com.

Patient Label Stamp Here

Lehigh Valley Hospital
Mild Traumatic Brain Injury—Physician Discharge Checklist

Instructions. This form is to be completed prior to discharging the above trauma patient, in accordance with the Mild Traumatic Brain Injury (MTBI) protocol. The purpose of this checklist is to simplify the process of safely discharging this trauma patient who has been diagnosed with MTBI, and who has no other injuries requiring hospital admission. This form confirms that the patient has satisfied safe discharge criteria and will be released from either the trauma bay or from an "observation room" in the Emergency Department. *Please check off the appropriate boxes, and hand this sheet to the Team Leader on call or place into the Red Book.*

Checklist (Check only if yes)

- | | |
|---|---|
| <input type="checkbox"/> Normal head CT | <input type="checkbox"/> C-spine clinically cleared |
| <input type="checkbox"/> GCS score 15 | |
| <input type="checkbox"/> Low-risk symptoms* | Or |
| <input type="checkbox"/> No drugs/EtOH | |
| <input type="checkbox"/> Not currently on anticoagulants | <input type="checkbox"/> C-spine cleared radiographically |
| <input type="checkbox"/> Reliable Adult | |
| <input type="checkbox"/> Lives within 30 min | |
| <input type="checkbox"/> CXR - wnl | |
| <input type="checkbox"/> Pelvis X-ray - wnl, or | |
| <input type="checkbox"/> Pelvis X-ray - n.d. | X-rays reviewed by radiologist |
| <input type="checkbox"/> Discharge pack given to patient | <input type="checkbox"/> Yes <input type="checkbox"/> No |
| <input type="checkbox"/> Follow-up appointments with other specialists (e.g., Ortho) given to patient | |
| <input type="checkbox"/> Index card with patient's name, address, and phone number attached to this sheet | |

*Low-risk symptoms = asymptomatic, mild headache/dizziness, or small scalp lac

Once this form is completed, place into the Red Book, or give to Team Leader. Thank you.

Signature of physician completing form

Date

Fig. 1. Lehigh Valley Hospital Physician Discharge Checklist.

although the data do not clearly identify which one is better or best.

C. Level III

1. Patients sustaining MTBI as an isolated diagnosis following a complete trauma evaluation may, at the discretion of the responsible physician, be discharged from the emergency department/trauma evaluation area if they fulfill certain "safe discharge" criteria.
2. Postconcussive symptoms include headache, dizziness, memory problems, and other symptoms that occur acutely in approximately 50% of MTBI patients, and in 33% at 3 months from injury. These symptoms may identify a subgroup of patients at subsequent increased risk for prolonged cognitive deficits as a result of their injury.
3. Neuropsychological testing of MTBI patients in the acute setting has been suggested to identify patients at high risk for prolonged cognitive deficits; however, it needs further study.

IV. SCIENTIFIC FOUNDATION

A. Definition, Epidemiology, and Natural History of MTBI

Although various authors give modifications to the definition of MTBI,^{3,5,6} an acceptable working definition for

Table 1 Patterns of Posttraumatic Symptoms At Discharge and 3-Month Follow-Up^a

Symptom Pattern	At Discharge, No. (%)	At 3 Months, No. (%)
None	457 (41.2)	183 (33.8)
Headaches	249 (22.5)	61 (11.3)
Memory problems	43 (3.9)	17 (3.1)
Dizziness	20 (1.8)	16 (2.9)
Weakness	22 (1.9)	7 (1.3)
Nausea	14 (1.3)	0
Numbness	0	12 (2.2)
Tinnitus	0	6 (1.1)
Double vision	0	6 (1.1)
Headaches and memory problems	38 (3.4)	13 (2.4)
Headaches and dizziness	41 (3.7)	13 (2.4)
Headaches and nausea	27 (2.4)	0
Headaches and weakness	17 (1.5)	0
Headaches and numbness	12 (1.1)	0
Headaches and tinnitus	0	9 (1.7)
Numbness and tinnitus	0	7 (1.3)
Headaches, memory problems, and dizziness	0	6 (1.1)
Headaches, dizziness, and tinnitus	0	6 (1.1)
Total	940 (84.7)	362 (66.8)

^a From Chambers J, Cohen SS, Hemminger L, Prall JA, Nichols JS. Mild traumatic brain injuries in low-risk trauma patients. *J Trauma*. 1996;41:976-980.

Evidentiary Table: Mild Traumatic Brain Injury—A Literature Review

First Author	Year	Reference	Data Class	Conclusions
Definition, Epidemiology, and Natural History of MTBI¹⁻⁸				
Alexander MP	1995	Mild traumatic brain injury: pathophysiology, natural history, and clinical management. <i>Neurology</i> . 45: 1253-1260.	III	Review article, literature review.
Gennarelli TA	1986	Mechanisms and pathophysiology of cerebral concussion. <i>J Head Trauma Rehabil</i> . 1:23-29.	III	Review article.
Levin HS	1996	Outcome from mild head injury. In: Narayan RK, et al., eds. <i>Neurotrauma</i> . NY: McGraw-Hill.	III	Book chapter: Outcome from Mild Head Injury.
Stein SC	1996	Outcome from moderate head injury. In: Narayan RK, et al., eds. <i>Neurotrauma</i> . NY: McGraw-Hill.	III	Book chapter: Outcome from Moderate Head Injury.
Kraus JF	1988	The epidemiology of mild, uncomplicated brain injury. <i>J Trauma</i> . 28:1637-1643.	III	Retrospective 1-year review of MHI; 80% of diagnoses were concussion; cost \$6 million in 1981 dollars.
Williams DH	1990	Mild head injury classification. <i>Neurosurgery</i> . 27:422-428.	II	Prospective study of patients with uncomplicated MTBI who had 97% good recovery rate using neuropsychologic measures 6 months postinjury compared to complicated MTBI (84%) and moderate CHI (73%).
Jennett B	1978	The problem of mild head injury. <i>Practitioner</i> . 221: 77-82.	III	According to Jennett, there will be a few patients each year that deteriorate despite MTBI. Early CT may ameliorate this problem.
Oppenheimer DR	1968	Microscopic lesions in the brain following head injury. <i>J Neurol Neurosurg Psychiatry</i> . 31:299-306.	III	Five cases of MTBI where brains were examined histopathologically, revealing microglial reaction (clusters) suggestive of anatomic injury.
Diagnosis⁹⁻³¹				
Shackford SR	1992	The clinical utility of computed tomographic scanning and neurologic examination in the management of patients with minor head injury. <i>J Trauma</i> . 33:385-394.	III	This Western Trauma Association multicenter review concluded it was safe to discharge patients to home with a normal neurologic exam and normal head CT.
Stein SC	1992	Mild head injury: a plea for routine early CT scanning. <i>J Trauma</i> . 33:11-13.	III	Retrospective: all patients admitted. No home follow-up.
Livingston DH	1991	The use of CT scanning to triage patients requiring admission following minimal head injury. <i>J Trauma</i> . 31:483-489.	II	Prospective: 63% phone follow up. All underwent head CT; >80% discharged.
Mohanty SK	1991	Are CT scans for head injury patients always necessary? <i>J Trauma</i> . 31:801-805.	III	Retrospective study presented at 1990 AAST; 12/348 head CT abnormal in patients without focal neurologic deficits. Critical of routine head CT.
Dunham CM	1996	Compelling evidence for discretionary brain computed tomographic imaging in those patients with mild cognitive impairment after blunt trauma. <i>J Trauma</i> . 41:679-686.	III	Used admission GCS and cranial soft tissue injury (CSTI) index to predict need for head CT. Advocated selective head CT approach.
Marshall LF	1991	A new classification of head injury based on computerized tomography. <i>J Neurosurg</i> . 75:S14-S20.	III	Nice paper on reading head CTs for trauma.
Camins MB	1996	Radiologic studies and cost-effectiveness in head injuries. <i>Bull Am Coll Surg</i> . 81:16-18, 47.	III	The American College of Surgeons opinion on the role of various radiographic modalities in CHI.
Lloyd DA	1997	Predictive value of skull radiography for intracranial injury in children with blunt head injury. <i>Lancet</i> . 349:821-824.	II	Neurologic exam, and not the presence/absence of skull fracture on x-ray, was more predictive of injury. Some methods problems.
Culotta VP	1996	Clinicopathological heterogeneity in the classification of mild head injury. <i>Neurosurgery</i> . 38:245-250.	III	Incidence of brain scan abnormalities differed between among GCS scores of 13-15: 4% when GCS = 15; 16% when GCS = 14; 28% when GCS = 13.

First Author	Year	Reference	Data Class	Conclusions
Masters SJ	1987	Skull x-ray examinations after head trauma. <i>N Engl J Med.</i> 316:84–91.	II	Prospective, multi-institutional study. May omit plain skull films in low-risk patients.
Murshid WR	1994	Role of skull radiography in the initial evaluation of minor head injury: a retrospective study. <i>Acta Neurochir.</i> 129:11–14.	III	Midline of skull fracture in GCS 13–15 population 11%. Neurosurgical intervention in 3%.
Servadei F	1998	Skull fracture as a risk factor of intracranial complications in minor head injuries: a prospective CT study in a series of 98 adult patients. <i>J Neurol Neurosurg Psychiatry.</i> 51:526–528.	II	Prospective (n = 98) but limited study. Presence of skull fracture predicts need for neurosurgical intervention.
Feurman T	1988	Value of skull radiography, head computed tomographic scanning and admission for observation in cases of minor head injury. <i>Neurosurgery.</i> 22:449–453.	III	Retrospective study which suggests patients may be discharged to home if initial brain CT is negative.
Miller EC	1996	Minor head trauma: is computed tomography always necessary? <i>Ann Emerg Med.</i> 27:290–294.	II	Prospective study, but no control group, not randomized. Represents the strongest data to support selective use of initial brain CT in GCS 15 MTBI patients.
Harad FT	1992	Inadequacy of bedside clinical indicators in identifying significant intracranial injury in trauma patients. <i>J Trauma.</i> 32:359–363.	III	In GCS \geq 13, abnormal CT rate 18%. Neurosurgical intervention in 4%.
Jeret JS	1993	Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. <i>Neurosurgery.</i> 32:9–16.	II	Equally strong data to oppose Miller et al. ²² which concludes that cranial lesions cannot be excluded based on clinical findings. Brain CT is required.
Borczuk P	1995	Predictors of intracranial injury in patients with mild head trauma. <i>Ann Emerg Med.</i> 25:731–736.	III	Over 1400 patients received; age >60, signs of basilar skull fracture, cranial soft tissue injury were high-risk variables in MTBI patients.
Taheri PA	1993	Can patients with minor head injuries can be safely discharged home? <i>Arch Surg.</i> 128:289–292.	III	Yes, patients with minor head injuries can be safely discharged home.
Ros SP	1989	Should patients with normal cranial CT scans following minor head injury be hospitalized for observation? <i>Pediatr Emerg Care.</i> 5:216–218.	III	Small series concluding discharge to home is safe, but no time period for observation is specified.
Stein SC	1990	The value of computed tomographic scans in patients with low-risk head injuries. <i>Neurosurgery.</i> 26:638–640.	III	Recommended initial brain CT if (+) LOC or amnesia as 5% of their MTBI patients needed neurosurgery which was predicted by CT.
Stein SC	1991	Is routine computed tomography scanning too expensive for mild head injury. <i>Ann Emerg Med.</i> 20:1286–1289.	III	The study is limited as it only compared cost of CT scan vs. admission.
Hsiang JN	1997	High-risk mild head injury. <i>J Neurosurg.</i> 87:234–238.	II	Advocates role of initial brain CT. Among those patients with an abnormal scan, 10% required neurosurgery. When brain scan normal, 0%.
Dacey RG Jr	1986	Neurosurgical complications after apparently minor head injury. <i>J Neurosurg.</i> 65:203–210.	III	Another older study concluding pressure of skull fracture associated with need for neurosurgical procedure and necessary role of initial brain scan.
Role of Neuropsychiatric Testing ^{31–46,69}				
Rimel RW	1981	Disability caused by minor head injury. <i>Neurosurgery.</i> 9:221–228.	II	424/538 patients followed-up at 3 months s/p minor head injury; 80% had PCS with evidence of organic brain damage, emotional stress as major etiologies (not legal); 34% remained unemployed.
Barth JT	1983	Neuropsychological sequelae of minor head injury. <i>Neurosurgery.</i> 13:529–533.	III	71/1248 patients followed-up at 3 months s/p minor head injury. “Significant %” had cognitive impairment as determined on three different tests.

Evidentiary Table: Mild Traumatic Brain Injury—A Literature Review (Continued)

First Author	Year	Reference	Data Class	Conclusions
Hughenholz H	1988	How long does it take to recover from a mild concussion? <i>Neurosurgery</i> . 22:853–858.	I	In tests of attention and information processing, 22 concussed patients were significantly slower than controls at 1 and 3 months. Improvement was seen, and by the first month were equal. Excellent reference.
Bohnen N	1992	Neuropsychological deficits in patients with persistent symptoms 6 months after mild head injury. <i>Neurosurgery</i> . 30:692–696.	II	Patients with postconcussive symptoms 6 months after injury perform less well on tests of attention than injured patients without PCS and control. Small numbers, questionable methods.
Chambers J	1996	Mild traumatic brain injuries in low-risk trauma patients. <i>J Trauma</i> . 41:976–980.	III	Phone survey, small numbers. Found that 11% of blunt trauma victims had PCS at 2 months.
Gentilini M	1985	Neuropsychological evaluation of mild head injury. <i>J Neurol Neurosurg Psychiatry</i> . 48:137–140.	III	Neuropsychologic testing in 50 MTBI patients was equivalent to 50 core-controls at 1 month.
Beers SR	1992	Cognitive effects of mild head injury in children and adolescents. <i>Neuropsychol Rev</i> . 3:281–320.	III	A doctoral thesis summarizing a comprehensive review of mild head injury studies in children.
Veltman RH	1993	Cognitive screening in mild brain injury. <i>J Neurosci Nurs</i> . 25:367–371.	III	166 patients with mild TBI (all GCS 13–15) had screening with Neurobehavioral Cognitive Status Exam (NCSE) prior to discharge with additional cognitive testing as outpatient if NCSE abnormal; 39% had abnormal results of NCSE; 97% of those with follow-up testing (39) had cognitive deficits; 25% of MTBI patients had positive cognitive screen while in hospital which correlated with deficits in follow-up 3 weeks later.
Parker RS	1994	Neurobehavioral outcome of children's mild traumatic brain injury. <i>Semin Neurol</i> . 14:67–73.	III	Case review format of various neuropsychologic sequelae in MTBI.
Cicerone KD	1996	Attention deficits and dual task demands after mild traumatic brain injury. <i>Brain Inj</i> . 10:79–89.	III	15 patients referred 18 months after MTBI had slower processing speeds than a community control group.
Fischer FP Jr	1981	Postconcussive hospital observation of alert patients in a primary trauma center. <i>J Trauma</i> . 21:920–924.	III	Retrospective 6-month review. Only skull fracture (43/333) associated with need for admit/neurosurgical sequelae.
High WM Jr	1990	Recovery of orientation following closed-head injury. <i>J Clin Exp Neuropsychol</i> . 12:703–714.	II	84 patients followed after CHI (20% had GCS of 13–15). Return of orientation occurred first to person, then place, and finally time.
Bassett SS	1990	Neuropsychological function in adolescents sustaining mild closed head injury. <i>J Pediatr Psychol</i> . 15:225–236.	II	29 adolescents tested after CHI due to MVC (65% had GCS 13–15) were compared to 29 healthy adolescents. Tests included Wechsler Intelligence Scale, Wechsler Memory Scale, Buschke Selective Reminding Test, Trail Making Test, Wisconsin Card Sorting Test, and Controlled Oral Word Association Test. Even with mild head injuries, there was evidence of difficulty with abstract reasoning and verbal memory and learning.
Haaland KY	1994	Recovery of simple motor skills after head injury. <i>J Clin Exp Neuropsychol</i> . 16:448–456.	II	40 patients with acute head injury (75% had GCS of 11–15) were compared to 88 controls. Finger-tapping and grip strength were measured 1 month and 1 year postinjury. Finger tapping improved in the injured group at 1 year but was slower than controls at both time points. Grip strength was poorer at 1 month for injured group and improved at 1 year; both groups were equivalent at 1 year. Conclusion: motor deficits may persist as long as 1 year after head injury.
Reimer W	1995	The neuropsychological spectrum in traumatically head-injured persons. <i>Brain Inj</i> . 9:55–60.	III	125 patients with traumatic brain injury were administered a battery of tests as they entered a rehabilitation program. Acquired knowledge was affected less than neuropsychologic function (memory, visual, and sensory perception) by brain injury.

First Author	Year	Reference	Data Class	Conclusions
Kreutzer JS	1996	Validation of a neurobehavioral functioning inventory for adults with traumatic brain injury. <i>Arch Phys Med Rehabil.</i> 77:116–124.	II	Outpatient neuropsychologic testing s/p MTBI with objective scoring systems correlated with patient perception of postinjury neurologic problems. These may be used clinically to assist in patient treatment and follow-up.
Management ^{17,28,31,47–64,70–72}				
Watson MR	1995	The post-concussion state: neurophysiological aspects. <i>Br J Psychol.</i> 167:514–521.	II	72% of MTBI patients had resolution of postconcussion symptoms at 6 weeks. Those that don't should have neuroelectrophysiologic studies done, according to these authors.
Klein M	1996	Long-term persisting cognitive sequelae of traumatic brain injury and the effect of age. <i>J Nerv Ment Dis.</i> 184:459–467.	III	Descriptive study with methodologic problems.
Macciocchi SN	1996	Neuropsychological functioning and recovery after mild head injury in collegiate athletes. <i>Neurosurgery.</i> 39:510–514.	III	10 NCAA athletic departments in the study. Young health MTBI patients generally recover rapidly and completely from MTBI.
Coonley-Hoganson R	1984	Sequelae associated with head injuries in patients who were not hospitalized: a follow-up survey. <i>Neurosurgery.</i> 14:315–317.	III	Retrospective/telephone survey. Methodologic weaknesses. Headaches, dizziness, and drowsiness in 27%, 11%, and 9% at 1 week. Two thirds followed head sheet instructions.
Klauber MR	1989	Determinants of head injury mortality: importance of the low risk patient. <i>Neurosurgery.</i> 24:31–36.	II	Differences in outcome of patients with CHI comparing hospitals lie in the care of the “low-risk” patients.
Wisner DH	1993	Head injury from a general surgeon's perspective. <i>Adv Trauma Crit Care.</i> 8:183–216.	III	Literature review and synopsis from a prominent academic traumatologist.
Rhodes M	1996	Role of the trauma surgeon in neurotrauma. In: Narayan RK, et al., eds. <i>Neurotrauma.</i> NY: McGraw-Hill.	III	Guidelines for when to consult with neurosurgeons, as developed by trauma and neurosurgeons at Lehigh Valley Hospital in Allentown, Pennsylvania.
Lawler KA	1996	Guidelines for evaluation and education of adult patients with mild traumatic brain injuries in an acute care hospital setting. <i>J Head Trauma Rehabil.</i> 11:18–28.	III	Literature-based recommendations on the evaluation and education of MTBI patients prior to discharge.
Wilberger JE Jr	1997	Development of guidelines for severe head injury. <i>Bull Am Coll Surg.</i> 82:29–33.	III	The American College of Surgeons Advisory Council data-based guidelines for severe CHI.
Anonymous	1997	Practice parameter: the management of concussion in sports. <i>Neurology.</i> 48:581–585 (see also pp 575–580).	III	The American Academy of Neurology's Report of the Quality Standards Committee (follows EAST's evidence-based format).
Bailes JE	1999	<i>Sports-Related Concussion.</i> St Louis, MO: Quality Medical Publishing; 115–139.	III	Book chapter: Diagnosis and management of head injury.
Culotta VP	1996	Clinicopathologic heterogeneity in the classification of mild head injury. <i>Neurosurgery.</i> 38:245–250.	III	Retrospective review found 10 times higher rate for surgery in patients with GCS of 13 compared to those with GCS of 15.
Dacey RG Jr	1986	Neurosurgical complications after apparently minor head injury: assessment of risk in a series of 610 patients. <i>J Neurosurg.</i> 65:203–210.	II	Prospective study of 610 patients: recommended discharge of patients with GCS of 15 and normal head CT. Cost analysis performed, only 11% of patients had CT scan performed.
Davis RL	1995	The use of cranial CT scans in the triage of pediatric patients with mild head injury. <i>Pediatrics.</i> 95:345–349.	III	Retrospective review: safe to discharge patients with GCS of 15 and normal head CT.

Evidentiary Table: Mild Traumatic Brain Injury—A Literature Review (Continued)

First Author	Year	Reference	Data Class	Conclusions
Gomez PA	1996	Mild head injury: differences in prognosis among patients with a Glasgow Coma Scale score of 13 to 15 and analysis of factors associated with abnormal CT findings. <i>Br J Neurosurg.</i> 10:453–460.	III	Retrospective review: significantly higher incidence of surgical intervention and neurologic complications with GCS 13–14.
Kay T	1993	Neuropsychological treatment of mild traumatic brain injury. <i>J Head Trauma Rehabil.</i> 8:74–85.	III	Guidelines presented for neuropsychological treatment of MTBI.
Marshall LF	1983	The National Traumatic Coma Data Bank, part 2: patients who talk and deteriorate—implications for treatment. <i>J Neurosurg.</i> 59:285–288.	II	Prospective study: 34/325 patients had a verbal GCS score of at least 3 prior to deterioration, 18 of these 34 died.
Reilly PL	1975	Patients with head injury who talk and die. <i>Lancet.</i> 2:375–377.	III	Case review of 66 patients who died after initially talking after injury. 75% associated with hematomas.
Rhodes M	1993	Selective neurosurgical consultation for trauma. <i>J Trauma.</i> 35:979.	III	Stratified neurosurgical consultation based on clinical and CT findings into URGENT, NONURGENT, and NO CONSULTATION categories.
Riesgo P	1997	Delayed extradural hematoma after mild head injury: report of three cases. <i>Surg Neurol.</i> 48:226–231.	III	Case reports of delayed extradural hematomas.
Rockswold GL	1993	Patients who talk and deteriorate. <i>Ann Emerg Med.</i> 22:1004–1007.	III	Review of subject of patients who deteriorate neurologically.
Rockswold GL	1987	Analysis of management in thirty-three closed head injury patients who “talked and deteriorated.” <i>Neurosurgery.</i> 21:51–55.	III	Retrospective review: 33/215 patients “talked and deteriorated,” most commonly due to subdural hematoma, 44% died, emphasized importance of rapid diagnosis and intervention.
Saunders RL	1984	The second impact in catastrophic contact-sports head trauma. <i>JAMA.</i> 252:538–539.	III	Case report of neurologic deterioration after sequential minor impacts.
Stein SC	1990	The value of computed tomographic scans in patients with low-risk head injuries. <i>Neurosurgery.</i> 26:638–640.	III	Retrospective review in which all patients had head CT performed and were admitted. They concluded that patients could be discharged if CT and neurologic exam were normal.
Postconcussive Symptoms^{65–68}				
McMordie WR	1988	Twenty-year follow-up of the prevailing opinion on the posttraumatic or postconcussional syndrome. <i>Clin Neuropsychol.</i> 2:198–212.	III	Neurosurgeons and neuropsychologists express divergent views on the postconcussion syndrome in this 1987 survey.
Alves WM	1986	Understanding posttraumatic symptoms after minor head injury. <i>J Head Trauma Rehabil.</i> 1:1–12.	II	Prospective study of 847 adults helps define characteristics of MTBI. 30% of patients had persistent headache at 6 months, yet authors show that for most patients, postconcussive symptoms resolve by 3 months.
Bohnen N	1992	Post-traumatic and emotional symptoms in different subgroups of patients with mild head injury. <i>Brain Inj.</i> 6:481–487.	III	Attempt to distinguish between post-MTBI cognitive symptoms vs. emotional suggests the former are more common in MTBI patient.
Szymanski HV	1992	A review of the post-concussion syndrome. <i>Int J Psychiatry Med.</i> 22:357–375.	III	Review of the American and European literature on PCS.

these guidelines is an injury caused by blunt acceleration/deceleration forces which produces a period of unconsciousness for 20 minutes or less and/or brief retrograde amnesia, a GCS score of 13 to 15, no focal neurologic deficit, no intracranial complications (e.g., seizure activity), and normal CT findings.¹⁻³ MTBI is one of the most common neurologic disorders, having a national prevalence exceeded only by migraine headache,¹ with one trauma system reporting MTBI as representing 80% of all head injury admissions.³

One theory for the neuropathology of MTBI is a predominantly diffuse axonal injury (DAI) caused by shear forces in the brain created by sudden deceleration. Reviews by Alexander¹ and Gennarelli² are supportive of this anatomic injury theory. Oppenheimer⁸ demonstrated microscopic lesions in the brain following head injury where patients died from other causes. The earliest lesions are detectable 15 hours after injury and include microglial cell proliferation, petechial hemorrhages, and other signs of DAI.⁸

B. Diagnosis

Soon after the introduction of CT in 1974, clinical reports established head CT (hCT) as the mainstay in the diagnosis of brain injury and neurotrauma. Although the diagnosis of MTBI is largely clinical by definition, most authors recommend hCT to confirm the absence of focal injury, bleeding, or occult trauma. There is no uniform agreement as to what constitutes a "positive" hCT examination,^{3,9,14} nor is there agreement as to whether all patients with MTBI should routinely undergo hCT. Stein and Ross,¹⁰ from the Cooper Union Trauma Center, justify routine hCT in MTBI based on their experience with 1500 MTBI patients, 17% of whom had positive findings on hCT, and 58 patients required surgery. All patients had met MTBI criteria, and none with normal hCT scans had neurologic deterioration while being observed. Additional support for the use of routine hCT in the evaluation of MTBI comes from a multicenter study by the Western Trauma Association.⁹ This study found that 59/2112 patients (3%) required craniotomy based on hCT findings, despite having a normal neurologic examination. However, all 1170 patients with normal hCT scans did well without surgical intervention. The authors differentiated between abnormal hCT scans (showing chronic abnormalities or minor soft tissue injuries), positive hCT scans (acute abnormalities including contusion, skull fracture), and relevant positive hCT scans (acute injuries to the brain, e.g., bleed). As expected, craniotomy rates were high in the positive and relevant positive groups, but not in the abnormal subcategory.⁹

A prospective, uncontrolled study by Miller et al. of 1382 patients concluded hCT may be utilized selectively in MTBI patients with clinical findings.²² Dunham et al.¹³ concluded similarly in a 1996 retrospective study. There are, however, few additional studies to support that conclusion.

Although most studies on neurotrauma radiography suggest that the presence of skull fractures on either plain film^{7,9,42} (the presence of a skull fracture increased the frequency of a relevant positive hCT by 3×) or hCT^{9,10,13} is associated with a higher

rate of intracranial pathology, the same studies also point out that the absence of skull fractures does not eliminate the risk for significant intracranial injury. Similarly, in the pediatric literature, Lloyd et al.¹⁶ prospectively showed that plain skull radiography was not a reliable predictor of intracranial injury and should be reserved for suspected penetrating injury or when nonaccidental mechanisms are suggested. They also recommended plain skull radiography if the age was less than 2 years.

C. Role of Neuropsychological Testing

In 1968, Oppenheimer⁸ emphasized that, even in so-called minor head injury, anatomic damage to the brain was observed on histopathologic study of sectioned brain tissue beginning at 15 hours postinjury. The diffuse and often occult nature of this brain injury is further documented in a review by Alexander¹ which cites additional histopathologic evidence in both animal and human models. Following this neuroanatomic "validation," reports of significant disability in cognitive function of patients with MTBI and grossly normal neurologic examinations became more frequent in the literature in the 1980s.

Rimel et al.³² reported on 538 MTBI patients with 80% follow-up at 3 months after injury in which 79% had persistent headaches and 59% reported memory problems. These data led to additional investigations of cognitive deficits in the post-MTBI patients, and a number of studies evaluated the role of neuropsychologic testing to define these conditions.⁶⁹ In a prospective study of patients with concussion compared with normal controls, Hugenholtz et al.³⁴ demonstrated significant impairment in patients with concussion who performed tasks specifically requiring attention and information processing, especially during the first month after injury. By the end of the first month, however, reaction times were not significantly different between the two groups. Gentilini et al.³⁷ showed no difference in neuropsychological test results between MTBI patients and nonconcussed case controls performed 1 month after injury. Although most studies demonstrated differences in selective testing between MTBI patients and noninjured controls, compelling evidence for the utility of neuropsychological testing in the acute management of MTBI is lacking. Veltman and colleagues³⁹ performed cognitive screening in the acute setting in 166 MTBI patients during the initial hospitalization using a 20-minute examination, known as the Neurobehavioral Cognitive Status Examination (NCSE).

The NCSE was administered by occupational and speech therapists. Follow-up contacts were made by a registered nurse. They found that 20% of those screened acutely tested positive, and of these, there was a good correlation with outpatient cognitive test abnormalities. The major weakness of this study was that the authors failed to identify predictive factors in the patients with positive screen, and it did not address whether screening all MTBI patients for cognitive deficits in the acute setting was worthwhile.

D. Management

CT scan of the brain is the cornerstone test in the evaluation of traumatic brain injury. The literature generally supports the use of CT scanning for all cases of MTBI in which at least one of the following is present: loss of consciousness; posttraumatic amnesia (PTA); confusion or impaired alertness.^{9–11,28,31} Stein and Ross¹⁰ retrospectively studied patients admitted with GCS of 13 to 15 and loss of consciousness or amnesia and showed that a significant percentage of these patients had abnormalities on CT scan. As expected, an inverse relationship between GCS and CT scan abnormalities was found. For GCS of 13, 14, and 15, the respective percentages of CT abnormalities were 38%, 24%, and 13%. In addition, more than 10% of patients with an initial GCS of 13 required surgery, whereas approximately 3% required surgery when GCS was 15.²⁸ In a study of 3370 patients with initial GCS of 13 to 15, Culotta et al.¹⁷ found that surgery was required for 4.5% of patients with initial GCS of 13 and 0.4% of those with GCS of 15.

Studies of patients who may be categorized as “talk and deteriorate” also support the practice of scanning all MTBI patients.^{58,59,62,63} In this manner, finding significant lesions on CT may allow earlier treatment before deterioration or allow close observation of patients who may otherwise have been discharged home. Although delayed neurologic deterioration is more likely with a lower initial GCS, cases of fatal deterioration from GCS of 15 have been reported.^{55,61}

For those patients with a GCS of 15, no neurologic or cognitive abnormalities, and a normal brain CT, including absence of skull fracture, it is reasonable to discharge them home with a reliable adult. This conclusion is supported by findings from the Western Trauma Association Multicenter Study in which 2766 isolated head injury patients with initial GCS of 13 to 15 were reviewed.⁹ None of the 1170 patients with a normal CT scan required neurosurgical intervention. In addition, they estimated that a 58% decrease in hospital charges would have been realized if patients in this category were discharged home from the emergency department. At Lehigh Valley Hospital, safe discharge criteria have been developed and are used in the form of a Physician Discharge Checklist (see Fig. 1). Dacey et al.³¹ prospectively studied 610 patients with admission GCS of 13 to 15 and also recommended discharge of patients with GCS of 15 and a normal CT scan. Stein and Ross²⁸ found that, in a retrospective study of 658 patients with an initial GCS of 13 to 15, none of the 542 patients with a normal hCT scan showed deterioration or required surgery.

Extending their study to a total of 1538 patients revealed results that corroborated their initial study.¹⁰ None of the 1339 patients with a normal initial CT scan deteriorated neurologically, had delayed CT abnormalities, or required surgery. Livingston et al.¹¹ concurred with this approach based on a prospective review of 111 patients. Davis et al.,⁵⁵ in a retrospective review of 400 children less than 18 years old with initial normal hCT, found that only one patient required surgery, and it was for a delayed subdural hematoma in a patient on warfarin for heart disease.

Neurosurgical Consultation

Rhodes et al.⁶⁰ developed criteria for neurosurgical consultations in which all trauma patients are initially seen by the trauma surgeons. Consultation was not obtained in cases of MTBI deemed to be “low risk.” These patients had a negative hCT scan and possibly minimal symptoms, such as headache or dizziness, but no alteration of consciousness, amnesia, or other more significant findings. Nonurgent consultation was recommended in cases of “moderate-risk” MTBI, including patients with any of the following: alteration of consciousness, amnesia, posttraumatic seizure, prolonged vomiting, and less severe abnormalities on CT scan, such as nondepressed skull fracture, small contusion without mass effect, and minimal edema.

A primary objective for observation of MTBI patients is the immediate detection of any neurologic deterioration. In particular, patients who exhibit a declining neurologic status, including progressive lethargy, pupillary dilatation, or focal neurologic deficit not explained by systemic sources, should have urgent CT scanning and neurosurgical consultation.

E. Postconcussive Symptoms

For some time after MTBI, postconcussive symptoms (PCS) are part of the normal recovery process and not a complication.³⁶ Table 1³⁶ shows that 43.5%, or almost one-half, of MTBI patients experience PCS. At 3 months after injury, these symptoms decreased to 33%, with headaches accounting for many of the symptomatic complaints. Bohnen et al.³⁵ compared concussed patients with PCS to concussed patients without PCS and showed that at 6 months postinjury, tests of selective attention were performed less well in the group with PCS compared with the group without PCS. One consideration for our MTBI protocol would be to identify those patients experiencing PCS in the acute setting and provide them with a 20-minute cognitive screening examination.

For patients admitted with confusion or lethargy which does not clear after several days, it is appropriate to consider rehabilitation medicine evaluation and possible referral to a rehabilitative facility. If symptoms are less debilitating but persistent, then outpatient follow-up may be indicated.

V. SUMMARY

- A. MTBI has defined clinical diagnostic criteria, the hallmark of which is a transient neurologic deficit, along with a diagnostic study confirming the absence of acute skull fracture or pathology.
- B. CT of the brain is the gold standard diagnostic study for MTBI patients and should be performed on all patients sustaining a transient neurologic deficit secondary to trauma. A patient with a normal hCT scan has a 0 to 3% probability for neurologic deterioration, usually in a patient with a GCS of 13 or 14.
- C. Neuropsychological testing may assist in the diagnostic work-up to identify high-risk patients during their acute hospitalization, and/or be used in 1 to 2 months

to evaluate patients with persistent postconcussive symptoms.

- D. The majority of MTBI patients recover completely within 1 month from MTBI. More is necessary to make data-based recommendations on the management and prognosis in the minority who do not recover in that time frame.

VI. FUTURE INVESTIGATION

Many aspects of mild traumatic brain injury (as with all blunt brain injury) remain confusing, particularly with regard to the spectrum of clinical outcomes that may result. The role of psychomotor testing such as cognitive testing, in an attempt to further characterize the injury, needs additional application and study. The common occurrence of MTBI lends itself to meaningful analysis, within both an institutional and a multi-institutional format. Enhanced characterization of the MTBI injury will allow more appropriate utilization of the many subspecialists involved in posttraumatic care, including the trauma surgeon, neurologist, physiatrist, physical, cognitive, and occupational therapists, psychiatrists, and primary care physicians.

As a practice management guideline, the committee objective was to review the available and pertinent literature on MTBI, categorize the data based on strength of study design/findings, and determine whether any recommendations could be made for management of the disorder based on that review.

The committee of coauthors represented a group of experienced trauma surgeons from a variety of hospital settings. The group felt several practical controversies presently existed in the routine management of MTBI, including, for example, the role of CT scan in diagnosis, the necessity for admitting patients sustaining isolated MTBI, and the question of simply, "What is the clinical significance in terms of predicted neurologic sequelae?" for the typical MTBI patient. The literature review addressed these questions, and elaborated on the role and utility of neuropsychiatric testing for MTBI patients.

Although numerous clinical studies are suggested by this practice management guideline, none had been started at the authors' institutions at the time of submitting this manuscript. Whether utilization of neuropsychiatric testing in our trauma population will in fact improve outcomes, reduce costs, or have a significant impact on the incidence of complications is asked by the guideline. We believe that this guideline, because of the effort made to address common clinical issues in the management of MTBI, will be useful in stimulating further study of these very important questions.

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