PRACTICE MANAGEMENT GUIDELINES FOR THE MANAGEMENT OF MILD TRAUMATIC BRAIN INJURY:

The EAST Practice Management Guidelines Work Group

James G. Cushman, MD,[1] Nikhilesh Agarwal, MD,[2]
Timothy C. Fabian, MD,[3] Victor Garcia, MD,[4] Kimberly K. Nagy, MD,[5]
Michael D. Pasquale, MD,[1] Arnold G. Salotto, MD,[1]

[1] Lehigh Valley Hospital, Allentown, PA
[2]Wyntrebrooke S.A., York, PA
[3] University of Tennessee, Memphis, TN
[4] Children's Hospital Medical Center, Cincinnati, OH
[5] Cook County Hospital and Rush University, Chicago, IL

© Copyright 2001 Eastern Association for the Surgery of Trauma

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 score of 13 to 15, no focal neurological deficit, no intracranial complications (e.g. seizure activity), and normal computed tomography (CT) findings.[1-3] In 1995, MTBI ranked third behind only abrasions and contusions as the most common ICD-9 code at our institution. In the pediatric age groups, the diagnosis of concussion, as an isolated injury or as the most significant diagnosis, was the single most reason for admission. 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.[17,28,31,54-68] Finally, the complicated and poorly understood issues surrounding post-traumatic and emotional symptoms in patients with MTBI are discussed in several publications.[69-72]

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.

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. 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 non-contributory or redundant. Subcommittee members for this EAST document followed a similar process which yielded a total of 76 citations.

III. Recommendations

A. Level I

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

B. Level II

© Copyright 2001 Eastern Association for the Surgery of Trauma

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 neurologic deficit secondary to trauma.

2. MTBI patients perform less well on complicated tasks requiring prolonged attention and rapid response times when compared to controls, and this deficit resolves in the majority of patients by one month post-injury. 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 neuropsychologic testing. A variety of tests can be performed, 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. Post-concussive 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 these guidelines is 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 score of 13 to 15, no focal neurological deficit, no intracranial complications (e.g. seizure activity), and normal computed tomography (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] Over 400 adults, and approximately 100 children, are admitted annually with MTBI (unpublished LVH trauma registry data).

The neuropathology of MTBI is felt to be predominantly a diffuse axonal injury (DAI) caused by shear forces in the brain created by sudden deceleration. Oppenheimer[8] 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

© Copyright 2001 Eastern Association for the Surgery of Trauma

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. While the diagnosis of MTBI is largely clinical by definition, most authors recommend a hCT to confirm the absence of focal injury, bleeding, or occult trauma. There is no uniform agreement as to what constitutes a "positive" hCT,[3,9,14] nor is there agreement as to whether all patients with MTBI should routinely undergo hCT. Stein and Ross 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.[10] All patients had met MTBI criteria, and none with normal hCTs 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 exam. However, all 1170 patients with a normal hCT 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 of 1382 patients concluded hCT may be utilized selectively in MTBI patients with clinical findings.[22] Dunham et al. concluded similarly in a 1996 retrospective study.[13] There are, however, few additional studies to support that conclusion.

While 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 3X) 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 non-accidental mechanisms are suggested. He also recommended plain skull radiography if the age was less than 2 years.

C. Role of neuropsychological testing

In 1968, Oppenheimer[8] emphasized that, even in so-called "minor" head injury, anatomical damage to the brain was observed on histopathologic study of sectioned brain tissue beginning at 15 hours post-injury. The diffuse and often occult nature of this brain injury is further documented in a review by Alexander[1] 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. In a prospective study of patients with concussion compared with normal controls, Hugenholtz et al. demonstrated significant impairment in patients with concussion who performed tasks specifically requiring

© Copyright 2001 Eastern Association for the Surgery of Trauma

attention and information processing, especially during the first month after injury.[34] By the end of the first month, however, differences in reaction times were not significant different between the two groups. Gentilini et al.[37] showed no difference in neuropsychological test results between MTBI patients and non-concussed case controls performed one month after injury. While most studies demonstrated differences in selective testing between MTBI patients and non-injured controls, compelling evidence for the utility of neuropsychological testing in the acute management of MTBI is lacking. Veltman and colleagues 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).[39]

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; post-traumatic amnesia (PTA); confusion or impaired alertness.[9-11,28,31] Stein and Ross[10] 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 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.[60,62,66,67] In this manner, finding significant lesions on CT may allow earlier treatment prior to deterioration or allow close observation of patients who may otherwise have been discharged home. Although delayed neurological deterioration is more likely with a lower initial GCS, cases of fatal deterioration from GCS of 15 have been reported.[57,65]

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 the1170 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. 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 Ross[28] 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 showed deterioration or required surgery.

© Copyright 2001 Eastern Association for the Surgery of Trauma

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 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.,[56] 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.[64] 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 and possibly minimal symptoms, such as headache or dizziness, but no alteration of consciousness, amnesia, or other more significant findings. Non-urgent consultation was recommended in cases of "moderate risk" MTBI, including patients with any of the following: alteration of consciousness, amnesia; post-traumatic seizure; prolonged vomiting; and less severe abnormalities on CT scan, such as non-depressed skull fracture, small contusion without mass effect, and minimal edema.

A primary objective for observation of MTBI patients is the immediate detection of any neurological 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.

Concussion in Sports

The decision about when to return to play in sports-related MTBI has been addressed by several authors. The importance of this issue involves the potential ability to prevent more severe brain injuries by identifying the patient at higher risk. Bailes[54] recommended a management scheme for athletes with concussion based on a modification of the Colorado Medical Society Guidelines for the Management of Concussion,[63] guidelines from Cantu,[55] as well as his own experience. Concussion is graded as 1 (mild), 2 (moderate), and 3 (severe), and all recommendations assume the patient is asymptomatic prior to return to play.

Grade 1 (mild): Confusion with no amnesia or loss of consciousness. If confusion clears within 20 to 30 minutes, then allow return to play, otherwise, may return within 1 week of being asymptomatic. After a second mild concussion in the same season, do not return to play for 2 weeks; must be asymptomatic at least 1 week and have a normal CT scan. Terminate season if a third mild concussion occurs. Return next season if asymptomatic.

Grade 2 (moderate): Confusion with amnesia, no loss of consciousness. May return to play only after appropriate evaluation and asymptomatic for 1 week. After a second moderate concussion, may return to play only after asymptomatic for 1 month and CT scan documented to be normal. Terminate season if a third moderate concussion occurs and consider termination of contact sports indefinitely.

© Copyright 2001 Eastern Association for the Surgery of Trauma

Grade 3 (severe): Any loss of consciousness. Urgent transport to hospital for evaluation and CT scan. May return to play after asymptomatic for at least 2 weeks, if loss of consciousness < 1 minute and CT scan is normal. For loss of consciousness > 1 minute, do not return to play for at least 1 month and must be asymptomatic for at least 2 weeks. Terminate season if a second severe concussion occurs and consider termination of contact sports indefinitely.

The objective of any guidelines for concussion in sports is to, first, recognize the more serious brain injuries and second, to prevent repeated concussions within a short period of time. Devastating outcomes may occur even in cases of repetitive mild concussions without loss of consciousness, as shown by Kelly et al.[59] In their report, a 17-year-old football player suffered two mild concussions separated by one week during which time he continued to have headaches but no other complaints. Subsequent to the second injury, his confusion quickly cleared, but shortly thereafter he progressed to brain death due to uncontrolled intracranial hypertension believed to be caused by a severe hyperemic response, based on autopsy findings. Other cases of "second impact syndrome" have also been reported which emphasizes the importance of these guidelines.[61,68]

E. Post-concussive symptoms

For some time after MTBI, post-concussive symptoms (PCS) are part of the normal recovery process and not a complication.[36] Table 1[36] shows that 43.5%, or almost one-half, of MTBI patients experience PCS. At three 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 post-concussive symptoms (PCS) to concussed patients without PCS and showed that at six months post-injury, tests of selective attention were performed less well in the group with PCS group compared to 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 exam.

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 patients with a normal hCT has a 0 to 3% probability for neurologic deterioration, usually in patients with a GCS 13 and 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 post-concussive symptoms.

© Copyright 2001 Eastern Association for the Surgery of Trauma

D. The majority of MTBI patients recover completely within one 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 regards 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, both within an institution and in a multi-institutional format. Enhanced characterization of the MTBI injury will allow more appropriate utilization of the many subspecialists involved in post-traumatic care, including the trauma surgeon, neurologist, physiatrist, physical, cognitive and occupational therapists, psychiatrists, and primary care physicians.

© Copyright 2001 Eastern Association for the Surgery of Trauma

VII. REFERENCES

1. Alexander MP. Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. Neurology 1995;45:1253-1260.
2. Gennarelli TA. Mechanisms and pathophysiology of cerebral concussion. J Head Trauma Rehabil 1986;1:23-29.
3. Levin HS. Outcome from mild head injury. In Narayan RK, Wilberger JE Jr, Povlishock JT, (eds.) Neurotrauma. New York: McGraw-Hill, 1996, pp. 749-754.
4. Stein SC. Outcome from moderate head injury. In Narayan RK, Wilberger JE Jr, Povlishock JT, (eds.) Neurotrauma. New York: McGraw-Hill, 1996, pp. 755-765.
5. Kraus JF, Nourjah P. The epidemiology of mild, uncomplicated brain injury. J Trauma 1988;28:1637-1643.
6. Williams DH, Levin HS, Eisenberg HM. Mild head injury classification. Neurosurgery 1990;27:422-428.
7. Jennett B. The problem of mild head injury. The Practioner 1978;221:77-82.
8. Oppenheimer DR. Microscopic lesions in the brain following head injury. J Neurol Neurosurg Psychiatry 1968;31:299-306.
9. 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 1992;33:385-394.
10. Stein SC, Ross SE. Mild head injury: A plea for routine early CT scanning. J Trauma 1992;33:11-13.
11. Livingston DH, Loder PA, Koziol J, Hunt CD. The use of CT scanning to triage patients requiring admission following minimal head injury. J Trauma 1991;31:483-489.
12. Mohanty SK, Thompson W, Rakower S. Are CT scans for head injury patients always necessary? J Trauma 1991;31:801-805.
13. Dunham CM, Coates S, Cooper C. Compelling evidence for discretionary brain computed tomographic imaging in those patients with mild cognitive impairment after blunt trauma. J Trauma 1996;41:679-686.
14. Marshall LF, Marshall SB, Klauber MR, et al. A new classification of head injury based on computerized tomography. J Neurosurg 1991;75:S14-S20.
15. Camins MB. Radiologic studies and cost-effectiveness in head injuries. Bull Am Coll Surg 1996;81:16-18, 47.
16. Lloyd DA, Carty H, Patterson M, Butcher CK, Roe D. Predictive value of skull radiography for intracranial injury in children with blunt head injury. Lancet 1997;349:821-824.
17. Culotta VP, Sementilli ME, Gerold K, Watts CC. Clinicopathological heterogeneity in the classification of mild head injury. Neurosurgery 1996;38:245-250.
18. Masters SJ, McClean PM, Arcarese JS, et al. Skull x-ray examinations after head trauma. Recommendations by a multidisciplinary panel and validation study. N Engl J Med 1987;316:84-91.
19. Murshid WR. Role of skull radiography in the initial evaluation of minor head injury: A retrospective study. Acta Neurochir 1994;129:11-14.
20. Servadei F, Ciucci G, Pagano F, et al. Skull fracture as a risk factor of intracranial complications in minor head injuries: A prospective CT study in a series of 98 adult patients. J Neurol Neurosurg Psychiatry 1998;51:526-528.

© Copyright 2001 Eastern Association for the Surgery of Trauma

21. Feuerman T, Wackym PA, Gade GF, Becker DP. Value of skull radiography, head computed tomographic scanning and admission for observation in cases of minor head injury. Neurosurgery 1988;22:449-453.
22. Miller EC, Derlet RW, Kinser D. Minor head trauma: Is computed tomography always necessary? Ann Emerg Med 1996;27:290-294.
23. Harad FT, Kerstein MD. Inadequacy of bedside clinical indicators in identifying significant intracranial injury in trauma patients. J Trauma 1992;32:359-363.
24. Jeret JS, Mandell M, Anziska B, et al. Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. Neurosurgery 1993;32:9-16.
25. Borczuk P. Predictors of intracranial injury in patients with mild head trauma. Ann Emerg Med 1995;25:731-736.
26. Taheri PA, Karamanovkian H, Gibbons K, Waldman N, Doerr RJ, Hoover EL. Can patients with minor head injuries be safely discharged home? Arch Surg 1993;128:289-292.
27. Ros SP, Ros MA. Should patients with normal cranial CT scans following minor head injury be hospitalized for observation? Pediatr Emerg Care 1989;5:216-218.
28. Stein SC, Ross SE. The value of computed tomographic scans in patients with low-risk head injuries. Neurosurgery 1990;26:638-640.
29. Stein SC, O'Malley KF, Ross SE. Is routine computed tomography scanning too expensive for mild head injury. Ann Emerg Med 1991;20:1286-1289.
30. Hsiang JN, Yeung T, Yu AL, Poon WS. High-risk head injury. J Neurosurg 1997;87:234-238.
31. Dacey RG Jr, Alves WM, Rimel RW, Winn HR, Jane JA. Neurosurgical complications after apparently minor head injury. Assessment of risk in a series of 610 patients. J Neurosurg 1986;65:203-210.
32. Rimel RW, Giordani B, Barth JT, Boll TJ, Jane JA. Disability caused by minor head injury. Neurosurgery 1981;9:221-228.
33. Barth JT, Macciocchi SN, Giordani B, Rimel R, Jane JA, Boll TJ. Neuropsychological sequelae of minor head injury. Neurosurgery 1983;13:529-533.
34. Hugenholtz H, Stuss DT, Stethem LL, Richard MT. How long does it take to recover from a mild concussion? Neurosurgery 1988;22:853-858.
35. Bohnen N, Jolles J, Twijnstra A. Neuropsychological deficits in patients with persistent symptoms six months after mild head injury. Neurosurgery 1992;30:692-696.
36. 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.
37. Gentilini M, Nichelli P, Schoenhuber R, et al. Neuropsychological evaluation of mild head injury. J Neurol Neurosurg Psychiatry 1985;48:137-140.
38. Beers SR. Cognitive effects of mild head injury in children and adolescents. Neuropsychol Rev 1992;3:281-320.
39. Veltman RH, VanDongen S, Jones S, Buechler CM, Blostein P. Cognitive screening in mild brain injury. J Neurosci Nurs 1993;25:367-371.
40. Parker RS. Neurobehavioral outcome of children's mild traumatic brain injury. Semin Neurol 1994;14:67-73.
41. Cicerone KD. Attention deficits and dual task demands after mild traumatic brain injury. Brain Injury 1996;10:79-89.
42. Fischer RP, Carlson J, Perry JF. Postconcussive hospital observation of alert patients in a primary trauma center. J Trauma 1981;21:920-924.

© Copyright 2001 Eastern Association for the Surgery of Trauma

43. High WM Jr, Levin HS, Gary HE Jr. Recovery of orientation following closed-head injury. J Clin Exp Neuropsychol 1990;12:703-714.
44. Bassett SS, Slater EJ. Neuropsychological function in adolescents sustaining mild closed head injury. J Pediatr Psychol 1990;15:225-236.
45. Haaland KY, Temkin N, Randahl G, Dikmen S. Recovery of simple motor skills after head injury. J Clin Exp Neuropsychol 1994;16:448-456.
46. Reimer W, Van Patten K, Templer DI, Schuyler B, Gross A, Yanovsky A. The neuropsychological spectrum in traumatically head-injured persons. Brain Injury 1995;9:55-60.
47. Coonley-Hoganson R, Sachs N, Desai BT, Whitman S. Sequelae associated with head injuries in patients who were not hospitalized: A follow-up survey. Neurosurgery 1984;14:315-317.
48. Klauber MR, Marshall LF, Luerssen TG, Frankowski R, Tabaddor K, Eisenberg HM. Determinants of head injury mortality: Importance of the low risk patient. Neurosurgery 1989;24:31-36.
49. Wisner DH. Head injury from a general surgeon's perspective. Adv Trauma Crit Care 1993;8:183-216.
50. Rhodes M. Role of the trauma surgeon in neurotrauma. In Narayan RK, Wilberger JE Jr, Povlishock , (eds.) Neurotrauma. New York: McGraw-Hill, 1996, pp. 989-994.
51. Lawler KA, Terregino CA. Guidelines for evaluation and education of adult patients with mild traumatic brain injuries in an acute care hospital setting. J Head Trauma Rehabil 1996;11:18-28.
52. Wilberger JE Jr. Development of guidelines for severe head injury. Bull Am Coll Surg 1997;82:29-33.
53. Practice parameter: The management of concussion in sports (summary statement). Neurology 1997;48:581-585.
54. Bailes JE. Sports-related concussion. St. Louis, Missouri: Quality Medical Publishing. 1999;115-139.
55. Cantu RC. Guidelines for return to contact sports after a cerebral concussion. Physician Sports Med 1986;14:75-83.
56. Davis RL, Hughes M, Gubler KD, Waller PL, Rivara FP. The use of cranial CT scans in the triage of pediatric patients with mild head injury. Pediatrics 1995;95:345-349.
57. Gomez PA, Lobato RD, Ortega JM, De La Cruz J. 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. Br J Neurosurg 1996;10:453-460.
58. Kay T. Neuropsychological treatment of mild traumatic brain injury. J Head Trauma Rehabil 1993;8:74-85.
59. Kelly JP, Nicholas JS, Filley CM, Lillehei KO, Rubinstein D, Kleinschmidt-DeMasters BK. Concussion in sports. Guidelines for the prevention of catastrophic outcome. JAMA 1991;266:2867-2869.
60. Marshall LF, Toole BM, Bowers SA. The National Traumatic Coma Data Bank. Part 2: Patients who talk and deteriorate: Implications for treatment. J Neurosurg 1983;59:285-288.
61. McQuillen JB, McQuillen EN, Morrow P. Trauma, sport, and malignant cerebral edema. Am J Forensic Med Pathol 1988;9:12-15.
62. Reilly PL, Graham DI, Adams JH, Jennett B. Patients with head injury who talk and die. Lancet 1975;2:375-377.

© Copyright 2001 Eastern Association for the Surgery of Trauma

63. Guidelines for the management of concussion in sports. Colorado Medical Society, Denver 1991.
64. Rhodes M, Lester M, Morrow M. Selective neurosurgical consultation for trauma. [Abstract.] J Trauma 1993;35:979.
65. Riesgo P, Piquer J, Botella C, Orozco M, Navarro J, Cabanes J. Delayed extradural hematoma after mild head injury: Report of three cases. Surg Neurol 1997;48:226-231.
66. Rockswold GL, Pheley PJ. Patients who talk and deteriorate. Ann Emerg Med 1993;22:1004-1007.
67. Rockswold GL, Leonard PR, Nagib MG. Analysis of management in thirty-three closed head injury patients who "talked and deteriorated". Neurosurgery 1987;21:51-55.
68. Saunders RL, Harbaugh RE. The second impact in catastrophic contact-sports head trauma. JAMA 1984;252:538-539.
69. McMordie WR. Twenty-year follow-up of the prevailing opinion on the posttraumatic or postconcussional syndrome. Clin Neuropsychol 1988;2:198-212.
70. Alves WM, Coloban ART, O'Leary TJ, Rimel RW, Jane JA. Understanding posttraumatic symptoms after minor head injury. J Head Trauma Rehabil 1986;1:1-12.
71. Bohnen N, Twijnstra A, Jolles J. Post-traumatic and emotional symptoms in different subgroups of patients with mild head injury. Brain Injury 1992;6:481-487.
72. Szymanski HV, Linn R. A review of the postconcussion syndrome. Intl J Psychiatry Med 1992;22:357-375.
73. Kreutzer JS, Marwitz JH, Seel R, Serio CD. Validation of a neurobehavioral functioning inventory for adults with traumatic brain injury. Arch Phys Med Rehabil 1996;77:116-124.
74. Watson MR, Fenton GW, McClelland RJ, Lumsden J, Headley M, Rutherford WH. The post-concussional state: Neurophysiological aspects. Br J Psychiatry 1995;167:514-521.
75. Klein M, Houx PJ, Jolles J. Long-term persisting cognitive sequelae of traumatic brain injury and the effect of age. J Nerv Ment Dis 1996;184:459-467.
76. Macciocchi SN, Barth JT, Alves W, Rimel RW, Jane JA. Neuropsychological functioning and recovery after mild head injury in collegiate athletes. Neurosurgery 1996;39:510-514.

© Copyright 2001 Eastern Association for the Surgery of Trauma

Table 1 Patterns of posttraumatic symptoms at discharge and 3-month follow-up.[36]

Symptom pattern	At Discharge n (%)	At 3 months n (%)
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)**

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.

© Copyright 2001 Eastern Association for the Surgery of Trauma

MILD TRAUMATIC BRAIN INJURY: A LITERATURE REVIEW

First Author	Year	Reference	Data Class	Conclusions
DEFINITION, EPIDEMIOLOGY, AND NATURAL HISTORY OF MTBI[1-8]				
Williams DH	1990	Mild head injury classification. *Neurosurgery 27:422-428*	II	Prospective study of patients with uncomplicated MTBI who had 97% good recovery rate using neuropsychologic measures 6 months post - injury compared to complicated MTBI (84%) and moderate CHI (73%).
Oppenheimer DR	1968	Microscopic lesions in the brain following head injury. *J Neurol Neurosurg Psychiatry 31:299-306*	III	Five cases of MTBI where brains were examined histopathologically, revealing microglial reaction (clusters) suggestive of anatomic injury.
Jennett B	1978	The problem of mild head injury. *The Practitioner 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.
Gennarelli TA	1986	Mechanisms and pathophysiology of cerebral concussion. *J Head Trauma Rehabil 1:23-29*	III	Review article.
Krauss JF	1988	The epidemiology of mild, uncomplicated brain injury. *J Trauma 28:1637-1643*	III	Retrospective 1-year review of MHI; 80% of diagnoses were concussion; cost $6 million in 1981 dollars.
Alexander MP	1995	Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. *Neurology 45:1253-1260*	III	Review article, literature review.
Levin HS	1996	Outcome from mild head injury. In Narayan RK, et al, (eds.) Neurotrauma 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.) Neurotrauma NY:McGraw-Hill	III	Book Chapter - Outcome From Moderate Head Injury

© Copyright 2001 Eastern Association for the Surgery of Trauma

First Author	Year	Reference	Data Class	Conclusions
DIAGNOSIS[9-31]				
Masters SJ	1987	Skull x-ray examinations after head trauma. *N Engl J Med 316:84-91*	II	Prospective, multi -institutional study. May omit plain skull films in low-risk patients.
Livingston DH	1991	The use of CT scanning to triage patients requiring admission following minimal head injury. *J Trauma 31:483-489*	II	Prospective: 63% phone follow up. All underwent head CT. >80% discharged.
Jeret JS	1993	Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. *Neurosurgery 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.
Miller EC	1996	Minor head trauma: is computed tomography always necessary? *Ann Emerg Med 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.
Hsiang JN	1997	High-risk mild head injury. *J Neurosurg 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%.
Lloyd DA	1997	Predictive value of skull radiography for intracranial injury in children with blunt head injury. *Lancet 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.
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. *J Neurol Neurosurg Psychiatry 51:526-528*	II	Prospective, (n=98) but limited study. Presence of skull fracture predicts need for neurosurgical intervention.
Dacey RG Jr	1986	Neurosurgical complications after apparently minor head injury. *J Neurosurg 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.
Feuerman T	1988	Value of skull radiography, head computed tomographic scanning and admission for observation in cases of minor head injury. *Neurosurgery 22:449-453*	III	Retrospective study which suggests patients may be discharged to home if initial brain CT is negative.

© Copyright 2001 Eastern Association for the Surgery of Trauma

First Author	Year	Reference	Data Class	Conclusions
Ross SP	1989	Should patients with normal cranial CT scans following minor head injury be hospitalized for observation? *Pediatr Emerg Care 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. *Neurosurgery 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.
Marshall LF	1991	A new classification of head injury based on computerized tomography. *J Neurosurg 75:S14-S20*	III	Nice paper on reading head CTs for trauma.
Mohanty SK	1991	Are CT scans for head injury patients always necessary? *J Trauma 31:801-805*	III	Retrospective study presented at 1990 AAST. 12/348 head CTs abnormal in patients without focal neuro deficits. Critical of routine head CTs.
Harad FT	1992	Inadequacy of bedside clinical indicators in identifying significant intracranial injury in trauma patients. *J Trauma 32:359-363*	III	In GCS $\geq$13, abnormal CT rate 18%. Neurosurgical intervention in 4%.
Stein SC	1991	Is routine computed tomography scanning too expensive for mild head injury. *Ann Emerg Med 20:1286-1289*	III	The study is limited as it only compared cost of CT scan vs. admission.
Shackford SR	1992	The clinical utility of computed tomographic scanning and neurologic examination in the management of patients with minor head injury. *J Trauma 33:385-394*	III	This Western Trauma Association multicenter review concluded it was safe to discharge patients to home with a normal neuro exam and normal head CT.
Stein SC	1992	Mild head injury: A plea for routine early CT scanning. *J Trauma 33:11-13*	III	Retrospective: all patients admitted. No home follow -up.
Taheri PA	1993	Can patients with minor head injuries can be safely discharged home? *Arch Surg 128:289-292*	III	Yes, patients with minor head injuries can be safely discharged home.
Murshid WR	1994	Role of skull radiography in the initial evaluation of minor head injury. A retrospective study. *Acta Neurochir 129:11-14*	III	Midline of skull fracture in GCS 13 -15 population 11%. Neurosurgical intervention in 3%.

© Copyright 2001 Eastern Association for the Surgery of Trauma

First Author	Year	Reference	Data Class	Conclusions
Borczuk P	1995	Predictors of intracranial injury in patients with mild head trauma. *Ann Emerg Med 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.
Camins MB	1996	Radiologic studies and cost -effectiveness in head injuries. *Bull Am Coll Surg 81:16-18, 47*	III	The American College of Surgeons opinion on the role of various radiographic modalities in CHI.
Culotta VP	1996	Clinicopathological heterogeneity in the classification of mild head injury. *Neurosurgery 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.
Dunham CM	1996	Compelling evidence for discretionary brain computed tomographic imaging in those patients with mild cognitive impairment after blunt trauma. *J Trauma 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.

© Copyright 2001 Eastern Association for the Surgery of Trauma

First Author	Year	Reference	Data Class	Conclusions
ROLE OF NEUROPSYCHIATRIC TESTING[31-46,73]				
Hugenholtz H	1988	How long does it take to recover from a mild concussion? *Neurosurgery 22:853-858*	I	In tests of attention and information processing, 22 concussed patients were significantly slower than controls at 1 & 3 months. Improvement was seen, and by the 1st month were equal. Excellent reference.
Rimel RW	1981	Disability caused by minor head injury. *Neurosurgery 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.
Bassett SS	1990	Neuropsychological function in adolescents sustaining mild closed head injury. *J Pediatr Psychol 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.
High WM Jr	1990	Recovery of orientation following closed -head injury. *J Clin Exp Neuropsychol 12:703-714*	II	84 patients followed after CHI (20% had GCS - 13-15). Return of orientation occurred first to person, then place, and finally time.
Bohnen N	1992	Neuropsychological deficits in patients with persistent symptoms six months after mild head injury. *Neurosurgery 30:692-696*	II	Patients with post -concussive symptoms 6 months after injury perform less well on tests of attention than injured patients witho ut PCS and control. Small numbers, questionable methods.
Haaland KY	1994	Recovery of simple motor skills after head injury *J Clin Exp Neuropsychol 16:448-456*	II	40 patients with acute head injury (75% had GCS=11-15) were compared to 88 controls. Finger -tapping and grip strength were measured 1 month and 1 year post -injury. 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.
Kreutzer JS	1996	Validation of a neurobehavioral functioning inventory for adults with traumatic brain injury. *Arch Phys Med Rehabil 77:116-124*	II	Outpatient neuropsychologic testing s/p MTBI with objective scoring systems correlated with patient perception of post -injury neurologic problems. These may be used clinically to assist in patient treatment and follow-up.

© Copyright 2001 Eastern Association for the Surgery of Trauma

First Author	Year	Reference	Data Class	Conclusions
Fischer FP Jr	1981	Postconcussive hospital observation of alert patients in a primary trauma center. *J Trauma 21:920-924*	III	Retrospective 6-month review. Only skull fracture (43/333) associated with need for admit/neurosurgical sequelae.
Barth JT	1983	Neuropsychological sequelae of minor head injury. *Neurosurgery 13:529-533*	III	71/1248 patients followed-up at 3 months s/p minor head injury. "Significant %" had cognitive impairment as determined on 3 different tests.
Gentilini M	1985	Neuropsychological evaluation of mild head injury. *J Neurol Neurosurg Psychiatry 48:137-140*	III	Neuropsychologic testing in 50 MTBI patients was equivalent to 50 core-controls at one month.
Beers SR	1992	Cognitive effects of mild head injury in children and adolescents. *Neuropsychol Rev 3:281-320*	III	A doctoral thesis summarizing a comprehensive re view of mild head injury studies in children.
Veltman RH	1993	Cognitive screening in mild brain injury. *J Neurosci Nurs 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. *Semin Neurol 14:67-73*	III	Case review format of various neuropsychologic sequelae in MTBI.
Reimer W	1995	The neuropsychological spectrum in traumatically head-injured persons. *Brain Injury 9:55-60*	III	125 patients with traumatic brain injury were a dministered 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.
Cicerone KD	1996	Attention deficits and dual task demand s after mild traumatic brain injury. *Brain Injury 10:79-89*	III	15 patients referred 18 months after MTBI had slower processing speeds than a community control group.
Chambers J	1996	Mild traumatic brain injuries in low -risk trauma patients. *J Trauma 41:976-980*	III	Phone survey, small numbers. Found that 11% of blunt trauma victims had PCS at 2 months.

© Copyright 2001 Eastern Association for the Surgery of Trauma

First Author	Year	Reference	Data Class	Conclusions
MANAGEMENT[17,28,47-68,74-76]				
Marshall LF	1983	The National Traumatic Coma Data Bank. Part 2: Patients who talk and deteriorate: Implications for treatment. *J Neurosurg 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.
Dacey RG Jr	1986	Neurosurgical complications after apparently minor head injury: Assessment of risk in a series of 610 patients. *J Neurosurg 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.
Klauber MR	1989	Determinants of head injury mortality: Importance of the low risk patient. *Neurosurgery 24:31-36*	II	Differences in outcome of patients with CHI comparing hospitals lie in the care of the "low-risk" patients.
Watson MR	1995	The post-concussional state: Neurophysiological aspects. *Br J Psychol 167:514-521*	II	72% of MTBI patients had resolution of post-concussion symptoms at 6 weeks. Those that don't should have neuroelectrophysiologic studies done, according to these authors.
Reilly PL	1975	Patients with head injury who talk and die. *Lancet 2:375-377*	III	Case review of 66 patients who died after initially talking after injury. 75% associated with hematomas.
Coonley-Hoganson R	1984	Sequelae associated with head injuries in patients who were not hospitalized: A follow-up survey. *Neurosurgery 14:315-317*	III	Retrospective/telephone survey. Methodologic weaknesses. Headaches, dizziness, and drowsiness in 27%, 11%, & 9% at 1 week. 2/3 followed head sheet instructions.
Saunders RL	1984	The second impact in catastrophic contact-sports head trauma. *JAMA 252:538-539*	III	Case report of neurologic deterioration after sequential minor impacts.
Cantu RC	1986	Guidelines for return to contact sports after a cerebral concussion. *Physician Sports Med 14:75-83*	III	Review article: guidelines for management of sports-related concussion.
Rockswold GL	1987	Analysis of management in thirty-three closed head injury patients who "talked and deteriorated." *Neurosurgery, 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.
McQuillen JB	1988	Trauma, sport, and malignant cerebral edema. *Am J Forensic Med Pathol 9:12-15*	III	Review of pathophysiology of cerebral edema after sequential concussion in sports.

© Copyright 2001 Eastern Association for the Surgery of Trauma

Stein SC	1990	The value of computed tomographic scans in patients with low -risk head injuries. *Neurosurgery 26:638-640*	III	Retrospective review in which all patients had a head CT performed and were admitted. They concluded that patients could be discharged if CT and neurologic exam were normal.
First Author	**Year**	**Reference**	**Data Class**	**Conclusions**
Anonymous	1991	Guidelines for the management of concussion in sports. Colorado Medical Society, Denver	III	Guidelines recommended for the management of concussion in sports.
Kelly JP	1991	Concussion in sports: Guidelines for the prevention of catastrophic outcome. *JAMA 266:2867-2869*	III	Case report of neurological deterioration after repeated mild head trauma and guidelines for sports.
Kay T	1993	Neuropsychological treatment of mild traumatic brain injury. *J Head Trauma Rehabil 8:74-85*	III	Guidelines presented for neuropsychological treatment of MTBI.
Rhodes M	1993	Selective neurosurgical consultation for trauma. *J Trauma 35:979*	III	Stratified neurosurgical consultation based on clinical and CT findings into URGENT, NONURGENT, and NO CONSULTATION categories.
Rockswold GL	1993	Patients who talk and deteriorate. *Ann Emerg Med 22:1004-1007*	III	Review of subject of patients who deteriorate neurologically.
Wisner DH	1993	Head injury from a general surgeon's perspective. *Adv Trauma Crit Care 8:183-216*	III	Literature review and synopsis from a prominent academic traumatologist.
Davis RL	1995	The use of cranial CT scans in the triage of pediatric patients with mild head injury. *Pediatrics 95:345-349*	III	Retrospective review: safe to discharge patients with GCS of 15 and normal head CT.
Rhodes M	1996	Role of the trauma surgeon in neurotrauma. In Narayan RK, et al, (eds.) Neurotrauma NY: McGraw-Hill	III	Guidelines for when to consult with neurosurgeons, as developed by trauma and neurosurgeons at Lehigh Valley Hospital in Allentown, Pennsylvania.
Culotta VP	1996	Clinicopathologic heterogeneity in the classification of mild head injury. *Neurosurgery 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.
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. *Br J Neurosurg 10:453-460*	III	Retrospective review: significantly higher incidence of surgi cal intervention and neurologic complications with GCS 13 -14.

© Copyright 2001 Eastern Association for the Surgery of Trauma

Klein M	1996	Long-term persisting cognitive sequelae of traumatic brain injury and the effect of age. *J Nerv Ment Dis 184:459-467*	III	Descriptive study with methodological problems.

First Author	**Year**	**Reference**	**Data Class**	**Conclusions**
Lawler KA	1996	Guidelines for evaluation and education of adult patients with mild traumatic brain injuries in an acute care hospital setting. *J Head Trauma Rehabil 11:18-28*	III	Literature-based recommendations on the evaluation and education of MTBI patients prior to discharge.
Macciocchi SN	1996	Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery 39:510-514*	III	10 NCAA athletic departments in the study. Young health MTBI patients generally recover rapidly and completely from MTBI.
Anonymous	1997	Practice parameter: The management of concussion in sports. *Neurology 48:581-585 (see also p 575-580)*	III	The American Academy of Neurology's Report of the Quality Standards Committee follows EAST's evidence -based format).
Riesgo P	1997	Delayed extradural hematoma after mild head injury: report of three cases. *Surg Neurol 48:226-231*	III	Case reports of dela yed extradural hematomas.
Wilberger JE Jr	1997	Development of guidelines for severe head injury. *Bull Am Coll Surg 82:29-33*	III	The American College of Surgeons Advisory Council data -based guidelines for severe CHI.
Bailes JE	1999	Sports-related concussion. *Quality Medical Publishing, 115-139*	III	Book chapter - Diagnosis and management of head injury.

© Copyright 2001 Eastern Association for the Surgery of Trauma

First Author	Year	Reference	Data Class	Conclusions
POST-CONCUSSIVE SYMPTOMS[69-72]				
Alves WM	1986	Understanding posttraumatic symptoms after minor head injury. *J Head Trauma Rehabil 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 patien ts, post-concussive symptoms resolve by 3 months.
McMordie WR	1988	Twenty-year follow-up of the prevailing opinion on the posttraumatic or postconcussional syndrome. *Clin Neuropsychol 2:198-212*	III	Neurosurgeons and neuropsychologists express divergent views on the post-concussion syndrome in this 1987 survey.
Bohnen N	1992	Post-traumatic and emotional symptoms in different subgroups of patients with mild head injury. *Brain Injury 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. *Intl J Psychiatry Med 22:357-375*	III	Review of the American and European literature on PCS.

© Copyright 2001 Eastern Association for the Surgery of Trauma