Controversies in the evaluation and management of minor blunt head trauma in children

David Schnadower, Hector Vazquez, June Lee, Peter Dayan and Cindy Ganis Roskind

Purpose of review

We present data from recently conducted research regarding controversial aspects of the evaluation and management of children with minor blunt head trauma.

Recent findings

Clinicians frequently but at times indiscriminately perform computed tomography scans for children with minor blunt head trauma resulting in potentially harmful radiation exposure. Recent guidelines recognize the limited but increasing data available to make strong recommendations regarding appropriate neuroimaging decisions. Investigators have derived and validated clinical prediction models to accurately identify patients with substantial traumatic brain injury, though no clear definitive rule exists. Children younger than 2 years appear to have a higher risk of intracranial injury following minor head trauma. These patients can be difficult to assess, with the evidence suggesting the need for a more conservative approach to diagnostic imaging. We present current and accepted definitions of concussion along with risk factors and treatment for postconcussion syndrome. Current return-to-play guidelines suggest that athletes who have sustained concussion should not resume play until symptoms have resolved because of the possibility, though rare, of second impact syndrome.

Summary

Research in the management of children with minor head trauma is actively evolving. We present a review of recent developments that can influence current clinical practice.

Keywords

brain concussion, craniocerebral trauma, head injuries (closed), postconcussion syndrome, skull fractures

Curr Opin Pediatr 19:258–264. © 2007 Lippincott Williams & Wilkins.

Division of Pediatric Emergency Medicine, Morgan Stanley Children's Hospital of New York Presbyterian, Columbia University College of Physicians & Surgeons, New York, USA

Correspondence to David Schnadower MD MPH, 622 West 168th Street, PH-137, New York, NY 10032, USA
Tel: +1 212 305 9825; e-mail: ds2194@columbia.edu

Current Opinion in Pediatrics 2007, 19:258–264

Abbreviations

CI	confidence interval
CT	computed tomography
ED	Emergency Department
GCS	Glasgow Coma Scale
LOC	loss of consciousness
RR	relative risk
SIS	second impact syndrome
TBI	traumatic brain injury

© 2007 Lippincott Williams & Wilkins
1040-8703

Introduction

Traumatic brain injury (TBI) from unintentional blunt trauma is the leading cause of death and disability among US children younger than 19 years of age, resulting in 7440 deaths, 642 000 emergency department (ED) visits and 65 000 hospitalizations annually [1]. Approximately 50–80% of patients presenting to the ED with head trauma have minor head trauma [2,3], which reasonably includes those with a Glasgow Coma Scale (GCS) or modified GCS of 14–15 but is defined differently among studies [3,4,5•,6,7].

Although computed tomography (CT) scans are the diagnostic test of choice to evaluate children with minor head trauma, clinicians vary widely in their use, ranging from less than 5% to upwards of 50% of patients presenting to the ED [1,5•,8–11]. In the US, less than 4–8% of head CT scans performed in children with minor head trauma display any evidence of TBI [2,3,7,12] and overall, less than 0.5% of children with minor blunt head trauma require neurosurgical intervention [5•,7,13]. There is, therefore, valid concern that CT scans are overused and that they may be detecting a number of clinically inconsequential findings that require no intervention [3,14,15].

Much controversy exists in the management of children with minor blunt head trauma, particularly regarding the use of CT with its associated radiation risks, the assessment of the risk of TBI (particularly in children under 2 years of age) and the diagnosis and management of concussions and postconcussion syndrome. Our objective is to provide a review of the recent literature for each of these topics.

Clinical question: in children exposed to a single head CT, what are the risks of adverse effects from radiation?

There is concern that CT scans expose children to potentially harmful radiation doses. Children are considerably more sensitive to radiation than adults due to rapidly dividing cells associated with growth. Since children have longer life expectancies, they have more opportunity to express radiation damage, particularly cancer. Although 'as low as reasonably achievable' radiation dose guidelines have been advocated for pediatric CT, there is concern that they are not applied universally and that there may be widespread underestimation of radiation risks among patients and healthcare professionals [16–18]. A pediatric head CT with adjusted settings delivers 30 millisieverts to the brain, whereas a nonadjusted adult-based protocol delivers twice as much radiation. A single head CT exposes children to 200–600 times as much radiation as typical postero-anterior and lateral chest radiographs [19].

Brenner *et al.* [20,21] used data from radiation exposure of atomic bomb survivors in Japan to estimate that the lifetime attributable risk for fatal cancer from one head CT ranges from one per 2000 scans for young infants to one per 5000 scans in older children. In addition, a recent population-based cohort study from Sweden noted an association between early childhood exposure to radiation in doses similar to a single head CT and poor cognitive performance and academic achievement at 18 years of age [22]. These potential risks underscore the importance of using CT scans selectively, implementing 'as low as reasonably achievable' radiation dose guidelines, and promoting research in alternative imaging technologies.

Clinical question: in children presenting to the ED with acute, minor blunt head trauma, are there clinical predictors and prediction models that accurately identify patients with substantial traumatic brain injury?

In a metaanalysis of 16 studies that included a total of 22 420 children with minor head trauma, Dunning *et al.* [4] described individual risk factors associated with the presence of TBI. In his study, the presence of skull fracture [relative risk (RR) 6.1, 95% confidence interval (CI) 3.4–11.2], focal neurological signs (RR 9.4, 95% CI 2.9–30.8), loss of consciousness (LOC) (RR 2.2, 95% CI 1.2–4.2) and GCS less than 15 (RR 5.5, 95% CI 1.6–19) was statistically associated with TBI, whereas headache (RR 1.0, 95% CI 0.6–1.7) and vomiting (0.88, 95% CI 0.7–1.2) were not. The presence of seizures (RR 2.8, 95% CI 0.9–9) showed a trend toward an association with TBI.

As these individual risk factors do not predict the presence of TBI with sufficient sensitivity, investigators have sought to derive and validate clinical prediction models that use combinations of factors to accurately identify those with TBI and/or clinically important head injury (Table 1). Unfortunately, each study in Table 1 used different inclusion criteria, making comparisons among them difficult. Additionally, the investigators frequently did not confine the study populations to those with minor head trauma. The studies performed by Haydel and Shembekar [7] and Oman *et al.* [14] are validation studies of clinical prediction rules derived in adults and children. Both rules have high sensitivities but low specificities, making it unclear if implementation of the rules may actually increase the use of CT scans if applied to less symptomatic populations (who would not meet each study's inclusion criteria). Palchak *et al.*'s [3] single-center derivation of a clinical decision rule resulted in prediction models that would identify those either with a positive CT or who required acute intervention. Dunning *et al.* [5•] recently published the largest study to derive a clinical decision rule to identify pediatric patients at high risk of clinically significant head injury (defined as death, need for neurosurgical intervention or abnormality on a CT scan). The prediction rule is complex and, like other models, includes predictors that are difficult to assess in young patients. If applied, this rule may theoretically increase the number of CT scans performed in the UK (from 3% to 13%). This study also included patients with very minor head trauma and CTs were performed very infrequently. As for all newly derived prediction models, validation must occur prior to clinical use [23].

Clinical question: in children younger than 2 years presenting to the ED with minor blunt head trauma, are there clinical predictors that accurately identify patients with traumatic brain injury or skull fracture?

Although the data presented above include children younger than 2 years, this group is often considered independently. Signs and symptoms are difficult to assess, particularly in the very young, and the threshold for obtaining imaging studies has traditionally been lower in this population [2,24–26]. The incidence of skull fractures and intracranial injury following minor head trauma seems to be higher in this age group. Skull fractures occur in 2% of all children following minor head trauma [5•] whereas the incidence is as high as 11% in children under 2 years of age [27]. Furthermore, 15–30% of patients under 2 years with a skull fracture will have TBI and the presence of skull fracture is a clear predictor of TBI [3,14,26,28].

An American Academy of Pediatrics expert panel issued consensus guidelines specific for the management of minor head trauma in children younger than 2 years [26]. They recommended that clinicians perform CT scans on patients at high risk for TBI, defined as those with any of the following: signs of depressed or basilar skull fracture, acute skull fracture, altered mental status,

Table 1 Recent clinical decision rules to predict substantial head injuries in children

Author	Sample size	Inclusion criteria	Outcome(s)	Clinical decision model to predict outcome(s)	Sensitivity (95% CI)	Specificity (95% CI)
Haydel, 2003 [7] (Validation)	14/175 (Outcome/N)	Children 5–17 yrs, GCS = 15, normal neurological exam, and + LOC	Any acute traumatic intracranial lesion on CT	Headache, emesis, intoxication, trauma above the clavicles, seizure or short-term memory deficit	100% (73–100%)	26% (19–33%)
Oman, 2006 [14] (Validation*)	138/1666 (Outcome/N)	All children ≤18 yrs with blunt head trauma who had head CT	Clinically important intracranial injury based on expert consensus (does not include all acute traumatic intracranial CT findings)	Significant skull fracture, altered level of alertness, neurological deficit, persistent vomiting, scalp hematoma, abnormal behavior, coagulopathy	98.6% (95–99.8%)	15% (13–17%)
Palchak, 2003 [3] (Derivation)	N = 2043, 1271 of whom had CT	Children < 18 yrs with nontrivial blunt head trauma	TBI requiring acute intervention (105/2043 patients had this outcome) Any TBI on CT (98/1271 who had CT had this outcome)	Abnormal mental status, clinical signs of skull fracture, history of vomiting, scalp hematoma in children ≤ 2 yrs of age, headache	100% (97–100%) 99.0% (94–100%)	43% (41–45%) 26% (23–28%)
Dunning, 2006 [5•] (Derivation)	281/22,772 (Outcome/N)	All children <16 yrs with any severity of head injury	Clinically significant head injury (death, need for neurosurgical intervention or abnormality on CT) Note: only 766 (3.3%) had CTs performed	History: Witnessed LOC>5 min, amnesia > 5 min, drowsiness, ≥3 vomits, suspicion of non-accidental injury, seizure. Exam: GCS<14 or GCS<15 if <1 year, suspicion of penetrating or depressed skull injury or tense fontanelle, signs of basilar skull fracture, focal neurological exam, or bruise, swelling or laceration>5 cm if <1 year of age. Mechanism: high speed traffic accident, fall > 3 meters, high speed injury from an object.	98.6% (96–99.6%)	87% (86.5–87.4%)

*Sample is a subset of a larger population of adults and children from whom decision rule was derived; CI, confidence interval; CT, computed tomography; GCS, Glasgow Coma Scale; LOC, loss of consciousness; TBI, traumatic brain injury.

focal neurological findings, bulging fontanel, vomiting more than five times or for more than 6 h after injury, or LOC for 1 min or longer. The panel recommended either imaging or observation for 4–6 h after the injury for patients at intermediate risk of TBI, which included patients vomiting three to four times, LOC for less than 1 min, history of lethargy or irritability resolved at the time of examination, nonacute skull fracture (>24–48 h), higher force mechanism, scalp hematoma (particularly if large or in a nonfrontal location), fall onto hard surface, unwitnessed trauma with possibility of significant mechanism, and head injury with no clear history of trauma [26]. Finally, the panel recommended discharge under the care of a reliable caretaker for patients at low risk for TBI, defined as those that are asymptomatic 2 h after injury, had a low energy mechanism of injury (e.g. fall < 3 ft) and are older than 12 months [26]. In these balanced recommendations the authors recognize the relative lack of data, particularly regarding variables such as height of fall, timing of loss of consciousness and the size of scalp hematomas.

Data from Greenes and Schutzman [27] provide useful information regarding which young patients with scalp swelling are at higher risk of skull fracture and TBI. In a prospective cohort study of 422 infants younger than 2 years with minor head trauma and isolated scalp swelling, they found that the size and location of a scalp hematoma were predictive of skull fracture. The odds ratio (OR) for the presence of skull fracture in patients with a large scalp hematoma (defined as 'large, boggy area') was 27.0 (95% CI 8–90.5). Hematomas in the parietal and temporal areas were more likely to be associated with skull fractures, with ORs of 38.2 (95% CI 12.5–115.7) and 16 (95% CI 3.1–85.2), respectively [27].

There is controversy regarding the role of skull radiographs as a screening tool for children under 2 years of age with possible skull fracture, as negative skull radiographs may obviate the need to perform a CT in a patient at low risk for TBI [25,29–31]. Skull radiographs have significant advantages as they require no sedation and expose children to 100 times less radiation than head CT [19,32]. TBI, however, can occur in the absence of skull fractures [2,26,33,34], and skull radiographs do not provide information on intracranial anatomy. In Dunning *et al.*'s metaanalysis [4], skull radiographs were found to have a sensitivity of 59% and a specificity of 88% in detecting TBI. There is also concern that skull radiographs are often misread by nonradiologists [34]. Chung *et al.* [35] noted that the sensitivity of skull radiographs read by pediatric emergency medicine attendings for the detection of skull fractures was 76% (±15%) and the specificity was 84% (±14%) compared with interpretations by a pediatric radiologist [35]. When choosing an imaging modality, clinicians must therefore consider the risk of

TBI, the availability of a CT scanner, the expertise at imaging interpretation and the need for sedation. We recommend that clinicians perform a CT for children younger than 2 years with an acute skull fracture on skull radiographs because the presence of skull fracture significantly increases the likelihood of TBI.

Clinical questions: what is the risk of, and what are the risk factors for, postconcussion syndrome? Does postconcussion syndrome result in long-term sequelae?

The term concussion is usually applied to a subgroup of patients with minor head trauma, although multiple definitions have been proposed. The American Academy of Neurology [36] defines concussion as 'a trauma induced alteration in mental status that may or may not involve loss of consciousness' but typically includes confusion and amnesia. In 2004, the Second International Conference on Concussion in Sport [37] defined sports concussion as a complex physiological process following head trauma resulting in rapid onset of short-lived functional impairment, though in some cases associated with prolonged postconcussive symptoms, and typically associated with a normal CT scan. MRI, single photon emission CT and PET scans may demonstrate subtle abnormalities such as small areas of hemorrhage, contusion, or axonal injury [38–41]. Imaging findings do not seem to be closely associated with functional outcome [40,41].

Postconcussion syndrome has been defined as the persistence beyond 7–10 days of the injury of any one of the following symptoms not present before injury: headaches, easy fatigability, sleep disturbances, dizziness, irritability, aggressiveness, anxiety, depression, missed work, relationship troubles, personality change, trouble with simple math, and trouble with short-term memory [37,42]. The concept of postconcussion syndrome as a comprehensive entity is somewhat controversial, with limited data available and substantial overlap of symptoms with psychiatric illnesses [43–46]. Nevertheless, the presence of postconcussion syndrome has been reported in as many as 13–35% of children 6 weeks to 3 months following 'minor' head trauma, defined in most studies as GCS 13–15 and symptoms such as amnesia, LOC, or vomiting [47–49].

Researchers have sought to identify risk factors for the development of postconcussion syndrome with the hope that early identification of those at risk may improve outcomes. In adults, female gender, prior axis I and axis II disorders, severe balance deficits, severe pain, poor performance on neurocognitive tests, presence of dizziness, headache, skull fracture or a serum protein S-100B above 50 μg/l on presentation to the ED have been found to be associated with a higher risk of postconcussion syndrome [50–53]. Pediatric data are limited. Studies suggest that postconcussion syndrome is more likely to develop in patients with low GCS score and anxiety upon presentation and in those with a history of prior head injuries, learning disabilities and neuropsychiatric disturbances [47–49,54,55]. Levels of biochemical serum markers such as S-100B, c-tau, and neuron-specific enolase have not been clearly shown to correlate with the onset, duration, or severity of postconcussion syndrome; however, they may be useful when considered in conjunction with other clinical factors [38,56–60].

Most children with postconcussion syndrome will have resolution of symptoms by 3 months after injury. Some children, however, may have more subtle lasting difficulties, such as behavioral and school problems [48,61,62]. Anderson *et al.* [63,64•] prospectively demonstrated no significant differences on cognitive measures or behavioral function 30 months after injury, but found some deficit in story recall and verbal fluency in children with mild head injury as compared with matched noninjured controls.

Clinical question: in children with postconcussion syndrome, are medications or psychosocial–behavioral measures effective in decreasing the severity of symptoms?

Early diagnosis, education and implementation of multidisciplinary intervention strategies may help alleviate disabilities from postconcussion syndrome. Postconcussive symptoms have been shown to decrease after an educational intervention in the form of an information booklet [48] and early supportive care [65]. In a small randomized controlled trial bed rest was not shown to improve outcome [66]. Some patients may warrant referrals to specialty services such as physical therapy for postural imbalances, neurology for headaches, behavioral therapy for attention/hyperactivity symptoms, or concussion-specific follow-up clinics [67].

Few data exist to evaluate the potential benefit of pharmacological treatment of postconcussion syndrome. Symptomatic management with nonsteroidal antiinflammatory medications for postconcussion headache is encouraged, with consideration for therapies directed at tension-type headache and migraine [68]. Newer modalities such as occipital nerve block are being studied [69] and treatments previously aimed at severe brain injury, such as corticosteroids, and hyperbaric oxygen therapy, are potential interventions for future investigation [70]. Antidepressants, anxiolytics, methylphenidate alone or in combination with psychotherapy, and cytidine 5′ diphosphocholine (CDP) have also been used though large randomized controlled trials have not been performed [71–73]. Although the effectiveness of existing therapies is unclear, ED clinicians should provide anticipatory guidance

regarding postconcussive syndrome and recommend pediatric follow-up for any child who sustains a concussion. Follow-up will allow clinicians and parents to explore potential behavioral and pharmacological therapies if symptoms persist.

Clinical question: in children with concussion, are published return-to-play guidelines effective in preventing second-impact syndrome?

Second-impact syndrome (SIS) refers to the vulnerability of the brain following repeated trauma during the postconcussive period. It is hypothesized that loss of cerebrovascular autoregulation can lead to vascular congestion, swelling, and transtentorial brainstem herniation when re-injury occurs while still symptomatic from a prior concussion [74]. The existence of SIS is controversial and has only been described in case reports. McCrory and Berkovic [75] analyzed 17 published cases and found that 11 out of 17 cases had no witnessed second impact, and no cases could be classified as 'definite SIS'. Nevertheless, various return-to-play guidelines have been advocated to prevent SIS and repeated damage from multiple concussions [36,76]. It is generally accepted that an athlete must not return to play until all concussive symptoms have completely resolved, and a similar conservative approach is uniformly recommended in children and adolescents [76]. One of the more recent and more accepted return-to-play guidelines comes from the Second International Conference on Concussion. They propose [37] that initially there should be no activity but, once an athlete is asymptomatic, they may proceed to light aerobic exercise and then to a stepwise, gradual progression to full game play. A substantial percentage of patients with concussive symptoms seen by medical providers do not receive specific follow-up instructions [77–79]. Return-to-play guidelines, though lacking high-level evidence, have become standard of care, and should be conveyed to ED patients.

Conclusions

Although there are no perfect individual predictors for TBI in children with minor head trauma, guidelines have been published and clinical prediction models are being developed that help clinicians predict which patients have a low risk of TBI and can forego neuroimaging. More data will be especially useful to aid clinicians in the evaluation of children younger than 2 years who can be difficult to assess and for whom we presently recommend a more conservative approach to diagnostic imaging. While precise estimates are lacking, many children experience postconcussive symptoms. We recommend pediatric follow-up after a concussion and appropriate behavioral intervention with or without pharmacologic therapy for children with persistent symptoms. Finally, return-to-play guidelines, though lacking high-level evidence, have become standard of care, and clinicians should convey this information to patients and their parents.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 363).

1 Langlois JA, Rutland-Brown W, Thomas KE. Traumatic brain injury in the United States. Atlanta, Georgia: CDC, National Center for Injury Prevention and Control; January 2006.

2 Quayle KS, Jaffe DM, Kuppermann N, *et al.* Diagnostic testing for acute head injury in children: when are head computed tomography and skull radiographs indicated? Pediatrics 1997; 99:E11.

3 Palchak MJ, Holmes JF, Vance CW, *et al.* A decision rule for identifying children at low risk for brain injuries after blunt head trauma. Ann Emerg Med 2003; 42:492–506.

4 Dunning J, Batchelor J, Stratford-Smith P, *et al.* A meta-analysis of variables that predict significant intracranial injury in minor head trauma. Arch Dis Child 2004; 89:653–659.

5 • Dunning J, Daly JP, Lomas JP, *et al.* Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. Arch Dis Child 2006; 91:885–891.

This is the largest derivation study to date of a prediction rule to identify children with minor head trauma at high risk of TBI.

6 Kuppermann N. Intracranial injury in minor head trauma. Arch Dis Child 2004; 89:593–594.

7 Haydel MJ, Shembekar AD. Prediction of intracranial injury in children aged five years and older with loss of consciousness after minor head injury due to nontrivial mechanisms. Ann Emerg Med 2003; 42:507–514.

8 Haydel MJ, Preston CA, Mills TJ, *et al.* Indications for computed tomography in patients with minor head injury. N Engl J Med 2000; 343:100–105.

9 Klassen TP, Reed MH, Stiell IG, *et al.* Variation in utilization of computed tomography scanning for the investigation of minor head trauma in children: a Canadian experience. Acad Emerg Med 2000; 7:739–744.

10 Livingston DH, Lavery RF, Passannante MR, *et al.* Emergency department discharge of patients with a negative cranial computed tomography scan after minimal head injury. Ann Surg 2000; 232:126–132.

11 Blackwell CD, Gorelick M, Holmes JF, *et al.* Pediatric head trauma: changes in use of computed tomography in emergency departments in the United States over time. Ann Emerg Med 2006; 1 December [Epub ahead of print].

12 Homer CJ, Kleinman L. Technical report: minor head injury in children. Pediatrics 1999; 104:e78.

13 Osmond MH, Klassen TP, Stiell IG, Correll R. The CATCH rule: a clinical decision rule for the use of computed tomography of the head in children with minor head injury [abstract]. Acad Emerg Med 2006; 13 (Suppl 1):11.

14 Oman JA, Cooper RJ, Holmes JF, *et al.* Performance of a decision rule to predict need for computed tomography among children with blunt head trauma. Pediatrics 2006; 117:e238–e246.

15 Stiell IG, Lesiuk H, Wells GA, *et al.* The Canadian CT Head Rule Study for patients with minor head injury: rationale, objectives, and methodology for phase I (derivation). Ann Emerg Med 2001; 38:160–169.

16 Lee CI, Haims AH, Monico EP, *et al.* Diagnostic CT scans: assessment of patient, physician, and radiologist awareness of radiation dose and possible risks. Radiology 2004; 231:393–398.

17 The ALARA (as low as reasonably achievable) concept in pediatric CT intelligent dose reduction: multidisciplinary conference organized by the Society of Pediatric Radiology; August 18–19, 2001. Pediatr Radiol 2002; 32:217–313.

18 Thomas KE, Parnell-Parmley JE, Haidar S, *et al.* Assessment of radiation dose awareness among pediatricians. Pediatr Radiol 2006; 36:823–832.

19 National Cancer Institute. Radiation risks and pediatric computed tomography (CT): a guide for healthcare providers. Bethesda, Maryland; 2002.

20 Brenner D, Elliston C, Hall E, *et al.* Estimated risks of radiation-induced fatal cancer from pediatric CT. AJR Am J Roentgenol 2001; 176:289–296.

21 Brenner DJ. Estimating cancer risks from pediatric CT: going from the qualitative to the quantitative. Pediatr Radiol 2002; 32:228–233; discussion 242–244.

22 Hall P, Adami HO, Trichopoulos D, *et al.* Effect of low doses of ionising radiation in infancy on cognitive function in adulthood: Swedish population based cohort study. BMJ 2004; 328:19.

23 Forsyth R. The CHALICE rule: ready for prime time? Arch Dis Child 2006; 91:877–878.

24 Greenes DS, Schutzman SA. Clinical indicators of intracranial injury in head-injured infants. Pediatrics 1999; 104:861–867.

25 Gruskin KD, Schutzman SA. Head trauma in children younger than 2 years: are there predictors for complications? Arch Pediatr Adolesc Med 1999; 153:15–20.

26 Schutzman SA, Barnes P, Duhaime AC, *et al.* Evaluation and management of children younger than two years old with apparently minor head trauma: proposed guidelines. Pediatrics 2001; 107:983–993.

27 Greenes DS, Schutzman SA. Clinical significance of scalp abnormalities in asymptomatic head-injured infants. Pediatr Emerg Care 2001; 17:88–92.

28 Schutzman SA, Greenes DS, Schutzman SA, *et al.* Pediatric minor head trauma. Ann Emerg Med 2001; 37:65–74.

29 Leaman AM, Rysdale E, Leaman AM, *et al.* Can we abolish skull x rays for head injury? [comment]. Arch Dis Child 2006; 91:374.

30 Reed MJ, Browning JG, Wilkinson AG, *et al.* Can we abolish skull x rays for head injury? Arch Dis Child 2005; 90:859–864.

31 Macgregor DM, McKie L, Macgregor DM, *et al.* CT or not CT – that is the question: whether 'tis better to evaluate clinically and x ray than to undertake a CT head scan! Emerg Med J 2005; 22:541–543.

32 Mazonakis M, Damilakis J, Raissaki M, *et al.* Radiation dose and cancer risk to children undergoing skull radiography. Pediatr Radiol 2004; 34:624–629.

33 Greenes DS, Schutzman SA. Occult intracranial injury in infants. Ann Emerg Med 1998; 32:680–686.

34 Lloyd DA, Carty H, Patterson M, *et al.* Predictive value of skull radiography for intracranial injury in children with blunt head injury. Lancet 1997; 349:821–824.

35 Chung S, Schamban N, Wypij D, *et al.* Skull radiograph interpretation of children younger than two years: how good are pediatric emergency physicians? Ann Emerg Med 2004; 43:718–722.

36 Practice parameter: the management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. Neurology 1997; 48:581–585.

37 McCrory P, Johnston K, Meeuwisse W, *et al.* Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. Br J Sports Med 2005; 39:196–204.

38 Akhtar JI, Spear RM, Senac MO, *et al.* Detection of traumatic brain injury with magnetic resonance imaging and S-100B protein in children, despite normal computed tomography of the brain. Pediatr Crit Care Med 2003; 4:322–326.

39 Bazarian JJ, Blyth B, Cimpello L. Bench to bedside: evidence for brain injury after concussion: looking beyond the computed tomography scan. Acad Emerg Med 2006; 13:199–214.

40 Hofman PA, Stapert SZ, van Kroonenburgh MJ, *et al.* MR imaging, single-photon emission CT, and neurocognitive performance after mild traumatic brain injury. AJNR Am J Neuroradiol 2001; 22:441–449.

41 Hughes DG, Jackson A, Mason DL, *et al.* Abnormalities on magnetic resonance imaging seen acutely following mild traumatic brain injury: correlation with neuropsychological tests and delayed recovery. Neuroradiology 2004; 46: 550–558.

42 Diagnostic and statistical manual of mental disorders IV. Washington DC: American Psychiatric Association; 1994. pp. 704–706.

43 Mickeviciene D, Schrader H, Nestvold K, *et al.* A controlled historical cohort study on the postconcussion syndrome. Eur J Neurol 2002; 9:581–587.

44 Necajauskaite O, Endziniene M, Jurieniene K. Prevalence, clinical features and accompanying signs of posttraumatic headache in children. Medicina (Kaunas) 2005; 41:100–108.

45 Mickeviciene D, Schrader H, Obelieniene D, *et al.* A controlled prospective inception cohort study on the postconcussion syndrome outside the medicolegal context. Eur J Neurol 2004; 11:411–419.

46 Nacajauskaite O, Endziniene M, Jureniene K, *et al.* The validity of postconcussion syndrome in children: a controlled historical cohort study. Brain Dev 2006; 28:507–514.

47 Mittenberg W, Wittner MS, Miller LJ. Postconcussion syndrome occurs in children. Neuropsychology 1997; 11:447–452.

48 Ponsford J, Willmott C, Rothwell A, *et al.* Impact of early intervention on outcome after mild traumatic brain injury in children. Pediatrics 2001; 108:1297–1303.

49 Yeates KO, Luria J, Bartkowski H, *et al.* Postconcussive symptoms in children with mild closed head injuries. J Head Trauma Rehabil 1999; 14:337–350.

50 Bazarian JJ, Atabaki S. Predicting postconcussion syndrome after minor traumatic brain injury. Acad Emerg Med 2001; 8:788–795.

51 Savola O, Hillbom M. Early predictors of postconcussion symptoms in patients with mild head injury. Eur J Neurol 2003; 10:175–181.

52 Evered L, Ruff R, Baldo J, *et al.* Emotional risk factors and postconcussional disorder. Assessment 2003; 10:420–427.

53 Sheedy J, Geffen G, Donnelly J, *et al.* Emergency department assessment of mild traumatic brain injury and prediction of postconcussion symptoms at one month post injury. J Clin Exp Neuropsychol 2006; 28:755–772.

54 Ponsford J, Willmott C, Rothwell A, *et al.* Cognitive and behavioral outcome following mild traumatic head injury in children. J Head Trauma Rehabil 1999; 14:360–372.

55 Greiffenstein MF, Baker WJ. Premorbid clues? Preinjury scholastic performance and present neuropsychological functioning in late postconcussion syndrome. Clin Neuropsychol 2003; 17:561–573.

56 Bazarian JJ, Zemlan FP, Mookerjee S, *et al.* Serum S-100B and cleaved-tau are poor predictors of long-term outcome after mild traumatic brain injury. Brain Inj 2006; 20:759–765.

57 Begaz T, Kyriacou DN, Segal J, *et al.* Serum biochemical markers for postconcussion syndrome in patients with mild traumatic brain injury. J Neurotrauma 2006; 23:1201–1210.

58 De Kruijk JR, Leffers P, Menheere PP, *et al.* Prediction of posttraumatic complaints after mild traumatic brain injury: early symptoms and biochemical markers. J Neurol Neurosurg Psychiatry 2002; 73:727–732.

59 Nygren De Boussard C, Fredman P, Lundin A, *et al.* S100 in mild traumatic brain injury. Brain Inj 2004; 18:671–683.

60 Stranjalis G, Korfias S, Papapetrou C, *et al.* Elevated serum S-100B protein as a predictor of failure to short-term return to work or activities after mild head injury. J Neurotrauma 2004; 21:1070–1075.

61 Hawley CA. Reported problems and their resolution following mild, moderate and severe traumatic brain injury amongst children and adolescents in the UK. Brain Inj 2003; 17:105–129.

62 McKinlay A, Dalrymple-Alford JC, Horwood LJ, *et al.* Long term psychosocial outcomes after mild head injury in early childhood. J Neurol Neurosurg Psychiatry 2002; 73:281–288.

63 Anderson V, Catroppa C, Morse S, *et al.* Outcome from mild head injury in young children: a prospective study. J Clin Exp Neuropsychol 2001; 23:705–717.

64 • Anderson VA, Catroppa C, Dudgeon P, *et al.* Understanding predictors of functional recovery and outcome 30 months following early childhood head injury. Neuropsychology 2006; 20:42–57.

This prospective observational study of children with head injury evaluates long-term outcome of children following head injury as well as risk factors for poor outcome.

65 Wade DT, King NS, Wenden FJ, *et al.* Routine follow up after head injury: a second randomised controlled trial. J Neurol Neurosurg Psychiatry 1998; 65:177–183.

66 de Kruijk JR, Leffers P, Meerhoff S, *et al.* Effectiveness of bed rest after mild traumatic brain injury: a randomised trial of no versus six days of bed rest. J Neurol Neurosurg Psychiatry 2002; 73:167–172.

67 Cook RS, Schweer L, Shebesta KF, *et al.* Mild traumatic brain injury in children: just another bump on the head? J Trauma Nurs 2006; 13:58–65.

68 Lew HL, Lin PH, Fuh JL, *et al.* Characteristics and treatment of headache after traumatic brain injury: a focused review. Am J Phys Med Rehabil 2006; 85:619–627.

69 Hecht JS. Occipital nerve blocks in postconcussive headaches: a retrospective review and report of ten patients. J Head Trauma Rehabil 2004; 19:58–71.

70 McCrory P. New treatments for concussion: the next millennium beckons. Clin J Sport Med 2001; 11:190–193.

71 Mittenberg W, Canyock EM, Condit D, *et al.* Treatment of postconcussion syndrome following mild head injury. J Clin Exp Neuropsychol 2001; 23:829–836.

72 Jin C, Schachar R. Methylphenidate treatment of attention-deficit/hyperactivity disorder secondary to traumatic brain injury: a critical appraisal of treatment studies. CNS Spectr 2004; 9:217–226.

73 Levin HS. Treatment of postconcussional symptoms with CDP-choline. J Neurol Sci 1991; 103 (Suppl):S39–S42.

74 McCrory P. Does second impact syndrome exist? Clin J Sport Med 2001; 11:144–149.

75 McCrory PR, Berkovic SF. Second impact syndrome. Neurology 1998; 50:677–683.

76 Cantu RC. Return to play guidelines after a head injury. Clin Sports Med 1998; 17:45–60.

77 Bazarian JJ, Veenema T, Brayer AF, *et al.* Knowledge of concussion guidelines among practitioners caring for children. Clin Pediatr (Phila) 2001; 40:207–212.

78 Pleacher MD, Dexter WW. Concussion management by primary care providers. Br J Sports Med 2006; 40:e2; discussion e2.

79 Bazarian JJ, McClung J, Cheng YT, *et al.* Emergency department management of mild traumatic brain injury in the USA. Emerg Med J 2005; 22:473–477.