PRACTICAL SUMMARIES IN

ACUTE CARE

A Focused Topical Review of the Literature for the Acute Care Practitioner

The facts behind the controversy: High-dose steroids in spinal cord injury

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Introduction

Acute spinal cord injury (SCI) is an often devastating event, with consequences of lifelong neurological deficits and significant disabilities. There are an estimated 14,000 victims of traumatic SCI in the United States annually, more than 90% caused by motor vehicle collisions, affecting a disproportionately young population.[1] The emergency physician is faced with the multiple challenges of treating the trauma patient while ensuring proper spinal immobilization, obtaining appropriate imaging, and applying limited treatment options to minimize neurologic sequelae.

SCI occurs in two phases. Direct mechanical injury to the spinal cord initiates a secondary response encompassing a cascade of vascular and cellular changes leading to inflammation, edema, and neuronal ischemia. Also, inflammatory mediators, calcium-mediated cellular injury, and lipid peroxidation play significant roles in this second phase of SCI. For the past 30 years, management of acute SCI has included administration of high-dose steroids, based largely upon three well-designed randomized clinical trials (National Acute Spinal Cord Injury Studies [NASCIS I, II, and III]). Intravenous high-dose methylprednisolone (MPSS) theoretically blunts lipid peroxidation and hydrolysis of cellular membranes.

Despite the physiologic risks of steroids and limited clinical evidence showing benefits, the potential neuroprotective effects of steroid treatment have made these protocols an implied standard of care. However, subsequent trials have produced conflicting evidence, and the role of steroids in acute SCI treatment remains a contentious issue. While 99% of Level I trauma centers follow the high-dose steroid protocol suggested by the NASCIS II/III, only half of the practitioners polled believed in the evidence.[2] The aim of this paper is to review the most pertinent studies and current literature that guide our practice today, and discover the facts behind the controversy regarding the role of high-dose steroids in acute spinal cord injury.

The implied standard of care

Source: Bracken MB, et al. A randomized controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury. Results of the Second National Acute Spinal Cord Injury Study. *New Engl J Med* 1990; 322:1405-11.

This seminal double-blind placebo-controlled trial was the foundation for current SCI treat-

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ment protocols. The trial involved 487 patients divided into three groups, receiving methylprednisolone, naloxone, or placebo. Methylprednisolone was administered as a 30 mg/kg bolus and 5.4 mg/kg/hr for 23 hours, and naloxone as a 5.4 mg/kg bolus followed by 4.0 mg/kg/hr for 23 hours. Pharmacologic agents were administered within 8 hours of acute closed SCI in 95% of participating patients. Neurological exams were scored based upon motor strength (clinical scale 0 to 5) and sensation to pinprick and touch (scale of 1 to 3 in 29 dermatomes where *1* indicated *absent*, *2* for *abnormal*, and *3* for *normal* sensation). These exams were performed at the time of admission, at 6 weeks, and again at 6 months.

There was no reported effect on motor scores at any time, but there was a statistically significant improvement in sensory scores at the 6-month neurological exam (pinprick, 10.0 vs 6.6; $P = 0.012$; and touch, 8.7 vs 5.9; $P = 0.042$). Complication and mortality rates were similar in the treatment and placebo groups. Patients treated more than 8 hours from time of injury had results inferior to placebo. These findings led the authors to conclude that high-dose steroid treatment with methylprednisolone is indicated for the treatment of acute SCI within 8 hours of the injury.

Commentary

To date, there had been few large, multicenter randomized controlled trials that strove to answer the question: Should steroids be administered for acute spinal cord injury? The NASCIS I did not show any benefit from methylprednisolone, but the dose was considered to be inadequate based upon animal studies. The dose was increased in this study, and it was the first study to indicate a statistically significant improvement in sensory function at 6 months.

A follow-up article by the same group demonstrated a statistically significant improvement in motor function from 6 months to 1 year after SCI ($p = 0.03$) if treated less than 8 hours from initial injury.[3] However, at the 1-year mark, the changes in pinprick and touch sensation were no longer statistically significant. Furthermore, the motor function gains were determined by post hoc analysis. If all randomized patients were considered, there was actually no overall benefit in neurological function among the three groups after 1 year. While criticism of the post hoc analyses has led many to question the validity of the study's results, even modest improvement in motor function can lead to important functional gains in SCI victims. This study's results led to the use of high-dose methylprednisolone becoming an implied standard of care.

Timing and consequences

Source: Bracken MB, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. *JAMA* 1997; 277(20):1597-1604.

Known as NASCIS III, this double-blind, randomized controlled study attempted to define the ideal duration of methylprednisolone therapy for SCI. A total of 499 patients were randomized into three groups: one group received a 48-hour infusion of methylprednisolone; the second group a 24-hour course within 8 hours after injury; and the third group received a 2.5 mg/kg bolus infusion of tirilazad mesylate (a potent lipid peroxidation inhibitor) every 6 hours for 48 hours. Patients' motor and sensory functions were examined

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immediately after injury, at 6 weeks, and at 6 months after therapy.

Motor function was tested bilaterally in 14 segments, each segment scored from 0 to 5, for a total maximum score of 70 for all normal responses. Sensory function was tested for 29 sensory levels on a scale of 1 to 3 (similar to NASCIS II) for a total possible score of 87. The group that received a 48-hour methylprednisolone infusion had an improvement of 3.6 motor points at 6 weeks ($p = 0.04$) and 3.7 motor points at 6 months ($p = 0.06$) compared with the 24-hour treatment group.

The 48-hour methylprednisolone group was further divided into two subgroups, one that received steroids within 3 hours of injury, the other receiving treatment 3 to 8 hours after SCI. The 3-to-8 hour treatment subgroup received the greatest benefit with a motor score improvement of 7.2 points ($p = 0.008$). Patients in the tirilazad arm had significantly worse motor function than those randomized to either steroid infusion group. However, those receiving tirilazad had notably fewer incidences of severe pneumonia ($p < 0.02$) and severe sepsis ($p < 0.07$) compared with the 48-hour methylprednisolone group.

Researchers concluded that patients receiving methylprednisolone within 3 hours of injury should continue to receive the medication for 24 hours, and treatment initiated within 3 to 8 hours of SCI should be continued for 48 hours.

Commentary

This important study established the duration of methylprednisolone infusions based upon time of injury. In NASCIS III, researchers were able to demonstrate statistically significant improvements in motor function in patients treated within 3-8 hours of injury for 48 hours. However, when one closely evaluates the motor and sensory scoring scales used, questions arise regarding the *clinical significance* of the point improvements. The motor scale equates a 1-point improvement from 2 to 3 with that from 4 to 5. There is a functionally crucial difference between being able to overcome gravity at all versus being able to overcome additional resistance; for most functional activity, grade 4 motor activity is sufficient.[4] That being said, any gain of antigravity strength, especially in the setting of cervical spinal cord injury,[5] can be an important benefit.

Critics of the NASCIS trials maintain that the neurologic benefits of high-dose methylprednisolone therapy were determined only through post hoc analysis. No other study has been able to verify the primary outcomes. A Japanese study attempted to reproduce the subgroup results, but its methodology suffered from randomization and intention-to-treat outcome issues that has led many critics to disregard the findings.[6]

High-dose steroid therapy has inherent risks. While all three NASCIS trials showed an increased rate of pneumonia and sepsis, NASCIS III presented statistical significance of these complications occurring more often than placebo. This study proved that while the 48-hour treatment regimen is beneficial, it has higher risks than the 24-hour protocol – making the timing of when to begin treatment after injury an important issue.

Is steroid therapy evidence based?

Source: Short DJ, et al. High-dose methylprednisolone in the management of acute spinal cord injury – a systematic review from a clinical perspective. *Spinal Cord* 2000;38:273-86.

In this meta-analysis, Short and colleagues presented a thorough review of three clinical trials and six cohort studies examining high-dose steroids in acute SCI. Articles were obtained through a MEDLINE search of articles from 1966-1999. Inclusion criteria included high-dose steroids administered within 12 hours of injury, and separate reporting of outcome measures for steroid and nonsteroid treatment groups. According to the authors, studies with questionable validity were excluded from the review.

Researchers included two Level I evidence trials. The Bordeaux study randomized 106 patients to one of four treatment groups: high-dose MPSS, nimodipine, MPSS and nimodipine, and no treatment. Analysis of ASIA scores (total motor, pinprick, and touch) at 1 year did not show any difference between the treatment groups. The previously discussed NASCIS II study was quoted directly, "Considering all randomized patients at 1 year, there were no significant differences in the neurological function by the treatment group, although patients treated with methylprednisolone showed a slight advantage over those receiving placebo on all three neurological parameters." Furthermore, the authors criticized the post hoc analysis by stating that, "the figures…endorse strong consideration of chance subgroupings and…influences other than the treatments given." The 1994 Otani Japanese study, with its randomization and outcome measure flaws, was reviewed as Level II-1 evidence, concluding that there were no statistically significant changes found in either sensory or motor scores at 6 weeks or at 6 months.

None of the retrospective studies in this paper showed a benefit from high-dose steroid use. Included

were two Level II-2 and four Level II-3 publications. Two of the level II-3 studies showing no benefit from MPSS at discharge did not detail the neurological examination. The paper also included two Level II-3 studies that were retrospective analyses of penetrating trauma cases.

The researchers concluded that the small body of evidence does not support significant beneficial treatment from high-dose methylprednisolone administration. Furthermore, the authors stated that the 12 animal studies reviewed supported the contention that "high-dose methylprednisolone (within 8 or 12 h of injury) should be excluded from consideration as an intervention for acute spinal cord injury."

Commentary

The authors approached this meta-analysis with a rigorous methodology, promoting the concept that evidence-based recommendations for clinical interventions must, "advise caution in applying results from nonrandomized groups of patients." However, the authors compared randomized prospective trials showing benefit of MPSS with retrospective analyses of nonrandomized trials, the majority with deleterious outcomes. The contrast in the level of evidence represented makes it difficult to reach the authors' conclusion that the evidence cannot support administration of high-dose steroids and may in fact lead to higher morbidity and mortality.

This review lends heavy suspicion to the purported benefit of high-dose steroid protocols in the setting of acute SCI. One may question whether the included studies were influenced by selection bias because Short and his colleagues commented in the discussion that the papers excluded from their review would not have substantially changed the conclusion. While none of the studies analyzed showed strong support for steroid use, it should be noted that there are no randomized trials reproducing the NASCIS II subgroup results. Also, there exists only weak clinical evidence supporting the conclusion of deleterious neurological results from MPSS.

Are end organs affected?

Source: Kubeck JP, et al. End-organ effects of high-dose human equivalent methylprednisolone in a spinal cord injury rat model. *Spine* 2006;31(3):257-61.

Despite widespread use of high-dose steroids in human SCI patients, few studies have been performed to assess the actual effects of high-dose steroids on end organs. This prospective study utilized a spinal cord injury model in 48 rats, divided into treatment and control groups. Treatment groups received high-dose methylprednisolone within 1 hour of injury. Liver, lung, intestine, heart, kidney, and spleen were harvested at varying intervals (from 0 to 48 hours of injury) and examined for histologic effects.

The spleen was the most affected organ, with significant lymphocytic depletion at 4 hours and continuing to 48 hours. Specimens of intestinal mucosa revealed dilated and autolyzed/necrotic mucosa starting from 16 hours post injury. Eosinophilic pulmonary infiltrates were found in all groups regardless of time from injury. No significant changes were found in cardiac, kidney, or hepatic tissues.

Commentary

Studies have been performed to evaluate the effect of methylprednisolone on the actual spinal cord,[7] but there has been little literature on the effects of high-dose steroids on end organs. It is paramount to weigh the risks of any treatment against the benefits. Infection, overwhelming sepsis, and gastrointestinal bleeding are potential complications of MPSS upon which many critics hinge their dissention. Two decades after the NASCIS II study, which did not show statistically significant increased infections, Kubeck and colleagues demonstrated end-organ effects of high-dose steroids. Profound lymphocyte depletion predisposes the patient to wound infection and sepsis, as the patient is functionally immune-compromised in the vital early resuscitation phase. Effects on intestinal mucosa may also manifest as gastrointestinal bleeding.

Another small study suggested that methylprednisolone may cause myopathy in the high doses administered after SCI.[8] Qian and colleagues expanded their discussion further to suggest that the natural course of improvement in steroid-induced myopathy may be the reason for the modestly increased motor scores in patients who received steroids in NASCIS II.

Source: McCutcheon EP, et al. Acute traumatic spinal cord injury, 1993-2000. A population-based assessment of methylprednisolone administration and hospitalization. *J Trauma* 2004; 56:1976-83.

McCutcheon and his colleagues performed a retrospective analysis on SCI patients who presented to South Carolina hospitals during an 8-year period. They compared differences in hospital length of stay, acute care charges, and other variables as a function of methylprednisolone administration. The study sample was composed of 75% randomly selected patients dis-

charged with the diagnosis of traumatic SCI. Of the 1227 SCI patients analyzed, 48.7% received high-dose steroids; patients admitted through emergency departments and major trauma centers were as much as six times more likely to receive MPSS. Younger patients (age < 19 years) were also twice as likely to be treated with steroids as older patients. The patients who received MPSS treatment had acute care charges that (even after adjustment for injury severity) were on average $16,845 greater than those not receiving steroids. Based upon the data collected, the authors concluded that methylprednisolone usage was associated with significantly longer hospital stays (17.5 days vs 13.8 days, $p < 0.01$); but adjustment for the injury severity, neurological level of the lesion, and inhospital death resulted in a reduction of hospital length of stay from 4 to 2.5 days.

Commentary

Studies such as this one are important for institutional decisions regarding degrees of protocol adherence. In many cases in this review, patient body weights were not uniformly available, raising questions regarding the consistency of recommended protocols. This study also illustrates how the use of high-dose steroids for SCI is not a clinically established standard of care. While 91.3% of the surveyed emergency physicians reported agreement with the NASCIS protocol, less than half the patients received steroid therapy. There appeared to be a selection bias in those receiving MPSS – with a higher incidence found in Level I and Level II trauma centers and in patients with greater severity spinal lesions.

The association between high-dose steroid use and higher costs/length of stay can be accounted for by numerous factors, including infectious complications. One must remember that the group of patients who predominantly received steroids in this study had a higher likelihood of suffering more severe trauma and being admitted to a trauma center. The more severe SCI patients received more procedures, putting them at increased risk for infections. A 1997 study similarly found that there were significantly higher incidences of pneumonia, duration of mechanical ventilation, and intensive care unit length of stay in steroid-treated patients as compared with those who did not receive high-dose methylprednisolone with SCI.[9] These conclusions reiterate the necessity of weighing the benefits of MPSS with the very real risks.

Penetrating injury

Source: Levy ML, et al. Use of methylprednisolone as an adjunct in the management of patients with penetrating spinal injury: Outcome analysis. *Neurosurgery* 1996;39(6):1141-9.

This retrospective analysis examined if high-dose steroids could be used as a treatment adjunct for penetrating cord injuries. Of the study's 232 patients in a county Level I trauma center with single penetrating SCI with no head injury, 71% did not receive steroids, and 21% received MPSS within 8 hours of injury according to the NASCIS II protocol and dosing. The authors concluded that there was no benefit of high-dose steroid administration in patients with penetrating missile injury; treatment and control groups appeared to be homogeneous.

There was no objective (NASCIS II neurologic scale) or subjective improvement in functional outcomes, including autonomy after injury or the ability to ambulate. There was no observed significant increased rate of urinary tract infection, pneumonia, sepsis, or duration of hospitalization in the high-dose steroid treatment group.

Commentary

Outcomes from penetrating injury to the spinal cord remain universally poor despite advances in surgical and medical treatments. No significant relationships between therapeutic intervention and functional recovery have been reported, and no patients with complete motor loss have been able to regain significant neurological function.[10]

A similar study using dexamethasone (4-6 mg every 4-6 hours) in penetrating SCI was performed, which showed no improvement in neurological outcome.[11] Another retrospective review of spinal cord gunshot wound victims showed no improvement in patients with either methylprednisolone or dexamethasone.[12] Interestingly, little increase in adverse events in steroid-treated patients was observed in all three of these studies. Based upon these studies, the use of methylprednisolone in penetrating SCI cannot be supported.

The pediatric population

Source: Faillace WJ. Management of childhood neurotrauma. *Surg Clin North Am* 2002;82(2):349-63.

In this review article, Faillace provides a thorough review of acute stabilization, medical, and surgical management of pediatric neurotrauma, including traumatic SCI. SCI in children occurs as a function of age, with head control being a major factor in determining injury. In age 0-1 years, SCI is primarily due to birth trauma. Children aged 2-9 years have a higher incidence of SCI from

falls and motor vehicle collisions. Sports-related injuries increase from age 9 to 16 years, and injuries from motor vehicle collisions predominate as the leading cause after age 16 years.

Pediatric spinal cord injuries still account for only 1% of all new SCI cases. The spine does not mature until approximately age 10 years. The lack of surrounding musculature, coupled with immature bone and a relatively large head-to-neck ratio can promote transient, self-reducing displacements of the vertebral column. In neonate cadaver studies, the vertebral column could be stretched for 2 inches without any disruption, while the underlying spinal cord could tolerate only a quarter-inch of disruption. For this reason, the clinician should consider the possibility of spinal cord injury without radiographic abnormality (SCIWORA).

No randomized studies of MPSS in the pediatric population exist, and children were not included in the NASCIS studies. In this review, Faillace advocated the use of high-dose steroids (at the same dose indicated by NASCIS II/III) in children with SCI based upon the implied standard of care generated by the NASCIS II study.

Commentary

SCI in children is a relatively rare occurrence, and very little literature specifically addresses the use of high-dose steroids in the pediatric population. There are no pediatric clinical trials specifically addressing this issue, and furthermore, children were excluded from the NASCIS II/III studies. There is little evidence to support or refute the use of methylprednisolone in children. As Faillace suggests, as long as high-dose steroid use is prevalent in adult SCI treatment, the same theory of steroid use should apply to the pediatric population. Special consideration must be taken with potential cases of pediatric SCI because radiographic images can be misleading. Incidence of SCIWORA in various studies ranges from 4.5% to 20% of children with spinal injuries.[13]

New therapies

Source: Lopez-Vales R, et al. FK506 reduces tissue damage and prevents functional deficit after spinal cord injury in the rat. *J Neurosci Res* 2005; 81(6):827-36.

This is a compelling prospective study that compared the use of the potent immunosuppressant FK506 with methylprednisolone (30 mg/kg) in SCI rat models. A control group received saline infusion, and an additional group received FK506 2 mg/kg intravenously followed by daily intraperitoneal injections (0.2 mg/kg).

Outcome measures included locomotor activity at time of injury and at 3, 7, and 14 days after injury, stabilization on an inclined plane, motor evoked potentials, and spinal cord tissue histological analysis for tissue sparing. Rats treated with FK506 had significantly better outcomes than those treated with either methylprednisolone or saline. FK506 was shown to be superior in histological parameters and increasing MEP amplitudes; more importantly, however, FK506 rats had improved neurological outcomes.

Rats treated with repeated FK506 doses had significantly better outcomes than those treated with a single bolus dose. Rats in the methylprednisolone group did not have results statistically significant from the controls. Thereby, the authors concluded that FK506 has potential as a pharmacologic therapy in the treatment of acute SCI and it should be considered for human trials.

Commentary

FK506 is an attractive potential intervention for SCI. It already has been FDA-approved for use in humans for preventing allograft rejection in transplant patients. Limitations of this study include the constraint of using a rat model and mechanism of injury. SCI was induced photochemically (directly on an open spinal cord) rather than mechanically. Nevertheless, the study is compelling in that a drug already approved for human use shows potential as a more effective adjunctive therapy than the current 'standard of care.' Besides being a potent immunosuppressant with very few known side effects, FK506 also protects against neuronal ischemia, excitotoxicity, and neurotrophic changes.[14]

There have been several other research studies that compared FK506 with methylprednisolone in the rat model; one such study found that the two agents used together provided better neuroprotection than either agent used alone.[15] Further studies are needed to assess the clinical and functional outcomes of human SCI patients with FK506, however the theoretical and preliminary ventures into this topic are encouraging.

Recommendations

Spinal cord injury is undoubtedly one of the most disabling traumatic injuries, with 11,000 new cases in the United States each year.[16] The costs of health care for the long-term management of a single SCI patient are staggering. As a result, clinicians hope to find a 'magic bullet' that could decrease the inflammation and edema, thereby limiting potential damage to the spinal cord. However, as evidenced

by Bracken's NASCIS II and III studies, there is little evidence to support significantly improved clinical outcomes from high-dose methylprednisolone therapy. Stratification of data showed a subgroup of patients in whom MPSS appeared to be of benefit, but this ad hoc analysis has been criticized. Systematic clinical reviews repeatedly show that the evidence does not support the use of routine high-dose steroid administration. Moreover, studies that indicate higher complication rates, end-organ effects of steroids, and longer hospital stay all seem to place high-dose steroid use in a less favorable light. Nevertheless, as McCutcheon demonstrated, the vast majority of surveyed emergency physicians accepted MPSS for the acute management of SCI.

This will likely continue to be a contentious topic and source of medicolegal litigation—and particularly true in the pediatric population, where physicians have been blamed for not offering the 'standard of care,' despite the lack of any studies regarding efficacy or even safety of high-dose methylprednisolone use in children.

Until a well-formulated prospective study is published showing a definitive lack of MPSS benefit, it is difficult to implement evidence-based recommendations against the protocol. However, the Canadian Neurosurgical Society, Canadian Spine Society, and the Canadian Association of Emergency Physicians have linked the available Level I and II evidence and revised their formal recommendations, no longer considering MPSS as their standard of care. The American Trauma Life Support organization now lists high-dose methylprednisolone as "*a* recommended treatment" rather than "*the* recommended treatment" for acute SCI.

Clinical studies of new pharmacologic agents still are needed to pursue an effective medical treatment of acute SCI. Until firm institutional level decisions regarding SCI are made, or a new therapy with proven benefits is established, physicians should continue to utilize high-dose steroids in daily practice as the best option for a disease process with a history of poor outcomes.

References

1. Thurman DJ, et al. Traumatic brain injury in the United States: A public health perspective. *J Head Trauma Rehabil* 1999; 14(6):602-15.
2. Vellman PW, et al. Administration of corticosteroids for acute spinal cord injury: The current practice of trauma medical directors and emergency medical system physician advisors. *Spine* 2003;28:941-7.
3. Bracken MB, et al. Methylprednisolone or naloxone treatment after acute spinal cord injury: 1-year follow-up data. *J Neurosurg* 1992;76:23-31.
4. Nesathurai, et al. Steroids and spinal cord injury. *J Trauma* 1998;45(6):1088-93.
5. Hugenholtz H. methylprednisolone for acute spinal injury: Not a standard of care. *CMAJ* 2003; 168(9):1145-6.
6. Otani, et al. Beneficial effect of methylprednisolone sodium succinate in the treatment of acute spinal cord injury. *Sekisui Sekizui* 1994; 7:633-47.
7. Oudega M, et al. Long term effects of methylprednisolone following transection of adult rat spinal cord. *Eur J Neurosci* 1999;11(7):2453-64.
8. Qian T, et al. High-dose methylprednisolone may cause myopathy in acute spinal cord injury patients. *Spinal Cord* 2005; 43(4):199-203.
9. Gerndt SJ, et al. Consequences of high-dose steroid therapy for acute spinal cord injury. *J Trauma* 1997;42:279-84.
10. Yashon et al. Missile injuries of the spinal cord and cauda equina. Spinal Injury 2ed. Norwalk: Appleton-Century-Crofts; 1986: 294-305.
11. Simpson RK Jr, et al. Treatment of acute penetrating injuries of the spine: A retrospective analysis. *J Trauma* 1989;29:42-6.
12. Heary RF, et al. Steroids and gunshot wounds to the spine. *Neurosurgery* 1997;41(3):583-4.
13. Hill SA, et al. Pediatric neck injuries. A clinical study. *J Neurosurg* 1984;60:700-6.
14. Bavetta S, et al. The effects of FK506 on dorsal column axons following spinal cord injury in adult rats: neuroprotection and local regeneration. *Exp Neurol* 1999;158:382-93.
15. Sosa I, et al. Immunosuppressants: Neuroprotection and promoting neurological recovery following peripheral nerve and spinal cord lesions. *Exp Neurol* 2005;195:7-15.
16. National Spinal Cord Injury Statistical Center. *Spinal Cord Injury Facts and Figures at a Glance*. 2005.

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After completing this activity, participants must complete the evaluation form provided at the end of each semester (May and November) and return it in the reply envelope provided to receive a certificate of completion. When an evaluation form is received, a certificate will be mailed to the participant.

CME QUESTIONS

31. What is the dosing of methylprednisolone as suggested by NASCIS II?

a. 5.4 mg/kg bolus followed by 4.0 mg/kg/hr infusion for 23 hours
b. 30 mg/kg bolus followed by 5.4 mg/kg/hr infusion for 23 hours
c. No bolus, start the infusion at 5.4 mg/kg/hr
d. 4-6 mg bolus every 4 to 6 hours

32. Which one of the following is the primary cause of spinal cord injury (SCI) in the United States?

a. Non-accidental trauma
b. Sports injuries
c. Penetrating injury
d. Motor vehicle collision

33. According to histological end-organ studies, which organ is most affected by high-dose steroid administration?

a. Spleen
b. Lung
c. Kidney
d. Liver

34. Which of the following characteristics is associated with higher incidence of methylprednisolone use?

a. Penetrating spinal cord injury
b. Presentation to a major trauma center
c. Elderly patient
d. Spinal cord injury > 8 hours

35. According to the studies in the article, which of the following statements regarding use of high-dose steroids in penetrating spinal cord injury is true?

a. There is a demonstrated higher rate of wound infection.
b. Initiation of therapy within 3 hours had an improved outcome.
c. There is no statistically significant benefit of high-dose steroid administration.
d. Steroid therapy is recommended in patients with multiple penetrating wounds to the spinal cord.

Answers: 31. b; 32. d; 33. a; 34.b; 35.c

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Trauma Reports

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Between 800,000 and 1,000,000 annual emergency department (ED) visits are for patients with head injury; 80% of these involve minor head injuries; 35% to 61% of patients seen in the ED undergo some form of radiographic evaluation.[1-4]

The use of computed tomography (CT) imaging specifically has seen exponential growth in the ED during the last 30 years. The first unit was installed at the Mayo Clinic in 1973; within only a few years, CT had virtually eliminated the use of pneumoencephalography and arteriography in head trauma.

In addition, the advent of CT scanning resulted in a reduction in the morbidity and mortality of head-injured patients when compared to historical controls studied by other methods.[5] This study also demonstrated a reduction in the number of skull radiographs, cerebral angiograms, and unnecessary surgical exploration. Early surgical intervention in head-injured patients with extra-axial hematomas was made possible with the advent of CT and was shown to improve morbidity and mortality in this population.[6,7]

Radiologic Evaluation of Head Trauma: Identifying the Spectrum of Injuries

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As with all radiographic studies, an organized approach to interpretation of head CT imaging, as well as other cranial radiographic studies, is imperative. Missed radiographic findings account for 12% to 15% of all malpractice settlements.[8,9] Some studies have demonstrated that missed CT scan interpretations are significant among those without specific training in CT interpretation.[10,11] Fortunately, physicians with proper training have a high degree of accuracy in interpretation of radiographs when compared with that of radiologists.[12-18] The authors review indications for radiographic imaging, interpreting images, and specific injuries.

— The Editor

Basics Principles of Radiology

Traditional radiographs record images on film or computer, which convey information about the size, shape, and distribution of tissues within a patient. Radiographs are a two-dimensional representation of a series of x-ray beams projected through a

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three-dimensional object. The image represents a combination of black, gray, and white shades that are determined by the absorption and scattering of x-rays by each tissue through which the beam is projected. Tissues that possess little radiodensity (e.g., air or fat) allow the beam to pass easily through the body with little scatter or absorption, exposing the radiographic film and producing a black image. Very radiodense tissues (e.g., metal, calcium, or bone) obstruct the beam and produce a white image. Each radiographic plate is coated with chemicals that emit a fluorescent flash when struck by the beam, thus augmenting the actual effect of the x-ray beam. More recently, the photographic plate has been modified so that the image can be digitized and converted to a computer image. Most other tissues, especially those containing water, produce a gray image. The x-ray beam will pass through many tissues en route to the film, and the final shading will reflect the summation of all tissues through which the beam has traversed.

Plain radiographs are described in terms of the path that the beam takes from the x-ray tube to the film. For example, a plain radiograph of the skull shot from front to back is an anterior to posterior (AP) projection. If directed from one side through to the other, the radiograph is a lateral view. The oblique view is obtained by directing the beam obliquely.

In CT, images represent the same spectrum of radiographic densities as plain radiography but repeated dynamic imaging allows reconstruction of the body part in the axial plain. Newer scanners allow coronal, sagittal, and even three-dimensional reconstructions. Despite the advances in CT technology, the average cost is approximately $125 per study.[19]

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The basic components of the CT scanner are the x-ray tube, detectors, and computers that allow for image reconstruction. Repeated slices are constructed as the beam rotates 360° around the patient while the table advances. Each CT image generated represents a matrix of picture elements or pixels. Each pixel is assigned an attenuation coefficient based upon its ability to absorb or scatter x-rays. This attenuation coefficient is expressed in Hounsfield units (HU), which vary from -1000 U (air) to +1000 (cortical bone). CT images can be viewed in a variety of 'windows' that accentuate tissues of a specific attenuation. In most trauma head CT protocols, 5-mm thick cuts are performed through the posterior fossa; 10-mm thick cuts are taken in the supratentorial region and will be displayed in brain, bone, and blood windows.

Newer high resolution scanners have thinner cuts, achieving submillimeter thickness. More recently, faster helical or spiral CT units that move the patient through the scanner in a continuous fashion and scan in a helical pattern around the patient have been introduced, reducing the scanning time to 20 to 30 seconds. This is particularly useful for imaging anatomy that is constantly in motion such as the chest.

Magnetic resonance imaging (MRI) uses computer technology to convert radiofrequency signals into shades of black, gray, and white. Instead of x-ray beams, an external magnetic field and radiofrequency waves are used to produce the image. The basic principle of MRI is the detection of spinning protons. When the patient is placed in the bore of the magnet, the magnetic fields of the protons align with the stronger magnetic field of the scanner. Radio waves are then intermittently and briefly applied to the tissue being examined to enhance the spin frequency by causing the protons to resonate (Larmor frequency) at a higher energy level. As the tissues return to their resting state, a radiofrequency wave is emitted and then detected by the receiving coil. The computer then converts the intensity of the detected signal to shades of black, gray, or white. The result is a three-dimensional plot of proton densities.

As noted, a signal is detected as the protons return to their resting state. This relaxation phase occurs by two major pathways: longitudinal or T1 decay, and transverse or T2 decay. Images generated from signals in the early portion of the relaxation phase (called T1-weighted sequence) produce a unique image in which fat appears as white and gray image contrast is excellent. During late relaxation (T2-weighted sequence), water appears white and fat appears gray. The T1-weighted sequence and T2-weighted sequence provide two perspectives on the same image. T1 images provide more useful architectural information; T2 images depict pathology more accurately because water (high in hydrogen protons) accumulates in many pathologic conditions (e.g., ischemia, edema, and tumor). In addition to producing images of high quality, MRI does not rely on radiation and thus, does not carry the biologic risk of x-rays.

Iodinated contrast agents used in x-ray-generated images are not useful for MR studies. Instead, paramagnetic agents such as gadolinium are used to enhance the image. Gadolinium acts by reducing T1 relaxation times and thus, improving the contrast

Table 1. High-Risk Criteria for Minor Head Injury [21]

- Glascow coma scale score < 15 after 2 hours
- Suspected open or depressed skull fracture
- Any sign of basilar skull fracture
- Vomiting ≥ 2 episodes
- Age ≥ 65 years

between tissues. It is most useful for delineating tumors and infections.

Indications for Radiographic Imaging in Head Trauma

CT scanning has virtually replaced plain skull radiographs in imaging the patient with head injury. Some physicians still will obtain plain radiographs in patients with suspected depressed skull fractures or with metallic foreign bodies. Plain radiography had previous utility in determining the presence of linear skull fractures. While 66% of patients with severe head trauma have an associated skull fracture, only 25% to 35% of patients with skull fractures have underlying cranial pathology.[20] The primary indications for obtaining skull plain films are to evaluate nonaccidental trauma (i.e., to look for fractures in different stages of healing), to assess ventricular shunt integrity, and as an adjunct to CT in penetrating skull trauma. It has been shown that the number of radiographs ordered for medicolegal considerations varies from 10% to 46%.[21,22]

Although MRI has the advantage of producing images with superior anatomic detail, CT provides several important advantages in the setting of trauma. The more rapid study completion time and greater availability of CT imaging makes it a far more useful study. Additionally, CT is as reliable as MRI in demonstrating intracranial lesions requiring operative intervention. Finally, CT has none of the stringent safety requirements of MRI imposed by the strong magnetic fields surrounding the scanner. MRI has a prominent role, however, in evaluating patients with subacute and chronic injuries. In these patients, MRI offers better discrimination between lesions and normal brain tissue, whereas subacute hemorrhage may appear isodense with normal brain tissue on CT.

Patients with altered level of consciousness documented by a Glascow coma scale (GCS) score less than 13 will require CT imaging in the acute setting. Controversy exists around the role of CT imaging in minor head trauma, defined as patients with blunt head injury, amnesia or disorientation or documented loss of consciousness, and a GCS score of 13 to 15. Several scoring systems have been suggested,[23-25] with the larger, validated Canadian CT rule[23] outperforming other published rules in adults with head trauma. Adults with risk factors delineated by the Canadian CT rule require emergent CT scanning. (See *Table 1.*) Using these criteria for obtaining a head CT in minor trauma, the authors noted a sensitivity of 100% and a specificity of 68.7% in detecting injuries requiring neurosurgical intervention. Additionally, they found a sensitivity of 98.4% and a specificity of 49.6% for clinically important brain injuries. Finally, the Canadian CT rule reduced the number of CT studies by almost 50%. Some additional patient populations must be considered for early use of CT. These include intoxicated patients,[26,27] patients with bleeding disorders,[28] patients with shunts from prior neurosurgical procedures,[29] and patients who return after initial evaluation following a head injury.[30] Other authors have independently found age greater than 65 years to be a risk factor for significant head injury.[31,32] However, even with a clinical model that includes these high-risk patients, it has been suggested that 100% sensitivity for intracranial injuries may not be achieved.[33,34] Patients with no loss of consciousness, normal neurologic findings, no vomiting, no amnesia, and minimal scalp injury can be discharged with careful instructions including 24 hours of close observation.

Clinicians should have a lower threshold for imaging infants and children with head injury as clinical features can be particularly subtle.[35] Independent risk factors for closed head injury include headache, vomiting, an altered mental status, physical signs of a basilar skull fracture, and focal neurological deficits. Importantly, a scalp hematoma alone signifies an increased risk of intracranial injury in infants younger than 1 to 2 years and careful consideration of CT imaging.[35,36] One additional consideration is the fact that significant pathology can arise after a presumably normal CT scan. This occurrence is particularly true of cerebral contusions, which commonly develop hours to days after injury. Even delayed epidural[37] and subdural[38] hematomas have been reported.

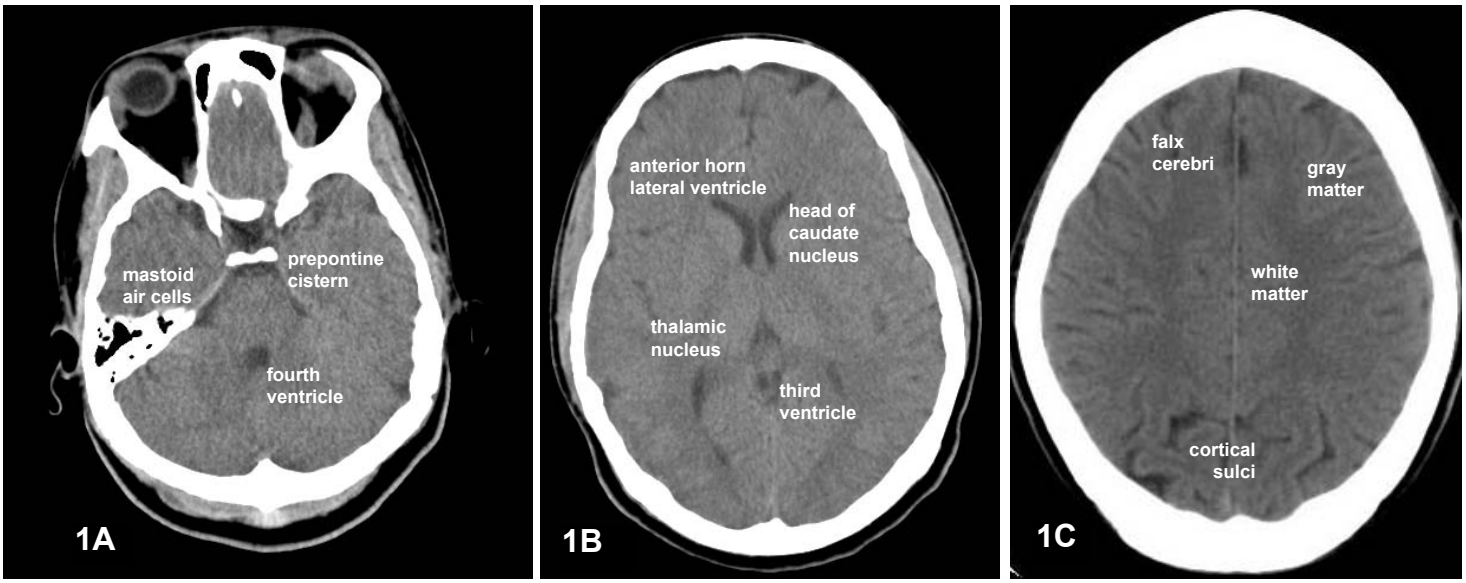
Interpretation of Radiographs in Head Trauma

As with other body systems, practitioners must have an organized approach in viewing radiographs of the cranial vault and its internal structures. Because of the importance of CT scanning in assessing the patient with head trauma, we will focus primarily on findings with this modality. The general principle is to divide head-injured patients into the following broad categories: normal intracranial findings, diffuse intracranial injury, and focal intra-axial or extra-axial findings.

Remember that the patient is placed supine on the CT table and the axial images are constructed as if one is viewing from the feet upward. As a result, the patient's left is on the right side of the image as with plain radiography. This is also true for MR images. It also should be remembered that black, gray, and white are defined by the tissue densities similar to plain radiography. However, specific tissues may be accentuated by viewing the various window settings as described previously.

The initial evaluation of the CT scan should consider the technical adequacy of the study. The axis of the x-ray beam is angled 20° downward along a line extending from the lateral canthus to the external acoustic meatus. If the patient's head is tilted as it enters the gantry, an oblique rather than a true axial cut is obtained. This can obscure an abnormality or create the appearance of an abnormality in a normal brain. The CT scout image is useful when assessing the technical quality of the study by demonstrating the orientation of the images, as well as the number of images produced and the anatomic area scanned. The

Figure 1. Cross-sectional Image of Brain at Various Levels



scout image is also valuable in assessing the skull for fractures that can be visible on this image. Images should also be evaluated for motion artifact or artifacts related to the presence of bullets or other dense foreign bodies.

Interpretation of CT imaging requires a thorough understanding of the cross-sectional anatomy of the brain. A complete review of neuroanatomy is beyond the scope of this article, however, excellent reviews on this subject are available.[39] With each slice, specific anatomic landmarks should be located as described below. Specific focus on symmetry, the cerebrospinal fluid (CSF) spaces, brain tissue, the skull, and soft tissues should be reviewed on brain window settings. Brain windows are used to highlight cerebral anatomy. Bone windows better delineate skull fractures and pneumocephaly. Blood windows more clearly demonstrate intracranial hemorrhage, which can be subtle, especially near the inner table of the skull. Localized soft-tissue swelling over the skull may be an indication of head trauma and may yield clues as to the location of coup and contre-coup injuries.

By convention, CT images progress cephalad from the foramen magnum to vertex of the skull. Important structures to identify on the lower images are the fourth ventricle, which lies on the midline, dorsal to the brainstem. Ventral to the brainstem are the prepontine and supracellar cisterns (*Figure 1A*). Because CSF is clear, these fluid-filled spaces appear dark gray. Air-filled spaces (e.g., the mastoid air cells and paranasal sinuses) are black on the image. Blood in either the CSF or sinuses will alter their appearance by increasing their densities. The posterior fossa structures demonstrated on lower CT images also include the cerebellar hemispheres and the dural sinuses.

Proceeding cephalad, the inferior portions of the cerebral hemispheres and the third and lateral ventricles can be seen (*Figure 1B*). The third ventricle is an important landmark for intracranial mass effect causing midline shift. The ambient cisterns that surround the upper pons are also visible and are important in assessing mass effect on the brainstem from injury to the temporal lobes. The third ventricle is flanked by the thalamic nuclei and each frontal horn of the lateral ventricles lies adjacent to the head of the caudate nucleus. These gray matter structures are of intermediate density as compared with the dark CSF in the adjacent ventricle.

Continuing cephalad, the cerebral cortex occupies virtually the entire image (*Figure 1C*). One should appreciate a distinct border between gray and white matter structures. In addition, the cortical sulci should be well delineated and should appear more prominent in the older brain. On the higher images, the falx cerebri becomes visible and becomes a second important midline structure. Ensure that equal volume occupies the right and left halves of the cranial vault and that no structure has crossed the midline.

Finally, there are three questions that you should consider as you view the images of a head CT scan:

- Is there blood in or around the brain?
- Is there a shift in the midline structures?
- Is there evidence of brain edema?

In the CT scan of a patient with acute injury, clotted blood is typically identified as a high density focus. Clotted blood tends to assume a characteristic shape depending upon the nature of the underlying abnormality, as will be discussed later. Next, one looks at all of the images for asymmetrical displacement of intracranial structures across the midline. Important landmarks that are useful midline indicators include the third and fourth ventricles and the falx cerebri. In addition, the ventricles may be obliterated by mass effect from edema or hematoma. Small volumes of blood in CSF can make the subarachnoid spaces appear isodense with brain, essentially obliterating the ventricles, sulci, and basal cisterns.

Table 2. Mnemonic for Reviewing Head CT Scan[14]

BLOOD:
- Epidural hematoma
- Subdural hematoma
- Intraparenchymal hemorrhage
- Intraventricular hemorrhage
- Subarachnoid hemorrhage

CAN (CISTERNS): EXAMINE FOR BLOOD AND EFFACEMENT
- Circummesencephalic
- Suprasellar
- Quadrigeminal
- Sylvian

BE (BRAIN):
- Symmetry
- Gray-white differentiation
- Shift
- Hyper- or hypodensity
- Pneumocephalus

VERY (VENTRICLES):
- Effacement
- Shift
- Blood

BAD (BONE): FRACTURES, SOFT-TISSUE INJURY, BLOOD IN SINUSES

Finally, one should appreciate a clear delineation between the gray and white matter structures in the brain. Significant edema reduces or eliminates these density differences to produce a more uniform, isodense appearance of brain tissue. Edema may manifest as either a localized or diffuse hypodense area of brain (usually 12 to 24 HU) with ill-defined gray/white boundaries resulting from increased brain water content. Edema also may result in the loss of sharp delineation of sulci. An area of suspected edema can be compared with the contralateral hemisphere to better appreciate the abnormality.

Perron[14] has suggested that the pneumonic "Blood Can Be Very Bad" be used to ensure that the examiner has reviewed all of the important features of the CT scan (*Table 2*).

MRI has not gained widespread use in the acute management of the injured patient due to a number of factors including cost, availability, speed of scanning, potential for motion artifact, limitations on the use of life support equipment in the strong magnetic field and insensitivity to bone involvement. On the other hand, MRI does not utilize ionizing radiation, can better discriminate between similar tissues, is better at defining cerebral edema, and is not complicated by bone artifacts. It is also able to provide delineation of structures in three orthogonal planes (axial, sagittal and coronal); CT scanning is generally limited to the axial plane when imaging the acutely injured patient. Small parenchymal injuries and collections of blood at the vertex and skull base are better elucidated by MRI. At least one study has demonstrated the superiority of MRI in identifying intracranial lesions;[40] however, these radiologic findings did not affect clinical management. The basic anatomy and approach are similar to reviewing CT scans. In the setting of acute trauma, MRI may be useful in assessing the extent of parenchymal injury, the exact location of extra-axial blood collections (i.e., subdural, epidural, and subarachnoid hemorrhage), the integrity of vascular structures using MR angiography, and potentially to determine the metabolic impact of injury using MR spectroscopy.

Figure 2. Linear Skull Fracture

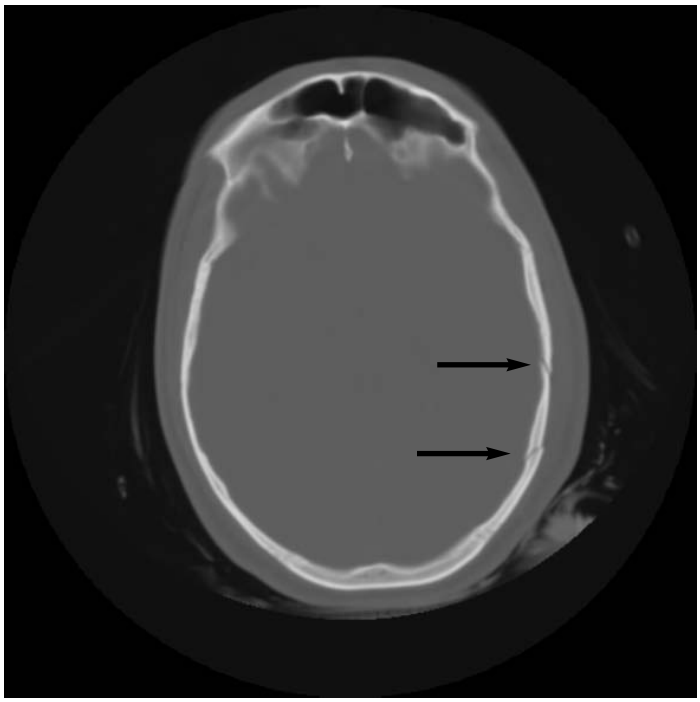


Figure 2. Black arrows indicate site of fracture.

Specific Injuries

Skull fractures can be identified using plain radiographs or CT scans. When the latter is used, bone windows best define the fracture line. Skull fractures are described as *linear*, *basilar*, or *depressed*. Diastatic fractures occur along existing suture lines. In the plain radiograph, a linear fracture appears as a clean sharply demarcated radiolucent line that is typically wider in its central portion and tapered at both ends. Linear fractures appear more sharply delineated than vascular grooves or suture lines. Fractures can further be differentiated from suture lines by the irregular corticated border seen with sutures. On a CT scan, the fracture line may be readily apparent. However, the fracture may be difficult to appreciate if it is transverse in its orientation, lying within the plane of the image. Examination of the scout film may reveal the fracture line (*Figure 2*). One should look for evidence of focal soft-tissue swelling in the scalp to identify areas of potential skull fractures. MRI is not useful in the assessment of skull fractures because this imaging modality requires that protons be mobile, and the protons of cortical bone are immobile.

Figure 3. CT Scans Demonstrating Depressed Skull Fracture, Basilar Skull Fracture, and Cerebral Contusion

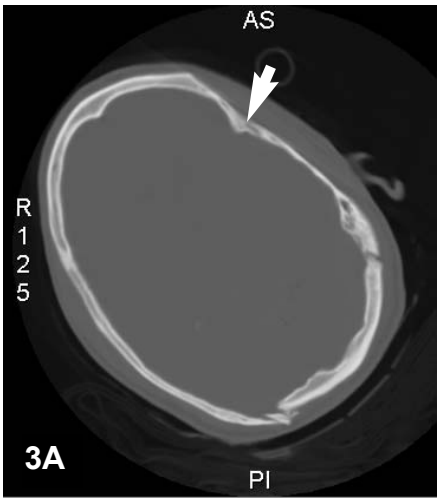


Figure 3A. Depressed Skull Fracture. Arrow denotes site of depression.

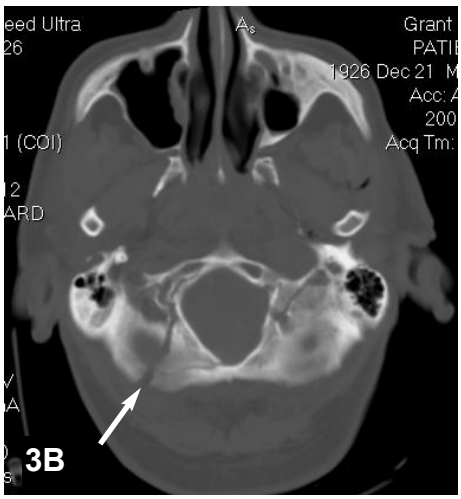


Figure 3B. Basilar Skull Fracture. Arrow demonstrates fracture through the skull base.

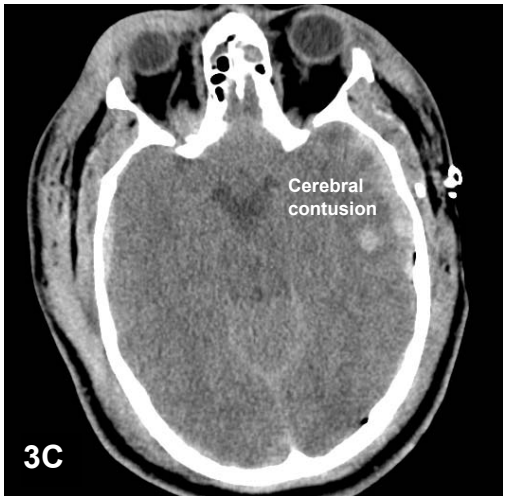


Figure 3C. Cerebral Contusion.

Thus, cortical bone appears uniformly black with MRI.

A depressed skull fracture is seen as overlapping cortical shadows on plain radiographs (*Figure 3A*). These injuries can be further elucidated by obtaining tangential views in the area of the fracture. Skull fractures are typically the result of direct trauma to the skull, most commonly in the frontal or parietal regions of the skull. A depressed skull fracture is believed to be significant when the deepest portion of the fracture extends below the inner table of the skull. These fractures are typically associated with injuries to brain parenchyma and dural tears. CT scanning offers the advantage of accurately defining the depth of the fracture and demonstrating any underlying brain injury.

Finally, basilar skull fractures are typically defined by clinical findings of raccoon eyes, Battle's sign, hemotympanum, CSF rhinorrhea, or otorrhea. A Towne view taken with plain skull radiographs occasionally may demonstrate the fracture. CT images of the posterior fossa with 1 to 2 mm cuts are the best method of demonstrating the fracture site (*Figure 3B*). Extension of the fracture line through the outer table or into an air-filled cavity (e.g., paranasal sinuses, nasal cavity, mastoid air cells) defines a compound basilar skull fracture. Pneumocephalus may accompany a compound basilar skull fracture. Basilar skull fractures through the petrous or sphenoid bone also may result in cranial nerve injuries and are best demonstrated on high-resolution CT.

Intracranial blood is classified as being *intra-axial* (within the brain substance) or *extra-axial*. The most common intra-axial traumatic lesions are cerebral contusions, hematomas, and shearing injuries. Contusions are ill-defined collections of blood along the brain surface or within the brain substance, whereas hematomas are well-circumscribed. Contusions are typically seen in the gray matter but may extend into the subcortical white matter (*Figure 3C*). There is typically a high density focus representing hemorrhage surrounded by low density edema. These are typically defined as *coup* or *contrecoup* injuries. Coup injuries occur beneath the surface of the skull at the site of impact, whereas contrecoup injuries occur on the opposite side of the brain along the line where the force was directed. Contusions have been found in high association with depressed skull fractures. MRI also may be used to define cerebral contusion. T2-weighted images demonstrate a low intensity signal (hematoma) surrounded by a high-intensity signal (edema).

Cerebral hematomas occur in a distribution similar to cerebral contusions and are most common in the frontal and temporal regions of the brain (*Figure 4*). Hematomas may be the result of the dissection of the clot through the brain white matter or the disruption of a penetrating vessel in this region. Large clots can actually dissect through the white matter into the ventricular system that may result in obstructive or communicating hydrocephalus. Cerebral hematomas are found in association with other brain injuries in 50% of cases, and intraventricular extension occurs in 33% of cases; 20% of these lesions are bilateral.[41-42] On CT, hematomas are identified by distinct collections of hyperdense blood surrounded by a hypodense area of edema in the acute injury and serum from clot degradation in the subacute injury. Typically, these are found in the frontal or anterior temporal lobe. On MRI, cerebral hematomas are similar in appearance on T2-weighted images in that the clot is hypointense and the edema or serum is hyperintense. Delayed appearance of cerebral hematomas 1 to 7 days after injury has also been reported.[43,44]

Shear injuries typically occur at the junction of the gray and white matter of the brain as the result of rotational forces, resulting in disruption of axons and their small accompanying blood vessels. These injuries can produce diffuse cerebral edema but also may appear as multiple distinct hemorrhagic foci varying from punctate to a few centimeters in diameter. Cisternal and ventricular compression may be seen but is not common in acute injury. Typically, lesions are found in four areas: the corpus cal-

Figure 4. Cerebral Hematoma

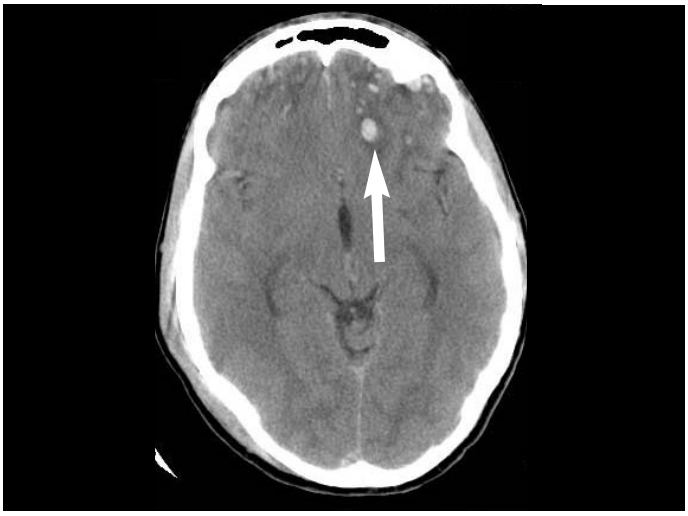


Figure 4. Cerebral Hematoma. Arrow indicates area of cerebral hematoma.

Figure 5. Subdural Hematoma

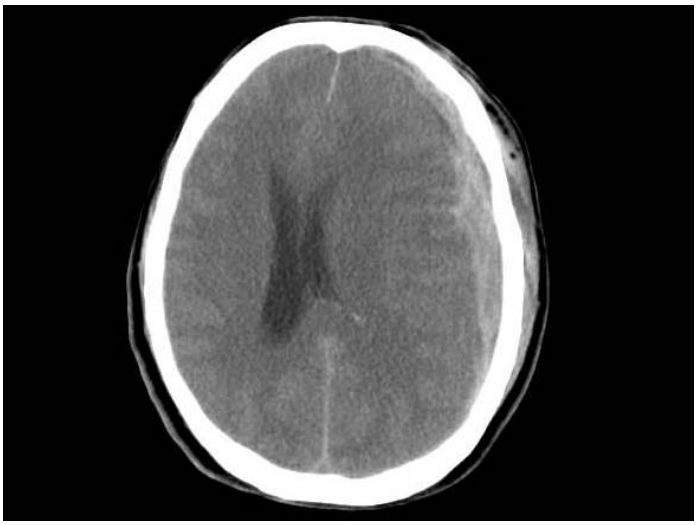


Figure 5. Subdural Hematoma. Note midline shift of lateral ventricle.

losum, the corticomedullary junction, the upper brainstem, or the basal ganglia. Hemorrhage into the corpus callosum may extend into the ventricular system where it is seen more easily.[45] It has been demonstrated that only one-fourth of shearing injuries initially present with visible hemorrhage. Thus, a negative CT study does not eliminate the possibility of shearing injury. Delayed presentations of diffuse axonal injury also have been reported.[46]

MRI has been shown to better demonstrate findings of diffuse axonal injury.[47,48] MRI should be performed when there is a disparity between the patient's clinical picture and the CT image. A high intensity signal is seen on T2-weighted images in the four regions where typical lesions occur. However, when demonstration of microhemorrhage is required, the preferred scan technique uses gradient-refocused imaging. This technique is highly sensitive to the presence of iron within hemoglobin.

Extra-axial injuries include subdural hematoma, epidural hematoma, and traumatic subarachnoid hemorrhage including intraventricular hemorrhage. In each case, the characteristic findings are defined by the relationship of the bleeding source to the brain and the meninges.

A subdural hematoma collects between the inner layer of the dura and the subarachnoid space when there is a tear in the bridging veins. The subdural space extends over both sides of the tentorium, cerebellar surfaces, convexities of the brain, and the interhemispheric fissure. Because a subdural hemorrhage lies deep to the dura, it can cross suture lines. Occasionally, a rupture of a parenchymal artery may produce these lesions. Subdural hematomas typically are associated with underlying brain injury and, therefore, carry a poor prognosis. Typical associated injuries include cerebral hematoma or contusion. Associated skull fractures are rare.

A typical subdural hematoma appears as a crescent-shaped collection of blood between the inner table of the skull and brain parenchyma (*Figure 5*). The classic shape may be modified in patients with previous injuries or meningeal infection. Small subdural injuries may be difficult to identify (S*ee* Pitfalls *section, page 8.*) and also may coexist with an epidural hematoma. These injuries tend to extend from front to back. They also may extend around the frontal or occipital pole into the interhemispheric fissure or beneath the brain along the tentorium. These injuries are often associated with displacement of intracranial contents by mass effect.[49] Collapse of the ventricular system and midline shift commonly are associated with acute subdural hematoma. In the acute phase of bleeding associated with a subdural hematoma, MRI is very insensitive in defining this injury and has gained a prominent role in the detection and defining the extent of subacute and chronic subdural hematomas.

An epidural hematoma is typically lens-shaped with blood collecting between the inner table of the skull and the dura. Because the dura attaches to the skull at suture lines, an epidural hematoma does not cross suture lines. Classically, an epidural hematoma results from a skull fracture in 85% to 95% of cases.[50] The most common site of an epidural hematoma is in the temporoparietal region and is due to disruption of the main trunk of the middle meningeal artery (*Figure 6*). This also may give rise to epidural blood on the floor of the middle cranial fossa. Disruption of the anterior trunk of the middle meningeal artery gives rise to frontal epidural hematomas. Other injuries are venous in origin and result from the tear of a dural sinus.

Epidural hematomas are almost exclusively unilateral injuries. An epidural hematoma in the posterior fossa is typically caused by venous bleeding; occipital fractures may be seen. These lesions account for 4% to 13% of epidural hematomas.[51,52] These injuries must be identified early; with prompt neurosurgical intervention a good prognosis can be expected. The size of the lesion depends upon the rate of bleeding, the time from injury to presentation, the severity of injury, and the formation of clot. Large epidural hematomas compress adjacent brain tissue, cause

Figure 6. Epidural Hematoma

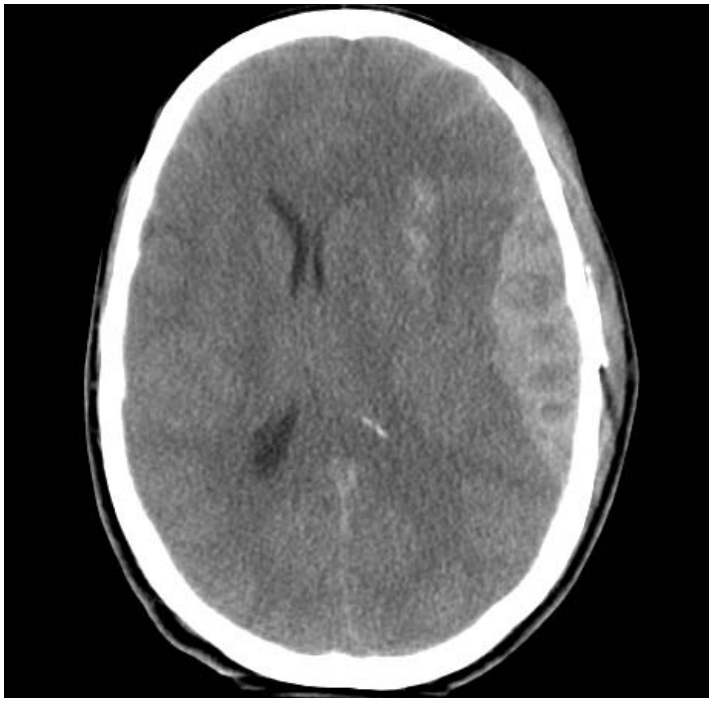
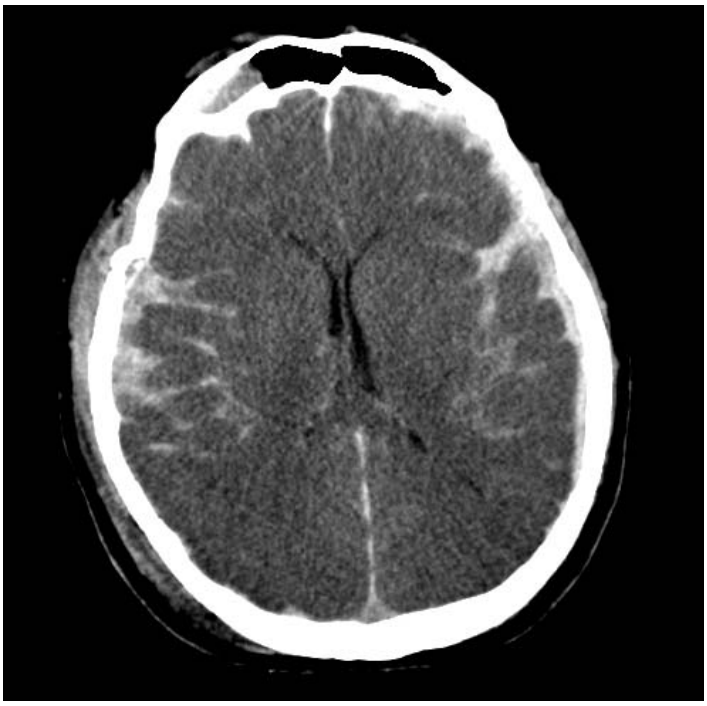


Figure 7. Traumatic Subarachnoid Hemorrhage



collapse of the ventricular system, and result in brain herniation. The lesion may not be uniform in appearance with an area of less-dense fluid representing both active bleeding and breakdown of clot.[50]

Finally, epidural hematomas commonly are associated with cerebral contusion or subdural hematoma.[53] Because these lesions must be promptly evacuated surgically, there is typically no role for MRI in acute management of epidural hematomas. Finally, it should be mentioned that epidural hematomas may have a delayed presentation arising hours to days after injury. These are typically found in areas beneath known skull fractures and frequently occur after evacuation of other intracranial lesions.[54]

In traumatic subarachnoid hemorrhage, disruption of the leptomeningeal vessels leads to bleeding, which tracks into the subarachnoid space. These collections course along the cerebral sulci and also may be seen along the interhemispheric fissure (*Figure 7*). Blood also can track into the ventricular system and can be visualized along the surface of the brain or layering dependently in the occipital horns of the lateral ventricle. Traumatic subarachnoid hemorrhages tend to be more focal than the spontaneous subarachnoid hemorrhage associated with a ruptured aneurysm.

Finally, disruption of the subependymal veins can result in traumatic intraventricular hemorrhage (*Figure 8*). This appears as a hyperdense localized collection within the ventricular system following trauma. Blood also may be seen in association with traumatic subarachnoid hemorrhage that extends into the ventricular system as described above.

Pitfalls in Radiology Interpretation

Bone averaging (*Figure 9*) occurs where a high-density structure (e.g., bone) abuts a low density structure (e.g., brain tissue). Where this occurs, the high and low density pixels are 'averaged' and give the appearance of an intermediate density that can mimic blood. Similarly, areas of calcification within the brain (e.g., the pineal gland, the choroid plexus, within the basal ganglia, or even a partial cut through the bony floor of the skull) may be mistaken for blood. Additionally, the falx cerebrum and tentorium tend to calcify with age and may be mistaken for subdural blood.

Small collections of intra-axial (cerebral contusion) or extra-axial blood (subdural or epidural hematoma or subarachnoid hemorrhage) located near the inner table of the skull may be difficult to appreciate, especially if only brain windows are used. The high density of blood may be indistinguishable from the adjacent cortical bone, a fact particularly true for extra-axial hematomas and cerebral contusions located in the parietal vertex and inferior temporal lobe. Extra-axial hematomas or subarachnoid blood that collects along the transverse plane may be difficult to distinguish from bone when present along the floor of the posterior fossa (*Figure 10*). Collections in the vertex or along the tentorium are better detected when coronal images are obtained.

CT does not provide adequate contrast between structures with similar densities, especially when one considers the high water content of brain structures. Artifact from metal structures (e.g., bullet fragments and aneurysm clips) can degrade the CT image by a phenomenon known as 'beam hardening,' where high density structures (e.g., the skull) may produce a streak artifact that can obscure the images.

Figure 8. Traumatic Intraventricular Hemorrhage

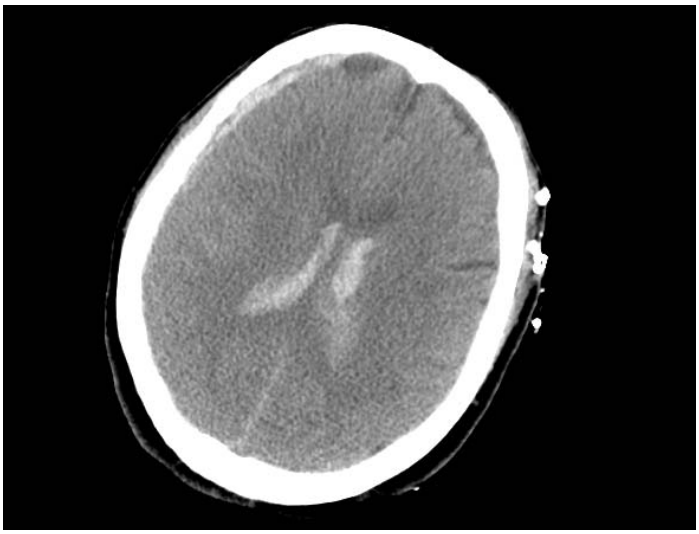


Figure 8. Traumatic Intraventricular Hemorrhage. Note that this patient also has a right subdural hematoma in the frontoparietal region.

Figure 10. Tentorial Hemorrhage

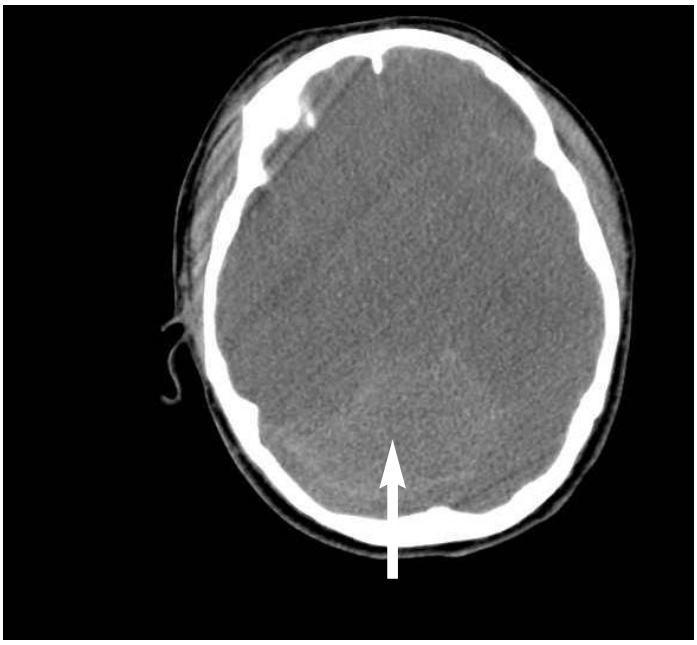


Figure 10. Tentorial Hemorrhage. Arrow depicts blood along the tentorium.

An important CT indicator of brain swelling is effacement of the subarachnoid spaces surrounding the brainstem (basal cisterns). However, blood within the basal cisterns can make them isodense with the brainstem, mimicking herniation.

Interhemispheric blood is often confused with thickening of the falx cerebri. The falx may be prominent along its posterior portion. However, subdural blood that collects along the falx can be difficult to distinguish from the more benign condition (*Figure 11*). When there is significant thickening along the anterior portion of the falx or when there is outlining of the adjacent sulci, interhemispheric blood must be suspected. Localized subdural hematomas may be found along the interhemispheric fissure. These are the result of tears in bridging veins and tend to occur in older patients with whiplash injury. Interhemispheric subdural hematomas tend to have a straight medial border and a convex lateral margin. The bleeding from a parasagittal subdural hematoma tends to remain contained in this region by arachnoid granulations. In addition, subarachnoid or subdural hemorrhage in the posterior falx may produce a high density ('pseudo-delta') sign that can be confused with the image of dural sinuses in a contrast-enhanced CT scan. Subarachnoid hemorrhage along the interhemispheric fissure is characterized by increased density outlining the parasagittal cortical sulci.

The sensitivity of CT may be limited when imaging subacute injuries. Subdural hematomas in the elderly patient are notorious for their subacute presentation. This relates to the breakdown of hemoglobin over time, with blood clot becoming isodense with brain and less visible on CT (usually around two weeks after the injury). In this circumstance, MRI becomes a more sensitive imaging modality for defining subacute or chronic collections of blood that will demonstrate a high-intensity signal on T1-weighted images.

Figure 9. Bone Averaging

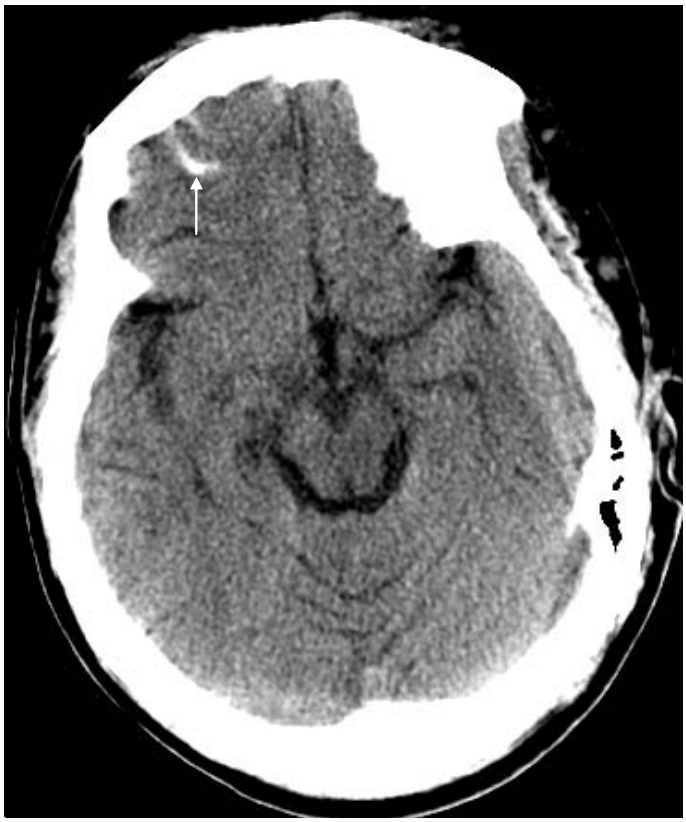


Figure 9. **Bone Averaging**. White arrow depicits bone averaging artifact from orbital roof originally interpreted as subarachnoid blood.

Figure 11. Subarachnoid Blood Along Falx

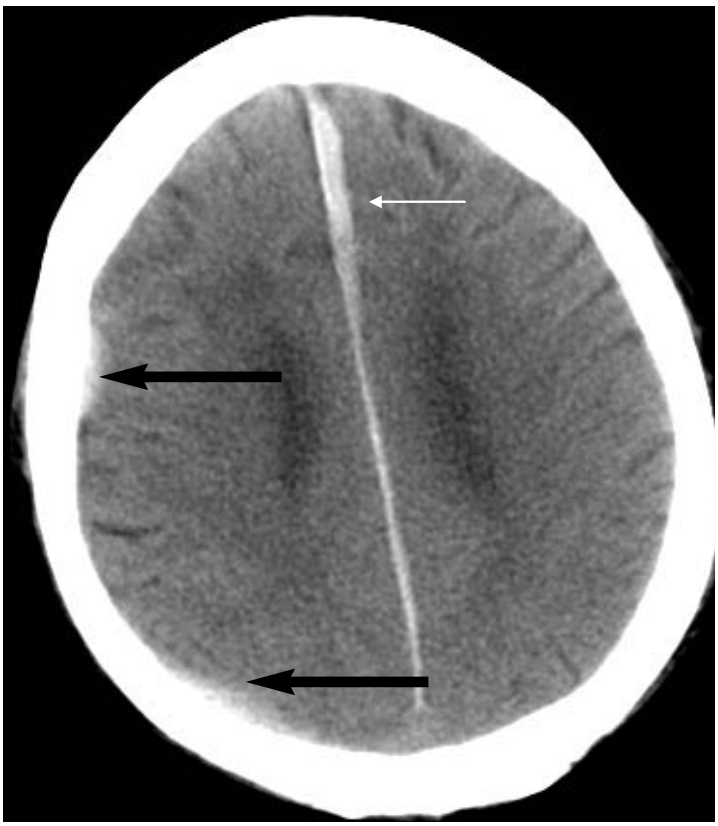


Figure 11. Subarachnoid Blood Along Falx. White arrow depicts blood along the falx. Black arrows demonstrate subdural hemorrhage along frontal and parietal convexities.

Conclusions

The clinician should be aware of the role of the various imaging modalities in the assessment of the patient with head trauma. CT imaging has revolutionized the evaluation of head injury, providing a rapid and reliable method for detecting acute traumatic hemorrhage and facilitating early surgical intervention when appropriate. Skull radiographs and MRI have limited roles in the management of early head injury. The clinician should be aware of the characteristic shape and location of intra-axial and extra-axial blood collections. In addition, when interpreting CT scans in the acute setting the clinician should be cognizant of difficulties in interpreting these radiographs caused by artifacts inherent in the imaging modality and unusual presentations of the resultant injury.

References

1. McCraig LF, Ly N. National Hospital Ambulatory Medical Care Survey: 2000 Emergency Department Summary. *Advance Data* 326:1-26, 2000.
2. Mayhue FE, Rust DD, Aldag JC, et al. Accuracy of interpretation of emergency department radiographs: Effects of confidence levels. *Ann Emerg Med* 1989;18:826.
3. Reisdorff EJ. Radiologic studies in the ED at Ingham Regional Medical Center, Lansing, MI (unpublished data). January 1996.
4. Gratton MC, Salomone JA III, Watson WA. Clinically significant radiograph misinterpretation at an emergency medicine residency program. *Ann Emerg Med* 1990;19:497.
5. Zimmerman RA, Bilaniuk LT, Gennarelli T. Computed tomography of shearing injuries of the cerebral white matter. *Radiology* 1978; 127:393-396.
6. Miller JD, Tocher JL, Jones PA. Extradural hematoma – earlier detection, better results [editorial]. *Brain Inj* 1988;2:83-86.
7. Servadei F, Piazzi G, Seracchioli A, et al. Extradural hematoma: An analysis of the changing characteristics of patients admitted from 1980 to 1986: Diagnostic and therapeutic implication in 158 cases. *Brain Inj* 1988;2:87-100.
8. Berlin L. Malpractice and radiologist, update 1986: An 11.5 year experience. *AJR* 1986;147:1291.
9. Trautlein J, Lambert RL, Miller J. Malpractice in the emergency department – Review of 200 cases. *Ann Emerg Med* 1984;13:709.
10. Alfaro D, Levitt MA, English DK, Williams V, Eisenberg R. Accuracy of interpretation of cranial computed tomography scans in an emergency medicine residency program. *Ann Emerg Med* 1995; 25:169-174.
11. Arendts G, Manovel A, Chai A. Cranial CT interpretation by senior emergency department staff. *Australas Radiol* 2003;47:368-374.
12. McLain PL, Kirkwood CR. The quality of emergency room radiograph interpretation. *J Fam Pract* 1985;20:443.
13. Overton DT. Quality assurance assessment of radiograph reading accuracy by emergency medicine faculty (abs). *Ann Emerg Med* 1987;16:503.
14. Perron AD, Huff JS, Ullrich CG, Heafner MD, Kline JA. A multicenter study to improve emergency medicine residents' recognition of intracranial emergencies on computed tomography. *Ann Emerg Med* 1998 Nov;32(5):554-562.
15. Eng J, Mysko WK, Weller GE, Renard R, Gitlin JN, et al. Interpretation of Emergency Department radiographs: a comparison of emergency medicine physicians with radiologists, residents with faculty, and film with digital display. *AJR* 2000;75:1233-1238.
16. Galasko CSB, Monahan PRW. Value of re-examining x-ray films of outpatients attending accident services. *BMJ* 1977;1:643.
17. de Lacey G, Barker A, Harper J, et al. An assessment of the clinical effects of reporting accident and emergency radiographs. *Br J Radiol* 1980;53:394.
18. Rhea JT, Potsaid MS, DeLuca SA. Errors of interpretation elicited by a quality audit of an emergency radiology facility. *Radiology* 1979;132:277.
19. Stockburger WT. CT imaging, then and now. *Radiol Management* Nov/Dec 2004:20-27.
20. Macpherson BC, Macpherson P, Jennett B. CT evidence of intracranial contusion and haematoma in relation to the presence, site and type skull fracture. *Clin Radiol* 1990;42:321.
21. de Lacey GJ, Barker A, Wignall V, et al. AJR for requesting radiographs in an accident department. *BMJ* 1979;1:159.
22. Eliastam M, Rose E, Jones H, et al. Utilization of diagnostic radiologic examinations in the emergency department. *J Trauma* 1980; 20:61.
23. Stiell IG, Wells GA, Vandernheen K, et al. The Canadian CT head

rule for patients with minor head injury. *Lancet* 2001;357: 1391-1396.
24. National Institute for Clinical Excellence (NICE). Head injury triage, assessment, investigation and early management of head injury in infants, children and adults. *Clinical Guideline 4.* London: NICE 2003;96-7.
25. Haydel MJ, Preston CA, Mills TJ, Luber S, et al. Indications for computed tomography in patients with minor head injury. *NEJM* 2000;343:103-105.
26. Nagy KK, Joseph KT, Krosner SM, et al. The utility of head computed tomography after minimal head injury. *J Trauma* 1999; 46:268-270.
27. Cook LS, Levitt MA, Simon B, Williams VL. Identification of ethanol-intoxicated patients with minor head trauma requiring computed tomography scans. *Acad Emerg Med* 1994;1:227-234.
28. Reinus WR, Swerner FL Jr. Clinical prediction of emergency cranial computed tomography results. *Ann Emerg Med* 1994;23: 1271-1278.
29. Schutzman SA, Greenes DS. Pediatric minor head trauma. *Ann Emerg Med* 1992;37:65-74.
30. Voss M, Knottenbelt JD, Peden MM. Patients who reattend after head injury. *BMJ* 1995;311:1395-1398.
31. Mack LR, Chan SB, Silva JC, Hogan TM. The use of head computed tomography in elderly patients sustaining minor head trauma. *J Emerg Med* 2003;24:157-162.
32. Arienta C, Caroli M, Balbi S. Management of head-injured patient in the emergency department: a practical protocol. *Surg Neurol* 1997;48:213-219.
33. Ibanez J, Arikan F, Pedraza S, et al. Reliability of clinical guidelines in detection of patients at risk following mild head injury: Results of a prospective study. *J Neurosurg* 2004;100:825-834.
34. Borczuk P. Predictors of intracranial injury in patients with mild head trauma. Predictors of intracranial injury in patients with mild head trauma. *Ann Emerg Med* 1995;25:731-736.
35. Schutzman SA, Greenes DS. Pediatric minor head trauma. *Ann Emer Med* 2001;37:65-73.
36. Isaacman DJ, Poirier MP. Closed head injury in children. *Pediat Emerg Care* 2002;18:48-52.
37. Milo R, Razon N, Schiffer J. Delayed epidural hematoma: A review. *Acta Neurochir* 1987;84:13-23.
38. Snoey ER, Levitt MA. Delayed diagnosis of subdural hematoma following normal computed tomography scan. *Ann Emerg Med* 1994;23:1127-1131.
39. Huddle DC, Glazer M, Chaney DB. Emergency imaging of the brai. In: Schwartz DT, Reisdorff E (eds.). *Emergency Radiology*. New York:McGraw-Hill;2000:385-442.
40. Pitts LH. The role of neuroimaging in minor head trauma. *Ann Emerg Med* 1991;20:1387-1388.
41. Zimmerman RA, Bilaniuk LT, Gennarelli T, et al. Cranial computed tomography in diagnosis and management of acute head trauma. *AJR* 1978;131:27.
42. Dublin AB, French MD, Rennick JM. Computed tomography in head trauma. *Radiology* 1977;122:365-369.
43. Dolinskas C, Bilaniuk LT, Simmerman RA, et al. Computed tomography of intracerebral hematomas I. Transmission of CT observations of hematoma resolution. *AJR* 1977;129:681-688.
44. Kishore PRS. Delayed sequelae of head injury. *J Computed Tomogr* 1980;4:287-295.
45. Abraszko RA, Zurynski YA, Dorsch NW. The significance of traumatic intraventricular hemorrhage in severe head injury. *Br J Neurosurg* 1995;9:769-773.
46. Corbo J, Tripathi P. Delayed presentation of diffuse axonal injury: A case report. *Ann Emerg Med* 2004;44:57-60.
47. Gentry LR, Godersky JC, Thompson B, et al. Prospective comparative study of intermediate-field MR and CT in the evaluation of closed head trauma. *AJR* 1988;150:673-682.
48. Gentry LR, Thompson B, Godersky JC. Trauma to the corpus callosum: MR features. *AJNR* 1988;9:1129-1138.
49. Forbes GS, Sheedy PF II, Piepgras DG, et al. Computed tomography in the evaluation of subdural hematomas. *Radiology* 1978; 126:143-148.
50. Zimmerman RA, Bilaniuk LT. Computed tomographic staging of traumatic epidural hematoma. *Radiology* 1982;144:809.
51. Ammirati M, Tomita T. Posterior fossa epidural hematoma during childhood. *Neurosurgery* 1984;14:541-544.
52. Garza-Mercado R. Extradural hematoma of the posterior cranial fossa: Report of severe cases with survival. *J Neurosurg* 1983; 59:664-672.
53. Tapiero B, Richer E, Laurent F, et al. Post-traumatic extradural hematomas. *J Neuroradiol* 1984;11:213-226.
54. Fukamachi A, Misumi S, Kaneko M, et al. Traumatic extradural hematomas with delayed development. *Comput Radiol* 1984;8: 197-201.

CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Questions

1. Which of the following are indications for a patient to receive a head CT?
   A. Glascow coma scale score of 13
   B. Open skull fracture
   C. Signs of a basilar skull fracture
   D. All of the above

2. Which one of the following groups of patients should physicians have a lower threshold for obtaining a head CT following trauma to the head?
   A. Intoxicated patients
   B. Patients taking antidepressant medications
   C. Patients with a history of a previous concussion
   D. Patients from prisons

3. In children, clinicians should have a lower threshold for obtaining a head CT after head trauma. Which of the following is *not* an independent risk factor for closed head injury?
   A. Crying
   B. Altered mental status
   C. Large scalp hematoma in a child younger than 1 year
   D. Focal neurologic deficit

4. The CT scout image is useful when assessing the technical quality of the study by demonstrating the orientation of the images as well as the number of images produced and the anatomic area scanned.
   A. True
   B. False

5. MRI has been shown to be superior to CT for the detection of:
   A. acute epidural hematomas.
   B. acute subdural hematomas.
   C. diffuse axonal injury.
   D. None of the above

6. Which of the following statements is *not* true regarding an epidural hematoma?
   A. It is usually a unilateral injury.
   B. The lesion is typically lens shaped.
   C. It is associated with a skull fracture in 10-15% of cases.
   D. The size of the lesion depends upon the rate of bleeding, time from injury to presentation, and severity of the injury.

7. Typically there is no role for the use of MRI in the acute evaluation of an epidural hematoma.
   A. True
   B. False

8. Which of the following statements regarding traumatic subarachnoid hemorrhage is true?
   A. Disruption of the leptomeningeal vessels leads to bleeding.
   B. Blood may track into the ventricular system and can be visualized along the surface of the brain or layering dependently in the occipital horns of the lateral ventricle.
   C. Traumatic subarachnoid hemorrhage tends to be more focal than the spontaneous subarachnoid hemorrhage associated with a ruptured aneurysm.
   D. All of the above

9. An important indicator of brain swelling is blood accumulation within the lateral ventricles.
   A. True
   B. False

10. Which of the following statements regarding interhemispheric blood is *not* true?
   A. It is often confused with thickening of the falx cerebri.
   B. Significant thickening along the anterior portion of the falx or outlining of the adjacent sulci should lead the clinician to suspect interhemispheric blood.
   C. Typically these are a result of an arterial bleed.
   D. Interhemispheric subdural hematomas tend to have a straight medial border and a convex lateral margin.

Answers:
1. D
2. A
3. A
4. A
5. C
6. C
7. A
8. D
9. B
10. C

In Future Issues:

The use of ultrasound in the hypotensive trauma patient

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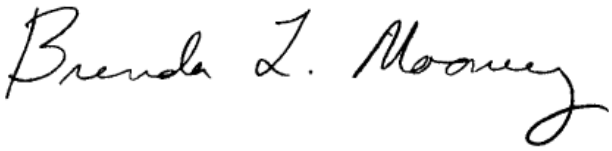

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