Pediatric Trauma

Initial Evaluation and management

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Head Injury

- Closed head injury
- Penetrating head injury

Closed Head Injury without Fractures

- Head injury is the most common cause of death and disability in children
- Approximately 7000 die each year
- Nearly 4 times as many permanently disabled

Mechanism of Injury

- Focal Impact
  - Cause focal injuries, with focal deficits
- Inertial forces
  - Generally result in diffuse damage
  - Often associated with decreased level of consciousness (GCS)

Types of Injuries

- Concussion
- Diffuse Axonal Injury
- Contusion
- Subarachnoid and Intraventricular Hemorrhage
- Shaking-Impact Syndrome

Concussion

- An injury to the head sufficient to cause loss of consciousness or amnesia of the event
- At the beginning of the continuum of angular acceleration/deceleration injury
- Most often from a blunt head injury

Presentation

- Child involved in low-velocity head impact
- Usually brief loss of consciousness (may be prolonged)
- No focal neurologic deficits

BOX 47–1
Modified Coma Score for the Pediatric Population[20]

Eye opening	
Spontaneously	4
To speech	3
To pain	2
None	1
Verbal response	
Oriented	5
Words	4
Vocal sounds	3
Cries	2
None	1
Motor response	
Obeys	5
Localizes	4
Flexion	3
Extension	2
None	1

Imaging Studies

- No absolute guidelines
- All patients with persistent symptoms should have a CT
- Plain skull films may be sufficient in infants with minimal injury, and NO neurologic deficits

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Observation

- Children with mild symptoms may be observed at home with a reliable caretaker over the next 24 hours
- Patients with more severe injuries, or with an adverse home situation should be admitted for monitoring
- Sedation should be minimized to allow for serial neurologic exams
- Repeat imaging is indicated if there is a risk for progression of intracranial injuries, or a worsening neurologic exam

Seizures

- Early post-traumatic seizures are more common in children than adults
- Usually generalized, mental status returns to baseline quickly
- Focal seizures more indicative of focal brain injury
- Not associated with long-term epilepsy

Expanding mass lesion

- Usually epidural hematoma
- If venous bleeding, may become symptomatic in a delayed manner (days after injury)
- May be progression of contusions

BOX 47–2
Surgical and Nonsurgical Management of Epidural Hematomas[36,44,75]

Surgical indications

- Focal neurologic exam, oculomotor palsy, increasing drowsiness
- Focal, significant cortical compression. Usually observed in clots >15 mm in largest width
- Brainstem herniation
- EDH volume 30–55 cc
- EDH with midline shift or uncal herniation, especially temporal lobe clot
- Concomitant intraparenchymal lesion or subdural hematoma with mass effect
- Associated fracture that transverses a major dural vessel in child that is neurologically impaired

Nonsurgical indications

- Patient is neurologically intact with only headache, nausea, vomiting, and irritability. Sixth nerve palsy without a posterior fossa clot is accepted.
- Hematoma is within the frontal, parietal, or occipital region. Small PFEDH may be observed that do not compress the cerebellar cortex, fourth ventricle, or brainstem.

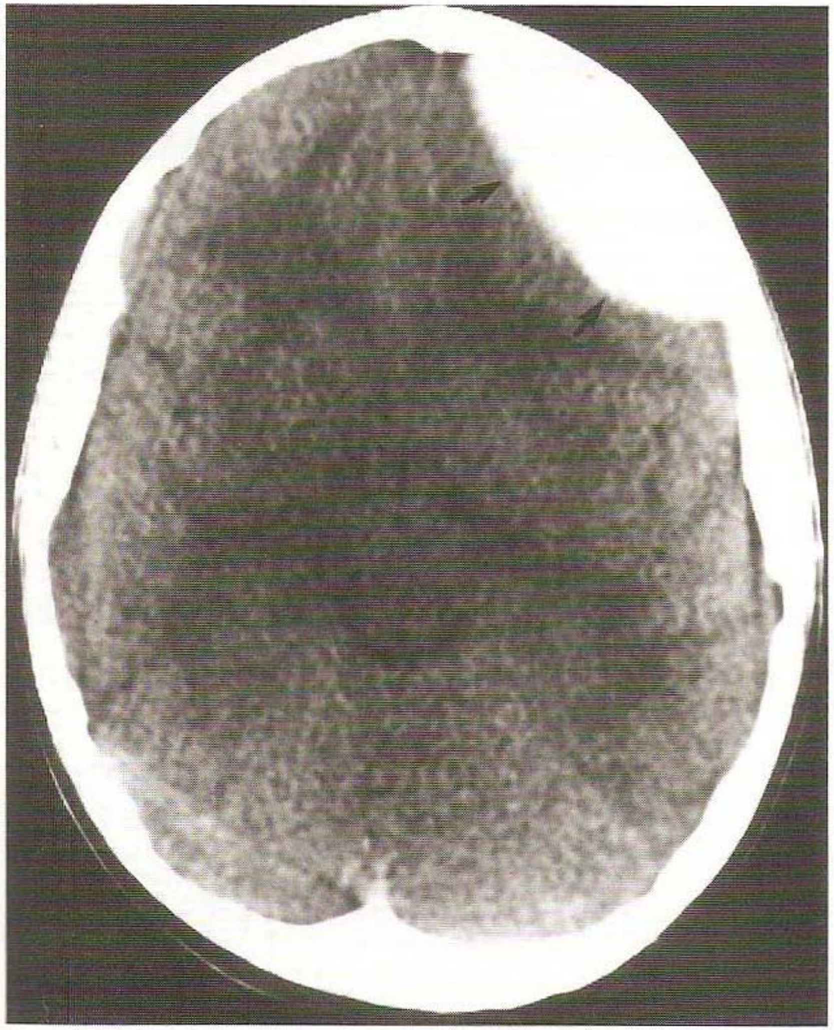
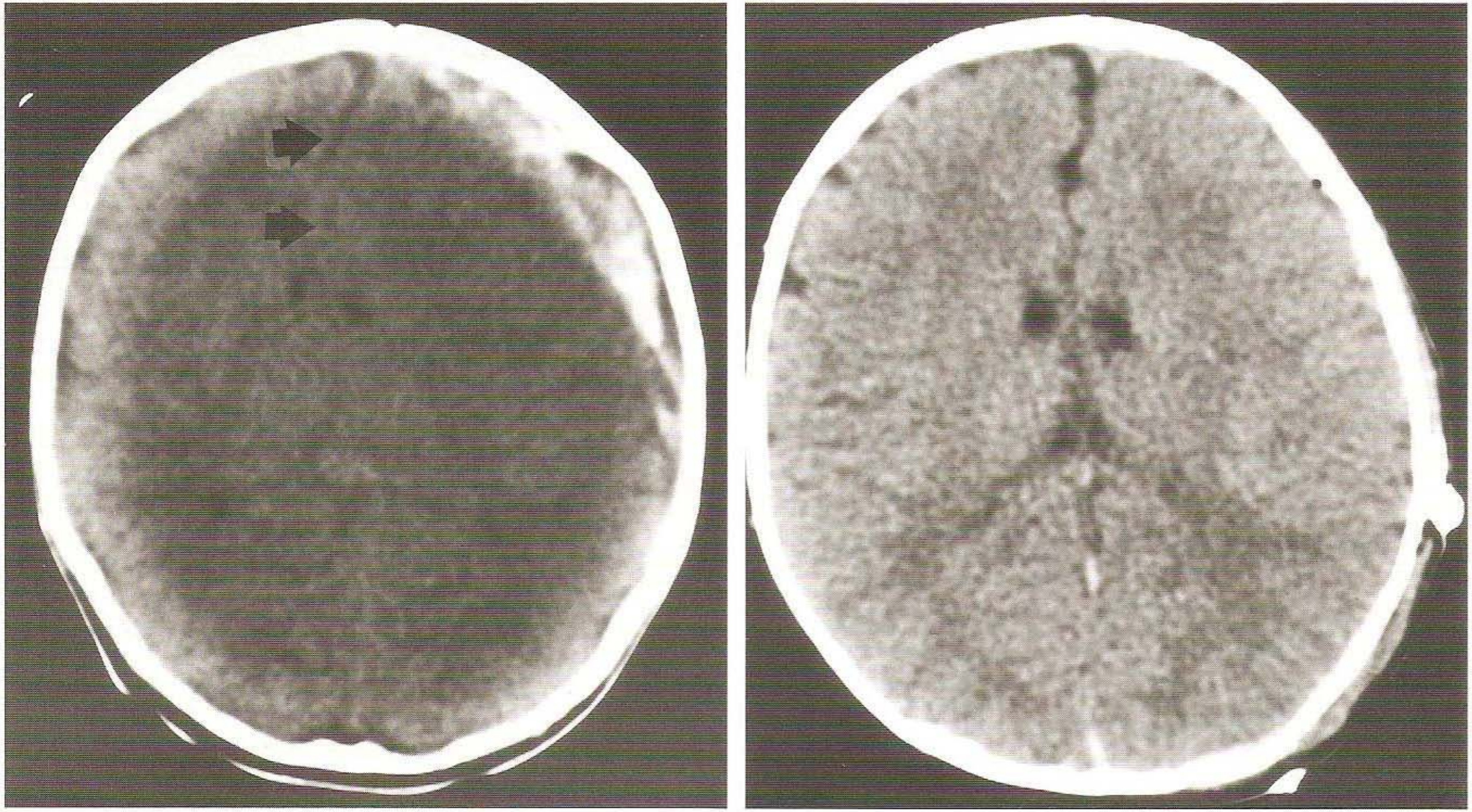


Figure 47–1. Acute frontal EDH in a 14-year-old boy with history of being hit by a baseball bat in the head 1 day earlier. This was accompanied by one episode of loss of consciousness and a CT scan was done for continued headache and lethargy. Initial GCS was 15 and then deteriorated to a GCS of 11. Arrows demarcate the cortical compression. This child underwent craniotomy without complications. Some pediatric neurosurgeons would have continued to observe this patient since the neurologic exam was nonfocal.



A B

Figure 47–4. **(A)** Large left frontal SDH with a significant midline shift (arrows). Patient was comatose and underwent emergent evacuation. **(B)** Follow-up scan demonstrates the midline structures to be normal and the ventricles open.

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Hyponatremia/Cerebral Edema

- Gradually decreasing consciousness
- May also have seizures

Vascular dissection

- Rare after minor trauma
- Most common with associated skullbase fractures
- Usually complain of focal neck pain

Diffuse Axonal Injury

- Usually applied to patients with loss of consciousness>6 hours, without a mass lesion on CT, or other known etiology
- Radiologically, see scattered petechial hemorrhages in the deep white matter, corpus callosum and brainstem on CT
- MRI shows diffuse white matter injuries
- Pathology shows axonal tears with retraction balls seen on high-power microscopy

Mechanism

- Result of large acceleration/deceleration forces
- Associated with motor vehicle accidents in older children and adolescents
- Younger children are often pedestrians struck by autos
- Rare in infants due to differences in anatomy

Presentation

- Immediate LOC
- Often accompanied by posturing, with fluctuating GCS
- May include cranial nerve dysfunction, including pupillary abnormalities

Management

- Initial CT shows no surgical lesion
- Placement of intracranial pressure monitoring device
- Repeat imaging if significant decrease in neurologic exam or elevated ICP

Hospital Course

- Later may develop a triad of hypertension, hyperhidrosis and hyperthermia
- Subsequently may go through period of agitation before regaining normal consciousness
- Extent of recovery is extremely variable

Contusion

- Focal lesion from an impact (coup and contre-coup)
- Usually progress over first few days, often cause delayed neurologic deterioration
- Associated with significant local brain edema

Figure 44–4. CT scan obtained 1 day after injury in an 8-year-old girl who was struck by a car and thrown many feet through the air before landing. The hematoma and contused tissue was evacuated to control ICP and prevent brainstem compression.

Management

- Medical management of seizure prophylaxis, ICP control and prevention of excessive edema first line
- Surgical evacuation indicated if progressive mass causes significant deterioration
- Goal is to remove adequate hematoma/infarcted brain to decompress normal brain tissue and prevent secondary injury

Subarachnoid and Intraventricular Hemorrhage

- Commonly occurs over convexities in association with contusion/after focal impact
- Diffuse basilar blood seen in occipito-cervical distraction injuries in infants or toddlers (acceleration/deceleration injury)
- Extent of parenchymal injury varies, but must prevent further injury due to instability
- Intraventricular blood most commonly seen with DAI, rarely requires specific intervention

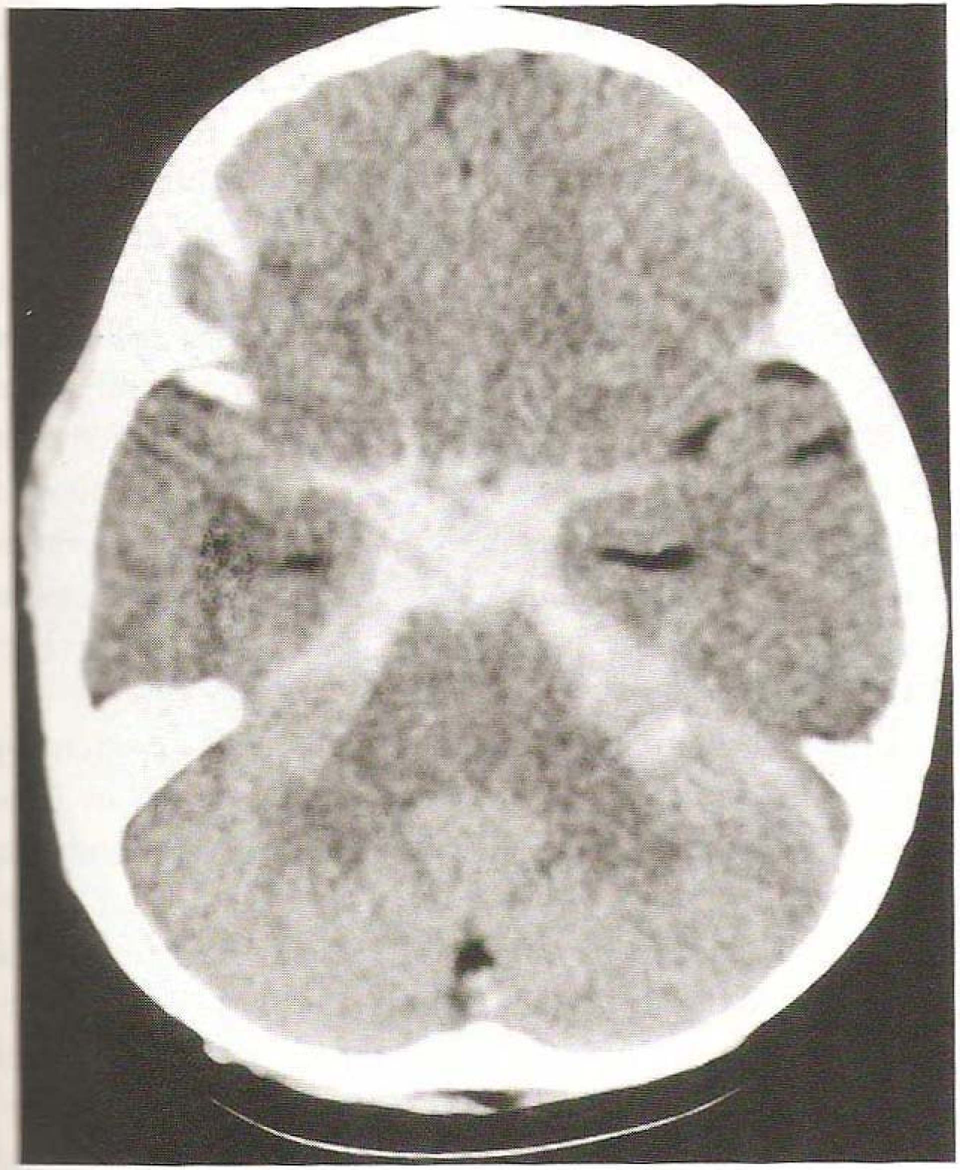


Figure 44–5. CT scan showing massive subarachnoid hemorrhage in an 8-month-old girl who was restrained in a forward-facing infant seat during a high-speed automobile crash. Examination showed a cervicomedullary distraction injury. The patient was neurologically intact on admission.

Shaking-Impact Syndrome

- Non-accidental trauma
- Mechanism involves large forces with sudden deceleration of the head
- Often have associated spine injuries
- Immature brain probably more susceptible to this kind of injury

BOX 52–1
Suspicious Scenarios and Signs and Symptoms for Spinal Column Trauma in Children

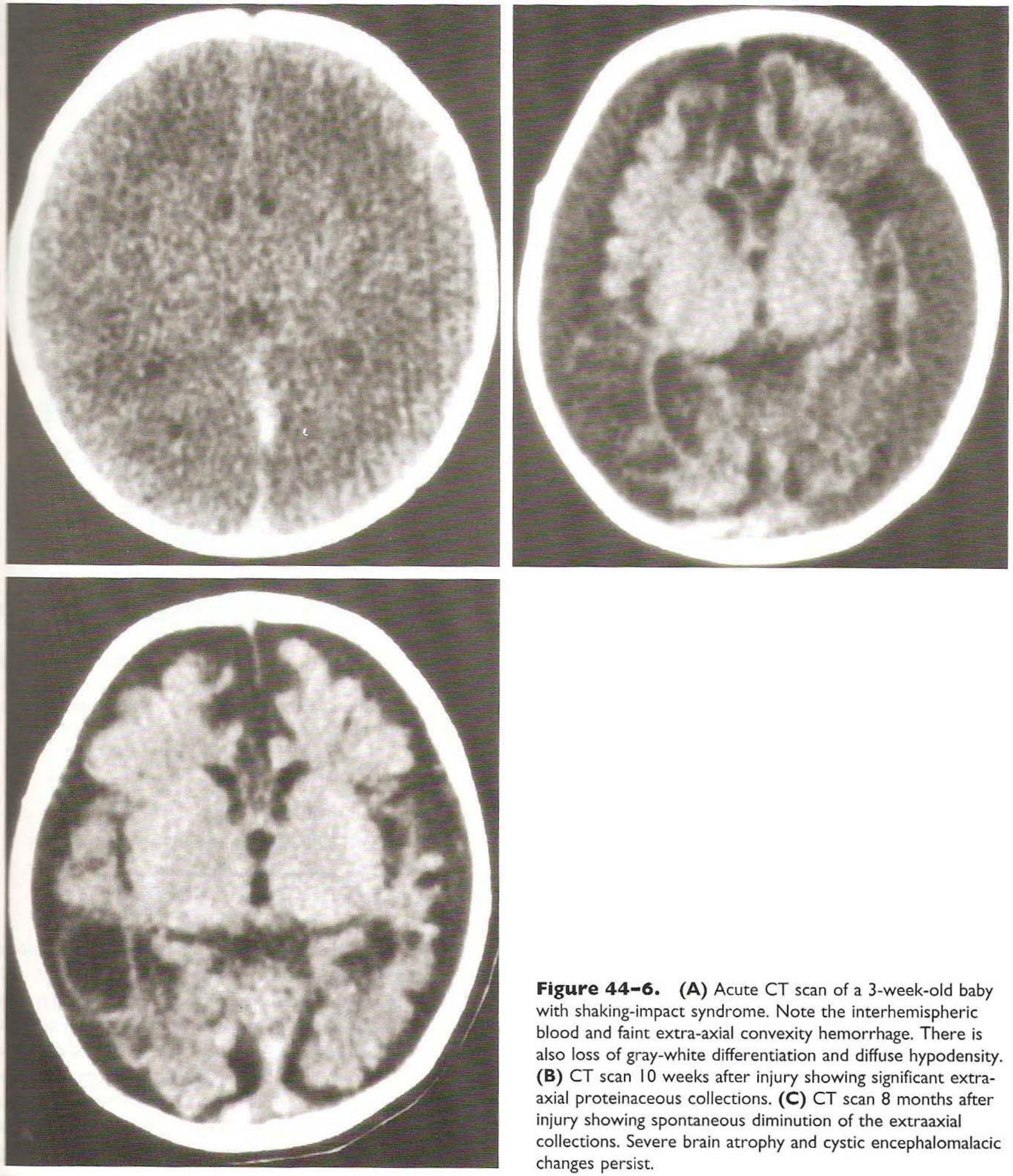
Child abuse	Seatbelt bruises of abdomen or neck
Ejection from vehicle or car seat	Subcutaneous emphysema consistent with "clothesline" injury of cervical spine and trachea
Pedestrian struck by vehicle	Crepitance or displacement of spinous processes
Child holding head in rigid position, neutral or otherwise	Vehicle track marks across back
Apnea following trauma Any neurological deficit	Heart rate of 80 with hypotension, consistent with spinal shock

Presentation

- Caretaker often describes only new onset of symptoms (lethargy/seizure/difficulty breathing, etc…) with no or only minor trauma
- Examination of child shows decreased consciousness, and often external signs of trauma (bruising, soft-tissue swelling)
- Further exam for retinal hemorrhages (present in about 75% of patients)
- Radiologic survey for skeletal injuries (present in about half)

Brain Imaging

- CT or MRI will show subarachnoid or subdural hemorrhages, often of various ages mixed
- Parenchymal injury may be minimal, or may show infarction of most of the brain with diffuse loss of grey-white junction (visible on CT within 24-48 hours)



A B C

Figure 44–6. **(A)** Acute CT scan of a 3-week-old baby with shaking-impact syndrome. Note the interhemispheric blood and faint extra-axial convexity hemorrhage. There is also loss of gray-white differentiation and diffuse hypodensity. **(B)** CT scan 10 weeks after injury showing significant extra-axial proteinaceous collections. **(C)** CT scan 8 months after injury showing spontaneous diminution of the extraaxial collections. Severe brain atrophy and cystic encephalomalacic changes persist.

Management

- ABC's-including intubation and circulatory support if necessary
- Rule out other organ system injuries that may require immediate treatment
- Seizure management/prophylaxis
- Control of ICP
- Surgical evacuation of mass lesions when necessary
- Search for other causes (e.g. coagulopathy, metabolic disease, vascular malformation)

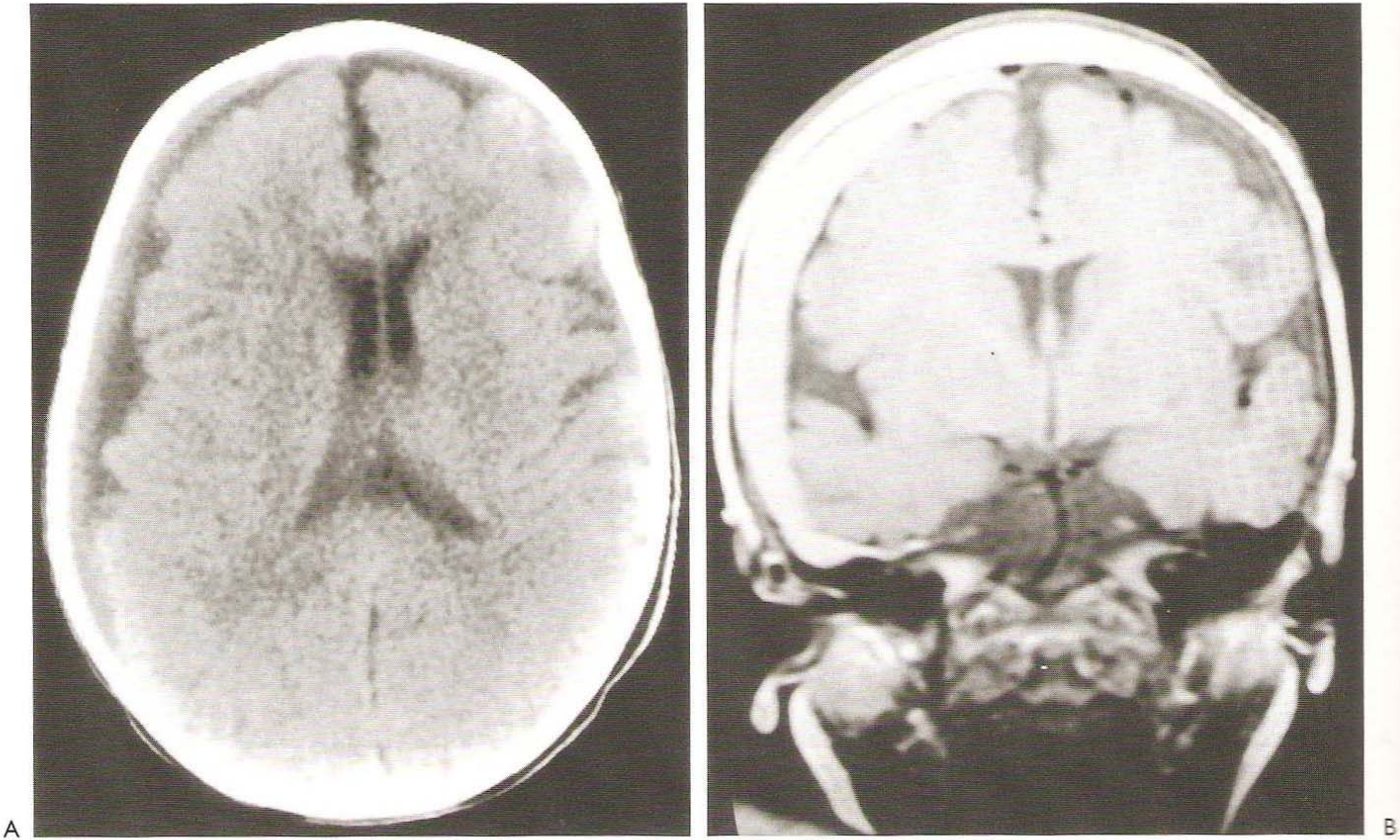


Figure 44–7. **(A)** CT scan and **(B)** MR scan of a 9-month-old girl who developed seizures and progressive developmental deterioration after a history of a minor fall at home. Note the asymmetrical bilateral subdural collections and widened Sylvian fissures. The infant was found to have glutaric aciduria, a metabolic disease associated with subdural hemorrhage and neurological decline, that may be accelerated after infection or minor trauma.

Posttraumatic Seizures

- Impact seizures (at time of event) noted in 12% of children admitted for mild head injury (concussion)
- Early seizures (within 1 week of trauma) in 10% overall
- 30-50% of patients in high-risk group (severe brain injury, focal cortical injury)
- 95% occur within the first 24 hours after trauma

BOX 47–4
Factors That Increase the Risk of Posttraumatic Seizures[71]

GCS < 8

Intracranial or subdural hematomas, diffuse cerebral edema

Open, depressed skull fracture

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TABLE 46–2.

Anticonvulsant Medications Useful in the Management of Posttraumatic Seizures

Drug	Initial Dosage (mg/kg/day)	Maintenance (mg/kg/day)	Serum Levels (μg/ml)	Half-life (hours)
Carbamazepine	5	15–20	4–12	7–27
Diazepam*#	0.2 (max 10 mg)	n/a	n/a	n/a
Lorazepam*#	0.1 (max 5 mg)	n/a	n/a	n/a
Phenobarbital	15–20	3–6	20–40	37–73
Phenytoin#	10–15	4–8	10–20	5–14

* Acute use only, generally for status epilepticus.
# Can be administered rectally.

Closed Skull Fractures

- 20% of children admitted with a head injury have a concommitant skull fracture
- May be simple linear fracture, or more complex comminuted, depressed or skull base
- Convexity linear skull fractures in an infant are usually not associated with any brain injury
- Fractures in older children, or more complex fractures increase the likelihood of finding an intracranial injury (roughly 100X)
- The worse the fracture, the worse the brain injury

Linear Skull Fractures

- 2/3 of all skull fractures
- 30% of patients do not have signs of external trauma suggesting presence of the skull fracture, may be remote to site of cranial impact
- Infants with large subgaleal hematomas associated with the injury must be monitored for hemodynamic stability
- If patient is completely normal neurologically, and has a normal CT (other than fx), then risk of deterioration is almost 0

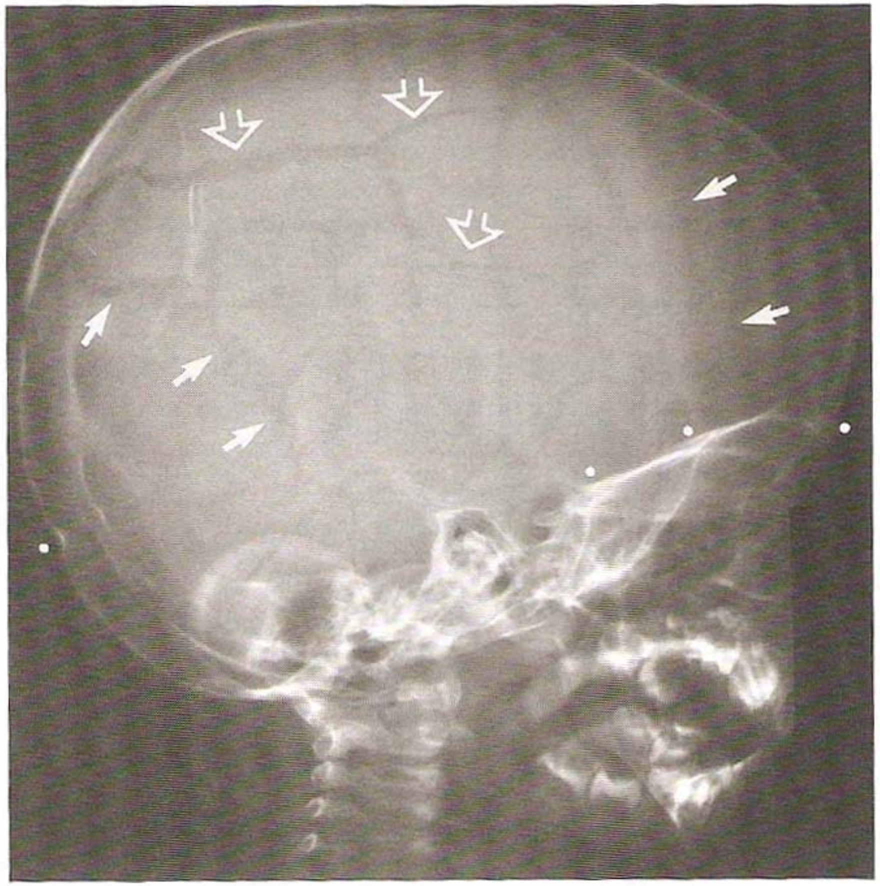


Figure 45–1. Plain skull radiography is the most sensitive means of detecting linear skull fractures, and the best way to determine the presence of multiple or complex fractures. The diagnosis of a linear fracture of the cranial vault requires only a knowledge of the normal sutural anatomy in the child and the location of the stronger areas of the skull that can serve as buttresses to a fracture. Some of these buttresses are in the nasofrontal region, the pterion, and the inion (small dots). Simple linear fractures (open arrows) will extend between two sutures (solid arrows), two buttresses, or between a suture and a buttress. Occasionally, a suture itself "fractures" and become diastatic, a phenomenon that still fits within the described schema.

Depressed Skull Fractures

- 25% of all childhood skull fractures
- 50% of patients with depressed skull fractures are children
- Caused by high-impact focal injury
- Dural laceration noted in only 10% of patients
- 80% involve frontal or parietal bone
- Most common fracture in neonates (80%)

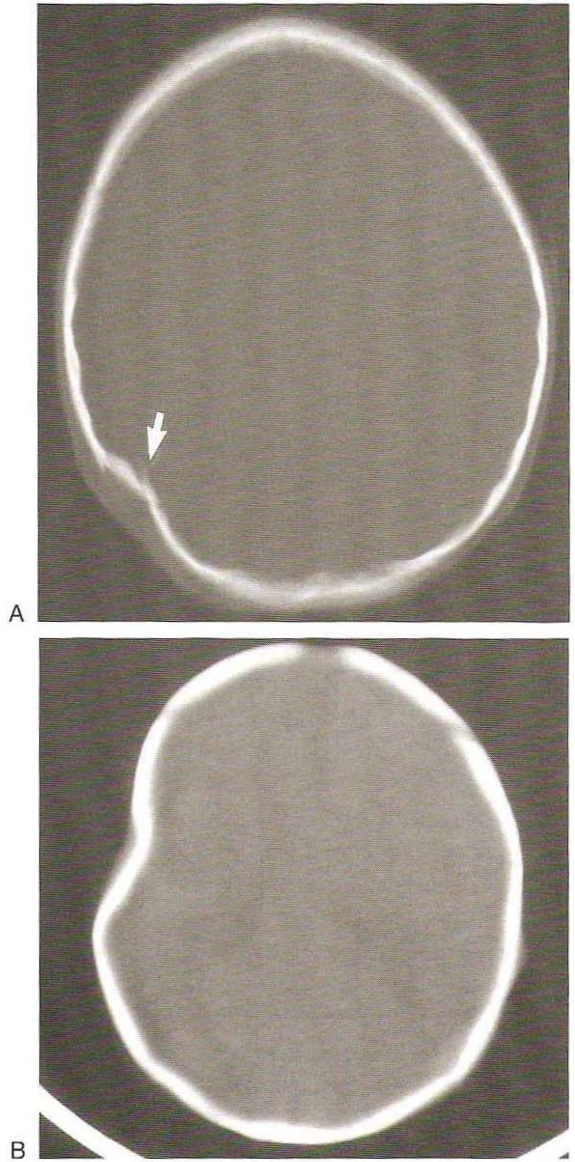


Figure 45–2. Depressed skull fractures. **(A)** CT scan showing a characteristic depressed fracture in an older child. Note the "greensticking" of the fracture and the small point of bone that commonly occurs on the inner surface of the fracture. **(B)** The characteristic "ping-pong" fracture in a newborn infant. These types of depressed fractures are likely to have a smooth inner surface; most reform without intervention.

Management

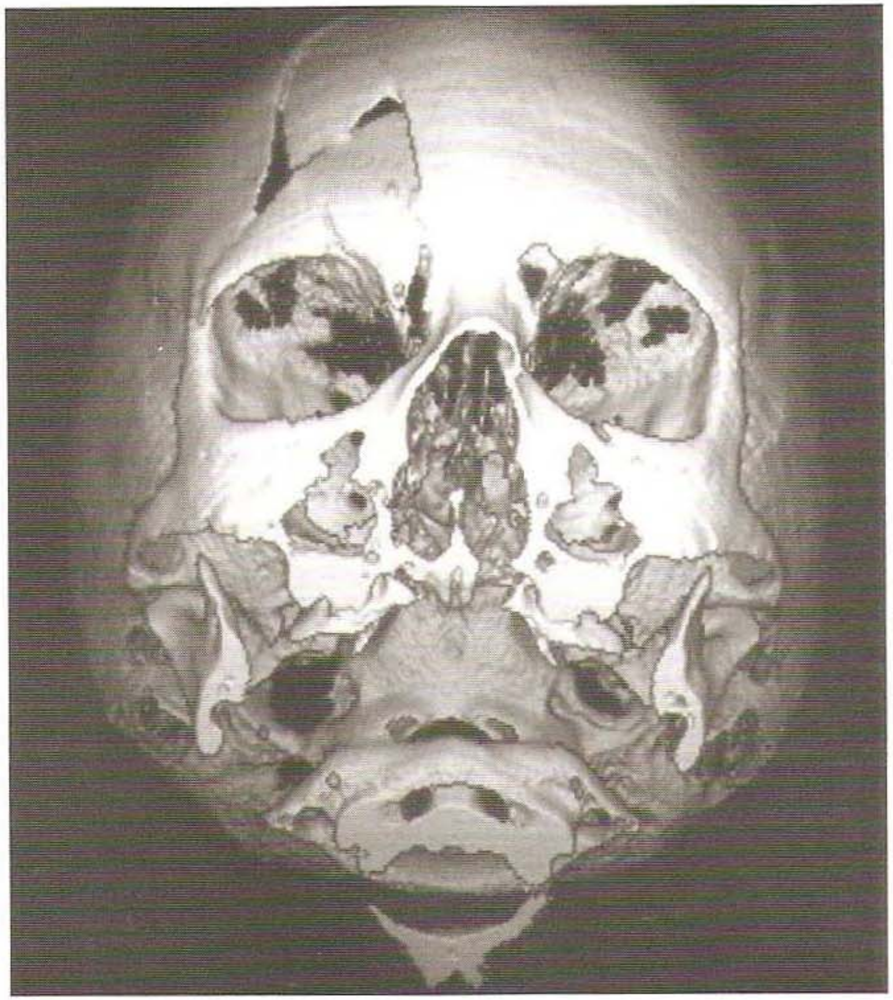
- Neonatal “ping-pong” fractures should be treated immediately if there is underlying intracranial injury
- Isolated fractures can be observed, as many will spontaneously improve as the child grows

Management

- Fractures in older children should be elevated if there is
  - A significant cosmetic deformity
  - Depression of >1cm
  - Underlying brain injury
  - Dural laceration
  - Surgery is safe (does not involve major sinuses)

Comminuted/Complex Fractures

- As these are commonly associated with severe brain injury, cranial reconstruction is often delayed until ICP issues are resolved
- Fractures involving the facial bones often require reconstruction for cosmetic and functional concerns



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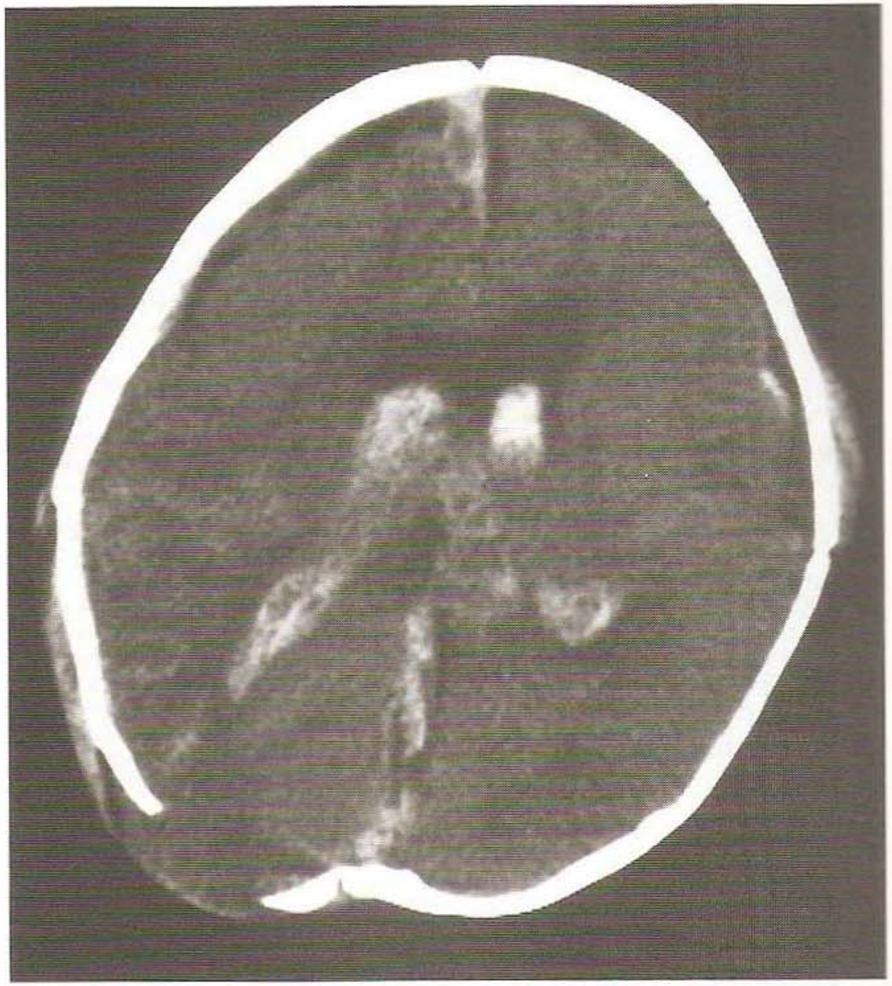


D

Figure 45-3. *(continued)* **(C)** A three-dimensional reconstruction of a CT scan showing a comminuted fronto-orbital fracture. This fracture was successfully reconstructed using absorbable microplates. **(D)** A "burst" fracture, demonstrated on a CT scan of a severely beaten infant. These types of injuries are associated with severe brain injuries. Survivors of the brain injury will need to undergo reconstruction of the dura and the calvarium.

Compound Fractures

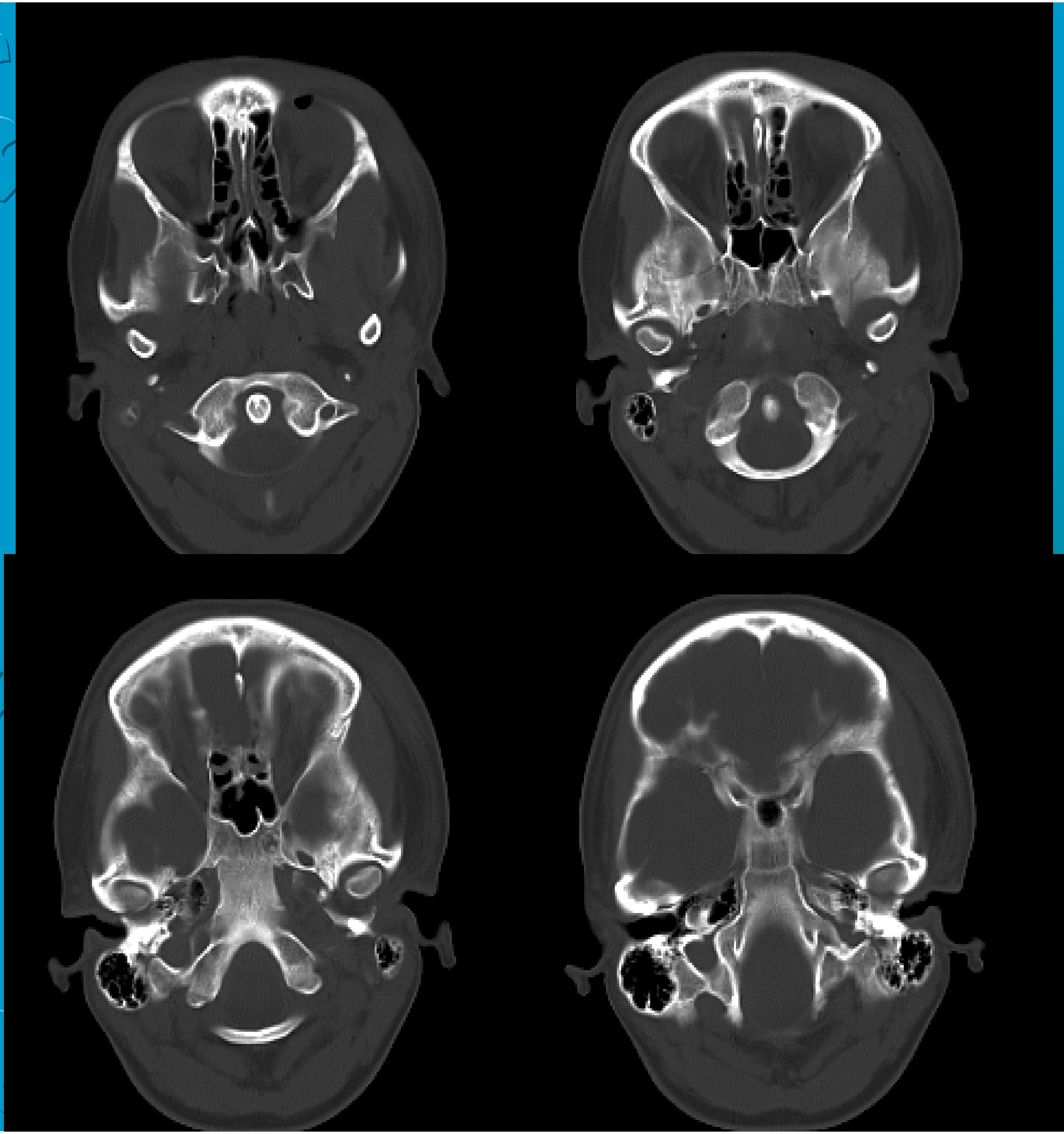
- Contaminated by exposure to the environment
- Include open fractures, but also those involving the paranasal sinuses and middle ear

Frontal Sinus Fractures

- Frontal sinus does not appear until 8-10 years of age
- Significant for intracranial issues only when posterior wall is involved
- May be accompanied by rhinorrhea or pneumocephalus
- If dural compromise persists, then surgical intervention is required

Basilar Skull Fractures

- Can be associated with injury to the vasculature, cranial nerves, or ocular/auditory structures
- Cannot be diagnosed on plain films-rquires high-resolution CT
- Occur in <10% of children with head injuries



Presentation

- Anterior fossa fractures associated with periorbital swelling/ecchymoses (Raccoon’s eyes), more commonly associated with rhinorrhea
- Temporal fossa/petrous fractures accompanied by ear pain, swelling, ecchymoses (Battle’s sign).
  - May see hemotympanum or external canal hemorrhage
  - May involved otorrhea or rhinorrhea (through Eustachian tube)
  - May have decreased hearing

Temporal Bone Fractures

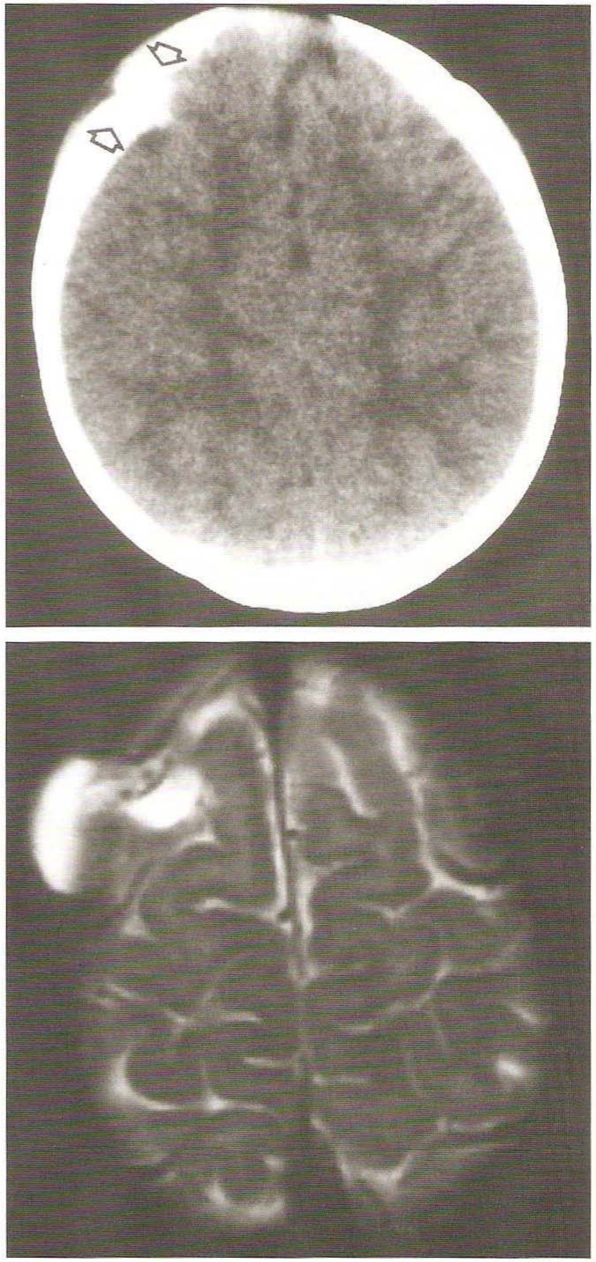
- Longitudinal
  - Extends anteriorly along petrous ridge
  - Not associated with long-term complications
  - 80% of fractures
- Transverse
  - Extends through petrous bone, can disrupt cochlear/vestibular structures
  - Often associated with permanent hearing loss/vestibular dysfunction/facial nerve palsies
  - 20% of fractures

Management

- ENT consult to assess hearing/facial nerve deficits
- Observation for possible rhinorrhea/otorrhea-may present days or weeks after injury
- No indication for prophylactic antibiotics
- Conservative management if CSF leak occurs (almost all resolve spontaneously)
- If necessary, lumbar drain can be placed for a few days, rarely surgical intervention is required

Growing Skull Fractures

- Complicate less than 1% of childhood fractures
- Caused by dural tear followed by persistent CSF leak/cyst that prevents bony healing
- Treated by surgical repair of dural defect
- Can be diagnosed clinically in 4-6 weeks follow-up after skull fracture



A

B

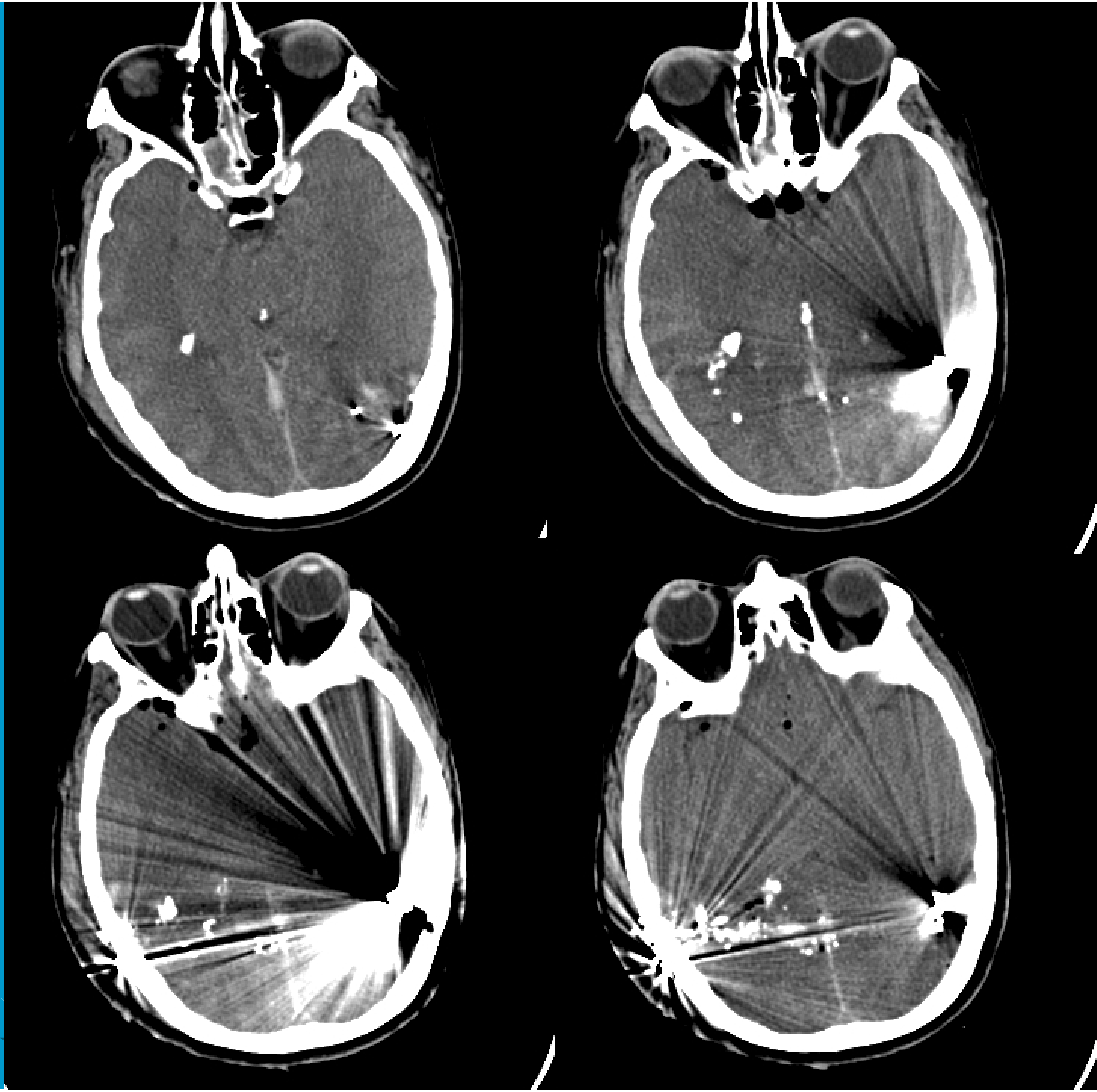
Figure 45–5. A growing skull fracture. **(A)** The initial CT scan shows the fracture and the underlying brain injury (arrows). **(B)** Within a few weeks, the T2-weighted axial MR scan confirms the development of a leptomeningeal cyst that has erupted through the fracture site.

Penetrating Injuries

- Common causes
  - Accidental injury with sharp objects
  - Warfare
  - Accidental shooting
  - Suicide
  - Homicide

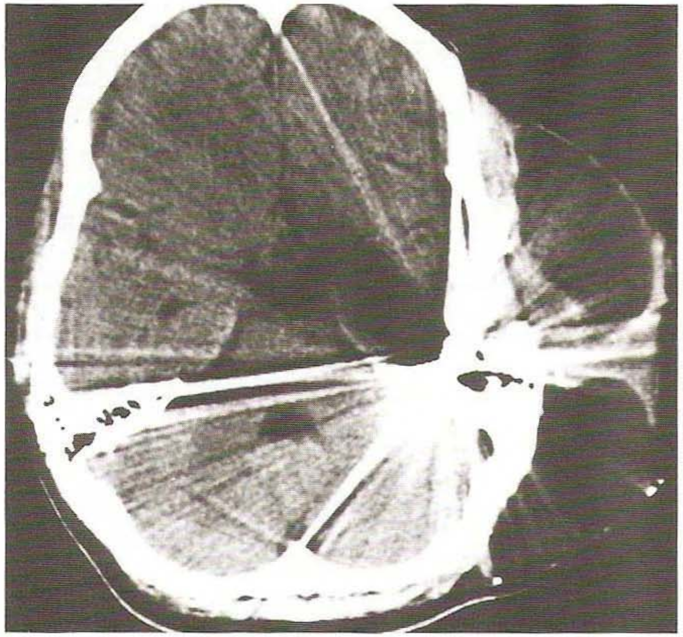
Presentation

- ABCs usually addressed in the field
- Usually have a focal neurologic deficit associated with tract of injury
- If brainstem is involved, may include altered consciousness
- Associated injuries may affect hemodynamic stability, and patients must be screened by the trauma surgery service
- Excessive hemorrhage may be noted if the patient has developed DIC due to release of thromboplastin from injured parenchyma



Management

- CT scan is study of choice
- Frequent neurochecks to monitor for developing mass lesions or increased ICP
- Follow-up CT at 6 hours (delayed hematomas most commonly occur 3-8 hours after initial injury)
- Surgery indicated
  - To remove foreign bodies to prevent secondary complications (infection, post-traumatic aneurysm, seizures)
  - To prevent further bleeding, edema and gliosis
  - To eliminate mass effect
  - Placement of intracranial pressure monitor
  - GCS>5
- Administration of antibiotics in the early period is variable. If desired, choice of antibiotic should cover appropriate contaminating organisms
- Contrast-enhanced imaging indicated in febrile patient to assess for possible abscess development
- Seizure prophylaxis for early seizures is effective, but patients are at increased risk (up to 50%) of late seizures



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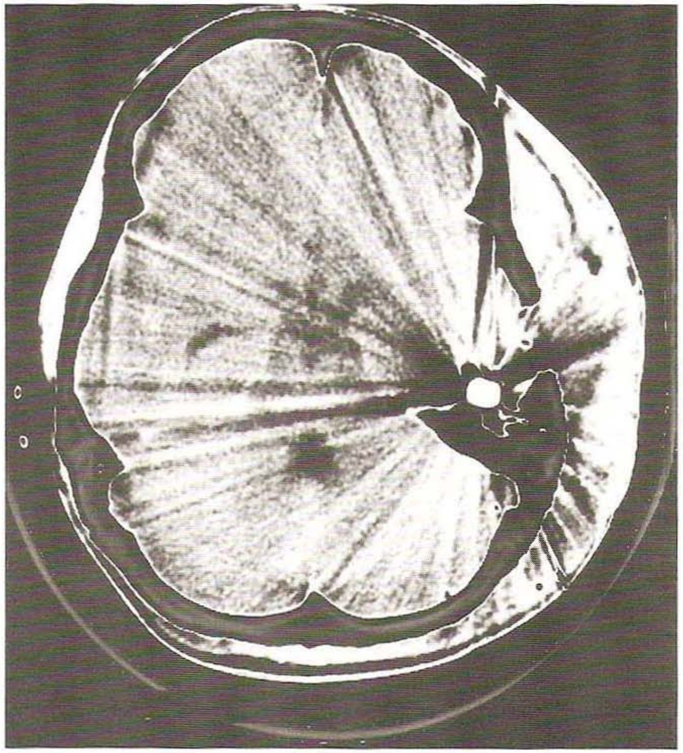

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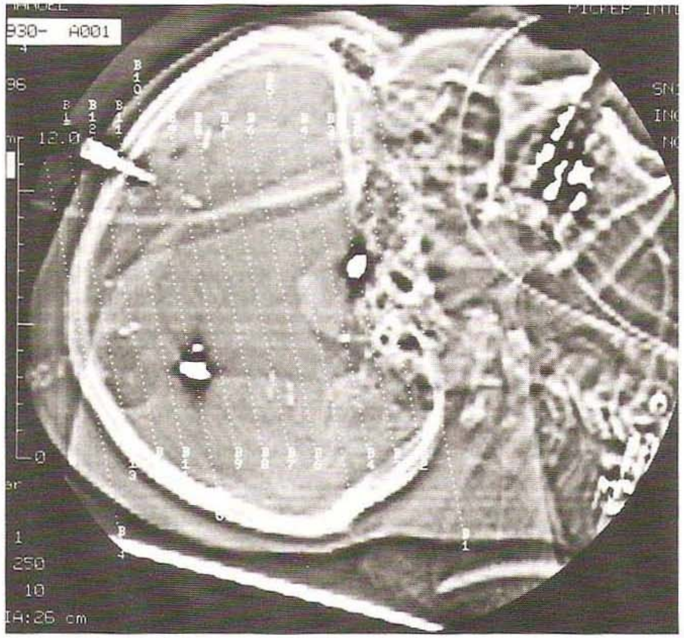

C

Figure 46–10. **(A)** Unenhanced CT scan demonstrating shrapnel lodged in the petrous ridge with scatter artifact. **(B)** After bacterial meningitis, a pseudomeningocoele developed at the site of earlier debridement without dural closure. **(C)** Note the high parietal entrance site and the additional shrapnel. A ventricular catheter is also present.

ICU Management

- Goals are to
  - Optimize substrate delivery and cerebral metabolism
  - Prevent herniation
  - Target mechanisms involved in secondary injury

Initial Resuscitation

- Must address ABCs first
- Hypoxia and hypotension increase morbidity and mortality
- Rapid-sequence, neuroprotective intubation for
  - GCS<10
  - Drop in GCS of 3 points
  - Anisocoria >1mm
  - C-spine injury compromising breathing
  - Apnea
  - Hypercarbia (PCO2>45)
  - Loss of gag reflex
  - Spontaneous hyperventilation with PCO2<25

Initial Resuscitation

- Cardiovascular assessment for adequate perfusion
- Resuscitation fluid should be isotonic crystalloid, followed by colloid and or blood

Worsening neurological status

- Sedation
- Seizures
- Expanding mass lesion
- Cerebral edema
- Hyponatremia
- Vascular dissection with CVA

Intracranial Pressure Monitoring

- No specific guidelines for children, but is reasonable to use adult guidelines
  - Abnormal CT and GCS<9
  - Abnormal neuro exam with normal CT, complicated by hypotension or posturing

Figure 44–3. MR imaging (HASTE sequence) 1 day after injury in a 13-year-old boy with diffuse axonal injury. Note the large right epidural hematoma, which was not present on the initial CT scan. The second study was obtained because of progressive significant elevation in ICP, which is not typical for "pure" diffuse axonal injury in children.

Cerebral Blood Flow Monitoring

- Stable xenon-enhanced CT
  - Shows regional differences in blood flow, can be used to assist in management decision
- Radioactive xenon imaging
  - Provides some real-time data for detection of changes in regional flow, but cannot assess anatomical abnormalities
- Transcranial doppler
  - Only assesses flow through distal ICA/MCA distributions
  - Limited utility in trauma

Monitoring Cerebral Metabolism

- Jugular venous saturation has not been studied in children, may be technically difficult due to smaller vessels
- Intraparenchymal PO2 monitor in adults can be used in clinical management of ICP/perfusion-only provides focal information, and is invasive
- PET imaging limited by long acquisition times, more useful after patient has stabilized to predict recovery

Maintenance

- Goals are to maintain cerebral perfusion
- CPP can be lower than in adults
  - 40-50 in infants/toddlers
  - 50-60 in young children
- BP support with pressors and inotropes
- CVP/cardiac output monitoring

Sedation/Paralysis

- Ideal to use short-acting agents to allow for neuromonitoring
- Use agents that do not increase ICP
  - Narcotics
  - Benzodiazepines
  - Small doses barbiturates
- Paralysis has been associated with increased nosocomial pneumonia and longer ICU stay
- Increased doses of sedatives/analgesics during routine care procedures to prevent agitation

CSF Drainage

- Only possible with catheter placement, may be technically difficult in a child
- Allows for therapeutic treatment of increased ICP
- Does increase risk of meningitis/ventriculitis

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Head Positioning

- HOB 30o decreases ICP
- Midline positioning improves venous drainage

Osmotic Agents

- Mannitol
  - Dehydrates brain parenchyma due to blood-brain barrier
  - Improves rheology, and allows for decreased arterial blood volume with autoregulation
- Hypertonic saline
  - Perhaps has less renal toxicity
  - Used more commonly in children to manage ICP-Na+ up to 170 without evidence of adverse effects in multiple studies
  - Can be administered as a drip or bolus infusion

Hyperventilation

- Can be used for short-term vasoconstriction to assist in ICP management
- Should not maintain PCO2<30mm Hg due to risk of ischemia
- Should prevent hypercarbia

Barbiturates

- Used to manage refractory increased ICP by decreasing cerebral metabolic demand
- Should have continuous EEG monitoring- endpoint is burst suppression
- Increases risk of hypotension and nosocomial pneumonia

Hypothermia

- Contradictory results in many studies due to increased risk of complication
- Use of moderate hypothermia (32o for 24-48hrs) may be used for refractory increased ICP
- Prevention of hyperthermia more important

Decompressive Craniectomy

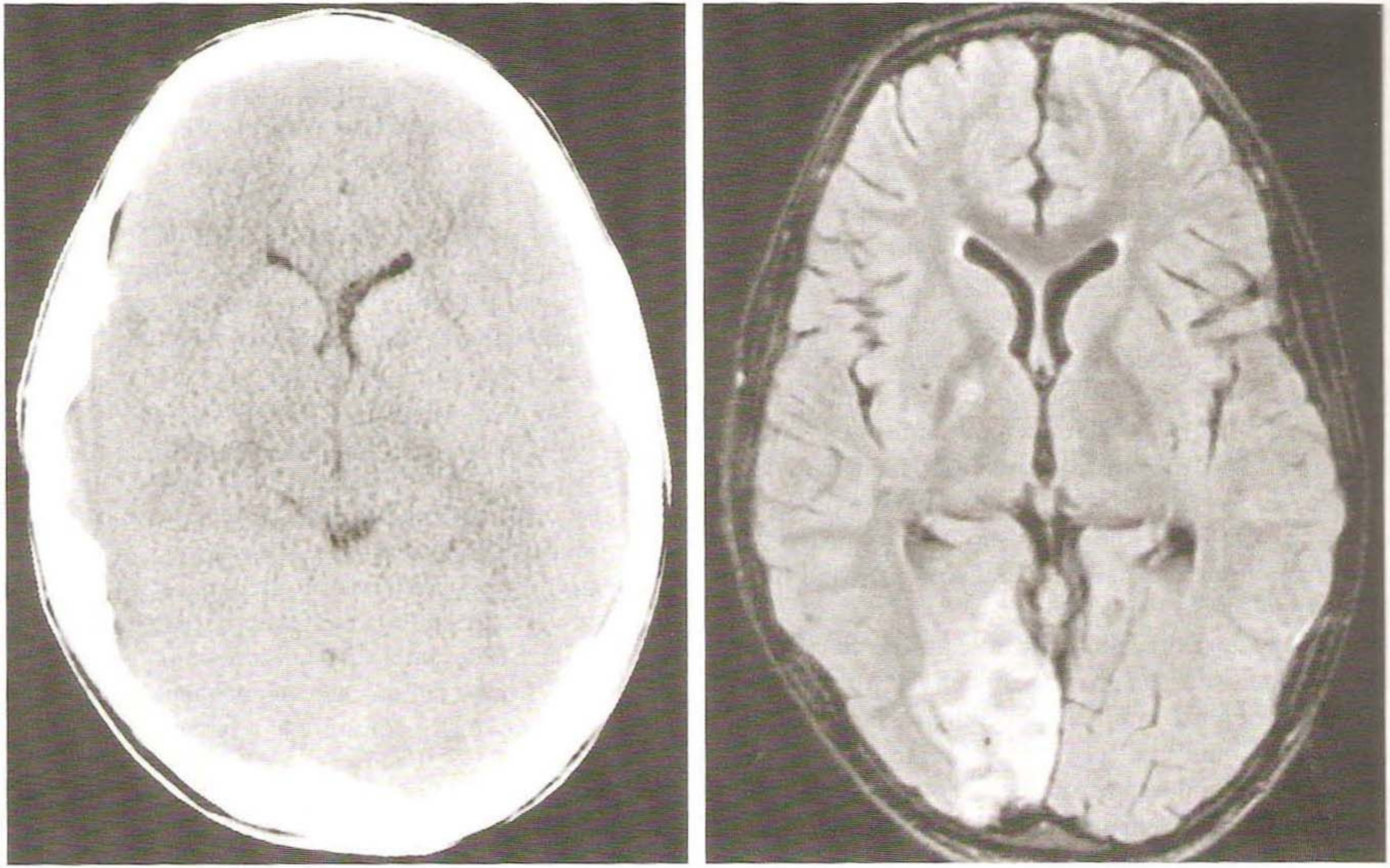
- No specific guidelines for use
- Can reduce ICP, better outcomes likely if performed early (<48hrs after injury)

Controlled Arterial Hypertension

- If autoregulation is intact, mild hypertension (100-140torr) induces vasoconstriction, and reduces ICP in adults
- Data about specific values is not available for children (who have lower baseline pressures)
- Unknown effects on development of edema, possible worsening of hemorrhage

Complications

- Seizure prophylaxis in severe traumatic brain injury
- Prevention of hyponatremia (can be due to SIADH or cerebral salt wasting)
- Nutrition support
- Glucocorticoid use in NOT indicated in head injury



A B

Figure 44–1. **(A)** Initial CT scan of a 9-year-old boy who struck his head against a concrete wall while running. He sustained a brief loss of consciousness, regained alertness, but developed headache and intermittent vomiting. On the second postinjury night he became progressively irritable, developed seizures, and then showed extensor posturing with dilated pupils and bradycardia. Serum sodium was 120 mM/l. **(B)** MR scan showing postherniation infarctions in posterior cerebral artery distribution and deep gray matter. Hemiparesis and hemianopsia persist.

Follow-up

- If and when the child's mental status has returned to baseline, and has adequate oral intake, he may be discharged
- Parents should be informed about possible post-concussive symptoms including headaches, dizziness, nausea, irritibility, difficulty with memory/concentration
- Patients may not participate in contact sports until all symptoms have resolved, + 1 week (general consensus, although does not guarantee against increasingly severe head injuries with future impacts)
- Follow-up may be with primary physician or with neurosurgeon

Rehabilitation and Outcome

- Motor and Visual-Motor Deficits
- Language and Communication Deficits
- Behavioral Changes
- Cognitive Dysfunction
- Academic Achievement

Motor and Visual-Motor Deficits

- Observed in nearly all children with moderate-severe injury
- Usually have mild residual deficits
  - Slower motor response time
- Developmental stage at time of injury affects future development-slower if patients injured before skill was fully developed

Language and Communication

- Expressive language more affected than receptive, recovers more slowly
- Young children have persistent deficits in written language
- Discourse most likely to have persistent deficits (not tested by routine assessment measures)

Behavior

- Increase in problem behaviors, decrease in adaptive behaviors
- Confusion, disorientation, agitation, withdrawal, disinhibition
- If pre-existing issues, may worsen even with mild head injury
- Severe injury greatly increases risk of persistent problems
- Young males at highest risk of persistent decline in adaptive behaviors

Cognitive Dysfunction

- IQ often decreased, but is a global measure, and may not detect specific cognitive deficits
- Performance IQ more affected than verbal IQ (may be associated to motor deficits)
- Memory impairment most common deficit-most associated with left hemisphere damage
- Verbal memory deficits may not be as noticeable until adolescence (when normal children develop this skill)
- Attention deficits may worsen memory function-younger children at risk for long-term effects
- Executive functions significantly affected in young children, and with frontal lobe injury

Academic Achievement

- Combined deficits in many areas lead to poor school performance
- Special education services to assist in environmental factors can improve outcomes
- Deficits may become more pronounced as the child grows older, since they have greater difficulty attaining new skills

Predictors of Outcome

- Injury Variables
  - Severity
  - Type and Extent of Injury
  - Secondary Injuries
- Pre-injury Variables
  - Age at Injury
  - Behavioral History
  - Premorbid Family Functioning
- Post-injury Variables
  - Family Functioning
  - Rehabilitation services

Primary Treatment

- Limited studies in children
- Inpatient vs Outpatient dependent on severity of injury
- Goals are to reintegrate into home and school setting
  - Prevent secondary complications
  - Retrain lost skills
  - Learn compensatory strategies

Interventions

- Psychosocial
  - Counseling for emotional issues
  - Behavior management with differential reinforcement
- Cognitive
  - Strengthen previously learned skills/patterns
  - Train new behaviors to compensate for impairments

Adjunct Treatments

- Parental Interventions
  - Parental support groups/stress management
- School-based Interventions
  - Home instruction
  - Slowly increase length of school day
  - Special education with classroom modifications
  - Self-instruction training

Conclusions

- Pediatric head trauma covers a large clinical spectrum
- Initial evaluation is important to ascertain extent of injury
- Management is directed toward minimizing any further secondary injury/complications
- Early and intensive rehabilitation after moderate to severe trauma improves long-term outcomes