Crush Injuries to the Head in Children

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ALTHOUGH THE MAJORITY of head injuries in children and adults involve dynamic loading conditions, some patients suffer static loading. Static loading occurs when forces are applied slowly to the head, and it produces a much different pattern of injuries. Crush injuries are usually described in the context of industrial accidents, but in our experience, these injuries are not rare in children. We report a series of seven crush injuries in young children admitted during a period of 29 months and describe our experience in the evaluation and treatment of this complex entity. Patient ages ranged from 15 months to 6 years. In four cases, the child's head was run over by a motor vehicle backing up in a driveway or parking lot. In the three other patients, the static loading occurred when the child climbed or pulled on a heavy object, which then fell over with the child and landed on the child's head. One child with cervicomedullary disruption died shortly after his arrival at the hospital. The others showed varying degrees of soft tissue injury to the face and scalp, with Glasgow Coma Scale scores ranging from 7 to 15. Computed tomograms and magnetic resonance images showed multiple and often extensive comminuted calvarial fractures, as well as subarachnoid and parenchymal hemorrhages. All patients had basilar cranial fractures. There was one cervical spine injury but no major vascular injuries. One child had pituitary transection, four had cranial nerve palsies, and another developed a delayed cerebrospinal fluid rhinorrhea 18 months after injury. All children made good cognitive recoveries, with some having relatively mild fixed focal deficits. Despite their alarming initial history and appearance, children who survive the acute period of a crush injury to the head have a good long-term prognosis, reflecting the ability of the brain and cranium to withstand quasi-static loading even in the early years of life. (Neurosurgery 37:401–407, 1995)

Key words: Basilar cranial fracture, Children, Cranial fractures, Crush injuries, Head injury

Crush injuries to the head are usually reported in adult populations and most often involve industrial accidents. In clinical practice, we find that the vast majority of head injuries are caused by dynamic, or rapidly applied, loading forces that cause the head to be quickly accelerated or decelerated. These are forces that are applied briefly and generally occur in ≤200 ms (6). Of the greatest clinical consequence are large dynamic forces with a significant rotational component, which lead to major shear strains and diffuse disruption of the brain parenchyma itself. Crush injuries, however, are produced by static or quasi-static applied forces, which are defined as those that occur over a longer period of time (>200 ms) and are applied over a large area (as opposed to a point). These static forces squeeze or slowly deform an object, like the cranium, until it is crushed beneath the load. The injuries produced to the head by static forces are quite different from the injuries produced by dynamic forces.

Little has been written about static loading injury in children. Crush injuries in natural disasters, such as earthquakes, have been described, and some children are included, but these situations are uncommon in clinical practice (12). Crush injuries involving automatic garage doors have been reported, but asphyxia is the predominant pathophysiology in these patients (2). More often in neurosurgical practice, static or quasi-static loading injuries occur in children when the patient's head is crushed beneath a moving vehicle or when a heavy object has been pulled down accidentally and has pinned the child's head. In the majority of patients seen in clinical practice, injuries are biomechanically mixed, including a dynamic component and a static component. The me-

chanical tolerance of the cranium of infants and young children to static loading has not been well characterized; therefore, when there is history of a crush injury in a child, it has been difficult to predict the injury types, complications, and expected outcome. Our series includes seven young children with head injuries that involve a major static load component; these children were admitted to our hospital during a 29-month period. Clinical and radiological findings, as well as management and outcome, are discussed.

PATIENTS, FINDINGS, AND OUTCOME

Patient population, mechanisms, and clinical findings

Children ranged in age from 15 months to 6 years (mean age, 48 mo). There were two girls and five boys (*Table 1*). In four cases, the child's head had been run over by an automobile, van, or truck while the vehicle was backing up at low speed, usually on a concrete or asphalt surface. In three cases, the child had climbed or pulled on a heavy object (a 400-lb [180-kg] stone fireplace, a 27-in [68.58-cm] television and stand, and a 100-lb [45-kg] clock); the object had fallen over with the child, and the child's head was pinned under the object.

Patient 4 underwent attempted resuscitation in the field and during transport but appeared clinically brain dead on arrival at the hospital; this child had palpable calvarial trauma, a large scalp avulsion, and an obvious cervical deformity. He died shortly after admission to the hospital, and radiological characterization of the injuries was not obtained. An autopsy revealed a fracture dislocation of the atlanto-occipital joint and cervicomedullary transection.

Three children were unresponsive and apneic at the scene or at the initial treatment facility. One of these children had seizures, but all improved after resuscitation.

When they arrived at the emergency department, all six surviving children localized pain or obeyed commands. Glasgow Coma Scale scores ranged from 7 to 15; however, because of intubation and/or eye swelling that precluded complete

TABLE 1. Patient Data[a]

Patient No.	Age/Sex	Mechanism of Injury	Examination Results[b]	CN	Radiological Findings	Surgery	Outcome
1	15 mo/F	Backed over by truck	Motor score 5, eyes swollen, intubated (seizures and unresponsiveness at scene)	III, VI, VIII	Fxs orbits, frontal, ethmoid, sphenoid, temporal bones; frontal and suprasellar hemorrhage	Repair orbital fxs, dural tears	Diabetes insipidus, development near age level, ptosis, amblyopia
2	4 yr/M	Climbed on 400-lb (180-kg) stone fireplace front, which fell over onto patient's head	GCS 8 (intubated), eyes swollen (apnea at scene)	None	Fxs frontal bone, orbits, ethmoids	Elevation fx, dural repair	Hyperactive (pre-injury), returned to baseline
3	6 yr/M	Pulled 27-in (68.58-cm) television and stand over onto head	GCS 12, CSF rhinorrhea and otorrhea	VI, VIII	Fxs occiput, including foramen magnum and clivus, orbits, ethmoids, temporal bone	Transnasal endoscopic repair of meningocele, LP shunt	Down's syndrome, returned to baseline; hearing loss
4	4 yr/M	Backed over by car	Clinically brain dead, massive scalp swelling and avulsion	Unknown	Cervicomedullary transection at autopsy		Died
5	34 mo/F	Pulled 100-lb (45-kg) clock over onto head	GCS 15, periorbital swelling	None	Fxs frontal bone, orbits, ethmoids	None	Hyperactive (pre-injury), returned to baseline
6	22 mo/M	Backed over by van	GCS 10, left hemiparesis (flaccid with agonal respiration at scene)	VI, VII, X	Fxs occiput, including foramen magnum, clivus, temporal bone; ligamentous hemorrhage, occiput-upper cervical spine; sigmoid sinus occlusion; cerebellar and pontine hemorrhage	None	Developmental skills range from age level to a few months behind, mild facial weakness, left vocal cord paralysis
7	6 yr/M	Backed over by small bus	GCS 10 (intubated)	II, III, VI, VIII	Fxs orbits, sphenoid, facial bones, mandible; epidural and orbital blood	Repair facial fxs	At cognitive baseline, normal vision, mild hearing loss

[a] CN, cranial nerve; Fx, fracture; GCS, Glasgow Coma Scale score; CSF, cerebrospinal fluid; LP, lumboperitoneal.
[b] Results of the physical examination performed when the patient was admitted to the hospital.

assessment, these scores did not always accurately reflect the neurological status. All children had marked to massive soft tissue swelling and abrasion, usually involving the face and portions of the calvaria. One child had otorrhea and rhinorrhea, and another had a palpable frontal depression.

Four children had cranial nerve (CN) palsies. One had a VIth nerve palsy; one had unilateral IIIrd and VIth nerve palsies; one had a IIIrd and bilateral VIth nerve palsies and unilateral optic nerve dysfunction; and one had VIth and VIIth nerve palsies and unilateral vocal cord paralysis. In several of these children, periorbital fractures and swelling also contributed to deficits in extraocular motility. In addition, four children with temporal basilar fractures had hearing deficits apparent in the acute period.

Extracranial injuries occurred in three of the surviving children. Patient 1, whose head and trunk were run over by a truck, also had a cervical spine injury at occiput–C1, along with a liver laceration and a sacral fracture. Patient 3, whose head was injured by a television set, also had a fractured clavicle. Patient 7, whose head was run over by a small bus, also had a shoulder fracture.

Radiological findings

All children had multiple calvarial and basilar cranial fractures (*Figs. 1–3* and *Table 1*). These involved the facial bones, the orbits, and the frontal, sphenoid, ethmoid, occipital, and temporal bones. Fractures extended through the clivus or the planum sphenoidale in three patients and through the foramen magnum in two patients. Fractures were comminuted and separated but were minimally displaced in most cases, except in one child with significant depression of the frontal region. None of the radiological examinations of these patients showed optic nerve compression or impingement by bone fragments.

Small extra-axial hemorrhages were common in the epidural, subdural, and subarachnoid spaces. Parenchymal contusions and lacerations underlying fracture sites were also seen, although no hemorrhages had mass effect warranting surgical evacuation. In addition, one child had an orbital hematoma, one had a suprasellar hematoma suggestive of pituitary avulsion, and one had a hemorrhage in the cerebellum and brachium pontis.

The findings on plain cervical spine radiographs were normal in all but one child, in whom the radiographical findings suggested a widening of the occiput–C1 distance. Three children with clinical or computed tomographic (CT) evidence of involvement of the posterior cranial base or cervical region underwent magnetic resonance (MR) imaging and MR angiography. The large vessels all appeared patent without evidence of dissection or other abnormality; Patient 6 had a sigmoid sinus occlusion. MR images showed that Patient 6 also had a widening of the occiput–C1 distance and had ligamentous hemorrhage in the posterior cervical spine at this level.

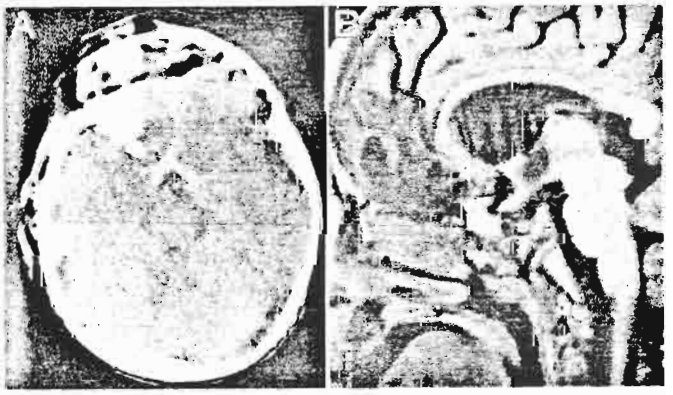

FIGURE 1. Patient 1. *A*, CT scan on the day of the injury shows the frontal fractures, the intracranial air, and the intraparenchymal and suprasellar hemorrhage. *B*, sagittal T1-weighted MR image obtained 5 months after the injury shows the frontal encephalomalacia and the loss of the high signal previously seen in the region of the posterior pituitary gland.

Hospital course and surgery

The patients were admitted to the Trauma Service and managed by a multidisciplinary team, including staff members from general surgery, critical care, neurosurgery, plastic surgery, otorhinolaryngology, ophthalmology, and orthopedics. A physician from the Neurosurgery Division assumed primary care when a significant underlying brain injury was suspected. All children were managed with cervical spine immobilization until a spine injury was ruled out by clinical or radiological criteria.

Patient 1, whose CT scan showed suprasellar hemorrhage, developed diabetes insipidus during the 2nd day after injury, but the diabetes insipidus resolved after 2 days. On the 3rd day after the injury, during the surgical repair of the orbital fractures, surgeons noticed that cerebrospinal fluid (CSF) was emanating from the inferior frontal fracture sites. Frontotemporal craniotomy and exploration revealed a large dural tear extending from the lateral aspect of the inferior frontal region to the cribriform plate. Dural repair was accomplished with temporalis fascia, and a vascularized pericranial graft was used to cover the fractures at the frontal floor. After several days of hyponatremia, permanent diabetes insipidus developed and has required continuing management with desmopressin acetate.

Patient 2 underwent early surgical repair of a depressed frontal fracture and dural laceration extending through the frontal cranial base and cribriform plate; this was managed with a vascularized pericranial graft to the floor of the frontal fossa. Patient 3, who had presented initially with transient otorrhea and rhinorrhea, developed late rhinorrhea 18 months after the injury. Iohexol cisternography revealed a small nasal meningocele, which was the source of the CSF leak. During surgery, a lumbar spinal catheter was placed, and fluorescein was injected intrathecally. The leak site was visualized by transnasal endoscopy, and the meningocele was reduced by bipolar coagulation and nasal packing. After several days of lumbar drainage, a lumboperitoneal shunt was placed.

Patient 5 required no surgical intervention for her multiple cranial fractures. Her pneumocephalus resolved, and her clinical recovery was uneventful, with persistence of her baseline hyperactivity.

Patient 6 remained intubated for airway control, both because of local swelling and because of decreased mental status. He underwent intracranial pressure monitoring measured by ventriculostomy. A mild left hemiparesis was present. At the time of the injury, he was treated with cervical immobilization and doses of methylprednisolone appropriate for spinal cord injury. MR images demonstrated pontine and cerebellar hemorrhage, as well as ligamentous hemorrhage at occiput–C1. Intracranial pressure was never significantly elevated, and the child improved with respect to mental status during the 1st week after the injury. Unilateral vocal cord paresis became apparent after he had improved sufficiently for extubation. He was maintained in a hard collar for 4 weeks, at which time flexion/extension spine radiographs showed no instability.

Patient 7 was treated at the time of the injury with methylprednisolone for possible optic nerve injury, which was manifested by an afferent pupillary defect and decreased acuity. His vision improved, and he underwent uncomplicated elective repair of orbital fractures on the 6th day after the injury. However, 2 days after surgery, he developed hyponatremia (lowest serum sodium, 117 mmol/L) with lethargy and seizures. He required intubation and aggressive medical management of fluids and electrolytes, and he received corticosteroids for possible pituitary insufficiency.

None of these children were treated with prophylactic antibiotics for CSF leaks, and none developed meningitis or other infectious complications. No clinically apparent delayed vascular complications occurred (e.g., stroke, traumatic aneurysm, or symptomatic sinus thrombosis).

The two youngest children (Patients 1 and 6) underwent inpatient rehabilitation to assist them in regaining their motor and other developmental skills. All other surviving children were discharged to their homes; outpatient follow-up was performed by the appropriate physicians, including staff from the otolaryngology, ophthalmology, endocrinology, and other pertinent services.

Outcome

The mean duration of follow-up was 26 months (range, 10–39 mo). A neuropsychological or developmental evaluation or school assessment was performed for each child. Of the three children who pulled heavy objects onto their heads, one had Down's syndrome, and two were considered hyperactive by their families before the injury. Post-treatment evaluations showed that all three had returned to their cognitive and behavioral baselines. The three children who survived being run over by vehicles also have done well, with most returning to their developmental baselines and all continuing to make appropriate milestones. Patient 6, who was one of the more severely impaired at the time of the injury, initially showed significant motor and expressive language delays; these skills have continued to improve, and 18 months after injury, his skills are at or just a few months behind his age level. In all these children, more subtle neuropsychological deficits may become apparent later in life; this is particularly true for Patient 1, whose follow-up studies show frontal lobe encephalomalacia.

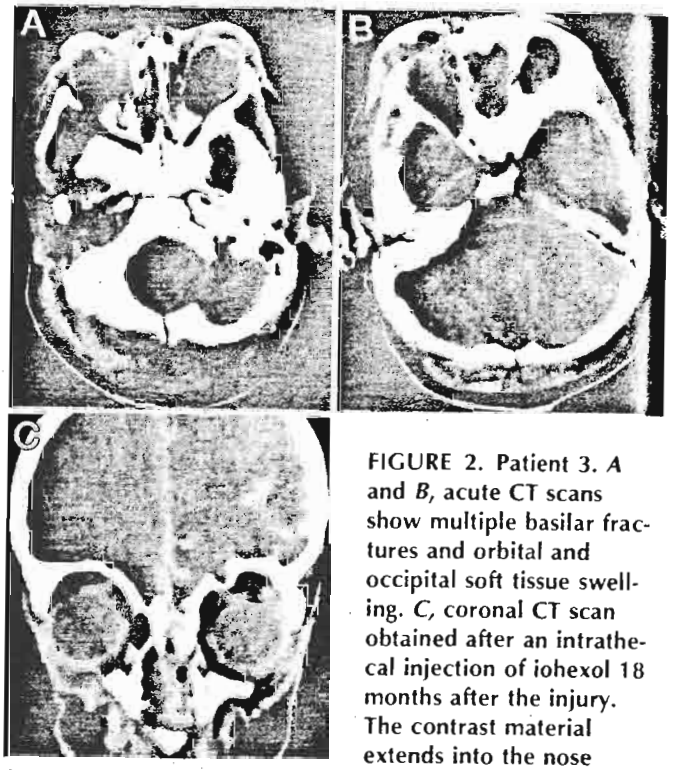

FIGURE 2. Patient 3. *A* and *B*, acute CT scans show multiple basilar fractures and orbital and occipital soft tissue swelling. *C*, coronal CT scan obtained after an intrathecal injection of iohexol 18 months after the injury. The contrast material extends into the nose through a defect in the left side of the ethmoid bone.

Extraocular motility eventually returned or significantly improved in all children, as did facial function in the child with a nerve palsy in CN VII. Vocal cord paralysis (one patient) and hearing loss (three patients) have persisted. Patient 1 developed amblyopia caused by ptosis; this condition responded to eyelid elevation and patching. Patient 7, who had decreased unilateral visual acuity (caused by optic nerve injury) at the time of his accident, has now fully recovered his visual acuity.

With respect to endocrine dysfunction, Patient 1, who had a pituitary stalk avulsion, developed permanent diabetes insipidus requiring desmopressin acetate (see the discussion of Patient 1 in the following section). In a second child with hyponatremia and possible steroid deficiency, steroids were tapered off 2 months after injury; this patient has not required further endocrine intervention.

DISCUSSION

Crush injuries involve the application of force over a relatively prolonged interval (usually >200 ms) and over a wide area. In the patients described here, as in most injuries seen in the clinical setting, the forces involved most likely included both a dynamic component and a static component. The children whose heads were run over by a vehicle were first thrown to the ground, which involves a dynamic load, and then suffered injuries from

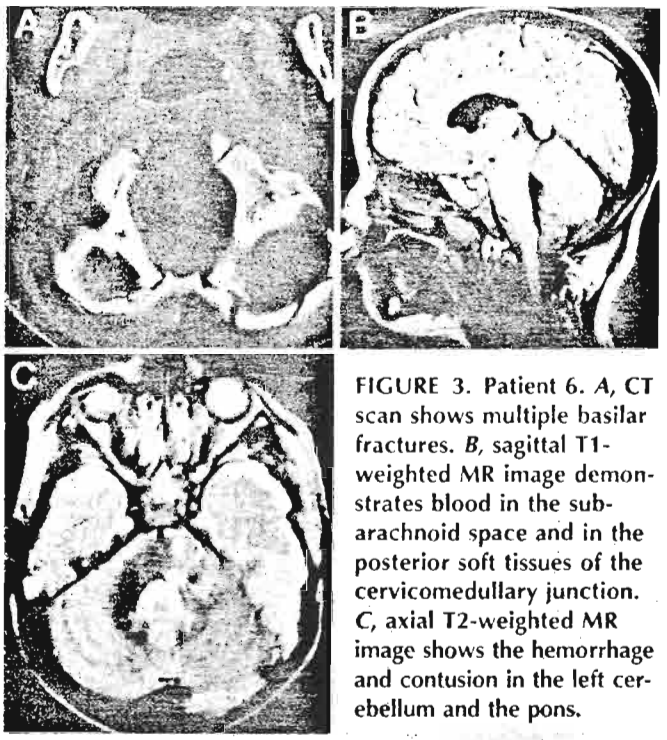

FIGURE 3. Patient 6. *A*, CT scan shows multiple basilar fractures. *B*, sagittal T1-weighted MR image demonstrates blood in the subarachnoid space and in the posterior soft tissues of the cervicomedullary junction. *C*, axial T2-weighted MR image shows the hemorrhage and contusion in the left cerebellum and the pons.

the static load. Likewise, the children who pulled heavy objects onto their heads first fell for a short distance along with the object, involving mainly translational dynamic forces, and then they were further injured by a more prolonged application of a static or quasi-static force. Although we cannot know exactly the magnitude of the dynamic component that occurs when the child struck the ground or was struck by either the moving vehicle or the falling object, the patterns of injury suggest that the significant static load associated with these heavy objects was a major determinant of the specific injuries seen. All these children sustained multiple fractures through the cranial base, which is the pattern most commonly described in adult crush injuries, and most of them had relative preservation of the brain itself (6).

Basilar fractures in children have been reported most often when the children are passengers or pedestrians involved in motor vehicle accidents; these have been estimated to occur in 6 to 14% of pediatric head injuries (7, 9). Multiple basilar fractures are rare under these inertial conditions. However, the fractures associated with crush injuries occur both at the site of contact of the crushing object and remotely, because the force is transmitted throughout the cranial base. This phenomenon explains our finding of fractures through the clivus and foramen magnum under static conditions with little neurological consequence; such fractures are usually associated with significant or fatal brain injury under inertial conditions (3).

This series demonstrates the effectiveness of the cranium in protecting the brain from applied force, even in young children. Although one child died and others suffered some continuing effects, the remarkable degree of brain preservation seen after static or quasi-static loading conditions with objects weighing at least 100 lb (45 kg) is greater than might be anticipated intuitively. This protection is probably afforded by the mechanical properties of both the cranium and the brain itself. In term infants, extreme molding at birth that could result in damaging brain distortion is limited by the interlocking of the cranial bones at the sutures, allowing the cranium to behave as a more rigid encasing structure (10). This interlocking is accomplished by the interdigitation of the bone at the sinusoidal suture lines, which helps prevent extreme overriding of the bone plates. This phenomenon appears to be present in the crush injuries in our series of young children as well, in which the crania sustained multiple fractures, but the bony displacement was limited.

When the cranium is not deformed, it transmits the high pressure exerted on its exterior to the brain tissue inside. Fortunately, because of the mechanical properties of the brain itself, some protection is offered in the setting of static loading by the high bulk modulus of brain tissue, which enables the cranium to withstand very high hydrostatic pressures without deforming. When the pressure or the force is applied nonuniformly and rapidly, especially when rotational acceleration-deceleration forces are involved, the result may be major parenchymal damage, including diffuse axonal injury (4, 6). The children in this series did not sustain this type of injury, but rather their injuries showed parenchymal involvement that reflected focal displacement or deformation beyond the tolerance of the overlying cranium.

The parenchymal injuries seen in these patients were predominantly focal and occurred most often in the cortex in association with an overlying fracture. These contusions and small extra-axial collections did not require surgical evacuation for mass effect. However, such contusions may contribute to long-term morbidity; for example, in Patient 1, significant unilateral frontal encephalomalacia developed. In some children, small scattered areas of abnormality of the deeper parenchyma were also seen (including the basal ganglia, the internal capsule, the cerebellum, and the brain stem) and were related to more remote tissue strains. As in other clinical settings in which small lesions are seen in these regions in children, clinical recovery occurred routinely, and no child had prolonged unconsciousness, which may occur in diffuse axonal injury. Endocrine abnormalities related to shear strains at the cranial base affecting the pituitary axis should be anticipated in a crush injury; these abnormalities can be predicted on the basis of either sphenoid involvement or suprasellar hemorrhage. Such abnormalities may complicate management at the time of the injury, as they did in two of our patients.

CN deficits have been reported in approximately 25% of children with basilar fractures; of these, about one-half are permanent (7). Four of six surviving patients in this series exhibited CN deficits, including CNs II, III, VI, VII, VIII, and X. Some olfactory injuries may have been undetected because of the patient's young age or inability to cooperate. The relatively high incidence of CN palsies is not surprising if we consider the multiplicity of basilar fractures in this group. Although most deficits improved over time, those involving extraocular motility resolved the most completely, with others tending to persist; this finding is similar to results in other series (7).

Although we might expect to see a frequent occurrence of optic nerve injury in association with orbital and sphenoidal fractures from crush injuries, only one patient had evidence of an optic neuropathy. In this series, no patient had an impingement on the optic canal that required optic nerve decompression. In one older child with unilateral decreased vision, rapid improvement was seen with corticosteroid treatment. Whether optic nerve decompression should be considered in patients in whom canal impingement is seen radiographically or in whom vision does not improve with medical management remains controversial; this issue has not been studied thoroughly in children (5, 8, 14).

Considering the frequency of CN injuries, we might expect damage to the great vessels; however, such damage was not encountered in this series, and an asymptomatic sigmoid sinus occlusion was the only vascular complication. MR imaging and MR angiography facilitate screening for vascular and cervical injuries; these studies were a part of the routine evaluation of any patient whose injury was not confined to the frontal vault.

Facial fractures in this series were typical for those occurring in young children, involving, for the most part, minimally displaced and "greenstick" type fractures characteristic of the relatively resilient facial skeleton in this age group (1). In general, a conservative approach is taken toward managing these fractures because the ability to remodel bone seems to be greater in young children than in older patients and because a conservative approach avoids the risk of adversely affecting later growth. In our experience, major facial bone displacement is more likely to occur from a high-speed motor vehicle trauma or a fall from heights, similar to the injuries reported in a series of adult patients (13). In our series, the fractures were repaired in two patients in whom there was bony displacement around the orbit or zygoma that we thought would probably not resolve spontaneously. Major unstable fractures of the midface (i.e., Le Fort III fractures) did not occur, which probably reflects the resilience of the immature facial skeleton to static loading, similar to the resilience seen in the calvarium (1).

Despite the frequent finding of dural tears by clinical criteria, radiological criteria, or surgical findings, only one of our patients (Patient 3) had CSF otorrhea or rhinorrhea; this frequency is less than that reported in other series of pediatric basilar cranial fractures (9). In Patient 9, mild post-traumatic hydrocephalus probably contributed to a pseudomeningocele and the delayed spinal fluid rhinorrhea that required surgical intervention. The use of intrathecal fluorescein was helpful in identifying the precise site of this child's CSF leak and allowed transnasal endoscopic repair. However, intrathecal fluorescein is not without risk, including neurological deficits and seizures, so it should be used with caution, especially when the patient is under general anesthesia and such complications may not be apparent (11).

Cervical injuries in this series were caused by the distraction that occurred when the head and cervical spine were stretched apart as they were run over by a vehicle tire. In Patient 4, this distraction was fatal; however, in the surviving child (Patient 6), late instability did not occur. When the patient's age or level of cooperation limited the clinical examination, MR imaging was helpful in assessing cervical spine injuries.

A coordinated, multidisciplinary approach to these patients is extremely helpful; this approach includes the involvement of staff from general and plastic surgery, otolaryngology, ophthalmology, orthopedics, and rehabilitation. Although the mechanism of injury and the patient's initial appearance may lead to a pessimistic assessment, children who do survive a crush injury have a good outlook for long-term recovery and should be managed aggressively in both the acute and rehabilitative phases.

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COMMENTS

The authors report seven cases of crush injuries to children; in four cases, the child's head was run over by a motor vehicle, and in the other three cases, the child pulled on a heavy object, which then fell on the child's head. The injury sustained was more of a static or quasi-static loading force resulting in a crush injury, particularly in those children whose head was run over, rather than the much more common rapid deceleration or dynamic/shearing type of head injury. These crush injuries result in basilar cranial fractures and often extensive facial injuries. The injury to the brain tends to be much less severe than the very common rapid deceleration type of injury. The message of this series of patients is that the neurological outcome often tends to be much better than one would anticipate at the time of the initial assessment.

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This article by Duhaime et al. focuses on a type of trauma that has received little attention in the literature but is not rare in a pediatric practice. The authors point out the reasons for the injuries and the good survival despite dramatic neuroradiological studies. Their review of the injury dynamics and biomechanics is excellent. The return to function of damaged cranial nerves is high, rather higher than in my experience, and I would hesitate to quote such high figures to the parents of an injured child. The authors had no patients with major vessel injury, but they do discuss the need for at least a magnetic resonance angiogram before surgery when the fractures and injury are beyond the frontal fossa. I think that more emphasis should be placed on the possibility of vascular injury, even in those children who have orbital injuries that may not require neurosurgery. The other specialists involved in the care of these children are unlikely to be aware of the possible vascular injury to the carotid or anterior cerebral artery; thus, the neurosurgeon may have to do that thinking for them.

The authors do not provide much discussion of the ideal timing for the elective correction of these orbital and frontobasal injuries in the comatose child, but most of the children reported did not have prolonged unconsciousness. Usually, the ideal timing is considered to be that point at which the intracranial pressure returns to normal, even if the coma persists.

Finally, the article does not address whether, if surgery is required for superior or lateral orbital deformity, bifrontal craniotomy is necessary as part of that correction to repair dural tears and to reconstruct the cranial base. Unless the injury to the frontal fossa is truly minor, I like to add the frontal craniotomy as part of the initial operation. Such a craniotomy helps avoid cerebrospinal fluid leakage both at the time of injury and later; in addition, it supplies an excellent source of bone for any grafts that might be necessary. The emphasis on a team approach to these injuries is sage and should be followed in all patients.

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ANNOUNCEMENT

1996 Van Wagenen Fellowship Applications

As of July 1995, The American Association of Neurological Surgeons will be accepting applications for the 1996 Van Wagenen Fellowship. The Fellowship application is available to any neurosurgical resident in his or her last year of training who is a citizen of any North American country. The fellowship requirements include that this continued training take place outside of the North American continent for a period of not less than 6 months. The deadline for submission of applications is November 30, 1995.

Applications will be mailed to all neurosurgical residents whose residency training ends in 1996. Additional applications may be obtained via faxed request to Chris Ann Philips, AANS Grants Coordinator, at 708/692-2589.