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## Repair of “Growing” Skull Fracture

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Growing fracture is a rare complication of skull fracture occurring in infancy and early childhood. This late complication of skull fracture is also known as a leptomeningeal cyst. “Growing” fracture is somewhat of a misnomer, but it is characterized by progressive diastatic enlargement of the fracture line. Although skull fracture is a common occurrence in the pediatric age groups, the incidence of growing fracture is only 0.05 to 1% among skull fractures in childhood.

### Patient Selection

The usual presentation of the growing fracture is a progressive, often pulsatile, lump on the head. Neurological symptoms such as seizure, hemiparesis, and mental retardation are less frequent. Often these patients are perfectly asymptomatic, and a palpable mass or widening of the fracture line is the sole sign of neurological sequelae noted incidentally by the parents. Usually a growing fracture develops within a few months following the initial skull fracture, but it may not be recognized for many years. Growing skull fractures usually occur during the first 3 years of life (most often during infancy), and almost never occur after 8 years of age. Although fractures may form in any part of the skull, the most common site for growing fracture is over the skull vault in the parietal region. Dural laceration is always present along the fracture line, and it is an essential factor for the development of a growing fracture. The dural laceration enlarges with the growing fracture. Computed tomography (CT) or magnetic resonance imaging (MRI) often demonstrates a focal dilatation of the lateral ventricle near the growing fracture. Lack of resistance of both dura and skull leads to focal amplification of the pulse wave of the intracranial pressure, causing herniation of the brain or subarachnoid space through the fracture line and the dural defect. The “growth” of the fracture line is caused by bone resorption due to continuous pulsatile pressure at the edge of the fracture line. A rapidly developing infantile brain and associated pathological conditions such as brain edema or hydrocephalus also contribute an outward driving force to cause brain herniation through the dural and skull defect. This pulsatile force of the brain during the period of its rapid growth produces the brain herniation through the dural laceration and fracture line, causing the enlargement of the fracture line of the thin skull.

One of the risk factors for the development of a growing fracture is the severity of head trauma. A linear skull fracture with underlying hemorrhagic contusion of the brain suggests a severe injury, significant enough to cause a dural laceration. Initial CT scans for the evaluation of head trauma in patients who ultimately develop a growing fracture usually reveal significant hemorrhage or contusion subjacent to the skull fracture. When a growing fracture is inspected at the time of surgical repair, the herniated brain is seen to be developing a cerebromeningeal cicatrix. In some cases, loculated subarachnoid cerebrospinal fluid (CSF) cyst(s) may be noted with underlying gliotic, atrophic brain. Although the loculated subarachnoid space may become cystic (leptomeningeal cyst), true leptomeningeal cysts are rare. The cystic changes in the growing fracture usually represent cystic encephalomalacia.

Depressed fractures usually do not cause growing fractures, but a linear fracture extending from the depressed fracture can lead to a growing fracture. The child who on initial x-ray films of the skull has diastasis of the fracture >4 mm is considered to be at risk for future development of a growing fracture. Diastasis of a cranial suture, however, is an unusual site for a growing fracture.

A growing fracture at the skull base can occur in an older age group, especially where the bone is thin such as in the orbital roof, if a linear fracture is accompanied by a dural laceration. Growing fracture and a meningoencephalocele can develop with a similar mechanism as those occurring in the skull vault of the young patient.

### Radiological Studies

X-ray films of the skull show wide diastases of the fracture line. If initial skull films are available, one can compare the films to confirm “growth” of the fracture line during the interval. When multiple fractures are noted in the same patient, healing of the fracture in one area may be noted as opposed to a growing fracture in another area. The fracture line can cross the coronal or lambdoid sutures but is usually limited to one parietal bone.

Neuroimaging such as CT and MRI provide information regarding the sequelae within the growing fracture and any intracranial pathological changes. Furthermore, if they are available from the time of initial trauma, it should be possible to demonstrate progressive changes. It is not unusual that the initial neuroimaging shows hemorrhagic contu-

sion, or subarachnoid or extraparenchymal hemorrhage. At the time of discovery of the growing fracture, neuroimaging demonstrates the diastasis of the fracture line and often cystic lesions near the fracture site. These cystic lesions represent encephalomalacia, a loculated arachnoid cyst, or cortical atrophy. The ipsilateral ventricle tends to show focal porencephalic dilatation with ipsilateral shift of the midline structure. This phenomenon may be due not only to lack of dural resistance but also to cerebral atrophy.

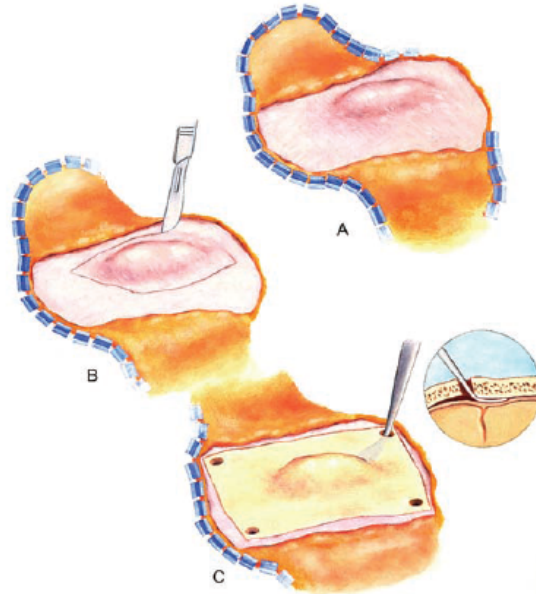
### Management

Surgical intervention is indicated with a growing fracture line, seizure disorder, or progressive neurologic deficits. A progressive cystic degeneration in the brain that has herniated through the dural and cranial defects can occur; therefore, surgical correction is recommended in young children even when seizures or other neurological symptoms or signs are absent. However, incidental, asymptomatic, and stable fractures in late childhood or adulthood probably do not require surgery. The goal of surgery for growing skull fractures is to repair the dural laceration and cranial defect, and to resect seizure foci. Growth of the growing fracture may arrest after CSF diversion shunting by a decrease of the CSF pulse pressure, but this does not correct a seizure disorder. Placing a shunt for primary treatment of these patients is not advised unless hydrocephalus is present. Shunting for nonhydrocephalic patients creates undesirable shunt dependency.

### Operative Procedure

The scalp incision should be large enough to expose the entire length of the skull defect. An S-shaped or semicircular skin incision is made, and the scalp flap is turned subgaleally, leaving the underlying periosteal tissue intact (Fig. 7-1A). By palpation, the entire length of the cranial defect covered by pericranium is exposed in surgical view. The site of the cranial defect is often bulging and may be accompanied by blush appearance due to an underlying subarachnoid cyst. As the cranial defect is dissected by incising the pericranium along the edge of the bony defect (Fig. 7-1B), soft tissues adherent to the edge of the cranium defect are scraped off by a sharp dissector.

The surgeon should remember that the dural edge is invariably larger than the cranial defect, and that the pericranium is directly adherent to the underlying cerebral tissue at the cranial defect. An effort to expose the dural edge by removing the cranial edge should not be undertaken, as this procedure is often complicated by removing the dura simultaneously with the skull bone due to the adhesive nature of the dural edge. To identify the dura, several bur holes are made away from the skull defect with a distance of at least 50% of the width of the cranial defect. At this time, a large enough amount of pericranium is removed from the neighboring skull to use it for repair of the dural defect. Once the dura is identified at each bur hole site, the dura is separated from the inner table of the skull toward the defect (Fig. 7-1C). A craniotomy is made around the skull defect by connecting the bur



**Figure 7-1** (A) The scalp flap is turned subperiosteally. The cranial defect is usually covered by the pericranium. (B) The pericranium is incised along the edge of the cranial defect. Then, the edge of the cranial defect is exposed by scraping off the soft tissues adherent to it. (C) The pericranium is removed from the surrounding skull surface and preserved for dural repair. Four bur holes are made in the surrounding skull for a craniotomy. After the confirmation of intact dura matter under the bur hole, the dura is separated from the bur hole toward the cranial defect. The surgeon should not attempt to identify the dura by removing the bone from the edge of the cranial defect. The craniotomy is performed on both sides of the growing fracture. The two bone flaps are removed and preserved for autologous bone cranioplasty.

holes with a craniotomy. Two pieces of the craniotomy flap are obtained, one from each side of the growing fracture.

After the craniotomy is completed (Fig. 7-2A), reactive periosteal tissue and the cerebromeningeal cicatrix are identified in the dural defect. Under magnified vision by means of surgical loupes, the cicatrix including the periosteal tissue is lifted, and all abnormal tissue is separated and transected using a bipolar cautery until normal white matter is exposed (Fig. 7-2B). The edge of the dura is separated from the cerebral tissue, carefully avoiding trauma to the cerebral blood vessels. In this region, abnormal tissue such as cystic changes or xanthochromic discoloration due to previous hemorrhage is often noted.

After adequate debridement of the cicatrix at the growing fracture and freeing of the intact dural edge from the cortical surface, the dural defect is closed using the periosteal graft (Fig. 7-2C). Autologous pericranium is preferable to cadaver dura. A watertight closure of the dura is important to avoid a recurrence of the growing fracture or postoperative CSF leakage.

Each of the obtained craniotomy flaps is split at the diploic space with an osteotome, separating it into inner and outer tables (Fig. 7-2D). The cranial defect is then repaired



by laying in the split autologous skull grafts. Usually four pieces are laid next to each other side by side to fill the cranial defect. These flaps are secured to each other with either nylon sutures or stainless steel wires through drill holes (Fig. 7-2E). These flaps are further secured to the craniotomy edge. If the defect of the skull is too large or the skull is too thin to separate into inner and outer tables, one may consider autologous rib grafts. These autologous bone grafts are well incorporated, and healing is excellent. Foreign materials such as methyl methacrylate should be avoided for cranioplasty in the growing skull.

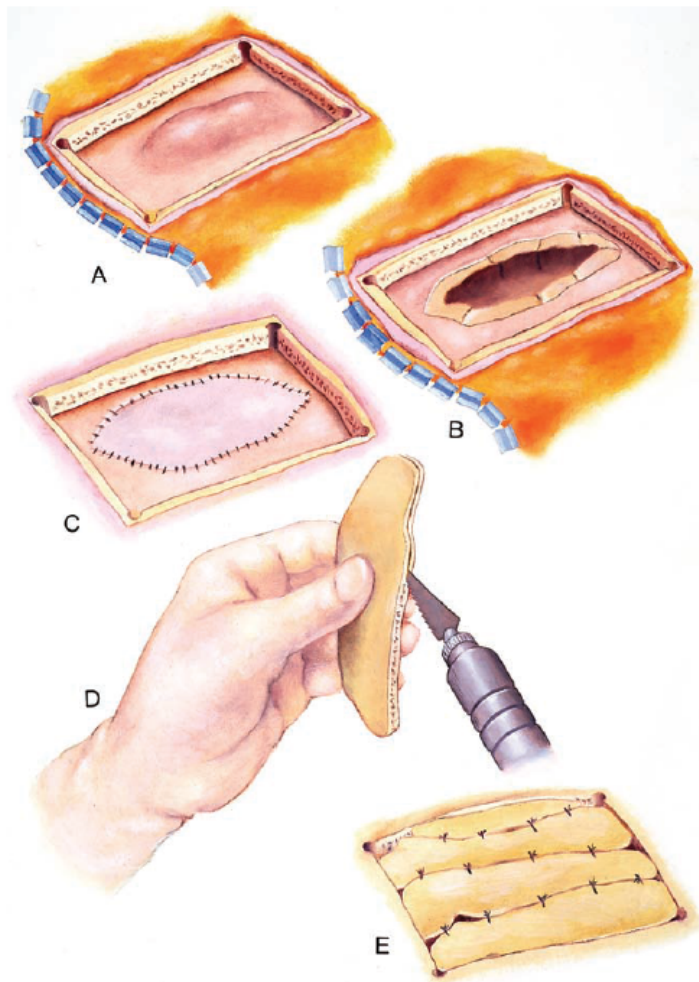
### Specific Considerations

The growing fracture may extend toward a dural venous sinus such as the superior sagittal or lateral sinus. Although these venous sinuses were spared from direct injury at the initial trauma, direct exposure of them is not advised or

necessary. When the fracture line extends perpendicularly to these sinuses, the closest end to the sinus does not need dural repair. However, if the growing fracture is parallel and near to the sinus, dural repair may be difficult due to the lack of enough dural edge next to the sinus. In these cases, one may repair the dural defect with a periosteal graft sutured to the periosteum of the skull above the sinus.

### Postoperative Management Including Possible Complications

CSF diversion shunting has been recommended for persistent postoperative CSF leakage from the craniotomy wound. It is justified if coexisting hydrocephalus is evident, or if CSF leakage occurs despite adequate repair of the growing fracture. A lumboperitoneal shunt or temporary lumbar CSF drainage is to be considered under these circumstances.



**Figure 7-2** (A) After the craniotomy, the intact dura mater is exposed around the dural defect, which is covered by the periosteum. Underneath the overgrowing periosteum is a cerebromeningeal cicatrix that is removed using bipolar cautery and sharp dissection until healthy white matter is exposed. (B) After all pathological tissues have been removed, the edge of the surrounding dura is separated from the intact cortical surface. (C) The previously removed periosteum is used to repair the dural defect. A watertight closure is achieved with 4-0 sutures. (D) The bone grafts are split at the diploic space between the inner and outer tables by means of an osteotome. (E) The obtained split bone flaps are used to repair the cranial defect. The bone flaps are secured to each other and to the edge of the cranial defect with nylon sutures or stainless steel wires.