Bifrontal decompressive craniotomy for massive cerebral edema

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A large bifrontal craniotomy was used in selected patients judged to carry an unusually high mortality risk due to brain swelling secondary to brain trauma. The procedure enabled exploration of both hemisphere convexities, evacuation of accumulated blood and necrotic brain, and decompression of swollen brain. This report reviews 73 cases operated on at the Massachusetts General Hospital since March, 1962; 18% of these patients survived. The largest experience was with post-traumatic cerebral edema, although intractable edema secondary to mass lesions, hemorrhage, toxic encephalopathy, and pseudotumor cerebri was also treated.

KEY WORDS · brain injury · cerebral edema · bifrontal craniotomy

Several methods of alleviating brain swelling can be instituted with varying intervals of effectiveness. Ventricular puncture or hyperventilation may provide precious time to begin supplementary measures. Hyperosmotic agents, urea and mannitol, act promptly to reduce intracranial pressure. The glucocorticoids, methylprednisolone and dexamethasone, suppress elevated intracranial pressure over longer periods.

We do not recommend bifrontal decompressive craniotomy when any of the methods above are sufficient to remove the threat to the patient’s life. It is reserved for patients in whom the likelihood of death is felt to be great in spite of these methods. We no longer use subtemporal decompression for this purpose.

Operative Procedure

The operation is carried out with an endotracheal tube in place. General anesthesia is used in active patients; it may not be required in comatose patients, but adequate ventilation is maintained. In the event that the patient’s level of consciousness rises, inhalation agents may be used. The patient is placed in the supine position with his face up. A large bicoronal skin flap is used, with the limbs placed just behind the normal hair line, extending inferiorly to the zygoma on both sides and curving anteriorly to the midline; it is carried subperiosteally to the level of the supraorbital ridges. Heavy silk sutures are placed at the base, and the flap is retracted forward of the operative field.

The reference points for the bone flap are: a burr hole over the frontal sinus; burr holes in the zygomatic portion of the frontal bone at the anterior insertion of the temporalis muscle; a burr hole 1 cm posterior to the coronal suture in the midline; and two burr holes laterally in the temporal region near the coronal plane of the midline burr hole.
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The burr holes are connected by a saw and the frontal bone removed, ordinarily in two halves. The dura is usually tense and bulging. It is incised bilaterally above the supraorbital ridges to the sagittal sinus anteriorly. Near the pterion the dura is opened in a "Y" shape to facilitate a "fishmouth" opening of the dura. Two heavy silk suture ligatures are placed through the most rostral part of the superior sagittal sinus and tied. The sinus and falx are divided by scissors. Failure to totally divide the falx restricts the frontal expansion of the brain. In severely swollen brains the frontal lobes may shift anteriorly, and the temporal lobes may begin to ease up over the sphenoid wing.

This wide exposure allows identification of contused and lacerated brain. Large acute hematomas may be easily removed under direct visualization. The temporal, parietal, and usually the occipital surfaces can be inspected by careful retraction of the brain with broad retractors or the palm of the operator's hand. Wide visualization of the brain surface is a principal virtue of this procedure. Bleeding points on the cortical surface are controlled with either coagulation or with bits of Gelfoam. The dura is not closed, and the dural defect is covered with frontal periosteum or temporal fascial grafts. The bone flap may be stored in the bone bank or sterilized and replaced at a later date.
temporal muscle, galea, and skin are carefully closed, with care taken that the layers overlap to exclude a direct tract which would invite cerebrospinal fluid leaks.

Several options exist during the course of the procedure, depending upon the patient’s condition and the operative findings. If the situation is one of great urgency, angiography may be omitted because the exploration is adequate without this time-consuming diagnostic preparation. The procedure may be begun by making the temporal incisions and a burr hole or subtemporal craniectomy to explore the lateral surfaces before proceeding to the larger operation. Unilateral rather than bilateral craniotomy may be done.

Postoperatively, close observation is necessary. Patients may require ventilatory and other systemic support. The large bone defect serves as a guide to the status of the intracranial pressure. A finger may be inserted cautiously beneath the dressing for palpation of the tension of the brain. We usually limit fluids to 1000 to 1400 cc per day, often using a .45% sodium chloride and 2.5% dextrose and water mixture. Steroid therapy, instituted preoperatively, is usually continued and hyperosmotic agents used if needed.

A wide variety of methods of cranioplasty have been used in these patients. We currently favor preservation of the bone in sterile packets which are frozen at --30°C and irradiated with electrons to a dosage of 2 million rads. Cranioplasty by any of the usual methods is done about 3 months postoperatively or as judgment indicates.

Characteristics of Patients

In this series of 73 patients, there were 51 males and 22 females with ages ranging from 3 months to 84 years. Traumatic injury affected the largest group of patients (50), whereas subarachnoid hemorrhage (16), gunshot wounds (6), and massive cerebral infarction (1) were less frequent.

Of the 73 patients, six were apneic upon arrival in the emergency ward, six others had irregular respirations, 68 were comatose, responding only to pain or not at all. Decerebrate postures were exhibited on one or both sides by 65. One or both pupils were dilated in 65, and pinpoint fixed in three. The plantar responses were noted extensor in 68 and not noted in three.

Results

In this group of very high risk patients, 13 survived (18%); these cases are summarized in Table 1. An additional 16 patients showed definite neurological improvement following the procedure but succumbed to other causes: cardiac arrest, gastrointestinal hemorrhage, or pulmonary emboli 3 to 38 days postoperatively. Eleven of the survivors were patients suffering from traumatic injury, and the survival in this subgroup was 22% (11 of 50) while in the subgroup of subarachnoid hemorrhage it was 12.5% (2 of 16). No patients with gunshot wounds nor the one with infarction lived. Of the six patients who were apneic preoperatively, two survived. All but one of the survivors had been comatose or had responded only to painful stimuli. Bilaterally dilated and fixed pupils had been present preoperatively in seven survivors. The oldest survivor was 48 years old. At the time of decompression, 33 patients required evacuation of at least one intracranial hematoma, including six of the 13 surviving patients (46%).

The quality of survival was excellent in five patients, the oldest 23 years; they were neurologically intact and returned to work or school. Four patients had some residual deficits but were capable of full self-care and a satisfactory life. Four patients were sent to chronic care facilities.

Of the 60 patients who died, death came within 24 hours for 25 patients, and 87% had succumbed by the 10th day.

Discussion

Cranial decompression for increased intracranial pressure secondary to a mass lesion was performed by Horsley who advocated a large skull defect in the right temporal region and a free dural graft. In 1905 Cushing described a subtemporal decompression in which a 6 x 8 bone defect was created, the dura mater cut in stellate fashion, and the temporalis muscle, fascia, galea, and scalp closed. In 1912 Hudson described the placement of a large osteoplastic flap in the occipitoparietal region for decompression in patients in whom a direct attack was not carried out for mass lesions. Spiller and Frazier advocated palliative operations for brain tumors, recommending that the basal part of the right temporal bone be removed.
### TABLE 1
**Decompressive bifrontal craniotomy in 13 survivors**

<table>
<thead>
<tr>
<th>Age, Sex</th>
<th>Conc.</th>
<th>Respirations</th>
<th>Pupils</th>
<th>Motor</th>
<th>Reflexes</th>
<th>Etiology, Pathology</th>
<th>Postop Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 M</td>
<td>coma</td>
<td>spont.</td>
<td>bilat. dilated fixed</td>
<td>bilat. decereb. rigidity</td>
<td>areflexia, bilat. Babinski</td>
<td>trauma, cerebral contusion</td>
<td>self care, on rehab, R hemiparesis, speech normal</td>
</tr>
<tr>
<td>23 M</td>
<td>resp. to pain</td>
<td>spont.</td>
<td>R dilated fixed, L react.</td>
<td>L hemiplegia</td>
<td>bilat. hyperreflexia, bilat. Babinski</td>
<td>trauma, cerebral edema, contusion</td>
<td>neurol. &amp; mentally normal</td>
</tr>
<tr>
<td>17 F</td>
<td>resp. to pain</td>
<td>spont.</td>
<td>bilat. dilated fixed</td>
<td>bilat. decereb. rigidity</td>
<td>bilat. hyperreflexia, bilat. Babinski</td>
<td>trauma, bilat. cerebral contusion</td>
<td>neurol. &amp; mentally normal</td>
</tr>
<tr>
<td>14 M</td>
<td>coma</td>
<td>apnea</td>
<td>bilat. dilated fixed</td>
<td>bilat. decereb. rigidity</td>
<td>areflexia, bilat. Babinski</td>
<td>trauma, fracture, epidural hematoma, bilat. cortical laceration</td>
<td>neurol. &amp; mentally normal, 64th day: return to school</td>
</tr>
<tr>
<td>12 M</td>
<td>resp. to pain</td>
<td>spont.</td>
<td>R dilated fixed, L react.</td>
<td>spont. movmt. reduced, R more than L</td>
<td>R. hyperreflexia, bilat. Babinski</td>
<td>trauma, fracture, bilat. cortical laceration, cerebral contusion</td>
<td>expressive dysphasia, 58th day: neurol. &amp; mentally normal</td>
</tr>
<tr>
<td>33 M</td>
<td>coma</td>
<td>spont.</td>
<td>bilat. dilated fixed</td>
<td>bilat. decereb. rigidity</td>
<td>bilat. Babinski</td>
<td>subarach. hemorrhage, cerebral edema</td>
<td>alert, oriented, L. hemiplegia</td>
</tr>
<tr>
<td>34 M</td>
<td>coma</td>
<td>spont.</td>
<td>bilat. dilated fixed</td>
<td>bilat. decereb. rigidity</td>
<td>areflexia</td>
<td>trauma, R subdural hematoma, R intracerebral hemorrhage, bilat. cerebral contusion</td>
<td>continuous coma</td>
</tr>
<tr>
<td>14 M</td>
<td>coma</td>
<td>spont.</td>
<td>bilat. dilated fixed</td>
<td>flaccid paralysis</td>
<td>areflexia, bilat. Babinski</td>
<td>trauma, L subdural hematoma, bilat. cerebral contusion</td>
<td>never alert, chronic care</td>
</tr>
<tr>
<td>27 M</td>
<td>coma</td>
<td>spont.</td>
<td>R dilated fixed, L constricted fixed</td>
<td>L. decereb. rigidity</td>
<td>areflexia, bilat. Babinski</td>
<td>trauma, R subdural hematoma, cerebral contusion</td>
<td>no spost movmt, chronic care</td>
</tr>
<tr>
<td>48 M</td>
<td>resp. to pain on R</td>
<td>spont.</td>
<td>bilat. sluggish reaction</td>
<td>R movmt, L hemiparesis</td>
<td>R hyperreflexia, bilat. Babinski</td>
<td>subarach. hemorrhage, cerebral edema</td>
<td>feeding tube, chronic care</td>
</tr>
<tr>
<td>38 M</td>
<td>coma</td>
<td>apnea</td>
<td>bilat. constricted, fixed</td>
<td>flaccid paralysis</td>
<td>areflexia, bilat. Babinski</td>
<td>trauma, cerebral edema, L contusion</td>
<td>home, mild dysphasia, works occasionally</td>
</tr>
<tr>
<td>4 M</td>
<td>semicoma, bilat. purposive movmt</td>
<td>spont.</td>
<td>equal and reactive</td>
<td>bilat. purposive movmt</td>
<td>R hyperreflexia, bilat Babinski</td>
<td>trauma, L cortical laceration, R cerebral contusion</td>
<td>mentally normal, L ptosis</td>
</tr>
<tr>
<td>17 F</td>
<td>coma</td>
<td>spont.</td>
<td>bilat. dilated fixed</td>
<td>flaccid paralysis</td>
<td>areflexia</td>
<td>trauma, L subdural hematoma, pulping frontal &amp; temporal lobes</td>
<td>neurol. &amp; mentally normal, returned to work at 15 mos</td>
</tr>
</tbody>
</table>
The concept of internal decompression with subtotal removal of the mass and dural closure is attributed to McKenzie. Botterel in 1950 reported amputation of the nondominant temporal lobe as a life-saving procedure in patients in whom exploration of the tentorial incisura failed to relieve the herniation. Gurdjian and Thomas in 1964 felt that the craniectomy of Cushing and the craniectomy of others in the temporal area were usually not successful, and they favored combining intracranial mass removal and bone decompression. Clark, et al., reported two cases in which massive total craniectomy was done, but both patients died, and thus evidence favoring use of Clark's procedure is lacking.

We feel that bifrontal craniotomy is a more physiological procedure than most of the alternatives because it allows decompression of both cerebral hemispheres simultaneously with expansion anteriorly and superiorly. It avoids lateral brain shifts and pressure of the incisura upon the midbrain. The bone edge does not wedge in upon the swollen brain and contribute to impaired venous circulation. It facilitates inspection of both hemispheres and removal of acute subdural or intracerebral hematoma as was done in 45% of the patients in our series. In one case, a point of hemorrhage was located posteriorly beyond access via the bone opening, and an occipitoparietal craniectomy was necessary to control a large lacerated cortical artery. Six patients had concomitant intracerebral hemorrhages which may have been undetected by less extensive exploration. Sixteen patients (22%) showed definite neurological improvement but died of other causes. Six patients who died without overt signs of midbrain compression, the decision was made intraoperatively to conduct bifrontal craniotomy because the brains were swollen and further swelling was anticipated. We feel the survivors of this group illustrate the difficulty in making an accurate prediction of death. The precise criteria of selection of patients probably vary somewhat.

The quality of the neurological and mental function of the survivors, we think, has been reasonably good. The number of massively disabled long-term survivors has been small. Because 87% of those who died did so within 10 days, we do not feel that this procedure needlessly prolongs the life of a hopelessly damaged patient. In general, we believe that the younger survivor from a decerebrate state probably has a better potential for good neurological recovery than does the adult, as Robertson and Pollard have noted. However, their experience that patients over 15 surviving decerebration do not make useful recoveries is not borne out in our cases.

We hope that careful analysis of cases will prompt further study of the most feasible application of the procedure. In the interim, we submit the following indications as a guide to the decision to use this procedure:

1. Coma: totally unresponsive or responsive only to deep pain
2. Unilaterally or bilaterally dilated and fixed pupils
3. Apnea
4. Decerebrate posturing: unilateral or bilateral, either spontaneously or in response to a noxious stimulus.

Preferably, at least two of the indications above should be present. In any instance in which a more limited procedure appears to provide adequate provision of additional intracranial space either by virtue of evacuation of hematoma or room for expansion into a defect, we favor withholding bifrontal craniotomy. However, we have come to believe as do Gurdjian and Thomas that limited cranial defects provide trivial volumes of space in patients suffering from massive brain injury and swelling. Nevertheless, we deplore instances reported to us of patients in whom this procedure was used for relatively modest injuries.

We are unable to prove with unequivocal documentation from these cases that their survival is uniquely attributable to the bi-

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frontal craniotomy. In this respect, the procedure resembles many other methods in use for surgical management of brain trauma. We submit that the time is ripe for critical, controlled, and well-documented evaluation of the various indications and methods of clinical management of brain trauma and brain swelling.

We have been encouraged to continue the use of bifrontal craniotomy on the basis of observations that several patients survived after exhibiting apnea and/or dilated pupils, clinical signs commonly preceding death in patients so affected. Our clinical impression that bifrontal craniotomy was useful for severe brain trauma and swelling was at the outset greeted with major skepticism by our neurosurgical colleagues. Over the subsequent years, however, most of our staff has come to the position of endorsing its use, providing it be restricted to the high risk category described. We have been encouraged by the interest of our senior staff and residents to publish this technique and invite its appraisal by other neurosurgeons. We have presented our appraisal of the case material, not so much as proof of its superiority over other methods, but rather as provocation for further critical appraisal of its use.

Summary

Extensive decompression of the brain when needed can be achieved by broadly removing the frontal bone bilaterally, incising the dura supraorbitally, and dividing the sagittal sinus and falx.

In addition to providing an abundance of additional space to accommodate brain swelling, the operative exposure permits an opportunity for unusually thorough exploration of the cerebral convexities. By virtue of the forward displacement of the brain, lateral shifts of the midbrain are minimized as is impingement of the remaining bone edge upon the surface draining veins.

Of the 73 patients exhibiting coma, apnea, dilated pupils, or decerebrate postures, 18% survived.

The procedure is proposed for cases in which death is likely to supervene without effective decompression and as an adjunct to other therapies such as hyperosmolar agents and steroids to combat brain swelling.

References


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