Hemicraniectomy in the management of acute subdural hematoma

JOSEPH RANSOHOFF, M.D., M. VALLO BENJAMIN, M.D., E. LYLE GAGE, JR., M.D., AND FRED EPSTEIN, M.D.

Department of Neurosurgery, New York University Medical Center, 550 First Avenue, New York, New York

Acute subdural hematomas requiring surgical drainage as a life-saving procedure shortly after injury have been associated with mortality rates as high as 90%. Important factors include early diagnosis, complete clot removal, control of active bleeding, and provision for control of subsequent cerebral edema. In a series of 35 patients who had large, unilateral acute subdural hematomas and were unconscious and decerebrate, the mortality rate was reduced to 60%, and 28% returned to normal activity, through the use of a radical procedure. Following establishment of airway and intravenous Mannitol, emergency angiography was carried out to demonstrate the exact intracranial pathology. A hemicranial bone flap was utilized to aid in clot removal and control of hemorrhage from bridging veins and/or cerebral lacerations. The bone flaps were discarded and the dura not closed to afford postoperative decompression. Plating of the skull defects was carried out at a later date when indicated.

Key Words: head injury · acute · subdural hematoma · decompression · hemicraniectomy · cerebral edema · cerebral laceration · intracranial hemorrhage

The destiny of patients with acute subdural hematomas requiring surgical intervention in the first 24 to 48 hours has been quite grim. We define "acute subdural hematoma" as that demanding emergency surgery for preservation of life within 2 days of the initial head injury. Reported mortality rates of up to 80% to 90% are indeed disheartening, particularly in the light of continued advances in neuroradiology, neuroanesthesia, respiratory assistance, as well as the widespread use of cortical steroids, Mannitol, and hypothermia. As evidence of the poor results of surgery we note the statements of some authors who maintain that they do not even operate on the acute clot. While death in these patients occurs as a result of brain-stem compression, torsion, and secondary pontine hemorrhages, only a few show pathological evidence of what might be considered as primary brain-stem injury. In fact, in our experience primary lesions of the brain stem in patients surviving the initial trauma are quite uncommon. Therefore, we have felt that a greater percentage of these patients should be saved through early diagnosis and prompt, adequately-designed surgical intervention.

The numerous factors influencing the outcome in these patients are complexly interrelated and require consideration at this time. As mentioned, if the severity, extent, and location of the initial injury to the brain renders the patient immediately unconscious with signs of severe brain-system dysfunction such as apnea, areflexia, and bilateral fixed dilated pupils, it is highly unlikely that the subsequent removal of a subdural hematoma
Hemicraniectomy for acute subdural hematoma

will affect the outcome. If, however, the individual has not received such an obviously fatal injury, what are the factors contributing to his chances of survival?

Certainly the location and severity of sublethal associated brain injuries, such as contusions and lacerations, will influence the ultimate outcome. Several extracerebral factors are important. Mortality rates appear to rise sharply in patients over 30 years. Associated medical illnesses, including alcoholism, even further accelerate the slope of this curve. Extracerebral trauma, particularly that leading to shock and hypoxia, lower the patient’s chances for recovery.

Time itself plays a role in the prognosis of this group in two quite different ways. The rapidity of clot expansion and accompanying brain compression is important in the eventual outcome of the individual case. Massive brain shifts can be tolerated if the process occurs over a long period of time, while a rapid shift may be fatal. Delay in recognition of the problem creates another time factor.

These patients are all too frequently put through a system of emergency medical care which not only is cumbersome but also costly. This is particularly true for those patients with rapidly developing hematomas whose neurological signs mimic those of a primary brain-stem injury, and this leads to a negative approach to the problem.

In our search for improved therapy for acute subdural hematomas three aspects of the problem seemed important; namely, early recognition, complete clot removal with control of all sources of bleeding, and provision for the inevitable secondary cerebral edema. The diagnostic measures and operative techniques to be described have developed as a result of these considerations.

Although neurosurgeons have at times advocated wide osteoplastic flaps, most have recommended multiple burr holes, craniectomies, and small cranial flaps. In our experience, these small openings have proved inadequate for removal of the entire solid hematoma or controlling the original source of hemorrhage. Moreover, these methods do almost nothing for the compressed and at times lacerated and contused brain or the inevitable associated cerebral edema.

For the past 36 months we have attempted to minimize the time delay in recognizing this clinical situation and once having made the diagnosis have performed extensive exposures and discarded the bone flaps in an attempt to achieve our goals of complete clot removal, hemostasis, and postoperative decompression.

Clinical Material

The patients under consideration were those suffering from unilateral acute subdural hematoma associated with predominantly unilateral underlying cerebral contusions and lacerations in whom, however, the subdural clot was the major factor requiring life-saving surgery within the first 48 hours after injury. Thirty-five patients fell into this restricted category, out of a very much larger patient population of acute craniocerebral trauma. Patients with subacute and chronic subdural hematomas, epidural hematomas, intracerebral clots or diffuse cerebral swelling and contusions were among those excluded from this study.

Evaluation and Treatment

Diagnostic Evaluation

Following the usual emergency measures, including the provision for an adequate airway and for respiratory assistance when indicated by blood gas determinations or on clinical grounds, preliminary neurological evaluation was carried out. Osmotherapy (Mannitol, 3 gm/kg) was administered to patients with alteration in vital signs, abnormal pupillary responses, and/or rapidly progressing neurological signs. In addition, a brief survey of the patient’s general medical status and exclusion of other serious systemic trauma was conducted. Because we believe that cerebral angiography is the most dependable method currently available for the exact definition of intracranial pathology following acute trauma, these patients were then all studied by emergency cerebral angiography. Biplane simultaneous technique was used with the pressure injector set at 220 lb/sq in., 12 cc of renographin being injected. Cross compression studies or bilateral carotid injections were employed when indicated.

Angiographic findings in these 35 patients were typical for acute subdural hematoma. Each demonstrated a superficial avascular area averaging 2 cm thick and covering the entire hemisphere. The avascular area varied
between 1 and 3 cm at its thickest point which often was in the middle fossa. Anterior cerebral shifts averaged 1.2 cm, and internal cerebral venous shifts 2 cm. There were also many instances of displacement of the posterior communicating-posterior cerebral complex which suggested uncal-hippocampal herniations. In addition, 20 instances of associated cerebral contusions and swelling, and 10 examples of traumatic cerebral vasospasm, were noted.

Preoperative Clinical Status

To evaluate the results of therapy it is imperative to know the clinical condition prior to surgery. All patients selected for this operative procedure were not only unconscious, but showed signs of brain-stem dysfunction and alteration of vital signs: 15 patients were bilaterally decerebrate, 17 unilaterally decerebrate, one bilaterally decorticate, while two manifested a mixed picture, at times being decorticate and occasionally showing semipurposeful response to noxious stimuli.

All patients had pupillary abnormalities; 27 had unilaterally dilated pupils, five had bilaterally fixed and dilated pupils, and three had miotic, poorly reactive pupils.

Thirty-one patients had respiratory abnormalities; six were apneic, eight had the typical Cheyne-Stokes pattern, 12 were tachypneic, and five bradypneic. Four patients had a normal respiratory pattern. Blood pressures were quite variable and ranged from 210/100 to 100/60. Pulse rate was extremely variable, ranging above 100 in 22 cases and below 90 in 13 cases.

Operative Procedure

When the clinical diagnosis of a large unilateral subdural hematoma as the primary intracranial mass lesion was confirmed, the following procedure was employed. Under general endotracheal anesthesia, a high temporal burr hole was placed to provide an immediate release of intracranial pressure and, in addition, to exclude the possibility of a subacute hematoma that could be evacuated through a conventional opening. When an acute subdural was encountered, a skin flap was extended from the glabella along the midline, terminating 4 cm above the external occipital protuberance. The skin incision was carried laterally to the level of the transverse sinus, and a one-layer skin flap including the periosteum was turned. A frontoparietal, occipital, and temporal bone flap was then removed to reveal almost the entire surface of the hemisphere; this was rapidly accomplished by the use of the high-speed air craniotome. The temporal squama was rongeured to the floor of the temporal fossa, with the neurosurgeon making absolutely certain that no shelf of bone remained that might prevent subsequent lateral shift of swollen temporal lobe. The bone flap was discarded or placed in the bone bank. The dura was widely opened and hinged at the attachment of the superior sagittal sinus.

Through this exposure it was possible to carry out a complete removal of all solid and liquid hematoma. The inferior surfaces of the frontal and temporal lobes were inspected for areas of clot and contusion. Estimates of the clot volumes varied from 150 to 350 cc. Bleeding from brain lacerations was controlled, and badly macerated brain was resected, if necessary. The bridging veins along the sagittal and transverse sinuses were inspected for active bleeding and were often found to be the source of the subdural hematoma.

When hemostasis was satisfactory, the dura was laid over the surface of the brain, with no attempt at closure. An epidural drain was placed and connected to a closed, sterile drainage system (which was removed in 24 to 36 hours). The scalp was closed in a one-layer on-end mattress technique with No. 30 stainless steel wire.

Postoperative Care

In the postoperative period respiratory assistance was maintained, as indicated by alterations in blood gases. Hypothermia was used only to maintain body temperatures within normal ranges. Antibiotics were given only when needed to combat specific infections of respiratory, urinary, or central nervous system origin. Steroids were not administered in this group; anticonvulsants were.

Results

Fourteen patients in this series survived, giving an over-all survival rate of 40%. Seven of the 35 patients have fully recovered and returned to their normal occupations.
Hemicraniectomy for acute subdural hematoma

Three patients have a residual mild hemiparesis and will be employable. Excellent or good results were therefore obtained in 28% of the entire series. Two patients have severe hemiparesis, and one of these is also aphasic. Two semicomatose patients await transfer to a nursing home for long-term chronic care. In nine patients the cranial defect has been plated with stainless steel wire and acrylic; these secondary procedures have been performed 6 weeks to 3 months following the primary operation.

A comparison of the preoperative findings in the fatal and nonfatal groups brings out the following points of interest. Of the 21 nonsurviving patients, 17 had increased blood pressures ranging from 210/105 to 170/80; the remainder showed relatively normal pressures. The surviving group of patients had blood pressures ranging from 190/100 to 160/60, the impression being, therefore, that the survivors showed slightly lower preoperative blood pressures although not of statistical significance. Pulse rate was variable, with no particular distribution between survivors and nonsurvivors. All fatalities had preoperative respiratory abnormalities: 12 were tachypneic, three had Cheyne-Stokes respiration, one bradypnea, four were apneic, and one had shallow, irregular respirations of a normal rate. The respiratory disturbances in the surviving patients were less severe than in the nonsurvivors. Four were tachypneic, four had Cheyne-Stokes respirations, and four had a normal respiratory pattern. Two surviving patients were apneic for a short period just prior to surgery.

All but three patients had at least one dilated pupil. Among the fatal cases, four had bilaterally fixed, dilated pupils, and 15 unilaterally dilated, non-reacting pupils. In the surviving group, one patient had bilaterally fixed and dilated pupils, 12 had unilaterally fixed, dilated pupils, and three had meiotic, sluggishly reacting pupils. The alternate pupils in this group ranged from sluggish to non-reactive, but were not maximally dilated.

In the fatal group, four patients showed no response to painful stimuli; the remainder reacted to all types of stimulation by exhibiting bilateral decerebration, which at times was spontaneous. In the surviving group, two patients showed almost no response to pain, four exhibited bilateral, and six unilateral decerebration. Two had a mixture of decorticate and semipurposeful response to pain.

Finally, it is worth noting that all patients died who had a clot exceeding 200 cc or who were apneic for any significant period of time; four out of five patients with bilaterally fixed and dilated pupils also died. The four patients who had a normal respiratory pattern survived.

Discussion

Many surgical procedures have been advocated in the treatment of acute subdural hematomas, among these burr holes, craniectomies, osteoplastic flaps, subtemporal decompression, and occasionally temporal lobectomy.\textsuperscript{1,4,6,11,15,18} Despite all efforts, the reported mortality rate has remained at approximately 80% to 90%. In our clinic, a retrospective analysis of 40 patients operated on via burr holes or routine craniectomies over a 5-year-period disclosed a mortality rate of 85%. Those patients who survived surgery improved transiently and then deteriorated 24 to 72 hours later. In most cases, death resulted from the ravages of cerebral edema as well as continued intracranial bleeding with recurrent clots, uncal-hippocampal herniation, and secondary brain-stem compression. In an attempt to overcome these problems hemicraniectomy was undertaken in the present series of 35 patients. With the extensive exposure afforded by this operation, all clot was easily removed, active bleeding controlled, and a large dural and bone decompression provided (Fig. 1).

In many cases when the tamponade effect of the clot was removed, extensive bleeding ensued from the torn veins. However, with the wide exposure, bleeding was rapidly controlled whether it occurred from torn bridging veins adjacent to the midline or from beneath the temporal lobe. In our previous experience with smaller exposures, bleeding from remote areas was occasionally overlooked and was only fully appreciated at postmortem examination when a recurrent collection of blood or clot was discovered.

Postoperative arteriograms in six patients confirmed the clinical impression that the swollen brain was bulging into the decompression; in addition, despite massive hemispheric edema, there was no shift of midline...
Fig. 1. Operative photograph showing hemi-craniectomy exposure of right hemisphere after most of the subdural clot was removed. Note severe temporal lobe contusions and scattered sub-pial hemorrhages. A small amount of residual clot overlies the occipital pole. This patient was actively bleeding from torn bridging veins beneath the temporal lobe.

structures, nor angiographic evidence of uncal-hippocampal herniation. The temporal lobe was consistently shifted upward and outward into the craniectomy, decompressing the incisural notch (Fig. 2).

Of the 35 patients, only four died within 48 hours of surgery from the effects of increasing intracranial pressure. The remaining fatalities survived 5 days to 6 weeks and generally succumbed to the medical complications of prolonged coma. Postmortem examination in four patients who died within 2 weeks of surgery disclosed unilateral hemispheric edema without shift of midline structures. In only one instance was there evidence of brain-stem compression. In each case the swollen brain had been decompressed through the hemicranial defect.

Moody, et al., demonstrated the effectiveness of massive cranial decompression in an experimental model of postcompression cerebral edema. Twenty dogs were severely traumatized with an epidural pressure balloon. Ten were treated with massive bone decompression and the remainder served as controls. In the first group, seven were alive at the end of 10 days. In the control group, all were dead within 12 hours. The brain of each of the surviving dogs had swollen into the decompression.

Steroid therapy for the treatment of post-traumatic cerebral edema was withheld in our patients in an attempt to reduce the number of variables which might account for improved survival rate. Studies by Wood and others suggest, however, that steroid therapy may be of value in the treatment of
Hemicraniectomy for acute subdural hematoma

post-traumatic cerebral edema. In the future we will most likely employ steroids as well as the hemicraniectomy procedure.

It should be emphasized that certain extracerebral factors contribute to the mortality rate in our series. Many of the patients were chronic alcoholics, malnourished, or generally debilitated. These individuals were susceptible to a host of complications, the most severe of which was massive pulmonary infection. Postoperatively, these patients commonly improved neurologically, only to succumb to pneumonia a few days later. It would be anticipated that a greater number of patients would survive in a more heterogeneous population.

Finally, what controls are there to substantiate our conclusion that this method of handling the acute unilateral subdural hematoma with preoperative signs of brain-stem dysfunction is of real value? Obviously, for instance, a series in which the hemicraniectomy flap was turned, the clot removed and hemostasis established, and the bone flap then discarded or replaced on a random basis would be of great scientific merit. We have not yet, however, been able to bring ourselves to this level of scientific objectivity, particularly in view of the fact that without exception every one of these patients demonstrated a very tight swollen hemicraniectomy site for periods of up to 2 weeks after the surgical procedure. We felt that this was relatively conclusive evidence that the decompression was indeed being utilized for the purpose for which it was designed.

With the introduction of hemicraniectomy, our current survival rate has increased from 15% to 40%. We do not feel, however, that any final conclusions can be drawn from this type of comparison. It is obvious that these patients received special attention from the attending resident and nursing staffs as a recognized group of "study patients." This must have contrasted sharply with the same group of patients in the previous years who inevitably were handled with a degree of pessimism that influenced the outcome of their therapy. In the strictest sense, therefore, we are without satisfactory control information to substantiate our clinical impressions concerning the value of this method of treatment. We present this small series of patients in the hope that others will join us in our attempts to further evaluate this procedure.

Summary

Despite all forms of surgical intervention, the mortality rate of acute subdural hematoma has been reported to be as high as 80% to 90%. In an effort to improve these results, we subjected 35 unconscious patients with acute unilateral subdural hematoma to hemicraniectomy (within 48 hours of injury). In this procedure most of the hemicranium on the affected side was removed, providing excellent visualization of the greater part of the involved hemisphere. All blood clot was removed, and bleeding from lacerations and torn bridging veins was controlled. The bone plate was discarded and the dura left open, providing for post-operative decompression.

In this group of patients the mortality rate has been reduced to 60%, and a functional survival rate of 28% has been achieved.

References

5. Darmody WR: Hypothermia for acute subdural hematoma. Paper presented before the 54th Annual Meeting, Society of Neurological Surgeons. Detroit, Michigan, April, 1963
17. Rowbathom GF: Acute Injuries of the Head; Their Diagnosis, Treatment, Complications and Sequels. Baltimore, Williams and Wilkins, 1942