The role of craniectomy in the treatment of chronic subdural hematomas

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A consecutive series of 48 adult patients with a chronic subdural hematoma is reported. These patients
were treated according to a protocol consisting of a sequence of conventional surgical procedures ranging
from simple burr-hole drainage to craniotomy and subdural membranectomy. Seven patients (15%) con-
tinued to demonstrate severe neurological dysfunction, or suffered acute neurological deterioration after
completion of this protocol. However, after undergoing excision of the cranial vault overlying the hematoma
site, six of these seven patients demonstrated a significant clinical improvement. Based on analysis of these
seven cases, the authors suggest that craniectomy be considered in those patients who suffer a symptomatic
reaccumulation of subdural fluid following craniotomy and membranectomy, or who demonstrate further
neurological deterioration as a result of cerebral swelling subjacent to the hematoma site. However, this
procedure probably has no efficacy once extensive cerebral infarction has occurred.

KEY WORDS cerebral swelling chronic subdural hematoma craniectomy head injury recurrent subdural hematoma surgical treatment

FOR those few patients who become critically ill as
a result of a chronic subdural hematoma (SDH), the mortality of this disease remains relatively high. For example, only 2% of the patients in the series reported by McKissock, et al., were comatose immediately prior to surgery. However, the mortality in this small group was 25%, which was more than four times that of the series as a whole. Although newer therapeutic modalities apparently provide reasonable alternatives to burr-hole drainage, none has proven efficacious in the treatment of chronic subdural hematomas that become intractable or life-threatening.

In our series of 48 consecutive adults with chronic SDH’s, we have encountered two groups of patients who may benefit from excision of the cranial vault overlying the hematoma site, despite the presence of coma and signs of brain-stem dysfunction. The first group is comprised of patients who suffer progressive neurological impairment as a result of multiple reac-
cumulations of subdural fluid. The intractability of these hematomas is invariably associated with a fixed, expanded subdural space that is the result of failure of the brain to re-expand following evacuation of the
hematoma. In these cases, craniectomy presumably allows the scalp and dura to invaginate into, and thus obliterate, this residual subdural space. In contrast, the second group of patients who may benefit from craniectomy includes those who demonstrate evidence of cerebral swelling following evacuation of a chronic SDH. In such cases, craniectomy initially provides an external decompression that allows expansion of the swollen cerebrum away from the brain stem, and subsequently permits obliteration of any sub-
dural space remaining after the swelling subsides.

These two clinical syndromes are undoubtedly rare. However, their incidence may be relatively high within the small group of patients who remain in danger of dying despite exhaustion of conventional treatment techniques. This paper presents the results of craniectomy in these two groups of patients with chronic SDH’s.

Clinical Material and Methods

Forty-eight patients, aged 20 to 86 years, received surgical treatment for a chronic SDH in the 6 years since we first incorporated craniectomy into our treat-
ment protocol at Plymouth General Hospital. A sub-
dural hematoma is classified as "chronic" according
to McKissock's temporal criterion. Cranietomy
was performed in seven of these patients (15%).

Neurological Grading

The neurological condition of all patients was
graded at hourly intervals and sequentially recorded
on their charts. The neurological grade represents a
composite of focal signs and the level of con-
sciousness. When these two parameters did not
suggest the same grade, greater emphasis was placed
on the latter. This grading system is comparable to
those used in several previously reported series of
patients with chronic SDH.s,14,16,24 It is as follows:

Grade 1 = patient alert and oriented; spontaneous
conversation and purposeful movements; absent
or only mild symptoms or focal signs (headache
or reflex asymmetry)
Grade 2 = patient drowsy or disoriented; limited
verbal and motor responses to questions or com-
mands; moderate focal signs (hemiparesis)
Grade 3 = patient not conversant; utters sounds
and makes purposeful motor responses to nox-
iou stimuli; severe focal signs (hemiplegia); in-
cludes patients who are awake but demented
Grade 4 = no vocalization; nonpurposeful, stereo-
typed, or absent motor responses to noxious
stimuli; abnormal vegetative functions (abnor-
mal respiratory pattern, anisocoria).

Table 1 shows the 48 patients in this series, classified
according to their preoperative neurological grade.

Treatment Protocol

All 48 patients were treated according to a stan-
dardized treatment protocol based upon changes in
neurological grade and on operative findings. The ini-
tial diagnosis of chronic SDH was made by cerebral
angiography, radionuclide brain scan, or by com-
puterized tomographic (CT) scan.
1. At minimum, posterior frontal, posterior
parietal, and inferior temporal burr holes were drilled,
even if the preceding radiological study suggested a
more restricted lesion. Following drainage of the
hematoma, any residual subdural space was explored
by gently passing a soft rubber catheter between the
burr holes. Incomplete drainage of the hematoma was
inferred from resistance to the passage of the catheter
or from evacuation of a quantity of fluid that the sur-
geon judged insufficient to account for the depth of the
residual subdural space. In such cases a craniotomy
was immediately performed.
2. The subdural space was tapped percutaneously
through the posterior parietal burr hole 72 hours after
surgery. The patient's head was positioned to enhance
gravitational drainage of any residual fluid.
3. Patients whose brains failed to re-expand im-
mediately after burr-hole drainage were kept flat in
bed for 5 to 7 days.
4. Postoperative neurological deterioration that
could not be explained by a cardiorespiratory or
metabolic disorder was evaluated by cerebral
angiography or, more recently, by CT scan. The first
symptomatic reaccumulation of subdural fluid was
treated by reopening the burr holes. The criteria for
immediately proceeding with a craniotomy were the
same as above. A second symptomatic recurrence was
treated by a large craniotomy and excision of the ex-
ternal subdural membrane. The surgical technique
was similar to that reported by Svien and Gelety21 and
by Smyth and Livingston.19
5. A symptomatic reaccumulation of subdural fluid
following craniotomy and membranectomy was treated
by excision of all of the cranial vault overlying the
pathologically expanded subdural space. Care was
taken to place the scalp incision well outside the
margin of the craniectomy in order to prevent wound
dehiscence as the scalp flap invaginated through the
cranial defect.
6. A craniectomy was also performed if at any
point in the hospital course neurological deterioration
was associated with radiographic and/or intraoper-
eative evidence of cerebral swelling. Every effort was
made to decompress the entire surface of the swollen
lobe. Dural closure under tension was avoided by in-
corporating a fascial graft or Silastic sheet implant
into the closure. Patients with cerebral swelling were
also placed on dexamethasone (24 mg/day) for 7 days
and hyperventilated for 36 to 72 hours.
7. Acrylic cranioplasty was offered to these
patients 6 to 9 months after craniectomy.

Clinical Data

The clinical histories of the seven patients who un-
derwent craniectomy are summarized in Table 2. The
mean age of these patients was 63 ± 5 years (SE),
which was significantly older (p < 0.05 by Student's t-
test) than the 41 patients (mean age, 55 ± 1.8 years)
who were successfully treated without craniectomy.
The craniectomy patients also differed from the other
patients in the series in terms of preoperative
neurological grade. Virtually all patients in the series
who became Grade 4 during their hospital course met
the criteria for craniectomy. Indeed, six of the seven
patients treated by craniectomy were comatose im-
mediately before that operation, and the remaining
patient was in Grade 3.

The craniectomy patients comprised two distinct
groups based on operative findings. The first group
(Cases 1, 2, 6, and 7) consisted of patients whose brain
failed to re-expand following burr-hole drainage of
their chronic SDH. They subsequently suffered symp-
tomatic fluid reaccumulations within their capacious
subdural space. The extent of their postoperative
recovery diminished with each recurrence and, as a
result, their neurological condition progressively
deteriorated. With the exception of Case 2, burr-hole evacuation of the initial hematomas (90 to 200 ml) failed to produce neurological improvement that was as much as one grade. In none of these cases did the surgeon suspect incomplete drainage of the hematoma at the time that the burr holes were drilled. The subsequent percutaneous taps of the subdural space with a Scott cannula drained as much as 60 ml of fluid. Before 4 and 8 days postoperatively, these four patients developed a recrudescence of their presenting signs and symptoms. The burr holes were reopened and 50 to 100 ml of fluid was evacuated. In two cases, a craniotomy was then performed because of evidence (which was subsequently confirmed) that a portion of the hematoma was loculated. In the other cases, a craniotomy was performed several days later after further neurological deterioration had occurred. Both free and loculated fluid was found in these cases also. In none of these four cases did the surgeon observe any degree of brain re-expansion prior to closure of the craniotomy. Nevertheless, all of these patients improved at least one neurological grade within 12 hours after craniotomy. However, each patient again deteriorated within the next 7 days. Progression of neurological dysfunction was generally rapid, with deterioration of one or two neurological grades occurring over periods of only 4 to 8 hours. The one patient (Case 2) whose craniotomy was re-explored after only 24 hours had a fresh SDH. The source of the bleeding could not be identified. The brain had not re-expanded following the previous craniotomy and membranectomy and remained 2 to 3 cm below the dura following evacuation of the acute SDH. The patient demonstrated signs of uncal herniation, and the surgeon therefore placed a high priority on preventing any further episodes of cerebral compression. The bone flap was thus removed. The scalp flap began to become deeply concave after 3 weeks. The other three patients in this clinical group deteriorated at the end of the 1st week following craniotomy. At craniectomy, the subdural space was distended with coagulated proteinaceous fluid. There was no evidence of recent hemorrhage. The brain did not re-expand following evacuation of the fluid in any of these cases.

The second group of patients who underwent craniectomy (Cases 3, 4, and 5) also had symptomatic recurrent chronic SDH's, which were ultimately treated by craniotomy and membranectomy. In addition, they were found to have cerebral swelling subjacent to their hematomas. The hematomas themselves were unremarkable and showed no gross signs of acute hemorrhage. In these cases, brain re-expansion began immediately after surgery, but the patient fluctuated between Grades 3 and 4. On the 1st postoperative day, her condition stabilized in Grade 4 and the oculomotor palsy recurred. At the time of re-exploration of the craniotomy, the dura was found to be very tense, but only a skim of fresh hematoma was found beneath it. The gyri were pale and swollen. After the bone flap was removed, the scalp remained tense for almost a week before gradually invaginating through the cranial defect.

**Results**

Postoperative assessments of neurological function were obtained in all 48 patients treated according to our standardized protocol (Table 1). In all cases, the most recently recorded neurological grade was used. These grades were determined at an average of 20 months after the definitive operation (range, 6 weeks to approximately 20 months). One of these patients was first noted to have papilledema in the recovery room. In both of these cases, the scalp flaps remained tense for several days before gradually softening and flattening, and ultimately becoming deeply concave. The other patient in this group (Case 3) deteriorated from Grade 2 to Grade 3 on the 3rd day after burr-hole drainage of her chronic SDH. She rapidly improved one grade following evacuation of 30 ml of nonclotting, serosanguinous fluid through a Scott cannula. Six hours later she again became Grade 3, but improved once more after 60 ml of fluid was evacuated through her reopened burr holes. She remained in stable condition for a week, but then became increasingly somnolent and hemiparetic over a period of 3 hours. Papilledema was noted for the first time. She developed an oculomotor nerve palsy ipsilateral to her hematoma as she was taken to the operating room. Craniotomy disclosed a multiloculated chronic SDH that contained a small amount of freshly clotted blood. The brain immediately began to re-expand, but the dura was closed without difficulty. The oculomotor palsy resolved immediately after surgery, but the patient fluctuated between Grades 3 and 4. On the 1st postoperative day, her condition stabilized in Grade 4 and the oculomotor palsy recurred. At the time of re-exploration of the craniotomy, the dura was found to be very tense, but only a skim of fresh hematoma was found beneath it. The gyri were pale and swollen. After the bone flap was removed, the scalp remained tense for almost a week before gradually invaginating through the cranial defect.

**TABLE 1**

<table>
<thead>
<tr>
<th>Preop Grade</th>
<th>Total Cases</th>
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<tbody>
<tr>
<td>Postop Grade</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>17</td>
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<td>7</td>
<td>3</td>
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<tr>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>38</td>
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</table>

*Preoperative grades are correlated with postoperative results. Preoperative grade refers to the grade before final operation in cases in which multiple surgical procedures were performed. Postoperative grade refers to status at the most recent follow-up examination.
to 5 years). No patient died of any cause within 30 days of his last operation or, to the best of our knowledge, died any time thereafter as a result of a recurrent chronic SDH.

The most recent follow-up neurological grade of each craniectomy patient is recorded in Table 2. In addition, these seven patients were assessed according to the outcome scale developed by Jennett and Bond. This scale of social function de-emphasizes the role of returning to work as an outcome determinant, and is thus particularly useful in evaluating our craniectomy patients, all of whom were approaching or beyond retirement age. Four patients (Cases 2, 3, 4, and 6) eventually attained the best outcome category by resuming their premorbid life styles and responsibilities. Two patients (Cases 1 and 7) were classified in the second outcome category, "disabled but independent." Only one patient (Case 5), who was classified as "severely disabled," had an unsatisfactory outcome. Although conscious and conversant, he remained demented and hemiplegic and consequently remained in the hospital until his death 6 weeks after craniectomy. Death was attributed to complications of congestive heart failure, which antedated his hospitalization. Autopsy demonstrated cortical and subcortical infarction subjacent to his previous hematoma.

No patient has suffered a further symptomatic reaccumulation of subdural fluid following craniectomy. We have not encountered either focal neurological deficits or epilepsy which have been reported to be late complications of large cranial defects.

Discussion

The seven patients who underwent craniectomy can be distinguished from the other 41 patients with surgically treated chronic SDH's on the basis of clinical parameters previously reported to be associated with a poor prognosis. These factors include: 1) poor preoperative neurological condition, 2) advanced age, and 3) need for multiple operations. Despite the fact that in each case all three of these adverse prognostic signs coincided, the majority of these patients eventually resumed all of their previous activities, and in only one case was the outcome clearly unsatisfactory.

Nevertheless, craniectomy is not a panacea for all patients who become critically ill as a result of a chronic SDH, since the indications for this operation are quite specific. Craniectomy is not an appropriate initial surgical procedure, even for patients who present in coma. Data reported by Svien and Gelety indicate that a substantial number of these patients can be cured by burr-hole drainage. One reason for performing a craniectomy is to obliterate the residual subdural space that persists due to failure of the chronically compressed brain to re-expand. Fox and McCullough have similarly advocated craniectomy in cases in which recurrent symptomatic chronic SDH's occur as a complication of shunting procedures for normal-pressure hydrocephalus. Pathological enlargement of the subdural space predisposes to hematoma reaccumulation, presumably because the pressure in this compartment becomes lower than that in adjacent capillaries, thus encouraging fluid transudation or actual hemorrhage. Removal of the cranial vault overlying the enlarged subdural space causes progressive obliteration of the latter as the scalp and dura invaginate through the cranial defect as a result of the gradient between the atmospheric and subdural pressures. The efficacy of craniectomy in this clinical setting is based on prevention of further reaccumulations of subdural fluid that might again cause

<table>
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<th>Clinical courses of seven craniectomy patients</th>
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<table>
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<tr>
<th>Clinical Data</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
<th>Case 6</th>
<th>Case 7</th>
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<tr>
<td>age (yrs)</td>
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<td>60</td>
<td>69</td>
<td>68</td>
<td>65</td>
<td>82</td>
<td>62</td>
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<td>M</td>
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<td>18</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>1</td>
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<tr>
<td>interval from burr holes to craniectomy (days)</td>
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<td>5</td>
<td>10</td>
<td>14</td>
<td>4</td>
<td>12</td>
<td>15</td>
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<td>no. of operations neurological grade before burr-hole drainage</td>
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<td>4</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>best grade between burr holes &amp; craniectomy</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>before craniectomy</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>at follow-up exam</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>interval from craniectomy to follow-up exam</td>
<td>2 yrs</td>
<td>5 yrs</td>
<td>5 yrs</td>
<td>4 mos</td>
<td>6 wks</td>
<td>4 mos</td>
<td>4 mos</td>
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*Interval from onset of symptoms if history of trauma was not obtained.

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cerebral compression. When failure of cerebral re-expansion is associated with marked neurological dysfunction, but not with a recurrent SDH, there is no reason to believe craniectomy will be efficacious. This clinical condition probably reflects the structural and functional consequences of progressive diminution of the vascular bed subjacent to the chronic SDH, and may be more common than fluid reaccumulation in patients who have persistent disability following craniotomy.

Symptomatic swelling of the white matter subjacent to a chronic SDH is undoubtedly rare, and was recognized in only 6% of our patients, all of whom were comatose at the time that the diagnosis was made. Browder and Rabiner obtained autopsy evidence of regional cerebral swelling in patients who died as a result of chronic SDH. Furthermore, regional swelling has been identified radiologically in some patients who failed to recover or continued to deteriorate following evacuation of a chronic SDH. Several investigators have reported that after evacuation of a chronic SDH, the intracranial pressure (ICP) may temporarily exceed the preoperative level by as much as 30%. The etiology of cerebral swelling following drainage of a chronic SDH remains unclear, although Tabaddor has suggested that the postoperative “rebound” of the ICP may be due to a temporary increase in cerebral blood flow or blood volume or may be attributable to vasogenic edema. In our Case 5, cerebral swelling was probably associated with acute, extensive cerebral infarction. In such cases, the cerebral swelling is virtually an epiphenomenon, and craniectomy can be expected to have little, if any, efficacy.

Our few patients with cerebral swelling were treated by a combination of decompressive craniectomy and glucocorticoid and respiratory therapy. We have not excluded the possible efficacy of a more aggressive pharmacological regimen, which might include osmotic and/or loop diuretics. Such regimens have been reported to encourage resorption of chronic SDH’se but have generally been abandoned in favor of surgical drainage in patients who are deteriorating or critically ill. The role of pharmacological alternatives to craniectomy therefore remains undefined, both for patients with cerebral swelling, and for those dying as a result of intractable subdural fluid reaccumulations.

Summary

Inclusion of craniectomy in a surgical protocol for treatment of patients with a chronic SDH may allow the surgeon to salvage some patients who might otherwise be expected to die on the basis of such unfavorable prognostic factors as marked neurological impairment, advanced age, and failure of antecedent surgical procedures. In such patients, craniectomy should be considered if neurological deterioration following craniotomy is associated with reaccumulation of fluid in a persistently enlarged subdural compartment. Craniectomy may also be beneficial in the rare cases in which neurological deterioration is associated with extensive swelling of white matter subjacent to the hematoma. This operation should not be performed routinely, since the majority of patients with chronic SDH’s can be cured by more conservative techniques, and not all patients whose chronic SDH’s become life-threatening will satisfy the above clinical criteria.

Addendum

Since our initial experience with the seven cases reported from our series in Plymouth, craniectomy has been successful in two additional patients with chronic SDH’s treated at the University of Virginia.

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References

Cranieotomy for subdural hematomas


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