Surgical Management of Primary Intracerebral Hemorrhage*

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For the treatment of primary intracerebral hemorrhage (hemorrhage without an underlying aneurysm or arteriovenous malformation) we have been pursuing a uniform surgical approach during the past 6 years.11 Our patients during this interval comprised a consecutive, unselected series which was representative of the problem within a given population. We used the method of immediate angiography and early direct surgical evacuation of the hematoma when demonstrated. We believed this would lead to the lowest possible mortality for the series as a whole, even though mortality in the operated patients might be high. From retrospective study of this series we hoped to derive an estimate of the over-all usefulness of surgical evacuation of the hematoma, the lowest mortality and morbidity that might be achieved with combined surgical and medical care, and the reasons for failure particularly among those patients receiving operative treatment.

Case Material

The Georgetown University Neurosurgical Service, with 60 beds incorporated in four affiliated hospitals, receives about 85 patients with spontaneous intracranial bleeding yearly, or approximately 500 cases over the last 6 years. Only those under direct supervision of the senior author were uniformly managed for purposes of this study. Most were at the University Hospital, which accommodates an average cross section of serious medical problems, has an active emergency service, and receives referred cases from nearby community hospitals.

Our sampling included 130 patients with spontaneous intracranial bleeding, 66 of whom showed no underlying vascular abnormality or neoplasm (Table 1). There were no primary cerebellar hemorrhages. Two brain stem hemorrhages, diagnosed on clinical grounds, were excluded, leaving 64 patients. Most of the early cases were admitted to the Medical or Neurology Service with consultation requested within the first 24 hours; later the cases were admitted directly to the Neurosurgical Service.

The male-to-female ratio was 1.5 to 1, and the age distribution is shown in Table 2. All except 2 patients had underlying hypertensive arteriosclerotic disease of some degree. In addition there were instances of diabetes mellitus, chronic emphysema, advanced polycystic renal disease, chronic congestive myocardial failure, hepatic cirrhosis with a bleeding dyscrasia; one patient was on anticoagulation therapy for myocardial ischemia. In many, the blood pres-

<table>
<thead>
<tr>
<th>TABLE 1</th>
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<tbody>
<tr>
<td><strong>Cases of spontaneous intracranial hemorrhage</strong></td>
</tr>
<tr>
<td>Etiology</td>
</tr>
<tr>
<td>Saccular aneurysms</td>
</tr>
<tr>
<td>A-V malformations</td>
</tr>
<tr>
<td>No vascular abnormality</td>
</tr>
<tr>
<td>Neoplasms</td>
</tr>
<tr>
<td><strong>Total</strong></td>
</tr>
</tbody>
</table>

* Hemorrhages occurring before 1 month prior to admission not included.

**TABLE 2**

**Age and distribution in cases of primary intracerebral hemorrhages**

<table>
<thead>
<tr>
<th>Age in Years</th>
<th>Number of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-29</td>
<td>2</td>
</tr>
<tr>
<td>30-39</td>
<td>14</td>
</tr>
<tr>
<td>40-49</td>
<td>5</td>
</tr>
<tr>
<td>50-59</td>
<td>21</td>
</tr>
<tr>
<td>60-69</td>
<td>13</td>
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<tr>
<td>70-79</td>
<td>8</td>
</tr>
<tr>
<td>90</td>
<td>1</td>
</tr>
</tbody>
</table>

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sure became extremely elevated following the ictus but returned to normal after removal of the clot.

**Clinical Management**

In nearly all cases, angiography was carried out within 24 hours after referral or, when admitted via the emergency service, prior to transfer to a hospital bed. Excessive elevation of blood pressure was controlled with intravenous and intramuscular Apresoline (hydralazine hydrochloride, Ciba) or Serpasil (reserpine, Ciba) or, in a few cases, an intravenous drip or Arfonad (trimethaphan camysylate, Roche). Tracheotomy or insertion of a cuffed intratracheal tube was performed when there was respiratory distress, aspiration pneumonitis, or certainty of prolonged coma. Early initiation of gastric tube feeding was used in preference to prolonged intravenous maintenance.

Many of the patients were in an intensive care unit fully equipped with electronic monitoring and experienced nursing personnel; all patients were under dual medical-surgical management.

Surgery was carried out within the first 24 hours in all except two patients. The technique used was craniotomy, a 2 to 3 cm cortical incision over a superficial portion of the clot, and removal of the hematoma by suction and saline irrigation. A penrose drain was left in the hematoma cavity for 24 hours. Lesions of the internal and lateral capsules were evacuated through small cortical incisions in the frontal, parietal, or temporal regions depending upon the direction of maximal clot extension. Preliminary twist-drill needle aspiration was used in two cases. All cases received general anesthesia with hyperventilation. Mannitol and urea were used postoperatively on occasion. Hypothermia was not used. Patients treated in the last 3 years received corticosteroids in large doses. Patients admitted to the Medical and Neurology Services usually had lumbar punctures. However, in severe cases this was deferred.

**Clinical and Angiographic Localization of the Hemorrhage**

With increasing experience we were able to localize the hemorrhage accurately from clinical findings and a detailed history. This depended chiefly on the rate of development of an abnormal state of consciousness and lateralizing sensory or motor symptoms. Hemorrhages originating in the capsular or thalamic regions usually showed both flaccid hemiplegia and alteration of consciousness. In lateral capsular hemorrhages the motor symptoms developed more slowly, followed by impairment of consciousness. Hemorrhages extending into the frontal, occipital, or temporal lobes were more apt to have intense headache, followed by alteration of consciousness and less severe lateralized motor signs. When the hemorrhage was large and a detailed history unavailable, upper brain stem signs obscured the picture, and localization became inaccurate.

Angiographically, clots extending into the frontal, temporal, or parietooccipital areas were accurately localized by appropriate surface artery displacement. In smaller capsular and paracapsular hemorrhages, displacement of the penetrating branches of the middle cerebral trunk and the anterior choroidal artery were the key findings. Medical displacement indicated an external capsular origin and lateral displacement origin in the basal ganglia or thalamus. Widening of the space between the Sylvian arteries and the anterior cerebral arteries on the anteroposterior view (so-called spreading of the U) was seen in both types.

In general, clots not precisely delineated angiographically were, on clinical grounds, small capsular lesions with minimal threat to survival. In only one case was pneumography employed in addition to angiography because the latter was of poor technical quality; a capsular clot was identified by shift of the third ventricle. In patients without major lateralizing signs or symptoms, three-vessel angiography was carried out in search for an aneurysm.

**Clinical Classification of Patients**

We classified our patients according to probability of survival as indicated by the neurological status during the first 1–2 hours after admission or referral. Three groups, defined on clinical grounds, were so derived (Table 3).

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**Group 1** (7 patients) included those whose survival was certain, precluding recurrence of bleeding. The objective lateralizing symp-
toms were minimal. The patients were alert or only slightly drowsy.

In Group 2 (33 patients) survival was uncertain. All had major laterализing and focal neurological deficits and their states of consciousness ranged from drowsy to deep stupor. Aside from alteration of consciousness none showed evidence of diencephalic or mesencephalic involvement by extension or compression.

In Group 3 (24 patients) there were clear-cut upper or lower brain stem signs consistent with extension of hemorrhage to these areas or compression from beginning uncal herniation. In this group death was clinically certain, and most were in extremis.

From the onset of the hemorrhage the clinical status was in a state of flux. Group assignment was based upon the status during the first hours under our care. This was the interval necessary to establish the diagnosis and initiate preliminary treatment consisting of an adequate airway and control of elevated blood pressure. Neither spinal fluid pressure, severity of pre-existing hypertension, nor age played a role in this classification.

Results

These 64 patients are considered as a surgical series because each was evaluated and managed with a view to surgical intervention. Thirty-seven underwent surgery for clot evacuation. Of the 64, 24 died, making a total management mortality of 37% (Table 4). None in Group 1 underwent surgery and all survived. In Group 2 surgery was deferred in 9 patients because, with one exception, we failed to demonstrate a significant mass lesion angiographically. Presumably these patients had small capsular or paracapsular clots.

All had bloody spinal fluid with increased pressure. The exception was a 90-year-old patient whose capsular clot was clearly defined but surgery deferred because of advanced senility. He became the only mortality of the 9 cases. In the 24 who underwent craniotomy there were 2 deaths, or an operative mortality of 8% (Table 5). Because death was not imminent at the time treatment started, these deaths were considered operative failures. In neither case, however, was death of direct cerebral origin. These two cases will be described in detail.

Case 1. A 38-year-old man with a history of severe hypertension was admitted to Georgetown University Hospital on January 19, 1966, 3 hours after abrupt onset of headache and right-sided weakness which had rapidly progressed to complete para-
Table 5

Mortality in surgical and non-surgical cases

<table>
<thead>
<tr>
<th>Group</th>
<th>Surgical Survived</th>
<th>Surgical Died</th>
<th>Non-surgical Survived</th>
<th>Non-surgical Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22 (8%)</td>
<td>2</td>
<td>0</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>2</td>
<td>2 (8%)</td>
<td>2</td>
<td>1 (12%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>3</td>
<td>10 (77%)</td>
<td>3</td>
<td>11 (100%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Total</td>
<td>25 (92%)</td>
<td>12 (32%)</td>
<td>15</td>
<td>12 (100%)</td>
</tr>
</tbody>
</table>

Sis and aphasia. Examination 1 hour after onset revealed a blood pressure of 220/120. The patient was alert, mute, did not follow verbal commands, and had a flaccid paralysis of the right arm and leg. A lumbar puncture revealed bloody cerebrospinal fluid with a pressure of 220 mm. He was given 30 mg of Apresoline intravenously. A left carotid arteriogram showed a mass lesion in the insula with extension into the frontal lobe.

Operation. The patient was immediately taken to the operating room. A large frontotemporal flap was turned. The dura was very tense. The clot was identified by a ventricular needle passed through the middle frontal gyrus. A 2-cm cortical incision was made and carried down through white matter to the clot, which was removed by suction. There was no hemorrhage or bleeding provoked by the removal. The brain became slack; a 1/4 inch penrose drain was placed in the cavity and the dura closed.

Postoperative course. There was no immediate change in the patient’s neurological status, but within 24 hours some reflex activity returned in the arm and leg, and on the second postoperative day he followed simple commands although his speech was unintelligible. Elevated blood pressure was controlled with Serpsal. After his fourth postoperative day he was somewhat less responsive. A lumbar puncture showed an initial pressure of 170 mm with xanthochromic spinal fluid. Thereafter he remained unchanged until the tenth day when his respirations suddenly became shallow and rapid, increasing to 40 per minute, and within a few minutes thereafter he was apneic, with cardiac arrest. An electrocardiogram revealed a complete heart block. Vigorous cardiac and respiratory resuscitation was initiated without avail, and the patient died. Permission for autopsy was not granted. The clinical impression was a pulmonary embolus.

Case 2. A 71-year-old man was admitted to Georgetown University Hospital on May 29, 1964. He had a large abdominal aortic aneurysm, for which surgery had been deferred because of severe generalized arteriosclerosis, and a history of multiple myocardial infarctions with mild cardiac decompensation for which he had been on digitalis. There was no history of hypertension. During the preceding 3 months he had frequent syncopal episodes.

Three hours before admission he noticed the abrupt onset of weakness of the left arm and leg and loss of vision. He was alert during examination in the emergency room. Blood pressure was 170/104. There was a left homonymous hemianopsia and a spastic left hemiparesis involving the arm and leg equally. The patient tended to ignore the left extremities. A lumbar puncture revealed an initial pressure of 180 mm of cerebrospinal fluid; the fluid was grossly bloody. During the first 12 hours following admission the patient’s state of consciousness declined, and he became unresponsive to painful stimulation.

Operation. A right carotid arteriogram 14 hours after admission showed a mass in the temporoparietal area on the right. A right temporoparietal craniotomy was performed immediately. The underlying dura was extremely tense. The clot was found through a 2 cm cortical incision in the posterior temporal lobe; the lesion extended into the inferior parietal area and posterior internal capsule and was estimated to be about 30 cc in amount. When hemostasis was complete a 1/4 inch penrose drain was placed in the cavity and the dura closed.

Postoperative course. Within 24 hours after surgery the patient moved the left leg and the left upper arm upon command. He had a series of left-sided focal seizures which were brought under control with phenobarbital and dilantin. On the second postoperative day he developed a low-grade temperature, tachycardia, and tachypnea. Chest x-rays demonstrated pneumonitis and severe emphysema. He remained in respiratory
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distress primarily from emphysema, and tracheotomy was performed. His cardiac status declined, and he died on his seventh postoperative day in congestive heart failure. An autopsy demonstrated reaccumulation of at least 1/2 of the clot in the lateral capsular region. There was severe atherosclerosis of the cerebral arteries.

In Group 3 the management mortality was 86%. Thirteen of the 24 patients underwent surgery. In the others it was deferred or impossible for the following reasons: six died of respiratory failure during the initial hours of evaluation and three of these prior to angiography; one died during angiography; in one, surgery was declined because of associated polycystic disease with uremia and a life expectancy of a few months. Each of the remaining three patients was moribund; family support for surgical intervention was withheld because of the patient’s advance age and associated debilitating disease.

In the 13 operated patients there were 10 deaths, an operative mortality of 77%. Because survival was not anticipated in this group, success in these three cases was almost certainly due to surgical intervention, which will be described in detail.

Case 3. A 20-year-old woman was admitted to the Neurosurgical Service on September 10, 1963. She was 2 months postpartum. On the night before admission she complained of a severe headache and was given codeine by her family doctor. Thereafter she had an uneventful night but in the morning was found unresponsive and was rushed to the hospital.

On examination she did not respond to voice. The left pupil was dilated and fixed to light. Blood pressure was 110/60. There was increased tone in the right arm and decerebrate posturing in response to painful stimulation. There was bloody cerebrospinal fluid and a pressure of 220 mm. Bilateral carotid arteriograms showed a mass lesion in the left frontal lobe.

Operation. A left frontal craniotomy was performed 6 hours after admission. The dura was extremely tense. The clot encountered in the midportion of the frontal lobe was removed through a 2-cm cortical incision over the anterior portion of the middle frontal gyrus. It extended deeply toward the internal capsule and was estimated to be 40 cc in volume. When hemostasis was completed, a 1/4-inch penrose drain was left in the cavity and the dura closed.

Postoperative course. The patient’s clinical status remained stable for 2 days and thereafter showed gradual, sustained improvement. When discharged 2 weeks after surgery she was walking, with approximately 50% recovery of the right spastic hemiparesis. There was a moderately severe expressive aphasia. Within 6 months, use of the right arm was close to normal. Speech recovered except for a very mild expressive aphasia. She has been followed for 3 years and carries on her normal responsibilities as a housewife. She has had approximately six seizures and requires dilantin, phenobarbital, and myololine for control.

Case 4. A 64-year-old woman was admitted to the emergency room 3 hours after abrupt onset of severe right-sided headaches and weakness of the left arm and leg. She was responsive to voice and had a severe left hemiplegia. A lumbar puncture revealed bloody cerebrospinal fluid with an initial pressure of 300 mm. During the first hour her condition deteriorated so that she was unresponsive to voice and the right pupil was maximally dilated and nonreactive to light. The left arm and leg assumed decerebrate posturing with painful stimulation, and there were bilateral Babinski signs.

Operation. A right carotid arteriogram 1 hour after admission showed a large mass in the right temporal lobe. Upon arrival in the operating room 2 hours after admission the patient was bilaterally decerebrate. Prior to induction of anesthesia a twist-drill hole was made over the right temporal area and approximately 30 to 40 cc of blood evacuated via a No. 16 ventricular needle. A large hematoma occupying the temporal lobe and extending into the insula was removed through a right temporal craniectomy. A tracheotomy was performed at the end of the procedure.

Postoperative course. After surgery the patient became apneic and required periodic respirator support. Her clinical status was unchanged for 5 days but thereafter grad-
ually improved. Three weeks later she could follow simple commands and use her right arm and leg normally. Her recovery was complicated by urinary tract infections and aspiration pneumonitis. The tracheotomy was discontinued after 1 month and she was discharged 4½ months after surgery. At that time she was normally responsive, feeding herself, and able to sit in a chair. However, she could not walk. There was some recovery of movements of the left ankle and feeble flexion movements of the left fingers. She was placed in a nursing home where she died of pneumonia 1 month later.

Case 5. A 65-year-old woman was admitted on November 11, 1966. She had a history of mild hypertension. Nine days prior to admission she was found lying on a sofa unable to move her left side, but she responded to voice and appeared alert. She was immediately admitted to another hospital where a lumbar puncture revealed bloody spinal fluid; the pressure was not recorded. Her neurological status remained unchanged for approximately 7 days but because of aspiration pneumonitis a tracheotomy was performed. After 7 days she became unresponsive and the right pupil dilated and fixed to light. She was transferred to this hospital.

At the time of admission her temperature was 102°, blood pressure 110/70, and respirations 30. There was no response to voice, and she had left flaccid hemiplegia with decerebrate responses to pain. The right pupil was dilated and fixed and the left small and unreactive. Forty minutes after admission a right carotid arteriogram showed a mass lesion in the right parietal lobe.

Operation. Two hours after admission, a right parietal craniotomy was performed, and a clot, estimated at 40 cc, was totally evacuated. The cavity extended to the midportions of the hemisphere and capsular area.

Postoperative course. During the first postoperative day, the patient became responsive to voice but was decerebrate on the left side. By the third postoperative day the left arm and leg became flaccid, and thereafter her neurological status stabilized. The remainder of her hospital course was complicated by pneumonitis, bilateral tracheo-

bronchitis, and hyponatremia. She had an episode of acute respiratory and cardiac arrest, secondary to aspiration, which responded to vigorous resuscitation. At the time of discharge, 48 days after surgery, she was normally alert, able to feed herself, and had recovered fair movement of the left hand. There was a partial left homonymous hemianopsia. She was transferred to a nursing facility for further rehabilitation.

Relationship of Hematoma Site to Survival

Anatomical localization of the hematomas from angiographic or pathological findings was possible in 50 of the 64 patients. Generally the sites were either paracapsular (24 cases), or lobar (26 cases) (Table 6). In most of the lobar cases, excluding the temporal lobe, the clot seemed to originate from the external capsule. In the massive hemorrhages causing rapid death (Group 3) the clot not only occupied a lobe but extended extensively through the central structures and ventricles.

Localization of the clot bore a striking relationship to the mortality and the presenting clinical status (Tables 7 and 8). Twenty-two of the 25 patients in Group 3 had paracapsular or capsular clots, whereas only 2 of the 23 in Group 2 were similarly located. The total mortality for Groups 2 and 3, irrespective of surgery, was 11% in the lobar cases and 92% in the capsular, paracapsular series. In the operated cases the mortality was 4% for the lobar clots and 79% for the capsular clots. All three survivors in Group 3 had lobar clots.

The large proportion of lobar clots in this series is at variance with other reported autopsy series in which 10% of the clots are lobar while 60% are capsular, paracapsular, or thalamic. This difference is explained by the probability of small capsular clots in the non-operated cases of Group 2, and our

<table>
<thead>
<tr>
<th>TABLE 6</th>
<th>Hematoma site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site</td>
<td>Group 2</td>
</tr>
<tr>
<td>Lobar</td>
<td>20</td>
</tr>
<tr>
<td>Capsular</td>
<td>5</td>
</tr>
</tbody>
</table>
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Analysis of Operative and Non-operative Deaths

In the 64 patients there were 24 deaths; autopsies were obtained in all but one case.

Group 2. The two operative deaths in Group 2 have been described. The single non-operative death in this group was a 90-year-old patient with severe senile dementia. The clot was in the external capsule and measured about 25 cc. There was no evidence of uncal herniation, and the patient died of pneumonia.

Group 3. Autopsies were obtained after the 10 operative deaths in this group. Three of these patients were apneic, or nearly so, at the time of induction of anesthesia and, following surgery, required respirator support. Each died within a few days without improving. In all three there was marked reaccumulation of the clot with extension into the third ventricle and hypothalamus.

One patient died 3 days postoperatively with a myocardial infarction. She had received anticoagulation therapy. The autopsy showed a large infarction surrounding the previous site of hemorrhage. Most likely her original pathology was hemorrhagic infarction. One patient died in cardiac arrest immediately after surgery, and autopsy demonstrated severe pulmonary edema and persisting clot in the thalamus and third ventricle.

The remaining four patients showed some clinical improvement following surgery but within a few days gradually deteriorated and expired with upper brain stem involvement. In all of these there was marked reaccumulation of the clot and extension to the ventricular system.

TABLE 7

<table>
<thead>
<tr>
<th>Site</th>
<th>Total Cases</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lobar</td>
<td>24</td>
<td>3 (11%)</td>
</tr>
<tr>
<td>Capsular</td>
<td>14</td>
<td>22 (92%)</td>
</tr>
</tbody>
</table>

TABLE 8

Hematoma site related to surgical mortality
(Groups 2 and 3)

<table>
<thead>
<tr>
<th>Site</th>
<th>Total Cases</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lobar</td>
<td>24</td>
<td>1 (4%)</td>
</tr>
<tr>
<td>Capsular</td>
<td>14</td>
<td>11 (79%)</td>
</tr>
</tbody>
</table>

In the 11 patients in Group 3 not undergoing surgery, autopsies demonstrated parasellar clots with ventricular extension in nine and frontal lobe and ventricular extension in two.

Summary. Seven of the 12 autopsies in operated patients showed significant reaccumulation of the hematomas which conceivably contributed to the death of four. Four died of medical complications at a time when their neurological status was improving.

Partial reaccumulation of the hematoma in patients whose operation had been successful was suggested by a prolonged convalescence in some and repeatedly by high lumbar puncture pressures in others. In two younger normotensive patients with purely lobar hemorrhages, angiography repeated 2 weeks after surgery suggested persistence of the hematoma. The evidence suggests, therefore, that reaccumulation of the clot was a major technical problem and contributed to operative mortality.

Quality of Survival

The wide diversity in age, education, economic status, and occupation among the patients precluded a meaningful evaluation of survival when restricted to specific criteria alone. For example, slight impairment of skilled movements of the fingers would be incapacitating for a watchmaker but of minor importance for a politician. In contrast, a trivial motor aphasia would be of little consequence in the former occupation but disaster for the latter. Therefore, we categorized survival as either satisfactory or unsatisfactory according to the following definitions. A satisfactory survival was defined as ability of that individual to carry out normal daily activities in an independent and responsible way and resume some of his
customary pleasure pursuits. Unsatisfactory survival included patients who remained absolutely dependent and restricted to a chair or bed existence. Neither of these categories was occupation-dependent, but follow-up relating to this criterion alone showed that among 18 survivors who, prior to the ictus, were actively employed and the sole support of their families or themselves, 10 returned to their former jobs, which ranged from clerical to managerial. Eight were unable to return to any form of employment.

The follow-up periods ranged from 3 months to 6 years. In Group 1 there were six satisfactory recoveries; two of these resumed former occupations. In one patient there was a complication of angiography resulting in a right hemiplegia which cleared only in part. Later he became completely self-reliant and can be considered a satisfactory result. One patient is still hospitalized for a renal tumor.

In Group 2, of the eight survivors who were operated upon, five were satisfactory and three unsatisfactory. Among the 22 surviving operative patients treated surgically, 15 made satisfactory recoveries and seven unsatisfactory. There were three late deaths in the seven unsatisfactory cases. These were due respectively to hepatic failure 3 months later, cerebral infarction 4 months later, and pneumonia in a bedridden elderly patient. These late deaths were not a direct consequence of the hemorrhage, which, however, had initiated the series of hospitalizations that ultimately ended in death.

The three survivors in Group 3 have been described in detail. Case 3 was a satisfactory survival. In Case 4 the patient died from pneumonia in a nursing home 1 month following discharge. In Case 5 the patient is still undergoing rehabilitation but is making a good recovery 4 months after surgery and this may be considered a satisfactory result.

In summary, 45% of the total 64 patients and 73% of the 40 survivors made a satisfactory recovery.

Discussion

During the past decade there have been scattered reports of the results of surgery in primary intracerebral hemorrhage.\(^1\)\(^2\)\(^3\)\(^4\)\(^5\)\(^6\)\(^7\)\(^8\)\(^9\)\(^10\)\(^11\)\(^12\) These have emphasized the presence of pre-existing hypertension, the timing of surgery, the localization of the clot, and the presenting clinical status as the principal factors in determining surgical mortality. Most reports were retrospective studies with undisclosed factors in the selection of cases; a variety of surgical techniques were employed in each.

Because, as they stated, "benefits of surgery for these lesions were not established," McKissock, et al.,\(^12\) undertook a controlled random trial.

They reported a 65% mortality for the surgical group compared to 51% for the non-surgical group. We had a 32% mortality for the surgical group and 37% for the entire series. Both were significantly lower than those of McKissock, et al.\(^12\) Judged by levels of consciousness and from the surgical standpoint, the valid comparison is between our Group 2 operated cases with an 8% mortality and their entire operated series with a mortality of 65%. It is not possible to analyze this wide discrepancy accurately because the report by McKissock, et al., did not include autopsy findings on their operative deaths. However, some of their cases with capsular or thalamic clots were apparently small, and other succumbed from reaccumulation of the clot as evidenced by their failure to achieve operative benefit in predominantly lobar clots.

If a clear-cut, arterial phase angiographic demonstration of the clot is required as a criterion for operation, our experience indicates that over-all mortality may be reduced to approximately one-third. Regardless of how rapidly the patient is brought to medical care, further reduction is not likely because of the incidence of large medially-arising hemorrhages which promptly dissect into the thalamus and ventricles. This is in line with ideas first advanced by Fazio\(^6\) and is comparable to present day mortality rates for the management of bleeding saccular aneurysms.

Failure to achieve the goal of two-thirds survival suggests serious associated medical diseases, inherent in an older age group with hypertensive arteriosclerotic disease, or technical failures, mostly reaccumulation of the clot. The frequency of the latter suggests there is a potential for recurrent bleeding from the original source, as with aneurysms. For the future we propose routine reoperation when evidence of recurrence appears
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during the first few postoperative days.

An estimate of the applicability of surgical evacuation can be made retrospectively. If we combined the patients with clots too small for certain angiographic definition and those with overwhelming hemorrhages leading to death during the interval for transportation to the hospital and preliminary evaluation, there would be 27, or 42% of the series. A few of those remaining may be excluded for reasons of advanced senility or complicating disease, leaving approximately 50% as surgical candidates. Within the broad category of primary spontaneous intracerebral hemorrhage, there are secondary groups such as hemorrhage from minute congenital arteriovenous malformations, hemorrhage secondary to minor trauma, not historically established, and hemorrhage into ischemic infarcts. In our series there were two young normotensive patients with lobar hemorrhages, two with evidence of infarction adjacent to the hematoma, and one with a possibility of prior trauma. Inclusion of these does not significantly alter the results as they relate to the most common etiology, hypertensive arteriosclerotic disease.

Summary

In a series of 130 patients with spontaneous intracranial bleeding, there were 64 whose hemorrhage was of hemispheric origin without an underlying aneurysm, arteriovenous malformation, or neoplasm. All were evaluated by early angiography. With the exclusion of patients in whom a clot could not be demonstrated and those who succumbed during the emergency evaluation, 37 patients had immediate hematoma evacuation. The operative mortality was 32% for the entire operated group, but only 8% for the patients not moribund upon arrival. The operative mortality in the lobar cases was 4%, contrasted with 89% in the capsular cases. The most important surgical complication was reaccumulation of the clot. We suggest that only 50% of such patients are possible candidates for surgical evacuation of the hematoma. One can expect that about 66% will survive and that among these about 75% will have a satisfactory result in terms of comfort and usefulness.

References