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TRAUMATIC ACUTE SUBDURAL HEMATOMA

Major Mortality Reduction in Comatose Patients Treated within Four Hours

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Abstract To discover which factors contributed to recovery after surgical intracranial decompression, we reviewed the records of 82 consecutive comatose patients with traumatic acute subdural hematoma (ASDH) who were treated in a single center under a uniform protocol. The delay from injury to operation was the factor of greatest therapeutic importance. Patients who underwent surgery within the first four hours had a 30 per cent mortality rate, as compared with 90 per cent in those who had surgery after four hours ($P<0.0001$). Other important prognostic variables included results of the initial neurologic examination, sex, multimodality-evoked potentials, and postoperative intracranial pressure (ICP). If all patients with traumatic ASDH were taken directly to hospitals equipped to diagnose and remove the hematoma within four hours of injury, mortality rates could be reduced considerably. (N Engl J Med. 1981; 304:1511-8.)

ACCIDENTS are the leading cause of death in the United States in persons from the ages of one to 45.[1] Half the 105,000 accidental deaths per year are due to motor-vehicle accidents, and the majority of these patients have severe head injuries.

The annual number of deaths due to head injury is unknown[2]; however, traumatic intracranial hematomas, especially acute subdural hematoma (ASDH), carry the highest mortality rate. The mortality rate reported in comatose patients with traumatic ASDH has been appallingly high: 60 to 90 per cent.[3-9] The ASDH is usually accompanied by associated primary brain damage, and the high mortality has previously been ascribed to the underlying brain damage.[4,10] This belief has fostered a fatalistic attitude among many physicians and has frustrated modern neurosurgery in its attempts to deal effectively with this problem.

A few studies[5,10-13] have suggested that prompt surgical evacuation of the traumatic ASDH might reduce this high mortality rate, as has been the experience with epidural hematomas.[14] To test the hypothesis that a reduction in the time interval between injury and surgical decompression could lower mortality in ASDH, we undertook a review of 82 consecutive comatose patients with traumatic ASDH. If this hypothesis is true, a considerable diminution in the mortality of ASDH could substantially decrease the high rate of accidental death in this country.

METHODS

Background for Patient Selection

This retrospective study was derived from data on 366 consecutive patients with severe non-missile head injuries who were admitted to the Division of Neurological Surgery, Medical College of Virginia, from December 1972 through February 1980. All patients younger than two years were excluded. After resuscitation, all patients included in this study were unable to speak intelligible words, even after noxious stimuli, and were unresponsive to verbal commands. They had negative drug and toxic-substance screens but were able to breathe spontaneously. During the eight-year period, 82 comatose patients with traumatic ASDH (with or without associated intracerebral hematomas) were admitted; these patients represent 22 per cent of the total series. All these patients had a large midline brain shift (greater than 5 mm), and all were treated surgically by craniotomy. The subdural clot was the major contributing factor causing the brain shift in each patient. The interval of time from the injury to the surgical decompression was obtained and recorded in 76 of the 82 patients. In six patients, the onset of injury could not be determined.

Treatment Protocol in the Patient with ASDH

After admission to the emergency room and resuscitation, general and neurologic evaluations were undertaken, and x-ray films of the skull and lateral x-ray films of the cervical spine were obtained. Unless the patient was in respiratory distress, endotracheal intubation awaited neurologic evaluation and interpretation of cervical-spinal x-ray films.

The patients were intubated and artificially ventilated after administration of pancuronium, 4 mg. Ventilation was adjusted to obtain a partial pressure of arterial carbon dioxide of 25 to 30 mm Hg and a partial pressure of arterial oxygen above 100 mm Hg if possible. All patients were initially given 20 mg of intravenous dexamethasone, 500 mg of intravenous phenytoin over 30 minutes, and 60 mg of intramuscular phenobarbital.

Computed axial tomography was used as the diagnostic method of choice. If computerized scanning was not available or if the patient was too unstable to be moved to the x-ray facility (i.e., if there was persistent hypotension below 95 mm Hg systolic), air ventriculography was performed in the emergency room. On recognition of a lesion requiring surgical decompression, 100 g of mannitol was rapidly infused intravenously in an attempt to decompress the brain medically during preparations for surgery and to compensate for the interval until actual evacuation of the clot. Mannitol was given because patients with ASDH have very high intracranial pressure (ICP) on admission to the emergency room.[15]

Rapid temporal craniectomy with partial evacuation of the clot immediately preceded temporofrontoparietal craniotomy in all cases, as previously described.[11] When appropriate, contused and necrotic brain tissue was removed from the anterior frontal and temporal regions.

After craniotomy, all patients had either a ventricular catheter or a subarachnoid screw inserted on the side opposite the craniotomy for postoperative monitoring of ICP. Persistent elevation of mean ICP over 20 mm Hg was treated with hyperventilation, drainage of cerebrospinal fluid, mannitol, or a combination of these methods. In some recent cases, barbiturates were used. A study of multimodality-evoked potential was recorded in 40 patients. Particular attention was directed toward anticipation of medical and surgical complications of head injury in the environment of an intensive-care

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unit. When patients were consistently obeying verbal commands and had resolution of all medical problems, they were transferred for rehabilitation that consisted of daily physical, speech, and occupational therapy. All surviving patients have been followed for one year with serial complete neurologic examinations, repeat computerized scans, and studies of evoked potentials.

Multimodality-Evoked Potentials

The neurologic examination can be misleading in the early postoperative period. Patients are recovering from anesthesia, or they may be iatrogenically paralyzed for control of ventilation. Therefore, in the last 40 patients, we relied on electrophysiologic data to assess brain function during the early postoperative period.

A complete study of multimodality-evoked potentials consisted of observation of visual, auditory, and somatosensory evoked cortical potentials and auditory and somatosensory evoked brain-stem potentials.[16,17] Electroretinograms, eighth-nerve action potentials, and peripheral-nerve action potentials were recorded when necessary to verify the integrity of the peripheral receptors of each of the sensory systems mentioned above. The technical aspects of obtaining and analyzing the evoked-potential data in comatose patients with head injuries have been described elsewhere.[16] Although evoked-potential studies were performed on Days 1, 4, 14, 90, 180, and 365 after injury, only data from the earliest study were used in this report. All studies were graded prospectively, and the investigator had no knowledge of the patient's condition or ultimate outcome. The data were entered into a computerized data bank after completion of each study.

Electrophysiologic evidence of brain-stem dysfunction was considered present when both the auditory and somatosensory evoked brain-stem potentials were severely abnormal or absent.[17] Hemispheric dysfunction was considered present electrically when the visual, somatosensory, or auditory cortical responses were severely abnormal or absent bilaterally. If both the brain-stem and hemispheric evoked potentials were severely abnormal or absent, the patient's electrophysiologic dysfunction was described as disseminated.[17]

Statistical Analysis

Comparison of various proportions summarized in Tables 1, 2, and 3 were made by the chi-square test. The time delay from injury to operation is expressed as the mean ±S.E.M. The mean time intervals were compared by the Aspin-Welch t-test after normalizing logarithmic transformation was applied for the time interval. A significant difference was indicated by a P value below 0.05. A more detailed analysis was performed with a multivariate discrete technique based on both linear and logistic models.

Results

Of 366 comatose patients with severe closed head injury, 82 patients had ASDH, 61 had either an epidural or intracerebral hematoma, and 213 had diffuse nonsurgical brain injury.

Of the 82 patients with ASDH, 62 were men. The mortality rate was 57 per cent, and 34 per cent had a functional recovery, which was defined as either a good recovery or moderate disability on the Glasgow Outcome Scale.[18] Only seven patients (9 per cent) were either severely disabled or vegetative.

Age

Patients with an ASDH were significantly older than patients with other head injuries (P<0.01). On the other hand, age was not different between survivors and nonsurvivors of ASDH (Table 2) or between male and female patients (Table 3).

Table 1. Variables Influencing Outcome in Acute Subdural Hematoma (ASDH) as Compared with All Other Severe Head Injuries.

Variable	Severe Head Injuries, Excluding ASDH	ASDH	Significance of Difference *
No. of patients	284	82	—
Age (yr)	26	41	P<0.01
Sex (%)			
Male	76	75	NS
Female	24	25	NS
Mortality (%)	29	57	P<0.0001
Severe morbidity (severe disability/vegetative) (%)	9	9	NS
Functional recovery (good recovery/moderate disability) (%)	62	34	P<0.0001
Mode of injury (vehicular accident) (%)	79	55	P<0.0001
Postoperative intracranial pressure (%)			
<20 mm Hg	59	53	NS
Uncontrollable	15	24	NS
Initial neurologic examination (%)			
Bilaterally absent pupillary light reflexes	29	45	P<0.01
Absent or impaired oculomotor function	33	60	P<0.0001
Decerebrate or flaccid	32	47	P<0.02
Time from injury to surgical decompression			
No. of patients	47 †	76 ‡	—
Mean time lapse ±S.E.M. (min)	326±34.1	325±27.3	NS

*NS denotes not significant.

†Forty-seven of the 61 craniotomies performed for intracerebral hemorrhage contusions or epidural hematoma. In 14 patients, the period between injury and surgical decompression could not be determined.

‡Seventy-six of the 82 patients with ASDH. In six patients, the onset of injury could not be determined.

Sex

Seventy-five per cent of all patients with head injuries and of all patients with ASDH were male. Of the patients with an ASDH, men had a 65 per cent mortality rate. There was a significantly lower mortality rate in women: 35 per cent (P<0.05). Eighty-five per cent of the 47 deaths due to ASDH were in men. The incidences of raised ICP, neurologic abnormalities, and associated cerebral contusions and the mode of injury did not differ significantly between the sexes. On average, women underwent surgery two hours earlier than men (P<0.02) (Table 3).

Mode of Injury

Fifty-five per cent of the patients with ASDH had vehicle-related accidents (automobile, motorcycle, or pedestrian), as compared with 79 per cent of the patients with other head injuries (P<0.0001). The frequency of vehicular accidents was not significantly different between any of the subgroups of patients with ASDH (Tables 2 and 3).

Initial Neurologic Examination

None of the patients with an ASDH were speaking coherent words or obeying verbal commands on the initial neurosurgical evaluation. The motor, pupillary, and oculomotor responses of all patients are summarized in Table 1. Bilaterally absent pupillary light reflexes were demonstrated in 45 per cent of the patients with ASDH, as compared with 29 per cent of the patients with all other types of head injury ($P<0.01$). A greater portion of patients with an ASDH (60 per cent) had impaired or absent oculocephalic or oculovestibular reflexes, in contrast to only 33 per cent of the patients with other head injuries ($P<0.001$). Decerebrate posturing or flaccidity was present in 47 per cent of patients with an ASDH, as compared with 32 per cent of the other comatose patients ($P<0.02$). As shown in Table 2, neurologic dysfunction significantly influenced the outcome in patients with an ASDH ($P<0.05$). No difference in neurologic dysfunction was noted between male and female patients with ASDH.

ICP

Postoperative ICP was monitored in all patients for at least three days. Uncontrollable ICP (defined as pressure greater than 60 mm Hg, sustained, and unaltered by ventricular cerebrospinal-fluid drainage, mannitol, or hyperventilation) was present in 24 per cent of the patients with ASDH and 15 per cent of the patients with other head injuries. The peak ICP was less than 20 mm Hg in 53 per cent of the patients with ASDH; this rate was similar to that of 59 per cent in patients with all other head injuries (Table 1). ICP was not significantly different between male and female patients. Outcome in patients with an ASDH was substantially influenced by ICP. In 79 per cent of the patients who had a functional recovery, the postoperative ICP did not exceed 20 mm Hg ($P<0.001$). Only 30 per cent of the patients who died had ICP below 20 mm Hg. Almost half the patients who died after evacuation of an ASDH had uncontrollable ICP.

Table 2. Comparison of Variables Influencing Outcome in Acute Subdural Hematoma.

Variable	Death	Functional Recovery	Significance of Difference *
No. of patients	47	28	—
Age (yr)	41	41	NS
Sex (%)			
Male	85	54	P<0.01
Female	15	46	P<0.01
Mode of injury (vehicular accident) (%)	62	43	NS
Postoperative intracranial pressure (%)			
<20 mm Hg	30	79	P<0.001
Uncontrollable	43	0	P<0.0001
Cerebral contusion or hematoma (%)			
Present	67	53	NS
Absent	33	47	NS
Multimodality-evoked potentials (no. of patients)			
Normal	4	19	P<0.001
Abnormal	15	0	
Initial neurologic examination (%)			
Bilaterally absent pupillary light reflexes	57	25	P<0.02
Absent or impaired oculomotor function	70	43	P<0.05
Decerebrate or flaccid	56	29	P<0.05
Time from injury to surgical decompression			
No. of patients	44	26	—
Mean time lapse ±S.E.M. (min)	390±38.5	170±18.3	P<0.0001

*NS denotes not significant.

Table 3. Comparison of Variables Influencing Outcome among Men and Women with Acute Subdural Hematoma.

Variable	Women	Men	Significance of Difference *
No. of patients	20	62	—
Age (yr)	42	39	NS
Mortality (%)	35	65	P<0.05
Functional recovery (%)	65	24	P<0.002
Mode of injury (vehicular accident) (%)	60	53	NS
Postoperative intracranial pressure (%)			
<20 mm Hg	50	54	NS
Uncontrollable	15	27	NS
Cerebral contusion or hematoma (%)			
Present	66	64	NS
Absent	34	36	NS
Initial neurologic examination (%)			
Bilaterally absent pupillary light reflexes	40	47	NS
Absent or impaired oculomotor function	45	65	NS
Decerebrate or flaccid	40	50	NS
Time from injury to surgical decompression			
No. of patients	18	58	—
Mean time lapse ±S.E.M. (min)	227±35.3	347±32.3	P<0.02

*NS denotes not significant.

Type of Intracranial Mass Lesions

ASDH accounted for 82 of 143 mass lesions in this series (57 per cent). Fifty-three of the 82 patients with an ASDH (65 per cent) had an associated intracerebral contusion or hematoma, which was also evacuated at the time of craniotomy. The presence or absence of an intracerebral hematoma or contusion did not significantly alter the outcome (Table 2), and the incidences of cerebral contusions were almost identical among our female and male patients.

Multimodality-Evoked Potentials

Multimodality-evoked potentials were recorded in 40 of the 82 patients with ASDH (49 per cent). Eighty per cent were men, and 20 per cent were women. The

mortality in the 40 patients with ASDH in whom we conducted evoked-potential studies was 48 per cent (Table 2). Men had an 89 per cent mortality rate, as compared with only 25 per cent among women. Twenty-five of the 40 patients (63 per cent) had normal or only mildly focally abnormal evoked potentials; 19 of these patients had a functional recovery, two were severely disabled or vegetative, and four died. The four patients who died and had normal evoked potentials died of such delayed secondary systemic insults as delayed splenic rupture, myocardial infarction, or septicemia. The remaining 15 patients (38 per cent) had severe abnormalities in multimodality-evoked potentials of both the hemispheres and the brain stem — that is, disseminated brain dysfunction as detected by the electrical studies; all 15 died.

Time Interval to Surgical Decompression

Because of the time delay associated with cerebral angiography,[4,11,13,14] we relied on emergency (twist-drill) air ventriculography from 1972 to mid-1975 for rapid determination of ventricular compression, shift, and ICP, using this method as the primary diagnostic test to determine the need for surgical intervention. Since 1975, virtually all the head injuries in our hospital have been evaluated by emergency computerized scan. The promptness of surgical decompression has been measured from the time of injury to the time of actual surgical decompression. The mean delay between injury and surgical intervention was 325 minutes in 76 of the 82 patients with ASDH and 326 minutes in our last 47 consecutive craniotomies for surgical trauma other than ASDH. Outcome was significantly improved by rapid surgical decompression in all patients with an ASDH ($P<0.0001$) and in the women as compared with the men ($P<0.02$). The average patient with an ASDH who had a functional survival underwent operation three hours sooner than the average nonsurvivor. Women had better recovery than men and underwent surgery an average of two hours earlier.

Multivariate Analysis of Data

A discrete multivariate analysis showed that sex, ICP, results of the initial neurologic examination, and time to surgery were all significantly related to outcome. The significance levels determined by this analysis were as follows: $P<0.004$ for time, $P<0.006$ for sex, $P<0.03$ for results of the neurologic examination, and $P<0.04$ for ICP. We did not consider multimodality-evoked potentials in the multivariate analysis because we had only 40 complete studies from the 82 patients. However, no interaction effect among the variables tested was significant.

In our study, the degree of neurologic deficit in the patients was not related to the time delay, as might be expected. This point is supported by two key factors. First of all, our protocol was designed so that we would operate on hematomas as rapidly as possible in all comatose patients, regardless of the other neurologic findings. Secondly, the severity of a neurologic deficit is not necessarily related to the time lapse in the early period after injury but is certainly dependent on the severity of the injury. Twenty of our severely injured patients with a combination of decerebration, impaired or absent oculocephalic or oculovestibular reflexes, and bilaterally absent pupillary light reflexes underwent surgical decompression, on the average, at about the same time as the others, if not sooner. Apparently, recognizing that a neurosurgical emergency existed was easier for the emergency-room or referring physician in a decerebrate patient than in a combative, flailing, alcoholic patient who fell.

Discussion

Several investigators[11,19-24] have stated that younger patients have better quality of survival, tolerate longer periods of coma or decerebration, and have fewer life-threatening medical or surgical complications of head injury (i.e., gastric ulcer or pneumonia). Richards and Hoff[8] described 100 patients with ASDH, with an average age of 47 years. These authors contrasted the average age of 36 years in survivors with that of 51 years in nonsurvivors. Gutterman and Shenkin[5] described 14 patients with decerebration and ASDH, with an average age of 48.6 years. The average age of survivors was 44 years, and that of nonsurvivors was 51. Our 82 patients with ASDH were 15 years older, on the average, than the 284 patients with other head injuries ($P<0.01$). This factor may have influenced the mortality in our patients with ASDH as compared with that of our patients with other head injuries. However, within our series of patients with an ASDH, outcome was not influenced by age.

Few studies of ASDH have mentioned the influence of gender on outcome. Most large series consist of 75 to 80 per cent men. Jamieson and Yelland[24] reported on 553 patients with subdural hematoma, 80 per cent of whom were men, and noted that complicated subdural hematomas (hematomas with cerebral contusions) were more prevalent in men by a ratio of 4:1, as compared with simple hematomas, in which the sex ratio was only 3:1. From these data, Jamieson postulated that women with an ASDH were generally less severely injured than men. Braakman and his co-workers[19] demonstrated in 1980 that women had predictably better outcomes after severe head injury. Among our 82 patients 25 per cent were women, but women made up 46 per cent of the patients who had a functional recovery. Men were in worse neurologic condition on initial evaluation than women, although this difference was not statistically significant (Table 3). Men and women were also statistically comparable with regard to ICP, mode of injury, and age. The significant variable with respect to the more favorable outcome in women was not that

women were less severely injured but that they were treated on the average about two hours sooner than men in our series ($P<0.02$).

Jamieson,[24] Cooper,[4] and Richards[8] and their colleagues found that the injuries in more than 50 per cent of patients with ASDH were precipitated by falls or assaults. Vehicular accidents were ranked second in frequency but first in mortality. In our series, vehicular accidents occurred in 55 per cent of our patients with ASDH and ranked first in mortality.

In most reports, intracranial hypertension has been defined as mean pressure in excess of 15 to 20 mm Hg.[12,25-27] McKissock reported that five of six patients with ASDH had lumbar pressures over 15 mm Hg.[27a] Shigemori et al.[12] found in 15 patients that five patients with ICP above 70 mm Hg 24 to 48 hours after the operation had 100 per cent mortality, and that only two of seven patients with ICP between 35 and 70 mm Hg survived. Three of their patients with ICP below 35 mm Hg survived to have a functional recovery. Sustained, uncontrollable intracranial hypertension over 60 mm Hg occurred in 15 per cent of our patients with head injuries, 24 per cent of all patients with an ASDH, and 43 per cent of those who died after an ASDH. The ICP of patients who had functional recovery from an ASDH was less than 20 mm Hg in 79 per cent of the cases. Only 30 per cent of the nonsurvivors had a peak ICP of less than 20 mm Hg. Of all our patients who were continuously monitored for at least 72 hours, 53 per cent of those with ASDH had a peak ICP of less than 20 mm Hg, which was similar to that of our patients with other head injuries. Postoperative elevation of ICP was associated with a poor outcome in the series of Shigemori et al. and in ours.

Evoked-potential findings in this series of patients with head injury and ASDH confirm the findings in other studies using multimodality-evoked potentials for prognosis in comatose patients with head injury.[16,17] When the postoperative evoked potentials were normal and there was no serious secondary systemic insult, the electrical data correctly forecast functional recoveries in 90 per cent of our patients with ASDH. Furthermore, all patients with severely abnormal evoked-potential data died. Postoperative evoked-potential studies appear to be an important means of evaluating the clinical efficacy of prompt surgical decompression.

Of the several factors that influence mortality, the results of a neurologic examination obtained before surgical decompression of a traumatic intracranial mass lesion can be a major determinant of outcome. This point has been well documented by Jennett et al.[18,28,29] and others.[8,9,13,19-21,30] McLaurin and Tutor[31] stated that level of consciousness was the most important guide to the need for surgical intervention and was of considerable prognostic value. Eighteen per cent of their 90 patients were conscious up to the time of operation; they had only six per cent mortality. The remaining 82 per cent of their patients were comatose and had 77 per cent mortality. To avoid this bias, we selected only patients who were comatose before surgical intervention. Motor, oculomotor, and pupillary dysfunction before surgery were key determinants of outcome, as illustrated in Table 2. McLaurin and Tutor[31] and Richards and Hoff[8] found a 75 per cent mortality rate associated with pupillary abnormalities, as compared with 35 per cent in patients whose pupils were normal. Jamieson and Yelland[24] noted 85 per cent mortality in patients with bilateral pupillary abnormalities. In our series, 25 per cent of the patients with bilaterally absent pupillary light reflexes had a functional recovery.

The presence of decerebrate rigidity before operation also had a devastating effect on outcome. Mortality in patients with ASDH and decerebration has been reported to range from 65 to 90 per cent.[3-9,24] Only 29 per cent of our patients with decerebrate rigidity had a functional recovery.

Prompt surgical intervention to evacuate an ASDH has been emphasized in the head-injury literature,[5,7,10,11,13,32] but little evidence is available to support its efficacy. Most of the stress has been on prompt evacuation of extradural hematomas.[5,7,14,33] For instance, Mendelow and his colleagues in Edinburgh[14] pointed out in 83 patients with extradural hematomas that the average delay from the time of deterioration of the level of consciousness until the surgical decompression was 15.7 hours in nonsurvivors and 1.9 hours in survivors. Putnam and Cushing[34] focused attention on ASDH as a surgically curable pathologic entity, but Chambers[10] was one of the first to emphasize that prompt surgical removal of a traumatic intracranial hematoma, whether extradural or intradural, was beneficial even if the the patient's neurologic condition appeared terminal. Gutterman and Shenkin demonstrated that patients with traumatic decerebration associated with either an extradural or intradural hematoma had 40 per cent mortality when craniotomy began within four hours of the onset of decerebration but had 100 per cent mortality when the procedure began after six hours.[5] Cooper et al.,[4] in a retrospective analysis of 50 cases of traumatic ASDH, reported that in 45 fatalities an average of six hours elapsed from admission to operation, as compared with 4.4 hours in the five survivors.

Richards and Hoff, in 1974, recorded a 75 per cent mortality rate in 100 patients with an ASDH who had surgery an average of seven hours after injury.[8] Emergency air ventriculostomy and computed tomography have allowed us to reduce the time from injury to operation to 5.4 hours in the average patient with an intracranial hematoma, including patients with an ASDH (Table 1). Functional recovery from an ASDH, however, was associated with an average of only 2.8 hours of delay (Table 2).

Figure 1 illustrates the fact that patients who underwent surgery for an ASDH within four hours of

the injury had a 60 to 70 per cent functional-recovery rate, as compared with only 10 per cent in those who had the operation more than four hours after the injury ($P<0.0001$). Severe morbidity and mortality were three times greater in patients who underwent surgery after four hours from the injury. Rapid removal of the ASDH prevents prolonged preoperative increased ICP and brain shift and therefore diminishes the probability of subsequent focal-tissue ischemia or brain-stem compression or both. In addition, the longer the delay between injury and admission, the greater the adverse effects of hypoxemia and hypotension — common secondary insults associated with head injury.[32]

Comparison with Other Series of Patients with ASDH

In assessing a patient with an ASDH, the rate of evolution of the mass lesion is usually gauged from the time between the injury and the operation, but it has also been gauged from the moment of neurologic deterioration or admission to the operation. Earlier investigators[3,9,23,24,31] have reported that comatose patients with an ASDH who had the operation within 24 hours of injury had a very high mortality rate. According to Browder[3] and Cooper et al.,[4] little progress has been made since 1943 to improve the mortality rate of more than 80 per cent observed in patients with ASDH (Table 4).

Our mortality rate in 82 patients with ASDH was 57 per cent, with good to moderate recoveries in 34 per cent. These data compare favorably with the results of other investigators[3-5,7,24,31] (Bricolo A, Turazzi S. Personal communication) (Table 4). The mortality rate in patients with ASDH ranged from 57 per cent in our series to 90 per cent in that of Cooper et al.[4] Forty-eight of the 50 patients described by Cooper et al.[4] were decerebrate or had abnormal unilateral or bilateral pupillary light responses. Limiting patient selection to these severe cases biased these workers' results and added to the pessimistic

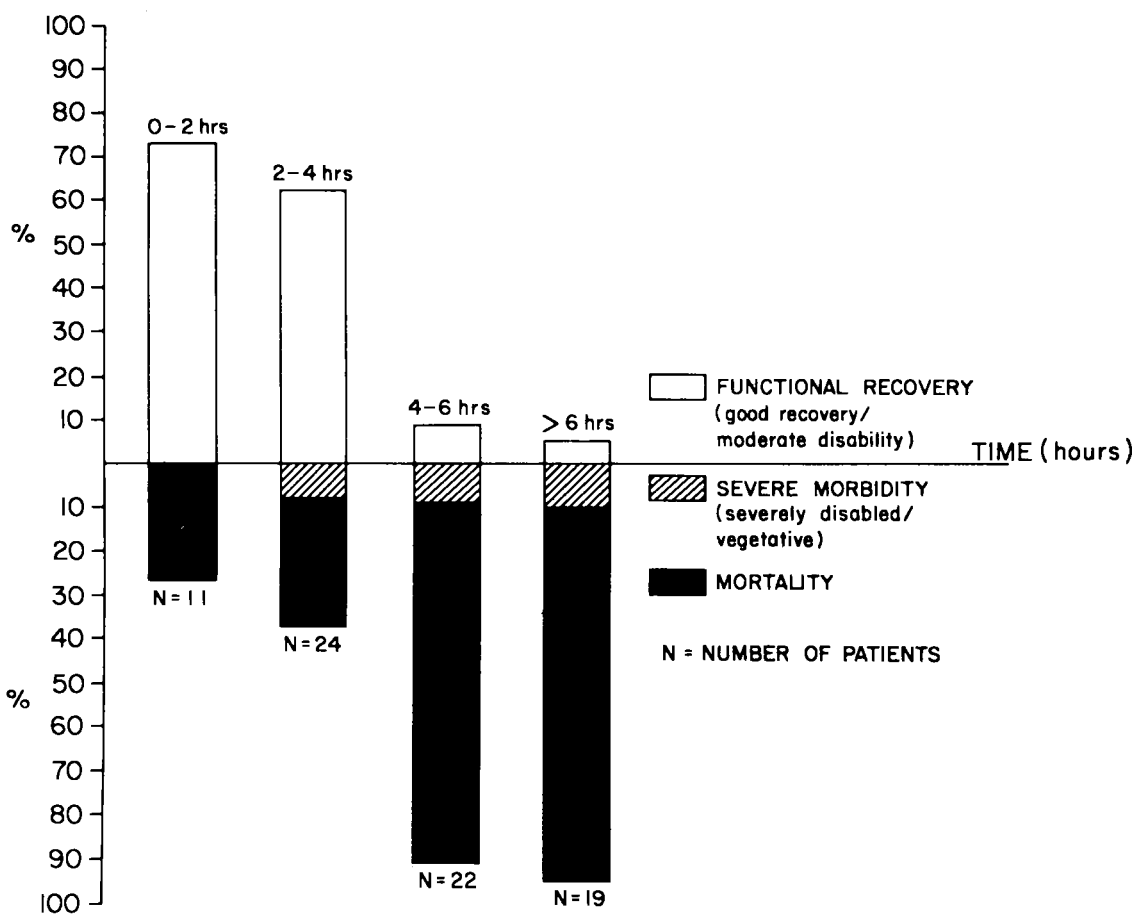

Figure 1. Influence of Time Delay from Injury to Surgical Intervention on Outcome in 76 Patients with Acute Subdural Hematoma.

In six other patients, the onset of injury could not be determined. There was a significant increase in mortality when the delay exceeded four hours ($P<0.0001$). The range of time delay in the group who underwent surgery more than six hours after injury was 6.2 to 18.3 hours.

Table 4. Reported Studies of Acute Subdural Hematoma in Comatose Patients.

Series (Year)	No. of Patients	Mortality	Functional Recovery
		%	
Browder (1943)[3]	51	82	—
McLaurin and Tutor (1961)[31]	74	73	16
Gutterman and Shenkin (1970)[5]	14	65	35
Ransohoff et al. (1971)[7]	35	60	28
Jamieson and Yelland (1972)[24]	207	63	—
Richards and Hoff (1974)[8]	100	75	14
Cooper et al. (1976)[8]	50	90	4
Bricolo and Turazzi (1980) *	94	67	26
Our series (1981)	82	57	34

*Bricolo A, Turazzi S. Personal communication, June 1980.

attitude toward the treatment of the traumatic ASDH.

In our series, the patients with less severe neurologic deficits on admission and low postoperative ICP had significantly better outcomes. Age did not significantly affect outcome among our 82 comatose patients with ASDH, but older patients were more prone to ASDH; therefore, age may have predisposed this group of patients to a higher mortality rate than that of patients with other head injuries. This possibility has previously been recognized,[4,8,11,17,24] as have the facts that men have worse outcomes than women and are more prone to ASDH, and that motor-vehicle-related accidents are a less common cause of ASDH than of other types of head injuries.

Of most practical importance, however, was the finding that comatose patients with ASDH who underwent rapid surgical decompression (within four hours of injury) had decreased mortality: 30 per cent (Fig. 1). This mortality rate represents an improvement over that of any published series and over the low rate of 57 per cent in all 82 patients with ASDH in our series. The mortality rate of 30 per cent in patients with ASDH treated within four hours approaches our rate of 29 per cent in patients with other severe head injuries who did not have a subdural hematoma (Table 1). Rapid transport to a hospital that is capable of providing prompt diagnosis and surgical decompression within four hours of the injury will substantially reduce mortality in patients with traumatic ASDH. Since ASDH develops in approximately 25 per cent of patients who are admitted while comatose from head injury, this information is critically important for rescue squads, emergency-room physicians, and tertiary physicians who are directly involved in the transport, diagnosis, and treatment of these patients.

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MEDICAL PROGRESS

GLUCAGON AND THE A CELL

Physiology and Pathophysiology

(First of Two Parts)

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IN this review we examine the role of glucagon, a polypeptide produced by the islets of Langerhans and a biologic antagonist of insulin. The effects of glucagon balance those of insulin so that fluctuations in blood sugar levels in response to sudden changes in fluxes of fuels are prevented.

By definition, relative hyperglucagonemia is present in all forms of diabetes in which glucose production exceeds glucose disposal and thus causes "endogenous hyperglycemia"; in the absence of glucagon-like biologic activity the massive hepatic overproduction of glucose and ketones observed in uncontrolled diabetes does not occur, even in the total absence of insulin. The hyperglucagonemia in Type I diabetes results from loss of B cells, which deprives A cells of the influence in changing insulin levels, an important regulator of glucagon secretion. Glucagon and glucose levels in Type I diabetes can be restored to normal simply by delivering insulin in a manner that simulates the normal pattern of insulin secretion. In Type II diabetes, however, the cause of the relative hyperglucagonemia is unclear, and factors other than or in addition to insulin deficiency may be involved.

UNderstanding of the physiology and pathophysiology of the glucagon-secreting pancreatic A cell has advanced substantially during the past decade. The vital role of normal A-cell function in the maintenance of intercibal hepatic fuel production has been convincingly established, and the metabolic consequences of abnormal A-cell function in diabetes are well defined.[1-3] In this review of recent progress in the study of glucagon the A cell is shown to be an essential component of the normal islets of Langerhans — highly organized communities of peptide-secreting cells that perform one of the most important of physiologic functions, the distribution of fuels to the tissues of the body according to need. We emphasize that disorganization of intercellular relations within these cellular communities causes dysfunction of the A cells and serious aberrations in fuel homeostasis.

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THE A CELL AND FUEL HOMEOSTASIS

Appropriate distribution of fuels to the tissues of an organism in accordance with ever-varying supply and demand requires that special priority be given to the fuel needs of the most vital of all tissues, the brain, for which glucose is normally the sole source of energy. Normal cerebral function requires the delivery of approximately 6 g of glucose during each hour. This requirement is not readily fulfilled if the arterial plasma glucose level falls below a critical level. The dependence of the brain on glucose thus creates a constant need for a specific fuel that transcends in importance all competing needs and explains the remarkably staunch defense against hypoglycemia that nature has evolved. In this defense glucagon has its most important role.

Morphofunctional Relations of the Islets

The glucagon-secreting A cells, one of the four types of islet cells identified to date in human beings, have a distinctive and nonrandom distribution within the islets.[4] They form the outer rim of the cortex of each islet (Fig. 1*a*) and make up about 25 per cent of the endocrine pancreas.[5] In the ventral pancreas they are sparse, with pancreatic polypeptide-containing cells taking their place.[6] The insulin-containing B cells constitute at least 60 per cent of the islet and form its medulla.[5] Somatostatin-containing D cells are