TRAUMATIC SUBDURAL HEMATOMA—ACUTE, SUBACUTE AND CHRONIC
AN ANALYSIS OF SEVENTY OPERATED CASES

FRANCIS ECHLIN, M.D.*

New York, N. Y.

(Received for publication September 21, 1948)

In the past it has been common practice to class all subdural hematomas as acute or chronic. Acuteness has been estimated on the basis of elapsed time from the injury, or on the degree of associated brain damage. According to one classification, all hematomas that cause death or come to operation before the 21st day after a cranio-cerebral injury are described as acute (Kennedy and Wortis8). In another classification, cases of subdural hematoma that still show unhealed or acute brain injury are placed in the acute group, the others being classed as chronic (Munro15). In still another classification, an acute subdural hematoma is regarded as the result of a severe head injury, usually with a fracture of the skull and more or less extensive brain laceration, whereas a chronic subdural hematoma is considered to be unassociated with severe brain injury and the history of trauma, as a rule, is slight or absent (Peet17).

As Munro15 has pointed out, any classification of subdural hematomas into groups is artificial, for as Munro,13 Laudig, Browder and Watson,11 and King10 have clearly demonstrated, the chronic form of traumatic subdural hematoma, so well described in the literature6,8,12,14,18,19,20 is nothing more than a later variant of the acute phase.

Acute hematomas become chronic only gradually, and progressive symptoms appear as the hematoma enlarges by the acquisition of further fluid, usually through the process of osmosis5,16,21 Therefore if any classification is to be used it should clearly indicate that there is a gradual transition between the so-called acute and chronic hematomas.

The purpose of this paper is to combat the present tendency to class all hematomas as acute or chronic. Such a classification is misleading and fails to emphasize the existence of a large group of subdural hematomas which, neither on clinical nor pathological grounds, can be satisfactorily classed as acute or chronic. If this is not clearly recognized, the average observer may fail to diagnose the subdural hematoma that ends in a fatality about the end of the first week to several weeks after injury.

In reviewing the cases of subdural hematoma to be presented, it is apparent that they fall roughly into three classes, on the basis of the clinical course, type of hematoma found at operation, and degree of associated brain injury; namely, acute, more or less subacute, and chronic. There is con-
siderable overlap between these three classes of hematoma, on the basis of
the criteria mentioned, but the existence of a subacute variety (as first sug-
gested by Bucy, Gurdjian and Webster) as well as an acute and chronic
one, is borne out on analysis of the findings in patients operated upon at
different time intervals following trauma.

MATERIAL

The material presented is based on a study of 70 patients with subdural
hematomas operated upon during the 20-month period from January, 1941
to July, 1942, on the Neurosurgical Service, 3rd Surgical (New York Uni-
versity) Division at Bellevue Psychiatric Hospital. During this interval
approximately 7916 patients entered the hospital with a history or signs of
recent head injury. Of these, 1415 showed signs of intracranial injury, con-
sidered severe enough to admit them to the neurosurgical ward. It was
among this group that most of the 70 subdural hematomas were found. A
few of the more chronic ones, however, were transferred from the psychiatric
or medical services, having been received there with a diagnosis other than
head injury.

These statistics show that the incidence of subdural hematomas was less
than 1 per cent in the entire series of head injuries, and less than 5 per cent
in the group of cases of severe head trauma.

Of these 70 patients with subdural hematoma, 50 were operated on by
me, and 20 by Henry Wigderson, but with 3 exceptions I followed them all
pre- and postoperatively.

Thirty-five additional patients, suspected of having a subdural collec-
tion of blood as the cause of their symptoms, were explored. In many of these
cases blood was found in the subdural space but they were not classified as
subdural hematomas.

In order to emphasize the difference in the clinical course of the illness,
and in the pathological nature of the hematomas in the various patients,
they will be analyzed in groups according to the time elapsed since injury.
Analysis of other data pertinent to the diagnosis and treatment of subdural
hematoma will follow.

The clinical course and nature of the hematoma found in patients
operated upon within 24 hours following trauma

There were 10 patients in this group. Trauma was severe in all and
consciousness was lost immediately following injury. At no time did a lucid
interval follow. The condition changed from coma to stupor in 5 patients,
and to semi-stupor in the remaining 5. After thus showing some degree of
improvement, stupor became progressively deeper in each instance. The
hematoma removed at operation consisted of a large, currant-jelly-like clot
with some fluid blood in 9 cases, and fluid blood alone in 1. Of the 10 pa-
tients in this group only 1 survived. The others all died within 5 days.
Autopsy revealed extensive traumatic brain injury in all cases.
Comment. These 10 patients suffered from a severe brain injury plus a fresh subdural hematoma. Death in 9 cases was due almost if not entirely to direct brain trauma, the hematoma, although large, being incidental. Survival in 1 case may be attributed to removal of the hematoma.

The clinical course and pathological nature of the hematoma in patients operated upon 2 to 7 days after injury

There were 11 patients with subdural hematoma operated on 2 to 7 days after injury. Trauma was severe with immediate loss of consciousness in 9 cases. Two patients had moderate trauma and did not lose consciousness.

Nine of the patients were admitted to the hospital immediately after injury, 1 was brought to the hospital 12 hours following injury, and 1 in 24 hours. All the patients remained on the neurosurgical ward until operation.

One of the patients continued to be comatose from the time of injury to operation on the 3rd day. Eight patients, after recovering consciousness, were very drowsy and disoriented and subsequently became much more drowsy or stuporous prior to operation. In 5 cases the onset of sustained stupor was sudden and in 4 of these the stupor was precipitated by a seizure. One patient had a lucid interval for 5 minutes after injury and then rather rapidly became stuporous. Stupor persisted 24 hours and was replaced by drowsiness which progressively deepened. The final patient did not lose consciousness with injury but went home in an alcoholic condition; 19 hours later he had a seizure, became comatose for about 1 hour and following this he was confused, drowsy and aphasic; 24 hours later he had another seizure, became progressively comatose and was operated upon.

The hematomas removed at operation from the 11 patients consisted of relatively fresh blood, in the form of large (9 cases) or small (2 cases) currant-jelly-like clots, accompanied by varying amounts of dark brown to black fluid.

Four of these patients survived and 7 died.

Comment. The clinical course, as well as the pathological nature of the hematomas, was acute or relatively acute in these 11 patients. The clinical course in most cases was directly influenced, in an important manner, by an associated brain injury.

The clinical course and nature of the hematoma in patients operated upon from the 7th to the 21st day following injury

There were 30 patients in whom a large subdural hematoma was found at operation 7 to 21 days after injury. In 19 cases operation was performed on the 10th to the 15th day following trauma.

A history of head trauma was obtained in all cases. In 27 the trauma was accompanied by immediate loss of consciousness. In 2 the injury was mild, without loss of consciousness, and in 1 the history is indefinite. Twenty-four of the patients were brought to the hospital within a few hours of the
accident. The other 6 were admitted within 8 days following injury. All of
the patients, with 1 exception, were under observation on the neurosurgical
ward throughout the course of their illness. The exception was a patient who
signed out of the hospital on the 5th day after injury, to be returned in stupor
8 days later.

One of the most important clinical signs in arriving at a diagnosis in
this group of 30 cases was the state of consciousness. All of the patients who
lost consciousness after injury subsequently regained it. Ten of the patients
remained drowsy and disoriented throughout, and before operation was
undertaken drowsiness had become progressively deeper.

Fourteen patients became mentally alert and cooperative but showed
some degree of disorientation. Each of these 14 individuals later went
through a period of progressive, usually fluctuating drowsiness preceding
operation.

Only 6 patients could be described as reaching a state of mental clarity
following their injury, but 4 of these subsequently became drowsy and then
suddenly stuporous prior to operation. In 3 instances the stupor was pre-
cipitated by a convulsive seizure. In the remaining 2 patients the diagnosis
was made by encephalogram before the onset of severe symptoms.

On a pathological basis it was found at operation that 15 of the hema-
tomas, in this group of 30 patients, more closely resembled the so-called
“chronic” than the “acute” variety of subdural hematoma, and consisted
of dark, coffee-colored fluid containing, in 5 cases, small, soft blood clots in
various stages of liquefaction. In 6 of these 15 patients thin membranes
surrounded the hematoma, in 1 case as early as the 8th day after injury. The
other 15 hematomas varied in consistency from currant-jelly-like clots with
black, tarry or brown fluid to 2 cases of so-called subdural hydroma contain-
ing canary-yellow fluid.

In this group of 30 patients, 23 lived and 7 died.

Comment. This entire group of 30 patients, operated on 7 to 21 days after
an acute injury of the brain, would ordinarily be classed as having an acute
subdural hematoma. However, 20 of the patients followed a subacute
clinical course, in that they became alert, cooperative and showed signs of
improvement for a week or more. In 15 of the 30 cases the pathological na-
ture of the hematoma removed at operation more closely resembled that
seen in “chronic” cases.

Progressive symptoms and signs of brain dysfunction in most of the
cases was due to a subdural hematoma. Trauma may have disturbed the
normal physiology of the brain and rendered it more susceptible to the
pressure from a hematoma in some instances, but, in the majority, trau-
matic brain injury itself was not the cause of progressive neurological symp-
toms and signs. On clinical and pathological grounds then, as well as on the
degree or part played by an associated brain injury, many of these cases are
best described as subacute.
The clinical course and nature of the hematoma in patients operated on from the 22nd to the 31st day after injury

There were 10 patients in this group and a history of trauma to the head was obtained in all. Nine patients lost consciousness with injury and in 1 there was no history of unconsciousness. Five of the patients were taken directly to the hospital from the scene of the accident and remained until after operation. Three were admitted 3 to 20 days after injury. Two were admitted immediately following injury, left the hospital in several days, and were readmitted 25 to 27 days later.

Of the 10 patients, 4 became mentally clear for varying periods and the remainder became alert but showed some memory defect or disorientation. Five of the patients later showed a progression in their disorientation with drowsiness. Five did not show progressive drowsiness, but other features in their clinical pictures suggested the possibility of a subdural hematoma and the diagnosis was confirmed by encephalogram.

At operation the hematomas in the 10 patients resembled the usual fluid ones seen in many chronic cases, but 4 patients had in addition soft, brownish-colored clots and 1 patient, operated on 31 days after injury, an organized hematoma of the consistency of liver. Membranes were present about the hematoma in at least 4 cases.

All of the 10 patients in this group survived.

Comment. According to one classification, all of these patients would be regarded as suffering from a chronic subdural hematoma since they were operated on more than 21 days from the time of injury. According to another classification, many of the patients would fall into the class of acute subdural hematoma, since the majority of them had a relatively recent brain injury which certainly was not completely healed in all cases.

Actually, at least 5 of the 10 patients followed a subacute clinical course and, although they became alert for varying intervals, nevertheless remained disoriented and later showed progressive disorientation and drowsiness before operation. Five other patients in this group, in whom a suspected hematoma was verified by encephalogram, could be classed as either subacute or chronic, but certainly not as acute on the basis of the clinical course nor pathological findings.

CHRONIC SUBDURAL HEMATOMA

Nine patients in the series were classed as suffering from a chronic subdural hematoma. A history of severe trauma was obtained in only 2 cases and these individuals were admitted 30 to 42 days thereafter. No definite history of trauma was obtained in the remaining 7 patients, although this does not exclude such an etiology.

Eight of the patients had shown progressive drowsiness before they were seen by the neurosurgical staff. Six of them were disoriented, 1 appeared psychotic and 2 were in coma on admission to the neurosurgical ward. Only 1 patient was alert and cooperative pre-operatively. The diagnosis of an in-
TRAUMATIC SUBDURAL HEMATOMA

tracranial lesion in this patient and 1 other was made by encephalogram.

All of the 9 patients had hematomas which were fluid with surrounding membranes. In 7 cases the fluid was of a brown color and in 2 it was yellow. A few mm. of soft, shaggy, brown clot was present on the inner surface of the outer membrane in 4 cases.

Seven of the patients survived and the 2 who were in coma at the time of operation died.

LOCALIZING SIGNS IN SUBDURAL HEMATOMAS

All of the 70 patients described in this paper showed abnormalities in the neurological examination.

In 29 cases there were localizing signs which correctly indicated the side of the hematoma, but in 12 the signs pointed to a cerebral lesion on the side opposite the hematoma. In 21 cases the neurological findings were inconclusive so far as localization was concerned. There were 8 cases of bilateral subdural hematoma. In 3 of these the neurological examination indicated a probable unilateral lesion, and in 5 the signs were not localizing.

The most common signs present were enlargement of a pupil, a slight facial weakness of central type, a diminished abdominal reflex unilaterally, inequality in the deep reflexes, a positive Babinski, and a slight weakness of one arm or leg. These signs when present from the time of admission were of little value in diagnosis or in localization. Signs that appeared at a later date, however, and especially when progressive, were of distinct value in making the presumptive diagnosis of subdural hematoma.

Marked hemiparesis to hemiplegia was present in 14 cases. In 8 patients the weakness was on the same side as the hematoma and in 6 on the opposite side. In no case was there positive evidence that a marked hemiparesis or hemiplegia was due solely to a subdural hematoma, with the exception of 2 cases of chronic hematoma. This is a most important point in the differential diagnosis between subdural hematoma and spontaneous or traumatic intracerebral lesions.

In 9 cases a marked weakness of one arm or leg was present from the time of admission, shortly after injury, and direct brain trauma probably was the chief causative factor. Of these 9 cases, 5 were among the patients operated upon in the first 24 hours. In 3 patients, who had moderate weakness of one side, a hemiplegia developed following a convulsive seizure.

Localizing value of a unilateral dilatation of the pupil. Inequality of the pupils was noted in 48 patients with a unilateral subdural hematoma. In 24 patients, at the time of operation, the larger pupil was on the same side as the hematoma and in 24 on the opposite side. In many of these cases there had been some fluctuation in the relative size of the pupils.

A relatively marked dilatation of one pupil was present at the time of operation in 25 cases. In these patients the large pupil was on the side of the hematoma in 14, and on the opposite side in only 7 cases. Quite marked enlargement of one pupil, however, was also noted in 4 patients with a bilateral hematoma.
From the above it is seen that a marked dilatation of one pupil is of some localizing significance, whereas a slightly dilated pupil is of no value in indicating the side of the hematoma.

CONVULSIVE SEIZURES

Convulsive seizures occurred in 17 patients. In the patients under discussion it is impossible to say whether the seizures were due to direct brain injury, alcoholism (of which there was a history in the majority of patients) or the subdural hematoma. Some of the patients may have had chronic epilepsy. However, the high incidence of convulsive seizures is a serious complicating factor in the presence of a brain injury and subdural hematoma, and frequently precipitated a hemiparesis or a continuing stupor in an already drowsy patient.

SUDDEN ONSET OF STUPOR OR COMA

In 11 patients, who were already drowsy, there was a sudden onset of persisting stupor or coma, and in 7 this was preceded by a convulsive seizure. Four of the patients in whom a seizure precipitated stupor were among the group operated upon on the 2nd to the 6th day after injury.

THE OPTIC FUNDI

Papilloedema was uncommon in this series of cases during the first 2 weeks after head trauma.

CAUSES OF DEATH

There were 25 deaths among the 70 operated cases. Of these, 9 occurred in the group operated upon in 24 hours after trauma, and may be attributed largely to associated brain injury.

Of the remaining 16 patients, 10 were in coma and 4 in stupor at the time of operation and were helped little by the removal of the hematoma. Five of the patients operated upon before the 31st day after trauma could probably have been saved by earlier operation, and this is certainly true of the 2 with chronic subdural hematoma.

It is very evident that if a patient is allowed to become comatose or deeply stuporous his chances of survival are greatly lessened even though the hematoma is evacuated.

Evans and Scheinker's\textsuperscript{4} demonstration that an expanding lesion may produce actual thromboses of large vessels as a result of herniation of cerebral tissue over the free edge of the tentorium or beneath the falx, may explain the coma and lack of recovery following operation in these cases.

At the time of operation 11 patients were comatose (exclusive of those operated upon in the first 24 hours) and 10 of them died; 13 patients were stuporous and 4 of these died.

One patient died of an operative infection and 1 of meningitis 4 months postoperatively.
PULSE, CEREBROSPINAL FLUID PRESSURE AND BLOOD PRESSURE

During the study a quite detailed analysis of the relationship between blood pressure, pulse, cerebrospinal fluid pressure and state of consciousness has been made, but will not be presented in detail here. The findings are in keeping with those reported by Browder and Meyers.\textsuperscript{1} In summary: a pulse rate below 60 was a common finding in subdural hematoma, being recorded in 32 cases, and was noted in the presence of a normal CSF pressure in 6 cases. In 13 cases when the CSF pressure was over 300 the pulse rate was over 60.

The CSF pressure was recorded on the day of operation in 48 cases. In 15 patients the lumbar puncture pressure was below 160 and in 33 above this level. In 17 patients the pressure was above 290 and in 13 it was 300 mm. of water or over. The lumbar CSF pressure bore no direct relationship to the state of consciousness. It was normal or below normal in 10 patients who were stuporous and elevated in many patients who were alert. The blood pressure likewise showed little if any relationship to the CSF pressure except perhaps in the first 24 hours after injury.

INCOMPLETE REMOVAL OF SUBDURAL HEMATOMA

In some cases, so-called recurrent subdural hematomas are due to incomplete removal of the clot at the first operation. The following cases are examples of incomplete surgical removal of a hematoma.

Four patients were operated upon on the 7th to the 14th day after injury. In each case the subdural space was exposed through bilateral posterior parietal burr holes and a subdural hematoma was found, consisting of coffee-colored fluid and a few clots. A thin membrane was present in 3 of the cases. In all patients a large quantity of the dark coffee-colored fluid was obtained, and it was felt that the patient's hematoma had been adequately removed. In addition a rubber tissue drain was left in the subdural space, through a stab wound in the scalp, for 24 hours.

Two of the patients improved temporarily, but within 1 to 2 weeks became progressively drowsy and presented other signs suggesting the presence of subdural hematoma. The other 2 patients failed to improve following operation. These 4 patients were all re-explored through a subtemporal decompression on the side of their original hematoma. In each case a large currant-jelly, or milk-chocolate type of clot, plus old fluid, was found.

Three of the patients survived and 1 died.

It was this type of experience that indicated that some subdural hematomas, although they may appear to be largely fluid, may contain large clots which cannot be adequately removed nor visualized through the posterior parietal approach.

OPERATIVE APPROACH

Following the above-mentioned experience it became our custom to explore for all subdural hematomas through the bilateral temporal approach. With the patient in a semi-sitting position supported by a small posterior
neck rest, a burr hole is made just anterior to and above each ear. The
patient, however, is draped so that subtemporal decompression and occipital
burr holes for ventriculography may also be carried out, if necessary. The
great majority of subdural hematomas will be found through this approach.
Some will be found only after passing a curved instrument into the subdural
space, which may be done with ease in this region. When a semi-solid clot is
present it can be evacuated after enlarging the subtemporal decompression.
One of the obvious advantages of this approach is that if a large opening in
the bone must be made to remove the clot the decompression is adequately
protected by the temporal muscle and the fascia, which is not the case in the
posterior parietal approach. Another advantage is the one already men-
tioned, that the solid portion of the clot most often lies in the temporal
region and incidentally frequently covers the lateral portion of the temporal
lobe to the base of the middle fossa. Clots in this latter region are not rare,
as suggested in the literature. When exploration through the temporal ap-
proach fails to reveal a hematoma, it is obvious that additional burr holes
may be indicated.

It was necessary to turn a bone flap in only 1 case in the series of 70, and
this was to remove an organized liver-like clot, 31 days after injury.

SUMMARY

On clinical and pathological grounds, there is a group of patients who
present the picture of an acute subdural hematoma and another group
which may be classed as having a chronic subdural hematoma. In addition,
there is a large number of individuals with subdural hematomas, who suffer
from symptoms that are more correctly classed as subacute. Many of the
patients in this latter group, although they remain drowsy or confused in
some instances, have had time to recover, or largely recover, from their
initial brain injury, but lapse into deepening drowsiness or show other signs
of a severe progressive intracranial lesion in a week to several weeks after
trauma. At operation, the hematomas in these individuals vary remarkably
in structure, and pathologically, at times, more closely resemble the chronic
than the acute type of lesion.

It is not with terminology that this paper is concerned, but rather with
stressing the fact that many patients, suffering from large collections of
blood in the subdural space, do not present an acute nor a chronic picture
clinically or pathologically. Unless this is stressed the uninitiated may fail
to be on the alert for the subdural hematoma that kills the patient at the
end of the first week to several weeks after injury.

REFERENCES

TRAUTOMATIC SUBDURAL HEMATOMA


