SUBDURAL HYGROMA
A REPORT OF SEVEN CASES
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The subdural space still remains an unsolved mystery in medicine. The anatomical details and studies of the subdural space are meagre indeed.\textsuperscript{15,28} Penfield made anatomical studies of the subdural space in dogs and estimated the contents to be a few drops to one cc. per kilogram. Estimations of the normal amount of fluid in the subdural space in humans have not been made, many neurosurgeons feeling that no subdural fluid is normally present. Operations at the vertex of the skull obviously fail to reveal the fluid because of its gravitation to the base. This basal subdural fluid has been repeatedly shown to me by Dr. Temple Fay\textsuperscript{9} in his Gasserian ganglion operations by the transtemporal approach. By nicking the subtemporal dura, large quantities of subdural fluid well out of the wound, insuring ample and easy approach to the Gasserian ganglion.

The origin of excessive subdural fluid accumulations, designated subdural hydroma by Dandy,\textsuperscript{8} has likewise created heated controversies. Dandy has listed three causes. Trauma is the foremost cause, resulting in arachnoidal tears with leakage of cerebrospinal fluid into the subdural space; secondly, subdural effusions may arise secondary to infection of overlying bone, as in mastoiditis; and thirdly, accumulations are occasionally seen secondary to a communicating hydrocephalus with tearing of the arachnoid at the basal cisterns. Most neurosurgeons believe that excessive collections of subdural fluid are the result of tearing of the arachnoid, the rent acting as a ball-valve mechanism preventing the return of the cerebrospinal fluid to its original confines. Adson\textsuperscript{7} was fortunate to observe such a tear in the arachnoid in one of his cases. All cases of traumatic subdural fluid effusions are probably not due to a tear in the arachnoid. McConnell's\textsuperscript{17} case No. 7 was quite interesting. The first drill opening made 7 hours after injury failed to reveal any subdural fluid while 9 days later, on re-exploration, a copious stream of yellow subdural fluid was evacuated despite repeated spinal fluid drainages. If a tear in the arachnoid were responsible for the subdural collection in this case, it should have been found at the first exploration. The author has had a similar experience (see Case 7).

The pathology of the fluid removed has likewise offered difficulties since gross contamination with blood occurs when the dura is opened. However, the protein content as reported by various investigators varies from 30 to 5000 mgm. per 100 cc. The fluid is variously described as clear, xanthochromic, or blood-tinged. The amount may vary from 30 cc. to 2300 cc. (Cohen's case\textsuperscript{6}).
SUBDURAL HYGROMA

HISTORICAL REVIEW

The first descriptions of post-traumatic subdural hygromas were written by Professor Payr\(^1\) of Germany during the first World War in 1916. His original descriptions under the name of meningitis serosa traumatica are complete in every detail. Little has since been added to his classical work in the *Medizinische Klinik*. He reported 4 cases: one occurring in the frontoparietal area, two over the temporal lobe and one in the posterior fossa. These four cases were due to tangential bullet wound injuries of the skull.

Dr. Charles Mayo\(^2\) in 1894 operated upon a brain cyst which undoubtedly appears to be a true subdural hygroma. Dr. Naffziger's report\(^3\) of 1924 was important as it stimulated a growing interest in this condition among neurosurgeons in this country. He discussed the etiology and symptomatology, and outlined the most rational treatment for the condition.

In 1927 there appeared the interesting report by Ira Cohen.\(^4\) He described a case in which a massive accumulation of subdural fluid had caused displacement and compression of the brain with focal and increased intracranial pressure symptoms.

With the exception of the writing of Dandy\(^5\) in 1932 in Dean Lewis’ *Practice of Surgery*, a decade had passed before interest was again revived by the reports of the Mayo group (Love\(^6\) in 1937, Walsh and Shelden\(^7\) in 1937, and Da Costa and Adson\(^8\) in 1941).

In England, the first and perhaps most extensive report was that of Adams McConnell\(^9\) in 1941. His 32 cases were well described and verified by operation. He divided them into 4 groups: Group 1—patients whose condition was deteriorating, usually within 48 hours of injury and characterized by an expanding lesion (8 cases); Group 2—patients who were in the process of recovery from definite general cerebral contusion, but in whom full recovery seemed to have been checked—passive space-occupying lesion (6 cases); Group 3—patients suffering from headache, dizziness, and vague symptoms of nervous instability dating from a head injury (4 cases); and Group 4—patients in whom headache and occasionally some definite nervous symptom, constant for the individual, started some time after a head injury—best operative results (12 cases).

In 1942 Scott\(^10\) reported 3 cases of prolonged stupor associated with subdural hygromas and in the following year he\(^11\) described a case of acute subdural hygroma which simulated the syndrome of an extradural hemorrhage. Siros\(^12\) in 1942 reported a case in a 9-year-old boy which was successfully treated surgically. In the following year Ley, Roca de Vinals and Sard\(^13\) wrote on collections of the subdural space.

With the advent of World War II an interesting group of subdural effusions secondary to blast concussion has been described by Abbott, Due and Nosik.\(^1,2,3\) These authors reported a total of 37 cases stressing, in particular, the psychiatric symptoms and the pre- and postoperative responses to standard psychometric examinations. Haynes\(^14\) added three more case reports in *War Medicine* in 1944. In the same year, McConnell\(^15\) added 5 cases...
of post-traumatic amnesia due to subdural hygromas. All cases were verified by operation.

Table I outlines the clinical features of the cases reported up to the present time including the author's seven cases. All of McConnell's cases are not included but only the most significant and detailed case was chosen from each of the four groups.

CASE REPORTS

Case 1. Mr. R. J., age 45, admitted August 1944.

Patient was struck in left temporal area with handle of a compressor hammer. Rendered unconscious immediately. Seen immediately following the accident but presented no focal signs. X-rays of the skull were normal. He had headache, however, and was instructed to return on the following day. Somewhat improved for the next three days, but headache persisted and therefore he was admitted for observation. Lumbar puncture: spinal fluid clear; pressure 10 mm. of Hg. Following this procedure the headache improved and he was discharged.

Oct. 8, 1944. Readmitted because of return of generalized headache and weakness of left arm. Grip notably diminished on left. Marked fatigue and nervousness. Deep tendon reflexes slightly increased on left. Fundi normal. Re-examination of skull by roentgenography showed the pineal gland to occupy a normal position. Blood counts, urinalysis and serology were normal.

Fig. 1. Case 1. Encephalogram. Obliteration of subarachnoid channels over both hemispheres. A bilateral subdural hygroma was disclosed at operation.
<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Unconsciousness</th>
<th>P.R.</th>
<th>B.P.</th>
<th>Focal Symptoms</th>
<th>Other Cerebral Symptoms</th>
<th>Increased Intracerebral Pressure</th>
<th>Spinal Fluid</th>
<th>Subdural Fluid</th>
<th>Roentgenograms</th>
<th>Surgery and Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Love 1937</td>
<td>34</td>
<td>F</td>
<td>5 mins.</td>
<td>70.71</td>
<td>+</td>
<td>Argyll-Robertson</td>
<td>Headache, belligerent, irritable, poor memory.</td>
<td>Protein, 90 mgm. %, + Wassermann, Gold curve</td>
<td></td>
<td></td>
<td>Unilateral trephine. Good recovery.</td>
<td></td>
</tr>
<tr>
<td>Walsh and Shelden 1933</td>
<td>9</td>
<td>M</td>
<td>1–2 hrs.</td>
<td>60.15</td>
<td>90/60</td>
<td>+</td>
<td>Drowsy, restless, irritable.</td>
<td>Tense dura</td>
<td>90–120 cc. slightly bloody fluid</td>
<td>Normal skull</td>
<td>“English” decompression. Good recovery.</td>
<td></td>
</tr>
<tr>
<td>McConnell 1941</td>
<td>48</td>
<td>M</td>
<td>10 days</td>
<td>140.6</td>
<td>90/78</td>
<td>+ Rt. dilated</td>
<td>Semi-comatose, bilateral grasp reflex.</td>
<td>Tense dura</td>
<td>3 cc. yellowish fluid, protein 40 mgm. %</td>
<td>1 oz. on rt., less on lt., dark yellow fluid, protein 190 mgm. %</td>
<td></td>
<td>Bilateral trephines. Expired.</td>
</tr>
<tr>
<td>Group 2</td>
<td>22</td>
<td>M</td>
<td>10 days</td>
<td>40–50</td>
<td>—</td>
<td>0 Rt. larger than lt.</td>
<td>Stupor followed by hyperexcitable state, dementia.</td>
<td>—</td>
<td>Considerable blood-stained fluid</td>
<td>Stellate fracture rt. parietal bone</td>
<td>Right temporal trephine. Good recovery.</td>
<td></td>
</tr>
<tr>
<td>Group 4</td>
<td>33</td>
<td>M</td>
<td>Stunned an instant</td>
<td>50.2</td>
<td>—</td>
<td>0</td>
<td>Left-sided headaches, tiredness, inability to concentrate.</td>
<td>Ventricular pressure 100 mm. Hg.</td>
<td>—</td>
<td>Free flow of clear fluid 24 hrs. after operation</td>
<td>Normal ventriculogram</td>
<td>Left temporal trephine. Good recovery.</td>
</tr>
<tr>
<td>Author</td>
<td>Age</td>
<td>Sex</td>
<td>Unconsciousness</td>
<td>P.R.</td>
<td>B.P.</td>
<td>Focal Symptoms</td>
<td>Pupils</td>
<td>Other Cerebral Symptoms</td>
<td>Increased Intracerebral Pressure</td>
<td>Spinal Fluid</td>
<td>Subdural Fluid</td>
<td>Roentgenograms</td>
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<tr>
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</tr>
<tr>
<td>Scott</td>
<td>1</td>
<td>child</td>
<td>5 days after convulsion</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
<td>Lt. dilated</td>
<td>18 cc. moderately bloody fluid</td>
<td>4 ozs. clear fluid</td>
<td>Linear fracture lt. frontal bone</td>
<td>Unilateral trephine.</td>
</tr>
<tr>
<td>Case 2</td>
<td>19</td>
<td>M</td>
<td>26 days</td>
<td>100/60</td>
<td></td>
<td>+ Equal</td>
<td>+</td>
<td>Prolonged stupor, restlessness</td>
<td>Tense dura</td>
<td>Clear fluid</td>
<td>3 ozs. clear fluid rt., 1 oz. clear fluid lt.</td>
<td>Fracture rt. orbit and lt. frontal bone</td>
</tr>
<tr>
<td>McConnell</td>
<td>1942</td>
<td>19 hrs.</td>
<td>130/0</td>
<td>110/65</td>
<td>0</td>
<td>Fingopoint</td>
<td>+</td>
<td>Semi-coma, Kernig reflex, Cheyne-Stokes respirations</td>
<td>Pressure not recorded on manometer</td>
<td>Blood-stained fluid</td>
<td>Yellowish fluid spurted rt., small amount rt.</td>
<td>Circular fracture lt. parietal and occipital bones</td>
</tr>
<tr>
<td>Case 3</td>
<td>8</td>
<td>F</td>
<td>20 hrs.</td>
<td>96, 38</td>
<td></td>
<td>+ Equal</td>
<td>+</td>
<td>Semi-coma, hemiplegia</td>
<td>Pressure not recorded on manometer</td>
<td>Blood-stained fluid</td>
<td>Yellowish fluid spurted rt., small amount rt.</td>
<td>Circular fracture lt. parietal and occipital bones</td>
</tr>
<tr>
<td>Case 4</td>
<td>52</td>
<td>M</td>
<td>7 hrs.</td>
<td>60-70</td>
<td></td>
<td>+ Rt. dilated, fixed</td>
<td>Confusion, incontinence</td>
<td>None</td>
<td>Blood-stained; pressure 100 mm. H₂O</td>
<td>Clear fluid</td>
<td>3 ozs. golden fluid rt., 500 mgm. %; 4 ozs. golden fluid lt., 500 mgm. %</td>
<td>Fracture lt. temporale bone</td>
</tr>
<tr>
<td>Case 5</td>
<td>12</td>
<td>M</td>
<td>1 hr.</td>
<td>70,—</td>
<td>102/75</td>
<td>0</td>
<td>—</td>
<td>Headache.</td>
<td>Increased 180 mm. H₂O</td>
<td>Large amount clear fluid bilaterally, protein 2000 mgm. %</td>
<td>—</td>
<td>Unilateral trephine.</td>
</tr>
<tr>
<td>Scott</td>
<td>9</td>
<td>M</td>
<td>1 hr.</td>
<td></td>
<td>10</td>
<td>+ Miotic</td>
<td>+</td>
<td>Jacksonian convulsions, stupor.</td>
<td>Tense dura, pressure 12 mm. Hg</td>
<td>2 ozs. clear fluid</td>
<td>Transverse fracture frontal bone</td>
<td>Unilateral trephine.</td>
</tr>
<tr>
<td>McConnell</td>
<td>1944</td>
<td>4½ hrs.</td>
<td>44,—</td>
<td></td>
<td>0</td>
<td>Amnesia, confused.</td>
<td>—</td>
<td>Blood-stained fluid, pressure 132 mm. H₂O, protein 50 mgm. %</td>
<td>95 cc. on rt., 300 cc. on lt., protein 180 mgm. %</td>
<td>Normal skull</td>
<td>Bilateral trephines.</td>
<td>Recovery.</td>
</tr>
<tr>
<td>Case 22</td>
<td>23</td>
<td>M</td>
<td>Hazy 3½ hrs.</td>
<td></td>
<td></td>
<td>—</td>
<td>0</td>
<td>Headache, dizziness, dullness, lack of sense of responsibility</td>
<td>—</td>
<td>Protein 20 mgm. %</td>
<td>Large amount bloody xanthochromic fluid on lt., small amount on rt.</td>
<td>Obliteration of subarachnoid patterns on both sides</td>
</tr>
<tr>
<td>Haynes</td>
<td>63</td>
<td>F</td>
<td>No history of trauma</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Hallucinations, delirium.</td>
<td>2D rt., secondary optic atrophy ft.</td>
<td>Normal pressure, normal analysis</td>
<td>Large amount xanthochromic fluid</td>
<td>No ventricular distortion. Lack of subarachnoid patterns over lt. frontal lobe.</td>
<td>Left frontal craniotomy. Partial recovery.</td>
</tr>
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<tr>
<td>Case 2</td>
<td>22</td>
<td>M</td>
<td>3 days 144, 32 160/110 0 —</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Prolonged stupor.</td>
<td>None</td>
<td>Pressure 120 mm. Hg.</td>
<td>5 ozs. clear fluid</td>
<td>None</td>
<td>Normal</td>
</tr>
<tr>
<td>Abbott, Due &amp; Nosik</td>
<td>1943</td>
<td>2 cases</td>
<td>Case 7</td>
<td>34</td>
<td>M</td>
<td>2 days 72, - 130/80 — —</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Headache, vertigo, loss of memory.</td>
<td>Yes</td>
<td>Pressure 200 mm. Hg. protein 30 mgm. -</td>
</tr>
<tr>
<td>Abbott, Due &amp; Nosik</td>
<td>1943</td>
<td>2 cases</td>
<td>Case 7</td>
<td>38</td>
<td>M</td>
<td>Dazed several days — — — 0 —</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Septic, ataxia, impaired hearing, irritability, inability to concentrate.</td>
<td>—</td>
<td>None</td>
</tr>
<tr>
<td>Wyess</td>
<td>45</td>
<td>M</td>
<td>1½ hrs. 60–80 120/80 + Equal</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Headache, hemiparesis, irritability.</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>3 ozs. xanthochromic fluid on rt., 3 oz. on lt. Protein 4000 mgm. %</td>
</tr>
<tr>
<td>Wyess</td>
<td>1944</td>
<td>2 cases</td>
<td>Case 1</td>
<td>29</td>
<td>M</td>
<td>Duration? 50, 16 100/60 0</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Apathetic, headache, drowsiness, dizziness.</td>
<td>2D on lt.</td>
<td>2D on rt.</td>
</tr>
<tr>
<td>Wyess</td>
<td>1944</td>
<td>2 cases</td>
<td>Case 1</td>
<td>28</td>
<td>F</td>
<td>Momentary 80, 20 — — —</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Restlessness, mania, profane.</td>
<td>Normal fundi</td>
<td>None</td>
</tr>
<tr>
<td>Wyess</td>
<td>1944</td>
<td>2 cases</td>
<td>Case 1</td>
<td>28</td>
<td>F</td>
<td>Duration? 75, 18 100/60 — —</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Headache, nervousness.</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Wyess</td>
<td>1944</td>
<td>2 cases</td>
<td>Case 1</td>
<td>28</td>
<td>F</td>
<td>About 1 hr. 80, 20 100/60 + Equal</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Aphasia, hemiplegia, intermittent stupor, restlessness.</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Wyess</td>
<td>1944</td>
<td>2 cases</td>
<td>Case 1</td>
<td>43</td>
<td>M</td>
<td>1 hr. 80, 20 100/60 + Equal</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Headache, pain over rt. eye, irritability, nervousness.</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Wyess</td>
<td>1944</td>
<td>2 cases</td>
<td>Case 1</td>
<td>42</td>
<td>M</td>
<td>? — — +</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Drowsiness, hemiparesis, restlessness.</td>
<td>—</td>
<td>None</td>
</tr>
</tbody>
</table>
Oct. 11. Encephalogram (Fig. 1). Obliteration of the fluid pathways over the convex surfaces of both hemispheres. Normal filling of ventricular system without any displacement. Headaches appeared to be aggravated by encephalography. Normal spinal fluid analysis.

Oct. 17. Bilateral exploratory trephines—local anesthesia. Two trephines made over pre-motor areas. On opening dura on the right, there was an immediate gush and spurt of slightly yellowish fluid. When the dural incision was enlarged, at least 3 to 4 ozs. of subdural fluid were evacuated. On the left side a similar pathology was seen except that the subdural fluid was less in amount. At completion of procedure the brain was pulsating and had fallen away from the undersurface of the dura so that two rubber tube drains could be easily introduced into the subdural space.


Oct. 29. Patient discharged symptom-free.

Comment. Of striking interest is the fact that the ventricles, on encephalography, showed no displacement in spite of the existing left monoparesis. Obliteration of the subarachnoid patterns was helpful in the diagnosis. The headaches appeared immediately following the injury while the monoparesis was delayed in onset, suggesting a possible delayed occurrence of the subdural hygroma.


In severe automobile accident in August 1944. Not unconscious. Following this he began having "dizzy spells" which he said were like a "hangover." The spells subsided but returned two weeks following the injury. The attacks were accompanied by malaise and bilateral supra-orbital headache. Intermittent diplopia started two months later.

On admission: Temperature 98.5°F.; pulse 48; respirations 16. No localizing signs. Weakness of the left sixth nerve. Bilateral papilledema of 2 D. Roentgenograms of the chest and skull were normal except for an airless right maxillary antrum. Blood counts, urinalysis, sedimentation rate and serology were normal.

Oct. 17. Bilateral exploratory trephines—local anesthesia. Because of the papilledema and absence of localizing signs with previous history of trauma, it was decided to make bilateral exploratory burr openings over the premotor areas. If the pathology could not be discovered by this procedure, it was decided to tap the ventricle from this position and proceed with ventriculography. Accordingly, the burr openings were made over the premotor areas. The dura was tense but not discolored. On nicking the dura, clear subdural fluid spurted out. Approximately 4 ozs. of clear subdural fluid were obtained from each side. An attempt was made to tap the right frontal horn of the lateral ventricle. The ventricle was collapsed and only 5 cc. of fluid could be obtained. A small amount of air was introduced and the cannula withdrawn. Two rubber tube drains were placed to the subdural space. The ventriculogram was indeterminate but some air was seen between the hemispheres, indicating a normal position of the midline structures.

Oct. 18. Alert and conscious. Free of all headache. The pulse had increased from 48 to 80 and the respirations from 10 to 22. The dressing was quite wet on both sides, indicating profuse subdural drainage.

Oct. 23. Symptom-free except for mild diplopia. Papilledema had receded to 1 D. in each eye.

Oct. 29. Discharged symptom-free. Mere blurring of nasal disc margins. Diplopia had subsided. He was seen at two-week intervals for five months. The optic discs were normal. He was back at work as an electric welder and symptom-free.

Comment. Here was an interesting problem of papilledema without localizing signs appearing after a head trauma which was unattended by loss
of consciousness. The small collapsed ventricles should caution one against ventricular puncture in these cases.

Case 3. T. S., age 13, admitted Nov. 9, 1944.

Patient was struck by an automobile and rendered unconscious momentarily. While in the receiving ward he was uncontrollable, restless, and resisted all attempts to aid him. A fractured right radius was also present. There were no localizing neurological signs at this time except for an equivocal Babinski on the right.

On admission: Temperature 96°F.; pulse 80; respirations 20. Spinal fluid pressure 6 mm. of Hg.; fluid blood-tinged.

The outstanding feature was the complete change of personality. He was restless, profane, insulting and unkind. He had a particular dislike for doctors and nurses. The mental reaction, at times, was almost that of a manic psychosis. His cries would awaken the entire ward. For the first three days he slept almost continuously, but could be aroused readily. His mother stated that prior to the accident he was a most model and polite gentleman.

On the third day he was re-examined for any focal neurological signs. The subgaleal hematoma was diminished in size but still fluctuant. The pupils were miotic but equal, responding sluggishly to light. There was mild impairment of facial movements on the right which may have been due to the contusion over the right cheek. The extremities were moved freely. The deep tendon reflexes were active and equal. Babinski and Hoffmann signs could not be elicited.

Nov. 15. Spinal puncture: pressure 6 mm. of Hg.; 20 cc. of clear fluid slowly removed. X-ray of skull: stellate fracture in the right temporal region involving greater wing of sphenoid bone.

There was a considerable depression of fragments. An additional linear fracture was seen in the right parietal bone with some separation of the parieto-temporal suture.


Nov. 17. Bilateral exploratory trephines. The incision on the right was made over the temporal area just anterior to and above the ear. On separating the peristeum from the bone a large gush of chocolate-colored fluid, approximately 3 ozs., escaped from the subgaleal collection. The temporal bone was fractured in several places and there was a marked indentation in the sphenoid ridge with depression of the temporal lobe. The fragments were elevated and removed piece-meal by rongeur. A small epidural collection of clotted blood was removed by suction. A large collection of clotted blood was seen beneath the temporal muscle dissecting the muscle free from the peristeum. This was likewise removed by suction. One large, loose fragment of bone, which extended to the base of the skull, was not removed for fear of serious bleeding.

The dura was tight and not discolored. A moderate amount of clear yellow subdural fluid escaped on incising the dura. The drainage increased with manipulation of the head and retraction of the brain by means of a brain spoon. The underlying cerebrum appeared normal. A drain was placed through the temporal muscle and dura, to the subdural space.

A single burr opening on the left showed a tight dura. There was likewise a moderate amount of subdural fluid present but on this side it was clear and colorless. One tube drain was placed in the subdural space.

Nov. 18. There was noted a marked and sudden change in personality. He was quiet, took his medication, and refused to swear. He permitted examination. He ate solid food for the first time during his illness. There was an amnesia for his past episodes of profanity and misbehavior. The head dressing was quite wet from subdural fluid drainage.

Nov. 19. He permitted a change of his dressing and removal of the subdural drains.


Nov. 23. Discharged symptom-free except for moderate emotional instability and increased psychomotor activity.

When seen 6 weeks later he was nearly normal except that he still showed an increased psychomotor activity and a tendency to be quite talkative.
Comment. The marked improvement in personality following drainage of the subdural fluid was quite dramatic. Psychotic episodes are not uncommon with subdural hygromas. McConnell cites a patient who required commitment but who fortunately was trephined in time. The result was excellent.


Eight years previously she was in an automobile accident. She was rendered unconscious and was hospitalized for an indeterminate period. Details of the injury could not be recalled. Shortly following the accident she complained of severe pain over the vertex of the skull. She became subject to intermittent headaches which started in both temporal areas, finally becoming generalized. The headaches were aggravated by noises and excitement. The neurological examination was entirely negative. An encephalogram, performed on a previous admission, revealed a normal ventricular system but a failure to completely visualize the subarachnoid patterns.

Dec. 4. Bilateral exploratory trephines—avertin-local anesthesia. The dura was full but not tense. On opening the dura a large amount of clear fluid was seen to escape. By rotating the head and depressing the cortex with a brain spoon, approximately 3 to 4 ozs. of clear fluid were obtained from each side. On the right a cortical vein was inadvertently opened and profuse bleeding occurred which was controlled by cautery.

Dec. 5. Complete relief of headache. There was a moderate weakness of the left foot presumably due to the bleeding from the Rolandoic vein.


She was seen at four-week intervals for the next 8 months. She felt well but still had occasional headaches in the temporal areas.

Comment. In this case there were prolonged headaches without focal neurological signs, due to a chronic bilateral subdural hygroma. Relief was almost complete with surgical drainage of the subdural space.

Case 5. Miss P. H., age 8, admitted May 3, 1944.

Eight days prior to admission she was struck by a truck while crossing the street and immediately lost consciousness. She regained consciousness at a near-by hospital, but a right hemiplegia and motor aphasia were recorded. Spinal puncture showed frankly bloody fluid. Two days following the injury she became quite drowsy but could be aroused. Six days later spinal puncture was repeated and the cerebrospinal fluid was xanthochromic. Following this procedure she became more alert and spoke a few coherent words. Periods of alertness would alternate with periods of stupor. Seven days after the injury the respirations became irregular and the pulse rate slowed to 52. On the next day she was transferred to the neurosurgical service at Temple University Hospital.

Examination on admission: Temperature 100°F.; pulse 60; respirations 16. The blood pressure was 104/66. She was drowsy and yet quite restless. A confusion was noted over the left eye and there was a mild degree of ptosis on this side. Her speech was limited to whining and crying. Occasionally a word or two could be spoken. The pupils were equal at 4 mm. and the response to light was prompt. There was a central type of facial weakness on the right. There was marked weakness of the right arm but the lower extremity on this side showed slight paresis. Bilateral Babinski responses could be easily evoked. A hematoma was present over the right clavicle and was due to an underlying fracture. The spinal fluid pressure was 10 mm. of Hg.; 16 cc. of straw-colored fluid were slowly withdrawn. There was a linear fracture of the left parietal bone.

May 5. Bilateral exploratory trephines—avertin-local anesthesia. On the left side the dura was tight but not markedly discolored. On opening the dura a large amount of xanthochromic fluid squirted out under pressure. Approximately 3 ozs. of fluid were removed. A single tube drain was placed to the subdural space. On the right side a similar pathology was found but
SUBDURAL HYGROMA

the amount of subdural fluid was less, approximately 1 oz. A similar drain was placed to the subdural space.

May 6. She was lucid and alert. She said "yes" and "no." The dressing was profusely wet from the drains in the subdural space.

May 8. Restlessness markedly subsided. Speech was still explosive but the words could be said with less difficulty.

May 11. Hemiparesis diminished markedly. She was able to form short sentences.

May 12. Able to count and form sentences.

May 15. Marked improvement in the aphasia. Now able to carry on a limited conversation. Hemiparesis had completely cleared but there was ataxia of both upper limbs, particularly on the right.

May 18. Discharged. She was seen at two- and three-week intervals for the following eight months. She is totally well and attending school. Her grades are above the average. She plays the piano well.

Comment. Prolonged stupor, and restlessness, with focal neurological signs (aphasia and hemiparesis), were the outstanding features due to a bilateral subdural hygroma. Complete relief of symptoms followed drainage of the subdural space.


A month before admission he was struck over the right fronto-temporal area with a heavy piece of lumber which fell from a height of 25 feet. Duration of unconsciousness was one hour. He was hospitalized at another institution and remained there but four days. During his hospital stay he had right-sided headaches and pain down the left arm. His headaches persisted and became increasingly severe. There were likewise irritability, nervousness, tremor and memory impairment. A semi-circular scar was present on the right frontal area. This was extremely sensitive to touch. The left eye was artificial. There were no localizing neurological findings.

Jan. 31. Encephalogram (Fig. 2). The initial spinal fluid pressure was 10 mm. of Hg.; protein was 35 mgm. per cent. A subdural air shadow was seen over the right cerebral hemisphere. There was a failure of visualization of the subarachnoid patterns over the left cerebral hemisphere. No shift of midline structures. The right lateral ventricle did not fill, but the left lateral ventricle had a normal appearance.

Feb. 5. Bilateral exploratory trephines—averin-local anesthesia. The dura was not tense or discolored. When the dura was opened on both sides, a stream of clear xanthochromic fluid in copious amounts (3 to 4 ozs.) welled out of the wound. The brain fell away from the dura and was seen to freely pulsate. Rubber tube drains were placed in the subdural space and fluid was seen to pulsate in the ends of the rubber tubes at the completion of the procedure.

There was an immediate marked amelioration of all symptoms. The headache, tremor and nervousness had completely abated. Because of profuse drainage, the drains were allowed to remain for 3 days. The patient had been given sulfadiazine prophylactically (1 gm. every 4 hours) two days preoperatively and 5 days postoperatively. The subdural fluid contained 117 mgm. per cent protein.

Comment. The encephalogram indicates a drainage of the subdural space on the right side and yet the spinal-fluid protein was but 35 mgm. per cent, while the subdural fluid obtained at operation was 117 mgm. per cent. The case is of interest in that it demonstrates how typical post-concussional symptoms may be relieved by drainage of the subdural space.

Case 7. Mr. H. T., age 42, admitted Oct. 10, 1943.

He was seen to fall from the top of a machine into a pit below, a distance of some ten feet. He was apparently not unconscious and was able to climb a ladder out of the pit without sup-
Vomiting immediately ensued. He was immediately taken to the emergency admission ward at Temple University Hospital. He was quite euphoric and insisted that he was well. At that time the pupils were equal and responded promptly to light. There were four lacerations about the right ear and occipital area. A bloody cerebrospinal fluid leak from the right ear was noted. Spinal puncture: pressure 10 mm. of Hg.; fluid bloody. Within half an hour the right pupil dilated and the left side became paretic. Drowsiness followed. An emergency right temporal exploratory trephine was carried out. A transverse fracture line was noted running almost parallel to the squamous suture of the temporal bone. The trephine opening was made below the fracture line. When the bone was removed a small epidural hemorrhage was present. The dura was blue and tense. On opening the dura chocolate-colored clots of blood escaped from the wound. These were evacuated by irrigation and suction with a soft rubber catheter. The brain was tight and herniated into the wound, indicating an already existing intrinsic cerebral edema. Two drains were placed in the subdural space. At the completion of the procedure the pupils were nearly equal and responded promptly to light. During the next three days he still had an incomplete left hemiplegia. Spinal puncture was repeated. The pressure was low and 5 cc. of xanthochromic fluid were removed. Because drowsiness and restlessness had increased, bilateral exploratory trephine openings were made in each parietal area. The dura was slightly discolored. On opening the dura, a large amount of subdural xanthochromic fluid escaped. There was more subdural fluid on the right side than on the left. Drains were placed in the subdural space. Drowsiness and hemiplegia per-

![Fig. 2. Case 6. Encephalogram. Note the obliteration of the subarachnoid spaces on the right and the presence of the subdural air shadow on the left. The left ventricle failed to fill but the right showed no displacement. A bilateral subdural hygroma was disclosed at operation.](image)
sisted for three days. The right pupil was larger than the left and did not respond to light. Gradually, the hemiplegia cleared, restlessness subsided and he made a nearly complete recovery.

Three months later he was back at his former occupation. One year later his only difficulty was a slight impairment in concentration and a tendency to tire at the end of the day.

Comment. Persistent drowsiness and focal neurological signs subsided after bilateral drainage of the subdural space. It is notable that the subdural hygroma occurred some time after the first operation and was responsible for the stormy convalescence.

INCIDENCE OF SUBDURAL HYGROMAS

Regarding the frequency of occurrence of this pathological entity, there are few statistics. The best are those of McConnell, who reported subdural effusions in 32 out of 63 patients operated upon for head trauma. Certainly the incidence is higher than has been heretofore suspected.

The frequency is higher in males than in females, most likely due to the severity of occupation and engagement in the theaters of war (blast effusions). In reviewing the cases reported in the literature, there were 26 males and 5 females. The ages varied from 1 to 69 years. The greatest incidence appeared in the second decade of life (9 cases).

The total number of cases is tabulated below:

<table>
<thead>
<tr>
<th>Author</th>
<th>Number of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mayo, 1894</td>
<td>1</td>
</tr>
<tr>
<td>Payr, 1916</td>
<td>4</td>
</tr>
<tr>
<td>Cohen, 1927</td>
<td>1</td>
</tr>
<tr>
<td>Love, 1937</td>
<td>1</td>
</tr>
<tr>
<td>Walsh and Shelden, 1937</td>
<td>1</td>
</tr>
<tr>
<td>Da Costa and Adson, 1941</td>
<td>1</td>
</tr>
<tr>
<td>McConnell, 1941</td>
<td>32</td>
</tr>
<tr>
<td>Siros, 1942</td>
<td>1</td>
</tr>
<tr>
<td>Scott, 1942</td>
<td>3</td>
</tr>
<tr>
<td>McConnell, 1942</td>
<td>4</td>
</tr>
<tr>
<td>Scott, 1943</td>
<td>1</td>
</tr>
<tr>
<td>Abbott, Due and Nosik, 1943*</td>
<td>7</td>
</tr>
<tr>
<td>Abbott, Due and Nosik, 1943*</td>
<td>27 (37 less 10 of 1st report)</td>
</tr>
<tr>
<td>McConnell, 1944</td>
<td>5</td>
</tr>
<tr>
<td>Haynes, 1944</td>
<td>3</td>
</tr>
<tr>
<td>Wycis, 1945</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>99 cases</td>
</tr>
</tbody>
</table>

* The subdural effusions and hematomas are not separated in the reports of Abbott, Due and Nosik, hence the total number of subdural hygromas is less than the total number listed above.

SYMPTOMATOLOGY

A history of injury (direct or blast) is usually available. There follows a period of unconsciousness which may vary from moments to weeks. The cases associated with blast concussion may show only an initial state of “daze” which may progress to confusion.
Headache, dizziness, nervousness, irritability, restlessness, inability to concentrate, loss of memory, confusion, focal neurological signs (aphasia, hemiparesis, hemiplegia, dilated pupil), drowsiness, semi-coma, and prolonged stupor are frequent post-traumatic symptoms.

In reviewing the detailed case reports of the literature, the following statistics were available when these symptoms were reported. The total number of cases reviewed was 33.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Number of Times Recorded</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>12</td>
</tr>
<tr>
<td>Dizziness</td>
<td>5</td>
</tr>
<tr>
<td>Nervousness and irritability</td>
<td>7</td>
</tr>
<tr>
<td>Loss of memory and confusion</td>
<td>6</td>
</tr>
<tr>
<td>Restlessness</td>
<td>4</td>
</tr>
<tr>
<td>Aphasia</td>
<td>5</td>
</tr>
<tr>
<td>Hemiparesis or hemiplegia</td>
<td>6</td>
</tr>
<tr>
<td>Drowsiness</td>
<td>4</td>
</tr>
<tr>
<td>Semi-coma</td>
<td>3</td>
</tr>
<tr>
<td>Stupor</td>
<td>7</td>
</tr>
</tbody>
</table>

Headache appears to be the most frequent symptom. It may commence immediately at the time of injury or may make its appearance months later. The headache is usually generalized but may be unilateral and more pronounced on the side of the greatest effusion. Altering head posture may aggravate the headache, particularly when the patient bends forward. The exact mechanism of subdural headache is unknown; however, it is believed to be due to a dural stretch by the entrapped fluid. The marked and dramatic relief following drainage of the subdural space would appear to substantiate this theory. The headache may be temporarily relieved by repeated spinal drainages; however, it is occasionally made worse due to the inability to empty the subdural space with further retraction of the brain from the dura.

Dizziness is likewise a frequent and often persistent symptom following cerebral concussion. It may be the sole symptom for years; however, it is usually associated with the above-named symptoms. The dizziness is usually of an indefinite nature; however, in some cases, a true turning sensation may be described by the patient. In none of the author’s cases was a true nystagmus or a true directional vertigo recorded. The causes for post-traumatic vertigo are not definitely known. Spiegel believes that this type of vertigo is the result of vasomotor disturbances of the brain as well as the labyrinth. The sudden increase of intracranial pressure at the instant of concussion is transmitted to the labyrinth as well as other parts of the skull and this impairs vestibular function. This theory is substantiated by the observations of Brunner. After concussing the skulls of guinea pigs he found circulatory disturbances, diapedesis, and transudation of fluid into the endo- and peri-lymphatic spaces of the inner ear (otitis interna vasomotorica). According to McConnell this symptom is usually the result of cerebral confusion and remains unaltered despite surgical drainage of the subdural hygroma. The
statistical studies of Friedman, Brenner and Denny-Brown attempted to show that there was remarkably little evidence that post-traumatic dizziness was related to vestibular damage. It is often difficult to decide whether dizziness is psychogenic or organic. In such instances it is frequently necessary to determine the presence of a lesion of the labyrinth or the vestibular pathways. Brunner found in uncomplicated post-concussional vertigo lack of marked cochlear disturbance with a normal or increased labyrinthine excitability. Baumoe1 and Marks were able to show a difference in tonus between the two labyrinths, there being a directional preponderance to one side.

Nervousness and irritability were recorded in seven instances of the 33 cases reviewed. Restlessness was recorded in but four instances. However, the author feels that this symptom has a greater incidence than would appear from statistical analysis.

Loss of memory, confusion and amnesia are not uncommon. In an interesting paper by McConnell, six cases of prolonged post-traumatic amnesia were found to be due to subdural effusion in five instances. McConnell suggested the following sequence of events: “Concussion caused a lengthy period of anoxemia; during this period a subdural effusion developed, interfered further with cerebral function and so prolonged the existing amnesia. The duration of amnesia may be regarded as a measure of the sums of the effects of the initial injury plus subsequent interference with cerebral function.” Following blast concussion, Abbott, Due and Nosik reported a high incidence of confusion, memory loss and lowered responses to the Hartford Shipley Retreat and Rorschach tests.

Occasionally the patient becomes delirious, disoriented and even manic so that commitment to an asylum has been necessary.

Focal neurological signs are frequently present. These are usually a hemiparesis or hemiplegia associated with or without aphasia. In the blast concussion group and the amnesic group reported by McConnell, focal neurological signs were sparse.

Pupillary anomalies are important signs. The presence of a dilated pupil on the side of the largest effusion is a good operative indicator. A dilated pupil was recorded in but 6 instances of the 33 cases reviewed. Bilateral miosis was recorded in 3 instances.

Occasionally, one may see the classical triad of headache, vomiting and papilledema, thus mimicking a brain tumor (see Case 2).

**DIAGNOSIS**

As a rule, in the acute cases, the differentiation from a subdural hematoma is difficult and can be settled only by trephining the skull. The persistence of headache, vertigo, irritability, memory impairment and mental confusion after the acute stage of a cerebral trauma has passed should suggest the possibility of a subdural hygroma. The appearance of headache some weeks or months later is not uncommon. Prolonged stupor was par-
ticularly emphasized in the reports of Scott. Post-traumatic amnesia as a result of subdural effusion has been stressed by McConnell. The diagnostic characteristics of subdural effusions following blast concussion are mental confusion, absence of localizing signs, and a diminished response to psychometric tests.

In the acute cases, the subdural effusion may act as an expanding lesion, thus mimicking a subdural hematoma. In the chronic cases, the subdural effusion acts as a passive space-occupying lesion, delaying recovery. The persistence of symptoms or delay of recovery following a head injury should thus arouse the suspicion of the presence of a subdural hygroma.

**SPINAL PUNCTURE**

Spinal puncture is not diagnostic and cannot differentiate this lesion from that of subdural hematoma. The pressure may be subnormal, normal or increased. The fluid may be clear, xanthochromic or blood-tinged.

In the cases analyzed in Table I, the recorded pressures varied from 6 to 15 mm. of Hg. while the protein content varied from 30 to 180 mgm. per cent.

**SUBDURAL PUNCTURE**

In this report the group of subdural hygromas occurring in infants is not included. Ingraham and Matson made an excellent study of subdural hematomas and hygromas in infancy and established the diagnostic value of subdural puncture. The technique of the method is fully described in their paper.

**ROENTGENOGRAPHY**

A fracture may or may not be present and is not necessarily correlated with the severity or size of the subdural effusion. In the cases recorded in Table I, a fracture was present in 15 instances. No predilection for any specific site was conducive to the production of the hygroma. A depressed fracture was noted in two cases.

Pneumoencephalography may be helpful. The most characteristic finding is the absence of subarachnoid filling over one or both hemispheres (Figs. 1 and 2). This blotting out of the subarachnoid patterns was stressed by Abbott, Due and Nosik in the blast concussion group. In Table I this feature was noted in seven cases.

The presence of subdural air shadows on one or both sides with a fluid level raises a controversial point. Some feel that these shadows are due to a technical error in performance of the encephalogram and merely represent a rent in the arachnoid with passage of air into the subdural space. This probably occurs during vomiting or coughing at the time of the procedure. A normal spinal fluid protein would seem to justify this conclusion, since the emptying of a true subdural hygroma should cause an elevation in the average sample of cerebrospinal fluid, particularly if the subdural collection be xanthochromic or bloodstained.
SUBDURAL HYGROMA

Others feel that the presence of subdural air shadows represents a true emptying of a subdural hygroma through a rent in the arachnoid that occurred formerly at the moment of injury or at the time of encephalography. These cases should show an improvement following such drainage of the subdural space.

It is the author’s practice to verify the presence or absence of a subdural hygroma in these cases by trephining, if symptoms persist following encephalographic drainage.

Ventricular shadows may be small and show little or no displacement in bilateral subdural hygromas. Under such circumstances, ventricular puncture becomes difficult and not without danger, particularly if one attempts to puncture in the premotor area. In unilateral subdural hygromas the roof of the ventricle is depressed on the affected side or the entire body may show displacement away from the side of the lesion. In other cases, the ventricle may appear displaced toward the side of the lesion. Again, in case of doubt, bilateral trephine openings should be made.

SURGICAL TREATMENT AND RESULT

It is now generally agreed that the operative treatment is the simple bilateral trephining of the skull. The opening may be conveniently placed over the frontal, temporal or parietal areas. Because of gravitation of the fluid burr openings placed low in the temporal areas, with patient in the sitting position, would be ideal for maximal drainage.

The dura is not discolored unless an underlying hematoma is present. The dura is frequently found to be tense and when it is opened, a gush of clear, xanthochromic or blood-tinged fluid escapes. The brain attempts to fill the opening but by depression of the cortex with a brain spoon and rotating the head, a considerable quantity of fluid can be obtained. After repeating this procedure several times, the brain falls away from the dura and can be seen to pulsate. Drains (cigarette or rubber tube) are placed beneath the dura and allowed to remain for 24 to 48 hours. Longer periods of drainage offer potentialities of infection and hence should be avoided. Once the drains are in place, fluids are forced by mouth and vein to encourage the cerebrum to regain its former confines.

Other operative procedures have been tried, some of which were puncture of the corpus callosum (Payr, 1916), subtemporal decompression (Naffziger, 1924, Cohen, 1927), “English” decompression (Walsh and Shelden, 1937) and even craniotomy (Haynes, 1944).

The statistics of Table I show that clear subdural fluid was recorded ten times, xanthochromic fluid fourteen times, and blood-tinged fluid but four times. The protein content ranged from 100 to 5000 mgm. per cent. In the group reported by Abbot, Due and Nosik, the protein content averaged 300 mgm. per cent. The amount of fluid obtained may vary from a few cc. to a liter. Cohen recorded 2500 cc. of subdural fluid which was obtained with nine different drainages.
In the review of the cases (Table I) bilateral trephines were recorded in 18 cases while unilateral trephine was recorded in 12.

Three patients died. Twenty-six patients made complete recoveries while three made partial recoveries.

SUMMARY

1. The literature on subdural hygromas has been reviewed.
2. Statistical analysis was conducted in thirty-three cases which were reported in detail.
3. Seven additional cases were contributed to the literature.
4. Symptomatology, diagnosis and roentgenography were discussed.
5. The incidence of subdural hygroma is certainly greater than is recognized and the author urges bilateral trephination in cases which present symptoms as outlined in this report.
6. Bilateral trephination of the skull is the surgical procedure of choice.

REFERENCES

9. Fay, T. Personal communication.