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One Hundred Cases Of Subdural Hematoma From 1930 To 1955 At The Henry Ford Hospital

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A chronic subdural hematoma is more likely to occur following a minor head trauma than an acute severe head injury. In 1914 Trotter stated that subdural hematoma always had a traumatic origin and emphasized that the head injury was often minor. In the same year he also published his excellent paper in which physiologic peculiarities of the cerebral circulation in subdural hematoma were studied in four different stages. The first, or stage of compensation, is present in the earliest period of the development of this lesion when a certain amount of limited encroachment upon the intracranial cavity can occur without interference with the cerebral circulation. He stated that this was possible because the cerebral spinal fluid found in the area of the hemorrhage could be displaced without producing any effect in function, and because the veins in the same region could undergo a certain amount of compression without restriction of the venous return sufficient to cause congestion.

With the increase in size of the hematoma the second, or stage of venous obstruction, is soon reached. Compression of the veins restricts the venous circulation, causing the veins to collapse. A state of cyanosis in the brain results. The venous congestion produces a characteristic alteration of cerebral function, namely, an increased excitability of the cerebral tissue. This is a local change.

If the hematoma is not evacuated the third, or stage of capillary anemia, follows. This is due to increased pressure in which the remaining blood in the capillaries is squeezed out of the affected part of the brain, which assumes the dead white color of capillary anemia. This is responsible for the subsequent fourth stage of paralysis of function. Thus, paralytic symptoms after a head injury are not always due to actual destruction of the brain substance, and may, in the majority of cases, indicate a compression which has advanced to the stage of capillary anemia.

The great majority of subdural hematomas are located in the lateral aspect of the fronto-parietal region. The size of the hematoma varies from a few centimeters in diameter to cover the complete hemisphere. Its thickness also varies from a few millimeters to four or five centimeters.

Many authors believe that chronic subdural hematomas gradually increase in size. Peet and Kahn stated that repeated bleeding from the highly vascularized outer membrane with rapid increase in the size of the hematoma would best explain the frequent exacerbations and remissions in the symptoms.

Gardner believes that the liquid contents of the hematoma is augmented with cerebrospinal fluid drawn into the cyst by osmotic pressure.

Chronic subdural hematoma is now recognized much more frequently than twenty-five years ago. In 1925 Putman and Cushing found only eleven cases in the records at Peter Bent Brigham Hospital from the previous nine years.

In 1935 Frasier reported six cases from 1932 to 1933 at Union Hospital, Philadelphia. Horrax and Poppen reported eighteen cases in 1937 in a period of two years. Furlow reported sixteen cases in 1936. Kunkel and Dandy reported forty-eight cases in 1939 on the neurosurgical service of the Johns Hopkins Hospital between

*Division of Neurological Surgery.
Subdural Hematoma

1914 and 1935. With the introduction of ventriculography in 1918 this condition began to be recognized more frequently and many times was found during the placement of bur holes for ventriculographic purposes.

In our series there has been a definite increase through the years (Fig. 1). From 1930 to 1934 eight cases were seen; from 1935 to 1939 the number rose to eleven; from 1940 to 1944 it showed a marked increase and twenty-three cases were seen. For the period 1945 to 1949 the number of cases dropped to sixteen, but, finally, for the last period 1950 to 1955 forty-two cases were seen in this hospital. This apparently marked increase in frequency of occurrence may be due to the greater number of head injuries, but a considerable part of the increase is due to our present knowledge of its etiology and symptoms.

TRAUMA

Since the description in 1857 by Virchow9 of the condition which he names "pachymeningitis hemorrhagica interna" there has been considerable controversy as to its etiology and pathogenesis. In 1914 Trotter10 stated that in the formation of subdural hematoma trauma was always present. In 1872 Sperling11 injected blood into the subdural space of dogs and found that organization began within a week and that the membrane formation was found after three weeks. He carried out similar experiments using defibrinated blood and did not find any membrane, which led him to the conclusion that organization was the result of the fibrin irritating the dura. Microscopically the findings corresponded in all points with those found in pachymeningitis hemorrhagica interna as originally described by Virchow9.

Similar experiments were made by Spiller12 in 1899. He nicked the superior longitudinal sinus in cats and dogs sufficiently to produce subdural hemorrhage, and by sacrificing the animals at intervals from six hours to five weeks, was able to study the various steps in the formation of chronic subdural hematoma. His conclusions
Francisco Gomez

were that evidence of new membrane formation was quite distinct after five days, that organization begins near the dura, that after five weeks the membrane is distinctly formed and that subdural hemorrhage may be one of the causes of the formation of a subdural membrane.

Trauma to the head is now accepted as the major etiological factor. Other causes are blood dyscrasias as described by Jones and Knighton\(^{13}\), metastatic carcinoma, and ruptured aneurysms. As a rule the injury is comparatively mild, in fact, in many cases so slight as to have been entirely forgotten until careful questioning recalled the occurrence. History of trauma is a very important factor in the diagnosis of subdural hematoma, as stated by other authors. However, in our series it was found that in twenty-nine patients it was not possible to elicit a history of head injury, and I certainly agree with Trotter\(^1\) that the role played by trauma in the formation of chronic subdural hematoma is often discounted because of its minor nature. This is especially true in the older patients, who have some degree of cortical atrophy, in whom the cortical veins draining into the sagittal sinus are under tension and any minor trauma in the antero-posterior or postero-anterior direction can very easily tear these vessels at their dural attachment. Bleeding then takes place into the subdural space with subsequent development of a hematoma. Grant\(^{14}\) in 1935 reported a case caused by the comparatively insignificant force of the breakers encountered in surf bathing.

In the one hundred cases reviewed at this hospital from the period 1930-1955 (Table I) seventy-one cases had a history of trauma. Of these, fifteen (21.1%) were children and fifty-six (78.9%) were adults. Of the fifty-six adults, the younger and older groups were studied. Above the age of fifty there were twenty-six cases (46.4%) and below the age of fifty, thirty cases (53.6%). The youngest of this group with head trauma was two weeks old, and the oldest seventy-six years, with an average age of 37.4 years.

<table>
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<th>History of Trauma</th>
<th>71 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults</td>
<td>56 (78.9%)</td>
</tr>
<tr>
<td>Children</td>
<td>15 (21.1%)</td>
</tr>
<tr>
<td>Adults Above age 50</td>
<td>26 cases (46.4%)</td>
</tr>
<tr>
<td>Adults Below age 50</td>
<td>30 cases (53.6%)</td>
</tr>
<tr>
<td>No History of Trauma</td>
<td>29 cases</td>
</tr>
<tr>
<td>Adults</td>
<td>26 (89.6%)</td>
</tr>
<tr>
<td>Children</td>
<td>3 (10.4%)</td>
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<tr>
<td>Adults Above age 50</td>
<td>19 cases (73.0%)</td>
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<tr>
<td>Adults Below age 50</td>
<td>7 cases (27.0%)</td>
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<tr>
<td>The Youngest of All</td>
<td>2 weeks</td>
</tr>
<tr>
<td>The Oldest of All</td>
<td>76 years</td>
</tr>
<tr>
<td>Average Age</td>
<td>42.4 years</td>
</tr>
</tbody>
</table>

Table I. Trauma in Subdural Hematoma
**Subdural Hematoma**

In the twenty-nine cases with no history of head injury, twenty-six were adults (89.6%) and three children (10.4%). The adults were again subdivided, and above the age of fifty years, nineteen cases (73%) were seen, and below the age of fifty, seven cases (27%). Here we emphasize that in older people who develop subdural hematoma a history of head trauma is not frequently obtained or is so trivial that the patient forgets to mention it. The youngest adult was sixteen years old, and the oldest eighty-two years, with an average age of 56.6 years. The over-all average age was 42.4 years.

**RACE AND SEX**

In our series ninety-three patients were white and seven colored. Of the ninety-three white patients, seventy-three (78.4%) were male and twenty (21.5%) female. The number of females with subdural hematoma in this hospital is higher compared with other reports such as that of Kunkel and Dandy in which five patients of forty-eight were female (10.4%).

**CLASSIFICATION**

Our subdural hematomas were classified as acute from zero to forty-eight hours, subacute from two days to two weeks, and chronic with a history of more than two weeks since onset of symptoms or head injury. Of the one hundred cases, seventeen were acute, twenty-eight subacute, and fifty-five chronic. Bilateral subdural hematomas were seen in thirty cases.

**SYMPTOMS AND FINDINGS**

Unconsciousness after head injury was present in twenty-eight cases, but of these seventeen (60%) corresponded to acute subdural, leaving only eleven (39.3%) in the subacute and chronic classification. This again emphasizes that minor or trivial head injuries, without loss of consciousness, are most often the cause of the chronic lesions.

The period between the head injury or beginning of symptoms and the removal of the subdural mass, whether at operation or at autopsy, varied from two to ten weeks with an average of 7.7 weeks. There was a child with a history of head injury for ten years in whom a calcified subdural hematoma was found.

Headache was the most frequent symptom in the subacute and chronic cases and seventy-eight (93.9%) complained of headache. This was localized to the side of the subdural in twenty (24%) cases. Convulsions were reported in seventeen patients. Mental confusion, drowsiness and fluctuating level of consciousness was found in almost every case. Peet had stated that mental confusion, either intermittent or continuous, was one of the most important diagnostic symptoms.

Of the findings from the neurological examination inequality of the pupils was present in twenty-five cases, being dilated on the side of the lesion. Here again Peet emphasized that this is the most positive localizing sign. Dilatation of the pupil is due to the herniation of the uncus underneath the tentorium, the third nerve being then compressed in its upper portion by the posterior cerebral artery which crosses it superiorly, thus interrupting the parasympathetic fibers to the iris. Early papilledema was seen in thirteen cases, bilateral papilledema in only five, and hemiparesis in forty.

Lumbar puncture was performed in fifty-four patients, thirty-three (61%) had
Francisco Gomez

FINDINGS:

INEQUALITY OF PUPILS ............................................ 25 cases
EARLY PAPILLEDEMA .................................................... 13 cases
BILATERAL PAPILLEDEMA ............................................. 5 cases
HEMIPARESIS ............................................................. 40 cases

LUMBAR PUNCTURE .................................................. 54

Fluid pressure
- Between 200* & 350 ........................................ 19 or 35.1%
- Above 350 ............................................................. 2 or 3.8%

Fluid characteristics
- Clear ................................................................. 45 or 87.3%
- Xanthochromic ................................................. 7 or 12.7%

Proteins & Pandy
- Below 50 Mg % ................................................ 434 or 79.6%
- Between 50 & 90 .................................................. 9 or 16.6%
- Above 90 ............................................................. 2 or 3.7%

* m.m. of water.

Table II. Physical Findings in Subdural Hematoma

normal spinal fluid pressure and in nineteen (35.1%) the pressure was between 200 and 350 m.m. of water and in two the pressure was above 350. The fluid was clear in forty-seven (87.3%) cases and bloody or xanthochromic in seven cases. Proteins and Pandy were normal in forty-three (79.6%) cases. Between 50 and 90 mgm.% in nine cases, and in two cases the proteins were 139 and 190 mgm.%, respectively.

DIAGNOSTIC STUDIES

Arteriograms (Fig. 2) were carried out in ten patients. A diagnosis of subdural hematoma was made in nine cases. Electroencephalograms (Fig. 3) were taken in twenty patients; in four patients the findings were suggestive of a subdural hematoma. X-rays of the skull (Fig. 4) revealed a shift of the pineal in five cases. Pneumoencephalograms (Fig. 5) were done in five cases, being of diagnostic help in two cases. Thirty-eight cases were correctly diagnosed, or strongly suspected, prior to the operation. The remainder of sixty-two cases had a tentative diagnosis of cerebral arteriosclerosis, post-traumatic syndrome, cerebrovascular accident, extradural hematoma, or brain tumor before surgery or autopsy.
Subdural Hematoma

Fig. 2 Right Carotid arteriogram showing displacement to the left of the anterior cerebral artery and "crescent" deformity of vessels of middle cerebral area, in a large chronic subdural hematoma.

Fig. 3 There is gross asymmetry over the two hemispheres with suppression of background activity on the left, in association with irregular Delta activity seen more on the left temporal region. Right side essentially normal except for some minor disturbance in the form of subalpha activity. A large subacute subdural hematoma was drained.
Fig. 4 Towne view A, showing displacement of calcified pineal gland to the right. Lateral view B, shows downward displacement of pineal gland. A large fronto temporo parietal chronic subdural hematoma was removed.

Fig. 5 Pneumoencephalogram showing flattening of roof and inferior displacement of frontal horn of lateral ventricle, due to the presence of a large chronic fronto parietal subdural hematoma.
Subdural Hematoma

DIAGNOSTIC STUDIES:

ARTERIOGRAMS: ........................................ 10
Correctly diagnosed .................................. 9 or 90%

ELECTROENCEPHALOGRAMS: ..................... 20
Suspected diagnosis ................................ 4 or 20%

SHIFT OF PINEAL GLAND ..................... 5

PNEUMOENCEPHALOGRAMS .................. 5
Diagnosed correctly ............................... 2 or 40%

OPERATIVE MORTALITY:

ACUTE SUBDURAL .................................. 55.5%
SUBACUTE SUBDURAL ............................. 4.9%
CHRONIC SUBDURAL .............................. 16.6%

PROGNOSIS:

SURVIVAL ........................................ 68 patients
Good Results ..................................... 58 or 85.3%

Table III. Diagnosis Operative Mortality and Prognosis in Subdural Hematoma

OPERATIVE TECHNIQUE AND MORTALITY

The majority of adults had bilateral parietal and frontal bur holes. However, in seventeen cases an osteoplastic bone flap was necessary. In children a craniotomy was the rule, in order to remove the inner subdural membrane from the brain surface to facilitate re-expansion and normal development of the brain. Of the seventeen acute subdural lesions, thirteen died. Of the twenty-eight subacute subdural lesions, five died, and from the fifty-five chronic lesions fourteen died. Of the seventeen acute cases nine were taken to the operating room and of these five died. Of the twenty-eight subacute, twenty-one came to operation, and one died. Of the fifty-five chronic cases, forty-eight went to operation and of these nine (16.6%) died, which is slightly below some other hospitals in which 20% operative mortality in the chronic group was the average; however, Munro, reviewing sixty-two cases in 1934, found an operative mortality of 41.2%; however, he included both acute and chronic forms. Our over-all mortality (operative) in all forms was 19.2%.

A good prognosis depends upon an early diagnosis, before medullary or brain stem findings appear. Once this stage is reached the mortality is high. Of the sixty-eight patients that survived (Table III) fifty-eight had good results and, if adults, they were back to their usual work. If children, they had a normal somatic and mental development. The remaining ten had poor results, and patients continued complaining of headaches or had convulsive seizures. The children had slow mental development or uncontrollable seizures and had to be confined to an institution.

42
A chronic subdural hematoma consists of a large collection of bloody fluid enclosed by a definite membranous sac, the outer wall being adherent to the inner surface of the dura while the inner wall lies on the arachnoid. The gross appearance is striking. Usually diagnosis is indicated by the dark bluish color of the dura, and upon opening of the dura a tense, glistening purplish membrane from which the dura can be rather easily separated, but leaving many bleeding points. The membrane varies from one to a few millimeters of thickness, the contents varies in color from bloody to chocolate and usually contains dark, partially broken down clots. After evacuation of the fluid blood and irrigation, a thin translucent bluish-green membrane is found pressed tightly against, but not adherent to the arachnoid. After removal of the inner membrane the pia-arachnoid is seen covering a congested brain cortex (Fig. 6).

Microscopic examination of the outer membrane shows a layer of connective tissue adherent to the dura. The outer layer frequently shows areas of hyalinization while on the inner surface new fibroblasts can be seen extending into the underlying clot. This membrane is quite vascularized with new formed capillaries which in the inner surface are sometimes of unusual size. Large, irregular spaces lying in a horizontal plane, lined with a single layer of “mesothelial” cells and generally containing normal blood have been repeatedly described in the outer membrane, and they seemed to be characteristic.
Subdural Hematoma

of a chronic subdural hematoma and have not been found in hematomas situated elsewhere (Fig. 7A).

The inner membrane consists of a thin layer of connective tissue covered with a single layer of "mesothelial" cells. It is avascular and generally is stained by blood pigments (Fig. 7B). Chemical analysis shows the presence of bilirubin. This is

Figure 7 A.—Outer membrane showing the typical vascular lakes in the inner portion adjacent to the hematoma. These lakes can be seen better on right upper quadrant.

Figure 7 B.—Inner membrane showing in the lower part the smooth mesothelial cell layer which lies against the lepto-meninges and makes its separation rather easy.
Francisco Gomez

produced by the reticuloendothelial cells in the meninges. The origin of the "mesothelial layer" of the inner membrane has not been definitely proved, but has been stated that they arise from cells wandering from the dura to the under surface of the clot. The semipermeable nature of the inner membrane has been proved by dialyzing experiments. This lends weight to the theory of augmentation of the fluid volume by the addition of cerebrospinal fluid from the subarachnoid space. However, this space must be largely obliterated by the pressure of the hematoma.

It has been claimed by several writers that the so-called chronic subdural hematoma is not subdural but is actually an intradural lesion. The most recent to expound this belief are Wells and Dickson17. They offer rather convincing proof from a careful pathological study of autopsy material that the cystic hematoma lies within the meningeal layer of the dura. If this is true, it would explain the outer and inner membranes whose formation has been so obscure.

Jelsma18 in 1930 reviewed forty-two cases from the literature with a confirmed diagnosis of chronic subdural hematoma. His average age was 39.2 years which is approximately the same as our series, 42.4 years. He stated that trauma was definite in 88% and that 98% may well have been traumatic and only two gave a history of no trauma. Here I found a marked discrepancy since we had 29% with no history of head injury. I feel this is due to the better knowledge at present of this disease and also because we are aware that subdurals are also quite frequent in older people in which the diagnosis of cerebrovascular accident, cerebral arteriosclerosis, or brain tumor are usually suspected.

The lesion should be strongly suspected in younger people who have a history of mild head injury followed by headaches and later develop drowsiness, mental confusion, hyperactive reflexes and mild hemiparesis. One should not wait until the patient is semicomatose, hemiplegic, with dilated pupils, bilateral Babinski's, which means brain stem pressure. Once he reaches this stage the prognosis is poor. In older people with a trivial head injury, or none, especially if moderate cortical atrophy is suspected, subdural hematoma should always be kept in mind since there is an increase of tension on the cortical veins which drain in the longitudinal sinus, facilitating their rupture and bleeding in the subdural spaces by any minor head injury. Here again, the headaches, mental confusion, drowsiness, and a fluctuating level of consciousness are very important helps in diagnosing this disease. The treatment should always be bilateral bur holes in the posterior parietal and frontal regions in order to drain the hematoma. In some patients an osteoplastic flap may be necessary if the patient has a thick inner or outer membrane that is inhibiting the expansion of the brain or if clotted hematoma has been left. In children the treatment is somewhat different, since subdural taps are always possible to perform, and by doing this several times the subdural space can be partially evacuated. Once the child is in good condition an osteoplastic bone flap can be turned in order to remove the membranes which in children is the most important step, since the inner membrane is tightly pressed against the surface of the brain and will impair further somatic and mental development of these children.

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46