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Subdural Haematoma in Infancy

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During the course of the last twenty years subdural haematoma in infancy has been increasingly accepted as a clinical entity which is more common than had previously been thought. Korwitz (1911), in a very large series of autopsies on still-born infants and those up to two years, found that 16 per cent. had intracranial haemorrhage and that the percentages were highest in the still-born and those dying within the first weeks of life. Subdural haematoma was the most common abnormality. More recently studies have shown that of deaths in the new born, between 25 and 50 per cent. are due to intracranial bleeding (De la Villa, 1942; McGuiness, 1943), though subdural haematoma is not as common a finding as in Korwitz's time. Although the number of cases associated with birth trauma may well be on the decrease, it is likely that in the past the condition has been overlooked on many occasions, particularly when in the chronic stage or an erroneous diagnosis such as hydrocephalus, cerebral palsy, pylorospasm or febrile convulsions has been made without the possibility of this disorder being borne in mind or the proper tests performed to establish the fact. Furthermore, antibiotics have resulted in saving the lives of many severe cases of acute meningitis which previously would have died, and it has been noted that in a large number of these cases there is an effusion into the subdural space complicating the primary disorder which, clinically and histologically, is indistinguishable from subdural haematoma due to other causes. An analysis of 24 cases is presented illustrating the problems of pathology, diagnosis and treatment.

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Historical Note

Although the written history of subdural haematoma in adults goes back to the sixteenth century, little attention was given to this condition in infants until the turn of the present century. Up to 1896 only 57 cases had been reported and none of these had been cured (Herter).

Finkelstein (1904) appears to have been the first person to make organised attempts at diagnosis and therapy, and recommended "puncture of the fontanelle," as a subdural tap was then called, with the injection of gelatin into the space to absorb the fluid and prevent its re-accumulation. This treatment was also recommended by Rosenberg (1913), who published the first large series of cases. Of his 60 patients, 30 died; and of the 15 who could be traced, very few were normal.

Apart from a few isolated references, interest in the condition then waned until after the classical work of Putnam and Cushing (1925) had served to put together the clinico-pathological picture in adults.

Interest was revived by Sherwood (1930), who published a comprehensive historical review and reported nine cases. His patients were treated by aspiration of the haematoma

alone. On this conservative regime three of his cases were normal, one died and the other five were either institutionalised or retarded. He wondered if operation would be advantageous.

Peet and Kahn (1932) undertook the radical therapy and reported nine cases. Once the diagnosis was established by subdural tap, these authors turned down a craniotomy flap and removed the haematoma and membranes. Four patients did well and subsequently developed into normal children, while five died.

Two years later Naffziger and Brown (1934) observed that the clinical picture in infants varied from that of adults and that the problems were quite different. They pointed out that . . . "infants do not tolerate satisfactorily the sudden change in the content of the sacs with the resulting alterations in the intracranial pressure relations . . . on the other hand, one is confronted with the importance of relieving the intracranial pressure and compression of the brain before permanent alterations occur." They treated five cases by aspiration alone or with surgical drainage through trephine openings. Of these, two died, one had impaired vision and mental retardation, one was normal and one was still under treatment. They stressed the importance of distinguishing between hydrocephalus of congenital origin and subdural haematoma, and they believed that . . . "if sufficient attention were given to this problem, subdural haematoma would be found to occur frequently."

This work paved the way for the basic studies of Ingraham, Heyl and Matson, who have reported the largest number of cases so far treated (219). While agreeing with Naffziger and Brown on the basic principles of treatment, they feel that removal of the subdural fluid alone is inadequate and hold that when the infant is sufficiently improved by slow decompression, radical removal of the membranes, if present, is essential if the child is not to suffer lasting damage.

Other reports have been published by Dowman and Kahn (1942), Elvidge and Jackson (1942), Kinley, Riley and Beck (1951), Gutkelch (1953), Smith, Dormont and Prather (1951), Everly Jones (1953), Wertheimer (1956), Ormiston (1956), Hankinson and Amador (1956).

Aetiology and Pathology

Although many aetiological agents have been suggested, only two of them are known to be of common occurrence—trauma and meningitis. Personal search of the literature published in the last forty years discloses some 535 cases of subdural haematoma which have been reported in varying detail. Of these, 132 were directly related to meningitis, while of the remaining 403, 177 had an antecedent history of trauma, or traumatic, or abnormal birth (excluding caesarian section). Two hundred and twenty-one cases were of mixed or unknown aetiology. In most cases the primary cause was completely unknown, while in others, congenital syphilis, diphtheria (Rosenberg), otitis media (Penfield, 1923), mastoiditis and brain abscess (Chambers, 1925), haemorrhagic disease of the newborn, scurvy, sepsis, pertussis, venous congestion (Sherwood, 1932), saggital sinus thrombosis (Gutkelch, 1953) and cerebrovascular anomalies (Peterman, McLean, Matson and Ingraham, Authors) have been suggested. Because the clinical presentation and problems associated with this cryptogenic group, the largest group of all in fact, closely resemble those associated with trauma, they will be considered as one mixed group. In addition, it has been accepted for many years that even minor degrees of trauma may be responsible for the development of this condition in the adult (Trotter, 1914). It may well be that in a number of cases minor degrees of trauma may have escaped notice, while some parents may have been reticent of admitting to such a history for fear of being considered negligent.

The meningitic group will be discussed separately, mainly on account of present custom, although the resulting pathology and the problems associated with their treatment are identical with those of the former group.

In our series there are 14 traumatic cases, six of unknown aetiology and four following meningitis.

Traumatic or Cryptogenic Group (Mixed Group); 403 Cases Analysed; 177 Traumatic and 226 Unknown.—This disease is one of neonatal life. Of 135 cases in which the age at onset of the symptoms is noted, 79 (59 per cent.) appeared during the first three months of life. A history of trauma or abnormal birth was recorded in 43 per cent. of cases. In 122 cases in which the nature of the trauma was recorded, 25 (20 per cent.) have a history of post-natal injury and the remainder one of abnormal birth. The group is therefore distributed as follows:

Postal-natal head injury	9 per cent.
Birth injury	34 per cent.
Unknown aetiology	57 per cent.

When the files of 50 infants admitted to University Hospital, New York City, for non-neurological complaints were examined, only 16 per cent. had a history of abnormal birth, or less than half that of the corresponding value in the subdural group.

Skull fractures were noted in only 25 of the 403 cases, or 5 per cent., and fractures of the long bones occurred in 13 cases (2.5 per cent.). Eight of these latter cases have no history of trauma and this seems to suggest that even quite major traumata may pass unnoticed or that the parent is unwilling to make the admission. Of 287 cases, 186 (or 65 per cent.) were in males. Matson and Ingraham have suggested that the increased incidence is due to the fact that males are slightly larger than females at birth, and Perlstein and Hood have shown that there is an increased incidence of intracranial birth injury in both premature and large infants. All these figures indicate that whereas trauma is an important factor, it is not the only one.

Hypoprothrombinaemia and vitamin K lack are known to occur in the newborn, the deficiency becoming increasingly marked during the first 72 hours of life. It is particularly low in premature infants. This deficit of prothrombin, throughout the first months of life, renders the infant more susceptible to the effects of head trauma than its elders, since small vessels, which if ruptured would normally clot at once, may continue to ooze insidiously for a longer time instead. Sharpe (1925 and 1926) has shown that some degree of intracranial bleeding may occur in as many as 10 per cent. of apparently normal births.

Whereas in the past there was much discussion about the origin of the condition, most are now of the opinion that a venous haemorrhage is the first occurrence, followed by organisation and the formation of a membrane around the clotted blood, and a number of bleeding points have actually been observed in adults—the superior longitudinal sinus (Oldberg), parietal veins Laudig *et al.*, Authors), bridging and arachnoidal veins (Leary), while several authors have seen tears of the tentorium cerebelli and straight sinus in infants.

Anderson has reported three cases in which a subdural haematoma complicated an operation for the relief of hydrocephalus. It seems that in these three cases intracranial pressure had been high prior to surgery and that when this was relieved, following ventriculo-ureterostomy, the thinned cortex tended to pull away from the vault, thereby tearing the veins running to the superior longitudinal sinus.

It may well be that when at delivery the compressive effects of forceps, prolonged or precipitate labour are suddenly removed, there is a rapid readjustment of the suture lines, a sharp drop in intracranial tension, a pulling away of the cortex from the dura, with laceration of the bridging veins, clotting of the blood being delayed by hypoprothrombinaemia.

Trotter (1914) observed that the brain was protected from lateral movement by the falx, but that when movement of the head was in the sagittal plane there was nothing to prevent the brain from continuing to travel if the head stopped sharply, thereby putting the arachnoidal bridging veins on the stretch and even rupturing them, which likelihood is enhanced by the softness, easy mouldability and rapid growth of the infant skull. Herein may be the cause of the haemorrhage in the post-natal traumatic group.

It has been suggested that the falx and tentorium serve to limit the extent of the bleeding centrally and posteriorly. Of 401 cases, 291 (72 per cent.) are bilateral, and this suggests that numerous vessels are torn on any one occasion. One of Peet and Kahn's cases communicated beneath the falx, and possibly one of ours (R.G.). The clots are usually situated in the temporo-parietal region and are rarely found elsewhere, although they may occur in the midline (Ormiston, 1956), in the posterior fossa (Coblentz, 1938; Crawford, 1935; Ormiston, 1956) and also several in which, the subdural membranes extending down into the thoracic region, the symptomatology mimicked a bulbar palsy. We have thought that at times the fluid may extend all the way down into the lumbar region.

The initial bleeding cannot be long sustained, since the mere presence of blood within the subdural space must increase the intracranial pressure and tamponade the bleeding point. The future course of the patient seems to depend upon the amount of blood extravasated and on the possibility of its being connected with the subarachnoid space and draining away, when no symptoms will arise. If the quantity of blood is large and confined to the subdural space there will be an early increase of intracranial pressure, with rapid onset of symptoms; if the quantity is small, the onset of symptoms is delayed. During the course of the first few days after the haemorrhage the blood clots and haemolysis commences. Zollinger and Gross have shown

that when blood is haemolysed it is five times more osmotically active than when unhaemolysed. Being closed in a pocket between the dura and the arachnoid, it is believed that the clot begins to attract tissue fluid of low osmotic pressure from surrounding interstitial spaces, and Gardner (1932) in fact has placed cellophane sacs containing blood in the subdural spaces of dogs and observed an increase in size.

At the same time organisation commences. Capillaries, round cells and fibroblasts invade the clot from the inner surface of the dura, and in the course of the next few days a thin, fibrous membrane is laid down. From the edge of the clot the process of fibrosis creeps inwards on the arachnoidal surface of the haematoma. Thus, rather than becoming a solidly organised mass, a hollow cyst forms by this ingrowth of granulation tissue from the side of the clot on to the inner, or arachnoidal, surface (illustration 1). The arachnoid itself, however, plays little part in this process of organisation, although it may become a little glazed in colour. The inner membrane resembles that of the dural surface, but is invariably much thinner. At operation this distinction into two membranes may or may not be apparent and depends upon the amount of fluid that has been drained preoperatively and the degree of organisation. It seems for some reason at present unknown that this particular region of the body cannot remove an haematoma in the usual manner and can only make a poor attempt at organisation.

During the course of the next few days or weeks there is a progressive increase in the size of the fluid content as well as a progressive breakdown of the osmotically active proteins to diffusable sizes. The haematoma, therefore, increases to such a size as to make itself manifest or, much more rarely, becomes stabilised at a size which is inadequate to produce serious symptoms. As the haematoma enlarges, further haemorrhages may occur from other veins which become stretched or from torn granulation tissue, and the condition becomes aggravated. The rate at which changes occur in the fluid and in the production of membranes was estimated by Munro and Merritt in adults. In an analysis of 47 cases in which the exact date of trauma was known they found that from four to five

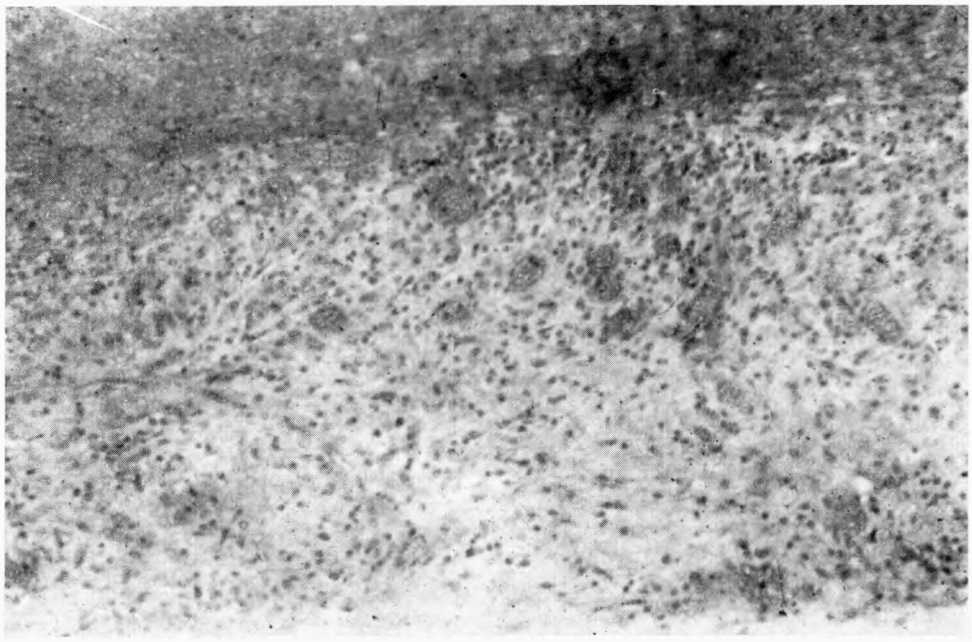
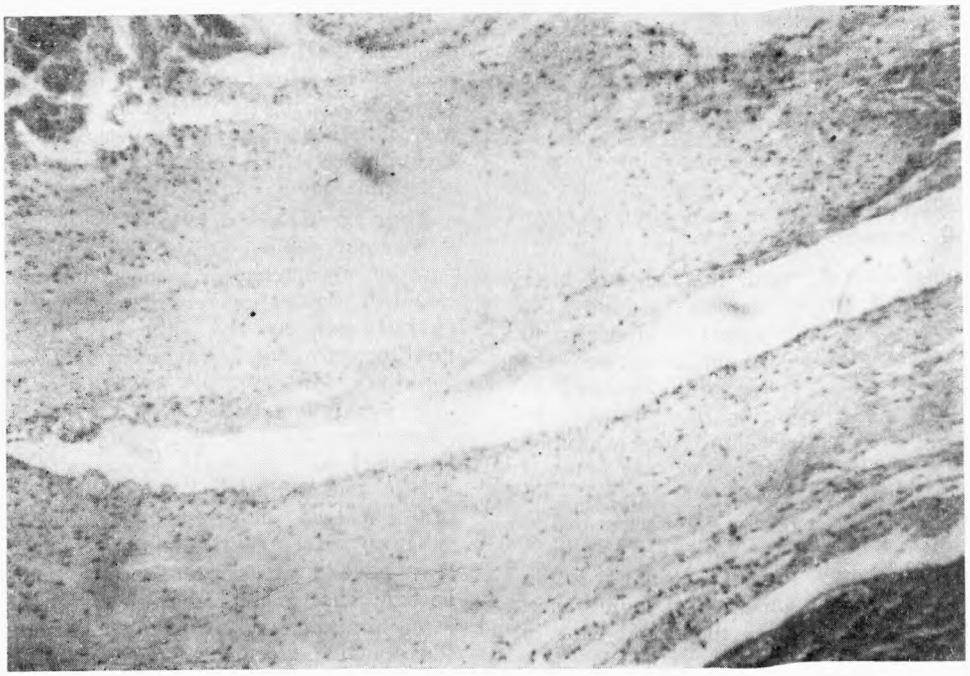
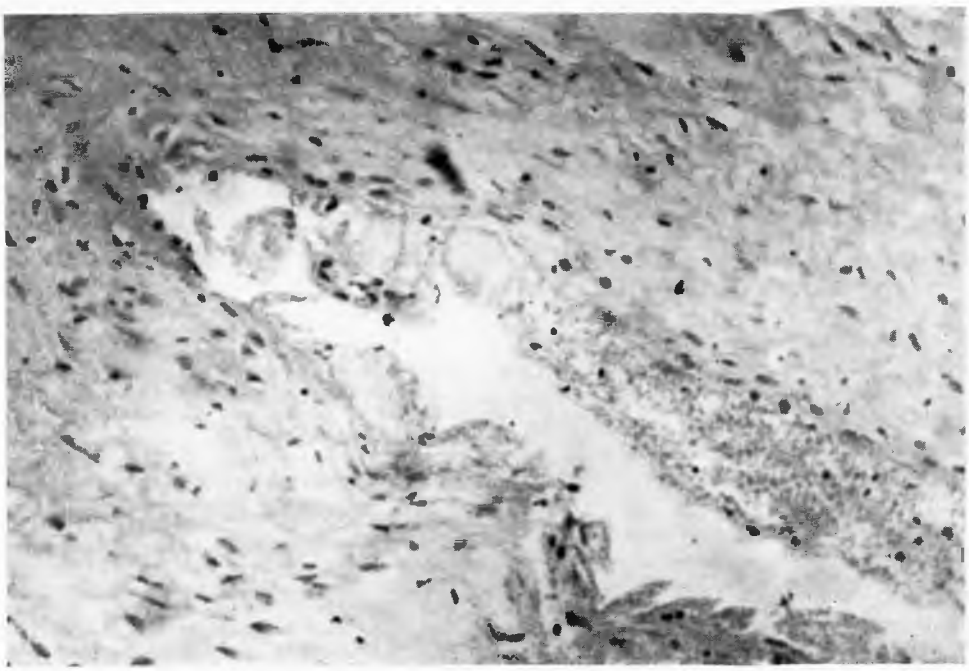


Illustration 1a.—Medium power cross section of the outer membrane which has been separated from the dura mater. Organisation is proceeding from the lower portion of the picture which was formerly attached to the dura mater upwards into the blood clot, which can be seen on the upper portion of the picture. Capillaries, round cells and fibroblasts are steadily invading the clot from below, leaving behind them an organised membrane as they advance into the clot.





Illustrations 1b and 1c.—The edge of the sac showing the continuity of inner membrane (upper) with outer membrane (lower). The cavity contains some red blood corpuscles which will later on be organised.

days after the injury there was a definite thin layer of fibroblasts beneath the dura mater, and that by 17 days there was a thin membrane on the arachnoidal surface of the clot as well. No membrane was seen in any of our cases before the seventeenth day after the occurrence of the haematoma, and then it was very thin. In one patient there was no membrane on one side at 96 days. Five cases operated on after 96 days showed thick membranes.

Munro and Merritt found that, shortly after the occurrence of the haematoma in adults, the protein value of the supernatant fluid begins to rise and reaches its maximum at around the twentieth day. It then drops rapidly until, at about thirty days, it begins to stabilise. In several of our cases whose fluid was examined for protein the value was found to vary between 2.5 gm. per cent. at seven days, 0.9 gm. per cent. at 56 days and up to 5.2 gm. per cent. at 180 days in different patients. Both these findings suggest that the whole process of disintegration of blood, as well as organisation of membranes, occurs much more slowly in the infant than in the adult.

The average duration of haematoma in 11 of our cases, in which the date of origin can be accurately fixed, is 61 days. In a chart prepared by Munro and Merritt the average duration of 41 adult cases from trauma to operation is 39 days or two-thirds of the time it takes for a haematoma in an infant to come to operation. It seems that this delay may be due both to the slower process of organisation and destruction of the blood clot and the decompressive abilities of the infant skull (illustration 2).

From the standpoint of intracranial mechanics there seem to be two very important factors in the infant which are not present in the adult and which account for the difference in symptomatology in chronic cases. In the adult the chronic subdural haematoma produces its symptoms by the interaction of increased intracranial pressure and a space occupying lesion pressing on a non-expansible skull and well developed brain. The infant, on the other hand, has open fontanelles and suture lines, which provide a ready means of decompression.

Papilloedema, vomiting, headache and stupor are accepted symptoms in the adult, but in the child enlargement of the head and opening of the suture lines may well be all that occur. It is this expansibility of the immature skull, therefore, that is one of the most important causes of chronicity of the condition in infants.

Another and extremely important factor is the growth of the infant brain. Coppelletta and Wolbach have estimated that the infant brain doubles in size during the first three months and again during the next six months. The brain may fail to develop at the usual rate, so that a mentally retarded infant is produced and, by remaining small, allow space for the haematoma.

Four cases showed unusual fluid findings, but it was not possible to determine accurately the date of occurrence of the haematoma nor to establish the aetiology. These cases are all characterised by low values for the subdural fluid protein, which range from 31.4 mg. per cent. to 141.3 mg. per cent. Two developed thin membranes, while the other two did not. These cases fall into that group which has been described as "subdural hygroma" and which Munro and Merritt feel is caused by the admixture of blood with cerebro-spinal fluid in the subdural space. With their lower protein values they have a greater tendency to stabilise and become chronic and may well represent some group intermediate between the traumatic and the infective (meningitic) types.

Examination of the clinical types shows that they can be divided into three groups: 1, Acute; 2, Chronic; 3, Chronic Relapsing (Davidoff and Dyke).

1. *The Acute Type* manifests itself at any time and there is frequently a history of trauma. There is a severe haemorrhage, together with a greater rise in intracranial pressure than the brain and skull can tolerate. The child shows early signs of acutely increased intracranial pressure and possibly signs of brain injury as well. The latent period is short and deterioration rapid.

Case Report No. 1:

P.D., age six days. The patient weighed 9 lbs. at birth and was the third offspring of a healthy mother. Labour was spontaneous, with a normal presentation, but was rapid and was completed in two hours. The child appeared well for six days, when she had a generalised convulsion. On admission to University Hospital a tense fontanelle 7 x 5 cm., a wide sagittal suture and occasional twitchings on the left side were noted. The right pupil was larger than the left. The child vomited after each feeding, occasionally projectile, and had a slight bronchitis. Skull X-rays showed no fracture. A subdural tap revealed 8 c.c. of bloody fluid from the right subdural space; the left side contained no fluid. The child was treated by aspiration of the subdural space, but despite this her head continued to enlarge, and five days after admission measured 37.5 cm. as compared to 36 cm. on admission.

Despite repeated subdural taps which ultimately became less productive, the child became grey and stuporous and the fontanelles very tense. The subdural

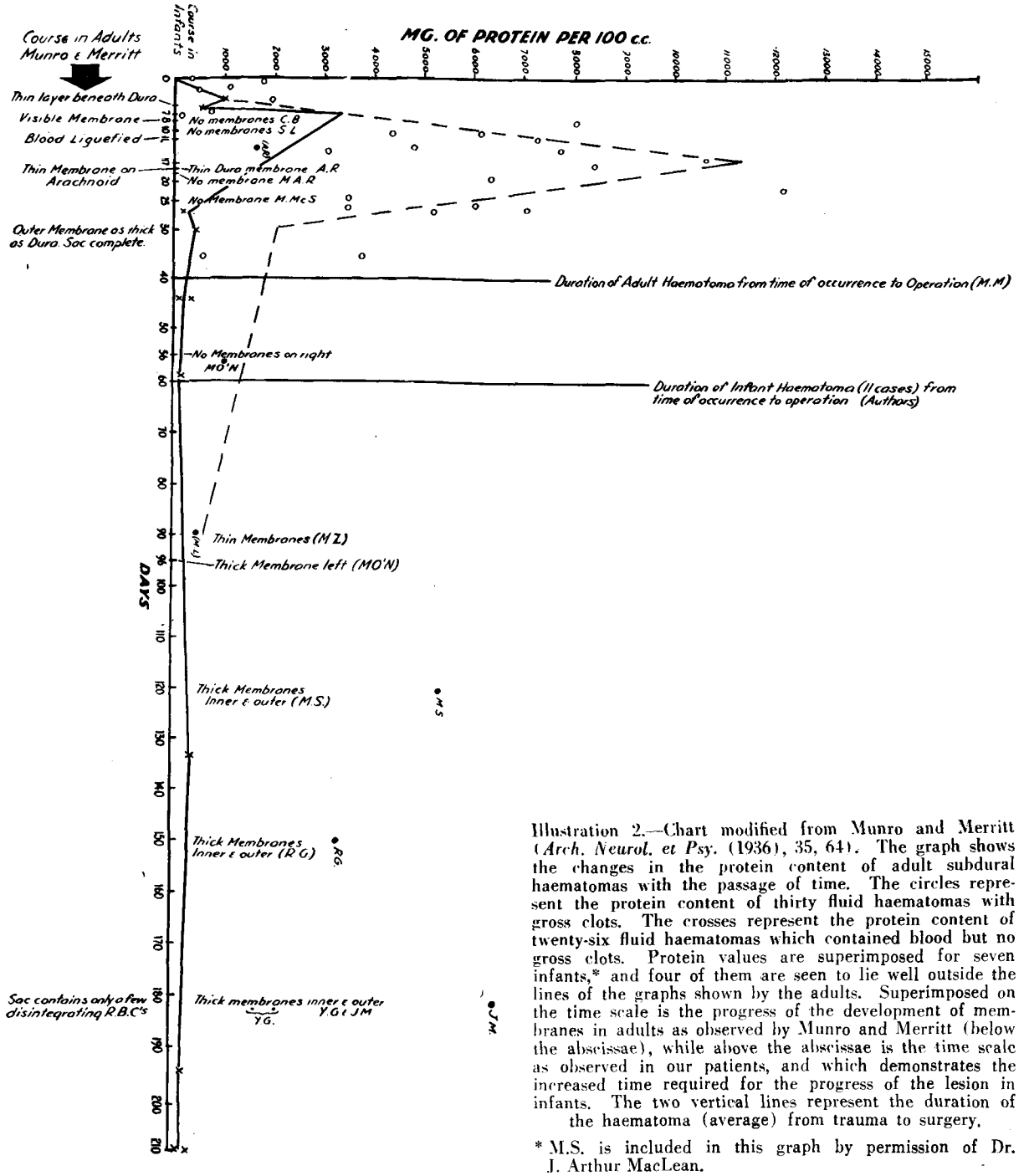


Illustration 2.—Chart modified from Munro and Merritt (*Arch. Neurol. et Psy.* (1936), 35, 64). The graph shows the changes in the protein content of adult subdural haematomas with the passage of time. The circles represent the protein content of thirty fluid haematomas with gross clots. The crosses represent the protein content of twenty-six fluid haematomas which contained blood but no gross clots. Protein values are superimposed for seven infants,* and four of them are seen to lie well outside the lines of the graphs shown by the adults. Superimposed on the time scale is the progress of the development of membranes in adults as observed by Munro and Merritt (below the abscissae), while above the abscissae is the time scale as observed in our patients, and which demonstrates the increased time required for the progress of the lesion in infants. The two vertical lines represent the duration of the haematoma (average) from trauma to surgery.

* M.S. is included in this graph by permission of Dr. J. Arthur MacLean.

fluid at this time consisted of thick old blood. Surgical intervention was forced by the patient's condition nine days after admission, when a trephine hole was made on the right side and very many old clots were washed out. There were no membranes. The space was drained and the wound closed. The patient improved and continued to do well. There are no sequelae three years following operation.

In this case a large amount of bleeding occurred, presumably at the time of labour. Treatment by aspiration was attempted, but was unsuccessful because of the thickness of the blood and clots. Symptoms occurred so rapidly that liquefaction did not have time to occur and aspiration had to be abandoned in favour of a trephine operation.

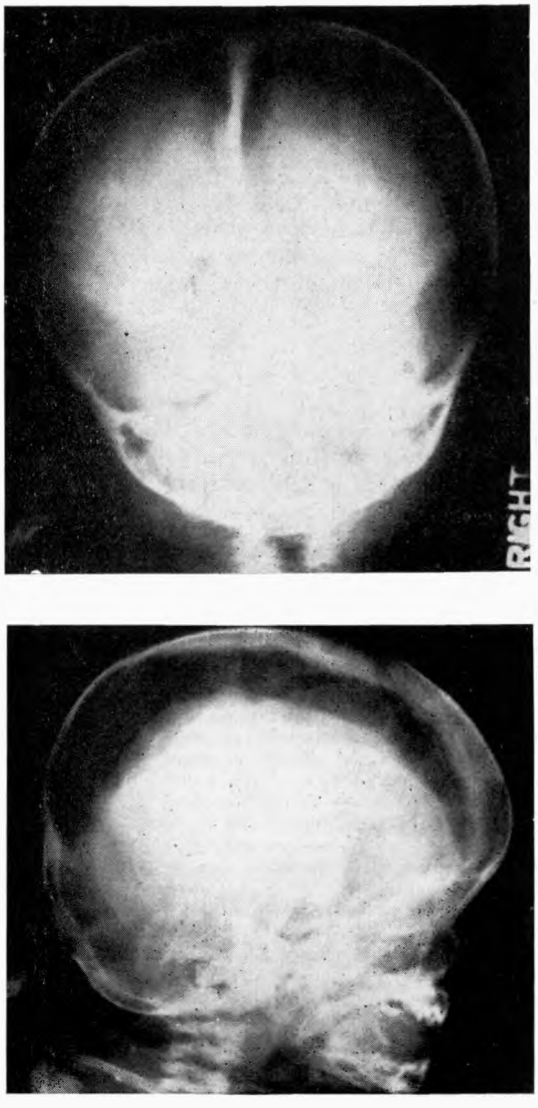


Illustration 3.—Air in the subdural space (Dr. T. I. Hoen). (Case report No. 2.)

2. *The Chronic Type of Late Infancy or Early Childhood.*—A history of trauma may or may not be present and vague symptoms may have been noted for one or more months. Ultimately the abnormality is observed or acute symptoms supervene.

Case Report No. 2:

R.G., male, age five months. There was no history of birth trauma. The child was admitted because of difficulty in eating, vomiting and left-sided convulsions of one month's duration. The anterior fontanelle and sutures were open and bulging. Electro-encephalography showed diffuse abnormality. Subdural tap with the injection of air demonstrated large bilateral subdural haematoma with marked cerebral agenesis (illustration 3), and membranes were removed through enlarged bilateral trephine holes. When last heard of two years and four months later, he was doing well, but was still slightly retarded. The electroencephalogram had become normal.

Here the skull allowed a decompression and the failure of development of the brain likewise created space for the enlarging lesion. However, despite this, the lesion expanded more rapidly than the decompressive abilities of the skull and brain could tolerate and he developed signs of increased intracranial pressure.

Case Report No. 3:

J.F., male, 2½ years, was admitted because of retarded development. There was a history of prematurity and of birth injury, labour being induced and lasting approximately 13 hours. On admission his head was found to be small for his age and he made noises, but said no words. There were no abnormal neurological findings. Pneumoencephalography was suggestive, in that there was poor filling of the subarachnoid spaces. Trephine holes showed bilateral subdural haematoma with membranes on both sides, which were removed by craniotomy. When last seen, approximately 1½ years later, he was found to be doing well. He was much more alert and communicative than previously and was classed by his pediatrician as only slightly subnormal.

In this case the child's head was not enlarged; if anything, it was slightly small. The space occupying lesion was present at the expense of the normal development of the brain.

3. *Chronic Subdural Haematoma Dating from Infancy.*—These cases show essentially the long term results of those of Group 2, which have not been detected earlier. There were no cases of this nature in this series, but their existence has been well documented by Davidoff and Dyke (1938), Critchley and Meadows (1932-33), Goldhahn (1930), Wadsworth-Schwartz (1952) and Spiller and McCarthy (1899), all of whom have reported cases in adults or older children who clearly have harboured the lesion since infancy. Seizures and mental retardation are the commonest symptoms.

This group underlines the importance of early diagnosis and treatment if permanent damage is to be avoided.

MENINGITIC GROUP

Cured cases of acute meningitis are commonly associated with a severe morbidity and, whereas meningococcal meningitis was at one time the commonest infective disorder of the meninges, Anglin *et al.* (1952) found that *H. influenzae* was now more common and accompanied by the more severe sequelae. Bloor *et al.* (1950) found that among 39 cases of meningitis due to *H. influenzae*, there were 10 deaths and 11 poor results. Among the 11 poor results there were one severe behaviour problem, three with bilateral pyramidal signs or hydrocephalus, one hemi-paretic and one was deaf; 10 were retarded.

In a review of the autopsy records of the children's hospital in Boston, McKay, Ingraham and Matson (1953) found that 25 patients out of a total of 218 dying from acute meningitis had been noted as having significant amounts of fluid in the subdural space. Smith *et al.* (1951) suggest that this fluid may occur frequently, since in 43 proven cases of meningitis under the age of two years they found increased quantities of subdural fluid in 20 of them. They found that the incidence of those suffering from effusions was highest in children under one year. This observation is supported by McKay *et al.*, who found fluid in 27 of 46 case (56 per cent.) under the age of one year, five of 45 (11 per cent.) between one and two years, and one of 44 (2.2 per cent.) between the ages of two and three. Of 36 other recorded cases, the average age was seven months and only one of these was over the age of one year. McKay *et al.* found that there was no uneven sex incidence except in so far as, in their series, males suffered from meningitis more than females.

Of 132 reported cases, the infecting agent has been as follows:—

H. influenzae	42 times
D. pneumoniae	39 ,,
N. meningococcus	19 ,,
Sterile culture	17 ,,
H. influenzae and pneumococcus	3 ,,
Streptococcus	4 ,,
Staphylococcus	2 ,,
B. coli	2 ,,
Paracolon bacillus	1 ,,
P. auruginosa	1 ,,
M. tuberculosis	1 ,,

It is felt that the clinical course of patients in the past may have been rendered more hazardous by pressure on the brain caused by this fluid, and that subsequently organisation of the exudate with membrane formation may be responsible for the continuation of that pressure, with long-term morbidity in some cases. Moreover, the significant reduction in the mortality effected by the antibiotics may result in an increase in this type of lesion in the severer cases and the development of abnormalities previously seen but rarely.

Fluid may be detected in the subdural space in significant quantities quite early in the disease, and when this occurs is evidence of a very severe illness. In one of our cases it was found on the third day. On the other hand, in another case it was found on the nineteenth day, although taps had been performed as early as the second day because of the child's poor condition, while in one of Smith's cases it was not found until the sixty-second day.

Smith *et al.* have likened the process to that of a pleural effusion. Gitlin (1955) has examined the protein content of subdural fluids obtained from infants whose lesions were either traumatic or inflammatory. In both cases he finds that there is a disproportionate increase in the amount of smaller moleculed serum albumen compared to globulin. This suggests that some degree of capillary damage has occurred in both cases, presumably of an inflammatory nature, and that in both cases the fluid is at least in part exudative in nature.

This opinion is supported by Crosby and Bauer (1956), who have examined the radioactivity of the skull after the ingestion of P^{32} in these cases. They find that P^{32} activity is greatest when membranes are present and that the level of radioactivity varies with the aetiology, cases associated with malnutrition having a lower value than those associated with trauma. These authors believe that their findings support the thesis that all subdural collections are effusions resulting from change in the vascular permeability and that osmosis forms only a part of the process.

The fluid, in the first instance, is either turbid or xanthochromic. Organisms can occasionally be grown from it, but of the antibiotics, penicillin at least enters the subdural space in quantities adequate to control susceptible infections (Arnold, 1951). Where the fluid is turbid there is usually a considerable outpouring of fibrin which appears to involve both arachnoid and dura mater, and while at operation it can be readily removed from the latter, it may not be so freely separated from the arachnoid. Organisation of these flecks of fibrin is responsible for the dural and arachnoidal thickening and

Table 1

Chart showing changes in spinal fluid and subdural fluid (C.P.). In this case fluid did not appear until two weeks after the start of the illness and while the spinal fluid was returning to normal. The appearance of convulsions was the indication for subdural puncture. Fluid then continued to form in increasing quantities, though it was not always removed "in toto" on each and every occasion that the space was tapped, and then began to diminish in quantity, though at operation it was obtained from both sides. On the right side the fluid became increasingly bloody and dark, though the protein value did not increase very much and the number of white cells remained more or less constant.

C.P.—Aged 7 Months.

Lumbar Puncture						Right Subdural Space						Left Subdural Space					
Date	*Colour*	*WBC*	*Prot. (mg. %)*	*Sugar (mg. %)*	*Cult.*	*Amt.*	*Colour*	*Cells*	*Prot. (g. %)*	*Sugar (mg. %)*	*Cult.*	*Amt.*	*Colour*	*Cells*	*Prot. (g. %)*	*Sugar (mg. %)*	*Cult.*
2/21	Yellow Grey	3,000	280	25	H. Inf. B.												
2/25	370 Turbid	12,000	180														
2/26	Turbid	85% Polys 14,000	268	100				Neg.						Neg.			
2/27	Turbid	6,000	320	87													
2/28	Turbid	85% Polys 2,500	216	93													
3/1	Clear	370	210	64													
3/4	Yellow	125	200	70													
3/10	60-100 Clear	90	190	78													
3/11						4 c.c.	Yellow	71,500 RBC 7,500 WBC	3.79		Ster.	4 drops					
3/12						½ c.c.						20	Yellow	1,400 RBC 130 WBC	1.054 1.054		Sterile Sterile
3/13						6 c.c.	Yellow	4,400 RBC 56 WBC	1.7	71		3					
3/14												22	Turbid	1,200 RBC 1,585 WBC	1.47	69	

3/17	Clear										15	Dark Yellow				
3/18											POLYETHYLENE CATHETER IN SUBDURAL SPACE					
3/20											15	Xantho	9,300 RBC 130 WBC	1.3		
3/21											5	Xantho	95 RBC 32 WBC	1.06		
3/22					12	Xantho	4,470 RBC 280 WBC	1.7			10	Xantho	3,090 RBC 120 WBC			
3/23					18	Xantho	19,500 RBC 133 WBC	1.35								
3/24					16	Yellow	13,860 RBC 640 WBC	1.1	74		20	Light Yellow	2,640 RBC 226 WBC			
3/25					5	Xantho					8	Light Yellow				
3/26					12	Dark Yellow	4,300 RBC 500 WBC	1.68			15	Light Yellow	830 RBC 70 WBC	.90		
3/27					6	Dark Xantho	39,000 RBC 4,700 WBC	1.35			8	Light Yellow	5,560 RBC 180 WBC	.50		
3/28					16		34,500 RBC 710 WBC	2.0			0					
3/29											0					
3/31					12	Very Dark	64,000 RBC 660 WBC	2.6	64							
4/1					16	Xantho		1.9	80		20	Light Xantho		1.15	74	
4/4											BURR HOLES. LT. ONLY.					
4/10					7	Bloody					0					
4/12					13	Bloody										
4/16					6											
5/3					Right Craniotomy—Small Membranes											

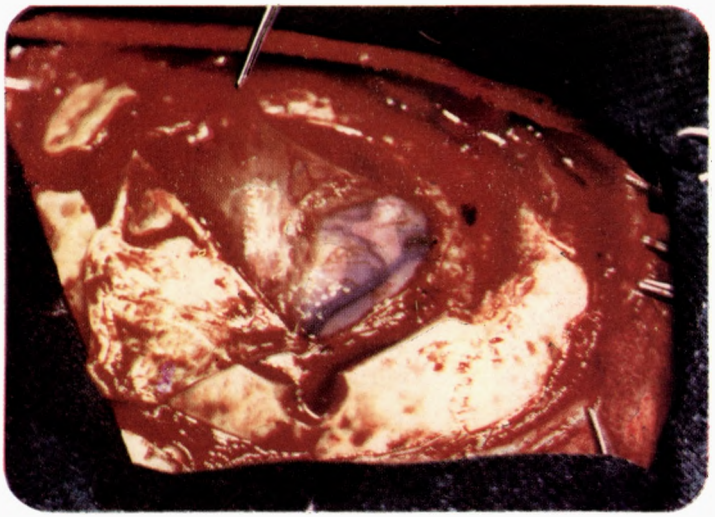


Illustration 4a.—*The dura mater has been reflected together with the outer membrane, and the inner membrane is held in the forceps and is being separated from the underlying and slightly glazed arachnoid (Dr. I. S. Cooper). (Meningitic group.)*

also, in some cases, for cortical scarring and adhesion.

As the inflammatory process subsides the production of fluid usually lessens, until eventually none can be obtained at subdural tap. On the other hand, it sometimes happens that the fluid, far from becoming less in quantity, increases and may become blood-stained, so that a dark-brown fluid may be obtained. That this colouration is not primarily due to bleeding caused by trauma at the time of the subdural tapping is shown by our case, L.B., in which the fluid discovered at ventriculography was a dark brown, yet no subdural taps had been performed, and presumably it originates by rupture of distended vessels in the inflamed meninges. Clearly, however, it is possible for bleeding to be caused by traumatic attempts to obtain fluid (see case report No. 6). When the fluid becomes deeply xanthochromic and bloody, or is infected or turbid, the tendency towards membrane formation appears to be greater than when it is light yellow; on the other hand, the length of time that the fluid is present may be significant. A typical sequence of changes found in cerebro-spinal and subdural fluids is illustrated by Table I (C.P.).

The process of the development of the membranes appears to be identical in all cases, no matter what the primary aetiology. There is the same invasion of the clot or the fibrinous exudate by fibroblasts, histiocytes and capillaries from the inner aspect of the dura, with the laying down of a thin fibrous membrane which thickens as organisation proceeds. A similar process occurs on the inner surface, but the flecks of fibrin cause the membrane to become more intimately related to the arachnoid and thicker than in the traumatic type (illustration 4a, b and c).

Of 40 reported cases, 25 (62 per cent.) were bilateral and 15 (38 per cent.) were unilateral.

Clinical cases can be classified into two groups, acute and chronic.

The Acute Lesion.

Case Report No. 4:

E.P., a male child, aged six months, was admitted to Bellevue Hospital, New York City, on the 30th January, 1955. He had a three-day history of convulsions with cough, fever and irritability for two days and vomiting for one day. On the day of admission he developed a slight paresis of the right leg and arm. He had had a lumbar puncture after his first convulsion which had been negative, and he had been given Gantrisin syrup. On admission he had a temperature of 103.5° F. and was stuperous and irritable. His neck was stiff and there was hyperreflexia, a bilateral babinski response and a right hemiparesis. There was a slightly bulging fontanelle and the disc margins were blurred.

W.B.C. was 18,000/c.mm. with 61 per cent. lymphocytes. Lumbar puncture showed a clear fluid, with 387 white cells, 77 per cent. being lymphocytes. Protein was 90 mg. per cent. and sugar was 62 mg. per cent. Culture grew beta-haemolytic streptococci. The child continued to have right-sided convulsions and on the same day subdural taps were performed. That on the left side yielded 8 c.c. of turbid yellow fluid. Subdural tapping on the right side was negative. Penicillin, chloromycetin and sulphadiazine were administered.

The following day subdural taps on the right side produced 12 c.c. of cloudy pink fluid with only 1 c.c. from the left side. Lumbar puncture on the third day showed a marked improvement, while a small quantity of turbid fluid was obtained from the left subdural space and 28 c.c. of pinkish fluid from the right side.

The child was very much improved, convulsions had ceased and he was taking fluid by mouth.

Subdural taps were not repeated until the eighth day, when the left side was dry, but the right side yielded 30 c.c. of bloody and xanthochromic fluid.

Two days later a further 40 c.c. of xanthochromic fluid were obtained from the right side, and on the eleventh day after admission the right side again yielded 30 c.c. of grossly bloody and xanthochromic fluid. The left side, which had been turbid at the start and was the first side to become positive, remained negative.

On the 11th February the child's condition was excellent, the temperature had been normal for several days and the C.S.F. normal for five days. Under general anaesthesia a trephine hole was made on the left side. This showed a thick outer membrane which, when opened, released about 10 c.c. of a dark xanthochromic fluid. A trephine hole was then made on the right side, and when a very large cavity extending down beside the frontal lobe was seen with a membrane on the surface of the dura, this was converted into a small flap. The membrane was removed from the dura. There was no inner membrane. The whole of the frontal lobe was seen to be compressed medially to accommodate a very large quantity of xanthochromic fluid, possibly as much as 70 c.c., which was lying in the subdural space. Penrose drains were inserted bilaterally and the wounds closed. Drainage of clear fluid was profuse and the protein content of the fluid obtained from the right side, from which all blood clot had been removed, was 1.8 gm. per cent. The drains were removed on the second day, despite the presence of some discharge, to prevent excessive loss of protein.

On the 25th February left temporeparietal craniotomy was performed. A thick outer membrane was found, extending forward under the dura, anteriorly to the flap and upwards towards the fontanelle. This was removed piecemeal and histologically was identical with the organising membrane which is found in the chronic type of haematoma. There was no inner membrane, but the arachnoid was dotted with grey areas of fibrinous material which, on removal, also showed early organisation. Unlike the inner membrane of the traumatic variety, which is easily separable from the arachnoid, flecks of fibrin, some of which measured up to 1 cm. in length and several mm. wide and deep, were part of the arachnoid and had to be excised from it. For this reason not all the bits of fibrin were removed for fear of doing further damage to the underlying cortex, but the dangers inherent in the organisation of this material with the formation of duro-arachnocortical scars were fully appreciated.

Nonetheless, as much of the membrane as possible was removed and the wound closed without drainage. Pneumoencephalography performed later showed only a slight dilatation of the anterior horn of the left lateral ventricle. When last seen, three months after discharge, the child appeared completely normal.

At the time of admission the child was extremely ill with meningitis and the superimposed lesion. Drainage of the subdural fluid from the left side alleviated the right-sided convulsions almost immediately and, by repeated subdural taps, the pressure was kept low until the inflammatory reaction had had time to subside and the child's condition was sufficiently improved to permit trephination and craniotomy. However, the fact that drainage of fluid continued profusely from the right side with a high protein value, even after operation, suggests that the inflammatory process was still present at that time and the risks of reaccumulation of fluid might have been minimised by delaying surgery further. Nevertheless, fluid did not reaccumulate in the subdural space.

The Chronic Type.

Case Report No. 5:

L.B., a boy, aged 3¼ years. On the 23rd May, 1952, the patient had a chill followed by convulsions and fever. He became stuporous and finally comatose and was admitted to hospital. The diagnosis was acute meningitis. C.S.F. studies showed 53 W.B.C. with 24 polymorphs and 29 lymphocytes/cu. mm. and a protein of 100 mg. Culture, however, was negative. He was treated with antibiotics. He remained comatose, and

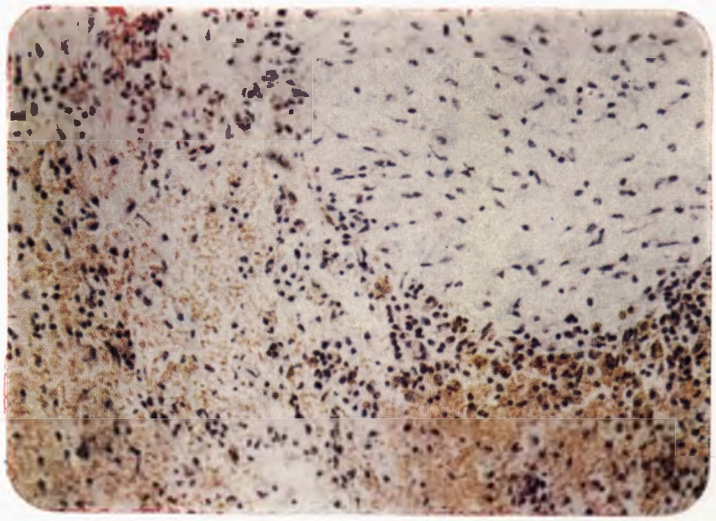


Illustration 4b.—*Section of the outer membrane in the above case (L.B.). Note the area of advancing organisation with the well-developed membrane in the top right-hand corner. (Meningitic group.)*

two days later his C.S.F. white cell count had risen to 1,300/cu. mm., 92 per cent. of which were polymorphs.

By the 30th May he had become conscious once more, but was aphasic. He tended to become drowsy, and repeatedly his condition was found to be improved by lumbar puncture. He presented a considerable diagnostic problem, developing papilloedema and making no improvement in his aphasia. His EEG showed a depressed voltage in the right parieto-occipital area, with some abnormality on the left side, but not suggestive of encephalitis. His head was not enlarged. On the 9th July bilateral trephine holes were placed in the parietal regions and bilateral subdural haematomata with membranes disclosed. The membranes were removed subsequently by craniotomy and the patient did very well, speech returning to normal in several months (illustration 5). Unfortunately the bone flap on the left side became infected and required removal. When last seen he was apparently mentally normal.

In this case the child suffered from acute meningitis, but the complicating subdural haematoma was not discovered until one and a half months later, when signs of increased intracranial pressure developed. The membranes showed active organisation with phagocytosis of degenerating red blood corpuscles, which are present throughout the newly-formed membrane. This section should be compared with those of the traumatic group (illustrations Nos. 1a and b and 4).

The further pathology of such lesions was well described by Spitz *et al.* (1945), who described two such cases which came to autopsy—one in a boy of three years and one in a man of 61 years of age. Both had purulent exudates over the surface of the brain, with exudates on the dura as well as the arachnoid, and the boy also had an organising duro-arachnoidal adhesion. In the latter case there was also a necrotising involvement of the bridging veins which had allowed their infected contents to escape. The organising purulent exudate over the dura resembled that of a traumatic subdural haematoma.

The mode of spread of the infection to the subdural space has raised some speculation. In Spitz's cases it appeared as though it had passed through thrombosed and ruptured dural veins. However, in the case of E.P. (No. 4), the fibrinous exudate was intimately involved with the arachnoid and, although the organism could not be cultured from the turbid fluid obtained from the left side, this former suggests that the normal lining effect of the arachnoid had broken down and the membrane had allowed penetration by the inflammatory process.

In our group of four cases there was one death which contains some interesting autopsy findings and is therefore reported further.

Case Report No. 6:

R.C., a two-months-old child, was admitted to Bellevue Hospital on the 15th July, 1955, with a three days history of fever, laboured breathing and coughing. On examination he was found to be lethargic, with a full fontanelle and moist rales over both bases. White blood count was 9,000 cu. mm. Lumbar puncture showed a cloudy fluid with 127 wbcs/cu. mm., 60 per cent. being polymorphs, a protein of 110 mg. per cent. and a sugar of 20 mg. per cent. and, on smear, showed numerous pneumococci confirmed by culture. He was treated with penicillin and Gantrisin. The patient had intermittent seizures on the day after admission, with periods of apnoea and the passage of black tarry stools during the next two days. The temperature fell and rose again. He was given a blood transfusion. On the 19th July his condition was still poor and he had several focal convulsions, the seizures starting each time in his left leg. The fontanelle was bulging and pulsating. Subdural puncture showed only a few drops of whitish fluid from the left subdural space. He remained in a very poor condition for several days and was given intrathecal pancreatic dornase.

A further subdural puncture was performed on the 27th July, when 10 c.c. of xanthochromic and cloudy fluid were removed from the left side and 12 c.c. from the right side. The following day the right side yielded 9 c.c. (1,500 wbcs/cu), protein 2 gm. per cent. and sugar 68 mg. per cent., while the left side yielded

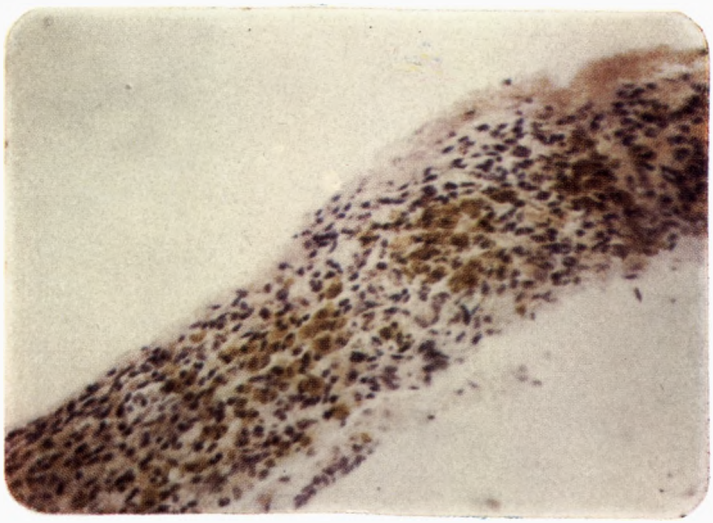


Illustration 4c.—*Section of the outer membrane of a traumatic case. Note the similarity to the inflammatory membrane with fibroblasts, histiocytes containing haemosiderin and red blood corpuscles in various stages of organisation.*

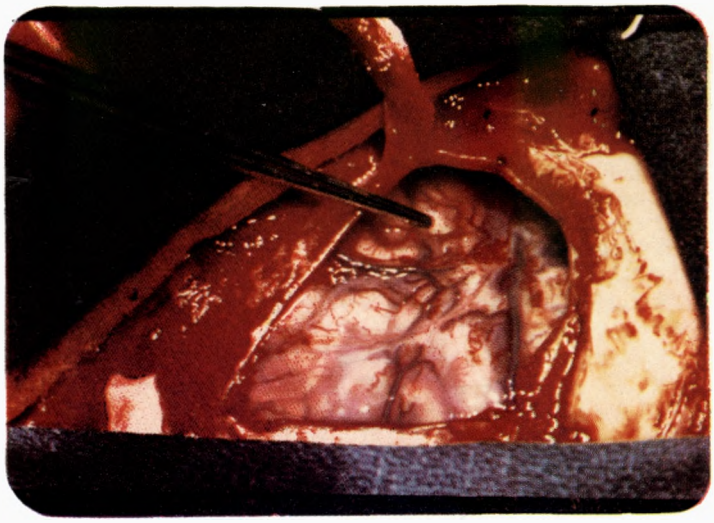


Illustration 5.—*The subdural sac has been removed and the cavity wherein it lay has been exposed. The arachnoid is glazed and contains some small portions of fibrin inferiorly. The forceps are pointing to an area of infarcted brain (Dr. I. S. Cooper).*

5 c.c. (1,000 wbc, 30,000 RBCs, protein 2.5 gm. per cent., sugar 68 mg. per cent.). The following two days the fluid was obtained in diminishing quantities, but it became steadily more bloody and xanthochromic. On 31st August a secondary invader, *H. influenzae para B.* was grown from this subdural fluid and antibiotic therapy was restarted. The infection rapidly subsided. Subdural taps were continued, and after being negative for one day produced increasing quantities of bloody fluid with an extremely high protein value and frequently with the suggestion of very recent bleeding.

On the 10th August bilateral trephine was performed and, on the left side, much fluid was obtained, but no membranes were present. Because of the child's poor condition no attempt was made to remove them, and drains were inserted. On the fourth post-operative day, when he was apparently beginning to eat well and do satisfactorily, he suddenly collapsed and ceased to breathe.

At autopsy there were numerous ante-mortem subdural clots still present and numerous purulent yellowish areas throughout the subarachnoid spaces, especially over the left cerebral hemisphere and around the base of the brain and in the interpeduncular space. There was congestion of the cerebral veins and pial capillaries. There was a large encapsulated subdural haemorrhage over the upper part of the right hemisphere and a similar one over the left hemisphere, near the superior longitudinal sinus.

This patient certainly died from the effects of the overwhelming infection, but the interesting and important point was the encapsulated blood clot over the cerebral hemispheres just at the point where the needles had been penetrating the dura mater overlying the anterior fontanelle. It seems likely that this patient was tapped too frequently and possibly unskilfully, and that the underlying subdural effusion was complicated by bleeding resulting from trauma to the meninges, with the formation of an encapsulated haematoma. There was no herniation of brain nor depression of the cortex, so it is unlikely that this haematoma was the cause of death: however, the organisation of the blood clot and the formation of membranes is one of the very things that we are aiming to avoid by removal of the effusion by tapping. Clearly, improperly performed subdural aspirations may be as harmful as no aspiration at all.

Meneghello and Aguilo (1951) and Hankinson and Amador (1956) report several cases in which it appears that the meningitis occurred in patients already harbouring a subdural haematoma, and from the record one of Sherwood's cases may have been similarly affected.

Signs, Symptoms and Associated Findings

(a) *Traumatic and Cryptogenic Group*

The symptoms and signs of 339 reported cases are tabulated in Table IIA (including Authors cases) as follows:—

Table IIA

		Per cent.
189	cases of convulsions	55
153	cases vomiting	48
135	cases hyperreflexia	42
133	cases tense fontanelle	38
121	cases fever	36
108	cases irritability	32
91	cases coma, stupor	28
83	cases retinal haemorrhages	26
79	cases large head	24
61	cases paresis	18
54	cases anaemia	17
32	cases increased W.B.C.	10
26	cases pallor	8
22	cases diarrhoea	7
22	cases failure to gain weight and malnutrition	7
10	cases open sutures	3
8	cases feeding problem	3
8	cases impaired vision	3
7	cases prominent scalp veins	3
7	cases eyes turned down	3
3	cases broncho-pneumonia	1

The following symptoms and signs in the author's 20 cases occurred with the following frequency:—

Table IIB

	Per cent.
15 cases convulsions	75
7 cases hyperreflexia	35
7 cases vomiting	35
7 cases irritability	35
6 cases coma, stupor	30
5 cases tense fontanelle	25
4 cases retarded	20
3 cases paralyses	15
3 cases enlarged head	15
3 cases retinal haemorrhages	15
2 cases open sutures	10
2 cases fever	10
1 case vitreous haemorrhage	5
1 case prominent scalp veins	5

As previous authors have noted, convulsions are the most frequent symptom and are found in 55 per cent. of cases. These, however, may be misconstrued as teething convulsions, febrile convulsions or idiopathic epilepsy.

A ten-months-old girl (B.A.S.) was admitted to hospital semi-comatose. She had been ill for only one day and had had a convulsion. She was discharged with the diagnosis of febrile convulsions and the correct diagnosis was not established until seizures reappeared and she developed a hemiplegia.

Vomiting occurred in 48 per cent. of cases. In four of our seven cases it was projectile and in the other three varied from a slight dribbling over the edge of the mouth to more obvious regurgitation. Although vomiting is a frequent symptom in this and other intracranial conditions, it is understandable that when it is the only symptom a diagnosis of gastro-intestinal disorder is made first.

A two-months-old girl (M.Z.), brought to hospital with the complaint of dribbling vomiting, was diagnosed as having pylorospasm and she was discharged. Persistence of regurgitation required re-admission and subdural taps demonstrated the presence of a subdural haematoma.

Hyperreflexia is present in 42 per cent. of cases, which may be bilateral or unilateral. Fifty-eight patients, or 18 per cent., showed some paresis pre-operatively as well. Three, or 16 per cent., of our patients had this finding. The most severe case was that referred to above (B.A.S.), who, after having severe convulsions, developed an area of infarction with paralysis of the arm. It is most important that cases such as these, relatively few though they may be, are not labelled cerebral palsy without adequate investigation (as happened in one of Marinacci's cases), because this diagnosis carries with it the connotation of incurable disease.

Varying degrees of drowsiness, ranging from retardation to stupor and coma, are found in one-third of the patients. Sometimes this symptom may not be noticed by the parents until quite late, when irreparable damage has been done (e.g., case report No. 3). In another case a child of 7½ months (T.F.P.) was admitted in poor condition with the complaint that he was sleepy, did not play or grasp things nor lift his head, and that he had a poor appetite and was constipated. On physical examination the findings were those of a stuperous child with a high-pitched cry and hyperactive deep tendon reflexes. The fontanelles were closed. A subdural tap was performed through the coronal suture, and on the right side 20 c.c. of slightly bloody and xanthochromic fluid obtained. Despite this, his condition remained poor and he died the next day. Autopsy was refused.

A tense fontanelle is a sign that is easily missed unless a specific examination is made for it. Nonetheless it was present in 38 per cent. of all cases. In other cases it is normal, or may even be depressed if the child is dehydrated from vomiting, or collapsed. Eighty-three infants (26 per cent.) showed retinal haemorrhages and nine of these, or 3 per cent. of the total, showed papilloedema or secondary optic atrophy. These figures almost certainly do not represent a true proportion, because Statten found haemorrhages in 17 of 23 cases for which they were examined. It is likely that this condition has not been noticed, either because it has not been looked for or because of the difficulties involved in making the examination. However, haemorrhages were found in only three of our patients, despite the fact that a search was made in each. One of ours showed, in addition, a severe haemorrhage in both eyes, causing temporary blindness, which disappeared after the haematoma was removed.

Enlargement of the head is found in one case in four, and in only half of these is the enlargement so gross as to be noticed by the parent. This condition may be diagnosed rather vaguely as "hydrocephalus," and without any investigation a rather gloomy prognosis, without justification, given. Not only do a number of cases of hydrocephalus respond to therapy, but occa-

sionally one of these is found to be mimicked by a chronic subdural haematoma (*cf.* case report No. 2).

Skull X-rays were performed on all our patients, but were abnormal in only three of them—spreading of the sutures twice and fracture of the occipital bone once. Only one patient had a pre-operative pneumoencephalogram, and probably the only use for this test in this condition is in the investigation of cases with mental retardation and few other symptoms. Its routine use in the presence of increased intracranial pressure is contraindicated. Air was injected into the haematoma on three occasions (illustration No. 3), but in each case the diagnosis had already been established by the subdural tap and it served only to outline the extent of the space. One patient M.McS.) was found to have a fractured clavicle as an incidental finding.

Little help is to be gained from an examination of the cerebro-spinal fluid. Xanthochromia is supposed to be a common finding in this condition, yet was present in only two of 13 cases in which a lumbar puncture was performed and was associated with an increase in the protein content. Both these children had had recent traumata and it seems likely that the xanthochromia was due to the escape of blood into the cerebro-spinal fluid at the time of injury rather than a diffusion of blood pigment from the haematoma itself. There was a slight increase in the white cell count in four cases.

The electroencephalogram has not been found to be of great value in diagnosis. It was normal in four cases where a haematoma was present. In seven others it showed abnormality in the parieto-temporo-occipital area on one side or the other, even where bilateral membranes were present; while in two cases it showed a diffuse abnormality without localisation. Marinacci *et al.*, however, found a definite depression of amplitude over the region of the haematoma in seven cases with focal abnormalities also.

The condition may be masked by concurrent disease, coryza, otitis, tonsillitis, anaemia, pallor and wasting. Fever is present in 36 per cent. of cases and the white blood count elevated in 10 per cent., so that where these are the most obvious symptoms it is quite understandable that the basic pathology may be missed.

The extremely variegated clinical picture that may be produced by this disease is illustrated by the admission diagnoses of the 20 cases in this series:—

Subdural haematoma	9
S.D.H. or hydrocephalus	2
S.D.H. or cerebral palsy	1
Pylorospasm	3
Febrile convulsions	2
Tetany	1
Retardation	1
No diagnosis	1

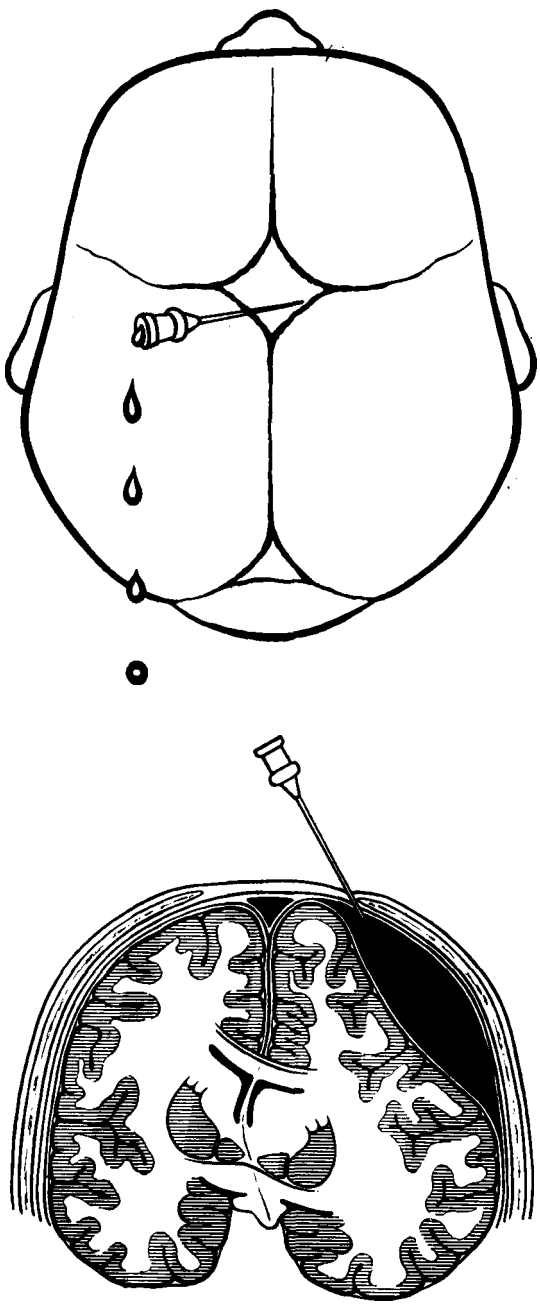
Matson and Ingraham have emphasised that the diagnosis was only to be made for certain by subdural tap. This principle has been followed in every one of our cases in which the diagnosis was suspected and in which it was practicable to perform the test. Of our 20 traumatic cases, 17 had a subdural tap and this was diagnostic in 15 of them, in that fluid was obtained; the remaining two were doubtful. Because it was felt on other grounds that a haematoma might be present, despite the doubtful tap, bilateral trephine holes were placed in these patients, with positive results. In three other patients the diagnosis was established by trephine on the basis of clinical evidence and in the presence of closed suture lines. This suggests, since the diagnostic taps were done by a variety of medical men, that in most hands this test is only correct in 88 per cent. of cases, and it should be emphasised that where there are strong enough grounds for believing that a haematoma is present, even when the subdural tap is negative, exploratory trephine seems justified. It is usually in the recent case in which gross clots clog the needle that difficulty is encountered with the interpretation of the results. Ormiston (1956) had a similar unsatisfactory experience.

Subdural tap is a procedure that is approached with considerable timidity by a great many physicians, who are unaware of the simplicity of its performance. Although the technique is easily mastered, it is not, however, a procedure that should be undertaken without adequate preparation.

In the first place, a needle is being introduced into a space whose reaction to foreign material is known to differ from the rest of the body, and therefore great concern must be exercised to ensure that no infection occurs.

Secondly, ill-judged and unskilled efforts to obtain fluid may result in some cortical damage, if not actually in the production of a subdural haemorrhage itself.

Because the haematoma usually lies in the fronto-parieto-temporal regions, the puncture



Illustrations 6a and 6b.—A subdural puncture. The needle enters at the lateral angle of the fontanelle in a downward and lateral direction. After two or three millimetres the dura is pierced and the stillette withdrawn. Any further advance except in very large haematomata risks traumatising the cortex. (Illustrations by kindness of Miss Patricia Archer.)

should be performed through the lateral angle of the anterior fontanelle or the coronal suture. Only on rare occasions, if the haematoma is suspected of being elsewhere, should the location differ. With the skin well shaved and using full aseptic technique, a small lumbar puncture needle should be inserted at the lateral angle of the fontanelle in an inferolateral direction. The resistance of the dura mater will be felt at a depth of two to three millimetres, and as soon as this is penetrated the stillete should be removed. Aspiration with a syringe should not be practised and fluid should be allowed to drip slowly into a sterile container (illustration 6).

Sometimes, on tapping the subdural space, a quantity of clear fluid is obtained. This fluid may come from the subdural space or it may come from the subarachnoid space which has been accidentally opened. It is very unusual to be able to get more than about 1 c.c. of fluid from the subarachnoid space of a child without waiting a long time unless there is gross cerebral agenesis, so that if more than that amount is obtained it must be assumed to be coming from the subdural space.

The fluid should be immediately examined for cells and protein. A small quantity should be withdrawn for cell count and the rest should be centrifuged and the protein in the supernatant fluid estimated. This can then be compared with that of the cerebro-spinal fluid obtained in the lumbar region if there is cause for doubt as to its origin. Protein values as high as 11 gm. per cent. were reported by Merrit and Munro (1936) in adults. The highest value noted by us was 5.7 gm. per cent. (J.M.).

(b) *Post-Meningitic Group*

The signs and symptoms and associated findings of 115 cases in which a subdural haematoma or effusion complicated meningitis are recorded:

Table III

		Per cent.
69 cases	irritability	61
58 cases	persistent fever	53
34 cases	convulsions	35
31 cases	listlessness proceeding to coma	26
27 cases	focal signs	23
22 cases	vomiting	19
19 cases	bulging fontanelle	15
14 cases	increased head size	13
5 cases	dehydration	5
5 cases	hyperreflexia	4.3
5 cases	stiff neck	4.3
4 cases	anorexia	3.4

	Per cent.
4 cases opisthotonus	3.4
4 cases otitis media	2.7
2 cases chronically increased intracranial pressure	1.7
2 cases diarrhoea	1.7
2 cases chronic meningitis	1.7
2 cases blurred discs	1.7

In one of our four cases the symptoms were those of chronically increased intracranial pressure with aphasia after subsidence of the meningitis, while in the others there was a profound toxaemia, with convulsions in two cases and increasing fever with attacks of opisthotomus in the last one.

Failure of the disease to respond rapidly to treatment, shown by continuance of the fever, irritability or convulsions in about one-third of the cases, are the most important suggestive points. McKay, Ingraham and Matson believe that if the temperature does not subside within 72 hours, or if the symptoms have not made a substantial improvement before that time, the subdural space should be tapped for the presence of fluid.

We have felt, however, that the occurrence of repeated convulsions is an indication for subdural tapping, even before the 72 hours have elapsed and have been gratified to find that they have been rapidly relieved by the removal of fluid when present. On the other hand, if fluid is not found on the first occasion, but the child's condition remains poor, subdural taps should be repeated as indicated later.

In our cases fluid was found at periods varying between the third day (first day of admission) and the forty-seventh day. (In this latter case, L.B., fluid had clearly been present for some time, since membranes were present.)

Arnold (1951) has noted that the skin overlying the fontanelle may be inflamed, and the fact that a bulging fontanelle does not cease to bulge after lumbar puncture is very suggestive of the presence of fluid.

Retinal haemorrhages are not found in this group and fundi are usually normal, though blurring of the discs was found in two cases.

Focal signs, such as paresis, are present in the same percentage in both aetiologies, and since they are presumably directly related to pressure on the cortex, this is to be expected.

TREATMENT

(A) *Prophylactic.*—The incidence of those cases associated with birth trauma can and is being reduced by increasing standards of obstetrics and the maintenance of an adequate vitamin K intake by the mother.

(B) *Therapeutic.*—Our general routine of treatment is essentially similar to that recommended by Matson and Ingraham (1949) and has been as follows:—

(1) Early decompression by repeated subdural tapping, usually on alternate sides, on alternate days, removing between 10-20 c.c. of fluid on each occasion. If the fluid rapidly disappeared (after two or three tappings) and the child remained well, no further therapy was given.

(2) If fluid continued to form, and particularly if it became increasingly xanthochromic, taps would be continued until the child's condition became much improved, signs of active infection subsided and the child became fit for operation. Bitemporal trephine holes were then made and the fluid drained. If the child's condition were good enough and membranes were present, craniotomy would be performed on one side on that occasion, the trephine holes being so placed that they could easily be converted into a small temporo-parietal flap. In the infectious cases great care must be exercised to ensure that the inflammatory process has settled down and that all cultures have been sterile for some time, as infection of the bone flap or re-accumulation of fluid is likely to occur. If indicated, craniotomy on the other side would be performed one or two weeks later.

TRAUMATIC AND CRYPTOGENIC GROUP

Two patients were treated by subdural tap only. One was a 21-day-old hypertonic infant (E.H.), in whom initial taps produced 2 c.c. of fluid on the right side and ½ c.c. from the left side. Subsequent taps produced less fluid and, improving, he was discharged for observation and continued to do well. The second patient (T.F.P.) was admitted as an acute problem and did not do well, despite subdural tapping. His condition was too poor for surgery. Therefore only one patient did well on aspiration alone, and he clearly had only a small haematoma.

In seven cases operation had to be undertaken before conditions were judged optimum. Two of these were neonatal. Fluid did not drain well by subdural tap, largely owing to blood clots, and, the patients' condition deteriorating, surgical intervention was undertaken. In another (R.L.), surgery had been undertaken as an

emergency procedure, after subdural taps had been doubtful, on an infant in very poor condition who did not survive. The last case (R.G.—case report No. 2) had a very large chronic haematoma, which it was felt inadvisable to drain repeatedly because of the great protein and electrolyte loss involved. After diagnosis had been established by subdural tap, the membranes were removed through enlarged trephine holes. Despite the sub-optimal conditions, all these patients except one did well.

One patient was treated by aspiration and trephine only at the optimum time (K.R.). No membranes were present and she made an excellent recovery.

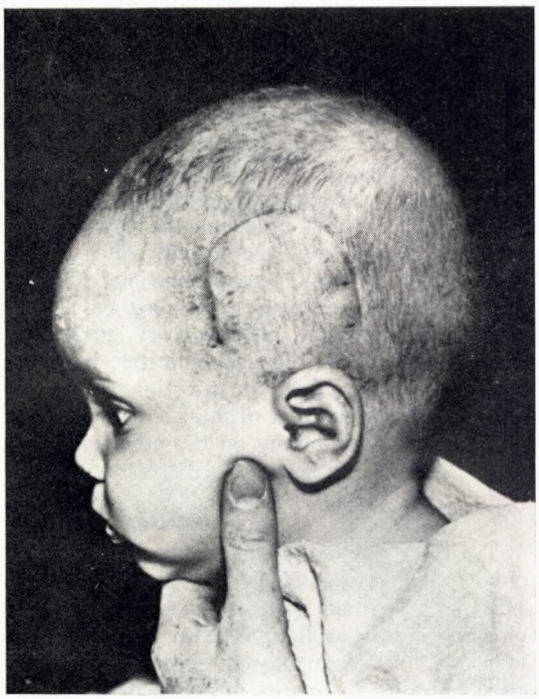


Illustration 7.—Wound immediately after removal of the stitches to show position.

Seven patients had repeated subdural taps and subsequently underwent craniotomy with removal of membranes. Two cases required unilateral craniotomy only; the rest required bilateral craniotomy.

In three patients the condition was diagnosed by bilateral trephine. In two, trephine alone was satisfactory; while in the other, bilateral craniotomy, with removal of the membranes, was necessary. These three patients did well.

A total of 11 unilateral or bilateral trephine operations and 18 craniotomies were performed on 18 patients. Seven patients had bilateral and four had unilateral craniotomies. One patient (R.L.) died in the immediate post-operative period. This gives an operative mortality of one death in 29 operations (3.4 per cent.) and a surgical case mortality of one case in 18 (5.5 per cent.). Of 20 cases, however, two died—an over-all case mortality of 10 per cent.

Of the 18 patients who survived, treatment was complicated in only one of them (A.J.), who developed a small area of necrosis of the scalp, necessitating a small plastic procedure.

Post-operatively, pyrexia was frequent, and every effort was made to keep the temperatures of such patients below 101.5° F. by the use of cold sponges, oxygen tents and, if necessary, ice water enemas. In no case did severe hyperthermia occur. No operation was commenced without a satisfactory intravenous infusion running, and this was maintained post-operatively until the haemoglobin had been checked and the infant was found to be taking fluids satisfactorily. In every case the infant had responded satisfactorily by the day following operation and was taking fluids by the second post-operative day (illustration 7).

Meningitic Group

Attention was directed in the first instance to the control of the infection, and for this purpose penicillin, gantrisin and chloromycetin were used. Intrathecal pancreatic dornase was used in two cases in an effort to cut down the organisation of fibrin. Subdural taps were performed on three patients. After the presence of fluid had been established the patients were tapped frequently, as before, to keep the intracranial pressure as low as possible and relieve the compressive effects of the fluid.

These were continued until either the production of fluid ceased or the patient's general condition was considered adequate to allow drainage by trephine and examination of the subdural space for the presence of membranes. If membranes were present, they were removed by craniotomy.

In one case (C.P.), instead of tapping the space repeatedly, a small polyethylene catheter was inserted into the subdural space through a spinal needle and connected to a drainage bottle. Drainage was not very good because of the tendency for the tube to curl up, and movements

of the child made it difficult to keep the whole apparatus sterile. However, it seems a worth while method if it will drain properly and saves the repeated traumata to the dura mater of subdural tapping (see case report No. 6).

The fourth case has already been described (case report No. 5).

Three unilateral or bilateral trephine operations, five craniotomies and one bone flap removed were performed on four patients. There was one death.

FOLLOW-UP STUDIES

Traumatic Group.—Of the 18 patients who were discharged from hospital, follow-up is complete to August, 1955, on all of them. The longest period is five years and ten months (J.M.) and the shortest three months (M.L.). One patient has since died of an unrelated cause (F.C.—pneumonia) and has been excluded from the series. He had, however, been well up to that time, with fewer seizures, but had been slightly retarded. Thus 17 are now available for study.

Thirteen have been classed as normal children. Four have some residual disability—three of them are mentally retarded (though two are greatly improved compared to their pre-operative state) and one has a hemiplegia, but is a bright child in all other respects. Therefore, of those 17 still living, 13 or 76 per cent. are normal, two or 12 per cent. are improved though still backward, and two or 12 per cent. have a residual neurological disability. This series of cases is compared with four other series similarly treated (Table IV).

Matson and Ingraham (1949) reported the largest series. They have had no hospital deaths since 1944 and have had only six deaths in 169 cases (mortality 2.9 per cent.). Five children died after discharge from hospital from causes unrelated to their disease. Seven had residual seizures, two were blind, two hemiplegic and two had facial weakness. They believe that over 70 per cent. are developing normally.

Statten (1948) presented an interesting and comparative series of cases, with one group treated prior to 1945 by haphazard and radical methods and one group treated subsequent to that date by the principles laid down by Matson and Ingraham. Of 13 cases treated in the first group, only three were known to be living and eight were dead—a mortality of 61 per cent. Of the group treated after 1945, ten were normal, three were retarded and two were dead. Two died before their condition could be adequately improved for surgery.

Good results speak for themselves. It is, however, of great importance to analyse the poor results and to determine the factors responsible. In our series two patients died; the first (R.L.) was an infant of three months and had been ill for only four days prior to admission. Subdural taps were not performed for two days and, when they were inconclusive, were not repeated for a further two days, by which time the fontanelle was very tense, she had developed spasticity of the upper arm and was almost moribund. Bilateral haematomata were drained through trephine holes as an emergency procedure, but the child died the next day. Autopsy was refused. It is possible this child might have

TABLE IV

Comparison of Five Series of Treated Subdural Haematomata in Infancy.
(Non-Meningitic Cases)

Series	*No. of Cases*	*Hospital and Disease Mortality*	*Deaths Unrelated to Condition and Post-Discharge*	*Number Living and Followed*	*Number of Normal Children*	*Number of Children with Some Mental or Physical Defect*
The author's	20	2 (10%)	1	17	13 (76%)	4 (24%)
Gutkelch	18	3 (16.6%)	—	15	11 (73%)	4 (27%)
Statten	15	2 (13%)	—	13	10 (79%)	3 (21%)
Matson and Ingraham	169	6 (2.9%)	5	158	Over 70%	Under 30%
Kinley, Riley and Beck	22	1 (4.4%)	—	18	13 (72%)	5 (28%)

been saved by more energetic treatment following the first subdural tap and serves to demonstrate the fact that there is little time to lose before decompression is commenced in the acute case. The second fatality in our series was also seen as an emergency problem at the age of seven months (T.F.P.). Subdural tap was not performed for seven days after admission, when the patient's condition was very poor. He died before surgery could be undertaken. It is impossible to escape the conclusion that had there been a greater awareness of the condition and treatment instituted sooner, the result might have been more satisfactory.

Four children have some residual disability. M. O'N. is mentally retarded and has poor vision. He had been vomiting since being taken home from hospital at birth and had recently had seizures and drowsiness. On the day of admission to hospital subdural taps were found to be positive bilaterally, but he did not drain well and intervention was forced after two days by his deterioration. After drainage of the right side a subdural tap on the left side was found to be negative and he was sent home.

Six weeks later re-admission was necessary for seizures and poor vision. Membranes were removed from the left side by craniotomy. It is possible that this visual defect, if not the mental retardation, might have been avoided by placing a left-sided trephine hole at the time of his first operation, instead of relying on a subdural tap. Although only six weeks elapsed between his first and second admission, this has been long enough for the development of permanent damage. The conservative treatment of the left side involved an accepted risk which, justified in the case of E.H., caused sequelae in this case.

Two other cases are mentally retarded. However, they are improved over their pre-operative condition, and it must be borne in mind that "one cannot make a silken purse out of a sow's ear," so that these cases must be classed as improved rather than failures of treatment.

B.A.S. has a residual hemiplegia. The consequences of her misdiagnosis have already been discussed.

Meningitic Cases

Three cases are alive and well in periods varying from three months to three years and three months. One patient has a residual disability (L.B.), in that the bone flap required removal after a second craniotomy. He awaits a plastic procedure at a later date. These cases cannot be compared with any other series except in so far as they form part of a group of cases of acute meningitis, and the results can only be properly assessed within the framework of such a group. Nevertheless a case such as L.B., where a residual aphasia only disappeared after membrane removal (there was no improvement between the time the fluid was drained off and the membrane removed) can only serve to improve the figures of any series of which they form a part.

The one case that died has been previously discussed.

Discussion—The Problems of Subdural Haematoma

Numerous problems remain unsolved in this rather peculiar lesion.

Whereas the aetiology in a number of cases is clear cut, either trauma or meningitis being the provoking factor, a large number of cases have no known aetiology, although hypoprothrombinaemia may be an important factor in many of them. Symptomatically, most of the latter cases resemble either the acute or the chronic condition, while sometimes the subdural fluid has a very low protein value and sometimes high values with thick membranes. In infants there is clearly some other factor rendering the infant skull liable to the production of the lesion, over and above the direct effects of trauma or infection.

During the course of the last century the origin of the membrane which appeared in adults and was then called pachymeningitis haemorrhagica interna, caused much discussion. Sperling and Wiglesworth believed that bleeding occurred first and was followed by organisation of the clot and production of the membranes, while Virchow believed that the bleeding occurred after membrane formation. Nowadays we know that an identical membrane may be formed, either by the organisation of a blood clot or of an inflammatory exudate. The only difference between them is the greater tendency towards duro-arachnoidal adhesion in the inflammatory group.

It is by no means clear, at the present moment, what is the difference between the subdural space and other tissue spaces in relation to the absorption of blood. In the majority of cases of extravasation elsewhere, even into the pleural

cavities, it is rare to find organisation of any but the biggest haematomata, and nowhere do we observe the steady increase in size seen so regularly in the subdural haematomata.

Whereas the theory of osmotic attraction appears to provide a satisfactory explanation for the increase in size of the traumatic group, it can scarcely be the only factor in the inflammatory variety. Gitlin's and Crosby and Bauer's observations on subdural fluids tend to suggest that both types of subdural fluid (traumatic and inflammatory) have an exudative element and the very close histological resemblance of the membrane produced in both groups imply that the mere presence of blood within the subdural space is irritating and sets up an inflammatory reaction with outpouring of fluid and invasion of the clot or exudate by phagocytic cells. Indeed, it is only on the basis of the perpetuation of the inflammatory reaction that one can account for the occasional persistence in the production of fluid, even after complete surgical drainage of the space, and sometimes even after the complete removal of the membranes.

In one of our traumatic cases (A.R.) aspiration was necessary several times after an operation for removal of membranes which had been left quite dry, to evacuate accumulating fluid which bulged the skin over the trephine holes around the bone flap. It seems that the old and rather mechanical theory of osmotic pressure must be considered outmoded, and whereas this undoubtedly plays a large part in the process, we have now to think of it as a combination of osmotic pressure and inflammatory reaction together.

Convulsions are not a common symptom in subdural haematoma in adults, yet they occur as a presenting symptom in over 50 per cent. of cases in the mixed and in 35 per cent. of the meningitic group. Presumably that is due to increased irritability of the infant cortex compared to that of the adult, since removal of the fluid by tapping frequently results in a remarkable amelioration of the patient's condition. In the majority of our cases suffering from seizures a left-sided seizure was related to a right-sided clot, and vice versa, while bilateral or generalised convulsions were usually found when bilateral lesions were present. There are many cases of convulsions occurring soon after birth, many of which are not amenable to treatment; nonetheless the possibility of this eminently curable lesion being the basic cause should always be borne in mind and the proper diagnostic measures taken to exclude it.

Persistent fever was found in 53 per cent. of the post-meningitic cases and 35 per cent. of the traumatic cases. This tends to support the thesis that even though the primary bleeding may be caused by trauma in certain cases, the subsequent reaction of the meninges is inflammatory in both cases, with fever following thereon and producing a similar pathological picture.

Much discussion has developed around the significance of the subdural membrane and the necessity for its removal. Few surgeons feel that the membranes are of any significance in the adult unless they are so thick that the subdural space does not collapse when the fluid is removed; but in the infant, on the other hand, many feel that their removal, especially of the inner membrane, is essential if brain damage is not to occur. It is held that the membrane does not grow at the same rate as the skull and brain itself and stretches across the vault of the skull like the string of a bow, compressing the underlying brain. Sherwood's and Naffziger and Brown's series, which did not do particularly well on drainage alone, and the good results obtained by craniotomy also, have led us to agree that in the majority of cases removal of the membranes should be carried out whenever they are present. On the other hand, Everley Jones (1952) has presented a series following meningitis in which craniotomy was not carried out on any patients, and states that all are doing well. Under these circumstances one has to agree that the question as to which patients should be subjected to radical surgery and which should not is as yet *subjudice*. From our own experience we have felt that when membranes are present, better results are obtained when they are removed than when, if of any thickness, they have been left *in situ*.

On the other hand, there can be no question regarding the advisability of removing fluid when it is present, even though in a number of the meningitic cases it may be reabsorbed as the inflammation subsides. The presence of fluid causes pressure on the brain, and the remarkable alleviation in symptoms obtained on tapping the subdural space bears ample testimony to the value of the procedure. In addition, removal of the fluid removes some cells and fibrin, all of which are likely to contribute towards the presence and thickness of the membranes.

Appendix A SUMMARY OF CASES

Initial	*Age and Sex*	*Birth and Neonatal History*	*History of Present Illness*	*Symptoms and Signs*
P.D.	6 days. Female.	Spontaneous normal delivery; 2 hr. labour.	Patient was well from birth until day of admission.	Left-sided convulsion; vomiting. Head 36 cm. Wide suture. Rt. pupil larger than lt.
M. McS.	2 days. Female.	24 hr. labour; forceps for foetal distress.	Cyanosed since birth; artificial respiration and oxygen supplied.	Cyanosis. Bruise over right eye. Hyperreflexia. Dehydration.
C.B.	12 months. Female.	Injury to nerves of arm during delivery; bruised by forceps.	Patient hit head one week prior to admission and again three days p.t.a. Vomiting and convulsions after second blow.	Left-sided convulsions. Projectile vomiting. Hyperreflexia.
E.H.	21 days. Male.	Normal.	Vomiting since soon after birth. Irritable at all times. Hypertonic.	Vomiting. Hypertonicity. Irritability. Hyperreflexia.
R.L.	3 months. Female.	Spontaneous normal delivery. Normal neonatal course.	Well until four days prior to admission.	Convulsions. Drowsiness. Fever. Fontanelle normal. Skull flattened posteriorly. Nasal congestion.
T.F.P.	7 months. Male.	Membranes ruptured one week prior to delivery. Labour lasted one hour. Post-natal course normal.	Has always been sleepy. Never ate much. Never lifted head. Did not grasp objects.	Sleepy. Does not play, grasp things, lift head. Is a poor sucker and has a poor appetite. Wide awake during exam. and hyperactive. High-pitched cry. Head 42 cm. Fontanelles closed. Hyperreflexia.
S.L.	3 months. Female.	Forceps, but perfectly normal after.	Hit head one week and again three days before admission.	Screaming. Vomiting. Irritable. Semi-comatose. Convulsions. Fontanelle full. Fixed pupils; eyes to right. Hyperreflexia. Haemorrhages in fundi and vitreous. Left ankle clonus.
M.A.R.	5½ months. Female.	Normal.	Well until 2½ weeks p.t.a. when spanked. Went limp and then spastic. Hospitalised; no diagnosis.	Convulsions 2½ weeks ago and again after similar trauma on day of admission. Irritable. Bulging fontanelle. Internal strabismus. Otitis media. Right patellar jerk absent. Brudzinski positive.

TRAUMATIC

Subdural Tap Spinal Tap	*Treatment*	*Operative Findings*	*Progress*	*Follow-up and Result*
Subdural tap: Right 8 c.c. Left dry. Spinal tap: Xanthochromic, 51 mg. protein per cent.	Patient's condition became very poor despite subdural taps. Head enlarged to 37.5 cms. Surgery forced because of poor state.	Right enlarged trephine. Numerous clots. No membranes.	Very good after drainage established.	3¼ years post-op. One seizure during febrile attack, otherwise normal.
First subdural tap negative; second positive. Left 4 c.c. Right 7 c.c. Spinal tap, protein, 42.6 mg. per cent.	Patients condition became very poor so that trephination became essential.	Left trephine. Numerous clots. No membranes. Right side later negative.	Very good after drainage established.	3½ years post-op. Normal child.
None.	Bilateral trephine.	Bilateral trephine. Clots on right side. No membranes. Left normal.	Very good after drainage established.	3 years and 1 month post-op. Normal child.
Subdural tap: Right 3 c.c. Left ½ c.c. Spinal tap not done.	No fluid obtained after two subdural taps. Child discharged for observation.	No operation.	Very good after 2 taps.	3¾ years post-op. Normal child.
Subdural tap: Left 2½ c.c. clear fluid. Protein 9 mg. per cent. Spinal tap: 10 wbc, 1,000 rbc Pandy. pos.	Subdural taps were unsatisfactory and the child's condition became precarious. Bilateral trephination was forced.	Bilateral trephine showed bilateral clots but no membranes.	Died 1 day after surgery.	Dead.
Subdural tap: Right 20 c.c. xanthochromic fluid. Spinal tap: protein 30.6 mg. per cent.	Patients condition became rapidly worse and was not relieved by subdural tap. He went down hill and died 7 days after admission. Right side only contained fluid.	No operation.	Died 1 day after establishment of diagnosis and drainage.	Dead.
Pos. bilaterally.	Patients condition not adequately improved by subdural taps. Trephine essential.	Bilateral. Blood clots. No membranes.	Very good after drainage.	Three months later normal.
Subdural taps positive bilaterally. Spinal tap: elevated pressure, protein 227 mg. per cent., wbc 132.	Improved on subdural tapping. Later had craniotomy.	Membranes with bilateral haemangiomata of the cerebral hemispheres.	Very good after establishment of drainage.	3¾ years post-op. Well, but has internal strabismus.

Appendix A

SUMMARY OF CASES

Initial	*Age and Sex*	*Birth and Neonatal History*	*History of Present Illness*	*Symptoms and Signs*
K.R.	5 months. Female.	Labour induced because of full term. Forceps and scars therefrom. Neonatal course normal.	Patient was well until 10 a.m. on day of illness, when she had a spasm lasting 3-5 mins. She became cyanotic and pale. Had three more attacks that day.	Convulsions. Lethargy. Soft fontanelle. Fundi normal.
B.A.S.	10 months. Female.	Normal delivery. Postnatal condition normal.	Had a convulsion following fever on day of first admission. Dieg-febrile convulsions. Re-admitted later with hemiplegia.	Convulsion. Hemiplegia (second adm.). Pupils constricted and not reacting. Semi-comatose. Twitching right lower limb. Hemiparetic.
Y.G.	6 months. Female.	Premature birth.	Enlarging head and retinal haemorrhages seen at three weeks.	Vomiting. Convulsions. Large head. Retinal haemorrhages. Hyperreflexia. Open fontanelle. Does not sit up.
M. O'N.	8 weeks. Male.	Caesarian section. B. wt. 8 lb. 9 oz. Irritable since taken home.	Patient cried continuously since coming home. Was very irritable and fretful, and one day he had a seizure; head and eyes turned to the right.	Continuous crying. Seizures. Drowsy hyperreflexia. Fontanelle not bulging. Mild U.R.I.
J.M.	6 months. Male.	Normal delivery twin. B. wt. 4 lb. 12 oz.	Spat up food from time of going home. Had a convulsion one week p.t.a. Vomits 15 minutes after feeding; projectile.	Seizures. Vomiting. Failure to gain weight; larger head than twin. Irritable. Visible scalp veins. Multiple retinal haemorrhages. Ant. font. 3 fingers, soft.
A.R.	12 months. Male.	Normal birth.	Fell two weeks p.t.a. Unconsciousness.	Vomiting. Lethargy. Anorexia. Irritability. Convulsions. Left internal strabismus. Retinal haemorrhage. Head increased in size.
R.G.	5 months. Male.	Delivery three weeks before term. Normal. Difficulty in eating since birth.	Occasional difficulty in eating since birth. Vomiting projectile; two months. Had a convulsion one month ago.	Projectile vomiting. Sits with support only. Difficulty in eating. Convulsions. Enlarged head. Anterior fontanelle and sutures open and bulging. Fundi normal. Mentally retarded.
J.F.	2½ years. Male.	Born at seven months. Labour induced and lasted 13 hrs. Difficulty in breathing; 24 hrs.	Retarded development. Talked 1½ years. Walked 23 months.	Retarded development in walking and talking. Makes noises but no words. Small head. No abnormal neurological findings.

TRAUMATIC—(*Continued*)

Subdural Tap Spinal Tap	*Treatment*	*Operative Findings*	*Progress*	*Follow-up and Result*
Subdural taps positive bilaterally. Protein 141 mg. per cent. Spinal tap: protein 18.5 mg. per cent.	Patient improved on subdural tapping alone. Bilateral trephine holes performed to allow drainage and establish the presence of membranes.	20 c.c. of fluid on right, none on left. No membranes.	Very good after establishment of drainage.	3¾ years post-op. Normal child.
Subdural tap not done. Spinal tap: I.P. 290 cm. of fluid. Protein 18.5 mg. per cent.	Patient's condition demanded immediate treatment. Left-sided trephine holes inserted. Right-sided trephine holes made subsequently, negative.	Bloody fluid with necrosis of temporal and frontal lobes.	Good after establishment of drainage. Hemiplegia persisted.	4¼ years post-op. Well. Residual hemiplegia improving.
Positive bilaterally.	Repeated subdural taps, followed by bilateral craniotomy.	Bilateral membranes.	Very good.	Three months post-op. Normal child.
Subdural tap positive bilaterally. Protein 900 mg. per cent. No spinal tap.	Had several subdural taps but did not improve, in fact convulsions became worse and drowsiness more marked. Craniotomy forced because of degeneration.	Right fronto-temporal flap-fluid subdural. Haematoma drained. No membranes. Left side done 1½ months later, demonstrating membranes.	Fair after second admission.	3¾ years post-op. Retarded. Poor vision.
Subdural tap bloody on right with xanthochromic. Negative on left. No spinal tap.	Repeated subdural taps until dry. Craniotomy for removal of membranes. Left side dry. Right always prolific.	Right craniotomy. Thick membranes.	Good after drainage established.	5¾ years post-op. Normal child.
Positive bilaterally.	Subdural taps followed by bilateral trephine when fluid re-accumulated. Left craniotomy with removal of thin membranes.	Membranes on left side.	Very good after membrane removal.	Eight months later normal.
Subdural tap positive bilaterally. Protein 2.98 gm. per cent. Spinal tap not done.	Bilateral craniotomy (enlarged trephine holes) after diagnosis established.	Bilateral thick membranes.	Very good after craniotomy.	Much brighter child one year ten months later. Slightly retarded.
No subdural tap. Spinal tap: Protein 14 mgm. per cent. WBC 23/cu.mmc.	After diagnosis was suspected on PEG, bilateral trephine holes made for confirmation, followed by craniotomy.	Bilateral thin membranes.	Did well after craniotomy.	Much brighter child than pre-op. one year eight months later.

Appendix A

SUMMARY OF CASES

Initial	*Age and Sex*	*Birth and Neonatal History*	*History of Present Illness*	*Symptoms and Signs*
M.Z.	2 months. Female.	F.T. forceps delivery. In oxygen for three days. B. wt. 9 lb. 11½ oz.	Vomited after each feeding since birth. Also gets attacks of clenching her fists and shaking.	Vomiting. Fontanelle depressed. Assymetrical head.
F.C.	3 months. Male.	Breech presentation; labour 4½ hrs. Neonatal course good.	Convulsions two weeks p.t.a. which lasted 1½ minutes and recurred five minutes later. Hospitalised elsewhere; no diagnosis.	Convulsions. Hyperreflexia. Fontanelles soft. ? Blind.
D.G.	3 months. Male.	Normal spontaneous delivery; labour 2 hrs. Neonatal period normal.	Was well until two days p.t.a., when he had a generalised convulsion; with eyes rolling up and extremities trembling, recurring q. 3h c cyanosis.	Convulsions. Head flattened on left side. Ant. Font. open. Pupils assymetrical, left/right ankle clonus during convulsion. Developed left Hemiplegia.
A.J.	9 months. Female.	Ten months pregnancy; 24 hrs. labour.	Very little gain in weight since birth. Frequent vomiting.	Vomiting. Failure to do well. Fever. Acute and chronically ill. Tense fontanelle. Kernig positive. Ears injected.

Appendix B

Initial	*Age and Sex*	*History*	*Signs and Organisms*	*Indications for Subdural Tap*
E.P.	6 months. Male.	Cough three days. Fever two days. Irritability. Vomiting. Paresis of right side. Convulsion one day.	Irritable. Stiff neck. Bulging fontanelle. Hyperreflexia. Semi-comatose. Right hemiparesis. Bilateral Babinski. Organism, *B. haemolytic strep.*	Bulging fontanelle. Convulsion. Hemiparesis.
C.P.	7 months. Male.	Rhinitis. Fever. Irritability.	Acutely ill. Lethargic. Cough and dullness Right base. Stiff neck. Grunting respiration. Organism, *H. influenzae.*	Increased fever. Opisthotonus.
R.P.	2 months. Male.	Runny nose. Temp. elevated three days. Lethargic.	Full fontanelle. Organism, pneumococci.	Bulging fontanelle. Convulsions.
L.B.	3¼ years. Male.	Fever three days. Convulsions three days. Lethargic.	Comatose. Spastic facial reactions. Organism unknown.	None performed.

TRAUMATIC—(*Continued*)

Subdural Tap Spinal Tap	*Treatment*	*Operative Findings*	*Progress*	*Follow-up and Result*
Subdural taps positive bilaterally. Spinal tap bloody. Proteins 817 mgm.	Bilateral subdural drainage by tapping until condition satisfactory for craniotomy.	Bilateral membranes. Large sylvian vessels on left side. Right side, outer membrane only seen.	Did well after craniotomy.	One year six months post-op. Very active; acts like normal child.
Subdural taps positive bilaterally. No spinal tap.	Bilateral subdural taps performed but were insufficient and trephine operation forced by patient's poor condition. Discharged but re-admitted and after subdural tap positive on left side, craniotomy performed.	Membranes.	After removal of membranes, patient was much improved.	Died after discharge from pneumonia. Excluded from follow-up. Dead.
Subdural taps positive bilaterally. Protein 98 mgm. per cent. Spinal tap normal.	Had subdural taps, but these were inadequate and bilateral trephine holes were made because of increasing seizures and paresis. Later had left craniotomy.	Thin membranes on left side.	Did well after craniotomy.	Two years post-op. Good. Normal child.
Subdural tap positive bilaterally.	Repeated subdural taps to reduce pressure. Right and left craniotomy to remove membranes.	Bilateral membranes Arteriovenous angioma over right side.	Good after craniotomy.	Normal child one year seven months later.

MENINGITIC GROUP

Day of Illness. Taps First Performed	*Day of Taps First Positive*	*Subdura Fluid Culture*	*Treatment*	*Operative Findings*	*Progress and Result.*
Third day.	Third day.	Negative.	Antibiosis. Subdural taps. Bilateral craniotomy.	Membranes bilaterally; much fibrin on left side.	Steady improvement after first subdural tap and antibiotics. Normal child three months later.
Fourth day.	Seventeenth day.	Negative.	Antibiosis. Subdural taps. Pancreatic dornase intrathecally. Left trephine. Right craniotomy.	Thin membranes right side.	Improved after first positive tap. Good. Normal child four months later.
Six days.	Fourteen days.	Negative at first. Later *H. para flu B.*	Antibiosis. Pancreatic dornase, blood transfusion, subdural taps and trephine.	Bilateral fluid right membranes.	Died four days post-op. Much inflammatory material in basal regions. Bilateral encapsulated haematomata beneath anterior fontanelle.
—	—	—	Subdural haematoma disclosed at venticulography for increased intracranial pressure and aphasia.	Bilateral membranes.	Left bone flap infected and removed. Aphasia disappeared. Doing well three years post-op.

REFERENCES

ANDERSON, F. M. (1952). *Pediatrics*, 10, 11.

ANGLIN, G. S., MACNAUGHTON, G. A. & SILVERTHORNE, N. (1952). *Canad. med. Assoc. J.*, 6, 435.

ARNOLD, G. G. (1951). *J. Ped.*, 39, 191.

——— (1952). *J. Ped.*, 40, 757.

BLOOR, B. M., GRANT, R. S. & TABRIS, S. A. (1950). *J. Amer. med. Assoc.*, 142, 241.

CHAMBERS, T. R. (1925). *J. med. Soc. New Jersey*, 22, 209.

CRAWFORD, R. (1935). Commenting on a paper by Sharpe, W. *J. Amer. med. Assoc.*, 104, 959.

CRITCHLEY, M. & MEADOWS, S. P. (1932-33). *Proc. Roy. Soc. Med.*, 26, 306.

COBLENTZ, R. G. (1938). *Surgery*, 4, 194.

——— (1940). *Surgery*, 8, 771.

COPPOLETTA, J. M. & WOLBACH, S. B. (1933). *Am. J. Path.*, 9, 55.

CROSBY, R. M. & BAUER, R. E. (1956). *J. Neurosurg.*, 13, 140.

DAVIDOFF, L. M. & DYKE, C. G. (1938). *Bull. Neurol. Inst. New York*, 7, 95.

DE LA VILLA, L. (1942). *Rev. Clin. Espan.*, 7, 158.

DOWMAN, C. E. & KAHN, E. A. (1942). *South. Surgeon*, 11, 154.

ELVIDGE, A. R. & JACKSON, I. (1949). *Am. J. Dis. Child.*, 78, 635.

FINKELSTEIN (1904). *Berl. Klin. Wochnschr.*, 41, 403.

GARDNER, W. J. (1932). *Arch. Neurol. et Psychiat.*, 27, 847.

GITLIN, D. (1955). *Pediatrics*, 16, 345.

GOLDHAHN, J. (1930). *Deutsche Zietschrift fur Chirugie*, 224, 323.

GUTHKELCH, A. N. (1953). *Brit. med. J.*, 31st Jan., 233.

HANKINSON, J. & AMADOR, L. V. (1956). *Brit. med. J.*, 21st July, 122.

HERTER, J. (1898). *Am. J. Ment. Sc.*, 116, 202.

HERTZBERGER, E., ROTEM, Y. & BRAHAM, J. (1956). *Arch. Dis. in Childhood*, 31, 44.

HOOD, P. N. & PERLSTEIN, M. A. (1955). *Pediatrics*, 16, 470.

INGRAHAM, F. D. & HEYL, H. L. (1939). *Amer. Med. Assoc.*, 112, 198.

INGRAHAM, F. D. & MATSON, D. D. (1949). *Advances in Pediatrics*, IV, *Interscience*.

JONES, H. E. (1952). *Lancet*, 3rd May, 891.

KINLEY, G., RILEY, H. D. & BECK, C. (1951). *J. Ped.*, 38, 667.

KORWITZ, H. L. (1914). *Virchows Arch. f. path. Anat.*, 215, 233.

LAUDIG, G. H., BROWDER, E. J. & WATSON, R. A. (1941). *Annals Surg.*, 113, 170.

LEARY, T. (1934). *J. Amer. med. Assoc.*, 103, 389.

MARINACCI, R. & MARINACCI, G. (1951). *Bull. Los Ang. Neurol. Soc.*, 16, 253.

MCGUINESS, F. G. (1943). *Canad. med. Assoc. J.*, 48, 206.

MCKAY, R. J., INGRAHAM, F. C. & MATSON, D. D. (1953). *J. Amer. med. Assoc.*, 152, 5.

MCLEAN, J. A. (1953). (Personal communications.)

MENEGHELLO, J. & AGUILO, C. (1951). *Rev. Chilen. De Ped.*, 23, 91.

MORELLO, A. & LEVY, L. F. (1956). *Minerva Pediatrica*, Anno VIII, 6 (February), 30.

MUNRO, D. & MERRITT, H. (1936). *Arch. Neurol. et Psy.*, 35, 64.

NAFFZIGER, H. C. & BROWN, H. A. (1934). *S. Clin. North America*, 14, 1465.

OLDBERG, E. (1945). *Medical Clinics of North America*, 29, 62.

ORMISTON, G. (1956). *Brit. med. J.*, 21st July, 126.

PEET, M. M. & KAHN, E. A. (1932). *J. Amer. med. Assoc.*, 98, 1851.

PENFIELD, W. G. (1923). *Am. J. Dis. Child.*, 26, 383.

PETERMAN, M. G. (1943). *Diseases of the Nervous System*, 4, 307.

PUTNAM, T. J. & CUSHING, H. (1925). *Archives of Surgery*, 11, 329.

ROSENBERG, J. (1921). *Ergebn. d. inn. Med. u. Kindern.*, 20, 549.

——— (1913). *Berl. klin. Wochnschr.*, 50, 2272.

SCHWARTZ, C. W. & COLLINS, L. C. (1952). *The Skull and Brain Roentgenologically Considered*. C. C. Thomas, Springfield, Ill., 110.

SHARPE, W. & MACLAIRE, R. (1925). *Surg. Gynaec. Obstet.*, 41, 583.

SHERWOOD, D. (1930). *Am. J. Dis. Child.*, 39, 980.

SMITH, M., DORMONT, R. & PRATHER, G. (1951). *Pediatrics*, 7, 34.

SPILLER & MCCARTHY (1899). *J. Nerv. & Ment. Dis.*, 26, 687.

SPITZ, E., POLLACK, A. & ANGRIST, A. (1945). *Arch. Neurol. et Psy.*, 53-54, 144.

STATTEN, TAYLOR (1948). *Canad. Med. Assoc. J.*, 58, 63.

TROTTER, W. (1914). *Brit. J. Surg.*, 2, 271.

ZOLLINGER, R. & GROSS, R. E. (1934). *J. Amer. Med. Assoc.*, 103, 245.