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Subdural Hematomas in Infants

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INDEX

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I. SUBDURAL HEMATOMAS IN INFANTS

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The purpose of this report is to evaluate the problems presented by subdural hematomas in infants. These problems principally include an investigation into the etiology, pathology, the proper therapy, and the prognosis. There seems to be a distinct difference between those hematomas secondary to trauma and those secondary to inflammation. When surgically exposed there is much variation in these hematomas and they respond to treatment in a variable manner. Likewise the necessity of surgical attack on all hematomas can be questioned.

In order to help evaluate these variations in subdural hematomas, an attempt was made to obtain follow-up data on the 39 children treated during infancy for subdural hematomas between the years 1942 and 1951 at the University Hospitals. For indispensable cooperation, the authors are grateful to the Department of Pediatrics and to Child Psychiatry.

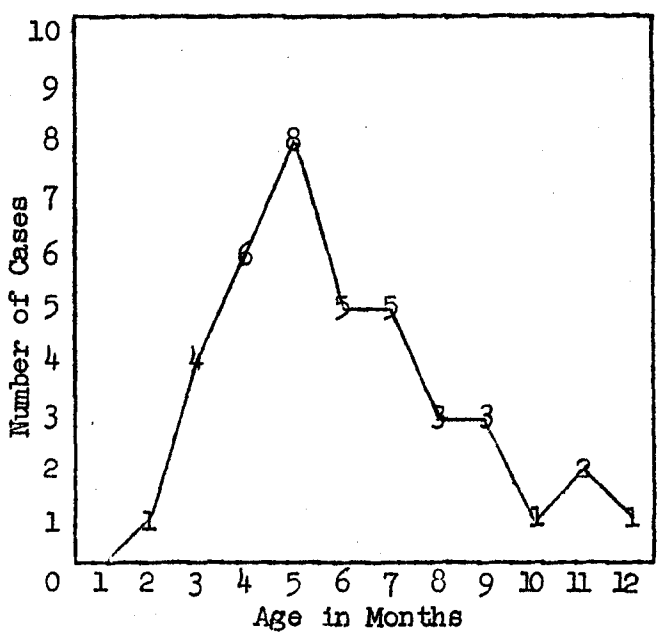
INCIDENCE

There are few reports in the literature on subdural hematomas in infants prior to 1930 when Sherwood[26] reviewed nine cases. Two years later, Peet and Kahn[23] also reported nine cases. Following this there were occasional articles (Naffziger and Brown[21], Coblentz[2], Rogatz[25], Dowman and Kahn[4]); but, it was not until 1944 when Ingraham and Matson[14] reported 98 cases that the lesion became clinically well recognized and delineated. Subsequently, Statten[31] in 1948 reported 28 cases and Elvidge and Jackson[6] in 1949 reviewed 55 cases. The 39 cases included in this report were encountered over a ten year period from 1942 to 1951. Thirty-five of these occurred during the five year period from 1946 to 1951. In the previous five years only four cases were encountered. This increased frequency of detection of subdural hematomas in infants is attributed to greater cognizance of the lesion and the routine inclusion of it in the differential diagnosis of pediatric problems. It has been said that the frequency of subdural hematomas in infants is largely proportional to the intensity with which they are sought[14].

The majority of cases reported in the literature not only have been in children under one year of age but most of them have occurred in children under the age of six months (Ingraham and Matson[14], Statten[31]). Figure I shows the age incidence of the thirty-nine cases included in this report. The youngest patient was two months old and the oldest twelve months. The average age was six months. The majority were detected between the third and seventh month of life.

FIGURE I

Age frequency of Subdural Hematomas in Infants.



ETIOLOGY

Virchow[33] in 1857 gave the first clear

description of the structure of subdural hematomas and of the encompassing membrane. He erroneously concluded the condition was primarily an inflammatory reaction of the dura with subsequent organization of the inflammatory membrane; and, secondarily, hemorrhage occurred into this organized area producing the hematoma. This was the classical pachymeningitis hemorrhagica interna chronica. Finkelstein[7] in 1911 described the histopathology of six cases but also related the cause to infection. However, Leary[17] in 1934 extensively restudied the problem and concluded that trauma, sometimes often very minor, rather than infection, was usually the inciting cause of the hematoma. Ingraham and Heyle[12] in 1938 agreed that trauma was undoubtedly the basic causative factor but postulated that poor nutrition was certainly a predisposing condition. Ingraham and Matson found in almost 50 per cent of their patients evidence of infection somewhere in the body at the time of hospitalization. This usually was of the respiratory, gastro-intestinal, or urinary tract. They felt, however, that this might only be a manifestation of the poor health associated with subdural hematomas. Ingalls[10] reported on eight infants with subdural hematomas and all had evidence of scurvy. For several years it was felt that scurvy, Vitamin K deficiency, or any other diseases giving rise to a bleeding diathesis, increased the possibility of clot development. According to Heilig et al[9] this is a logical deduction which is difficult to disprove; but, they pointed out that in Ingall's cases, no vitamin C levels were obtained and that the malnutrition may well have been an effect rather than the cause of the hematoma. They also point out that recent evidence indicates that even when maternal ascorbic acid levels are low, fetal levels are almost invariably maintained, at least in the neonatal period.

In over 50 per cent of Ingraham and Matson's cases there was a definite history of trauma and in 30 per cent of their cases the trauma was sustained at birth. This high frequency of birth trauma, as a cause of subdural hematomas, was observed also by Statten[31] and by Chase[1]. Statten reviewed 28 cases and also concluded that birth injury was the most common form of trauma. Overriding of the parietal bones during the moulding of the head as it passes through the pelvis causes tearing of the poorly supported cerebral veins as they enter the fixed sagittal sinus. Chase, in a thorough anatomical study of the meninges and their relationship to the brain, quite convincingly demonstrated that moulding of the infant's head during delivery produced tension on the intracranial soft parts, rupture of the delicate veins bridging from the dura to the cortex, and laceration of the falx and tentorium along with the contained vessels, with production of the hematoma. Rupture of vessels in midline structures such as the falx or tentorium may explain the frequent bilaterality of the hematomas in infants. He stressed that tentorial splits are more frequent in premature than in full term infants partly because of the greater immaturity of the fibers of the dural septa in prematurity. Five of Statten's 28 cases were in premature infants. He felt prematurity to be a contributory cause because the poorly developed bone failed to give proper protection to the underlying structures and also because the dura mater is more friable and vascular in premature infants.

Postnatal trauma as a cause of subdural hematomas in infants undoubtedly is more frequent than elicited in the history since they are so frequently due to minor injury. A blow on the head by a small rapidly moving object may produce a lacerated scalp or a depressed skull fracture but seldom does it produce a subdural hematoma. However, when a child's head hits the floor or other hard object, there may be a general shifting of the cranial contents forward or backward with tearing of the bridging veins to produce a hematoma. This type of injury happens so often to children that it is not even recorded in the history.

Subdural effusion of xanthochromic

fluid may result from acute bacterial meningitis (McKay et al[18], Smith et al[28], Steinberg and Murphy[30]). The exact mechanism for such effusions is difficult to explain. In many respects these subdural effusions in meningitis are similar to subdural hematomas. The majority of these effusions have been observed with meningitis due to H. influenzae but some of those reported in literature have been associated with infections due to N. meningitides, Ps. aerugenosa, D. pneumoniae, Salmonella and paracolon bacilli (Smith et al)[28].

In this series of 39 patients, 7 of the mothers stated they had difficulty during pregnancy or at the time of delivery. In two of the seven, Caesarean sections were performed because of an infant-pelvic disproportion. Four of the patients were one of twins. In five patients, there was a definite history of postnatal trauma. In 10 infants, there was evidence of an infection other than in the central nervous system; and, in four, there was a history of antecedent central nervous system infection. In none of the children was a vitamin deficiency apparent clinically nor was there evidence of vitamin deficiency by laboratory tests although tests were carried out in only a few of the 39 patients.

FIGURE II

Possible etiologic factors in patients with subdural hematomas.

History or findings of infections other than C.N.S.		10
otitis media	3	
pneumonia	1	
measles	1	
upper respiratory infection	5	
C.N.S. infection		4
influenza meningitis	2	
pneumococcal meningitis	1	
etiology?? meningitis	1	
Postnatal trauma		5
Difficulty during pregnancy		7
One of twins		4
Negative history		9
TOTAL		39

PATHOLOGY

Following a traumatic hemorrhage into the subdural space there is no evidence that the arachnoid or dura can bring about any appreciable degree of absorption (Leary)[17]. However, an attempt by the body is made to wall off the resultant clot from the dura and arachnoid (Putnam and Cushing[24], Cohn[3], Wertheimer and Dachaume[34]). During the first or second week after the hemorrhage fibroblasts together with new capillaries grow out from the undersurface of the dura and invade the blood clot producing a layer of granulation tissue. Histiocytes enter and begin to carry away debris from the breakdown of cells in the clot. During this stage the membrane is adherent to the dura and any attempt to separate it results in considerable oozing due to rupture of these new capillaries (Ingraham and Matson[12]). Gradually the membrane becomes firmer and more well established. The fibroblasts continue to proliferate, lay down collagen, and mature. Some investigators feel this prolification from the dura extends to eventually surround the clot but others (Putnam and Cushing[24]) feel that the inner membrane differs histologically from the outer and is produced by cells growing out from the arachnoid. It is true that the inner membrane is less adherent to the arachnoid and is more of a thin mesothelial membrane than the outer, adherent, dense fibroblastic membrane but whether they have different sites of origin is unclear.

As breakdown of the center of the clot continues a fluid high in protein content is formed. As the molecules of this fluid break down, through Donnan's equilibrium theory, more fluid is drawn through the permeable membrane into the center of the clot thereby gradually enlarging the clot (Zollinger and Gross[35], Gardner[8]). This expanding intracranial mass elevates the intracranial pressure.

Internal hydrocephalus is often found in association with subdural hematomas. This is probably due (Jackson and Werner[15], Elvidge and Jackson[6], Penfield[22]) to blockage of the areas of cerebrospinal

fluid absorption.

Subdural effusions occuring with bacterial meningitides have a different appearance at the time of operation than those apparently due to trauma. The enclosed fluid is apt to be less blood tinged, less xanthochromic. It may be somewhat opalescent due to a higher white cell count. The fluid often is loculated in numerous smaller cysts within the pia-arachnoidal membrane. The inner membrane is adherent to the arachnoid and pia and often to the cortex of the brain. Attempts to separate such an adherent membrane from the cortex may lead to tearing of the latter. The outer membrane usually can be separated easily from the dura. Microscopic sections of the membrane reveal granulation tissue infiltrated by clumps of white cells. The membrane is continuous with and may completely enmesh the arachnoid and pia and extend into the cortex. It is believed that effusions occur into the space between the membranes in much the same manner that the fluid increases in amount in subdural hematomas secondary to trauma.

SYMPTOMS AND SIGNS

There is no absolutely characteristic clinical picture of subdural hematomas in infants but there are certain symptoms and signs that should make one alert to the diagnosis. The commonest of these are enlarging head, bulging fontanelle, convulsive seizures, vomiting or persistent feeding problem, irritability or undue drowsiness and paresis of extremities. The triad emphasized by Ingraham and Matson[14] should make one immediately suspicious of a hematoma. (1) Failure to gain weight normally, refusal of feedings, hyperirritability or irregular temperature swings, accompanied by (2) an abnormally increasing head circumference, and (3) history of difficult labor or delivery.

The symptoms and signs presented by this series of 39 patients are shown in chart 3. Some patients had more than one presenting symptom or sign.

FIGURE III

Symptom or sign	No. patients
Vomiting	23
Seizures	17
Increased head size	13
Increased irritability	13
Lethargy	7
Temperature elevations	12
Feeding problem	5
Retarded development	6
Diarrhea	4
Paralysis	3
Stupor	3

There is nothing characteristic about the type of vomiting. It may or may not be of a forceful projectile type. It often occurs immediately after food ingestion and this can be very disconcerting when it occurs in a child who takes food poorly.

The seizures may vary from short petit mal to prolonged grand mal attacks. Often times the seizures, if focal, are followed by a postseizure palsy of the involved extremity.

The increase in head size usually is not great. In this series it averaged only 2.1 centimeters above the normal circumference. The important feature is a combination of increased head size and either bulging fontanelle and/or one of the other symptoms listed. It is emphasized that by no means do all infants with subdural hematomas have enlarged heads nor do they all have bulging fontanelles. Fourteen of the thirty-five infants in this series had normal head circumferences. It is possible that the enlarged head is due not so much to the mass of the hematoma as to the presence of an associated hydrocephalus (Penfield[22]). The hydrocephalus results from blockage by the hematoma of the subarachnoid spaces over the cerebral hemispheres thereby preventing absorption of the cerebrospinal fluid.

DIAGNOSIS

The diagnosis of a subdural hematoma in an infant is established by puncture of the subdural space. These punctures should be made with a short bevelled No. 20 lumbar puncture needle containing a stylet. The needle should be inserted at the lateral angle of the anterior fontanelle and directed laterally or posteriorly. It should pierce obliquely through the skin, galea, periosteum, and dura. Care must be taken to avoid traumatizing the cortex. The needle point should be advanced slowly and the stylet frequently withdrawn to prevent too deep penetration. All lateral motion of the needle must be avoided. With such precautions the procedure is relatively safe. In a normal infant only a few drops of fluid, under no tension, will be obtained from the subdural space. The presence of xanthochromic or pink, blood-tinged fluid in amounts greater than three or four cubic centimeters is abnormal. Occasionally this abnormal fluid will spurt out; but at times it will appear to be under no increased pressure. The reason for the later circumstance is that in infants the ununited sutures permit the cranium to expand thereby preventing appreciably increased intracranial pressure.

The presence of blood-tinged fluid is indication of recent hemorrhage. Xanthochromic fluid is evidence of old hemorrhage and is due to the breakdown of hemoglobin pigments.

Lumbar spinal fluid and ventricular fluid in these children are normal in color and protein content in contradistinction to the abnormal hematoma fluid.

The majority of subdural hematomas when they come to operation are extremely large and extend over the entire posterior frontal, parietal, superior temporal and anterior occipital lobes so that a laterally directed needle through the anterior fontanelle will encounter the hematoma. Occasionally a hematoma will be located only over the frontal region as was the case in one infant in this series or only over the occipital region as in another. In these instances the diagnosis can be established by directing the aspirating needle anteriorly from the anterior fontanelle or by inserting it through the lambdoid suture. It is advisable to make these punctures in infants strongly suspected of having hematomas and in whom the usual punctures do not reveal the hematoma. If after subdural punctures, there is still a question of the presence of a hematoma, trephine holes are made to establish more certainly the diagnosis. If at that time a hematoma is found and if the patient is in good condition, a craniotomy may be performed and the hematoma removed. If the child is not in good condition, craniotomy is postponed.

Roentgenograms of the skull are always made. Not only do they help in demonstrating a fracture line if present but they may be useful as collaborating evidence of the presence of increased intracranial pressure. The presence of abnormal x-rays may aid in the differential diagnosis.

TREATMENT

There are three principal methods of treatment of infants with subdural hematomas. (1) Aspiration alone, (2) Craniotomy alone and (3) Aspiration followed by craniotomy. Aspiration of the subdural space can be used as a treatment in infants with acute subdural hemorrhages in which fresh and unclotted blood is withdrawn at the time of aspiration. In these instances no membrane has developed because of the acuteness of the hemorrhage and if the patients are trephined, usually no membrane is found. A single or sometimes two such aspirations suffice to clear the hemorrhage. In infants with chronic subdural hematomas aspiration of the subdural space can be used to relieve temporarily the increased intracranial pressure and thus allow the general condition of the infant to improve; but, generally, aspiration is not effective in producing permanent cure. The aspirated fluid usually re-

accumulates within twenty-four hours; and, even if it did not, the encompassing membrane usually present in chronic subdural hematoma inhibits growth of the brain during this period of most rapid increase in brain volume.

Growth of the brain has been demonstrated by Scommon and Dunn to form a parabolic curve when plotted graphically. The period of most rapid growth lasts for only the first two or three years. After the age of three years, there is only a slight increase in intracranial contents. The increase in head size is due largely to thickening of the bones of the skull and growth of the accessory sinuses and bones of the face. The encompassing membrane prevents this normal brain expansion so that removal of the membrane is necessary to effect a cure.

The second method of treatment is to perform a craniotomy immediately after the diagnosis of a chronic subdural hematoma is established. At the time of the craniotomy the hematoma along with the encompassing membrane is removed. This effects a permanent cure; but, this method of therapy has lead to a relatively high operative mortality rate.

It was because of the high operative mortality rate with the immediate craniotomy that Ingraham and Matson evolved the policy of daily subdural aspiration on alternate sides for a period of ten to fourteen days in an attempt to keep the intracranial pressure close to normal while the infants general physical condition was built up to a point permitting a craniotomy. By this method the operative mortality rate has been greatly lowered.

The operative mortality rate reported in the literature has varied almost directly with the magnitude of the procedure. Sherwood[26] reported nine cases treated with aspiration alone with only one death. However, of the remaining eight only three were normal, and the rest had sequelae, principally mental retardation. On the other hand, Peet and Kahn[23] in 1932 reported on nine cases in whom craniotomies were done without preliminary multiple subdural aspirations. Five of the nine infants expired during or immediately after surgery. The surviving four infants apparently did very well. They advocated surgical removal of the membranes inspite of the high mortality. As mentioned above, Ingraham and Matson[14] believed the best results were obtained by preliminary subdural aspirations followed by trephination and later craniotomy with removal of the membranes. They reported an operatively mortality of nine patients (9%) in their series of ninty-eight infants. Three died during the operation and six died later during hospitalization. One died from poor general health, two from meningitis, and in the other three the cause of death was not determined.

The policy of therapy of subdural hematomas in this series was to first establish the diagnosis by subdural puncture. Following this several more subdural punctures were performed in order (1) to ascertain if the hematoma could be cured with aspiration alone, and (2) in order to better prepare the patient for surgery. If decreased amounts of fluid were obtained with each aspiration and if it seemed as though there were a chance of effecting a cure by simple aspirations, the aspirations were continued. If after five or six such aspirations the hematoma cavity hadn't disappeared, and if the patient were now in relatively good general condition, a craniotomy was performed and the hematoma with its encompassing membrane removed. At the time of operation it was considered best to make a trephine hole over the suspected hematoma and if a lesion with its membrane were encountered the head was rotated and a trephine hole made over the opposite hemisphere. If a hematoma and membrane were encountered here also, a very large osteoplastic craniotomy flap was performed on this second side and both the inner and outer membranes removed. It is necessary to remove about all of the outer and inner membrane possible because unremoved membranes will prevent

the brain from expanding and will permit reaccumulation or recurrence of the hematoma. Because these hematomas often extend over the entire hemisphere it is emphasized that the craniotomy must be very large. About ten days after the first operation, the hematomas and membrane are similarly removed from opposite sides.

Children seem to tolerate these procedures well. Pre-operatively, infusion of glucose or Ringer Solution is established; blood loss is replaced during the operation. Anesthesia used is local procaine (1%) supplementated by rectal pentothal.

The direct operative mortality in this series of thirty-nine infants was three (7.9%). One patient died of blood loss during the procedure. Two died postoperatively, one death was secondary to a bowel obstruction and the other one to increased intracranial pressure. In one of the thirty-nine cases in this series, a second operation was necessary to remove remaining membrane because of recurrence of the hematoma.

In this series of thirty-nine cases, twenty-eight (72%) had bilateral hematomas. The lesions were located over most of the hemisphere in thirty-three (85%), over only the parietal area four (10%), over only the frontal area in one (2.5%), and over only the occipital area in one (2.5%).

There was a well established inner and outer membrane in twenty-nine, (75%), and an outer membrane only in eight (21%). In two cases (5%) there was a well established inner and outer membrane on one side and only an outer on the other side. In three cases (9%), the membrane was atypical. It was in these three that infection seemed to be the etiological factor. In these cases the membrane was similar to those occurring secondary to meningitis as described above.

We attempted to follow the infants who had been treated by simple aspiration. Although we obtained excellent cooperation from the Record Room, we were unable to locate any records in the cross-indexed files of infants who had been treated in that fashion. We realize that there have been infants who have been treated in this fashion and that this portion of our study is unsatisfactory. According to the reports in the literature, this mode of therapy is inadequate because of the greater morbidity if the membrane is not removed. This is the logical deduction when one considers that the remaining membrane would prevent proper expansion and development of the cerebral hemisphere and permit scarring and traction on the cortex.

PROGNOSIS

In order to ascertain the prognosis of the patients treated for subdural hematomas, an attempt was made to follow-up the 39 cases included in this report. As shown in Figure 4, three (8%) expired in the hospital.

FIGURE IV

Follow-up on Infants operated upon for Subdural Hematomas.

Total No. Cases	Died in Hosp.	Followed		Not Followed Adequately
		Died	Living	
39	3(8%)	2(4%)	28(72%)	6(16%)

An adequate follow-up was obtained in 30 of the remaining 36 cases. In one of the six cases not included in the follow-up, data was obtainable for a period of five months after discharge from the hospital, but it is felt that the length of this period is inadequate to ascertain accurately the final outcome. The remaining five could not be followed because they had moved and no forwarding address was available. Of the 30 cases that were followed after discharge from the hospital, two have expired, one from a virus pneumonia and the other from status epilepticus. Twenty-eight patients were followed for a period ranging from one year to eight years with a mean follow-up period of 3.3 years. At the time of final follow-up examination on these 28 patients, an attempt was made to evaluate the children both with regard to mental and to physical capabilities. The results of the psychometric evulation are shown in figure 5.

FIGURE V

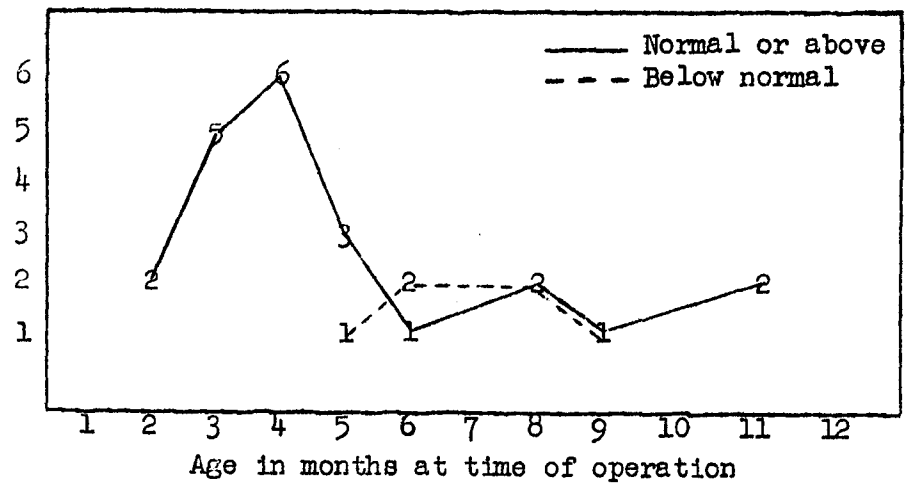
Psychometric Evaluation

Evaluation	Total No.	Evaluated by:		
		Psychiatry	Neurosurg. or Peds	Mother
Normal or above	22(79%)	7(32%)	13(60%)	2(8%)
Below normal	6(21%)	3(50%)	3(50%)	

In figure six is shown the age in months at time of operation of the children who are now considered to be of normal or above normal according to psychometric evaluation and also the age of those found to be below normal on psychometric evaluation. Although, the total number is too small to be statistically reliable, it would seem that those infants operated upon before six months of age had a greater chance of being normal according to psychometric evaluation than those operated upon after that age.

FIGURE VI

Psychometric evaluation relative to age at time of operation.



In figure seven is shown the evaluation of the electroencephalographic changes. Unfortunately none of these children had an electroencephalogram preoperatively.

FIGURE VII

Electroencephalogram evaluation of infants operated upon for subdural hematomas.

	Normal	Focal Dysrhythmia	Generalized Dysrhythmia	Focal and General
NUMBER	3	3	2	3

In a report by Marinacci et al it was concluded that the encephalogram was of a definite help in the diagnosis. They found a definite suppression of activity over the hematoma in seventy-five per cent of the children. The degree of suppression of activity was not necessarily consistent with the size of the hematoma, but was consistent with the degree of injury produced by compression of the cerebral cortex. In figure eight is shown the number and frequency of the persistent neurological abnormalities found in these children at time of follow-up study.

FIGURE VIII

Residual abnormalities in children following subdural hematomas.

Blind	1
Ocular imbalance	1
Paresis	
leg	1
arm and leg	1
Convulsions	1
General retardation-Physical and mental	6
Normal	17
TOTAL-Adequately followed	28

Invariably, the admission symptoms of vomiting, increased head size, increased irritability, lethargy, feeding problems, etc., disappeared after surgery.

SUMMARY

1. A review of the historical, etiological and pathological features of subdural hematomas in infants has been presented.
2. A summary of 39 infants treated for subdural hematomas at the University of Minnesota Hospitals, Minneapolis, is given.
3. The following impressions are obtained from this review:
   a. Post meningitis subdural accumulation of fluid vary from those secondary to trauma in that the inner membrane is difficult to remove because of its adherence to the cortex.

b. Chronic subdural hematomas can seldom be cured simply with multiple aspiration of the subdural space.

c. By a combination of repeated subdural aspiration and craniotomy to remove the membranes, good therapeutic result can be obtained. At the time of craniotomy all the encompassing membrane must be removed to prevent recurrence of the hematoma and/or residual neurologic abnormalities.

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II. MEDICAL SCHOOL NEWS

Coming Events

Mar. 20	E. Starr Judd Lectureship in Surgery; "Some Observations on the Treatment of Carcinoma of the Pancreas," Dr. Thomas G. Orr, Professor of Surgery, University of Kansas; Owre Amphitheater; 8:15 p.m.
Mar. 24-26	Continuation Course in Therapeutics for General Physicians
Apr. 7-9	Continuation Course in Surgery for General Physicians
Apr. 8	George E. Fahr Lectureship; "Coarctation of the Aorta," Dr. Robert E. Gross, Ladd Professor of Children's Surgery, Harvard Medical School, and Surgeon-in-Chief; Children's Hospital, Boston; Owre Amphitheater; 8:15 p.m.
Apr. 14-19	Continuation Course in Proctology for General Physicians
Apr. 17-19	Continuation Course in Obstetrics for Specialists

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Continuation Course in Therapeutics

The University of Minnesota will present a continuation course in Therapeutics to be held at the Center for Continuation Study from March 24-26, 1952. This course is intended primarily for doctors of medicine engaged in general practice and will cover a rather wide field of interest. Among other things, the treatment of certain gastro-intestinal conditions, the treatment of certain household poisonings, and the management of acute chronic alcoholism will be discussed. One-half day will be devoted to the problem of psychotherapy and another half-day will be given over to the consideration of various problems and peripheral vascular disease. The faculty will include clinical and full-time members of the staff of the University of Minnesota Medical School and the Mayo Foundation.

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Faculty News

Dr. Wesley W. Spink, Professor, Department of Medicine, addressed the Boston City Hospital House Officers Association in Boston on the subject, "Intracellular Parasitism in Brucellosis," on Friday, February 19. He also visited at the Army Medical Graduate School in Washington, D. C. on February 29.

Dr. Owen H. Wangensteen, Professor and Chairman, Department of Surgery, attended the Fifth Annual Gastric Cancer Conference in Cincinnati, Ohio, on March, 3, 4, and 5. He presented a paper on "Cancer of the Esophagus and the Stomach." He also attended the meeting of the Board of Directors of the American Cancer Society there on March 2.

Dr. Claude R. Hitchcock, Fellow in Surgery, also attended the Fifth Annual Gastric Cancer Conference and presented a paper entitled, "Experimental Studies with the Nematode Parasite, Gongylonema Neoplasticum, (Spiroptera Neoplasticum) and Vitamin A Deficient Diets Affecting the Forestomach of the Rat: Comparison of Results with Those of J. Fibiger."

Dr. F. John Lewis, Assistant Professor, Department of Surgery, will attend the meeting of the Central Surgical Association in Toronto, Canada, on March 6-8, and will speak on "Clinical Uses of the Artificial Kidney."

III

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

March 10 - 15, 1952

Monday, March 10

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom, Todd Amphitheater, U. H.

11:30 - Physical Medicine Seminar; Evaluation of Disability of the Hand; Kenath Sponsel; 142 Chemical Engineering Bldg.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - Physiology Seminar; 214 Millard Hall.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; Meningococcus Meningitis; R. Disenhouse; Sixth Floor West, U. H.

4:30 - 5:30 Dermatological Seminar; M-346, U. H.

4:30 - Public Health Seminar; 15 Owre Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

7:30 - Fracture Grand Rounds; Dr. Zierold; Sta. A.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Monday, March 10 (Cont.)

Veterans Administration Hospital

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; Conference Room; Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

3:30 - Psychosomatic Rounds; C. K. Aldrich; Bldg. I.

Tuesday, March 11

Medical School and University Hospitals

8:30 - Conference on Diet Endocrines and Cancer; M. B. Visscher; 116 Millard Hall.

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:00 - 1:30 Selected Topics, Permeability and Metabolism; Nathan Lifson; 129 Millard Hall.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases by Ancker Hospital Staff; Drs. Aurelius, D. Peterson, and Ogden; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 2:30 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Infectious Disease Rounds; Dr. Hall.

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

Tuesday, March 11 (Cont.)

Veterans Administration Hospital (Cont.)

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery Tumor Conference; Conference Room, Bldg. I.

1:00 - Surgery Chest Conference; T. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff Bldg. III.

3:30 - 4:20 Clinical Pathological Conference; Conference Room, Bldg. I.

Wednesday, March 12

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Allen Judd and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Surgery Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:30 - 1:20 Radio-Isotope Seminar; 12 Owre Hall.

1:30 - Conference on Circulatory and Renal Systems Problems; M. B. Visscher; 116 Millard Hall.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

5:00 - 6:00 Vascular Conference; Todd Amphitheater, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

8:00 - Pediatric Allergy Rounds; Lloyd Nelson; 4th Floor.

Wednesday, March 12 (Cont.)

Minneapolis General Hospital (Cont.)

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:30 - Pediatric Staff Meeting; Orthodontics in Everyday Practice; C. E. Rudolph; 4th Floor Annex.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:30 - Pediatric Rounds; E. J. Huenekens and Robert Ulstrom; 4th Floor.

2:00 - 4:00 Infectious Disease Rounds; 8th Floor.

4:00 - 5:00 Infectious Disease Conference; Classroom, 8th Floor.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, March 13

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

3:30 - Medicine-Pediatric Infectious Disease Conference; Heart Hospital Auditorium.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 X-ray Seminar; Clinical Study of Colonic Ruptures in the Course of Barium Enema; Elliott Lasser and Norman Zheutlin; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

Thursday, March 13 (Cont.)

Minneapolis General Hospital (Cont.)

8:30 - Neurology Rounds; William Heilig; 4th Floor.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

1:00 - Fracture-X-ray Conference; Dr. Zierold; Classroom.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery Roentgen Conference; Conference Room, Bldg. I.

Friday, March 14

Medical School and University Hospitals

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Criteria of Normality in Clinical Electrocardiography; Ernst Simonson; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

3:30 - 4:30 Advanced Neurophysiology Seminar; E. Gellhorn; 111 Owre Hall.

4:00 - 5:00 Dermatology Seminar; W-321, U. H.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

11:00 - Pediatric-Surgery Conference; Dr. Wyatt, Forrest Adams; Classroom, Sta. I.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

Friday, March 14 (Cont.)

Minneapolis General Hospital (Cont.)

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Microscopic-Pathology Conference; E. T. Bell; Conference Room, Bldg. I.

1:30 - Chest Conference; Wm. Tucker and J. A. Meyers; Ward 62, Day Room.

3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, March 15

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater,

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

Minneapolis General Hospital

8:00 - Pediatric Rounds; George Lund; 5th Floor.

11:00 - 12:00 Medical - X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

11:00 - Pediatric Clinic; C. D. May and Floyd Denny; Classroom, 4th Floor.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Hematology Rounds; P. Hagen and E. F. Englund.