SUBDURAL EFFUSIONS WITH RESPIRATORY INFECTIONS IN THE FIRST YEAR OF LIFE

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Since the papers of McKay et al.^{3,2} in 1950 and 1953 drawing attention to the occurrence of subdural effusions with meningitis, it has become commonplace to consider this complication when cases of meningitis, especially those due to *H. influenzae* and *S. pneumoniae*, fail to pursue their expected course with antibiotic treatment. Although the explanation for the occurrence of subdural effusion with meningitis remains obscure, at least the lesions are both intracranial and the complication therefore the more easily remembered.

Other diseases have been associated with subdural effusions,³⁻⁵ especially inflammatory disease of the respiratory system and intestinal tract, and in some cases apparently simple malnutrition. Subdural effusions occurring at the time of birth from injury or haemorrhagic disease have long been known. Because there seems no immediate reason why infections of the respiratory and intestinal tracts should be found with subdural effusions, the association is therefore the less easily remembered, and so we report here 9 cases of respiratory infections in which this complication impeded recovery. The respiratory disease varied from tracheo-bronchitis to frank pneumonia, and all cases responded to treatment with antibiotics, so that no undue anxiety was caused by the respiratory disease *per se* and a virus aetiology appeared unlikely.

The explanation for these effusions remains obscure, and no patient died, except one who was readmitted 7 months after discharge and died of broncho-pneumonia and at whose autopsy no trace of the subdural effusion was found. No operations other than subdural taps were performed, and the follow-up examinations have not revealed any sequelae of local or general brain damage. All these cases occurred in Coloured* and Native children, and over the same period no European cases were seen.

Before reporting on the series of 9 cases as a whole we submit the case histories of two of the patients (cases 1 and 7).

CASE HISTORIES

Case 1. F.S., Coloured male, aged 5 months

This child was seen by one of us (W.W.) before admission. For 3 days he was feverish and restless and took his feeds poorly, and on the day before admission he developed a cough and became dyspnoeic; no convulsions, diarrhoea or vomiting. There was no history of any previous illness or of birth trauma. On examination he was found to be a well-nourished child, very ill and markedly dyspnoeic, with a temperature of $102 \cdot 4^{\circ}$ F but not dehydrated. Air entry to both lungs was good, but mild bronchospasm was present, and there were scattered rhonchi in both lung fields with crepitations over the upper zone of the right lung. There were no abnormal physical signs in the central nervous system, and the fontanelle was normal. The other systems were normal. Chest X-ray showed increased broncho-vascular markings and

* The Coloured race is derived from a mixture of European with Malay, Hottentot, Bantu (Native) or Bushman Stock. there were areas of patchy consolidation in the right upper zone. Further investigations are reported in Table I.

A diagnosis of broncho-pneumonia was made, and the child was treated with chloramphenicol and given oxygen and alevaire during the first day in hospital. After 48 hours he was afebrile and markedly improved generally, and the chest was clear. He continued to take his feeds poorly. On the 3rd day it was noticed that he had marked head retraction and slight tenseness of the anterior fontanelle. A lumbar puncture was done and the cerebrospinal fluid (CSF) was clear, colourless, and normal on analysis. A subdural tap was then performed and 15 c.c. of bloody xanthochromic fluid was found on the right side (Table II). After the first tap there was a marked improvement and less retraction of the head, and the child fed better.

Subdural taps were subsequently done on one or both sides at intervals ranging from 1 to 3 days, yielding 10-30 c.c. of fluid at each tap during the following 7 weeks. The quantity then diminished, and the spaces became dry after 9 weeks. A syringe was not used to withdraw fluid and surgical drainage was not carried out. At the second tap 1 c.c. of methylene blue was injected into the right subdural space and a lumbar puncture performed 4 hours later. There was no dye in the CSF, which showed that the fluid found at subdural tap did not communicate readily with the subarachnoid space. Subdural air studies and a pneumoencephalogram were done (Table II).

While in hospital he made good progress and gained $2\frac{1}{2}$ lb. in weight. On discharge there were no signs of mental retardation. At follow-up 7 months later he was a healthy and normal child (Table III).

Case 7. M.H., Coloured male, aged 1 year

This child was ill for 3 days before admission, with fever and a non-productive cough, and was refusing his feeds. On the day of admission he became short of breath and irritable, and had a generalized convulsion, which lasted for a few minutes. His temperature on admission was 104°F, he was fully conscious but irritable, and dyspnoeic, and looked very ill. There was no cyanosis, and he was not dehydrated. The chest showed rib retraction at the costal margins, and over the right side in front dullness to percussion and bronchial breathing were observed. There were no adventitous sounds. In the abdomen the liver was palpable about one finger below the costal margin and the tip of the spleen could be felt. The fontanelle was open, but not bulging, and marked neck stiffness was present, with positive Kernig and Brudzinski signs. There were no other positive physical findings, and nothing significant in the past history. Chest X-ray (Fig. 1) showed dense consolidation of the entire right upper lobe. The lumbar-puncture fluid was normal. Further investigations are set out in Table I.

A diagnosis of lobar pneumonia with a febrile convulsion was made, and the child treated with penicillin and sulphadimidine. Within 24 hours the temperature came down to normal and remained so. On the 3rd day the lumbar puncture was repeated and a subdural tap performed because there was marked head retraction and the child was sucking very poorly. He had no respiratory distress at this stage, and the fontanelle was not bulging. From the left subdural space 16 c.c. of straw-coloured fluid was aspirated and 8 c.c. from the right side (Table II).

Subdural air studies were done at the time of the first tap; 10 c.c. of air was put into the left subdural space and X-rays taken (Fig. 2). Blood was taken through the anterior fontanelle from the sagittal sinus and a free flow was obtained, showing that there was no thrombosis. Subdural taps were performed

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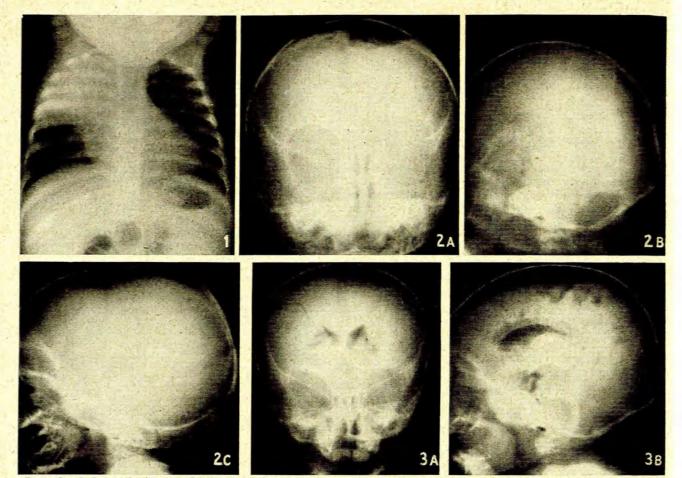


Fig. 1. Case 7. Pneumonia, right upper lobe (on admission).

Fig. 2.4. Case 7. Left subduragram. P.A. view in upright position. Note limitation of air shadow by falx cerebri.

Fig. 2B. Case 7. Left subduragram. Prone position. Lateral view.

daily, and gradually diminishing amounts of fluid were obtained until on the 7th day the space was dry.

After the first tap the baby started taking the feeds well, the head retraction improved, and he gained weight during the following days.

He was discharged well on the 10th day and readmitted a week later for a lumbar encephalogram (Fig. 3) and X-ray of the chest. The chest lesion was now virtually resolved, and he had gained 1 lb. in weight since discharge. At follow-up $4\frac{1}{2}$ months later, he was found to be healthy and normal, and to have gained a further 3 lb. (Table III).

STUDIES IN THE NINE CASES

Clinical Features

The clinical findings in this series are summarized in Table I. All the 9 children were admitted with chest infections, and 7 of them had radiological indications of pneumonia. The remaining 2 cases with clear X-rays had the physical signs of bronchitis and bronchiolitis.

Head retraction in all cases was the sign which drew attention to disease of the central nervous system. Only 2 cases showed abnormal fontanelles. In 6 cases the children were noted to be sucking poorly or refusing their feeds. One child had a convulsion, one vomited, and in 3 the neck was judged to be stiff. In 2 cases the children had been treated as out-patients and were admitted to hospital because of the development of head retraction.

Fig. 2C. Case 7. Left subduragram. Upright. Lateral view. Fig. 3A. Case 7. Lumbar encephalogram. P.A. upright. Normal ventricles. Air in left subdural space. Fig. 3B. Case 7. Lumbar encephalogram. Lateral upright. Air present in subarachnoid and subdural spaces. Ventricles normal.

In 5 cases subdural fluid was obtained from both sides at the initial tap, and in 2 of the remaining cases bilateral subdural effusions occurred later. In 2 cases unilateral effusions only were found. During the period under review a considerable number of cases of respiratory disease were treated as out-patients, and did not develop head retraction. Wherever this sign was found, subdural tap revealed an effusion. Usually this effusion was straw-coloured, but occasionally it was bloody. This is not to say that a subdural tap is the first examination to be made. Where signs of irritation of the central nervous system occur in the presence of infection, the first disease to be excluded must be meningitis, so that a lumbar puncture always preceded a subdural tap. In one case the CSF contained 12 cells per c.mm. and was sterile.

Following subdural taps an immediate improvement was noticed in the child's behaviour and its willingness to take feeds, and the head retraction disappeared in a few days.

The fluid obtained by subdural taps differed markedly in all cases from the fluid obtained at the same time by lumbar puncture. In 7 cases unilateral subdural air studies were made, and in no case did the air inserted cross the mid-line or fill the fissures of the brain. In case 1 a dye

TABLE I. CLINICAL FEATURES ON ADMISSION

| | | | | | | 5 | | Symptoms | | | | | - | | | | | | | | | A TANK |
|-------------|--------------|-----|------|--|--|---------------------------------|--|----------|--------------|-------|----------|-----------|-------------|------------|---|------------------------------------|------------|----------------|---------------------|--------------------|--|---|
| Case Number | Age (months) | Sex | Race | Date of Admission and Discharge (1958) | Weight on Admission and Discharge (lb. and oz.) | Duration of symptoms in days | Diagnosis on Admission | Cough | Irritability | Fever | Vomiting | Diarrhoea | Convulsions | Fontanelle | X-ray Chest | Blood Count | Wassermann | Mantoux 1/1000 | Rectal Swab | Throat Swab | Lumbar Puncture | Remarks |
| F.S. | 5 | M | Col. | 21 Jul. 1 Oct. | 12, 9 14, 11 | 3 | Broncho- pneumonia | + | + | + | - | 1 | - | N | Patchy consol. R upper zone | Hb 10.0 g% wbc 7,800 P 57% | - | - | - | C. albicans | 0 | |
| R.Z. | 3 | М | Nat. | 16 Aug. 1 Oct. | 11, 0 11, 11 | 5 | Lobar pneumonia | + | + | + | + | 15 | - | N | Consol. L lower lobe and lingula | Hb 10.5 g% | 0 | 4 | | 0 | 0 | 1 |
| 3 L.W. | 4 | F | Col. | 19 Aug. 20 Oct. | 5, 11 7, 11 | 3 | Bronchitis. Gastro- enteritis | 4 | - | + | + | + | - | N | NAD | Hb 11.0 g% wbc 15,600 P 38% | 0 | - | | C. albicans | 0 | |
| 4 С.Н. | 6 | M | Col. | 2 Sep. 5 Oct. | 12, 5 13, 13 | 28 | Broncho- pneumonia | + | - | 1 | + | 1 2 3 4 | 1000 | N | Patchy consol. R lower lobe and L base | Hb 9·9 g% wbc 21,350 P 45% | 0 | - | | 0 | Cells 12/c.mm Prot. 10 mg% Glob. neg. Chlor. 630 mg% Sugar N Culture sterile | Chest infection. Treated as out- patient. Head retraction. |
| 5.M. | 7 | F | Nat. | 13 Oct 8 Nov. | 8, 0 8, 5 | 3 | Lobar pneumonia | + | 1. | t | - | 1-1 | | N | Consol. L upper lobe. Periosteal thickening of humeri | Hb. 9·5 g% P 40% | + | 140 | - | Staph. aureus | N | Stiff neck |
| 6 М.В. | 6 | F | Col. | 13 Nov. 26 Nov. | 12, 8 11, 15 | 3 | Lobar pneumonia | + | - | + | - | - | 1 | N | Consol, lingula | Hb 9.5 g% wbc 23,850 P 69% | 0 | - | - | K. pneu- moniae | N | Stiff neck |
| 7 М.Н. | 12 | м | Col. | 29 Nov. 9 Dec. | 15, 6 16, 13 | 3 | Lobar pneumonia | + | + | + | - | - | + | N | Consol. R upper lobe | Hb 9.6 g% wbc 8,700 P. 79% | 2 | - | - | H. influ- enzae | N | Convulsion |
| 8 B.M. | 5 | м | Col. | 12 Dec. 23 Dec. | 10, 9 12, 9 | 28 | Bronchio- litis. Gastro- enteritis | + | + | + | + | - | - | N | NAD | Hb. 12.0 g% wbc 11,000 P 58% | - | 1 | | 0 | N | Head retraction |
| 9 A.S. | 6 | м | Col. | 29 Dec. 28 Feb. 59 | 13, 5 14, 0 | 14 | Broncho- pneumonia. Gastro- enteritis | + | - | + | + | + | | N | Patchy consol. R upper lobe | Hb. 11 0 g% wbc 14,500 P 68% | 0 | - | S. typhi- murium | E. coli. | 0 | |

Col=Coloured, Nat=Native, +=present, -=absent, Q=not done, N=normal, NAD=nil abnormal, Hb=haemoglobin, wbc=white cells/c,mm, P=polymorphonuclear leucocytes, R=right, L=left, P=right, P=right

| Case No. | History of Injury | Date of Admission (1958) | Date of First Tap | Reason | Fontenelle | 14 | +1 +1 | F | luids at First | Tap | | | Cubdunal | Lumbar Encephalogram | Remarks |
|-----------|-------------------------|--------------------------------|----------------------|---|------------|----------|--------|----------------------|--------------------------|-----------------|----------------|---------|--------------------------------------|---|--|
| | | | (1958) | | | | Side | Quan- tity ml. | Appear- ànce | Protein mg.% | Cells c.mm. | Culture | Subdural Air Studies (1958) | | |
| 1 F.S. | Nil | 21 Jul. | 27 Jul. | Head retraction. Bulging | Bulging | Subdural | R | 15 | Bloody | 300 | +++ | S | 5 Aug. L | 13 Aug. Air. in L subdural space. Ventricles N | 16 taps. 170 ml. in all, R 80 ml. L 90 ml. |
| | | | 27 541. | fontanelle. Sucking poorly | Buiging | CSF | 312.50 | 20 8210 | Clear | 10 | 3 | S | | | |
| | Nil NVD | 16 Aug. | 25 Aug. | Head retraction. Sucking poorly | N | Subdural | R L | 53 | Bloody Straw | 180 | 39. | S | 0 | 3 Sep. Air in both sub- dural spaces. Ventricles N | 13 taps. 35 ml. in all, R 5 ml. L 30 ml. |
| | | | | | | CSF | | 75-127 | Clear | 10 | 0 | S | | | |
| 1.W. | Nil NVD | 19 Aug. | 13 Sep. | Head retraction. Sucking poorly | N | Subdural | R L | 33 | Bloody Straw | 100 | 12 | S | 14 Sep. L | 29 Sep. Air in both sub- dural spaces, | 9 taps. 23 ml. in all, R 8 ml. L 15 ml. |
| 1 | | | 111 | | | C.S.F. | | | Clear | 10 | 0 | S | | Ventricles N | |
| C.H. N | Nil NVD | 2 Sep. | 2 Sep. | Head retraction. | N | Subdural | R | 2 · | Yellow | | A. 1. 1. 1. | S | 0 | 25 Sep. N | 8 taps. 4 ml. in all, |
| | | | | | | C.S.F. | 1.5150 | 1.00 | Clear | 10 | 12 | S | | | R 4 ml. L nil. |
| 5 S.M. | Nil NVD | 13 Oct. | 15 Oct. | Head retraction. Sucking poorly | N | Subdural | R | 10 | Yellow | 50 | 27 | S | 15 Oct. R | 4 Nov. N | 6 taps. 42 ml. in all, R. 31 ml. L 11 ml. |
| | | | 10 000 | | | CSF | 1 | 1.0 | Clear | 10 | 0 | S | | | |
| 6 М.В. | Nil NVD | 13 Nov. | 19 Nov. | Head retraction. Stiff neck. Sucking poorly | N | Subdural | R L | 4 12 | Bloody Yellow clot | 10 | 3 | S | 19 Nov. L | 25 Nov. Air in L subdural space. Ventricles N | 4 taps. 19 ml. in all, R 4 ml. L 15 ml. |
| | | | | | | CSF | | | Clear | 10 | 0 | S | | | |
| 7 M.H. | Nil NVD | 29 Nov. | 2 Dec. | Head retraction. Convulsion. Stiff neck. | N | Subdural | R L | 8 16 | Straw Straw | 30 | 9 | S | 2 Dec. L | 17 Dec. Air in L subdural space. | 5 taps. 51 ml. in all, R 27 ml. L. 24 ml. |
| | | 15.25 | and the | Sucking poorly | S. 9 1 | CSF | | 11575 | Clear | 10 | 3 | S | | Ventricles N | |
| 8 B.M. | Nil NVD | 12 Dec. | 12 Dec. | Head retraction. Stiff neck. Vomiting | N | Subdural | R L | 12 1 | Yellow Yellow | 180 | 135 | S | 12 Dec. R | 18 Dec. N | 4 taps. 25 ml. in all, R 20 ml. L 5 ml. |
| | 1.50 | | | , ching | | CSF | 1.1 | | Clear | 20 | 6 | S | | | |
| 9 A.S. | Nil NVD | 29 Dec. | 30 Dec. | Head retraction. Bulging fontanelle | Bulging | Subdural | L | 20 | Straw | 40 | + | S | 2 Jan. 59 L | 31 Jan. 59 | 12 taps. 190 ml. in all, |
| 14.15. | | 29 Dec. | so Dec. | | Buiging | CSF | 10 . | 11.112-1 | Clear | 20 | 6 | S | | N | R. nil. L 190 ml. |

TABLE II. INVESTIGATION OF SUBDURAL AND LUMBAR FLUIDS

NVD=Normal vertex delivery. N=Normal. O=Not done. R=Right. L=Left. S=Sterile.

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| Case No. | Age (months) | Interval since discharge (months) | Symptoms | Milestones | Head circum- ference on admission and follow-up (inches) | Weight on discharge and follow-up (lb. and oz.) | Fon- tan- elle | Remarks |
|-------------|-----------------|--|--|-----------------------------|--|--|----------------------|---|
| 1 | 14 | 7 | Coryza | Normal | 16 18 | 14, 11 17, 0 | N | |
| 2 | 11 | 7 | Malnutrition. Gastro- enteritis | Sitting but not crawling | 16 17 | 11, 11 14, 7 | N | Delayed milestones probably due to malnutrition. |
| 3 | 12 | 6 | None | Sitting but not crawling | 16 | 7, 11 11, 13 | N | Delayed milestones probably due to malnutrition. |
| 4 | 14 | 7 | Broncho- pneumonia. Malnutrition | Normal | 16½ 17½ | 13, 13 13, 3 | N | Died 4 days after 2nd admis- sion. Autopsy—broncho-pneu- monia. Nil abnormal intra- cranially. |
| 5 | 13 | 5 <u>1</u> | None | Normal | 163 | 8, 5 14, 15 | N | X-rays of bone show resolution of syphilitic process. |
| 6 | 12 | 5 | None | Normal | 16 16 ³ | 11, 15 15, 9 | С | |
| 7 | 16 | 41 | None | Normal | 19 19 | 16, 13 19, 15 | N | |
| 8 | 9 | 4 | None | Normal | 174 | 12, 9 15, 9 | N | |
| 9 | 17 | - | | | E I | 14, 0 | - | Case from afar. No reply to follow-up letter. |

TABLE III. FOLLOW-UP EXAMINATION

N=Normal.

was injected into the subdural space and did not appear in the CSF.

The radiological studies by lumbar encephalogram in no case showed any dilatation of the ventricular system. However, the distribution of air varied considerably and is discussed below.

Technique of Subdural Tap

Taps are usually made through the anterior fontanelle, since the posterior fontanelle is closed at birth or soon after. The scalp is shaved to expose the fontanelle and surrounding scalp, and the skin is prepared as for any surgical procedure.

It must be remembered that, once air is inserted into the subdural space, the fluid will move freely in all directions, so that meticulous positioning may be necessary. For the initial puncture it is sufficient to lay the baby on its back, suitably restrained, and insert the needle at right angles at the lateral margins of the fontanelle.

A 20-gauge lumbar-puncture needle with a short bevel should be used. After passing through the scalp a strong resistance will be felt at a depth between one-eighth and one-quarter of an inch. This is the dura mater and ommediately beneath lies the subdural space. Normally not more than a few drops of clear fluid are obtained. If no fluid is obtained the needle, with stylette reinserted, is passed

C = Closed.

forward, laterally and backwards for a short distance, as indicated by the arrows in Fig. 4. Care must be taken not to advance the needle deeply or the cortical veins may be injured. Alternatively, if possible, the needle may be inserted at right angles through the coronal suture.

In the newborn, or when the posterior fontanelle has been reopened by increased intracranial pressure, the same procedure may be carried out through the lateral margins of the posterior fontanelle to obtain fluid which has tracked backwards under the influence of gravity. A lateral insertion is essential to avoid the sagittal sinus.

After aspiration the puncture should be sealed with a sterile collodion dressing.

The aspiration should be repeated daily until less than 10 c.c. is obtained, or until the fontanelle ceases to bulge. Thereafter less frequent taps will suffice. Not more than 15 c.c. should be aspirated on each side on any one occasion.

Follow-up Studies

Details of the follow-up examination are set out in Table III. No local or general sequelae were observed during the period under review, which ranged from 4 to 7 months after discharge from hospital. One case was readmitted with broncho-pneumonia and died, and at autopsy no anatomical disorder was found within the cranium—in particular no membrane to indicate where the subdural effusion had been. It must be stated that, in this case, the amount of fluid

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withdrawn was small, and subdural air studies were not done.

In 2 cases the milestones were retarded, but this appeared to be a physical rather than an intellectual defect. Coloured children are lighter in weight than their European counterparts⁶ and malnutrition with delayed milestones is common.

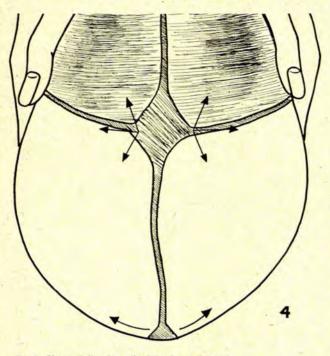


Fig. 4. Sites and directions of subdural taps (see text).

These studies do not support the plea of Williams *et al.*⁴ that open operation and stripping of the subdural membrane is necessary to avoid the possibility of permanent damage. However the follow-up period is short and further studies are required.

One case of syphilis is included, and is discussed below.

DISCUSSION

The aetiology of subdural effusions has exercised the minds of many from Trotter7 (1914) and Penfield8 (1923) to the experiments of Finberg et al.9 (1959), and the subject has been reviewed from time to time.¹⁰⁻¹² One supposes from reading the earlier papers that trauma due to external violence, however trivial, was considered an essential factor. It is notable that the large majority of cases occur in infants while the fontanelle is still open and before the sutures of the membranous cranium unite. It would be expected, therefore, that there would be more spring than in the older, more rigid skull, and that the release following an external impact would be more violent. For the same reason, shearing stresses would be the more easily produced. On the other hand, it is much easier to tap the subdural space at this age, and therefore disease is more likely to be discovered than later, when burr-holes have to be made.

More recently the hypothesis of internal violence has been brought forward, such as occurs with pneumoencephalography¹³ and hypernatraemia.⁹ In these circumstances it is suggested that the brain is stretched away from the surrounding cranium by rapid reduction in its volume, and in the process the bridging veins are torn.

In either event the principle is that a haemorrhage from veins bridging the subdural space occurs, soon followed by membrane formation and encapsulation. Fibrin and blood clot collect at the periphery of the space, and more fluid is attracted by osmosis, so that there is a tendency for the effusion to increase in size. Experimentally this has been done in animals with 'cellophane' bags¹⁴ and with an excised subdural haematoma sac.¹⁵ A tear in the arachnoid may also add cerebrospinal fluid.¹⁶

Any bleeding tendency, particularly that occurring in the first few days of life, may exaggerate any traumatic factor or cause spontaneous bleeding.

But it is not certain that haemorrhage is an essential precursor of subdural effusion, although it is difficult to postulate another cause of exudation. The anatomy of the subdural space has been compared to the synovial lining of a joint¹⁷ in that the dura, being of mesodermal origin, has a capillary bed and is therefore capable of exudation in its own right. The inner side of the subdural space is lined by the arachnoid membrane, which is of ectodermal origin and lacks a capillary bed. Exudation can therefore only occur from the capillaries of the dura, and the arachnoid can only fill the subdural space with blood or cerebrospinal fluid.

In our series all the fluids obtained at the first tap were yellow, and of high protein content, and the majority contained no obvious blood. But since the cavity was not directly seen, it may well be that in cases where no blood was found the space was lined with blood clot and the needle withdrew fluids from the central cyst.

Had the stress of coughing been responsible for rupturing the veins bridging the subdural space, one would have expected reports of subdural effusions to be common in whooping cough, but no such reports have been seen. Our cases were not clinically dehydrated, and did not exhibit scurvy or other bleeding tendency. No history of external violence was obtained nor anything to suggest that they had not progressed mentally in a normal manner.

In case 5 the Wassermann reaction was positive and there was radiological evidence of active syphilitic bone disease. This recalls the pachymeningitis haemorrhagica interna of Virchow, originally postulated as an inflammatory cause of subdural haematoma, and now generally discarded in favour of the traumatic theory. Syphilis, then a common disease, was regarded as a cause. In this case the child was treated with penicillin and his progress has been good and healing of the bony lesions has occurred.

In case 7 a puncture of the sagittal sinus was performed at the same time as a subdural tap, and fluid blood was freely obtained, excluding a thrombophlebitis of that sinus.

Air studies were made by two methods, viz. the insertion of air direct into the subdural space by tapping, and into the subarachnoid space by lumbar puncture. The risks of the production of a subdural effusion by lumbar encephalogram are known,¹³ but in all cases the presence of a subdural effusion had been proved before the encephalogram was undertaken. The time interval between subdural and subarachnoid air studies varied from 6 to 29 days; the details are set out in Table II, and Fig. 2 is a sample. It was not uncommon to find air in the subdural space after lumbar encephalogram (cases 1, 3, 6, 7) and in these cases, except case 3, it was found on the side on which the subduragram had previously been done. It is possible, therefore, that the air had not yet been absorbed, though the interval was as long as 15 days in case 7. In case 3 only a left-sided subduragram had been done, but after encephalogram air was found in both subdural spaces. It is possible that a tear in the arachnoid, as postulated by Munro,16 had permitted the subarachnoid air to spread to the subdural space. This view is supported by case 2, where air was found in both subdural spaces after encephalogram, though no subduragram had been done. A leakage of air at routine taps may, however, have been responsible. In cases 4, 5, 8 and 9 no air was found in the subdural spaces after encephalogram, which was done in cases 5, 8 and 9 from 6 to 29 days after subduragram (in case 4 no subduragram was done). It cannot be said, therefore, that a subarachnoid tear is present in all cases or, if it is, that it is sufficiently great to permit free passage of air between the spaces.

Since the brain trebles in size in the first year of life, any membrane adherent to its surface may interfere with the development of the underlying cortex. Williams et al.4 have therefore advised that the subdural membrane should be stripped to avoid the possibility of permanent damage. We have not done this and have contented ourselves with repeated tappings in one case for as long as 9 weeks, before securing dryness.

Our patients' subsequent progress and follow-up examination have not suggested that they have been handicapped by their illness, or that they were mentally retarded children before their illness began.

SUMMARY

1. Nine cases of respiratory disease are described complicated by subdural effusions. All recovered without sequelae after antibiotic therapy and repeated subdural taps. No open operative procedures were undertaken.

2. The results of follow-up examinations are recorded.

3. No evidence of direct trauma was found in any case, and no evidence of infection of the dubdural space. One case was syphilitic.

4. No cause of this complication has been proved. There is evidence that a tear in the arachnoid may have been present in some cases. The aetiology of subdural effusions in general has been discussed.

OPSOMMING

1. Nege gevalle van respiratoriese siektes, gekompliseerd deur subdurale effusies, is beskryf. Al die pasiënte het herstel sonder gevolge na antibiotiese terapie en herhaalde subdurale aftappings. Geen ope operatiewe behandelings is gedoen nie.

2. Die resultate van opvolg ondersoeke word vermeld.

3. Geen bewys van direkte trauma of infeksie van die subdurale spasie is in enige geval gevind nie. Een van die gevalle was sifilities.

4. Geen oorsaak vir hierdie komplikasie is bewys nie. Daar is bewys dat in sommige gevalle 'n skeur in die arachnoiëd was. Die etiologie van subdurale effusies in die algemeen is bespreek.

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REFERENCES

- 1. McKay, R. J., Morisette, R. A., Ingraham, F. D. and Matson, D. D. (1950): New Engl. J. Med., 242, 20.
- McKay, R. J., Ingraham, F. D. and Matson, D. D. (1953); J. Amer. Med. Assoc., 152, 387.
- 3. Guthkelch, A. N. (1953): Brit. Med. J., 1, 233.
- 4. Williams, J. M. and Stevens, H. (1954): Ann. Surg., 139, 287.
- 5. Herzberger, E., Rotem, Y. and Braham, J. (1956): Arch. Dis. Childh., 31, 44.
- 6. Woodrow, E. P. and Robertson, L. (1950): S. Afr. Med. J., 22, 761.

- Trotter, W. (1914): Brit. J. Surg., 2, 271.
 Penfield, W. G. (1923): Amer. J. Dis. Childh., 26, 383.
 Finberg, L., Luttrell, C. and Redd, H. (1959): Pediatrics, 23, 46.
 Ingraham, F. D. and Matson, D. D. (1949): Advanc. Pediat., 4, 231.
- 11. Smith, M. H. D. (1956): Ibid., 8, 165.
- Editorial (1959): Lancet, 1, 505.
 Smith, H. V. and Crothers, B. (1950): Pediatrics, 5, 375.
- 14. Gardner, W. J. (1932): Arch. Neurol. Psychiat., 27, 847.
- Zollinger, R. and Gross, R. E. (1934); J. Amer. Med. Assoc., 103, 245.
 Munro, D. (1942): New Engl. J. Med., 227, 87.
- 17. Leary, T. and Edwards, E. A. (1933): Arch. Neurol. Psychiat., 29, 691.