ACUTE SUBDURAL HAEMATOMA IN INFANCY AND CHILDHOOD

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Although the clinical pictures of the various types of subdural haematoma merge into one another, it is helpful to consider separately an acute variety unassociated with intrinsic cerebral injury, because in children it presents an urgent problem requiring prompt diagnosis and treatment if fatalities are to be avoided. The peak incidence is in early life, at a time when, as Ingraham and Matson (1944) have pointed out, the significance of trauma is most difficult to assess. In this paper we shall consider rapid compression of the brain by surface clots as a paediatric emergency. The following case is an example of the condition and is reported because it raises a diagnostic difficulty which has received but scant attention.

Case Report

The patient, a 13-month-old girl, was admitted to hospital on December 9, 1949. She was one of a pair of binocular twins. Her mother had been treated for anaemia, hyperemesis, and hydramnios during the pregnancy, but the child was delivered normally at term and caused no anxiety. Head moulding was not excessive. She was bottle-fed from birth and given orange juice and cod-liver oil daily. Her progress was normal; at the time of admission to hospital she weighed 21 lb. (9.5 kg.) and all the incisors were present. According to her mother she had always been pale and had bruised easily. There had been no spontaneous haemorrhages. In addition to her twin sister there are two other children, and all are healthy. Her father has suffered from urticaria and her mother from anaemia, which has responded to iron treatment. There is no consanguinity.

At 4.45 p.m. on December 9 the child, who was sitting playing on the floor, suddenly heeled over and had clonic convulsions involving the right arm and leg. After a few minutes she became limp and stuporous. When seen at 5.30 p.m. she was still in this condition. A bruise on the right cheek and one on each leg contrasted with her general pallor. The left lid was closed and the pupil was dilated but reacted to light. All limbs were completely flaccid and there was no neck rigidity. A history of injury could not be obtained, though minor bumps and tumbles could not be excluded.

By the time she was admitted to hospital three-quarters of an hour later the picture had changed in certain particulars. There was no ptosis and the pupils were equal; the eyes were deviated to the left. The right arm was flaccid and externally rotated; the other limbs moved normally. The right biceps-jerk was absent, the right knee- and ankle-jerks were exaggerated, and the right plantar response was extensor; the abdominal reflexes were absent; the fundi were normal; the head was not enlarged; and the anterior fontanelle was closed. The pulse was irregular, the rate being 80. Respirations were sighing, and she yawned frequently. She was still in a stuporous state but could be made to swallow fluids. Lumbar puncture was performed and 3.5 ml. of clear colourless fluid was withdrawn. It contained protein, 40 mg. per 100 ml.; chlorides, 620 mg.

per 100 ml.; and 2 cells per c.mm. Culture was sterile. A blood count showed: red cells, 2,700,000 per c.mm.; Hb, 44%; platelets, 102,000 per c.mm.; white cells, 20,000 per c.mm. (neutrophils 70%, lymphocytes 26%, monocytes 4%). A blood culture was sterile.

By next morning her colour was better and she was easier to feed, but there was still no spontaneous activity and the neurological signs were unchanged. The improvement was not maintained and by evening she had become ashen grey and semicomatose. The pulse had slowly risen during the day to 120 and the temperature to 102° F. (38.9° C.). At a second lumbar puncture the pressure was 180 mm. and the fluid was clear and colourless.

On December 11 two small flame-shaped haemorrhages were seen in the temporal half of the right fundus, one above and one below the disk. There was ptosis of the left lid; both pupils were dilated, but reacted briskly to light. The eyes still deviated to the left. There was now a right flaccid hemiparesis. The right plantar response was still extensor. The temperature had risen to 103.8° F. (39.9° C.) and the pulse to 130. Coma deepened and the child could be fed only with a stomach tube. At 9 p.m. clonic convulsions involving the right arm and leg occurred and lasted several minutes. The child died at 1 a.m. on December 12 after a further convulsion.

Pathological Findings.—At necropsy the body was that of a pale well-nourished child. A large, recently clotted subdural haemorrhage was found on the left side, extending from the posterior part of the frontal lobe backwards and downwards over the temporal and occipital lobes. There was no evidence of membrane formation. On a parietal branch of the left middle cerebral artery a small tag was seen. When this was teased out under water and examined with a hand lens no aneurysm could be made out and no tear was seen. Its significance remains obscure. The brain was normal and no fracture of the skull was found. The other organs were normal macroscopically. On microscopy the spleen and liver were normal and there was no evidence of extramedullary haemopoiesis.

Comment

The clinical picture was one of a rapidly progressive intracranial lesion with localizing signs. Subarachnoid haemorrhage and meningitis were excluded on the C.S.F. findings. The impairment of consciousness, combined with right-sided hemiplegia and incomplete left third nerve palsy, was taken to indicate the development of a tentorial herniation due to increasing intracranial haemorrhage rather than an inflammatory or thrombotic process. Diagnosis of a surface clot, as opposed to cerebral haemorrhage, was rendered difficult by the absence of a history of injury and by the acuteness of the illness; their differentiation is discussed below. No cause, such as scurvy or purpura haemorrhagica, could be found to account for the anaemia and the tendency to bleed, nor was the source of subdural bleeding discovered at post-mortem examination.

Discussion

This case illustrates the need to consider a rapidly forming subdural haematoma as an alternative diagnosis to spontaneous cerebral haemorrhage in the young, who are so liable to unwitnessed and unheeded injuries. When the clinical pictures are compared it is surprising that their differentiation is so seldom discussed. Both have an acute onset with disturbance of consciousness and share the secondary effects of intracranial bleeding, such as fever, leucocytosis, and retinal haemorrhages. Localizing signs are expected in cerebral apoplexy and are also common with subdural bleeding. Neither the progression of the focal or general signs nor the presence

of a unilateral dilatation of the pupil can always be relied upon to distinguish between the two lesions. The findings on lumbar puncture may be more helpful. Few red cells should be present in the cerebrospinal fluid in acute subdural haematoma without brain laceration, in contrast to the grossly bloody fluid found by Aring and Merritt (1935) in over 70% of cases of cerebral haemorrhage. The special methods of investigation are disappointing once puncture of the subdural space becomes impossible owing to fusion of the cranial sutures. Storch and Munro (1938) found encephalography of little use in all but large chronic subdural haematomata. Angiography is capable of yielding valuable information, but is technically difficult in a gravely ill child and is likely to lead to dangerous delay in operating.

The practical importance of this problem is brought out by Sheldon's (1927) analysis of 10,150 consecutive post-mortem examinations at the Hospital for Sick Children, Great Ormond Street. He found 50 cases of intracranial haemorrhage in children under 12 years of age; of these, only eight were attributed to trauma. The number of cases in the first year of life was much greater than in subsequent years even if those due to labour are excluded. He found that, in contrast to the commonness of intracerebral haemorrhages in adults, in his series the majority of haemorrhages occurred outside the brain substance. This difference in site is doubtless to be attributed to the rarity of vascular degenerations in childhood. Of the 50 cases of haemorrhage 1 was extradural, 11 were subdural, 16 were subarachnoid, 16 were cerebral, 4 were cerebellar, and 2 were in the brain stem. Eight of the intracerebral haemorrhages resulted from embolism during the course of malignant endocarditis and one was associated with chronic nephritis.

These figures, if representative, suggest that if these diseases can be excluded an intracranial haemorrhage in a child is twice as likely to be outside the brain as in its substance. This fact, when coupled with the difficulties of clinical diagnosis, leads us to believe that if a child has an acute progressive intracranial lesion and the C.S.F. is clear, there is no alternative to exploration of the subdural space on one or both sides, even if this policy results in a certain number of unnecessary operations.

Summary

A case of acute subdural haematoma occurring in infancy is described.

Difficulties in diagnosis are discussed.

It is shown that a child with symptoms and signs of a progressive intracranial lesion and with a clear C.S.F. should be regarded as an immediate surgical problem, even in the absence of a history of trauma.

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The need for varied and interesting play material for very young children is stressed in a new booklet—*Play with a Purpose*—published by H.M.S.O. for the Ministry of Health (price 1s.). It has been written mainly to help and advise those in charge of groups of young children in nurseries, and shows the value of simple play material in helping the all-round development of the child. Pictures of the type of toys most suitable to the child at various stages of his development are accompanied by many useful hints.

A CASE OF HYPERSENSITIVITY TO PETHIDINE IN A WOMAN IN LABOUR

BY

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Pethidine was first introduced as a spasmolytic and analgesic drug by Eisleb and Schaumann (1939). Since then the pharmacology and clinical possibilities of the drug have been fully investigated. Among its other uses pethidine has been found to be of great value as an analgesic in labour. This was first described by Benthin (1940); since then other workers have confirmed the importance of pethidine in this field, so that now it is probably the most widely used analgesic in labour, and can be given by midwives on their own responsibility.

The comparatively mild toxic effects of pethidine are always given favourable emphasis in accounts of the use of this drug. In animals, however, with the use of larger doses per kilogram than in man, pethidine causes hypotension, respiratory depression, and bradypnoea. In very large doses apnoea may occur (Gruber, Hart, and Gruber, 1941). In man a marked transitory fall of blood pressure also occurs, within three to four minutes, when the drug is given intravenously, and a moderate fall, maximum in 40 minutes, when given by intramuscular injection. This was reported by Branwood (1943), but in his series no alarming symptoms were noticed.

Cases have been described in which pethidine has caused a more severe reaction—e.g., apnoea, severe hypotension, or collapse—but these have occurred after giving it by intravenous injection. Therefore it seems of interest to record the following case of hypersensitivity to pethidine in a woman in labour, in which the drug was given by intramuscular injection.

Case History

A married woman aged 24 was admitted in labour at full term on January 2, 1951. She had felt well throughout her pregnancy, which had been quite normal. Labour began at 1.30 a.m. on January 2, and the patient was admitted to hospital at 6.30 a.m. On admission the clinical findings were as follows: B.P. 120/80; urine, N.A.D.; height of fundus, 40 weeks; presentation, vertex L.O.A.; head engaged; foetal heart heard; pains 1 in 10. On rectal examination the cervix was 3 cm. dilated. Pethidine, 100 mg., was given by intramuscular injection at once.

Labour progressed normally, and the cervix was fully dilated at 8.50 a.m. The patient was then encouraged to bear down with the pains. At 9.40 a.m. she suddenly became cyanosed and developed a transient urticarial rash on face, abdomen, thighs, and legs in quick succession. The rash had disappeared from the face by the time it appeared on the legs. She complained of feeling faint. The pulse was imperceptible, and the blood pressure could not be recorded. Until this time the foetal heart had been heard, but five minutes after this attack began she delivered normally a fresh stillborn male infant. The placenta and membranes followed 20 minutes later.

After delivery the blood pressure still could not be recorded, and the urticarial rash appeared again on the face, with some oedema of the lips and eyelids. This gradually disappeared, and by 10 a.m. a weak pulse could