6th hour (Schönebeck *et al.*, 1973)—that is, much less than the lowest dose with teratogenic effects in rats. Of course it cannot be excluded that the drug might have a teratogenic effect in man at a lower dose.

Our patient was informed of these facts. Yet she was strongly determined to carry her pregnancy through, and 5-FC therapy was definitely indicated because of her septicaemia. It should be emphasized that the pregnancy had advanced past the period of gestation most dangerous from the teratogenic point of view.

As her SGPT values were raised even before the institution of 5-FC treatment it is not likely that 5-FC should have caused the rise in transaminase. It was probably a result of liver toxicity from the candida septicaemia.

Like most other drugs 5-FC should not be given to women during early pregnancy. If a patient develops a yeast fungus septicaemia in early pregnancy and if there is strong indication for keeping the pregnancy going, we find 5-FC to be the safest preparation for the time being.

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Expanding Skull Fracture of Childhood

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Expanding skull fractures in children have been described for many years, but mostly in specialist neurological or radiological journals or in overseas journals (Lende and Erickson, 1961; Bandi *et al.*, 1969; Gruber, 1969; Goldstein *et al.*, 1970; Adeloye, 1971). The purpose of this report is to bring the condition to the attention of a wider readership.

Case Report

A boy aged 1 year 4 months was admitted to hospital having fallen from a first floor window on to a hard surface. His birth and progress to the time of the accident had been normal. On admission he was deeply unconscious, pale, and sweating and had a large haematoma in the right occipital region of the scalp. An *x*-ray picture showed a long linear fracture in the right parietal region of the skull (fig. 1). Frequent right-sided convulsions were treated with intravenous phenytoin sodium, and the child gradually regained consciousness over the next three days. He was then found to have a mild left-sided hemiparesis and a left homonymous hemianopia.

He was next admitted to hospital aged 2 years 8 months because of a prolonged left-sided convulsion, having been previously well since the time of the accident. There was now a palpable defect in the right parietal region of the skull and a corresponding defect was seen on an *x*-ray picture (fig. 2). Repeat *x*-ray examination 21 months later showed the defect to be unchanged (fig. 3).

After the second admission the child remained free of fits for 19 months on phenytoin sodium, but then developed brief left-sided clonic fits without loss of consciousness which rapidly increased in frequency up to five a day. The addition of phenobarbitone and sulthiame in turn to the anticonvulsant therapy produced no improvement, but the attacks stopped soon after giving acetazolamide, and at the time of writing he has been free of attacks for nine months on phenytoin sodium and acetazolamide.

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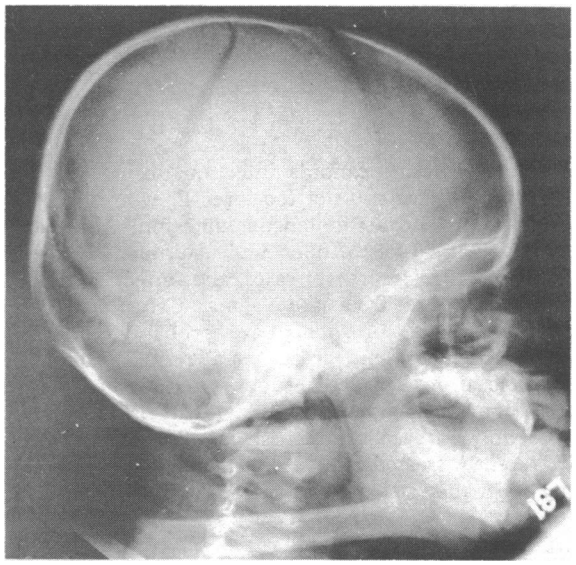

FIG. 1—*X*-ray picture of skull at time of first admission.

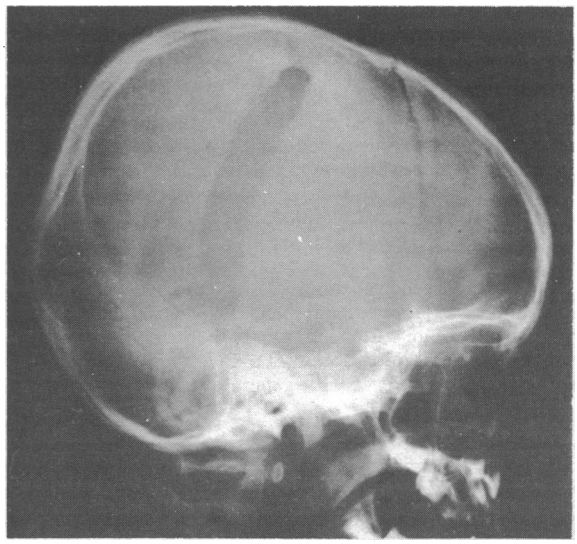

FIG. 2—*X*-ray appearance at time of second admission.

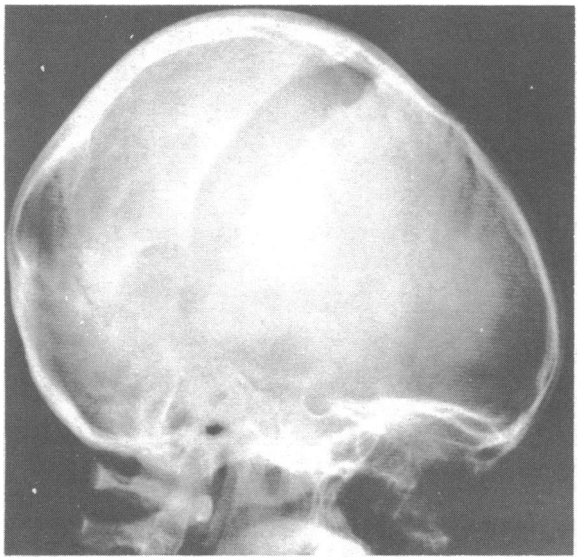

FIG. 3—Appearances at follow-up examination.

On examination at 4 years 6 months he was slightly overactive. He had a mild left-sided hemiparesis and minimal weakness of the left external rectus oculi muscle. The left optic disc was abnormally pale and the right normal. Visual acuity 3/6 in the right eye and 3/36 in the left had not changed significantly over a period of two years. There was a left homonymous hemianopia. A non-bulging defect in the right parietal region of the skull was palpable and measured 9 cm × 2 cm.

Comment

The first report of an expanding skull fracture was published in 1816 by John Howship (Lende and Erickson, 1961). The condition is seen only in children, though similar lesions have been produced experimentally in mature animals (Freeman *et al.*, 1969). A variety of names have been applied to the condition, including leptomeningeal cyst, subdural hygroma, fibrosing osteitis, traumatic meningocele, and cerebrocranial erosion (van Wigmont, 1972), but recently the term expanding (or growing) skull fracture has become accepted.

Tavernas and Ransohoff (1953) suggested, as a result of their clinical observations, that the lesions were the result of a dural tear under the fracture and subsequent herniation of a pouch of arachnoid between the edges of the bone with consequent erosion of bone aided by the pulsation of the brain. Goldstein *et al.* (1967) found in experiments on puppies that incision of both dura and arachnoid was necessary to produce an expanding fracture, but extension of the incision into the brain or into the lateral ventricle did not increase the likelihood of this occurrence.

The incidence of this condition after skull fracture in childhood is unknown, but Vas and Winn (1966) recalled 26 children with previous skull fractures for repeat *x*-ray examination and found one expanded fracture.

Most authors have recommended surgical repair of the cranial defect, but Ramamurthi and Kalyanaraman (1970) reported on four patients who did well without surgery, and they pointed out that air studies almost invariably show enlargement of the lateral ventricle on the side of the fracture indicating cerebral atrophy resulting from brain damage sustained at the time of the injury rather than subsequent compression by a cyst. They thought that surgery was unnecessary in the absence of a large defect or bulging of the scalp. Stein and Tenner (1972) disagreed with this view. They described six cases in which brain tissue was found within the defect by arteriography, and argued that in the absence of surgery such brain tissue would be liable to further trauma and scarring. In three of their patients the size of the defect and unsightly bulging of the scalp would alone have been sufficient indication for surgery. The fourth was operated on because of a swelling and cranial defect 10 days after injury, no other clinical details being given, and of the last two patients one had been asymptomatic and unaware of the defect in the three years since his accident, and the other had a moderate-sized skull defect which had not enlarged over three years and a minimal hemiparesis which was also non-progressive.

The present patient is not troubled by his cranial defect, his fits are well controlled medically, and his neurological signs have not progressed. Surgery is therefore not thought necessary.

I wish to thank my colleague, Dr. J. S. Oldham, for asking me to see this patient.

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