Discussion

In these patients the diagnosis of migraine is indisputable, but, largely because of the widespread and erroneous belief that complicated migraine is associated with vascular anomalies, the occurrence of such incapacitating symptoms only after blows on the head has in each patient given rise to fears of some more serious condition. The complete absence of physical signs and of sequelae did not suggest that elaborate investigations were needed.

There are few references to similar symptoms. Whitty (1967) mentioned the case of a patient who had migraine induced by boxing in his youth, and Graham (1968) referred to "youngsters with classical migraine who develop a characteristic attack when hit on the head in a football match," but presumably not while attempting to head the ball, which under American rules would result in more comprehensive injuries.

Migraine induced in this way is not common among footballers and is not mentioned in standard texts on sporting injuries. Inquiry at two Football League clubs did not reveal any further examples, but it cannot be known how often a promising career in football or boxing is given up on this account. In the professional footballer, threatened with the loss of an enjoyable and lucrative occupation, attempts at prophylactic treatment are clearly justified, but in my limited experience only doubtfully successful. A boxer subject to visual field defects could scarcely be encouraged to pursue so dangerous a career.

I found that in the traditional paramedical lore of the football trainer there is some confusion between "concussion" and headache. The ball weighs from 14 to 16 ounces (400 to 450 g) at the beginning of the game, and although the modern waterproof ball does not increase in weight by the 20% common with the leather ball on a wet pitch, the impact, even when travelling at the quite ordinary speed of 30 miles (48 km) an hour, is not inconsiderable. The footballer is trained to meet the ball, from whatever direction it comes and whatever its intended destination, in a re-

stricted area in the midline in the frontal region. The neck muscles are contracted and the force of the contact may be increased by a sharp movement of the head. It is claimed that correct heading of the ball avoids all ill effects but in a recent 15-minute demonstration by five professionals arranged for my benefit one finished up with a headache.

My patient (Case 1), a most skilful player, was certain that it was the accidental unexpected blow that was most likely to produce an attack. The implications of migraine prodromata induced by minor head injury have been discussed by Haas and Sovner (1969), who drew attention to their occurrence in children and to the understandable but needless alarm that results. They inclined to the view that the blow caused traction on the arteries at the base of the brain, with subsequent spasm. It is reasonable to suppose that an unexpected blow to the side of the head would be more likely to cause arterial distortion than the anticipated and minutely controlled impact of deliberate skilled heading. Other mechanisms and factors may be involved, including violent physical exertion, but the time relations clearly implicate the trauma as the immediate trigger. Any unitary theory of the cause of the migraine attack should therefore take into account migraine occurring immediately or within a minute or two after minor head trauma and at no other time.

My thanks are due to Mr. Denis Follows, Secretary of the Football Association, for information and to Mr. Kenneth Fish, of Oxford United Football Club, for a practical demonstration of heading a football.

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MEDICAL MEMORANDA

Unusual Presenting Symptoms in Neuroblastoma

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Although neuroblastoma is one of the commonest tumours in children (Willis, 1962) the diagnosis is often made late in the disease when there are already widespread metastases. This is because the presenting symptoms are so variable (Thurman and Donaldson, 1967), and in the early stages of the disease they are often non-specific. Careful history-taking often discloses that the child has had several weeks or even months of vague ill health sometimes associated with

intermittent abdominal pain or variable bone pains, which may be accompanied by a limp.

Neuroblastoma in the mediastinum may cause compression of the superior vena cava or lead to collapse of a lung from pressure on a main bronchus. Occasionally Horner's syndrome may result from pressure on the cervical sympathetic chain. Ptosis or unequal pupils in a baby are not very obvious signs and do not at once suggest a diagnosis of mediastinal tumour.

Abdominal neuroblastoma arising in the adrenal gland or from the sympathetic chain may cause only a small palpable tumour when there are already widespread bone metastases present. Where there is a large abdominal tumour this has usually infiltrated surrounding structures by the time of diagnosis and may have extended through the intervertebral foramina causing neurological signs.

The more generalized symptoms may be due to the leucoerythroblastic anaemia caused by bone marrow infiltration by malignant cells. The child may be pale, lacking in energy, and failing to gain weight. Occasionally secretion of pressor amines by the tumour itself may cause hypertension with headaches and chronic diarrhoea.

Two cases that presented with particularly unusual symptoms are reported.

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Case 1

A girl aged 4 years was admitted to the Hospital for Sick Children for the investigation of blindness. Two months previously she had complained of aching legs and had become nervous and unco-operative. Treatment with amitriptyline (Tryptizol) had partially relieved these symptoms, but four days before admission she had begun to bump into things and could no longer feed herself because she could not see.

On admission she had minimal vision in both eyes and could not pick up small objects. She could just distinguish colour. The pupils were equal but there was no response to light in the right eye and only a minimal response to light in the left eye. Adduction was impaired in both eyes. The fundi were normal. No other cranial nerves were involved. The deep tendon reflexes were increased but the abdominal reflexes were absent and the plantar responses were flexor. Sensation was unimpaired.

The haemoglobin was 10.2 g/100 ml and the W.B.C. 4,900/mm³ (normal differential). The blood film showed a leucoerythroblastic anaemia. The E.S.R. was 60 mm in 1 hr. An x-ray picture of the skull showed multiple small lytic areas in the bones of the vault and base of the skull, the pituitary fossa was poorly outlined, and the optic foramina showed poorly-defined margins. Skeletal survey showed multiple osteolytic lesions in the pelvis and long bones.

On air encephalography the lateral ventricles were symmetrically dilated and the anterior recesses of the third ventricles were displaced upwards and deformed by a suprasellar mass. The 4-hydroxy 3-methoxy mandelic acid (HMMA) excretion was 160 µg/24 hr (upper limit of normal at this age 3.3 µg/24 hr).

During her stay in hospital she became completely blind. The left pupil became fixed and both eyes became proptosed. Her condition rapidly deteriorated and she died a month later. Permission for necropsy was not obtained.

Case 2

A boy aged 10 years had developed intermittent pain in the right loin two months before admission to this hospital for investigation of possible spinal abscess. The loin pain became worse and he began to limp and complain of pain in his back and knees. He ran an intermittent fever and had "night sweats."

Examination showed an obviously unwell child with pallor and sweating. He had wasting and weakness of the muscles of both legs with bilateral ankle clonus. The haemoglobin was 9.0 g/100 ml and the W.B.C. 4,000/mm³ (normal differential). Blood film showed a leucoerythroblastic anaemia. The E.S.R. was 55 mm in 1 hr. X-ray examination of the spine showed narrowing of the intervertebral space between the 11th and 12th thoracic vertebrae.

An extradural abscess was suspected because of the fever and raised sedimentation rate. Aspiration of pus was attempted without success. He was then transferred to this hospital for myelography. A smooth compression at the level of the body of D. 11 was found. Exploration showed no extradural collection. The bones in the area were noted to be soft and a biopsy specimen was taken. This showed destruction of the trabeculae by a malignant neoplasm composed of round cells suggestive of metastatic neuroblastoma.

Further investigations at this stage showed an HMMA excretion of 6.3 µg/24 hr (upper limit of normal at this age 4.5 µg/24 hr). A skeletal survey showed osteolytic and sclerotic lesions throughout the pelvis and osteolytic lesions in the ribs and long bones.

He was given a short course of palliative radiotherapy to relieve

the bone pain and offset the impending paraplegia. A blood transfusion was also given and weekly intravenous injections of vincristine and cyclophosphamide were begun. His condition rapidly deteriorated, however, and he died a month later. Permission for necropsy was not obtained.

Comment

The diagnosis of neuroblastoma may be delayed because it is not thought of as a possibility in a child with unusual symptoms. In Case 1 it was at first thought that the patient had a suprasellar tumour or optic glioma. Blindness is an extremely rare presenting sign of neuroblastoma (Shubert *et al.*, 1969). The symptoms in Case 2 at first suggested an extradural abscess. An extradural tumour in the spine of a child, however, is commonly due to neuroblastoma or less frequently to lymphosarcoma (Marsden and Steward, 1968).

The diagnosis in both children was made by the presence of widespread bone deposits together with a raised excretion of HMMA. A bone marrow biopsy was not done in either case, but this might have provided further confirmatory evidence with infiltration by malignant cells. Both children had the leucoerythroblastic anaemia which is typical of malignant marrow infiltration.

Neuroblastoma should be considered as a possibility in any unexplained neurological condition. Persistent pains in the bones or joints or a limp should lead to a full skeletal survey to detect all lesions. The discovery of anaplastic cells in the bone marrow may lead to confusion with acute leukaemia, as occasionally may multiple bone lesions.

Unexplained weight loss, fever, anaemia, hypertension, and recurrent diarrhoea may all be presenting signs of neuroblastoma which require further investigation.

Estimation of the urinary catecholamines should always be done in these cases (Gitlow *et al.*, 1970). Although not positive in every case of neuroblastoma this provides a useful screening test together with a skeletal survey and bone marrow biopsy. In some patients the primary tumour is not obvious and the diagnosis depends on these investigations alone.

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