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Computed tomography in childhood epilepsy

Sir,

Children attending the school at the David Lewis Centre for Epilepsy are a selected group, as with few exceptions they are residential pupils from various parts of the United Kingdom and therefore are likely to have severe and often multiple handicaps. It seemed probable that a group of such children might show a high incidence of significant abnormalities on computed tomography.

The children attending the school at the David Lewis Centre and examined by computed tomography over several years numbered 222; there were 154 boys and 68 girls, aged 7 to 19 years. The results showed that 152 were normal. There were 28 with evidence of some degree of generalised atrophy and 30 with focal atrophy. Nine showed calcification and in three the findings were compatible with a cerebral tumour. Abnormalities in a third of the children is in agreement with the findings of Bachman *et al*[1] and Yang *et al.*[2] Bachman *et al* studied 98 children with chronic seizure disorders, and computed tomograms identified structural abnormalities in 30%, almost half having generalised or focal atrophy. Two per cent showed possible evidence of unsuspected cerebral tumours. In the study of Yang *et al*, 256 computed tomograms were performed to aid the evaluation of children with seizure disorders. There were abnormalities in 33% that were found mainly among those with partial seizures and generalised seizures of known aetiology, but also in those with neonatal seizures and in those with abnormal neurological findings and focal slowing shown on electroencephalography. Five patients were found to have cerebral tumours, one a porencephalic cyst, and one extraventricular communicating hydrocephalus. The use of computed tomography among children with non-specific mental retardation also seems to be unhelpful,[3] but when the mental retardation is associated with infantile spasms the scan is often abnormal, showing evidence of tuberous sclerosis or agenesis of the corpus callosum.[4]

Therefore it seems unlikely that a selected group of children such as those attending a special residential school will show more abnormalities than those seen in hospital outpatient clinics. Also the use of routine computed tomography in the investigation of children with epilepsy seems unjustified, although it will be indicated if there are symptoms and signs suggestive of a focal lesion. Twenty two children who showed thickening of the skull vault, and often proliferation of the intracranial sinuses, and 25 children with evidence of cerebral atrophy were assessed for correlations with the antiepileptic drugs that had been given and with the possible timing of the damage to the brain. Phenytoin and carbamazepine had been given to an equal number in both groups, which did not support the role of these drugs in causing thickening of the skull bones. The onset of seizures was more frequent during the first year in the first group, which may suggest an impairment of cerebral development at an early stage, which is also indicated by the thickening of the bones.

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Henoch-Schönlein purpura secondary to trauma

Sir,

The aetiology of Henoch-Schönlein purpura (HSP) is controversial. Various bacterial and viral infections together with drug and food sensitivity have been identified as possible aetiological factors.[1] No mention of trauma as a causative agent has been found in the literature. It is postulated that a traumatic event produces a shower of antigenic material of joint contents or tissue breakdown products which may initiate HSP.

We describe two cases where trauma preceded the symptoms of HSP.

Case reports

CASE 1

A 2 year old boy was brought to casualty with a limp after a fall the previous day. He had a swollen and tender right ankle. Radiological investigation showed an undisplaced fracture of the metaphysis of the right fibula. He was treated conservatively and later discharged. Two days later he returned to casualty with a painful left ankle. He was irritable, his temperature was 37·1°C, and he had a maculopapular rash. HSP was diagnosed and subsequently he developed abdominal discomfort, lymphadenopathy, proteinuria, and purpura. The bruise overlying his right ankle was yellow whereas the purpura of the HSP were blue/black.