ABC of 1 to 7 (Revised)

H B VALMAN

RECURRENT HEADACHE

Migraine

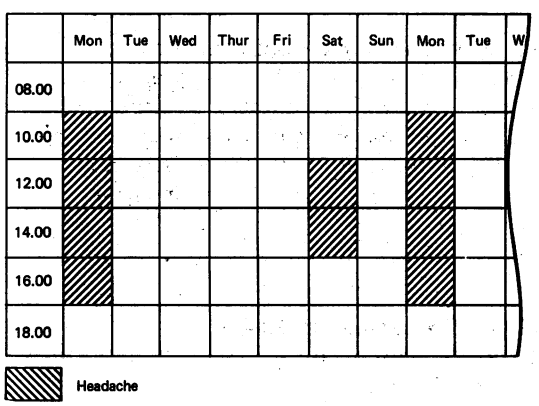
Emotional tension

Muscle contraction

Intracranial pathology

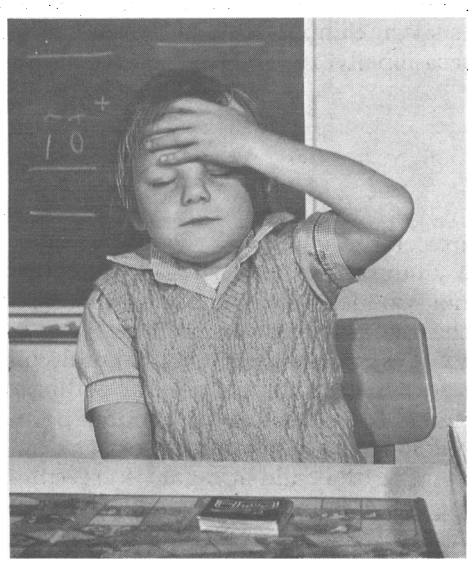
Any acute illness with pyrexia may cause headache, but if there is drowsiness, vomiting, photophobia, neck stiffness, or purpura an emergency lumbar puncture needs to be considered to exclude meningitis. Recurrent headaches are due to migraine, emotional tension, muscle contraction, or intracranial pathology. Emotional factors may precipitate attacks of migraine. Muscle contraction headaches occur if there is repeated clenching or grinding of the teeth. Detailed physical examination is essential on the first visit, and reassessment is needed during the first six months after the onset of headaches to exclude a cerebral tumour which did not produce localising symptoms or signs initially. The blood pressure should be measured and the fundi examined in every child with headache. Migraine occurs in about 4% of children, and tension headaches probably have about the same incidence.

Migraine



The pain of migraine is usually accompanied by nausea or vomiting and is relieved by sleep. There is often intolerance to light or noise and there may be marked pallor. The pain lasts for hours and there is complete freedom from pain between attacks. In about 20% of the patients there is a hemicranial distribution of the pain, and in about another 20% there is vertigo or light-headedness. Only about 5% of children with migraine have a visual aura. Migraine can occur at any age, but it is rare under the age of 5 years.

Ninety per cent of children with migraine have parents or siblings with this condition, so the absence of a family history throws some doubt on the diagnosis. As 50% of all children have a family history of migraine the *presence* of this history is not helpful in diagnosis. Although they may have been called migraine, the details of the relatives' headaches may show that they have the features of emotional tension headaches.



Psychological stress is the commonest trigger factor of attacks, and school is often implicated. The child may have difficulty in keeping up with his peers or may fear impending examinations. Children are often seen by a doctor for the first time at the beginning of the new school year in September, but in other families the mother may cope until March or April. Some of these children are progressing well at school but pursue a very hectic life afterwards. The importance of specific foods is controversial but a mother may have observed that a particular food such as chocolate or cheese may consistently precipitate symptoms. This occurs in about 10% of children. Provided only one type of food is implicated, it can be excluded from the diet. Any more extensive alterations should be supervised by a paediatric dietitian. A head injury or acute upper respiratory tract infection may precipitate a series of attacks, but the importance of acute sinusitis either as a trigger factor in migraine or as a specific cause of recurrent headache has probably been exaggerated. Physical activity to exhaustion, mild hypoglycaemia due to missing a meal, excessive exposure to sun, or a lack of sleep may precipitate attacks in susceptible children.

Avoid trigger factors

Paracetamol±metoclopramide

Propranolol

Management

There are no abnormal signs on examination and no investigations are indicated when the diagnosis is clear clinically. The diagnosis is explained to the whole family, including the child, and it is pointed out that most children have exacerbations of six months' duration within a 2–4 year period of school-related exacerbations followed by a remission which may last between nine years and indefinitely. Avoidance of trigger factors may need exploration with the help of a school report and sometimes assessment by a psychologist. If the symptoms have been present for less than six months a further physical examination will be needed until enough time has elapsed to exclude an intracranial lesion. Although this possibility needs to be considered, it need not be transmitted to the parents, but many parents will be worried about the possibility of a tumour and the value of a normal examination can be emphasised.

Treatment of an acute attack is more likely to be effective if it is given early. A supply of an anti-emetic (for example, metoclopramide) and paracetamol should be kept at school as well as at home. If this is not effective the child should be allowed to lie down in a darkened room for half an hour. If there are several attacks each week regular continuous prophylactic treatment with propranolol may be recommended for six months by a paediatrician. Behaviour modification techniques have been successful where parents are motivated and staff with the necessary skills are available.

Emotional tension

The headache is often present every day, usually starting in the afternoon and continuing to the evening. It is described as an ache, tightness, or pressure affecting any part of the head. It is commonly frontal but may be felt in the temporal or occipital regions. Poor school attendance is common, with absence from school for weeks at a time. Evidence of environmental factors causing anxiety at school and at home should be sought, and there may be additional physical symptoms such as pain in the abdomen or limbs which complete the picture. There may be overt symptoms of psychiatric disturbance such as depression, disruptive behaviour in group activities, or destruction of property.

Management

The absence of physical signs confirms the diagnosis and helps the family to accept it. Specific investigations are seldom required, but a further assessment is needed to allow the parents to consider any further relevant factors, to discuss the school report with them, to plan further management, and to confirm the absence of abnormal physical signs if the history is short. Simple changes in the child's routine or environment or, occasionally, referral to a child psychiatrist, may be needed.

Intracranial lesions

Physical examination

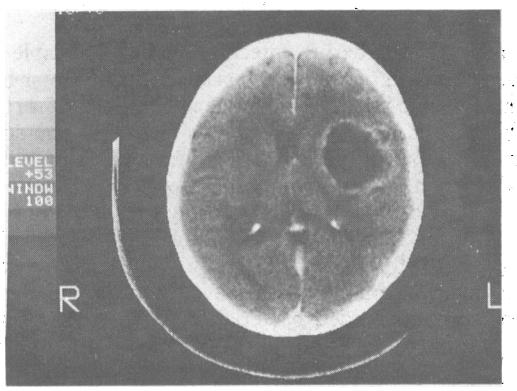
Fundi

Gait

Hemiparesis

Most intracranial lesions are cerebral tumours or vascular malformations, but a few are subdural haemorrhages or intracranial abscesses. These lesions do not produce a specific clinical picture, but there are some pointers which make the diagnosis more likely.

Abnormal physical signs are present in the majority of children with intracranial lesions either when they are first seen or within four months of the onset of symptoms. About half of the children have papilloedema, and other common signs are disturbances in gait or hemiparesis. Headache which wakes the child at night, is present on waking in the morning, or is aggravated by coughing suggests an intracranial lesion.



The following are indications for referral to a paediatrician or paediatric neurologist, who will usually arrange computed tomography of the brain as the first investigation.

(1) Abnormal neurological signs during or after headache.
(2) Fits with headache.
(3) Nocturnal or early morning headache, especially if the history is shorter than six months or if the headaches are increasing in frequency or severity.
(4) Recent school failure, change in behaviour—especially apathy or irritability—or failure to grow in height.
(5) Change in quality or distribution of headache.
(6) Extremely severe incapacitating headaches.
(7) Age less than 5 years.

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Lesson of the Week

Eczema herpeticum: a potentially fatal disease

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Forty to fifty per cent of children are seropositive to herpes simplex virus type 1 by the age of 5, and over 90% of urban dwellers are seropositive by adulthood.[1] The incidence of children with eczema contracting herpes simplex is high. In most cases this leads to a mild disorder, which may go unrecognised. Rarely, however, it may present as a severe fulminating disease with widespread lesions associated with a recognised mortality.[2] Early identification and treatment may be life saving. We report on a child with eczema in whom herpes simplex infection was not diagnosed until a week after the infection had erupted.

Herpes simplex virus infection of children with atopic eczema may lead to a serious and sometimes fatal condition; early diagnosis and treatment with acyclovir may be life saving

Case history

A 1 year old boy attending our dermatology clinic for atopic eczema was brought to the casualty department. His eczema had been managed with liquid paraffin:white soft paraffin (50:50), 1% hydrocortisone ointment, and regular baths with an emollient of liquid paraffin and lanolin (Alpha Keri oil) and emulsifying ointment. One week previously a vesicular eruption had developed and was diagnosed by his family doctor as chickenpox. No antiviral treatment was prescribed. During the week the boy had become lethargic and febrile, but despite being reviewed by his doctor two days later he continued to deteriorate.

On arrival in the casualty department he was extremely ill. He was feverish (38·5°C) and his skin was covered by a herpetiform eruption in which nearly all the vesicles had ruptured leaving a skin loss of around 20%. The clinical diagnosis was eczema herpeticum. He was 10% dehydrated with sunken eyes, reduced skin turgor, and cold extremities. Investigations showed that he had lost protein (albumin 15 g/l) and potassium (2·2 mmol/l). A full blood count disclosed bone marrow suppression (haemoglobin 82 g/l, white cell count $4·2\times10^9$/l (neutrophils 20%), platelet count 81×10^9/l). Skin swabs grew *Staphylococcus aureus* and *Streptococcus pyogenes*. No viral particles were seen on electron microscopy, but herpes simplex virus was isolated four days after culture in a human embryonic lung fibroblast and confirmed as type 1 by direct immunofluorescence (Syva UK).

He was given plasma and intravenous solutions containing potassium (7 mmol/kg/day) and began intravenous acyclovir and broad spectrum antibiotics. He was nursed in a heated room with paraffin mixture applied to reduce evaporation. Despite these measures his temperature fell to 34°C over eight hours. He developed paralytic ileus by 14 hours after admission and the potassium concentration fell to 2·0 mmol/l. Despite more potassium being given he had a cardiac arrest at 16 hours; a cardiographic recording showed asystole.

He was successfully resuscitated with intravenous adrenaline, potassium, and atropine and a regular heart beat was achieved by 11 minutes; during this time he had received cardiac massage and ventilation by an endotracheal tube. He was given sodium bicarbonate to counter a metabolic acidosis (pH 7·1). Though he started breathing spontaneously two minutes after the return of his heart beat, he was electively hyperventilated and given intravenous mannitol to reduce cerebral oedema.

During the next 24 hours he developed spontaneous cutaneous and gastric bleeding due to disseminated intravascular coagulation, confirmed by low platelet count, prolonged prothrombin time, reduced fibrinogen titres, and

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