tympanotomy was performed and a fistula was found in the foot plate of the stapes. The stapes was removed and the oval window was plugged with tempralis fascia. Since then (18 months) no further attacks of meningitis have occurred.

The second case was a girl, born following a normal pregnancy, and delivery, to healthy unrelated parents. Her development was normal except for deafness discovered at 12 months of age. She was fitted with a hearing aid in both ears. She has had three attacks of pneumococcal meningitis at the age of 21 months, 23 months, and 27 months, and was referred to the Hospital for Sick Children. Examination revealed a normal healthy child with a hearing aid in both ears. Throat, nose and right ear were normal; the left ear showed air bubbles and fluid in the middle ear. The tympanic membrane was dull. There was no other abnormality. Her investigations also were not diagnostic: haemoglobin and white cell count were normal. Serum immunoglobulins IgG and IgA were normal, IgM slightly raised. Nitroblue tetrazolium test was normal; cerebrospinal fluid (CSF) showed no cells and no organisms were cultured. Protein was 0 · 1g/1 (normal); skull radiographs and tomograms of the mastoids were normal. Metrizamide cisternogram did not demonstrate a leak into sinuses, and was normal. CSF flowed through the subarachnoid space. A technetium brain scan also showed normal CSF distribution and no abnormal accumulation was demonstrated. Hearing test confirmed severe bilateral senorineural hearing loss. She also had an exploratory tympanotomy, and this showed a leak of perilymph from the central part of the footplate of the stapes. The stapes was removed from the oval window and the cavity was plugged with tragal perichondrium and post-auricular muscle. She has remained free from attacks of meningitis for the past 42 months.

Recurrent meningitis in the presence of other systemic infections may indicate an immunological disorder. However, when it occurs without systemic infections, an anatomical defect is likely. When confronted with a child with the triad recurrent meningitis, hearing loss (especially sensori-neural) and evidence of middle ear pathology even without abnormality in the tomographic study of temporal bones, then labyrinthine fistula should be suspected. Detailed investigations, as in both our cases, may fail to show a site of CSF leak. Therefore in such a child exploratory tympanotomy should be undertaken.

We thank Mr John Evans and Mr Robert Pracy for allowing us to report these children.

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Errata

In the October issue of the *Journal of Neurology, Neurosurgery and Psychiatry* the letters by Quinn *et al* and by Plant were printed without their figures. We apologise to these authors and publish below the letters in full.

Insulin-induced hypoglycaemia does not abolish chorea

Sir: Pathological changes occur in the hypothalamus in Huntington's disease.[1] Insulin tolerance tests have been used to examine hypothalamic function in such patients, and mild abnormalities of growth hormone secretion have been described.[2–4] In the course of such an investigation, Keogh *et al*[2] noted that chorea ceased some 30 min after the insulin injection and was not evident for the next 60 to 75 min in all of the twelve patients studied. They did not think that this dramatic change was due to an altered level of consciousness, for "all patients were awake throughout the investigations and were checked repeatedly to see that they were capable of verbal communication". Subsequently, Lavin *et al*[3] described similar observations in another group of eight patients with Huntington's disease, in all of whom chorea disappeared for at least an hour within about half-an-hour of the insulin injection. Such a dramatic effect on chorea might provide some clue as to the pathophysiology of that movement disorder, so we have repeated the study concentrating on the effect of insulin-induced hypoglycaemia on the chorea.

Five patients with Huntington's disease (four males and one female; aged 30 to 70 years; with disease duration from 2 to 13 years; four on no drugs and one on tetrabenazine 25 mg three times daily) with obvious chorea were studied. After an overnight fast, blood was withdrawn for glucose estimation, and insulin (0·1 mg/kg) was injected into the opposite arm. Blood sugar and clinical response were measured every 10 min for 60 min, and then every 20 min for a further 60 min. The severity of chorea was rated using a specially designed scale described in detail elsewhere.[5] In addition the number of choreic movements occurring at rest in one selected region, such as an eye, finger or toe depending on the individual patient, was counted over a 60 sec period. Blood sugar fell below 2·0 mmol/l and symptoms and/or signs of hypoglycaemia developed in all subjects. However, the intensity of chorea did not alter. Three subjects fell asleep during the test, and chorea disappeared in two, but on arousal their chorea was of the same severity as before insulin. Unfortunately, insulin-induced hypoglycaemia had no effect on chorea in our patients.

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