The above experience does highlight the benefit of a local HDU facility. Stabilisation and close monitoring is not only good practice, essential for patient care but should reduce the work of the transport team when it arrives. When funding and patterns of care are reviewed locally, more attention should be given to ensuring that local facilities in the form of HDU beds are available. They are not mini PICUs but they do have a purpose.

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## A self-fulfilling prophesy?

Carroll and Brookfield[1] quote a widely used definition of febrile convulsion in their second paragraph: "an epileptic seizure occurring in a child aged from 6 months to 5 years, precipitated by fever arising from infection outside the nervous system in a child who is otherwise neurologically normal". The authors then go on to say that only a tiny percentage of children with febrile convulsions have meningitis. By definition though, that percentage is 0%.

I dispute the assertion that more experienced staff are less likely to recommend lumbar punctures. Over the years, most people miss the occasional case of meningitis and become doubly wary of "absence of meningeal signs" thereafter. Meningeal signs are often misunderstood too; many Senior House Officers believe Kernig sign to have something to do with pain in the back (rather than just a feeling of tightening in the hamstings). With neck stiffness, they sometimes expect the neck to be rigid rather than just slightly stiff on extreme flexion.

Even viral meningitis is very good at causing sensorineural hearing loss. Unless we routinely start antibiotics and request audiology on all children who have had a convulsion with fever, we still need to do lumbar punctures.

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## The relationship between *Helicobacter pylori* infection and iron deficiency: seroprevalence study in 937 pubescent children

*Helicobacter pylori* infection has been reported to be associated with various unexpected manifestations in childhood. One of them is iron deficiency anaemia at puberty. In 1999, we conducted a double blind, placebo controlled trial in pubescent children with iron deficiency anaemia and coexisting *H pylori* infection.[1] We found that *H pylori* eradication led to resolution of iron deficiency. We have carried out a study of seroprevalence to examine the epidemiological relationship between *H pylori* infection and iron deficiency anaemia at puberty. Haemoglobin, serum iron, total iron-binding capacity, serum ferritin, and serum IgG Antibodies to *H pylori* were measured in 937 Korean children (475 boys and 462 girls). Their ages ranged from 10 to 18 years. The prevalence of *H pylori* infection was compared between groups, based on the presence or absence of anaemia, hypoferritinemia, iron deficiency, and iron deficiency anaemia. The levels of hemoglobin, serum iron, total iron binding capacity, transferrin saturation, and serum ferritin were obtained according to the presence or absence of *H pylori* infection.

The prevalences of anaemia, iron deficiency, iron-deficiency anaemia, and *H pylori* infection were 8.1%, 9.1%, 3.1%, and 20.8%, respectively. The *H pylori* positive rates in anaemia, hypoferritinemia, and iron deficiency group were 34.2%, 29.5%, and 35.3%, respectively, compared to 19.6% in the non-anaemia group ($p=0.003$), 19.2% in the non-hypoferritinemia group ($p=0.005$), and 19.4% in the non-iron deficiency group ($p=0.001$). The *H pylori* positive rate in the iron deficiency anaemia group was 44.8% in comparison with 20.0% in the non-iron deficiency anaemia group ($p=0.001$). Haemoglobin and iron levels did not show any significant differences between the *H pylori* positive and negative groups. The serum ferritin level was significantly lower in the *H pylori* infected group ($p=0.0002$).

The associations between iron status and *H pylori* were largely restricted to girls rather than boys. We speculate that this is because female adolescents are more vulnerable to iron deficiency. *H pylori* may affect iron absorption metabolism in the stomach and exacerbate the iron deficit in adolescents, especially girls, whose iron is supplied marginally, with anaemia ensuing promptly.[2]

We believe that this is the only large scale study in children showing an association between *H pylori* infection and iron deficiency. When children at puberty are found to have iron deficiency that is refractory to iron supplementation, *H pylori* infection can be considered to be a possible cause of iron deficiency.

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## Acute ataxia complicating Langerhans cell histiocytosis

Some of the statements in the interesting short report by A Polizzi *et al*[1] can be challenged. It is incorrect to suggest that cerebellar ataxia has been reported "only occasionally" in children and that it is commoner in adults with Langerhans cell histiocytosis (LCH). Diabetes insipidus is the only CNS complication that is more common than cerebellar disease and though the precise relative incidence of cerebellar ataxia in children and in adults is unknown, because all published series are institution based, there is no reason to suspect that proportionately more adults suffer this complication. It is also misleading to suggest that the patient described by Polizzi *et al* represents a "unique" occurrence. Cerebellar ataxia may be present at diagnosis or appear during follow up and may be progressive or static. More details of the clinical and pathological spectrum of CNS involvement by LCH can be found in a recent review.[2]

As the authors point out, pituitary-hypothalamic axis involvement is caused by direct infiltration of these structures by pathological Langerhans cells ("LCH cells") and accompanying inflammatory cells. In patients who develop ataxia, cerebellar biopsy usually reveals only gliosis and demyelination, but CD1a-positive cells have been demonstrated in a few instances. Therefore, it is likely that the cerebellar lesions are caused by "LCH cell" infiltration followed by cytokine and chemokine mediated neural damage. The same sequence, with fibrosis as the end point, occurs in the liver and lungs of other LCH patients. Immune mechanisms may also be involved, as suggested by Polizzi *et al*, because CD8-positive T cells are also found in the cerebellar biopsies (Grois NG, personal communication). It is unlikely, however, that they represent the primary pathogenetic process. In other words, it is improbable that cerebellar involvement represents a "paraneoplastic syndrome" (ie an autoimmune disorder), as suggested by Polizzi and colleagues, a view supported by the fact that cerebrospinal fluid (CSF) "anti-neural" antibodies have not been detected in two studies (Grois N *et al* and Donadieu J *et al*, unpublished observations).

The combination of reticulo-nodular pulmonary shadowing and ataxia, as in this case, makes the diagnosis of LCH a real possibility. Given the onset of fatal pneumothorax soon after the child's discharge from hospital, a lung CT scan would almost certainly have shown cystic change and led to tissue diagnosis via lung biopsy or bronchial lavage. With chemotherapy, the prognosis for pulmonary LCH is usually favourable, even if complicated by pneumothorax.[3]

It is generally recommended that patients with suspected LCH are referred to paediatric oncologists and/or managed on the international evidence-based trials of the Histiocyte Society, which include recommendations for the management of CNS complications.[2–4] More details of the Histiocyte Society's activities and their contact address can be found on the Society's website: www.histio.org-society.

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