techniques such as the thiobarbituric acid method or boronate affinity chromatography is indicated.

References


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Rotavirus encephalitis

Sir,
Ushijima et al describe rotavirus encephalitis1 and benign convulsions in children with rotaviral gastroenteritis.2 We report briefly a further case of probable rotavirus encephalitis.

A boy of 20 months was admitted with a two day history of profuse watery diarrhoea. Admission was precipitated by a tonic clonic fit lasting about 10 minutes. There was no other history of fits in the child or his family, and his psychomotor development had been normal. A second fit occurred at the referring hospital and he had two further fits within 24 hours of admission to the infectious disease unit. He never became febrile.

On examination he was not feverish and fully conscious. Examination of cerebrospinal fluid was entirely normal, plasma concentrations were sodium 135 mmol/l, potassium 4.4 mmol/l, chloride 99 mmol/l, bicarbonate 19 mmol/l, urea 4.6 mmol/l, glucose 3 mmol/l, calcium 2-21 mmol/l, phosphate 1.31 mmol/l, and haemoglobin 124 g/l; the white cell count was 10.1×10⁶/l. Chest and skull x ray pictures, and computed tomogram of the brain were all normal. Cultures of urine and blood were sterile. No pathogens were cultured from swabs of the throat or nose, and no virus was detected in cerebrospinal fluid, urine, or swabs from the throat and nose. Paired serum samples showed no evidence of recent infection with rubella, mumps, herpes hominis, varicella zoster or measles virus, nor with influenza virus A or B, adenovirus, chlamydia, Clostridium burnetti, or Mycoplasma pneumoniae. An enzyme immunoassay showed rotavirus in the faeces but no rotavirus antibody was detected in the cerebrospinal fluid.

The first electroencephalogram showed a general excess of slow activity most marked in the left temporal region and suggestive of encephalitis. Follow up studies showed gradual improvement with return to normal rhythms four months after admission. During this time his behaviour was aggressive and disruptive, but this settled as the results of the electroencephalograms improved. He continues to develop normally at the time of writing.

The patient was treated with intravenous acyclovir for five days and phenobarbitone. Anticonvulsant treatment was discontinued at the onset of the behavioural abnormalities which did not improve. In retrospect it seems our patient had benign convulsions associated with rotavirus encephalitis but without specific antibody in the cerebrospinal fluid, as described by Ushijima et al.2

I thank Dr J Stevenson for permission to report this case and the Regional Virus Laboratory, East Birmingham Hospital for the ELISA for rotavirus antibody.

References


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Noonan’s syndrome and neurofibromatosis

Sir,
In the February 1987 issue Shuper et al described a 12 year old boy alleged to be suffering from Noonan’s syndrome and neurofibromatosis.1 There seems little doubt that their patient had Noonan’s syndrome but the evidence for coexisting neurofibromatosis is based solely on the presence of 20 or more cafe au lait spots, 10 of which we are told were, at the time of writing, over 1.5 cm in diameter.

In the introduction these authors state, ‘Multiple cafe au lait spots are regarded as pathognomonic of von Recklinghausen’s neurofibromatosis and have not been reported with Noonan’s syndrome.’ (my italics). In support of this statement, which is incorrect, they quote a paper by Mendez et al.2 Dr Mendez, however, in the same issue of the American Journal of Medical Genetics (July 1985) wrote a review article in which she stated ‘...Noonan’s syndrome individuals frequently have multiple pigmented moles, cafe au lait spots and other pigmented dysplasias’.3 Furthermore, in two other articles in the same issue of the journal, it is made abundantly clear that cafe au lait spots are a common finding in Noonan’s syndrome alone.4 5 Dr Mendez tells me (personal communication) that in her experience in Brazil at least 10% of patients with Noonan’s syndrome have cafe au lait spots.

The Israeli boy reported by Shuper et al should, therefore, have been regarded at that stage as having Noonan’s syndrome. As he was, however, clearly prepubertal at the time of writing, it is still possible that he may develop (perhaps in late adolescence or early adulthood) neurofibromata or indeed other manifestations of the peripheral form of von Recklinghausen’s neurofibromatosis that would then permit a diagnosis of neurofibromatosis Noonan’s syndrome, a recently described and rare concurrence.6 7

Authors commenting on cafe au lait spots in Noonan’s syndrome tend not to give details of the size of the cutaneous lesions in their case reports. In future it would
Selective examinations on starting school

Sir,

I would strongly agree with the call by O’Callaghan and Colver to abolish the routine medical examination at school entry. They describe a system of selective examination and provide some documentation about the process and about findings. Of course to prove conclusively that such a system is better than routinely examining every child at school entry would require a prospective study of some magnitude, but there is nevertheless much anecdotal evidence to support the notion that the routine medical examination at school entry is an outmoded concept that is an inefficient use of time and resources.

In Australia, as well as in England, the routine medical examination at school entry has been a longstanding tradition. It is a superficially attractive concept to examine every child at school entry, giving some children a clean bill of health and detecting problems in others that can be appropriately treated in order to prevent subsequent sequelae. A more critical analysis of this practice, however, raises a number of issues:

1. It is probable that most important problems have already been detected because most children now have access to a regular source of health care.
2. It follows therefore that there is a low yield of abnormalities detected at school entry that are both new findings and are important in terms of the child’s ongoing health and school performance.
3. The natural history of some of the conditions detected (for example, serous otitis media) is such that there does not exist an intervention which is widely accepted and which is known to make a difference to outcome.
4. Detection of problems does not necessarily result in appropriate treatment or management, because there is usually a reliance on compliance of parents for ongoing referral, etc.
5. There is overwhelming evidence that important problems at school have to do with the sequelae of chronic illness as well as developmental, behavioural, and psychosocial issues, rather than the sorts of findings detected by a routine physical examination.

A recent Australian study concluded that abolishing the school entry examination would allow a ‘redistribution of existing resources in order to concentrate more on ensuring effective management of identified problems’. There are many methodologies that could be implemented if resources were freed from school entry examinations and applied to the assessment and management of children in need. Callaghan and Colver have described one that obviously works in their school district, and it is inevitable that the needs of school children are better served by such a system than by routine examinations. The authors also argue against routine neurodevelopmental examinations at school entry—I would concur strongly with this as well. It would seem a more productive use of time to confine such detailed assessments to the small group of children who present with difficulties of learning or behaviour rather than to administer such examinations routinely.

References


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Enuresis in children

Sir,

The personal account of enuresis by a 16 year old girl is a reminder that many areas in the United Kingdom lack special facilities for dealing with this common and distressing condition. Management of enuresis requires specific treatment for the symptom and also help for family difficulties, which were evidently present here. Most children with wetting problems have a poor self image, which needs to be bolstered. If a child is referred for diurnal enuresis I ask the head teacher if someone will take him or her regularly to the toilet in a friendly discreet way. The mother is asked to do the same at home. With
Noonan's syndrome and neurofibromatosis.

T P Mann

Arch Dis Child 1988 63: 224-225
doi: 10.1136/adc.63.2.224-a

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