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

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Rotavirus encephalitis

Sir,

Ushijima *et al* describe rotavirus encephalitis[1] 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 feverish.

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 $10{\cdot}1\times10^9$/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.

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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 pigmentary 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