This case shows that within 9 months of HIV seroconversion and at a CD4 lymphocyte count within the normal range, damage to the gastrointestinal mucosa can occur. Treatment with zidovudine produced significant improvement in small-bowel architecture, this being associated with a resolution of diarrhoea and improved biochemical indicators of malabsorption.

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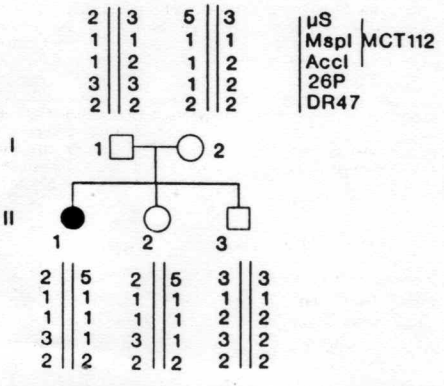
Genetic diagnosis of Friedreich's ataxia

SIR,—Friedreich's ataxia is inherited as an autosomal recessive trait. The Friedreich's ataxia locus has been mapped to chromosome 9q13–21·1,[1] and all families with typical clinical features show genetic homogeneity.[2] Two tightly linked marker loci, D9S15 (defined by probe MCT112) and D9S5 (defined by probes DR47 and 26P), have generated a linkage group within 1·4 centimorgans (cM) of the Friedreich's ataxia gene,[3,4] and Wallis et al[5] made the first prenatal diagnosis, which can be offered to families with an accuracy of 99% or more.[4,6] The disorder can also be diagnosed before symptoms develop. However, the poor long-term outlook and the lack of treatment for the disease raise several ethical issues about such studies.

In a linkage disequilibrium study of Friedreich's ataxia information about disease onset and evolution and genetic counselling about its autosomal recessive heredity pattern was provided for families. Seven families asked for predictive diagnosis of 15 seemingly healthy children aged under 15 years. Extended haplotypes were constructed with five DNA polymorphisms, three from D9S15 locus and two from D9S5 locus. Four were restriction fragment-length polymorphisms (RFLPs) and one recognised a polymorphic microsatellite sequence[7] (figure). 26P/*Bst* XI RFLP at D9S5, and the MCT112/microsatellite showed high polymorphism, with polymorphism information contents of 0·55 and 0·79, respectively. For RFLPs generating probe hybridisation,



Segregation of haplotypes at Friedreich's ataxia region of chromosome 9, showing identical pattern in subjects II-1 and II-2.

Polymorphic DNA markers are shown in the same order as numerical alleles on chromosomes.

DNA was extracted and digested with enzymes, followed by fractionation and Southern blotting onto 'Hybond-N' (Amersham International). The membranes were hybridised overnight with a DNA probe that was labelled with ^{32}P by a random labelling primed method. MCT112/microsatellite polymorphism was examined by polymerase chain reaction and products were electrophoresed on 6% polyacrilamide gel.

As expected 14 children had a different haplotype to that of the affected sibling in at least one chromosome. A 7-year-old girl showed the same chromosome haplotypes as her 10-year-old affected sister (figure). At the time of the test this girl showed knee deep tendon hyporeflexia, suggesting the presence of Friedreich's ataxia.

Predictive diagnosis in Friedreich's ataxia is a challenge for both physicians and parents since there is no specific treatment for this disease, and rehabilitation has not modified the natural history. Prediction of the development of the disease can be very stressful for patients and their parents. Thus we believe that predictive diagnosis should only be done when the physician needs to confirm clinical suspicion, as in our patient (II-2), or at the parents specific request.

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Screening dyspepsia by serology to *Helicobacter pylori* in children

SIR,—Dr Sobala and colleagues' message (July 13, p 94) is very important in clinical practice. Young adults with dyspepsia who are seronegative for *Helicobacter pylori* and who are not NSAID users do not have peptic ulcer disease. We are evaluating a screening policy in children, and have shown that abnormal concentrations of IgG and/or IgA against *H pylori* identified infected children with 95% sensitivity and 84% specificity.[1] In addition, increased concentrations of specific antibodies were found in 6% of Italian children, irrespective of the presence of chronic dyspepsia. The infection seems to be unusual before 10 years of age, although seropositivity has been detected in children younger than this,[1] and some infected infants have been described.

We obtained serum samples prospectively from 46 children (age range 8–18 years) evaluated for recurrent epigastric pain. *H pylori*-specific IgG and IgA were measured by ELISA. 22 children were seropositive (8 were seropositive for IgG, 6 for IgA, and 8 for both IgG and IgA). 26 children (4 IgG seropositive, 6 IgA seropositive, 7 IgG and IgA seropositive, and 9 seronegative) were evaluated by upper gastrointestinal endoscopy with biopsy; 5 (4 IgG and 1 IgG and IgA seropositive) of the 22 seropositive children had refused endoscopy.

The table shows diagnoses and *H pylori* status. All 11 children with *H pylori*-associated chronic gastritis had raised concentrations