true in his national epidemiological study. The reasons for dysrhapic developmental accidents may lie in the genotype, in the pre- or postconceptional environment, or in both. If so, is there a case for studying preventive vitamin supplementation in mothers after the occurrence of a minor developmental mishap like the one reported?

J-U WALTHER AND J A RAEBURN
*Pettenkoferstrasse 8a,
 D 8000 München 2,
 West Germany; and
 Western General Hospital,
 Edinburgh EH4 2XU.*

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Translocations, social class, and Adam and Eve

SIR,

Two points in the paper of Tierney *et al* (Balanced rearrangements of the autosomes: results of a longitudinal study of a newborn survey population. *J Med Genet* 1984;21:45-51) require comment.

Firstly, the authors speak of a lower mean social class accentuating an apparent association of a higher IQ in children with familial reciprocal translocations (their table 5). Their mean social class differed 'significantly' from that of the Robertsonian group: 3.43 vs 2.43. But this is not a valid approach. Social class grouping is not a continuous metrical variable; it is a five-point categorisation. The difference between social classes I and II is not, say, twice as much as between II and IV. Perhaps it would be useful for editors to insist upon the convention of using Roman numerals: 3.43 may have passed muster, but I doubt that III.XLIII would.

Secondly, Tierney *et al* propose that the somewhat higher IQ in just seven children with familial balanced rearrangements might reflect evolutionary advance. Now, I am quite prepared to believe that Adam and Eve arose (as brother and sister) following a chromosomal change in an ancestral primate.

Vogel and Motulsky refer to this explicitly on page 449 of their text *Human genetics* (1st ed), and implicitly in putting Dürer's woodcut of Adam and Eve on the front cover. But that 'giant saltation' was, surely, a one-off event. It is difficult to conceive of mini-saltations (hops and skips?) occurring at a rate of several per generation, and which so reorder the genome that the cerebral phenotype is improved. Speculation is fine (and fun), but one must resist the temptation to go overboard.

Otherwise, I found this an interesting and useful paper, particularly in respect of the data concerning the phenotypes of children with de novo apparently balanced rearrangements.

R J M GARDNER

*Department of Paediatrics and
 Child Health,
 University of Otago,
 Dunedin,
 New Zealand.*

Tracheo-oesophageal dysraphism

SIR,

I am alarmed by Dr David's wish (*J Med Genet* 1984;21:74) to get his hands on "whoever first used the awful term tracheo-oesophageal dysraphism and wish to disclaim responsibility, though I cannot remember where I came across the phrase when I first used it (*Lancet* 1980;ii:80). I thought 'dysraphism' (defective fusion) was an acceptable way of getting around the clumsiness of 'tracheo-oesophageal fistula and/or oesophageal atresia'. What would Dr David suggest?

As for the 'probably spurious' increase in NTD in sibs of children with TED, both negative studies cited by Dr David came from low frequency areas, and Baird's study did not measure the number of sibs but only failed to find a sib with NTD reported to the BC registry. Drs Ilyina and Lurie (*J Med Genet* 1984;21:73-4) place our study in the 'low incidence' group, but in fact Montreal had a frequency of over three per 1000 during the time of the study. The figure for 'Canada' given is for British Columbia, a low incidence area. Before giving up the idea of an increase in NTD in sibs of children with TED, I would like to see negative data from a high incidence area.

F C FRASER

*Division of Community Medicine,
 Memorial University,
 St John's,
 Newfoundland,
 Canada A1B 3V6.*