

included in the series. We need this information, because without it the assertion that "no amputation should be undertaken without preoperative vascular assessment" cannot be sustained.

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*This letter has been shown to Mr Cumming, whose reply follows.—ED. L.

SIR,—We agree that calling for "an aggressive approach to amputation" might be open to misinterpretation. However, we advise "an aggressive approach to knee joint preservation", and this statement is unambiguous. Some patients were treated by above-knee amputation on the basis of thermographic and pressure studies, in conjunction with clinical judgment. The complete record of all major lower-limb amputations done in Tayside in 1981-85 will be published elsewhere. It would have been inappropriate to include details of patients undergoing above-knee amputation in a paper concerned with the fate of the below-knee amputee.

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LACK OF SERUM IMMUNE RESPONSE TO BLASTOCYSTIS HOMINIS

SIR,—The protozoa *Blastocystis hominis* is a frequent inhabitant of the intestinal tract¹ but its role as an agent of diarrhoeal disease is controversial. Some reports indict the parasite when present in large numbers in diarrhoea^{2,3} but other investigators see its presence in diarrhoeal syndromes as fortuitous,⁴ the symptoms being due to undetected pathogens or to bowel dysfunction of non-infectious origin.

Four patients aged 12, 37, 40, and 82 years presented with diarrhoeal syndromes associated with *B. hominis* that had been present for 2-18 months. The patients presented with intermittent diarrhoea and abdominal pain, associated with bloating in two cases. All four had taken a short course of metronidazole. Two recovered promptly; one improved then relapsed and was treated with a second course of metronidazole before he recovered completely; and one patient was lost to follow-up. Large numbers of *B. hominis* (more than 5 per high-power field) were observed in stool specimens from all four patients. Stools were negative on culture for *Salmonella*, *Shigella*, *Campylobacter*, *Yersinia*, and *Aeromonas* spp. No parasites other than *Blastocystis* sp were detected on direct stool examination or on iron haematoxylin stained smears from concentrated samples.

We have examined the immune responses of these patients' sera to specific *B. hominis* polypeptides by immunoblot analysis. Antigens were derived from *B. hominis* recovered from a patient excreting large numbers of organisms. The protozoan was axenised under anaerobic conditions using a biphasic medium consisting of inspissated whole egg slant overlaid with Locke's solution containing 11% human serum.⁵ The medium was pre-reduced for 7 days before culture and media incorporated antibiotics. Axenised organisms were washed, sonicated, and boiled in reducing buffer containing β -mercaptoethanol and sodium dodecylsulphate. The samples were resolved on 12% sodium dodecylsulphate-polyacrylamide gels and transferred to nitrocellulose paper.⁶ Nitrocellulose blots were stained with amido-Schwartz and blocked with 5% ovalbumin. A 1/100 or 1/25 serum dilution was then reacted with the blots for 16 h, after which they were washed and probed with ¹²⁵I-labelled protein A.

No specific serum antibody response directed against any *B. hominis* protein component was detected on immunoblots, even after exposure of autoradiographs for 6 days. Adequate protein was used, as visualised by amido-Schwartz staining of blots, and the pattern of *B. hominis* polypeptides was distinct from the protein pattern of axenisation medium when it was subjected to electrophoresis.

Other non-invasive gastrointestinal pathogens, such as *Giardia lamblia*⁷ and rotavirus,⁸ elicit a specific serum immune response which can be monitored by immunoblot, as do pathogens localised to the genital tract.⁹ The immunoblot assay we used detected IgG;

perhaps *B. hominis* provokes a secretory IgA antibody response or maybe the humoral immune system is not activated. However, our findings do not support a role for *B. hominis* in diarrhoeal disease.

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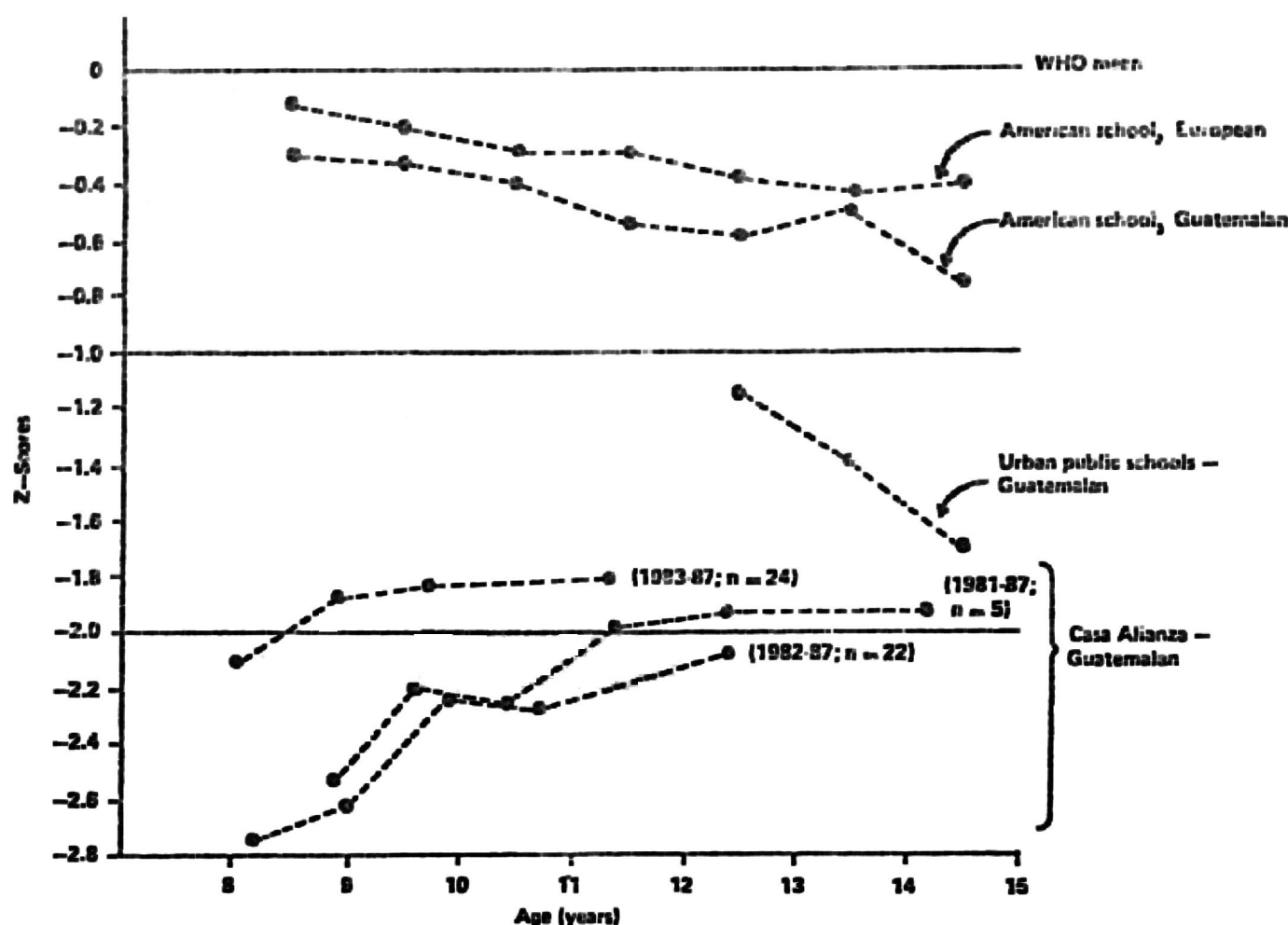
EVIDENCE FOR CATCH-UP GROWTH IN ADOLESCENCE

SIR,—Catch-up growth is defined as the process of recovery from an acute episode of infection or malnutrition and the longer-term process during which a poor previous history of growth is compensated for, resulting ultimately in a normal or near-normal body size.¹ The Institute of Nutrition of Central America and Panama (INCAP) longitudinal study on growth and development in eastern Guatemala¹ showed that for pre-school children food supplementation had its greatest impact on the growth of those with low weight and length for age as well as on the growth of those who had diarrhoea. Information on compensatory growth during adolescence, a period of accelerated growth, is lacking, however.

At INCAP data on growth during adolescence, collected in several studies in Guatemala, have been examined. Among the aspects of research interest is catch-up growth in chronically growth-retarded adolescents. One of these studies was of school-age boys with acute and chronic protein-calorie malnutrition from poor families, who had been residing for a long time in Casa Alianza, a foster home in Antigua Guatemala. At this home the children have enjoyed proper food, they benefit from both preventive and treatment-oriented health services, and there is a comprehensive educational programme. The children are measured periodically and data on 51 children admitted in 1981-83 have been analysed. Standardised measurements were done by INCAP personnel six, five, and four times, respectively, during the period 1981-87. Only children on whom consecutive measurements were done every year since admission to Casa Alianza were included in these analyses.

Mean Z-scores of height-for-age are compared with the WHO reference growth curve in the figure. The Casa Alianza boys have remained below the WHO growth curve and below curves for children attending the American School^{2,3} or state (public) schools⁴ in Guatemala City. The American School pupils, representing the richer families in Guatemala, were categorised as "Guatemalan" (home language Spanish and all four grandparents born in Guatemala) or "Europeans" (home language not Spanish, parents born in Western Europe [excluding Spain] or the United States). The public schools' pupils constitute a representative sample of Guatemalan adolescents from poor families attending secondary schools in Guatemala City.

The mean Z-scores of height-for-age of children from both the American School and from the public schools are negative and tend to fall during adolescence. The mean Z-score for the Casa Alianza children are also negative but they tend to improve during adolescence, suggesting catch-up growth.



Mean Z-scores of height-for-age in young adolescents.

The relation between initial Z-scores of height-for-age and weight-for-height, age, and time in the programme and changes in Z-scores of height-for-age since admission in the Casa Alianza children was studied by multiple regression analyses. The dependent variable was change in Z-score of height-for-age (ΔZ_h) and the independent variables were age (a), Z-scores of height-for-age (ΔZ_h) and weight-for-height (ΔZ_w) at admission, and time in programme (t). All four independent variables were significant predictors of the magnitude of ΔZ_h , and the multiple regression equation, for 129 children, was:

$$\Delta Z_h = 0.217 + 0.020a - 0.547Z_h - 0.435Z_w + 0.003t^2$$

Our findings suggest that catch-up in height is greater for children who were older and more stunted and wasted on admission to the Casa Alianza programme and that it is related to the square of the time in the programme.

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REPETITION STRAIN INJURY

SIR,—I read with interest your Aug 8 editorial and subsequent correspondence (Sept 26). With a bound the great Australian disease of "kangaroo paw" has arrived in Britain. Those of us who have followed the forthright and often vituperative letters in the *Medical Journal of Australia* since 1984 will not be surprised to note the differences of opinion between your correspondents such as Dr Awerbach, who doubts that the disease exists, and Dr Williams, who says that the condition is common in athletes. Moreover, claims for tenosynovitis and allied conditions are increasing in Britain, possibly driven by trades unions. Others report epidemics

among professional musicians.¹ Professor Brooks invokes semantics and suggests that "regional pain syndrome" might be a better term, though this does not seem to get us much further forward with either diagnosis or treatment.

I hope that the polarisation of view seen in Australia will not be repeated elsewhere and that a reasonable view will be taken. Like many "diseases" RSI is multifactorial and embraces the pathological, the physiological, and the psychosocial. There is ample evidence that repetitive activities can be combined with abnormal strain to produce physical abnormalities,^{2,3} and this is a fact of life to orthopaedic surgeons who treat athletes. To deny that such injuries can occur in the course of work is nonsensical, though the incidence of repetition strain injury in certain groups such as visual display unit operators in Australia suggests that psychological and social factors may be lowering the threshold for complaints of aching due to simple physiological strain.

Let us recognise that strenuous repetitive work can cause physical damage with conditions ranging from tenosynovitis to simple muscular aching, but let us also take into account the high incidence of natural discomfort⁴ and the fact that symptoms arising in the course of work are perhaps less well tolerated than those arising at play or in sport.

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SIR,—Professor Brooks and Dr Awerbach comment on your editorial on repetition strain injury (RSI). There are further reasons to be cautious before generalising from the Australian epidemic. I have reviewed 3976 cases of RSI in one workforce, and, in contrast to European studies of tenosynovitis, could not demonstrate a linear dose-response relation between RSI and key-stroke rate or duration of work with a keyboard. The morbidity was severe, 16% of cases being absent from work for 26 weeks or more.¹ This epidemic has been a watershed in Australian occupational health because it has highlighted the emergence of a new class of occupational health problems. The traditional concept of occupational disease being