

that time. Because of this reflex we doubt the efficacy of screening boys for undescended testes at 1 year and at school entry. The figures from Tamhne *et al* confirm our opinion that too many boys will be operated on unnecessarily because of retractility of the testes.

In 1989 a retrospective cohort study concerning the localisation of the testes from birth until puberty of 853 boys born in 1973 and living in West Friesland (The Netherlands) was done. In this study, which has been submitted for publication, we found a considerable number of boys with one or two undescended testes, that when previously measured, had been registered as scrotal. In all these boys the testes had assumed a normal scrotal position at puberty. This supports our advice that if the testes were descended no further screenings are necessary.

We agree with Tamhne *et al* that there is a need to set clear guidelines for the diagnosis of undescended testes and for referral pathways. This is important especially to prevent unnecessary operations.

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Revised criteria for diagnosis of coeliac disease and medical audit

SIR,—In the recently published revised criteria from the European Society for Paediatric Gastroenterology and Nutrition (ESPGAN) for the diagnosis of coeliac disease,¹ emphasis has shifted from the need for repeated biopsies to the response to dietary gluten withdrawal, combined with initial histological appearance of the jejunal biopsy.

It might be thought that this could decrease the use of this unpleasant and time consuming diagnostic procedure. However this may not be the case.

As part of a medical audit programme the clinical and histological details of jejunal biopsies performed in the Royal Liverpool Children's Hospital between January and May 1990 were reviewed.

Analysis of the data showed: (1) Out of 46 attempted biopsies, 10 were initially unsuccessful. (2) Of the 36 children biopsied, only four biopsies were performed after a gluten challenge, the rest were for initial diagnosis. (3) The number of jejunal biopsies for the first five months of 1990 was similar to the whole of 1989 (36 *v* 39) yet proportionately more children started a gluten free diet (11/36 (30%) *v* 10/39 (25%)). (4) Children referred from an outlying hospital were more likely to have a biopsy specimen compatible with coeliac disease (50% *v* 25%). (5) Eleven children commenced a gluten free diet, but only six had the characteristic 'flat' small intestinal mucosa of coeliac disease.

It was concluded that: (1) There was a significant chance of procedural failure with jejunal biopsy in children. (2) Few biopsies were performed after gluten challenge indicating that the practice of diagnosing coeliac disease by repeated biopsies had already lapsed.² (3) The increased number of biopsies in 1990 reflected a more aggressive diagnostic approach. (4) Gluten free diet was often commenced in the absence of characteristic histological criteria. With the new criteria these children would require gluten challenge.

It is therefore to be hoped that the use of the revised ESPGAN criteria will reduce dependency upon multiple jejunal biopsies and the inappropriate use of gluten free diet as highlighted by our medical audit.

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Crohn's disease

SIR,—Dr Puntis and colleagues have reported in this journal the first case of granulomatous lung involvement in a child with Crohn's disease.¹ It is clear from many previous reports that pulmonary involvement is actually common in chronic inflammatory bowel disease, although only a minority of patients have symptoms or overt signs. Bonniere and colleagues have demonstrated, in 22 adults with Crohn's disease who had no pulmonary symptoms, reduced serum activity of angiotensin converting enzyme, lymphocytosis on bronchoalveolar lavage, increased superoxide anion production from activated alveolar macrophages, and abnormal pulmonary function tests.² They concluded that most patients with Crohn's disease have latent pulmonary involvement. After recent findings in this unit, we are now able to suggest a mechanism for this previously unexplained phenomenon.

We have demonstrated production of the cytokine tumour necrosis factor- α (TNF- α) by single macrophages in colonic biopsies from patients with both Crohn's disease and ulcerative colitis,³ and have found raised serum concentrations of TNF- α in patients with relapsed colonic disease⁴; we consider that chronic TNF- α elevation may contribute to anorexia and growth failure. TNF- α has also been implicated in granuloma formation⁵; granuloma epithelioid cells are in fact activated and transformed macrophages. TNF- α mRNA was found in large quantities within these experimentally induced hepatic granulomas and anti-TNF- α monoclonal antibodies caused both rapid granuloma regression and reduction of TNF- α mRNA content.

TNF- α production is actually increased by exposure of macrophages to TNF- α itself,^{5,6} so that chronic elevation of serum TNF- α might lead to TNF- α production by tissue based macrophages. Pulmonary alveolar macrophages produce several times more TNF- α on stimulation than do circulating monocytes,⁷ and we suggest that raised serum TNF- α concentrations in chronic inflammatory bowel disease could stimulate alveolar macrophages to produce more TNF- α , which

would then act within the pulmonary micro-environment.

It is now clear that TNF- α can cause lung damage, and it is viewed as an important mediator in the adult respiratory distress syndrome (ARDS), which frequently supervenes in conditions such as septicaemia where serum TNF- α concentrations are grossly raised. Alveolar lavage fluid contains measurable TNF- α of pulmonary origin in ARDS.⁸ Lesser elevation of serum TNF- α produces a more subtle derangement of pulmonary function, demonstrated well by the abnormal lung function tests found in patients treated for malignancy with recombinant TNF- α .⁹

We think that this mechanism would account for the activated alveolar macrophages found by Bonniere *et al*,² for the subtle lung function abnormalities found in many patients with active disease, for pulmonary granuloma formation, and for the more florid ARDS like picture found in many patients with life threatening toxic dilatation, where serum TNF- α may be much higher.⁴

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VIDEO REVIEWS

The Six Week Check of Infants. A video training package. Commissioned by: Nottingham Health Authority. Production Company: Chrysalis Television Midlands. Cost of training package (video and manual): £195; additional trainer's manuals £6 each. For details contact: Mrs B Whitchurch, Training Office, Notting-