come to reconsider the 1970 European Society of Paediatric Gastroenterology and Nutrition (ESPGAN) diagnostic criteria for gluten sensitive enteropathy, as we do not entirely agree with the timing of the diagnostic intestinal biopsy proposed by our Italian Group of Paediatric Gastroenterology at the meeting in Trieste, May 1987, and recently published. A multicentre study indicates that at least in the vast majority of diagnoses of gluten sensitive enteropathy (more than 90%) are made in young children who are referred with signs of malabsorption, who are then found positive for the antigliadin antibody test. At the Trieste conference it was stated that these so called typical cases do not need a gluten challenge and can be readily and definitely diagnosed by a single biopsy specimen showing a flat mucosa at the first work up.

Looking back on our hundreds of biopsies we have noticed that whenever the clinical and laboratory data strongly suggested gluten sensitive enteropathy, we nearly always found a flat mucosa at the first intestinal biopsy. It appears then that the initial biopsy recommended by ESPGAN and retained by the Trieste recommendation seems seldom informative as its result is highly predictable in typical cases. In these cases (supported by a positive antigliadin antibody test and abnormal intestinal permeability test) we suggest that the initial biopsy is avoided and a six to 12 month period of gluten free diet is commenced (figure).

As the first years of a gluten free diet seem to have an imprinting effect on the long term compliance, we believe that the gluten challenge is, at present, unavoidable in the youngest patients because it demonstrates the persistence of, or the disease to the patient’s family. At the end of a three to six month period of gluten challenge (or less if symptoms develop) a biopsy is highly recommended as the relapse is sometimes evident only at the histological level. This single biopsy will confirm the diagnosis of gluten sensitive enteropathy in most cases. If the mucosa looks normal the patient can be left on a free diet and periodically checked to exclude the possibility of a late response to gluten. In our opinion a short period of gluten ingestion, as we propose, should not expose the patient to significant risks.

The above approach is less invasive than the ESPGAN scheme and fully exploits the diagnostic role of the antigliadin antibody test at the first assessment of the patient. We also suggest adding the sugar intestinal permeability test, which has proved to be a reliable screening procedure for gluten sensitive enteropathy. For atypical or late onset cases we agree that an intestinal biopsy is always mandatory during the first diagnostic phase. A conclusive note of caution is needed. It is well known that long term treatment of gluten sensitive enteropathy is often unsatisfactory. This is especially true for patients who have found ‘sensitive’ doctors who are overzealous in their wish to avoid unpleasant biopsies. So far we have followed the ESPGAN diagnostic criteria for coeliac disease with satisfactory results both for patients and for us. Nevertheless, in the light of the new, non-invasive diagnostic tools, we also agree on the need of revising the ESPGAN recommendations. This attempt should be made with the contribution of experts from several European centres.

The above conclusions are based on a review of the literature and on personal data from several centres.

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Urinary growth hormone excretion

Sir,—The paper by Walker and colleagues on the use of urinary growth hormone excretion as a screening test for growth hormone deficiency is of great interest. Words of caution are needed, however, before universal adoption of this test by paediatricians and general practitioners as already proposed.

Of the two clinical groups studied, one (group 2) consisted of patients with previously diagnosed growth hormone deficiency who had been treated for variable periods with growth hormone. It is therefore highly probable, though not stated in the paper, that the timing of the urinary and serum investigations in this group for growth hormone deficiency was by a considerable period, with the serum investigations at the time of diagnosis preceding the urinary one by months or years. In effect the urinary growth hormone estimation has been estimated retrospectively, which makes it difficult to evaluate its usefulness as a prospective test. Little if anything is known about deterioration of growth hormone secretion with time in patients with growth hormone deficiency, especially if they are on replacement treatment. Such data should not be used in the evaluation of the prospective role of urinary growth hormone excretion as a screening test for growth hormone deficiency.

The two different methods of deriving comparisons between overnight urinary growth hormone excretion and peak serum growth hormone concentration after stimulation makes the value of combining the data to give a coefficient of correlation suspect. In addition, as the authors themselves state, urinary growth hormone excretion is log distributed which invalidates regression analysis. Examination of the figure would suggest that at least for the subjects who were growth hormone deficient, if any correlation exists at all it could possibly be negative. There are clearly several factors that may influence urinary growth hormone excretion, and more detailed statistical analysis of the separate groups might have been more informative. The correlation coefficients would of course have been less impressive. The value of a non-invasive test of growth hormone secretion in children would be immense. Although of considerable promise, the study of Walker and colleagues does not yet justify the general introduction of urinary growth hormone estimations and more validation is necessary to establish the full characteristics of this potentially valuable addition to our range of investigations.

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Dr Walker comments:

The value of a non-invasive test of growth hormone secretion in children would be immense. Although of considerable promise, the study of Walker and colleagues does not yet justify the general introduction of urinary growth hormone estimations and more validation is necessary to establish the full characteristics of this potentially valuable addition to our range of investigations.

The purpose of presenting the relationship between overnight urinary growth hormone and peak serum response to conventional provocation tests (fig 4) was purely to demonstrate that this simple urine test is as good as the currently available blood tests in identifying those children whose growth problems are due to a relative deficiency of growth hormone. By using all those studied prospectively the relationship between the log transformed urinary growth hormone concentrations and peak serum response to provocation holds (r=0.65, p<0.001) and according to advice from our statisticians is quite valid. To undertake ‘a more detailed statistical analysis’ would attach far too great a significance to this relationship especially with the controversy surrounding the interpretation of provocation tests.

The data that best evaluates the role of urinary growth hormone excretion as a screening test for growth hormone deficiency is from the children with short stature in group 3. These were all studied prospectively with concurrent urine and blood tests and the urinary growth hormone results compared with the reference range from a normal population (fig 3). In fact we have since studied at least another 100 similar children and have not yet missed a child with growth hormone deficiency at the back of low serum growth hormone concentrations in response to provocation but a normal urinary growth hormone. Such is our experience and confidence in the method we suggest as it is the first line screen for growth hormone deficiency and offer it as part of our regional endocrine service to Wessex paediatricians.
Urinary growth hormone excretion.

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