systemic lupus erythematosus. *Am J Med* 1981; **70**: 875–81.

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Reply

SIR—We would like to thank Castañeda *et al* for pointing out our apparent omission of five other cases of protein losing enteropathy (PLE) with systemic lupus erythematosus (SLE) previously recorded in the literature. We felt, however, that certain other points which they raised were not entirely valid.

Firstly, although it is true that our patient does not quite fulfil the modified ARA criteria for SLE, clearly she does not have clinical features of any other connective tissue disease and cannot therefore be categorised as mixed connective tissue disease. This is a situation which frequently arises in practice and such patients are therefore best classified within the SLE syndrome. The presence of RNP antibodies and absence of DNA antibodies is quite compatible with a diagnosis of SLE. Secondly, we feel that it is fairly obvious from our list of negative investigations that reasonable steps were taken to exclude the other possible causes of PLE. Thirdly, it is also clearly stated that our comments about the pathogenesis of the odema are purely speculative and we do indeed refer to the possibility of an immune complex-mediated vasculitis as a possible mechanism. There was, however, no clinical evidence of vasculitis elsewhere.

Finally, our patient is of Anglo-Saxon origin.

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Reference

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Coeliac disease presenting with intestinal pseudo-obstruction

SIR,—Thank you for the opportunity to comment upon Dr Cluysenaer and Dr van Tongeren's letter (*Gut* 1985; **26**: 538). Our findings do not support their hypothesis because although we did not measure vitamin E levels, there was no evidence of ceroid deposition in full thickness biopsies of the jejunum, ileum and colon of our patient (*Gut* 1984; **25**: 1003–8).

Ceroid accumulation has long been recognised in

association with proven or suspected coeliac disease^{1–4} but its presence is generally considered to give rise to no symptoms.⁵ In only one previous case was pseudo-obstruction a reported feature.¹ Ceroid deposition has also been recorded in a case of pseudo-obstruction associated with scleroderma.⁶ As focal muscle atrophy with fibrous replacement can account for the motility disturbance of this condition, however, there seems little need to invoke a role for the ceroid pigment, especially as its presence has not been documented in other reports.^{7,8} Furthermore, ceroid has also been shown in cystic fibrosis, biliary atresia, and cirrhosis in childhood⁴ and in chronic pancreatitis in adults,⁹ all conditions which could result in malabsorption of vitamin E, but which are not associated with pseudo-obstruction. Thus, the presence of ceroid pigment may merely be a reflection of the vitamin E status of a patient rather than be causally related to intestinal motor dysfunction.

A role for vitamin E deficiency *per se* in pseudo-obstruction still remains a possibility, especially as such deficiency in animals may produce central nervous system effects and nutritional muscular dystrophy.¹⁰ The mechanism of vitamin E deficiency is ill-understood, some effects being reversible by antioxidants, others by selenium and yet others responding only to vitamin E replacement.¹⁰ Any possible effect of vitamin E on neuromuscular dysfunction in coeliac disease might thus be independent of the finding of ceroid deposition which is thought to accumulate because of the antioxidant deficiency.

The association rediscovered by Cluysenaer and van Tongeren is potentially important, but further studies are required to clarify any relationship between vitamin E deficiency, ceroid deposition, coeliac disease, and pseudo-obstruction. It would thus be important to know, for example, how many of their patients without ileus had ceroid deposition, and whether there was any correlation between this, vitamin E or selenium concentrations, and intestinal transit time.

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