It is apparent from the results (Table) that there is a close relationship between the urinary F.D.P. concentration and the disease activity. Even in the apparently "static" group three patients were excreting increased concentrations of urinary F.D.P. Noteworthy is the observation that 10 individuals judged to have progressive renal impairment only two had elevated serum F.D.P. while all had increased urinary excretion of these products. Highest urinary F.D.P. excretion concentrations were observed in polycystic disease with renal failure, hydronephrosis, lupus nephritis, post-sclerotic nephritis, and renal failure due to progressive proliferative glomerulonephritis. Serial urine studies in the patient with lupus nephritis, who was pregnant, showed close correlation with both her biochemical and clinical condition. Serum F.D.P. in this patient were less helpful and did not accurately reflect the marked fluctuations which occurred in the urinary F.D.P.

We agree with Dr. Clarkson and his colleagues that in certain groups of patients the urinary F.D.P. are derived from the renal glomerulus and do not represent filtration from the systemic circulation. In this context it is of interest that we have observed two patients with disseminated intravascular coagulation but with normal renal function. Both patients excreted greater than 20 mg/ml, but in neither patient were we able to detect F.D.P. in the urine. We note that the Edinburgh workers were unable to detect urinary F.D.P. in patients with uncomplicated polycystic disease. We also were unable to detect breakdown products in this type of patient, but in one patient with polycystic disease and progressive impairment of renal function the urinary F.D.P. were greater than 20 mg/ml, and it may be that in this group also urinary fibrin/fibrinogen breakdown products may be of value in assessing the progression of the renal lesion.

Though an estimation of the 24-hour excretion of F.D.P. provides a more accurate assessment of renal involvement such collections are difficult to carry out on an outpatient basis. Excretion of some value in phase the results of our obtained on random urine samples collected during an afternoon outpatient clinic.

We would like, therefore, to reiterate the view expressed by Dr. Clarkson and his colleagues that estimation of urinary F.D.P. may afford a simple but sensitive means of assessing the activity of the disease process in patients with certain types of renal disorder.—We are, etc.,

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Treatment of Myocardial Infarction

Sir,—Dr. G. R. Fearnley (21 August, p. 473) in commenting upon the findings of the European working party's statistically-controlled trial of streptokinase therapy in acute myocardial infarction (7 August, p. 325) expresses a personal view that thrombolytic therapy is unlikely to be useful in the treatment of this disease. Nevertheless, as with other such therapeutic problems, the role of thrombolytic therapy in this disease will be defined on the basis of scientific evidence rather than by disputation.

While the evidence is still incomplete, it should be noted, particularly since this evidence is not fully cited by the European working party, that this study is one of a number of trials in which apparently similar streptokinase dosage schedules have been employed. Of these studies, three others show statistically-significant benefit in favour of streptokinase, although in study randomisation was less than perfect. Two studies show a trend in favour of streptokinase treatment, and in the last study, with very low total mortality, there was no treatment preference. Clearly, these clinical-trial data offer substantial encouragement to investigators developing such new approaches to the treatment of acute myocardial infarction. I am, etc.,

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The Gut and Dermatitis Herpetiformis

Sir,—I consider your leading article on the gut and dermatitis herpetiformis (2 October, p. 5) exceptionally ill-informed and misleading. The article itself is contradictory; it starts with the sentence "enteropathy may be associated with dermatitis herpetiformis" (no references), but in the second paragraph reports a study which shows that enteropathy is almost invariably present (21 out of 22 patients studied). However, your article should really be criticized for failing to refer to the many studies and ready access to considerable literature on the subject, which has answered many of the questions raised by you and is also at variance with a number of the statements in your article.

Your article looks for evidence as to whether the enteropathy in dermatitis herpetiformis is the same as that in coeliac disease. You rightly state the histological appearances of the small intestinal lesion are similar (but reported 1966); that there is evidence of diminishing severity of the lesions in the distal small intestine (first reported 1967 and 1968) and there is evidence of gluten sensitivity in patients with dermatitis herpetiformis. However, you then find it difficult to equate the syndrome (dermatitis herpetiformis enteropathy) with coeliac disease. As long ago as 1967 when it was first suggested the enteropathy in dermatitis herpetiformis was due to gluten sensitivity further evidence was presented to show that the enteropathy in the two diseases had a similar aetiopathology. A high incidence of increased faecal excretion of folate, iron, and M globulin deficiency, and of splenic atrophy was found in patients with dermatitis herpetiformis and is known to occur in coeliac disease. Further evidence that both diseases are similar has since been forthcoming in that anticoagulant tissue antibodies have been reported in both disorders.

The next point where your article is incorrect is that though you state categorically that treatment of dermatitis herpetiformis with a gluten-free diet does not benefit the cutaneous lesions, you fail to mention the reports where a gluten-free diet has been shown to influence and in some instances control the eruption. This naturally leads to the following question (which you raised but could not answer) as to whether patients with dermatitis herpetiformis should be treated with a gluten-free diet.

In favour of treatment with a gluten-free diet it can be stated that in some patients the rash can be controlled by diet alone. Secondly, definite clinical (subjective and objective) improvement does occur in a number of patients with dermatitis herpetiformis. This is similar to patients with coeliac disease starting a gluten-free diet. Thirdly, as mentioned above, a large proportion of patients with dermatitis herpetiformis are on diet and further, deficiency of folate has been shown to improve with a gluten-free diet. Finally, the question of reticulosis of the small bowel has to be considered. You say that dermatologists are not aware that these patients with dermatitis herpetiformis are specially liable to develop such neoplasms. However, you fail to mention that treatment with a gluten-free diet has already been suggested for the very reason by a dermatologist (and a patient under my care with dermatitis herpetiformis has developed a lymphoma of the small bowel—before starting a gluten-free diet).

The above are definite points (all but the last you fail to mention) in favour of treating patients with dermatitis herpetiformis with a gluten-free diet. However, in practice, because of the social and practical problems of adhering to a gluten-free diet, I find it is not possible to treat all patients with this diet, and it has been my policy to consider each patient individually, rather than lay down any definite rule.—I am, etc.,

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Congenital Leukaemia and Mongolism

Sir,—Having failed to find a clinical report of congenital leukaemia in association with mongolism in the medical literature of this country, I should like to record the following case history.

A female mongol infant was born at Edgware General Hospital on 29 August 1970.
Physical signs were observed at once which suggested a blood disorder. Respiration was established without difficulty and the lungs appeared to expand normally but mild cyanosis persisted. Clinically and radiologically there was no evidence of congenital heart disease, but the liver was considerably enlarged. No glands were felt and the spleen was impalpable at the initial examination, but in view of the known association of Down's syndrome with congenital leukaemia a blood count was obtained about eight hours after birth. 

Arrangements were also made for chromosome studies to be undertaken, but as the infant was born during the August Bank Holiday investigations were necessarily limited. The blood count showed Hb 18 g/100 ml, haemocrit 54, M.C.H.C. 33, W.B.C.s 320,000/mm³ (neutrophils 10%, lymphocytes 8%, monocytes 4%, metamyelocytes 10%, myelocytes 18%, promyelocytes 10%, myeloblasts 40%). No platelet count was done. There was no evidence of sepsis, the mother's W.R. was negative, blood group of mother and infant was O Rh+ and the indirect Coombs test was negative, so leukemic reaction to sepsis, congenital syphilis, or erythroblastosis appeared to be excluded.

The baby became dyspnoic and more cyanosed, the liver increased in size, and the spleen became palpable before she died 48 hours after birth. The mode of death was very similar to that described by Pierce who found that the majority of infants exhibiting signs of leukaemia at birth died within a few days, often from respiratory insufficiency caused by atelectasis or pulmonary infiltration. The transient leukaemia-like blood picture sometimes seen in mongols after birth need not be considered in this case since the condition proved fatal and was clearly of intra-uterine origin and long standing. Postmortem examination showed the lungs to be expanded, with congestive changes and patchy atelectasis. There was no pneumonia. The liver was grossly enlarged, parenchyma being obscured by extensive extramedullary haemopoiesis, predominantly leukoblastic. Kidneys showed foci of haemopoietic tissue adventitia but no lesions similar to those of myeloblastic leukaemia. —I am, etc.,

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Wegener's Granulomatosis

Sir,—It is surprising that your leading article on 'Wegener’s Granulomatosis' (21 August, p. 446) fails to mention the very striking results obtained in this condition with the use of large doses of cyclophosphamide. Each of the patients treated had extensive pulmonary and central nervous system involvement and lesser degrees of renal involvement and sinusitis. Corticos- teroid therapy had proved without effect in all cases.

There was rapid improvement in signs and symptoms in four patients after cyclophosphamide had been started. Almost all evidence of disease disappeared. In one patient it was possible to discontinue the drug for 20 months and in another for 12 months. The other two patients were asymptomatic for three and a half years on continued low doses of cyclophosphamide.—I am, etc.,

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Halothane Hepatitis

Sir,—Mr. J. D. Langdon quotes Professor W. W. Mushin and colleagues (3 July, p. 18) as an authority for his statement that "there is no longer any doubt that in a small number of cases halothane administration does precipitate hepatitis." It is unfortunate that the summary which appears in bold print at the beginning of the paper by Professor Mushin and his colleagues is so misleading and open to misinterpretation, because in a subsequent letter Professor Mushin and Dr. M. Rosen state quite clearly that they did not intend to imply that halothane is the causative factor in "post-halothane jaundice." (14 August, p. 431).

The study by Professor Mushin and his colleagues established the high incidence of postoperative jaundice following two operations in less than one month. Since halothane is now used in 90% of anaesthetic procedures in this country it is therefore important that efforts be made to establish whether or not halothane is a co-incidental factor in the great majority of cases when postoperative jaundice occurs. As yet, no data have been provided which implicate halothane further than that (24 July, p. 245).—We are, etc.,

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1 Langdon, J. D., British Dental Journal, 1971, 131, 94.

Non-specific Backache

Sir,—The formation of a Back Pain Club such as your leading article describes (2 October, p. 4) is a welcome event. Backache spoils so many people’s lives, and provides one of the major causes of industrial invalidism today. Its importance was emphasized by a survey ("The Sore Back") in Glasgow of over six million episodes off work in 1966. This revealed that middle-aged employees with back trouble stayed away from work for an average of five months. Since there must be many people in that city who spend a week or two in bed and then return to work, this figure implies that a sizeable proportion absented themselves for a year. Here is a challenge indeed, involving millions of public money.

Naturally diagnosis leading to logical treatment and the detection of symptoms devoid of organic basis are both essential. Future progress must rely on research, but surely the first step is to disseminate to every hospital the large body of clinical knowledge that has already been accumulated, and to make available within the N.H.S. for the first time treatments well known to succeed. This plan could be implemented immediately.

Little is to be gained by lumping difficult cases together under the vague term "non-specific backache." There is a simple way out that I have employed daily since 1937: diagnostic epidural local anaesthesia. This injection has proved a reliable criterion for differentiating disc backache from non-disc backache. If a patient with a lesion of a lumbar moving part finds that his movements become painless after the external aspect of the dura mater and of the nerve roots has been rendered insensitive, a disc lesion must be responsible. The fluid injected (procaine 1:200) does not enter a joint, nor cause a detectable nerve palsy, nor reach the anterior aspect of the psoas or the longitudinal ligament against which a protruded disc engages. Where the fluid runs is clearly shown by the contrast radiographs (Plates 44-48) reproduced in succeeding editions of my book.2

I suggest that the Club need not start from scratch, but can base its further investigations on facts alreadyascertained and knowledge already in existence.—I am, etc.,

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1 Symposium on Clinical Problems of Practice, Proceedings of the Royal College of Practitioners, 1967, 13, 60.