LETTERS TO THE EDITOR

Prediction of severity of acute pancreatitis

SIR,—We read with great interest the article by Fan *et al* (*Gut* 1989; **30**: 1591–5) in which they have shown that two factors (serum urea and plasma glucose) were as good as the conventionally used multifactor scoring system of Imrie *et al*[1] and Ranson *et al*[2] using nine and 11 factors respectively for clinical and biochemical assessment of severity of acute pancreatitis. The major drawbacks of multifactor include (a) use of too many factors, (b) need for a longer duration (48 hours) before assessment of severity can be made, and (c) effect of treatment on various assessment parameters during 48 hours of observation.

Although fascinating, it seems unlikely that the authors' new approach – the use of a discriminant value of the two factors (serum urea >7·4 mmol/l and plasma glucose >11·0 mmol/l) in assessment of severity of pancreatitis – will stand the test of time because of the following reasons. Firstly, the raised serum urea has a very non-specific value as it can be altered because of dehydration, repeated vomitings, poor intake, and other non-pancreatic factors like gastrointestinal bleeding and renal dysfunction. Secondly, the occurrence of upper gastrointestinal bleeding (occurring in 10–20% of patients with acute pancreatitis)[3] may significantly affect the serum urea concentration even though it may have no relation to severity of pancreatitis.

Thirdly, the authors' explanation that high serum urea concentration could be a reflection of poor physiologic reserve of major organ system does not seem to have convincing scientific appeal.

Moreover as the plasma glucose intolerance and incidence of diabetes mellitus increase with age and the authors fail to mention whether or not underlying diabetes mellitus was ruled out in their patients with acute pancreatitis, it is possible that a proportion of their patients may have had raised plasma glucose secondary to pre-existing glucose intolerance or diabetes mellitus rather than because of underlying severe pancreatitis.

Finally, we believe that from the standpoint of the clinical management there is no harm in waiting for a day or two to observe the course of acute pancreatitis on conservative treatment even though the course may alter (maybe for the good) the score of the multifactor scoring system, rather than rush to predict the severity of acute pancreatitis at admission.

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1 Imrie CW, Benjamin IS, Ferguson JC, *et al*. A single-centre double-blind trial of trasylol therapy in primary acute pancreatitis. *Br J Surg* 1978; **65**: 337–41.
2 Ranson JHC, Rifkind KM, Turner JW. Prognostic signs and nonoperative peritoneal lavage in acute pancreatitis. *Surg Gynecol Obstet* 1976; **143**: 209–19.
3 Seorgel KH. Acute pancreatitis. In Sleisenger MH, Fordtran JS, eds. *Gastrointestinal disease, pathophysiology, diagnosis and management*. 4th ed. Philadelphia: Saunders, 1989: 1823–4.

Reply

SIR,—We agree that raised serum urea and blood glucose at the time of admission may be influenced by many factors. This inadequacy was reflected by the relatively low predictive value of positive and was fully discussed in the report. However, all the possibilities leading to raised serum urea mentioned by Dr Arora and Acharya were definitely related to a severe attack of acute pancreatitis and I cannot agree that gastrointestinal bleeding is unrelated to severity.[1] Patients with underlying diabetes mellitus were not specifically defined in our report. However, diabetic patients with underlying major organ dysfunction are certainly at high risk of developing systemic complications of acute pancreatitis and deserve to be carefully monitored and aggressively treated at admission.

The policy of waiting for 48 hours to monitor the course of the disease and to collect complete data for grading of severity is not justifiable in modern day medicine. In our previous report,[2] 13·8% of patients deteriorated within 48 hours. With adequate treatment, fewer patients did so.

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1 Frey CF, Stanley CJ, Eckhauser F. Haemorrhage. In: Bradley EL III, ed. *Complications of pancreatitis, medical and surgical management*. Philadelphia: Saunders, 1982: 96–123.
2 Choi TK, Mok F, Zhan WH, Fan ST, Lai ECS, Wong J. Somatostatin in the treatment of acute pancreatitis: a prospective randomised controlled trial. *Gut* 1989; **30**: 223–7.

Why do patients with ulcerative colitis relapse?

SIR,—In reply to my letter (*Gut* 1990; **31**: 959) Riley *et al* state that of the many studies I quoted 'all are uncontrolled.' This is incorrect and if unchallenged may lead further research workers to discount those criticisms regarding inappropriate methodology which remain valid and thus perpetuate the likelihood of further needless waste of research effort. My 1959 article[1] stated that 98 radiotherapy patients were used as control subjects and interviewed according to the same protocol as the 173 ulcerative colitis patients, while McMahon *et al*[2] used healthy siblings as controls in their investigation of 23 patients by means of psychometric tests including the Minnesota Multiphasic Personality Inventory and psychiatric interviews. In another investigation of 35 patients entitled 'Psychopathology of ulcerative colitis' Roubicek and Martonova[3] used 20 healthy subjects as controls and confirmed the limited value of standard psychiatric tests in these emotionally guarded colitis subjects by means of sensitive interviewing and the Thematic Apperception Test designed to penetrate emotional defences.

Riley *et al* are right to emphasise the continuing need for 'controlled clinical trials' but if the questions asked are irrelevant to pathogenesis, or the instruments of investigation are too blunt for the purpose asked of them, no amount of control data will help. They may even deceive people into thinking that proper scientific rigour has been applied.

Riley *et al* appear to have listened to commonly recited, but uncorroborated views of others, rather than checked the original sources. Pelser and I[4] have given examples of how this has often delayed scientific progress for years.

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1 Paulley JW. Ulcerative colitis: a study of 173 cases. *Gastroenterology* 1959; **16**: 566-76.
2 McMahon AW, Schmitt P, Paterson JF. Personality differences between inflammatory bowel disease patients and their healthy siblings. *Psychosom Med* 1973; **35**: 91-103.
3 Roubicek J, Martonova F. Psychopathology of ulcerative colitis. *Cesk Psychiatr* 1957; **53**: 220-30.
4 Paulley JW, Pelser HE. *Psychological management for psychosomatic disorders*. Heidelberg: Springer Verlag, 1989: 38, 62, 324-5.

Effects of albumin infusion in cirrhotic patients

SIR,—Intravenous albumin infusion has been reported as an effective treatment of hyponatraemia in cirrhotic patients with ascites (McCormick *et al*, *Gut* 1990; **31**: 204–7). The derangement in renal sodium handling in cirrhosis is well known; however, the mechanisms mediating this abnormality remain incompletely defined.[1-3] Changes in effective circulatory volume trigger hormonal alterations inducing sodium and water retention. A large proportion of cirrhotic patients with ascites formation show decreased effective plasma volume, activated vasoconstrictor hormone systems, hypoalbuminaemia, and hyponatraemia.[4,5]

We investigated 15 patients with liver cirrhosis and ascites (5 women, 10 men, aged 52–65 years). Patients were on longterm diuretic treatment and a low sodium diet containing 30 mmol/day of sodium. An intravenous infusion of 20% albumin was given in a dose of 1 g/kg. The diuretic and natriuretic responses as well as the albumin induced changes in vasoactive hormone profile were measured.

Albumin infusion induced nearly a fourfold increase in diuresis and sodium excretion in nine of 15 patients (group A), with the normalisation of serum sodium (Table). Albumin also increased the plasma level of atrial natriuretic factor (ANF) to normal, while decreasing the high plasma renin activity

Urine flow rate (U_V), sodium excretion ($U_{Na}V$), serum concentrations of sodium (Na) and albumin (alb), and plasma concentrations of atrial natriuretic factor (ANF), plasma renin activity (PRA), and vasopressin (AVP) (mean (SEM))

	U_V (ml/min)	$U_{Na}V$ (μmol/min)	Na (mmol/l)	alb (g/l)	ANF (fmol/ml)	PRA (ng/ml/h)	AVP (pg/ml)
Group A (n=9):							
Control	0·7 (0·2)	40 (6·8)	130 (1·4)	29·1 (1·5)	19·5 (30)	4·4 (1·0)	8·5 (1·5)
Albumin	2·4 (0·2)*	147 (20)*	135 (1·2)*	34·0 (1·2)*	49·5 (6·6)*	1·9 (0·3)†	6·5 (0·8)
Group B (n=6):							
Control	1·2 (0·3)‡	60 (8·0)‡	136 (2·0)‡	34·0 (0·7)‡	36·7 (3·9)‡	0·44 (0·09)§	4·0 (0·5)‖
Albumin	0·9 (0·2)	73 (11)	136 (1·5)	52·0 (1·2)*	31·0 (2·9)	0·23 (0·05)*	5·9 (1·7)

*p<0·001 *v* control; †p<0·01 *v* control; ‡p<0·001 *v* group A-control; §p<0·01 *v* group A-control; ‖p<0·05 *v* group A-control.