

Mr John Alexander-Williams

New President of the British Society of Gastroenterology 1986/7

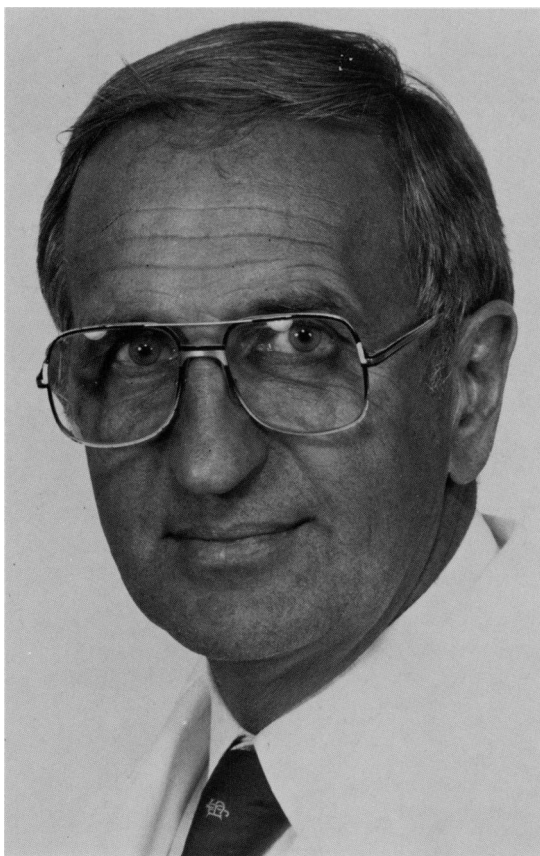
Mr John Alexander Williams becomes the President of our Society in its Jubilee year. The Society could not have chosen a better President to head its business in the year marking the 50th anniversary of its existence.

Mr Alexander Williams brings to the post a brilliant and distinguished career in clinical surgery and surgical research and an analeptic personality which will provide a proper focus for official celebrations.

Mr Alexander Williams trained at the University of Birmingham and is a consultant surgeon at the Birmingham General Hospital. He has been Hunterian Professor at the Royal College of Surgeons on

two occasions and his many distinctions include visiting professorships in Australia, Switzerland, United States, The Netherlands, South Africa, Egypt, and France. He has lectured and chaired scientific meetings all over the world and has held office in numerous national and international societies, editorial boards, and committees.

His department has a steady flow of postgraduate Fellows from all corners of the globe. Mr Alexander Williams's surgical and research interests embrace the management of gastric and biliary disease, and Crohn's disease. He has written extensively on the sequelae of gastric operations, inflammatory colonic disease, and perianal conditions and is an international authority in these areas. His immense energy and enjoyment of life spill over into skiing and swimming and he is also interested in painting, drawing, and writing. Foreign travel and joking are idiosyncratic pursuits of his. We look forward with pleasure to his Presidency of the British Society of Gastroenterology.



Correspondence

Effect of cimetidine in pancreatic steatorrhoea

SIR,—May we reply to the letter from Drs Schöni and Kraemer (*Gut* 1986; 27: 350–1) about our recent report¹ on the effect of cimetidine on fat digestion and solubilisation in cystic fibrosis? We did not examine the effect of cimetidine on fat absorption in our study, but it is reasonable to argue that it will depend, in part, on the effect on fat solubilisation. We cited six studies in which adjunctive cimetidine treatment improved fat absorption.

Whether this improves nutrition is more contentious as there are many determinants of nutritional status in cystic fibrosis. We agree that no long term trial has shown that cimetidine treatment improves nutritional status, but we think that this reflects problems in the design of published trials rather than the efficacy of the treatment.

A clinical trial investigating the efficacy of cimetidine in improving nutrition clearly should recruit only patients in whom the clinician would consider using the drug—that is, those who are severely malnourished, who have severe steatorrhoea, and in whom adjunctive cimetidine has proved effective in improving fat absorption (not all patients respond). In trials using enteric coated pancreatin (the most widely used preparation in the UK) in the 'conven-

tional' period, a non-enteric-coated preparation should be substituted in the 'cimetidine' period as enteric coating delays tablet dissolution even at pH 6. Finally, and most importantly, the diet should be changed between the two periods to take full advantage of cimetidine's effect. An improvement in fat excretion from, say, 50 g/d to 10 g/d would give the patient an extra 360 Kcal/d from fat and correspondingly less from protein and perhaps carbohydrate. This is insufficient to improve nutritional status markedly. What cimetidine does is to decrease the slope of the regression of fat excretion on fat intake, as shown by the Toronto group.² Patients whose intake is, as it usually is, limited by symptomatic steatorrhoea should therefore be able to eat as well as absorb more with adjunctive cimetidine treatment. Only by maximising fat intake separately during the 'conventional' and 'cimetidine' periods can the full nutritional effect of the drug be assessed. In effect, the trial is of a therapeutic strategy based on cimetidine, rather than of the pharmacological effect of the drug.

In our experience the effect of cimetidine, in carefully selected patients, is usually obvious both in terms of fat absorption and nutritional status. If it is not, or if airways resistance increases on routine monitoring, the drug should be stopped. Whether improving nutrition by this means improves pulmonary function is a more worthy, but as yet unanswered, problem for investigation in a controlled trial.

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References

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- 2 Durie PR, Bell L, Linton W, Corey ML, Forstner GG. Effect of cimetidine and sodium bicarbonate on pancreatic replacement therapy in cystic fibrosis. *Gut* 1980; **21**: 778-86.

Dietary essential fatty acids and peptic ulcer disease
SIR,—In a recent leading article (*Gut* 1986; **27**: 239-42) Hollander and Tarnawski presented data to support their hypothesis that the decline in peptic ulcer disease over the past few decades is associated with the increased intake of the dietary essential fatty acid, linoleic acid, during the same period.

Their evidence was both epidemiological and metabolic and may be criticised on both grounds.

The authors provide experimental evidence that the intragastric administration of either linoleic or arachidonic acid reduces the severity of rat gastric ulceration. Both acids were presented in the free form together with an appropriate solubilizer. Free fatty acids or their salts (soaps) are not consumed by man whose dietary fat is entirely esterified mainly as triglyceride and with small amounts of phospholipid. Proximal to the pancreatic duct (where the great majority of peptic ulcers occur), dietary fat remains esterified and in the non-aqueous phase. As such it is not available for exchange with intestinal cells. Furthermore, arachidonic acid is consumed by man in extremely small amounts because arachidonic is neither a component of vegetable fats, nor is it a component of the triglycerides of animal fats. Together these account for over 99% of our dietary fat. The small amount of dietary arachidonic acid consumed by man (ca 25 mg/d or 0.03% fatty acids) is derived from meat and milk phospholipids. Even this remains esterified proximal to the availability of pancreatic lipase. It is thus difficult to see how free linoleic acid or arachidonic acid could be made available in the human gut in that region where ulceration normally occurs and hence a local effect is difficult to envisage.

To hypothesise that increased local cytoprotection of the intestinal mucosa may arise from an increased metabolic pool of arachidonic acid, through intakes of dietary linoleic acid is also difficult to accept. Arachidonic acid is found almost exclusively in the phospholipid fraction of serum lipoproteins and cell membranes where it contributes about 25% of the fatty acids in the Sn-2 position. The turnover of this pool is extremely slow in unstimulated cells and consequently a prolonged period of essential fatty acid deficiency is required to reduce the phospholipid levels of arachidonic acid. Such a deficiency state occurs in man only under extreme circumstances such as the misuse of parenteral nutrition. On the other hand, raising the intake of dietary linoleic acid does not increase the cellular pool of arachidonic acid as that is determined by the mass of phospholipid (and hence membrane) per cell. There is no evidence from animal studies to suggest that dietary corn oil, rich in linoleic acid, increases cell or serum phospholipid arachidonic acid above that achieved when the nutritional requirements for essential fatty acids are met.

The authors propose that the factor linking the fall in the incidence of peptic ulcer disease and the rising intake of essential fatty acids is prostaglandin E₂ (PGE₂). The eicosanoids are undoubtedly linked to peptic ulcer disease but, surely, any factor