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Tumour necrosis factor and platelet activating factor in stool during salmonellosis

EDITOR,—We read with interest the work of Harendra de Silva *et al* (*Gut* 1993; **34**: 194–8) reporting interleukin 6 and tumour necrosis factor (TNF) in the stool of children with *Shigella dysenteriae* infection. We are particularly interested by data reporting the absence of TNF in the stool of patients with Salmonella infection.

Platelet activating factor (PAF) is a phospholipid mediator implicated in gastric ulceration and ischaemic bowel necrosis.[1] Tumour necrosis factor generates PAF production in human monocytes.[2] A close relation has been reported between PAF and TNF in the gastrointestinal tract where PAF mediates TNF induced damage.[3,4] Platelet activating factor and TNF have been reported in the stool of patients with inflammatory bowel disease.[5,6] Furthermore PAF is released in the stool of patients with bacterial (*Salmonella*, *Clostridium difficile*) but not with viral (rotavirus, adenovirus) or parasitic (*Cryptosporidium*) diarrhoea.[7] The lack of TNF in the stool of patients with *Cryptosporidium* or rotavirus may be related to the absence of a faecal PAF. The lack of TNF in the stool of patients with salmonellosis is, however, surprising and suggests, for the first time, that in some gut inflammatory states TNF is not essential for the amplification or initiation, or both of PAF release. To confirm this hypothesis it could be of interest to assess faecal TNF concentrations, for example during *Clostridium difficile* colitis. The lack of TNF and interleukin 6 in the stool of patients with salmonellosis strengthens the putative role of PAF in the ulceration and inflammation seen in the gastrointestinal tract of these patients.

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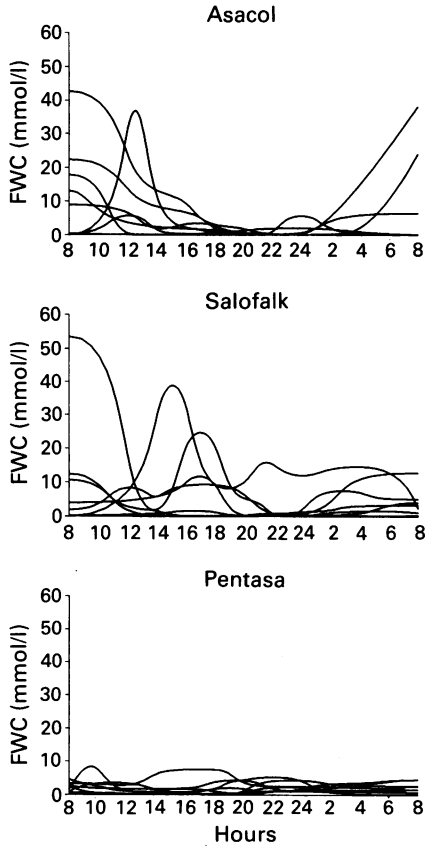
Oroileal transit of 5-aminosalicylic acid

EDITOR,—We read with much interest the elegantly performed study by Goebell *et al* (*Gut* 1993; **34**: 669–75) concerning the fate of 5-aminosalicylic acid (5-ASA) from Salofalk in the small intestine. The study elucidates some important aspects on the bioavailability of 5-ASA (pH of the gut lumen, the intestinal transit time).

We would like to comment, however, on the interpretation of the results. The authors conclude, that 30% of the ingested dose passed the ileum in solution, which is similar to the results from ileostomates on Salofalk.[1] Another 10% was found in the urine, and it is therefore concluded that 60% reach the colon in unreleased form. The design of the study does not permit this conclusion because the localisation of undissolved tablets was not assessed. In fact, some tablets could still be retained in the stomach. As the authors point out, the gastric retention time is highly variable.

Moreover, when the Salofalk tablet dissolves, its content of 5-ASA is released within 30 minutes,[2] so mean values showing that 1·5% of the content is released in the duodenum, 5·7% in the jejunum, and 12·7% in the ileum are misleading. Individual data for the six subjects would yield more accurate information.



Concentration of 5-aminosalicylic acid in ileostomy fluid. FWC=faecal water concentration.

We have studied ileostomy patients during steady state treatment with different 5-ASA preparations.[1] The subjects were given 2 g 5-ASA daily (two tablets of Salofalk (250 mg) four times daily, 400 mg Asacol five times daily, and Pentasa 500 mg four times daily) half an hour before the meals, as suggested by Goebell *et al*, and the concentration of 5-ASA was measured in the ileostomy output for 24 hours. Despite the dose being given four–five times daily, only one–two peak concentrations were seen for Asacol and Salofalk, and a lower but steady concentration during Pentasa (Figure), emphasising the importance of the size of the drug formulation for gastric retention time.

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1 Christensen LA, Fallingborg J, Abildgaard K, Jacobsen BA, Sanchez G, Hansen SH, *et al*. Topical and systemic availability of 5-aminosalicylate: comparisons of three controlled release preparations in man. *Aliment Pharmacol Therap* 1990; **4**: 523–33.
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Comparison of computed tomography, endosonography, and intraoperative assessment in TN staging of gastric carcinoma

EDITOR,—We read the report by Ziegler *et al* (*Gut* 1993; **34**: 604–10) and were impressed by the quality of the computed tomography and endogastric ultrasonograms, and by the accuracy of their endosonographic assessment of gastric tumours and node state compared with subsequent histological examination. The argument, however, which the authors use to justify their conclusion that 'endogastric ultrasonography should be introduced into the preoperative assessment of patients with gastric carcinoma' is flawed.

The authors have presented no data that support the claim of the final paragraph of their paper that 'as endogastric ultrasonography has by far the highest sensitivity and specificity for correct TN classification, the introduction of this technique in the preoperative diagnostic programme allows much better selection of inoperable patients'. They describe a comparison between computed tomography, endogastric ultrasonography, and intraoperative clinical assessment in a series of 108 patients, all of whom had total gastrectomy for their gastric tumours. They do not describe the computed tomographic findings, endogastric ultrasonographic findings or clinical assessment in any patient with inoperable tumours, and the data they present, while interesting, cannot therefore be used to support their claim relating to the selection of inoperable patients.

As clinicians, most of us would be very interested in any technique that would permit the reliable preoperative prediction of inoperability, and in some ways the paper has missed an opportunity to make an assessment of the potential clinical usefulness of endosonography. The authors must have imaging data that relate to patients who were subsequently found to be inoperable at laparotomy, and we would be very interested to see these results,

particularly of their endogastric ultrasonographic assessment. Furthermore, in terms of the avoidance of a formal laparotomy in patients who are subsequently deemed inoperable, diagnostic laparoscopy may well have a significant part to play, and we would also be interested in any data the authors have that specifically compare laparoscopy with the modalities described in their paper.

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Bile duct injury after laparoscopic cholocystectomy

EDITOR,—I read with interest the report of Davids *et al* (*Gut* 1993; **34**: 1250–4) describing their experience of ERCP in the management of patients with bile duct injuries after laparoscopic cholecystectomy. We too have found an increasing incidence of such injuries after laparoscopic cholecystectomy, compared with open cholecystectomy, as the first procedure becomes more widely performed. I agree that early ERCP is the investigation of choice and like Davids *et al* have found that bile duct dilatation has not always been detected by abdominal ultrasound.

In my opinion the authors failed to mention the most probable cause for strictures in the mid common bile duct. I believe these are caused by thermal damage produced by the diathermy hook during dissection. There is a risk of such injury when an L shaped hook is used. While dissecting with the tip of the hook, the heel can cause unnoticed damage possibly due to conduction of electricity along a previously placed metal clip. The delayed necrosis of the bile duct and the resultant very localised inflammatory reaction would, I think, explain the initial 'silent' anicteric period to which the authors refer. It would also explain the very localised area of dense fibrosis, which I have personally found surrounding the bile duct stricture.

I have treated two such patients, who have been referred to me, by hepaticojejunostomy. Although I accept that endoscopic stenting will be possible in many of these patients, I disagree that it should be used preferentially in young patients simply because 'a proximal hepaticojejunostomy seems unattractive' and endoscopic stenting seems 'less invasive'. Longterm efficacy should be the yardstick by which any treatment is judged rather than the degree of therapeutic 'invasion' necessary. The authors at present are in no position to recommend endoscopic stenting after such a short follow up. Successfully performed hepaticojejunostomy has been shown to be effective in the long term and not associated with significant morbidity. I would suggest that the effects of chronic culminative low grade sepsis, which are associated with the longterm use of indwelling endoprostheses mitigate against its use in the young. An admittedly difficult operation when performed by someone experienced in the field, offers the best chance of a longterm cure particularly if performed early and before infection has been introduced into the area.

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NOTES

Gut 1993; **34**: 1297–9. The editor would like to make clear that the recommendations in this paper apply to adults and are not relevant to children.

Budd-Chiari Syndrome

The Third International Symposium on Budd-Chiari Syndrome will be held in London on 11–13 April 1994. Further information from Professor K E F Hobbs, University Department of Surgery, Royal Free Hospital, Pond Street, London NW3 2QG. Tel: 44 71 435 6121; fax: 44 71 431 4528.

Gastrointestinal motility

The 7th European Symposium on Gastrointestinal Motility, will be held in Toulouse, France on 7–9 July 1994. Further information from: Lionel Bueno, Europa Organisation, 40 Boulevard des Récollets, BP 4406, F-31405 Toulouse, France. Tel: 33 61 32 66 99; fax: 33 61 32 66 00.

Digestive endoscopy course

The European Postgraduate Gastro-Surgical School is organising this course at the Academic Medical Center of the University of Amsterdam, Amsterdam, The Netherlands on 8–9 September 1994. For information contact: Helma Stockmann, Managing Director European Postgraduate Gastro-Surgical School, Room G4–109.3, Academic Medical Center, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands. Tel: 31 20 5663926; fax: 31 20 6914858.

European Pancreatic Association

The 26th meeting of the European Pancreatic Association will be held in Bologna, Italy on 8–10 September 1994. Further information from: Professor Lucio Gullo, Institute of Medicine and Gastroenterology, St Orsola Hospital, 40138 Bologna, Italy. Fax: 39 51 392538; tel: 39 51 6364129.

Bile acids in gastroenterology

The XIIIth International Bile Acid Meeting: Bile Acids in Gastroenterology will be held in San Diego, California on 30 September–2 October, 1994. Further information from: Cass Jones, Professional Conference Management, Inc, 7916 Convoy Court, San Diego, CA 92111 USA. Tel: 619 565 9921; fax: 619 565 9954.

Endoscopic Surgery

The 2nd Asian Pacific Congress of Endoscopic Surgery will be held in Hong Kong from 19–23 June 1995. Further information to Dr Sydney Chung, Department of Surgery, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong. Tel: 852 636 2627; fax: 852 645 3602.