Effects of olsalazine and sulphasalazine on jejunal and ileal water and electrolyte absorption in normal human subjects

A H Raimundo, D H Patil, P G Frost, D B A Silk

Abstract

The effect of sulphasalazine and olsalazine on jejunal and ileal water and electrolyte absorption was investigated in normal subjects by a steady state intestinal perfusion of a physiological glucose bicarbonate electrolyte solution in the absence and presence of increasing concentrations of each drug. (Olsalazine 0-25 g/l, 1-0 g/l, jejunum; 0-5 g/l, 1-0 g/l, ileum; sulphasalazine 0-25 g/l, 0-5 g/l, 2-0 g/l jejunum; 1-0 g/l, 2-0 g/l, ileum.) In the jejunum olsalazine at 1-0 g/l significantly inhibited water, sodium, chloride, and potassium absorption (p<0-05). In the ileum olsalazine at 0-5 and 1 g/l significantly inhibited glucose uptake (p<0-04) and water absorption (p<0-03). In the jejunum sulphasalazine had a dose related and significant inhibitory effect on water, bicarbonate, and sodium absorption and at 2-0 g/l an inhibitory effect on chloride, potassium (p<0-005), and glucose (p<0-05) absorption. In the ileum sulphasalazine had no significant effect on water and electrolyte absorption. All inhibitory effects were rapidly reversible. These data show that unexplained diarrhoea in patients with ulcerative colitis treated with olsalazine may occur as a consequence of inhibition of water and electrolyte absorption in the small intestine and that the mechanisms of inhibition of sulphasalazine and olsalazine are different.

Sulphasalazine has been widely used for mild or moderately severe attacks of ulcerative colitis and has been the mainstay of maintenance treatment for the prevention of relapse. After oral ingestion about 75% of the dose of sulphasalazine remains intact until it reaches the colon where it is split by bacterial azo reductases into salicylic acid and 5 aminosalicylic acid. The 5 aminosalicylic acid is considered to be the active moiety of sulphasalazine, exerting its therapeutic effect locally on the inflamed mucosa. Side effects, which occur in about one third of patients with inflammatory bowel disease, are thought to be related to the salicylapyridine moiety which is readily absorbed from the colon. To overcome the high incidence of side effects a new sulphafree component olsalazine (disodium azosalicylate) has been synthesised (Pharmacia AB, Sweden). Two molecules of 5 aminosalicylic acid are linked by an azo bond to form olsalazine, and when it is taken orally, most passes unchanged through the small intestine into the colon where complete azo reduction occurs releasing two molecules of 5 aminosalicylic acid. Recent clinical studies show that olsalazine is effective in controlling mildly active distal ulcerative colitis as well as being an effective agent for maintaining remission. Although it has been shown that olsalazine was tolerated by patients who were unable to tolerate sulphasalazine, unexplained diarrhoea occurred in 9-8% of patients treated with the drug. Recent animal studies suggest that sulphasalazine may exert a deleterious effect on intestinal glucose, water, and electrolyte absorption. The aim of the present study was to extend these findings to the investigation of the effect of the drug on water, glucose, and electrolyte transport in the normal human small intestine. Surprisingly, there seemed to be no published data on the effect of sulphasalazine on water and electrolyte handling by the small intestine. We therefore investigated the effect of sulphasalazine on glucose, water, and electrolyte absorption. All experiments were performed in normal subjects using an in vivo steady state perfusion technique.

Methods

Intestinal perfusion experiments were carried out in a total of 28 normal subjects, mean age 26 years, range 20-45 years. All gave informed consent and the study was approved by the Ethical Committee of Central Middlesex Hospital.

Of the 28 subjects, 16 had jejunal perfusions and 12 ileal perfusions. After an overnight fast each of the 16 subjects undergoing jejunal perfusions swallowed a double lumen perfusion tube incorporating a proximal occluding balloon. The tube was allowed to pass until the occluding balloon was situated just beyond the duodenal-jejunal flexure. The final position of the tube was checked fluoroscopically to confirm that the 30 cm perfusion segment was positioned in the proximal jejunum just beyond the ligament of Treitz. After an overnight fast each of the 12 subjects undergoing ileal perfusions swallowed a similar perfusion tube incorporating a proximal occluding balloon. The tube was modified to contain a 15 cm perfusion segment. The tube was allowed to pass until the 15 cm perfusion segment was judged radiologically to be situated in the distal ileum. In practice, the distal end of the tube was sited 180-220 cm from the teeth of each subject.

Test solutions contained bicarbonate ions (30 mmol/l) and ileal positioning was confirmed by comparing the differential handling of bicarbonate ions in the jejunum and ileum. This technique has been described. Perfusion solutions were continuously stirred and maintained
Effects of olsalazine and sulfasalazine on jejunal and ileal water and electrolyte absorption in normal human subjects

TABLE I  Composition of solutions

<table>
<thead>
<tr>
<th>Control solution: 1000 ml</th>
<th>Osmolarity mOsm/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>NaCl 105 mmol = 6.1 g</td>
<td>290</td>
</tr>
<tr>
<td>KCl 5 mmol = 0.37 g</td>
<td></td>
</tr>
<tr>
<td>NaHCO3 30 mmol = 2.52 g</td>
<td></td>
</tr>
<tr>
<td>Glucose 10 mmol = 1.9 g</td>
<td></td>
</tr>
<tr>
<td>Polyethylene glycol 4 g</td>
<td></td>
</tr>
<tr>
<td>C14 1 µg/L</td>
<td></td>
</tr>
</tbody>
</table>

Control + olsalazine 0-25 g/l
Control + olsalazine 0-5 g/l
Control + olsalazine 1 g/l
Control + sulfasalazine 0-25 g/l
Control + sulfasalazine 0-5 g/l
Control + sulfasalazine 1 g/l
Control + sulfasalazine 2 g/l

at 37°C in a water bath and were perfused at a rate of 15 ml/min using a constant infusion peristaltic pump (Watson-Marlow Ltd MHRE 200).

After a basal 30 minute perfusion with each solution, to achieve steady state conditions, three samples were taken from the distal collecting port, at 10 minute intervals, by siphoning into plastic containers and were chilled on crushed ice. The intestinal aspirates were then stored at 4°C until final analysis was carried out on the following day.

COMPOSITION OF PERFUSION SOLUTIONS AND EXPERIMENTAL DESIGN

The perfusion solutions were isosmolar with plasma and the composition is shown in Table I.

STUDIES WITH OLSALAZINE

The effect of olsalazine on jejunal glucose, water, and electrolyte absorption was investigated in seven subjects and the effect of olsalazine on ileal glucose, water, and electrolyte absorption in six subjects. In the jejunal experiments each subject was perfused first with a control glucose bicarbonate electrolyte solution (Table I). Two solutions of the same composition except for the addition of olsalazine (0-25 g/l; 0.7 mmol/l; 1 g/l; 2-8 mmol/l) were then perfused. The reversibility of the effects was checked by repeat perfusion of the control solution.

In the ileal studies the same protocol was adopted, except that the two test solutions contained 0.5 and 1 g/l of olsalazine (1.4 and 2.8 mmol/l respectively).

STUDIES WITH SULPHASALAZINE

The effect of sulfasalazine on jejunal glucose, water, and electrolyte absorption was investigated in nine subjects and its effects on ileal glucose, water, and electrolyte absorption in six subjects. In the jejunal experiments the same control solution was perfused in all subjects. Two of three test solutions containing sulfasalazine (0-25 g/l, 0.6 mmol/l n = 5; 0.5 g/l, 1-2 mmol/l n = 5; 2 g/l, 5 mmol/l n = 8) were then perfused following of reperfusion of the control solution in seven of nine of the normal subjects. In the ileal studies the same protocol was adopted except that the two test solutions contained 1 g and 2 g/l sulfasalazine (2.5 and 5 mmol/l respectively).

ANALYSIS

The C14-polypethylene glycol content was measured by liquid scintillation counting using a Beckman L5 7500 counter (Beckman R11C, High Wycombe, Bucks, UK). Quench correction was performed using the H number method. Before counting, samples were decolourised by incubating 1 ml aliquots with 0-5 ml domestic bleach (Melzone), at 50°C for 20 minutes, followed by vacuum extraction to remove excess chlorine. To prevent chemiluminescence these samples were then mixed with 5 ml distilled water and 10 ml Beckman-MP scintillation fluid and stored in the dark for four days before the radioactivity was counted.

Sodium, potassium, and bicarbonate from control solutions and intestinal aspirates were measured using a Beckman Astra autoanalyzer (Beckman Instruments, USA). Chloride concentrations were measured using a Chloride Meter (Corning Eel 920); glucose was measured by an ERIS analysis (Gluc-DH, Merck). Osmolality was measured by freezing point depression with an Advanced Osmometer (Advanced Instruments, Massachusetts, USA).

STATISTICS

Data are presented as mean (SE). Absorption rates of water and solutes were calculated using previously described formulas. The significance of perfusion of water, sodium, potassium, chloride, bicarbonate, and glucose compared to control values was determined by using the paired t test.

Results

EFFECTS OF OLSALAZINE

Jejunum (Table II)

When the control solution was perfused at the start of each study it was found that glucose and bicarbonate at concentrations of 10 and 35 mmol/l, respectively, significantly stimulated

<table>
<thead>
<tr>
<th>Water (n=7)</th>
<th>Sodium (n=7)</th>
<th>Potassium (n=7)</th>
<th>Chloride (n=7)</th>
<th>Bicarbonate (n=7)</th>
<th>Glucose (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>167 (17)</td>
<td>22.9 (2.9)</td>
<td>0.73 (0.09)</td>
<td>15.9 (2.2)</td>
<td>7.2 (0.6)</td>
<td>7.7 (1.0)</td>
</tr>
<tr>
<td>24 (4-6)*</td>
<td>17.6 (1-9)</td>
<td>0.51 (0.1)</td>
<td>11.3 (3)</td>
<td>7.9 (1.2)</td>
<td>6.4 (0.5)</td>
</tr>
</tbody>
</table>

*p<0.05 from control values (paired t test); **p<0.005.

igut: first published as 10.1136/gut.32.3.270 on 31 March 1991. Downloaded from http://gut.bmj.com/ on November 1, 2023 by guest. Protected by copyright.
the jejunal absorption of sodium, water, and electrolytes (p<0.05). The addition of 0.25 g/l of olsalazine resulted in a significant inhibition of jejunal water absorption (p<0.05) but had no effect on sodium, chloride, and potassium absorption. Glucose and HCO₃ absorption were also unaffected. Olsalazine at the higher concentration of 1 g/l had a significant inhibiting effect on water, sodium, and chloride uptake as well as potassium uptake (p<0.05). Olsalazine at 1 g/l did not affect glucose or bicarbonate uptake. The inhibitory effect of olsalazine on water and electrolyte uptake was rapidly reversible because the absorption values for water, sodium, and chloride during perfusion of the control solution at the end of the study were not significantly different from those at the beginning of the study.

**Jejunum (Table IV)**

As in the olsalazine experiments, glucose and bicarbonate in the control solution significantly stimulated jejunal water and electrolyte absorption during the initial perfusions. At all three concentrations studied (0.25, 0.5, and 2 g/l) sulphasalazine had a significant inhibitory effect on jejunal sodium and water absorption. The inhibitory effect of sulphasalazine was dose related. Sulphasalazine also had a significant inhibitory effect on jejunal bicarbonate uptake, which was also dose related. At the highest concentration of 2 g/l, sulphasalazine inhibited glucose uptake. The inhibitory effects of sulphasalazine were rapidly reversible. The absorption values during perfusion of the control solution at the end of the study were similar and not significantly different from those at the beginning of the study.

**Ileum (Table III)**

In the ileum olsalazine had a significant (p<0.04) inhibitory effect on water absorption and glucose uptake at both concentrations and had no effect on the ileal absorption of sodium, potassium, chloride, and bicarbonate. The inhibitory effect of olsalazine on water absorption and glucose uptake was rapidly reversible.

**Ileum (Table V)**

Sulphasalazine had no significant effect on water, electrolyte, and glucose uptake in the ileum.

---

**TABLE III**  Effect of intraluminal olsalazine on absorption of water, Na⁺, K⁺, CI⁻, HCO₃⁻, and glucose in the human ileum (mean (SEM))

<table>
<thead>
<tr>
<th></th>
<th>Water (ml/h/15 cm)</th>
<th>Sodium (mmol/l/15 cm)</th>
<th>Potassium (mmol/l/15 cm)</th>
<th>Chloride (mmol/l/15 cm)</th>
<th>Bicarbonate (mmol/l/15 cm)</th>
<th>Glucose (mmol/l/15 cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=6)</td>
<td>44.8 (11.1)</td>
<td>3.9 (4.6)</td>
<td>0.0 (0.15)</td>
<td>1.7 (3.4)</td>
<td>1.9 (0.6)</td>
<td>3.9 (0.9)</td>
</tr>
<tr>
<td>Olsalazine (n=6)</td>
<td>4.1 (14.7)*</td>
<td>2.8 (4.3)</td>
<td>-0.7 (0.16)</td>
<td>-3.7 (4.1)</td>
<td>1.4 (1.3)</td>
<td>3.9 (0.9)</td>
</tr>
<tr>
<td>Olsalazine (n=6)</td>
<td>15.1 (10.9)†</td>
<td>9.3 (2.7)</td>
<td>0.23 (0.1)</td>
<td>4.5 (2.1)</td>
<td>2.6 (0.7)</td>
<td>5.0 (0.72)**</td>
</tr>
<tr>
<td>Control (n=6)</td>
<td>44.7 (9.9)</td>
<td>2.6 (4.9)</td>
<td>0.03 (0.13)</td>
<td>6.2 (3.0)</td>
<td>5.0 (1.46)</td>
<td>6.1 (0.57)</td>
</tr>
</tbody>
</table>

*p<0.03 from control values (paired t test); †p<0.02; ‡p<0.04 from 0.5 g/l olsalazine.

---

**TABLE IV**  Effect of sulphasalazine on absorption of water, Na⁺, K⁺, CI⁻, HCO₃⁻, and glucose in the human jejunum (mean (SEM))

<table>
<thead>
<tr>
<th></th>
<th>Water (ml/h/15 cm)</th>
<th>Sodium (mmol/l/15 cm)</th>
<th>Potassium (mmol/l/15 cm)</th>
<th>Chloride (mmol/l/15 cm)</th>
<th>Bicarbonate (mmol/l/15 cm)</th>
<th>Glucose (mmol/l/15 cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=9)</td>
<td>153 (17.3)</td>
<td>21.2 (2.2)</td>
<td>0.58 (0.09)</td>
<td>12.1 (1.7)</td>
<td>10.4 (0.8)</td>
<td>7.6 (0.3)</td>
</tr>
<tr>
<td>Sulphasalazine (n=5)</td>
<td>134 (23)*</td>
<td>17.1 (3.6)*</td>
<td>0.45 (0.1)</td>
<td>12.2 (2.6)</td>
<td>8.8 (1.2)*</td>
<td>6.9 (0.4)</td>
</tr>
<tr>
<td>Sulphasalazine (n=5)</td>
<td>117 (22.7)*</td>
<td>15.8 (2.7)*</td>
<td>0.43 (0.1)</td>
<td>10.6 (1.8)</td>
<td>7.8 (1)*</td>
<td>6.9 (0.6)</td>
</tr>
<tr>
<td>Sulphasalazine (n=5)</td>
<td>39 (19.2)**</td>
<td>4.5 (2.5)**</td>
<td>0.02 (0.1)**</td>
<td>-0.05 (2.2)**</td>
<td>5.4 (0.6)**</td>
<td>6.8 (0.4)**</td>
</tr>
<tr>
<td>Sulphasalazine (n=8)</td>
<td>142 (21.5)</td>
<td>21 (3.1)</td>
<td>0.61 (0.12)</td>
<td>12.3 (3.3)</td>
<td>8.8 (1)</td>
<td>7.4 (0.4)</td>
</tr>
</tbody>
</table>

*p<0.05 from control values (paired t test); **p<0.005.
Discussion

The results of this study clearly show that olsalazine significantly inhibits jejunal water and electrolyte absorption from a glucose bicarbonate electrolyte solution perfused in normal human subjects. These findings are similar to the findings in animal experiments. In addition, sulphasalazine significantly inhibited water and electrolyte absorption. This is a new finding, as the effects of this drug have not been previously studied in humans. The inhibitory effect of both drugs was rapidly reversible and suggests that weak binding to an epithelial surface component is responsible rather than morphological damage to the brush border, as seen with neomycin.

The perfusion technique in this study measures only net luminal disappearance rates, so it is difficult to draw firm conclusions on the mechanism of the inhibitory effect of the two drugs in the jejunum. It seems, however, that in the jejunum the two drugs act differently. Sulphasalazine had a dose-related inhibitory effect on jejunal bicarbonate and sodium uptake (Table IV) and its effect may have been specific to bicarbonate-stimulated absorption, which in humans depends on $\text{H}^+/{\text{Na}}^+$ exchange.

Olsalazine, however, had no effect on jejunal bicarbonate or glucose uptake, suggesting a different effect on sodium and consequent water uptake.

A recent study by Sandberg-Gertzen et al. showed that ileostomy outputs during oral treatment with olsalazine (1 g and 2 g/day) were significantly increased whereas no change occurred after the administration of sulphasalazine (2 g/day). Given that our jejunal perfusion data indicate that both drugs have an inhibitory effect on sodium and water absorption in the jejunum, and only olsalazine was associated with increases in ileostomy effluent volumes, it seemed likely that the two drugs might exert different effects on ileal water and electrolyte handling. The ileal perfusions were carried out to test this hypothesis. The results of the ileal perfusion experiments showed that olsalazine had a significant inhibitory effect on ileal water absorption (at both concentrations). In contrast, sulphasalazine had no effect on ileal water and electrolyte absorption. This is of interest, particularly because of the suggestion that in the jejunum sulphasalazine may inhibit bicarbonate-stimulated absorption. Bicarbonate ions are secreted in the ileum so that at this site a bicarbonate-stimulated sodium and water mechanism does not occur. In contrast to sulphasalazine, olsalazine seems to exert an inhibitory effect on water absorption which persists throughout the unstimulated glucose bicarbonate solution perfused in normal human subjects. This inhibition is overcome by the apparently normally operative ileal function in the presence of the drug, then an explanation can be offered of why ileostomy effluent volumes do not increase after oral ingestion of the drug. Caution is required when extrapolating the findings of the present intestinal perfusion studies to the normal clinical setting. The in vivo perfusion technique used in the present study is based on assessments of ileal water absorption rates under steady-state conditions. Attempts were made in designing the jejunal experiments to approximate the loads of both sulphasalazine and olsalazine presented to the mucosa to those likely to occur normally after oral ingestion of therapeutic doses of the drugs.

Assumptions have to be made about such important aspects as drug solubility, gastric emptying rates, and postprandial jejunal flow rates. Taking into account these variables, the range of concentrations of olsalazine (0.5–1.0 g/l) and sulphasalazine (0.25, 0.5, and 2 g/l) perfused at 15 ml/min were chosen to simulate as far as possible the intraluminal loads presented to the jejunal mucosa after oral ingestion of the drugs. Ileal perfusion studies, using bicarbonate as a functional marker to confirm correct positioning of the test segment, are technically more demanding to perform than jejunal studies, and our previous experience has shown that this technique can be simplified by using a shorter test segment and higher infusion rates than in jejunal experiments. It is unlikely, therefore, that the drug load presented to the mucosa during the ileal perfusion experiments equates to those present after oral ingestion of therapeutic doses.

As a consequence, the concentrations of the two drugs used in the ileal studies were based on the results of the jejunal experiments rather than on calculations of the dose of the drugs likely to be present in the ileal lumen in the normal clinical setting.

A further notable point is that olsalazine has recently been shown to accelerate appreciably gastric emptying, mouth to caecum transit time, and whole gut transit time. This property might also be responsible for causing the observed increases in the ileostomy effluent volumes during sulphasalazine treatment. The effect of sulphasalazine on gastrointestinal transit has not yet been studied. The unexplained diarrhoea which has been reported in patients with ulcerative colitis treated with olsalazine occurs more commonly in patients with extensive disease. The effects of olsalazine shown here and in the ileostomy studies of Sandberg-Gertzen and her colleagues indicate that 24 hour colonic inflow volumes are likely to be higher than normal in patients treated with the drug. The absorptive function of diseased colonic mucosa obtained from ulcerative colitis patients is reduced. Patients who have extensive disease may therefore be unable to assimilate the common therapeutic inflow volumes associated with sulphasalazine treatment. A functional adaptation to an increase in colonic inflow has been shown to occur in patients without colonic disease. This in turn may explain why patients with distal ulcerative colitis treated with olsalazine have a relatively low incidence of unexplained diarrhoea.

We thank Mr J Rogers for help with the statistical analysis of the data and preparation of the manuscript, and Dr G K Grimble for his assistance with the biochemical analyses. We are grateful to Pharmacia Limited for their interest and financial support which formed part of the funding of this project.
274

Raimundo, Patil, Frost, Silk


16 Goerg KJ, Wanitschke R, Breiling K, Franke M. The effect of disodium-azoxos (DSA) on water and electrolyte transfer of the rat ileum and colon in vivo compared with sulphasalazine (SASP), 5-aminosalicylic acid (SA) and sulfapyridine (SP). Gastroenterology 1984; 86: 1091.


Reply

Sr,- We would like to thank Dr Bell and his colleagues for their comments. We are pleased to learn that they are now using the 13C-urea breath test (13C-UBT) for the detection of Helicobacter pylori and agree that the chromotographic purification of breath samples for isolation of 13CO, before mass spectrometry will help reduce the cost of the analysis. The European standard protocol, however, using either the pooled or single sample technique for breath collection, provides an even greater reduction in the overall cost of the technique. The quantity of isotope used in the European standard 13C-UBT (100 mg) is less than half that used in Graham’s original description of the 13C-UBT. Smaller quantities of isotope have since been used in several small studies without detrimental effect on the sensitivity or specificity of the test. The ability of the 13C-UBT to detect very low levels of H. pylori may, however, be impaired if very small amounts of 13C-urea are used. More specifically, although the trend to use smaller quantities of isotope is welcome, theoretically the intragastric concentration of the isotope should be slightly greater than the K max for the urease of H pylori.

R P H LOGAN
Department of Gastroenterology and Nutrition, Central Middlesex Hospital, London NW10 7NS

S DILL
Department of Gastroenterology, Universität Köln, Kanonstrasse Basel, Basel, Switzerland

Correspondence to: Dr R P H Logan.


BOOK REVIEW


There is much in this book which is satisfying to the reader, yet it suffers from a lack of continuity of information between widely separated chapters, which are certainly complications of the multiple author syndrome. There are two sections, one dealing with concepts of carcinogenesis and the other with the clinical management of premalignant conditions. Under the former there are two well written and instructive chapters on the principles of carcinogenesis and oncogenes. These are followed by contributions on epiphal renewal, DNA flow cytometry, and neoplastic progression in the gastrointestinal tract, and monoclonal antibodies in neoplastic and pre-neoplastic disorders of the large bowel. In the middle of these we are treated to a lengthy chapter on the subject of dysplasia, which is well written but almost entirely concerned with dysplasia in Barrett’s oesophagus and chronic ulcerative colitis. There is overlap with a subsequent chapter on inflammatory bowel disease in the second section on clinical management. Surprisingly, the chapter on dysplasia includes only a short paragraph on the diagnosis and classification of dysplasia in adenomas. One chapter only is devoted to the whole subject of gastrointestinal polyposis and polyposis syndromes. There is inadequate coverage of the epidemiology, genetics, pathology, and evolution of the adenoma-carcinoma sequence. The problems of the malignant potential of juvenile polyposis and the Peutz-Jeghers syndrome are ignored. A major weakness in many of the chapters is the lack of emphasis on the contribution of epidemiology to our understanding of premalignant states. Insufficient space is given to methods of investigation, particularly endoscopy. The main objective in the study of premalignant conditions and histopathological lesions must be prevention and early detection of cancer with reduced mortality. Yet the book provides no sense of thrust in this direction. It is a collection of essays, most of them individually very good, but without the continuity which makes for easy reading. A last criticism. Please could Dukes’ name be spelt correctly. It is the Dukes classification not Dukes’ classification. The production of the book is good with clear print and microphotographs of good quality. A pity that it leaves something to be desired.

B C MORSON

NOTES

Computers in Endoscopy

The 7th International Symposium on Endoscopic Ultrasound will be held in Munich, 14–15 June 1991. Information from: Dr med Thomas Rösch, 11 Medizinische Klinik und Poliklinik der TU, Klinikum rechts der Isar, Ismaninger Strasse 22, DW-8000 München 80, Germany. Tel: 089/4140 2263; fax: 089-4140 2747.

XVIth International Update on Liver Disease

The XVIth International Update on Liver Disease will be held at the Royal Free Hospital and School of Medicine, London, 11–13 July 1991. Information from: Professor Neil McIntyre, Academic Department of Medicine, Royal Free Hospital, Pond Street, London NW3 2QG. Tel: 017-794 0500, ext 3969.

British Society of Gastroenterology meeting

The 1991 Spring Meeting of the British Society of Gastroenterology was held on 10–12 April under the presidency of Professor Sir Robert Shields at the University of Manchester Institute of Science and Technology. UMIST, an important component of the vast Manchester education factory along the Oxford Road, is a new venue for the Society, but the programme was along traditional lines laid down within the last few years, with separate and extensive poster sessions each day complementing the oral presentations. There was no sign of the resurrection of the plenary session, but it is probably too soon for it to be reintroduced as a radical innovation; instead, key lectures served as central foci of the meeting. Professor S M Collins from McMaster University gave the International State of the Art Lecture on "Interactions between the immune and motor systems of the gut." Dr I Biarnason, the 1991 Avery Jones Research Medallist, spoke on NSAID-induced enteropathy, and Professor T J Peters gave a State Lecture on the molecular genetics of alcoholic liver disease. The inclusion of such lectures in the programme is to be commended as much as the trivial title, derived from advertising jargon, of "State of the Art" (with the absurd implication that lectures not so designated are incomplete or obsolete) is to be deplored. Perhaps the society might, in future meetings, emulate the royal colleges by using lectures as an opportunity to commemorate distinguished members who are no longer with us. On the social front, the programme maintained its reputation for innovative local hospitality by giving the endoscopists the ultimate video experience of dinner in Coronation Street at Granada TV. Accompanying persons were given a guided tour of a working cotton mill, perhaps to give them some idea of the working conditions which their companions experienced as house officers. And so to London in the summer.

Correction

Effects of olsalazine and sulphasalazine on jejunal and ileal water and electrolyte absorption in normal human subjects by Raimundo et al, March 1991; 32: 270–6. Table II gives data on the effect of olsalazine in the human jejunum; Table V gives data on the effect of sulphasalazine in the human ileum.