visual assessment of the tracings and photographic records. On reviewing the planimetry the reasons for deciding whether or not to change were unclear in four cases. Furthermore, traces of the dressing could often be seen, and the decision was thus not "blind." The crossover figures must, therefore, be viewed critically. It is noteworthy that when the four cases mentioned were excluded from the analysis the results still favoured cadexomer iodine ($p < 0.05$).

Contrary to the findings of Skog *et al*[3] we did not observe any significant effect on bacterial colonisation. Studies using a simulated wound model, however, have shown that although cadexomer iodine may not eliminate organisms, it does suppress growth for almost 18 hours and thereby significantly reduces bacterial mass.[7] The action of bacteria in inhibiting the healing of wounds may relate to the production of cytotoxic chemicals,[8,9] and reduction of bacterial mass for a substantial proportion of the day may be one of the mechanisms by which cadexomer iodine accelerates regrowth of the epithelium.

Ulcers in both groups healed well. This study shows that selected patients (average age 68 years) can manage their own ulcers effectively. Patient compliance was remarkably good.

Our former opinion, in common with that of many others, was that weekly dressing and bandaging provides adequate support for ulcers with minimal disturbance of new epithelium. On the basis of this study we suggest that daily bandaging and renewal of a non-adherent dressing may have distinct advantages over less demanding regimens. Bandages reapplied daily probably provide better support than those changed less regularly. Furthermore, many of our patients remarked how much the reduction in odour had improved their social lives. Although it takes time and patience to teach patients how to dress and bandage their ulcers—that is, five two hour sessions—this seems to be repaid in terms of both healing of ulcers and, eventually, a reduction in demands on the doctor and nurse. Cadexomer iodine seems to be useful in this context.

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SHORT REPORTS

Gastric emptying in chronic renal failure

Gastrointestinal symptoms almost invariably accompany advanced chronic renal failure, but little is known about the mechanisms responsible. Abdominal distension and nausea occur in patients with reflux oesophagitis, a condition known to be associated with delayed gastric emptying.[1] As there have been few investigations into the effect of uraemia on gastric motility we compared gastric emptying in patients with uraemia with that in healthy controls.

Patients, methods, and results

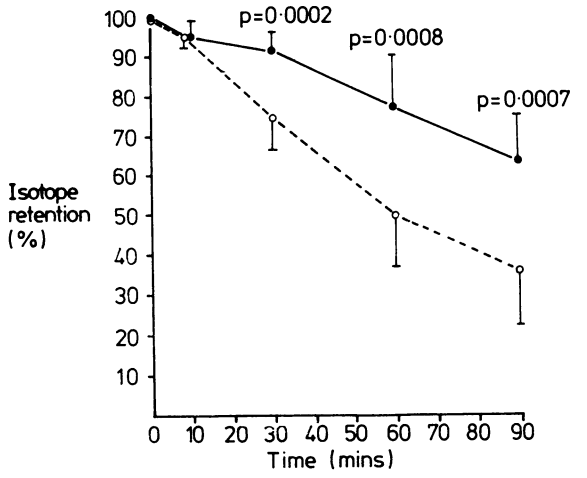
We studied 14 patients with uraemia (eight women), whose ages ranged from 21 to 61 (mean age 37). All had a glomerular filtration rate of less than 5 ml/min, and serum creatinine concentrations ranged from 690 to 1560 μmol/l (7·8 to 17·6 mg/100 ml). Four patients had started regular haemodialysis six to 12 months previously; the 10 others were studied shortly before joining the haemodialysis programme. The patients not undergoing dialysis were receiving a diet containing 20 g protein, and those undergoing dialysis consumed 60 g protein daily. All patients experienced intermittent nausea and vomiting, although these symptoms were less severe in the four undergoing haemodialysis. Barium meal studies in all patients showed no evidence of peptic ulceration. No patient took any drug likely to influence gastric motility—for example, metoclopramide or cimetidine—within 72 hours of the study.

Eleven healthy volunteers (seven women) aged 22-69 (mean age 44) were also studied.

After a six hour fast each subject ate a meal consisting of 30 g instant porridge, 8 g sugar, and 150 ml warm milk, to which 11·1 MBq (300 μCi) technetium-99m tin colloid had been added.[2] Each study began at 1500, and the four patients undergoing haemodialysis were studied on the day before a dialysis session. After eating the meal the subjects lay supine beneath a gammacamera linked to an on line computer. Successive 90 second images obtained over a 90 minute period were stored on magnetic discs. The gastric images were then delineated, and the total counts within this area were calculated for each of the 60 images. A curve showing gastric emptying was produced after adjustment for radioactive decay. Counts were expressed as a percentage of the maximum count.

No control subject experienced nausea or vomiting during the study, and no patient with renal failure vomited, although most described mild nausea and abdominal fullness.

The patients with uraemia showed significantly greater retention of isotope in the stomach as judged by counts at 30 minutes ($p < 0.01$), 60 minutes, and 90 minutes ($p < 0.05$) (Mann-Whitney U test). Three of the four patients undergoing dialysis had normal gastric emptying patterns. When the four



Mean (SD) proportion of radioisotope label left in stomach in patients with uraemia not receiving dialysis (●——●) and control subjects (○— — —○).

patients undergoing dialysis were excluded and the remaining patients with uraemia compared with the controls a pronounced delay in gastric emptying was apparent (figure).

Comment

Delayed gastric emptying was shown in patients with symptomatic uraemia compared with asymptomatic controls. This abnormality may be confined to patients with uraemia not undergoing dialysis, and our observation of normal gastric emptying in three of four uraemic patients receiving dialysis confirms a previous report, which found no disturbance in gastric motility in 10 patients with renal failure undergoing haemodialysis.[3]

Possible explanations for the delay in gastric emptying include electrolyte disturbance, uraemic toxins, raised plasma concentrations of gastrointestinal hormones, and dysfunction of the autonomic nervous system. The influence of symptoms such as nausea on gastric function must also be considered as it is not clear whether delayed gastric emptying causes these symptoms or is a consequence of them.

Our finding may aid the interpretation of pharmacokinetic data in patients with uraemia and it provides a rationale for using drugs that promote gastric emptying in uraemic patients with nausea and vomiting.[4 5]

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Primary lymphoma of the anal canal presenting as perianal suppuration

Primary lymphoma of the gastrointestinal tract is well documented, but to our knowledge lymphoma arising from the anal canal has not been reported. We report on a patient with anal lymphoma presenting as perianal suppuration.

Case report

An 89 year old woman presented with a two week history of throbbing perianal pain and discharge. There were no other symptoms attributable to gastrointestinal disease, although benign gastric ulceration had been diagnosed six months previously at endoscopy. Digital examination of the rectum then had not shown any abnormality but now showed a perianal abscess to the left of the anal margin that had discharged, leaving a large ulcerated area. Proctoscopy showed ulceration within the anal canal, but sigmoidoscopy to 17 cm did not show a lesion within the rectum. After debridement a biopsy specimen of pale, firm tissue at the base of the abscess cavity was obtained. General examination did not elicit palpable lymphadenopathy, hepatomegaly, or splenomegaly. Full blood count and differential white cell count were normal, as were results of liver function tests. A chest *x* ray film was normal.

Histological examination of the biopsy specimen showed a diffusely infiltrating undifferentiated tumour composed of large cells with vesicular nuclei containing several small nucleoli and with scanty surrounding cytoplasm. Staining of paraffin sections with monoclonal antibodies to leucocyte common antigen, cytokeratin, and epithelial membrane antigen was carried out using an indirect immunoperoxidase technique.[1] The cells of the tumour expressed leucocyte common antigen but not the epithelial antigens cytokeratin or epithelial membrane antigen. The tumour was therefore diagnosed as a high grade malignant lymphoma and classified as a centroblastic lymphoma (Kiel classification) or diffuse histiocytic lymphoma (Rappaport classification).

Subsequent computed tomography of the abdomen and pelvis showed no enlargement of the lymph nodes, no masses, and a normal liver and spleen. Gastroscopy showed that the previous gastric ulcer had healed completely after treatment with cimetidine. After a course of radiotherapy, consisting of 4000 cGy (4000 rad) given in 20 fractions over four weeks, the lesion had resolved satisfactorily, and the patient was alive and well three months after treatment.

Comment

Lymphoma in the large bowel accounts for about 10% of all gastrointestinal lymphomas,[2 3] but although the rectum is the commonest site within the large bowel, no series has described primary anal disease. The patient in this report appears to have had anal lymphoma as the case satisfied criteria for primary gastrointestinal lymphoma.[4] Palpable lymphadenopathy and enlargement of lymph nodes were absent, total and differential white cell counts were normal, and although laparotomy was not performed, computed tomography did not detect further intra-abdominal disease.

The presentation of primary lymphoma as perianal suppuration is interesting in view of the recent description of lymphoma arising from lymphoid tissue associated with mucous membranes.[5] Lymphoid tissue in the anal canal is aggregated around the anal glands in the intersphincteric plane, and infection of these glands is thought to be crucial in the pathogenesis of perianal abscess. Thus blockage of the glands by neoplastic infiltration probably contributed to the clinical picture.

Initial histological diagnosis was complicated by the undifferentiated nature of this tumour, and only after immunohistochemical staining with monoclonal antibodies could lymphoma be diagnosed definitely. This highlights the importance of such studies in poorly differentiated neoplasia, and some anal tumours reported as anaplastic carcinoma on purely histological grounds might, in fact, be lymphomas. Clearly, it is important to distinguish between carcinoma and lymphoma, and we would urge the use of immunohistochemical studies when the histogenesis of any anal tumour is in doubt.

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Fatal immune haemolysis associated with nomifensine

We report a fatal haemolytic reaction after treatment with nomifensine.

Case report

A 36 year old woman was admitted to Scunthorpe General Hospital as an emergency in February 1985, having collapsed within an hour of taking one tablet of nomifensine (Merital, 100 mg). She had taken the drug previously for one week but had stopped 10 days before presentation because of dizzy spells; there was no evidence of jaundice or red urine. On admission she was conscious but uncommunicative, pale, cyanosed with shallow