Discussion

These findings show that ranitidine 150 mg at bedtime is superior to enprostil 35 μg at bedtime in preventing relapse of duodenal ulcer. The extreme differences in relapse rates were surprising because enprostil 35 μg has been claimed to have antisecretory effects comparable[10] or superior[11] to those of cimetidine 600 mg. Relapse rates in the ranitidine group were comparable with rates reported in other recent trials.[3 4] By contrast, the relapse rates in the enprostil group were of the same magnitude as those described after placebo[2] and similar to rates found in our population after treatment for duodenal ulcer healing.[20 21] Hence enprostil, which is also inferior to ranitidine for duodenal ulcer healing,[15 22] cannot be recommended for the prevention of ulcer relapse.

The dose of enprostil used in this study inhibits acid secretion, and the therapeutic benefit, if any, is explicable by this effect rather than by any mythical "cytoprotection" which may be achieved at lower doses.[14] These results and our previous findings in studies on duodenal ulcer healing[15] and duodenal ulcer haemorrhage[23] question the clinical relevance of promoting cytoprotective properties of prostaglandin analogues for duodenal ulcer treatment.

We thank Mrs Rigmor Petersen for secretarial help; the staff of our endoscopy units for valuable support; and Syntex Inc, Palo Alto, California, for the study drugs.

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(Accepted 27 February 1987)

SHORT REPORTS

Irritable bowel syndrome as a cause of chronic pain in women attending a gynaecology clinic

Chronic lower abdominal pain and pelvic pain is a common problem in gynaecological practice. Most of these women undergo diagnostic laparoscopy, which shows a normal pelvis in up to 90% of cases.[1] Despite the lack of a definite abnormality further gynaecological procedures are often undertaken, which may include surgery.

The irritable bowel syndrome is common and a recognised cause of chronic pain. It is associated with many non-colonic features, including dyspareunia,[2] and is more prevalent in young women.[3] A diagnosis of the irritable bowel syndrome is made on the history alone, and, although results of special investigations are by definition negative, the diagnosis should not simply be one of exclusion.[4]

The purpose of this study was to see whether patients attending a gynaecological clinic with chronic pain might be suffering from the irritable bowel syndrome by taking a detailed history using Manning's questionnaire, which has been shown to identify successfully such patients.[4]

Patients, methods, and results

Fifty patients with chronic abdominal pain who had been shown to have no pelvic disease at laparoscopy were studied at their follow up visit. The women were aged between 18 and 35 and had had symptoms for at least three months. Each patient was asked the questions listed in the table and then examined for the physical signs. The table shows the number of positive responses to each question.

At least 26 patients admitted to looser stools at the onset of pain, pain eased by defecation, distension, mucus per rectum, hard pellet stools, or dyspareunia. Similarly, 38 patients had a tender, palpable colon and 36 rectal tenderness per vagina. Thirty patients had at least three symptoms associated with the irritable bowel syndrome and these patients were very likely to have the disorder. A further eight patients had two symptoms of the irritable bowel syndrome. Only three patients had none of these symptoms.

Incidence in 50 patients of symptoms and signs commonly present in the irritable bowel syndrome

	No of patients
Looser stools at onset of pain	26
More frequent bowel movement at onset of pain	21
Pain eased after bowel movement (often)	32
Visible distension	31
Feeling of distension	42
Mucus per rectum	29
Feeling of incomplete emptying	15
Hard pellet stools	33
Dyspareunia	41
Rectal tenderness per vagina	36
Tender, palpable colon	38

Comment

The questionnaire has been shown to identify successfully those patients with the irritable bowel syndrome.[4] Dyspareunia is not one of Manning's symptoms but has been shown to be associated with the syndrome.[2] It is another manifestation of a tender colon. Similarly, a tender colon and rectal tenderness are associated with the syndrome.[5] The combination of chronic pain and three symptoms of the irritable bowel syndrome supported the diagnosis in 30 (60%) of the patients in this study. A further eight patients may have been suffering from the condition with two symptoms. As the irritable bowel syndrome may be present without an abnormal bowel habit[3] the diagnosis was not excluded in the other 12 patients. The presence of

bowel tenderness in 38 patients further supported the diagnosis of the irritable bowel syndrome.

It was not entirely unexpected that so many young women with chronic abdominal pain would have the irritable bowel syndrome, which affects up to one fifth of an apparently healthy population. Though equally prevalent in men and women, those who consult a doctor are mainly women.[3] Being women with pain they are commonly referred to a gynaecology clinic, where details of their bowel habit are not sought. Only a third of patients with the irritable bowel syndrome have colicky pain alone, the others having dull pain or both types of pain.[5] Hence the type of pain alone cannot identify those with the irritable bowel syndrome, and details of their bowel habit must be sought.

Manning *et al* have shown that a detailed history can help towards a confident diagnosis of the irritable bowel syndrome in patients with chronic abdominal pain and avoid unnecessary investigations. Our study suggests that the irritable bowel syndrome is a common cause of chronic pain in women referred to a gynaecologist and should be sought by a detailed history in each case.

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(Accepted 6 January 1987)

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Cockroach dermatitis: an occupational hazard

Cockroaches are common inhabitants of kitchens, dining rooms, and food stores in hospitals, hotels, and houses and are important mechanical vectors of infectious disease. Reactions to cockroaches themselves, however, have rarely been recorded. We describe a patient with a severe cutaneous reaction to cockroaches in a hospital record store.

Case report

A 51 year old woman employed as a medical records clerk at another hospital presented with an intensely itchy eruption on the face, neck, hands, and knees, which had developed after she had been clearing old hospital case notes from a derelict hut. She gave no history of atopy or of other skin disorder. The appearance was of urticated erythematous papules coalescing into plaques, and clinically suggested an insect bite reaction. The hut was inspected by a pest controller, who found no evidence of mites, bed bugs, or other arthropods, but when the patient resumed work in the area the eruption recurred. When the interior of the hut was subsequently dismantled copious insect debris was discovered; examination of this showed numerous fragments of the German cockroach *Blatella germanica*.

The patient has had no subsequent symptoms. Prick testing with cockroach mix allergenic extract (Dome/Hollister-Stier) produced a negative reaction at 15 minutes but a 1 cm itchy erythematous papule at two hours. The result of epicutaneous testing was negative, as was that of prick testing of 20 healthy controls.

Comment

Cockroaches are widely encountered in the human environment, and cockroach extracts may produce positive responses to prick tests in exposed people, especially those with a history of atopy.[1] Cockroach specific IgE may be found in the serum of atopic subjects with a history of exposure to cockroaches.[2] Clinical manifestations of cockroach sensitivity are, by contrast, rare. Asthma has occasionally been reported,[3,4] and cutaneous reactions manifested as contact dermatitis or contact urticaria have been reported in only four cases,[1,3,4] all in laboratory assistants with a prolonged history of exposure to cockroaches in their work.

Our case was unusual because a history of cockroach exposure was not apparent. Given the widespread exposure of man to cockroaches and the known capacity of their antigens to induce allergic sensitivity, possibly this type of reaction is less rare than the paucity of published accounts might suggest.

We thank Dr Nicholas Burgess of the Royal Army Medical College for examining specimens and for entomological advice.

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(Accepted 5 March 1987)

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Lower oesophageal contractility as an indicator of brain death in paralysed and mechanically ventilated patients with head injury

The use of neuromuscular blocking drugs in neurosurgical patients receiving mechanical ventilation presents special problems in assessing the level of consciousness and cerebral function. In addition, brain death cannot be diagnosed in a paralysed patient.

Measuring lower oesophageal contractility has been shown to be of value in assessing the depth of anaesthesia in anaesthetised and paralysed patients.[1] The physiological basis of the technique relies on the anatomy and innervation of the oesophagus. In man and the American opossum[2] the muscles of the lower half of the oesophagus are composed of smooth fibres and so are not directly affected by neuromuscular blocking drugs. Oesophageal activity is measured as peristaltic (provoked lower oesophageal contractility) or non-propulsive (spontaneous lower oesophageal activity).

We have assessed lower oesophageal contractility as a guide to outcome in patients after head injury requiring neuromuscular paralysis and mechanical hyperventilation.

Patients, methods, and results

Sixteen patients with head injury admitted to the surgical intensive care unit for controlled hyperventilation and neuromuscular paralysis were studied over six months. The Glasgow coma scale was assessed on admission. Lower oesophageal contractility was monitored by an Antec Lectron 301 (Antec Systems Ltd, Oxford). The device consists of a disposable oesophageal probe coupled to the monitoring unit. The probe has a distal saline filled pressure sensing balloon and an adjacent pneumatically inflatable balloon designed to provoke a response from the oesophagus. The probe was introduced through the mouth and positioned so that the tip was 35 cm from the lips. The "provoking" balloon was inflated for five seconds and then deflated, the cycle being repeated every three minutes. Spontaneous activity in the oesophagus was recorded as the number of spontaneous contractions per minute. Provoked activity was monitored as the peak pressure of any contraction occurring within 10 seconds of inflating the balloon. The monitor memorises values of spontaneous and provoked activity for at least 24 hours of continuous recording.

Neuromuscular blockade was achieved with pancuronium by continuous infusion. Sedation was given as indicated clinically. Monitoring was continuous for the first eight hours and thereafter carried out for two hour periods twice a day up to 48 hours. The Glasgow coma scale was assessed again before discharge of the patients from the unit.

Results are presented as mean values and standard deviation (SD).

Patients with spontaneous lower oesophageal contractility—Eleven patients had spontaneous activity recorded in the oesophagus. Their mean age was 31·8 years (range 16-52) and mean Glasgow coma score on admission 9·0 (1·8). The mean spontaneous activity was 1·2 (0·7) contractions/min and mean provoked activity 40 (18) mm Hg. Ten patients recovered and were discharged from the unit; the remaining patient died of the adult respiratory distress syndrome.

Patients with absent spontaneous lower oesophageal contractility—Five patients did not have spontaneous activity in the oesophagus at any time. Their mean age was 32·1 years (range 30-45) and mean Glasgow coma score on admission 3·6