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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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.

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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

(1·1). All five patients continued to exhibit provoked activity (mean 28·5 (9·6) mm Hg). In all these patients the diagnosis of brain stem death was subsequently established by specific neurological criteria.

Comment

Our most striking finding was that of the 16 patients monitored by the technique, those with no spontaneous lower oesophageal contractility at any time, but with provoked activity intact, were invariably subsequently diagnosed as brain dead clinically after the action of pancuronium had worn off or been reversed. In this institution brain death is diagnosed using the criteria set out by the royal colleges and faculties of the United Kingdom.[3] In addition, carotid arteriography is performed to show cessation of cerebral circulation. The loss of spontaneous contractility in the presence of continuing provoked activity is explained by the normal physiology of the human oesophagus. The principal nerve supply is derived from the vagus nerve. The lower oesophageal muscle is supplied from intramural and paraoesophageal nerve plexuses. Vagal afferent and efferent fibres connect the oesophageal plexuses with the brain stem nuclei of the vagus nerve, which in turn are connected with higher cortical centres. The mechanisms governing production of non-propulsive activity are unknown, though acoustic stimuli as well as other forms of external stimuli can provoke this response.[4,5] Thus it appears that an intact pathway between the brain and oesophagus is required for non-propulsive activity to occur. By contrast, secondary activity (peristaltic) has been recorded in the isolated opossum oesophagus ex vivo and so does not require control by higher centres.[2] Hence in brain dead patients spontaneous lower oesophageal contractility would not be expected, whereas provoked contractility should be intact, as we have found.

We conclude that monitoring lower oesphageal contractility may be very useful in the early identification of brain death and may help to predict outcome in patients with head injury.

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Effect of combined implants of oestradiol and testosterone on libido in postmenopausal women

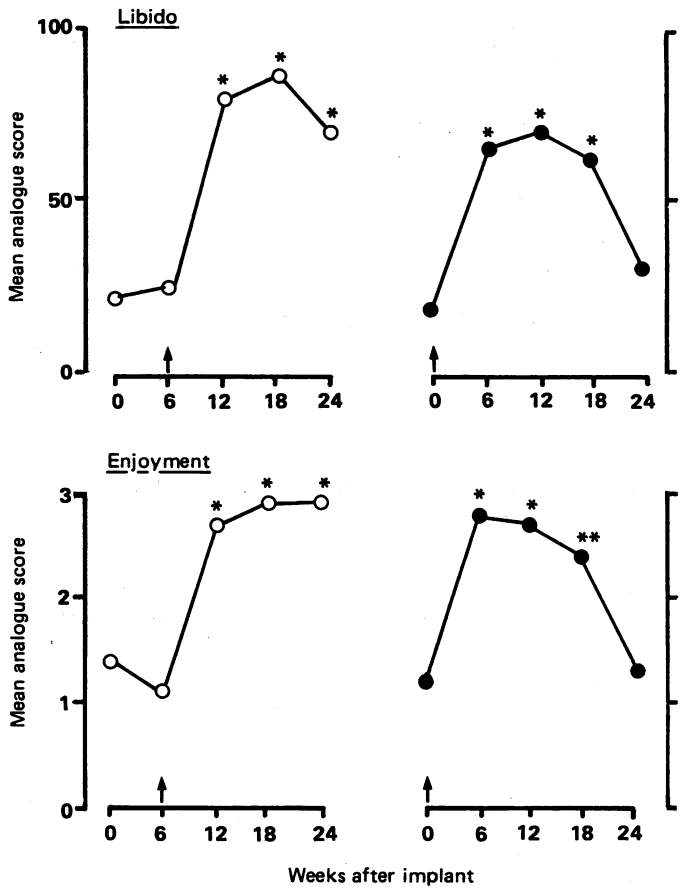
We have shown that a loss of libido in postmenopausal women can be cured with combined implants containing oestradiol 40 mg and testosterone 100 mg.[1] Dow *et al* reported that implants of oestradiol alone were as effective as combined implants.[2] We set out to discover whether additional testosterone was required by postmenopausal women whose lack of libido had persisted despite adequate oral oestrogen replacement. We also tried to discover whether a 50 mg dose of testosterone would be effective.

Patients, methods, and results

Twenty women were recruited from the menopause clinics at Prince Henry's and Royal Women's Hospitals, Melbourne. They had suffered from a severe loss of libido despite doses of oral oestrogens and progestogens that adequately relieved other main symptoms, such as hot flushes and vaginal dryness. After the initial clinical assessment patients were allocated randomly (by means of a table of random numbers) to one of two groups receiving different treatment: either a single implant of oestradiol 40 mg or a combined implant of oestradiol 40 mg and testosterone 50 mg (Organon). A single blind design was used. In the single implant group a failure to improve significantly after an initial six weeks' observation was taken as an indication for the addition of a testosterone implant 50 mg.

Current treatment with oral oestrogens was stopped, and after two weeks baseline blood samples were taken for hormonal and biochemical measurements, and the single or combined implant was inserted. The subsequent assessments were made at intervals of six weeks, and at each visit norethisterone 2·5 mg daily for 10 days was prescribed.

Self rating analogue scales were used to assess the severity of symptoms, with a score of 0 signifying the most severe form of the symptom and a score of 100 indicating freedom from the symptom.[1] Sexual enjoyment was assessed on a 0–3 rating scale. Plasma testosterone concentration was measured as described.[1] Differences within groups were analysed with Duncan's multiple range test and differences between groups compared by unpaired Student's *t* test.



Effects of initial single implant of oestradiol (○) and combined implant of testosterone and oestradiol (●) on libido and sexual enjoyment (analogue scales) over 24 weeks. Arrow indicates time of insertion of testosterone implant in group initially given oestradiol alone. *p<0·01, **p<0·01, Duncan's multiple range test.

The mean (SD) ages of the single and combined implant groups were 48·2 (5·2) and 43·5 (7·6) years, respectively; the mean number of years since menopause was 5·6 (3·9) and 7·8 (4·8), respectively. Nine of the combined implant group and all 10 in the single implant group had had hysterectomies, and three from each group had had oophorectomies. After six weeks the loss of libido in the single implant group remained, while the combined group showed significant symptomatic relief (p<0·01; figure). All subjects in the combined implant group were satisfied with their treatment after six weeks. Eight in the single implant group chose to have a testosterone implant at the first follow up visit at six weeks; the other two stopped coming because of dissatisfaction with the treatment. The testosterone implant resulted in rapid relief of symptoms in these eight subjects, persisting for up to 18 weeks (figure). The mean peak testosterone concentrations after testosterone implantation in both groups slightly exceeded the upper limit of the normal range (3 nmol/l), reaching 3·5 nmol/l in the combined implant group and 3·7 nmol/l in the group given testosterone at six weeks. There were no significant changes in the concentrations of cholesterol, its subfractions, or serum triglycerides in either group.