(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.

We thank Antec Systems Ltd and Roche Pharmaceuticals for their help and sponsorship during this study, and Mrs E Agrebi and Miss R Vincent for preparing the manuscript.

1 Evans JM, Davies WL, Wise CC. Lower oesophageal contractility: a new monitor of anaesthesia. *Lancet* 1984;ii:1151-4.
2 Christensen J, Lund GF. Esophageal response to distension and electrical stimulation. *J Clin Invest* 1969;48:408-19.
3 Royal Colleges and Faculties of the United Kingdom. Diagnosis of brain death. *Br Med J* 1976;ii:1187-8.
4 Faulkner WB. Objective esophageal changes due to psychic factors. *Am J Med Sci* 1940;200:796-803.
5 Stacher G, Schimierer G, Landcraf M. Tertiary esophageal contractions evoked by acoustical stimuli. *Gastroenterology* 1979;77:49-54.

(Accepted 19 January 1987)

Surgical Intensive Care Unit, University Hospital of Geneva, Geneva, Switzerland
M E SINCLAIR, MB, FFARCS, senior lecturer in anaesthesia
P M SUTER, MD, chief

Correspondence to: Dr M E Sinclair, Nuffield Department of Anaesthetics, John Radcliffe Hospital, Headington, Oxford OX3 9DU.

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

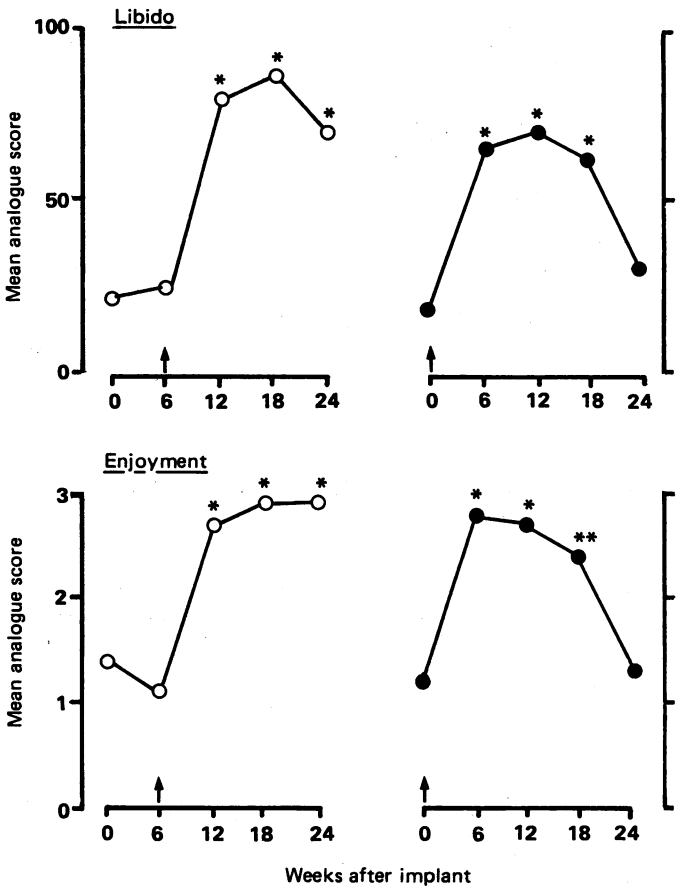
We have shown that a loss of libido in postmenopausal women can be cured with combined implants containing oestradiol 40 mg and testosterone 100 mg.[1] Dow *et al* reported that implants of oestradiol alone were as effective as combined implants.[2] We set out to discover whether additional testosterone 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.

Comment

Our data contrast with those of Dow *et al*[2] in that they suggest that added testosterone is beneficial in alleviating psychosexual symptoms. One reason for the difference may be the selection process: Dow *et al* studied an unselected group of patients attending their menopause clinic while we chose patients in whom oral oestradiol had failed to relieve psychosexual symptoms. Perhaps only in this group is testosterone specifically effective. No significant side effects were experienced.

We are grateful to Peter Dennis, Ken Outch, Nick Balazs, and Mohan Bangah for hormone and lipid assays, to Jill Volfsbergs for secretarial help, and to Robyn Redman for the artwork. Organon Australia kindly donated the implants.

1 Burger HG, Hailes J, Menelaus M, Nelson J, Hudson B, Balazs N. The management of persistent symptoms with oestradiol-testosterone implants: clinical, lipid and hormonal results. *Maturitas* 1984;6:351-8.
2 Dow MGT, Hart DM, Forrest CA. Hormonal treatments of sexual unresponsiveness in postmenopausal women. *Br J Obstet Gynaecol* 1983;90:361-6.

(Accepted 23 January 1987)

Prince Henry's Hospital, Melbourne, Australia 3004
HENRY BURGER, MD, FRACP, director, medical research centre and department of endocrinology
JEAN HAILES, MB, BS, clinical assistant, menopause clinic
JOY NELSON, MB, BS, clinical assistant, menopause clinic

Endocrine Unit, Royal Women's Hospital, Melbourne, Australia
MARGARET MENELAUS, MB, BS, clinical assistant

Correspondence to: Dr Burger.

Medical leeches as sources of wound infection

Leeches are used in plastic surgery to help relieve venous congestion after microsurgery: they ingest blood until satiated and then detach themselves. Owing to the anticoagulant properties of the animals' saliva the puncture wound continues to bleed. We report six cases in which use of leeches was followed by wound infections due to *Aeromonas hydrophila*.

Patients, methods, and results

We use about 100 leeches (an average of 10 per patient) each year. During the past three years six wound infections caused by *A hydrophila* developed in patients on whom leeches had been used; this represents an infection rate of 20%. The table summarises details of the six cases.

Comment

Whitlock *et al*[1] suggested that the medicinal leech (*Hirudo medicinalis*) was a potential source of infection when used in plastic and reconstructive surgery because it carries *A hydrophila* within its gut. The leech has no proteolytic gut enzymes and relies on the bacterium to digest blood. The organism has also been isolated from the anterior and posterior suckers of leeches and from the mucous trail and water in which they were kept.[1] *A hydrophila* has been implicated in three types of infection. It has been reported to be the causative organism in 2% of patients with diarrhoea,[2] and infections may occur after injuries sustained while swimming in contaminated water[3] and in immunocompromised patients.[4] The organism is occasionally carried in faeces.[2]

Although infection after the use of leeches has been reported only once previously,[5] our findings suggest that leeches may be an important cause of wound infections. These infections are generally characterised by the onset of inflammation and suppuration over 24 hours accompanied by moderate fever and leucocytosis. The proteolytic action of the bacterium may explain its effect on muscle; in case 5 a graft of muscle was destroyed by infection. The infection responded to antibiotic treatment in all but one case, in which it settled spontaneously before sensitivities were known. Surgical drainage and debridement were used to remove necrotic material.

Despite our findings leeches will remain useful in plastic and reconstructive surgery to treat venous congestion. When wound infection occurs after their application aeromonas should be looked for carefully. All but one of the strains isolated from our patients were resistant to penicillin and ampicillin; antibiotic sensitivity tests should always be performed. To treat an aeromonas infection an antibiotic resistant to β lactamases (for example, Augmentin) should be given orally or a cephalosporin (for example, cefuroxime or cephradine) intravenously.

Data on patients with A hydrophila wound infections

Case No	Date of operation	Indication	Surgical procedure	Time to positive swab (days)*	Clinical picture	Sensitivity to ampicillin/ penicillin	Treatment	Outcome
1	May 1983	Carcinoma of nasal septum	Forehead flap to upper lip	26†	Wound discharge	−	Cefuroxime	Settled
2	Aug 1983	Traumatic amputation of right thumb tip	Cross finger flap	2	Tenosynovitis	−	Augmentin	Settled
3	Sept 1984	Traumatic amputation of right hand	Microsurgical repair	11	Pin site, wound discharge	−	Debridement	Settled
4	Aug 1986	Midtarsal fracture of left foot	Instep flap to bare bone	4	Wound discharge and skin necrosis	−	Cefuroxime, debridement	Settled
5	Sept 1986	Cranioplasty	Microsurgical latissimus dorsi free tissue transfer	1	Infected haematoma	+	Amoxycillin, debridement	Settled
6	Nov 1986	Degloving injury of right hand	Microsurgical repair	7	Wound discharge and skin necrosis	−	Cefuroxime, debridement	Settled

*Time from first application of leeches to date of first positive swab result.
†Patient underwent staged procedures with leeches applied after each stage.

1 Whitlock MR, O'Hare PM, Sanders R, Morrow NC. The medicinal leech and its use in plastic surgery: a possible cause of infection. *Br J Plast Surg* 1983;36:240-4.
2 Millership SE, Curnow SR, Chattopadhyay B. Faecal carriage rate of Aeromonas hydrophila. *J Clin Pathol* 1983;36:920-3.
3 Rosenthal SG, Bernhardt HE, Phillips JA. Aeromonas hydrophila wound infection. *Plast Reconstr Surg* 1974;53:77-9.
4 Bulger RJ, Sherris JC. The clinical significance of Aeromonas hydrophila: a report of two cases. *Arch Intern Med* 1966;118:562-4.
5 Dickson WA, Boothman P, Hare K. An unusual source of hospital wound infection. *Br Med J* 1984;289:1727-8.

(Accepted 19 January 1987)

Department of Plastic and Jaw Surgery and Department of Medical Microbiology, Frenchay Hospital, Bristol BS16 1LE
N S G MERCER, FRCS, registrar in plastic surgery
DEBORAH M BEERE, MB, BS, senior registrar in medical microbiology
A J BORNEMISZA, FIMLS, senior medical laboratory scientific officer
P THOMAS, FIMLS, chief medical laboratory scientific officer

Correspondence to: Mr Mercer.

Primary myelodysplastic syndrome and cancer

Myelodysplastic syndrome is a preleukaemic clonal abnormality of haemopoietic stem cells.[1,2] We have looked at the occurrence of non-haematological malignancy in 138 patients with this condition, to find out whether or not they have an increased risk of developing other cancers either before or after myelodysplastic syndrome has been diagnosed.

Methods and results

Between October 1982 and May 1986 myelodysplastic syndrome (defined by conventional criteria[1,2]) was diagnosed in 138 patients. No patient with coexistent