natural resistance to a number of antibiotics including those routinely bactericidal for Gram-negative bacilli, such as ampicillin, third-generation cephalosporins and amino-glycosides. It is usually susceptible to trimethoprim–sulfamethoxazole, imipenem and rifampicin. Among cases of adult meningitis, five immunocompromised patients died and the four others were cured without ill effects. The seriousness of the disease is more from the terrain on which it occurs rather than the virulence of the germ. One patient without immunosuppression with post-encephalitic meningitis was cured with monotherapy poorly penetratable to cerebrospinal fluid (CSF) or poorly bacteriostatic (amikacin for 13 days, chenomycarin erythomycin for 25 days and the CSF cultures remaining positive for 25 days).

To our best knowledge, this is the first case report of an association between *F. meningosepticum* septicaemia and bacterial meningitis in a patient with AML. Our patient could have been an healthy carrier of *F. meningosepticum*, which became highly pathogenic because of persistent neutropenia. The high frequency of *F. meningosepticum* for early introduction of appropriate antibiotic therapy and the role of haematological remission in cure without sequelae.

### Case report

A 55-year-old man was working in his garden when he injured his left thumb with a lanced. He licked his finger to relieve pain. Five days later, he presented with left thumb nail bed haematoma, finger pain and fever. On examination he was pyrexial (37.8°C) and had a tender, swelling, left thumb with subcuticular abscess of the nail folds. X-Ray examination was normal. The nail was removed and amoxicillin/clavulanic acid (500/125 mg tid) was prescribed for 10 days. Two weeks later he remained febrile (37.5°C), and had left thumb pain and swelling. Radiologic examination revealed lytic bone defects on the terminal phalanges of the left thumb and soft-tissue swelling. The terminal phalanges was treated with curettage. The operative material showed an acute supplicative response and necrosis of tissue. Culture of the bone yielded, three days later, heavy growth of *E. corrodens*. The pathogen was susceptible in vitro to penicillin, ampicillin, and tetracycline and resistant to clindamycin, cephalosporins, chloramphenicol and gentamicin. Amoxicillin, 300 mg tid, was prescribed for 14 days. The thumb healed quickly and all symptoms promptly subsided.

As *E. corrodens* is an endogenous oral bacterium it is not surprising that the most common clinical sources of this organism are human bite wounds, head and neck infections and respiratory tract infections. Several clinical manifestations including soft-tissue infections or osteomyelitis caused by this organism, have also been described. Thus, most infections caused by *E. corrodens* involve areas contaminated by oral secretions. Probable, in our patient, the organism was inoculated when he licked his finger. It is a matter for speculation why amoxicillin/clavulanic acid failed to halt the development of osteomyelitis, in spite of the fact that the organism was susceptible in vitro to ampicillin. Presumably the daily dose and length of treatment were not sufficient to cure the infection, especially if osteomyelitis were already present when the patient was seen for the first time.

### Pressor effect of metoclopromide in phaeochromocytoma

**Sir**, Metoclopromide is a widely used anti-emetic in hospital practice. Apart from its well known anti-emetic action, it is generally well tolerated. It has a little known pressor effect in normal individuals but can produce a massive rise of blood pressure in phaeochromocytoma. We describe a patient with phaeochromocytoma given metoclopromide where, fortunately, the outcome was good.

### Case report

A 34-year-old woman, with recently diagnosed hypertension and a past history of neurofibromatosis Type 1, was referred with episodic dizziness, sweating, recurrent headaches and nausea. Her blood pressure was initially 208/134 mmHg. Control of blood pressure had been poor over the past few weeks, despite nifedipine and atenolol prescribed by her general practitioner. On admission to hospital her blood pressure was 150/90 mmHg. Multiple cafe au lait spots and axillary freckling were noted. She had had a right below-knee amputation some years previously for neurofibromatosis. Fundoscopy revealed grade 111 hypertensive changes. Physical examination was otherwise normal. Shortly after admission she complained of severe nausea and vomited once. She was given intramuscular metoclopromide (10 mg). Within minutes the patient became profoundly unwell, clammy, pale, and vomited again. Her blood pressure rose to 280/160 mmHg. Intravenous labetalol (50 mg) was infused over 20 minutes which relieved her symptoms and lowered the blood pressure to 160/120 mmHg. A presumptive diagnosis of phaeochromocytoma was made and the patient was given an alpha-blocker, doxazosin, in addition to the beta-blocker, atenolol. Nifedipine was discontinued on admission. Computed tomography (CT) scan of the abdomen revealed a 9 cm³ mass in the left adrenal gland. A 24-hour urine collection contained a 180 μmol of normal metadrenaline (normal < 4 μmol) and more than 70 μmol of metadrenaline (normal < 4 μmol). The patient underwent left adrenalecetomy and histological examination of the tumour confirmed the diagnosis of phaeochromocytoma.

Metoclopromide-induced hypertensive crisis was first described in 1976. Later this effect was found with phenoxybenzamine and clonidine. However, metoclopromide is a selective inhibitor of the adrenergic medulla which is blocked by both metoclopromide

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