Letters to the Editor

The role of *Candida albicans* in the pathogenesis of food-intolerant irritable bowel syndrome

Sir,

Middleton and colleagues¹ are to be congratulated in demonstrating that there is no conclusive link between overgrowth of intestinal *Candida albicans* and the symptomatology of irritable bowel syndrome (IBS).

Sadly, I suspect that the popular health magazines and alternative practitioners who persuade patients that these symptoms are directly linked to the pseudoscience of 'leaky bowels' and candida toxins² are unlikely to alter their views.

I have now seen a large number of patients with myalgic encephalomyelitis (postviral fatigue syndrome) who have a coexistent IBS. Almost without exception, a consultation with an alternative practitioner – some of whom are medically qualified – leads to the diagnosis of 'candida' and the recommendation of a highly restricted diet, probiotics, and antifungal drugs including nystatin and ketoconazole. Many general practitioners are perfectly willing to comply with patients' requests to prescribe these drugs despite the fact that no proven fungal infection has been identified.

As a result I have become increasingly concerned that this unjustified use of such medication for long periods of time (which some patients are now receiving) could result in the development of resistant strains of *Candida albicans* and other fungal infections. Armed with the evidence from Middleton's paper, I hope that doctors will now call a halt to prescribing potentially toxic drugs for an infection which does not appear to be involved in the pathology of IBS.

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Antifibrinolytic therapy for haemoptysis related to bronchial carcinoma

Sir,

Antifibrinolytic therapy has been used successfully in a number of conditions where bleeding may be associated with increased fibrinolysis. These include severe upper gastrointestinal haemorrhage,¹ bleeding associated with disseminated intravascular coagulation,² and bleeding after cardiac operations.³ However, the use of antifibrinolytic treatment in the management of haemoptysis associated with bronchial carcinoma does not seem to have been reported.

An 85 year old man was admitted in August 1990 with anginal pain not responsive to sublingual glyceryl trinit-

rate. Chest X-ray performed on this admission revealed a small left upper lobe opacity highly suggestive of bronchial carcinoma. Sputum cytology was negative for neoplastic cells. On follow-up the patient was free of chest symptoms, apart from occasional anginal attacks responsive to sublingual glyceryl trinitrate. In May 1991 he suffered painless rectal bleeding of dark red blood, barium enema revealed diverticular disease and bleeding settled on conservative management.

In late November 1991 he presented with a 2 month history of persistent haemoptysis with fresh or dark red blood, occurring daily mainly first thing in the morning, and varying from traces to half an eggcup in amount. The general practitioner had prescribed antibiotics on two occasions with no benefit. A diary confirmed haemoptysis was occurring every day and most recorded amounts were of 1–2 teaspoonfuls. Chest X-ray revealed that the left upper lobe opacity had grown to 6 cm diameter.

In view of the distress that the haemoptysis was causing it was decided to try the effect of antifibrinolytic therapy. Tranexamic acid (Cyclokapron) was given in a dose of 1 g three times a day by mouth, and the patient continued to keep a diary. After 2 days of therapy haemoptysis had cleared, and apart from spotting of blood on one day there has been no further haemoptysis over a period of one month, despite stopping therapy after 2 weeks of treatment.

It is therefore suggested that antifibrinolytic therapy may have a place in the management of persistent troublesome haemoptysis caused by bronchial carcinoma, at least as a temporising measure.

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Listeria meningitis – neurological and psychiatric sequelae

Sir,

Psychiatric syndromes associated with central nervous system listeriosis were recently reviewed by Duncan.¹ Included were reports of a manic depressive illness leading to suicide, three cases presenting with schizophrenic symptoms, and reports of disturbed behaviour

and brain damage in children.² Orland and Daghestani described a patient with catatonia occurring during listeria meningoenkephalitis,³ and Kellner *et al.* described a previously intelligent 68 year old man who, several months after recovering from meningoenkephalitis, complained of decreased intellect and short-term memory, difficulty in concentration and a generalized apathy, and lack of drive.⁴

An earlier review of 54 cases of central nervous system listeriosis showed that 20 cases developed focal neurological signs such as dysarthria, dysphasia, 6th and 7th cranial nerve palsies and nystagmus.⁵ We describe a patient with central nervous system listeriosis who developed several of the above psychiatric and neurological sequelae, as well as another complication which, to our knowledge, has not been previously reported – loss of the swallowing reflex.

An 83 year old previously well woman was admitted with a 2 day history of fever and increasing confusion. The initial diagnosis was of a pneumonia and she was given a 500 mg dose of intravenous amoxycillin after blood cultures had been taken. However, her condition rapidly deteriorated and re-examination showed that she had developed neck stiffness and a positive Kernig's sign as well as a temperature of 40°C. The cerebrospinal fluid was cloudy with white cells: 378 per µl (98% polymorphs); red cells: 945 per µl; protein 8 g/l; glucose: 3.8 mmol/l; microscopy showed Gram-positive rods.

The results suggested listeria meningitis and the patient was started on high dose intravenous amoxycillin (12 g/day) and gentamicin. Blood cultures subsequently grew *Listeria monocytogenes* (later typed 4b). Cerebrospinal fluid cultures showed no growth presumably because antibiotics had been administered before the lumbar puncture.

The pyrexia settled after 4 days of antibiotics, coinciding with a general improvement in her condition. However, she remained aphasic and was unable to

swallow safely and therefore nasogastric feeding was commenced. One month into her admission she began to speak and was able to swallow small amounts of semi-solid food. Over the next few weeks she fully regained her swallowing reflex and could eat normally, and at the same time her speech improved. Her main problem 3 months following admission is slow rehabilitation due to lack of drive and apathy, and she has poor short-term memory.

This case demonstrates several psychiatric and neurological sequelae of central nervous system listeriosis previously described: dysphasia, dysarthria, apathy, and loss of memory and drive. The patient also demonstrated loss of the swallowing reflex for several weeks following recovery from central nervous system listeriosis, and we believe that this should be included in the list of neurological sequelae of central nervous system listeriosis.

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