Letters to the Editor

Botulism

Sir,
Dr P.D. Welsby's condensed review of botulism¹ contained statements that could be misconstrued and do not reflect the experience gained from recent outbreaks.^{2–4} He describes the onset with oculo-bulbar symptoms as the usual presentation 'if they are not found dead', and suggests that gastroenteric symptoms are notable for their absence. Oculo-bulbar manifestations with a descending paralysis provide the most characteristic presentation. This is true of all types including type B, which is the commonest form of botulism in Europe and was the cause of the hazelnut yoghurt outbreak. The development in some patients of weakness in the lower limbs before that in the arms may help to differentiate the condition from Guillain-Barré syndrome (GBS). However, in approximately 40% of patients more protean parasympathetic symptoms such as blurring of vision, dizziness and hypotension preceded the development of muscular weakness.⁵ One-third developed abdominal cramps, vomiting, diarrhoea or constipation. Gastro-intestinal manifestations are a potent source of danger and were the cause of death in the one patient who died in that outbreak. She succumbed to an acid pneumonia following sudden gastric dilatation with regurgitation in the presence of oropharyngeal paralysis. Another patient developed gastric dilatation as his first symptom and was unable to do up his trousers. If the airway is protected and ventilation maintained the mortality from type B botulism should not approach 10%. There was no evidence, even retrospectively, of unexplained deaths that might have been attributed to botulism. However, with types A and E a more rapid progression would have been expected, with the risk of a higher mortality.

The presence of fever² or drowsiness^{2,3} does not exclude botulism. Many patients and their doctors interpreted a red, dry throat as tonsillitis. In the early stages a raised protein or cell count in the cerebrospinal fluid may also be absent in GBS. A false-positive edrophonium test can certainly be obtained in botulism.^{5,6} Positive electromyographic tests showing post-tetanic facilitation can be diagnostic but may be difficult to obtain in the most severely affected.⁷

Antitoxin therapy is not of proven value except in type E intoxication, and carries the added danger of an anaphylactoid reaction from horse serum. If used, it should be given early and should precede the early use of guanidine or 3,4-aminopyridine, which may enhance the uptake of toxin at the neuromuscular junction. Some patients, including children, reported a subjective improvement after its use at a relatively late stage.² Antibiotics are best reserved for secondary complications except in the presence of infantile or other toxico-infective forms of botulism.

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Gynaecomastia after long-term administration of domperidone

Sir,
Domperidone, a dopamine antagonist that does not freely cross the blood–brain barrier, has found wide use as an anti-emetic in cancer chemotherapy and as a useful promotility agent in patients with upper gastrointestinal symptoms. The peripheral effects of the drug include an increase in the tone of the gastro-oesophageal sphincter and an increase in the rate of gastric emptying.¹

Adverse reactions to domperidone are uncommon. However, those that have been reported are consistent with its action as a dopamine antagonist. An oculogyric crisis has been reported in an infant given the drug, and one case of gynaecomastia and galactorrhoea in a male infant has been recorded.^{2,3}

The anterior pituitary lies outside the blood barrier, and in common with other dopamine antagonists domperidone causes a rise in serum prolactin. There have been no previous reports of clinical adverse effects of dopamine-induced hyperprolactinaemia in adults. We report the first case of gynaecomastia in an adult after long-term administration of domperidone.

The patient, a 63 year old man, presented with a 3-month history of tender left gynaecomastia. There was no history of trauma or alcohol abuse. Twenty years previously he underwent a Pólya gastrectomy for a benign gastric ulcer. One year previously he had a gastroscopy for symptoms of nausea and vomiting and had been started on domperidone 20 mg 3 times a day with symptomatic improvement. His liver function tests, serum gonadotrophins and human chorionic gonadotrophin estimations were all normal. His serum prolactin was raised on 2 occasions one month apart at 894 $\mu\text{mol/l}$ and 609 $\mu\text{mol/l}$ (NR 50–360 $\mu\text{mol/l}$). X-rays of his pituitary fossa were normal. After withdrawal of the drug his prolactin fell to 88 $\mu\text{mol/l}$ with a resolution of his gynaecomastia. We recommend caution in the long-term