Drop attacks in the elderly: effect of pyridostigmine

Sir,

Elderly people are prone to sudden unexplained falls, usually categorized as 'drop attacks', a definition excluding those in whom the falls are accompanied or preceded by changes in the level of consciousness or by vertigo.^{1,2} The event is unpredictable and hence potentially dangerous, and constitutes the major type of home accident amongst people over the age of 65.³ The basic abnormality to which the falls may be attributable has not so far been clarified, but is not usually considered to be connected with the frequent cardiac or cerebral vasculopathy common in the age group.²

An approach to the question of the underlying pathophysiology of the disorder was put forward by Weiner *et al.*⁴ who found postural reflex impairment in 44% (severe) and 24% (moderate) of 34 such patients. The possibility of the immediate cause of the fall being a failure to generate tension in the quadriceps muscles sufficiently quickly to maintain erect posture has also been mooted.⁵ This concept of a causal momentary failure in the peripheral neuromuscular circuit is somewhat reminiscent of the familiar clinical sign in spastic paraparesis characterized by sudden collapse of muscle resistance to passive stretching.

Preventive treatment has so far been limited to various means of special attention to daily care of susceptible individuals^{3,6} with medication limited to that required for coexistent disorders (success has recently been reported⁷ following treatment of hypothyroidism). The concept referred to above of impaired maintenance of quadriceps tension, prompted a trial of the anti-cholinesterase pyridostigmine, as a possible means of somehow aiding maintenance of the neuromuscular contraction mechanism.

This agent has been prescribed in six individuals (three men, three women) aged 65–73 with typical drop attacks as defined, occurring several times a month. Two patients suffered from moderate hypertension; at the onset of therapy none had any abnormal neurological findings, but one female followed for 2 years subsequently developed signs and symptoms compatible with multiple system atrophy. In this patient, the two other females and one male, drop attacks ceased with onset of therapy. The male patient agreed to suspend medication in advance of electromyography studies for possible myasthenia gravis (which were negative); attacks returned 2 weeks later and ceased once more on resumption of treatment. Two females refused to stop therapy. All patients were started and kept on a dosage of 60 mg pyridostigmine twice daily and none of them reported any undesirable side effects on this regime. In those responding favourably, benefit resulted from the onset of treatment and was subsequently maintained, the longest period being 4 years to date.

This limited series may not justify conclusions at present, but I believe the drug to be worth trying in patients whose falls cannot be attributed to any clear aetiological causation.

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Splenic abscess due to *Eikenella corrodens*

Sir,

Eikenella corrodens is a slow-growing, aerobic and facultatively anaerobic Gram-negative rod, which is a normal inhabitant of the human oral cavity, and upper respiratory and gastrointestinal tracts.¹ The diseases associated with this organism range from periodontitis and human wound infections to more serious infections.² This report describes our experience of a splenic abscess caused by *E. corrodens*.

A 40 year old man with history of antrectomy with Billroth II anastomosis was admitted because of fever, chills and left-sided lower thoracic pain for 5 days. The patient was febrile. His breath sound on auscultation at the right base was diminished and there was a tenderness on deep pressure in the left costovertebral area. Laboratory data revealed a leucocyte count of $12.6 \times 10^9/l$ with 77% neutrophils. Chest, X-ray showed elevation of the left hemidiaphragm and a left-sided pleural effusion. Antibiotic therapy with erythromycin was administered. After 6 days of treatment, ultrasonographic examination of the abdomen revealed an enlarged spleen and perisplenic fluid collection, which was confirmed with computed tomography as a subcapsular spleen abscess. After that, the patient was treated with radiologically guided percutaneous drainage and clindamycin (2.4 g/day) plus gentamicin (240 mg/day), and he became afebrile in 24 hours. Seventy-two hours later, the antibiotic therapy was switched to ampicillin (12 g/day) because the culture of purulent material yielded *E. corrodens*. The results of blood cultures remained negative. The therapy was prolonged for 10 days and amoxycillin orally was continued until 25 days of therapy were completed.

Splenic abscesses are unusual; the incidence is between 0.2–0.7% in a population-based autopsy study.³ The most common organisms are Gram-positive cocci, anaerobes, aerobic Gram-negative rods and *Candida* spp.³ To our knowledge *E. corrodens* as a causative agent of splenic abscess has been reported only once before.⁴ In the case presented here, the microorganism might have reached the spleen by either haematogenous dissemination from oral cavity some weeks or months after the surgery into the subcapsular blood collection caused by operation, or spread from stomach to traumatic haematoma via the splenic vein or direct extension in the surgery.