As well as this central finding, the results of our study provide further support for a role of muscle afferents in proprioception.[4] Controls were significantly less accurate in reproducing knee joint position when initial test movements differed from subsequent target seeking movements. This has possible implications for the common physiotherapeutic practice of re-educating active movement through the medium of passive movement.

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Worsening of myasthenia gravis on treatment with imipenem/cilastatin

Myasthenia gravis may be exacerbated by a number of antibiotics which impair neuromuscular transmission, including the aminoglycosides, tetracyclines, and the polypeptide group.[1] Several reports have suggested that ampicillin and erythromycin may have similar effects.[2 3]

We describe here the worsening of myasthenia gravis on treatment with imipenem/cilastatin. Imipenem is a member of a new class of β lactam antibacterial drugs: the carbapenens. It is combined with cilastatin, a renal dehydropeptidase inhibitor, to inhibit renal degradation and has a wide spectrum of activity against Gram positive, Gram negative, and anaerobic bacteria, and against many multiresistant strains of bacteria.

CASE REPORT

A 45-year-old man presented with a three week history of progressive diplopia, facial weakness, and respiratory difficulties. Myasthenia gravis with malignant thymoma had been diagnosed 10 years previously; thymectomy was performed at that time and histology showed a mixed lymphocytic/epithelial cell tumour. He had a left upper lobectomy for pleural based metastases three years later. For the next seven years he remained well, requiring only 120 mg of pyridostigmine bromide daily.

At presentation mechanical ventilation was necessary. He was treated with azathioprine, steroids were gradually added to a dose of 60 mg prednisolone daily, and he received a course of plasma exchange.

Two months after admission he was extubated. At this stage he was receiving 150 mg pyridostigmine every three hours, azathioprine 150 mg each day, and prednisolone 60 mg on alternate days. Plasma exchange was continued with an exchange of two litres on a fortnightly basis. Six weeks after extubation a catheter was inserted into the right subclavian vein for easy venous access. He improved slowly. His forced vital capacity (FVC) increased from 0·3 l on admission to 2·4 l with moderate fatigue of the ocular and bulbar muscles. He subsequently developed superficial cellulitis at the site of the catheter. *Staphylococcus aureus* was grown and he was treated with flucloxacillin 500 mg intravenously three times a day. *Serratia marcescens* was grown on a routine sputum specimen taken two weeks previously and thus imipenem/cilastitin 500 mg intravenously four times a day was added.

His myasthenia deteriorated dramatically over the next 48 hours. His FVC decreased to 1·8 l with diplopia, marked bilateral facial weakness, and severe bulbar weakness. He also noted increased weakness in his arms and proximal leg muscles and had difficulty in walking. Serum calcium, urea, and electrolytes were normal. He responded positively to edrophonium 10 mg intravenously, with an improvement of the ocular bulbar and limb weakness. His FVC increased to 2·3 l. The imipenem/cilastatin was discontinued and the other drugs were left unchanged. Plasma exchange was not repeated at this stage due to the presence of infection; despite this, he improved over the next 24 hours and after 48 hours was back to his baseline state. The cellulitis resolved after six days and the flucloxacillin was discontinued after two weeks.

Various antibiotics may interfere with neuromuscular transmission and the mechanisms of action may include: a presynaptic effect leading to impaired release of acetylcholine, a post-synaptic curare-like blockade of the acetylcholine receptor, or a combination of the two mechanisms. The aminoglyclosides act pre-synaptically and post-synaptically, while the tetracyclines have a curare-like action.[1] The mechanisms of action of ampicillin, erythromycin, ciprofloxacin, and the polypeptides are unclear.[2-4]

A discussion with staff at Merck, Sharp and Dohme showed that one case had been reported to their adverse event database of myasthenia gravis developing in a patient receiving imipenem/cilastatin. To our knowledge there has been no published case. Our patient developed a worsening of his myasthenia while receiving this drug. His concurrent cellulitis may have been an additive factor, but this persisted long after the patient returned his to baseline state.

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Acceptability of electroconvulsive therapy to patients with Parkinson's disease

Some patients with Parkinson's disease have been shown to respond to electroconvulsive therapy (ECT).[1-3] It is not currently offered as a treatment for Parkinson's disease in the UK as clinicians do not consider it acceptable because of the stigma surrounding what may be seen as a psychiatric treatment and a fear of ECT by the patients based on a lack of information. This is surprising as non-drug treatments such as fetal tissue transplantation are more invasive and carry a higher risk than ECT. A consultant neurologist, while canvassing the opinions of patients attending a movement disorder clinic to assess the feasibility of a trial, found that patients were not keen to consider ECT as a treatment.

We decided to investigate this further with a larger group of patients in a standard manner using a questionnaire. Patients were asked a series of questions, including whether they would give consent to ECT if it were of definite benefit and, if so, would they agree to ECT as they were at that time or only if their disease became worse. Their opinion on consent to stereotactic transplantation was also sought.

All respondents were current patients selected from the Parkinson's disease register based at the department of neurology at the Institute of Psychiatry. Sixty-five questionnaires were distributed, of which 50 were completed. Twenty-six of these were administered to patients waiting in the outpatient department and 24 were sent out and returned by patients through the post. The mean age of the patients completing the questionnaire was 66·3 years and the mean duration of illness was 7·9 years. Twenty-eight of the respondents were men and 22 women. Fifty per cent of respondents had no other coexisting medical disorder. Twenty-eight per cent of respondents said they would consider ECT as a treatment at their present stage of illness if it was definitely known that ECT was of benefit in Parkinson's disease. A further 32% said they would only consider ECT if their illness became worse, and 36% said they would never consider it.

When asked about stereotactic transplantation, 16% of respondents said they would consider it at their present stage of illness and 28% only if their illness became worse. Fifty-two per cent said they would not consider it under any circumstances.

We conclude that patients with Parkinson's disease are more likely to accept ECT as a potential treatment than stereotactic transplantation. Had the patients been counselled or given further information about ECT, the proportion of respondents in this study agreeing to have it may have been greater. We suggest, in the light of these findings, that although ECT is not yet of proved value, it could potentially be a safe and effective option in Parkinson's disease, and that it is acceptable to a sizeable minority of patients. Those patients for whom drug treatment is proving unsatisfactory or in whom depressive features are prominent may benefit the most from ECT, but there is still a need for a definitive trial and our findings will have a bearing on the design of such a trial.

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