Letters

Stabilisation of a severe case of P.O.E.M.S. syndrome after tamoxifen administration.

Sir: In the absence of consistently effective treatment for plasma cell dyscrasia with polyneuropathy, organomegaly, endocrinopathy, M protein, and skin changes (P.O.E.M.S. syndrome), it seemed of interest to report a possible response to tamoxifen therapy.

A 56 year old man had been treated since 1982 for a sensorimotor neuropathy with oedema of the lower limbs. Since 1980 he had suffered from impotence, testicular atrophy, hypertrichosis and gynaecomastia. Nerve biopsy showed moderate decrease of myelinated fibres, without specific uptake in immunofluorescence and with no amyloid deposits. A monoclonal lambda IgG dysglobulinaemia (17 g/l, IgA 2.5 g/l, IgM 0.15 g/l) appeared as a complication after 6 months. Vertebra D12 was radio-opaque, and the diagnosis of plasmacytoma was confirmed by guided biopsy (the specimen showed preferential, especially lambda, IgG uptake in plasma cells). Iliac bone biopsy showed moderate plasmacytic infiltration. The C.S.F. protein level was 3 g/l, with an immunoelectrophoretic profile comparable to that of serum (IgG = 0.40 g/l).

Different treatments were undertaken: radiotherapy of vertebra D12 (3,300 rads) in 1982; sequential chemotherapy associating melphalan and prednisone (3 treatments) or melphalan, lomustine, cyclophosphamide and prednisolone (6 treatments); and combinations of vincristine, doxorubicin and dexamethasone according to the protocol of advanced multiple myeloma refractory to alkylating agents.¹ This last treatment resulted in medullary aplasia with septicæmia. All these treatments were ineffective. Weakness worsened in the lower limbs and spread to the upper limbs, so that in 2 years' time the patient was bedridden with intense pain refractory to symptomatic therapy. Generalised melanoderma developed, followed by pleural and peritoneal effusions and pericarditis with tamponade. Throughout this period the electrophoretic peak remained stable.

Out of desperation, after having read an article by Narasimhan² on the use of tamoxifen in the treatment of refractory lymphoma, we undertook this therapy (10 mg twice a day) in February 1985. Within 3 months, clinical improvement was spectacular, with disappearance of the oedema

and serous effusions and with partial and gradual regression of neurological signs (with resumption of physical activities, particularly walking, and disappearance of pain). There was moderate improvement in nerve conduction velocities. Two and a half years later, the general state of the patient is good, and his biochemical values remain stable.

In a study of 102 Japanese cases of P.O.E.M.S. syndrome,³ T. Nakanishi noted the possibility of improvement of neurological and other symptoms in a minority of patients by administration of prednisone and cyclophosphamide alone or in association. However, the majority of patients died (often of heart failure) after a mean survival period of 33 months. Several cases of improvement after irradiation of a solitary plasmacytoma have also been reported.⁴

The improvement in our patient after tamoxifen administration, when all other treatments had failed, suggests that this agent may be another therapeutic arm to combat the rapidly developing P.O.E.M.S. syndrome. Tamoxifen is ordinarily used as an antioestrogen in certain breast cancers and has also demonstrated its efficacy in a few cases of malignant lymphopathies.² Its action seems to stimulate the activity of natural-killer lymphocytes. Might there be a relationship with the gonadotrophic endocrine component of the P.O.E.M.S. syndrome, which has been particularly studied by Bardwick *et al*?⁵

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Unilateral essential tremor after wrist immobilisation: a case report

A 62 year old right handed decorator fell down stairs and fractured his wrist. This was treated by immobilisation in a scaphoid plaster from thumb to elbow for 6 weeks. When the plaster was removed the subject immediately noticed a tremor in his right hand which was intermittent and posturally dependant. With the wrist in a neutral or flexed position no tremor was present, but when the wrist was extended a marked tremor arose.

Prior to the accident he had been aware of some tremor in the outstretched fingers but it was so small that it did not interfere with living and was too slight for him to seek advice. There was no family history of tremor, and no problem with the left hand.

On examination the only abnormality was in the right arm. The right hand was normal in flexion and the neutral position. As soon as a posture involving extension was assumed a severe flexion-extension tremor appeared at the wrist. Writing could also provoke it. The frequency of the tremor was 7Hz, and it was absent during other postures of the wrist. The left hand was tremor-free. There was no dystonia, no Parkinsonian features and no loss in dexterity in the fingers.

In the subsequent 4 years the tremor has not spread to the opposite arm nor worsened in the right arm. It has not been improved by mysoline.

The clinical diagnosis was of an essential tremor, revealed clinically by the injury or more likely by the immobilisation. The history of some finger unsteadiness previously might suggest that the effect of the injury and its treatment was to raise a subclinical tremor to the clinical level. Its exquisite dependence on wrist posture might suggest that a peripheral trigger, possibly from an unbalancing of muscular afferents was important, although a central origin related to motor command cannot be excluded.

Dystonia have been described following peripheral injury,¹ and there is a case of an intention tremor following trauma but this was in association with clinical evidence of a cerebellar lesion.² The present subject's clear relation between injury and tremor appears to be a novel observation and begs the