followed by administration of prednisolone (50 mg/day) and melphalan (6 mg for 10 days) each month with monthly intravenous injections of cyclophosphamide (300 mg). Thereafter, the patient showed gradual improvement in motor, respiratory, and renal functions (figure 2, table). Four months after treatment, the tissue pressure of the quadriceps femoris in the supine position fell to 47 mm Hg. Serum IgA concentrations were consistently less than 200 mg/dl. There were no serious side effects of DMSO and an unpleasant breath odour was the patient’s main concern. Nine months after treatment, we noted a levelling off or a slight decline in some variables. Sixteen months after treatment, the patient aspired his secretions and died.

AL-amyloidosis results from conversion by proteolysis of mononclonal light chains into reactive β-fibrils, which can be recognised by Congo red staining. Light chain deposition disease is another pathological state associated with plasma cell dyscrasia.1 In our patient IgA λ plasmacytoma and skeletal muscle pseudohypertrophy, simultaneous deposition of AL-amyloid and λ light chains2 were shown by amyloid staining, immunohistoch- emistry, and electron microscopy. Involvement of shoulder muscle and possible factor causing motor impairment in our patient was a decreased range of motion, predominantly affecting proximal joints. Involvement of shoulder joints showing shoulder pad sign3 is pathognomonic of AL-amyloidosis.4 A factor hampering mobility is increased muscle tissue pressure reflected as wooden firm- ness. Increased muscle pressure is not produced by amyloid infiltration alone but may be related to deposition of amyloid, 4-sulfuric acid and silicon in muscles,5 tense muscle fascia secondary to amyloid deposition, or impaired tissue perfusion by amyloid angiopathy. The pressure is further increased by the activity to the point that it interferes with muscle blood flow. The goal of treatment in amyloidosis is to prevent further deposition of amyloid and to promote its resorption. In our patient, plasmapheresis and DMSO treatment resulted in an appreciable level of improvement in motor, respiratory, and renal func- tions. The ability of DMSO to make amyloid fibrils soluble for digestion has been demonstrated.6 Amyloid/ light chain-derived materials dislodged from various organs are likely to impair renal function. Therefore, to remove these breakdown products and the precursor mononclonal immunoglobulins, plasmapheresis was combined with DMSO. Because of the grave prognosis and disabling symptoms of amyloid associated muscle pseudohypertrophy, the trial of plasmapheresis and DMSO may be warranted even though the improvement may be moderate and of limited duration.

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Opossums, a rare complication of cocaine misuse

Opossums is a rare eye movement disor- der, mostly seen in postural ecephalopathy or occult neuroblastoma in children, or as a paraneoplastic phenomenon in adults. It rarely occurs after giving drugs or toxins. A single report of opossumus-myoelnic syndrome in association with cocaine use has been described in this journal.1 We pres- ent a patient with opossumus, myeloclonus, and ataxia.

A 29 year old man was admitted to hospi- tal with vertigo, nausea, and vomiting. He was unable to stand and walk, because his legs were shaking. The first symptoms had occurred after taking heroin until a few weeks oculomotor examination showed only sporadic horizontal ocular myoclonus in vertical movements. Follow up four months after his admission to the hospital yielded no ocular or horizontal myoclonus and the ataxia and the patient stated that he felt per- fectly well.

In our patient opossumus was very likely associated with taking cocaine. After exten- sive diagnostic evaluation no other complication could be found. The disorder appeared after incidental misuse of cocaine and was self limiting. One other such patient was described.

Various neurological complications of cocaine are known. Neurovascular disor- ders, either haemorrhagic or ischaemic, can occur after taking the drug.2 Seizures and migraine are other neurological complica- tions. Interestingly, increases in brain sero- tonin by inhibition of its uptake is an effect of cocaine. Maybe our patient, who had migraine, was more sensitive to the effect of cocaine, as severe serotonin dysfunc- tion has been reported in patients with migraine. The lack of any anatomical substrate supports this. On the other hand, a direct toxic effect of cocaine or on orofacial sympathethic sub- stances cannot be ruled out.

Further evidence of increased risk of mortality from Parkinson’s disease

It is often considered that since the intro- duction of levodopa treatment, there has been little difference in mortality from Parkinson’s disease compared with the general population.1 However, to date, only two or three studies have investigated the mortality in a group of patients with Parkinson’s disease compared with a matched control group. Rajput et al, in their review of case notes, found a mortality for patients with Parkinson’s disease 1.6 times that of controls,2 and the community based prospective case-control survey of Ebenezer et al in Aberdeen found a 2.35-fold higher death rate.3 In a recent issue of this Journal, Ben- Slomo and Marmot published the results of a long term community based prospective survey showing a 2.6-fold increased risk of mortality for Parkinson’s disease.4 We report the results of a prospective population based survey of subjects aged 65 and over that pro- vides further evidence of increased mortality due to Parkinson’s disease.

The population studied was a representa- tive, randomly selected sample of 2792 sub- jects, aged 65 and over, living at home in Gironde, France (PAQUID study), com- posed of 1122 men (40-2%) and 1670

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