to emphasise that bladder problems may be an early feature of cauda equina compression.

We thank Mr CG West and Mr T Farrington who were also involved in the treatment of this patient. We also thank Mrs T Pearce for the typing of this letter.

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Facial nucleus involvement in post-paralytic hemifacial spasm?

Sir: We report two unusual cases in which post-paralytic hemifacial spasm followed the injury of a very peripheral branch of the facial nerve. Such cases are unlikely to be explained by the peripheral hypotheses of the pathogenesis of post-paralytic hemifacial spasm. A 65-year-old man developed twitches in his right face 12 months after he accidentally cut himself near the right jaw with sharp sheers (Fig. 1A). The second patient, a 52-year-old man, complained of twitches around his right eye spreading over three weeks to the whole face; 10 months previously, his face had been accidentally cut by sharp glass near the right eye (Fig. 1B). In both cases the wound was clean without a haematoma, and there was no loss of consciousness. There was a slight weakness of the right orbicularis oris in the first patient, and of the orbicularis oculi in the second. A mild contracture at rest of the whole right hemiface was noticed in both cases, as well as a paradoxical co-activation of the mimic muscles on blinking or voluntary activation of the frontalis, orbicularis oculi and orbicularis oris. No other neurological signs emerged during a 5-years follow-up.

Electrophysiological examination of the right frontalis, orbicularis oculi and orbicularis oris muscles showed a mild reduction of MUPs in the orbicularis oris in the first patient and in the orbicularis oculi in the second; a subcontinuous activity of low amplitude MUPs at rest and the co-activation of all the muscles recorded during their separate voluntary activation were also noticed in both patients. These findings were consistent with post-paralytic hemifacial spasm.

Lamy1 first proposed the theory of misdirection of regenerating fibres to explain the abnormal associated movements in the facial muscles after incomplete recovery from facial nerve palsy. Another suggested mechanism was the formation of an "artificial synapse", or ephapse, at the site of the nerve injury when transaxonal excitation would give rise to a reverberating short-circuit to provoke mass muscle contraction and twitches.2,3 These hypotheses can explain cases of spasms occurring in a localised sector of the face following a lesion of a peripheral branch of the facial nerve.4 However, both the theories are insufficient, in our opinion, to explain the phenomena we observed. Regeneration of a single peripheral branch is unlikely to be so widespread as to reach all the muscles of the face. The formation of an ephapse at the site of the injury does not explain the spasm of the whole face. The occurrence of associated movements of the mimic muscles of the whole face after injury of single
branches of the facial nerve has been reported before. Such observations led Wartenberg to suggest that post-paralytic hemifacial spasm arose in the facial nerve muscles following peripheral as well as nuclear lesions of the seventh nerve. Ferguson postulated that functional reorganisation in the facial nucleus might occur following a partial deafferentation of the motor neurons after lesions of the nerve. This rearrangement, enhancing nuclear excitability might cause the development of hemifacial spasm. Indeed, an increased nuclear excitability has been found electrophysiologically in patients suffering from postparalytic contracture and mass movements. In our opinion, post-paralytic hemifacial spasm is likely to be due to central, not peripheral, mechanisms.

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The depression of myotonic dystrophy: response to imipramine.

Sir: Myotonic dystrophy is the most common of the inherited neuromuscular disorders, affecting up to 13.5 per cent 100,000 population. The neuromuscular symptoms which give the disorder its name—myotonia (delayed muscle relaxation associated with repetitive electrical depolarisation after a single contraction) and progressive distal weakness—can vary from incapacitating to undetectable. Other symptoms such as abnormal oesophageal and gastrointestinal motility, defective cardiac contraction and conduction, dysfunctional uterine contractions, cataracts, and extrathyroidal hypometabolism may not be present in all patients and may vary greatly in severity. Our recent study of myotonic dystrophy patients demonstrated that these patients uniformly have a major depressive disorder as defined by the American Psychiatric Association DSM-III criteria. We have now studied a group of myotonic dystrophy patients to determine the response of the depression to tricyclic antidepressant therapy.

A group of 16 consecutive myotonic dystrophy patients evaluated at the Red River Valley Chapter Muscular Dystrophy Association Clinic, Fargo, North Dakota were included in this study. The patient group consisted of six women and 10 men ranging in age from 21 to 49 years (mean age 37.5 years). At the time of the pre-treatment evaluation, a separate semi-structured psychological interview (including scoring of the Hamilton Rating Scale for Depression) was conducted. The tricyclic antidepressant imipramine was then prescribed in an initial dose of 25 mg at bedtime. The imipramine dosage was rapidly increased to 100-150 mg at bedtime, in order to achieve a therapeutic tricyclic blood level of 100-200 ng/ml (once within the therapeutic range, blood levels were checked monthly). Each patient was independently evaluated by the psychologist 12 to 30 weeks (average 26 weeks) after starting drug treatment.

All 16 myotonic dystrophy patients fulfilled the DSM-III criteria for major depressive disorder. The mean pre-treatment Hamilton Rating Scale for Depression score of the 16 patients was 43.6 (SD 12.3) with a range from 22 to 60. The average pre-treatment Hamilton Scale scores for the ten males (41.2, SD ± 13.1) and the six females (47.7, SD ± 9.2) did not differ. During treatment all patients had sufficient resolution of symptoms to fulfill no longer the DSM-III criteria for major depressive disorder. In all 16 patients the Hamilton Scale scores improved (average reduction in score was 23.1 points). The average during-treatment changes in scores (figure 1) was statistically significant (Student’s t test) at p < 0.001.

Indolence, moodiness, shyness, apathy, and lack of energy and motivation are characteristics that have usually been ascribed to myotonic dystrophy patients. Although a detailed psychoanalytic report attributed the personality disturbance of myotonic dystrophy to the neuromuscular symptomatology, most other reports have claimed that the psychological disturbances are not explicably by the degree of neuromuscular disability. Our previous study suggested that these psychological abnormalities were a part of the symptomatology of depression, and our findings now suggest that this depression in myotonic dystrophy responds to tricyclic antidepressant therapy. The improvement of the depression in all 16 patients is especially noteworthy. Presumably, myotonic dystrophy patients have a genetically-determined defect in central nervous system amine function which results in the depressive syndrome. We believe that further multidisciplinary studies of this unique disease, myotonic dystrophy, may be helpful in understanding the basic mechanisms of affective illness.

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