branches of the facial nerve has been reported before.[5-7] Such observations led Wartenberg[8] 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[9] 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 postfacial palsy contracture and mass movements.[10] 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.[1] 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[2] demonstrated that these patients uniformly have a major depressive disorder as defined by the American Psychiatric Association *DSM-III* criteria.[3] 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[4]) 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 50 weeks (average 26 weeks) after starting drug treatment.

All 16 myotonic dystrophy patients fulfilled the *DSM-III* criteria[3] 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 fulfil 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.[1 5-8] Although a detailed psychoanalytic report[9] attributed the personality disturbance of myotonic dystrophy to the neuromuscular symptomatology, most other reports have claimed that the psychological disturbances are not explicable by the degree of neuromuscular disability.[10-13] Our previous study,[2] 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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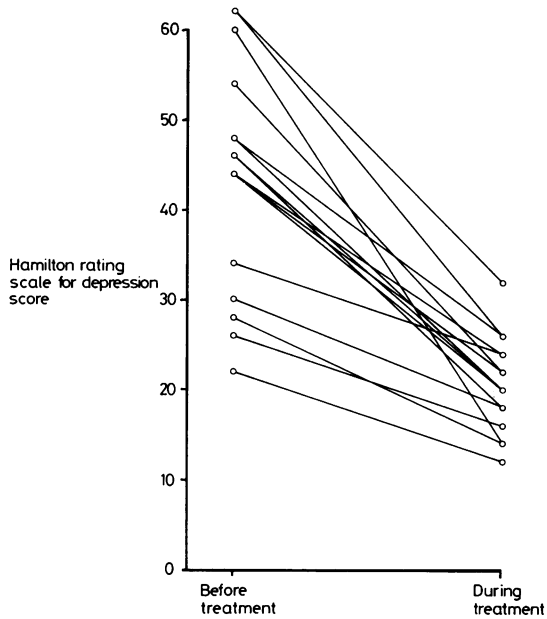

Fig 1 *Scattergram showing pre-treatment and during-treatment Hamilton Rating Scale for Depression scores of sixteen myotonic dystrophy patients treated with imipramine. Lines connect the pre-treatment and during-treatment values for each patient. The scores have been doubled.*

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Enuresis in chronic epiletic patients

Sir: Accurate assessment of the number of fits and an understanding of the natural history of seizures are essential to any long term study of anticonvulsant action. Thus the effect of ageing on seizure frequency in any population shows a 34–38% fall in incidence per decade.[1] In the present study, 82 adult residents of a centre for patients with epilepsy were observed over a three month period during which the nursing staff paid particular attention to the incidence of incontinence of urine, usually in sleep occurring in the absence and the presence of an observed fit.

The table shows that the incidence of enuresis exceeded the total number of observed or reported fits. Daytime fits were more frequent than nocturnal. The small number of nocturnal fits without micturition suggests that the observer factor was most important. These findings appear to contrast with the accepted premise that most fits occur when falling asleep or on waking. It is possible that many episodes of incontinence were due to seizures. Of the recorded episodes of incontinence, one-tenth occurred during daylight hours, not necessarily when the resident was dosing.

When the incidence of incontinence without an observed fit was analysed, 18 residents had enuresis (and one encopresis) as the sole involuntary manifestation during the period of observation. Of these, seven residents accounted for 279 reported episodes and one resident was incontinent every night. If these seven residents are excluded, only 100 episodes of involuntary sphincter dysfunction could be regarded as epileptic equivalents, that is, the sole manifestation of a seizure.

Particular attention was paid to the seven residents with persistent enuresis (average age 51 years) and to the six residents with attacks of encopresis (average age 57 years). Two of the residents with persistent enuresis were female, the others male. None of the affected residents had other evidence of urinary or bowel disorders to suggest that these problems were due to non-neurological dysfunction. Twelve of the thirteen had sustained epilepsy following head injury, invariably with complications such as alcoholism,

Table

	Patients	*Attacks*
Daytime fits		
Continent:	27	66
Incontinent:	31	118
Nocturnal fits		
Continent:	6	35
Incontinent:	12	40
Enuresis: (without observed fit)	36	355
Encopresis: (without observed fit)	6	15