

## Matters arising

Sir: Drs Martyn and Osmond (August 1989) suspect that we have overestimated disease duration, based on the assumption that for any individual, prevalence day is a random event; they are correct. No practical advice emerges from their interesting theoretical account but fortunately we can provide some factual information. Prevalence is a function of incidence or mortality and duration; statistics derived in south Wales between 1985-8, give a figure for duration which varies from 14.8-41.3 years depending on diagnostic classification (clinically definite or suspected) and whether figures for incidence or mortality are applied to this formula. Clearly this is of little help. Alternatively, the duration of disease can be measured in a cohort of cases known to have died in the interval between paired estimates of prevalence; in our own survey, the time between estimated year of onset or diagnosis and death from all causes in 31/37 patients registered between 1985-8 and dead by January 1st, 1988 in whom details were available was 21.7 and 17.3 years respectively. The recorded cause of death, usually registered without necropsy information, was interpreted as being due to multiple sclerosis (usually pneumonia) in 20/37, indirectly related in a further two, unrelated in 14/37 and not recorded in one.

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### Successful therapeutic intervention in a schizophrenic patient with blepharospasm

Sir: The report by Elston *et al*<sup>1</sup> correlating abnormal blinking with blepharospasm and postulating the likely underlying factor to be basal ganglia dysfunction, stimulated me into what was both an unconventional and successful therapeutic intervention in a schizophrenic patient with blepharospasm.

The case is a 42 year old man who has suffered with a schizophrenic illness for nine years with prominent negative symptoms. Until two years ago he was functioning well in the community, attending a work rehabilitation programme regularly, occasionally socialising and managing to spend the majority of his free time watching television and reading.

Over these two years he had apparently developed blepharospasm which had progressively worsened. About three months ago he was absent the majority of time from his work rehabilitation programme being unable to walk to work because of the severity of the blepharospasm and was totally unable to watch television or read. Two months ago I started him on Clonazepam 1 mgm bd increasing after several weeks to 1.5 mgm bd, which has had a very beneficial clinical effect. His episodes of severe blepharospasm now only occur one day per fortnight on average and he has not missed any days at his rehabilitation centre for the last four weeks. He is also able to watch television for 30 minute periods, and in the last week has been able to read the newspaper.

From recent clinical studies<sup>2</sup> Clonazepam has been shown to have a beneficial effect on tardive dyskinesia (also a result of basal ganglia abnormality) and to improve abnormal tracking movements seen in schizophrenic patients.<sup>3</sup> These effects suggest a common mechanism of action by increasing the inhibitory neurotransmitter GABA in this region. It seems that Clonazepam merits further investigation for abnormal eye movement in schizophrenic patients, including blepharospasm, and for the possible treatment of abnormal eye movements in a broader category of patients.

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### References

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- 2 Coyne L *et al*. The effects of benzodiazepines, neuroleptics and tardive dyskinesia on smooth pursuit eye movements in clinical schizophrenics. *Arch Gen Psych* 1988;45: 833-40.
- 3 Thoker GK *et al*. Brain and aminobutyric acid abnormality in tardive dyskinesia. *Arch Gen Psych* 1987;44:522-32.

### Prognosis in stroke

Sir: It is puzzling that in their study of the prognosis for stroke patients Lincoln *et al* (April 1989) paid no attention to the side of the hemiplegia, which must inevitably influence the nature of the perceptual and apraxic problems of such patients. It is a curious fact that few papers dealing with the prognosis for stroke patients from the motor point of view make the distinction between

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left and right hemiplegias, though there are some exceptions.<sup>1-3</sup> From these it appears that right-hemiplegics tend to make better progress, despite aphasia, while patients with a spatial disorder (more likely to be due to a right hemisphere lesion), and patients with a left hemiplegia have poorer sitting balance and a higher death-rate. It is, however, noteworthy that in one of these studies<sup>1</sup> the criterion for walking was walking between parallel bars, and presumably the same applied to climbing stairs, in which the left-hemiplegics did worse. It must be remembered that bars virtually eliminate the need to control the centre of gravity, and a test between bars is a test of progression, not walking.

The difference in terms of postural stability between a right and a left hemiplegia deserves more attention than it has hitherto received. It stands to reason that a right-handed, right-footed person must use his left side as his postural base in most activities, and that therefore a left hemiplegia, with or without perceptual difficulties, will cause more instability than a right hemiplegia, hence more bewilderment, anxiety and depression. In examining patients with strokes clinicians place themselves at an immediate disadvantage by examining them on a couch, or between bars, or using a support, all of which eliminate either the effect of gravity or the need to counterpoise a limb.

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- 3 Wade DT, Hewer RL, Wood VA. Stroke: influence of patient's sex and side of weakness on outcome. *Arch Phys Med Rehabil* 1984;65:513-6.