Outcome prediction in comatose patients: significance of reflex eye movement analysis

Sir: The report by Mueller-Jensen et al. correctly emphasises the importance of reflex eye movements in predicting outcome from coma. Reflex eye movements were not included in the original descriptions of the Glasgow Coma Scale but have been emphasised for diagnostic purposes since 1966 and for prognostic purposes since 1977. There are two additional points to be made.

Although initially absent oculovestibular responses are a poor prognostic sign, we agree that by themselves, they do not invariably predict a poor outcome. In our comprehensive study of 500 patients in non-traumatic coma, 122 lacked oculovestibular responses when first examined, and 3% of them ultimately improved so that they were able to live independently. We also agree that initial absence of both pupillary and oculovestibular responses (or of pupillary and corneal responses or of oculovestibular and corneal responses) is incompatible with the resumption of an independent life.

Although preserved tonic conjugate responses to oculovestibular stimulation is a good prognostic sign early in the course of coma, it is a bad sign if it persists. We noted a similar observation in discussing vegetative patients, namely that preserved, spontaneous, roving, conjugate eye movements at 7 days were associated with a 72% chance of the patient remaining comatose or vegetative. Similarly, we noted 83 patients who by 7 days still had tonic conjugate responses to oculovestibular stimulation, and 64% of them never improved beyond the vegetative state. The prolonged (1 week) absence of cortical influences on spontaneous or reflex eye movements is an ominous sign.

Matters arising

Do white matter changes on MRI and CT differ from vascular dementia due to Alzheimer's disease?

Sir: Erkinjuntti et al. report white matter lesions on MRI in 100% of patients with multi-infarct dementia (MID) and about 35% of patients with Alzheimer's type dementia (ATD) (figures extrapolated from their data). A number of observations arise from this paper. They correctly state that Besson et al. report proton density values in MID to be lower than in ATD. Proton density values in ATD are higher than those of controls, and controls are the same as MID. Erkinjuntti et al. did not measure proton density. They are, however, incorrect in their statement that T1 values were lower in both types of dementia compared with controls. Besson et al. demonstrated that white matter T1 was highly significantly increased in ATD and MID compared with controls. Erkinjuntti et al. did not examine normal subjects. There was a tendency for white matter T1 in MID to be higher than in ATD but this did not reach statistical significance. These findings are thus compatible with those of Erkinjuntti et al. The differences in instrumentation employed in the two studies must also be borne in mind.

Our first study was carried out on a 0.4T (1.7 MHz) resistive system. We have since examined another group of patients with MID, ATD and normal elderly control subjects using a 0.08T (3.4 MHz) system with intercalated saturation recovery/inversion recovery pulse sequence with TR 1000 ms and a tau 200 ms. This study shows that in addition to infarcts, two types of white matter lesion are observed. These are periventricular white matter change (PVWMC) of very high T1 value (> 390 ms) and lower intensity lesions further removed from the surface of the lateral ventricles than the PVWMC and of lower T1 intensity (T1, 310–390 ms) than PVWMC. PVWMC is present in both types of dementia but the lower intensity lesions are present in ATD and correlate positively with age. These results are therefore compatible with those of Erkinjuntti et al.

One of our patients imaged at the age of 63 years, died 3 years later. The image (IR display) shows areas of increased T1 in white matter adjacent to the posterior and anterior horns of the lateral ventricles (fig 1).