Loss of unilateral eye closure and right parietal lesion

Sir: Cases of inability to close the eyes voluntarily (apraxia of eye closure¹) and cases of inability to maintain the eyes closed (eyelid motor impersistence^{2,3}) are known. We report a patient in whom the acquired inability to close the eyes affected alternatively either one or the other eye.

A 70 year old man was investigated for epileptic Jacksonian seizures of the left upper limb. The seizures began in the left eyelid and then spread to the entire left side of the body. A CT scan showed a right parietal tumour (fig). The patient underwent operation and the tumour proved to be a glioblastoma. A month after surgery clinical examination revealed no motor or sensory deficits or lateral homonymous hemianopia. On the other hand, there he had a constructional apraxia and a mild left spatial hemineglect. A disorder of eyelid control was also noted. The patient was able to open and to close his eyes when requested but when asked to keep his eyes closed any attempt to communicate or any mental effort such as calculating was accompanied by instantaneous opening of the two eyes, in spite of repeated orders by the examiner. Often, the left eye opened first, followed after a few seconds by the right one. If the patient was asked again to close his eyes, he did it easily, but any attempt at verbal contact resulted in the re-opening of the two eyes, simultaneously or with the left one first.

A month later the constructional apraxia and neglect disappeared and the patient was

able to maintain his eyes closed in a normal way. He now complained, however, being unable to hunt (his favourite hobby) because he was unable to aim with either eye. The patient was right-handed and he used his right eye for aiming. Simultaneous closure of the two eyes, voluntarily or reflexly, was possible but unilateral closure of only either the right or of the left eye was impossible; the two eyes closed simultaneously or remained open. The patient's previous practice of hunting established that he was able to aim with either his right or his left eye before his illness and hence to close each eye independently. The only way the patient could still hunt was to maintain his left eye hidden by a headband. Clinical examination was still completely normal without any facial palsy.

Classically cortical lesions are considered to affect contralateral face movements, in particular mouth muscles and to a lesser extent orbicularis oculi. It is reported that each hemisphere controls the orbicularis oculi muscles of both sides, with a contralateral predominance.⁴ This anatomical organisation explains eyelid motor impersistence in patients with a unilateral lesion. In our patient the unilateral lesion also caused a bilateral eyelid dysfunction and an inability to close separately either the right or the left eye. This phenomenon was not a minor form of paralysis: all the eyelid movements were completely normal when bilateral and conjugate. The face movements also were normal (reflex, automatic and voluntary). The disorder was solely an inability to dissociate the two eyelid movements. We believe that this phenomenon is an apraxic disorder: a unilateral eyelid closure apraxia. The eyelid signs of our patient were of two types: at first there was a motor eyelid impersistence, followed 2 months later by a unilateral eyelid closure apraxia. Perhaps, these should be seen as two stages of the same syndrome.

Fig CT scan showing the right parietal glioblastoma.

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Disappointing results of increasing benzodiazepine dose after the development of anticonvulsant tolerance

Sir: For several years we have been studying benzodiazepine (BDZ) anticonvulsant tolerance in humans¹ and in an animal model.^{2,3} Clinically, it seems a fairly widely held (although as yet undocumented) view that the development of such tolerance cannot be effectively treated simply by increasing the BDZ dose. We have attempted to investigate this problem by examining data from a defined group of patients, suffering from complex partial and/or grand mal seizures, who had been in a preliminary evaluation of the anticonvulsant efficacy of N-desmethyloclobazam (NDMC),³ the active metabolite of clobazam. All patients had been given both clobazam and NDMC on separate occasions and we documented every instance where the BDZ dose had been increased when relapse followed initial improvement. Clobazam, usually given 20 mg/day initially, was increased by 10 mg increments, but because NDMC was only available in 30 mg capsules the dose of this drug ($t_{1/2} \sim 46$ h) was increased from 30 mg/day to 30 mg and 60 mg on alternate days. Our data (table) show that on only one of thirteen occasions did an increase in dose produce substantial (clinically relevant) improvement. In this instance the patient (No. 4) was given clobazam 20 mg/day for only 6 weeks before his dose increase and in view of his weight (85 kg), and hence relatively low dose (mg/kg), it seems possible that only a very limited degree of tolerance had developed during this time. Interestingly, animal studies in our laboratories suggest that in benzodiazepine tolerant mice the expected rightwards shift of the anticonvulsant dose response curve is accompanied by a considerable reduction in the maximum response (unpublished data). This finding, which is supported by similar observations on tolerance to the behavioural suppressant effects in rats,⁴ would appear to