nasogastric tube was inserted and intravenous fluids (0-9% NaCl) were started. An iv bolus injection of 10 mg of diazepam given over five minutes did not affect seizure activity (fig). Further injections of 20 and 20 mg iv boluses of diazepam were injected after one and four hours respectively with a decrease in the number of seizures (up to 1/hour).

On the second day, an iv infusion of diazepam was started at a dose of 40 mg/24 hours and the same infusion rate was maintained for the third day. As seizures were continuing (1-2/hour) and with increasing frequency, on the fourth day the dosage of diazepam was doubled (that is, 80 mg/24 hours). No effect was observed and the rate of infusion of diazepam was kept at 80 mg/24 hours for the fifth day. During the first five days the fits were occurring at a frequency of 6-7/hour. Since an intravenous preparation of phenytoin (a very effective drug in status epilepticus) is not available in Italy, lamotrigine was started at 7 am on the fifth day (30 hours after doubling the infusion rate of diazepam). Consent was obtained from the Protocol Review Committee and from the patient's parents.

A 600 mg loading dose of lamotrigine (100 mg capsules, Wellcome Research Laboratories, Beckenham, UK) was given over four hours (200 mg x three administrations at two-hour intervals), followed by two additional 200 mg doses over the next 20 hours. Five hours after starting lamotrigine the fits became less frequent (five fits during the subsequent six hours and three during the following 13 hours). As the seizures were well controlled (two to three per day), after three days the patient was discharged on phenobarbital (100 mg twice a day), carbamazepine (400 mg three times a day) and lamotrigine (200 mg twice a day). The range of plasma drug concentrations (μg/ml) over the observation period were: phenobarbital 30-7-356, carbamazepine 69-10-3, diazepam 0-9-1-8 and lamotrigine 0-4-4-9.

Our patient had a generalised convulsions and status epilepticus, a condition which is known to be particularly resistant to treatment and to present frequent spontaneous remissions. However, both the inefficacy of diazepam at the increased rate infusion (that is, 80 mg) and the marked fall in frequency of fits five hours after lamotrigine administration suggest that the termination of status epilepticus in our patient may reasonably be attributed to lamotrigine. Single oral doses of drug (120-240 mg) had been found to be rapidly effective within one to two hours in reducing interictal spike activity and the photosensitive range in epileptic patients. In addition, lamotrigine has been recently reported to dramatically reduce very frequent generalised tonic-clonic seizures and atypical absences and to stop the occurrence of non-convulsive status episodes.

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First description of myasthenia gravis in Spain

Non medical classical literature occasionally yields detailed observations on some neurological disease which was unrecognised at the time. In the eighteenth century old literary traditions such as idealistic descriptions of protagonist's, were replaced by a more realistic approach; heroes were portrayed as normal human beings, and this resulted in the description of real people complete with their medical conditions.

Sir Thomas Willis is usually credited with the first description of myasthenia gravis in western medical literature.1 His report in 1672 of a woman who was able to "speak freely" on occasions but at other times because "mute as a fish" has been ascribed to some authors to hysteria rather than myasthenia. Masteller2 recently suggested the existence of a still earlier historical description of myasthenia in an American Indian, in 1644. The first well documented case of myasthenia published in English medical literature was reported by Samuel Wilks of Guy's Hospital, London in 1877. The term myasthenia gravis was coined in 1895 by Jolly.3

In the Spanish literature, the first report of myasthenia appeared in a book written by Benito Perez Galdos, a famous Spanish writer living in the second half of the nineteenth century. In Tristana,4 published in 1892, Galdos wrote: ”From her life full of work she was left with a nervous weakness and weakness of the eyelid muscles. She was only halfway, and this with difficulty on certain days, or at times when certain winds ruled, sometimes reaching the point where she had to lift up her upper eyelid with her fingers when she wanted to see well any person. In addition, she was ill from her chest, and when the winter came along she was very ill." Myasthenia gravis seems the most likely explanation for this clear description of an old woman with intermittent ptosis.

Galdos was an excellent observer of Spanish society during his lifetime. In addition to his well-recognised literary merits, he reserves the credit for the first description of myasthenia in the Spanish literature.

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MATTERS ARISING

Neuropsychological deficits in patients with minor head injury after concussion and mild concussion

I read with interest the report by Drs Leinninger, et al, further documenting “organic” mentation deficits in patients with “minor head injury.” Unfortunately, Dr Leinninger and colleagues have failed to describe the basis for characterising their patients as having sustained “minor head injuries,” other than implying that these patients suffered only brief periods of loss of consciousness. The emphasis on “organic” neurological deficits in patients with “minor head injury” is extremely inappropriate and long overdue.

In my view, however, the major problem in this area is in the definition of “minor head injury.” In most series the patients are considered to have suffered only “minor head injury” if they have suffered short duration loss of consciousness and no major disturbance of language or motor or visual functions. Most neurologists would affirm that only a relatively limited portion of the brain is actually involved in these specific four areas of brain function, and patients can easily sustain damage to large areas of brain tissue focally or diffusely without disruption of these particular and relatively limited areas of brain. Thus patients classified in this simplistic fashion as having suffered “minor head injury” may in reality have suffered loss of considerable portions of their brain and the discovery of “organic” neurological deficits should come as no surprise.

It would be much more helpful to the medical community and to those with this epidemic disease if the term “mild head injury” were confined to patients documented not to have lost brain tissue either focally or diffusely. However, diffuse loss of brain tissue...
First description of myasthenia gravis in Spain.

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