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Taphoorn et al reply:

We thank Ambrogio and colleagues for their comments on our paper.[1]

They describe a patient with features, as they argue, of both myasthenia gravis and Lambert Eaton myasthenic syndrome (LEMS). The neurophysiological data recorded are compatible with LEMS, as was so in our patient. The combination, however, of LEMS and myasthenia gravis in the patient reported on is less evident than in our patient and in several patients described in the literature, for no antibodies against acetylcholine receptors were detected in serum.[2-5]

The only features suggesting myasthenia gravis are the fluctuating oculobulbar symptoms, which are not exclusive for myasthenia gravis.[6] Moreover we doubt if the manner of death, explained by the authors as a myasthenic crisis, really adds to the diagnosis myasthenia gravis in this patient.

We do not believe the so called "overlap myasthenic syndrome" to be a separate clinical entity; it merely is a combination of the two auto-immune diseases (LEMS and myasthenia gravis) in one patient.

As to the therapeutic implications, patients with a combination of LEMS and myasthenia gravis may be treated with corticosteroids, effective in both diseases.[7,8] Our patient, on a 40 mg prednisone alternate day dose, is still in a good clinical condition.

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Trial of ganglioside GM1 in acute stroke

Sir: In their therapeutic trial, recently reported in this Journal, Dr Hoffbrand and colleagues did not find GM1 therapy "to be of value in the treatment of acute stroke".[1] This indication seems in contrast with two previous clinical studies,[2,3] but the differences in the experimental designs, which are expression of two different approaches concerning the use of GM1 in stroke, may explain the discrepancies of the results.

In our trial[2] GM1 treatment was started two weeks after the onset of neurological deficits (when antioedema therapy had already been stopped) and it was continued daily for six weeks. At the end of this period the naturally occurring recovery after stroke was significantly enhanced by the drug. In our opinion GM1 seems to play a role in functional recovery by stimulating the adaptative reorganisation and the complex mechanisms of the neuronal plasticity (that is neuronal sprouting).

Hoffbrand and colleagues started GM1 therapy within 72 hours from the onset of the neurological deficits and continued for four weeks. It seems to us that such an approach might eventually show the antioedema effectiveness of GM1 (no other antioedema drugs were mentioned), but this drug effect has still to be demonstrated. Furthermore, this treatment could not clearly evaluate GM1 role in enhancing functional recovery because therapy was stopped too early.

Nevertheless, it must be pointed out that Hoffbrand's study reveals two data which are in partial accordance with our findings: (1) as regards mortality, the prognosis is better for patients on therapy with active drug: nine patients in placebo group and five patients in GM1 group died in Hoffbrand's trial, while in our study three patients in placebo and no one in GM1 group died (see patients and methods section of our paper); (2) in Hoffbrand's study, the mean increase in Barthel Index from the end of the first to the sixth month was 7·7 for the placebo group (from 72·3 to 80·0) and 18·0 for the GM1 group (from 70·2 to 88·2): probably such a wide difference is not statistically significant because the study group is not sufficiently large and homogeneous.

On the whole, these data indicate a possible effectiveness of GM1 in the treatment of stroke. We think that the real efficacy of the drug will be demonstrated not only by a larger study, but also through more selective criteria regarding the stroke (ischaemic or haemorrhagic) and the time of therapy (beginning and duration).

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