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Lambert-Eaton myasthenic syndrome

The Lambert-Eaton syndrome is thought to be an autoimmune disorder, and immunoglobulin G (IgG) antibodies from the patients with the syndrome have been shown to act on voltage-dependent calcium channels at nerve terminals to reduce calcium entry and hence acetylcholine release. Patients with the syndrome, however, manifest widespread adrenergic as well as cholinergic dysfunction of the autonomic nervous system, and the IgG from the patients with the syndrome sometimes fails to reduce quantal content in mice.¹ Therefore, the pathophysiology associated with the syndrome may not be solely due to the autoantibodies. We had a patient with the syndrome whose muscle response during surgery indicated the possible involvement of factor(s) other than IgG.

A 63-yr-old male with chronic muscle weakness had a lobectomy of the lung performed for adenocarcinoma. The patient was premedicated with atropine, and the trachea was intubated after a small dose of droperidol and fentanyl but without a muscle relaxant. Anaesthesia was maintained with 50 per cent nitrous oxide and epidural mepivacaine (two per cent). Neuromuscular twitch response was monitored with an ABM-100 (Datex). The stimulating electrodes were placed over the ulnar nerve, and the recording electrodes over the hypothenar muscles

FIGURE NMT = neuromuscular trend, PTF = post-tetanic facilitation.

(Figure 1). After ligation of the left upper pulmonary vein, twitch height increased gradually and reached 120 per cent of the preoperative value within 5.5 hr. Post-tetanic facilitation, which was observed at the start of the operation, disappeared at the end of the operation. Although the skin temperature was not monitored, the rectal temperature was 37.6 and 38.2° C at the times of ligation of the pulmonary vein and at the end of operation, respectively. Electrolyte imbalance was not observed during the operation. This immediate improvement of the reduced muscle response does not seem to be explained by the involvement of IgG as a single aetiology, because IgG has a half-life of about 21 days. A factor or factors other than IgG seem to be involved in the myasthenic syndrome observed in our patient. We suggest that this (or these) factor(s) is secreted from the tumour, affects neuromuscular transmission, and has a relatively short half-life. Our patient may be unusual, because the histology of the tumour showed it to be an adenocarcinoma and not small-cell carcinoma.

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