but this binding did not appear to involve the acetylcholine recognition site of the receptor. Nevertheless, D. B. Drachman (Johns Hopkins University, Baltimore) had induced electrophysiological evidence of MG in immunosuppressed mice by repeated injections of MG globulins over a period of 10 days.

On the other hand, J. Keesey (University of California, Los Angeles) was unable to demonstrate any inhibitory effect of MG serum on α -Bgt binding to mouse diaphragm; E. X. Albuquerque (University of Maryland, Baltimore) had found no reduction in acetylcholine sensitivity in rat or human endplates after treatment with serum from MG patients; and injection of serum globulins or lymphocytes into rats produced no electromyographical evidence of neuromuscular block (T. Namba, Maimonides Medical Center, Brooklyn). Were these negative results due to immunoglobulins being unable to penetrate the intact neuromuscular junction? B. Fulpus (University of Geneva), using α -Bgt-labelled muscles and radioactively-labelled anti- α -Bgt *in vivo* and *in vitro*, clearly showed that the antibody was able to penetrate and bind to the motor endplate.

The second day was concerned with genetic, immunological and clinical aspects of MG. Histocompatibility antigens HL-A₂ and HL-A₃ were shown to be more prevalent in MG patients than in the normal population and divided the patients into two main groups: HL-A₂ being more common in older patients with thymomas and circulating antibodies to skeletal muscle, and HL-A₃ being associated with young females with thymic hyperplasia and no circulating antibodies (M. J. Oosterhuis, Wilhelmina Gasthuis, Amsterdam). The relative proportion and absolute numbers of T and B lymphocytes in MG were reported by several speakers, but no consistent pattern emerged from most of these studies. G. Birnbaum, however (Cornell University Medical College, New York), found decreased T-cell function in young, hyperplastic patients, and a significant rise in T cells and T-cell function after thymectomy. Further immunological studies may well be easier to relate to the disease process if tissue typing is included.

The effect of immunosuppressant therapy was discussed and various regimens for steroid treatment suggested. One interesting aspect presented by G. Matell (Karolinska Institute, Stockholm) was drainage of the thoracic duct undertaken in severe cases. These patients improved remarkably after 24–48 hours of drainage, although their condition deteriorated within 1–2 weeks of stopping the treat-

ment. In contrast, injection of the cell-free lymph back into the same patients produced deterioration within 30–60 minutes.

The animal models presented at this meeting showed considerable similarity to MG. Circulating antibodies raised against solubilised AChR can bind to, and directly block the receptor *in situ*; cell-mediated hypersensitivity to the receptor can produce symptoms of an acute nature, and it may be a necessary prelude to the humoral response in rats. Whether there is a circulating antibody in MG directed against the acetylcholine recognition site of the AChR, and capable of directly blocking the receptor, is still doubtful. It is possible, however, that the immunoglobulins that bind to solubilised human receptors, as shown independently by Appel and Lindstrom, have an indirect inhibitory effect on neuromuscular transmission *in situ*.

Denervation and re-innervation

from Shin-Ho Chung

BEFORE a foetal muscle is innervated, the fibre is 'supersensitive' to the chemical transmitter acetylcholine over its entire length. When a motor nerve forms an excitable junction with the muscle fibre, the chemosensitive area shrinks progressively and becomes restricted to the junction and its immediate surroundings (Diamond and Miledi, *J. Physiol., Lond.*, **162**, 393; 1962). A similar process occurs in adult muscle fibres following regeneration after destruction of the nerve (Miledi, *J. Physiol., Lond.*, **151**, 1, 1960; Axelsson and Thesleff, *J. Physiol., Lond.*, **147**, 178; 1959). Interest in this system stems partly from the fact that, during their supersensitive period, muscle fibres accept innervation from foreign

Pollution from plants

from Peter D. Moore

It is both well known and a source of considerable concern that the particulate matter of the atmosphere contains a burden of toxic heavy metals (see *Nature*, **247**, 510; 1974). In south-west England attempts have been made to monitor the fallout of heavy metals by analysing their levels in superficial leaf washings and in cuticular layers on vegetation (see, for example, Little, *Environ. Pollut.*, **5**, 173; 1973). The reverse situation is described by Beauford, Barber and Barringer in this issue of *Nature* (page 35); they have shown that heavy metals from the soil are absorbed by plant roots, are translocated and eventually released into the atmosphere in particulate form, perhaps with fragments of cuticular waxes. It has been realised for several years that plants can generate atmospheric particulate matter, but this is the first clear demonstration, using radioactive tracer techniques, that the movement of heavy metals from soil to atmosphere may accompany this process.

This being so, it may be necessary to reconsider the use of vegetation as a convenient filter by which heavy metal fallout from the atmosphere can be monitored. It is not usually known what proportion of cuticular and epicuticular metal is derived from the soil through the plant. In many situations the soil levels of heavy metals also reflect atmospheric fallout, in which case the leaf washings will still provide a useful index, but this is not so

where metals in soils are derived from other sources.

But perhaps the most important implication of this work, as the authors point out, is that industrial activity is not the sole source of atmospheric heavy metals. The question which must now be asked concerns the respective importance of these two sources. Here an answer may be found in palaeoecological work by Lee and Tallis (*Nature*, **245**, 216; 1973), who analysed the lead contents of peat samples taken from depths down to 2 m at an upland site in Derbyshire. Lead contents of almost 600 p.p.m. were found in the upper 20 cm of peat, but these fall to about 40 p.p.m. at depths below 20 cm (dated by radiocarbon determination at pre 1460 AD). At depths below 125 cm, the leaf level falls yet further to a fairly steady value of about 10 p.p.m. These samples predate the Roman conquest, during which there was some lead mining and smelting, hence it must reflect the background level of atmospheric lead, to which the vegetation is presumably making its contribution. Thus, even if this background were entirely due to the production of metal-containing particulate matter from plants, the current industrial and domestic production of lead still exceeds it by sixty times. In the midlands of England, therefore, atmospheric lead production by vegetation must be regarded a very minor contribution to the atmospheric content of heavy metals when compared with human sources.