diseases represented in the control group, however, do not necessarily lead to severe wasting of tissues—for example, cervical spondylosis, herpes zoster, or transient ischaemic attacks. It seems to us that the reported results do not exclude the possibility that it is ALS which causes a slight increase in the plasma lead levels, and not lead that causes ALS.

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Reference

Conradi, Sebastian, Ronnevi, Lars-Olof, and Vesterberg, Olof (1978). Increased plasma levels of lead in patients with amyotrophic lateral sclerosis compared with control subjects as determined by flameless atomic absorption spectrophotometry. *Journal of Neurology, Neurosurgery, and Psychiatry*, **41**, 389–393.

SIR,—Encouraged by the interest that our article has aroused, we agree with our colleagues that some points could be discussed further. The degree of immobilisation of our patients varied, but most of them were still able to walk at the time of the study. It is evident that dehydration and wasting might well have influenced the results. As is known, lead can be mobilised from the skeleton in certain conditions, such as immobilisation and after trauma. This is manifested by a rise in whole-blood lead, which, however, our patients did not show. Therefore, the suggestion made by Göthe and Ekenvall that such a mobilisation of lead could give an isolated increase in the plasma levels of lead is purely hypothetical so far.

The difference in the plasma levels of lead between our ALS patients and control subjects is admittedly small, and the levels in all the patients are also clearly lower than those recently reported by Cavalleri *et al.* (1978) in a sample of lead workers not showing neuromuscular symptoms. Statements like "lead (that) causes ALS," are of course completely unjustified in our present state of knowledge, nor have we made such a statement. Still, it is of great importance that the potential role of lead in the pathogenesis of this tragic disease continues to be studied for the following reasons:

1. In ALS patients, earlier overexposure to lead and mercury has been demonstrated unambiguously (Pierce-Ruhland and Patten, 1978), and lead contamination is worldwide.
2. There is evidence of increased lead levels in some tissues, particularly CSF, in ALS patients, as previously shown by us.
3. Peripheral paresis without loss of sensation has been reported as a common symptom in lead intoxication.

Individuals might exist who show an abnormal susceptibility to lead in a yet unknown manner. Abnormalities in the binding of lead to various constituents in the blood could also change the bioavailability of lead. We are currently studying different aspects of the binding of lead in blood of ALS patients. Further, lead might well interact with other substances in a multifactorial pathogenetic process.

For some years we have been trying to test with various methods the hypothesis that in ALS, the motoneurones take up increased amounts of lead through the motor endplates. Undoubtedly, the work has been complicated by the present poor knowledge of the normal distribution of lead in man, including generally accepted normal values in different tissues, such as plasma and CSF. The research on the pathogenesis of ALS has hitherto suffered from a lack of testable working hypotheses. This has probably had a negative influence on the intensity of research activity in this field. The efforts of proving or disproving the above hypothesis could bring valuable new information and also stimulate further research work on this important problem.

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References

Cavalleri, A., Minoia, C., Pozzoli, L., and Baruffini, A. (1978). Determination of plasma lead levels in normal subjects and lead-exposed workers. *British Journal of Industrial Medicine*, **35**, 21–26.

Pierce-Ruhland, R. A., and Patten, B. M. (1978). Cross-sectional analytic case control study of antecedent events in motor neurone disease. In *Proceedings of the IVth International Congress on Neuromuscular Diseases*, Montreal, September 1978. Abstract No. 337.

Corrections

Ballistic elbow flexion movements in patients with ALS

In the paper by Dr Hallett in the March issue on page 235, line 20 in the left-hand column should begin Ag1–Ag2. . . .

Stapedius reflex in multiple sclerosis

In the paper by Dr Hess in the April issue on page 332, lines 7, 8, and 9 of the legend to Fig. 1 should read Calibration of the abscissa: 50 mm = 1 second; calibration of the ordinate: 1 mm. . . .

Notices

Change of Editor

As announced in the editorial in the May issue, Professor C. D. Marsden will be taking over as Editor from January 1980. From now on, therefore, authors should address their manuscripts to the Editor, *Journal of Neurology, Neurosurgery, and Psychiatry*, BMA House, Tavistock Square, London WC1H 9JR.

Change of reference style

From now on papers should be prepared according to the Vancouver style also described in the May editorial. The main change concerns references which should be numbered in the text in the order in which they are mentioned and listed numerically at the end of the paper. Full details are given inside the front cover of each issue of the journal.