Book reviews and letters

subject spread over more than a century is in itself a daunting task. Hécaen has chosen well, keeping a scrupulously fair balance between papers of differing points of view. Space does not permit a closer examination of the individual contributions. Suffice it to say that they are by leading figures in the subject such as Broca, Jackson, Babinski, Milner, Kimura, Gazzaniga, Sperry, and Hécaen.

The papers are arranged in three main sections dealing with (a) the discovery and development of the concept of cerebral dominance (10 papers), (b) functional hemispheric asymmetry (16 papers), and (c) cerebral organisation in lefthanders (four papers). Each paper is preceded by a linking and summarising commentary. Both in terms of number of papers and subject matter, section (b) is by far the most important—in fact one might ask whether the attempt to provide a comprehensive historical point of view at the same time as presenting some of the compelling issues in current research has meant that some of the more recent literature has had to be omitted purely on grounds of space.

In his concluding remarks, Hécaen indicates some of the directions that future research on cerebral dominance might now profitably take, given the existence of the necessary research techniques—for example, what precise role do subcortical structures play in lateralisation, is dominance exclusively a matter of genetic predetermination (an old question in itself), is dominance specific to *Homo sapiens*, and so on?

For the French reader, this book will provide an invaluable survey of the subject. The English reader will probably wonder—and with justification—why a comparable English anthology on cerebral dominance has not yet appeared.

M. K. C. MACMAHON

Terminology of Communication Disorders: Speech, Language, Hearing By L. Nicolosi, E. Harryman and J. Kresheck. (Pp. xv+273; illustrated; \$14.50.) Williams and Wilkins: Baltimore. 1978.

The aim of this work is to provide "a comprehensive dictionary/sourcebook containing definitions of the terminology used in [the fields of speech, language, and hearing] and in allied areas in one

manageable volume." Judged by this courageous criterion, the work is not a success. Spot checks reveal the absence of a whole host of terms used in communication disorders of which a handful are *arteriosclerosis*, *endoscopy*, *hemianopia*, *migraine*, *sphincter*, *thrombosis*, and *Veau operation*. In mitigation one could argue that the field of communication disorders is so vast, encompassing as it does large chunks of the medical, psychological, and linguistic sciences, that to provide comprehensive coverage of the terminology would entail compiling an extremely large and expensive volume. And so the authors have tended to concentrate, it seems, on articulatory, phonatory, and audiological disorders—though for what reason is not stated. Looked at purely from these points of view, the coverage can be described as both adequate and balanced. Within the area of language disorders, however, the coverage is far from adequate. Thus one finds *aneurysm* but not *angioma*, *arcuate fasciculus* but not *thalamus*. Even *basal ganglia*, *disconnection syndrome*, and *upper/lower motor neurone* fail to be listed.

The definitions are generally crisp and to the point, although one might register dissatisfaction with the vagueness of some of them, for example *light lateral*, *Doppler effect*, and *kernel*, and the downright inaccuracy of others, for example *implosive*. The drawings and photographs accompanying some of the entries are of good quality on the whole, but some are confusing, for example the labelling of the vocal and ventricular folds on pages 91 and 220, and the reversal of positions of the epiglottis and oesophagus on page 91. One diagram, that of the Cardinal Vowel chart, is horrendously wrong. Interspersed through the text are various tables listing, for example, the muscles used in speech and the classification of degrees of hearing impairment—all relevant and welcome in a book of this type.

The final section of the book (about a seventh of the total space) is devoted to an annotated tabulation of milestones in child development and the numerous diagnostic and screening tests available in the field of communication disorders. Such information is no doubt of value, but I wonder if the space could not have been better used for further entries in the dictionary.

Despite these reservations, the book is the best we have on the subject of

terminology, although it has few competitors. Anyone working or just straying into the multidisciplinary world of communication disorders would be advised to have access to it.

M. K. C. MACMAHON

Neurotransmitter Systems and their Clinical Disorders Edited by N. J. Legg. (Pp. 240; illustrated; £7.60.) Academic Press: London. 1978.

It used to be said (and often still is) that neurology is a diagnostic speciality, and an anatomical diagnosis was regarded as the acme of the tyro. Recent advances in neurochemistry and neurophysiology, however, have indicated that this is by no means sufficient to understand neurological disorders and, more significantly, allow hope that much more can be done for our patients. This book contains a comprehensive account of many of the growing points in this exciting field. Many chapters are well written, giving a reasonable but not dogmatic picture of what is happening without leaving the reader disoriented. I particularly liked those of Gray on structure of synapses and Bird on Huntington's chorea. Several aspects are not given the prominence they deserve—for example, cholinergic loss in Alzheimer's disease, and the whole field of the encephalins—and some chapters lack clinical relevance. But this is a good book to help in keeping abreast of the times.

ANGUS MCINNES

Letters

Increased plasma lead levels in patients with amyotrophic lateral sclerosis

SIR,—We have read the article by Conradi *et al.*, about increased plasma lead levels in patients with amyotrophic lateral sclerosis (Conradi *et al.*, 1978) with great interest. It seems to us that a possible explanation for the statistically increased plasma lead levels (the values are in themselves normal) might be increased lead mobilisation from the skeleton due to a serious disease causing immobilisation of the patients, severe wasting, and decreased food intake within a fairly short time.

It is not possible to judge the extent of immobilisation in the control group from the diagnoses only. Some of the

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 over-exposure 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 bio-availability 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 motoneurons 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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- Pierce-Ruhland, R. A., and Patten, B. M. (1978). Cross-sectional analytical 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 ms and 1 second; calibration of the ordinate: 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.