Functional integrity of the structural unaffected left hemisphere in crossed aphasia

We would like to comment on the article by Cappa *et al*[1] in which a PET study on crossed aphasia was reported. The authors state: "Neither CT scanning nor MRI can exclude the presence of a left hemispheric functional impairment. Functional imaging methods, such as single photon emission computed tomography (SPECT) and positron emission tomography (PET), have been used in a handful of cases to assess regional cerebral blood flow and metabolism in patients with crossed aphasia." In their two patients, PET scan has shown a functional depression of both hemispheres in the acute stage. Their report is particularly interesting as they suggest that, in the acute stage of a crossed aphasia, only PET can provide information on the functional state of the structurally unaffected left hemisphere; one of the conclusions is that such a functional impairment of the left hemisphere may play an important role in the development of language disturbances in crossed aphasia, thus suggesting a bihemispheric representation of language in these patients.

We have some remarks about the authors' results: (1) as reported by the authors themselves, the first patient was unfortunately not age matched with the control group and was 79 years old; it has been recently shown that cerebral oxidative metabolism decreases with aging.[2] (2) In the first patient, hypometabolism in the left hemisphere was mild compared with the marked crossed cerebellar diaschisis. (3) In the second patient, the bilateral improvement of metabolism on the second PET examination did not really help to distinguish the crucial site responsible for language disturbances.

Regarding the assessment of the functional condition of the left hemisphere, our two cases of crossed aphasia previously reported showed abnormalities in standard EEG and quantified EEG (QEEG) that presented a good relationship with CT and MRI findings, suggesting a functional integrity of the left hemisphere.[3,4] Aphasia is usually related to cortical-subcortical lesions and, in these cases, EEG and QEEG have a good sensitivity and specificity in detecting abnormalities in the affected hemisphere,[5-8] as well as in more widespread diseases.[9] We thus consider our finding of a functional integrity of the left hemisphere assessed by EEG and QEEG to be correct.

We believe the discrepancy between our findings and those of Cappa *et al*[1] is due to the extreme complexity in the physiopathology of crossed aphasia and to the uncertain knowledge, as yet, about brain lateralisation of language in these patients.

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Cappa et al *reply:*

Drs Bandini and Primavera point to a discrepancy between our findings of reduced glucose metabolism in the left hemisphere of two patients with crossed aphasia, and their own results of normal EEG activity in the unaffected hemisphere of right handed patients with language disturbances and right hemispheric pathology.

As a general comment, we think that a direct comparison of EEG findings with measurements of local metabolism and blood flow is probably unwarranted, given the differences between the methods. It is noteworthy that, in a recent review devoted to transcallosal diaschisis, Andrews[1] concluded that the data on contralateral electrical activity in the acute period after a unilateral lesion were "inconclusive", while blood flow and metabolism showed a consistent decrease, followed by gradual return to baseline.

Considering Bandini and Primavera's specific remarks: (1) Case 1 was not age matched with the control group. We consider it to be unlikely that this age difference played a crucial role in the comparison. Although oxygen consumption decreases slightly with normal aging,[2] several studies have confirmed the lack of a significant decline in absolute values of glucose consumption (see, for example, ref 3). In any case, it must be underlined that the reductions in our patient were in the 30–50% range in comparison with control values. (2) A direct quantitative comparison between crossed cerebellar and transcallosal diaschisis is of limited interest, given the present uncertainty about the mechanisms underlying these phenomena.[4] Both findings are well documented in the medical literature on PET.[5,6] (3) The increase of metabolism is needed bilateral, as clearly indicated by the lack of interaction in analysis of variance. This finding is in agreement with the participation of the contralateral hemisphere in the early phase of recovery, both in patients with "standard" aphasia and in patients with atypical language dominance. Our specific hypothesis, that the latter patients may be more liable to remote effects of focal lesions, remains open to further investigation.

In conclusion, we think that the differences between our report and Bandini and Primavera's findings are mainly due to the different methods. The subject of crossed aphasia, as well as of other instances of atypical cerebral dominance, is far from being completely understood, and remains a crucial research area for the understanding of the neural correlates of cognitive functions.

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BOOK REVIEWS

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Brain's Disease of the Nervous System/ 10th Edition. Edited by JOHN WALTON. (Pp 801, Price: £95.00) 1993. Oxford University Press. ISBN 019-261969-1.

When the first edition of Brain's, "Diseases of the Nervous System" was published in 1933, its initial reception did not suggest a long life expectation; and yet now a review of the tenth edition is invited, some 60 years later!

The book was first reviewed in "Brain", and although the piece was unsigned, the somewhat acerbic style is still recognisable. Several of the points made in the review can be considered in assessing the later and this, the latest, edition.

"The crux of the medical writer's problem today is not what he is to include but what he should omit . . . it is a defect of some modern textbooks that their authors have never formulated their problem in these terms and have left it obscure as to whom they wish to reach."

Lord Walton maintains, in his preface,

that his book will *"inevitably stand as a permanent monument to Lord Brain's clinical expertise, to his thoughtful approach to neurological medicine and to his outstanding literary skills."* With this in mind, the traditional structure is maintained, but the text has been largely rewritten, following the O.U.P. style. Lord Walton has acceded to the general view that the individual author can no longer prepare a comprehensive manuscript on neurological disease and he has recruited some younger authors, introducing much new written material and illustrations. He includes a chapter on rehabilitation. He does not clearly identify the audience for whom he has prepared the book.

"Gowers prepared himself for his task by long apprenticeship in the practice of medicine, he wrote because he had something to impart and not simply because there was room on the market for a book on his subject."

". . . crystallised his own experience. It was not a compilation. A textbook of the first rank can be written on no other foundation, and the authority of much current medical writing suffers by the departure from this soundprinciple." Lord Walton the editor certainly fulfils these early criteria.

The first edition was criticised for attempting to *"embody all the most recent and recondite advances in neurology and neuropathology, the speculative as well as the established,"* where, *"nothing has been missed, and the result is rather a concise encyclopaedia than working guide to neurology. Anatomy and physiology too, have been treated at unusual length . . . much of both these subjects is of purely academic interest and lacks practical application."* In this the tenth edition the two subjects are still included but in a much modified and applied fashion.

In the 1940 edition, Brain included a new chapter on the "Psychological manifestations of organic nervous disease" and repeated this in his post-war edition of 1947. Again, Walton has continued the tradition with a section on Neuropsychological Syndromes written by a clinical neurologist and "Psychiatric presentations in neurological practice" are addressed in the final two pages.

This edition will not deserve the criticism that *"whilst embodying the new in his book the author has not always omitted the views which this has rendered obsolete"* Edition one, *"was not free from contradictions on important points"* and *"doubt was felt as to the book's purpose. It provides too much and too indiscriminately for the practitioner, too little and too superficially for the neurologist."* With this constant problem in producing a text book short of an encyclopaedia, what has the author to achieve? Simply a review of the subject matter; a careful account of the commonly occurring disorders; a mention of the less common, and adequate references to take the student to further reading. This Walton has achieved by way of comprehensive chapter referencing. The text is broken up, at times into fragments which are too small. This can make reading difficult. Whilst there are no inaccuracies in large areas of the text, the index is flawed. For example, chronic fatigue syndrome appears not on p 249 (subarachnoid haemorrhage) but on 349. However, on discovery there is a very well reasoned and reasonable account of this contemporary and controversial problem.

It is pleasant to meet some old acquaintances. The gentleman with severe endocrine exophthalmos still looks out at us after 24 years, but apart from the dermatome map from the "Pocket atlas of Anatomy", unchanged from my fourth edition, (March, 1951), the illustrations are new and the picture of ophthalmic Grave's disease is so clear it should be retained!

As anticipated, the text is well written, carefully edited and I would judge this to be a very good book; certainly to be preferred to many of the alternatives on this side of the Atlantic. Now with a shared authorship, future editions will be assured.

JB FOSTER

Frontal Lobe Function and Dysfunction. Edited by H S LEVIN, H M EISENBERG and A L BENTON. (Pp 427; Price: £40.00). 1992. Oxford University Press. ISBN 0-19-506284-1.

Historical problems in frontal lobe research have included a tendency to expect the whole of the frontal lobes (nearly half the hemispheres) to have a single function, and a parallel tendency to try to delineate one characteristic clinical frontal lobe syndrome. There has also sometimes been insufficient regard for the immense connectivity of frontal cortex with other cortical areas and with subcortical areas, where lesions can produce elements of putative "frontal" syndromes. It is misleading to define "frontalism" on the basis of one or a few clinical tests such as the Wisconsin Card Sorting test, and a clear distinction must be preserved between the functional and the anatomical level of analysis. This excellent book, which summarizes much recent thinking on the subject, demonstrates that further understanding of the frontal lobes will involve firstly a more refined taxonomy of functions and secondly a neuroanatomical and neurophysiological mapping of specific functions.

Among Diamond's dicta are (1) use more than one task linked to a given neural substrate (convergent validity); (2) study the role of other neural regions in the same tasks (divergent validity); and study other tasks linked to other neural circuits (in other words, seek double dissociations); (3) use the *same* tasks when comparing populations rather than ones which are merely similar; and (4) study qualitative aspects of performance (why does the patient fail?). There is evidently a long way to go before these ideals are attained.

After Benton's useful historical introduction to the prefrontal region the first part of the book discusses anatomy, supporting Damasio's claim, in a stimulating epilogue, that progress in this area will depend in great degree on a better understanding of connectivities.

Subsequent sections cover clinical aspects of cognition; motor function (with a useful and provocative clinical chapter on this topic by Heilman and Watson); behaviour; development; and rehabilitation. The arrangement sometimes seems a little arbitrary. Although there is a section on integration of experimental studies with clinical data, a fuller impression of how anatomical and physiological and behavioural studies in primates relate to humans comes from reading the book as a whole. In contrast to most multi-author books, this one retains sufficient consistency of concepts and terminology to be largely intelligible as a continuous text.

Has the recent evolution of the frontal lobes led to a qualitative rather than merely quantitative departure from simple stimulus-response models of cerebral function? Did our frontal lobes liberate us from automatic behaviour, enabling us *not* to do things? The book takes a modern, cognitive viewpoint but an older behaviourist perspective is detectable in places.

Operational concepts of volition and consciousness are closely related and probably inseparable. If they are characteristically frontal lobe attributes, and if frontal cortex is heavily involved in the sort of "central" or non-modular processes which Fodor termed isotropic, it is small wonder that the moorland still looks rather bare.

CHRISTOPHER D WARD

Neurological Examination Made Easy. By GERAINT FULLER. (Pp 220 Illustrated; Price: £9.95 P/bk). 1993. Edinburgh, Churchill Livingstone. ISBN 0-443-04294-2.

This is another **made easy** book for medical students and it makes neurological examination far more complex than it actually is. It starts with an assumption that neurological examination can be used as 'screening tool' or as 'investigative tool'. I think it is a part of a good medical examination that forms the basis of the practice of medicine rather than a 'tool'.

The language used is very simple and the illustrations useful. In particular the figures used for demonstrating the sensory loss with use of small sections of spinal cord is a good idea and does convey useful information. The figures for abnormalities of the optic fundus, however, are confusing and I think colour pictures rather than line drawings make a better impact. There is an attempt to fit most of the examination findings in a form of flow chart, and the title for each is 'simplified approach'. I think it makes it complicated and there are always difficulties when you try to fit patients in flow diagrams.

Power testing and grading can always be controversial. MRC grades were designed largely to record power in poliomyelitis trials and research. For the clinician a good description as to what the patient can and can not do with a particular muscle is far more relevant. Medical students when taught neurological examination should learn to do just that rather than giving a 'number' to the weakness. This apart, the book flows very well and can certainly be recommended to students of medicine.

ATUL BINIWALE

Clinical Geriatric Neurology. Edited by LAURIE BARCLAY. (Pp 513; Price: £82.00). 1993. Waverly Europe Ltd. ISBN 0-8121-1610-0.

Most of the major symptoms in old age have a neurological component—unsteadiness, falls, intellectual impairment, incontinence. Though physical dependence is commonly caused by neurological disease it is not always recognised that much can usefully be done by way of preventative measures, early treatment and rehabilitation, to improve the wellbeing and independence of our elders. This book aims to outline the principles and provide practical guidance for the undergraduate and younger doctor dealing with neurological