Matters arising

evidence for axial sparing that has yet appeared. The effect is apparent from the bar graphs of the fig, and standard parametric tests establish its significance statistically. The merits of the experiment should not be obscured by the mistaken conclusion, however, for the results are of interest not only as proof of axial sparing but for their bearing on other issues concerning apraxia.

From a clinical point of view, the magnitude of the axial sparing effect is as important as its statistical significance. The data of Poeck *et al* give the first quantitative estimates of the size of the effect. On the average, aphasic patients make 83% more errors on non-axial commands than they do on axial commands. The increase is 52% for Global, 70% for Wernicke, 93% for Broca, and 116% for Amnesic patients. Axial sparing is thus a major feature of apraxia in all four forms of aphasia.

These percentage increases are directly proportional to the percentage of correct responses for each group. The non-significant interaction term in the multivariate tests establishes statistically that the size of the axial sparing effect is independent of the specific form of aphasia. This is an important finding, for it indicates that axial sparing is an inherent feature of ideomotor apraxia, not an occasional or ancillary phenomenon. Theories of the neural mechanism responsible for apraxia therefore need to explain not only why axial sparing occurs, but why it appears uniformly in different forms of aphasia.

Apart from its implications for axial sparing the study of Poeck *et al* challenges two current beliefs about ideomotor apraxia. In nonfluent forms of aphasia (Broca, Global) buccofacial apraxia is generally thought to be more severe than limb apraxia.^{8,9} In the present analysis, however, they are not significantly different. In the individual case, of course, buccofacial or limb apraxia may occur in isolation; but the data indicate that such differences average out over a series of patients with the same form of aphasia. Apraxia is also widely believed to be less severe in Wernicke than in nonfluent forms of aphasia.^{9,10} The present results, however, show apraxia to be more severe in the Wernicke than in the Broca group for all four types of movement (fig). Assessments of relative incidence or severity are of course always subject to sampling bias due to the selection and classification of patients. The results of Poeck *et al* nevertheless merit attention because the carefully balanced design of their experiment minimises many sources of bias that operated in earlier studies.

It is thus the two forms of aphasia with

marked verbal comprehension deficits (Global, Wernicke) that show the most severe ideomotor apraxia here. One might suppose that the test scores for these groups are lowered by their failure to understand some of the commands. Two additional findings reported by Poeck *et al*, however, speak against that explanation. First, the ordering of patient groups by severity is the same for performance to imitation, where verbal comprehension is not involved. Second, the authors present a classification of the errors to axial commands which shows that the type of error is proportionately the same for each group. If comprehension were a significant factor the category that includes failures to understand commands would be disproportionately large in the Global and Wernicke groups.

Failures of comprehension therefore do not appear to explain why the Global and Wernicke groups have the most severe degrees of apraxia. The most likely hypothesis is that the severity of apraxia reflects the average size of lesion in the four groups. In a group study of this kind such an effect could be present even though the correlation between severity of deficit and lesion size may be very small for individual cases. The fact that the pattern of apraxic deficits is the same in the different forms of aphasia, with a strong axial-sparing effect and no significant differences between the three non-axial types of movement, suggests that apraxia cannot be localised to cortical regions associated with specific types of language disorder. Yet it is also true that ideomotor apraxia is practically never seen except in association with some form of aphasia.^{10,11} The lesions for apraxia thus appear to be strongly localised to the language territory of the brain, yet not to any of the subregions of that territory associated with specific aphasic syndromes.

This picture fits nicely with the disconnection model of apraxia proposed by Geschwind. Apraxia for limb and oral commands he attributes to disruption of the pathway from Wernicke's area to the ipsilateral premotor area and thence via the corpus callosum to the contralateral premotor area of the other hemisphere. Any unilateral lesion that interrupts this pathway would almost inevitably produce an aphasia, and the larger the lesion the more likely it is to involve the critical connections. That would explain why the movement disorders of apraxia are strongly associated with the language disorders of aphasia, and also why axial sparing is uniform in all forms of aphasia. Finally, as all non-axial commands must be transmitted over the same pathway, the theory explains why the average severity of

apraxia is the same for movements of the arm, leg, and oral musculature, as the data indicate.

Geschwind's disconnection theory thus accounts for the major features of apraxia brought out by the experiment of Poeck *et al*, as well as for the phenomenon of axial sparing itself. Many details of the theory are still open to question, but its main assumptions receive striking confirmation from these new findings.

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Poeck and Willmes reply

Dr Howes has looked at our data from a perspective that differs from ours in a critical respect. We had chosen to investigate whether performance in the five conditions (oral, arm, leg, bimanual, and axial movements) was different in any one of the four standard aphasia subgroups. This amounts to admitting that the overall null hypothesis: no difference between any of the conditions of apraxia testing for the four patient groups might be tenable. In contrast, Dr Howes has taken Dr Geschwind's view for granted and

has focused his analysis on the problem whether axial movements are less impaired than oral movements. These different approaches necessitate different allocation of type-I error levels for the statistical tests employed. Consequently, we have applied the multiple test procedure by Holm,¹ comparing all pairs of conditions in all aphasia groups, which requires a sizeable type-I error reduction for each single comparison. Dr Howes maintains that his analysis of our data has demonstrated "axial sparing" because of the greater power of the statistical procedures applied by him. With all due respect we should prefer the view that the differences in results are mainly due to the different hypotheses tested. This becomes evident if one formalises the sequence of steps in Dr Howes' and in our (PLW) analysis:

$RH_{\text{Howes}} = => SP: SH: \mu_{\text{axial}} > \mu_{\text{oral}}$
(one-sided alternative)

$RH_{\text{PLW}} = => SP: SH_i: \mu_i = \mu_j, i \neq j \in$
{arm, leg, oral, bimanual, axial} (with
two-sided alternatives),

where RH stands for research hypothesis, SP for statistical prediction, SH for statistical null- or alternative hypothesis(es), μ for (theoretical) mean proportion correct. Obviously, the type-I error level for the individual statistical test must be different for both approaches.

Dr Howes strongly argues in favour of some and against other statistical procedures. We take the liberty to disagree: Nonparametric tests are not, per se, liable to error. It is understood that in a single computer run descriptive statistics are determined as well as the nonparametric test which helps to recognise erroneous data input.

We are afraid that bar graphs without information on interindividual variability of scores (that was included in our table 4) do not easily permit conclusions by mere inspection.

The computation of the probability value of $1/4^4$ would be correct only if the performances in the four different tasks were (statistically) independent, but each single patient carried out all tasks.

The application of the t test requires that the dependent variable has interval scale properties. This implies that equal differences in percentages correct indicate equal differences in performance at any point of the scale. It is absolutely uncertain whether, for example, a difference between two and four items correctly indicates the

same difference in ability on the alleged underlying (latent) dimension, that is, apraxia, as the difference between eight and ten. Consequently, we do not concur with the view that an overall 83% increase of errors from axial to non-axial commands reflects a salient increase in severity of apraxia.

Finally, application of log-linear models to contingency tables with (positively) dependent entries can lead to grossly anti-conservative results.^{2,3}

Of still greater importance is a problem that is neglected in many experimental investigations in neuropsychology. Comparison of raw scores or percentages correct for sets of tasks tacitly assumes that these tasks make demands of equal difficulty. This assumption is, however, quite often not justified. The assembly of tasks in any study on apraxia, including our own, is neither based on an explicit theory of complex motor performance nor on an empirically based selection (as opposed to "invention") of items, let alone the standardisation procedures required for any psychometric test. In this respect, apraxia research is still in a prescientific stage as compared with aphasia research.

While we do not agree that our data lend support to Geschwind's claim on the preservation of axial movements in ideomotor apraxia we suggest that an in-depth study on the varieties of apraxia still has to be done. This study would have to consider three basic prerogatives: (1) selection of tasks according to modern teaching of motor physiology instead of borrowing tasks from Liepmann's armamentarium,⁴⁻⁷ (2) construction of a psychometrically valid test battery, (3) standardisation of that battery on a sample of several hundred apraxic patients, a number that would permit the application of psychometric single case analysis procedures.⁸

It might well be that such an investigation would lead to a revision of the conception of apraxia.

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Post epileptic headache and migraine

Sir: We read with interest the paper on post epileptic headache and migraine by Schon and Blau.¹ The authors studied 100 epileptic patients and found that 50% of them complained of a post-ictal headache. Seizures were divided into "minor" and "major". Most of the patients had headache after "major" seizures whereas only 16 had headache after "minor" seizures. As considered by the authors, headache following generalised convulsions may be caused by general metabolic changes and an increase in cerebral blood flow up to 300%-400%, as shown in animals.² On the contrary, headache following minor seizures is less easily explained by focal reactive hyperaemia.³⁻⁶ Such headache, that may have migraine features, may be a model worthy of further study as suggested by Schon and Blau.¹ However, for this purpose a classification of minor seizures should be kept in mind, since the category of "minor" seizures comprises different epileptic mechanisms and of course different kinds of seizures. This facet of the problem was completely overlooked by the authors. In a paper of ours⁷ we showed that among 174 patients with "minor" seizures (comprising single partial seizures, complex partial seizures, typical absence and myoclonic absences), a post-ictal headache, in some cases with migraine features, was present only after partial complex seizures. If further confirmed, this observation may have physiopathological implications. In fact, focal metabolic and blood flow changes due to neuronal discharge are present both