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

Matters arising

in partial elementary and partial complex seizures.³⁻⁶ So these changes and a possibly related Leao's spreading depression⁸ cannot explain why headache follows partial complex seizures but not partial elementary seizures. A different mechanism therefore should operate in post-ictal headache. Dana-Haeri *et al*⁹ and Pritchard *et al*¹⁰ reported a significant rise in prolactin levels after complex partial seizures but not after partial elementary ones. Such hormonal changes suggest involvement of hypothalamic nuclei in complex partial seizures. Does neuronal discharge involving noradrenergic and serotonergic pathways originating in the locus coeruleus and brainstem trigger the vascular changes that may be responsible for headache? A similar "central" mechanism has been hypothesised for migraine.¹¹ We think that to obtain useful information on the mechanism of post-ictal headache, further studies should be performed taking into consideration seizure physiopathology and completed by neuroendocrinological and neurophysiological findings.

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A double-blind controlled trial of high dose methylprednisolone in patients with multiple sclerosis

Sir: High dose methylprednisolone seems to be a promising treatment in multiple sclerosis patients.¹

Some questions though remain unanswered by the authors: firstly, did the benefit last longer than four weeks? From the study by Rose *et al*² it is known that the difference in recovery between ACTH and placebo-treated multiple sclerosis patients suffering from a relapse was no longer statistically significant after four weeks. Secondly, was the severity of relapse comparable in both groups? Clinical disability scores were higher in methylprednisolone treated patients than in controls; this might have reflected more serious relapse in the former which usually results in relatively more recovery as measured by steps on the EDSS score. Thirdly, in the chronic progressive group, the actively treated patients were also more disabled on the Kurtzke Scale than the controls; they improved markedly because of a decrease on spasticity.

The authors did not state whether there was any difference between the groups with regard to physiotherapy and treatment with muscle relaxants.

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Milligan *et al* reply:

The trial was designed only to assess short-term effects of high dose pulsed intravenous methylprednisolone (iv MP); we did not feel justified in withholding corticosteroids for longer than four weeks in placebo treated patients, especially those in whom disability increased during the trial. Forty-eight of 50 participants were reviewed 13-35 (mean 23, SD6) months later, under open conditions and in some cases after receiving additional treatment with corticosteroids. Mean disability status scores at entry, one month and later follow-up were 5.4, SD1.8, 4.2, SD2.4 and 4.9, SD2.8 in the iv MP treated group compared with 4.2, SD1.7, 4.1, SD2.0 and 4.8, SD2.8 in placebo treated patients. Four patients had improved, eight were unchanged and 12 worse in the iv MP group at late follow up; corresponding figures for placebo treated patients were 6, 8 and 10. Patients in relapse have not been distinguished from those with chronic progressive disease in this analysis. As expected, there is therefore no long-term benefit following the intention to treat with a single course of iv MP.

In assessing the balance between beneficial and side effects, eight adverse events occurred during 1,116 patient months of follow-up of which seven developed in actively treated patients but none was severe and only two (pain in the dorsal spine and hip, each without radiological abnormality) possibly related to iv MP.

Owing to a randomisation bias, patients treated with iv MP during relapse had higher disability scores at entry than those in the placebo group. Numerically, there was therefore a greater potential for recovery following iv MP treatment; however, we are unaware of evidence indicating that more severely affected patients do actually achieve greater spontaneous recovery over one month than individuals with milder relapses and there are considerable difficulties in attaching significance to numerical changes in the disability status scale which is not linear. Patients in relapse who were most severely affected at presentation did not make a disproportionate contribution to our statistical analysis of results (see fig 1¹). It is therefore likely that the clinical improvement observed was a consequence of treatment. These arguments also apply to the chronic progressive group in which there is even less