erythematosus patients, CSF analysis is only abnormal in about 50% of patients with slight protein and IgG elevation. The initial reports of the value of low CSF C4 levels have not been substantiated. The same is true for SCF cyclic GMP levels, CSF immune complexes, lymphocytotoxic antibodies and DNA antibodies. EEG, CT scan, angiographic changes are not diagnostic. The use of 15-oxygen and $C^{15}O_2$ scan⁴ is promising, but too sophisticated for routine use. Recently, Morrow and others⁵ have commented that existing individual immunological tests are generally poor indices of disease activity (notably so with cerebral manifestations and thrombocytopenia). Monitoring the activity of systemic lupus erythematosus remains a difficult task.

In conclusion, early diagnosis of cerebral systemic lupus erythematosus still relies on high clinical suspicion. In difficult cases a combination of various immunological tests including immunofluorescence study of skin biopsy, may be useful in making a definite diagnosis so that steroid therapy can be commenced early.

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Anticonvulsant peripheral neuropathy

Sir: The article by Shorvon SD, and Reynolds EH, entitled *Anticonvulsant peripheral neuropathy: a clinical and elec-*

trophysiological study of patients on single drug treatment with phenytoin, carbamazepine or barbiturates,¹ represents progress in the attempt to determine neurotoxic side effects occurring from anti-convulsive medication. However, a few statements regarding earlier clinical studies need to be corrected.

(1) Shorvon and Reynolds write: "Hopf² reported a reversible slowing in nerve conduction in human volunteers . . .". The study by Hopf does not allow this conclusion as only two patients were electrophysiological examined after the medication was discontinued. A closer look at Shorvon's and Reynolds' table 1 p621—*Previous clinical and electrophysiological studies of phenytoin-induced peripheral neuropathy*—reveals a number of mistakes, or at least undue simplifications.

(2) The study by Lovelace and Horwitz³ is said not to have used a control group. Table 4 p74 in Lovelace's and Horwitz's study shows that controls were used, although the reader might be left with some doubt about how the controls were obtained.

(3) The patients reported in the study by Eisen *et al*⁴ were all treated for more than ten years. It should therefore read correctly in Shorvon's and Reynolds' table 1 >10. In contrast to the information Shorvon and Reynolds give in table 1, Eisen *et al*⁴ stated in their abstract that their patients had normal serum folate concentrations. Furthermore Eisen *et al*⁴ did compare the electrophysiological measurements with the results derived from 70 controls, which again is not mentioned in Shorvon and Reynolds table 1.

(4) Some of the information which Shorvon's and Reynolds' table 1 gives about a Polish study is incorrect. Zebrowska-Szymusik⁵ described on p429 that one patient had a serum diphenylhydantoin concentration in the toxic range. However, table IV p430 in Zebrowska-Szymusik's paper contains probably a typographical error, as the reader may get the impression that a diphenylhydantoin serum concentration of 10 mg/ml is considered to be in the toxic range. Zebrowska-Szymusik's Table III p429 shows that five patients (11.9%) has absent or weak reflexes. In my view it cannot be concluded from the information given by Zebrowska-Szymusik that the patients in her study received other drugs besides phenytoin.

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Shorvon and Reynolds reply:

Table 1 in our paper is no more than a summary of previous papers as an introduction to our own work. It was not our purpose nor did we discuss these previous publications in any detail. Professor Danner's comments and interpretation of the papers by Hopf, Lovelace and Horwitz, and Eisen *et al* are, to say the least, debatable. However, we are grateful to him for pointing out two errors in our table.

(1) The length of phenytoin treatment in the paper by Eisen *et al* should have read "more than 10 years" and not "10 years". This was an error overlooked at the proof stage. (2) The incidence of clinical neuropathy in the paper by Zebrowska-Szymusik should have read 8.9% and not 0%. This was due to a mistranslation from the original Polish.

None of Professor Danner's comments have any bearing on our own findings and conclusions.

Treatment of acquired aphasia: speech therapists and volunteers compared

Sir: In a recent issue of *J Neurol Neurosurg Psychiatry*, David, Enderby and Bainton¹ reported the results of a multicentre study comparing the effects of speech therapists and untrained volunteers on aphasia out-

come following stroke. In this study, aphasic patients were randomly assigned to speech therapists or untrained volunteers for 30 hours of individual treatment. The volunteers were provided with a detailed description of the patient's communication problems, suggestions for the manner in which to interact with the patient, and instructed to "encourage the patient to communicate as well as possible". The speech therapists gave each patient such therapy as they thought appropriate for the 30 treatment hours. Response to treatment was measured with overall scores from the *Functional Communication Profile*² (FCP) after 2, 4, 8, and 12 weeks of treatment and after cessation of treatment. Two pre-treatment baseline assessments using the FCP were taken one week apart. Results revealed that patients in both groups improved and that there were no significant differences between the groups at the pre-, within, and post-treatment intervals. Similar findings were obtained for both high and low level patients treated by speech therapists and volunteers.

Several important issues need to be considered before the results of this study can be used to support the employment of untrained volunteers in treatment of aphasic patients. Without the benefit of inter- and intra-examiner reliability data, the conclusion that aphasic patients will respond to treatment by untrained volunteers as well as to that provided by speech therapists, or vice versa, is untenable. In scoring the FCP, patients are rated as "normal", "good", "fair", "poor", and "none" on 45 communicative behaviours including broad performance categories, such as "reading letters", "understanding movies", "saying phrases" and "talking on the telephone". Determination of a rating requires familiarity with the patient and necessitates a subjective interpretation of the patient's abilities relative to his pre-morbid state. The examining clinicians in this study were not familiar with the patients they tested. David and her colleagues indicated that they were able to reduce inter-observer variation by providing suitable training, but do not tell us what this variation was. The fact that several therapists in different centres are involved in the administration of the FCP further heightens the need for reliability data.

A second major methodological problem that arises from the David study concerns the baseline pre-treatment measures. Baseline measures essentially imply steady state performance. Both groups of subjects reflected marked improvement from BL 1

to BL 2. Since the patients received no treatment during this time, this improvement must be attributed to other factors. Some of these might include (1) added spontaneous remission, (2) a placebo effect, or (3) increased patient familiarity with the test interview situation. Baseline measures are ordinarily employed in single case design research.³ In group studies baseline levels are essentially impossible to obtain inasmuch as some members of the group improve, some do not change and some may deteriorate during the baseline phase. Without the benefit of a stable baseline, treatment effects are difficult to ascertain.

Analysis of variance results indicated that both groups responded to treatment with significant changes in FCP scores. This improvement, however, was primarily restricted to baseline changes, and improvement from BL 2 to the first within-treatment measure. Beyond this point both groups improve minimally. This may result from the fact that 30 hours of treatment is minimal for an aphasic patient. The data provided in several large efficacy studies⁴⁻⁶ suggest that much more treatment is required to obtain positive changes.

Additional information is needed to determine the value of volunteers as surrogate clinicians. It is not stated whether a volunteer worked with one or several patients. If they worked with more than one patient, it is possible that some may have become more proficient therapists as the study progressed. It would be helpful to know if volunteers were screened before being assigned to patient care responsibilities. Information as to the educational levels and socioeconomic status of the volunteers would also be useful in determining their role as adjunct therapists. While the importance of significant others in the total rehabilitation of the aphasic person is vital, it is important to remember that volunteers in this study were directed by the speech therapists.

The authors state that the most essential finding of their study was that treatment had a positive effect on a group of late referrals as well as early referrals. This information is highly supportive of earlier investigations^{7,8} that have shown chronic aphasic patients to improve significantly following intensive speech and language therapy. The David *et al* study supports the contention that treatment, in any form, is beneficial to the aphasic patient. Information as to the effect of types of treatment, must come from further research.^{9,10}

This project was approved by the Research

and Development Committee of the Veterans Administration Medical Center, Portland, Oregon.

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David replies:

We welcome Marshall and Golper's comments on our paper. We did not include our own reliability data on the FCP in this paper because it is a well standardised test in its own right. However, for the reasons which Marshall and Golper cite we considered it important that the interscorer reliability should be checked. We undertook two studies: (1) Two therapists (RD and PE) together interviewed 15 aphasic