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 electrophysiologically 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-