peripheral nerves. Ten patients had neuroretinitis, two children had paresis of the facial nerve, and three adult women complained of neuralgia.⁷ One case study presented a peripheral facial nerve paralysis as a complication of CSD.⁸

Up until now CIDP has never been reported as a neurological complication of CSD.

Given the history and clinical course, the electrophysiological and nerve biopsy findings, coupled with the strongly positive serology to *B henselae*, we think that the CIDP in this patient is a direct complication of CSD. CIDP is an autoimmune process in which both humoral and cellular factors are thought to participate in the pathogenesis. Wheeler *et al* also suggested an immune response as a pathophysiological mechanism responsible for CSD encephalopathy.¹ In our patient a delayed myelin destruction is induced by sensitised macrophages, originally activated by the Bartonella infection. Therefore, we hypothesise that the pathophysiology of both central and peripheral nervous system complications after a CSD infection shares a similar immunological mechanism.

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CORRESPONDENCE

"Can't you use another vaccine"? Postrabies vaccination encephalitis

I read with interest the letter of Chau *et al* related to iatrogenic disseminated encephalomyelitis, in a man bitten "by his own apparently normal dog".¹ Whereas I appreciate the novelty of documenting such entities with brain MRI, I wish to question the reasoning behind the practice of antirabies vaccination in similar doubtful circumstances. I have commented on that issue in another occasion.² I have also documented instances of excessive enthusiasm, often encountered in situations such as those under discussion, in a research project conducted at the Pasteur Institute in Tehran, Iran.³ There is no justification for vaccinating a person

simply because of fear when the remedy itself is to be feared even more, as documented by Chua *et al*.

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The authors reply:

We thank Derakhshan for his comments on our case report of iatrogenic disseminated encephalomyelitis after use of the suckling mouse brain postrabies exposure vaccination. We were not responsible for the postexposure vaccination, which was administered in a provincial hospital in central VietNam. However, in the circumstances (and in the absence of the human diploid cell tissue culture vaccine) we think that it was appropriate to use the vaccine in this case. The dog had previously been well behaved and it was highly uncharacteristic for it to bite its owner. After the event the dog disappeared into the forest and was not seen again. Hence, it was not possible to retrieve the brain for analysis, as is usual in most cases.

The mortality from rabies is essentially 100%, a figure that can be reduced dramatically by the expeditious use of the suckling mouse brain vaccine after exposure. We agree with Derakhshan's comments on excessive enthusiasm for any medication, and obviously the relative risks and potential benefits must always be balanced. In a disease with a 100% mortality, where a potentially effective treatment is associated with a severe side effect in only 1:27 000 cases it would seem reasonable to use the treatment. At this centre we vaccinate 2000 people every year after a dog bite, we see about 50 people a year die of rabies. We would therefore anticipate seeing a case of iatrogenic disseminated encephalomyelitis after use of the suckling mouse brain postrabies exposure vaccination once every 13.5 years. In the same period we would see 675 people dying from the disease.

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Sleep benefit in Parkinson's disease

We read with great interest the report by Bateman *et al* on sleep benefit in Parkinson's disease.¹ The authors state that no objective study has been undertaken about sleep benefit. We must object to that, as our group has recently published an extensive objective study about sleep benefit, which includes objective motor examinations, levodopa plasma concentration determinations, and polysomnographies.² Moreover, we would like to briefly discuss some of the authors' findings in the light of our own results.

The authors studied 20 patients with the motor part of the UPDRS, at waking (apparently twice), and after medication during "on" and "off". At each rating, the patients

completed an "activities of daily living" (ADL) questionnaire. The authors also administered ADL questionnaires to heterogeneous groups of outpatients.

In their results, the authors comment on six out of 16 patients of the first series: "When they awoke these patients performed as well as when they were 'on' due to medication". Unfortunately, they give no data at all, such as mean UPDRS scores and ranges, for baseline state and "on" time schedules and hours of the ratings. Furthermore, no information is provided to account for the remaining four patients who were included in the first series but do not appear in the results.

The findings by Bateman *et al* contrast with our own results, in that patients with sleep benefit performed only slightly better in the morning compared with those without. A clear "on" compared with baseline was found in our study both in patients with and without sleep benefit after intake of their regular medication. We concluded from our data that sleep benefit was much smaller than expected. A morning baseline function as good as a drug induced "on", as described by Bateman *et al*, would be indeed a sleep benefit of considerable magnitude. On the other hand, a drug induced "on" similar to the morning baseline state could theoretically also point to an insufficiently drug treated patient group. In any case, as no data are given to allow comparisons, one is confined to speculate why sleep benefit could be so much greater in British patients than the Argentinian population, where sleep benefit, although objectively existing, was quite a subtle phenomenon.

The authors further state that a "strong correlation" was found between ADL and UPDRS, and conclude that ADL may serve as a "more objective" instrument to measure sleep benefit. Unfortunately they do not indicate if the correlation was found at any point in time or if all evaluations were lumped together, as no correlation index or graph is given.

In the second sample of the study, 113 patients completed an ADL questionnaire at three points in time (at waking, best, and worst) before any drug intake. This was done at home. The authors determined that sleep benefit was present when the mean ADL score difference between best and worst was more than 12—that is, when strong variations occurred in baseline score before medication. The validity of this arbitrary definition deserves some discussion. Firstly, to take this variation as a criterion for sleep benefit may lead to a confusion with motor fluctuations. As the ADL score has a maximum of 52 points, an absolute score difference of 12 as a prerequisite for sleep benefit will lead to the exclusion of patients with smaller fluctuations irrespective of sleep benefit. So their own definition could have biased the authors' finding that patients with sleep benefit had a younger disease onset, longer disease duration, and more frequent use of bromocriptine. All this might also occur in a fluctuating subgroup of patients and an association of sleep benefit with fluctuations has been previously described.³

Secondly, it is necessary to be cautious in considering ADL questionnaires as an objective measure to determine the presence of sleep benefit. The patient rating is based on how well he thinks he could perform at a given moment, and as we pointed out in our study, a large difference between self perceived motor function and objective motor function may occur in sleep benefit.