peripheral nerves. Ten patients had neuroretinitis, two children had paresis of the facial nerve, and three adult women complained of neuralgia.[2] One case study presented a peripheral facial nerve paralysis as a complication of CSD.[5]

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.

PATRICIA M MCNEILL
AAD VERRIPS
REINIER A MULLAART
FONS J M GABREËLS
*Department of Pediatric Neurology, Neuromuscular Centre Nijmegen, University Hospital Nijmegen, PO Box 9101, 6500 HB Nijmegen, The Netherlands*

ANNEKE W M GABREËLS-FESTEN
JAN G M KNIBBELER
*Department of Neurology*

Correspondence to: Dr A Verrips
A.Verrips@ckskg.azn.nl

1 Wheeler SW, Wolf SM, Steinberg EA. Cat scratch encephalopathy. *Neurology* 1997;**49**:876–8.
2 Carithers HA, Margileth AM. Cat scratch disease. Acute encephalopathy and other neurologic manifestations. *Am J Dis Child* 1991; **145**:98–101.
3 Carithers HA. Oculoglandular disease of Parinaud: a manifestation of cat scratch disease. *Am J Dis Child* 1978;**132**:1195–200.
4 Reed JB, Scales DK, Wong MT, *et al*. Bartonella henselae neuroretinitis in cat scratch disease. Diagnosis, management and sequelae. *Ophtalmology* 1998;**105**:459–66.
5 Walter RS, Eppes SC. Cat scratch disease presenting with peripheral facial nerve paralysis. *Pediatrics* 1998;**101**:E13.

# 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 apparantly 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*.

I DERAKHSHAN
*415 Morris Street, Suite 405, Charleston, West Virginia 25301, USA*
*Irajsudi@AOL.com*

1 Chau NVV, Hien TT, Sellar R, *et al*. "Can't you use another vaccine"? Postrabies vaccination encephalitis. *J Neurol Neurosurg Psychiatry* 1999;**67**:555–6.
2 Derakhshan I. Is the Negri body specific for rabies? A light and electon microscopical study. *Arch Neurol* 1975;**32**:75–9.
3 Derakhshan I, *et al*. Light microscopical diagnosis of rabies; a reappraisal. *Lancet* 1978;i: 302–3.

*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 enthusisasm 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.

NGUYEN VINH CHAU
TRAN TINH HIEN
JEREMY FARRAR
*Centre for Tropical Diseases Ho Chi Min City, 190 Ben Ham Tu, Quan 5, Ho Chi Min City, Vietnam*

### 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 heterogenous 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.

Finally, we would like to add a word of caution and remind the authors that it would be wise to avoid deriving the measure of "objective duration" of sleep benefit from three scales filled in at home by patients without any further instructions than to fill them at waking, and during best and worst before drug intake.

As the only dopamine agonist mentioned in this study is bromocriptine, we would be grateful to know what year the study was conducted in.

In any case, although "little is known about sleep benefit"[1] any study concerning this phenomenon should certainly attempt to increase knowledge and avoid a further increment of confusion.

BIRGIT HÖGL
*Universitätsklinik für Neurologie, Anichstrasse 35, A-6020 Innsbruck, Austria*

OSCAR GERSHANIK
*Centro Neurológico, Hospital Francés, La Rioja 951, 1221 Buenos Aires, Argentina*

Correspondence to: Dr Birgit Högl
birgit.ho@uklibk.ac.at

1 Bateman DE, Levett K, Marsden CD. Sleep benefit in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1999;**67**:384–5.
2 Högl BE, Gómez-Arévalo G, García S, *et al*. A clinical, pharmacologic and polysomnographic study of sleep benefit in Parkinson's disease. *Neurology* 1998;**50**:1332–9.
3 Factor SA, McAlarney T, Sanchez-Ramos JR, *et al*. Sleep disorders and sleep effect in Parkinson's disease. *Mov Disord* 1990;**5**:280–5.

*Bateman replies:*

The difference in results is due to a different definition of sleep benefit. Sleep benefit as defined in our paper refers to mobility as good as "on" on waking, which wears off over a variable period. Högl *et al* define sleep benefit as "self perceived mobility in the morning before drug intake as better than during the rest of the day."[1]

The purpose of the first part of our study was to verify the existence of sleep benefit as we had defined it, particularly in view of the findings of Högl *et al* that "patients with sleep benefit had a small improvement between night and morning" and "sleep benefit patients were clearly in the "off" state during baseline motor examination". Our inpatient study showed that six out of the 16 "who were studied from the moment of waking" performed as well as when they were "on" due to medication. Subsequently they spontaneously turned "off" to an identical state to "off" after medication. Four patients could not be studied from the moment of waking as they awoke before the investigator! We clearly found that sleep benefit is as good as "on" after medication and wears off, not an intermediate stable state between "on" and "off" as Högl *et al* have defined it, by subsequently giving these patients their normal medication on the same day and monitoring their response by half hourly UPDRS and ADL scores.

Aware of the patient's misperceptions about sleep benefit, we wished to confirm as objectively as possible the findings from our outpatient questionnaire by using the ADL rating scales. The inpatient study showed a correlation between motor UPDRS "on" score and ADL "on" score of $r$=0.72, $t$=4.42, p<0.001 and sleep benefit ratings $r$=0.62, $t$=3.35, p<0.01. The ADL maximum score is 125. It consists of 25 items that can be rated on a five point scale. Dr P Brown, originator of the scale, suggested that a change of 12 would be sufficient to confirm sleep benefit. As we were aware that sleep benefit, confirmed by personal observation, can represent a substantial change in motor performance, this criterion seemed reasonable. The correlation between motor UPDRS and ADL scores in our study was good, showing that the ADL scores are generally a reliable measure, although there will inevitably be exceptions.

Our study showed that sleep benefit as we defined it was generally a feature of patients with young onset Parkinson's disease. A 73 year old patient described in their paper, with disease onset at 62, would be unlikely to have sleep benefit as we defined it. Their paper, as their figure 2 shows, refers to a different phenomenon.

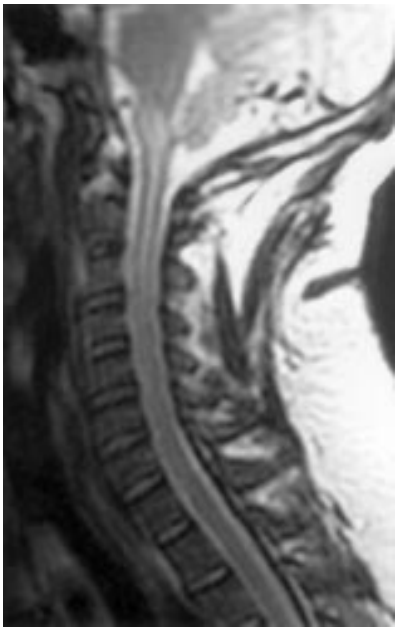
D E BATEMAN
*Royal United Hospital, Combe Park, Bath BA1 3NG, UK*

1 Högl BE, Gómez-Arevalo G, Garcia S, *et al*. A clinical, pharmacologic and polysomnographic study of sleep benefit in Parkinson's disease. *Neurology* 1998;**50**:1332–9.

## Intracranial dural fistula as a cause of diffuse MRI enhancement of the cervical spinal cord

We read the recent short report by Bousson *et al*[1] on spinal MR findings in a patient with progressive myelopathy and intracranial dural arteriovenous fistula with great interest.

We recently had a 42 year old man admitted as an emergency with a 3 week history of stepwise altered sensation in both lower limbs ascending to the torso which progressed to weakness involving his legs and hands. Two years before this he had an episode of severe backache associated with a tight band of pain around the waist and significant bilateral leg weakness. Resolution ocurred only after 4 months, when he was able to walk normally. On the current admission examination showed a spastic tetraparesis; there was minimally increased tone in the upper limbs, mild weakness of the small muscles of both hands, and marked pyramidal weakness of the legs with extensor plantar responses. He was unable to support his weight and was in urinary retention. He had a sensory level at T5 although dorsal column function was preserved.



*MRI of lower brain and spinal cord.*

Brain and spinal cord MRI showed increased signal in the medulla extending into the upper cervical cord down to C4 (figure). Slightly prominent vessels were seen overlying the right cerebellar hemisphere and a varix was visible close to the torcula. There was no enhancement in the cord or medulla and no abnormal flow voids in the spinal veins. The changes were thought to represent a spinal cord infarct and in view of the "stuttering" course in his history we proceeded to cerebral angiography. This showed an arteriovenous fistula supplied by the left middle and posterior meningeal artery and both occipital arteries. Venous drainage was into prominent varices lying just to the left of the midline and in front of the transverse sinus and then on the transverse sinus itself.

After an unsuccessful attempt at embolisation via the arterial route, the fistula was occluded by packing the varix with Guglieni detachable coils.

He made an uneventful recovery; after 2 months of intensive neurorehabilitation he recovered full function in his upper limbs and now has sufficient power in his legs to be able to walk with the aid of crutches.

This case emphasises that an intracranial arteriovenous fistula should be included in the differential diagnosis of increased signal on MRI of the cervical cord, even when dilated veins are not, as in this case, very apparent. Prodromal symptoms can occur and a careful history in a patient with ascending paraparesis and tetraparesis is essential. Endovasculaar occlusion at these fistulae can lead to useful inprovement in neurological function.

FADY G JOSEPH
PIA AMSLER
C M WILES
*Department of Neurology, University Hospital of Wales, Cardiff*

S F S HALPIN
*Department of Neuroradiology*

Correspondence to: Dr F G Joseph
Fadyjoseph@netscapeonline.co.uk

1 Bousson V, Brunereau L, Vahedi K, *et al*. Intracranial dural fistula as a cause of diffuse MR enhancement of the cervical spinal cord. *J Neurol Neurosurg Psychiatry* 1999;**67**:227–30.

## Inverse relation between Braak stage and cerebrovascular pathology in Alzheimer predominant dementia

Goulding *et al*[1] carried out a preliminary retrospective postmortem analysis of 25 patients (13 men, 12 women, mean age 80.7 years) with the clinical diagnosis of Alzheimer-type dementia (only one with suspected multi-infarct dementia) and a 36.4% frequency of the ApoEε4 allele. Eighteen brains (89%) with neuritic Braak stage ⩽4 had either additional cerebrovascular lesions (n=14), or Lewy bodies (n=l), or both (n=6), with a significant inverse correlation between cerebrovascular lesions and Braak stage. Forty eight per cent of the brains showed small focal infarcts, and only 20% disclosed "pure" Alzheimer's disease pathology. No association between the ε4 allele and any pathological variable was found. Based on these data, the authors emphasised the importance of screening for concomitant pathology in Alzheimer's disease, in which a cerebrovascular component has been suggested as an