to run) and manifested as spasm of the left side of the face and flexor spasm of the left arm and left leg. There were no premonitory symptoms and consciousness was normal during the attack. Attacks lasted for a few seconds only and he felt perfectly well afterwards. Neurological examination was normal. Brain MRI was normal and three EEGs performed over the course of the next year were all normal. There were no biochemical or haematological abnormalities; copper studies were normal. A diagnosis of paroxysmal kinesigenic choreoathetosis was made. He was allergic to carbamazepine and did not respond to sodium valproate.

Lamotrigine(3,5-diamino-6-[2,3-dichlorophenyl]-1,2,4-triazine) is a use dependent blocker of voltage gated sodium channels and has a similar anticonvulsant profile to phenytoin and carbamazepine in animal models.[5] Lamotrigine at a dose of 50 mg twice daily, completely abolished the involuntary movements. On two occasions, these returned on stopping lamotrigine and were abolished by reinstituting treatment. Our patient has taken this for over 2 years without ill effect. He now only experiences symptoms of paroxysmal kinesigenic choreoathetosis if he omits his medication. The response to lamotrigine may provide insight into the pathogenesis of the disease, suggesting that it may be caused by an ion channel defect; these are known to be responsible for some paroxysmal neurological conditions.[6] As far as we are aware, this is the first report of the successful use of lamotrigine to treat paroxysmal kinesigenic choreoathetosis.

A C PEREIRA
W J LOO
Department of Clinical Neurology, Ipswich Hospital, Ipswich, IP4 5PD, UK

M BAMFORD
Department of Paediatrics

S J WROE
Department of Clinical Neurology, Ipswich Hospital, Ipswich, IP4 5PD, UK

Correspondence to: Dr S J Wroe

1 Lishman WA, Symonds CD, Witty CW, *et al*. Seizures induced by movement. *Brain* 1962;**85**:93–108.
2 Kertesz A. Paroxysmal kinesigenic choreoathetosis: an entity within the paroxysmal choreoathetosis syndrome. Description of 10 cases and 1 autopsied. *Neurology* 1967;**17**: 680–90.
3 Demirkiran M, Jankovic J. Paroxsmal dyskinesias: clinical features and classification. *Ann Neurol* 1995;**38**:571–9.
4 Marsden CD, Luders HO. Paroxysmal choreoathetosis. In: Luders HO, ed. *Supplementary sensorimotor area*. Vol 70. Philadelphia: Lippincott-Raven; 1996;467–70.
5 Leach MJ, Lees G, Riddall DR. Lamotrigine: mechanisms of action. In: Levy RH, Mattson RH, Meldrum BS, eds. *Antiepileptic drugs.* 4th ed. New York: Raven Press, 1995;861–9.
6 Hanna MG, Wood NW, Kullmann DM. Ion channels and neurological disease: DNA based diagnosis is now possible, and ion channels may be important in common paroxysmal disorders. *J Neurol Neurosurg Psychiatry* 1998;**65**: 427–31.

Chronic inflammatory demyelinating polyneuropathy as a complication of cat scratch disease

Cat scratch disease (CSD) was first described in 1950 as a benign regional lymphadenitis. This infection is caused by *Bartonella henselae*. The clinical range of CSD has expanded beyond the classic presentation. In 5%–20% of the infected patients the disease may spread to other organs. However, neurological complications associated with CSD are rare, with encephalopathy being by far the most common form (90%) of nervous system involvement. Encephalopathy occurs in 2%–3% of patients and is more common in adults than in children with the onset varying from a few days to months after diagnosis of CSD.[1] Other known neurological manifestations, often in combination with encephalopathy, are neuroretinitis, oculoglandular disease of Parinaud, myelopathy, radiculopathy or abducens nerve, and facial nerve paresis. We report on a 3 year old boy who developed chronic inflammatory demyelinating polyneuropathy (CIDP) 6 weeks after identification of CSD.

A previously healthy 3 year old boy presented in a paediatric clinic with regional lymphadenitis of the submandibular and suboccipital glands. He was afebrile and did not have any other symptoms. There was no hepatosplenomegaly. No neurological signs were present on physical examination. As the boy might have been scratched by one of his pet cats, in accordance with a small cut on his scalp, CSD was suspected. It was serologically confirmed (enzyme linked immunosorbent assay (ELISA) *B henselae* IgG> 850 U/l; IgM> 250U/l) and he was treated with clarithromycin.

Six weeks after the onset of CSD, he showed difficulty in walking, inability to run or climb stairs, and frequent falls. Slowly progressive pain and a gait disorder were noted over a course of a further 8 weeks. On admission to our department there were no general signs and only a small submandibular lymph node remained. Mental state and cranial nerve examination were normal. He showed a symmetric distal muscle weakness in all limbs. A marked sensory ataxia was noted. We found mild root pain on a straight leg raising test. Deep tendon reflexes were very low to absent, especially in the legs. Plantar responses were flexor.

Serum concentrations of liver enzymes and glucose, and a protein spectrum were normal. Laboratory tests for adeno, RS, corona, influenza 1–2–3, parainfluenza 1–2–3, sendai, mumps, measles, herpes simplex and varicella zoster viruses, *Mycoplasma pneumoniae, Chlamydia psittaci, Coxiella burnetti, Mycobacterium tuberculosis* and atypical mycobacteria were negative. Antinuclear factor and ANCA were negative. Levels of complement factor 3 and 4 were normal. ELISA *B henselae* IgG in serum, 3.5 months after outbreak of lymphadenitis, was still >850 U/l. IgM specific antibodies now were <200 U/l. Examination of CSF showed 13 mononuclear leucocytes/μl, no polynuclear leucocytes, raised protein concentration (558 mg/l, normally 160–310 mg/l), three oligoclonal bands in CSF on isoelectric focusing, and a slight intrathecal IgG synthesis (51 mg/l, normally 3.2–15 mg/l). IgG and IgM specific antibodies against *B henselae* were negative in two CSF samples. Polymerase chain reaction (PCR) on *B henselae* in CSF and in serum was negative. Enolase, myelin basic protein, S100, lactate and glucose concentrations in CSF were normal.

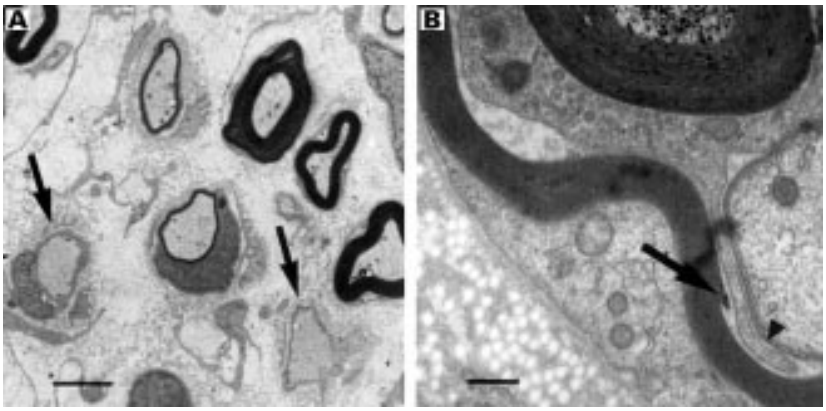
Electroencephalography, cranial CT, and gadolinium enhanced MRI of the thoracolumbar region were normal. Nerve conduction studies showed a marked decrease of motor nerve conduction velocities in all limbs (median nerve 20 m/s). In sensory conduction studies no response could be elicited. The EMG was normal.

The sural nerve biopsy showed many demyelinated axons and signs of early remyelination, but no onion bulbs (figure A). The density of myelinated fibres was slightly below normal, which could be attributed partly to intrafascicular oedema. Myelinated fibre size histogram was bimodal. There were no endoneurial infiltrates, or signs of vasculitis. Electron microscopy showed macrophage induced demyelination (figure B). Polymerase chain reaction (PCR) on *B henselae* in sural nerve fragments was negative.

The history and clinical presentation, combined with CSF findings and the results of EMG and light and electron microscopy studies, are compatible with the diagnosis of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP).

He was treated with prednisone (15 mg every other day for 1 month) and the dose was reduced slowly in the course of 4 months. His motor and sensory function recovered completely and deep tendon reflexes reappeared. Nerve conduction studies 1 year after the onset of CSD were normal.

Neurological complications of CSD are rare and predominantly of the CNS. Myelitis has been described in combination with encephalopathy[2] and radiculopathy was reported only in combination with encephalomyelitis. Ophthalmological problems are oculoglandular disease of Parinaud[3] and neuroretinitis.[4] In a study by Carithers and Margileth of 76 patients with CSD and neurological complications, 15 patients showed signs of dysfunction of cranial or



(A) Sural nerve. Low power electron micrograph. Demyelinated fibres (arrows) and thinly remyelinated fibres. Bar=2.0 μm. (B) Sural nerve; detail of myelinated fibre. Macrophage has invaded the myelinated fibre; process of macrophage is extending between axon and myelin sheath (arrow head). The damaged inner major dense line is terminating as a small dark swelling (arrow). Bar=0.2 μm.

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.