



Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.



CASE REPORT

Idiopathic polyradiculoneuropathy in a Bengal cat: electrophysiological findings and 1 year follow-up

Nicolas Granger DVM, MRCVS, DECVN*, **Catherine E Stalin** MA, Vet MB,
Thomas B Harcourt Brown MA, Vet MB,
Nick D Jeffery BVSc, PhD, DECVN, DECVS, FRCVS

*Department of Veterinary Medicine,
The Queen's Veterinary School
Hospital, University of Cambridge,
Madingley Road, Cambridge CB3
0ES, United Kingdom*

This report describes a rapidly progressive loss of motor function in a 16-month-old male neutered Bengal cat, beginning in the pelvic limbs and progressing to involve all limbs and rendering the cat non-ambulatory. The neurological examination revealed flaccid tetraparesis with decreased spinal reflexes but preserved conscious proprioception and skin sensation. Extensive electrophysiological tests were conducted including electromyography, motor and sensory peripheral nerves potential recordings and 'late' potentials, defining the electrodiagnostic characteristics of this disease. Based on the electrophysiological findings, a generalised proximal and predominantly axonal neuropathy affecting the ventral (motor) nerve roots was suspected. As no aetiology was identified, this disease was classified as idiopathic polyradiculoneuropathy. Over a year, the cat presented three separate episodes of tetraparesis, each with a spontaneous complete recovery, consistent with the reportedly good prognosis for this disease.

Date accepted: 13 March 2008

© 2008 ESFM and AAEP. Published by Elsevier Ltd. All rights reserved.

A 16-month-old male neutered Bengal cat was presented to the The Queen's Veterinary School Hospital for a rapidly progressive loss of motor function in all four limbs. Clinical signs appeared insidiously over 2–3 days, beginning in the pelvic limbs and progressing to involve all limbs and rendering the cat non-ambulatory, before stabilising the following week. The cat was examined 8 days after becoming unable to walk. The cat had had a similar episode at 6 months of age; at that time, motor function improved significantly over 2–3 weeks and took 3 months to return to normality. The cat was vaccinated as a kitten, but not since, and lived exclusively indoors.

No abnormalities were detected during general physical examination, except marked, generalised muscle atrophy (paraspinal and appendicular muscles) with sparing of the cranial musculature. The neurological examination revealed flaccid tetraparesis but the cat remained

able to hold the head in a normal position (see [Supplementary material](#)). Flexor reflexes, tibialis cranialis and extensor carpi radialis reflexes were decreased and the cutaneous trunci reflex was absent bilaterally. Conscious proprioception and skin sensation were well preserved. Cranial nerve reflexes were normal and no change in vocalisation was reported.

The clinical findings were consistent with a generalised neuromuscular disorder, for example, a myopathy, junctionopathy or a primarily motor neuropathy. A cause could not be identified through examination of serum biochemistry and complete blood cell count. Serology for evidence of exposure to feline coronavirus was negative. Exposure to toxoplasmosis was also negative as evidenced by normal serum IgG and IgM levels, on two consecutive blood samples taken 3 weeks apart.

Ten days after the onset of the tetraparesis, the cat's neurological signs were unchanged and electrodiagnostic (Sapphire unit, Medelec, Oxford Instruments, UK) investigations were

*Corresponding author. E-mail: ng311@cam.ac.uk

conducted under general anaesthesia (propofol (Rapinovet; Schering–Plough; 6.5 mg/kg, IV), sevoflurane (Sevoflurane Baxter; Baxter), oxygen) (Cuddon 2002).

Electromyography (EMG) revealed widespread abnormal spontaneous electrical activity (graded as ++; range: 0–4+ (Kimura 2001)), consisting primarily of positive sharp waves (Fig 1) and more occasional fibrillation potentials in most of the appendicular and paravertebral muscles. The EMG of the head muscles was normal.

The left-side ulnar, tibial and peroneal nerves were examined by nerve conduction studies, as previously recommended in cats (Cuddon 2002), and the results are summarised in Table 1. The residual M wave latency and F ratio were calculated using previously described formula (Cuddon 2002). Compound muscle action potentials (CMAPs) were markedly reduced in amplitude for all the tested nerves (Fig 2 and Table 1) (Malik and Ho 1989). Temporal dispersion of the CMAP was found in the tibial and peroneal nerves and the peroneal CMAP was polyphasic (Fig 2). Motor nerve conduction in the tibial, peroneal and ulnar nerves was at the lower end of the expected range compared to normal values in cats (Table 1) (Malik and Ho 1989). Conduction block was found in the ulnar nerve, as evidenced by finding a proximal/distal CMAP amplitude ratio of 0.4 with a change in duration of 7%. The sensory wave of the peroneal nerve was easily recorded and of normal conduction velocity (Table 1) (Redding and Ingram 1984).

In order to distinguish distal from proximal (nerve roots) disease, residual M wave latencies and F wave studies were examined. Residual M wave latencies of the three tested nerves were

within normal limits, whereas the latencies of F waves were increased compared with normal values in cats (Table 1) (Knecht et al 1985, Malik and Ho 1989). The F ratio was markedly increased (Cuddon 1995). Finally, examination of cisternal cerebrospinal fluid (CSF) revealed a normal cell count and a normal total protein concentration. The CSF cytology was normal.

Based on the above electrophysiological findings, a generalised proximal and predominantly axonal neuropathy affecting the ventral (motor) nerve roots was suspected. The absence of identified aetiology implies a categorisation as idiopathic polyradiculoneuropathy (IP).

The cat was discharged 4 days after the initial consultation (14 days after the onset of tetraparesis) and was already showing signs of improvement. Passive physiotherapy over the next 2 weeks was advised. The cat was reported to be able to walk almost normally 1 month later, although mildly weak. At that time, the cat was presented again to the referring veterinarian for similar, but more profound, neurological signs. This third bout regressed following 2 weeks hospitalisation for supportive treatment. Four months after this last episode, the cat was described as normal by the owners. The last contact with the owner was at 11 months after the electrodiagnostic investigation and the cat was normal.

This report describes the clinical and electrophysiological findings in a cat presented with a rapidly progressive flaccid tetraparesis, and the clinical follow-up of the cat over a year. It adds to the literature a detailed neurological description of a polyradiculoneuropathy in a cat, thus aiding other veterinarians in recognising this disease. This report also adds new

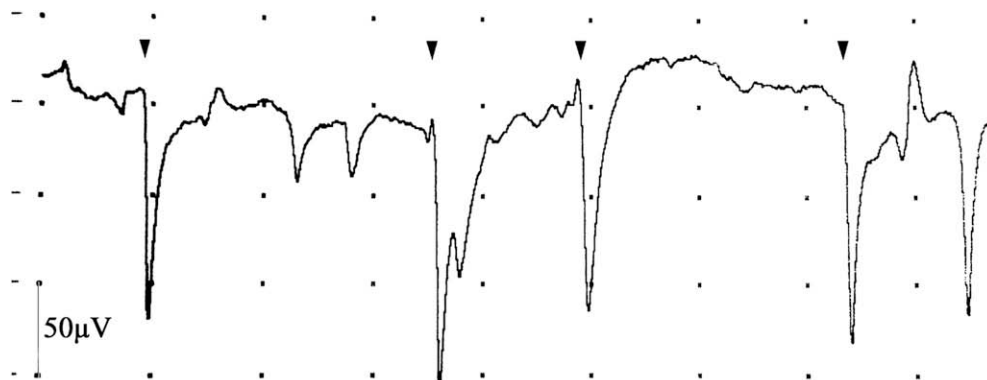


Fig 1. Electromyographic recording obtained from the presented cat under general anaesthesia. After inserting a concentric needle in the tibial cranial muscle, abnormal spontaneous electrical activity was recorded, consisting primarily of positive sharp waves (arrowheads).

Table 1. Numerical results of the electroneurographic examination of the cat

	Stimulated nerve		
	Tibial	Peroneal	Ulnar
CMAP amplitude (distal stimulation)	2.7	7.5	3.5
Normal CMAP amplitude (distal stimulation) (Malik and Ho 1989)	20.9 ± 3.4	30.9 ± 6.6	18.8 ± 4.7
CMAP amplitude (proximal stimulation)	2.7	7.5	1.4
Normal CMAP amplitude (proximal stimulation) (Malik and Ho 1989)	15.6 ± 4.0	29.0 ± 6.2	15.7 ± 4.8
Motor nerve conduction velocity	75	83	83
Normal motor nerve conduction velocity (Malik and Ho 1989)	101.4 ± 12.9	88.3 ± 17.8	120.0 ± 24.4
Sensory nerve conduction velocity	NT	99	NT
Normal sensory nerve conduction velocity (Redding and Ingram 1984)	80.2 ± 7.9	85.3 ± 6.8	89.2 ± 10.3
M wave residual latency	1.47	2.01	1.35
Normal M wave residual latency (Malik and Ho 1989)	1.8 ± 0.4	1.9 ± 0.6	1.3 ± 0.3
F wave latency	15.2	17.4	12.5
Normal F wave latency (Knecht et al 1985)	9.5 ± 1.0	NR	8.4 ± 0.9
F ratio	3.1	2.8	2.9
Normal F ratio (Cuddon 1995)	1.75 ± 0.2	NR	1.80 ± 0.21

CMAP's amplitudes are expressed in mV, latencies in ms, distances in cm and conduction velocities in m/s. Distal stimulation: tibial nerve = hock, peroneal nerve = stifle, ulnar nerve = carpus; proximal stimulation: tibial and peroneal nerves = trochanteric fossa, ulnar nerve = elbow. NT = not tested. NR = not reported. Normal values are expressed as mean ± standard deviation.

information about the natural course of the disease as this case showed three reversible episodes of subacute tetraparesis within 1 year (Gerritsen et al 1996). Finally, very specific electrophysiological tests such as the study of late potentials (F wave) and the sensory potentials have been conducted in our case. The recording of those potentials is usually more difficult than motor potentials because the amplitude of the waves is in the range of the microvolts (mV).

Our case was presented with a very rapidly developing symmetric flaccid tetraparesis and markedly decreased flexor reflexes, but normal patellar reflexes, preserved sensation (persistence of conscious proprioception and normal skin sensation), and normal cranial nerve reflexes. This clinical picture is very similar to the acute IP described in dogs and Guillain-Barré syndrome (GBS) in humans (Cuddon 1998, Hughes and Cornblath 2005). In many

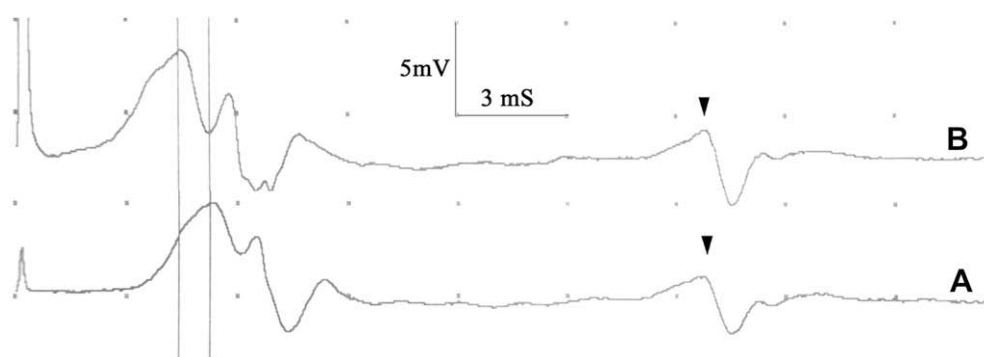


Fig 2. Motor nerve conduction study of the left peroneal nerve of the presented cat. The lower trace (A) shows the recording of potentials obtained after the proximal stimulation of the peroneal nerve and the upper trace (B) shows the recording after the distal stimulation. The first initial peaks that appear on the left of the traces are the stimulation artefacts. The next potentials represent the CMAPs of the peroneal nerve and the last potentials are the late potential or F wave (arrowheads). The amplitudes of the CMAP are reduced (7.5 mV for both the distal and proximal CMAP; normal CMAP amplitude – distal stimulation: 30.9 ± 6.6, normal CMAP amplitude – proximal stimulation: 29.0 ± 6.2) (Malik and Ho 1989). Note the polyphasic shape of the CMAPs. The vertical bars are positioned at the level of the first peak of each CMAP obtained in traces A and B. The distance between the proximal and distal stimulation points on the cat, divided by the time elapsed between the two vertical bars allow the calculation of the conduction velocity of the nerve impulse between the stimulation points, which was normal in this case (83 m/s, normal nerve conduction velocity: 88.3 ± 17.8) (Malik and Ho 1989).

animals affected by peripheral nerve diseases, the myotatic reflexes (as the patellar reflex) are preserved, whilst flexor reflexes become notably depressed, as in the case reported here. It has been proposed in human medicine, that the myotatic reflexes may be useful for the distinction between axonal and demyelinating polyneuropathy (patients with demyelinating disease have a significantly greater loss of their myotatic reflexes) (van Dijk et al 1999). In our case, the electrodiagnostic results were more indicative of an axonal type polyradiculoneuropathy, which may account for the preservation of the patellar reflexes. In previously described cats, the patellar reflex was recorded as absent in all the cases and post-mortem examination of two of the cases revealed a mixed involvement of the axons and the myelin (Gerritsen et al 1996).

The differential diagnosis in cats for acute progressive tetraparesis should include polyneuritis of possibly immune or infectious origin, polyneuropathy resulting from exposure to a toxin or polymyopathy, including that associated with electrolyte disturbance such as hypokalaemia. In cats, only two case reports of polyneuritis have been published (Lane and deLahunta 1984, Malik et al 1991). Although one would be tempted to include lymphoma in the differential diagnosis, this disease has not been associated with IP in animals. In humans, immune mechanisms triggered by lymphoma (paraneoplastic neuropathy) may initiate damage to the peripheral nervous system (Magne et al 2005, Wanschitz et al 2006).

Electrodiagnostic tests are crucial in precisely localising the site of a peripheral nerve lesion (ie, nerve root, axonal, axon terminals, neuromuscular junction, etc) and guide the clinician regarding the value of obtaining a peripheral nerve biopsy. If the disease affects only the nerve roots, the histopathological diagnosis from the peripheral nerve is frequently non-specific and no help in the diagnosis (Gerritsen et al 1996). Therefore, electrodiagnostic tests, which are minimally invasive, are of far greater value. The electrodiagnostic findings obtained on the presented cat were very similar to those reported in dogs and in acute motor axonal neuropathy in humans, which is one of the three main subtypes of GBS (Cuddon 1998, Hughes and Cornblath 2005). In our case, abnormal spontaneous electrical activity recorded in the muscles, combined with decreased amplitude of the CMAP suggest axonal loss. The nerve conduction velocities, which reflect the integrity of the myelin sheath

of the peripheral nerves, were only moderately reduced. Those findings, therefore, demonstrate a balance towards greater axonal than demyelinating lesions.

In dogs, the distal peripheral nerve trunks and the dorsal nerve roots (sensory) are much less severely affected than ventral nerve roots (motor) (Cummings and Haas 1966). The sensory potentials are usually easily recorded and have normal conduction velocity (Cuddon 1998), as obtained in the presented case for the sensory potential of the peroneal nerve. The study of the late potential (F wave) provides definitive evidence of the involvement of the nerve roots. The F wave represents a purely motor event and is a means of assessing the ventral nerve root (motor). In peripheral nerve diseases, the F wave latency is increased, as observed in our case. To assess the relative distribution of nerve pathological findings (proximal versus distal versus equal distribution), the F ratio can be used. The F ratio is obtained with a specific formula that basically divides the latency of the F wave by the latency of the M wave. If the ratio is greater than the reference value, it indicates more severe involvement of the proximal segment of the nerve, as demonstrated in our case.

GBS in humans is thought to be immune-mediated, based on epidemiological, histological and experimental results and has been associated with recent infection with *Campylobacter jejuni*, vaccination or following surgical procedures (Hughes et al 1999, Hughes and Cornblath 2005). Preliminary data in dogs have failed to demonstrate a clear association between the disease and an underlying infectious cause. No suggestive historical information was found in the history and clinical examination of the presented cat (Murray et al 2002). In dogs and humans, an albumino-cytological dissociation is observed with analysis of lumbar CSF but the presented cat has had a cisternal sample which had a normal total protein concentration (Murray and Cuddon 2002, Hughes and Cornblath 2005).

IP has a good prognosis in dogs and humans (Cuddon 1998, Hughes and Cornblath 2005). In previous reports in cats, the initial neurological progression reached a peak at 3 days, with spontaneous remission within 4–6 weeks, which is a very similar course to our cat (Gerritsen et al 1996). In the series of Gerritsen et al (1996), seven out of nine cats recovered completely. Relapses can occur in dogs but occur more rarely in cats (Gerritsen et al 1996). A chronic relapsing polyradiculoneuritis has been described in a cat

presenting abnormal high stepping gait, ataxia, muscle twitching and loss of sensory perception interrupted by several episodes of temporary remission. This condition differs from our case where the motor function was primarily affected (Flecknell and Lucke 1978). During the recovery period, support to eat and drink, manual expression of the bladder and prevention of pressure sores are required. Respiratory complications can also be seen in this condition and should be monitored during the recovery period.

IP in cats is a rapidly progressive disease leading to flaccid tetraparesis or tetraplegia, decrease or loss of spinal reflexes, but usually remarkably preserved sensation. The clinical signs are not characteristic but refer to a generalised neuromuscular disease. Electrodiagnostic tests help to precisely localise the lesion, although it does not provide an aetiological diagnosis. Thus, the diagnosis is based on combining the results of the neurological examination, the electrophysiological findings, and possibly the history of a recurrent disease. This disease should be recognised by veterinarians as it exhibits a marked discrepancy between the severity of the clinical signs and the overall good prognosis, although the early prognosis may be guarded because of possible respiratory failure. Because complete recovery is expected, it is of major importance for the animal to base the prognosis on the combination of the electrodiagnostic findings and natural evolution of the disease and not on the initial dramatic neurological signs.

Acknowledgement

This study was supported in part by a grant from the RCVS Trust.

Supplementary material

Supplementary material associated with this article can be found in the online version at doi: [10.1016/j.jfms.2008.03.008](https://doi.org/10.1016/j.jfms.2008.03.008).

References

Cuddon PA (1995) Assessment of nerve root function in neurologic disease (abstract) In: *Proceedings of the ACVIM 13th Annual Vet Med Forum*, pp. 913–917.

- Cuddon PA (1998) Electrophysiologic assessment of acute polyradiculoneuropathy in dogs: comparison with Guillain–Barre syndrome in people. *Journal of Veterinary Internal Medicine* **12** (4), 294–303.
- Cuddon PA (2002) Electrophysiology in neuromuscular disease. *Veterinary Clinics of North America* **32** (1), 31–62.
- Cummings JF, Haas DC (1966) Coonhound paralysis. An acute idiopathic polyradiculoneuritis in dogs resembling the Landry–Guillain–Barre syndrome. *Journal of Neurological Science* **4** (1), 51–81.
- Flecknell PA, Lucke VM (1978) Chronic relapsing polyradiculoneuritis in a cat. *Acta Neuropathologica* **41** (1), 81–84.
- Gerritsen RJ, van Nes JJ, van Niel MH, van den Ingh TS, Wijnberg ID (1996) Acute idiopathic polyneuropathy in nine cats. *Veterinary Quarterly* **18** (2), 63–65.
- Hughes RA, Cornblath DR (2005) Guillain–Barre syndrome. *Lancet* **366** (9497), 1653–1666.
- Hughes RA, Hadden RD, Gregson NA, Smith KJ (1999) Pathogenesis of Guillain–Barre syndrome. *Journal of Neuroimmunology* **100** (1–2), 74–97.
- Kimura J (2001) Types of electromyographic abnormalities. In: Kimura J (ed), *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. New York: Oxford, pp. 339–369.
- Knecht CD, Redding RW, Wilson S (1985) Characteristics of F and H waves of ulnar and tibial nerves in cats: reference values. *American Journal of Veterinary Research* **46** (4), 977–979.
- Lane JR, deLahunta A (1984) Polyneuritis in a cat. *Journal of the American Animal Hospital Association* **20**, 1006–1008.
- Magne N, Foa C, Castadot P, Otto J, Birtwisle-Peyrottes I, Thyss A (2005) Guillain–Barre syndrome and non-Hodgkin's lymphoma. Report of one case and review of literature. *Review Medicale de Bruxelles* **26** (2), 108–111.
- Malik R, Ho S (1989) Motor nerve conduction parameters in the cat. *Journal of Small Animal Practice* **30**, 396–400.
- Malik R, France MP, Churcher R, Mc Donald B (1991) Prednisolone responsive neuropathy in a cat. *Journal of Small Animal Practice* **32**, 529–532.
- Murray M, Cuddon PA (2002) Cerebrospinal fluid analysis in acute canine polyradiculoneuritis: albumin quotient and immunoglobulin G index determination using polyacrylamide gel electrophoresis (abstract). *Journal of Veterinary Internal Medicine* **16** (3).
- Murray M, Cuddon PA, Lappin MR (2002) Seroprevalence of various infectious agents in dogs with acute canine polyradiculoneuritis (abstract). *Journal of Veterinary Internal Medicine* **16** (3), 370.
- Redding RW, Ingram JT (1984) Sensory nerve conduction velocity of cutaneous afferents of the radial, ulnar, peroneal, and tibial nerves of the cat: reference values. *American Journal of Veterinary Research* **45** (5), 1042–1045.
- van Dijk GW, Wokke JH, Notermans NC, van Gijn J, Franssen H (1999) Diagnostic value of myotactic reflexes in axonal and demyelinating polyneuropathy. *Neurology* **53** (7), 1573–1576.
- Wanschitz J, Dichtl W, Budka H, Loscher WN, Boesch S (2006) Acute motor and sensory axonal neuropathy in Burkitt-like lymphoma. *Muscle and Nerve* **34** (4), 494–498.