

NOTE *Internal Medicine***Usefulness of Combined Electrophysiological Examinations for Detection of Neural Dysfunction in Cats with Lumbar Hematomyelia**Seiichi OKUNO¹⁾, Takayuki KOBAYASHI¹⁾ and Kensuke ORITO^{2)*}¹⁾*Animal Clinic Kobayashi, 715-1, Sakai, Fukaya, Saitama 366-0813, and* ²⁾*Department of Veterinary Pharmacology, School of Veterinary Medicine, Azabu University, Fuchinobe, Sagamihara, Kanagawa 229-8501, Japan*

(Received 23 March 2005/Accepted 8 August 2005)

ABSTRACT. We conducted combined electrophysiological examinations including F-wave, motor nerve conduction velocity (MNCV), spinal cord-evoked potential (SCEP), and needle electromyography (EMG) in two cats involved in traffic accidents that consequently developed hind limb paralysis caused by lumbar hematomyelia. F-wave could no longer be elicited within 3 days after the accident, and the MNCV and compound muscle action potential (CMAP) amplitude decreased in a time-dependent manner, with CMAP no longer being evoked after 7 or 8 days. EMG showed abnormalities such as fibrillation and positive sharp waves after 6 to 8 days. These results suggest that such combined electrophysiological examinations may provide objective, quantitative data for motor nerve dysfunction in cats with lumbar hematomyelia.

KEY WORDS: electrophysiological examinations, feline, hematomyelia.

J. Vet. Med. Sci. 67(12): 1265-1268, 2005

Hematomyelia is caused by a fracture or luxation of the spine, or an extrusion of intervertebral disk material [1, 7, 8, 14]. Acute damage to the feline spinal cord of cats has been shown to evoke hemorrhage and neuron necrosis in the gray matter within 4 h, and cavitation of the central portion of the spinal cord has been observed a few months after the trauma [11, 16]. Motor neuron function has never recovered once complete motor neuron death has occurred in the gray matter of the lumbar spinal cord. Abnormalities in lower motor neurons (LMN) that are representative signs of hematomyelia with motor neuron necrosis are usually confirmed by conventional neurological examinations such as postural reactions and spinal reflexes. These yield, however, only a subjective estimation that is influenced by the individual ability and experience of the examiner, and they cannot objectively detect motor nerve dysfunction in detail. Electrophysiological examinations have proved to be feasible diagnostic tools for the assessment of functions not only of the central nervous system (CNS) but also of the peripheral nervous system [3-5, 13, 15]. However, there have been no reports on the usefulness of combined electrophysiological studies for detecting dysfunctions of the peripheral and central nervous systems in spinal cord injuries. In the present study, we conducted F-wave, motor nerve conduction velocity (MNCV), needle electromyography (needle EMG), and spinal cord-evoked potential (SCEP, Dorsum potential) in 2 cats involved in traffic accidents and that showed a lack of spinal reflexes in their hind limbs due to hematomyelia. The usefulness of such combined electrophysiological examinations for the detection of motor nerve dysfunctions was confirmed.

The care and handling of the animals were in accordance

with the Azabu University Animal Experiment Guidelines, April 2000. Electrophysiological examinations were conducted without general anesthesia in Case 1 since the animal showed no sensations in the caudal portion of the trunk and hind limbs. In Case 2 myelography and the electrophysiological examinations were performed simultaneously under general anesthesia with isoflurane (Isoflu, Dainippon Pharmaceutical, Osaka, Japan). Rectal temperature was maintained at $38.5 \pm 0.5^\circ\text{C}$ using circulating water-heating pads. All electrophysiological examinations were conducted with a polygraph (Neuropack MEB-5508, Nihon Koden, Tokyo, Japan). F-wave was conducted as described previously [10]. In the MNCV examination, the needles for electrical stimulation were positioned at two sites along the tibial nerve with the cathode placed distally. Electrodes for the recording and reference as well as the conditions of single electrical stimulation were the same as those for the F-wave examination. In SCEP, a disk electrode was positioned over the midline between the spinous process of the L5 and L6 vertebrae for recording, and the reference was placed 2 cm lateral from the recording electrode. Needle-tipped stimulating electrodes were positioned as in the F-wave examination. The electrical stimuli lasted 0.2 ms, and rectangular waves were applied at a rate of 3 Hz. The stimulating intensity was adjusted to produce visible flexions of a digit. The averaged data of 100 responses were used. Needle EMG was obtained using a concentric needle electrode inserted into the muscles of the right pelvis and hind limb, including the middle gluteal, quadriceps, cranial tibial, gastrocnemius, and interosseous muscles.

Case 1: A 2-year-old male mix-breed cat involved in a traffic accident the previous day was referred to us with no voluntary movement in both hind limbs. No postural reactions such as conscious proprioception or any spinal reflexes including flexor, gastrocnemius, cranial tibial, or patellar reflexes were obtained. No acral pain sensation was

* CORRESPONDENCE TO: ORITO, K., Department of Veterinary Pharmacology, School of Veterinary Medicine, Azabu University, Fuchinobe, Sagamihara, Kanagawa 229-8501, Japan.

observed. These results suggested paralysis of both hind limbs. A fracture or luxation of the spine could not be identified with radiography. We started to treat this cat with medication of prednisolone and enrofloxacin (Baytril, Bayer Medical, Tokyo, Japan).

Electrophysiological examinations were performed 2, 4, 5, 6, and 8 days after the accident. No F-waves could be elicited throughout the study (Fig. 1). MNCV and compound muscle action potential (CMAP) amplitude were within the normal range 2 days after the accident, but then decreased in a time-dependent manner as shown in Table 1. Eight days after the accident, CMAP could no longer be evoked. In a needle EMG examination, potentials indicating denervation such as fibrillation and positive sharp waves were recorded in all muscles examined 6 and 8 days, and 8 days after the accident, respectively (Fig. 2). SCEP could not be elicited throughout the examination. Myelography under general anesthesia 8 days after the accident revealed intramedullary swelling of the lumbar spinal cord between L3 and L6 vertebrae, without spinal cord compression due to extradural material. A spinal cord hemorrhage between L5 and L6 was confirmed by hemilaminectomy. Clinical signs were not improved by medication and hemilaminectomy.

Case 2: A one-year-old castrated male mix-breed cat involved in a traffic accident the previous day was referred to us with a lack of voluntary movement in both hind limbs. Paralysis of both hind limbs was revealed by neurological examinations. A fracture or luxation of the spine could not be identified with radiography. We treated this cat with prednisolone. However, clinical signs did not improve. Three days after the accident, F-waves and SCEP could not be evoked. MNCV and CMAP amplitude of the distal portion of the tibial nerve were within the normal range (Table 1). Myelography revealed lumbar spinal cord swelling between the L3 and L6 vertebrae without compression due to extradural material. Seven days after the accident, F-wave, MNCV, and SCEP could not be elicited. In a needle EMG examination, fibrillation and positive sharp waves were found in all muscles examined (Table 1). Hemilaminectomy of the lumbar spine from L4 to L6 revealed swelling and a lack of blood flow on the surface of the spinal cord. No disk material was found in the vertebral canal. Hemorrhage in the gray matter of the lumbar spinal cord was revealed on autopsy.

F-wave has proven advantageous for the evaluation of the conductivity and neuron activity in proximal portions of the motor nerves since the waves occur after impulses have traveled the entire length of the motor nerves. F-wave conduction velocity and F-wave persistence (occurrence rate) reflect the function of the motor nerve conductivity and motor neuron activity [3, 6]. MNCV is a feasible index for the evaluation of conductive function in the distal portion and limited areas of the motor nerves. Needle EMG showing distinctive potentials, such as fibrillation or positive sharp waves, are directly linked with muscle denervation [17]. SCEP reflects the sensory tract functions of the spinal

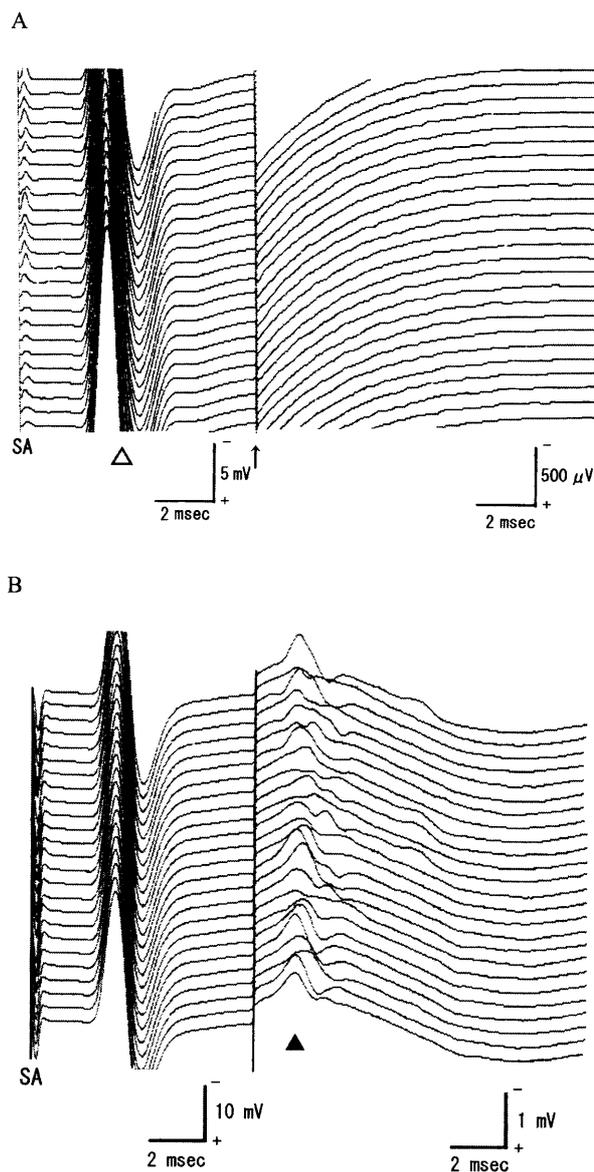


Fig. 1. A. F-wave examination 2 days after a traffic accident in Case 1. Recording of 25 stimuli in raster mode revealed that F-wave could not be elicited, i.e., F-wave persistence was 0%. Note that the recording sensitivity changed (arrow) after obtaining the compound muscle action potential (CMAP, white triangle). SA is an artifact. A negative polarity is indicated by an upward deflection. B. F-waves obtained from a clinically normal cat (black triangle).

cord [4]. Although these electrophysiological examinations have been widely used in veterinary medicine [2, 4, 9], there have been no reports on the usefulness of combining the above electrophysiological studies for the detecting dysfunctions of the peripheral and central nervous systems in spinal cord injuries.

In the present study, an F-wave could not be evoked in either cat 2 or 3 days after their accidents, but all the electro-



Fig. 2. Needle electromyography (EMG) in the gastrocnemius muscle in Case 1 showed abnormal muscle potentials with a positive sharp wave (black triangle) and fibrillation (white triangle) 8 days after the traffic accident. A negative polarity is indicated by an upward deflection.

Table 1. Changes in electrophysiological parameters in two cats after their traffic accidents

A. Case 1

Days after traffic accident	2	4	5	6	8
F-wave	Not elicited				
MNCV	75.0 m/sec	65.6 m/sec	51.9 m/sec	50.0 m/sec	Not elicited
CMAP amp	42.0 mV	38.0 mV	14.5 mV	1.6 mV	Not evoked
EMG	Normal	Normal	Normal	Fib	Fib PSW
SCEP	Not elicited				

B. Case 2

Days after traffic accident	3	7
F-wave	Not elicited	Not elicited
MNCV	95.0 m/sec	Not elicited
CMAP amp	35.0 mV	Not elicited
EMG	Normal	Fib PSW
SCEP	Not elicited	Not elicited

MNCV; motor nerve conduction velocity

CMAP amp; compound muscle action potential amplitude

EMG; electromyography

SCEP; spinal cord-evoked potential

Fib; fibrillation

PSW; positive sharp wave

physiological examinations, including F-wave, MNCV, and needle EMG, showed abnormalities after 6 or 7 days. In case 1, MNCV and CMAP amplitude decreased in a time-dependent manner, and CMAP was no longer evoked after 8 days. These results suggest that motor neuron dysfunctions in the spinal cord due to lumbar hematomyelia might occur within 3 days, and that denervation and wallerian degeneration in the distal portion of the motor nerve axons due to necrosis might occur between 3 and 6 days after a hematomyelia. Axon degeneration might lead to a decrease in CMAP amplitude, resulting in muscle inactivity and atrophy. Delay in the nerve conduction velocity might be caused by limb hypothermia due to muscle dysfunction [12]. Although conventional neurological examinations effectively reveal paralysis of both hind limbs, they do not provide objective information on nerve dysfunction.

Indeed, conventional neurological examinations performed on all days showed the same results, i.e. hind limbs paralysis, and never provide progressive pathophysiological conditions for the motor nerves. Electrophysiological examinations, however, should prove useful for quantitative evaluations of the central and peripheral nervous systems.

The present study revealed that F-wave abnormalities appeared earlier than those of needle EMG and MNCV in cats with hematomyelia. To our knowledge, this is the first report in the field of veterinary medicine to demonstrate that abnormality of neuronal activity in the spinal cord can be detected by F-wave examination, and that motor nerve dysfunctions in the central and peripheral nervous systems can be detected using repetitive, combined electrophysiological examinations.

REFERENCES

1. Bagley, R.S. 2000. *Vet. Clin. North. Am. Small. Anim. Pract.* **30**: 133–153.
2. Cuddon, P.A. 1998. *J. Vet. Intern. Med.* **12**: 294–303.
3. Fisher, M.A. 1992. *Muscle Nerve.* **15**: 1223–1233.
4. Holliday, T.A. 1992. *Vet. Clin. North. Am. Small. Anim. Pract.* **22**: 833–857.
5. Johnsen, B. and, Fuglsang-Frederiksen, A. 2000. *Neurophysiol. Clin.* **30**: 339–351.
6. Kimura, J. 1978. *Muscle Nerve.* **1**: 250–252.
7. Kornegay, J.N. 1991. *Probl. Vet. Med.* **3**: 363–377.
8. LeCouteur, R.A. 2003. *J. Feline Med. Surg.* **5**: 121–131.
9. Mizisin, A.P., Shelton, G.D., Burgers, M.L., Powell, H.C. and Cuddon, P.A. 2002. *J. Neuropathol. Exp. Neurol.* **61**: 872–884.
10. Okuno, S., Kobayashi, T. and Orito, K. 2002. *Am. J. Vet. Res.* **63**: 1262–1264.
11. Rawe, S.E., Lee, W.A. and Perot, P.L. 1978. *J. Neurosurg.* **48**: 1002–1007.
12. Rutkove, S.B. 2001. *Muscle Nerve.* **24**: 867–882.
13. Shahani, B.T. and Young, R.R. 1980. pp. 290–304. *In: Electrodiagnosis in Clinical Neurology* (Aminoff, M.J. ed.), Churchill Livingstone Co., New York.
14. Shores, A. 1992. *Vet. Clin. North. Am. Small. Anim. Pract.* **22**: 859–888.
15. Tonzola, R.F., Ackil, A.A., Shahani, B.T. and Young, R.R. 1981. *Ann. Neurol.* **9**: 305–308.
16. Wagner, F.C., Van Gilder, J.C. and Dohrmann, G.J. 1977. *Paraplegia* **14**: 245–250.
17. Wheeler, S.J. and Sharp, N.J.H. 1994. pp 54–55. *In: Small Animal Spinal Disorders: Diagnosis and Surgery.* Mosby-Wolfe, London.