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Tibial Nerve Somatosensory Evoked Potentials in Dogs with Degenerative Lumbosacral Stenosis

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Objective—To determine somatosensory evoked potentials (SEPs) in dogs with degenerative lumbosacral stenosis (DLS) and in healthy dogs.
Study Design—Clinical and experimental study.
Animals—Dogs with DLS (n = 21) and 11 clinically normal dogs, age, and weight matched.
Methods—Under anesthesia, the tibial nerve was stimulated at the caudolateral aspect of the stifle, and lumbar SEP (LSEP) were recorded percutaneously from S1 to T13 at each interspinous space. Cortical SEP (CSEP) were recorded from the scalp.
Results—LSEP were identified as the N1–P1 (latency 3–6 ms) and N2–P2 (latency 7–13 ms) wave complexes in the recordings of dogs with DLS and control dogs. Latency of N1–P1 increased and that of N2–P2 decreased as the active recording electrode was moved cranially from S1 to T13. Compared with controls, latencies were significantly delayed in DLS dogs: .8 ms for N1–P1 and 1.7 ms for the N2–P2 complex. CSEP were not different between groups.
Conclusions—Surface needle recording of tibial nerve SEP can be used to monitor somatosensory nerve function of pelvic limbs in dogs. In dogs with DLS, the latency of LSEP, but not of CSEP, is prolonged compared with normal dogs.
Clinical Relevance—In dogs with lumbosacral pain from DLS, the cauda equina compression is sufficient to affect LSEP at the lumbar level.
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Introduction

SOMATOSENSORY EVOKED POTENTIALS (SEPs) elicited by stimulation of the posterior tibial nerve in humans have been used as a noninvasive recording technique to assess nerve root and spinal cord function.[1–7] Sciatic nerve spinal SEP (SSEP) and cortical SEP (CSEP) have been recorded experimentally in dogs[8,9] and cats[10,11] involving thoracolumbar spinal cord injury caused by compression or impact forces. These experimental studies in mammals were performed to investigate the generators of the SEP and to test the usefulness of animal models to provide answers for SEP findings in human subjects with spinal cord, conus medullaris, and cauda equina diseases. The cauda equina has been investigated in experimental traction[12] or compression[13–19] studies in dogs. Delamarter et al[15] developed a dog model of lumbar spinal stenosis to study the pathophysiology of cauda equina syndrome. The cauda equina was constricted experimentally to simulate chronic compression. Dogs with 75% constriction of the diameter of the cauda equina had significant neurologic abnormalities. CSEP revealed abnormalities that preceded neurologic signs.[13–19]

SSEP and CSEP have been used in clinical studies for diagnosis and localization of spinal cord and cauda equina diseases in humans.[20–27] In patients with conus medullaris or cauda equina lesions (e.g., midline disc compression, trauma, tumor) configurational changes

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were found in SSEP before such changes were evident in standard electromyographs or nerve conduction velocity tests.[20,21,25] It was concluded that in cauda equina compression each major component of the SEP may be absent or the peak could have reduced amplitude and prolonged latency.

SSEP and CSEP after tibial or sciatic nerve stimulation have been recorded and described extensively in clinically normal dogs[28–35] and cats.[36,37] In contrast, reports on the use of SEP in clinical studies are rare. In dogs with acute compressive thoracolumbar spinal cord disease, caused by intervertebral disk extrusion, SEP recorded at T10–T11 differed significantly from a control group whereas L7–S1 recordings were not different between groups.[38] SEP were of value in determining prognosis for recovery after treatment of spinal injury because significant differences were found between the favorable and unfavorable outcome groups within the spinal injury group.[38] The potential usefulness of SEP in dogs with cauda equina compression was reported previously but more refinement and standardization was needed before being clinically useful.[39] Cuddon et al[35] concluded that evaluation of tibial nerve cord dorsum evoked potentials can be used to accurately assess functional severity and distribution of abnormalities in proximal sensory nerves, dorsal nerve roots, and spinal cord dorsal horns in dogs with suspected neuropathy, radiculopathy, or myelopathy involving the lumbosacral intumescences.

Our objective was to evaluate waveform, latencies, and amplitudes of lumbar SEP (LSEP) and CSEP in dogs with degenerative lumbosacral stenosis (DLS) and compare them with age and weight matched control dogs.

MATERIALS AND METHODS

Animals

SEP were recorded in dogs with DLS (n = 21) and age and weight matched, clinically normal dogs (n = 11).

DLS dogs (15 males, 6 females) were 6 Bouviers, 4 German Shepherds, 3 Labrador retrievers, 2 Rottweilers, 1 Bernese Mountain dog, 1 Dalmatian dog, 1 Airedale terrier, 1 Great Dane, 1 French Briard, and 1 cross breed. Body weight ranged from 22 to 70 kg (median, 33 kg) and age ranged from 1 to 13 years (median, 7 years). All dogs were referred with a history of cauda equina syndrome. Clinical signs were low back pain, lameness, muscle weakness, and muscle atrophy in one or both pelvic limbs. All dogs had pain upon the lumbosacral pressure test during lordosis of the caudal vertebral column. Normal postural reactions and spinal reflexes were present. Moderate-to-severe cauda equina compression at the lumbosacral region from DLS was confirmed with radiography, epidurography, and computed tomography (CT).

Control, normal dogs (8 males, 3 females) were 3 Belgian Shepherds, 1 German Shepherd, 3 Greyhounds, 1 Labrador retriever, 1 Bouvier, 1 Rottweiler, and 1 Boxer. Body weight ranged from 21 to 39 kg (median, 29 kg) and age ranged from 1 to 13 years (median, 7 years). Dogs had no history of low back pain, had no abnormalities during orthopedic and neurologic examination, and the lumbosacral area were considered normal on radiographs, with no signs or secondary changes associated with DLS. CT was not performed.

Anesthesia

SEP recordings were performed under anesthesia. Dogs were premedicated with 40–80 μg medetomidine/kg metabolic bodyweight. Anesthesia was induced with a single intravenous (IV) dose of 1–2 mg/kg propofol. Dogs were intubated and anesthesia was maintained with 4 mg propofol/kg/hour administered IV by an infusion pump. Electrocardiography, capnography, and pulse oximetry were used for monitoring. Body temperature was measured at regular intervals during each recording session. Infrared lamps and a warm-water bed were used to prevent a decrease in rectal temperature $>1^\circ$C. Dogs were positioned in right and subsequently left lateral recumbency for SEP recording.

Tibial Nerve Stimulation

The tibial nerve was stimulated, using 2 sensory needle electrodes (DISA 13L60, Danica Medical Instruments, Leusden, The Netherlands), placed at a depth of 1 cm caudal and proximal of the fibular head. Stimuli for SEP recording were square-wave pulses of .2 ms duration, applied at a rate of 1/s (1 Hz), generated by a stimulator (Model S-88, Grass Medical Instruments, Quincy, MA) and started by a computer signal. The stimuli were delivered to a Grass stimulation isolation unit (Model SUI 5, Grass Medical Instruments) and a constant current unit (Model CCU 1A, Grass Medical Instruments) controlling the stimulus intensity. The stimulus intensity was set at 8–12 V causing a clear, visually detectable, digital extension and tarsal flexion. After completion of SEP recordings following stimulation of the left tibial nerve, the dog was turned on the other side and recordings were repeated after stimulation of the right tibial nerve.

SEP Recording Technique and Data Acquisition

The dog and the examination table were grounded with separate electrodes during all measurements.

LSEP were recorded with 3 sensory needle electrodes. The indifferent electrode was placed ipsilateral to the stimulus side on the iliac crest near the bone and free of muscle. Another reference electrode was placed subcutaneously contralateral to the stimulus side on the iliac crest. The active recording electrode was placed in the dorsal midline between 2 spinous processes in the interspinous ligament near the interarcuate space and dorsal laminar bone. The active recording electrode was advanced from the lumbosacral region to the thoracolumbar region. Two measurements of LSEP were recorded at every junction from L7–S1 to T13–L1. The exact interverte-

bral space was localized by palpation of the spinous processes starting from L7.

CSEP were recorded with 3 sensory needle electrodes. The active recording electrode was placed contralateral to the stimulus side at the level of vertex (Cz) near the bone of the skull. The indifferent electrode was placed subcutaneously ipsilateral to the stimulus side in the neck halfway between the atlas and the thoracic inlet. Another reference electrode was placed subcutaneously ipsilateral to the stimulus side at the level of the shoulder.

The stimulus started data acquisition by the computer, functioning as an averager. The SEP response signal was preamplified, fed to a variable amplifier (total gain 20,000–100,000; frequency range 2 Hz–2 KHz) and to a 16-bit analog-to-digital converter, interfaced to the computer. The sampling rates were 10 KHz for LSEP and 5 KHz for CSEP and for both 512 data points were collected. Usually 16 to 64 measurements were averaged by the computer. When 2 independent recordings from each dog for each location on the vertebral column were repeatable, they were stored for further analysis. When recordings were not repeatable, they were discarded.

Using computer software, individual components of the LSEP and CSEP waveforms were identified and latencies and amplitudes of waveform peaks and troughs were measured. Negativity was up in all recordings.

Calculations and Statistics

The Fisher's Exact test was used to test for differences in proportions of identified waves between DLS and control groups.

At each location on the vertebral column, mean (± SEM) latency and mean amplitude was calculated from 4 recordings (2 from the left and 2 from the right side). Peak-to-peak amplitudes were calculated for the N1–P1-N2–P2 complex by adding up the absolute values of the maximum peak (N1 or N2) and trough (P1 or P2) deflections at each recording site. Thus, a value was obtained representing the total amplitude height of the N1–P1-N2–P2 complex of the SEP. Spearman's rank test for trend analysis was used to assess whether the latency of individual wave components had a tendency to increase or decrease as the distance between stimulation and recording increased.

The mean (± SEM) value for latency and amplitude was calculated from the values from T13 to S1, representing a mean value for the trend line graph of each wave component. Student's t-test for independent samples was used to compare means between the DLS and control groups. Statistical significance was assumed at $P < .05$.

RESULTS

Lumbar SEP

Waveform. LSEP were recorded between T13 and S1 (Figs 1A, B). At shorter latencies (0–15 ms) usually 2 prominent peaks were detected and they were labeled N1 and N2. Both peaks were each followed by deflections

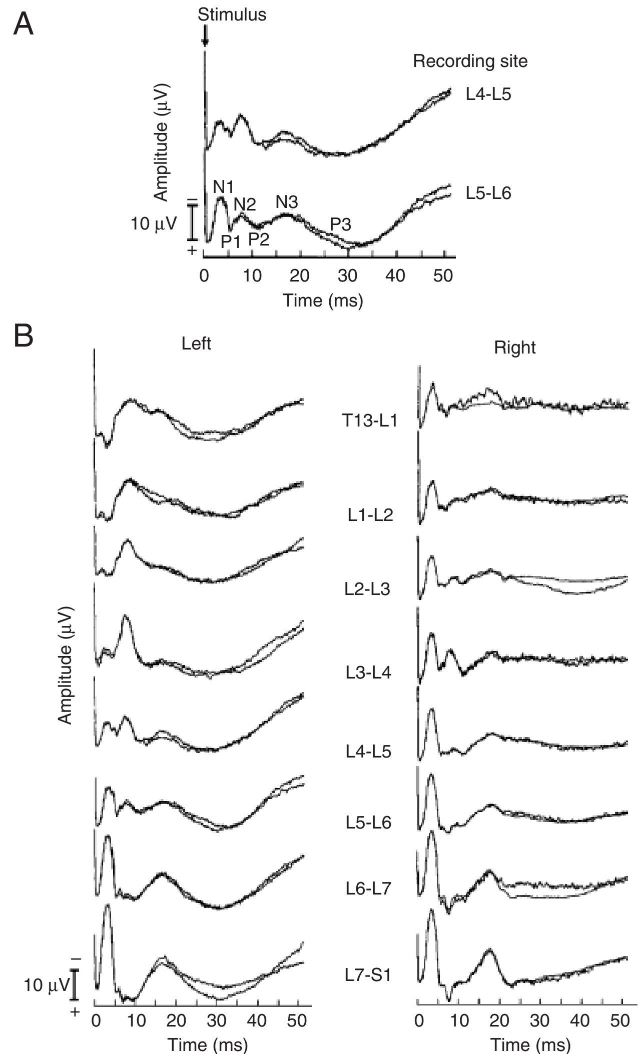

Fig 1. Representative example of a double series of tibial nerve lumbar somatosensory evoked potentials (LSEP) in a 7-year-old female Rottweiler with lumbosacral pain from degenerative lumbosacral stenosis. (A) Wave components of LSEP are marked N1, P1, N2, P2, N3, and P3. (B) LSEP were recorded between T13 and S1.

that were labeled as P1 and P2 (Fig 1A). The time span of the N1–P1 complex was approximately 5 ms and that of the N2–P2 complex 5–10 ms, and they were usually identified between L3 and S1. The N1–P1 complex disappeared in most recordings as the recording electrode moved cranially but was still identified in 20–30% of recordings at T13–L1 (Fig 1B). The N2–P2 complex was seen as a small wave embedded in the descending leg of N1 between L6 and S1 and became more prominent at L3–L4 (Fig 1B). Especially, the N2 peak was most prominent between L3 and L5 where the detection rate was maximum (80–100%). The identification percentage of the N2–P2 complex decreased to 20–30% between T13

and L3. The N1–P1 and N2–P2 complexes were equally identified in both control and DLS groups.

A third wave, the N3–P3 complex, was detected at longer latencies (15–50 ms) and its time span ranged from 15 to 30 ms (Figs 1A, B). The N3–P3 complex was identified more often in the control group than in the DLS group and the difference was significant at recording site L5–L6 (Fisher's Exact test, $P<.05$).

Wave latency. A trend was recorded for the latency of the N1–P1 complex to decrease and for the latency of the N2–P2 complex to increase as the active recording electrode was moved cranially from S1 to T13 (Fig 2). More specific, the latencies of N1 remained constant, whereas the latencies of P1 decreased by 1 ms (Fig 2). Change in latency of N1 was not significant for DLS or control groups. The decrease in latency of P1 was significant for the control group (Spearman's rank test, $r=-1.00$, $P<.001$) but not for the DLS group. The most prominent shift in latency occurred in the N2–P2 complex. The mean latencies of N2 and P2 increased by 1–2 ms as the active recording electrode was moved from S1 to T13 (Fig 2). The increase in latency of N2 was significant for both the DLS (Spearman's rank test, $r=1.000$, $P<.01$) and control groups (Spearman's rank test, $r=.833$, $P=.05$). The increase in latency of P2 was significant for the DLS group (Spearman's rank test $r=.929$, $P=.001$) but not for the control group.

The mean latency values were calculated (Table 1) and are a numeric representation of the mean (height level = latency) of each trend line graphed (Fig 2). Latencies of the N1–P1 and N2–P2 complexes were higher in the DLS group compared with the control group (Fig 2). The mean latency was significantly higher in the DLS group than in the control group: .8 ms for the N1–P1 complex and 1.7 ms for the N2–P2 complex (Student's t-test for independent samples, $P<.01$; Table 1).

Table 1. The Mean (± SEM) Latency and Amplitude of Tibial Nerve Lumbar Somatosensory Evoked Potentials (SEP) in Dogs with Degenerative Lumbosacral Stenosis (DLS) and Control Dogs

Lumbar SEP Wave Components	Latency* (ms)		Amplitude* (μV)	
	DLS (n = 21)	Control (n = 11)	DLS (n = 21)	Control (n = 11)
N1	3.8 ± 0.7[b]	3.5 ± 0.9	5.0 ± 0.3	5.2 ± 0.6
P1	5.6 ± 0.1[c]	4.8 ± 0.1	−1.3 ± 0.3[c]	0.2 ± 0.2
N2	8.6 ± 0.2[c]	7.4 ± 0.2	3.0 ± 0.3	2.8 ± 0.2
P2	13.0 ± 0.2[c]	11.3 ± 0.3	−3.0 ± 0.3[c]	−1.0 ± 0.4
N3	24.5 ± 0.7	24.4 ± 0.6	6.0 ± 0.5[b]	8.6 ± 0.6
P3	35.5 ± 0.9[c]	40.1 ± 0.6	−6.0 ± 0.9[a]	−8.9 ± 0.9
Peak-to-peak†			9.9 ± 0.4[c]	6.9 ± 0.8

*Latency and amplitude of values measured betweenT13 and S1.

†Sum of the absolute values of the maximum peak (N1 or N2) and trough (P1 or P2) deflections.

[a] $P<.05$,

[b] $P<.01$, and

[c] $P<.001$ compared with control value, Student's t-test for independent values.

The N3–P3 complex of the LSEP occurred between latencies 24 and 40 ms for the DLS and control group. The latency of N3 decreased significantly (Spearman's rank test $r=-.714$, $P<.05$) for the control group as the active recording electrode was moved cranially. The mean latency of N3 was 24 ms for both control and DLS groups. The mean latency of P3 was significantly lower for the DLS group than for the control group (Student's t-test for independent samples, $P<.001$; Table 1).

Wave amplitude. The mean amplitudes of N1 were maximal at L7–S1 (11–12 μV) and gradually decreased as the active recording electrode moved cranially to T13 (1–3 μV; Figs 1, 3). The mean amplitudes of N2 were minimal at L7–S1 (1–2 μV) and gradually increased as the recording electrode moved cranially, reaching its maximum at L3–L5 (4 μV), and remaining constant at T13–L3 (Figs 1, 3). The mean P1 and P2 amplitudes, but not mean N1 and N2 amplitudes, were significantly different between the control group and the DLS group (Student's *t*-test for independent samples, $P<.001$; Table 1).

Peak-to-peak amplitudes decreased significantly for the control group (Spearman's rank test $r=-.976$, $P<.01$) as the active recording electrode moved cranially from S1 to T13 (Fig 4). The mean peak-to-peak amplitude of the N1–P1–N2–P2 complex was significantly higher in the DLS group than in the control group (Student's t-test for independent samples, $P<.001$; Table 1).

The mean amplitude for the N3 and P3 wave components were significantly higher (2–3 μV) for the control

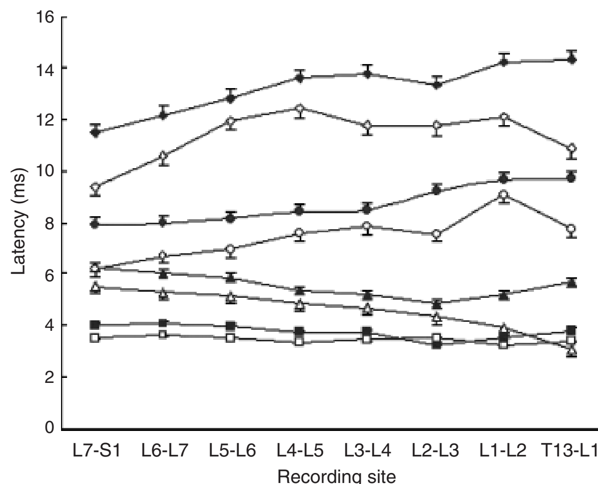

Fig 2. The mean (± SEM) latencies (ms) of wave components of tibial nerve LSEP recorded between T13 and S1 in 21 dogs with degenerative lumbosacral stenosis [N1 (—■—), P1 (—▲—), N2 (—●—), P2 (—◆—)] and in 11 control dogs [N1 (—□—), P1 (—△—), N2 (—○—), P2 (—◇—)].

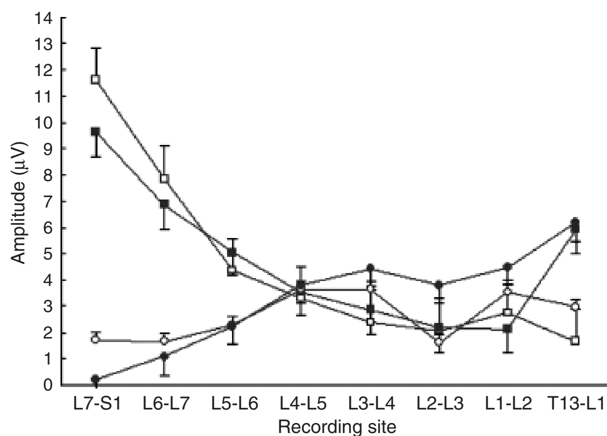

Fig 3. The mean (± SEM) amplitudes (µV) of wave components N1 and N2 of tibial nerve LSEP recorded between T13 and S1 in 21 dogs with degenerative lumbosacral stenosis [N1 (—■—), N2 (— • —)] and in 11 control dogs [N1 (—□—), N2 (— ○ —)].

group than for the DLS group (Student's t-test for independent samples, $P < .05$; Table 1).

Cortical SEP

A prominent CSEP wave complex with a 30 ms duration, labelled N2–P2, was most commonly detected between 20 and 50 ms after stimulation (Fig 5). Less common was a small complex N1–P1 of 10 ms duration, preceding N2–P2, and a broad complex N3–P3 of 50 ms duration, following N2–P2. The N1–P1 complex of the CSEP was recorded significantly more often (Fisher's Exact test, $P < .05$) in the control group (91%) than in the DLS group (57%) which was not the case for N2–P2 and N3–P3 complexes.

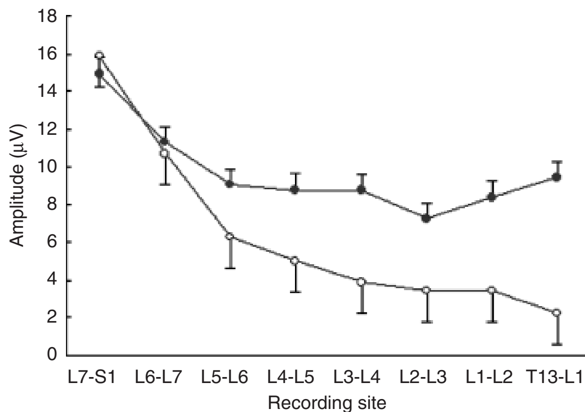

Fig 4. The mean (± SEM) peak-to-peak amplitudes (i.e., the sum of absolute amplitude values of the most positive deflection and the most negative deflection) of the wave complex (N1–P1–N2–P2) in tibial nerve LSEP, recorded between T13 and S1, in 21 dogs with degenerative lumbosacral stenosis (— • —) and in 11 normal dogs (— ○ —).

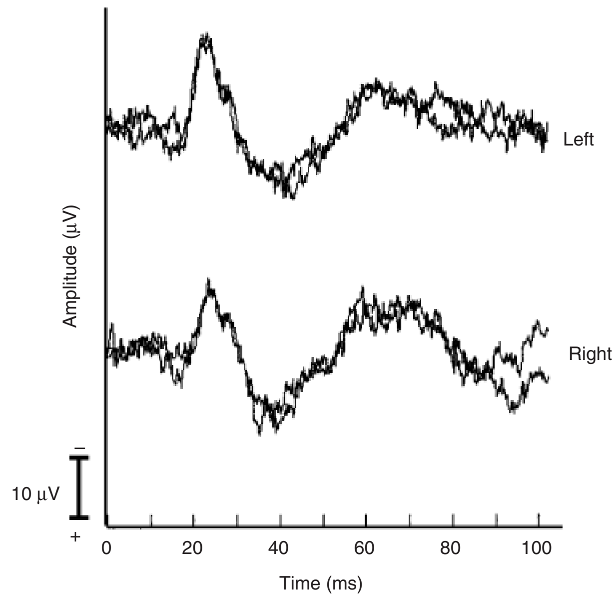

Fig 5. Representative example of a series of tibial nerve cortical SEP, recorded from the scalp vertex, in a 7-year-old female Rottweiler with lumbosacral pain from degenerative lumbosacral stenosis.

The mean latencies and amplitudes of tibial nerve CSEP were not different between the control and DLS groups (Table 2).

DISCUSSION

We found that the latencies of tibial nerve SEP recorded from the surface in dogs with cauda equina compression, were prolonged compared with those in control dogs.

Table 2. The Mean (± SEM) Latency and Amplitude of Tibial Nerve Cortical Somatosensory Evoked Potentials (SEP) Measured from the Scalp in Dogs with Degenerative Lumbosacral stenosis (DLS) and Control Dogs

Cortical	Latency (ms)		Amplitude (µV)	
SEP Wave Components	DLS (n = 21)	Control (n = 11)	DLS (n = 21)	Control (n = 11)
N1	11.6 ± 0.6	12.6 ± 0.5	1.0 ± 0.3	1.4 ± 0.3
P1	18.8 ± 0.9	18.9 ± 0.5	−1.2 ± 0.2	−1.3 ± 0.4
N2	28.5 ± 1.4	29.7 ± 1.0	1.9 ± 0.2	2.3 ± 0.5
P2	43.7 ± 2.2	47.4 ± 1.7	−2.0 ± 0.3	−2.0 ± 0.3
N3	66.4 ± 3.5	69.0 ± 1.9	2.4 ± 0.3	2.9 ± 0.7
P3	88.4 ± 1.7	86.7 ± 3.2	−2.0 ± 0.6	−1.8 ± 0.3
Peak-to-peak*			4.0 ± 0.4	4.6 ± 0.6

*Sum of the absolute values of the maximum peak (N1 or N2) and trough (P1 or P2) deflections.

Tibial nerve SEP recorded over the lumbosacral and caudal lumbar area reflect the quantity and timeliness of arrival of the afferent volley (sensory fibers) as well as the responsiveness of the pool of spinal neurons and interneurons in synaptic connection with the afferent fibers in the lumbosacral enlargement of the spinal cord.[6,7] The first major negative wave of the SSEP reflects the ascending volley of impulses in the dorsal roots, and the second negative wave reflects segmental postsynaptic activity and activity of spinal cord interneurons.[1–3,5,7,25] The configuration of lumbosacral SEP in humans[1–3,5] resembles the N1–P1–N2–P2 wave complex we recorded in dogs; however, the latencies of corresponding waves are different between humans and dogs. In dogs, latencies were 3–4 ms for N1 and 6 ms for N2 at L7–S1, and 7 ms for N2 at L4–L5, which is 35%, 50%, and 55%, respectively, of their human counterparts. There is a linear relation between latencies of tibial nerve SEP and body size in healthy dogs.[34] Therefore, smaller LSEP latencies in dogs compared with humans were most likely related to difference in body size. Moreover, the ascending action potential enters the spinal cord at L6 in dogs (compared with L1 in humans), possibly contributing to shorter latencies for the spinal cord dorsum potential (N2) in dogs.

LSEP and CSEP recorded in control dogs were similar in waveform, latency and amplitude as those previously reported in healthy dogs.[8,28–35] Tibial nerve LSEP was reported to consist classically of a cauda equina (dorsal root) evoked potential, best recorded as a negative peak between L7 and S1 (latency 3–4 ms), and a cord dorsum evoked potential, best recorded as a negative peak between L3 and L6 (latency of 6 ms).[28,30,32,33,35] We identified the third LSEP wave complex detected at longer latency that remained largely unchanged when moving the active recording electrode from S1 to T13, whereas its amplitude was strongly dependent on an undisturbed recording electrode configuration. The N3–P3 wave complex was a far-field potential generated by a change in volume conductor circumference and orientation, i.e., transition from pelvic limb to the trunk of the dog.[40] The DLS and control dogs were comparable in body conformation and thus generated a similar N3–P3 far-field volume-conducted evoked potential.

The LSEP in healthy dogs may be used to accurately assess functional severity and distribution of abnormalities in proximal sensory nerves, dorsal nerve roots, and spinal cord dorsal horns in dogs with suspected neuropathy, radiculopathy, or myelopathy involving the lumbosacral intumescences.[35] In human clinical study investigating LSEP and CSEP in patients with lesions of the conus medullaris and cauda equina it was concluded that each major component of the SEP can be absent or the peak can have a reduced amplitude and a prolonged latency.[25] The degree of impairment of the SEP was correlated with the degree of severity of the cauda equina lesion. Recording of SEP with surface electrodes represented a reliable test for the detection of mild cauda equina abnormalities.[25]

In an experimental dog model of lumbar spinal stenosis developed by Delamarter et al,[15] relative degrees of constriction of the cauda equina were performed by inserting a nylon constriction device. Dogs with 25% constriction of the caudal equina had no neurologic deficits and only mild changes in CSEP; those with 50% constriction had mild initial motor weakness and major changes in CSEP, and those with 75% constriction had paralysis of the tail, urinary incontinence, and dramatic changes in CSEP.[15] Cystometric measurements were noted to become a flat line with 75% of compression.[13,14] Constriction of the cauda equina at 50% was the critical point, resulting in loss of SEP, reflexes, neurologic deficits, and histologic abnormalities on necropsy examination of the cauda equina.[17,18]

Clinical signs of dogs with DLS we report may be best compared with those of dogs with 25% constriction of the cauda equina in the model of Delamarter et al.[15] None of our dogs had clinical neurologic deficits; however, we were able to detect significant SEP abnormalities in the DLS group compared with the control group. Latencies of the N1–P1 wave complex (reflecting the ascending volley of impulses in the dorsal roots), and latencies of the N2–P2 wave complex (reflecting postsynaptic activity and activity of the spinal cord interneurons) were prolonged in the DLS group compared with the control group. In another report where 26 dogs with cauda equina compression were investigated, latencies of tibial nerve SEPs and nerve conduction velocity were "largely normal" but it was stated that the recording technique needed more refinement and standardization.[39] Using the technique we report, future clinical studies using LSEP measurements may be used to confirm left or right-sided cauda equina compression, to differentiate between spinal cord and cauda equina diseases, to monitor the recovery of nerve function after decompressive surgery, and to determine the prognostic value of treatment in relation to outcome.

Histological changes in nerve roots from mechanical compression are nerve root edema, loss of myelin, and Wallerian degeneration of the motor nerve roots distal to the constriction and of the sensory nerve roots proximal to the constriction.[17,18,41] These changes in nerve roots may be responsible for the prolonged LSEP latencies we observed. The compressive lesion of the cauda equina at L7–S1 induced a delay of the ascending volley of impulses in the afferent sensory pathways in the dorsal roots, and an additional indirect delay at the level of the interneurons and postsynaptic pathways in the lumbosacral en-

largement of the spinal cord. The delay at the interneuron level may be caused by an imbalance between inhibitory and stimulatory interneurons set-off by the incoming disturbed and delayed ascending action potential.[42] This might have resulted in an increased activity at the interneuron level (more impulses in feedback loops), thereby delaying the start of the final compound action potential to ascend the spinal cord.[42] Increased activity at the interneuron level may explain an increased amplitude of the evoked potential at this level. Indeed, we found greater peak-to-peak amplitudes for the N1–P1-N2–P2 complex in the DLS group than in the control group.

Controversies still exist about the number and cerebral location of the source generators of the CSEP after tibial nerve stimulation.[43] The thalamus generates a wave component at 30 ms of latency and the contralateral parietal somatosensory cortex generates 1 or 2 wave components at 40 and 50 ms of latency after tibial nerve stimulation in humans.[43] The wave conformation of the CSEP in the control dogs in our study resembled the human CSEP equivalent.[43] There were no significant differences between CSEP latencies of the DLS and control groups. The initial delay in latency because of a compressive lesion on the cauda equina at L7–S1 in the DLS group was significant at low lumbar levels but at longer latencies it became relatively smaller and insignificant at the somatosensory cerebral cortex. This is in agreement with the experimental dog model described by Delamarter et al where 25% cauda equina constriction resulted in only mild changes in CSEP.[13–15] Apparently, in the dogs with DLS that have no neurologic signs, cauda equina compression was sufficient to affect latency of SEP at the lumbar level, but not at the cerebral level.

We concluded that surface recording of tibial nerve SEP is a valuable technique to monitor somatosensory nerve function of pelvic limbs in dogs. Latencies of LSEP were prolonged in dogs with DLS and cauda equina compression compared with those in control dogs. LSEP are a useful contribution to the diagnostic investigation of DLS and may be used to evaluate the recovery of nerve function after decompressive surgery.

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REFERENCES

1. Delbeke J, McComas AJ, Kopec SJ: Analysis of evoked lumbosacral potentials in man. J Neurol Neurosurg Psychiatry 41:293–302, 1978
2. Dimitrijevic MR, Larsson LE, Lehmkuhl D, et al: Evoked spinal cord and nerve root potentials in humans using a non-invasive recording technique. Electroencephalogr Clin Neurophysiol 45:331–340, 1978
3. Philips II LH, Daube JR: Lumbosacral spinal evoked potentials in humans. Neurology 30:1175–1183, 1980
4. Seyal M, Emerson RG, Pedley TA: Spinal and early scalp-recorded components of the somatosensory evoked potential following stimulation of the posterior tibial nerve. Electroencephalogr Clin Neurophysiol 55:320–330, 1983
5. Yiannikas C, Shahani BT: The origins of lumbosacral spinal evoked potentials in humans using a surface electrode recording technique. J Neurol Neurosurg Psychiatry 51:499–508, 1988
6. Chiappa KH: Short-latency somatosensory evoked potentials: methodology, in Chiappa KH (ed): Evoked Potentials in Clinical Medicine. New York, NY, Raven Press, 1990, pp 320–330
7. Jeanmonod D, Sindou M, Mauguière F: The human cervical and lumbo-sacral evoked electrospinogram. Data from intra-operative spinal cord surface recordings. Electroencephalogr Clin Neurophysiol 80:477–489, 1991
8. Parker AJ: Evoked cisterna cerebomedullaris potentials in the clinically normal dog. Am J Vet Res 39:1811–1815, 1978
9. Parker AJ: Comparison of the averaged evoked cisterna cerebellomedullaris potential, cortical potential, and neurologic examination in the diagnosis of spinal cord injury of the dog. Am J Vet Res 39:1816–1821, 1978
10. Schramm J, Hashizume K, Fukushima T, et al: Experimental spinal cord injury produced by slow, graded compression. J Neurosurg 50:48–57, 1979
11. Schramm J, Krause R, Shigeno T, et al: Experimental investigation on the spinal cord evoked injury potential. J Neurosurg 59:485–492, 1983
12. Fujita Y, Yamamoto H: An experimental study on spinal cord traction effect. Spine 14:698–705, 1989
13. Bodner DR, Delamarter RB, Bohlman HH, et al: Urologic changes after cauda equina compression in dogs. J Urol 143:186–190, 1990
14. Delamarter RB, Bohlman HH, Bodner D, et al: Urologic function after experimental cauda equina compression. Cystometrograms versus cortical-evoked potentials. Spine 15:864–870, 1990
15. Delamarter RB, Bohlman HH, Dodge LD, et al: Experimental lumbar spinal stenosis. Analysis of the cortical evoked potentials, microvasculature, and histopathology. J Bone Joint Surg Am 72:110–120, 1990
16. Delamarter RB, Sherman JE, Carr JB: 1991 Volvo Award in experimental studies. Cauda equina syndrome: neurologic recovery following immediate, early, or late decompression. Spine 16:1022–1029, 1991
17. Kim NH, Yang IH, Song IK: Electrodiagnostic and histologic changes of graded caudal compression on cauda equina in dog. Spine 19:1054–1062, 1994
18. Kim NH, Yang IH: A study of motor and sensory evoked potentials in chronic cauda equina compression of the dog. Eur Spine J 5:338–344, 1996
19. Wada N, Akatani J, Shikaki N, et al: Somatosensory evoked potentials produced by stimulation of the dorsomedial nerves innervating the tail in dogs. J Vet Med Sci 64:251–253, 2002

20. Ertekin C, Mutlu R, Sarica Y, et al: Electrophysiological evaluation of the afferent spinal roots and nerves in patients with conus medullaris and cauda equina lesions. J Neurol Sci 48:419–433, 1980
21. Wexler I: Spinal evoked potentials in the diagnosis of cauda equina compression. Electromyogr Clin Neurophysiol 22:521–524, 1982
22. Ertekin C, Sarica Y, Uckardesler L: Somatosensory cerebral potentials evoked by stimulation of the lumbo-sacral spinal cord in normal subjects and in patients with conus medullaris and cauda equina lesions. Electroencephalogr Clin Neurophysiol 59:57–66, 1984
23. Lehmkuhl LD, Dimitrijevic MR, Renouf F: Electrophysiological characteristics of lumbosacral evoked potentials in patients with established spinal cord injury. Electroencephalogr Clin Neurophysiol 59:142–155, 1984
24. Riffel B, Stöhr M, Körner S: Spinal and cortical evoked potentials following stimulation of the posterior tibial nerve in the diagnosis and localization of spinal cord diseases. Electroencephalogr Clin Neurophysiol 58:400–407, 1984
25. Lehmkuhl LD, Dimitrijevic MR, Zidar J: Lumbosacral evoked potentials (LSEPs) and cortical somatosensory evoked potentials (SEPs) in patients with lesions of the conus medullaris and cauda equina. Electroencephalogr Clin Neurophysiol 71:161–169, 1988
26. Yiannikas C: Short-latency somatosensory evoked potentials in peripheral nerve lesions, plexopathies, radiculopathies, and spinal cord trauma, in Chiappa KH (ed): Evoked Potentials in Clinical Medicine. New York, NY, Raven Press, 1990, pp 463–468
27. Aalfs CM, Koelman JHTM, Aramideh M, et al: Posterior tibial nerve somatosensory evoked potentials in slowly progressive spastic paraplegia: a comparative study with clinical signs. J Neurol 240:351–356, 1993
28. Holliday TA, Weldon NE, Ealand BG: Percutaneous recording of evoked spinal cord potentials of dogs. Am J Vet Res 40:326–331, 1979
29. Kornegay JN, Arvle EM, Purinton PT, et al: Somatosensory-evoked potential in clinically normal dogs. Am J Vet Res 42:70–73, 1981
30. Van Gestel MA: Electrospinogram bij de hond. Tijdschr Diergeneeskd 111:1185–1188, 1986
31. Steiss JE, Wright JC: Maturation of spinal-evoked potentials to tibial and ulnar nerve stimulation in clinically normal dogs. Am J Vet Res 51:1427–1432, 1990
32. Holliday TA.: Electrodiagnostic examination. Somatosensory evoked potentials and electromyography. Vet Clin N Am Small Anim Pract 22:833–857, 1992
33. Poncelet L, Delauche A, Vinals C, et al: Effect of bilateral stimulation on spinal evoked potentials in dogs. Am J Vet Res 53:1305–1308, 1992
34. Poncelet L, Michaux C, Balligand M: Influence of body size on tibial nerve somatosensory evoked potentials in dogs. Am J Vet Res 54:178–182, 1993
35. Cuddon PA, Delauche AJ, Hutchison JM: Assessment of dorsal nerve root and spinal cord dorsal horn function in clinically normal dogs by determination of cord dorsum potentials. Am J Vet Res 60:222–226, 1999
36. Redding RW, Lee AH, Wilson SG: Spinal-evoked potentials and spinal conduction velocity of the cat: reference values. Am J Vet Res 45:2175–2177, 1984
37. Sims MH, Selcer RR: Somatosensory-evoked and spinal cord-evoked potentials in response to pudendal and tibial nerve stimulation in cats. Am J Vet Res 50:542–545, 1989
38. Shores A, Redding RW, Knecht CD: Spinal-evoked potentials in dogs with acute compressive thoracolumbar spinal cord disease. Am J Vet Res 48:1525–1530, 1987
39. Kornberg M, Bichsel P, Lang J: Electromyography and spinal evoked potentials in the canine cauda equina syndrome. Schweiz Arch Tierheilk 131:287–289, 1989
40. Stegeman DF, van Oosterom A, Notermans SLH: Theoretical aspects of far-field somatosensory evoked potential generation, in Barber C, Blum T (eds): Evoked Potentials III. The Third International Evoked Potentials Symposium, 1986, Berlin, Germany. Boston, MA, Butterworths, 1987, pp 261–264
41. Takahashi N, Yabuki S, Aoki Y, et al: Pathomechanisms of nerve root injury caused by disc herniation: an experimental study of mechanical compression and chemical irritation. Spine 28:435–441, 2003
42. Chiappa KH: Short-latency somatosensory evoked potentials: interpretation, in Chiappa KH (ed): Evoked Potentials in Clinical Medicine. New York, NY, Raven Press, 1990, pp 371–399
43. Valeriani M, Insola A, Restuccia D, et al: Source generators of the early somatosensory evoked potentials to tibial nerve stimulation: intracerebral and scalp recording study. Clin Neurophysiol 112:1999–2006, 2001