Assessment of cord dorsum potentials from caudal nerves in anesthetized clinically normal adult dogs without or during neuromuscular blockade

James O. Campbell, DVM, PhD; Natasha J. Olby, Vet MB, PhD; Jonathan A. Hash, BA; B. Duncan X. Lascelles, BVSc, PhD

Objective—To assess the feasibility of measuring cord dorsum potentials (CDPs) in anesthetized clinically normal dogs after caudal nerve stimulation, determine the intervertebral site of maximum amplitude and best waveform of the CDP, and evaluate the effects of neuromuscular blockade.

Animals—8 male and 4 female dogs (age, 1 to 5 years).

Procedures—Dogs were anesthetized, and CDPs were recorded via needles placed on the dorsal lamina at intervertebral spaces L1-2 through L7-S1. Caudal nerves were stimulated with monopolar electrodes inserted laterally to the level of the caudal vertebrae. Dogs were tested without and during neuromuscular blockade induced with atracurium besylate. The CDP latency and amplitude were determined from the largest amplitude tracings.

Results—CDPs were recorded in 11 of 12 dogs without neuromuscular blockade and in all dogs during neuromuscular blockade. The CDP was largest and most isolated at the L4-5 intervertebral space (3 dogs) or the L5-6 intervertebral space (9 dogs); this site corresponded to the segment of insertion of the first caudal nerve. Onset latencies ranged from 2.0 to 4.7 milliseconds, and there was no effect of neuromuscular blockade on latencies. Amplitudes of the CDPs were highly variable for both experimental conditions.

Conclusions and Clinical Relevance—CDPs were recorded from all dogs tested in the study; neuromuscular blockade was not critical for successful CDP recording but reduced muscle artifact. This technique may be useful as a tool to assess the caudal nerve roots in dogs suspected of having compressive lumbosacral disease or myelopathies affecting the lumbar intumescence. (Am J Vet Res 2013;74:616–620)

The CDP is a stationary-evoked potential arising in dorsal horn interneurons after stimulation of peripheral nerves or nerve roots. Results of experiments involving intramedullary recordings, osmic acid-induced lesions, and spinal cord hemisection in decapitated and decerebrated cats support the conclusion that the CDP is a purely sensory phenomenon. The waveform consists of a large, slow negative deflection, which is preceded by an initial small triphasic wave and followed by a slow positive wave. The large negative potential that defines the waveform represents depolarization of dorsal horn interneurons, whereas the initial triphasic spike represents the incoming volley in the primary afferent axons. The potential is generated in the dorsal gray matter over 1 to 2 spinal cord segments, with the largest waveform generated in the spinal cord segments where the stimulated dorsal nerve roots penetrate the spinal cord.

In addition to early basic experimental data, there are examples of CDPs for dogs and cats recorded as part of spinal-evoked potential studies in intact animals following tibial and ulnar nerve stimulation. Details of CDP latencies and magnitudes have been established for those nerves in dogs. Procedures have also been described for CDP measurement after caudal nerve stimulation, but there are no data on normal latencies and magnitudes or the reliability of those measures to our knowledge. The purposes of the study reported here were to determine whether CDPs could be reliably recorded following caudal nerve stimulation in anesthetized adult dogs, which intervertebral location resulted in the largest caudal nerve CDP amplitude.
and what effect administration of atracurium besylate had on the latency, magnitude, and subjective form of the potential. The ultimate goal of this study was to develop an electrophysiological tool for functional assessment of lumbosacral compressive lesions of peripheral nerves and nerve roots that pass over the lumbosacral space within the spinal canal in dogs.

**Materials and Methods**

**Subjects**—Eight male and 4 female purpose-bred large mixed-breed dogs that were 1 to 5 years of age and weighed 23.0 to 32.1 kg were used. Dogs were acclimated to the housing facility for at least 3 months before being used in experiments. Within a 48-hour period prior to participation in experiments, all dogs underwent physical and neurologic examinations performed by the investigators; no abnormalities were found in any dog. The lumbar, lumbosacral, and sacral axial portions of each dog’s skeleton appeared normal, as determined from lateral radiographic views obtained 2 weeks prior to the study. Among the dogs, there were no remarkable abnormalities detected via CBC or serum biochemical analysis performed during a 48-hour period prior to the start of the experiments.

**Anesthesia**—Anesthesia was induced with propofol (4 mg/kg, IV) after administration of acepromazine maleate (0.05 mg/kg, IM) and morphine sulfate (0.1 mg/kg, IM). Each dog was intubated, and anesthesia was maintained with an inhaled isoflurane and oxygen mixture. Rectal temperature was maintained between 36.2° and 38.2°C with circulating warm water blankets throughout the experiments.

**Electrophysiology protocol**—Nerve stimulation, recording and averaging of waveforms, and measurement of CDP latencies and amplitudes were performed with an electrophysiologic unit. Cathode and anode monopolar stimulating needles were inserted into the muscles on the dorsolateral aspect of the tail (sacrocaudalis dorsalis lateralis, sacrocaudalis dorsalis medialis, and intertransversarius dorsalis caudalis) until each needle tip made contact with a caudal vertebra approximately 4 cm from the tail base (Figure 1). This distance was chosen to allow the technique to be applied to dogs with docked tails. The ground electrode was placed subcutaneously several centimeters proximal to the cathode needle.

Monopolar recording electrodes were placed at each intervertebral space from L1-2 to L7-S1; needles were inserted on the dorsal midline and advanced toward the intervertebral spaces until they contacted the dorsal laminae. Their placement was confirmed and adjusted with radiographic guidance (Figure 2). The distance between the cathode and each of the recording electrodes was measured at the skin surface. Reference electrodes were placed subcutaneously 4 to 6 cm lateral to each recording electrode. Recordings were made from 4 consecutive intervertebral spaces simultaneously (data series 1). Square waves (each 0.2 milliseconds in duration) were applied at a rate of 3.1 Hz, and stimulus strength was increased until a tail twitch was elicited. Five hundred responses were averaged. Atracurium besylate (0.2 mg/kg) was then administered IV, and the procedure was repeated (data series 2). Before repeating the procedure, neuromuscular blockade was confirmed with loss of paw twitch in response to 4 consecutive stimulations of the ulnar nerve with a peripheral nerve stimulator after atracurium administration.

The first 6 dogs on which experiments were performed were euthanized with an IV overdose of pentobarbital immediately after the experiment was completed and while the dogs were still anesthetized, and each cauda equina was exposed by dissection. The last 6 dogs on which experiments were performed were allowed to recover from anesthesia as part of a related experiment; these dogs were later euthanized, and each cauda equina was exposed by dissection. In 7 of the 12 dogs, the first caudal nerves were traced to their source spinal cord segment and the vertebral level of these spinal cord segments was recorded.

**Data analysis**—The intervertebral site at which the largest amplitude CDP was elicited was recorded; for 7 dogs, this site was compared with results of the dissection. The latency of CDP onset was determined from the largest amplitude tracings. The onset of each CDP was assessed as the takeoff point of the large negative cord dorsum peak (the triphasic spike was not always present; Figure 3). The distance from stimulating cathode to recording electrode...
was divided by the latency to calculate conduction velocity. The amplitudes were determined for the same peaks used to determine latencies and were measured from baseline to the peak of the negative potential. All 3 variables were expressed as mean, SD, and median.

The latency data were assessed for normality via the Kolmogorov-Smirnov test, and the data distribution was found to be nonnormal. The effect of neuromuscular blockade was evaluated by comparing the distance/latency values and amplitudes before and during neuromuscular blockade via a Wilcoxon signed rank test (a value of \( P < 0.05 \) was considered significant). The relationship between latency and the distance between recording and stimulating electrodes was evaluated via the Pearson product moment correlation coefficient.

**Results**

Cord dorsum potentials were successfully recorded after caudal nerve stimulation without neuromuscular blockade in 11 of 12 dogs and in all dogs after neuromuscular blockade with atracurium. Recording from consecutive intervertebral sites allowed observation of CDPs as well as moving potentials at multiple sites both with and without neuromuscular blockade. In all cases, the CDP was largest at a single intervertebral site: at the L5-6 intervertebral space in 9 dogs and at the L4-5 intervertebral space in 3 dogs. This site was consistent with the location of the insertion of the first caudal nerve in the spinal cord as determined via gross examination in 7 of the 12 dogs.

Six dogs had CDPs evident at multiple sites both with and without neuromuscular blockade. In 3 dogs, CDPs were measured at the site cranial to and the site caudal to the site where the largest CDP was recorded; in the other 3 dogs, CDPs were measured either at the site cranial to or the site caudal to the site where the largest CDP was recorded. The site where the largest CDP was recorded was not affected by neuromuscular blockade, but subjectively, the onset of the CDP was

![Figure 3—Representative CDPs in 2 anesthetized dogs (A and C; B and D) in the absence of (A and B) and during (C and D) neuromuscular blockade (achieved via IV administration of atracurium besylate). For 1 dog, moving potentials are visible in the traces recorded from intervertebral spaces L7-S1 and L6-7 without (A) and during neuromuscular blockade (C); the arrow in panel A illustrates that the moving potential may obscure the point of onset of the CDP. For the other dog, moving potentials were not recorded under either condition (B and D). Horizontal increments represent intervals of 5 milliseconds, and vertical increments represent intervals of 1 \( \mu V \).](image)
being investigated.2 CDPs a few centimeters adjacent to the site of the segment of the first caudal nerve likely reflect the ability to record CDPs detected cranial and caudal to the site of insertion consistently recorded as were the CDPs (Figure 3). The of the largest CDP in most dogs but were not as con-
siderate to the site where the first caudal nerve inserted vertebral space (9 dogs) and L4-5 intervertebral space (3 dogs). Caudal nerve CDPs were largest at the L5-6 inter-
verse small CDPs (< 2.0 µV). In the absence of neuromuscular blockade, the CDP latency range was 2.0 to 4.7 milliseconds during neuromuscular blockade. As for the CDPs, neuromus-
cular blockade had no effect on the presence of moving potentials.

The range of onset latencies was 2.1 to 4.7 milliseconds in the absence of neuromuscular blockade, and 2.0 to 4.7 milliseconds during neuromuscular blockade (Figure 4). The descriptive statistics for the variables of interest were summarized (Table 1). The distance between stimulating and recording electrodes ranged from 19.46 to 30.5 cm (mean ± SD, 23.75 ± 3.12 cm; median, 23.5 cm). There was no significant correlation between distance and latency (r² = 0.55; P > 0.05). The distance/latency value ranged from 57.03 to 122.5 m/s, and there was no effect of neuromuscular blockade on either the CDP latency or distance/latency value (P > 0.05). Compared with latencies, amplitudes within and between dogs with and without neuromuscular blockade were highly variable, ranging from 4 to 22.2 µV in the absence of neuromuscular blockade and from 2 to 28 µV during neuromuscular blockade.

**Discussion**

In the present study, CDPs were successfully measured in all 12 study dogs, although neuromuscular blockade was necessary to obtain the waveforms for one of the dogs. Caudal nerve CDPs were largest at the L5-6 intervertebral space (9 dogs) and L4-5 intervertebral space (3 dogs); the site where the largest CDP was recorded corresponded to the site where the first caudal nerve inserted into the cord, on the basis of findings of gross dissection in 7 dogs. The first 5 caudal spinal segments are closely grouped over < 1 vertebral level because the spinal cord tapers; thus, most input from the tail is located close to the site of insertion of the first caudal nerve.12 The presence of CDPs at multiple intervertebral sites in 5 dogs is consistent with findings of a previous study,13 and the smaller CDPs detected cranial and caudal to the site of insertion of the first caudal nerve likely reflect the ability to record CDPs a few centimeters adjacent to the site of the segment being investigated.2

Moving potentials were recorded caudal to the site of the largest CDP in most dogs but were not as consistently recorded as were the CDPs (Figure 3). The presence of moving potentials in the same tracing as the largest CDP occasionally seemed to obscure the onset of the CDP and may have contributed to variability of latency measurements. A prior study11 that assessed CDPs after tibial nerve stimulation in clinically normal dogs also revealed that presumed moving potentials were variably present on the rising phase of the large negative peak of the CDP, causing difficulty in identifying the onset for latency measurements. Strategies can be developed to mitigate this type of error. For example, latencies can be measured to a predetermined level of deviation from the baseline. However, such strategies require a consistent baseline, which is also difficult to define.

Muscle artifact has been shown to interfere with assessment of spinal-evoked potentials when stimulation is performed close to the recording site.14 In the present study, there was no effect of neuromuscular blockade on the amplitudes or on the latencies of the CDPs for the study dogs. This was not unexpected because the potential is considered to be a purely sensory phenomenon and neuromuscular blockade does not interfere with nerve conduction velocity.15 However, neuromuscular blockade subjectively made it easier to determine CDP onset for several dogs. In 1 dog, no waveforms were elicited in the absence of neuromuscular blockade, but a CDP could be recorded during neuromuscular blockade. However, the lack of waveforms in the absence of neuromuscular blockade was likely a result of technical problems rather than because of waveforms being obscured by muscle artifact. Thus, although neuromuscular blockade is not critical to obtaining diagnostic cord dorsum recordings following stimulation of the caudal nerves, when the waveforms obtained are difficult to interpret, a clinician should consider eliminating muscle artifact in this manner.

In the present study CDP amplitudes were extremely variable among dogs, making it unlikely that

---

**Table 1—Latency and amplitude of caudal nerve CDPs obtained from anesthetized dogs without and during neuromuscular blockade (achieved via IV administration of atracurium besylate).**

<table>
<thead>
<tr>
<th>Variable</th>
<th>During neuromuscular blockade (n = 12)</th>
<th>Without neuromuscular blockade (n = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency (ms)</td>
<td>Mean ± SD 2.97 ± 0.36</td>
<td>Median 2.85 ± 0.31</td>
</tr>
<tr>
<td>Distance/latency*</td>
<td>82.48 ± 19.78</td>
<td>80.36 ± 17.42</td>
</tr>
<tr>
<td>Amplitude (µV)</td>
<td>9.05 ± 5.94</td>
<td>6.30 ± 2.85</td>
</tr>
</tbody>
</table>

*Value represents the distance from stimulating cathode to recording electrode divided by the latency (ie, conduction velocity).
amplitude could be used as an objective clinical assessment of dorsal horn function in this species. It is difficult to place needles at each vertebral level in exactly the same position relative to the spinal cord, even with the aid of radiographic imaging. In addition, the cross-sectional area of the spinal cord increases at the intumescence and then rapidly tapers, thereby changing the distance between the dorsal laminae and the spinal cord along its length. These differences may well account for some of the variability in CDP amplitude.

The relatively low variability of caudal nerve CDP latency in the present study has suggested that this measurement may serve as a tool to assess the caudal nerve roots in dogs suspected of having compressive lumbosacral disease or myelopathies affecting the lumbar intumescence, providing information in addition to that gathered from clinical evaluation and diagnostic imaging. In all study dogs, the stimulating needles were placed approximately 4 cm from the tail base; the lack of correlation of latency with the distance between the stimulation and recording sites likely reflected the relatively small variation of distances assessed in the present investigation. In a recent study, the latency of spinal-evoked potentials measured after tibial nerve stimulation in dogs with clinical signs of lumbosacral stenosis was found to be altered, compared with findings for healthy dogs. Those researchers detected delays in onset of evoked potentials, but data were averaged across waveforms measured from all intervertebral sites between L7-S1 and T13-L1. Use of caudal nerve CDPs allows for assessment of discrete, identifiable sites between L7-S1 and T13-L1. Use of caudal nerve CDPs in dogs without neuromuscular blockade appeared to improve the interpretability of recorded waveforms. Given that the largest CDP amplitude was recorded most commonly at the L3-6 intervertebral space, but also was detected at the L4-5 intervertebral space, it would appear prudent to obtain recordings from both interspaces in a patient. The latency of the CDP was relatively uniform in the dogs assessed, which suggests that this variable could potentially be a useful measure with which to determine whether compression at the LS junction identified via MRI in a dog is functionally relevant. On the other hand, the CDP amplitude was highly variable and less likely to be of clinical use. Future studies of caudal nerve CDP latency in dogs with lumbosacral compression are indicated.

References