Somatosensory Evoked Potentials Produced by Stimulation of the Dorsomedial Nerves Innervating the Tail in Dogs

Naomi WADA1), Junko AKATANI2), Noriko SHIKAKI2), Atsu TAGA2), Kazuhiro ITAMOTO2), Yasuo TAURA3) and Mikihiro TOKURIKI1)

1)Department of Veterinary Physiology, 2)United Graduated School of Veterinary Science and 3)Department of Veterinary Surgery, Yamaguchi University, Yamaguchi 753-8515, Japan

(Received 16 May 2001/Accepted 13 November 2001)

NOTE Surgery

The sacral and caudal segments of the spinal cord and their nerve roots are anatomically defined as the cauda equina. The clinically important nerves, the sciatic, pudendal, pelvic and coccygeal nerves, pass thorough the cauda equina. Lesions of the cauda equina produce clinical signs such as paresthesia of the tail or perineal region, and urinary and fecal incontinence (cauda equina syndrome (CES), [5]). Somatosensory evoked potentials (SEPs) produced by stimulation of the DMN reflect the activities of ascending neuronal pathways above the coccygeal spinal segments and may be a useful tool for examining cauda equina syndrome.

KEY WORDS: canine, cauda equina syndrome, somatosensory evoked potential.

ABSTRACT. To record the somatosensory evoked potentials (SEPs) produced by stimulation of tail nerves and determine the effects of acute compression of the cauda equina on SEPs. The subjects were 10 adult Beagles. SEPs were recorded after stimulating the dorsomedial nerves (DMN) innervating the tail. The cauda equina was compressed using a balloon catheter inserted into the vertebral arch. In SEPs, two negative and one positive peak were often observed. The compression of the cauda equina caused significant depression of the positive component. The SEPs produced by stimulation of the DMN reflect the activities of ascending neuronal pathways above the coccygeal spinal segments and may be a useful tool for examining cauda equina syndrome.

The subjects were clinically healthy adult Beagles (5 males and 5 females), weighing 7.8–10.7 kg. Each dog was injected intramuscularly with atropine sulfate (0.05 mg/kg) and midazolam (0.3 mg/kg, Dornicum, Roche Co., Basel, Swiss) followed by medetomidine (0.04 mg/kg, Domitor, Farmos Co., Turku, Finland). Ten to 20 min after injection of midazolam + medetomidine, stable narcosis was observed for 20–30 min. During experiments, the level of anesthesia was maintained by administration of isoflurane (2.5%)-oxygen. Recordings were obtained using enamel-coated copper wire with a diameter of 120 μm, with the tip uncoated for approximately 2–3 mm. These wires were inserted through a 25 or 26 gauge injection needle and the uncoated tip of the wire was bent for 2 mm. The recording electrodes were placed on the frontal head skin [6], the 10th thoracic vertebrae (T10) and the 4th lumbar vertebrae (L4). The recording electrodes at T10 and L4 were inserted along the cranial edge of the processus spinatus of the 11th thoracic vertebrae (T11) and the 5th lumbar vertebrae (L5), respectively. And the tip of the wire electrode was placed near the intervertebral foramen between T10 and T11, and L4 and L5, respectively. The reference electrodes for the recording electrodes at the frontal head skin, T10 and L4 were placed on the spinous process of the axis, T10 and L4, respectively. The rectal temperature was continuously monitored and maintained at approximately 37°C (37 ± 2°C) using a heating mat. Electrical stimulation of DMN on both sides (0.1 ms duration) was performed using paired needle electrodes inserted into ECL between the 3rd caudal vertebrae (Ca3) and the 4th caudal vertebrae (Ca4) levels (distance between paired electrodes: 1–1.5 cm) on both sides. Stimuli of intensity at 1.5–3 times threshold (1.5–3T) and square pulse were used in order to cause detectable movements of the tail. Signals from the recording electrodes were amplified using a bioamplifier (VC-11, Nihon Kohden Co., Tokyo, Japan) and stored on magnetic tape with the

stimulation signal (RD-135, TEAC Co., Tokyo, Japan). The signals were averaged over 200 sweeps using the MacLab/8S system off line. Latency and amplitude were measured on 256 averaged signals (MacLab/8S, AD Instruments Co., Castle Hill, Australia). The effects of experimentally induced acute compression of the cauda equina on SEPs were studied in 7 dogs. After making a hole in the left side of the vertebral arch of the 7th lumbar vertebrae (L7), a balloon catheter was inserted and placed on the ventral aspect of the cauda equina. Compression was performed by inflating the balloon by injection of saline (0.5 ml, 1.0 ml) at the 5–6th lumbar vertebrae (L5–L6). SEP recordings were made immediately after inserting the balloon catheter and 5 and 10 min after the saline injection. The results obtained in the present study were analyzed by use of a one-way ANOVA. Post-hoc analysis was performed by using a t-test with Bonferroni/Dunn adjustments. In all cases, significance was established at P<0.01.

Figure 1 illustrates typical examples of SEP waveforms recorded at the frontal head skin (the lower trace), T10 (the middle trace) and L4 (the upper trace) after stimulating

<table>
<thead>
<tr>
<th>Component</th>
<th>4th lumber vertebrae (L4)</th>
<th>10th thoracic vertebrae (T10)</th>
<th>the frontal head skin</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1</td>
<td>13.7 ± 0.98 µV</td>
<td>19.6 ± 1.78 µV</td>
<td>13.8 ± 4.96 µV</td>
</tr>
<tr>
<td>P1</td>
<td>54.9 ± 16.31</td>
<td>60.0 ± 6.19</td>
<td>59.1 ± 3.36</td>
</tr>
<tr>
<td>N2</td>
<td>97.0 ± 5.48</td>
<td>96.4 ± 6.86</td>
<td>96.6 ± 7.70</td>
</tr>
</tbody>
</table>

The columns show the latency (ms), amplitude (µV) and the number of dogs (n) in which each component was observed. The measuring method of amplitude used base to peak about N1 and peak to peak about P1. In more than 5 dogs (>50 %), the N1 and P1 components of the SEPs at L4, T10 and the frontal head skin were observed after stimulating DMN. Especially, the P1 component was observed at the frontal head skin of all dogs. Figure 2 shows the changes in the waveforms of SEPs at the frontal head skin and T10 following acute compression of the cauda equina in the same animal. Figure 2a shows an example of the effects of compression on SEP waveforms. The effects of compression on N1 and N2 were not significant and unstable, while the effects on P1 was marked. In general, with increased intensity of compression, greater reduction of the amplitude of P1 was observed. However, there was no significant effect on latency. Figure 2b shows the mean amplitude of P1 as percent of control and the standard errors before and after injection of saline into the balloon. The histograms show that increasing the acute compression increased the depression of P1 amplitude. Single and double asterisks indicate significant differences of P1 amplitude compared to control and 0.5 ml injection at 5 min, respectively. The difference between P1 amplitude at 5 min and 10 min after the injection of saline was not significant.

Compression of the cauda equina is relatively common and has been reported in dogs of various ages and breeds [3]
The compression of the cauda equina causes syndrome (CES) with various kinds of symptoms and diagnosis of CES is very important for veterinary neurology. The results of the present experiments clearly show that SEPs can be recorded after electrically stimulating the tail nerves DMN at L4, T10 and the front of head skin. DMN consists of spinal nerves below S3, and SEPs evoked by DMN stimulation reflect the neural pathways above the sacrococcygeal spinal segments. DMN is easily stimulated by paired needle electrodes, and produced peaks with fixed latency in most animals. Furthermore, SEPs were recorded in 70% of dogs at the front of head skin after stimulating DMN. Other investigators have shown that experimental compression of the cauda equina produced effects similar to the symptoms of CES [1, 2, 5].

In the present experiments, the method which used the balloon as a model of not partial pressure but overall pressure of the cauda equina domain, was performed, the compression of the cauda equina produced a significant change in P1 in SEPs evoked by DMN stimulation. These facts suggest that SEPs evoked by stimulation of DMN may be a useful tool for examining a part of CES. Moreover, the further experiment, SEPs may be able to measure the grade and part of damage in the cauda equina.

REFERENCES