J Vet Clin 19(3) : 277-282(2002)

Effects of Anesthetics on Somatosensory Evoked Potentials (SEPs) in Dogs

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Abstract : This study was designed to evaluate the effects of anesthetics on waveform of SEPs and to authorize possible anesthetic protocol for measurement of the somatosensory evoked potentials (SEPs). Thirteen anesthetic methods were used. The SEPs were recorded on two channels (between the 5th and 6th lumbar vertebra as the channel 1 and between the 11th and 12th thoracic vertebra as the channel 2) following stimulation of posterior tibial nerve. To analyze SEPs wave, latency and conduction velocity were measured. Among thirteen anesthetic methods, standard SEPs waveforms were observed in dogs anesthetized with following six methods: Acepromazine + Thiopental Na + Isoflurane, Acepromazine + Propofol + Isoflurane, Diazepam + Xylazine, Xylazine + Ketamine, Acepromazine + Propofol infusion and Propofol infusion. Above six methods could be used with sufficient anesthetic depth. The differences of latency and conduction velocity among six groups were minimal compared to general waveform of SEPs. These results indicate that the six anesthetic methods can be used for recording SEPs in the dog. In particular, Diazepam + Xylazine and Xylazine + Ketamine as injectable anesthesia are considered more convenient than other four methods in veterinary medicine.

Key words **:** dog, somatosensory evoked potentials, anesthetics

Introduction

The somatosensory evoked potentials (SEPs) permit non-invasive testing of the functional state of specific parts of the nervous system. In contrast, most imaging techniques, e.g., radiographs and myelographs, provide only anatomic information. SEPs thus extend the clinical neurologic examination and complements the common imaging procedures[11].

The SEPs are electrical events elicited from neurons, synapses, or axons when sensory axons in peripheral nerves are stimulated. SEPs of dogs and cats have been studied experimentally and clinically[14,18,29,32,33,35].

The application of SEPs technique to questions of neurological diagnosis, prognosis and management originate from the observation by Dawson[8].

Since the early investigations, the possibility that SEPs might be used to help establish the level, severity and evolution of traumatic spinal cord damage has been widely examined[28].

Evoked potentials were used to evaluate the function of the sensory neural pathways. The validity, reliability and sensibility of SEPs examines have been well documented[37,39]. SEPs were often used to monitor the functional integrity of the neurological pathways during surgical procedure[4,5,19,20,34] and to determine the effectiveness of the surgical procedure[1,3,12].

Despite a dramatic increase in the use of sensory evoked potentials (EPs) to monitor the integrity of neural pathways in anesthetized patients, published information dealing with the effect of different anesthetic agents on short-latency SEPs was relatively limited[36]. Clark et al indicated that most general anesthetics alter EPs[7], but these studies failed to provide information on quantitative effects of individual anesthetics on SEPs. Many reports have suggested that halogenated anesthetics should be avoided in patients whom SEPs were being monitored[10,24,26,31]. McPherson *et al* reported a greater decrease of amplitude of both upper and lower extremity evoked potentials after use of nitrous oxide compared to that with either enflurane or isoflurane[23].

Fewer channels may unduly prolong the examination and has many artifacts. Artifacts arising from skeletal muscles are the most troublesome and measurement of SEPs in dogs should need restraint[9,13]. So, it is not possible to measure the posterior tibial nerve SEPs without anesthesia in dogs used two channels. Furthermore, there have been no comparative studies on the effects of inhalation anesthetic agents and injectable anesthetic agents on SEPs.

In the present study, the effects of major anesthetics on waveform of SEPs were evaluated to authorize possible anesthetic protocol for recordings of SEPs in dogs.

Materials and Methods

Experimental animals

Neurologically intact, five, mixed breed, male dogs (2-5 years, 3-5 kg) were used for this experiment.

Apparatus for experiments

Nerve stimulation and SEPs recording were performed with Neuropack 2, MEM-7102 (Nihon Kohden, Japan) and subdermal Platinum needle electrode (E-2, Grass, USA) was applied on the two channels. Anesthetic apparatus were used FO-20S vaporizer (Acoma, Japan), ACE 3000 ventilator (Acoma, Japan) and Vet/Ox Plus (SDI, USA) as patient

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monitoring system.

Condition of SEPs stimulation and measurement

The posterior tibial nerve was stimulated via surface electrodes placed at the medial region between calcaneal tuberosity and distal part of tibia (Fig 1). The nature of the stimulation was electro-stimulation. Two hundred constant current impulses of 0.2 ms duration were delivered at the rate of 2 Hz to stimulate the posterior tibial nerve. The supramaximal stimulation intensity was used at least 2-3 times of the response threshold.

The SEPs were measured on two channels. Platinum needle electrodes were placed on the subdermal region between the 5th and 6th lumbar vertebra as the channel 1 and on the subdermal region between the 11th and 12th thoracic vertebra as the channel 2 (Fig 1).

The latencies and distances from the electro-stimulating point on the posterior tibial nerve to channel 1 and channel 2, and from the channel 1 to channel 2 were measured.

In the channel 1 and 2, the first upward beginning point (positive peak) was named as LP1 and TP1, respectively. The first downward beginning point (negative) was marked as LN1 and TN1 (Fig. 2).

LP1, LN1, TP1 and TN1 denotes first lumbar positive, first lumbar negative, first thoracic positive and first thoracic negative, respectively.

Condition for recording

Animals were placed on the nonconductive table in the room at ambient temperature. All recordings were made within 20 minutes after induction of anesthesia. Anesthetic withdrawal time in the experimental dogs was 2 weeks.

A signal averaging technique was used to cancel the randomly occurring EEG waves and to record the summated time-locked signals[25]. SEPs were averaged 500-1,000 times in each recording.

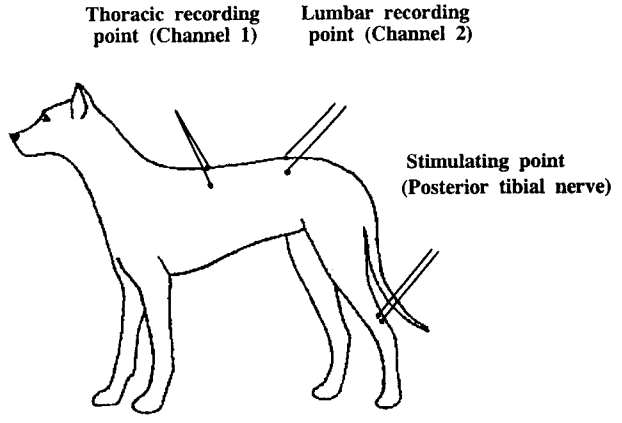

Fig 1. Diagram indicating the site of electrodes for channel 1, channel 2 and stimulating point. The reference electrodes of channel 1 and 2 were positioned 3 cm apart from recording electrodes.

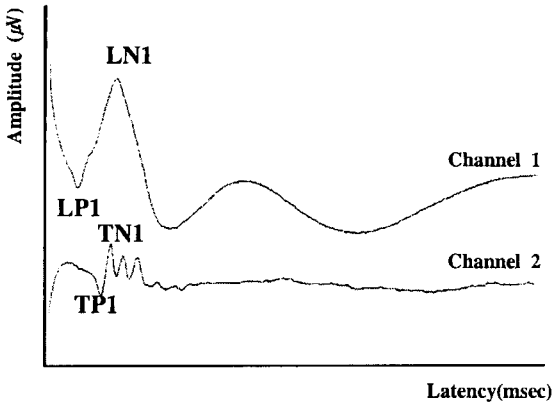

Fig 2. Schematic diagram showing lumbar (channel 1) and thoracic (channel 2) somatosensory evoked potentials recorded by stimulating the posterior tibial nerve. The amplitude of LP1 and LN1 (between L5 and L6) was higher than that of TP1 and TN1 (between T11 and T12).

Anesthesia

Thirteen anesthetic methods were examined by using eight injectable and one inhalation anesthetics.

Injectable anesthetics used were Acepromazine(Sedaject®, Samwoo, Korea) 0.5 mg/kg, Diazepam(Valium®, Roche Korea, Korea) 1 mg/kg + Xylazine(Rompun®, Bayer Korea, Korea) 2 mg/kg, Acepromazine 0.5 mg/kg + Ketamine(Keiran®, Hankuk United, Korea) 10 mg/kg, Acepromazine 0.2 mg/kg + Xylazine 2 mg/kg, Diazepam 1 mg/kg + Ketamine 5 mg/kg, Acepromazine 0.5 mg/kg + Propofol(Pofol®, Jeil, Korea) infusion, Medetomidine(Domitol®, Meiji, Japan) 0.1 mg/kg, Propofol infusion, Ketamine 15 mg/kg, Xylazine 1.5 mg/kg + Ketamine 10 mg/kg, and Zolazepam-Tiletamine(Zoletile®, Virvac, France) 7.5 mg/kg. Inhalation anesthetics used were Acepromazine 0.1 mg/kg + Thiopental Na (Thionyl®, Daehan, Korea) 15 mg/kg + Isoflurane(Aerane®, Ilsung, Korea) 2.5%, and Acepromazine 0.1 mg/kg + Propofol 6 mg/kg + Isoflurane 2.5%.

All of injectable anesthetics were administered intravenously. In Propofol infusion groups, propofol was administered at a dose of 6 mg/kg for induction of anesthesia, and then infused 0.8 mg/kg/min for the maintenance. As inhalation anesthesia, Thiopental Na or Propofol was injected intravenously to induce anesthesia after premedication of acepromazine maleate and the maintenance of anesthesia was carried out isoflurane with 100% oxygen. Oxygen was supplied by use of following equation[22]:

$$O_2 \text{ supply (L/min)} = 0.01 \times \text{Body weight (Kg)} \times \text{Respiratory rate (breath/min)}$$

Evaluation and analysis

1. Existence of standard SEPs. The presence of regular

SEPs waveform and of high frequency signal/noise ratio under each anesthetic protocol were examined at first.

2. Evaluation of SEPs waveform. Latencies and distances from electro-stimulating point to LP1, LN1, TP1 and TN1, and from channel 1 to channel 2 was recorded. And then, these measured latencies and distances were converted to the conduction velocity. The limits of normal range were mean±2.5SD[6]. The conduction velocity between two points can be calculated by use of following equation:

$$\text{Conduction velocity (m/sec)} = \frac{\text{distance (cm) of two points}}{\text{latency (msec) difference}} \times 10$$

3. Recording anesthetic maintenance time enough to measure SEPs in injection anesthesia.

Statistical analysis

To evaluate the statistical significance between SEPs components under Acepromazine + Thiopental Na + Isoflurane and those under other anesthetic methods, Post Hoc tests were performed by SPSS (ver 8.0).

Results

Existence of standard SEPs

Anesthetic methods showing standard SEPs waveforms were Acepromazine + Thiopental Na + Isoflurane (group I), Acepromazine + Propofol + Isoflurane (group II), Diazepam + Xylazine (group III), Xylazine + Ketamine (group IV), Acepromazine + Propofol infusion (group V), Propofol infusion (group VI).

Other anesthetic methods, Zolazepam + tiletamine, Diazepam + Ketamine, Ketamine, Acepromazine + Ketamine, Medetomidine, Acepromazine + Xylazine and Acepromazine, did not show standard waveform and various types of artifacts were observed in the SEPs waveforms.

Evaluation of SEPs waveform

The latencies and velocity of SEPs waveforms of Group I, Group II, Group III, Group IV, Group V and Group VI were as follows:

In the latency, stimulating point (SP)-LN1, SP-TP1 of group II, Ch1-Ch2 of group III, Ch1-Ch2 of group IV and SP-LP1, Ch1-Ch2 of group V were significantly different from that of group I ($p < 0.05$) (Table 1).

In the conduction velocity, SP-LN1 of group II, Ch1-Ch2 of group III, Ch1-Ch2 of group IV, SP-LP1 of group V and SP-LN1 of group VI had significant difference compared to group I ($p < 0.05$) (Table 2).

But, the differences of latency and conduction velocity among six groups were minimal compared to general waveform of SEPs and there did not alter channel 1 together with

Table 1. The latencies measured by SEPs in each group

Group	Number of dogs	Latency (msec)				
		SP-LP1	SP-LN1	SP-TP1	SP-TN1	Ch1-Ch2
I	12	2.65±0.40	4.84±0.67	4.36±0.53	5.15±0.61	2.50±0.50
II	21	2.92±0.40	5.35±0.48*	4.89±0.49*	5.50±0.50	2.58±0.23
III	17	2.76±0.59	5.04±0.65	4.32±0.71	4.99±0.71	2.22±0.50*
IV	14	2.93±0.37	4.81±0.51	4.52±0.32	5.07±0.32	2.13±0.19*
V	23	3.00±0.40*	5.09±0.63	4.51±0.72	5.24±0.51	2.24±0.24*
VI	9	2.74±0.35	4.34±0.87	4.53±0.21	5.03±0.23	2.29±0.39

Data are expressed as mean±SD. *Significantly different from Group I ($p<0.05$). Group I: Acepromazine + Thiopental Na + Isoflurane, Group II: Acepromazine + Propofol + Isoflurane, Group III: Diazepam + Xylazine, Group IV: Xylazine + Ketamine, Group V: Acepromazine + Propofol infusion, Group VI: Propofol infusion, SP: stimulating point.

Table 2. The conduction velocity measured by SEPs in each group

Group	Conduction velocity (m/sec)				
	SP-LP1	SP-LN1	SP-TP1	SP-TN1	Ch1-Ch2
I	103.7±11.8	56.8±6.3	92.0±7.9	78.0±7.9	52.0±7.3
II	94.3±12.3	51.1±4.5*	82.5±7.7	73.2±6.1	51.1±4.1
III	101.3±17.4	54.5±5.8	93.7±14.8	80.7±11.0	59.5±15.0*
IV	93.8±10.9	57.2±7.2	88.8±6.5	79.2±4.9	60.6±7.3*
V	91.9±12.2*	54.0±6.7	91.9±23.2	76.8±6.7	58.0±6.4
VI	100.6±14.6	64.7±13.2*	87.8±3.8	79.0±3.3	56.7±10.7

Data are expressed as mean±SD. *Significantly different from Group I ($p<0.05$). Group I: Acepromazine + Thiopental Na + Isoflurane, Group II: Acepromazine + Propofol + Isoflurane, Group III: Diazepam + Xylazine, Group IV: Xylazine + Ketamine, Group V: Acepromazine + Propofol infusion, Group VI: Propofol infusion, SP: stimulating point.

channel 2. Therefore, above six groups did not influence on SEPs.

Anesthetic maintenance time

The anesthetic maintenance time of group I, group II could be controlled by means of inhalation anesthesia. In addition, that of group V, group VI could be controlled by means of infusion anesthesia. The anesthetic maintenance times of group III and group IV using injectable anesthetics were 32 and 35 minutes, respectively. Therefore, all of these six groups had enough anesthetic maintenance time to measure SEPs.

Discussion

In this experiment, anesthetic methods showing standard SEPs waveforms were Acepromazine + Thiopental Na + Isoflurane, Acepromazine + Propofol + Isoflurane, Diazepam + Xylazine, Xylazine + Ketamine, Acepromazine + Propofol infusion, Propofol infusion. Their waveforms were regular, and had high signal/noise ratio. The other anesthetic methods such as Zolazepam + Tiletamine, Diazepam + Ketamine, Ketamine, Acepromazine + Ketamine, Medetomidine, Acepromazine + Xylazine and Acepromazine didn't show standard waveforms. It is considered that the waveforms can be measured on the sufficient anesthetic depth in which withdrawal reflex is absent in experimental animals.

These results indicate that six anesthetic methods can be used recording SEPs in the dog. In particular, Diazepam + Xylazine and Xylazine + Ketamine as injectable anesthesia are considered more convenient than other four methods in veterinary medicine.

Induction of anesthesia with barbiturates such as thiopental Na also causes little change in the early components used for monitoring, although cortical SEP latencies may be slightly increased[23]. The halogenated agents halogen, enflurane and isoflurane, which are widely used for maintenance of anesthesia to measure of SEPs. In this study, data from Acepromazine + Thiopental Na + Isoflurane were used as standard. But, the differences of latency and conduction velocity among six groups were minimal compared to general waveform of SEPs and there did not alter channel 1 together with channel 2. Therefore, above six groups did not influence on SEPs.

Premedicative drugs such as atropine, diazepam etc. have little effect on short-latency cortical or subcortical SEPs, although morphine and diazepam were found to attenuate the segmental activity recorded from the lumbar spinal cord following tibial nerve stimulation[14,21]. Premedication agents, such as narcotic analgesics and sedatives have been studied for the potential effect on SEPs. At premedication doses, these agents have little or no effect on the EPs[11,17]. In this experiment, diazepam did not induce significant changes in SEPs.

McPherson *et al* found SEP latencies increased but amplitudes unchanged following induction with a combination of thiopental and fentanyl. The decrease in the response amplitude and an increase of latency in the scalp-recorded potentials have been observed at induction doses of thiopental Na. The cervical potentials are relatively resistant to thiopental Na at these doses[23].

The halogenated agents halothane, enflurane and isoflurane, which are widely used for maintenance of anesthesia, are all found to cause a dose-related amplitude reduction and latency increase of cortical SEPs. Against a background of nitrous oxide, Pathak *et al* found halothane to abolish the cortical SEP at 1.0 MAC (minimum alveolar concentration) while equivalent concentrations of the other agents caused about 85% attenuation[26]. Peterson *et al*, on the other hand, found halothane to have the least effect, causing 80% attenuation at 1.5 MAC while isoflurane caused 95% attenuation[27]. All the halogenated agents prolong the central conduction time in a dose-dependent manner. In the study of Wang *et al*, 0.5% halothane increased the CCT (central conduction time) from a mean of 6.0 ms to 6.7 ms, while 2.0% halothane resulted in a further increase of up to 2 ms, independent of the degree of induced hypotension[38]. Subcortically generated SEPs components and propagated spinal cord potentials are affected minimally if at all by volatile anesthetics[9,31,40], which is one of their chief adventages for monitoring. However, potentials recorded from the lumbar spinal cord in sheep were attenuated by a mean of 35-40% by halothane in the high concentrations of 2% or 3%, with augmentation occasionally seen at 1%[2]. In the present study, isoflurane did not make significant changes in SEPs.

In man, the 'N1' segmental dorsal horn potential to be enhanced by thiopental, ketamine and the halogenated agents, but attenuated by morphine and fentanyl[21]. In this experiment, there were no alterations in SEPs caused by ketamine.

Continuous infusion of narcotics provides stable recordings, whereas bolus injections can affect both the evoked potentials and the wake-up test[26]. Kalkman *et al* recommended an alfentanil-propofol anesthetic technique for signal enhancement. In this experiment, propofol did not alter the SEPs[16].

Adjunct drugs administered during anesthesia, for example muscle relaxants, cardiovascular agents, antibiotics and phenytoin, appear to have little direct effect on SEPs[30]. In the present study, antibiotics were administered to the all dogs prior to experiment.

For measuring SEPs, anesthetic maintenance time was recorded to find out the availability of anesthetic protocol. Anesthetic maintenance period should be at least 25 minutes for recording SEPs. The anesthetic maintenance time of Acepromazine + Thiopental Na + Isoflurane, and Acepromazine + Propofol + Isoflurane could be controlled by means of

inhalation anesthesia. In addition, that of Acepromazine + Propofol infusion, and Propofol infusion could be controlled by means of infusion anesthesia. The anesthetic maintenance time of Diazepam + Xylazine, and Xylazine + Ketamine using injectable anesthetics were 32 and 35 minutes, respectively. Therefore, all of these six groups had enough anesthetic maintenance time to measure SEPs.

SEPs components between the anesthetic method using Acepromazine + Thiopental Na + Isoflurane and the other anesthetic methods was compared. In the latency, SP-LN1 and SP-TP1 of group Acepromazine + Propofol + Isoflurane, Ch1-Ch2 of group Diazepam + Xylazine, Ch1-Ch2 of group Xylazine + Ketamine and SP-LP1, Ch1-Ch2 of group Acepromazine + Propofol infusion were significantly different from those of group Acepromazine + Thiopental Na + Isoflurane ($p < 0.05$). In the conduction velocity, SP-LN1 of group Acepromazine + Propofol + Isoflurane, Ch1-Ch2 of group Diazepam + Xylazine, Ch1-Ch2 of group Xylazine + Ketamine, SP-LP1 of group Acepromazine + Propofol infusion and SP-LN1 of group Propofol infusion had no significant differece compared to group Acepromazine + Thiopental Na + Isoflurane ($p < 0.05$). The differences of latency and conduction velocity among six groups were minimal compared to general waveform of SEPs and did not alter Ch 1 with Ch 2. Therefore, the alteration of SEPs among these anesthetic protocols was not recognized.

Conclusion

The effect of the thirteen kinds of anesthetic methods on SEPs recording in dogs were examined. The six anesthetic methods out of them were satisfactory for the measurement of SEPs.

Two inhalation anesthetic protocol, Acepromazine + Thiopental Na + Isoflurane and Acepromazine + Propofol + Isoflurane, and two injectable anesthetic method, Diazepam + Xylazine and Xylazine + Ketamine, and two infusion anesthetic method, Acepromazine + Propofol infusion and Propofol infusion can be used for measuring SEPs by sufficient anesthetic depth.

Although all the above six methods are useful, for SEPs recording, Diazepam + Xylazine and Xylazine + Ketamine, the injectable anesthetics, are considered more convenient than the other four methods.

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마취제가 개의 Somatosensory Evoked Potentials (SEPs)에 미치는 영향

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요 약 : 본 실험은 각각의 마취방법이 체성감각유발전위 (SEPs) 파형에 미치는 영향을 알아보고 SEP의 측정에 적절한 마취방법을 찾고자 시행하였다. 임상적으로 건강하고 크기와 나이가 비슷한 다섯 마리의 잡종견을 대상으로 SEPs를 측정하고 각각의 측정값을 분석하였다. SEPs 측정을 위해 후 경골신경을 자극하였고 요추 5-6번 사이에서 channel 1의 LP1과 LN1, 흉추 11-12사이에서 channel 2의 TP1, TN1을 기록하였다. 실험에 사용한 마취방법 중, Acepromazine + Thiopental Na + Isoflurane, Acepromazine + Propofol + Isoflurane, Diazepam + Xylazine, Xylazine + Ketamine, Acepromazine + Propofol infusion, 및 Propofol infusion 등의 방법만이 SEPs 측정이 가능하였고, 파형은 명확하였으며, 측정에 요구되어지는 일정 시간인 25분 이상 동안 마취 유지가 가능하였다. 또한 각 마취군에서의 SEPs 파형을 Acepromazine + Thiopental Na + Isoflurane군과 비교해 보았을 때 latency의 경우, Acepromazine + Propofol + Isoflurane군의 ST(stimulating point)-LN1, SP-TP1, Diazepam + Xylazine 군의 Ch1-Ch2, Xylazine + Ketamine군의 Ch1-Ch2, Acepromazine + Propofol infusion군의 ST-LP1와 Ch1-Ch2에서 부분적으로 유의적인 차가 있었다. Conduction velocity의 경우, Acepromazine + Propofol + Isoflurane군의 ST-LN1, Diazepam + Xylazine군의 Ch1-Ch2, Xylazine + Ketamine군의 Ch1-Ch2, Acepromazine + Propofol infusion군의 ST-LP1, 그리고 Propofol infusion군의 ST-LN1의 측정값에서 유의적인 차가 있었지만 전반적으로는 전체적인 파형의 유의적인 변화는 없었다. 이상의 결과를 토대로 SEPs 측정시 흡입마취로는 Acepromazine + Thiopental + Isoflurane과 Acepromazine + Propofol + Isoflurane, 주사마취로는 Diazepam + Xylazine과 Xylazine + Ketamine, 점적마취로는 Acepromazine + Propofol infusion과 Propofol infusion 방법이 사용 가능한 것으로 확인되었다.

주요어 : 개, 체성감각유발전위, 마취