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ORIGINAL ARTICLE

# Transesophageal motor-evoked potentials, a novel method induced by transesophageal spinal cord stimulation, are less sensitive to anesthetics than transcranial motor-evoked potentials

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## Abstract

**Purpose** Intraoperative neurologic monitoring can be useful, but transcranial motor evoked potentials (TcMEPs) are sensitive to anesthetic agents. We compared the effects of anesthetics on the newly developed transesophageal motor evoked potentials (TeMEPs) with those on TcMEPs.

**Methods** Eleven pigs (25.6 ± 0.8 kg) were anesthetized by desflurane inhalation, remifentanil was maintained at 0.5 μg/kg/min until the end of the experiment. End-tidal desflurane concentration was then maintained at 7, 4, 10, and 13%, and TcMEPs and TeMEPs were measured at each concentration. Desflurane was then discontinued and propofol was infused at 10, 20, 40, and 60 mg/kg/h, and TcMEPs and TeMEPs were measured at each infusion dose. An electroencephalogram monitor was used to measure the hypnotic level.

**Results** Both desflurane and propofol anesthesia decreased bispectral index in a dose-dependent manner ($P < 0.0001$), replicating shallow (or adequate) to deep hypnotic levels in both anesthetic methods. The amplitude of TeMEPs was clearly larger than that of TcMEPs and was significantly larger at all anesthetic depths and all limb sites ($P < 0.0001$). Amplitudes of the lower extremities were lower than those of the upper extremities ($P < 0.0001$) for both TcMEPs and TeMEPs, but the amplitudes of TeMEPs were sufficiently large under desflurane as under propofol. The trend of concentration-dependent decrease in the amplitudes of TeMEPs under both anesthetics was not as apparent as in that of TcMEPs.

**Conclusions** TeMEPs are more tolerant to anesthesia than TcMEPs and may be a promising MEP monitoring technique for the lower corticospinal tract.

**Keywords** Intraoperative neurologic monitoring · Motor evoked potentials · Transcranial motor evoked potentials · Transesophageal motor evoked potentials

## Introduction

Neurological monitoring has been widely used to avoid neurological complications during surgery, and the accuracy of the monitoring is key to the precision of the surgical procedure [1–5]. Neurological monitoring is often performed under general anesthesia but is influenced by anesthetic drugs, making the choice of anesthetics and their dosage problematic [6–8].

Intraoperative monitoring of the corticospinal tract (CST) is commonly performed with motor evoked potentials (MEPs), where the motor cortex of the cerebrum is electrically stimulated and the impulses descending the CST are recorded as action potentials from the dominant muscle. Anesthetic management capable of detecting changes in the amplitudes of MEPs is required because the amplitudes of the muscle action potential waveform are monitored as a danger signal to the CST [4, 5], as indicated by its decreasing amplitudes [4, 5]. Most hypnotic drugs used in anesthesia act on synapses to reduce MEPs amplitudes in a dose-dependent manner. However, propofol, which is less likely to suppress evoked potentials (and thus produces larger MEPs amplitudes), is the first choice, and furthermore, anesthesiologists are required to keep the anesthetic depth as constant as possible to reduce the changes in amplitudes [6–8].

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Recently, transesophageal motor evoked potentials (TeMEPs), in which a special probe is inserted through the oral cavity to stimulate the spinal cord through the esophagus, have been newly developed [9–12]. This method cannot be used for monitoring brain function, such as intracranial lesions, because the stimulation site is the spinal cord, but it may be useful for spinal cord monitoring. In fact, TeMEPs has been reported to have a better response to spinal cord ischemia than TcMEPs [10, 12], with less variability in amplitudes [9] and fewer false positives [11]. As of September 2024, a phase III clinical trial is underway to evaluate the usefulness and safety of TeMEPs compared to TcMEPs as an intraoperative spinal cord monitoring method in patients undergoing aortic surgery who are at risk of spinal cord ischemia.

There are no reports on how sensitive TeMEPs are to the hypnotic drugs required to maintain adequate depth of anesthesia. This study was designed to compare TeMEPs to TcMEPs in large animal pigs, replicating shallow to deep anesthetic depths using propofol and desflurane, two drugs that are likely to be used as hypnotics in clinical MEPs monitoring [8]. We hypothesized that compared to TcMEPs, TeMEPs being a monitoring from the spinal cord to the peripheral muscle that omits the motor cortex to spinal cord pathway of the CST would be less influenced by hypnotic drugs than TcMEPs.

## Methods

### Animal preparation

This study was approved by the Institutional Animal Care and Use Committee of Hamamatsu University School of Medicine (approval number 23-087-01) on 8 April 2024. All experiments were conducted in accordance with the Animal Research Reporting of In Vivo Experiments guidelines. Eleven female swine, each weighing 25.6 ± 0.8 kg (mean ± SD; range: 24.4–26.6 kg) and approximately 2 months old, were studied in compliance with the relevant guidelines and regulations. The animals were provided with unrestricted access to water and food before the experiments. General anesthesia was induced via inhalation of 13% desflurane and oxygen, followed by tracheostomy using local anesthetics. General anesthesia was maintained with 7% end-tidal desflurane concentration (EtDes) alongside 1 l/min oxygen and 1 l/min air through mechanical ventilators. Exhaled gases were analyzed using an IntelliVue G5-M1019A (Philips Medical Systems, Eindhoven, The Netherlands). The ventilator was adjusted to maintain the end-tidal carbon dioxide partial pressure of 35–40 mmHg throughout the study. Electrocardiographic lead II was monitored using three cutaneous electrodes, and arterial blood pressure was measured by inserting a 16-gauge needle into the right femoral artery. The 12-gauge triple-lumen catheter was inserted into the right jugular vein, and remifentanil at 0.5 μg/kg/min was immediately administered continuously through the catheter until the end of the experiment. Local anesthetics were used for all catheter placements. After these preparation steps, EEG monitoring was started by positioning four cutaneous electrodes over the bilateral frontal occipital regions. Four channels of the EEG were amplified and digitally recorded, and processed EEG values (bispectral index: BIS and 95% spectral edge frequency: SEF) were collected electronically at intervals of 12 s. Each sensor was connected to an IntelliVue MX800 monitoring system (Philips Japan Medical Systems, Tokyo, Japan) to record heart rate (HR), mean arterial pressure (MAP) and EEG data. Nasal temperature was monitored and maintained at normal temperature in pigs with an electric heater and air conditioning. Saline was infused at 100 ml/h as a maintenance infusion.

### TcMEPs and TeMEPs monitoring

Neuromaster MEE 1232 Stimulator (Nihon Kohden, Tokyo, Japan) was used for both MEPs measurements. Transcranial stimulation was delivered via a pair of electrodes (cathode and anode), and corkscrew electrodes were placed in each parietal region, 2.0 cm above the eye and 1.0 cm lateral to the midline. Transesophageal stimulation was performed by inserting a custom-made silicon probe (UNIQUE MEDICAL, Tokyo, Japan) (Fig. 1) with a pair of electrodes (cathode and anode) on the tip into the mouth to a depth where the electrodes reached from the lower cervical vertebra to upper thoracic spine (Fig. 2). The probe depth was determined based on imaging in the pilot study to be as close as possible to the spinal cord. The evoked muscles were bilateral triceps brachii in the upper limbs and bilateral femoral biceps in the lower limbs, as in our previous study [13]. We used these proximal muscles because distal muscles were not optimal for sensing TcMEPs responses in our animals, and the triceps brachii and femoral biceps were the most appropriate for obtaining maximal TcMEPs amplitudes under baseline conditions in each animal. MEPs responses were recorded using a pair of subdermal needle electrodes (active and reference). The active electrode (cathode) was positioned over the muscle belly, while the reference electrode (anode) was placed over the corresponding tendon. Both transcranial and transesophageal stimuli were performed with a stimulus intensity of 200 mA (maximum stimulus intensity of Neuromaster MEE 1232 Stimulator), a pulse duration of 0.5 ms, and five train stimuli were delivered with an inter-stimulus interval of 2 ms. To compare amplitudes between both MEPs, the stimulus intensity and technique did not change throughout the study for each animal.

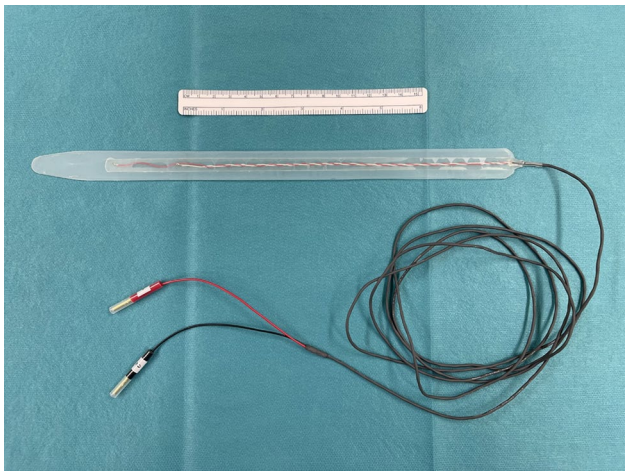

**Fig. 1** The silicon probe for transesophageal motor evoked potentials with two electrodes, an anode, and a cathode, placed at the tip (a custom-made device; UNIQUE MEDICAL, Tokyo, Japan)

## Experimental protocol

After animal preparation, TcMEPs were measured twice consecutively steps under 7% EtDes. Five minutes later, TeMEPs were measured twice consecutively. Five minutes after that, TcMEPs were measured twice consecutively again, and five minutes later, TeMEPs were measured twice consecutively (resulting in four measurements of TcMEPs and TeMEPs at the same anesthetic depth). After measurements under 7% EtDes, the EtDes was changed to 4% and 10 min later, TcMEPs and TeMEPs were measured in the same manner. The EtDes was further changed to 10% and 13%, and TcMEPs and TeMEPs were measured in the same manner at each concentration.

Desflurane inhalation was then discontinued and 1% propofol was started at 25 ml/h (10 mg/kg/h in a 25 kg pig). 30 min after the start of propofol administration, TcMEPs and TeMEPs were measured four times each, as they were at each desflurane concentration. Propofol was then increased to 50 ml/h and 20 min later, TcMEPs and TeMEPs were measured in the same manner. TcMEPs and TeMEPs were also measured at 100 ml/h and 150 ml/h in the same manner. After the completion of the study, the animals were euthanized by stopping the ventilator under the administration of 150 ml/h of propofol.

## Statistical analysis

Data are presented as means ± SDs or median (75th-25th percentile). A repeated-measures one-way analysis of variance (ANOVA) was employed to analyze the differences in HR, MAP, BIS and SEF across each anesthetic depth. If the ANOVA indicated significant differences, the Scheffé F-test for multiple comparisons was performed. The Kruskal–Wallis test was used to analyze the differences in MEPs amplitudes among each limb and between each anesthetic depth. If the Kruskal–Wallis test indicated significant differences, a Steel–Dwass test for multiple comparisons was performed. Statistical significance was set at $P < 0.05$.

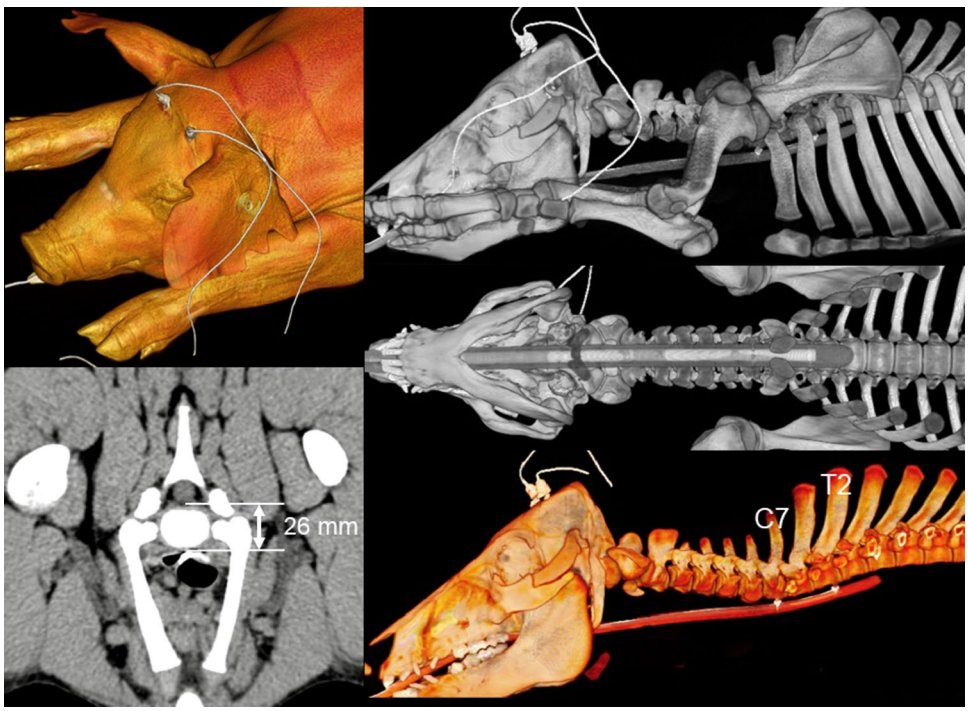


**Fig. 2** The computed tomography image of a pig with the placement of corkscrew electrodes for transcranial motor evoked potentials and a probe for transesophageal motor evoked potentials. The anode is positioned at the level of the second thoracic vertebra (T2) and the cathode is positioned at the level of the seventh cervical vertebra (C7). Distance from the probe to the spinal cord is approximately 26 mm

## Results

All pigs survived until the end of the study. HR, MAP, BIS, and SEF are presented in Table 1. HR, MAP and BIS decreased in a dose-dependent manner with both anesthetic methods. SEF decreased in a dose-dependent manner up to 10% desflurane but remained low and unchanged with increasing doses of propofol.

Although both TcMEPs and TeMEPs induced body movements in pigs, TeMEPs clearly elicited greater movements than TcMEPs. There were no instances of TeMEPs failure or obvious adverse events. The probe depth was set as close as possible to the spinal cord based on imaging in the pilot study, but there was no significant difference in TeMEPs amplitude with slight variations in probe depth. Additionally, in humans, shallow probe placement may cause strenuous upper limb movements due to cervical alpha motor neurons stimulation [11], but in pigs, no upper limb movements were observed.

**Table 1** Heart rate (HR), mean arterial pressure (MAP), bispectral index (BIS) and 95% spectral edge frequency (SEF) at each anesthetic depth

| | HR (beats/min) | MAP (mmHg) | BIS | SEF (Hz) |
|---|---|---|---|---|
| Desflurane | | | | |
| 4% | 201 ± 40 | 115 ± 20 | 81 ± 6 | 21 ± 2 |
| 7% | 151 ± 35[a] | 76 ± 17[a] | 70 ± 7[a] | 18 ± 3[a] |
| 10% | 150 ± 26[a] | 57 ± 11[b] | 48 ± 14[b] | 15 ± 2[b] |
| 13% | 139 ± 21[a] | 53 ± 11[b] | 20 ± 12[c] | 16 ± 5[a] |
| *P*-value | < 0.0001 | < 0.0001 | < 0.0001 | < 0.0001 |
| Propofol | | | | |
| 10 mg/kg/h | 209 ± 46 | 95 ± 17 | 73 ± 8 | 19 ± 3 |
| 20 mg/kg/h | 197 ± 45 | 96 ± 20 | 64 ± 9[a] | 17 ± 2 |
| 40 mg/kg/h | 172 ± 44[a] | 91 ± 16 | 33 ± 16[b] | 18 ± 6 |
| 60 mg/kg/h | 150 ± 44[b] | 71 ± 15[c] | 7 ± 8[c] | 17 ± 6 |
| *P*-value | < 0.0001 | < 0.0001 | < 0.0001 | 0.045 |

Data are expressed as mean values ± SD. *P*-value indicates the significance between each anesthetic depth

[a]Significant difference vs. each variable at 4% or 10 mg/kg/h

[b]Significant difference vs. each variable at 4 and 7% or 10 and 20 mg/kg/h

[c]Significant difference vs. all other anesthetic depth

Figure 3 shows one of the typical TcMEPs and TeMEPs measurements in the present study. In general, TcMEPs were clearly smaller in amplitude than TeMEPs and were easily suppressed with increasing doses of hypnotics, whereas TeMEPs were not suppressed at any anesthetic depth. Specifically, the amplitudes of TcMEPs and TeMEPs at each anesthetic depth are shown in Tables 2 and 3. The amplitudes of TcMEPs decreased in a dose-dependent manner under both desflurane and propofol anesthesia. Amplitudes of TcMEPs in the lower extremities were lower than those in the upper extremities ($P < 0.0001$). Desflurane anesthesia suppressed TcMEPs amplitude more strongly than propofol anesthesia, and at 13%, lower extremity amplitudes were absent in all animals. In contrast, the amplitudes of TeMEPs were clearly larger than those of TcMEPs and were significantly larger at all desflurane concentrations, all propofol doses, and all limb sites ($P < 0.0001$).

Amplitudes of TeMEPs in the lower extremities were lower than in the upper extremities ($P < 0.0001$), similar to TcMEPs. However, the amplitudes were sufficiently large under desflurane anesthesia as under propofol anesthesia (153 [343-30] [median {75th-25th percentile}] in the left lower limb and 39 [196-23] in the right lower limb under 13% EtDes, and 122 [369-32] and 105 [148-61] under 60 mg/kg/h of propofol, respectively). A dose-dependent trend was observed in TeMEPs under both anesthesia methods

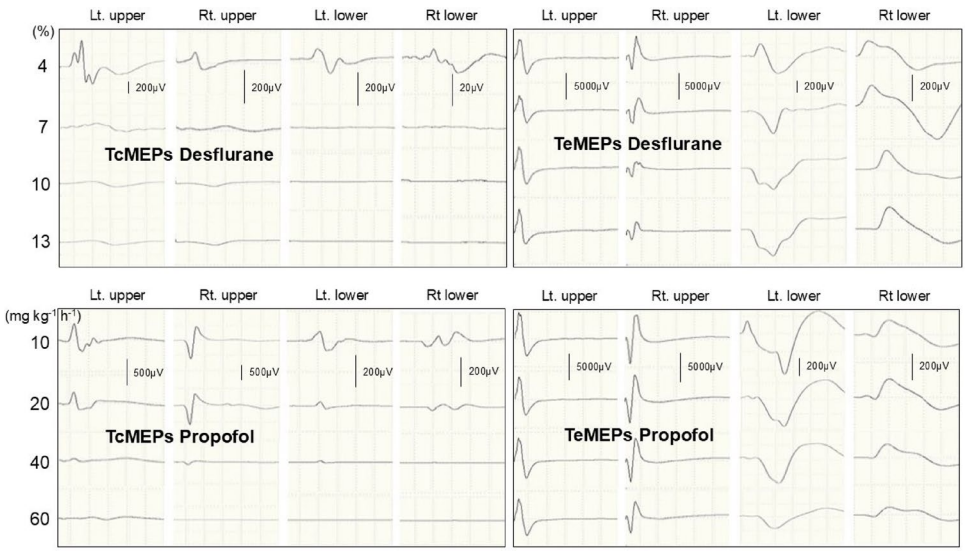


**Fig. 3** Changes in transcranial and transesophageal motor evoked potentials (TcMEPs and TeMEPs) waveforms under each hypnotic dose. Vertical voltages are different for each, but the same between the four hypnotic doses of each anesthetic method at the same limb site. *Lt. (or Rt.) upper* Left (or Right) upper limb; *Lt. (or Rt.) lower* Left (or Right) lower limb

**Table 2** The amplitudes (μV) of transcranial motor evoked potentials (TcMEPs) at each anesthetic depth

| | Left upper limb | Right upper limb | Left lower limb | Right lower limb |
|---|---|---|---|---|
| Desflurane | | | | |
| 4% | 16 (41–9) | 32 (51–16) | 0 (15–0) | 0 (12–0) |
| 7% | 16 (28–8) | 16 (26–10)[a] | 0 (6–0) | 0 (0–0)[a] |
| 10% | 15 (60–5) | 14 (23–5)[a] | 0 (0–0)[b] | 0 (0–0)[a] |
| 13% | 12 (69–0) | 12 (19–5)[a] | 0 (0–0)[b] | 0 (0–0)[b] |
| *P*-value | 0.419 | < 0.0001 | < 0.0001 | < 0.0001 |
| Propofol | | | | |
| 10 mg/kg/h | 202 (430–91) | 80 (262–34) | 67 (160–12) | 24 (55–13) |
| 20 mg/kg/h | 114 (249–40) | 72 (337–24) | 13 (62–0) [a] | 8 (18–0)[a] |
| 40 mg/kg/h | 25 (85–9) [b] | 38 (63–17) [b] | 0 (6–0)[b] | 0 (0–0)[b] |
| 60 mg/kg/h | 17 (42–6) [b] | 20 (34–9)[c] | 0 (4–0)[b] | 0 (0–0)[b] |
| *P* value | < 0.0001 | < 0.0001 | < 0.0001 | < 0.0001 |

Data are expressed as median values (75th-25th percentile). P-value indicates the significance of dose dependence for each limb

[a]Significant difference vs. each limb at 4% or 10 mg/kg/h

[b]Significant difference vs. each limb at 4 and 7% or 10 and 20 mg/kg/h

[c]Significant difference vs. all other propofol doses

**Table 3** The amplitudes (μV) of transesophageal motor evoked potentials (TeMEPs) at each anesthetic depth

| | Left upper limb | Right upper limb | Left lower limb | Right lower limb |
|---|---|---|---|---|
| Desflurane | | | | |
| 4% | 5640 (7935–2427) | 3074 (5085–1469) | 215 (481–49) | 127 (261–37) |
| 7% | 6760 (9760–4664) | 3699 (5820–1970) | 251 (479–76) | 105 (323–55) |
| 10% | 5455 (8335–2731) | 2360 (4115–1440) | 132 (326–34) | 107 (310–27) |
| 13% | 4930 (6153–2685)[a] | 2285 (4201–609)[a] | 153 (343–30) | 39 (196–23) |
| *P* value | 0.025 | 0.025 | 0.075 | 0.126 |
| Propofol | | | | |
| 10 mg/kg/h | 5275 (6883–2900) | 4570 (5334–1543) | 356 (617–216) | 272 (538–154) |
| 20 mg/kg/h | 4843 (5605–2901) | 4799 (5245–1625) | 493 (794–105) | 189 (830–164) |
| 40 mg/kg/h | 4247 (5430–2978) | 3830 (5195–1442) | 240 (569–46) | 123 (182–95) [b] |
| 60 mg/kg/h | 4195 (5395–2333) | 2935 (4803–920) | 122 (369–32)[b] | 105 (148–61)[b] |
| *P* value | 0.058 | 0.096 | < 0.0001 | < 0.0001 |

Data are expressed as median values (75th-25th percentile). P-value indicates the significance of dose-dependence for each limb

[a]Significant difference vs. each limb at 7%

[b]Significant difference vs. each limb at 10 and 20 mg/kg/h

which was more pronounced in the lower extremities than in the upper extremities, though less apparent than with TcMEPs.

## Discussion

The present study demonstrated that TeMEPs have larger amplitudes at any depth of anesthesia for both desflurane and propofol anesthetics than TcMEPs stimulated at the same stimulus intensity. Additionally, for these anesthetics, the dose-dependent decrease in MEPs amplitudes was not as pronounced for TeMEPs as for TcMEPs.

This experiment varied the doses of hypnotics, which can significantly affect MEPs and examined their effects on MEPs at relatively shallow to deep anesthetic depths. Although the doses of both desflurane and propofol were higher compared to clinical use, hypnotic levels were accurately assessed by EEG and allowing for the reproduction of a wide range of anesthetic depths. While there is no formal validation of BIS use in pigs, our previous pharmacokinetic-pharmacodynamic experiments in pigs suggest that BIS and SEF can accurately assess hypnotic levels in pigs [14–17].

SEF values decrease with increasing depth of anesthesia in both pigs [15, 16] and humans [18], but both tend to plateau around 15 Hz and above. When evaluating SEF and BIS values, desflurane anesthesia produces shallow-to-deep anesthesia, while propofol anesthesia produces nearly adequate depth-to-deep anesthesia. This study allowed us to investigate the effects of both anesthetic methods on each type of MEPs at appropriate anesthetic depths, reflecting clinical practice.

TeMEPs is a method of electrically stimulating the spinal cord and recording evoked potentials from peripheral muscles, similar in concept to spinal cord stimulation motor evoked potentials (SpMEPs) [19]. In SpMEPs, electrodes are inserted into the epidural space to stimulate the spinal cord, but in TeMEPs, the spinal cord is stimulated noninvasively from the esophageal (ventral) side, rather than dorsally as in SpMEPs. TeMEPs were first introduced in 2015 by cardiovascular surgeon Tsuda et al. with the final goal of detecting spinal cord ischemia in aortic surgery more accurately than TcMEPs [9]. They demonstrated using dogs, that TeMEPs required less stimulus intensity for maximal supramaximal stimulation and exhibited less variability in the amplitudes of individual stimuli compared to TcMEPs [9]. Similarly, using dogs, they showed that TeMEPs responded quicker than TcMEPs to spinal cord ischemia induced by aortic balloon dilation at the level of the 8-10th thoracic vertebrae, and was a better predictor of prognosis for paraplegia [10]. When TeMEPs were first designed, it utilized monophasic stimulation with an electrode inserted into the esophagus as the cathode and an electrode placed subcutaneously on the 4-5th thoracic vertebrae as the anode. However, the group later adopted biphasic stimulation, as used in this experiment, with two electrodes placed on the tip of a probe positioned in the esophagus. They showed that TeMEPs were clinically feasible and safe with a lower false-positive rate of spinal cord ischemia than TcMEPs in 18 patients underwent descending aortic or thoracoabdominal aortic repair [11]. In dogs, biphasic stimulation of TeMEPs, like monopolar stimulation, has also been reported to produce a more stable and rapid response to spinal cord ischemia than TcMEPs [12]. As of September 2024, a multicenter phase III clinical trial is underway to evaluate the utility and safety of TeMEP compared to TcMEP as an intraoperative spinal cord monitoring method in a larger number of aortic surgery patients at risk of spinal cord ischemia.

TeMEPs achieved such large amplitudes compared to TcMEPs due to differences in how they stimulate the nervous system. When the cortical motor cortex is stimulated transcranially with TcMEPs, D waves are generated by direct stimulation of the axons of subcortical pyramidal cells, and I waves are generated by stimulation of interneurons synaptically connected to the pyramidal cells. When these waves reach the anterior horn cells of the spinal cord, postsynaptic potentials accumulate, raising the cell membrane potentials, and when thresholds are exceeded, the anterior horn cells fire, evoking compound muscle action potentials. Because general anesthetics act on synapses, I waves are suppressed and excitatory postsynaptic potentials do not accumulate in the anterior horn cells of the spinal cord, train pulse stimulation is used to force the accumulation of excitatory postsynaptic potentials and fire the motor nerves. TeMEPs, on the other hand, evoke responses in peripheral muscles directly from the anterior horn cells of the spinal cord by bypassing the pathway from the motor cortex to the anterior horn cells and stimulating the spinal cord directly from the esophagus via the vertebral body. TeMEPs can stimulate the spinal cord and thus can provide stronger stimulation to the anterior horn cells of the spinal cord than transcranial stimulation, without being affected by the attenuation of I waves.

In the present study, TcMEPs did not produce sufficient amplitudes at all desflurane concentrations and at higher propofol doses, consistent with clinical observations. Typically, MEPs amplitude in the lower extremities are lower than in the upper extremities, and TcMEP cannot be used for spinal cord monitoring if the lower extremity amplitude is absent in baseline measurements. Our findings indicate that TcMEPs are challenging to perform under desflurane anesthesia in most cases. When TcMEPs is used for neurologic monitoring, propofol should be chosen over desflurane, and it is important to maintain a constant depth of anesthesia without overdosing [8]. In contrast, TeMEPs can achieve amplitudes similar to those seen with propofol even with desflurane anesthesia and are more tolerant of the changes in anesthetic depth. TeMEPs have the potential to minimize the influence of anesthesia on the MEPs amplitudes and may offer anesthesiologists greater flexibility in anesthetic choice and depth, as compared to TcMEPs.

There are several limitations in this study. First, MEPs measurements with propofol anesthesia were performed after desflurane anesthesia. To reduce the effect of residual propofol on MEPs measurements under desflurane anesthesia, measurements with desflurane anesthesia, which has a faster washout, were performed first. However, $0.6 \pm 0.2\%$ EtDes remained at the beginning of each MEPs measurement under propofol anesthesia, the results might differ if MEPs measurements had been performed under propofol anesthesia from the start. Additionally, this experiment measured MEPs under the administration of remifentanil. Although analgesics are considered to have little effect on MEPs [8], it is unclear how much the 0.5 μg/kg/min of remifentanil affected MEPs. Furthermore, this experiment was conducted on healthy animals without spinal cord abnormalities and used proximal muscles as the evoked muscles. In humans, proximal muscles are more susceptible to anesthetics than distal muscles [20]; therefore, our findings may not fully reflect actual clinical conditions or surgical

patients. Finally, a potential problem with TeMEPs itself is the risk of esophageal mucosal injury; Tsuda et al. found no damage to the esophageal mucosa in two dogs after the experiment [9], but this needs further verification.

In conclusion, TeMEPs are more tolerant of anesthetics and depth of anesthesia than TcMEPs, which is currently commonly performed and may reduce false positives due to anesthetics. It may be useful as one method of MEPs monitoring, although its safety to the esophageal mucosa and usefulness for spinal and spinal cord disease need to be investigated further.

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**Data availability** The data supporting the findings of this study is available from the corresponding author upon reasonable request.

## Declarations

**Conflict of interest** All authors have no conflicts of interests.

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