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Power spectral analysis of the electroencephalogram during induced total spinal block

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Abstract
Purpose. To evaluate the effects of total spinal block (TSB) on brain function, TSB-induced changes in cortical electrical activities were analyzed using power spectral analysis of an electroencephalogram (EEG).
Methods. Six patients suffering from chronic pain who were undergoing TSB therapy were studied. TSB was established with intrathecal 1% lidocaine ($0.3\,ml\cdot kg^{-1}$) injected through the C1–2 lateral intervertebral space. Mechanical ventilation was continued via a laryngeal mask until the recovery of respiration. The EEG recording was started before TSB induction and continued until 10 min after extubation. The following processed EEG parameters were monitored: spectral edge frequency-90% (SEF90), spectral median frequency (SMF), and relative power in the frequency bands of δ, θ, α, β, and the δ ratio [(α + β)/δ].
Results. TSB induced an unconscious state more than 40 min in all patients. During TSB, 12–13 Hz in SEF90 and 6–7 Hz in SMF were observed. These values are consistent with the previously reported prearousal threshold from general anesthesia. The other EEG descriptors did not change during the TSB-induced unconscious state.
Conclusion. The dissociation of cortical electrical activities and the clinical coma-like condition may be characteristic of the TSB-induced unconscious state.

Key words Total spinal block · Electroencephalogram · Power spectral analysis · Chronic pain

Introduction

Induced total spinal block (TSB) was historically used for anesthesia during surgical procedures in the upper part of the body, including the neck, head, and thorax [1]. Since Tsumura et al. first described the resulting relief of pain in posttraumatic cervical syndrome in 1971 [2], TSB has been applied to relieve various types of neck- or head-related persistent pain that do not respond to conventional pharmacological or nonpharmacological treatment. In general, TSB is induced with the injection of 1%–1.5% mepivacaine or lidocaine at a volume of $0.3–0.4\,ml\cdot kg^{-1}$ into the cervical subarachnoidal space. The injection of the local anesthetic immediately produces a coma-like condition in the patient, so that adequate ventilatory support is necessary until the recovery of consciousness. Although the Ministry of Public Welfare in Japan has accepted TSB as a standard therapy for persistent pain, the underlying mechanism of pain relief in this procedure still remains unclear.

Recent evidence has strongly suggested the supraspinal modulation of pain perception [3–5]. Various clinical interventions that target the central nervous system (CNS) have been reported to relieve intractable pain [6–9]. Since TSB clearly targets supraspinal organs and produces the characteristic "coma-like" unconscious state in patients, we assumed that the impact of TSB on the brain is involved in the mechanism of pain relief. The aim of the present study was, therefore, to evaluate the TSB-induced changes in central nervous activities. To achieve this purpose, we used power spectral analysis of an electroencephalogram (EEG). This analysis has been applied to evaluate the changes in the cortical electrical activities induced by general anesthetics and is reported to have high sensitivity and specificity for the prediction of arousal from anesthesia [10–12].

Patients and methods

After we had obtained the approval of our institutional ethical committee and written informed consent from the patients, six patients (three women and three men, aged 22–52 years) undergoing TSB therapy were enrolled in this study. All of the patients suffered from

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chronic pain in various upper body parts, including the head, neck, and limbs. Multiple medications and nerve-blocking interventions applied in our pain clinic provided only limited pain relief. The present intensity of pain in each patient was evaluated by using a visual analog scale (VAS) ranging from 0 (no pain) to 100 (the worst pain imaginable) before TSB induction without any daily medication or intervention. VASs were also recorded a day after TSB and subsequently in the follow-up period.

The patients were premedicated with intramuscular atropine ($0.01\,mg \cdot kg^{-1}$) 30–45 min prior to TSB. An intravenous catheter was inserted into the unaffected forearm vein before the treatment. The patients were placed in the supine position. An electrocardiogram, SpO_2, and noninvasive blood pressure were continuously monitored before the induction of TSB and until the full recovery of the patients. TSB was established with an intrathecal injection of 1% lidocaine at a volume of $0.3\,ml \cdot kg^{-1}$ using a 23-G needle advanced through the C1–2 lateral intervertebral space under X-ray vision. This approach, with a safe access to the subarachnoidal space, has been used for transcutaneus cordotomy [13]. To avoid possible discomfort before the complete loss of consciousness, the patients were given propofol intravenously at the minimal sleeping dose ($0.8–1.4\,mg \cdot kg^{-1}$) simultaneously with the injection of lidocaine. A laryngeal mask was placed immediately after the patients lost consciousness, and artificial ventilation was maintained until the recovery of sufficient spontaneous breathing.

For two-channel continuous recording of the EEG, silver/silver chloride cup electrodes were placed at C3–4 (positive) and A1–2 (negative) against the forehead as a common ground (FPZ), according to the international 10–20 system. The impedance of all electrodes was maintained at less than 3000 Ω. The EEG was recorded, analyzed, and stored by a p-EEG monitor system (Dräger, Lübeck, Germany). Bandpass filters were set at 0.5–30 Hz, and the amplifier sensitivity was 200 μV. On-line power spectral analysis was performed by fast Fourier transformation. The epoch length for EEG acquisition was 2 s. The power spectrum was displayed as density spectral array. The following quantitative EEG parameters were generated by the p-EEG system: spectral edge frequency-90% (SEF90), spectral median frequency (SMF), and relative power in the frequency bands of δ (0.5–4 Hz), θ (4–8 Hz), α (8–13 Hz), β (13–30 Hz), and the δ-ratio $[(\alpha + \beta)/\delta]$. The EEG recording was started before the induction of TSB and continued until 10 min after the removal of the laryngeal mask. Data from 30 consecutive epochs, excluding artifacts, were averaged in each of the generated parameters at the following time points: before TSB induction; 5, 15, and 30 min after the induction; at eye opening; and 5 min after extubation.

Data were expressed as means ± SD. The changes in the parameters during TSB were analyzed with repeated-measure analysis of variance (ANOVA), and the Wilcoxon signed-ranks test was used to compare values between each time point. *P* values less than 0.05 were considered to indicate statistically significance.

Results

The clinical profiles of the six patients are summarized in Table 1. Needle access to the C1–2 intrathecal space was achieved under local anesthesia without serious bleeding or paresthesia in all patients. During TSB, bradycardia (heart rate <50 bpm) occurred in four of the six patients close to eye opening. They were successfully treated with intravenous atropine, and no other critical arrhythmia was observed throughout the procedure. Blood pressure and SpO_2 were stable throughout TSB (data not shown). The laryngeal mask was removed when each patient showed a full response to verbal commands. The mean time from the injection of

Table 1. Clinical features of six patients and the changes in visual analog scale (VAS) of pain after total spinal block (TSB)

Patient no.	Age/Sex	Diagnosis	VAS change (pre/post-TSB)[a]	Duration of VAS < 50 (mo)
1	25/F	CR	78/8	3
2	22/F	UK	95/22	0.5
3	46/M	CHD	85/40	8
4	52/M	CRPS I	72/34	0
5	42/M	PTCS	77/21	14[b]
6	46/F	CRPS I	91/84	NA

CR, Cervical radiculopathy; UK, unknown source; CDH, chronic daily headache; CRPS I, complex regional pain syndrome type I; PTCS, posttraumatic cervical syndrome; NA, not applicable

[a] Post-TSB VAS is an evaluation on a day after TSB in each patient

[b] Lost to follow-up

lidocaine to the removal of the laryngeal mask was 54.5 ± 9.1 min (range, 42–68). No prolonged impairment of consciousness or respiration was observed. None of the patients felt any discomfort or developed memory during TSB. Five of the six patients experienced satisfactory pain relief (>50% reduction in VAS recorded a day after the procedure) after TSB. Long-lasting pain relief (VAS < 50) was obtained in two of the patients (Table 1).

Figure 1 shows the changes in SEF90 during TSB ($P < 0.0001$). With the induction of TSB, SEF90 significantly decreased from the preinduction value of 19.8 ± 3.1 Hz to 13.8 ± 2.0 Hz at 5 min, and was kept at 12.5 ± 1.8 Hz until 30 min postinduction ($P < 0.05$ at 5 min, $P < 0.01$ at 15 and 30 min *vs* preinduction). It increased to 23.3 ± 2.5 Hz when the patients opened their eyes ($P < 0.05$ *vs* preinduction) and decreased to 17.8 ± 4.8 Hz 5 min after the removal of a laryngeal mask (not significant *vs* pre-induction). The changes in SMF during TSB ($P < 0.011$) are shown in Fig. 2. Although the minimal value of 6.4 ± 2.6 Hz was observed at 30 min after TSB induction, the change in SMF was not statistically significant, except at eye opening (12.9 ± 3.2 Hz, $P < 0.02$ *vs* preinduction value of 8.3 ± 3.7 Hz). The relative band powers and the δ-ratio are presented in Table 2. The relative β power increased to 50.2 ± 19.2% at eye opening as compared with preinduction (28.3 ± 11.4%, $P < 0.02$). However, it did not change significantly during the period the patients were unconscious. The changes in all other relative band powers did not reach statistical significance, with wide deviations throughout TSB.

Discussion

To our knowledge, the processed EEG has not been previously applied to analyze the effects of local anes-

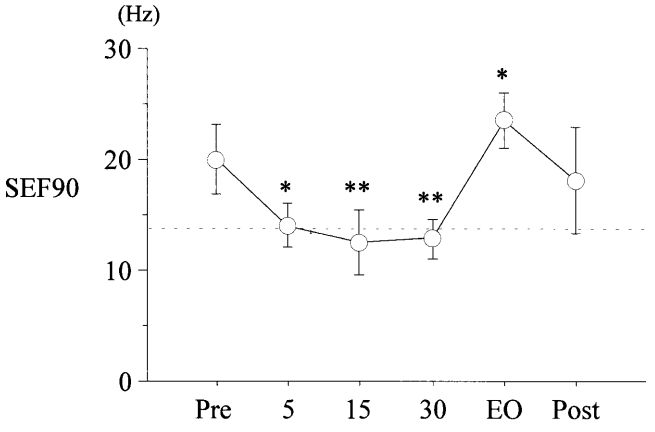

Fig. 1. Changes in spectral edge frequency-90% (*SEF90*) during induced total spinal block (TSB) ($P < 0.0001$). Time points: *Pre*, before TSB induction; *5*, *15*, *30*, minutes after TSB induction; *EO*, at eye opening; *Post*, 5 min after extubation. *$P < 0.05$, **$P < 0.01$ *vs* preinduction value. *Broken line* represents the previously reported arousal threshold from general anesthesia [11, 12]

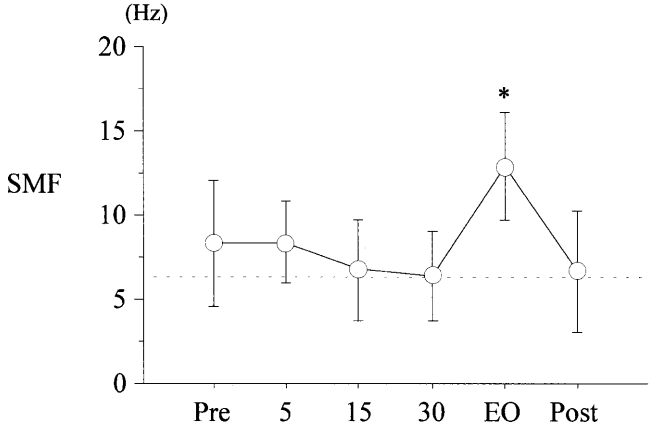

Fig. 2. Changes in spectral median frequency (*SMF*) during induced total spinal block (TSB) ($P < 0.011$). Time points: *Pre*, before TSB induction; *5*, *15*, *30*, minutes after TSB induction; *EO*, at eye opening; *Post*, 5 min after extubation. *$P < 0.02$ *vs* preinduction value. *Broken line* represents the previously reported arousal threshold from general anesthesia [12,13]

Table 2. Relative band powers (%) and δ ratio [(α + β)/δ] during total spinal block (TSB)[a]

Time	δ	θ	α	β	δ ratio
Pre	42.1 ± 22.4	10.1 ± 10.6	19.6 ± 11.2	28.3 ± 11.4	1.0 ± 0.8
5 min	27.9 ± 12.4	13.8 ± 5.0	37.8 ± 9.9	22.1 ± 20.5	2.2 ± 1.0
15 min	43.4 ± 21.2	13.0 ± 8.5	28.4 ± 25.9	15.2 ± 14.9	1.8 ± 2.6
30 min	45.1 ± 16.5	11.3 ± 2.8	26.6 ± 18.5	12.3 ± 9.9	1.1 ± 1.1
EO	16.7 ± 12.2	14.7 ± 13.4	18.5 ± 14.8	50.2 ± 19.2*	3.2 ± 2.9
Post	36.6 ± 28.8	11.1 ± 13.5	26.5 ± 15.3	25.8 ± 14.0	3.2 ± 4.6
P (ANOVA)	0.071	0.923	0.379	0.003	0.421

[a] Data are expressed as means ± SD. Time points: Pre, before TSB induction; 5 min, 15 min, 30 min, time after TSB induction; EO, at eye opening; Post, 5 min after extubation
* $P < 0.02$ *vs* preinduction value

thetics given intrathecally on the electrical activities of the brain. The frequency of 12.5 Hz in SEF90 observed 15 and 30 min after TSB induction in the present study is comparable to the previously reported threshold value (14 Hz) for predicting arousal from general anesthesia with isoflurane or propofol [10] and isoflurane-nitrous oxide [11]. The 6.4 Hz recorded 30 min after TSB induction is also consistent with the 6 Hz value that was reported as a threshold of awareness during isoflurane-nitrous oxide anesthesia [11,12]. These results, combined with the fact that none of the enrolled patients developed intraoperative memory, suggest that TSB kept the patients under "light" anesthesia in terms of cortical electrical activities. We found no significant change in any of the relative band powers during the unconscious state. These results also suggest the minimal suppressive effect of TSB on cortical electrical activities. Although rapid loss of consciousness and decrease in SEF90 could be achieved by the bolus administration of propofol in this study, the "coma-like" condition lasted over 40 min in all the patients. Therefore, the bolus propofol is not likely to be responsible for the prolonged unconscious state. The discrepancy between the suppressed levels of cortical electrical activities and the practical finding indicating deep coma may be a characteristic unique to TSB-induced unconsciousness.

The mechanisms of TSB-induced unconsciousness are not entirely understood. Based on the observation that no changes occurred in the raw surface EEG during accidental high spinal anesthesia, Huvos et al. [14] suggested that deafferentiation is responsible for unconsciousness. However, TSB induced by cervical injection of local anesthetics has been reported to affect the EEG. Ide et al. [15] found nonresponsive α-like activities accompanied by transient spindle waves. These resembled the "α-like coma" pattern that is typically observed in brain stem injury [16]. They confirmed in monkeys that the distribution of methylene blue in an applied volume ($0.6\,mg \cdot kg^{-1}$) of local anesthetics is limited to the ventral surface of the brain stem, with a thin spread of the dye on the surface of the cortex and the cervical spinal cord. Yanagida [17] reported a high incidence of the appearance of "spike and wave complex" in the EEG under a subconvulsive blood concentration of mepivacaine ($2.7\,\mu g \cdot ml^{-1}$) during TSB. These authors suggested that the direct inhibition of the brain stem reticular formation by local anesthetics caused the clinical "coma-like" condition in patients. In contrast with these findings, the power spectral analysis in this study demonstrated a less suppressive effect of TSB on the cortical electrical activities, without any convulsive activities. These discrepancies may have been caused by the differences in the concentration or the volume of the local anesthetics applied. The initial administration of propofol could alternatively explain the lack of seizure activity [18]. Nonetheless, the characteristic "coma-like" condition accompanied by the "light" changes in the processed EEG parameters achieved by our TSB procedure also suggests the selective suppression of brain stem activities by intrathecal local anesthetics. This hypothesis could be supported by a previous study in cats demonstrating that initial suppression of midbrain electrical activities by intravenous local anesthetics produces relatively minor changes in the cortical EEG, although the suppression was not highly selective in this model [19]. These authors also reported the development of cortical seizure activities in the context of the progression of the electrical suppression levels in the midbrain.

Recently, direct evidence using neuroimaging that demonstrates the effect of human cerebral cortex activity on pain experience has been accumulating [3–5]. The involvement of central mechanisms in pain perception may explain the limited effects of peripheral nerve blockade in chronic intractable pain. Accordingly, several interventions targeting the CNS have been reported to relieve persistent pain, such as hypnosis [6,7], stereotactic operation [8], and electroconvulsion [9]. The characteristic unconsciousness produced by TSB may be related to such central modulations. The reported convulsive therapies have suggested the "dynamic" central interventions as a key mechanism for pain relief. However, we found no convulsive electrical activities in any of the patients. TSB may have more "static" effects on the brain, such as that produced by hypnotic therapy. Although no definitive conclusions can be drawn from the present results because of the limited number of patients and measurements, TSB theoretically blocks almost all afferent impulses toward the brain, which could never be achieved with the other previously reported interventions. This total isolation of the brain with less suppressed cortical activities may have an impact on the brain and modulate individual pain perception. Since TSB is one of the most invasive interventions, further studies are required to clarify its efficacy and mechanisms of pain relief.

In conclusion, the power spectral analysis revealed that TSB produced levels of consciousness that resembled the previously reported prearousal thresholds from general anesthesia. The total isolation of the brain with less suppressed cortical activities may be characteristic of the TSB-induced unconscious state.

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