

Effects of spinal anesthesia on the electroencephalogram in the elderly

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Abstract : Elderly patients frequently fall asleep during spinal anesthesia without sedatives. We investigated effects of spinal anesthesia on electroencephalogram (EEG) in elderly patients. Elderly patients were randomly assigned. Patients in Group C (n = 8) received an epidural catheter with no anesthetics as control ; patients in Group S (n = 8) received spinal anesthesia. Subsequently, processed EEG data were monitored for 5 minutes. Spinal anesthesia induced significant decreases in 90% spectral edge frequency (SEF90), whereas the control group had no change in SEF90. It was concluded that spinal anesthesia induces decreased SEF90, indicating suppressed cortical activity in early phases of sensory blockade in elderly patients.

Key words : Spinal anesthesia ; electroencephalogram ; cortical activity ; elderly.

INTRODUCTION

Patients undergoing spinal anesthesia become drowsy without sedatives (1). Indeed, spinal anesthesia decreases hypnotic requirements in human and animal (1-5). However, the effects of spinal anesthesia on electroencephalogram (EEG) indicating cortical activity are unclear. In clinical experience, elderly patients tend to fall asleep during spinal anesthesia. Therefore, we hypothesized that elderly patients will show suppressed EEG activity with spinal anesthesia. However, prolonged bed rest in a quiet environment may induce somnolence by itself. To rule out this possibility and test our hypothesis, we conducted the experiment immediately after the spinal injection and measured the standard EEG in elderly patients (≥ 65 yr).

METHODS

This study was approved by the School of Medicine, Keio University institutional ethical committee, and written informed consent was obtained from 16 patients ASA I-II (≥ 65 yr), undergoing transurethral bladder tumor resection. Female subjects were excluded to rule out the pos-

sibility of abnormalities in sleep status secondary to menopause (6). Other exclusion criteria included a history of back pain, neurological or psychiatric disease. No premedication was given. Monitoring included non-invasive blood pressure, electrocardiogram and pulse oximetry. The patients received a preload of acetated Ringer's solution (200~500 ml). The EEG electrodes were placed according to the international 10-20 system and their impedance was kept below 5000 Ω . The EEG was recorded and analyzed using a processed EEG monitor system, version 3.01 (Dräger, Lübeck, Germany), and SEF90 and the raw EEG waveforms were stored and displayed on a Microsoft Windows-compatible computer. In the postoperative off-line data analysis, the raw EEG waveforms were inspected to exclude artifacts due to electromyogram or eye movements. During data collection, patients were instructed to relax with their eyes closed and to avoid unnecessary conversation to affect stable processed EEG (pEEG) data.

Subjects were randomly assigned to one of two groups. Following the collection of baseline pEEG data, subjects were placed in the left lateral position. In Group S (n = 8), spinal anesthesia was performed at the L3-4 interspace with a 25-gauge pencil-point needle using 10 mg of glucose-free bupivacaine (0.5%) and the level of sensory block was evaluated by alcohol swab during 30 min. In Group C (n = 8), an epidural catheter without anesthetics was inserted at the L3-4 interspace.

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Subsequently, subjects were returned to the supine position and data was collected for five minutes. This epidural catheter group was used as a control group.

The analog raw EEG signal was digitized for a period of 2 seconds, constituting one epoch. Data were averaged over 10 consecutive artifact-free epochs collected at the end of 5 minutes over the course of the baseline and post-intervention periods by an anesthesiologist blinded to the goals of this study.

The following quantitative EEG parameters were generated by the pEEG system : SEF90, relative power in the frequency bands of δ (1.5 – 4 Hz), θ (4 – 8 Hz), α (8 – 12 Hz), β (> 12 Hz), and the δ ratio ($\alpha + \beta/\delta$). Statistical analyses were performed using the Wilcoxon matched-pair signed-ranks test or the Mann-Whitney U test, and $P < 0.05$ was considered as significance.

RESULTS

No patients were excluded from the study. There were no differences between two groups in terms of patient demographics (Table 1). In Group S, sensory blockade reached T5, T6 or T7. No subjects suffered from hypotension, bradycardia or oxygen desaturation.

Table 2 shows effect of spinal anesthesia on EEG variables and mean arterial pressure compared with control. There were no significant inter-group differences in baseline SEF90, relative band powers. No significant EEG variable changes were observed between pre-intervention and post-intervention in Group C. In contrast, significant decreases in SEF90

Table 1

Patient demographic data

	Control group (n = 8)	Spinal group (n = 8)
Age (years)	73 ± 3	74 ± 8
Height (cm)	165 ± 2	160 ± 3
Weight (kg)	61 ± 9	59 ± 8

Values are presented as mean ± SD.

There were no statistically significant differences between the two groups.

($P = 0.009$) and relative β -band powers ($P = 0.028$) and significant increases in relative θ -band powers ($P = 0.0168$) were observed in Group S.

As a representative example from Group S subjects, Fig. 1 show the time course of changes in SEF90 after spinal anesthesia and shows a significant decrease in SEF90 after spinal anesthesia.

DISCUSSION

We evaluated the effects of spinal anesthesia on EEG in elderly patients. We found that spinal anesthesia induced decreases in SEF90 and relative β -band power and an increase in relative θ -band power, which is consistent with the changes in EEG characteristic of the onset of somnolence (7, 8).

Previous two studies failed to demonstrate EEG suppression with spinal anesthesia (4, 9). These studies consisted of a too heterogenous population, which we thought to be the factor that affected the results. From our clinical experience, elderly patients are likely to fall asleep during spinal anesthesia. In an animal study, spinal anesthesia blocks ascending somatosensory

Table 2

Baseline and post-intervention EEG variables and MAP in control group and spinal groups

	Control group (n = 8)		Spinal group (n = 8)	
	baseline	post-intervention	baseline	post-intervention
SEF90 (Hz)	19.2 ± 3.1	18.4 ± 2.3	22.5 ± 1.5	16.5 ± 3.0 ^a
β (%)	32.0 ± 13.4	28.3 ± 9.0	40.7 ± 8.2	21.4 ± 10.7 ^b
α (%)	14.8 ± 2.9	15.8 ± 3.5	10.8 ± 2.9	18.7 ± 12.7
θ (%)	9.6 ± 2.6	8.2 ± 3.0	7.9 ± 1.8	11.1 ± 1.1 ^c
δ (%)	43.6 ± 14.3	47.7 ± 7.8	40.6 ± 8.8	48.8 ± 16.9
δ ratio	1.4 ± 0.4	1.5 ± 0.3	1.7 ± 0.2	1.5 ± 0.4
MAP (mmHg)	99 ± 12	100 ± 10	100 ± 9	98 ± 7

Values are presented as mean ± SD.

^a $P = 0.009$ between baseline and post-intervention in spinal group.

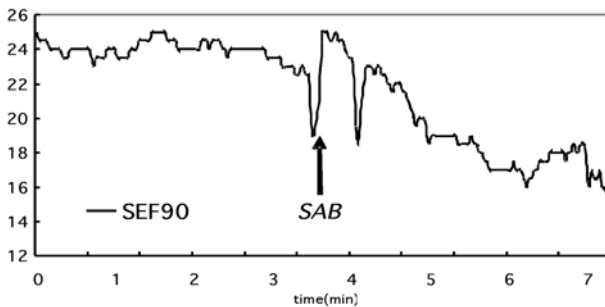
^b $P = 0.028$ between baseline and post-intervention in spinal group.

^c $P = 0.0168$ between baseline and post-intervention in spinal group.

EEG = electroencephalogram.

SEF90 = 90% spectral edge frequency.

MAP = mean arterial pressure.



SAB = Subarachnoid block (spinal anesthesia)

Fig. 1. — Time course of changes in 90% spectral edge frequency (SEF90) during spinal anesthesia block from a single representative subject in spinal group.

transmission to mildly depress the excitability of reticulo-thalamo-cortical arousal mechanisms (10). As sensorimotor disturbances are observed in an aging animal model (11, 12), we speculated that not only sensorimotor disturbance but also the reticulo-thalamic system may be disturbed in elderly patients thus possibly explaining why they are more sensitive to effect of spinal block. Therefore, we decided to restrict our population to elderly patients.

Although a decrease in cerebral blood flow due to reduction in arterial pressure might reduce cortical activity (13), we observed no significant changes, beyond the autoregulation of cerebral blood flow, in arterial pressure throughout the course of our study.

This study has limitations. First, the length of study period (five minutes) may be too short. However, lying on the bed in a quiet environment may induce somnolence, particularly in elderly patients with their eyes closed. This tendency is dependent on the patient's psychological state and may compound or mask effects of spinal anesthesia. To exclude these possibilities and elucidate the effect of spinal anesthesia, we measured EEG variable changes for 5 min immediately after drug administration.

Secondly, sedation was not measured using other method such as the Observer's Assessment of Sedation/Awareness (OAA/S). However, this is not suitable for evaluating the mild sedative effect of spinal anesthesia. One study demonstrated that EEG changes correlate with consciousness (14) allowing us to believe that patients were sedated during suppressed EEG activity.

Recent studies suggest that the involvement of the ascending somatosensory system in spinal anesthesia may induce somnolence (10, 15). A small amount of lidocaine injected into the epidural

space induces sedation, while intravenous lidocaine does not (15), suggesting that the sedation caused by epidural anesthesia was due to the anesthetic block itself rather than by direct cerebral effects. These findings thus indicate that the ascending somatosensory system rather than the direct effects of local anesthetics on the brain may be involved.

In conclusion, we demonstrated that spinal anesthesia induces EEG suppression early in the onset of sensory blockade in elderly patients and changes in EEG are due to inhibitory effects of spinal anesthesia on ascending somatosensory transmission.

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