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Original contribution

Comparison of the effects of propofol and midazolam on the cardiovascular autonomic nervous system during combined spinal and epidural anesthesia☆

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Abstract

Study Objective: To investigate the effect of propofol and midazolam on cardiac autonomic nervous system (CANS) activity during combined spinal-epidural anesthesia.

Design: Prospective, clinical study.

Setting: Operating room of a university hospital.

Patients: Forty ASA physical status I and II patients scheduled for knee surgery.

Intervention: Patients were randomized to receive sedation with either propofol or midazolam.

Measurements: Heart rate (HR), HR variability (HRV), systolic arterial pressure (SAP), and SAP variability (SAPV) were used for the analysis. These values were measured at the preanesthetic period, after intrathecal injection for spinal anesthesia, after sedation with propofol or midazolam, and just before the end of surgery with sedation. Cross-spectral analyses of the HR and SAP data were assessed to quantify the frequency-related coherence spectra and phase spectra.

Main Results: Spinal anesthesia itself had no effect on power spectral changes in both groups. After sedation, as for HRV, high-frequency (HF) power (HF, 0.15-0.40 Hz) did not change, whereas low-frequency (LF) power (LF, 0.04-0.15 Hz) and LF/HF, an indicator of CANS balance, significantly decreased with propofol. Further, coherence in cross-spectra presented depression in the LF band area after sedation with propofol. Before the end of surgery with sedation, LF and LF/HF in both HRV and SAPV were correlated with age in those with propofol; however, scarce relation was observed in those who received midazolam.

Conclusions: Propofol was more potent than midazolam in causing CANS activity to be sympatholytic during combined spinal and epidural anesthesia and which was correlated with age only with propofol.
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1. Introduction

Heart rate (HR) and blood pressure are depressed when the analgesia level approaches cephalad with spinal or epidural anesthesia. This phenomenon is considered to be the result of decreased sympathetic and increased parasympathetic tone in the balance of cardiac autonomic nervous system (CANS) activity [1]. Midazolam and propofol, useful for sedation during regional anesthesia, tend to lead cardiac autonomic balance to become sympatholytic and parasympathotonic by the additive effects of these drugs and anesthesia [2]. Furthermore, as young patients are more apt to show bradyarrhythmia or fall into cardiac arrest, the most serious complication, especially with propofol [3,4], the age factor possibly could affect sympatholytic or parasympathotonic reactions in these situations. Depressed baroreceptor reflex and peripheral sympathetic nervous activity were reported as a cause of propofol [5], although those effects have not been well demonstrated in a range of relatively young ages. Thus, we conducted this study to elucidate 2 hypotheses as to the effects of propofol and midazolam on CANS during combined spinal-epidural anesthesia. First, we studied the difference in the effects of the 2 sedatives on the cardiac nervous system, and then we investigated age-related differences on autonomic nervous output.

To clarify these hypotheses, we assessed HR variability (HRV) and systolic arterial pressure (SAP) variability (SAPV) using time-domain and frequency-domain analyses. Moreover, baroreflex function was assessed using cross-spectral analysis between HR and SAP.

2. Materials and methods

After approval of the study protocol by the Hiroshima University Hospital's institutional ethics committee and written informed consent from all the subjects were obtained, we studied 40 consecutive ASA physical status I and II patients below 50 years of age and undergoing arthroscopic knee surgery. Exclusion criteria were a history of cardiovascular and respiratory disease, as well as any patients receiving hypnotics or sedatives. Patients were randomly assigned via a computer-assisted randomization method to 2 groups: those who received sedation with propofol and those who received midazolam during anesthesia.

Patients were premedicated with 0.5 mg of atropine intramuscularly 60 minutes before anesthesia. After a peripheral intravenous catheter was secured, patients were monitored with electrocardiography, radial arterial pressure, capnography via nostril sampling, pulse oximetry, and HR monitoring. Patients were placed in a surgical-side-down lateral decubitus position, at which time an epidural catheter was placed cephalad at either the L1/L2 or L2/L3 intervertebral space for optional anesthesia in the event of prolonged surgery and postoperative pain relief. Spinal anesthesia was commenced at L3/L4 with an intrathecal injection of hyperbaric 0.5% tetracaine in 2.0 mL of 10% dextrose. Each patient was maintained in this position for 15 minutes so as to achieve sufficient extension of analgesia. Sensory level was determined by pin-prick response. No local anesthetic was given through the epidural space at the time of catheter placement, or within 2 hours after intrathecal injection.

After the dermal level of analgesia was confirmed, either propofol or midazolam was started. Patients in group P received propofol with bolus of 0.5 mg kg^{-1}, followed by a 4 to 6 mg kg^{-1} h^{-1} continuous infusion, whereas those in group M received midazolam with 0.06 to 0.08 mg kg^{-1} bolus, followed by a 3 to 5 mg h^{-1} continuous infusion. When surgery lasted more than 2 hours, a continuous epidural injection of 1.5% mepivacaine was commenced at a rate of 3 to 4 mL h^{-1}. Throughout the operation, patients were kept supine and oxygenated by face mask. Those who received ephedrine for hypotension as SAP decreased below 80 mm Hg and/or atropine for bradycardia below 50 bpm were excluded from the study. Quality of sedation was ascertained continuously with a spectral edge frequency of 90% (SEF90) within a range of 9 to 13 Hz by processed electroencephalogram (Dräger Medical, Lübeck, Germany).

Signals were recorded digitally onto a magneto-optical disk (DR-M3, TEAC, Tokyo, Japan) from a patient monitor (AS/3, Datex, Helsinki, Finland) at a sampling rate of 500 Hz. We also performed an off-line, beat-to-beat analysis for the recorded signals and obtained successive data of HR, BP, and endtidal CO_2 ($ETCO_2$). Mean HR, mean BP (mean arterial pressure), and respiratory frequency were computed from the stored signals, and data were processed for HRV and SAPV, as has been documented previously [1]. The time points of data acquisition were as follows: preanesthetic period (S1), 20 minutes after intrathecal injection when the dermal level of analgesia was confirmed (S2), 30 minutes after commencement of sedation (S3), and just before the end of surgery under sedation (S4). The dermal level of analgesia was confirmed again at S4. All patients were instructed to breathe at ease and remain calm with their eyes closed.

Heart rate variability was assessed using fast Fourier transformation–based power spectral analysis. Sequential R-R interval tachograms were obtained by resampling 1024 instantaneous HR data at 4 Hz for 256 seconds. The power spectral component at the LF (0.04-0.15 Hz) and HF (0.15-0.40 Hz) band widths, and total power (TP, 0.01-0.50 Hz) were computed by integrating the area under the curve and were expressed as a natural logarithm [6]. The ratio of LF to HF (LF/HF) was calculated as an index of sympathovagal balance. Changes in SAPV band frequency analysis were assessed as the same as HRV band frequency. We carried out cross-spectral analyses between HR and SAP data to quantify the frequency-related coherence and phase. This cross-spectral analysis has the same meaning as the correlation coefficient in a linear regression analysis [7] and

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