学位論文　博士（医学）乙

Effects of spinal anesthesia and sedation with dexmedetomidine or propofol on cerebral regional oxygen saturation and systemic oxygenation a period after spinal injection

（脊髄くも膜下麻酔と脊髄くも膜下麻酔中のプロポフォール・デクスメデトミジンを用いた鎮静が脳組織酸素飽和度・経皮的酸素飽和度に与える影響）

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Journal of Anesthesia
https://doi.org/10.1007/s00540-020-02816-5

ORIGINAL ARTICLE

Effects of spinal anesthesia and sedation with dexmedetomidine or propofol on cerebral regional oxygen saturation and systemic oxygenation a period after spinal injection

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Received: 16 January 2020 / Accepted: 12 June 2020

Abstract

Purpose To evaluate changes in cerebral regional oxygen saturation (rSO_2) after spinal anesthesia and compare the changes in rSO_2 and systemic oxygenation between dexmedetomidine sedation and propofol sedation.

Methods Thirty-six patients scheduled to undergo transurethral surgery under spinal anesthesia were randomly assigned to the dexmedetomidine ($n = 18$) and propofol groups ($n = 18$). We used near-infrared spectroscopy sensors to measure rSO_2, and obtained data from each side were averaged. After oxygen insufflation, baseline measurements of mean arterial blood pressure (MAP), heart rate, rSO_2, pulse oximetry saturation (SpO_2), bispectral index, and body temperature were made. After spinal anesthesia, we measured these parameters every 5 min. Twenty minutes after spinal injection, dexmedetomidine or propofol administration was started. We measured each parameter at 10, 25, and 40 min after the administration of dexmedetomidine or propofol.

Results The baseline rSO_2 in the dexmedetomidine group was $71.3 \pm 7.3\%$, and that in the propofol group was $71.8 \pm 5.6\%$. After spinal anesthesia, rSO_2 in both groups decreased significantly (dexmedetomidine group: $65.4 \pm 6.9\%$; propofol group: $64.3 \pm 7.4\%$). After administering sedatives, rSO_2 was equivalent after spinal anesthesia. rSO_2 was comparable between the two groups. MAP and SpO_2 were significantly higher in the dexmedetomidine group than in the propofol group.

Conclusion Spinal anesthesia decreased rSO_2; however, the decline was not severe. Dexmedetomidine and propofol did not compromise cerebral oxygenation under spinal anesthesia. Nevertheless, MAP and SpO_2 were more stable in dexmedetomidine sedation than in propofol sedation. Dexmedetomidine may be suitable for spinal anesthesia.

Keywords Dexmedetomidine · Near-infrared spectroscopy · Propofol · Spinal anesthesia

Introduction

Spinal anesthesia is commonly used for surgeries below the umbilicus, but it sometimes causes hypotension. It was found that cerebral blood flow (CBF) decreases after spinal anesthesia [1]. Neuraxial anesthesia, including spinal anesthesia, has been reported to possess a sedative property [2, 3]. We reported in a previous study that epidural anesthesia decreased the bispectral index [2]. Sedation decreases the cerebral metabolic rate (CMR) of oxygen. The CMR changes parallel with the change in CBF. However, because of the concurrent effects of decreased blood pressure and sedation, the CBF can decrease beyond CMR-CBF coupling after spinal anesthesia, resulting in compromised cerebral oxygenation.

Sedation is sometimes required in spinal anesthesia, and dexmedetomidine and propofol are the most commonly used drugs for this purpose. Dexmedetomidine has cerebrovascular constricting property [4], whereas propofol has no effect on the cerebral blood vessels [5]. In addition, dexmedetomidine was found to decrease the CBF more than the CMR [6]. Conversely, propofol decreased the CBF less than the CMR [6]. In spinal anesthesia, CBF may decrease to a greater extent under sedation with dexmedetomidine than under sedation with propofol. Thus, cerebral oxygenation may be

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Published online: 18 June 2020

more impaired under dexmedetomidine sedation than under propofol sedation.

Regarding the sedation with dexmedetomidine or propofol in the intensive care unit, the arterial blood pressure was comparable between the two groups, whereas the heart rate (HR) was significantly lower in the dexmedetomidine group [7]. On the other hand, propofol, but not dexmedetomidine, yielded increased airway resistance and bronchoconstriction index [8]. Hence, systemic oxygenation may be better preserved in dexmedetomidine sedation.

Near-infrared spectroscopy is used to measure regional cerebral tissue oxygen saturation (rSO_2). It has been reported that the rSO_2 can be used to monitor cerebral ischemia [9] and that it reflects the cerebral perfusion [10, 11]. Moreover, rSO_2 measurement appears to be superior to transcranial Doppler for monitoring the adequacy of cerebral perfusion and oxygenation [12]. Therefore, the rSO_2 should reflect the CBF.

To the best of our knowledge, no previously published study has compared the value of rSO_2 in propofol sedation and the dexmedetomidine sedation under spinal anesthesia. In the present study, we tested the following hypotheses. First, cerebral oxygenation is compromised under spinal anesthesia. Second, under spinal anesthesia, dexmedetomidine impairs the cerebral circulation more than does propofol. Third, systemic oxygenation is preserved better in dexmedetomidine than in propofol sedation.

Materials and methods

The study was approved by the institutional review board of the University of Yamanashi (study No. 1252) and was registered in the University Hospital Medical Information Network Clinical Trials Registry under study number UMIN000026225. Written informed consent was obtained from all patients.

We enrolled 46 patients who were scheduled to undergo elective transurethral resection of bladder tumor under spinal anesthesia combined with sedation using dexmedetomidine or propofol at the University of Yamanashi Hospital between January 2015 and February 2018. All patients had an American Society of Anesthesiologists ASA physical status class of I–III. Patients with cerebral diseases were excluded. Using a computer-generated randomization list, patients were randomly assigned to one of two groups based on the sedative agents; dexmedetomidine or propofol group.

Patients did not receive premedication. Before anesthesia, the sensors for near-infrared spectroscopy (SAFB-SM, Covidien, Dublin, Ireland) were pasted on the bilateral forehead for rSO_2 measurement. The obtained data from each side were averaged. A bispectral index (BIS) sensor was also pasted on the forehead. Monitoring included electrocardiography, non-invasive automated blood pressure measurement, pulse oximetry (SpO_2), and body temperature measurement using an earphone-type infrared tympanic thermometer (CE Thermo, Nipro, Tokyo, Japan). Oxygen was given at 2 L/min through a nasal cannula. After oxygen insufflation, baseline measurements of mean arterial blood pressure (MAP), HR, rSO_2, SpO_2, BIS, and body temperature were made. Next, spinal anesthesia was performed using 2.3–2.5 mL hyperbaric 0.5% bupivacaine. Measurements were taken every 5 min until 20 min after spinal anesthesia, at which time intravenous dexmedetomidine or continuous propofol administration was started. Dexmedetomidine was given at 4 μg/kg/h for 10 min, and then the dose was decreased to 0.4 μg/kg/h. Propofol was administered using a target-controlled infusion at 2 μg/mL. The sedation depth was titrated to a BIS of 50–70. MAP, HR, rSO_2, SpO_2, BIS, and body temperature measurements were made at 10, 25, and 40 min after the start of dexmedetomidine or propofol administration.

Fluid management was conducted by the attending anesthesiologist. To maintain MAP and HR, 5 mg ephedrine or 0.05 mg phenylephrine was used when systolic blood pressure decreased by 30% compared with baseline or < 90 mmHg. Airway patency was maintained by preventing upper airway obstruction using jaw thrust maneuver if needed. A clinically unsafe decline of rSO_2 was defined as over 20% decrease from baseline or below 50%. If the patient had a clinically unsafe decline of rSO_2, the study was stopped.

The primary outcome was the change in rSO_2 after spinal anesthesia and during dexmedetomidine or propofol sedation under spinal anesthesia. The secondary outcome was the change in SpO_2 during dexmedetomidine or propofol sedation under spinal anesthesia. We also evaluated the changes in MAP and HR during dexmedetomidine and propofol sedation under spinal anesthesia.

We used Stat Flex version 6.0 (Artec, Osaka, Japan) for statistical analysis. Power analysis revealed that a sample size of 18 patients per group was sufficient to provide 80% power with an α level of 0.05 to detect mean differences of 7% in rSO_2. The spread of spinal anesthesia was compared using the Mann-Whitney test. Values are presented as medians and quartiles. Within-group comparisons of changes in MAP, HR, rSO_2, SpO_2, and BIS were examined via a two-way analysis of variance (ANOVA) and Dunnett post hoc comparisons. Between-group differences in MAP, HR, rSO_2, SpO_2, and BIS were performed using a two-way ANOVA and Newman–Keuls post hoc test. Values are presented as means ± standard deviations. A P-value < 0.05 was considered statistically significant.

Results

All the participants had an ASA physical status class I or II. No participant had severe cardiovascular or respiratory disease. Thirteen (6 in the propofol group, 7 in the dexmedetomidine group) patients had well-controlled hypertension, and 7 (6 in the propofol group, 1 in the dexmedetomidine group) had mild chronic obstructive pulmonary disease. Of 46 eligible patients, 10 were excluded because of data acquisition error. Therefore, 36 patients (dexmedetomidine group: $n = 18$; propofol group: $n = 18$) were included in the final analysis (Fig. 1). There were no significant differences between the two groups as regards demographic characteristics, except for sex (Table 1). Body temperature was maintained at approximately 36.5 °C during the study period. Median levels of sensory block at 15 min after spinal anesthesia were T8.67 (interquartile range, T6.00–T9.00) in the dexmedetomidine group and T8.88 (interquartile range, T6.00–T10.00) in the propofol group; there was no significant difference between the two groups ($P = 0.523$). No patient experienced cardiorespiratory collapse. Two patients needed jaw thrust maneuver due to upper airway obstruction; still, SpO_2 did not drop below 90%.

Figure 2a shows that the MAP decreased at 20 min after spinal anesthesia. In the dexmedetomidine group, the MAP significantly increased at 10 min after dexmedetomidine administration ($P < 0.05$, compared with 20 min after spinal anesthesia), but decreased after that. In the propofol group, the MAP decreased after propofol administration. After dexmedetomidine or propofol administration, the MAP was higher in the dexmedetomidine group than in the propofol group. Figure 2b shows that HR did not change after spinal anesthesia. In both groups, the HR decreased significantly at 10 min after sedative administration (dexmedetomidine: $P < 0.01$; propofol: $P < 0.05$), and there was no significant difference between the two groups.

Table 1 Patients' characteristics

	Dexmedetomidine group ($n = 18$)	Propofol group ($n = 18$)	P value
Age	71 ± 11	74 ± 10	0.3569
Sex (male/female)	17/1	12/6	0.0352
Height	163.3 ± 7.5	161.4 ± 7.4	0.4381
Weight	60.4 ± 9.9	61.0 ± 8.0	0.8434

Figure 3a shows that SpO_2 did not change significantly in the dexmedetomidine group. Conversely, SpO_2 decreased after propofol administration. Figure 3b shows that the BIS did not change after spinal anesthesia. After sedative administration, the BIS decreased significantly in both groups (dexmedetomidine: $P < 0.05$; propofol: $P < 0.01$), with no significant difference between the groups.

Figure 4 shows that the rSO_2 increased after oxygen insufflation relative to the baseline values. The baseline mean rSO_2 was 71.3 ± 7.3% in the dexmedetomidine group and 71.8 ± 5.6% in the propofol group. At 20 min after spinal anesthesia, the rSO_2 significantly decreased in both groups to 65.4 ± 6.9% ($P < 0.01$) and 64.3 ± 7.4% ($P < 0.01$), respectively. After dexmedetomidine loading (10 min after the start of dexmedetomidine), the rSO_2 slightly decreased (63.8 ± 7.0%), but the decline in rSO_2 was not statistically significant. Under propofol sedation, the rSO_2 was equivalent to that after spinal anesthesia. The rSO_2 values were comparable between the two groups. No patient developed a clinically unsafe decline in rSO_2.

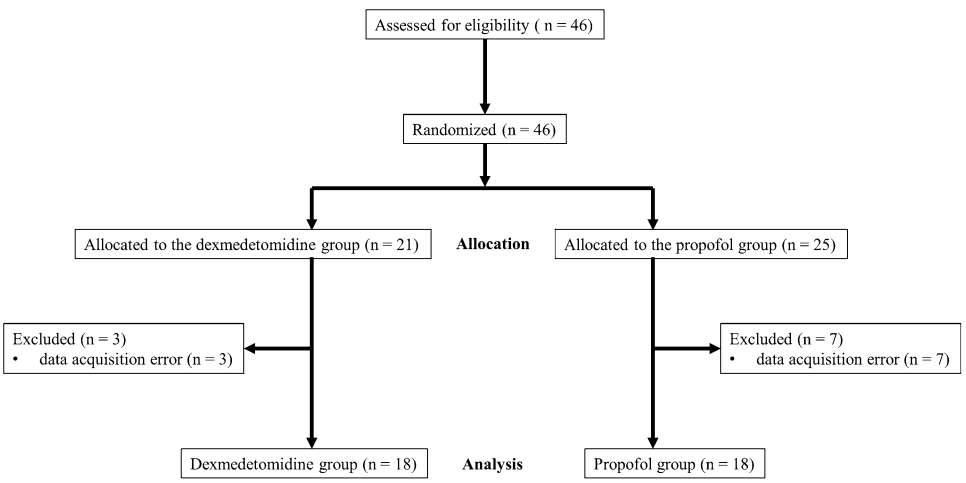

Fig. 1 Flow chart of patient allocation and analysis

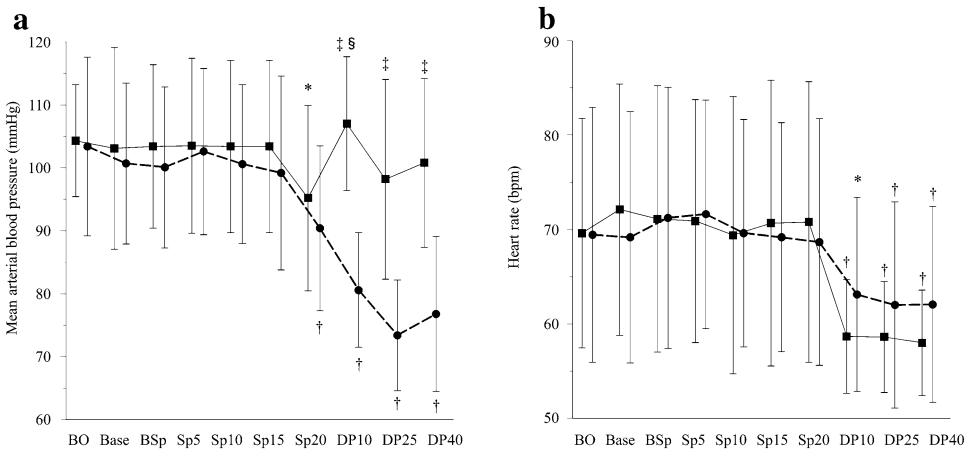

Fig. 2 Change of mean arterial blood pressure (MAP) (**a**) and heart rate (HR) (**b**) in the dexmedetomidine group (filled black square) and the propofol group (filled black circle). 20 min after the spinal anesthesia, MAP decreased in the both groups. After the dexmedetomidine application, MAP increased temporarily. After the propofol application, MAP decreased significantly. Heart rate did not change after the spinal anesthesia. After the dexmedetomidine or propofol application, HR decreased significantly. * $P < 0.05$, compared with baseline. † $P < 0.01$, compared with baseline. ‡ $P < 0.01$, corresponding propofol group. § $P < 0.05$, compared with Sp20. BO: before oxygen insufflation, Base: baseline, BSp: just before spinal anesthesia, Sp5: 5 min after the spinal anesthesia, Sp10: 10 min after the spinal anesthesia, Sp15: 15 min after the spinal anesthesia, Sp20: 20 min after the spinal anesthesia, DP10: 10 min after the dexmedetomidine or propofol application, DP25: 25 min after the dexmedetomidine or propofol application, DP 40: 40 min after the dexmedetomidine or propofol application

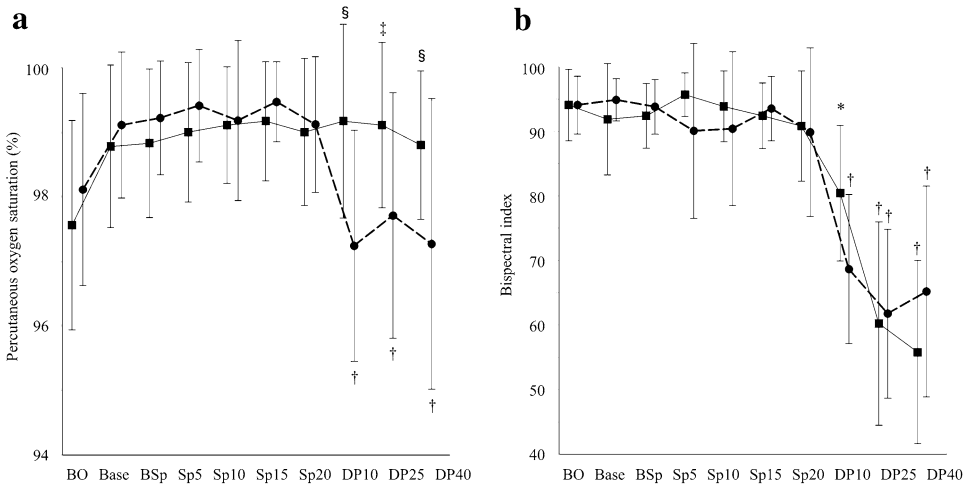

Fig. 3 Change of percutaneous oxygen saturation (SpO_2) (**a**) and bispectral index (BIS) (**b**) in the dexmedetomidine group (filled black square) and the propofol group (filled black circle). In the dexmedetomidine group, SpO_2 did not change significantly. In the propofol group, SpO_2 decreased after the sedation with propofol. In the both groups, BIS decreased after the sedation with either dexmedetomidine of propofol. * $P < 0.05$, compared with baseline. † $P < 0.01$, compared with baseline. ‡ $P < 0.05$, corresponding propofol group. § $P < 0.01$, corresponding propofol group. BO: before oxygen insufflation, Base: baseline, BSp: just before spinal anesthesia, Sp5: 5 min after the spinal anesthesia, Sp10: 10 min after the spinal anesthesia, Sp15: 15 min after the spinal anesthesia, Sp20: 20 min after the spinal anesthesia, DP10: 10 min after the dexmedetomidine or propofol application, DP25: 25 min after the dexmedetomidine or propofol application, DP 40: 40 min after the dexmedetomidine or propofol application

Discussion

This is the first study to compare the CBF in dexmedetomidine sedation and propofol sedation under spinal anesthesia by using near-infrared spectroscopy. In the present study, we found that the rSO_2 decreased after spinal anesthesia. However, no further reduction in the rSO_2 was noticed during sedation with either dexmedetomidine or propofol. In addition, those declines in rSO_2 were not

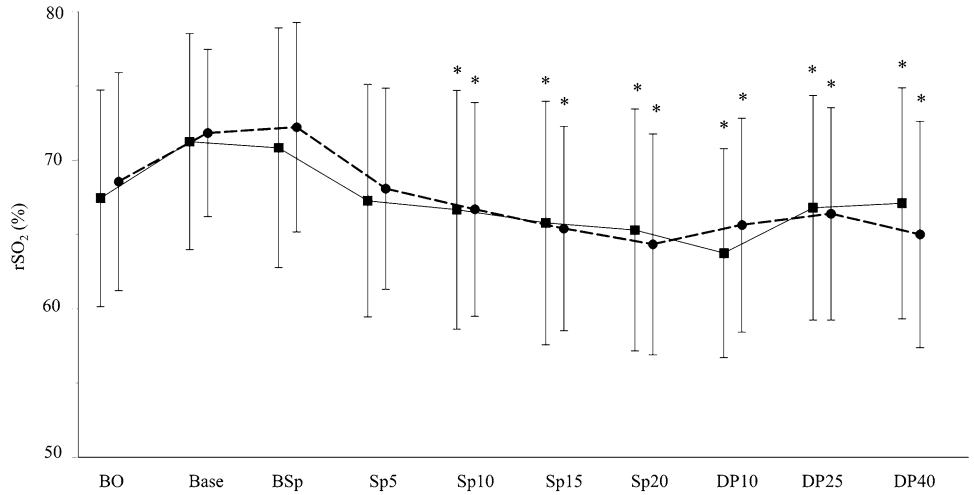

Fig. 4 Change of rSO_2 in the dexmedetomidine group (filled black square) and the propofol group (filled black circle). rSO_2 decreased from 10 min after the spinal anesthesia. rSO_2 remained low values, however, no further decline was observed. * $P < 0.01$, compared with baseline. BO: before oxygen insufflation, Base: baseline, BSp: just before spinal anesthesia, Sp5: 5 min after the spinal anesthesia, Sp10: 10 min after the spinal anesthesia, Sp15: 15 min after the spinal anesthesia, Sp20: 20 min after the spinal anesthesia, DP10: 10 min after the dexmedetomidine or propofol application, DP25: 25 min after the dexmedetomidine or propofol application, DP 40: 40 min after the dexmedetomidine or propofol application

clinically unsafe. We also found that the MAP and SpO_2 were more stable in the dexmedetomidine group than in the propofol group. These results suggest that compared to propofol, dexmedetomidine has the advantage of improved systemic circulation, including the cerebral region, under spinal anesthesia.

This study shows that the rSO_2 decreased after spinal anesthesia. The changes in rSO_2 under spinal anesthesia are not well known. One study showed that the rSO_2 did not change significantly in patients who underwent spinal anesthesia for urological surgery [13]. Others reported a significant decline in rSO2 after spinal anesthesia for cesarean delivery [14]. The subjects of the present study were elderly patients. In geriatric patients (> 60 year.) who underwent surgical fixation of the neck of the femur, the frequency of rSO_2 reduction below the baseline level was significantly higher with spinal than with general anesthesia [15]. In elderly patients, the cardiac output decreased after spinal anesthesia [16]. Furthermore, among patients who underwent spinal anesthesia for open surgical repair of a hip fracture, the systolic blood pressure and CBF were significantly lower in those > 75 years of age than in those aged < 60 years [1]. The decline in rSO_2 in this study may be due to a decrease in cardiac output and CBF caused by spinal anesthesia though we did not directly measure cardiac output and CBF. Another possible explanation for this decline in rSO2 may be the redistribution of blood flow after spinal anesthesia. In general anesthesia, redistribution of heat occurs from the central core to the periphery due to anesthetic-induced vasodilation [17]. It has been reported that sympathetic nervous system activity is well maintained compared to parasympathetic activity in geriatric patients [18]; therefore, peripheral blood vessels are potentially constricted in elderly patients. The peripheral sympathetic block caused by spinal anesthesia may induce extensive peripheral vessel dilation. The blood flow redistribution from the cranial to the peripheral vessels in spinal anesthesia may be the cause of decline in rSO_2 after spinal anesthesia.

Dexmedetomidine has been found to exert a cerebrovascular constricting property, [4] and dexmedetomidine-induced sedation has been shown to decrease the CBF [19]. Dexmedetomidine was reported to decrease the CBF in the face of an unaltered CMR in isoflurane-anesthetized dogs [20]. Thus, cerebral circulation may be compromised under dexmedetomidine sedation. In contrast, propofol has no direct effect on cerebral arteries [5], yet it is known to cause a decrease in the CBF accompanied by a concurrent decrease in the CMR [21]. Cerebral circulation would be well preserved under propofol anesthesia. We speculated that the rSO_2 should decrease more in dexmedetomidine than in propofol sedation. However, the rSO_2 was comparable between the two groups in the present study. Drummond et al. [22] reported that dexmedetomidine decreased the middle cerebral artery velocity in parallel with a reduction in the CMR in healthy humans. The result suggested that CMR-CBF coupling is preserved during dexmedetomidine sedation in humans. Moreover, the CMR reduction was less under propofol sedation than under dexmedetomidine sedation in humans [23]. Cerebral circulation may not be impaired in dexmedetomidine

sedation in humans. In addition, in the present study, the MAP and SpO_2 were higher in dexmedetomidine sedation than in propofol sedation. We recently reported that the rSO_2 changed along with the MAP [24]. The rise in MAP may be partly responsible for the comparable rSO_2 changes between dexmedetomidine and propofol sedation despite the cerebrovascular constricting property of dexmedetomidine. Regarding the correlation between SpO_2 and rSO_2, there have been contradictory reports. One study suggested that SpO_2 had a significant effect on cerebral oxygenation [25]. Another study found no significant correlation between cerebral and arterial oxygenation [26]. We also found previously that arterial oxygenation did not affect rSO_2 [24]. Thus, SpO_2 would not have an effect on rSO_2 results.

Regarding the effects of dexmedetomidine and propofol on hemodynamics, Chang et al. [27] reported that the blood pressure was lower in dexmedetomidine sedation without a loading dose than in propofol sedation in critically ill patients. Contrary to their findings, Wu et al. [28] reported that the MAP was higher in dexmedetomidine sedation with a loading dose than in propofol sedation during esophagogastroduodenoscopy. Our results concur with those of the latter study as the MAP was higher in dexmedetomidine sedation than in propofol sedation. The hypotensive effect of dexmedetomidine is mediated by alpha-2 adrenoceptor activation in the central nervous system [29], and its hypertensive effect is mediated by activation of the vascular alpha-2B adrenoceptors [30]. The effect of dexmedetomidine on blood pressure is biphasic [31]. Dexmedetomidine decreased the blood pressure in lower plasma concentrations, while it increased the blood pressure in higher plasma concentrations [31]. In Chang's study, a loading dose of dexmedetomidine was omitted [27], whereas a loading dose was employed in Wu's [28] and our study. Therefore, the plasma concentration of dexmedetomidine was lower in Chang's study than in Wu's and our study. The higher plasma concentration of dexmedetomidine could be responsible for the higher MAP in dexmedetomidine sedation. On the other hand, propofol reduces sympathetic nervous activity [32]. Spinal anesthesia also reduces sympathetic nervous activity depending on the area of anesthesia. In this study, the median sensory blockade level in the propofol group was T8.88. Generally, the sympathetic block level was higher than the sensory block level. Hence, the decrease in blood pressure in the propofol group was caused by the combined sympathetic blockade effect of propofol sedation and spinal anesthesia. Chang et al. [27] also reported that HR was lower in dexmedetomidine sedation than in propofol sedation. In agreement with their study, the HR in this study was lower in dexmedetomidine sedation than in propofol sedation, although the difference was not significant. However, there was no need to treat for bradycardia in dexmedetomidine sedation. For sedation in spinal anesthesia, dexmedetomidine may be preferred because the blood pressure is more stably maintained.

In this study, SpO_2 was significantly higher in dexmedetomidine than in propofol sedation. In some studies, respiratory depression did not develop in dexmedetomidine sedation [28, 31, 33]. In contrast, propofol produced dose-related collapsibility of the upper airway and respiratory depression [34]. The difference in SpO_2 between dexmedetomidine and propofol sedation may be due to the disparity in the effects on respiration between both drugs. Our result corresponds with Yoon's finding that SpO_2 was significantly lower in response to propofol than to dexmedetomidine [33]. The depth of sedation may affect respiration; however, the BIS was similar in both groups. Thus, the depth of sedation was comparable between dexmedetomidine sedation and propofol sedation in this study. Regarding the effects on respiration, dexmedetomidine may be preferable for sedation under spinal anesthesia.

The rSO_2 is influenced by cardiac output that correlates with the mean arterial pressure and heart rate [35]. We observed in the present study that propofol induced stronger suppression of the circulation than dexmedetomidine. However, values of rSO_2 were comparable between dexmedetomidine- and propofol-induced sedation. Propofol causes respiratory depression not only by airway obstruction [36] but also by central respiratory suppression [37]. In this study, propofol suppressed respiration even though the value of BIS was between 50 and 70 with TCI 2 μg/mL. We implemented airway management with jaw thrust when the patient experienced airway obstruction. However, the respiratory suppression due to propofol sedation might have induced hypercarbia. We have previously shown that rSO_2 was associated with $PaCO_2$ [38]. Hypercarbia induced by propofol could have increased the rSO_2 values. It is possible that circulatory suppression-induced a lowering effect on rSO_2 and that the hypercarbia-induced increase of rSO_2 balanced this effect. Consequently, the values of rSO_2 were very similar for both dexmedetomidine sedation and propofol sedation.

It has been accepted that BIS decreases with the sedation level. Mean BIS at deep sedation was reported to be 49 ± 16 [39]. Others reported that median BIS values for moderate to deep sedation were between 50 and 70 [40]. We controlled the dose of sedatives, as the value of BIS would be ranging from 50 to 70 in this study. Target BIS values in this study might be adequate. Nevertheless, one study reported that the BIS values at moderate sedation and deep sedation during dexmedetomidine sedation were 65.6 ± 7.1 and 43.8 ± 5.3, and those during propofol sedation were 73.6 ± 3.7 and 53.6 ± 7.6, respectively [41]. The BIS values at moderate and deep sedation by dexmedetomidine sedation were significantly lower than those by propofol sedation [41]. It might have been better to determine more

detailed target BIS settings for dexmedetomidine sedation and propofol sedation.

This study has some limitations. First, the observational period was short because the duration of transurethral resection of the bladder tumor was short. Spinal anesthetic block height lowers with procedure duration; differences in spinal block level might affect the rSO_2. Nevertheless, most operations that are performed under spinal anesthesia should be short. Second, we did not measure arterial carbon dioxide pressure. Arterial catheter insertion is not common, and it is too invasive for the transurethral resection of bladder tumors. We reported previously that the arterial carbon dioxide pressure affected the rSO_2 [24]. The effects on respiration are different between dexmedetomidine sedation and propofol sedation. Therefore the arterial carbon dioxide pressure may have been higher under propofol sedation than under dexmedetomidine sedation in this study. Furthermore, muscle contraction on the forehead or face influences the value of BIS [42]. In this study, because the patients were under sedation, muscle contraction on the forehead or face might have occurred during anesthesia.

Conclusions

Spinal anesthesia decreased the rSO_2, but the decline was not clinically unsafe. Sedation with dexmedetomidine and propofol under spinal anesthesia does not compromise cerebral circulation. However, the MAP and SpO_2 were more stable in dexmedetomidine sedation than in propofol sedation. Thus, dexmedetomidine may be more suitable for short-term sedation in spinal anesthesia.

Acknowledgments We would like to thank Editage (http://www.editage.com) for English language editing.

Compliance with ethical standards

Conflict of interest statement All authors have no conflict of interest.

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