



# REVISTA BRASILEIRA DE ANESTESIOLOGIA

Official Publication of the Brazilian Society of Anesthesiology  
[www.sba.com.br](http://www.sba.com.br)



## SPECIAL ARTICLE

# Dexmedetomidine and propofol infusion on sedation characteristics in patients undergoing sciatic nerve block in combination with femoral nerve block via anterior approach

Abdulkadir Yektaş\*, Funda Gümüş, Ayşin Alagol

Anesthesiology and Reanimation Clinic, Bağcılar Training and Research Hospital, Istanbul, Turkey

Received 28 November 2013; accepted 2 January 2014

Available online 5 February 2014

### KEYWORDS

Dexmedetomidine;  
Propofol;  
Sedation;  
Nerve block

### Abstract

**Objective:** Dexmedetomidine is an  $\alpha$ -2 adrenergic agonist having wide range of effects including sedation in mammalian brain, and has analgesic as well as sympatholytic properties. This study aimed to compare the effects of dexmedetomidine and propofol infusion on sedation characteristics in patients undergoing combined sciatic nerve and femoral nerve block via anterior approach for lower limb orthopedic procedure.

**Methods:** Forty patients, who were between 18 and 65 years old, this study was made at anesthesiology clinic of Bağcılar training and research hospital in 08 September 2011 to 07 June 2012, and underwent surgical procedure due to fractures lateral and medial malleol, were included. Sciatic nerve and femoral nerve block were conducted with an anterior approach on all patients included in the study, with an ultrasonography. The patients were randomly divided into dexmedetomidine [Group D ( $n = 20$ );  $0.5 \mu\text{g kg}^{-1} \text{h}^{-1}$ ] and propofol [Group P ( $n = 20$ );  $3 \text{mg kg}^{-1} \text{h}^{-1}$ ] infusion groups.

**Results:** The vital findings and intra-operative Ramsay sedation scale values were similar in both groups. Time taken for sedation to start and time required for sedation to become over of Group D were significantly higher than those of Group P ( $p < 0.001$  for each).

**Conclusions:** Substitution of dexmedetomidine instead of propofol prolongs the times to start of sedation, the times to end of sedation and duration of sedation.

© 2014 Sociedade Brasileira de Anestesiologia. Published by Elsevier Editora Ltda. All rights reserved.

\* Corresponding author.

E-mails: [akyektas722000@yahoo.co.uk](mailto:akyektas722000@yahoo.co.uk), [akyektas@hotmail.com](mailto:akyektas@hotmail.com) (A. Yektaş).

**PALAVRAS-CHAVE**

Dexmedetomidina;  
Propofol;  
Sedação;  
Bloqueio de nervo

## Características da sedação com infusão de dexmedetomidina e propofol em pacientes submetidos ao bloqueio do nervo ciático em combinação com bloqueio do nervo femoral via abordagem anterior

**Resumo**

**Objetivo:** Dexmedetomidina é um agonista  $\alpha_2$ -adrenérgico que tem uma ampla gama de efeitos, incluindo sedação do cérebro de mamíferos, e propriedades tanto analgésicas quanto simpato-líticas. Este estudo teve como objetivo comparar os efeitos de dexmedetomidina e propofol sobre as características da sedação em pacientes submetidos ao bloqueio combinado dos nervos ciático e femoral via abordagem anterior em procedimento ortopédico de membro inferior.

**Métodos:** Quarenta pacientes, entre 18 e 65 anos, submetidos a procedimento cirúrgico por causa de fraturas lateral e medial do maléolo, foram incluídos neste estudo, conduzido no Departamento de Anestesiologia do, Bağcılar Training and Research Hospital de 8 de setembro de 2011 a 7 de junho de 2012. O bloqueio dos nervos ciático e femoral foi feito via abordagem anterior em todos os pacientes incluídos no estudo, com ultrassonografia. Os pacientes foram randomicamente divididos em dois grupos para as infusões de: dexmedetomidina (grupo D [n = 20];  $0,5 \mu\text{g kg}^{-1} \text{h}^{-1}$ ) e propofol (grupo P [n = 20];  $35 \text{mg kg}^{-1} \text{h}^{-1}$ ).

**Resultados:** Os sinais vitais e os valores da escala de sedação de Ramsay no período intraoperatório foram semelhantes em ambos os grupos. Os tempos de início e término da sedação no grupo D foram significativamente maiores do que os no grupo P ( $p < 0,001$ , respectivamente).

**Conclusão:** O uso de dexmedetomidina em vez de propofol prolonga os tempos de início, término e duração da sedação.

© 2014 Sociedade Brasileira de Anestesiologia. Publicado por Elsevier Editora Ltda. Todos os direitos reservados.

**Introduction**

Anxiety may lead to hypertension, arrhythmia and increase in myocardial oxygen consumption by causing higher sympathetic stimulation in patients undergoing surgical procedure under local or general anesthesia. Generally, sedation and non-surgical pain management during a surgery in patients undergoing surgical procedure under regional anesthesia has become an important issue in the anesthesiology practice.<sup>1</sup> Primary aim of the sedation includes providing comfort to patients, eliminating anxiety, maintenance of hemodynamic stability and restraining patient from moving. Dexmedetomidine is an  $\alpha_2$  adrenergic agonist which is more selective than clonidine and has wide range of effects including sedation in mammalian brain without causing anesthesia as well as has analgesic and sympatholytic properties.<sup>2</sup> The most important advantage of dexmedetomidine is lack of properties that may cause respiratory depression, although it may cause deep sedation at therapeutic doses.<sup>3</sup> Owing to such novel properties, dexmedetomidine may be a safer drug to provide sedation in patients undergoing peripheral nerve block. It has been used for sedation in various anesthesia procedure like MRI, spinal anesthesia involving wide range of patients including infants and children.<sup>4</sup> High dose of dexmedetomidine has been successfully used in pediatric magnetic resonance imaging (MRI) sleep studies.<sup>5</sup> Additionally, in a case report it was reported that dexmedetomidine was successfully and safely used for sedation during spinal anesthesia of a very old patient.<sup>6</sup> Dexmedetomidine has also been used effectively for the sedation of infants and children during spinal anesthesia in combination with ketamine preserving cardiovascular and respiratory functions.<sup>7</sup> Favorable results have been obtained by dexmedetomidine sedation

during septoplasty surgery under local anesthesia in terms of satisfaction from anesthesia and surgery.<sup>8</sup> However, it has not been used for sedation in various other regional anesthesia procedures such as lower limb nerve block.

Propofol is being safely and successfully used for a long time during any intervention and imaging technique requiring sedation in patients with spontaneous respiration, as well as in regional anesthesia and peripheral nerve blocks. Various studies have shown that propofol is a preferable agent for sedation when used in combination with opioids since its efficacy starts and ends easily and dose titration is easily performed.<sup>9,10</sup> Bilateral brachial plexus block has been successfully performed with propofol-ketamine sedation under ultrasonography guidance.<sup>11</sup>

Sciatic nerve block via anterior approach can be performed under ultrasonography guidance and such an approach is very comfortable for the patient; femoral nerve block as well can be performed at the same time in the same region.<sup>12,13</sup>

The present study was aimed to compare the effects of intravenous continuous infusion of dexmedetomidine and propofol on the sedation characteristics of patients undergoing sciatic nerve block and femoral nerve blocks through anterior approach.

**Materials and methods**

After the study was approved by the local ethics committee of Yeditepe University Medical Faculty (approval date and number: 02.08.2011; 130), this study was made at anesthesiology clinic of Bağcılar training and research hospital, İstanbul, Turkey in 08 September 2011 to 07 June 2012,

40 patients, who were between 18 and 65 years old, in the American Society of Anesthesiologists (ASA) classification I–II,<sup>14</sup> and had undergone surgical procedure due to lateral and medial malleol fractures, were included in the study after the approval of local ethics committee and written informed patient consent were obtained. This study has been prepared in accordance with the principles of the Helsinki declaration.

Age, gender, height and weight of the patients, as well as concomitant diseases, history for drug use and smoking, anesthesia technique, surgery duration, tourniquet duration, ASA classification, systolic and diastolic blood pressures, peripheral oxygen saturation, heart rate, onset of sedation, level of sedation, time of termination of sedation, were recorded.

Inclusion criteria:

1. Patients of between 18 and 65 years old.
2. Patients with ASA I–II.
3. Patients with lateral and medial malleol fractures.

Exclusion criteria:

1. Patients with vascular disease, cardiac disease [I–II degree atrioventricular (AV) block].
2. Metabolic-renal-hepatic disease.
3. Pregnancy.
4. Hemodynamic instability.
5. Drug use that is likely to cause metabolic acid–base imbalance.
6. History for steroid use and allergy.
7. Contraindications to regional anesthesia.
8. Alcohol – drug addiction.
9. Those who did not graduate from primary school were excluded from the study.

The Ramsay sedation scale<sup>15</sup> (RSS) was used to assess the level of sedation. The modified Aldrete scoring system<sup>16</sup> was used to assess recovery from anesthesia. The patients having no premed were divided into two groups [Group dexmedetomidine (D;  $n=20$ ) and Group propofol (P;  $n=20$ )] on the day of surgery according to the computerized randomization scheme.<sup>17</sup> Electrocardiography, non-invasive arterial blood pressure measurement and peripheral pulse oximetry were performed after the patients were admitted into the surgery room and basal values of their vital signs were recorded. For sedation, Group D received dexmedetomidine (Precedex, Abbott, Rock Mount, NC, USA) infusion at a dose of  $1\ \mu\text{g kg}^{-1}$  for the first 10 min and  $0.5\ \mu\text{g kg}^{-1}\ \text{h}^{-1}$  throughout the surgery. Patients in Group P received 2% propofol (Propofol-<sup>®</sup>Lipuro, B. Braun, Melsungen AG, Germany) at a dose of  $1\ \text{mg kg}^{-1}$  as the loading dose (in a minute) prior to the block and then infusion was started at a rate of  $3\ \text{mg kg}^{-1}\ \text{h}^{-1}$  and continued at the end of surgery. Both groups simultaneously received 0.9% NaCl at a rate of  $10\ \text{mL kg}^{-1}\ \text{h}^{-1}$  for the first hour and continued at a rate of  $5\ \text{mL kg}^{-1}\ \text{h}^{-1}$ . Vital signs were measured at 5 min intervals after dexmedetomidine or propofol infusion was started, and the time to start sedation (RSS score between 2 and 4) was recorded. Sciatic nerve block and femoral nerve block were performed 10 min after dexmedetomidine infusion and 5 min after propofol infusion via anterior approach using

Stimuplex<sup>®</sup> A (21 G 0.80–150 mm) block needles (B. Braun, Melsungen AG, Germany), of which the isolated tip was 30°, under ultrasonography (Diagnostic ultrasound system, Model SDU 450 XL Class-1 type B, Shimadzu Corporation, Yokohama, Japan) guidance together with nerve stimulator (Stimuplex HNS nerve stimulator, Braun, Melsungen, Germany). A total of 40 mL anesthetic solution including 30 mL of 0.5% isobaric bupivacaine and 10 mL of 2% lidocaine was prepared. The nerves were stimulated by a stimulus with 2 Hz frequency and 1 mA flow; stimulus intensity was gradually decreased to 0.4 mA.

In femoral nerve block,<sup>18</sup> contractions of vastus medialis, vastus intermedialis and vastus lateralis were observed individually and the dispersion of local anesthetic was demonstrated by ultrasonography (linear probe) during the infusion of 20 mL of local anesthetic mixture. In sciatic nerve block,<sup>19</sup> when the plantar flexion, dorsiflexion and eversion of the foot were seen, the dispersion of local anesthetic was demonstrated by ultrasonography (convex probe) during the infusion of 20 mL of local anesthetic mixture.

After the block was performed in both groups, the block was evaluated by cold compress with ice battery; time to start sensory block was recorded. On the 30th min after the block, the patients were transferred for the surgery and RSS scores were recorded every 5 min. All patients were followed up by the same anesthesiologist.

Dexmedetomidine and propofol infusion rates were fixed in both groups, when needed, to fix the sedation levels between 2 and 4 according to the RSS.

Dexmedetomidine and propofol infusions were discontinued at the end of surgery and the patients were transferred to the postoperative care unit. The patients, then, transferred to the clinic when their modified Aldrete scores were  $\geq 9$ .

## Statistical analysis

The 10-case pre-study that we conducted showed that the mean artery pressure in the dexmedetomidine group was  $82 \pm 5\ \text{mm Hg}$  and  $76 \pm 5\ \text{mm Hg}$  in the propofol group; as the mean artery pressure difference is 6 mmHg and the standard effect size is 1.2 with 95% power and 5% fallibility,  $n=20$  for each group. All data for the study was evaluated with the SPSS package program for Windows 11.05. All data were expressed as mean  $\pm$  standard deviation. The normality analysis of the data was conducted with the Kolmogorov–Simirnov test. The study groups were also compared by chi-square test with respect to age, height, body weight, gender, ASA classification. The study groups were compared by independent sample *t* test in terms of arterial blood pressure, heart rate and peripheral oxygen saturation used. The study groups were also compared using the Mann–Whitney *U* test with Bonferroni correction with respect to sedation levels during surgery according to RSS, and time to start and end sedation. A *p* value  $<0.05$  was considered statistically significant.

## Results

Age ( $p=0.901$ ), height ( $p=0.852$ ), body weight ( $p=0.112$ ), gender ( $p=0.714$ ), ASA classification ( $p=1000$ ), surgery

**Table 1** Distribution of general characteristics of the study groups.

	Group D	Group P	<i>p</i>
Age (year)	38.05 ± 12.03	38.55 ± 12.30	0.901
Height (mm)	169.95 ± 10.22	170.50 ± 8.20	0.852
Body weight (kg)	72.55 ± 16.65	79.80 ± 10.77	0.112
Gender (male/female)	15/5	16/4	0.714
ASA (ASA I/ASA II)	15/5	17/3	1.000
Surgery duration (min)	80.75 ± 26.27	89.80 ± 33.81	0.351
Tourniquet time (min)	67.55 ± 22.88	69.22 ± 27.33	0.654

D, dexmedetomidine; P, propofol.

Data are expressed as mean ± standard deviation or number where appropriate.

duration ( $p=0.351$ ) and Tourniquet time ( $p=0.654$ ) of the patients are presented in Table 1. There were no significant differences between Group D and Group P in terms of these data.

There were no significant differences between the groups in terms of arterial blood pressure, heart rate, and peripheral oxygen saturation measured every 5 min after dexmedetomidine or propofol infusion was started. Blood pressure values of the patients are presented in Table 2 and heart rate and peripheral oxygen saturation values are presented in Table 3.

RSS scores measured every 5 min after the start of surgery are presented in Table 4. There was no significant difference between the groups in terms of intraoperative RSS scores.

Mean time to start sedation (RSS scores between 2 and 4) after the start of dexmedetomidine or propofol infusion was found to be significantly longer in Group D as compared to that in Group P ( $8.10 \pm 1.07$  and  $3.80 \pm 0.83$ , respectively;  $p < 0.001$ ). Time to recovery from sedation following discontinuation of dexmedetomidine or propofol infusion (modified Aldrete sedation scale score = 9) was significantly

**Table 2** Comparison of intraoperative blood pressure values between the study groups.

Blood pressure (mm Hg)	Group D	Group P	<i>p</i>
<i>Basal</i>			
Systolic	128.70 ± 16.46	133.05 ± 14.88	0.386
Diastolic	73.85 ± 11.88	74.90 ± 12.44	0.786
<i>5th min of the infusion</i>			
Systolic	114.40 ± 30.97	122.80 ± 21.20	0.324
Diastolic	70.05 ± 14.94	71.35 ± 11.05	0.756
<i>5th min after peripheral nerve block</i>			
Systolic	114.45 ± 9.38	119.10 ± 16.68	0.284
Diastolic	70.00 ± 10.55	68.35 ± 9.90	0.613
<i>15th min after peripheral nerve block</i>			
Systolic	124.55 ± 15.77	121.00 ± 17.18	0.50
Diastolic	75.05 ± 13.29	71.45 ± 12.52	0.383
<i>5th min of the surgery</i>			
Systolic	122.95 ± 13.96	123.50 ± 15.97	0.908
Diastolic	74.50 ± 13.56	74.25 ± 10.09	0.948
<i>10th min of the surgery</i>			
Systolic	126.15 ± 15.07	126.85 ± 15.80	0.887
Diastolic	79.85 ± 13.39	76.55 ± 13.79	0.447
<i>20th min of the surgery</i>			
Systolic	126.40 ± 16.06	126.95 ± 32.11	0.946
Diastolic	78.95 ± 10.74	78.25 ± 16.13	0.823
<i>30th min of the surgery</i>			
Systolic	129.40 ± 14.95	131.75 ± 13.70	0.607
Diastolic	79.15 ± 14.25	78.10 ± 12.13	0.803

D, dexmedetomidine; P, propofol.

Data are expressed as mean ± standard deviation.

**Table 3** Comparison of the study groups in terms of heart rate and peripheral oxygen saturation.

	Group D	Group P	<i>p</i>
<i>Heart rate (beat min<sup>-1</sup>)</i>			
Basal	83.90 ± 18.47	83.40 ± 11.77	0.919
5th min of the infusion	76.25 ± 19.25	78.35 ± 12.72	0.686
5th min after peripheral nerve block	69.20 ± 16.91	76.80 ± 11.50	0.105
5th min of the surgery	65.95 ± 13.98	75.45 ± 11.89	0.056
10th min of the surgery	66.45 ± 13.11	71.15 ± 11.73	0.239
20th min of the surgery	64.45 ± 11.78	71.25 ± 11.79	0.076
30th min of the surgery	70.40 ± 13.52	76.30 ± 13.66	0.178
<i>Peripheral oxygen saturation (%)</i>			
Basal	98.35 ± 1.27	97.90 ± 2.49	0.476
5th min of the infusion	98.35 ± 1.84	98.90 ± 2.36	0.417
5th min after peripheral nerve block	98.85 ± 1.57	99.22 ± 1.82	0.519
5th min of the surgery	99.25 ± 0.91	99.20 ± 2.50	0.934
10th min of the surgery	99.25 ± 1.02	98.90 ± 3.37	0.659
20th min of the surgery	99.35 ± 0.81	99.80 ± 3.37	0.482
30th min of the surgery	99.25 ± 0.85	98.80 ± 3.78	0.606

D, dexmedetomidine; P, propofol.

Data are expressed as mean ± standard deviation.

**Table 4** Comparison of the groups in terms of intraoperative Ramsay sedation scale scores.

	Group D	Group P	<i>p</i>
<i>Ramsay sedation scale scores</i>			
5th min of the surgery	2.80 ± 1.28	2.85 ± 1.42	0.912
10th min of the surgery	3.15 ± 1.27	3.15 ± 1.14	0.966
20th min of the surgery	3.15 ± 1.23	3.15 ± 1.09	0.978
30th min of the surgery	3.30 ± 0.92	3.50 ± 0.94	0.495

D, dexmedetomidine; P, propofol.

Data are expressed as mean ± standard deviation.

longer in Group D than that in Group P ( $22.30 \pm 3.32$  min and  $9.90 \pm 2.10$  min, respectively, [Tables 5 and 6](#);  $p < 0.001$ ).

With regard to adverse events, one patient in Group D developed bradycardia ( $<60$  rate  $\text{min}^{-1}$ ) and was treated with 0.5 mg atropine intravenously.

Twelve patients were excluded because of inadequate block (9 sciatic and 3 sciatic + femoral).

In Group D, one patient did not even reach the RSS level of 2 with  $0.5 \mu\text{g kg}^{-1} \text{h}^{-1}$  dose of dexmedetomidine whereas

2 patients reached the level of RSS 5–6; however, 4 patients in the Group P had an RSS level of 5–6.

## Discussion

Benzodiazepines, propofol,  $\alpha$ -2 adrenoceptor agonists such as clonidine and dexmedetomidine, sevoflurane, ketamine and opioids are used to reduce procedure-associated fear and anxiety to enhance comfort to patient.<sup>20</sup> Although

**Table 5** The time it took for the Ramsay sedation scale points to achieve the range of 2–4 after the dexmedetomidine and propofol infusion was initiated in the groups.

	Group D	Group P	<i>p</i>
Time passed for the Ramsay sedation scale points to achieve the range 2–4 (min)	8.10 ± 1.07	3.80 ± 0.83	* <0.001

D, dexmedetomidine; P, propofol.

Data are expressed as mean ± standard deviation.

\* When Group D and Group P are compared in terms of their Ramsay sedation scale points achieving the range of 2–4 after the infusion of dexmedetomidine and propofol was initiated, this period was longer in Group D than in Group P and there were statistically significant differences present between Group D and Group P,  $<0.001$ .

**Table 6** The time it took for the modified Aldrete sedation scale points to achieve 9 after the dexmedetomidine and propofol infusion was suspended.

	Group D	Group P	<i>p</i>
Time passed for the modified Aldrete sedation scale points to achieve 9 (min)	22.30 ± 3.32	9.90 ± 2.10	* <0.001

D, dexmedetomidine; P, propofol.

Data are expressed as mean ± standard deviation.

\* When Group D and Group P are compared in terms of their modified Aldrete sedation scale points achieving 9 after the infusion of dexmedetomidine and propofol was suspended, this period was longer in Group D than in Group P and there were statistically significant differences present between Group D and Group P, <0.001.

dexmedetomidine is being used in intensive care units for a long time for sedation, there is limited information about its use for intraoperative sedation.<sup>4</sup> In the present study, we aimed to evaluate whether dexmedetomidine can be used for sedation during peripheral nerve block and to investigate the effects of dexmedetomidine on sedation characteristics in comparison to propofol.

Dexmedetomidine is an  $\alpha$ -2 receptor agonist. Studies have shown that  $\alpha$ -2 receptor agonists have analgesic, sedative-hypnotic and sympatholytic properties.<sup>21,22</sup> There are studies suggesting that dexmedetomidine is an effective drug in intraoperative sedation.<sup>23,24</sup> Shehabi et al.<sup>25</sup> performed dexmedetomidine infusion (0.2–0.7  $\mu\text{g kg}^{-1} \text{h}^{-1}$ ) for an average of 71.5 h for sedation of 60 critical patients in the intensive care unit with a fixed dose infusion to achieve score between 2 and 4. They fixed the infusion dose that RSS score would be between 2 and 4. They concluded that dexmedetomidine can be used for an effective sedation and as a substitute analgesic in critical patients without causing significant changes in vital signs within 24 h period. In the present study, we used dexmedetomidine for sedation during anterior sciatic nerve block in combination with femoral nerve block. We fixed the dexmedetomidine infusion dose to be 0.5  $\mu\text{g kg}^{-1} \text{h}^{-1}$  and applied a loading dose of 1  $\mu\text{g kg}^{-1}$  for 10 min prior to the infusion; thus, In our study, 2 out of 23 patients had reached the level of RSS 5–6 with 0.5  $\mu\text{g kg}^{-1} \text{h}^{-1}$  dose of dexmedetomidine; however, these patients did not develop respiratory depression and their vital findings were within physiological limits. No significant change was observed in the vital signs of the patients in Group D and vital signs were also similar to that of Group P.

Intravenous use of sedative and hypnotic drugs during regional anesthesia as supportive medication enhances patient comfort<sup>26,27</sup> with minimum morbidity and mortality and maintains cardiovascular stability by lowering perioperative stress.<sup>28</sup> In the present study, we aimed to enhance patient comfort with dexmedetomidine and propofol infusion and observed that the similar sedation had been achieved in Group D as compared to that in Group P.

An ideal sedation during regional anesthesia requires an open airway, a reliable sleep state, minimally influenced cardiovascular system, and a rapid recovery period from anesthesia.<sup>1</sup> MRI sleep studies conducted on the children with obstructive sleep apnea have demonstrated that dexmedetomidine requires low rate of airway intervention as compared to propofol. Although dexmedetomidine has caused significant decrease in heart rate and propofol has caused significant decrease in arterial blood pressure, the

procedure has not been discontinued and MRI sleep study has been completed successfully.<sup>29</sup> In the present study, none of the patients required airway support and no significant difference was observed between the groups in terms of vital signs. However, time to recovery from anesthesia was found significantly longer in Group D as compared to Group P.

Intraoperative sedation during regional anesthesia enhances the quality of local and regional anesthesia by providing optimum patient comfort with minimum morbidity and mortality. Nevertheless, it is very difficult to provide adequate level of sedation due to the variabilities in the patient expectations regarding sedation level, differences in intraoperative conditions, and pharmacokinetic and pharmacodynamic properties of the agents. In the present study, we tried to preserve the adequate level of sedation by successful adjustment of infusion rates of dexmedetomidine and propofol. The RSS scores of the patients were found to be similar in both study groups.

It is stated that dexmedetomidine is frequently used for sedation in intensive care units and that it is an agent with which patient cooperation is better as compared to other drugs.<sup>30,31</sup> In the present study, the level of sedation was kept between 2 and 4 according to RSS and no significant difference was obtained between the study groups in terms of sedation at that level; thus, the cooperation levels of the study groups were found to be similar.

Dexmedetomidine provides dose-dependent sedation and prolongs the sensorial block. However, it has been stated that dexmedetomidine may cause unintended hemodynamic impairment, and nausea and vomiting.<sup>31</sup> Neither nausea, vomiting nor unintended hemodynamic impairment was observed in the present study.

Intravenous  $\alpha$ -2 adrenoceptor agonist administration causes a decrease in heart rate, and temporary increase in arterial blood pressure and systemic vascular resistance due to the activation of postjunctional vascular  $\alpha$ -2 adrenoceptors. Subsequently, long-term decrease in heart rate and blood pressure is observed due to the decrease in central sympathetic tonus and vagal activity.<sup>4,31</sup> Dexmedetomidine provides a predictable stable hemodynamics when administered as continuous infusion. In the present study, blood pressure and heart rate values were found similar in both study groups. In Group D, one patient developed bradycardia and was treated with 0.5 mg atropine intravenously. Moreover, patients with hypovolemia, vasoconstriction or severe cardiac block had already been excluded from the study since they were likely to develop hypotension and bradycardia.

The most important advantage of dexmedetomidine is that it does not cause respiratory depression, although it may cause deep sedation at therapeutic doses.<sup>3,32,33</sup> In our study, 2 out of 23 patients in Group D had reached the level of RSS 5–6 with 0.5  $\mu\text{g kg}^{-1} \text{h}^{-1}$  dose of dexmedetomidine; however, these patients did not develop respiratory depression and their vital findings were within physiological limits.

There are similar studies, which used dexmedetomidine for intraoperative sedation in awake patients.<sup>24,25</sup> Arain et al.<sup>17</sup> compared dexmedetomidine and propofol during intraoperative sedation and found no difference in psychomotor performance and respiratory rate during recovery. They also found that later onset and termination of sedation was observed in the dexmedetomidine group as compared to the propofol group, whereas postoperative analgesia was better and analgesic consumption was lower in the dexmedetomidine group. In the present our study, the effects of dexmedetomidine and propofol on the postoperative analgesic characteristic of sciatic–femoral block were found similar since half-lives of dexmedetomidine and propofol was shorter than action time of sciatic–femoral block. However, consistent with the above-mentioned study, later onset and termination of sedation was observed in dexmedetomidine group than propofol group in the present study.

### Limitation

The result of the primary measurement of this study is the RSS values after dexmedetomidine and propofol sedation. Future studies that show that dexmedetomidine can be used for sedation during the intra-operative period and that present the effects of dexmedetomidine on the characteristics of the sciatic–femoral block with an anterior approach should be encouraged. The RSS used to identify the degree of sedation is subjective; it is limited due to the patient providing verbal evaluations. RSS is a subjective measurement; however, it has been described previously.<sup>15</sup> RSS does not provide objective data regarding the sedation status of the patient. RSS values depend on the particular pharmacodynamic and pharmacokinetic characteristics of the sedative drug on different people.<sup>34</sup> The patients' vital findings were evaluated and their compatibility with the RSS values were compared. Verbal dialogs were continued with the patient in order to perform a correct and accurate sedation evaluation. The patients were informed on RSS before the operation. The RSS inquiry was performed on all patients by the same anesthetist. Future studies will study using objective methods to identify the degree of sedation while intra-operatively using dexmedetomidine.

### Conclusion

In conclusion, it was observed that sedation could be achieved in the range of RSS 2–4 via dexmedetomidine at a dose of 0.5  $\mu\text{g kg}^{-1} \text{h}^{-1}$  or propofol infusion at a dose of 3  $\text{mg kg}^{-1} \text{h}^{-1}$  administered for sedation purposes on patients undergoing sciatic–femoral nerve block with an anterior approach. It was considered that dexmedetomidine had propofol-like effects on patients undergoing

a sciatic–femoral nerve block with an anterior approach and that it could be used in place of propofol. However, it was observed that some patients could have RSS levels of 5–6 via the infusion of dexmedetomidine at a dose of 0.5  $\mu\text{g kg}^{-1} \text{h}^{-1}$  or propofol at a dose of 3  $\text{mg kg}^{-1} \text{h}^{-1}$ . Similarly, it was observed that some patients could experience inadequate sedation via dexmedetomidine infusion. It was observed that the time between sedation onset and ending with dexmedetomidine at a dose of 0.5  $\mu\text{g kg}^{-1} \text{h}^{-1}$  was longer than sedation time induced via propofol at a dose of 3  $\text{mg kg}^{-1} \text{h}^{-1}$  at a statistically significant level for patients undergoing sciatic–femoral nerve block with an anterior approach.

### Conflicts of interest

The authors declare no conflicts of interest.

### References

1. Olkkola KT, Ahonen J. Midazolam and other benzodiazepines. *Handb Exp Pharmacol.* 2008;182:335–60, [http://dx.doi.org/10.1007/978-3-540-74806-9\\_16](http://dx.doi.org/10.1007/978-3-540-74806-9_16).
2. Mantz J, Josserand J, Hamada S. Dexmedetomidine: new insights. *Eur J Anaesthesiol.* 2011;28:3–6, <http://dx.doi.org/10.1097/EJA.0b013e32833e266d>.
3. Hoy SM, Keating GM. Dexmedetomidine: a review of its use for sedation in mechanically ventilated patients in an intensive care setting and for procedural sedation. *Drugs.* 2011;30:1481–501, <http://dx.doi.org/10.2165/11207190-000000000-00000>.
4. Ok HG, Baek SH, Baik SW, Kim HK, Shin SW, Kim KH. Optimal dose of dexmedetomidine for sedation during spinal anesthesia. *Korean J Anesthesiol.* 2013;64:426–31, <http://dx.doi.org/10.4097/kjae.2013.64.5.426>.
5. Siddappa R, Riggins J, Kariyanna S, Calkins P, Rotta AT. High-dose dexmedetomidine sedation for pediatric MRI. *Paediatr Anaesth.* 2011;21:153–8, <http://dx.doi.org/10.1111/j.1460-9592.2010.03502.x>.
6. Kunisawa T, Hanada S, Kurosawa A, Suzuki A, Takahata O, Iwasaki H. Dexmedetomidine was safely used for sedation during spinal anesthesia in a very elderly patient. *J Anesth.* 2010;24:938–41, <http://dx.doi.org/10.1007/s00540-010-1025-z>.
7. McVey JD, Tobias JD. Dexmedetomidine and ketamine for sedation during spinal anesthesia in children. *J Clin Anesth.* 2010;22:538–45, <http://dx.doi.org/10.1016/j.jclinane.2010.03.002>.
8. Dogan R, Erbek S, Gonencer HH, Erbek HS, Isbilen C, Arslan G. Comparison of local anaesthesia with dexmedetomidine sedation and general anaesthesia during septoplasty. *Eur J Anaesthesiol.* 2010;27:960–4, <http://dx.doi.org/10.1097/EJA.0b013e32833a45c4>.
9. Mahfouz AK, Ghali AM. Combined use of remifentanyl and propofol to limit patient movement during retinal detachment surgery under local anesthesia. *Saudi J Anesth.* 2010;4:147–51, <http://dx.doi.org/10.4103/1658-354X.71570>.
10. Neumann MM, Davio MB, Macknet MR, Applegate RL. Dexmedetomidine for awake fiberoptic intubation in a parturient with spinal muscular atrophy type III for cesarean delivery. *Int Obstet Anesth.* 2009;18:403–7, <http://dx.doi.org/10.1016/j.ijoa.2009.05.002>.
11. Toju K, Hakozaiki T, Akatsu M, Isosu T, Murakawa M. Ultrasound-guided bilateral brachial plexus blockade with propofol–ketamine sedation. *J Anesth.* 2011;25:627–9, <http://dx.doi.org/10.1007/s00540-011-1238-9>.

12. Uz A, Apaydin N, Cinar SO, et al. A novel approach for anterior sciatic nerve block: cadaveric feasibility study. *Surg Radiol Anat.* 2010;32:873–8, <http://dx.doi.org/10.1007/s00276-010-0642-4>.
13. Imbelloni BE, Rava C, Gouveia MA. A new, lateral, continuous, combined, femoral-sciatic nerve approach via a single skin puncture for postoperative analgesia in intramedullary tibial nail insertion. *Local Rec Anesth.* 2013;6:9–12, <http://dx.doi.org/10.2147/LRA.S37261>.
14. Tan WP, Talbott VA, Leong QQ, Isenberg GA, Goldstein SD. American Society of Anesthesiologists class and Charlson's Comorbidity index as predictors of postoperative colorectal anastomotic leak: a single-institution experience. *J Surg Res.* 2013;4804:00489–497, <http://dx.doi.org/10.1016/j.jss.2013.05.039>.
15. Balaban B, Yağar S, Özgök A, Koç M, Güllapoğlu H. A randomized, placebo-controlled study of pregabalin for postoperative pain intensity after laparoscopic cholecystectomy. *J Clin Anesth.* 2012;24:175–8, <http://dx.doi.org/10.1016/j.jclinane.2011.06.027>.
16. Yazar MA, Inan N, Ceyhan A, Sut E, Dikmen B. Postoperative analgesic efficacy of intravenous dexketoprofen in lumbar disc surgery. *J Neurosurg Anesthesiol.* 2011;23:193–7, <http://dx.doi.org/10.1097/ANA.0b013e31820d1ebb>.
17. Arain SR, Ebert TJ. The efficacy, side effects, and recovery characteristics of dexmedetomidine versus propofol when used for intraoperative sedation. *Anesth Analg.* 2002;95:461–6, <http://dx.doi.org/10.1213/01.ANE.0000019085.69108.A3>.
18. Altermatt FR, Corvetto MA, Veneqas C, et al. Brief report: the sensitivity of motor responses for detecting catheter-nerve contact during ultrasound-guided femoral nerve blocks with stimulating catheters. *Anesth Analg.* 2011;113:1276–8, <http://dx.doi.org/10.1213/ANE.0b013e31822c9887>.
19. Ota J, Sakura S, Hara K, Saito Y. Ultrasound-guided anterior approach to sciatic nerve block: a comparison with the posterior approach. *Anesth Analg.* 2009;108:660–5, <http://dx.doi.org/10.1213/ane.0b013e31818fc252>.
20. Borgeat A, Aquirre J. Sedation and regional anesthesia. *Curr Opin Anesthesiol.* 2009;22:678–82, <http://dx.doi.org/10.1097/ACO.0b013e31832f3320>.
21. Chrysostomou C, Schmitt CG. Dexmedetomidine: sedation, analgesia and beyond. *Expert Opin Drug Metab Toxicol.* 2008;4:619–27, <http://dx.doi.org/10.1517/17425255.4.5.619>.
22. Parikh DA, Kolli SN, Karnik HS, Lele SS, Tendolkar BA. A prospective randomized double-blind study comparing dexmedetomidine vs. combination of midazolam-fentanyl for tympanoplasty surgery under monitored anesthesia care. *J Anaesthesiol Clin Pharmacol.* 2013;29:173–8, <http://dx.doi.org/10.4103/0970-9185.111671>.
23. Rozet I. Anesthesia for functional neurosurgery: the role of dexmedetomidine. *Curr Opin Anesthesiol.* 2008;21:537–43, <http://dx.doi.org/10.1097/ACO.0b013e3183280edafd>.
24. Rozet I, muangman S, Vavilala MS, et al. Clinical experience with dexmedetomidine for implantation of deep brain stimulators in Parkinson's disease. *Anesth Analg.* 2006;103:1224–8, <http://dx.doi.org/10.1213/01.ane.0000239331.53085.94>.
25. Shehabi Y, Ruettimann U, Adamson H, Innes R, Ickeringill M. Dexmedetomidine infusion for more than 24 hours in critically ill patients: sedative and cardiovascular effects. *Intensive Care Med.* 2004;30:2188–96, <http://dx.doi.org/10.1007/s00134-004-2417-z>.
26. O'Sullivan MJ. Patient comfort in regional anesthesia. *Anesth Analg.* 2008;106:349–50, <http://dx.doi.org/10.1213/01.ane.0000297283.37494.ab>.
27. Sinha A. Patient comfort during regional anesthesia: the obvious and the obscure. *J Clin Anesth.* 2007;19:1–2, <http://dx.doi.org/10.1016/j.jclinane.2006.10.003>.
28. Powers KS, Nazarian EB, Taypriks SA, et al. Bispectral index as a guide for titration of propofol during procedural sedation among children. *Pediatrics.* 2005;115:1666–74, <http://dx.doi.org/10.1542/peds.2004-1979>.
29. Mahmoud M, Gunter J, Donnelly LF, et al. A comparison of dexmedetomidine with propofol for magnetic resonance imaging sleep studies in children. *Anesth Analg.* 2009;109:745–53, <http://dx.doi.org/10.1213/ane.0b013e3181adc506>.
30. Mason KP, Lerman J. Review article: dexmedetomidine in children: current knowledge and future applications. *Anesth Analg.* 2011;113:1129–42, <http://dx.doi.org/10.1213/ANE.0b013e31822b8629>.
31. Bindu B, Pasupuleti S, Gowd UP, Gorre V, Murthy RR, Laxmi MB. A double blind, randomized, controlled trial to study the effect of dexmedetomidine on hemodynamic and recovery responses during tracheal extubation. *J Anesthesiol Clin Pharmacol.* 2013;29:162–7, <http://dx.doi.org/10.4103/0970-9185.111665>.
32. Prasad SR, Simha PP, Jagadeesh AM. Comparative study between dexmedetomidine and fentanyl for sedation during mechanical ventilation in post-operative paediatric cardiac surgical patients. *Indian J Anaesth.* 2012;56:547–52, <http://dx.doi.org/10.4103/0019-5049.104572>.
33. Afonso J, Reis F. Dexmedetomidine: current role in anesthesia and intensive care. *Rev Bras Anesthesiol.* 2012;62:118–33, [http://dx.doi.org/10.1016/S0034-7094\(12\)70110-1](http://dx.doi.org/10.1016/S0034-7094(12)70110-1).
34. Ihmsen H, Saari TI. Dexmedetomidine. Pharmacokinetics and pharmacodynamics. *Anaesthesist.* 2012;61:1059–66, <http://dx.doi.org/10.1007/s00101-012-2114-1>.