Original Article
Comparison of dexmedetomidine and propofol used for drug-induced sleep endoscopy in patients with obstructive sleep apnea syndrome

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Abstract: Background: Surgical operations are alternative treatments in persons with Obstructive Sleep Apnea Syndrome who cannot tolerate continuous positive airway pressure therapy. Drug-Induced Sleep Endoscopy is a method with which somnolence is pharmacologically induced and collapse is evaluated through nasal endoscopy in patients with Obstructive Sleep Apnea Syndrome. Aims: We aimed to evaluate efficiency of dexmedetomidine or propofol used for sedation in patients undergoing drug-induced sleep endoscopy. Methods: A total of 40 patients aged between 18 and 65 years old in the ASA STATUS I-II group were included in the study. After premedication with midazolam 0.05 mg/kg intravenously, patients were randomly divided into two groups and administered intravenous (iv) propofol with the loading dose of 0.7 mg/kg for 10 minutes, followed 0.5 mg/kg/h infusion (Group P); or dexmedetomidine with the loading dose of 1 mcg/kg for 10 minutes, followed by 0.3 mcg/kg/h infusion (Group D). Haemodynamic and respiratory parameters, Bispectral index score, Ramsey sedation score, time to achieve sufficient sedation, surgeon’s and patients’ satisfaction, postoperative Aldrete score and side effects were recorded. Results: Time to achieve sufficient sedation, Bispectral index scores at 5, 10 and 15th. minutes intraoperatively, first Aldrete score in the recovery room, \( \text{SpO}_2 \) values and respiratory rates all over the surgical procedure and in the recovery room were found lower in Group P (\( P<0.05 \)). Bispectral index scores, mean arterial pressure and heart rate in the recovery room were significantly lower in Group D (\( P<0.05 \)). Conclusion: Dexmedetomidine may be preferred as a safer agent with respecting to respiratory function compared with propofol in obstructive sleep apnea patients who known to be susceptible to hypoxia and hypercarbia.

Keywords: Obstructive sleep apnea syndrome, drug induced sleep endoscopy, sedation, propofol, dexmedetomidine

Introduction
Obstructive Sleep Apnea (OSAS) is a syndrome characterized by the episodes of apnea or hypopnea due to obstruction in the upper airways [1]. The primary pathology is chronic intermittent hypoxia, resulting in the development of systemic inflammation and several comorbidities causing morbidity and mortality such as hypertension and stroke [2]. Surgical procedures of the upper airways are the treatment options especially for the patients who cannot tolerate continuous positive airway pressure therapy (CPAP) [3].

Drug-Induced Sleep Endoscopy (DISE) is a method with which somnolence is induced pharmacologically and obstruction in the upper airways is evaluated through nasal endoscopy in patients with OSAS [3-5].

Patients with OSAS are at a high risk for anesthesia-related mortality and morbidity. It is suggested that general anesthesia should be pre-
ferred to the deep sedation produced without providing safety of the airway. Whereas, general anesthesia applied on the OSAS patients for simple diagnostic and interventional procedures may eliminate the chance for being an ‘outpatient’ [6, 7].

Use of sedative drugs providing rapid recovery without respiratory and cardiac depression in DISE procedures in these patients who are prone to develop hypoxia and hypercapnia will provide a safer sedation method. Benzodiazepines and propofol have been used alone or in combination for DISE [8, 9]. Dexmedetomidine is a high selective α-2 adrenergic receptor agonist with anxiolytic and analgesic effects. As dexmedetomidine does not depress respiratory function and has a wide safety margin, it is preferable for anesthetists using in sedoanalgesia in the invasive procedures at the operation theatre as well as used in the intensive care unit [8-11].

In the literature, there are limited studies about the use of sedoanalgesic drugs in DISE. In this randomized, double blinded clinical study, we aimed to compare the sedative, haemodynamic and respiratory effects and, surgeon and patient satisfaction of dexmedetomidine and propofol in patients with OSAS undergoing DISE.

**Methods**

**Study design, data collection and procedures**

Our study was conducted on 40 patients in ASA STATUS I-II, aged between 18-50 years old, considering OSAS diagnosed by polysomnography undergoing nasal endoscopy under sedation for DISE procedure by the anesthesiology department of Erzincan University Mengücekgazi Training and Research Hospital.

Patients under 18 years old, drug or alcohol abusers or those having history of chronic analgesic use, patients who know to have allergy against the study drugs, those with IInd-IIIrd degree A-V block, patients with psychiatric disorders and those have Mallampati scores of III-IV were excluded from the study.

Following a fasting for 8 hours, patients were taken to the operating room and a peripheral intravenous cannulation on the dorsal side of the hand was performed. A balanced crystalloid (ISOLYTE-S, BRAUN, USA) 6 mL/h infusion was applied for 20 minutes. The infusion was maintained during the operation with a rate of 8 mL/h. Mean arterial pressure (MAP), peripheral oxygen saturation (SpO₂), heart rate (HR), respiratory rate (RR) (PHILPS MP-20 Philips Electronics Japan, Ltd, Tokyo, Japan) and Bispectral index Score (BIS) (BISTM Complete 2-Channel Monitor and 4 Electrode Sensor, both from Covidien, Mansfield, MA, US) values and Ramsey Sedation Scores (RSS) were recorded.

All the patients were administered intravenous (iv) bolus of 0.05 mg/kg midazolam (DORMICUM, DEVA ILAC, ISTANBUL TURKEY) and randomly divided into two groups: In Group P (n=20), patients were administered iv propofol (PROPOFOL-LIPURA, B.BRAUN, MELSUNGEN AG, GERMANY) with the loading dose of 0.7 mg/kg for 10 minutes followed by the maintenance dose of 0.5 mg/kg/h; and in Group D (n=20), patients were administered iv dexmedetomidine (PRECEDEX, HOSPIRA, NORTH ROCKY MOUNT, USA) with the loading dose of 1 mcg/kg for 10 minutes followed by the maintenance dose of 0.3 mcg/kg/h. The maximal dexmedetomidine dose to be infused was planned as 0.6 mcg/kg/h. Dose of the drugs was titrated by increasing with 0.1 mg/kg/h propofol in Group P and 0.1 mcg/kg/h dexmedetomidine in Group D at 5 minute intervals until a sufficient sedation level was achieved.

Loading and maintenance doses of the study drugs were infused by using infusion pumps (INFUSOMAT SPACE, B.BRAUN, MELSUNGEN AG, GERMANY) in both groups. Patients who did not reach to the desired sedation level despite the increased doses were excluded from the study. Infusion was terminated at the end of nasal endoscopy.

All patients received 2 L/minutes oxygen during the procedure through a nasal cannula.

Heart Rate, MAP, RR, SpO₂, RSS and BIS values were recorded at 5 minute intervals; at 5, 10, and 15 minutes during the surgical procedure. Time to achieve sufficient sedation was recorded as minute (min).

Time to achieve sufficient sedation was defined as the duration between initiation of the drug infusion and the time when RSS:4 and BIS <75 values were obtained [12, 31]. Aldrete recovery scores, haemodynamic and respiratory parameters, BIS, RSS and side effects, if occurs, were
recorded when the patients arrived to the recovery room (0), and at 10, 20 and 30 minutes. After monitoring 30 min in the recovery room, patients were allowed to be taken to the ward with an Aldrete score >9, then monitored in the ward for 4 hours and discharged at the end of this duration. patients’ and surgeon’s satisfaction were recorded using 7-point likert-like verbal rating scale [32].

In case of heart rate dropped under 50 beats/minute and continued for 15 seconds in the intraoperative period, it was considered as bradycardia and atropine 0.5 mg iv was administered; similarly in case of MAP dropped by more than 30% of the initial value and continued for 60 seconds, rate of iv crystalloid infusion was raised to 20 ml/minute and hypotension therapy was planned. Oxygen administration of 5 L/minutes with a mask if SpO₂ <92 and positive pressure ventilation with Ambu when the RR fell below 8 was added to the treatment protocol.

**Statistical analysis**

Statistical analyses were carried out using the Statistical package for Social Sciences for Windows version 15.0 (SPSS, Chicago, IL, USA). Descriptive statistics for each variable were determined. Normality of the data distribution was assessed with the Kolmogorov-Smirnov test. Normally distributed data were expressed as mean ± standard deviation. Median and minimum-maximum values were used for variables without normal distribution. Data with normal distribution were compared by Student’s t test Comparisons of continuous variables with asymmetric distribution were made by using the Mann-Whitney U test. A P value less than 0.05 were considered significant.

**Results**

No significant difference was found between the groups in terms of age, Body Mass Index (BMI), ASA scores, duration of the operation and, patient and surgeon’s satisfaction (P>0.05, Table 1).

When haemodynamic data were examined, no significant difference was found between the groups in terms of intraoperative MAP and HR (P>0.05, Table 2; Figure 1). Whereas at the measurements in the recovery room, both MAP

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**Table 1. Demographic data, duration of surgery, patient and surgeon’s satisfaction scores**

<table>
<thead>
<tr>
<th></th>
<th>Group P</th>
<th>Group D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>43.3±10.6</td>
<td>47.4±11.6</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.9±3.9</td>
<td>29.5±4.1</td>
</tr>
<tr>
<td>ASA Scores (min-max)</td>
<td>1 (1-2)</td>
<td>1 (1-2)</td>
</tr>
<tr>
<td>Duration of Surgery (min)</td>
<td>5.6±1.4</td>
<td>5.1±0.8</td>
</tr>
<tr>
<td>Patients’ Sat (min-max)</td>
<td>7 (6-7)</td>
<td>7 (6-7)</td>
</tr>
<tr>
<td>Surgeon’s Sat (min-max)</td>
<td>6 (6-7)</td>
<td>7 (6-7)</td>
</tr>
</tbody>
</table>

BMI, Body Mass Index; ASA Sc, American Society of Anaesthesologists Score; Patient’s Sat, Patients’ Satisfaction Score; Surgeon’s Sat, Surgeon’s Satisfaction Score.

**Table 2. Haemodynamic and respiratory data**

<table>
<thead>
<tr>
<th>Periods (min)</th>
<th>0</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>R 0</th>
<th>R 10</th>
<th>R 20</th>
<th>R 30</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP (mmHg)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Group P</td>
<td>94±7.5</td>
<td>90.6±8.4</td>
<td>83.7±7.2</td>
<td>81.3±7.6</td>
<td>81.7±7.9&quot;</td>
<td>86.6±7.6&quot;</td>
<td>90.2±7.3&quot;</td>
<td>92.9±6.9&quot;</td>
</tr>
<tr>
<td>Group D</td>
<td>90.2±7.1</td>
<td>85.6±7.9</td>
<td>82.7±8.2</td>
<td>77.6±10</td>
<td>71.8±7.9</td>
<td>73.4±7.8</td>
<td>75.2±8</td>
<td>79.2±8</td>
</tr>
<tr>
<td>HR (min⁻¹)</td>
<td></td>
<td></td>
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<tr>
<td>Group P</td>
<td>82.9±6.9</td>
<td>74.1±5.9</td>
<td>69.7±5.1</td>
<td>67.6±5.4</td>
<td>69.3±5.1&quot;</td>
<td>74.3±5.2&quot;</td>
<td>79.3±5.8&quot;</td>
<td>82.3±5.9&quot;</td>
</tr>
<tr>
<td>Group D</td>
<td>78.2±8.6</td>
<td>74.1±8.4</td>
<td>70.2±8.1</td>
<td>67.9±7.8</td>
<td>65.3±6.2</td>
<td>66.9±6.6</td>
<td>69.3±6.9</td>
<td>74.6±8</td>
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<tr>
<td>SpO₂ (%)</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
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<tr>
<td>Group P</td>
<td>98±1.2</td>
<td>96.4±1.3&quot;</td>
<td>94.7±1.3&quot;</td>
<td>93.9±1.5&quot;</td>
<td>94.4±1.6&quot;</td>
<td>95.8±1.4&quot;</td>
<td>96.3±1.2&quot;</td>
<td>96.8±1.2&quot;</td>
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<tr>
<td>Group D</td>
<td>98±1.3</td>
<td>97.8±1.3</td>
<td>97.4±1.6</td>
<td>96.9±1.3</td>
<td>96.6±1.3</td>
<td>97.2±1.2</td>
<td>98.1±1</td>
<td>98.4±1</td>
</tr>
<tr>
<td>RR (min⁻¹)</td>
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</tr>
<tr>
<td>Group P</td>
<td>15.8±1.4</td>
<td>13±0.9&quot;</td>
<td>11±0.9&quot;</td>
<td>10±0.8&quot;</td>
<td>10.2±1&quot;</td>
<td>12.2±1&quot;</td>
<td>13.2±1.5&quot;</td>
<td>14.2±1.1</td>
</tr>
<tr>
<td>Group D</td>
<td>16.6±2.1</td>
<td>15.9±2</td>
<td>15.2±2.1</td>
<td>14.7±1.9</td>
<td>14.2±1.8</td>
<td>14.7±1.5</td>
<td>15.5±1.5</td>
<td>15.9±1.6</td>
</tr>
</tbody>
</table>

R, recovery; MAP, Mean Arterial Pressure; HR, Heart Rate; RR, Respiratory Rate. *, P<0.05, when compared with Group D; **, P<0.001, when compared with Group D.
and HR values were found to be significantly lower in Group D (P<0.05, P<0.001, Table 2).

When respiratory data were evaluated, both SpO₂ and RR were lower in Group P at all measurements (P<0.05, P<0.001, Table 2; Figures 2, 3). The lowest SpO₂ value was found as 91% and the respiratory rate as 9; then, oxygen support was needed 1/20 patients in Group P (P>0.05).

Time to achieve an adequate sedation was found to be significantly shorter in Group P (P<0.001, Table 3). Ramsey sedation scores were higher in Group P at the intraoperative 10th minute (P<0.001), while no significant difference was observed between the groups in terms of RSS in the other measurement times (Table 3). Bispectral Index scores were significantly lower in Group P at 5, 10 and 15 minutes (P<0.001). However, no significant difference was found between the groups in terms of the BIS scores at time to admission to the recovery room (P>0.05). BIS values were significantly higher at 10, 20 and 30 minutes in the recovery room in Group P (P<0.001, Table 3).

Aldrete score was found to be lower in Group P when arrived to the recovery room (P<0.001), no significant difference was found at other times of evaluation with respect to aldrete scores (Table 3).

No side effect was observed in both groups such as nausea, vomiting and des-ert mouth.

Discussion

Obstructive Sleep Apnea Syndrome (OSAS) is characterized by the episodes of apnea or hypopnea due to obstruction in the upper airways. If these patients are left untreated, systemic inflammation may develop and cause to several diseases leading to morbidity and mor-
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Surgical therapy is applied as an alternative option in severe OSAS patients and in the patients who cannot tolerate CPAP [3]. Operations which directly target localization of the obstruction are the most successful surgical method in these patients. Nasal endoscopy during the natural sleep is known to be the most ideal diagnostic method to determine localization of the pathology. Drug-Induced Sleep Endoscopy (DISE), has been applied for the first time by Pringle and Craft [13], in which the upper airways are evaluated with fiberoptic nasal endoscopy during sleep produced by sedative drugs [1, 4, 5, 14].

The most suitable option in the endoscopic procedures for both endoscopists and patients is to use a safe sedative agent which will not cause respiratory and cardiac dysfunctions [6]. We preferred propofol which is commonly used in the endoscopic procedures for sedation in patients undergoing DISE and, compared with dexmedetomidine which is recently being considered in the sedoanalgesic protocols [6, 7, 9].

Propofol has been used in different doses for sedation [6, 7, 9, 10, 12, 16, 17]. In some studies, sleep could not be produced at the desired level with bolus administration of propofol 1 mg/kg [15], and 2 mg/kg [20]. Moreover, the authors reported desaturation with inadequate sedation [15, 20]. We administered propofol by infusion; loading dose followed by maintenance, instead of bolus injection. Whereas, in a sedoanalgesia protocol performed in patients undergoing vitreoretinal surgery; when propofol was administered by iv infusion with 0.7 mg/kg loading and 0.5-2 mg/kg/h maintenance, respiratory rate and oxygen saturation have been found decreased with these doses [16]. Kaygusuz et al. [9] reported increase in the respiratory rate and decrease in oxygen saturation with propofol compared to dexmedetomidine. The authors had used loading dose of propofol as 6 mg/kg/h given for 10 minutes and maintenance dose was planned as 2.4 mg/kg/h [10]. In another study by Prachanpanich et al. [17] propofol was loaded with a dose of 1 mg/kg for 10 minutes followed by maintenance dose of 3 mg/kg/h; respiratory rate was found to be lower and need for oxygen support was found to be higher in the propofol group. In our study, we combined propofol with midazolam 0.05 mg/kg and defined the loading dose of propofol as 6 mg/kg/h given for 10 minutes and maintenance dose was planned as 2.4 mg/kg/h [16]. In another study by Prachanpanich et al. [17] propofol was loaded with a dose of 1 mg/kg for 10 minutes followed by maintenance dose of 3 mg/kg/h; respiratory rate was found to be lower and need for oxygen support was found to be higher in the propofol group. In our study, we combined propofol with midazolam 0.05 mg/kg and defined the loading dose of propofol as 0.7 mg/kg for 10 minutes with the controlled infusion technique and, the maintenance dose of propofol was defined as 0.5 mg/kg/h. Both SpO₂ and respiratory rates were lower in patients in propofol group compared to the patients administered dexmedetomidine. Although we did not observe severe desaturation in our patients, one of the patients in propofol group needed oxygen support. We also found that BIS values decreased and sedation score increased more rapidly in the propofol group; thus time to achieve adequate sedation was shorter in this group.

<table>
<thead>
<tr>
<th>Table 3. RSS: 4T, BIS, Ramsay and Aldrete’s scores</th>
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<tbody>
<tr>
<td><strong>RSS: 4T (min)</strong></td>
</tr>
<tr>
<td>Group P</td>
</tr>
<tr>
<td>Periods (min)</td>
</tr>
<tr>
<td>RSS (min-max/med)</td>
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<tr>
<td>Group P</td>
</tr>
<tr>
<td>Group D</td>
</tr>
<tr>
<td>BIS (ort± SD)</td>
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<tr>
<td>Group P</td>
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<tr>
<td>Group D</td>
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<tr>
<td>ALD Sc (min-max/med)</td>
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<tr>
<td>Group P</td>
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<tr>
<td>Group D</td>
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</tbody>
</table>

R, recovery; RSS 4T, Ramsay Sedation Score: 4 Time; BIS, Bispectral Index Score; RSS, Ramsay Sedation Score; ALD Sc, Aldrete score; *, P<0.05; **, P<0.001, when compared with Group D.
Sedoanalgiesic agents for drug induced sleep endoscopy

Dexmedetomidine is a high selective α-2 adreneric receptor agonist and as a result of the stimulation of these receptors, central sympathethic activity decreases and, sedative and anxiolytic effects reveal [8, 11]. This drug is known to show adrenergic effect with rapid loading. Therefore, loading dose is recommended to be administered slowly at least 10 minutes for the sympatholytic effect [6]. We applied the loading dose for 10 minutes and did not cause occurrence of sympathetic activity. Although dexmedetomidine temporarily increases the blood pressure and heart rate in the beginning, this effect is replaced by drops in the blood pressure and heart rate [8, 11]. Some of the sedoanalgesia studies comparing dexmedetomidine and propofol reported that both drugs decrease MAP and HR with no significant differences observed between them [7, 9, 16]. It was demonstrated in some studies that, despite using widely, propofol caused bradycardia and hypotension; and dexmedetomidine had cardioprotective effects [10, 25]. On the other hand, several studies in the literature demonstrated that dexmedetomidine is more associated with hemodynamic instability than propofol [6, 26]. In our study, although propofol and dexmedetomidine were similar in terms of the haemodynamic parameters in the intraoperative period, MAP and HR values were lower in dexmedetomidine group in the recovery room after the drugs were discontinued (P<0.05, P<0.001). In addition, MAP and HR values raised in propofol group compared to the basal values at the 30 minute of recovery, neither MAP nor HR reached to basal values in the dexmedetomidine group. But this condition did not cause delayed recovery.

In a review on pediatric patients by Lin et al [18], use of dexmedetomidine in DISE was found to induce a natural sleep-like sedative response and thus enables a more accurate diagnosis of the localization and degree of the obstruction. Loading dose of dexmedetomidine has been applied as 1 mcg/kg for 10 minutes iv; maintenance dose used to be initiated with 0.2-0.4 mcg/kg/h and titrated with 0.1 mcg/kg/h in every five minutes [6, 8, 9, 14, 19]. Similarly, we applied the loading dose as 1 mcg/kg/h for 10 minutes and the maintenance dose as 0.3 mcg/kg/h.

Combined use of propofol, benzodiazepine, opioids or dexmedetomidine provides more advantages due to the synergistic effect compared to the using of these agents alone [7, 9]. Muller et al. [26] has found that dexmedetomidine alone was not as effective as propofol combined with fentanyl for providing conscious sedation. We administered iv midazolam prior to loading of the study drugs in this study. We aimed to take advantage of the synergistic effect by adding midazolam to propofol and dexmedetomidine. We found that BIS values decreased and sedation score increased more rapidly in the propofol group; thus time to achieve adequate sedation was shorter in the propofol group. On the other hand, in dexmedetomidine group, we observed adequate sedation as well as respiratory stability.

Gross et al. [22] demonstrated that, there is a tendency to decrease in physiological responses against hypoxia and hypercapnia in OSAS and, susceptibility increases against anesthetic agents which have effects causing respiratory depression. Consistent with data from our study, the sedoanalgesia studies comparing propofol and dexmedetomidine have found higher respiratory rate and oxygen saturation in the blood in the dexmedetomidine group and they explained that propofol might cause hypotonia and depress breathing due to its muscle relaxant effects. They reported that dexmedetomidine provided a better respiratory stability and was more attractive drug for anaesthetist due to its the wide margin of safety [8-10, 16, 17, 23, 24]. Our results support these studies; in spite of we used low dose propofol with controlled infusion instead of bolus injection, peripheral oxygen saturation and respiratory rates were higher in dexmedetomidine group than propofol group. In addition, the lowest SpO₂ value was recorded in propofol group during the procedure and oxygen support was needed.

BIS is a widely used method in sleep studies and the scores between 75-90 shows light sleep and the scores between 20 and 75 indicate to a deep sleep wave [27-29]. We found that BIS values higher and RSS was lower in dexmedetomidine group during the sedation.

In one study, use of propofol in DISE was criticized and the authors stated that this drug caused more hypotonia and muscle relaxation, lead to deeper sleep and might cause incorrect evaluations about the obstruction [8]. It was demonstrated in some studies that, patients
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sedated with dexmedetomidine are more cooperative than those sedated with propofol and that, dexmedetomidine provided a faster recovery period and earlier returning to the consciousness level [7, 10, 12]. In another study, time of stay in the recovery room was found similar with dexmedetomidine and propofol [16]. This was supported by our results; in this study Aldrete score at time of the admission to the recovery room were higher in dexmedetomidine group. In contrast, it was reported that, time of stay in the recovery room was longer in patients who received dexmedetomidine, thus it was concluded that the use of this drug as a sedoanalgesic agent should be limited [6, 7, 19, 26, 30].

In a study comparing propofol, benzodiazepines, opioids and dexmedetomidine for sedation patients and surgeons satisfaction were provided in all the drug groups [7]. Whereas, in another sedoanalgesia study comparing dexmedetomidine and propofol, surgeons satisfaction was similar in both groups, while patients satisfaction was higher in the dexmedetomidine group. The authors attributed this to dexmedetomidine provided a natural sleep-like pathway [16]. In our study, surgeons and patients satisfaction was found similar in propofol and dexmedetomidine groups.

In conclusion, our results suggest that, propofol provided rapid and deeper sedation and haemodynamic stability, and dexmedetomidine provided respiratory stability as well as adequate sedation for DISE. As patients with OSAS have higher risk to develope hypoxia and hypercapnia we suggest that, dexmedetomidine has advantages to propofol for sedation in patients with OSAS undergoing DISE.

Disclosure of conflict of interest

None.

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