MEDICAL SCIENCE MONITOR

CLINICAL RESEARCH

e-ISSN 1643-3750
© Med Sci Monit, 2015; 21: 2125-2133
DOI: 10.12659/MSM.893884

Received: 2015.02.16
Accepted: 2015.03.16
Published: 2015.07.22

Comparison of Dexmedetomidine versus Propofol for Sedation after Uvulopalatopharyngoplasty

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEFG Jihong Xu
ABCF Chunji Jin
BCDE Xiaopeng Cui
ADF Zhou Jin

Department of Anesthesiology, General Hospital of Shenyang Military Region, Shenyang, Liaoning, P.R. China

Corresponding Author: Jihong Xu, e-mail: jihongxu@126.com
Source of support: This study was funded by the Liaoning Provincial Natural Science Foundation of China (2013020168)

Background: Adequate sedation is important in the post-anesthesia care unit (PACU) following uvulopalatopharyngoplasty (UPPP) to ensure patient comfort and decrease the duration of mechanical ventilation (MV), PACU stay, and bleeding. This study aimed to compare dexmedetomidine and propofol as sedatives after UPPP in the PACU.

Material/Methods: We randomized 124 mechanically ventilated adults following UPPP who were managed in the PACU of the General Hospital of the Shenyang Military Region between January 2014 and June 2014, to receive either dexmedetomidine or propofol. The patients in the propofol group received an infusion of propofol (3 mg/kg/h) titrated up to 6 mg/kg/h to attain a Ramsay sedation score ≥4. The dexmedetomidine group patients received 1.0 μg/kg of dexmedetomidine over a period of 10 minutes and then 0.5 to 1.0 μg/kg/h infusion to maintain a Ramsay sedation score ≥4.

Results: Bispectral index (BIS) values were significantly lower in the dexmedetomidine group than in the propofol group at Ramsay sedation scores of 4 and 5. The mean times to spontaneous breathing, waking, and extubation were shorter in the dexmedetomidine group. Tramadol requirement was significantly reduced in the dexmedetomidine group ($P<0.05$). Incidence of cough during the extubation process in the propofol group was higher than in the dexmedetomidine group. After extubation, Bruggemann comfort scale (BCS) and Rass agitation scores (RASS) were decreased in the dexmedetomidine-sedated patients.

Conclusions: Dexmedetomidine provides safe and effective sedation for post-UPPP surgical patients and significantly reduces the use of analgesics, with minimal adverse effects.

MeSH Keywords: Deep Sedation • Propofol • Dexmedetomidine

Full-text PDF: http://www.medscimonit.com/abstract/index/idArt/893884

2669 5 2 36

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Background

Obstructive sleep apnea syndrome (OSAS) is a highly prevalent disorder affecting about 4% of adults, and is associated with repetitive episodes of transient oxygen desaturation during sleep [1]. OSAS is an independent risk factor for a number of cardiovascular diseases [2–4]. Uvulopalatopharyngoplasty (UPPP) is a routine procedure for OSAS [5]. Although UPPP expands pharyngeal cavity and improves upper respiratory tract obstruction, it is often complicated by edema, strictures, bleeding, increased hypopharyngeal secretions, and decreased pharyngeal airway-protective reflexes [6]. Therefore, patients are prone to have airway re-obstruction and apnea during recovery from anesthesia.

Adequate sedation is important in the post-anesthesia care unit (PACU) following UPPP to ensure patient comfort and decrease the duration of mechanical ventilation (MV), PACU stay, and bleeding [7]. The anesthetic propofol is commonly used in the ICU for sedation of the ventilated postsurgical patient [8]. However, propofol sedation may also cause respiratory depression, which may be accentuated by the concurrent use of opioids [9,10].

Dexmedetomidine is a highly specific alpha-2-adrenergic receptor agonist that possesses sedative, anxiolytic, and analgesic effects. At clinically effective doses, continuous sedation with intravenous dexmedetomidine does not interfere with the normal course of ventilator weaning and extubation because it does not depress respiratory drive or decrease arterial oxygen saturation [11,12]. These characteristics may facilitate extubation after UPPP. The aim of this study was to retrospectively compare the effects of dexmedetomidine versus propofol as sedatives following UPPP in the PACU.

Material and Methods

Subjects

After approval of the ethics committee of the General Hospital of Shenyang Military Region (China), a total of 150 patients undergoing UPPP and recovering from general anesthesia were studied. All subjects were diagnosed with OSAS based on symptoms such as heavy and loud snoring, witnessed apneas, and/or daytime sleepiness and choking during sleep. Each subject underwent clinical assessment, testing for complete blood count, liver and kidney function, and cardiac enzymes. Patients also completed the Epworth sleepiness scale (ESS). Overnight polysomnography (PSG) was performed. The patients were considered ineligible if they had unstable angina or acute myocardial infarction in the last 30 days, uncontrolled diabetes, morbid obesity (BMI >40), ejection fraction below 30%, or were treated with neuromuscular blocking agents. Patients were also excluded if they had a history of alcohol or drug abuse or their neurologic condition was difficult to evaluate.

Study design

Our study included 124 subjects classified into 2 groups. Patients in the propofol group (n=61) received an infusion of propofol (3 mg/kg/hr), which was titrated up to a maximum of 6 mg/kg/h until they were adequately sedated (Ramsay sedation score ≥4). Patients in the dexmedetomidine group (n=63) received 1.0 μg/kg of dexmedetomidine over a 10-min period and then 0.5 to 1.0 μg/kg/h infusion to maintain a Ramsay sedation score ≥4. If a dexmedetomidine-sedated patient could not be maintained within the desired sedation range and the infusion rate was already at the recommended maximum of 1.0 μg/kg/h, the patient received intravenous injection of propofol (3 mg/kg/hr) until Ramsay sedation score was at least ≥4.

Tramadol (1 mg/kg) was allowed for pain relief in both groups. Staff determined the need for tramadol depending upon the signs of pain [e.g., sweating, increased blood pressure, and elevated heart rate (HR)] before extubation or when the score was no more than 2 on the Bruggemann comfort scale (BCS) assessed 10 min after extubation by direct communication with the patient.

Intraoperative anesthetics, narcotics, and other medications were standardized by the Department of Anesthesiology by induction with propofol 2 mg/kg in combination with midazolam 2 mg, sufentanil 0.5 μg/kg, and rocuronium bromide 0.8 mg/kg. The video laryngoscopes were used for intubation. Anesthesia was maintained with propofol (3–8 mg/kg/h) and remifentanil (3–15 μg/kg/h) administered with an infusion pump, and 0.5–2.5% sevoflurane administered by vaporization. Depth of anesthesia was adjusted to maintain BIS values within the 40–60 range. Blood pressure and heart rate (HR) were maintained within 20% of awake values. Rocuronium bromide was administered intravenously as required. Propofol, remifentanil, and sevoflurane were stopped when the patients were transferred to the PACU after the operation.

In the PACU, mechanical ventilation was stopped when end-tidal (ET) anaesthetic concentration dropped to 0.2%. Respiration was then manually assisted until recovery of spontaneous breathing (tidal volume >6 mL/kg). Each patient was given 5 L/min of oxygen via oxygen insufflation after recovery of adequate spontaneous ventilation, followed by 2 L/min oxygen through a nasal cannula after extubation. Sedative infusion was continued for 3 h, and then patients were extubated. A patient was considered ready for extubation if awake or arousable, cooperative and comfortable, and if fraction of inspiration O_2 (FiO_2) was less than 0.4 and blood oxygen saturation

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

(SpO2) exceeded 96%. In addition, the partial pressure of O_2 in arterial blood (PaO_2) should be over 80 mmHg, partial pressure of carbon dioxide in the blood ($PaCO_2$) below 50 mmHg, tidal volume greater than 6 mL/kg, spontaneous respiratory rate lower than 25/min, with steady circulatory function and no hemorrhagic secretions observed in the upper respiratory tract. Methylprednisolone (40 mg) was administered intravenously before extubation. The patients were extubated in the lateral position. Patients with a modified Aldrete score between 9 and 10 were transferred to the ward.

The electrocardiogram, HR, noninvasive systolic and diastolic arterial blood pressures, SpO_2, respiratory rate, BIS (BIS XP 3.4 monitor, Aspect Medical Systems, Newton, MA) were recorded preoperatively (T0), when sedation was initiated (T1), and at the following timepoints: 30 min after dexmedetomidine or propofol initiation (T2), extubation (T3), 10 min after extubation (T4), and at discharge from the PACU (T5).

Other recorded variables included:

1. The number of cases receiving tramadol.
2. Time to recovery of spontaneous breathing.
3. Time to extubation: defined as the time interval between stopping dexmedetomidine or propofol and the fulfillment of extubation criteria.
4. Time to discharge from PACU.
5. Ramsay sedation scores [13] were assessed at T2 and Rass agitation scores (RASS) [14] were assessed at T3. At T3, if RASS score was at least 3 points, the patients were treated with analgesics (tramadol) and sedatives (dexmedetomidine or/and propofol with an infusion pump). Sedatives were continued for 30 min and then discontinued to awaken the patients. The RASS score was assessed for the second time. If RASS score was at least 3, the sedation was continued as before until RASS score was 2 or less. If RASS score was 2 or less, sedatives were discontinued and the patients regained consciousness gradually.
6. Cough responses during extubation were scored as 1=no incidence of cough; 2=smooth extubation, slight coughing (1~2 coughs); 3=moderate coughing (3~4 coughs); 4=severe or repetitive coughing (5~10 coughs); 5=patient discomfort, poor extubation (>10 severe coughs) [15].
7. BCS were assessed at T4 (10 min after extubation). BCS: 0=continuous pain; 1=no pain without movement, but serious pain when breathing deeply or coughing; 2=no pain without movement, but mild pain when breathing deeply or coughing; 3=no pain, even when breathing deeply; and 4=no pain when coughing) [16].
8. Potential adverse drug reactions were noted during PACU: bradycardia (HR <50 bpm), tachycardia (HR >100 bpm), hypotension [mean arterial pressure (MAP) was less than 30% of the baseline], hypertension (MAP was more than 30% of the baseline), respiratory depression (respiratory rate ≤8 bpm or SpO2 ≤90% for a duration exceeding 5 min), glossoptosis, nausea or vomiting, rigors, and bleeding.

Statistical analysis

Statistical analysis was performed using a commercial software package (SPSS 17.0, Chicago, IL). This sample size for the study was based on tramadol consumption in the PACU after UPPP surgery, assuming a requirement of rescue tramadol 0.72 (SD0.23) mg/kg with dexmedetomidine-ketamine-based anesthesia [17]. To detect a 30% reduction in tramadol requirements in the PACU after surgery, 60 subjects per treatment group would be needed for a study with an alpha level of 0.05 and a beta level of 0.2 (80% power). Sample size was increased by 15% to account for the miss rate.

Continuous variables with a normal distribution are reported as mean (standard deviation, SD). Categorical variables are expressed as numbers and percentages. Changes in hemodynamic variables were tested with repeated-measures ANOVA. The LSD method was used for multiple comparisons. Independent-samples t-test was performed on all other quantitative variables obtained. Differences in the incidence of adverse events were analyzed by chi-square test. A P-value less than 0.05 was considered statistically significant.

Results

Sixty-one patients in the propofol group and 63 patients in the dexmedetomidine group were analyzed (Figure 1). The baseline characteristics of the study population are shown in Table 1. Operative time was about 40 min, and no additional muscle relaxants were administered. The 2 groups were similar in age, BMI, apnea-hypopnea index (AHI), neck size, and ESS score. There were no significant differences in anesthesia time and operative time between the 2 groups. All subjects had daytime awake oxygen saturation exceeding 92% (Table 1).

Figure 2 shows the HR, noninvasive systolic and diastolic arterial blood pressures, SpO_2, $ETCO_2$, and BIS at each point of time. In the propofol group, HR, systolic blood pressure (SBP), and diastolic blood pressure (DBP) significantly increased at the time of extubation (T3) in contrast to dexmedetomidine-sedated patients who manifested non-significant changes in SBP and DBP ($P>0.05$). Dexmedetomidine sedation decreased the HR significantly after its initiation and was significantly less than in the propofol group. Oxygen saturation decreased and $ETCO_2$ increased at 30 min after propofol initiation. The SPO_2 and $ETCO_2$ were not changed in the dexmedetomidine group (both $P<0.05$, in comparison with the propofol group). BIS values were significantly lower in the dexmedetomidine group than in the propofol group at Ramsay sedation scores of 4 and 5 ($P=0.045$).

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

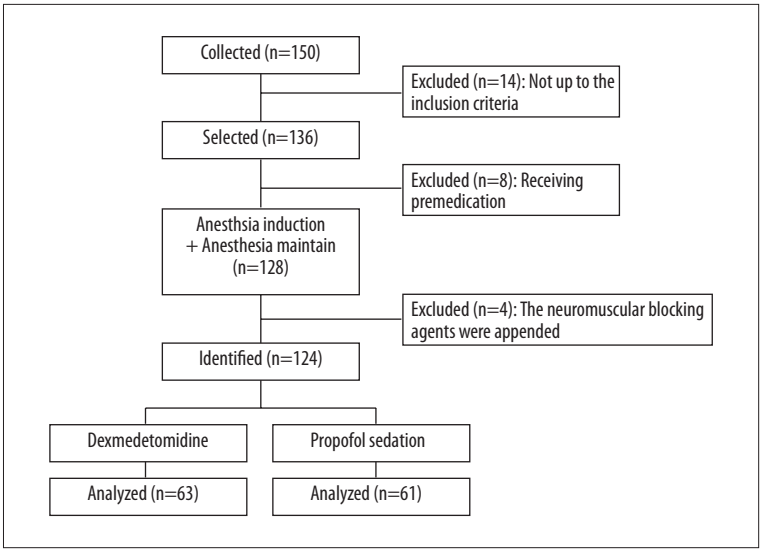

Figure 1. Flow diagram of the trial.

Table 1. Baseline characteristics of the patients receiving dexmedetomidine or propofol sedation.Values are mean (SD)or number (percent). There were no significant differences between groups.

Characteristic	Dexmedetomidine group (n=63)		Propofol group (n=61)		P value
Age, yrs	45.2	(12.0)	45.6	(11.2)	0.537
Male sex (%)	54.0	(85.7)	53.0	(86.9)	0.826
BMI, kg/m^2	32.0	(6.5)	31.4	(7.3)	0.342
AHI	45.1	(10.6)	46.3	(8.0)	0.781
Neck size, cm	44.2	(2.9)	45.8	(2.2)	0.652
ESS	15.0	(3.0)	14.0	(5.0)	0.302
Basal SpO_2, %	94.6	(2.6)	95.0	(3.1)	0.511
Min SpO_2, %	67.2	(10.6)	65.4	(12.7)	0.124
The largest loudness of Snore, dB	74.2	(13.2)	76.8	(10.7)	0.220
Hemoglobin, g/L	148.9	(15.3)	150.2	(12.9)	0.319
Duration of anesthesia, min	66.4	(17.1)	68.5	(18.3)	0.083
Duration of surgery, min	45.7	(15.6)	43.4	(15.2)	0.462

BMI – body mass index; AHI – apnea-hyponea index; ESS – Epworth sleepiness scale.

There were no significant differences in Ramsay sedation scores between groups during assisted ventilation (T2) (P=0.259). After extubation, BCS and RASS in the dexmedetomidine group were lower than in the propofol group (P=0.024 and 0.042, respectively). During assisted ventilation, the dexmedetomidine-sedated patients required less tramadol than patients in the propofol group (P=0.001) (Table 2). Coughing during extubation occurred more frequently and was more severe in the propofol group compared to the dexmedetomidine group (P<0.001) (Table 3).

The time to spontaneous breathing in the dexmedetomidine group was significantly shorter than in the propofol group (P=0.035). Patients fulfilled the criteria of extubation earlier in the dexmedetomidine group (P=0.028) (Table 4).

No patient was reintubated after extubation. None of the patients experienced postoperative bleeding that required any intervention. There were fewer dexmedetomidine-sedated patients requiring treatment for emergent adverse reactions than in the propofol group (P=0.002). There were no differences

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

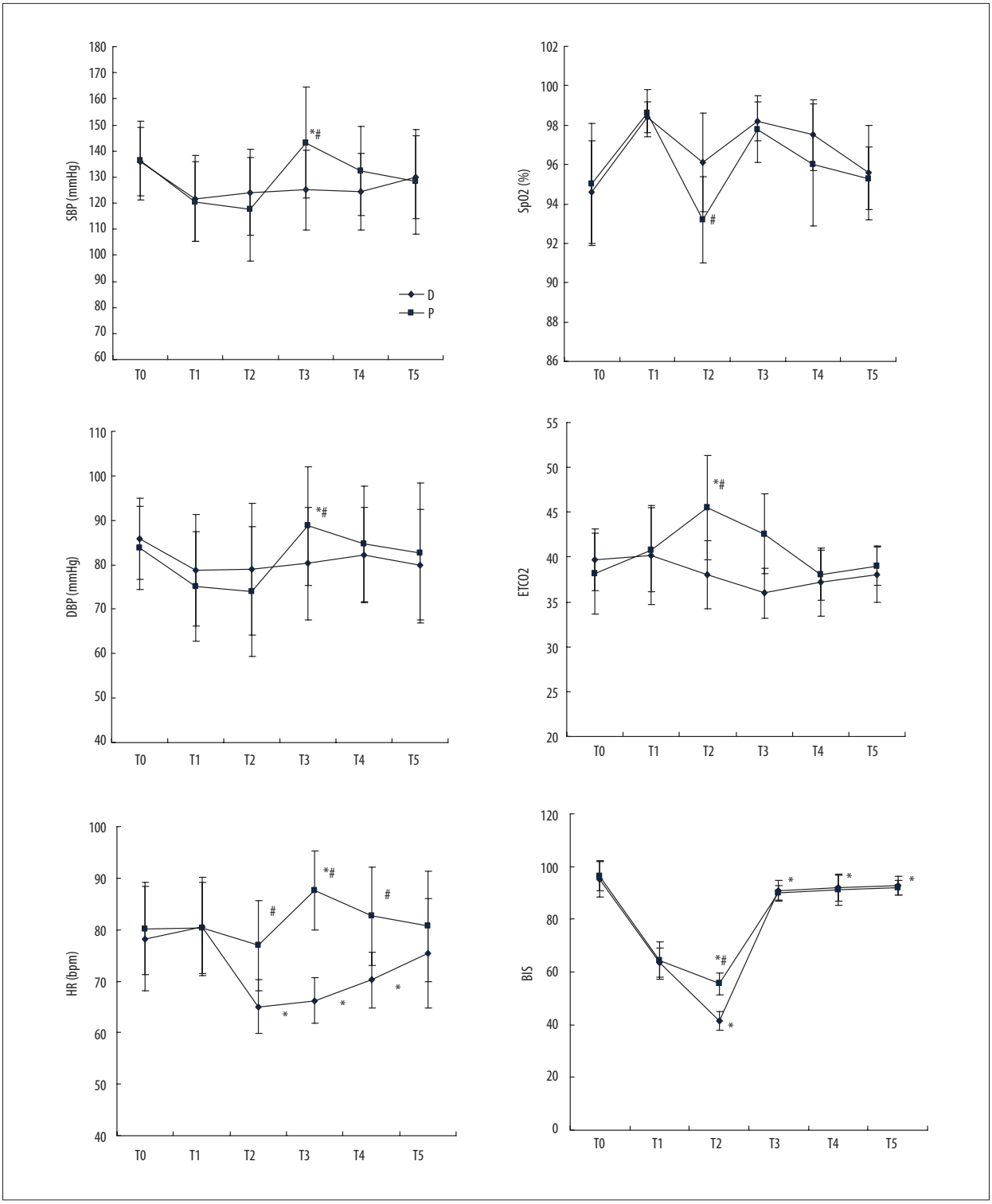

Figure 2. Changes of vital signs (noninvasive arterial blood pressure, HR, SpO_2, $ETCO_2$, and BIS) of the 2 groups in the PACU. SBP – systolic blood pressure, DBP – diastolic blood pressure, HR – heart rate, $ETCO_2$ – end-tidal CO_2, SpO_2, BIS – bispectral index, which was obtained for each timepoint (T0: preoperative, T1: initiation of sedative infusion, T2: 30 min after sedative infusion, T3: during extubation, T4: 10 min after extubation and T5: at discharge from PACU). Data are shown with mean ±SD. # P<0.05 for the difference between the 2 groups at the same timepoint. * P<0.05, when compared with T1 within the same group.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Table 2. Sedation, agitation scores in PACU and number of patients receiving tramadol. Values are mean (SD) or number (percent). *A P-value less than 0.05 was considered statistically significant.

Characteristics	Dexmedetomidine group (n=63)		Propofol group (n=61)		P value
Ramsay sedation scores	4.57	(0.62)	4.74	(0.52)	0.259
RASS	0.8	(0.7)	1.5	(1.2)	0.042*
BCS	2.53	(1.32)	1.46	(0.76)	0.024*
Number of patients receiving tramadol (%)	35.0	(55.6)	52.0	(85.2)	0.001*

RASS – Rass agitation scores; BCS – Bruggemann comfort scale.

Table 3. Coughing incidence during extubation in the patients receiving dexmedetomidine or propofol sedation. Values are number(percent). *A P-value less than 0.05 was considered statistically significant.

	Total cough number	Occasional cough number (1–2 times)	Regular cough number (3–4 times)	Frequent cough (5–10 times)	Choking or severe cough (more than 10 times)
Dexmedetomidine	27 (42.9)	16 (25.4)	10 (15.9)	1 (1.6)	0
Propofol	43 (70.4)	15 (24.6)	23 (37.7)	5 (8.2)	0
χ^2	11.824	0.982	15.137	3.941	
P	<0.001*	0.322	<0.001*	0.047*	

Table 4. Time to spontaneous breathing, extubation and PACU stay in patients receiving dexmedetomidine or propofol. Values are mean (SD). *A P-value less than 0.05 was considered statistically significant.

Time	Dexmedetomidine group (n=63)		Propofol group (n=61)		P value
Time to spontaneous breathing (min)	6.5	(2.1)	10.5	(5.6)	0.035*
Time to extubation (min)	8.5	(4.0)	15.1	(8.3)	0.028*
Length of PACU stay (min)	235.6	(15.7)	242.5	(17.5)	0.124

between the 2 groups in the length of stay in the PACU. Four categories – hypertension, tachycardia, bradycardia and respiratory depression – were significantly different between groups. Bradycardia occurred significantly more frequently in the dexmedetomidine group (P=0.002). Hypertension, tachycardia and respiratory depression occurred significantly more frequently in the propofol group (P=0.038 and 0.027, respectively, Table 5).

Discussion

Dexmedetomidine, a a2-adrenoreceptor agonist well known for its anti-anxiety, sedative, analgesic, anaesthetic-sparing and respiratory-sparing effects, is also a perfect candidate for premedication [18]. In the PACU of our department, we administrated dexmedetomidine to patients after UPPP for sedation. In our study, analgesic consumption, pain intensities, sedation and agitation scores, cardiovascular and respiratory variables, and adverse reactions such as nausea, vomiting, and rigors were compared for 4 hours after operation. The results show that dexmedetomidine-based sedation was safe and effective for postsurgical UPPP patients.

Our results also showed that at comparable Ramsay scores, BIS values were lower with dexmedetomidine sedation than with propofol. The results are similar to those of Recart et al. [19]. Dexmedetomidine sedation differs from that of propofol-induced sedation [20]. Patients who receive dexmedetomidine are quite comfortable and are still arousable and responsive to

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Table 5. Adverse reactions during recovery in the patients receiving dexmedetomidine or propofol. Values are number (percent). *A P-value less than 0.05 was considered statistically significant.

	Dexmedetomidine group (n=63)		Propofol group (n=61)		χ^2	P value
At least one adverse event	35	(55.6)	50	(82.0)	10.028	0.002*
Hypotension	3	(4.7)	4	(6.6)	0.188	0.665
Hypertension	5	(7.9)	13	(19.7)	4.468	0.035*
Tachycardia	5	(7.9)	12	(19.6)	4.290	0.038*
Bradycardia	13	(20.6)	2	(3.3)	9.177	0.002*
Respiratory depression	4	(6.3)	12	(19.6)	4.895	0.027*
Rigors	0		1	(1.6)	1.041	0.308
Nausea or vomiting	2	(3.2)	2	(3.3)	0.201	0.654
Glossoptosis	3	(4.8)	4	(6.6)	0.188	0.665

stimuli. Arain et al. [21] evaluated the intraoperative sedative effects of dexmedetomidine and propofol, and demonstrated that although sedation with dexmedetomidine was achieved slowly, similar effects were found between groups 25 min after initiation of infusion.

Pain was the main cause of agitation in patients recovering from UPPP, reflected by frequent complaints of throat pain and discomfort after extubation [22]. A previous study showed that visual analogue scale (VAS) was 4~6 points in a quiet condition after UPPP [23]. Prompt and effective management of pain is one of the most effective measures to prevent and treat postoperative complications following UPPP. Opioid analgesics are commonly used to manage postoperative pain, but in large doses they may cause respiratory depression. In our study, dexmedetomidine-sedated patients required significantly less tramadol and had decreased agitation during extubation. This analgesic-sparing effect of dexmedetomidine is well documented in previous studies [24–26].

We compared the UPPP patients receiving dexmedetomidine or propofol during recovery from extubation in the PACU. Under similar levels of sedation, dexmedetomidine efficiently inhibited the stress response around tracheal extubation, whereas propofol increased blood pressure and HR. Dexmedetomidine initiation was associated with decreased HR and transient hypertension, which is consistent with other reports in the literature. No dose reduction or discontinuation of dexmedetomidine has been reported in any patient [27,28].

The effect of dexmedetomidine in reducing the incidence and severity of cough during extubation is consistent with the study of Aksu et al. [29], in which injection of 0.5 μg/kg dexmedetomidine before extubation effectively alleviated the airway responses and suppressed the cough response and hemodynamic fluctuations. In addition, the treatment did not prolong patient recovery time. Dexmedetomidine also prevented the occurrence of nausea, vomiting, and chills after extubation [30]. In this study, the incidence of nausea, vomiting and chills was less in patients receiving dexmedetomidine. Glossoptosis is the most common respiratory complication after extubation. None of the 124 patients in this study had glossoptosis, and the influence of dexmedetomidine and propofol on glossoptosis was similar. The time to extubation and the time to recovery of spontaneous breathing were significantly shorter in the dexmedetomidine group compared with the propofol group. Patients in the dexmedetomidine group were easy to wake and recovered consciousness more quickly and completely. These results suggest that the extubation quality was better in the dexmedetomidine group than in the propofol group.

Respiratory depression during the UPPP recovery period is commonly caused by the residual effects of anesthetics such as opioids. Assisted ventilation or ventilation control should be performed until the recovery of spontaneous breathing, and antagonist must be used with caution to prevent agitation [31]. Similar to our results, Goyagi et al. [32] reported that dexmedetomidine did not significantly prolong the recovery time of spontaneous breathing and the eye-opening time compared with propofol. Indeed, several other studies with dexmedetomidine support observations that dexmedetomidine used at clinical doses did not depress respiratory drive [33–35]. However, some patients may have respiratory depression due to the interaction between dexmedetomidine and residual anesthetics and muscle relaxants. Itagaki et al. [35] reported that geriatric

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

patients presented with respiratory depression 90 min after extubation following infusion of 0.26 μg kg h dexmedetomidine for 3.5 h, indicating that higher doses or prolonged administration of dexmedetomidine caused respiratory depression. In this study, respiratory depression occurred in 12 cases after drug withdrawal and before extubation in the propofol group, and in 4 cases after extubation in the dexmedetomidine group. These data suggest that inappropriate application of dexmedetomidine in patients of different ages resulted in respiratory depression [36].

Agitation scales were monitored occasionally by an assessor who rated the level of agitation on the basis of a single observation and interaction with the patient. Discrete observations may fail to account for changes in sedation level that may occur between assessments. Plasma drug concentrations were not measured, and therefore, cannot be assumed to have remained constant or similar in patients.

Conclusions

This study provides strong evidence supporting postoperative sedation by dexmedetomidine in patients following UPPP. This drug can induce suitable depth of sedation and the analgesic effect, significantly reduce the use of analgesics without clinically significant respiratory depression and agitation during extubation, and shorten the time to extubation. Thus, the better extubation quality provided by dexmedetomidine shows promising postoperative sedation effects.

Acknowledgments

We thank the talented and supportive members of the ENT Department (ear, nose, and throat) Surgery, Department of Anesthesiology, and the nursing staff of the Intensive Care Unit.

Conflict of interest

We declare that we have no conflict of interest.

References:

1. Young T, Palta M, Dempsey J et al: The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med, 1993; 328: 1230–35
2. Gunnarsson SI, Peppard PE, Korcarz CE et al: Obstructive sleep apnea is associated with future subclinical carotid artery disease: Thirteen-year follow-up from the wisconsin sleep cohort. Arterioscler Thromb Vasc Biol, 2014; 34: 2338–42
3. McMillan A, Bratton DJ, Faria R et al: Continuous positive airway pressure in older people with obstructive sleep apnoea syndrome (predict): A 12-month, multicentre, randomised trial. Lancet Respir Med, 2014; 2: 804–12
4. Cepeda-Valery B, Acharjee S, Romero-Corral A et al: Obstructive sleep apnea and acute coronary syndromes: Etiology, risk, and management. Curr Cardiol Rep, 2014; 16: 535
5. Braga A, Grechi TH, Eckeli A et al: Predictors of uvulopalatopharyngoplasty success in the treatment of obstructive sleep apnea syndrome. Sleep Med, 2013; 14: 1266–71
6. Kandasamy T, Wright ED, Fuller J, Rotenberg BW: The incidence of early postoperative complications following uvulopalatopharyngoplasty: Identification of predictive risk factors.J Otolaryngol Head Neck Surg, 2013; 42: 15
7. Abdelmageed WM, Elquesny KM, Shabana RI et al: Analgesic properties of a dexmedetomidine infusion after uvulopalatopharyngoplasty in patients with obstructive sleep apnea. Saudi J Anaesth, 2011; 5: 150–56
8. Lonardo NW, Mone MC, Nirula R et al: Propofol is associated with favorable outcomes compared with benzodiazepines in ventilated intensive care unit patients. Am J Respir Crit Care Med, 2014; 189: 1383–94
9. Chen KZ, Ye M, Hu CB, Shen X: Dexmedetomidine *vs.* remifentanil intravenous anaesthesia and spontaneous ventilation for airway foreign body removal in children. Br J Anaesth, 2014; 112: 892–97
10. Chen L, Wang M, Xiang H et al: Prediction of effect-site concentration of sufentanil by dose-response target controlled infusion of sufentanil and propofol for analgesic and sedation maintenance in burn dressing changes. Burns, 2014; 40: 455–59
11. Belleville JP, Ward DS, Bloor BC, Maze M: Effects of intravenous dexmedetomidine in humans. I. Sedation, ventilation, and metabolic rate. Anesthesiology, 1992; 77: 1125–33
12. Venn RM, Hell J, Grounds RM: Respiratory effects of dexmedetomidine in the surgical patient requiring intensive care. Crit Care, 2000; 4: 302–8
13. Herr DL, Sum-Ping ST, England M: Icu sedation after coronary artery bypass graft surgery: Dexmedetomidine-based versus propofol-based sedation regimens. J Cardioth Vascul Anesthesia, 2003; 17: 576–84
14. Walsh TS, Everingham K, Frame F et al: An evaluation of the validity and potential utility of facial electromyelogram responsiveness index for sedation monitoring in critically ill patients. J Crit Care, 2014; 29: 886 e881–87
15. Nishina K, Mikawa K, Maekawa N, Obara H: Fentanyl attenuates cardiovascular responses to tracheal extubation. Acta Anaesthesiol Scand, 1995; 39: 85–89
16. Tang QF, Qian YN, Qiu YH et al: Effects of preemptive intravenous lornoxicam on the analgesic efficacy of epidural morphine and expression of chemokines in women undergoing hysterectomy. Cur Ther Res Clin Exp, 2009; 70: 1–97
17. Cheng X, Huang Y, Zhao Q, Gu E: Comparison of the effects of dexmedetomidine-ketamine and sevoflurane-sufentanil anesthesia in children with obstructive sleep apnea after uvulopalatopharyngoplasty: An observational study. J Anaesthesiol Clin Pharmaco, 2014; 30: 31–25
18. Sun Y, Liu C, Zhang Y et al: Low-Dose Intramuscular Dexmedetomidine as Premedication: A Randomized Controlled Trial. Med Sci Monit, 2014; 20: 2714–19
19. Recart A, Gasanova I, White PF et al: The effect of cerebral monitoring on recovery after general anesthesia: A comparison of the auditory evoked potential and bispectral index devices with standard clinical practice. Anesth Analg, 2003; 97: 1667–74
20. Turan A, Dalton JE, Kasuya Y et al: Correlation between bispectral index, observational sedation scale, and lower esophageal sphincter pressure in volunteers using dexmedetomidine or propofol. Med Sci Monit, 2012; 18(10): CR593–96
21. Arain SR, Ebert TJ: The efficacy, side effects, and recovery characteristics of dexmedetomidine versus propofol when used for intraoperative sedation. Anesth Analg, 2002; 95: 461–66
22. Davis PJ, Finkel JC, Orr RJ et al: A randomized, double-blinded study of remifentanil versus fentanyl for tonsillectomy and adenoidectomy surgery in pediatric ambulatory surgical patients. Anesth Analg, 2000; 90: 863–71
23. Bell KR, Cyna AM, Lawler KM et al: The effect of glossopharyngeal nerve block on pain after elective adult tonsillectomy and uvulopalatoplasty. Anaesthesia, 1997; 52: 597–602
24. Triltsch AE, Welte M, von Homeyer P et al: Bispectral index-guided sedation with dexmedetomidine in intensive care: A prospective, randomized, double blind, placebo-controlled phase ii study. Crit Care Med, 2002; 30: 1007–14
25. Martin E, Ramsay G, Mantz J, Sum-Ping ST: The role of the alpha2-adrenoceptor agonist dexmedetomidine in postsurgical sedation in the intensive care unit. J Inten Care Med, 2003; 18: 29–41

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

26. Venn RM, Grounds RM: Comparison between dexmedetomidine and propofol for sedation in the intensive care unit: Patient and clinician perceptions. Br J Anaesth, 2001; 87: 684–90
27. Shehabi Y, Ruettimann U, Adamson H et al: Dexmedetomidine infusion for more than 24 hours in critically ill patients: Sedative and cardiovascular effects. Inten Care Med, 2004; 30: 2188–96
28. Maze M, Scarfini C, Cavaliere F: New agents for sedation in the intensive care unit. Crit Care Clin, 2001; 17: 881–97
29. Aksu R, Akin A, Bicer C et al: Comparison of the effects of dexmedetomidine versus fentanyl on airway reflexes and hemodynamic responses to tracheal extubation during rhinoplasty: A double-blind, randomized, controlled study. Curr Ther Res Clin Exp, 2009; 70: 209–20
30. Massad IM, Mohsen WA, Basha AS et al: A balanced anesthesia with dexmedetomidine decreases postoperative nausea and vomiting after laparoscopic surgery. Saudi Med J, 2009; 30: 1537–41
31. Virtaniemi J, Kokki H, Nikanne E, Aho M: Ketoprofen and fentanyl for pain after uvulopalatopharyngoplasty and tonsillectomy. Laryngoscope, 1999; 109: 1950–54
32. Goyagi T, Tanaka M, Nishikawa T: Oral clonidine premedication reduces induction dose and prolongs awakening time from propofol-nitrous oxide anesthesia. Can J Anaesth, 1999; 46: 894–96
33. Kasuya Y, Govinda R, Rauch S et al: The correlation between bispectral index and observational sedation scale in volunteers sedated with dexmedetomidine and propofol. Anesth Analg, 2009; 109: 1811–15
34. Hsu YW, Cortinez LI, Robertson KM et al: Dexmedetomidine pharmacodynamics: Part i: Crossover comparison of the respiratory effects of dexmedetomidine and remifentanil in healthy volunteers. Anesthesiology, 2004; 101: 1066–76
35. Itagaki T, Uchisaki S, Adachi Y et al: apnea and severe respiratory depression induced by dexmedetomidine after general anesthesia in intensive care unit. Masui, 2009; 58: 1534–37
36. Ramsay MA, Luterman DL: Dexmedetomidine as a total intravenous anesthetic agent. Anesthesiology, 2004; 101: 787–90

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]