

Dexmedetomidine in the Supratentorial Craniotomy

Supratentorial Kraniotomilerde Dexmedetomidin' in Yeri

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Abstract

Objective: In this double-blind prospective clinical study, we investigated the effects of fentanyl and dexmedetomidine as adjuvant agents in supratentorial craniotomies on the following: hemodynamic changes during perioperative and recovery periods, brain edema perioperatively, recovery times and side effects, such as hypertension, shivering, nausea and vomiting.

Materials and Methods: Thirty consenting ASA physical status I-II patients undergoing intracranial tumor surgery were randomly divided in two groups. In group D (n=15), dexmedetomidine was infused as a 1 µg/kg bolus dose 10 minutes before induction of anesthesia and maintained with 0.4-0.5 µg/kg/min during the operation. In group F (n=15), animals were given fentanyl 0.02 µg/kg/min as an infusion for anesthesia maintenance. At induction, fentanyl was given as a 2 µg/kg dose in group D and as a 4 µg/kg dose in group F. Hemodynamic changes, recovery times and postoperative side effects were recorded before induction, during the perioperative period and 24 hours postoperatively.

Results: In group D; MAP and HR values after intubation, after skull clamp insertion and after extubation were lower than in group F (p<0.05). In group D, cerebral relaxation scores were also significantly lower. Recovery times were found to be shorter in group D as compared to group F; the same trend was observed for the supplemental opioid requirement. During the postoperative period, there was no shivering, nausea or vomiting in group D, but in group F, 3 patients complained of shivering, and 2 patients experienced nausea and vomiting.

Conclusion: In our study, we found that dexmedetomidine controlled the hemodynamic changes better than fentanyl perioperatively, after extubation and during the early postoperative period. Our results suggest that dexmedetomidine is safer and more effective in controlling hemodynamic changes during surgical stimulation than the standard agents used in neuroanesthesia.

Key Words: Craniotomy, Dexmedetomidine, Hemodynamics, Neurosurgery

Özet

Amaç: Supratentorial kraniotomide, adjuvan olarak kullanılan deksmedetomidin ile fentanilin, peroperatif ve derlenme dönemindeki uyarılara verilen hemodinamik cevaplar ile peroperatif beyin ödeminin önlenmesindeki etkinliklerini, anesteziden derlenme sürelerini ve postoperatif periyottaki hipertansiyon, titreme, bulantı ve kusma gibi yan etkileri önlemedeki etkinliklerini araştırdık.

Gereç ve Yöntem: Elektif intrakranial tümör cerrahisi yapılacak ASA I-II, 30 hasta randomize iki gruba ayrıldı. Grup D: Deksmmedetomidin infüzyonu yapılan, Grup F: Fentanil infüzyonu yapılan grup. Grup D'de indüksiyondan 10 dk önce, 1 µg/kg bolus infüzyon olarak deksmedetomidin verildi. İndüksiyonda grup D'de 4 µg/kg fentanil, grup F'de ise 2 µg/kg fentanil verildi. İndüksiyon sonrasında grup D'de deksmedetomidin 0.4-0.5 µg/kg/sa, grup F'de fentanil 0.02 µg/kg/dk dozlarında idame infüzyon olarak devam edildi. İndüksiyon öncesinde, peroperatif dönemde, postoperatif ilk 24 saatte hemodinamik veriler ile derlenme süreleri ve postoperatif yan etkiler kaydedildi.

Bulgular: Grup D'de entübasyon sonrası, çivili başlık sonrası ve ekstübasyon sonrası OAB ve KAH değerleri istatistiksel olarak anlamlı şekilde düşük bulundu. Grup D'de beyin relaksasyon skorları istatistiksel olarak anlamlı şekilde daha düşük bulundu. Grup D'de anlamlı olarak derlenme süreleri kısa ve ek narkotik ihtiyacı daha azdı. Grup D'de postoperatif dönemde üşüme, titreme, bulantı ve kusma görülmezken, grup F'de 3 hasta da üşüme ve titreme, 2 hasta da ise bulantı-kusma görüldü.

Sonuç: Çalışmamızda, deksmedetomidinin peroperatif, ekstübasyon sonrası ve erken postoperatif dönemdeki hemodinamik cevapları fentanile kıyasla daha iyi kontrol ettiğini saptadık. Deksmmedetomidinin, nörocerrahi hastalarında, cerrahi uyarılara verilen hemodinamik cevapların kontrolünde, standart kullanılan ajanlara kıyasla daha güvenilir ve efektif olduğuna inanmaktayız.

Anahtar Kelimeler: Beyin cerrahisi, Exmedetomidine, Hemodinami, Kraniyotomi

Introduction

In neuroanesthesia, the appropriate surgical conditions ensure that the brain is minimally affected by the procedure, without jeopardizing autoregulation of the cerebral circulation. Rapid recovery from neuroanesthesia and early neurological examination are also required [1]. In addition, it has been reported that the prevention of the hypertensive responses at the recovery stage is an important issue with regards to reducing the extent of intracranial hemorrhage [2].

Another important consideration is the duration of effects mediated by drugs used to depress respiration after extubation. The disadvantage of fentanyl is prolonged respiratory depression when repeated doses are administered [3]. It has been reported that dexmedetomidine, which is an α -2 agonist agent that has recently been used in neuroanesthesia, decreases the hemodynamic response to endotracheal intubation, catecholamine discharge, and surgical stress, thus providing hemodynamic stability [4]. Also, treatment with dexmedetomidine reduces the need for opioid and anesthetic agents [5].

Received: May 04, 2010 / Accepted: May 11, 2010

Summary of this manuscript presented in: 2007 TARK Kongresi, Antalya

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doi:10.5152/eajm.2010.19

This study sought to compare fentanyl, one of the standard drugs used in neuroanesthesia, with dexmedetomidine, with regard to the hemodynamic responses to stimulants administered during surgery, recovery criteria, and side effects during the postoperative period, including shivering, nausea, and vomiting.

Materials and Methods

Our study included 30 cases of ASA (American Society of Anesthesiologists) grade I-II patients aged 18-65 years, who were scheduled to undergo operations for supratentorially placed tumors. The study was approved by the Ethical Committee of Gaziantep University Medical Faculty. Informed consent was obtained from each patient. The following patients were excluded from the study: women who were pregnant and lactating; patients with hepatic or renal disorders; patients who had sensitivity to opioids or any of the drugs used; patients who had taken opioids, benzodiazepines or tricyclic antidepressants within 48 hours prior to the study; patients with neurological diseases (Parkinson's, Alzheimer's, Multiple Sclerosis, Amyotrophic Lateral Sclerosis, Guillain's-Barre); alcohol and drug addicts; patients with psychiatric diseases; patients with active infections; and patients whose body mass index (BMI) was greater than 30 kg/m².

The patients were randomized in two groups. The first group received dexmedetomidine infusions (group D; Precedex®, flk, 100 µg mL/L, Abbott Lab., North Chicago, USA), whereas the second group was treated with fentanyl infusions (group F). Both groups consisted of 15 patients each.

Patients who had not been premedicated were placed in a supine position. Patients taken into the operation room were evaluated using applied electrocardiography (ECG) from the standard DII derivation, heart rate (HR), oxygen saturation (SpO₂), systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), end-tidal carbon dioxide (ETCO₂), and temperature (Siemens SC 7000 monitor, Sweden). The gender, age, body weight, and operation time of each case was recorded. The blood pressures (SAP, DAP, MAP), HR and SpO₂ values recorded before the induction were considered as basal values. The same parameters were recorded one minute after the induction, at one and five minutes after intubation, before and one minute after the skull clamp was placed, before starting the skin incision and one minute after the incision. For group D, these values were recorded after the loading infusion at a dose of 1 µg/kg dexmedetomidine and for 10 minutes before the induction. These measurements were recorded every 10 minutes during the operation and at one minute after extubation.

For anesthesia induction, the patients in group D were induced with 2 µg/kg of fentanyl (Fentanyl Citrate-Abbott, USA), 5 mg/kg of thiopental, and 0.1 mg/kg of vecuronium bromide (Norcuron, ORGANON, Netherlands); the patients inhaled 100% oxygen for 3 minutes, and then endotracheal intubation was applied. For anesthesia maintenance, 0.7% isoflurane (Forane-Abbott, USA) in a mixture of 50% oxygen and 50% air was administered. The patients were given, when necessary, additional doses of vecuronium bromide and fentanyl. Dexmedetomidine was continued at a rate of 0.4-0.5 µg/kg/min as the maintenance infusion.

In group F, induction was achieved with 4 µg/kg of fentanyl, and the same dose of thiopental and vecuronium bromide. Anesthesia maintenance was the same as in group D, except for the administration of dexmedetomidine. Fentanyl was continued at a rate of 0.02-0.03 µg/kg/min as the maintenance infusion.

A right internal jugular vein catheter was placed for central venous pressure (CVP) monitoring, a radial artery catheterization was initiated for invasive blood pressure monitoring and blood gas analysis, and a urine catheter was put in place.

Before the skull clamp was put in place, the patients in both groups received fentanyl at a dose of 2 µg/kg as IV, and the skin region to be nailed was infiltrated with 3-5 ml 2% lidocaine. Mechanical ventilation was applied to the patients in both groups such that the tidal volume was 8-10 ml/kg and PaCO₂ was 28-32 mmHg.

During the operation, an increase in MAP of more than 15 mmHg with respect to the basal value with a HR remaining over 90 beats/min for more than one minute was evaluated as mild-insufficient anesthesia. In such cases, an additional 2 µg/kg of fentanyl was administered to patients in either group to increase the infusion rates by 20%.

MAP under 60 mmHg was considered as hypotension, and the infusion dose was reduced accordingly. If this dose was insufficient, then 5 mg of ephedrine was administered. A HR remaining under 50 beats/min for one minute was considered as bradycardia, in which case 0.01 mg/kg atropine IV was administered. Atropine and ephedrine needs were recorded. Before the dura was opened, 20% mannitol at a dose of 1 g/kg was infused over 15 minutes. After the dura was opened, the same surgeon was asked to evaluate the cerebral swelling status using a cerebral relaxation score (1: perfect, no swelling; 2: minimal swelling; 3: substantial swelling, no medication required; and 4: severe swelling, medication required [6]). The dexmedetomidine and fentanyl infusions were continued until the skin sutures were initiated. When all the skin sutures were completed, the administration of anesthetic gases was stopped. Neuromuscular block was antagonized with 0.02 mg/kg of atropine and 0.05 mg/kg of neostigmine. After sufficient respiratory activity was observed and consciousness was regained, the patient was extubated. If sufficient respiratory activity could not be achieved within 15 minutes after the administration of anesthetic drugs was ceased, then anesthesia was antagonized with 0.1 mg of naloxone to address the effect of residual opioid.

After the anesthetic drugs were discontinued, extubation time, the time taken to open eyes after verbal stimulation, and orientation time were assessed and recorded. Patients were evaluated using the Aldrete score (ADS) [7] as an early waking test during the postoperative period, at 5-minute intervals. The time taken for each patient to achieve an ADS score of 8 was recorded. The doses of fentanyl and dexmedetomidine were recorded as well as the use of additional drugs. The patients were taken to the intensive care unit at the end of the operation, and the hemodynamic parameters, SpO₂, and side effects, such as shivering and nausea-vomiting, were recorded for a period of 24 hours.

The statistical data were recorded with SPSS 13.0. Mean±standard deviation values were determined using descriptive analyses. Comparisons of the two groups with regard to hemodynamic, respiratory, biochemical data and recovery periods were carried out with the Mann-Whitney U-test; the Wilcoxon sign test was used for intra-group comparisons. Gender, side effects and need for additional medication were analyzed via χ^2 or Fisher's exact test, as appropriate. Values of $p < 0.05$ were accepted as significant.

Results

The clinical and demographical data of the patients included in our study are presented in Table 1. No statistically significant differ-

ence was found between the groups in terms of age, weight, gender or operation time. Before the induction of anesthesia, blood pressure values, HR, and SpO₂ values were similar between the groups.

In group D, the blood pressure values and HR values for dexmedetomidine after a loading infusion of 10 minutes were significantly lower with respect to the values observed prior to sedation ($p < 0.05$). However, these decreases were less than 20% and within physiological limits. No statistically significant difference was found with regard to SpO₂ values. In group D as compared to group F, there was a statistically significant decrease in MAB value measurements after induction, one minute after intubation, after the skull clamp, after extubation, and during the 2nd hour postoperatively ($p < 0.05$) (Figure 1).

The HR values were significantly lower one minute after intubation, after the skull clamp, at the 30th to 150th min during the operation period, after extubation, and at 2, 4, and 8 hours postoperatively in group D ($p < 0.05$) (Figure 2).

In Group D, there was no difference in MAP values after the operation as compared to before the operation. In Group F, higher values were observed after the induction as compared to before the induction; however, measurements obtained after skull clamp were higher than values observed before the induction ($p < 0.05$). There was no statistically significant difference between the MAP values observed after intubation and those obtained after extubation.

In Group D, HR values before induction, values one minute after intubation, and values observed after skin incision were similar. The HR values observed after skull clamp were lower than the values observed prior to the induction of anesthesia ($p < 0.05$). However, this value was within physiological limits. In Group F, the HR values at one minute after the intubation, after the skin incision, and after the skull clamp did not exhibit significant differences as compared to the values obtained before the induction of anesthesia.

No statistically significant difference was found between groups with regard to SpO₂ values obtained before the induction, after the induction, during the perioperative period, or after extubation. In the blood gas analysis performed after extubation, no statistically significant differences were found between groups for either PaO₂ or PaCO₂ values. There was no statistically significant difference between the two groups for ETCO₂ measurements.

When the groups were evaluated with regard to the cerebral relaxation score, the median (range) in Group D was significantly

lower than in group F [1(1-2) vs. 3(1-3), $p < 0.0001$] (Table 2). Recovery was faster in group D than group F ($p < 0.0001$) (Table 3). The amount of additional narcotics needed was higher in Group F than in group D (546.66 ± 195.91 vs. 261.66 ± 110.14 μ g) ($p < 0.05$).

In Group D, shivering and nausea-vomiting were not observed in any of the patients during the postoperative period, but in group F, shivering was observed in 3 patients and nausea-vomiting in 2

Table 1. Demographic variables for patients (mean \pm SD)

	Group D (n=15)	Group F (n=15)	p-value
Age	39.93 \pm 11.90	43.53 \pm 10.28	NS
Body weight (kg)	71.13 \pm 8.19	76.26 \pm 9.46	NS
Gender (F/M)	8/7	6/9	NS
Operation time (min)	221.93 \pm 24.59	218.26 \pm 31.73	NS

(D) dexmedetomidine; (F) fentanyl; (NS) non-significant ($p < 0.05$)

Table 2. Cerebral Relaxation Scores (CRS) between groups (values are [n(%)])

	Group D (n=15)	Group F (n=15)	p-value
No swelling (score 1)	12 (%80)	1 (%6,6)	<0.0001
Minimal swelling (score 2)	3 (% 20)	6 (% 40)	NS
Important swelling (score 3)	0 (%0)	8 (%53,3)	0.001
Severe Swelling (score 4)	0 (%0)	0 (%0)	NS
Overall CRS [median(range)]	1(1-2)	3(1-3)	<0.0001

(D) dexmedetomidine; (F) fentanyl; (NS) non-significant ($p < 0.05$)

Table 3. Recovery Criteria (mean \pm SD)

	Group D (n=15)	Group F (n=15)	p-value
Time to reach ADS 8 (min)	9.06 \pm 3.63	15.66 \pm 2.60	$p < 0,0001$
Extubation time (min)	4.40 \pm 2.22	9.80 \pm 2.27	$p < 0,0001$
Eye opening time (min)	6.43 \pm 3.29	12.50 \pm 3.26	$p < 0,001$
Response to verbal stimulation (min)	7.76 \pm 3.58	14.60 \pm 3.68	$p < 0,0001$
Cooperation time (min)	9 \pm 3.76	15.66 \pm 4.07	$p < 0,002$

(D) dexmedetomidine; (F) fentanyl ($p < 0.05$)

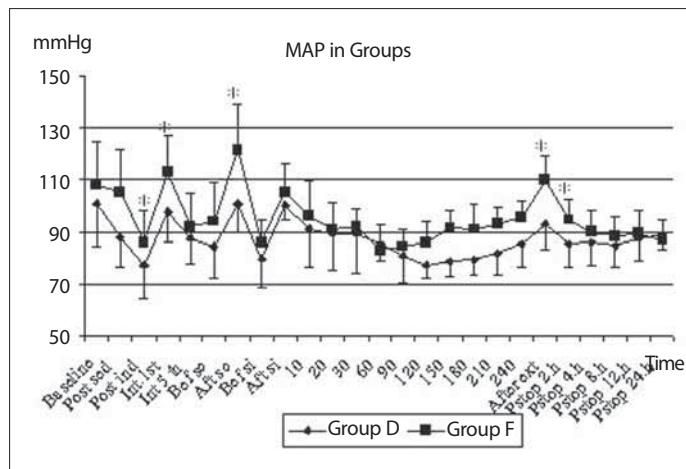


Figure 1. Changes in mean arterial blood pressure (MAP) between groups, * $p < 0.05$.

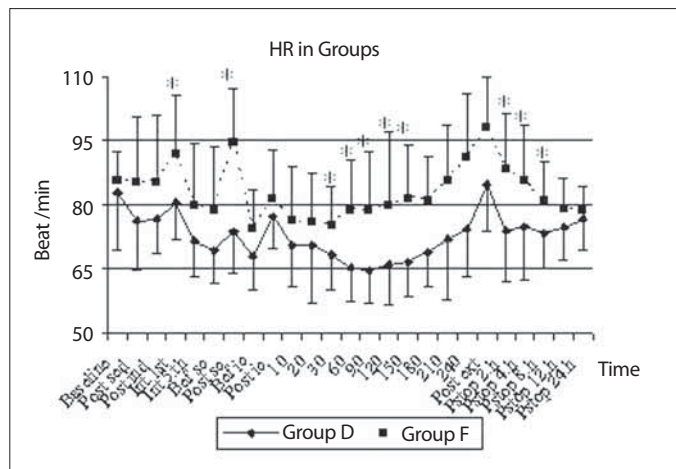


Figure 2. Changes in heart rate (HR) between groups, * $p < 0.05$.

patients. In Group D, 0.5 mg of atropine was administered to one patient; 10 mg of ephedrine was administered to two patients; in group F, 0.5 mg of atropine was administered to one patient, and naloxone was given to one patient as additional medication. There was no significant difference between the groups.

Discussion

In this study, patients with supratentorial tumors were operated upon. These patients developed hypertension due to surgical stress. Heart rate increases in the periods after intubation, after skull clamp, after skin incision, and after extubation were lower in magnitude for the group in which dexmedetomidine was administered as an adjuvant agent. However, these values were 20% higher than the values observed before the induction of anesthesia and were at physiological levels. Larger increments were recorded in the fentanyl group, especially the values observed after skull clamp as compared to the values observed before the induction of anesthesia. A faster recovery and fewer side effects were recorded in the dexmedetomidine group than in group F. Furthermore, during the postoperative period, hemodynamic parameters were lower in the dexmedetomidine group.

Alpha-2 adrenergic agonists have properties that reduce sympatholytic, sedative, and anesthetic requirements as well as provide hemodynamic stabilization. It is also known that dexmedetomidine exhibits an analgesic effect without inducing respiratory depression. Tanskanen *et al.* used dexmedetomidine on the patients undergoing intracranial tumor surgery. They showed that dexmedetomidine depresses tachycardia and the hypertensive response developing at intubation and at extubation better than placebo [1]. In the same study, the authors reported that, during the intraoperative period, dexmedetomidine reduced SAP by 20% in comparison to control group levels.

Bekker *et al.* [8] reported that dexmedetomidine administered during neuroanesthesia reduces the need for opioids, leads to fewer antihypertensive treatments, and provides better hemodynamic stability during incision.

Lawrence *et al.* [9] showed that dexmedetomidine given before the induction as a single dose of 2 µg/kg IV controls the hemodynamic responses to tracheal intubation and extubation as well as HR changes during the intraoperative period, in comparison to control treatment.

We think that the hemodynamic values remained more stable due to the depressed stress response. In our study, hypertension and tachycardia attacks were more effectively controlled in the dexmedetomidine group, especially during the periods when the stress response is pronounced.

Taittonen *et al.* [10] showed that, after premedication with dexmedetomidine, SAP and DAP decreased by 11% and HR decreased by 18%. In our study, we observed a decrease of 13% in SAP, 9% in DAP, and 9% in HR after the bolus infusion of dexmedetomidine for 10 minutes. Although these changes represented statistically significant differences, each parameter remained at physiologically acceptable levels.

In our study, we found that hemodynamic values, as well as HR values one minute after the intubation, were lower in group D as compared to group F. MAP and HR values in group D one minute after the intubation were compared to the values observed after the induction of anesthesia. There was no significant difference. There was also no significant difference when these values were compared for Group F; however, we found the measurements one minute after the intubation were higher than control values.

Many general and local anesthetics have been used to depress the response to skull clamp. Jamali *et al.* [11] reported that when the brain surgery patients were given 0.8 µg/kg sufentanyl and 4.5 µg/kg fentanyl before skull clamp application, MAP and SAB decreased by 10 mmHg. However, these values returned to baseline after the application of a skull clamp. The authors also stated that HR was reduced by 10/min in the sufentanyl group and 8/min in the fentanyl group.

Although the hemodynamic measurements before the skull clamp placement were similar, after the skull clamp was placed, we recorded lower values for blood pressure and HR in group D. When we compared the MAP values after the skull clamp was placed with values obtained before the induction of anesthesia in Group D, there was no significant difference; however, HR was lower after the clamp than before. In Group F, while the MAP values after skull clamp were significantly higher than the preinduction values, HR increased after the skull clamp, but this increase was not significant. When we evaluated blood pressures and HR after extubation, we observed significantly lower measurements in group D as compared to group F.

In the studies conducted by Ebert *et al.* [12] and Belleville *et al.* (13), dexmedetomidine doses over 2 µg/kg minimized respiratory depression as compared to the control group. It was also shown in various studies that while dexmedetomidine exhibited sedative and analgesic effects, it did not cause respiratory depression and did not lead to reductions in PaO₂ or CO₂ retention [12, 14]. Also, consistent with the literature, we observed no significant difference in the SpO₂ values before and after the dexmedetomidine infusion.

Tanskanen *et al.* [1] showed that the dexmedetomidine infusion provided faster recovery after anesthesia without inducing respiratory depression after extubation. The extubation time was shorter in dexmedetomidine-treated patients as compared to fentanyl-treated patients. Another clinical study [5] determined that when dexmedetomidine was administered as a single dose IV before the induction of anesthesia, the period for recovery from the anesthesia after the operation was shortened.

Our results showed no significant difference between the two groups in terms of SpO₂ values after extubation. In our study, significantly shorter recovery periods were observed in Group D as compared to group F (Table 3). Therefore, it is suggested that dexmedetomidine might be more advantageous than fentanyl in cranial surgery anesthesia. No respiratory depression was observed in either group; however, rapid recovery was observed after treatment with dexmedetomidine. Other studies [12, 15] showed that dexmedetomidine does not cause a decrease in PaO₂ or CO₂ retention.

According to our results, no significant differences were observed between the groups with regard to PaO₂ and PaCO₂ values after extubation.

It is well known that the α-2 agonists have little effect on ICP, that direct activation of cerebral α-2 receptors causes cerebral vasoconstriction and that decreasing the MAP and CBF provides a clinically superior surgical area [16]. Ard *et al.* [17] reported that dexmedetomidine provides better operational conditions due to minimal cerebral swelling, which is due to the decrease in CBF. Even though we did not have the opportunity to directly measure the effects of dexmedetomidine on CBF in our study, cerebral swelling (as a reflection of CBF in combination with the cerebral relaxation score) was higher in group F as compared to group D (Figure 3). Therefore, dexmedetomidine treatment resulted in improved cerebral pressures.

In a prospective clinical study [18] that investigated dexmedetomidine as an adjuvant agent, the authors found that the duration of PACU stay was significantly shorter in the dexmedetomidine group.

Although patients in the dexmedetomidine group had fewer hypertensive episodes in our study, we determined the blood pressures and HR values at the 2nd hour postoperatively to be significantly lower in group D as compared to group F. Therefore, dexmedetomidine provides better control of postoperative hemodynamics than fentanyl, minimizing postoperative complication risks.

Aantaa *et al.* [19] showed that when 1 µg/kg of dexmedetomidine was used as premedication, the thiopental dose necessary for the induction of anesthesia was reduced by 55%. In a similar study, the authors reported that dexmedetomidine, administered as premedication, reduced the dose of IV anesthetic agents required, as well as the need for opioids during the intraoperative period [10].

In our study, the amount of additional narcotics required was significantly lower in group D as than group F.

Postoperative complications such as nausea, vomiting and shivering affect postoperative comfort. Several studies [16, 20] have shown that dexmedetomidine reduces postoperative shivering. In a clinical study conducted to investigate nausea-vomiting [9], the dexmedetomidine group required fewer antiemetics than the control group.

In our study, no patient in group D exhibited nausea, vomiting or shivering. However, in group F, shivering was observed in three patients, and nausea was observed in two patients.

Hypotension and bradycardia are sometimes reported in connection with dexmedetomidine. Aryan *et al.* [20] encountered bradycardia in only one of the 39 patients who were infused with dexmedetomidine and followed up in the intensive care unit; the infusion did not have to be stopped for this patient. Therefore, it was considered that the development of bradycardia did not represent a clinical problem. In another study [5], hypotension (SAP <90 mmHg) was observed in four patients in the group that received dexmedetomidine before the induction; fluid infusion was sufficient among treated patients; and no patient required ephedrine. The authors of this study encountered atropine requiring bradycardia in one patient after induction and in two patients during the postoperative period. No hypotension was observed in group F, whereas bradycardia was observed in one patient after induction.

In our study, we observed only one case of hypotension and one case of bradycardia in group D, and only one case of bradycardia in Group F. Neither hypotension nor bradycardia was observed in any patient during the postoperative period.

In conclusion, dexmedetomidine is effective during supratentorial tumor surgery for controlling perioperative hemodynamic responses and inducing cerebral relaxation as well as for reducing fentanyl consumption during the perioperative period. This shortens the recovery time and improves postoperative respiratory functions.

Conflict of interest statement: The authors declare that they have no conflict of interest to the publication of this article.

References

1. Tanskanen PE, Kytta JV, Randell TT, Aantaa RE. Dexmedetomidine as an anaesthetic adjuvant in patients undergoing intracranial tumour surgery: a double-blind, randomized and placebo-controlled study. *Br J Anaesth* 2006; Nov;97: 658-65.
2. Basali A, Mascha EJ, Kalfas I, Schubert A. Relation between perioperative hypertension and intracranial hemorrhage after craniotomy. *Anesthesiology* 2000; 93: 48-54.
3. Guy J, Hindman BJ, Baker KZ, et al. Comparison of remifentanyl and fentanyl in patients undergoing craniotomy for supratentorial space-occupying lesions. *Anesthesiology* 1997; 86: 514-24.
4. Mantz J. Dexmedetomidine. *Drugs Today (Barc)* 1999; 35: 151-7.
5. Yildiz M, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu S. Effect of dexmedetomidine on haemodynamic responses to laryngoscopy and intubation : perioperative haemodynamics and anaesthetic requirements. *Drugs R D* 2006; 7: 43-52.
6. Gelb AW, Salevsky F, Chung F, et al. Remifentanyl with morphine transitional analgesia shortens neurological recovery compared to fentanyl for supratentorial craniotomy. *Can J Anaesth* 2003; 50: 946-52.
7. Aldrete JA. The post-anesthesia recovery score revisited. *J Clin Anesth* 1995; 7: 89-91.
8. Bekker A, Sturaitis MK. Dexmedetomidine for neurological surgery. *Neurosurgery* 2005; 57: 1-10.
9. Lawrence CJ, De Lange S. Effects of a single pre-operative dexmedetomidine dose on isoflurane requirements and peri-operative haemodynamic stability. *Anaesthesia* 1997; 52: 736-44.
10. Taittonen MT, Kirvela OA, Aantaa R, et al. Effect of clonidine and dexmedetomidine premedication on perioperative oxygen consumption and haemodynamic state. *Br J Anaesth* 1997; 78: 400-6.
11. Jamali S, Archer D, Ravussin P, et al. The effect of skull-pin insertion on cerebrospinal fluid pressure and cerebral perfusion pressure: influence of sufentanil and fentanyl. *Anesth Analg* 1997; 84: 1292-6.
12. Ebert TJ, Hall JE, Barney JA, et al. The effects of increasing plasma concentrations of dexmedetomidine in humans. *Anesthesiology* 2000; 93: 382-94.
13. Belleville JP, Ward DS, Bloor BC, et al. Effects of intravenous dexmedetomidine in humans. I. Sedation, ventilation, and metabolic rate. *Anesthesiology* 1992; 77: 1125-33.
14. Talke P, Chen R, Thomas B, et al. The hemodynamic and adrenergic effects of perioperative dexmedetomidine infusion after vascular surgery. *Anesth Analg* 2000; 90: 834-9.
15. Johnson KB, Egan TD. Remifentanyl and propofol combination for awake craniotomy: case report with pharmacokinetic simulations. *J Neurosurg Anesthesiol* 1998; 10: 25-9.
16. Cormack JR, Orme RM, Costello TG. The role of alpha2-agonists in neurosurgery. *J Clin Neurosci* 2005; 12: 375-8.
17. Ard JL, Jr., Bekker AY, Doyle WK. Dexmedetomidine in awake craniotomy: a technical note. *Surg Neurol* 2005; 63: 114-6.
18. Bekker A, Sturaitis M, Bloom M, et al. The effect of dexmedetomidine on perioperative hemodynamics in patients undergoing craniotomy. *Anesth Analg* 2008; 107: 1340-7.
19. Aantaa RE, Kanto JH, Scheinin M, Kallio AM, Scheinin H. Dexmedetomidine premedication for minor gynecologic surgery. *Anesth Analg* 1990; 70: 407-13.
20. Aryan HE, Box KW, Ibrahim D, et al. Safety and efficacy of dexmedetomidine in neurosurgical patients. *Brain Inj* 2006; 20: 791-8.