Egyptian Journal of Anaesthesia (2014) **30**, 261–266

Egyptian Society of Anesthesiologists

Egyptian Journal of Anaesthesia

www.elsevier.com/locate/egja
www.sciencedirect.com

Research Article

Dexmedetomidine is an effective adjuvant to subtenon block in phacoemulsification cataract surgery

Ashraf M. Eskandr [a,*], **Abd El-Azeem A. Elbakry** [a], **Osama A. Elmorsy** [b]

[a] *Department of Anaesthesia, Faculty of Medicine, Menoufiya University, Egypt*
[b] *Department of Ophthalmology, Faculty of Medicine, Menoufiya University, Egypt*

Received 30 August 2013; revised 15 January 2014; accepted 18 January 2014
Available online 7 February 2014

KEYWORDS

Analgesia;
Dexmedetomidine;
Intraocular pressure;
Subtenon

Abstract *Background:* Researches to find a better adjuvant in regional anesthesia are still continued until now.
Dexmedetomidine prolongs anesthesia and analgesia of local anesthetics in various neural blocks as well as the onset of sensory and motor block. The objective of the present study was to evaluate the effect of adding dexmedetomidine to local anesthetics on the sensory and motor block of the subtenon block in patients undergoing phacoemulsification cataract surgery.
Methods: Sixty patients of American Society of Anaesthesiologists (ASA) grade I–III, aged between 18 and 70 years, scheduled for phacoemulsification cataract surgery were randomly assigned to two equal groups. Group C (control group) received 2 ml of a mixture of 2% lidocaine and 0.5% bupivacaine and Group D (dexmedetomidine group) received 2 ml of a mixture of 2% lidocaine and 0.5% bupivacaine plus dexmedetomidine (0.5 μg/kg). Onset and duration of sensory and motor block was recorded. Pain during administration of anesthesia and during surgery was graded using the verbal analogue scale and recorded. Intraocular pressure, hemodynamic, and sedation parameters were recorded before and after surgery.
Results: Onset of both sensory and motor block was significantly decreased in group D ($P < 0.001$, $P = 0.004$ respectively), and duration of sensory and motor block was more prolonged in group D than in group C ($P < 0.001$, $P = 0.961$). Pain during administration of anesthesia was

* Corresponding author. Address: 3 Yassin Abdelghafar St., Shibeen Elkoom, Menoufiya, Egypt. Tel.: +20 01001960697.
E-mail address: ameskandr@yahoo.com (A.M. Eskandr).
Peer review under responsibility of Egyptian Society of Anesthesiologists.

Production and hosting by Elsevier

 http://dx.doi.org/10.1016/j.egja.2014.01.007

significantly lower in group I compared with group II, and more patients in group I compared with group II were pain free, without a significant difference between the two groups. Intraocular pressure was significantly decreased in group D ($P < 0.001$). More sedation score was observed in group D ($P = 0.022$). Heart rate and mean arterial blood pressure were insignificantly decreased in group D more than in group C.

Conclusion: Dexmedetomidine is a safe and effective adjuvant to subtenon block in phacoemulsification cataract surgery.

1. Introduction

Dexmedetomidine is a novel selective α_2 receptor agonist that produces sedation and analgesia without causing respiratory depression [1]. It also allows patients to respond to verbal commands during the sedation; easy conversion from sleeping to awakening is possible [2]. Therefore, dexmedetomidine has been used in various clinical fields, such as sedation in the intensive care unit, radiological examination of pediatric patients, awake intubation, shockwave lithotripsy, endoscopic examination [3–7] and an adjuvant to local anesthetics [8,9].

Many studies were done to evaluate its effect as sedative when administered intravenously and other studies were done to evaluate its effect on analgesia when added to local anesthesia in axillary-supraclavicular and infraclavicular plexus, intrathecal, epidural and perineural block [10–16]. But no one, till now studied its effect when used as adjuvant to local anesthetics in subtenon block. So, the present study was scheduled to study the effects of adding dexmedetomidine to local anesthetics on the sensory and motor block of the subtenon block in patients undergoing phacoemulsification cataract surgery.

2. Methods

After approval of the local ethical committee and obtaining written informed consent, 60 patients, ASA grade I–III and aged 18–70 years of both sex scheduled for elective phacoemulsification cataract surgery with sub-Tenon's anesthesia were included in this randomized blind study which was done in Menoufiya University Hospitals. Exclusion criteria included the usual contraindications for regional anesthesia, coagulation abnormalities, impaired mental status, uncontrolled glaucoma, recent surgical procedure on the same eye, and refusal of the patient. A peripheral intravenous catheter was inserted, and monitoring included continuous electrocardiography, pulse oximetry, and automated noninvasive blood pressure measurement. Before induction of blockade, benoxinate hydrochloride 0.4% drops were instilled and no IV sedative/hypnotic medication was used before or during the block. Patients were assigned randomly through closed envelop method to receive single-injection. All punctures were performed by the same person (anesthetist or surgeon) using a 25-gauge needle. The needle was inserted to contact the conjunctiva between the eyeball and the semilunaris fold, at a depth of less than 1 mm, with the bevel directed toward the globe. The needle was then shifted slightly medially, displacing the semilunaris fold and caruncle away from the eyeball. The needle was advanced in an anteroposterior direction, with the globe directed slightly medially by the needle, until a 'click' was perceived, at a depth of B15–20 mm. At this moment, the globe returned to the primary gaze position. This point represents a reliable depth marker that confirms the episcleral location of the tip of the needle. In each group, the local anesthetic solution was injected after an aspiration test, in group D (Dexmedetomidine group) 2 ml of a mixture of 0.5 μg/kg dexmedetomidine and equal parts of 0.5% bupivacaine and 2.0% lidocaine was injected and in group C (Control group) 2 ml of a mixture of equal parts of 0.5% bupivacaine and 2.0% lidocaine was injected. Demographic data included age, gender, weight, and height were recorded. Duration of surgery, onset and duration of sensory and motor block were recorded. Pain during anesthesia administration and surgery was recorded. Patients rated pain during injection of anesthetics and during surgery using the verbal analog scale, with scores ranging from 0 to 4 (grade 0, no pain; grade 1, mild pain; grade 2, moderate pain; grade 3, severe pain; and grade 4, maximum pain). Also, hemodynamic parameters (HR and MAP), sedation score and intraocular pressure were recorded before and after surgery. Sensory block duration or duration of analgesia was defined as the time from injection of local anesthetic mixture to complete recovery from pain sensation or the first need of rescue analgesia was measured and recorded. Motor block duration was described as the time from injection of local anesthetic to complete recovery of motor function in all ocular muscles. Ocular akinesia (immobility) of the globe during surgery was scored. A 12-point scale described by Brahma et al. [17] was used in which akinesia of ocular movements in each quadrant was scored between 0 and 3 (0, no block; 1, partial akinesia unsuitable for surgery; 2, partial but sufficient akinesia; 3, total akinesia); the final score was the total of these four subscores; hence, the minimum score possible was 0 and the maximum was 12 (3×4). The patient's level of sedation was assessed using the inverted observer's assessment of alertness/sedation scale [18], with a score of 1 = completely awake, 2 = awake but drowsy, 3 = asleep but responsive to verbal commands, 4 = asleep but responsive to tactile stimulus, 5 = asleep and not responsive to any stimuli.

There were no available previous data to depend on for calculation of the sample size required in the present study so that a pilot study was conducted on a number of 10 patients given subtenon block and resulted in an increase in the duration of the sensory block from 88.6 ± 4.79 min in the control group to 181.1 ± 4.1 min in the group where dexmedetomidine was added. The sample size was calculated to be 25 patients, so we decided to include 30 patients in each group in the study. We used GraphPad Stat Mate version 2 statistics program for power analysis.

Statistical analysis was performed using SPSS version 10. Results were expressed as the mean ± SD as indicated. A Student's t test was used to compare the quantitative variables between the two groups. Chi-square analysis was used to

Table 1 Demographic data and duration of surgery.

	Group D (n = 30)	Group B (n = 30)	P-value
Age (years)	57.97 ± 11.75	58.03 ± 11.41	0.984
Weight (kg)	74.6 ± 9.66	75.23 ± 7.54	0.779
Height (cm)	166.07 ± 5.64	166.03 ± 5.8	0.978
Sex (F/M)	10/20	12/18	0.789
Duration of surgery(min.)	26.2 ± 6.8	28.6 ± 5.62	0.142

Group C: control group, Group D: dexmedetomidine group, M: male, F: female, n = number of patients. Data were expressed as mean ± standard deviation and number of patients.

compare qualitative values between the two groups. $P < 0.05$ was considered significant.

3. Results

There were no significant differences between the two groups with regard to age, weight, height, gender, and duration of surgery (Table 1). As regards pain during administration of anesthesia, there was a significant difference between both groups for grades 0 and 1 ($P < 0.05$) and an insignificant difference between the two groups for the other grades (Table 2). In addition, there was an insignificant difference between the studied groups with respect to pain during surgery (Table 2) despite more patients being pain free in dexmedetomidine group than in control group during surgery. The onset of sensory block was significantly shorter in the dexmedetomidine group as compared with the control group ($P < 0.001$). Also, the onset of motor block (globe akinesia) was significantly shorter in the dexmedetomidine group than in the control group ($P = 0.004$) (Table 3). As regards, the duration of analgesia or sensory block (the time interval from injection of local anesthetic to first analgesic intake) was significantly longer in the dexmedetomidine group as compared with the control group ($P < 0.001$) (Table 3). There was no significant difference between the two groups ($P = 0.961$) as regards, the duration of akinesia (Table 3). Akinesia score was better in dexmedetomidine group than in control group but with insignificant difference between the two groups (Table 4). The intraocular pressure showed a significant decrease between the preoperative and postoperative values in dexmedetomidine group and between the two groups ($P < 0.001$) with insignificant difference in the control group (Table 5). The study showed more significant increase in numerical sedation score in the dexmedetomidine group ($P = 0.022$) than in the control group (Table 5). Mean arterial blood pressure was more decreased in the dexmedetomidine group than in the control group with insignificant difference within and between them (Table 5). In relation to heart rate, there was high significant decrease in dexmedetomidine group ($P < 0.001$) and insignificant difference in the control group between the preoperative and postoperative measures with high significant difference between the two groups ($P < 0.001$) (Table 5).

4. Discussion

The present study demonstrated that adding dexmedetomidine to lidocaine and bupivacaine mixture in subtenon block produces a significant rapid onset of sensory and motor block, significant prolongation of analgesia and insignificant prolongation of globe akinesia, and decreasing IOP with safe hemodynamic changes and sedative effect.

Dexmedetomidine is a new alpha-2 agonist which has got numerous beneficial effects [14]. It acts on both pre- and post-synaptic sympathetic nerve terminal and central nervous system thereby decreasing the sympathetic outflow and norepinephrine release causing sedative, anti-anxiety, analgesic, sympatholytic and hemodynamic effects [13,14]. Various clinical studies on intravenous dexmedetomidine resulted in significant opioid sparing effects [6].

In previous animal studies, dexmedetomidine has been reported to enhance sensory and motor blockade along with increased duration of analgesia [7–10]. In humans, dexmedetomidine has also shown to prolong the duration of block and postoperative analgesia when added to local anesthetic in various regional blocks as axillary, supraclavicular and infraclavicular brachial plexus, intrathecal, epidural and perineural blocks [11–16]. These previous study results were coincident with the present study results which showed significant decrease in onset of sensory and motor block with prolongation of sensory and motor block duration. These effects

Table 2 Pain during anesthesia and surgery.

Grade	Pain during anesthesia			Pain during surgery		
	C group (n = 30)	D group (n = 30)	P-value	C group	D group	P-value
0	20(66.7%)	28(93.3%)*	0.024	27(90%)	29(96.7%)	0.612
1	9(30%)	2(6.7%)*	0.045	2(6.7%)	1(3.3%)	1.000
2	1(3.3%)	0	1.000	1(3.3%)	0	1.000
3	0	0	–	0	0	–
4	0	0	–	0	0	–

Group C: control group, Group D: dexmedetomidine group, n = number of patients. Data were expressed as number of patients (%).
* $P < 0.05$: significant.

Table 3 Onset and duration of sensory and motor block.

	C group (n = 30)	D group (n = 30)	P-value
Onset of sensory block (min.)	2.43 ± 0.74	1.65 ± 0.63*	<0.001
Onset of motor block (min.)	3.03 ± 1.35	2.1 ± 1.06*	0.004
Duration of sensory block (min.)	87.9 ± 4.9	182.6 ± 5.1*	<0.001
Duration of motor block (min.)	159.73 ± 7.32	166.33 ± 5.94	0.961

Group C: control group, Group D: dexmedetomidine group, n = number of patients. Data were expressed as mean ± standard deviation.
* $P < 0.05$: significant.

of dexmedetomidine can be explained by central and peripheral actions. The central actions are mediated through α_2 adrenoceptors, which are situated at locus coeruleus and dorsal horn of spinal cord [19]. The peripheral actions of dexmedetomidine on peripheral nerve blocks are mediated through four mechanisms; these mechanisms are centrally mediated analgesia, α_2B adrenoceptor mediated vasoconstrictive effects, attenuation of inflammatory response and direct action on peripheral nerve [20]. This direct action can be explained on the basis of many studies, proposing that α_2 agonists (clonidine, dexmedetomidine) by enhancing activity-dependent hyperpolarization generated by the Na/K pump during repetitive stimulation, increases the threshold for initiating the action potential causing slowing or blockage of conduction [21–23].

The intraocular hypotensive effect of dexmedetomidine in the present study is consistent with previous several studies on α_2 agonists. Dexmedetomidine was effective in preventing the rise of the IOP in response to succinylcholine and endotracheal intubation [24]. Dexmedetomidine infusion as an adjunct to local analgesia in ophthalmic surgery was effective in reduction in the IOP significantly [25]. The drug was also found to reduce the IOP by 34% after a single i.v. dose of dexmedetomidine 0.6 μg/kg [26]. Similar effects were shown in elderly patients during cataract surgery [27,28]. Also, Yazbeck-Karam and co-workers studied supplementation of retrobulbar block with clonidine in vitreoretinal surgery showed a decrease in IOP [29]. On the contrary, when Lee and colleagues, infused dexmedetomidine as a supplement to isoflurane anesthesia, they found no IOP lowering effect [30]. This difference can be explained as Lee and colleagues measured IOP at 3 times; the base line, before the loading dose was given and 1 min after intubation and the loading dose started 10 min before induction of anesthesia, as the time from the loading dose induction till the last measurement was 10–11 min which is not a sufficient time for maximal effect of dexmedetomidine, while in the present study, we compare between the baseline and the postoperative measurement.

The effect of dexmedetomidine on the IOP may be caused by a direct vasoconstrictor effect on the afferent blood vessels of the ciliary body, which results in reduction in aqueous humor production [31]. Moreover, it could increase outflow of

Table 4 Ocular movement during surgery.

Akinesia score	C group (n = 30)	D group (n = 30)	P-value
0	1 (3.3%)	0	1.000
2	3 (10%)	1 (3.3%)	0.612
4	6 (20%)	7 (23.3)	1.000
6	6 (20%)	7 (23.3%)	1.000
8	12 (40%)	13 (43.4%)	1.000
10	2 (6.7%)	2 (6.7%)	1.000
12	0	0	–

Group C: control group, Group D: dexmedetomidine group, n = number of patients. Data were expressed as number of patients (%).

Table 5 Intraocular pressure, sedation score and hemodynamic parameters.

	C group (n = 30)	D group (n = 30)	P-value
Intraocular pressure			
Preoperative	16.17 ± 1.51	16.1 ± 1.09	0.838
Postoperative	16.47 ± 1.46	15.23 ± 0.57*	<0.001
P-value	0.437	<0.001	
Sedation score			
Preoperative	1.07 ± 0.25	1.07 ± 0.25	*1.000*
Postoperative	1.03 ± 0.18	1.33 ± 0.55*	*0.006*
P-value	0.561	0.022	

	Heart rate			MAP		
	C group	D group	P-value	C group	D group	P-value
Hemodynamic parameters						
Preop	74.73 ± 6.23	74.27 ± 6.14	0.774	93.03 ± 10.47	93.13 ± 10.99	*0.971*
Postop	74.47 ± 5.69	68.43 ± 3.89*	<0.001	91.07 ± 7.9	89.32 ± 6.66	*0.357*
P-value	0.867	<0.001		0.416	0.11	

Group C: control group, Group D: dexmedetomidine group, n = number of patients. Data were expressed as mean ± standard deviation.
* $P < 0.05$: significant.

the aqueous humor caused by a reduction in the sympathetically mediated vasomotor tone of the ocular drainage system [32]. Additionally, its associated hemodynamic response could contribute to the IOP lowering effect [33].

Significant decrease in heart rate and mean arterial blood pressure from the baseline was reported in many studies, when dexmedetomidine was added to local anesthetics [13–15,27,34]. The decrease in heart rate and mean arterial blood pressure caused by α-2 agonist can be explained by their central action decreasing the sympathetic outflow and norepinephrine release. Although the decrease in heart rate and mean arterial blood pressure reported in dexmedetomidine group, it never was less than 20% of the baseline values which proved that the use α-2 agonists provides a hemodynamic stability during the intra- and post-operative periods.

There was a significant increase in sedation score in dexmedetomidine group with arousable effects, which can be explained by the central action of dexmedetomidine as some amount of systemic absorption of the drug could be present, this is in accordance with other studies [11,19].

In conclusion, the present study demonstrated that adding dexmedetomidine (0.5 μg/kg) to a mixture of 2% lidocaine and 0.5% bupivacaine in subtenon block for patients undergoing cataract phacoemulsification surgery, resulted in a significant rapid onset and prolongation of analgesia and akinesia with decreased IOP and stable hemodynamic changes. So, we recommend more studies including large number of patients to confirm our study findings about the usage of dexmedetomidine as a safe and effective adjuvant to subtenon block.

Conflict of interest

No conflict of interest.

References

[1] Gerlach AT, Dasta JF. Dexmedetomidine: an updated review. Ann Pharmacother 2007;41:245–52.

[2] Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anesth Analg 2000;90:699–705.

[3] Bergese SD, Khabiri B, Roberts WD, Howie MB, McSweeney TD, Gerhardt MA. Dexmedetomidine for conscious sedation in difficult awake fiberoptic intubation cases. J Clin Anesth 2007;19:141–4.

[4] Siddappa R, Riggins J, Kariyanna S, Calkins P, Rotta AT. High-dose dexmedetomidine sedation for pediatric MRI. Paediatr Anaesth 2011;21:153–8.

[5] Tan JA, Ho KM. Use of dexmedetomidine as a sedative and analgesic agent in critically ill adult patients: a meta-analysis. Intensive Care Med 2010;36:926–39.

[6] Muller S, Borowics SM, Fortis EA, Stefani LC, Soares G, Maguilnik I, et al. Clinical efficacy of dexmedetomidine alone is less than propofol for conscious sedation during ERCP. Gastrointest Endosc 2008;67:651–9.

[7] Kaygusuz K, Gokce G, Gursoy S, Ayan S, Mimaroglu C, Gultekin Y. A comparison of sedation with dexmedetomidine or propofol during shockwave lithotripsy: a randomized controlled trial. Anesth Analg 2008;106:114–9.

[8] Bekker A, Sturaitis M, Bloom M, Moric M, Golfinos J, Parker E, et al. The effect of dexmedetomidine on perioperative hemodynamics in patients undergoing craniotomy. Anesth Analg 2008;107:1340–7.

[9] Bulow NM, Barbosa NV, Rocha JB. Opioid consumption in total intravenous anesthesia is reduced with dexmedetomidine: a comparative study with remifentanil in gynecologic videolaparoscopic surgery. J Clin Anesth 2007;19:280–5.

[10] Esmaoglu A, Yegenoglu F, Akin A, Turk CY. Dexmedetomidine added to levobupivacaine prolongs axillary brachial plexus block. Anesth Analg 2010;111(6):1548–51.

[11] Sarita SS, Varshali MK, Sushma DL, Ruchika R. Comparison of dexmedetomidine and clonidine (α_2 agonist drugs) as an adjuvant to local anesthesia in supraclavicular brachial plexus block: a randomised double-blind prospective study. Indian J Anesth 2012;56:243–9.

[12] Ammar AS, Mahmoud KM. Ultrasound-guided single injection infraclavicular brachial plexus block using bupivacaine alone or combined with dexmedetomidine for pain control in upper limb surgery: a prospective randomized controlled trial. Saudi J Anaesth 2012;6(2):109–14.

[13] Kanazi GE, Aouad MT, Jabbour-Khoury SI, Al Jazzar MD, Alameddine MM, Al-Yaman R, et al. Effect of low-dose dexmedetomidine or clonidine on the characteristics of bupivacaine spinal block. Acta Anaesthesiol Scand 2006;50(2):222–7.

[14] Sukhminder JS, Sukhwinder KB, Jasbir K, Gurpreet S, Vikramjit A, Sachin G, et al. Dexmeditomidine and clonidine in epidural anaesthesia: a comparative evaluation. Indian J Anaesth 2011;55:116–21.

[15] Sukhminder JB, Vikramjit A, Jasbir K, Amarjit S, Parmar SS. Comparative evaluation of dexmedetomidine and fentanyl for epidural analgesia in lower limb orthopaedic surgeries. Saudi J Anaesth 2011;5:365–70.

[16] Abdallah FW, Brull R. Facilitatory effects of perineural dexmedetomidine on neuraxial and peripheral nerve block: a systematic review and meta-analysis. Br J Anaesth 2013 June;110(6):915–25.

[17] Brahma AK, Pemberton CJ, Ayeko M, Morgan LH. Single medial injection peribulbar anaesthesia using prilocaine. Anaesthesia 1994;49:1003–5.

[18] Chernik DA, Gillings D, Laine H, Hendler J, Silver JM, Davidson AB, et al. Validity and reliability of the observer's assessment of alertness/sedation scale: study with intravenous midazolam. J Clin Psychopharmacol 1990;10:244–51.

[19] Popping DM, Elia N, Marret E, Wenk M, Tramèr MR. Clonidine as an adjuvant to local anaesthetic for peripheral nerve and plexus blocks: a meta-analysis of randomized trials. Anesthesiology 2009;111:406–15.

[20] Brummett CM, Amodeo FS, Janda AM, Padda AK, Lydic R. Perineural dexmedetomidine provides an increased duration of analgesia to a thermal stimulus when compared with a systemic control in a rat sciatic nerve block. Reg Anesth Pain Med 2010;35:427–31.

[21] Dalle C, Schneider M, Clergue F, Bretton C, Jirounek P. Inhibition of the I(h) current in isolated peripheral nerve: a novel mode of peripheral antinociception? Muscle Nerve 2001;24:254–61.

[22] Kosugi T, Mizuta K, Fujita T, Nakashima M, Kumamoto E. High concentrations of dexmedetomidine inhibit compound action potential in frog sciatic nerve without α_2 adrenoceptor activation. Br J Pharmacol 2010;160:1662–76.

[23] Brummett CM, Hong EK, Janda AM, Amodeo FS, Lydic R. Perineural dexmedetomidine added to ropivacaine for sciatic nerve block in rats prolongs the duration of analgesia by blocking the hyper polarization-activated cation current. Anesthesiology 2011;115:836–43.

[24] Mowafi HA, Aldossary N, Ismail SA, Alqahtani J. Effect of dexmedetomidine premedication on the intraocular pressure changes after succinylcholine and intubation. Br J Anaesth 2008;100(4):485–9.

[25] Abdalla MI, Al Mansouri F, Bener A. Dexmedetomidine during local anesthesia. J Anesth 2006;20:54–6.
[26] Scheinin B, Lindgren L, Randell T, Scheinin H, Scheinin M. Dexmedetomidine attenuates sympathoadrenal responses to tracheal intubation and reduces the need for thiopentone and preoperative fentanyl. Br J Anaesth 1992;68:126–31.
[27] Virkkila M, Ali-Melkkila T, Kanto J, Turunen J, Scheinin H. Dexmedetomidine as intramuscular premedication for day-case cataract surgery. A comparative study of dexmedetomidine, midazolam and placebo. Anaesthesia 1994;49:853–8.
[28] Virkkila M, Ali-Melkkila T, Kanto J, Turunen J, Scheinin H. Dexmedetomidine as intramuscular premedication in outpatient cataract surgery: a placebo-controlled dose-ranging study. Anaesthesia 1993;48:482–7.
[29] Yazbeck-Karam VG, Siddik-Sayyid SM, Abi Nader EL, Barakat DE, Karam HS, Cherfane GM, et al. Supplementation of retrobulbar block with clonidine in vitreoretinal surgery: effect on postoperative pain. J Clin Anesth 2011;23(5):393–7.
[30] Lee YY, Wong SM, Hung CT. Dexmedetomidine infusion as a supplement to isoflurane anaesthesia for vitreoretinal surgery. Br J Anaesth 2007;98:477–83.
[31] Macri FJ, Cervario SJ. Clonidine. Arch Ophthalmol 1978;96:2111–3.
[32] Vartiainen J, MacDonald E, Urtti A, Rouhiainen H, Virtanen R. Dexmedetomidine-induced ocular hypotension in rabbits with normal or elevated intraocular pressures. Invest Ophthalmol Vis Sci 1992;33:2019–23.
[33] Georgiou M, Parlapani A, Argiriadou H, Papagiannopoulou P, Katsikis G, Kaprini E. Sufentanil or clonidine for blunting the increase in intraocular pressure during rapid-sequence induction. Eur J Anaesthesiol 2002;13:519–22.
[34] Cheung CW, Ng KF, Choi WS, Chiu WK, Ying CL, Irwin MG. Evaluation of the analgesic efficacy of local dexmedetomidine application. Clin J Pain 2011;27(5):377–82.