

Dexmedetomidine used to provide hypotensive anesthesia during middle ear surgery

Farah Nasreen · Shahjahan Bano · Rashid Manzoor Khan · Syed Abrar Hasan

Abstract This study was carried out to assess the hypotensive effect of low dose dexmedetomidine (DEX) infusion during middle ear surgery. 42 ASA grades I and II patients of either sex aged 18–45 years undergoing elective middle ear surgery were randomly divided into two groups of 21 each. Group I received placebo bolus and infusion of saline at a rate similar to DEX in Group II. Group II received 10–15 min prior to induction of anesthesia 1 µg/kg IV bolus DEX diluted in 10 ml of normal saline over 10 min. Immediately thereafter an infusion of 0.4 µg/kg/hr of DEX commenced. Standard anesthetic technique was used. Halothane was titrated to achieve a mean arterial pressure 30% below the control value (value taken just after premedication). We observed that a statistically significant reduction in the percentage of halothane required to reduce MAP 30% below control value occurred in patients receiving DEX infusion ($1.3 \pm 0.4\%$) in comparison to those receiving placebo ($3.1 \pm 0.3\%$). Patients receiving DEX infusion had a better surgical field. The mean awakening time was significantly reduced in patients of Group II (9.1 ± 2.7 min) when compared to patients of Group I (12.8 ± 2.2 min).

We conclude that DEX can be safely used to provide hypotensive anesthesia during middle ear surgery.

Keywords Dexmedetomidine · Hypotensive anesthesia · Middle ear surgery

F. Nasreen¹ · S. Bano¹ · R. M. Khan¹ · S. A. Hasan²

¹Department of Anaesthesiology,

²Department of Otorhinolaryngology,

J. N. Medical College AMU,

Aligarh - 202 002,

UP, India

F. Nasreen (✉)

E-mail: kazmifarah@ymail.com

Introduction

The need for intentionally reducing blood pressure to hypotensive level in a variety of surgical procedures is gaining popularity today. The practice of middle ear surgery under anesthesia has undergone a revolution with the introduction of hypotensive anesthesia to provide a relatively bloodless field while using an operating microscope [1]. Innumerable techniques/agents have been advocated to achieve hypotension during anesthesia. These have ranged from the use of inhalational agents such as halothane, isoflurane to various drugs such as nitroprusside [2]. Recent publications have demonstrated that the addition of clonidine, an α_2 agonist can enhance hypotensive anesthesia during middle ear surgery [3–5]. Dexmedetomidine (DEX), a newer prototype of α_2 agonists has similar effects as clonidine but efficacy of its peri-anesthetic infusion for induced hypotension and bloodless surgical field during middle ear surgery has yet to be evaluated. The present study was designed to evaluate the effect of low dose DEX infusion on the requirement of halothane to lower mean arterial pressure 30% below control values, quality of blood less field, and awakening time in patients undergoing middle ear surgery.

Materials and methods

Following the approval by institutional Ethics Committee, 42 adult ASA grades I and II patients undergoing middle ear surgery were selected. The patients coming for tympanoplasty, those with history of anticipated difficult airway and cardiorespiratory disease were excluded from the study. Patients were divided into 2 groups of 21 each on a random basis.

All patients were premedicated 15–20 min prior to induction of anesthesia with Inj. Midazolam 0.025 mg/kg, Inj. On-dansetron 0.1 mg/kg & Inj. Tramadol 2 mg/kg. In the operating room ECG, pulse oximetry, non-invasive blood pressure and neuromuscular function using PNS were monitored.

Group I (control group): Received 10–15 min prior to induction of anesthesia placebo bolus and infusion of saline at a rate similar to DEX in Group II.

Group II (study group): Received 10–15 min prior to induction of anesthesia, 1 μ g/kg bolus DEX diluted to 10 ml in NS over 10 min. Immediately thereafter an infusion of 0.4 μ g/kg/hr of DEX commenced. This infusion was terminated 5–7 min prior to end of surgery.

After preoxygenating the patients for 3 min, anesthesia was induced with inj propofol 2 mg/kg and vecuronium 0.1 mg/kg intravenously. Trachea was intubated with cuffed orotracheal tube and anesthesia was maintained with 60% nitrous oxide in oxygen and halothane titrated to achieve a mean arterial pressure (MAP) 30% below the control value.

Control value Value taken just after premedication.

At the end of the surgery residual neuromuscular blockade was reversed with inj. neostigmine 0.04 mg/kg and atropine 0.02 mg/kg i.v. Concentration of halothane recorded in percentage every 15 min till conclusion of surgery.

Surgical field in terms of visual blood loss estimation as reported by the surgeon was recorded.

Grades of surgical field

Grade I: Blood less field not hampering surgery

Grade II: Mild bleeding requiring occasional suctioning

Grade III: Excessive bleeding hampering surgery despite suctioning

Awakening time following reversal of neuromuscular blockade was recorded. This duration comprised from administration of reversal of neuromuscular blockade till sustained eye opening (for >5 sec) on command.

All person responsible for recording parameters were not aware of the nature of infusion (DEX/SAL).

Statistics

The collected data was tabulated and expressed as mean \pm SD. The result was analysed by unpaired t test. Value of $p < 0.05$ was considered significant.

Results

Table 1 shows the demographic data of Group I and II. There was no statistically significant difference between the mean ages and sex of the patients in the two groups ($p > 0.05$).

There was a significant reduction ($p < 0.05$) in the percentage of halothane required to reduce mean arterial pressure 30% below control value in Group II (Table 2).

In Group II, majority of the patients had Grade I surgical field, whereas none of the patient had Grade III surgical field (Table 4). Thus, it is evident that patients receiving

Table 1 Demographic data

Group	Age (yrs)	Sex (M:F)
I (n = 21)	25.2 \pm 8.9	1.6:1
II (n = 21)	23.8 \pm 7.0	1.1:1
P value	>0.05 ns	>0.05 ns

Values are mean \pm SD ns= not significant

Table 2 Comparison of mean percentage of halothane required in two groups to reduce mean arterial pressure 30% below control value

Group	% Halothane	SD
I	3.1	\pm 0.3
II	1.3	\pm 0.4
P value	<0.05 s	

Values are mean \pm SD s = significant

Table 3 Percentage of halothane used in different ranges in either group

Group	<1.0 %	1.1–2.0%	2.1–3.0%	>3.0%
I (n=21)	-	-	10	11
II (n=21)	4	15	2	-

Table 4 Assessment of surgical field by operating surgeon

Group	Grade I		Grade II		Grade III	
	No. of Patients	%	No. of Patients	%	No. of Patients	%
I	7	33.3	10	47.6	4	19.0
II	13	61.9	8	38.0	-	-

Table 5 Comparison of awakening time (min) in two groups

Group	Mean	SD
I	12.8	\pm 2.2
II	9.1	\pm 2.7
P value	<0.05 s	

s = significant

DEX (Group II) had a better surgical field as compared to patients receiving placebo (Group I).

We find that the mean awakening time was significantly reduced in Group II as compared to Group I ($p < 0.05$) (Table 5).

Discussion

A number of techniques/agents have been advocated to achieve hypotension during middle ear surgery. Amongst the pharmacological agents, DEX is the most recently introduced drug to provide hypotensive anesthesia during middle ear surgery [6].

The results of our study indicate that the use of DEX infusion during middle ear surgery reduced the percentage of halothane required to decrease mean arterial pressure 30% below control value. These findings confirm with a previous study which showed that use of DEX reduces requirement of inhalational anesthetics [7, 8]. The probable mechanism of reducing blood pressure by DEX is attributed to stimulation of peripheral alpha 2 adrenoceptors of vascular smooth muscle. This results in decrease in blood pressure and heart rate secondary to inhibition of central sympathetic outflow.

In the present study, it is evident that patients receiving DEX (Group II) had a better surgical field as compared to patients receiving placebo (Group I). These findings can be attributed to the fact that DEX reduces sympathetic activity, resulting in lower blood pressure and reduced heart rate thereby decreasing blood loss at the surgical site and improving the quality of surgical field [6].

In our study there is reduction in mean awakening time following administration of DEX. These findings are consistent with another study which evaluated the sedative effect of DEX in middle ear patients [9]. This finding can be explained by two reasons. Firstly, the use of DEX reduces the requirement of halothane which can prolong awakening time. Secondly, patients receiving DEX are sedated yet easily arousable, a unique feature not observed with other sedatives and inhalation agents [10, 11].

In the present study, none of the patients developed any complications during perioperative period. None of the patients developed heart rate <50 bpm following bolus or infusion of DEX at any stage during intraoperative period. All the patients were hemodynamically stable and none of them required vasopressor support or bolus administration of fluids to maintain hemodynamic status. In the recovery room, patients were monitored for 45 min and then shifted to wards. During this period no complications were noted and also no adverse events were reported during their stay in the ward subsequently.

Thus we conclude that DEX in the doses used in our study, can be safely administered to provide hypotensive anesthesia during middle ear surgery keeping

the hemodynamic fluctuations within the physiological range.

References

1. Kerr AR (1977) Anesthesia with profound hypotension for middle ear surgery. *Br J Anaesth* 49:447–452
2. Fairbairn ML, Eltringham RJ, Young PN and Robinson JM (1986) Hypotensive anesthesia for microsurgery of the middle ear. A comparison between isoflurane and halothane. *Anesthesia* 41:637–640
3. Toivonen J and Kaukinen S (1990) Clonidine Premedication: A useful adjunct in producing deliberate hypotension. *Acta Anaesthesiol Scand* 34:653
4. Marshall JM, Gomez L, Martos C, Sanchez C, Martinez MC and Delgado M (2001) Clonidine decreases intraoperative bleeding in middle ear surgery. *Acta Anaesthesiol Scand* 45: 627–633
5. Stocche RM, Louis VG, Marlene PD and Oswaldo MJ (2003) Clonidine leads to induce hypotension in middle ear surgery. *Rev Bras Anestesiol* 53:1–10
6. Ulger MH, Demirbilek S, Koroglu A, Borazan H and Ersoy MO (2004) Controlled hypotension with dexmedetomidine for middle ear surgery. *J Inon Univ Med Fac* 11:4
7. Aho M, Lehtinen AM, Erkola O, Kallio A and Korttila K (1992) The effect of intravenously administered dexmedetomidine on perioperative haemodynamics and isoflurane requirements in patients undergoing abdominal hysterectomy. *Anesthesiology* 74:997–1002
8. Khan ZP, Munday IT, Jones RM, Thornton C, Mant TG and Amin D (1999) Effects of Dexmedetomidine on isoflurane requirements in healthy volunteers. Pharmacodynamic and pharmacokinetic interactions. *Br J Anaesth* 83:372–380
9. Raul A, Vega S, Carolina C, Silvia S, Elba B and Victoria D (2005) Dexmedetomidine: A New Alpha-2 agonist anesthetic agent in infusion for sedation in middle ear surgery with awake patient. *Anesthesiology* 103:623
10. Venn RM, Bradshaw CJ, Spencer R, Brealey D and Feneck R (1999) Preliminary UK experience of dexmedetomidine, a novel agent for postoperative sedation in the intensive care unit. *Anesthesia* 54:1136–1142
11. Curtis FG, Castiglia YMM, Stolf AA, Ronzella E, Vani SMD, Nascimento Jr P (2002) Dexmedetomidine and Sufentanil as Intraoperative Analgesics. Comparative study. *Rev Bras Anestesiol* 52:525–534