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Influence of dexmedetomidine-propofol-isoflurane and medetomidine-propofol-isoflurane on intraocular pressure and pupil size in healthy dogs

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ABSTRACT: The present prospective, randomised, double-blinded clinical study was designed to investigate the commonly used anaesthetic combinations of dexmedetomidine-propofol-isoflurane and medetomidine-propofol-isoflurane on intraocular pressure and pupil size in dogs. Forty client-owned healthy dogs with no ocular abnormalities, average body weight of 25.7 ± 13.1 kg (mean ± SD) and aged 3.7 ± 2.7 years, were enrolled. Twenty four males and 16 females were included. Dogs were allocated randomly to receive dexmedetomidine *i.v.* at 0.005 mg/kg, dexmedetomidine at 0.01 mg/kg, medetomidine at 0.01 mg/kg or medetomidine at 0.02 mg/kg. Ten minutes later anaesthesia was induced in all dogs with propofol and maintained with isoflurane in oxygen-air. Intraocular pressure, pupil size, heart rate, respiratory frequency and arterial blood pressures (SAP, DAP) were measured prior to (baseline) and at 10 (before propofol), 20, 30, 40, 50 and 60 min after dexmedetomidine or medetomidine administration. Oxygen saturation of haemoglobin (SpO_2) and end-tidal CO_2 concentration ($EtCO_2$) was monitored following anaesthesia induction. Data were analysed using Anderson-Darling and Bartlett's tests for data distribution and homogeneity of variance confirmation and ANOVA followed by Dunnett's tests for multiple comparisons. Changes were considered significant when $P < 0.05$. Following drug administration, pupil size, heart rate and respiratory frequency decreased significantly within groups, but did not differ between groups. No significant changes in intraocular pressure, SAP and DAP within and between groups, and SpO_2 or $EtCO_2$ between groups, were observed. Comparable doses of dexmedetomidine or medetomidine combined with propofol and isoflurane induced reductions in pupil size, respiratory frequency and heart rate, however, without a significant influence on intraocular pressure or arterial blood pressure.

Keywords: alpha-2 agonist; premedication; eye; ophthalmology

The alpha-2 adrenoceptor agonist medetomidine and its enantiomer dexmedetomidine are often used in small animal anaesthesia for sedation or general anaesthesia premedication. Medetomidine is a mixture of two enantiomers – dexmedetomidine and the pharmacologically inactive levomedetomidine. The haemodynamic and respiratory effects of comparable doses of dexmedetomidine and medetomidine are very similar (Murrell and Hellebrekers 2005).

The pressure generated by the ocular aqueous humour on the eye's fibrous layer generates intraocular pressure (IOP). The IOP level is influenced, among others, by choroidal blood flow and the tonus of extraocular muscles on the globe, which can be affected by alpha-2 adrenoceptor agonists. These drugs can influence ocular blood inflow and outflow and intraocular vascular tonus, which can affect IOP (Murphy 1985). The intraocular vascular tonus is regulated by the partial arterial pressure of carbon dioxide ($PaCO_2$) that influences ocular blood flow (Hvidberg et al. 1981). Additionally, changes in arterial blood pressure (systolic arterial pressure) can induce alteration of the IOP (Cunningham and Barry 1986).

The first report of the influence of systemically administered medetomidine on IOP and PS in dogs was presented by Verbruggen et al. (2000). They

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described an increase of IOP in four dogs, and a decrease of IOP in 10 dogs, 5 min after administration of medetomidine at 0.15 mg/m^2 body surface area (corresponds to approximately 0.005 mg/kg). In all 14 dogs medetomidine induced miosis. Kanda et al. (2015) described the effects of medetomidine on IOP and PS in dogs at five different doses. A significant decrease in IOP was observed 6 h after 0.08 mg/kg medetomidine when compared to IOP at 0.25 and 0.50 h, although there were no significant changes in IOP from baseline. In dogs treated with 0.005, 0.01, 0.02 and 0.04 mg/kg medetomidine, there were no significant changes in IOP. Pupil size (PS) did not change significantly after any of the medetomidine treatments when compared with the baseline value.

Artigas et al. (2012) administered dexmedetomidine at 0.005 mg/kg to dogs and measured IOP at 10 and 20 min after drug administration. No significant difference in IOP measurements between T0 and T10 were observed; however, a significant decrease was observed at T20. A significant degree of miosis was observed at T10 after sedation. At T20, the PS increased slightly; however, there was no statistically significant difference between T0 and T10 min. Based on these findings, it was concluded that dexmedetomidine, in combination with mydriatics, may be used in ophthalmic surgical or diagnostic procedures, which require complete dilation of the pupil.

Jaakola et al. (1992) studied the effects of a single intravenous dose of 0.0006 mg/kg of dexmedetomidine on IOP in humans. After dexmedetomidine administration, there was a 34% reduction in IOP.

In our previous study (Rauser et al. 2012) we described the effects of the medetomidine-butorphanol and dexmedetomidine-butorphanol combinations on IOP and PS over a 60 min observation period. We used dexmedetomidine and medetomidine, both at 0.3 mg/m^2 (corresponds to approximately 0.01 mg/kg). Following drug administration, IOP increased significantly at T10 min; the IOP for dexmedetomidine was significantly higher than for medetomidine. There were no significant differences in IOP between groups at any other time points. At T30 and T40 min after drug administration, IOP in both groups significantly decreased below baseline.

Intraocular pressure in dogs under general anaesthesia could be influenced by induction and maintenance agents. Non-significant influence of propofol on IOP in dogs was described by Batista et al. (2000). However, Hofmeister et al. (2008) reported an increase in IOP after propofol administration. Effects of separately administered isoflurane on IOP in dogs have not been reported yet. The influence of isoflurane or sevoflurane in adults was described by Yoshitake et al. (1992). They reported a non-significant reduction in IOP up until 30 min of isoflurane or sevoflurane administration. There are no studies that have reported the effects of comparable doses of medetomidine or dexmedetomidine administered alone prior to general anaesthesia on IOP and PS. The aim of the present study was to determine changes in IOP and PS values, together with cardiorespiratory parameters, after the administration of medetomidine or dexmedetomidine in combination with propofol for induction, and isoflurane for maintenance of general anaesthesia in dogs.

MATERIAL AND METHODS

All procedures were carried out in accordance with the present laws of the Czech Republic and with the consent of the Ethics Committee of the University of Veterinary and Pharmaceutical Sciences, Brno. The techniques described below are very similar to commonly used clinical procedures. Our study was run as a prospective, randomised, double-blinded clinical trial using similar methodology to what was reported in our previous publication (Rauser et al. 2012).

Animals. Dogs with no ocular abnormalities, scheduled for stifle surgery were enrolled in this study. Only dogs with ASA (American Society of Anaesthesiologists) physical status I or II were included. An ophthalmic examination, including observation of eyelids and conjunctiva, slit lamp-biomicroscopy of cornea, anterior chamber, lens and vitreous and applanation tonometry (without pupil dilatation), was performed by an experienced individual blinded to the treatment groups. Only dogs without ophthalmic abnormalities and with IOP measured at 10–25 mmHg (Renwick 2002) prior to sedation were included. Dogs with any health problems, eye pathologies or higher or lower IOP were excluded.

Study protocol. Dogs were randomly allocated by means of blind drawing lots – coloured balls in a black pocket, and were divided into one of four

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groups, each containing 10 animals. Following intravenous (*i.v.*) catheterisation of a cephalic vein, baseline (T0) measurements were performed. Group DEX-5 dogs then received *i.v.* dexmedetomidine at 0.005 mg/kg (Dexdomitor, Orion Pharma, Finland), group DEX-10 dogs received *i.v.* dexmedetomidine at 0.01 mg/kg, group MED-10 dogs received *i.v.* medetomidine at 0.01 mg/kg (Domitor, Orion Pharma, Finland) and group MED-20 dogs received *i.v.* at medetomidine 0.02 mg/kg. A single investigator (Zapletalova) obtained all the measurements and was unaware of which drug had been administered.

In all dogs, 10 min after sedative administration, selected parameters were measured and recorded (see below). Subsequently, anaesthesia was induced with *i.v.* propofol (Norofol, Norbrook, North Ireland), administered slowly using small boluses (0.5 mg/kg, every 30 s) until a plane of anaesthesia suitable for endotracheal intubation was achieved. The total dose of propofol was recorded. Thereafter, all dogs were orotracheally intubated and connected to a semi-closed rebreathing anaesthesia system. Anaesthesia was maintained with isoflurane 1.5% (Aerrane, Baxter, Belgium) in oxygen-air (FiO_2 0.6). All dogs were maintained in lateral recumbency during measurement procedures without any surgical stimulation.

Measurements. In all dogs, intraocular pressure (IOP), pupil size (PS), heart rate (HR), respiratory frequency (f_R) and systolic and diastolic arterial pressures (SAP, DAP) were measured and recorded before sedation (T0) and 10 (before propofol administration), 20, 30, 40, 50 and 60 min after drug administration (T10, T20, T30, T40, T50, T60). Oxygen saturation of haemoglobin (SpO_2), and end-tidal carbon dioxide concentrations ($EtCO_2$) were measured after onset of anaesthesia, intubation and connection to the anaesthesia system at T20, T30, T40, T50, T60.

The intraocular pressure of each dog was measured using applanation tonometry (TonoPen XL, Medtronic, Jacksonville, FL USA). Prior to measurements for each new patient, the rubber cover was replaced and the tonometer was calibrated. During measurement of IOP, each dog was positioned in left lateral recumbency, with the head maintained in a relaxed fashion at the level of the thorax. The dog's head was not below the level of the body in order to avoid both fixation and compression in the cervical area or the globe itself. In all dogs IOP was measured on the same eye – right eyes of dogs in left lateral recumbency or left eye of dogs in right lateral recumbency. The globe was gently fixed in a central position using the conjunctiva and anatomical forceps. Pupil size was measured using a paper ruler placed on the cornea.

The heart rate was measured by auscultation (prior to sedation) and by 3-lead electrocardiography (after sedation). Leads were applied on both front and left hind limbs. The respiratory frequency was measured by observation of chest wall movement (before sedation) and by capnography curve analysis (after sedation). Blood pressure was measured non-invasively using a cuff applied to the front limb. Cuff width was 40% of the circumference of the limb. Collected data included systolic and diastolic arterial pressure. The sensor for $EtCO_2$ measurement was attached to the end of the patient's endotracheal tube and $EtCO_2$ was measured using the side-stream technique. Oxygen saturation of haemoglobin was measured using a sensor applied to the tip of the patient's tongue. Vital-sign monitors were used to measure heart rate and respiratory frequency, $EtCO_2$ and SpO_2 (Datex Cardiocap II, Datex-Ohmeda, Finland), and SAP and DAP (Cardel 9401, Midmark, UK).

Statistical analysis. All parameters – IOP, PS, HR, f_R, SAP, DAP, SpO_2 and $EtCO_2$ – were measured at the same time points in DEX-5, DEX-10, MED-10 and MED-20 groups and were compared to each other. Intraocular pressure, PS, HR, f_R, SAP and DAP measured at times T10, T20, T30, T40, T50 and T60 were also compared with values at time T0.

Statistical analysis was performed using Minitab software (Minitab 16 Statistical Software 2010, State College, PA, USA). Anderson-Darling and Bartlett's tests were used to confirm normal distribution of data and homogeneity of variance, respectively. All variables were compared between groups at each specific time point using one-way analysis of variance (ANOVA) techniques. For multiple comparison of IOP between time points within each tested group, Dunnett's test was used. $P < 0.05$ was considered statistically significant.

RESULTS

Forty healthy dogs comprising 24 males and 16 females aged (mean ± SD) 3.7 ± 2.7 years, and weighing 25.7 ± 13.1 kg, were enrolled in this study. There

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Table 1. Changes in intraocular pressure, pupil size, heart and respiratory rate, systolic and diastolic arterial pressures, end-tidal CO_2 and oxygen saturation of haemoglobin in dogs at selected times after premedication (mean ± SD)

Groups	Time after administration (min)						
	baseline	10+	20	30	40	50	60
Intraocular pressure (mmHg)							
DEX-5	19 ± 4	18 ± 5	19 ± 8	21 ± 6	20 ± 7	22 ± 7	22 ± 6
DEX-10	19 ± 7	22 ± 7	18 ± 6	20 ± 9	18 ± 6	19 ± 8	22 ± 8
MED-10	22 ± 7	20 ± 6	21 ± 7	21 ± 4	20 ± 5	19 ± 6	20 ± 7
MED-20	17 ± 5	21 ± 7	22 ± 10	19 ± 8	19 ± 5	18 ± 6	18 ± 6
Pupil size (mm)							
DEX-5	7 ± 2	4 ± 1*	4 ± 2*	4 ± 2*	4 ± 2*	4 ± 1*	4 ± 1*
DEX-10	6 ± 2	4 ± 2	3 ± 1*	3 ± 2*	3 ± 1*	4 ± 1	3 ± 1
MED-10	8 ± 2	5 ± 2	4 ± 1	4 ± 1	4 ± 2	3 ± 2	6 ± 2
MED-20	7 ± 3	5 ± 2*	4 ± 1*	3 ± 1*	4 ± 2*	3 ± 2*	5 ± 2*
Heart rate (beats/min)							
DEX-5	113 ± 19	67 ± 28*	51 ± 25*	65 ± 31*	75 ± 20*	74 ± 20	84 ± 20
DEX-10	125 ± 29	77 ± 38*	68 ± 18*	93 ± 20	101 ± 26	109 ± 33	115 ± 39
MED-10	101 ± 28	67 ± 29*	62 ± 38*	60 ± 25*	69 ± 26*	75 ± 23*	84 ± 14*
MED-20	101 ± 17	69 ± 30*	66 ± 24*	84 ± 22*	96 ± 27	94 ± 27	90 ± 23
Respiratory frequency (breaths/min)							
DEX-5	63 ± 20	18 ± 10*	13 ± 9*	13 ± 5*	15 ± 7*	18 ± 10*	20 ± 15*
DEX-10	46 ± 27	25 ± 23	17 ± 8*	16 ± 8*	19 ± 8*	22 ± 8*	29 ± 20*
MED-10	90 ± 54	20 ± 6*	20 ± 16*	14 ± 7*	14 ± 4*	16 ± 6*	13 ± 5*
MED-20	78 ± 35	35 ± 36	17 ± 5*	14 ± 5*	18 ± 4*	22 ± 7*	22 ± 7
Systolic arterial blood pressure (mmHg)							
DEX-5	148 ± 19	152 ± 18	138 ± 21	126 ± 27	115 ± 22	123 ± 26	133 ± 24
DEX-10	140 ± 34	141 ± 21	133 ± 25	136 ± 29	133 ± 30	139 ± 33	137 ± 31
MED-10	162 ± 24	162 ± 37	142 ± 20	151 ± 38	150 ± 30	143 ± 22	150 ± 26
MED-20	168 ± 40	161 ± 29	142 ± 30	159 ± 18	151 ± 22	146 ± 24	145 ± 47
Diastolic arterial blood pressure (mmHg)							
DEX-5	105 ± 27	120 ± 22	103 ± 20	87 ± 17	82 ± 18	79 ± 23	91 ± 23
DEX-10	80 ± 23	97 ± 22	88 ± 25	98 ± 23	92 ± 27	99 ± 30	103 ± 27
MED-10	116 ± 21	124 ± 31	107 ± 23	103 ± 47	107 ± 28	99 ± 26	101 ± 20
MED-20	105 ± 41	123 ± 41	95 ± 32	112 ± 17	102 ± 21	98 ± 24	104 ± 24
End-tidal CO_2 (kPa)							
DEX-5			5.1 ± 0.8	4.7 ± 1.2	5.2 ± 0.6	5.0 ± 0.6	4.9 ± 0.4
DEX-10			4.5 ± 0.6	4.7 ± 0.7	4.8 ± 0.8	4.7 ± 0.9	4.4 ± 0.8
MED-10			5.5 ± 0.7	5.2 ± 0.9	5.1 ± 0.9	4.9 ± 1.2	5.1 ± 1.0
MED-20			4.8 ± 0.6	4.8 ± 0.8	4.7 ± 0.6	4.6 ± 0.5	4.4 ± 0.6
Oxygen saturation of haemoglobin (%)							
DEX-5			99 ± 2	97 ± 3	98 ± 1	98 ± 1	98 ± 1
DEX-10			96 ± 3	98 ± 3	96 ± 6	97 ± 3	97 ± 4
MED-10			99 ± 1	99 ± 2	99 ± 1	98 ± 2	99 ± 2
MED-20			98 ± 1	98 ± 1	99 ± 1	97 ± 2	97 ± 3

+Measurement before propofol administration

*Significant decrease in measured parameters within group compared to baseline

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were no significant differences between groups with regard to sex, body mass, age or measured parameters at baselines (IOP, PS, HR, f_R, SAP and DAP).

The propofol dose used for induction of anaesthesia in the DEX-5 group was 1.5 ± 1.2 mg/kg (mean ± SD), in the DEX-10 group 2.0 ± 0.7 mg/kg, in the MED-10 group 1.4 ± 0.8 mg/kg and in the MED-20 group 1.4 ± 0.4 mg/kg. There were no significant differences in the propofol dose used for anaesthesia induction between groups.

We detected no significant differences in IOP within and between groups at any observation time points (Table 1).

Significant decreases in PS in MED-20 and DEX-5 within each group at all observation times compared to T0, and in DEX-10 at T20, T30 and T40 compared to T0, were recorded. No significant differences in PS were detected between groups at any observation time points (Table 1). None of the forty dogs exhibited increased PS compared to baseline at any of the time points.

Significant decreases in HR in MED-10 at all time points compared to T0, in MED-20 at T10, T20, T30 compared to T0, in DEX-5 at T10, T20, T30, T40 compared to T0 and in DEX-10 at T10, T20 compared to T0, were recorded within each group. There were no significant differences in HR between groups at any time points (Table 1).

Within groups, significant decreases in f_R in MED-10 and DEX-5 at all time points compared to T0, in MED-20 at T20, T30, T40, T50 compared to T0 and in DEX-10 at T20, T30, T40, T50, T60 compared to T0 were detected. We detected no significant differences in f_R between groups at any time points (Table 1).

No significant differences in SAP, DAP within and between groups and SpO_2 and $EtCO_2$ between groups were detected at any time points (Table 1).

In three dogs of DEX-5, four dogs of DEX-10, four dogs of MED-10 and four dogs of MED-20 surgery began between T40 and T60. Variables measured at T50 and T60 in the above-mentioned dogs were therefore excluded from evaluation.

DISCUSSION

Ten minutes after premedication, IOP, PS and other measured parameters were influenced by alpha-2 adrenoceptor agonists only. At this time point, we detected no significant differences in IOP within and between groups. Despite alterations in IOP in several dogs, these changes were insignificant and IOP remained within physiological limits (10–25 mmHg). Our observations are not in agreement with those of Verbruggen et al. (2000), where an increase in IOP in four dogs, and a decrease in 10 dogs, 5 min after administration of medetomidine at 0.15 mg/m^2 body surface area (corresponds to approx. 0.005 mg/kg), was reported. The dose of medetomidine in the present study was two to four times higher when compared to the dose used by Verbruggen et al. (2000). In our previous study (Rauser et al. 2012), where we used comparable doses of medetomidine (0.3 mg/m^2) or more than twice the higher dose of dexmedetomidine used here (0.3 mg/m^2), we detected an increase in IOP 10 min after premedication before propofol administration. However, in that study we used an alpha-2 adrenoceptor agonist in combination with butorphanol and the patient was positioned in ventral recumbency. It is possible that patient positioning could cause these differences, as the ventral position can significantly increase IOP (Hvidberg et al. 1981). Supine body position has been shown to increase IOP in humans, but Broadwater et al. (2008) showed that this was not the case in conscious dogs in sternal recumbency. Kanda et al. (2015), who administered similar doses of medetomidine (0.01 and 0.02 mg/kg), did not observe significant changes in IOP.

Interestingly, Jaakola et al. (1992) described a decrease in IOP after administration of dexmedetomidine in humans. However, they used a lower dose of dexmedetomidine (0.0006 mg/kg). Artigas et al. (2012) used a dose of dexmedetomidine similar to what was administered in our DEX-5 group, and reported similar results at T10. However, at T20 they detected a significant decrease in IOP. In our study, the IOP at T20 was measured after anaesthesia induction by propofol and connection to an anaesthetic breathing system. Therefore, IOP was under the influence of propofol and isoflurane. Batista et al. (2000) described insignificant changes in IOP after propofol administration, which is in agreement with our findings. Hofmeister et al. (2008) reported an increase in IOP after propofol administration, which runs counter to our observations. However, the insignificant IOP changes after propofol administration observed in the present study were probably due to the lower dose of

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propofol used. Our and Hofmeister's findings are not in agreement with those reported in humans, where propofol decreased IOP by 40% (Mirakhur et al. 1987).

Effects of separate administration of isoflurane on IOP in dogs have not yet been reported. Almeida et al. (2004) used sevoflurane and desflurane in dogs after propofol induction. Measurements showed normal IOP values in both groups, and IOP values did not differ between groups. Yoshitake et al. (1992) described the effects of isoflurane or sevoflurane on IOP in adults. After sevoflurane or isoflurane administration IOP was reduced up until 30 min without significant differences between groups. On the basis of these facts we assume similar insignificant effects of isoflurane on IOP in dogs. Therefore, the observed changes in IOP in the present study can be attributed above all to different premedication.

The measurement at T20 was performed in all dogs after endotracheal intubation. It is reported that orotracheal intubation increases IOP (Warner et al. 1997) due to the patient coughing during laryngeal stimulation, an increase in extraocular muscle tone, or an increase in sympathetic tone. However, this increase in IOP is only transient and, therefore, was not observed in the current study.

Intraocular pressure can be influenced by patient recumbency (Hvidberg et al. 1981; Broadwater et al. 2008). All of the patients in the current study were positioned in lateral recumbency. There is no information relating specifically to the effects of a lateral recumbent position on intraocular pressure, but it is known that intraocular pressure is affected by extraocular muscle tone, scleral rigidity, and aqueous humour production and drainage (Brunson 1980). Aqueous humour production and drainage, in turn, can be influenced by changes in central nervous system output, blood pressure and venous drainage (Cunningham and Barry 1986). In the present study no significant differences in arterial blood pressure within and between groups were observed. This is unexpected as Murrell and Hellebrekers (2005) reported an initial increase in systemic blood pressure after alpha-adrenoceptor agonist administration. The stable blood pressure measurements in the present study may be due to the lower dose of medetomidine and dexmedetomidine administered, which would explain why there was no significant variation in IOP. Some studies reported a decrease in mean arterial pressure after propofol induction (Brussel et al. 1989; Lerche et al. 2000), whereas others have shown no effect (Quandt et al. 1998). In the present study a change in blood pressure after propofol administration (T20) was not observed and, this again, may be due to the lower dose of propofol administered.

Pypendop and Verstegen (1998) used doses of medetomidine (10 and 20 mg/kg) similar to the ones administered in the present study. They detected a significant transient increase in arterial blood pressure, which returned to baseline or lower within 10 min after medetomidine administration. It is possible that the rise in arterial blood pressure was missed in the present study because the first measurements after medetomidine or dexmedetomidine administration were taken after 10 min. An increase of arterial blood pressure causes a decrease in IOP. This is transient due to vasoconstriction of the choroidal and retinal vessels, which reduce intraocular blood volume (Riva et al. 2011).

Intraocular pressure can also be affected by changes in the partial pressure of oxygen (PaO_2) and partial pressure of carbon dioxide ($PaCO_2$) in arterial blood (Cunningham and Barry 1986). Neither PaO_2 nor $PaCO_2$ were measured in the present study. We monitored SpO_2 and $EtCO_2$ only, which is not comparable to PaO_2 or $PaCO_2$. However, SpO_2 and $EtCO_2$ values were in physiological ranges without differences between groups. Therefore, O_2 or CO_2 levels would not have influenced IOP.

Verbruggen et al. 2000 suggested that peripheral alpha-2 receptors exist in the eye and may be involved in the physiological regulation of IOP, which is not regulated by miosis only. Miosis was not accompanied by changes in IOP induced by medetomidine administration. The suppression of sympathetic activity induced by alpha-2 adrenoceptor agonists inhibits constriction of the iris dilator muscle, which is innervated primarily by sympathetic nerves. However, inhibition of the iris dilator muscle induced by alpha-2 adrenoceptor agonists does not induce miosis; it only inhibits mydriasis (Kanda et al. 2015). Pupillary constriction increases aqueous humour outflow from the anterior chamber, which decreases IOP (Gelatt and Brooks 1999). Nevertheless, in the present study, a significant decrease in PS was observed when compared with the insignificant changes in IOP. This result suggests that the changes in PS were not related to IOP, which is in agreement with the

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hypotheses and findings presented by Kanda et al. (2015) and Verbruggen et al. (2000).

In small animals, alpha-2 adrenoceptors can induce vomiting. Sinclair (2003) observed vomiting after medetomidine administration in 8–20% of dogs. Smith and Walton (2001) described increases in IOP in 45% of children who vomited. Changes in IOP in vomiting dogs have not yet been reported. However, we assume similar changes in IOP in vomiting dogs. In our study, we did not observe vomiting; therefore, an influence of vomiting on IOP can be excluded. In summary, our study shows that comparable doses of dexmedetomidine or medetomidine induce significant reductions in PS, HR and f_R, however, without a significant influence on IOP or SAP and DAP. Both alpha-2 adrenoceptor agonists, at the doses used in the present study, are a good option for ocular examination or surgical procedures in dogs when a specific control of IOP is required. However, these must be used in combination with mydriatics in procedures requiring complete dilation of the pupil.

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