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Research Article

Anaesthesia monitoring in ASA II dogs: A comparison of dexmedetomidine and acepromazine as premedication

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Abstract

Dexmedetomidine and acepromazine are potent muscle relaxant and tranquillizer respectively. Both the drugs lack comparative monitoring studies in dogs in India. The present study was aimed to compare the effects of dexmedetomidine and acepromazine as premedication agents in ASA II dogs undergoing propofol-induced and isoflurane-maintained anaesthesia. The clinical study included 13 dogs. Group D dogs (n=7) received dexmedetomidine 10 mcg/kg and Group A dogs (n=6) received acepromazine 0.05 mg/kg intramuscularly along with butorphanol 0.2 mg/kg and glycopyrrolate 0.01 mg/kg as pre-anaesthetic drugs. The top-up required, post-induction apnea, heart rate, respiration rate, temperature, blood pressure, mechanical ventilation, oxygen supplementation, isoflurane requirement, end tidal carbon dioxide, electrocardiography and hemato-biochemical parameters were recorded. The data was analyzed using student *t*-tests. Post-induction apnea was common in Group D and isoflurane requirement was higher in the Group A. The heart rate reduced significantly after premedication in both the groups, and was prominent in Group D. The blood pressure was significantly higher in the Group D after induction and decreased later, however, it decreased significantly after pre-medication in Group A and continued to fall. The recovery was faster in the Group D dogs, although the quality of recovery was similar in both groups. In conclusion, both dexmedetomidine and acepromazine are safe as pre-anaesthetic agents in ASA II dogs. Dexmedetomidine provides deeper sedation, anaesthetic sparing and faster recovery than acepromazine when combined with butorphanol and glycopyrrolate, with stable intraoperative parameters in ASA II dogs anesthetized with propofol and isoflurane.

Keywords: Acepromazine, Blood pressure, Butorphanol, Dexmedetomidine, Heart rate

Highlights

- Dexmedetomidine and acepromazine are safe as pre-anaesthetic agents in ASA II dogs
- Dexmedetomidine provides deeper sedation, anaesthetic sparing and faster recovery than acepromazine

INTRODUCTION

Premedication is an essential part of veterinary anaesthetic protocols. It is aimed at decreasing anxiety, easy handling, smooth induction, and decreasing induction doses. Selection of preanaesthetic agents greatly enhances perioperative recovery and patient safety, especially in companion animal practice. Dexmedetomidine is a highly selective α2-adrenoceptor agonist, is used extensively for its potent sedative and analgesic (Murrell & Hellebrekers, 2005). It decreases sympathetic tone through central inhibition of norepinephrine release, producing dose-related bradycardia and variable blood pressure responses. It induces sleep-like sedation, reduces anxiety, and has sympatholytic and anaesthetic-sparing effects with minimal respiratory effects (Mahmoud & Mason, 2015). Acepromazine is a phenothiazine derivative, has tranquilising effects due to dopamine receptor antagonism and peripheral α1-adrenergic blockade (Murrell & Hellebrekers, 2005). Although devoid of intrinsic analgesic activity, it yields consistent sedation and muscle relaxation (Steffey & Mama, 2007). Glycopyrrolate is an anticholinergic pre-anaesthetic used to counter bradycardia (Lemke et al., 1993). Butorphanol is a partial kappa and sigma receptor agonist, causing mild to moderate respiratory depression and transient heart rate alterations (Trim, 1983). Propofol a short-acting intravenous anaesthetic, is preferred for induction due to its rapid onset and smooth recovery, although it can cause dose-related respiratory and cardiovascular depression (Lerche et al., 2000). Isoflurane, used commonly for maintenance, is characterised by low solubility, low hepatic metabolism, and ease of titration

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but also causes peripheral vasodilation (Hall et al., 2001; Steffey & Mama, 2007). The American Animal Health Association (AAHA) recommends, ASA (American Society of Anaesthesiologist) classification in animals as well (McMillan & Brearley, 2013), with ASA II including the dogs and cats with mild systemic diseases and dogs with well compensated mild mitral valve degeneration may also be included.

This study compares dexmedetomidine and acepromazine as premedications in ASA II dogs induced using propofol and maintained on isoflurane.

MATERIALS AND METHODS

A total of 13 client-owned dogs classified into ASA II were included in the study. The dogs were undergoing surgery for the procedures: ear hematoma (n=2), nasal lacrimal abscess (n=1), patellar luxation (n=1), cherry eye (n=2), femur head ostectomy (n=1), eye extirpation (n=2), elbow hygroma (n=2), total ear canal ablation (n=1), entropion (n=1). The dog's age ranged from 0.4 to 7.5 years and weighed between 10 and 60 kg. The dogs were advised 12 hours off feed and 6-8 hours off water prior to surgery. The dogs were randomly divided into two groups: Group D (n=7): Received a combination of dexmedetomidine (5-10 µg/kg IM), butorphanol (0.2 mg/kg IM), and glycopyrrolate (0.01 mg/kg IM), and Group A (n=6): Received a combination of acepromazine (0.05 mg/kg IM), butorphanol (0.2 mg/kg IM), and glycopyrrolate (0.01 mg/kg IM).

All pre-medications were administered intramuscularly in a single syringe into the lumbar muscles. After 10-15 minutes, the anaesthesia was induced with propofol (2–4 mg/kg IV to effect) and was maintained on isoflurane in 100% oxygen delivered via an endotracheal tube. The heart rate (HR), respiratory rate (RR), rectal temperature (RT), and blood pressure (BP) were monitored and recorded at baseline, after premedication, following induction, and at 5-minute intervals throughout the anaesthetic period. The peripheral oxygen saturation (SpO_2), end-tidal carbon dioxide ($EtCO_2$), and reflexes-including pedal withdrawal, palpebral response, and jaw tone-were assessed and recorded during anaesthesia to evaluate anaesthetic depth.

The blood samples were collected at four time points: prior to premedication (baseline), after premedication, during maintenance of anaesthesia, and during recovery. Electrocardiography (ECG) was also performed and recorded at these same time points to evaluate cardiac rhythm and to detect any perioperative arrhythmias.Anaesthetic monitoring was done using a Philips IntelliVue multiparameter monitor and a BPL ECG machine. At the end of surgery, the dogs were disconnected from isoflurane and were kept on 100% oxygen for 5 minutes before shifting to recovery room. The quality of recovery was assessed by coughing, time of sternal recumbency and ability to stand.

Statistical analysis: The data was statistically analysed using Microsoft Excel 2021. Unpaired *t*-tests were used to compare parameters between groups. Results were considered statistically significant at $p<0.01$ and $p<0.05$.

RESULTS

Induction parameters: The duration of anaesthesia was non-significantly more in Group A which was due to surgical procedure and was independent of anaesthetic agent. Top-up anaesthesia was required in one dog of each group (Table 1). All the dogs showed relaxed jaws, except one of Group A. Post-induction apnea was present in 4 dogs (57.14%) of Group D and 3 (50%) of Group A. The incidence of apnea was slightly more in Group D due to synergistic effect of propofol and dexmedetomidine combined with depression on the respiratory system. Overall, the dogs in Group D showed better induction compared to those in Group A.

Maintenance parameters

Isoflurane: The Isoflurane requirement was non-significantly higher in Group A dogs from the start of anesthesia as compared to Group D (Fig. 1a).

Table 1. Table showing the mean±SE induction parameters of ASA II dogs

	Duration of anesthesia (min)	Top up (mL)	ET size (ID)	Jaw tone (0=relaxed) (1=not relaxed)	Apnea (0=absent 1=present)
Group D (n=7)	32.14±6.44	One dog, 14.28%	8.86±0.47	0=7	0=3, 42.86% 1=4, 57.14%
Group A (n=6)	57±9	One dog, 16.67%	8.17±0.65	0=5, 83.33% 1=1, 16.67%	0=3, 50% 1=3, 50%

Oxygen flow rate: The mean oxygen flow rate was constant throughout the surgery in both the groups except for 2 dogs of Group A, where a higher flow rate was required.

Heart rate: The heart rate of the dogs of Group D was significantly ($p=0.03$) lower than that of Group A at presentation and reduced to almost 50% after the pre-anesthesia while it reduced to almost 14% for Group A (Fig. 1b). The drop in heart rate from 0 to 10 minutes was significant for both the groups ($p=0.00087$ for Group D and $p=0.035$ for Group A). Throughout the anesthesia the heart rate of Group D dogs was significantly lower that of Group A dogs. After 10 minutes of switching off the Isoflurane anesthesia, the heart rate almost came to normal in Group A while it took 12-13 minutes in Group D. At almost 15 minutes in recovery, the heart rate of both the groups' dogs, shoot more than the pre-anesthesia stage.

Rectal temperature: No significant change in the temperature was recorded at different time intervals among the dogs. But within the group a significant drop in the temperature was recorded in Group D after pre-anesthetic administration at $p<0.01$. However, for Group A dogs, the temperature drop was not significant for the first 20 minutes but later became significant up to 60 minutes. Dexmedetomidine causes a higher drop in temperature than acepromazine but Isoflurane also adds to drop in temperature. In recovery, the temperature could not come to pre stage up to 15 minutes in both the groups.

Respiration rate: The respiration significantly dropped in both the groups after induction (at 20 min) (when comparing 10-20 min) as few dogs also showed apnea at induction (Fig. 1c). At recovery, the respiration rate increased with duration but took more time to come to pre stage in Group D as compared to Group A.

Mucous membrane (MM)/Capillary refill time (CRT) (in secs): MM/CRT was recorded to evaluate blood perfusion to tissues. MM was a pink/roseate colour, and CRT was <2 seconds in all the cases of both the groups for the anaesthetic duration. In one dog of Group A had CRT 0 seconds during recovery.

Eyeball position: The eyeball position was ventromedial in all the dogs of both the groups during anesthesia except for one dog of Group D at 0 min. During recovery most of the dog's eyeball returned to central (light plane) within 5 minutes of switching off the Isoflurane.

Palpebral reflex: The palpebral reflex was absent in all the dogs of Group A at all time intervals, while it was present in 2 dogs of Group D at 0 and 10 minutes. However, palpebral reflex returned back within 5 minutes of switching off the Isoflurane in both the groups.

Pedal reflex: The pedal reflex was absent in all the dogs of both groups except one dog of Group D at 0 minutes. During recovery, the pedal reflex returned back in 3 dogs of Group D and one dog of Group A at 5 minutes. The reflex returned in most of the dogs of Group D in 10 minutes.

Jaw tone: The jaw tone was absent in all the dogs of both the groups during anesthesia except for one dog of Group D at 10 minutes, indicating presence of adequate depth and good level of muscle relaxation throughout the period of anaesthetic maintenance in both the protocols.

Oxygen haemoglobin saturation (SpO_2): No significant change in the oxygen haemoglobin saturation was recorded in between the two groups at different time intervals but when comparing the SpO_2 between 0 and 10 minutes in Group D, the SpO_2 dropped significantly due to initial apnea incidence.

Blood pressure

Systolic arterial pressure (SAP): After induction and 10 minutes into the Isoflurane the Group A dogs showed a significantly reduced SAP in comparison to Group D (Fig. 1d). The systolic arterial pressure readings at the different intervals in Group D display a non-significant decreasing pattern, though it was less at presentation also. An increase in SAP was recorded at 50 minutes in Group D and 60 minutes in Group A.

Diastolic arterial pressure (DAP): The DAP was significantly low in Group A dogs in comparison to Group D dogs at 20, 30 and 40 minutes (i.e after induction and for few minutes further). The DAP in Group D increased after pre-anesthetic and during Isoflurane and reduced back to normal in the end while in Group A, the DAP mostly reduced after pre-anesthesia and further during Isoflurane (Fig. 1e).

Mean arterial pressure (MAP): Similar to the DAP, the MAP also reduced significantly in Group A at 20, 30 and 40 minutes. The MAP increased after pre-anaesthesia and further during Isoflurane in Group D while decreased in Group A (Fig. 1f).

End tidal carbon dioxide ($EtCO_2$): No significant change in the $EtCO_2$ values were found among the groups and remained within the normal range throughout anaesthesia.

Intermittent positive pressure ventilation (IPPV): The IPPV was required in one dog each of both the groups after induction (during induction apnea).

Electrocardiography (ECG): Out of 7 dogs of Group D, one dog showed inverted T at base ECG and 3 dogs showed sinus bradycardia after induction. While in Group A, 2 dogs showed inverted T at base and after induction and during anaesthesia. One dog among these showed sinus tachycardia during anaesthesia.

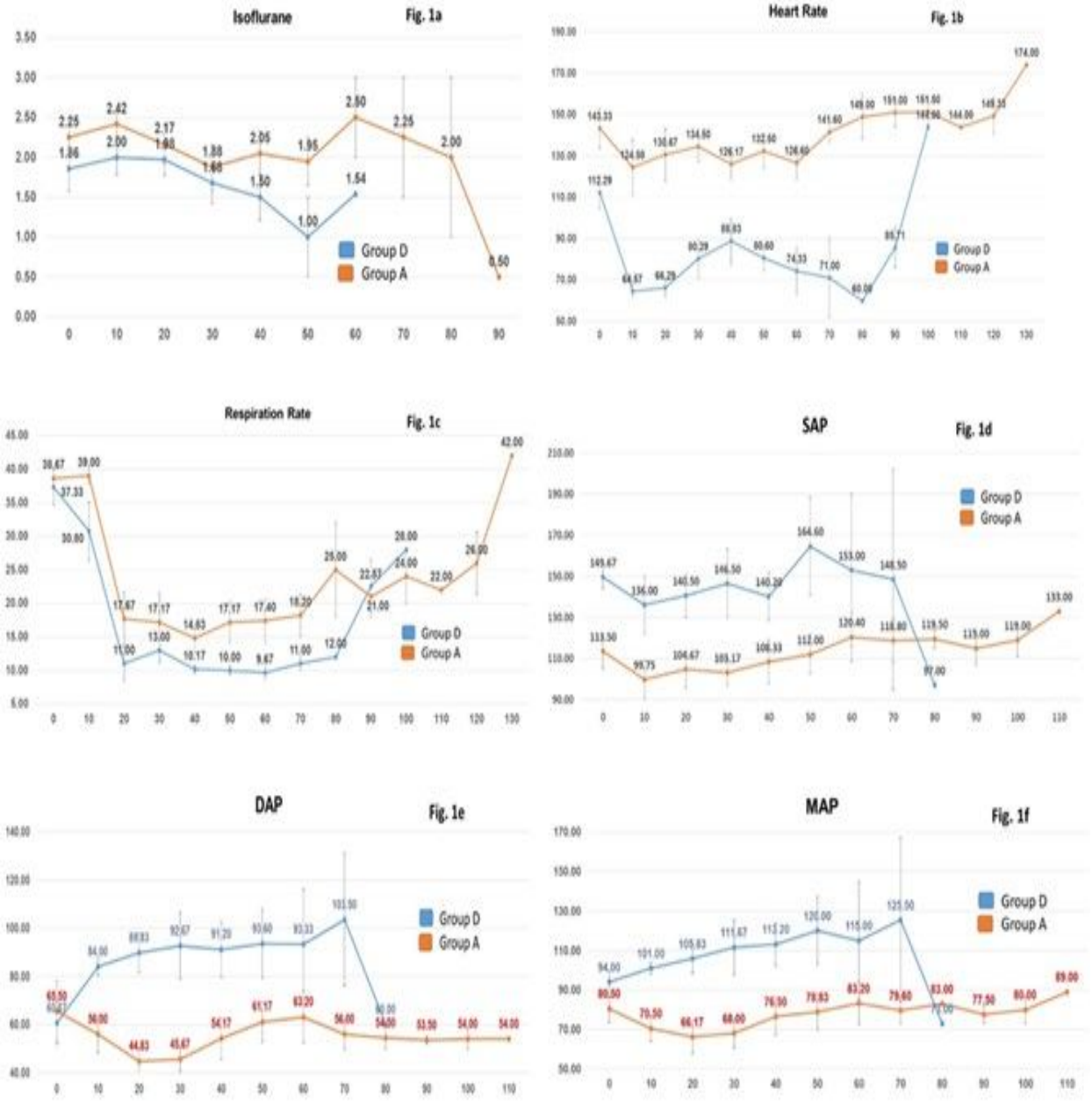

Fig. 1. Line graphs showing the comparative demonstration of Mean±SE values of Isoflurane % (a), Heart rate (b), Respiration rate (c), SAP (d), DAP (e), MAP (f) in Group D and Group A dogs at various time interval

Haemato-biochemical parameters: The haemoglobin (Hb) reduced after pre-medication in Group D but not in Group A, however it reduced in both the groups during maintenance, which was significant in Group A as compared to Group D (Table 2). The packed cell volume (PCV) also reduced in both the groups and was significant during maintenance between the groups where, it was more reduced in Group A. The total leukocyte count (TLC) was found to be reduced during anaesthesia and recovery but not significantly. The platelet values declined in both groups with all consecutive intervals, and was significant after pre-medication. The alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphatase (ALKP), blood urea nitrogen (BUN) and creatinine showed a reduction in values after premedication and further during maintenance but were not significant between and within the groups.

Recovery parameters: None of the dogs showed coughing in any of the groups. Three dogs of Group D showed sternal recumbency before recovery while all were in lateral recumbency in Group A. After recovery, Group A showed sternal recumbency in 3 dogs while rest were lateral only. While in Group D, 5 dogs were in sternal recumbency and 2 were standing after recovery. Recovery was smooth in both groups, although Group D dogs regained sternal recumbency and ambulation faster. Mild bradycardia was noted in some dogs in Group D during early recovery, but no significant complications were observed in either group.

Both dexmedetomidine and acepromazine are safe as pre-anaesthetic agents in ASA II dogs. Dexmedetomidine provides deeper sedation, anaesthetic sparing and faster recovery than acepromazine when combined with butorphanol and glycopyrrolate, with stable intraoperative parameters in ASA II dogs anesthetized with propofol and isoflurane.

Table 2. Table showing the mean haemato-biochemical values for ASA II dogs

Group	Pre-anaesthetic	After Pre-anaesthetic	During Maintenance	During Recovery
		Haemoglobin		
Group D	12.6±0.52	11.8±0.84	10.59±0.45*	10.57±0.49
Group A	9.43±2.03	9.5±1.51	7.37±1.17*	7.52±1.22
		TLC		
Group D	13900±1375.98	13471.43±1043.74	12228.57±1042.14	12271.43±972.9
Group A	15900±4956.48	15466.67±2688.7	10566.67±1826.41	10066.67±1366.18
		PCV		
Group D	34.81±0.77	31.4±1.96	28.31±1.14*	29.09±1.17*
Group A	27.55±4.31	25.98±3.43	20.85±2.71*	21.12±2.91*
		Platelets		
Group D	237333.33±19484.47	173142.86±34483.56*	184428.57±30969.92	177857.14±30936.52
Group A	345500±43016.47	281166.67±33044.83*	256333.33±40729.73	210550±45619.39
		ALT		
Group D	30.5±3.5	28.14±1.56	26.57±2.61	26.43±1.31
Group A	53±34	29.33±7.14	24.33±6.98	23.83±6.26
		AST		
Group D	44.5±2.5	38.43±4.07	33.57±3.95	35.43±4.87
Group A	31±3.46	51.67±7.27	34.67±2.39	32.33±1.41
		ALKP		
Group D	81.5±33.5	61.57±9.2	61.14±10.01	64.29±13.69
Group A	235.67±87.17	84.83±49.72	91.67±44.94	77.5±36.06
		BUN		
Group D	17.67±3.08*	12.57±1.43	12.57±1.7	12.14±1.58
Group A	9.2±1.46*	8.67±1.69	9±1.69	8.5±1.5
		Creatinine		
Group D	0.96±0.08	0.9±0.1	0.86±0.1	0.86±0.09
Group A	0.78±0.12	0.7±0.13	0.77±0.09	0.75±0.08

*Level of significance ($p<0.05$)

DISCUSSION

The Isoflurane has a sparing effect on dexmedetomidine (Aantaa et al., 1997). Dexmedetomidine reduces the heart rate more than acepromazine (Martin-Flores et al., 2019; Pan et al., 2021). Dexmedetomidine and butorphanol when used together had a significant depressant effect on heart rate (Barletta et al., 2011). Dexmedetomidine also causes a concentration-dependent decrease in heart rate (Pypendop et al., 2011). This decreasing trend may also be due to the cardiopulmonary suppressing effect of both dexmedetomidine and propofol combined. The reduction in heart rate observed was not life-threatening at any interval in the present study. Both dexmedetomidine and acepromazine dogs showed absent pedal and jaw tone reflex during anaesthesia, with acepromazine dogs having better muscle relaxation. The muscle relaxation property of dexmedetomidine has not been discussed in literature but there has been a report on the role of dexmedetomidine in the management of post-operative muscle spasm (Trinh & Villaluz, 2022). An inverted T wave could be due to normal physiological variations in the canines or underlying cardiac pathology (Mukherjee et al., 2015).

Acepromazine, increases heat loss, thereby reducing the body temperature by vasodilation (Grasso et al., 2015). Respiratory depression has been reported post propofol induction, leading to a decrease in respiration rate (Keegan & Greene, 1993) and dexmedetomidine may have some synergistic effect on respiratory depression with propofol.

A transiently higher mean and systolic arterial blood pressure just after administration of dexmedetomidine is reported (Pan et al., 2021). Acepromazine causes arterial hypotension through α_1-adrenergic receptors in isoflurane-anaesthetised dogs (Martin-Flores et al., 2019).

In conclusion, both dexmedetomidine and acepromazine are safe as pre-anaesthetic agents in ASA II dogs. Dexmedetomidine provides deeper sedation, anaesthetic sparing and faster recovery than acepromazine when combined with butorphanol and glycopyrrolate, with stable intraoperative parameters in ASA II dogs anesthetized with propofol and isoflurane.

Conflict of interest: All the authors declare to have no conflict of interest.

Author's contribution: MS: Recording of data writing and analysing it; VS: Scrutinizing the work, analysis and writing; NK, NS: Anaesthesiologists in clinical cases.

Data availability statement: Data set generated during research need to be available from the corresponding author upon reasonable request.

Ethical statement: This study was approved by the vide letter no. V-11011(13)/2/2024-CPCSEA-DAFD from the Committee for the Purpose of Control and Supervision of Experiments on Animals, New Delhi, for a period of one year.

Use of artificial intelligence tools: No use of artificial intelligence tools has been done.

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