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RESEARCH ARTICLE Indian Journal of Animal Research

Sedative, Analgesic and Anaesthetic Evaluation of Propofol in Combination with Butorphanol, Dexmedetomidine and Acepromazine in Clinically Healthy Dogs

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10.18805/IJAR.B-5446

ABSTRACT

Background: Propofol is intravenous short acting general anaesthetic most popular drug for induction and maintenance of anaesthesia in veterinary practice due to its rapid induction and smooth recovery. However, as sole general anaesthetic, it is unsatisfactory because of its poor analgesic property and causing hypotension and apnoea. Therefore, it commonly used in combination with opioid or alpha 2 agonist to minimize the untoward effects. The aim of the present study was to evaluate the sedative, analgesic and anaesthetic efficacy of propofol in combination with butorphanol, dexmedetomidine or acepromazine premedication in dogs.

Methods: The present study was conducted on 18 healthy adult dogs of either sex and randomly divided into three groups (ButP, DexP and AceP) with six animals in each. Glycopyrrolate @ 0.02 mg/kg was administered intramuscularly 10 minutes prior to the anaesthetic administration in all the dogs. The animals of group ButP, DexP and AceP were premedicated intramuscularly with butorphanol @ 0.3 mg/kg b.wt., dexmedetomidine @ 10 µg/kg b.wt. and acepromazine @ 0.4 mg/kg b.wt. respectively. Ten minutes later propofol was injected intravenously at a dose of 7 mg/kg b.wt. to induce general anesthesia. The following clinical parameters were recorded *viz*., onset of sedation, onset of anaesthesia, degree of analgesia, extent of muscle relaxation, duration of anaesthesia and complete recovery. All the data were analyzed using SPSS v 25.0 statistics software program and presented as mean±Standard Error.

Result: The onset of sedation and induction of anaesthesia was earlier in group DexP followed by AceP and ButP. Group DexP exhibited significantly ($P<0.05$) longer duration of anaesthesia and complete recovery than groups ButP and AceP. All the groups had excellent analgesia; however animals of group DexP showed longer duration of analgesia. All the reflexes were abolished completely with longer duration of muscle relaxation in group DexP. The above anaesthetic study suggests that propofol in combination with dexmedetomidine; butorphanol or acepromazine can be safely used for inducing surgical anaesthesia in dogs. However, dexmedetomidine-propofol combination produced prolonged duration of anaesthesia with better quality suitable for long surgical procedure in dogs.

Key words: Acepromazine, Analgesic, Anaesthetic, Butorphanol, Dexmedetomidine, Glycopyrrolate, Propofol, Sedative.

INTRODUCTION

General anaesthesia is commonly done, especially in dogs, to assist routine surgical operations and also to complete life-saving surgeries. Sleep, amnesia, muscle relaxation and analgesia are all desirable effects of an anaesthetic. However, as single agent cannot generate all of these effects, therefore combinations of drugs are used known as balanced anaesthesia (Thurmon and Short, 2007). Various investigators have observed that alpha-2 adrenoceptor agonists (xylazine, medetomodine, detomidine and dexmedetomidine), phenothiazines (chlorpromazine, prochlorperazine, trifluprомazine and acepromazine) and non-barbiturate (propofol) produce sedation and analgesia (Hall *et al.,* 2001; Ahmad *et al.,* 2013 and Rangel *et al.,* 2020). Propofol (2-6 di-isopropyl-phenol) is commonly used in human anaesthetic practice and is also becoming popular for induction and maintenance of anaesthesia in veterinary practice particularly in dogs and cats and is not related to barbiturates, euganols, or steroid anaesthetics. Propofol anaesthesia is characterized by rapid onset, lack of cumulative effects and absence of excitatory effects on induction, during maintenance and recovery (Bufalari *et al.,* 1997). However, propofol as sole general anaesthetic is unsatisfactory because of its poor

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How to cite this article: Dewangan, R., Pal, S., Sharda, R., Khutey, B.B., Kumar, I. and Kurrey, L. (2024). Sedative, Analgesic and Anaesthetic Evaluation of Propofol in Combination with Butorphanol, Dexmedetomidine and Acepromazine in Clinically Healthy Dogs. Indian Journal of Animal Research.1-10. doi:10.18805/IJAR.B-5446

Submitted: 15-07-2024 **Accepted:** 29-11-2024 **Online:**30-12-2024

analgesic property. Consequently, for major surgical procedures must be combined with potent analgesic drugs such as opioids and alpha 2 agoinsts (Redondo *et al.,* 1999). The mechanism of action of propofol is exactly unknown but its indues depression by enchancing the effects of the inhibitory neurotransmitter GABA and decreasing the metabolic activity of the brain (Concas *et al.,* 1991).

Glycopyrrolate is a synthetic quaternary ammonium compound, anticholinergic with no central effects and is about five times more effective as atropine sulphate. It has a powerful and long-lasting antisialagogue effect. Glycopyrrolate inhibits cholinergic transmission by blocking peripheral muscarinic receptors. Butorphanol is a synthetic opioid and is a partial agonist at μ and an agonist at kappa opioid receptors. Butorphanol is mainly utilised as an analgesic and sedative in cats and dogs (Marini *et al.,* 1992). Dexmedetomidine an alpha-2 adrenergic agonist is an active optical enantiomer isolated from the racemic compound medetomidine. It is mainly used for sedation, analgesia and as an anaesthetic adjunct in operations requiring complete intravenous anaesthesia to minimise anaesthetic requirements (Miller, 2009). Acepromazine is a phenothiazine tranquilizer that depresses the reticular activating system and inhibits dopamine receptors in the CNS, resulting in drowsiness. Because of the dopamine inhibition in the chemoreceptor trigger zone, it also possesses antiemetic, antihistaminic, antiarrhythmic and antishock characteristics (Turi and Muir, 2011). The aim of this study was to evaluate the sedative, analgesic and anaesthetic efficacy of propofol in combination with butorphanol, dexmedetomidine or acepromazine in clinically healthy dogs.

MATERIALS AND METHODS

The present work was carried out during January to December 2021 in confinement of Department of Veterinary Surgery and Radiology at College of Veterinary Science and A.H., Anjora, Durg (C.G.) India.

Anaesthetic design

The study was conducted on 18 clinically healthy dogs of either sex weighing between 10 to 20 kg body weight and were randomly divided into three groups *viz.*, ButP, DexP and AceP, comprising of 6 animals in each. All dogs were dewormed with Praziplus (Albendazole 300 mg with Praziquental 25 mg) Tab. @ 1 Tab. / 10 kg body weight orally fifteen days before the start of anaesthestic study. The animals were fasted overnight and the drinking water was withheld for 4 hours before the anaesthetic trial. The animals were kept under uniform feeding and managemental practices throughout the experiment. Ten minutes prior to the anaesthetic administration, all dogs were administered with glycopyrrolate @ 0.02 mg/kg b.wt. intramuscularly. The animals of group ButP, DexP and AceP were premedicated intramuscularly with butorphanol @ 0.3 mg/kg b.wt., dexmedetomidine @ 10 µg/kg b.wt. and acepromazine @ 0.4 mg/kg b.wt. respectively. After administration of preanesthetic, the animals were kept undisturbed in a calm environment to record the onset of sedation. Induction of anaesthesia was done with propofol @ 7 mg/kg b.wt. intravenously in all the animals until the pedal reflex abolished and dogs were intubated with suitable endotracheal tube (4.5 to 8.5 OD mm) with guidance of laryngoscope and then subsequently various observations were recorded upto 120 mins.

Parameters studied

Evaluation of sedation, analgesia and anaesthesia was done on the basis of Onset of Sedation (Minutes), Induction of Anaesthesia (Minutes), Duration of Anaesthesia (Minutes), Quality of Anaesthesia and Recording of various reflexes and responses. The quality of anaesthesia was recorded on a scale 1 to 4 where 1 represents poor anaesthesia, 2-fair anaesthesia, 3-good anaesthesia and 4-excellent anaesthesia. The degree of analgesia was scored (0-3) by pinching of the inter-digital skin of the foot and vigorous squeezing and twisting or pinching of digit or pad. Jaw relaxation, palpebral reflex, pedal reflex, response to intubation and anal sphincter relaxation were recorded at 0 (base value), sedation, 5 min after sedation, after induction and at 5, 15, 30, 45, 60 and 120 min after administration of propofol in all the groups. Relaxation of the jaw was taken as a measure of muscle relaxation. It was evaluated by observing the resistance to opening of the jaw while pulling apart the lower and upper jaws. It was scored from 0-3 scale. The status of the palpebral reflex was recorded as a measure of depth of sedation at same time interval as for relaxation of jaw reflex. It was measured by observing a blink of the eye lids on touching the area around the medial canthus of the eyes with the index finger, scored from 0-3. The status of the pedal reflex was recorded as a measure of the depth of analgesia. It was assessed by observing the withdrawal reflex to the pinching of the inter-digital skin of the hind foot of the animal. The response of animal to pedal pinching was graded from 0-3. Response to intubation was recorded to assess the feasibility of intubation. If the animal allowed easy intubation, the endotracheal tube was left *in situ* and return of the laryngeal reflex was recorded when the animal started coughing and graded from 0-4. Relaxation of anal sphincter was graded from 0 to 3 depending upon the extent of relaxation (Table 1).

Additional parameters like recovery time (Minutes), time to extubation (Minutes), head rightening (Minutes), sternal recumbency time (Minutes), standing time (Minutes) and complete recovery time (Minutes) were also recorded. Complications *viz.* nausea, vomition, salivation, lacrimation, muscle twitching *etc*. were recorded during and after anaesthesia in each group of animals if observed.

Statistical analysis

The data collected were statistically analysed using analysis of variance (ANOVA) and Duncan's multiple range tests (DMRT). The mean and standard error of the recorded values were calculated. Comparison within group and

between groups was made using SPSS v25 statistics software program and data were presented as Mean±S.E. The subjective data generated from the scoring of various parameters were analysed using the Kruskal Wallis Test. Statistically significant differences were considered at 5 percent level (5%).

RESULTS AND DISCUSSION

Onset of sedation

Decrease in spontaneous activity in all the animals was observed after administration of preanaesthetic agent. However, marked and early sedation with lowering of the head was observed in animals of group DexP as compared to group ButP and AceP where mild sedation occurred as depicted in Table 2. The onset of sedation and recumbency was significantly ($P<0.05$) earlier in Group DexP than other groups, due to the onset of action of dexmedetomidine owing to its lipophilic property (Amarpal *et al.,* 1996). All the animals remained conscious but were unable to stand when disturbed. No case of salivation and vomiting were observed in all the three groups. Comparison between groups revealed rapid onset and profound sedation after administration of dexmedetomidine in group DexP. There was excellent sedation in group DexP as compared to group ButP and AceP while both groups showed mild sedation. The sedative/hypnotic effects of dexmedetomidine are mediated through pertussis-sensitive inhibitory G protein in locus coeruleus resulting in hyperpolarization and reduced nerve conduction (Kuusela *et al.,* 2000). The faster onset of sedation was recorded with dexmedetomidine in the present study as also confirmed by various workers (Ahmad *et al.,* 2013 and Verma *et al.,* 2021). The use of premedicants in the present study was aimed in relieving anxiety in order to smoothen anaesthetic induction, maintenance and recovery phase.

Induction of anaesthesia (minutes)

Induction of anaesthesia was rapid, smooth and free from any untoward reactions like struggling and paddling in all the three groups. Shorter onset of anaesthesia was observed in group DexP (0.49±0.03 min.) as compared to group ButP (0.55±0.02 min.) and AceP (0.53±0.02 min.). Induction of anaesthesia was quicker in animals premedicated with dexmedetomidine as compared to that premedicated with butorphanol or acepromazine. This might be due to the effect of dexmedetomidine which produces sufficient degree of sedation prior to induction with propofol. Rapid onset of anaesthesia was recorded in all the three groups in the present study which might be due to the high lipid solubility of propofol and ability to rapidly cross blood-brain barrier. Propofol is rapidly redistributed from the brain to other tissues and is also efficiently eliminated from plasma by hydroxylation, which explains its short action and rapid recovery. During propofol anaesthesia, there was rotation of eyeball in rostroventral position in light to moderate surgical anaesthesia. At the

Table 1: System of recording of various reflexes and responses (adapted and modified after Amarpal *et al.,* 1996).

Parameters	0	1	2	3	4
Analgesia	Strong reaction to pinching of the inter-digital skin of the foot	Weak responses to pinching of the inter-digital skin of foot	Occasional response to pinching of the inter-digital skin of the foot	No response to pinching of the inter-digital skin of the foot	-
Relaxation of jaw	Not allowing to open the jaws	Resistant to opening of the jaws and would close quickly	Less resistance to opening of the jaws and would close slowly	No resistance and jaws would remain open	-
Palpebral reflex	Quick blink	Slow blink	Very slow and occasional blink	No blink	-
Pedal reflex	Strong withdrawal	Slow withdrawal	Occasional withdrawal	No withdrawal	-
Response to intubation	Do not permit entry of tube into the mouth	Allow entry but will chew	Allow deeper entry but will cough	Difficult incubation with coughing	Easy intubation without coughing
Anal sphincter relaxation	Complete anal relaxation	Moderate anal relaxation	Mild anal relaxation	Absence of anal relaxation	-

end of the study, the position of eyeball was central which might be due to loss of the tone of the eye muscles and increased depth of anaesthesia (Hall *et al.,* 2001). Downward rotation of eyeball was observed after induction and during surgical anaesthesia with propofol and was in accordance with Anandmay *et al.* (2012) and Bayan *et al.* (2020) in dogs. All the reflexes were abolished completely after induction of propofol anaes thesia in all three groups suggesting that the surgical stage of anaesthesia had reached which are in agreement with earlier researchers under propofol anaesthesia in dogs (Kim *et al.,* 1999; Kandpal *et al.,* 2005; Dewangan *et al.,* 2010; Jena *et al.,* 2014 and Mate and Aher, 2019).

Record of various reflexes and responses

Analgesia was better in group DexP as compared to groups ButP and AceP after sedation with various preanaesthetics (Fig 1). Similar findings were also reported

Table 2: Time taken for occurrence of various signs associated with sedation after administration of butorphanol, dexmedetomidine or acepromazine in dogs in different groups.

Group (n=6)	Salivation (min)	Ptosis (min)	Nodding (min)	Head down (min)	Sternal recumbency (min)	Lateral recumbency (min)
ButP	-	2.25 ± 0.36^{Ba}	3.33 ± 0.42^{Bb}	6.0 ± 0.45^{Bc}	7.33 ± 0.22^{Bd}	9.0 ± 0.26^{Be}
DexP	-	2.25 ± 0.17^{Aa}	2.33 ± 0.21^{Ab}	4.0 ± 0.26^{Ac}	5.50 ± 0.22^{Ad}	6.83 ± 0.31^{Ae}
AceP	-	2.67 ± 0.21^{Ba}	4 ± 0.26^{Bb}	5.67 ± 0.42^{Bc}	6.83 ± 0.31^{Bd}	9.71 ± 0.31^{Be}

ABC - Value bearing different superscript vary significantly (P<0.05) among groups.

abcde - Value bearing different superscript vary significantly (P<0.05) within groups.

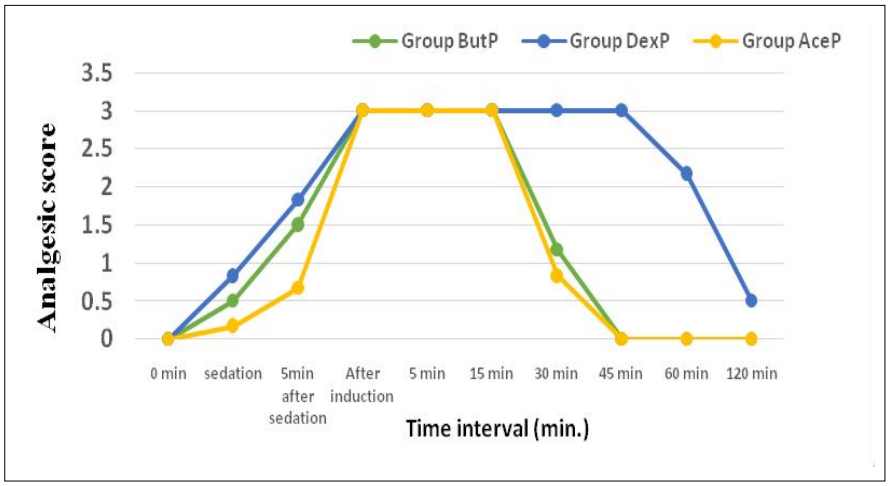

Fig 1: Effect on analgesic score at various time interval in different groups.

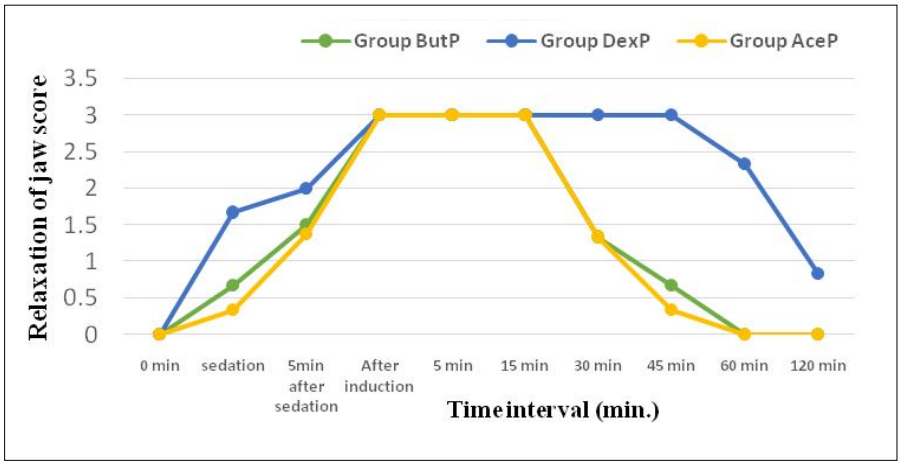

Fig 2: Effect on relaxation of jaw score at various time interval in different groups.

by Kumar *et al.* (2022 a) after intravenous dexmedetomidine in dogs. Jaw muscle relaxation was moderate in group DexP and mild in group ButP and AceP after sedation with premedicants (Fig 2). After induction with propofol, excellent analgesia and jaw muscle relaxation was present in all the groups but persisted for longer duration in group DexP. This could be attributed to synergistic interaction between the α-2 agonist and propofol. Analgesic action of dexmedetomidine is mainly through spinally and interruption of nociceptive pathways to the ventral root of the dorsal horn which reduces spinal reflexes (Talukder and Hikasa, 2009). Ahmad *et al.* (2013) documented that alpha-2 agonists produce profound muscle relaxation when used alone or in combination with opioid antagonists. Palpebral reflex decreased moderately in animals of group DexP and reflecting mild decrease in group ButP and AceP after sedation with preanaesthetics (Fig 3). After induction with propofol, there was complete loss of palpebral reflex in all the groups indicated by absence of eyelids blink which might be due to synergistic interaction of preanaesthetics like butorphanol, dexmedetomidine and acepromazine with propofol. Pedal reflex abolition was better in group DexP as compared to groups ButP and AceP after sedation with various preanaesthetics (Fig 4). There was complete loss of pedal reflex following induction with propofol in animals of all the groups suggesting that surgical stage of anaesthesia has been reached which persisted for longer period up to 45 min. in animals of group DexP which could be attributed to the synergistic interaction between the α-2 agonist and propofol. Post induction with propofol, analgesia was excellent in all the groups as there was no response to pinching of inter-digital skin of the foot which persisted for longer duration in group DexP. Endotracheal intubation was not possible in animals after sedation with butorphanol, dexmedetomidine and acepromazine (Fig 5). Easy intubation was possible without coughing in all the animals after induction with propofol anaesthesia. Loss of laryngeal reflexes along with endotracheal intubation persisted up to 15 min. in group ButP and AceP whereas intubation was maintained for longer period up to 60 min. in animals of group DexP which could be attributed to the synergistic

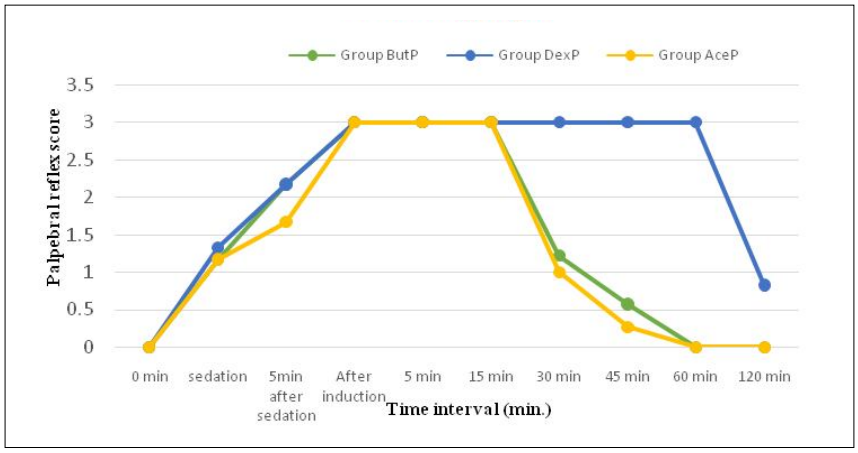

Fig 3: Effect on palperbral reflex score at various time interval in different groups.

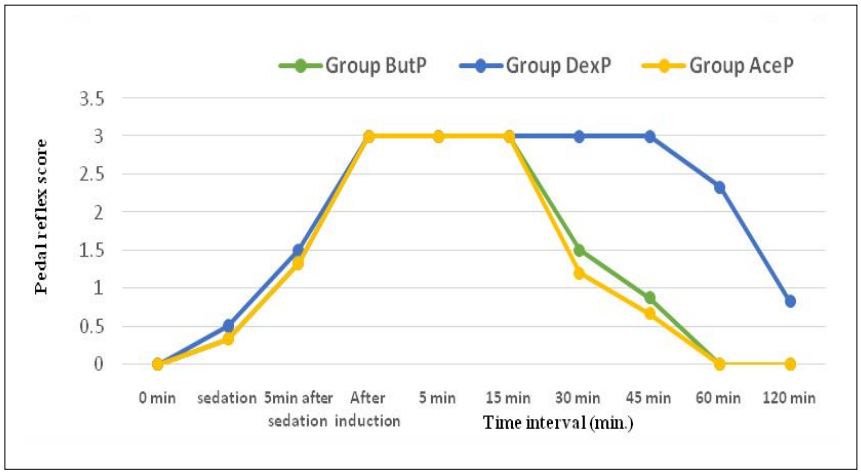

Fig 4: Effect on pedal reflex score at various time interval in different groups.

interaction between the α-2 agonist and propofol. Complete depression of laryngeal reflex occurred after propofol induction in the present study led to easy intubation by single attempt in all the groups might be due to combined action of preanaesthetics with propofol. Amengual *et al.* (2013) also noted rapid induction of anaesthesia facilitating easy endotracheal intubation in dogs anaesthetised with propofol. Mild anal sphincter muscle relaxation was observed in group ButP and Acep after sedation with butorphanol and acepromazine respectively whereas, dexmedetomidine resulted in moderate anal sphincter muscle relaxation in group DexP (Fig 6). Complete anal sphincter muscle relaxation was observed after induction with propofol might be due to synergistic interaction of preanaesthetics like butorphanol, dexmedetomidine and acepromazine with propofol. Propofol also has good muscle relaxation property opined by Venugopal *et al.* (2002) in dogs. In group DexP animals, after dexmedetomidine-propofol administration, there was excellent muscle relaxation for longer duration which could be due to prior administration of dexmedetomidine activating alpha-2 adrenoceptors present in the spinal cord (Branson *et al.*, 1993). Results of the present study were in accordance with study of Kumar *et al.* (2022 a and b) who observed deep sedation alongwith analgesia and relaxation of muscle in dogs after intravenous dexmedetomidine which might be due activation of alpha2 adrenoceptor in CNS.

Quality of anaesthesia

The quality of anaesthesia was judged by abolition of various reflexes (palpebral, pedal, jaw and anal reflexes), extent of muscle relaxation and analgesia after sedation and following induction with propofol. Sedation was good after administration of glycopyrrolate-dexmedetomidine whereas it was poor to fair after administration of glycopyrrolate-butorphanol and glycopyrrolate-acepromazine (Fig 7). Quality of anaesthesia was excellent after administration of propofol and characterized by rapid induction, short duration, excellent muscle relaxation and analgesia. These effects are due to rapid uptake of propofol into the CNS and redistribution from the brain to other

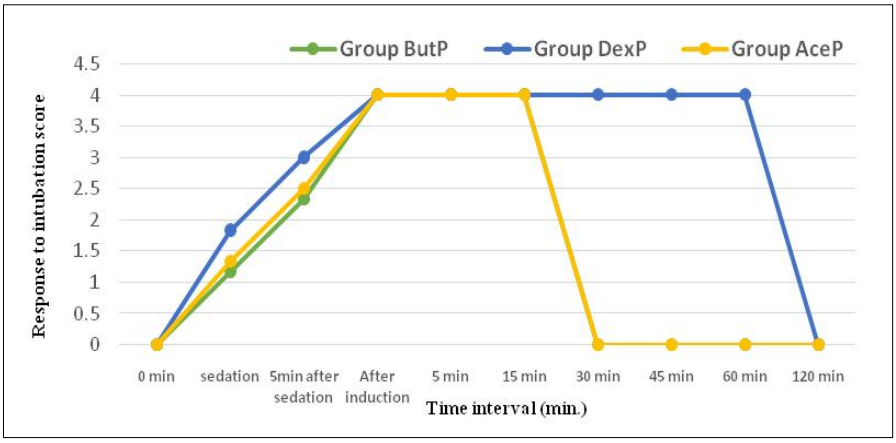

Fig 5: Effect on response to intubation at various time interval in different groups.

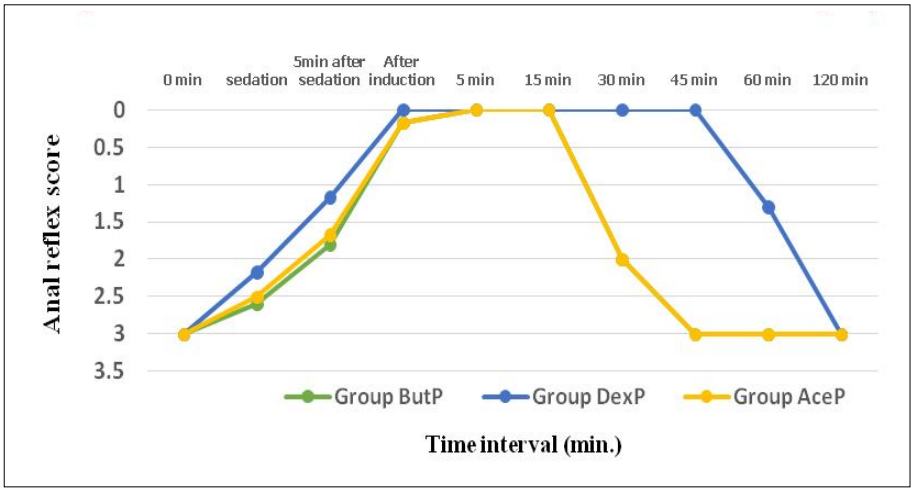

Fig 6: Effect on anal reflex score at various time interval in different groups.

tissue leading to efficient elimination from plasma by metabolism (Zoran *et al.,* 1993) produced effective general anaesthesia in canines. In the present study, all the reflexes *viz.* palpebral, pedal, jaw and anal reflexes were completely abolished with longer duration of muscle relaxation along with analgesia recorded in group DexP up to 45 min. as compared up to 15 min. in groups ButP and AceP post anaesthesia. This might be due to synergistic interaction between the α-2 agonist (dexmedetomidine) and propofol. The present findings are in agreement with Dewangan *et al.* (2010); Jena *et al.* (2014) and Mate and Aher (2019) after propofol anaesthesia in dogs.

Duration of anaesthesia (minutes)

The duration of anaesthesia in group DexP was significantly (P<0.05) longer (58.28±1.45 min.) than group ButP (18.56±2.04 min.) and AceP (15.82±0.91 min.). Longer duration of anaesthesia in animals of group DexP might be due to synergistic action of dexmedetomidine with propofol. Comparison between the groups duration of anaesthesia were statistically significant (P<0.05). Similarly, Sarode (2015) recorded the total duration of anaesthesia as 62.6±10.87 min. in dogs anaesthetized with atropine-dexmedetomidine-propofol. Anandmay *et al.* (2012) reported a significantly (P<0.05) longer duration of anaesthesia in dogs after administration of propofol in combination with buprenorphine (14.00±1.38 min.) as compared to propofol alone (10.26±1.11 min.). The above findings are in confirmatory with Surbhi *et al.* (2010); Jena *et al.* (2014) and Mate and Aher (2019) after propofol anaesthesia in dogs in combination with different preanaesthetic. In the present study, butorphanol or dexmedetomidine or acepromazine was combined with propofol to prolong the duration of anaesthesia and produce profound analgesia with good muscle relaxation. However, a longer duration of anaesthesia was observed in dogs premedicated with dexmedetomidine as compared to butorphanol or acepromazine.

Recovery Time (minutes)

Recovery time was significantly (P<0.05) longer for group DexP as compared to group ButP and group AceP (Table 3). Longer recovery time from anaesthesia in animals of group DexP might be due to synergistic action of dexmedetomidine with propofol whereas shorter recovery time in animals of group ButP and AceP revealed faster rate of metabolic clearance of propofol from the body. The present findings of recovery time in group DexP corroborates with the findings of Jena *et al.* (2014) who observed recovery time of 13.67±1.02 min. and Bayan *et al.* (2002) who noted recovery time of 19.92±0.40 min. during propofol anaesthesia in dogs without premedication. Kojima *et al.* (2002)

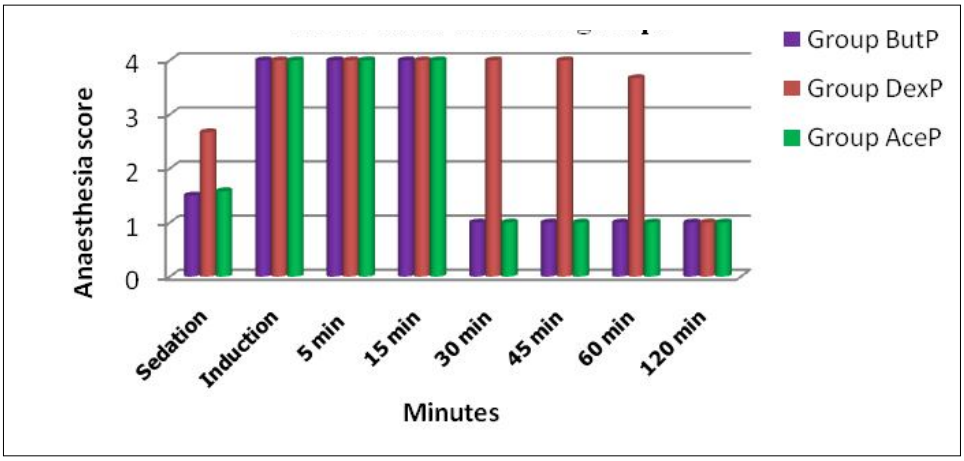

Fig 7: Quality of anaesthesia score at various time intervals in different groups.

Table 3: Recovery from anaesthesia in dogs of different groups.

Parameters (min)	Groups (N=6)		
	ButP	DexP	AceP
Start of recovery time	18.56±2.04^{B}	58.28±1.54^{C}	15.82±0.91^{A}
Time to extubation	19.32±2.03^{B}	63.75±0.68^{C}	17.85±0.76^{A}
Head rightening	22.15±2.17^{A}	71.37±1.30^{B}	19.67±0.67^{A}
Sternal recumbency time	24.67±2.30^{A}	82.50±3.45^{B}	24.50±2.06^{A}
Standing time	28.50±2.38^{A}	86.83±3.82^{B}	30.67±4.56^{A}
Complete recovery time	35.84±3.02^{A}	91.26±3.53^{C}	35.08±4.13^{B}
Complications (If any)	-	Urination	Straightening of legs

ABC - Values bearing different superscript vary significantly. (P<0.05) between groups.

recorded prolonged recovery time in dogs administered with acepromazine-butorphanol-propofol as compared with propofol alone in dogs.

Time to extubation of tube, head rightening, sternal recumbency time, standing time and complete recovery time was significantly (P<0.05) longer for group DexP followed by group ButP and AceP (Table 3) as a result to synergistic action of dexmedetomidine with propofol resulting in deeper sedation and reduced metabolic activity to delay redistribution and metabolism of the drugs. The shorter duration observed in animals of group ButP and AceP signify faster rate of metabolic clearance of propofol from the body. All the animals recovered very smoothly, excitement free with no shivering and struggling after propofol anaesthesia. The difference in complete recovery time from anaesthesia in between groups was statistically significant (P<0.05) in group DexP and non-significant in group ButP and AceP. Hughes and Nolan (1999) reported extubation time, lifting of head and standing time as 30±7 min.; 59±12 min. and 105±13 min. respectively after propofol anaesthesia in greyhounds. Bufalari *et al.* (1997) reported that raising of head in dogs after 18.29±7.37 min. and 21.07±6.25 min. following stood significantly sooner with minimal signs of ataxia at 35.12±09.35 and 35.19±02.39 minutes in acepromazine-propofol and butorphanol-propofol anaesthesia respectively. Ko *et al.* (1996) reported 73.5±19 minutes of sternal recumbency in dogs premedicated with medetomidine and butorphanol following propofol anaesthesia. Sternal recumbency time of group DexP in the present study was in agreement with the results obtained by Rafee *et al.,* (2015) who observed that dexmedetomidine given IV in dogs @ 20 µg/kg under went in lateral recumbency at 90 minutes. Matthews *et al.* (2004); Ambros *et al.* (2008) and Mate and Aher (2019) reported longer duration of recovery in animal who received sedative as preanaesthetic which might be due to additive effect of sedative like dexmedetomidine or midazolam with propofol.

Complications (If any)

Salivation, defecation, nausea, vomition and lacrimation were absent in animals of all the three groups. In present study, no salivation was observed in any of the group which could be attributed to glycopyrrolate antimuscarinic effect. The above findings are in accordance with the Bufalari *et al.* (1997). Voluntary urination was recorded in 5 out of 6 animals in group DexP after reappearance of pedal reflex which might be due to α2-agonist mediated inhibition of release of antidiuretic hormone in dogs or osmotic diuretic effect of increase blood glucose by α2- agonist. Similar findings have been reported by Jena *et al.* (2014) after xylazine or dexmedetomidine with propofol in dogs.

Straightening of legs was recorded in 2 out of 6 animals in group AceP at the time of recovery where acepromazine was premedicated with propofol which might be due to hyper sensitivity response to noise. Yawning was also recorded in 3 out of 6 animals in group AceP after sedation with acepromazine which could be attributed to light state of anaesthesia where dog opens the jaw, curl the tongue and simulate a yawn (Lumbs and Jones, 1996).

CONCLUSION

Anaesthetic study suggests that dexmedetomidine-propofol induces excellent quality of surgical anaesthesia with prolonged duration (58.28±1.45 min.) as compared butorphanol-propofol and acepromazine-propofol in dogs. Therefore, propofol in combination with dexmedetomidine, butorphanol or acepromazine may be safely used for inducing surgical anaesthesia in dogs.

ACKNOWLEDGEMENT

The present study was supported by Dau Shri Vasudev Chandrakar Kamdhenu Vishwavidyalya (DSVCKV), Chhattisgarh.

Disclaimers

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Informed consent

All animal procedures for experiments were approved by the Committee of Experimental Animal care and handling techniques were approved by the University of Animal Care Committee.

Conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this article. No funding or sponsorship influenced the design of the study, data collection, analysis, decision to publish, or preparation of the manuscript.

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