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Clinico-Physiological and Haemodynamic Alterations Following Propofol Induction and Premedication with Butorphanol, Dexmedetomidine or Acepromazine in Dogs

Rukmani Dewangan, Sumeet Pal, Raju Sharda, S. K. Tiwari, Durga Chaurasia, B. B. Khutey and H. K. Ratre

Dept. of Veterinary Surgery and Radiology, College of Veterinary Science and A.H., Anjora, Durg (C.G.), Dau Shri Vasudev Chandrakar Kamdhenu Vishwavidyalya (DSVCKV), Chhattisgarh (491 001), India

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Corresponding dewanganrukmani@gmail.com

0000-0002-5826-2391

ABSTRACT

The present study was conducted during February-October 2021 at Department of Veterinary Surgery and Radiology, College of Veterinary Science & A.H. Anjora, Durg (C.G.), India to evaluate the alternations on clinico-physiological and haemodynamic parameters following propofol induction in dogs premedicated with butorphanol or dexmedetomidine or acepromazine. Eighteen adult dogs of either sex were randomly divided into three groups (BP, DP and AP) with six animals in each. Ten minutes prior to the anaesthetic administration, all the dogs were administered glycopyrrolate @ 0.02 mg kg^{-1} I/M. The animals of group BP, DP and AP were premedicated intramuscularly with butorphanol @ 0.3 mg kg^{-1} b.wt., dexmedetomidine @ 10 µg kg^{-1} b.wt. and acepromazine @ 0.4 mg kg^{-1} b.wt. respectively. General anaesthesia was induced with propofol @ 7 mg kg^{-1} b.wt. intravenously. Clinical parameters were recorded following induction with propofol. Physiological and haemodynamic parameters were recorded before (0), 5 min. after sedation, induction and at 10, 20, 40, 60 and 120 min. post propofol anaesthesia. The onset of sedation and anaesthesia was quicker in group DP followed by AP and BP. Duration of anaesthesia and complete recovery were significantly (p<0.05) longer in group DP as compared to group BP and AP. The physiological and haemodynamic parameters showed transient changes which were compensated and remained within normal range during the study period without producing any deleterious effect. Thus, propofol can be safely used as induction agent in dogs premedicated with either dexmedetomidine or butorphanol or acepromazine and does not produce any adverse effect on cardiopulmonary system. However, dexmedetomidine-propofol combination provided longer duration of anaesthesia in dogs as compared to other groups.

KEYWORDS: Acepromazine, anaesthetic, butorphanol, dexmedetomidine, haemodynamic, physiological and propofol

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1. INTRODUCTION

Canines undergo many surgical interventions which necessitate the need of a safe and effective anaesthetic that can produce sleep, amnesia and muscle relaxation. There is no single anaesthetic agent available till date that can provide these desirable effects by itself. Therefore, a combination of sedatives and other anaesthetics have been widely used in animal practice to attain desirable general anaesthesia. Today in an era of balanced anaesthesia which can be achieved by appropriate use of multiple drugs and characterized by unconsciousness, analgesia, muscle relaxation and alteration of autonomic reflexes. The combination of complementary drugs permits use of decreased dose of each drug to achieve anaesthesia with reduction in their commensurate side effects (Grimm et al., 2001). Premedication of animals before induction of anaesthesia provides significant advantage in terms of cardiovascular stability, analgesia and quality of recovery (Lemke, 2007). A good preanaesthetic is needed before induction of anaesthesia with propofol to produce desired surgical anaesthesia. Considering the advantages of dexmedetomidine as sedative, butorphanol as an opioid analgesic and acepromazine as a phenothiazine tranquilizer, were used as premedicants in the present study to propofol induction.

Anticholinergic premedication has been recommended with α_2 agonists to prevent bradyarrhythmias and potential reduction in cardiac output produced by these agents which has been widely adopted within most veterinary practices. Glycopyrrolate inhibits cholinergic transmission by blocking peripheral muscarinic receptors and is a synthetic quaternary ammonium compound with no central effects. It is about five times more effective than atropine and has a powerful and long-lasting antisialagogue effect. Opioids are the most commonly used analgesics to supplement anaesthesia for tolerance of surgical procedures due to their efficacy, rapid onset of action and safety. Butorphanol tartrate is a centrally acting agonist antagonist type of opioid that provides sedation, short duration analgesia and reduces the dose of intravenous anaesthetics for induction (Koc et al., 2006). The alpha 2 adrenergic agonists are useful adjuncts to anaesthesia because of their sedative, anxiolytic and analgesic effects. Among alpha2 agonists, dexmedetomidine is an active optical isomer of medetomidine (Ahmad et al., 2013) and selective steroisomers used in anaesthesia because of more predictable pharmacokinetics and pharmacodynamics, compared to their racemic mixture (Uilenreef et al., 2008). Acepromazine is a phenothiazine tranquilizer that depresses the reticular activating system and inhibits dopamine receptors in the CNS, resulting in drowsiness. It has a longer half-life in young animals because it is processed by the liver and removed by the kidney. It induces mild to moderate tranquilization, muscle relaxation and a decrease in spontaneous activity attributable principally to central dopaminergic antagonism (Adediran and Adetunji, 2021). Because of the dopamine inhibition in the chemoreceptor trigger zone, it also possesses antiemetic, anticonvulsant, antispasmodic, hypotensive and hypothermic properties (Yohannes, 2018).

Propofol (2-6 di-isopropylphenol) is a nonbarbiturate, nonsteroid, short acting general anaesthetic that is associated with a rapid induction and good quality recovery, but may cause hypotension and apnoea. Propofol is an intravenous hypnotic agent commonly administered intravenously for induction and maintenance of anaesthesia by bolus or continuous infusion in dogs and produces unconsciousness in a rapid, smooth and safe fashion in healthy animals (Lerche et al., 2000). The mechanism of action of propofol is exactly unknown but it produces anaesthetic effect by acting at $GABA_A$ receptors and the fast redistribution from the brain to other tissues and rapid effective clearance from body by metabolism accounts for the brief action and smooth emergence. However, propofol as sole general anaesthetic is unsatisfactory because of its poor analgesic property. Consequently, for major surgical procedures must be combined with potent analgesic drugs such as opioids and alpha 2 agoinsts (Adediran and Adetunji, 2021). Since review reveals very scanty literature on the use of propofol with glycopyrrolate, butorphanol, dexmedetomidine and acepromazine in dogs, therefore, the aim of present anaesthetic study was to evaluate the alternations on clinico-physiological and haemodynamic parameters following propofol induction in dogs premedicated with butorphanol, dexmedetomidine andacepromazine.

2. MATERIALS AND METHODS

2.1. Place of work

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

2.2. Study design

Eighteen healthy dogs of either sex weighing between 10 to 20 kg body weight were randomly divided into three groups viz., BP, DP and AP, comprising of 6 animals in each. All dogs were dewormed with Praziplus (Albendazole 300mg 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^{-1} b.wt. intramuscularly. The animals of group BP, DP and AP were premedicated intramuscularly with butorphanol @ 0.3 mg kg^{-1} b.wt.,dexmedetomidine @ 10 µg kg^{-1} b.wt. andacepromazine @ 0.4 mg kg^{-1} b.wt. respectively. General anaesthesia was induced with propofol @ 7 mg kg^{-1} b.wt. intravenously in animals of all the groups and dogs were intubated with suitable endotracheal tubeof (4.5 to 8.5 OD mm) with guidance of laryngoscope.

2.4. Evaluation of clinico-physiological parameters

The clinical parameters assessed were onset of sedation, induction of anaesthesia, duration of anaesthesia, and complete recovery from propofol anaesthesia. The physiological parameters included heart rate, respiratory rate and rectal temperature which were recorded at before (0), 5 min. after premedication/sedation, following induction and at 10, 20, 40, 60 and 120 minutes after propofol anaesthesia.

2.5. Evaluation of haemodynamic parameters

Various heamodynamic parameters viz., blood pressure, SpO_2 and capillary refill time were recorded at before (0), 5 min. afterpremedication/sedation, following induction and at 10, 20, 40, 60 and 120 minutes after propofol anaesthesia. Systolic blood pressure (SBP) and diastolic blood pressure(DBP) were determined using a non-invasive system with the cuff placed on thefore leg over the metacarpal artery and recorded by the veterinary patient monitor(New Gen Medical Systems) Mean arterial pressure (MAP) was calculated using the formula MAP=DP+1/3(SP-DP). Haemoglobin oxygen saturation (SpO2) was monitored by the pulse oximeter using multiparameter veterinary patient monitor and recorded with the sensor probe placed on the lateral surface of the tongue or ear pinna of each dog. Capillary refill time (seconds) was monitored by pressing the gingival mucosa digitally.

2.6. Statistical analysis

The data collected was 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 were analysed through statistics software program (SPSS-2017; v25.0) and data was presented as Mean±S.E. Statistically significant differences were considered at 5 percent level (5%) of significance.

3. RESULTS AND DISCUSSION

3.1. Clinical parameters

There was decrease in spontaneous activity in all the animals after administration of preanaesthetic agent. All the animals remained conscious but were unable to stand when disturbed. No cases of salivation and vomiting were observed in all the three groups. Mild sedation was observed in group BP and AP at 9.0±0.26 min. and 9.71±0.31 min respectively. Whereas in group DP, there was marked sedation with lowering of the head with early lateral recumbency in 6.83±0.31 min (Figure 1). Comparison between groups revealed rapid onset and profound sedation after administration of dexmedetomidine in group DP. In the present study, the onset of sedation and recumbency was earlier in Group DP than other groups, due to the onset of action of dexmedetomidine owing to its lipophilic property (Amarpal et al., 1996). Alvaides et al. (2008) reported increased onset of sedation within 2 to 5 minutes following dexmedetomidine administration. Ahmad et al. (2013) reported onset of sedation at 4.50±0.96 minutes after intramuscular injection of dexmedetomidine in dogs. In group DP, there was excellent sedation as compared to group BP and AP while both groups showed mild sedation. The sedative/hypnotic effects of dexmedetomidine are mediated through pertussis-sensitive inhibitory G proteinin locus coeruleus resulting in hyperpolarization and reduced nerve conduction (Kuusela et al., 2000). Acepromazine induces mild to moderate sedation and a main behavioural effect in canines by blocking or antagonising the post-synaptic D2 receptors. Contrary to our finding, Posner (2007) and Roon et al. (2007) documented profound to moderate sedation after acepromazine administration. The faster onset of sedation was recorded after sedation with dexmedetomidine in the present study confirmed with our observation by various workers (Amarpal et al., 1996 and Ahmad et al., 2013). Similarly, Arunkumar et al. (2017) reported onset of sedation at 2.05±0.19 min. after dexmedetomidine administration in dogs and Pircioet al. (1976) recorded mild sedation in dogs when injected butorphanol @ 0.5 mg kg^{-1} bwt. IM. Sharma and Bhargava (2007) reported onset of anaesthesia at 60.83±6.88 seconds after administrating triflupromazine-propofol in the dog. The use of premedicants in the present study was aimed in relieving anxiety in order to smoothen anaesthetic induction, maintenance and recovery phase. Propofol as sole agent lacks analgesic property which necessitates the use of premedicants with it as to provide preemptive analgesia.

The induction of anaesthesia lasted for 0.55±0.02, 0.49±0.03, and 0.53±0.02 min. in groups BP, DP and AP respectively (Figure 1). In the present study, the shorter induction of anaesthesia was observed in group DP as compared to group BP and AP. Induction of anaesthesia was quicker in animals premedicated with dexmedetomidine as compared to with butorphanol or acepromazine. This might be due to the effects of dexmedetomidine which produces sufficient degree of sedation prior to induction with propofol. Arunkumar et al. (2017) reported induction time of 57.33±0.99 seconds

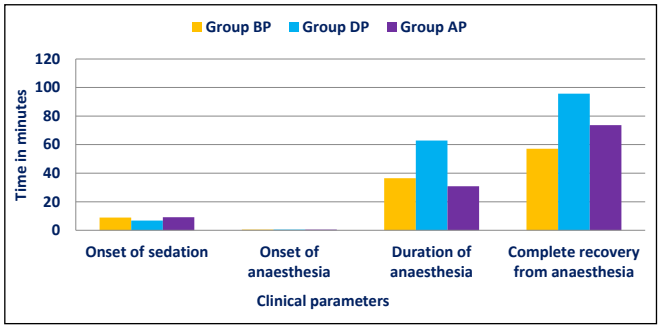

Figure 1: Showing onset of sedation, induction, duration and complete recovery from anaesthesia following propofol induction in dogs

after dexmedetomidine-propofolanaesthesia in dogs. The induction of anaesthesia was 36.00±1.86 sec. and 28.00±2.00 sec. after administration of propofol alone and in combination with buprenorphine in dogs (Anandmay et al., 2012). 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. In the present study, downward rotation of eyeball was observed after induction with propofol and during surgical anaesthesia. Similarly, Bayan et al. (2020) also noted a shorter induction time of34.67±2.12 sec. after propofol anaesthesia in dexmedetomidine and butorphanol premedicated dogs. The duration of anaesthesia in group DP was significantly (p<0.05) longer (58.28±1.45 min.) than group BP (18.56±2.04 min) and AP (15.82±0.91 min.) (Figure 1). Longer duration of anaesthesia in animals of group DP might be due to synergistic action of dexmedetomidine with propofol. Similarly, Meshram (2015) recorded longer mean duration of anaesthesia in dogs anaesthetized with dexmedetomidine-propofol (48.73±3.13 min.) as compared to diazepam-propofol (25.86±2.32 min.) and propofol alone (13.16±0.75 min.). Sarode (2015) recorded the total duration of anaesthesia as 62.6±10.87 min in dogs anaesthetized with atropine-dexmedetomidine-propofol.

Complete recovery from propofol anaesthesia was significantly (p<0.05) longer in group DP (91.26±3.53 min.) followed by group AP (35.08±4.13 min.) and group BP(35.84±3.02 min) respectively (Figure 1). The difference in the complete recovery from anaesthesia in between groups was statistically significant (p<0.05) in group DP and non-significant in group BP and AP. All the animals recovered very smoothly, excitement free with no shivering and struggling after propofol anaesthesia. Longer complete recovery from anaesthesia in animals of group DP might be due to synergistic action of dexmedetomidine with propofol resulting in deeper sedation and reduced metabolic activity to delay redistribution and metabolism of the drugs. On the other hand, shorter complete recovery time was observed in animals of group BP and AP. Bufalari et al. (1997) reported dogs premedicated with acepromazine or butorphanol stood significantly sooner with minimal signs of ataxia at 35.12±09.35 and35.19±02.39 minutes, respectively after propofol anaesthesia. Mate and Aher (2019) noted complete recovery time from anaesthesia after intravenous administration of dexmedetomidine-butorphanol and dexmedetomidine-midazolam as preanaesthetic with propofol anaesthesia in dogs which was 56.0±13.41 min. and 38.71±8.57 min. respectively

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 Bufalari et al. (1997). Voluntary urination was recorded in 5 out of 6 animals in group DP 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 increased blood glucose by α2- agonist. Similar findings have also 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 AP 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 AP 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 (Lumb and Jones, 1996).

3.2. Physiological parameters

3.2.1. Heart rate (beats per minute)

In group BP, a non-significant decrease in heart rate was observed after sedation with butorphanol which further decreased non-significantly after induction with propofol up to 20 min. post anaesthesia. Later on, the values increased and returned to near normalcy by 120 min. In group DP, a non- significant increase in heart rate was observed after sedation with dexmedetomidine which further decreased significantly (p<0.05) after induction with propofol up to 40 min. post anaesthesia. However, the values increased and returned to base value by 120 min. interval. Group AP showed a non-significant decrease after administration of acepromazine-propofol anaesthesia up to 10 min. post anaesthesia. Later on, these values increased and returned to the normal physiological range by 120 min (Table 1). There was a non-significant decrease in heart rate in group BP and AP after administration of butorphanol and acepromazine respectively whereas heart

Table 1: Effects on physiological parameters following propofol induction in dogs at various time intervals in different groups

Parameters	Group (n=6)	0 (min)	5 min after sedation (min)	After induction (min)	10 (min)	20 (min)	40 (min)	60 (min)	120 (min)
Heart rate (beats/ min.)	BP	92.17^{Acd} ±3.33	86.83^{Aabcd} ±2.82	84.17^{Aabc} ±2.80	82.17^{Aab} ± 2.70	80.17^{Aa} ±2.63	85.17^{Aabcd} ±2.44	89.67^{Abcd} ±2.39	93.67^{Acd} ±2.82
	DP	95.33^{Ac} ±3.16	99.67^{Bc} ±3.02	92.83^{Abc} ±3.05	89.33^{Aabc} ±2.58	84.50^{Aab} ±2.72	81.17^{Aa} ±2.65	90.17^{Aabc} ±3.34	94.17^{Abc} ±5.21
	AP	94.83^{Aab} ±3.47	92.00^{ABab} ±3.70	86.00^{Aab} ±3.84	83.00^{Aa} ±3.54	87.17^{Aab} ±3.88	89.17^{Aab} ±3.81	92.83^{Aab} ±3.72	95.17^{Ab} ±3.24
Respiration rate (breath/min.)	BP	22.67^{Ae} ±0.79	20.67^{Bcd} ±0.73	18.25^{Bbc} ±0.70	16.65^{Bb} ±0.56	14.78^{Ba} ±0.42	16.47^{Bb} ±0.43	20.87^{Bc} ±0.67	21.33^{Ade} ±0.84
	DP	21.64^{Ae} ±0.73	16.33^{Ad} ±0.61	13.50^{Ac} ±0.62	12.00^{Abc} ±0.48	11.33 ± 0.42^{Aab}	10.17^{Aa} ±0.48	14.5^{Ad} ±0.43	16.83 ± 1.48^{Ae}
	AP	22.17 ± 0.60^{Ae}	20.33^{Bd} ±0.33	18.33^{Bc} ±0.21	16.17^{Bb} ±0.31	14.33^{Ba} ±0.21	15.83^{Bb} ±0.31	18.17^{Bc} ±0.60	21.83^{Ae} ±0.31
Rectal temperature (°F)	BP	101.03^{Aa} ±0.68	100.95^{Aa} ±0.47	100.48^{Aa} ±0.40	100.17^{Aa} ±0.58	99.4^{Aa} ±0.63	99.8^{Aa} ±0.63	100.3^{Aa} ±0.64	101.08^{Aa} ±0.65
	DP	101.05^{Ac} ± 0.28	101.01^{Ac} ± .24	100.73^{Abc} ±0.32	100.57^{Abc} ±0.33	99.83^{Aab} ±0.38	99.38^{Aa} ±0.38	99.32^{Aa} ±0.49	100.23^{Aabc} ±0.31
	AP	101.28^{Ad} ± 0.24	100.52^{Ac} ± 0.20	100.12^{Abc} ±0.17	99.68^{Aab} ±0.26	99.43^{Aa} ±0.31	99.78^{Aab} ±0.27	100.61^{Abc} ±0.18	101.03^{Ac} ±0.08

ABC: Values bearing different superscript vary significantly. (p<0.05) between groups; abcde: Values bearing different superscript vary significantly. (p<0.05) within groups

rate increased non-significantly after administration of dexmedetomidine in group DP. Butorphanol has been reported to cause a mild decrease in heart rate with minimal cardiovascular effect (Trim, 1983; Greene et al., 1990). It has been reported that butorphanol facilitates the increase in parasympathetic tone and thereby contributes to bradycardia (Ko et al., 2000). A non-significant change in heart rate has been reported in dogs treated with buprenorphine and acepromazine (Stepien et al., 1995). Reduction in heart rate was reported in dogs premedicated with acepromazine at the dose rate of 0.1 mg kg^{-1} intramuscular (Bufalari et al., 1997). In the present study, animals of group DP showed an initial increase in heart rate after sedation with dexmedetomidine which is in accordance with earlier studies in which pre-emptive administration of anticholinergic like atropine or glycopyrrolate was capable of reversing α-2 agonist-induced bradycardia in dogs (Ko et al., 2001) and caused initial tachycardia (Alibhai et al., 1996). The accelerated heart rate might be due to the effect of the anticholinergic drug (glycopyrrolate) administrated along with dexmedetomidine as preanaesthetic agent which was capable of increasing heart rate by reversing α-2 agonist-induced bradycardia (Neto et al., 2004). Contrary to our study, Ahmad et al. (2013) and Santosh et al. (2013) reported decrease in heart rate after dexmedetomidine administration in dogs which might be attributed due to higher dose of dexmedetomidine used in their study. There was a decrease in heart rate after administration of propofol in all the three groups and thereafter showed a progressive increasing trend to reach the base value by 120 min. of the observation period. The decreased in heart rate after propofol administration might be due to propofol-induced vasodilation leading to a fall in systemic vascular resistance as well as dose-related depression of myocardial contractibility (Duke, 1995). The above findings are in agreement with Dewangan et al. (2010) and Anandmay et al. (2012) following propofol anaesthesia in dogs. However, Surbhi et al. (2010) and Suthar et al. (2018) recorded an increase in heart rate after propofol anaesthesia which might be due to effect of propofol.

3.2.2. Respiration rate (breaths per minute)

In the present study, respiratory depression was more marked in animals of group premedicated with dexmedetomidine as compared to butorphanol and acepromazine. There was a significant (p<0.05) decrease in respiratory rate from the base value up to 10 min. after butorphanol-propofol anaesthesia in group BP. The animals

of group DP showed a significant (p<0.05) decrease in respiratory rate up to 120 min. with a peak decrease at 40 min. after dexmedetomidine-propofol anaesthesia. The animals of group AP showed a significant (p<0.05) decrease in respiratory rate at 10 min. However, these values returned to base line by 120 min. interval in all three groups (Table 1). Apnoea was frequently observed after induction of propofol anaesthesia in dogs (Morgan and Legge, 1989). Apnoea of 30 seconds or longer has been observed at an incidence rate of 25% in dogs receiving propofol (Nolan et al., 1993). In the present study, transient apnoea was observed immediately after propofol administration which lasted for 30-40 seconds in all the three groups. Respiratory depression and apnoea, are the most reported adverse effect of propofol anaesthesia and proportional to rate of infusion of propofol (Maney et al., 2013). Transient apnoea of 19.5 seconds was reported with rapid injection of propofol administration and 28.8 seconds with slow injection of propofol administration in dogs premedicated with acepromazine and morphine (Murison, 2001). In the present study, there was significant (p<0.05) decrease in respiration rate after induction with propofol in all the three groups. This reduction in respiration rate might be attributed to combined effect of preanaesthetics viz. butorphanol, dexmedetomidine and acepromazine with propofol.Propofol can induce significant depression of respiratory functions characterized by a reduction in the rate of respiration by depressing central inspiratory drive and ventilator response to arterial carbon dioxide response. Induction of anaesthesia with propofol also led to a decrease in respiratory rate in dogs which are in agreement with earlier studies (Jena et al., 2014 and Arunkumar et al., 2017).

The decrease in respiration rate in group BP might be due to the direct depressive effect of butorphanol in the medullary centre in the general and respiratory centre in particular. The results of the present study are in agreement with the findings of Benson and Tranquilli (1992) who documented respiratory effect of butorphanol on small animal anaesthesia. Opioids in combination with propofol increase the probability of respiratory depression during anaesthesia (Short and Bufalari, 1999). Similarly, Anandmay et al. (2012) also reported significant decrease in respiration rate after propofol alone and in combination with buprenorphine in atropinized dog. Severe respiratory depression was recorded in group DP due to synergistic effect of dexmedetomidine and propofol. It might be due to the direct depressant action of alpha 2- agonist like dexmedetomidine which has been known to produce respiratory depression caused by activation of the α-2 adrenergic pathway which led to inhibition of locus coeruleus neurons (Oyamada et al., 1998). The result of the present study was in conformity with the findings of Amarpal et al. (1996) who documented that administration of medetomidine or detomidine in dogs cause a decrease in respiratory rate with minimal effects on blood gases. There was significant decrease in respiration rate after administration of acepromazine in group AP, which confirms the finding of lower respiration rate in the patients treated with acepromazine (Bufalari et al., 1997). In contrast to this, Bigby et al. (2017) reported that respiration is rarely affected by acepromazine at therapeutic dosages.

3.2.3. Rectal temperature (°F)

In animals of group BP and AP, a non-significant decrease in rectal temperature was observed after sedation with butorphanol and acepromazine respectively which further decreased non-significantly up to 20 min after administration of propofol anaesthesia. But in group DP, a non-significant decrease in the rectal temperature was observed after administration of propofol in combination with dexmedetomidine which persisted up to 60 min. However, these values returned to normalcy by 120 min. in all three groups (Table 1). In the present study, there was a non-significant decrease in rectal temperature in all the groups after sedation with preanaesthetics and induction of anaesthesia with propofol but remained within the physiological limits. A decrease in rectal temperature after administration of preanaesthetic and anaesthetic might be attributed to a decrease in heat production due to a least muscular activity and also direct effect of drugs on the hypothalamus (Virtanen, 1989). In group BP, a decrease in rectal temperature was observed after administration of butorphanol which might be due to decrease in body temperature by reducing basal metabolic rate and through heat loss via respiratory system, especially in panting animals (Thurmon et al., 1996; Ku Kanich and Wiese, 2015). In the animals of group DP, a non-significant decrease in the rectal temperature was also observed after sedation with dexmedetomidine which might be due to activation of alpha2 C drenoceptors by dexmedetomidine which mediate hypothermia (Lemke, 2007) in combination with the reduction in muscular activity and BMR. On the contrary to our study, Ahmad et al. (2013) reported a non-significant increase in rectal temperature in dogs after administration of dexmedetomidine alone at 20 µg kg^{-1} I/M. In group AP, a decrease in rectal temperature was observed after administration of acepromazine which is a phenothiazine agent and known to interfere with thermoregulatory mechanisms leading to decrease in body temperature (Thurmon et al., 1996; Hall et al., 2001; Wagner, 2002). The decrease in rectal temperature in the present study can be attributed to synergistic action of preanaesthetic (butorphanol, dexmedetomidine,

and acepromazine) and propofol causing depression of thermoregulatory centre. Similar findings were observed by Sharma and Bhargava (2007) in dogs under triflupromazine-propofol anaesthesia. Surbhi et al. (2010) also observed significant reduction in rectal temperature after premedication with medetomidine-butorphanol and propofol anaesthesia in dogs.

3.3. Haemodynamic parameters

Systolic blood pressure (mmHg) decreased significantly in group BP and AP after 5min. sedation with glycopyrrolate-butorphanol (from 123.33±2.17 to 112.0±2.27 mmHg) and glycopyrrolate-acepromazine (from 120.17±2.48 to 109.17±2.33 mmHg) respectively which further significantly decreased up to 20 min. (from 112.0±2.27 to 100.83±1.25 mmHg) and (from 109.17±2.33 to 100.50±2.17 mmHg) respectively after induction with propofol. While in group DP, a significant increase in systolic blood pressure was observed 5 min. after sedation with glycopyrrolate-dexmedetomidine (from 122.0±1.15 to 128.17±0.90 mmHg) which further significantly decreased up to 40 min. (from 128.17±0.90 to 113.33±1.63 mmHg) after induction with propofol. Later on, SBP increased and returned to near normalcy by 120 min (Figure 2). Similarly, Smith et al. (1993) reported a significant decrease in SAP after propofol administration in dogs treated with acepromazine (SAP, 178 mm of Hg before vs 128 mm of Hg after propofol) and with acepromazine/butorphanol (SAP, 184 mm of Hg before vs 98 mm of Hg after propofol).

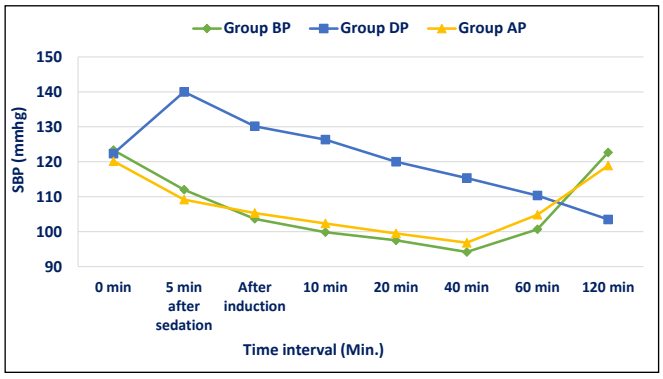

Figure 2: Effect on systolic blood pressure (mm Hg) following propofol induction in dogs at various time intervals in different groups

Diastolic blood pressure (mmHg) also decreased significantly in group BP and AP, 5 min. after sedation with glycopyrrolate-butorphanol (from 81.67±1.23 to 76.17±2.29 mmHg) and glycopyrrolate-acepromazine (from 80.33±0.92 to 76.33±1.50 mmHg) respectively which further significantly decreased after induction with propofol up to 10 min. (from 76.17±2.29 to 69.17±1.38 mmHg) and 20 min. (from 76.33±1.50 to 70.17±2.51 mmHg) respectively. While in group DP, a significant increase in diastolic blood pressure was observed 5 min. after sedation with glycopyrrolate-dexmedetomidine (from 80.83±0.79 to 91.33±0.88 mmHg) which further significantly decreased after induction with propofol up to 40 min. (from 91.33±0.88 to 77.33±1.41 mmHg). Later on, DBP increased and returned to near normalcy by 120 min.interval(Figure 3). Mean blood pressure (mmHg) decreased significantly in animals of group BP and AP, 5 min. after sedation with glycopyrrolate-butorphanol (from 95.06±0.74 to 88.11±1.71 mmHg) and glycopyrrolate-acepromazine (from 93.61±1.18 to 87.28±1.50 mmHg) respectively which further significantly decreased after induction with propofol up to 10 min (from 88.11±1.71 to 79.22±0.94 mmHg) and 20 min (from 87.28±1.50 to 80.28±2.29 mmHg) respectively. While in group DP, a significant increase in mean blood pressure was observed 5 min after sedation with glycopyrrolate-dexmedetomidine (from 94.56±0.74 to 103.61±0.59 mmHg) which further significantly decreased after induction with propofol up to 40 min. (from 103.61±0.59 to 89.33±1.05 mmHg). Later on, MAP increased and returned to near normalcy by 120 min. interval (Figure 4).

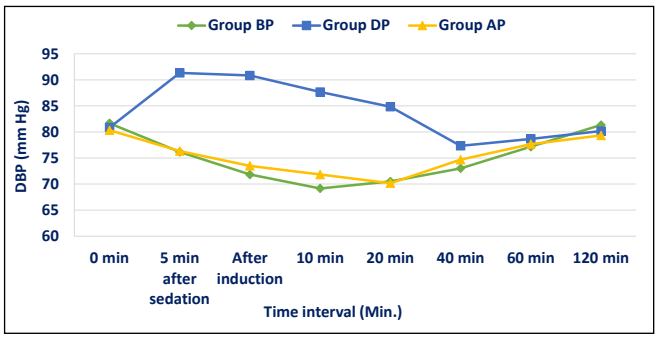

Figure 3: Effect on diastolic blood pressure (mmHg) following propofol induction in dogs at various time intervals in different groups

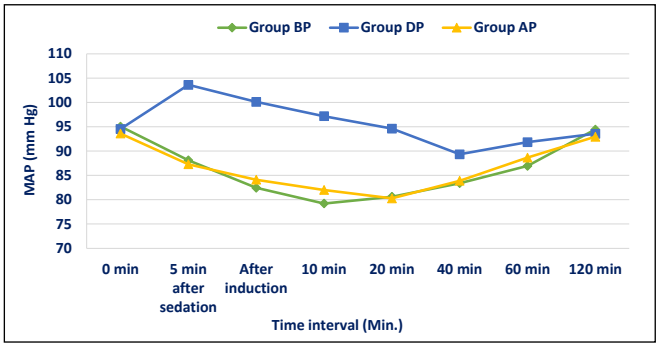

Figure 4: Effect on mean arterial pressure (mmHg) following propofol induction in dogs at various time intervals in different groups

A decrease in blood pressure is a common finding when propofol is used to induce anaesthesia in dogs (Cattai et al., 2018). Decrease in diastolic pressure with propofol induction had also been reported by Jenna et al. (2014) and Bolaji-Alabi et al. (2018) which could be as propofol causes a transient decrease in blood pressure mainly due to peripheral vasodilation, decreased sympathetic outflow and myocardial depression. The reduction in mean arterial pressure in dogs premedicated with acepromazine contributes to hypotension during general anaesthesia by causing a decrease in arterial blood pressure due to its alpha-antagonism and resultant vasodilation and hypotension (Monteiro et al., 2017). Bufalari et al. (1997) recorded marked decreases in MAP, reflecting a significant drop in DAP induced by a propofol-butorphanol combination which might suggest the decrease in peripheral vascular tone mediated by butorphanol-propofol. Hypotension induced by vasodilatation may be observed when acepromazine is used as a preanaesthetic, especially at higher dosages (Bufalari et al., 1997). Similarly, Rafee et al. (2015) also reported a significant increase in SBP, DBP and MBP after administration of dexmedetomidine alone or with an opioid may be due to high blood concentration of dexmedetomidine and atropine. Anticholinergic agents like atropine and glycopyrrolate are capable of causing hypertension (Alibhai et al., 1996). Lemke et al. (1993) also reported increased blood pressure after anticholinergic administration with alpha-2-agonists.The increase in DBP after sedation with glycopyrrolate-dexmedetomidine might be due to stimulation of peripheral α-2Bagonist receptors mediated transient initial hypertension of variable duration (Vainio et al., 1989). Propofol causes a transient decrease in diastolic blood pressure mainly due to a decrease in peripheral vascular resistance, direct negative inotropic action, decrease sympathetic outflow and myocardial depression (Cullen and Reynoldson, 1993).

The reduction in SBP, DBP and MBP following propofol administration arises primarily as a result of the vasodilatory effect of propofol which could be due to both a reduction of sympathetic tone and a direct effect on a smooth muscle. Similarly, Sooryadas et al. (2011) also reported decreased systemic arterial BP due to peripheral vasodilation following propofol induction. Induction of anaesthesia with propofol in the dog has been demonstrated which results in a dose-dependent decrease of systemic vascular resistance (Cattai et al., 2018). The SBP, DBP and MBP in the present study showed a significant decrease after propofol induction premedicated with butorphanol or dexmedetomidine or acepromazine and remained within the physiological range. Contrary to the current study, Dar et al. (2019) reported non- significant changes in diastolic arterial pressure after propofol induction in dogs premedicated with diazepam-butorphanol.

Animals of group BP and AP showed significant decrease in SpO_2 (%) after sedation with glycopyrrolate-butorphanol and glycopyrrolate-acepromazine respectively which further decreased significantly after induction with propofol up to 20 min (from 98.17±0.31 to 91.33±0.71%) and (from 97.83±0.31 to 91±0.37%) respectively (Figure 5). Later on, SpO_2 increased significantly and returned to near normalcy by 120 min. While in group DP, a significant decrease in SpO_2 (%) was observed after sedation with glycopyrrolate-dexmedetomidine which further decreased significantly after induction with propofol up to 40 min (from 98.33±0.21 to 91.17±0.48%) post anaesthesia. Later on, these values increased significantly and returned to normalcy at 120 min.interval. The SpO_2 value ranged from 87.17±0.40 to 98.33±0.21 in all the three groups of animals at various time intervals. Bufalari et al. (1997) reported an initial reduction in oxygen saturation (SpO_2) below 90% following propofolinduction in dogs premedicated with acepromazine, butorphanol, and acepromazine-butorphanol and was corrected with oxygen supplementation following which SpO_2 remained within the acceptable clinical range. Similarly, decreased SpO_2 has been reported following administration of butorphanol-medetomidine or dexmedetomidine in propofol anaesthetized dogs (Gupta, 2010; Surbhi et al., 2010). The present findings were in accordance with Lerche et al. (2000) and Jenna et al. (2014) which could be because of propofol causing respiratory depression and ultimately decreased respiration rate. Contrary to our study, Bolaji-Alabi and Adetunji (2018) and Suthar et al. (2018) reported non-significant reduction in SpO_2 after propofol anaesthesia. SpO_2 provides an estimate of the percent haemoglobin saturated with oxygen and monitoring of the SpO_2 is an excellent non-invasive, readily available diagnostic method

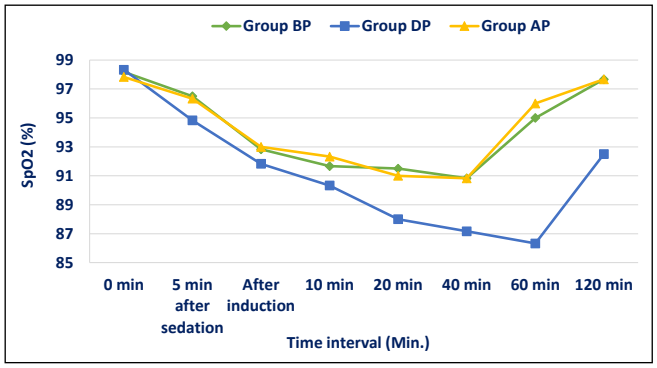

Figure 5: Effect on SpO_2 (%) following propofol induction in dogs at various time intervals in different groups

that provides early warning of desaturation (Pachtinger, 2013). In the present study, a decrease in $SpO_2$was observed in groups BP, DP and AP after sedation with glycopyrrolate along with butorphanol, dexmedetomidine and acepromazine which further decreased after induction with propofol. Later on, SpO_2 increased to near base value by 120 min. of the study period. It indicates that healthy dogs were used in this study and were able to maintain oxygenation without supplementation. The initial decrease in SpO_2 in animals of group DP might be attributed to vasoconstriction caused by the combined effect of α-2 agonist like dexmedetomidine (Kuusela et al., 2000) and propofol (Thurmon et al., 1994). Low arterial oxygen concentration could also be caused by respiratory depression due to sedation (Leppanen et al., 2006). A decrease in respiration rate was recorded in all the groups after sedation with butorphanol or dexmedetomidine or acepromazine and after induction with propofol might also be responsible for reduced SpO_2 in the present study. Similarly, respiratory depression observed might be due to the combined effect of butorphanol (Kuo and Keegan, 2004); dexmedetomidine (Kuusela et al., 2000); acepromazine (Bufalari et al., 1997) and propofol (Bayan et al., 2002).

The capillary refill time in all the animals at 0 (base value), 5 min after sedation and after induction and at 10, 20, 40, 60 and 120 min post propofol anaesthesia was recorded less than 2 seconds. The capillary refill time observed in the present study did not showed any significant change throughout the study period. Capillary refill time (CRT) is the time taken for a capillary bed to refill with blood following digital pressure on the gum. It normally takes less than two seconds for the colour to return but any circulatory failure increases the capillary refill time (Kumar, 1996). Capillary refill time was used to assess the adequacy of peripheral perfusion. Pink mucous membranes and a rapid capillary refill time indicates good peripheral blood flow. Excessive depth of anaesthesia would cause the mucous membranes to become pale and capillary refill time to increase. Tissue perfusion is usually decreased when the gums are pale, rather than pink, and the capillary refill time (CRT) exceeds 1.5 seconds, or the mean arterial pressure (MAP) is less than 60 mmHg (Hall et al., 2011). In the present study, cyanosis or pallor mucus membranes was not observed at any time interval in animals of the three groups because only surgical anaesthesia was induced and dose of propofol was appropriate. CRT also provides information on the state of homeostasis and should be less than 1.5 to 2.0 seconds (Hubbell, 2006). A capillary refill time of less than 2 seconds was observed in the present study and confirms the findings of Girard et al. (2010) in dogs administered intravenous medetomidine or butorphanol (alone or in combination).

4. CONCLUSION

The results of the present study suggest that glycopyrrolate-dexmedetomidine-propofol provides adequate and longer duration of anaesthesia as compared to glycopyrrolate-butorphanol-propofol and glycopyrrolate-acepromazine-propofol in dogs. Hence, propofol can be used safely as an induction agent in dogs premedicated with glycopyrrolate-butorphanol, glycopyrrolate-dexmedetomidine as physiological and haemodynamic parameters showed transient changes which remained within physiological limits and were compensated within study period.

5. ACKNOWLEDGEMENT

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