EVALUATION OF CLINICOPHYSIOLOGICAL AND HAEMATOBIOCHEMICAL PARAMETERS IN PROPOFOL-SEVOFLURANE ANAESTHESIA ALONE AND IN COMBINATION WITH DEXMEDETOMIDINE AND BUTORPHANOL IN ATROPINIZED DOGS

Kanika Tiwari[1*], Narendra Singh Jadon[2], Priyanka Pandey[3], Manjul Kandpal[2] and Jyotsana Bhatt[1]
Department of Veterinary Surgery and Radiology, College of Veterinary and Animal Sciences, Govind Ballabh Pant University of Agriculture and Technology, Pantnagar-263 145, Uttarakhand, India

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ABSTRACT

The aim of present study was to evaluate the clinico-physiological and haemato-biochemical effects of propofol induction and sevoflurane maintenance anaesthesia alone and in combination with dexmedetomidine and butorphanol for producing balanced anaesthesia in canine patients. Twelve healthy dogs of either sex were randomly divided into two groups propofol-sevoflurane (PS) and dexmedetomidine-butorphanol-propofol-sevoflurane (DBPS) with six animals in each group. Atropine sulphate @ 0.04 mg/kg was administered subcutaneously to all the animals. In group DBPS, after administration of atropine sulphate 5 minutes later dexmedetomidine @ 5 µg/kg i/v and butorphanol @ 0.1 mg/kg i/v were administered, respectively. Ten minutes later propofol was administered i/v till effect and maintained with sevoflurane inhalant anaesthesia in both the groups to induce surgical stage of general anaesthesia. The clinico-physiological, haemodynamic and hemato-biochemical parameters were recorded at various time intervals in both the groups of animals. All the data were analysed using SPSS statistics software program and presented as mean±SE. Significantly ($p<0.05$) shorter induction time and significantly ($p<0.05$) longer duration of anaesthesia, recovery time, sternal recumbency time, standing time, complete recovery time and per cent reduction in MAC of sevoflurane was recorded in group DBPS than in group PS. Induction dose reduced in group DBPS after the administration of sedative and analgesic drugs as compared to group PS where no sedative and analgesic drugs were administered. The extent of muscle relaxation, degree of analgesia and abolition of pedal and palpebral reflexes was excellent in group DBPS as compared to group PS. Significant ($p<0.05$) decrease in heart rate in the animals of group DBPS whereas significant ($p<0.05$) decrease in systolic, diastolic and mean arterial pressure in group PS was recorded at respective time interval. Both the groups showed significant ($p<0.05$) decrease in respiration rate and haemoglobin oxygen saturation at respective time interval. Transient changes within physiological limits were observed in haematobiochemical parameters which reached to baseline values by 24 hours. The above anaesthetic study suggests that propofol in combination with dexmedetomidine and butorphanol can be safely used for inducing anaesthesia and for performing longer surgical procedures in dogs.

***Key words*:** Atropinized dog, butorphanol, dexmedetomidine, propofol, sevoflurane

Introduction

Administration of preanaesthetic drug reduces the dose of general anaesthetic agent upto 50%, reduces the salivary secretion and secretion of mucus glands of respiratory tract. They are also used to calm the canine patient so that anaesthetic agents could be injected without any struggling or frightness. Therefore, combination of sedative and analgesic along with other anaesthetic drugs attain desirable effects of general anaesthesia. Among sedatives, dexmedetomidine is a potent and highly selective alpha-2 adrenergic agonist, has sympatholytic, amnestic, sedative and analgesic properties (Carollo *et al*., 2008). Butorphanol, an agonist-antagonist narcotic or analgesic produces mild sedation and limited short duration of analgesia for visceral pain and less painful procedures in the animals (Ahsam *et al.,* 2020). The chemical structure of butorphanol is similar to morphine but its analgesic potency is 3-5 times that of morphine. Propofol is an ultra-short acting non-barbiturate intravenous anaesthetic drug which offers rapid and smooth induction, short period of unconsciousness with minimal analgesia and excitement free recovery in small animal patients (Berry, 2015). Sevoflurane is a volatile ether inhalation anaesthetic agent with lower blood-gas coefficient which produces rapid induction and recovery in the animals (Haitjema and Cullen, 2001). Review reveals scanty literature on the use of dexmedetomidine or butorphanol and its combination with propofol and sevoflurane in dogs. Keeping in view the above, the aim of this study was to evaluate the clinico-physiological, haemodynamic, haemato-biochemical effects of propofol-sevoflurane alone and its combination with dexmedetomidine and butorphanol in canine patients for inducing adequate anaesthesia in dogs with minimal side effects.

Materials and Methods

Ethical statement

All methods employed in this study effort were approved by the Institutional Animal Ethics Committee (IAEC), College of Veterinary and Animal Sciences, Pantnagar, Uttarakhand.

Selection of animals

The present anaesthetic study was conducted on twelve

[1*]PhD. Scholar and corresponding author E mail: ttkanika13@gmail.com, Mob no. 9411701876; [2]Professor; [3]Assistant Professor, Molecular Biology and Genetic Engineering, College of Basic Sciences and Humanities, Govind Ballabh Pant University of Agriculture & Technology, Pantnagar, Uttarakhand. email: shuklapriyanka5@gmail.com

clinically healthy adult dogs aged 1.5 to 8 years and weighing 10-40 kg presented to Dr. I. P. Singh Veterinary Clinical Complex and Trauma Centre, College of Veterinary and Animal Sciences, Govind Ballabh Pant University of Agriculture & Technology, Pantnagar for various surgical affections. They were randomly divided into two groups viz., PS and DBPS, comprising of 6 animals in each group.

Preparation of animals

All animals were kept off fed for 12 hours before administration of anaesthesia. The dogs were subjected to a detailed clinical examination before the start of anaesthetic study and the base values (0 min) for physiological, haemodynamic and haemato-biochemical parameters were recorded as reference. The site was prepared aseptically for administration of drugs and collection of blood samples along with secured intravenous catheterization.

Anaesthetic protocols

All the 12 animals were randomly divided into two groups designated as group PS and DBPS. The animals of all the two groups were premedicated with atropine sulphate @ 0.04 mg/kg subcutaneously. 10 minutes later, the animals of group PS received propofol intravenously till effect for induction and kept as control group. The animals of group DBPS was premedicated with dexmedetomidine @ 5 µg/kg i/v and butorphanol @ 0.1 mg/kg i/v after five minutes interval respectively. Ten minutes later propofol was administered slowly as i/v bolus till effect to induce surgical stage of anaesthesia. Animals of both the groups were maintained with sevoflurane inhalant anaesthesia (1 MAC) after endotracheal intubation.

Clinical parameters

The following clinical parameters were assessed after propofol administration viz., induction time, degree of analgesia, extent of muscle relaxation, palpebral and pedal reflex, duration of anaesthesia, recovery time, sternal recumbency time, standing time, complete recovery time, required doses of induction agents and per cent reduction in MAC of sevoflurane after administration of preanaesthetic drugs. Induction time was recorded as time elapsed from injection of drugs to abolition of pedal reflex. Degree of analgesia was recorded by pinprick reflex, i.e., a reflex on a gentle prick of a needle on the medial aspect of the thigh and interdigital web of the hindfoot. Extent of muscle relaxation was recorded by observing the tone of jaw muscle, flaccidity of abdominal muscles and resistance of the limb to flex by the animals. Palpebral reflex was noted by tapping the skin at the medial canthus of the eye while pedal reflex was noted by pinching the interdigital web of hindfoot of the dog. Score card of muscle relaxation, response to stimuli in pedal and palpebral reflex and response to noxious stimuli by pin prick were studied in animals of both of groups, presented in Table 1 and 2, respectively and were recorded at 0 minute, 5 minute after preanaesthetic administration and then at 5, 10, 15, 20, 30, 45, 60, 75 and 90 minutes after propofol anaesthesia. Duration of anaesthesia was recorded as the time elapsed from the induction time, i.e., time of abolition of pedal reflex to the time of reappearance of pedal reflex in the anaesthetized animal. Recovery time was recorded as the time from discontinuation of drug to the reappearance of pedal reflex in the anaesthetized animal. Sternal recumbency time was recorded as the time from discontinuation of drug to regaining of sternal recumbency by the animal. Standing time was recorded as the time from discontinuation of drug to regaining of standing position by the animal. Complete recovery time was recorded as the time elapsed from discontinuation of drug to the time animal walk unassisted. Any complications viz. nausea, vomiting, salivation, lacrimation, muscle twitching etc. were noted during and after anaesthesia in all the animals.

Table 1: Score card of muscle relaxation for animals of both the groups

Score	Type of muscle relaxation	Description
1	No muscle relaxation	Tightly closed jaws, stiff limbs resisting all attempts to flex and tight abdominal muscles.
2	Mild relaxation	Moderate resistance on opening the jaws and flexing the limbs; mild flaccidity of the abdominal muscles.
3	Moderate relaxation	Mild resistance on opening the jaws and flexing the limbs; moderate flaccidity of the abdominal muscles.
4	Excellent relaxation	No resistance on opening the jaws and flexing the limbs; completely flaccid abdominal muscles.

Physiological parameters

The physiological parameters included rectal temperature, heart rate and respiration rate were recorded at 0 minute, 5 minute after preanaesthetic administration and then at 5, 15, 30, 45, 60, 75, 90 and 180 minutes after propofol anaesthesia.

Haemodynamic parameters

The haemodynamic parameters included systolic arterial blood pressure, diastolic arterial blood pressure, mean arterial blood pressure and haemoglobin oxygen saturation were recorded at 0 minute, 5 minute after preanaesthetic administration and then at 5, 15, 30, 45, 60, 75, 90 and 180 minutes after propofol anaesthesia.

Table 2: Score card of pedal reflex, palpebral reflex and analgesia for animals of both the groups.

Clinical parameters	Score			
	0	1	2	3
Pedal reflex	Intact and strong (strong withdrawal)	Intact but weak (animal responding slowly)	Intact but very light (slow and occasional response)	Abolished
Palpebral reflex	Intact and strong (quick blink)	Intact but weak (slow response)	Very weak (very slow and occasional)	Abolished
Analgesia	Strong reaction to pin prick (No analgesia)	Weak response to pin prick (Mild analgesia)	Occasional response to pin prick (Moderate analgesia)	No response to pin prick (Excellent analgesia)

Haemato-biochemical parameters

The haematological parameters included haemoglobin, total erythrocyte count, total leucocyte count, differential leucocyte count was estimated by automatic hematology analyzer (Medonic Cell Counter). The biochemical parameters included serum glucose, serum total protein, serum albumin, serum creatinine, serum urea nitrogen, serum aspartate amino transferase and serum alanine amino transferase were estimated using commercial test kit (Erba®) in semi-automatic biochemical analyzer. Both the parameters were recorded at 0 minute, 30 minute, 1 hour, 6 hour and 24 hour during anaesthetic study.

Statistical analysis

The mean and standard error of the recorded values were calculated and data was analyzed using simple t-test with Microsoft Excel 2019 and analysis of variance (ANOVA) with SPSS statistics software program. Non-parametric data was analysed by Kruskal-Wallis H test. $p<0.01$ and $p<0.05$ were considered as significant.

Results and Discussion

Clinical parameters

The mean induction time was significantly ($p<0.01$) shorter in animals of group DBPS (32.83±0.60 sec) than in group PS (68.00±0.96 sec). Shorter induction time in animals of group DBPS might be due to synergistic action of dexmedetomidine and propofol respectively. Bisht *et al*. (2018) also reported shorter induction time in dogs preanaesthetized with dexmedetomidine prior to induction with propofol. The mean duration of anaesthesia was significantly ($p<0.01$) higher in animals of group DBPS (81.83±1.58 min) than in group PS (54.83±2.85 min). The dose dependent sedation produced by preanaesthetic administration of dexmedetomidine and butorphanol influenced the duration of anaesthesia in group DBPS which was also noted by Khattri *et al.* (2013) in buffalo calves preanaesthetized with dexmedetomidine and butorphanol and induced with propofol.

The mean recovery time, mean sternal recumbency time, mean standing time and mean complete recovery time were 5.83±0.54 min, 8.83±0.54 min, 10.33±0.80 min and 11.50±0.92 min in group PS whereas 8.33±0.42 min, 10.83±0.31 min, 13.33±0.49 min and 14.83±0.60 min in group DBPS, respectively. In group DBPS, mean recovery time, mean sternal recumbency time, mean standing time and mean complete recovery time were significantly ($p<0.01$) more as compared to group PS which might be due to premedication of sedative and analgesic drugs. Similar results were also reported by Jadon *et al.* (1998); Singh *et al.* (2013) and Saini *et al.* (2017) after premedication of alpha 2-agonist drugs. Although recovery was smooth, free from excitement and uncomplicated in both the groups after anaesthesia. Induction dose of propofol calculated for group PS was 5.00 ± 0.06 mg/kg and in group DBPS was 2.16 ± 0.01 mg/kg. Preanaesthetic administration of dexmedetomidine and butorphanol reduces the induction dose of propofol which was also reported by Hoy and Keating (2011) and Bisht *et al*. (2018) in dexmedetomidine premedicated and propofol induced dogs. Per cent reduction in the MAC of sevoflurane was 18.19±2.62 in group II which agreed with the finding of Bisht *et al*. (2018) who has also noted reduction in the MAC of sevoflurane in dexmedetomidine premedicated dogs. Muscle relaxation was moderate after 5 minutes of preanaesthetic administration of dexmedetomidine and butorphanol in group DBPS.

However, excellent quality of muscle relaxation for longer duration was observed after induction with propofol in group DBPS as compared to group PS (Fig. 1). Pedal and palpebral reflexes were very weak in group DBPS after 5 minutes of preanaesthetic administration of dexmedetomidine and butorphanol, which got completely abolished at 5 minutes post-induction with propofol in both the groups. Abolition of pedal and palpebral reflexes for longer duration were observed in

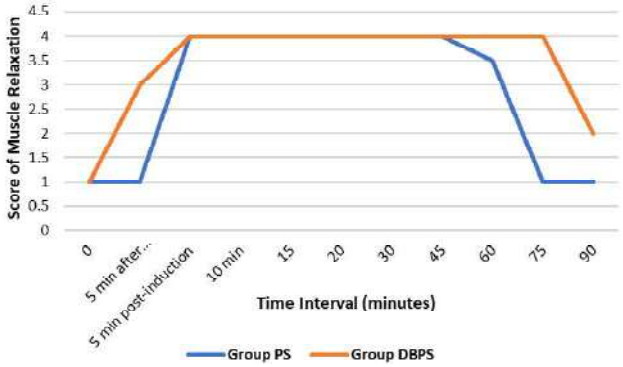

Fig. 1: Scoring of muscle relaxation in both the groups at various time intervals.

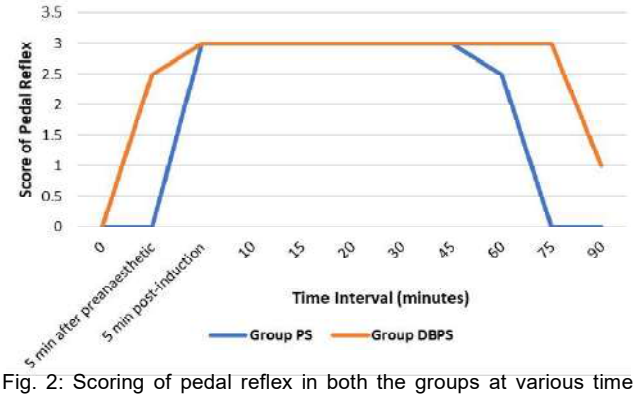

Fig. 2: Scoring of pedal reflex in both the groups at various time intervals.

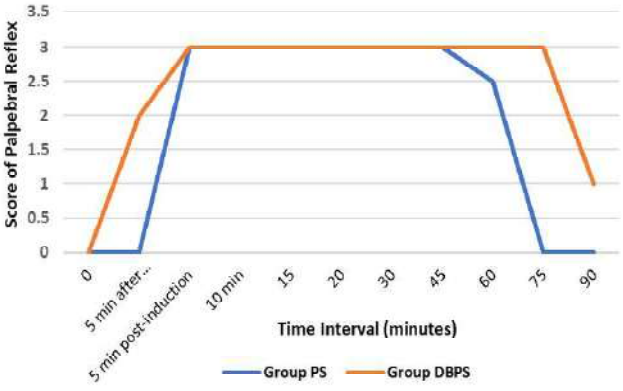

Fig. 3: Scoring of palpebral reflex in both the groups at various time intervals.

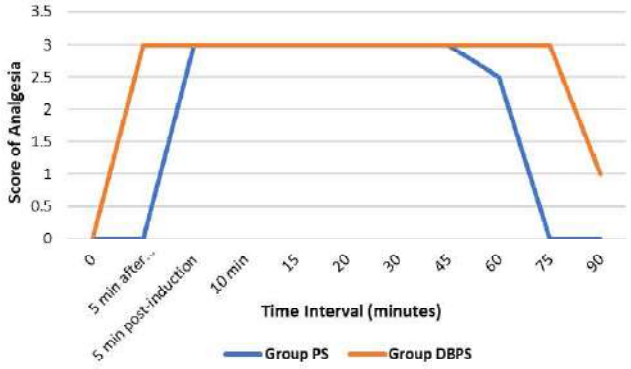

Fig. 4: Scoring of analgesia in both the groups at various time intervals.

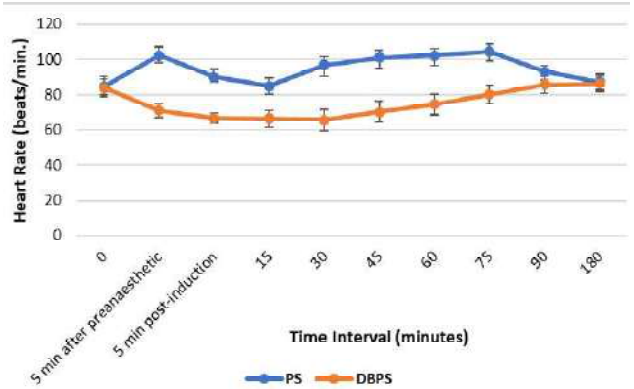

Fig. 6: Effect on heart rate in both the groups at various time intervals.

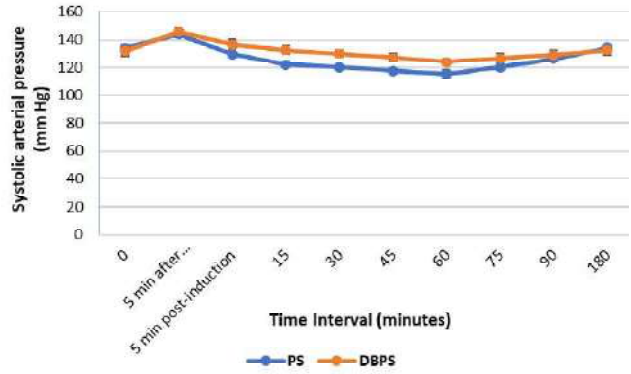

Fig. 8: Effect on systolic arterial pressure in both the groups at various time intervals.

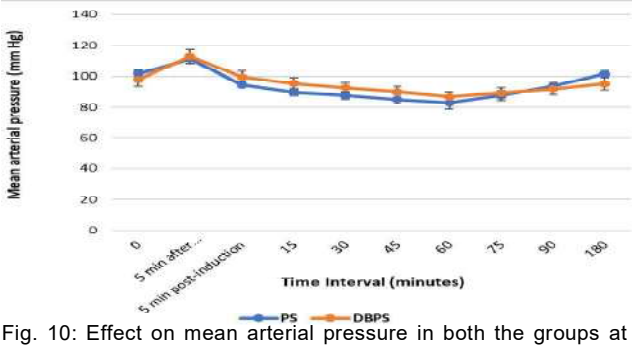

Fig. 10: Effect on mean arterial pressure in both the groups at various time intervals.

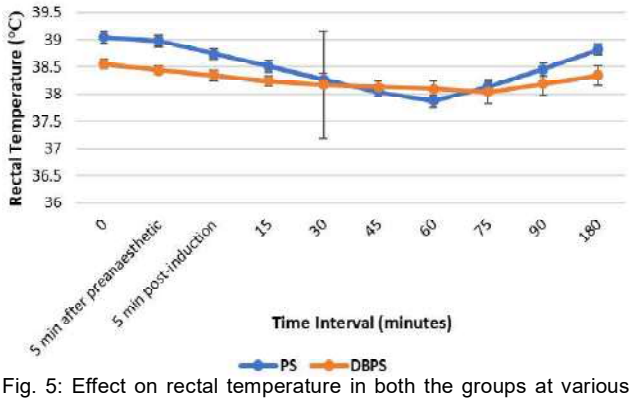

Fig. 5: Effect on rectal temperature in both the groups at various time intervals.

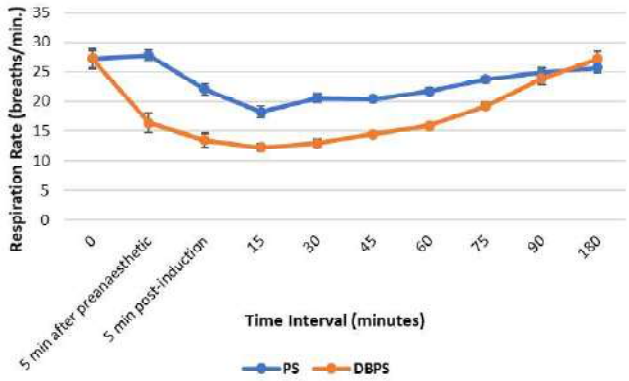

Fig. 7: Effect on respiration rate in both the groups at various time intervals.

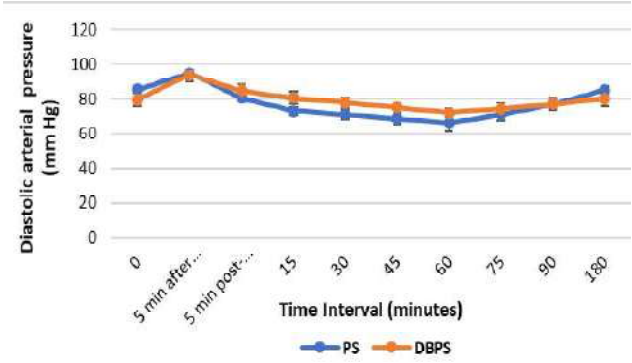

Fig. 9: Effect on diastolic arterial pressure in both the groups at various time intervals.

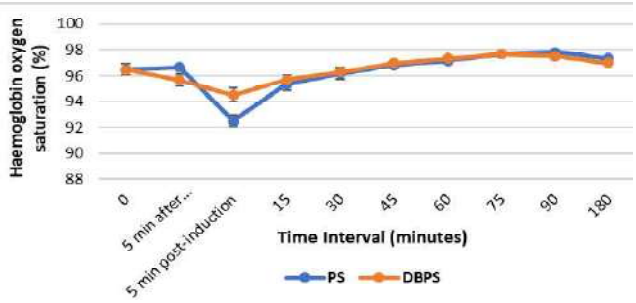

Fig. 11: Effect on haemoglobin oxygen saturation in both the groups at various time intervals.

group DBPS as compared to group PS (Fig. 2 & 3). Dexmedetomidine as alpha 2 agonist is known to produce good muscle relaxation which might be attributed to inhibition of intraneuronal transmission of impulses at the level of CNS by alpha 2 agonist (Lemke, 2007). Similar results were also noted by Khattri *et al*. (2013) and Sharma *et al.* (2014) which might be attributed to synergistic effect of the drugs used in the study. Moderate analgesia was observed in group DBPS after 5 minutes of premedication with dexmedetomidine and butorphanol, which thereafter produces excellent quality of analgesia for longer duration at 5 minutes post-induction with propofol in the animals of group DBPS as compared to animals of group PS (Fig. 4). Excellent analgesia might be attributed to the synergistic action of butorphanol and dexmedetomidine as propofol provides minimal analgesia which was also noted by Kuusela *et al*. (2000) and Neto *et al.* (2007). The analgesic effect of dexmedetomidine is mediated at spinal level and by interruption of nociceptive pathways to the ventral root of the dorsal horn which reduces spinal reflexes (Talukder and Hikas, 2009). Butorphanol exerts its analgesic effect by inhibiting the transmission of nociceptive stimulation in the dorsal horn of the spinal cord, activating descending inhibitory pathways, inhibiting supra spinal afferent pathways and decreasing the release of neurotransmitters in the spinal cord (Schnellbacher, 2010).

Physiological parameters

In group PS, a non-significant decrease in rectal temperature was recorded from 5 min after preanaesthetic administration to 5 min post-induction. In group DBPS, non-significant decrease was recorded from 5 min after preanaesthetic administration to 75 min post-induction. Thereafter, the values returned to the base line at 180 minutes (Fig. 5). This might be due to decrease in production of body heat due to sedation and reduced muscular activity. Activation of alpha-2C adrenergic receptors by dexmedetomidine might also have contributed to hypothermia (Lemke, 2007). Heart rate was significantly ($p<0.05$) decreased from 5 min after preanaesthetic administration to 30 min post-induction in group DBPS which might be due to reflex bradycardia and induced vasoconstriction caused by alpha-2 agonist (Fig. 6). Bradycardia was observed in the present study which supports the reports of earlier studies (Bisht *et al*., 2016). It has been reported that butorphanol facilitates the increase in parasympathetic tone and thereby contributes to bradycardia (Ko *et al*., 2000). There was significant ($p<0.05$) decrease in respiration rate from 5 min to 15 min post-induction in group PS and from 5 min after preanaesthetic administration to 15 min post-induction in group DBPS, which might be due to dose dependant depression produced by dexmedetomidine and propofol (Fig. 7). Similar results were also reported by Kandpal *et al*. (2005) and Kumar *et al.* (2016) after preanaesthetic administration of alpha-2 agonist. All the physiological parameters exhibited transient changes and were maintained within the normal physiological limits in all the animals during the study period.

Haemodynamic parameters

In group PS, systolic arterial blood pressure and diastolic arterial blood pressure was significantly ($p<0.05$) decreased at 15 minutes to 75 minutes post-induction whereas it was non-significantly increased at 5 min after preanaesthetic administration to 15 minutes post-induction and non-significant decrease was observed at 30 minutes to 90 minutes time interval in group DBPS as compared to baseline value (Fig. 8 & 9). Similarly, mean arterial blood pressure was significantly ($p<0.05$) decreased at 30 min to 60 min post-induction in group PS whereas it was non-significantly increased at 5 min after preanaesthetic administration and non-significantly decreased at 5 min to 60 min post-induction in animals of group DBPS. Thereafter, it increased gradually after 75 min in both the groups (Fig. 10). Initial rise in arterial pressure may be due to dexmedetomidine which increases the arterial blood pressure due to vasoconstriction which results from the stimulation of alpha-2B adrenoreceptors in the smooth muscles of blood vessels by the dexmedetomidine (Bisht *et al.,* 2018). However at later stage, arterial blood pressure was decreased which could be due to the predominant action of alpha-2A adrenoreceptors by dexmedetomidine with other anaesthetics (Lemke 2007). Haemoglobin oxygen saturation was significantly ($p<0.05$) decreased at 5 min post-induction in both the groups as compared to baseline values which could be due to either respiratory depressant effect of propofol alone or combined effect of preanaesthetic drugs dexmedetomidine and butorphanol along with induction agent propofol (Fig. 11). Similar findings were also reported by Singh *et al.* (2013) and Bisht *et al.* (2018).

Haemato-biochemical parameters

In group DBPS, the values of haemoglobin was significantly ($p<0.05$) decreased from 1 hour to 6 hour interval which might be attributed to blood pooling in spleen as a result of reduced sympathetic tone caused by alpha-2 agonist drugs (Jadon *et al.,* 1995). No significant changes were observed in the total erythrocyte count, total leucocyte count and differential leucocyte count at respective time interval in both the groups (Table 3). In group PS, serum glucose was significantly ($p<0.05$) increased at 1 hour whereas it was significantly ($p<0.05$) increased from 30 min to 6 hour in group DBPS, which might be either due to inhibitory effects of dexmedetomidine on alpha-2 receptors of beta cells which inhibit insulin secretion and increase glucose production (Ahmad *et al*., 2013) or increased liberation of glucocorticoid during anaesthesia (Singh *et al.,* 2010). Non-significant decrease in total protein was recorded from 30 minutes to 1 hour in both the groups and serum albumin was non-significantly decreased from 30 minutes to 1 hour in group PS whereas it was significantly ($p<0.05$) decreased from 30 minutes to 6 hour in group DBPS as compared to baseline values, which might be attributed to inter-compartmental shifting of fluid which leads to haemodilution (Jadon *et al.,* 1995; Saini *et al.,* 2019). Non-significant rise in serum urea nitrogen from 30 min to 1 hour in group PS, significant ($p<0.05$) rise at 1 hour in group DBPS and non-significant rise in creatinine from 30 min to 1 hour in both the groups which might be attributed to the action of anaesthetic drugs on the temporary inhibition of blood flow in the renal system which decreased the glomerular filtration rate (Umar and

Table 3: Mean ± SE of haematological parameters in group I and II

Parameters	Groups	Time Interval				
		0 min	30 min	1 hr	6 hr	24 hr
Hb(g/dl)	PS	12.61±0.33	12.46±0.32	11.81±0.32	11.89±0.32	12.55±0.29
	DBPS	12.47±0.22^{a}	12.24±0.14^{ab}	11.54±0.14^{c}	11.91±0.11^{bc}	12.48±0.18^{a}
TEC (10^6/µL)	PS	6.67±0.36	6.50±0.36	6.35±0.37	6.49±0.36	6.68±0.35
	DBPS	6.65±0.33	6.37±0.32	6.19±0.32	6.30±0.33	6.60±0.35
TLC (10^3/µL)	PS	10.71±0.43	10.29±0.43	10.05±0.42	10.18±0.42	10.57±0.44
	DBPS	11.05±0.38	10.44±0.39	10.14±0.36	10.33±0.33	10.87±0.37
N (%)	PS	71.99±1.72	72.60±1.77	72.91±1.72	72.72±1.78	72.26±1.73
	DBPS	68.95±2.04	69.73±2.03	70.14±1.98	69.69±2.04	69.17±2.09
L (%)	PS	23.33±1.76	22.17±1.72	21.43±1.54	21.83±1.56	22.33±1.76
	DBPS	26.67±2.09	24.83±1.97	23.91±1.97	25.31±2.05	26.00±1.91
M (%)	PS	6.00±0.36	5.33±0.21	5.00±0.00	5.10±0.28	5.58±0.37
	DBPS	5.89±0.48^{a}	4.35±0.49^{ab}	3.65±0.54^{b}	4.75±0.47^{ab}	5.00±0.36^{ab}
E (%)	PS	1.50±0.22	1.85±0.31	2.05±0.26	1.93±0.21	1.63±0.31
	DBPS	1.33±0.21^{b}	2.03±0.23^{ab}	2.33±0.21^{a}	1.89±0.18^{ab}	1.67±0.25^{ab}

The pair of values with different superscript vary significantly ($p<0.05$)

Table 4: Mean ± SE of biochemical parameters in group I and II

Parameters	Groups	Time Interval				
		0 min	30 min	1 hr	6 hr	24 hr
Glucose (mg/dl)	PS	73.08±2.60^{b}	81.33±2.57^{ab}	86.41±2.49^{a}	82.45±2.58^{ab}	76.83±2.62^{ab}
	DBPS	80.39±2.86^{c}	96.47±2.84^{ab}	106.72±2.84^{a}	99.70±2.61^{a}	85.65±2.70^{bc}
Total protein (g/dl)	PS	6.36±0.36	6.21±0.37	6.12±0.39	6.16±0.36	6.28±0.35
	DBPS	6.82±0.14	6.53±0.16	6.38±0.13	6.45±0.13	6.75±0.13
Serum albumin (g/dl)	PS	2.99±0.12	2.71±0.11	2.67±0.11	2.73±0.12	2.88±0.12
	DBPS	2.93±0.09^{a}	2.50±0.09^{bc}	2.40±0.09^{c}	2.51±0.09^{bc}	2.83±0.09^{ab}
Serum urea nitrogen (mg/dl)	PS	20.81±0.88	22.15±0.90	22.94±0.93	22.62±0.94	21.91±0.94
	DBPS	20.52±0.85^{b}	22.69±0.86^{ab}	23.88±0.83^{a}	22.31±0.82^{ab}	22.54±0.81^{ab}
Serum Creatinine (mg/dl)	PS	1.43±0.14	1.57±0.13	1.61±0.14	1.52±0.13	1.41±0.14
	DBPS	1.40±0.14	1.48±0.14	1.53±0.14	1.45±0.14	1.32±0.15
Alanine amino transferase (IU/L)	PS	34.44±1.47	36.36±1.56	37.81±1.57	36.75±2.18	33.57±1.46
	DBPS	34.65±1.38	37.19±1.43	39.01±1.64	37.66±1.39	34.36±1.36
Aspartate amino transferase (IU/L)	PS	27.21±1.45	27.61±1.62	27.96±1.59	27.51±1.62	26.27±1.60
	DBPS	27.28±1.49	27.93±1.37	28.38±1.57	27.94±1.59	26.61±1.55

The pair of values with different superscript vary significantly ($p<0.05$)

Adam, 2013; Mazumdar *et al.,* 2015). There was non-significant increase in the values of serum alanine amino transferase and serum aspartate amino transferase from 30 min to 1 hour interval and reached to baseline values at 24 hours in both the groups (Table 4). It was in accordance with the results of Sharma *et al.* (2014) and Bisht *et al.* (2016). The permeability of cell membrane in liver or muscle tissues changes due to hypoxia created as a result of respiratory depression due to administration of alpha-2 adrenergic agonist which in turn causes changes in the level of liver enzymes (Kalim *et al.,* 2011). The values of haematobiochemical parameters returned to their baseline values by 24 hours in animals of both the groups.

Conclusion

The findings of the present study conclude that dexmedetomidine-butorphanol-propofol-sevoflurane anaesthetic combination produce satisfactory anaesthesia, excellent quality of muscle relaxation and analgesia, complete abolition of pedal and palpebral reflexes with transient changes in physiological and haematobiochemical parameters as compared to propofol-sevoflurane anaesthesia. Hence, dexmedetomidine @ 5 µg/kg i/v and butorphanol @ 0.1 mg/kg i/v can be recommended for clinical use in the dogs undergoing propofol and sevoflurane anaesthesia.

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