Indian Journal of Animal Sciences **88** (8): 887–891, August 2018/Article

Clinicophysiological and haematobiochemical effects of dexmedetomidine-propofol-sevoflurane anaesthesia in dogs

DEVENDER SINGH BISHT[1], NARENDRA SINGH JADON[2], DEEPTI BODH[3] and MANJUL KANDPAL[4]

Gobind Ballabh Pant University of Agriculture and Technology, Pantnagar, Uttarakhand 263 145 India

Received: 7 March 2018; Accepted: 17 May 2018

ABSTRACT

The present study was conducted to evaluate the clinicophysiological and haematobiochemical effects of dexmedetomidine in dogs undergoing propofol-sevoflurane anaesthesia. Twelve apparently healthy adult dogs were divided into two groups having 6 animals each. Animals of group I received atropine sulphate @ 0.04 mg/kg s.c. + dexmedetomidine @ 10 µg/kg i.v. while animals of group II were administered atropine sulphate @ 0.04 mg/kg s.c. + dexmedetomidine @ 15 µg/kg i.v. Anaesthesia was induced with propofol (as i.v. bolus till effect) and maintained with sevoflurane. Clinicophysiological and haematobiochemical parameters were recorded at different intervals. Quicker attenuation of clinical reflexes was observed in both groups. Induction time was significantly lower while duration of anaesthesia, recovery time, standing time, complete recovery time and percent reduction in MAC of sevoflurane was significantly higher in group II. Non-significant differences in induction dose of propofol, physiological and haematobiochemical parameters were observed in both groups. Significant decrease in heart rate, respiration rate, rectal temperature, haemoglobin oxygen saturation and significant increase in mean arterial pressure was recorded in both the groups. Transient significant decrease in haemoglobin, total leukocyte count, total erythrocyte count and transient significant increase in glucose, urea nitrogen, creatinine, alanine aminotransferase, aspartate aminotransferase and cortisol was recorded in both the groups. Erythrocyte sedimentation rate increased significantly while insulin level decreased significantly in both groups. Both anaesthetic combinations used in the present study produced satisfactory anaesthesia and muscle relaxation, therefore can be suggested for clinical use in canine patients undergoing propofol-sevoflurane anaesthesia.

Key words: Anaesthesia, Dexmedetomidine, Dog, Propofol, Sevoflurane

A number of drugs including alpha-2 agonists have been used to produce different levels of sedation and analgesia in canine patients. Dexmedetomidine, a potent and highly selective alpha-2 adrenergic agonist having sympatholytic, sedative, amnestic and analgesic properties (Carroll *et al.* 2008) has been described as a safe adjunct in many clinical applications. Propofol is a rapidly metabolized, nonbarbiturate sedative/hypnotic anaesthetic drug which offers rapid, smooth induction and recovery from anaesthesia in small animal patients (Glowaski and Wetmore 1999). Sevoflurane is a volatile anaesthetic agent with low blood-gas partition coefficient which produces rapid induction and recovery of anaesthesia (Haitjema and Cullen 2001).With the exception of few studies (Bisht *et al.* 2016) limited information is available on the clinicophysiological and haematobiochemical effects of dexmedetomidine-propofol-sevoflurane anaesthesia in dogs. The aim of present study was to evaluate the effects of two doses of dexmedetomidine on the clinico-physiological and haematobiochemical parameters in canine patients under propofol and sevoflurane anaesthesia.

Present address: [1]Ph.D. Scholar (drdsbisht5@gmail.com), [2]Professor and Head (drjadonns12 @rediffmail.com), [3]Assistant Professor (deeptibodh@yahoo.in), [4]Associate Professor (manjulkandpal@yahoo.com), Department of Surgery and Radiology, College of Veterinary and Animal Sciences.

MATERIALS AND METHODS

The study was conducted in accordance with the guidelines laid down by the Institutional Animal Ethics Committee. Twelve clinically healthy, adult dogs of either sex aged 3–5 year and weighing 10–15 kg were included in the study. All dogs were kept off feed for 12 h before administration of anaesthetic agents. The dogs were divided randomly into two groups having six animals each. Premedication was done with atropine sulphate (@ 0.04 mg/kg b.wt. s.c.), followed by dexmedetomidine (@ 10µg/kg b.wt. i.v.) in group I and dexmedetomidine (@ 15µg/kg b.wt. i.v.) in group II after 5 min. Ten minute later, anaesthesia was induced with propofol (as bolus i.v. till effect) in both groups followed by endotracheal intubation. Maintenance of anaesthesia was done with sevoflurane (in oxygen, 2l/min) at vaporizer setting of 2.2% (1 MAC). An equilibrium period of 15 min was allowed before proceeding with measurements for MAC of sevoflurane by tail clamp method (Itami *et al.* 2013). The average of lowest MAC

Table 1. Score card of muscle relaxation in animals of group I and II

Score	Description
1 (no muscle relaxation)	Tightly closed jaws, stiff limbs resisting any attempt to flex and tight abdominal muscles
2 (mild muscle relaxation)	Moderate resistance to opening of the jaws and flexing of the limbs, mild flaccidity of the abdominal muscles
3 (moderate muscle relaxation)	Mild resistance to opening of the jaws and flexing of the limbs, moderate flaccidity of the abdominal muscles.
4 (excellent muscle relaxation)	No resistance to opening of the jaws and flexing of the limbs, completely flaccid abdominal muscles

Table 2. Score card of pedal and palpebral reflexes in animals of group I and II

Score	Description
1	Excellent response to stimulus
2	Moderate response to stimulus
3	Mild response to stimulus
4	Completely abolished response

Table 3. Various clinical parameters recorded in animals of group I and II

Parameter	Description
Induction time (sec)	Time elapsed from propofol administration to induction of anaesthesia (adjudged by loss of pedal reflex)
Duration of anaesthesia (min)	Time elapsed from induction of anaesthesia to first spontaneous movement of any body part (after detachment of sevoflurane)
Recovery time (min)	Time from discontinuation of sevoflurane to first spontaneous movement of any body part
Sternal recumbency time (min)	Time from discontinuation of sevoflurane administration to regaining of sternal recumbency
Standing time (min)	Time from discontinuation of sevoflurane administration to regaining of standing position
Complete recovery time (min)	Time from discontinuation of sevoflurane administration to walking of dog without any ataxia

with negative tail clamp response and highest MAC with positive tail clamp response was taken as MAC for any particular measurement. Blood samples were collected at 0 min, 30 min, 60 min, 6 h and 24 h interval for haematobiochemical evaluation. Haemoglobin (Hb), total erythrocyte count (TEC), total leucocyte count (TLC), erythrocyte sedimentation rate (ESR) were estimated by standard procedures (Jain 1986). Serum glucose, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were estimated using commercial test kits (Autospan®, Arkray Healthcare Pvt. Ltd, Surat, India). Serum insulin and cortisol were estimated using commercial insulin and cortisol ELISA kits (Calbiotech Inc. Spring Valley, CA, USA). Serum urea nitrogen and creatinine were estimated using commercial kits (ERBA, Transasia Bio-Medicals Ltd., Solan, Himachal Pradesh, India). Clinical parameters, viz. induction time, duration of anaesthesia, recovery time, sternal recumbency time, standing time, complete recovery time, required doses of induction agent and sevoflurane MAC reduction were recorded. Physiological parameters, viz. heart rate (HR), respiration rate (RR), rectal temperature (RT) and haemoglobin oxygen saturation (SPO_2) were recorded at 0 min, 15 min, 30 min, 60 min, 90 min, 6 h and 24 h interval. Score card of muscle relaxation, response to stimuli in pedal and palpebral reflexes and description of various clinical parameters recorded in animals of group I and II are presented in Tables 1, 2 and 3, respectively. Required dose of propofol (mg/kg b.wt.) was calculated after completion of each trial. Data were analyzed by using SPSS software version 15 (SPSS, Inc., Chicage, IL). Means at different time intervals between two groups were compared using one way analysis of variance (ANOVA) and Dunkan's multiple range tests (DMRT).

RESULTS AND DISCUSSION

Mean±SE values of clinical, physiological, haematological and biochemical parameters in group I and II are presented in Tables 4, 5, 6 and 7, respectively. Induction time was significantly ($P<0.05$) lower in group II (Table 4) which was in accordance with findings of Bisht *et al.* (2016) who reported significantly lower induction time in dogs administered higher dose of dexmedetomidine. Duration of anaesthesia was significantly ($P<0.01$) higher in group II (Table 4). The dose dependent sedation produced by different doses of dexmedetomidine influenced duration of anaesthesia in group II. Bisht *et al.* (2016) reported increase in level of sedation with incremental doses of dexmedetomidine in dogs. Although recovery from anaesthesia was smooth and uneventful in both groups, significantly ($P<0.05$) longer recovery time, standing time and complete recovery time in group II (Table 4) might be due to administration of higher dose of dexmedetomidine. Contrary to this, Bisht *et al.* (2016) reported non-significant differences in recovery times in dogs administered dexmedetomidine-etomidate-sevoflurane anaesthesia. Hoy and Keating (2011) reported that low dose of dexmedetomidine reduces propofol infusion dose but does not affect its pharmacokinetics or recovery time after induction. Propofol induction dose was non-significantly lower in group II which was in accordance with findings of Kussela *et al.* (2001) who observed significant effect of dose level of premedicant on induction dose of propofol in dexmedetomidine premedicated dogs. Induction dose of propofol in group I (2.46±0.19 mg/kg) and group II (2.45±0.20 mg/kg) was in accordance with the reported induction dose of 2.2–3.3 mg/kg (Dewangan *et al.* 2010).

Percentage reduction in the MAC of sevoflurane was significantly (P<0.05) higher in group II. Bisht *et al.* (2016) reported similar findings in dogs administered dexmedetomidine-etomidate-sevoflurane. Adequate muscle relaxation was observed in animals of both the groups. Muscle relaxation score was 1 at 5 min, 3 at 10 min, thereafter increased gradually to score of 4 from 15 min onwards to 60 min (group I) and 75 min (group II). The muscle relaxation property of dexmedetomidine was attributed to inhibition of alpha-2 adrenoceptors in intraneuronal level of central nervous system (Lemke 2007). Median pedal and palpebral reflex score was 1 at 5 min, 2 till 15 min, then increased gradually to score of 4 from 20 min onwards to 75 min before decreasing to score of 1 at 90 min interval in both groups. Both reflexes were depressed mildly after dexmedetomidine administration but abolished completely after propofol administration suggesting that surgical stage of anaesthesia has reached. Combined synergistic effects of drugs administered along with dexmedetomidine and high specificity of dexmedetomidine for alpha-2 adrenoreceptors might have caused attenuation of reflexes (Jena *et al.* 2014).

Table 4. Mean (±SE) of clinical parameters in animals of group I and II

Parameter	Group I	Group II
Induction time (seconds)	179.00±23.05	108.00±7.73*
Duration of anaesthesia (minutes)	70.00±23.05	85.00±7.73**
Recovery time (minutes)	3.17±0.64	6.48±0.38*
Sternal recumbency time (minutes)	7.61±0.87	8.31±0.78
Standing time (minutes)	11.00±0.89	13.00±2.79*
Complete recovery time (minutes)	14.00±0.94	24.16±4.71*
Dose of propofol (mg/kg)	2.46±0.19	2.45±0.20
MAC of sevoflurane (% reduction)	13.60±0.08	22.70±0.20*

*Values differ significantly between groups (P<0.05). **Values differ more significantly between groups (P<0.01)

Significant (P<0.01) decrease in heart rate from 15 min to 90 min in both groups might be attributed to reflex bradycardia caused by alpha-2 agonist induced vasoconstriction (Lemke 2007). Bradycardia observed in

Table 5. Mean (±SE) of physiological parameters in group I and II

Parameter	Group	Time interval						
		0 min	15 min	30 min	60 min	90 min	6 h	24 h
Heart rate (beat/min)	I	82.1±21.10	70.83±7.54**	65.33±4.37**	61.33±7.03**	60.1±4.48**	81.5±3.08	82.1±2.82
	II	84.6±0.97	72.33±3.06**	67.00±3.25**	64.3±2.18**	62.83±2.61**	81.6±2.15	81.5±1.93
Respiration rate (breath/min)	I	18.1±0.77	15.33±1.83**	14.13±3.29**	14.2±1.04**	16.83±1.07	17.7±1.97	16.5±0.97
	II	18.5±3.23	14.17±2.22**	15.00±0.49**	13.0±0.97**	15.83±0.46**	19.6±0.73	18.8±1.40
Rectal temperature (ºC)	I	38.6±0.41	38.40±0.55	38.07±0.55**	37.4±0.40**	38.0±0.39**	38.2±0.28**	38.6±0.17
	II	38.7±0.27	37.96±0.55**	37.90±0.59**	37.60±0.42**	38.00±0.34**	38.2±0.37**	38.7±0.30
Mean arterial pressure (mm Hg)	I	106.82±12.02	140.6±14.82**	90.48±7.21**	78.75±10.09**	79.30±5.30**	102.8±2.72	104.9±6.07
	II	108.62±1.63	142.4±1.94**	91.00±2.38**	80.39±4.50**	79.00±2.03**	104.9±2.25	102.4±3.29
Haemoglobin oxygen saturation (SpO_2) (%)	I	97.83±0.18	96.67±0.73**	96.33±0.54**	97.00±0.28*	97.07±0.73*	96.67±0.54**	97.23±0.40
	II	98.00±0.00	97.33±0.61*	96.17±0.34**	97.10±0.59**	97.53±1.00*	97.40±1.05*	97.83±0.72

*Significantly different from base value (P<0.05). **Significantly different from base value (P<0.01)

Table 6. Mean (±SE) of haematological parameters in group I and II

Parameter	Group	Time interval				
		0 min	30 min	60 min	6 h	24 h
Haemoglobin (g/dl)	I	12.37±0.30	12.23±0.32	11.60±0.42*	11.63±0.42*	12.7±0.34
	II	12.40±0.48	12.13±0.61	12.22±0.60	11.60±0.40*	12.30±0.67
Total leukocyte count (10^3/µl)	I	6.60±0.23	6.00±0.28**	5.90±0.16**	6.38±0.30	6.38±0.14
	II	5.99±0.38	5.70±0.24	5.50±0.37**	5.49±0.24**	5.80±0.46
Total erythrocyte count (10^6/µl)	I	5.85±0.18	5.62±0.14*	5.47±0.20**	5.50±0.07**	5.80±0.14
	II	5.87±0.12	5.73±0.21	5.70±0.18	5.70±0.32	5.90±0.23
Erythrocyte sedimentation rate (mm/hr)	I	2.50±0.24	2.67±0.46	3.10±0.34**	2.90±0.37	2.40±0.37
	II	2.33±0.23	2.50±0.23	3.00±0.40**	2.67±0.46	2.40±0.72

*Significantly different from base value (P<0.05). **Significantly different from base value (P<0.01)

Table 7. Mean (±SE) of biochemical parameters in group I and II

Parameter	Group	Time interval				
		0 min	30 min	60 min	6 h	24 h
Glucose (mg/dl)	I	75.0±5.29	89.33±6.73**	98.33±9.24**	112.5±6.60**	80.00±5.23
	II	74.1±5.18	90.50±6.75**	103.8±7.93**	114.1±3.29**	78.50±5.05**
Insulin (µU/ml)	I	14.00±1.30	12.67±1.29*	11.90±1.04**	11.00±0.80**	13.00±1.30
	II	14.00±1.15	12.50±1.04*	12.10±1.43**	10.83±0.72**	12.53±1.28
Cortisol (µg/dl)	I	2.83±0.20	3.00±0.23	3.25±0.25**	3.30±0.22**	2.90±0.20
	II	2.65±0.04	2.87±0.04	3.40±0.03**	3.50±0.05**	2.84±0.05
Blood urea nitrogen (mg/dl)	I	22.50±1.22	24.33±2.93	25.50±4.81*	24.00±1.77	24.10±1.67
	II	19.50±1.76	21.50±1.83	23.67±3.98**	22.10±3.01**	21.50±2.45
Creatinine (mg/dl)	I	0.75±0.04	0.85±0.11	0.92±0.18**	0.89±0.04*	0.69±0.04
	II	0.73±0.07	0.89±0.08**	0.93±0.09**	0.82±0.03	0.68±0.04
Alanine aminotransferase (IU/l)	I	32.50±1.87	34.83±2.83	36.50±4.04**	33.50±1.76	32.83±1.48
	II	34.00±2.99	36.67±3.06	39.50±3.29**	35.50±3.39	33.17±1.91
Asparatate aminotransferase (IU/l)	I	22.00±4.69	25.67±2.71	28.67±1.67**	25.33±2.71*	24.67±2.71
	II	24.83±2.11	26.00±2.68	29.17±1.93**	25.60±2.48	24.33±1.83

*Significantly different from base value (P<0.05). **Significantly different from base value (P<0.01).

present study supported the findings of earlier studies (Kuusela *et al.* 2001, Bisht *et al.* 2016). Significant (P<0.01) decrease in respiration rate from 15 min onwards till 60 min (group I) and 90 min (group II) might be due to dose dependent depression in respiration rate produced by dexmedetomidine. Further decrease in respiration rate in both groups may be explained by the action of propofol with dexmedetomidine. Significant (P<0.01) decrease in rectal temperature from 30 min to 6 h (group I) and 15 min to 6 h (group II) might be attributed to a possible decrease in heat production due to sedation and decreased muscular activity. Activation of alpha-2C receptors by dexmedetomidine in both groups might also have contributed to hypothermia (Lemke 2007). Mean arterial pressure increased significantly (P<0.01) at 15 min, then decreased significantly (P<0.01) from 30 min to 90 min in both groups. Initial rise in mean arterial pressure may be attributed to high dose of dexmedetomidine which produces hypertension due to vasoconstriction resulting from stimulation of alpha-2B adrenoceptors in the smooth muscles of blood vessels. Decreased mean arterial pressure at later stages could be due to predominant action of alpha-2A adrenoceptors by dexmedetomidine with other anaesthetics (Lemke 2007). Significant (P<0.01) decrease in haemoglobin oxygen saturation from 15 min onwards till 6 h interval in both groups could be due to combined effect of dexmedetomidine induced vasoconstriction along with respiratory depressant effect of propofol. Pooling of blood in spleen as a result of reduced sympathetic tone caused by dexmedetomidine (Jadon *et al.* 1995) might have caused significant (P<0.05) decrease in values of haemoglobin, total leukocyte count and total erythrocyte count. The stress during analgesic period and involvement of subcortical pathways in ACTH regulation might have led to significant (P<0.01) increase in erythrocyte sedimentation rate at 1 h interval in both groups (Steyn 1969). Significant (P<0.01) increase in glucose and simultaneous (P<0.01) decrease in insulin levels from 30 min to 6 h interval in both groups might be 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). Bisht *et al.* (2016) reported similar finding in dogs administered dexmedetomidine-etomidate-sevoflurane. Increased level of stress during anaesthesia, increased depth of anaesthesia and non-significant effect of dexmedetomidine and propofol on cortisol levels during surgery and anaesthesia might have caused significant (P<0.01) increase in cortisol levels in both groups (Du *et al.* 2015). Significant (P<0.01) rise in serum urea nitrogen at 60 min (group I; P<0.05) and from 60 min to 6 h (group II; P<0.05), and significant (P<0.01) rise in creatinine at 60 min (group I) and from 30 min to 60 min (group II) might be due to the effect of drugs used during anaesthesia which produce temporary inhibition of renal blood flow resulting in decreased glomerular filtration rate (Umar and Adam 2013). Mazumdar *et al.* (2015) reported similar finding in dog administered dexmedetomidine. Values of serum alanine amino-transfersae and aspartate aminotransferase increased significantly (P<0.01) at 1 h interval and reached base values towards the end of study period in both groups. This was in accordance with findings of Sharma *et al.* (2014) and Bisht *et al.* (2016). The permeability of cell membrane to liver or muscle tissues changes due to hypoxia created as a result of alpha-2 agonist induced respiratory depression which in turn causes changes in level of liver enzymes (Kalim *et al.* 2011). The values of different haematobiochemical parameters returned to their base levels by 24 h in animals of both the groups. Non-significant differences in haematobiochemical parameters were observed between animals of two groups at respective time intervals.

From the results of the present study, it was concluded that both anaesthetic combinations produce satisfactory anaesthesia and muscle relaxation but their use was

associated with transient changes in physiological and haematobiochemical parameters. Dexmedetomidine @ 10 µg/kg and 15 µg/kg body weights can be suggested for clinical use in dogs undergoing propofol-sevoflurane anaesthesia.

ACKNOWLEDGEMENTS

The authors would like to thank the Dean, College of Veterinary and Animal Sciences; Dean, Post Graduate Studies, Director (Research) and Director (Experiment Station) for providing all necessary facilities for the conduct of research work.

REFERENCES

Ahmad R A, Amarpal, Kinjavdekar P, Aithal H P, Pawde A M and Kumar D. 2013. Potential use of dexmedetomidine for different levels of sedation, analgesia and anaesthesia in dogs. *Veterinarni Medicina* **58**: 87–95.

Bisht D S, Jadon N S, Kandpal M and Bodh D. 2016. Clinicophysiological and haematobiochemical effects of dexmedetomidine-etomidate-sevoflurane anaesthesia in dogs. *Indian Journal of Veterinary Surgery* **37**: 77–81.

Carroll C L, Krieger D, Campbell M, Fisher D G, Comeau L L and Zucker A R. 2008. Use of dexmedetomidine for sedation of children hospitalized in the intensive care unit. *Journal of Hospital Medicine* **3**: 142–47.

Dewangan R, Tiwari S K, Sharda R and Nath K. 2010. Clinicophysiological and cardiopulmonary response to xylazine-propofol anaesthesia in dogs. *Indian Journal of Veterinary Surgery* **31**: 127–29.

Du Y, Chen Y J, He B and Wang Y W. 2015. The effects of single-dose etomidate versus propofol on cortisol levels in pediatric patients undergoing urologic surgery: a randomized control trial. *Anesthesia and Analgesia* **121**: 1580–85.

Glowaski M M and Wetmore L A. 1999. Propofol: application in veterinary sedation and anesthesia. *Clinical Techniques in Small Animal Practice* **14**: 1–9.

Haitjema H and Cullen L. 2001. Clinical experience with sevoflurane in dogs. *Australian Veterinary Journal* **79**: 339–41.

Hoy S M and Keating G M. 2011. Dexmedetomidine. *Drugs* **71**: 1481–1501.

Itami T, Kawase K, Tamaru N, Ishizuka T, Tamura J, Miyoshi K, Umar M A, Inoue H and Yamashita K. 2013. Effects of a single bolus intravenous dose of tramadol on minimum alveolar concentration (MAC) of sevoflurane in dogs. *Journal of Veterinary Medical Science* **75**: 613–18.

Jadon N S, Kumar A, Singh H and Singh H. 1995. Clinical, haematological and biochemical effects of detomidine and ketamine anaesthesia in dogs. *Indian Journal of Animal Sciences* **65**: 967–69.

Jain N C. 1986. Hematologic techniques. *Schalm's Veterinary Hematology*. 4th ed. Lea and Febiger, Philadelphia, PA. pp 20–86.

Jena B, Das J, Nath I, Sardar K K, Sahoo A, Beura S S and Painuli A. 2014. Clinical evaluation of total intravenous anaesthesia using xylazine or dexmedetomidine with propofol in surgical management of canine patients. *Veterinary World* **7**: 671–80.

Kalim O, Tiwari S K, Sharda R and Vishwakarma P. 2011. Haematobiochemical response to lumbar epidural anaesthesia using bupivacaine alone and in combination with certain analgesics in buffalo calves. *Iranian Journal of Veterinary Science and Technology* **3**: 17–24.

Kuusela E, Raekallio M, Vaisanen M, Mykkanen K, Ropponen H and Vainio O. 2001. Comparison of medetomidine and dexmedetomidine as premedicants in dogs undergoing propofol-isoflurane anesthesia. *American Journal of Veterinary Research* **62**: 1073–80.

Lemke K A. 2007. Anticholinergics and sedatives. *Lumb and Jones' Veterinary Anaesthesia and Analgesia*. 4th ed. (Eds) Tranqilli W J, Thurmon J C and Grimm K A. Blackwell Publishing Ltd., Oxford.

Mazumdar H, Sarma B, Sarma K K and Mazumdar A. 2015. Haemato-biochemical effects of dexmedetomidine in dog. *International Journal of Recent Scientific Research* **6**: 5301–03.

Sharma R, Kumar A, Kumar A, Sharma S K, Sharma A and Tewari N. 2014. Comparison of xylazine and dexmedetomidine as a premedicant for general anaesthesia in dog. *Indian Journal of Animal Sciences* **84**: 8–12.

Steyn D G. 1969. Adrenal cortical response to halothane anaesthesia. *Journal of South African Veterinary Medical Association* **40**: 353–67.

Umar M A and Adam M K. 2013. Effects of combination of ketamine medetomidine anaesthesia on haematology and some serum chemistry parameters in dogs. *Nigerian Veterinary Journal* **34**: 808–13.