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Effects of Desflurane and Isoflurane on Postanaesthetic Recovery Characteristics with Hepatic and Renal Functions in Dogs

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Abstract: The aim of the study, was to investigate the effects of desflurane and isoflurane on postanaesthetic recovery characteristics with hepatic and renal functions in dogs. Sixteen adult mongrel dogs of both sexes weighing between 16-21 kg were equally divided into 2 groups. Anaesthesia was induced with 0.3 mg kg^{-1} midazolam and 10 mg kg^{-1} thiopental intravenously. Maintenance of anaesthesia was continued with 7.2-8% desflurane or 1.3-1.5% isoflurane. Heart and respiration rates, arterial haemoglobin oxygen saturation and rectal temperatures were monitored before, during, 1 h and 1 day after anaesthesia periodically and postanaesthetic recovery score times were also observed. Serum alanine aminotransferase, aspartate aminotransferase, γ-glutamyltransferase, blood urea nitrogen, total bilirubin and creatinine levels were measured in venous blood samples. Postanaesthetic recovery scores including time to standing, time to reaching sternal recumbency, eye opening and time to extubation were found shorter in desflurane group than isoflurane group ($p<0.05$). Alanine aminotransferase, aspartate aminotransferase and γ-glutamyltransferase activities were non-significantly increased in the isoflurane group and there were no change between groups. Total bilirubin and creatinine levels were non-significantly decreased, blood urea nitrogen levels were non-significantly increased within normal range during and after the anaesthesia in both groups. The present study reveals 2 important results. Firstly, desflurane provided faster anaesthetic induction, recovery with less excitation and rapid recovery times ($p<0.05$) and the dogs stood up in shorter time than those of isoflurane. Secondly, this study indicates that both volatile agents have no harmful side effects on renal and hepatic functions in dogs.

Key words: Desflurane, isoflurane, dog, postanaesthetic recovery scores, renal and hepatic functions

INTRODUCTION

Desflurane is a new inhalation anaesthetic agent with rapid induction and fast recovery due to its low blood/gas solubility ratio (0.42) (Clutton, 1998; O'Keeffe and Healy, 1999; Go´mez-Villamandos *et al*., 2000; Go´mez-Villamandos *et al*., 2006). Animal studies suggest that desflurane undergoes the least metabolism compared to all the volatile agents (Koblin *et al*., 1989). Isoflurane undergoes approximately 0.2% metabolism and desflurane metabolism is estimated to be 1-10th of that (Koblin *et al*., 1989; Schmidt *et al*., 1999). This indicates that tissue toxicity is unlikely (Sutton *et al*., 1991). Desflurane anaesthesia has generally not been associated with deterious effects on the liver and kidney (O'Keeffe and Healy, 1999). However, recent studies (Nishiyama *et al*., 1998; Tiainen *et al*., 1998; Schmidt *et al*., 1999) have reported that isoflurane and desflurane anaesthesia cause liver injury in man. There are several studies evaluating hepatic function during desflurane anaesthesia in humans (Sutton *et al*., 1991; Zaleski *et al*., 1993; Wissing and Kuhn, 2000) and isoflurane in dogs (Topal *et al*., 2003). Isoflurane is also used as a common inhalational anaesthetic agent, but its clinical application related to desflurane is limited in the veterinary practice. While there have been several studies (Tsai *et al*., 1992; Smith *et al*., 1994; Dexter and Tinker, 1995; Dupont *et al*., 1999; Hedenqvist *et al*., 2001; Ozturk and Altug, 2007) suggesting therapid emergence of desflurane compared with isoflurane, to date, no studies have compared isoflurane directly with desflurane on recovery characteristics with hepatic and renal effects in dogs. Thus, the present study was designed to compare the postanaesthetic recovery characteristics of desflurane and isoflurane inhalation anaesthetics and their hepatic and renal haemodynamic effects in dogs.

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MATERIALS AND METHODS

Animals and experimental procedures: The dogs were supplied from Antakya Municipality Dog Care House. The experiments were conducted in accordance with the Animal Research Ethics Committee of University of Mustafa Kemal, Faculty of Veterinary Medicine. The animals were vaccinated and observed for 30 days prior to the experimental procedure. Based on a clinical examination such as electrocardiographic and ultrasound evaluations of the heart, liver and kidneys and complete blood count, parasite profiles and serum biochemical analyses, all dogs were classed as ASA (American Society of Anaesthesiologists) class I. Sixteen healthy mongrel dogs, 18.2±5.4 years old and weighing (means±SD) 17.8±3.2 kg were used in this study and the dogs were randomly divided into 2 equal groups (for each groups; male: 4, female: 4). There were no significant differences between groups with respect to sex, age or body mass. Anaesthesia theatre temperature was kept at between 18-22°C and humidity, 60-65%. Food, but not water, was withheld for at least 12 h before the start of the anaesthesia protocols.

Induction and inhalation anaesthesia procedures: Anaesthetic protocols were designed as midazolam + thiopental + desflurane (DES group) and midazolam + thiopental + isoflurane (ISO group). Ten minutes prior to induction of anaesthesia, the dogs received atropine sulphate (Atropan® 2 mg mL^{-1}; Vetas, Istanbul, Turkey) at the dose rate of 0.04 mg kg^{-1} intramuscularly to control salivation and then an intravenous catheter was placed in the cephalic vein to infuse Lactated Ringer's solution (Eczacibasi-Baxter, Istanbul, Turkey) at the rate of 10 mL $kg^{-1}h^{-1}$. Anaesthesia was induced with 0.3 mg kg^{-1} midazolam (Dormicum® 5 mg mL^{-1}, Roche, Istanbul, Turkey) and 10 mg kg^{-1} thiopental (Pentotal Sodyum® 0, 5 g, Abbott, Istanbul, Turkey) intravenously (1/3; with rapid infusion, its remainder; slow infusion) in all animals. No supplement dosages were administered thereafter. The dogs were positioned in left lateral recumbency during anaesthesia. Following endotracheal intubation with a cuffed tube, a semi-closed circle rebreathing anaesthesia system was connected to the endotracheal tube and the animals were delivered 100% oxygen. The initial vaporizer settings were started with 1.5% isoflurane or 8.0% desflurane in oxygen (3 L min^{-1}) for induction of inhalant anaesthetics and anaesthesia maintenance was continued for 70 min with 7.2% desflurane (Suprane®, Baxter, Munich, Germany) or 1.3% isoflurane (Aerrane®, Baxter, Illinois, USA). Carbon dioxide was removed from the circuit using soda lime. At the end of anaesthesia procedure, the inhaled anaesthetics were discontinued and the lungs were ventilated with 100% oxygen at a fresh gas flow rate of 3 L min^{-1}. The concentration of desflurane and isoflurane were also chosen according to previous reports in dogs. The vaporizer and the monitoring devices were calibrated before the study according to manufacturer's instructions. Anaesthesia maintenance was performed in the 2 groups with an anaesthesia machine (AMS 200, Ankara, Turkey) and vaporizers (Desflurane-Datex Ohmeda Tec 6 Plus vaporizer, USA; Isoflurane-Blease Datum® vaporizer, England). The induction times of inhalant agents were also calculated from time to starting of the volatile gas infusion and sings of associated with the 3rd stage (plane 2) of general anaesthesia (Thurmon *et al*., 1999).

Postanaesthetic recovery characteristics: The trachea was extubated when a regular spontaneous breathing pattern had been reestablished and then all dogs observed in the Post-Anaesthesia Care Unit (PACU) for the next 3 h and the animal care unit for 1 day. Time to reaching a Postanaesthetic Recovery Scores (PARS) including time to extubation, time to standing, time to reaching sternal recumbency, time to return of head control, tongue control, eye opening and absence of anal reflex and adverse effects during the recovery period such as presence or absence of vomiting, coughing and excitation were recorded. Time to extubation was measured from time interval between the disconnection of the volatile anaesthetic maintenance and the removal of the endotracheal tube. Time to sternal recumbency was recorded from time in minutes from time to extubation until voluntary return to sternal recumbency. Time to standing were also recorded from time in minutes from time to extubation to stand up.

Cardiopulmonary responses: Heart Rate (HR, beats min^{-1}), Respiration Rate (RR, beats min^{-}),[1] arterial oxyhaemoglobin saturation (SpO_2, %) and rectal temperature (°C) of all animals in this experiment were monitored at baseline, 20 (induction), 50, 70 and 90 min (anaesthesia maintenance) and then 1 h after and one day after, respectively. Respiration rates (RR, beats min^{-1}) were evaluated with auscultation of lungs and rectal temperatures were measured using a digital thermometer. Heat lamps were used to maintain rectal temperature at approximately 38°C during anaesthesia. Heart Rate (HR, beats min^{-1}) and SpO_2 (%) were monitorised using a pulse oximeter (Criticare 504DX Pulse Oximeter, Waukesha, USA), which was noninvasively placed on ears.

Evaluating hepatic and renal functions: Venous blood samples were taken at baseline, 20 (induction), 50 and 90 min (anaesthesia maintenance) and then 1 h after and 1 day after, respectively. Serum was separated by centrifugation at 3000 rpm for 5 min, collected into a microfuge tube. The obtained serum samples were evaluated on the same day. Serum Alanine Aminotransferase (ALT), aspartate Aminotransferase (AST), γ-Glutamyl-Transferase (GGT), Blood Urea Nitrogen (BUN), Total bilirubin (Tbil) and Creatinine (Cr) levels were measured using Teco diagnostic kits in an autoanalyser (Autolab, Analyzer Medical System, Roma, Italia) for 2 groups.

Statistical analysis: Data are reported as means±standard deviation. Statistical evaluations were accomplished with the standard statistical software (SPSS version 13, Chicago, IL, USA). Continuous variables were tested for normality with the Kolmogorov-Smirnov test. Data were analysed with repeated measures ANOVA for the time effect in each group; when indicated, a Bonferroni's correction was performed for post hoc comparisons. The differences between groups at each time point were evaluated with use independent t-test. Differences were considered significant when $p<0.05$.

RESULTS

Induction and inhalation anaesthesia findings: The induction of anaesthesia with midazolam and thiopental was rapid and smooth and endotracheal tubes were inserted without any trouble in all dogs. Induction of the inhalation anaesthesia was generally smooth, apart from airway irritation was observed in 2 animals in the desflurane group. The times of the inhalation induction were found shorter in desflurane group than isoflurane group (Fig. 1, $p<0.01$). In desflurane group, more coughing (in 4 of 8 cases) was experienced immediately after extubation when compared to isoflurane (in 1 of 8 cases).

Postanaesthetic recovery findings: Emergence from anaesthesia was generally smooth and also vomiting was not seen in any cases. Time to reaching a PARS such as time to extubation, time to standing, time to reaching sternal recumbency, time to return of head control, tongue control, eye opening and absence of anal reflex were presented in Fig. 1. PARS times were found shorter in desflurane group than isoflurane group. The differences of time to standing, time to reaching sternal recumbency, eye opening and time to extubation between desflurane and isoflurane group after anaesthesia were statistically significant (Fig. 1, $p<0.05$). The dogs in desflurane group stood up in shorter time with less excitation than those of isoflurane.

Cardiopulmonary responses: The changes of heart rate, respiration rate and arterial oxyhaemoglobin saturation (SpO_2) measurements in the groups and the statistical differences between the groups were presented in Table 1. The heart rate of animals in both groups increased at induction and maintenance of anaesthesia ($p<0.005$). The respiration rate significantly decreased at induction and maintenance of anaesthesia in both groups ($p<0.05$). The level of SpO_2 significantly decreased at induction of anaesthesia with thiopental ($p<0.05$) and slightly non-significantly increased during anaesthesia in each group. During and after anaesthesia, the differences of respiration rate and SpO_2 between desflurane and isoflurane groups were not found significant, but the increases of the heart rate in desflurane group were found significantly higher than isoflurane group ($p<0.05$).

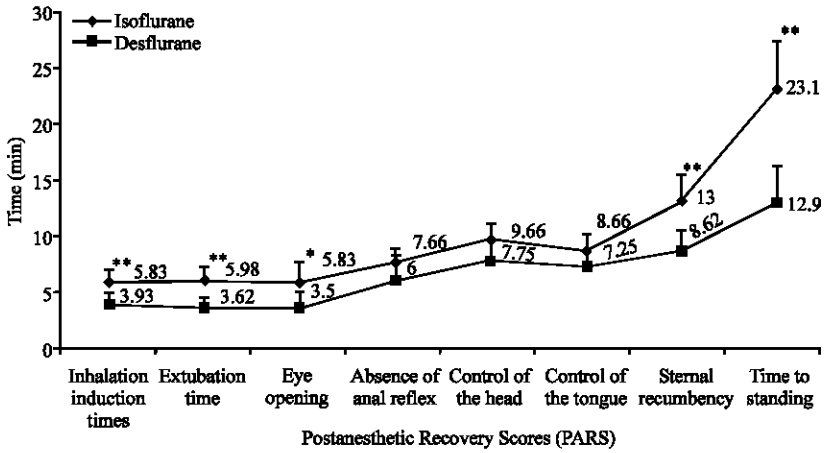

Fig. 1: Mean times to reaching Postanaesthetic Recovery Score (PARS) and inhalation induction times in dogs. Data are expressed as mean±SD; *$p<0.05$ versus the desflurane group, **$p<0.01$ versus the desflurane group

Table 1: Cardiopulmonary parameters before, during and after Desflurane (DES) and Isoflurane (ISO) anaesthesia in dogs

	Before anaesthesia	Induction and inhalation anaesthesia maintenance (min)					After anaesthesia	
Parameters	baseline	20th	30th	50th	70th	90th	1 h after	24 h
HR (Beats min^{-1})								
DES	101±15	190±35§	168±26§	146±29*†	148±30†*	153±33†*	127±23**	102±18
ISO	88±15	160±27§	147±32§	122±19	116±15	112±14	94±15	92±13
RR (Beats min^{-1})								
DES	30.8±9.8	30.1±21	19.5±7.8	14.2±7.8†	15.9±5.5	18.0±4.9	28.6±12	29.7±9.7
ISO	31.6±9.6	28.0±11	20.8±8.5	15.6±6.3†	15.8±4.7	17.6±4.7	33.8±8.1	21.4±3.1
SpO_2 (%)								
DES	96.8±1.6	92.1±2.1†	96.1±3.3	97.2±1.7	97.8±1.8	97.9±1.3	95.7±2.5	96.5±1.7
ISO	95.9±2.1	90.3±1.6†	97.8±3.1	97.4±1.5	98.2±1.5	97.8±1.3	95.3±2.3	95.8±2.7

HR: Heart Rate, RR: Respiration Rate; SpO_2: arterial oxyhaemoglobin saturation; Data are expressed as mean±SD; †: $p<0.05$ compared with before anaesthesia the measurements in the group; §: $p<0.005$ compared with before anaesthesia the measurements in the group; *$p<0.05$ versus the ISO group; **$p<0.01$ versus the ISO group

Table 2: The markers of hepatic and renal functions before, during and after Desflurane (DES) and Isoflurane (ISO) anaesthesia in dogs

	Before anaesthesia	Induction and inhalation anaesthesia maintenance			After anaesthesia	
	baseline	20th min	50th min	90th min	1 h after	24 h
ALT U L^{-1}						
DES	41.4±23.2	35.6±22.3	32.1±29.2	24.8±12.8	41.3±23.0	34.2±25.9
ISO	37.4±12.4	34.8±12.9	35.6±10.5	32.3±12.1	46.5±22.9	46.2±18.4
AST U L^{-1}						
DES	37.7±31.1	34.0±25.6	31.5±24.5	31.2±7.2	29.5±29.8	33.7±37.2
ISO	36.5±19.9	35.4±13.7	35.8±14.6	36.1±10.7	43.4±16.6	45.8±27.4
GGT U L^{-1}						
DES	4.1±3.5	4.2±4.2	4.1±1.2	4.0±2.0	4.3±3.1	4.3±3.7
ISO	4.2±3.0	4.3±2.4	4.4±2.8	4.3±1.5	5.1±1.1	5.5±1.4
Cr mg dL^{-1}						
DES	0.89±0.3	0.83±0.3	0.77±0.1	0.77±0.1	0.78±0.2	0.81±0.1
ISO	0.95±0.3	0.78±0.1	0.76±0.1	0.71±0.1	0.74±0.1	0.77±0.06
Tbil mg dL^{-1}						
DES	0.07±0.04	0.04±0.02	0.03±0.02	0.04±0.02	0.03±0.02	0.05±0.04
ISO	0.06±0.02	0.05±0.02	0.04±0.02	0.05±0.03	0.03±0.02	0.05±0.02
BUN mg dL^{-1}						
DES	22.2±3.7	23.4±4.0	25.7±1.9	24.6±1.3	24.9±2.0	24.3±2.2
ISO	21.2±7.7	21.1±8.0	21.3±7.7	23.0±8.0	22.8±3.8	27.1±3.8

Data are expressed as mean±SD; There were no statistically significant differences between the study groups ($p<0.05$)

Hepatic and renal function markers: The values of ALT, AST, Cr, Tbil, BUN and GGT in groups and between groups were presented in Table 2. In the isoflurane group, ALT, AST and GGT activities non-significantly increased 1 h and one day after anaesthesia. In the desflurane group, these enzymes retained within normal ranges. Tbil and Cr levels were non-significantly decreased within normal range during and after anaesthesia in both groups. BUN levels in desflurane and isoflurane groups were non-significantly increased during and after anaesthesia.

DISCUSSION

The Minimum Alveolar Concentration (MAC) of desflurane and isoflurane in dogs has been reported between 7.2 and 10.3% (Doorley *et al*., 1988; Hammond *et al*., 1994; Go´mez-Villamandos *et al*., 2006) and 1.28 and 1.30% (Steffey and Howland, 1977; Steffey *et al*., 1994), respectively. In this study, the MAC of both volatile anaesthetics were selected on the basis of earlier reports corresponding with desflurane (Doorley *et al*., 1988; Hammond *et al*., 1994; Go´mez-Villamandos *et al*., 2006) and isoflurane (Steffey and Howland, 1977; Steffey *et al*., 1994). We initially used a vaporizer anaesthetic concentration goal of 1.5% isoflurane and 8.0% desflurane for induction of anaesthesia. After reaching time to surgical anaesthesia the concentrations (%) of volatile gases were reduced to 1.3% for isoflurane and 7.2% (1 MAC) for desflurane in maintenance of anaesthesia. These rates were seen adequate to maintain anaesthesia in the dogs.

Desflurane has been shown to result in a more rapid emergence and faster recovery than isoflurane in human (Tsai *et al*., 1992; Smith *et al*., 1994; Dexter and Tinker, 1995; Dupont *et al*., 1999), rabbit (Hedenqvist *et al*., 2001) and rats (Öztürk and Altug, 2007). Tsai *et al*. (1992) stated that the quality of recovery was also felt to be better, with less shivering and less delirium in the desflurane patients. Martin *et al*. (2001) also reported that dogs anaesthesized with desflurane stood up between 12.5-13.5 min, in addition, Dexter and Tinker (1995) found that human patients given desflurane were discharged a mean of

4.4 min earlier than those given isoflurane. In our study, times of the inhalation induction and PARS times including time to standing, time to reaching sternal recumbency, eye opening and time to extubation were shorter in desflurane group than isoflurane group, respectively as presented Fig. 1 ($p<0.05$). Recovery scores were also superior in the desflurane group as reported by Martin *et al.* (2001). In addition, Smith *et al.* (1994) noticed that eye opening and time to tracheal extubation were shorter as found in our study. The data obtained in this study were consistent with the studies mentioned above. The results may be explained by the fact that desflurane has a lower blood-gas solubility coefficient than that of isoflurane (0.42 versus 1.4), indicating that induction and recovery from isoflurane anaesthesia is more rapid (Sutton *et al.*, 1991; Clutton, 1998; Öztürk and Altug, 2007). To our knowledge, the present study is the first report, which compared in detail the influence of desflurane and isoflurane administration on recovery characteristics in dogs.

The circulatory effects of desflurane are parallel to those of isoflurane (Weiskopf *et al.*, 1995; Clarke *et al.*, 1996). As with isoflurane, if the concentration of desflurane is increased rapidly to concentrations exceeding 1 MAC, sympathetic activity and heart rate then increase (Clarke *et al.*, 1996). In the study, although both anaesthetic agents increase heart rate, the increases of the heart rate during desflurane were significantly higher than that of isoflurane. These findings might be an indication for a more depressant effect on the cardiac vagal activity of desflurane in comparison with isoflurane as reported (Picker *et al.*, 2001).

As the other inhaled anaesthetics, desflurane causes dose-dependent respiratory depression. The magnitude of respiratory depression seems to be similar that of isoflurane and it is expressed in drastic decreases of respiration rate (Clarke *et al.*, 1996). Desflurane extremely irritates to the airways with its sympathetic stimulation effects (Clarke *et al.*, 1996), with concentrations of 6% or more causing coughing, breath holding and laryngospasm, making it unsuitable to use for inhalation induction in both children and adults (Zwass *et al.*, 1992). In this study, the levels of respiration rate significantly decreased during anaesthesia in both groups ($p<0.05$). In the desflurane group was experienced more coughing immediately, after extubation than isoflurane. It shows that isoflurane is less irritating to the airways compared to desflurane in dogs. These findings are consistent with other reports noting that desflurane is an airway irritant (Zwass *et al.*, 1992; Clarke *et al.*, 1996). When spontaneous respiratory inspiration and expiration were allowed in slight anaesthesia, frequent respiration was seen during the desflurane induction. This finding is similar to that reported by Clarke *et al.* (1996).

Karzai *et al.* (1998) showed that desflurane and isoflurane decrease arterial oxyhaemoglobin saturation (SpO_2) compared to propofol anaesthesia. Dupont *et al.* (1999) found that isoflurane and desflurane provided similar haemodynamic effects and arterial oxygenation. Weiskopf *et al.* (1995) reported that desflurane maintains myocardial, hepatic, intestinal and sceletal muscle blood flow, although, isoflurane decreases intestinal and skeletal muscle blood flow in a dose dependent manner and decreases tissue perfusion at various degrees, depending on systemic hypotension in vasculary beds in dogs. In the present study, SpO_2 levels significantly ($p<0.005$) decreased immediately after receiving midozolam and thiopental, but during desflurane and isoflurane maintenance non-significantly increased SpO_2 without significant difference between 2 volatile anaesthetics in regard to arterial oxygenation. This finding was contrary to the report by Weiskopf *et al.* (1995) and Karzai *et al.* (1998), whereas it was consistent with research by Dupont *et al.* (1999). In this study, SpO_2 was >95% during desflurane and isoflurane administration in all dogs. The occurrence that isoflurane and desflurane increased arterial oxygenation was probably as a result of the fact that they were delivered in oxygen.

Elevated serum levels of aminotransferase and bilirubin activity have been regarded as the gold standard for anaesthetic-related hepatic toxicity in humans (Zaleski *et al.*, 1993; Nishiyama *et al.*, 1998; Wissing and Kuhn, 2000) and animals (Bernard *et al.*, 1990; Topal *et al.*, 2003). Sutton *et al.* (1991) and Zaleski *et al.* (1993) reported that desflurane affected liver function minimally or not at all in adults. In addition, Wissing and Kuhn (2000) expressed that there was no any observed toxic effect on liver of desflurane exposure. This may be related to its minimal biodegradation and the rapid elimination after anaesthesia. Nishiyama *et al.* (1998) states that isoflurane increases the levels of AST, ALT and Tbil and leads to hepatic damage. Topal *et al.* (2003) reported that isoflurane significantly increased ALT and AST activities 2 days after anaesthesia and GGT activities significantly increased seven days after anaesthesia. In the present study, ALT, AST and GGT levels non-significantly increased without resulting in hepatic damage in isoflurane group 1 h and one day after anaesthesia. These findings were inconsistent with those of Nishiyama *et al.* (1998) and Topal *et al.* (2003) results. On the other hand, these enzymes retained in the normal reference limits in desflurane group. These findings confirm that isoflurane and desflurane affect liver functions minimally and not cause hepatotoxicity in dogs. The levels of ALT and AST in desflurane group were consistent with previous human reports (Sutton *et al.*, 1991; Zaleski *et al.*, 1993; Wissing and Kuhn, 2000). Conversely, the findings of the present study were

inconsistent with those of Steffey *et al.* (2000) results, indicating AST activity increased by desflurane in horses.

Nephrotoxicity, directly attributable to halothane and isoflurane, or their metabolites has not been reported in dogs (Clutton, 1998). In addition, there is no evidence of about renal toxicity of desflurane, even after prolonged exposure. Eger *et al.* (1997) examined markers of renal function (urinary albumin, glucose, α-glutathione-S-transferase and serum creatinine and blood urea nitrogen) before and for up to 7 days after exposure to 8 h of 1.25 times MAC desflurane and they found no evidence of renal injury. As for hepatic function, desflurane only minimally affects renal function in human patients (Sutton *et al.*, 1991; Weiskopf *et al.*, 1995), dogs (Merin *et al.*, 1991) and horses (Steffey *et al.*, 2000). Zaleski *et al.* (1993) noticed that desflurane and isoflurane seemed to be unaffected both renal function and blood flow. In addition, Wissing and Kuhn (2000) reported that Tbil and Cr remained unchanced or decreased. In our study, it was seen that Tbil and Cr levels non-significantly decreased and also BUN levels were non-significantly increased during and after anaesthesia in both groups ($p>0.05$, Table 2). The levels of Cr and Tbil obtained are consistent with Wissing and Kuhn's results (2000). Therefore, it may be suggested that these agents not attributed nephrotoxicity and occured similar renal effects.

CONCLUSION

The present study reveals 2 important results. Firstly, desflurane provided faster anaesthetic induction, time to standing, time to reaching sternal recumbency, eye opening and tracheal extubation in a shorter time and more rapid recovery than isoflurane. Secondly, this study suggests that both volatile agents have similar effects on kidney and liver functions without lead to renal and hepatic damage in dogs.

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