# Anesthetic Induction with Propofol versus Ketamine Pre and Post Lower Pole Nephrectomy in Dogs

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**Abstract:** This study was performed on fourteen Mongrel dogs to compare anesthetic induction in healthy, as well as, partially nephrectomized dogs using bolus intravenous administration of either propofol 4 mg/kg b.wt. in group I (before nephrectomy) and group II (one month after lower pole nephrectomy) or ketamine 10 mg/kg b.wt. in group III (before nephrectomy) and group IV (one month after lower pole nephrectomy). The quality of induction and recovery, the occurrence of cardiovascular and respiratory side effects and serum biochemical parameters were investigated. The results revealed that, anesthetic induction time did not change significantly in dogs before and after nephrectomy under the effect of either propofol or ketamine. Meanwhile, ketamine induced significantly longer weak time and down time than did propofol in corresponding groups. RRF was significantly longer in nephrectomized than non nephrectomized dogs under the effect of both agents while, recovery time was significantly longer in nephrectomized than non nephrectomized dogs under the effect of propofol. Ketamine caused significantly longer recovery time than did propofol in corresponding groups. There were no significant differences in induction and recovery scores before and after nephrectomy in dogs anesthetized with either propofol or ketamine. However, propofol caused significantly better induction and recovery than did ketamine in corresponding groups. Propofol caused significant decrease but ketamine caused significant increase in heart rate and respiratory rate in both nephrectomized and non nephrectomized dogs. Meanwhile, they did not significantly alter rectal temperature. ECG tracings showed only change in heart rate without arrhythmias. Significant increases in AST, LDH, CPK, urea and creatinine were observed in all groups with minor disparity from one to another group. It could be concluded that ketamine had better cardiopulmonary effect than propofol but the later was superior in the quality of induction and recovery. Lower pole nephrectomy in dogs had minimal impact on the modality of the effect of either agent.

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**Key words:** Dog – ketamine – nephrectomy - propofol

## 1. Introduction

Induction of short-term anesthesia can be accomplished by use of intravenous (IV) anesthetic drugs such as propofol and dissociative agents. Propofol (2,6-di-isopropylphenol) is a nonbarbiturate sedative/hypnotic drug that is rapidly metabolized in the animal. The best features of this drug include the rapid induction of anesthesia, short duration of action, lack of excitatory side effects on induction and recovery, and no significant cumulative effects on repeated administration ***(Muir and Gadawski, 1998).*** However, the induction of anesthesia with propofol is often associated with a marked decrease in systemic arterial blood pressure in dogs ***(Brüssel et al., 1989)***. Respiratory depression and apnea are reported to be the most consistent and important side effects in animals receiving IV propofol ***(Muir and Gadawski, 1998)***. However, the drug is a suitable choice in patients with preexisting liver or kidney disease ***(Glowaski and Wetmore, 1999)***. Propofol is highly lipophilic and rapidly metabolized primarily to inactive glucuronide conjugates, the metabolites being excreted in the urine. In man, liver disease and renal failure have little effect on pharmacokinetic parameters and it seems likely that extrahepatic mechanisms contribute to the metabolism of propofol, but this has not been investigated in any detail in other animals ***(Hall et al., 2001).***

Ketamine is a phencyclidine derivative. It is an N-methyl-D-aspartate receptor antagonist with analgesic and anesthetic properties. Unlike many anesthetics, ketamine usually causes an increase in heart rate and arterial blood pressure as a result of increased sympathetic efferent activity ***(Wong and Jenkins, 1974)***. However, these cardiovascular effects may be unacceptable in some circumstances leading to the development of hypertension and tachycardia ***(Karapinar et al., 2006)***. Ketamine also has been associated with violent recoveries, muscle hypertonicity and convulsions in dogs ***(Haskins et al., 1985).*** Rapid recovery following intravenous bolus ketamine administration is by rapid

redistribution of ketamine from the CNS to other tissues, primarily fat, lung, liver, and kidney ***(Lanning and Harmel, 1975).*** Clinically, animals with renal dysfunction or obstruction to urine flow also have prolonged sleep times when larger doses of ketamine are given ***(Short, 1987)***. Generally speaking, dissociative anesthetics should be given cautiously to animals that have significant hepatic or renal dysfunction ***(Lin, 2007).*** Few, if any, studies were performed to assess intravenous anesthetic induction in nephrectomized or partially nephrectomized dogs. However, no data were available about the effect of propofol or ketamine in nephrectomized dogs.

The aim of this study was to compare anesthetic induction in healthy, as well as, partially nephrectomized dogs using bolus intravenous administration of either propofol or ketamine. The quality of induction and recovery, the occurrence of cardiovascular and respiratory side effects and serum biochemical parameters were investigated to determine whether either agent might be suitable for use as part of an induction technique in healthy or partially nephrectomized dogs. In addition, this study also aimed at evaluating the influence of partial (lower pole) nephrectomy on the effect of both agents in dogs.

## 2. Material and Methods

This study was performed on 14 Mongrel dogs before and one month after being used for an advanced experimental surgical technique (Permanent renal tourniquet for lower pole nephrectomy). Dogs were deemed to be healthy on the basis of physical examination and serum biochemical analysis.

Dogs were randomly assigned into two sets each of seven dogs. Each set received one treatment in two occasions. The first animal set received propofol (Diprivan®, AstraZeneca UK Limited, United Kingdom, 1% emulsion) as bolus intravenous induction agent before (Group I), as well as, one month after partial nephrectomy (Group II), while the second animal set received ketamine (Ketamine® 100, Pantex, Holland, 10% solution) as bolus intravenous induction agent before (Group III), as well as, one month after partial nephrectomy (Group IV). Dogs in the propofol set weighed 14.2 ± 3.7 kg (mean ± SD) and dogs in the ketamine set weighed 13.3 ± 3.1 kg.

This protocol was approved by local research ethics committee of Faculty of Veterinary Medicine, Suez Canal University. Food was withheld at least 8 hours prior to each animal being anesthetized. The dogs were weighed individually on the same scale. Thereafter, they were assigned for induction of anesthesia with propofol 4 mg/kg IV or ketamine 10 mg/kg IV.

An IV catheter was placed in the cephalic vein. All the dogs were anesthetized between 9:00 and 14:00 o'clock. The assigned induction drug was injected intravenously by hand at a rate of 10% of the total volume given as a bolus every 6 seconds until the total volume was injected ***(Sams et al., 2008)***. Loss of jaw tone and ability to intubate the trachea without resistance was assessed and marked to estimate induction score by a single, blinded observer. The dog's tracheas were intubated and they were allowed to breathe spontaneously on room air.

Pulse rate, respiratory rate, and rectal temperature were recorded before injection (baseline), and then every 5 minutes after injection for an hour even after the dogs began to swallow and the trachea was extubated. ECG tracings, lead II with paper speed 25 mm/seconds, were recorded at 5 and 10 minutes after injection of both induction agents to evaluate the electrical function of the heart.

Blood samples were taken from cephalic vein through the canula immediately before induction, and at 30, and 120 minutes as well as 24 and 48 hours after injection in clean dry tubes for serum biochemical analysis. Serum was collected and marked for later biochemical estimation. Serum biochemical analyses that included serum aspartate transferrase (AST) serum lactate dehydrogenase (LDH), creatine phosphokinase (CPK), serum urea and creatinine were estimated using a spectrophotometric method.

Weak time (the time in seconds elapsed from the end of injection of the induction agent to the time when the animal showed ptosis of the head) and induction time or down time (the time, in seconds, from the end of injection to sternal/lateral recumbency) were exactly recorded. In addition, return of righting reflex, RRF time, (the time, in minutes, elapsed from the end of injection to the ability of the animal to raise the head) and recovery time (the time, in minutes, from the end of injection to the time when the dog was able to walk unassisted) were also recorded. Quality of induction and recovery scoring *(Table, 1)*, and the incidence of side effects, e.g. muscle twitching, apnea (no spontaneous breathing for more than 20 seconds) and salivation, were also recorded.

**Statistical analysis:**

Values of HR, RR and RT, as well as, serum biochemical values were analyzed using one-way analysis of variance (ANOVA) for comparison of means between the groups and Dunnet multiple range test was used to compare the means at different intervals, within the group, to the baseline values. The data were presented as the mean ± SD. Induction

and recovery scores were analyzed primarily using Kruskal-Wallis test to estimate the mean ranks and to test the significance between groups; thereafter, Mann-Whitney U test was used to calculate the P value and test the differences between each two groups. The data were presented as the mean ranks. Significance was accepted at $p < 0.05$ ***(Snedecor and Cochran, 1967)***. The statistical analysis was performed using IBM SPSS 19.0.0 for windows.

**Table (1): Criteria used to evaluate the quality of induction and recovery in non nephrectomized as well as, nephrectomized dogs induced with propofol and ketamine.**

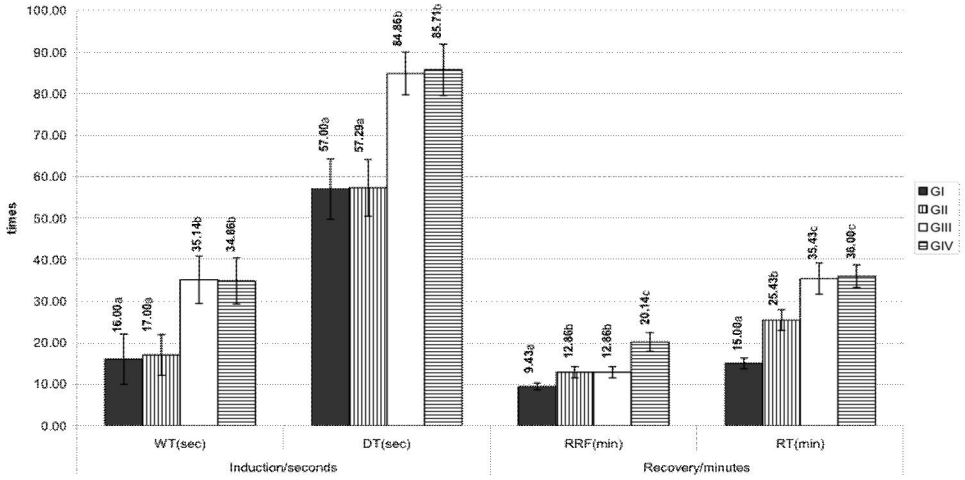
| |
|---|
| Induction quality scoring* |
| (1) Good = smooth induction, rapidly assumes recumbency, no signs of excitement, easy tracheal intubation |
| (2) Fair = slightly prolonged, mild excitement, reflex response to tracheal intubation |
| (3) Poor = obvious excitement, jumps or attempts to stand after recumbency, inability to intubate trachea |
| Recovery quality scoring* |
| (1) Good = smooth, easy transition to alertness, resumes sternal position, stands in a reasonable amount of time and is able to walk with minimal ataxia |
| (2) Fair = transient excitement or whole body movements, some struggling, hyper-responsiveness that disappears once dog stands unassisted but with moderate ataxia |
| (3) Poor = stereotypical behavior, e.g. circling, premature attempts to stand, prolonged struggling |

*Modified by ***Prasinos et al. (2005)*** after ***Lin et al. (1997)*** and ***Carroll et al. (1998).***

## 3. Results

Weak time and down time showed non significant difference between group I and group II. There was also non significant difference between group III and group IV. On the other hands, ketamine in group III and group IV induced significantly longer weak time and down time than did propofol in groups I and group II respectively. RRF times were significantly longer in group II and group IV than those in group I and group III respectively. Recovery time in group I was significantly shorter than that of group II, while, there was non significant difference between groups III and group IV. Ketamine in group III and group IV induced significantly longer recovery time than did propofol in group I and Group II respectively *(Fig. 1)*.

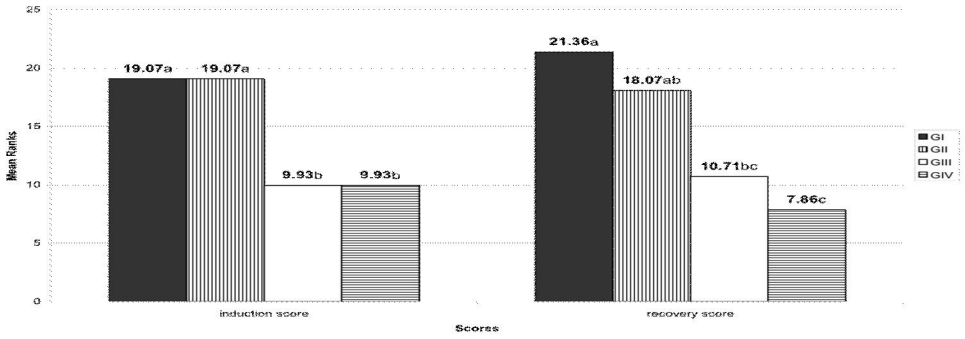
Fig. (1): Induction and recovery times in all groups




Induction score, mean ranks, showed non significant differences between group I and group II, as well as, between group III and group IV. On the other hands, propofol in non nephrectomized dogs (group I) and nephrectomized ones (group II) caused significantly better induction than did ketamine in non nephrectomized dogs (group III) and nephrectomized ones (group IV). Recovery score showed non significant difference between group I and group II, between group II and group III and also between group III and group IV. Propofol in non-nephrectomized (group I) and nephrectomized (group II) dogs caused significantly higher recovery score than did ketamine in non- nephrectomized (group III) and nephrectomized (group IV) dogs respectively *(Fig. 2)*. Three dogs, after ketamine injection (2 in

group III and 1 in group IV), showed abnormal behavior during recovery. They showed a state of delirium and abnormal voice. After recovery, the former two dogs in group III had moderate ataxia for 15 minutes.

Fig. (2): Induction and recovery scores (Mean Ranks) in all groups




Group I and group II showed significant decrease in heart rate *(Fig. 3)* and respiratory rate *(Fig. 4)*, compared to their baseline values, allover the follow-up period (60 min.). Group III showed significant increase in HR from 5 min. to 40 min. post injection compared to the baseline values. RR showed also significant increase from 5 min. to 35 min. post injection. Group IV showed significant increase in HR and RR from 5 min. to 40 min. post injection compared to the baseline values.

ECG tracings *(Fig. 5)* showed no arrhythmias. Only, there were changes in heart rate which was higher after ketamine injection than that after propofol in both nephrectomized and non nephrectomized dogs. Rectal temperature *(Fig. 6)* showed non significant difference in all groups allover the study period compared to the baseline values.

Fig. (3): Heart rate (Mean±SD) before and after injection of the agents in all groups

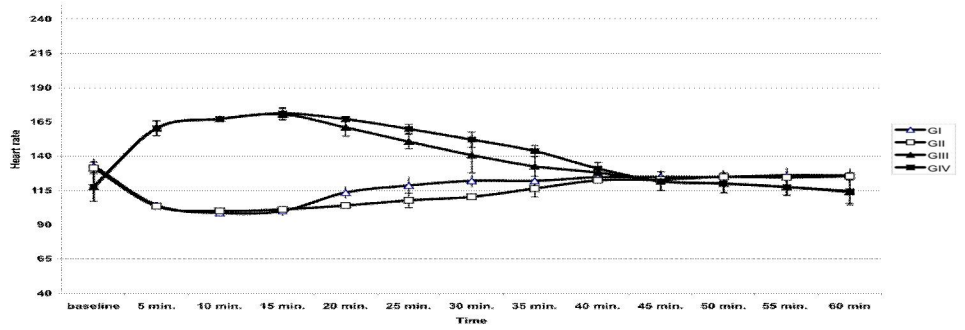
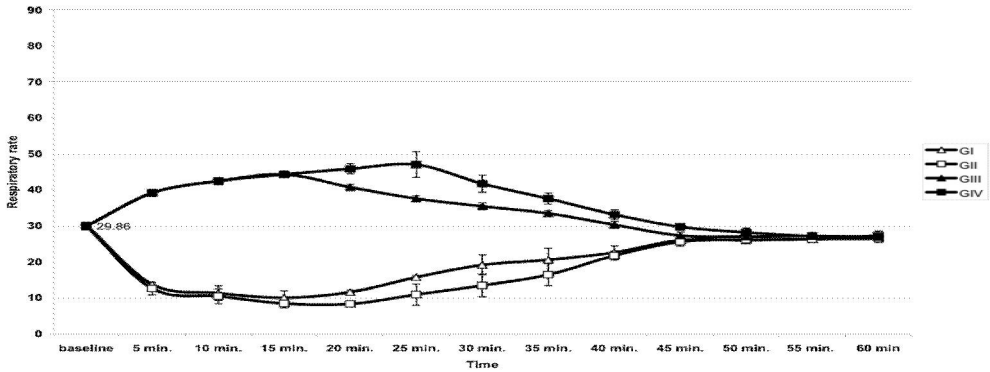



Fig. (4): Respiratory rate (Mean±SD) before and after injection of the agents in all groups



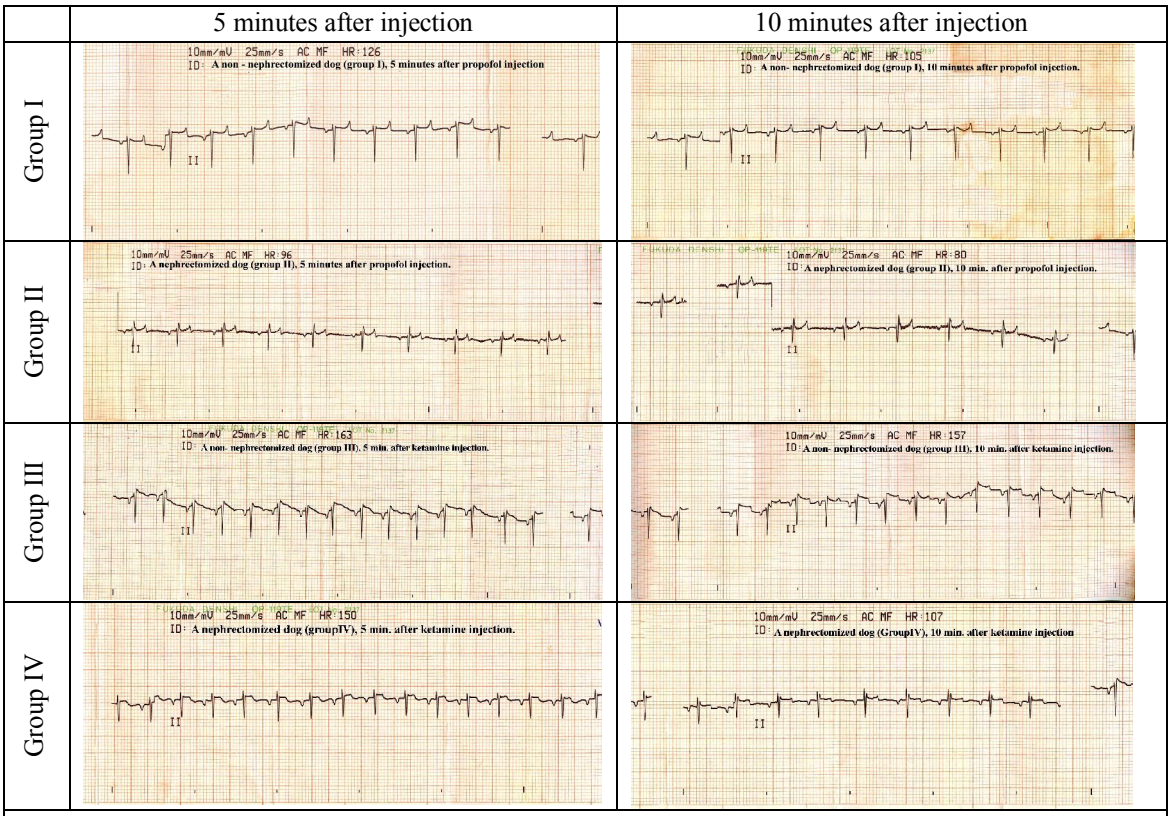
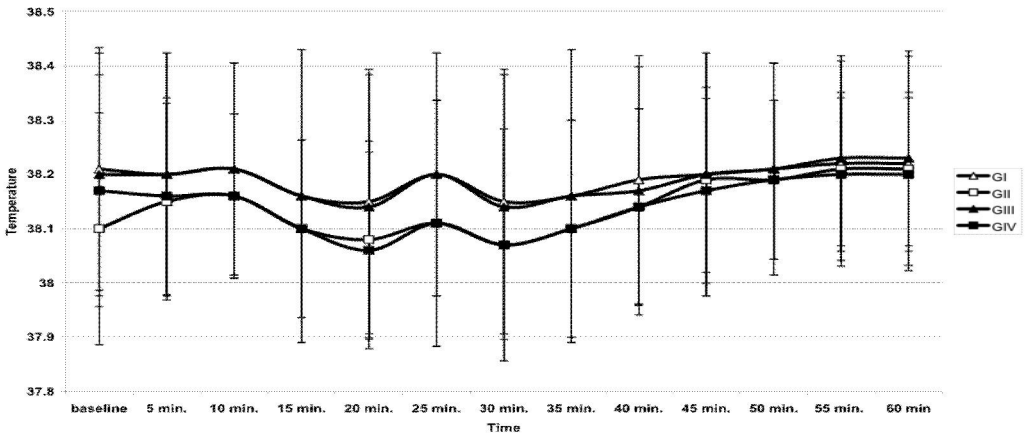



**Fig. (5): ECG tracings, lead II with paper speed 25 mm/seconds in dogs at 5 and 10 minutes after propofol in group1 (non nephrectomized) and group II (nephrectomized) as well as ketamine in group III (non nephrectomized) and group IV (nephrectomized) showing only differences in heart rates without arrhythmias.**

Muscle twitching during anesthetic duration was observed in 1 out of 7 dogs in group III and 1 out of 7 dogs in group IV, while propofol did not induce muscle twitching in group I or group II. Apnea was recorded in 3 out of 7 dogs in group I at the period between 7 and 11 minutes after propofol injection and 4 out of 7 dogs in group II at the period between 10 and 16 minutes after propofol injection, while ketamine did not induce apnea in group III and group IV. Salivation was observed in 3 dogs in group III and 2 dogs in group IV.

Fig. (6): Rectal temperature (Mean±SD) before and after injection of the agents in all groups



There were significant increases in AST values *(Fig. 7)* in all groups at 30, 120 min. and 24 hrs after injection, while non significant difference was observed at 48 hrs after injection compared to baseline values. LDH *(Fig. 8)* increased significantly at 30, 120 min and 24 hrs after injection in group I and group III, while group II and group IV showed only significant increase at 30 and 120 min after injection compared to the baseline values. Serum CPK *(Fig. 9)* increased significantly at 30, 120 min and 24 hrs after injection in group I, III and group IV, while group II showed only significant increase at 30 min after injection compared to the baseline values. Serum urea *(Fig. 10)* increased significantly at 30, 120 min and 24 hrs after injection in group I, II and group III, while group IV showed significant increase at 30, 120 min and 24, 48 hrs after injection compared to the baseline values. Serum creatinine *(Fig. 11)* increased significantly at 30, 120 min and 24 hrs after injection in group I and group III, while group II and group IV showed significant increase only at 30 min after injection compared to the baseline values. In all serum biochemical parameters, baseline values were significantly higher in group II and group IV (nephrectomized dogs) than those in group I and group III (non nephrectomized dogs).

Fig. (7): Serum AST (Mean±SD) before and after injection in all groups

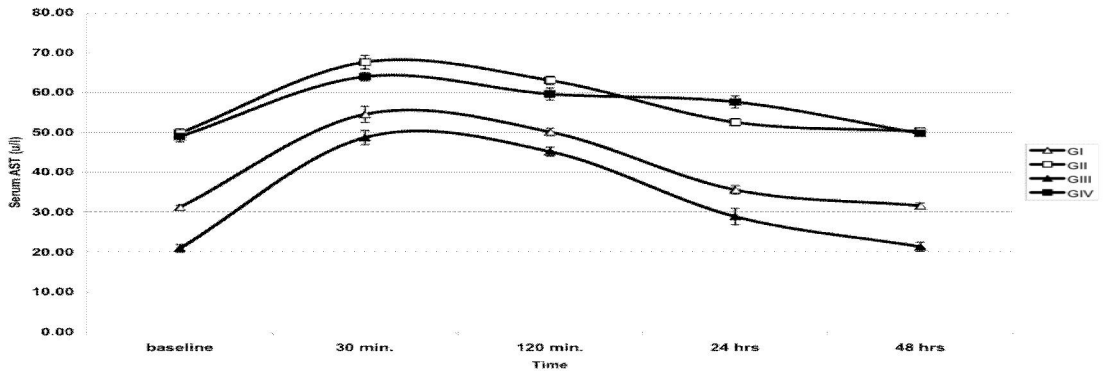



Fig. (8): Serum LDH (Mean±SD) before and after injection in all groups

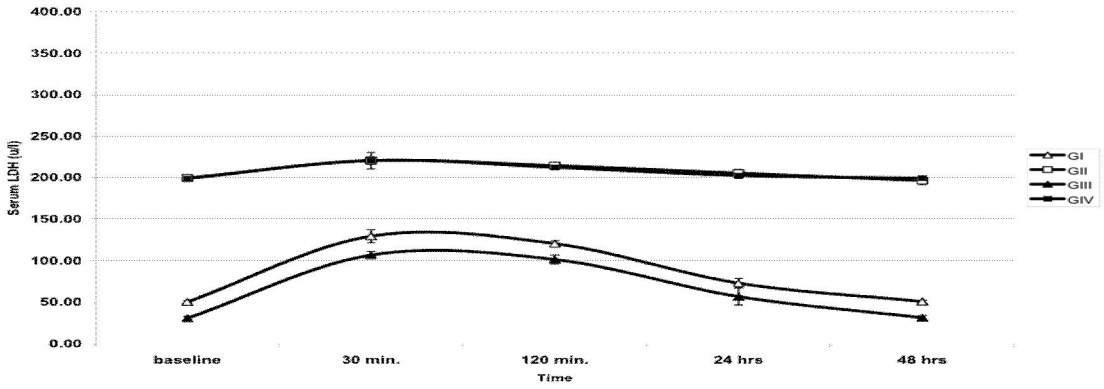


Fig. (9): Serum CPK (Mean±SD) before and after injection in all groups

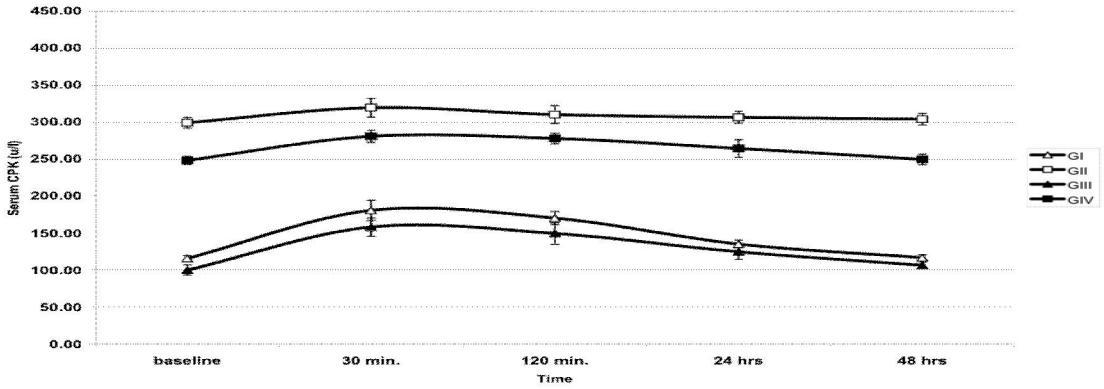



Fig. (10): Serum Urea (Mean±SD) before and after injection in all groups

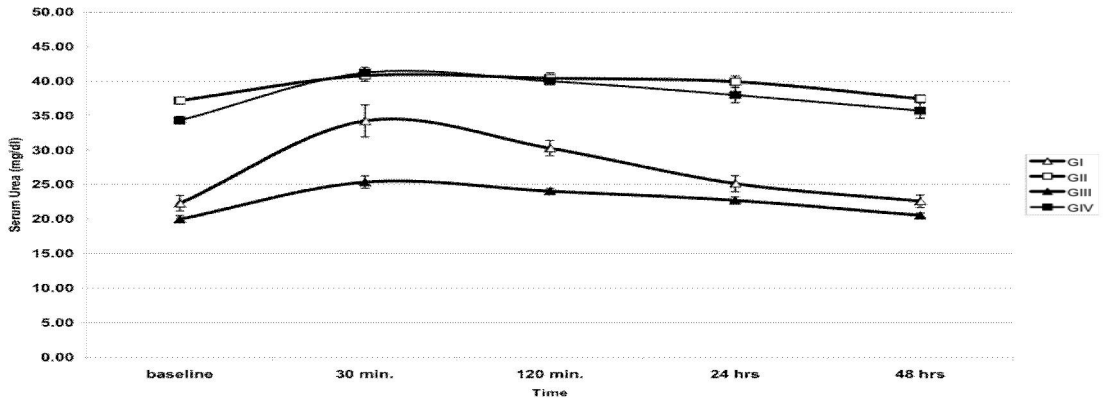
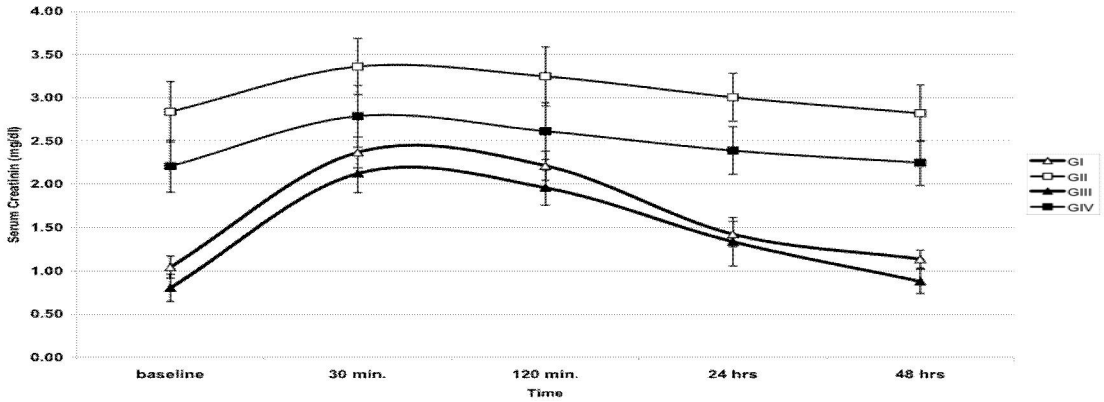



Fig. (11): Serum Creatinin (Mean+SD) before and after injection in all groups




## 4. Discussion

In our study, anesthetic induction (weak time and down time) did not change significantly in dogs before and after nephrectomy under the effect of either propofol or ketamine. On the other hands, ketamine induced significantly longer weak time than did propofol in corresponding groups. Similarly, ***Short and Bufalari, (1999) and Wagner and Hellyer, (2000)*** stated that, propofol is an intravenous anesthetic that has a rapid onset, short duration of action, rapid metabolism and does not result in biologically active metabolites. ***Zoran et al. (1993)*** reported that, rapid onset of propofol action is caused by rapid uptake into the CNS. Meanwhile, ***Lin (2007)*** stated that, because of its small molecular weight, a pKa near the physiological pH (7.5), and high lipid solubility, ketamine has a rapid onset of action, with maximal effect occurring in approximately 1 minute.

In this study, partial nephrectomy could not alter the common feature of the rapid anesthetic induction with either propofol or ketamine.

RRF was significantly longer in nephrectomized than non nephrectomized dogs under the effect of both agents. Recovery time was significantly longer in nephrectomized than non nephrectomized dogs under the effect of propofol but not ketamine. Ketamine caused significantly longer recovery time than did propofol in corresponding groups. ***Zoran et al. (1993)*** explained that, the short action and rapid smooth emergence of propofol result from rapid redistribution from the brain to other tissues and efficient elimination from plasma by metabolism. ***Guitton et al. (1998)*** explained that, the excretion rates of propofol glucuronide and of both glucuronide conjugates of the metabolite 1,4-di-isopropylquinol paralleled the corresponding plasma concentrations, but only for the first 15 hrs after induction of anesthesia. Thereafter, the water-soluble glucuronides are excreted via the kidney by the process of glomerular filtration. Meanwhile, termination of effect after a single bolus of ketamine is caused by rapid redistribution of the drug from brain to other tissues ***(Lin, 2007)***. ***White et al. (1982)*** had interpreted formerly that, the α-elimination phase of ketamine lasts only a few minutes, and the β-elimination half life is 2 to 3 hours. The compound is metabolized extensively by the hepatic cytochrome P450 system, by N-demethylation; its primary metabolite, norketamine, is only one third to one fifth as potent as the original compound. ***Aroni et al. (2009)*** added that, the metabolites of norketamine undergo renal excretion. Partial nephrectomy, in this study, had a noticeable effect on RRF and recovery times. It prolonged RRF time after injection of both propofol and ketamine, while it prolonged recovery time after propofol injection.

In this study, there were no significant differences in induction and recovery scores before and after nephrectomy in dogs anesthetized with either propofol or ketamine. However, propofol caused significantly better induction and recovery than did ketamine in corresponding groups. Similarly, ***Muir and Gadawski, (1998)*** reported that, the best features of propofol include the rapid induction of anesthesia, short duration of action, lack of excitatory side effects on induction and recovery, and no significant cumulative effects on repeated administration. In addition, propofol has been associated with high quality anesthetic recoveries in horses ***(Mama et al., 1996; Oku et al., 2003 and Boscan et al., 2006)***. They added that, recoveries from short anesthetic periods have been described to be smooth and calm, and with few attempts to stand. Both standing quality and overall recovery quality from the bolus injection of propofol were excellent, and all horses stood on their first attempt ***(Rezende et al., 2010)***. On the other hands, abnormal behavior, which may progress to delirium, may occur during emergence from ketamine ***Lin (2007).*** Depression of the inferior colliculus and medial geniculate nucleus leading to misperception of auditory and visual stimuli may be responsible for this reaction ***(White et al., 1982)***. Emergence reactions are characterized by ataxia, increased motor activity, hyperreflexia, sensitivity to touch, and sometimes violent recovery ***(Beck, 1976; Wright, 1982 and Velisek and Mares, 1990)***. These reactions usually disappear within several hours without recurrence. Abnormal behavior, in this study, was observed in three dogs and ataxia was also observed in two dogs after ketamine injection.

***Bleeker et al. (2008)*** hypothesized that reabsorption of propofol and its metabolites by the kidney is a major process in the overall elimination and that the resulting compounds are gradually conjugated and excreted in the urine. This may support our result that recovery not induction times were significantly longer in nephrectomized than non nephrectomized dogs under the effect of propofol. Conversely ***Hall et al.(2001)*** reported that, in man, liver disease and renal failure have little effect on pharmacokinetic parameters and it seems likely that extrahepatic mechanisms contribute to the metabolism of propofol, but they admitted that, these findings had not been investigated in any detail in other animals timely. On the other hand, clinically, animals with significant hepatic dysfunction do not metabolize ketamine as rapidly as do healthy animals ***(Short, 1987)***. He added that, animals with renal dysfunction or obstruction to urine flow also have prolonged sleep times when larger doses of ketamine are given**.** In our study a bolus intravenous ketamine dose was used and the resulted RRF time was longer but recovery time was not significantly longer in nephrectomized than non nephrectomized dogs. Generally speaking, ***Lin (2007)*** advised that, dissociative anesthetics should be given cautiously to animals that have significant hepatic or renal dysfunction**.**

In this study, propofol caused significant decrease but ketamine caused significant increase in heart rate and respiratory rate in both nephrectomized and non nephrectomized dogs. They both did not significantly alter rectal temperature. Partial nephrectomy did not affect these common features of both drugs. Similarly, ***Smith et al. (1993)*** reported that adverse cardiovascular and respiratory side effects have been reported after propofol injection, including hypotension, hypoventilation and apnea. ***Goodchild and Serrao (1989)*** explained that the

decrease in arterial blood pressure is believed to result from the combined effects of impaired myocardial contractility and a decrease in systemic vascular resistance. In this study, propofol caused apnea in 3 out of 7 non nephrectomized dogs and 4 out of nephrectomized ones. ***Kashiwagi et al. (2004)*** reported that, hypoventilation and apnea are mediated centrally via depression of central inspiratory drive and the ventilatory response to carbon dioxide. There is evidence that the incidence and severity of these adverse effects are increased when propofol is administered as a rapid bolus ***(Stokes and Hutton, 1991)*** and this may be of concern if propofol is to be used to achieve rapid anesthetic induction and endotracheal intubation in dogs. On the other hand, unlike many anesthetics, ketamine usually causes an increase in heart rate and arterial blood pressure as a result of increased sympathetic efferent activity ***(Wong and Jenkins, 1974)***. ***Hall et al. (2001)*** suggested that, this effect is probably due to an increase in circulating catecholamines caused by ketamine blocking the reuptake of noradrenaline by adrenergic nerve terminals. However, these cardiovascular effects may be unacceptable in some circumstances leading to the development of hypertension and tachycardia ***(Karapinar et al., 2006)***. On the other side, ***Booth (1988)*** reported that dissociative anesthetics, when given alone, differ from most other anesthetics in that they do not depress ventilatory responses to hypoxia. In dogs anesthetized with ketamine, respiratory rate and minute volume decrease initially, but both return to baseline values within 15 minutes ***(Haskins et al., 1985).***

In this study, ECG tracings showed no arrhythmias, only change in heart rate which was higher after ketamine injection than that after propofol in both nephrectomized and non nephrectomized dogs. Partial nephrectomy did not affect the figure of the tracing under the effect of both agents. These findings were in accordance with ***Branson and Gross (1994)*** who reported that, propofol is not inherently arrhythmogenic, but may enhance the arrhythmogenic effects of epinephrine. Propofol, in our study was used with a small bolus dose that could not enhance epinephrine induced arrhythmia. Similarly, ***Hall et al. (2001)*** reported that, cardiac arrhythmias are uncommon in animals under ketamine anesthesia and the minimal arterial blood pressure is always similar to and rarely less than the preanesthetic level.

Significant increases in AST, LDH, CPK, urea and creatinine were observed in all groups in this study with minor disparity from one to another group. Baseline values were significantly higher in nephrectomized dogs than those in non nephrectomized ones. The influence of lower pole nephrectomy on the action of either propofol or ketamine was minimum and could be neglected. ***Lucia and Jacqueline (2009)*** reported that, AST is not an organ-specific and skeletal muscle contains the highest concentration followed by liver and cardiac muscle. The enzyme half life is about 22 hours in dogs and may increase due to muscle trauma and muscle diseases. They added that, LDH is an enzyme that catalyzes the conversion of lactate to pyruvate. It is found in variety of tissues including liver, heart and skeletal muscles. It increases in muscle and heart diseases. ***Madej and Stańczyk (1975)*** stated that, CPK is a leakage enzyme present in high concentration in the cytoplasm of myocytes. It has a very short half life about 1 hour**.** They found also that ketamine anesthesia in dogs and cats causes a slight reversible damage to the liver and kidneys and increases the activity or reticuloendothelial cells in the organism.

We hypothesized that, elevation of AST, LDH and CPK, in this study, might be due to muscle twitching and liver action after ketamine injection as well as, due to liver and myocardial action after propofol injection. These findings were supported by ***Chen, et al. (2000)*** who reported that, there are several lines of evidence indicating that propofol induces subclinical and reversible disturbance in hepatocellular integrity in human by affecting the serum level of hepatic enzymes in vivo after long term infusion. Our findings also were in agreement with ***Franco et al. (2004)*** who stated that, the increased CPK and AST serum activity in ketamine-treated group imply that anesthesia induced alteration in skeletal and cardiac muscles. They added that, such alterations were considered transient and returned to baseline values rapidly. Generally, ***Corssen et al. (1968)*** and ***Dundee et al. (1978)*** reported that, hepatic dysfunction following clinical use of ketamine, and other dissociatives, is not evident in either people or dogs. They added that, a significant increase in serum concentrations of liver enzymes has been observed in people anesthetized with a ketamine infusion and dogs given higher intramuscular doses.

***Burkholder (2000)*** suggested that 15-20% of dogs have renal insufficiency as renal blood flow and glomerular filtration rate reduced under the effect of anesthesia. He added that, even if anesthesia increases sympathetic stimulation as in case of dissociative anesthesia, renal blood flow also reduced. Pre-anesthetic fasting caused uremia which might affect the drug activity and toxicity. It affects the ability of albumin to bind to drugs and increase the proportion of free drug in plasma ***(Fishman, 1970)***.

It could be concluded that, although ketamine had better cardiopulmonary effect than propofol, the later was superior in the quality of induction and recovery. Lower pole nephrectomy in dogs had minimal impact on the modality of the effect of either agent. The only evident alteration was the relatively prolonged recovery times especially after propofol injection. Further investigations are needed to discover the effect of the variation in the size and/or location of the nephrectomized portion of the dogs' kidney on the anesthetic effect.

## 5. Acknowledgement and Correspondence

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## 6. References

***Aroni, F.; Iacovidou, N.; Dontas, I.; Pourzitaki, C. and Xanthos, T. (2009):*** Pharmacological aspects and potential new clinical applications of ketamine: reevaluation of an old drug. *J. Clin. Pharmacol., 49 (8): 957-964.*

***Beck, C. C. (1976):*** Vetalar (ketamine hydrochloride): A unique cataleptoid anesthetic agent for multispecies usage. *J. Zoo Anim. Med., 7: 11-38.*

***Bleeker, C.; Vree, T.; Lagerwerf, A. and Willems-van Bree, E. (2008):*** Recovery and long-term renal excretion of propofol, its glucuronide, and two di-isopropyl quinol glucuronides after propofol infusion during surgery. *British Journal of Anaesthesia, 101 (2): 207–212.*

***Booth, N. H. (1988):*** Intravenous and other parenteral anesthetics. *In: Booth, N, H., McDonald, L. E, eds. Veterinary Pharmacology and Therapeutics. Ames, IA: Iowa State University Press, 212.*

***Boscan, P.; Steffey, E. P.; Farver, T. B.; Mama, K. R.; Huang, N. J. and Harris, S. B. (2006):*** Comparison of a high (5%) and low (1%) concentrations of micellar microemulsion of propofol formulation with a standard (1%) emulsion in horses. *Am. J. Vet. Res., 67(9):1476-1483.*

***Branson, K. E. and Gross, M. E. (1994):*** Propofol in veterinary medicine. *J. Am. Vet. Med. Assoc. 204: 1888-1890.*

***Brüssel, T.; Theissen, J. L.; Vigfusson, G.; Lunkenheimer, P. P.; Van Aken, H. and Lawin, P. (1989):*** Hemodynamic and cardio dynamic effects of propofol and etomidate: negative inotropic properties of propofol. *Anesth. Analg. 69, 35–40.*

***Burkholder, W. J. (2000):*** Dietary considerations for dogs and cats with renal disease. *J. Am. Vet. Med. Assoc. 216: 1730-1734.*

***Carroll, G. L.; Hooper, R. N. and Slater, M. R. (1998):*** Detomidine-butorphanol-propofol for carotid artery translocation and castration or ovariectomy in goats. *Vet. Surg., 27, 75–82.*

***Chen, T. L.; Wu, C. H.; Chen, T. G.; Tai, Y. T.; Chang, H. C. and Lin, C. J. (2000):*** Effect of propofol on functional activities of hepatic and extrahepatic conjugation enzyme systems. *Br. J. Anesth. 84 (6) 771-776.*

***Corssen, G.; Miyasaka, M. and Domino, E. F. (1968):*** Changing concepts in pain control during surgery: Dissociative anesthesia with CI-581—A progress report. *Anesth. Analg., 47 (6): 746-759.*

***Dundee, J. W.; Fee, J. P. H.; Moore, J.; McIlroy, P. D. and Wilson, D. B. (1978):*** Liver function studies after ketamine infusions. *Br. J. Clin. Pharmacol. 6 (5): 450-451.*

***Fishman, R. A. (1970):*** Permeability changes in experimental uremic encephalopathy.*Archives of Internal Medicine 126: 835-837.*

***Franco L. G.; Fioravanti, M. C. S.; Damasceno, A. D.; Borges, A. C.; Soares, L. K.; Rabelo, R. E. and da Silva, L. A. F. (2004):*** Assessment of serum enzymatic markers of cardiomyocytes injury in female dogs submitted to ketamine S(+), atropin and xylazine association. *Acta Cirurgica Brasileria 24(1): 36-42.*

***Glowaski, M. M. and Wetmore, L. A. (1999):*** Propofol: application in veterinary sedation and anesthesia. *Clin. Tech. Small Anim. Pract. 14, 1–9.*

***Goodchild, C. S. and Serrao, J. M. (1989):*** Cardiovascular effects of propofol in the anaesthetized dog. *Br. J. Anaesth., 63(1):87-92.*

***Guitton, J.; Buronfosse, T.; Desage, M.; Flinois, J. P.; Perdrix, J. P.; Brazier, J. L. and Beaune, P. (1998):*** Possible involvement of multiple human cytochrome P450 isoforms in the liver metabolism of propofol. *Br. J. Anaesth., 80 (6): 788–795.*

***Hall, L. W.; Clarke, K. W. and Trim, C. M.(2001):*** Veterinary Anesthesia (10th ed.). W. B. Saunders, London, UK, pp.123–124.

***Haskins, S. C.;Farver, T. B. and Patz, J. D. (1985):*** Ketamine in dogs. *Am. J. Vet. Res. 46:1855-1860.*

***Karapinar, B.; Yilmaz, D.; Demirag, K. and Kantar, M. (2006):*** Sedation with intravenous ketamine and midazolam for painful procedures in children. *Pediatr. Int. 48, 146–151.*

***Kashiwagi, M.; Okada, Y.; Kuwana, S.; Sakuraba, S.; Ochiai, R. and Takeda, J. (2004):*** A neuronal mechanism of propofol-induced central respiratory depression in newborn rats. *Anesth. Analg., 99, 49-55.*

***Lanning, C. F. and Harmel, M. H. (1975):*** Ketamine anesthesia. *Annu. Rev. Med. 26: 137-141.*

***Lin, H. C. (2007):*** Dissociative anesthetics. *InTranquilli W. J., Thurmon J. C. Grimm K. A.* eds. Lumb & Jones' Veterinary Anesthesia and Analgesia, 4th ed. Blackwell Publishing, Ames. Iowa. p. 301.

***Lin, H.C.; Purohit, R. C. and Powe, T. A. (1997):*** Anesthesia in sheep with propofol or with xylazine-ketamine followed by halothane. *Vet. Surg., 26, 247–252.*

***Lucia, A. and Jacqueline, C. W. (2009):*** Liver enzyme elevations in dogs: Physiology and Pathophysiology. *Compendium Vet. Com. CE Article 1, 408-414.*

***Madej, J. A. and Stańczyk, J. F. (1975):*** Effect of ketamine anesthesia on enzyme activity in organs of dogs and cats. *Anaesth Resusc Intensive Ther., 3(4): 297-303.*

***Mama, K. R.; Steffey, E. P. and Pascoe, P. J. (1996):*** Evaluation of propofol for general anesthesia in premedicated horses. *Am. J. Vet. Res. 57, 512–516.*

***Muir, W. W. and Gadawski, J. E. (1998):*** Respiratory depression and apnea induced by propofol in dogs. *Am. J. Vet. Res. 59, 157–161.*

***Oku, K.; Yamanaka, T.; Ashihara, N.; Kawasaki, K.; Mizuno, Y. and Fujinaga T. (2003):*** Clinical observations during induction and recovery of xylazine-midazolam-propofol anesthesia in horses. *J. Vet. Med. Sci., 65 (7): 805-808.*

***Prassinos, N. N.; Galatos, A. D. and Raptopoulos, D. (2005):*** A comparison of propofol, thiopental or ketamine as induction agents in goats. *Vet. Anesth. Analg. 32, 289-296.*

***Rezende, M. L.; Boscan, P.; Stanley, S. D.; Mama, K. R. and Steffey, E. P. (2010):*** Evaluation of cardiovascular, respiratory and biochemical effects, and anesthetic induction and recovery behavior in horses anesthetized with a 5% micellar micro emulsion propofol formulation. *Vet. Anaesth. Analg., 37, 440–450.*

***Sams, L.; Braun, C.; Allman, D. and Hofmeister, E. (2008):*** A comparison of the effects of propofol and etomidate on the induction of anesthesia and on cardiopulmonary parameters in dogs. *Veterinary Anaesthesia and Analgesia, 35, 488–494*

***Short, C. E. (1987):*** Dissociative anesthesia. *In: Short, C. E., ed. Principles and Practice of Veterinary Anesthesia. Baltimore: Williams and Wilkins, 158.*

***Short, C. E. and Bufalari, A. (1999):*** Propofol anesthesia. *Vet. Clin. North. Am. Small Anim. Pract. 29 (3):747-778.*

***Smith, J. A.; Gaynor, J. S.; Bednarski, R. M. and Muir, W. W. (1993):*** Adverse effects of administration of propofol with various preanesthetic regimens in dogs. *J. Am. Vet. Med. Assoc., 202, 1111–1115.*

***Snedecor, G. W. and Cochran, W. G. (1967):*** Statistical Methods, *6th edn, (Oxford and TBH, New Delhi).*

***Stokes, D. N. and Hutton, P. (1991):***Rate-dependent induction phenomena with propofol: implications for the relative potency of intravenous anesthetics. *Anesth. Analg., 72(5): 578-583.*

***Velisek, L. and Mares, P. (1990):*** Anticonvulsant action of ketamine in laboratory animals. *In: Domino EF, ed. Status of Ketamine in Anesthesiology. Ann Arbor, MI: NPP: 541.*

***Wagner, A. E. and Hellyer, P. W. (2000):*** Survey of anesthesia techniques and concerns in private veterinary practice. *J. Am. Vet. Med. Assoc., 217:1652–1657.*

***White, P. F.; Way, W. L. and Trevor, A. J. (1982):*** Ketamine- Its pharmacology and therapeutic uses. *Anesthesiology. 56 (2):119-136.*

***Wong, D. H. W. and Jenkins, L. C. (1974):*** An experimental study of the mechanism of action of ketamine on the central nervous system. *Can. Anaesth. Soc. J., 21, 57-67.*

***Wright, M. (1982):*** Pharmacologic effects of ketamine and its use in veterinary medicine. *J. Am. Vet. Med. Assoc., 180 (12):1462-1471.*

***Zoran, D. L.; Riedesel, D. H. and Dyer, D. C. (1993):*** Pharmacokinetics of propofol in mixed breed dogs and greyhounds. *Am. J. Vet. Res., 54 (5): 755-760.*