A COMPARATIVE EVALUATION OF HALOTHANE AND ISOFLURANE ANAESTHESIA FOR OVARIOHYSTERECTOMY IN BITCHES

NAGARAJ, M.

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
VETERINARY COLLEGE, BANGALORE
KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY, BIDAR

JULY, 2009

A COMPARATIVE EVALUATION OF HALOTHANE AND ISOFLURANE ANAESTHESIA FOR OVARIOHYSTERECTOMY IN BITCHES

Thesis submitted to the
Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar
in partial fulfillment of the requirements
for the award of the degree of

Master of Veterinary Science

In

VETERINARY SURGERY AND RADIOLOGY

By

NAGARAJ, M.

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
VETERINARY COLLEGE, BANGALORE
KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY, BIDAR

JULY, 2009

KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY, BIDAR.

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
VETERINARY COLLEGE, BANGALORE

CERTIFICATE

This is to certify that the thesis entitled "***A COMPARATIVE EVALUATION OF HALOTHANE AND ISOFLURANE ANAESTHESIA FOR OVARIOHYSTERECTOMY IN BITCHES***" submitted by **Mr. NAGARAJ, M.** I.D. No. MVHK-518 in partial fulfillment of the requirements for the award of **MASTER OF VETERINARY SCIENCE** in **VETERINARY SURGERY AND RADIOLOGY** of the Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar is a record of bonafide research work carried out by him during the period of his study in this University under my guidance and supervision and the thesis has not previously formed the basis for the award of any degree, diploma, associateship, fellowship or other similar titles.

Bangalore,
July , 2009

(Dr. R. NAGARAJA)
Major Advisor

APPROVED BY:

Chairman : _____
(Dr. R. NAGARAJA)

Members : 1. _____
(Dr. M. DEVARAJ)

2. _____
(Dr. M. S. VASANTH)

3. _____
(Dr. L. RANGANATH)

4. _____
(Dr. V. GIRISH KUMAR)

Affectionately dedicated

to my beloved family members

Parents		*Brothers*
Sri S.R. Munirathnaiah Setty	&	*Harish, M.*
Smt. Jayalakshmamma		*Vasudeva, M.*

ACKNOWLEDGEMENT

I place an record my deep sense of gratitude and heartfelt thanks to my Major Advisor ***Dr. R. NAGARAJA****, Professor, for his scholarly guidance, timely advice, constant encouragement and inspiring moral support during all the stages of this study. His sustained interest in my research work and timely help enabled me to complete the investigation in time.*

I am equally grateful to ***Dr. M.S. Vasanth****, Dean Veterinary College Hassan and* ***Dr. L. Ranganath****, Professor and Head, Dept. of Veterinary Surgery and Radiology, for their timely advice and guidance throughout my postgraduate programme. I will be very greatful to all my teachers, who thought me a wild life surgery, lameness, radiology, disciplined surgery, how to analyse, tackle veterinary emergency cases and veterinary client education.*

I am very much grateful to my Advisory Members, ***Dr. M. Devaraj****, Registrar, Karnataka Veterinary, Animal And Fisheries Sciences University and* ***Dr. V. Girish Kumar****, Professor and Head, Dept. of Veterinary Bio-chemistry for their useful suggestions and guidance during the study.*

I take this occasion to thank ***Dr. Nalini Pradeep****, Professor and Head (Retd.), Dept. of Veterinary Pathology Veterinary College, Bangalore for her timely advice and guidance.*

I thank all the staff of Dept. of Veterinary Surgery and Radiology, for their affectionate help and moral support, extended during the course of this study.

I am indebted to my close friends and my fellow postgraduate colleagues ***Drs. Mahendra, Mahesh, Yeshwanth, Gaurav, Ravi K.B.****,* ***Bhanuprakash, K.C.*** *and* ***Dr. Chandan*** *for their unforgettable support and affection all through my work.*

I am also grateful to my junior colleagues ***Drs. Vishal, Saiprakash, Venkatagiri, Vani*** *and undergraduate students* ***Manikantha, Shashank, Vishwaradya, Ashwini*** *and* ***Gokula*** *group who were always there with their cordial help and cooperation during my research work.*

I wish to thank ***Dr. H.A.K. Nayeem, Dr. Onkarappa, Dr. Vinay Kumar*** *and* ***Dr. Prashanth*** *working in State Livestock Breeding and Training Center, Hesaraghatta for there constant encourage.*

I heartily acknowledge the constant inspiration, moral and financial support of my beloved Father ***Sri S.R. Munirathnaiah Setty*** *and my ever loving Mother* ***Smt. Jayalakshmamma*** *and encouragement of dear Brothers* ***Harish, M.*** *and* ***Vasudeva, M.*** *with gratitude throughout my study.*

I extend my thanks to ***Sri Vinayaka Printers****, Hebbal for neat printing and binding in time and I also thanks for those who have knowingly and unknowingly helped in accomplishment of this thesis.*

Bangalore,
July , 2009

(NAGARAJ MUNIRATHNAIAH SETTY)

CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. INTRODUCTION

II. REVIEW OF LITERATURE

III. MATERIALS AND METHODS

IV. RESULTS

V. DISCUSSION

VI. SUMMARY

VII. BIBLIOGRAPHY

VIII. ABSTRACT

I. INTRODUCTION

The pioneering studies on clinical usage of inhalation anaesthetics began with usage of gaseous anaesthetics such as nitrous oxide and cyclopropane, and volatile liquids *Viz.*, ethyl chloride, diethyle ether or chloroform. Presently the latest entrants to the list of inhalation anaesthesia *Viz.*, enflurane, isoflurane, desflurane and sevoflurane, which are increasingly becoming popular than injectable anaesthetics because of their unique properties among general anaesthetics. Inhalants are eliminated from the body primarily through the lungs whereas hepatic inactivation and renal elimination are not strictly necessary to permit a patient to regain consciousness from inhalation anaesthesia. The safety feature of inhalation anaesthetics is mainly attributed to their volatile nature, which facilitates its easy absorption and elimination (80 percent or more) through lungs and only a minor portion (20 percent or less) to undergo biotransformation.

Halothane is a multi halogenated ethane, clear volatile liquid, non-inflammable and possess properties of rapid induction, having good potency and rapid elimination from system with minimal side effects to the patients. In dogs, halothane having a MAC value of 0.87% tends to cause dose dependent cardiac and respiratory depression (Hall, 1957) in addition frequent cardiac arrhythmias during clinical use (Tranquilli, 1986). Frequent exposure to halothane is known to result in hepatotoxicity in patients (Topal *et al.*, 2003).

Isoflurane is an isomer of the inhalant anaesthetic enflurane, introduced into veterinary practice during early eighties. Similar to fluorinated hydrocarbon

halothane, isoflurane is nonflammable and non-explosive in clinically effective concentrations. Isoflurane with MAC value of 1.31% is slightly less potent than halothane in dogs. Similar to other anaesthetics isoflurane also causes hypothermia, dose dependent cardiac and respiratory depression (Meyer *et al.*, 1984). Compared to halothane, isoflurane posses lower blood solubility and faster induction rate, which facilitates early recovery from anaesthesia (Jones and Seymour, 1986). Thus the popularity of isoflurane in veterinary practice reflects its cardiovascular stability, low blood solubility, resistance to hepatic metabolism and more of patient safety (Werner, 1987; Martinez *et al.*, 1996; Mutoh, 2001; Guochang Hu *et al.*, 2002; Topal *et al.*, 2003 and Kilic and Isler, 2005).

Therefore usage of anaesthetics having characteristics of rapid induction and early elimination with minimal side effects is not only advantageous to the patient but it also minimizes the possible health hazards to both the surgeon and operating team and animal care takers. In view of above, the present study was conducted with the following objectives:

1. To assess and compare clinical efficacy of halothane and isoflurane anaesthesia in bitches undergoing routine ovariohysterectomy procedure.
2. To asses and compare the effect of halothane and isoflurane inhalant anaesthetics on clinical parameters *Viz.*, rectal temperature (° F), respiratory rate (per minute) and heart rate (per minute) in bitches undergoing ovariohysterectomy procedure.
3. To assess and compare the influence of halothane and isoflurane anaesthesia on haematological and biochemical parameters *Viz.*, total erythrocyte count (TEC),

packed cell volume (PCV), hemoglobin concentration (Hb), total leukocyte count (TLC), differential leukocyte count (DLC), platelet count (PLT), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), plasma creatinine, total plasma protein (TPP), electrolytes (sodium, potassium and chloride ions) and blood glucose levels during and after anasesthesia.

4. To record complications and emergencies if any, during induction, maintenance and recovery phases of inhalant anaesthesia in bitches subjected for routing ovariohysterectomy procedure.

II. REVIEW OF LITERATURE

The available literature on comparative evaluation of halothane and isoflurane anaesthesia for ovariohysterectomy in bitches is reviewed under the following headings.

2.0 Preanaesthetic medication

Inhalation anaesthesia is often initiated with premedication using sedatives, tranquilizers and anticholinergic drugs. The choice of premedication varies with species, temperament, physical status of the patient and the procedure to be performed as well the personal preferences. During inhalation anaesthesia premedication aid in restraining, by reducing apprehension and also decreases the quantity of anaesthetic agents required to produce general anaesthesia. Premedicants are usually administered i/v or s/c 15 to 20 min prior to induction.

Wiersig (1974) reported epinephrine or nor epinephrine induced ventricular fibrillation in experimental dogs anaesthetized with combination of ultra short acting barbiturates and halothane. The treatment of barbiturate-halothane anaesthetized dogs with acepromazine maleate, chlorpromazine hydrochloride or propranolol before administration of the catecholamine's protected the dogs against ventricular fibrillation.

Lang *et al.* (1979) studied the effect of acetylpromazine on canine haematology and found that consistent fall in haematological values in first hour after

administration. They attributed this could be due to adrenergic suppression causing relaxation of splenic capsule.

Jones and Snowdon (1986) studied the cardiovascular and respiratory effects of the inhalation anaesthetic agent isoflurane in dogs. Anaesthesia was induced with thiopentone after premedication with acepromazine. Isoflurane was administered with nitrous oxide and oxygen by spontaneous ventilation after base line values had been determined. Arterial blood pressure decreased as the concentration of administered isoflurane increased. Isoflurane produced a profound and dose related respiratory depression as measured by the increase in end tidal carbon dioxide levels. Isoflurane administration did not produce any visible muscle twitching.

Mansell and Parry (1992) studied the effect of acepromazine maleate, xylazine and thiopentone on the packed cell volume, plasma protein content, factor VIII activity and Von Willebrand factor antigen concentration of blood in normal dogs. These variables were measured in dogs with haemophilia A, administered with acepromazine maleate and thiopentone. They reported that both the packed cell volume and plasma protein content decreased after the administration of either acepromazine maleate or xylazine.

Smith *et al.* (1993) studied the effects of propofol on anaesthetic induction in forty dogs anaesthetized with isoflurane under four preanaesthetic regimens like anaesthesia without preanaesthetic drugs; or with preanaesthetic administration of acepromazine (0.1 mg/kg of body weight, i/m), diazepam (0.2 mg/kg, i/v), or acepromazine (0.02 mg/kg) and butorphanol (0.4 mg/kg, i/m). The data recorded

about parameters like heart rate, systolic arterial blood pressure (SAP), respiration, quality of induction and recovery shows that the SAP significantly decreased after propofol administration in dogs treated with acepromazine and with acepromazine and butorphanol.

Wright *et al.* (1996) measured the cardiac electro-physiological variations before and after intravenous administration of atropine (0.04 mg/kg of body weight) and propanolol in isoflurane-anaesthetized healthy dogs. They concluded that cardiac electro-physiological variations vary widely among healthy, isoflurane-anaesthetized dogs.

Robertson *et al.* (2001) conducted a study to assess the influence of preanaesthetic administration of acetylpromazine (acepromazine) or morphine and fluids on urine production, arginine vasopressin (previously known as antidiuretic hormone) concentrations, mean arterial blood pressure, plasma osmolality, PCV, and concentration of total solids during halothane anaesthesia and surgery in dogs. They observed that compared with values for acetylpromazine, preoperative administration of morphine resulted in significantly lower urine output during the surgical period.

Teixeira-Neto *et al.* (2001) studied the effects of atropine and methotrimeprazine on epinephrine induced ventricular arrhythmias in halothane anaesthetized dogs {ten mixed-breed dogs were assigned to 3 treatments (saline, atropine, and methotrimeprazine)} in a randomized complete block design. Anaesthesia was induced and maintained with halothane (1.5 % minimum alveolar concentration) in oxygen. They analyzed the heart rate and arterial blood pressure

found that the arrhythmogenic dose of epinephrine increased in atropine and methotrimeprazine treated groups.

Hui Cheng Chen *et al.* (2003) used acepromazine as one of the premedicant for dogs under going different surgical procedures and came out with results indicating that treatment with ephedrine (0.2 mg/kg i/v) in hypotensive dogs under isoflurane anaesthesia caused an increase in mean arterial pressure.

Adams *et al.* (2006) used acepromazine as premedicant to evaluate the non-depolarizing neuromuscular blocking drug cis-Atracurium in dogs with Porto systemic shunts and compare with clinically normal dogs and concluded that cis-Atracurium may have a use in veterinary anaesthesia for producing neuromuscular blockade in dogs with hepatic insufficiency, including those with Porto systemic shunt.

Steagall *et al.* (2006) employed acepromazine as one of the premedicant to evaluate the isoflurane sparing effects of lidocaine and fentanyl administration by constant rate infusion during surgery in dogs and found that administration of fentanyl resulted in greater isoflurane sparing effect than did lidocaine.

Brodbelt *et al.* (2006) carried out a case control study, undertaken retrospectively from dogs anaesthetized at the Queen Mother Hospital for Animals, Royal Veterinary College (from February 1999 to April 2002) to identify the major risk factors associated with anaesthetic related mortality and estimated the mortality risk in a United Kingdom canine referral practice population. Potential risk factors

assessed included patient health status, age, premedication with acepromazine, premedication with opioids, overall premedication and induction with thiopentone or propofol, maintenance of anaesthesia with halothane or isoflurane and duration of anaesthesia. They observed that the use of acepromazine was associated with reduced risk of death compared to other premedicants.

2.1 Halothane anaesthesia

Hall (1957) reported that induction of anaesthesia with halothane was smooth and rapid and recovery of anaesthesia was also rapid in dogs. During anaesthesia depression in respiration, pulse rate and blood pressure was observed.

Fisher (1961) studied the effects of halothane anaesthesia in domestic animals. The anaesthesia caused a depression of the ventilation rate in all species and concomittent to this the respiratory acidosis occurred. Removal of the animal from the anaesthetic atmosphere led to the rapid return of the ventilation rate to normal as a consequence of recovery from the respiratory acidosis.

Carter (1964) successfully administered halothane through a standard Boyle's anaesthetic apparatus at a flow rate varying between 1.25 to 7.5 liters per min depending upon the size of dog.

Krister Iwarsson (1966) anaesthetized seven dogs with fluothane and observed that an increase in PCo_2 and PO_2 and decrease in pH and blood pressure during the course of anaesthesia.

Halnan (1967) administered halothane at the rate of 4 per cent for induction and 2.5 per cent of maintenance through a blease vaporizer and stated that this technique could be applied by a supervised lay assistant and easily learned. The anaesthetist knows exactly how much anaesthetic is being delivered.

Little John and Mitchell (1969) concluded that the accuracy of capillary samples of blood was acceptable for clinical investigation only in the case of pH but not for PCo_2 (pressure of Co_2) and HCo_3 (bicarbonate).

Bouda and Pavalica (1970) found that respiratory acidosis occur during halothane anaesthesia in cows. There was a fall in pH, an increase in PCo_2 and PO_2 after the anaesthesia, a gradual adjustment of the acid base state was observed and equilibrization took place within three hours.

Gates *et al.* (1971) studied the blood gas chemistry in cattle anaesthetized with halothane and reported that an increase in Co_2 value, an increase in O_2 tension and decrease in pH as anaesthesia deepened. But the above values returned to normal during recovery period. These results confirmed that acidosis developed during halothane anaesthesia but also demonstrated the efficiency of buffer system of the body in controlling the adverse effects.

Steffey *et al.* (1974) reported that halothane did not significantly reduce aortic pressure, pulmonary artery pressure, and cardiac out put, stroke volume, left ventricular work and total peripheral resistance in dogs.

Wiersig *et al.* (1974) recorded ventricular fibrillation in seven of fifteen halothane anaesthetized dogs following intravenous injection of epinephrine or nor epinephrine.

Skarda (1976) compared arterial and venous blood pressure for determination of blood gas acid base in the horse under halothane anaesthesia and concluded that blood obtained from jugular vein can be used for blood gas chemistry and clinical purposes instead of arterial blood.

Steffey *et al.* (1976) recorded a decrease in haematocrit and blood protein concentration in dogs and monkeys administered with halothane.

Bagshaw *et al.* (1978) reported a rapid rise in the rectal temperature in young male grey hound dogs anaesthetized with halothane and Suxamethonium.

Sharma (1978) reported that in dogs anaesthetized with halothane, the respiratory tidal volume directly related to the body weight and found no periodic deep breaths when compare to dogs anaesthetized with methoxyflurane.

Chambell *et al.* (1980) reported that halothane caused hypotension, thus reducing blood supply to liver resulting hypoxia in liver which may lead to hepatic damage.

Deyoung and Sawyer (1980) concluded that nitrous oxide was a useful adjunct to halothane anaesthesia inspite of its relatively low potency in dogs.

Pedersoli (1980) reported that an increase in concentration of serum bromide during and after halothane anaesthesia in dogs and discussed about the possible selective or stimulant levels of increased serum bromide concentration are discussed.

Rose *et al.* (1980) studied physiological and biochemical effects of intravenous fluids administration during halothane anaesthesia in dogs. They reported that, anaesthesia alone and also during anaesthesia there will be a decrease in temperature, mean arterial pressure, PCV, total protein and respiratory rate.

Sluij *et al.* (1983) reported that in inhalant anaesthetic administration the capillary and venous blood samples can be used instead of arterial blood samples for measurements of acid base and blood gas status in dogs in cases of severe impairment of lung function mainly hypoxemia, hypercapnoea and hypovolumia.

Taylor and Hall (1985) studied the effects of halothane and enflurane as maintenance anaesthesia in horses in which anaesthesia was induced with xylazine and ketamine. They observed that there was greater respiratory depression and no significant difference in arterial blood pressure, blood gases and haematological parameters.

Kelly *et al.* (1985) anaesthetized fourteen horses with halothane in oxygen for eight hours and reported a reduction in number of platelets which returned to normal within 24 hours.

Tranquilli *et al.* (1986) studied the alteration in the arrhythmogenic dose of epinephrine following xylazine administration to halothane-anaesthetized dogs. They

found that at the end of intravenous. Xylazine bolus administration, heart rate decreased and means arterial pressure was increased.

Leone *et al.* (1988) conducted a study to know the effect of equal local anaesthetic potency doses of lignocaine or bupivacaine in halothane-anaesthetized dogs. They found that both lignocaine and bupivocaine anaesthetics caused the depression of global haemodynamic function and regional myocardial dysfunction.

Gross *et al.* (1997) studied the effectiveness of the infraorbital and inferior alveolar nerves regional anaesthesia in halothane anaesthetized dogs for dental procedures and concluded that regional anaesthesia of the infraorbital and inferior alveolar nerves may effectively provide analgesia for dental procedures in dogs.

Torske *et al.* (1999) studied the cardiovascular effects of epidurally administered oxymorphone and oxymorphone-bupivacaine combination in six dogs anaesthetized with halothane. They recorded the heart rate, systematic and pulmonary arterial pressures, central venous pressure and cardiac output. They found that epidural administered oxymorphone uptake was rapid with decreased heart rate, whereas glycopyrrolate administration improved heart rate resulting in improved cardiac index.

Yang *et al.* (1999) studied the anaesthetic and cardiovascular effects of xylazine-fentanyl-azaperone and medetomidine-midazolam preanaesthetic medications and their combinations with antagonists (yohimbine, atipamezole) in halothane-anaesthetized dogs. They recorded the recovery (pedal reflex recurrence

time, extubation time, arousal time, standing time and walking time) parameters. They concluded that preanaesthetic administration of xylazine-fentanyl-azaperone or medetomidine-midazolam with their antagonists is suitable for rapid sedation, prevention of apnoea after intubation and rapid recovery after halothane anaesthesia.

Fox *et al.* (2000) studied the effects of ovariohysterectomy plus different combinations of halothane anaesthesia and butorphanol analgesia on behaviour in the bitches. Behaviour was monitored while the bitches were alone (non-interactive) and when routinely examined and handled prior to blood collection (interactive) and they concluded that behaviour of bitches during study period after ovariohysterectomy reflects as an ovariohysterectomy is a painful procedure.

Redondo *et al.* (2000) studied the percentage of halothane necessary for maintaining anaesthesia in dogs by using romifidine as a premedicant. The induction was carried out with propofol and the study concluded that the combination of romifidine, atropine, propofol, halothane and N_2O appears to be an effective combination for inducing and maintaining general anaesthesia in healthy dogs.

Brondani *et al.* (2004) evaluated the cardiovascular alterations and analgesia in fourteen dogs submitted to epidural administration of clonidine or romifidine to enable coxofemoral surgery. Anaesthetic was induced using propofol (8 mg/kg) and maintained using halothane and O_2 in spontaneous breathing. The heart rate and respiratory rate, systolic arterial rate, haemoglobin oxygen saturation and halothane concentration were assessed before anaesthetic induction and every 10 minutes until the end of the surgery. Samples of arterial blood were collected to assess pH, $PaCo_2$,

PaO_2, SaO_2 and HCO_3 - levels and reported that dogs with romifidine group had bradycardia, bradyarrhrythmia and hypertension.

Joubert (2004) monitored the anaesthetic depth in ten dogs based on the subjective assessment of the patient to determine if the AEP (auditory-evoked potential) monitor was useful in dogs. After clinical and otoscopic examination, dogs were premedicated with acetylpromazine and morphine and anaesthesia was induced with thiopentone and maintained with halothane. End-tidal carbon dioxide, temperature, pulse oximetry, blood pressure and the electrocardiogram were monitored and recorded every five minutes. He concluded that the AAI (A-line ARX-Index) Index shows good prospect for the evaluation of anaesthetic depth in dog's undergone surgery.

Lavor *et al.* (2004) used halothane as maintenance anaesthesia in twenty bitches undergoing caesarean section and their 129 puppies were divided into 4 groups according to the anaesthetic protocol. All dogs were premedicated with 0.22 mg/kg body weight midazolam intramuscularly, after which anaesthesia was induced with propofol (Group I, 5 bitches and 39 puppies), etomidate (Group II, 5 bitches and 25 puppies), thiopental (Group III, 5 bitches and 26 puppies) and epidural anaesthesia (Group IV, 5 bitches and 39 puppies). Anaesthesia was maintained with a semi-closed circuit containing halothane. Rectal temperature, heart rate, respiration rate, haemoglobin oxygen concentration and mean blood pressure were evaluated in the bitches, while heart rate, breathing effort, muscle movements, mucus colour, reflex irritability and haemoglobin oxygen saturation were measured in the puppies. It was

shown that all anaesthetic protocols were adequate for the bitches and only resulted in minimum changes in the measured parameters and found that Group IV was the best procedure for the puppies compared to the inject able anaesthetic protocols.

Rauser *et al.* (2004) studied the effect of epidural administration of lidocaine, fentanyl and their combination on the minimum alveolar concentration of halothane in 40 healthy dogs. Lidocaine, fentanyl, their combination (2 mg/kg of lidocaine and 0.005 mg/kg of fentanyl) and saline (as a control) administered epidurally and dose response to halothane was determined by means of minimum alveolar concentration (MAC). Basic vital parameters, such as heart rate, respiratory rate, saturation of haemoglobin with oxygen and end-tidal partial pressure of CO_2 were recorded and they concluded that epidural administration of the combination of above drugs could reduce the dose of general anaesthetics, which was important in management of critically ill patients.

Simeonova (2004) studied the changes in the parameters of acid-base status and blood gas analysis in three different anaesthesia schemes in dogs. The first groups of animals were subjected to standard inhalation anaesthesia using halothane. Those from the second group received balanced anaesthesia using pancuronium and the third group received epidural lumbosacral anaesthesia using lidocaine. Arterialized capillary blood samples were obtained at immediately before anaesthesia (0 min), during premedication (30 min), during deep anaesthesia (120 min), after recovery (about 140 min) and the next day (24 h). The acid-base and blood gas parameters were determined. They found that development of primary noncompensated

respiratory acidosis and over oxygenation during the deep stages of halothane and balanced anaesthesia

Simeonova *et al.* (2004) evaluated the oxidative effect of three different anaesthetic protocols in dogs 24 mixed breed dogs by measurement of plasma malondialdehyde (MDA) concentrations before, during and after anaesthesia. Group 1 was premedicated with atropine sulfate (0.02 mg/kg, s/c) and acepromazine maleate (0.1 mg/kg, i/m) and anaesthesia was induced with sodium thiopental (10 mg/kg, i/v) and maintained with 2.5 -3.0 vol% halothane. Group 2 received the same premedication and induction drugs but the maintenance of anaesthesia was with fentanyl citrate (0.01 mg/kg, i/v), halothane (0.5 vol %), pancuronium bromide (0.06 mg/kg, i/v) and controlled ventilation. Group 3 received the same premedication followed by lumbosacral epidural anaesthesia using 2% lidocaine (0.3 ml/kg). Group 4 received no drugs but blood samples were collected at the same times as the other 3 groups. Venous blood samples were collected from all animals to determine the concentration of MDA prior to anaesthesia (baseline), at the time of premedication (30 minutes), during the deep stage of anaesthesia (120 minutes), at the end of anaesthesia (140 minutes) and on the next day (24 h). The stated that halothane anaesthesia, was accompanied by statistically increased blood concentrations of MDA at 120 minutes and 24 hours in comparison to baseline and in comparison with the balanced anaesthesia, epidural and control groups.

2.2 Isoflurane anaesthesia

Meyer *et al.* (1984) used isoflurane anaesthesia as an adjunct to hypothermia for open heart surgery in a dog. They used surface cooling and isoflurane anaesthesia (0.75%) and found that after 130 minutes cooling oesophageal and rectal temperatures were 24 and 20°C respectively, blood pressure was 50/20 mm/Hg and heart rate was 45 beats / minute.

Jones and Seymour (1986) used isoflurane as an inhalation anaesthetic agent in 22 dogs and 21 horses undergoing a variety of surgical procedures. The authors documented that the cardiopulmonary changes with isoflurane anaesthesia were similar to those observed with halothane. Rapid changes in the depth of anaesthesia were achieved and recovery from anaesthesia was rapid in both dogs and horses, which was a reflection of the relative insolubility of isoflurane. Recovery from anaesthesia in the horses was particularly smooth and rapid with the animals spending a greater part of their recumbency in the sternal position, as opposed to lateral recumbency before standing in a well coordinated manner.

Jones and Snowdon (1986) in there experiment induced anaesthesia in 4 dogs with thiopental (12 mg/kg) 30 minutes after premedication with acepromazine (0.1 mg/kg, i/m.) and maintained by isoflurane. They found that arterial blood pressure decreased as the concentration of administered isoflurane increased. Isoflurane produced a profound and dose related respiratory depression as measured by the increase in the end tidal carbon dioxide levels. Isoflurane administration did not produce any visible muscle twitching.

Werner (1987) described the properties and use of the isoflurane that, it is fast acting, virtually non-toxic to the kidneys and liver, has insignificant effects on the heart but as with most inhalation anaesthetics, it lowers blood pressure and depresses inhalation. It is however, expensive and requires a vaporizer. Its use in veterinary practice is feasible if a closed circuit and low-flow delivery are used.

Tranquilli *et al.* (1988) studied the alterations in epinephrine-induced arrhythmogenesis after xylazine and subsequent yohimbine administration in isoflurane anaesthetized dogs. They found that yohimbine possessed a protective action against catecholamine-induced arrhythmias in dogs anaesthetized with isoflurane and xylazine.

Greene *et al.* (1992) quantitative EEG was assessed in six dogs anaesthetized with 1.8% end-tidal isoflurane concentration and following diazepam (0.2 mg/kg i/v) administration. They concluded that quantitative EEG provides a relatively non-invasive, objective measure of diazepam and flumazenil induced changes in cortical activity during isoflurane anaesthesia.

Schwartz *et al.* (1992) conducted a study to clarify the relationship between neuromuscular blocking agents (pancuronium) and anaesthetic potency by studying effect of on steady state electroencephalogram (EEG) burst suppression produced by isoflurane in dogs. They noted dogs received pancuronium 0.1 mg./kg showed the percent of the EEG was isoelectric increased from 21 ± 0.099 (mean ± SD) to 35 ± 0.11 ($P \leq 0.01$).

Matthews *et al.* (1995) used the isoflurane as an inhalant anaesthesia in 6 dogs to compare the readings from two Veterinary pulse oxymeters. The probes were placed on the toe and ear at all inspired oxygen concentrations and found that in the two locations from which readings were obtained, the two units performed quite differently.

Stekiel *et al.* (1995) measured the effects of inhaled isoflurane on hypoxemia induced changes in the diameter of small mesenteric veins, sympathetic efferent neural activity, heart rate, and arterial blood pressure. Simultaneous changes in these dependent variables were measured *in situ* in response to 40 seconds periods of sequentially administered 10%, 5%, 2.5%, and 0% inspired oxygen before, during and after either 0.75% or 1.5% vol/vol inhaled isoflurane in alpha-chloralose-anaesthetized rabbits and found that isoflurane inhibited hypoxia-mediated venoconstriction, increases in sympathetic efferent nerve activity, arterial hypertension and bradycardia.

Martinez *et al.* (1996) studied the pharmacokinetics, effects on renal function, and potentiation of atracurium-induced neuromuscular blockage after administration of a high dose of gentamicin in six isoflurane-anaesthetized dogs. They found that pre- and post-treatment values for serum urea nitrogen, serum creatinine, urine Creatinine-to- gamma -glutamyltransferase ratio and other urine analytes were not significantly different.

Martin *et al.* (1997) studied the hemodynamic effects of epidural ketamine in isoflurane anaesthetized dogs. Mean baseline values for heart rate, mean arterial

pressure, mean pulmonary artery pressure, central venous pressure, pulmonary capillary wedge pressure, cardiac index, stroke index, systemic vascular resistance and pulmonary vascular resistance were recorded and concluded that the epidural injection of 2 mg/kg of ketamine is associated with minimal haemodynamic effects during isoflurane anaesthesia in dogs.

Lee *et al.* (1998) studied the respiratory depressant and skeletal muscle relaxant effects of low-dose pancuronium bromide in spontaneously breathing, isoflurane anaesthetized dogs. They recorded the tidal volume, respiratory rate and minute ventilation throughout the study period and serial arterial blood gases were measured at intervals. Eye position scores, based on the degree of ocular rotation from a neutral gaze axis, were assigned by an ophthalmologist. They found that tidal volume and minute ventilation in high dose dogs decreased by 82% from baseline after injection of pancuronium bromide. Tidal volume and minute ventilation in LD dogs decreased 40% and 55%, respectively.

Martinez *et al.* (1998) studied the neuromuscular effects of doxacurium chloride in isoflurane anaesthetized dogs. In there study they recorded the onset of sedation (time from drug administration to maximal depression), duration (time from maximal depression) and recovery time.

Benson *et al.* (2000) studied the effect of medetomidine on the stress response induced by ovariohysterectomy in isoflurane anaesthetized dogs (In 12 healthy adult female laboratory dogs, weighing 16.8 to 25 kg). The blood samples were examined for epinephrine, nor epinephrine, ACTH, cortisol, insulin and glucose. The results

showed that premedication with medetomidine prevented or delayed the stress response induced by ovariohysterectomy in isoflurane anaesthetized dogs.

Hellyer *et al.* (2001) determined the effect of a constant rate infusion of fentanyl on minimum alveolar concentration of isoflurane and to determined the interaction between fentanyl and a benzodiazepine agonist (diazepam) and antagonist (flumazenil) in isoflurane-anaesthetized dogs and stated that fentanyl markedly decreased isoflurane MAC in dogs. Diazepam, but not flumazenil, further decreased isoflurane fentanyl MAC.

Guochang Hu *et al.* (2002) observed that isoflurane protects myocardium during ischemia reperfusion via a mechanism involving the adenosine triphosphate-sensitive potassium channels and reported that isoflurane inhibited neutrophil-endothelium interactions and the inflammatory response *in vitro* via a pathway independent of the adenosine triphosphate-sensitive potassium channels. This action could be involved in the cardio protection by isoflurane observed *in vivo*.

Muir and Wiese (2004) compared the effects of lactated ringer's solution (LRS) with those of a physiologically balanced 6% hetastarch plasma expander administered to 12 isoflurane anaesthetized dogs by inducing hypotension by blood withdrawal. Hemodynamic variables, pH, blood gas concentrations, PCV, serum electrolyte, total protein concentrations and colloid osmotic pressure (COP) were determined at baseline. They found that hemodynamic variables decreased after blood withdrawal but returned to baseline values more rapidly after infusion with a smaller volume of hetastarch solution when compared with the response by LRS infusion.

Whereas PCV and serum total protein concentration decreased after administration of either solution, COP decreased only after administration of LRS.

Sawyer *et al.* (2004) conducted a study to evaluate the performance of a veterinary oscillometric noninvasive blood pressure monitor in isoflurane anaesthetized dogs. They made an assessment to determine how closely indirect measurements were associated with direct measurements. Six mongrel dogs were anaesthetized with thiopental and maintained with isoflurane. They concluded that noninvasive blood pressure measurements with an oscillometric monitor provided an excellent means of detecting arterial hypotension in anaesthetized dogs.

Tobata *et al.* (2004) studied the effects of dopamine, dobutamine, amrinone and milrinone on regional blood flow in isoflurane anaesthetized dogs. In their study, they found that dopamine increased blood flow in ventricular myocardium, induced the increase in blood flow in the intestine and kidney at a low to middle dose. Dobutamine induced the highest increase in blood flow in ventricular myocardium and skeletal muscle at middle and high doses, amrinone and milrinone increased blood flow in ventricular myocardium almost same with catecholamines and milrinone decreased vascular resistance moderately in most other organs.

Boscan *et al.* (2005) characterized the effects of ketamine administration on the cardiovascular and respiratory systems and on acid-base balance and recorded the adverse effects of ketamine in isoflurane anaesthetized dogs (six healthy dogs). Cardiovascular, respiratory, acid-base variables, body temperature and urine production were recorded before and during noxious stimulation. Cardiac index,

stroke index, rate-pressure product, systemic vascular resistance index, pulmonary vascular resistance index, left ventricular stroke work index, right ventricular stroke work index, arterial oxygen concentration, mixed-venous oxygen concentration, oxygen delivery, oxygen consumption, oxygen extraction ratio, alveolar-arterial oxygen partial pressure gradient and venous admixture were calculated. They found that at the higher plasma ketamine concentrations, adverse effects such as spontaneous movement and profuse salivation. Myoclonus and dysphoria were observed during recovery in most dogs.

Chanoit *et al.* (2005) studied the hemodynamic effects of marbofloxacin (MBF) in isoflurane anaesthetized six dogs. They induced the anaesthesia with sodium thiopental and maintained with isoflurane. Cardiovascular variables were monitored throughout anaesthesia. Marbofloxacin was administered by an i/v bolus at 2 mg/kg, followed 10 minutes later by an infusion at a rate of 40 mg/kg/h for 30 minutes (total dose, 20 mg/kg). They found significant changes during infusion when a cumulative dose of 12 mg/kg had been given. They observed that maximal decreases at the end of the infusion were 16% in heart rate, 26% in systolic left ventricular pressure, 33% in systolic aortic pressure, 38% in diastolic aortic pressure, 29% in cardiac output and 12% in QT interval. All dogs recovered rapidly from anaesthesia at the end of the experiment.

Hideo Hashiguchi *et al.* (2005) studied the pharmacological preconditioning effect of isoflurane against ischemia in the heart brain and kidney in rats. They found that in the isoflurane preconditioning group (1.5% isoflurane for 20 min before renal

ischemia) serum creatinine (1.2 ± 0.7 and 1.1 ± 0.2 mg/dL) and blood urea nitrogen (99 ± 29 and 187 ± 31 mg/dL) were significantly smaller at 24 and 48 hours after reperfusion than in the non-preconditioning group (creatinine, 2.4 ± 1.2 and 2.9 ± 0.9 mg/dL, urea, 62 ± 19 and 79 ± 20 mg/dL).

Nahed-Saleh *et al.* (2005) studied the renal effects of the selective alpha 2-adrenoceptor agonist medetomidine in isoflurane anaesthetized dogs. Urine and blood samples were collected before and at 30, 60, 90 and 120 min following medetomidine injection. Mean arterial blood pressure, renal blood flow, glomerular filtration rate, urine volume (Uv), urine osmolality (Uosm), free water clearance, fractional clearance of sodium (FNa), plasma osmolality (Posm), plasma glucose levels and plasma antidiuretic hormone concentrations were measured. They obtained results showing that increased FNa and Posm and decreased Uosm. Plasma glucose levels initially increased and subsequently decreased.

Pypendop and Ilkiw (2005) determined the pharmacokinetics of ketamine and norketamine in six isoflurane anaesthetized dogs. They obtained results and stated that in isoflurane anaesthetized dogs, among individuals a high variability of disposition of ketamine exist.

Solano *et al.* (2006) determined the effect of intravenous administration of ketamine on the minimum alveolar concentration of isoflurane in six anaesthetized dogs. They found that salivation, regurgitation, mydriasis, increased body temperature and spontaneous movements were some of the adverse effects associated with the high plasma ketamine concentrations.

Andreas *et al.* (2006) studied the effect of isoflurane in neonatal rodents and recorded the arterial blood pressure, heart rate, blood gases and glucose in 10-day-old mice during 60 min of isoflurane anaesthesia with spontaneous or mechanical ventilation, as well as during 60 min of hypoxia-ischemia with isoflurane anaesthesia or without anaesthesia. They found that during isoflurane anaesthesia, hypoglycemia and metabolic acidosis occurred with spontaneous and mechanical ventilation.

Ward *et al.* (2006) conducted study in seven captive male African wild dogs (*Lycaon pictus*) and stated that an inject able medetomidine-ketamine-atropine combination with maintenance by gaseous isoflurane and oxygen provides an inexpensive, reliable anaesthetic for captive African wild dogs.

Rosati *et al.* (2007) evaluated the dose-related cardiovascular and urine output (UrO) effects of dopamine hydrochloride and dobutamine hydrochloride, administered individually and in combination in dogs during deep isoflurane anaesthesia. They concluded that in isoflurane anaesthetized dogs, a guideline dose for dopamine of 7 mug/kg/min is can be administered safely.

Sousa *et al.* (2007) studied the effects of isoflurane on Tei-index of myocardial performance in healthy dogs. They studied the effects of 1.0 MAC isoflurane anaesthesia on the pre ejection period (PEP), left ventricular ejection time (LVET), PEP/LVET ratio, isovolumic relaxation time (IVRT), stroke index (SI), cardiac index (CI), heart rate (HR) and the Tei-index in healthy unpremedicated dogs. They observed significant increases in PEP, PEP/LVET ratio, IVRT and TEI. The LVET

and HR did not change significantly, whereas the SI and CI decreased during anaesthesia. They concluded that isoflurane produced direct effects on the Tei-index.

Brandes *et al.* (2007) sub anaesthetic concentrations of isoflurane strongly depressed canine IHMNs *in vivo*. The neuronal response to 5-HT was also depressed by isoflurane, suggesting that anaesthetic activation of leak K channels, which is expected to result in a larger 5-HT response, was not a dominant mechanism in this depression.

2.3 Inhalant anaesthesia alone or combination and comparative studies

Klide (1976) studied the cardiopulmonary effects of isoflurane and enflurane inhalant anaesthetics in non-sedated, previously instrumented and awake dogs. They observed that enflurane depressed cardiopulmonary function to a greater extent than isoflurane and the depression of cardiopulmonary function from both agents increased with increasing depth of anaesthesia and enflurane produced muscle twitching, but isoflurane did not.

Steffey and Howland (1978) studied the potency of enflurane in dogs in comparison with halothane and isoflurane. Circulatory and respiratory responses were graded by increasing alveolar concentrations of enflurane in unpremedicated healthy dogs during conditions of spontaneous and controlled ventilation. They obtained the data indicates that equipotent concentrations of enflurane are at least as depressant to the cardiopulmonary system as halothane and isoflurane.

Steffey *et al.* (1983) studied the accuracy of isoflurane delivery by halothane specific vaporizers and observed that in comparison with concentrations of halothane, slightly higher concentrations of isoflurane were delivered from halothane vaporizers.

Bednarski *et al.* (1984) made a cost comparison of anaesthetic regimens in the dog and cat in various combinations of sedatives, tranquillizers and antimuscarinics (preanaesthetic drugs) and anaesthetic induction and maintenance drugs. They found that the combination of acetylpromazine-thiamylal-halothane was the least expensive regimen for both dog and cat, whereas drug combinations that included isoflurane as the maintenance drug were the most expensive.

Hellebrekers (1986) conducted a comparative study between isoflurane and halothane as inhalation anaesthetics in the dog and stated that there was no difference between the two anaesthetics in speed of induction. Both halothane as well as isoflurane caused the blood pressure to fall during maintenance anaesthesia. Increase in heart rate and decrease respiration rate with isoflurane, which also resulted in more severe respiratory acidosis than halothane. Recovery was quickest with isoflurane.

Muir and Hubbell (1988) determined the cardiopulmonary and chemical restraining effects of racemic ketamine and its enantiomers to dogs. They observed that the dogs recovering from isoflurane anaesthesia showed transient decrease in arterial blood pressure, left ventricular contractility, cardiac output and total peripheral vascular resistance. Arterial pH and the PO_2 values decreased after i/v administration of racemic ketamine or its enentiomers.

Zbinden *et al.* (1988) studied the uptake and elimination of halothane and isoflurane using a closed-loop anaesthetic system. Haemodynamic and respiratory variables were recorded and the anaesthetic partial pressures were measured in the inspired and expired air, as well as in the arterial, cerebrovenous and mixed venous blood. They concluded that the rate of uptake of isoflurane is more rapid than that of halothane from the alveolar space to the blood, but not from the blood to brain tissue. The rates of elimination from brain tissue and from blood were found to be similar for both agents.

Elizabeth *et al.* (1988) studied the renal function and pathological changes in 27 dogs undergoing pyometra operation maintained under halothane or isoflurane anaesthesia. They evaluated the complete blood count, PCV, serum values of creatinine, BUN, albumin, globulin, sodium, potassium, calcium, phosphorus, glucose, ALP and ALT. They found that the mean PCV was 38 ± 6 %, the mean RBC count was 5.5 ± 0.86 (10^6 cells/cumm), the mean platelet count was 159,070 ± 86,940 cells/mm ^{3}, the mean serum sodium value was 142.6 ± 5.1 mEq /lit, the mean serum potassium value was 4.2 ± 0.6 mEq/lit, the mean ALP value was 178.7 ± 163.5 IU/lit and the mean serum creatinine value was 0.69 ± 0.2 mg/dl.

Katoh and Ikeda (1994) conducted a study to assess the effect of sevoflurane on lung resistance and compliance, and its responsiveness to histamine in eight dogs in comparison with the effect of sevoflurane, isoflurane, enflurane and halothane on bronchoconstriction caused by histamine. Histamine (2, 4, and 8 μg/kg) were administered i/v and the values of pulmonary resistance (RL) and dynamic pulmonary

compliance (Cdyn) at the time of peak effect were recorded. They found that under 1 or 2 MAC anaesthesia, sevoflurane as well as the other three anaesthetics had no bronchoactive effects. All four anaesthetics, including sevoflurane, demonstrated inhibitory effect on increases in RL and decreases in Cdyn caused by histamine. They concluded that sevoflurane was less potent than halothane in attenuating changes in RL and Cdyn in response to i/v histamine administration.

Sloan *et al.* (1996) carried comparative study for sevoflurane and in terms of induction (loss of eyelash reflex, coughing and heart rate) and recovery characteristics (Times to eye opening, pain scores) with single-breath inhaled inductions of anaesthesia in humans. They found that during isoflurane induction time patients showed coughing, increased heart rate and during recovery, patients who received isoflurane had a lower pain scores.

Mutoh *et al.* (1997) evaluated cardiopulmonary effects of sevoflurane (Sevo), compared with halothane (Hal), enflurane (Enf), and isoflurane (Iso). He stated that cardiovascular effects of Sevo were greater than those of Hal, similar to those of Iso and less than those of Enf. Respiratory effects of Sevo were similar to those of Iso at all anaesthesia stages.

Tacke *et al.* (1998) in there study used sevoflurane in dogs that were tested and compared with isoflurane and halothane anaesthesia. They found that there was no significant depression of the cardiovascular system, kidney or hepatotoxic side effects after sevoflurane, isoflurane and halothane anaesthesia.

Neath *et al.* (2000) used isoflurane as maintenance anaesthesia to study the hemodynamic effects of intrathecal oxytocin in normal dogs. They recorded the baseline values of the mean for heart rate, mean arterial pressure, central venous pressure, cardiac output, systemic vascular resistance, mean pulmonary arterial pressure, pulmonary arterial occlusion pressure and pulmonary vascular resistance. They concluded that the intrathecal injection of 1.6 µg/kg of oxytocin is associated with minimal haemodynamic effects during isoflurane anaesthesia.

Mutoh *et al.* (2001) conducted study in dogs to characterize respiratory reflexes elicited by nasal administration of sevoflurane (Sevo), isoflurane (Iso) or halothane (Hal) in anaesthetized dogs and concluded that nasal administration of Sevo at concentrations generally used for mask induction of anaesthesia induced milder reflex inhibition of breathing, presumably via afferent neurons in the nasal passages, than that of Iso or Hal. Respiratory reflexes attributable to stimulation of the nasal passages may contribute to speed of onset and could promote a smoother induction with Sevo, compared with Iso or Hal.

Gerding *et al.* (2002) conducted a study to determine the onset and duration of pancuronium-induced paralysis and effects on intraocular pressure of 27 healthy dogs undergoing elective phacoemulsification surgery. The dogs were premedicated with intramuscular atropine-medetomidine, anaesthesia induced with thiopental and maintained with isoflurane.

Topal *et al.* (2003) studied the effects of halothane, isoflurane and sevoflurane anaesthesia on hepatic function and hepatocellular damage in twenty-one clinically

normal mongrel dogs {the dogs were 1-4 years old, and weighed between 13.5 and 27 kg} by comparing the activity of hepatic enzymes and bilirubin concentration in serum. The animals are divided into three groups and accordingly anaesthetized with halothane, isoflurane and sevoflurane. The Xylazine HCI (1-2 mg/kg) i/m was used as pre-anaesthetic medication. Anaesthesia was induced with propofol 2 mg/kg i/v. venous blood samples were collected before pre-medication, 24 and 48 h, and 7 and 14 days after anaesthesia. They found that in halothane group, serum AST and ALT activities significantly increased all the time after anaesthesia compared with baseline activities. But in the isoflurane group AST and ALT activities increased only between 2 and 7 days and in the sevoflurane group 7 days after anaesthesia and concluded that the use of halothane anaesthesia induces an elevation of liver enzymes activities more frequently than isoflurane or sevoflurane from 2 to 14 days after anaesthesia in dogs and the effects of isoflurane or sevoflurane anaesthesia on the liver in dogs is safer than halothane anaesthesia in dogs.

Pettifer and Hosgood (2004) determined the effect of inhalant anaesthetic and body temperature on peri-anaesthetic serum concentrations of transdermally administered fentanyl in dogs. Each dog received four treatments: isoflurane + normothermia (ISO-NORM), isoflurane + hypothermia (ISO-HYPO), halothane + normothermia (HAL-NORM) and halothane + hypothermia (HAL-HYPO). Direct arterial blood pressures and arterial blood gases were monitored. They found that the mean body temperatures during the anaesthetic period for the four treatments were: ISO-NORM = 37.7 ± 0.07 °C, ISO-HYPO = 35.8 ± 0.1 °C, HAL-NORM = 37.7 ± 0.06 °C, and HAL-HYPO = 35.8 ± 0.13 °C.

Topal *et al.* (2004) studied the effects of halothane, isoflurane and sevoflurane anaesthesia on end tidal CO_2 and arterial oxygen saturation (SpO_2) in dogs. They induced anaesthesia by propofol 2 mg/kg, i/v, maintained with halothane, isoflurane or sevoflurane in oxygen in the concentrations of 1.5, 2.0 and 3.0% respectively. They found that end-tidal CO_2 during halothane anaesthesia was lower than isoflurane and sevoflurane anaesthesia. SpO_2 in dogs of the halothane group was less than the other two groups and concluded that isoflurane or sevoflurane anaesthesia appears to be safer than halothane anaesthesia in dogs.

Denise *et al.* (2005) conducted a comparative evaluation of inhaled halothane, isoflurane, and sevoflurane anaesthetics in dogs during acute normovolemic hemodilution in 18 mongrel dogs, which were divided into three groups to receive either one of anaesthetic treatment. They measured the hemodynamics, oxygen transport and gastric pH before blood withdrawal, at the end of hemodilution, and 30 and 60 min after the end of hemodilution. They found that heart rate was more rapid in the sevoflurane group and thirty minutes after hemodilution, the cardiac index increased 88%, 86%, and 157% in the halothane, isoflurane and sevoflurane group, respectively.

Kilic and Isler (2005) studied the adverse anaesthetic effects in 70 dogs which are randomly divided into 5 equal groups: thiopental (Group 1); ketamine (Group 2); halothane (Group 3); isoflurane (Group 4); and enflurane (Group 5) they recorded that the adverse effect of the anaesthesia in Group 2 on the respiration and haemodynamic

values were determined to be less than those of the other four Groups. The reduction in respiration rate was significantly less in Group 1 than in Groups 3, 4 and 5.

Duke *et al.* (2006) premedicated the dogs undergoing ovariohysterectomy with acepromazine, hydromorphone and glycopyrrolate, anaesthesia was induced with thiopental to study the effect of nitrous oxide on halothane, isoflurane and sevoflurane requirements. They concluded that N_2O reduces halothane, isoflurane and sevoflurane requirements for surgical procedure and cardiovascular stimulation occurred when N_2O was used with isoflurane, less so with sevoflurane and not with halothane.

Lerche and Muir (2006) evaluated the effect of medetomidine on minimum alveolar concentration (MAC), respiratory rate, tidal volume, minute volume and maximum inspiratory occlusion pressure in halothane and isoflurane anaesthetized dogs. They determined the MAC of both inhalants before and 5, 30, and 60 minutes after administration of medetomidine (5µg/kg, i/v) and concluded that equipotent doses of halothane and isoflurane have differing effects on respiration that are most likely attributable to differences in drug effects on central respiratory centers.

Pottie *et al.* (2007) conducted study on healthy adult dogs undergoing desexing surgery which were premedicated with intramuscular acepromazine and morphine and anaesthesia was induced using halothane, isoflurane or sevoflurane delivered by mask or by intravenous propofol and maintained in standard fashion using one of the three inhalant agents. They found that hypothermia is a common complication of general anaesthesia and surgery.

Teixeira Neto *et al.* (2007) compared the cardio respiratory changes induced by equipotent concentrations of halothane (HAL), isoflurane (ISO) and sevoflurane (SEVO) before and after hemorrhage and concluded that inhaled anaesthetics should be used judiciously in animals presented with blood loss. However, if an inhalation agent is to be used under these circumstances, ISO may provide better hemodynamic stability than SEVO or HAL.

Pottie *et al.* (2008) compared the speed and quality of induction of general anaesthesia using three different inhalant agents and one intravenous agent, in healthy dogs undergoing desexing surgery. Less excitable dogs were not premedicated; others were premedicated with intramuscular acepromazine and morphine. Anaesthesia induction protocol was randomly assigned, with halothane, isoflurane or sevoflurane delivered by mask, or propofol delivered intravenously. Maximum vaporiser settings were used for inhalant inductions. Induction of anaesthesia was considered complete at the time of endotracheal intubation. Quality of induction was scored by the administering veterinarian. They found that isoflurane inductions were significantly faster than halothane inductions (2.86 ± 0.25 vs. 3.71 ± 0.22 min; mean ± SE, $P = 0.013$). Sevoflurane inductions (3.29 ±0.24 min) were not significantly different from either halothane (3.71 ± 0.22 min, $P = 0.202$) or isoflurane inductions (2.86 ± 0.25 min, $P = 0.217$). Induction with propofol (1.43 ± 0.13 min) was significantly faster than inhalant induction ($P < 0.001$ in each case). Premedication decreased the dose requirement and time to induction for dogs induced with propofol, but did not significantly change the time to intubation for inhalant inductions. Dogs administered propofol and premedication

were significantly more likely to have an excellent quality of induction, but there was no difference between inhalant agents in terms of induction quality.

Egger *et al.* (2009) conducted a study to determine, if the preanaesthetic administration of ephedrine would prevent anaesthesia-induced hypotension in dogs and cats. In this study they anaesthetized ten cats with acepromazine, butorphanol, ketamine, and isoflurane, and 8 dogs with acepromazine, morphine, propofol and halothane. Cats received ephedrine or saline 10 minutes after premedication. Dogs received ephedrine or saline at the time of premedication. They observed that, no difference in heart rate, respiratory rate, end-tidal CO2, rectal temperature, O2 saturation or cardiac rhythm among treatment groups.

Silva *et al.* (2009) Studied halogenated anaesthetic influence on renal function during hemorrhagic shock and resuscitation. They studied the comparative effects of three halogenated anaesthetics halothane, sevoflurane and isoflurane at equipotent concentrations (1 MAC) on renal responses after resuscitation. The dogs were splenectomized and hemorrhaged to hold mean arterial pressure at 40-50 mm Hg over 45 min and then resuscitated with the shed blood volume. Hemodynamic variables were measured at baseline, after 45 min of hemorrhage, and 15 and 60 min after resuscitation. Renal variables were measured at baseline and 15 and 60 min after resuscitation. They conclude that no difference can be detected between using equipotent doses of halothane, sevoflurane or isoflurane in relation to renal variables in dogs submitted to pressure-adjusted hemorrhagic shock and resuscitation.

III. MATERIALS AND METHODS

The purpose of the present study was to compare the clinical efficacy of halothane and isoflurane inhalant anaesthethetics in clinical cases of dogs undergoing ovariohysterectomy.

This study was conducted on 12 clinical cases of female dogs, which were presented to the Veterinary College Hospital, KVAFSU, Hebbal, Bangalore for ovariohysterectomy. These animals were randomly divided into two groups *Viz.*, Group A (Halothane) and Group B (Isoflurane) comprising of six dogs each.

3.1 Experimental procedure

3.1.1 Preparation of the animals for the experiment

All the animals were clinically examined, body weight were recorded and randomly assigned into one of the two groups. In all the animals solid food and water was withheld for 12 and 6 hours respectively, prior to study. The left flank area was prepared in the routine manner to perform ovariohysterectomy under aseptic conditions.

3.2 Preanaesthetic treatment

3.2.1 Atropine sulphate

Atropine sulphate (1 ml ampule, Indian Research Lab. Pvt. Ltd., West Bengal, 0.65 mg/ml) was administered at a dose rate of 0.04 mg/kg body weight

intramuscularly, 25 minutes prior to induction of inhalation anaesthesia to both the groups (Group A and Group B).

3.2.2 Acepromazine maleate

Acepromazine maleate (Acemav, inj. 50 ml vial, Mavlab, Australia, 10 mg/ml) was administered intramuscularly at the dose rate of 0.03 mg/kg body weight as a sedative, 20 minutes prior to induction of inhalation anaesthesia.

3.2.3 Ampicillin and Cloxacillin

Ampicillin and Cloxacillin (Megapan, 500 mg, inj Vet, Aristo Pharmaceutical Pvt. Ltd., Mumbai) was administered i/m at the dose rate of 22 mg/kg body weight, one hour prior to the induction of inhalation anaesthesia and continued postoperatively for five days (b.i.d).

3.3 Inhalation Anaesthetic agents

3.3.1 Halothane

Halothane (FLUOTHANE®, 250 ml bottle, Nicholas Piramal India Ltd., Madhya Pradesh.) was administered in combination with oxygen both during induction and maintenance of general anaesthesia in Group A animals.

3.3.2 Isoflurane

Isoflurane (FORANE®, 250 ml bottle, Abbott India Ltd., Mumbai) was administered in combination with oxygen both during induction and maintenance of general anaesthesia in Group B animals.

3.4 Induction of inhalant anaesthesia i.e. Halothane (Group A) and Isoflurane (Group B)

The animals were administered with Atropine sulphate and Acepromazine maleate i/m, at 25 minutes and 20 minutes respectively, prior to induction with halothane and animals were not disturbed at least for 15 minutes from the point of administration of atropine sulphate. The time taken for onset of sedation was recorded. Twenty minutes after premedication with Acepromazine maleate, anaesthesia was induced with halothane (for Group A) and isoflurane (for Group B) animals using close fitting face mask. In both groups inhalation anaesthesia was administered along with oxygen (1.5 liters per minute) till animal reaches surgical plane of anaesthesia, which was marked by the absence of pedal reflex, ventromedial deviation of eye ball and deep abdominal breathing with decreased respiratory rate. Surgical plane of anaesthesia was maintained throughout the period of surgery. The decision to increase or decrease the concentration of inhaled anaesthetic agent was made based on standard criteria for assessing depth of anaesthesia. These included heart rate, respiratory rate and pattern, pulse quality, palpebral reflex, jaw tone and response to movement and surgical stimulation. The goal was to achieve a satisfactory level of anaesthesia, which was sufficient to allow the ovariohysterectomy procedure without evidence of conscious response or subconscious movement by the patient. The time required for induction, average maintenance dose volume of inhalant anaesthesia, duration of surgery and recovery parameters were recorded.

3.5 Surgical procedure performed on bitches subjected for inhalation anaesthesia

Ovariohysterectomy was performed in all the animals by following either left or right flank incision method. After attaining surgical plane of anaesthesia, an oblique incision was made through upper flank area. The uterine horns and ovaries were approached through deeper incision via separation of skin, superficial fascia, deep fascia, caudoventrally external oblique muscle, carnioventrally internal oblique muscle, dorsoventrally transverse abdominis muscle and peritoneum. The uterus and ovaries were identified and grasped with thumb forceps. The suspensory ligament of the ovary was ruptured by traction, ovary was fully exteriorized from the abdomen and a large opening was made through broad ligament to expose the ovarian attachment and its blood vessels. Three clamp method was followed for ligature of ovarian pedicle. Using a No. 1-0 chromic catgut (Ethicon, Jhonson & Johnson Ltd, Baddi) a double ligature was applied to ligate the ovarian pedicle. Then the attachment between the ligature and the ovary was severed. After removing one ovary, the other ovary was located and removed in similar manner. The broad ligament was carefully freed from uterine horns with body and the blood vessels were identified. The uterine blood vessels were ligated on each side. A transfixating double ligature was used to encompassing entire uterus and severed just cranial to the transfixation ligature. The parietal layer of the peritoneum and transverse abdominus muscle was sutured by applying continuous pattern sutures using No.1-0 catgut. The internal and external oblique muscle layers were apposed separately by using No.1-0

catgut in simple interrupted suture pattern. Skin incision was closed by horizontal suture pattern with nonabsorbable sutures (polyamide).

3.6 Schedule of blood sample collection

In both the Groups, blood samples were collected at different time intervals through out the study. Whole blood samples collected in EDTA vials was used for complete blood count, blood glucose and total plasma protein estimation. Whereas separate set of serum samples that were stored in deep freezer was used for estimation of biochemical parameters at a later date. In both groups, blood samples were obtained at following time intervals during the study:

Sample No.	Blood sample collection
1	Fifteen minutes prior to preanaesthetic administration (0 minutes)
2	Fifteen minutes after preanaesthetic administration
3	One hour after induction of anaesthesia
4	Six hours after induction of anaesthesia
5	Twenty four hours after induction of anaesthesia
6	Forty eight hours after induction of anaesthesia
7	Ninety six hours after induction of anaesthesia
8	Seven days after induction of anaesthesia

3.7 Different parameters recorded during inhalation anaesthesia in bitches undergoing ovariohysterectomy

3.7.1 Onset of sedation

It was calculated as the time taken for the onset of symptoms like droopy eyelid, head down and attaining recumbency.

3.7.2 Induction of inhalation anaesthesia

It was calculated on the basis of total time required by an animal to get into the plane of surgical anesthesia, which was marked by absence of pedal reflex, ventromedial deviation of eyeball and deep abdominal breathing with decrease respiratory rate.

3.7.3 Average maintenance dose volume of anaesthesia (Vol %)

Average maintenance dose volume percentage (Vol %) of inhalant anaesthetic required to maintain surgical plane of anaesthesia was calculated by monitoring the reappearance of pedal reflex and central upward movement of eyeball from state of ventromedial deviation.

3.7.4 Surgical plane of anaesthesia during ovariohysterectomy procedure

It was marked as the duration of anaesthesia between abolition of pedal reflex and completion of surgical procedure. Completion of surgery was marked as completion of last skin suture.

3.7.5 Parameters recorded during recovery phase of anaesthesia

At the end of surgery, marked by closing of the last skin suture, the vaporizer setting was turned to 'OFF' position and the animals were allowed to breathe fresh oxygen for three additional minutes. Then animals were shifted to recovery room and video recording was performed to study the qualitative (noninteractive) and quantitative recovery parameters. During recovery period, except for blood sampling, dogs were left undisturbed until they were able to stand up and walk on their own. The point at which the animal stood up was the last piece of data that was recorded for qualitative parameters during recovery phase of inhalation anaesthesia.

3.7.5.1 Quantitative parameters recorded during recovery phase of anaesthesia

The average time for return of swallowing reflex, pedal reflex, head righting reflex, time taken for voluntary leg movement, time taken for sternal recumbency and time taken for animal to ambulate after cessation of anaesthesia during recovery period were recorded.

3.7.5.2 Qualitative parameters recorded during recovery phase of anaesthesia

The qualitative parameters were assessed based on videorecordings obtained during recovery phase of anaesthesia.

Start position(starting position of body), Position change(change of body position), End position(last position of body before attaining a recumbency or standing procedure), Head position, Ear position, Eye position, Tail position, Vocal sounds, Others (Arched back, stretching, rigid back, lip licking, draws leg up.)

3.7.6 Postoperative care of the animals

All the animals were subjected for routine postoperative care over the following 18-72 hours after surgery and discharged on 7th postoperative day.

3.8 Clinical parameters

Vital body parameters *Viz.*, rectal temperature (°F), heart rate (per minute) and respiratory rate (per minute) were recorded at 15 minutes prior to premedication (0 minutes, base line sample) and thereafter at every 15 minutes interval upto 60 minutes during recovery period.

3.9 Hematological parameters

The following haematological parameters were estimated using automatic blood cell counter (Sysmex-K800, Japan).

a. Total erythrocyte count (TEC, 10^6/cmm of blood)

b. Packed cell volume (PCV, %)

c. Hemoglobin (Hb, g %)

e. Platelet count (10^6/cumm)

d. Total leukocyte count (TLC, 10^3/cmm. of blood) and Differential leukocyte count (DLC %) were made as per standard procedure described by Coles and Campbell (1986).

3.10 Biochemical parameters

The following biochemical parameters were estimated using the auto analyzer (B.M. Hitachi, 704 Auto Analyzer, Japan).

a. Alanine aminotransferase (ALT, units/liter)

b. Aspertate aminotransferase (AST, units/liter)

c. Alkaline phosphatase (ALP, units/liter)

d. Plasma creatinine (mg/dl)

e. Total plasma protein (TPP, g/dl)

f. Electrolytes (sodium, potassium and chloride, mEq/liter) and Blood glucose (mg/dl) was determined by Ames Glucometer, Gx, USA.

3.11 Statistical analysis

The mean and standard error for all the data were computed. The variations in the clinical and biochemical parameters before, during and after anaesthesia at different time intervals for both within and between treatments were analyzed by single tailed unpaired 't' test. The data interpreted as per procedure described by Snedecor and Cochran (1996). The test of significance was fixed at five per cent for all the comparisons.

IV. RESULTS

A comparative study was conducted to evaluate the clinical efficacy of halothane and isoflurane anaesthesia with reference to clinical, haematological and biochemical parameters in six bitches each undergoing ovariohysterectomy. The results of the study are presented under the following headings:

Different parameters assessed during inhalation anaesthesia in bitches undergoing ovariohysterectomy (Group A and Group B animals)

4.1.1 Onset of sedation

In both Group A and Group B animals, onset of sedation was observed within four minutes after intramuscular administration of acepromazine maleate. In Group A and Group B animals optimum depth of sleep was at 7.04 ± 0.58 and 6.48 ± 0.28 minutes, respectively (Table 2).

4.1.2 Induction of inhalation anaesthesia

The mean duration to attain optimum level of induction for halothane anaesthesia (Group A) was 8.26 ± 1.55 minutes and for isoflurane anaesthesia (Group B) was 3.01 ± 0.16 minutes. In Group A animals halothane induction was smooth but took longer duration to attain surgical plane of anaesthesia. Whereas in Group B animals induction with isoflurane was quicker but animals resisted during initial uptake of isoflurane.

Table 1. Physical parameters recorded in Group A (Halothane) and Group B (Isoflurane) animals subjected for ovariohysterectomy procedure

Sl. No.	Animal No.	Body weight (kg)		Sl. No.	Animal No.	Body weight (kg)
1	A_1	10.0		**1**	B_1	11.0
2	A_2	13.5		**2**	B_2	12.6
3	A_3	12.8		**3**	B_3	10.4
4	A_4	9.67		**4**	B_4	9.8
5	A_5	8.90		**5**	B_5	10.6
6	A_6	7.54		**6**	B_6	11.5

Table 2. Comparative mean of different parameters recorded during induction, maintenance and recovery phases anaesthesia in Group A (Halothane) and Group B (Isoflurane) animals

Sl. No.	Parameters (minutes)	Group A	Group B
1	Time taken for onset of Sedation	7.04 ± 0.58	6.48 ± 0.28
2	Time taken for induction	8.26 ± 1.55	3.01 ± 0.16*
3	Duration of Anaesthesia for surgical procedure	33.68 ± 2.59	32.04 ± 1.59
4	Average maintenance volume of anaesthesia for group (Volume %)	3.34 ± 0.18	2.61 ± 0.25*
5	Time taken for regain of swallowing reflex from the point of termination of anaesthesia	9.53 ± 1.09	3.18 ± 0.43*
6	Time of return of pedal reflex from the point of termination of anaesthesia	15.04 ± 2.56	5.54 ± 0.51*
7	Time of return of head writing reflex from the point of termination of anaesthesia	25.44 ± 5.58	7.32 ± 0.95*
8	Time taken for voluntary leg movement from the point of termination of anaesthesia	17.75 ± 2.86	7.67 ± 1.15*
9	Time taken for sternal recumbency from the point of termination of anaesthesia	27.97 ± 4.91	10.28 ± 1.21*
10	Time taken for animal to attempt to stand from the point of termination of anaesthesia	42.89 ± 4.42	16.94 ± 4.17*

* Significant ($P \leq 0.05$)

4.1.3 Average maintenance dose volume of inhalation anaesthesia (Vol %)

The average maintenance dose volume of halothane (Group A) and isoflurane (Group B) anaesthesia required during ovariohysterectomy procedure were 3.34 ± 0.18 and 2.61 ± 0.25 (Vol %), respectively (Table 2).

4.1.4 Surgical plane of anaesthesia during ovariohysterectomy

During the present study, the average duration of anaesthesia required for completion of ovariohysterectomy procedure in Group A and Group B animals were 33.68 ± 2.59 and 32.04 ± 1.59 minutes, respectively (Table 2). Surgical plane of anaesthesia was accomplished and ascertained by absence of pinna reflex, pedal reflex or anal reflex and presence of ventromedial positioning of eye ball and skeletal muscle relaxation during the course ovariohysterectomy procedure in all the animals. No salivation or vomiting was observed in any of the animals and the entire course of inhalant anaesthetic regimen followed during ovariohysterectomy procedure was uneventful in both the groups (Table 3).

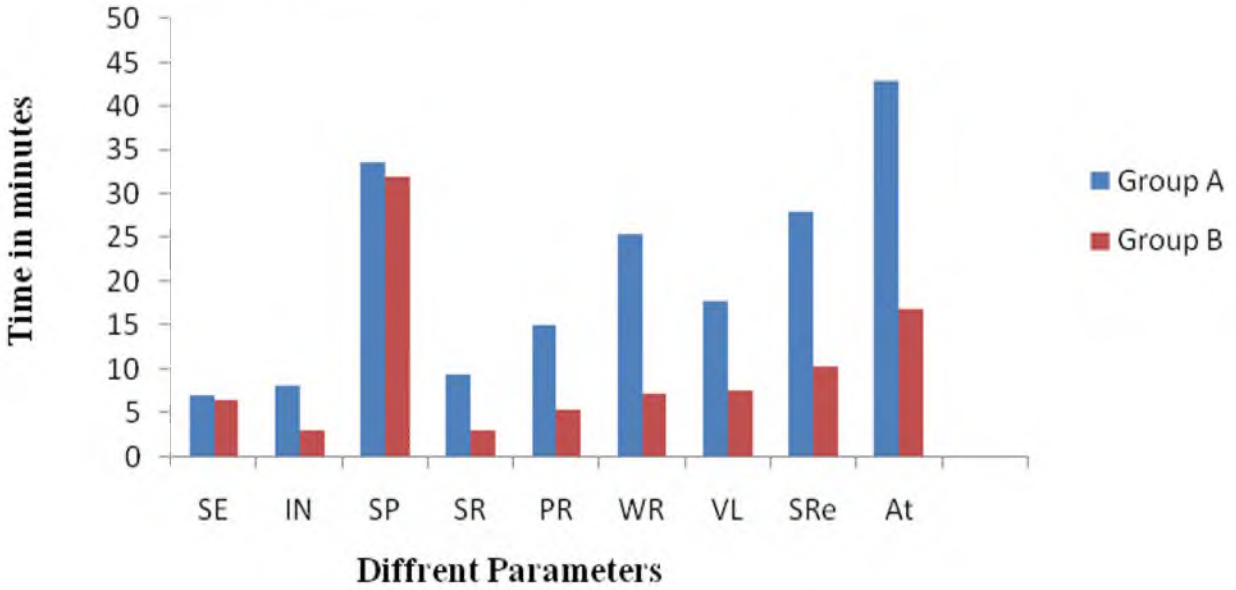
4.1.5 Quantitative parameters observed during recovery phase

In both groups, beginning of recovery phase of anaesthesia was marked from the point of cessation of anaesthesia, which coincides with the last skin suture applied. In Group A animals, the mean duration for return of swallowing reflex, pedal reflex, head righting reflex, voluntary leg movement, attainment of sternal recumbency and duration for animals to ambulate were: 9.53 ± 1.09, 15.04 ± 2.56, 25.44 ± 5.58, 17.75 ± 2.86, 27.97 ± 4.91 and 42.89 ± 4.42 minutes, respectively (Table 2, Fig.1).

Table 3. Various reflexes monitored during inhalation anaesthesia in Group A (Halothane) and Group B (Isoflurane) animals subjected for ovariohysterectomy procedure

Sl. No.	Reflexes	Group A			Group B		
		Present	Sluggish	Absent	Present	Sluggish	Absent
1	Salivation			6 (100%)			6 (100%)
2	Vomition			6 (100%)			6 (100%)
3	Skeletal muscle relaxation a. Good b. Moderate	6 (100%)			6 (100%)		
4	Pedal reflex		1 (16.66%)	5 (83.33%)			6 (100%)
5	Palpabral reflex		2 (33.34%)	4(66.66%)			6 (100%)
6	Position of eyeball a. Ventro medial b. Centre	5 (83.33%) 1(16.66%)			6 (100%)		
7	Pupil Status	6 (100%) Slightly dilated			6 (100%) Slightly dilated		
8	Pinna pinch reflex			6 (100%)			6 (100%)
9	Anal pinch reflex			6 (100%)			6 (100%)
10	Cutaneous analgesia		1(16.66%)	5(83.33%)			6 (100%)

Fig.1. Comparative mean of different parameters during sedation, induction and recovery phase of anaesthesia in Group A (Halothane) and Group B (Isoflurane) animals



SE → Time of onset of Sedation
IN → Induction time
SP → Time for surgical procedure
SR →Regain of swallowing reflex
PR →Time of return of pedal reflex from point of termination of anaesthesia
WR →Time of return of head writing reflex from point of termination of anaesthesia
VL →Time taken for voluntary leg movement from point of termination of anaesthesia
SRe → Time taken for Sternal recumbency from point of termination of anaesthesia
At → Time taken for animal to attempt to stand from point of termination of anaesthesia

In Group B animals, the mean duration for return of swallowing reflex, pedal reflex, head righting reflex, voluntary leg movement, attainment of sternal recumbency and duration for animals to ambulate were: 3.18 ± 0.43, 5.54 ± 0.51, 7.32 ± 0.95, 7.67 ± 1.15, 10.28 ± 1.21 and 16.94 ± 4.17 minutes, respectively. The above values were significantly lesser when compared to the mean values (duration) of halothane anaesthetized (Group A) animals (Table 2, Fig. 1).

4.1.6 Qualitative parameters recorded during recovery phase

Based on the interactive behavioral attributes, qualitative parameters were assessed as per the procedure described under the subheading 3.7.5.2.

In Group A, the animals use to sleep more, the position change was not so frequent may be due to residual effect of halothane in blood. During state of recovery the head position was lowered in all the animals. 50% of animals were alert and watchful at any movements (personnel) surrounding recovery room. Transition from anaesthesia to full consciousness is not smooth. Moderate paddling or other unconscious movements were seen all along recovery phase. Vocalisation was evident and most the animals had difficulty positioning themselves. In this group lip licking, licking of floor, legs and bandage were predominantly observed.

In Group B animals, recovery phase was relatively a smooth transition from anaesthesia to the state of full consciousness. No struggling, paddling, tremors or other unconscious movements observed. Moderate attempts of lip licking, licking of floor, legs and bandage were evident but at lesser frequency compared to the halothane group (Table 4).

Table 4. Qualitative parameters recorded during inhalation anesthesia in Group A (Halothane) and Group B (Isoflurane) animals subjected for ovariohysterectomy procedure

Behaviour	Start position	Position change	End position	Head position	Ear position	Eye position	Tail position	Vocal	Others *
Group A									
A1	Lateral recumbency	No	Stand	Level	Stand upright	Watch	Low	Yes	Lip licking, draws leg up
A2	Lateral recumbency	Slightly	Stand	Lowered	Stand upright	Watch	Low	Yes	Licking floor
A3	Lateral recumbency	Rarely	Stand	Lowered	Back	Drowsy	Surface	Yes	Hurdling closely
A4	Lateral recumbency	No	Stand	Lowered	Flat to side	Watch	Surface	Yes	Biting bed material
A5	Lateral recumbency	No	Stand	Lowered	Flat to side	Drowsy	Low	Yes	Licking legs
A6	Lateral recumbency	Rarely	Stand	Lowered	Back	Drowsy	Low	yes	Licking bandage
Group B									
B1	Lateral recumbency	Yes	Stand	level	Stand upright	Watch	Low	No	draws leg up
B2	Lateral recumbency	Yes	Stand	Lowered	Stand upright	Drowsy	Low	Yes	Urinated
B3	Lateral recumbency	Yes	Stand	level	Stand upright	Stare ahead	Low	No	Licking bandage
B4	Lateral recumbency	Yes	Stand	level	Flat to side	Watch	Low	No	draws leg up
B5	Lateral recumbency	Yes	Stand	level	Stand upright back	Watch	Low	No	Licking bandage
B6	Lateral recumbency	Yes	Stand	Lowered	Stand upright	Watch	Low	No	draws leg up

*Arched back, stretching, rigid back, lip licking, draws leg up.

4.1.7 Rectal Temperature (°F)

In Group A animals, the mean rectal temperature prior to preanaesthetic administration (from the point of acepromazine maleate administration) was 101.7 ± 0.48 ^{0}F. Fifteen minutes after preanaesthetic administration the mean rectal temperature was 101.7 ± 0.46 ^{0}F. During the course of halothane anaesthesia (maintenance and recovery period) the mean rectal temperature was 99.68 ± 0.33 ^{0}F, 99.18 ± 0.50 ^{0}F, 98.78 ± 0.46 and 98.85 ± 0.27 ^{0}C at 15 minutes, 30 minutes, 45 minutes and 60 minutes, respectively, which was significantly decreased from the base values (Table 5 and Fig. 2).

In Group B animals the mean rectal temperature prior to preanaesthetic administration (from the point of acepromazine maleate administration) was 101.9 ± 0.46 ^{0}F. Fifteen minutes after preanaesthetic administration the mean rectal temperature was 101.5 ± 0.42 ^{0}F. During the course of isoflurane anaesthesia (maintenance and recovery period) the mean rectal temperature was 100.3 ± 0.48 ^{0}F, 99.80 ± 0.53 ^{0}F, 99.48 ± 0.56 ^{0}F and 99.10 ± 0.26 ^{0} F at 15 minutes, 30 minutes, 45 minutes and 60 minutes, respectively, which was significantly decreased from the base values (Table 5 and Fig. 2).

4.1.8 Respiratory rate (Per minute)

In Group A animals the mean respiratory rate prior to preanaesthetic administration was 24.00 ± 2.38 per minute and at 15 minutes the mean respiratory rate was dropped to 21.17 ± 1.74 per minute. During the course of anaesthetic period

Table 5. Comparative results of clinical parameters recorded in Group A (Halothane) and Group B (Isoflurane) animals at different intervals (mean ± SE)

Parameters	Time interval					
	Before and after preanaesthesia		After induction			
	0 min.	15 min.	15 min.	30min.	45 min.	60 min.
Temperature (^{0}F)						
Group A	101.7 ± 0.48 ^{ax}	101.7 ± 0.46 ^{ax}	99.68 ± 0.33 ^{bx}	99.18 ± 0.50^{bx}	98.78 ± 0.46^{bx}	98.85 ± 0.27^{bx}
Group B	101.9 ± 0.46 ^{ax}	101.5 ± 0.42 ^{ax}	100.3 ± 0.48^{bx}	99.80 ± 0.53^{bx}	99.48 ± 0.56 ^{bx}	99.10 ± 0.26 ^{bx}
Respiratory rate (per min.)						
Group A	24.00 ± 2.38 ^{ax}	21.17 ± 1.74 ^{ax}	22.67 ± 1.92 ^{ax}	18.83 ± 2.04 ^{ax}	17.17 ± 1.42^{bx}	17.50 ± 1.58^{bx}
Group B	24.50 ± 3.07 ^{ax}	21.83 ± 2.99 ^{ax}	19.33 ± 1.70 ^{ax}	20.67 ± 2.21 ^{ax}	17.50 ± 1.25 ^{ax}	18.00 ± 1.26 ^{ax}
Heart rate (per min.)						
Group A	109.7 ± 9.98^{ax}	94.17 ± 11.30 ^{ax}	95.33 ± 11.76 ^{ax}	84.33 ± 11.16 ^{ax}	87.33 ± 8.17 ^{ax}	92.33 ± 9.80 ^{ax}
Group B	114.7 ± 8.14^{ax}	97.67 ± 9.75 ^{ax}	103.3 ± 8.34 ^{ax}	94.00 ± 7.52 ^{ax}	93.00 ± 6.03 ^{ax}	103.2 ± 7.37 ^{ax}

Means bearing any one common superscript either in rows or in columns don not differ significantly ($P \leq 0.05$)
Superscript in columns a & b, Superscript in rows x & y.

Fig. 2. Comparative mean of Temperature (°F) recorded in Group A (Halothane) and Group B (Isoflurane) animals

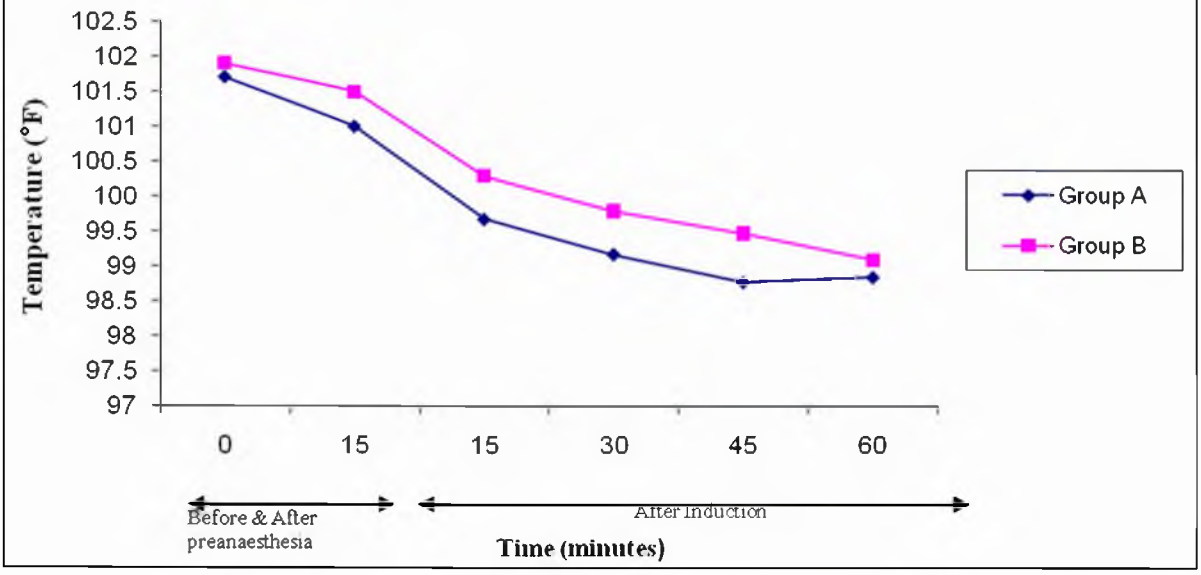
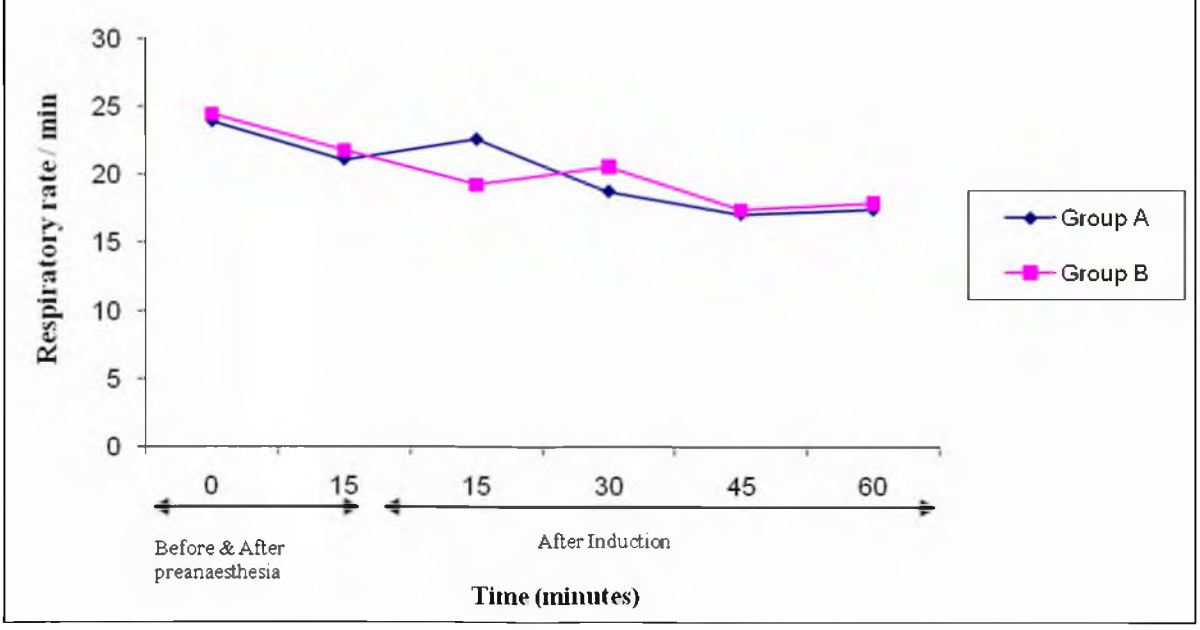


Fig. 3. Comparative mean of Respiratory rate (per minute) in Group A (Halothane) and Group B (Isoflurane) animals



(maintenance and recovery phase) the mean respiratory rate was 22.67 ± 1.92, 18.83 ± 2.04, 17.17 ± 1.42 and 17.5 ± 1.58 per minute at 15 minutes, 30 minutes, 45 minutes and 60 minutes, respectively. The mean respiratory rate observed at 45 and 60 minute interval following halothane induction was significantly lower than that of base value (Table 5 and Fig. 3).

In Group B animals the mean respiratory rate prior to preanaesthetic administration (from the point of acepromazine maleate administration) was 24.50±3.07/ min. Fifteen minutes after preanaesthetic administration the mean respiratory rate was 21.83±2.99/ min. During the course of isoflurane anaesthesia (maintenance and recovery period) the mean respiratory rate was 19.33 ± 1.70, 20.67 ± 2.21, 17.50 ± 1.25 and 18.00 ± 1.26/ min at 15 minutes, 30 minutes, 45 minutes and 60 minutes, respectively, which were non-significantly decreased from the base values (Table 5 and Fig. 3).

4.1.9 Heart rate (Per minute)

In Group A animals the mean heart rate prior to preanaesthetic administration was 109.7 ± 19.98 per minute and at 15 minutes interval the mean heart rate was dropped to 94.17 ± 11.30 per minute. During the course of anaesthetic period (maintenance and recovery phase) the mean the heart rate 95.33 ± 11.76, 84.33 ± 11.16, 87.33 ± 8.17 and 92.33 ± 9.80, at 15 minutes, 30 minutes, 45 minutes and 60 minutes, respectively (Table 5 and Fig. 4).

In Group B animals the mean heart rate prior to preanaesthetic administration was 114.7 ± 18.14 per minute and dropped to 97.67 ± 9.75 per minute at 15 minute interval. During the course of anaesthetic period (maintenance and recovery phase) the mean the heart rate 103.3 ± 8.34, 94.00 ± 7.52, 93.00 ± 6.03 and 103.2 ± 7.37, at 15 minutes, 30 minutes, 45 minutes and 60 minutes, respectively.

In both Groups the mean heart rate decreased non-significantly from basal mean values (Table 5 and Fig. 4).

4.2 Haematological parameters

4.2.1 Total erythrocyte count (TEC)

In Group A animals the mean TEC prior administrating preanaesthetic was 6.04 ± 0.4 10^6/cmm of blood and 15 minutes after preanaesthetic administration the mean TEC levels was dropped to 5.20 ± 0.38 million/cmm. One hour after induction of halothane anaesthesia the mean TEC level was decreased non-significantly 5.38 ± 0.30 10^6/cmm. At the end of 6, 24, 48, 96 and 168 hour (7 days) the mean TEC levels were: 5.68 ± 0.27, 6.07 ± 0.26, 6.38 ± 0.49, 6.55 ± 0.42 and 6.20 ± 0.40 10^6/cmm, respectively. None of the above values were significantly different (Table 6 and Fig. 5).

The mean TEC prior administrating preanaesthetic in isoflurane anaesthesia was 6.40 ± 0.31 10^6/cmm of blood and 15 minutes after preanaesthetic administration the mean TEC levels was dropped to 5.38 ± 0.36 10^6/cmm were the latter values was insignificant. One hour after induction of isoflurane anaesthesia the mean TEC level

Table 6. Comparative results of haematological parameters recorded in Group A (Halothane) and Group B (Isoflurane) animals at different intervals (mean ± SE)

Parameters	Time interval							
	Before and after preanaesthsia		After induction					
	0 min.	15 min.	1 hr.	6 hr	24 hr.	48hr.	96 hr.	168hr.(7 days)
TEC (10^6/cmm)								
Group A	6.04 ± 0.40 ^{ax}	5.20 ± 0.38 ^{ax}	5.38 ± 0.30 ^{ax}	5.68 ± 0.27 ^{ax}	6.07 ± 0.26 ^{ax}	6.38 ± 0.49 ^{ax}	6.55 ± 0.42 ^{ax}	6.20 ± 0.40 ^{ax}
Group B	6.40 ± 0.31 ^{ax}	5.38 ± 0.36 ^{ax}	5.80 ± 0.32 ^{ax}	5.97 ± 0.31 ^{ax}	6.19 ± 0.34 ^{ax}	6.48 ± 0.55 ^{ax}	6.67 ± 0.38 ^{ax}	6.53 ± 0.40 ^{ax}
PCV (%)								
Group A	36.37 ± 1.75 ^{ax}	34.75 ± 1.30 ^{ax}	36.65 ± 1.68 ^{ax}	36.30 ± 0.65 ^{ax}	36.97 ± 0.94 ^{ax}	36.85 ± 1.53 ^{ax}	39.95 ± 1.87 ^{ax}	36.53 ± 2.57 ^{ax}
Group B	40.15 ± 2.26 ^{ax}	37.63 ± 2.07 ^{ax}	39.07 ± 2.55 ^{ax}	38.68 ± 2.24 ^{ax}	40.33 ± 2.96 ^{ax}	41.95 ± 2.81 ^{ax}	43.15 ± 2.21 ^{ax}	38.48 ± 3.03 ^{ax}
Hb (g/dl)								
Group A	13.47 ± 0.98 ^{ax}	13.02 ± 1.20 ^{ax}	12.38 ± 0.99 ^{ax}	12.64 ± 0.87 ^{ax}	12.80 ± 0.18 ^{ax}	12.78 ± 0.40 ^{ax}	13.43 ± 0.71 ^{ax}	12.62 ± 0.71 ^{ax}
Group B	13.45 ± 0.61 ^{ax}	12.23 ± 1.09 ^{ax}	12.18 ± 0.68 ^{ax}	12.33 ± 0.79 ^{ax}	13.07 ± 0.62 ^{ax}	13.57 ± 0.79 ^{ax}	13.72 ± 0.80 ^{ax}	13.17 ± 1.02 ^{ax}
TLC (10^3/cmm)								
Group A	13.58 ± 3.22 ^{ax}	14.27 ± 2.72 ^{ax}	10.90 ± 1.49 ^{ax}	21.42 ± 4.98 ^{ax}	22.12 ± 4.34 ^{ax}	20.60 ± 4.15 ^{ax}	16.88 ± 1.81 ^{ax}	11.98 ± 0.77 ^{ax}
Group B	17.62 ± 2.24 ^{ax}	15.77 ± 1.85 ^{ax}	15.03 ± 1.70 ^{ax}	22.92 ± 2.90 ^{ax}	17.68 ± 2.27 ^{ax}	15.43 ± 0.97 ^{ax}	15.30 ± 1.70 ^{ax}	11.77 ± 0.83 ^{ax}
DLC%								
Neutrophil								
Group A	75.67 ± 8.29 ^{ax}	74.83 ± 7.79 ^{ax}	71.83 ± 8.57 ^{ax}	78.00 ± 8.56 ^{ax}	80.83 ± 6.56 ^{ax}	80.33 ± 3.85 ^{ax}	84.00 ± 1.06 ^{ax}	81.33 ± 0.91 ^{ax}
Group B	84.67 ± 0.66 ^{ax}	83.00 ± 1.00 ^{ax}	81.67 ± 1.38 ^{ax}	85.33 ± 1.96 ^{ax}	84.67 ± 1.83 ^{ax}	82.17 ± 1.32 ^{ax}	83.33 ± 0.49 ^{ax}	80.83 ± 1.24 ^{ax}
Lymphocyte								
Group A	23.50 ± 7.77 ^{ax}	22.67 ± 7.51 ^{ax}	23.00 ± 8.52 ^{ax}	23.33 ± 9.06 ^{ax}	19.17 ± 9.01 ^{ax}	12.83 ± 0.74 ^{ax}	15.83 ± 0.70 ^{ax}	15.00 ± 1.46 ^{ax}
Group B	14.50 ± 1.33 ^{ax}	13.83 ± 0.79 ^{ax}	14.67 ± 1.40 ^{ax}	11.67 ± 1.38 ^{ax}	12.00 ± 1.48 ^{ax}	12.83 ± 0.70 ^{ax}	15.17 ± 0.70 ^{ax}	16.00 ± 1.15 ^{ax}
Eosinophil								
Group A	2.33±0.21 ^{ax}	1.33±0.21 ^{ax}	1.66±0.21 ^{ax}	1.66±0.21 ^{ax}	1.33±0.21 ^{ax}	1.00±0.36 ^{ax}	1.83±0.30 ^{ax}	1.33±0.12 ^{ax}
Group B	2.16 ± 0.30 ^{ax}	2.00 ± 0.31 ^{ax}	2.00 ± 0.36 ^{ax}	1.83 ± 0.16 ^{ax}	2.50 ± 0.95 ^{ax}	1.80 ± 0.20 ^{ax}	4.00 ± 2.40 ^{ax}	2.20 ± 0.58 ^{ax}
Monocyte								
Group A	1.16±0.30 ^{ax}	1.50±0.34 ^{ax}	1.00±0.01 ^{ax}	1.16±0.30 ^{ax}	0.83±0.40 ^{ax}	0.33±0.21 ^{ax}	1.83±0.30 ^{ax}	1.16±0.30 ^{ax}
Group B	1.00±0.25 ^{ax}	1.50±0.34 ^{ax}	1.16±0.30 ^{ax}	1.16±0.16 ^{ax}	1.66±0.68 ^{ax}	1.00±0.36 ^{ax}	1.33±0.42 ^{ax}	1.16±0.30 ^{ax}
Basophil								
Group A	1.00±0.25 ^{ax}	1.00±0.44 ^{ax}	0.66±0.33 ^{ax}	1.00±0.36 ^{ax}	0.66±0.33 ^{ax}	0.66±0.33 ^{ax}	1.33±0.33 ^{ax}	1.16±0.16 ^{ax}
Group B	0.16±0.16 ^{ax}	1.66±0.42 ^{ax}	0.66±0.42 ^{ax}	0.66±0.16 ^{ax}	0.66±0.42 ^{ax}	0.33±0.21 ^{ax}	1.66±0.33 ^{ax}	1.50±0.22 ^{ax}
TPP (g/dl)								
Group A	7.26 ± 0.07 ^{ax}	7.71 ± 0.37 ^{ax}	7.23 ± 0.21 ^{ax}	6.93 ± 0.26 ^{ax}	7.13 ± 0.29 ^{ax}	7.68 ± 0.30 ^{ax}	7.51 ± 0.20 ^{ax}	7.31 ± 0.08 ^{ax}
Group B	6.70 ± 0.29 ^{ax}	6.94 ± 0.56 ^{ax}	6.44± 0.37 ^{ax}	6.42 ± 0.37 ^{ax}	6.60 ± 0.40 ^{ax}	7.14 ± 0.49 ^{ax}	7.18 ± 0.36 ^{ax}	6.63 ± 0.41 ^{ax}

Means bearing any one common superscript either in rows or in columns do not differ significantly ($P \leq 0.05$)
Superscript in columns a & b, Superscript in rows x & y.

Fig. 4. Comparative mean value of Heart rate per minute in Group A (Halothane) and Group B (Isoflurane) animals

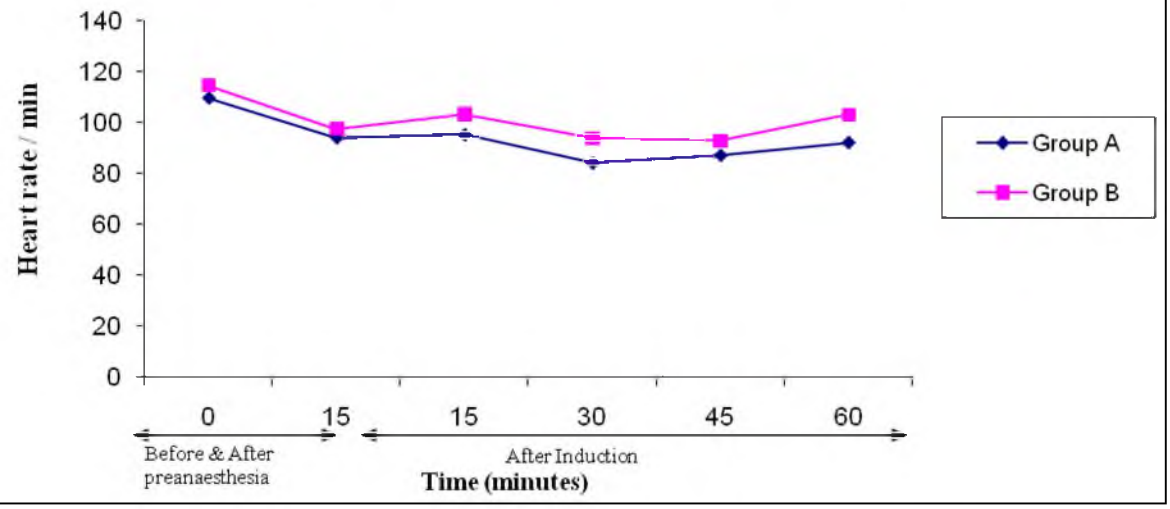
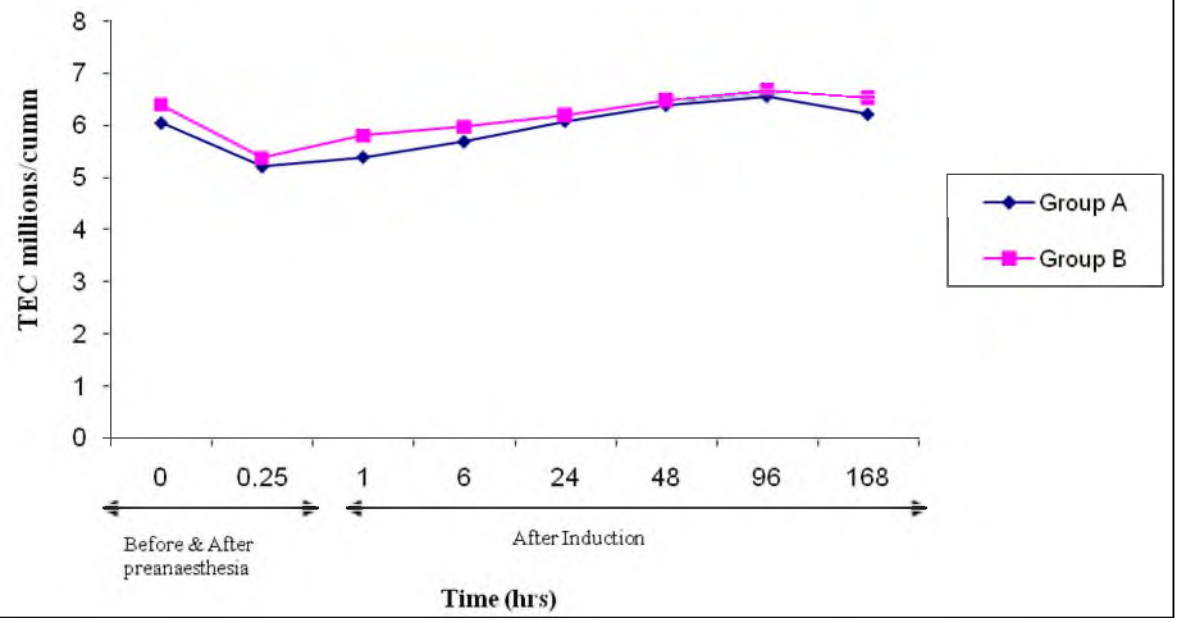


Fig. 5. Comparative mean of Total erythrocyte count in Group A (Halothane) and Group B (Isoflurane) animals



was decreased non-significantly 5.80 ± 0.32 10^6/cmm. At the end of 6, 24, 48, 96 and 168 hour (7 days) the mean TEC levels were: 5.97 ± 0.31, 6.19 ± 0.34, 6.48 ± 0.55, 6.67 ± 0.38 and 6.53 ± 0.40 10^6/cmm, respectively, which were insignificant (Table 6 and Fig. 5).

4.2.2 Packed cell volume (PCV)

The mean PCV was 36.37 ± 1.75 percent prior to preanaesthetic administration and at 15 minutes interval PCV level was dropped to 34.75 ± 1.30 percent. Following halothane induction the mean PCV levels returned to basal level were: 36.65 ± 1.68, 36.30 ± 0.65, 36.97 ± 0.94, 36.85 ± 1.53, 39.95 ± 1.87 and 36.53±2.57 percent at the end of 1 hour, 6 , 24 , 48 , 96 and 168 hour (7 days), respectively. None of the PCV values were significant from basal PCV levels (Table 6 and Fig. 6).

In Group B animals the mean PCV was 40.15 ± 2.26 percent prior to preanaesthetic administration and at 15 minutes interval PCV level was dropped to 37.63 ± 2.07 percent. Following isoflurane induction the mean PCV levels were: 39.07 ± 2.55, 38.68 ± 2.24, 40.33 ± 2.96, 41.95 ± 2.81, 43.15 ± 2.21 and 38.48 ± 3.03 percent at the end of 1hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. None of the PCV values were significant from basal PCV levels (Table 6 and Fig. 6).

4.2.3 Haemoglobin (Hb %)

In Group A animals, prior to preanaesthetic administration the mean Hb per cent was 13.47 ± 0.98 and after preanaesthetic administration the mean Hb level decreased to 13.02 ± 1.20. Following halothane induction the mean Hb levels were:

Fig. 6. Comparative mean of Packed cell volume (%) in Group A (Halothane) and Group B (Isoflurane) animals

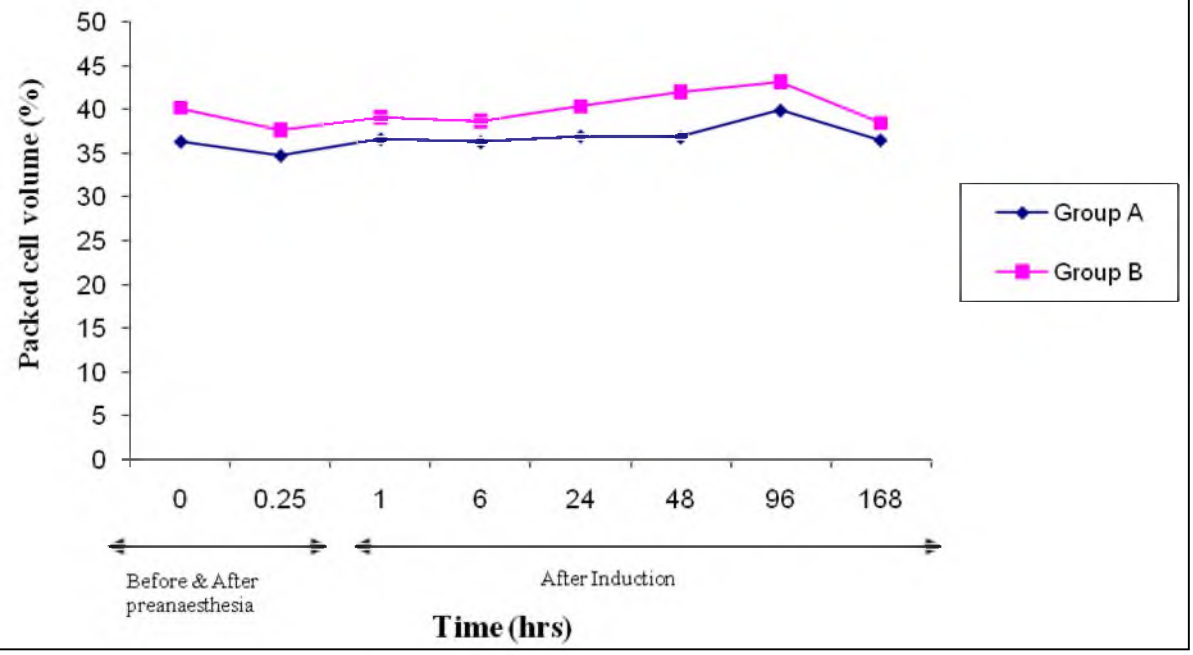
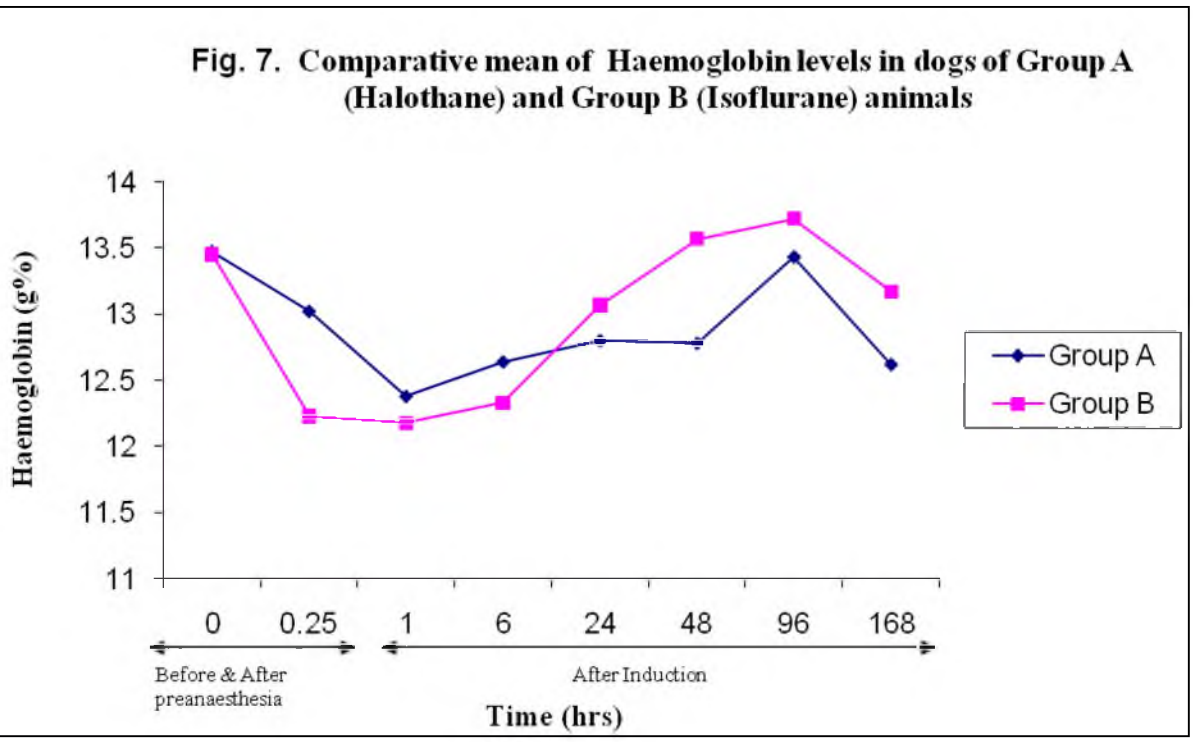


Fig. 7. Comparative mean of Haemoglobin levels in dogs of Group A (Halothane) and Group B (Isoflurane) animals



12.38 ± 0.99, 12.64 ± 0.87, 12.80 ± 0.18, 12.78 ± 0.40, 13.43 ± 0.71 and 12.62 ± 071 % at 1hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. The mean Hb values were decreased non-significantly from basal Hb values (Table 6 and Fig.7).

In Group B animals, prior to preanaesthetic administration the mean Hb per cent was 13.45 ± 0.61 and after preanaesthetic administration the mean Hb level was decreased to 12.23 ± 1.09. Following isoflurane induction the mean Hb levels were: 12.18 ± 0.68, 12.33 ± 0.79, 13.07 ± 0.62, 13.57 ± 0.79, 13.72 ± 0.80 and 13.17 ± 1.02 % at 1hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. The Hb values were decreased non-significantly from basal Hb values except at 48 and 96th hrs there was a non-significant increase in mean Hb level. (Table 6 and Fig. 7).

4.2.4 Total leukocyte count (TLC)

In Group A animals the mean TLC prior to the preanaesthetic administration was 13.58 ± 3.22 10^3/cmm of blood and after preanaesthetic administration the mean TLC level was increased to 14.27 ± 2.72 10^3/cmm. After halothane induction the mean TLC levels were: 10.90 ± 1.49, 21.42 ± 4.98, 22.12 ± 4.34, 20.60 ± 4.15, 16.88 ± 1.81 and 11.98 ± 0.77 10^3/cmm 1hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. There was no significant change in the TLC values observed throughout the period of study in Group A animals (Table 6 and Fig. 8).

In Group B animals, the mean TLC prior to the preanaesthetic administration was 17.62 ± 2.24 10^3/cmm of blood and after preanaesthetic administration the mean TLC level was decreased to 15.77 ± 1.85 10^3/cmm. After isoflurane induction the

Fig. 8. Comparative mean of Total leucocyte count in Group A (Halothane) and Group B (Isoflurane) animals

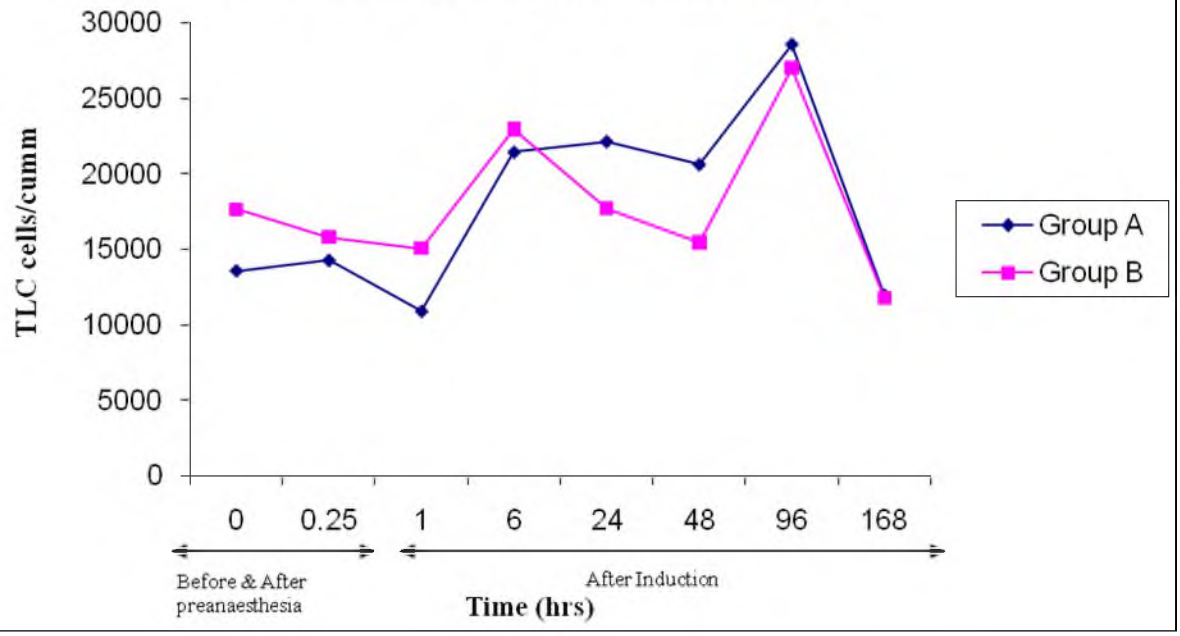
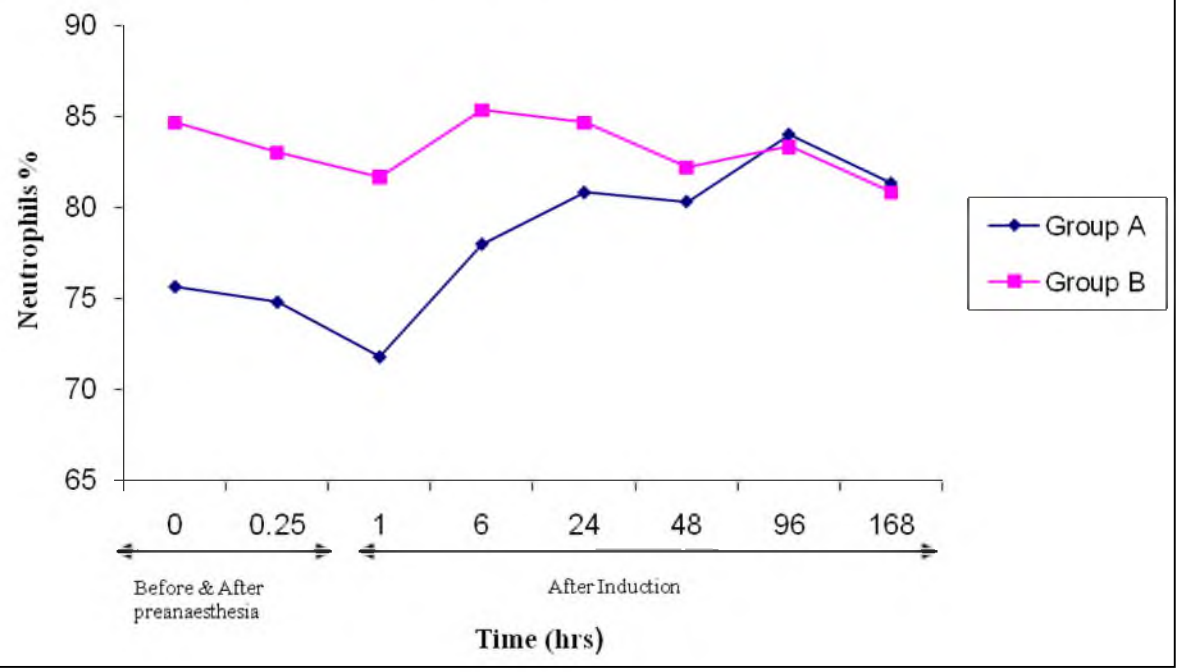


Fig. 9. Comparative mean of Neutrophil count in Group A (Halothane) and Group B (Isoflurane) animals



mean TLC levels were: 15.03 ± 170, 22.92 ± 2.90, 17.68 ± 2.27, 15.43 ± 0.97, 15.30 ± 1.70 and 11.77 ± 0.83 10^3/cmm 1hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. There was non-significant decrease in the TLC values observed throughout the period of study in Group B animals except transitory increase at 24th hr (Table 6 and Fig. 8).

4.2.5 Differential leukocyte count (DLC)

In Group A animals, prior to preanaesthetic administration the mean per cent of neutrophil, lymphocyte, eosinophil, monocyte and basophil were: 75.67 ± 8.29, 23.50 ± 7.77, 2.33 ± 0.21, 1.16 ± 0.30 and 1.00 ± 0.25 per cent, respectively. Following halothane induction, during the period of observation between 1 hour and 96 hours, there were no significant changes observed in mean values of neutrophil, lymphocyte, eosinophil, monocyte and basophile count. At the end of 168 hours the mean values of neutrophil, lymphocyte, eosinophil, monocyte and basophil count were 81.33 ± 0.91, 15.00 ± 1.46, 1.33 ± 0.12, 1.16 ± 0.30 and 1.16 ± 0.16 per cent, respectively. None of the DLC values were significant but transient increase and decrease within normal limit was observed (Table 6 and Fig. 9, 10, 11, 12 and 13).

In Group B animals, prior to preanaesthetic administration the mean value of neutrophil, lymphocyte, eosinophil, monocyte and basophil were: 84.67 ± 0.66, 14.50 ± 1.33, 2.16 ± 030, 1.00 ± 0.25 and 0.16 ± 0.16 percent, respectively. Following isoflurane induction, during the period of observation between 1 hour and 96 hours, there were no significant changes observed in mean values of neutrophil, lymphocyte, eosinophil, monocyte and basophile count. At the end of 168(7 days) hours the mean

Fig. 10. Comparative mean of Lymphocyte count (%) in Group A (Halothane) and Group B (Isoflurane) animals

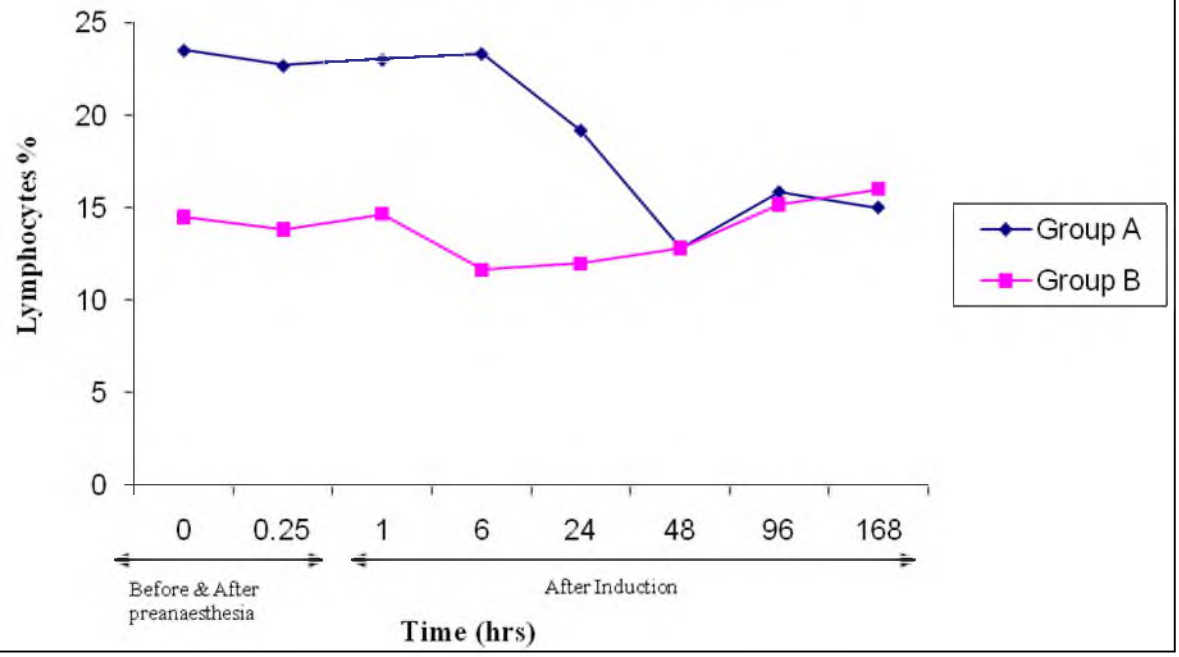


Fig. 11. Comparative mean of Eosinophil count in Group A (Halothane) and Group B (Isoflurane) animals

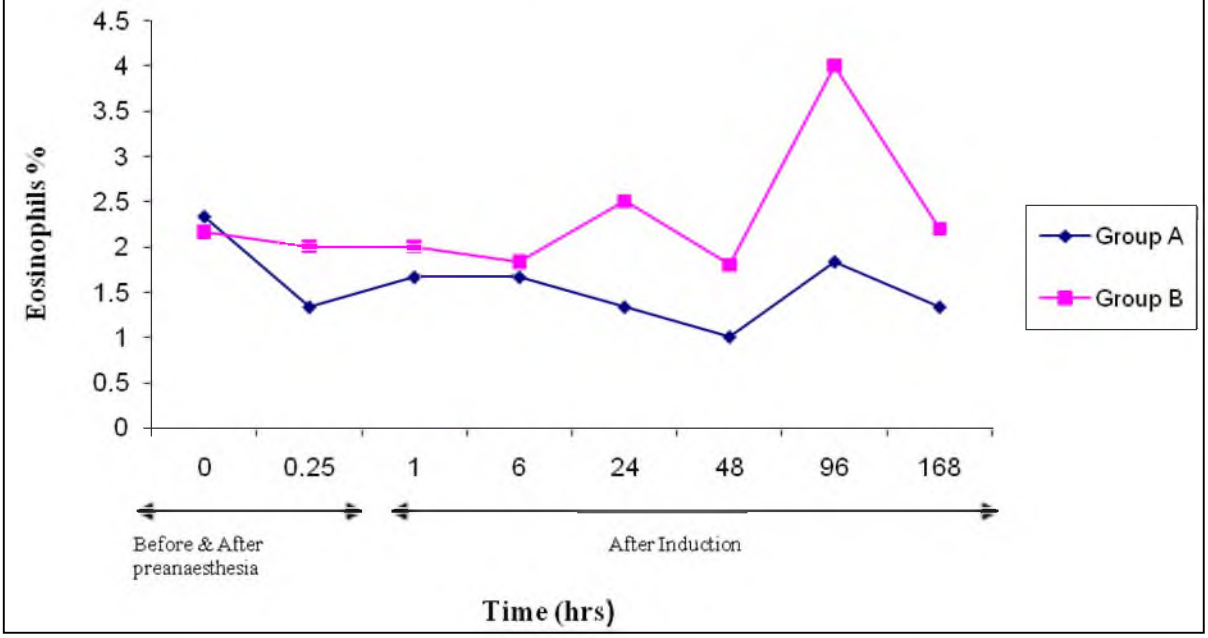


Fig. 12. Comparative mean of Monocyte count in Group A (Halothane) and Group B (Isoflurane) animals

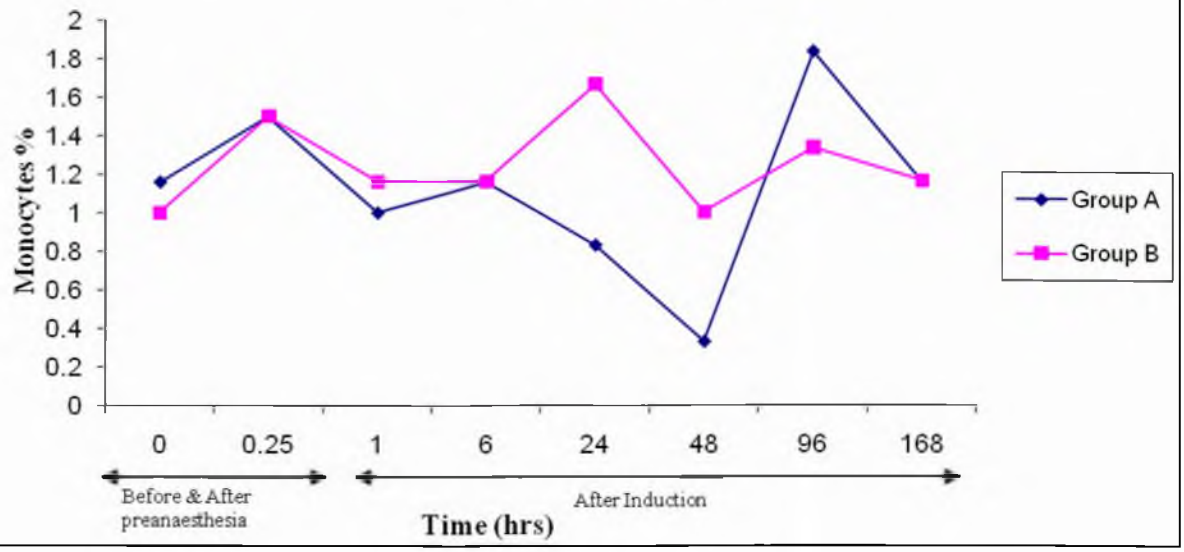
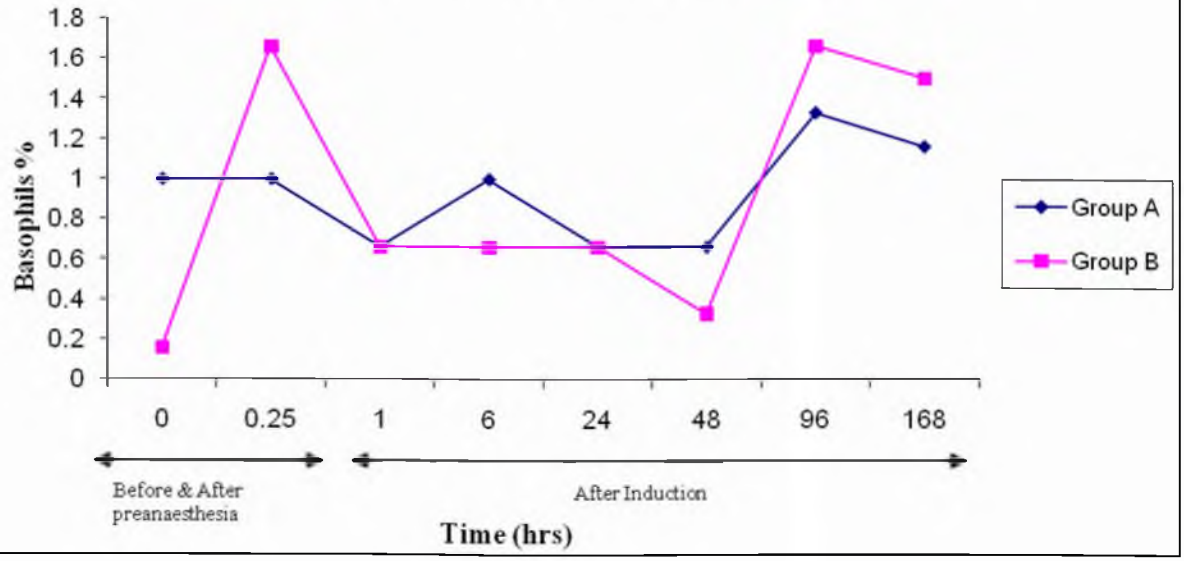


Fig. 13. Comparative mean of Basophil count in Group A (Halothane) and Group B (Isoflurane) animals



values of neutrophil, lymphocyte, eosinophil, monocyte and basophil count were 80.83 ± 0.49, 16.00 ± 1.15, 2.20 ± 0.58, 1.16 ± 0.30 and 1.50 ± 0.22 percent, respectively. None of the DLC values were significant but transient increase and decrease within normal limit was observed (Table 6 and Fig. 9, 10, 11, 12 and 13).

4.2.6 Platelet (PLT) count

In Group A animals, the mean Platelet count was 3.95±0.69 10^6/cumm prior to preanaesthetic administration and after preanaesthetic administration the mean PLT count was decreased to 2.47 ± 0.39. Following halothane induction and observation period the mean PLT count decreased non-significantly were: 2.77 ± 0.50, 2.89 ± 0.59, 3.33 ± 0.76, 3.33 ± 0.60, 3.55 ± 0.67 and 3.12 ± 0.67 10^6/cumm at 1 hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. None of the mean PLT count values were significant from mean basal PLT values (Table 7 and Fig. 14).

In Group B animals, the mean PLT count was 3.74 ± 0.72 10^6/cumm prior to preanaesthetic administration and after preanaesthetic administration the mean platelet count was decreased to 2.65 ± 0.38 10^6/cumm. Following isoflurane induction and during the course observation period the mean platelet count was: 3.27 ± 0.51, 3.29 ± 0.55, 3.81 ± 0.64, 3.52 ± 0.40, 3.60 ± 0.51 and 3.15 ± 0.67 10^6/cumm at 1 hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. The mean platelet count values were decreased non-significantly from mean basal PLT values except at 24^{th} hr there was a transient non-significant increase in PLT value was observed (Table 7 and Fig. 14).

4.3 Biochemical parameters

4.3.1 Alanine aminotransferase (ALT)

In Group A animals, the mean ALT level prior to the preanaesthetic administration was 40.00 ± 3.64 and after 15 minutes the mean ALT level was 43.17 ± 2.73 units/liter. After induction of halothane the mean ALT levels were: 42.17 ± 1.60, 43.67 ± 2.60, 40.83 ± 4.57, 50.0 ± 1.57, 52.0 ± 3.30 and 48.0 ± 4.13 at 1 hour, 6, 24, 48, 96 and 168(7 days), respectively. The mean ALT levels at 96 and 168th hour (7 days), respectively were increased significantly from basal ALT levels (Table 7 and Fig. 15).

In Group B animals, the mean ALT level prior to the preanaesthetic administration was 33.33 ± 3.80 and after 15 minutes the mean ALT level was 37.50 ± 4.49 units/liter. After induction of isoflurane the mean ALT level were: 36.17 ± 4.07, 37.67 ± 4.34, 36.83 ± 4.65, 41.67 ± 3.95, 41.67 ± 5.01 and 42.00 ± 5.16 at 1 hour, 6, 24, 48, 96 and 168(7 days), respectively. The mean ALT levels increased non-significantly within normal values after induction with isoflurane throughout the period of observation in study (Table 7 and Fig. 15).

4.3.2 Aspartate aminotransferase (AST)

In Group A animals, the mean AST level prior to administration of preanaesthetic and 15 minutes after preanaesthetic administration was 56.17 ± 4.22 and 65.33 ± 12.31 units/liter. After induction with halothane anaesthesia the mean AST levels were: 59.17 ± 8.53, 65.67 ± 11.29 and 78.0 ± 19.83units/liter at the end of

Table 7. Comparative biochemical parameters recorded in Group A (Halothane) and Group B (Isoflurane) animals at different intervals (mean ± SE)

Parameters	Time interval							
	Before and after preanaesthesia		After induction					
	0 min.	15 min.	1 hr.	6 hr	24 hr.	48hr.	96 hr.	168hr.(7 days)
Plt (lakhs/ cumm)								
Group A	3.95 ±0.69 ^{ax}	2.47 ± 0.39 ^{ax}	2.77 ± 0.50 ^{ax}	2.89 ± 0.59 ^{ax}	3.33 ± 0.76 ^{ax}	3.33 ± 0.60 ^{ax}	3.55 ± 0.67 ^{ax}	3.12 ± 0.67 ^{ax}
Group B	3.74 ± 0.72 ^{ax}	2.65 ± 0.38 ^{ax}	3.27 ± 0.51 ^{ax}	3.29 ± 0.55 ^{ax}	3.81 ± 0.64 ^{ax}	3.52 ± 0.40 ^{ax}	3.60 ± 0.51 ^{ax}	3.15 ± 0.67 ^{ax}
Blood Glucose (mg/dl)								
Group A	89.77 ± 11.01 ^{ax}	89.77 ± 6.05 ^{ax}	125.1 ± 12.89 ^{ax}	100.0 ± 9.22 ^{ax}	93.75 ± 9.84 ^{ax}	91.13 ± 10.94 ^{ax}	92.38 ± 8.36 ^{ax}	105.21 ± 7.97 ^{ax}
Group B	88.20 ± 11.28 ^{ax}	89.29 ± 6.01 ^{ax}	126.5 ± 12.62 ^{ax}	97.60 ± 9.80 ^{ax}	87.25 ±10.1 ^{ax}	83.55± 9.28 ^{ax}	85.03± 7.26 ^{ax}	97.00 ± 8.90 ^{ax}
ALT(SGPT) (IU/lit)								
Group A	40.00 ± 3.64 ^{ax}	43.17 ± 2.73 ^{ax}	42.17 ± 1.60 ^{ax}	43.67 ± 2.60 ^{ax}	40.83 ± 4.57 ^{ax}	50.00 ± 1.57 ^{ax}	52.00 ±3.30 ^{bx}	48.00 ±4.13 ^{bx}
Group B	33.33 ± 3.80 ^{ax}	37.50 ± 4.49 ^{ax}	36.17 ±4.07 ^{ax}	37.67 ± 4.34 ^{ax}	36.83 ± 4.65 ^{ax}	41.67 ± 3.95 ^{ax}	41.67 ± 5.01 ^{ax}	42.00 ± 5.16 ^{ax}
AST (SGOT) (IU/lit)								
Group A	56.17 ± 4.22 ^{ax}	65.33 ± 12.31 ^{ax}	59.17 ± 8.53 ^{ax}	65.67 ± 11.29 ^{ax}	78.00 ± 19.83 ^{ax}	87.83 ± 12.48 ^{bx}	96.17 ± 8.95 ^{bx}	85.83 ± 10.75 ^{bx}
Group B	47.33 ± 4.66 ^{ax}	62.50 ± 13.04 ^{ax}	61.17 ± 8.48 ^{ax}	73.33 ± 8.67 ^{bx}	78.83 ± 19.32 ^{ax}	85.17 ± 13.97 ^{bx}	87.67 ± 12.90 ^{bx}	91.00 ± 10.21 ^{bx}
ALP(IU/lit)								
Group A	112.7 ± 12.71 ^{ax}	112.3 ± 13.77 ^{ax}	186.3 ± 75.28 ^{ax}	221.7 ± 97.85 ^{ax}	288.8 ± 120.9 ^{ax}	147.2 ± 6.24 ^{ax}	129.0 ± 8.26 ^{bx}	145.7 ± 4.70 ^{bx}
Group B	116.8 ± 6.51 ^{ax}	108.8 ± 7.37 ^{ax}	103.2 ± 6.15 ^{ax}	112.0 ± 10.54 ^{ax}	135.8 ± 14.34 ^{ax}	118.5 ± 10.21 ^{ay}	112.7 ± 8.61 ^{ax}	117.0 ± 13.67 ^{ax}
Creatinine (mg/dl)								
Group A	0.94 ± 0.06 ^{ax}	0.92 ± 0.04 ^{ax}	1.04 ± 0.12 ^{ax}	0.96 ± 0.06 ^{ax}	1.13 ± 0.12 ^{ax}	1.18 ± 0.08 ^{ax}	1.11± 0.13 ^{ax}	1.07± 0.11 ^{ax}
Group B	0.97±0.05 ^{ax}	0.95 ± 0.02 ^{ax}	0.96 ± 0.08 ^{ax}	0.91 ± 0.07 ^{ax}	0.95 ± 0.11 ^{ax}	1.04 ± 0.05 ^{ax}	1.11 ± 0.12 ^{ax}	0.97 ± 0.06 ^{ax}
Electrolytes Sodium (mEq/lit)								
Group A	140.0 ± 1.18 ^{ax}	142.5 ± 1.17 ^{ax}	142.1 ± 1.66 ^{ax}	142.1 ± 2.08 ^{ax}	140.6 ± 1.31 ^{ax}	143.8 ± 1.97 ^{ax}	144.5 ± 1.00 ^{bx}	144.7 ± 1.99 ^{ax}
Group B	141.8 ± 1.21 ^{ax}	142.9 ± 0.82 ^{ax}	143.6 ± 1.57 ^{ax}	143.1 ± 1.61 ^{ax}	138.9 ± 2.56 ^{ax}	146.0 ± 1.10 ^{bx}	144.1 ± 1.02 ^{ax}	144.7 ± 2.01 ^{ax}
Potassium(mEq/lit)								
Group A	4.11 ± 0.10 ^{ax}	4.26 ± 0.18 ^{ax}	4.20 ± 0.18 ^{ax}	4.40 ± 0.17 ^{ax}	4.33 ± 0.26 ^{ax}	4.49 ± 0.13 ^{ax}	4.73 ± 0.12 ^{bx}	4.53± 0.10 ^{bx}
Group B	4.15 ± 0.15 ^{ax}	4.55 ± 0.12 ^{ax}	4.38 ± 0.10 ^{ax}	4.40 ± 0.08 ^{ax}	4.43 ± 0.13 ^{ax}	4.29 ± 0.14 ^{ax}	4.51 ± 0.11 ^{ax}	4.96 ± 0.19 ^{bx}
Chloride (mEq/lit)								
Group A	104.3 ± 1.83 ^{ax}	106.9 ± 2.10 ^{ax}	110.3 ± 2.57 ^{ax}	110.7 ± 3.30 ^{ax}	102.7 ± 0.99 ^{ax}	105.0 ± 1.87 ^{ax}	104.2 ± 1.59 ^{ax}	103.8 ± 2.30 ^{ax}
Group B	107.6 ± 2.24 ^{ax}	108.4 ± 1.79 ^{ax}	111.0 ± 2.11 ^{ax}	109.2 ± 1.35 ^{ax}	103.4 ± 2.54 ^{ax}	107.4 ± 1.84 ^{ax}	106.0 ± 1.74 ^{ax}	107.5 ± 2.68 ^{ax}

Means bearing any one common superscript either in rows or in columns do not differ significantly ($P \leq 0.05$)
Superscript in columns a & b, Superscript in rows x & y.

Fig. 14. Comparative mean of Platelet count in Group A (Halothane) and Group B (Isoflurane)animals

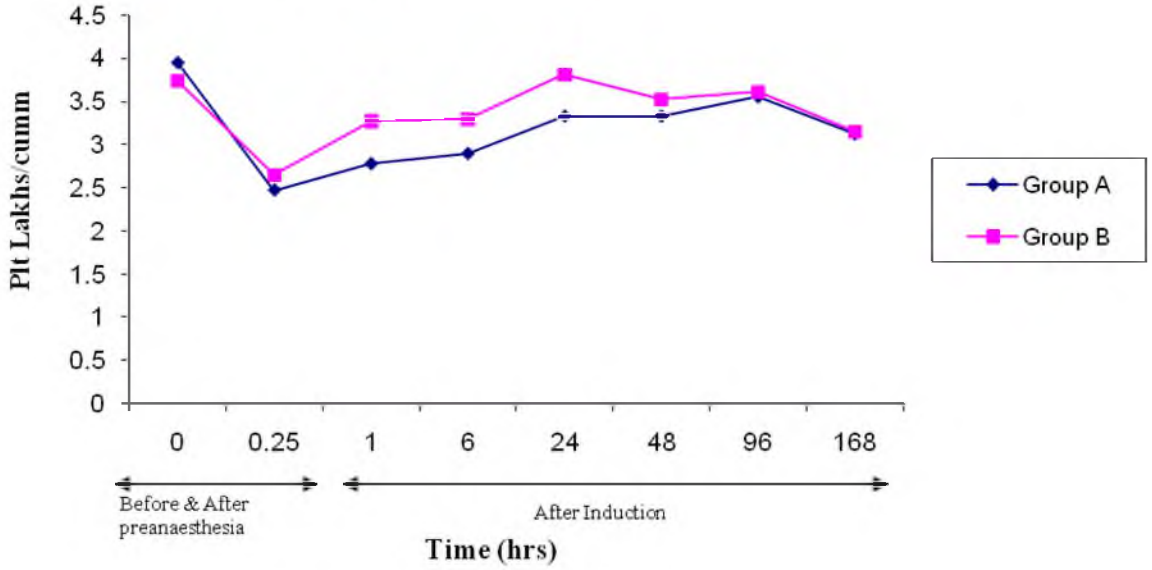
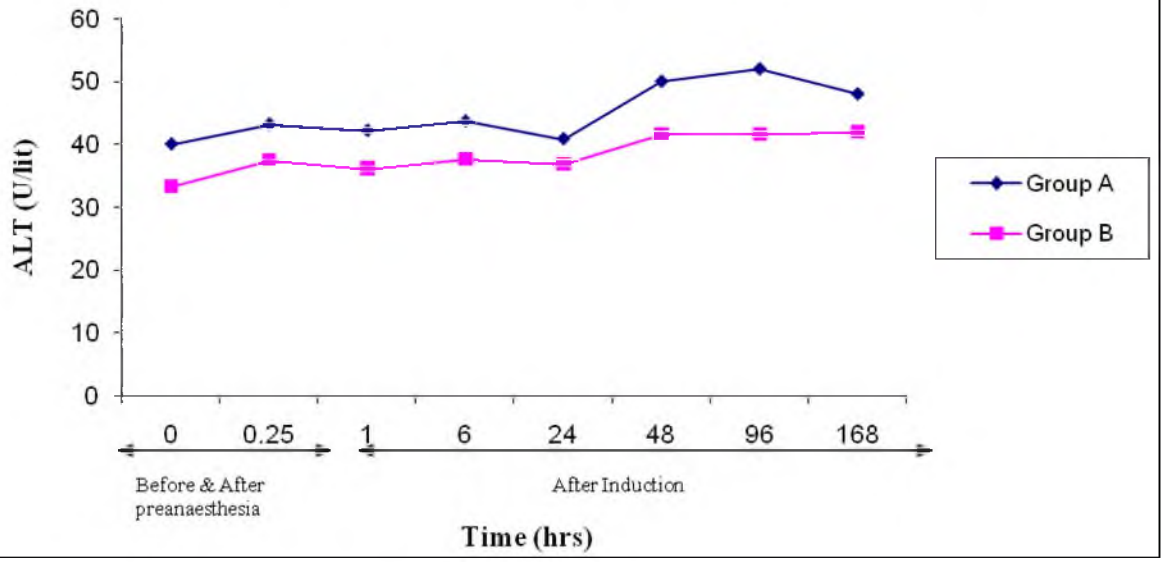


Fig. 15. Comparative mean of Alanine aminotransferase levels in Group A (Halothane) and Group B (Isoflurane) animals



6, 24 hour respectively. The mean AST level values 87.83 ± 12.48, 96.17 ± 8.95 and 85.83 ± 10.75 units/liter were increased significantly at 48, 96 and 168 hour (7 days) respectively with reference to basal mean AST levels. (Table 7 and Fig. 16).

In Group B animals, the mean AST level prior to administration of preanaesthetic and 15 minutes after preanaesthetic administration was 47.33 ± 4.66 and 62.50 ± 13.04 units/liter. After induction with isoflurane anaesthesia the mean AST levels were: 61.17 ± 8.48, 73.33 ± 8.67 (significant increase) and 78.83 ± 19.32 units/liter at the time factor of 1, 6 and 24 hour, respectively. The mean AST level values 85.17 ± 13.97, 87.67 ± 12.90 and 91.00 ± 10.21 units/liter were increased significantly at 48, 96 and 168 hour (7 days), respectively with reference to basal mean AST levels (Table 7 and Fig. 16).

4.3.3 Alkaline Phosphatase (ALP)

In Group A animals, the mean ALP level prior to preanaesthetic administration was 112.75 ± 12.71 and 15 minutes after preanaesthetic administration it was 112.33 ± 13.77 units/liter. At 1, 6, 24 and 48 hours after induction of halothane the mean ALP levels increased non-significantly were; 186.3 ± 75.28, 221.7 ± 97.85, 288.8 ± 120.9 and 147.2 ± 6.24 units per liter. Significantly increased mean values are noted as 129.0 ± 8.26 and 145.7 ± 4.70 unit/liter at 96 and 168 hour (7 days) (Table 7 and Fig. 17).

In Group B animals, the mean ALP level prior to preanaesthetic administration was 116.8 ± 6.51 and 15 minutes and after preanaesthetic administration it was 108.8

Fig. 16. Comparative mean of Aspertate aminotransferase levels in Group A (Halothane) and Group B (Isoflurane) animals

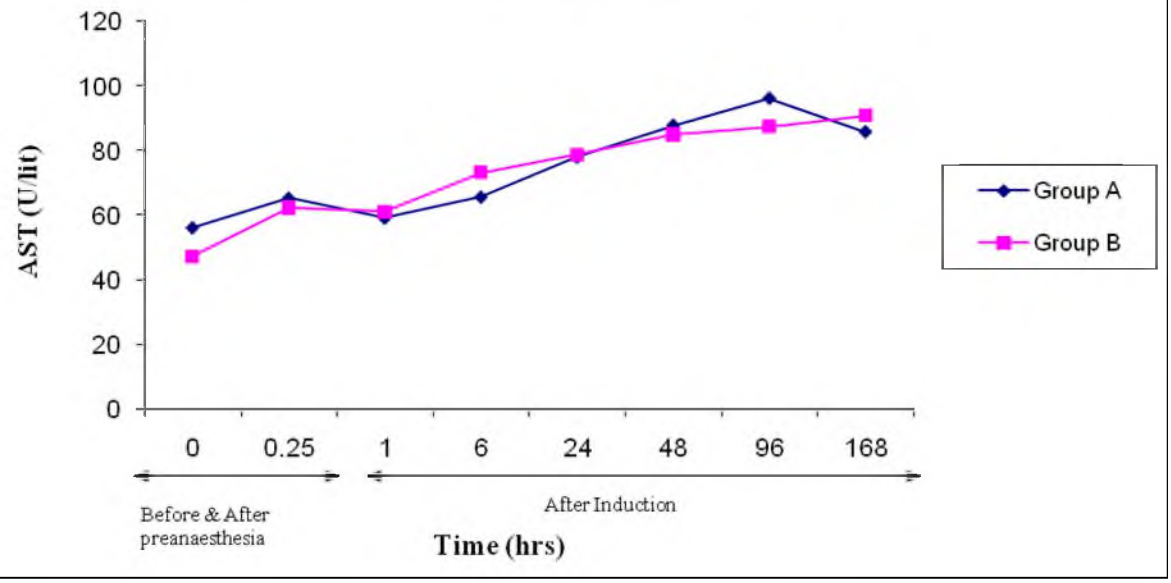
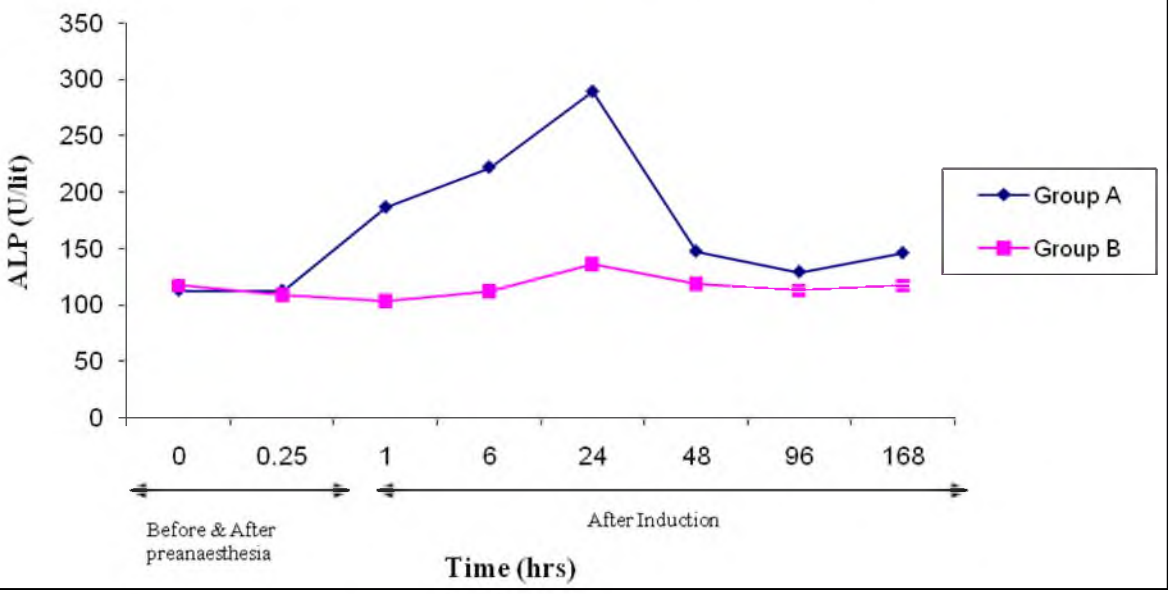


Fig. 17. Comparative mean of Alkaline phosphatase levels in Group A (Halothane) and Group B (Isoflurane) animals



± 7.37 units/liter. Following isoflurane induction the mean ALP level showing transient non-significant increase and decrease in comparison to basal mean value were; 103.2 ± 6.15, 118.5 ± 10.21, 112.7 ± 8.61, 117.0 ± 13.67 unit/liter at 1hour, 6, 24,48, 96 and 168 hours (7 days), respectively (Table 7 and Fig.17).

4.3.4 Creatinine

In Group A animals, before and 15 minutes after preanaesthetic administration the mean creatinine level were 0.94 ± 0.06 and 0.92 ± 0.04 mg/dl. After induction of halothane anaesthesia the mean creatinine level values were 0.96 ± 0.06, 1.13 ± 0.12 1.18 ± 0.08, 1.11 ± 0.13, and 1.07 ± 0.11 mg/dl at the end of 6, 24, 48, 96 and 168 hour (7 days) respectively. The creatinine values were increased non-significantly in comparison to mean creatinine basal values. (Table 7 and Fig. 18).

In Group B animals, before and 15 minutes after preanaesthetic administration the mean creatinine levels were 0.97 ± 0.06 and 0.95 ± 0.02 mg/dl, respectively. After induction of isoflurane anaesthesia the mean creatinine level values were: 0.96 ± 0.08, 0.91 ± 0.07, 0.95 ± 0.11, 1.04 ± 0.05, 1.11 ± 0.12 and 0.97 ± 0.05 mg/dl at 1hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. The mean creatinine values were transiently increasing (non-significant) and return to basal value at 48, 96 and 168th hr (Table 7 and Fig. 18).

4.3.5 Total plasma protein (TPP)

In Group A animals, the mean TPP value was 7.26 ± 0.07 g/dl prior to preanaesthetic administration medication and after preanaesthetic administration the

mean TPP value was increased to 7.71 ± 0.37. After halothane induction the mean TPP values were: 7.23 ± 0.21, 6.93 ± 0.26, 7.13 ± 0.29, 7.68 ± 0.30, 7.51 ± 0.20 and 7.31 ± 0.08 g/dl at 1 hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. During the course of study the mean TPP values were increased non-significantly at 48, 96 and 168th hr from the mean basal TPP values (Table 6 and Fig.19).

In Group B animals, the mean TPP level was 6.70 ± 0.29 g/dl prior to preanaesthetic administration medication and after preanaesthetic administration the mean TPP levels was increased to 6.94 ± 0.56. After isoflurane induction the mean TPP levels were: 6.44 ± 0.37 g/dl, 6.42 ± 0.37, 6.60 ± 0.40, 7.14 ± 0.49, 7.18 ± 0.36 and 6.63 ± 0.41 g/dl at 1 hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. During the course of study the mean TPP values were increased non-significantly at 48 and 96th hr from the mean basal TPP values (Table 7and Fig. 19).

4.3.6 Serum electrolytes

In Group A animals, prior to preanaesthetic administration the mean electrolytes: Na^+, K^+ and Cl^- ion levels were 140.0 ± 1.18, 4.11 ± 0.10 and 104.3 ± 1.83, mEq/L, respectively. After 15 minutes of preanaesthetic administration the mean levels of electrolytes: Na^+, K^+ and Cl^- were 142.5 ± 1.97, 4.26 ± 0.18 and 106.9 ± 2.10 mEq/L, respectively. Whereas, during the course of halothane general anaesthesia and during recovery phase, the respective mean electrolyte levels for Na^+, K^+ and Cl^- at 1, 6, 24, 48, 96 and 168th hour (7 days) were: 142.1 ± 1.66, 4.20 ± 0.18 and 110.3 ± 2.57; 142.1 ± 2.08, 4.40 ± 0.17 and 110.7 ± 3.30; 140.6 ± 1.31, 4.33 ± 0.26 and 102.7 ± 0.99; 143.8 ± 1.97, 4.49 ± 0.13 and 105.0 ± 1.87; 144.5 ± 1.00, 4.73

Fig. 18. Comparative mean of Creatinine levels in Group A (Halothane) and Group B (Isoflurane) animals

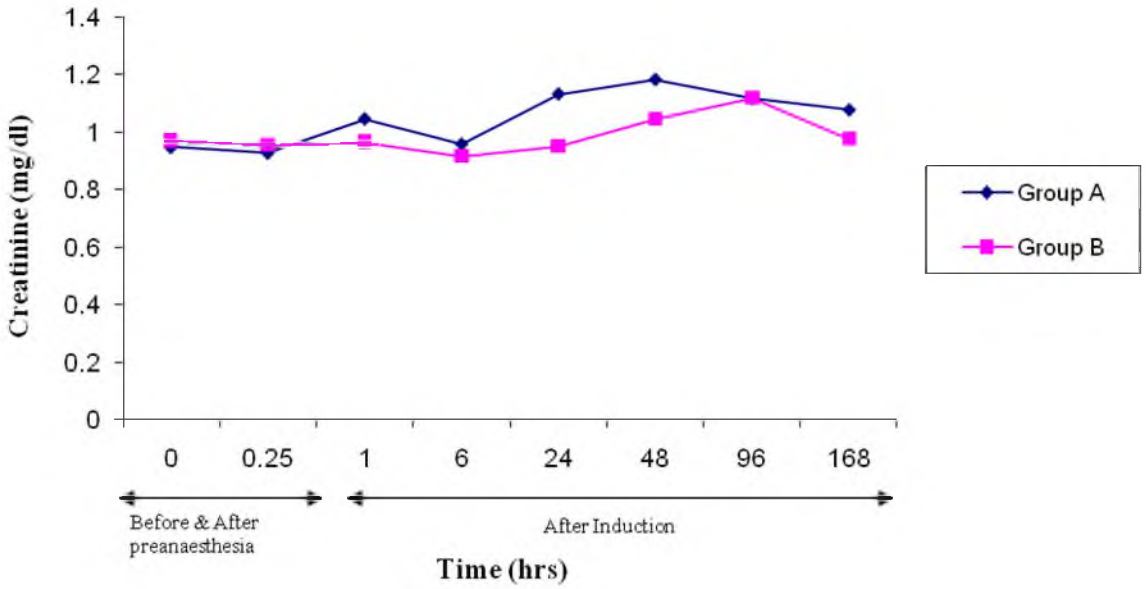
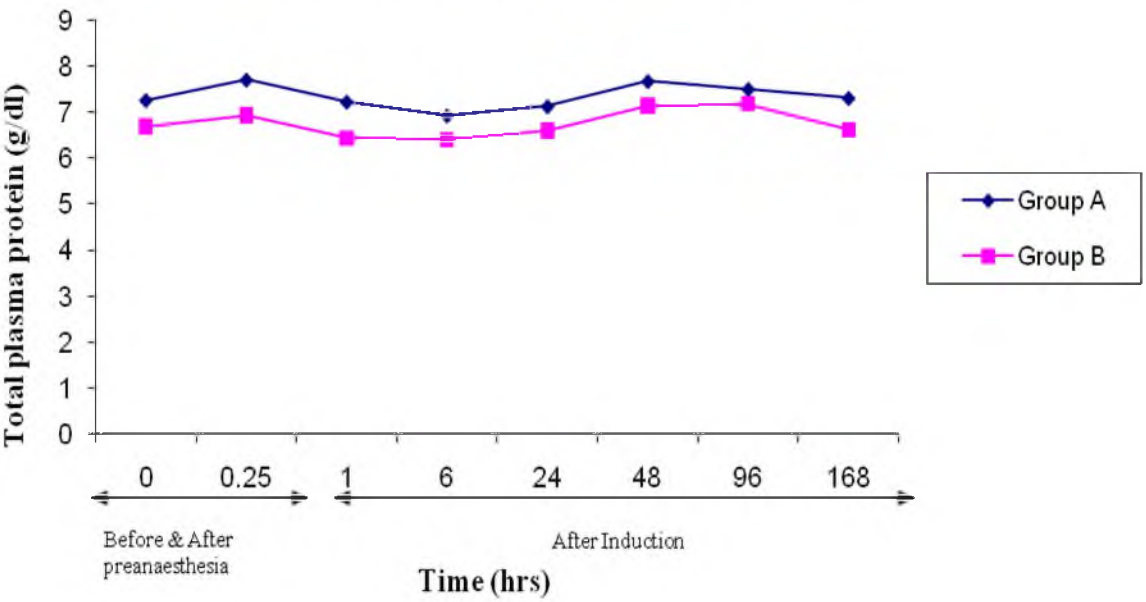


Fig. 19. Comparative mean of Total plasma protein levels in Group A (Halothane) and Group B (Isoflurane) animals



± 0.12 and 104.2 ± 1.59, and 144.7 ± 1.99, 4.54 ± 0.10 and 103.8 ± 2.30 mEq/L. No significant difference observed in Cl^- ion levels throughout the study period but at 96th hour the mean value of sodium and potassium ion levels were increased significantly (Table 7 and Fig. 20, 21and 22).

In Group B animals, prior to preanaesthetic administration the mean electrolytes: Na^+, K^+ and Cl^- ion levels were 141.8 ± 1.21, 4.15 ± 0.15 and 107.6 ± 2.24, mEq/L, respectively. After 15 minutes of preanaesthetic administration the mean levels of electrolytes: Na^+, K^+ and Cl^- were 142.9 ± 0.82, 4.55 ± 0.12 and 108.4 ± 1.79 mEq/L, respectively. Whereas during the course of isoflurane general anaesthesia and during recovery phase, the respective mean electrolyte levels for Na^+, K^+ and Cl^- at 1 hour; 6; 24; 48; 96 and 168th hour were: 143.6 ± 1.57, 4.38 ± 0.10 and 111.0 ± 2.11 mEq/L; 143.1 ± 1.61, 4.40 ± 0.08 and 109.2 ± 1.35 mEq/L; 138.9 ± 2.56, 4.43 ± 0.13 and 103.4 ± 2.54 mEq/L; 146.0 ± 1.10, 4.29 ± 0.14 and 107.4 ± 1.84 mEq/L; 144.1 ± 1.02, 4.51 ± 0.11 and 106.0 ± 1.74 mEq/L; 144.7 ± 2.01, 4.96 ± 0.19 and 107.5 ± 2.68 mEq/L, respectively (Table 7 and Fig. 20, 21 and 22). There was a non-significant transient fluctuation within limits observed in Cl^- ion level mean value but significantly increased Na^+ and K^+ ion levels were found at 48 and 168th hr in comparison to basal level values.

4.3.7 Blood glucose

In Group A animals, before the preanaesthetic administration the mean glucose level was 89.77 ± 11.01 and 15 minutes after preanaesthetic administration the mean glucose level was 89.77 ± 6.05 mg/dl. After induction of halothane anaesthesia the

Fig. 20. Comparative mean of Sodium in Group A (Halothane) and Group B (Isoflurane) animals

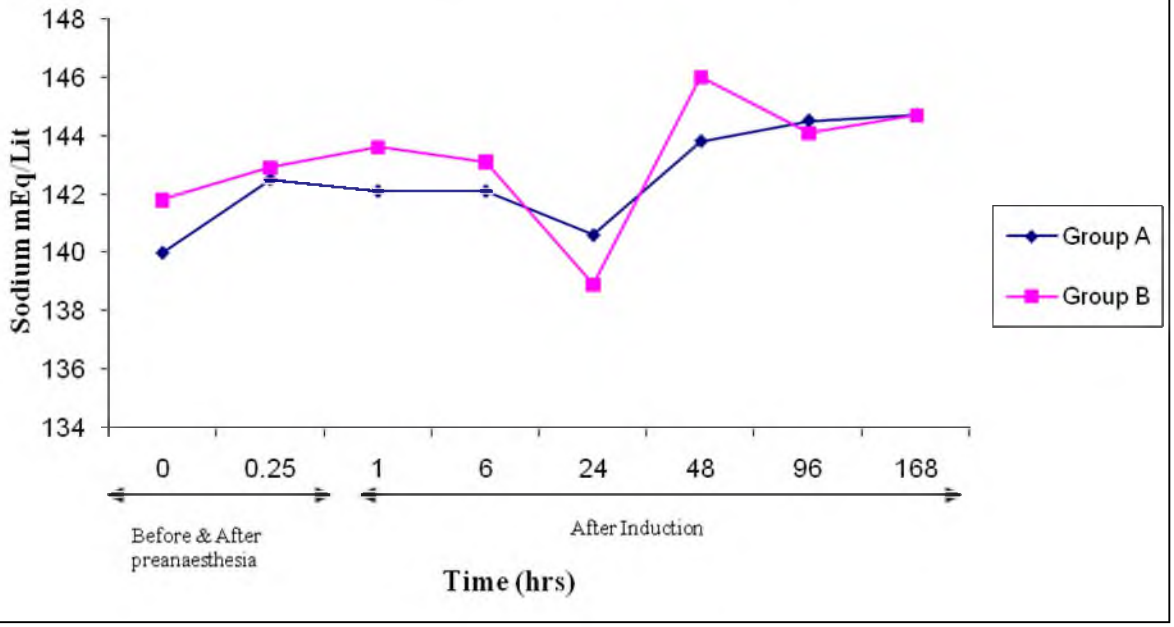
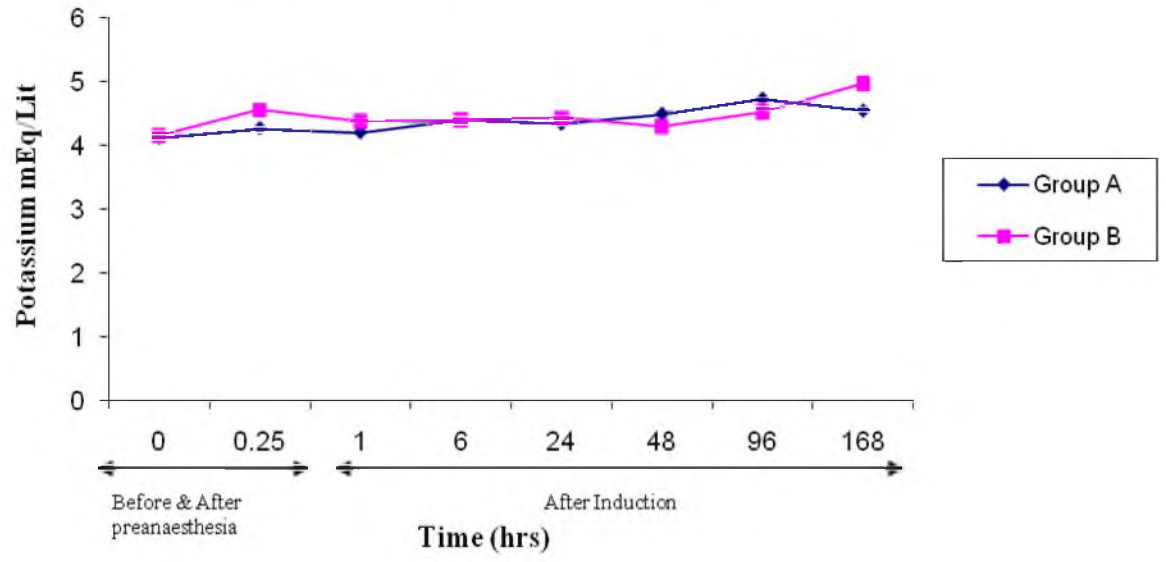


Fig. 21. Comparative mean of Potassium in Group A (Halothane) and Group B (Isoflurane) animals



mean glucose levels were: 125.1 ± 12.89, 100.0 ± 9.22, 93.75 ± 9.84, 91.13 ± 10.94, 92.38 ± 8.36 and 105.21 ± 7.97 mg/dl. at 1 hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. Non-significant increase in blood glucose level was observed during the course of study (Table 7 and Fig. 23).

In Group B animals, before the preanaesthetic administration the mean glucose level was 88.20 ± 11.28 and 15 minutes after preanaesthetic administration the mean glucose level was 89.29 ± 6.01 mg/dl. Following isoflurane anaesthesia the mean glucose levels were:126.5 ± 12.62, 97.60 ± 9.80, 87.25 ± 10.15, 83.55 ± 9.28, 85.03 ± 7.26 and 97.00 ± 8.90 mg/dl. at 1 hour, 6, 24, 48, 96 and 168 hour (7 days), respectively. Non-significant transient increase and decrease values within limits were observed during course of the study (Table 7 and Fig. 23).

Fig. 22. Comparative mean of Chloride in Group A (Halothane) and Group B (Isoflurane) animals

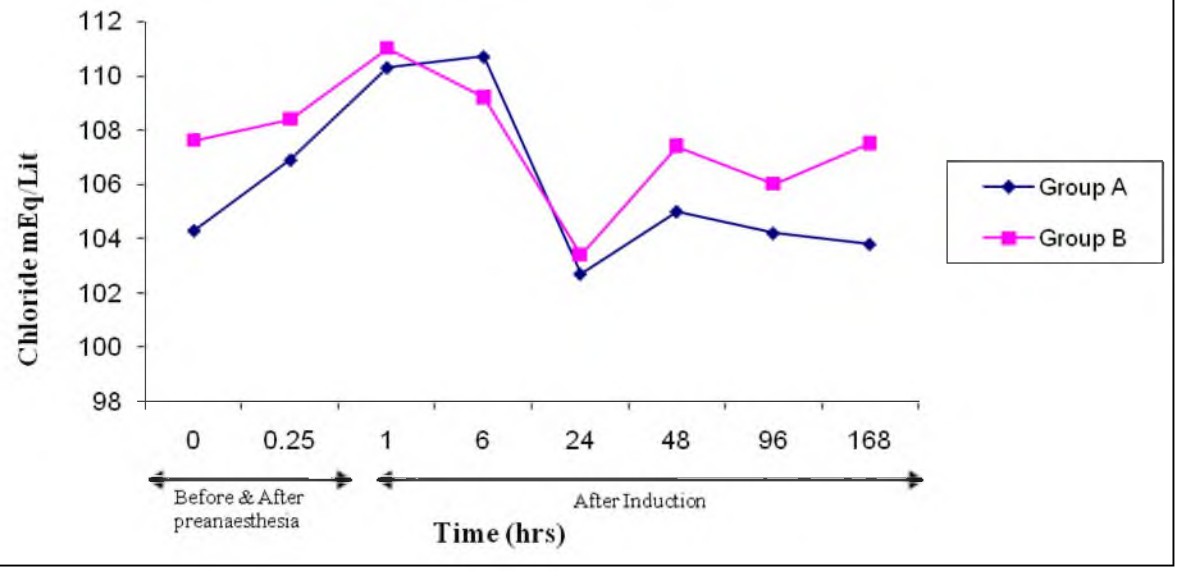
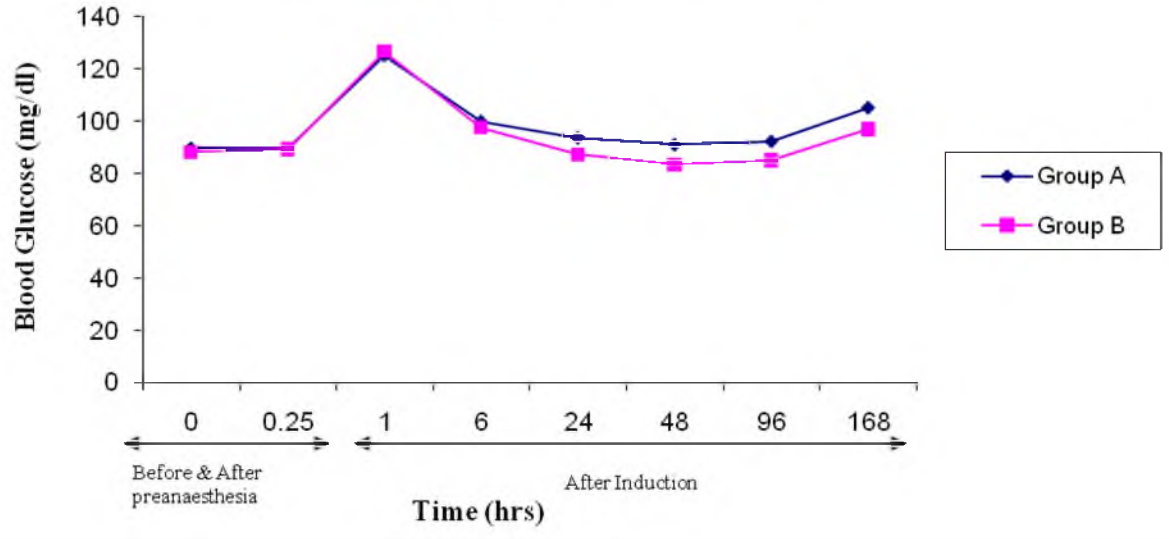


Fig. 23. Comparative mean of Blood glucose levels in Group A (Halothane) and Group B (Isoflurane) animals



V. DISCUSSION

The purpose of this study was to assess the comparative clinical efficacy of halothane and isoflurane inhalant anaesthesia in clinical cases of dogs undergoing ovariohysterectomy procedure. The results from this study are discussed under the following headings:

5.1 Different parameters assessed during inhalation anaesthesia in bitches undergoing ovariohysterectomy

5.1.1 Onset of sedation

During preanaesthetic period, from the point of acepromazine maleate administration. Optimum level of sedation was observed by a mean duration of 7.04 ± 0.58 and 6.48 ± 0.28 minutes in Group A and Group B animals, respectively. Acepromazine maleate is a phenothiazine derivative having potent neuroleptic property with relatively low toxic effects. It has been proved very safe compared to other premedicants for administering inhalation anaesthesia in dogs (Brodbelt *et al.*, 2006). Further, it is evident from the literature that Acepromazine maleate has been successfully employed as a premedicant for different studies (Jones and Snowdon 1986; Smith *et al.*, 1993; Wright *et al.*, 1996, Robertson *et al.*, 2001; Hui Cheng Chen *et al.*, 2003 and Steagall *et al.*, 2006). When administered in high doses prior to epinephrine administration, Acepromazine maleate effectively prevents cardiac arrhythmias and ventricular fibrillation in dogs during barbiturate, methoxyflurane and halothane anaesthesia (Wiersig *et al.*, 1974 and Teixeira-Neto *et al.*, 2001).

5.1.2 Induction of inhalation anaesthesia

During the present study, the halothane and isoflurane inhalation anaesthesia was administered by semi closed method, using a close fitting face mask. The mean duration to attain optimum level of induction for halothane (Group A) and isoflurane (Group B) was 8.26 ± 1.55 and 3.01 ± 0.16 minutes, respectively. In both the groups, inhalation anaesthesia (halothane or isoflurane) was administered in combination with pure oxygen at the flow rate of 1.5L/min. Aneasthesia induction was smooth and uneventful in all the animals. Successful administration of inhalation anaesthesia using a tight fitting mask or tracheal intubation has been reported by different researchers (Fisher 1961; Bednarski *et al.* 1984; Martin *et al.* 1997; Martinez *et al.* 1998 and Alexander *et al.* 2003).

5.1.3 Average maintenance dose volume of halothane and isoflurane anaesthesia (Vol %)

The average maintenance volume of anaesthesia for Group A (halothane) and Group B (isoflurane) animals was 3.34 ± 0.18 and 2.61 ± 0.25 (Vol %), respectively. Beneken Kolmer *et al.* (1975) in their experiment, maintained the halothane anaesthesia at flow rate of 0.5 to 1.0 vol %. Denise *et al.* (2005) used anaesthetic concentrations of 1 MAC for halothane, isoflurane and sevoflurane. Martin *et al.* (1997) used isoflurane as maintenance anaesthesia at the rate of 1.5 MAC. Lee *et al.* (1998) used isoflurane at 1.9 ± 0.1 Vol%. During present study the Vol % observed in both Group A and Group B animals was slightly at higher range than values reported in the literature. This may be attributed to the method (semi closed mask method)

used to administer anaesthesia or due to the type of equipment used (anaesthetic evaporator, mask and other accessories) as there might have been leakage or partial loss of anaesthesia at face-mask junction.

5.1.4 Monitoring surgical plane of anaesthesia during ovariohysterectomy procedure

During present study, the average duration of anaesthesia required for completion of ovariohysterectomy procedure was 33.68 ± 2.59 and 32.04 ± 1.59 minutes in Group A and Group B animals, respectively. In both the groups, no salivation or vomition was observed during the entire course of anaesthetic procedure which indicated the successful and effective preanaesthetic procedure followed during this study. The dosage of atropine sulphate administered during this study was effective in preventing possible untoward effects (salivation, vomiting and respiratory secretions) by way of its anticholinergic effect. Surgical plane of anaesthesia was ascertained by monitoring for absence of pinna reflex, pedal reflex or anal reflex and, presence of ventromedial positioning of eye ball and skeletal muscle relaxation during the course ovariohysterectomy procedure in both the group of animals. In both the groups, the entire course of inhalant anaesthetic regimen followed during ovariohysterectomy procedure in clinical cases of dogs was uneventful.

5.1.5 Quantitative parameters recorded during recovery

The beginning of recovery phase of anaesthesia was marked from the point of cessation of anaesthesia, which coincides with the last skin suture applied. In Group A animals, the mean duration for return of swallowing reflex, pedal reflex, head

righting reflex, voluntary leg movement, attainment of sternal recumbency and duration for animals to ambulate were: 9.53 ± 1.09, 15.04 ± 2.56, 25.44 ± 5.58, 17.75 ± 2.86, 27.97 ± 4.91 and 42.89 ± 4.42 minutes, respectively. These readings were in agreement with the similar observations from Yang *et al.* (1999) and Redondo *et al.* (2000).

In Group B animals, the average duration for return of swallowing reflex, pedal reflex, head righting reflex, voluntary leg movement, attainment of sternal recumbency and duration for animal to ambulate were: 3.18 ± 0.43, 5.54 ± 0.51, 7.32 ± 0.95, 7.67 ± 1.15, 10.28 ± 1.21 and 16.94 ± 4.17 minutes, respectively. In Group B animals the reappearance of reflexes were faster, recovery was smooth and the animals stood up with minimal efforts. The number of attempts made by animals to stand upright were fewer and seemingly uncomplicated when compare to that of Group A animals. This could be mainly attributed to the low solubility of isoflurane, which facilitated faster elimination from the body. Further, similar findings have been reported by Meyer *et al.* (1984); Jones and Seymour (1986); Hellebrekers (1986); Johnson *et al.* (1998) and Sloan *et al.* (1996).

5.1.6 Qualitative parameters recorded during recovery

In Group A, the animals used to sleep for longer duration, the position change was not so frequent this may be due to residual effect of halothane in blood. During recovery phase head position was lowered in all the animals. 50% of animals were alert and watchful at any movement (personnel) surrounding recovery room. Transition from surgical plane of anaesthesia to full consciousness state was not

smooth. Moderate paddling or other unconscious movements were seen all along the recovery phase. Vocalisation was evident and most of the animals had difficulty positioning themselves. In this lip licking, licking of floor, legs and bandage were predominantly observed. This may have been attributed to the restless suffered during recovery phase. Similar findings were recoded by Fox *et al.* (2000).

In Group B animals, recovery phase was relatively a smooth transition from anaesthesia to the state of full consciousness. No struggling, paddling, tremors or other unconscious movements observed. Moderate attempts of lip licking, licking of floor, legs and bandage were evident but at lesser frequency compared to the halothane group.

5.1.7 Rectal temperature

In both Group A and Group B animals, rectal temperatures were recorded at different interval period during the course of study period. Significant drops in rectal temperatures were observed in both the groups of animals after induction with halothane or isoflurane anaesthesia (15, 30, 45 and 60 minute's time point intervals). This may could have been due to the reduced activity of reticular activating system and depression of thermoregulatory centre along with decreased metabolic rate and reduced skeletal muscle activity during sedation and anaesthesia (Goodman and Gilman, 1980 & 2001). These observations are also in agreement with the findings of Meyer *et al.* (1984); Pettifer and Hosgood (2004) and Pottie *et al.* (2007);

5.1.8 Respiratory rate

In both Group A and Group B animals the respiratory rate was decreased after preanaesthetic administration and also during the period of induction, maintenance and recovery phase of inhalation anaesthesia. In Group A significant drop in respiratory rate was seen during recovery period both at 45 and 60 minute following induction compared to basal value. The decrease in respiratory rate might have been due to the dose dependent depressing effect of anaesthesia on higher respiratory centres. The results of present study are found to be in agreements with similar findings of Mutoh *et al.* (1997); Lerche and Muir (2006); Kilic and Isler (2005); Hellebrekers (1986) and Teixeira Neto *et al.* (2007).

5.1.9 Heart rate

In Both Group A and Group B animals, decreased heart rate was observed after preanaesthetic administration and at 15 minutes interval following induction of inhalant anaesthesia. Decrease in heart rate may have been due to the depression effect of halothane on myocardial contractility i.e. a direct cardio depressant effect on cardiac excitation-contraction coupling, which is mediated by an interaction with the L-type Ca^{2+} channel has been put forth (Kanaya *et al.* 1998). However, Mutho *et al.* (1997), Topal *et al.* (2004) and Teixeira Neto *et al.* (2007) reported that the heart rates were higher with isoflurane in comparison with halothane. In another study by Denise *et al.* (2005), it is stated that during acute normovolemic hemodilution in dogs the heart rate was not changed in isoflurane group whereas the heart rate was decreased in halothane group. This may be due to less depressant effect of baroreceptor-reflex

function with isoflurane in comparison to halothane. Duke *et al.* (2006) reported an increased heart rate in isoflurane-nitrous oxide combination and decreased heart rate in halothane-nitrous oxide group. Martin *et al.* (1997) observed no significant changes in heart rate after administering epidural ketamine in combination with isoflurane anaesthesia in dogs.

5.2 Haematological parameters

5.2.1 Total erythrocyte count, packed cell volume and haemoglobin

There was a slight drop in total erythrocyte count (TEC), packed cell volume (PCV) and haemoglobin (Hb) levels in both the groups at 15 minutes following preanaesthetic medication, which could have been attributed to spleenic pooling of blood constituent (Collette and Meriwether, 1965; Lang *et al.* 1979), shifting of fluid from extra vascular compartment to intravascular compartment to maintain normal cardiac output (Coles and Campbell 1986). No significant alterations in haematological data were observed in both within and between groups. Similar observations were recorded by Robertson *et al.* (2001); Muir *et al.* (2004) and Wilson *et al.* (2004).

5.2.2 Total leukocyte count, differential leukocyte count and platelet count

The total leukocyte count (TLC) was measured in both the groups throughout the period of study. Results revealed a non-significant increase in TLC levels at 6, 24, 48 and 96^{th} hrs in Group A animals. In Group B animals, a non-significant increase in TLC levels were observed only at 6^{th} and 24^{th} hrs when compared basal levels. Differential leukocyte count (DLC) in both Group A and Group B animals showed a

non-significant variation compared to the basal levels (Table No. 6). No significant alterations in TLC or DLC levels were observed in both within and between groups. In both Groups, the mean Platelet count decreased non-significantly from mean basal PLT values and no significant difference was observed within as well as among the Groups. These findings were in agreement with the similar observations from Collette and Meriwether, 1965; Lang *et al.* 1979.

5.3 Biochemical parameters

In Group A, alanine aminotransferase (ALT) increased significantly at 96 and 168th hrs whereas in Group B there was a non-significant increase throughout the period of study. No significant changes were found between the groups. In Group A, alkalinephosphatase (ALP) increased significantly at end of 96 and 168th hours (7th day) basal levels but in Group B there was a transient non-significant increase and decrease in comparison to basal mean value. At 48th hour time interval, significantly lower value observed in Group B in comparison to same time point interval of Group A. Aspartate aminotransferase (AST) increased significantly at 48, 96 and 168 (7th day) in both Group A and Group B. However, between the groups, no significant change was observed in AST levels. The increase in AST levels in both Groups might have been due to the possible hepatotoxic effect of the portion of anaesthetic component that underwent biotransformation in the liver. Topal *et al.* (2003) observed significantly increased serum AST and ALT activities all the time after anaesthesia compared with baseline activities in the halothane group, but in the isoflurane group AST and ALT activities increased only between 2 and 7 days. Authors opined that this was due to use of halothane anaesthesia (high blood solubility) that might have

induced an increased enzyme activity in liver more frequently than isoflurane (low blood solubility) in dogs. They also opined that isoflurane anaesthesia exerts minimal adverse effects on liver when compared to halothane anaesthesia in dogs.

Creatinine levels did not differ significantly in both within and between groups throughout the study period. In Group A, non-significant increase in creatinine levels were observed at 1, 24, 48 96 and 168th hrs during post-anaesthetic period. In Group B, creatinine levels non-significantly increased at 48 and 96th hrs. Similar findings were recorded by Martinez *et al.* (1996) in dogs administered with isoflurane and Ko JC *et al.* (2000) in carprofen administered healthy dogs anesthetized with propofol and isoflurane.

The mean TPP value in both groups increased non-significantly at 48, 96 and 168th hrs (only in Group A) but no significant alterations in TPP levels were observed in both within and between groups. Similar reports were documented by Wilson *et al.* (2004) in halothane anaesthetized dogs. However, Muir *et al.* (2004) reported a decrease in serum protein after administration of lactated Ringer's solution and a physiologically balanced 6% hetastarch plasma expander in isoflurane anaesthetized dogs where this could have been attributed the plasma diluting effect hetastach administered during their study.

In both, within and between the groups, serum chloride ion levels were varied (slight increase or slight decrease) non-significantly throughout the period of study. Whereas in Group A, at 96th hour, the mean value of sodium and potassium ions levels were increased significantly from that of basal levels. In Group B, the mean

sodium ion levels at 48th hour was significantly higher compared to basal levels. No significant changes either in potassium or sodium ions levels were observed between the groups.

A transitory non-significant increase in blood glucose levels was observed after induction in both Group A and Group B animals. This may have been due to surgical stress induced release of cortisol, which might have induced transitory gluconeogenesis leading to hyperglycaemia. Nahed-Saleh *et al.* (2005) reported initially increased and subsequently decreased Plasma glucose levels in medetomidine-isoflurane anaesthetized dogs. During present study, in blood glucose levels were observed between the groups.

VI. SUMMARY

A comparative assessment on clinical efficacy of halothane and isoflurane inhalant anaesthesia in clinical cases of dogs undergoing ovariohysterectomy was undertaken during this study. Usage of anaesthetic having characteristics of rapid induction and quicker elimination with minimal side effects is not only advantageous to the patient but it also minimizes the possible health hazards to both the surgeon or operating team and animal care takers (Werner, 1987, Topal *et al.* 2003, Kilic and Isler 2005). With these considerations in view, the present study was conducted with the following objectives: (i) to assess and compare clinical efficacy of halothane and isoflurane anaesthesia in bitches undergoing ovariohysterectomy procedure; (ii) to compare various clinical parameters like rectal temperature, respiratory rate and heart rate; (iii) to assess and compare the influence of halothane and isoflurane anaesthesia on haematological and biochemical parameters such as total erythrocyte count (TEC), packed cell volume (PCV), hemoglobin concentration (Hb), total leukocyte count (TLC), differential leukocyte count (DLC), platelet count(PLT), alanine aminotransferase (ALT), aspertate aminotransferase (AST), alkaline phosphatase (ALP), plasma creatinine, total plasma protein (TPP), electrolytes (sodium, potassium and chloride) and blood glucose and (iv) To record complications if any, during Anaesthesia and post anaesthetic period.

Twelve cases of female dogs were randomly allotted into two groups of six animals in each *Viz.*, halothane anaesthesia (Group A) and isoflurane anaesthesia (Group B). Results from the present study revealed that for ovariohysterectomy procedure in bitches, usage of isoflurane general anaesthesia was advantageous over halothane

anaesthesia in terms of speed and quality of induction, maintenance and recovery from anaesthesia. There were non-significant minor alterations in haematological parameters and no changes were observed in creatinine (Ko JC *et al.* 2000) and electrolytes levels. Significant alterations were noted with respect to ALT in halothane administered animals (Group A), which may have been attributed to the hepatotoxic effects of halothane in dogs (Topal *et al.* 2003). No such adverse evidence was observed in isoflurane administered animals (Group B). During post anaesthetic period, as compared to halothane, the animals anaesthetized with isoflurane were quicker in regaining reflexes, recovery was smooth and the animals were able to attain standing posture with minimal efforts.

In conclusion, isoflurane aneasthesia was comparatively advantageous over halothane anaesthesia in terms of ease of administration, maintenance and recovery with minimal adverse effects on physiological status of bitches subjected for ovariohysterectomy procedure.

VII. BIBLIOGRAPHY

ADAMS WA., MARK SENIOR. J., JONES R.S., WILLIAMS J and GLEED R.D., 2006. cis-Atracurium in dogs with and without porto-systemic shunts. *Vet. Anaest. Analg.*, **33**(1):17-23

ALEXANDER VALVERDE., TIMOTHY, E., MOREY., JORGE HERNANDEZ and WENDY DAVIES., 2003. Validation of several types of noxious stimuli for use in determining the minimum alveolar concentration for inhalant anesthetics in dogs and rabbits. *Am. J. Vet. Res.*, **8**:957-962

ANDREAS, W., LOEPKE, M.D., JOHN. C., MCCANN, B.S., DEAN KURTH, M.D. and JOHN, J. MCAULIFFE., 2006. The Physiologic Effects of Isoflurane Anesthesia in Neonatal Mice. *Anesth Analg.*, **102**:75-80

BAGSHAW, R.J., COX, R.H., KNIGHT, D.H. and DETWEILER, D.K., 1978. Malignant hyperthermia in grey hound. *J. Am.Vet. Med. Ass.*, **172**(1):61-62

BALI, I. M., DUNDEE, J. W. and ASSAF, R. A. E., 1975. Immediate changes in plasma potassium, sodium and chloride concentrations induced by suxamethonium. *B. J. Anaesth.*, **47:**393-397

BEDNARSKI, R.M., BEDNARSKI, L.S. and MUIR, W.W., 1984. Cost comparison of anesthetic regimens in the dog and cat. *J. Am.Vet. Med. Ass.*, **185**(8): 869-872

BENEKEN KOLMER, H. H., BURM, A. G., CRAMERS, C. A., RAMAKERS, J. M. and VADER, H. L., 1975. The uptake and elimination of halothane in dogs: A two or multicompartment system? II: Evaluation of wash-in and wash-out curves. *B. J. Anaesth.*, **47**:1169-1175

BENSON, G.J., GRUBB, T.L., NEFF DAVIS, C., OLSON, W.A., THURMON, J.C., LINDNER, D.L., TRANQUILLI, W.J. and VANIO, O., 2000. Perioperative stress response in the dog: effect of pre-emptive administration of medetomidine. *Vet. Surg.*, **29**(1): 85-91

BOSCAN, P., PYPENDOP, B.H., SLOANE, A.M. and ILKIW, J.E., 2005. Cardiovascular and respiratory effects of ketamine infusions in isoflurane-anesthetized dogs before and during noxious stimulation. *Am. J. Vet. Res.*, **66**(12): 2122-2129

BOUDA, J. and PAVALICA, J., 1970. Acid base changes in the blood of cows during halothane narcosis., *Acta. Vet. Brno.*, **39**:265-270. (*Vet. Bull.*, **41**:2588.1971)

BRANDES, I.F., ZUPERKU, E.J., STUCKE, A.G., HOPP, F.A., JAKOVCEVIC, D. and STUTH, E.A., 2007. Isoflurane Depresses the Response of Inspiratory Hypoglossal Motoneurons to Serotonin In Vivo. *Anesthesiology.*, **106**(4):736-745

BRODBELT D.C., HAMMOND R., TUMINARO, D., PFEIFFER, D.U and WOOD, J.L.N., 2006. Risk factors for anaesthetic-related death in referred dogs. *Vet. Rec.* **158**(16): 563-564

BRONDANI, J.T., NATALINI, C.C., RAISER, A.G., MAZZANTI, A. and PRATI, L., 2004. Analgesia epidural com clonidina ou romifidina em caes submetidos a cirurgia coxofemoral. Source. *Arquivo-Brasileiro-de-Medicina-Veterinaria-e-Zootecnia*. Publication, Belo Horizonte, Brazil: Escola de Veterinaria, Universidad Federal de Minas Gerais., **56**(2): 175-182

CARTER, H.E., 1964. Induction of anaesthesia in dogs by the inhalation of nitrous oxide, Halothane and oxygen. *Vet. Rec.*, **76**: 147-148.

CHAMBELL, D., FITCH, W. AND HUGHES, R.L., 1980. The effect of enflurane and halothane upon liver blood flow and oxygen consumption in the grey hound., *Br. J. Pharmacol.* **69**(2): 315

CHANOIT, G.P., SCHNEIDER, M., WOEHRLE, F. AND LEFEBVRE, H.P., 2005. Effect of marbofloxacin on cardiovascular variables in healthy isoflurane-anesthetized dogs. *Am.J. Vet.Res.*, **66**(12): 2090-2094

COLES, E. H. and CAMPBELL., TW., 1986. Veterinary *clinical* pathology, 4th edn. Saunders, Philadelphia, pp 279-291. 200. Comp *Clin Pathol* (2008) **17**:197–201

COLLETTE, W.L. and MERIWETHER, W.F., 1965. Some changes in the peripheral blood of dogs with administration of certain tranquilizers and narcotics. *Vet. Med.*, **60**: 1223.

DENISE, T.F., DENISE, A.O., ALINE, M. A., EUNICE, Y. T. and JOSÉ, O.C.A., 2005. A Comparative Evaluation of Inhaled Halothane, Isoflurane, and Sevoflurane during Acute Normovolemic Hemodilution in Dogs. *Anesth Analg.*, **100**:1014-1019

DEYOUNG, D.J. and SAWYER, D.C., 1980. Anaesthetic potency of nitrous oxide during halothane anaesthesia in the dog. *J. Am. An. Hosp. Ass.*, **16**(1): 125-128.

DHILLON, K.S. and PAUL, B.S., 1972. The effects of different premedications on the recovery time of dogs from halothane anaesthesia. *Cey.Vet.J.*, **20**:728. (*Vet.Bull.*, **43**:918.1973)

DUKE, T., CAULKETT, N.A. and TATARYN, J.M., 2006. The effect of nitrous oxide on halothane, isoflurane and sevoflurane requirements in ventilated dogs undergoing ovariohysterectomy. *Vet. Anaesth Analg.*, **33**(6):343-350

EGGER, C., MCCRACKIN, MA., HOFMEISTER, E., TOUZOT-JOURDE, G., ROHRBACH, BE., 2009. Efficacy of preanesthetic intramuscular administration of ephedrine for prevention of anesthesia-induced hypotension in cats and dogs. *Can. Vet. J.*, **50**(2):179-84

ELIZABETH, A. S., MERYL, P. LITTMAN., ROBERTSON, L. and KENNETH, C. BOVEE., 1988. Renal dysfunction in dogs with pyometra. *J. Am. Vet. Med. Assoc.* **193**(4): 457-464

FISHER, E.W., 1961. Observations on disturbances of respiration of cattle, horses, sheep and dogs caused by halothane anaesthaesia and changes taking place in plasma pH and plasma carbon di oxide content. *Am. J.Vet. Res.*, **22**:279-286

FOX, S. M., MELLOR, D. J., STAFFORD, K. J., LOWOKO, C.R.O and HODGE, H., 2000. The effect of ovarihyterectomy plus different combinations of halothane anaesthesia and butorphanol analgesia on behaviour in the bitches. *Res. Vet. Science.*, **68**: 265-274

GATES J.B.JR., BOTTA, J.A. and TEER, P.A., 1971. Blood gas and pH determination in cattle anesthetised with Halothane. *J. Am. Vet. Med. Ass.*, **158**:1678-1682 (*Vet. Bull.*, **91**:6571)

GERDING, P.A.JR., BENSON, G.J., HAMOR, R.E. and SCHAEFFER, D.J., 2002. Onset and duration of paralysis and effect on intraocular pressure of pancuronium bromide in isoflurane-anaesthetized dogs. *J.Ani.Vet.Adv.*, **1**(4): 186-189

GOODMAN, L.S. AND GILMAN, A., 1980. *The Pharmacological Basis of Therapeutics*. **VI Edn**., McGraw Hill Medical Pub., New York, pp. 395-413

GOODMAN, L.S. AND GILMAN, A., 2001. *The Pharmacological Basis of Therapeutics*. Eds. Hardman, J.G. and Limbird, L.E., X Edn., McGraw Hill Medical Pub., New York, pp. 599-601

GREENE, S.A., MOORE, M.P., KEEGAN, R.D. and GALLAGHER, L.V., 1992. Quantitative electroencephalography for measurement of central nervous system responses to diazepam and the benzodiazepine antagonist, flumazenil, in isoflurane-anaesthetized dogs. *J.Vet. Pharmacol. Ther.*, **15**(3):259-266

GROSS, M.E., POPE, E.R., O'BRIEN, D., DODAM, J.R. and POLKOW, H.J., 1997. Regional anesthesia of the infraorbital and inferior alveolar nerves during non-invasive tooth pulp stimulation in halothane-anesthetized dogs. *J. Am. Vet. Med. Ass.*, **211**(11):1403-1405

GUOCHANG HU., JAKOB V.J., M. RAMEZ SALEM., ZHI- QING ZHAO and GEORGE J. CRYSTAL, 2002. Isoflurane inhibits Neutrophil- Endothelium interactions in the coronary circulation: Lack of a role for Adenosine triphosphate sensitive potassium channels. *Anesth. Analg.*, **94**:849-856

HALL, L.W., 1957. Bromochloro trichloroethane (Flurathane) a new volatile anaestheic agent. *Vet. Rec.*, **69**: 615-618

HALNAN, C. R. E., 1967. Fluothane anaesthesia using a Blease vaporiser. *J. Small Anim. Pract.*, **8**: 717-724

HELLEBREKERS, L.J., 1986. Comparison of isoflurane and halothane as inhalation anaesthetics in the dog. *Vet. Quart.*, **8**(3):183-188

HELLYER, P.W., MAMA, K.R., SHAFFORD, H.L., WAGNER, A.E. and KOLLIAS, B.C., 2001. Effects of diazepam and flumazenil on minimum alveolar concentrations for dogs anesthetized with isoflurane or a combination of isoflurane and fentanyl. *Am.J.Vet.Res.*, **62**(4):555-560

HIDEO, H.M., HIROAKI, M., HIROSHI, M., MASANORI, M., TAKEHIKO, K. and KOJI, S., 2005. Isoflurane Protects Renal Function Against Ischemia and Reperfusion Through Inhibition of Protein Kinases, JNK and ERK *Anesth Analg.*, **101**:1584-1589

HUI CHENG CHEN., MELISSA D. SINCLAIR and DORIS H. DYSON., 2003. The Use of Ephedrine in Dogs under Isoflurane to Manage Hypotension: A Preliminary Investigation . *World Small Animal Veterinary Association World Congress Proceedings*

JOHNSON R.A., STRILER, E., SAWYER, D.C. and BRUNSON, D.B., 1998 Comparison of isoflurane with sevoflurane for anesthesia induction and recovery in adult dogs. *Am. J. Vet. Res.*, **59**(4):478-81

JONES, R.S. and SEYMOUR, C.J., 1986. Clinical experiences with isoflurane in dogs and horses. *Vet. Rec.*, **119**(1): 8-10

JONES, R.S. and SNOWDON, S.L., 1986. Experimental investigation of the cardiovascular and respiratory effects of increasing concentrations of isoflurane in the dog. *Res. Vet. Sci.*, **40**(1): 89-93

JOUBERT, K.E., 2004. Does the A-line ARX-Index provide a reasonable assessment of anaesthetic depth in dogs undergoing routine surgery? *J. Sou. Afr. Vet. Ass.*, **75**(3): 110-115

KANAYA, N., MANABE, M., TSUCHIDA, H. 1998. Comparative myocardial depression of Sevoflurane, isoflurane and halothane in cultured neonatal rat ventricular myocytes. *Anesth. Analg.*, **67**: 1041-1047

KATOH, T. and IKEDA, K.A., 1994. Comparison of sevoflurane with halothane, enflurane, and isoflurane on bronchoconstriction caused by histamine *Can. J. Anesth.*, **41**:1214-1219

KELLY, A.B., STEFFEY, E.P., MCNEAL, D. and WILLITS, N., 1985. Immediate and long term effects of halothane anaesthesia on equine platelet function. *J. Vet. Pharm. Therap.*, **8**(3): 284-289. (*Vet. Bull.*, **55:** 8058)

KILIC, S. and ISLER, C.T., 2005. Evaluation of different anesthetic agents in dogs. *Indian.Vet.J.*, **82**(6): 621-624

KLIDE, A.M., 1976. Cardiopulmonary effects of enflurane and isoflurane in the dog. *Am. J. Vet. Res.*, **37**(2):127-131

KO, J.C., MIYABIYASHI, T., MANDSAGER, R.E., HEATON JONES, T.G. and MAURAGIS, D.F., 2000. Renal effects of carprofen administered to healthy dogs anesthetized with propofol and isoflurane., *J. Am. Vet. Med. Assoc.*, **217**(3): 346-349

KRISTER and IWARSSON., 1966. Fluothane anaesthesia without artificially assisted respiration.Effects upon blood pressure, Blood gases and acid base equilibrium in surgical treatment of dogs. *Nord. Vet. Med.*, **18**:569-576

LANG, S.M., EGLEN, R.M. and HENRY, A.C., 1979. Acetylepromazine administration: its effect of canine haematology. *Vet. Rec.*, **105**: 397-398.

LAVOR, M.S.L., POMPERMAYER, L.G., NISHIYAMA, S.M., DUARTE, T.S., FILGUEIRAS, R. and ODENTHAL, M.E., 2004. Fetal and maternal effects of propofol, etomidate, thiopental and epidural anaesthesia in bitches undergoing elective caesarean section. *Ciencia-Rural.*, **34**(6): 1833-1839

LEE, D.D., MEYER, R.E., SULLIVAN, T.C., DAVIDSON, M.G., SWANSON, C.R. and HELLYER, P.W., 1998. Respiratory depressant and skeletal muscle relaxant effects of low-dose pancuronium bromide in spontaneously breathing, isoflurane-anesthetized dogs. *Vet. Surg.*, **27**(5): 473-479

LEONE, B.J., LEHOT, J.J., RUNCIMAN, W.B., WELDING, R.N., RAMSAY, J.G., ARVIEUX, C.C., RYDER, W.A. and FOEX, P., 1988. Effects of lignocaine and bupivacaine on regional myocardial function and coronary blood flow in anaesthetized dogs. *Br.J.Anaesthesia.*, **60**(6): 671-679

LERCHE, P. and MUIR, W.W.III., 2006. Effect of medetomidine on respiration and minimum alveolar concentration in halothane- and isoflurane-anesthetized dogs. *Am. J. Vet. Res.*, **67**(5): 782-789

LITTLE JOHN, A. and MITCHELL, B., 1969. Acid base and blood gas studies in horses. Acomparison of capillary and arterial blood samples for the estimation of acid base values in horses., *Res. Vet. Sci.*, **10**:260

MANSELL, P.D. and PARRY, B.W., 1992. Effect of acepromazine, xylazine and thiopentone on factor VIII activity and von Willebrand factor antigen concentration in dogs. *Aust. Vet. J.*, **69**(8):187-190

MARTIN, D.D., TRANQUILLI, W.J., OLSON, W.A., THURMON, J.C. and BENSON, G.J., 1997. Hemodynamic effects of epidural ketamine in isoflurane-anesthetized dogs. *Vet. Surg.*, **26**(6): 505-509

MARTINEZ, E.A., MEALEY, K.L., WOOLDRIDGE, A.A., MERCER, D.E., COOPER, J., SLATER, M.R. and HARTSFIELD, S.M., 1996. Pharmacokinetics, effects on renal function, and potentiation of atracurium-induced neuromuscular blockage after administration of a high dose of gentamicin in isoflurane-anaesthetized dogs. *Am.J.Vet.Res.*, 57(**11**): 1623-1626

MARTINEZ, E.A., WOOLDRIDGE, A.A., HARTSFIELD, S.M. and MEALEY, K.L., 1998. Neuromuscular effects of doxacurium chloride in isoflurane-anesthetized dogs. *Vet. Surg.*, **27**(3): 279-283

MATTHEWS, N.S., SANDERS, E.A., HARTSFIELD, S.M., MERCER, D. and WOOLDRIDGE, A.A., 1995. comparison of 2 pulse oximeters in dogs. *J. Vet. Emergency-and-Critical-Care.*, **5**(2): 116-120

MEYER, R.E., GLEED, R.D. and HARVEY, H.J., 1984. Isoflurane anesthesia as an adjunct to hypothermia for surgery in a dog. *J. Am. Vet. Med. Ass.*, **184**(11): 1387-1389

MUIR, W.W. and HUBBELL, J.A.E., 1988. Cardiopulmonary and anesthetic effects of ketamine and its enantiomers in dogs. *Am. J. Vet. Res.*, **49**(4): 530-534

MUIR, W.W.III. and WIESE, A.J., 2004. Comparison of lactated Ringer's solution and a physiologically balanced 6% hetastarch plasma expander for the treatment of hypotension induced via blood withdrawal in isoflurane-anesthetized dogs. *Am. J. Vet. Res.*, **65**(9): 1189-1194

MUTOH, T., KANAMARU, A., SUZUKI, H., TSUBONE, H., NISHIMURA, R. and SASAKI, N., 2001. Respiratory reflexes in spontaneously breathing anesthetized dogs in response to nasal administration of sevoflurane, isoflurane, or halothane. *Am. J. Vet. Res.*, **62**(3):311-319

MUTOH, T., NISHIMURA, R., KIM, H.Y., MATSUNAGA, S. and SASAKI, N., 1997. Cardiopulmonary effects of sevoflurane, compared with halothane, enflurane, and isoflurane, in dogs. *Am. J. Vet. Res.*, **58**(8):885-890

NAHED SALEH., AOKI, M., SHIMADA, T., AKIYOSHI, H., AMIN, H. and OHASHI, F., 2005. Renal effects of medetomidine in isoflurane-anesthetized dogs with special reference to its diuretic action. *J. Vet. Med. Sci.*, **67**(5): 461-465

NEATH, P.J., BROWN, D.C., HUGHES, D. and PERKOWSKI, S.Z., 2000. The hemodynamic effects of intrathecal oxytocin in normal dogs. *Vet. Surg.*, **29**(3): 272-277

O'BRIEN, J.J., 1966. Halthane as a complete anaesthetic in dog. *Vet. Rec.*, **79**:526-529

OTTO, K.A., 2007. Effects of averaging data series on the electroencephalographic response to noxious visceral stimulation in isoflurane-anaesthetized dogs. *Res. Vet. Sci.*, **83**(3):385-93

PEDERSOLI, W.M., 1980. Serum bromide concentrations during and after halothane anaesthesia in dogs. *Am. J. Vet. Res.*, **11**(2):77-80

PETTIFER, G.R. and HOSGOOD, G., 2004. The effect of inhalant anesthetic and body temperature on peri-anesthetic serum concentrations of transdermally administered fentanyl in dogs. *Vet. Anaesth. and Analg.*, **31**(2): 109-120

POTTIE, R., DART, C., PERKINS, N. and HODGSON, D., 2007. Effect of hypothermia on recovery from general anaesthesia in the dog. *Aust. Vet. J.*, **85**(4):158-162.

POTTIE, RG., DART, CM. and PERKINS, NR. 2008. Speed of induction of anaesthesia in dogs administered halothane, isoflurane, sevoflurane or propofol in a clinical setting. *Aust. Vet. J.*, **86**(1-2):26-31

PYPENDOP, B.H. and ILKIW, J.E., 2005. Pharmacokinetics of ketamine and its metabolite, norketamine, after intravenous administration of a bolus of ketamine to isoflurane-anesthetized dogs. *Am. J. Vet. Res.*, **66**(12): 2034-2038

RAUSER, P., LEXMAULOVA, L., VLASIN, M., FICHTEL, T. and LORENZOVA, J., 2004. Effect of epidural administration of lidocaine, fentanyl and their combination on the minimum alveolar concentration of halothane in dogs. *Vet. Med.*, **49**(11): 421-426

REDONDO, J.I., GOMEZ VILLAMANDOS, R.J., DOMINGUEZ, J.M. and SANTISTEBAN, J.M., 2000. Propofol or thiopentone as induction agents in romifidine-sedated and halothane-N2O-anesthetized dogs: a preliminary study. *Can. J. Vet. Res.*, **64**(4): 249-253

ROBERTSON, S.A., HAUPTMAN,J.G., NACHREINER, R.F. and RICHTER, M. A., 2001. Effects of acetylpromazine or morphine on urine production in halothane-anesthetized dogs. *Am. J. Vet. Res.*, **62**(12): 1922-1927

ROSATI, M., DYSON, D.H., SINCLAIR, M.D. and SEARS, W.C., 2007. Response of hypotensive dogs to dopamine hydrochloride and dobutamine hydrochloride during deep isoflurane anesthesia. *Am. J. Vet. Res.*, **68**(5):483-494

ROSE, R.J., CARTER, R.J. and ILKOW, J.K., 1980. Physiological and biochemical effects of intravenous fluid administration during halothane anaesthesia in dogs. *J. Vet. Pharma. Therap.*, **3**(2):111-119

SAWYER, D.C., GUIKEMA, A.H. and SIEGEL, E.M., 2004. Evaluation of a new oscillometric blood pressure monitor in isoflurane-anesthetized dogs. *Vet. Anaesth. Analg.*, **31**(1): 27-39

SCHWARTZ, A.E., NAVEDO, A.T. and BERMAN, M.F., 1992. Pancuronium increases the duration of electroencephalogram burst suppression in dogs anesthetized with isoflurane. *Anesthesiology.*, **77**(4):686-690

SHARMA, H.N., 1978. Respiratory patterns in anaesthetised dogs during surgery. *Vet.Rec.*, **103**(17):375-377

SILVA, AE., CASTIGLIA, YM., MÓDOLO, NS., ROBERTO, WM., BRAZ, LG., VANE, LA., VIANNA, PT. and BRAZ, JR. 2009. Does the choice of the halogenated anesthetic influence renal function during hemorrhagic shock and resuscitation? *Ren Fail.*, **31**(1):62-69.

SIMEONOVA, G., 2004. Acid-base status and blood gas analysis in three different anaesthesia schemes in dogs. *Turk-Veterinerlik-ve-Hayvanclk-Dergisi.*, **28**(4): 769-774

SIMEONOVA, G.P., TODOROVA, I.I., GADJEVA, V. and DINEV, D.N., 2004. Evaluation of lipid peroxidation associated with three anesthetic protocols in dogs. *Revue-de-Medecine-Veterinaire.*, **155**(12): 602-605

SKARDA, R., 1976. Determination of blood gas and acid base relationships in the horse under halothane anaesthesia with special reference to a comparison of arterial and venous blood. *J.Schweizer.Archiv.Fur.Terheclkride.*, **117**(12):713-730. (*Vet. Bull.*, 47:4011)

SLOAN, M.H., CONARD, P.F., KARSUNKY, P.K. and GROSS, J.B., 1996. Sevoflurane versus isoflurane: induction and recovery characteristics with single-breath inhaled inductions of anesthesia. *Anesthesia & Analgesia.*, **82:** 528-532

SLUIJ, F.J.V., DEVRIES, H.W., DEBRUIJNE, J.J. and VANDESIBROM, W.E., 1983. capillary and venous blood compared with arterial blood in the measurement of acid base. *Am. J. Vet. Res.*, **44**: 459

SNEDECOR, C. W. and COCHRAN, W. G., 1996. *In:* Statistical Analysis. **Eighth** edition.Oxford and IBH publishing co. New Delhi, pp.335-345.

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**(7):1111-1115

SOLANO, A.M., PYPENDOP, B.H., BOSCAN, P.L. and ILKIW, J.E., 2006. Effect of intravenous administration of ketamine on the minimum alveolar concentration of isoflurane in anesthetized dogs. *Am. J. Vet. Res.*, **67**(1): 21-25

SOUSA, M.G., CARARETO, R., DE NARDI, A.B., BRITO, F.L., NUNES, N. and CAMACHO, A.A., 2007. Effects of isoflurane on Tei-index of myocardial performance in healthy dogs. *Can. Vet. J.*, **48**(3):277-282

STEAGALL P.V., TEIXEIRA NETO F. J., MINTO B.W., CAMPAGNOL D and CORREA., 2006.Evaluation of the isoflurane-sparing effects of lidocaine and fentanyl during surgery in dogs. *J Am Vet Med Assoc.*, **229** (4) :522-527

STEFFEY, E.P. and HOWLAND, D., 1978. Potency of enflurane in dogs: comparison with halothane and isoflurane. *Am. J. Vet. Res.*, **39**(4): 573-577

STEFFEY, E.P. WOLINER, M.J. and HOWLAND, D., 1983. Accuracy of isoflurane delivery by halothane-specific vaporizers. *Am. J. Vet. Res.*, **44**(6):1072-1078

STEFFEY, E.P., GILLESPIE, J.R., BERRY, J.D., EGER, E.I. and RHODE, E.A., 1974. Circulatory effects of halothane and halothane nitrous oxide anaesthesia in dog controlled ventilation. *Am.J.Vet.Res.*, 35(**10**):1289-1293

STEFFEY, E.P., GILLESPIE, J.R., BERRY, J.D., EGER, E.I. and SCHOLLM, O.W., 1976. Effect of halothane and halothane nitrous oxide on haematocrit and protein concentration in dog and monkey. *Am. J. Vet. Res.*, **37**(8):959-962

STEKIEL, T.A., STEKIEL, W.J., TOMINAGA, M, STADNICKA, A, BOSNJAK, Z.J. and KAMPINE, J.P. 1995. Isoflurane-mediated inhibition of the constriction of mesenteric capacitance veins and related circulatory responses to acute graded hypoxic hypoxia. *Anesthe.Analg.*, **80**:994-1001

TACKE, S., XIONG, H. and SCHIMKE, E., 1998. Sevoflurane (SEVOrane) as an inhalation anesthetic in dogs in comparison with halothane and isoflurane. *Tierarztl. Prax. Ausg. K. Klientiere. Heimtiere.*, **26**(6):369-377

TAYLOR, P.M. and HALL, L.W., 1985. Clinical anaesthesia in the horse comparison of enflurane and halothane., *Equine. Vet. J.*, **17**(1):51-57. (Vet. Bull., **55:**3168)

TEIXEIRA NETO, F.J., LUNA, S.P., CRUZ, M.L., BRAZ, J.R., MASSONE, F. and NOGUEIRA, C.S., 2007. A Study of the effect of hemorrhage on the cardiorespiratory actions of halothane, isoflurane and sevoflurane in the dog. *Vet. Anaesth. Analg.*, **34**(2):107-116

TEIXEIRA NETO, F.J., MASSONE, F., LUNA, S.P.L., CAMACHO, A.A., S JUNIOR. and ISHIY, H.M., 2001. The effects of atropine and methotrimeprazine on the epinephrine-induced arrhythmias in halothane-anesthetized dogs. *Can. J. Vet. Res.*, **65**(2): 116-124

TOBATA, D., TAKAO, K., MOCHIZUKI, M., NISHIMURA, R. and SASAKI, N., 2004. Effects of dopamine, dobutamine, amrinone and milrinone on regional blood flow in isoflurane anesthetized dogs. *J. Vet. Med. Sci.*, **66**(9): 1097-1105

TOPAL, A., GUL, N. and GORGUL, O.S., 2004. Comparison of end-tidal CO2 and arterial oxygen saturation during halothane, isoflurane or sevoflurane anaesthesia in dogs. *Indian. Vet. J.*, **81**(12): 1345-1347

TOPAL, A., GUL, N., ILCOL, Y. and GORGUL, O.S., 2003. Hepatic effects of halothane, isoflurane or sevoflurane anaesthesia in dogs. *J. Vet. Med. Series.*, **50**(10): 530-533

TORSKE, K.E., DYSON, D.H. and CONLON, P.D., 1999. Cardiovascular effects of epidurally administered oxymorphone and an oxymorphone-bupivacaine combination in halothane-anesthetized dogs. *Am. J. Vet. Res.*, **60**(2): 194-200

TRANQUILLI, W.J., THURMON, J.C. and BENSON, G.J., 1988. Alterations in epinephrine-induced arrhythmogenesis after xylazine and subsequent yohimbine administration in isoflurane-anesthetized dogs. *Am. J. Vet. Res.*, **49**(7):1072-1072

TRANQUILLI. W.J., THURMON, J.C., BENSON, G.J. and DAVIS, L.E., 1986. Alteration in the arrhythmogenic dose of epinephrine (ADE) following xylazine administration to halothane-anesthetized dogs. *J. Vet. Pharma. Therap.*, **9**(2):198-203

WARD, D.G., BLYDE, D., LEMON, J. and JOHNSTON, S., 2006. Anesthesia of captive African wild dogs (Lycaon pictus) using a medetomidine-ketamine-atropine combination. *J. Zoo. Wildlife. Med.*, **37**(2):160-164

WERNER, R.E., 1987. Isoflurane anesthesia: a guide for practitioners. *Compendium-on-Continuing-Education-for-the-Practicing-Veterinarian*. **9**(6):603-606

WIERSIG, D.O., DAVIS, R.H. and SZABUNIEWICZ, M., 1974. .Prevention of induced ventricular fibrillation in dogs anaesthetised with ultra short acting barbiturates and halothane. *J. Am. Vet. Med. Ass.*, **165**(4):341-345 (*Vet.Bull.*, **45**:2831)

WILSON, D. V., EVANS, A. T., CARPENTER, R. E. and MULLINEAUX, D. R., 2004. The effect of four anaesthetic protocols on splenic size of dogs. *Vet. Anaesth. Analg.*, **31**(2): 109-120

WRIGHT, K.N., HINES, D.A. and BRIGHT, J.M., 1996. Cardiac electro physiologic measurements in dogs before and after intravenous administration of atropine and propranolol. *Am. J. Vet. Res.*, **57**(12): 1695-1701

YANG, HANSEOK., KWEON, OHKYEONG., WOO, HEUNGMYEONG. and NAM, TCHICHU., 1999. Anaesthetic and cardiovascular effects of xylazine-fentanyl-azaperone and medetomidine-midazolam preanesthetic medication. Combination with their antagonists in halothane-anaesthetized dogs. *Korean. J. Vet. Res.*, **39**(3): 616-627

ZBINDEN, A.M., THOMSON, D.A., WESTENSKOW, D.R., FREI, F. and MAERTENS, J., 1988. Anaesthetic uptake and elimination: is there a difference between halothane and isoflurane in the dog Anaesthetic uptake and elimination: is there a difference between halothane and isoflurane in the dog? *Br. J. Anaesthe.*, **60**(4):395-401

VIII ABSTRACT

A comparative evaluation of halothane and isoflurane anaesthesia for ovariohysterectomy in bitches was studied. During the present study the following parameters were assessed: (i) rectal temperature, respiratory rate and heart rate, (ii) haematological and biochemical parameters *Viz.*, total erythrocyte count (TEC), packed cell volume (PCV), hemoglobin concentration (Hb), total leukocyte count (TLC), differential leukocyte count (DLC), platelet count(PLT), alanine aminotransferase (ALT), aspertate amino transferase (AST), alkaline phosphatase (ALP), plasma creatinine, total plasma protein (TPP), electrolytes (sodium, potassium and chloride) and blood glucose and quantitative parameters such as average time for return of swallowing reflex, pedal reflex, head righting reflex, time taken for voluntary leg movement, time taken for sternal recumbency and time taken for animal to ambulate after cessation of anaesthesia during recovery period and also, qualitative parameters including: start position, position change, end position, head position, ear position, eye position, tail position, vocal and others (arched back, stretching, rigid back, lip licking, leg up position) were assessed based on video recordings obtained during recovery phase of anaesthesia. Results revealed, in both the Groups, there were non-significant minor alterations in clinical, haematological and biochemical parameters except in halothane Group, where ALT levels were increased significantly. In conclusion, isoflurane anaesthesia was comparatively advantageous over halothane anaesthesia in terms of ease of administration, maintenance and recovery with minimal adverse effects on physiological status of bitches subjected for ovariohysterectomy procedure.