# EFFICACY OF SEVOFLURANE FOR MAINTENANCE OF PROPOFOL ANAESTHESIA IN DOGS


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**Thesis submitted in partial fulfilment of the requirement for the degree of**

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**Faculty of Veterinary and Animal Sciences**
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**COLLEGE OF VETERINARY AND ANIMAL SCIENCES**
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# DECLARATION

I hereby declare that this thesis entitled **"EFFICACY OF SEVOFLURANE FOR MAINTENANCE OF PROPOFOL ANAESTHESIA IN DOGS"** is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title of any other University or Society.

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**K.S. RAGHAVAN**

# CERTIFICATE

Certified that the thesis, entitled **"EFFICACY OF SEVOFLURANE FOR MAINTENANCE OF PROPOFOL ANAESTHESIA IN DOGS"** is a record of research work done independently by Sri. **K.S. RAGHAVAN,** (2008-13-142) under my guidance and supervision and it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to him.

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*Dedicated to*

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# *Introduction*

# 1. INTRODUCTION

Surgery when necessitates to a compromised animal, choice of anaesthesia will be the foremost concern for the surgeon. In such instances the selection of anaesthetic agents, route and method of administration to produce a balanced anaesthesia is a challenge.

Advances in veterinary anaesthesiology derived from adoption of human anaesthetic agents, equipment and techniques, which began with the use of ether and nitrous oxide to the recently developed agents like sevoflurane and desflurane. A variety of injectable and inhalant anaesthetic agents are preferred in veterinary practice. Even though the injectable anaesthetics are an option in veterinary practice due to its advantages in administration, the merit of inhalant anaesthetics can not be ignored in prolonged surgery and that too in compromised patients. Maintenance of anaesthesia with inhalant agents is accepted because of their predictability of effects, the quality of the anaesthesia produced, and ability with modern equipment to accurately titrate the dose delivered (Pottie *et al.*, 2008).

In clinical practice the anaesthetist choose a balanced anaesthesia with the goal of patient safety, comfort and a quiet surgical field. Employment of modern anaesthesia monitoring systems creates a visual aid recorded over time that assists in determining the change in patient status during the anaesthetic period.

Rapid loss of consciousness and easy endotracheal intubation are the advantages to an intravenous method of induction. Proper sedation can enhance the quality and rapidity of induction. Premedication also prevents emergence delirium (Pottie *et al.*, 2008).

Glycopyrrolate is a quaternary nitrogen anticholinergic agent, having potent antimuscarinic activity, prolonged action and less effective penetration through blood brain and placental barriers than atropine.

Xylazine hydrochloride, a sedative analgesic, is an alpha 2 adrenoceptor agonist, produces muscle relaxation and anxiolysis. It is being safely used as a preanaesthetic agent in dogs.

Propofol is a sedative hypnotic drug with anaesthetic properties, producing short duration of anaesthesia without many side effects. Propofol is preferred to a volatile agent for a faster induction of anaesthesia (Pottie *et al.*, 2008), reduced chance of regurgitation and aspiration of stomach contents (Love *et al.*, 2007).

Sevoflurane is a noninflammable halogenated inhalant anaesthetic. It is less potent than the currently used inhalation agents like halothane and isoflurane in dogs, which has a minimum alveolar concentration (MAC) value of 2.36 per cent in dogs against 0.86 per cent for halothane and 1.28 per cent for isoflurane (Steffey, 1996). The main advantage of sevoflurane over halothane and isoflurane is its superior pharmacokinetic properties including low solubility in blood and tissues resulting in a faster induction and recovery characteristics (Haitjema and Cullen, 2001). The advantage of rapid kinetics such as improved control of depth of anaesthesia and ease of maintaining stable level of anaesthesia is approved for all species (Clarke, 1999).

There is a need to titrate drug dose against patient response, which is influenced by individual and species differences in drug kinetics and dynamics, disease status and concurrent therapy. Many compounding factors such as disease, breed and temperament of animal, environment and imposition of surgery inevitably affect the way of drug behaviour. So it is essential that the observations under real conditions encountered during everyday clinical veterinary practice be reputed. The glycopyrrolate, xylazine, propofol and sevoflurane combination will be a worthful anaesthetic regimen and reports on trials with this combination in clinical settings are scanty.

Hence the present study was undertaken with the objective to evaluate the efficacy of sevoflurane for maintaining the propofol induced anaesthesia in dogs.

# *Review of Literature*

# 2. REVIEW OF LITERATURE

## 2.1 DRUGS USED FOR THE STUDY

### 2.1.1 Glycopyrrolate

Proakis and Harris (1978) reported that glycopyrrolate, a muscarinic receptor antagonist, had the advantage of not crossing the blood brain barrier and placental barrier, resulting in decreased likelihood of central nervous system and foetal effects.

Short (1987) determined the effective preanaesthetic dose of glycopyrrolate in dog as 0.011 mg/kg body weight. Glycopyrrolate was superior to atropine in reducing gastric acidity and consequent Mendelson's syndrome, in human beings.

Jacobson *et al.* (1994) administered glycopyrrolate intravenously at the dose rate of 0.01 mg/kg body weight as a premedication 11 ± 2 min prior to butorphanol (0.2 mg/kg body weight) and xylazine (0.5 mg/kg body weight).

Adams (1995) recommended preanaesthetic dose of 0.01 mg/kg body weight glycopyrrolate parenterally in dogs. Glycopyrrolate in dogs could effectively diminish the volume and acidity of gastric secretions.

Dyson and Davies (1999) used glycopyrrolate effectively to treat bradycardia in anaesthetised dogs, and reported that dogs with less than 10 kg body weight were more resistant to the effects of glycopyrrolate and required a higher dose than the standard (0.01 mg/kg body weight i/v).

Lemke (2001) in a study in healthy dogs used intramuscular administration of glycopyrrolate at a dose of 0.01 mg/kg body weight, 15 min prior to administration of alpha-2 agonist romifidine.

Reyntijens *et al.* (2005) observed that glycopyrrolate at the rate of 0.06 mg/kg body weight administered as intravenous bolus during sevoflurane-remifentanil based anaesthesia in children prevented bradycardia.

Woods and Sladen (2009) opined that preoperative administration of glycopyrrolate should be considered as it could dry secretions and suppress upper airway vagal responses.

### 2.1.2 Xylazine

Raghavan *et al.* (1979) suggested the optimal preanaesthetic dosage of xylazine in dog as 1 mg/kg body weight and used xylazine two per cent solution subcutaneously 20 min before the administration of thiopentone sodium.

Knight (1980) reported that the analgesia and sedation produced by xylazine were not dose dependent and lasted only about 20 min with the analgesic effect being minimal in the extremities.

### 2.1.3 Propofol

The dose of propofol at the rate of 6-7 mg/kg body weight intravenously for induction of anaesthesia could be reduced to 50-75% upon premedication with xylazine at the rate of 1 mg/kg body weight intravenously (Short and Bufalari, 1999).

Sooryadas (2001) evaluated xylazine–propofol anaesthesia in atropine premedicated healthy and compromised dogs and reported that the combination was safe for surgery in both cases.

Kojima *et al.* (2002) observed a significant reduction in the induction dose of propofol up to 2.70 ± 7.00 mg/kg body weight upon premedication with 0.02 mg/kg body weight medetomidine and 0.30 mg/kg body weight midazolam. The degree of dose reduction and recovery time after induction with propofol was related to the depth of sedation induced by preanaesthetic medication.

Mohan (2006) studied the efficacy of glycopyrrolate and xylazine premedication on propofol anaesthesia in dogs. It could produce good muscle relaxation for caesarean section and reported 29 out of 38 puppies delivered were live and active.

Love *et al.* (2007) in a clinical study in dogs used 3.50 ± 1.00 mg/kg body weight propofol for induction of anaesthesia under acepromazine (0.03 mg/kg body weight) and pethidine (3 mg/kg body weight) pre medication.

Bras *et al.* (2008) observed an induction dose of propofol intravenous bolus injection as 6 mg/kg body weight upon premedication of dogs with morphine sulphate (0.50 mg/kg body weight) and acepromazine (0.03 mg/kg body weight) intramuscular.

According to Pottie *et al.* (2008) premedication with acepromazine and morphine could reduce the dose requirement of propofol for induction of anaesthesia in dogs. The dose of propofol required without premedication was 4.32 ± 0.61mg/kg body weight and it was 2.68 ± 0.24 mg/kg body weight with premedication.

Ramankutty (2008) evaluated and recommended the propofol - isoflurane anaesthesia with glycopyrrolate-xylazine premedication in healthy and compromised dogs as safe regimen.

### 2.1.4 Sevoflurane

According to Patel and Goa (1996) biotransformation of sevoflurane was dose independent and 1 to 5% of the absorbed dose was metabolised as against 0.2% for isoflurane.

Haitjema and Cullen (2001) reported that sevoflurane is less potent inhalant than halothane or isoflurane and hence a higher concentration was required for anaesthetic use. The low solubility of sevoflurane allowed rapid

equilibration between alveolar gas and blood to achieve a quick stable concentration.

The combination of alpha-2 agonist medetomidine, propofol and sevoflurane was effective for inducing and maintaining general anaesthesia in healthy cats. The cats breathed spontaneously. Sevoflurane in oxygen at a fresh gas flow of 200 ml/kg/min was delivered using a Bain breathing circuit. The vaporiser setting was adjusted to 3 to 4%, which was 1 to 3 times the MAC of sevoflurane for cats (Selmi *et al.,* 2005).

Kutter *et al.* (2006) maintained anaesthesia in healthy goat and sheep with approximately 1 MAC sevoflurane (2.70 ± 0.30% for goats; 3.30 ± 0.30% for sheep). Animals were premedicated with alpha- 2 agonist dexmedetomidine and ventilated mechanically. Here the heart rate, cardiac output and arterial oxygen tension decreased significantly.

Gomez-Villamandos *et al.* (2007) maintained anaesthesia with sevoflurane in Iberian lynx (*Lynx pardinus*) premedicated with medetomidine.

According to Steffy (1996) anaesthetic recovery from an inhalant agent depend not only on blood/ gas solubility, but on alveolar ventilation, cardiac output, and venous-to-alveolar anaesthetic partial pressure differences. An increase in cardiac output or decrease in ventilation would prolong recovery time.

Mutoh *et al.* (1997) opined that spontaneous ventilation was preferable as long as animals ventilate satisfactorily under the influence anaesthetics. Controlled ventilation found to reduce venous return and lower cardiac output. High $PaCO_2$ induced by spontaneous ventilation could stimulate sympathetic nerves and compensate for cardiovascular system depression.

A moderate increase in $PaCO_2$ had certain beneficial effects on the cardiovascular depression, where by the increased $PaCO_2$ was a potential

stimulator of the sympathetic nervous system and compensate for cardiovascular system depression (Mutoh *et al.*, 1997 and Polis *et al.*, 2001).

Polis *et al.* (2001) studied the influence of ventilation mode on cardiopulmonary parameters in sevoflurane anaesthetised dogs and observed that sevoflurane anaesthesia at 1.5 MAC could be safely used in premedicated healthy dogs but induced a relatively moderate cardiopulmonary depression during spontaneous and controlled ventilation. It was also observed that spontaneous ventilation mostly used during clinical anaesthesia was often accompanied by hypoventilation, which will increase the $PaCO_2$.

Boller *et al.* (2005) reported that the cardiac output was a major determinant of inhalation agent uptake rate. Authors reported no significant difference in volatile anaesthetic uptake between spontaneously breathing and mechanically ventilated subjects. However the rise in alveolar concentration towards inspired concentrations was more rapid when ventilation was increased. Increased ventilation affected drug wash into the functional residual capacity rather than anaesthetic uptake.

Kutter *et al.* (2006) maintained anaesthesia in healthy goat and sheep with sevoflurane that ventilated mechanically.

Laredo *et al.* (2009) in a study opined that increasing the depth of anaesthesia in spontaneous ventilation could depress the rate of ventilation and limited the uptake of anaesthetic vapour which otherwise with mechanical ventilation might lead to over dosage.

## 2.2 CLINICAL SIGNS

### 2.2.1 Signs of Sedation

According to Klide *et al.* (1975) xylazine at the dose rate of 2.2 mg/kg body weight could induce sedation within five min after intravenous administration and 10 min after intramuscular administration which lasted

approximately 20 min after intravenous and 30 min after intramuscular administration. Subjective sedative effects like lying down, medial rotation of eyeball and prolapse of nictitans with some degree of analgesia were also observed following the administration of xylazine.

The common clinical signs of sedation observed following xylazine premedication in atropinised dogs were winking of eyes, yawning, incoordination of movement and assumption of sternal recumbency with head down posture (Rajankutty, 1996).

Kandapal *et al.* (2005) studied clinico-physiological effects of xylazine in dogs and observed that the pedal reflex remained unaffected after xylazine administration.

The common clinical signs of sedation reported after premedication with glycopyrrolate-xylazine in healthy and compromised dogs were winking of the eyes, licking, scanty salivation, incoordination of movements with lowering of head and sitting on haunches followed by sternal recumbency with head down posture within 15 min (Mohan, 2006; Narayanan, 2007 and Ramankutty, 2008).

### 2.2.2 Signs of Induction Anaesthesia

Sooryadas (2001) reported sluggish palpebral reflex and ventromedial rotation of eyeballs during induction with propofol in healthy and compromised dogs premedicated with atropine and xylazine

Venugopal *et al.* (2002) observed that propofol-ketamine anaesthesia with or without premedication in dogs effectively abolished jaw tone during induction.

Kuusela *et al.* (2003) compared propofol infusion and propofol-isoflurane anaesthesia in dogs and reported that propofol infusion achieved a lighter anaesthesia than with isoflurane, so also a less decrease in palpebral reflexes with occasional distinct spontaneous blinking. Palpebral reflexes were rarely seen in isoflurane anaesthesia.

Easy endotracheal intubation was reported with administration of propofol in dogs premedicated with glycopyrrolate-xylazine combination (Mohan, 2006 and Ramankutty, 2008).

According to Pottie *et al.* (2008) induction with propofol (1.43 ± 0.13 min) was significantly faster in dogs than induction using halothane, isoflurane and sevoflurane. Authors also observed that premedication with acepromazine and morphine could further decrease the induction time.

**2.2.3 Time for Induction of Anaesthesia**

Branson *et al.* (2001) reported longer induction time of anaesthesia with sevoflurane (7.30 ± 3.10 min) than with injectable agents, propofol and thiopental (1.10± 0.40 min).

Sooryadas (2001) reported an induction time of anaesthesia as 4.20 ± 1.08 min in healthy dogs and 3.21 ± 0.83 min in compromised dogs premedicated with atropine @ 0.04 mg/kg body weight and xylazine @ 1 mg/kg body weight.

Venugopal *et al.* (2002) reported an induction time of anaesthesia as 4.86±0.006 min following intravenous administration of propofol-ketamine in dogs.

Mohan (2006) recorded an induction time of anaesthesia as 2.23 ± 1.04 min with propofol in dogs under glycopyrrolate- xylazine premedication.

Narayanan (2007) reported induction time of anaesthesia as 6.50 ± 0.43 min to 9.50 ± 0.72 min with ketamine in dogs premedicated with various combinations of glycopyrrolate, xylazine and midazolam.

Easy endotracheal intubation was reported following intravenous administration of propofol in dogs with glycopyrrolate-xylazine premedication (Mohan, 2006 and Ramankutty, 2008).

Pottie *et al.* (2008) reported an induction time of anaesthesia as 1.84 ± 0.21 min with propofol, 3.29 ± 0.24 min with sevoflurane, 2.86±0.25 min with isoflurane and 3.71 ± 0.22 min with halothane in acepromazine-morphine premedicated healthy dogs. Quality of induction was better with propofol than with inhalant agents.

Ramankutty (2008) reported the time of induction of anaesthesia as 1.11 ± 0.25 min and 0.83 ± 0.15 min respectively in healthy and compromised dogs under the propofol induced anaesthesia with glycopyrrolate-xylazine premedication.

### 2.2.4 Signs of Anaesthesia

The depth of anaethesia might have to be varied on the basis of the degree of stimulation and changing physiological conditions so as to balance adequate anaesthesia – analgesia and physiologic function (Soma and Klide, 1987).

Branson *et al.* (2001) worked out subjective scoring criteria in an anaesthetic study with 196 dogs to assess the quality of induction, maintenance, recovery and graded as poor, fair, good and excellent (Annexure III).

Sooryadas (2001) reported sluggish palpebral reflex and rolled down eye ball during induction and throughout the period of maintenance as salient ocular reflexes with xylazine-propofol anaesthesia in atropine premedicated healthy and compromised dogs. The duration of anaesthesia after induction reported was 14.03 ± 2.04 min in healthy dogs (with 5.09 ± 0.59 mg/kg body weight propofol) and 18.54 ± 1.64 min in compromised dogs (5.04 ± 0.49 mg/kg body weight propofol).

Bennet *et al.* (2008) in a study with sevoflurane in dogs determined the adequacy of anaesthesia by assessing jaw tone and eye position.

Ramankutty (2008) reported satisfactory depth of anaesthesia and excellent degree of muscle relaxation with propofol-isoflurane anaesthesia after glycopyrrolate-xylazine premedication in healthy and compromised dogs subjected to different surgical interventions.

Lozano *et al.* (2009) in an anaesthetic study categorised the level of sedation as not very sedated, slightly sedated, sedated and deeply/very sedated (Annexure II).

### 2.2.5 Recovery Time

Anaesthetic recovery depended not only on blood/ gas solubility, but on alveolar ventilation, cardiac output, and venous-to-alveolar anaesthetic partial pressure differences (Steffy, 1996).

Clarke (1999) reported that the solubility of sevoflurane in many tissues, particularly in fat was higher compared to isoflurane or desflurane (Fat blood partition coefficient: sevoflurane- 48, isoflurane- 45, desflurane- 27) so that there could be comparatively a chance for a slower anaesthetic recovery particularly in prolonged anaesthesia.

Branson *et al.* (2001) reported .the over all mean time from extubation to sternal recumbency as 10 ± 15 min during sevoflurane anaesthesia maintenance in dogs.

Haitjema and Cullen (2001) evaluated the surgical procedures on ASA status II dogs, maintained on sevoflurane anaesthesia after intravenous induction with thiopentone or diazepam with ketamine observed that the palpebral reflex returned within five min (ranged from 2 to 11min) in 68% of dogs; dogs extubated in three to 15 min (which occurred within 5 min of the return of the palpebral reflex) after discontinuing sevoflurane maintenance. 68% of dogs were able to lift their head within five min (ranged from 1-10 min) of being extubated. It was concluded that irrespective of various induction regimens, two – third of

the dogs studied reached sternal recumbency within 10 min (ranged from 1-20 min).

Polis *et al.* (2001) opined that emergence time in propofol induced inhalation anaesthesia was minimally influenced by propofol. High metabolic clearance rate and fast redistribution made plasma levels of propofol decline rapidly.

Polis *et al.* (2001) studied influence of halothane, isoflurane and sevoflurane in oxygen on clinical anaesthesia in healthy mongrel dogs and reported that sevoflurane anaesthesia indicated shortest emergence.

Sooryadas (2001) reported a recovery time of 14.72 min in healthy dogs and 20.9 min in compromised dogs with propofol anaesthesia.

Kojima *et al.* (2002) reported that the recovery time after induction with propofol was related to the depth of sedation induced by preanaesthetic medication.

According to Boller (2005) cardiac output contributed a major determinant in inhalation agent uptake.

Sahay and Dass (2005) evaluated effect of propofol in atropinised goat and observed smooth recovery and shorter recovery time.

Mohan (2006) reported that recovery time with-propofol anaesthesia in dogs under glycopyrrolate - xylazine premedication was 17.66 ± 1.81 min

Love *et al.* (2007) conducted a clinical study in dogs with sevoflurane anaesthesia. The mean time from the discontinuation of sevoflurane administration to extubation was 5.40 min, to head lifted was 8.40 min, position to sternal recumbency was 11.40 min and to standing posture was 24.40 min. It was opined that differences in depth of anaesthesia at the end of the procedures might have contributed to the variability in the recovery time.

Bennet *et al.* (2008) studied sevoflurane maintained anaesthesia in dogs undergoing elective surgeries and reported no relationship between duration of anaesthesia and the recovery time. They observed a head lift time of 18 ± 16 min, sternal recumbency time of 28 ± 22 min, and standing time of 48 ± 32 min

Jadon *et al.* (2008) maintained anaesthesia in atropine-diazepam premedicated puppies for 30-40 min using 2.5% sevoflurane and reported a recovery time of 9.20 ± 2.89 min

Mohamadnia *et al.* (2008) studied the recovery characteristics of sevoflurane maintained anaesthesia in sheep and reported post anaesthetic time to first swallow as 3.62 ± 0.98 min and ability to maintain head lifted for five min as 38.8 ± 16.6 min

Ramankutty (2008) reported mean recovery time of 27.50 min in healthy dogs maintained on 3% isoflurane, premedicated with glycopyrrolate-xylazine and induced with propofol. The significant difference of recovery time could not be observed in compromised dogs.

Topal (2008) evaluated sevoflurane and isoflurane anaesthesia in xylazine premedicated rabbits and reported significantly early recovery time with sevoflurane compared to isoflurane.

Lozano *et al.* (2009) compared the recovery time in dogs maintained under sevoflurane anaesthesia with isoflurane and desflurane. The observations recorded with sevoflurane anaesthesia were median time to extubation as seven min, (interquartile range (IQR) 5-7 min), median time to sternal recumbency as 9.5 min (IQR 7.25-11.75), five dogs attained standing position in less than 20 min, four dogs between 20 to 45 min and two dogs in more than 45 min

### 2.2.6 Recovery Quality

Haitjema and Cullen (2001) reported that, in their clinical experience with dogs of ASA status II, maintained on sevoflurane anaesthesia resulted excellent to good grades in the recovery quality.

Love *et al.* (2007) conducted a clinical study in dogs and observed significantly better quality of recovery with sevoflurane when compared to isoflurane.

Jadon *et al.* (2008) maintained anaesthesia in atropine - diazepam premedicated puppies for 30-40 min using 2.5% sevoflurane and reported a smooth recovery.

### 2.2.7 Undesirable Side Effects

Muir III and Gadawski (1998) reported respiratory depression and apnoea as the most likely adverse effects following intravenous bolus administration of propofol in dogs.

Clarke (1999) stated that in human, there was a need for adequate analgesia under sevoflurane anaesthesia as 32% patients had the major side effect of nausea and vomiting.

Branson *et al.* (2001) studied sevoflurane anaesthesia maintenance in 196 dogs and reported that hypotension, tachypnoea and apnoea were the most common undesirable side effects.

Haitjema and Cullen (2001) in their clinical experience in various surgical interventions in 22 dogs on ASA status II, maintained on sevoflurane anaesthesia after intravenous induction with thiopentone or diazepam with ketamine, resulted shivering in one dog, tachypnoea in two dogs and sneezing during recovery in one dog as adverse reactions.

Kojima *et al.* (2002) administered propofol as slow intravenous injection (>60 seconds), which contributed to stable respiratory conditions and could avoid undesirable side effects.

Kuusela *et al.* (2003) observed lower adrenaline concentration after propofol infusion at the end of the recovery period.

Sahay and Dass (2005) evaluated effect of propofol in atropinised goats and observed transient apnoea in 50% animals.

Kale *et al.* (2006) could reduce the incidence of apnoea by slow intravenous injection of propofol over a period of 30-40 seconds.

Wilson *et al.* (2006) studied the influence of sevoflurane on gastro-oesophageal reflux (GER) during anaesthesia in dogs and observed that there was a similar risk of GER as with halothane and isoflurane. Increased gastric acidity could also result in an increase in incidence of GER.

Love *et al.* (2007) in a clinical study in dogs preferred propofol to a volatile agent for induction in order to reduce chance of undesirable side effects like regurgitation and aspiration of stomach contents.

Sharma and Bhargava (2007) observed apnoea as a constant finding with propofol general anaesthesia in dogs premedicated with atropine and triflupromazine hydrochloride.

Bennet *et al.* (2008) administered medication to supplement analgesia during sevoflurane maintained anaesthesia in all dogs and also in post operative periods in most of the dogs.

Mohamadnia *et al.* (2008) studied the recovery characteristics of sevoflurane maintained anaesthesia in sheep and suggested pain as a possible cause for the disturbed recovery.

Pottie *et al.* (2008) administered meloxicam at the rate of 0.2 mg s/c for analgesia before the end of sevoflurane anaesthesia in dogs.

Ramankutty (2008) reported decreased respiratory rate and temporary apnoea as undesirable side effect up on induction of anaesthesia with intravenous rapid bolus administration of propofol in glycopyrrolate-xylazine premedicated dogs.

Yamashita *et al.* (2008) used carprofen at the rate of 4 mg/kg body weight subcutaneously (s/c) or meloxicam at the rate of 0.2 mg/kg body weight s/c, along with butorphanol 0.3 mg/kg body weight i/m one hour prior to induction of anaesthesia for preemptive analgesia in sevoflurane anaesthesia of dogs.

Lozano *et al.* (2009) studied recovery quality in 12 dogs maintained under sevoflurane anaesthesia and recorded smooth recovery except for one dog which had an unacceptable recovery signs such as vocalisation and seizure. The appearance of seizure activity could have resulted from its neurological condition and/or could have been potentiated by the inhalation agent.

### 2.2.8 Sevoflurane Vaporiser Concentrations

Branson *et al.* (2001) studied sevoflurane anaesthesia maintenance in 196 dogs and reported that the over all mean vaporiser setting at the beginning of maintenance was 3.6 per cent where as the over all mean vapouriser setting was 3.3 per cent.

Haitjema and Cullen (2001) in their clinical experience in various surgical procedures in 22 dogs on American Society of Anaesthesiologists (ASA) status II, maintained on sevoflurane anaesthesia after intravenous induction with thiopentone or diazepam with ketamine, observed that most dogs were initially maintained on a vapouriser setting between 3 to 7 per cent sevoflurane. They could reduce vapouriser setting to 3 per cent or less after 5 to 6 min of sevoflurane maintained anaesthesia. It was found that after 30 min of anaesthesia

maintained between 1 and 3 per cent sevoflurane on Bain's circuit 75% dogs required only less than 2.5 per cent sevoflurane.

Mendes *et al.* (2003) maintained cats for ovariohysterectomy under sevoflurane anaesthesia with 3.4% sevoflurane (1.3 MAC in cats) in oxygen at flow rate of 200 ml/kg/min delivered using a Bain's breathing circuit.

Gomez-Villamandos *et al.* (2007) maintained anaesthesia with sevoflurane in Iberian lynx (*Lynx pardinus*) using 5% sevoflurane in oxygen for induction and maintained anaesthesia with 2.80% sevoflurane to a plane of anaesthesia sufficient to avoid movements or changes in heart rate, respiratory rate or blood pressure.

A clinical study conducted by Love *et al.* (2007) in dogs, used 3-3.5% sevoflurane in 100% oxygen delivered via a non rebreathing system to increase the depth of anaesthesia and between vaporiser setting of 2-2.5% to maintain a light plane of anaesthesia. Vaporiser setting was fixed to 2% towards the end of anaesthesia.

Mutoh (2007) in a study in dogs used high inspired concentration of 4.8% sevoflurane in 100% oxygen at a flow rate of four liters per min for mask induction. The higher concentration of sevoflurane was used to speed the rate of equilibration in the alveoli.

Bennet *et al.* (2008) reported that there was no constant relationship between animal size and vapouriser setting. The fresh gas flow and anaesthetic concentration were individually adjusted for each dog.

Jadon *et al.* (2008) maintained anaesthesia in atropine-diazepam premedicated puppies for 30-40 min using 2.5% sevoflurane.

Topal (2008) used sevoflurane anaesthesia in xylazine premedicated rabbits and reported that induction of anaesthesia with vapouriser setting

of 6% sevoflurane and maintenance with a vapouriser setting of 3% sevoflurane (MAC = 3.70%).

Yamishita *et al.* (2009) observed that the sevoflurane MAC in older dogs (1.86 ± 0.29%) was significantly less than that for the younger dogs (2.25 ± 0.15%).

Singh *et al.* (2010) maintained surgical anaesthesia in critically ill canine patients with 3-3.5% sevoflurane.

## 2.3 PHYSIOLOGICAL PARAMETERS

### 2.3.1 Rectal Temperature, Pulse Rate and Respiratory Rate

#### *2.3.1.1 Premedication and Induction*

Jacobson *et al.* (1994) observed that administration of xylazine in glycopyrrolate premedicated dogs could cause significant decrease in breathing rate.

Muir III and Gadawski (1998) reported decrease in rectal temperature on intravenous administration of propofol in dogs. The authors attributed it to a decrease in shivering threshold and impaired thermoregulatory control. It was reported that increasing the dosage of propofol resulted in greater decrease in respiratory rate and slower recovery of respiratory rate to pre anaesthetic values.

Redondo *et al.* (1999) studied the effect of propofol in atropine-xylazine premedicated dogs and differences from basal values in rectal temperature were not reported after premedication. Authors reported decrease in respiratory rate after premedication. Increased pulse rate and lower respiratory rates were reported after induction.

Sooryadas (2001) evaluated xylazine–propofol anaesthesia in atropine premedicated healthy and compromised dogs and reported significant decrease in respiratory rate after premedication and induction.

Venugopal *et al.* (2002) observed significant decrease in rectal temperature and respiratory rate, and significant increase in pulse rate after propofol-ketamine anaesthesia with or without triflupromazine/diazepam premedication in dogs.

Kandapal *et al.* (2005) studied clinico-physiological effects of xylazine in dogs and reported a significant reduction in rectal temperature, which was attributed to decreased metabolic rate, muscle relaxation and central nervous system depression. The significant reduction in respiratory rate was also reported, which was attributed to direct depressant action on central nervous system

Sahay and Dass (2005) observed non significant decrease in rectal temperature with propofol administration in atropinised goats.

Kale *et al.* (2006) observed significant decrease in rectal temperature and significant decrease in respiratory rate in dogs with propofol anaesthesia.

Mohan (2006) reported decrease in rectal temperature following premedication with glycopyrrolate and xylazine in compromised dogs. Subsequent administration of propofol resulted in increase in pulse rate, decrease in respiratory rate and rectal temperature.

Sharma *et al.* (2006) observed significant hypothermia in neonate calves administered with xylazine.

Sharma and Bhargava (2007) observed non significant decrease in rectal temperature with propofol general anaesthesia in dogs premedicated with atropine and triflupromazine hydrochloride. There was decreasing trend in respiratory rate.

### *2.3.1.2 Maintenance with* Sevoflurane

Johnson *et al.* (1998) observed that during maintenance of anaesthesia with sevoflurane there was decreased respiratory rate compared to isoflurane in adult dogs.

Clarke (1999) reported a dose related respiratory depression with sevoflurane anaesthesia in dogs.

Branson *et al.* (2001) studied sevoflurane anaesthesia maintenance in dogs and reported significantly lower respiratory rate due to depression of both diaphragmatic function and central respiratory function.

Polis *et al.* (2001) studied influence of halothane, isoflurane and sevoflurane in oxygen on clinical anaesthesia in healthy mongrel dogs and reported higher respiratory rate for halothane and lower rate for sevoflurane and isoflurane.

Gomez-Villamandos *et al.* (2005) reported that the combination of alpha-2 agonist romifidine, propofol and sevoflurane was an effective drug combination for inducing and maintaining general anaesthesia in healthy dogs. Sevoflurane and propofol resulted in respiratory depression charecterised by a decrease in ventilatory parameters.

Bennet *et al.* (2008) studied sevoflurane maintained anaesthesia in dogs and observed that body temperature did not fall below 36°C. Authors also reported the spontaneous respiratory rate in the range between 13 ± 5 per min at 110 min and 17 ± 7 per min at 100 min

Jadon *et al.* (2008) maintained anaesthesia in atropine - diazepam premedicated puppies for 30-40 min using 2.5% sevoflurane and recorded non significant decrease in rectal temperature. Authors also reported significant decrease in respiratory rate.

Topal (2008) evaluated sevoflurane anaesthesia in xylazine premedicated rabbits, reported decreased respiratory rate and increased $SpO_2$% during maintenance compared to the base-line values.

Lozano *et al.* (2009) maintained 12 dogs under sevoflurane anaesthesia and recorded rectal temperature at the time of induction with propofol as 37.98 ± 0.59°C and at the time of recovery as 35.85 ± 0.89°C.

Singh *et al.* (2010) recorded a slight and gradual decrease in the body temperature under sevoflurane maintained anaesthesia in dogs. Authors attributed generalised sedation, inhibition of skeletal muscle movement, reduction in metabolic rate and depression of thermoregulatory centre as the reasons for the decrease in body temperature. Authors also observed decrease in the respiratory rate under sevoflurane maintained anaesthesia in both healthy and compromised dogs.

#### *2.3.1.3 Maintenance with Other Agents*

Narayanan (2007) reported increase in pulse rate following isoflurane maintained anaesthesia in dogs.

Ramankutty (2008) reported decrease in rectal temperature and increase in pulse rate following propofol-isoflurane anaesthesia in dogs.

### 2.3.2 Cardiopulmonary Parameters

#### *2.3.2.1 Premedication and Induction*

Klide *et al.* (1975) studied the cardiopulmonary effects of xylazine in dogs and opined that bradycardia caused by xylazine might be attributed to an increase in vagal tone and decrease in activity of sympathetic cardiac nerves

According to Knight (1980) muscle relaxation effect of xylazine was due to partial synaptic blockade in the central nervous system.

According to Haskins *et al.* (1986) xylazine should not probably be used in dogs in which preservation of tissue blood flow and oxygenation are high priorities until organ specific studies had been completed. Authors observed reduced oxygen delivery to the tissues as evidenced by increased oxygen utilisation ratio, decreased venous $PO_2$ and oxygen content after xylazine administration.

Jacobson *et al.* (1994) reported significant increase in heart rate, cardiac index and decrease in stroke index following intravenous administration of glycopyrrolate at the rate of 0.01 mg/kg body weight. Subsequent administration of xylazine was associated with significant decreases in cardiac index, stroke index, oxygen delivery and oxygen consumption.

Muir III and Gadawski (1998) opined that the initial increase in heart rate in the dogs administered with propofol alone most likely represented sympathetic activation associated with loss of unconsciousness.

Lemke (2001) observed that administration of glycopyrrolate prior to administration of alpha-2 agonist could reduce the incidence and severity of bradycardia in healthy dogs, albeit at the expense of an increased myocardial work.

Nicholson and Watson (2001) justified judicious use of anticholinergics particularly in combination with those drugs that tend to cause marked bradycardia, such as alpha-2 agonists.

Sooryadas (2001) reported decreased heart rate during the anaesthesia with propofol in healthy and compromised dogs premedicated with atropine and xylazine.

Kuusela *et al.* (2003) compared propofol infusion and propofol-isoflurane anaesthesia in dogs and reported that propofol infusion produced more respiratory depression than propofol - isoflurane anaesthesia

Reyntijens *et al.* (2005) administered glycopyrrolate as preanaesthetic during sevoflurane-remifentanil based anaesthesia in children and could prevent bradycardia. Authors preferred glycopyrrolate to atropine because, the latter had central effects that lead to more prolonged impairment of parasympathetic nervous system control of heart rate than equipotent doses of the former.

Kandapal *et al.* (2005) studied clinico-physiological effects of xylazine in dogs and reported that there was significant reduction in heart rate, which was attributed to direct depressant action of xylazine on central nervous system.

Kale *et al.* (2006) reported a significant increase in heart rate with propofol anaesthesia in dogs.

Sharma *et al.* (2006) observed significant bradycardia in neonate calves administered with xylazine.

Sharma and Bhargava (2007) studied clinical effect of propofol general anaesthesia in dogs premedicated with atropine and triflupromazine hydrochloride and found that there was significant increase in heart rate.

Bras *et al.* (2008) observed that propofol bolus injection for induction at the rate of 6 mg/kg body weight did not change heart rate significantly in dogs after premedication with morphine sulphate (0.5 mg/kg body weight) and acepromazine (0.03 mg/kg body weight) intramuscularly.

#### *2.3.2.2 Maintenance with* Sevoflurane

Nakaigawa *et al.* (1995) observed that sevoflurane did not significantly alter heart rate at 1 or 2 MAC in dogs compared with controls. The decrease in arterial blood pressure was not followed by increased heart rate, which might be attributed to either sympathetic nervous system depression or suppression of baroreceptor reflex tone with sevoflurane or both.

According to Patel and Goa (1996) opined that sevoflurane up to 2 MAC preserved hepatic arterial blood flow inspite of decreasing cardiac output. It preserved total hepatic and portal venous blood flow at 1MAC, but at 1.50 MAC total hepatic blood flow was reduced by 26% and portal venous blood flow by 31 % with further reduction at 2MAC.

Mutoh *et al.* (1997) in a study in dogs concluded that cardiovascular effects of sevoflurane were similar to those of isoflurane, greater than those of halothane and less than those of enflurane. It was also observed that prolonged anaesthesia with sevoflurane increased sympathetic nervous system activity and increased cardiac output. Respiratory effects of sevoflurane were similar to those of isoflurane at all anaesthesia stages. Sevoflurane was suggested to be safe up to a moderate surgical anaesthesia stage in dogs undergoing spontaneous ventilation. Although assisted ventilation might be needed to maintain normocapnia at deep anaesthesia stages, such respiratory depression was easily reversed by the property of rapid control of anaesthesia depth associated with lower blood / gas partition coefficient causing fewer problems.

Johnson *et al.* (1998) observed that during maintenance of anaesthesia with sevoflurane there was increased heart rate compared to isoflurane in adult dogs.

Clarke (1999) reviewed and stated a dose related respiratory depression charecterised by a fall in ventilatory frequency with sevoflurane anaesthesia in dogs.

Picker *et al.* (2001) observed that heart rate in dogs increased with increasing anaesthetic concentration and was more with desflurane but intermediate with sevoflurane and least with halothane. The difference in the degree of increase in heart rate between agents was attributed to differences in their vagolytic action. In dogs, unlike in human, vagal activity predominates in the control of heart rate.

Polis *et al.* (2001) studied influence of halothane, isoflurane and sevoflurane in oxygen on clinical anaesthesia in healthy mongrel dogs and reported increased heart rate for isoflurane and sevoflurane anaesthesia but little effect with halothane; lower blood pressure with sevoflurane; significantly lower inspiratory oxygen fraction with sevoflurane. It was concluded that there was no significant difference between three anaesthetics.

Gomez-Villamandos *et al.* (2005) reported that the combination of alpha-2 agonist romifidine, propofol and sevoflurane was an effective drug combination for inducing and maintaining general anaesthesia in healthy dogs. Sevoflurane and propofol resulted in respiratory depression characterised by a decrease in ventilation parameters.

Kutter *et al.* (2006) maintained anaesthesia in healthy goat and sheep with approximately 1 MAC sevoflurane (2.70 ± 0.30% for goats; 3.30 ± 0.30% for sheep). Animals were premedicated with alpha- 2 agonist dexmedetomidine and ventilated mechanically. Here the heart rate, cardiac output and arterial oxygen tension decreased significantly.

Bennet *et al.* (2008) studied sevoflurane maintained anaesthesia in oxygen and nitrous oxide in dogs and reported that the heart rate ranged between 110 ± 15 per min at 110 min and 119 ± 25 per min at 100 min

Jadon *et al.* (2008) recorded a significant increase in heart rate with 2.50% sevoflurane anaesthesia in atropine - diazepam premedicated puppies.

Topal (2008) evaluated sevoflurane anaesthesia in xylazine premedicated rabbits and reported increased heart rate and $SpO_2$% during maintenance compared to the base-line values.

Yamishita *et al.* (2009) observed that old dogs had mild to moderate cardio-respiratory depression when they were anaesthetised with an equipotent titre of sevoflurane compared with the young dogs. It was also observed that

peripheral haemoglobin saturation ($SpO_2$) was lower in older dogs compared with younger dogs.

#### *2.3.2.3 Maintenance with Other Agents*

Ramankutty (2008) reported an increased oxygen saturation level during propofol-isoflurane anaesthesia in dogs.

### 2.3.3 Electrocardiogram

According to Bolton (1975) the electrocardiogram (ECG) could detect even small discrepancies in myocardial oxygenation; early changes would be manifested in T wave and S-T segment. It was opined that life threatening arrhythmias could occur in dogs secondary to endotoxaemia, pyometra, uraemia, neoplasia and electrolyte imbalances.

Klide *et al.* (1975) in an anaesthetic study measured heart rate to assess cardiovascular function.

Haskins *et al.* (1986) studied the cardiopulmonary consequences of intravenous xylazine at the rate of 1 mg/kg body weight and reported sustained significant bradycardia, significant decrease in cardiac output, left ventricular work load, first and second degree heart block.

Nakaigawa *et al.* (1995) observed that sevoflurane did not affect atrioventricular conduction times in pentobarbital anaesthetised dogs.

Singh *et al.* (1997) observed that glycopyrrolate pretreatment to xylazine administration in horses prevented bradycardia, onset of second degree atrioventricular block and kept cardiac index sustained to near normal values.

Administration of sevoflurane at higher doses had been reported to be depressing myocardium and cause fall in cardiac output (Clarke, 1999).

According to Dyson and Davies (1999) the size of the dog produced a significant effect on baseline heart rate. It was higher in small dogs.

R-wave of ECG was correlated with the cardiac output and progressively decreased R wave amplitude, which indicated, decreased cardiac out put. A second possible reason for decreased R wave amplitude was increase in ventricular size. A close inverse relationship between end diastolic ventricular volume and R wave amplitude had been shown in patients with enlarged heart (Nystrom *et al.,* 1999).

Narayanan (2007) studied the electrocardiographic changes following glycopyrrolate-xylazine premedication in healthy dogs and recorded sinus arrhythmia and sino atrial block.

Bennet *et al.* (2008) monitored heart rate to determine the adequacy of sevoflurane maintained anaesthesia in dogs.

Jadon *et al.* (2008) recorded mild degree electrocardiographic changes as depression of ST segment, lengthening of QT interval and decrease in PR interval with 2.50% sevoflurane maintained anaesthesia in atropine-diazepam premedicated puppies for 30-40 min

Ramankutty (2008) studied the electrocardiographic changes following glycopyrrolate-xylazine premedication in healthy and compromised dogs and recorded first degree heart block, second degree heart block and sinus arrest.

Singh *et al.* (2010) reported significant increase in heart rate with sevoflurane anaesthesia in dogs.

### 2.3.4 Capillary Refill Time

Capillary refill time increased in dogs maintained on isoflurane anaesthesia (Narayanan, 2007 and Ramankutty, 2008).

### 2.3.5 Clotting Time

Short and Bufalari (1999) reported that clotting time was not affected with propofol.

Narayanan (2007) reported marginal variations in blood coagulation time in healthy dogs maintained on isoflurane anaesthesia.

## 2.4 HAEMATOLOGICAL PARAMETERS

### 2.4.1 Premedication and Induction

Singh *et al.* (1997) observed a decline in Volume of Packed Red Cells (VPRC) and Haemoglobin (Hb) following xylazine administration in horses. The authors attributed this effect to accumulation of erythrocytes in the spleen and other vascular reservoirs due to a decrease in sympathetic tone caused by xylazine and movement of fluid from the extravascular to the intravascular compartment in response to hyperglycemia.

Sooryadas (2001) evaluated xylazine-propofol anaesthesia in atropine premedicated healthy and compromised dogs. The haematological changes reported were decrease in haemoglobin concentration; total erythrocyte count and total leukocyte count after premedication. All these parameters increased after induction. The neutrophilia with decrease in lymphocyte count was observed after premedication in healthy animals while neutrophil count showed a decrease with increase in lymphocyte count after premedication in compromised animals

Venugopal *et al.* (2002) reported significant decrease in total leukocyte count, haemoglobin and volume of packed red cells during propofol-ketamine anaesthesia with or without premedication.

Mohan (2006) reported decrease in volume of packed red cells, haemoglobin concentration, total leukocyte count and an increase in the neutrophil count with decrease in lymphocyte count after glycopyrrolate–xylazine premedication in compromised dogs.

Narayanan (2007) studied the effect of glycopyrrolate-xylazine premedication in healthy dogs and reported significant increase in lymphocyte count after premedication.

Ramankutty (2008) reported significant increase in erythrocyte sedimentation rate in both healthy and compromised dogs after premedication with glycopyrrolate-xylazine.

Chandrashekarappa *et al.* (2009) observed nonsignificant fall in packed cell volume, haemoglobin and total leukocyte count upon general anaesthesia induced with propofol in dogs. These changes were attributed to spleenic pooling of blood constituents, shifting of fluid from extra vascular compartment to intravascular compartment to maintain normal cardiac output and also could be due to loss of blood during surgery.

### 2.4.2 Maintenance of Anaesthesia with Sevoflurane

Jadon *et al.* (2008) maintained anaesthesia in atropine - diazepam premedicated puppies for 30-40 min using 2.5% sevoflurane and recorded non significant alterations in haemoglobin, packed cell volume and differential leukocyte count.

Singh *et al.* (2010) observed stable haemodynamics and reported transient non significant decrease in haemoglobin and packed cell volume during sevoflurane maintained anaesthesia in dogs. Authors attributed these changes due to the pooling of blood cells in the spleen. Neutrophil and lymphocyte counts were not changed significantly during anaesthesia indicating least adverse effect.

Insignificant fluctuations in basophil, eosinophil and monocyte counts were also observed.

### 2.4.3 Maintenance of Anaesthesia with Other Agents

Narayanan (2007) reported increase in lymphocyte count and decrease in neutrophil count during anaesthesia in healthy dogs with isoflurane.

Ramankutty (2008) reported significant decrease in haemoglobin concentration, significant increase in erythrocyte sedimentation rate (ESR), and decrease in total leukocyte count (TLC) in dogs, decrease in volume of packed red cells maintained with propofol-isoflurane anaesthesia. ESR reported a decrease in compromised dogs. ESR was significantly increased after recovery in healthy and compromised dogs. TLC reported an increase at 24 hours post anaesthesia. There was decrease in lymphocyte and increase in neutrophil count during anaesthesia in healthy and compromised dogs. Marginal variations in eosinophils were reported. There was increase in monocyte count during anaesthesia

## 2.5 SERUM BIOCHEMICAL PARAMETERS

### 2.5.1 Premedication and Induction

Singh *et al.* (1997) observed decline in total protein (TP) following xylazine administration in horses.

Mohan (2006) reported increase in TP after premedication with glycopyrrolate and xylazine, which decreased after administration of propofol in compromised dogs

Sharma *et al.* (2006) observed significant decrease in alanine aminotransferase level (ALT) at 30 min in neonate calves administered with xylazine without any clinical significance attributed.

Jain *et al.* (2007) studied the biochemical effect of propofol-ether anaesthesia with xylazine or diazepam premedication in dogs and reported that the change in values of TP was not significant. It was opined that the gradual non significant increase in the level of ALT might be due to the disruption of parenchyma cells as well as increased cell permeability by intravenous use of propofol with triflupromazine, diazepam or xylazine as preanaesthetic.

Narayanan (2007) studied the effect of glycopyrrolate-xylazine premedication in healthy dogs and the salient biochemical changes reported were significant decrease in TP and marginal increase in blood urea nitrogen (BUN) after premedication.

Chandrashekarappa *et al.* (2009) observed that TP, ALT, AST and creatinine levels did not differ significantly upon general anaesthesia induced with propofol in dogs

### 2.5.2 Maintenance of Anaesthesia with Sevoflurane

Jadon *et al.* (2008) observed nonsignificant alterations in TP, BUN, creatinine and ALT with 2.5% sevoflurane anaesthesia in atropine - diazepam premedicated puppies.

Singh *et al.* (2010) reported gradual significant decrease in TP after administration of 3-3.5% sevoflurane in dogs. Authors observed non significant changes in serum creatinine excluding the possibility of deleterious effect of sevoflurane in kidney. Less change in BUN and nonsignificant difference in ALT were also reported.

### 2.5.3 Maintenance of Anaesthesia with Other Agents

Ramankutty (2008) reported nonsignificant alterations in (BUN), non significant decrease in creatinine level in dogs maintained with anaesthesia using propofol-isoflurane. The increase in AST and decrease in TP noted during anaesthesia were within normal physiological limits.

## 2.6 ANAESTHESIA IN COMPROMISED PATIENTS

Patel and Goa (1996) reported that sevoflurane up to 2 MAC preserved hepatic arterial blood flow in dogs in spite of decreased cardiac output and blood pressure. There was freedom from arrhythmogenic effects of endogenous and/or exogenous epinephrine with sevoflurane.

According to Singh *et al.* (1997) glycopyrrolate pretreatment produced improvement in cardiac index and tissue oxygen delivery without altering ventilation. It would be a beneficial effect in xylazine sedation in compromised patients or when sedation is to be followed by the administration of an anaesthetic agent.

Clarke (1999) reported that sevoflurane would not sensitise the heart to epinephrine induced cardiac arrhythmias. He also reported that sevoflurane, like isoflurane appeared to be effective in reversing bronchospasm. Hepatic arterial blood flow was preserved during deep anaesthesia with sevoflurane and had no chance for immune mediated hepatitis.

Short and Bufalari (1999) reported that propofol caused no significant alterations in pharmacokinetic parameters in human patients with renal disease and hepatic disease suggesting that it could be used safely in those patients.

Branson *et al.* (2001) reported absence of cardiac dysrrhythmias in sevoflurane anaesthetised dogs because the dysrrhythmogenic dosage of epinephrine was much higher.

Topal *et al.* (2003) studied the hepatic effects of halothane, isoflurane and sevoflurane anaesthesia in dogs. It was observed that halothane mostly affected post operative liver function than isoflurane and sevoflurane. Isoflurane induced an elevation in serum activities of liver enzymes more frequently than did sevoflurane between 2 and 7 days.

Bein *et al.* (2005) reported that sevoflurane preserved myocardial function better than propofol during a brief period of ischemia in patients undergoing minimally invasive coronary artery bypass grafting in humans. It was opined that sevoflurane might have protective properties against ischemia in patients with coronary heart disease.

Driessen *et al.* (2006) found that suppression of vasomotor tone in horses under sevoflurane anaesthesia was very less. This property of sevoflurane could potentially provide a benefit for its use in critically ill equine patients that were often affected by substantial vasodilation.

Nath *et al.* (2009) studied physiological, haematological and biochemical changes in bitches with pyometra before and after surgery. They reported fever as a variable sign, which decreased after surgery. There was no variation in heart rate. Anaemia was a constant feature. There was leukocytosis along with absolute neutrophilia, lymphopenia and monocytosis. The biochemical changes were high TP, elevated BUN and creatinine, high AST value and low alanine ALT value. Authors opined that infection and stress resulted in haematological changes. The hyperprotenaemia may be due to increased globulin production as a result of infection. Authors attributed immune complex deposition in the glomeruli as a cause for glomerulonephritis and proximal tubular damage leading to renal failure. Hepatic degeneration at higher endotoxin level along with inhibition of gluconeogenesis and break down of skeletal muscles might be the cause for raised AST level. Authors attributed inhibition of ALT synthesis in liver or hepatic membrane damage resulting in fewer quantity of ALT enzyme available for leakage into serum as a cause for low ALT level. The compromised bitches were premedicated with diazepam, followed by anaesthesia induced and maintained with ketamine. It took 3-7 days after surgery for a significant reduction in higher haematological and serological values.

Singh *et al.* (2010) reported that the combination of thiopental sodium and sevoflurane was safer as compared to the thiopental sodium and isoflurane as it

provided more cardiac and respiratory stability in critically ill canine patients. Higher values of total protein in compromised dogs compared to healthy dogs were also reported.

## 2.7 AMERICAN SOCIETY OF ANAESTHESIOLOGISTS PHYSICAL STATUS CLASSIFICATION

Haitjema and Cullen (2001) rated American Society of Anaesthesiologists Physical Status (ASA) status level to the dogs by the physical examination.

According to Bednarski (2007) ASA physical status level I and II patients appeared comparatively to be at less risk for anaesthetic complications where as physical statuses III through V were at greater anaesthetic risk.

Laredo *et al.* (2009) in an anaesthetic study assessed ASA status classification of dogs by a physical examination and measurements of packed cell volume and total protein.

Lozano (2009) in an anaesthetic study assessed ASA status classification of dogs by a physical examination and on medical history.

**Physical status classification of veterinary patients ( Ref. Bednarski, 2007)**

| ASA Status Level | Patient Description |
|---|---|
| I | Normal healthy patient |
| II | Non-incapacitating systemic disease (e.g., obesity, mild dehydration, and simple fractures) |
| III | Severe systemic disease not incapacitating (e.g. compensated renal insufficiency, stable congestive heart failure, controlled diabetes mellitus, or caesarean section) |
| IV | Severe systemic disease that is a constant threat to life (e.g., gastric dilation and volvulus) |
| V | Moribund, not expected to live 24 h irrespective of intervention (e.g., severe uncompensated systemic disturbance) |

ASA, American Society of Anesthesiologists.
Procedures performed under emergency conditions are denoted by placing an *E* behind the physical status number.

**Description of sedation score categories (Ref: Lozano *et al.*, 2009)**

| | |
|---|---|
| **Not very sedated** | Able to stand up and walk. Fully responsive, wagging the tail. No signs of depression, drowsiness, ataxia or altered character with respect to how it was without any medication. Resistance to catheterisation, to restraint or being placed on the table. |
| **Slightly sedated** | Able to stand up and walk. Fully responsive but slower to react. Not wagging tail. Mild degree of depression, drowsiness, ataxia or mild changes in character. Mild resistance to catheterisation, restraint or position on the table. |
| **Sedated** | Able to stand up but reluctant to walk and/or ataxic. Slow reaction to stimuli. Signs of depression, drowsiness, ataxia and changed character. No resistance to catheterisation, restraint or positioning on the table. |
| **Deeply/very sedated** | Unable to stand up and walk. Unresponsive to stimuli. Depressed, drowsy and sleepy. Easy catheterisation, no restraint needed, no response. |

**Subjective criteria used to score induction of anaesthesia**

**(Ref: Branson *et al.*, 2001)**

| | |
|---|---|
| **Poor** | Stormy, prolonged induction, with unexpected attempts to bite or escape (inappropriate for the individual animal's personality), injury risk to patient or attending personnel, inappropriate vocalisation, severe muscle fasciculations, urination, defecation. Difficult intubation. Profound alterations in heart rate, rhythm, or respiratory rate. |
| **Fair** | Moderate resistance to recumbency with longer periods of excitement, attempts to escape or evade handling or the mask. Moderate physical restraint required by attending personnel. Moderate, transient muscle movements. Moderate difficulty in intubation. Noticeable changes in heart or respiratory rate. |
| **Good** | Smooth transition to lateral recumbency except for transient short periods of mild voluntary or involuntary movements. Slightly longer induction period with mild muscle movement. Slight resistance to intubation. Detectable alterations heart or respiratory rate not requiring intervention. |
| **Excellent** | Smooth, rapid l recumbency without vocalisation, muscle movement, twitching or seizure-like activity. Minimal excitement, breath holding or objection to mask (if mask used). Little change in heart or respiratory rate from pre induction values. |

**Subjective criteria used to score maintenance of anaesthesia**

**(Ref: Branson *et al.*, 2001)**

| | |
|---|---|
| **Poor** | Inability to maintain immobility or cardiopulmonary stability without major alterations in anaesthetic plan. Apnoea requiring intervention. Inadequate muscle relaxation or movement in response to painful stimuli. |
| **Fair** | Minor alternations to anaesthetic protocol required. Noticeable response to painful stimuli but not on routine tissue handling. Moderate muscle tone. |
| **Good** | Slight abnormal cardiopulmonary parameters not requiring alteration in anaesthetic protocol. Minimal muscle tone. Minimal reflex response to painful surgical manipulation. |
| **Excellent** | Normal blood pressure, heart rate and respiratory rate maintained. Adequate muscle relaxation. No response to surgical manipulation. Ability to change surgical depth effectively and rapidly. |

**Subjective criteria used to score recovery from anaesthesia**

**(Ref: Branson *et al.*, 2001)**

| | |
|---|---|
| **Poor** | Marked excitement, incoordination, failed attempts to reach sternal recumbency or stand, vocalisation, injury risk due to prolonged ataxia. Salivation, vomiting, defecation, biting, or licking inappropriately |
| **Fair** | Mild vocalisation, incoordination, thrashing, seizure- like activity. |
| **Good** | Minimal incoordination or excitement. Moderate extension of extubation time and/ or sternal recumbency (more than 15 minutes). Mild or transient vocalisation |
| **Excellent** | No excitement or incoordination. Smooth return to sternal recumbency and standing posture (if possible). Minimal vocalisation, muscle fasciculatuions, or seizure-like activity. Less than 15 minutes from turning off anaesthetic gas source and extubation, and <15 additional miutes to sternal recumbency. |

**Table 1. Normal values of physiological, haematological and biochemical parameters of dogs (Courtesy: Benjamin, 1985 and Chauhan and Agarwal, 2006)**

| Physiological parameters | Range | Haematological parameters | Range | Biochemical parameters | Range |
|---|---|---|---|---|---|
| Rectal temperature (°C) | 37.50-39.20 | Haemoglobin concentration (g/dl) | 12-18 | Blood urea nitrogen (mg/dl) | 10-20 |
| Pulse rate (per min) | 90-100 | Volume of packed red cells (%) | 37-54 | Serum creatinine (mg/dl) | 1.0-2.7 |
| Respiratory rate (per min) | 10-30 | Erythrocyte sedimentation rate (mm/h) | 1-6 | Aspartate amino transferase (U/L) | 10-62 |
| Peripheral oxygen saturation ($SpO_2$) (per cent) | >95% | Total leukocyte count ($10^3/mm^3$) | 8.2-13.5 | Alanine amino transferase (U/L) | 25-92 |
| Capillary refill time (sec) | 1-2 sec | Lymphocytes (%) | 12-30 | Total protein (g/dl) | 6.1-7.8 |
| Clotting time (min) | 1-5 min | Neutrophils (%) | 60-75 | | |
| | | Eosinophils (%) | 2-10 | | |
| | | Monocytes (%) | 3-9 | | |
| | | Basophils (%) | 0-1 | | |

# *Materials and Methods*

# 3. MATERIALS AND METHODS

## 3.1 SELECTION OF ANIMALS

The study was conducted in 12 dogs of either sex, different age, breed and body weight brought for various surgical procedures at Veterinary hospitals Mannuthy and Kokkalai, College of Veterinary and Animal Sciences, Mannuthy.

All the dogs were clinically examined and broadly divided into two groups as Group I healthy animals and Group II compromised animals. The dogs were subjected to a detailed physical examination and their physical status for anaesthesia was rated as per status classification for veterinary patients adapted from the American Society of Anaesthesiologists (ASA). The medical history, levels of volume of packed red cells and total protein were also considered to assess the status for anaesthesia (Haitjema and Cullen, 2001; Bednarski, 2007; Laredo *et al.*, 2009; Lozano *et al.,* 2009).

## 3.2 DESIGN OF STUDY

Healthy dogs presented for surgical procedures belonging to ASA physical status I and II were included in Group I. Those dogs brought for surgical procedures in systemically compromised condition belonging to ASA physical status III and IV were included in Group II. The six animals of each group were numbered serially in the following pattern:

Group I $I_1, I_2, I_3, I_4, I_5, \& I_6,$

Group II $II_1, II_2, II_3, II_4, II_5, \& II_6.$

## 3.3 PREPARATION OF THE ANIMALS

All dogs in Group I were subjected to withholding of food and water for 12 hours prior to anaesthesia and in Group II being in compromised condition, avoided such routine preanaesthetic preparations. After stabilisation of animal's condition,

all clinical, cardiopulmonary, haematological and serological measurements were taken as baseline values. The site of surgery was prepared for aseptic surgery. The anaesthesia and surgical procedures were performed with informed consent obtained from the owners.

## 3.4 ANAESTHETIC PROTOCOL

Anaesthetic procedures were adopted in both Group I and II animals with the following protocol.

### 3.4.1 Preanaesthetic Medication (Plate 1)

Glycopyrrolate[1] at the rate of 0.011 mg/kg body weight, followed by xylazine[2] at the rate of 1 mg/kg body weight at 15 minutes interval were administered intramuscularly.

### 3.4.2 Induction of Anaesthesia (Plates 2 & 4)

Fifteen minutes after preanaesthetic medication, propofol[3] 1% emulsion at the rate of 4 mg/kg body weight was administered by slow intravenous bolus injection in the cephalic vein.

### 3.4.3 Endotracheal Intubation (Plate 5)

After induction of anaesthesia, dogs were positioned in lateral recumbency for endotracheal intubation via oral route using a cuffed endotracheal tube with sizes ranged from 6 to 9. Lignocaine hydrochloride jelly[4] was applied on the endotracheal tube to provide lubrication prior to intubation. The intubation was done using a laryngoscope with Macintosh blade. Cuff of the endotracheal tube was adequately inflated and secured in position with the help of a tape tied on the upper jaw.

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1. Pyrolate- Neon Laboratories Ltd., Andheri (E), Mumbai
2. Xylaxin- Indian Immunologicals Ltd., Hyderabad
3. Neorof- Neon Laboratories Ltd., Andheri (E), Mumbai
4. Xylocaine 2% jelly- Astra Zeneca Pharma India Ltd., Bangalore

### 3.4.4 Anaesthetic Apparatus (Plate 3 )

Phoebus[5] anaesthetic machine with Bain's circuit system and a calibrated agent specific sevoflurane vapouriser[6] was used. The vaporiser was filled by quick-fill system to the maximum level indicated. The maximum vapouriser setting possible in the vaporiser was 8%, minimum was 0.20%.

### 3.4.5 Maintenance of Anaesthesia

The endotracheal tube was connected to the breathing unit to deliver the inhalant anaesthetic. Anaesthesia was maintained with sevoflurane[7] in pure oxygen. Dogs were allowed to breathe spontaneously. The fresh gas flow and anaesthetic concentration were individually adjusted for each dog. At the onset of maintenance, vaporiser was set to release a higher concentration of sevoflurane and later on maintained at reduced concentration by observing the clinical signs to achieve adequate depth "to effect" surgical anaesthesia. The vaporiser was then turned to 2% towards end of surgery in all animals to achieve uniformity while recording the recovery time by avoiding differences in depth of anaesthesia at the end of the procedures (Lozano *et al.,* 2009).

### 3.4.6 Extubation

When spontaneous swallowing reflex was regained the endotracheal tube was removed after deflating the cuff.

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5. Phoebus-MA 201. Ferro Curves, Kolkatta
6. Meditec Orion Anaesthesia Vaporiser – Meditec International, England Limited, UK
7. Sevorane- Abbot India Ltd., Mumbai

## 3.5 SURGICAL MANAGEMENT

The animals were subjected to various surgical operations under strict aseptic conditions. Dextrose normal saline[8] was infused intravenously to all the animals at the rate of 10 ml/kg/hour immediately after induction of anaesthesia. The surgical procedures performed in Group I were tumour resections ($I_1$, $I_2$), herniorrhaphies ($I_3$, $I_4$ and $I_6$) and oopherectomy ($I_5$). Surgical procedures performed in compromised animals of Group II were tumour resection ($II_1$), ovariohysterectomy for pyometra ($II_2$, $II_3$, $II_4$, $II_5$) and caesarean section ($II_6$). Postoperatively all the dogs were given antibiotics, Ampicillin–cloxacillin[9] at the rate of 20 mg/kg body weight intravenously for two consecutive days and orally for next three days. The sutures were removed on the eighth postoperative day.

## 3.6 MAIN ITEMS OF OBSERVATION

Main items observed were signalment and anamnesis, clinical observations, physiological parameters, haematological parameters, serum biochemical parameters, post anaesthetic and post operative complications, if any.

### 3.6.1 Signalment and Anamnesis

Details like breed, sex, age, body weight, history, past and immediate present treatment, health status, disease / condition and the surgery performed before were gathered.

### 3.6.2 Clinical Observations

Subjective assessment was done on qualitative clinical parameters.

#### *3.6.2.1 Clinical Signs*

The salient clinical signs exhibited by the dogs following premedication, during induction, maintenance and recovery from anaesthesia were recorded. The

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8. Dextrose Normal Saline – Baxter, Tamil Nadu
9. Megapen- Aristo Lab, Maharastra

subjective criteria as a tool used to assess and record the level of sedation as 'not very sedated', 'slightly sedated', 'sedated' and 'deeply sedated' was adopted from the work of Lozano *et al.* (2009). The subjective criteria as a tool used to assess and record the level of qualities of induction, maintenance and recovery from anaesthesia were adopted from the work of Branson *et al.* (2001). Accordingly those qualities were rated as 'poor', 'fair', 'good' and 'excellent'.

#### *3.6.2.2 Sevoflurane Vaporiser Concentration*

The sevoflurane vapouriser concentrations for each case at the beginning of maintenance, during maintenance of anaesthesia and towards the end of surgical procedures were recorded.

#### *3.6.2.3 Time for Induction of Anaesthesia*

The time interval between administration of propofol and loss of pedal reflex (Knight, 1980 and Kandapal *et al.,* 2005) was recorded as time of induction of anaesthesia.

#### *3.6.2.4 Duration of Anaesthesia*

The time interval between onset of propofol anaesthesia with the disappearance of pedal reflex, and the time of switching off the sevoflurane anaesthetic vaporiser was recorded.

#### *3.6.2.5 Depth of Anaesthesia*

Depth of anaesthesia was assessed based on various clinical signs *viz.* disappearance of palpebral and pedal reflexes, the degree of muscle relaxation and the responses to the surgery. The light plane of anaesthesia was judged with ventromedial rotation of eyeball, the presence of a moderately brisk palpebral reflex and a moderately relaxed jaw tone (Love *et al.*, 2007). Medium plane of anaesthesia was judged with ventromedial rotation of eyeball, the absence of palpebral reflex and relaxed jaw tone. Deep plane of anaesthesia was judged with abolished palpebral reflex, central position of eye ball from its ventromedial rotation and relaxed jaw.

### *3.6.2.6 Degree of Muscle Relaxation*

Muscle relaxation was rated as poor, moderate, good or excellent, depending up on the resistance in opening jaws manually and by assessment of relaxation of abdominal muscles during surgery.

### *3.6.2.7 Recovery Time*

It was calculated as the time interval in minutes from switching off the sevoflurane vaporiser to the time when the animal could stand up and walk unassisted. Various components of recovery time recorded as given below.

| | |
|---|---|
| Extubation time | Time interval in minutes between switching off the sevoflurane vaporiser and the first appearance of swallowing reflex. |
| Head lift time | Time interval in minutes between switching off the sevoflurane vaporiser and the time when the animal lifted the head. |
| Sternal recumbency time | Time interval in minutes between switching off the sevoflurane vaporiser and the time when the animal regained sternal recumbency. |
| Standing time | Time interval in minutes between switching off the sevoflurane vaporiser and the time when unassisted weight bearing on all limbs was attained. |

### **3.6.3 Physiological Parameters** (Plate 6)

The physiological parameters were recorded before premedication, immediately before the induction of anaesthesia (after premedication), 30 minutes after administration of propofol (during maintenance), 30 minutes after the discontinuation of sevoflurane administration (during recovery) and at 24 hours post recovery (at 24 hours post anaesthesia).

### *3.6.3.1 Rectal Temperature*

Rectal temperature was recorded in degree Celsius (°C) using a clinical thermometer and a multiparamonitor[10].

### *3.6.3.2 Pulse Rate*

Pulse rate was recorded per minute by placing the bulb of one or more fingers on the skin over the femoral artery and applying gentle pressure.

### *3.6.3.3 Respiratory Rate*

Respiratory rate was recorded per minute by counting the number of costal movements during inspiration and expiration. Subsequent counter checking was done during anaesthesia by counting the inflation or deflation of the rebreathing bag.

### *3.6.3.4 Peripheral Haemoglobin Oxygen Saturation (Pulse Oximetry)*

Peripheral haemoglobin oxygen saturation ($SPO_2$) was monitored and recorded by connecting the transducer of the pulse oximeter (Multiparamonitor) to the margin of the tongue/ear flap (Allen, 1992).

### *3.6.3.5 Electrocardiogram (ECG)*

The electrocardiography was performed in Lead II, at a paper speed of 25 mm/second using a multiparamonitor. Impedances were kept at a low level using conductive gel for ECG between the electrodes and the skin. Heart rate and cardiac rhythm were monitored by real time visual inspection of the ECG in the monitor.

### *3.6.3.6 Colour of Visible Mucous Membrane*

The colour of conjunctival/gingival mucous membrane was noted during the period of observation.

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10. Planet 50 - Larson and Tubro Ltd, Pondicherry

#### *3.6.3.7 Capillary Refill Time*

The capillary refill time in seconds was recorded from refilling of the tongue capillary and the mucous membrane of the gums after gentle digital pressure on it.

#### *3.6.3.8 Clotting Time*

Clotting time in minutes was recorded employing capillary tube method (Chauhan and Agarwal, 2006). The clotting time was calculated as the time interval between the appearance of blood and the fibrin strands across the gap between the ends of the capillary tube.

### 3.6.4 Haematological Parameters

Blood samples were collected before and after premedication, 30 minutes after administration of propofol (during maintenance), 30 minutes after the end of sevoflurane administration (during recovery) and at 24 hours post recovery (at 24 hours post anaesthesia). Disodium salt of ethylenediaminetetraacetic acid (EDTA) was used as anticoagulant.

Haemoglobin concentration, volume of packed red cells (VPRC), erythrocyte sedimentation rate (ESR), total leukocyte count (TLC) and differential leukocyte count (DLC) were estimated (Feldman *et al.,* 2000).

### 3.6.5 Serum Biochemical Parameters

Blood samples without anticoagulant for serum were collected before and after premedication, 30 minutes after administration of propofol (during maintenance), 30 minutes after the end of sevoflurane administration (during recovery) and at 24 hours post recovery (at 24 hours post anaesthesia).

The estimation of blood urea nitrogen (BUN), creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT) and total protein (TP) was conducted using the diagnostic kits (Agappe Diagnostics Pvt. Ltd., Ernakulam) by Semi Auto Analyser (Secoman, France).

### 3.6.6 Postanaesthetic and Post Operative Observations

The animals were observed till complete recovery from anaesthesia and at 24 hours after the surgery. The clinical signs and the signs of complications, if any, were recorded.

## 3.7 STATISTICAL ANALYSIS

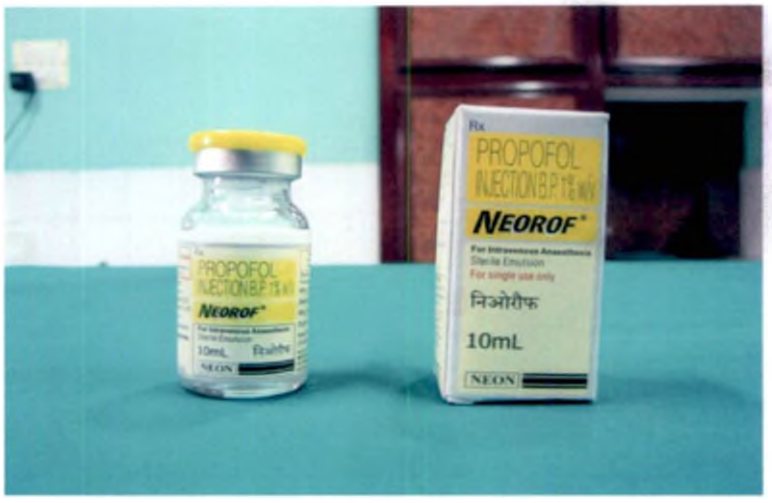
Statistical analysis was carried out using Students t-test for all the parameters recorded (Snedecor and Cochran, 1994). Paired t-test was used to analyse within groups and independent t-test was used to analyse between groups.

**Plate 1. Drugs Used for the Study**

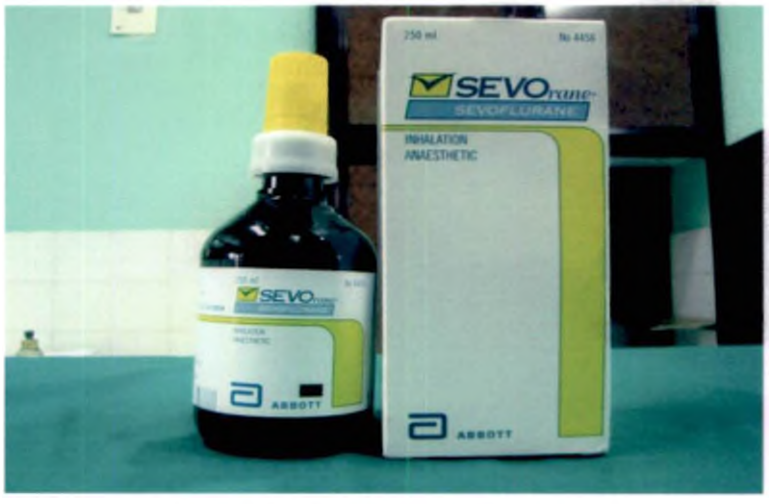

**A. PYROLATE (Glycopyrrolate)**


**B. XYLAXIN (Xylazine)**

**Plate 2. Drugs Used for the Study**

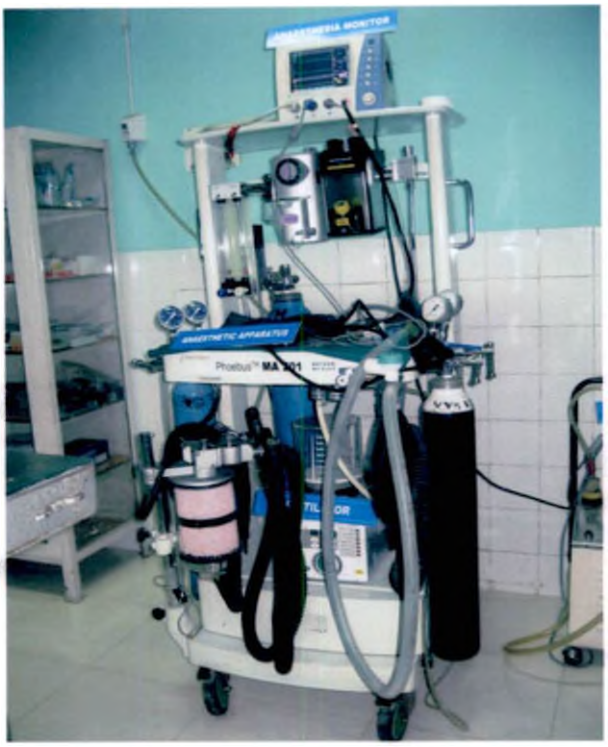



**A. NEOROF (Propofol)**




**B. SEVORANE (Sevoflurane)**

**Plate 3. Anaesthetic Equipment**

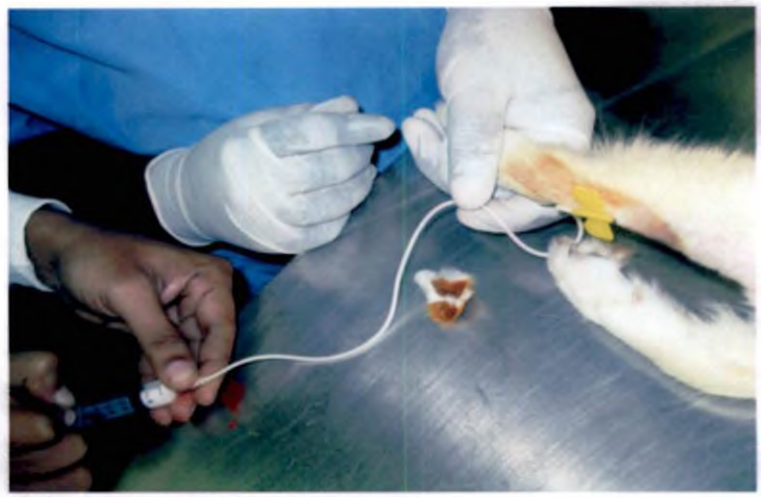


**A. Phoebus Anaesthetic Machine**

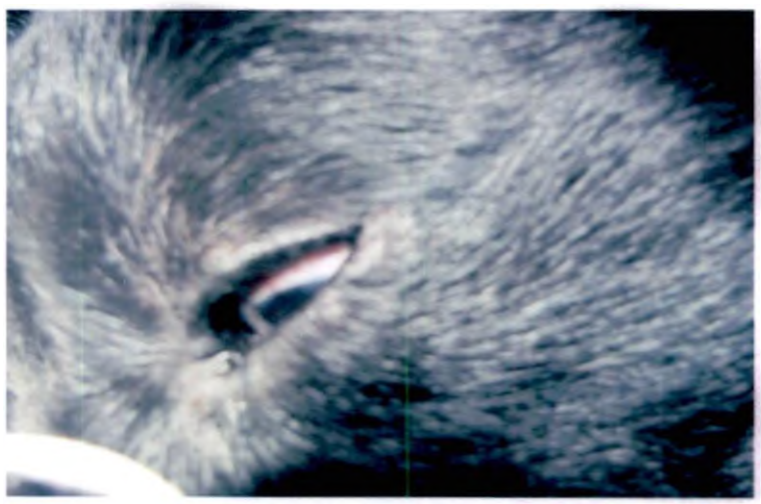

**B. Sevoflurane Vaporiser**

**C. "Quick Filling" Sevoflurane Vaporiser**

**Plate 4. Induction of Anaesthesia with Propofol**

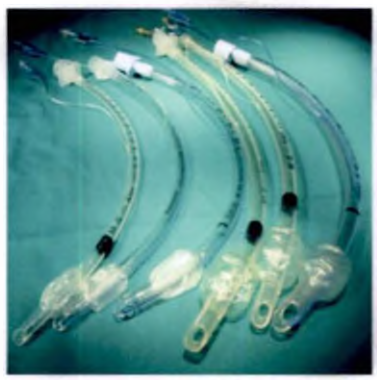


**A. Intravenous Bolus Injection in the Cephalic Vein**



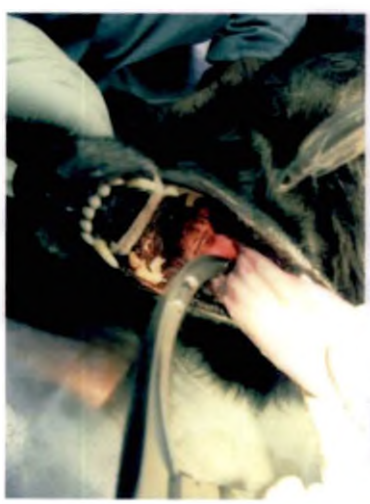
**B. Ventromedial Rotation of Eyeball**

**Plate 5. Endotracheal Intubation**

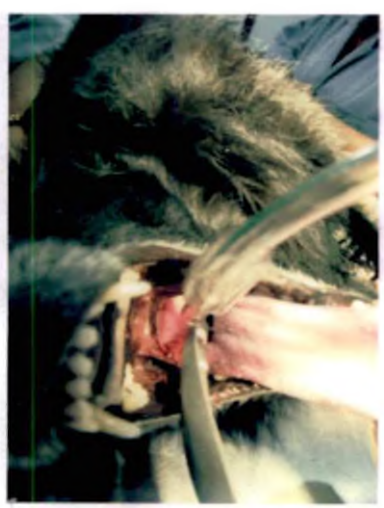


**A. Cuffed Endotracheal Tubes**

**B. Laryngoscope with Macintosh Blade**

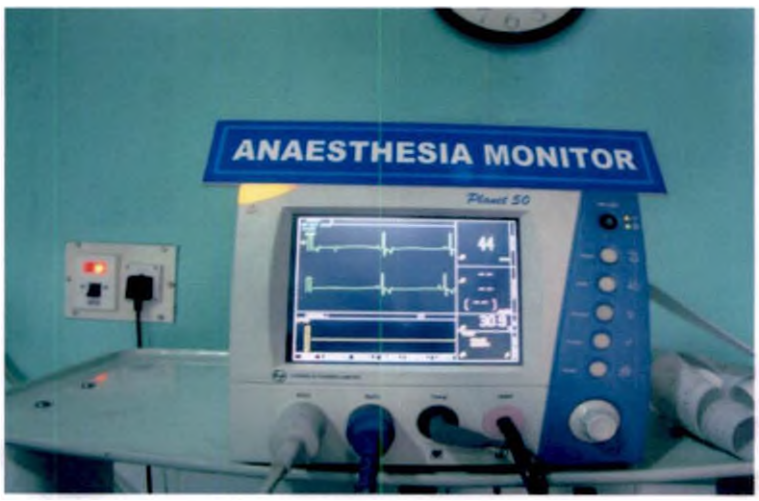


**C. Laryngoscope in Use**

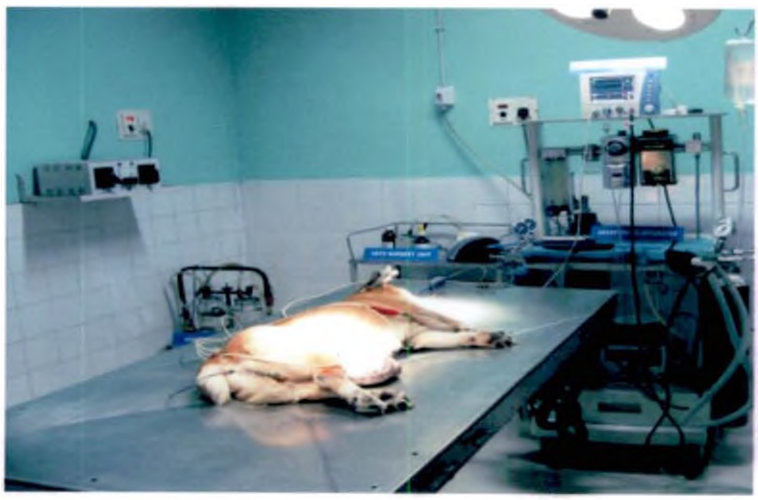


**D. Endotracheal Intubation**

**Plate 6. Anaesthesia Monitoring**




**A. Multipara Monitor**



**B. Multipara Monitor in Use**

# *Results*

# 4. RESULTS

Twelve dogs of either sex (4 males and 8 females), different age groups, breed, and body weight were subjected to various surgical procedures comprising of six healthy dogs and six dogs in compromised conditions. The preoperative preparations were found satisfactory in all the cases. All the surgical procedures were performed under propofol-sevoflurane anaesthesia with glycopyrrolate-xylazine premedication. Various parameters observed and recorded were statistically analysed. The results obtained were presented in Tables 2 to 15.

## 4.1. GROUP I

The animals of Group I belonged to healthy animals with ASA status I and II. The surgical procedures performed among Group I dogs were tumour resections ($I_1$ and $I_2$), herniorrhaphies ($I_3$, $I_4$ and $I_6$) and oopherectomy ($I_5$) (Table 2).

### 4.1.1 Signalment and Anamnesis (Tables 2 & 4)

There were three male and three female dogs with mean age of 6.33 ± 1.94 years (3 to 14 years) and mean body weight of 17.47 ± 3.08 kg (7.3 to 27 kg) in the group. None of the dogs had history of medical or surgical treatment during the past one month.

### 4.1.2 Clinical Observations (Table 6)

The animals were examined for physical condition and their physical status for anaesthesia (ASA status level) were rated as ASA I for three animals ($I_1$, $I_2$ and $I_6$) and ASA II for three animals ($I_3$, $I_4$ and $I_5$). The medical history, levels of volume of packed red cells (VPRC) and total protein (TP) were considered along with physical examination to judge the physical status. On

general clinical examination it was found that all the dogs were alert and active with normal physiological functions.

*4.1.2.1 Clinical Signs* (Table 8)

*4.1.2.1.1 Signs of Sedation* (Plate 7): Salient clinical signs indicative of sedation were winking of eyes, lowering of head, scanty salivation, incoordination in gait, ataxia, sitting on haunches followed by sternal recumbency with head down posture. The level of sedation assessed based on the clinical signs was 'slightly sedated' in two dogs ($I_3$ and $I_4$), 'sedated' in two dogs ($I_1$ and $I_2$) and 'deeply sedated' in two dogs ($I_5$ and $I_6$). Dogs that assessed with 'slightly sedated' score for sedation could able to stand up and walk, fully responsive to stimuli but slower to react and exhibited mild resistance to get placed on the table and intravenous catheterisation. Dogs that assessed with 'sedated' score exhibited incoordination of gait, ataxia, slow reaction to stimuli, nonresistant to get placed on the table and intravenous catheterisation. Dogs that were 'deeply sedated' could not able to stand up and walk, unresponsive to stimuli, nonresistant to get placed on the table and easy intravenous catheterisation.

*4.1.2.1.2 Signs of Induction of Anaesthesia*: Salient clinical signs indicative of anaesthetic induction were transition to lateral recumbency, relaxed jaw muscles, abolished pedal reflex, sluggish palpebral reflex, ventromedial rotation of eye balls and easiness to perform endotracheal intubation. Moderate to slight resistance to intubation was observed in four animals. The assessment of induction quality was fair in one dog ($I_3$), good in three dogs ($I_1$, $I_2$ and $I_4$) and excellent in two dogs ($I_5$ and $I_6$). Dogs that assessed with 'fair' score for induction quality had moderate resistance to lateral recumbency, mild voluntary movement, required moderate physical restraint by handling personnel, moderate jaw muscle tone and moderate difficulty in intubation. Dogs that assessed with 'good' score for induction quality had smooth transition to lateral recumbency, mild involuntary movement, mild jaw muscle tone and slight resistance to intubation. Dogs that assessed with 'excellent' score for induction quality had

smooth rapid lateral recumbency, no excitement, no jaw muscle tone and easy intubation.

*4.1.2.1.3 Signs of Anaesthesia:* All surgical procedures were performed in medium plane of anaesthesia, which was judged by ventromedial rotation of eyeball, the absence of palpebral reflex and relaxed jaw tone. Heart and respiratory rates were maintained steadily.

*4.1.2.1.4 Maintenance Quality:* The maintenance quality of anaesthesia with sevoflurane was good in one animal ($I_3$), excellent in five animals ($I_1$, $I_2$, $I_4$, $I_5$ and $I_6$). One dog that assessed with 'good' score for maintenance quality had minimal muscle tone and minimal reflex response to pain during surgical manipulation which got corrected by increasing the depth of anaesthesia. The ECG changes were sinus bradycardia and first and second degree heart blocks. Sudden variation in the rate of respiration observed in that animal was got corrected without any alteration in the anaesthetic protocol. All other dogs were assessed with 'excellent' score for maintenance quality had almost steady heart and respiratory rates, excellent muscle relaxation and non response to surgical manipulations. The anaesthetic depth could be changed effectively and rapidly in those dogs.

*4.1.2.1.5 Recovery Quality:* The dogs were observed during recovery for in coordination in the gait. Recovery quality was excellent in all the dogs.

*4.1.2.1.6 Undesirable Side Effects:* The undesirable side effect observed were regurgitation in one dog ($I_1$) after five minutes post anaesthesia.

***4.1.2.2 Sevoflurane Vaporiser Concentrations*** (Table 10)

At the beginning of maintenance of anaesthesia the sevoflurane vaporiser concentration was 6.17 ± 0.17%, which then gradually reduced to 2.67 ± 0.11% that achieved a light plane of anaesthesia. The deep plane of anaesthesia was achieved with 3.83 ± 0.11% vaporiser concentration. The vaporiser concentration

was turned to 2% in all animals towards the end of the surgical procedures (Fig.3).

*4.1.2.3 Time for Induction of Anaesthesia* (Table 11)

The time for induction of anaesthesia following the intravenous injection of propofol was 1.17 ± 0.17 min (Fig.1).

*4.1.2.4 Duration of Anaesthesia* (Table 11)

The duration of anaesthesia was 56.67 ± 4.09 min (Fig.1).

*4.1.2.5 Depth of Anaesthesia* (Table 8, 9)

The depth of anaesthesia was satisfactory in all the dogs. The surgical interventions performed were tumour resections ($I_1$ and $I_2$), herniorrhaphies ($I_3$, $I_4$ and $I_6$) and oopherectomy ($I_5$) (Plate 11).

*4.1.2.6 Degree of Muscle Relaxation* (Table 8, 9)

The degree of muscle relaxation was good in two animals ($I_1$ and $I_2$,) and excellent in four animals ($I_3$, $I_4$, $I_4$ and $I_6$) for performing the surgical procedures.

*4.1.2.7 Recovery Time* (Table 11)

The recovery time was 18.50 ± 3.19 min The different time components in the stages of recovery from the point of switching off the sevoflurane vaporiser were: - extubation time – 3.17 ± 0.48 min, head lift time - 8.50 ± 1.41 min, sternal recumbency time - 12.50 ± 2.06 min, and standing time - 18.50 ± 3.19 min (Fig.1 & 2).

### 4.1.3 Physiological Parameters (Table 12)

#### *4.1.3.1 Rectal Temperature*

The rectal temperature (°C) was 39.47 ± 0.17 and 38.9 ± 0.36, before and after premedication respectively. It was 38.12 ± 0.43, 38.48 ± 0.39 and 38.85 ± 0.27 during anaesthesia, recovery and at 24 hours respectively. There was decrease in rectal temperature after premedication, during anaesthesia, recovery and it increased to near base line value by 24 hours post operatively. The decrease in rectal temperature was significant ($p<0.05$) during anaesthesia (Fig.4).

#### *4.1.3.2 Pulse Rate*

The pulse rate (per minute) was 105.33 ± 1.86 and 110.83 ± 2.20, before and after premedication respectively. It was 118.83 ± 3.76, 110.67 ± 2.70 and 105.00 ± 1.71 during anaesthesia, recovery and at 24 hours respectively. There was significant increase in pulse rate after premedication ($p<0.01$), during anaesthesia $p<0.01$) and recovery ($p<0.05$). It reached to base line value by 24 hours post operatively (Fig.5).

#### *4.1.3.3 Respiratory Rate*

The respiratory rate (per min) was 28.67 ± 1.82 and 25.00 ± 1.91, before and after premedication respectively. It was 17.33 ± 1.48, 23.00 ± 1.55 and 28.33 ± 1.80 during anaesthesia, recovery and at 24 hours respectively. There was significant decrease ($p<0.01$) in respiratory rate after premedication, during anaesthesia and recovery. Respiratory rate started increasing during recovery and it reached to the base line value by 24 hours post operatively (Fig.6).

#### *4.1.3.4 Peripheral Haemoglobin Oxygen Saturation Level (Pulse Oximetry)*

The peripheral haemoglobin oxygen saturation ($SpO_2$) level (%) was 97.83 ± 0.31 and 97.00 ± 0.45, before and after premedication respectively. It

was 98.67 ± 0.61, 97.83 ± 0.48 and 96.5 ± 0.34 during anaesthesia, recovery and at 24 hours respectively. There was significant decrease ($p<0.01$) in peripheral haemoglobin oxygen saturation level after premedication. It increased during anaesthesia followed by a decrease to base line value later and observed significant decrease ($p<0.01$) at 24 hours post operatively (Fig.7).

### *4.1.3.5 Electrocardiogram (ECG)*

All the animals showed normal electrocardiographic recordings before premedication (Plate 11A), at the end of the anaesthesia and at 24 hours. ECG changes noticed following premedication were tachycardia ($I_5$) (Plate 11B) and first degree heart block ($I_3$) (Plate 11C). During anaesthesia the changes noticed were reduction in R amplitude ($I_1$ and $I_6$) (Plate 12A), sinus bradycardia ($I_3$) (Plate 12B), first degree heart block ($I_3$), second degree heart block ($I_3$) (Plate 12C), S-T segment depression ($I_3$ and $I_4$) (Plate 12C), S-T slurring ($I_4$) (Plate 12C), reduced R and T amplitude ($I_5$) (Plate 13A) and peaked T wave ($I_3$ and $I_5$).

### *4.1.3.6 Colour of Visible Mucous Membrane*

The colour of visible mucous membrane was pale roseate in all the dogs throughout the period of observation.

### *4.1.3.7 Capillary Refill Time*

The capillary refill time (in seconds) was 1.00 and 1.33 ± 0.05 before and after premedication respectively. It was 1.75 ± 0.16, 1.42 ± 0.11 and 1.00 during anaesthesia, recovery and at 24 hours respectively. There was significant increase ($p<0.01$) in capillary refill time after premedication, during anaesthesia and recovery ($p<0.05$). It was decreased to the base line value at 24 hours post operatively (Fig.8).

#### *4.1.3.8 Clotting Time*

The clotting time (min) was 3.17 ± 0.11 and 3.67 ± 0.11, before and after premedication respectively. It was 4.17 ± 0.11, 3.79 ± 0.15 and 3.33 ± 0.21 during anaesthesia, recovery and at 24 hours respectively. There was an increase in clotting time after premedication, during anaesthesia and recovery. It reached to the base line value at 24 hours post operatively. The increase in clotting time during recovery was significant ($p<0.01$) (Fig.9).

### 4.1.4 Haematological Parameters (Tables 13 and 14)

#### *4.1.4.1 Haemoglobin Concentration*

The haemoglobin concentration (g/dl) was 14.70 ± 0.69 and 14.62 ± 0.51, before and after premedication respectively. It was 12.25 ± 1.08, 13.08 ± 0.41 and 12.25 ± 1.36 during anaesthesia, recovery and at 24 hours respectively. There was decrease in haemoglobin concentration after premedication, during anaesthesia, recovery and at 24 hours post operatively. The decrease in haemoglobin concentration during anaesthesia was significant ($p<0.05$) (Fig.10).

#### *4.1.4.2 Volume of Packed Red Cells (VPRC)*

The volume of packed red cells (%) was 42.12 ± 1.84 and 41.48 ± 1.38, before and after premedication respectively. It was 36.73 ± 0.10, 36.68 ± 0.91 and 39.22 ± 0.87 during anaesthesia, recovery and at 24 hours respectively. There was decrease in VPRC after premedication, during anaesthesia, recovery and reached to baseline value at 24 hours post operatively. The decrease in VPRC during anaesthesia ($p<0.05$) and recovery ($p<0.01$) was significant (Fig.11).

#### *4.1.4.3 Erythrocyte Sedimentation Rate (ESR)*

The erythrocyte sedimentation rate (mm/h) was 2.17 ± 0.17 and 2.50 ± 0.22, before and after premedication respectively. It was 3.50 ± 0.22, 3.67 ± 0.21 and 2.33 ± 0.21 during anaesthesia, recovery and at 24 hours respectively. There

was marginal increase in ESR after premedication, during anaesthesia and recovery. It reached near to the base line value at 24 hours post operatively. The increase in ESR during anaesthesia and recovery was significant ($p<0.01$) (Fig.12).

#### *4.1.4.4 Total Leukocyte Count (TLC)*

The total leukocyte count ($10^3/mm^3$) was 14.50 ± 1.00 and 14.43 ± 1.54, before and after premedication respectively. It was 12.08 ± 1.12, 13.12 ± 1.40 and 20.33 ± 2.34 during anaesthesia, recovery and at 24 hours respectively. There was decrease in TLC after premedication, during anaesthesia and recovery. The decrease in TLC during anaesthesia was significant ($p<0.01$). It was significantly increased ($p<0.05$) at 24 hours post operatively (Fig.13).

#### *4.1.4.5 Differential Leukocyte Count (DLC)*

*4.1.4.5.1 Lymphocyte Count:* The lymphocyte count (%) was 21.08 ± 2.93 and 19.60 ± 3.20, before and after premedication respectively. It was 25.00 ± 3.98, 21.33 ± 3.70 and 21.10 ± 2.98 during anaesthesia, recovery and at 24 hours respectively. There was mild decrease in lymphocyte count after premedication but it was increased during anaesthesia. The lymphocyte count reached near to the base line value during recovery and at 24 hours post operatively (Fig.14).

*4.1.4.5.2 Neutrophil Count:* The neutrophil count (%) was 71.92 ± 2.73 and 74.83 ±3.32 before and after premedication respectively. It was 70.33 ± 3.99, 73.67 ± 3.82 and 70.00 ± 2.88 during anaesthesia, recovery and at 24 hours respectively. There was mild increase in neutrophil count after premedication, but decreased during anaesthesia followed by a marginal increase during recovery. It was decreased at 24 hours post operatively (Fig.15).

*4.1.4.5.3 Eosinophil Count:* The eosinophil count (%) was 4.00 ± 1.34 and 2.40 ± 0.55 before and after premedication respectively. It was 2.67 ± 0.56, 2.5 ± 0.34

and 6.00 ± 0.86 during anaesthesia, recovery and at 24 hours respectively. There was decrease in eosinophil count after premedication, during anaesthesia and recovery. It was increased at 24 hours post operatively (Fig.16).

*4.1.4.5.4 Monocyte Count:* The monocyte count (%) was 3.00 ± 0.45 and 3.17 ± 0.95 before and after premedication respectively. It was 2.00 ± 0.37, 2.50 ± 0.56 and 2.90 ± 0.57 during anaesthesia, recovery and at 24 hours respectively. There was increase in monocyte count after premedication and was decreased during anaesthesia, recovery and at 24 hours post operatively (Fig.17).

*4.1.4.5.5 Basophil Count*: The basophil count (%) was zero in all the dogs in the duration of the study.

**4.1.5 Serum Biochemical Parameters** (Table 15)

***4.1.5.1 Blood Urea Nitrogen (BUN)***

The blood urea nitrogen (mg/dl) was 11.50 ± 1.34 and 12.50 ± 1.65, before and after premedication respectively. It was 12.67 ± 1.91, 11.00 ± 1.46 and 12.00 ± 1.79 during anaesthesia, recovery and at 24 hours respectively. There was increase in BUN value after premedication, during anaesthesia and at 24 hours post operatively. All these fluctuations were within physiological limits (Fig.18).

***4.1.5.2 Creatinine***

The creatinine (mg/dl) was 1.05 ± 0.06 and 1.12 ± 0.12, before and after premedication respectively. It was 1.00 ± 0.11, 1.12 ± 0.11 and 1.20 ± 0.29 during anaesthesia, recovery and at 24 hours respectively. There was increase in creatinine after premedication and at 24 hours post operatively. All these fluctuations were within physiological limits (Fig.19).

### *4.1.5.3 Aspartate Aminotransferase (AST)*

The aspartate aminotransferase (U/L) was 39.00 ± 3.85 and 38.83 ± 2.61, before and after premedication respectively. It was 36.83 ± 6.01, 36.67 ± 6.46 and 73.5 ± 8.29 during anaesthesia, recovery and at 24 hours respectively. There was decrease in AST after premedication and during anaesthesia. It was significantly increased ($p<0.01$) at 24 hours post operatively (Fig.20).

### *4.1.5.4 Alanine Aminotransferase (ALT)*

The alanine aminotransferase (U/L) was 32.33 ± 3.65, and 34.33 ± 3.60, before and after premedication respectively. It was 30.67 ± 2.93, 29.17 ± 3.03 and 38.50 ± 3.40 during anaesthesia, recovery and at 24 hours respectively. The ALT value decreased during anaesthesia and recovery without any significance. There was non significant increase in ALT value after premedication and at 24 hours post operatively (Fig.21).

### *4.1.5.5 Total Protein (TP)*

The total protein (g/dl) was 6.60 ± 0.33 and 6.53 ± 0.35, before and after premedication respectively. It was 5.58 ± 0.26, 5.32 ± 0.40 and 6.45 ± 0.20 during anaesthesia, recovery and at 24 hours respectively. There was decrease in total protein value after premedication, during anaesthesia, recovery and at 24 hours. The decrease was significant ($p<0.05$) during anaesthesia and recovery (Fig.22).

### 4.1.6 Postanaesthetic and Postoperative Observations (Plate 10)

All the animals had rapid, smooth and uneventful recovery from anaesthesia. The surgical wound healing took the normal duration and was uneventful.

## 4.2 GROUP II

The animals of Group II belonged to ASA status III and IV. The surgical procedures performed among Group II dogs were tumour resection ($II_1$) ovariohysterectomies on account of pyometra ($II_2$, $II_3$, $II_4$ and $II_5$) and caesarean section $II_6$ (Table 3).

### 4.2.1 Signalment and Anamnesis (Tables 3 & 5)

There were one male and five female dogs with mean age of 7.83 ± 1.54 years (2.5 to 13.5 years), mean body weight of 15.43 ± 4.05 kg (6.00 to 29 kg) in the group. All the dogs had history of medical treatment from the onset of illness.

### 4.2.2 Clinical Observations (Table 7 & 11).

The animals were examined for physical condition and their physical status for anaesthesia was rated as ASA III for four animals ($II_3$, $II_4$, $II_5$ and $II_6$) and ASA IV for two animals ($II_1$ and $II_2$). The medical history, levels of volume of packed red cells (VPRC) and total protein (TP) were considered along with physical examination to judge the physical status. All the dogs had comparatively lower VPRC and TP. On general clinical examination it was found that two dogs ($II_1$ and $II_2$) with pale and four dogs ($II_3$, $II_4$, $II_5$ and $II_6$) with congested conjunctival mucous membrane. The animals were found generally dull, not taking food and water sufficiently.

#### *4.2.2.1 Clinical Signs* (Tables 9 )

*4.2.2.1.1 Signs of Sedation:* Salient clinical signs indicative of sedation were winking of eyes, lowering of head, scanty salivation, incoordination in gait, ataxia, sitting on haunches, followed by sternal recumbency with head down posture. The level of sedation assessed based on the clinical signs was 'sedated' in four dogs ($II_1$, $II_2$, $II_4$ and $II_5$); and 'deeply sedated' in two dogs ($II_3$ and $II_6$).

Dogs that assessed with 'sedated' score exhibited incoordination of gait, ataxia, slow reaction to stimuli, nonresistant to get placed on the table and intravenous catheterisation. Dogs that were 'deeply sedated' could not able to stand up and walk, unresponsive to stimuli, nonresistant to get placed on the table and easy intravenous catheterisation.

*4.2.2.1.2 Signs of Induction of Anaesthesia*: Salient clinical signs indicative of anaesthesia induction were smooth transition to lateral recumbency, relaxed jaw muscles, abolished pedal reflex, sluggish palpebral reflex, ventromedial rotation of eye balls and able to perform endotracheal intubation. The assessment of induction quality was good in four dogs ($II_1$, $II_2$, $II_4$ and $II_5$) and excellent in two dogs ($II_3$ and $II_6$). Dogs that assessed with 'good' score for induction quality had smooth transition to lateral recumbency, mild involuntary movement, mild jaw muscle tone and slight resistance to intubation. Dogs that assessed with 'excellent' score for induction quality had smooth rapid lateral recumbency, no excitement, no jaw muscle tone and easy intubation.

*4.2.2.1.3 Signs of Anaesthesia:* All surgical procedures were performed in medium plane of anaesthesia, which was judged by ventromedial rotation of eye ball, the absence of palpebral reflex and relaxed jaw tone. Heart rate and respiratory rate were maintained steadily with absence of all other reflexes.

*4.2.2.1.4 Maintenance Quality:* The maintenance quality of anaesthesia with sevoflurane was good in two animals ($II_1$ and $II_3$), excellent in four animals ($II_2$, $II_4$, $II_5$ and $II_6$) Two dogs that assessed with 'good' score for maintenance quality had minimal muscle tone and minimal reflex response to pain during surgical manipulation which got corrected by increasing the depth of anaesthesia. The ECG changes as first degree heart block, single ventricular premature contraction and sudden variations in the rate of respiration observed in those animals was got corrected without any alteration in the anaesthetic protocol. The other four dogs were assessed with 'excellent' score for maintenance quality had steady heart and respiratory rates, excellent muscle relaxation and non response to surgical

manipulations. The anaesthetic depth could be changed effectively and rapidly in those dogs.

*4.2.2.1.5 Recovery Quality:* The dogs were observed during recovery for in coordination in the gait. Recovery quality was assessed as excellent in all the dogs.

*4.2.2.1.6 Undesirable Side Effects:* Sneezing after five minutes post anaesthesia ($II_6$) and vocalisation in two dogs after eight hours post anaesthesia ($II_1$ and $II_4$) were observed.

***4.2.2.2 Sevoflurane Vaporiser Concentrations*** (Table 10)

At the beginning of maintenance of anaesthesia, the sevoflurane vaporiser concentration was 5.33 ± 0.11%, which then progressively reduced to 2.58 ± 0.08% and achieved a light plane of anaesthesia. The deep plane of anaesthesia was achieved with 3.67 ± 0.11% vaporiser concentration. The vaporiser concentration was turned to 2% in all animals towards the end of surgical procedures (Fig.3).

***4.2.2.3 Time for Induction of Anaesthesia***

The time for induction following the intravenous injection of propofol was 0.92 ± 0.05 min (Fig.1).

***4.2.2.4 Duration of Anaesthesia***

The duration of anaesthesia was 72.50 ± 4.47 min (Fig.1).

***4.2.2.5 Depth of Anaesthesia*** (Table 9)

The depth of anaesthesia was satisfactory in all the dogs. The surgical interventions performed were tumour resection ($II_1$), ovario-hysterectomy for pyometra ($II_2$, $II_3$, $II_4$ and $II_5$) and caesarean section ($II_6$) (Plate 14).

*4.2.2.6 Degree of Muscle Relaxation*

The degree of muscle relaxation was good in one animal ($(II_1)$) and excellent in five animals ($II_2$, $II_3$, $II_4$, $II_5$, and II6) for performing the surgical procedures.

*4.2.2.7 Recovery Time*

The recovery time was 29.00 ± 1.75 min The different time components in the stages of recovery from the point of switching off the sevoflurane vaporiser were:- extubation time - 5.00 ± 0.26 min, head lift time - 13.17 ± 0.79 min, sternal recumbency time - 18.50 ± 1.31 and standing time - 29.00 ± 1.75 min (Fig.1 & 2).

**4.2.3 Physiological Parameters** (Table 12)

*4.2.3.1 Rectal Temperature*

The rectal temperature (°C) was 38.60 ± 0.50 and 38.07 ± 0.41 before and after premedication respectively. It was 37.40 ± 0.37, 38.23 ± 0.25 and 38.47 ± 0.48 respectively during anaesthesia, recovery and at 24 hours. There was decrease in rectal temperature after premedication, during anaesthesia and recovery. It increased to near preanaesthetic value at 24 hours post operatively. The decrease in rectal temperature was significant after premedication ($p< 0.05$) and during anaesthesia ($p< 0.01$) (Fig.4).

*4.2.3.2 Pulse Rate*

The pulse rate (per min) was 103.50 ± 2.31 and 108.67 ± 1.58 before and after premedication respectively. It was 117.50 ± 2.60, 110.00 ± 1.61 and 101.83 ± 2.44 during anaesthesia, recovery, and at 24 hours respectively. There was significant increase ($p<0.01$) in pulse rate after premedication, during anaesthesia and recovery but found decreased at 24 hours post operatively (Fig.5).

### *4.2.3.3 Respiratory Rate*

The respiratory rate (per min) was 25.67 ± 0.80 and 20.67 ± 0.95 before and after premedication respectively. It was 16.50 ± 1.48, 24.83 ± 1.33 and 28.00 ± 0.86 during anaesthesia, recovery and at 24 hours respectively. There was significant decrease ($p<0.01$) in respiratory rate after premedication and during anaesthesia. It was decreased during recovery and significantly increased ($p<0.01$) at 24 hours (Fig.6).

### *4.2.3.4 Peripheral Haemoglobin Oxygen Saturation Level (Pulse Oximetry)*

The peripheral haemoglobin oxygen saturation ($SpO_2$) level (%) was 96.00 ± 0.52 and 95.17 ± 0.60 before and after premedication respectively. It was 98.00 ± 0.68, 95.67 ± 0.84 and 95.00 ± 0.37 during anaesthesia, recovery and at 24 hours respectively. There was significant decrease ($p<0.01$) in oxygen saturation level after premedication, but significantly increased ($p < 0.05$) during anaesthesia, which decreased again during recovery and significantly decreased ($p< 0.05$) at 24 hours (Fig.7).

### *4.2.3.5 Electrocardiogram (ECG)*

Electrocardiographic changes noticed following premedication were tachycardia ($II_2$ and $II_4$) and first degree heart block ($II_1$). During anaesthesia the changes noticed were reduction in R amplitude ($II_1$ and $II_6$), reduced P amplitude ($II_1$) (Plate 13B), change in polarity of T wave ($II_4$ and $II_6$) (Plate 13C), high P amplitude ($II_4$) (Plate 13C), reduced T amplitude ($II_2$ and $II_5$) (Plate 14A), S-T segment depression ($II_3$) (Plate 14B), peaked T wave ($II_3$) (Plate 14C), first degree heart block ($II_1$), tachycardia ($II_2$ and $II_4$) (Plate 15A) and single ventricular premature contraction ($II_1$) (Plate 15B).

*4.2.3.6 Colour of Visible Mucous Membrane*

The colour of visible mucous membrane was pale in two animals ($II_1$ and $II_2$) and congested in four animals.

*4.2.3.7 Capillary Refill Time*

The capillary refill time (in seconds) was 1.08 ± 0.08 and 1.54 ± 0.10 before and after premedication respectively. It was 2.17 ± 0.14, 1.67 ± 0.08 and 1.08 ± 0.08 during anaesthesia, recovery and at 24 hours respectively. There was significant increase ($p<0.01$) in capillary refill time after premedication, during anaesthesia and recovery. It decreased to the preanaesthetic value at 24 hours post operatively (Fig.8).

*4.2.3.8 Clotting Time*

The clotting time (min) was 3.92 ± 0.20 before and after premedication. It was 4.25 ± 0.11, 4.00 ± 0.22 and 3.92 ± 0.20 during anaesthesia, recovery and at 24 hours respectively.

The clotting time was not affected after premedication but there was significant increase ($p<0.05$) during anaesthesia, nonsignificant increase during recovery and returned to the preanaesthetic value at 24 hours post operatively (Fig.9).

**4.2.4 Haematological Parameters** (Tables 13 and 14)

*4.2.4.1 Haemoglobin Concentration*

The haemoglobin concentration (g/dl) was 10.80 ± 1.37 and 9.68 ± 1.12 before and after premedication respectively. It was 8.50 ± 1.22, 7.88 ± 1.09 and 10.10 ± 1.14 during anaesthesia, recovery and at 24 hours respectively. There was significant decrease in haemoglobin concentration after premedication ($p< 0.05$), during anaesthesia ($p<0.01$) and recovery ($p<0.01$). The haemoglobin concentration returned near to the preanaesthetic value at 24 hours post operatively (Fig.10).

### *4.2.4.2 Volume of Packed Red Cells (VPRC)*

The volume of packed red cells (%) was 29.80 ± 3.70 and 27.57 ± 3.53 before and after premedication respectively. It was 23.23 ± 3.17, 21.83 ± 2.87 and 28.22 ± 3.04 during anaesthesia, recovery, and at 24 hours respectively. There was significant decrease ($p<0.05$) in VPRC after premedication, during anaesthesia, and recovery ($p<0.01$). It increased to near preanaesthetic value at 24 hours post operatively (Fig.11).

### *4.2.4.3 Erythrocyte Sedimentation Rate (ESR)*

The erythrocyte sedimentation rate (mm/hr) was 6.67 ± 1.17 and 8.33 ± 1.58 before and after premedication respectively. It was 10.83 ± 1.89, 11.33 ± 1.93 and 8.50 ± 1.48 during anaesthesia, recovery, and at 24 hours respectively. There was significant increase in ESR after premedication ($p<0.05$), during anaesthesia ($p<0.01$), recovery ($p<0.01$) and at 24 hours ($p<0.05$) post operatively (Fig.12).

### *4.2.4.4 Total Leukocyte Count (TLC)*

The total leukocyte count ($10^3$ /$mm^3$) was 21.00 ± 4.17 and 20.18 ± 4.00 before and after premedication respectively. It was 15.05 ± 3.40, 18.43 ± 3.14 and 31.10 ± 7.40 during anaesthesia, recovery and at 24 hours respectively. There was significant decrease ($p<0.05$) in TLC after premedication and during anaesthesia followed by an increase at 24 hours post operatively (Fig.13).

### *4.2.4.5 Differential Leukocyte Count (DLC)*

*4.2.4.5.1 Lymphocyte Count:* The lymphocyte count (%) was 32.83 ± 2.74 and 29.67 ± 2.91 before and after premedication respectively. It was 28.00 ± 2.29, 25.83 ± 2.30 and 30.33 ± 2.39 during anaesthesia, recovery and at 24 hours respectively. There was significant decrease ($p<0.05$) in lymphocytes after premedication and again it was decreased during anaesthesia, recovery and at 24 hours post operatively (Fig.14).

*4.2.4.5.2 Neutrophil Count:* The neutrophil count (%) was 62.17 ± 2.77 and 66.00 ± 3.12 before and after premedication respectively. It was 67.00 ± 3.54, 68.67 ± 3.72 and 64.50 ± 2.35 during anaesthesia, recovery and at 24 hours respectively. There was significant increase ($p<0.01$) in neutrophil count after premedication. It was in increased state during anaesthesia, recovery and at 24 hours post operatively (Fig.15).

*4.2.4.5.3 Eosinophil Count:* The eosinophil count (%) was 1.83 ± 0.48 and 1.33 ± 0.42 before and after premedication respectively. It was 1.50 ± 0.43, 2.00 ± 0.58 and 1.17 ± 0.31 during anaesthesia, recovery and at 24 hours respectively. There was marginal decrease in eosinophil count after premedication and during anaesthesia. The marginal increase in eosinophil count was recorded during recovery and then decreased at 24 hours post operatively (Fig.16).

*4.2.4.5.4 Monocyte Count:* The monocyte count (%) was 3.17 ± 1.47 and 3.00 ± 1.06 before and after premedication respectively. It was 3.33 ± 1.43, 3.5 ± 1.46 and 3.83 ± 1.30 during anaesthesia, recovery, and at 24 hours respectively. There was mild decrease in monocyte count after premedication and then increased during anaesthesia, recovery and at 24 hours post operatively. All these fluctuations were non significant (Fig.17).

*4.2.4.5.5 Basophil Count*: There was one basophil cell before premedication in one dog ($II_3$), one appeared during anaesthesia in another dog ($II_5$), and at 24 hours in yet another dog ($II_6$). In all other animals and in all other observation periods it was zero.

**4.2.5 Serum Biochemical Parameters** (Table 15)

***4.2.5.1 Blood Urea Nitrogen (BUN)***

The blood urea nitrogen (mg/dl) was 60.17 ± 42.03 and 59.33 ± 40.03 before and after premedication respectively. It was 61.83 ± 41.16, 59.83 ± 41.70 and 69.17 ± 44.66 during anaesthesia, recovery and at 24 hours respectively.

There was non significant increase in BUN value from the preanaesthetic value during anaesthesia and at 24 hours post operatively. It was decreased after premedication and during recovery without any significance (Fig.18).

#### *4.2.5.2 Creatinine*

The creatinine (mg/dl) was 3.73 ± 2.60 and 3.75 ± 2.42 before and after premedication respectively. It was 3.70 ± 2.53, 3.73 ± 2.50 and 4.75 ± 2.98 during anaesthesia, recovery and at 24 hours respectively. There was marginal increase in creatinine value after premedication and at 24 hours (Fig.19).

#### *4.2.5.3 Aspartate Aminotransferase (AST)*

The aspartate aminotransferase (U/L) was 49.00 ± 3.97 and 49.00 ± 4.47 before and after premedication respectively. It was 42.50 ± 5.04, 47.67 ± 5.38 and 145.83 ± 49.57 during anaesthesia, recovery, and at 24 hours respectively. There was marginal increase in AST value after premedication and a non significant increase at 24 hours post operatively. It was decreased during anaesthesia and recovery without any significance (Fig.20).

#### *4.2.5.4 Alanine Aminotransferase (ALT)*

The alanine aminotransferase (U/L) was 51.00 ± 4.21 and 52.33 ± 3.08 before and after premedication respectively. It was 45.17 ± 2.77, 51.17 ± 3.76 and 62.33 ± 8.83 during anaesthesia, recovery and at 24 hours respectively. The ALT value increased during recovery and at 24 hours. There was decrease in ALT value after premedication and during anaesthesia without any significance (Fig.21).

#### *4.2.5.5 Total Protein (TP)*

The total protein (g/dl) was 6.15 ± 0.85 and 5.77 ± 0.83 before and after premedication respectively. It was 5.12 ± 0.70, 5.05 ± 0.67 and 5.78 ± 0.70 during anaesthesia, recovery, and at 24 hours respectively. There was significant

decrease ($p<0.05$) in total protein value after premedication, during anaesthesia and recovery. It was nonsignificantly decreased at 24 hours (Fig.22).

### 4.2.6 Postanaesthetic and Postoperative Observations (Plate 10)

All the animals had rapid, smooth and uneventful recovery from anaesthesia. The surgical wound healing took normal duration and was uneventful.

**Table 2. Signalment, disease/condition and surgery performed in Group I animals**

| Animal Number | Age (years) | Sex | Body weight (kg) | Breed | Disease/Condition | Surgery performed |
|---|---|---|---|---|---|---|
| $I_1$ | 9 | Female | 27.00 | German Shepherd Dog | Mammary tumour – Benign simple adenoma | Tumour resection |
| $I_2$ | 10 | Male | 20.00 | Non Descript | Tumour elbow joint – Kennel granuloma | Tumour resection |
| $I_3$ | 3 | Male | 9.50 | Spitz | Perineal hernia | Herniorrhaphy |
| $I_4$ | 6 | Male | 19.50 | Doberman | Perineal hernia | Herniorrhaphy |
| $I_5$ | 3 | Female | 21.50 | Non Descript | Neutering | Oopherectomy |
| $I_6$ | 14 | Female | 7.30 | Welsh Corgi | Inguinal hernia | Herniorrhaphy |
| Mean ± SE | 6.33± 1.94 | – | 17.47± 3.08 | – | – | – |

**Table 3. Signalment, disease/condition and surgery performed in Group II animals**

| Animal Number | Age (years) | Sex | Body weight (kg) | Breed | Disease/Condition | Surgery performed |
|---|---|---|---|---|---|---|
| $II_1$ | 8 | Male | 29.00 | German Shepherd Dog | Tumour elbow joint level – Malignant fibrous histiocytoma | Tumour resection |
| $II_2$ | 9 | Female | 20.30 | Doberman | Pyometra | Ovario-hysterectomy |
| $II_3$ | 9 | Female | 7.50 | Dachshund | Pyometra | Ovario-hysterectomy |
| $II_4$ | 13.5 | Female | 6.00 | Lhasa Apso | Pyometra | Ovario-hysterectomy |
| $II_5$ | 5 | Female | 6.80 | Spitz | Pyometra | Ovario-hysterectomy |
| $II_6$ | 2.5 | Female | 23.00 | German Shepherd Dog | Dystocia | Caesarean section |
| Mean ± SE | 7.83± 1.54 | – | 15.43± 4.05 | – | – | – |

**Table 4. Anamnesis of Animals in Group I**

| Animal Number | Present history | Duration of illness | Past history | Past treatment | Immediate present treatment |
|---|---|---|---|---|---|
| $I_1$ | A growth on right caudal inguinal mammary gland. | 6 months | 7 whelpings. Last whelping was 8 months back. | Antibiotic treatment for otitis | Nil |
| $I_2$ | A growth on right elbow joint level | 3 months | Enlargement in the size of the growth. | Nil | Nil |
| $I_3$ | A soft reducible swelling on right perineal region | 1 month | Increase in the size of the swelling | Administered laxatives | Nil |
| $I_4$ | A soft reducible swelling on right perineal region | 2.5 weeks | Increase in the size of the swelling | Perineal herniorrhaphy performed on left side one month back. | Nil |
| $I_5$ | Brought for Spaying | Nil | Alopecia | Administered ivermectin 3 months back | Nil |
| $I_6$ | A large sized soft swelling on right inguinal region | 1.5 years | Incidence of inguinal hernia 4 years back. | Inguinal herniorrhaphy 4 years back. | Nil |

**Table 5. Anamnesis of animals in Group II**

| Animal Number | Present history | Duration of illness | Past history | Past treatment | Immediate present treatment |
|---|---|---|---|---|---|
| $II_1$ | A growth on left elbow joint level | 3 months | Enlargement in the size of the growth. | Administered antibiotics and absorbents | Oral antibiotics and multivitamin |
| $II_2$ | Purulent yellow coloured discharge from vulva, anorexia. | 2 weeks | No mating so far. No improvement in disease condition with medical treatment. | Administered antibiotics, parenteral fluids and multivitamins. | Animal was on parenteral antibiotic and fluids. |
| $II_3$ | Purulent discharge from vulva, anorexia | 2 weeks | No mating so far. No improvement in disease condition with medical treatment. | Administered antibiotics, parenteral fluids and multivitamins. | Animal was on parenteral antibiotic and fluids. |
| $II_4$ | White discharge from vulva, anorexia and vomiting | 2.5 weeks | Animal improved in condition with the medical treatment adopted. | Administered antibiotics, parenteral fluids and multivitamins. | Animal was on parenteral antibiotic and fluids. |
| $II_5$ | White discharge from vulva, anorexia. | 1 week | Animal improved in condition with the medical treatment adopted. | Administered antibiotics, parenteral fluids and multivitamins. | Animal was on parenteral antibiotic and fluids. |
| $II_6$ | Whelping not in progress. | 2 days | On ultra sound scanning, foetal skeleton observed, no heart beat. Passed 64 days of gestation. | Medical induction of whelping | Traction per vaginum failed. |

**Table 6. Clinical observations on physiological status and rating of ASA status level for anaesthesia in Group I animals**

| Animal Number | Respiratory (rate/min ) | Pulse (rate/ min) | Rectal temperature (°C) | Colour of visible mucus membrane | VPRC (%) | Total protein (g/dl) | ASA status level |
|---|---|---|---|---|---|---|---|
| $I_1$ | 31 | 104 | 39.4 | Pale roseate | 36 | 6.9 | II |
| $I_2$ | 32 | 111 | 39.2 | Pale roseate | 42 | 5.9 | II |
| $I_3$ | 32 | 100 | 38.8 | Pale roseate | 47.2 | 6.7 | I |
| $I_4$ | 22 | 101 | 39.8 | Pale roseate | 47 | 6.2 | I |
| $I_5$ | 24 | 106 | 39.8 | Pale roseate | 38.3 | 5.9 | I |
| $I_6$ | 31 | 110 | 39.8 | Pale roseate | 42.2 | 8 | II |

**Table 7. Clinical observations on physiological status and rating of ASA status level for anaesthesia in Group II animals**

| Animal Number | Respiratory (rate/min) | Pulse (rate/ min) | Rectal temperature (°C) | Colour of visible mucus membrane | VPRC (%) | Total protein (g/dl) | ASA status level |
|---|---|---|---|---|---|---|---|
| $II_1$ | 28 | 102 | 38.8 | Pale | 13.3 | 6 | IV |
| $II_2$ | 24 | 108 | 39.4 | Pale | 26.3 | 7 | IV |
| $II_3$ | 26 | 104 | 38.6 | Congested | 33 | 6 | III |
| $II_4$ | 24 | 106 | 39.2 | Congested | 36.2 | 8.7 | III |
| $II_5$ | 28 | 108 | 39.4 | Congested | 31.8 | 2.4 | III |
| $II_6$ | 24 | 93 | 36.2 | Congested | 38.2 | 6.8 | III |

**Table. 8. Assessment on clinical signs of sedation, anaesthesia and recovery in Group I animals**

| Animal Number | Level of sedation | Induction quality | Maintenance quality | Recovery quality | Depth of anaesthesia | Degree of muscle relaxation |
|---|---|---|---|---|---|---|
| $I_1$ | Sedated | Good | Excellent | Excellent | Satisfactory | Good |
| $I_2$ | Sedated | Good | Excellent | Excellent | Satisfactory | Good |
| $I_3$ | Slightly sedated | Fair | Good | Excellent | Satisfactory | Excellent |
| $I_4$ | Slightly sedated | Good | Excellent. | Excellent | Satisfactory | Excellent |
| $I_5$ | Deeply sedated | Excellent. | Excellent. | Excellent | Satisfactory | Excellent |
| $I_6$ | Deeply sedated | Excellent. | Excellent. | Excellent | Satisfactory | Excellent |

**Table 9. Assessment on clinical signs of sedation, anaesthesia and recovery in Group II animals**

| Animal Number | Level of sedation | Induction quality | Maintenance quality | Recovery quality | Depth of anaesthesia | Degree of muscle relaxation |
|---|---|---|---|---|---|---|
| $II_1$ | Sedated | Good | Good | Excellent | Satisfactory | Good |
| $II_2$ | Sedated | Good | Excellent | Excellent | Satisfactory | Good |
| $II_3$ | Deeply sedated | Excellent. | Good | Excellent | Satisfactory | Excellent |
| $II_4$ | Sedated | Good | Excellent | Excellent | Satisfactory | Excellent |
| $II_5$ | Sedated | Good | Excellent | Excellent | Satisfactory | Excellent |
| $II_6$ | Deeply sedated | Excellent. | Excellent | Excellent | Satisfactory | Excellent |

**Table 10. Observations on sevoflurane vaporiser concentration for maintenance of anaesthesia in Group I and II animals (Mean ± SE)**

| Animal Group | Sevoflurane vaporiser concentration (%) | | | |
|---|---|---|---|---|
| | At the beginning** | Deep plane of anaesthesia | Medium plane of anaesthesia | Light plane of anaesthesia |
| I | 6.17 ± 0.17 | 6.17 ± 0.17 | 3.83 ± 0.11 | 2.67 ± 0.11 |
| II | 5.33 ± 0.11 | 5.33 ± 0.11 | 3.67 ± 0.11 | 2.58 ± 0.08 |

** $p < 0.01$

**Table 11. Observations on time of induction, duration of anaesthesia and recovery time in Group I and II animals (Mean ± SE)**

| Animals | Time of induction (min) | Duration of anaesthesia (min) * | Recovery time (min) * | | | |
|---|---|---|---|---|---|---|
| | | | Extubation time (min) | Head lift time (min) | Sternal recumbency time (min) | Standing time (min) |
| Group I | 1.17 ± 0.17 | 56.67 ± 4.09 | 3.17 ± 0.48 | 8.50 ± 1.41 | 12.50 ± 2.06 | 18.50 ± 3.19 |
| Group II | 0.92 ± 0.05 | 72.50 ± 4.47 | 5.00 ± 0.26 | 13.17 ± 0.79 | 18.50 ± 1.31 | 29.00 ± 1.75 |

* $p < 0.05$

**Table. 12. Observations on physiological parameters of Group I and II animals (Mean ± SE)**

n = 6

| Parameters | Group | Premedication | | During anaesthesia (30 min) | During recovery (30 min) | At 24 h |
|---|---|---|---|---|---|---|
| | | Before | After | | | |
| Rectal temperature (°C) | I | 39.47± 0.17 | 38.90± 0.36 | 38.12± 0.43 * | 38.48± 0.39 | 38.85± 0.27 |
| | II | 38.60 ± 0.50 | 38.07± 0.41 * | 37.40± 0.37** | 38.23± 0.25 | 38.47± 0.48 |
| Pulse rate (per min) | I | 105.33± 1.86 | 110.83± 2.20 ** | 118.83± 3.76 ** | 110.67± 2.70* | 105.00± 1.71 |
| | II | 103.50± 2.31 | 108.67± 1.58 ** | 117.50± 2.60 ** | 110.00± 1.61** | 101.83± 2.44 |
| Respiratory rate (per min) | I | 28.67± 1.82 | 25.00± 1.91 ** | 17.33± 1.48 ** | 23.00± 1.55 ** | 28.33± 1.80 |
| | II | 25.67± 0.80 | 20.67± 0.95 ** | 16.50± 1.48** | 24.83± 1.33 | 28.00± 0.86 ** |
| Peripheral haemoglobin oxygen saturation -$SpO_2$% (Pulse Oximetry) | I | 97.83± 0.31 | 97.00± 0.45** | 98.67± 0.61 | 97.83± 0.48 | 96.50± 0.34* |
| | II | 96 .00± 0.52 | 95.17± 0.60** | 98.00± 0.68* | 95.67± 0.84 | 95.00± 0.37* |
| Capillary refill time (sec) | I | 1.00± 0.00 | 1.33± 0.05** | 1.75± 0.16** | 1.42± 0.11* | 1.00± 0.00 |
| | II | 1.08± 0.08 | 1.54± 0.10** | 2.17± 0.14** | 1.67± 0.08** | 1.08± 0.08 |
| Clotting time (min) | I | 3.17± 0.11 | 3.67± 0.11 | 4.17± 0.11 | 3.79± 0.15** | 3.33± 0.21 |
| | II | 3.92± 0.20 | 3.92± 0.20 | 4.25± 0.11* | 4.00± 0.22 | 3.92± 0.20 |

* $p < 0.05$ ** $p < 0.01$

**Table.13. Observations on haematological parameters of Group I and II animals (Mean ± SE)**

| Parameters | Group | Premedication | | During anaesthesia (30 min) | During recovery (30 min) | At 24 h |
|---|---|---|---|---|---|---|
| | | Before | After | | | |
| Haemoglobin concentration(g/dl) | I | 14.70± 0.69 | 14.62± 0.51 | 12.25± 1.08 * | 13.08± 0.41 | 12.25± 1.36 |
| | II | 10.80± 1.37 | 9.68 ± 1.12 * | 8.50± 1.22** | 7.88± 1.09 ** | 10.10± 1.14 |
| Volume of Packed Red Cells(%) | I | 42.12± 1.84 | 41.48± 1.38 | 36.73 ± 0.10 * | 36.68± 0.91 ** | 39.22± 0.87 |
| | II | 29.80± 3.70 | 27.57± 3.53 * | 23.23 ± 3.17 * | 21.83± 2.87 ** | 28.22± 3.04 |
| Erythrocyte Sedimentation Rate (mm/h) | I | 2.17 ± 0.17 | 2.50 ± 0.22 | 3.50± 0.22** | 3.67± 0.21 ** | 2.33 ± 0.21 |
| | II | 6.67 ± 1.17 | 8.33 ± 1.58 * | 10.83 ± 1.89 ** | 11.33± 1.93 ** | 8.50 ± 1.48 * |
| Total Leukocyte Count ($10^3$ /$mm^3$) | I | 14.50± 1.00 | 14.43± 1.54 | 12.08 ± 1.12 ** | 13.12± 1.40 | 20.33± 2.34 * |
| | II | 21.00± 4.17 | 20.18± 4.00 * | 15.05 ± 3.40 * | 18.43± 3.14 | 31.10± 7.40 |

* $p < 0.05$ * * $p < 0.01$

**Table. 14. Observations on differential leukocyte count of Group I and II animals (Mean ± SE)**

n = 6

| Parameters | Group | Premedication | | During anaesthesia (30 min) | During recovery (30 min) | At 24 h |
|---|---|---|---|---|---|---|
| | | Before | After | | | |
| Lymphocytes (%) | I | 21.08± 2.93 | 19.60± 3.20 | 25.00± 3.98 | 21.33± 3.70 | 21.10± 2.98 |
| | II | 32.83± 2.74 | 29.67± 2.91 * | 28.00± 2.29 | 25.83± 2.30 | 30.33± 2.39 |
| Neutrophils (%) | I | 71.92± 2.73 | 74.83± 3.32 | 70.33± 3.99 | 73.67± 3.82 | 70.00 ± 2.88 |
| | II | 62.17± 2.77 | 66.00± 3.12 ** | 67.00± 3.54 | 68.67± 3.72 | 64.50± 2.35 |
| Eosinophils (%) | I | 4.00 ± 1.34 | 2.40 ± 0.55 | 2.67 ± 0.56 | 2.50 ± 0.34 | 6.00 ± 0.86 |
| | II | 1.83 ± 0.48 | 1.33 ± 0.42 | 1.50 ± 0.43 | 2.00 ± 0.58 | 1.17 ± 0.31 |
| Monocytes (%) | I | 3.00 ± 0.45 | 3.17 ± 0.95 | 2.00 ± 0.37 | 2.50 ± 0.56 | 2.90 ± 0.57 |
| | II | 3.17 ± 1.47 | 3.00 ± 1.06 | 3.33 ± 1.43 | 3.50 ± 1.46 | 3.83 ± 1.30 |
| Basophils (%) | I | 0.00 ± 0.00 | 0.00 ± 0.00 | 0.00 ± 0.00 | 0.00 ± 0.00 | 0.00 ± 0.00 |
| | II | 0.00 ± 0.00 | 0.00 ± 0.00 | 0.17 ± 0.17 | 0.17 ± 0.17 | 0.17 ± 0.17 |

* $p < 0.05$ ** $p < 0.01$

**Table. 15. Observations on biochemical parameters of Group I and II animals (Mean ± SE)**

n = 6

| Parameters | Group | Premedication | | During anaesthesia (30 min) | During recovery (30 min) | At 24 h |
|---|---|---|---|---|---|---|
| | | Before | After | | | |
| Blood Urea Nitrogen (mg/dl) | I | 11.50± 1.34 | 12.50± 1.65 | 12.67± 1.91 | 11.00± 1.46 | 12.00± 1.79 |
| | II | 60.17± 42.03 | 59.33± 40.03 | 61.83± 41.16 | 59.83± 41.70 | 69.17± 44.66 |
| Serum creatinine (mg/dl) | I | 1.05± 0.06 | 1.12± 0.12 | 1.00 ± 0.11 | 1.12± 0.11 | 1.20± 0.29 |
| | II | 3.73± 2.60 | 3.75± 2.42 | 3.70 ± 2.53 | 3.73± 2.50 | 4.75± 2.98 |
| Aspartate aminotransferase (U/L) | I | 39.00 ± 3.85 | 38.83 ± 2.61 | 36.83 ± 6.01 | 36.67 ± 6.46 | 73.50 ± 8.29** |
| | II | 49.00 ± 3.97 | 49.00 ± 4.47 | 42.50 ± 5.04 | 47.67 ± 5.38 | 145.83± 49.57 |
| Alanine aminotransferase (U/L) | I | 32.33 ± 3.65 | 34.33 ± 3.60 | 30.67 ± 2.93 | 29.17 ± 3.03 | 38.50 ± 3.40 |
| | II | 51.00 ± 4.21 | 52.33 ± 3.08 | 45.17 ± 2.77 | 51.17 ± 3.76 | 62.33 ± 8.83 |
| Total Protein (g/dl) | I | 6.60± 0.33 | 6.53± 0.35 | 5.58± 0.26* | 5.32± 0.40* | 6.45± 0.20 |
| | II | 6.15± 0.85 | 5.77± 0.83* | 5.12± 0.70* | 5.05± 0.67* | 5.78± 0.70 |

* $p < 0.05$ ** $p < 0.01$

**Plate 7. Clinical Signs of Sedation (Glycopyrrolate-Xylazine) ($I_5$)**

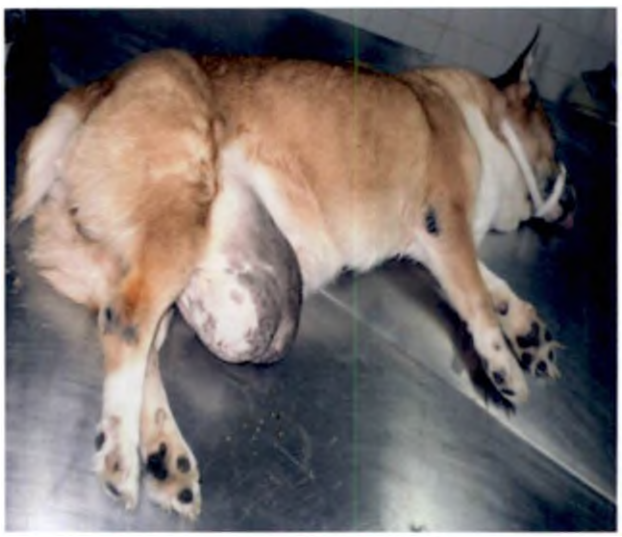
**A. Before Sedation**

**B. Incoordination of Gait**

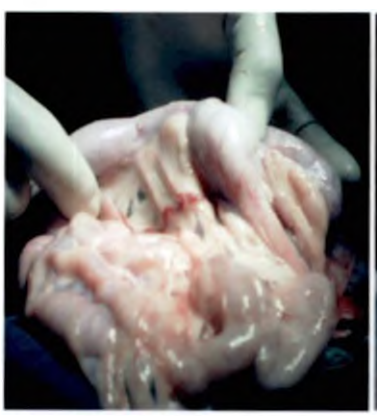
**C. Sitting on Haunches**

**D. Sternal Recumbency with Head Down Posture**

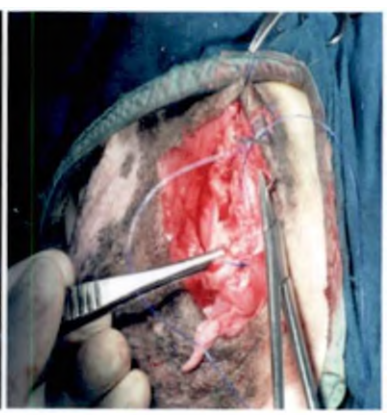
**Plate 8. Surgical Condition and Procedure – Group I Animal ($I_6$)**



**A. Inguinal Hernia**

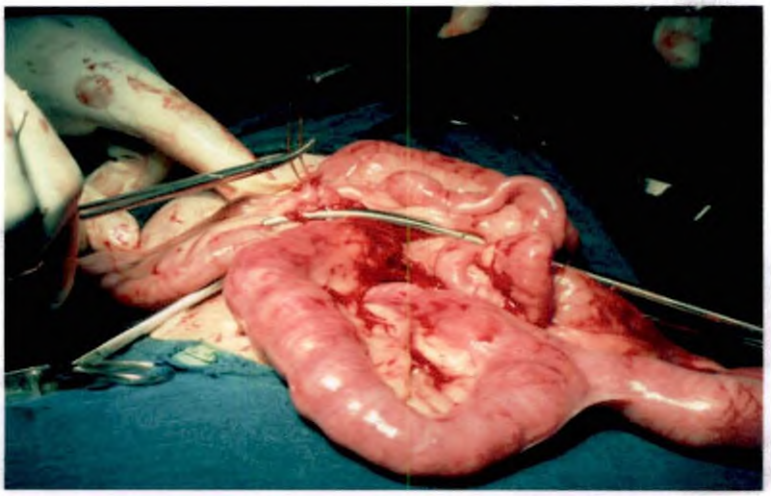


**B. Hernial Sac Contents**

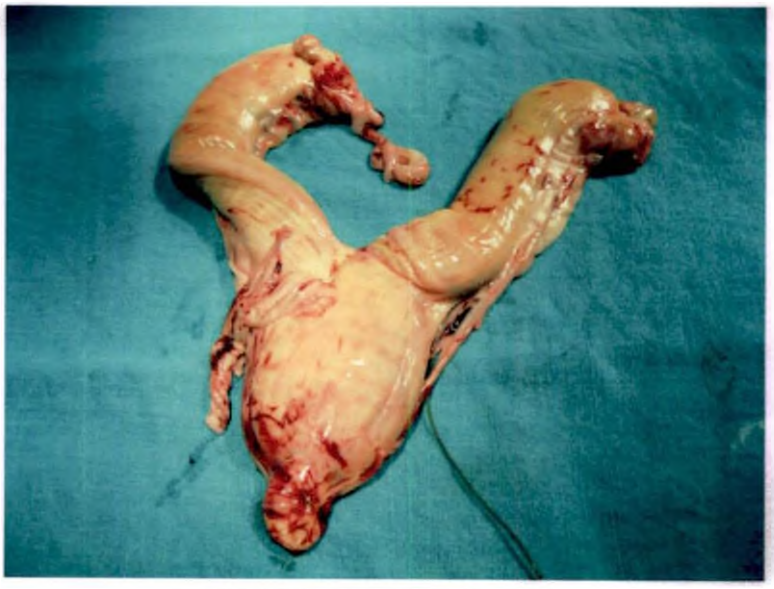


**C. Herniorrhaphy**

**Plate 9. Surgical Procedures - Group II Animals**

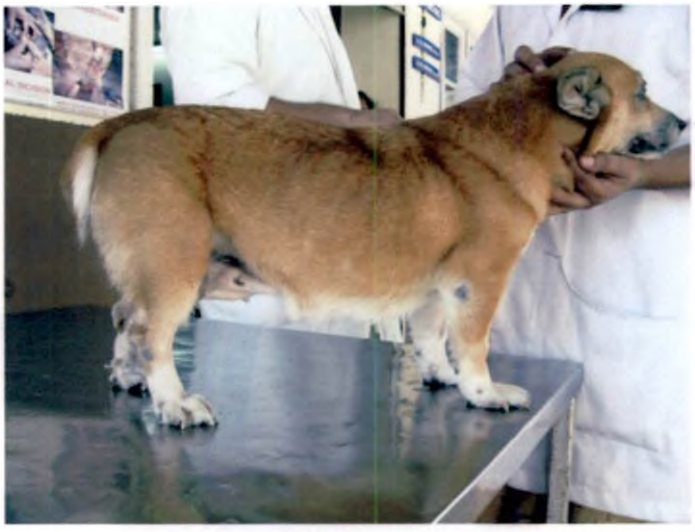


**A. Pyometra Ovariohysterectomy ($II_3$)**



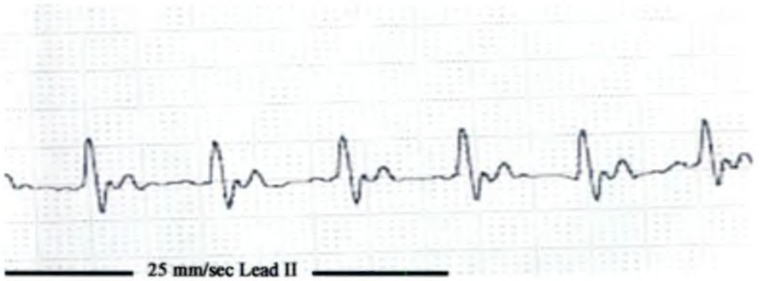
**B. Uterus-Pyometra ($II_2$)**

**Plate 10. Animals – 24 Hours Post Anaesthesia**

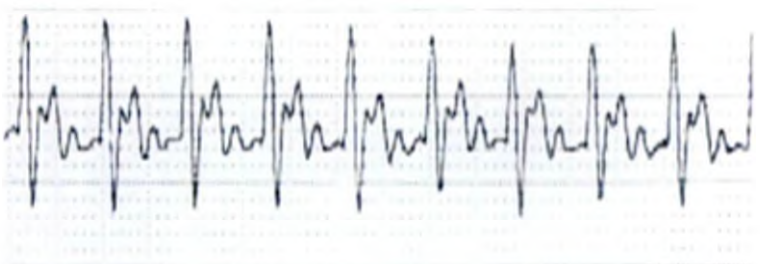


**A. After Inguinal Herniorrhaphy ($I_6$)**

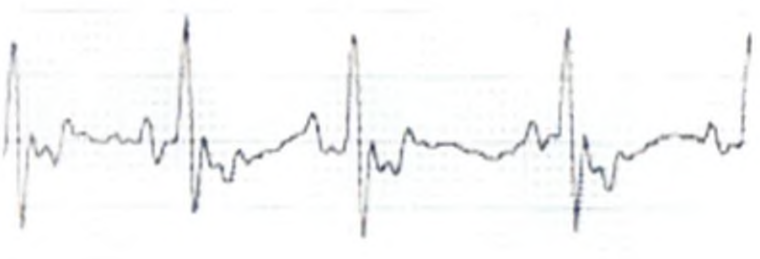
**B. After Pyometra Ovariohysterectomy ($II_4$)**



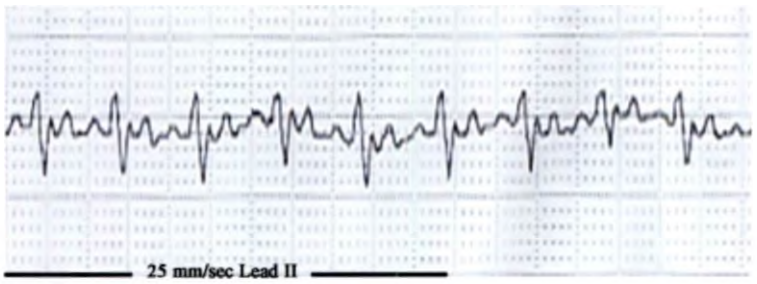

**11 A. Normal Electrocardiogram – Before Premedication ($I_1$)**



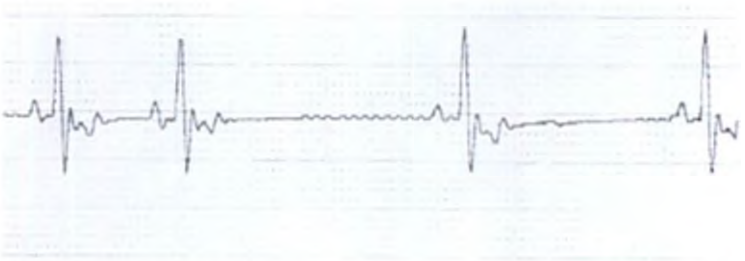
**11 B. Tachycardia – After Premedication ($I_5$)**



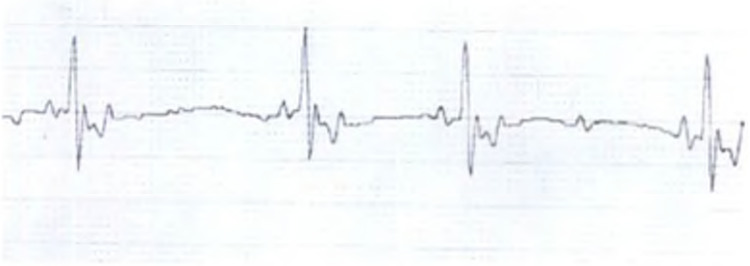
**11 C. First Degree Heart Block – After Premedication ($I_3$)**



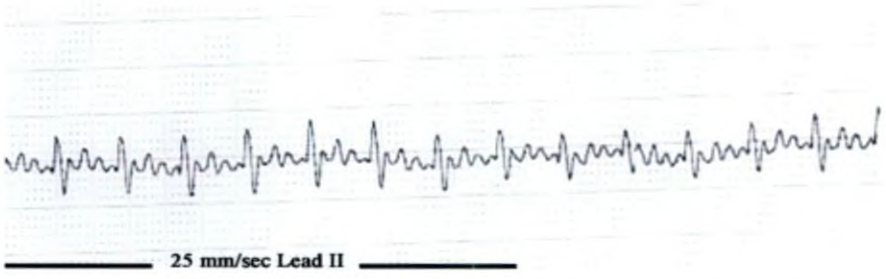

**12 A. Reduction in R Amplititude – During Anaesthesia ($I_1$)**



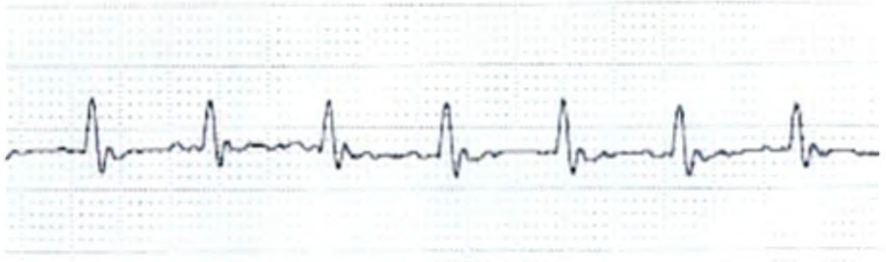
**12 B. Sinus Bradycardia – During Anaesthesia ($I_3$)**



**12 C. Second Degree Heart Block & S-T Slurring – During Anaesthesia ($I_3$)**

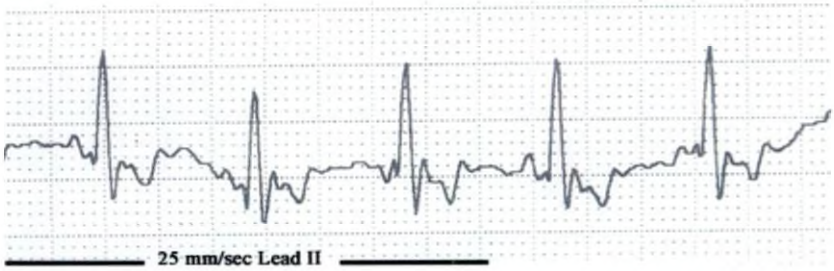



**13 A. Reduced R & T Amplitude – During Anaesthesia ($I_5$)**

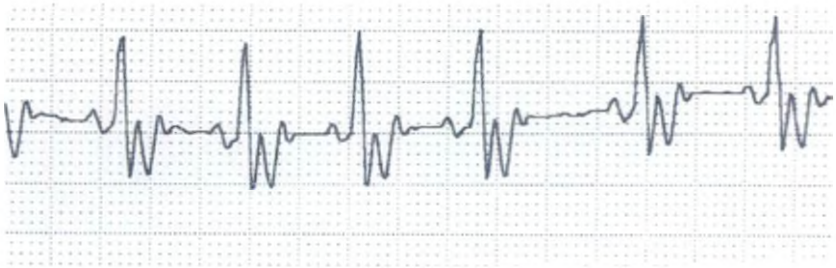


**13 B. Reduced P Amplitude – During Anaesthesia ($II_1$)**

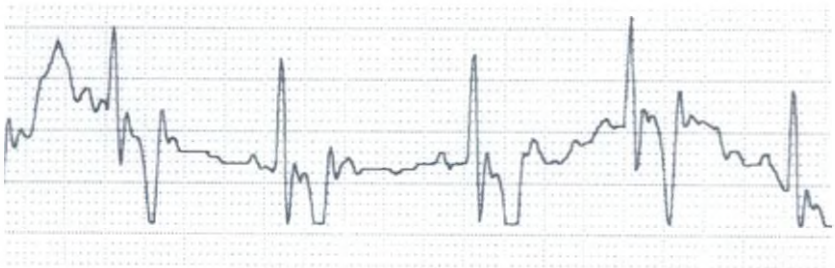
**13 C. High P Amplitude & Changed Polarity of T Wave – During Anaesthesia ($II_4$)**



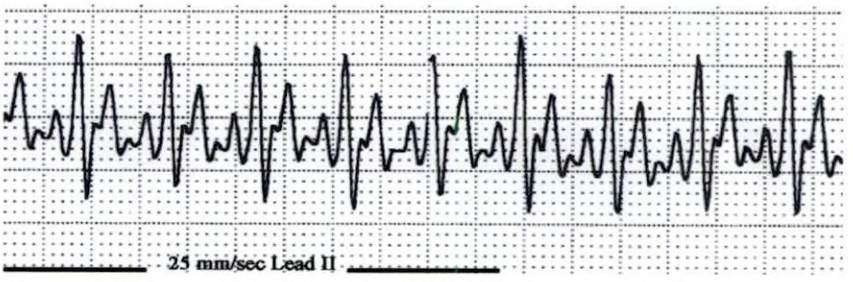

**14 A. Reduced T Amplitude - During Anaesthesia ($II_2$)**



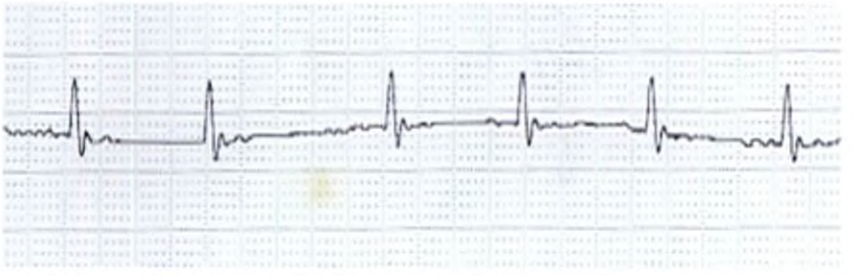
**14 B. S-T Segment Depression – During Anaesthesia ($II_3$)**



**14 C. Peaked T Wave – During Anaesthesia ($II_3$)**




**15 A. Tachycardia - During Anaesthesia ($II_4$)**



**15 B. Single Ventricular Premature Contraction - During Anaesthesia ($II_1$)**

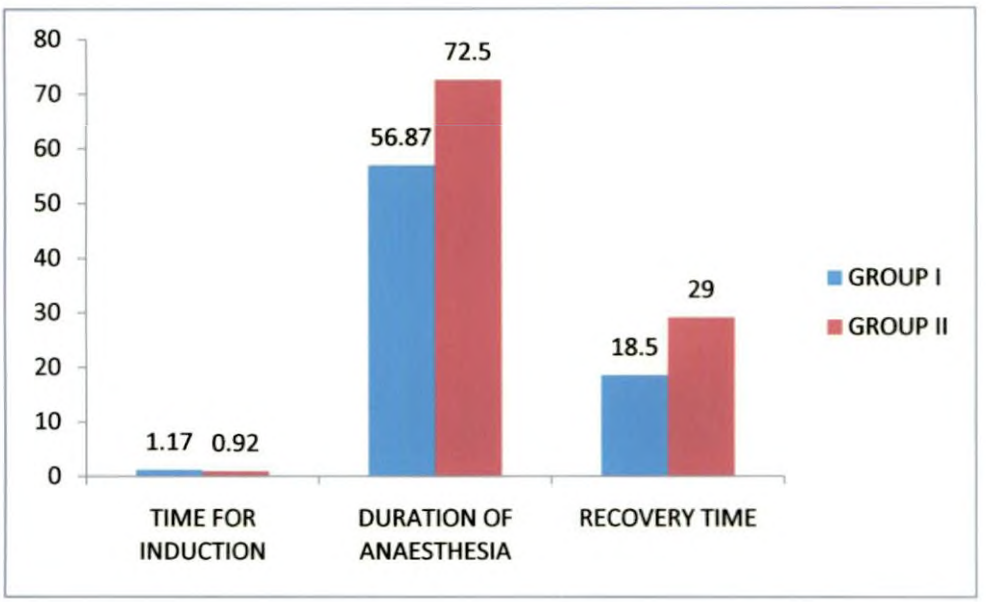



**Fig.1: Mean Time of Induction, Duration of Anaesthesia and Recovery Time in Group I and II Animals**

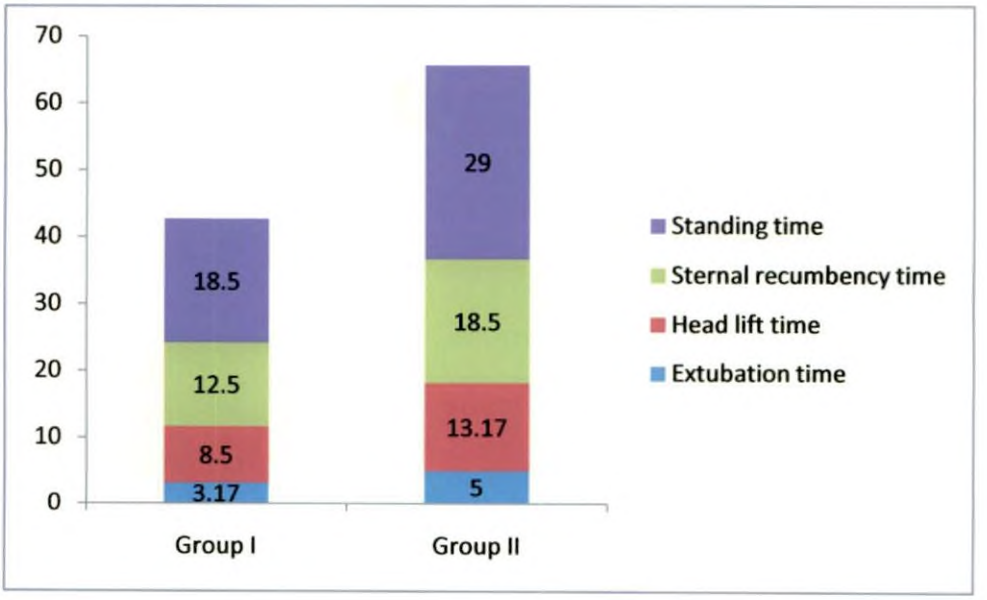



**Fig.2: Different Stages of Mean Recovery Time in Group I and II Animals**

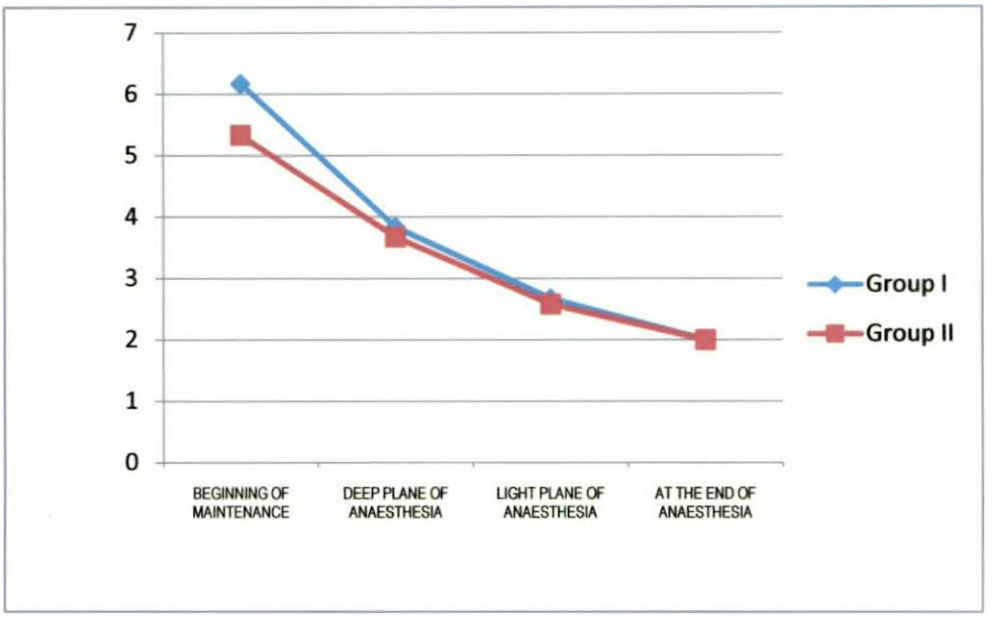



**Fig.3: Mean Vaporiser Concentration of Sevoflurane During Maintenance in Group I and II Animals**

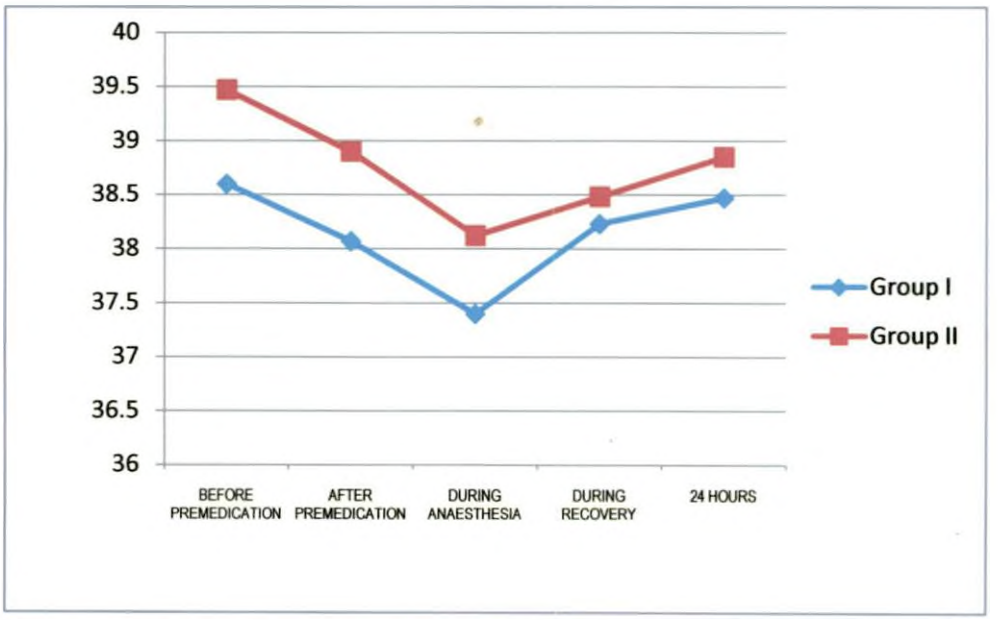



**Fig.4: Mean Rectal Temperature in Group I and II Animals**

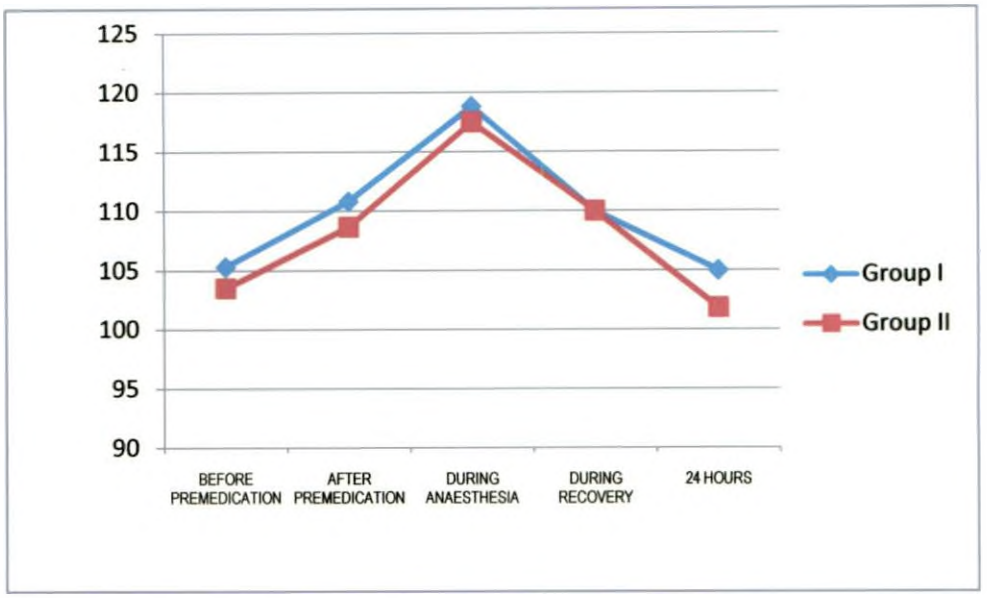



**Fig.5: Mean Pulse Rate in Group I and II Animals**

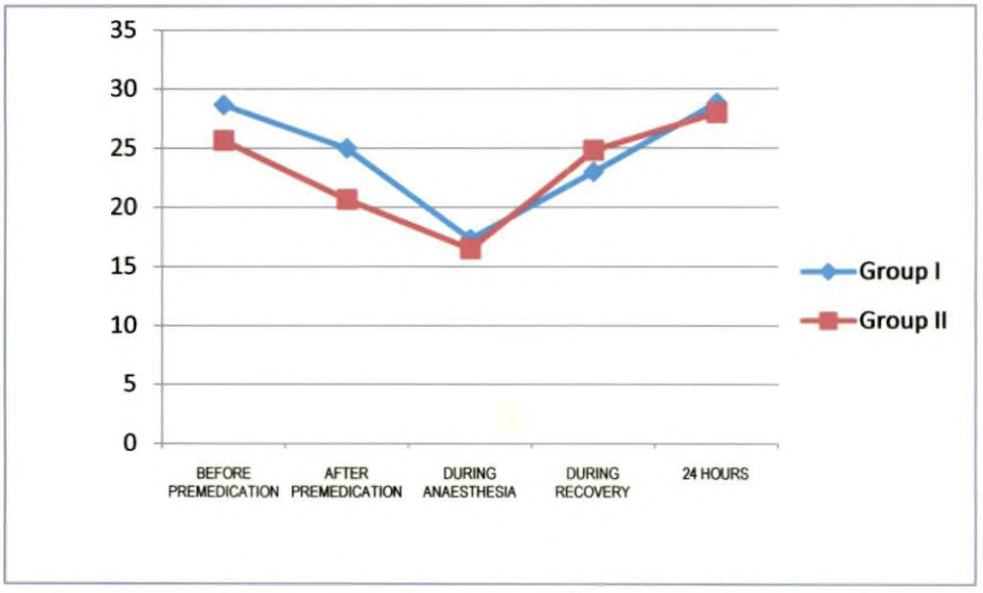



**Fig.6: Mean Respiratory Rate in Group I and II Animals**

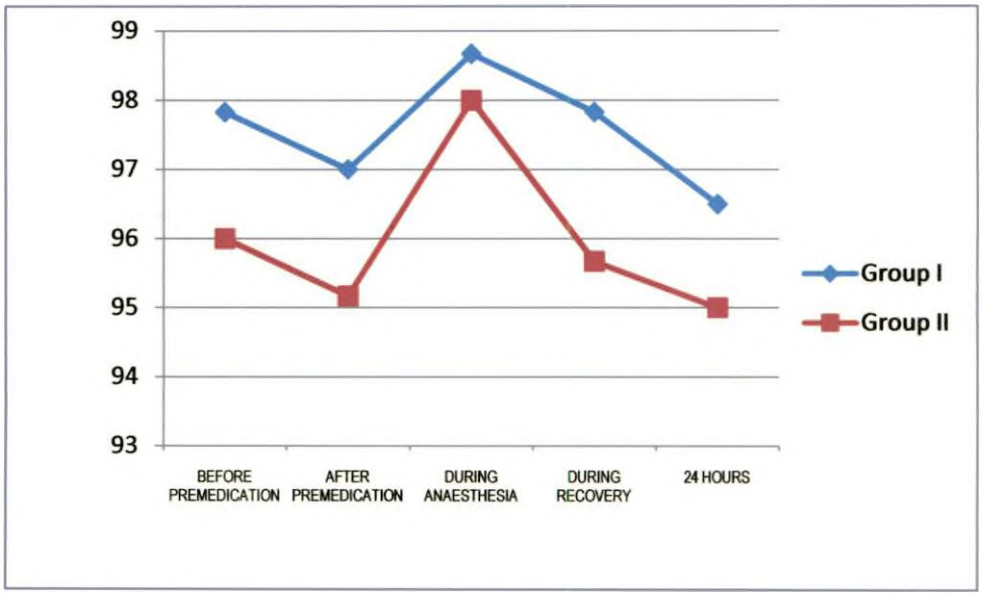



**Fig.7: Mean Peripheral Haemoglobin Oxygen Saturation in Group I and II Animals**

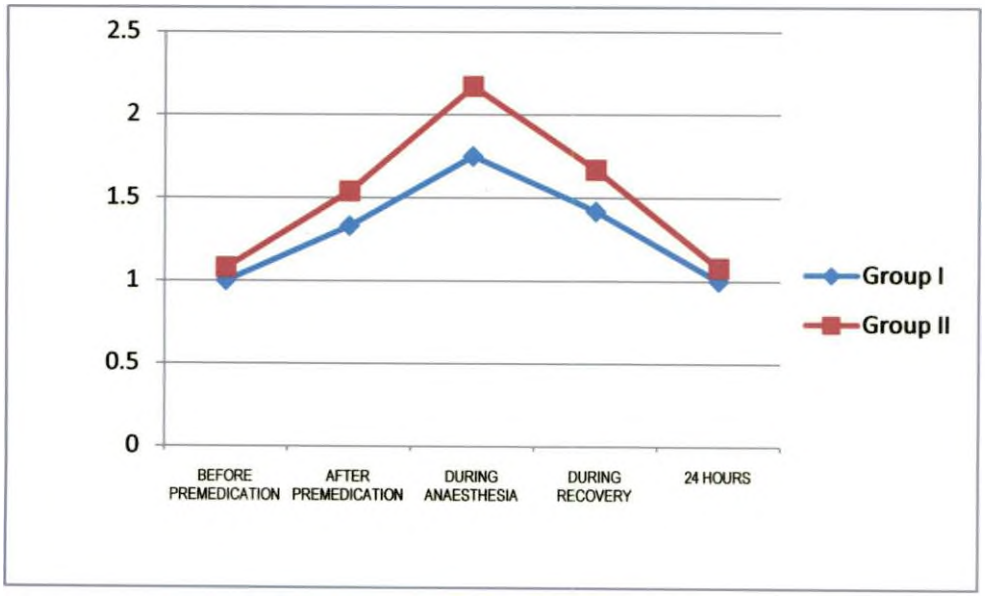



**Fig.8: Mean Capillary Refill Time in Group I and II Animals**

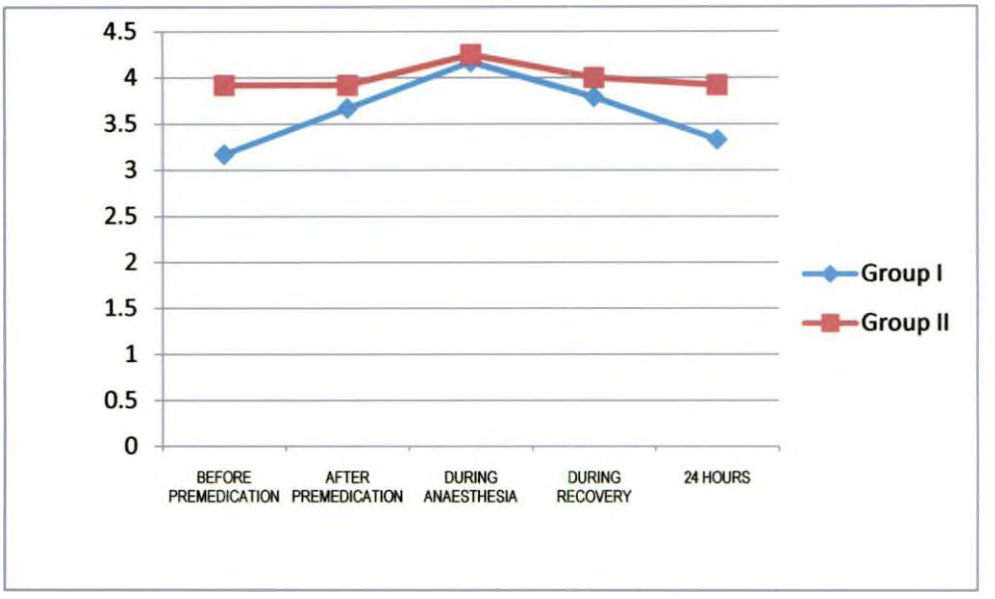



**Fig.9: Mean Clotting Time in Group I and II Animals**

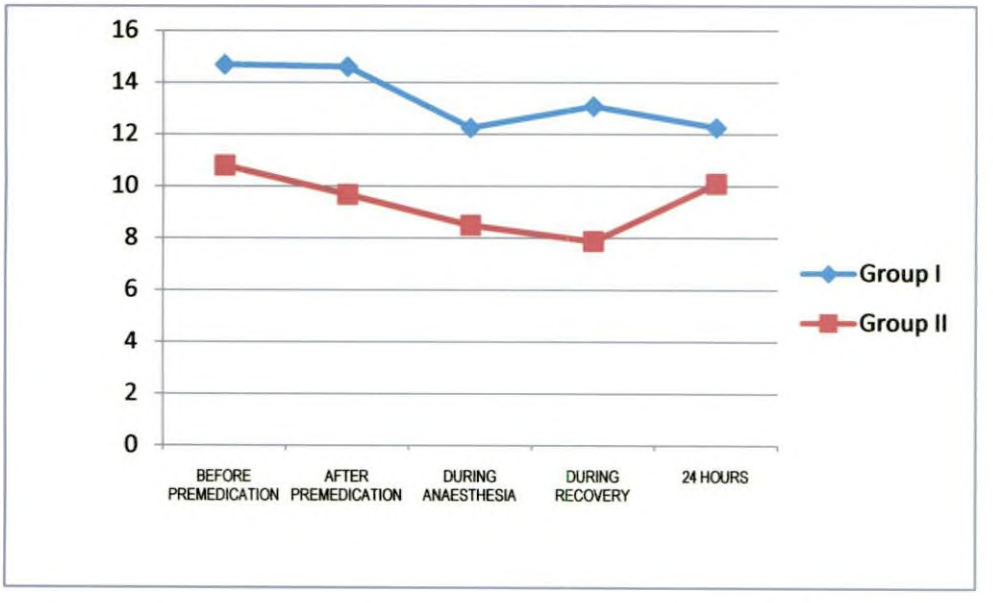



**Fig.10: Mean haemoglobin concentration in Group I and II Animals**

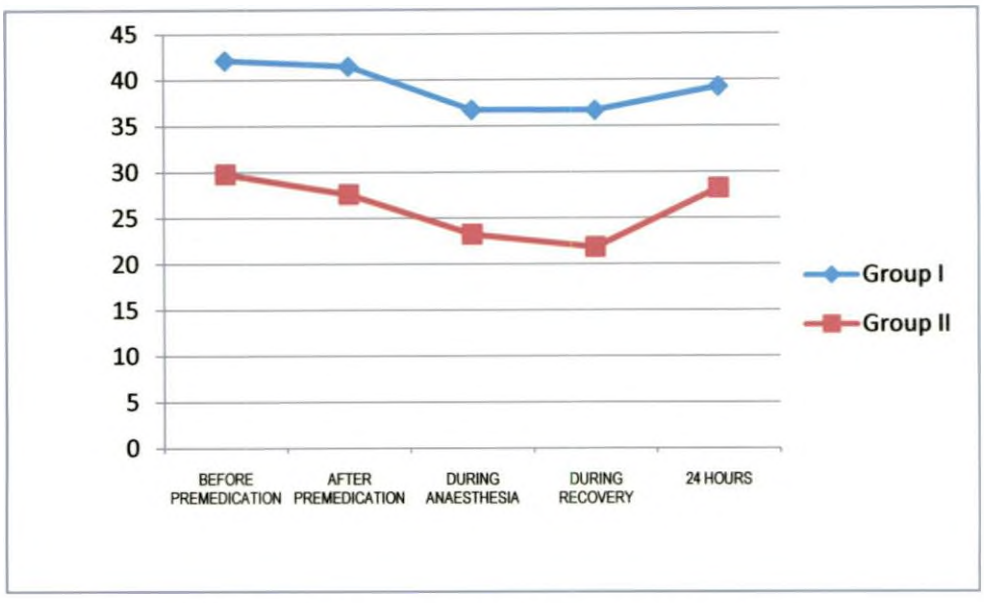



**Fig.11: Mean Volume of Packed Red Cells in Group I and II Animals**

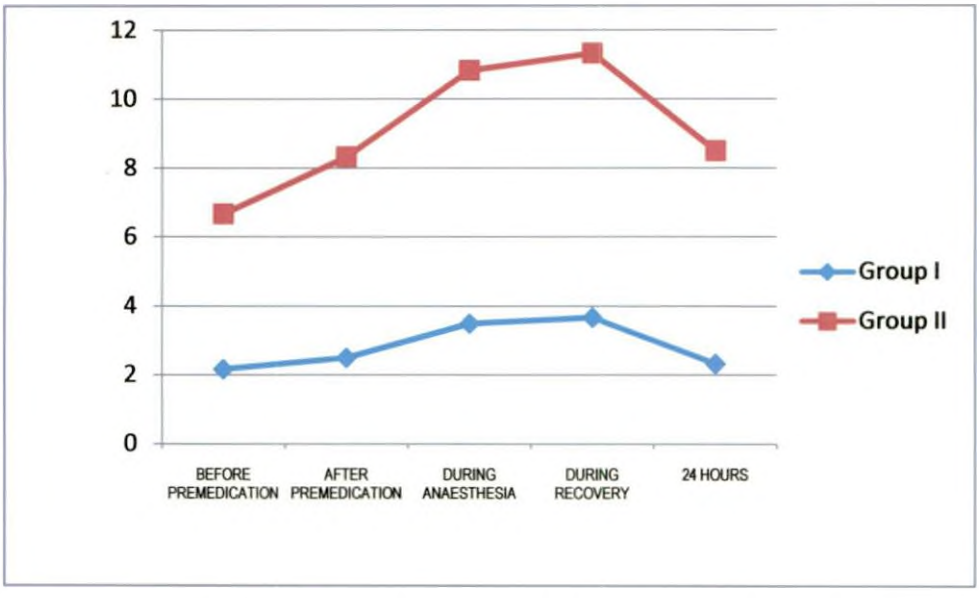



**Fig.12: Mean Erythrocyte Sedimentation Rate in Group I and II Animals**

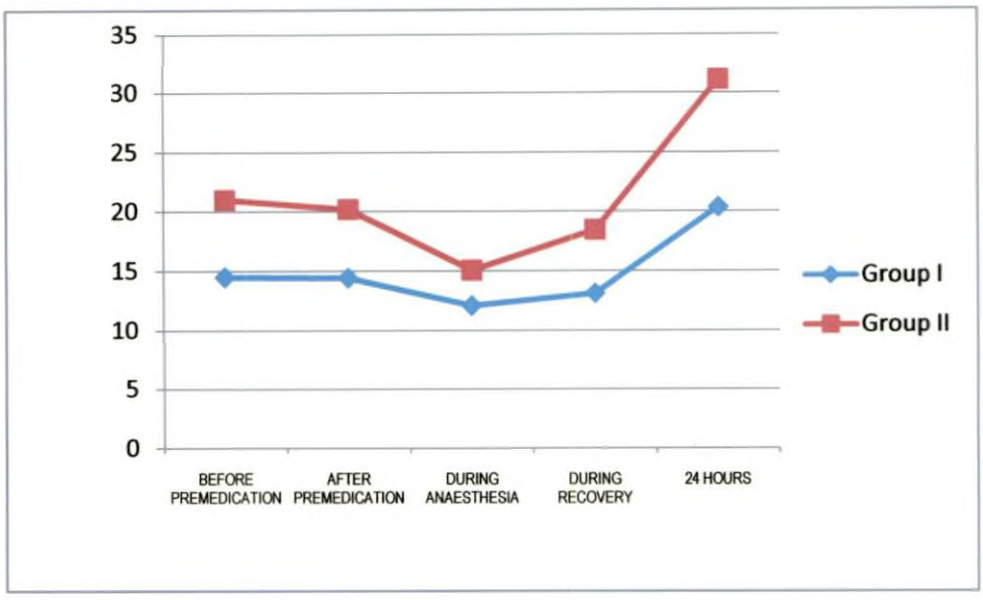



**Fig.13: Mean Total Leukocyte Count in Group I and II Animals**

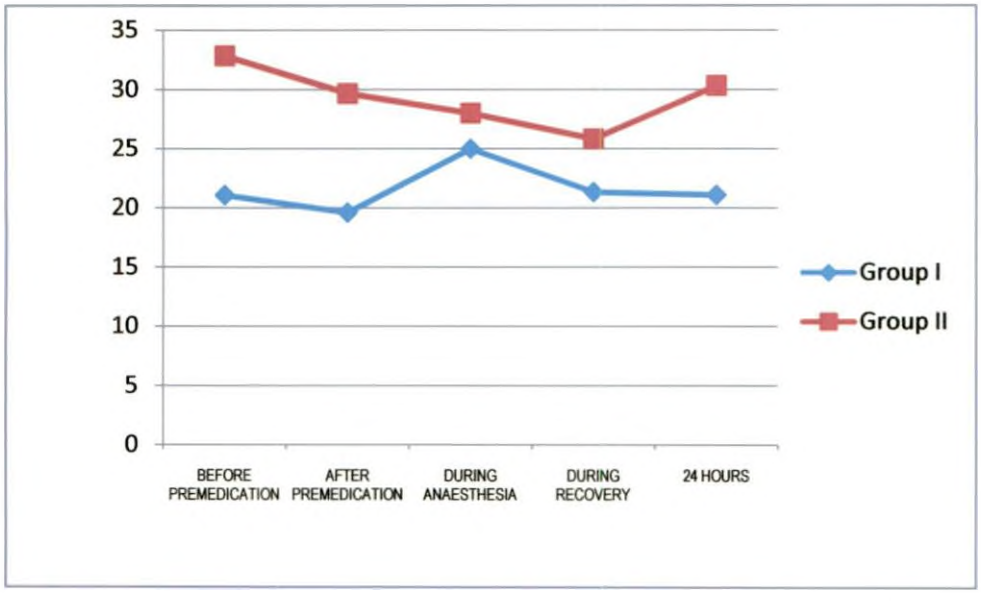



**Fig.14: Mean Lymphocyte Count in Group I and II Animals**

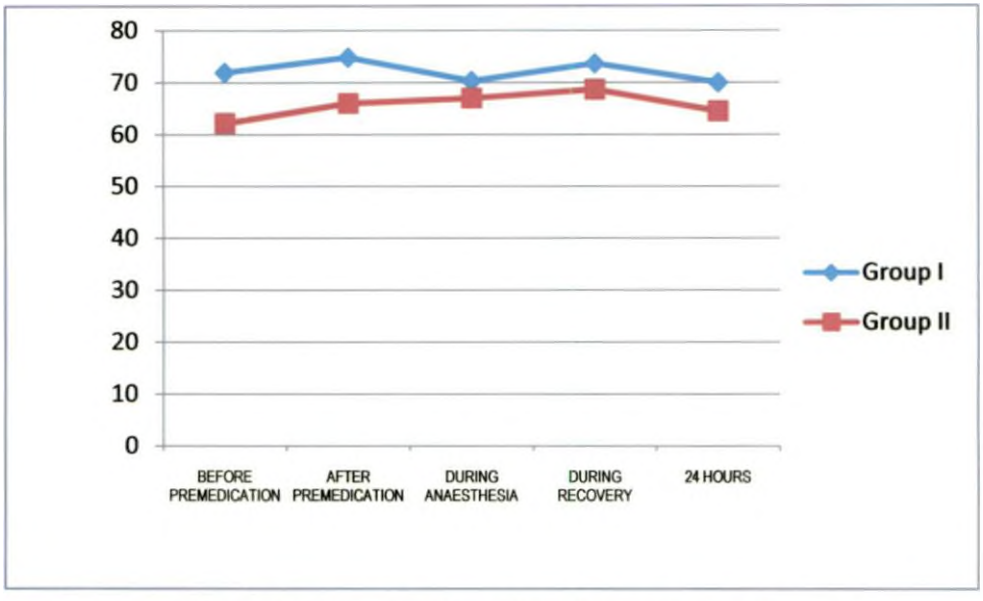



**Fig.15: Mean Neutrophil Count in Group I and II Animals**

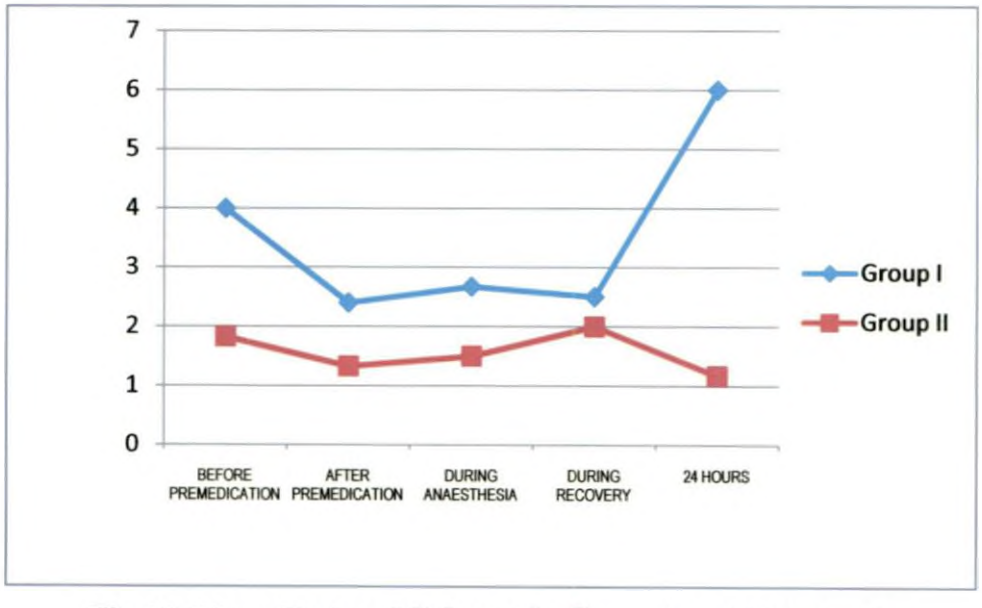



**Fig.16: Mean Eosinophil Count in Group I and II Animals**

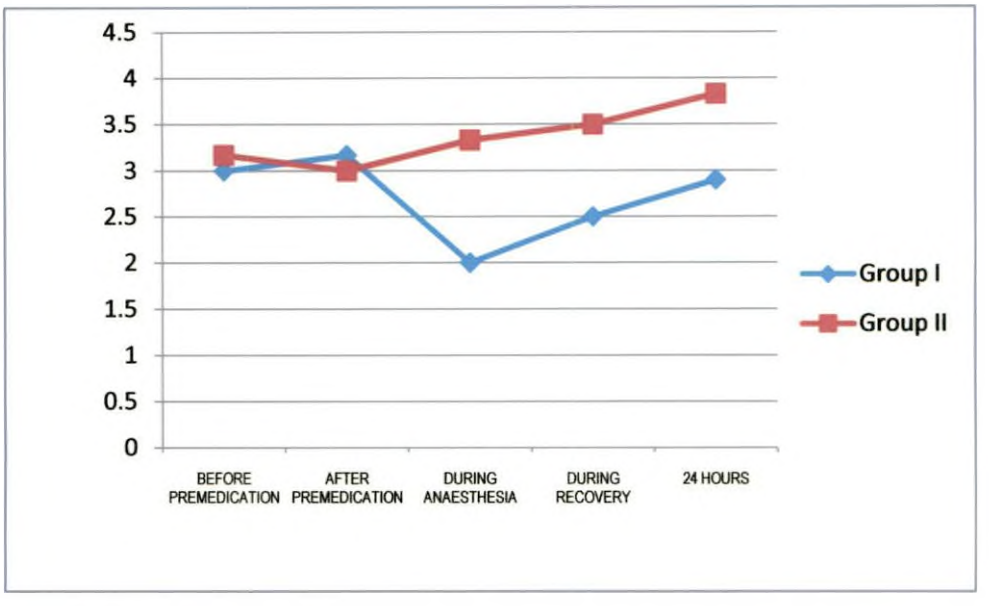



**Fig.17: Mean Monocyte Count in Group I and II Animals**

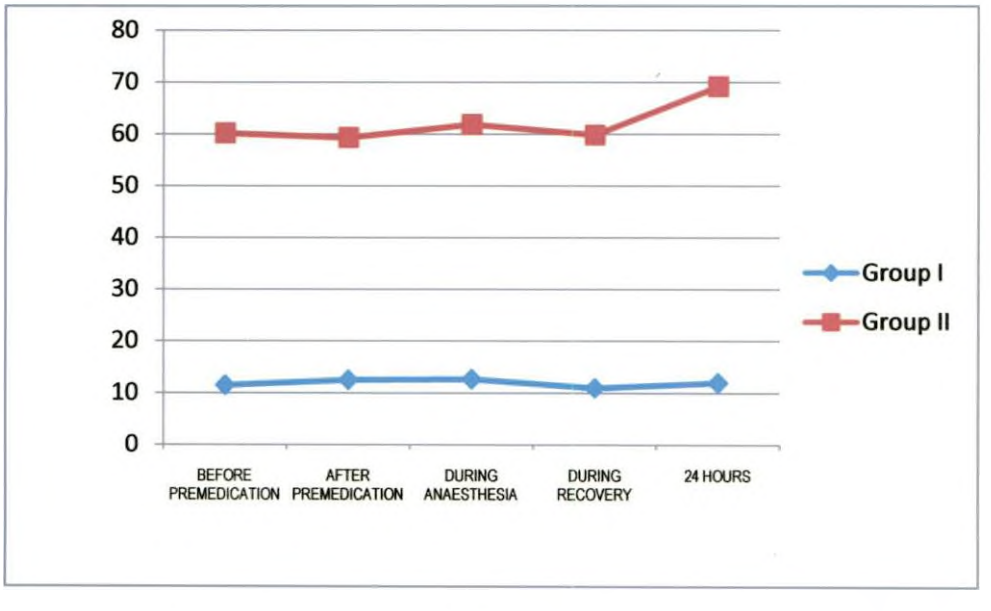



**Fig.18: Mean Blood Urea Nitrogen Level in Group I and II Animals**

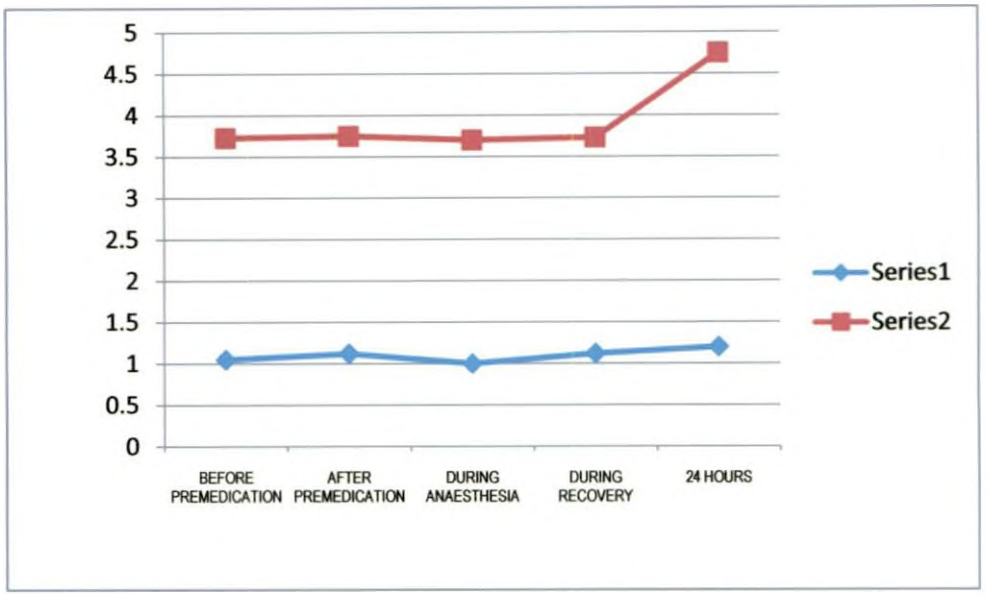



**Fig.19: Mean Serum Creatinine Level in Group I and II Animals**

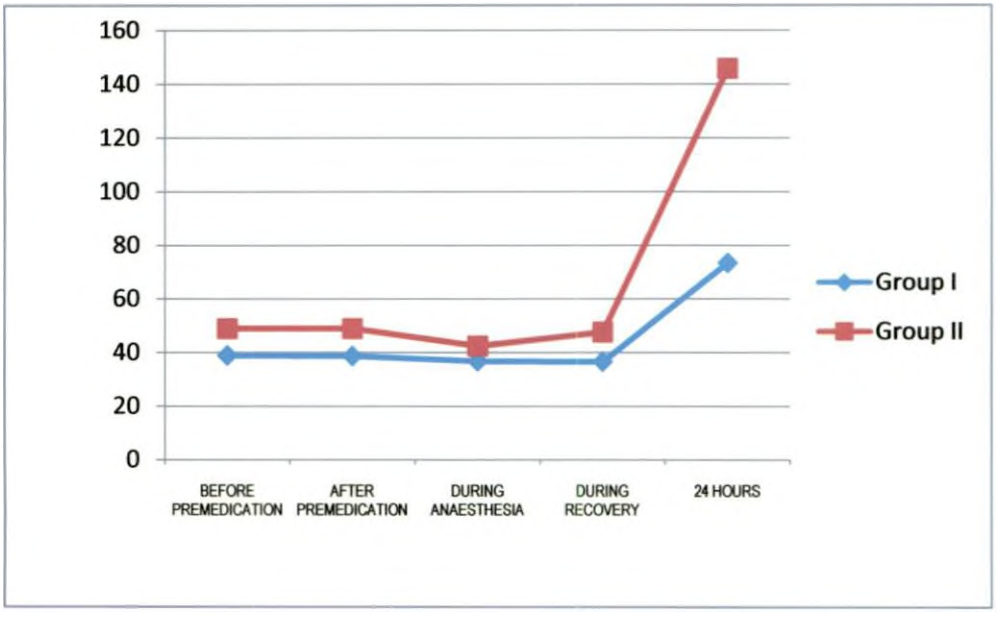



**Fig.20: Mean Aspartate Aminotransferase in Group I and II Animals**

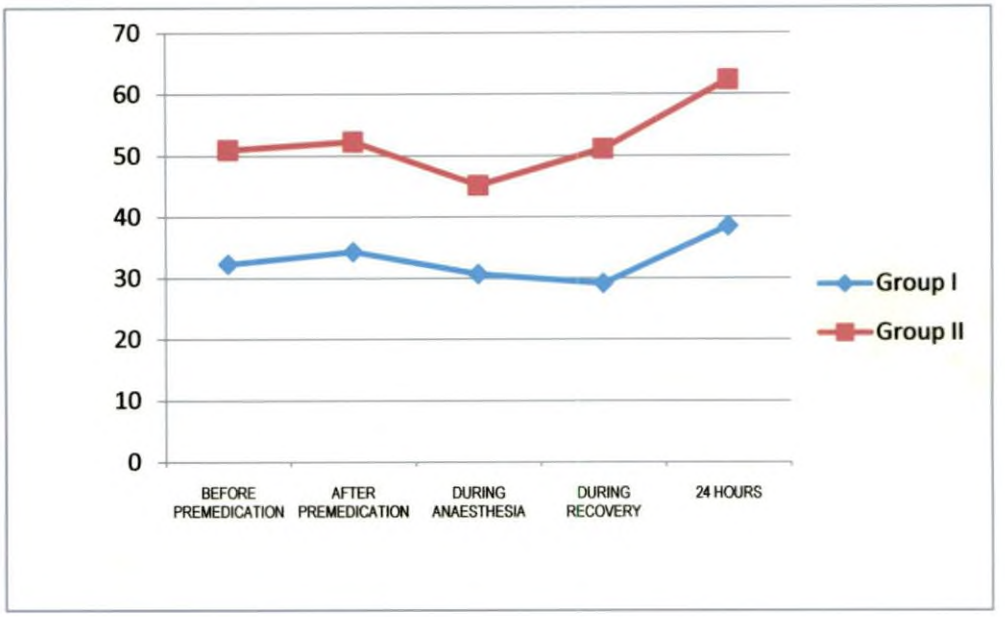



**Fig.21: Mean Alanine Aminotransferase in Group I and II Animals**

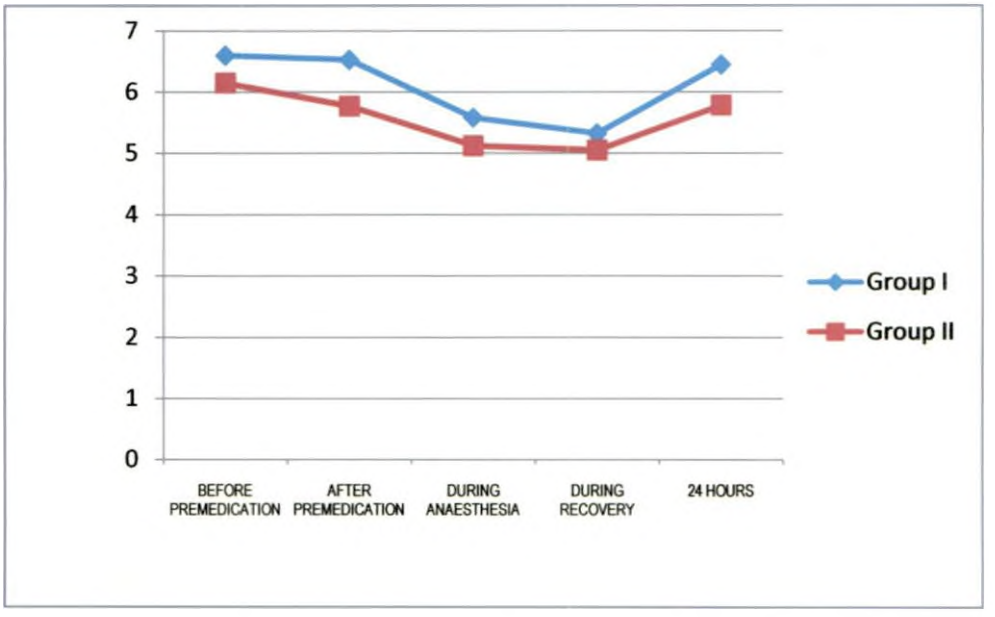



**Fig.22: Mean Total Protein in Group I and II Animals**

# *Discussion*

# 5. DISCUSSION

The study was conducted in 12 dogs of either sex, different age groups, breed and body weight brought for various surgical procedures at Veterinary hospitals Mannuthy and Kokkalai, College of Veterinary and Animal Sciences, Mannuthy.

Six healthy animals presented for surgical procedures were included in Group I and those six animals presented with compromised condition for surgical procedures were included in Group II.

All dogs in Group I were subjected to withholding of food and water for 12 hours prior to anaesthesia and in Group II animals being in compromised condition, avoided such routine preanaesthetic preparations.

Glycopyrrolate at the rate of 0.011 mg/kg body weight, followed by xylazine at the rate of 1 mg/kg body weight at 15 minutes interval were administered intramuscularly as premedication to animals of both the groups. Fifteen minutes later, propofol 1% emulsion at the rate of 4 mg/kg body weight was administered by slow intravenous bolus injection for induction of anaesthesia. Anaesthesia was maintained with sevoflurane (2.5 to 6.5%) in pure oxygen by Bain's circuit system utilising sevoflurane vaporiser.

Anaesthetic evaluation was carried out by continuous monitoring of clinical observations, physiological, haematological, biochemical parameters before and after premedication, during anaesthesia, recovery and at 24 hours.

## 5.1 SIGNALMENT AND ANAMNESIS

There were three male and three female dogs with mean age of 6.33 ± 1.94 years (3 to 14 years) and mean body weight of 17.47 ± 3.08 kg (7.3 to 27 kg) in the Group I. The Group II constituted one male and five female dogs with mean age of 7.83 ± 1.54 years (2.5 to 13.5 years) and mean body weight of 15.43

± 4.05 kg (6.00 to 29 kg). The dogs in both groups were of different breeds. There was no significant difference between groups in the age and body weight.

None of the dogs in the group I had the history of medical or surgical treatment during the past one month. All the dogs in the group II had the history of medical treatment with parenteral antibiotics and fluids from onset of illness. Inspite of medical treatment none of the dogs in Group II had shown promising recovery owing to multi organ involvement and hence decided surgical treatment as next option after stabilisation of the patient's condition.

## 5.2 CLINICAL OBSERVATIONS

The animals were examined for physical condition and their physical status for anaesthesia rated as American Society of Anaesthesiologists (ASA) status level I for three dogs ($I_3$, $I_4$ & $I_5$) and ASA status level II for three dogs ($I_1$, $I_2$ & $I_6$) in the group I; ASA status level III for four dogs ($II_3$ to $II_6$) and ASA status level IV for two dogs ($II_1$ & $II_2$) in the group II. The medical history, levels of volume of packed red cells (VPRC) and total protein (TP) were considered along with physical examination to judge the physical status in accordance with the step followed by Haitjema and Cullen (2001); Bednarski (2007); Laredo *et al.* (2009) and Lozano *et al.* (2009). Group II dogs had comparatively lower VPRC, TP and abnormalities in colour of mucous membrane and physiological functions on account of their compromised nature.

### 5.2.1 Clinical Signs

#### *5.2.1.1 Signs of Sedation*

The level of sedation was assessed based on the clinical signs and the degree of sedation was more pronounced in compromised dogs than in healthy dogs. The salient clinical signs of sedation like winking of eyes, lowering of head, scanty salivation, incoordination of gait, ataxia, and sitting on haunches followed by sternal recumbency with head down posture following xylazine

administration in dogs had also been reported by Rajankutty (1996), Mohan (2006), Narayanan (2007), and Ramankutty (2008). Salivation was scanty in all the dogs of both the groups and attributed to prior administration of the anticholinergic glycopyrrolate (Adams, 1998; Mohan, 2006; Narayanan, 2007; Ramankutty, 2008 and Woods and Sladen, 2009).

### *5.2.1.2 Signs of Induction of Anaesthesia*

The salient clinical signs of anaesthesia induction were transition to lateral recumbency, relaxation of jaw muscles, loss of pedal reflex, sluggish palpebral reflex, ventromedial rotation of eye balls, mild voluntary or involuntary movements and easiness in endotracheal intubation. The voluntary movements were totally absent in Group II animals. Deeply sedated animals ($I_5$, $I_6$, $II_3$ & $II_6$) exhibited excellent induction quality compared to animals with less degree of sedation. Sluggish palpebral reflex and ventromedial rotation of eye ball during induction with propofol are in agreement with the observations by Sooryadas (2001). Effectively abolished jaw tone during induction had been reported by Venugopal *et al.* (2002). Endotracheal intubation was possible in all the dogs which are in agreement with the findings of Mohan (2006) and Ramankutty (2008).

### *5.2.1.3 Signs of Anaesthesia*

The noted signs of anaesthesia were loss of palpebral and pedal reflexes, relaxation of jaw and abdominal muscles and ventromedial rotation of eye balls. The adequacy of anaesthesia during maintenance with sevoflurane to conduct the surgical interventions could be determined by monitoring these clinical signs as well as noting the changes in heart rate, respiratory rate and character as reported by Bennet *et al.* (2008).

### *5.2.1.4 Maintenance Quality*

All the dogs achieved a steady-state level of anaesthesia. The minimal muscle tone and reflex response to pain during surgical manipulation observed in one animal of Group I ($I_3$) and two animals of Group II ($II_1$ & $II_3$) were managed by increasing the depth of anaesthesia. The effect of xylazine and propofol vanished early in the maintenance period (Knight, 1980 and Sooryadas, 2001) and sevoflurane action dominated in major periods of anaesthetic maintenance. Sevoflurane provided a good controllable depth of anaesthesia for the maintenance. The ECG and respiratory rate changes observed in those three animals were transient. The increased degree of stimulation from surgical manipulations might be the reason for the reflex response to pain in three animals and hence depth of anaesthesia was increased as suggested by Soma and Klide (1987).

### *5.2.1.5 Recovery Quality*

Recovery quality was excellent in all the dogs of both the groups and it is in agreement with reports of Haitjema and Cullen (2001), Jadon *et al.* (2008) and Love *et al.* (2007).

### *5.2.1.6 Undesirable Side Effects*

The reduced dose rate of propofol at the rate of 4 mg/kg on account of xylazine premedication along with slow administration of propofol in the present study might have contributed to stable respiratory conditions and is in agreement with Kojima *et al.* (2002) and Kale *et al.* (2006). The respiratory depression and apnoea as undesirable side effects upon induction with rapid bolus injection of propofol reported by Muir III and Gadawski (1998), Kuusela, 2003, Sahay and Dass (2005), Sharma and Bhargava (2007) and Ramankutty (2008) were not observed in the present study. One animal in Group I ($I_1$) exhibited regurgitation and two animals in Group II ($II_1$ & $II_4$) vocalisation during recovery. These signs might be on account of visceral pain as suggested by Clarke (1999), Mohamadnia

*et al.* (2008) and Lozano *et al.* (2009). The post operative pain exhibited by those animals was managed with intramuscular administration of meloxicam at the rate of 0.2 mg/kg body weight, which is in agreement with the procedure adopted by Bennet *et al.* (2008) and Pottie *et al.* (2008) with sevoflurane anaesthesia. The analgesic effect of xylazine was short lived (Knight, 1980).

The incidence of regurgitation and aspiration of stomach contents were of reduced chance with propofol as reported by Love *et al.* (2007) but it might also be on account of the influence of sevoflurane on gastro esophageal reflux (GER) as reported by Wilson *et al.* (2006). Increased gastric acidity could result in more incidence of GER but glycopyrrolate premedication in the present study reduced such a chance (Short, 1987 and Adams, 1995).

One animal in Group II ($II_6$) exhibited sneezing as an undesirable side effect, which was also reported by Haitjema and Cullen (2001). The other undesirable side effects reported with sevoflurane anaesthesia like shivering, tachypnoea (Haitjema and Cullen, 2001; Branson *et al.*, 2001), apnoea (Branson *et al.*, 2001) and seizure activity (Lozano *et al.*, 2009) during recovery in dogs were not observed in the present study.

**5.2.2 Sevoflurane Vaporiser Concentrations** (Fig. 3)

The significant difference ($p<0.01$) in vaporiser concentration of sevoflurane at the onset of maintenance 'to effect' was observed between the groups; it was 6.17 ± 0.17% (2.61 MAC) in Group I animals and 5.33 ± 0.11% (2.26 MAC) in Group II animals. The higher concentration of sevoflurane used in the beginning of maintenance in the present study was to speed up the rate of equilibration in the alveoli as reported by Mutoh (2007).

The anaesthetic concentration was individually adjusted for each dog and apparently there was no constant relationship between animal size and age with vaporiser setting as reported by Bennet *et al.* (2008) and Yamishita *et al.* (2009).

The sevoflurane vapour concentration used to maintain medium depth of surgical anaesthesia was 3.83 ± 0.11% (1.62 MAC) in Group I animals and 3.67 ± 0.11% (1.56 MAC) in Group II animals, comparatively lower in Group II animals. The concentration of sevoflurane to achieve light plane of anaesthesia in Group I animals was 2.67 ± 0.11% (1.13 MAC) and in Group II animals, it was 2.58 ± 0.11% (1.09 MAC). Deep plane of anaesthesia was achieved with 6.17 ± 0.17% (2.61 MAC) sevoflurane in Group I animals and 5.33 ± 0.11% (2.26 MAC) in Group II animals.

The different sevoflurane concentrations used for anaesthesia reported in dogs were 3.3 per cent by Branson *et al.* (2001); 1.5 MAC by Polis *et al.* (2001); 3 to 3.5% to increase the depth of anaesthesia and 2 to 2.5% to maintain a light plane of anaesthesia by Love *et al.* (2007) and Singh *et al.* (2010). Jadon *et al.* (2008) used 2.5% sevoflurane for maintenance in dogs. Mendes *et al.* (2003) maintained cats for ovariohysterectomy under sevoflurane anaesthesia with vaporiser setting of 3.4% sevoflurane (1.3 MAC in cats).The mean end-tidal concentration of sevoflurane required to maintain anaesthesia in Iberian lynx was 2.8% (Gomez-Villamandos *et al.*, 2007).

There was no significant difference in vaporiser concentration of sevoflurane to achieve deep and light planes of anaesthesia between the two groups. The use of higher sevoflurane vaporiser concentration in the beginning of the maintenance and subsequent gradual reduction in the vaporiser settings later to continue the maintenance adopted in the present study was in agreement with the findings of Branson *et al.* (2001), Haitjema and Cullen (2001) in dogs, Gomez-Villamandos *et al.* (2007) in Iberian lynx and Topal (2008) in rabbits.

**5.2.3 Time for Induction of Anaesthesia** (Fig. 1)

The time for induction of anaesthesia following intravenous injection of propofol in Group I and Group II animals was 1.17 ± 0.17 and 0.92 ± 0.05 min respectively and is in accordance with observations of Ramankutty (2008). The

time for induction of anaesthesia in the present study was shorter when compared to induction of anaesthesia with propofol reported by Sooryadas (2001), Venugopal *et al.* (2002) and Mohan (2006). Time for induction of anaesthesia was comparatively shorter in Group II animals on account of their compromised status and is in agreement with the observation made by Sooryadas (2001). The faster induction using propofol observed in the present study is agreement with Pottie *et al.* (2008), where as induction time with sevoflurane alone for induction was not only longer but also not smooth (Branson *et al.,* 2001 and Pottie *et al.,* 2008).

The analgesic effect of xylazine observed was minimal in the extremities (Knight, 1980) and the pedal reflex observed unaffected with the administration of xylazine (Kandapal *et al.,* 2005) and hence the time of loss of pedal reflex was considered as the time of induction of propofol general anaesthesia.

**5.2.4 Duration of Anaesthesia** (Fig. 1)

The duration of anaesthesia was 56.67 ± 4.09 and 72.50 ± 4.47 min in Group I and II animals respectively. There was significant difference ($p<0.05$) in the duration of anaesthesia between groups. The surgical procedures conducted on Group II animals were of longer duration requiring prolonged anaesthetic maintenance than that of Group I animals.

**5.2.5 Depth of Anaesthesia**

The depth of anaesthesia as assessed from the surgical procedures performed was satisfactory in all the dogs. Sevoflurane enabled to alter the depth of anaesthesia rapidly. Heart and respiratory rates were maintained steadily. The surgical procedures performed were three tumour resections ($I_1$, $I_2$ & $II_1$), three herniorrhaphies ($I_3$, $I_4$ & $I_6$), one oopherectomy ($I_5$), four ovario-hysterectomies for pyometra ($II_2$ to $II_5$) and one caesarean section ($II_6$).

Propofol infusion in the present study produced a moderate level of anaesthesia for a short period of time and the dose of propofol administered was also less (Short and Bufalari, 1999; Kojima *et al.,* 2002; Love *et al.,* 2007 and Pottie *et al.,* 2008). It was during the propofol induced general anaesthesia the tracheal intubation was made possible, but sooner it resulted in appearance of ocular reflexes and occasional spontaneous blinking (Kuusela *et al.,* 2003). Sevoflurane administration immediately achieved the required depth of anaesthesia and it could be confined up to a medium plane of surgical anaesthesia in all the dogs that had undergone different surgical procedures (Mutoh *et al.*, 1997). Further increase was done to deepen the anaesthesia.

Ramankutty (2008) also achieved satisfactory depth of anaesthesia with propofol-isoflurane anaesthesia after glycopyrrolate-xylazine premedication in healthy and compromised dogs subjected to different surgical interventions.

### 5.2.6 Degree of Muscle Relaxation

Out of the twelve animals studied, the degree of muscle relaxation was good in four animals ($I_1$, $I_2$, $II_1$ & $II_2$) and excellent in eight ($I_3$ to $I_6$ and $II_3$ to $II_6$) animals for performing the surgical procedures. Excellent muscle relaxation was observed in majority of both healthy and compromised dogs. The degree of muscle relaxation achieved in the present study is in agreement with Mohan (2006) and Ramankutty (2008).

### 5.2.7 Recovery Time (Fig. 1 & 2)

The recovery time was 18.50 ± 3.19 and 29.00 ± 1.75 min in Group I and II animals respectively. It was significantly prolonged ($p<0.05$) in Group II animals compared to Group I animals.

The drugs other than sevoflurane administered in the anaesthetic protocol might not affect the recovery time (Proakis and Harris, 1978; Knight, 1980; Polis *et al.,* 2001 and Sooryadas, 2001).

This study did not measure the effect of sevoflurane on cardiac output or venous-to-alveolar anaesthetic partial pressure differences; the differences in recovery variables might also be attributable to disparity of these factors (Steffy, 1996). The prolonged duration of surgery and hence the long duration of anaesthesia in compromised dogs might have resulted in increased sympathetic tone. The increased sympathetic tone could increase cardiac output (Mutoh *et al.*1997), which is a major determinant of inhalation agent uptake (Boller, 2005). An increase in cardiac output or decrease in ventilation would prolong recovery time as well as increased solubility of sevoflurane in blood and tissue, particularly in fat as reported by Johnson *et al.* (1998), Clarke (1999) and Boller *et al.* (2005).

Early recovery observed in Group I animals is in agreement with Bennet *et al.* (2008). The recovery time observed in Group I animals in the present study is shorter than with propofol-isoflurane anaesthesia achieved by Ramankutty (2008). Sooryadas (2001) achieved a shorter recovery time of 14.72 min in healthy dogs and 20.9 min in compromised dogs with propofol anaesthesia. Mohan (2006) also achieved a shorter recovery time of 17.66 min with propofol anaesthesia in compromised dogs.

The different time components in the stages of recovery recorded from switching off the sevoflurane vaporiser in the present study were: Extubation time – 3.17 ± 0.48 min in Group I animals, 5.00 ± 0.26 min in Group II animals; Head lift time - 8.50 ± 1.41 min in Group I animals, 13.17 ± 0.79 min in Group II animals; Sternal recumbency time - 12.50 ± 2.06 min in Group I animals, 18.50 ± 1.31 min in Group II animals; Standing time - 18.50 ± 3.19 min in Group I animals; 29.00 ± 1.75 min in Group II animals. These mode of observations with sevoflurane anaesthesia were also recorded by Haitjema and Cullen (2001); Love *et al.* (2007); Bennet *et al.* (2008); Mohamadnia *et al.* (2008) and Lozano *et al.* (2009). The mean time from extubation to sternal recumbency in present study was 9.33 min in Group I animals and 13.5 min in Group II animals, which is in accordance with Branson *et al.* (2001).

## 5.3 PHYSIOLOGICAL PARAMETERS

### 5.3.1 Rectal Temperature (Fig. 4)

There was decrease in rectal temperature after premedication, during anaesthesia (significant in Group I – $p<0.05$; Group II – $p<0.01$), recovery and it was increased near to the preanaesthetic values in both groups by 24 hours post operatively. The decrease in rectal temperature after xylazine premedication was in contrast to the finding of Redondo *et al.* (1999). There was significant decrease ($p<0.05$) in rectal temperature after premedication in Group II animals. Mohan (2006) also reported reduction in rectal temperature with glycopyrrolate-xylazine premedication. The decrease in rectal temperature following xylazine administration had been reported in dogs (Kandapal *et al.,* 2005) and in calves (Sharma *et al.,* 2006 ); following propofol administration in dogs (Muir III and Gadawski, 1998; Venugopal *et al.*, 2002; Kale *et al.,* 2006; Mohan, 2006; Sharma and Bhargava, 2007) and goats (Sahay and Dass, 2005).

Bennet *et al.* (2008) reported that body temperature did not fall below 36°C under sevoflurane maintained anaesthesia in dogs, but Lozano *et al.* (2009) observed mean rectal temperature of 35.85°C in dogs maintained under sevoflurane anaesthesia. The lowest rectal temperature recorded in the present study was 38.12 ± 0.13°C in Group I animals and 37.4 ± 0.37°C in Group II animals during the period of maintenance. The decrease in rectal temperature during sevoflurane maintenance was significant in both the groups ($p<0.05$ in Group I, $p<0.01$ in Group II animals), which could be due to decreased metabolic rate, muscle relaxation, decreased shivering threshold, central nervous system depression and impaired thermoregulatory control as reported by Muir III and Gadawski, (1998) Kandapal *et al.* (2005) and Singh *et al.* (2010). The decrease in rectal temperature following propofol-isoflurane administration (Ramankutty, 2008) and with sevoflurane anaesthesia (Bennet *et al.,* 2008; Jadon *et al.,* 2008 and Lozano *et al.,* 2009) had also been reported in dogs.

**5.3.2 Pulse Rate** (Fig. 5)

There was significant increase in pulse rate after premedication ($p<0.01$), during anaesthesia ($p<0.01$) and recovery in both groups ($p<0.05$ in Group I and $p<0.01$ in Group II animals). It was decreased to preanaesthetic values at 24 hours in Group I animals and near to preanaesthetic value in Group II animals. Propofol had been reported to increase pulse rate (Redondo *et al.*, 1999; Venugopal *et al.*, 2002 and Mohan, 2006) following induction in dogs. Isoflurane had been reported to increase pulse rate in dogs by Narayanan (2007) and Ramankutty (2008).

**5.3.3 Respiratory Rate** (Fig. 6)

There was significant decrease ($p<0.01$) in respiratory rate after premedication and during anaesthesia in both the groups. There was significant decrease ($p<0.01$) in respiratory rate during recovery in Group I animals, and non significant decrease in Group II animals. The respiratory rate was increased at 24 hours in both groups with a significant increase ($p<0.01$) observed in Group II animals.

The significant reduction in respiratory rate as an effect of xylazine in dogs had been reported by Jacobson *et al.* (1994), Redondo *et al.* (1999), Sooryadas (2001) and Kandapal *et al.* (2005). Significant decrease in respiratory rate under propofol anaesthesia with or without premedication in dogs had been reported by Venugopal *et al.* (2002), Kale *et al.* (2006), Mohan (2006), Sharma and Bhargava (2007). A decrease in respiratory rate up on induction with propofol had been reported by Redondo *et al.* (1999) and Sooryadas (2001).

A dose related respiratory depression had been reported by Clarke (1999) with sevoflurane in dogs. Decreased respiratory rate in dogs during sevoflurane maintenance had also been reported by Johnson *et al.* (1998), Branson *et al.* (2001), Polis *et al.* (2001), Gomez-Villamandos *et al.* (2005), Jadon *et al.* (2008), Singh *et al.* (2010) and Topal (2008). The decreased respiratory rate with

temporary apnoea in propofol-isoflurane anaesthesia in dogs was reported by Ramankutty (2008).

Sudden variations in the rate of respiration as its decreased rate observed during deep anaesthesia stage in one animal of Group I ($I_3$) and in two animals of Group II ($II_1$ & $II_3$) were transient and easily reversed by rapid control of depth of anaesthesia (Mutoh *et al.,* 1997 and Laredo *et al.,* 2009).

**5.3.4 Peripheral Haemoglobin Oxygen Saturation Level (Pulse Oximetry)** (Fig. 7)

There was significant decrease ($p<0.01$) in peripheral haemoglobin oxygen saturation level after premedication in both the groups, but increased during anaesthesia in Group I animals and significantly increased ($p<0.05$) in Group II animals; which was decreased towards recovery and observed significant decrease ($p<0.05$) at 24 hours post operatively in both the groups. In all the stages $SpO_2$ was above 95% due to maintenance of anaesthesia with sevoflurane in oxygen and only because of withdrawal of 100% oxygen in the recovery period a decreased value within the physiological limit was observed.

A pulsatile blood flow could be maintained in all the animals (Nicholson and Watson, 2001) which indicate perfect tissue oxygen delivery with the anaesthetic protocol studied. Glycopyrrolate premedication in the present study was beneficial (Jacobson *et al.,* 1994) which could rectify the action of xylazine (Haskins *et al.*, 1986), in reducing oxygen delivery to the tissues. Increased $SpO_2$% had been also reported with sevoflurane anaesthesia by Topal (2008) and propofol-isoflurane anaesthesia by Ramankutty (2008).

The effect of age on $SpO_2$% was apparently not observed in this study as against the findings of Yamishita *et al.* (2009).

### 5.3.5 Electrocardiogram (ECG)

ECG changes noticed following premedication were tachycardia in three dogs ($I_5$, $II_2$ & $II_4$) and first degree heart block in two dogs ($I_3$ & $II_1$).

Administration of glycopyrrolate might have prevented the incidence of higher degree heart blocks (Singh *et al.,* 1997; Lemke, 2001) in premedication period of this study. Glycopyrrolate premedication had been reported to be beneficial in maintaining cardiac index (Jacobson *et al.,* 1994; Singh *et al.,* 1997) and Jacobson *et al.* (1994) also reported significant increase in heart rate. Dyson and Davies (1999), Lemke (2001), Nicholson and Watson (2001) reported its administration in children to prevent bradycardia during sevoflurane-remifentanil based anaesthesia (Reyntijens *et al.,* 2005). In dogs, unlike human, vagal activity predominated in the control of heart rate (Picker *et al.,* 2001).

The changes suggestive of myocardial hypoxia (Bolton, 1975) noticed during anaesthesia in Group I animals were S-T segment depression in two dogs ($I_3$ & $I_4$) and S-T slurring in one dog ($I_4$). The changes suggestive of myocardial hypoxia noticed in Group II animals were changes in polarity of T wave in two dogs ($II_4$ & $II_6$), high P amplitude in one dog ($II_4$), S-T segment depression in one dog ($II_3$) (Jadon *et al.,* 2008), peaked T wave in one dog ($II_3$) and single ventricular premature contraction in one dog ($II_1$). But these changes were not persisted and corrected spontaneously during recovery period in both groups.

Other changes observed during anaesthesia in healthy animals were decrease in R wave amplitude in two dogs ($I_1$ & $I_6$), reduced R and T amplitude in one dog ($I_5$), high T amplitude in two dogs ($I_3$ & $I_5$), sinus bradycardia in one dog ($I_3$), first degree heart block in one dog ($I_3$), and second degree heart block in one dog ($I_3$). The changes observed in compromised animals were reduced P amplitude in one dog ($II_1$), decrease in R wave amplitude in two dogs ($II_1$ & $II_6$), reduced T amplitude in two dogs ($II_2$ & $II_5$), tachycardia in two dogs ($II_2$ & $II_4$) and first degree heart block in one dog ($II_1$).

The decrease in R wave amplitude in two healthy and two compromised animals might be indicative of decreased cardiac output (Nystrom *et al.,* 1999) in the increased anaesthetic depth. Administration of sevoflurane at higher doses had been reported to be depressing myocardium and cause fall in cardiac output (Clarke, 1999) but rapid lowering of anaesthetic depth effected then and there in the present study might have avoided untoward effects.

Propofol had also been reported to increase heart rate (Muir III and Gadawski 1998; Kale *et al.,* 2006 and Sharma and Bhargava, 2007) following induction of anaesthesia in dogs.

Singh *et al.* (2010) observed a significant increase in heart rate with sevoflurane anaesthesia in dogs. Episodes of tachycardia also observed in two animals during the anaesthesia maintenance period. Such episodes had also been reported by Johnson *et al.* (1998), Polis *et al.* (2001), Bennet *et al.* (2008) and Jadon *et al.* (2008) in dogs and Topal (2008) in rabbits during sevoflurane anaesthesia. According to Picker *et al.* (2001) it may be due to decreased vagal tone as an effect of increased sevoflurane concentration.

In spite of premedication with glycopyrrolate, sinus bradycardia and second degree heart block were observed in one healthy dog ($I_3$); first degree heart block in a compromised dog ($II_1$) during anaesthesia maintenance, which might be due to increased anaesthetic depth. The spontaneous ventilation adopted in the present study might have often accompanied by some degree of hypoventilation (Mutoh *et al.,* 1997 and Polis *et al.,* 2001) and the possible moderate increase in $PaCO_2$ which could stimulate sympathetic nervous system and there by a beneficial effect on cardiovascular depression happened in those animals.

### 5.3.6 Colour of Visible Mucous Membrane

The colour of visible mucous membrane was pale roseate in all healthy dogs throughout the period of study indicating the stability of peripheral

circulation. On account of compromised nature, two animals ($II_1$ & $II_2$) had pale and four ($II_3$ to $II_6$) had congested mucous membranes in the Group II indicating toxaemia.

**5.3.7 Capillary Refill Time** (Fig. 8)

There was significant increase ($p<0.01$) in capillary refill time after premedication, during anaesthesia, recovery ($p<0.05$ in Group I animals) and then it was decreased to the preanaesthetic value at 24 hours post operatively in both the groups. Similar observations were reported by Narayanan (2007) and Ramankutty (2008) in dogs maintained on isoflurane anaesthesia.

**5.3.8 Clotting Time** (Fig. 9)

There was non significant increase in clotting time after premedication in Group I animals, unchanged in Group II animals; increased during anaesthesia in Group I animals and significantly increased ($p<0.05$) in Group II animals; there was significant increase ($p<0.01$) in clotting time during recovery in Group I animals and it was decreased in Group II animals, and then it was decreased to the preanaesthetic value at 24 hours post operatively in both groups. Clotting time reported to be not affected with propofol by Short and Bufalari (1999). Narayanan (2007) reported marginal variations in blood coagulation time in healthy dogs maintained on isoflurane anaesthesia.

5.4 HAEMATOLOGICAL PARAMETERS

In the present study, other than an increase in erythrocyte sedimentation rate (ESR) all parameters viz. VPRC and total leukocyte concentration (TLC) showed a decrease during sevoflurane anaesthesia in healthy animals. Dextrose normal saline administered after induction of anaesthesia intravenously to all animals of both groups might have resulted in haemodilution, but the influence of this factor has affected uniformily and hence not contributed to the variation in interpreting the haematological values.

The decrease in haemoglobin concentration, VPRC and TLC was observed after premedication, during anaesthesia and during recovery in the compromised dogs which might be due to spleenic pooling of blood constituents as reported by Singh *et al.* (1997) and Chandrashekarappa (2009).

**5.4.1 Haemoglobin Concentration** (Fig. 10)

There was decrease in haemoglobin concentration after premedication (significant in Group II, $p<0.05$) during anaesthesia (significant in both groups, Group I-$p<0.05$; Group II-$p<0.01$) and recovery (significant in Group II, $p<0.01$) in both groups. The decreased haemoglobin level reached near to the base line value at 24 hours post operatively in Group II animals, but in Group I animals the level remained decreased. The decreased haemoglobin concentration after premedication is in agreement with the observation made by Mohan (2006) in dogs with glycopyrrolate-xylazine premedication and Sooryadas (2001) in dogs with atropine-xylazine premedication.

Non significant alteration in haemoglobin concentration was reported in puppies under sevoflurane maintenance by Jadon *et al.* (2008) and with propofol anaesthesia by Chandrashekarappa *et al.* (2009). Transient nonsignificant decrease in haemoglobin concentration was reported by Singh *et al.* (2010) with sevoflurane anaesthesia in dogs.

**5.4.2 Volume of Packed Red Cells (VPRC)** (Fig. 11)

There was significant decrease in volume of packed red cells after premedication ($p<0.05$), during anaesthesia ($p<0.05$) and recovery ($p<0.01$), in both the groups except a non significant difference after premedication in Group I animals. The VPRC returned near to normal range by 24 hours in Group I animals but was in a decreased state in Group II animals. Significant decrease in VPRC had been reported in dogs after propofol-ketamine anaesthesia with or without premedication by Venugopal *et al.* (2002); reported decrease by Mohan (2006) and Chandrashekarappa *et al.* (2009) in propofol anaesthesia and in

propofol-isoflurane anaesthesia by Ramankutty (2008). Non significant alteration in VPRC was reported in dogs under sevoflurane maintenance by Jadon *et al.* (2008) and Singh *et al.* (2010).

**5.4.3 Erythrocyte Sedimentation Rate (ESR)** (Fig. 12)

There was significant increase in erythrocyte sedimentation rate in both the groups after premedication ($p<0.05$), during anaesthesia ($p<0.01$) and recovery ($p<0.01$) except in Group I animals after premedication, where the increase was not significant. Similar observations were reported by Ramankutty (2008) in healthy dogs with propofol-isoflurane anaesthesia, but reported a decrease in compromised dogs. The ESR reached to near baseline value at 24 hours in Group I animals, but remained significantly increased ($p<0.05$) in Group II animals.

**5.4.4 Total Leukocyte Count (TLC)** (Fig. 13)

The total leukocyte count showed decrease throughout the period of study in both the groups except at 24 hours where there was an increase. Similar changes were reported in propofol-ketamine anaesthesia with or without premedication by Venugopal *et al.* (2002); Mohan (2006) and Chandrashekarappa *et al.* (2009) in propofol anaesthesia and Ramankutty (2008) in propofol-isoflurane anaesthesia in dogs. The decrease in TLC observed after premedication is in agreement with Sooryadas (2001) in dogs with atropine-xylazine premedication.

**5.4.5 Differential Leukocyte Count (DLC)**

There was decrease in lymphocyte count and increase in neutrophil count after premedication in both the groups, which was significant in Group II animals; same trend observed during anaesthesia, recovery and at 24 hours in compromised dogs but the changes were not significant. The decrease in lymphocyte and increase in neutrophil count after premedication in compromised

dogs is in accordance with Mohan (2006). It agrees with Ramankutty (2008) under propofol-isoflurane anaesthesia in healthy and compromised dogs. The increase in lymphocyte count and decrease in neutrophil count was observed during anaesthesia in group I animals and similar observations in healthy dogs under isoflurane maintenance were made by Narayanan (2007). The lymphocyte count reached to base-line values during recovery and at 24 hours in Group I animals. The non significant alteration in differential leukocyte count observed in healthy dogs in the present study and was in accordance with the observations made by Jadon *et al.* (2008) in puppies maintained on 2.5% sevoflurane.

***5.4.5.1 Lymphocyte Count*** (Fig. 14)

There was decrease in lymphocyte count after premedication in Group I and II animals which was significant ($p<0.05$) in Group II, it increased during anaesthesia in Group I animals but in Group II animals noticed a decrease. The lymphocyte count reached to the preanaesthetic value during recovery and at 24 hours post operatively in Group I animals but decreased in Group II animals. All these fluctuations were non significant except after premedication in Group II animals.

***5.4.5.2 Neutrophil Count*** (Fig. 15)

There was increase in neutrophil count after premedication in Group I and II animals which was significant ($p<0.01$) in Group II; it decreased during anaesthesia in Group I animals but in Group II animals it increased. A mild increase during recovery was noticed in both the groups. It decreased at 24 hours post operatively in Group I animals and increased in Group II animals. All these fluctuations were non significant except after premedication in Group II animals. Non significant changes were observed in neutrophil count in dogs maintained with sevoflurane by Singh *et al.* (2010).

*5.4.5.3 Eosinophil Count* (Fig. 16)

There was decrease in eosinophil count after premedication and during anaesthesia in both the groups. The decrease in eosinophil count during recovery was observed in Group I animals but marginal increase in Group II animals. It increased at 24 hours post operatively in Group I animals but a marginal decrease in Group II animals. All these fluctuations were non significant in both groups. Marginal variations in eosinophils were reported by Ramankutty (2008) under propofol- isoflurane anaesthesia in dogs. Insignificant changes in eosinophil count were also reported by Singh *et al.* (2010) with sevoflurane anaesthesia in dogs.

*5.4.5.4 Monocyte Count* (Fig. 17)

There was marginal increase in monocyte count after premedication and then it was in decreasing trend during anaesthesia, recovery and at 24 hours postoperatively in Group I animals. There was marginal decrease in monocyte count after premedication and subsequently marginal increase was observed during anaesthesia, recovery and at 24 hours post operatively in Group II animals. The increase in monocyte count during anaesthesia was also reported by Ramankutty (2008) under propofol- isoflurane anaesthesia in dogs. Insignificant changes in monocyte count were also reported by Singh *et al.* (2010) with sevoflurane anaesthesia in dogs.

*5.4.5.5 Basophil Count*

Basophil counts observed in both the group were not significant.

5.5 SERUM BIOCHEMICAL PARAMETERS

There were only non significant fluctuations from the preanaesthetic values in the blood urea nitrogen (BUN) and creatinine levels in both the groups. Thus the anaesthetic protocol studied was safe for the renal function and is in agreement with Singh *et al.* (2010) with sevoflurane anaesthesia in dogs. Short

and Bufalari (1999) reported safe use of propofol in human patients with renal impairments. The non significant increases in BUN and creatinine levels observed in compromised animals indicate that there might have been alterations in glomerular filtration rate on account of toxaemia.

There was increase in aspartate aminotransferase (AST) and alanine amino transferase (ALT) at 24 hours post operatively in both the groups. The total protein was also decreased after premedication, during anaesthesia and during recovery in both the groups. Those effects might be due to higher concentration of sevoflurane (>2 MAC) causing reduction in hepatic blood flow as reported by Patel and Goa (1996) in dogs. In contrast, Clarke (1999) reported that sevoflurane could preserve hepatic arterial blood flow during deep anaesthesia. Topal *et al.* (2003) studied the hepatic effects of halothane, isoflurane and sevoflurane anaesthesia in dogs and also reported that the sevoflurane resulted in least elevation of serum activities of liver enzymes.

**5.5.1 Blood Urea Nitrogen (BUN)** (Fig. 18)

There was non significant increase in BUN level during anaesthesia and at 24 hours post operatively in both groups. The Group II being compromised animals, the BUN levels recorded as preanaesthetic value, during anaesthesia and at 24 hours were above the normal range and the same trend maintained during the period of anaesthesia. The fluctuations in BUN value were non significant in both groups. Non significant alterations in BUN level had been reported in dogs maintained anaesthesia with 2.5% sevoflurane by Jadon *et al.* (2008), 3-3.5% sevoflurane by Singh *et al.* (2010) and with propofol-isoflurane anaesthesia by Ramankutty (2008).

**5.5.2 Creatinine** (Fig. 19)

There were non significant fluctuations in creatinine value in Group I animals and the same in already existing higher serum creatinine level in Group II animals. It was increased at 24 hours post operatively in Group II animals.

Nonsignificant alterations in creatinine value had been reported in puppies maintained anaesthesia with 2.5% sevoflurane (Jadon *et al.,* 2008). Chandrashekarappa *et al.* (2009) reported no significant difference in the creatinine levels under general anaesthesia induced with propofol in dogs. Ramankutty (2008) evaluated the propofol-isoflurane anaesthesia in healthy and compromised dogs and reported non significant decrease in creatinine level during anaesthesia. Non significant alterations observed in creatinine level in healthy and compromised dogs in the present study excluded the possibility of deleterious effect on kidney on account of the anaesthetic regimen.

**5.5.3 Aspartate Aminotransferase (AST)** (Fig. 20)

There were non significant fluctuations in aspartate aminotransferase after premedication, during anaesthesia and recovery in both the groups. There was increase in AST at 24 hours post operatively in both the groups and it was significant ($p<0.01$) in Group I animals. In the Group I animals, there was mild increase (1.5-2 times from the preanaesthetic values) in AST at 24 hours. Out of the four pyometra cases in the Group II, three animals had mild to moderate increase (2 to 7 times from the preanaesthetic values) in AST at 24 hours. Higher endotoxins in pyometra might have resulted in hepatic degenerative changes, inhibition of gluconeogenesis and break down of skeletal muscles as reported by Nath *et al.* (2009). Chandrashekarappa *et al.* (2009) reported no significant difference in the AST level under general anaesthesia induced with propofol in dogs. Ramankutty (2008) studied the propofol-isoflurane anaesthesia in dogs and reported increased AST during anaesthesia but within the normal physiological limits.

**5.5.4 Alanine Aminotransferase (ALT)** (Fig. 21)

There was non significant increase in alanine aminotransferase after premedication and at 24 hours post operatively in both the groups. The result in the present study agrees with that of Jain *et al.* (2007) with propofol anaesthesia

in dogs. Non significant alterations in ALT had been reported in dogs maintained on anaesthesia with 2.5% sevoflurane by Jadon *et al.* (2008) and 3-3.5% sevoflurane by Singh *et al.* (2010). Chandrashekarappa *et al.* (2009) reported no significant difference in the ALT under general anaesthesia induced with propofol in dogs.

**5.5.5 Total Protein (TP)** (Fig. 22)

There was decrease in total protein after premedication in both the groups, and it was significant ($p<0.05$) in Group II animals. There was significant ($p<0.05$) decrease in the total protein during anaesthesia and recovery in both the groups which is in agreement with Singh *et al.* (2010). It was increased near to the preanaesthetic value in Group I animals at 24 hours postoperatively, where as it was below the preanaesthetic value in Group II animals.

The decreased total protein might be an indication of reduced liver function and the effect was more prominent in compromised animals.

Nonsignificant alterations in total protein level had been reported in puppies maintained on anaesthesia with 2.5% sevoflurane by Jadon *et al.* (2008). Jain *et al.* (2007) reported non significant changes in values of total protein under propofol-ether anaesthesia in dogs. Chandrashekarappa *et al.* (2009) reported no significant difference in the TP under general anaesthesia induced with propofol in dogs. Singh *et al.* (2006) observed a decline in total protein following xylazine administration in horses. The decreased TP observed after glycopyrrolate-xylazine premedication is in agreement with Narayanan (2008) in healthy dogs and Mohan (2006) in compromised dogs. Ramankutty (2008) evaluated the propofol-isoflurane anaesthesia with glycopyrrolate-xylazine premedication in healthy and compromised dogs and reported decreased TP during anaesthesia but within the physiological limits.

## 5.6 POSTANAESTHETIC AND POSTOPERATIVE COMPLICATIONS

All the animals of Group I and II had rapid, smooth and uneventful recovery from anaesthesia. All the animals of Group I and II had uneventful recovery from surgery.

# *Summary*

# 6. SUMMARY

The study was conducted in 12 dogs of either sex, different age groups, breed and body weight brought for various surgical procedures at Veterinary hospitals Mannuthy and Kokkalai, College of Veterinary and Animal Sciences, Mannuthy. Six healthy animals presented for surgical procedures were included in Group I and those six animals presented with compromised condition for surgical procedures were included in Group II.

Among the animals selected for the study, there were three male and three female dogs with mean age of 6.33 ± 1.94 years (3 to 14 years) and mean body weight of 17.47 ± 3.08 kg (7.3 to 27 kg) in the Group I and one male and five female dogs with mean age of 7.83 ± 1.54 years (2.5 to 13.5 years) and mean body weight of 15.43 ± 4.05 kg (6.00 to 29 kg) in Group II.. There was no significant difference between groups in the age and body weight. None of the dogs in the group I had the history of medical or surgical treatment during the past one month. Majority of the dogs in the group II had the history of medical treatment with parenteral antibiotics and fluids from onset of illness. Inspite of medical treatment none of the dogs in Group II had shown promising recovery owing to multi organ involvement and hence decided surgical treatment as next option after stabilisation of the patient's condition.

The animals were examined for physical condition and their physical status for anaesthesia rated as American Society of Anaesthesiologists (ASA)status level I for three dogs and ASA II for three dogs in the group I; ASA status level III for four dogs and ASA status level IV for two dogs in the group II. All dogs in Group I were subjected to withholding of water for 12 hours and food for 24 hours prior to anaesthesia and in Group II being in compromised condition, avoided such routine preanaesthetic preparations.

Same anaesthetic protocols were studied in animals of both the groups. Glycopyrrolate at the rate of 0.011 mg/kg body weight, followed by xylazine at the rate of 1 mg/kg body weight at 15 minutes interval were administered intramuscularly as premedication to animals of both the groups. Fifteen minutes later, propofol 1% emulsion at the rate of 4 mg per kg body weight was administered by intravenous bolus injection for induction of anaesthesia. Anaesthesia was maintained with sevoflurane (2.5 to 6.0%) in pure oxygen by Bain's circuit system utilising sevoflurane vaporiser.

The salient clinical signs of sedation observed in both groups were winking of eyes, lowering of head, scanty salivation, incoordination of gait, ataxia and sitting on haunches followed by sternal recumbency with head down posture. The degree of sedation was more pronounced in compromised dogs than in healthy dogs.

The time for induction of anaesthesia was 1.17 ± 0.17 min in Group I animals and in Group II it was 0.92 ± 0.05 min. Time for induction of anaesthesia was comparatively shorter in Group II animals. Deeply sedated animals exhibited excellent induction quality compared to animals with less degree of sedation. Endotracheal intubation was possible in all the dogs.

At the onset of maintenance, a higher vaporiser concentration of 5 to 6% sevoflurane was used which was significantly higher in Group I animals. The vaporiser concentration of sevoflurane required for maintenance was comparatively lower in compromised dogs. The sevoflurane vapour concentration used to maintain the depth of anaesthesia was 3.83 ± 0.11% (1.62 MAC) in Group I and 3.67 ± 0.11% (1.56 MAC) in Group II animals. All the dogs achieved a steady – state of anaesthesia. There was no significant difference in vaporiser concentration of sevoflurane to achieve deep and light planes of anaesthesia between the two groups. The animals achieved a light plane of anaesthesia with the vaporiser concentration of 2.6 – 2.7% sevoflurane.

The duration of anaesthesia was 56.67 ± 4.09 and 72.50 ± 4.47 min in Group I and II respectively. The surgical procedures conducted on compromised dogs required longer duration of anaesthesia for maintenance than that of animals in healthy condition.

The depth of anaesthesia as assessed from the surgical procedures performed was satisfactory in all the dogs. Sevoflurane provided a good controllable depth of anaesthesia for the maintenance.

Out of the twelve animals studied, the degree of muscle relaxation was good in three animals and excellent in nine animals for performing the surgical procedures.

Recovery quality was excellent in all the dogs of both the groups. The recovery time was significantly prolonged in compromised dogs compared to healthy dogs. It was 18.50 ± 3.19 min in Group I and 29.00 ± 1.75 min in Group II animals. The different time components in the stages of recovery recorded from switching off the sevoflurane vaporiser in the present study were: Extubation time – 3.17 ± 0.48 min in Group I, 5.00 ± 0.26 min in Group II; Head lift time - 8.50 ± 1.41 min in Group I, 13.17 ± 0.79 min in Group II; Sternal recumbency time - 12.50 ± 2.06 min in Group I, 18.50 ± 1.31 min in Group II; Standing time- 18.50 ± 3.19 min in Group I; 29.00 ± 1.75 min in Group II.

Vocalisation and regurgitation were the undesirable side effects observed during post anaesthetic period in two animals. The post operative pain by those two animals was managed with intramuscular administration of meloxicam at the rate of 0.2 mg/kg body weight. One animal had sneezing during the anaesthesia recovery period as an undesirable side effect.

There was decrease in rectal temperature after premedication, during anaesthesia and recovery in both groups. There was significant increase in pulse rate after premedication, during anaesthesia and recovery in both groups. There was decrease in respiratory rate after premedication and during anaesthesia in

both the groups. Peripheral haemoglobin oxygen saturation level increased during anaesthesia in both the groups.

Electrocardiographic changes noticed following premedication in Group I and II animals were tachycardia and first degree heart block. During anaesthesia the changes noticed in Group I and II animals in common were S-T segment depression, peaked T wave, reduction in R wave amplitude and first degree heart block. The changes observed particular to Group I animals were S-T slurring, reduced R and T amplitude, second degree heart block, and Sinus bradycardia; those particular to Group II were change in polarity of T wave, high P amplitude, peaked T wave, single ventricular premature contraction, reduced P amplitude, reduced T amplitude and episodes of tachycardia. All these changes were spontaneously corrected during recovery period.

The colour of mucous membrane was pale roseate in all animals of Group I during the periods of observation, while two animals of Group II had pale and four had congested mucus membranes.

In the present study, other than an increase in ESR, capillary refill time and clotting time, all haematological parameters like VPRC and TLC observed a decrease during sevoflurane anaesthesia in Group I and Group II animals. There had non significant fluctuations in the lymphocyte, neutrophil, eosinophil, monocyte counts on account of anaesthesia, except in Group II animals where there was significant decrease in lymphocyte and significant increase in neutrophil counts after premedication. The total protein significantly decreased during anaesthesia and recovery in Group I animals. There were only non significant fluctuations from the preanaesthetic values in the blood urea nitrogen, creatinine, aspartate aminotransferase and alanine aminotransferase levels in both the groups on account of anaesthesia, but the AST had significant increase recorded at 24 h in Group I animals.

All the animals of Group I and II had smooth and uneventful recovery from anaesthesia. All the animals of Group I and II had uneventful recovery from surgery.

The following conclusions could be drawn from the study:

(i) Xylazine (1 mg/kg body weight, i/m) with prior administration of glycopyrrolate (0.011 mg/kg body weight, i/m) resulted in satisfactory sedation in all the dogs.

(ii) Glycopyrrolate-Xylazine premedication with propofol induction (4 mg/kg, slow i/v bolus) permitted easy endotracheal intubation.

(iii) Propofol-sevoflurane anaesthesia with glycopyrrolate-xylazine premedication resulted in rapid smooth induction, excellent muscle relaxation and satisfactory depth of anaesthesia with good control during maintenance, smooth, uneventful and rapid recovery in both healthy and compromised animals.

(iv) Sevoflurane with a vaporiser setting of 2.5 to 6% in pure oxygen efficiently maintains propofol anaesthesia.

(v) The anaesthetic regimen studied is recommended for performing clinical surgical procedures in dogs.

# *References*

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# EFFICACY OF SEVOFLURANE FOR MAINTENANCE OF PROPOFOL ANAESTHESIA IN DOGS

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Abstract of the thesis submitted in partial fulfilment of the requirement for the degree of

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# ABSTRACT

The present study was aimed to determine the efficacy of sevoflurane for maintaining propofol anaesthesia in dogs. The study was conducted in 12 dogs of either sex, different age groups, breed and body weight brought for various surgical procedures at Veterinary hospitals Mannuthy and Kokkalai, College of Veterinary and Animal Sciences, Mannuthy. Six healthy animals for surgical procedures with American Society of Anaesthesiologists (ASA) status level I and II for anaesthesia were included in Group I and those six animals for surgical procedures in compromised condition with ASA status level III and IV were included in Group II.

Glycopyrrolate at the rate of 0.011 mg/kg body weight, followed by xylazine at the rate of 1 mg/kg body weight at 15 minutes interval were administered intramuscularly for premedication in both the groups. Fifteen minutes later, propofol at the rate of 4 mg per kg body weight was administered by intravenous bolus injection for induction of anaesthesia. Anaesthesia was maintained with sevoflurane (2.5 to 6.0 %) in pure oxygen by Bain's circuit system utilising sevoflurane vaporiser.

The salient clinical signs observed following premedication in both the groups were winking of eyes, lowering of head, scanty salivation, incoordination of gait, ataxia and sitting on haunches followed by sternal recumbency with head down posture. The time for induction of anaesthesia was 1.17 ± 0.17 and 0.92 ± 0.05 minutes in Group I and in Group II animals respectively. Endotracheal intubation was possible in all the dogs.

The maintenance quality was good to excellent in animals of both the groups. All surgical procedures were performed in medium plane of surgical anaesthesia. Sevoflurane maintenance provided a good controllable depth of anaesthesia with good to excellent degree of muscle relaxation.

The duration of anaesthesia was 56.67 ± 4.09 and 72.50 ± 4.47 minutes in Group I and II animals respectively. The depth of anaesthesia was satisfactory in all the dogs.

The recovery time was 18.50 ± 3.19 minutes in Group I and 29.00 ± 1.75 minutes in Group II animals Recovery quality was excellent in all the dogs of both the groups. Vocalisation, regurgitation and sneezing were the undesirable side effects observed during post anaesthetic period.

The rectal temperature and respiratory rate recorded a decrease while pulse rate and peripheral haemoglobin oxygen saturation level recorded an increase with sevoflurane maintained anaesthesia in both the groups.The electrocardiographic changes were only transient and got corrected spontaneously during recovery period in both the groups.The colour of mucous membrane was pale roseate in all animals of Group I. The two animals of Group II had pale and four had congested mucus membranes.

In general other than an increase in erythrocyte sedimentation rate, capillary refill time and clotting time all haematological parameters like volume of packed red cells and total leukocyte count observed a decrease during sevoflurane maintained anaesthesia. There had non significant fluctuations with in the physiological limits in the lymphocyte, neutrophil, eosinophil, monocyte counts and serum total protein levels on account of anaesthesia, except in Group II animals. There were only non significant fluctuations from the preanaesthetic values in the blood urea nitrogen, creatinine, aspartate aminotransferase and alanine aminotransferase levels in both the groups.

All the animals had smooth and uneventful recovery from anaesthesia and surgery.