

# CLINICAL EVALUATION OF DIAZEPAM- KETAMINE ANAESTHETIC REGIMEN IN MACACA RADIATA (BONNET MACAQUE)

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## ABSTRACT

*The study was conducted on 6 clinical cases of either sex of Macaca radiata referred for procedures such as vasectomy and translocation. The monkeys were administered with an anesthetic combination of diazepam 1 mg/kg bodyweight and ketamine 15mg/kg bodyweight. The findings revealed minimal changes in cardiopulmonary, haematobiochemical parameters, intra and post anesthetic complications and stress response following induction. The duration of anesthesia was  $27.50 \pm 0.58$  minutes. A significant decrease in mean intra ocular pressure during peak anesthesia and before recovery was observed.*

**Key Words:** Macaca radiata-Bonnet Macaque-Diazepam-Ketamine-anesthesia.

## INTRODUCTION

Macaca radiata (Bonnet Macaque) is the most common commensal monkey of South India. Vasectomy is indicated for control of their population which warrants chemical restraining. The increased population also is one factor that these monkeys invariably injured necessitating chemical immobilization to perform or carry out various surgical procedures

## MATERIALS AND METHODS

The study was conducted on six clinical cases of Macaca radiata of either sex referred for procedures such as vasectomy, translocation, radio- diagnosis and for surgical procedures. In all the cases feed and water were withheld for about 12 hours and 3 hours respectively to reduce the risk of aspiration. The monkeys were administered diazepam (1 mg/kg) and ketamine (15mg/kg) (Popilskis et al., 1992) either with hand held syringe or darted with blowgun. Teleinject dart syringes were used for darting with a blowgun.

Atropine sulphate was administered (0.05mg/kg) immediately after ataxia. The following parameters were studied: time for ataxia, time for induction, duration of anesthesia, time for recovery, quality of anesthesia, total dose, intra and post anesthetic complications, buccal temperature, intra ocular pressure. Cardio pulmonary and haematobiochemical parameters including cortisol were studied

## RESULTS AND DISCUSSION

Time taken for ataxia was longer when diazepam was combined with ketamine which could be attributed to the slow absorption rate of diazepam following intramuscular administration and high lipid soluble nature in the adipose tissue would delay the release (Woolfson, et al., 1980). Ataxia could be attributed to the central muscle relaxing effect of benzodiazepine derivative. Time for induction was  $4.68 \pm 0.10$  minutes. Diazepam in combination with ketamine induced anesthesia by its action on the part of the limbic system, thalamus and hypothalamus resulting in calming effect (Swindle et. al., 2002).

The duration of anesthesia was  $27.50 \pm 0.58$  minutes. The duration of anesthesia was controlled by plasma half life of ketamine and the quick recovery from ketamine anesthesia was due to the redistribution from the blood and the central nervous system to other tissues (Wright 1982).

The mean recovery time was  $36.17 \pm 0.61$  minutes. The early recovery from ketamine anesthesia was attributed to ketamine induced catecholamine, which enhanced the basal metabolism leading to faster elimination of ketamine (Luna et. al., 1997).

The quality of anesthesia was smooth and uneventful. Smooth induction could be attributed to the effect of ketamine that altered the reactivity of the central nervous system to various sensory impulses with out blocking sensory input at spinal or brain stem levels. The muscle relaxation could be attributed to the effect of diazepam mediated through central nervous system (Wright 1982).

Diazepam and ketamine at the dose rate of 1 mg/kg body weight and 15mg/kg body weight respectively induced and maintained anesthesia for a period of  $27.50 \pm 0.58$  minutes.

The buccal temperature reduced following induction with diazepam - ketamine which could be attributed to the anxiolytic action and reduction in skeletal muscle metabolism.

The dynamic movement of aqueous and vitreous humor and choroidal vascular volume maintained the intra ocular pressure. Decrease in arterial blood pressure could also decrease intra ocular pressure drastically. In the present study the intra ocular pressure decreased when diazepam was combined with ketamine which could be attributed to the decrease in extra ocular muscle tone caused by diazepam.

The mean heart rate and pulse rate elevated when ketamine alone was administered in animals due to the stimulation of sympathetic nerve trunks and diazepam, a benzodiazepine derivative

moderated the cardio stimulatory property of ketamine and maintained the heart rate. (Jackson et. al, 1978). The respiratory rate decreased from  $33.16 \pm 0.33$  (immediately after induction) to  $21.00 \pm 0.63$  (before recovery) which could be due to the effect of diazepam to potentiate the respiratory depressant effect of ketamine (Haskins et. al, 1986). The study reveled that diazepam was a useful adjunct to ketamine anesthesia as it maintained cardiopulmonary stability in *Macaca radiata*.

The blood urea nitrogen and serum creatinine values at different stages of anesthesia were within normal clinical limits revealing normal renal blood flow, tubular flow rate and urinary excretion (Malaga et. al., 1991). The mean serum protein decreased during peak anesthesia and before recovery which could be attributed to temporary haemodilution due to migration of interstitial fluid into the vascular system (Reddy et. al, 1991). The blood glucose levels increased up to  $143 \pm 0.36$  mg/dl, which could be attributed to the increased gluconeogenesis, inhibition of insulin secretion, stimulation of glucagons secretion and the exiting post receptor within the cell, which inhibited the action of insulin on glucose metabolism (Lassen and Weiser, 2004). The plasma values fluctuated between  $21.83 \pm 0.45$  and  $24.50 \pm 0.32$  ug per 100 ml. The increase in plasma cortisol level is often considered as a marker of physiological or physical stress which has wide ranging effects on many physiological systems. Ketamine did not modify the stress induced increase of cortisol in non-human primates (Pun et. al., 1989).

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