Global Veterinaria 13 (3): 293-296, 2014
ISSN 1992-6197

DOI: 10.5829/idosi.gv.2014.13.03.8562

Evaluation of the Subcutaneous Route for the Induction of Ketamine Anaesthesia in Dogs

Babalola Samuel Adeola

Department of Veterinary Surgery and Theriogenology,
Michael Okpara University of Agriculture Umudike, Abia State, Nigeria

Abstract: This study was undertaken to evaluate the administration of Ketamine hydrochloride anaesthetic subcutaneously (Dorsal neck fold) for the induction of anaesthesia in dogs. Ten (10) Nigerian indigenous dogs were used for the study. Ketamine was administered subcutaneously at a dose rate of 10 mg/kg b.wt. and the dogs evaluated for extent and level of anaesthesia. The basic physiologic parameters and some anaesthetic indices obtained were expressed as mean ± standard deviation, while the anaesthetic indices that cannot be express as their mean were summarised. At the end of the study, 10 mg/kg single dose administration of ketamine hydrochloride subcutaneously produced anaesthesia in the dogs.

Key words: Ketamine · Subcutaneous Route · 10mg/Kg · Dogs · Anaesthesia

INTRODUCTION

Like the phencyclidines, ketamine produces a trance-like state unique to the anaesthesia. This state results from an electrophysiological dissociation between the limbic and higher cortical systems and is termed dissociative anaesthesia [1-3]. Patients anesthetized with ketamine appear to be awake and have little higher cortical depression. At the same time, cortical awareness is blocked from external stimuli, including auditory, visual, or pain-related input. As in a dreamlike state, awareness of time during ketamine anaesthesia is also blunted [1-3]. Brainstem activity remains normal and processes for maintaining essential cardiac and respiratory functions are unaltered. The combinations of these traits in a single drug are remarkable [1-3]. Only under ketamine anaesthesia can a painful procedure be performed without the need for routine intubation, ventilatory control, or sophisticated cardiovascular monitoring. Ketamine has several other beneficial actions; bronchodilation makes ketamine a favoured induction agent for rapid-sequence intubation during status asthmaticus. The effects of ketamine on the central sympathetic system mediate bronchodilation, causing an increase in circulating catecholamine [1-3]. Ketamine has also been demonstrated to have a direct relaxant effect on airway smooth muscle, [1, 2, 4, 5]. Ketamine causes an increase in blood pressure and cardiac output; these two properties make ketamine a favoured anaesthetic agent for patients in shock [6]. Evidence suggests that the adrenergic effects of ketamine are mediated centrally, similar to those of cocaine and that, premedication with central depressants can blunt or abort the myocardial effects of ketamine, such sympatholysis has been demonstrated with droperidol and multiple benzodiazepines, including diazepam, flunitrazepam and midazolam hence their use in cases in which increases in cardiac output or blood pressure are undesirable (e.g., patients with severe coronary artery disease) [6]. The peripheral adrenergic response to ketamine is characterized by catecholamine release and nor-epinephrine re-uptake inhibition. Mean arterial pressure is typically elevated by approximately 25 mm Hg. Pulse rate, stroke volume and cardiac output also increase, although systemic vascular resistance is unaffected [7]. Ketamine has a depressant effect on the myocardium in patients who already have maximal circulating catecholamine levels (severe end-stage shock states) or in cases in which catecholamine activity is blocked [7]. Nonetheless, most patients in shock states

Corresponding Author: Samuel Adeola, Department of Veterinary Surgery and Theriogenology,
Michael Okpara University of Agriculture Umudike, Abia State, Nigeria.

benefit rather than get worse from administration of ketamine [7]. Ketamine increases coronary artery blood flow, although the potential benefit of this effect during ischemic states is offset by the drug's other inotropic actions [7]. In addition, ketamine blunts the myocardial response to catecholamine, making ketamine useful for decreasing epinephrine-induced dysrythmias [8]. The drug has other antidysrhythmic effects as well and was recently demonstrated to reduce reperfusion-induced ventricular fibrillation in animals [9].

MATERIALS AND METHODS

Experimental Animals: Ten (10) apparently healthy Nigerian indigenous dogs of ages 6 months to 1 year (using the dental eruption formulae), of both sex (7 males and 3 females) were purchased from the local market within the study area. The dogs were housed in the Ahmadu Bello University Veterinary Teaching hospital (ABU VTH) Small animal kennels and fed on rice and bean and left over food from a nearby restaurant: Water was supplied Ad libitum for the period of the study.

A detailed preanaesthetic evaluation of all the dogs was carried out and irrespective of the laboratory findings, all the dogs were treated with long acting oxytetracyclin (Avicycline® 20% "L.A". Inj. AVICO Jordan, 20 mg/kg i.m) used for systemic infection, praziquantel (10 mg/kg orally) and ivermectin (Avimec®-Inj. AVICO Jordan) (400ug/kg subcutaneously) for deworming the dogs and 0.06% permethrin powder was used topically to control ectoparasites. The dogs were allowed to acclimatise for two weeks before commencement of experiment. Using the convenience method of sampling, the dogs were numbered serially (numbers 1-10).

The dogs being in stable physiologic conditions and based on their individual body weight already determined using the bathroom scale (Camry® China) method. The dogs were each given Ketamine Hydrochloride 50% (Rotex® Medica, Tritau. Germany) subcutaneously at the dorsal neck fold, at a dose rate of 10 mg/kg. The physiologic parameters and anaesthetic indices were monitored and recorded at 10, 20, 30, 40, 50, 60 and 180 minutes post administration of the anaesthetic. Each dog serves as its own control. The basic physiologic parameters and some anaesthetic indices obtained were expressed as mean ± standard deviation calculated using GraphPad InStat3 statistical package, while the anaesthetic indices that cannot be express as their mean were summarised.

Parameters Assessed: Temperature: This was assessed using digital clinical thermometer (LCD pen style; TDB-3F)® rectaly. With a rotating motion, it was inserted into the rectum gently and held in position until beeped, then withdrawn and the figure on the LCD screen was read and recorded.

Pulse Rate: This was assessed by applying light digital pressure on the femoral artery to feel its pulsation. The numbers of pulse per minute was counted and recorded.

Respiratory Rate: It was assessed visually by observing the inspiratory and expiratory movements of the ribs. The inspiratory and expiratory phases were counted as one respiratory rate.

Anal Sphincter Opening and Pupillary Reflexes *(Response to the ordinary environmental light)*: Were assessed visually by examining the anal opening for constriction or relaxation while the response to ordinary environmental light was used for the pupillary reflex. Pupillary response to light or anal opening is denoted as, normal ++ and absence --).

Assessment of the Swallowing Reflex: Was done manually by applying light friction on the oesophagus to stimulate peristaltic movement of the oesophagus (Minimal reaction is denoted by +, normal by ++ and absence by --).

Muscle Relaxation: Was assessed by noting the limbs at rest as well as the presence or absence of muscular resistance when the limbs were flexed and extended.

Analgesia: was evaluated using rat tooth haemostatic forcep clamp at first ratchet lock at the interdigital space and the dog evaluated for response to pain, this was also use to evaluate the extent and level of anaesthesia.

Induction Time: Time between ketamine administration and when the dog becomes recumbent and disconnected. Duration of anaesthesia: Time between when the dog became recumbent, loss of sensory perception and when the dog becomes environmentally conscious by lifting up its head.

Recovery Time: Time from recumbency to sternal and from sternal to standing unaided.

Table 1: Mean ± standard deviation of the basic physiologic parameters and some anaesthetic indices of the experimental dogs.

Time (minute)	0	10	20	30	40	50	60	180
Temperature(°C)	38.65 ± 0.51	38.36 ± 0.28	38.38 ± 0.31	38.47 ± 0.35	38.33 ± 0.23	38.37 ± 0.41	38.40 ± 0.32	38.39 ±0.33
Pulse rate (b/m)	96.2 ± 9.4	115.3 ± 9.56	117.8 ± 9.56	111.8 ± 14.82	111 ± 13.35	107 ± 10.26	99.38 ± 6.99	98.54 ± 7.32
Respiratory rate(c/m)	35.80 ± 2.27	41.70 ± 2.76	42.1 ± 1.92	40.9 ± 1.71	40.2 ± 1.38	39.45 ± 2.52	39.15 ± 2.02	38.57±2.093
Induction time (minute)	3.5 ± 0.00							
Duration of anaesthesia(minute)	34.5 ± 6.92							
Recovery time(minute)	45 ± 10.94							

Table 2: Summary of some anaesthetic indices of the experimental dogs.

Time (minute)	0	10	20	30	40	50	60	180
Anal sphincter tone	++	--	--	--	+	++	++	++
Pupillary reflex	++	--	--	-	-	+	++	++
Analgesia	++	--	--	-	+	+	++	++
Muscle relaxation	++	--	--	-	+	+	++	++
Swallowing reflex	++	++	++	++	++	++	++	++

RESULTS AND DISCUSSION

The mean induction time of about 3 minutes using the subcutaneous route compared favourably with the 3 to 5 minutes induction time following intramuscular injection of ketamine [10]. This phenomenon cannot be easily explained and further studies may be necessary to fully elucidate this.

During the studies the rectal temperature, respiratory rate were maintained within normal range; swallowing reflex (which protect the animal from aspiration pneumonia and vomiting) was maintained while the pulse rate increased and returned to near normal after a short while. These findings agree with those of i.m and i.v route of administration of ketamine [1, 2, 3].

The mean duration of anaesthesia of about 34.5 minutes is superior to 10-25 minutes reported by Brander [10]. During this period there was muscular rigidity with body spasm which was severe in the hind limbs also analgesia, these continued for about 3 to 5 minutes after the dogs had recovered. These agreed with the findings reported by Hall, *et all.* and Brander [7, 10]. The dogs start to show signs of recovery at the mean time of 45 minutes characterised by mild analgesia with muscle relaxation and tonic-clonic spasms of limb muscles. This study has show that it is possible to induced anaesthesia with ketamine anaesthetic via the subcutaneous route (dorsal neck fold) in dogs.

ACKNOWLEGEMENT

I appreciate all the Technical staff of the small animal unit of the department of Veterinary Surgery and Radiology, Faculty of Veterinary Medicine, Ahmadu Bello University Zaria, Nigeria for their assistance toward the success of this work.

REFERENCES

1. Lau, T.T. and P.J. Zed, 2001. Does ketamine have a role in managing severe exacerbation of asthma in adults? Pharmacotherapy, 21(9): 1100-6
2. Petrillo, T.M., J.D. Fortenberry, J.F. Linzer and H.K. Simon, 2001. Emergency department use of ketamine in paediatric status asthmaticus. Journal of Asthma, 38(8): 657-61
3. Allen, J.Y. and C.G. Macias, 2005. 11w efficacy of ketamine in paediatric emergency Department, patients who present with acute severe asthma, Emergency medicine, 46(1): 43-50.
4. Nehama, J., R. Pass and A. Bechtler-Karsch, 1996. Continuous ketamine infusion for the treatment of refractory asthma in a mechanically ventilated infant: case report and review of the pediatric literature. Pediatric Emergency Care, 12(4): 294-7.
5. Sato, T., A. Matsuki, E.K. Zsigmond and S.F. Rabito, 1997. Ketamine relaxes airway smooth muscle contracted by endothelin. Anaesthesia Analgesia, 84(4): 900-6.
6. Doenicke, A., R. Angster and M. Mayer, 1992. [The action of S-(1)-ketamine on serum catecholamine and cortisol. A comparison with ketamine race mate], Anaesthetist, 41(10): 597-603.
7. Hall, L.W., K.W. Clarke and C.M. Trim, 2001. General Pharmacology of Injectable agents used in Anaesthesia. Veterinary Anaesthesia 10th Edition, W. B. Saunders. An imprint of Harcourt Publishers Limited, pp: 127-129.

8. Niiya, S., 1990. [The effect of ketamine on epinephrine-induced arrhythmias in dogs anesthetized with halothane-nitrous oxide]. *Masui*, 39(12): 1652-9.
9. Baczko, I., I. Lepran and J.G. Papp, 1997. Influence of Anaesthetics on the Incidence of Perfusion-induced Arrhythmias and Sudden Death in Rats. Journal of Cardiovascular Pharmacology, 29(2): 196-201.
10. Brander, G.C., 1991. Dissociative anaesthetics. Veterinary applied pharmacology and therapeutics. 5th edition, ELBS Bailliere Tindal London, pp: 370-372.