Effects of lumbosacral epidural ketamine and lidocaine in xylazine-sedated cats

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ABSTRACT

In order to determine the analgesic and cardiovascular effects of the combination of epidural ketamine and lidocaine, 6 sedated cats were studied. Six healthy, young cats were used in a prospective randomised study. Each cat underwent 3 treatments, at least 1 week apart, *via* epidural injection: (1) ketamine (2.5 mg/kg), (2) lidocaine (4.0 mg/kg), and (3) ketamine (2.5 mg/kg) plus lidocaine (4.0 mg/kg). Epidural injections were administered through the lumbosacral space. Analgesia, motor block, sedation, heart rate, arterial blood pressure, respiratory rate and arterial oxygen saturation were measured. Rectal temperature was compared before and after sedation as well as after epidural administration of the drugs. Epidural administration of the ketamine/lidocaine combination induced prolonged analgesia extending from the coccygeal to the T13-L1 dermatomes, leading to severe ataxia. Cardiovascular effects were significant in all treatments: heart rate decreased, but there was a minimal reduction in arterial pressure. It was concluded that adding a dose of ketamine to epidural lidocaine in cats is feasible and effective.

Keywords: analgesia, cats, epidural, ketamine, lidocaine, xylazine.

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INTRODUCTION

Epidural anaesthetic techniques have been shown to be effective in experimental cats[12]. Most commonly, veterinarians use this technique after induction of anaesthesia and before surgery, because of the difficulty of performing an epidural injection in a conscious cat[31]. Epidural anaesthesia can be used in cats for several procedures, such as orthopaedic surgery on a hind limb, or a caesarian section, in critically ill cats or in cats that are also on a light anaesthetic plane or heavily sedated[20]. Lidocaine is traditionally used for epidural block in dogs and cats[34], but can induce hypotension and neurotoxicity. In view of these alterations during epidural blockade, studies were conducted to determine the advantages of a specific sensory blockade. Researchers have studied several drugs or drug combinations, such as morphine in cats[36], opioids/lidocaine or bupivacaine in mice[2] and ketamine/lidocaine in goats[11]. If the effect of the combination is greater than the sum of the effects of individual agents, the interaction is considered synergistic. Synergistic interactions can occur when drugs affect different critical points along a common pathway[27].

Local anaesthetics have the potential to produce sensory, motor and sympathetic blockade by depressing axonal conduction of nerves. Usually, anaesthetists use 2 % lidocaine with this objective, but higher doses and concentrations induce temporary or permanent lesions of the nervous tissues[19,25]. The vasodilatation due to sympathetic blockade produced by epidurally injected local anaesthetics decreases the duration of anaesthesia[32] and induces hypotension[29]. Local anaesthetics can be used for hind limb and abdominal procedures, but larger volumes that would provide more cranial analgesia cannot be used, owing to the concomitant respiratory and cardiac depression[31].

Epidural administration of the N-methyl-D-aspartate (NMDA) receptor antagonist ketamine produces analgesia in animals[10,11,14]. Ketamine is commercially available as a racemic mixture of the 2 enantiomers, *S*(+)-ketamine and *R*(–)-ketamine. The NMDA receptor plays an important role in the development of neuropathic pain through activation of excitatory amino acids (glutamate) in the dorsal horn[26]. *S*(+)-ketamine, the left-handed optical isomer of racemic ketamine, has a 4-fold-higher affinity for NMDA receptors than right-handed *R*(–)-ketamine[38]. Ketamine is widely used in cats as a dissociative anaesthetic agent. Anaesthetic protocols incorporating ketamine provide better postoperative analgesia than protocols using thiopentone and halothane with or without butorphanol[35].

The objective of the present study was to determine whether the combination of lidocaine and *S*(+)-ketamine administered epidurally in healthy cats produces analgesia of longer duration with fewer side effects than epidural lidocaine or ketamine administered alone, at doses used in clinically veterinary medicine.

MATERIALS AND METHODS

The ethics committee of the Federal University of Mato Grosso do Sul approved the study protocol and experimental design.

Six young, healthy, domestic shorthair cats weighing 2.0–3.7 kg (mean ± standard deviation, 2.7 ± 0.6 kg) were used in this study. All cats were maintained in cages in the small animal anaesthesia room of the Faculty of Veterinary Medicine and Animal Science facilities throughout the experimental period. Each animal received 3 treatments in a randomly selected way, with at least a 1-week interval between each treatment. Observers were blind to the drugs and doses administered in each study.

Fifteen minutes before administration of epidural drugs, all cats were sedated with 1.0 mg/kg of 1 % xylazine intramuscularly. The lumbosacral area was clipped and the skin was prepared for aseptic placement of an epidural needle, after infiltration of the skin and subcutaneous tissues with 0.5 mℓ of the 1 % lidocaine. We maintained all animals in sternal recumbency for localisation of the lumbosacral epidural space. This space was identified by the depression between the last lumbar vertebra and the 1st sacral vertebra. A 22-gauge 2.5 cm epidural needle was placed at the epidural space at a 70–90° angle along the median plane,

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with the bevel facing cranially. Correct positioning of the needle was confirmed by the hanging-drop method and loss of resistance by injected air into the epidural space. Treatments were *S*(+)-ketamine (K; 0.05 mℓ/kg; 2.5 mg/kg; Cristália Chemical and Pharmaceutical Products Ltd, Brazil), 2 % lidocaine without adrenaline (L; 0.20 mℓ/kg; 4.0 mg/kg; Hipolabor Farmacêutica Inc, Sabará, Brazil) and *S*(+)-ketamine plus 2 % lidocaine (KL; *S*(+)-ketamine 2.5 mg/kg plus lidocaine 4.0 mg/kg). The volume of the drugs was always 1 mℓ/3.0 kg body weight, using sterile 0.9 % saline solution when necessary to achieve this volume. All drugs were injected at a rate of 0.5 mℓ/s into the epidural space of each experimental animal.

Heart rate (HR), arterial blood pressure (systolic (SAP), diastolic (DAP) and mean (MAP) arterial pressure), respiratory rate (RR), arterial oxygen saturation measured by pulse oximetry (SpO_2), rectal temperature (RT), analgesia, sedation and motor blocking were measured before drug administration (baseline), 5, 10 and 15 min after sedative drug administration and 15 min after epidural administration until the end of the anaesthetic period. The same investigator for the 3 treatments confirmed analgesia or anaesthesia. Any lack of analgesia (*e.g.* a strong positive response to a noxious stimulus) was determined after sedative drug administration, but before epidural administration of the drugs in any treatment.

After epidural injections standard painful stimuli were measured using a 25-gauge needle to assess superficial pain (needle prick to the skin) and with the response to the pressure of a haemostatic forceps (closed to the 1st ratchet) to assess deep pain. These painful stimuli were applied to the skin of both lateral aspects of the hind limbs, perineum and upper and lower abdominal wall. Each treatment ended when responses to a noxious stimulus were similar to the response observed before drug administration. Analgesia was determined using the following scale: 1 = strong reaction to painful stimulus, 2 = depressed reaction to painful stimulus, 3 = analgesia (no response to superficial needle-prick stimulation of the skin) and 4 = anaesthesia (no response to pressure of haemostatic forceps applied to the skin). Motor-blocking effects were evaluated using the following scale: 1 = normal motor function, 2 = mild motor incoordination (cat has difficulty maintaining a standing position), 3 = ataxic (cat is recumbent, with movement of hind limbs) and 4 = severely ataxic (cat is in sternal recumbency, in frog-like position, without movement of hind limbs; Fig. 1). During sedation, before and after epidural administration of the drugs, sedation was evaluated using the following scale: 1 = no sedative effects, 2 = mild sedation (reduced alertness, some response to acoustic stimuli), 3 = moderate sedation (drowsiness, head down, mydriasis, moderate palpebral reflex) and 4 = marked sedation (lateral or sternal recumbency, no response to acoustic stimuli, mydriasis, head down, weak palpebral reflex).

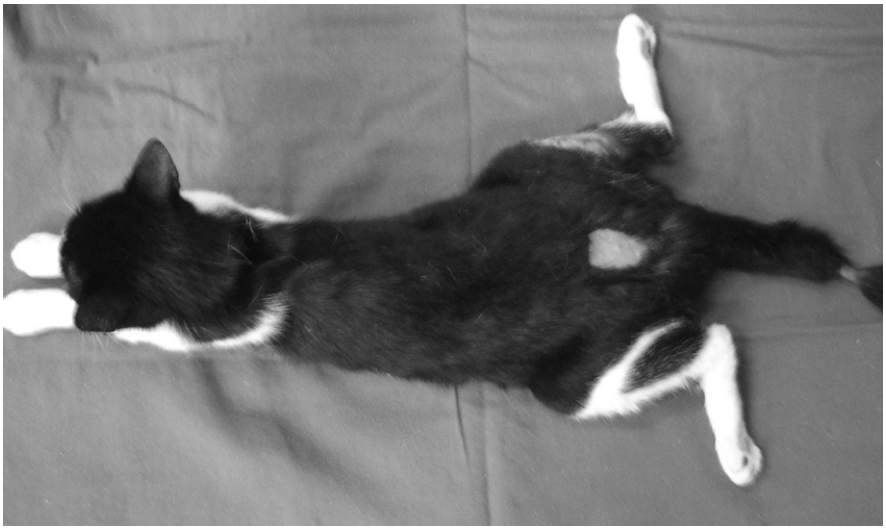

Fig. 1: **Cat in sternal recumbency without movement in the hind limbs and with the hind limbs spread open laterally** (frog-like position).

SAP, DAP and MAP were measured using a non-invasive device (RX-300[A] cardiac monitor; Transmai Equipamentos Médicos Hospitalares); the cuff was placed over the ulnar artery, on the forearm. Arterial blood pressure was measured by the oscillometric method. A sensor affixed to the clipped tail measured SpO_2. HR was measured using electrocardiography, RR was determined as the number of chest movements per minute, and RT was obtained with a digital thermometer. All cardiovascular and respiratory variables were recorded before noxious stimulation to establish baseline values.

To assess the diffusion length of soluble drugs within the epidural space at different intervals, we made superficial skin pricks and applied deep pressure with haemostatic forceps to the skin at adjacent dermatomic regions, beginning at the tail and proceeding cranially up to the caudodorsal rib areas. Response to noise and to sudden movements of personnel was also recorded.

All data were analysed using SAS software (SAS Institute). For each treatment, a randomised complete block design was used in which time was the treatment and each of the 6 animals was a block. For the dependent variables HR, RR, SAP, DAP, MAP and RT, an analysis of variance was performed and a *post hoc* Dunnett's test was applied when the treatment response differed from baseline (time 0). For analgesia, sedation and motor-dependent variables, non-parametric statistics were used (Friedman's test), followed by multiple comparisons for ranked data using Dunnett's test, with time 0 as a baseline. In each analysis, differences were considered significant if $P < 0.05$.

RESULTS

Sedation induced by xylazine was effective in all cats, with duration of 2 or 3 h (grade 3 or 4). Ketamine alone or in combination with lidocaine did not produce any sign of sedation compared with xylazine. However, animals in all treatment groups demonstrated severe ataxia (grade 4). Response to a pinprick or response to the pressure of haemostatic forceps provided a satisfactory indication regarding the efficacy of analgesia in the skin of the hind limbs, perineum and abdominal wall regions during the post-injection period in sedated cats.

Onset of complete analgesia (not significantly different; $P < 0.05$) was earlier in all treatments (2.2 ± 1.0 min) after epidural administration of the drugs. Duration of analgesia was comparable among the different treatments. The ketamine/lidocaine combination produced a longer duration of analgesia (137 ± 14.3 min) than did ketamine (70 ± 6.3 min) or lidocaine (69 ± 13 min) alone (Fig. 2). The cranial spread of analgesia obtained with the KL treatment was similar to K or L alone, extending to dermatomic region T13-L1. The ataxic effect and the analgesic effect of lidocaine alone were similar (66 ± 15 min and 69 ± 13 min, respectively), whereas the ketamine treatment induced a shorter period of ataxia (53 ± 9.6 min; $P < 0.05$). Although the KL treatment led to a period

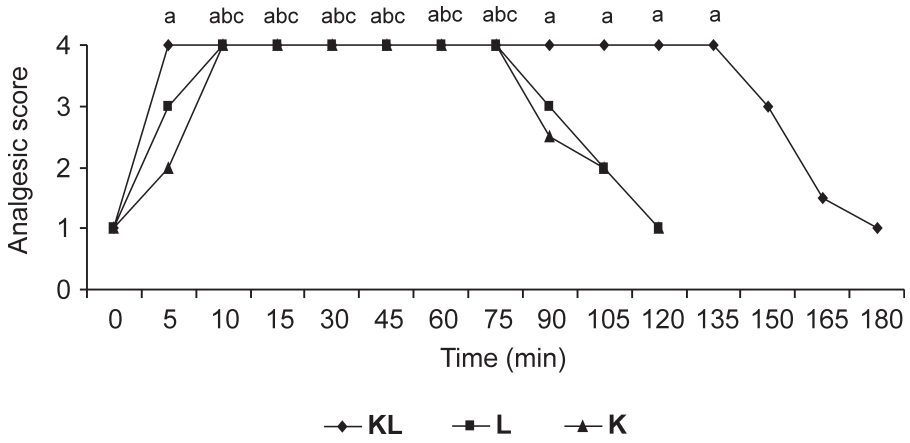

Fig. 2: **Median analgesia score in response a standard noxious stimulus to the hind limbs, perineum and abdominal wall after epidural administration of ketamine** (K 2.5 mg/kg), **lidocaine** (L 4.0 mg/kg) **or ketamine/lidocaine** (KL) **in 6 cats**. **The following scale was used: 1 = strong reaction to painful stimulus, 2 = depressed reaction to painful stimulus, 3 = analgesia** (no response to superficial needle-prick stimulation of the skin) **and 4 = anaesthesia** (no response to pressure of haemostatic forceps applied to the skin)**. Values for [a]KL, [b]L, and [c]K differed significantly** ($P < 0.05$) **from baseline** (time 0) **values.**

of ataxia of 96 ± 17 min, it induced analgesia for approximately140 min.

After sedation and epidural administration, all animals in all treatment groups had a significant decrease ($P < 0.05$) in HR compared with baseline; this decrease was more prolonged in the KL treatment group, starting from 15 min after injection and lasting until the end of the experiment. The K treatment decreased HR significantly ($P < 0.05$) at all time points, and the L treatment decreased HR significantly from 15 to 120 min after injection. Epidural injection of ketamine and of ketamine/lidocaine induced a significant decrease ($P < 0.05$) in SAP in the period from 75 to 105 min and 75 to 90 min, respectively. DAP decreased significantly in the K treatment group from 60 to 105 min after injection, and from 45 to 90 min in the KL treatment group. In addition, we observed an important decrease ($P < 0.05$) in MAP in the K treatment group at 60–105 min and in the KL treatment group at 30–105 min. Arterial pressures (SAP, DAP and MAP) did not show considerable alterations after the L treatment compared with baseline. K, L and KL treatments did not cause statistical differences in RR in any time point. SpO_2 decreased significantly in the K (10–15 min), L (5–60 min) and KL (5–15 min) treatment groups compared with baseline. The K, L and KL treatments induced a significant decrease in RT ($P <$ 0.05) but at different times: K treatment, 90–120 min; L treatment, 105–120 min; and KL treatment, 45–180 min (Tables 1 and 2).

DISCUSSION

In this study involving 6 cats, sedation before epidural administration of the study drugs was effective and permitted epidural punctures. Although alpha 2-adrenoceptor agonists induce moderate to deep sedation and analgesia in dogs and cats, this class of drugs does not produce the anaesthesia required for painful surgical interventions[16]. In some surgical procedures, such as orthopaedic surgery on a hind limb or a caesarean section, it is more humane and convenient to sedate the cat heavily[20]. Several studies have demonstrated that use of general anaesthesia is usually mandatory in the cat[1,20,31].

The results of this study establish that the epidural combination of ketamine and lidocaine produces analgesia in the regions of the skin of the hind limbs, perineum and abdominal wall in cats for a duration double that achieved with the administration of ketamine or lidocaine alone.

The associated hypotension produced by the sympathetic blockade occurs because of the epidural injection of local anaesthetic agents. This effect is particularly harmful in animals undergoing caesarian sections and in vulnerable, unstable patients[29]. The vasodilatation produced by sympathetic blockade induced by epidurally injected local anaesthetics decreases the duration of analgesia[14,32]. Varying sensitivities of nerves to local anaesthetics may result in clinically important differential blockade of somatic sensory fibres and pre-ganglionic sympathetic fibres[8].

Recently, there has been renewed interest in using diluted solutions of the agents in the epidural space to generate a sensory block loss or analgesia without interfering with motor function. However, in animals and humans, there is no evidence of neurological injury after repeated intrathecal injection of preservative-free

Table 1: **Cardiovascular variables in 6 cats given epidural injections of ketamine, lidocaine or ketamine/lidocaine.**

	Treatment	Baseline	Time (min)												
			5	10	15 (EA)	30	45	60	75	90	105	120	135	150	165	180
HR	K	158 ± 19	119 ± 2*	95 ± 11*	91 ± 11*	110 ± 13*	108 ± 18*	105 ± 19*	102 ± 28*	102 ± 27*	104 ± 19*	106 ± 22*	ND	ND	ND	ND
	L	134 ± 11	106 ± 23	107 ± 15	96 ± 16*	91 ± 14*	90 ± 18*	89 ± 22*	93 ± 21*	102 ± 31*	100 ± 27*	99 ± 24*	ND	ND	ND	ND
	KL	169 ± 26	151 ± 19	140 ± 16	125 ± 1*	112 ± 16*	106 ± 14*	106 ± 18*	105 ± 11*	113 ± 26*	123 ± 27*	128 ± 22*	129 ± 19*	136 ± 18*	129 ± 11*	123 ± 7*
SAP	K	152 ± 9	128 ± 19	121 ± 22	123 ± 10	146 ± 20	140 ± 22	135 ± 16	120 ± 15*	116 ± 13*	117 ± 14*	126 ± 18	ND	ND	ND	ND	ND
	L	134 ± 13	138 ± 18	133 ± 30	137 ± 29	137 ± 36	135 ± 29	126 ± 21	122 ± 24	120 ± 21	122 ± 23	125 ± 20	ND	ND	ND	ND	ND
	KL	152 ± 27	133 ± 15	122 ± 16	113 ± 18	121 ± 36	120 ± 23	115 ± 17	108 ± 17*	108 ± 13*	124 ± 16	133 ± 34	131 ± 25	139 ± 30	133 ± 17	131 ± 13
DAP	K	117 ± 15	87 ± 11	73 ± 18	64 ± 6	106 ± 15	99 ± 29	86 ± 21*	73 ± 21*	78 ± 9*	82 ± 16*	91 ± 14	ND	ND	ND	ND	ND
	L	96 ± 19	88 ± 23	80 ± 13	74 ± 18	76 ± 19	81 ± 29	73 ± 23	62 ± 17	71 ± 21	72 ± 17	80 ± 8	ND	ND	ND	ND	ND
	KL	107 ± 19	88 ± 9	89 ± 22	77 ± 18	76 ± 17	69 ± 30*	64 ± 21*	62 ± 22*	66 ± 21*	78 ± 24	83 ± 24	93 ± 18	94 ± 14	100 ± 13	101 ± 11
MAP	K	130 ± 12	106 ± 12	94 ± 16	90 ± 5	123 ± 14	117 ± 25	105 ± 16*	93 ± 15*	93 ± 6*	96 ± 18*	105 ± 13	ND	ND	ND	ND
	L	117 ± 10	107 ± 13	103 ± 18	99 ± 20	104 ± 24	103 ± 25	95 ± 17	89 ± 14	92 ± 16	90 ± 16	96 ± 13	ND	ND	ND	ND
	KL	128 ± 22	108 ± 11	104 ± 19	92 ± 17	93 ± 22*	90 ± 24*	85 ± 17*	82 ± 14*	85 ± 15*	98 ± 25*	105 ± 20	109 ± 17	115 ± 16	116 ± 12	116 ± 9

EA = epidural administration; HR = heart rate (beats/min); SAP = systolic arterial pressure (mmHg); DAP: diastolic arterial pressure (mmHg); MAP = mean arterial pressure (mmHg); K = ketamine (2.5 mg/kg); L = lidocaine (4.0 mg/kg); KL = ketamine 2.5 mg/kg + lidocaine 4.0 mg/kg; ND = not determined. Values are presented as mean ± standard deviation. *Denotes statistical significance from baseline ($P < 0.05$).

Table 2: **Respiratory rate, oxygen saturation and rectal temperature in 6 cats given epidural injections of ketamine, lidocaine or ketamine/lidocaine.**

	Treatment	Baseline	Time (min)												
			5	10	15 (EA)	30	45	60	75	90	105	120	135	150	165	180
RR	K	68 ± 30	54 ± 13	48 ± 13	48 ± 9	48 ± 12	52 ± 18	46 ± 13	46 ± 18	46 ± 13	47 ± 17	47 ± 19	ND	ND	ND	ND
	L	48 ± 14	49 ± 8	46 ± 9	43 ± 11	45 ± 7	44 ± 7	39 ± 8	36 ± 9	37 ± 8	38 ± 9	39 ± 10	ND	ND	ND	ND
	KL	43 ± 13	44 ± 6	47 ± 12	41 ± 9	37 ± 13	43 ± 15	40 ± 11	39 ± 10	36 ± 6	39 ± 11	38 ± 11	39 ± 9	38 ± 7	38 ± 3	40 ± 4
SpO_2	K	99 ± 2.3	96 ± 3.3	91 ± 1.6*	90 ± 1.4*	93 ± 3.5	94 ± 5.0	93 ± 7.5	96 ± 4.4	95 ± 3.0	97 ± 2.3	98 ± 1.0	ND	ND	ND	ND
	L	99 ± 1.2	93 ± 3.2*	91 ± 3.6*	88 ± 4.8*	90 ± 2.4*	94 ± 3.3*	95 ± 2.8*	95 ± 3.7	95 ± 1.5	95 ± 2.2	97 ± 2.1	ND	ND	ND	ND
	KL	100 ± 0.5	96 ± 2.5*	94 ± 3.3*	93 ± 1.9*	98 ± 1.8	99 ± 1.4	98 ± 0.8	98 ± 1.4	98 ± 0.8	98 ± 1.0	100 ± 0.5	99 ± 0.8	99 ± 0.6	99 ± 0.8	99 ± 0.5
RT	K	38.3 ± 0.5	38.6 ± 0.6	38.7 ± 0.6	38.5 ± 0.7	38.1 ± 0.9	37.8 ± 1.0	37.2 ± 1.4	36.9 ± 1.3	36.8 ± 0.9*	36.6 ± 0.7*	36.8 ± 0.6*	ND	ND	ND	ND
	L	38.4 ± 0.7	38.6 ± 0.5	38.6 ± 0.4	38.6 ± 0.6	38.2 ± 0.5	37.7 ± 0.7	37.5 ± 0.6	37.4 ± 0.8	37.3 ± 0.9	37.1 ± 0.8*	37.1 ± 0.9*	ND	ND	ND	ND
	KL	38.5 ± 0.5	38.5 ± 0.5	38.4 ± 0.4	38.4 ± 0.4	38.4 ± 0.4	37.6 ± 0.5*	37.2 ± 0.3*	37.1 ± 0.4*	37.1 ± 0.4*	37.0 ± 0.4*	36.9 ± 0.5*	36.8 ± 0.5*	36.9 ± 0.6*	37.4 ± 0.4*	37.6 ± 0.3*

EA = epidural administration; RR = respiratory rate (breaths/min); SpO_2 = arterial oxygen saturation measured by pulse oximetry; RT = rectal temperature: K = ketamine (2.5 mg/kg); L = lidocaine (4.0 mg/kg); KL = ketamine 2.5 mg/kg + lidocaine 4.0 mg/kg; ND = not determined. Values are presented as mean ± standard deviation. *Denotes statistical significance from baseline ($P < 0.05$).

ketamine[6]. In our study, lidocaine in combination with ketamine produced analgesic/anaesthetic block lasting twice as long as that observed with use of each drug alone. A high concentration of local anaesthetic inside the nerve fibres is required for a longer period of anaesthesia[15,23]. However, this high concentration can increase neurotoxicity and prolong hypotension[17,19]. Like local anaesthetics, ketamine caused a dose-dependent increase in the duration of the nociceptive response in horses[9] and goats[10]. In the present study, ketamine 2.5 mg/kg produced approximately 70 min of analgesia in regions tested, whereas this dose combined with lidocaine produced 140 min of analgesia. Lamont (2002)[21] recommended that cats be given lidocaine 4 mg/kg or bupivacaine 1.0 mg/kg , in a final volume of 1 mℓ per 4.5 kg body weight, with or without morphine (0.1 mg/kg). These combinations are useful for providing long-lasting postoperative analgesia with no loss of motor function. Ketamine is a non-competitive antagonist of NMDA receptors and interacts with opioid, monoaminergic and muscarinic receptors and with voltage-sensitive Ca^{2+} channels[18] in the spinal cord.

Some level of analgesia occurred in all 6 cats 2 to 4 min after epidural administration of ketamine, lidocaine or the ketamine/lidocaine combination in all anatomical regions, but intensity and duration varied. The K and L treatments had similar times between analgesic and motor blocks. The L treatment was associated with a longer period of ataxia compared with the analgesic period. Epidural injection of ketamine does not enable the drug to reach motor fibres, but when the drug is administered in combination with adrenaline, it causes motor blockage[5]. In this study, epidural administration of ketamine induced ataxia and recumbency, suggesting a possible action of the drug as a local anaesthetic. The ketamine/lidocaine combination induced a longer interval of ataxia, which, however, was shorter than the period of analgesia.

Systemically, ketamine affects the cardiovascular system, with increased HR, systemic and pulmonary arterial blood pressure, systemic and pulmonary vascular resistance[30] and respiratory depression in humans[7]. Ketamine probably increases HR for the most part through a centrally mediated, generalised increase in sympathetic tone[4]. Ketamine produces its sympathomimetic actions primarily by direct stimulation of central nervous system (CNS) structures[37]. In this study, K, L, and KL treatments decreased HR significantly. We suppose that this effect occurred because of sedation with xylazine before administration of the drugs, given that after epidural injections, HR remained stable until the end of the experiment. The central actions of alpha 2-adrenergic drugs, such as xylazine, decrease sympathetic outflow and increase parasympathetic outflow from the CNS and cause decreased adrenergic activity, hypotension and bradycardia[13]. The positive chronotropic effects of ketamine probably temporarily and partially counterbalance the bradycardic effect of alpha 2-adrenergic agonists[28]. In this study, all treatments decreased arterial blood pressure, with ketamine and the ketamine/lidocaine combination producing decreases significantly different from the control at several times. Other studies involving epidural or subarachnoid injections of ketamine alone or in combination did not induce alterations in arterial blood pressure in goats[11] or horses[14]. This finding was probably due to sedation with xylazine, volume, the application site or the animal species. In this study, epidurally administered ketamine, lidocaine and ketamine/lidocaine did not induce alterations in RR in cats. However, saturation of peripheral O_2 decreased in all treatments at different times. This decrease began before epidural administration of the drugs. Therefore, it was concluded that this alteration was due to pre-sedation with xylazine.

RT decreased at a later stage in all treatment groups in this study. Alpha 2-agonists have been reported to induce prolonged depression of thermoregulation[3,16]. These agents have also been found to depress hypothalamic noradrenergic alpha 2-receptors and cause hypothermia[24]. The decrease in body temperature after xylazine administration in the present study is in keeping with findings of other reports concerning use of alpha 2-agonist drugs in cats[3,16,33]. The decrease in RT can also be explained by the cutaneous vasodilatation caused by epidural injection of lidocaine[11] or ketamine[22]. Other factors such as reduced muscular activity, generalised sedation and direct effects on thermoregulation in the CNS by sedative and analgesic agents can lead to this decrease in RT in cats.

CONCLUSIONS

Administration of the combination of *S*(+)-ketamine (2.5 mg/kg) and 2 % lidocaine (4.0 mg/kg) *via* the epidural space is a more effective means of increasing duration of analgesia in healthy cats than the administration of ketamine or lidocaine alone, with a decrease in heart rate and little interference in arterial blood pressure. Epidural administration

of ketamine/lidocaine produces ataxia, but with a shorter duration compared with the period of analgesia. However, further investigations of the dose–response relationship needs to be undertaken.

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