MEETING ABSTRACTS

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Antinociceptive effect of intrathecally applied alpha$_2$ agonists (xylazine and detomidine) in sheep and the response to atipamezole

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This study evaluated the antinociceptive and physiologic effects of xylazine (X) and detomidine (D) administered intrathecally (IT) at the lumbosacral space, before and after the injection of atipamezole (A) IV.

The study was approved by the National Animal Protection Authorities. Five adult healthy female sheep were anaesthetized with propofol on four occasions to inject the following treatments IT: groups 1 and 2, 0.05 mg kg^{-1} X (2 mg mL^{-1} saline) IT; groups 3 and 4, 0.01 mg kg^{-1} D (0.5 mg mL^{-1} saline) IT (Waterman et al. 1988). Nociceptive threshold (TH) was tested by applying pulsed and stepwise enhanced direct current (Ludbrook et al. 1995) at one hind leg pastern and noting the current at the moment of foot lift. Maximum current applied was 40 mA. Baseline TH was measured twice before anaesthesia and every 10 minutes when the sheep regained consciousness. Atipamezole was given IV immediately after reaching maximum analgesic action of X and D as defined by two equal or decreasing TH values and measurements were continued for 90 minutes. The dose of A for groups 1 and 3 was 0.005 mg kg^{-1} (0.25 mg mL^{-1} saline) IV, and for groups 2 and 4 was 0.0025 mg kg^{-1} A (0.25 mg mL^{-1} saline) IV. Heart rate (HR), mean direct arterial pressure (MAP), PaO_2 and $PaCO_2$ were measured. The differences between measurements recorded before and after treatment were analysed using a paired *t*-test for the drug effects and a nonparametric Wilcoxon's rank sum test for the comparison between groups. A p-value < 0.05 was considered significant.

All sheep were able to stand before A IV. Threshold baseline value was 4.5 ± 1.7 (mean $\pm$ SD) mA for all animals. Xylazine caused a significantly higher TH rise (35.2 ± 1.8 mA), faster onset (21.1 ± 16.0 minutes) and longer duration of the TH enhancement (104.1 ± 8.6 minutes) than D (TH: 16.3 ± 7.8 mA, onset: 49.5 ± 28.4 minutes, duration: 59.3 ± 27.3 minutes). A significant increase in $PaCO_2$ was observed in the X and D treated animals, 0.39 ± 0.21 kPa (2.9 ± 1.6 mm Hg) and 0.39 ± 0.29 kPa (2.9 ± 2.2 mm Hg), respectively. Heart rate was significantly decreased by -21 ± 17 beats minute^{-1} for X animals and -13 ± 13 beats minute^{-1} for D. Mean arterial pressure (-9 ± 13 mm Hg for X and -1 ± 11 mm Hg for D animals) and PaO_2 0.65 ± 1.32 kPa (4.9 ± 9.9 mm Hg) for X and 1.45 ± 4.19 kPa (10.9 ± 31.4 mm Hg) for D animals) did not change significantly. The nociceptive threshold was not affected by A in any group. Threshold values of all X treated animals before A was 39.3 ± 1.4 mA and after was 37.2 ± 6.3 (group 1) and 40 ± 0 (group 2). Threshold values of all D treated animals before A was 21.0 ± 8.3 and after was 19.4 ± 7.3 (group 3) and 24.8 ± 8.0 (group 4).

At the dosages administered intrathecally in this study, X and to a lower degree D induce antinociception without major physiologic changes. Atipamezole up to 0.005 mg kg^{-1} IV does not affect the resulting antinociception as assessed by electrical stimulation.

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Do common anaesthetic protocols influence cerebral oxygenation?

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Near-infrared-spectroscopy (NIRS; Jöbsis 1977) has been used in humans to measure regional cerebral

oxygen saturation (rsO_2), decreases of which can be indicative of a critical cerebral O_2 supply (Kirkpatrick et al. 1998). This study investigated changes in rsO_2, the redox status of cytochrome a/a_3, and the cerebral total hemoglobin content using NIRS during different sedative and anaesthetic techniques in dogs.

Eighty four foxhounds and 32 beagles aged 12.9 ± 13.4 (6.3–94.4) (±SD (range)) months and weighing 24.8 ± 6.6 (2.7–43) kg were divided randomly into four groups. Group AM received 0.1 mg kg^{-1} acepromazine and 0.5 mg kg^{-1} l-methadone/fenpipramid (l-Polamivet) IV, group DM 0.5 mg kg^{-1} diazepam and 0.5 mg kg^{-1} l-Polamivet IV, and group MM 40 μg kg^{-1} medetomidine and 0.5 mg kg^{-1} l-Polamivet IV. Group P was anaesthetized (7 mg kg^{-1}) and maintained (0.3 mg kg^{-1} $minute^{-1}$) with propofol. Thirty minutes after sedation groups AM, DM and MM received 8 μg kg^{-1} naloxone IV, and at 35 minutes group MM received 200 μg kg^{-1} atipamezole IV. NRIS was monitored continuously (recording interval, 1 second). Blood gases were analysed before and every 5 minutes after sedation or induction, and at 2 and 7 minutes post-naloxone injection or end of propofol infusion. Descriptive and comparative statistics (global: Kruskal–Wallis test, level of significance $\alpha = 0.05$, group comparison: Mann–Whitney test, $\alpha = 0.0083$), and Spearman's correlation were determined using the statistical software SPSS.

Before drug administration rsO_2 was $65 \pm 7\%$ ($n = 109$). One minute after drug administration significant differences between groups were found (Table 1). There was a significant increase of about 8% in rsO_2 over time in the *p* group, whereas groups AM, DM and MM showed a significant decrease with the greatest reduction of 20% in group MM. One minute after naloxone IV pre-induction values were rapidly attained in groups AM ($65.2 \pm 6\%$, $n = 31$) and DM ($65.4 \pm 5.4\%$, $n = 24$) while in group MM ($52.1 \pm 10.8\%$, $n = 27$) pre-induction values of rsO_2 were not reached until 1 minute after atipamezole IV ($62.9 \pm 7.4\%$). Periodic breathing (MM) was mirrored

Table 1 Regional cerebral oxygen saturation (%) after sedation or propofol anaesthesia at 0 minute

	Time (minutes)							
	0	**1**	**2**	**3**	**4**	**5**	**10**	**30**
AM								
Median	64.5	65.4	61.7	62.1	60.1	58.8	55.2	61.3
25%-p.	60	60.8	58.1	56.9	58.1	54.9	50.1	55.2
75%-p.	68.7	69.5	68	65.8	64.2	64.4	60.7	63.9
n	30	29	29	30	30	30	30	29
DM								
Median	64.6	62.4	53.8	55.2	55.9	55.4	54.3	57.4
25%-p.	59.7	57	49.1	50	48.2	51.8	50	51.3
75%-p.	69.6	65	60.9	60	59.4	60.7	60.3	60.6
n	23	24	25	25	25	25	24	25
MM								
Median	67.1	61.3	53.6	49.3	51.4	52	50.6	50.4
25%-p.	60.3	56.2	42.2	39.2	43.8	42.8	43.5	42.8
75%-p.	69.6	64	58.8	57.3	57.2	57.3	56.9	55.5
n	25	25	26	26	26	26	27	27
P								
Median	66	67	68.6	69.3	69.2	69.7	70.5	70.1
25%-p.	60	62.5	63.7	65.4	64.2	65.7	65.6	64.4
75%-p.	71.1	69.8	71.7	73.2	73.3	74	75	76.1
n	26	25	26	26	26	26	26	25
Inter-group differences $p < 0.0083$		P:DM P:MM	AM:MM AM:DM P:DM P:MM	AM:all P:all	AM:all P:all	AM:MM P:all	P:all	AM:MM P:all

by variations of rsO_2. There were no significant changes or intergroup differences in the redox state of cytochrome a/a_3 or of total cerebral hemoglobin content indicating cerebral blood volume (Wyatt et al. 1990).

The 20% decrease in rsO_2 in group MM is in the range regarded as indicative of a critical cerebral O_2 supply in humans (Kirkpatrick et al. 1998). The effects of naloxone and atipamezole and the correlation with blood gas analysis demonstrate an influence of respiratory depression on the vascular cerebral O_2 status whereas the lack of cytochrome a/a_3 reduction indicates an undisturbed cellular O_2 supply (Jöbsis 1977). In spite of some limitations in terms of applicability (Alef 2002). NIRS seems to enable the influence of clinical routines on cerebral oxygenation to be monitored although further validation of NIRS in the dog and the cat is required.

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Short-term anaesthesia in piglets using a liquid injection, rebreathing, inhaler device

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An economical anaesthetic technique of short duration that can be administered to piglets in the field is desirable for humanitarian reasons, for castration, tail docking or other brief procedures. Using the principles of anaesthetic uptake and distribution, an inhaler was developed to vaporize and administer isoflurane to piglets.

The inhaler design consisted of a mask, vaporization chamber and a rebreathing bag. A stopcock provided access for injection of liquid isoflurane onto a wick contained in the vaporization chamber. Inspiratory and expiratory flow of air over the wick enhanced anaesthetic vaporization. The amount of liquid isoflurane required for induction and 2–3 minutes of surgical anaesthesia was calculated using the square root of time model proposed by Lowe & Ernst (1981) for liquid injection, closed circuit anaesthesia in people. Calculations were based on an assumed MAC of 1.4% and the achievement of a target alveolar concentration of 1.3 MAC to provide a surgical plane of anaesthesia. The appropriate isoflurane concentrations in the mask, inhaler, rebreathing bag and the piglet's FRC and tissues were calculated. Original calculations were based on metabolic size ($BW^{0.75}$) and then converted to weight (kg). Based on the piglet's scale weight, the total microliters of liquid isoflurane required were formulated into a table for field use. Isoflurane was injected into the inhaler stopcock followed by oxygen to fill the rebreathing bag and initiate vaporization. After the mask was placed over the piglet's nose a slide switch was activated to allow gases to move in and out of the inhaler and rebreathing bag. Fifty-seven male piglets weighing (mean $\pm$ SD 3.0 $\pm$ 0.7 kg) and aged 7.7 $\pm$ 1.0 days were randomly selected to receive anaesthesia prior to castration. Remaining littermates served as controls for assessing morbidity or mortality. The time to induction, recovery and total anaesthetic time were measured. The $P_E'CO_2$ was measured at the piglet's nostril immediately after the mask was removed at the end of the surgical procedure. Data were analysed in SAS using the Proc Mixed procedure.

Inductions were rapid, 47 $\pm$ 9 seconds, generally with minimal or no resistance. The duration of surgery was 1–2 minutes. Anaesthesia was adequate and recovery was rapid, 122 $\pm$ 44 seconds. Total time from start to standing was 260 $\pm$ 51 seconds. The $P_E'CO_2$ was 5.2 $\pm$ 1.1 kPa (39.4 $\pm$ 8.4 mm Hg). There was no morbidity or mortality associated with either group of piglets. After piglets were standing and mobile, they were returned to the sow and other littermates, where they immediately started nursing and were indistinguishable from littermates except by determination of ear notch number.

This technique provides safe, rapid anaesthesia and recovery that is appropriate for use by veterinarians for brief field procedures.

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The effect of low dose rocuronium bromide on eyeball position, muscle relaxation, and ventilation in dogs

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A central eyeball position is often required during sedation or anaesthesia to facilitate examination of the eye. However, use of neuromuscular blockade to produce a central eye position may result in depressed ventilation. This study evaluated the eyeball position, muscle relaxation and changes in ventilation during general anaesthesia after the IV administration of 0.1 mg kg^{-1} rocuronium.

With client consent, 12 dogs of different breeds, body mass 27.2 ± 11.8 kg, aged 5.6 ± 2.8 years (mean ± SD) were anaesthetized for ocular examination. Pre-anaesthetic medication was 0.01 mg kg^{-1} medetomidine and 0.2 mg kg^{-1} butorphanol IV. Anaesthesia was induced with propofol to effect and maintained with 10 mg kg^{-1} $hour^{-1}$ propofol by infusion. The dogs were placed in left lateral recumbency, their trachea intubated and connected to a circle breathing system ($FIO_2 = 1.0$). All dogs breathed spontaneously. The superficial peroneal nerve of the right hind leg was stimulated every 15 seconds with a train-of-four (TOF) stimulation pattern and neuromuscular function was assessed with an acceleromyograph (TOF-Guard). Adequacy of ventilation was measured with the Ventrak 1550. After 10 minutes of anaesthesia to allow stabilisation of baseline values, 0.1 mg kg^{-1} rocuronium was administered IV. Minute volume (VM), tidal volume (VT), respiratory rate (RR), $PE'CO_2$ and maximal depression of T1 and TOF ratio were measured. Data were analysed using a paired *t*-test. The changes in the eyeball position were recorded.

A total of 100 ± 33 seconds after the injection of rocuronium, T1 was maximally depressed to 62 ± 21% and the TOF ratio to 42 ± 18% of baseline values. Both variables returned to baseline after 366 ± 132 seconds (T1) and 478 ± 111 seconds (TOF). There was no significant reduction in VM (2.32 ± 1.1 L $minute^{-1}$), VT (124.1 ± 69.3 mL) and RR (10 ± 3.8 breaths $minute^{-1}$) and no increase in $PE'CO_2$ (6.5 ± 2.1 kPa (48.8 ± 16.1 mm Hg)) throughout the procedure. The eyeball rotated to a central position 35 ± 7 seconds after rocuronium IV and remained there for a minimum of 20 ± 7 minutes in all dogs.

We conclude that rocuronium at a dose of 0.1 mg kg^{-1} can be administered to dogs IV with minimal changes in ventilatory variables. The eyeball is fixed in a central position for at least 20 minutes, which greatly facilitates clinical examination.

A comparison of two different methods for dead space measurements in ventilated dogs

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The aim of the study was to compare two methods of measuring physiological dead space/tidal volume ratio (VD/VT) and alveolar dead space (VD_{ALV}). Measurements were obtained by automated single breath CO_2 analysis (Ventrak 1550/Capnoguard 1265 (V&C)) and classical calculations were carried out using the Enghoff–Bohr equation in anaesthetized dogs.

The V&C consists of a mainstream capnometer, a pneumotachometer, a signal processor, and computer software to determine continuous single-breath CO_2 analysis (SBT-CO_2). Eleven dogs of mixed breed (five female, six male) mean body mass 35 ± 10 kg, aged 9 months to 8 years were studied. Pre-anaesthetic medication was acepromazine (0.03 mg kg^{-1}) and methadone (0.1 mg kg^{-1}). Anaesthesia was induced with propofol given to effect and maintained with propofol (10 mg kg^{-1} $hour^{-1}$) and fentanyl (0.02 mg kg^{-1} $hour^{-1}$) by infusion. The dog's trachea were intubated and the carbon dioxide and flow sensor were placed between the tube and the Y-piece of a circle system ($FIO_2 = 1.0$). Controlled ventilation was started (tidal volume 10–15 mL kg^{-1}) and settings were not changed throughout the measurement period. Mixed expired PCO_2 ($PECO_2$) was measured by analyzing expired gas collected in a mixing box in the expiratory limb of the circle system. The dorsal pedal artery was cannulated for arterial blood sampling and analysis. Measurements were done every 15 minutes for 1 hour. The VD/VT was automatically calculated and displayed from the SBT-CO_2 analysis and also obtained using the Enghoff modification of the Bohr equation ($VD/VT = (PaCO_2 - PECO_2)/PaCO_2$). Alveolar dead space was determined by calculating the physiological dead space (VDphys = expired volume × (VD/VT)) and subtracting the anatomical dead space measured by SBT-CO_2. Values for VD/VT and VD_{ALV} obtained with both methods were compared using Students *t*-test.

The mean values from the automatic dead space calculation (VD/VT: 0.62–0.63; VD_{ALV}: 56.1–64.3 mL) did not differ significantly from those calculated

arithmetically (V_D/V_T: 0.62–0.63; $V_{D_{ALV}}$: 54.09–66.31 mL). The mean differences and standard deviation in V_D/V_T was 0.63 ± 0.00 and in $V_{D_{ALV}}$ 58.98 ± 4.28 mL for the two measurement techniques.

Our data indicate that V&C can be used for accurate noninvasive online V_D/V_T and $V_{D_{ALV}}$ measurements in anaesthetized ventilated dogs.

Adaptation of thermal threshold analgesiometry for NSAIDs in cats: effects of ketoprofen

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Nonsteroidal anti-inflammatory drugs (NSAIDs) are widely used to provide analgesia in clinical veterinary medicine, but there are few objective data evaluating this effect under controlled conditions in cats. Analgesia is more difficult to detect with acute analgesiometry after NSAIDs than after opioids. This investigation aimed to adapt the feline thermal analgesiometry method previously employed with opioids (Dixon et al. 2002) for use with NSAIDs.

Ketoprofen, a COX1 inhibitor licensed for cats was chosen. Six cats (2 neutered, four entire females, weighing 2.2–5.4 kg) were studied in two blinded randomized crossover trials each at least 2 weeks apart. Thermal thresholds (TT) were measured using the thermal threshold-testing device previously developed for cats. A heater element and temperature sensor in a small probe were held at constant pressure against the cats' shaved thorax with an elasticized band. Skin temperature was recorded before each test, then the heater activated. When the cat responded by flinching, turning or jumping the heater was turned off and the temperature recorded. In the first study TT were measured following subcutaneous (SC) injection of ketoprofen (2 mg kg^{-1}) or a similar volume of saline. In the second study, prior to TT, and under isoflurane restraint, a mild inflammatory focus was produced at the probe site by five SC injections of 5 mg kaolin in 0.1 mL saline at each corner and in the center of a 1.5-cm square. Saline or ketoprofen as in the first study were injected at the same time. Three baseline temperatures were recorded before any injections were given. Thermal thresholds were measured at 1 and 2 hours and then two-hourly for 24 hours. Data were analysed using ANOVA.

Baseline skin temperature increased (37.3 ± 0.5–38.1 ± 0.8 °C) 24 hours after saline injection in study 2 ($p < 0.05$) but did not change after any other treatment. Thermal thresholds decreased (40.0 ± 1.3 to 39.1 ± 0.4 °C) 16 hours after ketoprofen in study 1 ($p < 0.05$) and increased (41.6 ± 1.5–44.8 ± 6.1 °C) 16–24 hours after ketoprofen in study 2 ($p < 0.05$), with no significant changes after saline. No obvious increase in sensitivity to thermal stimulation after kaolin injection was detected although obvious inflammation was present for up to 36 hours and the cats responded to digital pressure at the treated site.

The method detected some effects of a COX1 selective NSAID and may be suitable for future NSAID studies in cats. However, a pressure stimulus (Dixon et al. 2000) may prove better than thermal, and it requires investigation.

Acknowledgments

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Influences of carprofen and the experience of the surgeon on post-castration pain in lambs and young sheep

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This study evaluated the influences of carprofen and the experience of the surgeon on post-castration pain in lambs and young sheep castrated in the field.

A total of 201, 1–6-week-old lambs with a mean body mass of 11.0 ± 2.4 kg (mean ± SD) were castrated after local application of 4 mL lidocaine (2%). Preoperatively lambs were given either 20 mg carprofen SC or an equal volume of saline in a randomized order. A further 34 sheep, aged 6 months, with a mean body mass of 36.0 ± 4.5 kg were castrated after local application of 7 mL lidocaine (2%). Preoperatively sheep received 2 mg kg^{-1} carprofen SC or an equal volume of saline in a randomized order. Lidocaine was injected SC in front of, behind and into each spermatic cord. All surgery was performed using the same Burdizzo clamp (jaw 45 mm wide, handles 225 mm) either by students or experienced veterinary surgeons. Pain was assessed preopera-

tively and at 24, 48 and 72 hours after castration by two independent observers unaware of the treatment group. Observers scored pain using a visual analog scale (100 mm) after applying gentle pressure onto the scrotal region (Thornton & Waterman-Pearson 1999). Swelling of the scrotal region was scored as 'severe', 'present but not severe' and 'not present'. The VAS scores were compared using a Mann–Whitney test, $p < 0.05$ was considered significant. Swelling was compared using Fisher's exact test.

In lambs, no significant differences in pain scores between those treated with carprofen or saline were noted by either observer ($p > 0.48$) at any of the three assessment times. However, the number of lambs with severe or present swelling of the scrotal region was significantly lower in those receiving carprofen (8% *versus* 18%, $p = 0.028$). Lambs castrated by students showed significantly higher pain scores (23% *versus* 13%, $p < 0.001$ at 24 hours; 20% *versus* 13%, $p = 0.0062$ at 48 hours; 16% *versus* 10%, $p = 0.0088$ at 72 hours) and significantly more swelling (18% *versus* 8%, $p = 0.0189$) of the scrotal region than those castrated by veterinary surgeons. Young sheep receiving carprofen preoperatively had significantly lower pain scores (12 mm *versus* 25 mm, $p = 0.037$) 24 hours after surgery compared to sheep receiving saline. No scrotal swelling was obvious in sheep.

Juvenile sheep but not lambs, receiving carprofen preoperatively showed reduced pain scores up to 24 hours after surgery. Carprofen reduced swelling of the scrotum in lambs. Lambs castrated by experienced surgeons scored lower VAS scores at all times and showed less swelling of the scrotal region.

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The isoflurane sparing effect of a medetomidine constant rate infusion in horses

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This clinical study analysed the anaesthetic sparing effect of a medetomidine constant rate infusion (CRI) during isoflurane anaesthesia in horses.

Forty healthy horses undergoing different types of orthopaedic and soft tissue surgeries were studied in a randomized trial. Orthopaedic surgeries were primarily arthroscopies and splint bone extractions. Soft tissue surgeries were principally castrations with one ovariectomy. All horses received 0.03 mg kg^{-1} acepromazine IM 1 hour prior to sedation. Group A (11 orthopaedic and nine soft tissue surgeries), was sedated with 1.1 mg kg^{-1} xylazine IV, group B (13 orthopaedic and seven soft tissue surgeries) with 7 μg kg^{-1} medetomidine IV. Anaesthesia was induced in both groups with 2.2 mg kg^{-1} ketamine and diazepam 0.02 mg kg^{-1} IV. Maintenance of anaesthesia was with isoflurane (ISO) in 100% oxygen, depth of anaesthesia was always adjusted by the first author. Group B received an additional CRI of 3.5 μg kg^{-1} $hour^{-1}$ medetomidine. Respiratory rate (RR), heart rate (HR), mean arterial blood pressure (MAP), $F_E'ISO$ and $F_E'CO_2$ were monitored with a methane insensitive monitor (Cardiocap 5, Ohmeda, Anandic, Diessenhofen) and noted every 5 minutes. Arterial blood was withdrawn for gas analysis (PaO_2, $PaCO_2$) 5 minutes after the induction of anaesthesia and every 30 minutes thereafter. Dobutamine (DOB) was given as a CRI to maintain mean arterial blood pressure above 70 mm Hg. Data were averaged over time (sum of measurements/number of measurements) and tested for differences between groups by unpaired *t*-tests.

There were no significant differences between the groups in terms of body mass (group A, 508 ± 73.7 kg; group B, 529.25 ± 78.4 kg) or duration of anaesthesia (group A, 125.5 ± 36 minutes; group B, 121.5 ± 48.4 minutes). The mean $F_E'ISO$ required to maintain a surgical plane of anaesthesia was significantly higher in group A (1.33 ± 0.13%) than in group B (1.07 ± 0.19%; $p = 2.78 \times 10^{-5}$). Heart rate was different between the two groups (group A, 42.2 ± 8.3; group B, 32.6 ± 3.5; $p = 8.8 \times 10^{-5}$). Dobutamine requirements were higher in group A (group A, 0.72 ± 0.24 μg kg^{-1} $minute^{-1}$; group B, 0.53 ± 0.23 μg kg^{-1} $minute^{-1}$; $p = 0.023$). Respiratory rate, $F_E'CO_2$, PaO_2, $PaCO_2$ were not different between the groups. Adjustment of anaesthetic depth subjectively was easier with the medetomidine infusion and isoflurane (group B) than with isoflurane as a sole agent (group A). In group A 12 horses and in group B five horses showed purposeful movements on 27 (A) and 12 (B) occasions. They were given thiopental (group A, 0.0114 mg kg^{-1} $minute^{-1}$; group B, 0.0023 mg kg^{-1} $minute^{-1}$). In group A, a further 17 horses were given ketamine to deepen anaesthesia (52 occasions, 0.00426 mg kg^{-1} $minute^{-1}$) whereas in group B only

nine horses needed ketamine (34 occasions, 0.00179 mg kg^{-1} $minute^{-1}$).

An infusion of 3.5 μg kg^{-1} MED during ISO anaesthesia resulted in a significantly reduced ISO requirement.

Ketoprofen reduces the minimum alveolar concentration (MAC) in dogs anaesthetized with isoflurane and fentanyl

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Non-steroidal anti-inflammatory drugs may potentiate the opioid induced reduction in volatile anaesthetic requirements (Gomez de Segura et al. 1998). This study determined the reduction in the MAC of isoflurane (ISO) produced by ketoprofen (KETO) in dogs anaesthetized with fentanyl (FENT) and ISO.

Six healthy female crossbred dogs, weighing 13.5 ± 1.3 (mean ± SD) kg and aged 3.0 ± 0.9 years were studied. Approval of the study was obtained from the institutional ethics committee. Anaesthesia was induced in all dogs via a facemask with 5% ISO in 5 L $minute^{-1}$ oxygen. The dogs' trachea were intubated and lungs were ventilated to maintain normocapnia ($P_E'CO_2$ 4.7–6 kPa, 35–45 mm Hg). A heating pad was used to maintain body temperature. The animals were anaesthetized four times at one week intervals with the following anaesthetic and analgesic protocols randomly administered. Study 1, MAC (ISO); Isoflurane MAC. Study 2, MAC (ISO + FENT); dogs anaesthetized with ISO received a loading dose of 30 μg kg^{-1} FENT IV over 20 minutes followed by a maintenance infusion of 0.2 μg kg^{-1} $minute^{-1}$ FENT. Study 3, MAC (ISO + FENT + KETO1); as study 2 plus 1 mg kg^{-1} KETO. Study 4, MAC (ISO + FENT + KETO2); as study 2 plus 2 mg kg^{-1} KETO. The MAC was determined in duplicate by applying a standard electrical stimulus (50 V, 50 H_2 over 60 seconds via two needles placed SC over the tarsus). The stimulus was applied 15 minutes after every step change in anesthetic concentration. The Wilcoxon test was applied to data to determine significant differences among MAC measurements.

Fentanyl significantly decreased MAC (ISO) from 1.27% ± 0.02% to 0.73% ± 0.08%, a reduction of 42% ($p < 0.05$). Ketoprofen 1 mg kg^{-1} further decreased the MAC value (although not statistically significantly) with a reduction of 47% from MAC (ISO) (0.67% ± 0.13%) and 8% from MAC (ISO + FENT). When KETO 2 mg kg^{-1} was given, the reduction in MAC was 50% compared to MAC (ISO) (0.63% ± 0.08%; $p < 0.05$) and 14% compared to MAC (ISO + FENT) $p < 0.05$.

Administration of KETO further reduces MAC (ISO) compared to levels observed with FENT alone. The observed reduction may have clinical advantages.

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Clinical use of mivacurium in the cat

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This study investigated the characteristics of neuromuscular blockade (NMB) produced by mivacurium in the cat. Mivacurium is a short acting nondepolarizing muscle relaxant, which undergoes rapid hydrolysis by plasma cholinesterase and is expected to have a predictable effect of short duration (Maehr et al. 1991; Gyermek et al. 1999).

Thirteen cats (seven females, six males) aged 2–14 years, body mass 3.5–5.9 kg, undergoing corneal surgery were anaesthetized with propofol (3–6 mg kg^{-1} IV) after pre-anesthetic medication IM with an opioid alone or in combination with acepromazine (0.02 mg kg^{-1}) or medetomidine (0.01 mg kg^{-1}). Anaesthesia was maintained with isoflurane (13 cats) or halothane (1 cat) in oxygen (33%) and nitrous oxide (66%) with mechanical ventilation. Cats were monitored using an ECG, side-stream capnography, and a Doppler flow sensor which was positioned on the pedal artery to estimate systolic blood pressure. The peroneal nerve was stimulated with a train-of-four (TOF) and Double Burst (DBS) pattern using a peripheral nerve stimulator, to assess the degree of NMB. Mivacurium IV (0.1 mg kg^{-1}) was administered over 10 seconds. The TOF stimulation was applied every 12 seconds until no twitch was apparent (onset time) and continued until the TOF ratio was considered to be 1. Response to DBS stimulation was then assessed until the ratio was 1. The same procedure was followed after recovery of a DBS ratio of 1, if administration of a further dose of mivacurium (0.05 mg kg^{-1}) was required. Times of onset of NMB, return of the 1st and 4th twitches, TOF and DBS ratio of 1, were recorded, and descriptive

analyses performed. The effects of mivacurium on heart rate (HR), systolic blood pressure (SAP) and variations in onset and recovery time of NMB between the first bolus and the subsequent doses were determined using a paired Student *t*-test.

The HR and SAP did not change significantly after administration of mivacurium. The onset time was significantly shorter ($p < 0.0001$) after administration of the second dose of mivacurium (48 ± 8 seconds) compared to the initial dose (107 ± 17 seconds). The time to return of the first twitch (11 minutes 12 seconds ± 3 minutes 26 seconds) and to DBS 1 (16 minutes 41 seconds ± 4 minutes 21 seconds) were longer after the first dose than after subsequent doses (8 minutes 56 seconds ± 2 minutes 2 seconds and 18 minutes 28 seconds ± 7 minutes 19 seconds, respectively). However, a second dose of mivacurium was administered in only three cats. Reversal of NMB was never required.

Mivacurium is a short acting muscle relaxant in the cat, without apparent cumulative effect after repeated dosing. The time to complete recovery in 97.5% of patients after a single bolus is estimated to be 25 minutes (mean ± 2 SD).

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Blood pressure and electrocardiographic effects of acepromazine in anaesthetized horses

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Acepromazine, a phenothiazine tranquilizer, causes hypotension in standing horses (Parry et al. 1982). However, a retrospective study (Taylor & Young 1993) showed that acepromazine pre-anesthetic medication did not affect arterial blood pressure (MAP) in anaesthetized horses. This study examined the effects of acepromazine on MAP during romifidine–ketamine–halothane anaesthesia in horses anaesthetized for various surgical procedures.

Forty-four horses were allocated by block randomization to groups A and B. Group A received acepromazine 0.05 mg kg^{-1} IM 30 minutes before induction of anaesthesia, group B did not. All horses received romifidine 0.1 mg kg^{-1} IV 5 minutes before anaesthesia was induced with diazepam 0.05 mg kg^{-1} and 2.2 mg kg^{-1} ketamine IV. The horses' trachea were intubated and horses breathed 50% oxygen and 50% nitrous oxide plus halothane (concentration adjusted as required clinically) from a circle breathing system. Nitrous oxide was discontinued after 10 minutes and analgesics, flunixin 1.1 mg kg^{-1} and either morphine 0.1 mg kg^{-1} or butorphanol 0.05 mg kg^{-1} (matched for horses undergoing the same procedure) administered IV. The facial or dorsal metatarsal artery was catheterized for direct measurement of MAP (every 10 min) and withdrawal of blood for gas analysis (every 30 min). The electrocardiogram (ECG) was monitored continuously with a 10 seconds printout obtained every 10 minutes. Intermittent positive pressure ventilation (IPPV) was instigated if $PaCO_2$ exceeded 9.3 kPa (70 mm Hg). Dobutamine was infused (1.0–5.0 kg^{-1}minute^{-1}) if MAP < 58 mm Hg and was continued until MAP > 70 mm Hg. Mean age, weight and duration of anaesthesia were compared between the groups using a *t*-test for independent samples. Gender distribution and numbers of horses requiring IPPV or dobutamine were compared between groups using a chi-squared test (with Yates correction). To compare MAP over time, the area under the curve (MAPAUC) was calculated and compared between groups using a *t*-test. Horses receiving dobutamine were excluded from MAPAUC and MAP comparisons. The ECG printouts were examined for arrhythmias.

There were no significant differences between groups ($p > 0.05$). Group A contained three stallions, 10 geldings and nine mares, aged 6.3 years (range 0.75–18). Group B comprised eight stallions, 11 geldings and three mares aged 7.3 (1–16) years. Duration of anaesthesia was group A 97 (50–140) minutes, group B 99 (50–160) minutes. Eight horses in group A and three in group B required IPPV. Nine horses in group A and four in group B received dobutamine. Mean arterial pressure ranged from 60 to 128 mm Hg in group A and 58–96 mm Hg in group B. Mean MAPAUC was 5941 mm Hg minute^{-1} in group A, in B 6000 mm Hg minute^{-1}. Atrial premature complexes were recorded from one horse in group B. No other arrhythmias were detected.

Although MAP was lower in the acepromazine group, this appeared unlikely to cause a clinical problem. The incidence of arrhythmias was too low to determine the influence of acepromazine in this study.

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Comparative study of the effects of acepromazine and structurally related compounds on the TNF-α release by monocytes stimulated by *Chlamydia pneumoniae*

Acepromazine (ACP), a member of the phenothiazine family, has antioxidant properties and interacts with reactive oxygen species produced by stimulated neutrophils (Serteyn et al. 1999). We found that ACP reduced the differentiation of monocytes induced by an overnight incubation with a crude *Chlamydia pneumoniae* extract (Serteyn et al. 2001). The same model was used to test the effects of phenothiazines on the TNF-α release by activated monocytes.

A crude *Chlamydia pneumoniae* extract was obtained by mechanical disruption and centrifugation (1 minute, 1500 r.p.m.) of 78 hours infected McCoy cells. Monocytes (THP1 cell line; 2×10^6 cells by assay) were incubated overnight with 30 μL of *Chlamydia pneumoniae* crude extract (equivalent to an endotoxin charge of 3.5 pg) in the presence or absence of phenothiazines (from 10^{-6} to 10^{-4} M) (Mouithys-Mickalad et al. 2001). For estimation of TNF-α release, the supernatants were collected, centrifuged (to eliminate the undifferentiated monocytes) and used for TNF-α measurements ($n = 6$) (Quantikine HS human TNF-α, R&D Systems, UK). Acepromazine was compared to other phenothiazines (chlorpromazine, trifluoperazine) or to structural analogues of phenothiazines (phenoxazine, thioxanthen-9-one and methylene blue). For each assay, cytotoxicity was evaluated by microscopic examination and blue trypan exclusion method. Mean values of TNF-α were compared by a Student *t*-test ($p < 0.05$).

TNF-α release by *Chlamydia*-treated THP1 was significantly decreased by ACP in a dose-dependent manner, 378 ± 30, 209 ± 38 and 189 ± 35 ng mL^{-1} for 10^{-6}, 10^{-5} and 10^{-4} M compared to the control values 385 ± 9 ng mL^{-1}. Similar inhibitions of TNF-α release were obtained with trifluoperazine (313 ± 25 and 265 ± 14 ng mL^{-1} at 10^{-6} and 10^{-5} M) and chlorpromazine (323 ± 29 and 227 ± 13 ng mL^{-1} at 10^{-6} and 10^{-5} M), but at 10^{-4} M, these two drugs were cytotoxic. The other structurally parent compounds increased significantly the TNF-α production: 630 ± 46 and 468 ± 60 ng mL^{-1} for thioxanthen-9-one and 547 ± 17 and 331 ± 111 ng mL^{-1} for methylene blue at 10^{-5} and 10^{-6} (M). At 10^{-4} M, the two compounds were cytotoxic. Phenoxazine increased the TNF-α production, slightly at 10^{-6} and 10^{-5} M (444 ± 39 and 424 ± 16 ng mL^{-1}, respectively) and significantly at 10^{-4} M (959 ± 30 ng mL^{-1}).

Further studies are needed to verify if the inhibition of TNF-α release by some phenothiazines could be linked to a reduction of the signal transduction, especially the NF-κB pathway. These results could be interesting for the anaesthesia or treatment of animals suffering from a systemic inflammatory reaction.

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The effects of pre-anesthetic administration of xylazine on the cardiovascular responses to dobutamine in halothane anaesthetized horses

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Studies evaluating the effects of dobutamine in horses do not consistently report increases in cardiac output despite increases in arterial blood pressure. The concurrent administration of the α_2 agonist clonidine, in people, inhibited the chronotropic effects of dobutamine and increased left ventricular stroke work (Zimpfer et al. 1982). Our study was performed to determine if pre-medication with an α_2 agonist affects the response to dobutamine in anaesthetized horses.

Eleven horses were anaesthetized on four separate occasions for one of four randomly assigned treatments; (I) no xylazine, no dobutamine (II) xylazine,

no dobutamine (III) no xylazine, dobutamine, and (IV) xylazine, dobutamine. Horses received 0.02 mg kg^{-1} of butorphanol IV 10 minutes prior to anesthetic induction. Two minutes prior to induction, groups II and IV received 0.5 mg kg^{-1} of IV xylazine. Anaesthesia was induced with 6–7 mg kg^{-1} of thiopental and maintained with halothane. End-tidal halothane concentrations were maintained between 1.1 and 1.2% in groups I and III, and 0.9–1.0% for groups II and IV. Heart rate, cardiac output, right atrial pressure, and systolic (SAP), diastolic (DAP) and mean (MAP) arterial pressure were recorded 30 minutes after beginning halothane anaesthesia (T10). Cardiac output was estimated using Lithium dilution (Linton et al. 2000). Baseline measurements were repeated twice, at 5-minute intervals (T5 and T0). At time 0 (T0), an IV infusion of either saline (100 mL $hour^{-1}$) or dobutamine (0.001 mg kg^{-1} $minute^{-1}$) was started and data recorded at 5-minute intervals for 30 minutes (T5 – T30). Stroke volume and systemic vascular resistance (SVR) were calculated. Data were analysed using repeated measures ANOVA ($p < 0.01$ significant) and Newman–Keuls for multiple comparisons.

Cardiac output and stroke volume increased over time in groups III and IV. Cardiac index was higher in groups III and IV than in groups I and II from T10 until completion of the study. Estimates of cardiac index at T30 for groups I–IV were 45 ± 9, 46 ± 11, 71 ± 11, and 78 ± 19 mL kg^{-1} $minute^{-1}$, respectively (mean $\pm$ SD). Stroke index was higher in groups III and IV than in groups I and II from T15 to T30. Values for stroke index at T30 for groups I–IV were 0.98 ± 0.19, 1.11 ± 0.18, 1.46 ± 0.21, 1.74 ± 0.33 mL kg^{-1}. Heart rate decreased from T10–T30 in groups I and II. Heart rate was greater in groups I and III than in groups II and IV at T5 and T0. Values for heart rate at T0 for groups I–IV were 48 ± 5, 42 ± 5, 50 ± 4, 43 ± 4 beats $minute^{-1}$. Systolic arterial pressure, DAP and MAP were higher in groups III and IV than in groups I and II from T5 to T30. There were no differences in SVR between groups.

Dobutamine at 0.001 mg kg^{-1} $minute^{-1}$ increased cardiac output, blood pressure, and stroke volume. Premedication with xylazine at 0.5 mg kg^{-1} did not appear to affect the response to dobutamine.

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Use of the Anemon Index to evaluate the quality of analgesia during fentanyl and sevoflurane anaesthesia in pigs

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Anemon I is a new monitoring system that can be used to evaluate autonomic nervous system reactivity in real time by showing a simple, easily interpretated quantitative index (0–200), the Anemon Index (AI) (Junke et al. 2000). This study used the AI to evaluate the quality of analgesia during sevoflurane and fentanyl anaesthesia in pigs.

Six healthy pigs, weighing 24.76 ± 3.40 kg, were induced to anaesthesia with 5% sevoflurane (SEVO) in 5 L $minute^{-1}$ oxygen. After endotracheal intubation SEVO was given at 1 MAC (2.66%) in 3 L $minute^{-1}$ oxygen. Fentanyl was infused IV at 50 μg kg^{-1} $hour^{-1}$ for the first 30 minutes of anaesthesia, discontinued for 30 minutes, and then infused at 100 μg kg^{-1} $hour^{-1}$ for another 30 minutes. Three mechanical noxious stimuli (needle prick, pin-prick and pressure on the abdomen) were applied for 15 seconds at 30, 60 and 90 minutes. The AI, ECG, invasive mean arterial blood pressure (MAP), heart rate (HR), SpO_2 by pulse oximetry, tidal volume, Fe'_{SEVO}, $Fe'CO_2$ and respiratory rate were recorded before induction (baseline), after induction, after intubation and extubation, and before and during noxious stimulation at 30, 60 and 90 minutes. Recovery times were recorded. Statistically significant differences were determined by ANOVA. Spearman rank-correlation was used to evaluate the relationship between AI and hemodynamic variables. A p-value of < 0.05 was considered significant.

A significant ($p < 0.01$) decrease in AI was recorded after anaesthetic induction, from 82.3 ± 21.1 to 52.7 ± 20.3. After intubation, AI increased slightly, but not significantly, to 71.7 ± 27.1. A significant ($p < 0.05$) increase of AI occurred after extubation. Nociceptive stimuli did not have any measurable effect either on AI or on recorded cardiovascular variables. There was no movement, respiratory changes,

or any other visible response to noxious stimulation. The AI did not change significantly with the different doses of fentanyl. Respiratory depression and apnoea were seen in all animals during the fentanyl infusion; therefore, pigs received intermittent positive pressure ventilation. Anaesthesia with sevoflurane and fentanyl resulted in a significant ($p < 0.001$) decrease in MAP. Heart rate did not change significantly. There was no correlation between AI and cardiovascular variables (HR and MAP).

Endotracheal intubation caused an increase and extubation a greater significant increase in the AI. This suggests that intubation and extubation may represent stressful events during general anaesthesia, although further studies are needed to validate the use of the AI in pigs. Sevoflurane anesthetic induction may not prevent the sympathetic stimulus caused by endotracheal intubation in pigs, as indicated by the increased AI values.

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Preliminary evaluation of subjective scoring systems for assessment of postoperative pain in horses

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This study examined the performance of two subjective pain scoring systems for evaluating equine postoperative pain, and investigated differences in pain scoring tendencies of veterinarians and grooms.

Fifteen horses were included in the study. Group 1 ($n = 8$) had chronic lameness and was admitted for elective arthroscopy under general anaesthesia, on one or two femoropatellar, femorotibial or tibiotarsal joint or digital flexor tendons. The anaesthetic protocol for each horse was similar but not standardized. Multi-modal peri-operative analgesia comprised: romifidine (100 μg kg^{-1} IV); flunixin (1.1 mg kg^{-1} IV); ketamine (2.2 mg kg^{-1} IV); morphine (0.12 mg kg^{-1} IV); phenylbutazone (4 mg kg^{-1} IV/PO). Group 2 ($n = 7$) included pain free controls. At 6 hours post-recovery from anaesthesia (PR) (group 1) or at 20.00 hours (group 2 with one limb bandaged), horses were filmed undisturbed in their stables for 90 seconds (dynamic behaviour, DB); thereafter, the surgery site and pharynx of each horse were palpated (and filmed) in a standardized manner (interactive behaviour, IB). Two observer groups, seven veterinarians and eight grooms, watched video footage of each horse and assigned pain scores using a visual analog scale (VAS) and a numerical rating scale (NRS). Observers assigned a pain score (VAS and NRS) for DB and IB separately and overall.

Statistical analysis (Minitab 13.0, Wilcoxon signed rank and Mann–Whitney *U*-tests) investigated differences in pain scores attributed to groups 1 and 2 horses, compared pain scores assigned by veterinarians and grooms, and examined differences in the performance of VAS and NRS techniques. There were significant differences in the pain scores assigned by veterinarians and grooms to groups 1 and 2 horses. When using DB or IB separately (but not combined) to score perceived pain, grooms assigned higher scores to group 1 than group 2 ($U = 81.5$, $p < 0.05$; $U = 82.0$, $p < 0.05$) using the VAS. There was no difference in NRS scores attributed by grooms to groups 1 and 2. Using DB and IB separately or combined, there was no difference in pain scores attributed to groups 1 and 2 by veterinarians using either VAS or NRS scoring systems. Using separate VAS scores for DB ($W = 32.5$, $p < 0.05$) and IB ($W = 26.5$, $p < 0.05$) and using combined (DB + IB) VAS scores, grooms awarded higher pain scores ($W = 27.0$, $p < 0.05$) than veterinarians to group 1. Using the NRS, vets and grooms did not score pain differently for group 1. For group 2, grooms scored pain significantly higher than vets when using the VAS to score IB separately ($W = 21.0$, $p < 0.05$); no other differences between grooms and veterinarians in pain scoring of group 2 (NRS or VAS, DB and IB separately or combined) were identified.

The performance of subjective pain scoring systems for assessment of equine postoperative pain varies according to the scale used, the behaviour evaluated (dynamic or interactive) and the observer group. While data suggest that grooms distinguished post-surgery horses from controls more successfully than vets and assigned higher pain scores to these horses, the specific behavioral criteria on which scores were assigned requires future investigation and identification.

A comparison of the haemodynamic effects of epidurally administered medetomidine and xylazine in dogs

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Alpha$_2$ agonists have a significant role in epidural anaesthetic techniques. However, there are few reports regarding epidural administration of these drugs especially in small animals (Greene et al. 1995; Keegan et al. 1995; Vesal et al. 1996). This study compared the haemodynamic effects of xylazine and medetomidine after epidural injection in dogs.

Six dogs (four females and two males) weighing 27.5 ± 3.39 kg, aged 5.6 ± 1.42 years were studied on two separate occasions one month apart. Dogs were sedated with 0.5 mg kg^{-1} diazepam IM and 0.1 mg kg^{-1} acepromazine IM. After 20 minutes, a lumbosacral epidural injection of 0.25 mg kg^{-1} xylazine was administered (group X). One month later, following the same sedation, 15 μg kg^{-1} medetomidine was administered epidurally (group M). Haemodynamic variables (ECG and indirect blood pressure (Doppler)), respiratory rate and rectal temperature were recorded before (baseline) and then every 5 minutes after the epidural injection, up to 60 minutes. Differences between groups were compared by a paired *t*-test. Within group changes were compared to basal values by ANOVA. A p-value of < 0.05 was considered statistically significant.

Both groups showed significant reductions in heart rate (106.3 ± 7.7 beats minute^{-1} baseline *versus* 67.7 ± 7.6 (group M); 91 ± 3.8 baseline *versus* 52.3 ± 9 (group X)) and mean arterial blood pressure (113.1 ± 12.3 mm Hg baseline *versus* 87 ± 11 (group M); 118 ± 7 baseline *versus* 91 ± 14 (group X)). There were no differences between groups in these variables. After epidural injection, first degree atrioventricular block was recorded significantly more often in group X (50% against 33%) but second degree block was significantly more frequent in group M (66% against 33%). Also 50% of dogs in group X and 66% in group M showed sinus arrest. Respiratory rate decreased significantly in both groups following the epidural injection (20.66 ± 0.66 minute^{-1} baseline *versus* 16.33 ± 4.77 (group M); 37.66 ± 0.56 baseline *versus* 16.33 ± 1.81 group X), but no differences between groups were observed. Rectal temperature decreased significantly in group X (38.16 ± 0.21) with respect to the basal measurement (39.30 ± 0.14 °C). In group M, there was no significant reduction in temperature, however, no statistical difference in rectal temperature was found between groups.

This study shows that 0.25 mg kg^{-1} xylazine and 15 μg kg^{-1} medetomidine produce similar, significant cardiovascular and respiratory changes following lumbosacral epidural administration in dogs.

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