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Awards

The Topcat Metrology Research Award

Dr Polly Taylor and Dr Michael Dixon (Topcat Metrology) regularly carry out research, working and publishing with our clients all over the world. Currently there are over 50 systems active in 10 countries.

To support the academic community, Topcat Metrology is offering a prize for the best paper on a pain or analgesia-related subject by a resident (or equivalent) presented at the AVA meeting in Davos. The prize is the use of any one of the Topcat systems for a research project for up to 6 months duration. Full support will be provided, including training at a mutually agreed location in Europe. The project could involve, for instance, investigating the analgesic effect of a drug in a particular species, a clinical trial using the Prodplus to assess disease-associated or postsurgical pain, a lab animal study using the Mousemet electronic von Frey system or a development study involving our temporal summation system. Selection will be by a panel of three judges, two of them international figures in veterinary analgesia and a third vote from the directors of Topcat Metrology. The prize is valid for two years. Topcat will supply the system as soon as the winner has secured funding for all other study costs including their travel to the agreed training location in Europe.

Resident Award

The prize is a free registration for the whole congress for one of the first 10 residents who submit their abstract. The abstract needs to be accepted as short communication or poster and the resident needs to be present at the congress. The prize will be drawn by lot and awarded at the welcome reception.

AVA Tombola

The prize is a voucher for the Schatzalp bar for one of the first 10 AVA-members who register for the congress. The prize will be drawn by lot and awarded at the welcome reception.

Overview Program AVA Spring Meeting 2012 Davos

WEDNESDAY 21st of March

18⁰⁰ Registration

18⁰⁰ Welcome reception in the Lobby and outside with AVA Tombola followed by “happy sledging”

THURSDAY 22nd of March: Blood kills?

7³⁰ Registration

8³⁰ Keynote lecture: Coagulation in the perioperative period: pathophysiology, monitoring and specific, goal-directed therapy

PD Dr. med. M. Ganter, University of Zurich, Switzerland

9³⁰ Keynote lecture: Optimizing haemostasis monitoring and therapy in veterinary medicine

Prof. Dr. med. vet. B. Wiinberg, University of Copenhagen, Denmark

10³⁰ Coffee break in the exhibition hall

11⁰⁰ Keynote lecture: Blood transfusion results in adverse outcome – the scientific evidence

Prof. Dr. med. D. Spahn, University of Zurich, Switzerland

12⁰⁰ Lunch Panorama Restaurant and “happy skiing”

12³⁰ ECVAA General Meeting in the Lobby

16⁰⁰ Abstract presentations (see overview oral abstract presentations)

17¹⁵ Coffee break in the exhibition hall

17³⁰ Abstract presentations (see overview oral abstract presentations)

19⁰⁰ Aperó

20⁰⁰ Conference Dinner in the Lobby

FRIDAY 23rd of March: Opioid therapy – friend or foe?

7³⁰ Registration

9⁰⁰ Keynote lecture: Hazards of opioids in acute and chronic pain therapy

PD Dr. med. H. Rittner, Universitätsklinikum Würzburg, Germany

10⁰⁰ Coffee break in the exhibition hall

10³⁰ Keynote lecture: Opium – Almighty God’s greatest cure

Prof. Dr. med. vet. E. Clutton, The University of Edinburgh, UK

11³⁰ Panel Discussion: “Controversies regarding the use of opioids”

PD Dr. med. H. Rittner, Universitätsklinikum Würzburg, Germany

Prof. Dr. med. vet. E. Clutton, The University of Edinburgh, UK

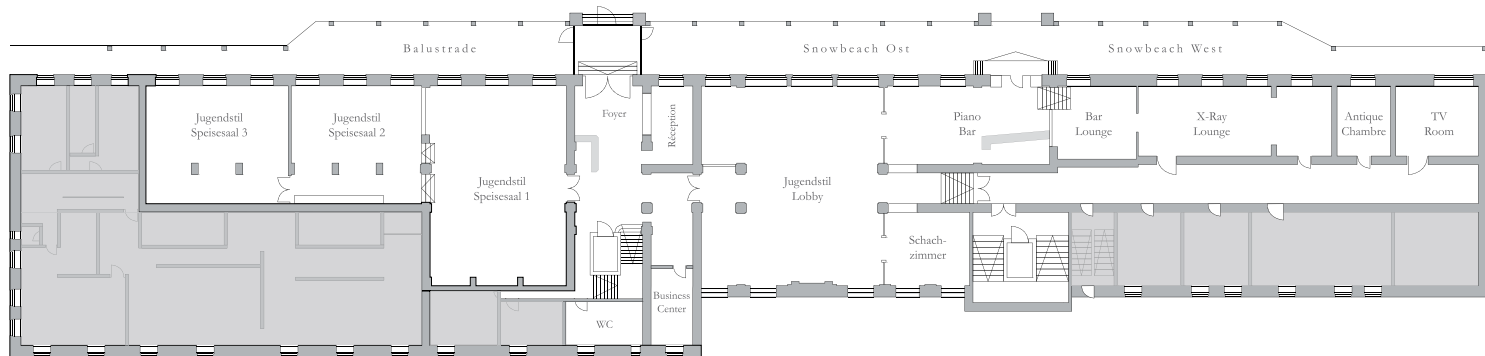
12¹⁵ Lunch Panorama Restaurant and “happy skiing”

12⁴⁵ AVA General Meeting in the Lobby

16⁰⁰ Abstract presentations (see overview oral abstract presentations)

17³⁰ Closing Ceremony

19⁰⁰ Moonshine-walk to “Alp Strela” with Fondue dinner



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	Room “Lobby“ <i>Chaired by Gabby Musk</i>	Room “Speisesaal 3“ <i>Chaired by Sabine Kästner</i>
16 ⁰⁰ - 16 ¹⁵	Mechanical nociceptive thresholds using four different probe configurations in horses. <i>PM Taylor, N Crosignani, MJ Dixon, C Lopes, AC Rosa, SPL Luna, JNP Puoli Filho</i>	The effect of pure oxygen or air/oxygen mixture on arterial blood oxygenation in spontaneously ventilated, isoflurane anaesthetised horses: a retrospective study <i>S Schauvliege, I Savvas & F Gasthuys</i>
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16 ⁴⁵ - 17 ⁰⁰	Comparative study of a new metamizole formulation and carprofen on postoperative pain in dogs undergoing ovariohysterectomy <i>KS Kalchofner Guerrero, R Wuhrmann, AMM Schwarz, S Feldmann, S Hartnack & R Bettschart-Wolfensberger</i>	Effects of a constant rate infusion of dexmedetomidine on the minimum alveolar concentration in sevoflurane anaesthetized ponies <i>M Gozalo Marcilla, K Hopster, F Gasthuys, L Hatz, AE Krajewski & S Schauvliege</i>
17 ⁰⁰ - 17 ¹⁵	Influence of dipyrone on minimal alveolar concentration (MAC) of sevoflurane and on thermal and mechanical threshold in beagle dogs <i>AF Schütter & SBR Kästner</i>	Measurements of trans-diaphragmatic pressure in ponies during total intravenous and isoflurane anaesthesia. <i>B Steblaj, S Schauvliege, K Pavlidou, I Savvas, F Gasthuys & Y Moens</i>

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KEYNOTE LECTURES

Coagulation in the perioperative period: pathophysiology, monitoring and specific, goal-directed therapy

PD Dr. med. Michael Ganter, University Hospital Zurich, Switzerland

Blood coagulation is a complex and tightly regulated physiological network of interacting proteins and cells with extensive sensitivity, amplification, and control pathways. The system represents a delicate balance between pro- and anticoagulant as well as pro- and antifibrinolytic activities. Clinically relevant phenotypes, i.e. bleeding and thrombosis immediately occur when this balance is disturbed. Treatment of a massive bleeding implies an interdisciplinary challenge for surgeons and anaesthetists. The distinctive task in perioperative coagulation management is to assess and judge both sides of the coagulation balance. Specific coagulation interventions can then be made to either side, preventing both overt bleeding and thrombosis (1, 2).

Current transfusion strategies and coagulation management are based on a detailed understanding of the coagulation physiology and a specific coagulation monitoring. In a bleeding patient, patient's medical history, clinical findings, routine and advanced laboratory coagulation testing as well as bed-side (point-of-care) coagulation monitoring helps to reliably and readily identify the underlying coagulation disorder. The key to success in terms of patient outcome is to keep the coagulation system in an optimal equilibrium so that bleeding is adequately controlled without thromboembolic adverse events (3). For any given patient, there is no simple answer to coagulation management, but instead, optimal coagulation intervention and management needs to be individually defined for each patient (1, 2, 4).

Advanced coagulation monitoring will employ a combination of routine laboratory coagulation tests, single factor measurements and whole blood, point of care coagulation testing, always keeping in mind the patient's history and clinical findings (5). Whole blood coagulation tests like Thrombelastography® (Haemonetics Corporation, Braintree, MA) or rotation Thromboelastometry® (Tem International GmbH, Munich, Germany) may overcome some of the limitations of routine laboratory coagulation tests and are increasingly being used in massive bleeding patients. With minimal time delays, they provide valuable information on overall kinetics of clot formation, clot strength, platelet function and overt fibrinolysis in whole blood (6). However, these tests are still in vitro assays; they do not reflect in vivo contributions of local tissue and the endothelium, tissue factor bearing cells, and blood flow to the naturally occurring coagulation process. Therefore, any coagulation test requires skilled

interpretation and clinical correlation in evaluating its significance for bleeding or thrombosis (7).

Patients with massive hemorrhage become coagulopathic due to several mechanisms. For example, trauma and shock directly activate the thrombomodulin-protein C pathway resulting in the acute coagulopathy of trauma and shock (8-10). Thereby, key players of the propagation phase of coagulation, the tenase (VIIIa-IXa) and prothrombinase (Xa-Va) complex are getting degraded and inactivated by activated protein C. Furthermore, plasminogen activator inhibitor 1, the principal inhibitor of tissue plasminogen activator and urokinase, activators of plasminogen and hence fibrinolysis is consumed through activated protein C, resulting in increased fibrinolysis. The developing coagulopathy then gets worse through the better known pathogenetic factors: consumption and dilution of coagulation factors, hypothermia and acidosis.

Fibrinogen is the substrate of coagulation and is usually the first coagulation factor to become critically low in massive bleeding (3). According to Hiippala et al. (11), fibrinogen levels fall below 1 g/L after a loss of 150% of the calculated blood volume. Factor II, V, VII and platelet levels become critical later, after a loss of >200% of the blood volume. However, these figures are very general and do not help greatly in the individual case. In addition, the arbitrary definition of the critical level determines when the corresponding level will be reached i.e. after what blood volume loss.

If patients present with clinical and objective signs of coagulopathic bleeding, treatment with allogeneic blood products (fresh frozen plasma, cryoprecipitate, platelet concentrates), factor concentrates, pharmacological interventions or a combination thereof has to be initiated. Evidence-based recommendations, like the one from the multidisciplinary Task Force for Advanced bleeding Care in Trauma (3), updated in 2010 are very helpful for optimal patient care.

Transfusion of allogeneic blood products is independently associated with increased mortality and major adverse cardiac and non-cardiac outcomes (12). One strategy to reduce bleeding and avoid allogeneic blood transfusion in surgical patients at increased risk of bleeding is the use of anti-fibrinolytics. Since marketing of the antifibrinolytic aprotinin has been suspended in 2007, only two anti-fibrinolytics remained commercially available for patient use, i.e. ϵ -aminocaproic acid and tranexamic acid (TXA). Both drugs are lysine analogues and inhibit fibrinolysis by competitively blocking the lysine binding site on plasminogen (13). Lysine analogues have shown to reduce blood loss and the need for allogeneic red cell transfusion, especially in cardiac, liver, orthopedic surgery and most

recently in trauma patients (CRASH-2 trial) (14-16). The lysine analogues (evidence is stronger for TXA than for ϵ -aminocaproic acid) were probably as effective as aprotinin in most studies but at lower costs (17).

How should all these aspects translate into perioperative, hemostatic management? First, we have to thoroughly understand the pathophysiology of the deranged coagulation system in massive bleeding, and in particular understanding that blood coagulation does not only consist of pro-coagulant proteins. We always have to consider the four elements of the coagulation system and keep them in balance, i.e. pro- and anti-coagulant as well as pro- and anti-fibrinolytic ‘subsystems’. Second, we have to carefully diagnose the main problem of the disturbed coagulation system with patient’s history, clinical findings and adequate blood tests. Since blood coagulation may change rapidly during massive hemorrhage, frequent re-assessment is necessary. Furthermore, we need to exactly know how to interpret the blood tests ordered, what they can tell us and where their limitations are. Third, we are to initiate the specific treatment needed by the individual patient early. The better we know the underlying pathophysiology, the better we can diagnose and treat our patients targeted to their individual needs.

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Optimizing Haemostasis Monitoring and Therapy in Veterinary Medicine

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The ability of a laboratory assay to reveal and correlate to clinical phenotype is crucial for rational hemostasis monitoring. The ideal hemostasis assay should therefore be able to identify both biochemical and cellular abnormalities in the hemostasis system and at the same time correlate to the clinical signs of the patient.

Effective haemostasis depends on an adequate number of functional platelets, an adequate concentration and activity of plasma coagulation and fibrinolytic proteins, and a normally responsive vasculature. The traditional diagnostic approach to the haemostatic system is usually performed with test of primary haemostasis (platelet count and buccal mucosal bleeding time) and secondary haemostasis through plasma based assays designed to further localize defects, such as the aPTT (intrinsic & common pathway) and PT (extrinsic & common pathway). The fibrinolytic system is traditionally evaluated with measurements of degradation products such as FDP's and D-Dimer and endogenous anticoagulant ability has been evaluated through, AT, protein C (PC) and protein S (PS). Additional specialized individual coagulation factor tests can be performed to further localize congenital defects. This traditional approach with plasma-based coagulation screening tests is optimized towards localizing the defective or deficient coagulation protein, but it is time consuming and the results often correlate poorly with the clinical phenotype.

The introduction of the cell-based model of hemostasis has increased our understanding of the complex biochemistry of physiologic and pathologic hemostasis and has forced a re-evaluation of the traditional view of the intrinsic and extrinsic pathways of coagulation. The cell based model of hemostasis has made it evident that *in vivo* hemostasis is influenced by numerous other pro- and anti-coagulant components than those present in blood plasma alone and it is important that the methods used for the diagnosis and monitoring of patients take this into consideration. Although citrated plasma contains many of the factors involved in coagulation, whole blood (WB) contains both the soluble factors and intravascular cells active in

physiologic and pathologic haemostasis, incorporating TF and phospholipid bearing cells. In particular TF expression in certain types of tissue and cellular components of the blood such as the activated platelets and leukocytes, supply a surfaces for initiation, amplification and propagation of clot formation and thus play key roles in hemostasis. These cellular and tissue components are themselves influenced by inflammatory- and immune-responses during disease.

Although these recent advances in our understanding of haemostasis have shown that the division between primary and secondary haemostasis is evidently not biologically accurate, the traditional methodology is still a useful diagnostic approach to diagnosing bleeding patients with congenital bleeding disorders. However, the new information helps to explain why it is often challenging to treat and monitor hypo- and hypercoagulable patients in a clinical situation, based on plasma assays alone, both with regard to progression of disease and the effectiveness of blood component and/or anticoagulation therapy. With the current knowledge that whole blood contains all the intravascular factors and cells participating in physiologic and pathologic hemostasis, incorporating TF and phospholipid-bearing cells, it is reasonable to assume that whole blood assays such as thromboelastography (TEG) may provide a more accurate reflection of *in vivo* hemostasis than the traditionally used plasma based hemostasis assays.

TEG is not a new method, but its potential use in assessing hemostatic disorders has resurfaced after the assay was automated and new activators were introduced, allowing for rapid and global assessment of hemostatic function in whole blood. More specifically, TEG evaluates all of the steps in hemostasis, including initiation, amplification, and propagation as well as fibrinolysis, including the interaction of platelets and leukocytes with the proteins of the coagulation cascade. Thus, TEG combines evaluation of the traditional plasma components of coagulation with the cellular components. Theoretically, all the TEG parameters are influenced by abnormal hemostasis; R and K values are increased and MA values are decreased in hypocoagulable states and opposite changes are observed in hypercoagulable states. Thus, TEG analysis should be able to distinguish pathologic from physiologic states and thus offers a welcome opportunity for point of care evaluation of overall hemostatic capability. TEG can potentially provide clinicians with the ability to

rapidly diagnose monitor and predict therapeutic response in veterinary patients with bleeding and/or thrombotic disorders. Consequently TEG could have a major impact on how management of such patients is approached in the future and potentially help advance and optimize the treatment of such patients significantly.

Blood transfusion results in adverse outcome – the scientific evidence

Prof. Dr. med. D. Spahn, University of Zurich, Switzerland

Hazard of Opioids in Acute and Chronic Pain Therapy

PD Dr. H. Rittner, Director, Pain Clinic, University Hospital Wuerzburg, Germany

Opioids are extensively used in acute as well as chronic pain therapy. Opioids are used postoperatively for acute pain therapy and in cancer pain as well as non-cancer pain for chronic pain therapy. In acute pain therapy the risks mainly include respiratory depression as well as sedation and nausea and vomiting. In chronic pain therapy, sedation and central nervous side effects are still common. These as well as nausea and vomiting are usually only a problem in the beginning. Addiction is always discussed but the clinical implications are limited if opioids are used with certain precautions. Most of the patients for acute and chronic opioid therapy need treatment for constipation as long as opioids are taken. Since the amount of opioids prescribed is constantly rising during the last years, other side effects of opioids came into focus. Effects of opioids on the hormonal balance seem to be restricted to the gonadotrophins. However the clinical implications so far are not very well studied. Opioid-induced immunosuppression has been demonstrated in cell culture experiments and in animal models. This is in striking contrast to the paucity of confirmatory studies in humans. In the lecture the major findings on opioid use and infectious complications in intensive care unit (ICU) patients, in patients with acute or chronic non-malignant pain, and in intravenous drug users (IDU) will be presented and discussed. Limitations of clinical studies include ethical concerns in randomized placebo-controlled trials (RCT) in acute postoperative pain and for a large part of ICU patients. In chronic pain treatment most studies only inadequately report infectious complications in relation to opioid use since their incidence is usually not considered to be drug related. Infectious complications in IDUs are very frequent but cannot easily be distinguished from risk behavior or risk environment. In summary small studies suggest that there are more risks in acute as well as chronic opioid use than previously considered, but sufficient RCT studies are still lacking. Nevertheless, opioids provide an excellent pain relief especially in acute as well as cancer pain with a relatively low risk potential compared to other drugs for pain therapy including non-steroidal anti-inflammatory drug or conanalgesics used in chronic pain therapy like antidepressants and anticonvulsants.

"Almighty God's Greatest Cure"

Prof. R. Eddie Clutton, The University of Edinburgh

Few medical drugs have received the same acclaim as the opiates¹: Paracelsus (1493-1541) called opium the 'stone of immortality' and mixed it with alcohol to produce "laudanum" [laudare - to praise]. Thomas Sydenham (1680) said, "Of all the remedies it has pleased almighty God to give man to relieve his suffering, none is so universal and so efficacious as opium". Later, Oliver Wendell Holmes (1809-94) proclaimed "Opium...the Creator himself seems to prescribe, for we often see the scarlet poppy, growing in the cornfields, as if it were foreseen that wherever there is hunger to be fed, there must also be pain to be soothed." William Osler (1849-1919) referred to it as 'God's own medicine'. Veterinarians have been less generous with praise although the long-standing mistrust of this (and other) analgesic classes appears to be waning – at least in the companion animal species. This parallels a burgeoning of information, an increased interest in animal pain, a broadening desire to treat it using new strategies and a recognition that euthanasia is an ideal form of analgesia.

These changes are readily discernible from the analysis of attitudinal studies: Hansen & Hardie 1993; Doohoo & Doohoo 1996; Doohoo & Doohoo 1998; Watson 1996; Capner 1999; Joubert 2001; Hewson 2001; Hugonard 2004; Williams 2005; Joubert 2006. The gender and age of respondents appear to be important in attitudinal changes, although geographical factors are confounded by time. Similar progress is being made in horses (Price 2012) cattle (Huxley 2006; Hewson 2007; Kilellend 2012) and laboratory animals (Richardson 2005; Stokes 2009; Coutlter 2009) albeit at a slower rate. Specism in opiate administration can be explained in a variety of ways

Horses

Despite well-established (Guinard 1899, Milks 1917), continuing and increasing (Mircuca, Clarke, Love) evidence to the contrary, the use of mu agonists continues to be condemned authoritatively in horses as a justification for investigating novel analgesics in this species. The case against opioids is based on their propensity to cause: excitation and box-walking; bizarre ingestive behaviour; adverse respiratory and gastro-intestinal effects combined with conflicting experimental evidence on their MAC-reducing and analgesic effects (Clutton 2010). Current reticence in the use of opioids in horses results from 1) poorly conducted

science including inadequate review of the non-English literature; 2) a failure to differentiate signs of pain and opioid side-effects; 3) inappropriate extrapolation from the human experience, 4) a scientific pre-occupation with testing adverse side-effects in small numbers of pain-free animals using limited experimental techniques lacking external validity; combined with a failure to recognize signs of pain in horses (Price)combined with an indifference to mild suffering (Green). CEPEF (Johnston) failed to implicate the use of opioids in equine peri-operative deaths although the role of opioids and opiates remains undecided.

Cats

Concerns with opiate use in *felidae* originated with Joel and Arndts (1925) who produced maniacal excitation after injecting 20 mg kg⁻¹. With time the benefits of opioids have been recognised in cats although reticence in its administration *cf* dogs persists.

Dogs

The use of opiates in dogs was considered by 18th century vivisectionists: Robert Hooke confided to Boyle after conducting a thoracotomy on a dog without anaesthetic, "...that he doubted there was an opiate strong enough to render the dog insensible under these conditions". Serturmer (1805) used morphine crystals in dog food to get rid of unwanted dogs and observed that morphine could evoke sleep and ultimately death in these animals. Since its introduction into veterinary practice side-effects attributed to morphine have included: excessive sedation; drug potentiation; euphoria; behaviour alteration (mania, dysphoria; hallucinations and rage reactions) cough suppression; intracranial hypertension; emetic effects, tolerance & dependence; respiratory depression; miosis / mydriasis; airway effects; adverse chest wall effects; adverse cardiovascular effects; adverse gastrointestinal (pancreatic, biliary, constipation) urinary retention; pruritus. Several of these are extrapolations from the human opiate literature and do not appear to be relevant in dogs. Other effects appear to become increasingly irrelevant as the requirement for analgesia increases. CEPSAF (Brodbelt) failed to implicate opiates in peri-anaesthetic death in small animals.

Human Beings

The clinical benefits of opiates in the clinical, particularly the palliative care setting, are marred more by their psychoactive rather than their physiological effects. Their long-term use being associated with tolerance and dependence. Constipation and sedation are also

regarded as undesirable features of prolonged administration. For these and other reasons, both acute, but particularly chronic pain are notoriously undertreated - at least in the USA. At higher levels of prescription, this results from concerns with prescribing drugs of interest to the DEA. At lower (nursing) levels, fear of side-effects including dependence are frequently cited.

Concerns with opiate side effects has engendered the proliferation of synthetic compounds, e.g., pethidine, methadone, buprenorphine, butorphanol, tramadolol, none of which have unthroned the "Queen of drugs" but remain popular in veterinary practice where opium has not found "universal efficacy" presumably because of unimportant selection criteria, misleading commercial promotion, misleading science, immunity from controlled drug legislation and a diminishing inability of some to recognize animal suffering and a disinclination to relieve it. Despite newly found (hyperalgesia; immunomodulation) and "traditional" side-effects morphine and (heroin) remain the most popular drugs for smoothing the avenues of death in the hospice environment.

Conclusion

Morphine remains the gold-standard against which other opioid drugs are judged. It remains the most commonly used drug in human palliative care. In medical practice, it has resisted repeated *coups d'etat* by synthetic and semi-synthetic usurpers. History does not record a butorphanol war, and Guerin Pierre Narcisse did not paint "Buprenorphine and Iris" (1811).

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Abstract Session Thursday

Room “Lobby”

Mechanical nociceptive thresholds using four different probe configurations in horses.

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Numerous mechanical stimuli have been used to measure mechanical nociceptive thresholds (MT). Since pressure = force/area, we evaluated the relationship between probe area and force readings of MT with different probes.

Eight horses (268 - 460 kg, 5 -10 years) were randomized in a 4-way crossover study. MT was measured using flat circular probe configurations (PC) of 1 mm (SHARP), 3.2 mm (BLUNT), one spring-mounted 1 mm pin that retained 1-2 N between tests (SPRING) and three 2.5 mm hemispherical pins mounted in an equilateral triangle (3P). A unit under remote control on the horse's back (Topcat Metrology Ltd) increased force at 1.2 N/sec in a pneumatic actuator on the dorsal surface of the metacarpus. Each horse was tested 5 times on each forelimb with each PC in random sequence, with 5 minutes between tests on one site. End point was a foot stamp. All tests were performed by one blinded operator. Mean MT was calculated for each horse for each PC, to generate a mean MT for each PC. Data were compared using Kruskal-Wallis ($P < 0.05$).

Mean $\pm$ SD MT were: SHARP 5.6 ± 2.3 , BLUNT 11.4 ± 3.4 , 3P 9.6 ± 4.6 , SPRING 6.4 ± 1.8 N. BLUNT was higher than SHARP and SPRING, but 3P was not different from any other. Larger contact area PCs produced higher MT than smaller PCs, but the relationship was not linear. BLUNT (area 7.7 mm^2) produced MT 2-fold higher than SHARP (area 0.75 mm^2) (area 10-fold greater). 3P (area 15 mm^2) produced more variable MT, less than 2-fold higher than SHARP (area 20-fold greater). SPRING did not affect the MT achieved by SHARP alone.

This demonstrates that PC has nonlinear effects on MT, probably related to probe profile and skin compression characteristics. It is important to define PC for measuring mechanical thresholds.

Approved by the UNESP Animal Experimentation Ethical Committee- protocol number 238/2011

Preliminary evaluation of a colo-rectal distension model for visceral nociceptive threshold testing in the donkey

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The donkey exhibits subtle signs of colic associated pain, consequently clinical assessment of colic pain is challenging. Visceral nociceptive threshold testing (VNTT) may mimic visceral distension that is common during colic, and could aid the assessment of analgesic management of colic pain. The study aim was to develop a minimally invasive VNTT model in donkeys.

Eight castrated male donkeys (105-160kg, 4-8 years) were studied. Following manual evacuation of faeces in rectum, the test balloon (latex (LB) or metallinised nylon (MNB)) was inserted, and connected to a pressure control system (MT1, Topcat Metrology). The balloon was inflated with air in a standardised manner, until a threshold response was observed or cut-off value (LB 500ml, MNB 46 mmHg) reached. Three data sets (1-2 per day, each comprising 4 tests at 15 intervals) were collected per animal for each balloon type, with LB evaluated first, followed by MNB.

Pressure remained constant during inflation of the LB due to its elasticity, therefore threshold volume was recorded; there was considerable variation in threshold volume between and within animals (253 ± 168 mL). The most frequent threshold response for the LB was tail swish (56% tests), followed by hind foot-lift, weight-shift, turning head, kicking, balloon expulsion, pinning back ears and front foot-lift. Due to the inelastic properties of the MNB, threshold pressure was recorded. Mean $\pm$ SD pressure was 32 ± 7 mmHg. Balloon expulsion was the most frequent threshold response for the MNB (53% tests), followed by turning head, leg shift, walking off, tail base lift, and hind foot-lift.

The two balloons produced different repertoires of threshold behaviours. The frequency of balloon expulsion with the MNB suggests that inflation produced an 'urge to defecate' rather than a response indicative of nociception. The utility of the LB was limited by the data variability. Neither balloon, placed rectally, was suitable for VNTT in the donkey.

Intravenous administration of tramadol has no antinociceptive effect in horses submitted to electrical and thermal stimuli

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Opioids may cause adverse effects in horses. This study evaluated the effect of tramadol on spontaneous locomotor activity (SLA) and thermal and electrical antinociception in horses.

Electrical stimulus was applied to one gelding horse and four mares (410 ± 25 kg) and thermal stimulus was applied to other three gelding horses and three mares (400 ± 15 kg). Both stimuli were applied to the shaved coronary region of the thoracic limb, according to Carregaro et al. (2007). The electrical and thermal hoof withdrawal reflex latencies (HWRL) were determined before and every 10 minutes for 60 and 90 minutes after IV administration of 2 and 3 mg.kg^{-1} of tramadol respectively. Head height, SLA, and behaviour were recorded for 180 minutes after 2, 3 or 5 mg.kg^{-1} of tramadol IV. Data were analysed by ANOVA followed by Tukey test ($P < 0.05$). SLA was measured by four pairs of orthogonally oriented infrared-photoelectric movement sensors in the stall.

Tramadol did not prolong the HWRL either to electrical (baseline 2.9 ± 0.4 ; maximal value at 50 min 3.6 ± 1.2 volts) or thermal (baseline and maximal value at 10 min 5.2 ± 2.3 seconds) stimuli. Head height and SLA also remained unchanged after tramadol. Horses receiving 3 and 5 mg.kg^{-1} of tramadol developed trembling in pectorals, triceps, and gluteus muscles and adopted a base-wide stance.

Tramadol neither induced sedation nor prolonged the HWRL after thermal or electrical stimuli in horses under experimental conditions and is unlikely that might be a useful analgesic under clinical painful conditions.

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Comparative study of a new metamizole formulation and carprofen on postoperative pain in dogs undergoing ovariohysterectomy

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The purpose of this study was to compare carprofen with a new oral formulation of metamizole in dogs undergoing ovariohysterectomy (OHE).

Twenty-three dogs were randomly assigned to one of two groups: Group M received metamizole (50 mg kg^{-1} PO) and group C received carprofen (4 mg kg^{-1} PO) one hour before anaesthetic induction and after 24 and 48 hours. Sedation was performed with medetomidine (0.005 mg kg^{-1}) and methadone (0.3 mg kg^{-1}) IM. Anaesthesia was induced with propofol and maintained with isoflurane and fentanyl. One blinded observer assessed postoperative sedation with a numerical score (0-3), and analgesia with a visual analogue scale, a dynamic interactive visual analogue scale, the Glasgow composite pain scale (CMPS), and a mechanical wound threshold device from 0.5 until 70 hours after the end of anaesthesia. Methadone (0.3 mg kg^{-1} IM) was administered if CMPS was $>6/24$ in ambulatory dogs, or $>5/20$ in non-ambulatory dogs. Venous blood samples were collected to quantify plasma levels of test drugs. Non-parametric Wilcoxon-Rank-Sum Tests (or Mann-Whitney U Tests) were used to assess the presence of differences in medians between the groups. $P < 0.05$ was considered to be significant.

Sedation scores were significantly higher at $T=0.5$ in group C (1.2 ± 1.03 versus 0.3 ± 0.48); CMPS scores were significantly higher at $T=0.5$ in group M (2.4 ± 1.51 versus 1.4 ± 3.17). Three dogs required rescue methadone (1/group M, 2/group C). Four dogs of group M vomited postoperatively. Carprofen and metamizole were well absorbed and median peak concentrations were reached within 12h(4-24) and 4h(4-16) for carprofen and metamizole respectively.

Both drugs provided adequate analgesia in dogs undergoing OHE. Duration of analgesia was found to be equal for both groups. Vomiting occurred in 40 % of dogs in group M.

The authors would like to thank Bayer Animal Health GmbH for sponsoring this study

Influence of dipyrone on minimal alveolar concentration (MAC) of sevoflurane and on thermal and mechanical threshold in beagle dogs

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The aim of the study was to evaluate possible MAC sparing and thermal and mechanical anti-nociception of dipyrone in dogs.

Two groups of 7 adult Beagle dogs (13.0 ± 3.2 kg; 18.2 ± 3.9 kg) were used in a randomised, blinded, controlled study design with a cross-over set up. After induction with 8 % sevoflurane in oxygen and endotracheal intubation a 1 hour stabilization period was allowed. Electrical stimulation was used for MAC determination. MAC was determined before 50 mg kg^{-1} dipyrone or placebo (NaCl 0.9 %) IV and after 1 hour and 4 hours. In a second trial, thermal (TT) and mechanical thresholds (MT) were determined before 50 mg kg^{-1} dipyrone or placebo and over 24 hours after treatment in awake dogs. Data were analyzed by analysis of variance for repeated measurements and paired t-test. Level of significance was 5 %.

Sevoflurane MAC was stable over 4 hours (2.79 vol%, 2.86 vol%, 2.88 vol%). No statistically significant change from baseline (2.74 vol%) occurred after dipyrone (2.78 vol% (1 hour); 2.79 vol% (4 hours)). Blood pressure and HR were not different between dipyrone and placebo. In the awake dogs MT and TT were not different between groups. At time point 75 min after dipyrone MT was significantly higher than baseline ($p < 0.001$). There were no more significant differences to baseline.

Dipyrone does not induce an anaesthetic sparing effect during sevoflurane anaesthesia in dogs. Acute cutaneous anti-nociception could not be detected after dipyrone

Comparison of analgesic efficacy of epidural methadone or ropivacaine/methadone with or without pre-operative oral tepoxalin in dogs undergoing tuberositas tibiae advancement surgery.

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The efficacy of 4 analgesia protocols in dogs undergoing tuberositas tibiae advancement was investigated in this randomised, blinded study.

Thirty-two client owned dogs (n=8 treatment⁻¹) received an oral placebo (PM and PRM) or tepoxalin (10 mg kg⁻¹) tablet (TM and TRM) once daily during 1 week before surgery. Epidural methadone (0.1 mg kg⁻¹) (PM and TM) or the epidural combination methadone (0.1 mg kg⁻¹)/ropivacaine 0.75% (1.65 mg kg⁻¹) (PRM and TRM) was administered after induction of anaesthesia. Intra-operative fentanyl requirements (2 µg kg⁻¹ IV) and end-tidal isoflurane concentration after 60 minutes of anaesthesia (FE'ISO₆₀) were recorded. Post-operative analgesia was evaluated hourly from 1-8 and at 20 hours post-extubation with a visual analogue scale (VAS) and the University of Melbourne Pain Scale (UMPS). If VAS>50 and/or UMPS>10, rescue methadone (0.1 mg kg⁻¹) was administered IV. Analgesic duration (time from epidural until post-operative rescue analgesia) and standing times were recorded. Normally distributed variables were analysed with a F-test (α=0.05) or t-test for pairwise inter-treatment comparisons (Bonferonni adjusted α=0.0083). Non-parametric data were analysed with the Kruskal-Wallis test (α=0.05 or Bonferonni adjusted α=0.005 for inter-treatment comparison of post-operative pain scores).

More intra-operative rescue analgesia interventions were required in PM [2 (0-11)][median (range)] and TM [2 (1-2)] compared to PRM (0) and TRM (0). FE'ISO₆₀ was significantly lower in (PRM+TRM) compared to (PM+TM). Analgesic duration was shorter in PM (459 ± 276 minutes) (mean ± SD) and TM (318 ± 152 minutes) compared to TRM (853 ± 288 minutes), but not to PRM (554 ± 234 minutes). Standing times were longer in the ropivacaine treatments compared to TM.

Inclusion of epidural ropivacaine resulted in reduction of FE'ISO₆₀, avoidance of intra-operative fentanyl administration, delayed and reduced need for post-operative rescue analgesia (if combined with tepoxalin) and delayed standing times. Tepoxalin improved post-operative analgesia in treatment TRM.

The effect of a lidocaine/bupivacaine mixture administered into the infraorbital canal in dogs

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This study aimed to determine onset and duration of effect of lidocaine/ bupivacaine (LB) administered into the infraorbital canal in dogs.

Six healthy adult intact female hound dogs weighing 21.3 ± 1.4 kg were used. The dogs were anesthetized with intravenous propofol and maintained with isoflurane in oxygen and ventilated to maintain a mean $PE\dot{V}CO_2$ between 3.6-4.7 kPa at normothermia (37.8 °C) in lateral recumbency. Insulated stimulating needles were inserted in the gingiva lateral to both maxillary canine teeth (C) and the right or left fourth premolar (P4) and second molar (M2) teeth (random allocation). Insulated needles were inserted to record the digastricus reflex (DR). The applied current was adjusted to achieve a maximum DR for each site. Three baseline recordings were made at 10-minute intervals. These values were averaged. Lidocaine (2%) and bupivacaine (0.5%) (0.5 mL of each solution) were then administered into the infraorbital canal using a 27-gauge 3.2 cm needle and aspirating before each injection. Recordings were made at 5, 10, 15, 30, 45 and 60 minutes, then every 20 minutes for up to 7 hours. The areas (duration x amplitude) of the DR for the unblocked canine tooth were used to normalize the results for the blocked side at each time. Anything <15% of baseline was defined as a successful block.

5/6 canine teeth were blocked by 5 minutes and the remaining one was blocked by 10 minutes. The average duration of the block for C, P4 and M2 was 277, 127 and 0 minutes, respectively. Only 3/6 P4 were blocked.

An infraorbital block with LB, injected deep into the canal, produced a long lasting block of the gingiva next to C but failed to consistently block P4 or M2. These data suggest that other approaches should be used for adequate analgesia during tooth extraction caudal to C.

The effect of meloxicam on renal function in dogs premedicated with medetomidine and anaesthetized with propofol and isoflurane

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This study aimed to investigate the effect of meloxicam on renal function in dogs undergoing anaesthesia and elective surgery.

Dogs received medetomidine 0.015 mg kg^{-1} IM. Anaesthesia was induced with propofol ($1\text{--}3 \text{ mg kg}^{-1}$) IV and maintained with isoflurane in 100% oxygen. Dogs were randomly allocated to receive intravenously meloxicam 0.2 mg kg^{-1} or distilled water 0.04 ml kg^{-1} . Blood and urine samples were collected before meloxicam or placebo administration (T1) and at the end of anaesthesia (T2). Mean T1 to T2 time was 57.5 ± 6.7 minutes in meloxicam and 62.3 ± 16.1 minutes in placebo groups. All dogs received methadone 0.3 mg kg^{-1} and atipamezole 0.025 mg kg^{-1} after T2, dogs in placebo group also received meloxicam. Lactated Ringer's was administered at $10 \text{ ml kg}^{-1} \text{ hour}^{-1}$ during anaesthesia. Urine pH, specific gravity (SG), gamma-glutamyltransferase to creatinine (γ -GT/crea), alkaline phosphatase to creatinine (AP/crea) and urinary protein to creatinine (UPC) ratio, fractional excretion of sodium (FE_{Na}), diuresis and glomerular filtration index (GFRI) were determined. Mann-Whitney test was used to compare groups and Wilcoxon test to compare timepoints within groups (significance $p < 0.05$).

Urinary parameters were similar in both groups at both T1 and T2 (Table 1). Diuresis (meloxicam 3.78 , placebo $3.54 \text{ ml kg}^{-1} \text{ hour}^{-1}$) increased above reference values and GFRI remained within them (meloxicam 2.64 , placebo $2.44 \text{ ml minute}^{-1} \text{ kg}$), however no significant difference between the groups was observed.

Meloxicam had no effect on kidney function in normotensive healthy dogs in this study.

Table 1: Median values for urinary parameters

	Meloxicam (T1)	Meloxicam (T2)	Placebo (T1)	Placebo (T2)
SG	1.039	1.014 *	1.034	1.010 *
FE_{Na} (%)	0.326	0.872 *	0.299	0.945 *
UPC	0.14	0.22 *	0.08	0.20 *
γ -GT/crea	2.48	2.74	1.44	3.40 *
AP/crea	0.010	0.014 *	0.013	0.087

* significant difference between T1 and T2

Development of a questionnaire to assess chronic pain associated with feline degenerative joint disease

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There are no validated assessment tools for feline degenerative joint disease (DJD)-associated pain. We hypothesized that an appropriately developed subjective owner-completed instrument to assess chronic feline DJD-associated pain (Feline Musculoskeletal Pain Index-FMPI) would prove reliable and have responsiveness and criterion validity.

45 client-owned cats were involved in this study – 20 normal, and 25 with DJD. The FMPI sensitivity and validity was explored through a double blinded, stratified, randomized, placebo-controlled crossover clinical study over a 10 week period in 25 cats. Meloxicam (0.1 mg/kg on day 1 followed by 0.05 mg/kg daily) was used to effect pain relief, and FMPI subjective data were compared to objective outcome measures (accelerometry). Reliability and repeatability were assessed using Cronbach's alpha and Intra-Class Correlation values respectively. A linear mixed model and backwards stepping regression were used to assess responsiveness and criterion validity. Critical p-value was 0.05.

Reliability (Cronbach's alpha 0.90 and 0.93 respectively) and repeatability were good (ICC values over 0.8) for normal and DJD affected cats. The FMPI showed positive responses for placebo and treatment; however no treatment effects were detected using a linear mixed model. There were no treatment effects on objectively measured activity. Using a backwards-stepping regression model, percent of meloxicam target dose administered, temperament, and total FMPI score at baseline were the covariates that had most effect on FMPI. Controlling for significant covariates, most positive effects (FMPI and accelerometry) were seen for the placebo treatment.

This instrument (FMPI) showed a good internal consistency, however neither responsiveness nor criterion validity were detected.

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Descriptive evaluation of responses from small animal practitioners in Switzerland to a survey on practice of perioperative analgesia

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A survey was performed to evaluate the use of peri-operative analgesia in dogs and cats by veterinary practitioners and their need in continuous education.

The questionnaire was sent electronically and by regular mail to more than 1000 practicing veterinarians. Questions were grouped in 7 sections recording personal data, education in veterinary analgesia, general ideology regarding treatment of peri-operative pain, personal experience, assessment, and use of main analgesics to treat peri-operative pain.

A total of 252 received forms were analyzed. An equal number of males and females (mean age 50.5 ± 9 and 40.2 ± 9 years old respectively) participated. Based on 5 questions, 81.3% showed an excellent (5/5), 15.5% very good (4/5), and 2.8% good (3/5) motivation for the use of peri-operative pain therapy. The main reasons for use of analgesics were to relieve the patient from pain (62.7%) and to contribute to the overall healing (34.1%). When presenting specific surgical procedures, most veterinarians did not evaluate resulting postoperative pain; Most veterinarians reported to routinely administer analgesics before (71-96%) or after (2-22%) surgery, while a minority never did (0-8%) or only in presence of pain symptoms (0-8%). Most used analgesics were non-steroidal anti-inflammatory drugs (carprofen, meloxicam, tolfenamic acid) and opioids (butorphanol, buprenorphine). The main reason for opioid choice was its analgesic efficacy. In most clinics (97.1%), animals are evaluated for pain after recovery. Only 39.3% of veterinarians use loco-regional anaesthesia from which only 7.4% report ring-block techniques, 5.6% epidurals and 4.8% specific regional nerve blocks. Forty five veterinarians reported postoperative use of ketamine constant rate infusion, 38 lidocaine, 45 oral tramadol and only 6 oral gabapentin.

Swiss veterinarians appear to well recognize the need for perioperative pain. However, they seemed to be unsure in evaluating pain severity, distinguishing between opioids classes, and using loco-regional anaesthesia techniques.

Abstract Session Thursday
Room “Speisesaal 3”

The effect of pure oxygen or air/oxygen mixture on arterial blood oxygenation in spontaneously ventilated, isoflurane anaesthetised horses: a retrospective study

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It has been shown that 100% oxygen administration can cause atelectasis during anaesthesia in man and different animal species, including horse. In this study, the effect of FiO_2 on PaO_2 during isoflurane anaesthesia in horses in a clinical setting was evaluated.

Two hundred anaesthetic records were selected from a 3-year period. One hundred cases received pure oxygen (group O) and another 100 received air/oxygen mixture (group M) as a carrier gas during spontaneous ventilation. Inclusion criteria were: age 2-15 years, weight 300-600 kg, ASA status 1-2, premedication with acepromazine ($20 \mu\text{g kg}^{-1}$), romifidine ($80 \mu\text{g kg}^{-1}$), and morphine (0.1 mg kg^{-1}), induction of anaesthesia with midazolam (0.06 mg kg^{-1}) and ketamine (2.2 mg kg^{-1}), maintenance with isoflurane, anaesthetic duration at least 20 min, non-abdominal surgery, spontaneous ventilation. Inspiratory oxygen fraction (FiO_2), PaO_2 , PaCO_2 , f_r , SAP, DAP, $\text{E}'\text{CO}_2$, and $\text{E}'\text{iso}$ were recorded. Areas under the curve were calculated for the above variables throughout anaesthesia and standardised (divided) by the total anaesthetic duration (AUCst). P(A-a)O_2 [according to the formula $\text{P(A-a)O}_2 = (\text{Pbar-PH}_2\text{O}) \times \text{FiO}_2 - \text{PaCO}_2/0.9 - \text{PaO}_2$] and $\text{PaO}_2/\text{FiO}_2$ were calculated. For statistical analysis t-test, Mann-Whitney test, and regression analysis were used.

The anaesthetic duration was 41.8 ± 14.2 min in group O and 39.3 ± 15.3 in group M. The mean $\pm$ s.d. AUCst FiO_2 was 0.784 ± 0.009 in group O and 0.604 ± 0.007 in group M (Mann-Whitney U test, $p < 0.0005$). The AUCst PaO_2 was 209 ± 101.8 mmHg in group O and 169 ± 73.2 mmHg in group M (Mann-Whitney U test, $p = 0.01$). The AUCst P(A-a)O_2 was 289 ± 96.3 mmHg in group O and 201 ± 76.9 mmHg in group M (t test, $p < 0.0005$). The AUCst $\text{PaO}_2/\text{FiO}_2$ was 264 ± 115.2 mmHg in group O and 280 ± 114.8 mmHg in group M (Mann-Whitney U test, $p = 0.337$). AUCst FiO_2 correlates significantly although weakly with AUCst P(A-a)O_2 ($p < 0.0005$, $r^2 = 0.276$).

According to the results of the present study, FiO_2 may have an effect on PaO_2 , although no clinical hypoxia was detected.

Influence of ketamine or xylazine constant rate infusion on quality of anaesthesia, cardiopulmonary function and recovery in isoflurane anaesthetized horses- a clinical trial

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With balanced anaesthesia, anaesthetic depth, cardiovascular function and recovery might be influenced differently by the drugs used.

Fifty-one horses undergoing elective surgery were randomly assigned to three treatment groups (each $n = 17$). After premedication with acepromazine, xylazine, butorphanol anaesthesia was induced with ketamine-midazolam and maintained with isoflurane alone (ISO), isoflurane with $1 \text{ mg kg}^{-1} \text{ hour}^{-1}$ ketamine (IK; stopped 20 minutes before end of anaesthesia) or $1 \text{ mg kg}^{-1} \text{ hour}^{-1}$ xylazine (IX). End-tidal isoflurane (ETISO) was adjusted according to a score by the same anaesthetist. Dobutamine was infused to maintain MAP $\geq 70 \text{ mmHg}$. Arterial blood gases, HR, fr, MAP, cardiac output were measured and cardiovascular indices calculated. Groups ISO and IK received xylazine before recovery. Recovery was scored from 11 (ideal) to 100 (worst). Data were analysed using one-way ANOVA and repeated measures ANOVA ($p < 0.05$).

Mean $\pm$ standard deviation averaged ETISO was significantly lower in IX (0.95 ± 0.07) and IK (0.97 ± 0.08) than in ISO (1.16 ± 0.13). Group IX had significantly higher averaged MAP (mmHg) (90 ± 13), in combination with lower HR, without dobutamine, than ISO (71 ± 7) and IK (76 ± 7), which required dobutamine at 0.53 and $0.36 \text{ } \mu\text{g kg}^{-1} \text{ minute}^{-1}$, respectively. Differences in other cardiopulmonary parameters did not reach statistical significance. Recovery score was 21 ± 6 , 26 ± 11 and 19 ± 9 in ISO, IK and IX, respectively.

Cardiopulmonary function could be maintained with each protocol. Xylazine results in pronounced reduction of anaesthetic requirements and blood pressure support.

A comparison of head/tail rope-assisted versus unassisted recoveries of horses after partial intravenous general anaesthesia

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The aim of this study was to evaluate the quality of recovery after partial intravenous general anaesthesia (PIVA) in horses, comparing head/tail ropes-assisted and unassisted recoveries.

Client owned adult horses (n = 777) undergoing general anaesthesia as part of elective, emergency or diagnostic procedures between 2004-2010 were used. The unassisted recoveries were evaluated retrospectively from archived protocols (n = 539), the head/tail ropes-assisted recoveries prospectively (n = 238).

General anaesthesia was maintained using isoflurane in 100% oxygen and an infusion of ketamine ($20 \mu\text{g kg}^{-1} \text{ minute}^{-1}$), midazolam ($0.3 \mu\text{g kg}^{-1} \text{ minute}^{-1}$), xylazine ($5 \mu\text{g kg}^{-1} \text{ minute}^{-1}$) until the end of anaesthesia, which typically results in an endtidal isoflurane concentration of 0.63 - 0.93%. During recovery all horses were scored by various observers (modified from Donaldson et al 2000), which includes the parameters duration of recovery, attempts to standing and recovery score defined by signs of ataxia and attitude of the horse. Descriptive statistics, ANOVA and multivariate covariant analysis were performed to evaluate influence of age, weight and duration of anaesthesia on the parameters. Data is given as [mean $\pm$ standard deviation (range)].

There was no significant difference in the number of attempts to stand unassisted [1.6 ± 1.1 (1-8)] and assisted [1.5 ± 0.7 (1-3)]. There was a significant difference in duration of recovery, unassisted patients recovering faster [27.6 ± 13.1 minutes (1-105)] than assisted [30 ± 14 minutes (5-102)] ($p=0.021$). There was no significant difference in recovery score between unassisted [13.1 ± 7.1 (7-41)] and assisted patients [14.1 ± 6.7 (7-35)].

A clear advantage of head/tail ropes-assisted over unassisted recoveries after PIVA could not be demonstrated.

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Effects of a constant rate infusion of dexmedetomidine on the minimum alveolar concentration in sevoflurane anaesthetized ponies

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The aim was to determine the effects of dexmedetomidine (DEX) constant rate infusion (CRI) on the minimum alveolar concentration (MAC) in sevoflurane anaesthetized ponies.

In a prospective, randomized, crossover, blinded, experimental study, 6 healthy ponies (mean 294 ± SD 51 kg, 12.7 ± 2.8 years) were included. Each pony was anaesthetized twice (3 week washout period). Anaesthesia was induced with sevoflurane in oxygen (via nasotracheal tube), the pony placed on the surgical table (T0) and anaesthesia maintained with sevoflurane in oxygen/air (FiO₂ = 55%). Ponies were randomly allocated to receive DEX 3.5 µg kg⁻¹ IV (T10-T15) followed by DEX CRI at 1.75 µg kg⁻¹ hour⁻¹ or an equivalent volume saline. After T60, MAC determination was initiated. Stimuli consisted of constant current electrical stimuli at the skin of the lateral pastern region. Triplicate MAC estimations were obtained and then averaged in each pony. Monitoring included pulse oximetry, ECG, anaesthetic gas monitoring and invasive arterial blood pressures. Arterial blood gases were determined every 20-30 minutes. Normocapnia was maintained by artificial ventilation in both treatments. Analysis of variance with treatment and period as fixed factors were used to detect differences between treatments (α = 0.05).

Sevoflurane MAC was decreased from 2.42 ± 0.55% to 1.07 ± 0.21% ($p < 0.001$) by the DEX CRI (53 ± 15% reduction). Heart rate (36 ± 3 *versus* 42 ± 5 beats minute⁻¹) ($p = 0.049$), SAP (104 ± 7 *versus* 141 ± 15 mmHg) ($p < 0.001$), MAP (83 ± 4 *versus* 114 ± 8 mmHg) ($p < 0.001$) and DAP (69 ± 4 *versus* 94 ± 6 mmHg) ($p < 0.001$) were significantly lower with DEX CRI. The PaO₂ was similar between treatments.

A DEX CRI at the reported dose reduces significantly the MAC of sevoflurane. Potential cardiovascular effects should be considered.

Measurements of transdiaphragmatic pressure in ponies during total intravenous and isoflurane anaesthesia.

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The aim of the study was to compare diaphragmatic contractility in TIVA and isoflurane anaesthetized ponies.

Six healthy ponies weighing 286 ± 60.9 kg, aged 12.7 ± 2.8 years were included in this randomized, experimental cross-over study. Following premedication with romifidine [$80 \mu\text{g kg}^{-1}$ intravenously (IV)], general anaesthesia was induced with midazolam (0.06 mg kg^{-1} IV) and ketamine (2.5 mg kg^{-1} IV) and maintained with either isoflurane in 60% oxygen ($\text{FE}'_{\text{Iso}} = 1.1\%$, treatment ISOFLURANE) or with a combination of romifidine ($120 \mu\text{g kg}^{-1} \text{ h}^{-1}$ IV), midazolam ($0.09 \text{ mg kg}^{-1} \text{ h}^{-1}$ IV) and ketamine ($3.3 \text{ mg kg}^{-1} \text{ h}^{-1}$ IV) (treatment TIVA) while breathing 60 % oxygen. The ponies were positioned in right lateral recumbency. One catheter was placed with the cuff in the distal oesophagus and one with the cuff in the stomach. To measure transdiaphragmatic pressure (TDP), the lumina of both cuffs were connected to a differential pressure transducer, which was zeroed before use. Position of the catheters was confirmed during inspiration (positive pressure in the stomach and negative in the oesophagus). TDP measurements were performed using a Mueller's manoeuvre, which consists of inspiration against a closed airway. The airway was closed by manually occluding proximal end of the endotracheal tube during inspiration every 5 minutes in 3 consecutive breaths, over a period of 60 minutes. Statistical analysis was performed using the Wilcoxon signed ranks test.

In 2 ponies from the TIVA group, the gastric catheter could not be positioned properly, so these ponies were excluded from the data analysis. TDP was higher during treatment ISOFLURANE (29.37 ± 1.00 mmHg) than during TIVA (14.57 ± 7.38 mmHg) (difference not significant; $P = 0.068$).

The tendency for greater diaphragmatic contractility in isoflurane anaesthetized ponies compared to TIVA anaesthetized ponies needs to be confirmed in more animals.

Measurement of transcranial bioimpedance in horses during general anaesthesia

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Prolonged periods in dorsal recumbency increase intracranial pressure in horses during general anaesthesia (Brosnan *et al* 2008). Transcranial bioimpedance (TCBI) quantified cerebral oedema in piglets (Lingwood *et al*, 2002). This study aimed to establish the effect of body position on TCBI changes in anaesthetized horses.

TCBI was measured in 16 (11 male and 5 female) horses undergoing general anaesthesia for surgery; after sedation with romifidine, anaesthesia was induced with ketamine and diazepam and maintained with sevoflurane vaporized in oxygen administered via a large animal anaesthetic machine (Tafonius, Vetronics). Flunixin and morphine were administered. Optimum and consistent electrode placement (caudal to the zygoma) in relation to the calvarium had been confirmed in cadavers using computerised tomography. An Impedimed SBF7 tetrapolar bioimpedance spectroscopy was used to measure TCBI before induction of anaesthesia (baseline), every 10 minutes during anaesthesia and in the initial recovery period.

The characteristic impedance at zero frequency was extrapolated using Cole-Cole analysis and plotted against time after induction. Impedance changed with duration of anaesthesia. Of 9 horses in dorsal recumbency, 7 showed increases in impedance over time, and 6 of 7 horses in other positions (left or right lateral) showed decreases in impedance over time. ANOVA revealed a non-significant relationship between change in TCBI over time and positioning ($p=0.08$). There was a statistically significant ($p<0.05$) relationship between changes in TCBI over time, and sex. However, 6 of the 9 horses in dorsal recumbency were stallions or geldings which may have confounded the result.

Transcranial impedance changes in anaesthetized horses follow a pattern suggesting ICP increases in anaesthetized horses are proportional to time. These changes may prove to be greater in animals in dorsal rather than lateral recumbency, and so warrant further investigation.

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Upregulation of intra-articular mu-opioid receptors in an acute equine synovitis model
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Mu-opioid receptors (MOR) have been demonstrated in equine synovial membranes (Sheehy et al. 2003).

This study assesses up-regulation (increase in number and density) of MOR in equine synovial membrane during acute inflammatory conditions. In a placebo-controlled cross-over design, lipopolysaccharide (LPS) was injected into middle carpal joint of seven healthy ponies. Arthroscopy and synovial membrane biopsy were performed under general anaesthesia at baseline (3 weeks before LPS injection), T48 and T672 hours after LPS injection, with ponies assigned to receive either phenylbutazone (PBZ 2.2 mg/kg PO BID for 7 days) or placebo (order of treatment randomised) from 2 hours post-LPS with butorphanol as rescue analgesia. Ponies were scored clinically for pain and lameness and the density and staining pattern of MOR in synovial membrane biopsies was evaluated using immunohistochemical techniques. Normally distributed data were tested using a linear mixed model for repeated measures, using univariate post-hoc tests with Bonferroni's correction. Categorical data were tested by means of Friedman and post-hoc Wilcoxon signed-rank tests.

LPS injection consistently induced a transient synovitis. Pain and lameness were significantly attenuated by treatment with PBZ ($p < 0.05$ at T8 and at T8 and T24 respectively). Up-regulation of MOR in the inflamed synovial membrane could be demonstrated in the placebo treated animals ($p < 0.05$). In PBZ-treated animals up-regulation was not seen ($p = 0.48$), but there was no significant difference at any time point between PBZ- and placebo-treated animals.

Up-regulation of MOR could indicate increased efficacy of intra-articular treatment with opioids during acute inflammatory conditions.

Reference

Sheehy, JG, Hellyer, PW, Sammonds, GE et al. (2001) Evaluation of opioid receptors in synovial membranes of horses *Am J Vet Res* 62, 1408-1412.

Lithium dilution and pulse contour analysis for cardiac output measurement in calves: a comparison with the thermodilution technique

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Sequential cardiac output measurements with lithium dilution ($\dot{Q}_{t_{LiDCO}}$) and pulse contour analysis ($\dot{Q}_{t_{PulseCO}}$) were compared to thermodilution measurements ($\dot{Q}_{t_{Thermo}}$) in calves.

Eight calves (2.5 - 8 months, 140 ± 51 kg) undergoing prolonged (120 - 630 minutes) anaesthesia for an intestinal loop study were used. After premedication (0.1 mg kg^{-1} midazolam and either $2 \text{ } \mu\text{g kg}^{-1}$ fentanyl (3 calves) or 0.1 mg kg^{-1} morphine (5 calves) intravenously), anaesthesia was induced with propofol ($2\text{-}4 \text{ mg kg}^{-1}$ intravenously) and maintained with isoflurane in oxygen/air (inspiratory O_2 fraction 55%) and either a fentanyl ($0.1 \text{ } \mu\text{g kg}^{-1} \text{ minute}^{-1}$) or morphine ($0.1 \text{ mg kg}^{-1} \text{ hour}^{-1}$) infusion respectively. Besides routine monitoring and cardiovascular support (fluids and dobutamine to maintain MAP above 70 mmHg), $\dot{Q}_t$ was measured using the 3 techniques at 30 minute intervals (a maximum of 10 measurements per calf). Thermodilution measurements (Swan-Ganz catheter, iced saline) were performed in triplicate; values which deviated $>10\%$ from the median value were rejected. The PulseCO software was calibrated using the first triplicate thermodilution measurement; calibration was not repeated thereafter. For the LiDCO technique, lithium chloride ($4.5 \text{ } \mu\text{mol kg}^{-1}$) was injected through the proximal port of the Swan-Ganz catheter. The overall means of the 3 techniques were compared using t-tests. The effects of time, body temperature and different cardiovascular variables on these differences were analyzed using mixed model ANOVA.

Overall, $\dot{Q}_{t_{LiDCO}}$ and $\dot{Q}_{t_{PulseCO}}$ were respectively (mean $\pm$ SD) $9.9 \pm 24.3\%$ and $11.1 \pm 24.1\%$ higher than $\dot{Q}_{t_{Thermo}}$ ($P < 0.01$). Although $\dot{Q}_{t_{Thermo}}$ remained quite stable, the difference with the other techniques increased significantly over time. The difference between $\dot{Q}_{t_{PulseCO}}$ and $\dot{Q}_{t_{Thermo}}$ was significantly influenced by SAP, DAP, MAP, packed cell volume and body temperature.

The LiDCO and PulseCO techniques are not interchangeable with the thermodilution technique for cardiac output measurement in calves.

The influence of certain drugs on the response characteristics of the lithium sensor used in the LiDCOplus Hemodynamic Monitor

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The study aim was to examine the effect of certain drugs on the function of the LiDCO sensor.

Test drugs and lithium were dissolved in saline to produce the following solutions: saline, saline-lithium, saline-drug, and saline-drug-lithium. The drug concentrations were overlapping the range of clinical interest as estimated from published literature. These 38°C solutions were pumped through the LiDCO sensor in predetermined order. Sensor voltages were measured using the LiDCOplus Hemodynamic Monitor. Lithium and drug-induced voltage changes were determined ($n = 5$). Differences between lithium-induced voltage changes in the absence and presence of drugs indicated erroneous lithium detections (paired t-test, $p < 0.05$). The resulting biases in lithium detection were translated to biases in LiDCO measurements using a published formula (Linton et al. 1993).

Clonidine, detomidine, dexmedetomidine, medetomidine, romifidine, xylazine, ketamine, s-ketamine, lidocaine, and rocuronium caused concentration dependent increases in baseline sensor voltages and negative biases in lithium detection which were mathematically equivalent to $> +10\%$ bias in LiDCO measurements. The drug-induced changes in baseline sensor voltages correlated with predicted biases in LiDCO measurements ($r^2 = 0.91$). Atipamezole, acepromazine, butorphanol, diazepam, midazolam and guaifenesin caused minimal or no interaction with the sensor ($< +10\%$ bias in LiDCO).

Based on these findings and published pharmacokinetic data xylazine, ketamine, lidocaine and rocuronium treatments may cause clinically relevant biases in LiDCO. These findings need to be confirmed in vivo. Relevant (>3 mV) changes in baseline sensor voltages due to the presence of drugs may indicate possible interactions with the LiDCO sensor.

Reference

Linton RA, Band DM, Haire KM (1993) A new method of measuring cardiac output in man using lithium dilution. *Br J Anaesth* 71, 262-266.

The authors thank the LiDCO Ltd. for supporting the study and Mr. Eric Mills and Dr. David Band for personal communication.

Pharmacokinetics and pharmacodynamics of a 5 $\mu\text{g kg}^{-1} \text{ hour}^{-1}$ fentanyl infusion in conscious cats

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The pharmacokinetics and pharmacodynamics of fentanyl in cats administered as a constant rate infusion have not yet been described.

Seven healthy adult cats (five castrated males, 2 females, 3.7-5.2kg) were studied using a randomized, blinded crossover design with eight days between treatments. Cats were anesthetized with sevoflurane in oxygen for placement of a jugular and contralateral cephalic catheter. Thorax and lower thoracic limbs of each cat were shaved for thermal (TT) and mechanical threshold (MT) testing respectively. The following day, treatments were either 5 $\mu\text{g kg}^{-1}$ fentanyl followed by 5 $\mu\text{g kg}^{-1} \text{ h}^{-1}$ fentanyl infusion (Group F) for two hours or an equivalent volume of 0.9% saline solution (Group S: Control) given as loading dose (LD) and infusion. Sedation scores, MT, TT and blood samples were obtained prior to drug treatment (baseline), 0.25, 0.75, 1, 1.5, 2, 2.25, 2.5 2.75, 3, 4, 6, 8, 10, 14 and 26 hours after LD administration. Data were analyzed by repeated measures ANOVA with correction for multiple comparisons. Probability values < 0.05 were deemed significant.

Fentanyl induced mild sedation during the infusion. One cat salivated profusely after fentanyl LD, no other adverse effects were observed. For the duration of the infusion TT and MT were significantly different between groups, except for TT one hour after LD administration. Thermal thresholds were significantly higher than baseline ($44.2 \pm 0.3^\circ\text{C}$) between 0.25 (peak: $50.0 \pm 2.9^\circ\text{C}$) and 0.75 hours. MT were significantly higher than baseline ($5.3 \pm 0.8 \text{ N}$) between 0.25 (peak: $11.8 \pm 2.9 \text{ N}$) and 1 hours. During the two hour infusion, fentanyl plasma concentrations decreased from 4.0 ± 1.5 to $2.7 \pm 1.1 \text{ ng ml}^{-1}$ and were associated with antinociception. Fentanyl plasma concentrations less than $1.3 \pm 0.2 \text{ ng ml}^{-1}$ (2.25 hours after LD) were not associated with antinociception.

Results indicate a fentanyl infusion (5 $\mu\text{g kg}^{-1} \text{ h}^{-1}$) may provide mechanical and thermal antinociception for this infusion period.

Abstract Session Friday
Room “Lobby”

Non-invasive continuous positive airway pressure delivered using a paediatric helmet in dogs recovering from general anaesthesia: a feasibility study

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Non-invasive Continuous Positive Airway Pressure (CPAP) represents an effective ventilatory support for people recovering from anaesthesia and during intensive care (Squadrone et al. 2005). The aim of this study was to evaluate the feasibility of CPAP administered with a helmet in healthy dogs.

Fifteen healthy female dogs recovering from general anaesthesia after elective ovariohysterectomy were enrolled in this study. All dogs received the same standardized anaesthetic protocol (acepromazine, morphine, propofol and isoflurane in oxygen). After extubation in all dogs, the head was placed in a paediatric helmet connected to a venturi valve supplied with medical air. In all patients the air flow was set to 10 L minute⁻¹ in order to deliver a total flow of 50 L minute⁻¹. Dogs received the following sequence of treatments: zero CPAP (NOCPAP1), CPAP of 5 cmH₂O (CPAP) and again zero CPAP (NOCPAP2). Each treatment phase lasted 15 minutes and at the end of each phase an arterial blood sample was withdrawn. Throughout the study the following data were collected: pressure and FiO₂ inside the helmet, MAP, *f_r*, HR, sedation score (0 = awake, 10 = deep sedation) and tolerance to the helmet (0 = excellent, 4 = poor).

The *f_r* (10.5 ± 1.4 breaths minute⁻¹) and PaCO₂ (5.2 ± 0.3 kPa) were significantly lower at CPAP compared to NOCPAP1 (16.0 ± 2.3 breaths minute⁻¹ and 7.3 ± 0.5 kPa) and NOCPAP2 (15.0 ± 1.6 breaths minute⁻¹ and 6.3 ± 0.5 kPa). The PaO₂ was higher at CPAP (14.0 ± 0.5 kPa) compared to NOCPAP1 (10.7 ± 0.9 kPa) and NOCPAP2 (11.5 ± 0.7 kPa). Tolerance and sedation scores were similar and adequate at all study times.

The results of this study demonstrated that non-invasive CPAP applied using a helmet is a feasible and effective treatment in dogs recovering from general anaesthesia.

Reference

Squadrone V, Coha M, Cerutti E et al. (2005) Continuous positive airway pressure for treatment of postoperative hypoxemia: a randomized controlled trial. JAMA 293, 589-595.

Comparison of the effects of propofol or alfaxalone for anaesthesia induction and maintenance on respiration in cats

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This study was conducted to compare the effects of propofol and alfaxalone on respiration in cats.

Twenty cats undergoing ovariohysterectomy, premedicated with medetomidine $10 \mu\text{g kg}^{-1}$ IM and meloxicam 0.3 mg kg^{-1} SC were randomly assigned to two groups: group A ($n=10$) receiving alfaxalone $5 \text{ mg kg}^{-1}\text{minute}^{-1}$ and $10 \text{ mg kg}^{-1} \text{ hour}^{-1}$ IV for induction and maintenance of anaesthesia; group P ($n=10$) received propofol $6 \text{ mg kg}^{-1}\text{minute}^{-1}$ and $12 \text{ mg kg}^{-1} \text{ hour}^{-1}$ IV for induction and maintenance of anaesthesia. After endotracheal intubation the tube was connected to a non-rebreathing system delivering oxygen 100%. The rate of the anaesthetic drug was adjusted in both groups ($\pm 0.5 \text{ mg kg}^{-1} \text{ hour}^{-1}$) every 5 minutes according to a predefined scoring sheet including HR, MAP and clinical signs. If apnea for more than 30 seconds, $\text{PE}'\text{CO}_2 > 7.3 \text{ kPa}$ or hemoglobin saturation (SpO_2) $< 90\%$ occurred, ventilation was controlled by manual ventilation. Methadone 0.1 mg kg^{-1} was administered postoperatively IM.

Data were analyzed using Fisher exact test with $p \leq 0.05$ for significance.

Two and 8 of the cats in group A and P had to be ventilated. The difference between group A and P was significant ($p=0.02$). Two vs 2, 0 vs 4 and 0 vs 6 cats in group A vs P showed apnea ($p=1$), $\text{PE}'\text{CO}_2 > 7.3 \text{ kPa}$ ($p=0.08$) or $\text{SpO}_2 < 90\%$ ($p=0.01$). The mean $\pm$ SD anaesthesia induction dose was 11.6 ± 0.3 and $11.7 \pm 2.7 \text{ mg/kg}$ (administered to effect until tracheal intubation was possible) and anaesthesia maintenance dose was 10.7 ± 0.8 and $12.4 \pm 0.5 \text{ mg kg}^{-1} \text{ hour}^{-1}$ for alfaxalone and propofol, respectively.

We conclude that alfaxalone has less influence on respiration than propofol in cats. Alfaxalone might be the preferable drug to induce and maintain anaesthesia in cats when no possibility for controlled ventilation is available.

Combining dexmedetomidine, ketamine, and butorphanol for sedation in dogs: A new optimization method

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A dose-finding study was conducted to obtain the optimal combination of dexmedetomidine, ketamine, and butorphanol administered intramuscularly to healthy dogs for minor therapeutic or diagnostic procedures.

Eighty dogs were tested in a blinded, randomized, prospective clinical trial. First, eight clinically relevant combinations were tested in 50 dogs. The quality of each combination was rated using a purposefully developed negative score (NS; 0-23.5), to judge the quality of sedation, side effects, and need for additional anaesthetics. Descriptive statistics was performed to compare the combinations. Then the combinations were divided into “promising” and “unsatisfactory” subgroups and their centroids P_c and U_c were determined before a new combination (N) was calculated using the formula $N = P_c + 1.3 * (P_c - U_c)$. The combination N was tested in six dogs and then it replaced the worst of the previous eight combinations. The same procedure was repeated after each new combination until no further improvement was achieved (Berenbaum 1990; Sveticic et al. 2003).

The optimal combination found was dexmedetomidine $5 \mu\text{g kg}^{-1}$, ketamine 1 mg kg^{-1} , and butorphanol 0.3 mg kg^{-1} with a median NS of 1.5 (interquartile range 1.5-2.4). In all twelve dogs receiving this combination, the quality of sedation was satisfactory as none of the dogs required additional anaesthetics within the first 20 minutes of the procedure or showed severe side effects.

The application of this optimization method allowed the calculation of an optimal drug combination for sedation in healthy dogs. This combination can now be used in a larger clinical trial to support its clinical utility.

References

- Berenbaum MC (1990) Direct search methods in the optimisation of cancer chemotherapy regimens. *Br J Cancer* 61, 101-109
- Sveticic G, Gentilini A, Eichenberger U et al. (2003) Combinations of Morphine with Ketamine for Patient-controlled Analgesia: A New Optimization Method. *Anesthesiology* 98, 1195-1205

Acknowledgment: This project was partially funded by the Veterinary Services of the Swiss Army.

The effect of dilution of alfaxalone on induction dose requirement in dogs

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A study assessing alfaxalone in cats suggested dilution of alfaxalone reduced the induction dose (Zaki et al. 2009). We compared dose rates required for induction of anaesthesia in dogs using undiluted and diluted alfaxalone.

Following a sample size calculation, sixty-one dogs were block randomised according to weight (0-10, 10-20, 20-30 kg) to receive alfaxalone undiluted or diluted to 5 mg mL⁻¹ with water for injection. Each group was subdivided to receive alfaxalone intravenous infusion at 0.5 or 1 mg kg⁻¹ minute⁻¹. Acepromazine 0.03 mg kg⁻¹ and buprenorphine 0.02 mg kg⁻¹ were administered IM and an intravenous catheter placed. The IV infusion of alfaxalone was started. Once jaw tone was lost and the tongue could be protracted for placement of a laryngoscope, the infusion was stopped. Endotracheal intubation was attempted. If reaction occurred the infusion was restarted until endotracheal intubation was possible. Subjective scores were assigned for temperament, body condition score (BCS), sedation, induction quality, ease of intubation and transition to maintenance anaesthesia by the same observer unaware of treatment (1 representing the best score possible). Data were pooled, tested for normality and groups compared using t tests or Mann Whitney with $p < 0.05$ considered significant.

Groups were similar for weight, age, BCS, pre-anaesthetic medication to induction times and scores for temperament, sedation, induction, intubation. Transition to maintenance scores (1 – 3) were higher in the diluted group 1 (1 – 3) compared to undiluted 1 (1 – 2) ($p = 0.01$). Median dose was lower in the diluted 1.05 (0.55 – 2.07) compared to the undiluted group 1.3 (0.76 – 3.4 mg kg⁻¹) ($p = 0.0029$). One dog (diluted group) displayed excitement; five (diluted group) and one (undiluted group) showed muscle twitching.

Dilution of alfaxalone resulted in a reduction in dose required for induction of anaesthesia when infusion rate was controlled.

Reference

Zaki S., Ticehurst K.E. and Miyaki Y. (2009) Clinical evaluation of Alfaxan-CD® as an intravenous anaesthetic in young cats. *Aust. Vet. Journal* 87, 82-87.

The effect of butorphanol on the incidence of dexmedetomidine-induced emesis in the cat.

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Butorphanol is reported to prevent emesis in dogs when administered before treatment with cisplatin (Moore et al. 1994). The objective of this study was to determine butorphanol's anti-emetic effect when co-administered with dexmedetomidine in cats.

Seven male and 7 female domestic shorthair cats, aged 14-84 months (median, 78 months) and weighing 1.7-5.5 kg were used in a randomised, blinded, controlled, crossover study. Each cat received intramuscularly five different treatment protocols: A (dexmedetomidine 25 µg kg⁻¹); B (dexmedetomidine 20 µg kg⁻¹ plus butorphanol 0.2 mg kg⁻¹); C (dexmedetomidine 20 µg kg⁻¹ plus butorphanol 0.1 mg kg⁻¹); D (dexmedetomidine 25 µg kg⁻¹ plus butorphanol 0.2 mg kg⁻¹) and E (dexmedetomidine 20 µg kg⁻¹). Episodes of emesis, signs of nausea (non-productive retching, salivation and licking) and time to lateral recumbency were recorded, while sedation was scored subjectively 8 minutes after treatment. The Friedman test and the Cochran's Q-test were used to analyse data. Significance was evaluated at the 5% level.

Time to lie down and sedation scores did not differ significantly between the groups. Cats in groups B, C and D had a significantly lower incidence of emesis (0/14, 0/14, and 1/14, respectively) compared with cats in groups A (10/14) and E (11/14). The incidence of cats with nausea was significantly higher for animals in groups A (10/14) and E (12/14) compared to animals in groups B (4/14) and D (3/14).

Butorphanol co-administered with dexmedetomidine significantly reduced the incidence of emesis induced by dexmedetomidine in the cats of this study.

Reference

Moore AS, Rand WM, Berg J et al. (1994) Evaluation of butorphanol and cypiroheptadine for prevention of cisplatin-induced vomiting in dogs. J Am Vet Med Assoc 205, 441-443.

The Effect of Age and Body Mass on Responses to Anaesthesia in Pigs

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The study's objective was to assess the interaction between age, body mass and the effects of an anaesthetic drug combination in pigs.

Thirty-six male pigs were anaesthetized on three occasions (S1-3) for CT scanning, aged 105, 137 and 166 days respectively. The pigs' mean ($\pm$ SD) body mass (kg) at S1-3 were: 57.2 ± 4.4 , 88.4 ± 6.2 and 114.7 ± 7.6 . Medetomidine ($5 \mu\text{g kg}^{-1}$), azaperone (1 mg kg^{-1}) ketamine (5 mg kg^{-1}) and midazolam (0.25 mg kg^{-1}) were combined in the same syringe and injected intramuscularly. If venous cannulation was not possible after 5 minutes, 2-3% isoflurane in oxygen and N_2O was delivered by mask until it was, then stopped. If anaesthetic depth was inadequate for CT scanning, a full dose (midazolam 0.25 mg kg^{-1} , ketamine 2 mg kg^{-1}) or half dose was administered intravenously. The relationship between mass and the time in minutes from sedation to four end-points (Table 1) were assessed using regression analysis and linear mixed effect models (LME). LMEs were used to assess the effects of isoflurane and intravenous anaesthetics on these end-points.

On each occasion, there was no significant relationship between mass and the five end-points. Isoflurane significantly influenced the time the animals took to move, attempt to stand and finally stand following sedation ($P < 0.037$); intravenous agents had no effect on the time to the first attempt to stand or when finally standing ($P > 0.585$).

Body mass did not significantly influence the response of pigs to the anaesthetic described.

	S1	S2	S3
Induction: first recumbency	2.94 ± 1.57	3.64 ± 3.65	3.98 ± 1.70
Recovery: first movement	57.53 ± 19.75	96.29 ± 57.99	53.97 ± 22.02
first attempt to stand	112.00 ± 38.53	162.29 ± 51.00	137.81 ± 52.14
finally standing on four legs	161.75 ± 46.64	207.23 ± 47.39	182.50 ± 60.30

Table 1. The time (minutes) from sedation to each endpoint is given as mean $\pm$ SD.

Abstract Session Friday

Room “Speisesaal 3“

Cardiorespiratory, anaesthetic and analgesic effects of two doses of morphine combined with medetomidine and alphaxolone in rabbits

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Various medetomidine combinations have been studied in rabbits, however medetomidine, morphine and alphaxalone anaesthesia has not been evaluated.

Ten New Zealand white rabbits were anaesthetised twice, receiving medetomidine 200 µg kg⁻¹ with 1 mg kg⁻¹ (MOR1) or 2 mg kg⁻¹ (MOR2) morphine IM. After preoxygenation, anaesthesia was induced with alphaxalone (dose determined following pilot work, 10 mg kg⁻¹ IV). The trachea was intubated and 60% oxygen provided. Measurements of Hr, fr, MAP, arterial pH, PaO₂ (KPa), PaCO₂ (KPa) and SaO₂ were taken at baseline, after premedication and a mean calculated of values taken every 5 minutes for 90 minutes after induction. Duration of surgical anaesthesia (defined as absent ear-pinch and toe-pinch reflexes) was recorded. Data were compared with t-tests.

Compared to baseline, HR (MOR1: 236 ± 16 vs 162 ± 45; MOR2: 224 ± 15 vs 151 ± 31), MAP (MOR1: 92 ± 10 vs 60 ± 20; MOR2: 89 ± 10 vs 66 ± 11), fr (MOR1: 224 ± 28 vs 85 ± 49; MOR2: 240 ± 47 vs 72 ± 55) and SaO₂ (MOR1: 97.5 ± 0.6 vs 89.1 ± 4.8; MOR2: 97.3 ± 1.2 vs 86.3 ± 1.1), decreased after premedication, (fr significantly different between groups). Postinduction apnoea of 22 ± 5 (MOR1) and 44 ± 5 minutes (MOR2) was observed. Manual ventilation was instigated until spontaneous ventilation resumed. Mean pH during anaesthesia was lower than baseline (MOR1: 7.41 ± 0.47 vs 7.24 ± 0.04; MOR2: 7.37 ± 0.52 vs 7.23 ± 0.05) PaCO₂ increased from baseline during anaesthesia (MOR1: 4.1 ± 0.2 vs 8.2 ± 1.3; MOR2: 3.8 ± 0.5 vs 8.4 ± 1.5 kPa). Surgical anaesthesia lasted longer with MOR2.

These doses of morphine with medetomidine and alphaxalone in rabbits produced surgical anaesthesia, but with profound cardiorespiratory depression.

Ketamine decreases the cerebral binding of the selective serotonin-2A receptor radioligand ^{123}I -R91150 in cats.

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Antagonism at the postsynaptic serotonin (5-HT_{2A}) receptors could explain the antidepressant effects of ketamine (Diazgranados et al., 2010). This effect was studied using micro-single photon emission computed tomography.

Six six-year-old cats were used in this study. The radioligand ^{123}I -R91150, a 5-HT_{2A} receptor antagonist, was injected IV. Anaesthesia was induced and maintained with a continuous rate infusion (CRI) of propofol ($8.4 \pm 1.2 \text{ mg kg}^{-1}$ followed by $0.22 \text{ mg kg}^{-1} \text{ minute}^{-1}$) 75 minutes after tracer administration and the first acquisition was started 15 minutes after induction of anaesthesia. After this acquisition propofol ($0.22 \text{ mg kg}^{-1} \text{ minute}^{-1}$) was combined with ketamine (5 mg kg^{-1} followed by $0.023 \text{ mg kg}^{-1} \text{ minute}^{-1}$), the second acquisition started 15 minutes later. Semiquantification, (with the cerebellum, a region devoid of 5-HT_{2A} receptors, as a reference region) was performed to calculate the 5-HT_{2A} receptor binding indices (parameter for available receptor density) in the frontal and temporal cortex. The binding indices were analyzed with paired t-tests.

The addition of ketamine resulted in decreased binding indices in the right frontal (1.25 ± 0.22 compared to 1.45 ± 0.16 , $p = 0.004$), left frontal (1.34 ± 0.15 compared to 1.49 ± 0.10 , $p = 0.019$), right temporal (1.30 ± 0.17 compared to 1.45 ± 0.09 , $p = 0.019$) and left temporal cortex (1.41 ± 0.20 compared to 1.52 ± 0.20 , $p = 0.032$).

This study shows that alterations of the 5-HT_{2A} receptor status can be studied with ^{123}I -R91150 micro-single photon emission computed tomography in cats. Furthermore an interaction between ketamine and the 5-HT_{2A} receptors resulting in a decreased binding of ^{123}I -R91150 in the frontal and temporal cortex was demonstrated. Whether the decreased radioligand binding resulted from a direct competition between ketamine and ^{123}I -R91150, or from a decreased affinity of the 5-HT_{2A} receptor caused by ketamine, remains to be elucidated.

Reference

Diazgranados N, Ibrahim L, Brutsche NE, et al. (2010) A randomized add-on trial of an N-methyl-D-aspartate antagonist in treatment-resistant bipolar depression. *Arch Gen Psychiatry* 67(8),793-802.

The influence of chronic morphine on the canine cerebral 5-HT_{2A} receptor availability measured with SPECT

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A major drawback of opioid therapy is tolerance development. Drugs acting on the serotonergic (5-HT) system have been studied to prevent this problem. This study examines the effect of chronic opioids on cerebral 5-HT_{2A} receptors.

Brain 5-HT_{2A} receptor availability was estimated in seven healthy five-year-old female neutered Beagle dogs pre- and post- chronic morphine treatment (oral sustained release morphine 20 mg twice daily for ten days) with ¹²³I-5-I-R-91150 (a 5-HT_{2A} selective radioligand) and Single Photon Emission Computed Tomography (SPECT). Before and on the last day of morphine treatment, SPECT scans were performed 90 minutes after ¹²³I-5-I-R-91150 injection. Dogs were premedicated with dexmedetomidine (375 µg m⁻² body surface area, IM). Anesthesia was induced with propofol (2.80 ± 0.63 mg kg⁻¹ IV) and maintained with isoflurane in oxygen. 5-HT_{2A} receptor binding indices (BI) for the frontal, parietal, temporal and occipital cortex and the subcortical region were calculated by semiquantification with the cerebellum (devoid of 5-HT_{2A} receptors) as a reference region. Data were analyzed using a mixed-model with treatment as a fixed effect and dog as a random effect.

Chronic morphine treatment significantly ($p \leq 0.05$) lowered 5-HT_{2A} BI's in the right and left frontal cortex, the right and left temporal cortex, the right and left parietal cortex, and the subcortical region.

The decreased cerebral 5-HT_{2A} receptor availability following chronic morphine treatment suggests an interaction between the opioid and serotonergic system, the exact nature of this interaction remains to be elucidated.

Acknowledgement

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Effect of Dexmedetomidine versus Acepromazine-Methadone premedication on limb-to-lung circulation time in dogs

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The study compares limb-to-lung circulation times in dexmedetomidine or acepromazine-methadone premedicated dogs.

Twenty client owned ASA 1 and 2 dogs from both genders were randomly assigned to receive dexmedetomidine 0.01 mg kg^{-1} IM or acepromazine 0.04 mg kg^{-1} and methadone 0.2 mg kg^{-1} IM. Anaesthesia was induced with propofol and maintained with isoflurane, adjusted to achieve similar anaesthetic depth in all patients. Mechanical ventilation was started immediately ($20 \text{ breaths min}^{-1}$, inspiratory to expiratory ratio 1:2) and V_t adjusted to achieve $PE'CO_2$ $3.9\text{-}5.3 \text{ kPa}$. After ten minutes arterial blood gas analysis was performed and 5 minutes later a single dose of sodium bicarbonate 0.5 mEq kg^{-1} was administered intravenously over 10 seconds, starting the injection at the start of inspiration. The time interval between the start of bicarbonate injection and the recording of the highest $PE'CO_2$ was measured and defined as limb-to-lung circulation time. Age, body weight, HR, MAP, $FE'iso$, pH, $PaCO_2$, bicarbonate concentration and circulation time were compared between groups with Student's t-test ($p < 0.05$).

Increase in $PE'CO_2$ was less in dexmedetomidine group ($1.7 \pm 0.2 \text{ kPa}$ vs. $1.9 \pm 0.2 \text{ kPa}$). Circulation time was longer in the dexmedetomidine group (27 ± 5.1 versus 20 ± 2.3 seconds). Body weight was lower in the dexmedetomidine group ($23.3 \pm 6.8 \text{ kg}$ versus 30.6 ± 3.9). Mean arterial blood pressure was higher in the dexmedetomidine group (92 ± 9 vs. $73 \pm 7 \text{ mmHg}$).

The slower circulation time after dexmedetomidine premedication needs to be considered when administering fast acting drugs to effect e.g. for induction of anaesthesia.

Effects of midazolam on quantitative electroencephalographic (EEG) parameters in light and deep alfaxalone anaesthesia in cats

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The EEG allows characterization of drug effects on the central nervous system (CNS). The hypothesis was tested whether the administration of midazolam during alfaxalone anaesthesia has a paradoxical stimulating effect on CNS activity in cats.

Seven healthy, adult domestic shorthair cats (4.1 ± 1.2 kg) were anaesthetized twice in a randomized, experimental trial with a wash-out period of eight days. Alfaxalone was given by target controlled infusion (Graseby3500, CCIP software) aiming at $4 \mu\text{g ml}^{-1}$ for induction, followed by $3 \mu\text{g ml}^{-1}$ (groupL) or $6 \mu\text{g ml}^{-1}$ (groupD) for maintenance. The EEG was recorded continuously from three subcutaneous electrodes. Midazolam (MZ) (0.25 mg kg^{-1}) was administered as IV bolus injection after recording a baseline EEG. Eucapnia ($4.4\text{--}6.0 \text{ kPa}$) and normothermia were maintained. Photoc stimulation ($5\text{--}50 \text{ Hz}$) and hyperventilation (target $\text{PE}'\text{CO}_2$ of 3.3 kPa for 2min) were applied for EEG activation. Total EEG power (P_{tot}), relative power, median frequency (MF), 95% spectral edge frequency (SEF95) and Narcotrend index (NI) were analysed offline (20s epochs). Data were analyzed by Wilcoxon signed rank tests ($p < 0.05$).

The NI was significantly lower in groupD than in groupL. Baseline P_{tot} , MF and SEF95 were significantly lower in groupD. In groupL the NI, P_{tot} , MF and SEF95 showed a significant decrease after MZ. In groupD relative α -power and SEF95 decreased significantly after MZ. Photoc driving occurred more frequently in groupD than in groupL.

In alfaxalone anaesthetized cats MZ did not induce CNS stimulation. The NI was able to distinguish light and deep anaesthesia at the chosen target concentrations.

Pharmacokinetics of carprofen in broiler chickens

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Carprofen improved mobility in broiler chickens and hens with induced arthritis (McGeown et al. 1999, Hocking et al. 2005); however, carprofen dose and efficacy varied between studies. Our study aimed to describe carprofen pharmacokinetics in broiler chickens since they are unknown.

Twenty-seven Gait Score 3, Cobb broilers aged 42-46 days were randomly divided into three groups and given 15, 25, or 35 mg kg⁻¹ carprofen SC. Cloacal temperature was measured and 2 ml blood withdrawn immediately before carprofen administration (T0) and 1, 3, 6 and 24 hours afterwards. Plasma was harvested, and total plasma carprofen concentration and R and S enantiomers were measured by HPLC. Area under the curve (AUC) for plasma carprofen concentration over the 24 hours was calculated for each dose and compared using a one-way ANOVA. Similar analysis compared AUC for cloacal temperature. Data are presented as mean ± SD, statistical significance was set at $P \leq 0.05$.

The AUC for carprofen concentrations were significantly different between doses ($P = 0.0004$). Median (95% ± CI) peak plasma concentrations of carprofen occurred 3 - 6 hours after administration and were 17.07 (± 13.28-27.19), 23.13 (± 20.20-28.23), 31.37 (± 16.80-48.24) µg ml⁻¹ in the three groups respectively; concentrations were negligible 24 hours later. Cloacal temperatures were within normal limits (41.2 ± 0.3 °C) at T0 and T24. Cloacal temperature decreased after carprofen administration; AUCs were significantly different between groups ($P = 0.04$). Lowest cloacal temperatures were 40 °C in the 35 mg kg⁻¹ group, and occurred 6 hours after carprofen administration.

Decreased body temperature following carprofen administration to apyrexia animals has not been previously reported in any species. The decrease reported here may have been induced by carprofen. The mechanism requires elucidation.

References

Hocking PM, Robertson GW, Gentle MJ (2005) Effects of non-steroidal anti-inflammatory drugs on pain-related behaviour in a model of articular pain in the domestic fowl. *Res Vet Sci* 78, 69-75.

McGeown D, Danbury TC, Waterman-Pearson AE, Kestin S (1999) Effect of carprofen on lameness in broiler chickens. *Vet Rec* 144, 668-671.

Poster Session Room “Lobby”

The influence of ketamine on regional cerebral perfusion and on the serotonin-2A receptor in the canine brain.

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Ketamine induces antidepressant effects, possibly by interacting with the serotonin (5-HT_{2A}) receptor (Diazgranados et al., 2010). This interaction was investigated using Single Photon Emission Computed Tomography (SPECT) with ¹²³I-R91150.

Seven six-year-old Beagles were used in this study. For the perfusion study, ^{99m}Tc-Technetium-Ethyl Cysteinate Dimer was injected IV during general anaesthesia with a continuous rate infusion (CRI) of propofol (0.4 mg kg⁻¹ minute⁻¹) and during a CRI of the combination of propofol (0.2 mg kg⁻¹ minute⁻¹) and ketamine (0.03 mg kg⁻¹ minute⁻¹) on two separate occasions to create a perfusion pattern map representing the regional cerebral blood flow. For the receptor binding study, ¹²³I-R91150 was injected IV, anaesthesia was induced and maintained with propofol (6.9 ± 0.6 mg kg⁻¹ followed by 0.4 mg kg⁻¹ minute⁻¹ IV) 90 minutes after tracer administration and the first acquisition was started 15 minutes after induction of anaesthesia. After this acquisition propofol (0.2 mg kg⁻¹ minute⁻¹) was combined with ketamine (2 mg kg⁻¹ followed by 0.03 mg kg⁻¹ minute⁻¹) and the second acquisition was started 15 minutes later. For both studies, semiquantification, with the cerebellum, a region devoid of 5-HT_{2A} receptors, as a reference region, was performed to calculate the perfusion indices (PIs) and the 5-HT_{2A} receptor binding indices (BIs) (parameter for available receptor density) in the frontal, parietal, temporal and occipital cortex and the subcortical region. Both PIs and BIs were analyzed with paired t-tests.

Adding ketamine to the propofol CRI significantly increased PIs in the left frontal (1.06 ± 0.03 compared to 0.99 ± 0.04, $p = 0.01$) and right parietal cortex (1.01 ± 0.05 compared to 0.92 ± 0.03, $p = 0.01$). 5-HT_{2A} BIs were not significantly altered by ketamine.

Ketamine caused regional perfusion alterations but no acute changes in the 5-HT_{2A} receptor status measured with ¹²³I-R91150 SPECT.

Reference

Diazgranados N, Ibrahim L, Brutsche NE, et al. (2010) A randomized add-on trial of an N-methyl-D-aspartate antagonist in treatment-resistant bipolar depression. *Arch Gen Psychiatry* 67(8),793-802.

Oral transmucosal and intramuscular administration of dexmedetomidine and buprenorphine in healthy adult cats

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The purpose of this study was to compare the sedative and antinociceptive effect of dexmedetomidine and buprenorphine after oral transmucosal (OTM) and IM administration in healthy adult cats.

Following a randomized blinded crossover protocol (1 month wash-out), a combination of dexmedetomidine ($40 \mu\text{g kg}^{-1}$) and buprenorphine ($20 \mu\text{g kg}^{-1}$) was given OTM (buccal cavity) or IM (quadriceps muscle) in 6 female neutered cats, weighing 5.3 to 7.5 kg. The sedative effect was measured with a Numerical Rating Scale (NRS) at baseline, at 15 and 30 minutes, as well as at 1, 2, 4 and 6 hours after treatment. Simultaneously, analgesia was scored by a Dynamic and Interactive Visual Analogue Scale (DIVAS) based on the response to an ear pinch and by the cat's nociceptive response to a mechanical threshold exerted by a pressure rate onset device. At the same time points, buccal pH and respiratory and heart rate were measured. Side-effects such as salivation, vomiting and behavioural changes were observed. Signed rank tests were performed.

For the NRS, a significant difference ($p = 0.03$) was found in the overall median value between the OTM group (2.64; range 2.81-1.14) and the IM group (2.98; range 3.52-1.52). Nociceptive thresholds increased after both treatments but there was no significant difference in overall median values. The DIVAS was higher in the OTM group, although not significantly. Buccal pH was consistently between 8 and 8.5. Salivation was noted shortly after OTM administration ($n = 2$). Onset of vomiting was observed within 30 min after administration in both groups (OTM: $n = 4$, IM: $n = 3$).

In healthy adult cats, the OTM administration of dexmedetomidine and buprenorphine produced less sedation than the IM administration. Nociceptive thresholds were increased following OTM and IM administration without significant difference between both groups.

Antinociceptive effects of low dose ketamine infusions in conscious cats.

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Subanesthetic concentrations of ketamine have gained acceptance for acute and chronic pain management in dogs, horses and humans. The antinociceptive effects of two low dose ketamine infusions in cats using mechanical and thermal threshold models were studied.

Eight healthy adult cats (six castrated males, two females, 3.7-6.7 kg) were studied in a randomized, blinded crossover design with minimum eight days between treatments. The thorax and lower thoracic limbs of each cat were shaved for thermal (TT) and mechanical threshold (MT) testing respectively, and a cephalic catheter was placed for drug administration. Treatments were 0.5 mg kg⁻¹ ketamine followed by a continuous rate infusion of ketamine of either 5 (Group K5) or 23 (Group K23) µg kg⁻¹ minute⁻¹ for 2 hours, or an equivalent volume of 0.9% saline solution given as loading dose and infusion (Group S: Control). Sedation scores, MT and TT were obtained prior to drug treatment (baseline) and 0.25, 0.5, 0.75, 1, 1.5, 2, 2.25, 2.5, 2.75, 3, then every 30 minutes for 7 hours, and at 10, 12, 14 and 26 hours after administration of loading dose. Data were analyzed by repeated measures ANOVA with correction for multiple comparisons. $P < 0.05$ was deemed significant.

Ketamine induced mild sedation during the infusion and no other adverse behavioral effects were observed. Thermal threshold was significantly higher than baseline (K5: 44.5±0.7 °C; K23: 44.5±0.5 °C) at 15 minutes in the K5 group (46.8±3.5 °C) and at 45 minutes in the K23 group (47.1±4.1 °C). In the K23 group TT was significantly increased compared to S and K5 at 45 minutes. In K5 at 15 minutes MT (9.6±4.0 N) was different to baseline (6.1±0.8 N) and to the S group (5.9±2.3 N).

Results indicate that low dose ketamine infusions minimally affect thermal and mechanical antinociception in cats.

Physiological effect of sedation with a combination of subcutaneous alfaxalone and butorphanol in hyperthyroid cats

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To evaluate the sedative, respiratory and cardiovascular effects of subcutaneously administered alfaxalone and butorphanol in a group of hyperthyroid cats

A prospective single-centre study of twenty nine client-owned hyperthyroid cats. Cats were examined and sedated with alfaxalone ($2\text{--}3\text{ mg kg}^{-1}$) and butorphanol (0.2 mg kg^{-1}) administered subcutaneously. The objective was to achieve an end-point sedation with minimal resistance to handling and oral medication with an I^{131} therapy capsule, whilst maintaining an intact gag reflex. Sedation score, HR, fr , MAP, SAP and DAP were recorded every 15 minutes following drug administration for a total observation period of 45 minutes. Sedation score utilised physical criteria including posture, eyelid reflex, globe position, jaw tone and response to stimuli (Alvaides et al., 2008). A Dunnett's test was used to compare all parameters, except sedation, at each time point with the control time (0 minutes). Sedation score at each time point was assessed using a one-sided sign test.

Four of the 29 cats were excluded from analysis due to incomplete data or incomplete dosing. Twenty cats received 3 mg kg^{-1} and 5 cats received 2 mg kg^{-1} of alfaxalone. For the 3 mg kg^{-1} group the maximum median sedation score of 10 (range 4–18) was reached 45 minutes after injection ($p < 0.0001$). The lowest mean $\pm$ SD HR ($190.6 \pm 46.1\text{ beats minute}^{-1}$), fr ($33.0 \pm 15.1\text{ breaths per minute}$), SAP ($134.1 \pm 24.2\text{ mmHg}$) MAP ($108.9 \pm 23.1\text{ mmHg}$) and DAP ($92.2 \pm 24.4\text{ mmHg}$) were observed 30 minutes after injection. There were significant decreases in fr observed at all time points ($p < 0.001$). There were significant decreases in SAP, DAP and MAP at 30 minutes ($p < 0.001$).

Subcutaneously administered alfaxalone and butorphanol can be used for sedation in hyperthyroid cats undergoing short procedures. Transient cardiovascular and respiratory depression may be observed.

Reference

Alvaides RK, Teixeira Neto FJ, Aguiar AJA et al. (2008) Sedative and cardiorespiratory effects of acepromazine or atropine given before dexmedetomidine in dogs. *Vet Rec* 162, 852–856.

The influence of meloxicam and anaesthesia on occult blood in faeces in dogs

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This study investigated the influence of meloxicam and anaesthesia on presence of faecal occult blood in healthy dogs.

Six dogs were studied on two occasions, firstly receiving 0.06 ml kg⁻¹ water for injection and secondly 0.1 mg kg⁻¹ meloxicam, administered orally once daily for ten days with a 10 day washout period between experiments. On day 10 of treatment, dogs were anaesthetized with isoflurane (FE'ISO 0.7 to 2.0 %) in 100% oxygen for 3 hours after premedication with methadone 0.2 mg kg⁻¹ subcutaneously and induction to general anaesthesia with midazolam 0.15 mg kg⁻¹, ketamine 0.5 mg kg⁻¹ and propofol 3 mg kg⁻¹ intravenously. Samples of the first morning faeces were collected on days 1, 4, 7, 10, 11 and 12. Hemoccult test® (guaiac method) was used to detect occult blood. Test cards were read immediately and 48 hours later.

Occult blood in faeces was found positive at both test readings in one dog (day 1) and only at immediate reading in another dog (day 4) in placebo group. In meloxicam group, the positive results were obtained on days 7 to 12 (Table 1).

After anaesthesia, positive results were found only in meloxicam treated dogs. Reduced perfusion of gastrointestinal mucosa during anaesthesia in addition to meloxicam impediment of prostaglandin synthesis, may have contributed to the presence of occult blood in faeces.

Table 1: Occult blood in faeces in meloxicam treated dogs

dog	A		B		C		D		E		F	
day	0h	48h	0h	48h	0h	48h	0h	48h	0h	48h	0h	48h
7	-	-	-	-	-	-	-	-	-	-	-	+
10	-	-	-	-	-	+	+	+	-	+	-	+
11	-	-	-	-	-	-	+	+	-	-	+	-
12	-	-	-	-	+	-	-	-	-	-	+	+

Cardiorespiratory, anaesthetic and analgesic effects of three continuous rate infusions (CRI) of dexmedetomidine in dogs anaesthetized with a CRI of alfaxalone

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The objective was to assess the cardiorespiratory, anaesthetic, analgesic and recovery effects of two CRI dexmedetomidine in CRI alfaxalone anaesthetized dogs.

Six healthy beagles were instrumented for haemodynamic measurements the same day of the experiment with sevoflurane. Afterwards, three treatments were administered to each dog with a 7 day interval: saline (control group, C) or dexmedetomidine at 1 (group LDA) or 2 (group HDA) $\mu\text{g kg}^{-1}$ IV as a loading dose followed by alfaxalone induction and maintained with alfaxalone at $0.07 \text{ mg kg}^{-1} \text{ min}^{-1}$ and dexmedetomidine at 0 (C), 0.5 (LDA) or 1 (HDA) $\mu\text{g kg}^{-1} \text{ h}^{-1}$ IV during 90 minutes. Cardiac index (CI), systemic vascular resistance index (SVRI), stroke volume index (SI), contractility (dPmx), HR, MAP, SAP, DAP, fr , $\text{PE} \text{ CO}_2$, central venous pressure (CVP), bispectral index (BIS), blood-gases and the degree of anaesthesia and analgesia were recorded at predetermined intervals. The times and quality of recovery were scored. A 2-way ANOVA for repeated measurements followed by Bonferroni adjustments was applied between groups. Within the treatment, one-way ANOVA repeated measures with Dunnett's test to compare the baseline with each time point ($p \leq 0.05$).

A decrease in HR (C: 143 ± 20 ; LDA: 78 ± 18 ; HDA: 87 ± 19 $\text{beats} \cdot \text{minute}^{-1}$) and CI (C: 3.98 ± 0.64 ; LDA: 2.02 ± 0.68 ; HDA: 2.42 ± 0.68 $\text{L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$) and an increase in SVRI (C: 2415 ± 1739 ; LDA: 5061 ± 1782 ; HDA: 6584 ± 1120 $\text{dyn} \cdot \text{s} \cdot \text{cm}^{-5} \cdot \text{m}^{-2}$) occurred at 1 and 2 $\mu\text{g kg}^{-1}$ dexmedetomidine. These changes remained during the anaesthetic period, more pronounced with HDA. Respiratory depression was higher after alfaxalone induction, overalls in groups premedicated with dexmedetomidine (fr C: 19 ± 7 ; LDA: 11 ± 5 ; HDA: 9 ± 2 $\text{breaths} \cdot \text{minute}^{-1} / \text{PaO}_2$ C: 86.7 ± 20.2 ; LDA: 56.2 ± 7.4 ; HDA: 58.1 ± 8.4 mmHg). Lower values of BIS was obtained with HDA (C: 85 ± 11 ; LDA: 84 ± 10 ; HDA: 67 ± 10). Quality of recovery increased in dexmedetomidine groups without any differences in recovery times.

The combination alfaxalone-dexmedetomidine improves the degree of anaesthesia and analgesia and the quality of the recovery, with a great cardiorespiratory depression, more pronounced with the high dose dexmedetomidine.

Anaesthetic management during surgical correction of a cardiac defect under beating-heart cardiopulmonary bypass in a Maine Coon cat: a case report

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Open heart surgery using cardiopulmonary bypass (CPB) is very high risk because of potential complications including hemodilution, hypercoagulation, and electrolyte imbalance. We describe here the first report of surgical treatment of cor triatriatum sinister in a cat using CPB.

A 14-month old, 5.9-kg, male castrated Maine Coon cat was referred for a 3-month history of inappetence and weight loss and a 3-day history of dyspnea. Clinical examination showed pale mucous membranes and acute dyspnea. Cardiogenic pulmonary oedema was evident on thoracic radiographs. After medical stabilisation, cor triatriatum sinister was diagnosed by echocardiography and angiography, and surgical treatment under heart-beating CPB was scheduled.

Ketamine and midazolam were administered intramuscularly as a premedication. Following a 10-minute preoxygenation anaesthesia was induced with intravenous etomidate, then maintained with isoflurane in 100% oxygen, after endotracheal intubation. A left lateral thoracotomy was performed. A venous cannula was introduced into the right atrium and an arterial cannula into the descending thoracic aorta. CPB was then established using a neonatal circuit previously primed with cross-matched blood, lactated Ringers solution, mannitol, bicarbonate and heparin, and moderate hypothermia (34°C) was induced. Analgesia was performed using intercostal blocks, morphine, ketoprofen. Post-operative management included blood pressure monitoring, blood-gas, haematology, thoracic radiography and echocardiography. Treatments consisted of slow transfusion of whole blood, crystalloid IV fluid therapy, antibiotics, corticosteroids, diuretics and aspirin. Post-operative complications were transient hyperthermia and moderate thoracic effusion.

The cat was hospitalised in the Intensive Care Unit for 6 days after surgery, then discharged with antibiotic and furosemide treatment. Follow-up evaluation was performed regularly up to 6 months after surgery and showed excellent general status, increased activity and weight gain.

Owner-assessed indices of quality of life in cats and the relationship to the presence of chronic pain-DJD

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The objectives of the study were to describe the types of items considered important by owners for their cats' quality of life (QoL); to describe the proportion of these items that involve mobility; to evaluate what patient factors, including the severity of DJD-associated pain, affect this distribution.

This prospective, observational study included 166 client-owned cats with varying degrees of chronic DJD. A weighted QoL questionnaire was used to collect data. QoL items were categorized into six behavioral domains and further designated by the degree of mobility involved. Items were divided into active (AA; e.g. running), inactive (IN; e.g. sleeping) and items with implied activity (IA; e.g. sitting on window seat). Distributions of AA, IA and IN items were described. Backwards stepping regression analysis was used to assess the effect of cat, owner and veterinarian-assessed variables on QoL item distributions.

840 client-generated items were evaluated. Regardless of pain-DJD status, 40 % of items listed involved mobility, while 60 % were "inactive" items. Increasing age was associated with decreased AA frequency ($P < 0.0001$) and increased IN frequency ($P < 0.0001$). Increased bodyweight was associated with decreased IA frequency ($p = 0.0012$). No other variables affected score distribution. Overall QoL score was significantly lower in cats with worse temperament ($p = 0.0029$) and higher DJD scores ($p = 0.01$); age was not significant in this model.

These results highlight the need to assess non-active items that owners consider constitute QoL to fully assess the impact of diseases like chronic pain-DJD.

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Repeatability and comparison of thermal threshold latency measures in normal dogs and dogs with pelvic limb osteoarthritis

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Chronic pain may be associated with altered sensory processing. Quantitative sensory testing (QST) can test for secondary hyperalgesia and central nervous sensitization. The objectives were to determine whether thermal threshold latencies (TTL) could be measured in client-owned dogs, were repeatable and were different between normal dogs and dogs with pelvic limb OA.

A high-powered light source from a validated thermal threshold device delivered a ramped thermal stimulus to the underside of the pad of the third digit (Wegner et al. 2008). Dogs were standing and minimally restrained. Time to thermal withdrawal was measured using an automated cut-off when the limb was moved. Five tests were performed on each pelvic limb at each time point. Clinically normal dogs ($n = 23$) and dogs with OA-associated pelvic limb pain ($n = 11$) were tested on two occasions 2 weeks apart. Right and left limb TTL were pooled and mean data were evaluated using paired and unpaired comparisons and Bland Altman plots.

Thermal thresholds were successfully measured in 34/34 client-owned dogs without prior training. Week-to-week TTL did not vary significantly in normal ($p = 0.95$) or OA dogs ($p = 0.71$). Although there was no significant difference between control group TTL (15.6 ± 3.0 s) and OA group (18.3 ± 5.6 s) ($p = 0.09$), OA TTL values were higher.

Thermal thresholds can be measured in client owned dogs with no training, and are repeatable from week to week. Further data are required to determine if OA results in thermal hypoalgesia.

Evaluation of meloxicam and tepoxalin in cats

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There have been recent concerns surrounding the use of NSAIDs to treat pain in cats. This study reviewed clinical prescriptions of meloxicam (Metacam®) and tepoxalin (Zubrin®) in cats, and evaluated the effects on different blood and urine diagnostic values.

Medical records (n = 262) were obtained from the Morrisville Cat Hospital. The criteria for inclusion in this review were cats with clinical pathology panels before and after courses of NSAIDs. Age, diagnosis, NSAIDs prescriptions, dose, duration, pre-existing diseases, concurrent medications, adverse events were recorded. Two-tailed paired Wilcoxon tests were performed for exploring significant changes ($p < 0.05$) between initial versus final laboratory tests (chemistry and CBCs panels, urinalyses and T4 tests).

A total of 67 medical records fit the inclusion criteria (n = 21 and n = 46, meloxicam and tepoxalin respectively). Average doses administered were 0.012 and 12.09 mgkg⁻¹day⁻¹ (meloxicam and tepoxalin respectively). The median prescription durations were 81 (4-1604) and 10.5 (2-807) days for meloxicam and tepoxalin respectively. Suspected adverse events (resulting in medication being stopped) were reported for meloxicam (23.8 %, 5/21 cats) and tepoxalin (8.69 %, 4/46 cats) a median of 81 and 4 days respectively after the prescription started. There were no statistically significant changes for chemistry and CBC panels, urinalyses and T4 tests, except for a decrease in urinary protein ($p = 0.02$) for tepoxalin. Several parameters were significantly altered in meloxicam cats but remained in the normal range.

Further investigation of safety and efficacy is warranted for tepoxalin.

Pulse pressure and stroke volume variations in a model of hemorrhagic shock in mechanically ventilated dogs – preliminary results

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The objective was to study pulse pressure and stroke volume variations (PPV, SVV) as volume status indicators.

Four beagles anesthetized with propofol and rocuronium were instrumented for thermodilution cardiac output, PPV, and SVV measurements (LidCOplus). Hemorrhagic shock was induced targeting MAP of 40 mmHg. Blood was replaced followed by hetastarch-induced hypervolemia (20 mL kg⁻¹). Data collected at baseline (B), after hemorrhage (H), after transfusion (T), and after hetastarch (HES) included cardiac index (CI, L min⁻¹ m⁻²), stroke volume index (SVI, mL m⁻²), PPV (%), SVV (%), central venous pressure (CVP, mmHg). Analysis was performed with repeated measures ANOVA ($p \leq 0.05$).

Blood loss was 40 to 51.8%. At H, MAP was 28 ± 4 (mean $\pm$ SD). CI and SVI decreased from 5.1 ± 2.2 and 48.7 ± 15.5 (B) to 1.2 ± 0.6 and 9.7 ± 4.5 (H) ($p = 0.0028$; 0.0004), and normalized after transfusion (T: 5.6 ± 2.1 and 46.4 ± 16.9 ; $p = 0.0012$, 0.0006). Neither CI nor SVI changed after HES. SVV and PPV increased from 10 ± 2.2 and 9.5 ± 0.6 (B) to 38.5 ± 7.6 ($p < 0.0001$) and 38.4 ± 4.8 ($p < 0.0001$) (H), normalizing after transfusion (7.8 ± 2.6 ; 8.8 ± 1.5 , $p < 0.0001$). CVP did not change after hemorrhage. It increased after transfusion (H: 1.8 ± 3.7 , T: 15.4 ± 5.3 ; $p = 0.0002$), but not after HES.

PPV and SVV are reliable indicators of hypovolemia in dogs. CVP failed to reflect profound hemorrhage in dogs.

Thermal and mechanical nociceptive threshold testing in pregnant sheep

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There is limited information on analgesia for sheep. We compared analgesic regimes in pregnant ewes after laparotomy by measuring thermal (TT) and mechanical (MT) nociceptive thresholds.

Ewes (128 days gestation) underwent laparotomy for another research project. TT and MT were measured before, and 2, 6, 24 and 48 hours after surgery. Thermal stimuli (cut-off 60 °C) were delivered to the lateral aspect of the metatarsus via a skin-mounted probe, and mechanical stimuli to the contralateral site via a pneumatically driven 1.5 mm diameter pin. Each test was performed 5 times, alternating thermal and mechanical stimuli, with 10 minutes between thermal stimuli. At the end of surgery ewes received either: 100 µg hour⁻¹ transdermal fentanyl patch (medial thigh) (group FP) (n=8), or 3 µg kg⁻¹hour⁻¹ intra-peritoneal medetomidine osmotic pump (group IPM) (n=8) inserted immediately prior to closure. Data were analysed using the Kruskal-Wallis RS Test ($p < 0.05$). Once a significant effect was identified, pairwise comparisons were performed using paired Wilcoxin RS tests. To compensate for multiple hypotheses testing, $p < 0.005$ was considered significant.

Prior to surgery mean $\pm$ SD TT was $56.1 \pm 5.0^{\circ}\text{C}$ (FP) and $55.6 \pm 5.5^{\circ}\text{C}$ (IPM); MT was 6.3 ± 2.6 N (FP) and 8.0 ± 5.0 N (IPM). In FP there was no significant change in either TT or MT over time. In IPM there was no significant change in MT over time but TT increased at 2 hours to $59.2 \pm 3.0^{\circ}\text{C}$ ($p = 0.003$). Skin temperature (ST) ranged from 33.0 - 34.7°C and did not change over time. There were no significant differences between groups in TT, MT or ST. Administration of intra-peritoneal medetomidine ($3 \mu\text{g kg}^{-1} \text{ hour}^{-1}$) by an osmotic pump increases the thermal nociceptive threshold in the immediate post operative period in pregnant sheep. Medetomidine

Cardiorespiratory and anaesthetic effects of a continuous rate infusion of fentanyl in dexmedetomidine-morphine-propofol-isoflurane anaesthetized sheep

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Fentanyl patch has been evaluated in sheep under orthopaedic experimental surgery (Arhern et al. 2009), but no studies of fentanyl infusion in sheep has been published.

A prospective, randomized, blinded controlled study was performed in twenty adult, female sheep (41.1±4.5 kg) to determine the anaesthetic and cardiorespiratory effects of a continuous rate infusion (CRI) of fentanyl in sheep anaesthetized with isoflurane. Sheep were sedated with dexmedetomidine (4 µg kg⁻¹, IV) and morphine (0.2 mg kg⁻¹, IV). Anaesthesia was induced with propofol IV to effect, and maintained with isoflurane in 100% oxygen. Ten sheep received fentanyl 10 µg kg⁻¹ hour⁻¹ IV (group F) and ten received saline (group P) during anaesthesia. Endtidal isoflurane concentration (FE'iso) required for maintenance based on reflexes and cardiovascular parameters were recorded. The following were recorded every 5 minutes: FE'iso, FE'CO₂, fr, HR, SAP, DAP, MAP, cardiac index (CI), systemic vascular resistance index (SVRI), stroke volume index (SVI), left ventricular contractility (dPmx) and central venous pressure (CVP). Arterial blood-gases and electrolytes were measured at baseline, 5 minutes after induction and every 30 minutes thereafter. Quality of sedation, induction and recovery were assessed using a numerical rating scale and compared with Mann Whitney tests (p<0.05). Recovery times were recorded from stopping anaesthetic administration. Cardiorespiratory parameters mean values during the whole procedure and recovery times were compared using unpaired t-tests (p<0.05).

FE'iso (1.1±0.3% vs 1.3±0.6%) was significantly lower in group F (p=0). No differences in recorded data were seen apart from in SVRI that was significantly lower in group F (36.9±13.8 vs 46.7±29.5 dynes seconds cm⁻⁵) (p=0.005). Sodium (141.2±6.4 vs 137.4±7.2 mmol L⁻¹) (p=0.01) and Calcium total (1.02±0.12 vs 0.94±0.11 mmol L⁻¹) (p=0.001) were significantly higher in group F.

Fentanyl CRI did not affect the cardiorespiratory parameters in sheep anaesthetized with Isoflurane, and it reduced FE'iso 18.6%.

Ahern B, Soma L, Boston R, Schaer T (2009). Comparasion transdermally administered fentanyl and intramuscularly administered buprenorphine during and following orthopedic surgery in sheep. Am J Vet Res. Mar;70(3):418-22

Chemical immobilization of chimpanzees (*Pan troglodytes*) using a combination of detomidine and ketamine

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To determine if detomidine-ketamine can be used for chemical immobilization of chimpanzees.

Twenty-one adult captive chimpanzees (12 males, 9 females, age 8-46 years, weighing 40.4-68.4 kg) were immobilized for routine check-up with detomidine-ketamine (50 $\mu\text{g kg}^{-1}$ and 4 mg kg^{-1} in groups 1 (n=8) and 2 (n=7), 60 $\mu\text{g kg}^{-1}$ and 5 mg kg^{-1} in group 3 (n=6)) IM in the pelvic limbs by dart. Groups were based on social structure and immobilized on different days. A period of 8 minutes in group 1 and 15 minutes in groups 2+3 was respected before removing the animals from their enclosures. Body temperature, lactate and glucose values (at the beginning and end of clinical examination), and peripheral haemoglobin saturation and PR (grouped per 5 minutes) were measured as long as immobilization was sufficient. The induction (darting-immobilization), total anaesthetic (induction-full recovery) and recovery times with or without atipamezole were recorded.

Immobilization was observed 4.3 (2.2-20) minutes after darting. Early handling of chimpanzees often resulted in arousal. Additional ketamine IM ($2.90 \pm 1.23 \text{ mg kg}^{-1}$) was required (group 1 n = 5, 2 n = 3, 3 n = 3). Correctly dosing based on estimated weight was difficult. Administered dose of detomidine ($\mu\text{g kg}^{-1}$) and total ketamine (mg kg^{-1}) in group 1 was 52.58 ± 6.02 and 5.93 ± 1.6 respectively; in group 2 59.59 ± 5.74 and 5.92 ± 1.18 ; in group 3 62.31 ± 6.00 and 6.88 ± 2.13 . Most animals were hypoxaemic (SpO_2 92 (74-97)% and hypothermic ($35.9 \pm 1.3^\circ\text{C}$). Occasionally, bradycardia ($59.6 \pm 11.3 \text{ bpm}$) and mild hypoglycaemia ($81 \pm 14.9 \text{ mg dL}^{-1}$) were observed. Lactate remained acceptable (1 (<0.8 -5.3) mmol L^{-1}). Atipamezole ($121.1 \pm 23.9 \mu\text{g kg}^{-1}$) IM assured an acceptable quality of recovery after 12.9 (5-58.5) minutes. Duration of immobilization varied (75.9-177.3 min) when no reversal agent was administered (n=4).

Detomidine (60 $\mu\text{g kg}^{-1}$) and ketamine (5-6 mg kg^{-1}) can be used for immobilization of chimpanzees for minimally invasive procedures.

Use of a commercial bellows-foot pump for mechanical ventilation during large animal field anaesthesia

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The aim of the study was to evaluate efficiency of an adapted commercial bellows-foot pump used to ventilate horses during prolonged field anaesthesia.

A commercial 2.5 L bellows-foot pump with a custom made ball-operated expiratory valve was used to ventilate five two years old Haflinger stallions (350 ± 15 kg) during field anaesthesia for castration in lateral recumbency. Horses were pre-medicated with acepromazine ($0.03 \text{ mg kg}^{-1} \text{ IM}$), detomidine ($0.015 \text{ mg kg}^{-1} \text{ IV}$) and butorphanol ($0.01 \text{ mg kg}^{-1} \text{ IV}$). Anaesthesia was induced with ketamine ($2.2 \text{ mg kg}^{-1} \text{ IV}$) and midazolam ($0.1 \text{ mg kg}^{-1} \text{ IV}$). Endotracheal intubation was performed and anaesthesia maintained with total intravenous anaesthesia (midazolam $0.09 \text{ mg kg}^{-1} \text{ hour}^{-1}$, ketamine $3.3 \text{ mg kg}^{-1} \text{ hour}^{-1}$ and xylazine $0.3 \text{ mg kg}^{-1} \text{ hour}^{-1}$). The bellows-foot pump and the expiratory valve were connected to the endotracheal tube. Oxygen (6 L minute^{-1}) was supplied via tubing into the open expiratory valve or via access port on the foot pump. Horses were monitored clinically and an anaesthetic monitor provided spirometry, respiratory gas analysis and pulse oximetry. Arterial blood samples were analysed with a portable blood gas analyser. When PaCO_2 exceeded 5.9 kPa , intermittent positive pressure ventilation was provided by quick consecutive compressions (2-3) of the pump delivering a tidal volume of approximately 10 ml kg^{-1} . The frequency was guided by end tidal CO_2 and blood gas analysis.

In all horses PaCO_2 could be maintained $< 6.65 \text{ kPa}$ ($6.18 \pm 3.06 \text{ kPa}$, mean $\pm$ SD) until end of anaesthesia (44 ± 11 minutes) using a mean respiratory rate of 6.3 (range 4-10). Peak inspiratory pressure was $24 \pm 6.6 \text{ cmH}_2\text{O}$. Inspired oxygen concentration ranged from 26-46 % ($36 \pm 7\%$) and PaO_2 from $8.38\text{-}11.03 \text{ kPa}$ ($10.1 \pm 0.933 \text{ kPa}$).

The adapted bellows foot pump can be used to provide effective mechanical ventilation during large animal field anaesthesia.

The use of Monte Carlo Simulation to evaluate the influence of measurement errors on parameters of gas exchange during equine anaesthesia

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This study investigates the influence of measurement error on the calculation of the residual isoflurane gradient (RIG) in anaesthetized horses. The RIG is the alveolar- to-arterial partial pressure difference for isoflurane corrected for alveolar dead space and shunt using a complicated equation with 20 directly measured variables. RIG was previously calculated in 6 horses in 3 body position/ventilation mode combinations (Bergadano et al. 2002) and yielded physiologically unexplainable negative values. The hypothesis was that negative values were due to random measurement error.

A statistical method, the Monte Carlo Simulation, was used to study influence of measurement error on the RIG. MC-Simulation propagates input uncertainty through a model to determine output uncertainty. Using documented measurement errors for all measuring devices (input) used in the previous study and the dedicated equations to calculate RIG, 18 datasets representing horse/body position/ventilation mode combinations containing each 1000 possible values for the RIG (output) were generated and analyzed. The simulated mean of the RIG varied between -0.15 kPa and 0.38 kPa. All standard ranges were within the interval from -1.56 kPa to 1.43 kPa. The mean relative error calculated from the 18 datasets was 990%. Fifteen out of 18 datasets contained 8 to 90% negative values.

The algebraic sign of the RIG defines the direction of gas exchange for isoflurane at the alveolar-capillary membrane. Positive values represent a gas flow from alveolus to capillary blood which we would expect during steady state anaesthesia. Negative values represent a gasflow from capillary to the alveolus which is physiologically unlikely. It can be concluded from the MC analysis that under the conditions of this study the existence of true negative values cannot be ruled out.

The Monte Carlo Simulation was useful to evaluate the influence of measurement errors on scientific results.

Reference

Bergadano A, Moens Y, Lerou J et al. (2002) Does the positioning of horses influence the end-expired to arterial to mixed venous isoflurane partial pressure differences? Proceedings of the 8th World Congress of Veterinary Anaesthesia, Knoxville, USA. p.133

Thermal threshold testing for standardized assessment of nociception in horses - comparison between different stimulation sites and acepromazine alone or combined with buprenorphine

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The aim of the study was to compare thermal thresholds (TT) determined at the head or body to assess cutaneous anti-nociception in horses.

The study was carried out as a randomized, blinded, controlled trial with cross-over design. Eleven horses (5 - 23 years, 600 ± 95 kg) were used. Thermal thresholds were determined by incremental contact heat applied to the skin above the nostril (N) or the withers (W). Safety cut out was 54°C. Horses were treated IV with saline (S), acepromazine (0.05 mg⁻¹ kg⁻¹) (ACE) or acepromazine and buprenorphine (0.0075 mg⁻¹ kg⁻¹) (AB). Single stimulations were performed 15 minutes prior and 15, 45, 75, 105, 165, 225, 285, 405 and 525 minutes after treatment. Sedation score, gastrointestinal auscultation score and occurrence of skin lesions were recorded. Data were analysed with analysis of variance for repeated measurements followed by Dunnett's t tests (SAS 9.2.). Alpha was set at 5%.

There were no significant differences in TT between N and W with any treatment. The TT remained constant after S (N: 50.4 ± 3.0°C, W: 49.4 ± 3.5°C) and there was no difference in TT between S and ACE. After AB there was a significant increase above baseline in TT for 405 minutes at N and W. Restlessness occurred 30 - 90 minutes after AB in 7 horses. All horses had reduced to absent borborygmi after AB for 165 to 495 minutes.

Thermal stimulation at both described body areas gives consistent results in the assessment of cutaneous nociception in horses.

Preliminary results from the clinical evaluation of S-ketamine as a single intravenous bolus to induce general anaesthesia in horses undergoing field surgical castration

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This prospective clinical trial investigates the use of a single IV bolus of S-ketamine at 2.5 mg kg⁻¹ to allow for surgical castration of horses.

Healthy young males (1-5 years) scheduled for field castration were enrolled. After IV romifidine (0.1 mg kg⁻¹) and L-methadone/fenpipramide (50/2.5 µg kg⁻¹), general anaesthesia was induced with IV S-ketamine (2.5 mg kg⁻¹) and diazepam (0.05 mg kg⁻¹), and oxygen insufflated in a nostril. Heart rate, pulse quality, *fr*, FE'CO₂, and pulse oxymetry were continuously monitored. Lidocaine 2% was administered intratesticular. The horses were left recovering unassisted but under continuous observation and the recovery quality was scored.

Preliminary results are presented for 19 horses. Three horses required a rescue S-ketamine bolus (1 mg kg⁻¹) before the end of surgery at 13.5, 14.5, and 17 minutes after induction. Median times and scores for anaesthetic and surgical events are presented in Table 1. Strong palpebral reflex occurred at 14 (IQR, 11-15.5) minutes before sternal recumbency, and appeared to be a good predictor of recovery time. Nystagmus only appeared temporarily in 5 horses.

General anaesthesia achieved from a single IV bolus of S-Ketamine at 2.5 mg kg⁻¹ was satisfactory with good quality of induction and recovery, but of short duration.

	Median	25% percentile	75% percentile	Minimum	Maximum	n
Induction score (/5)	1	1	2	1	4	19
Recovery score (/5)	1	1	2	1	3	15
Induction-end of surgery	14.5	13	16	11	18	16
Induction-palpebral	14	11.5	16.5	7	22	17
Induction-first movement	25	23	28	16	32	15
Induction sternal	28	26	31	20	34	15
Induction standing	29	26	34	20	49	15

Table 1. Quality scores (1=best; 5=worst) and time (minutes) elapsed between induction of anaesthesia and the occurrence of recorded events.

Effects of constant rate infusions of dexmedetomidine associated with butorphanol, and dexmedetomidine alone in conscious horses

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Dexmedetomidine and butorphanol administered by constant rate infusion (CRI) may be useful for continuous sedation in horses. The hemodynamic and sedative effects of dexmedetomidine CRI alone or with butorphanol were evaluated.

Six adult horses ($432 \pm 25 \text{ kg}^{-1}$) randomly underwent two 90 minute CRI treatments 7 days apart: dexmedetomidine (D) and dexmedetomidine/butorphanol (DB). After instrumentation while unsedated all horses received D: dexmedetomidine ($3.5 \mu\text{g.kg}^{-1}$ bolus IV, followed by $5 \mu\text{g.kg}^{-1}.\text{hour}^{-1}$ CRI); or DB: dexmedetomidine ($3.5 \mu\text{g.kg}^{-1}$ bolus IV, and $3.5 \mu\text{g.kg}^{-1}.\text{hour}^{-1}$ CRI) and butorphanol (0.02 mg.kg^{-1} bolus IV, and $0.024 \text{ mg.kg}^{-1}.\text{hour}^{-1}$ CRI). Sedation scores, cardiac output (measured by thermodilution) and respiratory variables were recorded before sedation, during (5, 15, 30, 60 and 90 minutes) and after infusions (15, 30 and 60 minutes). Student's t-test and ANOVA, followed by the Tukey-Kramer method were used to compare cardiorespiratory values (mean $\pm$ SD), and Friedman's and Dunn's tests were used for non-parametric analysis ($p < 0.05$).

Cardiac index and heart rate decreased during both treatments in a similar manner (D: 60 ± 9 to $38 \pm 4 \text{ mL.min}^{-1}.\text{kg}^{-1}$ and DB: 63 ± 6 to $37 \pm 4 \text{ mL.min}^{-1}.\text{kg}^{-1}$; D: 44 ± 3 to $29 \pm 4 \text{ beats.min}^{-1}$ and DB: 43 ± 5 to $30 \pm 4 \text{ beats.min}^{-1}$) at 5 minutes of infusion. Systemic vascular resistance index increased from baseline in D (129 ± 15 to $210 \pm 14 \text{ dynes.s.cm}^{-5}.\text{kg}^{-1}$) and in DB (132 ± 19 to $218 \pm 25 \text{ dynes.s.cm}^{-5}.\text{kg}^{-1}$) at same moment. Sedation scores (head drop D: 47% and DB: 43%, at 15 minutes of infusion), and recovery from ataxia were similar between treatments. Head position remained lower than 50% of baseline until 30 minutes of CRIs.

Both CRIs provided steady sedation for 30 minutes and were considered adequate sedation protocols for short diagnostic procedures in standing horses.

Sublingual administration of detomidine in horses: sedative effect, analgesia and detection time

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Detomidine is a frequently used sedative in horses, placed on the prohibited substances list of the Fédération Equestre Internationale (FEI). The objectives of the present study were to examine the quality of sedation, presence of analgesia and the detection time in an official FEI referenced laboratory after sublingual administration.

A single dose of 40 $\mu\text{g kg}^{-1}$ detomidine oromucosal gel was administered sublingually to ten healthy Dutch warmblood mares. Blood samples, acquired by jugular venipuncture, and urine samples, obtained by catheterisation, were collected before and for eight days after administration and examined by a FEI laboratory. Sedation was evaluated by measuring head height and scoring the reaction to auditory and combined auditory/sensory stimuli. Mechanical Nociceptive Thresholds (MNTs) were used to assess analgesia. Furthermore HR was measured and ataxia was scored. Mixed model analyses were performed with a Poisson distribution for nonparametric data.

All plasma and urine samples were negative from 48 and 60 hours post administration respectively. Decrease in HR and head height was maximal at 40 and 60 minutes, respectively. Maximal decrease in the response to stimuli was observed at 100 minutes. Ataxia was maximal at 60 minutes. At 40 and 80 minutes MNTs were significantly increased compared to baseline. All parameters, except the MNTs of two locations, returned to baseline values within 24 hours post administration.

In conclusion, sublingual detomidine provided significant sedative effects and showed analgesic efficacy based on MNTs. Maximum detection time was 60 hours and a withdrawal time of 72 hours may be recommended.