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Feline Health Topics

for veterinarians

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The Effects of Xylazine on Cardiac Function

Editor's Note: During the past three years the Birmingham Feline Fancier's Cat Club of Birmingham, Alabama has sponsored a Summer Fellowship Program in Feline Medicine at the Cornell Feline Health Center. The following report summarizes the study worked on by the 1984 fellowship recipient, Nancy Dunkle (Purdue University). Her advisor for the project was Dr. Sydney N. Moise. The study was published in the *American Journal of Veterinary Research*, 47:10 (2212-2216), 1986.

Although the sedative/analgesic drug xylazine (Rompun^R, Haver-Lockhart Laboratories) is approved for use in the cat, there are associated side effects of the drug. Reported non-cardiovascular side effects include prolonged depression of the thermoregulatory function, hyperglycemia, and vomiting. The use of a parasympatholytic agent (i.e. atropine sulfate or glycopyrrolate) in conjunction with xylazine has been recommended to prevent marked bradycardia or atrioventricular block. The safety of this drug combination is questionable, as well as the use of xylazine by itself.

The purpose of this study was to evaluate cardiac function in cats sedated with xylazine or the combination of xylazine and glycopyrrolate. The method chosen to evaluate cardiac function was M-mode echocardiography, a non-invasive technique. Nine 13- to 18-month-old cats (5 males, 4 females) were studied. Each cat was evaluated during (1) manual restraint (2) after IM injection of 0.55 mg of xylazine/kg of body weight (3) after 2.2 mg IM of xylazine/kg and (4) after 0.011 mg IM of glycopyrrolate/kg followed 10 minutes later by 2.2

mg IM of xylazine/kg. Treatments were administered randomly with 72 to 120 hours between treatments. The results of the study showed that left ventricular function was decreased when xylazine was administered.

Conclusions from the study were:

- (1) Conscientious cardiovascular monitoring of a cat is paramount when xylazine or xylazine/parasympatholytic combination is administered.
- (2) The use of parasympatholytic drugs may not improve overall cardiac function. However, the bradycardiac effect of xylazine can be so profound that it may warrant the administration of a parasympatholytic drug (i.e. glycopyrrolate or atropine).
- (3) Cats with compromised cardiac function should not be sedated with xylazine. ■

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Isoflurane: A New

Charles E. Short,

Although there are many advantages of using methoxyflurane or halothane for clinical anesthesia in small animal practice, there are a variety of complications as well as circumstances where other methods of anesthesia are justified. Halothane sensitizes the myocardium to catecholamines. In those patients with myocardial sensitivity, such as traumatic myocarditis, halothane could increase the likelihood of life-threatening ventricular arrhythmias during the surgical procedure. It became apparent, because of the extent of biotransformation of both halothane and methoxyflurane, that patients with hepatic dysfunction were not good candidates for their use. Neither were these good candidates for injectable anesthesia. It was shown the breakdown of methoxyflurane released the fluoride ion which caused renal toxicity. Its renal toxicity made it less desirable in animals with renal failure. Furthermore, a few practitioners developed health problems because of their sensitivity to chronic low-level exposure to halothane or methoxyflurane.

Since halothane and methoxyflurane were not always the ideal agents to use for inhalant anesthesia in veterinary medicine, the development of enflurane and isoflurane was of significant interest to us. Both these inhalant agents showed less renal and hepatic responses because there was less biotransformation of the drug during the elimination process. They did not appear to be as arrhythmogenic as halothane and had faster recovery periods than methoxyflurane. Unfortunately, at high concentrations, enflurane did not provide the needed muscle relaxation in some animals and in others could produce slight seizure-like activity. This problem was usually seen in animals receiving higher concentrations of enflurane than needed for surgical anesthesia, but the margin between surgical anesthesia and the concentration which resulted in inadequate muscle relaxation was small. As a result, isoflurane appears to be the promising agent for use in veterinary practice of the recent developments.

Structure and Physical Properties

Isoflurane is a methyl ethyl ether having many properties similar to enflurane. It is a stable compound and is nonflammable. Its molecular weight of 184.5 is the same as enflurane, slightly less than halothane, and more than methoxyflurane. It has a boiling point of 48.5°C compared with 50.2 for halothane. At 20°C, the vapor pressure of isoflurane is 239.5 mmHg compared to 244.2 mmHg for halothane. As a result, isoflurane can be administered in a halothane-type vaporizer with the delivered concentrations being very similar to those expected if halothane were used. Isoflurane has an ethereal odor with mild pungency.

Feline Health Topics

A publication for veterinary professionals

The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats everywhere, by developing methods to prevent or cure feline diseases, and by providing continuing education to veterinarians and cat owners. All contributions are tax-deductible.

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Inhalant Anesthetic

D.V.M., M.S.

Minimum Alveolar Concentrations

The anesthetic potency of isoflurane when compared to diethyl ether, demonstrates equal analgesia and slightly less muscle relaxation. It is more potent than enflurane, but less than methoxyflurane. There is greater muscle relaxation with isoflurane than enflurane. Likewise, recovery from isoflurane is very rapid especially compared to other anesthetic agents.

Uptake and Distribution

There is relatively low solubility of isoflurane in blood and tissues which provides a rapid increase in alveolar concentration of isoflurane during induction of anesthesia and accounts for the rapid decrease during recovery. The solubility in blood for isoflurane as indicated by the blood gas partition/coefficient at 37°C is 1.4 compared to 2.4 and 12.0 for halothane and methoxyflurane respectively. There is little isoflurane absorbed in the fat (fat to gas partition coefficient of 60 for isoflurane, 138 for halothane and 635 for methoxyflurane).

Clinical Responses

The responses by the cerebral system to isoflurane are similar to halothane and methoxyflurane. The degree of central nervous system depression is dose related, is not associated with seizure-like activity at high concentration, and provides classic anesthetic responses as measured by the electroencephalogram.

The greatest change in the cardiovascular system is usually noted during induction where there can be a significant decrease in blood pressure with high concentrations. During the maintenance of anesthesia with isoflurane, blood pressure will be normally stable unless there are contributing factors to cause changes. There is an absence of ventricular arrhythmias. One does not usually observe significant ECG changes during the use of isoflurane. Cardiac output is not as depressed with isoflurane as

with halothane and arterial blood pressures have been stable and at acceptable levels.

Isoflurane can cause marked depression of the respiratory system. Surgical stimulation slightly increases alveolar ventilation. Respiratory depression usually will occur before cardiovascular depression.

Renal or hepatic toxicity have not been demonstrated to isoflurane. There appears to be little metabolism of isoflurane and as a result, less of the fluoride metabolites are found following isoflurane anesthesia.

Summary

The initial results of use of isoflurane in veterinary hospitals has demonstrated advantageous responses in high risk animal patients. Its use in these high risk individuals has included animals suffering from traumatic myocarditis and patients with renal and hepatic dysfunction. It has been used by veterinarians with known sensitivity to other inhalant anesthetics with dramatically reduced occupational health adversities. As a result, isoflurane is an agent which can be selectively considered in veterinary practice. ■

This article was adapted from a seminar presented at the 78th Annual Conference for Veterinarians (January 1987) at the NYSCVM, Cornell University, Ithaca, NY.

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Complicated Ulcers

Thomas J.

One of the most frequent ocular disorder of companion animals is simple ulcerative keratitis. The uncomplicated nature of most corneal erosions and ulcers and the remarkable healing capability of the corneal epithelium generally assure rapid and complete resolution of such lesions. Chronic and/or recurrent corneal ulceration (complicated ulcerative keratitis) occurs far less frequently than simple ulcerative keratitis, but demands careful diagnostic assessment to minimize delayed healing.

Ocular Diagnostic Tests

Ocular diagnostic tests yield invaluable information for the accurate assessment of the causes of complicated ulcerative keratitis. Schirmer's tear test (STT), corneal and conjunctival culture collection and exfoliative cytology, fluorescein dye staining, and tonometry are simple to perform. The practitioner ultimately saves time and improves diagnostic accuracy by performing those tests that are congruent with clinical signs.

The rules governing use of these tests are few but inflexible. Every animal presenting with mucoid or mucopurulent ocular discharge must undergo Schirmer's tear testing prior to instillation of any diagnostic eyedrops such as topical anesthetics, dilating agents, or collyria. Normal feline values are reported to be 17 mm/minute (+/- 6 mm SD), though in actual practice many asymptomatic cats show values less than 10-12 mm. The STT values should be used to confirm the diagnostic suspicion of keratoconjunctivitis sicca (KCS), but not used alone to suggest it when the characteristic mucoid discharge is absent.

Again, prior to the instillation of diagnostic eyedrops, a sample for viral or bacteriologic culture should be collected if the practitioner feels the results may be useful in diagnosis. Nearly all diagnostic eyedrops and collyria contain preservatives which may negate an otherwise positive culture.

Regardless of the apparently superficial nature of a presented eye problem, a complete ocular examination must be performed bilaterally on every patient. This should include assessment of eye-related reflexes (palpebral, pupillary, corneal, menace, conjugate eye movements), the external eye, anterior and posterior segments. A cursory ocular examination may overlook lagophthalmos, exophthalmos, corneal hypesthesia, and other subtle abnormalities which may cause complicated keratitis.

Diagnostic accuracy demands that the practitioner consistently consider all diagnostic possibilities. The following provides basic information on the various causes of complicated ulcerative keratitis.

Degenerative Corneal Disorders

Corneal Mummification:

Corneal mummification or sequestration is a form of ulcerative keratitis which affects both mixed breed and purebred cats. It is characterized by the appearance of chronic, usually superficial corneal ulceration which may involve one or both corneas simultaneously or sequentially. Unresponsive to conventional topical therapy, the condition progresses to involve brownish discoloration of the involved cornea. Eventually, black plaques develop which resemble large foreign bodies.

The cause of mummification has not been documented. Possible etiologies which have been suggested are keratoconjunctivitis sicca (KCS), lagophthalmos-related corneal dryness, and minor entropion.

Suggested treatments include both medical and surgical options. Intensive topical tear replacement combined with antibiotic and atropine therapy may resolve the lesions. If entropion is present, correction is necessary. Surgical superficial-to-deep keratectomy may be the most expedient therapy for treatment of the well-developed black plaques. Owners should be

Chronic Keratitis in the Cat

Kern, D.V.M.

counseled that the condition is often bilateral and may recur despite successful medical or surgical therapy.

Dryness-Related Keratitis:

Corneal dryness occurs because tears are inadequately produced or delivered to the conjunctival sac, or tears are inadequately distributed over the corneal and conjunctival surfaces. KCS, documented by low to absent STT values, results from tear secretory failure or impairment of access of tears to the eye, presumably from damage to the lacrimal gland or ductules. While the causes of KCS are several (infectious, toxic, immune-mediated, congenital malformation, iatrogenic, idiopathic), documentation of its cause in an individual patient is difficult.

Problems of tear distribution are just as important but may be much more subtle. Inefficient blinking (lagophthalmos) results from facial nerve paresis or paralysis; conformational disorders which cause exophthalmos or poor globe/eyelid contact; facial, orbital, or eyelid swelling from trauma or inflammation; and others. Schirmer's tear test values are usually normal in these conditions; thus, assessment of the problem depends upon the completeness of the ocular examination.

Therapy for STT-documented KCS and secondary ulceration is best aimed at adequate topical tear replacement and conventional symptomatic corneal ulcer treatment. This author's preference is to combine aqueous and viscous (ointment) tear replacements at each of four to six treatments daily, with the tear solution preceding the ointment instillation. If STT values are recordable, oral 1 or 2 percent pilocarpine is empirical; however, many cats may not accept it at any dose. Animals with cardiac insufficiency, cardiac arrhythmias, pancreatitis, or chronic diarrhea are not candidates for pilocarpine therapy, as the drug may aggravate these conditions. Also, prior to anticipated long-term oral pilocarpine usage, the practi-

tioner should document the drug's efficacy with rising STT values (without additional tear replacement therapy). The drug's poor palatability and side effects should not be inflicted on animals incapable of responding to it.

The long-term prognosis for vision retention by animals afflicted with KCS is poor. Most animals benefit from short-term antibiotic and topical antibiotic-corticosteroid therapy to treat their secondary bacterial conjunctivitis and to retard the corneal melanosis, neovascularization and scarring which progress inexorably to blindness. In general, the more severe the tear deficiency, the more difficult adequate medical therapy is to achieve, and the faster vision is lost. Parotid duct transposition (PDT) is a potentially useful adjunct to medical tear replacement for animals with severe KCS which is not manageable by topical therapy and/or oral pilocarpine treatment alone. The chemical composition of saliva is dissimilar to that of tears; thus, saliva is a non-physiologic tear substitute probably inferior to the artificial tear replacements commercially available. The secretion's wetness and availability partially compensate for these gross deficiencies. However, these shortcomings cause certain recurrent problems which both owner and clinician need remember prior to surgical parotid duct transposition. Despite its drawbacks, PDT is a valuable option for selected patients and well-informed owners.

Corneal Dystrophy

The existence of an epithelial corneal dystrophy which promotes chronic, superficial corneal ulceration has not been confirmed in cats, as it has in dogs. Nevertheless, many cats develop such chronic corneal erosions, characterized by the presence of a flap of non-adherent epithelium surrounding the erosion.

Successful therapy of these non-healing erosions is based upon removal of the non-adherent epithelial flap. This author's preferred

technique is to debride the loose epithelium with a cotton-tipped applicator wetted with saline or collyrium. Epithelium is removed until a border of adherent epithelium is identified. Caustic agents such as tincture of iodine, Lugol's iodine, or trichloroacetic acid are not used. In my experience, such chemical treatment greatly encourages potentially harmful neovascularization which serves to delay healing further. Debridement is followed by treatment with topical atropine and antibiotic drops or ointments for 7 to 14 days.

There is a good probability for recurrence in the initially affected eye or the other eye, a fact worth emphasizing to owners. Even with prompt debridement and appropriate follow-up, healing may take several weeks. If the practitioner recognizes the presence of a nonadherent epithelial flap adjacent to a corneal erosion at first presentation, he/she should expect delayed healing and counsel owners of the prolonged course.

Metabolic Causes of Keratitis

Experimental evidence derived from laboratory animal models of diabetes mellitus, as well as clinical evidence from studies of diabetic humans, suggests that diabetic individuals form defective corneal epithelial basement membrane. The defective membrane causes predisposition to spontaneous corneal erosion.

Animals which show signs of endocrinopathy and present with non-healing corneal erosions should undergo laboratory tests to confirm the suspected disorders.

Neurotrophic Keratitis

The ophthalmic branch of the trigeminal nerve provides the corneal epithelium and stroma with sensation via numerous nerve terminals within and between the epithelial and stromal layers. Catecholamines and, presumably, other substances are secreted by these terminals which exert a major influence on the normal epithelial regeneration cycle. If the cornea is denervated by injury or dysfunction of this nerve, spontaneous, slowly healing corneal erosions may follow. The hallmark for the diagnosis of this disorder is presence of corneal

hypesthesia. Corneal sensation assessment is accomplished by using a wisp of cotton loosened from the tip of an applicator prior to topical anesthesia. If the erosion occurs unilaterally, sensation comparison between normal and affected sides is recommended. Typically, erosions of this nature involve the central cornea which is exposed between blinks.

Confirmation is difficult and purported causes are vague. Occasionally, animals which have suffered traumatic proptosis of the globe appear to develop corneal anesthesia and chronic keratitis. It is unclear whether traumatic injury directly to the cornea might also be responsible.

Therapy of neurotrophic keratitis includes use of topical antibiotics and atropine. Soft contact lens placement or tarsorrhaphy may accelerate healing. Prognosis for resolution of corneal hypesthesia is guarded.

Infectious/Immune-Mediated Causes of Keratitis

Non-healing superficial corneal erosions rarely are infected by bacteria. Typically, bacterial keratitis promotes progressive cellular infiltration, rapid neovascularization, and stromal loss, resulting in apparent deepening of the corneal defect. Alternatively, even benign-appearing acute or chronic corneal erosions are at greater risk of becoming infected than the intact corneal surface. Spontaneous bacterial infection of the cornea rarely occurs without damage to the corneal epithelial surface, which allows pathogenic bacteria to adhere and then to colonize the eye. Thus, routine use of topical antibiotic therapy for even minor corneal abrasion in animals is defensible defensive medicine.

The feline rhinotracheitis virus, a herpesvirus, has been associated with ulcerative keratitis in young cats and with non-ulcerative stromal keratitis in adult and aged cats. The ulcerative keratitis is probably secondary to corneal epithelial destruction by the virus. The relationship between the feline stromal keratitis and the viral infection is open to question.

(continued on page 8)

Memorial Program Has Many Benefits

The natural death of a pet is difficult for a client to accept; but when it involves a conscious decision to euthanize a faithful feline companion, it is a very traumatic experience. This is a critical time; a time when your client may choose another veterinarian for future pet care. You want to express your compassion and understanding to your client to ease their bereavement ... but how? **The Cornell Feline Health Center's Memorial Program** was specifically designed to communicate your feelings in a heartfelt and consoling way.

Benefits of the Program

The Memorial Program has multiple benefits. Clients find solace in the tribute given in their pet's name. Furthermore, since the contributions are used to support the vital work of the Center, your memorial gift demonstrates your commitment to the continued progress of feline medicine. The treatments and/or methods of disease prevention developed by the Center are routinely applied in veterinary practices, such as the vaccination program for feline respiratory diseases. Ultimately, the progress made by the Center benefits you and your client by helping feline patients live longer and healthier lives.

Presently, over 350 veterinarians participate in the memorial program. Many have reported that their clients have been extremely appreciative, and thus, it has strengthened the veterinarian-client relationship.

How It Works

Complete a memorial card after collecting your euthanasia/disposal fee. Forward the card with your contribution to the Cornell Feline Health Center. (The amount of the contribution is at your discretion; however, most participating veterinarians give \$10 to \$15 per cat.) Upon receipt of your contribution we will mail a personalized acknowledgement letter to your client informing him/her of your thoughtful gesture in their pet's name.

The memorial program is designed to help your client through the bereavement phase. At the same time it allows you to take an active role in advancing feline medicine while building your own practice. Don't lose out on this valuable opportunity; return the coupon in this brochure to order your supply of memorial cards. ■

COUPON FOR MEMORIAL CARDS

(Complete and return this form to: Cornell Feline Health Center, College of Veterinary Medicine, Schurman Hall, Ithaca, NY 14853-6401)

Yes, I want to participate in the Cornell Feline Health Center's Memorial Program. Please send me:

10 cards 15 cards 25 cards

(name)

(street address)

(city)

(state)

(zip)

Keratitis (continued from page 6)

Traumatic Keratitis

Traumatic injuries to the corneal epithelium, basement membrane, and anterior stroma undoubtedly are responsible for many instances of delayed healing of otherwise unremarkable corneal erosions and ulcers. Physical disruption of the epithelial basement membrane may promote abnormal epithelial duplication without normal adhesion. These probably are indistinguishable clinically from spontaneous erosions presumably due to corneal basement membrane dystrophies. Therapy, similar to that recommended for dystrophic corneal erosions, may be successful.

Toxic Keratitis

Occasionally, corneal erosions occur in small animals secondary to toxic injury to the cornea. This more commonly occurs after inadvertent instillation of shampoos, insecticides, or household chemicals. Thorough cleansing of the affected eye immediately after chemical instillation may minimize the injury. Topical antibiotic and atropine therapy is indicated until resolution of the injury.

Summary

Performing a complete eye exam and utilizing the appropriate diagnostic tests is imperative for successful treatment of complicated ulcerative keratitis cases. ■

This article was adapted from a seminar presented at the spring 1987 veterinary conference at Cornell University, Ithaca, NY.

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★ JOB OPPORTUNITY... ★ Feline Extension Specialist

The Cornell Feline Health Center will be expanding its professional staff to include a Feline Extension Specialist. We will be looking for a veterinarian with at least 3 years of clinical experience in feline practice who is adept at interacting with a wide range of individuals.

Duties of the position will include (1) developing the Feline Consultation and Diagnostic Service of the Center (2) serve as assistant director (3) provide information for publications (4) develop innovative approaches to promote the Center's goals.

Interested persons should contact the Director, Cornell Feline Health Center, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853.



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