Compendium

VETERINARY PRACTICE ISSUES

Compendium

APRIL 2005 (VOL 27, NO 4)

Controlling Avian Pain

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Abstract

Little information is available on avian pain and analgesia, and species differences make choosing appropriate analgesics difficult. This article reviews the current literature on avian analgesia, citing both experimental studies and clinical observations. Information on preemptive analgesia and balanced anesthesia is given. Each category of analgesics is discussed, and specific references to birds are provided when possible. Doses for potential analgesics are also provided as reported in the literature.

Analgesia is the relief of pain without loss of consciousness. Analgesics function by decreasing stimulation of the ascending spinal pathways or by activating the endogenous descending pain modulation pathways. Recognizing pain and anxiety in animals is critical for appropriate analgesic selection and pain relief. Furthermore, timely administration of analgesics is important because persistent pain perception can have a negative effect on homeostasis and healing.[1,2]

Lack of adequate information on pain and analgesia makes choosing an appropriate analgesic difficult. Recognizing the signs of pain in birds is complicated by confounding factors such as differences between acute and chronic pain as well as behavioral differences between domestic and wild animals, predator and prey species, and individuals. However, if a procedure or injury involves tissue damage and/or the bird demonstrates changes in posture (i.e., guarding), temperament (i.e., aggressive or passive), or behavior (i.e., lack of eating or activity), the veterinarian should assume that the bird is in pain.[3]

Controlling pain involves pharmacologic, physical, environmental, and behavioral management.[1] Any pain management program should include proper care and nonpharmacologic methods of analgesia such as supporting or bandaging the traumatized area; modifying the environment with appropriate choices and location of perches, bedding, food, and water; and providing a dry, warm, quiet, nonstressful environment. Reducing fear and anxiety with anxiolytics, tranquillizers, and muscle relaxants can decrease muscle tension and central nervous system (CNS) activity.[4]

Research evaluating pain thresholds and changes in them after administering analgesics is limited in birds.[5] Also, there is limited information available on the pharmacokinetics and pharmacodynamics of analgesics in birds. Nevertheless, pharmacologic intervention should be used as it would be in mammals; when possible, observations and clinical studies should be reported in the literature.

Preemptive Analgesia

Tissue injury can induce prolonged changes in CNS function that later influence responses to afferent inputs and contribute to postoperative pain.[6] *Nociception* is the term used to refer to pain perception. Nociceptive information that reaches the spinal cord can produce central sensitization (i.e., a state of spinal neuron hyperexcitability). Studies in mammals show that pain-induced neural changes can be prevented by administering analgesics before injury induces spinal hyperexcitability and pain-related behaviors.[6] In addition, analgesics are less effective when administered after prolonged central excitability or after pain behavior has already been established. Preemptive analgesia blocks sensory nociceptive stimuli from onward transmission, thus reducing overall pain experienced by an animal.[6]

Balanced Anesthesia

Balanced anesthesia refers to administration of several drugs to prevent excess physiologic derangements by any single drug during or after anesthesia.[7] However, most birds are usually anesthetized solely with an inhaled anesthetic (frequently isoflurane).[7] During isoflurane anesthesia, the CNS is depressed sufficiently to prevent pain perception, but isoflurane anesthesia does not provide postoperative analgesia.[8] In fact, all inhaled anesthetics can be hyperalgesic (i.e., increased nociception) at very low concentrations (i.e., those obtained during recovery from anesthesia) by enhancing C-fiber activity.[9] Patients may perceive noxious stimulation from their wounds to be more intense than if no anesthetic were present.[9] Violent recoveries from inhalant anesthesia have been noted in birds,[7] and if hyperalgesia is also present when inhaled anesthetics are at a low concentration, some of this behavior during recovery may be attributable to intense pain. Providing appropriate perioperative analgesia may improve recovery in birds.

Despite a good margin of cardiovascular stability in a variety of mammals,[8] isoflurane at high concentrations can depress both the cardiovascular and respiratory systems in birds, particularly those that are debilitated.[7] A balanced approach to anesthetizing birds may minimize the adverse effects of any single drug and maximize analgesia. In chickens, μ- and k-opioid agonists decrease requirements for isoflurane in a dose-dependent manner.[7] However, combining opioids with isoflurane can cause respiratory depression with little effect on the heart rate and mean arterial pressure.[7] In mammals, opioids and α_2 agonists are usually chosen for acute, sharp pain, whereas NSAIDs are often administered for inflammation and chronic pain.[1]

Opioids

Opioids exert their actions by binding to specific membrane receptors distributed throughout central and peripheral nervous system structures involved in transmission, modulation, and sensation of pain (**TABLE 1**). The three main classes of opioid receptors are μ, d, and k. In mammals, μ receptors are most commonly associated with pain relief, but specific d- and k-opioid agonists can also modulate pain at spinal and supraspinal sites. Opioids can produce analgesia in birds but with variable and conflicting results.[5,7,10,11] Clinical use of opioids has been hindered by lack of published information concerning possible differences in opioid actions between birds and mammals and among different avian species. In mammals, μ- and k-opioid agonists are often used to provide analgesia and CNS depression during anesthesia, resulting in overall reduction in the required concentration of volatile anesthetics.[7] Side effects such as sedation and respiratory depression can be readily reversed with naloxone or naltrexone, but this also terminates analgesia.[1]

In pigeons, the effect of μ- and k-opioid agonists appears to be similar to that in mammals. Autoradiographic studies of the forebrain of pigeons shows a predominance of k receptors compared with those in mammals, but both μ- and k-opioid agonists are capable of producing analgesia.[12] Pigeons are able to discriminate between an intramuscular injection of morphine (a μ-opioid agonist) and saline but are unable to distinguish μ-like compounds from k-like ones. In comparison, mammals are able to distinguish μ-like compounds from k-like ones, perhaps suggesting that the discriminative effects of these two classes of drugs share a common mechanism of action in pigeons.[12] Differences in responses to opioid analgesics may be related to the proportion of subclasses of opioid receptors in different species,[4] but more research is necessary to determine opioid function in birds.

In chickens, initial studies using high doses (i.e., 200 mg/kg) of morphine produced analgesia in a toe-pinch test; however, more recent studies demonstrated morphine analgesia at much lower doses (5 to 30 mg/kg) using alternative nociceptive tests.[13] Chickens can be trained to associate color with the presence of analgesics in their food. Chickens (both healthy and lame) selected food with the highest dose of morphine when given three choices (i.e., food with 8.6, 49, or 430 mg/kg of morphine).[14] Chickens without lameness may have showed an obvious preference for morphine because of its euphoric effect.[14] In domestic fowl, morphine can produce either hypo- or hyperalgesia during thermal and chemical nociceptive tests. Genetic factors play an important role in determining sensitivity to opioid analgesic effects. Hyperalgesia displayed in domestic fowl is strain dependent, naloxone sensitive, and mediated primarily by μ-receptor activation at CNS loci.[15]

Buprenorphine is a partial agonist that binds readily to μ receptors and has some k-antagonist properties. Being a partial agonist, it does not induce the same degree of effect as a full agonist, such as morphine, and is effective only for treating mild to moderate pain. Buprenorphine has reportedly been clinically effective in birds,[2] but in African grey parrots, large doses produced no significant analgesic effect.[4,5] With very high doses, however, there may be a reduced analgesic effect mediated by stimulation of μ-opioid receptors.[16]

Butorphanol is a weak antagonist at the μ receptor but a strong agonist at the k receptor and is used commonly in small and large animal anesthesia for premedication and analgesia. In mammals, butorphanol produces analgesia in a dose-dependent manner with fewer respiratory-depressant effects compared with morphine. In parrots, butorphanol (1 mg/kg) administered during isoflurane anesthesia decreased the amount of isoflurane required during application of a painful stimulus (called *isoflurane-sparing effect* or *reduction in minimum anesthetic concentration*) by 25% in cockatoos and 11% in African grey parrots, but the change was not significant in blue-fronted Amazon parrots.[11,17] However, care should be taken when interpreting this kind of information because isoflurane can be spared through sedation rather than analgesia.[8] Another study was unable to demonstrate a reduction in the amount of halothane required to anesthetize turkeys during surgery with the addition of butorphanol (0.1 mg/kg), but birds treated with butorphanol had fewer responses to noxious stimuli than did controls.[18] Butorphanol (1 mg/kg) significantly increased the threshold to electrical stimuli in half of the conscious African grey parrots tested.[5]

Table 1. Opioids for Controlling Avian Pain[a]

Drug	Dose (mg/kg)	Route	Comments	Species	Reference(s)
Buprenorphine	0.01–0.05	IM	It was effective for 8–12 hr.	Psittacines Raptors Pigeons Waterfowl	Clyde[2], Beynon[37]
	0.1	IM	There was no effect in African grey parrots.	Psittacines	Paul-Murphy et al[5]
Butorphanol	0.1	IM	It did not decrease the halothane requirement. There were fewer responses to noxious stimuli than in control birds.	Domestic turkey	Reim and Middleton[18]
	0.2–2	IM	It was effective for 3–8 hr.	Not given	Clyde[2], Ritchie and Harrison[29]
	1	IM	It decreased the isoflurane requirement (median effective dose).[b] There were some effects on respiration.	Psittacines	Curro et al[11], Curro[17]
	3	IM	Readminister it every 2–4 hr.	Not given	Clyde[2]
	1–3	Not given	Some subjects became hyperalgesic when administered 6 mg/kg.	Hispanolian parrots	Paul-Murphy and Ludders[38]
	1–4	IM, PO	Readminister it every 2–4 hr. Administer it as needed, but do not exceed four times per day.	Not given	Clyde and Paul-Murphy[4]
Codeine	30	IM	It increased the jump latency to the thermal nociceptive stimulus.[b]	Chicken	Hughes[39]
Fentanyl	0.2	IM	It had little analgesic effect. There was an initial excitement phase in some subjects.	Cockatoo	Paul-Murphy and Ludders[38]
Morphine	0.1, 1, and 3	IV	There were dose-dependent isoflurane-sparing effects. There was a decrease in the minimum anesthetic concentration.[b]	Chicken	Concannon et al[7]
	2.5–30	IM, SC	Pain was relieved.	Not given	Ritchie and Harrison[29]
	30	IM	There was a diminished flight response to electric shock.[b]	Chicken	Bardo and Hughes[10]

[a]Doses are anecdotal as reported in the literature except where noted. Doses can vary dramatically between species and individuals as well as between healthy and debilitated individuals.
[b]Supported by experimental evidence.

Nonnarcotic Analgesics

Steroidal Antiinflammatories (Corticosteroids)

Corticosteroids may reduce pain by suppressing response to chemical, thermal, traumatic, or inflammatory injury through reduced fibroblast proliferation, macrophage response to the migration inhibition factor, sensitization of lymphocytes, and response to mediators of inflammation (**TABLE 2**). The combination of long-acting local anesthetics (i.e., bupivacaine) and corticosteroids has been shown to reduce postoperative discomfort in humans.[19] Betamethasone is a powerful steroidal antiinflammatory drug that reduces pain associated with degenerative hip disorders in adult male turkeys.[20] Intraarticular injection of sodium urate produces acute synovitis with inflammatory changes such as swelling, increased joint temperature, and sensitization of the joint capsule receptors lasting at least 3 hours. In this model, betamethasone decreased inflammation in chickens.[21]

Corticosteroids can alter response to endogenous or parenterally administered opioids. In rats, administering a potent synthetic corticosteroid such as dexamethasone can reduce antinociception induced by μ-opioid agonists while potentiating k-opioid agonists. Administering corticosteroids may therefore reverse stress-induced analgesia (i.e., that brought about by repeated stressful or painful stimuli) by acting at the μ receptor; thus these drugs should be administered with caution to stressed patients.[22] The risk of immunosuppression and other potential complications makes NSAIDs preferable in many situations.[4]

Table 2. Antiinflammatories for Controlling Avian Pain[a]

Drug	Dose (mg/kg)	Route	Comments	Species	Reference(s)
Steroids					
Betamethasone	0.1	IM	It has been used to treat degenerative hip disorders in turkeys and uric acid–induced arthritis in chickens.[b]	Turkeys Chickens	Duncan et al[20], Hocking et al[21]
Dexamethasone	0.05–2	IM, IV	Use with caution.	Most	Ritchie and Harrison[29]
Methylprednisolone acetate	0.5–1	IM, PO	It is not usually used for analgesia.	Most	Ritchie and Harrison[29]
Prednisolone	7	PO	Administer twice daily. It has been used as an antiinflammatory.	Most	Smith[40]
	5 mg in 2.5 ml of water	PO	Administer two drops twice daily. It has been used as an antiinflammatory.	Most	Smith[40]
Prednisolone sodium succinate	0.5–1	IM, IV	It has been used as an antiinflammatory. Use as a one-time treatment only.	Not given	Ritchie and Harrison[29]
Prednisone	6.7	PO	Administer twice daily, then use a decreasing dose. It is antiinflammatory and antipruritic.	Not given	Smith[40]
NSAIDs					
Aspirin (acetylsalicylic acid)	5	PO	Administer three times daily.	Not given	Ritchie and Harrison[29], Paul-Murphy and Ludders[38]
	325-mg tablet dissolved in 250 ml of drinking water	PO	Change the water three times daily because it alters the taste and smell of water.	Not given	Ritchie and Harrison[29]
Carprofen	1	SC	It alleviated chronic lameness.[b]	Broiler chickens	McGeown et al[27]
	2–10	IM	—	Not given	Bishop[41]
	2–4	PO	Administer two or three times daily. Higher doses may be needed for the oral route.	Not given	Paul-Murphy and Ludders[38]
	10	IM	—	Raptors Pigeons Waterfowl	Beynon[37]
Flunixin meglumine	1	IM	Administer once daily. There is potential nephrotoxicity.	Not given	Paul-Murphy and Ludders[38]
	2	IM	It has been used as an antiinflammatory.	Not given	Bishop[41]
	3–5	IM	—	Not given	Clyde[2]

	5	IM	The physiologic action appears to be approximately 12 hr. The duration of analgesia has not been assessed clinically.[b]	Mallard ducks	Machin et al[31]
	1–10	IV, IM	It can cause GI upset or ulceration. It can be nephrotoxic in some species (it is contraindicated in cranes). A high dose (10 mg/kg) can cause regurgitation and tenesmus in budgerigars.	Not given	Clyde and Paul-Murphy[4], Ritchie and Harrison[29]
Ibuprofen	5–10	PO	Administer two or three times daily. Use a pediatric suspension for small birds.	Not given	Paul-Murphy and Ludders[38]
Ketoprofen	2	IM	Administer one, two, or three times daily.	Not given	Bishop[43]
	5	IM	The physiologic action appears to be approximately 12 hr. The duration of analgesic effect has not been assessed clinically.[b]	Mallard ducks	Machin et al[31]
	5–10	IM	—	Raptors Pigeons Waterfowl	Beynon[37]
Meloxicam	0.1	PO	—	Not given	Paul-Murphy and Ludders[38]
Phenylbutazone	Not given	Topical	There was a mild reduction in pain-related behavior after partial beak amputation.[b]	Chickens	Ritchie and Harrison[29]
	3.5–7	IV, PO	Administer two or three times daily. GI ulcerations are possible. Do not use if hepatic, renal, or cardiac abnormalities exist. Do not administer SC or IM.	Psittacines	Clyde[2], Paul-Murphy and Ludders[38]
	20	PO	Administer three times daily. Do not administer SC or IM.	Raptors	Beynon[37]
Piroxicam	0.5	PO	Administer twice daily. It is used to treat chronic osteoarthritis.	Not given	Paul-Murphy and Ludders[38]

[a]Doses are anecdotal as reported in the literature except where noted. Doses can vary dramatically between species and individuals as well as between healthy and debilitated individuals.
[b]Supported by experimental evidence.

NSAIDs

Prostaglandins (PGs) are important local mediators of inflammation and pain and are also known to lower the activation threshold (i.e., response to pain at a lower stimulus level) to thermal, mechanical, and chemical stimulation (**TABLE 2**). NSAIDs control pain by inhibiting the cyclooxygenase (COX) enzyme that prevents PG production. Drugs that inhibit PG biosynthesis in mammals produce analgesia by decreasing inflammation at the site of injury and also through transmitter mechanisms in the spinal cord. PGs are involved in modulating avian pain responses, and physiologic mechanisms involving PGs are similar to those described in mammalian models.[23]

PG synthesis is mediated by one of two isoforms of COX enzymes: COX-1 and -2. The COX-1 enzyme is constitutive (i.e., part of the normal enzyme complement of a cell) and present at relatively constant concentrations, whereas COX-2 is inducible and concentrations of it increase in response to a stimulus. COX-1 produces PGs that have a cytoprotective function in tissue such as the gastric mucosa, kidneys, reproductive tract, and CNS. Similarly, thromboxane production in platelets is a COX-1-mediated process. Until recently, NSAIDs were believed to have exerted their therapeutically beneficial effects primarily by inhibiting COX-2, whereas drugs that inhibit COX-1 were responsible for some of the toxic side effects, such as gastric ulceration, renal papillary damage, and extended clotting time.[24] Consequently, there has been a shift in focus to drugs that inhibit COX-2.[25] However, it appears that COX-1 contributes to the inflammatory process and that COX-2-selective inhibitors may not be as efficacious as mixed inhibitors in their antiinflammatory actions. Both COX-1 and -2 are constitutively expressed in the CNS, and their relative expression varies depending on species.[24] Renal perfusion in hypovolemia is supported by PGs, but studies indicate that both COX-1 and -2 are present in the kidneys of some species.[25]

It is likely that both COX-1 and -2 are important in antinociception, but more research is necessary to distinguish their effects. Flunixin, ketoprofen, and carprofen have COX-1 and -2 actions,[26] and it is well recognized that these NSAIDs are capable of producing potent analgesia in both mammals[24] and birds.[27,28] Recommended doses of flunixin range from 1 to

10 mg/kg,[29] but no experimental data are available to confirm analgesia at low doses. In addition, flunixin administered to parrots did not produce an isoflurane-sparing effect,[17] but NSAIDs have not been shown to reliably reduce the inhaled anesthetic requirement in any species.[6] Chickens were able to maintain their pretrimming feed intake levels over the first 24 hours after phenylbutazone was applied to their beaks,[28] and this was longer than in untreated birds. Lame chickens preferentially selected food with carprofen at three doses (3.4, 34.3, and 343 mg/kg) rather than food without analgesics, and a dose of 1 mg/kg of carprofen raised pressure thresholds (i.e., decreased response to pressure) for at least 90 minutes after a subcutaneous injection.[14] In another study,[27] carprofen increased the speed and walking ability of rapidly growing broiler chickens with chronic lameness. Other NSAIDs have been used in birds with some success, although renal toxicity and gastrointestinal (GI) effects have been noted in some clinical cases. Flunixin meglumine appears to produce more adverse effects in birds than do other NSAIDs.[4]

Pharmacokinetic studies with broiler chickens indicate that peak plasma levels of carprofen are reached 1 to 2 hours after a subcutaneous dose.[27] Unfortunately, pharmacokinetic data cannot be extrapolated between species[24] and plasma levels of NSAIDs likely do not reflect physiologic or pharmacologic activity because NSAIDs contain weak acids, are highly protein bound, and tend to accumulate in areas of inflammation.[30] In addition, NSAIDs block access of arachidonic acid to its binding site on the COX enzyme, thus preventing conversion to thromboxane B_2. Consequently, thromboxane B_2 may be used to estimate the length of NSAID action. In mallard ducks, flunixin (5 mg/kg) and ketoprofen (5 mg/kg) suppressed thromboxane B_2 levels for up to 12 hours, suggesting that their physiologic action may be that long,[31] but further studies are necessary.

α_2-Adrenergic Agonists

As in mammals, sensitivity to noxious stimuli in birds is susceptible to adrenergic modulation. α_2-Adrenergic agonist activation can produce sedation, anxiolysis, and analgesia as well as reduce the minimum alveolar concentration of inhalant anesthetics. Because α_2-adrenergic agonists (e.g., xylazine, medetomidine) can cause muscle tremors, respiratory depression, and movement (in response to noise) in birds,[32] these drugs are often combined with ketamine. Disadvantages of α_2-adrenergic agonists include hypertension following intravenous bolus injections, hypotension, bradycardia with partial atrioventricular block,[33] dose-dependent hypothermia resulting from decreased thermogenesis, increased postoperative fluid requirements, sedation, and respiratory depression. Although including α_2-adrenergic agonists can be useful in premedication for balanced anesthesia during painful procedures, α_2-adrenergic agonists are not usually administered after surgery. Atipamezole is a highly potent, specific, competitive α_2 antagonist of centrally and peripherally located α_2 adrenoceptors that quickly relieves unwanted side effects, but administration also reverses analgesia.[32]

Ketamine

Ketamine is a dissociative anesthetic and an *N*-methyl-d-aspartate glutamate receptor antagonist.[34] Ketamine is often combined with sedatives such as α_2-adrenergic agonists and benzodiazepines for premedication or general anesthesia for minor procedures. At low doses, ketamine can enhance analgesia by preventing *N*-methyl-d-aspartate receptor-mediated sensitization in the CNS. Therefore, low-dose ketamine can be useful for preemptive analgesia in major surgeries and also for postoperative analgesia because it may abolish hypersensitivity once it is established.[34] Although ketamine prevents sharp, superficial pain effectively, it does not control visceral, dull pain. Thus analgesia produced by ketamine alone is not adequate for laparotomy or orthopedic surgery.[34]

Local Anesthetics

Local anesthetics (i.e., lidocaine, bupivacaine) function by blocking ion channels, thereby preventing generation and conduction of pain impulses (**TABLE 3**). Local anesthesia before tissue trauma can reduce postoperative pain significantly because it prevents nociceptor sensitization and therefore avoids central changes secondary to activation of pain pathways.[1] Local nerve blockade before nerve transection in amputation can decrease the prevalence of "phantom limb" pain in humans.[1] Although local anesthesia is sufficient for pain relief, it does not reduce stress that may be induced by physical restraint and handling of an awake bird. Sedation or general anesthesia should also be considered during stressful or prolonged procedures.

Birds may be more sensitive than mammals to the toxic effects of local anesthetics because lower doses (2.7 to 3.3 mg/kg) of bupivacaine in birds produce toxic effects[35] compared with higher doses (3.5 to 4.5 mg/kg) in dogs.[36] It is recommended that the lidocaine dose not exceed 4 mg/kg in birds because seizures and cardiac arrest can result from overdosing. However, chickens receiving higher doses of bupivacaine (2.7 to 3.3 mg/kg) showed signs of toxicosis (e.g., recumbency with outstretched legs, drowsiness) and distress immediately after injection.[35] Other possible side effects of

local anesthesia include depression, drowsiness, ataxia, nystagmus, muscle tremors, and hypotension.[36]

The length of action of local anesthetics in birds is unknown. In mammals, lidocaine is shorter acting (60 to 120 minutes) than bupivacaine (240 to 360 minutes).[36] In domestic fowl, bupivacaine has produced effective analgesia in two pain models. Chickens that had bupivacaine applied to their beak stumps after amputation were able to maintain their pretrimming feed intake levels over the first 4 hours.[28] In uric acid-induced, hock-joint pain, intraarticular bupivacaine (2 mg/kg) increased feeding, pecking, and standing behaviors while resting declined proportionally, and the behavior of treated birds was indistinguishable from that of the control group.[35]

Table 3. Local Anesthetics for Controlling Avian Pain[a]

Drug	Dose (mg/kg)	Route	Comments	Species	Reference(s)
Bupivacaine	2	Intraarticular	The intraarticular sodium urate injection has been associated with musculoskeletal pain.[b] Toxicosis occurs at >2.5 mg/kg.	Chickens	Duncan et al[20]
Bupivacaine (50:50 mixture with dimethyl sulfoxide)	Not given	Topical	There was reduced pain-related behavior after partial beak amputation.[b]	Chickens	Glatz et al[28]
Lidocaine	Not given	SC (infiltration)	The dose should not exceed 4 mg/kg.	Not given	Paul-Murphy and Ludders[36]

[a]Doses are anecdotal as reported in the literature except where noted. Doses can vary dramatically between species and individuals as well as between healthy and debilitated individuals.
[b]Supported by experimental evidence.

Conclusion

Pain perception in birds is likely analogous to that in mammals. Invasive and painful procedures should always be accompanied by appropriate analgesia and anesthesia. When choosing an analgesic for a bird, practitioners should consider the level of pain and treat it as they would in mammals. Pharmacologic intervention is important, but physical, environmental, and behavioral management should not be overlooked.[3] Although avian pain management is in its infancy, research and clinical studies demonstrate the benefit of using opioids, steroidal antiinflammatories, and NSAIDs as well as other analgesics such as α_2-adrenergic agonists, ketamine, and local anesthetics. Assessing analgesic efficacy is extremely important because the dose and choice of analgesic may vary widely among species. The information in this article is meant as a guide rather than a recommendation for managing pain in birds. There is clearly a need for further clinical investigations, and both successes and failures should be reported in the veterinary literature to expand the limited information available.

*A companion article on the physiology and evaluation of avian pain appeared in the February 2005 issue.

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