CE

Avian Pain: Physiology and Evaluation

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ABSTRACT:

Information on avian pain and analgesia is limited. However, an understanding of pain physiology and associated behavior specific to avian species can help practitioners recognize pain and treat it effectively. This article provides background information on peripheral and central processing of pain information in birds. Evaluation of pain and changes in behavior are described, and stress-induced analgesia and changes in attention that produce analgesia are also discussed.

Birds represent the most abundant and diverse class of air-breathing vertebrates, including approximately 9,700 species. However, practitioners encounter only a few species from select orders, usually including Anseriformes (e.g., ducks, geese, swans), Columbiformes (e.g., pigeons, doves), Galliformes (e.g., chickens, turkeys, pheasants), ratites (e.g., ostriches, emus), Falconiformes (diurnal birds of prey; e.g., eagles, hawks, falcons), Strigiformes (e.g., owls), Psittaciformes (e.g., parrots), Piciformes (e.g., toucans), and Passeriformes (e.g., perching birds, including finches and canaries). In mammals, most information about pain has been obtained from laboratory, companion, and farm animals, and there is a wide range of responses to painful stimuli among mammalian species, breeds, and individuals. Information about pain in birds is limited and has been derived from only a handful of species from the following orders: Anseriformes,[1] Columbiformes,[2] Galliformes,[3–5] and Psittaciformes.[6,7]

It is generally accepted that birds perceive pain similarly to mammals. Birds have neurologic components to respond to painful stimuli and endogenous antinociceptive (antipain) mechanisms to modulate pain,[8] and some pharmacologic agents administered for pain in mammals also modulate pain pathways and behavioral responses to painful stimuli in birds.[7,9,10] Pain perception allows animals to minimize their exposure to potentially harmful stimuli. Birds often do not indicate pain in an obvious manner because species that may be preyed on are less likely to display overt pain-associated behavior that may attract attention from predators.[11] Considerable variation in behavioral responses to pain may occur among avian species, breeds, strains, or individuals, and there is no reliable or universal indicator of pain.[12] Most practitioners are able to recognize acute severe pain, but chronic pain may go undetected, especially if practitioners are unfamiliar with the normal behavior of the species. Therefore, it is advisable to treat for pain when dealing with any condition expected to cause pain, especially if known to be painful in humans (see box on page 99).

This article provides information on pain physiology and related behavior that may improve practitioners' understanding of pain mechanisms and, therefore, knowledge of appropriate analgesic therapies in birds. This article is based on the available literature on avian pain, but interpreting experimentally produced measures of pain reflexes can be problematic because they may not represent a measure of true pain behavior.[13]

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Perspectives on Pain

A consensus statement from the Cross-Species Approach to Pain and Analgesia Workshop stated "Animals feel pain" with the following caveats[a]:

Although it is unclear at this time at what taxonomic level nociception is associated with pain and whether all species, including humans, feel pain with the same qualities and intensities, operationally, vertebrates and some invertebrates feel pain.

If researchers were to step back from the neurophysiologic evidence that birds feel pain and take a more common-sense approach, it would be clear to them that birds must feel pain in order to survive in their environment. Unquestionably, the experience of pain must be aversive and unpleasant to motivate a bird or any other animal to change its behavior. In plain language, pain hurts regardless of the species. Because veterinarians do not know what animals are feeling when they are injured, we must extrapolate from the only species that can directly communicate its feelings to us—namely humans. Veterinarians are obligated to advocate on behalf of animals; therefore, it is appropriate to assume that if an animal has been injured, it is experiencing pain. Rather than wait until we have been convinced beyond a shadow of a doubt that surgery or trauma has caused pain, it is incumbent on veterinarians to assume that pain is present after injury unless proved otherwise.

What about the argument that veterinarians can never know whether a bird is experiencing pain following an injury and that making such an assumption puts us at risk of anthropomorphism? If we hold up the experience of human pain as the "gold standard," we find that it is not so easy to measure and assess human pain even though we are all the same species and we can ask human patients what they are experiencing. In fact, some chronic painful conditions in humans (e.g., fibromyalgia) are poorly understood, diagnosed, and treated and represent an enormous unmet medical need. Pain is an individual sensory and emotional experience, which means that it may not be possible for one person to know for certain the pain of another person. This uncertainty is the nature of pain and does not mean that pain is not real. Similarly, just because an animal (or a bird in the case of this review) may have a different pain experience from that of humans does not mean that the animal's pain does not exist. Veterinarians are challenged to determine whether avian patients are suffering from pain and what the most effective therapy would be. Dr. Machin has done a marvelous job of focusing attention on the important questions regarding pain in birds (namely How do we diagnose and treat it?) rather than being distracted by the question of whether birds actually feel pain. The task before us is not easy, but it is certainly worthy of all of our efforts.

This review points to the complexity of pain and should encourage biomedical scientists and veterinarians to collaborate on ways to further our understanding of the recognition and treatment of avian pain. A group of veterinarians, veterinary nurses, and allied professionals who were troubled that painful animals are still inadequately treated by our profession formed the International Veterinary Academy of Pain Management (IVAPM) in September 2003. The IVAPM is a multidisciplinary organization dedicated to improving the identification, prevention, and relief of pain and distress in all animals cared for by the veterinary profession. The intent of the IVAPM is to provide a forum for all parties interested in animal pain to work together to improve the understanding of pain and raise the standard of care for treating animals in pain. For more information, visit the IVAPM online at www.cvmbs.colostate.edu/ivapm or send an email to ivapm@colostate.edu.

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[a]Paul-Murphy J, Ludders JW, Robertson SA, et al: The need for a cross-species approach to the study of pain in animals. *JAVMA* 224:692–697, 2004.

PAIN PATHWAYS

A noxious stimulus is one that may damage tissue, and a receptor sensitive to a noxious or potentially noxious stimulus is a nociceptor. The physiology of pain involves two processes[13]:

- A peripheral process involving detection and transmission of information concerning potential tissue damage
- A central process governing the cerebral response to this information

Peripheral Nervous System

Three types of nociceptors have been identified in birds:

- High-threshold mechanothermal
- Mechanical
- Thermal

High-threshold mechanothermal nociceptors are polymodal pain receptors because they have more than one function.[12] They respond to temperatures above

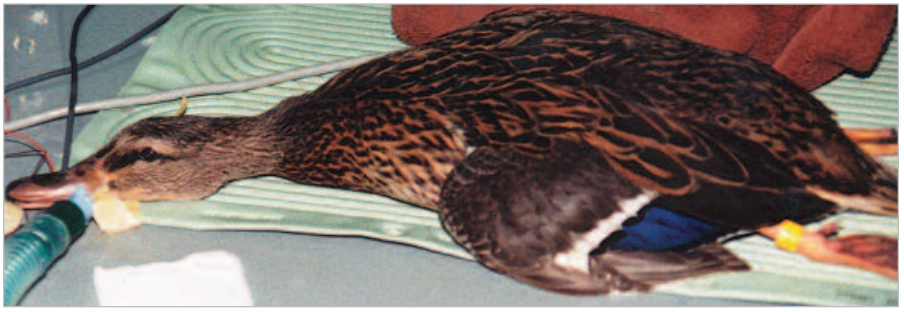

Figure 1. **Isoflurane-anesthetized mallard duck undergoing assessment for ketoprofen analgesia.**

104°F (40°C) and mechanical stimulation and have been identified in pigeons,[2] waterfowl,[1] and chickens.[12] Impulse conduction in these nociceptors is very slow and comparable with that of mammalian unmyelinated C fibers, which arise from cutaneous free-nerve endings that transmit pain. Increasing the magnitude of the stimulus increases the number of responses. Some fibers show a continuous response up to the highest temperature tested (132.8°F [56°C]), whereas other fibers show a clear peak in response at a lower temperature, and increasing stimulus intensity beyond this temperature results in a reduced response.[14]

Mechanical nociceptors are likely equivalent to myelinated Aδ and unmyelinated C fibers originating from cutaneous free-nerve endings in mammals; they have been reported in chickens[15] and waterfowl.[1] The receptive fields of high-threshold mechanical nociceptors are similar to those of polymodal nociceptors, but some have larger receptive fields. Increasing the stimulus strength increases the response, which may be either linear or exponential.[14]

Thermal nociceptors without mechanical sensitivity respond in a manner similar to polymodal nociceptors.[16] They are either Aδ or C fibers and have been reported in pigeons[2] and chickens.[12] These receptors appear to be less sensitive to cold than are corresponding receptors in mammals, but the threshold of heat nociceptors tends to be higher in avian species compared with that in mammals. This is not surprising because body and skin temperatures are higher (105.8°F to 107.6°F [41°C to 42°C]) in birds[2]; however, when comparing physiologic responses of nociceptors in chickens with those in mammals, discharge patterns and receptive field size are very similar.[15]

Tissue injury and acute pain can affect both peripheral and central nervous systems in mammals and alter sensitivity to subsequent stimuli. This sensitization may be characterized by one or more of the following[13]:

- Pain response at a lower stimulus level (i.e., lower activation threshold)
- Increased response to noxious stimuli
- Quicker response to pain (i.e., shorter response latency)
- Longer response to stimulation (i.e., persistent pain)
- Increased response to a given stimulus intensity or spontaneous activity
- Spread of pain to uninjured tissue and hyperalgesia (i.e., increased response to a stimulus that is normally painful)

Central sensitization occurs when the threshold required to activate dorsal horn neurons decreases following nociceptor activity. Recruitment of other nerves as a result of increased output or increased excitability causes changes in receptive field properties.[13]

Peripheral sensitization results when tissue damage causes a drop in pH and release of inflammatory mediators (e.g., histamines, bradykinins), to which small unmyelinated C fibers are sensitive. The nociceptors become responsive to a wider range of stimuli or respond abnormally to sustained stimuli of another type.[13] C fiber activity may be further altered by prostaglandins (PGs) in both mammal and avian species

Birds have neurologic components to respond to painful stimuli; therefore, appropriate analgesia should be provided during painful procedures.

by lowering activation threshold during tissue injury and inflammation.[17] In addition, eicosanoids and prostaglandin E_1 and E_2 sensitize small-diameter sensory nerve fibers to thermal and mechanical stimulation. Substance P appears to be an important mediator of components of the inflammatory response, and its release results in peripheral vasodilation and further sensitization of the C fiber's peripheral ending.[18] In avian sensory neurons, exposure to prostaglandin E_2 produces a dose-dependent increase in the release of substance P through inward flux of calcium.[17]

Central Nervous System

As in mammals, pain signals in birds are transmitted from receptors to several areas of the midbrain and forebrain by multiple ascending spinal pathways. The spinal cord dorsal horn in mammals has 10 layers (laminae) that can be distinguished based on cell size and distribution, whereas chickens have six laminae.[15] Nociceptive information is transmitted to the lamina I and outer lamina II of the dorsal horn via Aδ and C primary afferent fibers.[19] In birds, the distribution of the neurons is similar to that of the nociceptive spinothalamic tract cells in monkeys and cats.[18] Neurons receive input from substance P–containing axon terminals, which also appear to play an important role in neurotransmission of pain in birds.[18] One study suggests that melatonin may also be important in transmitting sensory information such as pain signals.[19] As in mammals, α_2-receptors may also be important in nociception. Distribution of these receptors has been mapped in chickens and closely resembles patterns seen in mammals.[20] The similarities

between avian and mammalian pain pathways lend further support to the hypothesis that birds are capable of perceiving pain.

The central nervous system plays a major role in processing all noxious information. As in mammals, endogenous opioid systems appear to modulate central processing in birds.[8] In mammals, several endogenous opioids, including endorphin and enkephalin, act on the opioid receptors μ, κ, and δ to inhibit pain. Birds have opioid receptors in similar proportions to those in humans.[21] Opioid receptors are detectable in chick embryos from 10 days of age in vivo and are concentrated in areas thought to play key roles in sensory input processing and memory.[22] Distribution of β-endorphin and enkephalin-like immunoreactivity in the avian telencephalon is similar to that in the mammalian telencephalon,[8] indicating that the binding sites are similar. Evidence demonstrates that the endogenous opioid system plays an important role in pain modulation and perception because male broiler chickens with degenerative joint disease affecting the hip walked more slowly after receiving an injection of naloxone (an opioid antagonist).[23]

Figure 2. Mallard duck with a catheter for frequent blood sampling during a pharmacodynamic study evaluating thromboxane levels after NSAID treatment.

EVALUATING PAIN AND PAIN-ASSOCIATED BEHAVIOR

Recognizing pain and anxiety in animals is critical for appropriate analgesic selection and pain relief. Experimental evidence shows that there is a relationship between activation of nociceptors and behavioral evidence of pain.[12,24] Nociceptive stimuli that have been used to investigate pain in birds include electric shock,[25,26] comb pinch,[27] feather removal,[3] cutaneous thermal stimulation,[5,26–28] injection of intraarticular sodium urate[29] or formalin,[30] and oral presentation of algogenic (pain-producing) substances.[31] Although there are no reliable or universal indicators of pain, birds tend to respond to noxious stimuli with a fight-or-flight response (i.e., escape reactions, vocalization, excessive movement)[3] and/or conservation–withdrawal responses (i.e., no escape attempts or vocalization and immobility).[12,27] The conservation–withdrawal response after noxious cutaneous thermal stimulation is typified in chickens by crouching and immobility (i.e., a crouched posture, with the head drawn into the body and the eyes partially or fully closed).[12,32]

In chickens, acute pain (such as feather removal) is usually characterized by wing flapping and/or vocalization, decreased head movement, increased heart and respiratory rates, and increased blood pressure.[3] Electric shock or comb pinch also produces acute pain, resulting in active avoidance behavior involving vigorous escape attempts (i.e., jumping and wing flapping) with some vocalization.[26] In comparison, inappetence, inactivity, and a "puffed-up" appearance are often demonstrated when birds are exposed to prolonged pain.[3] Prolonged pain by continual feather removal tends not to produce an exaggerated escape response; instead, birds crouch in an immobile state. Changes in blood pressure and electroencephalogram arousal immediately after feather removal suggest the presence of a pain sensation.[32] Subsequent monitoring of the electroencephalogram shows characteristic high-amplitude low-frequency activity similar to that seen during sleep or catatonic states.[32]

As in mammals, endogenous opioid systems appear to modulate central processing of pain information in birds.

Immobility is a complex behavioral reaction to a painful[31,32] and/or fear-inducing (restraint) stimulus.[32,33] Procedures that increase fear prolong the immobility reaction, whereas procedures that reduce fear attenuate

the response.[32] Immobility may be an evolutionary antipredator strategy to prevent further damage produced by struggling and to allow escape, if possible.[3] The functional significance of changing behavior from active escape to crouching immobility may also be related to learned helplessness. This behavioral pattern develops when an animal experiences traumatic events that are aversive and continue to occur despite attempts by the animal to reduce or eliminate them.[3] Studies in mammals suggest that learned helplessness may also produce some analgesia[32] (Figures 1 and 2).

Thermal trauma, which occurs during partial beak amputation in chickens, involves cutting and cautery, resulting in full-thickness burns. Pain experienced at amputation results from massive injury discharge in the nerve fibers, lasting approximately 15 seconds. However, after the initial discharge, no abnormal pattern of response to cutaneous stimulation was detected in any of the sensory receptors in the beak stump for 4.5 hours after amputation. The absence of change in peripheral neural input following beak amputation may suggest an absence of pain during this period. A similar pain-free period has also been observed in humans following full-thickness burns. Normal beak use after amputation occurs for approximately 6 hours after amputation, but by 24 hours after amputation, chickens were less mobile, were unwilling to peck at the environment, and had decreased food and water intake.[34] More detailed studies of beak use after amputation demonstrated guarding behavior, hyperalgesia, and significant reductions in environmental pecking, preening, beak wiping, and head shaking, all of which persisted for 6 weeks after surgery.[14] In one study, inactivity was observed as long as 56 weeks after surgery. These responses as well as altered food intake, reduced weight gain, and egg production[34,35] provide evidence of chronic pain in birds. Decreased activity is also common in humans with chronically painful conditions.

In addition to behavioral evidence, anatomic and physiologic evidence support the presence of chronic pain following partial beak amputation. Adjacent to the scar tissue, damaged and regenerating nerve fibers formed extensive neuromas.[16] Electrophysiologic recordings from nerve fibers innervating these neuromas were abnormal for trigeminal afferent fibers. The most characteristic abnormality was the presence of abnormal spontaneous neural activity in the trigeminal nerve from the beak stump 5 to 83 days after initial amputation.[16] Neural activity arising from trigeminal neuromas was similar to that reported in experimental neuromas in rats, mice, and cats. Studies on peripheral nerve injury and subsequent neuroma formation in mammals have suggested that abnormal activity from regenerating axons is implicated in postamputation stump pain.[16]

Response to noxious stimuli can vary greatly from species to species. Electric shock and thermal tests have been used effectively in gallinaceous birds, but one study of conscious parrots (i.e., African grey parrots *Psittacus erithacus erithacus* and *Psittacus erithacus timneh*) found that the large variation among responses prevented meaningful quantitative assessment of the temperature threshold.[26] In comparison, it was possible to identify when some African grey parrots became aware of the electrical stimulus because they would look down at their foot or chew on the wire. The amount of stimulus that caused the bird to lift its foot could be measured reliably, and response was attenuated with opioids.[7]

In contrast to domestic mammals, birds indicate pain in a less obvious manner and tend to respond to noxious stimuli with a fight-or-flight and/or conservation–withdrawal response that may be difficult for practitioners to interpret.

STRESS-INDUCED ANALGESIA

A wide variety of repeated stressful or painful stimuli can induce temporary reductions in responsiveness to noxious stimuli (called *stress-induced analgesia*).[32] Stress from social isolation can also alter pain perception in species that normally live in groups.[36] The two most common indications of social separation in domestic chicks are distress vocalizations and stress-induced analgesia.[37] Birds experiencing less stress (i.e., those held and tested in familiar large pens) showed significantly less pain-coping behavior compared with birds tested in novel pens.[36,37]

Stress-induced analgesia caused by social separation is poorly understood. In mammals, two theories describe the role of endogenous opioids in social separation. One theory (social separation–opioid stimulation) suggests that opioid system activity is stimulated by stressful experiences such as social separation, whereas another theory (social separation–opioid withdrawal) suggests the opposite—social isolation places an animal in a state similar to opioid withdrawal. Birds that become separated from conspecifics elicit distress vocalizations in an attempt to reestablish social contact. This isolation causes a state of endogenous opioid withdrawal, leading to disinhibition of distress vocalizations, whereas the presence of social companions stimulates the release of endogenous opioids, inhibiting vocalizations.[38] Opioid agonists tend to decrease distress vocalizations, whereas opioid antagonists increase separation-induced distress vocalization. However, when morphine was administered to isolated chicks, there was no change in response to thermal nociception. Adrenergic, cholinergic, dopaminergic, GABAergic, and serotoninergic manipulations produce only modest effects on distress vocalization. These results suggest that some separation-stress behaviors are mediated by opioid systems (i.e., distress vocalization), whereas others are mediated by nonopioid systems (i.e., stress-induced analgesia and hyperthermia).[37,38]

Benzodiazepine agonists can modulate stress in many animal models and thus influence the results of nociceptive tests while having no effect on nociception itself. As with morphine, the benzodiazepine agonist chlordiazepoxide reverses distress vocalizations in chick social separation, but unlike morphine, chlordiazepoxide also reverses stress-induced analgesia. It appears that benzodiazepine is less behavior-specific than morphine in modulating separation-stress behaviors in chicks.[38] Although stress may produce analgesia in some situations, stress alone should not be considered necessary or sufficient to induce analgesia.

ANALGESIA PRODUCED THROUGH CHANGES IN ATTENTION

Pain associated with trauma and disease can be chronic and often involves inflammation. Behavioral responses to pain are complex and can be influenced by changes in an animal's attention (level of awareness). In chickens, changes in attention can significantly suppress

pain and reduce lameness during an experimentally induced chronic pain stimulus (i.e., injection of sodium urate into the joint, which mimics articular gout).[39] Hypoalgesia can be produced by diverting attention in situations designed to increase feeding motivation or motivation to explore.[24] Complete analgesia or marked hypoalgesia was observed in birds deprived of food for 16 hours and then given access to food following sodium urate injection. This could be completely reversed by naloxone, suggesting that the analgesia may be opioid-mediated. When introduced to novel surroundings, birds behaved as normal; alert birds and attentional mechanisms are preoccupied with exploring a new physical and/or social environment.[39]

Distraction and attention-focusing strategies have been used to help humans cope with chronic low-level pain. Coping is based on the cognitive action of switching attention; thus when patients were fully engaged in a task, they did not process pain at the same time. The absence of pain-related behavior does not necessarily indicate an absence of pain. Expression of pain in birds, similar to that in humans, can be altered by the motivational state of the individual, but the analgesia experienced is likely only temporary.[39]

Although the precise mechanism is unknown, there is evidence that the peripheral nervous system plays a significant role in inflammation.[40] Some evidence suggests that changes in attention resulting in reduced pain may also directly influence inflammation, leaving only the general tissue reaction.[24] Studies on the neural activity in the medullary dorsal horn of monkeys suggest that attention-dependent changes in sensory discrimination and affective components of pain are mediated at the early stages of sensory processing. If similar changes occur while processing nociceptive information at the spinal level, the activity of the peripheral nervous system may also be affected.[24] Reduced pain perception and inflammation were demonstrated following attentional changes in chickens.[24] However, more research is necessary before clear conclusions can be drawn.

Watch for an upcoming article on controlling avain pain.

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1. Which of the following is evidence of pain perception in birds?

a. Like mammals, birds have the appropriate neurologic components.
b. Birds have endogenous antinociceptive mechanisms to modulate pain.
c. Pharmacologic agents administered for pain in mammals also modulate pain pathways in birds.
d. Birds have appropriate behavioral responses to painful stimuli.
e. all of the above

2. Which statement regarding nociceptors in birds is incorrect?

a. Birds have three types of nociceptors that respond to mechanical and thermal stimulation.
b. Avian discharge patterns and receptive fields are very similar to those in mammals.
c. Sensitization of avian nociceptors can result in persistent pain.
d. High-threshold mechanical nociceptors originate from encapsulated nerve endings.
e. PGs can play a role in sensitizing peripheral nociceptors.

3. Sensitization is characterized by a

a. higher threshold of activation.
b. decreased response to noxious stimuli.
c. shorter response latency.
d. decreased response to a stimulus.
e. perception of low-level pain.

4. Which statement regarding pain perception in birds is incorrect?

a. Birds have endogenous opioid receptors that modulate central processing of pain.
b. Substance P is important in neurotransmission of pain.
c. Pain information is transmitted to laminae I and II via primary afferent fibers.
d. PGs are important in pain modulation.
e. none of the above

5. A polymodal pain receptor

a. rapidly conducts action potentials.
b. transmits pain signals through myelinated fibers.
c. responds to both mechanical and thermal stimuli.
d. responds to increased stimulus magnitude with a decreased response.
e. adapts rapidly at low stimulus intensities.

6. Which statement(s) regarding endogenous opioids in birds is correct?

a. Endogenous opioid systems modulate central processing of noxious information in birds.
b. Endogenous opioid receptors are found in chicken embryos.
c. Endogenous opioids can be effectively antagonized by naloxone.
d. all of the above
e. none of the above

7. Conservation–withdrawal responses to pain stimuli are

a. characterized by vocalization and attempts to escape.
b. characterized by immobility and no attempt to escape.
c. most commonly associated with acute pain.
d. not associated with a painful stimulus.
e. a and c

8. Partial beak amputation in domestic chickens resulting in thermal trauma

a. can be followed by a pain-free period lasting several hours.
b. disrupts normal beak use for a few minutes.
c. has little impact on food and water intake.
d. is not associated with chronic pain.
e. none of the above

9. Which nociceptive test has poor accuracy in African grey parrots?

a. electric shock
b. injection of intraarticular sodium urate
c. feather removal
d. thermal stimulation
e. formalin injection

10. Stress-induced analgesia can be demonstrated in birds when they

a. experience repeated painful stimuli.
b. are separated from social companions.
c. are exposed to novel conditions.
d. b and c
e. all of the above