CONSIDERATIONS IN CARE FOR AVIAN HEAD TRAUMA

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Anatomy and Physiology of the Avian Head

The avian skull is designed to support and protect cranial structures with a minimum of weight. Bones in the skull are extensively fused and, because the bird does not have to bear the weight and stress of chewing teeth, the jaws are designed as lightweight struts that support the beak. In addition, because birds are heavily dependent on visual cues, there are large areas in the skull for ocular placement.

The orientation of the brain within the skull varies by bird group. The brain in avian species with long narrow skulls, such as herons, is oriented horizontally, but most birds have almost vertically oriented brains. The orbits of the eyes take up a large proportion of the midline space leaving little room for the brain in most bird skulls. As an extreme example, the brain of the American woodcock is rotated close to 120° off the horizontal axis and lies almost upside down in the skull with the foramen magnum pointing in a forward direction. This anatomic configuration allows the woodcock eye to be positioned at the extreme posterior aspect of the skull, thus enabling the bird to probe for invertebrate food deep in the mud while simultaneously staying alert for aerial predators.

In comparison to mammals, birds have markedly enlarged optic lobes, which serve an associative and coordinating function for visual information. In fact, in some species, the diameter of the optic nerve is larger than that of the cervical spinal cord. Most birds have a poor sense of smell and this is evidenced by small olfactory lobes. The cerebral hemispheres are smooth, and both the corpus striatum and the cerebellum are large, reflecting the large role that instinctive behavior and the need for muscular coordination play in these species. As in mammals, the majority of the cranial nerves is associated with the brainstem.

Because there are many functions associated with the avian head, a traumatic brain injury (TBI) has the potential to cause a wide variety of physiological abnormalities. Physiological functions that may be affected with TBI include vision, hearing, equilibrium, olfaction, gross and fine motor control, among many others.

What Happens with Traumatic Brain Injury?

The magnitude of initial impact to the brain will determine the severity of the primary brain injury. Oftentimes, a contrecoup injury is sustained after the initial impact to the avian head. Physical trauma may include dural tears, epidural or subdural bleeding, contusions, lacerations, and hematomas which can lead to diffuse axonal injury. Hypovolemic shock may be an indication of occult bleeding into the brain.
Secondary injuries to the brain are mediated by enhanced activity of the excitatory neurotransmitters, the generation of reactive oxygen species and the production of pro-inflammatory cytokines. These lead to further neuronal cell damage and death which, in turn, result in cerebral edema, increased intracranial pressure (ICP), compromise to the blood-brain barrier and alterations in cerebrovascular reactivity.

There are three major components to brain tissue. These include the brain parenchyma, arterial and venous blood supply and cerebrospinal fluid (CSF). Because the skull is a rigid compartment, any increase in volume in one of these components means that the volume of the other components must decrease. If this does not occur, then ICP will increase, resulting in compromise in cerebral perfusion and ischemia of brain tissue. Increases in ICP also result in increased CO₂ levels in the brain, an elevation in mean arterial pressure and reflex bradycardia.

**Diagnostic Modalities for Avian Head Trauma**

Many of the same diagnostic tools used in small or large animal medicine can be utilized in cases of avian head trauma. However, there are limiting factors due to differences in avian anatomy and physiological function from mammalian cases of TBI. Diagnostic tools include physical and neurological examination, various radiographic modalities, bloodwork, CSF analysis and electroencephalograms (EEG).

Human studies have shown that approximately 60% of patients with TBI have concurrent injuries to other major organs. This rate may be even higher in avian patients given the smaller size of their body in relationship to the traumatic incident. Consequently, a thorough physical examination to detect other injuries is essential. Airway, breathing and circulation must be addressed prior to any other abnormalities. It is important to recognize a bird in hypovolemic shock or with hypoxemia. Ideally, a minimum hematological database should include packed cell volume, total protein, blood glucose, and electrolyte levels. In addition, because the avian eyes occupy such a large proportion of the skull, ophthalmic trauma is often seen concurrently with head trauma. Thus, a thorough ophthalmic examination should be conducted during the physical examination.

Clinical signs of head trauma may include external evidence of bleeding and bruising, an alteration in mental status, development of ataxia or a degree of paresis, head tilt, nystagmus, tremors or other involuntary movements, and seizure activity. As in other species, ataxia or paresis can be due to injury to central nervous system (CNS) motor control functions. Head tilts and nystagmus result from an asymmetrical injury to the central or peripheral vestibular system. Intention tremors indicate cerebellar disease while general body tremors result from multifocal or diffuse CNS lesions.

The neurological examination begins with careful observation of the patient’s mental status. Is the bird lethargic or showing hyperactivity? A bird in recumbence may be painful, e.g. with an extremity fracture, weak, e.g. due to emaciation, or lethargic due to a neurological abnormality. Changes in attitude, such as decreased aggression in predatory species, may indicate a central or peripheral neurological issue. Normal coordinated movement requires functional cerebral,
cerebellar, vestibular and proprioceptive pathways and any disruption in these pathways will result in abnormal coordination. Lack of precise or finely tuned movements indicate cerebral dysfunction. Paraparesis or paraplegia result from some disruption in the neural pathways to the affected limb(s).

Cranial nerve (CN) assessment in birds can be accomplished through step by step review of nerve function. The ability to avoid visual obstacles indicates intact optic nerve and cranial pathways. Many birds do not respond to a menace response since this is a learned behavior so the absence of this response should not be interpreted as an indication of visual deficits. When attempting to menace a bird, care should be taken not to create air currents that can lead to a false menace reaction. If a true menace reaction is obtained, this can be interpreted as the bird having normal reaction initiation in the cerebellum and intact CN II and V pathways. While birds do have some striated muscle component to their irises, a normal pupillary light reflex (PLR) should be obtained in the bird if it is examined in a dark room and a bright light is used. Normal PLR requires intact CN II and III. The consensual reflex is not evaluated in birds due to complete decussation of the optic nerves and the fact that light can be seen across the spongy bony septum between orbits. CN II, IV and VI keep the globe in a normal position within the orbit and any abnormal positioning may be due to disruption in the corresponding nerve. Because there is also a striated muscle component to the nictitans in birds, a third eyelid prolapse does not directly indicate loss of sympathetic nerve innervation as is seen in Horner’s syndrome in other species. CN V is responsible for facial and eyelid sensation while CN VII produces the motor response to facial stimulation. CN V and VII also contribute to jaw position and function. CN VIII is involved in maintaining equilibrium and sensory perception of noise and damage to this nerve results in a spontaneous horizontal nystagmus with the fast phase away from the side of the lesion. Because the avian eye has relatively little movement within the bony orbit, nystagmus in birds is manifested by complete movement of the head/eye. Dysfunction of CN IX may be shown with signs of dysphagia and voice loss. Abnormalities of the vagus nerve (CN X) are subtle but may include regurgitation, voice change, increased heart rate and decreased gastrointestinal motility. CN XI provides general somatic efferent nerves to the skeletal muscles of the neck and dysfunction may result in poor neck movement. Lastly, CN XII innervates the tongue, tracheal and syringeal muscles so that dysfunction can result in deviation in the position of the tongue.

Postural reactions are more difficult to evaluate in birds because the wings are non-weight bearing. Normal postural reactions require intact sensory and motor pathways as well as functional processing and integration in the CNS. Postural reactions may be seen via assessment of behaviors such as perching vs. knuckling of the foot, wing extension and return to normal position, normal flapping when the bird is held and then dropped in space, and normal extensor postural thrust reaction.

As in mammals, lesion localization is essential for proper treatment and assessment of prognosis. Lesions in the cerebral cortex may result in change in mentation, poorly tuned fine motor movement, contralateral proprioceptive loss, postural reaction deficits, and contralateral vision loss. Birds with midbrain lesions appear sleepy, may have decerebrate rigidity, and exhibit varying degrees of paresis and strabismus. Ventrolateral strabismus is associated with CN III dysfunction while dorsomedial strabismus is an indication of CN IV damage. Caudal brainstem
Lesion signs may include irregular respiration, ataxia, paresis, postural reaction deficits, and dysphagia. Cerebellar lesions result in intention tremors, dysmetria, ataxia, and incoordination while maintaining normal muscular strength. Birds with vestibular lesions may exhibit head tilt, nystagmus, ataxia, and rolling or circling behavior.

Radiographs may be helpful in TBI diagnosis though the multiple super-imposed and pneumatized bones in the avian skull make interpretation difficult. CSF collection is done through the foramen magnum between the cerebellum and the dorsal surface of the medulla though this is contraindicated in birds with increased ICP and those that are an anesthetic risk. Lumbar puncture is not possible because of the synsacrum and the absence of the cauda equine in birds. A CT scan can be useful to locate intracranial lesions though an MRI examination provides the advantage of imaging the caudal brainstem in the caudal fossa of the skull without artifact, an increase in soft tissue detail and the creation of images in various planes. EEGs have been done in birds though the results may be difficult to interpret due to lack of normal values in many species. Monitoring of ICP is very useful for directing TBI therapy though, unfortunately, there is no documentation on its use in avian species. This may be because of the difficulty in placing the monitoring device through the lightweight, spongy avian skull.

General Considerations in Treatment of TBI

The primary goals of TBI therapy are to maintain normal blood pressure, prevent increased ICP, prevent neuronal edema and ischemia and prevent any brainstem herniation from occurring. Clinical signs of TBI will develop over time – therefore, animals should be treated and monitored for at least 48 hours before they are considered to be stable.

Much of the therapy for avian TBI is based on techniques used in small animal medicine. Thus, as in these species, recommendations for limiting fluid volume are now contraindicated due to resulting dehydration and hypotension. Restoration of cranial perfusion pressure (CPP) is accomplished through the use of crystalloids and/or colloids with careful monitoring of response to fluid therapy to avoid overhydration and an increase in CPP. Hypertonic saline is used in cases of profound hypovolemic shock and cerebral edema, though the effects are short acting unless colloids are also used in fluid therapy. Recommendations in small animal medicine are 4 ml/kg 7.5% NaCl or 5.3 ml/kg 3% NaCl over 2-5 minutes. This should be followed by colloid administration, e.g. Hetastarch® at 10-20 ml/kg given over 15 minutes and then finally by continued crystalloid administration with LRS or 0.9% NaCl.

Oxygenation and ventilation should be maintained and their effects monitored through assessment of respiratory rate and pattern, mucous membrane color and auscultation. Pulse oximetry is not considered a reliable means of determining oxygenation in birds. Arterial blood gases may be analyzed as a means of determining PaO2 and PaCO2 levels. The use of hyperventilation to increase oxygen rates and decrease CO2 levels is not generally useful because this may lead to cerebral hypoperfusion and increase the risk of brain ischemia. Transfusions should be considered as a means to deliver additional oxygen to tissues.

Mannitol is considered to be one of the first treatments used for patients with increased ICP after TBI. This drug decreases cerebral edema through promotion of fluid shifts from intracellular
and interstitial areas of the brain into the vasculature. In small animals, the effects peak one hour after administration and effects persist for 6-8 hours. However, its use is contraindicated in hypovolemic patients and, as discussed above, it is difficult to monitor ICP in birds in order to accurately determine the effects of mannitol. Mannitol therapy should always be followed by crystalloid administration. Recent studies have shown that there may be more benefit with the use of 3% NaCl over mannitol.

General recommendations for treating TBI patients include keeping them in a cool, dark and quiet environment and elevating the head to 30° above horizontal with the bird in a recumbent position. This position increases venous drainage from the head without changes in cerebral oxygenation. Birds with TBI should also be placed in an oxygen cage set between 20-40% O2. Other considerations include avoiding hyperglycemia since this condition can induce lactic acidosis which leads to oxygen free radical production and consequent CNS damage. In addition, hyponatremia should be avoided as this can exacerbate cerebral edema. Narcotic analgesics should be used judiciously as they can cause hypoventilation and increased ICP.

Seizure activity in birds can lead to hyperthermia, hypoxemia and cerebral edema. Thus, aggressive treatment of this condition in TBI patients is necessary in order to avoid increased ICP. Anticonvulsant drugs used in birds include diazepam given 0.5 – 1.0 mg/kg IV or IO and phenobarbital at 1-2 mg/kg IV/IO or IM q 6-12 hours.

**Monitoring Patients After TBI**

Continuous monitoring of patient status after TBI is necessary in order to evaluate the effectiveness of treatment as well as determining prognosis. Monitoring includes evaluation of neurological status, blood pressure, cardiac and respiratory status, body temperature, and hematological values.

The prognosis is fair to good in patients with minor, non-progressive injuries. Signs such as severe bilateral miosis and diminished consciousness can indicate acute severe CNS trauma and increased ICP. Dilated pupils and an absence of PLR, stupor or coma, and an abnormal respiratory pattern usually indicate brainstem involvement and carry a grave prognosis. Delayed development of brainstem signs usually signals forebrain herniation from progressive edema or hemorrhage.

**Conclusion**

Both pet and wild birds are often admitted to veterinary practices and rehabilitation centers with head and ocular trauma. Understanding the pathophysiology of TBI is helpful in evaluating treatment options while careful evaluation of neurological status is important in determining prognosis. Treatment should be based on current standards of care for TBI in the veterinary and human literature.
Suggested Reading and Resources


