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Laryngeal Paralysis in a Loggerhead Sea Turtle (*Caretta caretta*)

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Abstract: A cold-stunned, sub-adult, female loggerhead sea turtle (*Caretta caretta*) was found stranded in December 2012 on Cape Cod, Massachusetts. The turtle was treated routinely; however, she did not gain weight as expected during rehabilitation. Eleven weeks after stranding, staff noticed stertorous upper airway sounds. Oral examination revealed a unilateral laryngeal paralysis-like condition resulting in partial airway obstruction. Magnetic resonance imaging and electromyography showed abnormalities of the abductor arytenoideae muscle. Muscle and hepatic biopsies were collected for histologic and toxicologic assessment to seek an underlying cause of the disorder. The turtle did not recover from anesthesia, and necropsy revealed a large amount of hemorrhage from the liver biopsy site. Unilateral myofiber atrophy of the abductor arytenoideae muscle was the predominant histological finding.

Key Words: *Caretta caretta*, cold-stunned, laryngeal paralysis, loggerhead, pathology, turtle.

Introduction

The loggerhead sea turtle (*Caretta caretta*) is circumglobal in distribution throughout temperate and tropical seas of the Atlantic, Pacific, and Indian Oceans. The loggerhead sea turtle is listed as Endangered by the International Union for Conservation of Nature (IUCN, 2014), and various populations are listed as Endangered or Threatened under the United States Endangered Species Act (U.S. Fish and Wildlife Service [USFWS], 2015). Juvenile and subadult loggerhead turtles utilize New England waters as seasonal foraging habitat during warmer months of the year (Morreale and Standora, 2005). As water temperature decreases below 10°C (50°F) in autumn, turtles that do not migrate to warmer waters may become cold-stunned along the New England coast (Still *et al.*, 2005). Cold-stunned turtles have low body temperatures, cease swimming, float on the surface of the water, and are prone to stranding (Wyneken *et al.*, 2006). Such turtles may be affected by significant physiologic derangements, such as respiratory acidosis, hyperkalemia, and severe bradycardia, and pathologic conditions such as pneumonia and sepsis (Wyneken *et al.*, 2006; Innis *et al.*, 2009). Live, stranded, cold-stunned turtles are often rescued and transported to rehabilitation centers for evaluation and treatment.

Case Report

A cold-stunned, 25 kg, subadult, female loggerhead sea turtle was recovered from Cape Cod, Massachusetts by volunteers and staff of the Massachusetts Audubon Society, Wellfleet Bay Wildlife Sanctuary, in December 2012 and was admitted to the New England Aquarium Animal Care Center (Quincy, Massachusetts). The animal was assessed by physical examination, radiography, and hematologic and plasma biochemical analyses and was determined to be in stable condition. It had a partially healed, minimally displaced carapace fracture beginning at the junction of the fourth vertebral and left fourth and fifth costal scutes, extending laterally to the carapace margin. There was minimal necrotic tissue and the coelom was not violated. The turtle was managed as a routine cold-stun and trauma case using established protocols including fluid therapy, gradual warming, and antibiotics (42 mg/kg oxytetracycline [Bio-Mycin 200, 200 mg/ml, Boehringer Ingelheim Vetmedica, Inc., St. Joseph, MO] IM once weekly for 8 wk) (Harms *et al.*, 2004; Wyneken *et al.*, 2006; Innis, 2014). The turtle responded well to treatment and began eating voluntarily 10 days after admission. Appetite remained fair, but was subjectively low for the species, and the turtle failed to gain weight as expected over the course of rehabilitation. Serial

hematologic and plasma biochemical data revealed no significant findings.

Eleven weeks after admission to the hospital, exaggerated respiratory effort and loud respiratory sounds were noted. Examination revealed stertorous upper airway sounds during inspiration and increased inspiratory effort. Oral examination was considered normal but was limited in the conscious animal. Radiographic assessments of the lungs and trachea were normal. One week later, antiparasitic treatment (25 mg/kg praziquantel [Praziquantel, Fish Remedies, LLC, Vero Beach, FL] PO q3h for three doses in one day) was provided based on species-specific pharmacokinetic data due to the high prevalence of spirorchid trematode infection in this species (Jacobson *et al*., 2003).

Clinical signs persisted 3 wk after onset. Further examination and tracheobronchoscopy were performed under anesthesia (5 mg/kg ketamine [Ketaset® 100 mg/ml, Pfizer Animal Health, New York, NY] IV; 0.05 mg/kg dexmedetomidine [Dexdomitor® 0.5 mg/ml, Pfizer Animal Health] IV). As typical for sea turtles, the turtle was apneic after induction. Oral examination showed mild hyperemia of the glottis mucosa, with the glottis remaining in the normal closed position while apneic. Lidocaine 0.4 mg/kg topically (Lidocaine 2%, Hospira, Lake Forest, IL) was administered on the glottis as local anesthetic for bronchoscopy. Direct bronchoscopy showed mild hyperemia of the proximal 2–3 cm of the trachea, with no apparent ulceration or plaques. The remainder of the trachea, bronchi, and anterior lung fields were considered normal, with only small aggregates of foamy mucus. Several mucus samples were collected for cytology, aerobic culture, and polymerase chain reaction (PCR) testing for chelonian herpes viruses (University of Florida, Zoological Medicine Infectious Disease Laboratory, Gainesville, FL). Upon completion of bronchoscopy, the turtle was intubated and manually ventilated with air at an airway pressure of 10 cm water and a rate of one breath per minute until spontaneous respiration was achieved. Anesthesia was reversed with 0.5 mg/kg atipamezole (Antisedan®, Pfizer Animal Health) IM, and 0.05 mg/kg atropine (Atropine sulfate 0.54 mg/ml; Med-Pharmex Inc., Pomona, CA) IM was given due to bradycardia. As spontaneous ventilation resumed, oral examination revealed consistent absence of right glottis movement during inspiration, similar to that seen in dogs affected by laryngeal paralysis (Fig. 1). The left glottis cartilage moved well. It could not be determined whether this finding was due to true paralysis or secondary to topical lidocaine usage; however, similar findings had not been seen by the authors during numerous previous sea turtle procedures in which topical lidocaine was applied to the glottis. Cytologic results were unremarkable and aerobic culture and herpes PCR were negative.

Stertorous airway sounds persisted after anesthetic recovery. To assess if the absence of the right glottis movement was a persistent problem or a transient result of topical lidocaine use, manual restraint was used 10 days later for reassessment of glottis function while conscious. The examination showed consistent lack of lateral movement of the right side of the glottis, which was flaccidly inhaled into the tracheal opening during inspiration, resulting in approximately 50% obstruction of the trachea.

To better characterize the nature of the presumptive laryngeal dysfunction and to assess for possible anatomic abnormalities, a neurologic examination and additional diagnostic

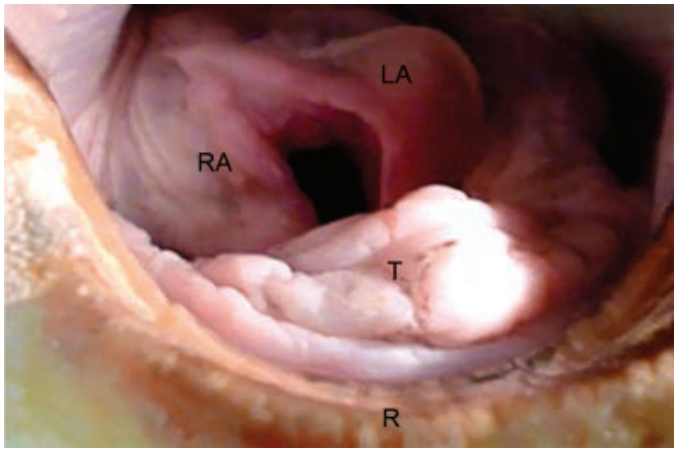

Figure 1. Right laryngeal paralysis in a loggerhead sea turtle; right anteroposterior oblique view. The turtle is inspiring. The left arytenoid (LA) is well abducted while the right arytenoid (RA) remains on mid-line. T = tongue; R = rhamphotheca.

testing was pursued 6 wk after the onset of clinical signs. The conscious neurologic physical examination was unremarkable. Anesthesia was induced with ketamine and dexmedetomidine as previously described, and the turtle was maintained on isoflurane (Aerrane, Baxter HealthCare Corporation, Deerfield, IL) in oxygen with intermittent, positive-pressure ventilation. Laryngeal examination during intubation confirmed previous observations of right laryngeal paralysis. Electromyography (EMG), magnetic resonance imaging (MRI), and computed tomography (CT) were conducted. With the turtle in dorsal recumbency, EMG needles were inserted through the skin on the ventral lateral aspect of the larynx, targeting the abductor arytenoideae muscles. The EMG showed fibrillation and positive sharp waves in the abductor arytenoideae muscle with the right side more-severely affected than the left side. These EMG findings were consistent with muscle membrane instability, which can be seen from myositis or denervation of the affected muscles. MRI was performed prior to and after administration of intravenous contrast medium (gadopentetate dimeglumine contrast medium 100 mg/kg IV; Magnevist®, 469 mg/ml, Bayer HealthCare Pharmaceuticals, Inc., Wayne, NJ). T2-weighted spin echo parasagittal images showed an ill-defined hyperintensity of the right abductor arytenoideae muscle (Fig. 2) (Fraher *et al*., 2010). T1-weighted precontrast images revealed no abnormalities but, following contrast administration, inhomogenous and predominantly peripheral enhancement of the right abductor arytenoideae muscle was seen (Figs. 3, 4). Differential diagnoses for the MRI findings included denervation, inflammation–myositis, and neoplasia. A CT scan of the head was performed but showed no significant findings (Arencibia *et al*., 2006). Anesthesia was reversed with 0.5 mg/kg atipamezole IM and the turtle recovered smoothly. The neurologic examination and diagnostic evaluation study supported a clinical diagnosis of laryngeal paralysis.

Ten days later, due to persistent symptoms, treatment with 50 mg carprofen (Rimadyl® 100 mg tablets, Pfizer Animal Health) PO every 3 days (empirical dose) and adjunctive weekly acupuncture treatment for 3 wk was initiated. The acupuncture treatment protocol was comprised of distal acupoints on the limbs and head. Points were chosen accord-

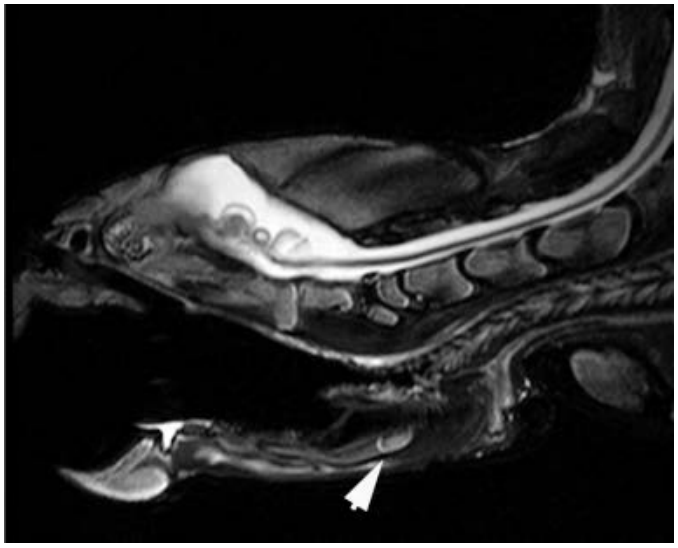

Figure 2. Right parasagittal T2-weighted spin echo magnetic resonance image of the head of a loggerhead sea turtle with laryngeal paralysis. There is focal hyperintensity of the abductor arytenoideae (arrow).

ing to classical Traditional Chinese Medical theory, with consideration made for maintaining needle retention while limiting stress on the turtle during treatment. Acupuncture points GV 20, Yintang, KI3, and LI4 were needled at 1- to 4-mm depths using 0.16- and 0.18-mm ga needles (Seirin J-Type Acupuncture Needles #1/#2, Seirin Corporation, Kyoto, Japan). Needles were retained for a minimum of 20 min.

Underlying disorders such as hypothyroidism, toxicoses (e.g., lead toxicity, biotoxins), and nutritional deficiencies were considered, as these have been associated with laryngeal dysfunction in other species (Jaggy *et al.*, 1994; Allen, 2010; Bedford *et al.*, 2013). Although there are minimal comparative data available for loggerhead sea turtles, free

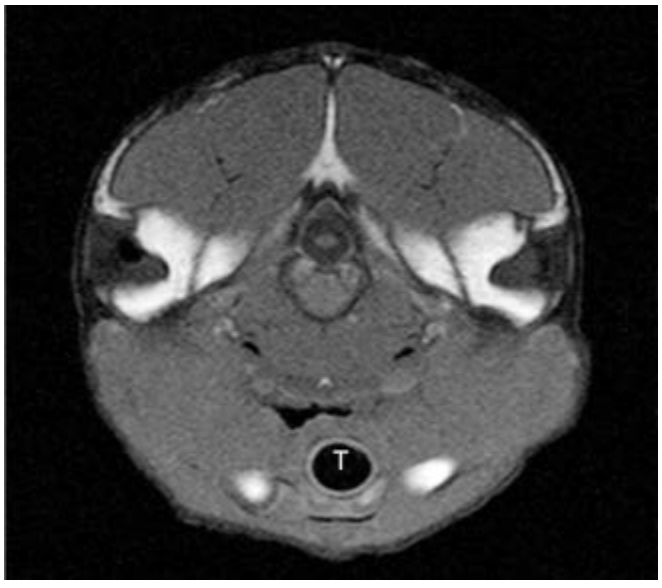

Figure 3. Axial T1-weighted spin echo magnetic resonance image of the head of a loggerhead sea turtle with laryngeal paralysis (prior to administration of contrast agent). No abnormalities are noted. T = trachea.

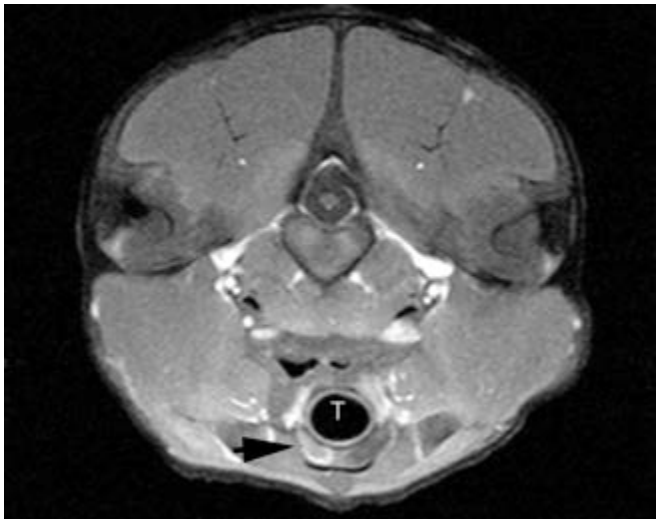

Figure 4. Axial T1-weighted spin echo magnetic resonance image of the head of a loggerhead sea turtle with laryngeal paralysis (after administration of contrast agent). There is contrast enhancement (predominantly peripheral enhancement) of the right abductor arytenoideae (arrow). T = trachea.

thyroxine (2.2 pg/ml), blood lead (<0.02 ppm), plasma vitamin E (8.74 µg/ml), and selenium (386 ng/ml) concentrations were considered to be normal in comparison to values for domestic species. Nonetheless, vitamin E and selenium treatment was initiated with vitamin E–selenium injection (E-Se 68 IU/ml Vit. E, 2.5 mg/ml Se, [vitamin E–Se] Schering-Plough Animal Health Corp., Union, NJ) at an intended dose of vitamin E 1.5 IU/kg IM. However, a 10-fold overdose was given due to human error, and no further doses were given thereafter.

Due to persistent clinical signs and lack of response to non-steroidal anti-inflammatory therapy, acupuncture, and vitamin E–Se therapy, further diagnostics were performed to rule out other underlying diseases 10 wk after onset of the clinical signs. The turtle was premedicated with 1 mg/kg morphine (morphine sulfate injection, 15 mg/ml IM, West-Ward Pharmaceuticals Corporation, Eatontown, NJ). Anesthesia was induced with ketamine and dexmedetomidine as previously described and maintained with sevoflorane (SevoFlo®, Abbott Animal Health, 200 Abbott Park Road, Abbott Park, IL) in oxygen with intermittent positive-pressure ventilation. Prefemoral laparoscopy revealed immature ovaries and normal gastrointestinal serosa and lung surfaces. The liver was mottled pink to tan and a 5-mm cup biopsy was collected for histology and culture. An additional 2-g liver biopsy was collected for toxicologic analysis using monopolar radioendosurgical scissors. Minimal bleeding occurred immediately after sampling. The coelom was closed routinely, and the turtle was repositioned into dorsal recumbency for laryngeal muscle biopsy and culture. Atipamezole 0.5 mg/kg IM was administered to begin anesthesia recovery, and the turtle was repositioned into sternal recumbency for esophagogastroscopy. Endoscopy showed no gross lesions, but several areas of the stomach mucosa had mild hyperemia and a slightly rough surface. Four gastric mucosal biopsies were collected. An orogastric tube was placed under endoscopic guidance for later administration of barium sulfate to begin a gastrointestinal radiographic contrast series. Cloacoscopy was performed to allow ure-

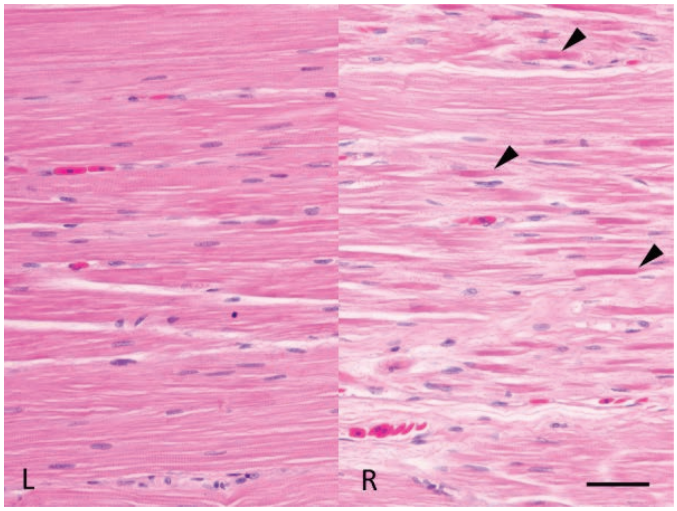

Figure 5. Histology of the left (L) and right (R) abductor arytenoideae muscles. Individual and groups of myofibers within a section from the right muscle are shrunken and stain intensively (black arrowheads), which is suggestive of atrophy and compression by surrounding unaffected muscle. The section from the left muscle is unremarkable. Hematoxylin and eosin. Scale bar = 50 μm.

thral catheterization for collection of 20 ml of urine for biotoxin analysis. After completing other procedures, 3.6 g/kg barium sulfate (Liquid E-Z-Paque®, 600 mg/ml, E-Z-EM Inc., Lake Success, NY) was administered into the stomach.

After surgery, the animal showed no sign of anesthetic recovery over several hours despite three doses of 0.05 mg/kg naloxone (Naloxone HCl, 0.4 mg/ml, Hospira Inc) IM, two doses of 0.5 mg/kg atipamezole IM and IV, one dose of 2.5 mg/kg doxapram (Dopram®, 20 mg/ml, West-Ward Pharmaceuticals Corporation, Eatontown, NJ) IV, and 1 mEq/kg sodium bicarbonate (Sodium Bicarbonate, 1 mEq/ml, Hospira Inc.) in 20 ml/kg lactated ringers solution (Lactated Ringer's Injection, USP, Hospira Inc.) SC. Intermittent positive pressure ventilation was maintained initially with room air, followed by oxygen. Analysis of venous blood samples showed moderate hyperglycemia, mild respiratory acidosis, and hypoxemia, which worsened over time despite oxygen therapy and an appropriate ventilation rate. Heart rate remained relatively stable at 28–32 beats per minute but no reflex responses were detectable. Supportive care, including ventilation, was continued overnight but the animal died the following morning.

Gross necropsy revealed a significant amount of bleeding from the liver biopsy site and approximately 200 ml of coagulated and uncoagluated blood throughout the coelomic cavity, representing approximately 10 percent of the turtle's total blood volume. There were multifocal zones of pectoral muscle discoloration that were presumed to be associated with IM injection sites. Histopathological examination of biopsy samples and tissues collected at necropsy did not reveal a clear cause of death; however, notable findings included abnormalities involving skeletal muscle and the urinary bladder. Shrunken, intensely eosinophilic individual and groups of myofibers were observed in a longitudinal section of the right abductor arytenoideae muscle (Fig. 5). These histological changes were uniform among affected myocytes (monophasic) and were suggestive of compression of atrophied fibers by unaffected adjacent muscle. Unfortunately, a representative transverse section could not be obtained due to the small size and orientation of muscle. This finding was not observed in the contralateral abductor muscle. In addition, a focal area of muscle regeneration was observed within the ventral laryngeal musculature. No associated fibrosis, lesions affecting intramuscular nerves, or inflammation were present. Additional findings included a relatively severe, predominantly mononuclear cystitis, with no etiologic agent seen, and granulomatous inflammation with fibrosis and intralesional bacteria at presumptive IM injection sites. No brain lesions were seen histologically. All cultures (aerobic, anaerobic, and fungal) from the liver and laryngeal muscle biopsies were negative.

Analyses for the presence of saxitoxin (enzyme-linked immunosorbent assay [ELISA]) and brevetoxin (receptor binding assay) were conducted postmortem using urine and liver samples collected 1 day prior to death, feces collected during necropsy, and archived plasma samples collected on the day of admission and 1 month after admission (NOAA National Ocean Service, Marine Biotoxins Program, Charleston, SC). Results showed undetectable concentrations of both toxins in all samples.

Discussion

Laryngeal paralysis is common in dogs and horses, but to the authors' knowledge has not been reported in reptiles. Laryngeal paralysis can be congenital, hereditary, or acquired. Causes of acquired laryngeal paralysis include inflammatory disorders (infectious and non-infectious), traumatic injuries (e.g., bite wounds, collar or leash injuries, surgical trauma), toxins (e.g., lead, organophosphates), cervical neoplasia, and paraneoplastic syndromes (Allen, 2010; Kitshoff *et al*., 2013). Laryngeal paralysis has been associated with hypothyroidism, myasthenia gravis, and generalized neuromuscular disease, but many cases are idiopathic (Allen, 2010; Kitshoff *et al*., 2013). The most-effective treatment for severely affected dogs and horses is unilateral cricoarytenoid lateralization, also called "tie-back surgery," during which a suture is placed between the cricoid cartilage and the arytenoid cartilage to permanently open the arytenoid cartilage (Kitshoff *et al*., 2013). Other less-common surgical treatment options include arytenoidectomy (a partial laryngectomy), ventriculectomy or vocal cordectomy (or both), permanent tracheostomy, and a neuromuscular pedicle graft (Kitshoff *et al*., 2013). None of these surgical treatment choices are an option for an aquatic turtle that must routinely fully close its glottis to avoid aspiration of water.

Based on all available data, the most parsimonious diagnosis for the present case was laryngeal paralysis of unknown etiology but likely due to denervation atrophy. While it appeared clinically that the turtle developed the condition during the rehabilitation process, in light of the pre-existing carapace fracture, it is possible that laryngeal paralysis was trauma-related. The turtle was generally unthrifty throughout rehabilitation in comparison to conspecific cases. However, no substantial underlying disease process was identified through a suite of diagnostic testing. Although clinical signs, imaging, and histologic findings suggested unilateral disease, EMG findings indicated that the left abductor arytenoideae was also affected but to a much lesser degree than the right. The sensitivity of an EMG for detecting subtle

neuromuscular changes is considered superior to that of an MRI (McDonald *et al*., 2000).

The therapeutic interventions in this case were not successful but were reasonable. Antimicrobial therapy is often required for debilitated and traumatized sea turtles, as they are prone to bacterial pneumonia, osteomyelitis, sepsis, and other infections (Orós *et al*., 2005; Innis *et al.*, 2009). Oxytetracycline is commonly used in sea turtles, and pharmacokinetic data support its use in loggerhead sea turtles (Harms *et al*., 2004; Innis, 2014). It is likely that the observed pectoral muscle lesions could have been caused by repeated oxytetracycline injections or other IM injections, but this remains speculative at this time. The findings should prompt clinicians to closely monitor sea turtles during long-term intramuscular drug use.

There are no clear guidelines for non-steroidal anti-inflammatory use in sea turtles, thus clinicians often extrapolate doses and dosing intervals. The carprofen dose used in the present case is similar to that used in dogs, while the dosing interval was selected conservatively. There are no data to support or refute a specific carprofen dosing interval in sea turtles at this time. Acupuncture treatment was selected because the authors have seen a favorable clinical response in several other neurological or musculoskeletal cases in aquatic animals, including sea turtles. Vitamin E–Se therapy was also deemed reasonable, as vitamin E deficiency is common in captive marine animals and vitamin E-responsive neuromyopathies have been described (Hill *et al*., 2001; Bedford *et al*., 2013). It is not clear how the 10-fold vitamin E–Se overdose may have affected this case. Other therapies that were considered included corticosteroids, low-level laser therapy, and doxepin. Anecdotal observations suggest that doxepin, a tri-cyclic antidepressant, may be effective in dogs with laryngeal paralysis, but controlled studies are lacking (Sumner and Rozanski, 2013). It is likely that these options would have been tried if the turtle had survived longer. The failure of the turtle to recover from the surgical biopsy procedure could have been due to an undiagnosed underlying disease, an adverse drug reaction, an adverse reaction to monopolar surgical current, or other cause(s). However, given that it had recovered uneventfully from previous anesthetic episodes, and in light of the necropsy findings, it is likely that the cause of death was hypovolemia secondary to bleeding from the liver biopsy site. Although minimal bleeding was observed laparoscopically at the time of surgery, it is possible that hemorrhage began later due to repositioning or increasing blood pressure during recovery. An underlying coagulopathy due to carprofen use or other causes cannot be excluded. A substantial liver biopsy was required for toxicologic evaluation. For future cases, greater caution should be employed to ensure adequate hemostasis of such biopsy sites.

The urinary bladder pathology seen in this case is not common in sea turtles, and its cause could not be determined. It is possible that this disease process contributed to the general unthrift of this animal. In general, detection of lower urinary tract disease in aquatic animals is challenging, as their urination is generally not observed.

Although there are a number of descriptions of the muscular anatomy of the head and larynx of turtles, the detailed anatomy and nomenclature of the laryngeal musculature of loggerhead sea turtles are not clearly defined (Wyneken, 2001; Sacchi *et al*., 2004; Fraher *et al*. 2010; Arencibia *et al*., 2012; Jones *et al*., 2012). For example, in a description of the cross-sectional anatomy and MRI imaging of the head of loggerhead sea turtles, the musculature is simply referred to as the "laryngeal muscle" (Arencibia *et al*., 2012). Here we have elected to use the term "abductor arytenoideae" to apply to the muscle that opens the glottis based on the nomenclature of this muscle in another sea turtle species, the leatherback turtle (*Dermochelys coriacea*) (Fraher *et al*., 2010). Other authors have referred to this muscle in some turtle species as the dilator laryngis (Sacchi *et al*., 2004). Additional anatomic and functional descriptions of these muscles in loggerhead sea turtles are warranted.

In summary, this report describes clinical signs and diagnostic findings in a loggerhead sea turtle with laryngeal paralysis. While the underlying cause of the disease was not determined and case management was unsuccessful, this case should prompt clinicians to consider laryngeal dysfunction as a possible cause of upper airway signs in sea turtles.

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