

What You Need to Know to Care for Tortoises

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The goal of this lecture is to familiarize you with tortoises, how to house them, feed them and treat them. The first part of this lecture will cover care of tortoises, how to house and feed them (covered in 'Hibernation (Brumation) Problems of Tortoises'). The second part, starting here, covers how to diagnose and treat them. Blood collection by jugular venipuncture is easier as a multi-person procedure with a butterfly catheter. For most chelonians over a kilogram sedation is recommended with 10–20 mg/kg Telazol IM or SC, then draw blood 15 to 20 minutes later. Another site for intravenous access is the subcarapacial venous plexus venipuncture (SCVP) but is fraught with lymph contamination and can result in spinal compromise, thus the jugular vein is preferred for phlebotomy. Lymph contamination has been shown to statistically decrease values for many chemistry values.

Mycoplasmosis, chronic nasal sinusitis, is a slowly debilitating disease causing nasal discharge that rarely kills tortoises. Several species are implicated including *Mycoplasma agassizii*, *M. testudineum* and unidentified *Mycoplasma*, but not *M. testudinis*. Clinical signs include clear serous to tenacious mucoid bubbling to mucopurulent nasal discharge, sometimes also from the eyes, conjunctivitis and palpebral edema. Clinical signs may appear within 1–2 weeks of exposure. Nasal discharge is always abnormal in tortoises, and typically caused by mycoplasmosis, but also can be caused by herpes virus, ranavirus, adenovirus, intranuclear coccidiosis, paramyxovirus, chlamydophilosis, reovirus, foreign bodies, gastric reflux or regurgitation and oronasal fistulas (see Nasal Discharge in Tortoises for more information on treatment and diagnosis). *Mycoplasma* is spread directly via nasal exudates and is very contagious as tortoises often greet one another nose to nose. Many tortoises are chronically infected and have decreased appetite. Tortoises with acute infections may be inappetent and lethargic with clear nasal discharge. Chronic cases can have erosion of the nares and anterior beak; hatchlings can have enlargement and distortion of the snout (reminiscent of atrophic rhinitis in swine), bubbling from the nares or have clogged nares and have lost weight or feel light.

Nutritional secondary hyperparathyroidism (NSHP) is common and is obvious with soft shells, inability to lift the shell while walking, splayed legs, and decreased appetite. Tortoise shell should feel very much like skull, solid, with no soft areas (except pancake tortoises). Young leopard tortoises raised by the author develop a firm shell within about a year, desert tortoises may take longer. Calcium deficient females probably produce calcium deficient hatchlings. Clinical signs vary dependent on age of exposure to a calcium deficient diet. Small tortoises' fail to grow or gain weight, have soft shells, splay out their legs, have overgrown nails, fail to lift their plastron while walking and are often not eating. Young desert tortoises weighing less than 20 grams, with soft shells, and anorexia, carry a grave prognosis and are also protein deficient. Stomach tubing nutritional support (LaFaber Omnivore Care) every few days and calcium may encourage eating of thawed frozen green peas (Jarchow, personal communication), which are protein rich. If the patient is eating and defecating it should recover if the diet is corrected and UV light is provided. Anorexic NSHP turtles die despite intensive treatment, McArthur suspects many are in kidney failure.

If the tortoise developed normally prior to adoption of a calcium/protein deficient diet the shell may feel firm but the marginals curl dorsally, the seams collapse inwardly, the bridge widens vertically, the plastron scrapes while walking, the inner rear nails wear excessively while the outer nails overgrow, the body may appear too large for the shell, the anterior maxillae may curve more like a parrot's beak, penile or cloacal prolapse may be present and the normally straight ventral or arched dorsal midline may curve laterally or ventrally. Pyramidal shell growth often accompanies calcium deficiency, however pyramidal shell growth is not solely caused by calcium deficiency; it is probably multifactorial in origin. Good nutritional history, radiographs and blood work can give you additional insights into diagnosis in patients with minimal clinical signs. Calcium deficiency on radiographs can be subtle initially, look for decreased bone opacity, such as faint pectoral and pelvic opacity, and poor cortical definition or thinning.

Both ionized and total calcium may be normal to low and there may be inverse calcium to phosphorus (i.e., < 1:1). With obvious clinical signs diagnostics are not needed for diagnosis.

Calcium gluconate (360 mg/ml) at 1 ml/kg SID to BID or powdered calcium carbonate, Ca lactate or Ca citrate on food should be continued for months until the shell feels like solid bone, the patient is gaining weight and eating well. Obviously ultraviolet light and intensive nutritional restructuring are in order. Calcitonin has been recommended at 50 IU/kg IM, repeat in 1 week, once normocalcemic or one week after starting calcium supplementation as well as vitamin D (400 IU IM, repeat in 1 week). Do not give calcitonin if hypocalcemic. Recently calcitonin has become very expensive.

Dog gnaw trauma is common. Neighboring dogs will dig under fences or grab tortoises through gaps in the fencing. Small chelonians can be devoured without a trace. A dog that shows interest in tortoises should never be trusted with a tortoise. Raccoons and opossums enter yards at night to prey on tortoise limbs. Lawn mowers, automobiles and large hoofed stock can all injure tortoises. Dog gnaw trauma and shell repair include extensive wound cleaning and lavage, debridement of dead or devitalized tissue, wet to dry bandages, antibiotics, pain medications, fluid and nutritional support, and repair, once clean, with epoxy patches, screws and cerclage. Keep the wound open and draining as long as possible. Medical honey and sugar can be used on infected wounds, once clean switch to silver sulfadiazine cream. See Flemming 2014, for review of shell injuries.

Tortoises have an especially large penis which may be pink, purple or black. Phimosis and paraphimosis should not be used to describe penile prolapse, chelonians have no prepuce, nor is penile prolapse caused by constriction of the cloacal opening or cloacal retraction. Penile prolapse is common with NSPH as the tortoise's body outgrows the confines of a small shell. Penile prolapse can also be secondary to straining associated with foreign bodies, constipation, bladder or cloacal uroliths, or parasites, spinal injury or tearing of the retractor penis muscles. Irritation of the penis can also result from infection or breeding. If the penis is viable it should be cleaned under anesthesia and replaced in the cloaca. A purse string suture around the cloaca with a small opening for defecation is recommended for three weeks. If the penis is necrotic or repeatedly prolapses it can be amputated (tortoises don't urinate through their penis). Amputation is straightforward in small tortoise but in larger chelonians, it is more complicated (see 'Chelonian Surgery' or Innis, Boyer 2002).

Adult male Sulcatas can lacerate their penis while mating or masturbating which causes profuse intermittent cloacal hemorrhage. Anesthesia and suturing is required. Overgrown beaks and nails are often accompanied by malnutrition and must be treated in tandem; well fed healthy tortoises typically don't need beak trims. Sedation with 10–20 mg/kg Telazol makes dremeling the beak much easier.

Hepatic lipidosis is common in older tortoises with a history of fruit and grocery store vegetable diets, especially females that don't breed or tortoises that don't hibernate. Often there is other disease, such as mycoplasmosis or a cystourolith, or the tortoise may be lethargic and anorexic on emergence from hibernation. Typically there is a gradual reduction in appetite and activity; the tortoise may be more selective, eating only fruits. Weakness is present, manifest as dragging the plastron while walking. In advanced cases the patient is flaccid, edematous, lethargic, weak (head and legs easily extended), anemic, hypoalbuminemic and hypocalcemic. Tortoises ordinarily do not vomit unless there is GI obstruction. Liver enzymes and bile acids are often normal as there is no breakdown of liver cells, triglycerides and cholesterol may be elevated. Reptiles do not convert biliverdin to bilirubin, therefore icterus is not present. Endoscopic liver biopsy and histopathology is needed for definitive diagnosis. Fatty livers are swollen, friable, pale tan to white, ooze white to yellow fluid when cut, and float in formalin. Hepatic lipidosis patients are poor candidates for anesthesia or surgery, thus many are treated with presumptive diagnosis. Treatment consists of long term nutritional support via esophagostomy tube into the stomach until the patient is eating well on its own. This may take several months. Wright recommended an elemental diet initially, LaFebers Emerald for Herbivores (Lafeber Co, Cornell, IL), then after 3 to 7 days switching to Oxbow's Critical Care for Herbivores (Oxbow Animal Health, Murdock, NE). Stomach tube 20 mls/kg/day, for the first few days, then increase to 30 mls/kg/day, both should be divided into multiple feedings. Multiple small feedings are better than less frequent larger feedings. Flush the tube after each feeding with water. If the patient regurgitates check tube placement and environmental temperature, and back off feeding for a day or two. Too large a volume of food will cause immediate

regurgitation. LaFebers Emerald for Herbivores has a source of vitamin K activity and vitamin B12. Other supplements, such as L-carnitine (250 mg/kg PO q 24 hrs), methionine (50 mg/kg PO q 24 hrs), taurine, S-adenosylmethionine (30 mg/kg PO q 24 hrs), lactulose (0.5 ml/kg PO q 24 hrs), and *Silybun marianum* (extract from milk thistle, 4–15 mg/kg PO q BID to TID) can also be used, however no prospective clinical trials have been conducted to evaluate efficacy of any of these drugs in reptiles. 20 mg/kg ceftazidime SC q 72 hrs is given for 3 weeks until the esophagostomy tube forms a tight seal.

Desert and sulcata tortoises are prone to cystic calculi, largely composed of urates, compounds composed of uric acid and sodium, potassium, or calcium. Cystic calculi can obstruct or torse the colon, and lead to bladder thickening or necrosis. Stones are undoubtedly painful and can lead to slow decline of the tortoise. Stones predominate in the left lobe of the bladder (due to the larger right liver lobe) and can often be palpated prefemorally. Radiographs reveal a laminated radio dense calculus, or calculi. Etiology is unknown, inappropriate diet, more acidic urine, inhibition or loss of bladder cilia, protozoal infections, and lack of access to water, soaking and urination, could be culprits. Concurrent hepatic lipidosis is common and is a contraindication to surgery. Plastral celiotomy is generally needed for most bladder stones (see Chelonian Surgery for discussion and aftercare), bladder stones cannot be dissolved, as in mammals. Tortoises that are larger than 15 cms with non-laminated stones that are less than half the length of the prefemoral fossa are candidates for cystourolith removal through the prefemoral fossa (see Mangone, Johnson 1998), which is much less invasive affording a faster recovery. To do this the stone should be easily smaller than the prefemoral fossa opening, which is rarely the case. The bladder is marsupialized to the prefemoral skin, or exteriorized and incised, and the stone is broken down and removed through the prefemoral fossa, cutting the plastron may increase exposure. Juvenile tortoises with flexible plastrons can be cut on midline with a scalpel and the stone removed through an H shaped plastral incision, which can be wired shut. Most tortoises that are healthy prior to surgery recover well, but may not eat for several weeks. Always place an esophagostomy tube after plastral celiotomy.

Cloacaliths arise from cystoliths that pass from the bladder and become lodged within the pelvis. Do not confuse a cloacalith with an egg lodged in the pelvis. Either must be extracted per cloaca, cloacaliths require extensive drilling and breakdown with forceps to remove under anesthesia. Occasionally a tortoise presents with extreme cloacal dilation secondary to a previous chronic cloacalith or pelvic egg that passed. Bladder prolapse sometimes occurs with bladder stones and can be reduced through the cloaca with endoscopic guidance if caught early.

Gastrointestinal impaction can be partial or complete. Malnourished tortoises may seek and eat small rocks if on a calcium deficient diet. Sand is inadvertently ingested stuck to food. Clinical signs include decreased appetite to anorexia, listlessness, lack of defecation, regurgitation or straining to defecate. Radiographs often reveal impactions at the distal transverse colon as it turns posteriorly for the descending colon in the caudal left coelom, just cranial to the inguinal fossa, with widespread impaction proximally. Distended loops of bowel may be visible in the lung fields on lateral radiographs. Small amounts of gravel and foreign bodies will often pass without treatment and may be considered an incidental finding if the tortoises continues to eat and defecate well. Partial impactions are most common and treated with repeated stomach tubing of mineral oil, fluids and shallow warm water soaks twice daily for 30 minutes and nutritional support if still eating. If the tortoise isn't eating, an esophagostomy tube allows fluids, nutrition, and medications, such as antibiotics and intestinal stimulants, to be delivered. Serial radiographs every 2 to 4 weeks allow progress to be monitored. As long as the tortoise continues eating and defecating foreign bodies should pass. Celiotomy is indicated if medical management fails, or in anorexic animals, if not hypoalbuminemic or anemic. Surgery is easiest by milking material into the cecum from the transverse and ascending colon, then exteriorizing the cecum for enterotomy and extraction of foreign bodies.

Prolapses of the colon, cloaca, hemipenis, bladder and oviduct are possible. Penis prolapse is easy to identify and likewise a cloacal prolapse can be identified by a clitoris. Once edematous, colon and bladder can be difficult to distinguish, the oviduct may have longitudinal striations. Radiographs and bloodwork are useful to identify predisposing causes such as calcium deficiency, uroliths and GI foreign bodies. Most prolapses can be slowly reduced under anesthesia and checked with cloacal laparoscopy

while infusing fluids. A purse string suture with enough opening for feces is recommended for 3 to 6 weeks. The author had one bladder prolapse spontaneously reduce after soaking in warm water overnight, as unlikely as that seems.

Herpesvirus virus causes stomatitis, glossitis, nasal discharge and death in a wide variety of tortoises especially *Gopherus*, *Testudo* and some *Geochelone*. Outbreaks often occur after introduction of Russian tortoises, *Testudo horsfieldi*, which may be a carriers or symptomatic. Several deaths in a collection are common with signs of anorexia, emaciation (temporal atrophy), dehydration (sunken eyes), clear nasal discharge and discrete to multifocal diphtheric white plaques progressing to caseous debris in the caudal oropharynx. There seems to be variability in virus strain pathogenicity or individual immunity. Aggressive treatment with acyclovir (80 mg/kg PO SID x 21 days), fluid/nutritional support, and broad-spectrum antibiotics are important. Necropsy is an easy way to confirm disease with experienced reptile pathologists. Diagnosis can be confirmed by PCR. See Nasal Discharge in Tortoises for more information.

Testudinine intranuclear coccidiosis (TINC) is an emerging disease that can spread quickly in large collections that also presents with nasal discharge and deaths. Clinical signs include rapid weight loss, weakness, nasal discharge, gasping respiration, and swollen erythematous necrotic cloacal crusts. Diagnosis is typically by post-mortem histopathology or PCR of combined conjunctival, choanal and cloacal swabs. The University of Florida has a 24 test TINC panel which is great for screening collections for this highly contagious disease. See Nasal Discharge in Tortoises for more information.

Ulcerative dermatitis of the ventral shell margins can be caused by *Aeromonas hydrophili*. Ulcerated areas should be thoroughly cleaned with chlorhexidine, rinsed and treated with systemic antibiotics and silver sulfadiazine (Silvadene Cream 1%, Monarch Pharmaceuticals, Bristol, TN). Scutes slough from underlying bacterial or fungal infection. Infected detached scutes and bone should be debrided back to healthy tissue (under anesthesia), scrubbed with chlorhexidine, painted with silver sulfadiazine and bandaged. Continue cleaning with bandage changes every other day until healthy granulation tissue set ups, scutes will regrow. Systemic antibiotics are indicated based on culture and sensitivity.

Amblyomma ticks harboring heart water disease (*Ehrlichia* {*Cowdria*} *ruminatum*) were found in the environment around tortoises in Florida and led to a ban on importation of leopard, sulcata and *Kinixys* spp. into the United States in 2000. Ticks are rare on tortoises in captivity in southern California, but present within the indigenous range of desert tortoises.

Salmonella, *Shigella* and *Campylobacter* spp. are common in tortoises, warn owners about proper handling to prevent zoonosis. Intestinal commensals, such as oxyurids (multiple species of pinworms), *Nyctotherus*, *Balantidium coli* and most *Entamoeba* species, are probably normal flora and may help digestion. However epizootics have been reported for *E. invadens* in many chelonians. *E. invadens* is highly contagious, has a direct life cycle and can spread rapidly through a collection. Treatment consists of 50 mg/kg metronidazole EOD for 5 to 10 treatments. Ascarids and strongyles should be treated with 100 mg/kg fenbedazole, repeated in 14 days. Warn owners that live worms may be passed in feces. Recently, 66 mg/kg oxfendazole, or 100 mg/kg fenbendazole, both given via stomach tube, to Hermann's tortoises was shown to eliminate shedding of oxyurid ova after 12 and 31 days, respectively, a second dose was not required. Cestodes can be treated with 8 mg/kg praziquantel SC, repeated in 14 days. Pre-paid follow up fecal collection containers are recommended in chelonians to determine efficacy. Environmental clean up is always recommended.

Renal disease is less commonly encountered and diagnosis is not as straightforward as in mammal medicine. Reptiles don't concentrate their urine, urine is not sterile and, in tortoises, the urinary tract is not easily accessible. Tortoises are uricotelic so blood urea nitrogen and creatinine are less useful. Uric acid levels > 10 mg/dl, or BUN > 40 mg/dl are abnormal, in desert tortoises, both can increase with dehydration. Aggressive rehydration over 72 hrs should correct dehydration but not renal disease (see 'Reptile Anesthesia' for a discussion of fluid treatment). In Mediterranean (*Testudo*) tortoises, uric acid levels > 16.8 mg/dl indicate renal failure, > 25.2 mg/dl causes urate crystal deposition in tissue (gout), and > 33.6 mg/dl becomes life threatening. Hyperuricemia associated with renal disease represents > 2/3's loss of renal mass. In renal failure, there may also be elevation of potassium, phosphorus, AST, and LDH, these parameters are not specific nor sensitive to renal tissue. Reptile kidneys also have high ALT and AP activity but these are not often elevated because they are released into urine not blood. Normal

tortoises have basic urine, catabolic tortoises have acidic urine, but again, this is not specific to renal disease nor always present. Renal disease diagnosis is difficult ante-mortem without renal biopsy and, as in other species, there are myriad potential etiologies. Noninfectious causes of renal disease are often more common, chronic, result from poor husbandry and include degenerative changes to the glomeruli or tubules. Treatment of chronic renal disease in tortoises involves fluid therapy (soaking, epicoelomic or intravenous), nutritional support, and allopurinol (50 mg/kg PO q 24 hrs x 30 days then q 72 hrs thereafter). If hypocalcemic, give 10 mg/kg calcium gluconate PO q 12–24 hrs, if hyperphosphatemic, give aluminum hydroxide 100 mg/kg PO q 12–24 hrs. Monitor bloodwork q 2–4 weeks. Failure to urinate after 10 days of fluid therapy is a poor prognostic indicator. Infectious causes tend to be acute and less common. Flagellated protozoans, such as the diplomonad, *Hexamita*, and the trichomonad, *Monocercomonas*, may ascend from the GI tract into the kidneys and cause tubulointerstitial nephritis, tubular necrosis and mineralization. Clinical signs include anorexia, polydipsia, diarrhea and death, concurrent other disease if often present. Metronidazole at 50 mg/kg EOD for 5 treatments or 100 mg/kg, repeated in 10 days, via stomach tube or capsule on food is recommended.

Females sometimes fail to lay eggs, which are palpable in the inguinal fossa. Females are often pacing their enclosure and may have dug several nests without laying anything. Most chelonians have hard shelled eggs easily distinguishable on radiographs. Sliders are very responsive to oxytocin at 2–10 IU/kg IM q 60 minutes for 2 doses, then q 120 minutes for 2 more doses, or 2–10 IU/kg IV at the same interval for 3 doses. Intravenous administration resulted in faster egg laying in red eared sliders (Ianni, Parmigiani *et al.* 2014). Jarchow recommends oxytocin, 1–3 IU/kg IM, repeat in 48 hrs if the initial dose wasn't effective. Lutalyse (prostaglandin F2alpha) alone will induce egg laying in 90% gravid female sliders, map turtles, cooters and softshells, with one injection, and may also work in tortoises. Dosage is 1 (> 5 kg turtles) to 1.5 (1–5 kg turtles) to 2 mg/kg (< 1 kg turtles) IM. Celiotomy is indicated if oxytocin is ineffective. Eggs very rarely get lodged in the pelvic inlet. If present they may be expelled with digital palpation of the prefemoral fossae under anesthesia, or must be broken down and removed per cloaca.

Tortoises can fall into swimming pools and appear drowned, but tortoises can survive underwater for incredibly long periods, perhaps through anaerobic metabolism. An ECG or Doppler ultrasound can be used to see if there is a heartbeat or pulse. Holding the tortoise head down and mouth open while pumping its legs in and out can result in return to breathing. Intermittent positive pressure ventilation is recommended until breathing well. Doxapram, 5 mg/kg IV and mannitol, 1–2 grams/kg IV can stimulate breathing and urination (Johnson, Jarchow 2002). Antibiotics are recommended for 3 weeks.

Corneal ulcers are common in squinting tortoises and are often secondary to foreign bodies (such as sand, hay or foxtails), or bacterial or fungal infections. Treatment includes topical pain medications and antibiotics, systemic antibiotics, cleaning and flushing the eye under anesthesia and temporary tarsorrhaphy.

Exuberant epiplastrons are occasionally present in male *Geochelone sulcata* and *Gopherus agassizii*. If the epiplastron interferes with the male's ability to eat, it can be amputated and sealed with epoxy. Most do not require any treatment.

Tortoise care and medicine is changing rapidly. Veterinarians need to educate themselves to advise on both health and disease and become advocates for, what should be, long lived patients.

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