Avian and Exotic Animal Clinic

Faculty of Veterinary Medicine

University of Veterinary and Pharmaceutical Sciences Brno, Czech Republic

with

Clinic for Avian, Reptile and Fish Medicine

University of Veterinay Medicine -Vetmeduni, Vienna, Austria

and

Czech Association of Zoo and Wildlife Veterinarians (CAZWV)

MINI-SYMPOSIUM

TERRAPINS, TURTLES, TORTOISES - BIOLOGY, VETERINARY MEDICINE, WILDLIFE

photo: Chris Van Wyk

08-03-2010 Brno

10-03-2010 Vienna

Preface

They all times fascinated us and we must feel responsibility for well-being of chelonians in their natural habitats as well as in captivity. They make us happy and we have to do the best for them. We keep them in our houses, but it was not their choice.

At home, a thousand days are good,
abroad, half a day is sorrow.
Chinese proverbial saying

I hope this mini-symposium will inspire veterinary students, veterinarians in practice and young scientist to study all aspects of veterinary medicine regarding with biology, clinical veterinary medicine and wildlife protection. We invited the leading specialists from Australia and the United States of America, Robert Johnson, BVSc MACVSc CertZooMed BA and Charles J. Innis, DVM, for their excellent lectures about veterinary practice with turtles, tortoises and terrapins. Exotic medicine is fast becoming 'mainstream' in clinical veterinary practice. The modern technologies undoubtedly enable veterinarians to pursue more advanced medical and surgical therapies for chelonian patients.

Z d e n ě k K n o t e k

Photo Johannes Pfleiderer

Mini-symposium: Terrapins, turtles, tortoises - biology, veterinary medicine, wildlife

Želvy ve veterinární praxi tří kontinentů

University of Veterinary and Pharmaceutical Sciences Brno

Faculty of Veterinary Medicine

8th March 2010

Posluchárna PKMZ

13.00 - 13.45 Význam biochemického a hematologického vyšetření při klinickém vyšetření želv. (*The value of haematological and biochemical profile in clinical examination of tortoises and turtles*).

MVDr. Anna Hrdá, MVDr. Zora Knotková, CSc.,
prof. MVDr. Zdeněk Knotek, CSc. Dipl ECZM (herpetology)

Avian and Exotic Anim. Clinic, Univ. Vet. and Pharm. Sci Brno (CZ)
Clinic for Avian, Reptile and Fish Medicine, Vetmeduni, Austria

13.50 - 14.35 Biology and Veterinary Medicine in Marine Chelonians I

Charles J. Innis, DVM

New England Aquarium, Boston, USA

Coffee break

14.50 - 15.35 Biology and Veterinary Medicine in Marine Chelonians II

Charles J. Innis, DVM

New England Aquarium, Boston, USA

15.40 - 17.10 Biology and Veterinary Medicine in Australian Chelonians

Robert Johnson, BVSc MACVSc CertZooMed BA

South Penrith Veterinary Clinic, NSW, Australia

Mini-symposium: Terrapins, turtles, tortoises - biology, veterinary medicine, wildlife

University of Veterinary Medicine – Vetmeduni Vienna

Lecture hall G

10th March 2010

13.00 - 13.45	Advances in Veterinary Medicine in Chelonians Zdeněk Knotek, DVM, PhD Dipl ECZM (herpetology) Avian and Exotic Animal Clinic, Univ. Vet. and Pharm. Sci Brno, (CZ) Clinic for Avian, Reptile and Fish Medicine, Vetmeduni, Austria
13.50 - 14.35	Biology and Veterinary Medicine in Marine Chelonians I Charles J. Innis, DVM New England Aquarium, Boston, USA
	Coffee break
14.50 - 15.35	Biology and Veterinary Medicine in Marine Chelonians II Charles J. Innis, DVM New England Aquarium, Boston, USA
15.40 - 17.10	Biology and Veterinary Medicine in Australian Chelonians Robert Johnson, BVSc MACVSc CertZooMed BA South Penrith Veterinary Clinic, NSW, Australia

Speakers

Anna Hrdá, DVM
Dr. Anna Hrdá graduated from the Faculty of Veterinary Medicine, University of Veterinary and Pharmaceutical Sciences Brno in 2009. In October 2009 she started her PhD study at the Avian and Exotic Animal Clinic. The scientific work of dr. Hrdá is focused on methods of the clinical diagnosis of metabolic diseases in exotic animals. Her hobbies are dog training, reptile breeding and dancing.

Charles J. Innis, VMD
Dr. Charles Innis received his bachelor's degree in Biology from Cornell University in 1990 and his VMD from the University of Pennsylvania School of Veterinary Medicine in 1994. He was in private practice working with small animals and exotics from 1995-2005, and has been working full time at New England Aquarium since 2005, where he is currently the Director of Animal Health. He is a Clinical Assistant Professor in the Department of Environmental and Population Health at Tufts University, Cummings School of Veterinary Medicine. Dr. Innis is a member of the IUCN Tortoise and Freshwater Turtle Specialist Group, the IUCN Marine Turtle Specialist Group, and is Past President of the Association of Reptilian and Amphibian Veterinarians. He has authored many publications on the medical and surgical care of reptiles.

Robert Johnson BVSc MACVSc CertZooMed BA CMAVA
Robert Johnson graduated from the University of Sydney in 1976. He runs a small animal, wildlife and reptile practice in Penrith, New South Wales with his veterinarian wife Jane and works as a clinical veterinarian at Taronga Zoo, Sydney. He is President of the Unusual and Exotic Pets Group of the Australian Veterinary Association. Robert is a member of the Australian College of Veterinary Scientists (Feline Medicine) and holds a Certificate in Zoological Medicine from the Royal College of Veterinary Surgeons. Research interests include the in situ conservation of the Fijian Crested Iguana, *Brachylophus vitiensis*, the collection of baseline haematological and biochemical data of captive and free living Australian herpetofauna and the identification of the protozoal microflora of the short beaked echidna, *Tachyglossus aculeatus*. For relaxation Robert draws cartoons, tries to speak Italian and German and enjoys bushwalking, but not all at the same time.

prof. Zdeněk (Sid) Knotek, DVM, Ph.D, Dipl ECZM (herpetology)
Sid graduated from Brno Vet University in 1982. He is head of the Avian and Exotic Animal Clinic, University of Veterinary and Pharmaceutical Sciences Brno. His current focus involves medicine and surgery in exotic animals, especially in reptiles. Sid has also teaching activities at Vetmeduni Vienna, having lectures for Purdue, Utrecht, Kosice and Ljubljana Vet Universities. He is past president of the European Association of Zoo and Wildlife Veterinarians (EAZWV), president of CAZWV. Sid supervises the joint teaching projects at Brno and Vienna Vet Universities, clinicaly oriented programme for students and veterinarians - Summer School for Exotic Medicine and Surgery, and the intensive training course of European School for Advanced Veterinary Studies – Exotic Pets Medicine and Surgery. He is a diplomate of the European College of Zoological Medicine (herpetology) and member of the international committee of the Association of Reptilian and Amphibian Veterinarians.

VÝZNAM BIOCHEMICKÉHO A HEMATOLOGICKÉHO VYŠETŘENÍ PŘI KLINICKÉM VYŠETŘENÍ ŽELV

MVDr. Anna Hrdá[1]

MVDr. Zora Knotková, CSc.[1]

prof. MVDr. Zdeněk Knotek, CSc. Dipl ECZM (herpetology)[1,2]

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Želvy jsou tradičními pacienty veterinárních pracovišť. Před chovatelem jsou schopny skrývat projevy onemocnění, takže jsou veterinárnímu lékaři prezentovány až při výrazném zhoršení zdravotního stavu. K častým problémům patří chronické poruchy metabolismu, jejichž klinické příznaky jsou nespecifické (apatie, anorexie, svalová slabost apod.). Pozornost se tedy zaměřuje na nepřímé diagnostické metody, zahrnující i hematologické a biochemické vyšetření krve.

Stanovení hematologického a biochemického profilu krve je nezbytnou součástí kvalitního klinického vyšetření u želv. Interpretace výsledků není snadná a praktické klinické využití vyžaduje poměrně velkou zkušenost a současné respektování výsledků, dosažených ostatními vyšetřovacími metodami. U želv existuje více míst vhodných k odběru krve. Doporučovaným způsobem je odběr krve z jugulární vény, při kterém je riziko kontaminace vzorku lymfou minimální. Takový odběr je však u mnoha druhů želv technicky náročný a lze proto využít i odběr z dorsální ocasní žíly nebo subkarapaxiálního plexu. Bezpečně lze odebrat zhruba 10 % z celkového objemu krve, přičemž objem krve plazů je odhadován mezi 5 až 8 % tělesné hmotnosti. Plazma je jako vzorek pro biochemické vyšetření preferována před sérem, neboť srážení krve je u plazů časově náročné a objem získaného vzorku je u plazmy oproti séru výrazně vyšší. Jako antikoagulační látka je vhodný heparin (heparinát sodný, případně lithium heparinát). Hematologické vyšetření zahrnuje stanovení hematokritu, koncentrace hemoglobinu, celkového počtu erytrocytů a leukocytů a určení diferenciálního rozpočtu leukocytů. Počty červených a bílých krvinek se stanovují v Bürkerově komůrce s použitím Natt-Herrickova roztoku. Diferenciální rozpočet se stanovuje v krevním nátěru obarveném dle Pappenheima. Biochemické vyšetření plazmy se běžně provádí jako u savců na automatických analyzátorech. Mezi základní

biochemické parametry, které je vhodné u želv sledovat, patří celková bílkovina, glukóza, kyselina močová, ALP, ALT, AST, CK, Ca a P. Doporučeno je též určit koncentraci albuminu a vypočítat A/G poměr.

Početnou skupinu pacientů tvoří želvy s chronickými poruchami metabolismu. U terestrických druhů želv se v jarních měsících setkáváme s projevy syndromu posthibernační anorexie. Vyšetřením plazmy u nich zaznamenáváme hyperfosfatémii, hypoglykémii zvýšenou koncentraci kyseliny močové a sníženou koncentraci celkové bílkoviny.

Metabolické poruchy skeletu jsou u želv v zájmových chovech velmi časté. Příčinami jsou převážně neadekvátní podmínky chovu, ale v některých případech bývá potvrzeno i chronické onemocnění ledvin (sekundární nutriční nebo renální hyperparathyroidismus). Deficit iontů kalcia v periferní krvi pacient kompenzuje uvolňováním rezerv z krunýře a kostí, ale po vyčerpání zásob bývá vyšetřením zaznamenána hypokalcémie. Pravidelným nálezem je zvýšená aktivita alkalické fosfatázy a zvýšená koncentrace fosforu.

Intravitální diagnostika onemocnění jater u želv je poměrně komplikovaná. U pacientů lze pozorovat anémii, leukocytóza může být potvrzením zánětlivého procesu. Z biochemických parametrů sledujeme při chronických onemocněních jater snížení koncentrace celkové bílkoviny a některých případech i hypoglykémii. Pro nepřímé hodnocení stavu jater, zvláště u chronických onemocnění, je vhodné hodnotit koncentraci žlučových kyselin v periferní krvi. Předpokládá se, že ke zvýšení koncentrace žlučových kyselin v krvi dochází při závažném narušení funkce jater, při hepatoportálních zkratech nebo při obstrukci žlučových cest. Počet uveřejněných studií, které se zabývají hodnocením koncentrace žlučových kyselin u želv, je však doposud omezený a přesné vymezení souvislostí mezi změnami koncentrace žlučových kyselin v krvi a jednotlivými typy jaterních onemocnění u želv zatím chybí. Pro posouzení stavu jater se jeví jako vhodné určení plazmatické koncentrace biliverdinu. Toto vyšetření však zatím v našich podmínkách žádná laboratoř neprovádí.

V hematologickém profilu pacientů s chronickým postižením ledvin bývají pravidelně zjišťovány snížené hodnoty červené krevní řady (neregenerativní anémie), při zánětlivých onemocněních

může dojít k navýšení počtu leukocytů, azurofilie a monocytóza. U pacientů s postižením ledvin je pravidelným nálezem zvýšená koncentrace fosforu v plazmě a s tím související změna v poměru vápníku a fosforu. V důsledku ztrát albuminu bývá snížená koncentrace celkové bílkoviny a pozorujeme rovněž zvýšení aktivity AST a CK. Ke zvýšení hladiny kyseliny močové dochází většinou až v pokročilém stádiu onemocnění.

V období folikulogeneze jsou v periferní krvi zjišťovány zvýšené koncentrace vápníku, celkové bílkoviny, cholesterolu, triacylglycerolů a vyšší aktivita ALP. Při retenci snášky vajec nemusí být v biochemickém ani hematologickém profilu zaznamenány žádné výrazné odchylky, výjimkou může být zvýšená koncentrace fosforu při komplikacích vyvolaných distenzí močového měchýře.

K akutním případům u želv patří různá poranění, způsobená pokousáním jiným zvířetem či pádem, při kterých dochází k frakturám krunýře nebo končetin. I v takových případech je vhodné provést alespoň orientační hematologické vyšetření pro odhad ztrát krve. S ohledem na konkrétní situaci je však v mnoha případech dávána přednost posouzení zbarvení sliznic a provedení rentgenologického vyšetření. Příkladem akutního stavu je i přítomnost cizího tělesa v trávicím traktu nebo těžká obstipace. Před zahájením chirurgického řešení takových případů provádíme kompletní hematologické a biochemické vyšetření. Pokud jsou zaznamenány výrazné odchylky od běžných hodnot, je nutné pacienta před chirurgickým zákrokem nejdříve stabilizovat.

Tabulka č. 1 Hematologický profil nejčastěji chovaných druhů želv

Druh	Hemoglobin	Hematokrit	Erytrocyty	Leukocyty	Heterofily	Eozinofily	Bazofily	Monocyty	Lymfocyty
	g/l	l/l	10^{12}/l	10^{9}/l	10^{9}/l	10^{9}/l	10^{9}/l	10^{9}/l	10^{9}/l
Testudo hermanni Testudo graeca	37 - 81	0,25 – 0,40	0,50 -1,40	6,00 – 16,00	0,72 – 13,00	0 – 0,80	0	0 – 1,6	0,72 – 10,20
Trachemys scripta elegans	59 - 89	0,12 – 0,26	0,40 – 0,80	9,70	3,29	0	0,19	0,10	3,88

Tabulka č. 2 Biochemický profil nejčastěji chovaných druhů želv

Druh želvy	Celková bílkovina	Glukóza	ALP	ALT	AST	Kyselina močová	Ca	P
	g/l	mmol/l	µkat/l	µkat/l	µkat/l	µmol/l	mmol/l	mmol/l
Testudo hermanni Testudo graeca	20 - 75	6,2 – 14,4	1,0 – 18,5	<0,1 – 2,1	0,3 – 8,6	119 - 487	1,5 – 4,6	1,2 – 2,6
Testudo horsfieldi	24,3 – 68,5	8,9 -12,0	1,8 – 7,2	0,5 – 1,2	0,5 - 1,6	37,6 – 150,0	1,7 – 2,9	0,7 – 1.9
Trachemys scripta elegans	16,1 – 33,7	2,1 – 3,4	1,8 – 5,2	0,1 – 0,2	0,9 – 2,6	14,1 – 240,4	1,8 – 3,0	1,3 – 2,0

ADVANCES IN VETERINARY MEDICINE IN CHELONIANS

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Determination of morphologic characteristic of different peripheral blood cells and plasma chemistry profile of chelonians was the purpose of many studies. Research is still continuing on normal healthy tortoises, turles and terrapins as well as on patients suffering from different metabolic diseases[1-4]. The results of independent trials show a significant degree of variation due to different animal selection methods and technical differences in blood sample treatment. Blood that is exposed to heparin for several hours will usually not stain as well as slides made immediately after collection. Haemolytic destruction of chelonian cells by EDTA was observed. Classification of white blood cells in reptiles is inconsistent, because variable criteria have been used to categorise these cells. For the more exact characterisation of different types of blood cells in a group of healthy tortoises (*Testudo horsfieldi*) the commercial kits were used[4]. Ten different types of blood cells were determined: erythrocytes, thrombocytes, lymphocytes, monocytes, type-I cells (heterophils), type-II cells (eosinophils), type-III cells (azurophils), type-IV cells (basophils), type-Ia cells (toxic heterophils) and type-V cells (polychromatophil erythrocytes). The presence of eosinophils varies among species of reptiles. Eosinophils are present in chelonians as well as in crocodilians. Cells which are known as **heterophils** and **eosinophils** are present in chelonians. The main difference between them is the shape of granules. Sometime it could be difficult to distinguish the type of granules with the basic Pappenheim´s smears, because the cytoplasm is filled with them. We suggest that Granulocolor® would be appreciated in cases of special importance. Pappenheim method is absolutely sufficient for routine laboratory examination of tortoise's hemogram. The **heterophils** are characterized by oval or sharpened bright red cytoplasmic granules. The nucleus is bluish, in an off-centre position, mostly segmented. The **basophils** in reptile blood smears stand out by their exquisite blue granules filling in the cytoplasm and concealing even the nucleus situated in the central position. There is a risk of diluting and washing away basophilic granules in case when inconsiderate sample processing is applied. Inconsistent classification of **monocytes** and leukocytes with **azurophilic granules** in the cytoplasm has been a traditional issue in reptile haematology[1-2]. Differentiating

between the two types of leukocytes in reptiles takes thorough preparation of samples and much experience on the part of the person doing the count. The azurophils are mononuclear cells with a dominantly stained nucleus mostly in an off-centre position and blue-grey cytoplasm containing prominent azurophilic granules. Their shares in peripheral blood differ depending on the reptile genus and species[5]. Plasma chemistry profile in chelonians involves analysis of well separated plasma for the concentration of total protein, glucose, uric acid, alkaline phosphatase, alanine aminotransferase, aspartateaminotransferase, calcium, phosphorus and bile acids[6-9]. The hyperuricaemia may indicate renal damage in reptiles, but uric acid levels do not increase significantly until the extensive damage of kidney[10]. Hyperphosphataemia seems to be more reliable indicator of renal insufficiency in chelonians. Indeed, the phosphorus-calcium ratio could be a sensitive parameter for the diagnosis of renal disease. The peripheral blood concentrations of calcium, cholesterol and triglycerides may correspond to the metabolic activity requirement, which is lower in adult males than in females during the breeding season. The mechanism of hormonal control (oestrogen levels) for seasonal changes of calcium, cholesterol and triglycerid levels in blood of female chelonians is expected. There is still a lack of information concerning the existence of a feasible method for monitoring the chronic liver failure in reptiles. Studies that have been performed to characterize the bile acids in reptiles demonstrate that a variety of different bile acids are produced, nevertheless 3-α bile acids appear to be conserved amongst all reptile groups. As the clinical symptoms of liver failure in reptiles are unspecific the attention is aimed at methods of indirect diagnostics, such as diagnostic imaging, clinical hematology and plasma chemistry. Monitoring of bile acids concentrations in peripheral blood of chelonians is currently the centre of attention, because elevated values are supposed to be the results of liver diseases.[11]

Different techniques of veterinary endoscopy serve as feasible diagnostic and minimally invasive methods in exotic animal medicine and surgery[12]. Endoscopy in chelonians has developed from methods for sexing monomorphic reptiles[13]. It started to be very important method for clinical diagnosis and different endoscopic techniques were developed for reptilian patients. Nowadays it includes diagnostic endoscopy (with a form of guided biopsy) and minimally invasive endosurgery. Basic examinations of the mouth cavity and cloaca can be realised in chelonians under mild anesthesia[14]. General anesthesia (isoflurane) is required for advanced endoscopy – laparoscopy and cystoscopy. Carbon dioxide is the insufflation gas of choice. Reccommended coelomic pressure is 2 – 4 mm Hg, with a CO_2 flow rate of 0.5 – 1.0 l/min. Gas insufflation was

the preferred technique, but irrigation with sterile saline solution is better for cloacoscopy[14]. The most feasible coelioscopic approach in chelonians is via the small perforation of the prefemoral fossa skin. Cloacal endoscopy is feasible method of direct evaluation the contents of the urinary bladder and indirect control of the gonads (follicles)[15-17]. Not only visualization, but tissue biopsies are required to investigate unclear pathological situations. Endoscope-guided biopsies allow the surgeon to collect biopsies from particular locations, especially when only portions of the organ are altered. The most common biopsies in chelonians are those of the liver and kidneys.

Recently, coacoscopy and non-invasive cystoscopy have been described in details in chelonians[16-17]. It has been reported that dystocias in turtles present almost one third of all egg retention cases in reptiles but no final conclusions regarding the prevalence of dystocia among the various species of chelonians can be made[18]. Chelonian eggs could pass through urodeum into the urinary bladder, particularly if they have been induced to lay with oxytocin[19]. In comparison with dystocias in snakes or lizards, the diagnoses and treatments of egg retention in chelonians would be more difficult. The very common problem is the proper timing. Even with radiographs, the decision as to whether the eggs are retained and the treatment is necessary would not be easy. Adult female turtles and terrapins should produce eggs without any form of visual or physical contact with the male. To determine the optimal time for the veterinary intervention is nearly impossible. Although instigation of treatment on normal female is not recommended, delayed treatment is associated with the risk of complications. Several methods of invasive and non-invasive treatment protocols have been described[20-22]. If hormonal stimulation failed and eggs have been identified in the urinary bladder, surgical intervention by the plastral approach seems be the best choice. We found the method of cloacoscopy-assisted removal the eggs from urinary bladder as a valuable non-invasive alternative to a classical coeliotomy in chelonians. The transplastral approach has its advantages as well as disadvantages. The whole procedure is lasting for many minutes to hours (depending on the chelonian species and the plastron width) and it is followed with weeks of healing the surgical site. A soft-tissue approach to the coelom and urinary bladder would be valuable alternative method. The surgical site showed good healing by three weeks after surgery. The method is feasible in large species of chelonians only. Two methods should be used for distension of the cloaca and urinary bladder – insufflation with a gas (with air or CO_2) or irrigation with the sterile fluid solution. The later method is more feasible. Moreover,

aggressive flushing the bladder and cloaca with the sterile fluid is optimal for cleaning the mucosa and removal the small pieces of the broken shell and content of the destroyed egg.

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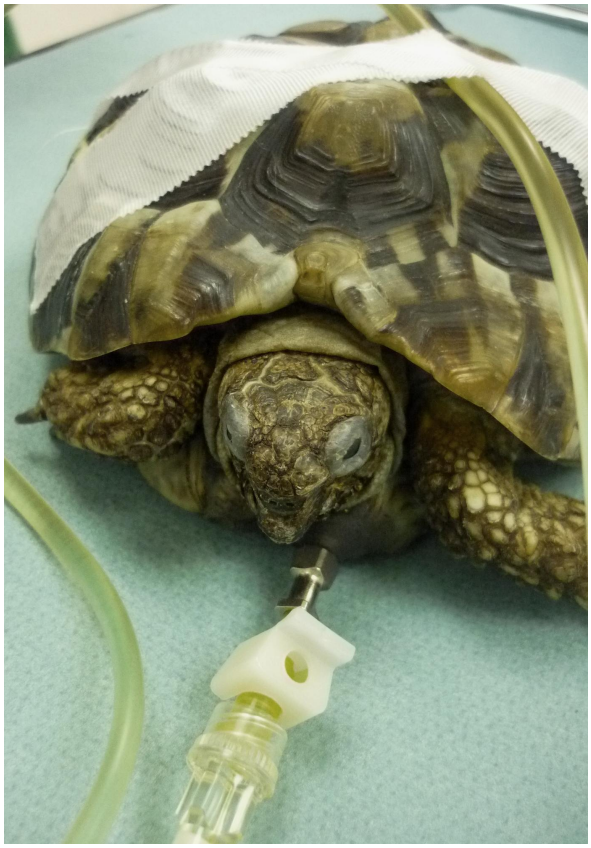

MEDICAL MANAGEMENT OF MARINE TURTLES

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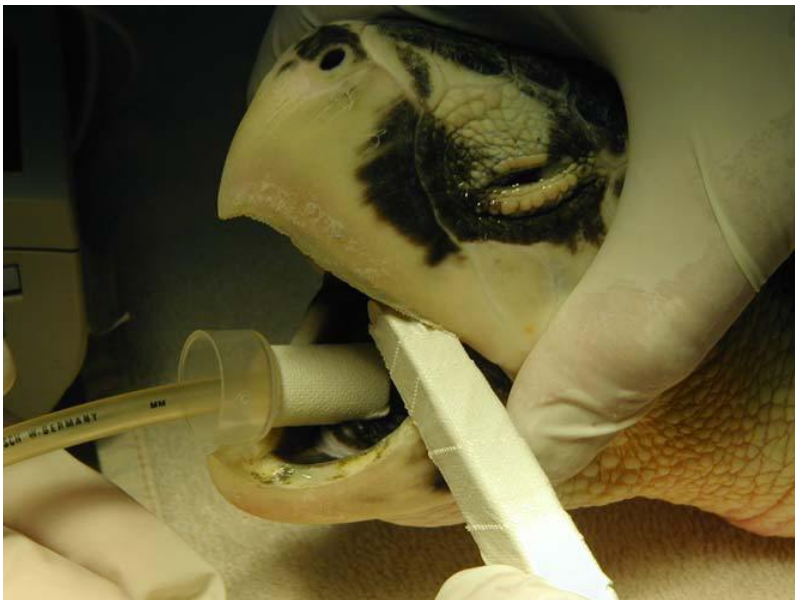

INTRODUCTION TO MARINE TURTLES

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Marine turtles are highly migratory turtles species that hatch on land, migrate offshore as hatchlings and usually avoid land until maturity when females return to nest in the same general regions where they hatched. With the exception of the Australian flatback turtle, all have a prolonged oceanic life stage. Most return to coastal waters to feed for a number of years before reaching puberty.[1] Leatherbacks typically remain oceanic until they are more than a meter in length. In all species, large juvenile and adult sizes migrate among feeding grounds seasonally. Generally, hatchlings and oceanic stage turtles are carnivorous, but as they age they tend to switch diets. For example, green turtles become mostly herbivorous, loggerheads tend to eat bottom dwelling mollusks and crustaceans, Kemp's ridley turtles feed on crabs, cnidarians, and

mollusks, olive ridleys tend to feed on surface or water column dwelling crustaceans, hawksbills feed on sponges and other reef dwelling organisms, and leatherbacks feed on jellyfish, large oceanic salps and pyrosomes. The diet of flatbacks is less well studied but appears to include mollusks, soft corals, and jellyfish.[2]

Once the turtles become sexually mature, they mate in the water either at mating grounds or in coastal waters near nesting beaches. Most mating is just prior to the each nesting season. Females store sperm during a nesting season but do not appear to store sperm more than 1 yr. Most sea turtles nest multiple times in a nesting season but do not nest every year. After breeding, they migrate to feeding grounds.[3]

Seven extant genera representing two families (Dermochelyidae vs. Cheloniidae).[4] The key characters include the presence or absence of complete longitudinal ridges along the carapace vs. a hard keratinous shell. All species are characterized by flipper-like fore limbs, a somewhat reduced shell, and a short neck. No sea turtle can retract its head or limbs into the shell. The sexes become externally dimorphic at puberty. A mature male has long, prehensile tails that extense well beyond the carapace margins and the vent is closer to the tail tip than to caudalmost part of the plastron. The tails of mature females (and immature turtles) are short and seldom extend beyond the carapace except in leatherbacks. The vent is close the plastron in females.[5] All species are long-lived and late-maturing. They produce many small eggs, small hatchlings (14-45 g at hatching) and parental care is limited to nest site selection. Most young do not survive to adulthood.[6]

The leatherback (*Dermochelys coriacea*) is the sole surviving species in a lineage (Dermochelyidae) that is at least 110 million yr old. The leatherback has a black carapace, usually with white spots, and a light plastron, the shell surface is smooth or even waxy. Leatherbacks lack distinct head scales as adults, have no claws, and have a thin keratin covering
on the jaws. Hatchlings are black with longitudinal stripes.[5,7] Scutes (the keratinous plates of the shell) overlie the bony shell. They are important in species identification. The carapacial scutes, named by location, are the *marginals*, along the carapace periphery; laterals (=costals), *vertebrals* (= centrals) along the dorsal midline. The single most cranial marginal just behind the neck is the *nuchal*. The most caudal marginal scutes are the pair of *supracaudals* (= postcentrals). On the plastron, the paired scutes (anterior to posterior) are the *gular*, *humeral*, *pectoral*, *abdominal*,

femoral and *anal*; unpaired *intergular* and *interanal* scutes are not always present. *Inframarginal* scutes, located between the carapace and plastron form the bridge.[5]

The six extant hard-shelled species (cheloniids) have persisted for between ~20-60 million yr depending on species.[4] The species can be distinguished from one another by the number and patterns of prefrontal scales on the head, the form of the head, the number of claws, and the numbers and carapace shape and pattern.[4,5,7] The scute patterns of the plastron are generally not diagnostic for species, but can serve as external landmarks for internal structures. The cheloniid marine turtle species that persist today are *Caretta caretta* (loggerhead), *Chelonia mydas* (green turtle), *Eretmochelys imbricata* (hawksbill*), Lepidochelys kempii* (Kemps ridley), *Lepidochelys olivacea* (olive ridley), and *Natator depressa* (flatback).[4,5,7] Several races of green turtles are occasionally listed as species (the Pacific black turtle and the Japanese green turtles are among these), but modern taxonomists define *Chelonia mydas* as a single species but with regional differences in morphology.[8]

Wyneken covers the anatomy of marine turtles in detail.[5] Briefly, there are a number of systems in marine turtles that are distinct from those of other turtles. Several are important when interpreting diagnostic images and understanding normal behavior. In the head, there greatly hypertrophied lachrymal glands (salt glands) dorsal, medial, and caudal to the eye, and lateral to the relatively small braincase. These metabolically active glands are often larger than the brain and eyes and play important roles in osmoregulation in these species that take in saltwater; they are responsible for the continuous production of salty, viscous tears.

The tongue is not protrusable. However it moves it is moved up and down in the mouth during ventilation and apnea, and during buccal pumping. The glottis is located in the central caudal aspect of the tongue and is usually closed except during ventilation, when the glottis maybe elevated to abut the choanae. Esophagus of all sea turtles is lined with large cone-shaped papillae that point caudally. These papillae are keratinous and trap food will excess seawater is expelled from the moth of nostrils during feeding.[5,7]

The bony shell of cheloniid marine turtles changes with age. In hatchlings, the carapace of formed of distinct ribs that articulate with modified vertebrae. As the turtles age, peripheral bones

ossify supporting the marginal scales, and intermembranous bone is laid down along the ribs (forming the pleural bones) in a medial to lateral progression. As a result, fontanels between the distal pleurals and the peripheral bones decrease in size with age. The hatchling plastrón appears as a ring of paired bones and a single entoplastron bone extending ventral to the heart. As the turtles age, all the plastron bones increase in size so that the plastron and bridge bečíme increasingly ossified with age. However, the central plastron remains as an unossified fibrous fontanelle.[5] In the leatherback the ribs and vertebrae remain as distinct structures throughout life. The plastron remains as a thin ring of bone. A networks of small thing bony plates, embedded in blubber form former a tough but flexible shell that lies superficial to the ribs and is covered by skin and more blubber.[5] The flipper skeleton in marine turtles is composed of elongated phalanges, metacarpals, and flattened carpals. The there is extensive fibrous connective tissue stiffening the flipper and limited muscles. The radius and ulna are short. When resting, it is common in all marine turtles for them to flex the flipper at the elbow so the flipper lies along side the shell and the tip points caudally. When swimming, the greatest movements are at the shoulder joint which lies inside the shell[5] and the flipper blade is extended and twisted. All marine turtles are considered to be imperiled species (listed by international entities such as the IUCN and most are also listed by government agencies). Marine turtles populations are managed worldwide. In U.S. waters, the National Marine Fisheries Service manages species and permits; the U.S. Fish and Wildlife Service manages the same species and permits when they are on land. Harvest and trade is prohibited in the United States and in many waters used by sea turtles.[7] As migratory species, marine turtles may pass through or reside in both waters that prohibit harvest and those that allow it.

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MEDICAL CARE OF SEA TURTLES

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Physical Examination

All animals should be weighed and the core body temperature recorded (measured from the cloaca). In addition, measurements such as straight and curved carapace lengths (SCL, CCL), straight and curved carapace widths (SCW, CCW) and straight plastron length (SPL) should be collected. These data provide a proxy for estimating maturity as well as a baseline for future assessment and progress.[1] A minimal data base of complete laboratory analysis (complete blood count, plasma chemistries), whole body radiographs and, when necessary, microbiologic cultures should be collected. In some situations (such as investigation of a disease outbreak) blood and serum should be banked. A comprehensive neurologic examination should be performed on all patients presenting with any neurologic abnormalities, spinal or head trauma. Chrisman et al. published a komplete neurologic assessment protocol based on her work with five normal, and six neurologically abnormal animals.[2]

Blood Collection

The University of Florida has an ongoing database available on their website (http://accstr.ufl.edu/blood_chem.htm) for Florida sea turtles. Normal values reported in recent literature.[3-5] Gicking et. al. reported plasma protein electrophoresis values of the Atlantic loggerhead sea turtle (*C. carretta*).6 Blood is best collected from either the supravertebral (dorsal occipital or cervical) sinus or the external jugular vein. Whitaker also discusses blood sample collection from the metatarsal vein. Either of these sites can be used for intravenous catheter placement. This author prefers a simple cutdown approach using the external jugular. A standard small mammal jugular catheter is used. Intravenous fluids, medications and if needed, parenteral nutrition can all be administered via the intravenous catheter.

Cytology

Cytologic analysis is another valuable tool for diagnosing various problems. Fecal analysis, via

floatation, sedimentation and direct saline smears should be performed on all new patients, as well as hospitalized and resident sea turtles on a regular basis. Coelomocentesis and CSF analysis are also commonly performed.

Diagnostic Imaging

Radiology, ultrasound, special imaging such as CT or MRI and nuclear scintigraphy are all valuable tools for diagnosing ill and injured sea turtles.[7]

Endoscopy

As with other species of reptiles, endoscopy can be used for many indications in sea turtles.8 Endoscopy allows a minimally invasive approach to various body areas that are not readily attainable by conventional means. In endangered species, this benefit alone justifies the expense of the equipment. This author routinely utilized both rigid and flexible endoscopy in sea turtles. Patients with green turtle fibropapilloma (GTFP) are endoscoped as part of their disease screening. In addition, flexible endoscopy is used for gastrointestinal (foreign bodies, obstructions) and respiratory cases. Coelioscopy is part of the standard data base analysis for all patients with GTFP. Many of these patients are debilitated, so general anesthesia is not necessary. At most, light sedation with a local analgesic at the site of insertion is all that is used. Lidocaine (2 - 4 ml 2%) is infused in the mid-prefemoral fossa bilaterally. Although it is possible to access both sides of the coelomic cavity from one entry point, bilateral visualization, and, the addition of a second entry site makes triangulation and use of adjuctive endosurgical devices more efficient.

Common Medical Problems

Surveys from various geographic locations around the world report similar conditions and problems.[9] In addition to natural disease, the sea turtles also have to deal with various forms of human impact.[10]

Trauma

Boat strikes, entrapment (entanglements) and shark bites are the most common cause for morbidity and mortality. A sequela of trauma, one that is often overlooked, is suffocation/drowning. Salt water drowning is a serious condition. Open wounds are best treated as such. Rarely do sea turtles present with fresh trauma (an exception being boat strikes where the

animal may be rescued immediately). As a result, debriding, packing (with sugar or honey when dry-docked), systemic antibiotics, analgesics and fluid support are appropriate. Depression fractures of the shell and skull should be gently elevated (generally with sedation or anesthesia). Loose pieces can be discarded because they usually will not reattach. Large, fresh fractures of the carapace, plastron or skull can be wired or plated. After fixation the animal should be dry-docked for several days to allow a surface healing before placement back in the water. If the wounds are old it is best to leave them as is (other than proper cleaning and debriding) as many will self-heal by secondary intention. This is evidenced by the many wild sea turtles seen with significant wounds that have healed without any veterinary intervention.

Fibropapillomatosis

Common to sea turtles in tropical waters world-wide is a proliferative disease known as green turtle fibropapilloma (GTFP). First described in 1938 in the green turtle (*Chelonia mydas*), the disease which is caused by a herpesvirus,[11] is now found in all species of sea turtles except for the leatherback (*Dermochelys coriacea*). The reason that the disease has crossed species and has become more prevalent is not known, however, the belief is that human-induced environmental factors (ocean warming, algal blooms, pollution, etc.) may be co-factors. This debilitating disease is characterized by the proliferation of cutaneous and visceral fibropapillomas. The fibropapillomas (FP) themselves are benign, but *en mass* these lesions debilitate the animals, causing anemia, immunosuppression and increased susceptibility to other disease.[12-14]

MRI has been shown to be superior and more sensitive at identification of internal FP,[15] which are most commonly found in the lung tissue. The liver, gall bladder, intestinal tract (both mesentery and mural), the kidneys and in the shell are other locations where FP has been seen. If internal FP exists, the patient's prognosis is grave and the animals are immediately euthanatized and necropsied. To date, it appears that the most effective method of control GTFP is surgical removal. The CO2 LASER has revolutionized removal of the masses in turtles. Minimal hemorrhage occurs in turtles, even in the larger lesions.[16] Post-removal, the LASER can be used to shrink and seal the site so that no sutures are necessary. The patients are started on antibiotics and analgesics postop, held out of the water for 24 hr, then placed back in their tanks. No formal studies have been conducted to determine the amount of time FP animals need to be held post mass removal before being released back to the wild. Empirically, patients are kept for 1 yr. If no

regrowth occurrs in that time, the animals are released. If FP returns, the procedure/time clock starts over.

Fish Hooks and Gastrointestinal Injury

The gastrointestinal tract, in some fashion, is commonly involved with sea turtle morbidity. Ingestion of fish hooks, fishing line, plastic bags, garbage and more can all contribute to both fast and slow deaths in these animals.[17,18] Fish hooks may be readily apparent in the oral cavity or if swallowed, with the line trailing from the mouth or cloaca. They may be occult and incidental findings on survey radiographs. Most hooks are designed to rust and eventually be eliminated by the animal. However, if they bečíme lodged, or if the line is pulled, it is possible for the hook to perforate the intestinal tract, causing a potential life-threatening coelomitis. Additional problems with hooks and lines include strangulation of one or both flippers with trailing line, erosions to the lateral commissures of the mouth from the line, and lead intoxication secondary to the ingested sinkers. In general, whenever there is a hook, there is usually a line. If the hook cannot be reached with a scope or the grasper and examination suggests perforation or a lead sinker is present, every attempt should be made to retrieve it surgically. Several approaches have been described.[19-21] If not possible to access the hook via the oral or supravertebral route, nearly the entire intestinal tract can be reached through either pre-femoral fossa.

Entanglement

Entanglements are common with fish hooks, nets and trap lines. The offending fibers need to be carefully removed, and the tissue tested for viability. Strangulation injuries, not uncommonly, devitalize limbs. Amputation is generally the only option in these cases. Intestinal impactions are likewise potentially lethal. These impactions can lead to buoyancy problems, pressure necrosis and intestinal perforations. Mineral oil gavages (0.25% body weight), lactulose (15 – 60 ml total dose per day) and psyllium (1 – 3 g per turtle per day) have been used with success at the MSTH. Avoid docusate (stool softener) as it has been shown to be toxic in reptiles.[22]

Flotation (Buoyancy) Abnormalities

There are several potential causes for “floater,” sea turtles that are unable to maintain proper buoyancy or have difficulty in diving. Trauma, which causes small or large tears in the lung, causes air to leak from the lung where it then gets trapped in the coelom. The lung tears act as “one-way” valves, and the air cannot return back into the lung. As a result, a pneumocoelom

develops and the animal floats and bobs at the surface like a cork. Any gas producing coelomic infection can result with the sea turtle floating. Intestina impactions, where ileus is a consequence of the blockage, frequently manifests with buoyancy problems.

Animals with neurologic damage, such as spinal trauma may not be able to dive properly, and appear to have buoyancy problems. Cold stunned animals, simply from weakness, may not be able to dive and as a result, are found floating on the surface.

There likely are many other causes that have yet to be elucidated. Grimm and Mader[23] performed a study with 33 healthy *C. mydas*, evaluating their coelomic pressure during the respiratory cycle. Mean resting coelomic pressures were 0 mm Hg. Peak inspiratory pressure was –3 mm Hg. Although not definitive, this study suggests that measurement of coelomic pressures may aid in diagnosis.

Treatment and correction of floating problems centers on identifying and reversing the cause. In general, if the problem can be found (lung injury, intestinal impaction, etc.) the floating can be corrected.

Contamination with Oil, Fuel and Other Toxins

Fossil fuels, heavy metals and environmental toxins are a problem for sea turtles worldwide.[24-28] Oil and the byproducts can be problematic both through ingestion and surface contamination. Heavy metals and toxins are ingested through water and food contamination. Not unexpectedly, high levels of toxicants are seen in areas of greater human populations. Lutcavage et al[24] reported that oiled turtles had up to a four-fold increase in white blood cell counts, a 50% reduction in red blood cell counts, and red blood cell polychromasia. Most serum blood chemistries (e.g., BUN, protein) were within normal ranges, although glucose returned more slowly to baseline values than in the controls.[24] Lutcavage et al[32] observed that the physiologic insults resolved with a 21-day recovery period, but pointed out that the long-term biologic effects of oil on sea turtles remain completely unknown.

Parasites

All wild sea turtles have a natural burden of endo- and ectoparasites.[29-32] In a healthy sea turtle host these rarely cause problems. However, when stressed with disease, trauma or environmental imbalances these parasites can cause pathology. Helminths such as trematodes and nematodes are a part of the normal flora. Monogenetic and digenetic trematodes are found in many species of

sea turtles. The monogenetic trematode *Lophotaspis vallei* resides mainly in the stomach and upper small intestine of loggerhead. They are not known to cause any damage to their hosts. Most digenetic trematodes have evolved to a level of mutual coexistence and little or no pathology is inflicted. These live in nearly every soft tissue organ in the body of their reptilian hosts, although most reside in the gastrointestinal tract. Spirorchiidae, group of digenetic flukes whose adults live in blood vessels and the heart, are pathogenic. Sometimes the adults may be numerous enough to block smaller vessels (capillaries) causing ischemia and damage to the tissue not being fed or oxygenated. The fluke eggs can accumulate, causing the same problem and some of those eggs that leave the gut are trapped by the host forming granulomas. Raidal et. al.[32] evaluated causes of morbidity and mortality in juvenile *C. mydas* off the coast of western Australia. Histopathologic examination demonstrated severe multifocal to difuse granulomatous vasculitis, aggregations of spirorchid fluke eggs and microabscesses throughout various tissues including intestines, kidney, liver, lung and brain. Adnyana, Ladds, and Blair[33] evaluated praziquantal in six spontaneously infected *C. mydas*. After treatment the animals were necropsied and evaluated. The absence of flukes in treated, but not control turtles, indicated that a 1 day course of treatment at a dose rate of 3 H 50 mg/kg body weight, administered orally, was effective. Jacobson et. al.,[34] in a phramacokinetic study in *C. caretta* found that oral administration of 25 mg of praziquantel/kg 3 times at 3-hr intervals may be appropriate for treatment of loggerhead sea turtles with spirorchidiasis. *Caryospora cheloniae*, a coccidial pathogen, was implicated in the deaths of 70 wild green sea turtles, *C. mydas*, in southeast Queensland, Australia over 6 wk in spring 1991. Based on the necropsy of 24 turtles, there was a severe enteritis or encephalitis.[31] Prior to this report, *C. cheloniae* had only been reported in farm-raised *C. mydas*. *Eimeria caretta* has been found in loggerheads, *C. caretta*, but has not been associated with pathology.[35] Marine leeches (*Ozobranchus margoi*) are found in large numbers on green turtles (*C. mydas*), Atlantic hawksbill (*E. imbricata*), loggerhead (*C. caretta*), and Atlantic ridleys (*L. kempii*). High populations of this leech usually are seen on severely emaciated turtles. Fresh water baths will usually suffice to rid the turtles of the leeches.

Infectious Diseases

Sea turtles are susceptible to a myriad of infectious diseases. Bacterial, fungal and viral disease are commonplace, especially in animals that have been immunocompromised from reasons

already discussed.[9,13,32,36-42] Signs vary with pathogen and condition, but, generalized debilitation is common to most.

Neoplasia

Neoplasia has been documented in wild sea turtles.43,44 Other than that discussed with the GTFP, treatment of neoplasia in sea turtles has not been reported.

Weak, Leathargic and Moribund

There are many reasons for a sea turtle to present in a weak, lethargic or moribund condition. These animals may wash up on the shore, or be found floating in the ocean. Systemic disease, heavy parasite burdens, toxins, trauma or blood loss due to trauma and starvation or combinations of these conditions, as well as others, can all be contributing factors. Cold Stunned Animal Present Obtunded or Moribund. Finding multiple animals in a similar condition within a short period of time is a cause for alarm. Two recent epizootics involving loggerheads (*C. caretta*) have been reported.[45,46] Dubbed "lethargic syndrome" and "debilitated loggerhead syndrome," these occurred in the fall/winter 2000-2001 and 2003 in Florida and along the Southeast Atlantic coast of the United States, respectively. There were many similarities in both events. In general, the strandings were emaciated, sub-adult loggerheads in various stages of ill health or were dead. Many of the sea turtles were covered with small barnacles on both their shells and skin. It is not uncommon to find epibiota (barnacles, bryozoans, and other encrusting organisms) on a sea turtle's shell; however, they are not usually found on the skin of healthy animals. Neurologic and electrophysiologic examinations, and pathologic changes within peripheral nerve specimens supported a diagnosis of either demyelinating polyneuropathy and/or neuromuscular junction transmission block.[45] Eighteen loggerheads were necropsied and histopathology indicated generalized and neurologic spirorchiidiasis (*Neospirorchis* sp.).

Evaluation for heavy metals, organophosphates and environmental toxins showed some minor perturbations from the normal in some of the affected animals, but none were considered high enough to be the sole cause of the outbreak. The authors concluded that the clinical signs and pathologic changes seen in the affected loggerheads resulted from combined heavy spirorchiid parasitism and possible chronic exposure to a novel toxin present in the diet of the loggerheads.[45]

Therapeutics

Principles of treatment for sea turtles should follow standard guidelines. Efforts are being made to establish sea turtle-specific drug dosages.[47-50] Following initial diagnostics and establishment of a treatment plan, the sea turtles should be housed in isolation pools. Dehydrated and parasite covered sea turtles should receive a soaking in a freshwater pool. The freshwater will assist in the correction of the dehydration and also help rid the animal of the ectoparasites. Normal seawater should be used in the rehabilitation and hospitalization tanks. The salinity should be approximately 35 parts per thousand (ppt). Chlorine can be added to the salt water (0.5 mg/L to achieve a lever of 0.5 parts per million [ppm]) to reduce bacterial and algal growth. The water should be tested as chlorine levels greater than 1.0 ppm can be irritating to the turtle's eyes.[4] The water temperature at subtropical facilities generally does not need to be regulated; however, at more tropical or more temperate location the water temperature may need regulation. Indole pools should be kept between 25°C and 30°C (77°F to 86°F). Water that is too cold can be immunosuppressive, depress appetite and delay healing, and water that is too warm can cause hyperthermia and also have other metabolic consequences.[4] Partial shade is required in outdoor tanks. Medications are generally administered either orally, intramuscularly (i.m.), intracoelomically (i.ce.) or intravenously (i.v.). The general adage "if the mouth works, use it," does apply to sea turtles. However, in animals that are debilitated, have gastrointestinal stasis or are large and dangerous, the per os (p.o.) route may not be practical. Intramuscular injections can be administered in the pectoral or pelvic limbs. I.ce. medications and fluids are best administered in the prefemoral fossa cranial to each thigh.

Anesthesia

Sea turtles present a unique challenge as anesthetic patients. These animals are capable of shunting their pulmonary circulation (dive response) and holding their breath for several hours. Sea turtles can be pre-medicated prior to induction. For potentially painful procedures, administration of analgesics prior to the event is recommended. Moon and Stabenau51 discuss un-premedicated direct orotracheal intubation and isoflurane induction in *L. kempii*. An isoflurane concentration of 3.4 ±0.3% provided anesthetic induction in 7 ±1 min. The mean duration of the recovery was 241 ±31 min. The duration of the recovery phase was not affected by the duration of anesthesia, type of carrier gas, method of ventilatory weaning, or use of selected pharmacologic agents. The recovery phase was characterized by hypoxemia, progressive

acidemia, hypercapnia, and lactic acidosis. Awakening in the turtles was preceded by a characteristic tachycardia and tachypnea. All sea turtles recovered from isoflurane anesthesia without apparent adverse effects within 24 hr. Chittick et. al used pre-medications in their study on 13 loggerhead sea turtles.[52] Anesthesia was induced with a premedication cocktail of medetomidine (50 μg/kg, i.v.) and ketamine (5 mg/kg, i.v.), the patient was intubated and then maintained with sevoflurane (0.5% to 2.5%) in oxygen. Sevoflurane was delivered with a pressure-limited intermittent-flow ventilator. Administration of sevoflurane was discontinued 30 to 60 min prior to the end of the surgical procedure. Atipamezole (0.25 mg/kg, i.v.) was administered at the end of surgery. Median induction time was 11 min (range, 2 – 40 min, n = 11). Median delivered sevoflurane concentrations 15, 30, 60, and 120 min after intubation were 2.5 (n = 12), 1.5 (n = 12), 1.25 (n = 12), and 0.5% (n = 8), respectively. Heart rate decreased during surgery to a median value of 15 beats/min (n = 11). End-tidal partial pressure of CO2, ranged from 2 to 16 mm Hg (n = 8); median blood gas values were within reference limits. Median time from atipamezole administration to extubation was 14 min (range, 2 – 84 min, n = 7). The authors concluded that a combination of medetomidine and ketamine for induction and sevoflurane for maintenance provides safe, effective, controllable anesthesia in injured loggerhead sea turtles. At surgical planes of anesthesia some form of mechanical ventilation is necessary. The tidal volume can be estimated to be approximately 50 ml/kg. Ventilatory pressure should not exceed 15 cm H2O and the rate should be approximately 2 – 8 breaths/min.3 The anesthetic carrier gas should be either 100% Oxygen or a 50:50 mixture of oxygen and nitrous oxide. If nitrous oxide is used it should be discontinued at least 5 min prior to the end of surgery. In general, reflexes such as jaw tone, palpebral reflex and flipper pinch can be a crude estimate of anesthetic depth. Heart/pulse rate may be monitored with a ultrasound, a pulse oximeter or Doppler flow detector. None of these techniques offer information regarding the physiologic status of the animal while anesthetized, but do provide a means to monitor heart rate and trends. An electrocardiogram (ECG) can be used, however, since the reptilian heart can beat even after death, the tracing may be of little value. Post anest etic recovery may take from minutes to hours, depending on the pre-anesthetic and anesthetic used, the length of the procedure, the patient's body temperature and individua variation. An intubated patient should never be left unattended. Prior to recovery, there is generally an increase in both spontaneous respirations and heart rate.[51]

Pain Management

There have been no published, refereed studies on the use of analgesics in sea turtles.

Surgery

Surgical techniques are similar to those employed in other chelonian species. Just as in other water turtles, return of sea turtles to the aquatic environment will be based on procedure, anesthesia and risk assessment. Shell repair is also similar to other chelonians.[53,54] An exception here is that the marine environment tends to favor healing for small or open wounds. This author tends to leave small cracks and shell deficits open to allow healing by secondary intention. Sea turtles have an amazing ability to heal providing supportive care and attention to sepsis prevention is addressed. An example is that of a green turtle (*C. mydas*) with a plastron wound that opened to the coelomic cavity.[55] Over the course of 4 wk and an abundance of nursing care, the turtle was returned to a pool. Evaluation 1yr later showed that the patient made a komplete recovery. Of interest to note is the effect of the marine environment on skin sutures. Govett et. al. evaluated tissue response to four different, absorbable skin sutures.[56] Chromic gut, polyglyconate, polyglactin 910, and poliglecaprone 25 were used in 258 turtles to close a wound produced at the time of laparoscopic sex determination. Gross and histologic tissue reactions to the different suture types were analyzed. Histologically, polyglactin 910 produced a greater tissue reaction than any other suture type. Poliglecaprone 25 and polyglyconate caused the lest tissue reaction of the four suture types examined in sea turtle skin. All skin closures are performed using a horizontal, everting pattern. In areas where tension, motion or dehisence is a concern, plastic stints (made from intravenous line tubing) are added for additional strength. The stints are removed after 3 – 4 wk, with the skin sutures being removed 2 or more wk later. Several sea turtle-specific surgeries have been described in the literature.[19-21,57] In general, surgical approaches are similar to those for any other chelonian. The colomic cavity can be approached from either prefemoral fossa. In small animals, endoscope assisted visualization of internal organs or endosurgery may be the techniques of choice. Amputations are generally performed at either the shoulder or hip joints, although, distal amputations are possible. Sea turtles handle amputations well. After surgery the animals are placed in large pools to provide rehabilitation and exercise. It does not take the animals long to learn to swim and steer, regardless of whether the amputation was front or rear.

Necropsy

Because of the intrinsic value of these animals, all sea turtles that die or are euthanatized must be necropsied. In some regions performing a complete post mortem examination, documenting the findings, and reporting them to the appropriate authorities is a legal requirement. A comprehensive, sea turtle-specific necropsy protocol is available via the Internet at the University of Florida's website http://www.vetmed.ufl.edu/sacs/wildlife/seaturtletechniques/ and a downloadable, printable necropsy form can be found at http://www.vetmed.ufl.edu/sacs/wildlife/seaturtletechniques/necropsyreport2.htm.

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INTERPRETATION OF PLASMA BIOCHEMICAL, HEMATOLOGIC, ACID-BASE, AND BLOOD GAS DATA OF SEA TURTLES

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Plasma biochemical, hematologic, acid-base, and blood gas analyses are a routine part of the medical management of sea turtles, and are also important for health evaluation of free-ranging sea turtles. This document provides an introduction to interpretation of such data, but assumes that the clinician has general familiarity with clinical pathology of reptiles and blood-gas interpretation. The reference list provided herein is not exhaustive, and the reader should seek additional original references as cited in the manuscripts listed below. It assumed that the leader is aware of sea turtle medicine chapters in the general reptile and zoological medicine texts, and these are not specifically cited herein. The University of Florida maintains a useful database of sea turtle biochemical values (University of Florida. Establishing plasma biochemical and hematocrit reference intervals for sea turtles in Florida. Available at http://accstr.ufl.edu/blood_chem.htm. Accessed April 27, 2009). Blood may be collected from sea turtles from several sites including the dorsal cervical sinus (most common), post-occipital sinus, sub-carapacial sinus, interdigital veins, and dorsal coccygeal vein. Sampling arterial blood is difficult without surgical cut-down, which is rarely done in clinical patients. As such, the majority of sea turtle literature (including acid-base studies) is based on data acquired from venous samples. Chelonian hematology and plasma biochemistry results vary greatly with different laboratory methodologies, venipuncture sites, species, age, gender, and season.[1-7] Comparisons of clinical data to the published literature must be done thoughtfully. There are many published accounts of hematologic, acid-base, and plasma biochemical data for sea turtles. These studies provide an abundance of comparative data that may be of use to the clinician. However, some of these studies describe apparently healthy freeranging animals, while others describe debilitated animals in rehabilitation.[1,2,6-13] Some studies provide data for anesthetized sea turtles, turtles caught in trawl nets, nesting females, turtles that have completed rehabilitation, ill wild turtles, or long-term captive turtles.[12-18] It is important that the clinician interprets this data within the context of the conditions of the study. Heparin is often used as an anti-coagulant for sea turtle blood, and most sea turtle biochemical studies are based on heparinized plasma samples, but

serum may also be used. No significant differences were found in leatherback turtle (*Dermochelys coriacea*) plasma biochemical values between samples collected in sodium heparin vs. lithium heparin.[11] Blood should be processed immediately after collection if possible; however, a recent study in loggerhead sea turtles (*Caretta caretta*) found no significant changes in plasma biochemical parameters (except for gamma glutamyltransferase) when whole blood was stored under refrigeration for 24 hr prior to centrifugation and analysis.[19] As in many reptiles, sea turtle blood testing often lacks sensitivity and specificity. However it appears that some analytes may be very useful for assessing the physiologic status, prognosis, and clinical progress of sea turtles. Several of these analytes are discussed below. For those analytes not discussed, the clinician should make conclusions based on standard medical principles until additional data is available. Healthy carnivorous sea turtles may have much higher blood urea nitrogen (BUN) levels than terrestrial vertebrates; however, debilitated sea turtles often have relatively low BUN levels.[2,3,6-9,12,13] While the reason for low BUN levels is unclear, it is possible that it is related to decreased urea production under conditions of anorexia and/or reduced hepatic function. Interestingly, sea turtles sometimes show increasing BUN values during rehabilitation even though they have not yet accepted food, which suggests that anorexia alone may not explain the reduced BUN seen at admission.[12] BUN values may be low at the time of admission, and increase over the course of rehabilitation. Relatively low BUN values are seen in nesting female leatherback turtles in comparison to leatherbacks in northern Atlantic feeding grounds (Innis, unpublished data, October 2008).[11,16] Green turtles (*Chelonia mydas*) are more herbivorous than other sea turtle species and have relatively lower BUN values.[7-9,17] The phenomenon of low BUN values in ill turtles (some of which have poor renal function) vs. high BUN values in healthy turtles (with normal renal function) is counterintuitive to clinicians that work mainly with mammals. One study found higher BUN values in green turtles with fibropapillomas compared to unaffected green turtles.[17] Uric acid values tend to be low in healthy sea turtles (generally <2 mg/dl).[2,3,6-9,12,13] In coldstunned Kemp's ridley turtles (*Lepidochelys kempii*), severely elevated uric-acid values are associated with a poor prognosis.[13] Specific studies to correlate uric values and renal function or pathology in sea turtles have not yet been conducted. Both hypoglycemia and hyperglycemia are common in ill sea turtles.[13] Possible causes of hypoglycemia in these cases include exhaustion, prolonged anorexia, and sepsis. Hyperglycemia in reptiles may reflect a stress response, overcompensation of gluconeogenic mechanisms, liver disease, pancreatic disease,

and/or exogenous dextrose administration. In general, glucose values of surviving turtles stabilize over several weeks. In contrast to many other vertebrates, healthy sea turtles often have lower total plasma kalcium concentrations and often have an inverse calcium:phosphorus ratio.[2,3,6,9,11,13,16] It is possible that this inverse calcium: phosphorus ratio is normal for these species. However, the clinician should also consider other causes including juvenile bone growth, nutritional secondary hyperparathyroidism, etc. Elevated phosphorus values are sometimes seen in very ill sea turtles, presumably related to poor renal function. These cases often show simultaneous elevation of uric acid, potassium, and sodium values. Ionized calcium values have recently been reported for Kemp's ridley turtles, and were lower than those reported for several other reptile species.[12] Low total protein and albumin levels have been reported in injured loggerhead sea turtles and cold-stunned Kemp's ridley turtles.[13] While the cause of hypoalbuminemia is unknown, it could be the result of prolonged anorexia, reduced hepatic function, protein-losing nephropathy, enteropathy, etc. Protein electrophoresis data have been published for several sea turtle species, but clinical use of this data has not yet become routine.[11] Globulin often represents 50-70% of the total protein value in sea turtles.[2,3,6-9,12,13] Clinical impressions indicate that globulin levels may increase in sea turtles with inflammatory and infectious diseases, but specific published data is lacking at this time. A variety of plasma enzyme levels are often measured, however, the tissues of origin for these enzymes have not been specifically determined in sea turtles. In snakes and lizards these enzymes are found in a variety of tissues, thus may be non-specific indicators of tissue injury. In sea turtles, lactate dehydrogenase, aspartate aminotransferase, alkaline phosphatase, and kreatine kinase appear to more useful than gamma glutamyltransferase and alanine aminotransferase.[13] Elevated serum/plasma levels of tissue enzymes have been noted in cold-stunned or injured turtles. [13] In some cases, enzymes remain moderately elevated during rehabilitation. Possible reasons for persistent enzyme elevation in these turtles include rapid tissue growth, undetected pathology, and exertion due to confinement in captivity. Interpretation of electrolyte values is done using standard principles. Debilitated sea turtles often have elevated sodium, potassium, chloride, magnesium (ionized and total), and ionized kalcium concentrations, presumably due to a combination of dehydration, reduced renal and salt gland function, and ingestion/aspiration of sea water.[12,13] However, a spectrum of derangements is seen, and some individuals may be hypokalemic or hypocalcemic. Severe elevations of podium and potassium values are often

associated with mortality.[13] Consistent with observations in other reptiles, values for bilirubin, gamma glutamyltransferase, alanine aminotransferase, and creatinine are generally low in sea turtles, and these parameters are generally considered to be of little clinical importance in reptile medicine.[2,3,6-9,12,13] Sea turtle hematocrit values are generally in the high-20's to mid-30's percent.[2,3,5,11-13,17] High hematocrit values are often seen with dehydration (often with high sodium and chloride values). Anemia can be seen in sea turtles after acute trauma (blood loss), or with chronic pathology (e.g., bacterial or fungal pneumonia). Characterization of anemia follows standard principles. The regenerative response to anemia may take weeks to months. Studies of white blood cell counts in sea turtle species have reported values ranging from 2,000-25,000 cells/µl.[2-5,10,11,13,17,20] Initial white blood cell counts for cold-stunned turtles are often elevated in comparison to convalescent values.13 High white blood cell counts may reflexy inflammation, immune response, physiologic stress, and/or systemic pathology. Most hematology studies of sea turtles describe the majority of leucocytes as heterophils, followed by moderate numbers of lymphocytes, and smaller numbers of monocytes, eosinophils, and basophils. However, there is some inconsistency and disagreement over the nomenclature of sea turtle leucocytes. For example, in one study of Kemp's ridley turtle hematology, the granulocytes are described as large eosinophils and small eosinophils, rather than heterophils and eosinophils.[20] Some reports in loggerhead turtles describe azurophils rather than monocytes.[2] A number of acid-base and blood gas studies have been conducted for sea turtles. Initially, most of these studies were conducted to study the basic physiologic responses to diving, temperature variability, etc. More recently, the effect of a variety of stressors, including forced submergence, experimentally induced hibernation, general anesthesia, and trawling has been evaluated.[12,14 16,18] Older acid-base and blood gas studies utilized analyzers that were calibrated directly to the body temperature of the turtle. Newer studies generally use patient-side analyzers, which perform analysis at 37ºC. While the subject of temperature-correction of acid-base and blood-gas data for ectotherms is somewhat controversial, the majority of sea turtle physiology and clinical medicine papers utilize mathematical formulae to correct the data for the turtle's body temperature (reporting both the raw data, and temperature-corrected [TC] data).[12,14-16,18] These formulae, as well their derivation, can be found in several recent publications.[12,14,18] In general these formulae are considered to be more appropriate for sea turtles than mammalian-based formulae that may be used by the analyzer. However, two sea turtle studies have reported that analyzer-corrected values for pH and

pCO2 were similar enough to manually calculated values to be clinically useful. In contrast, analyzer-corrected values for pO2 did not agree well with manually calculated values, differing by up to 70%.[12,14] In addition, one must ignore inherently inaccurate data generated by the analyzer. For example, reptile hematocrit values generated by automated analyzers are often inaccurate when compared to the manual hematocrit; and any data derived by using this inaccurate hematocrit is also inaccurate (e.g., hemoglobin). Other analyzer-calculated values that rely on principles of mammalian physiology (e.g., oxygen saturation of hemoglobin) are also inherently inaccurate for sea turtles. Venous pHTC in healthy sea turtles at 25ºC is generally 7.4-7.6, varying somewhat aminy different studies.[12,14-16,18] Acidosis (both respiratory and metabolic) may be seen in ill, injured, or anesthetized turtles. Elevated venous pCO2 values are common. Since pCO2 and pO2 values vary directly with temperature, one must interpret the results relative to the temperature of the turtle. For example, in Kemp's ridley turtles at 25ºC, the pCO2TC is generally around 30 torr; however, in cold-stunned Kemp's ridleys at 10ºC, the pCO2TC that is associated with a good prognosis is lower (e.g., 20 torr), and a pCO2TC of 30 torr is considered to be elevated.[12] Lactate concentrations have been reported for sea turtles exposed to moderate stressors such as pound net capture and general anesthesia as well as more physiologically demanding stressors such as long voluntary dives, trawl net capture, or experimental forced submergence.[12,14,16,18] High lactate concentrations have been described in cold-stunned sea turtles and anesthetized sea turtles, presumably due to anaerobic metabolism caused by hypoventilation and reduced perfusion.[12,14,16]

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Medical management of cold-stunned sea turtles

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Abstract: "Cold-stunning" of free-ranging sea turtles in the United States and Europe has been reported for many years, and typically occurs when water temperatures drop below 12ºC (54ºF). A high percentage of cold-stunned turtles are already dead when they are found stranded on the beach. Rehabilitation of live cold-stunned turtles may be successful. Cold-stunned turtles are generally affected by metabolic disturbances, dehydration, and renal failure. Common plasma biochemical abnormalities include hypoglycemia, hyperglycemia, hypernatremia, hyperchloremia, hyperphosphatemia, hypocalcemia, hypoproteinemia, hypoalbuminemia, hypokalemia, hyperkalemia, hypermagnesemia, reduced BUN, and elevated plasma enzyme concentrations. Pathology of the alimentary, respiratory, integumentary, nervous, sensory, and urogenital systems is common. Medical management is directed at correction of metabolic and respiratory derangements, gradual increase of body temperature, and treatment of systemic pathology. Diagnostic tests including needle aspirate, biopsy, tracheal wash, bacterial and fungal culture, ultrasound, endoscopy, magnetic resonance imaging, computed tomography, and nuclear scintigraphy may be used to thoroughly evaluate the patient during rehabilitation.

Key Words: cold-stunning, sea turtle, pathology, Kemp's ridley, *Lepidochelys kempii*

"Cold-stunning" of free-ranging sea turtles in the United States and Europe has been reported for many years.[1-9] While the reasons for these cold-stun events are not completely understood, it is thought that geographic, oceanographic, and meteorologic conditions are involved.[5] Cold-stunning typically occurs when water temperatures drop below 12ºC (54ºF).[1-5] In some areas, predictable late autumn cold-stun events are seen when turtles fail to leave summer foraging grounds for warmer waters.[1-3,5-7] Cold-stunning may affect all species of sea turtles, although leatherback turtles are much less likely to be affected as they can maintain their body temperature above that of the ambient water, and are known to forage in cold waters. In the northeastern United States, the most commonly affected species is Kemp's ridley turtle (*Lepidochelys kempii*). Cold-stunned turtles become weak, float on the surface of the water, and are washed onto

beaches.[9] Unfortunately, 35-85% of cold-stunned turtles are already dead when they are found on the beach.[2,4,10]

A grading scale for cold-stunned turtles has been proposed, ranging from Class I turtles which are relatively alert and active, to Class IV turtles which are moribund.[11] The success rate for rehabilitation of cold-stunned turtles is variable, but is generally in the range of 50-70%.[2] Excluding turtles that die within the first three days of presentation, the success rate approaches 90%.[12] Many turtles that die within the first three days show no indication of being alive other than weak cardiac contraction as seen with echocardiography. One could argue that some of these animals are clinically dead, and thus should not necessarily be considered treatment failures. The authors do not classify an animal as dead until no cardiac activity at all is visible with echocardiography, or until obvious rigor mortis is noted.

Upon collection from the beach, cold-stunned turtles should be maintained at the temperature at which they were found, and transported to a rehabilitation center. Experience has shown that immediate, rapid warming results in higher mortality than controlled, gradual warming as discussed below.

At New England Aquarium, the following protocol is completed for each turtle upon hospitalization. Examination is conducted rapidly, and ideally in a cool room, to prevent warming of the patient. Core body temperature is measured via cloacal probe. Mentation, activity, and attitude are assessed. Heart rate is determined by Doppler monitor or echocardiography. Bradycardia is common, with heart rate at admission generally 1-12 beats per min. Respiratory rate is determined visually. Apnea or severe bradypnea is common, with respiratory rates at admission often between one breath per min to one breath per 15-20 min. Physical examination is conducted, standard body measurements are taken (carapace length, plastron length, etc), and the turtle is weighed. Oral exam is performed, and the oral cavity is cleaned, as there is often sand in the mouth and pharynx. Fluorescent corneal stain is used to detect corneal damage or ulceration, which is commonly seen secondary to desiccation, mechanical trauma, and predation. Traumatic wounds and fractures are common. Hydration status is assessed subjectively and objectively (see below). Nutritional status and body condition is assessed. Euthanasia may be considered in cases of severe brain or spinal trauma; severe, bilateral ocular trauma; or if multiple flippers are

amputated. Be conservative with euthanasia decisions as turtles can often heal from severe injuries. Any sea turtle euthanasia plan must be discussed and approved by federal authorities.

Hematocrit, electrolytes, blood glucose, acid-base, and blood gas status should be assessed at admission using a point-of-care analyzer (see Interpretation of Plasma Biochemical, Hematologic, Acid-Base, and Blood Gas Data of Sea Turtles in this volume for additional information).[13] In the author's experience, complete blood count (CBC) and complete plasma biochemical panels are not entirely necessary or useful during the first few days of hospitalization, but are very useful, and used most economically for longer-term monitoring of the patient.[14] CBC and chemistry panels may be performed every one to two weeks for several weeks, and then monthly for the remainder of hospitalization. Clinical laboratory findings often indicate metabolic disturbances, dehydration, and renal failure. Common plasma biochemical abnormalities include hypoglycemia, hyperglycemia, hypernatremia, hyperchloremia, hyperphosphatemia, hypocalcemia, hypoproteinemia, hypoalbuminemia, hypokalemia, hyperkalemia, hypermagnesemia, reduced BUN, and elevated plasma enzyme concentrations.[10,13-15] Lactate levels may be elevated secondary to hypoventilation, poor perfusion, and anaerobic metabolism.[13] Complete blood cell counts often reveal heterophilic leucocytosis.[14,16] Blood gas evaluation generally reveals metabolic and respiratory acidosis.[13] Hematocrit, electrolytes, blood glucose,
and blood gas values are monitored daily to every other day for the first two weeks of convalescence. Acid-base and electrolyte abnormalities often resolve with general supportive care. Persistence and exacerbation of acidosis, hypercarbia, hyperkalemia, hypernatremia, hyperphosphatemia, and hyperuricemia are negative prognostic signs.[14] Common signs of improvement include resolution of anemia, normalization of pH, blood gas, and electrolyte values, resolution of leucocytosis, increasing BUN, and normalization of plasma enzyme levels.[10,14] In a recent retrospective study of 208 cold-stunned Kemp's ridley turtles, turtles that died had significantly greater plasma concentrations of sodium, chloride, potassium, calcium, phosphorus and uric acid than turtles that survived.[14] For survivors, convalescent values for BUN and calcium were significantly greater than initial values, while convalescent values for glucose, sodium, and uric acid were significantly lower than initial values.[14]

The initial treatment plan is based on physical examination, blood glucose, electrolyte, and blood gas status. Turtles should initially be kept within 2-3ºC (3-5ºF) of their core body temperature. Animals are placed on towels or padding in an incubator that increases the core temperature to 13ºC (55ºF) over the first 24 h. The majority of animals are given hypotonic balanced electrolyte and glucose solution (1 part lactated ringer's solution, 2 parts 2.5% dextrose/0.45% saline) at 10-20 ml/kg SC. This is often divided between a morning and afternoon dose. Fluid temperatures are kept at 10-13ºC (50-55ºF) initially to prevent rapid warming. Subsequent fluid boluses are adjusted to the patient's current core body temperature. Some clinicians prefer to use intracoelomic fluids, while others feel that intracoelomic fluids are not absorbed well, and may cause iatrogenic coelomitis. There is little objective data in this regard. The choice of crystalloid products for use in sea turtles is largely empirical, but should be based on the current metabolic and hydration status of the patient. Dextrose-containing fluids are avoided in hyperglycaemic patients. Hypoglycemic patients (plasma glucose<80 mg/dl) may benefit from intravenous 50% dextrose (1ml/5kg).[11] The authors prefer to dilute dextrose to a strength of 10% with 0.9% saline prior to infusion. Sodium bicarbonate, calcium gluconate, potassium chloride, etc. may be added to fluids to address physiologic derangements as needed. In animals with very poor cardiac contractility and severe bradycardia, atropine is often effective. For apneic animals, doxapram is often effective. Doses and routes of administration of such drugs are based on principles of small animal emergency medicine, and are determined at the discretion of the clinician. Intravenous boluses of these drugs can be easily given, and are frequently used by the author. Endotracheal intubation and positive pressure ventilation may be required for hours to days. All turtles get a supervised shallow freshwater bath adjusted to their core body temperature within the first 24 h of hospitalization, and are evaluated for their ability to move and swim voluntarily. Stronger turtles may be given access to shallow seawater within 24-48h, while weaker turtles continue with shallow freshwater bathing until their hydration is improved. Freshwater helps to remove epibiota, and also hydrates the patient if ingested. Each turtle receives a "swim plan" according to its activity level, attitude and overall physical condition. Supervised swims are permitted in shallow pools until the animals are strong enough for deep water activity. After the first day of care, patient temperature is raised by 3ºC (5ºF) per day until the temperature reaches 25ºC (78ºF). More rapid warming may exacerbate acidosis and hyperkalemia. Parenteral

antibiotics +/- antifungal therapy are initiated early in the course of rehabilitation due to the high incidence of bacterial and fungal infections in these presumably immunocompromised patients.

All turtles should have dorsal/ventral, anterior/posterior, and lateral whole body radiographs and flipper radiographs after the core temperature has been stabilized to room temperature. Periodic blood culture should be considered throughout rehabilitation. *Enterococcus* sp. septicemia is seen has been documented in approximately a dozen cold-stunned Kemp's ridley turtles in the past five years (Innis et al, unpublished data, January 2009). Cold-stunned sea turtles are generally not fed until their hydration and electrolytes have normalized and they are able to swim on their own (generally 1-2 wk). These patients are often affected by gastrointestinal stasis, and premature feeding can cause additional complications. Body condition, attitude and activity level should be taken into account prior to instituting forcefeeding. In many cases turtles will begin feeding in several weeks voluntarily. Initially, only small amounts of fish fillet are offered. Once regular defecation is observed, and radiographs reveal absence of gastrointestinal stasis, larger meals may be offered. Cold-stunned turtles may be affected by pathology of the alimentary, respiratory, integumentary, nervous and sensory, and urogenital systems.[17,18] Necrotizing enterocolitis and bacterial or fungal pneumonia are frequently encountered. Parasites and parasitic lesions may be seen. In many cases, however, the extent and severity of pathologic and parasitic lesions are minimal, suggesting that other factors such as metabolic, respiratory, and electrolyte derangements, hypothermia, and drowning likely cause mortality in many cold-stunned turtles.[17] Diagnostic tests including needle aspirate, biopsy, tracheal wash, bacterial and fungal culture, ultrasound, endoscopy, magnetic resonance imaging, computed tomography, and nuclear scintigraphy may be used to thoroughly evaluate the patient during rehabilitation.[12,19,20] Osteolytic appendicular skeletal lesions are sometimes seen during rehabilitation of cold-stunned turtles, and may be associated with joint swelling and lameness.[19,20] Such lesions often develop after one to two months of hospitalization, and may or may not be associated with bacterial osteomyelitis.[20] These lesions have some similarity to phalangeal lesions seen in humans after frostbite injuries. Monitoring of lesions through serial radiographs and nuclear scintigraphy may be useful.[19,20] Such lesions generally re-model and clinically resolve over a four to six month period.[19,20] Serial radiographs and computed tomography have also proven useful for monitoring lung lesions of cold-stunned turtles.

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Dystocia in Australian freshwater turtles

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Introduction

Dystocia in captive Australian freshwater turtles is reasonably common. The condition has been categorised as obstructive or non-obstructive in nature. [1] Obstructive dystocias usually involve the inability to pass eggs through the oviduct and cloaca due to anatomical changes relating to the egg or the gravid female. Oversized, fractured, ectopic or deformed eggs may contribute to dystocia. Maternal abnormalities include pelvic abnormalities, oviductal stricture, or other factors such as abscesses, cystic calculi, or eggs sticking together. It is often difficult to establish the aetiology of non-obstructive dystocias. Many are thought to be due to poor husbandry, including incorrect temperature, inadequate or no available nesting site, malnutrition, dehydration, or infection. [2] In captive reptiles poor physical condition including obesity and poor muscle tone can contribute to dystocia. Injury is rarely a contributing factor to dystocia in captive turtles.

Free living turtles

In Australia free living freshwater turtles (*Chelodina* spp.) are commonly presented for treatment of injuries, usually sustained as a result of being hit by motor vehicles. It is the impression of the author that dystocia in these animals can be a direct or indirect result of trauma. Fractures to the carapace and plastron may have a direct physical effect on egg laying, or the presence of pain alone may lead to problems with oviposition. Often turtles with minor fractures or minimal displacement respond well to pain relief using non-steroidal antiinflammatory agents such as meloxicam (Metacam®, Boehringer) or tolfenamic acid (Tolfedine®, Ausrichter) and medical induction. However, turtles with significant disruption of the normal anatomy, including constriction of the cloacal aperture between the plastron and carapace can suffer from an obstructional dystocia. These animals usually require surgical treatment of the condition involving coeliotomy, salpingotomy and fixation of fractures.

Diagnosis

Diagnosis of dystocia involves establishing whether the turtle is in fact having difficulty passing eggs and is actually due to lay. It is difficult to distinguish dystocia from a normal gravid state. A good history and thorough physical examination will aid this diagnosis in captive reptiles. Consequently, it is more difficult to establish whether a free living animal is dystocic or not. Signs including obvious caudal abdominal masses, straining, cloacal prolapse, and increased respiratory rate will occur in dystocic chelonia. Injured turtles should be investigated thoroughly. Unfortunately little is known about the gestation period of free living common eastern long-necked turtles. It is known that females may also store sperm for up to 13 months leading to out of season oviposition, given the right climatic conditions (Michael Frith, pers comm).

Signs of listlessness and weakness are common in dystocic lizards but often turtles show no outward signs of discomfort. Radiography is a useful diagnostic aid in these reptiles. Gravidity is often an incidental finding when injured animals are radiographed. [3] Egg position, size and shell thickness can be gauged reasonably accurately from radiographs. Retained eggs often have a thickened shell or abnormal shape. Turtles that are still gravid after the nesting season of late spring and early summer should be induced if there are no obvious signs of obstruction.

Diagnosis of gravidity by palpation of the coelomic cavity via the prefemoral fossa is often difficult. The process can be facilitated by placing the caudal half of the turtle in lukewarm water and patiently waiting for her to relax. This may take several minutes. Some reports indicate that the texture of eggs will give some guide as to whether a turtle is due to lay (Michael Frith, pers comm). Eggs will harden towards the end of the gestation period.

Treatment

Unlike lizards, chelonia appear to tolerate much longer dystocic periods and rarely present as emergencies. Provision of a suitable environment is paramount as often failure to provide this is a major contributing factor to dystocia. Supportive treatment includes intracoelomic fluids, taking care not to inadvertently damage any eggs by injection, and assisted feeding. Physical

manipulation of eggs is usually difficult or impossible in chelonia due to the confining properties of the carapace and plastron.

Medical

Hormonal stimulation may be used in cases of non-obstructive dystocia. Oxytocin, stimulating oviductal contraction, is frequently used and recommended dose rates range from 1-20 IU/kg bodyweight. [4,5,6] It is reportedly successful in over 90% of cases, when administered within 48 hours of the onset of dystocia. [5] Arginine vasotocin, the reptilian equivalent has been used but is unavailable commercially.[1] Medical induction of oviposition appears to be more successful in chelonia compared with lizards and snakes. Additional treatments include priming with calcium and propanolol. There are little data on the benefits of this adjunct therapy, [4] however some reports mention that propranolol may be useful in decreasing oviductal sympathetic tone in gravid reptiles, allowing endogenous hormones to be more effective in stimulating oviposition. [7] There are reports that unlike mammals, difficulty in delivering eggs is not due to primary uterine inertia or hypocalcaemia.[4] Often warm water baths assist in induction. [3,4] Prostaglandins have also been used in reptiles with limited success.[7,8]

Surgical

Surgical removal of eggs is indicated in cases of obstructive dystocia in turtles. Plastrotomy provides easy access for the surgeon but requires a longer rehabilitation time and may require dry-docking in the case of freshwater turtles. The prefemoral approach provides less easy access but much faster healing and recovery times. Egg retention in the urinary bladder has been reported in a Florida cooter turtle, *Pseudemys floridana floridana* subsequent to trauma.[5] Egg retention in the urinary bladder should be suspected when there is radiographic evidence of caudal midline egg location.[5] Further support for the diagnosis is failure of conventional treatment for egg retention and/or a history of trauma.[5] Additional diagnostics including ultrasonography may be required if there is no response to hormonal induction.

Anaesthesia in freshwater turtles

The author's preferred anaesthetic induction agent for *Chelodina longicollis* is alfaxalone (Alfaxan CD-RTU®, Jurox), a quick acting and safe anaesthetic used at the dose rate of 8mg/kg

via the jugular vein. Lignocaine gel is applied to the glottis to relax the glottal opening. Endotracheal intubation is achieved using a modified intravenous catheter (12-14g). Anaesthesia is maintained using isoflurane and oxygen at 2-3% at a flow rate of 1L/minute, using an Ayres T-piece. Intermittent positive pressure ventilation is delivered at a rate of 2 respirations per minute. Anaesthetic monitoring is carried out using a doppler monitor. The author has used this drug in over 100 common eastern long-necked turtles for both induction of general anaesthesia and as a short acting anaesthetic for minor procedures such as placement of oesophagostomy tubes and shell repairs.

The eggs

Eggs from Chelodina spp. placed in a container of moistened vermiculite and water in equal quantities can be incubated at 28°C. It is the experience of the author that often reptile eggs delivered surgically or induced hormonally have a decreased chance of hatching. The timing of elective induction corresponding with a normal gestation cycle may increase the likelihood of viable eggs. Diapause has been recorded in the eggs of free-living Australian chelid turtles which could complicate the interpretation of a "normal" incubation period in these species.[9]

Conclusion

Dystocia in the class Reptilia is a common problem. Often in turtles it is difficult to distinguish between a normal gravid state and dystocia. Injured free-living turtles should be sexed and radiographed. Female turtles with shell fractures should be examined closely to assess whether the injuries may lead to, or are causing dystocia. A risk-benefit analysis of such animals should be done to determine the most favourable outcome for the turtle. The eggs of dystocic turtles that are rescued during the egg-laying season of late spring to early summer, and hormonally induced, stand a better chance of hatching than those that are treated out of season.

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An anatomical approach to prognosis in traumatic injuries of the common eastern long-necked turtle

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Not all creatures protect their young.
A turtle lays its eggs in the sand and departs.
- Jeffrey Masson[1]

Introduction

Shell injuries are the most common reason free-living Australian freshwater turtles are presented for veterinary treatment. There is a need for wildlife rescuers, rehabilitators and veterinarians to devise a simple method of triaging injured turtles in order to avoid any unnecessary suffering, and to a lesser extent, waste valuable resources, both human and financial. This paper tries to address this problem in a logical and simple way. Common eastern long-necked turtles (*Chelodina longicollis*) presented for treatment at South Penrith Veterinary Clinic were examined and had injuries charted. Records were kept of injury patterns, treatments and outcomes over a 16 month period.

Biology

The common eastern long-necked turtle, belongs to the sub-order Pleurodira (side-neck) and the family Chelidae containing three genera of long-necked piscivores (fish eaters), two of which occur in Australia, *Macrochelodina* spp. and *Chelodina* spp.. Semi-aquatic in its habits, *C. longicollis* occupies inland waterways, swamps, lagoons and creeks of the eastern mainland of Australia. Mating usually occurs in spring (September – October) and egg-laying mid November (Michael Frith, herpetologist, pers comm). Hatchlings generally emerge from their nests during March and April; however non-seasonal hatchings have been recorded as early as August and in late October. Although the time of egg-laying was unknown in these cases it does however provide evidence that *C. longicollis* eggs can "overwinter". Embryos of the broad-shelled turtle, *Macrochelodina expansa*, undergo diapause; however
C. longicollis do not (Michael Frith, herpetologist, pers comm).

Chelonian anatomy

Musculoskeletal system

The turtle shell consists of a carapace, two bridges and a plastron. The carapace is made up of 50 bones derived from the ribs, vertebrae and dermal elements. The plastron has evolved from the clavicles, interclavicles and gastralia (abdominal ribs).[2] Scutes, or keratin plates, cover the bony layer, but do not exactly overlap the underlying bones. Both the scutes and the underlying bone are capable of regeneration. The pelvic and pectoral girdles are contained within the rib cage. The appendicular skeleton of Australian freshwater turtles is typical of other vertebrates, except that the atlas and axis are fused. The remaining cervical vertebrae are large and elongated. A large muscle mass necessary for movement of the neck runs along the dorsal body of turtles closely attached to the coelomic surface of the carapace. It consists of the *M. retrahens capitus collique*, small muscles attached to the cervical vertebrae and a very large *M. longissimus dorsi*. Expansion of the

M. longissimus dorsi, results in forward movement of the head. Contraction of *the same muscle* in conjunction with either relaxation or contraction of *M. retrahens capitus collique* will result in a rapid sideways movement of the neck.[3]

C. longicollis is a "suck and gape" feeder, swimming after its prey, whereas the larger broad-shelled turtle is a "strike and gape" feeder. Common anatomical features of the "strike" mode feeders are a long neck and streamlining of the skull. In contrast, *C. longicollis* has a rounded head. "Strike" mode feeders also have a more flattened head, binocular vision, large and elongated *M. longissimus dorsi*, and a large body relative to the head and neck. Neural bone plates (dorsal midline) are also larger in order to buttress these muscles during striking. The larger body to head and neck ratio prevents retropulsion when striking. [3]

Skin

The skin covers the limbs, neck, head, cloacal and tail areas. Typical of most freshwater turtles Australian species usually have soft skin compared with terrestrial chelonia.

The coelomic cavity

The coelomic cavity is lined by the pleuroperitoneum. There is no diaphragm; however a membrane, the *septum horizontale*, separates the lungs dorsally from the viscera ventrally.[4]

Respiratory system

In pleurodiran (side-necked) turtles the trachea is single and not bifurcate. The lungs are large saccular organs taking up the bulk of the dorsal subcarapacial space. There is no diaphragm. The pig-nosed turtle is the only free-living cryptodiran (straight-necked) species in Australia. In turtles belonging to the order Cryptodira the trachea is usually bifurcated quite rostrally. Respiration in the typical mammalian sense is not possible in the turtle due to the confining aspects of the shell and the absence of a diaphragm. A complex system of muscles helps to expand and deflate the lungs in conjunction with limb movements. Chelonians do not rely upon negative thoracic pressure (due to the absence of the diaphragm) so penetrating wounds to the coelomic cavity will not necessarily cause deflation of the lungs or dyspnoea. Some Australian species of turtles, such as the Fitzroy River turtle, *Rheodytes leokops,* absorb oxygen through the cloaca, allowing for prolonged periods of submersion. The lungs of this species are quite small compared with other Australian freshwater turtles (David Vella, pers comm). *C. longicollis* can also spend extended periods underwater.

"In one serendipitous observation, a turtle (C. longicollis) found submerged during winter had not moved for at least four weeks (as indicated by a small water-logged stick placed on the carapace)"[5]

Gastrointestinal system

Freshwater turtles have a mouth with a large gape, comprising a horny beak (rhamphotheca) and the lower jaw or mandible. The hyoid apparatus and oesophagus are large compared with most other vertebrates. These features facilitate the swallowing of large amounts of water along with the food or prey item. Dietary preferences vary in Australian chelonia. For example,
C. longicollis is carnivorous and piscivorous in its habits, whereas the Macquarie short-necked turtle, *Emydura macquarii macquarii,* is an omnivore. The stomach is simple and muscular, lying ventrally and to the left side of the coelomic cavity. The small intestine is relatively short compared with most mammals. The liver is large and bilobed. The large intestine is short and terminates in the cloacal coprodeum. The gall bladder is present in freshwater turtles. The pancreas is similar in structure and function to other vertebrates.

Urogenital system

The kidneys are situated quite caudally and dorsally in the retrocoelomic cavity. The urinary bladder is well developed in chelonia. It is bilobed and flattened in structure. When distended it is balloon like and the bladder wall is quite transparent. Male turtles have a single intromittent organ, unlike the Squamata (snakes and lizards) which have two hemipenes. Compared with the hemipene of snakes and lizards, it does not invert. The penis normally rests within the retroperitoneal cavity and protrudes from the cranial cloaca in a caudoventral direction during coitus. The urethra is absent. The oviduct of female turtles is paired. Lying dorsal to the bladder, it is quite long and convoluted, looking like a large cestode when in the non-gravid state. Unlike other reptiles the urogenital ducts of chelonia enter the neck of the bladder instead of the urodeum. [4] Eggs and follicles in the gravid female occupy a large proportion of the coelomic space.

Gender determination

Gender may be determined in most Australian side-necked turtles by physical examination. Male *C. longicollis* are generally smaller than females. The tail may be longer in males compared with females. The plastron in males is concave caudally. Females have a uniformly flat plastron. Aged female specimens develop a convex plastron. The caudal notch of the plastron is more v-shaped in the male. The penis may occasionally protrude from the cranial cloaca during handling.

Methods

Charting techniques and record keeping

Thirty two injured common eastern long-necked turtles were presented for treatment at South Penrith Veterinary Clinic from February 2005 to June 2006. All turtles underwent a full physical examination. Radiographs were taken of all female turtles and turtles with limb and neck injuries, paresis or paralysis.

A template of the dorsal and ventral view of *C. longicollis* was used to record the pattern of fractures (Figure 1). The date and location of rescue, identification details, treatments and outcome were noted. The weight, straight carapace length (SCL) and gender were also recorded.

Repair techniques

A variety of techniques were used for the repair of shell injuries, including the use of dental composite, orthopaedic wiring, external coaptation using K wires, pins and screws. Wounds were cleaned and debrided with saline or chlorhexidine 0.05% and fracture surfaces ground back to healthy tissue using a Dremel® tool. Alfaxalone (Alfaxan®-CD, Jurox, Australia) was the anaesthetic of choice, used at a dose rate of 8mg/kg body weight, achieving a good surgical plane of anaesthesia for most simple repairs. For longer procedures turtles were intubated and maintained on isoflurane and oxygen. An oesophagotomy tube was placed in one turtle. Routine antibiosis was used in all cases. Pain relief was provided using butorphanol tartrate (0.1mg/kg SC) and tolfenamic acid at a dose rate of 4mg/kg SC q24-72h. Several turtles required long term care in a moist environment that excluded submersion. This varied from placing turtles in tubs of shallow water to using a custom made device ("turtle steamer"©). The latter consists of one plastic tub placed inside another, with heated water in the bottom tub and holes drilled in the upper tub in order to provide a more humid but not "wet" environment.

Criteria for euthanasia

The criteria for euthanasing turtles included the presence of compound fractures, extensive soft tissue damage or exposure, paresis or paralysis of the head or limbs and infection.

Summary of results

Injury sites and outcomes of free-living common eastern long-necked turtles treated at South Penrith Veterinary Clinic from 21/2/2005 to 5/6/2006 are presented in Table 1. A total of 32 turtles were triaged; 23 were male and 9 female. Eleven were released, 9 died or were euthanased, five placed in medium or long term care and 7 lost to follow up. Shell injuries were recorded in 30 animals, the remaining two having suffered head injuries. Injuries affecting the carapace were the most common type, divided into carapace alone, 6 turtles, carapace and plastron 8, carapace and bridge 9 and carapace, bridge and plastron 2 turtles. One turtle with a plastron and bridge fracture had no carapacial injury. Three turtles had only plastron fractures and one turtle had a single bridge fracture. Of the 9 turtles that died or were euthanased, 8 were males and only one did not have a bridge fracture. Five of the euthanased turtles had soft tissue damage or exposure of soft tissue. Seven of the animals with bridge fractures also had carapace fractures. One turtle had a single carapace fracture. Only one turtle was presented during the winter.

Females were presented for treatment from October to March only. Injured males were presented over a wider period, January to April, one during June, and the remainder from September to November.

Discussion

Females versus males

Significantly more males than females were presented for treatment. Females were more commonly seen in late spring, summer and early autumn whereas there was no obvious seasonal trend with respect to males. Generally males roam more widely than females. Female turtles usually stay close to waterways, only travelling greater distances during and after the nesting period.

Nature of injuries

Mortality rates appear to be higher in turtles with bridge injuries. Of the nine that were euthanased or died only one had not suffered a bridge injury. The bridges are integral parts of the shell separating the carapace and plastron and providing structural support in the protection of the viscera. There appears to be a higher chance of serious internal injury if one or both bridges are fractured. The presence of soft tissue damage or the exposure of internal organs is not a good prognostic sign. The largest muscle group in the body, the *M. retrahens capitus collique* and *M. longissimus dorsi* run along the dorsal midline of the turtle from the head to the caudal body. Within the coelomic cavity the muscle group is firmly attached to the carapace. Transverse fractures crossing the midline can sever this tissue leading to a partial or complete inability to retract or flex the neck. Deep carapacial injuries may cause lung trauma but as previously mentioned do not appear to lead to pronounced dyspnoea.

As well as obvious fractures of the carapace, bridges and plastron, other injuries may occur. Paresis or paralysis of the neck may result in an inability to retract the head under the carapace. One turtle (56T15) was suffering from ankylosing spondylosis of the cervical vertebrae and could not retract its head. The neck condition would have been of a chronic nature, increasing the risk of head injury. Limbs that do not retract normally may be fractured or paretic. Asymmetry with respect to limb function and posture are usually signs of serious injury. Due to their poor

prognosis, turtles with bridge injuries should be radiographed and fully assessed prior to starting a treatment plan. All female turtles should be radiographed. Injured female turtles that are gravid should be assumed to be dystocic, especially if presented out of the egg-laying season. Gravid turtles should be medically induced. If oviposition does not occur within 12-24 hours obstructive dystocia is suspected. Coeliotomy and salpingotomy may be required in these cases.

Occasionally turtles will respond well to treatment but lose condition several months after the initial injury. Abscesses involving internal organs or longstanding infections are often found in these cases.

Conclusion

A full physical examination and a good understanding of turtle anatomy and biology are paramount to achieving a satisfactory outcome for injured turtles. Clinicians should become competent in sexing turtles in order to facilitate the triage process. Turtles with compound or multiple injuries affecting the bridge have a poor prognosis.

Important points

- Don't let the sun set on an injured dystocic turtle.
- Ensure carers keep turtles in their preferred optimal temperature zone (POTZ), which is 25-28^{o}C for the common eastern long-necked turtle.
- Ensure carers can cater with more complex turtle husbandry issues such as "dry-docking" and egg incubation, otherwise all your good work will be to no avail.
- Ensure carers give progress reports.
- Radiography is a most useful diagnostic aid.
- Endoscopy is useful in assessing patency of the pleuroperitoneum and damage to internal organs.
- Resources such as these injured turtles should be used in health surveillance studies (e.g. ranavirus monitoring) that will benefit the greater environment and biosecurity of Australia.

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Update 2010

A total of 185 turtles were triaged between 21/02/2005 and 15/02/2010. Males outnumbered females (105/80). One hundred and thirty four had incurred a head, carapace, bridge or plastron injury (Table 2). Of the remainder, nineteen turtles had no obvious injury; twelve showed signs of abscess, soft tissue or chronic injury; ten were attacked by dogs; five had swallowed fish hooks; and five were “not doing well”. Turtles that were euthanased or died had a higher percentage of carapace and bridge injuries compared with those that were released. Released animals were also more likely to have just a single injury.

Table 1. Injury sites and outcomes of free-living common eastern long-necked turtles treated at South Penrith Veterinary Clinic (21/2/2005 – 5/6/2006)

Date/ID	Sex	Carapace	Plastron	Bridge	Soft tissue	Tissue exposure	Other injuries, gravid state	Outcome
21/2/05 WIRES N1	M	X	X					Released
23//2/05 WIRES3	M	X						Died
25/2/05 MOP1	M		X					Released
26/2/05 MOP2	M	X	X		X	X		Euthanasia
26/2/05 WIRES4	M	X		X(1)				Released
2/3/05 Cranebrook	F	X	X	X(1) abrasion			Dog attack	Released
4/3/05 MOP6	M	X	X	X(1)				Euthanasia
21/3/05 WIRESW8	M	X		X(2)				Released
14/4/05	M	X		X(1)				?
11/4/05 WIRES N2	M	X						?
29/9/05 MOPBC	M	X		X(1)			X	Released
10/10/05 56T2	F	X	X				Gravid	Longterm care
21/10/05 56T3	M	X						Released
24/10/05 WIRES1290	F						X*	Longterm care
31/10/05 56T4	F	X	X					?
31/10/05 56T5	F			X(1)			Gravid #radius/ulna	Euthanasia
4/11/05	F	X	X					Released
28/11/05 56T8	M	X		X(1)	X			Died
10/11/05 56T6	M	X		X(2)	X	X		Died
11/11/05 56T7	M		X					?

11/1/06 Pebbles	F		X	X				Released
17/1/06 56T8	M	X		X(1)				Euthanasia
18/1/06 56T9	F	X						?
20/1/06 56T10	M	X		X(2)	X Neck		X**	Died
23/1/06 56T11	M	X	X				***	?
1/3/06 56T13	M	X		X(1)	Haem		# humerus	?
2/3/06 56T12	F	X	X					Released
8/3/06 56T14	M	X						Released
13/3/06 56T15	M	X		X(1)		X		Euthanasia
3/4/06 56T16	M				X		X****	Longterm care
7/4/06 Mulgoa Rd	M	X					R head tilt	Care
5/6/06 56T17	M		X					Care

(Injuries are motor vehicle trauma unless indicated)
Bridge injuries are described as unilateral (1) or bilateral (2).

* WIRES 1290 #mandible, #maxilla, gravid
** 56T10 Neurological signs - unable to retract neck, spasticity of 4 limbs.
*** 56T11 LHL absent - old injury
**** 56T15 Head injuries, eye trauma. Torticollis. Ankylosing spondylitis of cervical vertebrae.

Table 2 :Turtles with shell fractures or head injuries 21/02/2005-15/02/2010

D/E/R	Single	Head	Carapace	Plastron	Bridge	Total
Died N(%)	3(14.3)	1(4.8)	17(81)	8(38.1)	15(71.4)	21
Euthanased N(%)	7(21.9)	6(18.8)	28(87.5)	8(25)	19(59.4)	32
Released N(%)	37(46.3)	4(5)	60(75)	32(40)	24(30)	80
Lost to follow up		1(0.07)				1
Total	**47(35)**	**12(9)**	**105(78.4)**	**48(35.8)**	**58(43.3)**	**134**