
THE CHELONIAN SHELL IN HEALTH AND DISEASE: IT'S WHAT'S OUTSIDE THAT COUNTS

Bruce S Levine, DVM, Dipl ABVP Avian, Exotic Companion Mammal, Companion Animal

Huntington Beach, CA 92648 USA blevine@earthlink.net

ABSTRACT

The shell is a unique structure characterizing the order Chelonia. Composed of living tissue, care must be taken to minimize tissue damage and preserve blood supply when treating disorders of this structure.

Introduction

The shell is a unique structure characterizing the order Chelonia. First appearing in the Permian era¹⁷ it has evolved in most species as a calcification involving the ribs, dermis, and intercostal muscles. Histologically, the shell consists of inner cancellous bone surrounded dorsally and ventrally by a layer of compact bone.⁸ The dorsal shell is called the carapace and the ventral the plastron. Bony sutures allow for shell growth and mobility in hinged species. The shell is covered externally by a series of keratin scutes. The margins of the external scutes enlarge by keratin production by the underlying epidermal cells. The underlying bony scutes grow by cartilaginous osteogenesis on the margins. The sutures of the keratin scutes and the underlying osseous scutes do not align which helps in maintaining the shell's structural integrity. Vertebral and intercostal vessels and nerves supply the carapace while intercostals and epigastrics supply the plastron.

The keratinaceous carapace is divided into paired marginal and costal scutes, and as well as unpaired vertebral and nuchal scutes. The plastron consists of the paired gular, humeral, pectoral, abdominal, femoral, and anal scutes. In some species intragular, paired axillary and inguinal scutes may also be present on the plastron. The abdominal scutes meet the marginal scutes of the carapace to form the lateral bridges. (Fig. 1)

Unique among tetrapod vertebrates, the pectoral and pelvic girdles are found ventral to the ribs and respiration is achieved by the actions of modified abdominal muscles pushing and pulling on the lungs while expanding and contracting the coelomic cavity. In some species buccal respiration is also achieved by the pharynx pulsing air into the trachea.

Most turtle shells are constructed along the models of the keystone arch and the cupola.¹⁴ The carapace may be highly elevated or shallow varying by environmental and ecologic constraints. Strengthened by its marked convexity and its thickened periphery, the entire structure is protected from vertical collapse by the strong bridges connecting the plastron to the peripheral bones.



Disorders

Congenital Shell Defects

Premature scute closure is commonly seen in many species of chelonian. Although the resultant deformity of the carapace may affect structural function, often these abnormalities are not clinically significant.⁴

The gular process is especially prominent in male Testudinae like the California desert tortoise. Abnormalities can range from dorsal curling of the distal end causing abrasion of the ventral cervical area to complete forward obstruction of the head. It is unknown whether the etiology is congenital or poor husbandry. These can be trimmed for surgically correction.

Nutritional

Nutritional hyperparathyroidism is an all too common problem in young growing chelonians. All vegetable diets poor in calcium and/or vitamin D₃ result in fibrous osteodystrophy of the shell and subsequent growth deformities.³ Often the shell is flattened dorsoventrally and deformities and fractures of the long bone can occur as well. Although more common in squamates, kyphosis with or without neurologic deficits can also result from improper nutrition. Dietary correction is the key treatment but often does not reverse the structural deformities.

Metabolic

Renal failure is seen in older chelonians, particularly tortoises. Etiology is often idiopathic though bacterial, viral, and nutritional causes have been demonstrated in some cases. The resultant renal hyperparathyroidism can lead to shell lesions such as loosening of the scutes, oozing cracks or generalized softening of the shell.¹⁰ Neurologic signs such as tremors and convulsions can be seen as well. Diagnosis can be based on finding hypocalcemia and hyperphosphatemia, enlarged kidneys on cloacal palpation in selected species, and renal biopsy. Treatment with potent vitamin D analogues such as diethyltarchestrol in early cases may achieve palliation of some clinical signs but the long term prognosis is poor. This disorder has also been seen as a congenital problem in hatchling California desert tortoises.

Infectious

Infections of the shell are more common in aquatic chelonians than tortoises.^{7,15} These are usually related to inadequate husbandry such as poor water quality, rough substrates, inappropriate temperatures, lack of ultraviolet light, wrong diet and insufficient basking areas. Signs range from superficial ulceration of the keratinatous scutes to deep ulcerations and deep shell abscesses. Septicemia is often present in these patients as well. Treatment consists of debridement, systemic and local bacterial or fungal antimicrobials, and correction of the general husbandry as well.

Idiopathic

Pyramiding is often seen in tortoises that have been raised under unnatural conditions of humidity and diet.¹² It results in the vertebral and costal scutes having the central areolae sharply elevated. In some species it has been demonstrated to occur in the wild as well as in captivity. The condition often first manifests in the post-hatchling months, or occasionally later in life. Current belief is that the condition derives from excessively dry conditions, and when hatchlings are raised on a substrate such as wet sphagnum, the shells develop normally.

Dyskeratosis is associated with abnormal conformation and loss of keratin integrity of the shell of Californian desert tortoises (*Gopherus agassizii*).⁹ Gross lesions include white discoloration, flaking and peeling of the scutes, with irregular pitting and chipping. Deep defects may expose dermal bone. Lesions occur most consistently in the plastron, and extend from the seams into the scutes. Microscopically, dyskeratosis is characterized by crevices, clefts and thinning of the epidermal horny layer. Soil, plant debris, necrotic cellular debris, and occasionally, bacteria and fungi often accumulate in these defects. The lesions occur in both sexes and in all sizes of adults. The cause of mortality and the pathogenesis of the lesion has not been determined. A genetic predisposition is unlikely because multiple populations of desert tortoises are affected. Toxic or nutritional causes have been suggested.

Traumatic

Car accidents, dog bites, and miscellaneous injuries can result in shell cracks, punctures, lacerations, and dislodgements. Lavage and thorough debridement of necrotic shell should be performed to prevent infections and wet dry bandages can be used in small puncture wounds or to protect the tissues until a more permanent repair can be performed. Preoperative physical examination, imaging, and/or laboratory examination must be undertaken to determine the extent of internal soft tissue damage.

Loss of keratin scutes is commonly seen. Over time (years) the underlying bony plate necrotizes and a new scute will regrow in the area. Treatment is minimal intervention.¹

There are many methods to repair shell damage.^{2,5,6,13} In young growing chelonians with soft shells, small holes can be drilled and the bony plates sutured together until healing (usually 1-2 mo). In adults, the shell can be repaired with cerclage wires, surgical bone plates, metal bridges and/or epoxy fiberglass patches.

Fractures or avulsions involving the carapacial-cutaneous border can be repaired by drilling small holes in the edges of the carapace with suturing of the skin to the shell in a horizontal mattress pattern. A similar method utilizing simple interrupted sutures can be used to repair luxations of hinged plates.

Lost shell fragments can be addressed by long term bandaging with or without metal bridges or by covering with light sensitive dental acrylic prosthesis. Infected areas should be thoroughly debrided and implanted with methylmethacrylate impregnated antibiotic beads before closure.



Surgery

Coeliotomy is the most common reason for shell surgery,⁴ though carapacial-cutaneous or plastron-cutaneous flaps have been utilized for exposure of the deeper areas of the pectoral girdle.¹⁰ A circular dermal rotary tool, a cast cutter, or stryker saw can be used to create an inwardly slanting flap in the plastron or carapace. The shell membrane is then cut along the flap on three sides, leaving the soft tissues attached on the third side to allow blood supply. The anterior edge is best to leave intact on the posterior coelomic approach and the medial or posterior edge is left intact over the pectoral and pelvic girdles. Closure is achieved by overlying patches of epoxy and fiberglass cloth or tape. In younger patients, metal bridges or patches of plumber's putty can be utilized for closure without interfering with further shell growth.¹⁶

Conclusion

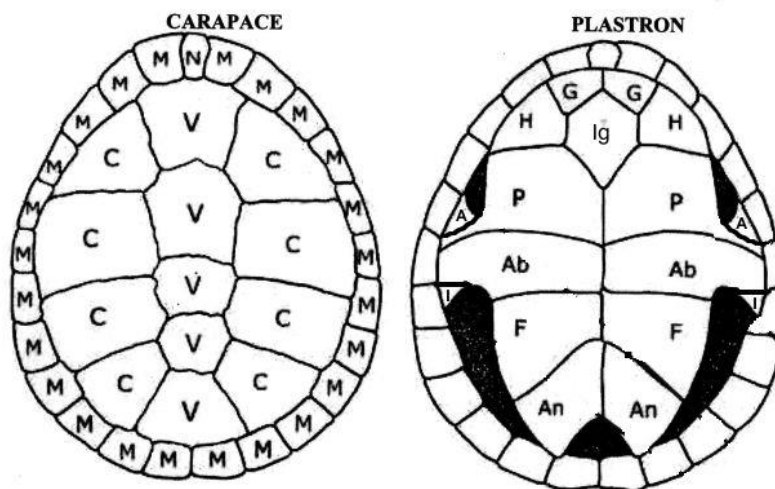
The tortoise shell is a unique structure in the animal kingdom, but common vertebrate dermatologic principles can be used in treating its abnormalities.

LITERATURE CITED

1. Barten SL. 2006. Shell damage *In* Mader D, Reptile Medicine and Surgery, 2nd Ed., 896.
2. Bennett RA. 1989. Reptilian surgery part II: management of surgical diseases. *Comp Cont Educ Pract Vet* 111-112.
3. Cooper JE. 2006. Dermatology *In* Mader D, Reptile Medicine and Surgery, 2nd Ed., 204-205.
4. Frye FL. 1991. Biomedical and Surgical Aspects of Captive Reptile Husbandry, 2nd Ed., Melbourne FL, Krieger Publishing.
5. Harwell G. 1989. Repair of injuries to the chelonian plastron and carapace *In* Kirk RW, Current Vet Therapy 10: Small Anim Pract.
6. Heard DJ. 1999. Shell repair in turtles and tortoises: an heretical approach, *Proc North Am Vet Conf*.
7. Hernandez-Divers SJ et al. 2009. Investigation of shell disease in map turtles (*Graptemys* spp.). *J Wildl Dis*, 45(3):637-652.
8. Hirasawa T, Nagashima H, Kuratani S. 2013. The endoskeletal origin of the turtle carapace. *Nature Commun*, 4: 2107 doi:10.1038/ncomms3107.
9. Homer BL et al. 1999. Characterization of soluble shell scute proteins of California desert tortoises (*Gopherus agassizii*), *Proc ARAV*, 108.
10. Lawton MPC. 1996. Partial plastronectomy in a *Testudo graeca* *Proc ARAV*, 40-42.
11. Mader D. 2006. Metabolic bone disease *In* Mader D, Reptile Medicine and Surgery, 2nd Ed., 847-848.
12. Pritchard PCH. 2012. Evolution and structure of the turtle shell *In* Wyneken J, Biology of Turtles: From Structures to Strategies of Life, 70.
13. Richards J. 2001. Metal bridges – a new technique of turtle shell repair. *J Herpetol Med Surg*, 11:31.
14. Rivera G, Stayton CT. 2013. Effects of asymmetry on the strength of the chelonian shell: a comparison of three species. *J Morphol*, 274(8):901-908.

15. Scheelings TF, Tesdorpf R, Hooper C, Stalder K. 2012. *Chromobacterium violaceum* isolation from a Macquarie turtle (*Emydura macquarii*). J Herpetol Med Surg, 22:22-24.
16. Tamukai K. Flap closure method using epoxy putty in plastron osteotomy in chelonians. Exotic DVM, 12,1:5-11.
17. Tyler R. Lyson, et al. 2013. Evolutionary origin of the turtle shell. Curr Biol, 30.

Figure 1. Scutes of the carapace and plastron.



N= Nuchal M= Marginal C= Costal V= Vertebral G= Gular H= Humeral Ig= Intergular P= Pectoral
Ab= Abdominal F= Femoral An= Anal A= Axillary I= Inguinal

