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DECOMPRESSION SYNDROME IN FOSSIL MARINE TURTLES

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ABSTRACT

Avascular necrosis of bone occurs in Tertiary and Quaternary marine turtles, as it does in Cretaceous mosasaurs. Assessment for potential underlying pathophysiology suggests decompression syndrome as the etiology of this bone pathology. The pathophysiology of decompression syndrome, and predisposing and protective factors are reviewed.

INTRODUCTION

Avascular necrosis of the humeral head results in loss of viability (death) of bone. The devitalized bone often becomes necrotic and liquefied, subsequent to the loss of vascular supply, so that the stresses of normal use result in fracture of the surviving subchondral bone (Resnick et al., 1981; Feldman et al., 1981). The result of such fractures is discrete collapse of the articular surface. This complication of decompression syndrome, which has been observed in man, was recently discovered in Cretaceous mosasaurs (Rothschild and Martin, 1987). The present report describes avascular necrosis in Cretaceous marine turtles and Cenozoic marine and freshwater Chelonians.

METHODS

Cretaceous marine turtle specimens in the collection of the University of Kansas Museum of Natural History were examined for avascular necrosis of the humeral head, as evidenced by focal subsidence (e.g. collapse) of the articular surface. Specimens were also examined in the Institut Royal des Sciences Naturelle des Belgique (Brussels, Belgium), the Red Mountain Museum (Birmingham, Alabama) and the Auburn University Museum (Auburn, Alabama). Specimens found to have focal subsidence of the humeral articular surface were sectioned for confirmation of avascular necrosis. A simple coronal cross-section was adequate for examination. The sections were subjected to radiologic and electron probe analysis and to bright field, polarizing, fluorescent, and scanning electron microscopic examination. Polarizing and fluorescent microscopy of the sections revealed trabecular patterns, the disruption of which characterize avascular necrosis. Scanning electron microscopy revealed ultrastructure alterations, and permitted precise placement of the electron probe utilized for bismuth analysis. Standard ionization chamber technique was utilized for assessment of radiation levels.

RESULTS

Examination of Cretaceous marine turtles contemporary with the mosasaurs revealed a specimen of *Desmatocheles* (Desmatochelyidae) manifesting avascular necrosis (Table 1) of the humeral heads. This was seen as focal subsidence within the articular surface of the intact specimen (Fig. 1A) and by collapse of the necrotic zone as seen in cross section (Fig. 1B). Evidence of avascular necrosis was also found in both Eocene (*Eosphargis*: Dermochelyidae and Cheloniidae, species indet.) and Oligocene (*Oligochelone* and Cheloniidae, species indet.) turtles. Avascular necrosis was also present in *Porthochelys* (Toxochelyidae), and *Protostega*

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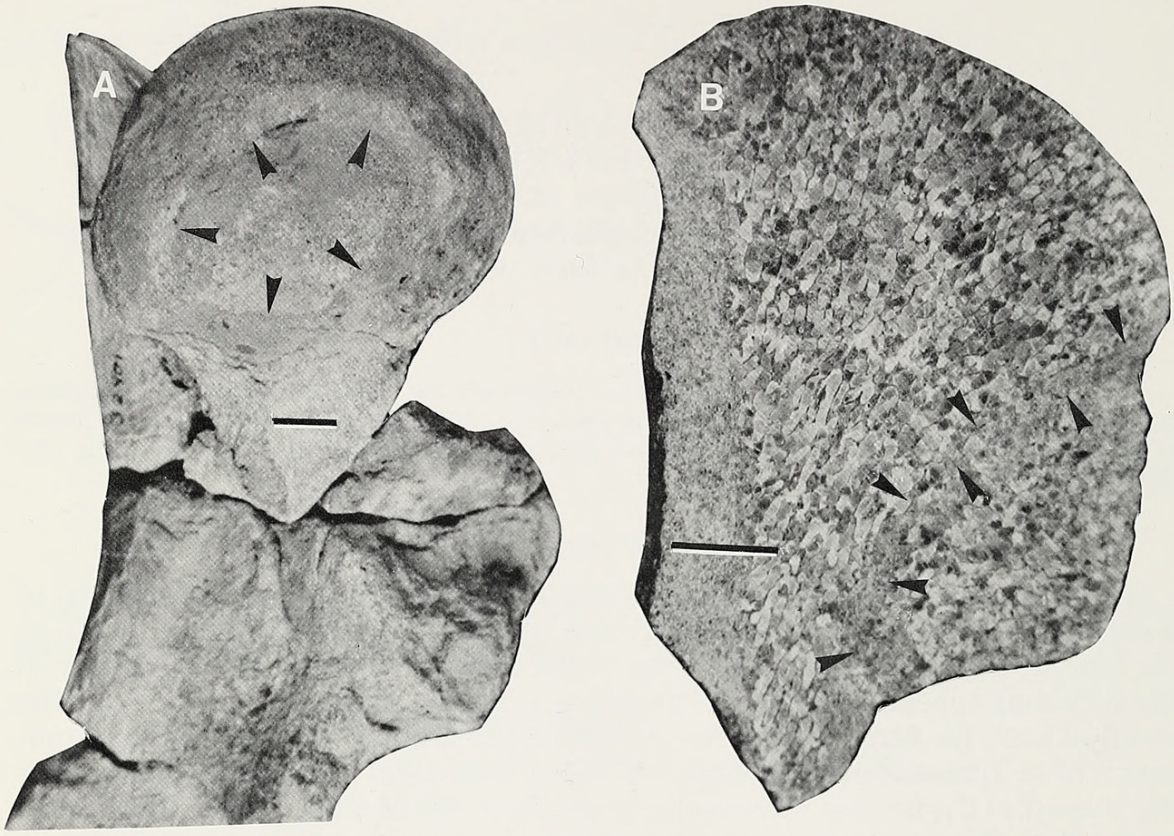

Fig. 1.—*Desmatochelys* humerus showing A, focal subsidence on articular surface of head (arrows) and B, collapsed necrotic zone in cross-section of head.

dixie (Protostegidae) the Cretaceous. Avascular necrosis was not observed in *Psephophorus, Lytoloma,* and *Trionyx,* although only limited numbers of humeri were available for examination. Microscopic examination of sectioned material revealed only focal linear loss of definition of trabeculae and collapse of subchondral bone. Trabecular architecture was intact and normal extrinsic to the zone of involved bone. Electron probe analysis revealed no evidence of significant bismuth exposure. Radiation levels in affected and unaffected specimens were similar.

Avascular Necrosis in Turtles

Avascular necrosis is an easily recognized pathology, characterized by the appearance of articular surface collapse. Its presence is confirmed in 11% of fossil Cheloniidae examined and in four other marine turtle families (Table 1). The differential diagnosis of avascular necrosis in turtles is as previously reported for mosasaurs: radiation poisoning, bismuth poisoning, and decompression sickness (Rothschild and Martin, 1987). Lack of variation in radiation levels and absence of bismuth as determined by electron probe analysis suggest decompression sickness as the etiologic factor. As *Protostega* shared a common habitat with the mosasaurs, (evidenced by mosasaur bite lesions in a *Protostega* carapace (RMM 2255) in the Red Mountain Museum collection), it is possible that *Protostega* was subjected to the same pathophysiologic mechanism in development of avascular necrosis as the mosasaurs. Sudden changes in depth (e.g. rapid surfacing), possibly

Table 1.—*Incidence of avascular necrosis of turtle humeral heads.*

Taxon	Epoch	Specimens examined	Frequency %
Cheloniidae			
Caretta caretta	Holocene	8	0
Oligochelone rupeliensis	Oligocene	9	11
species indet.	Oligocene	3	33
species indet.	Eocene	15	7
Toxochelidae			
Porthochelys laticeps	Cretaceous	1	100
Ctenochelys cf. *tenuitesta*	Cretaceous	1	0
species indet.	Cretaceous	3	0
Lytoloma gosseleti	Eocene	2	0
Protostegidae			
Protostega dixie	Cretaceous	2	50
Desmatochelyidae			
Desmatochelys sp.	Cretaceous	4	50
Dermochelyidae			
Psephophorus rupeliensis	Oligocene	2	0
Eosphargis gigas	Eocene	4	50
Testudinidae			
species indet.	Pleistocene	9	0
Trionychidae			
Trionyx levalensis	Eocene	3	0

related to escape from mosasaurs or other predators, may have played a major role in the development of the lesions.

Decompression Syndrome in Non-Turtle Vertebrates

Decompression syndrome is commonly thought of as a phenomenon dependent upon breathing an exogenous air supply, but this requirement is not absolute. Decompression illness has also been noted in man subsequent to breath-holding dives (Pauley, 1965; Kooyman et al., 1973; Strauss, 1970). Repeated two minute dives of 20 to 40 m have resulted in the decompression syndrome (Kooyman et al., 1973). Pauley (1965) reported neurologic manifestations of decompression syndrome in man after sixty 15 to 20 m dives performed over a five hour period. The decompression syndrome etiology was confirmed by the "dramatic response" of symptomatology to recompression. Pauley (1965) also reported three similar cases occurring after 20 "bottom drops" during Norwegian escape-tank training. Polynesian (Tuamoto) pearl divers manifest an intriguing dichotomy of susceptibility to decompression illness. Those who performed 40 to 50 two minute dives to greater than 30 m, with three to four minutes between dives, or who participated in vigorous 4 to 5 hr diving sessions to 50 m developed decompression illness (Pauley, 1965; Strauss, 1970). Those who maintained at least ten minute intervals between dives (a different "diving habit" pursued at an adjacent island) did not develop decompression syndrome. The Ama (Japanese diving women), who dive to more shallow depths or maintain a longer surface interval, are apparently unaffected by decompression disease (Anderson, 1966; Strauss, 1970).

Decompression syndrome has also been documented in other vertebrates. Berk-

Table 2.—*Physiological mechanisms promoting vertebrate protection from decompression syndrome (derived from Anderson, 1966; Denison et al., 1971; Strauss, 1970).*

Decreased lung surface area for gas exchange
—Small lung volume
—Thoracic wall collapse
—Exhalation prior to descent
—Cartilage/muscle airway support (minimizes air trapping)
Augmented, targeted circulation
—Intracardiac blood shunting
—Increased intravascular volume
—Increased hematocrit
—Decreased red blood cell size
—Elasticity of blood vessels and sinuses
—Anuria-water conservation
—Vasoconstriction (e.g. of muscle beds)
Protection from hypothermia
—Decreased ratio of surface area to mass
Metabolic
—Decreased activity of
—histamine
—serotonin
—bradykinin
—smooth muscle acting factor
—Altered complement activation

son (1967) reported the death of a seal from decompression syndrome after forced surfacing from 300 m. Gas bubbles have been noted in blood samples obtained from diving birds at various depths. After four dives to 4 atm of pressure, nitrogen tension is increased to two to four times the original; after four dives to 7.8 atm nitrogen pressure increased to five times the basal value. After 2.5 minutes of submersion nitrogen increased from 82.4 to 89.5% of total gases (Kooyman et al., 1973). Most vertebrates avoid decompression syndrome only because of the briefness of their dives (Kooyman et al., 1973), but decompression syndrome can and does occur with prolonged submergence in a variety of vertebrate taxa.

The pathophysiology of decompression syndrome is more complex than simple vascular occlusion by nitrogen bubble emboli. Major causative factors may be hemoconcentration and red blood cell agglutination, resulting in decreased tissue perfusion (Strauss, 1970). Risk factors (derived from Strauss, 1970) for development of decompression syndrome include dehydration, fatigue, increased hemoconcentration, stress, impaired circulation, hypothermia, systemic disease, injury, and obesity.

Physiological protective mechanisms are listed in Table 2. However, the impact of the protective mechanisms is not straightforward. For example, the polycythemic (increased packed red blood cell content of blood) response may in some instances actually reduce circulation due to its effect on blood viscosity (Lutz and Bentley, 1985).

Decompression Syndrome in Turtles

Berkson (1967) first identified decompression syndrome in turtles. He reported that blood drawn from turtles under pressure forms bubbles in the syringe and

further noted the gas emboli death of a turtle after rapid decompression from 18.7 atm. Berkson also noted that turtle blood was supersaturated with respect to nitrogen at a given atmospheric pressure and that stepwise decompression also produced caisson disease (decompression sickness). The susceptibility of turtles to decompression syndrome is further substantiated by the observation of avascular necrosis, a phenomenon previously noted in mosasaurs and attributed to decompression syndrome (Rothschild and Martin, 1987).

While diving habits of some turtles are known, the impact on turtles of these risk and protective factors during diving has been the subject of limited study. The leatherback turtle (*Dermochelys coriacea*) is known to dive almost continuously with short surface intervals and can dive to 475 m. *Lepidochelys olivacea,* the olive ridley sea turtle, can dive to 300 m (Eckert et al., 1986). The loggerhead turtle (*Caretta caretta*) utilizes its lungs as a major oxygen storage system and spends only 1% of its time on the surface (Lutz and Bentley, 1985). Some turtles, such as *Pseudemys scripta* (Glass and Wood, 1983), have significant cutaneous respiration which could conceivably protect them from decompression sickness; 65% of the total CO_2 elimination in *Trionyx mucita* is non-pulmonary.

There is evidence that some of the physiological protective mechanisms may be non-operative in turtles. Lutz and Bentley (1985) noted that turtles inhale prior to dives, enhancing their risk. Lung collapse does occur in turtles at 80–160 m (Berkson, 1967), which leaves a substantial range of depth in which gas exchange continues.

Avascular necrosis has been identified in *Desmatocheles, Prostostega, Porthochelys, Oligochelone, Eosphargis,* and several indeterminate Cheloniidae. Factors which might affect susceptibility to this phenomenon, apparently related to decompression syndrome, include depth of dives, time at depth, surface time, lung type, cutaneous respiration, complement responses to micro-bubble formation, and perhaps even nitrogen excretion in the form of ammonium carbonate (Strauss and Sampson, 1986).

A general survey of contemporary turtle humeri would allow identification of susceptible species. Correlation of occurrence of avascular necrosis with diving habits, risk factors, and protective mechanisms in extant turtles may allow assessment of the relationship between risk factors and protective mechanisms.

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