

DECOMPRESSION SYNDROME IN PLESIOSAURS (SAUROPTERYGIA: REPTILIA)

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ABSTRACT—Recognition of avascular necrosis through propodial head subsidence in fossils indicates that plesiosaurs were susceptible to decompression syndrome and implies deep, prolonged or repetitive diving behavior for these animals. Contrary to the situation in mosasaurs, plesiosaur vertebrae were not targets of avascular necrosis. This suggests that the anastomosing internal vascularization of plesiosaur vertebral centra was functionally adaptive. No phyletic trend is evident in the susceptibility of plesiosaurs to the pathology, although stem group sauropterygians were almost never affected (not surprising for near-shore, shallow water animals). Cryptoclididae were the only plesiosaur family not regularly afflicted with decompression syndrome. The difference between this and other groups is most likely to have been behavioral and/or ecological.

INTRODUCTION

The presumed diving habits of certain extinct marine vertebrates have been identified through the recognition of a specific paleopathology, avascular necrosis (Rothschild and Martin, 1987, 1993; Moltani et al., 1999). The occurrence of avascular necrosis in Cretaceous mosasaurs (Reptilia, Squamata, Mosasauoidea) documents their susceptibility to caisson's disease ('the bends') or decompression syndrome (Rothschild and Martin, 1987). This 'window' on prehistoric behavior implies deep or repetitive diving habits or rapid changes of depth for mosasaurs and perhaps for Tertiary and Cretaceous marine turtles that also share the pathology (Rothschild, 1987, 1991). It has been unclear whether the susceptibility to avascular necrosis extended to other fossil reptiles (Rothschild, 1990).

The apparent absence of avascular necrosis in plesiosaur vertebrae was previously interpreted that plesiosaurs might have been spared the disease and by inference, 'the bends' (Rothschild, 1990). Why has avascular necrosis not been seen in plesiosaurs? Were plesiosaurs better adapted for repetitive, prolonged or deep diving than the mosasaurs and turtles, whose environment they at times shared? Did plesiosaurs not engage in such behavior to the extent that they became susceptible? Recent recognition of humeral and femoral head subsidence in plesiosaur fossils in museum collections suggests that these animals were susceptible to decompression syndrome in the manner seen in some fossil marine turtles (Rothschild, 1987, 1991). Plesiosaurs did, indeed, develop avascular necrosis.

Avascular necrosis is a pathology that includes the death of bone and can result from decompression syndrome (Rothschild, 1982). Decompression syndrome occurs in humans and other vertebrates subsequent to deep, prolonged, or closely repetitive subaqueous dives followed by inadequate staged decompression, or by rapid changes in depth (Pauley, 1965; Andersen, 1966; Strauss, 1970; Kooyman et al., 1973; Strauss and Sampson, 1986). Supersaturation of nitrogen in blood and tissues, because of increased ambient pressures (+1 atm at 10 m; +2 atm at 20 m, etc.) can result in nitrogen bubble formation subsequent to decompression. Loss of viability and death of bone may take place because of vascular occlusion, compromising nutrient availability in the watershed regions supplied by the affected vessel (Feldman et al., 1981; Michel et al., 1982; Ratchliffe, 1985; Resnick and Niwayama, 1988; Strauss and

Sampson, 1986). Typically, such blockage is the result of the formation of in vivo nitrogen bubble emboli, although hemocoagulation and red blood cell agglutination, resulting in decreased tissue perfusion, may be important causative factors as well (Strauss, 1970).

Specific types of vertebrae and proximal subarticular portions of femora and humeri are especially susceptible to necrosis (Rothschild and Martin, 1987; Resnick et al., 1981). The resultant loss of mechanical integrity makes necrotic bone in the propodials susceptible to normal compression stresses (such as those created by vigorous or prolonged muscle activity during swimming) across the glenoid and acetabulum, in the shoulder and hip joint, respectively (Feldman et al., 1981; Michel et al., 1982; Rothschild, 1982; Ratchliffe, 1985; Resnick and Niwayama, 1988). The damaged bone partly collapses, producing a visible subsidence zone.

Decompression syndrome is typically envisioned as a pathology associated with breathing from an external air supply in the case of humans, but it clearly can be manifested following breath-hold dives as well (Pauley, 1965; Strauss, 1970; Kooyman et al., 1973). Decompression syndrome occurs with repetitive (e.g., 40–50) dives of 2 minutes duration, to depths greater than 30 meters in humans, if the interval between dives is limited to only 2–4 minutes. It does not occur if the individual dives are spaced at 10 minute intervals (Pauley, 1965; Andersen, 1966; Strauss, 1970; Kooyman et al., 1973). In other extant vertebral groups, decompression syndrome has been documented notably in certain turtles, snakes, birds and pinnipeds (Berkson, 1967; Kooyman et al., 1973; Seymour, 1982), although protective mechanisms to prevent compressed nitrogen from entering their systemic circulations have also been noted (Berkson, 1967; Kooyman and Sinnett, 1982; Seymour, 1982; Leith, 1989). Protective mechanisms may include adaptive changes in behavior, morphology and/or physiology. Most vertebrate groups avoid decompression syndrome by virtue of the brevity of their dives (Kooyman et al., 1973), although with prolonged submergence, it can occur in most vertebrate taxa, modern cetaceans being the most obvious exception. Deep-diving whales perhaps avoid decompression syndrome by storing extra oxygen in an extensive vascular network, the retia mirabilia (Young, 1981) and/or compression of pulmonary gas into non-exchanges areas of the lung (Seymour, 1982).

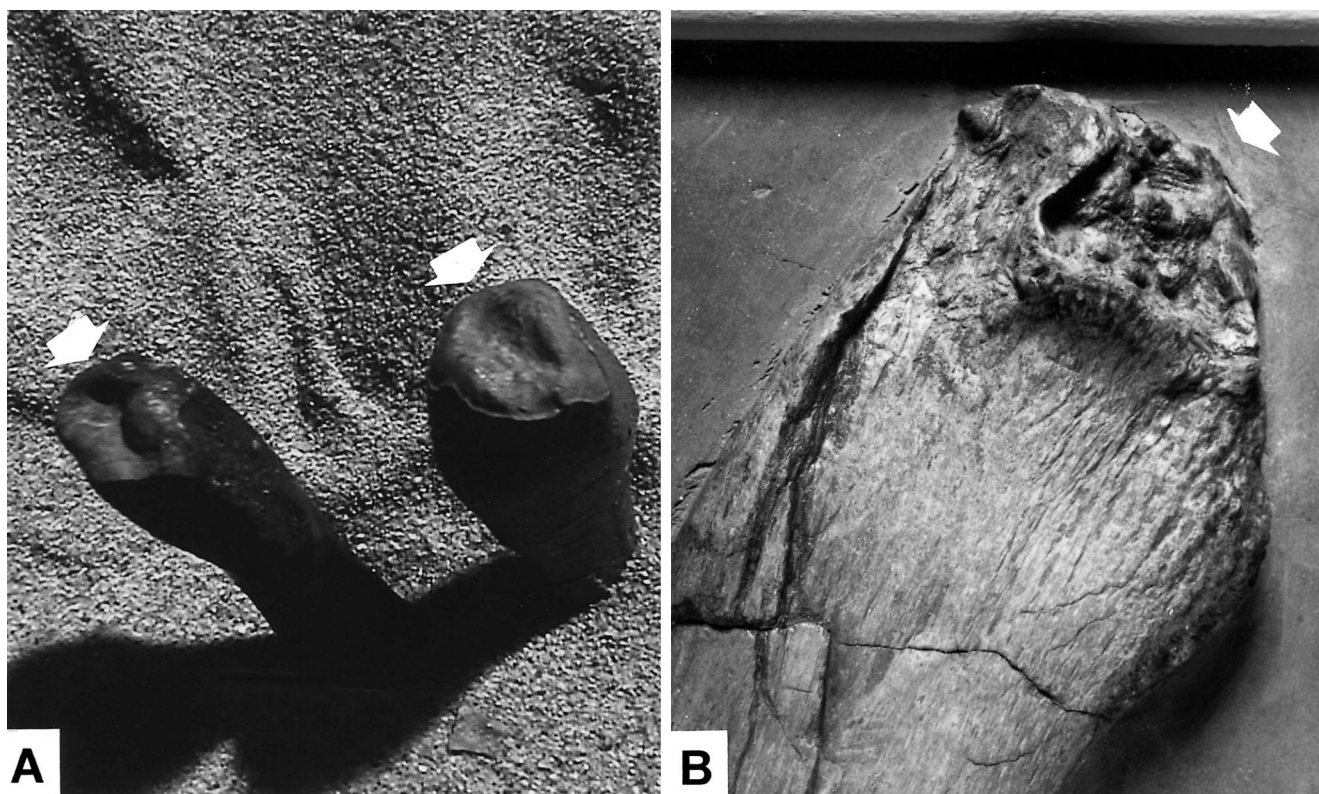


FIGURE 1. Avascular necrosis in *Neusticosaurus* PIMUZ T3935 humerus (A, B). Avascular necrosis in elasmosaurid FMNH P12752 (C). A), Areas of subsidence (arrows) noted at proximal articular surfaces. B), Defect in proximal articular surface (arrow). Irregular, ill-defined residual defect base is classic for avascular necrosis, as seen in humans.

METHODS

A systematic study of plesiosaur fossils was undertaken to assess the occurrence of propodial bone avascular necrosis and focal subsidence, any potential evolutionary changes in its frequency and to identify the structural characters that rendered their vertebrae resistant to avascular necrosis.

The humeri and femora of plesiosaurs were examined for gross evidence of avascular necrosis within the proximal articular surfaces of the bones, as evidenced by focal subsidence (collapse). Cross-sections of plesiosaur vertebrae, all non-necrotic, were analyzed for physical factors that could have prevented susceptibility to avascular necrosis. As many plesiosaurs are curated/exhibited as slab mounts, the areas of interest in these fossils are often inaccessible. This study examined only fully prepared, three-dimensionally preserved specimens and those slab-mounted examples in which the propodial articular surfaces are clearly visible. Radiologic facilities (sufficient to penetrate fossil bone) were not available at sites where affected specimens were curated.

Abbreviations—AMNH, American Museum of Natural History, New York City; CM, Carnegie Museum of Natural History, Pittsburgh, Pennsylvania; CMC, Cincinnati Museum Center, Cincinnati, Ohio; FMNH, Field Museum of Natural History, Chicago, Illinois; FHSM, Fort Hays State University (Sternberg Museum), Fort Hays, Kansas; BMNH, The Natural History Museum, London, England; LACM, Los Angeles County Museum of Natural History, Los Angeles, California; UCMP, Museum of Paleontology, University of California at Berkeley, Berkeley, California; NMC, National Museum of Canada, Ottawa, Ontario, Canada; NMNH, National Museum of Natural History (Smithsonian), Washington, D.C.; OMNH,

Oklahoma Museum of Natural History, Norman, Oklahoma; PIMUZ, Palaeontologisches Institut und Museum, Universitaet Zurich, Zurich, Switzerland; ROM, Royal Ontario Museum, Toronto, Ontario, Canada; RTM, Royal Tyrrell Museum, Drumheller, Alberta, Canada; SDSM, South Dakota School of Mines, Rapid City, South Dakota; TM, Tate Geological Museum, Casper, Wyoming; TTU, Texas Tech University Museum, Lubbock, Texas; KU, University of Kansas Museum of Natural History, Lawrence, Kansas; UW, University of Wyoming Geological Museum, Laramie, Wyoming; and YPM, The Yale Peabody Museum of Natural History, New Haven, Connecticut.

RESULTS

Macroscopic examination of 576 humeri and 479 femora reveals subsidence of the articular surface (Fig. 1) in 32 and 10 respectively (Tables 1, 2). These lesions were easily distinguished from osteochondrosis (failure of cartilage cone transformation into bone). The latter is a sharply defined focal articular surface defect, lacking elevated margins (Resnick and Niwayama, 1988; Rothschild and Martin, 1993). The sharply defined margins are clearly absent in Figure 1.

The frequency of affliction of humeri (5.6%) is significantly greater (Chi square (2 tail, 1 df) = 8.228, $P > 0.005$) than that in femora (2.1%). Sample size is sufficiently large to compare frequency of avascular necrosis across family groups within Plesiosauria. The frequency of occurrence typically is indistinguishable among the groups, with the exception of Cryptoclididae. Absence of avascular necrosis in the Cryptoclididae is statistically significant (Fisher exact test, $P = 0.0258$). The fre-

TABLE 1. Frequency of subsidence in plesiosaur and nothosaur humeri and femora.

	Humeri	Femora
Plesiosauridae		
<i>Attenborosaurus</i>	1	—
<i>Plesiosaurus</i>	7/30	2/34
Cryptoclididae		
<i>Cryptoclidus</i>	1/23	14
' <i>Tricleidus</i> '	2	2
Thaumatosauridae		
<i>Eurycleidus</i>	3	3
<i>Leptocleidus</i>	1	1
<i>Megalneusaurus</i>	2/2	2
<i>Rhomaleosaurus</i>	2	2
<i>Simolestes</i>	4	4
<i>Thalassiodracon</i>	2	6
' <i>Plesiosaurus</i> ' <i>macrocephalus</i>	2	1
Elasmosauridae		
<i>Aphrosaurus</i>	3/7	3/5
<i>Colymbosaurus</i>	1	—
' <i>Elasmosaurus</i> '	5	8
<i>Hydrotherosaurus</i>	1/4	1/2
<i>Muraenosaurus</i>	2	1
<i>Scanisaurus</i>	4/26	30
<i>Styxosaurus</i>	—	1
<i>Thalassomedon</i>	1/3	2
' <i>Cimoliasaurus</i> ' <i>limnophilus</i>	2	2
' <i>Thalassiosaurus</i> ' <i>ischiadicus</i>	—	1
Pliosauridae		
<i>Liopleurodon</i>	4	6
<i>Macroplata</i>	1	3
<i>Peloneustes</i>	2	2
<i>Plesiopleurodon</i>	5	1/9
<i>Pliosaurus</i>	2	1
' <i>Pliosaurus</i> ' <i>chiliensis</i>	3/7	1/15
<i>Polycotylus</i>	1	21
Polycotylidae		
<i>Dolichorhynchops</i>	1/5	1
<i>Polycotylus</i>	3/8	1/9
<i>Trinacromerum</i>	9	2
	2/8	1/2
Pachypleurosauridae		
<i>Neusticosaurus</i>	4/346	239
<i>Serpianosaurus</i>	33	26
<i>Silvestrosaurus</i>	1	2
Nothosauridae		
<i>Ceresiosaurus</i>	16	13
<i>Lariosaurus</i>	4	4
<i>Nothosaurus</i>	2	2

quency in Pliosauridae was not statistically different from that of Plesiosauridae (Fisher exact test, $P = 0.077$).

Assessment of variation in frequency within families is compromised by sample size. There is no variation within families, as exemplified by the Polycotylidae. Absence of avascular necrosis in *Polycotylus*, compared to the other Polycotylidae (*Dolichorhynchops* and *Trinacromerum*) reveals no statistically significant differences, whether measured according to humeral involvement (Fisher exact test, $P = 0.128$) or analyzing all elements (Fisher exact test, $P = 0.137$).

Avascular necrosis is not present in vertebrae of those plesiosaur specimens in which affected associated humeri or femora are present. Physical examination of cross sections of plesiosaur vertebrae reveals large vascular channels with multiple anastomoses. There is no evidence for the watershed type vascular supply required for susceptibility to avascular necrosis.

DISCUSSION

Plesiosaurs are extinct marine predatory reptiles that minimally ranged from the latest Triassic to the latest Cretaceous. They are sauropterygians characterized by the elaboration of

TABLE 2. Specimens with subsidence diagnostic of avascular necrosis.

<i>Plesiosaurus</i> sp. BMNH R32393 2 femora
BMNH R46486 humerus
BMNH 32395 humerus
BMNH 41859 humerus
YPM 421
YPM 462
YPM 456 (3 humeri)
<i>Cryptoclidus</i> BMNH R2413 humerus
<i>Megalneusaurus rex</i> UW 4602 2 humeri
Elasmosauridae YPM 1641 1 femur
KU 441 humerus
FMNH P12752 humerus and femur
TMM 9217 humerus and femur
<i>Muraenosaurus</i> sp. NMNH 419631 humerus
LACM 2802 humerus
<i>Muraenosaurus dubroviensis</i> BMNH 2427 humerus
<i>Muraenosaurus leedsi</i> BMNH R2427 humerus
<i>Styxosaurus snowii</i> SDSM 451 humerus
<i>Peloneustes philarchus</i> BMNH 47410 femur
<i>Pliosaurus</i> sp. BMNH 2437 humerus
FMNH p25312 humerus
<i>Pliosaurus andrewsi</i> BMNH R3891 humerus and femur
Polycotylidae SDSM 23020 humerus
<i>Dolichorhynchops</i> sp. FHSU VP-698 3/5 humeri, 1/3 femora
<i>Trinacromerum</i> sp. RTM 93.21.1 1/3 humeri and 1/1 femur
OMNH 071 2/2 humeri
<i>Neusticosaurus edwardsi</i> PIMUZ T3935 2 humeri
PIMUZ T3428 humerus
<i>Neusticosaurus pusillus</i> PIMUZ T3392 humerus

two pairs of hyperphalangic limbs, indicative of paraxial subaqueous propulsion, and by a more or less elongated neck and short, rigid thorax (Storrs, 1993). Most are presumed to have been piscivorous (Massare, 1987), although along with fish remains (Cope, 1871; Brown, 1904; Patterson, 1975; Storrs, 1995), cephalopod jaw elements and hooklets have been observed as stomach contents (Zhuravlyev, 1943; Tarlo, 1959; Sato and Tanabe, 1998) and tooth marks on isolated bones indicate that large pliosaurs preyed upon other reptiles (Clarke and Etches, 1992).

Preconceived notions often so direct our thinking that some patterns seldom get explored (Spodick, 1975). Such is the case with plesiosaurs and associated evidence of decompression syndrome. Premising an analysis for avascular necrosis in plesiosaurs on vertebral radiologic findings, as proved so useful in the study of mosasaurs, was ultimately misleading (Rothschild, 1990). Recognition of decompression syndrome in mosasaurs and turtles is based upon the identification of a specific associated pathology, avascular necrosis (Rothschild, 1987, 1991; Rothschild and Martin, 1987). Radiography of mosasaur vertebrae may be used to identify the pathognomonic band-like zones of dead/dissolved bone indicative of this pathology (Resnick and Niwayama, 1988; Resnick et al., 1981; Rothschild and Martin, 1987, 1993). However, mosasaur and turtle surveys also reveal analogous proximal humeral and femoral damage. Rather than simply a band of dead, liquefied bone in the propodial head, this mechanically-compromised region collapses subsequent to the fracture of the surviving subchondral bone due to the transmission of compressive forces, resulting from paraxial locomotion. The result is an appearance of partial articular surface collapse (subsidence). By focusing analysis in plesiosaurs on the search for linear radiolucent features within vertebrae (never found), propodial head subsidence in this group had been overlooked previously.

In the case of turtles, their vertebral construction is such that nutrient dependence upon a single vessel is minimal. Thus, the substrate for recognition of vertebral avascular necrosis does not exist in turtles. The large and anastomotic internal vascular

TABLE 3. Frequency of subsidence in plesiosaur and nothosaur humeri and femora.

	Humeri	Femora
Plesiosauridae	7/31	2/34
Cryptoclididae	1/25	16
Thaumatosauridae	2/16	19
Elasmosauridae	9/50	4/53
Pliosauridae	3/22	2/57
Polycotylidae	6/30	2/14
Pachypleurosauridae	4/380	267
Nothosauridae	22	19
Total	32/576	10/479

supply of plesiosaur vertebrae similarly provides a wealth of blood supply resources, such that occlusion of one vessel would not compromise bone nutrition. Therefore, vertebrae are not targets of avascular necrosis in plesiosaurs. The prominent nutritive foramina (foramina subcentralia) of plesiosaurs and the large degree of variability in their number and placement are seemingly relevant to the nonsusceptibility of plesiosaur vertebrae to avascular necrosis. Indeed, such enhanced vascularization, not evident in stem-group plesiomorphic sauropterygians, is potentially an evolutionary adaptation to greater, more vigorous diving habits in the Plesiosauria.

Sauropterygians display an ecological trend from littoral, shallow-water taxa to more fully ocean-going, open marine forms that is correlated both with stratigraphic and phylogenetic position (Sues, 1987; Storrs, 1991, 1993). Pachypleurosaur and stem-group Eusauropterygia (stem Nothosauriformes) were typically near-shore animals that lack foramina subcentralia. Plesiosaurs possess them as an autapomorphy. Although plesiosaurs do not exhibit vertebral avascular necrosis, they share with mosasaurs and turtles a humeral and femoral articulation susceptibility. Propodial head subsidence provides an indication of avascular necrosis, and therefore of decompression syndrome, in plesiosaurs (Tables 1–3). Indeed, plesiosaurs share with some turtles (Rhodin et al., 1981) a transepiphyseal vascularization of the chondrous and osseous regions of the propodial head (extensive vascularization across the growth plate). This morphology is also present in whales (Felts and Spurrell, 1965) and sirenians (Rhodin et al., 1981), but is otherwise unique. If transepiphyseal vascularization is a consequence of rapid growth (Rhodin, 1985), an increased susceptibility to avascular necrosis in turtles and plesiosaurs (see discussion of cetaceans, above) might be inferred, should the transepiphyseal blood supply be compromised, as e.g., by decompression syndrome (Rothschild, 1991). Among stem-group Sauropterygia, which lack transepiphyseal vascularization (Glen W. Storrs, pers. obs.), only a single genus of pachypleurosaur, *Neusticosaurus*, has been found to exhibit (rarely) humeral head subsidence (Fig. 1).

Examination of the differential occurrence of avascular necrosis in various plesiosaurian 'families' provides potential insight into their diving behaviors (Table 3). There does not yet exist a complete cladistic classification of the Plesiosauria, but a reliance on traditional family groupings does elucidate functional types at the very least, a result more relevant to our purposes. The implication is that the traditional Plesiosauridae, Elasmosauridae, Polycotylidae and Pliosauridae engaged in deeper or more repetitive diving, or initiated more rapid changes of depth, than did the Cryptoclididae. What about these groups led to different susceptibilities to pathology? Assuming, in the absence of evidence to the contrary, that plesiosaurs had not developed physiological adaptations to prevent decompression syndrome in their limbs as have apparently modern cetaceans, the differences are most likely to have been behavioral/

ecological. We have already noted an apparent morphological adaptation in their vertebrae.

No phyletic trend is evident, as avascular necrosis is seen in some of the earliest plesiosaurs (Plesiosauridae), but also in some of the last (Elasmosauridae, Polycotylidae). Thus, it appears that Plesiosauria as a whole did not develop any physiological adaptation over the course of evolutionary history. The cryptoclidids lack symptoms of decompression syndrome and were also Upper Jurassic and Cretaceous in age. Stem-group sauropterygians rarely exhibit the pathology, probably because of their occurrence in more littoral, shallow-water habitats.

Plesiosaurids include some of the earliest taxa. They, like the advanced elasmosaurids, often contain stomach stones or gastroliths within their body cavities. Some controversy remains surrounding the probable function of these stones, but one possibility is their use as a buoyancy compensation mechanism (Storrs, 1993; Taylor, 1993, 1994). Gastroliths, as opposed to hydrodynamic forces, do not rely upon the motion of the animal through the water for buoyancy compensation. Thus, in plesiosaurs with such stones, it may be assumed that they had a relatively greater dependency on slow maneuverability (Taylor, 1993). Ambush predation (as opposed to active pursuit) has, indeed, been suggested for these long-necked animals (Massare, 1988). Fast, relatively short-necked forms (e.g., polycotylids and pliosaurs) rarely contain large quantities of gastroliths, a circumstance suggestive of a different ecological niche (Storrs, 1993). Nevertheless, all of these groups exhibit a similar frequency of avascular necrosis, indicative of decompression syndrome. They must have been more or less equally exposed to the habits of deep, prolonged or repetitive diving, perhaps in search of their prey, or of rapid changes in depth, possibly during pursuit of prey or flight from predators. The greater incidence of head subsidence in humeri than in femora may indicate a greater reliance on the use of the forelimb, or at least greater compressive stresses there. Why cryptoclidids, a mesodiran group with advanced limb structure, should not frequently also exhibit avascular necrosis (1 of 25 humeri and 0 of 16 femora examined) remains a mystery. One can only speculate that some significant behavioral difference is in evidence (e.g., habitual occupation of the uppermost water column. Such is the limit of pathology as a new tool for paleoecological inference.

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