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NEURAL BONE SYNARTHRODIAL JOINT INFECTION CAUSING INTER-NEURAL SYNOSTOSIS AND LORDOTIC SPINAL DEFORMITIES IN CAPTIVE-RAISED SEA TURTLES (CHELONIIDAE)

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ABSTRACT

Analysis of lordotic spinal deformities occurring in captive-raised sea turtles (n=272; 65.1% deformed) demonstrated focal inflammatory lesions in inter-neural and neural-costal synarthrodial joint sutures leading to proliferative fibroblastic response, neural synostosis, and dorsal neural tethering. Continued normal endochondral ossification and growth of ventral thoracic centra caused eventual lordotic spinal deformities. Biochemical and histologic analysis demonstrated no evidence of nutritional fibrous osteodystrophy. Etiology was presumed to be microbial infection, possibly related to unrecognized ulcerative shell disease.

Key words : spinal deformity, lordosis, neural bones, joint infection, sea turtles, Chelonia mydas, Caretta caretta, Lepidochelys olivacea

INTRODUCTION

The development of spinal deformities in turtles has generated much previous interest. RHODIN et al. (1984) reviewed all previous literature on spinal deformities in sea turtles and calculated incidences of occurrence of deformities in the various populations examined : they found a total incidence of 0.11% spinal deformities in their review of 11,726 specimens, with the incidence of lordosis being

only 0.03%. COKER (1910) examined 208 hatchling Caretta caretta from transplanted nests and found a 0.5% incidence of spinal irregularities. He postulated that the cause of the spinal deformities was injury to the embryos produced by crushing or pressure from the sand surrounding the nest. Observations of 984 hatchling sea turtles from Queensland, Australia by MOORHOUSE (1933) indicated a 0.1% incidence of spinal deformities. DERANIYAGALA (1939) worked with Lepidochelys olivacea from Sri Lanka, and found a 0.3% incidence of spinal irregularities in wild turtles (n=378). A review by NIXON and SMITH (1949) listed the occurrence of spinal deformities in fourteen species of turtles belonging to five different families. These authors hypothesized that the irregularities occurred at various stages of embryonic development, during which the vertebral column was deformed into abnormal positions. Spinal deformities occur among sea turtles from West Africa according to VILLIERS (1958), however, there is no mention of incidence. Several species of sea turtles were raised by the Florida Department of Natural Resources as a conservation measure; 0.4% of the 2,500 turtles developed lordotic spinal deformities (WITHAM and FUTCH, 1977). There appears to be a high incidence of kyphotic and lordotic spinal deformities (1.2%; n=409) in wild adult Chelonia mydas in the Bali region of Indonesia (RHODIN et al., 1984).

Spinal deformities in non-marine turtles have been reported by several authors. PARKER (1901) described several specimens of Clemmys insculpta that exhibited spinal abnormalities in the same anatomical position; he regarded this property as evidence of early embryonic disruption affecting both ectodermal and mesodermal bone formation. In describing a distorted carapace of a tortoise, Testudo graeca, WANDOLLECK (1904) suggested that the condition might have resulted from embryonic arrest at some critical stage in development. HILDEBRAND (1930) studied the diamondback terrapin, Malaclemys terrapin, and suggested that the amount of oxygen available to developing embryos may be an important factor in producing spinal deformities. LYNN and ULLRICH (1950) reported on shell abnormalities in Chrysemys picta and Chelydra serpentina as a condition of suboptimal moisture relationships of developing turtles eggs. MITCHELL and YNTEMA (1973) produced scoliosis and other deformities in embryos of Chelydra serpentina experimentally exposed to the pesticide malathion. FRYE and CARNEY (1975) documented a case of parathyroid adenoma in a Geochelone carbonaria causing osteodystrophy and mild spinal deformity. LOPEZ JURADO et al. (1979) illustrated a lordotic Testudo hermanni believed to have become deformed as a result of exposure of the dorsal carapace to damage from a brush fire. WILHOFT (1980) reported the gradual appearance of lordosis during growth in a laboratory-raised Chelydra serpentina but did not document the etiology of the deformity. GUTZKE et al. (1987) described scute and tail deformities in Chrysemys picta caused by abnormal incubation temperatures, but found no evidence of increased deformities caused by altered moisture relationships.

Despite the various hypotheses advanced as to the cause of the deformities reported in the preceding papers, very few have attempted to investigate the underlying pathologic mechanisms. There have been no histopathologic studies of the deformities, nor have radiographic or serologic investigations been carried out. Without a basic understanding of the histological appearance of these deformities it would be difficult to advance hypotheses of etiology.

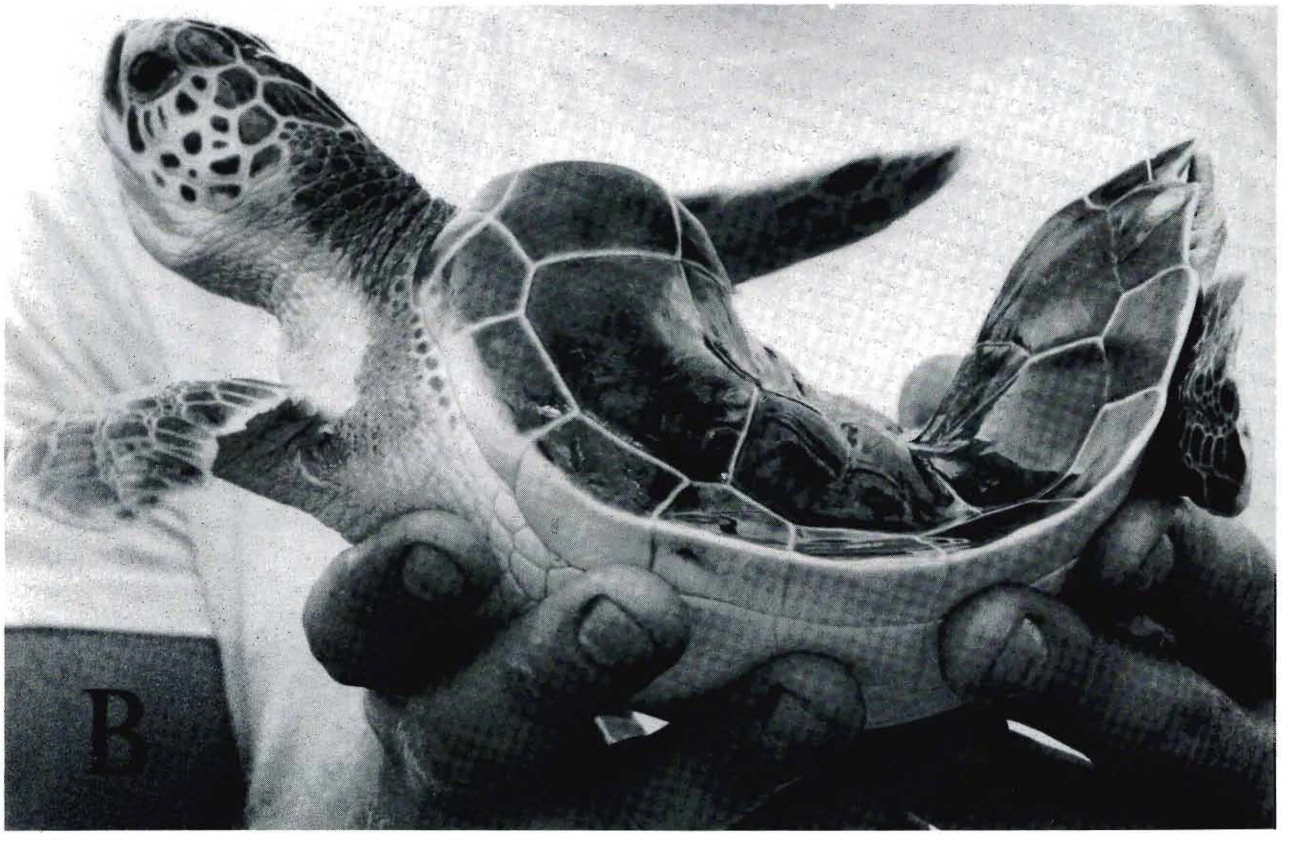
In this paper, we describe the post-embryonic development of lordosis (concave spinal deformity) in a group of artificially incubated and captive-raised sea turtles. We utilize anatomic, radiographic, histologic, and serologic investigations to characterize the condition and to compare the group of deformed captive sea turtles to both normal captive specimens and normal wild sea turtles. We document that the deformities occurred as a result of infection in the inter-neural and neural-costal bone synarthrodial joint sutures of young growing turtles, leading to premature inter-neural and neural-costal fusion and synostosis with subsequent development of lordosis through relative thoracic vertebral overgrowth associated with pathologic neural bone tethering. An infectious origin for the development of spinal deformities in growing turtles has not previously been recorded.

METHODS AND MATERIALS

A total of 304 sea turtles (Cheloniidae) were utilized in the study. A control group of 32 wild specimens of green sea turtles (Chelonia mydas), loggerheads (Caretta caretta), and Pacific olive ridleys (Lepidochelys olivacea) were either nesting adult females (n=20), beached adolescents (n=5), or natural nest hatchling mortalities (n=7). A study group of 272 captive specimens represented the same three species. Of this group, 82 were "Headstart" specimens of C. mydas from a single clutch collected at Fort Lauderdale, Florida, 86 were "Headstart" specimens of C. caretta, also from a single clutch collected at Fort Lauderdale, Florida, and 104 were hatchlings of a single clutch of L. olivacea eggs collected at Ostional, Costa Rica. All captive specimens were hatched following artificial incubation of transpor-

Figure 1. A. Deformed yearling *Caretta caretta*. B. Deformed yearling *Chelonia mydas*.

Figure 1



ted natural nests, and raised in Fort Lauderdale, Florida in large aquarium conditions. Daily diet included ample quantities of beef liver, whole fish, freezedried plankton, lettuce, and pulverized oyster chips.

Complete spinal columns of one-year and three-year old normal and deformed turtles were sectioned longitudinally and transversely. Histologic sections of decalcified thoracic vertebral and adjacent neural elements of five-month to three-year old normal and deformed turtles were prepared and processed in routine fashion (OGDEN et al., 1981) and stained with hematoxylin-eosin. Specimens were examined from deformed L. olivacea (n=4), C. caretta (n=4), and C. mydas (n=5), as well as normal specimens of the three species (n=5).

Radiographic investigation was carried out on both whole specimens and isolated thoracic vertebral columns (removing the paravertebral bony elements with a band saw to allow unobstructed lateral radiographs of the thoracic spine and midline neural elements). Radiographs using standard clinical techniques were obtained of deformed captive turtles (n=5), normal captives (n=5), and wild turtles (n=3).

Because nutritional deficiencies have been suggested as a possible cause of spinal deformities (nutritional fibrous osteodystrophy; see FRYE, 1981), serologic analysis of blood calcium, phosphorus, and magnesium content was performed. Serum samples were obtained via the paravertebral sinus (MENZIES et al., 1983) from wild nesting adult females (n=20) and juvenile captive turtles (n=20 total : n=7 normal, n=13 deformed). Approximately 5 ml blood was obtained from each animal, serum portions separated from the red blood cells (centrifuged at 1500 rpm for 10 min) and frozen prior to analysis. The samples were assayed on a random access auto-analyzer (Acclaim - Electro Nucleonics, Fairfield, NJ) and a discrete clinical analyzer system (ACA - DuPont Inc., Wilmington, DE). The calcium assay on the Acclaim and ACA is a dye-binding methodology where calcium forms a purple complex with o-cresol-phthalein complexone in an alkaline medium. The reaction is spectrophotometrically measured at a wavelength of 540 nanometers. The Aca phosphorus method measures the absorption of reduced phosphomolybdate complex at a UV wavelength of 340 nm. The Acclaim magnesium procedure is based upon a colorimetric detection of a metallochromic dye (wavelength of 500 nm) formed when magnesium and Calmagite complex. The Student's t-test was utilized to evaluate the significance of the serologic data.

RESULTS

Of the 272 artificially hatched and captive-raised sea turtles in this study, 177 (65.1%) developed varying degrees of lordotic spinal deformities (Fig.1). Lordosis is also known as "sway-back" and is defined as a concave deformity of the spine in a sagittal plane with the apex of the deformity pointing ventrally. The opposite condition of kyphosis ("hump-back") was not observed in this group. A few specimens additionally had very mild scoliosis associated with their severe lordosis. All specimens were normal in appearance at hatching and developed the deformity gradually during growth, becoming increasingly severe with time. In addition, all of the deformed specimens also developed carapacial scute abnormalities during growth. All hatchlings had normal scute patterns, but as the lordotic spinal deformities developed, the scute patterns also changed, with the gradual appearance of both fused and supernumerary scutes, becoming increasingly complex and distorted with time (see Fig.2). Of the 82 C. mydas hatchlings, 72 (87.8%) developed deformities, first becoming noticeable at about age 8 to 9 months. Of the 86 C. caretta hatchlings, 75 (87.2%) developed deformities, first appearing at about age 8 to 9 months. Of the 104 L. olivacea hatchlings, 30 (28.8%) developed deformities, appearing at about age 9 to 10 months.

The serologic analysis (see Table 1) demonstrated no significant differences in the calcium levels between wild nesting adult female turtles and the captive juveniles. However, the wild nesting female group had significantly higher concentrations of magnesium and lower levels of phosphorus than the captive juveniles. Despite the differences between wild and captive animals, there were no significant differences in the levels of calcium, phosphorus, or magnesium between the deformed and normal captive juveniles (see Table 2). The serologic analysis demonstrated no clear evidence of nutritional deficiencies as the cause of the deformities (see discussion below).

Radiographs and gross longitudinal sectioning of the thoracic spine and neural elements revealed that in deformed specimens there were specific patterns of deformity (see Fig. 4). The primary lesion appeared to be premature fusion or failure of segmentation of the dorsal neural elements with partial fusion of the neurals and continued normal growth of the ventral thoracic centra. This created a dorsal tethering effect and subsequent development of lordosis as the elements ventral to the spinal cord continued to grow but the dorsal elements slowed in their growth and fused. There was secondary extreme thickening of the neural bones at the apex of the deformity, with loss of neural bone sutures and the formation of a very dense and thick bony carapace. The ventral portions of the thoracic centra also become slightly lordotic at the apex of the deformity, but normal cartilaginous growth plates were maintained and the mild deformities were apparently only secondary to the primary dorsal fusion tethering effect. In two large three-year old C. mydas with severe deformities there were two separate areas of lordosis with intervening normal neural and thoracic elements. Both specimens had normal vertebrae and neurals at T1 and 2, T6, and T8-10 in one specimen, T9-10 in the other, with the areas

Figure 2. Carapacial scute pattern of severely lordotic 3-year old Chelonia mydas (see lateral radiograph of spine of this animal in Fig. 4). Scute pattern and shell shape were normal at hatching.

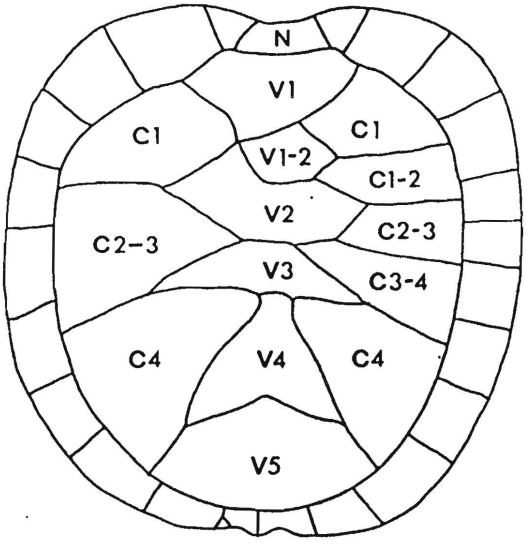


TABLE 1. Results of the serologic analyses: means, standard deviations, and t-test results of the wild population (nesting adult females) vs. the captive population (juveniles) [mixed *Chelonia mydas*, *Caretta caretta*, and *Lepidochelys olivacea*].

Analytes (millimoles/Liter)	Wild Population (n=20)		Captive Population (n=20)		t-test ($t_{.995}$=2.58,df=38)
	mean	S.D.	mean	S.D.	
calcium	1.65	1.1	1.40	0.6	t=0.86
phosphorus	1.58	0.5	3.60	1.4	t=2.77
magnesium	3.46	0.6	2.60	1.3	t=6.40

Table 2. Results of the serologic analyses : means, standard deviations, and t-test results of the normal captives vs. the deformed captives [mixed Chelonia mydas, Caretta caretta, and Lepidochelys olivacea].

Analytes (millimoles/Liter)	Normal Captives (n=7)		Deformed Captives (n=13)		t-test ($t_{.995}$=2.87, df=18)
	mean	S.D.	mean	S.D.	
calcium	1.40	0.5	1.40	0.7	t=0
phosphorus	3.50	1.2	3.65	1.5	t=0.4
magnesium	2.54	1.2	2.65	1.4	t=0.3

of lordotic involvement being T3-5 and T7 in one specimen, T3-5 and T7-8 in the other. In both, the anterior lordotic curve at T3-5 was the major curve accounting for most of the deformity. Normal elements were present at T6 in both specimens, separating the anterior and posterior curves from each other. The neural foramina for the spinal nerves at the deformed centra T3-5 and T7 (plus T8 in one specimen) were markedly compromised, forming narrow vertical fissures. At the normal T1-2 and T6 levels the spinal nerve foramina had a normal large round shape.

Histologic sectioning of the thoracic spine and neural elements in 5-month, 8-month, and 3-year old normal and deformed specimens demonstrated the sequential development of the lesions causing the gradual appearance of the lordotic deformities.

Sections of 5-month old normal specimens showed normal growth patterns with separate dermatomally-derived neural and costal bones occurring as separate early membranous ossifications. Separate sclerotomally-derived ventral thoracic vertebral centra, paramedian costal heads, and dorsal thoracic neural arches occurred as separate early endochondral ossifications (see KALIN, 1945 and BURKE, 1989 for further discussion). The inter-neural and neural-costal synarthrodial joints were wide open and composed of a thick fibrous acellular mesenchymal tissue.

In 8-month and 3-year old normal specimens the inter-neural and neural-costal bone synarthroses became increasingly complex with extensive bony interdigitations separated by a thick relatively acellular fibrous connective tissue (see Figs. 3A and 3B) (see also SUZUKI, 1963 for further discussion). The ventral thoracic centra, paramedian costal heads, and dorsal thoracic neural arches remained separated and growing through endochondral ossification in three separate directions from a triradiate cartilage just lateral to the spinal cord. The dorsal portions of the dorsal thoracic neural arches became fused with the ventral portions of the neural bones, as did the distal portions of the paramedian costal heads and the overlying membranous costal bones.

In deformed 8-month and 3-year old specimens these growth processes were disturbed. In 8-month old specimens focal inflammatory lesions (described below) caused disruption of normal synarthrodial inter-neural and neural-costal relationships. In 3-year old specimens the resultant deformities with synarthrodial fusions and resultant lordosis were evident.

Serial sections of an 8-month old mildly deformed C. caretta demonstrated focal areas of chronic inflammatory infiltrates invading inter-neural and neural-costal synarthrodial joints (see Fig. 3C). Not all synarthroses were involved. Posterior and anterior carapacial sutures were usually normal. Some mid-carapacial neural-costal sutures were affected on only one side of the midline, with the opposite side normal. The inflammatory lesion was characterized by a highly cellular infiltrate filling the fibrous synarthrosis, consisting of a moderate number of polymorphonuclear heterophils (SYPEK and BORYSENKO, 1988), many melanomacrophages and free melanin granules, as well as numerous proliferative fibroblasts (see Fig. 3D). In most sections there was no evidence of spread of the inflammatory tissue beyond the synarthrodial joints. There was no osteomyelitis of the adjacent neural or costal bones, no paravertebral abscesses, nor other inflammatory lesions. However, in one section, there was conti-

guous spread of inflammatory cells into the neural-costal synarthrosis from a superficial epidermal focus of chronic inflammatory destruction suggestive of a carapacial scute surface or inter-laminar sulcus lesion. In all sections there was normal endochondral ossification and growth of the ventral thoracic centra, the paramedian costal heads, and the ventral portions of the dorsal neural arches. The lesion occurring in the synarthrodial sutural joints could best be characterized as chronic focal synarthrodial joint infection with proliferative fibroblastic response. Though bacteria were not seen, and cultures unfortunately not obtained, the most likely etiology of the infection is presumed to be microbial.

Serial sections of a 3-year old severly deformed C. mydas demonstrated no active infection, but complete destruction of inter-neural and neural-costal synarthrodial joints with resultant inter-neural and neural-costal fusion. Neural and costal membranous bone growth had essentially ceased, but endochondral ossification of the ventral centra, paramedian costal heads, and ventral portions of the dorsal neural arches was progressing at a normal rate (see Figs. 5 and 6). This continuous normal ventral thoracic growth combined with dorsal neural-costal fusion had led to ventral overgrowth with dorsal tethering, leading to severe lordosis. In some sections the former neural-costal synarthroses remained as thin, straight, inactive fibrous fissures surrounded by inactive membranous bone (see Fig. 6B), extending in continuous fashion from the carapace surface to the neural foramina. In other sections there was more or less complete bony synostosis across the former synarthrosis.

In comparing the 8-month old specimen with the 3-year old specimen, it was clear that the focal inflammatory lesion with proliferative fibroblastic response had led directly to synarthrodial joint fusion; and that the infection had subsequently cleared, leaving the animal to become increasingly lordotic through continued normal ventral thoracic endochondral bone growth tethered by fused dorsal neural-costal membranous bone growth.

Of particular note is that anatomic, radiographic, and histologic analyses failed to reveal any evidence of nutritional fibrous osteodystrophy in either normal or deformed juvenile turtles (WALLACH and HOESSLE, 1968; ZWART and Van De WATERING, 1969; FRYE, 1981). All animals had normal endochondral ossification patterns and normal bone density. In addition, there was no evidence of osteitis deformans (Paget's disease) or other bone or joint pathology.

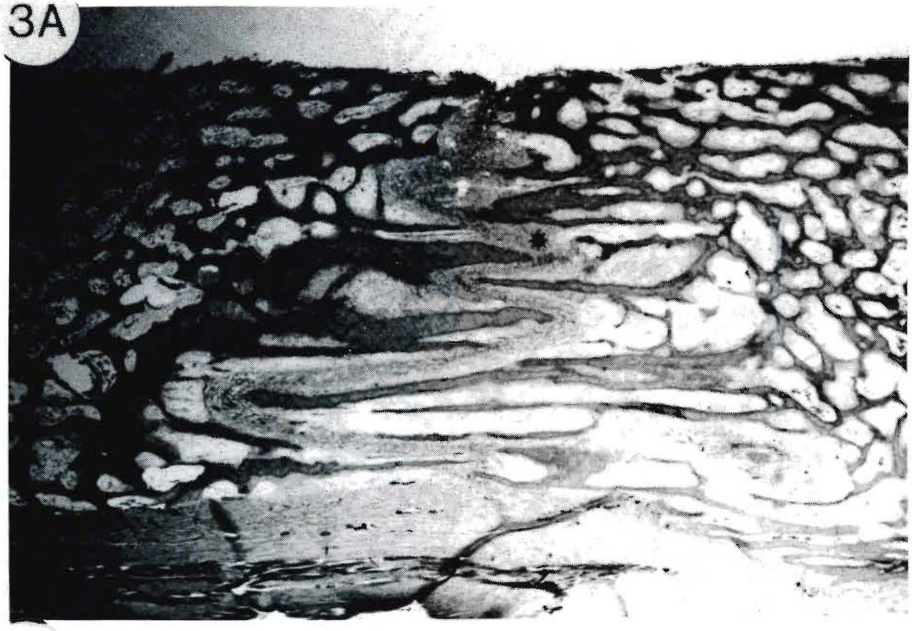
Figure 3. A. Normal inter-neural synarthrodial joint suture (at asterisk) in 3-year old normal Chelonia mydas (compare with deformed 3-year old C. mydas in Fig. 6B). Note extensive bony interdigitations from both neural bones with intervening fibrous connective tissue. B. Enlargement of synarthrosis at asterisk in A, showing relatively acellular fibrous connective tissue. C. Neural-costal synarthrodial joint suture in 8-month old minimally deformed Caretta caretta, showing highly cellular inflammatory infiltrate filling synarthrosis (at asterisk). D. Enlargement (same magnification as in B) of cellular inflammatory infiltrate at asterisk in C, showing polymorphonuclear heterophils, melanomacrophages, free melanin granules, and numerous proliferative fibroblasts.

Figure 4. Lateral radiograph of thoracic spine and midline neural elements of 3-year old Chelonia mydas with severe lordosis (lateral shell elements removed). Ventral thoracic vertebral centra T1-T9 labeled 1-9. Abnormal neural foraminal fissure labeled at F5, normal neural foramen labeled at F6.

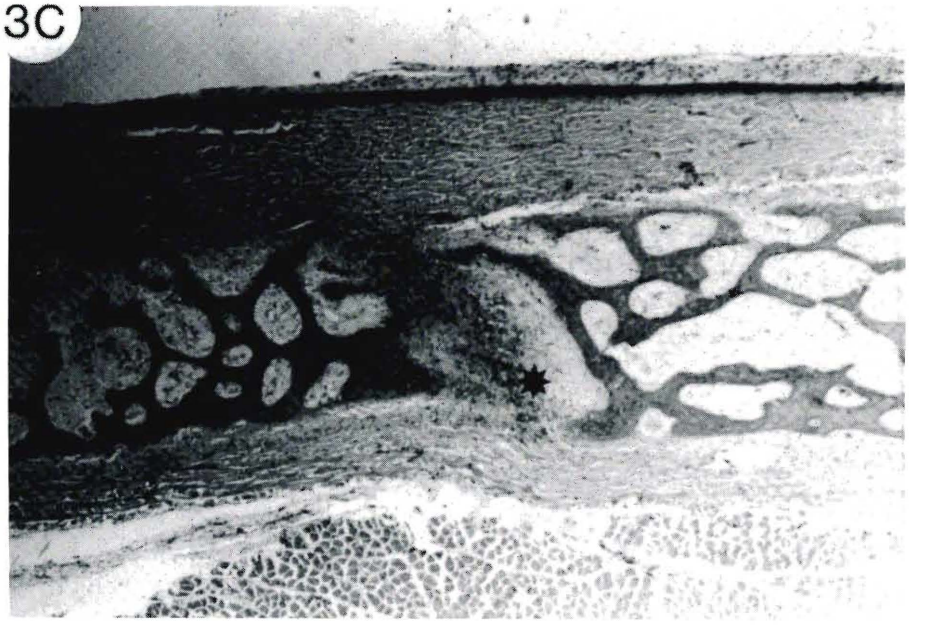
Figure 5. Transverse histological section of thoracic spine and neural elements of 3-year old severely lordotic Chelonia mydas (same specimen as in Figs. 2 and 4, section approximately through T4). V = ventral thoracic vertebral centra; s = spinal cord (highly compromised); R = paramedian costal (rib) heads; D = dorsal thoracic neural arches; N = neural bone; c = costal bone; m = paravertebral longitudinal muscles; A = box showing normal endochondral ossification, enlarged in Fig. 6A; B = box showing site of neural-costal synarthrodial fusion, enlarged in Fig. 6B.

Figure 6. A. Enlargement of box A in Fig. 5, showing normal endochondral ossification of ventral thoracic vertebral centra (for description of normal patterns of endochondral ossification and growth in sea turtles, see RHODIN, 1985). B. Enlargement of box B in Fig. 5, showing neural-costal synarthrodial fusion with thin, inactive fibrous suture (at asterisk) with inactive surrounding membranous bone (compare with actively growing synarthrosis seen in Fig. 3A).

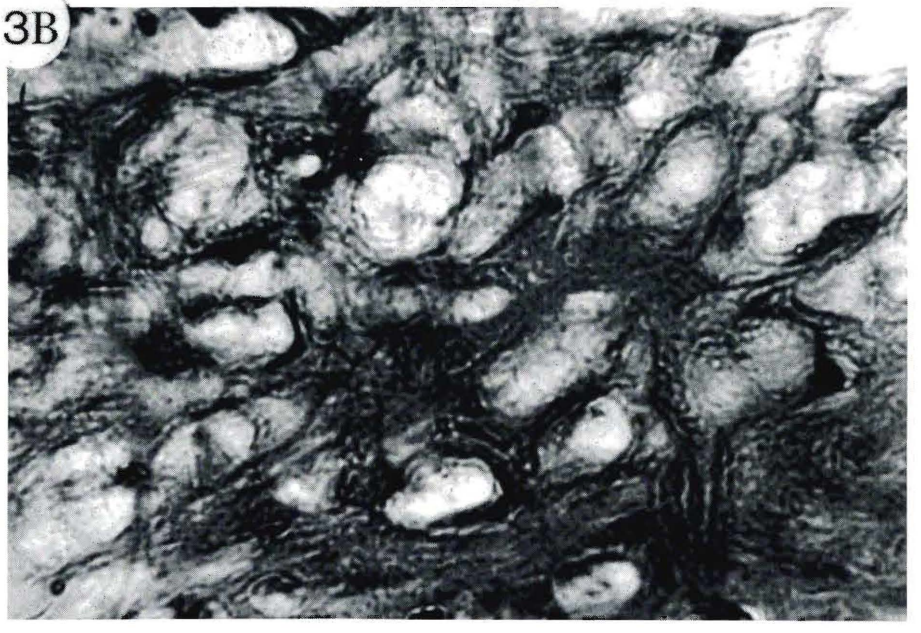
3A



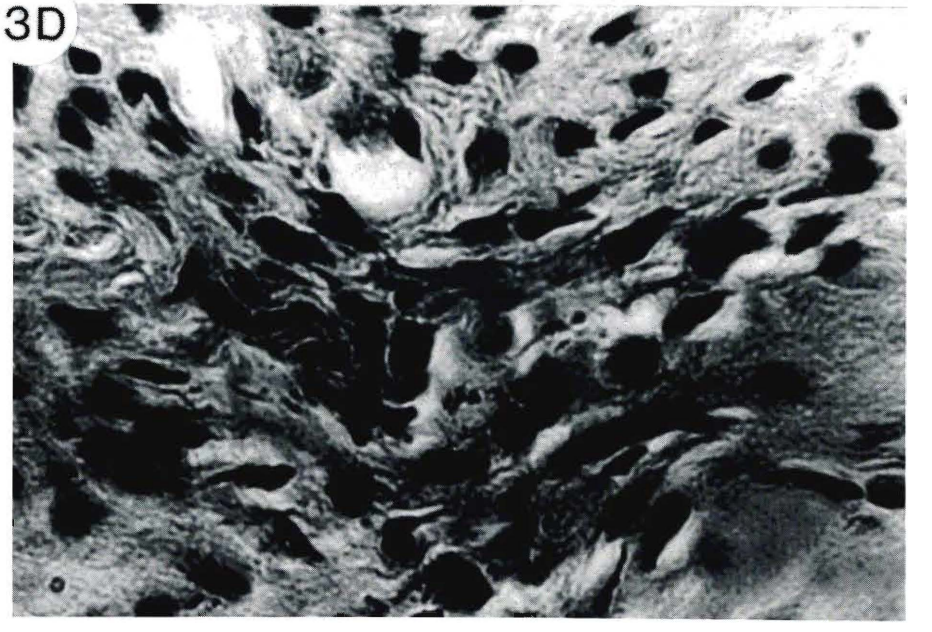
3C

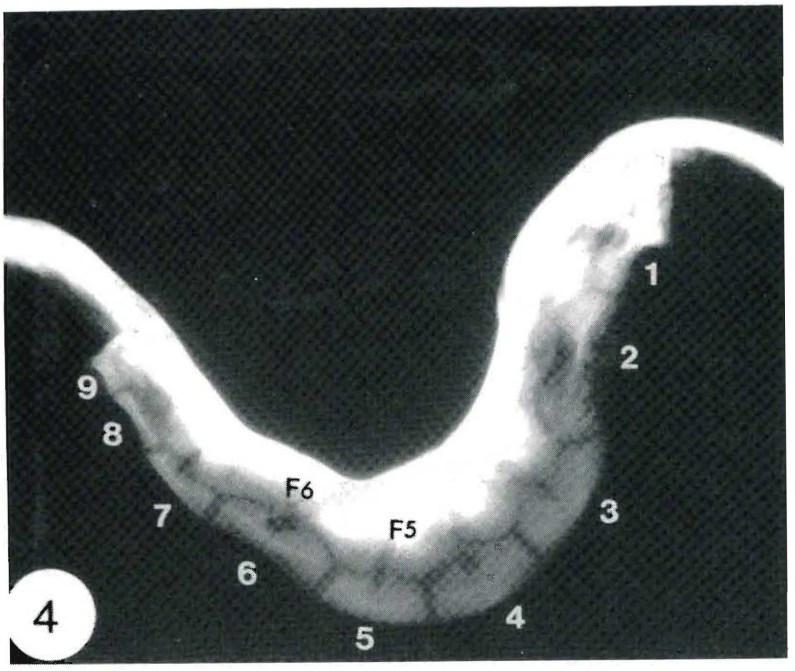


3B



3D





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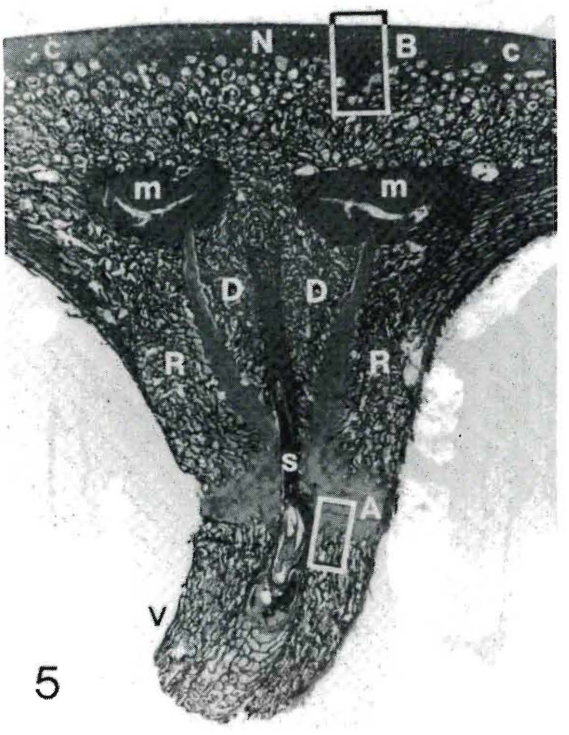

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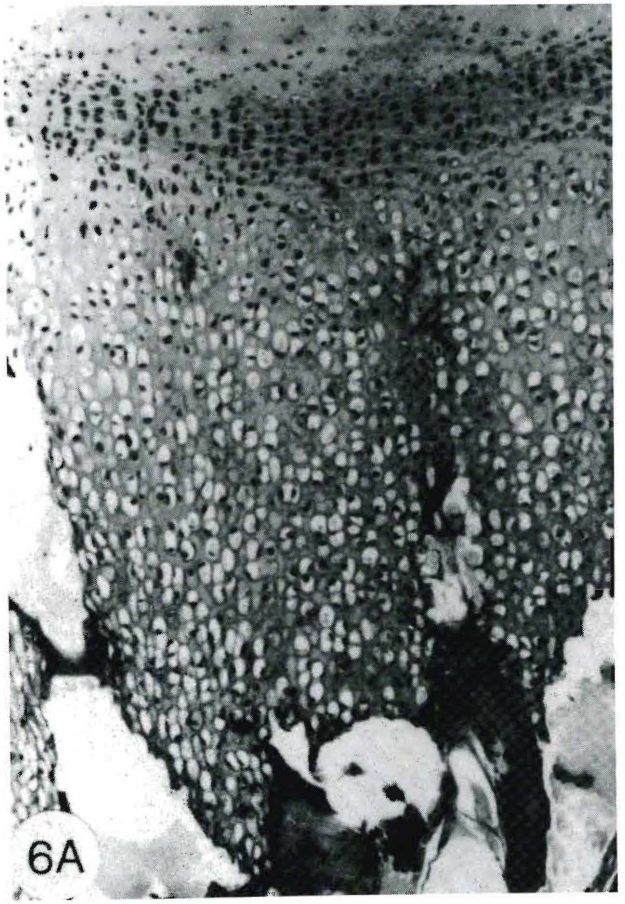
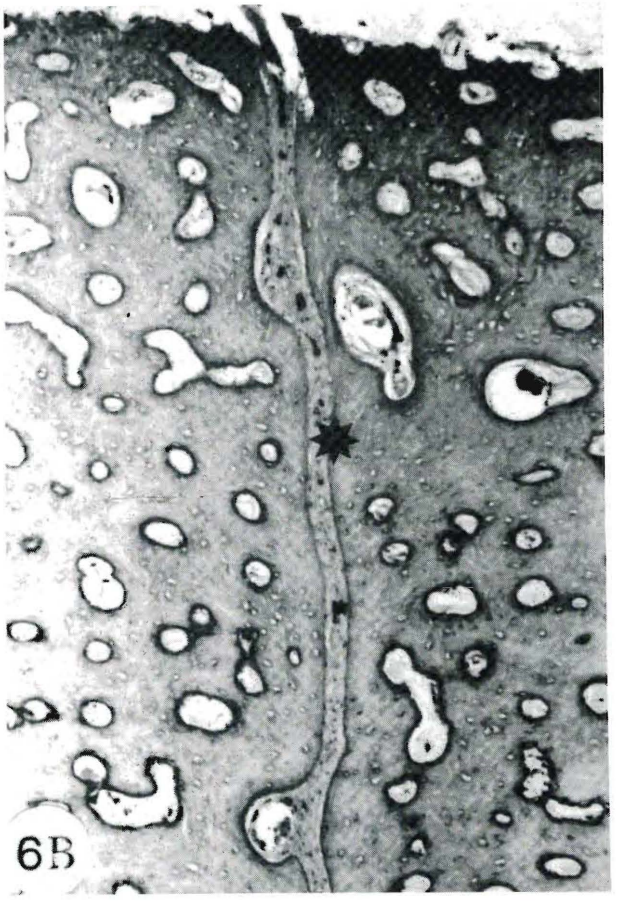


6A



6B

DISCUSSION

Serologic analysis failed to reveal any significant biochemical abnormalities in either the captive juvenile group in general or the deformed specimens in particular. Our results were relatively consistent with the findings of other authors (see DESSAUER, 1970; LUTZ and DUNBAR-COOPER, 1987). The high level of magnesium found in the wild nesting adult female group is in agreement with the findings of MAIZELS (1956) and LOCKWOOD (1961), who reported increased levels of total calcium and magnesium in reptiles during egg laying. SIMKISS (1962) and CLARK (1967) also found similar levels in nesting female turtles, where increased mobilization of calcium and magnesium from the skeleton into the blood made these ions more available for egg-shell production. The high phosphorus levels in the juvenile captive group is due to pituitary growth hormone which causes increased bone and tissue growth in young animals (DAVIDSOHN and HENRY, 1974). The captive juvenile group had serum calcium levels approximately equal to those of the wild female nesting group, and there were no significant differences in any of the analyte levels of the normal captive juveniles as compared to the deformed captive juveniles. Taking into account hormonal differences in the developmental stages of sea turtles, the serologic analysis is not suggestive of a nutritional disorder as being the underlying cause for the deformities. Even though it might be argued that the slightly lower calcium and magnesium and higher phosphorus levels in the captive group could be suggestive of some minimal nutritional deficiencies, the identical results when comparing normal with deformed captives essentially excludes nutritional factors as a direct cause of the deformities. In addition, histologic sections of deformed animals failed to reveal any evidence of nutritional fibrous osteodystrophy, while at the same time revealing an obvious focal inflammatory cause for the deformities.

The combination of radiographic, anatomic, and histologic analyses demonstrated unequivocally that the lordotic spinal deformities developed as a result of chronic focal inflammation of inter-neural and neural-costal synarthrodial joint sutures. This lead to synarthrodial fusion and inter-neural synostosis causing eventual lordosis as a result of continued normal ventral thoracic vertebral endochondral growth juxtaposed against pathologic dorsal neural bone tethering. In view of the high percentage (65.1%) of the captive specimens affected, it is hypothesized that the lesions are of an infectious microbial origin, representing a generalized zoonosis of the captive population occurring at about age 6 to 8 months. Both local Floridian species appear to have been equally susceptible to the presumed zoonosis, with 87.8% of the C. mydas and 87.2% of the C. caretta involved, whereas the exotic Costa Rican species L. olivacea with only 28.8% involved may have been more resistant. Despite some suggestive evidence on one histologic section (see description above) that the original lesion may have been a carapacial or sulcus infection with eventual spread into the neural synarthroses, there was no obvious evidence of carapacial infection of the living animals at or before the time that the deformities began to appear. However, most of the animals had fairly heavy carapacial algal growth, so scutes and sulci were not readily visible for inspection. It is possible that a microbial infection had developed in scute sulci and was simply not noticed because of the heavy overlying algal growth. The fact that scute patterns were normal at hatching and became abnormal with increasing spinal deformity (see Fig. 2) suggests that there was indeed infection of scute sulci and areas underneath the scutes. It is presumed that the scute sulcus infection then spread into the underlying epidermis and dermis and entered the inter-neural and neural-costal synarthrodial joint sutures, where the infection triggered a reactive proliferative fibroblastic response, which then eventually led to synarthrodial fusion and gradual lordotic spinal deformity.

Infection as a cause for spinal deformity has not previously been recorded for turtles. We find no evidence in the literature of documented cases of infectious-based spinal deformities in any reptiles, though FRYE and CARNEY (1974) and KIEL (1977) have described non-infectious osteitis deformans (Paget's disease) of snakes causing spinal deformity. In general, bone and joint infections have only rarely been documented in reptiles. ACKERMAN et al. (1971) described infectious arthritis in a teju lizard (Tupinambis teguixin), and OGDEN et al. (1981) documented septic arthritis with osteomyelitis in a leatherback turtle (Dermochelys coriacea). WALLACH (1975) and FRYE (1981) have described ulcerative shell disease in freshwater turtles. This condition is due to Beneckea chitinovora, a Gram negative bacillus which causes scute and bone ulcerations that give the carapace a pock-marked appearance. HUNT (1958) has reported on mycotic disease occurring in carapacial sulci and beneath scutes of Emys orbicularis causing shell rot and bone necrosis secondary to pathologic algal growth.

Though unrecorded among reptiles, the development of spinal deformity secondary to infection is well known in humans, where tuberculous spondylitis has in the past been a common cause of progressive kyphotic spinal deformity (LA ROCCA, 1982). Progressive lordotic deformity as described for turtles in this paper also occurs rarely in humans, but is usually caused by congenital failure of segmentation of the dorsal vertebral spinal elements combined with continued endochondral growth of ventral vertebral body elements (BRADFORD et al., 1982), a mechanism of "tethering" nearly identical to that described in this paper, though differing in etiology.

We are unable to document what type of agent may have been responsible for the infection causing the deformities in our study population. Unfortunately, tissue cultures were not obtained prior to death, and acid-fast or other organism-specific stains were not utilized. However, the absence of visible bacte-

ria on the sections does not exclude bacterial infection as a cause, since most cases of subacute or chronic bone and joint infections are not accompanied by visible bacteria or areas of purulence (OGDEN et al., 1981). We hypothesize that the infection was bacterial in origin, though viral or fungal agents cannot be excluded. Among bacteria, Gram negative organisms such as Beneckea often cause shell disease (WALLACH, 1975; MARCUS, 1980). Also commonly causing skin lesions (and potentially shell lesions) in turtles are the various species of Mycobacterium (RHODIN and ANVER, 1977). In addition, viral agents such as herpes and mixed bacterial flora commonly cause focal ulcerative dermatitis (gray-patch disease) and potentially shell disease, as described in captive-raised sea turtles by REBELL et al. (1975), GLAZEBROOK (1980), JACOBSON (1980a), and WILES and RAND (1987). Fungal infections can also cause skin and shell disease in turtles (HUNT, 1958; JACOBSON, 1980b).

Despite the fact that lordosis was extremely common in our population of captive-raised sea turtles, this condition has only been noted once previously in a captive population (WITHAM and FUTCH, 1977). These authors also noted that many of their young growing animals also developed focal skin necrosis (herpes-related gray-patch disease). In view of our own studies, it is possible that their lordotic deformities were a result of these necrotic lesions. Other major studies on disease problems in captive-raised sea turtles have not recorded spinal deformities of any kind (GLAZEBROOK, 1980; CLARY and LEONG, 1984; RAJAGOPALAN et al., 1984).

The histopathologic mechanism of infection causing synarthrodial joint fusion and neural tethering leading to spinal lordosis in growing turtles is a fascinating and previously unrecorded phenomenon. Unfortunately, we were unable to identify a causative agent. If this condition arises again in other captive-raised population of turtles, then careful attention should be paid to thorough culturing of both blood and carapace elements, as well as careful histologic examination with a wider spectrum of staining techniques, so that identification of an etiologic agent can be firmly established.

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