SPONDYLITIC CHANGES IN LONG-FINNED PILOT WHALES (GLOBICEPHALA MELAS) STRANDED ON CAPE COD, MASSACHUSETTS, USA, BETWEEN 1982 AND 2000

Authors: Sweeny, Melinda M., Price, Janet M., Jones, Gwilym S., French, Thomas W., Early, Greg A., et al.

Source: Journal of Wildlife Diseases, 41(4) : 717-727

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-41.4.717

Journal of Wildlife Diseases, 41(4), 2005, pp. 717–727

SPONDYLITIC CHANGES IN LONG-FINNED PILOT WHALES (*GLOBICEPHALA MELAS*) STRANDED ON CAPE COD, MASSACHUSETTS, USA, BETWEEN 1982 AND 2000

Melinda M. Sweeny,[1] Janet M. Price,[2] Gwilym S. Jones,[3] Thomas W. French,[4] Greg A. Early,[1] and Michael J. Moore[1,5]

[1] Woods Hole Oceanographic Institution, Woods Hole, Massachusetts 02543, USA
[2] Sepracor, Inc., 84 Waterford Dr., Marlborough, Massachusetts 01752, USA
[3] Northeastern University, Biology Department, 650 Huntington Ave., Boston, Massachusetts 02115, USA
[4] Division of Fisheries and Wildlife, 1 Rabbit Hill Rd., Westborough, Massachusetts 01581, USA
[5] Corresponding author (email: mmoorewhoi.edu)

ABSTRACT: The primary bone pathology diagnoses recognized in cetacea are osteomyelitis and spondylosis deformans. In this study, we determined the prevalence, type, and severity of vertebral pathology in 52 pilot whales, a mass stranding species that stranded on Cape Cod, Massachusetts, between 1982 and 2000. Eleven whales (21%) had hyperostosis and ossification of tendon insertion points on and between vertebrae, chevron bones, and costovertebral joints, with multiple fused blocks of vertebrae. These lesions are typical of a group of interrelated diseases described in humans as spondyloarthropathies, specifically ankylosing spondylitis, which has not been fully described in cetacea. In severe cases, ankylosing spondylitis in humans can inhibit mobility. If the lesions described here negatively affect the overall health of the whale, these lesions may be a contributing factor in stranding of this highly sociable species.

Key words: Ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis, *Globicephala melas*, long-finned pilot whale, stranded, vertebral pathology.

INTRODUCTION

Pilot whales, *Globicephala melas*, are highly social, traveling in pods of related members (Amos et al., 1993), and they strand both singly and en masse (Robson, 1984). A single whale may strand as a result of disease, starvation, or abandonment (Geraci and Aubin, 1977). Mass strandings of pilot whales have been attributed to a number of causes, including navigational error, such as disturbance to the echolocation system or coastal geomagnetic characteristics (Bernard and Reilly, 1999), or unfamiliarity with an area while feeding or migrating in or near shore (Dawson et al., 1985). Mass strandings have also been thought to be initiated by a sick (Duignan et al., 1995) or injured individual of a pelagic and tightly social species such as the pilot whale (Robson, 1984). Such a sick individual could subsequently induce other group members, healthy or sick, to come ashore (McLeod, 1986). Mass strandings of pilot whales occur on Cape Cod, Massachusetts (McFee, 1990; Wiley et al., 2001), among other places. Between 1982 and 2000 there were 14 mass stranding events and 56 single strandings in the New England region. All 14 mass strandings occurred on Cape Cod, with 50% (7/14) on Wellfleet beaches, 36% (5/14) on Eastham beaches, and 14% (2/14) in Barnstable. Of the single strandings 16% (9/56) occurred in Wellfleet, 9% (5/56) in Eastham, 11% (6/56) in Truro, 5% (3/56) in Provincetown, 28% (10/56) in various other locations in Massachusetts, and 41% (23/56) in other locations, including Maine and Rhode Island (Rubenstein, pers. comm.). Most single strandings resulted from whales that had previously died at sea and washed ashore dead.

There has been no systematic review of the vertebral pathology of specimens of mass-stranded individuals in Massachusetts. It is unclear if specific pathology may underlie some of these mass stranding events. Full necropsy with histopathology is often impractical because of the logistic constraints associated with mass strandings. The goal of this study was to determine if vertebral pathology was present and, if present, whether it severe enough

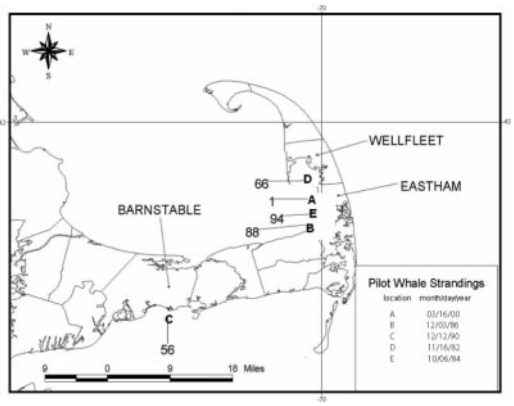

FIGURE 1. Map of pilot whale stranding locations, Cape Cod, Massachusetts, USA, and number of stranded animals. (A) Campground Beach, Eastham; (B) Boatmeadow Creek, Eastham; (C) Squaw Island, Hyannisport; (D) Lieutenant Island, Wellfleet; (E) Eastham.

to compromise health and possibly induce a stranding event. In this study, cleaned pilot whale spinal columns were examined to assess the nature and degree of vertebral lesions.

MATERIALS AND METHODS

Vertebrae

Four hundred seventy pilot whales stranded on Cape Cod from 1982 to 2000 (Rubenstein, pers. comm.). Bones from 52 animals (four mass stranding events, one single whale washed ashore dead) were collected on beaches and marshes of the eastern shore of Cape Cod Bay, Massachusetts, USA, from 1982 to 2000 (Fig. 1). The time interval between stranding and collection ranged from the time of stranding to as many as 6 yr. Bones were assigned to specific individuals on the basis of contiguity at the time of collection (Table 1). All bones were archived either at Northeastern University in Boston, Massachusetts (n=51), or at the University of Arkansas (n=1) and were identified by museum numbers (and field numbers, when available). When collected, most bones had already been cleaned by natural decomposition. Bones (collected before 1990) with dried tissue remains were boiled. After 1990, horse manure (followed by washing and drying) was used to remove the remaining tissue.

Examination

Mature animals were recognized by the presence of fused epiphyseal plates, while those with incomplete fusion or unfused plates were considered immature (Kompanje, 1995a). The vertebral formula for *G. melas* has been reported as cervical (7), thoracic (11), lumbar (12–24), and caudal (28–29) (Tomilin, 1967). For this examination, the vertebral regions were determined as follows: cervical vertebrae were highly compressed and naturally fused either C1-5, C1-6, or C1-7; thoracic vertebrae have articular facets for the ribs; lumbar vertebrae do not have articular facets for ribs or chevron bones; and the first 17 caudal vertebrae have articular facets for chevron bones, while the remainder are significantly reduced in size. Sex was determined either at the time of stranding or by morphometrics of the vertebrae (Van Beusichem, 2000).

The gross examination of each vertebra included an assessment of smoothness and regularity of surfaces, bilateral symmetry, anomalies, bone growths, fusion, fractures, or abnormal fistulae (Fig. 2). Any lesions found were ranked as follows: minor (those involving less than 10% of total vertebral surface area), moderate (involving 11% to 50% of vertebral surface area), and severe (covering greater than 50% of the vertebral surface area with or without fusion of two or more vertebrae).

For each animal, the following information was recorded: museum identification number, spinal region, presence/absence of pathology, measurement of pathology, sex (when available), epiphyseal fusion (maturity), and date and location of stranding. Digital photographs were taken (dorsal, ventral, right lateral, and left lateral views) of each bone with pathology and of a subsample of normal bones. A subsample of normal and diseased bones were radiographed as well as scanned with computed tomography (Siemens Somatom Emotion, Malvern, Pennsylvania, USA). The contrast and pixel grayscale levels of scanned images were adjusted to maximize the different structural features.

RESULTS

The number of vertebrae examined per animal varied from 1 to 68; the maximum number expected was 71 (Tomilin, 1967). Gross examination revealed osseous changes in 11 of 52 (21%) individuals examined (estimated prevalence is 21±5.6%) (Table 1). Osseous changes ranged from development of minor osteophytes to severe ankylosis of vertebral bodies; three had severe pathology (Table 1). In the 11 animals with lesions, two of three atlas/axis

TABLE 1. Samples examined from pilot whales stranded on Cape Cod, Massachusetts, USA.

Museum ID No.	Total vertebrae	Pathology (yes/no [y/n]	Sex[a]	Epiphyseal fusion (y/n)
Mass stranded animals				
Lieutenant Island, Wellfleet—16 November 1982 (n=66); 41°53′N, 70°01′W				
NUVC 2151	11	n	f	y
2157	3	y	f	y
2174	2	n	u	n
2173	1	n	f	y
2178	1	n	u	n
2176	1	n	u	n
2175	1	n	u	n
2172	5	n	f	y
2171	3	n	f	y
2179	1	n	f	y
2188	1	n	u	n
2189	1	n	u	n
2186	1	n	u	n
2187	1	n	u	n
2190	1	n	u	n
2160	1 (At/Ax)a	n	u	u
2152	36	y	f	y
2153	36	n	f	y
2116	31	y	f	y
2115	30	y	m	y
2133	34	n	u	n
2131	30	n	u	n
2124	1	n	u	n
2123	1	n	u	n
2128	1	n	u	n
2127	1	n	f	y
2126	1	n	u	n
2125	1	n	u	n
2129	1	n	u	n
2136	1	n	u	u
2143	15	n	f	y
2130	6	n	f	y
2117	11	y	f	y
1771	8	n	u	n
1772	6	n	u	u
1897	12	n	u	n
2344	1	y	f	y
Mass Stranded Animals				
Eastham-6 October, 1984 (n=94) 41° 49′ N, 70° 00′ W				
NUVC 2132	25	n	f	y
2119	13	n	u	n
2121	16	n	m	n
2118	11	n	f	y
2197	39	y	f	y
2198	31	n	f	y
2120	19	n	m	y
Mass stranded animals				
Boatmeadow Creek, Eastham—3 December 1986 (n=88); 41°48′N, 70°00′W				
NUVC 2204	58	n	f	n
2345	47	y	m	y
2148	47	n	f	y
2149	43	n	f	y
2150	54	y	f	y
Uarkb	61 + At/Ax	y	f	y
Mass stranded animals				
Squaw Island, Hyannisport—12 December 1990 (n=56); 41°37′N, 70°19′W				
NUVC 2340	61	n	u	n
Single stranded animals				
Campground Beach, Eastham—16 March 2000 (n=1); 41°51′N, 70°00′W				
NUVC 4247	44 + At/Ax	y	f	y

[a] f = female; m = male; u = unknown; At/Ax = atlas/axis.
[b] Uark = University of Arkansas.

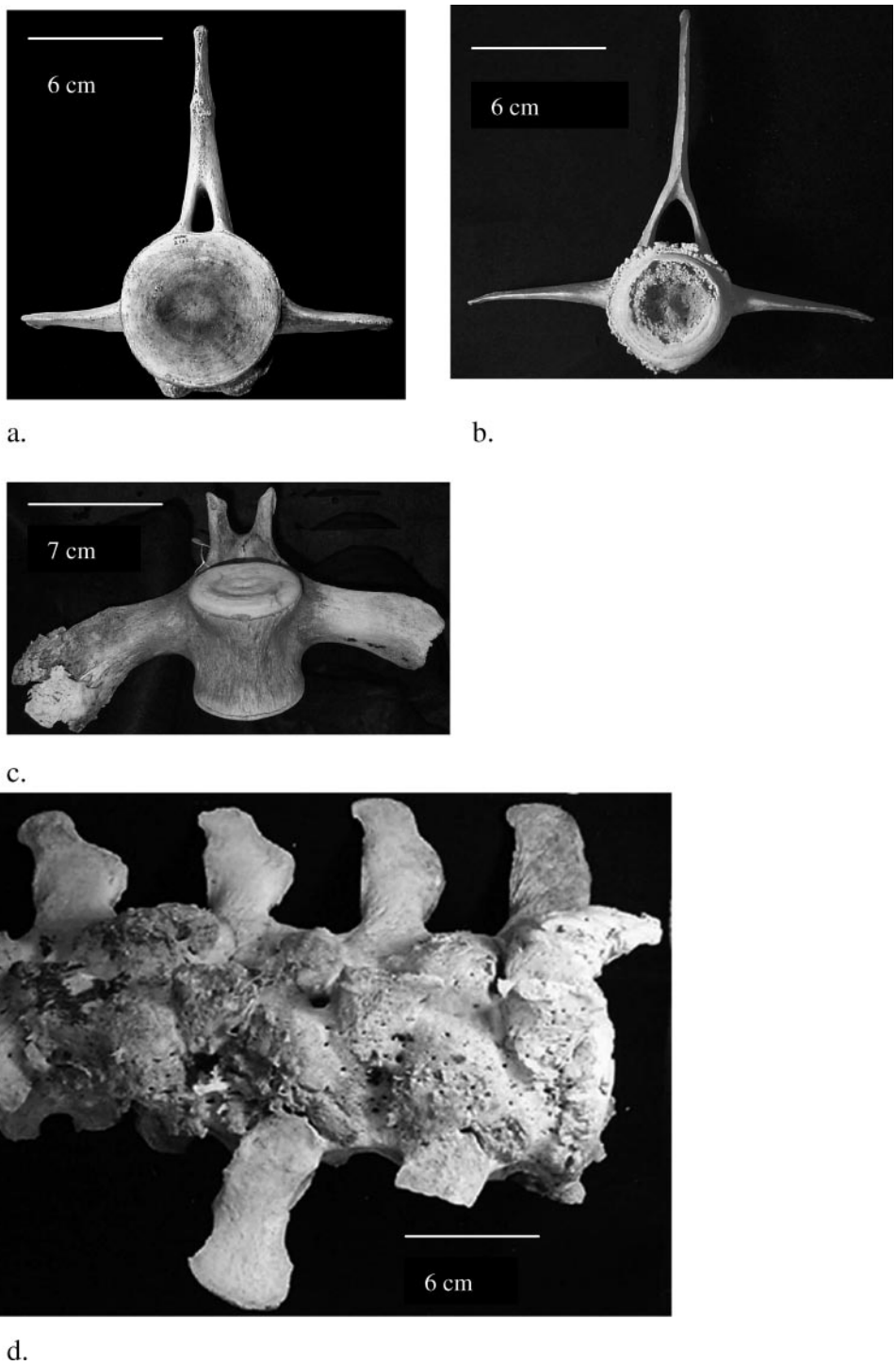

FIGURE 2. Gross morphology of pilot whale vertebrae. (a) Anterior view of normal pilot whale caudal vertebra (Ca6) (same sample as 3a and 4a) (NUVC 2157). (b) Posterior view of pilot whale lumbar vertebra (L11). Note subchondral cysts of vertebral body and periarticular syndesmophytes (same sample as 3c) (NUVC 4247). (c) Ventral view of pilot whale thoracic vertebra (T9). Note bony growth and erosion of distal transverse processes; more pronounced on the left. (NUVC 2345). (d) Right lateral view of four fused pilot whale caudal vertebrae (Ca9-12). Note syndesmophyte production and ankylosis (same sample as 4b, 4c, and 4d) (NUVC 4247).

bones, nine of the 45 (20%) cervical vertebrae complexes, 22 of the 77 (29%) thoracic vertebrae, 21 of the 103 (20%) lumbar vertebrae, 33 of the 120 (28%) caudal vertebrae, and one of one chevron bones had lesions. Pathology was present in both the naturally fused and unfused cervical vertebrae. Both NUVC 4247 (single stranding) and UArk (mass stranding), which had lesions in all regions, had cervical vertebrae that were fused normally but also exhibited abnormal bony growth and erosion.

The presence of skeletal abnormalities was not associated with any particular stranding location or date (Table 1). Three of the four mass stranding events (all of which occurred between October and December) had at least one whale with lesions. The only mass stranding without bone pathology was at Squaw Island, Hyannisport, Massachusetts, where only one of 56 stranded whales was examined, and this animal was a calf. Animals with lesions were from Lieutenant Island, Wellfleet (six of 38 whales examined, 16%); Eastham 1984 (one of six examined, 17%); Boat Meadow Creek, Eastham (three of six examined, 50%); and Campground Beach, Eastham (one single stranded whale). Three of six spatially related individuals from the 3 December 1986 stranding at Boat Meadow Creek had bone lesions.

Of the 427 animals that mass stranded on Cape Cod between 1982 and 2000, 234 (55%) were females, 95 (22%) were males, and 98 (23%) were of undetermined sex (Rubenstein, pers. comm.). The 52 whales sampled included 23 females (44%), four males (8%) and 25 animals of unknown sex (48%); 24 (46%) were immature, 25 (48%) were mature, and three (6%) were of undetermined age. Of the 11 animals with lesions, nine were females (82%), two were males (18%), and all were mature.

Some radiographs revealed a 'moth-eaten' appearance in the vertebral body, suggestive of osteoporosis (Fig. 3b, c), as well as peridiscal vacuolization (i.e., pitting; Fig. 3b–d). The computed tomography scans illustrated significant loss of intervertebral disk space and ossification (white areas) of the intervertebral disk in the fused caudal vertebrae of NUVC 4247 (Fig. 4c, d). Decreased central ossification in the vertebral body, also suggestive of osteoporosis (darker areas), was apparent in some specimens, along with peridiscal vacuolization (Fig. 4b, d). Bony excrescences appear to merge from the dorsal and ventral vertebral margins, while leaving radiolucent areas at the level of the intervertebral disk spaces.

DISCUSSION

Understanding the pathobiology of beached and beaching marine mammals is critical to their management. In the case of mass stranded odontocetes, understanding clinical observations is important for triage on the beach, possible treatment regimes, and in prognosis for recovery. In this study, vertebral lesions were found in at least 21% of pilot whales examined. This observation raises the following issues: 1) quality of the data, 2) differential diagnosis and pathogenesis of the lesion, 3) whether the lesions impair mobility, and 4) if the lesions initiate stranding behavior.

Data quality

At the time of collection, available vertebrae per carcass ranged in number from one to an entire set. These results are therefore a conservative estimate of both severity and prevalence of bone pathology, as we do not know the condition of the missing vertebrae and/or other animals in the stranding. Although collection of skeletal parts was undertaken systematically, some vertebrae may be from one individual, despite being represented by several different identification numbers, since bones are sometimes scattered by tidal action or scavengers. Such an overestimation in total number of individuals would also render the results conservative. If the total number of animals was overestimated, the actual prevalence of pathology would be

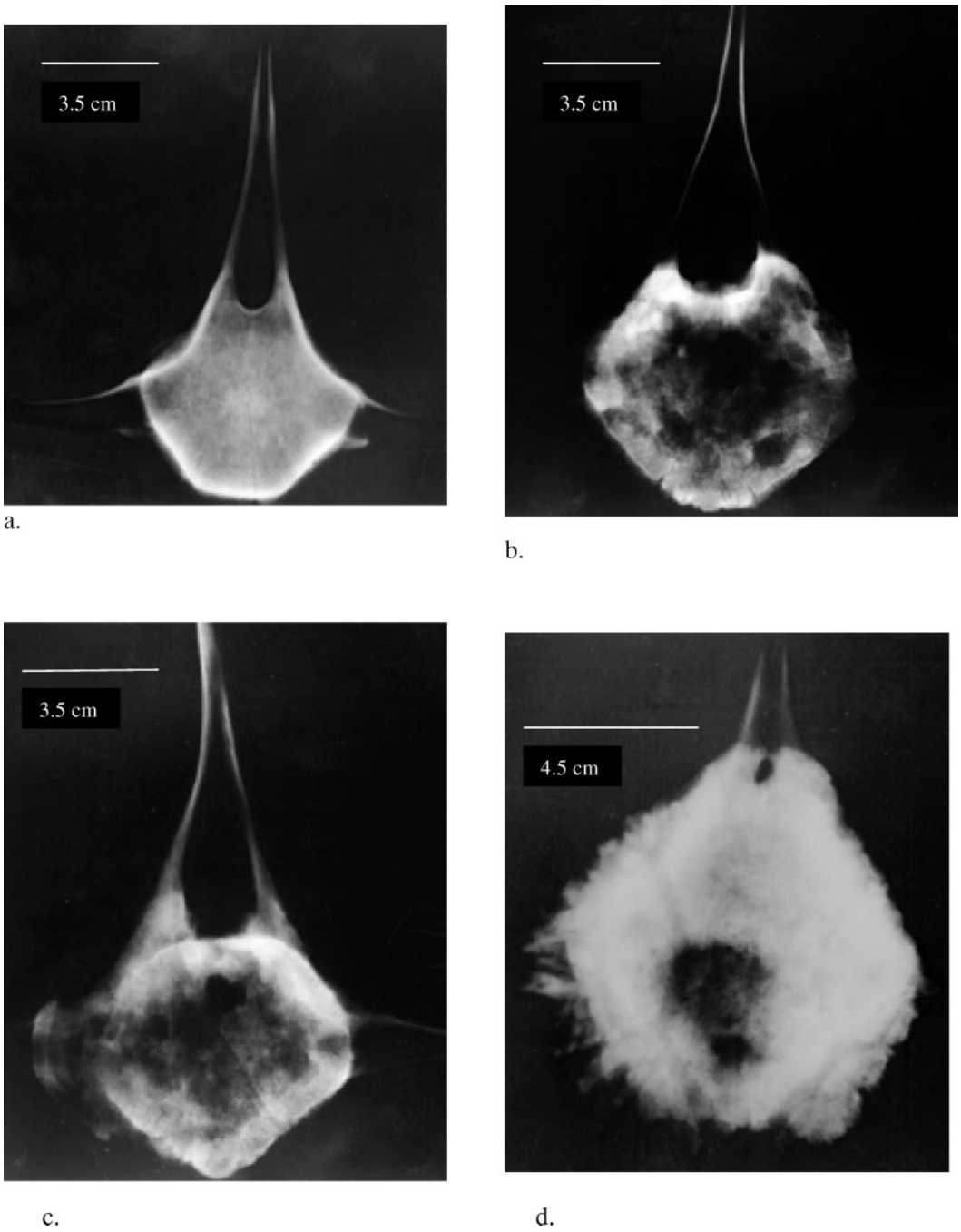

FIGURE 3. X-radiographs of pilot whale vertebrae. (a) Posterior view of normal pilot whale caudal vertebra (Ca6) (same sample as 2a and 4a) (NUVC 2157). (b) Anterior view of pilot whale lumbar vertebra (L12). Note 'moth-eaten' osteoporotic body (NUVC 4247). (c) Posterior view of pilot whale lumbar vertebra (L11) Note vacuolization, dark area in upper mid-body and osteoporotic body (same sample as 2b). (NUVC 4247). (d) Anterior view of pilot whale caudal vertebra (Ca15). Note 'shaggy' vertebral rim and narrowing of the neural canal. (NUVC 2344).

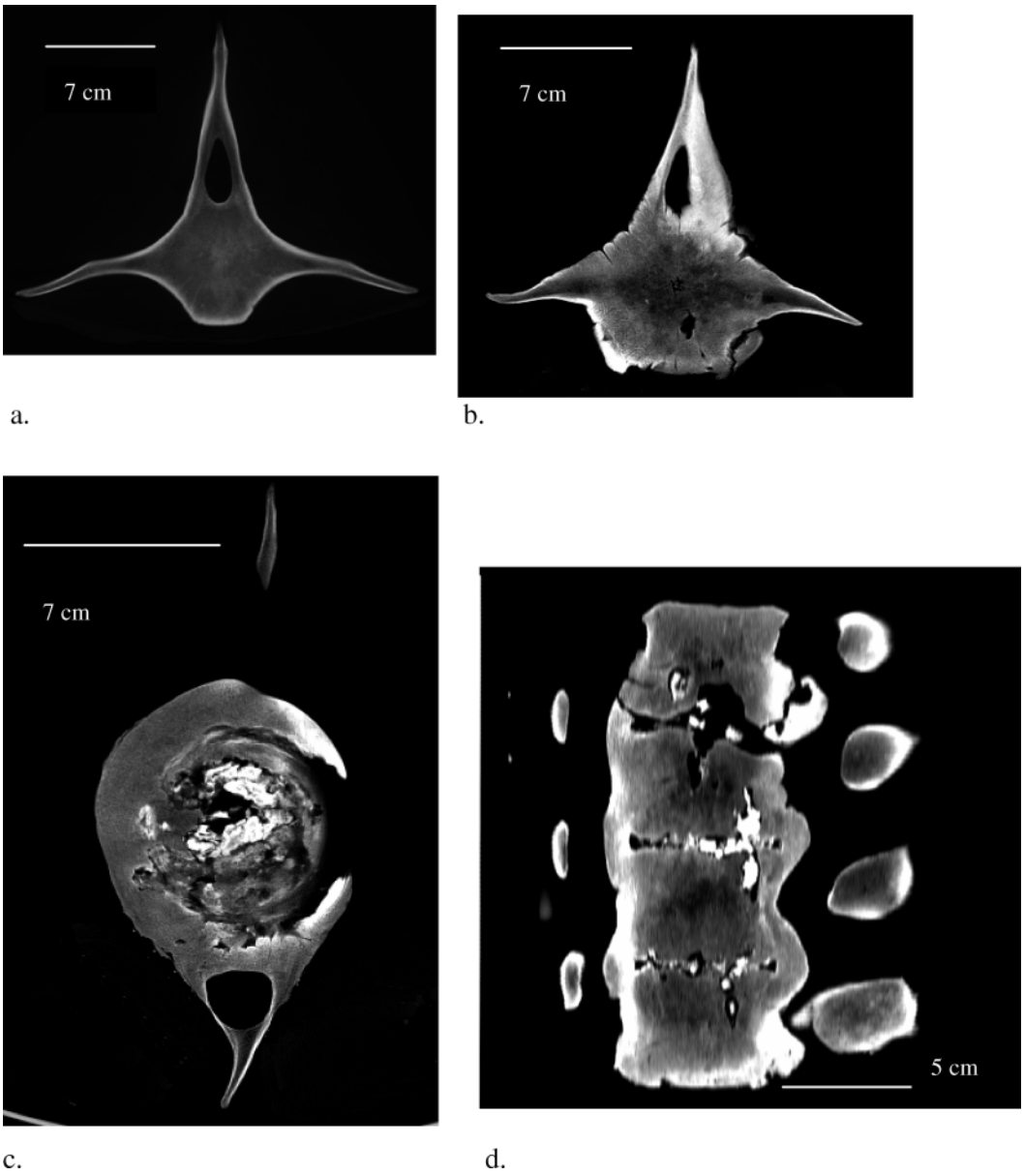

FIGURE 4. Computed tomographs of pilot whale vertebrae. (a) Anterior view of normal pilot whale caudal vertebra (Ca 6) (same sample as 2a and 3a) (NUVC 2157). (b) Anterior view of mid-body of one of four fused pilot whale caudal vertebra (Ca 9-12). Note hyperostosis of the spinal process, subchondral cysts, and osteoporosis (same sample as 2d, 4c, and 4d) (NUVC 4247). (c) Anterior view of intervertebral disk of one of four fused pilot whale caudal vertebra (Ca 9-12). Note syndesmophyte production and ossification of intervertebral disk (same sample as 2d, 4b, and 4d) (NUVC 4247). (d) Lateral view of four fused pilot whale caudal vertebrae (Ca 9-12). Note syndesmophyte bridging between vertebrae, ossified intervertebral discs, and subchondral cysts in the body (same sample as 2d, 4b, and 4c) (NUVC 4247).

higher. It is also possible that the total number of animals was underestimated, where multiple vertebrae were thought to represent different individuals but were actually from one animal. Spatial separation at the time of collection makes this unlikely.

Conservative or not, the data reveal that a large number of pilot whales stranded on Cape Cod had bone pathology. Pathology has also been reported in bones from pilot whales driven onto Newfoundland shores, the difference being that only one case of severe bone pathology (a block of four fused vertebrae) was found among hundreds of carcasses and bones in a "graveyard of flensed bones" (Cowan, 1966). Those pilot whale carcasses were left from a whale drive fishery, rather than from 'naturally' stranded pilot whales. Another source of nonstranded pilot whale carcasses is from the Faroe Islands, where a traditional drive fishery of pilot whales has been maintained since the archipelago was inhabited 1,000 yr ago (Bloch, 1998); no bone pathology has been reported from those pilot whales driven ashore (Bloch, pers. comm.). The stock structure of the North Atlantic long-finned pilot whale population is uncertain (Fullard et al., 2000), but Fullard et al. (2000) have proposed a stock structure that is correlated to sea surface temperature, including a cold-water population west of the Labrador/North Atlantic current and a warm-water population extending across the Atlantic in the Gulf Stream. Therefore, pilot whales from the Faroe Islands, Newfoundland, and those that stranded on Cape Cod may belong to the same population. Considering the number of lesions found in the present study, it is noteworthy that pathology has not been reported from nonstranding sources of potentially the same population.

Differential diagnosis

Although there are no other studies involving stranded, by-caught, or captive pilot whale bone pathology, there are studies on other stranded cetaceans with bony lesions, the diagnoses for which include spondylitis (Paterson, 1984), spondylosis deformans (Kompanje, 1995a), osteomyelitis (Kompanje, 1995b), and discospondylitis (Alexander et al., 1989). The differential diagnosis for the lesions described here includes osteomyelitis, spondylosis deformans, diffuse idiopathic skeletal hyperostosis (DISH), and ankylosing spondylitis (AS).

Vertebral osteomyelitis can have effusive bony growth and pitting, as seen in these cases; however, osteomyelitis does not usually span more than two vertebrae because of the resistive properties of intervertebral discs (Jubb et al., 1992). In this case, there are multiple animals with blocks of three and four fused vertebrae. In the case of spondylosis deformans, the costovertebral or apophyseal joints are not involved, and the intervertebral discs do not typically calcify (Resnick and Niwayama, 1988c). In this study, intervertebral disk calcification and costovertebral joint lesions are present. Therefore, both osteomyelitis and spondylosis deformans can be ruled out.

Diffuse idiopathic skeletal hyperostosis is characterized by flowing or undulating calcification and ossification along the ventrolateral aspect of at least four contiguous vertebral bodies, with relative preservation of intervertebral disk height in the involved vertebral segments. Additionally, there is an absence of apophyseal joint bony ankylosis (Resnick, 2002b). Lucent areas between the ossified ventral longitudinal ligament and ventral vertebral surfaces are also characteristic (Resnick, 2002b). Lesions in this study are present in apophyseal joints as well as intervertebral discs; therefore, DISH can also be ruled out.

The lesions found in this study have characteristics of AS. This disease belongs to a group of diseases in humans known as spondyloarthropathies; this group of diseases involves inflammation and eventual ankylosis of the sacroiliac joints (absent in

cetacean), costovertebral joint changes, calcification of intervertebral discs, and osteoporosis (Resnick and Niwayama, 2002a). Ankylosing spondylitis is described as a chronic, nonpurulent inflammatory disease of the axial joints and both sacroiliac joints in quadrupeds (Wollheim, 1993).

In humans, AS characteristically shows ossification within the annulus fibrosus in the form of syndesmophytes. This begins with cellular infiltration of plasma cells and lymphocytes, followed by fibrin accumulation and swelling in the outer layer of the annulus fibrosus; chondrocytes then form at the junction of the intervertebral disk and vertebral edge and, in the later stages of disease, these chondrocytes undergo calcification, with eventual bridging to neighboring vertebral joints (Resnick and Niwayama, 2002a). Joints affected by AS include the discovertebral junctions, costovertebral joints, and apophyseal joints, with potential fusion throughout the vertebral column (Resnick and Niwayama, 2002a) caused by inflammation not only in the joints but also in the entheses (i.e., ligament and tendon insertion points; Wollheim, 1993). As entheses become inflamed (enthesitis), they can eventually ossify, producing syndesmophytes, potentially forming a "bony" bridge between vertebrae (Resnick and Niwayama, 1988a); periostitis at the entheses is also observed. Radiographically, lesions can result in irregular or "shaggy" periarticular surfaces (Fig. 3d; Resnick and Niwayama, 1988a) with thin, flowing syndesmophytes extending from one vertebral body to the next, with surrounding eburnation (i.e., thickening) and excessive discal calcification (Fig. 4c). While the bone surface has produced extra bone, internally there is a loss of trabecular density (i.e., osteoporosity; Figs. 3b, 4b) within the vertebral body (Khan, 1993b).

At the onset of AS, clinical signs in afflicted humans include anorexia, weight loss, and a low-grade fever. As the disease progresses, 25–30% of humans develop anterior uveitis, inflammation of the ascending aorta and aortic valve, fibrosis and cavitation in the upper lobes and pleuritis of the lungs, and amyloid involvement of the kidneys (Resnick and Niwayama, 2002a). In humans, AS is caused by a genetically determined immune response to environmental factors (Resnick and Niwayama, 2002a). It has been suggested that some gram-negative bacteria, such as *Klebsiella* sp., may play a role in triggering ankylosing spondylitis, but the supporting evidence is controversial (Khan, 1993a). Examination of extraskeletal tissues was not possible in this study.

In this study of pilot whales, lesions are present in both costovertebral and discovertebral joints; in addition, there is calcification of intervertebral joints, osseous erosions, osteoporosis, and bony lesions that cross more than one intervertebral space, all features that are consistent with AS.

Effects on mobility

In humans, AS can cause pain and stiffness as well as debilitation (Resnick and Niwayama, 2002a). Assuming spinal flexibility has importance to other mammals, including pilot whales, the effects on mobility could be more significant. The principal locomotory organ of whales is the body axis (i.e., the vertebral column, spinal muscles of the back, and tail flukes) (Slijper, 1961). Cetaceans produce thrust by vertical movements of the tail and flukes (as opposed to the lateral strokes of fish) (Arkowitz and Rommel, 1985). An ankylosed spine not only causes a decrease in spinal mobility, but it also creates vulnerability as a result of a rigid vertebral column that cannot bend or rotate on impact to absorb traumatic stress (Resnick, 2002a). In the case of AS, the rigidity of the spine in fused sections also causes more vulnerability to fracture and osteoporosis (both present in this study) in other sections of the vertebral column (Palmer, 1990). The complication of likely spinal pain and fusion is serious for any organism

that must swim without an opportunity to rest to allow bone pathology to heal, in contrast to terrestrial mammals. Such pain is likely, even though pilot whales are essentially weightless in water, as the rib and chevron bones are force-bearing analogues acting as compression members on the vertebrae while the animal flexes and extends the vertebral column to swim.

Consequences of mass stranding

Bone pathologies have been documented in single stranded cetaceans (Kompanje, 1995a). Thus, a pilot whale with a severely compromised vertebral column may be likely to strand. However, would such an animal's beaching lead the healthy, related members inshore? Mass strandings of pilot whales have involved morbillivirus infection (Duignan et al., 1995), parasitic nerve damage (Morimitsu et al., 1987), and physical, clinical, and histologic evidence of illness (Walsh et al., 1991). No studies to date have investigated chronic, noninfectious diseases in mass strandings. This study reveals that over the past 18 years, 88 vertebrae from 11 animals and five separate pilot whale stranding events showed bone disease suggestive of AS that may have affected the whales' mobility.

There is a need for further investigation of the skeletal and extraskeletal features of ankylosing spondylitis and other osteologic pathology in pilot whales and other species of whales and dolphins. Histopathologic examination of periarticular and extraskeletal tissue for inflammatory changes, immunopathologic histochemistry, and more extensive epidemiologic studies are all needed to better understand the role of the observed changes in the mass stranding events commonly observed in this species.

ACKNOWLEDGMENTS

Thanks are due to Michael Scherer and Marine Research, Inc., to William Henry and Buzzards Bay Animal Hospital, and to Darlene Ketten, Scott Kramer, and Julie Aruda. We also thank Nancy McCartney at the University of Arkansas (Accession number 88-52-1). This study was funded in part by NOAA Fisheries Grant NA03NMF4390046. Conclusions and recommendations are those of the authors and do not necessarily reflect the views of the NOAA. Samples were collected under National Marine Fisheries Service authorization to the New England Aquarium. This paper represents Woods Hole Oceanographic Institution contribution number 11299.

LITERATURE CITED

ALEXANDER, J. W., M. A. SOLANGI, AND L. S. RIEGEL. 1989. Vertebral osteomyelitis and suspected diskospondylitis in an Atlantic bottlenose dolphin (*Tursiops truncates*). Journal of Wildlife Diseases 25: 118–121.

AMOS, B., C. SCHLOTTERER, AND D. TAUTZ. 1993. Social structure of pilot whales revealed by analytical DNA profiling. Science 260: 670–672.

ARKOWITZ, R., AND S. ROMMEL. 1985. Force and bending moment of the caudal muscles in the shortfin pilot whale. Marine Mammal Science 1: 203–209.

BERNARD, H. J., AND S. REILLY. 1999. Pilot whales *Globicephala* Lesson, 1828. *In* Handbook of marine mammals, S. Ridgway and R. Harrison (eds.). Academic Press, Boston, Massachusetts, pp. 245–279.

BLOCH, D. 1998. A review of marine mammals observed, caught, or stranded over the last two centuries in Faroese Waters. Shetland Sea Mammal Report 1997: 15–37.

COWAN, D. 1966. Pathology of the pilot whale (*Globicephala melaena*). Archives of Pathology 82: 178–189.

DAWSON, S. M., S. WHITEHOUSE, AND M. WILLISCROFT. 1985. A mass stranding of pilot whales in Tryphena Harbour, Great Barrier Island. Investigations of Cetacea 17: 165–173.

DUIGNAN, P. J., C. HOUSE, J. R. GERACI, G. EARLY, H. COPLAND, M. WALSH, G. BOSSART, C. CRAY, S. SADOVE, D. S. AUBIN, AND M. MOORE. 1995. Morbillivirus infection in two species of pilot whales (*Globicephala* sp.) from the western Atlantic. Marine Mammal Science 11: 150–162.

FULLARD, K. J., G. EARLY, M. P. HEIDE-JORGENSEN, D. BLOCH, A. ROSING-ASVID, AND W. AMOS. 2000. Population structure of long-finned pilot whales in the North Atlantic: A correlation with sea surface temperature? Molecular Ecology 9: 949–958.

GERACI, J., AND D. J. S. AUBIN. 1977. Mass stranding of the long-finned pilot whale, *Globicephala melaena*, on Sable Island, Nova Scotia. Journal of Fisheries Research Board Canada 34: 2196–2199.

JUBB, K. V. F., P. C. KENNEDY, AND N. PALMER. 1992. Bones and joints.*In* Pathology of domestic animals, K. V. F. Jubb, P. Kennedy, and N. Palm-

er (eds.). Academic Press, Boston, Massachusetts, pp. 100–125.

KHAN, M. A. 1993a. Pathogenesis of ankylosing spondylitis: Recent advances. Journal of Rheumatology 20: 1273–1277.

———. 1993b. Seronegative spondyloarthropathies. *In* Primer on the rheumatic diseases, H. Schumacher, J. Klippel, and W. Koopman (eds.). Arthritis Foundation, Atlanta, Georgia, pp. 189–193.

KOMPANJE, E. J. O. 1995a. Differences between spondylo-osteomyelitis and spondylosis deformans in small odontocetes based on museum material. Aquatic Mammals 21: 199–203.

———. 1995b. On the occurrence of spondylosis deformans in white-beaked dolphins *Lagenorhynchus albirostris* (Gray, 1846) stranded on the Dutch coast. Zooligische Mededekingen Leiden 69: 231–250.

MCFEE, W. E. 1990. An analysis of mass stranding of the long-finned pilot whale, *Globicephala melaena,* on Cape Cod. Master's Thesis, Northeastern University, Boston, Massachusetts, 90 pp.

MCLEOD, P. J. 1986. Observations during the stranding of one individual from a pod of pilot whales, *Globicephala melaena,* in Newfoundland. Canadian Field Naturalist 100: 137–139.

MORIMITSU, T., T. NAGAI, M. IDE, H. KAWANO, A. NAICHUU, M. KOONO, AND A. ISHII. 1987. Mass stranding of Odontoceti caused by parasitogenic eighth neuropathy. Journal of Wildlife Diseases 23: 586–590.

PALMER, R. W. 1990. Bone loss as well as bone formation is a feature of progressive ankylosing spondylitis. British Journal of Rheumatology 29: 498–499.

PATERSON, R. A. 1984. Spondylitis deformans in a bryde's whale (*Balaenoptera edeni* Anderson) stranded on the southern coast of Queensland. Journal of Wildlife Diseases 20: 250–252.

RESNICK, D. 2002a. Ankylosing spondylitis. *In* Diagnosis of bone and joint disorders, D. Resnick (ed.). W. B. Saunders Co., Philadelphia, Pennsylvania, pp. 1023–1081.

———. 2002b. Diffuse Idiopathic skeletal hyperostosis. *In* Diagnosis of bone and joint disorders, D. Resnick (ed.). W. B. Saunders Co., Philadelphia, Pennsylvania, pp. 1476–1504.

———, AND G. NIWAYAMA. 1988c. Degenerative diseases of the spine. *In* Diagnosis of bone and joint disorders: Articular diseases, D. Manke (ed.). W. B. Saunders Co., Philadelphia, Pennsylvania, pp. 1480–1561.

ROBSON, F. D. 1984. Strandings: Ways to save whales. The Science Press, Johannesburg.

SLIJPER, E. J. 1961. Locomotion and locomotory organs in whales and dolphins (Cetacea). Symposium of the Zoological Society London 5: 77–94.

TOMILIN, A. G. 1967. Cetacea. Israel Program for Scientific Translations, Jerusalem.

VAN BEUSICHEM, K. 2000. Skeletal development in the long-finned pilot whale, *Globicephala melas*. Master's Thesis, Northeastern University, Boston, Massachusetts, 103 pp.

WALSH, M., D. O. BEUSSE, W. G. YOUNG, J. D. LYNCH, E. D. ASPER, AND D. K. ODELL. 1991. Medical findings in a mass stranding of pilot whales (*Globicephala macrorhynchus*) in Florida. NOAA Technical Report NMFS 98, Miami, Florida.

WILEY, D. N., G. EARLY, C. A. MAYO, AND M. J. MOORE. 2001. Rescue and release of mass stranded cetaceans from beaches on Cape Cod, Massachusetts, USA, 1990–1999: A review of some response actions. Aquatic Mammals 27: 162–171.

WOLLHEIM, F. A. 1993. Spondyloarthropathies: Ankylosing spondylitis. *In* Textbook of rheumatology, H. Kelley, S. Ruddy, and C. B. Sledge (eds.). W. B. Saunders Co., Philadelphia, Pennsylvania, pp. 943–960.

Received for publication 2 July 2004.