

Short Note

Necropsy Report of a Fin Whale (*Balaenoptera physalus*) Stranded in Denmark in 2010

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There is little detailed information on stranded fin whales (*Balaenoptera physalus*) in the scientific literature (Notarbartolo di Sciara et al., 2003). In Denmark, at least eight fin whales stranded between the years 1603 and 1958 (Kinze, 1995). On 16 June 2010, a live subadult or adult male fin whale stranded in the Bay of Vejle (55° 69' N, 9° 58' E), Denmark. Despite several attempts, it was not possible to rescue the fin whale, which was only partially exposed by the water. The fin whale succumbed after 5 d stranded in shallow water. The dead fin whale was transported to a nearby pier, and a necropsy was performed by a multidisciplinary team, including biologists, conservationists, physicians, and veterinarians representing three universities and several museums. The fin whale was 17 m long and weighed 26 tons (measured on a truck scale), which suggested that the fin whale was emaciated. The age of the fin whale could not be determined as no ear plugs were sampled from the whale. However, based on its length, the fin whale was estimated to be approximately 5 y old (Aguilar & Lockyer, 1987). Externally, the fin whale had superficial cutaneous excoriations and small ulcerations, most likely related to physical trauma associated with the 5-d stranding period in shallow water. The fin whale was emaciated with scant subcutaneous and visceral fat deposits. The stomach was empty, and a mild inflammation, characterized by multifocal areas with redness and swelling, was present in the stomach wall. Only a small amount of brown fluid was present in the duodenum, jejunum, and ileum. The liver and spleen were enlarged and congested. In the wall of the urinary bladder, there

were several multifocal areas with moderate intramural hemorrhage. The lungs were atelectatic, and white to reddish frothy fluid exuded from cut surfaces. The bones were composted with elephant dung and macerated at 70° C in water with sodium carbonate and hydrogen-peroxide, then examined. For histology, tissue specimens were collected from the fused vertebrae and fixed in 10% neutral buffered formalin. Specimens were decalcified in 17% formic acid until a satisfactory texture was reached for cutting and further processing. Vertebrae 18 and 19 (lumbar region) were fused by syndesmophyte formation, leading to ankylosis (see Figure 1, top). The intervertebral disc could not be examined but was scanned with Computer Tomography (CT). The remaining organs had no apparent gross lesions.

Spiral computed tomography was performed at Aarhus University Hospital, Aarhus, Denmark, using a Siemens 64 PET/CT biograph (Siemens AG, Erlangen, Germany) with imaging parameters as follows: tube rotation, 360°/s; tube voltage, 120 kv; tube current, 330 mA; and collimation, 64 × 0.6 mm. Images were reconstructed in a 512 × 512 matrix with a 2-mm slice thickness using a B60s convolution kernel. Cross-sectionally, a layer of radiating periosteal exostoses was formed on the middle and ventro-lateral aspects of the vertebral body (see Figure 1, middle & bottom). The layer of new bone was most pronounced along the ventral aspect, which at gross inspection revealed an irregular rugged or crater-shaped surface of the vertebral bodies.

Histopathology was performed at the Danish Technical University and University of Copenhagen.



Figure 1. *Top:* Photo of the fused vertebrae 18 and 19. *Middle:* The surface of the three-dimensional Computer Tomography (CT) of the fused vertebrae shows periosteal exostoses on the mid-ventral and ventro-lateral aspects of the vertebral body. *Bottom:* A cross-sectional slice through the CT image shows that the layer of new bone was most pronounced at the ventral aspect, which at gross inspection revealed a rugged or crater-shaped surface of the vertebral bodies.

Organ samples included the liver, lung, heart muscle, stomach, spleen, lymph nodes, intestines, kidneys, adrenal glands, bladder, and bone, which were formalin-fixed and submitted for routine histologic processing and staining by hematoxylin and eosin. Histopathological findings included hemorrhages in several organs, including the bladder and heart muscle. The patchy distribution of areas with hemorrhagia was most likely consistent with lesions due to blunt trauma and high pressure, and it did not indicate disseminated intravascular coagulation (DIC), sepsis, agonal cardiovascular decompensation, acidosis, or

some other systemic process. Mild accumulations of extracellular bacteria were occasionally seen, and the bronchial epithelium was locally infiltrated with a few lymphoplasmacytic infiltrates. Additionally, necrosis and edema were observed in the bronchial walls, and blood was present in bronchial luminae. In a localized area of the lungs, neutrophils and macrophages were found. Accumulation of hemosiderin was found in the liver macrophages. Furthermore, diffuse atrophy of hepatocytes were loaded with lipofuscin pigment. In the kidneys, we found a focal area of mineralization within the tubular epithelial lining, sclerosis of a few glomeruli, and necrosis of the epithelial lining of the renal pelvis. Multiple thrombi were present within small-size vessels of the spleen. Histologically, the cortical bone tissue revealed a normal osteonal pattern with parallel arrangement of birefringent collagen arranged in concentric lamellae (see Figure 2A & B).

To retain the integrity of the skull, the brain could not be removed. Brain tissue was collected via the foramen magnum, and screening both brain and lung by PCR (Barrett et al., 1993) proved negative for morbillivirus. Serum was tested for antibodies against *Brucella* spp., and lymph nodes were cultured for *Brucella* spp. (MacMillan & Starck, 2000; Jungersen et al., 2006) with negative results. Samples from lung, lung lymph node, liver, spleen, and intestines were cultured for bacteria on blood agar (Blood agar base Number 2, Oxoid, Greve, Denmark) supplemented with 5% calf blood. Non-hemolytic *Escherichia coli* was recovered from lung, hilar and marginal lymph nodes, spleen, intestine, and liver. *Aeromonas hydrophila* was isolated from the lung. However, no associated inflammation was found, so the bacteria might have been a result of contamination.

Fin whales travel alone, in pairs or in small groups (Notarbartolo di Sciara et al., 2003). Single fin whales stranding may be caused by disease, starvation, abandonment (exclusion from the group), or other unknown reasons. None of the pathological findings from the fin whale described herein can account conclusively for the stranding. A possible reason for the stranding might have been the ankylosing spondylosis (also known as spondyloarthrosis) found between vertebrae numbers 18 and 19. It can be speculated that this might have restrained spinal mobility and contributed to stranding and death. This hypothesis is supported by the fact that skeletal abnormalities are common findings in many other cetacean species such as stranded humpback whales (*Megaptera novaeangliae*) (Groch et al., 2012) and long-finned pilot whales (*Globicephala melas*) (Sweeny et al., 2005). In white-beaked dolphins (*Lagenorhynchus albirostris*) from Danish waters, up to 25% of males and 62% of females strand with vertebral ankylosis (Galatius et al.,

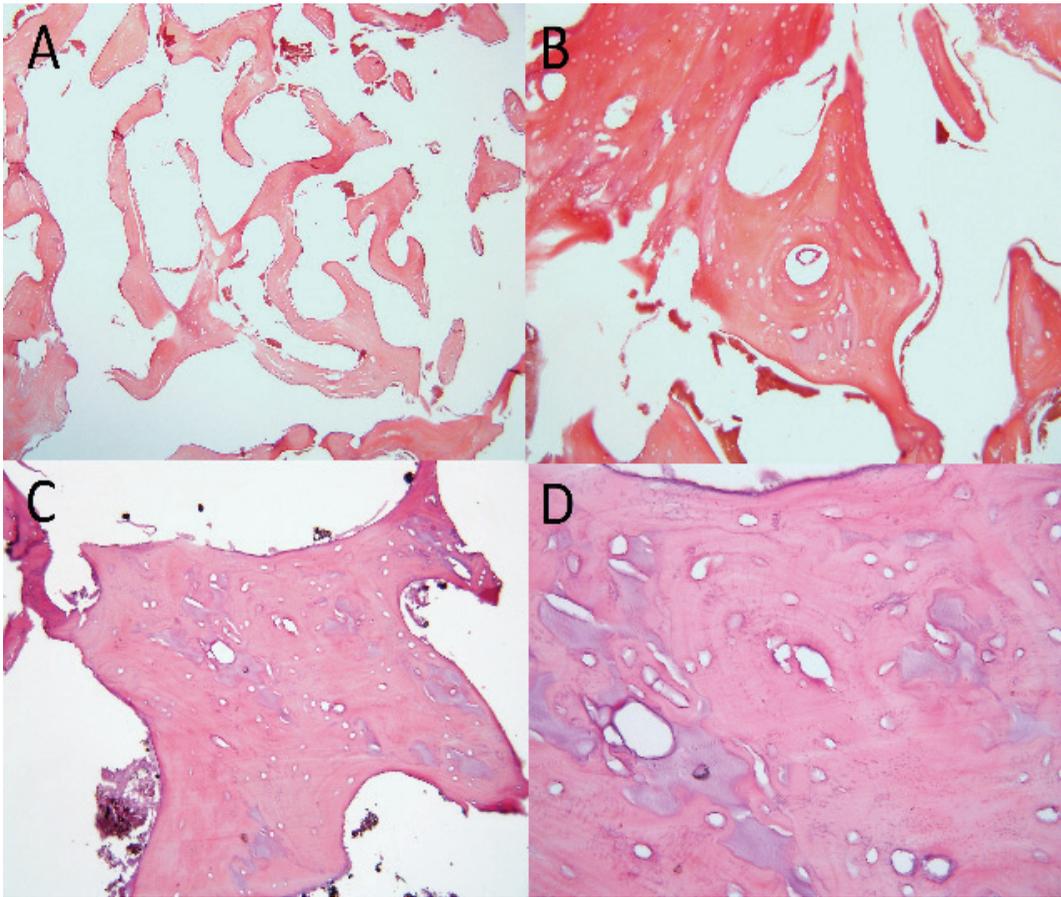


Figure 2. Histologically, the cortical bone tissue revealed a normal osteonal (cortical and lamellar) pattern with parallel arrangement of birefringent collagen arranged in concentric lamellae (A & B). This was also seen in the fin whale stranded in 1905 (C & D). A: x2.5, B: x10, C: x10, and D: x25.

2009). In addition, this is not the first bone pathology report in fin whale. An immature fin whale with fractures of the radius and ulna of the flippers has previously been described (Ogden et al., 1981) as well as other pathological skeletal changes found in other whales (Kompanje, 1999; Hellier et al., 2011). However, the fact that skeletal abnormalities are commonly found in fin whales might indicate that they are neither life-threatening nor the primary reason for stranding. Furthermore, only two of the vertebrae were fused, and no secondary fractures were observed. Therefore, the ankylosing spondylitis could just be a random finding without major clinical importance.

In addition to the fused vertebrae, other vertebrae as well as flipper bones were CT scanned, and no pathological changes were found. Initially, we estimated the mineral density of the vertebrae bones by use of Hounsfield units (HU), a standardized CT attenuation coefficient as it has been

shown to correlate with bone mineral density in vertebrae from living humans (Schreiber et al., 2011). We found a mean of 196 HU (-246 HU; -96 HU) in cervical vertebrae and a mean of 201 HU (-233; -165) in lumbar vertebrae, which in living humans would have indicated osteoporosis (Schreiber et al., 2011). However, by comparing them with similar bones from a male fin whale stranded in the Bay of Vejle in 1905 (-363 HU [-381; -339] and 483 HU [-506; -461], respectively), we realized that mineral density is probably not relevant to investigate in whale bones. The extremely low HU values of the 1905 fin whale are probably due to the drying-out process throughout the past hundred years and not related to decreased bone mineral density. Histologically, the cortical bone tissue revealed a normal osteonal pattern in both the 2010 (Figure 2A & B) and 1905 (Figure 2C & D) fin whales.

The bone pathology was the only significant lesion found on gross examination that might have been related to the stranding of this animal. The empty stomach and small amount of fluid in the intestines indicate that the fin whale had not eaten for several days, and this is also in agreement with the scarce amounts of subcutaneous and visceral fat deposits. The depletion of blubber and visceral fat stores might suggest that the fin whale had an insufficient caloric consumption for more than just a few days. Emaciation could also be considered as a possible contributory factor leading to stranding.

No morbillivirus or antibodies against *Brucella* spp. were detected by molecular screening or serology, respectively, in the present study. The non-hemolytic *E. coli* and *A. hydrophila* found in the lung and other organs were likely *postmortem* invaders or contaminants. These bacteria are often present in the environment (Janda & Abbott, 2010), and they were not associated to microscopic lesions.

We conclude that none of the pathological findings can account convincingly for the stranding of the fin whale. However, we speculate that ankylosing spondylosis may have constrained spinal mobility and contributed to its stranding and subsequent death. We cannot exclude it as just a random finding.

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