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giant Canada geese as the apparent prevalence is quite low. Park managers, though, should be aware of this problem

with measures taken to limit artificial feeding and to remove birds with this condition from the breeding population.

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Idiopathic Scoliosis in a Newborn Sea Otter, *Enhydra lutris* (L.)

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An adult female sea otter, captive since November 1976, was kept at the Seattle Aquarium, Seattle, Washington. Age at captivity was estimated at 3-5 yr. The animal's diet in the aquarium consisted of fish, clams and crabs. The animal was housed with four adult otters in a 211,200 liter pool, 4 m deep, separated from the public by glass walls but open to the seawater of Puget Sound via wire fencing. On 20 November 1982, it gave birth to a female infant. This was its first known pregnancy. The infant was not observed to move and was believed to be born dead. The mother carried the infant to a small ledge at the surface of the pool and proceeded to groom it for 30-40 min. The pup then fell into the pool and sank to the bottom, remaining there for about 5 hr, during which time its head was bitten off by one of the otters.

Five hr following the recovery of the carcass, a gross necropsy was performed at the aquarium, the lungs were infused with formalin and the entire animal, minus the head, was submitted to the Comparative Pathology Laboratory at the University of Washington. Tissues were fixed in 10% phosphate-buffered formalin, dehydrated in alcohol, cleared in xylene and embedded in paraffin. Seven-micron sections were stained with hematoxylin and eosin for general observations, Prussian

blue for hemosiderin, PAS for mucopolysaccharides, and Brown and Brenn for bacteria (Luna, 1968, *Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology*, 3rd Ed., McGraw Hill, New York, 258 pp.).

The carcass, without head and thoracic viscera, weighed 530 g. Length of the spine from neck base to rump was 195 mm. No significant gross lesions other than those attributable to the 2 day post-mortem interval were observed in any of the internal organs. When all abdominal viscera were removed, a 50 mm lateral deviation of the spine to the right was observed from the 5th thoracic vertebra to the thoracolumbar junction (Fig. 1). No gross vertebral or rib anomalies were seen. The deviation appeared to have been present for a long period of time and had resulted in considerable deformation in rib alignment. When the carcass was compressed craniocaudally, the deviation became more accentuated.

Histopathologic examination of the heart, aorta, trachea, esophagus, thymus, salivary gland, thyroid and adrenal glands, uterus, ovary and lungs failed to reveal any significant lesions. No structural abnormalities were observed in muscle, connective tissue or nerves. The lungs were congested, and some alveolar spaces were dilated from infusion of fixative. Some of the epithelial cells lining the bronchioles had a slight brownish discoloration consis-

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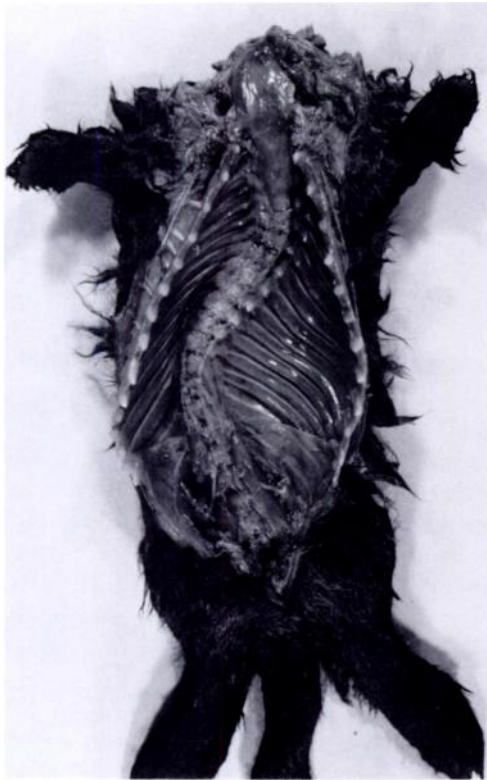


FIGURE 1. Carcass of infant sea otter with head and viscera removed, showing right lateral deviation of the vertebral column from T5 to the thoracolumbar junction.

tent with inspissated bile pigment from inhaled gastric contents. There was no other evidence of gastric content material in the lungs. The marked lobular architecture of the marine mammal lung was readily apparent.

Evidence of a moderate to severe inflammatory process was observed in the peritoneum covering the spleen, uterus, omentum, stomach and other abdominal organs. Along the serosal surface of the stomach the infiltrate often contained oblong, intensely basophilic, acicular deposits suggestive of calcified material. These findings were interpreted as indicative of bile peritonitis which developed in utero. There was no evidence of an infectious agent in any of the affected tissues.

The animal was a stillbirth; however, based on the absence of significant post-mortem change, the fetus must have been alive at the time of delivery. The scoliosis may have contributed in a significant way to the death of the infant by prolonging parturition. Uterine contractions during fetal expulsion could have caused a more severe spinal deviation and resulted in dystocia. A complicating factor of unknown cause was the presence of a bile peritonitis, most severe in tissues near the bile duct, which was only evident histopathologically.

Scoliosis has been classified according to both morphologic and etiologic features (Keim, 1978, CIBA Clinical Symposia 1978 Annual, pp. 2–30). Congenital scoliosis includes those cases with gross vertebral or rib malformations, such as hemivertebrae, incomplete vertebral segmentation, supernumerary ribs or rib fusion. Associated spinal and extraspinal congenital defects are observed often. Spontaneous congenital scoliosis has been reported in many domestic species. A genetic basis for the deformity has been demonstrated in rabbits (Sawin, 1964, Clin. Orthop. 33: 71–90) and chickens (Taylor, 1971, Avian Dis. 15: 376–390; McCarrey, 1981, J. Hered. 72: 6–10). Experimentally, congenital scoliosis has been induced in a variety of animal species by teratogens too numerous to be cited here.

Scoliosis without gross structural deformities may be secondary to neuropathies such as poliomyelitis, encephalitis or syringomyelia or to a variety of myopathies. Scoliosis of this type is rarely identified in the neonate inasmuch as time is required for the tendon or muscle imbalance to exert its scoliotic effect. A kepone-induced scoliosis was produced experimentally in sheepshead pupfish (*Cyprinodon variegatus* Lacepede) following 10 days of exposure to an organochlorine and was proposed to have been caused by neuromuscular dysfunction (Couch, 1977, Science 197: 585–587).

Cases of scoliosis in which no gross morphologic deformities or primary neuromuscular or connective tissue disorders can be identified are termed idiopathic. Females are affected seven times more frequently than males, and one of the most common idiopathic patterns is a right thoracic curve from T4, 5 or 6 to T11, 12 or L1 (Keim, 1978, op. cit.) such as seen in the present case. Studies on idiopathic infantile scoliosis have demonstrated it to be highly familial, with its prevalence increasing with increased maternal age (DeGeorge, 1967, J. Med. Genet. 4: 251–257). At least two researchers have postulated the condition to be inherited, possibly by a sex-linked dominant mode with variable expressivity and incomplete penetrance (Wynne-Davies, 1968, J. Bone Jt. Surg. 50-B: 24–30; Cowell, 1972, Clin. Orthop. 86: 121–131).

The cause of the scoliosis in the sea otter

of this report can only be speculative. While no gross vertebral, spinal or rib anomalies were noted, the absence of the animal's head precluded identification of any skull, palate or brain deformities which, if present, might have suggested a congenital or neurologic etiology. Although histopathology failed to reveal any structural abnormalities in nerves, muscle or connective tissue, primary disease in one or more of these areas cannot be ruled out. Unfortunately circumstances prevented histochemistry, muscle enzyme analysis, electromyography and cerebrospinal fluid analysis, tests which might have provided clues to the origin of the scoliosis.

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Spondylitis Deformans in a Bryde's Whale (*Balaenoptera edeni* Anderson) Stranded on the Southern Coast of Queensland

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The pathological vertebral condition known as spondylitis deformans in cetaceans has been discussed by a number of authors (Slijper, 1936, *In Capita Zoologica*, Martinus Nijhoff, The Hague, The Netherlands, 590 pp.; van Bree and Duguy, 1970, *Der Zoologische Garten* 39: 11–15). Slijper (1936, op. cit.) listed its occurrence in 39 cetaceans, one of which was a Bryde's whale. This paper reports a further example of spondylitis deformans, associated with spinal canal stenosis, in that species.

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On 30 October 1982 a Bryde's whale stranded and died on a sand-bank near the southern end of Stewart Island (25°38'S, 152°57'E) in the Great Sandy Strait. It measured 14.35 m in length, but its sex was not determined. The carcass, when affected by autolysis, drifted for some days before washing ashore on the western side of Fraser Island, approximately 5 km from the stranding site. The skeleton was collected by a team from the Queensland Museum on 15 February 1983 and was subsequently registered (QM JM 4386).

The spinal column between lumbar