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PATHOLOGICAL FINDINGS IN THE HAWAIIAN MONK SEAL

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ABSTRACT: Postmortem examinations were performed on 45 Hawaiian monk seals (*Monachus schauinslandi*) collected during field research on the beaches of the Northwestern Hawaiian Islands (USA) from 1981 to 1985. Both males and females of all age groups, perinatal through adult, were examined. Frequent findings included parasites, trauma, cardiovascular disease (congenital and acquired), and respiratory infections. Emaciation was a common condition. All animals except neonates were infected with parasites; infection was severe in several cases. Splenic hematopoiesis was a universal histopathologic finding. Some cases exhibited lesions consistent with renal, gastrointestinal, and toxic disorders; ectopic tissue calcification; gallstones; and ophthalmologic and dental problems.

Key words: *Monachus schauinslandi*, monk seal, pathology, emaciation, parasites, trauma, survey.

INTRODUCTION

The Hawaiian monk seal (*Monachus schauinslandi*) is an endangered pinniped which lives and breeds primarily in the Northwestern Hawaiian Islands (NWHI) (USA). In the recovery plan for the monk seal, collection of baseline data on diseases was given high priority, as such data could be important to the recovery effort (Gilmartin, 1983).

While not studied extensively, parasitism of the monk seal has been documented from necropsies and examinations of fecal samples: four species of cestodes, *Diphyllobothrium hians* (Markowski, 1952) = *Bothriocephalus* sp. (Chapin, 1925), *D. elegans* and *D. cameroni* (Rausch, 1969), and *D. rauschi* (Andersen, 1987) = *D. hians* (Rausch, 1969); two nematodes, *Contracaecum turgidum* (Chapin, 1925) and *Anisakis* sp. ova recently identified in scats by Dailey et al. (1988); one acanthocephalan, *Corynosoma rauschi* (Golvan, 1959); and one lung mite, *Halarachne laysanae* (Furman and Dailey, 1980). Trematoda ova, not identified to species, also have been reported (Dailey et al., 1988). Clusters of *Contracaecum* sp. were reported attached to the ulcerated gastric mucosa of a young monk seal (Whittow and Balazs, 1979). These lesions with embedded parasites were common findings in an investigation of mortality in monk seals at Laysan Island in 1978 (Gilmartin et al., 1980).

Serological evidence of a low incidence of calicivirus infection exists, and *Salmonella sieburg* and *S. minnesota* were isolated from rectal cultures of monk seals (Gilmartin et al., 1980; Gilmartin, unpubl. data). A toxic disease, ciguatera, also has been implicated in monk seal deaths (Gilmartin et al., 1980).

This report summarizes the necropsy findings from Hawaiian monk seals through a 5-yr period. The primary goal of compiling and examining these postmortem data was to determine whether significant pathological processes existed which might be mitigated by management actions to enhance the recovery of this endangered species.

MATERIALS AND METHODS

Necropsies on Hawaiian monk seals were conducted and tissue specimens collected from 1981 through 1985 at most NWHI locations: Kure Atoll (28°25′N, 178°10′W), Pearl and Hermes Reef (27°55′N, 175°45′W), French Frigate Shoals (FFS) (23°45′N, 166°10′W), and Lisianski (26°02′N, 174°00′W), Laysan (25°42′N, 171°44′W), and Necker (23°35′N, 164°42′W) Islands. Research staff presence at the islands varied greatly in duration, season, and number of personnel.

Dead animals were usually necropsied where they were found by the field biologists, with

TABLE 1. Summary of pathological findings in 42 Hawaiian monk seals necropsied during 1981 to 1985.

		Number of seals affected									
		Adult		Subadult		Juvenile		Pup		Neonate	
Pathological findings	n*	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male
Emaciation	24	5	4	2	3	3	1	0	2	1	2
Parasitism	37	10	7	2	3	7	5	1	2	0	0
Trauma	16	4	1	1	2	0	2	0	2	1	2
Infectious disease	10	2	2	0	1	1	1	0	0	1	2
Cardiovascular disease											
Congenital	5	0	0	0	1	0	2	1	1	0	0
Acquired	5	2	2	0	0	0	1	0	0	0	0
Respiratory disease	6	0	2	0	0	1	0	0	0	1	2
Incidental findings											
Ectopic calcification	6	1	4	0	0	0	1	0	0	0	0
Ophthalmologic disorders	2	1	1	0	0	0	0	0	0	0	0
Gallstones	2	2	0	0	0	0	0	0	0	0	0

* (n = the total of seals observed for each category).

abnormalities noted to the best ability of the prosector. Representative organ samples from apparently normal as well as diseased tissues were fixed in 10% buffered formalin for later histopathologic examination (Winchell, 1990). Routinely sampled tissues included lung, heart, liver, spleen, pancreas, intestine, kidney, adrenal, gonad, lymph node, muscle, and, when located, thyroid and thymus. Samples from other tissues were collected when abnormalities were suspected. Tissue samples were embedded, sectioned, and stained with hematoxylin and eosin by routine methods. Parasites collected from the digestive tract were preserved in alcohol or formalin.

Case information recorded during most necropsies included animal identification, sex, size classification (e.g., adult, subadult, juvenile, pup), necropsy date, and island. When available, historical information on individual seals was used in judging the relative importance of the histopathologic information in the overall picture of the mortality.

RESULTS AND DISCUSSION

General

Postmortem findings on 42 Hawaiian monk seals are summarized in Table 1. Findings have not been separated into primary and contributory factors, as such determinations would too often be speculative or arbitrary. Most of the listed conditions have the potential to be primary, secondary, or contributory factors in the disease process. No significant lesions were identified in three additional animals.

The necropsy data from this study suggest some categorical groupings, but these findings cannot be extrapolated to population losses because of variation in the duration of data collection among the islands, variable quality of postmortem examinations, and the very small sample (less than 5% of the estimated number of animals that died each year were recovered for necropsy). Some abbreviated case histories representing the categories in Table 1 are presented below.

Emaciation

Emaciation (lack of body fullness—characterized internally by a thin or absent fat layer, relatively thin blubber, and muscle wasting) in the seals examined was a common finding. Twenty-four of the animals were thin to severely emaciated. Early weaning and neonatal-maternal separations were the usual causes of pup emaciation. Starvation has commonly been reported in pinniped deaths (Keyes, 1965; Ridgway et al., 1975; Anderson et al., 1979; Fay et al., 1979; Dierauf, 1983). In mor-

talities with evident emaciation, it is usually difficult to determine whether malnutrition lowered resistance to disease or disease compromised an animal's ability to forage. For example, a 2- to 3-wk period of immobility and anorexia with progressive weight loss and debilitation were the primary clinical features at Laysan Island in 1978, where ciguatera and parasite infection were the probable causes of death in at least 50 seals (Gilmartin et al., 1980).

Parasitism

The intensity of parasite infection and associated lesions ranged from mild to severe; only newborn pups did not harbor gastrointestinal parasites. No new parasites were identified during the study.

The largest recorded gastric ulcer in this study was 7 cm in diameter. Usually, an animal had one or two large and many small to medium (1–3 cm) ulcers. Typically, the granulomatous scars and ulcers were more numerous in the older adults (e.g., 26 in a 17-yr-old female). No perforated ulcers were found. One juvenile seal in apparently good physical condition had parasites nearly occluding the lumen of the small bowel and had nine gastric ulcers with embedded nematodes. The ulcers were characterized by a thick pyogenic membrane overlying edematous granulation tissue. Peripheral and subserosal areas were chronically reactive with increased dense connective tissue, including large numbers of fibroblasts and histiocytes, and increased vascularity. Also evidenced was a bacteremia, probably originating from bite injuries inflicted by adult male seals. Hepatic lesions included centrolobular necrosis and fatty changes. It is difficult to ascribe a primary cause of death and determine the role of parasitism in cases such as this. Debilitation from heavy parasitism may have weakened the animal and predisposed it to attack by adult male seals and subsequent septic death, or conversely, the septic condition may have diminished parasite resistance resulting in an increased ante mortem infection.

Trauma

During the 5-yr study, the most common cause of lethal traumatic injuries (11 of 16 cases) was "mobbing" (Table 1). During a mobbing, large numbers ($\leq$27) of adult male monk seals attack a single seal in mating attempts, many actually mounting the subject (Johnson and Johnson, 1981; Alcorn, 1984; Johanos and Austin, 1988). Mobbing injuries are inflicted on adult females primarily but also immature seals of both sexes. The size class and sex of 11 mobbed seals included 4 adult females, 1 subadult female, 2 juvenile females, 2 subadult males, and 2 male pups.

Mobbing incidents usually resulted in large, deep, dorsal bite wounds where skin, blubber, and sometimes muscle, were removed. The secondary effects of the bite injuries and attempted breeding included exposure, dehydration, infection, debilitation, trauma to the reproductive tract, starvation due to inability to forage, and high risk of shark predation. The eventual consequence (recovery or death) of mobbing injuries to an individual appeared unrelated to the subjectively judged gross severity of the injuries.

In addition to dorsal wounds, edema, and subcutaneous hemorrhage, three young adult (5- to 6-yr-old) females suffered traumatic damage to the reproductive tract, one of which had a severe metritis. The other two had recently ovulated and one of these had recently weaned a pup. Among the 11 mobbing victims was a 2-yr-old female with histological evidence of pubertal follicular development at the time of death.

Four of the 11 mobbing victims had chest and pulmonary lesions consistent with severe trauma. Two victims were male pups (3.5- and 4.5-mo-old), one was a yearling female, and one a 4-yr-old male. An adult male was observed mounting the 3.5-mo-old pup in shallow water, biting its back and flippers, and then leaving it in respiratory distress. Congested, hemorrhagic pulmonary tissue and severely

bruised abdominal blubber were found at necropsy of the latter seal.

Only a few incidents were documented in which trauma unrelated to male aggression was associated with mortality. Deaths of three neonates were probably related to perinatal trauma as reported in other pinnipeds (Andersen et al., 1979). Findings in the neonates included traumatic liver damage and aspirated amniotic fluid in one pup and a large quantity of blood in the thoracic and abdominal cavities of the other two pups, including a hemopericardium in one of the latter pups. A weaned male pup died after it swallowed a spiny finned fish, tail first: The fins caught in the cervical esophagus, partially obstructing it and eventually necrosing through the esophageal wall. Local infection, inflammatory reaction, and pressure-induced necrosis of the tracheal wall were observed. Small pieces of the rotted fish were identified in the bronchial tree and had caused aspiration pneumonia.

Other sources of trauma which may lead to infections and deaths are shark attack and entanglement in marine debris (Alcorn and Kam, 1986; Henderson, 1990). These appear to occur at a low frequency in the population.

Infectious disease

Infectious disease was established from findings or was suspected in 10 cases. Septicemia was histologically identified in five cases, four of which were linked to bite-wounds inflicted by adult males and included dermal abscesses and infected gastric ulcers. One of the latter group was a pup, probably infected perinatally, that died shortly after birth. The mother of this pup was sighted with an extensive back wound of mobbing origin 2 wk prior to parturition.

The only seal with a bacteremia not associated with trauma had a ruptured renal abscess. Microscopic examination of a necrotic kidney revealed severe nephrolithiasis with secondary renal abscess formation and nephrocalcinosis. A myocardial infarction and moderate pulmonary emphysema with interstitial calcification and a heavy monocytic infiltrate were also found.

A presumed prenatal infection caused developmental abnormalities, including a grossly misshapen skull. This pup appeared uncoordinated and weak at birth (Alcorn and Buelna, 1989). Respiratory distress was punctuated by "wet" respiratory sounds and froth at the nostrils. It died at the termination of one of several convulsive episodes of increasing duration. Approximately 25% of the animal's ventrum and the entire tail were covered with granulomatous dermal nodules between 2.5 and 20.0 cm in diameter. The same type of lesion was found 3.5 cm distal to the pup's umbilicus on the umbilical cord. These nodules contained epidermal elements, including hair. The pup's eyes were affected bilaterally with buphthalmos. Glaucoma was secondary to obliteration of the Canal of Schlemm by inflammatory cells and reactive tissue. All body tissues and placenta were heavily and diffusely infiltrated with leukocytes, primarily neutrophils. Examination of pulmonary tissues revealed marked atelectasis, diffuse leukocytic infiltration, and aspirated amniotic fluid. Notably, the mother of this pup gave birth to apparently normal pups before and after this pup.

Congenital cardiovascular disease

Of five seals with congenital anomalies, two had a patent ductus arteriosus. One, a 1.5-yr-old male, died in captivity from a stress myopathy syndrome. He was found to have a 0.5 mm patency. Another juvenile male was found to have a 1.5 mm patency. A high prevalence of ductus arteriosus patencies (1.0–2.0 cm) in pups at FFS was confirmed in 1986 (W. G. Gilmartin, unpubl. data). Not all of these seals were premature. Patent ductus arteriosus was reported in several harbor seals (*Phoca vitulina*) found stranded along the Oregon coast (Stroud, 1979). It is not known

whether this is a pathologic condition in seals.

Other congenital findings included an anomalous moderator band in a subadult male, a 2.5 cm ventricular septal defect in a 4.5-wk-old female pup, and a patent foramen ovale in a 3-day-old male. The moderator band was discovered midchamber in the right ventricle, extending horizontally from the medial to lateral wall. The ventricular wall was thin (2 mm) relative to other hearts examined.

Findings in the pup with the patent foramen ovale included 200 ml of serosanguinous abdominal fluid; 50 ml of a pink-yellow pericardial fluid; congestion of the splenic, pulmonary, renal, and hepatic tissues; and splenic and hepatic hemosiderosis. The findings suggest that the patency was a significant contributing factor to the death.

Acquired cardiovascular disease

Acquired cardiovascular lesions were observed in four aged monk seals, two each, male and female, and a juvenile male. The two females were aged by their canine teeth, but all four seals exhibited physical characteristics and incidental necropsy findings consistent with old age. A 17-yr-old female had cerebral infarcts and recent laminar necrosis of the cerebral cortex with associated focal collections of neutrophils and erythrocytes. She also had severe focal arteriosclerosis with luminal occlusion in pancreatic arterioles and less severe lesions in the arterioles of the spleen and periadrenal fat. Severe congestion in pulmonary and hepatic tissues was suggestive of terminal heart failure.

An old male exhibited several cardiac and vascular changes, including myocarditis, myocardial calcification, myocardial necrosis, and calcification of the internal elastic lamina of the renal artery.

The other aged male died the morning after his capture. Severe passive congestion of pulmonary tissue and a chronic focal lipid pneumonia was reported. Focal calcification of the internal elastic lamina of the cardiac arteries and severe passive congestion with early cardiac cirrhosis of the liver were also observed. Centrilobular hepatic scarring suggested prior episodes of severe passive congestion. Hyperplasia of the vesicular and reticular adrenal cortical zones with atrophy of the glomerular zone was suggestive of a pre-neoplastic state. Adrenal changes may have lead to an aberrant stress response contributing to terminal heart failure. These data suggest that age-related cardiovascular changes may be significant factors in the death of aged seals.

Mild arteriosclerosis in the spleen of a juvenile (1.5-yr-old) male was a surprising finding. The cause of death was not apparent in this animal.

Respiratory disease

Respiratory disorders included two cases of chronic pneumonia with associated pleuritis, one case of aspiration pneumonia, two prenatal pneumonias associated with generalized infectious disease, and one newborn male where considerable atelectasis and aspirated amniotic fluid were the only lesions observed.

Partial atelectasis was commonly observed in newborns. This was not related to primary lung pathology, but likely to prolonged birth, non-viable individuals who lived only a short time, or animals sufficated by the placenta or their mothers. A male, a female born with generalized infection, and a female with evidence of perinatal trauma were found to have fluid-filled lungs, possibly amniotic fluid indicative of difficult parturition.

Primary pulmonary disease of unknown etiology was believed to be the cause of death in at least two animals: a 20-yr-old male with chronic pneumonic lesions and associated pleuritis and a 5-yr-old male with severe septal congestion, interstitial edema, pleural edema, and atelectasis.

Incidental findings

Hematopoiesis was evident in all spleen samples. Neither the sex, age, nor specific

disease processes correlated with the degree of splenic hematopoiesis. In many cases, hepatic tissue was too autolyzed for the identification of hematopoietic activity, but in two cases, some hepatic hematopoiesis was evident.

Ectopic tissue calcification was found in five cases: (1) focal calcification of the intramural arteries in the uterus of an adult female; (2) calcification in the elastic lamina of coronary arterioles in an aged male; (3) focally calcified connective tissue in a fibrotic area of the adrenal gland, focal renal deposits in the medula and basement membranes of proximal convoluted tubules, pulmonary deposits in the walls of septal blood vessels and within hyaline membranes of peripheral small bronchi—all found in the tissues of an old male; (4) focal calcification in the adrenal cortex of a 1-yr-old male with pseudogland formation in the glomerular layer and mild arteriolar sclerosis of the spleen; and (5) multiple sites of focal calcification in a 17-yr-old male seal, myocardial calcification adjacent to bacterial myocarditis with infarct and necrosis, interstitial calcification in pulmonary alveolar septa with bacterial pneumonia and emphysema, nephrocalcinosis in the kidney with abscess formation secondary to nephrolithiasis, and calcification of the internal elastic membrane of the renal artery.

Tissue calcification may be related to changes typical of old age or to disease states including chronic infection and endocrine and mineral imbalances. Primary hyperparathyroidism occurs at a low frequency in domestic and wild mammals, and lesions in these cases do not support such an etiology.

Signs of ophthalmologic disease are not commonly observed in wild monk seal populations and were also infrequently found at necropsy. Ophthalmologic problems of undetermined etiology were observed in two seals. Trauma or infectious disease were possible causes of corneal clouding in a 5-yr-old male that died of sepsis secondary to a mobbing wound. A 17-yr-old female had a central cicatricial scar on the opaque yellow cornea of one eye. Other findings included atherosclerosis, cerebral infarct, pancreatitis, and a pale yellow gallstone (1 × 3 mm).

In two seals, ages 17 and 24, very worn dentition was reported. This condition was probably more common than noted, although most older seals probably exhibited it. One aged male had severe osteolysis of both the mandible and maxilla, such as might be associated with root abscesses.

CONCLUSIONS

These findings are of importance to the monk seal recovery effort in that the information has led to (1) identification of some causes of mortality in the monk seal population; (2) information about general disease processes and age-related changes in monk seal organ systems; (3) compilation of a histologic data base for future evaluation of monk seal tissues; and (4) reevaluation of postmortem procedures resulting in implementation of changes which will allow gathering of more pertinent morphologic, physiologic, and pathologic data.

The data indicate that deaths from septicemia resulting from wounds received in adult male mobbing attacks account for more monk seal deaths than were previously believed associated with this behavior. Parasitism is a usual finding, but of undeterminable significance in monk seal disease processes. Importantly, there does not appear to be any disease phenomenon affecting the population in a manner which may significantly hinder recovery of the species.

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