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## FINDINGS IN PINNIPEDS STRANDED ALONG THE CENTRAL AND NORTHERN CALIFORNIA COAST, 1984–1990

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**ABSTRACT:** Personnel at The Marine Mammal Center (The Center) treated 1,446 stranded marine mammals recovered from the central and northern California (USA) coast from 1984 through 1990, including California sea lions (*Zalophus californianus*), northern elephant seals (*Mirounga angustirostris*), Pacific harbor seals (*Phoca vitulina richardsi*), northern fur seals (*Callorhinus ursinus*), Steller sea lions (*Eumetopias jubatus*), and Guadalupe fur seals (*Arctocephalus townsendi*). The primary disease findings in stranded California sea lions were renal disease, renal disease complicated by severe verminous pneumonia, verminous pneumonia, seizures of unknown etiology, and renal disease complicated by severe pneumonia of unknown etiology. Stranded elephant seals included pups, yearlings with dermatological problems, and neonates. Most harbor seals admitted to The Center were underweight and premature pups. Stranded northern fur seals included animals with seizures of unknown etiology and emaciated pups. Stranded Steller sea lions included underweight pups and aged adult females with pneumonia. Two Guadalupe fur seals had hemorrhagic gastroenteritis. Incidental findings at the time of stranding among the six species included verminous pneumonia and pneumonia of unknown etiology, renal disease, internal parasitism, ophthalmologic problems, gastrointestinal disorders, otitis externa, and external wounds.

**Key words:** *Zalophus californianus*, *Mirounga angustirostris*, *Phoca vitulina richardsi*, *Callorhinus ursinus*, *Arctocephalus townsendi*, *Eumetopias jubatus*, stranding, health problems, disease.

### INTRODUCTION

Numerous scientists have studied pinniped diseases based on information obtained from captive animals (Ridgway, 1972; Wallach, 1972; Sweeney, 1974a, b; Medway, 1980) and stranded pinnipeds from the wild (Schroeder et al., 1973; Sweeney and Gilmartin, 1974; Geraci and Sweeney, 1979; Stroud and Roffe, 1979; Dierauf, 1983). Studies of stranding causes and the medical care of stranded marine mammals provide valuable information concerning health care of these animals in captivity, and the assessment of health problems in the wild.

The Marine Mammal Center (The Center), Sausalito, California (USA), is a marine mammal rehabilitation facility. Pinnipeds which stranded along the central and northern California coast were hospitalized and released to the wild after successful medical treatment. From January 1984 through December 1990, 1,446 pinnipeds representing six species were

admitted to The Center. The six species were the California sea lion (*Zalophus californianus*), northern elephant seal (*Mirounga angustirostris*), Pacific harbor seal (*Phoca vitulina richardsi*), northern fur seal (*Callorhinus ursinus*), Steller sea lion (*Eumetopias jubatus*), and Guadalupe fur seal (*Arctocephalus townsendi*). The objective of this retrospective study was to identify the primary disease findings and incidental findings in pinnipeds, based on clinical evaluation and post mortem examination.

### MATERIALS AND METHODS

Pinnipeds were considered stranded and were recovered from the beach if they were on a high public use beach and would not retreat to the water, if they were obviously ill or underweight, or if after encouragement to return to the water they remained on the beach for 24 to 48 hr. Sick or injured marine mammals were not typically removed from established or known haul-out or rookery areas.

Routine procedures performed on all live stranded pinnipeds admitted to The Center included: a physical examination, blood collection

for complete blood count (CBC) and serum chemistry profile, fecal flotation assessment for the presence of parasites, and the initiation of an individual medical and husbandry program. Staff monitored patient progress on a daily basis. An animal was considered successfully rehabilitated if it was clinically healthy based on normal CBC and serum chemistry profile, satisfactory body weight, and exhibited normal behavior. Prior to release, rehabilitated pinnipeds were flipper tagged for potential future identification in the wild. Rehabilitated animals not fit for release to the wild were placed in zoological parks.

Medical and scientific staff performed a gross post mortem examination on animals that died during rehabilitation or animals dead on arrival (DOA). Pinnipeds that survived <24 hr at The Center were classified as DOA. Post mortem examinations were performed <18 hr of death. During necropsies tissues were collected on the basis of gross abnormalities and fixed in 10% buffered formalin. Tissues were sent to Pathology Service, Veterinary Medical Teaching Hospital, University of California, Davis, California (USA), or to California Veterinary Diagnostics (CVD), West Sacramento, California, for histopathological evaluation using hematoxylin and eosin stains.

Bacterial and fungal cultures were performed at CVD using methods from Carter (1984). In 1984, microscopic agglutination tests (Galton et al., 1962) using standardized *Leptospira pomona* antigen were performed on paired serum samples from six animals; four samples were taken on admission and before release, and two samples were taken on admission and immediately after death. Diagnostic testing in 1984 also included wet mount, darkfield microscopic evaluation of kidney or urine samples from 14 different California sea lions. In 1988, microscopic agglutination tests with known strains of leptospirosis were performed on four paired serum samples. The antibody titers to *Leptospira pomona* ranged from 6,400 to 12,800; and to *L. icterohaemorrhagiae* they ranged from 1,600 to 12,800. Two serum samples had titers of 12,800 to both *L. pomona* and *L. icterohaemorrhagiae* (N. A. Vedros, pers. comm.). Isolation of *Leptospira pomona* was never performed during the 1984 and 1988 outbreaks of renal disease.

Hematology was done at French Hospital, San Francisco, California, until 1989, and then at Marin General Hospital, Greenbrae, California. Serum chemistries were performed on a Dacos® Analyzer (Coulter Electronic, Inc., Hialeah, Florida, USA). Hematology was determined using a Coulter Counter® Model S-Plus IV (Coulter Electronic, Inc.).

The presence of internal parasites was evaluated by fecal flotation (Ovatector® Disposable Fecal Diagnostic System, B. G. S. Medical Products, Venice, Florida) and post mortem examination. Tentative identification was made using the diagnostic key of Dailey and Gilmartin (1980). Post mortem identification of *Contracium* was made following descriptions of Baylis (1936), *Parafilaroides* after Dailey and Hill (1970), and *Dipetalonema* from Taylor et al. (1961). Other helminths were identified based on descriptions by Margolis (1956). Parasites were deposited at The Center, the University of California, Davis, and Ocean Studies Institute in Long Beach, California.

Data were obtained from The Center's medical records dated January 1984 through December 1990. Recorded information included species, age, sex, admission weight, date of stranding, primary disease finding (PDF), incidental findings at the time of stranding, fate of animal (release, DOA, subsequent death, or relocated to another beach), laboratory results, and gross post mortem and histopathology results.

The PDF was defined as the most probable reason why the animal stranded. Incidental findings (IF) at the time of stranding were secondary health problems that may or may not have added to the overall debilitation of the animal, or incidental findings on post mortem examination. The primary disease finding and IF were diagnosed based on clinical data and pathologic examination. Data pertaining to PDF were placed into one of four categories: successfully rehabilitated and released or placed; animals which died during rehabilitation; DOA animals; and animals harassed by people or dogs, but found to be clinically healthy and released to another beach.

The diagnosis of a specific disease was not always possible to determine due to the lack of extensive diagnostic testing. Disease diagnosis was based on information available. For example, clinical diagnosis of renal disease was based on clinical signs which included polydipsia, depression, tucked-up posture (hind flippers tucked under body), leukocytosis, elevated blood urea nitrogen, phosphorus and creatinine levels, and hyperglobulinemia. Pathological diagnosis was based on kidney gross pathology and in some cases, renal histopathology.

Vermineous pneumonia as a PDF or IF was diagnosed clinically, based on lung auscultation, a fecal flotation positive for lungworm larvae, or post mortem examination. A clinical diagnosis of pneumonia of unknown etiology was based on lung auscultation, leukocytosis, and a fecal flotation negative for lungworm larvae. A

diagnosis of pneumonia of unknown etiology was supported by pathological findings in dead pinnipeds.

Traumatic injuries were segregated into specific categories. Fisheries interactions as a PDF included pinnipeds entangled in nets, fishing line, or fish hooks. Trauma as an IF included cutaneous abscesses, conspecific bite wounds, and lacerations. Eye injury was not included as an IF for animals listed as fisheries interactions as the PDF because fisheries interactions often involved head and corresponding eye injuries. Eye injuries as an IF included blindness, corneal ulcer, corneal edema, hypopyon, conjunctivitis, cataracts, trauma, and puncture wounds.

Gastrointestinal disorders as an IF were diagnosed clinically by observation of bloody feces or diarrhea, or on post mortem examination. Gastrointestinal disorders included gastritis, enteritis, and colitis. On gross observations of the gastrointestinal tract on post mortem examination, we did not distinguish between congestion due to stress or shock, inflammation due to unidentified pathogens, and post mortem autolysis. Histopathology of the gastrointestinal tract often was not performed.

On admission to The Center, hypoglobulinemia was diagnosed when serum globulin was <2.0 mg/dl, hyperbilirubinemia was evident when serum bilirubin was >15.0 mg/dl, and hypoglycemia was diagnosed when serum glucose was <80.0 mg/dl.

Age classifications were defined as the following: pups were under one year, yearlings were one to two years, subadults were between two years and adults, and adults were sexually mature animals. Neonatal elephant seal pups were identified by the presence of a complete lanugo coat, teeth not erupted from the gum line, and because they were not yet weaned. Premature pups were defined for Pacific harbor seals as pups with all or partial lanugo present.

Animals euthanatized due to poor probability of survival were administered Beuthanasia-D Special® (Schering Plough, Kenilworth, New Jersey, USA) at a dose of 1 ml/5 kg for otariids and phocids. The drug was administered intravenously in phocids. Otariids received an intracardiac injection at the same dosage after initial sedation using Rampun® (Hewer/Diamond Scientific, Mobay Corp., Animal Health Division, Shawnee, Kansas, USA) given intraperitoneally at a dose of 1 ml per 10 kg.

## RESULTS

We examined 1,446 stranded pinnipeds. Of these, 618 were released, 562 died during rehabilitation, 249 were DOA, and 17

healthy animals were relocated from harassment by dogs and humans on the beach. Post mortem examinations were performed on 730 (90%) of the dead animals.

*California sea lion:* Of the 765 California sea lions examined, 141 were female, 623 were male, and gender for one animal was not recorded. The uneven sex ratio can be attributed to the migratory patterns of this species (Ainley et al., 1982). By age class we examined 12 pups, 305 yearlings, 453 subadults, and 95 adults.

The major PDF's were renal disease (32%), renal disease complicated by severe verminous pneumonia (14%), verminous pneumonia (10%), seizures of unknown etiology (6%), and renal disease complicated by severe pneumonia of unknown etiology (5%) (Table 1).

Renal disease was a PDF in 243 California sea lions, and was observed most frequently in 1984 ( $n = 107$ ) and 1988 ( $n = 88$ ); it occurred primarily in male yearlings and subadults. Among the 243 California sea lions with renal disease as a PDF, 78 (32%) had gastrointestinal disorders as an IF; another 49 (21%) had vesicular lesions on the palmate surfaces of the flippers and oral mucosa suggestive of San Miguel sea lion virus infection (SMSV) (Gage et al., 1990) as an IF. Renal disease as an IF was seen every year of this study. Based on post mortem examination results of animals that did not clinically exhibit renal disease as a PDF, 13% of California sea lions had mild to moderate kidney pathology suggestive of renal disease as an IF.

Forty-seven (6%) of the 765 California sea lions, both male and female, were recovered exhibiting seizures of unknown etiology as a PDF (Table 1). Types of seizures ranged from short episodes to grand mal seizures. Based on the clinical diagnostic testing and pathologic examination, we found no clear etiology for the seizures.

Pneumonia was a common health problem in California sea lions, both as a PDF and as an IF. We found that 62 (26%) of

TABLE 1. Primary disease findings in California sea lions, 1984 to 1990.

Primary Disease Finding (n = 765)	Released <sup>a</sup>	Died <sup>b</sup>	DOA <sup>c</sup>
Renal disease (RD) (31.8%)	104	93	46
Verminous pneumonia & RD (13.9%)	17	50	39
Verminous pneumonia (9.8%)	19	30	26
Seizures (6.1%)	28	16	3
Unknown pneumonia (5.6%)	19	15	9
RD/unknown pneumonia (5.1%)	8	17	14
Transitional cell carcinoma (4.2%)	0	27	5
Gunshot (4.2%)	7	22	4
Underweight adult (3.9%)	23	3	4
Fisheries interactions (3.3%)	25	10	11
Unknown (2.7%)	3	0	18
G.I. ulcer perforation (1.4%)	0	7	4
Shark bite (1.0%)	3	2	3
Ulcerative enteritis (0.8%)	0	4	5
Hepatic failure (0.7%)	0	4	1
Underweight pups (0.5%)	3	1	0
Eye injury (0.5%)	2	1	1
Coccidiomycosis (0.3%)	0	1	1
Pyometra (0.3%)	0	1	1
Lymphosarcoma (0.1%)	0	1	0
Necrotic esophagitis (0.1%)	0	1	0
Otitis (0.1%)	1	0	0
Heart failure (0.1%)	0	1	0
Tar and oil on fur (0.1%) <sup>d</sup>	1	0	0
Totals	266	300	199

<sup>a</sup> Rehabilitated and released.<sup>b</sup> Does not include animals which died within 24 hours of admission.<sup>c</sup> Animals which died within 24 hours of admission or on the beach.<sup>d</sup> No evidence of ingestion.

the 243 California sea lions with renal disease as a PDF also had mild to moderate verminous pneumonia as an IF, and 14 (6%) had mild to moderate pneumonia of unknown etiology as an IF. Based on the clinical and post mortem examinations of all California sea lions (excluding animals with renal disease, verminous pneumonia, or pneumonia of unknown etiology as a PDF), we found that 29% had mild to moderate verminous pneumonia as an IF, and 4% had mild to moderate pneumonia of unknown etiology as an IF.

Trauma was recorded as a PDF for specific causes such as gunshot (4%), fisheries interactions (3%), and shark bites (1%). Trauma as an IF was present in 3% of all admitted California sea lions, and another 8% had an eye injury as an IF.

Internal parasites were a common find-

ing in stranded California sea lions. Gastric nematodes, tentatively identified as *Anisakis* sp. and *Contracaecum* sp., were most common, and were found in 532 (70%) of the sea lions. Gastric ulcers related to nematode infection were observed in 306 (61%) of 499 dead California sea lions. A nematode, tentatively identified as *Parafilaroides decorus*, was found in 442 (58%) of the sea lions. Other parasites tentatively identified were: *Diphyllbothrium* sp., found in 297 (39%) sea lions, *Zalophotrema* sp. and *Pricetrema* sp. found in 212 (28%) sea lions, hookworms (nematodes) in two (0.3%) sea lions and coccidia, cryptosporidia, and the nematode, *Dipetalonema* sp., each seen in one (0.1%) sea lion.

**Northern fur seal:** Of the 18 recorded fur seal strandings, there were 13 females, four males, and one animal of unrecorded

TABLE 2. Primary disease findings in northern fur seals, Steller sea lions and Guadalupe fur seals, 1984 to 1990.

Primary disease finding	Released <sup>a</sup>	Died <sup>b</sup>	DOA <sup>c</sup>
Northern fur seals ( <i>n</i> = 18)			
Emaciated pups (33.3%)	0	4	2
Seizures (22.2%)	1	1	2
Unknown pneumonia (22.2%)	3	0	1
Verminous pneumonia (11.1%)	0	1	1
Unknown (11.1%)	1	0	1
Steller sea lions ( <i>n</i> = 7)			
Underweight pups (71.4%)	3	1	1
Pneumonia (28.6%)	0	2	0
Guadalupe fur seals ( <i>n</i> = 2)			
Hemorrhagic gastroenteritis (100%)	0	2	0

<sup>a</sup> Rehabilitated and released.<sup>b</sup> Does not include animals which died within 24 hours of admission.<sup>c</sup> Animals which died within 24 hours of admission or on the beach.

gender. A total of 11 weaned pups, one yearling, and six adults stranded. Stranded animals included six emaciated pups and four animals with seizures of unknown etiology (Table 2). None of the emaciated pups survived. The four fur seals with seizures were admitted in 1988 along with the California sea lions having seizures. The etiology of the seizures was not determined.

Pneumonia as a PDF was common. Verminous pneumonia was a PDF in two animals, and pneumonia of unknown etiology was a PDF in four animals.

Internal parasites were not common in stranded northern fur seals because most of the animals were not old enough to be parasitized. Tentative diagnosis of parasites included two animals with *Anisakis* sp. and/or *Contracaecum* sp., two with *Parafilaroides decorus*, two with *Diphyllobothrium* sp., and one animal with a hookworm (nematode) infection.

**Steller sea lion:** Five female and two male Steller sea lions stranded during the study period (Table 2). Of these, five were underweight pups and two were adults with severe pneumonia of unknown etiology. Three of the five underweight pups were known to be orphans. One pup had pneumonia and seizures as an IF. One fe-

male had uterine fibroleiomyoma and bilateral, ovarian sex cord stromal tumors, while another adult female Steller sea lion had a moderate endometritis. Both had evidence of old gunshot wounds.

The two adult females had trematode and nematode infections. One pup had a lungworm (nematode) infection.

**Guadalupe fur seal:** Guadalupe fur seals are rare north of Mexico; thus, any stranding along the California coast is noteworthy. Two female Guadalupe fur seals, one pup and one yearling, stranded during 1984 and 1988. Both had hemorrhagic gastroenteritis (Table 2). The IF in the pup included emaciation, bilateral cataracts, and a pneumothorax. The yearling also was emaciated and had evidence of being entangled in a gill net. Nematodes and cestodes were noted on post mortem examination in the pup.

**Northern elephant seal:** Of the 427 stranded elephant seals, 183 were male, 240 female, and no gender was recorded for four animals. By age class, there were 255 pups and neonates, 171 yearlings, and one adult. The mean weight of elephant seal pups admitted to The Center was 41 kg (*n* = 255, SD = 10). The PDF's included 164 (39%) emaciated pups, 156 (37%) yearlings with dermatological problems,

TABLE 3. Primary disease findings in northern elephant seals, 1984 to 1990.

Primary disease finding (n = 427) <sup>c</sup>	Released <sup>b</sup>	Died <sup>c</sup>	DOA <sup>d</sup>
<b>Pups</b>			
Emaciation (38.6%) <sup>e</sup>	121	43	0
Underweight neonates (6.3%)	18	9	0
Trauma (3.7%)	10	6	0
Unknown pneumonia (2.8%)	3	7	1
Gastroenteritis (2.8%)	2	9	1
Verminous pneumonia (0.9%)	1	2	1
Hepatic failure (0.7%)	0	3	0
Oil and tar on fur (0.7%) <sup>f</sup>	2	1	0
Renal failure (0.7%)	1	2	0
Eye injury (0.2%)	1	0	0
Gall stones (0.2%)	1	0	0
Patent ductus arteriosus (0.2%)	0	1	0
Possible nocardiosis (0.2%)	0	1	0
Unknown (0.2%)	0	1	0
Dermatological problems (36.5%)	98	54	4
Verminous pneumonia (0.5%)	0	1	1
Trauma/shark bite (0.5%)	0	2	0
Unknown (0.2%)	0	1	0
Emaciation (0.2%)	1	0	0
Fisheries interactions (0.2%)	1	0	0
<b>Totals</b>	<b>259</b>	<b>144</b>	<b>8</b>

<sup>a</sup> Sixteen animals were relocated due to harassment by people and dogs.

<sup>b</sup> Rehabilitated and released.

<sup>c</sup> Does not include animals which died within 24 hours of admission.

<sup>d</sup> Animals which died within 24 hours of admission or on the beach.

<sup>e</sup> Percentages are of total.

<sup>f</sup> No evidence of ingestion.

and 27 (6%) underweight neonates (Table 3). The major cause of death of pups at The Center during rehabilitation was diagnosed as gastrointestinal disorders.

Eye injuries were seen in 54 (21%) of the 255 pups as an IF, and 33 (13%) of pups were admitted with injuries related to trauma as an IF. One of the neonates had patent ductus arteriosus (PDA) diagnosed on post mortem examination.

During the study period, 154 (37%) elephant seals were admitted with skin disease of unknown etiology as a PDF. Skin disease was characterized by mild to severe generalized, ulcerative, follicular dermatitis. By sex, 64 males, 87 females, and three elephant seals of unrecorded gender had skin disease. The disease appeared primarily in yearlings (1 to 2-yr-olds); however, six 9-mo-old pups also were admitted

with skin disease. Verminous pneumonia as an IF was seen in 17 (11%) of skin diseased elephant seals; 5 (3%) animals had pneumonia of unknown etiology as an IF. Eye injury as an IF was seen in 8 (5%) of skin-diseased elephant seals.

Internal parasites were common in northern elephant seals. Tentatively identified parasites included: *Contracaecum* sp. or *Antisakis* sp. were found in 200 (47%) elephant seals, lungworms (*Paraflarioide decorus* or *Otostrongylus* sp.) were found in 130 (30%) animals, tapeworms (*Dipyllobothrium* sp.) were found in 81 (19%) animals, and trematodes were found in 29 (7%) elephant seals.

**Pacific harbor seal:** Of 227 harbor seals treated, there were 135 males, 91 females, and one animal of unrecorded gender. In total, 212 pups stranded during the study

period, along with eight subadults and seven adults. Harbor seal pups admitted to The Center had a mean weight of 8 kg ( $n = 212$ ,  $SD = 2$ ). The PDF of stranded animals included 77 (34%) underweight pups and 70 (31%) premature neonates. The most common causes of harbor seal pup death were gastrointestinal disorders (Table 4).

The IF in admitted harbor seal pups included 15 (7%) eye injuries, 11 (5%) trauma, 9 (4%) pneumonia, and 2 (1%) otitis externa. Hypoglobulinemia, hyperbilirubinemia, and hypoglycemia were found in harbor seal pups and premature neonates. On admission, hypoglobulinemia was found in 58 (27%) of 212 hospitalized pups, hyperbilirubinemia in 25 (12%) of the pups, and hypoglycemia in 25 (12%) pups. On post mortem examination, ten premature neonates had patent ductus arteriosus; however, the heart was not consistently examined during the study period.

Seal pox as described by Hastings et al. (1989) occurred in 16 (7%) of all admitted harbor seals and was considered an IF.

Internal parasites were not a common finding in Pacific harbor seals admitted to The Center. Tentatively identified parasites included: *Contracaecum* sp. or *Anisakis* sp. found in eleven (5%) harbor seals, lungworms (*Otostrongylus circumlitis*) found in ten (4%) animals, trematodes found in three (1%) harbor seals, and tapeworms (*Diphyllbothrium* sp.) found in one (0.5%) animal.

#### DISCUSSION

We documented health problems in six species of pinnipeds along the California coast. Definitive diagnoses and extensive diagnostic testing of disease problems was not possible in most pinnipeds admitted to The Center due to the lack of scientific personnel and financial constraints.

In comparison to an earlier six year study at The Center (Dierauf, 1983) in which 303 pinnipeds were recovered from 1975 through 1980, this study involved 1,446 cases over a time frame of 7 yr. Escalating

TABLE 4. Primary disease findings in Pacific harbor seals, 1984 to 1990.

Primary disease finding ( $n = 227$ ) <sup>a</sup>	Re- leased <sup>b</sup>	Died <sup>c</sup>	DOA <sup>d</sup>
<b>Pups</b>			
Underweight (33.9%) <sup>e</sup>	38	39	0
Trauma (14.5%)	10	16	7
Umbilical infection (5.7%)	2	10	1
Pneumonia (4.8%)	0	5	6
Gastroenteritis (3.5%)	0	7	1
Premature neonates (30.8%)	33	24	13
Fisheries interactions (1.8%)	1	2	1
Pneumonia (1.3%)	1	1	1
Trauma/shark bite (0.8%)	0	1	1
Unknown (1.3%)	0	1	0
Hepatic failure (0.4%)	0	0	1
Pyometra (0.4%)	0	1	2
<b>Totals</b>	<b>85</b>	<b>107</b>	<b>34</b>

<sup>a</sup> One animal of this total was relocated due to harassment by people or dogs.

<sup>b</sup> Rehabilitated and released.

<sup>c</sup> Does not include animals which died within 24 hours of admission.

<sup>d</sup> Animals which died within 24 hours of admission or on the beach.

<sup>e</sup> Percent of total cases.

numbers of reported stranded animals may have resulted from increased public awareness of The Center's rehabilitation program, increased numbers of people on beaches, increased pinniped populations, or the occurrence of disease epizootics during the study period. The disease findings in this study are probably not proportionally representative of all problems in the wild pinniped population.

It often was difficult to determine a PDF. In many cases, extensive diagnostic testing and pathological evaluation were not performed due to financial constraints. Health problems not normally a problem in the wild may have resulted from or been aggravated by other diseases, stress of captivity, or shock.

Renal disease suggestive of leptospirosis is a well documented health problem in California sea lions (Medway, 1980; Dunn, 1990). Based on diagnostic testing performed in 1984 (Dierauf et al., 1985) and 1988 (Vedros, pers. comm.), the increase in animals with renal disease was believed



to be related to two separate outbreaks of leptospirosis. In this study, the possible causes of renal disease include dehydration and shock, toxins, or infectious agents including *Leptospira pomona*. Renal disease, believed to be due to *Leptospira pomona*, has been documented in the wild population and is considered an enzootic health problem (McIlhattan et al., 1971; Vedros et al., 1971; Smith et al., 1974; Gilmartin et al., 1976; Dierauf et al., 1985).

San Miguel sea lion virus has been documented in California sea lions (Sawyer, 1976; Smith and Latham, 1978). At The Center, the virus was isolated from the lesions of only one animal (Gage et al., 1990), but 49 other animals with a PDF of renal disease had vesicular lesions on the flippers and mucosa similar to those seen on the confirmed case.

Verminous pneumonia as a disease problem in California sea lions has been well documented (Sweeney, 1974a; Sweeney and Gilmartin, 1974; Dailey, 1986). The true prevalence of verminous pneumonia as a PDF or IF cannot be stated accurately without pulmonary tissue biopsy of every animal with pneumonia. A fecal flotation negative for lungworm larvae does not rule out verminous pneumonia as a disease problem, and a fecal flotation positive for lungworm larvae does not necessarily mean that lungworms are the cause of inflammation in the lungs of the host. As a result, the number of cases of verminous pneumonia and pneumonia of unknown etiology may be inaccurately diagnosed.

More extensive diagnostic procedures are needed to determine the etiology of seizures in California sea lions and northern fur seals. Routine diagnostics performed on seizure animals included complete physical examination, CBC, serum biochemistry, and fecal flotation; all laboratory results appeared normal. Nasal and tracheal cultures for selected viral, bacterial, and fungal infections were negative. Brain histopathology was performed on two animals, and revealed a mild meningoencephalitis in one animal. Two mass

strandings of California sea lions having seizures in southern California occurred in the late 1970's and the early 1980's (Sweeney, pers. comm.); however, no etiologic agents were identified. During 1986 and 1988 large numbers of pinnipeds with seizures stranded in the same location over a short period of time. This is suggestive of a toxic or infectious etiology.

Gastrointestinal disorders in California sea lions may be related to uremia, unknown viral, bacterial, or parasitic pathogens, stress, or shock. Intestinal disorder in all species was likely overdiagnosed on post mortem examination, as post mortem examinations often occurred several hours after death, and histopathology of the intestines was not performed on all dead animals.

Neoplasia in marine mammals has been documented in the past (Mawdesley-Thomas and Bonner, 1970; Mawdesley-Thomas, 1971; Stroud and Roffe, 1979; Brown et al., 1980; Joseph et al., 1986). Neoplasia was not a common finding in stranded pinnipeds during the study period; however, one California sea lion was tentatively diagnosed as having a lymphosarcoma and thirty-two were tentatively diagnosed as having transitional cell carcinoma. A tentative diagnosis of transitional cell carcinoma was based on histopathology in twenty-six of the thirty-two California sea lions. California sea lions diagnosed as having transitional cell carcinoma were euthanized and a complete post mortem examination was performed. It was impossible to determine the site of origin of the tumor, as there were numerous tumors and metastases throughout the body. Histopathological features included multiple metastases throughout the organs of the body, although the primary sites involved were the kidneys and uro-genital tract. Degree of infiltration of organs ranged from epithelial to submucosal to complete with little identifiable tissue remaining. Infiltrates were multilobulated, composed of large nests of neoplastic transitional cells which often were divided into packets by reactive fibrous connective tis-

sue. Neoplastic cells were characterized as large, polygonal, with pale eosinophilic or basophilic cytoplasm, and with large round to oval vesiculated nuclei with prominent nucleoli. The clinical signs of animals with transitional cell carcinoma included a swollen or prolapsed rectum, abdominal distension, and swollen hind flippers.

On admission, elephant seal and harbor seal pups were underweight or emaciated. In the wild, the average birth weight of elephant seals is 30 to 45 kg, and animals are weaned at 130 to 160 kg (McGinnis and Schusterman, 1981). The average normal birth weight of harbor seals is 9 kg, and animals are weaned after 4 to 6 weeks at double their birth weight (Bigg, 1981). It is not known from this study why elephant seal and harbor seal pups were found underweight and apparently abandoned or prematurely separated from their mother. It was not known if gastrointestinal disorders are the PDF or a health problem that occurred during rehabilitation. These animals often succumbed to gastrointestinal disorders during hospitalization; however, further diagnostics would have been necessary to isolate possible etiologies. Unidentified viral or bacterial pathogens, toxic pollutants, stress, shock, diet changes, or unknown changes in the marine environment may have played a role in gastrointestinal disorders. A small number of the gastrointestinal disorders were directly related to sand ingestion.

Rehabilitation of the harbor seal pups was a challenge due to their age and compromised physiologic state on admission. Bilirubin levels between 3.0 mg/dl and 15.0 mg/dl frequently were seen in pups with no ill effects, while pups with levels greater than 15.0 mg/dl were considered to have a fair to guarded prognosis. Hypoglobulinemia seen on admission probably was the result of poor suckling ability or maternal neglect. Hyperbilirubinemia is believed to be a nonpathologic transient condition of the neonatal harbor seal (Dierauf et al., 1984); pups with hypoglycemia likely were suffering from starvation.

It is controversial whether patent ductus

arteriosus (PDA) in premature harbor seal pups is physiologic or pathologic. In this study, PDA was not found in harbor seals estimated to be older than 10 days. The significance of the PDA in the single elephant seal neonate is unknown.

Skin disease in yearling elephant seals has been recognized for many years but is poorly understood. The problem may be a primary bacterial disease or a disorder secondary to an overall debilitating condition such as starvation, or stress and immunosuppression. Most elephant seals with skin disease are underweight (Morris, pers. comm.). Most cases of skin disease in this study were seen during the time of molt or just prior to the molt; however, occasional cases were seen throughout the year. In the wild, yearlings normally molt in the spring. The hair and the surface epidermal layer are both replaced during this process (McGinnis and Schusterman, 1981), which potentially makes the denuded skin susceptible to pathogens. A direct association between the molt and elephant seal skin disease has not been proven.

Parasites were a common finding in stranded pinnipeds. Nematodes, trematodes, and cestodes were found in the lungs, liver, bile duct, stomach, and intestines. A large number of the elephant seals and harbor seals admitted to The Center were neonatal or recently weaned pups; thus, parasites were not a common clinical or pathologic finding in these species.

Eye injury as an IF was a common occurrence in stranded pinnipeds. Sometimes blindness was a transient problem, and sight was recovered as body condition improved. Loss of sight in some cases may have been due to head or eye trauma, or malnutrition. Cataracts were seen in all ages of pinnipeds, and may be congenital, nutritional, traumatic, or due to senility.

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