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Marine Mammals: Introduction

Marine mammals are a diverse group of species that depend on marine environments for survival. In the USA and its territories, the Marine Mammal Protection Act of 1972 protects cetaceans, pinnipeds, sirenians, sea otters, and polar bears as marine mammals. The cetaceans consist of two major groups with different physiology and anatomy—toothed whales (Odontocetes) and baleen whales (Mysticetes). The pinnipeds consist of three major groups—true seals (Phocidae), eared seals (Otariidae), and walruses (Odobenidae). Sirenians (Sirenia) are of a single family that includes manatees and dugongs. The sea otter is a marine member of the Mustelidae, and the polar bear is the only member of the Ursidae that is considered marine.

Few pharmaceuticals or vaccines have been approved for use in marine mammals. Many recommendations can be made based on personal experience or published reports; however, sufficient documentation for official approval is unlikely, and caution is indicated.

Management

The general rule in maintaining marine mammals in captivity is to duplicate their natural environment as closely as possible. Most cetaceans live in marine habitats, although some species migrate into freshwater, and a few are adapted to river habitats. Baikal seals have adapted completely to freshwater. Marine cetaceans should be kept in water with a salinity of 25-35 g/L. Preferably this “salt” water should consist of balanced sea salts; however, captive animals have survived for long periods, in apparent health, in simple sodium chloride solutions. The pH of mid-ocean waters is 8.2-8.3; water for captive marine cetaceans should be maintained as close to this as possible.

Freshwater cetaceans also require water similar to that of their natural habitat. In the USA, the Marine Mammal Protection Act specifies that coliform bacterial counts of water for captive marine mammals must be ≤1000 MPN (most probable number per 100 mL).

The temperature tolerance range of each species of cetacean can be fairly wide, but the optimal temperature range is narrower. Temperature requirements should be evaluated carefully for any cetacean in captivity. Animals kept in the extremes of their temperature tolerance range are more susceptible to environmental and infectious disease. Inappropriately combining different species of animals for display purposes can result in compromises in which one or all species are held at temperatures that jeopardize their well-being.

Good air quality, especially in indoor facilities (10-20 air changes/hr) is as important as good water quality. Photoperiods, light spectral and intensity requirements, sound tolerances, and flight distance requirements are not well established for any cetacean. They undoubtedly vary among species with widely diverse habitats, as well as among individuals. Extremes in any of these factors should be considered detrimental in the absence of specific data for the species in question.

Most pinnipeds live in marine habitats. Their environmental requirements are similar to those of cetaceans except that pinnipeds can “haul out” on land. Although captive pinnipeds can be kept in freshwater if given additional salt in their diet, saltwater pools that meet the specifications listed above for cetaceans are preferred. Most pinnipeds obtain their metabolic water requirements in food and do not require access to freshwater if provided fish with a high-fat content. However, it is common practice to allow pinnipeds access to fresh water. Most pinnipeds are also more tolerant of cold temperatures than excessive heat. The considerations for cetaceans are equally valid for pinnipeds. Pools for captive pinnipeds should provide shelter from wind and some shade. Haul out requirements are different for each species, and some pinnipeds (eg, the northern fur seal) require very specific timing of access to land only at the pupping season.

Sirenians have water requirements similar to those of cetaceans, although the most common sirenian in the USA, the manatee, migrates between marine and freshwater environments seasonally. The manatee does better in captivity if salinity is changed seasonally to match migrations in the wild. It is a warmwater species.

The natural habitat of the sea otter is the Pacific northwest from northern California to Alaska. In captivity, the sea otter thrives best in a cold marine water system. Because the fur of the sea otter is its major protection against hypothermia, the water must be kept completely free of oils and organic material that could mat or damage the coat.

The polar bear naturally lives on arctic and subarctic ice. It has successfully adapted to captivity even in subtropical climates but is more susceptible to disease in warm climates. Polar bears traditionally have been provided with freshwater in captivity. Proper attention to filtration and water quality is beneficial.
**Restraint**

Marine mammals must be restrained for thorough examinations. Trained cetaceans and pinnipeds can be taught behaviors that facilitate examination and collection of diagnostic samples. For these animals, the presence of familiar attendants is important.

For complex procedures or untrained animals, the safest approach to restraining a cetacean is to remove it from the water. Captive enclosures should allow water drainage so that cetaceans can be stranded without the use of nets. As the animal begins to lose buoyancy in the draining water, it should be positioned over thick foam pads to minimize struggling and injury. Nets are an alternative for corralling or catching small cetaceans kept in sea pens or encountered in the wild; however, experienced personnel are required to minimize the risk of drowning or injury to the animal or staff. Netted cetaceans are placed on foam or specially designed stretchers that can be suspended above water level to support and restrain the animal.

Restrain of cetaceans on the foam depends on the procedure to be performed and the animal. Small cetaceans (dolphins) can often be restrained by the weight of 3 or 4 attendants—one person controls the peduncle of the tail fluke and the others apply weight to the animal's body. The pectoral fins should be placed alongside the animal in a natural position to avoid permanent damage. In larger cetaceans (whales), the powerful tail fluke may need to be secured with a loop over the tail stock, taking care to prevent abrasions or lacerations with the restraint loop.

Capturing pinnipeds is generally easier on dry ground, although small ones can be captured in the water with end-release hoop nets. Larger animals should not be netted in water but must be coaxed or driven from the water or have the water drained from their pool. On land, hoop nets can be used on larger animals, but cargo nets, baffle boards, and “come-along” poles also can be helpful. Once captured, small seals can be restrained like most other large mustelids. Hoop nets can be used for removing them from pools. Once they are out of the water, restraint bags, squeeze boxes, or other restraint devices for small wild carnivores can be used. Polar bears are large and dangerous, and manual restraint is not advised.

**Anesthesia**

Physiologic adaptations to diving and marine environments make general anesthesia of cetaceans and pinnipeds difficult. Anesthetic drugs commonly used in other animals often have narrow margins of safety or cause unexpected reactions in marine mammals. Tranquilizers, sedatives, and anesthetics should be administered to marine mammals only by personnel experienced in their use. Specialized anesthetic machines and respirators are required for cetaceans. Sirenians rarely require general anesthesia or tranquilization for treatment. Sea otters can be sedated with diazepam (0.2 mg/kg body wt) or tiletamine-zolazepam (1 mg/kg). Surgical anesthesia can be obtained with higher doses of tiletamine-zolazepam (2 mg/kg) or with halothane and nitrous oxide. Polar bears are routinely immobilized with etorphine, tiletamine-zolazepam, and other agents used IM. The required dose is highly dependent on the individual animal and environment.

Numbers indicate anatomical structures as follows:
1. blowhole  
2. cranium  
3. cerebral hemisphere  
4. cerebellum  
5. bony nares  
6. nasal cavity  
7. glottis  
8. nasopharyngeal sphincter  
9. esophagus  
10. arytenoid cartilage  
11. cricoid cartilage  
12. trachea  
13. eparterial bronchus  
14. tracheal cartilages  
15. thyroid cartilage  
16. epiglottic cartilage  
17. hyoid bone  
18. tongue  
19. oral cavity  
20. palate  
21. oropharyngeal sphincter

Common dolphin, nasopharynx, cross-section. Courtesy of Dr. James McBain.
Environmental Diseases

**Corneal Edema:**

Corneal opacity is frequently seen in captive pinnipeds kept in either freshwater or saltwater; it is also seen in captive cetaceans but is rare in wild animals. It can be due to various environmental problems. Transient cases can be caused by simply moving an animal to freshwater from saltwater, or vice versa. Lack of shade and excessive bright light have been implicated. Unsanitary water conditions (eg, high bacterial loads or overuse of oxidative disinfectants in the water) also have been associated with the disease. Nutritional deficiencies have been suggested as causes, but response to supplementation with vitamin C or A has not been dramatic. The condition is usually self-limiting if the underlying insult to the cornea is removed.

**Corneal Ulcers:**

These occur frequently in captive pinnipeds and cetaceans. They can be the result of direct trauma or the sequelae of unresolved or untreated cases of corneal edema. Diagnosis is by observation of epithelial defects on corneas stained with fluorescein. In trained animals, small lesions can be treated topically. In untrained animals, subconjunctival injections of antibiotics and steroid are required. Extensive lesions benefit from protection by suturing the eyelids. Deep ulcers or lacerations in danger of eroding Descemet’s membrane should be stabilized with a thin methylacrylate patch. As in corneal edema, successful resolution and prevention of recurrence depend on removal of the underlying cause.

**Foreign Bodies:**

Many captive marine mammals develop the vice of swallowing objects dropped into their pools. In cetaceans, the opening to the second compartment of the stomach is small, and foreign objects remain in the first compartment. In pinnipeds, the small pylorus prevents passage of most foreign bodies. Frequently, no clinical signs are evident. On occasion, anorexia, regurgitation, or lethargy may be seen. However, because gastric foreign bodies are a hazard to the animal, all efforts should be made to prevent their ingestion or to remove them once ingested. Sharp objects may cause gastric perforation. Other objects, including coins, can initiate ulceration, which can culminate in perforation. Diagnosis is often made by observing the animal swallow an object. Smaller animals can be radiographed, and small cetaceans can be palpated via the esophagus to establish the presence of foreign bodies. Animals occasionally regurgitate foreign bodies; however, assisted removal is usually indicated. Removal of the objects is usually best performed by gastroscopy, which is also used as a method of diagnostic confirmation. Training animals to retrieve for reward as a displacement to swallowing foreign objects is thought to be beneficial.
**Gastrointestinal Ulcers:**

GI ulcers are a significant problem in captive marine mammals. Ulcers of the first compartment of the cetacean stomach are a common necropsy finding and pose less severe clinical problems than do ulcers of the pyloric region or proximal duodenum. Gastric ulcers in pinnipeds frequently progress to perforation, which results in peritonitis and subsequent death. Gastric ulcers also occur in sirensians. Although ulcers in cetaceans perforate less frequently than in pinnipeds, they should be treated as a serious clinical problem. Although various etiologies, including parasitic damage and increased histamine content of spoiled fish, have been suggested, the disease must be considered primarily an environmental or stress-related condition. Dramatic environmental changes, including changes of personnel or companion animals, can precipitate serious GI ulceration in cetaceans or pinnipeds. Clinical signs include lethargy, partial anorexia, abdominal splinting, pallor, and occasionally regurgitation. Animals with bleeding ulcers show anemia and possibly leukocytosis. Diagnosis generally is based on identification of mammalian RBC in gastric washes; confirmation is by endoscopic visualization of the lesions. Palliative treatment of nonperforating ulcers consists of administration of cimetidine (6 mg/kg, t.i.d.) and alumina-gel-based antacids with or without simethicone, along with frequent small meals. The underlying cause must be identified and corrected for long-term successful treatment. Management of perforating ulcers with resulting peritonitis must include intensive broad-spectrum antibiotic and fluid therapy. As in man, stress-induced GI ulcers are more likely to develop in marine mammals that have previously had an ulcer than in animals that have not.

**Trauma:**

Traumatic lesions (eg, cuts, wounds from gunshots or propeller blades) are common in marine mammals. Propeller injuries are a major problem in manatees, which commonly enter heavily navigated recreational waters in Florida. Traumatic wounds should be cleaned, debrided, and generally allowed to heal as open wounds unless body cavities are breached. Antibiotics should be administered during convalescence to prevent gross infection. Maintenance of good water quality and a high plane of nutrition is beneficial to the healing process. Large wounds frequently heal uneventfully.

**Oil Exposure:**

Exposure of marine mammals to spills of petroleum hydrocarbons is a major concern. Sea otters (Enhydra lutris) are particularly susceptible to the impact of oil spills because of their natural grooming habits and their lack of an insulating blubber layer. Hepatotoxicity, renal toxicity, GI damage, and loss of homeothermic ability are important effects of exposure to petroleum hydrocarbons; however, the most devastating effects are due to direct pulmonary damage from inhalation of volatile hydrocarbons.

Experimental evidence suggests cetaceans and pinnipeds will avoid spills if possible (unlike sea otters) and are relatively resistant to oil from direct skin contact. Ingestion of large quantities of oil by these species is unlikely and although baleen fouling occurs in mysticete whales, it is usually resolved with 24-36 hr. Pinnipeds and cetaceans are susceptible to severe pulmonary damage due to inhalation of volatile hydrocarbons as are other mammals, including man. Efforts to reduce human exposure to hydrocarbons when dealing with oil-contaminated animals must be a top priority. Treatment of exposed animals includes removal of oil from both the skin, using mild detergents (eg, 2% New Dawn), and GI system (activated carbon gavage), along with physiologic supportive therapy. It is critical to recognize that capture, transport, and holding stresses appear to lower the threshold of hydrocarbon toxicity in these animals.

**Nutrition and Nutritional Diseases**

Generally, captive animals fed a diet that is solely or primarily fish are provided dead fish that have been frozen. The logistics and difficulty in providing this fish can lead to some special nutritional concerns. All fish are not of equal nutritional value; diets consisting of a single species of fish are unlikely to provide balanced nutrition for any animal. Similarly, one diet will not serve all piscivores equally. Only fish suitable for human consumption should be fed. See also nutrition: exotic and zoo animals, Nutrition: Exotic And Zoo Animals: Introduction. Frozen fish pose the added risks of improper storage and thawing; these procedures must be monitored carefully. Feed fish should be held at <30°F (-1°C) to reduce deterioration of their nutritional value through oxidation of amino acids and unsaturated lipids. Dehydration of frozen fish can also be a problem for animals that obtain their water from their food. Fatty fish should not be stored >6 mo. Few fish, with the possible exception of capelin, should be stored >1 yr. To retain optimal vitamin content and reduce moisture loss, frozen fish should be thawed in air under refrigeration. Thawing in water leaches away water-soluble vitamins. Thawing at room temperature encourages bacterial growth and spoilage.
The energy requirements of marine mammals vary with age, environmental temperatures, and condition. Young growing dolphins and smaller pinnipeds generally require 9-15% of their body wt in high-quality fish per day. Older animals may need only 4-9% of their body wt for maintenance. Larger species (whales, elephant seals) generally require less food (2-5% of body wt) as adults.

Sirenians thrive on a diet of hydroponic grass and various lettuces and vegetables, supplemented with high-protein monkey chow, carrots, bananas, and multivitamin-mineral supplements used particularly to balance calcium/phosphorus ratios. It is thought that sirenians ingest considerable animal protein incidentally during grazing in the wild. Intake requirements have been estimated at 7-9% of body wt daily. Sirenians are generally fed several times a day to accommodate their grazing feeding pattern.

Sea otters are usually fed diets consisting of various invertebrates (echinoderms, molluscs, occasional crustaceans) and fish. Adult animals require ~25-30% of their body wt in food each day.

Polar bears in the wild have high lipid diets, particularly in winter when they subsist heavily on seals. They are considered to have an exceptional dietary requirement for vitamin A, and some dermatologic conditions respond to daily supplementation of 20,000-1,000,000 IU in the diet. Polar bears are commonly fed large amounts of fish in captivity.

Neonatal Nutrition:

Young marine mammals are frequently encountered in strandings. Often they are not weaned and must be fed a diet resembling their dam's milk. In captivity, neonates may be abandoned by their parents and require artificial rearing. The milk of marine mammals has a high lipid content. Most species are carbohydrate intolerant, and neonates fed formulas with carbohydrates develop severe, life-threatening, bacterial gastroenteritis. Most neonatal marine mammals also require immense caloric density in replacement milks.

Phocid and otarid seals can be reared on the same formula, made by grinding 340 g of headless, tailless herring to a paste in 100 mL of water. This is put through a ricer to produce a fish mash, which is supplemented with a mixture of 150 mL lactated Ringer's, 1¼ tsp (8.3 g) sodium chloride, 400 mg thiamine, 400 IU vitamin E, ⅛ tsp (1.3 g) calcium gluconate powder, 250 mg vitamin C, 1 tsp (5 mL) safflower oil, 1 tsp (2.5 g) lecithin with 0% carbohydrates, and 1 powdered human multivitamin tablet. The mix is then blended with 200 mL of heavy whipping cream that has been treated with lactase enzymes at least several hours before. This formula can be stored refrigerated for up to 24 hr.

Pinniped pups should be fed every 4 hr in their first week of life; gradually, the amount of formula fed should be increased and the feedings dropped to five per day. Harbor seal pups should be tube fed until 2-3 wk old before they are weaned to small pieces of fish. Elephant seal pups require tube feeding until they are 4 wk old, when weaning can begin. California sea lion pups can be force fed fish as early as 4 wk of age and be free feeding by 6 wk. Neonatal walruses have been reared on formulas with a whipping-cream base extended with ground molluscs (clams) rather than fish. They also seem to tolerate carbohydrates reasonably well. Walruses have a much longer nursing period than other pinnipeds.

Neonatal cetaceans have longer nursing periods than pinnipeds. Success at bottle-rearing has been minimal. The fat content of cetacean milks varies considerably: bottle-nosed dolphin milk is ~17% fat (half that of most pinniped milks); beluga whale milk, 27%; harbor porpoise milk, 46%; and mysticete blue whale milk, 42%. Formulas similar to the pinniped formula, adjusted for fat content with oils, should be successful in neonatal cetaceans if the logistics of delivery are solved.

Neonatal sirenians begin to nibble sea grasses shortly after birth but may continue to nurse up to 18 mo. They can be reared on artificial milks with early weaning. Neonatal sea otters also have been reared successfully from birth on artificial formulas. Neonatal polar bears are extremely altricial and are a challenge because of an apparently immature immune system. Polar bear milk is high in fat (31%) and contains minimal lactose. Polar bears have been successfully reared on formulas with a whipping cream or oil base.

Thiamine Deficiency:

This can be seen in any piscivorous animal. Thiamine in the food is destroyed by the activity of thiaminase enzymes or antithiamine substances in the fish being fed. These active enzymes also destroy supplemental thiamine that is placed in fish if it sits for long periods before feeding. Clinical signs of thiamine deficiency are primarily of CNS disturbances. Affected animals may show anorexia, regurgitation, or ataxia. The condition can progress to seizures, coma, and death.

Animals with clinical signs of thiamine deficiency respond rapidly to IM injection of thiamine hydrochloride (up to 1 mg/kg body wt), followed by oral supplementation. Control usually involves supplemental thiamine at 25 mg/kg food, preferably administered 2 hr before a main feeding.
Vitamin E Deficiency (Steatitis, White Fat Disease):

The antioxidant properties of vitamin E are believed to play an important role in maintaining the integrity of cellular membranes. Oxidative processes during the storage of fish destroy vitamin E and other antioxidants. Steatitis has been induced experimentally in phocid seals, and relationships between vitamin E deficiency and hyponatremia are suspected. Captive piscivores are commonly supplemented PO with vitamin E at a rate up to 100 mg/kg of feed, which generally maintains high serum levels of the vitamin. This does not appear necessary if fish is properly stored and thawed.

Hyponatremia (Salt Deficiency, Addison’s Disease):

Hyponatremia in pinnipeds is closely related to adrenal exhaustion and development of Addison’s disease, which links the syndrome to environmental stressors rather than to a simple primary salt deficiency. It is most common in pinnipeds maintained in freshwater exhibits but can be seen in animals kept in saltwater. It is more common in phocid seals but occurs in otarids and other marine mammals. Signs include periodic weakness, anorexia, lethargy, incoordination, tremor, and convulsions. Serum sodium levels can fall to <140 mEq/L. Severely affected animals may collapse in Addisonian crisis, which can be fatal.

Emergency therapy consists of sodium chloride infusion and replacement corticosteroids. Long-term management of advanced cases requires mineralocorticoid supplementation in conjunction with oral sodium chloride supplements and periodic monitoring of serum sodium levels. Control consists of provision of saltwater pools or supplementation of sodium chloride (3 g/kg food) in the diet of captive pinnipeds maintained in freshwater pools. Animals on salt supplementation should have continuous access to freshwater.

Histamine Toxicity (Scombroid Poisoning, Mackerel Poisoning):

Scombroid fish (mackerel, tuna) and other dark-fleshed fish have a short shelf life, even when frozen at low temperatures. A complex of substances, including histamine formed by bacterial decarboxylation of the large amount of histidine found in the flesh of the fish, is responsible for the signs seen in affected marine mammals. The toxicity can also occur with nonscombroid fishes, including poorly handled herring, anchovies, or pilchard. It is most common in pinnipeds but is seen in other marine mammals. Clinical signs include anorexia; lethargy; a red, inflamed mouth or throat; and conjunctivitis and increased lacrimation. Occasionally vomiting, diarrhea, pruritus, urticaria, or postures indicative of abdominal pain are seen. Antihistamines, including cimetidine, may provide symptomatic relief, but the condition is generally self-limiting and the animal begins feeding within 2-3 days. In more severe or acute cases, epinephrine is effective in counteracting the histamine reaction. Cortisone and diphenhydramine hydrochloride can be beneficial in the face of respiratory difficulty. Control consists of avoiding scombroid fish in the diet or careful attention to their quality, storage, and handling when used.

Bacterial Diseases

Actinomycetes:

Nocardiosis is commonly reported in debilitated marine mammals. It has been diagnosed in the pilot whale, harbor porpoise, killer whale, false killer whale, spinner dolphin, and leopard seal. Infections due to Actinomyces spp also have been diagnosed in the bottlenosed dolphin. Successful treatment has not been reported, but the treatment of choice would be sulfonamide therapy, except in killer whales, in which sulfonamides are contraindicated.

Clostridial Myositis:

Severe myositis due to infections with Clostridium spp has been diagnosed in captive killer whales, pilot whales, bottlenosed dolphins, California sea lions, and manatees. All marine mammals are probably susceptible. The disease is characterized by acute swelling, muscle necrosis, and accumulations of gas in affected tissues, accompanied by a severe leukocytosis. Untreated, it can be fatal. Diagnosis is based on detection of gram-positive bacilli in aspirates of the lesions and is confirmed by anaerobic culture and identification of the organism. Treatment includes systemic and local antibiotics, surgical drainage of abscessed areas, and flushing with hydrogen peroxide. Commercially available, inactivated clostridial bacterins are used routinely in some facilities, although efficacy in marine mammals has not been studied. Botulism has been reported in captive California sea lions (Zalophus californianus) during an endemic outbreak of the disease in waterfowl. Affected animals stopped eating and appeared unable to swallow several days before dying.
Pneumonia:
The chief cause of death in captive marine mammals is believed to be pneumonia. It is not common in polar bears. Most cases of marine mammal pneumonia have significant bacterial involvement, and most organisms cultured from terrestrial species have been identified in marine mammals. Pneumonia often can be considered the result of mismanagement. Marine mammals require good air quality, including high rates of air exchange at the water surface in indoor facilities. Tempered air or acclimation to cold temperatures is also important to prevent lung disease, even in polar species. Animals acclimated to cold temperatures are usually quite hardy; however, sudden transition from warm environments to cold air, even with warmer water, can precipitate fulminating pneumonias, particularly in nutritionally or otherwise compromised animals. Clinical signs include lethargy, anorexia, severe halitosis, dyspnea, pyrexia, and marked leukocytosis. The disease can progress rapidly. Diagnosis is usually based on clinical signs and confirmed by response to therapy. Treatment consists of correction of environmental factors and intensive antibiotic and supportive therapy. The initial antibiotic is usually broad-spectrum, commonly cephalaxin (40 mg/kg, t.i.d. or q.i.d.); adjustments are based on cultures and sensitivities from blowhole or tracheal samples.

Erysipelas (Diamond Skin Disease):
Erysipelas can be a serious infectious disease of captive cetaceans and pinnipeds. The organism, *Erysipelothrix rhusiopathiae*, which causes erysipelas in pigs and other domestic species, is a common contaminant of fish. A septicemic form of the disease in marine mammals can be peracute or acute; affected animals die suddenly either with no prodromal signs or with sudden depression, inappetence, or fever. A cutaneous form that causes typical rhomboidal skin lesions is usually a more chronic form of the disease. Animals with this form usually recover with timely antibiotic treatment.

Necropsy of peracute cases generally fails to reveal grossly discernible lesions other than widespread petechiation. Diagnosis is based on culture of the organism from the blood, spleen, or body cavities. Arthritis has been found in animals that have died with the chronic form of the disease. Treatment of the peracute and acute forms has rarely been attempted because the absence of prodromal signs obscures the diagnosis. Animals with the dermatologic form usually recover with administration of penicillins, tetracyclines, or chloramphenicol, and supportive treatment.

Control seems primarily related to the provision of high-quality fish that is properly stored and handled. Vaccination is controversial and not implemented in many aquaria because of problems with the bacterin. Vaccine breaks are common. Vials of killed erysipelas bacterin should be cultured for surviving organisms before use in marine mammals. Modified live bacterins should be avoided for the initial vaccination. Fatal anaphylaxis can occur on revaccination. For this reason, some vaccination programs have been reduced to one-time administration even though antibody titers fall below the presumed effective level.

If cetaceans are to be revaccinated, sensitivity tests should be performed by injecting a small amount of bacterin submucosally on the lower surface of the tongue. Hypersensitive animals develop swelling and redness at the injection site within 30 min. Because the vaccine is extremely irritating, no more than 3-5 mL should be used at any one site, even in nonsensitive mammals. A long needle (≥2 in. [5 cm]) should be used to assure that the vaccine is deposited in the muscle and not between muscle and blubber, or a sterile abscess can result. Bacterin should be administered in the dorsal musculature anterior and lateral to the dorsal fin. Administration posterior to the dorsal fin can result in a severe tissue reaction, immobilizing the animal for several days. To maintain high antibody titers, a booster after 6 mo and annual revaccination are required.

Leptospirosis:
This has been diagnosed in otarid pinnipeds and bears. In seals, the disease is characterized by depression, reluctance to move, polydipsia, and pyrexia. It may also cause abortions and neonatal deaths in California sea lions and Northern fur seals. Lesions include a severe, diffuse, interstitial nephritis, with renal tubules packed with
spirochetes. The gallbladder may contain inspissated black bile, but hepatitis may not be apparent grossly. Hyperplasia of Kupffer's cells, erythrophagocytosis, and hemosiderosis are seen histologically. Gastroenteritis can be a feature. Antibodies to various Leptospira serovars (canicola, icterohaemorrhagiae, autumnalis, and pomona) have been identified in affected animals by fluorescent antibody techniques; however, only L pomona has been isolated from marine mammals. Treatment in pinnipeds is similar to that in dogs (Leptospirosis in Dogs). Control in captive animals requires serologic examination of new animals during quarantine. Captive animals can be vaccinated in endemic areas. Leptospira can infect man, and appropriate precautions should be taken.

**Tuberculosis:**

Marine mammals are susceptible to various mycobacteria. Unconfirmed tuberculosis has been reported in a stranded, wild bottle-nosed dolphin in the Mediterranean, and indirect evidence points to Mycobacterium bovis being possibly endemic in free-ranging otarids off the coast of Australia; otherwise, mycobacteriosis has been a disease of captivity. Pinnipeds, cetaceans, and sirenians have developed disease due to M bovis, M smegmatis, M fortuitum, M chelonae, and M marinum. Cutaneous and systemic forms are seen. There are strong indications that immunosuppression may be involved in the development of infections by the atypical mycobacteria. Intradermal testing with high concentrations of bovine or avian purified protein derivative (PPD) tuberculin can be used to screen exposed animals; however, anergy occurs. In pinnipeds, injections in the webbing of the rear flippers should be read at 48 and 72 hr. ELISA screening has identified antibodies in seals but requires further evaluation before it can be considered a screening test. Diagnosis is made by culture and identification of the organism from lesion biopsies, tracheal washes, or feces. Mycobacteriosis in marine mammals is an emerging disease and is probably of public health significance. (See also Tuberculosis: Introduction, Mycobacterial Infections other than Tuberculosis.)

**Miscellaneous Bacterial Diseases:**

Marine mammals are probably susceptible to the entire range of pathogenic bacteria. Bacteria other than those discussed above cause significant disease in marine mammals. Pasteurella multocida has caused several outbreaks of hemorrhagic enteritis with depression and abdominal distress leading to acute death in dolphins and pinnipeds. It has also been reported to cause pneumonia in pinnipeds. In dolphins, P haemolytica has been incriminated in hemorrhagic tracheitis that responded to chloramphenicol therapy. Plesiomonas shigelloides has been responsible for gastroenteritis in harbor seals. Pseudomonas pseudomallei has caused serious fatal outbreaks of disease in various marine mammals in captivity in the far east. Salmonella spp have caused fatal gastroenteritis in manatee and beluga whales. Staphylococcal septicemia has caused the death of a dolphin with osteomyelitis of the spine (pyogenic spondylitis). Another case of intradiskal osteomyelitis, due to Staphylococcus aureus, was treated successfully with a prolonged course of cefazolin sodium and cephalaxin. Staphylococcus aureus also has been incriminated in a fatal pneumonia in a killer whale. Vibrio spp infect slow-healing wounds of cetaceans managed in open sea pens.

**Mycotic Diseases**

Captive marine mammals seem particularly prone to fungal infections (see also Fungal Infections: Introduction). There is no definitive evidence of horizontal transmission of most of these; they appear to be secondary to stress, environmental compromise, or other infectious disease. Some systemic mycoses have distinct geographic distributions. Diagnosis is based on clinical signs and confirmed by identification of the organism in biopsy or, preferably, culture. Wet mounts in lactophenol or cotton blue may render an immediate diagnosis with some of the morphologically distinct fungi. Tissue smears cleared in warm 10% potassium hydroxide can be examined to identify characteristic fruiting bodies or hyphae.

Topical medication of pinnipeds for dermatophytosis is feasible. Smaller cetaceans can be kept out of water in a sling for 2-24 hr, provided areas of the body not being treated are kept moist. Otherwise, systemic therapy is used.

**Aspergillosis:**

Fatal pulmonary aspergillosis has been diagnosed in bottle-nosed dolphins and a California sea lion. Cutaneous aspergillosis has been seen in gray seals with concomitant mycobacteriosis. The respiratory form has been a postmortem diagnosis. Cutaneous lesions respond to topical povidone iodines with ketoconazole therapy (10 mg/kg, PO, daily).
Candidiasis:
This common mycotic disease in captive cetaceans occurs secondary to stress, unbalanced water disinfection with chlorines, or indiscriminate antibiotic therapy. Candidiasis is also reported in pinnipeds. In cetaceans, the lesions usually are found around body openings. At necropsy, esophageal ulcers are often found, particularly in the area of the gastroesophageal junction. In phocid pinnipeds, inflammation at the mucocutaneous junctions, particularly at the commissures of the mouth and around the eyes, anus, and vulva, is the common presentation. Diagnosis is based on identification of the yeast in cultures or biopsy. Candidiasis generally responds well to ketoconazole (6 mg/kg, PO, daily) along with correction of any environmental deficits. One anecdotal report suggests a possible toxic drug reaction to ketoconazole in a northern elephant seal (Mirounga angustirostris). Early detection and treatment is usually successful. Another opportunistic yeast, Cryptococcus neoformans, has been diagnosed in fatal advanced pulmonary disease in a bottlenosed dolphin.

Dermatophytosis:
Mycotic dermatitis due to Trichophyton spp or Microsporum canis generally responds to topical povidone-iodine or oral griseofulvin (or both).

Streptothricosis (Dolphin Pseudopox, Cutaneous Dermatophilosis):
Streptothricosis (Dermatophilus congolensis), a subcutaneous mycosis, has been reported in pinnipeds. It must be distinguished from sealpox. Simultaneous infections of streptothricosis and pox have been recorded in sea lions. Cutaneous streptothricosis usually manifests as sharply delineated nodules distributed over the entire body and usually progresses to death. Diagnosis is based on demonstration of the organism in biopsies or culture. Treatment with systemic antifungal drugs has been unsuccessful. Sporothrix schenckii, the cause of another subcutaneous mycosis, has been reported in a Pacific white-sided dolphin.

Systemic Mycoses:
The systemic mycoses of marine mammals are a zoonotic risk, and precautions should be taken to prevent infection when handling dead and diseased animals. Keloidal blastomycosis (Lobo’s disease) has been reported only in man and dolphins. Blastomycosis has caused fatal disease in California sea lions, a Stellar sea lion, northern fur seals, and polar bears. Lobomycosis due to infection with Loboa loboi is seen in the Atlantic bottlenosed dolphin. Fatal systemic histoplasmosis has been reported in a captive harp seal. Coccidioidomycosis has been found in the California sea lion and sea otter. Blastomycosis has been successfully treated with intensive management including 70 days of itraconazole (3.5 mg/kg, PO, daily) combined with antibiotic and supportive therapy when indicated.
**Zygomycetoses:**

*Mucor* spp and *Entomophthora* spp have caused fatal disease in the bottle-nosed dolphin and harp seal. *Fusarium* dermatitis occurs in California sea lions and gray seals. These should be considered diseases of debilitated animals; the underlying cause of the low host resistance to these opportunist infections must be corrected if therapy is to be successful. Amphotericin B is the therapy of choice for zygomycete infections, but newer imidazoles warrant consideration.

**Parasitic Diseases**

Marine mammals are susceptible to all of the major groups of parasites, including various nematodes, trematodes, cestodes, mites, lice, and acanthocephalans. Clinical experience with many of these is limited, while others are commonly seen in recently captured specimens.

**Acarasis:**

Nasal and lung mites are found in phocid and otarid seals. Lung mites cause rattling coughs. Nasal mites cause nasal discharge but apparently little discomfort. Diagnosis is made by identifying the mite in nasal secretions or sputum. The life cycles of these mites are unknown. Infections have been cleared rapidly with two injections of ivermectin at 200 µg/kg, 2 wk apart. Treatment of infected animals eliminates the problem in captive enclosures without environmental treatment. Mites have been associated with large, roughened lesions of the laryngeal area of cetaceans, but their overall significance or treatment is unknown.

Demodectic mange has been diagnosed in California sea lions. Nonpruritic, alopecic lesions with hyperkeratosis, scaling, and excoriation occur on the flippers and other body surfaces that contact the substrate. Diagnosis is made by deep skin scrapings and identification of the mite. Secondary bacterial infection that results in pyoderma occurs in chronic cases. Treatment is the same as in dogs ( *Mange in Dogs and Cats*). Predisposing factors in pinnipeds are unknown. The mites are not readily transmitted among contact animals.

Heavy infestations of sucking lice are common in wild pinnipeds and can cause severe anemia. The lice can be seen grossly and are readily transmitted. They are highly sensitive to chlorinated hydrocarbon insecticides. Rotenone powder is also effective. The affected animal must be removed from the water, allowed to dry before being dusted, and kept out of the water ≥12 hr. Treatments must be repeated in 10–12 days. Animals in captivity can be freed of parasites, provided that no new sources of infestation are introduced.

**Lungworm:**

Lungworms are common in all pinnipeds. Sea lions have *Parafilaroides decorus*, while true seals are usually parasitized by *Otostrongylus circumlitus*. The latter parasite is also found in the hearts of some phocids; however, it does not produce a microfilaremia. There are at least four species of lungworms in various cetacean hosts, including *Halocercus lagenorhynchi*, which has caused prenatal infections in the Atlantic bottle-nosed dolphin (*Tursiops truncatus*).

Lungworm infection can be diagnosed by examination of feces or bronchial mucus. Anorexia, coughing, and sometimes blood-flecked mucus are the first signs of pulmonary parasitism. Treatment of *P decorus* infection consists of mucolytic agents administered intratracheally, antibiotics to treat any concomitant bacterial pneumonia, and levamisole phosphate (15 mg/kg) daily for 5 days. Treatment of *O circumlitus* has been by intratracheal administration of levamisole phosphate (5 mg/kg) daily for 5 days. Cetacean lungworms probably are also susceptible to levamisole and ivermectin; however, the sudden deaths of two beluga whales (*Delphinapterus leucas*) injected IM with levamisole phosphate suggests this drug administered by that route may be contraindicated. A percentage of pinnipeds also show neurologic reactions to IM injection of levamisole, and PO or SC administration has been recommended.

Lungworm infections often remain asymptomatic for long periods; when an animal becomes debilitated for other reasons, clinical signs may appear. In captivity, lungworm infections are usually self-limiting if larvae are not introduced in fresh fish intermediate hosts. Feeding frozen fish prevents reinfection.
Heartworm:

Heartworms of the genus *Dipetalonema* are a common necropsy finding in pinnipeds but have not been reported in cetaceans or sirenians. Phocid seals are affected by *D. spirocauda*, and otarids are infected subcutaneously by *D. odendhali*. Both groups of pinnipeds can be infected with the canine heartworm *Dirofilaria immitis* in endemic areas; however, phocid seals are abnormal hosts. Dirofilariasis is diagnosed by identifying microfilariae in the blood. Transmission is by the same mosquitoes that bite dogs. A high-dose levamisole phosphate regimen (40 mg/kg, daily for 1 wk) has successfully cleared infection in captive pinnipeds, with the advantage of oral administration. Prevention in endemic areas has been successful with daily administration of diethylcarbamazine at 3.3 mg/kg body wt during the mosquito season. This drug is also effective as a larvicide. Preventive regimens using ivermectin (canine doses) have been used successfully in pinnipeds. (See also *Heartworm Disease: Introduction*.)

Other Nematodes:

The Anasakidae are pathogenic nematodes found in the stomach of marine mammals. Granulomas form at their attachment sites and can lead to blood loss, ulceration, and ultimately perforation and peritonitis. Raw fish is most often incriminated as the source of infection. Infections with *Contracaecum* spp are common in wild cetaceans and pinnipeds. Polar bears in captivity are prone to heavy ascarid infection. Gastric nematodes can be successfully treated with oral dichlorvos (30 mg/kg), fenbendazole (11 mg/kg), or mebendazole (9 mg/kg) given twice, 10 days apart. Ivermectin may be considered.

Hookworms (*Uncinaria* spp) are found in pinnipeds. Severe infections are known only in the fur seals. Newborn pups are infected via colostrum. Disophenol (12.5 mg/kg) or ivermectin (100 µg/kg) injected SC are effective against these parasites. Many species of a large spirurid nematode (*Crassicauda* spp) infect the cranial sinuses, major vessels, kidneys, and mammary gland ducts of cetaceans. Successful treatments are not documented but are potentially possible with systemic parasiticides.

Cestodiasis:

*Diphyllobothrium pacificum* is commonly found in sea lions, and heavy infection is thought to cause intestinal obstruction. Niclosamide (160 mg/kg) or praziquantel (10 mg/kg) are effective treatments. Other cestodes commonly seen include *D. lanceolatum* in phocid seals, *Diplogonoporus tetrapterus* in all pinnipeds, and *Tetrabothrium forsteri* and *Strobilocephalus triangularis* in cetaceans. Cetaceans are also commonly infected with subcutaneous tapeworm cysts throughout the blubber. These usually are the larval forms of tapeworms of sharks.

Trematodiases:

Fluke infections are common in pinnipeds and cetaceans; *Nasitrema* spp are found in the nasal passages and sinuses of cetaceans. Ova of these trematodes have been associated with necrotic foci in the brains of animals showing behavioral aberrations and have been incriminated as a cause of localized pneumonia in cetaceans. Infections are often accompanied by halitosis and brown mucus around the blowhole and occasionally by coughing.
Diagnosis is based on demonstration of typical operculated trematode ova in blowhole swabs or feces. Treatment with oral praziquantel (10 mg/kg, two treatments 1 wk apart) is usually effective. Reinfection can be prevented by not feeding fresh or live fish.

*Zalophotrema hepaticum* is an important hepatic trematode of the California sea lion; it causes biliary hypertrophy and fibrosis of the liver. Signs are usually seen in adults and include icterus, lethargy, and anorexia. Bilirubinemia and increased serum hepatic enzymes are common. Diagnosis is based on identification of trematode ova in the feces. Treatment with bithional at 20 mg/kg body wt has been successful.

Various other trematodes infect the stomach, intestines, liver, pancreas, and other abdominal organs of marine mammals. Pancreatic fibrosis due to trematodiasis is a common necropsy finding.

**Coccidiosis:**

Coccidia (*Eimeria phocae*) have been found in harbor seals with a fatal, bloody diarrhea. These coccidia are probably susceptible to anticoccidial drugs used against other species, e.g., amprolium.

There have been no reports of coccidiosis in other marine mammals. (See also Coccidiosis: Introduction, Coccidiosis of Cats and Dogs, Coccidiosis.)

**Viral Diseases**

**Adenovirus:**

Adenovirus has been isolated from a sei whale and bowhead whales and in livers from six young stranded California sea lions (*Zalophus californianus*) with hepatitis. No disease was noted in the cetaceans. Pinnipeds developed weakness, emaciation, photophobia, polydipsia, abdominal splintering, blood-tinged diarrhea, and eventually posterior paresis; a relative lymphopenia and monocytosis were seen. All pinnipeds developed pneumonia and died within 28 days.

The most prominent histologic lesion in all cases was hepatic necrosis. Massive coagulation necrosis without apparent zonal distribution occurred in some animals. Basophilic intranuclear inclusions in hepatocytes or granular amphophilic intranuclear inclusions in Kupffer’s cells were seen. No evidence of adenovirus was detected in the lungs. Adenovirus from California sea lions is not known to cause disease in man.

**Caliciviruses (San Miguel Sea Lion Virus):**

Caliciviruses have been isolated from otarid seals, walrus, Atlantic bottle-nosed dolphin (*Tursiops truncatus*), and opaleye fish (*Girella nigricans*). The marine caliciviruses appear to be serotypes of vesicular exanthema of swine virus (VESV, Vesicular Exanthema Of Swine: Introduction). Several species of mysticete cetaceans have antibodies to different serotypes of VESV. By 4 mo of age, most California sea lions have neutralizing antibodies to one or more of the serotypes. Opaleye fish are probably responsible for the endemic status of caliciviruses in marine mammals that inhabit the coastal waters of California. To date, infections have not been diagnosed in marine mammals in the Atlantic ocean.

The most consistent lesion in marine mammals is skin vesicles. In pinnipeds, the vesicles are most prevalent on the dorsal surfaces of the fore flippers. In dolphins, vesicular lesions have been seen in association with “tattoo” lesions and old scars. Vesicles are 1 mm to 3 cm in diameter. They usually erode and leave shallow, fast-healing ulcers, but occasionally vesicles regress and leave plaque-like lesions. Skin lesions usually resolve without treatment. Infection may cause premature parturition in pinnipeds. Affected pups have interstitial pneumonitis and encephalitis and fail to thrive.

Inoculation of marine caliciviruses into pigs causes vesicular lesions identical to those seen in vesicular exanthema. In man, heavy exposure to marine caliciviruses can result in neutralizing antibodies, and localized lesions in an accidental laboratory exposure as well as isolation of calicivirus from a clinically ill primate indicates that these viruses should be handled carefully.
**Herpesvirus:**

Herpesviruses have been isolated from neonatal harbor seals (Phoca vitulina), a California sea lion (Zalophus californianus), and a gray seal (Halichoerus grypus). Herpesvirus-like particles have been demonstrated in skin lesions from beluga whales (Delphinapterus leucas). Herpesvirus-like lesions occur in a wide variety of other pinnipeds and cetaceans.

Harbor seals develop nasal discharge, inflammation of the oral mucosa, vomiting, diarrhea, and fever, followed by coughing, pneumonia, anorexia, and lethargy that can result in death in 1-6 days. Morbidity can approach 100% in stressed seals in crowded conditions; mortality is ~50%. The incubation period appears to be 10-14 days.

In the California sea lion and gray seal, recurring circumscribed areas of alopecia ~0.5 cm in diameter were the primary sign of infection. Systemic disease, including pneumonia, can occur. Herpetic lesions in beluga whales are generally circular, up to 2 cm in diameter, and may appear slightly depressed with a target appearance or be raised and proliferative. The centers of some lesions are necrotic or may contain verrucous growths. Systemic infections have not been documented in the whales.

Necropsy findings include interstitial pneumonia, hepatomegaly with massive coagulation necrosis, and small erosions of the oral mucosa and skin. Intranuclear inclusions may be seen in biopsies of early skin lesions. In seals, interstitial pneumonia caused by herpesvirus must be distinguished from bronchial pneumonia caused by influenza virus. Other infectious organisms (eg, bacteria and parasites) may complicate herpesvirus pneumonia. Herpesvirus hepatitis must be differentiated from adenovirus hepatitis, which has intranuclear inclusion bodies.

In systemic infection, therapy is supportive. In a documented epidemic, oral acyclovir did not eliminate the infection but appeared to significantly shorten clinical signs in primary infections. Vaccination with 1 mL of trivalent poliovirus vaccine to control recrudescence of suspected herpesvirus lesions has been used with some success; although it reduced the severity of recrudescence in seals, there is a potential public health risk because live poliovirus may be shed after vaccination. Stress and immunosuppression are associated with recrudescence of latent infections. There is no evidence that the herpesviruses of pinnipeds or cetaceans are zoonotic.

**Influenza Virus:**

Two different influenza A viruses have been isolated from stranded harbor seals (Phoca vitulina) and two other subtypes from a stranded pilot whale (Globicephala melaena). Infection is probably common. Clinical signs in seals are dramatic. Even well-nourished animals become weak, incoordinated, and dyspneic, with occasional white or bloody nasal discharge. Swollen necks due to facial trapping of air escaping through the thoracic inlet is common. The single pilot whale had difficulty maneuvering, was extremely emaciated, and was sloughing skin. The incubation period during epidemics is ≤3 days. Many factors probably contributed to the explosive nature of the epidemics in the harbor seals. High population densities and unseasonably warm temperatures contribute to high mortality.

In the seals, pneumonia was characterized by necrotizing bronchitis and bronchiolitis and hemorrhagic alveolitis. In the pilot whale, the lungs were hemorrhagic and a hilar node was greatly enlarged. For differential diagnosis, see herpesvirus, above.

The virulence of epidemics has precluded attempts at intensive supportive care. Persons whose eyes were contaminated while doing necropsies, or by being sneezed on by affected seals, have developed keratoconjunctivitis within 2-3 days, and identical virus has been recovered. All affected people have recovered completely within 7 days without developing any antibody titers, which suggests that the reaction was local, as occurs with Newcastle disease virus.

**Morbillivirus:**

Phocid seals are susceptible to canine distemper virus (Canine Distemper: Introduction) and to a Morbillivirus (phocid Morbillivirus), which is closely related to but distinct from canine distemper virus. Generally, young seals are affected and show depression, anorexia, crusting conjunctivitis, nasal discharge, and dyspnea. Pneumonia develops and mortality can be high in previously unexposed animals. Outbreaks in wild harbor seals have been extensive in the North Sea. Seals vaccinated with canine distemper vaccine have been rendered immune to challenge with the virus (suspension of organ material) obtained from dead wild seals. Deaths of seals in Lake Baikal, Russia in 1987 were due to canine distemper virus.

A delphinid distemper virus (cetacean Morbillivirus), closely related to rinderpest (Rinderpest: Introduction) and peste des petits ruminants (Peste Des Petits Ruminants: Introduction), has been implicated in the deaths of harbor porpoises (Phocoena phocoena) and common dolphin (Delphinus delphis) off the coast of the UK, striped dolphins (Stenella coeruleoalba) in the Mediterranean, and bottle-nosed dolphins (Tursiops truncatus) in the western Atlantic and Gulf of Mexico.
**Poxvirus:**

Poxvirus has been identified morphologically in skin lesions of both captive and free-ranging pinnipeds and cetaceans. Lesions in California sea lions (Zalophus californianus), harbor seals (Phoca vitulina), and gray seals (Halichoerus grypus) probably are due to parapoxviruses; lesions in South American sea lions (Otaria byronia) and Northern fur seals (Callorhinus ursinus) probably are not. An orthopox virus has been isolated from pox-like lesions on a gray seal (H. grypus). An unclassified poxvirus has also been associated with skin lesions in Atlantic bottlenosed dolphins (Tursiops truncatus) and in a stranded Atlantic white-sided dolphin (Lagenorhynchus acutus).

Outbreaks typically occur in postweanling pinnipeds recently introduced into captivity. The incubation period is 3-5 wk. A break in the epithelial surface may be required to start an infection. Lesions can recur. Small, cutaneous, raised nodules (0.5-1 cm in diameter) occur on the head, neck, and flippers of affected pinnipeds. These may increase to 1.5-3 cm in diameter during the first week and may ulcerate or develop satellite lesions during the second week. After the fourth week, lesions begin to regress, although nodules are reported to persist as long as 15-18 wk. Areas of alopecia and scar tissue may remain after resolution.

Cutaneous poxvirus infections in cetaceans can occur on any part of the body but are most common on the head, pectoral flippers, dorsal fin, and tail fluke. They range from ring or pinhole lesions to black, punctiform, stippled patterns (“tattoo” lesions). Ring or pinhole lesions appear as solitary, 0.5-3 cm, round or elliptical blemishes, which sometimes coalesce. They are usually light gray and may have a dark gray border, although the reverse color pattern is also seen. Lesions may persist for months or years without any apparent ill effects to the animal. Major differentials include cutaneous streptothricosis and calicivirus. Poxvirus has not been isolated from pinnipeds or cetaceans. Diagnosis is based on the presence of eosinophilic, intracytoplasmic inclusion bodies in lesion biopsies and is confirmed by identification of typical poxvirus particles by electron microscopy.

Poxviruses of marine mammals do not appear to cause systemic infections. Although animals with cutaneous poxvirus lesions have died, other factors were responsible. Therapy to control secondary bacterial infections is indicated only when skin lesions suppurate. The parapoxviruses of pinnipeds can cause isolated lesions on the hands of persons not wearing gloves when in contact with infected animals.

**Miscellaneous Viral Diseases:**

A ringed seal in Norway was wounded and appeared confused; its overall condition deteriorated over 5 days, and it became aggressive. Rabies was confirmed by immunofluorescent examination of the brain. At the time, there was an epidemic of rabies in foxes in the area.

The only retrovirus identified to date in a marine mammal was a spumavirus isolated from recurring skin lesions in a California sea lion that subsequently died of Pasteurella pneumonia complicated with herpesvirus.

An enterovirus of unknown pathogenicity isolated from a rectal swab of a California gray whale has now been reclassified as a calicivirus. Antibodies, unassociated with disease, against human influenza virus (after challenge) and poliomyelitis virus were found in bottlenosed dolphins.

Severe enteritis and vomiting that rapidly lead to death in a captive beluga whale was suggestive of parvovirus enteritis, but no virus was isolated.

**Neoplastic Diseases**

Tumors in marine mammals are infrequent, although a wide variety has been reported. They are of little consequence except for malignant lymphoma in harbor seals, in which horizontal transmission can occur in a closed population.