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SEAL AND SEA LION MEDICINE

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Introduction

Seals and sea lions are commonly managed in display facilities and rehabilitated after stranding, with much of the medicine presented here learned from care of the California sea lion (*Zalophus californianus*), northern fur seal (*Callorhinus ursinus*), South American sea lion (*Otaria flavescens*), harbor seal (*Phoca vitulina*), gray seal (*Halichoerus grypus*), and northern elephant seal (*Mirounga angustirostris*). While disease prevalences vary widely across species and environment, much of the knowledge gained from care of these more common species may be applied to lesser known species.

Husbandry

Pools, Haul-Out Areas, and Enclosures

All pinnipeds require both water and haul-out space. Although seals and sea lions can survive without access to water for weeks at a time, they appear more content when given free access to water. Most pinnipeds will eat more readily when offered food in water, particularly in a rehabilitation situation. Fur seals will defecate and groom when given access to a pool; if left in a dry haul-out area, they may appear clinically depressed and fur quality may be compromised by contact with urine and feces. Pool design should aim at accommodating the behavioral and physical needs of the animals housed, as well as maintaining water quality (see **Chapter 31**). For otariids, the pool may be sunken below ground or raised, with access by ramps. As phocids do not have the climbing abilities of otariids, a sunken pool with the water level close to the edge to allow easy exit from the pool is preferred. Debilitated pinnipeds may have difficulty exiting pools, regardless of design, though gently sloping sides or ledges

just below the surface of the water both facilitate the egress of any species and allow seals to rest in shallow water.

Pinnipeds should be housed in salt water; however, they are often housed in freshwater systems due to economic or logistical constraints. Ophthalmic problems are more common in freshwater than in salt water, and in pools without shade (Dunn et al. 1996; Colitz 2010b; see section below entitled Eyes; see **Chapter 23**). Fur seals in particular should be housed in salt water, as they may not groom properly in freshwater, resulting in loss of fur integrity and poor thermoregulation. While freshwater is generally not available to wild pinnipeds, provision of freshwater (low-lying bucket or pan) to pinnipeds in salt water systems may help them maintain hydration, particularly during rehabilitation and when housed in particularly warm environments. When housed in freshwater, oral salt supplementation should be provided to prevent hyponatremia (Geraci 1972a; Lair et al. 2002).

Lighting should mimic the natural photoperiod for the species as closely as possible. Harbor seals maintained in continuous light conditions have had disrupted molt cycles that reverted to normal when a natural photoperiod was reinstated (Mo, Gili, and Ferrando 2000). Extremes of both heat and cold should be prevented, although in general most species are better able to tolerate cold than heat. Geraci (1986) states that healthy, robust harbor, gray, harp (*Pagophilus groenlandicus*), and ringed seals (*Phoca hispida*) can tolerate water at freezing temperatures, and air temperature below –20°C (–4°F), although a northern elephant seal died after being exposed suddenly to an outdoor temperature of –15°C (5°F) for 30 minutes. Hyperthermia can be avoided by providing access to shade, pools, or sprinklers, when ambient temperatures rise above 26°C (79°F). Hypothermia is rare, but can be a significant problem in thin, malnourished animals in rehabilitation. Provision of waterproof heating pads, plastic pads, platforms off concrete floors, or kennel areas with heat lamps, as well as permanent structures that provide protection from wind and rain, can help prevent hypothermia.

Feeding

Although wild pinnipeds feed on a variety of prey, managed animals are usually maintained on a diet of herring (*Clupeidae* spp.), smelt (*Osmeridae* spp.), mackerel (*Scombridae* spp.), capelin (*Mallotus villosus*), and squid (*Loligo* spp.). Care should be taken if feeding mackerel and other scombroid fish to ensure it has been appropriately stored to avoid scombroid toxicity. As herring is a relatively fatty fish, it is commonly fed to produce rapid weight gain. Details of the nutritional content of different diets and the methods to calculate caloric requirements of marine mammals are provided (see **Chapter 29**), while hand-rearing techniques are given in **Chapter 30**. As a rough guideline, young growing pinnipeds are fed 8–15% of their body weight of food per day, and older animals 4–8% per day. Thiamine at 25 to 35 mg/kg fish and vitamin E at 100 IU/kg fish are recommended to prevent nutritional disorders associated with a frozen fish diet. When supplementing an animal's diet, it is advisable to feed a fish containing the supplements prior to the main feed to ensure the animal receives all of its medications.

Restraint

The methods commonly used to restrain pinnipeds may be classified into behavioral, physical, mechanical, and chemical, with choice depending upon the objective. For example, if the desired objective is to perform an abdominal ultrasound, a mechanical squeeze may be safest for handlers and the animal, but may be suboptimal for effective ultrasound positioning. Different types of restraint are often used in combination. For example, a chemical sedative such as a benzodiazepine may be given to an animal to augment physical restraint. Many of these techniques are depicted well in Geraci and Lounsbury (1993).

Behavioral Restraint Behavioral restraint is an extremely effective technique for most captive pinniped species and can be either free- or protected-contact. The participation of an animal in its own health evaluations can be far less stressful and time-consuming than other restraint techniques, though the lack of a barrier in a free-contact situation may also be dangerous to people or animals (see **Chapter 39**).

Physical Restraint Physical restraint is limited by size and species, the animal's level of aggression and alertness, and the experience and physical ability of the restrainers. It is usually very safe for the animal, but human safety is a concern with larger animals. Physical restraint requires a thorough knowledge of the behavior and anatomy of the species being restrained. For example, larger otariids have tremendously strong forelimbs in comparison with phocids. The fore flippers may have to be secured by additional personnel to prevent the animal from gaining leverage and rising up (Gentry and Holt 1982). Creative use of towels, blankets, bags, and nets will aid physical restraint and increase the safety of personnel. A common method of restraint of a smaller pinniped (phocids under approximately 60 kg and otariids under approximately 30–40 kg) is to place a hoop net or wrap large towel over the animal's head to restrict vision and mobility prior to restraining. The primary restrainer can then control the head by holding the base of the skull with both hands and pushing the head toward the ground. The primary restrainer should straddle the animal by resting their knees on the ground and controlling the side-to-side and upward motion. It is critically important that the restrainer rest their body weight on their own knees and not on the animal, as the pressure can severely restrict the animal's respirations. Care must also be taken to ensure that the animal's nares and mouth are clear of netting or towels to allow full respirations.

Mechanical Restraint Mechanical restraint is limited by the availability of adequate equipment, the cost of which varies considerably. Many types of mechanical restraint devices have been used with pinnipeds, including chutes, herding boards, restraint boards, stretchers with straps, restraint boxes, squeeze cages, and slings (Cornell 1986; Gentry and Casanas 1997). As some mechanical restraint equipment can be very large and heavy, it may be difficult to use in field situations. In general, mechanical restraint devices are designed to maximize safety to human operators, but may pose some risk to the animal. Some restraint boards require a padded, hinged guillotine to secure the neck, and could obstruct the airway. Mechanical squeeze cages should be used with caution, and only by experienced personnel, since it is possible to use excessive pressure and cause trauma or interfere with adequate ventilation. Be aware that some of the mechanical restraint devices limit full access to the animal.

Chemical Restraint The ability to use chemical restraint relies on the expertise of the operators, and often requires the presence of a specially trained veterinarian (see **Chapter 26**). Some commonly used agents for sedation of phocids include diazepam, or midazolam +/– butorphanol intravenously (IV) or intramuscularly (IM; though diazepam has relatively poor IM absorption), and for anesthesia are IV propofol, alfaxalone, or tiletamine/zolazepam. Induction agents are likely to induce apnea; thus, the clinician should be prepared to intubate. Masking with isoflurane or sevoflurane is possible, but may prove difficult due to breath-holding; however, both are commonly used for anesthetic maintenance. Some common agents used in otariids include midazolam with medetomidine and butorphanol IM, which can be reversed with flumazenil, atipamezole, and naltrexone, respectively. Alternatively, midazolam with either alfaxalone or ketamine IM can be used effectively, although the ketamine dose is generally lower than that used in terrestrial mammals. Isoflurane and sevoflurane are also commonly used safely in conjunction with injectable agents, and smaller otariids can be readily masked to an anesthetic plane (with either inhalant) while manually restrained. Additional information on sedative and anesthetic drugs and dosages is given in **Chapters 26 and 27**.

Diseases

Details of viral, bacterial, fungal, protozoal, parasitic, and noninfectious diseases of pinnipeds are provided in **Chapters 14 and 17 through 21**, respectively. To avoid repetition, this chapter focuses on the clinical signs of these diseases, describes these by affected organ system for ease of differential diagnosis, and then discusses treatment. Drug dosages for recommended therapeutic agents are given in **Chapter 27**.

Integumentary System

Multiple viruses cause dermal lesions in pinnipeds, with sealpox and calicivirus among the most common. Documented sealpox viral infections are generally of the Parapoxvirus family (Becher et al. 2002; Nollens et al. 2006), though an Orthopoxvirus has been identified in two wild Steller sea lion pups in Alaska (Burek et al. 2005). Poxvirus infections typically occur in animals that have been recently weaned or are in a rehabilitation setting; then these infections spread rapidly among a susceptible population (Hastings et al. 1989; Müller et al. 2003; Nollens et al. 2005). These viruses cause pathognomonic lesions, which consist of round, raised, firm skin nodules 0.5 to 1 cm (0.4 in) in diameter that gradually increase in size over the first week, and may ulcerate or suppurate (see **Chapter 17**). Lesions commonly occur over the head and neck, but may also arise over the thorax and abdomen, perineal regions, or in the oral cavity (Müller et al. 2003; Nollens et al. 2005). Satellite lesions appear in the second week and may spread rapidly. Lesions are usually self-limiting and regress after 4–6 weeks, although some have persisted for months. Animals usually remain active when affected, although lesions around the lips and eyes may cause sufficient discomfort to reduce appetite. Marked neutrophilia and hyperglobulinemia may occur in association with nodule development. Diagnosis can be made through skin biopsy (see **Chapter 17**). Treatment is usually unnecessary, although broad-spectrum antibiotics may be needed to control secondary bacterial infections, and nonsteroidal anti-inflammatory drugs (NSAIDs) can be used to reduce discomfort associated with the lesions. In vitro studies suggest that cidofovir could be an effective antiviral in treating sealpox (Nollens et al. 2008). Sealpox is zoonotic and proper personal protective equipment (PPE) should be worn when handling affected individuals (see **Chapter 4**).

San Miguel sea lion virus is a calicivirus that in California sea lions causes vesicles on both dorsal and ventral surfaces of the flippers, occasionally around the lips, on the dorsum of the tongue, and on the hard palate (Gage et al. 1990; Smith and Boyt 1990; Van Bonn et al. 2000; see **Chapter 17**). The vesicles usually erode, leaving rapidly healing ulcers, but may become secondarily infected by bacteria, especially in malnourished and debilitated animals. Calicivirus has also been shown to cause gastroenteritis with the onset of infection, with signs of vomiting, abdominal pain, and diarrhea. Sea lions will respond to supportive care of fluid therapy and antibiotic coverage. Hematologic changes can include neutropenia, lymphopenia, and thrombocytopenia. The disease can progress to vesicles following the enteritis phase (Schmitt 2009). Diagnosis is confirmed with PCR or isolation of the virus from aspirated vesicular fluid or feces, but is often presumptive based on clinical appearance. Treatment is supportive, aimed at preventing secondary infection and enhancing nutritional status of the animal. Occasionally, stranded sea lions are observed with severe gangrenous necrosis of

the phalanges. Although development of these lesions has not been observed, it is suspected that they may result from vesicles that became secondarily infected with bacteria. These lesions are treated with debridement, topical wound care, and systemic antibiotic therapy based on culture and antibiotic sensitivity results. NSAIDs can also be used to reduce discomfort. A novel papillomavirus was identified in two California sea lions with proliferative and focally extensive skin lesions. PCR was used to characterize the virus and the lesions regressed, without treatment, after several months (Rivera et al. 2012).

Herpesviruses have been isolated from harbor and gray seals with small erosive skin lesions, and observed in epithelial plaques in harbor seals and California sea lions. Although infrequent, herpesviruses should be considered in the differential diagnosis of skin lesions, and skin biopsies should be examined for inclusions.

Morbillivirus dermatitis has been diagnosed in both a hooded seal (*Cystophora cristata*) and a harp seal. Skin lesions consisted of epithelial hyperplasia, hyperkeratosis, degeneration, and necrosis, and the systemic infection was fatal in both cases. Diagnosis of morbillivirus is described in **Chapter 17**.

Bacterial infections are common in pinnipeds, especially in dermal abscesses in stranded animals, though are rarely reported in the literature as the primary cause of dermal disease (see **Chapter 18**). Multifocal circular ulcers 1–2 cm (0.4–0.8 in.) in diameter have been observed in California sea lions and northern elephant seals. Histologically, these appear to be the consequence of vasculitis and thrombosis. Microabscesses are also common on the ventral abdomen of sea lions following septicemia. Diagnosis is based on the histological appearance of biopsies, and treatment with systemic antibiotics is recommended. Secondary bacterial infections are common with traumatic injuries and bite wounds. Antibiotic therapy should be selected based on culture and sensitivity when possible. Bite wound infections can lead to severe systemic disease, as in the case of one California sea lion, which developed a focal bacterial meningitis and paraparesis from a chronic dermal ulcer. *Escherichia coli* serovar *haemolytica* and *Clostridium perfringens* were identified as the primary underlying agents (Braun et al. 2015). Subcutaneous abscesses due to infection with *Mycobacterium chelonae* in a captive gray seal (Stoskopf et al. 1987) and *M. smegmatis* in a captive California sea lion (Gutter, Wells, and Spraker 1987) were diagnosed after culturing the organisms from aspirated fluid. The gray seal was treated successfully with minocycline, while the sea lion died with concurrent pulmonary abscesses. Methicillin-resistant *Staphyloccocus aureus* (MRSA) has been documented in a stranded harbor seal. Treatment was successful and based on culture and sensitivity (Fravel et al. 2011).

A number of fungal diseases of the skin have been described. Fungal acanthosis and alopecia associated with *Candida albicans* and *Fusarium* spp. infections typically occur at mucocutaneous junctions, around nail beds, and in the axillae. Lesions are most often observed in managed animals maintained in freshwater. Diagnosis is based on skin scrapings and fungal culture, PCR, and/or histological examination of biopsies. Topical treatment is difficult without limiting access to water, but systemic treatment of an elephant seal with fluconazole at 0.5 mg/kg was effective in clearing clinical signs (Gulland, unpubl. data)

Trychophyton rubrum infection caused multifocal to coalescing, ulcerative, and nonpruritic lesions over the lumbar region in a Patagonian sea lion. Oral terbinafine at approximately 2.3 mg/kg per os (PO) SID and a topical dilution of enilconazole over a period of 75 days were successful in clearing the infection (Quintard, Lohmann, and Lefaux 2015). Similar treatment was employed for two California sea lions with *Microsporum gypseum* dermatomycosis with complete resolution of lesions after 65 days of therapy. Lesions were well demarcated, depigmented, were covered in crusts, and were most extensive over the flippers (Sós et al. 2013). *Trichophyton mentagrophytes*, *Malassezia* spp., and *Yarrowia* (*Candida*) *lipolytica* were isolated in a group of captive harbor seals and gray seals that presented with erythematous, thickened, alopecic skin lesions. Lesions were primarily found over the face and flippers, particularly around the nail bed. Various treatments were initiated including topical treatment with miconazole and chlorhexidine, and systemic treatment with oral itraconazole at 5 mg/kg PO BID with variable responses. Environmental factors, including overchlorination of water and warm water temperatures, contributed to occurrence of disease (Pollock, Rohrbach, and Ramsay 2000). Cystofilobasidiales infection caused systemic mycoses in a California sea lion. This animal presented with ring lesions over the flippers, which progressed to dermal nodules over the flippers, abdomen, and muzzle. Itraconazole (2.5 mg/kg PO BID), and later voriconazole (4 mg/kg PO BID), failed to resolve the infection. Acute liver failure was noted, likely due to voriconazole toxicity, and thus caution is recommended in using voriconazole to treat fungal infections (Field et al. 2012).

Alopecia, broken hair shafts, and pruritus are common in debilitated seals and sea lions associated with lice infestation. Most infections are species specific; the California sea lion louse is *Antarctophthirius microchi*, and the harbor seal louse is *Echinophthirius horridis*. Lice may be observed with the naked eye and are readily treated with ivermectin, dichlorvos, or disophenol systemically, or with topical rotenone louse powder. Demodicosis, also characterized by alopecia and pruritus, has been observed in California sea lions, northern fur seals (Spraker, pers. comm.), and harbor seals (Kim, Lee, and Kwak 2015). Diagnosis based on histological detection in biopsies and treatment with Amitraz (0.01% once weekly) and ampicillin (10 mg/kg PO SID) has been effective in clearing clinical symptoms of demodectic mange (Sweeney 1986b; Kim, Lee, and Kwak 2015). *Pelodera strongyloides* parasites have caused mild superficial dermatitis and perifolliculitis in Pacific harbor seals. Diagnosis was made by biopsy and histology (McHuron et al. 2013).

Traumatic skin wounds are common in stranded animals. Net entanglements, fishhooks, and gunshot injuries are especially common in California sea lions (Goldstein et al. 1999). Diagnosis of gunshot injuries is dependent upon radiographic detection of lead fragments or pellets, or recovery of the projectile, although many wounds suggestive of exit wounds are observed in pinnipeds from which no evidence of gunshot can be detected. Differential diagnoses include bite wounds (usually paired holes of similar size) and bird damage. The characteristics of shark bite wounds vary with species of shark. Management of traumatic skin wounds is based on removal of foreign bodies and debris, debridement of devitalized tissue, control of infection, and promotion of healing, as in domestic animals. Many topical therapies have been employed including Betadine ointment, chlorhexidine scrubs, silver alginate dressings, platelet-rich plasma, Granulex sprays, honey, laser therapy, and many more. Choice of topical agents should be made on a case-by-case basis. Placing an animal in salt water rather than freshwater may enhance wound cleansing. Tetracycline and penicillin have been used to treat shark wounds, as *Vibrio* spp. and *Clostridium* spp. are frequently isolated from these wounds (Pavia et al. 1989; Klontz et al. 1993). Severe tissue avulsion from traumatic injury is managed best with debridement of necrotic tissue and topical treatment to encourage granulation and healing by secondary intention rather than surgical closure.

Rare neoplastic diseases of the skin have been diagnosed in pinnipeds. A cervical dermal melanoma was described in a 7-month-old stranded harbor seal. Diagnosis was made by fine needle aspirate and subsequent biopsy and histopathology, though the animal died during surgery to remove the mass. The melanoma was described as low grade, and no evidence of metastasis was found on histopathology (Morick et al. 2010). Pleomorphic liposarcoma was described in a captive South African fur seal that presented with a large, progressive, ulcerated mass over the right shoulder. The animal died, and pulmonary, hepatic, splenic, and lymph node metastases were noted on necropsy (Pervin et al. 2016). A cutaneous mast cell tumor was identified in an adult captive California sea lion. Biopsy was needed for diagnosis, and surgical excision was successful in removing the tumor (Staggs, Henderson, and Labelle 2016). An invasive cutaneous squamous cell carcinoma was documented in a Hawaiian monk seal, and surgical excision was elected (Doescher et al. 2010). Benign mammary hyperplasia resulting in a mammary mass development was identified in a managed subadult female sea lion and surgically excised with no recurrence (Schmitt, unpubl. data).

Alopecia and acanthosis have occurred in captive harbor seals that failed to molt when maintained in constant photoperiod (Mo, Gili, and Ferrando 2000). Diagnosis was based upon clinical history, and restoration of a natural photoperiod resulted in new hair growth. Cutaneous lupus erythematosus was diagnosed in a captive gray seal that for 9 years had continuous ulcerative nasal dermatitis and intermittent ulcerative dermatitis of the nail beds and dorsum of the body (Burns 1993). Treatments with systemic prednisone, antibiotics, and antifungals, and with topical steroids, and protection from ultraviolet radiation, were unsuccessful, and the seal died during the second week of treatment. A pruritic allergic dermatitis, with loss of guard hairs over the dorsum, was described in a captive sub-Antarctic fur seal (*Arctocephalus tropicalis*; Bodley, Monaghan, and Mueller 1999). Diagnosis was based on positive reactions to allergens prepared from weed, grass, tree pollens, and some insects. Symptomatic treatment with oral antihistamines was only partially successful, but specific allergen immunotherapy using 10 allergens was effective, despite side effects.

Alopecia of an unknown cause is well documented in juvenile Australian fur seals. Affected individuals are generally in poorer body condition than healthy conspecifics and have higher circulating T4 levels, possibly due to increased thermoregulatory demands. Higher levels of PCBs in association with thyroid disruption have been documented in this population, though thyroid disturbance does not appear to be the cause of the alopecia (Lynch, Keeley, and Kirkwood 2014). Guard hair alopecia of unknown cause has also been observed in stranded northern fur seals and Guadalupe fur seals (Field, unpubl. data).

A skin condition characterized by hyperkeratosis, alopecia, and ulceration has been well described in northern elephant seals, but its etiology remains obscure (Beckmen et al. 1997). This disease has been associated with decreased levels of circulating thyroxine (T4) and triiodothyronine (T3); however, thyroid function testing was normal in affected individuals (Yochem et al. 2008). Another ulcerative skin disease of obscure etiology has been described by Anderson et al. (1974) in gray seals. In 2011, a new ulcerative dermatitis disease syndrome was described in a number of Arctic pinniped species, including Pacific walruses (*Odobenus rosmarus divergens*), bearded seals (*Erignathus barbatus*), ringed seals (*Phoca hispida*), ribbon seals (*Phoca fasciata*), and spotted seals (*Phoca largha*). This unusual mortality event (UME) was characterized by generalized ulcers/erosions in Pacific walruses, and similar lesions with alopecia over the eyes, muzzle, hind flippers, tail, and trunk of ice seals. Affected individuals were more approachable and lethargic, with a tendency to haul out more frequently. Some mortality has been associated with the syndrome, and histopathology indicates significant involvement of the liver, lung, immune system, and the skin's vascular bed. To date, no associated bacterial, viral, or fungal agent has been identified, and no toxin or pollutant has been implicated in causing this disease (Burgess et al. 2013; Stimmelmayr et al. 2013).

Musculoskeletal System

Diseases involving the pinniped musculoskeletal system include infectious causes, trauma, and congenital abnormalities. Numerous bacteria can cause deep abscesses, myositis,

osteomyelitis, and arthritis (Thornton, Nolan, and Gulland 1998). Most of these bacteria are opportunistic, occurring following trauma, introduction through contaminated hypodermic needles or surgical instruments, or hematogenous spread, as a result of generalized sepsis (see **Chapter 18**). *Clostridium perfringens* has been isolated from cases of severe myositis following poor injection technique (Greenwood and Taylor 1978), and *Otariodibacter oris* was isolated from seals and sea lions with abscesses or osteomyelitis, as well as a variety other bacteria (Hansen et al. 2013).

Sarcocystis infection causes myositis, as well as neurologic and generalized disease in pinnipeds (see **Chapter 20**). Antemortem diagnosis may be based on clinical presentation, as well as antibody levels, with confirmation by histology and PCR of muscle biopsies. Treatment with oral ponazuril at 10 mg/kg for at least 4 weeks, along with supportive care (fluids and anti-inflammatory medication to reduce inflammation secondary to parasite die-off), has been clinically effective in some cases (Carlson-Bremer et al. 2012; Alexander et al. 2015), although optimal treatment duration is still unclear, and up to 3 months of treatment may be required to clear an infection (Mylniczenko, Kearns, and Melli 2008). Parasites, including the filariid *Acanthocheilonema odenhali* and inactive *Uncinaria* spp. larvae, may be found in muscles and fascia, but do not usually cause clinical signs of disease.

Trauma is especially common in free-ranging, stranded pinnipeds. Osteomyelitis affecting the extremities, in particular, is a common occurrence secondary to trauma (Thurman, Downes, and Barrow 1982), or superficial infections such as calicivirus (see Integumentary System above). Antimicrobial treatment is generally utilized in these cases. Fractures may require surgical repair or amputation, particularly with open, chronic, fractures and osteomyelitis (Bennett, Dunker, and Gage 1994; Lucas, Barnett, and Reiley 1999; Lewer et al. 2007; Malabia et al. 2011; Hespel et al. 2013; Garcia et al. 2015; see Surgery below). Rhabdomyolysis secondary to general anesthesia and surgery are described (Bailey et al. 2012), and patient positioning and support, as well as close perioperative and postoperative monitoring, is recommended for longer surgical procedures, particularly involving larger animals. Monofilament line entanglement or fishhooks in the skin often require general anesthesia to cut material free and clean wounds. Intervertebral disc protrusion, collapsed thoracic disc spaces, or spondylitis has been observed in wild animals, secondary to blunt force trauma, or managed animals performing repetitive behaviors, such as standing vertical against a wall (Schmitt, pers. comm.). The spinal cord in pinnipeds terminates between the 8th and 12th thoracic vertebrae, so any trauma to the cranial spine or collapsed thoracic disc space can result in paresis or paralysis of hind limbs.

Some neoplastic diseases can manifest clinical signs in the musculoskeletal system. Carcinomas may erode the lumbar spine, affecting neurological function, as well as potentially resulting in pathological fractures. Animals may exhibit hind-end paresis or paralysis (Gulland et al. 1996a). Lymphosarcoma has affected the bone marrow in a harbor seal (Stroud and Stevens 1980), and a rhabdomyosarcoma was noted in a free-ranging Steller sea lion (Zabka et al. 2004). High levels of polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichloroethane (DDT) have been associated with pathological bone lesions and reproductive failure in seals in the Baltic Sea, likely due to alterations in bone and thyroid homeostasis; however, the effects of these compounds remain to be fully characterized (Routti et al. 2008). A variety of congenital bone malformations and abnormalities have been reported in pinnipeds, including occipital bone dysplasia, atlantoaxial subluxation, and cribriform plate aplasia (Dennison et al. 2009; Maclean et al. 2008).

Physical examination, fine-needle aspiration with cytology and culture of the aspirate, muscle biopsy, radiographs, computerized tomography, and ultrasound (see **Chapters 24 and 25**) will all facilitate diagnosis of musculoskeletal problems. Treatment is dictated by the diagnosis, though certain techniques commonly used in other species may be difficult in pinnipeds, such as splinting. Initial wound care is similar to other species, and fracture repair of extremities has been performed using both internal and external fixator systems (see Surgery below). Bandaging is particularly difficult in these species due to their fusiform body shape, flipper shape, and aquatic environment. Wounds are often left open for second intention healing while allowing animals access to water where they appear to be more comfortable.

Digestive System

Otariids will show behavioral signs of discomfort with abdominal pain, such as fore flippers extended down over the ventral abdomen, or logging at the surface or clutching to the edge of a pool edge on their side with fore and hind flippers with a hunched abdomen. Dental disease in pinnipeds can be ascertained by animals showing complete inappetence, dropping fish, dysphagia, playing with food, or prehending food on one side and not the other.

Oral lesions are often viral or traumatic in origin. Herpesvirus, morbillivirus, and poxviruses can cause ulcerative oral lesions in pinniped species. Treatment is largely supportive, and targeted at controlling secondary bacterial infections and reducing discomfort. Trauma secondary to foreign bodies such as fishhooks or entanglements is also relatively common. Partial glossectomy was effective in removing necrotic lingual tissue secondary to fishhook entanglement in a Hawaiian monk seal (Barbieri et al. 2013). Megaesophagus and concurrent intestinal volvulus have been described in harbor seals in two separate cases. Clinical symptoms included aerophagia, regurgitation, vomiting, diarrhea, bloat, abdominal pain, and occasional inappetence. Megaesophagus was diagnosed using contrast radiography. Both were medically managed using gastric support medications, but eventually died from complications (Tuomi et al. 2011). Ingested foreign

bodies are not uncommon in dehydrated and malnourished seals and sea lions. On the East Coast, rescued "ice seals," such as harp and hooded seals, have been diagnosed with gastric impaction secondary to ingestion of rocks. Gastrotomy has been performed to remove rocks, with successful recovery and return to the wild (Schmitt, pers. comm.). Wild sea lions are diagnosed with monofilament line and fishhook ingestion commonly, and successful treatment is based on the degree of tissue damage, and if the hook can be removed with endoscopy or gastrotomy (Schmitt, pers. comm.)

Gastritis and gastric ulcers are common in pinnipeds. Stress and high burdens of gastric nematodes can cause gastric ulceration and chronic emesis. Gastric nematodes have also caused duodenal perforations leading to peritonitis and death in stranded California sea lions (Fletcher et al. 1998). Gastroprotectants and anthelmintics can be used to manage animals with suspected ulcers (see end of this section for details). Several novel *Helicobacter* spp. have been associated with gastritis in an Australian sea lion and in harp seals (Harper et al. 2003; Oxley, Powell, and McKay 2004). In the sea lion, recurrent episodes of anorexia and abdominal discomfort prompted endoscopy and subsequent biopsy of gastric and intestinal mucosa. *Helicobacter* spp. and *Wolinella* spp. were identified by PCR. Treatment included amoxicillin at 10 mg/kg PO BID and metronidazole at 10 mg/kg PO BID. Initially the animal's condition improved with therapy, but repeated flare-ups occurred over several years.

Primary neoplasms of the oral and gastrointestinal tracts have been documented in pinnipeds. Lingual squamous cell carcinoma has been described in a California sea lion (Sato et al. 2002), and esophageal squamous cell carcinoma has occurred in several aged captive harbor seals (Flower et al. 2014). Clinical symptoms included intermittent dysphagia, inappetence, regurgitation, and abnormal posturing. The tumors were often ulcerated and occurred near the gastroesophageal junction. Bloodwork abnormalities in these cases included azotemia, hyperproteinemia, hyperglobulinemia, and leukocytosis. Gastric carcinoma developed in a captive, aged South American sea lion. Vomiting, anorexia, and weight loss occurred, with hematemesis and melena in end stage disease (Yamazaki, Koutaka, and Une 2016).

Enteritis in pinnipeds can be caused by a variety of infectious agents. Viral causes of enteritis include morbillivirus, which has caused chronic ulcerative stomatitis and acute hemorrhagic enteritis in wild harbor seals in Europe (Jauniaux et al. 2001). In an outbreak of herpesvirus in juvenile harbor seals, early clinical symptoms included vomiting, diarrhea, and fever. Severe hepatic necrosis was found postmortem (Borst et al. 1986). Enteritis has been linked to bacteria, including *Clostridium* spp. and *Salmonella* spp. in different pinniped species. The interpretation of culture of these organisms from fecal samples is difficult, as they have been cultured from both clinically normal animals and those with severe hemorrhagic enteritis. Disseminated blastomycosis was identified in two California sea lions in different captive facilities. *Blastomyces dermatitidis* was the suspected primary pathogen, and postmortem findings included enteritis with subsequent rupture and peritonitis, infiltration of the spleen and liver, severe pyogranulomatous pneumonia, and ulcerative skin lesions (Zwick et al. 2000).

Most gastrointestinal parasites are part of the normal flora of free-ranging pinnipeds and do not significantly affect the host; however, they can be responsible for clinical disease. Nematodes may potentiate malnutrition in already compromised animals, especially young animals, and high parasite burdens have the potential to obstruct the intestinal lumen (Banish and Gilmartin 1992). Juvenile Hawaiian monk seals with cestode infections, primarily *Diphyllobothrium* spp., tend to be in poorer body condition than those without infections (Reif et al. 2006; Gobush, Baker, and Gulland 2011). Treatment with praziquantel at 5 mg/kg IM for 2 days in Hawaiian monk seals has shown some seasonal promise in improving body mass and survivorship (Gobush, Baker, and Gulland 2011), although higher doses have been used to clear cestode infection in rehabilitated monk seals. Acanthocephalans have caused gastrointestinal perforation and peritonitis in gray and harbor seals. Hookworm infections causing enteritis and bacteremia have been associated with increased wild California sea lion pup mortalities on San Miguel Island (Spraker et al. 2007). California sea lions appear to be the definitive host for enteric coccidian parasites, with only mild associated enteritis. This parasite, however, was implicated in causing protozoal lymphadenitis, hepatitis, myocarditis, and encephalitis in a neonatal harbor seal (Colegrove et al. 2011), suggesting varying interspecies pathogenicity (see **Chapter 20**).

Hepatitis has been associated with adenovirus infection in otariid species (Dierauf, Lowenstine, and Jerome 1981; Goldstein et al. 2011; Inoshima et al. 2013). Clinical signs of affected animals included diarrhea, anorexia, abdominal pain, posterior paresis, polydipsia, and photophobia. Bloodwork abnormalities include markedly elevated AST and ALT. Adenoviral infection can be fulminant and fatal (Inoshima et al. 2013). Hepatic necrosis and intranuclear inclusions are typical postmortem findings. To date, at least one novel adenovirus has been described in association with hepatitis, Otarine Adenovirus 1 (OtAdV-1; Goldstein et al. 2011). Several types of bacteria associated with hepatitis have been isolated from the livers of pinnipeds (Thornton, Nolan, and Gulland 1998). Mycotic agents including *Coccidioides immitis* have also affected the liver of California sea lions (Fauquier et al. 1996), and neoplastic diseases may manifest themselves in the liver of pinnipeds (see **Chapter 14**, **Table 14.1**)

Hemochromatosis has been observed in managed California sea lions and northern fur seals, but the etiology is obscure (Garcia et al. 2000; Clauss and Paglia 2012).

Primary pancreatic disease has been rarely reported, though may be underreported due to nonspecific clinical signs and lack of species-specific tests. Chronic pancreatitis has been reported in an adult captive California sea lion, with no identifiable underlying cause. The animal developed

secondary diabetes mellitus and was managed with glargine insulin injections, gastric protectants, and a high-protein, low-fat diet (Meegan et al. 2008). Pancreatic adenocarcinoma has been described in an adult captive Steller sea lion. Postmortem secondary lesions included pancreatitis, bile duct obstruction, hepatitis, and hepatic encephalopathy (Goertz et al. 2011).

Small amounts of rectal bleeding, irregular in frequency and of varying duration, in harbor seals and California sea lions have been associated with a postmortem finding of ileocecocolic intussusception (Lair and Lamberski, pers. comm). A case of antemortem diagnosis of ileocecocolic intussusception occurred in a captive harbor seal that initially presented for anorexia and a leukopenia with degenerative left shift. Malodorous diarrhea developed, with concurrent tenesmus and regurgitation. Abdominal radiographs revealed gas dilated loops of intestine, and an exploratory laparotomy revealed significant ischemic compromise leading to euthanasia (Heym et al. 2011). Congenital abnormalities, including cleft palate (Suzuki et al. 1992) and hiatal herniation, have been seen in stranded pinnipeds (Beekman 2008; Biancani et al. 2012). Laparoscopic gastropexy was successfully used to repair a hiatal hernia in a stranded weanling elephant seal (Greene et al. 2015).

Iatrogenic causes of gastrointestinal disease include feeding inappropriate formulas or spoiled fish, using poorly designed feeding tubes, or feeding at an inappropriate rate or volume. Young animals, especially when debilitated, often go through a period of regurgitation and malabsorption if formula is introduced too quickly. Adequate rehydration and a gradual reintroduction to complex diets may aid in decreasing the frequency of emesis and diarrhea (see **Chapter 30**). Impactions caused by solidifying formula in neonates have been seen and may also be prevented by feeding appropriate formulas, monitoring hydration, and a gradual introduction to complex diets.

Clinical symptoms associated with diseases of the digestive tract in pinnipeds include inappetence, emesis, regurgitation, icterus, melena, hematochezia, diarrhea, straining, and/or steatorrhea. Abdominal pain or discomfort is often manifested as inappetence, lethargy, or depression. Otariids with abdominal discomfort will often tuck their flippers to their abdomen. In the water, they may float with tucked flippers and a hunched back.

Diagnosis of gastrointestinal disease often requires a series of diagnostic techniques beginning with physical examination. Physical examination helps detect broken, missing, or worn teeth, oral ulcers, oral foreign bodies such as fish spines and fishhooks, abdominal distension, abdominal masses, a palpable fluid wave, perineal swelling, or prolapsed rectum. A complete blood count may help identify an infectious cause. Clinical chemistry findings may indicate specific organ involvement, hypoproteinemia, gastrointestinal hemorrhaging, and electrolyte imbalances associated with chronic emesis or diarrhea (see **Appendix 1**, **Tables A1.2** [Phocids] and **A1.3** [Otariids], Clinical Laboratory Values). Ultrasound may be used to identify a variety of abnormalities within the abdomen, including ascites, ileus, foreign bodies, and organ abnormalities. Abdominocentesis of a distended abdomen can differentiate peritonitis and hemoperitoneum. Culture and cytology of aspirated fluid may help further define disease. As many pinnipeds have large intra-abdominal vessels and large spleens, aspiration of frank blood does not necessarily indicate hemoperitoneum, and ultrasound-guided aspiration will help guide sample collection. Further diagnostics may include radiographs to detect gastric foreign bodies, gastric impaction, or constipation. Although it is often difficult to achieve good contrast in pinniped abdominal radiographs due to the relative lack of visceral fat, in young and thin animals, it is possible to achieve some indication of organ size, displacement, and intra-abdominal masses. Endoscopy can be used to diagnose gastric ulcers, gastritis, colitis, and gastric foreign bodies, as well as obtain gastric and colonic biopsies. Laparoscopic examination can enable direct visualization of the gastrointestinal serosa, as well as liver, pancreas, and associated structures. Biopsies of the liver and other tissues may be obtained either laparoscopically or by ultrasound-guided biopsy (see **Chapter 24**).

Supportive therapy for gastrointestinal disease is critical, as fluid, electrolyte, and protein abnormalities can quickly result in mortality if they are not resolved. Since many animals with gastrointestinal disease vomit or regurgitate, parenteral administration of fluids, medications, and potentially even nutrition should be provided (see above). GI protectants such as sucralfate, famotidine, ranitidine and omeprazole, antiemetics including maropitant citrate and ondansetron, and prokinetics, such as metoclopramide, can all be used in treating gastrointestinal disease. Attempts to induce emesis with apomorphine, xylazine, or hydrogen peroxide have been unsuccessful. Simethicone has been used to reduce bloating. Mirtazapine, orally and rectally, has been used to promote appetite with variable effects. Probiotics have been used in animals on chronic antibiotics or with chronic gastrointestinal disease, but little is known about their efficacy in pinniped species (see **Chapter 27**). Parenteral nutrition (PN) was administered to six severely malnourished (third-stage starvation) northern elephant seal pups or weanlings, using AminosynTM amino acid solution and Intralipid® lipid emulsion. A 19-gauge spring-reinforced intravenous catheter was placed into the epidural sinus, and PN was administered with either a portable syringe pump housed in a water-resistant case secured to the patient by a harness, or by "bolusing" the solutions over a 1- to 2-hour time period three times daily while the seal was confined in a smaller area (approx. 1.5 × 1.5 m [5 × 5 ft.] area; Frankfurter et al. 2014). CBC, serum chemistry, and electrolyte values were monitored regularly, and antibiotics and fluids were administered concurrently. Serum glucose, insulin, and glucagon levels indicated likely appropriate metabolic responses. Reintroduction of tube-fed oral electrolytes was initiated within 3–5 days, followed by a semielemental diet (Emeraid Piscivore®) for several days and

gradual incorporation of ground fish slurries. Although only one out of the six seals ultimately survived to be released, PN appeared to be safe and well tolerated by these gravely ill animals, and may be of great benefit to severely nutritionally compromised animals.

Respiratory System

Respiratory disease is common in pinnipeds, and these species are capable of masking severe disease. Canine distemper virus (CDV) has caused epizootics of pneumonia and death in Baikal seals (*Phoca sibirica*) and Caspian seals (*Phoca caspica*), and phocine distemper virus (PDV) has most frequently been associated with epizootics in harbor seals with occasional smaller mortality events in gray, harp, and hooded seals (Duignan et al. 2014). Ocular and nasal discharges, cough, cyanosis of mucous membranes, dyspnea, diarrhea, fever, and central nervous signs, such as depression or seizures, are observed in affected seals. Subcutaneous emphysema of the neck and thorax may occur as a sequelae to pulmonary damage, and seals may have difficulty swimming and diving (Siebert et al. 2010). Antemortem diagnosis may be detected by rising serum antibody titers, though animals may succumb to disease prior to developing a strong serologic response. Virus isolation is difficult, yet necessary to confirm identification of the virus. Treatment consists of supportive care, and controlling secondary bacterial infections that commonly cause death in infected seals (Baker and Ross 1992). Antibiotics effective against *Bordatella bronchiseptica*, *Corynebacterium* spp., and *Streptococcus* spp. are recommended. Although clinical recovery is documented, CDV has been isolated from asymptomatic carriers (Lyons et al. 1993). No commercially available vaccine for PDV currently exists, but commercially available attenuated CDV vaccine has been used to immunize stranded gray and harbor seals (Carter et al. 1992). Experimental inoculation of harbor seals with inactivated and subunit CDV vaccines have provided some protection from clinical disease (Visser et al. 1989, 1992; Van Bressem et al. 1991; Quinley et al. 2013). Most recently, vaccination of the wild population of Hawaiian monk seals (*Neomonachus schauinslandii*) was undertaken, because of the animals' low abundance, the fact that the population is naive to PDV, and that they have potential for exposure to a devastating PDV or CDV epizootic (Aguirre et al. 2007).

Influenza virus has also caused epizootics in harbor seals, with clinical signs similar to those in seals with PDV and CDV (Geraci et al. 1982; Anthony et al. 2012). These included dyspnea, lethargy, blood-stained nasal discharge, and subcutaneous emphysema, with pneumonia as the predominant postmortem lesion. There is evidence for interspecies transmission between birds, seals, and humans, suggesting that seals can both become infected and transmit influenza viruses to other species (Webster 1981; Goldstein et al. 2013). Phocine herpesvirus-1 (PhHV-1) has caused pneumonia in neonatal harbor seals in rehabilitation (Borst et al. 1986), while another herpesvirus was isolated from a California sea lion with acute hemorrhagic pneumonia (Kennedy-Stoskopf et al. 1986). Diagnosis of both infections is based on viral isolation, and treatment is supportive. Harbor seals with pneumonia associated with influenza virus were also infected with a mycoplasma, so therapy with antibiotics such as tetracyclines may be beneficial (Geraci et al. 1984).

Bacterial pneumonias are common in seals and sea lions, both as primary infections and secondary to viral and lungworm infections. A variety of organisms may be involved, although Gram-negative organisms are most common (Keyes, Crews, and Ross 1968; Sweeney 1986a; Spraker et al. 1995; Thornton, Nolan, and Gulland 1998; Haulena et al. 2006; Jang et al. 2010). Clinical signs include tachypnea, dyspnea, lethargy, and cough. Diagnosis is based upon auscultation of the chest, radiography of the lung fields, and bronchoscopy. Treatment with the appropriate systemic antibiotic may be based upon prediction of the likely organism, or culture and sensitivity of organisms from tracheal or bronchial washes (Johnson, Nolan, and Gulland 1998). Mucolytics such as acetylcysteine, and bronchodilators such as albuterol and aminophylline, have been used regularly on stranded harbor seals and California sea lions in rehabilitation.

Pneumonia in otariids may occur with heavy infestation of *Parafilaroides decorus*, although asymptomatic infection is common in young animals. *Parafilaroides gymnurus* infects alveoli of phocids, and *Otostrongylus circumlitus* may cause obstructive bronchitis and bronchiolitis in harbor, harp, and ringed seals, and yearling northern elephant seals (see **Chapter 21**). The degree of inflammatory response to *Parafilaroides* infections varies from none to marked suppurative and granulomatous pneumonia. Reaction may be more severe to dead and degenerate worms. Diagnosis depends upon detection of larvae in feces or sputum. Treatment with fenbendazole or ivermectin removes infection, but in severe cases, simultaneous treatment with antibiotics and either corticosteroids or nonsteroidal anti-inflammatories is strongly recommended to control secondary bacterial infections and reduce the inflammatory response to dying parasites. Interestingly, a *Brucella* spp. isolate was obtained from the lung of a harbor seal with *Parafilaroides* spp. infestation (Garner et al. 1997). Histological examination revealed most of the inflammation and *Brucella* spp. to be around the dead parasites. A more recent study confirmed these findings in stranded harbor seals (Lambourn et al. 2013); however, the role of *Parafilaroides* spp. in the epidemiology of *Brucella* infections remains unclear.

Pulmonary granulomas due to infection with *Mycobacterium pinnipedii* have been reported in captive and wild pinnipeds (Forshaw and Phelps 1991; Bastida et al. 1999; Cousins et al. 2003; Jurczynski et al. 2011). An enzyme-linked immunosorbent assay (ELISA) test (Cousins 1987) and intradermal tuberculin tests have been used for diagnosis of infection in live pinnipeds, although interpretation of results is difficult (Needham and Phelps 1990; Jurczynski et al. 2012), and successful treatment of

clinical cases has not been documented. Similar lesions may also result from fungal infections. *Coccidioides immitis* and *C. posadasii* infections are not uncommon in pinnipeds throughout California (Fauquier et al. 1996, Huckabone et al. 2015), and *Cryptococcus* spp. (Mcleland et al. 2012) and *Blastomyces dermatitidis* (Zwick et al. 2000) infections have also been diagnosed. Diagnosis is usually made postmortem based on histological detection of organisms and culture as disease is generally advanced. Treatment has rarely been described, but a walrus with coccidioidomycosis was treated successfully with voriconazole for years (Schmitt and Procter 2014).

Cardiovascular System

Anemia is common in young otariids as a consequence of hookworm (*Uncinaria* spp.) infestation, or secondary to malnutrition, and has also been reported in Mediterranean monk seals (A. Komnenou, pers. comm.). Affected animals are weak, are occasionally dyspneic, and have pale mucous membranes. Diagnosis of hookworm infestation is based on detection of ova in feces (see **Chapter 21**), although animals may remain anemic for weeks after patent infection ceases. Treatment with anthelmintics and supplementation with iron and vitamin B12 is usually effective. Nonregenerative anemia is seen in California sea lions as a consequence of chronic renal damage, usually as a result of leptospirosis (see below). Disseminated intravascular coagulation (DIC), characterized by bleeding from the nares, hematoma formation, thrombocytopenia, hypofibrinogenemia, and extended clotting times, is relatively common in stranded northern elephant seals (Gulland et al. 1996b). It may occur with septicemia or vasculitis associated with migrating *Otostrongylus* larvae (Gulland et al. 1997a). Diagnosis of *Otostrongylus* infestation in live seals during the prepatent period is not currently possible, although clinical signs and season of occurrence are highly suggestive of infection. Clinicopathologic changes include elevated white blood count greater than 40,000 with a left shift, reduced platelet count, and increased aminotransaminases (ALT and GGT). Serum amyloid A, an acute phase protein, has the potential to serve as a diagnostic tool in prepatent *Otostrongylus* infections in elephant seals prior to the development of clinical signs (Sheldon et al. 2015). *Otostrongylus* worms have also been found in the right ventricle and pulmonary arteries in California sea lions causing similar clinical signs as seen in elephant seals (Kelly et al. 2005). Elephant seals with signs of DIC are treated with antibiotics, corticosteroids, and supportive care, but therapy is rarely successful. A lysine analogue antifibrinolytic drug, ε-aminocaproic acid (EACA), shows promise in treating the bleeding associated with prepatent *Otostrongylus* arteritis in northern elephant seals (Kaye et al. 2016). Other cases of anemia have included hyperestrogenism-induced medullar aplasia in a gray seal (Lacave, pers. comm.) and hemolytic anemia of unknown origin in a northern fur seal (Chelysheva and Romanov 2008).

Cardiac insufficiency in pinnipeds can be caused by cardiomyopathy related to toxin or capture stress, bacterial endocarditis, and heartworm infestation. In California sea lions, domoic acid (DA) toxicosis can cause a degenerative cardiomyopathy associated with decreased cardiac contractility and cardiac output (Zabka et al. 2009; Barbosa et al. 2015). Serum troponin-I and EKG tracings are not predictive of the severity of DA-associated cardiomyopathy. Electrocardiograms described in otariids and phocids are consistent with other animals, and the ventricular activation (QRS complex orientation) falls into category B with swine, horses, and cetaceans (Hamlin, Ridgway, and Gilmartin 1972; Dassis et al. 2016). Both ventricles depolarize simultaneously in bursts of canceling activity, leading to potential limitations of using ECGs in pinnipeds for diagnosing cardiac pathology. Echocardiography is currently the only tool for diagnosis of cardiac insufficiency in sea lions affected by domoic acid. In South American fur seal pups, capture stress has also induced cardiomyopathy, characterized by myocardial contraction band necrosis and endothelial disruption (Seguel et al. 2014). Bacterial endocarditis caused by *Staphylococcus aureus* and *Escherichia coli* has been documented as a cause of mortality in seals and sea lions and should be ruled out for cardiac insufficiency in pinnipeds (Kim et al. 2002, Chinnadurai et al. 2009).

A variety of microfilarid species have been documented in pinnipeds (see **Chapter 21**). Infection by either the canine heartworm *Dirofilaria immitis* or the phocid parasite *Acanthocheilonema spirocauda* may cause dilatation of the pulmonary artery and right ventricle, and can be detected radiographically. Microfilaria observed in blood smears must be distinguished from those of the noncardiopathogenic fascial worm, *A. odenhali*. The vast majority of microfilaria noted in wild California sea lions are *A. odenhali*. Commercially available canine heartworm antigen tests cross-react with *A. odendhali* in California sea lions, and results from these tests should be interpreted with caution when diagnosing heartworm in sea lions (Krucik, Van Bonn, and Johnson 2016). Successful treatment of documented heartworm cases has not been described. Preventive treatment of captive animals in *D. immitis* endemic regions with ivermectin at 0.6 mg/kg every month during the mosquito season is recommended, as well as removal of lice from stranded animals, as the seal louse *Echinophthirius horridus* has been shown to transmit *A. spirocauda* (Geraci et al. 1981).

In phocids, patency of the foramen ovale (f.o.) and ductus arteriosus (d.a.) occurs longer after birth than is described in terrestrial mammals. The f.o. may be patent up to 7 weeks of age, and the d.a. may be patent up to 6 weeks of age without evidence of clinical consequence (Dennison et al. 2011a). Patency should only be considered abnormal if there is evidence of cardiac enlargement or hemodynamic derangement, and care should be taken not to fluid-overload the pups during initial days of treatment.

Urogenital System

Leptospirosis, caused by pathogenic spirochetes within the genus *Leptospira*, is well recognized in free-ranging California sea lions stranded in northern California. Although rarer, infection has also been reported in Steller sea lions, northern fur seals, Pacific harbor seals, and northern elephant seals (Smith et al. 1977; Stamper, Gulland, and Spraker 1998; Stevens, Lipscomb, and Gulland 1999; Colegrove, Lowenstine, and Gulland 2005; Cameron et al. 2008). Antibodies, providing evidence of prior exposure, have been detected in Hawaiian monk seals, New Zealand fur seals, and a bearded seal (MacKereth et al. 2005; Aguirre et al. 2007; Calle et al. 2008). Clinical signs are best documented in California sea lions, and include depression, anorexia, polydipsia, dehydration, vomiting, diarrhea, melena, oral ulcers, abdominal pain, and muscular tremors. Hematological changes include elevations in blood urea nitrogen, phosphorus, sodium, creatinine, and neutrophil count. However, asymptomatic chronic infection and leptospire shedding also occur (Prager et al. 2013, 2015).

Diagnosis is based on clinical signs and serum chemistry abnormalities consistent with leptospirosis, in addition to the absence of clinical signs suggestive of the other causes of azotemia, (i.e., amyloidosis, urogenital carcinoma, pyelonephritis, or severe dehydration). Infection can be confirmed through PCR detection of *Leptospira* DNA, or culture and isolation from urine or kidney tissue (Ahmed et al. 2012). To date, the only serovar isolated from free-ranging California sea lions and northern fur seals is *L. interrogans* serovar *pomona*, while both *L. interrogans* serovar *pomona* and *L. kirschneri* have been isolated from northern elephant seals (Smith et al. 1977; Cameron et al. 2008; Zuerner and Alt 2009; Delaney et al. 2014). Several other *L. interrogans* serovars have caused renal disease in both managed and free-ranging pinnipeds (Calle et al. 2003; Kik et al. 2006; Patchett et al. 2009). Sea lions with single microscopic agglutination test (MAT) titers over 1:100 are considered exposed, but clinically active cases of *L. interrogans* serovar *pomona* in sea lions usually have titers greater than 1:3200 (Colagross-Schouten et al. 2002). Cross-reaction with other *Leptospira* serovars with the MAT is common, and therefore a positive MAT titer against a particular serovar does not confirm infection with that serovar.

Treatment consists primarily of supportive care directed toward the clinical manifestations of the individual animal, such as fluids, gastric protectants, and analgesics. In vitro, *Leptospira* are susceptible to many antibiotics, including those in the penicillin and tetracycline families, and a 10- to 14-day course is recommended. In California sea lions, there is currently no evidence that penicillin-based antibiotics alone are effective in clearing an infection in vivo (Prager et al. 2015); however, a longer duration treatment course with tetracycline antibiotics may be effective in elimination of leptospiruria. Clinical signs and blood values can resolve with treatment, but, due to the severity of renal disease, roughly two-thirds of California sea lions presenting with clinical leptospirosis die despite treatment (Gulland et al. 1996c).

Renal disease may also occur as a consequence of renal calculi, congenital renal aplasia, and amyloidosis (see **Chapter 14**). California sea lions diagnosed with amyloidosis exhibited signs of renal disease, with elevated BUN, creatinine, and phosphorus, plus hypoalbuminemia (Chinnadurai et al. 2008; Colegrove et al. 2009). Premortem diagnosis of amyloidosis requires a renal biopsy, and diagnoses of congenital renal aplasia and renal calculi require radiography and ultrasound. Treatment of these rare conditions has not been reported.

Urogenital tumors are common in free-ranging California sea lions (see **Chapter 14**). Clinical signs in these animals usually result from pressure on ureters and invasion of local organs. Initial presentation is often nonspecific with signs of malnutrition, and signs suggestive of metastatic cancer include posterior paresis, perineal and scrotal edema, ascites, and vaginal or rectal prolapse. Ultrasound often reveals hydroureter and hydronephrosis caused by ureter obstruction. Additional diagnostic tools include abdominocentesis and cytology, radiology, and laparoscopic biopsy techniques. Treatment has not been attempted. Other tumors of urogenital origin include renal cell carcinoma in a Steller sea lion (Romanov et al. 2015), choriocarcinoma in a California sea lion (Fravel et al. 2013), and an ovarian interstitial cell tumor in a South American sea lion (Biancani et al. 2010).

Abortions and stillborn pups are frequently observed on pinniped rookeries. Leptospires (Gilmartin et al. 1976), herpesviruses (Dietz, Heide-Jorgensen, and Harkonen 1989), caliciviruses (Smith and Boyt 1990), *Coxiella burnetii* (Lapointe et al. 1999), high levels of DDTs (Gilmartin et al. 1976), and domoic acid (Goldstein et al. 2009) have all been reported in aborting pinnipeds. Unlike cetaceans, *Brucella* spp. have not been found to be the causative agent of abortion in pinnipeds despite some investigation. An aborted California sea lion fetus had positive immunostaining for *Brucella* spp. in the respiratory and reproductive tissue, and the placenta was culture positive; however, the dam suffered from concurrent DA toxicity, and thus the primary cause of the abortion remains unknown (Sidor et al. 2008). Vaginal prolapse has been observed in California sea lions and Australian sea lions (*Neophoca cinerea*; Read et al. 1982). Treatment of the latter by ovariohysterectomy was successful. Uterine torsions and ruptures have been observed in California sea lions with DA intoxication, and were believed to be consequences of severe convulsions (Gulland et al. 2000). In pregnant California sea lions intoxicated with DA, fetal and amniotic fluid may act as a reservoir of DA initially ingested by pregnant females. Clinical improvement is often observed in the adults after abortion of the pup. To induce abortion, dexamethasone given at 0.25 mg/kg IM SID for 3–5 days is usually successful. If there is no response to dexamethasone, prostaglandin F2 alpha (i.e., dinoprost tromethamine; Lutalyse®) at 250 µg/kg IM SID for 3 days can be attempted, though a lower dose may also be successful.

Endocrine System

Few primary endocrine disorders have been documented in pinnipeds. Both hyper- and hyponatremia are common in stranded animals (see **Chapters 8 and 29**), and may be consequences of inappropriate stress responses. Adrenal necrosis resulting from infection by a herpesvirus, PhHV-1, has been associated with severe electrolyte and glucose abnormalities in stranded neonatal harbor seals undergoing rehabilitation (Gulland et al. 1997b).

Hypothyroidism has been suspected as attributing to obesity in captive California sea lions, and seems to be responsive to treatment with exogenous thyroid hormone. One adult, captive, California sea lion developed diabetes mellitus secondary to chronic pancreatitis. The animal was managed with glargine insulin injections, gastric protectants, and a high-protein, low-fat diet (Meegan et al. 2008). Environmental exposure of ringed seals to persistent organic pollutants in the Baltic Sea appears to affect endocrine homeostasis in these animals, though long-term health effects have yet to be described (Routti et al. 2010).

Severely malnourished animals are frequently hypoglycemic, and intravascular access in hypovolemic, minimally responsive, otariids can be challenging in an emergency situation. Fravel et al. (2016) showed that intraperitoneal (IP) administration of a dextrose bolus (500 mg/kg) will increase blood glucose levels to the same degree as an IV bolus, and thus can be administered during a hypoglycemic crisis. In the authors' experience, this technique has resulted in the successful revival of numerous hypoglycemic California sea lion and northern fur seal pups.

Eyes

Pinniped eyes are characterized by a large globe, prominent tapetum lucidum, rounded lens, and a narrow, tear-shaped pupil (Miller, Colitz, and Dubielzig 2010). The visual system of pinnipeds is adapted to both aquatic and terrestrial habitats, and most pinnipeds have good vision below the surface of the water in low light and above the surface in bright light (Wartzok and Ketten 1999). Pinnipeds have very active lacrimal glands producing constant tears that protect the cornea. Lack of tearing is often used as an indication of dehydration.

Eye lesions are common in both free-ranging and captive pinnipeds (see **Chapter 23**; Stoskopf et al. 1985; Schoon and Schoon 1992; Haulena, McKnight, and Gulland 2003; Colitz et al. 2010a,b). There may be an increased frequency of eye lesions in animals that are maintained in freshwater environments (Sweeney 1986b; Dunn et al. 1996), but water quality, oxidation by-products, UV light exposure, viral infections, underlying uveitis, and trauma all contribute to the multifactorial etiology of eye lesions in captive pinnipeds. Corneal lesions are most frequently encountered, followed by cataracts, traumatic injuries, infectious processes, and neoplasia (Miller et al. 2013). Captive otariids are frequently affected by a form of progressive keratitis, characterized by corneal opacities, edema, recurrent ulceration, and blepharospasm (Colitz et al. 2010a). Chronic exposure to sunlight appears to be an important risk factor (see **Chapter 31**), and progression of the disease is associated with secondary bacterial and fungal infections. Oral nonsteroidal anti-inflammatory drugs help to control pain and uveitis, and topical cyclosporine or tacrolimus appears to diminish recurrence of active disease. Treatment of active infection is imperative, and topical (triple antibiotic suspensions, serum, or platelet-rich plasma) and oral medications typically include doxycycline to stabilize the corneal stroma and speed re-epithelialization, and for its ability to be secreted in the tear film (Solomon et al. 2000; Freeman et al. 2013). Various bacteria have been cultured from traumatic lesions, conjunctivitis, and keratitis in pinnipeds (Thornton, Nolan, and Gulland 1998), and targeted therapy following culture and sensitivity is advised.

Visually impaired pinnipeds will thrust their vibrissae forward if investigating noises or new surroundings. Although normal pinnipeds will also do this, visually impaired animals tend to exaggerate the action and extend their vibrissae for prolonged periods of time. The menace response may be difficult to evaluate, since the vibrissae are very sensitive to air movement. Visually impaired animals, if placed into new surroundings, may not avoid obstacles well, but can accommodate very quickly using tactile and acoustic cues, making diagnosis of blindness difficult. Ophthalmic examination is difficult in pinnipeds because of a prominent nictitating membrane, strong eyelids, and the ability to retract the globe into the ocular cavity. Very narrow pupils limit visualization of internal eye structures such as the lens and retina, and pinnipeds do not tend to dilate their pupils very well when topical mydriatic agents are applied to the cornea. Retrobulbar block has been utilized to reverse the ventral rotation that frequently follows anesthesia, and this produces excellent mydriasis to examine the interior of the globe (Gutierrez et al. 2016).

Cataracts are common in pinnipeds, and frequently lead to synechiae formation, anterior prolapse, and rupture of the globe (see **Chapter 23**). The lenses of young animals may be removed by phacoemulsification (Colitz et al. 2011; Esson et al. 2015), but lenses in older animals are harder than in many terrestrial species, and lensectomy is required in the majority of cases (Colitz et al. 2011). Lensectomy has also been performed following globe perforation in phocids (Colitz et al. 2013).

Treatment of ocular lesions of pinnipeds is similar to that of domestic animals (see **Chapter 23**). However, the use of saline washes, most readily given as saltwater baths, appears to help decrease corneal edema. Both oral and topical anti-inflammatories and analgesics are helpful to treat pain associated with uveitis and corneal ulcers.

Nervous System

Numerous infectious agents have been identified as the cause of neurologic disease in pinnipeds. Viral encephalitis

secondary to morbillivirus, herpesvirus, and influenza are commonly reported, with small to very large-scale outbreaks in wild populations (Geraci et al. 1982, 1984; Phillippa et al. 2009; Philip Earle et al. 2011; Duignan et al. 2014; also see Respiratory section above and **Chapter 17**). Several cases of West Nile Virus (WNV) and Eastern Equine encephalitis (EEE) virus have also been described in captive phocids (Stremme 2003; Dalton, Dickerson, and Wigdahl 2004; Gentz and Richard 2004; McBride et al. 2008). A single case of rabies has been described in a ringed seal (Odegaard and Krogsrud 1981), and a novel parvovirus was detected in the brain of a harbor seal with meningoencephalitis (Bodewes et al. 2013). Clinical signs of these diseases are similar and may include depression, lethargy, ataxia, coma, recumbency, tremors, seizures, and coma. Other body systems, particularly the respiratory system, may also be affected. Some viral infections may be diagnosed on the basis of rising antibody titers, although confirmation is often made postmortem on histological examination of brain tissue, using immunoperoxidase and immunofluorescent techniques. Treatment is supportive and may include fluids, anticonvulsants, anti-inflammatory medication, and antibiotics for secondary infection. L-lysine supplementation did not alter the course of herpesvirus infection of harbor seal pups in a rehabilitation facility (Guarasci et al. 2010), and the efficacy of antiviral medications in these species is unknown. Vaccination is recommended against WNV using a recombinant canarypox vaccine, because it induced presumptively protective antibody levels in Steller sea lions (Tuomi et al. 2014). Harbor seals (Schmitt, pers. comm.) and several phocid species, including endangered Hawaiian monk seals, have been safely vaccinated with a recombinant DNA vaccine against canine distemper virus (see Respiratory section above).

Protozoal, bacterial, and fungal infections can cause similar neurologic signs. Any number of bacteria may infiltrate the nervous system through hematogenous spread. Various fungi including zygomycetes (Sosa et al. 2013; Barnett et al. 2014), *Coccidioides* spp. (Huckabone et al. 2015), *Cryptococcus* spp. (Rosenberg et al. 2016), and *Scedosporium apiospermum* (Haulena et al. 2002) have been detected in nervous and other tissues (see **Chapter 19**). *Toxoplasma gondii* and *Sarcocystis* spp. infections have been found in numerous wild pinniped species and some captive individuals (see **Chapter 20**).

Biotoxin exposure can also cause severe central nervous disease in pinnipeds. Neuronal necrosis in the hippocampus of California sea lions is caused by domoic acid (DA) exposure (Scholin et al. 2000; see **Chapter 16**). Common neurological signs include seizures, tremors, and ataxia. Repeated exposure to DA results in permanent hippocampal damage with memory loss and often a chronic epileptic state (Goldstein et al. 2008; Buckmaster et al. 2014; Cook et al. 2016). Treatment for biotoxin exposure is supportive and includes anticonvulsants, fluid therapy, and anti-inflammatory medication. Control of seizures with lorazepam (longer antiseizure effect than other benzodiazepines), midazolam, diazepam, or other benzodiazepines, and phenobarbital is beneficial. Current treatment for DA toxicosis at The Marine Mammal Center includes phenobarbital (4 mg/kg IM twice daily for 2 days, then 2 mg/kg IM or PO twice daily for 5 days) and lorazepam (0.2 mg/kg IM twice daily for the first 1–2 days, or longer as needed to control seizures). Subcutaneous (SC) fluids and dexamethasone (in the absence of ocular ulcer) are also generally administered for the first few days. An additional 0.2 mg/kg lorazepam is given IM if seizures do not stop 10–15 minutes after the first dose, and a third dose may also be given. If seizure activity does not stop after three doses of at least 0.2 mg/kg lorazepam (over approximately 45 minutes), euthanasia is recommended due to poor prognosis. Alpha-lipoic acid (ALA), a powerful antioxidant that crosses the blood–brain barrier, is also currently being administered to sea lions that strand at TMMC with DA toxicosis (10 mg/kg SC once daily), in an effort to reduce oxidative damage secondary to neuronal damage and necrosis. Successful control of idiopathic seizures in a captive adult California sea lion has been achieved with 1 mg/kg SID oral phenobarbital (Gage, pers. comm.), and a young California sea lion with intracranial structural anomalies using oral phenobarbital 4 mg/kg once daily (Dold et al. 2005).

Various anomalous and developmental brain lesions have been identified in pinnipeds. Pneumocerebellum, attributed to gas bubble formation, was noted in two stranded California sea lions (Van Bonn et al. 2011, 2013). Intracranial space-occupying lesions, usually tumors, have caused seizures in captive sea lions. Although hydrocephalus occurs in young stranded elephant seals (Trupkiewicz, Gulland, and Lowenstine 1997), sudden death, rather than neurological signs, usually occurs. Other reported congenital and anomalous neurologic abnormalities in various pinnipeds include hemicerebral anomalies (McKnight et al. 2005), bilateral caudate nucleus inflammation (Dennison et al. 2011b), cerebral infarction (Stevens et al. 2010), and discospondylitis (Tuomi et al. 2004); and multicentric neurofibromatosis was diagnosed in a geriatric California sea lion (Rush, Ogburn, and Garner 2012).

Thiamine deficiency is common in pinnipeds fed with frozen fish, particularly fish with high thiaminase content, and can result in polioencephalomalacia with acute neurological signs. Antemortem diagnosis is based on clinical signs and lack of supplementation, coupled with diet evaluation and response to supplementation, as clinicopathologic findings are generally nonspecific (Croft et al. 2013). Thiamine-dependent enzyme activity (transketolase) in blood and tissue samples can also be measured. Hyponatremia presents with similar clinical signs, and is also diagnosed by a combination of history of lack of supplementation (particularly in freshwater), plasma biochemistry, and response to treatment with sodium chloride (Geraci 1972b). Electrolyte imbalances associated with renal disease and/or nutritional deficiencies may also cause neurological signs (see **Chapters 8 and 29**). Selenium toxicosis was reported for several captive California sea lions that were fed with a diet later found to be high in selenium (Edwards et al. 1989).

Surgery

Surgical procedures for pinnipeds are generally comparable to those of canids with some species-specific adjustments. Commonly performed procedures are described below, and many are summarized in Higgins and Hendrickson (2013). Ocular surgery is discussed in **Chapter 23**.

Surgical procedures of the integument generally include mass removal, biopsy collection, and wound debridement and care. Standard sterile and surgical technique should be followed, including clipping hair from the affected site, sterile preparation of the incision site, and sterile procedure. Care should be taken with fur seals to minimize clipping, due to these species' reliance on fur for thermoregulation. An alternative to clipping in fur seals is to part the hair down the incision line using a tight comb, such as a flea comb, and apply betadine gel to prepare the skin, a technique used in sea otter surgical procedures (gel prepared by combining 12 ml betadine solution with 4.5-ounce sterile, water-based, lubricant; Murray, pers. comm.).

As pinnipeds generally recline on, and often ambulate on their ventral surface, surgical approach should be dorsolateral when possible to avoid contact of the incision site (postoperatively) with the ground. The thick hypodermis (blubber layer) often necessitates good retraction for proper exposure. Closure of skin and deeper layers should include tension relieving suture patterns in most areas, due to high tension and pressure on tissues. The hypodermis has poor holding capacity, and in healthy individuals is at least several centimeters thick, and even thicker in more robust animals. Tension-relieving suture patterns in this location are particularly important, as this layer must be closed to reduce dead space. Incisions that are full thickness (into the abdomen), or deep tissues, should be closed with four or five layers to help prevent dehiscence. Staples and sutures are both effective in closing the skin. Pain control with opioids and/or NSAIDS is strongly recommended perioperatively and postoperatively for most invasive procedures (see **Chapter 27** for drugs and dosages).

The duration of time to return an animal to water postoperatively varies greatly, though is only occasionally reported. Return to water may depend largely on the procedure, species, status (free-ranging vs. captive), and incision location. Free-ranging California and Steller sea lions with satellite tag transmitters implanted into the abdomen were returned to enclosures with access to water within hours postsurgery (Horning et al. 2008). None of the animals experienced dehiscence or infection of the surgical site. In the authors' experience, contamination of surgical sites with urine and feces, especially over the abdomen and extremities, can lead to infection and dehiscence; thus, most animals are allowed full-time access to water within a matter of days to a week. Alternatively, animals may be given access to water daily for restricted periods of time for hygiene, feeding, and comfort. Contamination of a surgical site by waterborne pathogens is also possible; thus, regular monitoring of incision sites is required. If not allowed access to water, animals should be kept in a clean, dry area with a smooth surface, particularly if surgery was performed on the ventrum or extremities.

Surgery of the musculoskeletal system frequently involves long bone or phalanx repair or amputation, and skull trauma including dental surgery. Fracture repair of long bones is rarely reported in the literature. One report describes the placement of a string-of-pearls locking plate, impregnated with antibiotics, to repair a closed, complete transverse diaphyseal fracture of the tibia of a 2-week-old gray seal (Hespel et al. 2013). A yearling California sea lion that presented with osteomyelitis of the left carpus was successfully released following fusion of the joint, using external fixation (Field, unpubl. data; **Figure 41.1**).

Initial systemic antibiotic treatment of the infection with multiple different antibiotics was unsuccessful; thus, polymethylmethacrylate (PMMA) beads impregnated with amikacin were placed around the joint, two rows of pins were placed in the radius and metacarpals, and PMMA bars made of thermoplastic polymeric material were placed to stabilize the joint (**Figure 41.2a and b**).

The fixator was removed 6 weeks later, following successful joint fusion.

Amputation of phalanges, digits, or limbs is not uncommon when bone is exposed or infection cannot be controlled with antimicrobial drugs. A nerve block using a local anesthetic, such as lidocaine or bupivacaine, is strongly recommended for both pain control and to help reduce minimal alveolar concentration (MAC) for general anesthesia. Phalanx amputation is common and similar to other species. It is of particular importance to preserve as much of the surrounding tissue as possible by undermining around the affected bone(s) to allow adequate subcutaneous tissue and skin

Figure 41.1 External fixation device on the dorsal surface of a California sea lion yearling front flipper.

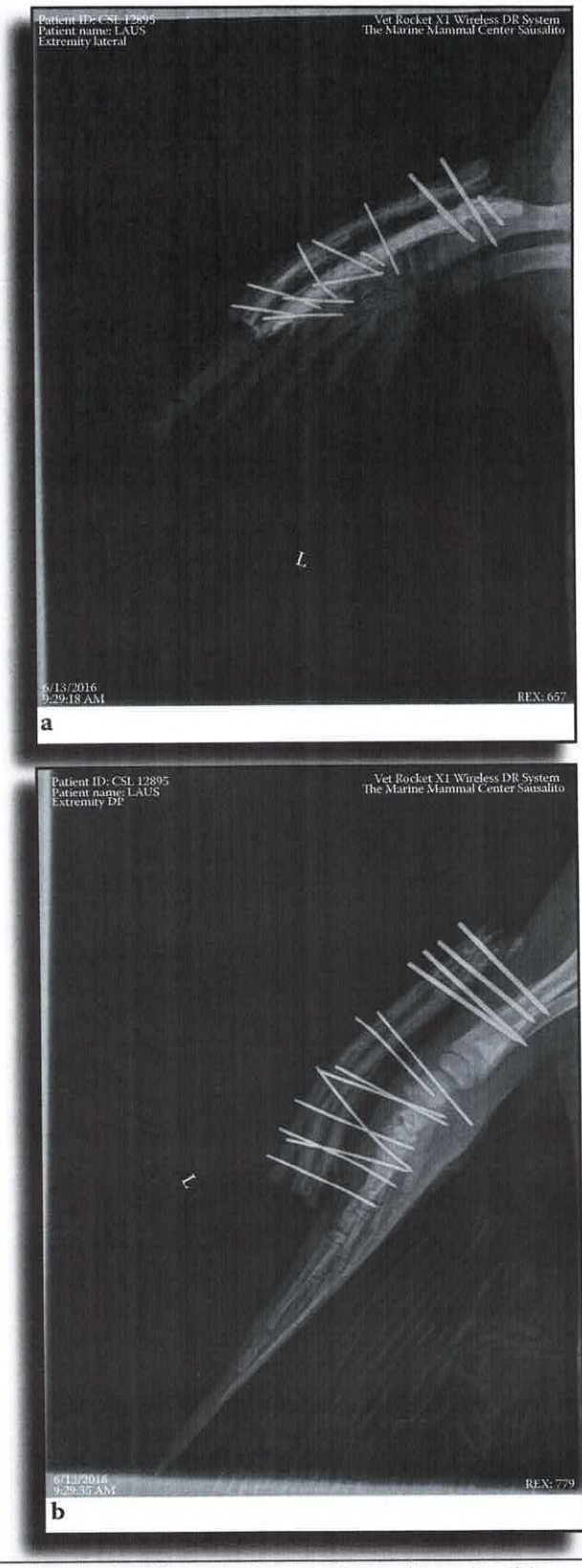

Figure 41.2 (a, b) Dorsoventral and lateral radiographs of the external fixation device of California sea lion yearling with osteomyelitis incorporating the left carpus. Polymethylmethacrylate antibiotic impregnated beads are visible in the joint.

closure. A dorsal approach is recommended, since these distal sites are in regular contact with the ground, and the dorsal surface is easily visualized for recheck examination. If insufficient tissue is present to entirely close the end of a joint or limb, mid-diaphyseal amputation of the next proximal bone is recommended to prevent high tension and dehiscence of the incision site.

Partial or complete front or hind flipper amputations have also been performed with return to normal, or near normal, locomotion. The impact of amputation surgery on the ability of free-ranging animals to successfully forage for live prey and have adequate mobility on land should be strongly considered prior to the procedure, and must be assessed prior to releasing the animal. **Figure 41.3** depicts the successful amputation of the right hind flipper of a yearling California sea lion at the level of the coxofemoral joint subsequent to severe limb damage (**Figure 41.4**).

The joint was approached from the lateral aspect and care was taken to isolate and ligate the blood vessels around the joint (Da Costa Gomez, pers. comm.). Soft tissue over the tarsus/tibiotarsus is particularly limited and difficult to close; thus, mid-diaphyseal amputation of the tibia and fibula with

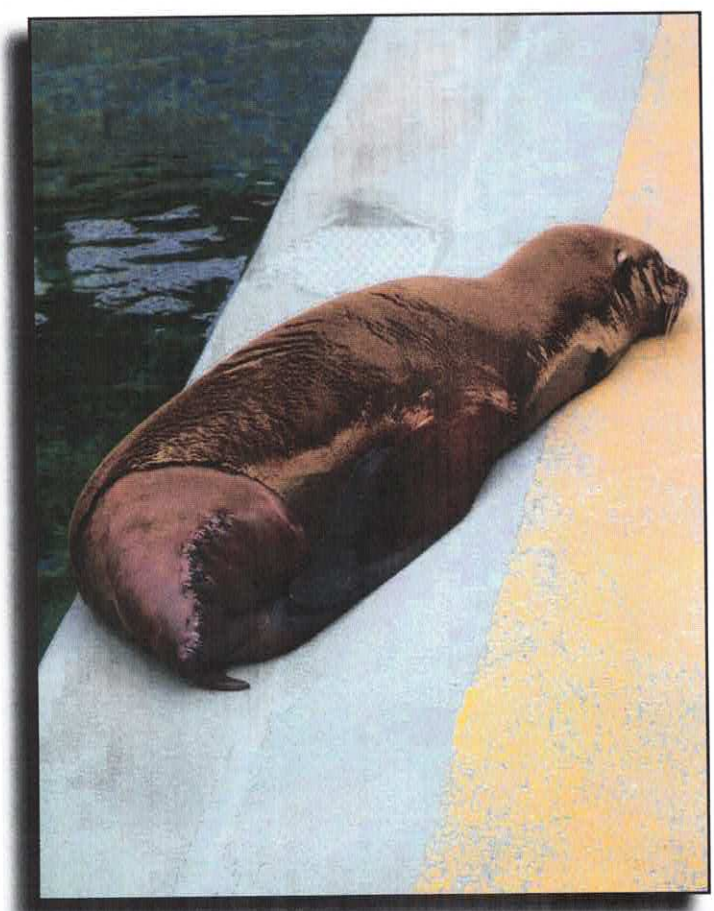

Figure 41.3 California sea lion yearling post-amputation of right hind flipper at the coxofemoral joint using a lateral surgical approach.

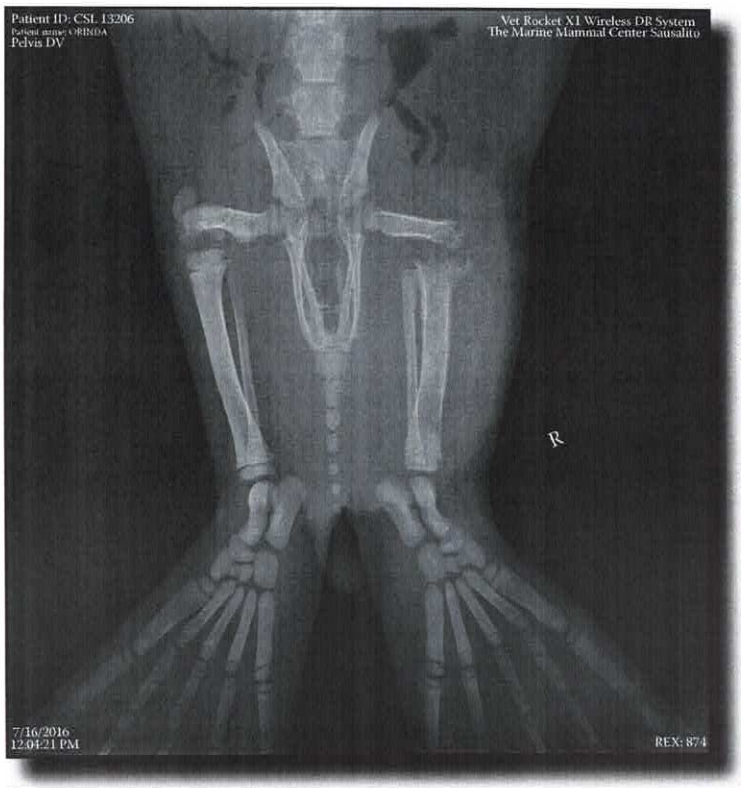

Figure 41.4 Dorsoventral radiograph of hind limbs of a California sea lion yearling prior to leg amputation. Findings include right femoral distal diaphyseal fracture with metaphyseal and epiphyseal lysis, displaced and lytic patella, and proximal tibial fracture with epiphyseal and diaphyseal lysis.

closure using surrounding muscle and skin is recommended. This technique is described by Garcia et al. (2015) for harbor seal weanlings suffering from necrotizing infections of the tarsus or tibiotarsal joint. Bandage maintenance is challenging in these species, even when maintained out of water, given their fusiform body shape and their aquatic environment.

Surgeries involving the pinniped skull are generally confined to dental surgery and mandibular repair secondary to trauma. Fractured and loose teeth should be extracted (see **Chapter 22**). Lewer et al. (2007) described a case of closed left mandibular fracture in a geriatric harbor seal, which healed successfully after 12 months with the use of an oral dental acrylic splint and cerclage wire. A bilateral mandibular fracture in a 12-year-old South African fur seal was reduced and plated using two 3.5 mm positioning and compression device (PCD) plates and autotype screws (Flanagan et al. 2009). Both fractures resolved within 2 months. A harbor seal pup that stranded with a closed, complete fracture of the right caudal mandible developed a bony sequestrum at the fracture site. A ventral approach was used to debride the lesion and place an intraosseous wire. Canine trabecular bone powder and equine lamellar cortical bone matrix were used, along with platelet-rich plasma as a graft. The seal did well postoperatively (Rosenberg et al. 2015).

Surgery of the respiratory tract of pinnipeds has not been reported in the literature, despite common occurrence of severe respiratory disease. Tracheal perforation secondary to entanglement in monofilament fishing line occurs occasionally in free-ranging pinnipeds. Successful repair of tracheal perforation in California sea lions has been accomplished by initial debridement to freshen wound edges, and release incisions of existing scar tissue, to decrease tension. Mucosal margins of the trachea are closed with simple interrupted sutures, if possible. Overlying muscle layers are closed with interrupted horizontal mattress and cruciate sutures followed by standard closure of subcutaneous and skin layers with tension-relieving sutures (Da Costa Gomez, pers. comm.)

Abdominal surgery is performed most commonly for gastrotomy, or for procedures involving the reproductive tract. Esophageal surgery for fishhook removal has been performed in Hawaiian monk seals, as well as partial glossectomy of necrotic lingual tissue for the same reason (Barbieri et al. 2013; Levine, pers. comm.). Gastric foreign bodies are not uncommon in pinnipeds and are often found incidentally on necropsy of free-ranging pinnipeds with no evidence of associated pathology. Many animals will regurgitate or vomit foreign objects, or objects may be retrieved using gastroscopy. Surgery is indicated for gastric impaction, gastric perforation, fishhooks embedded in tissue, or other severe disease. The pinniped stomach is strongly u-shaped, but is otherwise similar to the canine. Gastrotomy for fishhook removal has been performed in numerous Hawaiian monk seals (Levine and Barbieri, pers. comm.). Gastric impaction by rocks, sand, or other abnormal ingesta is frequently noted in juvenile harp and hooded seals that strand on the east coast of the United States (Helmick, Dunn, and St. Aubin 1995) and western European coast (Alonso-Farre et al. 2011). The reason for foreign material ingestion is unknown, and seals are often critically dehydrated with severe gastric disease on presentation. Rehydration and administration of mineral oil and small amounts of water through an orogastric tube may allow sand and smaller rocks to pass, or in some cases rocks can be removed through endoscopy or laparoscopically. Gastrotomy was successful in treating rock impaction in a stranded juvenile harp seal with concurrent severe pneumonia (**Figure 41.5**).

This harp seal was maintained on IV fluids and antibiotics, IM famotidine, and oral fluids and mineral oil for nearly 3 weeks until the pneumonia had resolved sufficiently to allow surgery (Field, Schuh, and Tuttle 2009).

Intestinal surgery has also not been reported in the literature, though a variety of intestinal diseases, including gastric torsion, gastric or mesenteric volvulus, intussusception, obstruction, and other surgical conditions have been found on necropsy. Laparotomy on a severely debilitated free-ranging Hawaiian monk seal with a jejunal intussusception was initially successful; however, the animal died several days later secondary to mesenteric torsion (Levine and Barbieri, pers. comm.). Though these severe intestinal diseases

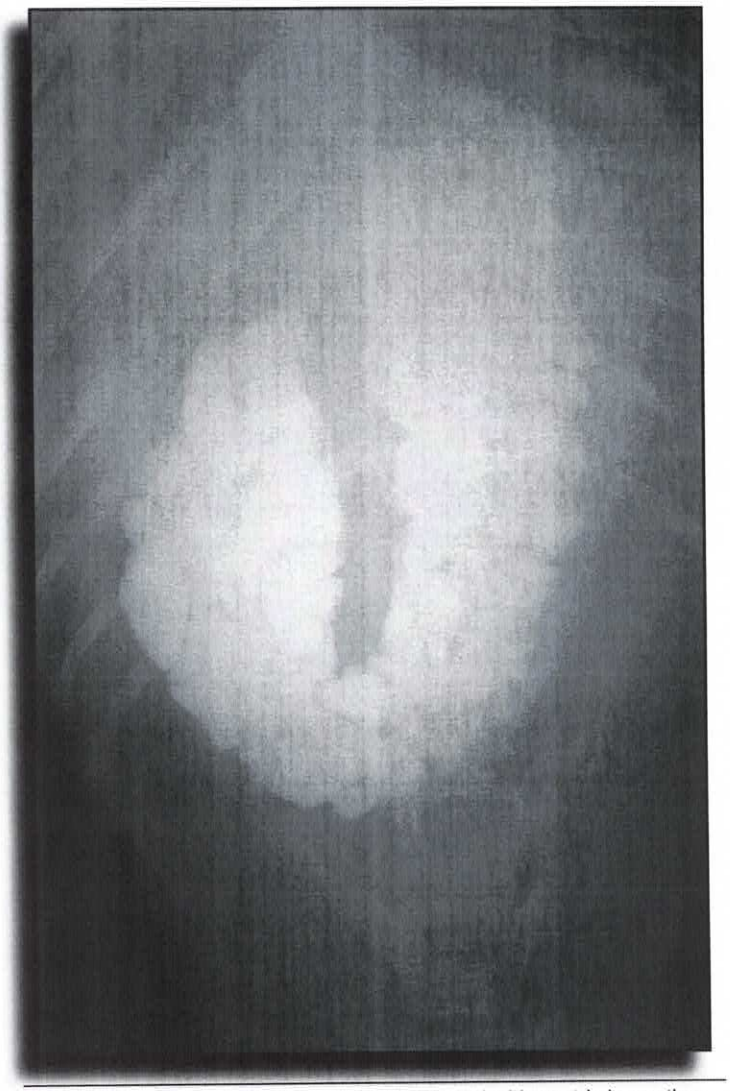

Figure 41.5 Radiograph of a juvenile harp seal with gastric impaction from rock ingestion.

appear relatively uncommon, a successful outcome generally requires rapid diagnosis and surgical intervention.

Surgery of the reproductive tract has been reported for both male and female pinnipeds. Ovariohysterectomy was performed in a South Australian fur seal with a vaginal prolapse; the procedure was performed similar to that of a dog, with a ventral midline incision, exteriorization of the uterus, ligation of the ovarian, broad ligament and uterine vessels, and ligation of the uterine body at the cervix, using overlapping horizontal mattress sutures followed by oversewing of the uterine stump (Read et al. 1982). Caesarian section has been performed in California sea lions (Schmitt, pers. comm.) and in an 8-year-old harbor seal with uterine torsion. In this case, the dam became acutely lethargic and anorexic, and the fetal heart rate and movement were reduced. The uterine torsion prevented cervical dilation and pup expulsion, and the pup was successfully resuscitated following surgical intervention. The uterus was closed in two layers, the abdomen in four layers, and the skin effectively sealed with Dermabond (Gili, pers. comm.). Most other reported abdominal procedures, including gastropexy for a hiatal hernia, ovariectomy (Dover et al. 2004), and tissue biopsies, have been performed laparoscopically (see **Chapter 25**).

In male pinnipeds, orchiectomy has been performed in a number of otariids (scrotal testes); however, this procedure is rarely performed in phocids because their testes are para-abdominal. Otariid testes can retract strongly, so castration of mature males during breeding season when the testes have descended may be advantageous. If out of season or if animals are immature, testes can be pushed into the scrotum with rectal manipulation. A prescrotal incision is generally recommended, and the procedure can be done closed or open. Open is recommended for mature animals to ensure adequate ligation of larger vessels. Two cases of partial penis amputation subsequent to persistent paraphimosis were reported in South African fur seals (Lacave, Guglielmi, and Mantratz 2008). One animal required partial os penis amputation and urethral reconstruction; the other required amputation of the tip of the penis; both recovered well.

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