Stranded juvenile pinnipeds are found frequently on coasts. It is not known in most cases whether these animals are abandoned, lost, or injured. Whatever their source, they must be fed, which in most instances means they must be force fed. Usually, they are newly weaned or about weaning age, thus they should be fed a diet resembling mother's milk, as well as 1 containing some solid nutrients to introduce them to eating fish. The following diet has been used successfully:

Lactated Ringer's solution	120 mL
Predigested protein (protein hydrolysate 1 g/30 mL)	25 mL
"Similac"	60 mL
Whipping cream	240 mL
Thiamine (500 mg)	1 mL
"Nutri-cal"	11.6 g
Herring	550 g

Protein	9.4%
Fat	18.5%
Water	70.0%
Carbohydrate	1.3%
Ash	0.84%
Calcium	0.06%

It is known that sea lion milk contains no lactose and that the species lacks intestinal dissacharidases (*see* above). However, the above diet has not caused any noticeable problems in the juveniles in which it has been used.

A much simpler diet for young seal pups is a blend of equal parts good quality fish and water to which has been added a pint of heavy cream and some multivitamins. This is fed via stomach tube at a rate of 8% of body wt/day in 4 divided doses. This diet is continued until pups start consuming whole fish at 3–5 weeks of age.

MANAGEMENT, HUSBANDRY, AND DISEASES OF MINK AND FOXES

MINK MANAGEMENT

The ranch for mink (*Mustela vison*) should be located on well-drained soil, well away from urban areas. Good husbandry and proper, regular manure disposal will help control odors. A guard fence around the farm aids in preventing the escape of mink, and keeping out feral or wild animals. Many wild species such as skunk (*Mephitis mephitis*) or raccoon (*Procyon lotor*) may act as vectors for diseases such as distemper.

Mink are housed individually in wire mesh pens raised above the ground. A nest box with a hole for entry is attached outside or placed within the pen. Wood used for the nest box should not be painted or treated with wood preservatives. Soft, awn-free marsh hay, chopped straw, wood shavings, or fine woodwool make suitable nest material. Nest boxes should be cleaned and nest material replaced as required, especially before a female whelps, and during cold weather.

Sheds may be used throughout the year, provided that they admit sufficient natural light to supply normal daylight hours and there is plenty of air circulation in the warmer months.

Mink feed may be supplied as a wet gruel placed on top of the wire or as a commercially prepared, dry, pelleted ration placed in feed hoppers. During the weaning and postweaning periods food is supplied on feeding trays placed on the floor of the run for small kits that cannot reach the top of the pen. Fresh water should always be available. Watering cups fastened to the outside of the pen with a lip protruding inside are commonly used. Automatic watering systems with individual nipples or flotation cups are used in sheds, temperature permitting. Cold storage facilities are necessary to freeze and store the meat portion of the ration. A day's supply of fish and meat byproducts is thawed, commercial cereal added, and the combined ration is mixed with water to a consistency that will remain on the wire of the pen without dropping through. Ready-mixed feeds are available in some areas. This may be delivered daily, ready to feed, or may be in frozen blocks, which are kept in cold storage and thawed as required. Dry pelleted diets are used on some ranches for part or all of the year. (*See also* NUTRITION: MINK, p 1185.)

Pelting usually is done in November or December. Several methods of killing are acceptable; including electrocution, carbon monoxide gas, or cervical dislocation. Nicotine sulfate injections and carbon dioxide inhalation are not considered to be humane.

Ranchers usually keep 1 male for each 5 female breeders. Mink are seasonal breeders; sexual activity is controlled by increasing periods of daylight. Artificial lights in the sheds must be used with caution since they may adversely affect photoperiod and interfere with the normal reproductive cycle. In the Northern hemisphere the breeding season begins in late February-early March and lasts about 4 weeks. Mating should occur within an hour after the female is placed in the male's pen. If fighting ensues they should be separated. Ovulation is induced by coitus. In females mated before mid-March it is standard practice to re-mate them after 7–8 days, often with an additional mating the following day; thus, individual females may be mated 2–3 times. Ova from 2 matings have been known to contribute to the same litter. There is a period of delayed implantation of the fertilized ova, so the apparent gestation period varies from 40–75 days.

Mink have 1 litter a year of 1–12 kits (average 4). Most litters are born during the last week in April and the first 2 weeks in May. Kits are blind, hairless, and weigh about 10 g when born, but grow rapidly throughout the summer to reach a weight of about 800 g (females) or 1,600 g (males) by October. Kits are weaned at about 6–8 weeks of age, and may be separated shortly thereafter and housed 1, 2, or 3 per pen. Adult mink are extremely agile, strong, and vicious. Handling requires the use of special leather gloves or wire catching cages.

DISEASES OF MINK

VIRAL DISEASES

ALEUTIAN DISEASE (Plasmacytosis)

A slow virus infection of mink, characterized by poor reproduction, gradual weight loss, oral and gastrointestinal bleeding, renal failure and uremia, and high mortality. All color phases of mink may be infected with Aleutian disease (AD), but light color phases genetically derived from the Aleutian color phase are most susceptible. The cause is a parvovirus not related to mink viral enteritis. Transmission occurs *in utero* and by direct or indirect contact with infected mink.

Following infection, mink frequently respond with marked increases in immunoglobulin levels. Immunoglobulins are unable to neutralize the AD virus and immune complexes form and deposit in various tissues, resulting in immune-complex glomerulonephritis and arteritis. Gross pathological changes include enlargement of the spleen; kidney changes, varying from swelling and

petechiation to atrophy and pitting; and enlargement of mesenteric lymph nodes. Histological lesions include plasma cell infiltration in the kidneys, liver, spleen, lymph nodes, and bone marrow; bile duct proliferation; membranous glomerulonephritis; and fibrinoid arteritis.

The disease is controlled through a test and slaughter program. Positive mink are identified by blood testing for specific antibody by counterimmunoelectrophoresis. All positive mink should be killed. Mink that are to be kept for breeding stock are tested in late fall (before selection of breeding stock and pelting) and in January or February (before breeding). New introductions to the herd should be tested.

There is no vaccination or effective treatment; positive females and their litters should be separated from the rest of the herd and pelted in season. The virus is present in the saliva, urine, feces, and blood of infected animals. Pens should be cleaned with 2% sodium hydroxide. Care should be taken to disinfect equipment after handling, vaccinating, or testing mink on infected farms. Wild birds and flies may serve as vectors, and their control is essential.

DISTEMPER

Mink of all ages are susceptible to canine distemper virus. The incubation period varies from 9–14 days. The virus may be recovered from infected mink 5 days before the appearance of clinical signs. Mink that have apparently recovered may continue to shed the virus for several weeks. Transmission may be direct (through contact or aerosol) or indirect (the virus may persist for a day or more in the environment).

Clinical signs include nasal and ocular discharge; hyperemia, thickening, and crustiness of the skin on the muzzle, feet, and ventral abdominal wall; neurological signs (convulsions and "screaming fits"); or a combination of these. At necropsy, in addition to skin lesions, there may be evidence of bronchopneumonia. Histological or fluorescent antibody examination may reveal intracytoplasmic or intranuclear inclusions or distemper antigen in epithelial cells of the urinary bladder, kidney, bile ducts, intestine, lung, trachea, and occasionally brain. Nonsuppurative encephalitis may be present in mink with neurological signs.

In outbreak situations, affected animals should be killed and the balance of the herd vaccinated as soon as possible. Mink kits should be vaccinated prophylactically at 9–10 weeks of age with a MLV vaccine. Adult mink should be vaccinated in alternate years. Immunization by either the parenteral or aerosol routes can be employed.

MINK VIRAL ENTERITIS

A highly contagious disease caused by a parvovirus related to but not identical with that of feline panleukopenia. Although the viruses of mink and feline enteritis are cross-immunogenic, they are not cross-infective under natural conditions. All ages are susceptible, but the disease is most serious in kits. Transmission usually occurs by the fecal-oral route, and the incubation period varies from 4–8 days.

Clinical signs include sudden anorexia; depression; watery, mucoid, blood-tinged diarrhea; dehydration; immunosuppression; and death. Characteristic gross lesions include a flaccid, dilated, hyperemic small intestine, containing liquid, fetid contents; some mink may die suddenly with no gross lesions. The main histological lesions include enteritis and lymphoid necrosis. Intestinal lesions are characterized by erosion of surface mucosa, blunting and attenuation of villi, and cryptal dilation. Ballooned epithelial cells may contain inclusion bodies similar to those of feline panleukopenia. Splenic and lymph node lesions include lymphoid depletion and necrosis.

Early in an outbreak, all mink showing signs should be killed and all clinically normal mink should be vaccinated immediately with a formalized tissue culture vaccine. Mink viral enteritis can be prevented by prophylactic vaccination programs. All mink should be vaccinated at 6–8 weeks of age with a combination mink viral enteritis-botulism vaccine. Annual vaccination is recommended in endemic areas. If female mink have experienced an outbreak or have been vaccinated against mink viral enteritis in January or February, their kits born in May and June should not be vaccinated until they are 11–12 weeks of age.

AUJESZKY'S DISEASE
(Pseudorabies)

Aujeszky's disease is reported occasionally in mink that are fed pork products contaminated with pseudorabies virus. Mortality may be high, and clinical signs are referable to the CNS (tonic and clonic convulsions; excitement, alternating with depression; and, in some cases, self-mutilation). The diagnosis is confirmed by virus isolation or serology. Since contaminated pork is the usual source of infection, all pork products should be cooked before they are fed to mink.

TRANSMISSIBLE MINK ENCEPHALOPATHY
(Mink scrapie)

Scrapie in mink is rare but has potential to cause high mortality in adult mink. The incubation period in experimental infections is 8 months or longer. Clinical signs are similar to those of scrapie in sheep (qv, p 607) and include hyperirritability, ataxia, compulsive biting, somnolence, coma, and death. Histologic lesions in brains of affected mink are similar to those of scrapie in sheep. Although mink have been experimentally infected by intracerebral inoculation of brain material from scrapie-infected sheep, and by feeding tissues from infected sheep, the means of natural transmission is unknown. Control measures cannot be suggested, except to exclude sheep byproducts from the ration in endemic areas.

EPIZOOTIC CATARRHAL GASTROENTERITIS

Millions of mink have been affected by an agent (most likely a virus) that causes an acute catarrhal gastroenteritis. The disease usually occurs in adult dark mink. Outbreaks occur most frequently during periods of stress, ie, during early fall molting, spring mating, and whelping seasons. The clinical signs (mucus in the feces and partial anorexia) rarely last more than 5–6 days. Death may occur if the affected mink is immunosuppressed by the Aleutian disease virus. There are no commercially available vaccines. Treatment is symptomatic and of questionable value. It is important to differentiate this condition from mink viral enteritis.

BACTERIAL DISEASES

BOTULISM

Botulism (qv, p 370) occasionally causes heavy losses in unvaccinated mink that consume feed containing Type C toxin. Usually, many mink are found dead within 24 hours of exposure to the toxin, while others show varying degrees of paralysis and dyspnea. Postmortem findings are nonspecific and are related to death from respiratory paralysis. Diagnosis is confirmed through inoculation of serum or filtered tissue from affected mink into mice. The immunotype of botulism toxin involved is determined by mouse protection tests using specific antitoxin.

Toxic feed should be removed, and stored feed or ingredients examined for the presence of toxin. Recovered mink are not immune to further challenge. Annual vaccination of kits and breeders with botulism (Type C) toxoid is recommended to prevent outbreaks.

HEMORRHAGIC PNEUMONIA

Pseudomonas aeruginosa may result in serious losses. Mink of all ages are affected, particularly during the stressful period of fall molt. Mink are usually found dead with no prodromal signs. A bloody nasal exudate may be observed at the time of death. Gross lesions include a severe hemorrhagic pneumonia with swelling and consolidation of 1 or more lung lobes. Treatment involves immediate vaccination of the entire herd or a "buffer zone" of animals around the focus of injection with a *Pseudomonas* bacterin and immediate administration of sodium sulfathiazole (1 oz/150 lb [410 mg/kg] of wet mixed feed) and an equivalent quantity of sodium bicarbonate for 7 days as a herd treatment. Care must be taken with dosage since sulfonamides are potentially toxic for mink. The mink should have ample water. *Pseudomonas* bacterins are available for prophylactic ranch vaccination.

TUBERCULOSIS

Mink, particularly Aleutian types, are susceptible to infection with avian, bovine, and human tubercle bacilli. Infection is usually food-borne, and the disease has become endemic on some ranches. Clinical signs include weight loss and in some cases, abdominal distension. Affected mink are severely emaciated and have enlarged spleen and lymph nodes. There may be miliary involvement of the lungs, liver, and other organs. The diagnosis is confirmed by identifying acid-fast intracellular organisms in smears from affected tissues. There is no treatment, and control consists of culling visibly affected mink, and feeding meat products from inspected processing plants. Tuberculin tests are generally ineffective in detecting infected mink.

URINARY INFECTIONS AND UROLITHIASIS

Urinary tract infections cause serious losses in female mink in late spring (during pregnancy and lactation) and in male mink in late summer and autumn (during the rapid-growth and furring period). Several predisposing factors have been suggested: hypovitaminosis-A; diethylstilbestrol toxicity; contamination of food, cages, or nest boxes by pathogenic bacteria; decreased water intake; or increased mineral intake.

Mink may die without showing clinical signs or they may have difficulty in urinating, dribble urine, and occasionally have hematuria. Gross postmortem findings include acute hemorrhagic cystitis or pyelonephritis, or both, usually associated with urinary calculi (magnesium ammonium phosphate) in the bladder or kidneys. A variety of organisms, including staphylococci, coliforms, and *Proteus* sp are commonly isolated.

In severe outbreaks, bacteriological culture and antibiotic sensitivity tests should be done, and the treatment added to the feed. The following will help to prevent the condition: good sanitation to reduce environmental contamination; increasing the water supply; and pelting out of families in which the condition is seen. When a continual problem exists (with magnesium ammonium phosphate calculi), feed grade (75%) phosphoric acid may be added to the feed (0.8 lb/100 lb [8 g/kg] of wet mixed feed) from March to early June and from mid-July to October, to reduce the pH of the urine: phosphoric acid should not be used in young mink. Salt (NaCl, 0.5%) may be added to the diet to increase water consumption.

MISCELLANEOUS BACTERIAL DISEASES

Various diseases or signs of disease, including septicemia, pneumonia, pleuritis, abortions, abscesses, cellulitis, and enteritis occur sporadically on mink ranches; occasionally, they may become herd problems. Many bacteria, including *Proteus, Klebsiella,* and *Campylobacter* spp, coliforms, streptococci, staphylococci, and salmonellae, have been isolated.

Antibacterial sensitivity tests should be done to determine the treatment. Drugs may be administered parenterally or in the feed or water. Dosage can be estimated on the basis of body weight—female mink weigh about 1¾–2 lb (800–1000 g), and males from 4–4½ lb (1.8–2.1 kg). Dosages recommended for cats should be used and adjusted for weight. However, some sulfonamides, eg, sulfaquinoxaline and sulfamethazine, and streptomycin should not be used in mink.

The source of infection should be determined and eliminated: eg, enteritis often is caused by contaminated or spoiled feed, and abscesses by injury from wire or splintered wood in the pens, awns in hay or straw used for bedding, or spicules of bone in the feed. Outbreaks of tularemia, anthrax, brucellosis, and clostridial infections have been caused by feeding contaminated feed, often containing tissue of animals that have died or are carriers of these infections. Careful selection of feed ingredients, and disinfection of equipment and pens are important in the prevention and control of many infections of mink. "Dead stock" should not be used as mink feed.

NUTRITIONAL DISEASES

Steatitis (yellow fat disease) occurs in young, rapidly growing mink as a result of excessive rancid unsaturated fatty acids or a deficiency of vitamin E in the diet. Affected mink may be found dead, or they may exhibit slight locomotor disturbances followed by death. Necropsy findings include yellow, edematous internal or subcut. fat that contains an acid-fast pigment. Control consists of removal of the source of the rancid fats and proper storage of feed. Stabilized vitamin E may be administered in the feed (15 mg/mink) for 2 weeks, and affected kits should be injected parenterally with 10–20 mg vitamin E for several days. The condition can be prevented by feeding a nutritionally sound diet.

Chastek paralysis (thiamine deficiency) results from feeding certain raw fish that contain the enzyme thiaminase. These include whitefish, freshwater smelt, carp, goldfish, creek chub, fathead minnow, buckeye shiner, sucker, channel catfish, bullhead and minnow, white bass, sauger pike, burbot, and saltwater herring. Affected mink gradually lose their appetite and weight, and die after terminal convulsions and paralysis. Thiaminase-containing fish should be thoroughly cooked at 181°F (83°C) for at least 5 minutes, or fed raw as a portion of the diet only on alternate days. Affected mink may be injected subcut. with 50 mg of thiamine hydrochloride, which results in rapid recovery. Adequate thiamine (brewer's yeast) should be present in the ration.

Because of the rapid growth of mink kits, **rickets** occurs when rations are deficient in vitamin D, calcium, or phosphorus. Affected kits usually crawl unsteadily in a frog-like posture, have rubbery bones, and are smaller than normal. The diet should be supplemented as required, and severely affected kits may be treated on an individual basis.

Nursing sickness occurs in lactating mink. Affected females become thin and weak, stop eating, and wander about aimlessly, ignoring their kits and sometimes carrying food in their mouths without eating it. Death occurs within a few days of onset of clinical signs. Gross postmortem lesions are nonspecific, but include lipidosis of liver and kidney, and dehydration. Kits from affected females must be weaned or fostered as soon as possible, and affected females should be tempted to eat with liver, freshly killed sparrows, etc. Intraperitoneal or subcut. treatment with electrolyte solutions often help reverse the dehydration. To control this condition, kits should be encouraged to start eating solid food as early as possible by placing trays of soft feed in the pen. Salt may be added (NaCl, 0.5% of ration) if it is not already present in the commercial cereal, and plenty of fresh water and feed should be available to nursing females at all times.

Cotton underfur usually indicates anemia, and may be caused by certain fish (Pacific hake, coalfish, whiting) that interfere with iron retention in the

mink. This condition can be prevented by thoroughly cooking the offending fish at 181°F (83°C) for at least 5 minutes, or by feeding it on alternate days.

Gray underfur and loss of guard hair occurs when high levels of uncooked eggs are fed to young mink. Avidin, a factor present in eggs, inactivates biotin, a vitamin required for pigmentation and hair growth. Affected mink may be injected with 1 mg biotin twice weekly for 4 weeks and biotin may be added to the ration. Biotin deficiency can be prevented by cooking eggs at 196°F (91°C) for 5 minutes.

POISONING

Lead poisoning may occur in mink that have ingested lead-containing paints from wire or other equipment. Affected mink gradually lose weight and die within 1–2 months with clinical signs consistent with either gastroenteritis or CNS disturbance. Individual mink may be treated with calcium EDTA as a chelating agent. Dicalcium phosphate or calcium gluconate and vitamin D should be added to the ration on affected ranches. All sources of lead should be removed.

Insecticides other than pyrethrum, piperonyl butoxide, and rotenone may be highly toxic to mink. The above insecticides should not be used on mink under 8 weeks of age, or where these mink can contact them (eg, nest boxes). Other insecticides should be avoided whenever possible.

Wood preservatives (chlorinated phenols, cresols) cause mortality of kits in the first 3 weeks of life, and occasionally, in older mink. They should not be used where mink can chew on treated wood (pens, nest boxes or nest litter). Care should be taken that shavings used as nest box litter do not contain wood preservatives.

Diethylstilbestrol-containing products cause reproductive failure and a high incidence of urinary tract infections in mink, and care should be taken not to include them in the ration. Similarly, **thyroid** and **parathyroid** glands included in meat trimmings fed to mink may result in reproductive failure if present at high levels.

Chlorinated hydrocarbons and **polychlorinated biphenyls (PCB)** contained in the ration have caused reproductive failure in mink. Mink appear to be exquisitely sensitive to **polybrominated biphenyls (PBB)**; 1 ppm in the ration has caused decreased litter size and viability of offspring.

DMNA: The addition of sodium nitrite as a preservative to stale herring meal results in the formation of dimethylnitrosamine (DMNA), which is very hepatotoxic in mink, causing hepatic degeneration, ascites, and extensive internal hemorrhage.

Sulfaquinoxaline upsets normal blood-clotting mechanisms of mink and causes extensive internal hemorrhage, which results in serious losses. **Streptomycin** is toxic to mink and its use should be avoided.

MISCELLANEOUS DISEASES

Fur-clipping and **tail-biting** are common vices of mink, and may be related to abnormal behavior patterns of captivity. Fur-clipping decreases the value of the pelt, and tail-biting frequently results in fatal hemorrhage. There is no effective treatment: all mink demonstrating these vices should be pelted.

Urinary incontinence (wet-belly disease) is a nonfatal condition usually affecting male mink in the late summer and autumn, characterized by dribbling of urine and staining of the pelt around the urinary orifice. Since affected areas of the pelt must be discarded, the condition is of economic importance. The cause of the condition is unknown but at least 3 factors, including the genetic strain, dietary fat level, and calcium-phosphorus ratio appear to have the greatest influence on the incidence of "wet belly". Affected animals should be pelted.

Starvation and **chilling** cause death in mink fed inadequate fat or provided with too little feed during the winter and early spring. Affected mink are thin, and may run until they collapse and die, or they may be found dead in their cages. Such deaths are commonest after a sudden decrease in environmental temperature, especially in the early spring when mink are being brought into breeding condition. Necropsy reveals emaciation and an absence of body fat, in some cases accompanied by hepatic lipidosis and gastric ulceration. This management disease must be differentiated from infectious diseases.

Gray diarrhea in mink resembles chronic pancreatic necrosis in dogs, and is characterized by a ravenous appetite and the passage of large amounts of gray, fetid feces. Affected mink appear to die of starvation. No pancreatic abnormalities, viruses, bacteria, or parasites have been demonstrated to be causes. Treatment is of questionable value.

Gastric ulcers and **hepatic and renal lipidosis** are common in mink, and are usually associated with other diseases or periods of stress that result in several days of inappetence. This condition is commonest during late gestation, during the stressful period of weaning kits, and during the fall period of furring up.

Hereditary diseases such as hydrocephalus, hairlessness, "screw neck," "bobbed tails," Ehlers-Danlos syndrome, hemivertebrae, and tyrosinemia occur occasionally and must be controlled by pelting of the sire, dam, and litter mates of the affected mink.

Coccidiosis occasionally causes losses in young mink. Affected animals have diarrhea, dehydration, and weight loss. Coccidiostats may be used to control outbreaks. Coccidiosis can be prevented through good sanitation and regular manure removal.

Myiasis: Flies of the genus *Wohlfahrtia* are the most important external parasites of mink. The female fly lays maggots directly on the skin of the kits. The larvae penetrate the skin and produce inflammation and lesions that resemble abscesses. Affected kits become restless, lose condition, and may die. Carbaryl or rotenone dust placed beneath the litter in the nest boxes beginning a few days prior to the occurrence of the flies may help prevent infestation. It should not be used for kits <3 days old. Treatment may be repeated once after a 14-day interval. (*See also* CUTEREBRA INFESTATIONS [SM AN], p 758.)

FOX MANAGEMENT

The importance of cleanliness in raising foxes (*Vulpes* spp) cannot be overemphasized. Raised, woven-wire-bottom pens should be used for raising ranch foxes. These pens disrupt the life cycle of many parasites by the feces dropping through the wire. Usually, foxes are kept in individual pens with an attached kennel.

The ration for ranch-raised foxes is roughly the same as that fed mink, which consists of a commercial cooked cereal with chicken, beef byproducts, and fish (qv, p 1185). Fox pellets are available commercially and have given satisfactory performance. The vixen usually shows signs of estrus in late January and February. The period varies between silver and blue foxes. Most farmers use a polygamous mating system, taking the female to the pen of the male. Most females are in standing heat for 2–3 days and are bred 2 or 3 times during this period. Many ranchers utilize vaginal cytology or electronic "rut gauges" to determine the proper time for best mating. Considerable research is being done on artificial insemination of foxes and this technique is becoming widely used in larger ranches. The gestation period is approximately 52 days. Foxes have 1 litter per year. Blue foxes should average 6–7 pups per litter, silver foxes 3–5 pups. Foxes are usually pelted in November and December.

DISEASES OF FOXES

DISTEMPER

Foxes are susceptible to canine distemper virus. The virus is easily transmitted between dogs, mink, ferrets, raccoons, and other susceptible species.