APPENDIX I:

ANIMAL DISEASE

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[1] Merck Veterinary Manual
8th Edition
Online Version
Published by
Merck & Co., Inc.
Whitehouse Station, NJ, USA

[2] National Animal Control Association Training Guide
Revised Edition 1995 P 15-7—15-11

[3] University of California at Davis Shelter Medicine WEB Page
http://www.vetmed.ucdavis.edu/msmp/protocols/cleaning/cleaning.htm

[4] http://www.thebark.com/ezine/features_specialFeatures/specialFeatures_04.html

[5] National Animal Control Association Training Guide
Revised Edition 1995 P 1-2—1-11

Appendix I Animal Disease

1.
Rabies

Rabies is an acute viral encephalomyelitis that principally affects carnivores and insectivorous bats, although it can affect any mammal. It is almost invariably fatal once clinical signs appear. Although rabies occurs throughout the world, a few countries are free of the disease due to successful eradication programs or to their island status and enforcement of rigorous quarantine regulations.

Etiology and Epidemiology:

Rabies is a rhabdovirus that characteristically is confined to one species in a given geographic area, although extension to other species is common. Identification of different variants, or ecotypes, by laboratory procedures such as monoclonal antibody analysis and polymerase chain reaction (PCR) has greatly enhanced understanding of rabies epidemiology. Generally, each ecotype is responsible for rabies transmitted between members of the same species in a given geographic area. From an epidemiologic perspective, it is preferable to use the name of the species acting as the reservoir as an adjective: rabies maintained by dog-to-dog transmission is canine rabies, whereas rabies in a dog as a result of extension from a different reservoir, eg, skunk (or fox), should be referred to as skunk (or fox, etc) rabies in a dog.

In North America, distinct ecotypes are responsible for rabies in dogs and coyotes in Mexico and south Texas, red foxes in Canada and New England, raccoons along the eastern seaboard, and gray foxes in Texas. Two different ecotypes are responsible for rabies in striped skunks in the south central states and striped skunks in the north central states. Other less common ecotypes, such as Arizona fox and hog-nosed skunk, also occur. The epidemiology in North American bats is somewhat confusing, but in general, each ecotype found in bats can be assigned to a predominant bat species. Extension from bats to terrestrial animals occurs infrequently but seems to involve cats more frequently than other animals. Most cases of rabies in man in the USA in the past decade have been caused by bat rabies ecotypes (especially the variant associated with a relatively rare species, *Lasionycteris noctivagans* , the silver-haired bat).

Reservoirs of rabies vary throughout the world. Canine rabies predominates in Africa, Asia, Latin America, and the Middle East. In North America and Europe, where canine rabies has been practically eliminated, rabies is maintained in wildlife. For many years, skunks were the most commonly reported rabid animal in the USA, but since 1991, rabid raccoons have been the most numerous. Canine rabies has recently become established in coyotes (*Canis latrans*) in southern Texas and has the potential to spread throughout much of the USA and Canada. Skunk, raccoon, and fox rabies are each found in fairly distinct geographic regions of North America, although some overlapping occurs. Bat rabies is distributed throughout the USA. In Europe, red fox rabies predominates. In parts of northern Europe, rabies in the raccoon dog is of increasing concern, and it now appears that rabies in serotine bats may be widely distributed in Europe. The vampire bat is an important reservoir in Mexico, Central and South America, and parts of the Caribbean and is the source of outbreaks in cattle and sometimes man. Other wild species play an important role in the maintenance of rabies in certain areas, including mongooses in the Caribbean and southern Africa, jackals in certain parts of Africa, and wolves in parts of northern Europe.

No cat-to-cat transmission of rabies has been recorded, and no feline ecotype is known. However, cats are very susceptible to all ecotypes, and extension is common. Virus is present in the saliva of rabid cats, and there have been reports of people developing rabies after being bitten by a rabid cat. Reported cases in domestic cats have outnumbered those in dogs in the USA every year since 1987.

Transmission and Pathogenesis:

Transmission is almost always by introduction of virus-laden saliva into the tissues, usually by the bite of a rabid animal. However, virus from saliva or tissue fluids may be introduced into fresh wounds or through intact mucous membrane (eg, ingestion). Virus may be present in the saliva and transmitted by an infected animal several days before onset of clinical signs (usually 3-5 days in domestic dogs and cats and up to 8 days in striped skunks). Rabies virus has not been isolated from skunk musk (spray).

The incubation period is both prolonged and variable; typically, the virus remains at the inoculation site for a considerable time. The unusual length of the incubation period is why postexposure treatment, including in man the practice of locally infiltrating hyperimmune serum, is possible. Most cases in dogs occur within 21-80 days after exposure, but the incubation period may be shorter or considerably longer. One reliably recorded case of rabies in man had an incubation period of almost 7 yr.

After replication within muscle cells near the site of inoculation, the virus travels via the peripheral nerves to the spinal cord and ascends to the brain. After reaching the brain, the virus usually travels efferently via peripheral nerves to the salivary glands. Therefore, it is assumed that if an animal was capable of transmitting rabies via its saliva, virus will be detectable in the brain.

Hematogenous spread is extremely unusual. Under most circumstances, there is no danger of aerosol transmission of rabies. However, aerosol transmission has occurred under specialized conditions in which the air contains a high concentration of suspended particles or droplets carrying viral particles. Such conditions may develop in laboratory settings where leaks in high-pressure systems expel virus into the air, or in bat caves where oral and nasal secretions are aerosolized by millions of wingbeats. It is hypothesized that aerosol infection occurs via direct attachment of the virus to olfactory nerve endings and, therefore, may even evade immunity provided by preexposure prophylaxis.

Clinical Findings:

Clinical signs of rabies are rarely definitive. Rabid animals of all species exhibit typical signs of CNS disturbance, with minor variations among species. The most reliable signs, regardless of species, are behavioral changes and unexplained paralysis. Behavioral changes may include anorexia, signs of apprehension or nervousness, irritability, and hyperexcitability (including priapism). The animal may seek solitude. Ataxia, altered phonation, and changes in temperament are apparent. Uncharacteristic aggressiveness may develop—a normally docile animal may suddenly become vicious. Commonly, rabid wild animals lose their fear of man, and species that are normally nocturnal may be seen wandering about during the daytime.

The clinical course is divided into three phases—prodromal, excitative, and paralytic. However, this division is of limited practical value because of the variability of signs and the irregular lengths of the phases. During the prodromal period, which lasts 1-3 days, animals show only vague CNS signs, which intensify rapidly. The disease progresses rapidly after the onset of paralysis, and death is virtually certain within 10 days after the initial onset of signs. Some animals die rapidly without marked clinical signs.

The term “furious rabies” refers to animals in which aggression (the excitative phase) is pronounced. “Dumb or paralytic rabies” refers to animals in which the behavioral changes are minimal or absent, and the disease is manifest principally by paralysis.

Furious Form:

This is the classical “mad-dog syndrome,” although it occurs in all species. There is rarely any evidence of paralysis during this stage. The animal becomes irrational and, with the slightest provocation, may viciously and aggressively use its teeth, claws, horns, or hooves. The posture and expression is one of alertness and anxiety, with pupils dilated. Noise invites attack. Such animals lose all caution and fear of natural enemies. Carnivores with this form of rabies frequently roam extensively, attacking other animals, including people, and any moving object. They commonly swallow foreign objects, eg, feces, straw, sticks, and stones. Rabid dogs chew the wire and frame of their cages, breaking their teeth, and will follow a hand moved in front of the cage, attempting to bite. Young pups apparently seek human companionship and are overly playful, but bite even when petted, usually becoming vicious in a few hours. Rabid skunks appear to seek out and attack litters of puppies or kittens. Rabid domestic

cats and bobcats attack suddenly, biting and scratching viciously. As the disease progresses, muscular incoordination and seizures are common. Death is the result of progressive paralysis.

Paralytic Form:

This is first manifest by paralysis of the throat and masseter muscles, often with profuse salivation and inability to swallow. Dropping of the lower jaw is common in dogs. Owners frequently examine the mouth of dogs and livestock searching for a foreign body or administer medication with their bare hands, thereby exposing themselves to rabies. These animals are not vicious and rarely attempt to bite. The paralysis progresses rapidly to all parts of the body, and coma and death follow in a few hours.

Species Variations:

Cattle with furious rabies are dangerous, attacking and pursuing man and other animals. Lactation ceases abruptly in dairy cattle. Instead of the usual placid expression, there is one of alertness. The eyes and ears follow sounds and movement. A common clinical sign is a characteristic abnormal bellowing, which may continue intermittently until shortly before death.

Horses and mules frequently show evidence of distress and extreme agitation. These signs, especially when accompanied by rolling, may be interpreted as evidence of colic. As with other species, horses may bite or strike viciously and, because of size and strength, become unmanageable in a few hours. Such animals frequently suffer self-inflicted wounds.

Rabid foxes and coyotes frequently invade yards or even houses, attacking dogs and people. The irrationality of behavior that can occur is demonstrated by the fox that attacks a porcupine; finding a fox with porcupine quills can, in most cases, support a diagnosis of rabies.

Rabid raccoons and skunks typically show no fear of man and are ataxic, frequently aggressive, and active during the day, despite their nocturnal nature. In urban areas, they often attack domestic pets.

In general, rabies should be suspected in terrestrial wildlife acting abnormally. The same is true of bats that are seen flying in the daytime, resting on the ground, attacking people and animals, or fighting. Insectivorous bats, though small, can inflict a wound with their teeth and should never be caught or handled with bare hands.

Rodents and lagomorphs rarely constitute a risk for rabies exposure, but each incident must be evaluated individually. Several reports of laboratory-confirmed rabies in woodchucks have been associated with the raccoon rabies epizootic in the eastern USA.

Diagnosis:

Clinical diagnosis is difficult, especially in localities where rabies is uncommon. In the

early stages, rabies can easily be confused with other diseases or with normal aggressive tendencies. Therefore, when rabies is suspected and definitive diagnosis is required, laboratory confirmation is indicated.

Immunofluorescence microscopy on fresh brain tissue, which allows direct visual observation of a specific antigen-antibody reaction, is the test of choice. When properly used, it can establish a highly specific diagnosis within a few hours. Brain tissues examined must include hippocampus, medulla oblongata, and cerebellum (and should be preserved by refrigeration with wet ice or cold packs). The mouse inoculation test or tissue culture techniques using mouse neuroblastoma cells (or both) are commonly used as a backup, but results rarely disagree with those of the immunofluorescence test.

Control:

Comprehensive guidelines for control in dogs have been prepared by the World Health Organization and include the following: 1) notification of suspected cases, and destruction of dogs with clinical signs and dogs bitten by a suspected rabid animal; 2) reduction of contact rates between susceptible dogs by leash laws, dog movement control, and quarantine; 3) mass immunization of dogs by campaigns and by continuing vaccination of young dogs; 4) stray dog control and destruction of unvaccinated dogs with low levels of dependency on, or restriction by, man; and 5) dog registration.

The Compendium of Animal Rabies Control, compiled and updated annually by the National Association of State Public Health Veterinarians (NASPHV), summarizes the most current recommendations for the USA and lists all USDA-licensed rabies vaccines. Many effective vaccines, both modified live virus and inactivated types, are available for use in dogs throughout the world; in the USA, all currently marketed vaccines (for any species) are inactivated. Recommended vaccination frequency is 1 or 3 yr. Several vaccines are also available for use in cats, and a few for use in ferrets, horses, cattle, and sheep. Because of the increasing importance of rabies in cats, vaccination of cats is extremely important. No vaccine is approved for use in wildlife kept as pets, and protective immunity from the commercially available vaccines has not been demonstrated in these species.

Until recently, the control of rabies in wildlife populations relied on the destruction of wildlife in an attempt to reduce the contact rate between susceptible animals; however, this proved ineffective. In Europe and Canada, use of oral vaccines distributed in baits to control fox rabies is widespread and effective. The disease has now been eradicated from Switzerland and curtailed in Ontario and in several western European countries. Use of a vaccinia-rabies glycoprotein recombinant vaccine is being investigated as a potential method to control wildlife (raccoon and coyote) rabies in North America and to assist in the control of dog rabies in developing countries.

Management of Suspected Rabies Cases—Exposure of Pets:

Where terrestrial wildlife or bat rabies is known to occur, any animal bitten or scratched

by a wild, carnivorous mammal (or a bat) not available for testing should be regarded as having been exposed to rabies. The NASPHV recommends that any unvaccinated dog or cat exposed to rabies be destroyed immediately. If the owner is unwilling to do this, the animal should be placed in strict isolation for 6 mo and vaccinated against rabies 1 mo before release. Some rabies authorities recommend vaccination at the beginning of the isolation period. If an exposed animal is currently vaccinated, it should be revaccinated immediately and closely observed for 45 days.

Exposure of Man:

When a person is exposed to an animal suspected of having rabies, the risk of rabies transmission should be evaluated carefully. Risk assessment should include consideration of the species of animal involved, the prevalence of rabies in the area, whether exposure sufficient to transmit rabies occurred, and the current status of the animal and its availability for diagnostic testing. Wild carnivores and bats present a considerable risk where the disease occurs, regardless of whether abnormal behavior has been seen. Any wild carnivore or bat suspected of exposing a person to rabies should be considered rabid unless proved otherwise by laboratory testing. This also applies to "pet" wildlife. Any healthy dog or cat, whether vaccinated against rabies or not, that exposes (bites or deposits saliva in a fresh wound or on a mucous membrane) a person should be confined for 10 days; if the animal develops any signs of rabies during that period, it should be humanely destroyed and its brain promptly submitted for rabies diagnosis. If the dog or cat responsible for the exposure is stray or unwanted, it should be destroyed as soon as possible and submitted for rabies diagnosis. Since the advent of testing by immunofluorescence microscopy, there is no value in holding such animals to "let the disease progress" as an aid to diagnosis.

Human Immunization:

Preexposure immunization is strongly recommended for all people in high-risk groups, such as veterinary practitioners, animal control officers, rabies and diagnostic laboratory workers, and people travelling to countries in which canine rabies is endemic or epizootic. However, preexposure prophylaxis cannot be absolutely relied on in the event of subsequent rabies exposure and must be supplemented by a limited postexposure regimen.

2.
Canine Parvovirus

Etiology and Pathophysiology:

The origin of the canine parvovirus has not been established. The virus is very stable in the environment, able to withstand wide pH ranges and high temperatures. It is resistant to a number of common disinfectants and may survive for several months in contaminated areas. Rottweilers, American Pit Bull Terriers, Doberman Pinschers, and German Shepherd Dogs are at increased risk of disease. Toy Poodles and Cocker Spaniels appear at decreased risk for developing the enteric disease. Mortality associated with canine parvovirus infection is reported to be 16-35%.

The virus is transmitted from direct contact with infected dogs. Indirect transmission, eg, from fecal-contaminated fomites, is also an important source of infection. The virus is shed in the feces of infected dogs for up to 3 wk after infection. Recovered dogs may serve as carriers and shed the virus periodically.

After ingestion, the virus replicates in lymphoid tissue of the oropharynx; from there, it spreads to the bloodstream. It attacks rapidly dividing cells throughout the body, especially those in the bone marrow, lymphopoietic tissue, and the crypt epithelium of the jejunum and ileum. Replication in the bone marrow and lymphopoietic tissue causes neutropenia and lymphopenia, respectively. Replication of the virus in the crypt epithelium of the gut causes collapse of intestinal villi, epithelial necrosis, and hemorrhagic diarrhea. Normal enteric bacteria, eg, *Clostridium perfringens* and *Escherichia coli* enter the denuded mucosa and gain entry to the bloodstream, resulting in bacteremia.

Clinical Findings:

Infected dogs are often asymptomatic. Clinical disease may be triggered by stress (eg, boarding), and clinical signs may be exacerbated by concurrent infection with opportunistic enteric pathogens (eg, *Salmonella* , *Clostridium perfringens* , *E coli* , *Campylobacter* , coronavirus, and various parasites. The dose of virus required to cause clinical disease may also be a factor. Prolonged contact with a dog shedding high levels of virus increases the likelihood of disease. The incubation period is 3-8 days. Viral shedding may begin on day 3, before the onset of clinical signs.

Initially, two common clinical forms of the disease were recognized—myocarditis and gastroenteritis. Myocarditis was seen in young pups, especially in the early neonatal period. Infection lead to myocardial necrosis with either acute cardiopulmonary failure (causing pulmonary edema, cyanosis, and collapse) or scarring of the myocardium and progressive cardiac insufficiency. However, myocarditis is no longer seen because effective immunization of bitches protects pups during this early period of life.

Gastroenteritis is most common in pups 6-20 wk old, ie, the period when maternal antibody protection falls and vaccination has not yet adequately protected the pup against infection. Most affected dogs (~85%) are <1 yr old. In dogs >6 mo old, intact males are more likely to develop enteritis than intact females, reflecting the tendency of male dogs to roam. Dogs with the enteric form suffer from an acute onset of lethargy, anorexia, fever, vomiting, and diarrhea. The feces are loose and may contain mucus or blood. The severity of clinical signs varies. Most dogs recover within a few days with appropriate supportive care; others can die within hours of the onset of clinical signs. A common complication is pulmonary edema or alveolitis.

Other clinical problems that have been associated with canine parvovirus include birth defects and infertility. However, supportive evidence is lacking.

Diagnosis:

Diagnosis is based on an appropriate history and the clinical signs and confirmed by a positive fecal ELISA test or hemagglutination test. Leukopenia or lymphopenia is seen in most infected dogs during the course of illness. Hypoalbuminemia, hyponatremia, hypokalemia, and hypochloremia may be seen. Serum ALT levels are increased in some dogs. Diagnosis may also be confirmed by a four-fold increase in serum IgG titer over a 7- to 14-day period, detection of serum IgM antibody to parvovirus in dogs that have not been vaccinated within the last 3-4 wk, or the detection of parvovirus particles in the feces using immunoelectronmicroscopy.

Treatment:

There is no specific therapy to eliminate the virus. Most dogs recover with appropriate supportive care directed to restoration of fluid balance. Oral electrolyte solutions may be used in mildly dehydrated dogs without a history of vomiting. More severely affected dogs should receive IV fluid therapy (lactated Ringer's and 5% dextrose with additional potassium chloride [10-20 mEq/L]) to counter dehydration and maintain fluid balance. Monitoring of electrolyte changes is advisable. Most dogs that survive the first 2-3 days of disease recover.

Persistent vomiting can be controlled with metoclopramide, 0.2-0.5 mg/kg, q.i.d., PO or SC, or 1-2 mg/kg/day, slow IV).

Routine use of antibiotics is discouraged. In more severe cases (eg, those with severe blood loss, fever, or loss of intestinal integrity), intestinal integrity is compromised, and these dogs are predisposed to bacteremia and septicemia. In these cases, trimethoprim-sulfa (15 mg/kg, b.i.d., SC or PO) for 5-10 days is advisable; in more severe cases, ampicillin (20 mg/kg, t.i.d., IV) and gentamicin (2.2 mg/kg, t.i.d., SC) for a maximum of 5 days is advisable.

Food and water should be withheld until vomiting has subsided. After this, small amounts of a bland diet (eg, cottage cheese and rice or a commercially available prescription diet such as Canine i/d® [Hill's]) should be offered frequently. A small volume of warm, salted meat broth should be given concurrently. If GI signs recur after feeding, the dog should be fasted for an additional 12-24 hr before feeding again. If food can be tolerated, the bland diet is continued for 7-14 days, after which the dog's regular diet can be gradually reintroduced.

Prevention and Control:

Contaminated areas should be thoroughly cleaned. Household bleach (1:30 dilution) or commercial products labelled for use against parvovirus are potent inactivators of the virus. The same solutions may be used as foot baths to disinfect boots. Disinfection of hands, clothing, and food and water bowls is recommended. Pups should be kept isolated from adult dogs returning from shows or field trials.

Vaccination is critical in the control of the disease. Variants of the virus have appeared since the disease was first recognized, but current vaccines protect dogs against all strains of the virus. Vaccines containing live attenuated canine parvovirus generally induce more effective immunity than inactivated virus vaccines. The high-titer canine parvovirus vaccines now available effectively protect puppies against viral challenge, even during the period when maternal antibody titers remain high enough to interfere with active immunization but have declined enough to predispose pups to infection. Three doses of vaccine are recommended at 6, 9, and 12 wk of age.

3.
Canine Distemper

Canine distemper is a highly contagious, systemic, viral disease of dogs seen worldwide. It is characterized by a diphasic fever, leukopenia, GI and respiratory catarrh, and frequently pneumonic and neurologic complications. The disease occurs in Canidae (dogs, foxes, wolves), Mustelidae (eg, ferret, mink, skunk), most Procyonidae (eg, raccoon, coatimundi), and some Viveridae (binturong).

Etiology and Pathogenesis:

Canine distemper is caused by a paramyxovirus closely related to the viruses of measles and rinderpest. The enveloped virus is sensitive to lipid solvents and most disinfectants and is relatively unstable outside the host. The main route of infection is via aerosol droplet secretions from infected animals. Some infected dogs may shed virus for several months.

Virus replication initially occurs in the lymphatic tissue of the respiratory tract. A cell-associated viremia results in infection of all lymphatic tissues, which is followed by infection of respiratory, GI, and urogenital epithelium, as well as the CNS. Disease follows virus replication in these tissues. The degree of viremia and extent of spread of virus to various tissues is moderated by the level of specific humoral immunity in the host during the viremic period.

Clinical Findings:

A transient fever usually occurs 3-6 days after infection and there may be a leukopenia (especially lymphopenia) at this time, but these signs may go unnoticed. The fever subsides for several days before a second fever occurs, which lasts <1 wk. This may be accompanied by serous nasal discharge, mucopurulent ocular discharge, and anorexia. GI and respiratory signs may follow and are usually complicated by secondary bacterial infections. An acute encephalomyelitis may occur in association with or immediately after the systemic disease, or in the absence of systemic manifestations. Hyperkeratosis of the footpads (“hardpad” disease) and epithelium of the nasal plane may be seen.

Neurologic signs are frequently seen in those dogs with hyperkeratosis. CNS signs include 1) localized involuntary twitching of a muscle or group of muscles (myoclonus, chorea, flexor spasm, hyperkinesia), such as in the leg or facial muscles; 2) paresis or paralysis, often beginning in the hindlimbs evident as ataxia, followed by ascending paresis and paralysis; and 3) convulsions characterized by salivation and chewing movements of the jaw (petit mal, "chewing-gum fits"). The seizures become more frequent and severe, and the dog may then fall on its side and paddle its legs; involuntary urination and defecation (grand mal seizure, epileptiform convulsion) often occur. A dog may exhibit any or all of these neurologic signs in addition to others in the course of the disease. Infection may be mild and inapparent or lead to severe disease manifest by most of the above signs. The course of the systemic disease may be as short as 10 days, but the onset of neurologic signs may be delayed for several weeks or months.

Chronic distemper encephalitis (old dog encephalitis, [ODE]), a condition often marked by ataxia, compulsive movements such as head pressing or continual pacing, and incoordinated hypermetria, may occur in adult dogs without a history of signs related to systemic canine distemper. Convulsions and neuromuscular twitching (chorea) do not seem to occur with ODE. Although canine distemper antigen has been detected in the brain of dogs with ODE by fluorescent antibody staining, dogs with ODE are not infectious and replication-competent virus has not been isolated. The disease is caused by an inflammatory reaction associated with persistent canine distemper virus infection in the CNS.

Lesions:

Thymic atrophy is a consistent postmortem finding in young puppies. Hyperkeratosis of the nose and foot pads may be present. Depending on the degree of secondary bacterial infection, bronchopneumonia, enteritis, and skin pustules may also be present. Histologically, canine distemper virus produces necrosis of lymphatic tissues, interstitial pneumonia, and cytoplasmic and intranuclear inclusion bodies in respiratory, urinary, and GI epithelium. Lesions found in the brain of dogs with neurologic complications include neuronal degeneration, gliosis, demyelination, perivascular cuffing, nonsuppurative leptomeningitis, and intranuclear inclusion bodies predominately within glial cells.

Diagnosis:

Distemper should be considered in the diagnosis of any febrile condition in puppies with multisystemic manifestations. While the typical clinical case is not difficult to diagnose, the characteristic signs sometimes fail to appear until late in the disease. The clinical picture may be modified by concurrent toxoplasmosis, neosporosis, coccidiosis, parasitoses, and numerous viral and bacterial infections. Distemper is sometimes confused with other systemic infections such as leptospirosis, infectious canine hepatitis, or Rocky Mountain spotted fever. Intoxicants such as lead or organophosphates can cause simultaneous GI or neurologic sequelae. A febrile catarrhal illness with neurologic sequelae justifies a clinical diagnosis of distemper. At necropsy, diagnosis is usually confirmed by histologic lesions or immunofluorescent

assay for viral antigen in tissues, or both. In dogs with multisystemic signs, conjunctival, tracheal, vaginal or other epithelium, or the buffy coat of the blood can be examined by immunofluorescent assay. These samples are usually negative when the dog is showing only neurologic manifestations or when circulating antibody is present (or both). The diagnosis can then be made by serologic demonstration of virus-specific IgM or an increased ratio of CSF to serum virus-specific IgG.

Treatment:

Treatments are directed at limiting secondary bacterial invasion, supporting the fluid balance and overall well-being of the dog, and controlling nervous manifestations. Antibiotics, electrolyte solutions, protein hydrolysates, dietary supplements, antipyretics, nasal preparations, analgesics, and anticonvulsants are used. No one treatment is specific or uniformly successful. Dogs may recover completely from systemic manifestations, but good nursing care is essential. Despite intensive care, some dogs do not make a satisfactory recovery. Unfortunately, treatment for neurologic manifestations of distemper are unsuccessful. If the neurologic signs are progressive or severe, the owner should be appropriately advised.

Prevention:

Successful immunization of pups with canine distemper modified live virus (MLV) vaccines depends on the lack of interference by maternal antibody. To overcome this barrier, pups are vaccinated with MLV vaccine when 6 wk old and at 2- to 4-wk intervals until 16 wk old. Measles virus induces immunity to canine distemper virus in the presence of relatively greater levels of maternal distemper antibody. An MLV measles vaccine and a combination of MLV measles and MLV canine distemper vaccine are available. These vaccines must be administered IM. Pups 6-7 wk old should receive the measles or combination vaccine and at least two more doses of MLV distemper vaccine when 12-16 wk old. Many varieties of attenuated distemper vaccine are available and should be used according to manufacturers' directions. Annual revaccination is suggested because of the breaks in neurologic distemper that can occur in stressed, diseased, or immunosuppressed dogs

4.
Feline Panleukopenia-Feline Distemper

(Feline infectious enteritis, Feline distemper)

Feline panleukopenia is a highly contagious, often fatal, viral disease of cats; it is most severe in kittens. Nowadays, the disease is seen relatively uncommonly by veterinarians, presumably as a consequence of the widespread use of effective vaccines. However, infection rates remain high in feral, unvaccinated feline populations.

Etiology, Transmission, and Pathogenesis:

The causative parvovirus (feline panleukopenia virus [FPV]) infects and destroys actively dividing cells of all Felids and some members of related families (raccoon, mink, coatimundi, and kinkajou). Antigenically, FPV is indistinguishable from mink enteritis virus and is closely related to canine parvovirus type 2. Rapidly dividing cells in bone marrow, lymphoid tissues, intestinal epithelium, and in very young animals, cerebellum and retina are most affected. In pregnant queens, the virus may spread transplacentally to infect rapidly dividing embryonic or fetal cells, which leads to embryonic death, mummification, abortion, and stillbirth. Alternatively, infection of kittens in the perinatal period may destroy the germinal epithelium of the cerebellum, which leads to cerebellar hypoplasia, incoordination, and tremor. Feline cerebellar ataxia has become a relatively rare diagnosis, because most queens provide protective passive immunity to their kittens during the period of susceptibility.

Virus particles are abundant in all secretions and excretions during the acute phase of illness and may be shed in the feces of survivors for up to 6 wk after recovery. The virus is extremely resistant to inactivation; it may survive ≥1 yr in a suitable environment and can be transported via fomites (eg, shoes, clothing, food bowls). However, it can be destroyed by a 6% solution of bleach (aqueous sodium hypochlorite). Cats are infected via the oronasal route by exposure to infected animals or their secretions or to fomites. Most free-roaming cats are exposed to the virus during their first year of life. Those that develop subclinical infection or survive acute illness usually mount a robust, protective immune response.

Clinical Findings:

Most infections are subclinical, as evidenced by the high seroprevalence of anti-FPV antibodies among unvaccinated, healthy cats. Those cats that do manifest signs of illness are usually <1 yr old. Typically, fever (104-107°F), depression, and anorexia develop after an incubation period of 2-7 days. Vomiting may develop 1-2 days after the onset of fever; it is usually bilious and unrelated to eating. Diarrhea tends to begin a little later. Extreme dehydration develops rapidly, even in the face of continued drinking. Physical examination reveals severe depression, dehydration, and sometimes abdominal pain. Abdominal palpation may induce vomiting. Thickened, turgid intestinal loops and mesenteric lymphadenopathy may be palpable. In young animals with cerebellar involvement, ataxia and tremors with normal mentation may be seen. Retinal lesions, if present, appear as discrete gray foci. The duration of illness seldom exceeds 5-7 days. Indeed, young kittens with peracute panleukopenia often die within 24 hr of the onset of observed clinical signs. Mortality is high, especially in young kittens; losses of 25-90% are typical.

Lesions:

There may be few gross lesions in peracute cases, although typically, dehydration and emaciation are marked. The earliest changes are edema and necrosis of the thymus and mesenteric lymph nodes. The bone marrow may appear semifluid and fatty. The bowel walls are usually thickened and turgid; excessive gas may be present in some bowel loops. The serosal surfaces of severely affected bowel loops may be hyperemic, with ecchymotic or petechial hemorrhages. The liver, kidneys, and spleen may appear

slightly swollen. Histologically, the intestinal crypts are usually dilated and contain debris consisting of sloughed necrotic epithelial cells. Blunting and fusion of villi may be present. Degeneration of hepatocytes and renal tubular epithelial cells is seen. Eosinophilic intranuclear inclusion bodies may be found in tissues in which viral replication has occurred.

Diagnosis:

A presumptive diagnosis is usually based on clinical signs and the presence of panleukopenia (nadir 50-3,000 cells/μL). Neutropenia is a more consistent feature of FPV infection than is lymphopenia. Lymphopenia is most likely to be seen during the period of viremia (2-7 days after infection). Total WBC counts of ≤2000 cells/μL are associated with a poor prognosis. During recovery from infection, there is typically a rebound neutrophilia with a marked left shift. Diagnosis can be confirmed by showing the presence of FPV antigen in feces. The CITE® canine parvovirus test kit appears to detect FPV antigen during the acute phase of infection.

Differential diagnoses include other causes of profound depression, panleukopenia, and GI signs. Salmonellosis, feline leukemia virus (FeLV), and feline immunodeficiency virus infections should be considered. There is recent evidence to suggest that the panleukopenia-like syndrome previously associated with FeLV infection is in fact caused by concurrent FeLV and FPV infections.

Treatment and Prevention:

Successful treatment of acute cases requires careful monitoring, vigorous fluid therapy, and supportive care. Electrolyte disturbances, acidosis, hypoglycemia, hypoproteinemia, anemia, and systemic infections commonly develop in severely ill cats infected with FPV, appropriate treatment should be administered. In addition to vigorous crystalloid infusion, transfusion of plasma or whole blood from an immune cat will help support plasma oncotic pressure, as well as provide some passive immunity. Parenteral, broad-spectrum antibiotic therapy is appropriate; however, nephrotoxic drugs (eg, gentamicin, amikacin) should be avoided or used with great care in dehydrated cats. Antiemetic therapy (eg, metoclopramide) may provide some relief and allow earlier enteral feeding. Parenteral nutrition may be beneficial in some cases.

Excellent inactivated and modified live vaccines are available for prevention of FPV infection. Live vaccines should not be given to cats that are pregnant, immunosuppressed, or sick, or to kittens <4 wk old. Most vaccine manufacturers recommend that kittens should be vaccinated at 8-10 wk of age and again at 12-14 wk, with annual revaccination thereafter. The last vaccination of the initial series should be given after the kitten is 12 wk old because transferred maternal passive immunity may compromise efficacy if the vaccine is given earlier. Exposure to virus should be avoided until 2 wk after the initial vaccination series has been completed. Vaccination provides solid, long-lasting (perhaps lifelong) immunity.

5.
Rhinitis and Sinusitis

Inflammation of the mucous membranes of the nose and sinuses may be acute or chronic.

Etiology:

Viral infection is the most common cause of acute rhinitis or sinusitis in dogs and cats. Feline viral rhinotracheitis (FVR), feline calicivirus (FCV), canine distemper, canine adenovirus types 1 and 2, and canine parainfluenza are most frequently incriminated. Chronic states exist for FVR and FCV, with intermittent shedding associated with stress. Bacterial rhinitis or sinusitis frequently is a secondary complication. Primary bacterial rhinitis is extremely rare in dogs but may be from infection with *Bordetella bronchiseptica* or *Pasteurella multocida* . Allergic rhinitis or sinusitis is a poorly defined atopy that occurs seasonally in association with pollen production, and perennially, probably in association with house dusts and molds. Smoke aspiration, inhalation of irritant gases, or foreign bodies lodged in the nasal passages also may cause acute rhinitis.

Chronic rhinitis most commonly is due to secondary bacterial infection after inflammation or trauma, foreign bodies, neoplasia, or mycotic infection. In cats, chronic rhinosinusitis is a frequent sequela of acute viral infections of the nasal and sinus mucosa that result in hyperplastic glandular and epithelial changes. Rhinitis or sinusitis may result when an apical tooth root abscess extends into the maxillary recess. Mycotic rhinosinusitis may be caused by *Cryptococcus neoformans* , *Aspergillus* spp , and *Penicillium* spp . Cats more often are affected with *Cryptococcus* sp than dogs, whereas aspergillosis is frequent in dogs but rare in cats.

Clinical Findings and Diagnosis:

Acute rhinitis is characterized by one or more of nasal discharge, sneezing, pawing at the face, respiratory stertor, open-mouth breathing, or inspiratory dyspnea. Lacrimation and conjunctivitis often accompany inflammation of the upper respiratory passages. Affected tissues are often hyperemic and edematous. The nasal discharge is serous but becomes mucoid as a result of secondary bacterial infection. If inflammatory cells infiltrate the mucosa, the discharge may become mucopurulent. Sneezing, in an attempt to clear the upper airways of discharge or exudate, is seen most frequently in acute rhinitis and tends to be intermittent in chronic rhinitis. Aspiration reflex (“reverse sneeze”), a short paroxysmal episode of inspiratory effort in an attempt to clear the nasopharynx of obstructing material, may also be seen. Respiratory stertor, open-mouth breathing, and inspiratory dyspnea occur when the nasal passages are narrowed from inflamed mucosa, glandular elements, and secretions. An acute unilateral nasal discharge, possibly accompanied by pawing at the face, suggests a foreign body. Neoplastic or mycotic disease is suggested by a chronic nasal discharge that was initially unilateral but becomes bilateral or that changes in character from mucopurulent to serosanguineous or hemorrhagic.

6.
Feline Leukemia

Despite the widespread use of vaccines, feline leukemia virus (FeLV) remains one of the most important causes of morbidity and mortality in cats. It causes a variety of malignancies, but persistent infection can also cause severe immunosuppression and profound anemia. The virus is present worldwide. In nature, FeLV infects domestic cats and a few other Felidae. In the laboratory, cells from a much wider range of species can be infected by some strains of the virus.

Etiology and Epidemiology:

FeLV is a retrovirus in the family Oncovirinae. Other oncoviruses include feline sarcoma virus, mouse leukemia viruses, and two human T-lymphotropic viruses. Although oncogenesis is one of their more dramatic effects, oncoviruses cause many other diseases, including degenerative, proliferative, and immunological disorders.

There are three main FeLV subgroups of clinical importance. Subgroup A viruses are found in all naturally infected cats. FeLV-A, the original, archetypical form of the virus, is efficiently transmitted among cats. FeLV-A viruses tend to be less pathogenic than viruses of the other subgroups, but some strains cause severe immunosuppression. Almost all naturally infected cats are originally infected by FeLV-A. Within the infected cat, FeLV-A is sometimes altered to produce FeLV-B and FeLV-C viruses. FeLV-B is found in ~50% of naturally infected cats, along with FeLV-A. The FeLV-A and FeLV-B together are more frequently associated with neoplastic diseases than is FeLV-A alone. FeLV-C viruses are isolated from only 1% of naturally infected cats, along with FeLV-A and sometimes both FeLV-A and FeLV-B. The presence of FeLV-C in an infected cat is strongly associated with the development of erythroid hypoplasia and consequent severe anemia. Viruses of all three subgroups are detected (but cannot be distinguished) by commonly used FeLV diagnostic test kits.

The incidence of FeLV infection is directly related to the population density of cats. Infection rates are highest in catteries and multicat households, especially when cats have access to the outdoors. In the USA, 1-2% of healthy stray urban cats are persistently viremic. Not surprisingly, much higher percentages of sick, “at risk” cats are found to be infected.

Persistently infected, healthy cats are the major reservoir of FeLV. Carriers excrete large quantities of virus in saliva. Lesser amounts of virus are excreted in tears, urine, and feces. Oronasal contact with infectious saliva or urine is the most likely mode of transmission. Nose-to-nose contact, mutual grooming, and shared litter trays and food dishes facilitate transmission. Bite wounds from infected cats are an efficient mode of

transmission but occur relatively infrequently in cats kept indoors 100% of the time. Bites may be a more important mode of transmission in indoor-outdoor cats.

Age resistance is significant. Young kittens are much more susceptible than adults. The virus may be transmitted vertically (in utero or by milk) or horizontally (by secretions and excretions). Because FeLV is a fragile, enveloped virus and because of age resistance, horizontal transmission between adults usually requires prolonged, intimate contact. In addition, the dose required for oronasal transmission of the virus is relatively high.

Although FeLV infects felids other than domestic cats, there is no important reservoir of infection in wild felids.

Pathogenesis:

After oronasal inoculation, the virus first replicates in oropharyngeal lymphoid tissue. From there, virus is carried in blood mononuclear cells to spleen, lymph nodes, epithelial cells of the intestine and bladder, salivary glands, and bone marrow. Virus later appears in secretions and excretions of these tissues and in peripheral blood leukocytes and platelets. Viremia is usually evident 2-4 wk after infection. The acute stage of FeLV infection (2-6 wk after infection) is rarely detected. It is typically characterized by mild fever, malaise, lymphadenopathy, and blood cytopenias.

In ≥70% of adult cats, viremia and virus shedding are transient, lasting only 1-16 wk. A few cats continue to shed virus in secretions for several weeks to months after they cease to be viremic. Virus may persist in bone marrow for a longer period, but even this latent, or sequestered, infection usually disappears within 6 mo. Some FeLV-exposed cats (≤30%) fail to mount an adequate immune response and go on to become persistently (ie, permanently) viremic. Persistently viremic cats develop fatal diseases after a variable time period. In a group of persistently FeLV-positive cats, 50% die within ~1 yr; 83% of infected healthy cats die within 3½ yr of detection of their infection.

Disorders Caused by FeLV:

FeLV-related disorders are numerous and include immunosuppression, neoplasia, anemia, immune-mediated diseases, reproductive problems, and enteritis.

The **immunosuppression** caused by FeLV is similar to that caused by feline immunodeficiency virus. There is an increased susceptibility to bacterial, fungal, protozoal, and other viral infections. The incidence of feline infectious peritonitis is increased in FeLV-infected cat colonies. Numbers of neutrophils and lymphocytes in the peripheral blood of affected cats may be reduced, and those cells that are present may be dysfunctional. Many FeLV-positive cats have low blood concentrations of complement; this contributes to FeLV-associated immunodeficiency and oncogenicity because complement is vital for some forms of antibody-mediated tumor cell lysis.

Infusion of complement-rich serum can cause lymphoma regression in some cats. Much of the immunodeficiency caused by FeLV is thought to be due to the high degree of viral antigenemia.

Lymphoid or myeloid tumors (eg, lymphoma, lymphoid leukemia, erythremic myelosis) develop in up to 30% of cats persistently infected with FeLV. FeLV-negative (ie, nonviremic) cats can also develop these tumors. Lymphoma is the most frequently diagnosed malignancy of cats. Most American cats with mediastinal, multicentric, or spinal forms of lymphoma are FeLV-positive. However, in some parts of the world, these forms of lymphoma are becoming much less common, and the proportion caused by FeLV is decreasing. This may be related to effective control of FeLV. Renal and GI forms of lymphoma are more likely to be found in FeLV-negative cats.

The **anemia** caused by FeLV is usually nonregenerative and normochromic. There is frequently an idiosyncratic macrocytosis. About 10% of FeLV-related anemias are hemolytic and regenerative. This form of anemia may be associated with hemobartonellosis or immune-mediated hemolysis, or both.

Immune complexes formed in the presence of moderate antigen excess can cause systemic vasculitis, glomerulonephritis, polyarthritis, and a variety of other immune disorders. In FeLV-infected cats, immune complexes form under conditions of antigen excess, because FeLV antigens are abundant and anti-FeLV IgG antibodies are sparse. These conditions are ideal for the development of immune-mediated disease.

Reproductive problems are common; 68-73% of infertile queens have been reported to be FeLV-positive, and 60% of queens that abort are FeLV-positive (although abortion is a relatively uncommon cause of feline infertility). Fetal death, resorption, and placental involution may occur in the middle trimester of pregnancy, presumably as a result of in utero infection of fetuses by virus transported across the placenta in maternal leukocytes. Occasionally, infected queens give birth to live, viremic kittens. Some of these may carry their infection into later life. Latently infected (ie, nonviremic) queens may pass virus on to their kittens in milk.

Enteritis may develop, and it resembles feline panleukopenia both clinically and histopathologically. Clinical signs include anorexia, depression, vomiting, and diarrhea (which may be bloody). Because of the concurrent immunosuppression associated with FeLV infection, septicemia may develop. Recent evidence suggests that FeLV and feline panleukopenia virus may act synergistically to produce this syndrome.

Other disorders may also develop. FeLV occasionally causes a neuropathy leading to anisocoria and hindlimb paralysis. Certain FeLV-induced lymphomas can produce identical clinical signs. If antineoplastic therapy is planned, it is important to distinguish neoplasia from neuropathy. FeLV can also cause quasineoplastic disorders such as multiple cartilaginous exostoses (osteochondromatosis).

Diagnosis:

The ELISA for FeLV tests for the presence of soluble FeLV p27 (CA) antigen in serum, plasma, or other body fluids. FeLV antigen may be present in the absence of intact, infectious viral particles. This is because excess FeLV antigens are released from infected cells free of viral particles. The ELISA test detects antigenemia rather than viremia. All ELISA-positive cats should have an indirect immunofluorescence assay (IFA) test done to confirm their status. The IFA tests for the presence of FeLV structural antigens (eg, p27 or other core antigens) in the cytoplasm of cells suspected to be FeLV-infected. In clinical practice, peripheral blood smears are usually used for the IFA test, but cytologic preparations of bone marrow or other tissues can also be used. The vast majority of IFA-positive cats are persistently viremic and have a poor long-term prognosis.

Diagnosis of FeLV-induced neoplasia is similar to that of other tumors. Cytological examination of fine-needle aspirates of masses, lymph nodes, body cavity fluids (eg, pleural effusion), and affected organs may reveal malignant lymphocytes. Bone marrow examination may reveal leukemic involvement, even when the peripheral blood appears normal. Biopsy and histopathologic examination of abnormal tissues is often necessary for diagnostic confirmation.

Treatment:

Ideally, an FeLV-infected cat would be identified early and treated to eradicate its retroviral infection before FeLV-related diseases had time to develop. Unfortunately, eradication of retroviral infections at any stage of disease is extremely difficult. Most infected cats are persistently viremic by the time their infection is diagnosed. Persistent viremia is notoriously difficult to reverse. In addition, FeLV-related disease(s) may already be present at the time of diagnosis, further complicating therapeutic efforts.

Antiviral drugs based on nucleoside analogs have, in general, been of little benefit in the management of FeLV-infected cats. Although AZT (3'-azido-3'-deoxythymidine), at 20 mg/kg, PO, t.i.d., can prevent the development of persistent viremia if given shortly after viral inoculation, it is of little benefit when given to persistently viremic cats. However, in recent studies, the degree of antigenemia was reduced in naturally infected cats treated with AZT at 5 mg/kg, SC, b.i.d., or with PMEA (9-[2-phosphonylmethoxyethyl]adenine) at 2.5 mg/kg, SC, b.i.d. Unfortunately, both AZT and PMEA cause adverse hematologic effects. In another study, PMEA (6.25 or more mg/kg/day, SC) enabled cats to resist infection with FeLV.

Interferon, up to 1.6×10^6 u/kg, SC, s.i.d., has been given alone and in combination with AZT (20 mg/kg, PO, t.i.d.) to FeLV-infected cats. In this study, the degree of antigenemia was significantly decreased, beginning 2 wk after treatment with human recombinant interferon alpha-2b, either alone or in combination with AZT administered PO. AZT alone had no effect on the degree of antigenemia. Unfortunately, rapid development of antibodies directed against the interferon molecule limited the efficacy

of this treatment to a period of 7 wk.

In another study, ultralow doses of leukocyte-derived human interferon-α were given to FeLV-infected cats (0.5 or 5.0 u, PO, s.i.d. for 7 days, every other week for 4 wk). Although the treatment was not effective in preventing persistent viremia, mean survival time was significantly increased in treated cats compared with placebo-treated controls. The lower dose was more efficacious than the higher dose.

Staphylococcal protein A is a bacterial cell wall component that binds the F_c portion of IgG. In one study, extracorporeal immunoadsorption of plasma from FeLV-infected cats was reported. In a few infected cats, viremia was reversed and lymphoma regression was induced. Staphylococcal protein A has also been given (6.6 mg/kg, IV, twice weekly with prednisone) for the treatment of hemolytic anemia associated with FeLV infection.

Other therapeutic options, including acemannan, BCG, diethylcarbamazine, levamisole, mixed bacterial toxins, muramyl peptides, killed parapoxvirus preparations, *Propionibacterium acnes* , and suramin, have been recommended for consideration in the treatment of FeLV-infected cats. However, relatively little work has been done to establish their efficacy. FeLV-induced lymphoid malignancies can be treated using combinations of antineoplastic drugs; most protocols include prednisone, vincristine, and cyclophosphamide.

Many FeLV-positive cats remain completely healthy for years. There is no clear evidence that healthy FeLV-positive cats benefit from any form of therapy during this period. Stress and sources of secondary infection should be avoided. The cat should remain indoors 100% of the time to reduce the risk of exposure to infectious agents and to prevent the infected cat from acting as a source of infection to other cats. Owners should be advised to watch for signs of FeLV-related disease, particularly secondary infections. If an FeLV-related disease develops, it should be diagnosed and aggressively treated.

Prevention and Control:

A test and removal program to rid catteries and multicat households of FeLV can be extremely effective if these guidelines are carefully followed: 1) All cats should be tested for FeLV viremia (IFA is best). 2) All viremic cats should be removed. 3) All dishes, litter pans, and bedding should be disinfected. 4) All movement of cats in and out of the cattery should be prevented. 5) All cats should be retested after 12 wk to detect cats that may have been incubating the virus at the time of the first test. 6) The quarantine can be lifted when all cats have tested negative on two consecutive occasions, 12 wk apart. 7) All cats should be tested and quarantined before introduction to the cattery. Ideally, two tests 12 wk apart should be done. 8) Breeding should be only to cats known to be FeLV-negative, and cats should be introduced only from FeLV-negative colonies.

FeLV vaccines are intended to protect cats against FeLV infection or, at least, to prevent persistent viremia. Types of vaccines include killed whole virus, subunit, and genetically engineered. Vaccines may vary in protective effect, and manufacturers' claims and independent comparative studies should be carefully noted. The following guidelines for vaccine use have been recommended: 1) Only healthy, febrile cats should be vaccinated. 2) Cats from a high-risk or unknown background should be tested for FeLV before vaccination. 3) All cats at risk of exposure to FeLV should be vaccinated. 4) FeLV-positive and FeLV-negative cats should be kept separated, even if the FeLV-negative cats have been vaccinated. 5) Although it may be preferable to use the same brand of vaccine for primary inoculations and boosters, use of a different brand for booster vaccinations should still produce an anamnestic response. This is because gp70 (SU) is the immunogen in all currently available vaccines. 6) Vaccines that prevent infection entirely are preferable to those that just prevent persistent viremia. 7) The brand of vaccine should be selected on the basis of clear research data. The preventable fraction should be mentioned in the vaccine manufacturers' product information.

7.
Respiratory Diseases Of Small Animals

Respiratory diseases are common in dogs and cats. Although clinical signs such as coughing and dyspnea are commonly referable to primary problems of the respiratory tract, they may also occur secondary to disorders of other organ systems (eg, congestive heart failure).

Both young and aged animals are at increased risk of developing respiratory disease. At birth, the respiratory and immune systems are incompletely developed; this facilitates the introduction and spread of pathogens within the lungs, and alveolar flooding may occur. In aged animals, chronic degenerative changes that disrupt normal mucociliary clearance and immunologic anergy may render the lungs more vulnerable to airborne pathogens and toxic particulates.

A varying flora of indigenous commensal organisms (including *Pasteurella multocida* , *Bordetella bronchiseptica* , streptococci, staphylococci, pseudomonads, and coliform bacteria) normally reside in the canine and feline nasal passages, nasopharynx, and upper trachea, and at least intermittently in the lungs without causing clinical signs. Opportunistic infections by these bacteria may occur when respiratory defense mechanisms are compromised by infection with a primary pathogen (eg, distemper, parainfluenza virus, or canine type 2 adenovirus in dogs, and rhinotracheitis virus or calicivirus in cats), other insults (eg, inhalation of smoke or noxious gases), or diseases such as congestive heart failure and pulmonary neoplasia. Secondary bacterial infections complicate the management of viral respiratory infections of both dogs and cats. Pathogens may continue to reside in the respiratory tract of convalescent animals. When stressed, these animals may relapse; they can also act as a source of infection for others. Poor management practices (eg, overcrowding) are often associated with poor hygienic

and environmental conditions, and the resultant stress increases both the incidence and severity of infections. Conditions that favor the spread of infections often occur in catteries, kennels, pet shops, boarding facilities, and humane shelters.

Congenital abnormalities, such as stenotic nares, elongation of the soft palate, and tracheal stenosis, can cause respiratory dysfunction. Neoplastic masses, degenerative changes of the airways, and tracheal collapse can result in dyspnea and other clinical manifestations of respiratory disease.

Tracheal collapse is most common in toy and miniature breed dogs and rare in cats. The etiology is unknown. Affected animals have a nonproductive honking chronic cough, and inspiratory or expiratory dyspnea. Frequently, they are obese and may have concurrent cardiovascular or other pulmonary disease (especially chronic bronchitis). Weight loss (if obese) is a critically important part of management. Other measures include exercise restriction, reduction of excitement and stress, and medical therapy, eg, antitussives, antibiotics, bronchodilators, and corticosteroids

8.
Toxoplasmosis

Toxoplasma gondii is a protozoan parasite that infects most species of warm-blooded animals, including birds and man, throughout the world.

Etiology and Pathogenesis:

Members of the cat family are the only known definitive hosts for *T gondii* and, therefore, serve as the main reservoir. Three infective stages of *T gondii* have been identified: tachyzoites (the rapidly multiplying form), bradyzoites (tissue cyst form), and sporozoites (within oocysts). Infection may be acquired by carnivorism, ingestion of feces containing oocysts, or congenitally.

In unexposed cats after ingestion of uncooked meat containing tissue cysts, *T gondii* initiates enteroepithelial replication. Bradyzoites are released from tissue cysts by digestion in the stomach and small intestine, invade intestinal epithelium, and undergo sexual replication, culminating in the release of oocysts (10 μm diameter) in the feces. Oocysts are first seen in the feces at 3 days after infection and may be released for up to 20 days after infection. After exposure to air for 24 hr, oocysts sporulate, become infective, and may persist in the environment for up to 1 yr. Cats generally develop immunity to *T gondii* after the initial infection and, therefore, only shed oocysts once in their lifetime.

In all warm-blooded animals, after ingestion of uncooked meat containing tissue cysts (carnivores) or feed contaminated with cat feces containing oocysts (herbivores), *T*

gondii initiates extraintestinal replication. Bradyzoites and sporozoites, respectively, are released and infect intestinal epithelium. After several rounds of epithelial replication, tachyzoites emerge and disseminate via the bloodstream and lymph. Tachyzoites infect tissues throughout the body and replicate intracellularly until the cells burst, causing tissue necrosis. Tachyzoites measure 4-8 × 2-4 μm in diameter and stain with Giemsa. Young and immunocompromised animals may succumb to generalized toxoplasmosis at this stage. Older animals mount a powerful cell-mediated immune response to the tachyzoites (mediated by cytokines) and control the infection, driving the tachyzoites into the tissue cyst or bradyzoite stage. Tissue cysts are usually seen in neurons and in cardiac and skeletal muscle. Individual cysts are 50-150 μm in diameter and consist of an argyrophilic wall enclosing hundreds of sporozoites that stain well with periodic acid-Schiff stain. Tissue cysts remain viable in the host for many years.

Clinical Findings:

The tachyzoite is the stage responsible for tissue damage; therefore, clinical signs depend on the number of tachyzoites released, the ability of the host immune system to limit tachyzoite spread, and the organs damaged by the tachyzoites. Because adult immunocompetent animals control tachyzoite spread efficiently, toxoplasmosis is usually a subclinical illness. However, in young animals, particularly puppies, kittens, and piglets, tachyzoites spread systemically and cause interstitial pneumonia, myocarditis, hepatic necrosis, meningoencephalomyelitis, chorioretinitis, lymphadenopathy, and myositis. The corresponding clinical signs include fever, diarrhea, cough, dyspnea, icterus, seizures, and death. *Toxoplasma gondii* is also an important cause of abortion and stillbirth in sheep and sometimes in pigs and goats. After infection of a pregnant ewe, tachyzoites spread via the bloodstream to placental cotyledons, causing necrosis. Tachyzoites may also spread to the fetus, causing necrosis in multiple organs. Finally, immunocompromised adult animals (eg, cats infected with feline immunodeficiency virus) are extremely susceptible to developing acute generalized toxoplasmosis.

Diagnosis:

Antemortem, serologic testing may be useful. Available tests include the Sabin-Feldman dye test, complement fixation, direct and indirect hemagglutination, latex agglutination, modified agglutination, ELISA, and indirect fluorescent antibody testing. During *T gondii* infection, IgM antibodies appear early but generally do not persist past 3 mo after infection. Therefore, increased IgM titers (>1:256) are consistent with recent infection. In contrast, IgG antibodies appear by the fourth week after infection and may remain increased for years during subclinical infection. Thus, to be useful, IgG titers must be measured in paired sera from the acute and convalescent stages (3-4 wk apart) and must show a 4-fold increase in titer. Additionally, CSF and aqueous humor may be analyzed for the presence of tachyzoites or anti- *T gondii* antibodies. Postmortem, tachyzoites may be seen in tissue impression smears. Additionally, microscopical examination of tissue sections may reveal the presence of tachyzoites, bradyzoites, or both. Finally, homogenates of tissue samples obtained at necropsy may be injected

intraperitoneally into mice. Some strains of *T gondii* are lethal to mice in as few as 5 days. Examination of Giemsa-stained peritoneal fluid or squash preparations of mouse brain tissue will reveal tachyzoites and bradyzoites. *Toxoplasma gondii* is morphologically similar to other protozoan parasites and must be differentiated from *Sarcocystis* sp (in cattle), *Sarcocystis neurona* (in horses), and *Neospora caninum* (in dogs).

Treatment:

For animals other than man, treatment is seldom warranted. Sulfadiazine (73 mg/kg) and pyrimethamine (0.44 mg/kg) act synergistically and are used widely in the treatment of man. Other sulfa drugs, including sulfamethazine and sulfamerazine, are also effective. However, these drugs are not effective against the bradyzoite stage and, therefore, *T gondii* infection is not completely eradicated. Clindamycin is the treatment of choice for dogs and cats, at 10-40 mg/kg and 25-50 mg/kg respectively, for 14-21 days.

Prevention and Zoonotic Risk:

Toxoplasma gondii is an important zoonotic agent. In some areas of the world, up to 60% of the human population has serum IgG titers to *T gondii* and, therefore, are likely to be persistently infected. Toxoplasmosis is a major concern for people with immune system dysfunction (eg, people infected with human immunodeficiency virus). In these individuals, toxoplasmosis usually presents as meningoencephalitis and results from the emergence of *T gondii* from tissue cysts located in the brain as immunity wanes rather than from primary *T gondii* infection. Toxoplasmosis is also a major concern for pregnant women because tachyzoites can migrate transplacentally and cause birth defects in human fetuses. Infection of women with *T gondii* may occur after ingestion of undercooked meat (particularly pork) or accidental ingestion of oocysts from cat feces.

All infective forms of *T gondii* are heat labile and are destroyed by dry heat at 150°F (65°C), boiling water, iodine, and ammonia. Therefore, toxoplasmosis can easily be prevented by several simple measures: All meat should be cooked thoroughly. Uncooked meat should not be fed to pet cats. Because it takes up to 24 hr for oocysts to become infective after exposure to air, litter boxes should be cleaned daily. Individuals should wash thoroughly after handling cat feces. Pregnant women should avoid handling cats and cleaning litter boxes.

Vaccines are not currently available for use in man. However, experimental vaccines have been used to decrease the incidence of abortion in sheep and to stimulate *T gondii* immunity in laboratory rodents.

9.
Mange in Dogs and Cats

Sarcoptic Mange (Canine Scabies):

Sarcoptes scabiei canis infestation is a highly contagious disease of dogs found worldwide. The mites are fairly host-specific, but animals (including man) that come in contact with infested dogs can also be affected. The adult mite is roughly circular in shape, without a distinctive head, and has four pairs of short legs. Females are almost twice as large as males. The entire life cycle (17-21 days) is spent on the dog, where the female burrows tunnels in the stratum corneum and lays her eggs. Sarcoptic mange is readily transmitted between dogs by direct contact. The incubation period is variable (10 days to 8 wk) and depends on level of exposure, body site, and number of mites transmitted. Asymptomatic carriers may exist. Intense pruritus is characteristic and is probably due to hypersensitivity to mite products. Primary lesions consist of a papular eruption that, due to self-trauma, develops thick crusts with secondary bacterial infection. Typically, lesions start on the ventral abdomen, chest, ears, elbows, and legs and, if untreated, become generalized. Dogs with chronic, generalized disease develop severe thickening of the skin with fold

with chronic, generalized disease develop severe thickening of the skin with fold formation and crust buildup, peripheral lymphadenopathy, and emaciation; dogs so affected may even die. "Scabies incognito" has been described in well-groomed dogs; these dogs, infested with sarcoptic mites, are pruritic, but demonstrating the mites on skin scrapings is difficult because the crust and scale have been removed by regular bathing.

Diagnosis of sarcoptic mange is based on the history of severe pruritus of sudden onset, possible exposure, and involvement of other animals, including man. Sometimes, making a definitive diagnosis is difficult because of negative skin scrapings. Concentration and flotation of several scrapings may increase chances of finding the mites. Several extensive superficial scrapings should be done of the ears, elbows, and hocks; nonexcoriated areas should be chosen. Fecal flotation may reveal mites or eggs. Even if mites are not found but the history and clinical presentation are highly suggestive of sarcoptic mange, trial therapy is warranted. The hair should be clipped, the crusts and dirt removed by soaking with a good antiseborrheic shampoo, and an acaricidal dip applied. Lime-sulfur is highly effective and safe for use in young animals; several dips 5 days apart are recommended. Phosmet has been successfully used according to label instructions. Amitraz is an effective scabicide, although it is not approved for this use, and there have been some reports of lack of efficacy. Ivermectin is not approved for this use, but 200 µg/kg, PO or SC, two treatments 2 wk apart, is very effective and usually curative. Ivermectin at this dosage is contraindicated in Collies and Collie crosses, and the heartworm status of the dog should be evaluated before treatment.

Notoedric Mange (Feline Scabies):

This rare, highly contagious disease of cats and kittens is caused by *Notoedres cati* , which can opportunistically infest other animals, including man. The mite and its life cycle are similar to the sarcoptic mite. Pruritus is severe. Crusts and alopecia are seen, particularly on the ears, head, and neck, and can become generalized. Mites can be found in skin scrapings. Treatment consists of lime-sulfur dips at 10-day intervals. Nonapproved treatments include amitraz at half the concentration used in dogs and ivermectin at 200 µg/kg, SC. Sudden death in association with the use of ivermectin in kittens has been reported.

Cheyletiellosis (Walking Dandruff):

Cheyletiella blakei infests cats, *C yasguri* infests dogs, and *C parasitovorax* infests rabbits, although cross-infestations are common, including human infestation. This disease is very contagious. Mite infestations are rare in flea endemic areas, probably due to the regular use of insecticides. These mites have four pairs of legs and prominent hook-like mouthparts. They live on the surface of the epidermis, and their entire life cycle (3 wk) is spent on the host. Clinical disease is characterized by scaling, a dorsal distribution, and pruritus, which varies from none to severe. Cats may develop dorsal crusting or generalized miliary dermatitis. Asymptomatic carriers

may develop dorsal crusting or generalized miliary dermatitis. Asymptomatic carriers may exist. The mites may not be easy to find, especially in animals that are bathed often; acetate tape preparations, superficial skin scrapings, and flea combing can be used to make the diagnosis. Weekly dippings with pyrethrins or lime-sulfur for 6-8 wk are necessary to eradicate the mites. Ivermectin is also an effective, but nonapproved, treatment. The environment should also be treated with a good insecticide.

Canine Demodicosis:

This common skin disease of dogs occurs when large numbers of *Demodex canis* mites inhabit hair follicles, sebaceous glands, or apocrine sweat glands. In small numbers, these mites are part of the normal flora of the skin of dogs and cause no clinical disease. The mites are transmitted from dam to puppies during nursing within the first 72 hr after birth. The mites spend their entire life cycle on the host, and the disease is not considered to be contagious. The pathogenesis of demodicosis is complex and not completely understood; evidence of hereditary predisposition for generalized disease is strong. Immunosuppression, natural or iatrogenic, can precipitate the disease in some cases. Other factors known to predispose to generalized demodicosis include systemic disease, estrus, and heartworm infection.

Two clinical forms of the disease exist. Localized demodicosis occurs in dogs <1 yr old,and 90% of these cases are thought to resolve spontaneously. Lesions consist of areas of focal alopecia and erythema. A percentage of these cases will progress to the generalized form. Generalized demodicosis is a severe disease with generalized alopecia, papules, pustules, and crusting. Lesions are usually aggravated by secondary bacterial infections, and pododermatitis is common. Dogs can have systemic illness with generalized lymphadenopathy, lethargy, and fever when deep pyoderma, furunculosis, and draining tracts are seen. Deep skin scrapings reveal mites, eggs, and larval forms in high numbers. Whenever generalized demodicosis is diagnosed in an adult dog, medical evaluation to identify an underlying systemic disease should be pursued.

Localized demodicosis can be treated by topical application of rotenone ointment or amitraz. The prognosis for this form is usually good. The only approved treatment for generalized demodicosis is whole body amitraz dips (0.025%) applied every 2 wk; the entire hair coat should be clipped, and a benzoyl peroxide shampoo should be used for its follicular flushing activity before the dip is applied. The secondary bacterial infection must be treated with the appropriate antibiotic. Therapy must be monitored by skin scrapings every three or four dips, and treatment should not be stopped until at least two consecutive negative scrapings are obtained. Other reportedly successful experimental treatments include high doses of milbemycin oxime (0.5-1 mg/day) or ivermectin (600 µg/kg, PO, daily). Corticosteroids are contraindicated in any animal diagnosed with demodicosis.

Feline Demodicosis:

Two species of mites cause disease in cats. *Demodex cati* is thought to be a normal inhabitant of feline skin. It is a follicular mite, similar to but narrower than the canine mite. The other species of *Demodex* remains unnamed; it is shorter, with a broad abdomen, and is found only in the stratum corneum. Feline demodicosis is uncommon. In localized demodicosis, there are one or several areas of focal alopecia on the head and neck. In generalized disease, alopecia, crusting, and secondary pyoderma of the whole body are seen. The generalized form has also been associated with other systemic disease, especially diabetes mellitus. In some cases, ceruminous otitis externa has been the only clinical sign. Pruritus is variable; both forms of the mite can cause similar disease, but cats infested with the unnamed mite are frequently pruritic. Diagnosis is made by skin scrapings, although mite numbers are often small. Medical evaluation is indicated in cats with generalized disease. Dermatophyte cultures are essential, because dermatophytosis and demodicosis can be concurrent conditions. Prognosis of generalized demodicosis can be unpredictable because of its potential relationship with systemic disease. Some cases will spontaneously resolve. Weekly lime-sulfur dips (2%) are safe and usually effective.