REVIEWED ARTICLE

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Biology and medicine of the domestic ferret: an overview

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ABSTRACT: In recent years, the domestic or European ferret (Mustela putorius furo) has become increasingly popular as a companion animal. This paper reviews our knowledge of the biology and physiology, nutrition, diagnostic procedures, restraint, and anesthetic procedures, and diseases of the ferret, and briefly outlines recommended preventive medicine procedures.

INTRODUCTION

The domestic or European ferret *(Mustela putorius furo)* is a member of the family Mustelidae, order Carnivora. Other mustelids include the mink *(Mustela vison)*, skunk *(Mephitis* sp., *Spilogale* sp.), weasel *(Mustela* spp.), badger *(Taxidea taxus)*, wolverine *(Gulo luscus)*, otter *(Lutra sp., Enhydra* sp.), marten *(Martes americana)*, and fisher *(Martes pennanti)*. Two species of ferrets are found in the United States, the domestic ferret and the black-footed ferret *(Mustela nigripes)*. The black-footed ferret is the only ferret indigenous to the US and it is highly endangered. No natural population of black-footed ferrets are known to occur in the wild, although a captive propagation program is currently underway to reintroduce captive-bred stock into its former range.

The domestic ferret was domesticated from the wild European polecat and was probably brought to North America by English settlers 300 years ago.[1] Two variations are recognized based on coloration: the wild type (or fitch) ferret is a pale yellow buff with a black mask, legs, and tail; and the albino type which is white with pink eyes. It is estimated that there are now over one million pet ferrets in the U. S., and the number is rapidly increasing. The legal status of ferrets as pets in the U. S. varies from state to state. For example, in California, ferrets were originally banned as pets, however, because of recent legislation it is now legal, under some circumstances, to own a neutered male ferret.[1] Because the laws in many states are changing, it is prudent to be aware of a state's law and requirements before a client purchases a ferret.

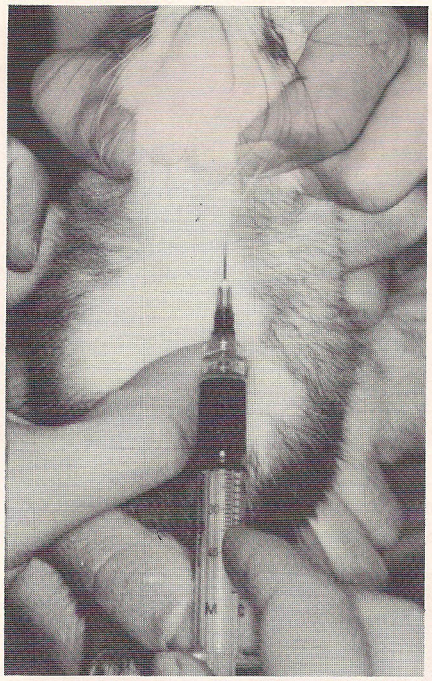

Fig.1. Jugular venipuncture in a ferret.

In summary, the ferret has become increasingly popular as a companion animal because of its small size, ease of care and maintenance, and inquisitive personality. Knowledge of ferret biology and its medical and management needs,

Fig.2. The "scruff" method of ferret restraint.

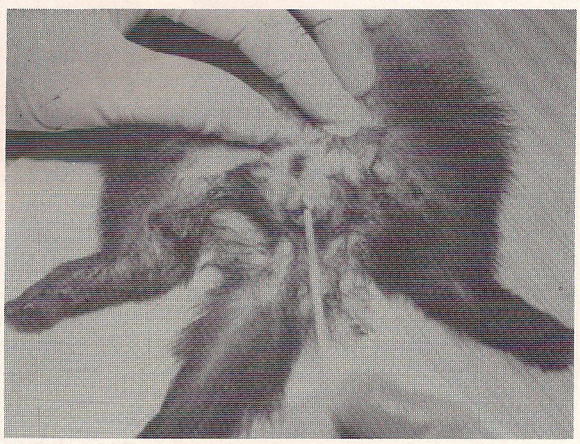

Fig.3. Partial rectal prolapse in a ferret secondary to diarrhea.

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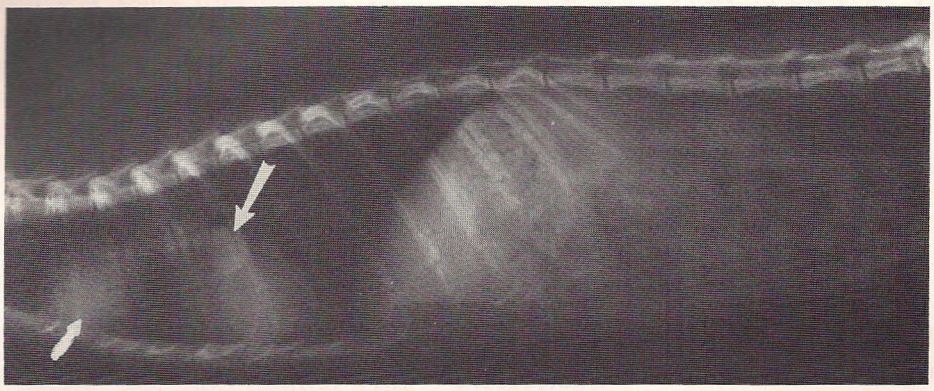
Fig. 4a.



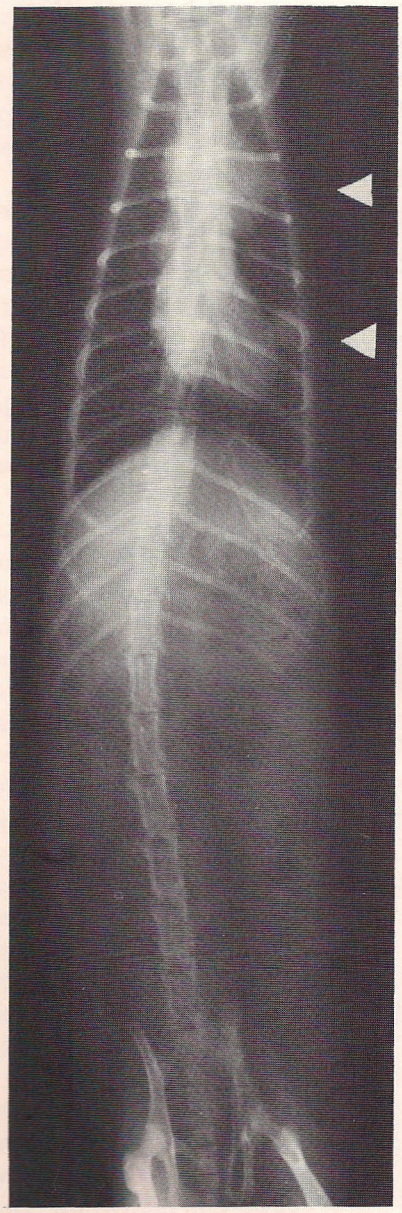


Fig.4a (above) and 4b.(below) Thoracic radiographs of a ferret with coccidiomycosis. Arrows point to two radiopaque areas in the cranial mediastinum and caudal right lung lobe, which correspond to intrathoracic fungal granulomas. (Courtesy of the J Zoo Wildl Med)

Fig.5. Insulinoma. Primary mass was excised from pancreas, and metastatic mass is present in spleen.

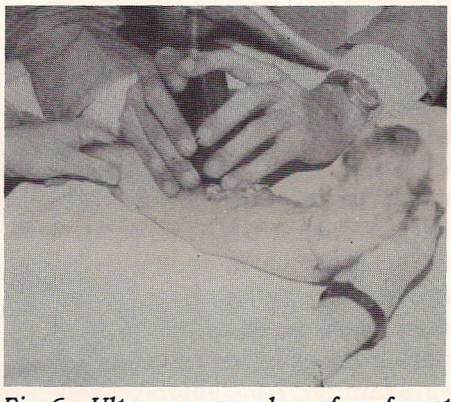


Fig.6. Ultrasonography of a ferret with hyperadrenocorticism.

including an active preventive medicine program, is essential to ensure good health and a high quality of life for this species.

BIOLOGY AND PHYSIOLOGY

The ferret, being a carnivore, is in many ways like the cat; however, it does have some unique anatomical differences. One is the presence of highly developed anal sacs that secrete a fluid called musk. The well-developed sebaceous glands also contribute to the musky smell. Another difference is that the ferret lacks a cecum and, therefore, has little capacity to digest fiber. The male ferret, unlike the cat, does have an os penis. The average life span is typically 7-8 years, although a ferret may live 9 or 10 years.

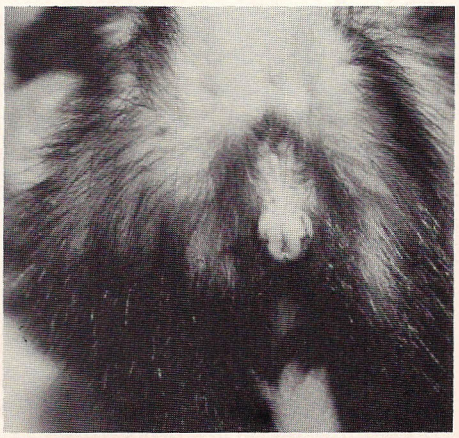


Fig.7. Vulvar swelling in an ovariectomized female ferret, associated with remnant ovarian tissue activity.

Males (hobs) are frequently about twice as large as females (jills). Male body weight typically ranges from 1.3 to 2.8 kg, with females weighing 0.5 to 1.2 kg.[2,3] There is a marked seasonal weight variation associated with the photoperiod. Weight is lost during the spring mating season and summer and is regained during the autumn months.[2]

Body temperature (rectal) ranges from 37.8 - 40° C (100.6 - 104°F), with an average of 38.8°C (102°F).[4] The ferret's normal respiratory rate is 33 - 36 breaths/minute.[2] Heart rate ranges from 210-245 beats/minute.[4] Electrocardiographic (ECG) measurements of wave amplitude are similar to those of the normal feline ECG, although the height of the ferret's R wave may approach but should not exceed 2.0 mV.[2] Radiographically, the cardiac silhouette may be interpreted incorrectly as having dilatation of the left ventricle.[2]

Hematologic values, serum chemistry values, and selected uri-

Fig.8. Rubber band foreign body removal from the small intestine of a ferret.

Fig.9. Anal sacculectomy in a ferret.

nalysis results in normal ferrets are presented in Tables 1-3, respectively. In general, hematologic and serum chemistry values are similar to those of the cat.[2]

NUTRITION

The nutritional requirements of ferrets have not been determined, but are assumed to be similar to those of cats and mink.[2] Ferrets are carnivores and may be fed cat or mink diets (dry or moist forms),[1] although feeding ferrets a high quality, dry feline growth diet is probably the most common practice. In some areas, a commercial ferret diet is also available. All rations should be checked to ensure that the protein levels are between 26-36%. A diet that is too low in protein may result in symptoms that include weight loss, neurological deficits, skin abnormalities, or reproductive problems. High fiber foods should be avoided because the ferret has little ability to digest fiber.

Ferrets can be adequately maintained on diets with fat levels from 15-30%. Fat is also a source of energy, so feeding a high energy diet with a large proportion of fat may cause the ferret to eat less because of the increased energy density. This can result in poor reproductive performance if the animal is no longer eating enough to meet its protein, vitamin, and mineral needs.

Specific carbohydrate requirements for ferrets are not known, however, most diets contain 22-44%. Although data is not available on the vitamin and mineral requirements of ferrets, diseases in ferrets due to a lack of vitamins have been reported, the most common of which is steatitis, or yellow fat disease which results from hypovitaminosis E and excess polyunsaturated fat in the diet. Steatitis is most often seen in young ferrets fed diets containing an excessive amount of fish or horsemeat.[5]

Small bones should be withheld from the diet to prevent their becoming lodged in the mouth or gastrointestinal tract.[2] Fresh water, in either a cup or drinking bottle, should be available ad libitum.

BLOOD COLLECTIONS

Blood for diagnostic purposes has been obtained from the jugular vein, cephalic vein, toenail clip, ventral tail artery, heart, femoral and jugular vein via cut-downs, retro-orbital sinus, saphenous vein, and tail clip.[1,2,6-9] For multiple blood samples, the surgical placement of a jugular catheter has been described.[1,10]

If a small blood sample ($\leq$ 0.5 ml) is required, the cephalic (using an insulin syringe) or saphenous veins may be used. If these veins are inaccessible, a toenail may be clipped and the blood collected into heparinized capillary tubes, but this procedure is generally more painful than other venipuncture techniques. For larger amounts, the preferred venipuncture site is usually the jugular vein. The jugular venipuncture technique is similar to that performed on a cat or dog. The ferret should be restrained with its front legs pulled down off the edge of the table and its head tilted back so the neck is extended (Fig. 1.); alternatively, the ferret's body may be wrapped in a towel and scruffed in dorsal recumbency for venipuncture. Some veterinarians prefer to clip the hair on the ventral neck to help visualize the vein. The vein is then distended by applying pressure on the vein proximal to the venipuncture site, and the needle is inserted into the vein. Once blood appears in the hub, the vein has been entered, so advancement of the needle should be stopped. After obtaining the sample, the syringe is withdrawn from the vein and pressure is applied to the injection site to stop the bleeding. In some cases, chemical restraint (preferably with isoflurane) may be required.

The ventral tail artery, located a few millimeters beneath the skin surface in a palpable groove on the proximal half of the tail on its ventral surface, has been used successfully by some practitioners. To perform this procedure, a 25 gauge needle is inserted cephalad at a shallow angle.[8]

Cardiocentesis can generally be performed without complication in anesthetized patients[6] in a laboratory setting, although it should not be used in client ferrets because of the potential for fatal tamponade or bleeding. In this technique, the anesthetized animal is placed in dorsal recumbency, and a 21 gauge or larger, 1½ inch needle is introduced at a 30° angle distal to the xiphoid process and directed cephalad into the animal's heart.

RESTRAINT AND ANESTHESIA

Manual restraint of ferrets often is adequate in situations where blood samples can be collected

quickly or the animal is at high anesthetic risk.[8] To prevent artifactual changes in the blood values, the animal must not be excessively excited. Warming the animal under an incandescent lamp or in a heat box for five minutes before sampling facilitates collection of blood.[8]

The ferret is best restrained when grasped at the scruff of the neck, with the other hand supporting the hind end (Fig. 2.). Ferrets strongly resist having their hind legs pulled backward. Therefore, some handlers stabilize their hindquarters by holding the base of the tail.

Chemical restraint of ferrets is commonly indicated because of the number of elective surgeries, emergency surgeries, and selected diagnostic procedures conducted, and in handling fractious patients. Table 4 presents the drugs commonly used in the sedation and anesthesia of ferrets. Food and water should be withheld from young, healthy ferrets at least six hours prior to anesthesia, or four hours in older (3 or more years old) ferrets in which insulinomas have not been ruled out. Depending on the procedure and duration of anesthesia, the ferret may need to be intubated with a 2-3 mm (I.D.) endotracheal tube or Cole tube. Placing of the tube usually requires lidocaine spray and direct visualization of the glottis.

BACTERIAL DISEASES

Abscesses caused by *Staphylococcus, Streptococcus, Pasteurella, Corynebacterium, Actinobacillus,* and hemolytic *E. coli* may occur sporadically secondary to penetrating wounds (e.g., bite wounds during breeding, penetration by ingested foreign bodies).[1] They can be prevented by reducing exposure to sharp objects in the ferret's food and environment, and by limiting breeding time. Diagnosis is based on aspiration of the fluctuant swellings (with or without draining tracts) for cytology and bacterial culture and sensitivity. Abscesses are often accompanied by pyrexia and neutrophilia. Treatment is standard as for other species; establish drainage, irrigate, and administer appropriate systemic antibiotics if indicated.

Bacterial pneumonias may be primary, or secondary to viral (e.g., influenza) infections or other illnesses (e.g., hyperadrenocorticism, megaesophagus).[1] Organisms may include *Streptococcus zooepidemicus, S. pneumoniae, E. coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Bordetella bronchiseptica,* and *Listeria monocytogenes.* Nasal discharge, dyspnea, pyrexia, lethargy, anorexia, and acute death may be observed. Diagnosis is based on clinical signs, radiographs, culture and cytology of tracheal wash samples, or necropsy findings. Differential diagnoses to consider are pleural effusion caused by heartworms (*Dirofilaria immitis*) or dilatative or hypertrophic cardiomyopathy, mycotic pneumonia, or neoplasia. Initial antibiotic therapy is modified by culture and sensitivity results or response to treatment. Gentamicin nebulization is recommended for neonatal patients.

Proliferative bowel disease is a diarrheal syndrome whose cause and effect has not been established.[11-13,15] A typical presentation is a young ferret with mucohemorrhagic or greenish diarrhea, extreme weight loss, and partial rectal prolapse (Fig. 3). Diagnosis is based on clinical signs, a palpably enlarged colon, and biopsy or necropsy findings of hyperplastic colonic mucosa. The small intestine may also be affected. Prognosis is guarded, but affected ferrets have reportedly responded to chloramphenicol at 50 mg/kg P.O., B.I.D. x 14 d.[14] Metronidazole (20 mg/kg P.O., B.I.D.), sulfasalazine, and tylosin also have been suggested as treatments,[15] and may become first-choice medications because of the current unavailability of oral chloramphenicol products.

Helicobacter (Campylobacter) jejuni can produce diarrhea (without proliferative colitis) in experimentally inoculated ferrets, and it has been isolated from asymptomatic carriers.[16] Since *H. jejuni* is a leading cause of human diarrhea, the zoonotic potential of ferret ownership must be considered. Isolation of *Helicobacter* from feces requires special culture techniques available through diagnostic laboratory services. *Helicobacter* colitis must be differentiated from Aleutian disease, salmonellosis, and eosinophilic gastroenteritis, which may also cause tarry stools (see "Viral Diseases" and "Miscellaneous Conditions").

Salmonellosis. Ferrets are susceptible to salmonellosis, and this should be considered in any ferret with gastroenteritis, although it is not believed to be common in pet animals.[1,17] The clinical syndrome does not differ significantly from that seen in other species. Ferrets may be asymptomatic carriers, or exhibit conjunctivitis, tarry stools, temperature fluctuations, dehydration, and lethargy. Diagnosis is based on culture of feces, or from liver, heart, blood, spleen, lymph nodes, intestinal contents, or bone marrow at necropsy. To avoid selection of resistant *Salmonella,* antibiotic therapy must be based on culture and sensitivity results. Fluid and electrolyte therapy is also a necessary component of treatment. Hypoglycemia and endotoxic shock may develop and require treatment.

Tuberculosis. Ferrets are highly susceptible to *Mycobacterium avium, M. bovis,* and *M. tuberculosis.*[18] Widespread infections were noted in research facilities prior to institution of proper ration preparation (i.e., raw poultry and meat, unpasteurized milk).[1] Feeding of commercially prepared foods reduces the risk appreciably. Ferret susceptibility to the disease should be kept in mind if treatment of an *M. avium* positive bird is contemplated in a ferret-owning household.

Posterior paralysis, weight loss, hepatomegaly, splenomegaly, and intestinal nodules have been reported.[18] *Mycobacterium bovis* is more likely to be disseminated than *M. avium* or *M. tuberculosis.*[1] Radiology can be used to detect nodular lesions. Exploratory laparotomy with biopsy of affected organs or

lymph nodes can lead to definitive diagnosis with acid fast bacilli on histopathology or culture. Intradermal skin testing is problematic in ferrets. Treatment is not recommended.

Leptospirosis. *Leptospira grippotyphosa* and *L. icterohaemorrhagiae* have been isolated from ferrets, but the incidence is low.[19] Ferrets exposed to domestic animals or rodents (as for rodent control) may be at some risk; however, vaccination is not currently recommended.

Listeriosis. *Listeria monocytogenes* may be considered in a differential for a ferret with central nervous system signs.[20] Rabies exposure must be definitively excluded before diagnosis is pursued. Cerebrospinal fluid (CSF) culture is necessary for diagnosis. Treatment with broad spectrum antibiotics would be indicated.

Botulism is a risk with feeding of uncooked food or soil-contaminated food. Ferrets are susceptible to toxins of *Clostridium botulinum* types A, B, and C.[21] As with other animals, clinical signs include blepharospasm, photophobia, lethargy, urinary incontinence, weight loss, ataxia, paralysis, and death. Toxin can be detected in food, gastrointestinal contents, blood, or serum of affected ferrets. Supportive therapy is necessary and antitoxin may be administered if available.

Actinomycosis (*Actinomyces*) or lumpy jaw in ferrets may result from the feeding of bones or bone fragments in the ration.[1] The incidence of infection, however, is low. Diagnosis is by isolation from lesions or special stains on histopathology. Differential diagnoses include neoplasia, and abscesses caused by *Staphylococcus* and *Streptococcus*. Treatment consists of antibiotic therapy (penicillins or tetracyclines), drainage, debridement, and irrigation.

Attention to proper feeding, therefore, can reduce the incidence of many of the above bacterial diseases in ferrets: salmonellosis, tuberculosis, botulism, campylobacteriosis, and actinobacillosis.

Mycoplasma spp. have been isolated from oral and nasal cavities of clinically normal ferrets; clinical significance is unknown.[1]

Chlamydia induces pneumonitis in experimentally infected ferrets; clinical significance is unknown.[1]

VIRAL DISEASES

Ferrets are extremely susceptible to the **canine distemper** (CD) virus, with mortality approaching 100%.[22] Clinical signs appear 7-10 days after exposure, with death occurring in 12-16 days with ferret adapted strains and 21-25 days with canine strains.[4] Early signs of CD include anorexia and mucopurulent ocular and nasal discharge. By 10-12 days, there is usually a rash under the chin and in the inguinal area,[2] and the foot pads may become hyperkeratotic. Animals that survive this catarrhal phase usually go into a central nervous system phase characterized by hyperexcitability, muscular tremors and/or convulsions, hypersalivation, coma, and death.[2,4,23] Treatment consists of supportive therapy, but is almost always futile. All ferrets should be prophylactically immunized against CD (see Table 5).

Ferrets are presumed to be extremely susceptible to **rabies** and capable of virus transmission.[2] Clinical signs seen in the ferret include anxiety, lethargy, and posterior paresis. If the ferret begins to show clinical signs, it should be euthanized and the brain sent to a laboratory for testing. Presently the U. S. Department of Agriculture has approved two killed rabies vaccines for the domestic ferret (see Table 5), although not all states have approved the vaccine for use. However, if a ferret bites someone, submission of the head for examination may be required regardless of the ferret's vaccination status.

Aleutian disease virus (ADV), a parvovirus, is infectious for ferrets but usually does not produce clinical disease. If clinical signs occur, they are nonspecific and include malaise, weight loss, melena, progressive posterior paralysis, urinary incontinence, and tremors. Hypergammaglobulinemia (greater than 20% of the plasma protein) occurs in ADV. Serologic testing (immunofluorescent antibody test, counter immunoelectrophoresis) is available to detect the presence of antibodies to ADV.[24] There is no treatment (other than supportive therapy) or prevention for this disease.

Several strains of human **influenza virus** are pathogenic for ferrets, with signs including sneezing, purulent nasal discharge, fever, anorexia, and listlessness.[2,25] Spontaneous recovery is the rule and is usually complete within one week of the onset of signs. Treatment consists of symptomatic therapy including intranasal antihistamines or other decongestants.

Ferrets are not considered susceptible to **feline panleukopenia, feline leukemia virus,** or the related viruses of **mink enteritis** and **canine parvovirus.**

MYCOTIC DISEASES

Systemic fungal disease is uncommon in ferrets, and is more likely to occur in animals maintained outdoors or those used for hunting.

Blastomycosis is contracted by inhalation of spore-laden dust. The disease is characterized by chronic granulomatous lesions in the lungs, or by chronic ulcerative skin lesions.[26] Radiographs reveal a nodular interstitial pneumonia ("snowstorm"), and cytology of lung aspirates or tissue imprints may demonstrate budding yeast forms. Definitive diagnosis is based on isolation of *Blastomyces dermatitidis* from lesions. Serology may also be helpful. Treatment is based on the canine protocol, with amphotericin B (0.25-1 mg/kg I.V. q 24-48 h until a total dose of .7-25 mg/kg is delivered), ketoconazole (10-30 mg/kg P.O. q 24 h X 60 d), supplementary fluid therapy, and monitoring of BUN. Jugular catheterization may facilitate I.V. treatments;[10] I.P. or in-

traosseous administration may be attempted if access to veins is lost.

Cryptococcosis (*Cryptococcus neoformans*) is associated with soil contact, especially when contaminated by bird feces. The primary lesion in ferrets is a meningoencephalitis.[27] Clinical signs may be absent prior to an abrupt death, or lethargy and neurological signs (including posterior paresis) may be noted. Antemortem diagnosis has not been made in ferrets, but CSF cytology (India ink preparation) or culture could be employed. Treatment would be with amphotericin B at 0.15 mg/kg I.V. three times per week for 2-4 months.

Histoplasmosis (*Histoplasma capsulatum*) is contracted by inhalation of dust containing the organism, which is a common soil isolate in the central U.S.[1] Clinical signs may include hepatosplenomegaly, abdominal pain, weight loss, pneumonia, lymphadenopathy, and ascites. Diagnosis is based on biopsy of affected organs with special histopathologic staining or by culture. Histoplasmosis is treated with amphotericin B.

Coccidiomycosis. *Coccidiodes immitis* has been reported in ferrets in the southwestern United States.[28] Clinical signs may include lethargy, weight loss, coughing, lameness, pyrexia, lymphadenopathy, splenomegaly, and draining tracts. Diagnosis is based on cytology and culture of draining wounds, chest radiographs (Fig. 4.), and serology. Successful treatment has not been reported, but if diagnosed early enough may respond to ketoconizole and amphotericin B.

Superficial mycoses include mucormycosis and dermatophytosis.

Mucormycosis is a fungal infection caused by *Absidia corymbifera* (order Mucorales).[1] It occurs secondary to ear mite (*Otodectes cynotis*) otitis. Clinical signs range from minor pruritus and ceruminous exudate, to lethargy and torticollis. Local invasion of the petrous temporal bone and brain is possible. Diagnosis is based on ear cytology (for mites and *Absidia* hyphae). Treatment is with an otitis medication of choice (e.g., Tresaderm®), preferably prior to local invasion.

Dermatomycoses (ringworm) caused by *Microsporum canis* and *Trichophyton mentagrophytes* are transmitted by contact with infected animals directly or via fomites.[1] The host range is broad and includes humans. Young animals are at greater risk. Lesions are randomly distributed discrete alopecic areas with erythema and crusts. *Microsporum canis* fluoresces under ultraviolet light, but generally diagnosis is based on skin scrapings and culture. Mild lesions may regress without treatment. Griseofulvin (25 mg/kg P.O., B.I.D.), with or without a topical antifungal medication, and cleaning and disinfection of the premises and bedding are recommended.

PARASITIC DISEASES

Ferrets are susceptible to a variety of external and internal parasites, and their diagnosis and treatment are similar to those used in the cat. Ferrets can be infested with fleas (*Ctenocephalides* spp.), ear mites (*O. cynotis*), and ticks. Mange (*Sarcoptes scabiei*) can occur in ferrets in either of two forms: generalized with patchy alopecia or localized to the feet. Treatment consists of ivermectin (0.2 - 0.4 mg/kg IM or SQ) (treatment of choice) and/or 0.5% carbaryl shampoos weekly for 3-5 weeks,[4] in conjunction with environmental cleaning.

Internal parasites in ferrets include coccidia (*Isospora*), *Toxoplasma, Sarcocystis, Giardia,* roundworms (*Toxascaris, Toxocara*), hookworms (*Ancylostoma*), tapeworms, and heartworms (*D. immitis*). Natural infection with heartworms was first reported by Carpenter and Hillman (1978),[29] but has since been observed in a number of ferrets.[30-32] Very few adult worms are required to cause signs of congestive heart failure (i.e., ascites and respiratory distress); these low numbers do not usually produce antigen levels high enough to generate a positive occult heartworm test. Heartworm prevention is indicated for ferrets in endemic areas (i.e., housing ferrets indoors during the mosquito season and/or administering diethylcarbamazine [2.75-5.5 mg/kg], milbemycin oxime [1.15-2.30 mg/kg], or ivermectin [6μg/kg] prophylactically. Avermectin B_1 a (0.2 mg/kg) and melarsoprol (100 mg/kg) as a single dose, 38-42 days after infection, have been shown to be effective against developing forms in ferrets.[33]

ENDOCRINE DISORDERS

Insulinoma or pancreatic endocrine neoplasia, is one of the most common endocrine neoplasias of ferrets.[1,34] Proliferation of pancreatic islet cells, predominantly beta cells, results in excessive production of insulin. Cells producing glucagon, somatostatin, and pancreatic polypeptide are also found in pancreatic endocrine neoplasms, but their effects are masked by the greater numbers of beta cells.[35] Hyperinsulinism leads to hypoglycemia, to which most clinical signs are referable.

Clinical signs commonly observed are episodic weakness, lethargy, hypersalivation, dehydration, ataxia, posterior paresis or paralysis, and seizures.[34-40] Hypoglycemia (glucose <60-80) is usually the only abnormality in the clinical chemistries. A presumptive diagnosis can be made quickly on an animal showing typical signs with the use of a glucometer by collecting a drop of blood from a toenail clip. Other rule outs for hypoglycemia include sample mishandling (failure to separate serum from red cells in a timely manner), other overwhelming neoplasia, diffuse liver disease, starvation, sepsis, and hypoadrenocorticism. These can usually be discounted through CBC, chemistry panel, and radiographs. Ultrasound examination might possibly reveal a pancreatic nodule. Although some authors question the diagnostic value of insulin/glucose ratios[34] and measurement of absolute insulin values, others believe that the following ratios may

aid in making the diagnosis:[41,42] G/I < 2.5 mg/uU; I/G > 0.3 uU/mg; and amended insulin glucose ratio >30. The amended insulin glucose ratio is calculated as follows:

$$\frac{\text{insulin in uU/ml} \times 100}{\text{glucose [mg/dl]} - 30}$$

Surgical excision of the insulin-secreting mass(es) is the initial treatment of choice, but affected ferrets eventually require medical therapy as well.[34,36] Medical management may be required for presurgical stabilization, if surgery is not an option, or if the problem recurs after surgery. Medical management may include frequent high protein meals, prednisolone at 0.5 - 1.0 mg/kg/day P.O., and diazoxide 10-40 mg/kg/day P.O.[34] Brewers yeast (containing chromium) (1/8-1/4 tsp. B.I.D. in food)[43] may also be added to the treatment protocol. Prednisolone is used for its gluconeogenic effects. Diazoxide directly inhibits insulin release, but may cause nausea, vomiting, and obesity. Somatostatin has been administered to humans with insulinomas; preliminary trials of its use in ferrets with insulinoma have been inconclusive.[34]

Surgery should be considered palliative, not curative, because pancreatic endocrine tumors in ferrets tend to metastasize, although the disease progresses slowly.[35] Metastatic sites include regional lymph nodes, liver, spleen (Fig. 5), duodenum, and mesentery. Although a nodule is usually visible in the pancreas, the surgery occasionally requires tactile detection of the mass(es). The patient should be maintained on 5% dextrose during surgery and blood glucose should be monitored periodically. Blood glucose often rises dramatically with the removal of the neoplasia, and transient to prolonged hyperglycemia is possible.[42] Fluids (LRS if hyperglycemia has resulted) should be continued and the ferret maintained N.P.O. for 24 hours post surgery to guard against pancreatitis. Blood glucose should be monitored until it stabilizes in the normal range, and then every three months thereafter. Survival times after diagnosis range from 4 to 24 months, with appropriate medical and surgical management.[34]

Hyperadrenocorticism (adrenal-associated endocrinopathy) in ferrets is generally caused by a functional adrenal adenoma or carcinoma. The left adrenal gland is more commonly affected than the right, but disease may be present bilaterally.[34] Excess cortisol or estrogens may be produced in some patients. Clinical features include progressive bilaterally symmetrical alopecia, thin skin, polyuria and polydipsia, weakness, increased susceptibility to infectious disease and, in some females, vulvar swelling if estrogens are being produced.[34,44] Abdominal radiographs and ultrasound examination (enlarged adrenal[s]) are useful diagnostic aids (Fig.6).[1,34,45-47] Additionally, a CBC may reveal an anemia, lymphopenia, and eosinopenia. Alkaline phosphatase is usually not elevated. ACTH stimulation and dexamethasone suppression tests have been described in normal ferrets,[48-50] but may or may not be diagnostic in ferret hyperadrenocorticism.[45] Exploratory laparotomy and adrenalectomy is the preferred treatment, especially if the tumor is unilateral.[1,45,51] The right adrenal gland, however, can be extremely difficult to remove because of its intimate association with the caudal vena cava. An exploratory may also reveal other concurrent neoplasms (i.e., insulinoma, lymphosarcoma, etc.). Alternately, medical treatments include mitotane (50 mg/kg P.O. q 24h x 7d, then q 72h until signs resolve), and some have attempted treatment with ketoconazole (15 mg/kg P.O. B.I.D.);[34] carcinomas, however, do not respond to medical treatment.

Diabetes mellitus is not well studied or characterized in domestic ferrets.[1] Diabetes mellitus was diagnosed in a black-footed ferret with polyuria, polydipsia, polyphagia, dehydration, weight loss, marked hyperglycemia (724 mg/dl), glycosuria, and ketonuria.[52] The patient was successfully maintained on intermediate-acting insulin (NPH Iletin, Eli Lilly and Co., Indianapolis, IN) for 16 months. Diagnosis and treatment of diabetes mellitus in ferrets may be based on feline protocols.

Hyperestrogenic anemia. Ferret females are seasonally polyestrous induced ovulators.[1] Vulvar swelling is maximal about 1 month after the onset of estrus and regresses 2-3 weeks following mating. Estrus can continue six months if the jill is not bred. The gestation period is 42 days, and, if fertilization fails, a pseudopregnancy of 40-42 days ensues. If the jill is not bred, high estrogen levels cause suppression of myeloid, erythroid, and megakaryocytic cell lines.[53-56] (Hobs administered exogenous estrogens respond similarly.)[53,57] Clinical signs derive from the bone marrow suppression and will be evident in about 50% of females experiencing prolonged estrus. These include bilateral symmetrical alopecia of the ventral abdomen and tail, anorexia, weight loss, vulvar swelling with serous to mucopurulent discharge, lethargy, bacterial infections (pneumonia, pyometra, septicemia) secondary to leukopenia, petechiae and ecchymoses secondary to thrombocytopenia (<20,000/ul), posterior paresis (hemorrhage in the CNS), and pale mucous membranes (PCV <20%, sometimes <10%).[1,53-56,58] Death may occur after 2 months (or longer) duration of estrus. By the time the ferrets are presented for treatment the condition is usually critical and many die. An estrous intact female with anemia can be assumed to have this condition, although the diagnosis may be obscured by secondary bacterial infections or CNS signs.[1]

The condition can be prevented in a variety of ways:[1] breeding; breeding to a vasectomized hob (induces pseudopregnancy); inducing ovulation (and hence pseudopregnancy) by administration of 20 ug GnRH I.M. or 50-

100 IU HCG S.Q. 10 or more days after the onset of estrus[59] (vulvar swelling should diminish in one week; if not, repeat the treatment); altering light cycles (14 h light: 10 h dark prevents estrus in many jills); and performing an ovariohysterectomy (OHE) (breeders usually have this done at weaning, which has reduced the incidence of this condition). Megestrol acetate also prevents estrus but introduces a high risk of pyometra and should not be used.

Treatment of hyperestrogenic anemia requires medical restoration of the oxygen carrying capacity of the blood (transfusion) and reduction of the high levels of estrogen surgically (OHE). Antiestrogens (e.g., clomifene, tamoxifen) are ineffective because they are estrogenic in ferrets.[1] Transfusions can be performed as necessary,[60] and transfusions may be multiple without cross matching as there are no detectable ferret blood groups.[61] Endogenous RBC production may be stimulated with B vitamins, iron dextran (10 mg/kg I.M. q 1 week), or nandrolone decanoate (1-5 mg/kg I.M. q 1 wk).[62] Antibiotics may be employed to prevent secondary bacterial infections during the period of leukopenia. Corticosteroids have been recommended to increase capillary integrity.[1] Some authors recommend conservative medical treatment (inducing ovulation, blood transfusion) to stabilize the ferret, followed by surgery.[53] Surgery results in a more rapid reduction in blood estrogen levels, but may require blood transfusions and careful hemostasis. Resolution of bone marrow suppression may take months.

Spayed female ferrets with vulvar swelling may have hyperadrenocorticism, ectopic or remnant ovarian tissue (Fig 7), or estrogen secreting neoplasia.

NON-ENDOCRINE NEOPLASIA

Lymphosarcoma is the most commonly reported non-endocrine neoplasia in ferrets.[1,63] Clinical signs are varied and depend on the organ(s) involved. Lymph nodes, spleen, thymus, liver, meninges, stomach, thoracic and abdominal cavities, and blood may be affected.[1,64-66] Careful attention to lymph node palpation is indicated in all ferrets during routine physical examinations. Diagnosis of lymphosarcoma may be based primarily on cytology and histopathology, although CBC, chemistry panel, radiographs, and ultrasound are all helpful. Lymphosarcoma may be responsive to chemotherapy with prednisolone/ vincristine/ cyclophosphamide,[66,67] prednisone/ asparaginase/ vincristine/ cyclophosphamide/ methotrexate,[67] and doxorubicin (usually in combination protocols). A CBC should be monitored at each treatment for signs of toxicity. A link between lymphosarcoma and Aleutian disease virus (a parvovirus) or a suspected ferret retrovirus has been suggested.[1]

Ovarian and other **reproductive organ tumors** are common forms of neoplasia in ferrets.[1] Clinical signs are usually related to fertility problems. Mammary tumors are uncommon.[1]

Skin and **subcutaneous tumors** also occur in ferrets, with mast cell tumors being the most common. Metastasis may occur with mast cell tumors, as well as with squamous cell carcinomas and sebaceous gland adenocarcinomas. Other less common skin tumors include sebaceous gland adenomas, hemangiomas, and tail chordomas/chondrosarcomas. All skin masses on ferrets should have an excisional biopsy performed for rapid diagnosis and treatment.

MISCELLANEOUS CONDITIONS

Dilatative and hypertrophic cardiomyopathy (DCM and HCM). Ferrets with cardiomyopathy have a history of chronic weight loss and lethargy, and may be dyspneic.[68] Diagnostic features include generalized cardiomegaly, pleural effusion, pulmonary congestion and edema on radiographs, and hepatosplenomegaly on palpation. An ECG can demonstrate PVC's, tall and wide QRS complexes, A-V block, and depressed ST segments.[1,69] Echocardiograms of DCM demonstrate four chamber enlargement and decreased cardiac output, fractional shortening, and contractility.[1] HCM also involves left atrial dilation evident echocardiographically, but the left ventricular free wall (and/or interventricular septum) is thickened and left ventricular contractility may be increased.[69] Furosemide (1-4 mg/kg I.M., then P.O., B.I.D.), and digoxin elixir (0.01 mg/kg P.O. q 12-48h) with monitoring of digoxin blood levels, may be used for treatment of resultant heart failure, although prognosis is guarded to poor.[68] There has been some speculation that incidence of DCM has decreased with the greater supplementation of taurine in cat foods, which are frequently fed to ferrets.

Urolithiasis is commonly encountered.[1,70] Frequent urination, straining, licking, blood-tinged urine, and occasionally complete blockage may be observed. Diagnosis and treatment are similar to that in cats, except that with complete blockage of a male ferret, urinary catheterization is difficult, if not impossible, due to the very narrow urethral diameter through the os penis. Cystocentesis provides temporary relief, and a urethrostomy may be necessary. Crystal and stone analysis should be performed if possible to guide dietary therapy (usually low magnesium and urinary acidification).[71-73] Since reports are lacking with regards to sterile versus bacterial cystitis, antibiotic treatment would be prudent pending culture and sensitivity results.

The inquisitive nature of young ferrets makes them prone to gastrointestinal foreign bodies (Fig. 8.).[1] Older ferrets may develop gastrointestinal obstruction secondary to hairballs. Presenting signs usually include anorexia and nausea, evidenced by hypersalivation, pawing at the mouth, and, occcasionally, vomiting. Diagnosis may be made with abdominal palpation and radiography (with or

without contrast media). Surgical removal of foreign bodies is necessary for resolution of the problem.

Gastric dilatation with subsequent shock has been reported in association with pyloric foreign bodies, prolonged fasting followed by overeating, or with sudden dietary changes.[1] *Clostridium welchii* may be associated with this condition, producing gas distension. Gastric dilatation is treated by surgical exploration and decompression, and therapy for shock.

Gastric ulcers can cause mild or severe disease.[1] *Helicobacter mustelae* has been associated with gastric ulcers in ferrets.[73] Clinical signs may include vague abdominal distress, vomiting, halitosis, blood in vomitus, and melena. Definitive diagnosis is difficult. If suspected, gastric ulcers can be treated empirically with amoxicillin (20 mg/kg P.O. q 24 h), gastrointestinal protectants, and cimetidine (5-10 mg/kg P.O. q 6-8 h).[74]

Eosinophilic gastroenteritis is a syndrome characterized by bloody, mucoid diarrhea, anorexia, and weight loss, with vomiting also noted in some patients.[75,76] Clinicopathologic findings include hypoalbuminemia (≤ 3.0 gm/dl) and eosinophilia (range 10-35%, absolute count 1190 to 4480, compared to normal range 0-7%). Eosinophilic gastroenteritis is diagnosed by exploratory laparotomy and histopathologic evaluation of stomach, small intestine, and mesenteric lymph node biopsies.

Treatment includes supportive care for the anorectic ferret; use maintenance requirements of 60 ml/kg/day and 200-300 kcal/kg/day in figuring your patient's needs.[1] Prednisone may relieve clinical signs (1.25-2.5 mg/kg P.O. q 24 h, gradually decreasing to q 48 h); it is generally administered for life. Although parasites have not been noted in cases of eosinophilic gastroenteritis, ivermectin 0.4 mg/kg S.Q. (repeated two weeks later) apparently resolved the syndrome in one ferret.[75]

Retained deciduous teeth may be present (permanent teeth erupt between 50-74 days of age),[1] and although these should be extracted promptly, they often come out in their own in time.

Periodontal disease is frequently observed, and prophylactic dental care should receive more attention in practice.

Splenomegaly is a common incidental finding, and has been associated with insulinoma, hyperadrenocorticism, Aleutian disease, lymphosarcoma, fungal disease, cardiac disease, hemangioma, hemangiosarcoma, and ferret life in general (idiopathic).[1] An association with clinical disease and a thorough workup including CBC, chemistry panel, radiographs, ultrasound, and fine needle aspirate or biopsy of the spleen should be performed to rule out the above diseases before considering splenectomy. In actuality, splenectomy in ferrets is seldom indicated.

Posterior paresis can be a clinical feature of multiple diseases: insulinoma, lymphosarcoma, tuberculosis, hyperestrogenic bone marrow suppression (hematomyelia), botulism, fungal myelitis, trauma, Aleutian disease, and rabies.

PREVENTIVE MEDICINE

Although many individuals assume that the primary role of a veterinarian in ferret management is to provide medical care for sick or injured animals, in actuality a veterinarian's role is to implement a strong preventive medicine program to reduce medical problems. Preventive medicine includes all the practices that strengthen genetic and immunological resistance to disease, provide sound nutrition, and minimize exposure to disease agents. Annual physical examination of young ferrets, semiannual examination (including CBC and fasting blood glucose) of ferrets over three years of age, vaccination for canine distemper virus and rabies (in some states), routine parasite examinations, dental prophylaxis, and, in some cases, heartworm prevention,[77] for example, are essential components of a good preventive medicine program and will contribute greatly to the overall length and quality of life for the ferret.

Two prophylactic surgical procedures are commonly practiced in the pet ferret. Males are castrated to decrease aggression, inappropriate urination, and body odor, and females are spayed to avoid pregnancy, false pregnancy, and pancytopenia during estrus and to reduce body odor.[78] Although animals can be descented to alleviate problems associated with voluntary anal gland expression during excitement or anxiety (Fig. 9), clients should be made aware that the majority of ferret body odor is associated with sebaceous secretions, which are markedly reduced after castration or ovariohysterectomy. Table 5 outlines a proposed schedule of vaccination and routine prophylactic care.

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Table 1: Hematologic values of normal ferrets[a]

Parameter	Albino ferrets		Fitch ferrets	
	Male	Female	Male	Female
Packed cell volume(%)	55.4 (44-61)[b]	49.2 (42-55)	43.4 (36-50)	48.4 (47-51)
Hemoglobin (g/dl)	17.8 (16.3-18.2)	16.2 (14.8-17.4)	14.3 (12-16.3)	15.9 (15.2-17.4)
Red blood cells (10^6/mm^3)	10.23 (7.3-12.18)	8.11 (6.77-9.76)	-	-
White blood cells (10^3/mm^3)	9.7 (4.4-19.1)	10.5 (4.0-18.2)	11.3 (7.7-15.4)	5.9 (2.5-8.6)
Differential (%)				
Bands	-	-	0.9 (0-2.2)	1.7 (0-4.2)
Neutrophils	57.0 (11-82)	59.5 (43-84)	40.1 (24-78)	31.1 (12-41)
Lymphocytes	35.6 (12-54)	33.4 (12-50)	49.7 (28-69)	58.0 (25-95)
Monocytes	4.4 (0-9)	4.4 (2-8)	6.6 (3.4-8.2)	4.5 (1.7-6.3)
Eosinophils	2.4 (0-7)	2.6 (0-5)	2.3 (0-7)	3.6 (1-9)
Basophils	0.1 (0-2)	0.2 (0-1)	0.7 (0-2.7)	0.8 (0-2.9)
Platelets (10^3/mm^3)	453 (297-730)	545 (310-910)	-	-
Reticulocytes (%)	4.0 (1-12)	5.3 (2-14)	-	-

[a] Data modified from Fox (1988)[1] and Thornton (1979).[3]
[b] Mean and range.

Table 2. Serum chemistry values of normal ferrets.[a]

Parameter	Albino Ferrets	Fitch ferrets
Alanine aminotransferase (ALT) (SGPT) (IU/L)	-	170 (82-289)
Alkaline phosphatase (IU/L)	23 (9-84)	53 (30-120)
Aspartate aminotransferase (AST) (SGOT) (IU/L)	65 (28-120)	-
Bilirubin, total (mg/dl)	< 1.0	-
Blood urea nitrogen (mg/dl)	22 (10-45)	28 (12-43)
Calcium (mg/dl)	9.2 (8.0-11.8)	9.3 (8.6-10.5)
Carbon Dioxide	-	24.9 (20-28)[c]
Chloride (mmo/L)	116 (106-125)	115 (102-121)
Cholesterol (mg/dl)	165 (64-296)	-
Creatinine (mg/dl)	0.6 (0.4-0.9)	0.4 (0.2-0.6)
Gamma glutamyltransferase (GGT) (IU/L)	-	5
Glucose (mg/dl)	136 (94-207)[b]	101 (62.5-134)
Lactic dehydrogenase	-	460 (241-752)[c]
Lipase (U/L)	-	0-200
Phosphorus (mg/dl)	5.9 (4.0-9.1)	6.5 (5.6-8.7)
Potassium (mmol/L)	5.9 (4.5-7.7)	4.9 (4.3-5.3)
Protein, total (g/dl)	6.0 (5.1-7.4)	5.9 (5.3-7.2)
Albumin (g/dl)	3.2 (2.6-3.8)	3.7 (3.3-4.1)
Albumin/globulin (g/dl)	-	1.8 (1.3-2.1)[c]
Globulin (g/dl)	-	2.2 (2.0-2.9)[c]
Sodium (mmol/L)	148 (137-162)	152 (146-160)
Triglycerides	-	18 (10-32)[c]

[a] Data modified from Fox (1988),[1] Thornton et al. (1979),[3] and Kawasaki (1992).[77]
[b] Mean and range.
[c] From males only.

Table 3. Selected urinalysis results in normal ferrets.[a]

Parameter	Male	Female
Volume (ml/24 hr)	26 (8-48)[b]	28 (8 -140)
Sodium (mmol/24 hr)	1.9 (0.4-6.7)	1.5 (0.2 - 5.6)
Potassium (mmol/24 hr)	2.9 (1.0-9.6)	2.1 (0.9 - 5.4)
Chloride (mmol/24 hr)	2.4 (0.7-8.5)	1.9 (0.3 - 7.8)
pH	6.5 - 7.5	6.5 - 7.5
Protein (mg/dl)	7 - 33	0 - 32

[a] Data from Thornton et al. (1979).[3]
[b] Mean and range.

Table 4. Drugs used commonly in the sedation and anesthesia of ferrets.[a]

Drug	Dosage	Route[b]
Sedatives/tranquilizers		
Acepromazine	0.2-0.5 mg/kg	IM, SQ
Xylazine	1.0 mg/kg	IM, SQ
Diazepam	1.0-2.0 mg/kg	IM
Ketamine	10-20 mg/kg	IM
Preanesthetics		
Atropine	0.05 mg/kg	IM, SQ
Acepromazine	0.1 - 0.25 mg/kg	IM, SQ
Anesthetics		
Ketamine	35 mg/kg (range 30-60)	IM
Ketamine-acepromazine (9:1)	20-35 mg/kg (K)	IM, SQ
	0.2-0.35 mg/kg (A)	IM, SQ
Ketamine-xylazine[c]	25 mg/kg (range 20-30) (K)	IM
	2 mg/kg (range 1-4) (X)	IM, SQ
Ketamine-diazepam	25-35 mg/kg (K)	IM
	2-3 mg/kg (D)	IM, SQ
Isoflurane[d]	1.5 - 3% maintenance	Inhal
Halothane	3-3.5 induction	Inhal
	0.5 - 2.5 maintenance	
Methoxyflurane	1-3% induction	Inhal
	0.3 - 0.5% maintenance	

a Modified from Ryland (1983),[2] Fox (1988),[1] and Burke (1988).[4]
b IM = intramuscular; SQ - subcutaneous; Inhal - inhalation.
c Use with caution. Burke 1988[4] has reported severe depression and two fatalities in ferrets given ketamine-xylazine mixtures. If ketamine-xylazine is used, xylazine is usually administered first, followed by ketamine, although may be given concurrently in same syringe.
d Anesthetic of choice.

Table 5. Proposed schedule of vaccinations and routine prophylactic care in the ferret.[a]

Age	Recommendation
4-6 weeks	CDV[b] vaccination if dam is unvaccinated
6-8 weeks	CDV vaccination; physical examination; fecal examination
≈ 10 weeks	CDV[c] vaccination; physical examination; fecal examination
12-14 weeks	CDV[c] vaccination; rabies vaccination[d]; physical examination, fecal examination (optional); see e.
6-8 months	Spay/castrate; fecal examination; remove musk glands (optional); see e
1 year	CDV booster; rabies booster[d]; physical examination including dental prophylaxis; fecal examination if indicated; CBC; see e.
2 years	CDV booster; rabies booster;[d] physical examination including dental prophylaxis; fecal examination if indicated; CBC; see e.
3 years and older (every 6 mos)	CDV booster; rabies booster (annual);[d] physical examination including dental prophylaxis, fecal examination if indicated; CBC; fasting blood glucose; see e.

a Modified after Ryland (1983)[2] and Burke (1988);[4]
b CDV = canine distemper vaccine (nonferret origin; MLV of chick-embroyo origin is recommended); Fromm-D®, Solvay Veterinary, or Fervac-D®, Harlan Sprague Dawley, Inc.
c Vaccinations are generally administered at 2-3 week intervals until the ferret is 12-14 weeks of age.
d Only a killed virus vaccine (Imrab 3® Rhone Merieux, Inc., or Prorab 1®, Intervet, Inc.) should be used; not approved in all states; need depends on circumstances.
e Heartworm prevention may be indicated in ferrets in endemic areas.

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