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Clinical Techniques

RESTRAINT

- Most pet ferrets are gentle, tractable, and are easy to restrain without assistance. Often only minimal restraint is needed when performing a physical examination. Some ferrets may be lightly restrained on the examination table. Others will need to be restrained in a firmer manner.
- Tractable ferrets can be lightly restrained on an examination or treatment table by placing one hand under the chest and lifting slightly.
- Energetic ferrets may be restrained by scruffing (Fig. 175-1). Use one hand to grasp the skin over the back of the neck and lift the ferret up, suspending all the limbs. Stroke the abdomen with a downward motion to relax the ferret. The ferret’s back may be supported with the other hand, or the ferret may then be reclined along the forearm of the arm used to scruff the ferret. Most ferrets will become very relaxed, although some young ferrets and some females may resist.
- Firm restraint is often required when administering vaccinations or when performing treatment procedures. Control the head by scruffing, or by cupping the back of the ferret’s neck and placing the thumb and fingers along the caudal border of the mandibles. Place the other hand over the pelvis to restrain the hindquarters on the table top with the hind legs underneath the body; do not pull the legs back.
- Aggressive ferrets, such as nursing females, kits, or ferrets raised with little human contact, are uncommon. Restrain these ferrets by the scruff of the neck, using the techniques previously described. Avoid using leather gloves, which are awkward. Use sedation if necessary.

DIAGNOSTICS

Blood Collection

There are several suitable sites for blood collection in ferrets:

- Cephalic vein
- Lateral saphenous vein
- Jugular vein
- Cranial vena cava
- Ventral tail artery

**Key Point** Do not perform cardiac puncture or retro-orbital bleeding.

Indications

- Cephalic or lateral saphenous venipuncture may be used to obtain small amounts of blood (<1.0 ml) for a packed cell volume (PCV), blood glucose, complete blood cell count (CBC), or serum biochemistry analysis.
- The jugular vein, cranial vena cava, cephalic vein, or ventral tail artery may be used to collect larger volumes of blood.
- Use the jugular vein to collect blood for transfusion.
### Other Considerations

- Sedation: Collection of blood from the jugular veins, cranial vena cava, or ventral tail artery may require sedation and/or the assistance of two people for restraint. Sedation is rarely required for venipuncture of cephalic, lateral saphenous, or jugular veins.
- If necessary, clip the hair over the venipuncture site to see the vein.
- The normal hematocrit of ferrets is high; draw three times as much blood as the volume of plasma or serum required. (See Tables 175-1 and 175-2 for blood values reported in normal ferrets.)
- The PCV, red blood cell count (RBC), hemoglobin, white blood cell count (WBC), and plasma proteins often rapidly decrease after induction of isoflurane anesthesia.

### Techniques

#### Cephalic Vein

- Collect blood from the cephalic vein in ferrets using the same restraint technique described for dogs and cats.
- Use an insulin syringe with a 28-gauge needle or a 1cc tuberculin syringe with a 25-gauge needle to collect volumes of blood up to 1.0ml. Larger volumes of blood may be collected with a 3cc syringe and a 25-gauge needle.
- Alternatively, place a 25-gauge needle in the vein and collect blood directly from the hub into small blood collection tubes.

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### Table 175-1. REFERENCE RANGES FOR HEMATOLOGIC VALUES IN FERRETS

| Value                  | Sex | Range         | Mean | Range         | Mean |
|------------------------|-----|---------------|------|---------------|------|
| **Hematocrit (%)**     | ♂️  | 46–57         | 49.1 | 44–61         | 55.4 |
|                        | ♀️  | 47–51         | 48.4 | 42–55         | 49.2 |
| **Hemoglobin (g/dl)**  | ♂️  | 15.2–17.7     | 16.1 | 16.3–18.2     | 17.8 |
|                        | ♀️  | 15.2–17.4     | 15.9 | 14.8–17.4     | 16.2 |
| **Red blood cells (×10^6/μl)** | ♂️  | 7.30–12.18    | 10.23| 6.77–9.76     | 8.11 |
|                        | ♀️  | 5.6–10.8      | 7.3  | 4.4–19.1¹     | 9.7  |
| **Neutrophils**        | ♂️  | 616–7020/μl   | 2059/μl | 11–82%       | 57.0% |
|                        | ♀️  | 725–2409/μl   | 1825/μl | 43–84%       | 50.5% |
| **Lymphocytes**        | ♂️  | 1728–4704/μl  | 3571/μl | 12–54%       | 35.6% |
|                        | ♀️  | 1475–5590/μl  | 3426/μl | 12–50%       | 33.4% |
| **Monocytes**          | ♂️  | 0–432/μl      | 176/μl | 0–9%         | 4.4% |
|                        | ♀️  | 100–372/μl    | 263/μl | 2–8%         | 4.4% |
| **Eosinophils**        | ♂️  | 112–768/μl    | 378/μl | 0–7%         | 2.4% |
|                        | ♀️  | 50–516/μl     | 214/μl | 0–5%         | 2.6% |
| **Basophils**          | ♂️  | 0–112/μl      | 50/μl | 0–2%         | 0.1% |
|                        | ♀️  | 0–172/μl      | 48/μl | 0–1%         | 0.2% |
| **Bands**              | ♂️  | 0–972/μl      | 235/μl | 0–248/μl     | 99/μl |
|                        | ♀️  | 0–248/μl      | 99/μl | 297–730      | 453  |
| **Platelets (×10^3/μl)** | ♂️  | 297–730      | 453  | 310–910      | 545  |
|                        | ♀️  | 310–910      | 545  |                      |      |
| **Mean corpuscular volume (μm³)** | ♂️  | 54  |                      |      |
|                        | ♀️  | 61  |                      |      |
| **Mean corpuscular hemoglobin (pg)** | ♂️  | 17.6 |                      |      |
|                        | ♀️  | 19.9 |                      |      |
| **Mean corpuscular hemoglobin concentration (%)** | ♂️  | 32.2 |                      |      |
|                        | ♀️  | 32.8 |                      |      |

* Males all castrated.
† Males all intact.

¹These white blood cell counts are higher than those currently seen in clinical practice. At our laboratories, the normal white blood cell count is 3–8 × 10³/μl, and most are 4–6 × 10³/μl.

Adapted with permission from Lee EJ, Moore WE, Fryer HC, Minocha HC: Hematological and serum chemistry profiles of ferrets (Mustela putorius furo). Lab Anim 16:133–137, 1982; and Thornton PC, Wright PA, Sacra PJ, Goodier TEW: The ferret, Mustela putorius furo, as a new species in toxicology. Lab Anim 13:119–124, 1979. Copyrights 1979 and 1982, Macmillan Magazines Limited.
Lateral Saphenous Vein

- The lateral saphenous vein runs diagonally across the lateral surface of the hindleg, just proximal to the hock.
- Use an insulin syringe with a 28-gauge needle or a 1-ml syringe with a 25-gauge needle to collect small blood samples (<1.0 ml).

Jugular Vein

- Several methods are described for jugular venipuncture:
  - The ferret may be placed in sternal recumbency at the edge of the table. Extend the head dorsally with the front legs held down, out of the path of the venipuncturist.
  - Alternatively, wrap the ferret in a towel with the front legs drawn back along the thorax, leaving the head and neck extended from the towel. Position the ferret in dorsal recumbency, and extend the head and neck by scruffing.
  - Restraining the ferret in the same manner described for the cranial vena cava (see below).

Cranial Vena Cava

- This procedure is referred to as cranial vena cava venipuncture, but, in reality, blood is collected from the jugular vein as it passes into the thoracic cavity at the thoracic inlet.

Key Point

Blood collection from the cranial vena cava requires complete immobilization of the ferret; otherwise, do not attempt the procedure. Use sedation or the help of two assistants for restraint.

- Do not use this site if intrathoracic disease (e.g., mediastinal mass, mega-esophagus) or coagulopathy is suspected.

Technique

1. Place the ferret in dorsal recumbency. One assistant restrains the head and neck in extension while holding the forelegs alongside the thorax. A second assistant restrains the hindquarters without pulling the rear legs back. Precise positioning facilitates the procedure.
2. Palpate the manubrium and locate the "notch" on either side where the manubrium and the first rib meet.
3. Insert a 3-ml syringe with a 25-gauge, 5/8-inch needle at either notch and direct the needle at a shallow angle (<45 degrees) along an imaginary line running from the notch toward the opposite rear leg.
4. Insert the needle to the hub and gently aspirate while withdrawing the needle.
5. If the ferret struggles, abort the procedure, and do not make a second attempt until the ferret is quiet. Ferrets that struggle persistently should be sedated for this procedure.

Ventral Tail Artery

- Venipuncture at this site may be painful.

Technique

1. Scruff the ferret and place it in dorsal recumbency (wrapping it in a towel may help with restraint), or anesthetize the ferret and place it in dorsal recumbency.
2. Prepare the site aseptically.
3. Use a 1- or 3-cc syringe and a 22- to 25-gauge needle for sample collection.
4. Insert the needle on the ventral midline of the tail at 30-degree angle toward the body approximately 2 to 3 cm from the anus.
5. Advance the needle to the bone; withdraw it slowly while applying a slight vacuum to the syringe.

### Table 175-2. REFERENCE RANGES FOR SERUM BIOCHEMISTRY VALUES IN FERRETS

| Value                  | Albino* | Fitch† |
|------------------------|---------|--------|
| Total protein (g/dl)   | 5.1–7.4 | 5.3–7.2 |
| Albumin (g/dl)         | 2.6–3.8 | 3.3–4.1 |
| Glucose (mg/dl)        | 94–207  | 62.5–134|
| Fasting glucose (mg/dl)| 10–45   | 12–45  |
| Blood urea nitrogen (mg/dl) | 0.4–0.9 | 0.2–0.6 |
| Sodium (mmol/L)        | 137–162 | 146–160|
| Potassium (mmol/L)     | 4.5–7.7 | 4.3–5.3 |
| Chloride (mmol/L)      | 106–125 | 102–121|
| Calcium (mg/dl)        | 8.0–11.8| 8.6–10.5|
| Phosphorus (mg/dl)     | 4.0–9.1 | 5.6–8.7 |
| Alanine aminotransferase (U/L) | 28–120 | 57–248§ |
| Aspartate aminotransferases (U/L) | 9–84 | 30–120 |
| Alkaline phosphatase (U/L) | <1.0 | 0–0.18 |
| Bilirubin (mg/dl)      | 64–296  | 119–209§|
| Cholesterol (mg/dl)    | 16.5–28 | 16–28§ |

*Combined values of male (N = 40) and female (N = 24) ferrets from Thornton PC, Wright PA, Sacra PJ, Goodier TEW: The ferret, Mustela putorius furo, as a new species in toxicology. Lab Anim 13:119–124, 1979.
†Combined values of intact male, female, and castrated male ferrets (total N = 13, aged 4–8 mo) from Lee EJ, Moore WE, Fryer HC, Minocha HC: Haematological and serum chemistry profiles of ferrets (Mustela putorius furo). Lab Anim. 16:133–137, 1982, except where noted.
‡From Brown S: Personal communication, 1995.
§Combined values from cardiac and orbital venipuncture of male ferrets (N = 16) from Fox JG: Normal clinical and biologic parameters. In: Fox JG (ed): Biology and Diseases of the Ferret. Philadelphia: Lea & Febiger, 1988, pp 159–173.
6. Apply direct pressure to the site for several minutes after the needle is withdrawn.

**Collection of Blood for Transfusion**

A detailed discussion of the techniques used for blood collection for transfusion and blood transfusion is presented in the Hematopoietic System section in this chapter.

**Diagnostic Imaging**

**Radiography**

- Use standard radiographic techniques (see Chapter 4), including sedation for correct positioning and to limit exposure of the technician, as well as high detail radiographic film and cassettes.
- When interpreting films, it helps to think of the ferret as an elongated cat.
- The kidneys are relatively short (about two lumbar vertebrae in length).
- Splenomegaly is a common radiographic finding.
- For barium-contrast radiography of the gastrointestinal (GI) tract, give 15 ml/kg of 20% barium solution PO via syringe feeding or lavage tube. Most ferrets will accept syringe feeding of barium. Normal GI transit time is about 3 to 4 hours.

**Ultrasound**

- Echocardiography may be used to evaluate the heart in ferrets with suspected cardiac disease (see “Cardiovascular Disease”).
- Other uses of ultrasound in ferrets include investigation of intra-abdominal or intra-thoracic masses, organomegaly, paraurethral cysts, or prostatic cysts.

**Bone Marrow Aspiration**

- The indications, guidelines, and techniques for bone marrow sampling are the same as those described for dogs and cats. (see Chapter 22)
- Preferred sites include the proximal femur and humerus. The iliac crest may be used for sample collection as well, but can be a difficult site to access.
- Sedation is required for bone marrow aspiration. Use a 20- or 22-gauge spinal needle with stylet for sample collection.

**Splenic Aspiration**

Fine-needle aspiration of the spleen has been performed successfully in ferrets and is a rapid means of evaluating splenic cytology. In ferrets, the only contraindication is suspected hemangiosarcoma of the spleen. Sedation is rarely necessary, but is recommended if the ferret persistently struggles.

**Technique**

1. Two assistants are recommended for restraint. Place the ferret in dorsal recumbency. One assistant should restrain the head and neck by scruffing the ferret with one hand. The other hand is used to restrain the forelimbs. A second assistant restrains the hind limbs by placing one hand around the pelvis.
2. Palpate the spleen and position it against the left lateral or ventral body wall.
3. Clip and prepare the site aseptically.
4. Insert a 25-gauge needle attached to a 3-ml syringe to the hub at a perpendicular angle to the skin and aspirate from the spleen.
5. When a small amount of bloody fluid is visualized in the needle hub, withdraw the needle and prepare slides routinely for cytology.

**Cystocentesis**

- Urine may be collected by cystocentesis using the same technique described for the cat.
- A 22- or 25-gauge needle on a 1- or 3-cc syringe may be used for sample collection.
- Anesthesia is recommended if the ferret is difficult to restrain.

**THERAPEUTIC TECHNIQUES**

**Intravenous Therapy**

- **Key Point** Sedation is often required for placement of a butterfly or indwelling intravenous (IV) catheter, or for small-volume IV therapy.
- For small-volume IV therapy (0.3–0.4 ml), use an insulin syringe. The cephalic or lateral saphenous veins are the preferred sites for injection. Sedation may not always be required for a single injection.
- A 25-gauge butterfly catheter may be used to administer larger, single-dose volumes into the cephalic vein.
- An indwelling catheter can be placed in the cephalic, lateral saphenous, or jugular vein. Sedation is usually required.
- Flush indwelling catheters with small volumes of heparinized saline solution to maintain catheter patency.
- Peripheral indwelling catheters can be placed rapidly and are useful in emergency situations and for surgery.

**Cephalic or Saphenous IV Catheter Placement**

**Technique**

1. Clip and prepare the site aseptically.
2. Tent the skin over the vein and make a small cut-down incision in the skin with a 22-gauge needle, taking care to avoid the vein.
3. Introduce a 1/4–1/2 inch indwelling catheter (20–24 gauge) through the cut-down incision and into the vein.
4. When catheterizing small or debilitated ferrets it is often helpful to attach a tuberculin syringe containing heparinized saline to the catheter hub after removing the stylet. The heparinized saline may then be flushed through the catheter, dilating the vein while the catheter is advanced. The person holding off the vein must release the vein if this technique is used.

5. Cap the catheter and tape it in place routinely.

**Jugular Catheter Placement**

**Technique**

1. Sedation is required for placement.

2. Place the anesthetized ferret in dorsal recumbency. Clip and prepare the site aseptically. Make a small cut-down incision over the jugular vein as described previously.

3. Introduce a 20-gauge or smaller catheter into the vein. Suture in place and place a tape bandage around the neck.

4. Ferrets often do not tolerate jugular catheters. Jugular catheters are not used very often unless venous access is required for treatment purposes.

**Intraosseous Catheter Placement**

- Sedation is required for placement.
- The femur is the preferred site for IO catheter placement.

**Technique**

1. Place the anesthetized ferret in lateral recumbency. Clip and prepare the site aseptically.

2. Wearing a sterile surgical glove, palpate the top of the greater trochanter, and locate the trochanteric fossa.

3. Introduce the tip of a 20- or 22-gauge spinal needle with stylet in place into the trochanteric fossa, and advance the needle parallel to the long axis of the femur through the cortical bone and into the medullary cavity. Flush with heparinized saline.

4. Suture into place.

**Subcutaneous and Intramuscular Injections**

- Restrain the ferret by scruffing. Some ferrets may require two people for restraint.
- SC injections may be given in the loose skin over the shoulders.
- IM injections may be given in the quadriceps, the semimembranosus-semitendinosus muscles of the hind limbs, or in the expaxial muscles of the lower back.
- Limit the volume of the IM injections, due to the small muscle mass of the ferret.

**Fluid Therapy**

- Fluids may be administered SC, IV, or IO, depending on the needs of the patient.
- The daily fluid requirement for ferrets has not been reported but can be estimated at 70 to 100 ml/kg/day. Adjust for dehydration and fluid loss.
- Administer SC fluids over the dorsal shoulder and thoracic region.
- IV or IO fluid therapy is used for a wide range of medical and surgical situations, and is recommended for ferrets that are >5% dehydrated.
- IV or IO fluids must be administered with an infusion pump. Fluids may be given as a continuous infusion, may be administered by continuous infusion, or may be given in 2 to 3 bolus doses over a 24-hour period.
- Ferrets may require the addition of dextrose to fluids because hypoglycemia is common.

**Oral Therapy**

- Oral medications are most easily given to ferrets in liquid form.
- If possible, compound medications formulated in tablet or capsule form into liquid suspensions, or crush and mix them with a sweet-tasting substance such as Nutri-Cal (Evsco Pharmaceuticals) feline hairball laxative, or fruit-flavored syrup, and administer by syringe.
- Ferrets suffering from insulinoma should not be given sugar-based treats or medications if at all possible. Hide medications in fatty acid supplements, vegetable oil, whipping cream, or meat baby food.

**Nutritional Support**

- Supplemental feeding is important in the management of anorectic or critically ill ferrets, and in the treatment and prevention of hypoglycemic episodes associated with insulinoma.
- Most ferrets can be force-fed dietary supplements by syringe. Once they acquire a taste for a given supplement, it may be possible to offer it in a bowl.
- Feed ferrets as much food as they will take comfortably (12–25 ml) 2 to 4 times daily.
- Foods useful for force-feeding include the following: meat baby foods, slurried cat or ferret food, and Science Diet A/D liquid soy-based formulas (e.g. Deliver 2.0, Mead Johnson Nutritional) may be added to the mixture to increase the calorie content and improve palatability.

**Drug-Dosing Guidelines**

- Use of all medications is considered off-label for the ferret; there are no approved drugs available for ferrets in the United States.
• Several exotic animal formularies are commercially available that include drug dosage information on ferrets.

• When dosing information is not available, use feline dosages with the following exceptions:
  • *Chloramphenicol*: (50 mg/kg) bid, IV, SC, IM, or PO.
  • *Aspirin*: (10–25 mg/kg) bid–tid PO (canine dosage).

• Many ferrets become lethargic when placed on enalapril (Enacard, Merck Agvet) for cardiac disease. Start with a very low dose (0.25–0.5 mg/kg) q48h PO. Some ferrets cannot tolerate more than every-other-day therapy.

• *Ivermectin* (0.06 mg/kg) q30d PO for heartworm prevention; (0.05 mg/kg) once 3 to 4 weeks after adulticide treatment as a heartworm microfilaricide; (0.50–1.0 mg/kg) PO, SC, repeat in 14 days for sarcoptic mites; (1 mg/kg) instill half the calculated dose into each ear and repeat in 14 days for ear mites.

### Blood Transfusions

A detailed discussion of the techniques used for blood collection for transfusion and blood transfusion is presented in the Hematopoietic System section in this chapter.

### Urinary Catheterization

• Sedation or isoflurane anesthesia facilitates urinary catheterization. The procedure may be difficult in ferrets with urethral disease or urethral calculi.

#### Males

1. Position in dorsal recumbency and prolapse the penis from the prepuce.
2. Identify the urethral opening, which lies ventral to the tip of the penis. It is helpful to place a 24-gauge IV catheter (with stylet removed) into the urethral opening as a guide.
3. Use a ferret urinary catheter (Slippery Sam ferret urinary catheter, Cook Veterinary Products) an open-ended tomcat catheter, or a #3.5-Fr. feeding catheter. A wire stylet may be useful.
4. Suture into place.

#### Females

1. Position in ventral recumbency with the hindquarters elevated and use a tomcat catheter or a 3.5-Fr. feeding tube with or without a stylet.
2. The urethral orifice is located on the ventral floor of the vaginal vault. Catheterize the urethra blindly or after identification using a vaginal speculum.

### Ferrets with urethral disease

• The catheter may only pass part way into the urethra (often to the pelvic flexure). This may be sufficient to allow retrograde flushing of urethral calculi into the bladder, or to empty the bladder of a ferret with urethral obstruction secondary to prostatic enlargement.

### Sedation

**Key Point** Isoflurane administered by face mask is the most convenient method to immobilize a ferret for procedures such as venipuncture and radiography. Induction and recovery are rapid.

Doses for parenteral agents used in ferrets are listed in Table 175-3.

• **Acepromazine** is useful for sedation.
• **Butorphanol tartrate** has been used at SC, IM, IV, but can cause very profound sedation in some ferrets.
• **Ketamine alone** does not produce effective muscle relaxation. Use in combination with acepromazine for minor surgical procedures, or with diazepam for more complicated procedures.
• **Medetomidine** is not analgesic; animals will respond to painful stimuli. Use with an analgesic agent. This agent is reversible (atapamazol).
• **Tiletamine-zolazepam** (Telazol, Fort Dodge) gives variable muscle relaxation but is useful for immobilization for procedures such as venipuncture, radiography, and electrocardiography. Recovery may be prolonged.
• **Xylazine** may cause bradycardia or vomiting, and is not recommended for use.

### Anesthesia and Analgesia

• Premedicate with parenteral agents followed by face-mask induction. Alternatively ferrets may be given

| **Table 175-3. DRUGS RECOMMENDED FOR CHEMICAL RESTRAINT AND ANALGESIA OF FERRETS** |
|---------------------------------|------------|----------|
| **Drug**                        | **Dosage (mg/kg)** | **Route**   |
| **Chemical Restraint**          |              |           |
| Acepromazine                    | 0.1–0.3      | IM, SC    |
| Ketamine                        | 25–35        | IM, SC    |
| plus acepromazine*              | 0.2–0.3      | IM        |
| Ketamine                        | 25–35        | IM        |
| plus diazepam                   | 2–3          | IM        |
| Ketamine                        | 10–25        | IM        |
| plus xylazine                   | 1–2          |           |
| **Analgesics**                  |              |           |
| Buprenorphene                   | 0.01–0.03 mg/kg q8–12h | SC, IM, IV |
| Butorphanol tartrate            | 0.05–0.5 mg/kg q8–12h | SC, IM    |
| Carprofen                       | 1 mg/kg q12–24h | PO        |
| Flunixin meglumine              | 0.5–2.0 mg/kg q12–24h | IM, IV |

*Use this combination for minor surgery.

IM, intramuscular; SC, subcutaneous.
inhalant agents without premedication via chamber
or face-mask induction.
• Isoflurane is the inhalant anesthetic most commonly
used in small mammal practice. Sevoflurane is used
in some practices as well. Pharmacokinetic and phar-
macologic isoflurane and sevoflurane are very
similar; sevoflurane smells better and is better toler-
ated during face-mask induction.
• Isoflurane does not provide analgesia. Administer an
analgesic pre- or intra-anesthesia if a painful proce-
dure is to be performed. Analgesic agents com-
monly used in ferrets include Buprenorphene
(0.01–0.03 mg/kg q8–12h SC, IM, IV), Butorphanol
(0.05–0.5 mg/kg q8–12h SC, IM), Carprofen (1
mg/kg q12–24 PO), and Flunixin meglumine
(0.5–2.0 mg/kg q12–24h IM, IV).
• Intubate ferrets to facilitate intermittent positive pres-
sure ventilation (IPPV). A 2.5 to 3.5 mm endotracheal
tube usually is suitable.
• The same planes of anesthetic depth reported for
dogs and cats occur in the ferret.
• Follow basic principles of anesthesia for small animals,
(see Chapter 2) and provide supplemental heat
during surgery; administer IV fluids (isotonic elec-
trolyte solutions supplemented with 2.5–5% dextrose)
during long procedures and for insulinoma surgery.

Infectious Diseases of the Ferret

VIRAL DISEASES

Canine distemper and influenza are the two most
common viral diseases of the ferret. Influenza is zoonotic
between humans and ferrets, and is typically passed from
humans to ferrets. Canine distemper is 100% fatal in the
ferret, making distemper vaccination imperative.

Canine Distemper

Etiology

• The canine distemper virus (CDV) is a paramyx-
ovirus. Transmission occurs through direct contact
with infected animals of any species, and through
contact with fomites such as in shoes or clothing.
• The incubation period for CDV in the ferret is typi-
cally 7 to 10 days; however, incubation for some
strains of CDV may take up to 21 days. (For discus-
sion of CDV in dogs, see Chapter 13).
• Intubate ferrets to facilitate intermittent positive pres-
sure ventilation (IPPV). A 2.5 to 3.5 mm endotracheal
tube usually is suitable.
• The same planes of anesthetic depth reported for
dogs and cats occur in the ferret.
• Follow basic principles of anesthesia for small animals,
(see Chapter 2) and provide supplemental heat
during surgery; administer IV fluids (isotonic elec-
trolyte solutions supplemented with 2.5–5% dextrose)
during long procedures and for insulinoma surgery.

Clinical Signs

• Early in the disease the only clinical sign may be a
mild unilateral or bilateral conjunctivitis.
• Pyrexia (>40°C), anorexia, and profuse mucopuru-
lent naso-ocular discharge develop as the disease
progresses.

Diagnosis

• Key Point Preliminary diagnosis of CDV may be
based primarily on the history, physical examina-
tion, and clinical signs, which are unlike those of
any other disease in the ferret.
• The history often reveals that the animal is unvacci-
nated, overdue for booster vaccination, or is improp-
erly vaccinated. There is often no evidence of direct
exposure to an infected animal because fomite trans-
mission can occur.
• Differential diagnoses early in the disease include
influenza and bacterial conjunctivitis. As the disease
progresses, development of the integumentary
lesions around the chin, lips, and footpads are
pathognomonic.
• A fluorescent antibody test can be performed on
blood smears, conjunctival scrapings, or mucous
membrane scrapings. This test is only useful in the
first days of disease onset; false-negative results are
common. Modified live vaccine strains of canine dis-
temper will not affect this test.
• Plasma samples can be submitted to measure anti-
body titer. A positive result is not always diagnostic of
disease because both infected and vaccinated ferrets
can have a positive titer. Unvaccinated ferrets with a
positive titer are likely infected with CDV.
• Fluorescent antibody staining may be performed on
imprints of the lymph nodes, bladder epithelium,
and cerebellum postmortem.
• Histopathologic diagnosis may be possible via identi-
fication of inclusion bodies in the cytoplasm or
nucleus of epithelial cells of the trachea, urinary
bladder, skin, gastrointestinal tract, lymph nodes,
spleen, and salivary glands.

Treatment

• Key Point Treatment for canine distemper is rarely
effective, and is limited to supportive care only.
Most affected animals must be euthanized.

• Disinfect the household thoroughly using 0.2%
Roccal (Upjohn), 0.75% phenol, or 2% to 5%
sodium hydroxide.
• In multiple animal households, remove all clinically
affected animals. Vaccinate the remaining animals
immediately.
**Prevention**

**Key Point** Preventing infection by vaccination is crucial in pet ferrets, since treatment is not effective.

- Vaccinate all ferrets in the household or facility. Currently only two vaccines are approved for use in ferrets: PureVax (Merial, Athens, GA), and Fervac-D (United Vaccines, Inc., Madison, WI). Give 1 ml SC, using the following schedule:
  - If the dam is vaccinated: Vaccinate kits at 8 weeks of age and repeat vaccination every 3 to 4 weeks until the kits are 16 weeks of age.
  - If the dam is unvaccinated: Vaccinate the kits at 6 weeks of age and repeat every 3 to 4 weeks until the kits are 16 weeks of age.
- Revaccinate annually. Sources claim that immunity lasts for 3 years; however, outbreaks have been known to occur 18 months after vaccination.
- Use of serum titers as a method to evaluate an animal’s current immunological status is unsubstantiated.
- Quarantine new ferrets and canines for 4 weeks before exposure to other resident animals. Vaccinate new animals immediately after acquisition at the beginning of the quarantine period.
- Use of Galaxy D (Schering-Plough Animal Health Co., Omaha, NE) for distemper vaccination has been described. Use of this product in ferrets is extra-label. This product has proved effective in preventing canine distemper in young ferrets; however, duration of immunity is unknown.
- Do not use CDV vaccines that contain canine parvovirus, adenovirus, or other viruses. It is not necessary to vaccinate for leptospirosis unless there is exposure to wild rodents.

**Influenza**

**Etiology**

The influenza virus is an orthomyxovirus. Ferrets are susceptible to influenza A and B; this is the only documented zoonotic disease of the ferret. Human-to-ferret transmission is more common than ferret-to-human transmission. Transmission occurs by direct contact with naso-ocular discharges, and via inhalation of aerosolized droplets.

- The incubation period is typically 2 to 10 days post-exposure.
- The clinical course of the disease is typically 7 to 14 days.

**Clinical Signs**

**Key Point** Influenza typically causes only mild illness and discomfort, and is usually self-limiting in an otherwise healthy animal.

- Clinical signs may include any combination of the following:
  - Sneezing with a clear, serous nasal discharge.
  - Mild conjunctivitis with serous ocular discharge. Crusting around the eyes may occur rarely.
  - A nonproductive cough that may be loud and paroxysmal, and often occurs more frequently at night.
  - Diarrhea. Vomiting may occur in rare cases.
  - Partial to total anorexia, listlessness, and fever.
  - Pneumonia, severe illness, or death may occur in neonates, geriatric patients, and in ferrets with concurrent diseases such as lymphosarcoma or insulinoma.
- Ferrets with underlying immunosuppressive disorders, especially lymphosarcoma, may develop repeated or cyclic episodes of influenza. Rule out lymphosarcoma by performing a complete blood cell count (CBC), bone marrow biopsy/cytology, or a peripheral lymph node biopsy (see Lymphoma in this chapter).

**Diagnosis**

Diagnosis is based primarily on the clinical signs, history, and physical examination.

- The history often indicates recent exposure to a human or another ferret with influenza or signs of upper respiratory tract disease.
- The overall physical condition often remains good, although slight or moderate dehydration may be present if the animal is not eating or drinking normal amounts.
- Differential diagnoses include the very early stages of canine distemper, GI rotavirus infection, and lymphosarcoma. If mucopurulent nasal or ocular discharge is noted, consider early CDV or a secondary bacterial infection.

**Treatment**

- Supportive care generally is sufficient.
- Encourage the ferret to eat and drink. Offer 1 to 3 tablespoonsfuls of Hill’s Science Diet A/D or strained meat baby food bid–qid if the animal refuses the regular diet.
- If indicated, give an oral electrolyte solution that is palatable to ferrets.
- If sneezing or coughing is excessive and interferes with eating or sleeping, give an antihistamine such as chlorpheniramine (1.0–2.0 mg/kg) bid–tid PO, or diphenhydramine (0.5–2.0 mg/kg) bid–tid PO. Nasal solutions containing phenylephrine may be used to relieve nasal congestion.
- Antibiotics are not necessary unless secondary bacterial infection is present.
- Antiviral medications such as amantadine (6mg/kg) bid PO (Symmetrel, ENDO Pharmaceuticals, Chadds...
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Ford, PA) may be useful in the treatment of ferrets with influenza. Zanamivir (12.5 mg/kg) once intra-nasally (Relenza, GlaxoSmithKline, Research Triangle Park, NC) has been shown experimentally to prevent influenza infection.

Prevention

Good hygiene is the key to prevention.

• Discuss the zoonotic potential with the client. Advise clients to wash their hands frequently, and to avoid holding the ferret near the face.
• In the veterinary hospital, do not allow influenza-infected personnel to handle ferrets, especially if the animal is a neonate, a geriatric patient, or a patient debilitated by serious disease.
• Vaccination is not recommended; only short-term immunity results, and the wide variation of the influenza virus makes appropriate vaccination difficult.

Rabies

Etiology

Rabies is caused by a rhabdovirus that results in fatal disease in ferrets. It is transmitted via contact with an infected animal’s saliva (see Chapter 15). This is a zoonotic disease; however, there has never been a report of ferret-to-human rabies transmission. Experimentally, the incubation period is 28 to 33 days.

Clinical Signs

• Behavioral abnormalities that range from anxiety and hyperactivity to lethargy.
• Neurologic signs such as ascending paralysis, ataxia, hyperparesthesia, and posterior paresis.

Diagnosis

Diagnosis is based on clinical signs, and/or a history of known or potential exposure to rabies.

• History may include a recent bite wound or exposure to a rabid animal.
• The ferret may be unvaccinated; however, development of rabies in vaccinated individuals has occurred in other animal species.
• Differential diagnoses include Aleutian disease, botulism, brain hypoxia from severe seizures, CNS neoplasia, insulinoma, intervertebral disc disease, and viral or bacterial encephalitis.
• Postmortem laboratory testing of brain tissue (fluorescent antibody staining [FAS] or virus isolation) confirms the diagnosis (see Chapter 15).

Treatment

• There is no treatment for rabies.
• Euthanize the suspect animal to protect humans and other animals in its environment. Submit animal for postmortem FAS testing of brain tissue.

• Key Point It has not been demonstrated that ferrets are carriers of rabies. Many public health facilities now recognize and accept the 10-day quarantine period for ferrets; however, in some states, unvaccinated ferrets involved in biting incidents will be euthanized and submitted for rabies testing. It is important to be familiar with state and local laws regarding vaccination requirements and the laws following a biting incident.

• Key Point The Compendium of Animal Rabies Prevention and Control recommends that ferrets be confined and observed for 10 days following human exposure. If signs compatible with rabies develop, the animal should be euthanized and protocols for rabies testing should be followed. Vaccinated ferrets exposed to a potentially rabid animal should be revaccinated and quarantined for 45 days. Euthanize any unvaccinated ferret exposed to a rabid animal.

Prevention

• Vaccination is the only prevention, and is mandatory in some states.
• Imrab 3 (Rhone Merieux) is an inactivated rabies vaccine that is currently the only rabies vaccine approved for use in ferrets. Administer at a dose of 1 ml SC.
• Vaccinate initially at 3 months of age. Revaccinate annually.

Aleutian Disease

Etiology

Aleutian disease (ADV) is caused by a parvovirus that affects both mink and ferrets. Transmission occurs by direct contact or via contact with fomites contaminated with any infected body fluid, including blood.

ADV produces a progressive immune-mediated disease accompanied by the deposition of antigen-antibody complexes in multiple organs of the body. The virus is prevalent in the ferret population, but the percentage of ferrets that develop clinical illness is low. In
one survey of 700 ferrets, 10% were serologically posi-
tive, but only two animals developed clinically active
disease. Some ferrets may be asymptomatic carriers,
while others may have natural immunity to the disease.

**Clinical Signs**

- **Key Point** The clinical signs of Aleutian disease are
extremely variable, and the incubation period can be
as short as 1 day or as long as 90 to 200 days.

- Ataxia, mild incoordination, posterior paresis, or
tremors may be the initial presenting signs. Initially
ferrets often continue to eat, and appear bright and
alert. As the disease progresses, paresis progresses to
the forelimbs, and wasting develops that may con-
tinue for weeks or months.

- Anorexia, lethargy, melena, and urinary inconti-
nence are seen in later stages of the disease.

- A slow wasting disease existing without neurologic
signs may also occur.

**Diagnosis**

- Diagnosis may be based on the history, clinical signs,
physical examination findings, the presence of high
serum total protein and hypergammaglobulinemia,
and a positive ADV test. Diagnosis is confirmed with
histopathology.

- Differential diagnoses include bacterial or viral
encephalitis, CNS neoplasia, canine distemper, lym-
phosarcoma, gastric foreign body, tuberculosis, inter-
vertebral disc disease, systemic mycoses, and rabies
(in cases with behavioral changes and sudden
paralysis).

- Exposure history is often not helpful because of the
prevalence of asymptomatic carriers.

- High serum total protein may be present. Serum
protein electrophoresis may demonstrate hypergam-
maglobulinemia (>20% of the total serum protein).

- Blood samples may be submitted for counterimmu-
noelectrophoresis testing (United Vaccines, Inc.,
Madison, WI) or enzyme-linked immunosorbent
assay (ELISA) testing (Avecon Diagnostics, Bath, PA).
An in-house saliva sample kit is available as well
(Avecon Diagnostics, Bath, PA).

- Histopathology demonstrates lymphocytic plasma-
cytic infiltration and perivascular cuffing in many
organ systems. The kidneys, liver, lymph nodes, and
spleen are often affected.

**Treatment**

- **Key Point** There is no effective treatment for Aleut-
ian disease. Provide supportive care and do not
allow contact between clinically ill animals and
healthy ferrets. Euthanasia is usually indicated
only for clinically affected animals.

- Asymptomatic ferrets that test FAS-positive for ADV
should not be euthanized because they may never
become clinically ill. Infected ferrets may remain
asymptomatic for life, but can remain persistently
infected. Other ferrets may develop nonpersistent,
self-limiting disease and fully recover.

- Administration of corticosteroid therapy and sup-
portive care may prolong the life of some ferrets with
clinically active disease.

**Prevention**

**Breeding colonies**

- Breeding colonies should be closed. New animals
should be ADV tested and quarantined prior to intro-
duction.

- Test all resident ferrets and remove serologically
positive animals from the population.

- ADV-negative animals should be retested in 6
months, before adding them to the colony, due to the
potentially long incubation period.

**Pets**

- It is not necessary to test a pet ferret unless it has been
exposed to a clinically ill animal.

- It is not necessary to euthanize a clinically normal,
non-breeding ADV-positive ferret or remove it from
contact with other pet ferrets. Advise the client,
however, that there is a slight possibility that the pet
may develop clinical illness.

- Do not house ferrets in close proximity to mink.

- Retest ADV-positive animals in 6 months since some
animals may eventually eliminate the virus and
become negative.

**Rotavirus**

Rotavirus causes gastrointestinal infection and a bright
green or yellowish-green diarrhea. Rotavirus is
described in the “Gastrointestinal System” section in
this chapter.

**Lymphosarcoma**

Lymphosarcoma is common in the ferret, and is dis-
cussed in the “Neoplasia” section in this chapter.

**BACTERIAL DISEASES**

**Common Bacterial Infections**

**Etiology**

*Staphylococcus*, *Streptococcus*, *Escherichia coli*, and other
common bacteria from the environment can be intro-
duced through penetrating wounds, punctures, abra-
sions, contact with mucous membranes, and by
inhalation or ingestion.
Abscessation is an uncommon form of bacterial infection in ferrets.

**Clinical Signs**

- An abscess may occur in any part of the body, including the anal glands, mammary tissue, mouth, mucous membranes, reproductive tract, respiratory tract, subcutis, and prostatic tissue.
- Body temperature may be ≤40°C if bacterial sepsis is present.
- Bacterial dermatitis causes thickened, irritated areas of skin. Affected ferrets may lick and chew these areas until they become denuded and ulcerated.
- Bacterial conjunctivitis causes a thick mucopurulent ocular discharge and swelling of the conjunctiva; corneal ulcerations may be present.
- Bacterial pneumonia causes lethargy, fever, anorexia, and dyspnea, and is often accompanied by mucopurulent nasal discharge and coughing.
- Bacterial mastitis occurs primarily in the lactating jill, and is accompanied by depression, fever, and anorexia. One or more mammary glands are swollen, discolored, and warm to the touch.
- Bacterial metritis may or may not cause a vaginal discharge; depression, fever, and partial or total anorexia are often present.
- Bacterial vaginitis causes a thick mucopurulent yellow-to-green vaginal discharge with little odor. Fever is usually absent, and the animal does not appear clinically ill.

**Diagnosis**

- Presumptive diagnosis is based on clinical signs, physical examination, demonstration of bacteria on routine cytology, results of bacterial culture, and sensitivity of the affected sites. The total WBC may demonstrate a marked leukocytosis (>10,000).

**Treatment**

- Treatment should consist of appropriate antibiotic therapy based on culture and sensitivity results, and surgical drainage or excision of the affected tissue when appropriate.
- Begin treatment with a broad spectrum antibiotic pending the results of culture and sensitivity testing, or when obtaining a culture is not feasible.
- Provide supportive treatment as needed, such as fluid therapy and nutritional support.

**Specific Treatment**

**Cutaneous Abscesses**

- Lance and thoroughly flush with an antiseptic solution. Keep the area open and flush twice daily until healing occurs by second intention.
- Administer oral antibiotics until signs of infection are gone and a healthy bed of granulation tissue is present.

**Mastitis**

See Mastitis in the Reproductive Disease section in this chapter.

**Uterine Infection (Metritis, Pyometria)**

See Uterine Infection in the Reproductive Disease section in this chapter.

**Respiratory Tract (Pneumonia)**

- If possible, perform a tracheal wash and submit samples for cytology, bacterial culture, and sensitivity testing.
- If pleural effusion is evident on radiography, perform thoracocentesis. Submit samples for cytology, bacterial culture, and sensitivity testing.
- Start oral broad-spectrum antibiotic therapy immediately, pending culture and sensitivity results. If pleural effusion is present, consider treating with a combination of clindamycin and cephalosporins (use cat dosages).
- Use of bronchodilating agents and/or nebulization therapy may be beneficial treatment modalities as well.

**Conjunctivitis**

- Treat conjunctivitis with a broad-spectrum ophthalmic ointment.
- Perform a fluorescent corneal staining test to rule out corneal ulcers. (see Chapter 133)

**Anal Gland Infection**

- Submit samples for bacterial culture and sensitivity testing.
- Begin treatment with broad-spectrum oral antibiotics. Modify treatment based on culture sensitivity results.
- Instruct owners to hot pack the affected area 5 to 10 minutes bid–tid.
- Treat until infection and swelling resolve, then perform anal sacculectomy.
- Continue antibiotic treatment 10 to 14 days postoperatively.

**Campylobacteriosis Infection**

- *Campylobacter* spp. typically causes GI disease (see "Gastrointestinal System").

**Salmonellosis**

- *Salmonella* spp. may rarely cause gastroenteritis in ferrets (see "Salmonella" within "Gastrointestinal System").
Botulism
- Botulism is a rarely encountered disease in the domestic ferret caused by the ingestion of food contaminated with the *Clostridium botulinum* toxin. *C. botulinum* is commonly found in the soil.
- Uncooked food or food contaminated with soil can be the source of the infection.

Tuberculosis
- Clinical cases of tuberculosis in the ferret are reported infrequently; however, ferrets are susceptible to bovine, avian, and human *Mycobacterium* spp. infections.
- The disease can be transmitted by ingestion of contaminated meat (poultry or meat), unpasteurized milk, or food contaminated by the droppings of infected wild or pet birds (see Chapter 19 for information about tuberculosis in dogs and cats).
- Clinical signs include chronic weight loss, and diarrhea that is unresponsive to treatment. Vomiting may occur as well in some cases.
- Diagnosis is based on history, clinical signs, and the exclusion of other diseases; it is confirmed by intestinal biopsy. Histopathologically granulomatous inflammation and acid-fast bacteria are identified. Infection may also be confirmed by culturing the organism, and with polymerase chain reaction (PCR) testing.
- Because of the zoonotic potential of this disease, treatment is not recommended. Affected animals should be euthanized.

Fungal Infections

Dermatophytosis

**Etiology**
Dermatophytosis is rare in the ferret and is typically caused by *Microsporum canis* and *Trichophyton mentagrophytes*. Dermatophytes are transmitted by direct contact with infected animals or contaminated bedding, caging, and fomites.

▼ **Key Point** Ferrets are usually not carriers of these organisms. Clinical disease is typically self-limiting. The most common source of infection of the pet ferret is the household cat.

**Clinical Signs**
- Young, debilitated, or geriatric ferrets are the most commonly affected.
- Lesions are consistent with those described in other species (see Chapter 42). Alopecia with erythema, inflammation, hyperkeratosis, superficial crusting, lichenification, and erythema are present. Pruritis is common and may lead to self-trauma and secondary pyoderma.

**Diagnosis**
- Diagnosis is based on the identification of the fungal agent on skin scrapings, fungal culture, or a positive Wood’s light examination (*M. canis*). (see Chapter 42).

**Treatment**
- Dermatophytosis is often self-limiting and may resolve without therapy. However, due to the zoonotic potential, treatment is recommended.
- Topical treatment includes the use of keratolytic shampoos, and/or lime sulfur dips. (see Chapter 42).
- Oral therapy consists of the administration of griseofulvin (25 mg/kg) PO sid for 21 to 30 days. Perform a CBC every 14 days while the ferret is receiving treatment.
- Disinfect the home by steam cleaning, the application of dilute (1:10) bleach or chlorhexidine solutions, and vacuuming thoroughly to remove infectious spores. Dispose of the vacuum cleaner bag after vacuuming is complete. A thorough cleaning of the heat ducts and air conditioner/heater filters is also recommended.

Other Fungal Infections
- Systemic mycoses are rare in the ferret; however blastomycosis, histoplasmosis, cryptococcosis, coccidioidomycosis, and aspergillosis have been reported.
- Consider these infections in the differential diagnosis of any systemic disease that is refractory to treatment and involves wasting, granulomatous lesions, persistent or recurring draining wound tracts, and respiratory tract disease.
- Diagnosis is based on the histopathological or cytological demonstration of the fungal organism in biopsies or aspirates (see Chapter 20).
- Complement fixation and precipitation tests have been used with variable success.
- Treatment is the same as described for the dog and cat (see Chapter 20).

Hematopoietic System

**Splenomegaly**
Primary disease of the spleen is uncommon. Splenomegaly is often a common incidental finding in a healthy adult ferret, or it may occur in association with
a wide variety of disease conditions. Perform a complete medical evaluation in all splenomegaly cases.

Etiology

- Splenomegaly may be a normal or incidental finding in some patients.
- Pathological causes of splenomegaly may include chronic immune stimulation, erythroid bone marrow insufficiency, extramedullary hematopoiesis (EMH), hypersplenism, heart disease, and neoplasia.
- Splenomegaly can occur concurrently with adrenal gland disease and insulinoma, but it is usually an incidental finding.
- Extramedullary hematopoiesis (EMH) may cause enlargement of the spleen. The etiology of EMH is unclear; compensation for myeloid insufficiency and chronic immune stimulation have been suggested as causes. Ferrets with EMH typically do not show evidence of hematological abnormalities. Grossly the spleen has a normal shape and color, but it appears enlarged.
- Hypersplenism may cause enlargement of the spleen, but is rare in the ferret.
  - Destruction of one or more blood cell lines by the splenic reticuloendothelial system occurs; affected ferrets will have anemia, leukopenia, thrombocytopenia, or pancytopenia.
- Bone marrow may be normal or hyperplastic in affected patients.
- Lymphoma is the most common neoplasia of the ferret spleen. Hemangioma or hemangiosarcoma may occur as well.
  - When splenic lymphoma is present the spleen typically has irregular borders and a nodular texture. White or tan nodules may be noted grossly on the surface of the spleen and in the parenchyma. Metastasis may be present.
- Splenic torsion and abscessation are rare in the ferret.

Clinical Signs

- The normal ferret spleen measures approximately 5 cm × 2 cm × 1 cm, and may be palpated in the left cranial abdominal quadrant. The texture of the spleen should be slightly firm, smooth, and the edges should be sharp.
- An enlarged spleen is often noted on abdominal palpation as a firm, elongated smooth mass extending down to the left side of the ferret abdomen, or crossing diagonally across the ventral abdomen from the left cranial abdominal quadrant to the caudal right abdominal quadrant.
- Abdominal distention may occur.
- Occasionally the spleen is so large and pendulous that the ferret can barely lift its abdomen off the ground.
- Abdominal discomfort due to splenomegaly appears to be uncommon in ferrets.

Diagnosis

- Perform a CBC, platelet count, serum biochemical analysis, bone marrow cytology, and whole body radiography.
- Diagnosis of hypersplenism is based on the presence of one or more cytopenias, normal to hypercellular bone marrow cytology/biopsy, and the absence of blood loss, infection, or neoplasia.
- Obtain whole body radiographs to delineate the borders of the spleen and to rule out other abnormalities such as cardiomegaly or hepatomegaly that may contribute to splenomegaly.
- Splenic aspiration or biopsy may be performed. Perform fine-needle aspiration of the spleen using a 22-gauge needle (see “Clinical Techniques”). Do not perform splenic aspiration if hemangiosarcoma is suspected.
- Perform an abdominal ultrasound to evaluate the spleen. When the splenic parenchyma appears irregular, an ultrasound-guided biopsy or fine-needle aspiration may be performed.
- Perform a splenic biopsy during abdominal exploratory surgery, particularly if the spleen is irregular or discolored.

Treatment

Treatment depends on the primary disease condition. Usually splenectomy is not necessary.

\[\text{Key Point}\] Indications for splenectomy are the same as for other species and include hypersplenism, splenitis, splenic abscess, torsion, rupture, neoplasia, and discomfort caused by excessive splenomegaly.

- To perform a splenectomy, follow the surgical guidelines for splenectomy in dogs and cats (see Chapter 25).
- Anemia may result after splenectomy; the decision to perform splenectomy should be made cautiously, and with consideration to the health of the ferret as a whole.
- Administer antibiotics and fluid therapy pre- and postoperatively.
- Monitor asymptomatic ferrets with splenomegaly with periodic physical examination, CBC evaluation, imaging, and splenic aspiration.

\[\text{ANEMIA}\]

The clinical approach to anemia in ferrets is the same as for other species. Anemias are classified as regenera-
tive or nonregenerative; treatment is directed at the specific cause.

**Etiology**

There are many causes of anemia in ferrets; decreased erythropoiesis, destruction of red blood cells, and blood loss contribute to anemia.

- **Nonregenerative anemia** (normocytic, normochromic, nonregenerative anemia) occurs when bone marrow hematopoiesis is disrupted. Bone marrow cytology of affected ferrets may appear normal.
- Decreased erythropoiesis may be caused by chronic metabolic disease (renal, hepatic), chronic inflammation, hyperestrogenism, bone marrow suppression, and neoplasia.
- Anemia of chronic disease can occur whenever long-term illness is present and is caused by decreased erythrocyte survival, decreased availability of iron, or a decreased response to the anemia treatment.
- Anemia associated with chronic inflammation is mediated by sustained inflammatory cytokine release.
- Hyperestrogenism may cause nonregenerative anemia due to estrogen-induced bone marrow suppression. Unspayed female ferrets, female ferrets with ovarian remnants, or hyperestrogenism associated with chronic adrenal disease may contribute to this syndrome.
- Myeloid and leukemic neoplasias can cause suppression of bone marrow erythropoiesis due to replacement of normal bone marrow by neoplastic or fibrotic changes.
- **Erythrocyte destruction** may cause anemia; causes include immune-mediated disease, toxins, parasitism, or septicemia.
- Idiopathic immune-mediated hemolytic anemia, and immune-mediated hemolysis secondary to viral disease or blood parasites have not been reported in the ferret.
- Drug-induced hemolysis and heavy metal toxicosis (including zinc) are potential causes of hemolytic anemia.
- **Anemia secondary to blood loss** may be secondary to trauma, hemostatic disorders, bleeding lesions, and parasitism.
- Bleeding lesions may be internal or external.
- Bleeding ulcers may lead to anemia, and may be associated with *H. mustelae* gastritis, gastrointestinal foreign body, or chronic use of ulcerogenic drugs.
- Parasitism is uncommon in the ferret. Coccidiosis in the young ferret or severe flea infestation may cause anemia.
- Hemostatic disorders include thrombocytopenia associated with estrogen toxicity, rodenticide poisoning, and liver disease.

**Clinical Signs**

- Clinical signs include weakness, pallor, lethargy, and inappetence. Jaundice may be seen if hemolysis is present.
- A soft systolic murmur is common in anemic ferrets.
- A swollen vulva is present in ferrets with persistent estrus, an ovarian remnant, and in some cases of adrenal gland disease. Hair loss may also be present on the shoulders and flanks.
- Ferrets with estrogen toxicity may have signs of thrombocytopenia such as petechiae, ecchymoses, and melena.
- Melena may be noted if GI bleeding is present.
- Palpate the spleen. Splenomegaly may be caused by hypersplenism and subsequent anemia.
- Check carefully for fleas. Perform a fecal examination.

**Diagnosis**

- Diagnosis is based on the medical history, the physical examination findings, and a complete diagnostic work-up that includes a CBC, reticulocyte count, serum biochemical analysis, whole-body radiographs, and bone marrow cytology if indicated.

**History**

- Obtain a careful history regarding possible blood loss, toxicity, and foreign body ingestion. Determine the duration of vulvar swelling (if present).

**Laboratory Tests**

- Characterize the anemia based on RBC parameters and hemoglobin concentration.
- The normal hematocrit for the ferret is 46% to 61%, higher than that of other animals. The erythrocyte count is higher as well; erythrocyte counts as high as 17.0 × 10^6 cells/µl have been reported.
- The normal reticulocyte count may be as high as 10%. Reticulocyte counts greater than 12% are indicative of a regenerative bone marrow response.
- Regenerative anemia is often the result of blood loss or hemolysis.
- Perform bone marrow aspiration (see Clinical Techniques), particularly if the anemia is nonregenerative, to identify infiltrative processes and assess the morphology of RBC precursors. Bone marrow cytology from animals affected with anemia of chronic disease may be normal. Bone marrow cytology is also indicated in ferrets with nonregenerative anemia that is unresponsive to treatment after 6 to 10 days.
- Obtain blood for blood lead concentration if lead poisoning is suspected.
Diagnostic Imaging

- Whole-body radiographs are indicated to rule out abdominal neoplasia, GI foreign body, and thoracic neoplasia. A GI contrast study may be helpful to rule out the presence of GI ulcers. Ultrasound may be helpful based on the rules out established.

Key Point

Anemia in an intact female ferret with a swollen vulva for more than 4 weeks most likely is due to estrogen toxicity.

Treatment

The objectives of treatment are to treat both the anemia and the underlying cause.

- General supportive care includes oxygen therapy, subcutaneous fluids, and nutritional supplementation.

Treatment of the Anemia

- Specific supportive care includes whole blood transfusion, iron dextran therapy, and the administration of erythropoietin.
- Oral iron supplements may be administered to replenish whole-body iron stores.
- Erythropoietin may be used to treat ferrets with nonregenerative anemia. Administer 100 U/kg three times per week until the PCV is stable, then administer 1 to 2 times a week. Continue to monitor the PCV, and titrate the dose as needed.

Blood Transfusion

- Indications for blood transfusion include the clinical status of the patient, a low packed-cell volume (PCV) of <12%, the specific cause of the anemia, and the potential for continued blood loss.
- Ferrets lack specific blood types; transfusion reactions are rare in the ferret; up to three transfusions from the same donor and transfusions from multiple donors are considered safe.
- The normal blood volume of an adult ferret may be calculated as 8% of the body weight.
- The ideal value of the PCV post-transfusion would be within the normal reference range; a more likely goal is 5% to 10% higher than the pre-transfusion PCV. For dosage guidelines, see Chapter 22.
- Before transfusion, administer a rapid-acting corticosteroid, such as dexamethasone sodium phosphate (6-8mg/kg once IV) or prednisolone sodium succinate (22mg/kg once IV), as a slow bolus infusion, and administer an antihistamine such as diphenhydramine (0.5-1mg/kg IV, IM, SC) to the recipient ferret.

Collection of Blood for Transfusion

- The normal blood volume of ferrets has not been reported, but is estimated to be 5% to 6% of the total body weight (approximately 60ml/kg). Twenty percent of the estimated blood volume (approximately 12ml) may be collected from healthy ferrets.
- The jugular vein is the preferred site for the collection of large volumes of blood for transfusion.
- Sedate the donor ferret and place it in dorsal recumbency.
- Use a butterfly catheter to collect the blood into a syringe containing an anticoagulant such as sodium citrate (0.1ml citrate per 0.9ml blood) or acid-citrate-dextran (ACD) (1ml per 6ml blood collected).
- Transfer the blood immediately to the recipient. Blood should be filtered as it is transfused to the recipient.
- Administer fresh blood transfusions through an indwelling catheter or via a butterfly catheter into the cephalic or jugular vein. If a vein is inaccessible, administer into the peritoneal cavity or via the intraosseous route into the proximal femur.
- Whole blood is commercially available from Marshall Farms (Marshall Pet Products Inc., Wolcott, NY).
- Hemoglobin substitutes such as Oxyglobin (Bioprure Corporation, Cambridge, MA) (11-15ml/kg) IV or IO over 4 hours may be used if whole blood is unavailable.
- Administer Oxyglobin slowly in normovolemic patients and in patients with renal disease, heart disease, or when the risk of pulmonary edema is present.

Treatment of the Cause of Anemia

- Stop bleeding (internal or external).
- Correct the underlying cause of GI bleeding, including medical therapy for GI ulceration (see "Gastrointestinal System"), surgery to remove GI foreign body, and antibiotics and supportive care for enteritis/colitis.
- For anemia of chronic disease, treat the underlying primary disease process.
- Address metabolic disease (renal, hepatic) if present.
- To correct estrogen toxicity in the intact female, terminate estrus (see below) and provide supportive care until the bone marrow is functional. Broad-spectrum antibiotic therapy is important for the control of sepsis in leukopenic patients. Estrogen toxicity associated with ovarian remnants is treated by surgical removal of the remnants when the ferret is stable enough for surgery. Estrogen toxicity associated with adrenal gland disease is treated by adrenalectomy. Preoperative care is the same as that described for the intact female (see below). (For diagnosis and treatment of adrenal tumors see “Adrenal Gland Disease.”)
- Treat fleas with any product that is safe for use in cats (see Chapter 45).
- Treat lead poisoning following the same protocols recommended for cats (see Chapter 22).
Anemia secondary to neoplasia is associated with a poor prognosis. Some cases of lymphoma may respond to treatment (see “Neoplasia”).

Termination of Estrus

Administer human chorionic gonadotropin (HCG) in a single injection of 100 IU (or 1000 USP) IM. Repeat this dose in 1 to 2 weeks if vulvar swelling has not diminished.

Alternatively, give gonadotropin-releasing hormone (GnRH) at a dose of 20 mg IM or SC; repeat in 2 weeks if necessary.

GnRH and HCG are effective only after the 10th day of estrus. Bone marrow toxicity is not immediately reversible with termination of estrus; the PCV continues to fall for days to weeks.

Monitor the PCV as a useful guide to therapy and prognosis:
- PCV > 25%—the prognosis is good and termination of estrus is the only therapy required.
- PCV 15% to 25%—the prognosis is guarded because the PCV level can decrease further after termination of estrus.
- PCV < 15%—the prognosis is poor and aggressive supportive care is indicated, including multiple blood transfusions until bone marrow function is restored.

Prevention

Some causes of anemia in ferrets can be prevented. Educate owners about proper husbandry techniques to avoid flea infestation, foreign body ingestion, and trauma.

Key Point To prevent estrogen toxicity, spay all female ferrets not used for breeding.

Neoplasia

INSULINOMA

Insulinoma (pancreatic beta-cell tumor) is one of the most common neoplasias of the ferret. Disease is most common in ferrets over 2 years of age, and results in progressive, cyclic, or persistent hypoglycemia.

Etiology

- The incidence of insulinoma is typically higher in ferrets in the United States than in ferrets in other countries. The cause(s) are unknown.
- Possible etiologies include a limited genetic pool and diet. Ferrets in the United States are typically fed processed foods containing large amounts of cereal grains. Ferrets in other countries are typically fed a more natural diet consisting of meats and whole prey items.

Clinical Signs

- Early signs may be subtle and transient. Cyclic or progressive episodes of profuse hypersalivation and pawing at the mouth (which is indicative of nausea), lethargy, depression, “stargazing,” and posterior paresis may be seen during periods of hypoglycemia.
- As the disease progresses, or during periods of inadequate food intake, symptoms become more pronounced and may progress to stupor or coma. Seizures may occur.
- Splenomegaly is a common, unassociated finding on physical examination (see “Splenomegaly” under “Hematopoietic System” in this chapter).
- Adrenal disease is often identified concurrently in many ferrets with insulinoma (see “Adrenal Neoplasia” in this chapter).

Diagnosis

Key Point Base a presumptive diagnosis of insulinoma on the history, clinical signs, and repeated evidence of hypoglycemia in the presence of normal or elevated blood insulin levels. Make a definitive diagnosis via surgical removal and histopathology of a pancreatic tumor or biopsy.

Fasting Serum Glucose

- A carefully monitored fast of 4 to 6 hours is sufficient.
- If necessary, obtain several samples over a period of several days.
- Normal fasting serum glucose concentration is 90 to 110 mg/dl. Ferrets with insulinoma often have a fasting serum glucose of 20 to 75 mg/dl. Ferrets with fasting serum glucose between 75 to 90 mg/dl are considered suspect and should be monitored.
- Do not fast the animal for more than 6 hours. Discontinue fast if signs of hypoglycemia occur. Prolonged fasting may lead to collapse, coma, or seizures. Feed the ferret a high-protein and high-fat meal as soon as possible after collection of blood.

Blood Insulin Values

- Measurement of blood insulin concentration is not consistently reliable in the ferret.
- False positive results may occur if liver disease, non-islet cell tumors, or sepsis are present.
- Blood insulin concentrations greater than 275 pmol/liter (or 38 μU/ml) are considered elevated in ferrets. However, if the ferret is severely hypoglycemic at the time of sample collection, the blood insulin
value may be normal because insulin and glucose are in a constant dynamic state.

**Serum Biochemical Profile and Complete Blood Cell Count**
- The serum biochemical profile is typically normal except for the presence of hypoglycemia.
- The CBC is typically normal.
- A slight elevation in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) may be noted. The cause is unknown and may incidental, or may indicate hepatic lipidosis due to chronic hypoglycemia or some hepatic pathology.

**Treatment**

**Key Point** Insulinoma is a progressive disease in ferrets. Educate owners how to recognize the signs of hypoglycemia and how to manage hypoglycemia at home (see “Medical Therapy” and “Hypoglycemia Episodes”).

- Treatment options include medical therapy and/or surgical therapy.
- Medical therapy will need to be adjusted as the disease progresses.
- Surgery is used as a management tool and is not curative.

**Medical Therapy**
Insulinoma is a progressive disease, even after surgical intervention. Medical therapy is often effective in controlling symptoms associated with insulinoma for 6 to 18 months. Frequent feeding is the first step in treatment. Add prednisone and diazoxide as clinical signs and hypoglycemia worsens.

- Feed high-quality protein and high-fat meals frequently, especially after exercise or a long sleep. Avoid foods containing sugar or excessive carbohydrates (except to treat hypoglycemic episodes); these foods cause short term hyperglycemia followed by a period of hypoglycemia 1 to 2 hours later.
- 1 to 3 tablespoons of Hill’s Science Diet A/D or meat-based baby foods may be given twice daily and as needed.
- Chromium has been anecdotally reported to stabilize blood glucose and insulin levels in humans. Brewer’s yeast, which is a rich source of chromium, has been beneficial in some ferrets with insulinoma. Give 1/8 to 1/4 tsp of Brewer’s yeast q12h in the high-quality protein, high-fat meal.
- When frequent feedings no longer control clinical signs, begin corticosteroid therapy. Administer a prednisone or prednisolone suspension at a starting dosage of 0.1 to 0.25 mg/kg bid PO. As clinical signs worsen, increase the dosage gradually as needed to control signs. When the dosage approaches (0.75–1.0 mg/kg) bid PO consider the addition of diazoxide.
- Corticosteroids promote hepatic gluconeogenesis and antagonize the effects of insulin at the cellular level. Ferrets appear to tolerate corticosteroids well; side effects are minimal at lower dosages. Corticosteroids are usually the first treatment of choice because they are inexpensive and cause few side effects in the ferret.
- Add diazoxide (Proglycem, Baker Norton Pharmaceuticals) (10 mg/kg/day) PO divided bid–tid when frequent feedings and corticosteroids no longer control clinical signs. The diazoxide dosage may be gradually increased up to 60 mg/kg/day PO divided bid–tid as needed. Diazoxide is expensive, and is not palatable for some ferrets. Potential side effects at high doses include hypertension, lethargy, depression, anorexia, and nausea.

**Treatment of Acute Hypoglycemic Episodes**
- Hypoglycemic episodes require specific therapy.
- Mild to moderately severe hypoglycemic episodes often can be treated successfully at home. Instruct the client to give the ferret honey or corn syrup.
- Owners should be taught how to identify hypoglycemic episodes and how to administer medications with a syringe.
- If the ferret has collapsed, rub honey or corn syrup on the gingiva (taking care not to be bitten).
- Once the animal is stabilized, owners should feed a high-protein meal, and make arrangements to bring the ferret in for a blood glucose test.
- Severe hypoglycemic episodes that do not respond to home therapy or that result in seizures require treatment in a veterinary hospital (see Chapter 35).
- Administer a slow IV bolus of 50% dextrose (0.5–2 ml) until the ferret responds. Give no more than necessary to stop signs of hypoglycemia. If the ferret continues to have seizures or remains comatose, place an IV catheter and administer fluids containing 5% dextrose. Also administer a corticosteroid.
- Anticonvulsant therapy (diazepam 1–2 mg IV to effect) may be necessary if the ferret is in status epilepticus. Follow anticonvulsant protocols for dogs and cats (see Chapter 127) while correcting the hypoglycemia.
- Modify the medical treatment as necessary, and consider surgical intervention.

**Surgical Therapy**
Surgical therapy is the treatment of choice even though surgical removal or debulking of pancreatic tumors or partial pancreatectomy is palliative and provides only temporary remission of signs (6 to 24 months).
Follow canine preoperative protocols (see Chapter 35). Fast the ferret for only 4 to 6 hours preoperatively to avoid hypoglycemia.

- Administer IV or IO isotonic fluids containing dextrose (2.5–5.0%) 1 to 2 hours preoperatively if possible, during surgery, and continue postoperatively until the ferret is stable and is eating and drinking.

- Evaluate the blood glucose concentration pre-, intra-, and postoperatively if possible.

- See Chapter 35 for information about the surgical removal of insulinoma.

**Key Point** Perform a complete abdominal exploratory; insulinomas can metastasize to the regional lymph nodes, liver, and spleen (uncommon). Concurrent adrenal tumors (see Adrenal Gland Disease) are common.

- If the spleen is enlarged and appears irregular or mottled, consider performing a complete or partial splenectomy and submit for histopathology.

**Postoperative Care and Complications**

- Postoperatively monitor the blood glucose concentration bid–tid until the ferret is discharged from the hospital. Many ferrets become euglycemic immediately after surgery. Some ferrets may remain hypoglycemic. Rarely, ferrets may become transiently hyperglycemic after surgery.

- Most ferrets will require resumption of medical therapy 2 to 6 months after surgery. Some ferrets will need medical therapy immediately postoperatively.

- Monitor blood glucose levels for 10 to 14 days after surgery and at 60 to 90 day intervals.

- *Iatrogenic pancreatitis* is rarely a problem in ferrets; however, as a precaution, withhold food and water for 12 hours postoperatively; give 2.5% to 5.0% dextrose IV during this period. Monitor blood glucose 1 to 4 times daily.

- *Transient diabetes mellitus* may occur postoperatively. Hyperglycemia and glucosuria may be present for 1 to 21 days postoperatively; generally no treatment is required.

- Histopathologic examination of the pancreatic mass may reveal hyperplasia, adenoma, and/or adenocarcinoma of the pancreatic beta cells, even within a single tissue specimen.

**Prognosis**

- The prognosis is guarded, but with surgery and medical treatment, ferrets have had a good quality of life for more than 1 year after diagnosis of insulinoma. The median survival time was 17 months (range, 0.5–40 months) in one study of 53 ferrets treated with surgery, medical management, or both.

**ADRENAL GLAND DISEASE/ADRENAL NEOPLASIA**

Adrenal tumors are common in ferrets, and occur with approximately the same frequency as insulinomas. Adrenal neoplasia and insulinomas often appear concurrently. Adrenal tumors have been identified in ferrets as young as 1 year of age, although they typically occur in ferrets >2 years of age.

- Adrenal gland disease in ferrets is not Cushing’s disease. Excessive sex steroids, not corticosteroids, are produced by a hyperplastic or neoplastic adrenal gland.

**Etiology**

- The etiology is unknown. Possible causes of adrenal disease in ferrets include early neutering, genetic factors, and lack of exposure to normal seasonal photoperiods. The incidence of adrenal neoplasia is higher in ferrets in the United States. In the U.S., ferrets are typically neutered at 6 weeks of age, and are housed indoors under artificial light cycles. Ferrets in Europe and Australia are typically housed outside, and are not neutered until 6 months of age.

- Adrenal neoplasia in ferrets causes a variety of clinical signs, and appears to be the result of excessive secretion of estrogens and androgens, not cortisol. Pituitary-dependent hyperadrenocorticism has not been documented in ferrets.

- Adrenal tumors most commonly arise from the adrenocortical tissue. Common histopathological findings include hyperplasia, adenoma, and adenocarcinoma.

**Clinical Signs**

- Signs include progressive, bilaterally symmetric alopecia, usually starting at the tail base and progressing cranially. Hair loss often starts in the early spring or fall. There may be a history of alopecia and spontaneous hair regrowth as well.

- Pruritis often is reported, along with excessive dryness of the skin and small excoriations. Thinning of the skin is common.

- An enlarged vulva, mimicking estrus, may be the only clinical sign in spayed females. Muco-purulent vulvar discharge may be noted. Castrated males may exhibit territorial marking and sexual behaviors, and may develop the strong body odor and oily hair coats of intact males. Mammary hyperplasia can occur in either sex.

- Male ferrets may present with partial to complete urinary obstruction. Persistent elevation of adrenal-derived androgenic hormones may cause development of prostatic hypertrophy, prostatic cysts, or periurethral cysts, which cause narrowing of the
urethra. Affected ferrets may present with stranguria, dysuria, azotemia, and severe metabolic derangement. Male ferrets that are described as straining to urinate should be treated as an emergency (see “Urinary System”).

- Atrophy of abdominal musculature and mobilization of fat to the ventral abdomen, leading to a pendulous appearance, may be seen.
- Atrophy of hind limb musculature and rear limb paresis can occur.
- Polyuria/polydipsia is uncommon but has been reported.
- Collapse, anemia, and petechiation resembling estrogen toxicity have been described in male and female ferrets with chronic or advanced adrenal disease (see Hematopoietic System).
- Enlarged adrenal glands may occasionally be noted on the physical examination. The left adrenal gland is easier to palpate than the right.
- Radiographs are not typically helpful in confirming this disease. Ultrasonography may be useful for identification of adrenalmegaly.
- CBC is typically unremarkable unless estrogen toxicity-like anemia is present. The serum chemistry profile is typically within normal limits unless insulinoma is present.

**Diagnosis**

- Female ferrets often present with a swollen vulva. Differential diagnoses include an intact female ferret, a female ferret with an ovarian remnant, and seasonal alopecia. Perform a serum steroid panel or administer human chorionic gonadotropin (100IU) IM to determine if a female ferret is unspayed or has an ovarian remnant.
- A plasma steroid hormone assay may be used to support the diagnosis. Elevated plasma concentration of estradiol, androstenedione, and/or 17-hydroxyprogesterone is a reliable indicator of adrenal gland disease (see Table 174-4). A hormone panel is commercially available through the Clinical Endocrinology Laboratory of the Department of Comparative Medicine at the University of Tennessee.
- The adrenocorticotropic hormone (ACTH) stimulation test and the low-dose dexamethasone suppression test are not useful in ferrets. Ferrets with adrenal gland disease do not produce abnormal concentrations of cortisol, and adrenal gland disease in the ferret appears to be independent of ACTH. Urine cortisol/creatinine ratio does not appear to be a specific indicator of adrenal gland disease.

### Table 175-4. STEROID HORMONE CONCENTRATIONS IN FERRETS

| Steroid                        | Range   |
|--------------------------------|---------|
| Androstenedione (nmol/L)       | 0–15    |
| Estradiol (pmol/L)             | 30–180  |
| 17-hydroxyprogesterone (nmol/L)| 0–0.8   |

From Clinical Endocrinology Laboratory, University of Tennessee College of Veterinary Medicine.

- Perform exploratory surgery to confirm the diagnosis.

**Treatment**

- Adrenal tumors can be managed medically or surgically. Surgical management is preferred and recommended.
- Medical treatment may cause clinical signs to regress, but will not stop growth of the adrenal tumor.

**Medical Therapy**

The goal of medical treatment is to decrease or eliminate the clinical signs of adrenal gland disease. Medical therapy will not stop or prevent the growth of an existing tumor, and should be reserved for ferrets that are poor surgical candidates, ferrets with inoperable bilateral adrenal tumors, or ferrets with recurrent adrenal gland disease.

- Medical treatments described in the literature include mitotane, ketoconazole, androgen receptor blockers, aromatase inhibitors, and gonadotropin-releasing hormone analogs.

**Gonadotropin-releasing hormone analogs.** There are two general types of GnRH analogs: GnRH agonists and GnRH antagonists. To date only GnRH agonists such as leuprolide acetate (Lupron Depot, TAP Pharmaceuticals Inc., Lake Forest, IL) have been used to control the signs of adrenal disease in the ferret. Of the medical treatments described, anecdotal reports suggest that leuprolide acetate has been most effective in alleviating dermal and urogenital signs of adrenal disease. Administer the 1 month depot formulation of leuprolide acetate at a dose of (250 µg/kg) IM every 30 days.

**Androgen receptor blockers** theoretically block the actions of androgens at the receptor site, and decrease or reverse the signs of adrenal gland disease. In human medicine these drugs are used to treat men with prostatic carcinoma or prostatic hyperplasia. FluTamide (Eulexin, Schering Corporation, Kenilworth, NJ) and bicalutamide (Casodex, AstraZeneca Pharmaceuticals LP, Wilmington, DE) have been used, primarily in male ferrets. Results are variable.
- **Aromatase inhibitors** such as anastrozole (Arimidex, AstraZeneca Pharmaceuticals LP) inhibit aromatase, an enzyme involved in estrogen production. Some ferrets show decreased evidence of adrenal gland disease symptoms when treated with this drug.

- **Mitotane** (0,p'-DDD) (Lysodren, Bristol-Myers Squibb Oncology, Princeton, NJ) is rarely effective in ferrets with adrenal gland disease, presumably because ferrets do not develop pituitary-dependent hyperadrenocorticism. If clinical signs do resolve, they will often recur as soon as the mitotane therapy is withdrawn.

**Key Point** Perform a fasting blood glucose test before starting mitotane therapy. Do not use mitotane if blood glucose is low (indicative of concomitant insulinoma). Mitotane causes a decrease in endogenous cortisol production; if insulinoma is present, serum glucose levels also may fall, causing a hypoglycemic crisis.

- Give mitotane (50 mg/kg) PO q24h for 7 days, then q48h until clinical signs start to resolve. At that time decrease q to q72h until signs are fully resolved, then maintain the ferret on 50 mg/kg once q7–30d as necessary.

- Mitotane must be compounded in 50-mg aliquots in #1 capsules. Capsules must be administered intact. Have owners coat the capsules with vegetable oil, push into the back of the throat, and follow with a palatable liquid or blenderized cat food to promote swallowing.

- The most common side effect of mitotane is hypoglycemia. Teach owners to recognize the signs of hypoglycemia, and have prednisone available at home. If side effects occur, discontinue mitotane and administer prednisone (1.0–1.25 mg) PO.

- If continuation of mitotane therapy is desired after a hypoglycemic crisis, administer concomitantly with prednisone (see Insulinoma).

- **Ketoconazole** is not effective in the treatment of adrenal disease in the ferret.

**Surgical Therapy**

Follow the adrenalectomy preoperative protocol described for dogs (see Chapter 33).

- Fast the ferret 4 to 6 hours preoperatively. Place an indwelling IV or IO catheter preoperatively, and administer fluids pre-, intra-, and postoperatively. If insulinoma is present concurrently treat and monitor appropriately.

- Perform a ventral midline laparotomy. Palpate and visualize both adrenal glands carefully. Normal adrenal glands are 5 to 8 mm × 2–3 mm in size, are pale pink in color, and are typically surrounded by fat.

- The left adrenal gland lies in a fat pad cranial to the left kidney. The right adrenal gland is located cranio-medial to the right kidney under the caudate liver lobe adjacent to the vena cava. It may be necessary to transect the hepatorenal ligament to fully visualize and palpate this gland.

- Adrenal changes may be subtle, especially in younger ferrets and because the adrenal glands are surrounded by fat. Visual changes, such as dark circular lesions and small raised cysts, may be present instead of gross enlargement.

- One or both adrenal glands may be affected. If only the left adrenal gland is affected, removal is relatively straightforward. If the right adrenal gland is affected, removal can be difficult because of the gland’s proximity to the vena cava and liver (see Chapter 33).

- If both adrenal glands are affected, remove the left adrenal gland and debulk the right adrenal gland. Bilateral adrenalectomy has been described in the ferret, but should be done with caution. Monitor postoperatively for development of acute adrenal hypocorticism. If acute AHC develops, treat as described for dogs (see Chapter 33).

**Key Point** Always perform a complete abdominal exploratory. Observe and palpate the pancreas at surgery for insulinomas, which often are found concurrently with adrenal neoplasia.

**Postoperative Care and Complications**

- Monitor fasting serum glucose levels every 60 to 90 days during mitotane therapy and after adrenalectomy, even if no pancreatic nodules were evident during surgery.

**Prognosis**

- The prognosis following successful surgery is good. A full resolution of clinical signs can be expected in many cases.

- Recurrent or continued symptoms of adrenal gland disease may be associated with development of a tumor on the remaining adrenal gland, or recurrence of an adrenal tumor due to metastasis (which is rare).

- Even without treatment, ferrets may survive up to 2 years or longer after diagnosis, although the hair loss is generally progressive.

- Potential sequelae to chronic adrenal gland disease include prostatic disease, bone marrow suppression, or mechanical interference with the vena cava (right adrenal gland).

**Pheochromocytoma**

Pheochromocytomas are adrenal tumors that arise from the adrenal medulla and produce excessive amounts of catecholamines. Pheochromocytomas have been reported in ferrets, but are rare. Treatment of choice is surgical removal of the affected gland.
LYMPHOSARCOMA

Lymphosarcoma (lymphoma) is common in ferrets of all ages, and is similar in presentation to the disease in cats and dogs (see Chapter 27). Three presentations may occur in the ferret and include lymphosarcoma, lymphocytic, and lymphoblastic forms.

Etiology

• A viral etiology has been hypothesized.

Clinical Signs

Clinical signs are variable, depending on the form of lymphoma present and the organ system involved.

• Lymphosarcoma: Solid tissue tumors are present in the organs or lymph nodes.
• Lymphocytic lymphoma: Adult ferrets are most commonly affected. The course and survival time can be long. Peripheral lymphadenopathy is typically present and metastasis to visceral organs may occur. The neoplastic cell identified on cytology or histopathology is a mature, well-differentiated lymphocyte.
• Lymphoblastic lymphoma: Young ferrets are most commonly affected. Leukemia and neoplasia in visceral organs occur early in the course of this form of the disease. Large immature lymphocytes are noted on cytology or histopathology.
• Other forms: Cutaneous lymphoma may occur in the ferret. Clinical signs that may accompany any form of lymphoma include:
  • Inappetence, lethargy, splenomegaly, and weight loss despite a normal appetite
  • Dyspnea, tachypnea, and exercise intolerance
  • Peripheral lymphadenopathy and/or abnormal CBC
  • Acute collapse, often with pyrexia
  • Fever of unknown origin
  • Cutaneous masses
  • Chronic diarrhea and/or rectal prolapse
  • Some ferrets are asymptomatic; lymphoma may be an incidental finding during evaluation for another medical problem.
  • Lymphoma tends to be a more acute, fulminant disease in younger animals.

Diagnosis

The method of diagnosis depends on the organ system involved.

• Obtain a thorough history and physical examination.
• Perform a CBC, platelet count, and a serum biochemistry profile. If the ferret is anemic, perform a reticulocyte count.
• Often the CBC and differential WBC counts are not diagnostic for lymphoma. The CBC may be normal or may reveal an absolute or relative lymphocytosis. Anemia, leukopenia, and thrombocytopenia may be seen. Abnormal lymphocytes may occasionally appear in the differential count.
• Persistent absolute lymphocyte counts greater than 3500 or a relative lymphocytosis (>60%) are considered suspicious; repeat the CBC in 4 to 6 weeks and perform a bone marrow biopsy and/or lymph node biopsy if the CBC results are repeatable or if lymphadenopathy is present.
• The serum chemistry profile may disclose elevated liver enzymes if the liver is involved; paraneoplastic syndromes are uncommon in the ferret.
• Perform thoracic and abdominal radiography and ultrasonography to evaluate for intra-thoracic and intra-abdominal masses.
• Perform fine-needle aspiration or biopsy of affected tissues for histological and cytological examination. Fine-needle aspiration of the spleen is usually inconclusive.
• Lymph node biopsy is often the most helpful diagnostic tool for diagnosis of lymphoma. If possible, biopsy an enlarged lymph node. When lymphadenopathy is not present, biopsy the popliteal lymphnode. The popliteal lymph node is the most accessible peripheral node for biopsy. Avoid biopsy of intra-abdominal lymph nodes if possible.
• Perform bone marrow aspiration to identify infiltration by neoplastic cells and the disease (see “Clinical Techniques”).

Treatment

Splenectomy

• If the spleen is involved, perform a splenectomy (see Chapter 25) to reduce the overall tumor load.

Chemotherapy

Chemotherapy for lymphoma may be successful (approximately 10% remission rate). In general, protocols have been adapted from feline medicine (see Chapters 26 and 27).

• Success of chemotherapy may be affected by the age of the ferret, concurrent disease (e.g., adrenal gland disease, insulinoma), concurrent medication, inappropriate use of and resistance to chemotherapeutic agents (ferrets treated with prednisone prior to chemotherapy), and the type of lymphoma present.
• Ferrets with bone marrow involvement or with solid tumors involving organs typically have a poor prognosis.
• Longer periods of remission tend to occur in individuals with adult onset or lymphocytic lymphoma.
• IV chemotherapeutic agents are given via butterfly catheter or small-gauge needle with the ferret under
sedation; face-mask administration of isoflurane is the most convenient and rapid method of sedation.
• One chemotherapy protocol is outlined in Table 175-5.

**Key Point** Monitor the CBC weekly. If the WBC falls below 2000 WBC/\(\mu\)l, or the RBC falls below 4 ¥ 10^6/\(\mu\)l discontinue vincristine for 1 week or more until the WBC count increases to at least 3000 WBC/\(\mu\)l.

• Palliative therapy may be attempted by administering oral prednisone (2.2 mg/kg) PO q24h.
• Supportive care is important (see “Nutritional Support for Insulinoma”).
• Consider referral to an oncologist if experience with chemotherapeutic agents is limited.

**OTHER TYPES OF NEOPLASIA**
• **Chordoma:** Chordomas are tumors that arise from notochord remnants. Tumors occur most often at the tip of the tail, but may occur in the cervical region as well.
• **GIT:** Gastric adenocarcinoma.
• **Reproductive tract:** Tumors include granulosa cell tumors, luteomas, and leiomyomas in intact females and in remnant tissue in spayed females. Sertoli cell tumors have been reported in intact males.
• **Skin and Subcutis:** See Dermatologic Diseases in this chapter.
• Other tumors reported in ferrets include chondroma, chondrosarcoma, fibroma, fibrosarcoma, hepatic adenocarcinoma, hemangiomia, hemangiosarcoma, mast cell tumor, mesothelioma, osteoma, osteosarcoma, schwannoma, squamous cell carcinoma, thymoma, and renal and pancreatic carcinomas.

### Table 175-5. CHEMOTHERAPY PROTOCOL II FOR LYMPHOMA*

| Week | Drug         | Dose             |
|------|--------------|------------------|
| 1    | Vincristine  | 0.07 mg/kg, IV   |
|      | Asparaginase | 400 IU/kg, IP    |
|      | Prednisone   | 1 mg/kg, PO, q24h and continued throughout therapy |
| 2    | Cyclophosphamide | 10 mg/kg, SC |
| 3    | Doxorubicin  | 1 mg/kg, IV      |
| 4-6  | As weeks 1–3 above but discontinue asparaginase |
| 8    | Vincristine  | 0.07 mg/kg, IV   |
| 10   | Cyclophosphamide | 10 mg/kg, SC |
| 12   | Vincristine  | 0.07 mg/kg, IV   |
| 14   | Methotrexate | 0.5 mg/kg, IV    |

• IV, intravenously; IP, intraperitoneally; PO, per os; SC, subcutaneously.
• Protocol is continued in sequence biweekly after week 14.
• From Rosenthal KE: Ferrets. Vet Clin North Am 24:19–20, 1994.

**Dermatologic Diseases**

### SEASONAL CHANGES IN THE SKIN AND HAIRCOAT

Ferrets may experience dramatic seasonal changes in the haircoat triggered by photoperiod changes. This change is most apparent in the intact animal. If one is unfamiliar with these changes, normal coat changes may be interpreted incorrectly as a medical problem.

**Key Point** Individual animals may exhibit different patterns of coat change each successive year.

**Haircoat**
• A normal, diffuse, gradual thinning of the coat typically occurs in the spring when the photoperiod is increasing and continues through the summer. The coat typically becomes shorter and darker at this time, and the face mask may appear or disappear. Focal alopecia should not be present.
• Some ferrets may experience a dramatic 1-day loss of the undercoat.
• A normal, but dramatic, loss of body weight (up to 40%) may occur at this time as well.
• Hair growth will reverse in the fall and winter. Coats typically become longer, thicker, and lighter. Body weight may change (up to 40%) as well.
• Females in estrus and males “in season” may show an even more marked hair loss but should not have areas of alopecia.
• Males typically lose hair in the inguinal area because of constant rubbing to mark territory; the mid- and caudal abdomen is often wet with urine.
• Neutered ferrets or ferrets kept under artificial lighting conditions often experience no coat changes.
• Neutering or spaying may cause temporary, diffuse alopecia hair thinning postoperatively, particularly if the animal was reproductively active at the time of surgery. The preoperative color pattern may not return.
• At any time of the year, regrowth of hair that has been shaved for medical procedures is slow. This is particularly true in the winter and summer when no active hair growth is occurring.

**Skin**
• Hair regrowth (regardless of the cause of alopecia) is often preceded by a blue to purple discoloration of the skin that can alarm the owner. This discoloration
is caused by new hairs growing through the dermis, and is most noticeable on the abdomen and face.
• Intact jills may exhibit a bluish discoloration of the skin during estrus. If ovariohysterectomy is performed while a jill is in estrus, this discoloration may occur approximately 10 days postoperatively.
• Pseudonails associated with hyperkeratosis of the footpads may occur in ferrets older than 2 years of age that are housed on carpet or linoleum surfaces. Trim pseudonails as necessary. Rub a small amount of petroleum jelly or vitamin E oil into the pads daily to help prevent lesions.

INFECTIOUS DISEASES

Bacterial Infections
Cutaneous bacterial infections in ferrets are typically manifested as abscesses or as a diffuse, ulcerative pyoderma.

Abscesses
• Abscesses may develop secondary to puncture wounds, bites, or may develop in the inguinal fat after traumatic injury (e.g., being stepped on). For diagnosis and treatment of abscesses, see “Infectious Diseases.”

Ulcerative Pyoderma
Ulcerative pyoderma is the second most commonly encountered form of bacterial dermatitis in the ferret.

Etiology
• Various bacteria can cause ulcerative pyoderma. The most common agents are Staphylococcus and Streptococcus spp.

Clinical Signs
• Focal alopecia with diffusely hyperemic, thickened, ulcerated skin may occur over any area of the body.

Diagnosis
• Perform a cutaneous punch biopsy (see Chapter 37) to rule out diffuse cutaneous mast cell tumor, which may have a similar gross appearance.
• Perform bacterial culture and sensitivity testing.

Treatment
• Administer systemic antibiotics based on culture and sensitivity testing. Antibiotics effective in the treatment of pyoderma in ferrets often include amoxicillin-clavulanate (Clavamox, SmithKline) (13–25 mg/kg) q12h PO and cephalosporins (use feline dosages).
• Topical treatments include twice-weekly cleansing with an antibacterial shampoo containing chlorhexidine or benzoyl peroxide. Daily application of an antibacterial cream may be beneficial if the lesion is small and localized.

Canine Distemper Virus Infection
Dermatologic lesions are quite prominent with CDV infection in ferrets.
• Dermatologic signs typically begin with hyperemia around the lips, chin, eyes, and sometimes the inguinal area. With time, crusts and skin thickening may appear.
• Hyperkeratosis of the foot pads occurs as the disease progresses.
• See Infectious Diseases in this chapter for a detailed discussion of CDV in ferrets; also see Chapter 13 for a discussion of CDV in dogs.

Dermatophytosis
• Microsporum canis and Trichophyton mentagrophytes are the most common causes of superficial mycotic infections in the ferret.
• See “Infectious Diseases” in this chapter for diagnosis and treatment.

EXTERNAL PARASITES

Fleas
• Flea infestation may be encountered in pet ferrets. Clinical signs are similar to those seen in cats (see Chapter 45).

Treatment
• Flea shampoos, dips, or powders containing pyrethrin may be used. Products containing lindane or organophosphates are not recommended for use in the ferret.
• Imidacloprid (Advantage, Bayer Corporation, Shawnee Mission, KS) (0.4ml) topically every 3 weeks has been reported to be effective. No adverse effects have been noted. This drug may be used in conjunction with lufenuron.
• Lufenuron (Program, Norvartis Animal Health, Greensboro, NC) (45 mg) PO every 4 weeks has been anecdotally reported to be effective. Advise clients that there is a 6- to 8-week period before flea numbers are observed to decline.
• Fipronil (Frontline, Merial LTD., Iselin, NJ). Half the cat dose has been anecdotally reported to be effective. This drug may be used in conjunction with lufenuron.
• Selamectin (Revolution, Pfizer, New York, NY). Administration of the cat dosage has been anecdotally reported to be effective.
• Flea collars are not recommended because they come off easily and small pieces can be ingested.
• Treat the environment for fleas.

Ear Mites
Ear mite infection in ferrets is caused by *Otodectes cynotis*, the same parasite that infects cats and dogs.

**Clinical Signs**
- Ferrets rarely exhibit pruritis, even with heavy mite infestation.
- Ferrets normally have large volumes of dark reddish-brown ear wax present in the ear canal, which resembles the debris present with *O. cynotis* infestation. If ear mite infestation is present, wax production may become excessive and cause occlusion of the external ear canal.
- *O. cynotis* infestation may be accompanied by secondary bacterial infection, leading to otitis media or otitis interna. Neurological signs such as head tilt and circling may occur (see Chapter 61 for techniques for the management of otitis media in cats).
- When chronic ear mite infestation is present, lichenification and a bluish pigment may appear on the inner surface of the pinnae. These changes are caused by a response to chronic irritation, and usually regress after treatment.
- Rarely, *O. cynotis* may colonize other parts of the body.

**Diagnosis**
- Examine all ferrets for ear mites; the incidence of infestation is high in some populations.
- Mites in the ear canal can often be visualized using an otoscope; however, otoscopic examination is often difficult because of the uncooperative nature of the patient and small size of the ear canal.
- Confirm the diagnosis by microscopic examination of ear debris.

**Treatment**
- Thoroughly clean the ears.
- Administer ivermectin (Ivomec, Merck-Sharp & Dohme Agvet), (1 mg/kg) divided into two doses. Instill one dose into each ear. Repeat in 2 weeks.
- Tresaderm (Merck Agvet, Rahway, NJ) may be used to treat ear mites in ferrets. Administer 2 drops in each ear q24h for 7 days, stop for 7 days, then repeat. This medication has been reported to be effective in treatment of ear mites in the ferret.
- Selamectin may be used for treatment of ear mites in the ferret. Use at the dose described for treatment of fleas.
- Bathe the ferret within 24 to 48 hours after treatment. Wash all bedding and treat all other potential hosts in the household (see Chapter 59).
- Topical treatments may not be effective due to the narrow size of the ear canal, and patient resistance to treatment.
- Persistent infections may be due to the presence of ear mites on the body, or failure to deliver the topical agent effectively. In such cases, parenteral administration of ivermectin (0.5 mg/kg) SC every 7 to 10 days for 2 treatments may be necessary. Do not use topical and parenteral ivermectin together.

Sarcoptic Mange

**Etiology**
*Sarcoptes scabiei* mites are transmissible between dogs and ferrets via contact with the infected hosts or their bedding. (See Chapter 44 for discussion about sarcoptic mange in dogs and cats.)

**Clinical Signs**
- Lesions are typically confined to the feet, which become hyperemic, swollen, and intensely pruritic. Crusting often occurs around the nails, and in severe cases the nails may slough.
- Generalized alopecia, accompanied by intense pruritis, occurs rarely.

**Diagnosis**
- A positive diagnosis is based on clinical signs, exclusion of differential diagnoses, and positive skin scrapings obtained from several sites (false negative results do occur).
- A common differential diagnosis is contact allergy. Similar lesions have been observed in ferrets housed on plastic-floor cages. These lesions resolve when the cage bottom is changed to wire or wood.

**Treatment**
- Advise clients of the zoonotic potential of this parasite.
- Treatment may need to be based on differential diagnoses; mites may be difficult to identify on skin scrapings.
- Administer ivermectin (0.5 mg/kg) SC every 7 days for three treatments.
- Lime sulfur dips may be used instead of ivermectin. Dip ferrets in 2% lime sulfur every 7 days until signs have resolved for 2 weeks.
- Wash all bedding and treat all potential contact hosts in the household.
Demodectic Mange

**Etiology**
Demodicosis is rare in the ferret.

**Clinical Signs**
- Otitis externa has been associated with demodicosis. This may be the only presenting sign.
- Localized alopecia accompanied by pruritis may occur.

**Diagnosis**
- Mites may be identified on routine skin scrapings and examination of ear canal debris.

**Treatment**
- Treatment can be difficult. Use ivermectin at the daily dose described for dogs (see Chapter 43).
- Do not use mitotane.

Estrus Alopecia
Allopecia may be seen in intact females that have been in estrus for 1 month or longer.

**Clinical Signs**
- Bilaterally symmetrical hair loss over the shoulders and flanks, which eventually progresses to involve the entire body. Hairs epilate easily, and the underlying skin appears normal.
- A grossly enlarged vulva indicates a state of estrus. Be aware that the ferret also may be anemic and thrombocytopenic (see “Anemia”).

**Diagnosis**
- Diagnosis is based on clinical signs in an intact female.

**Treatment**
- Perform an ovariohysterectomy (see Chapter 91) if the ferret is stable enough for the procedure, or induce ovulation with HCG (see “Termination of Estrus”; “Anemia”).
- Hair regrowth will recur rapidly after surgery or ovulation; however, changes in hair length, color, or thickness are common.

Adrenal Gland Disease
- Bilateral, symmetrical alopecia is a common sign of adrenal disease in the ferret (see “Adrenal Gland Disease”).

**Hypothyroidism**
- Hypothyroidism has not been documented in ferrets.

**NEOPLASIA**
Neoplasia of the skin is the third most common neoplasm reported in the ferret and commonly occurs in ferrets 1 year of age and older. Complete removal of skin masses using wide surgical excision followed by histopathology is recommended.
Mast Cell Tumors

Mast cell tumors are the most common skin masses encountered and are typically benign in the ferret.

- Individual tumors typically appear as slightly raised, flat, button-like cutaneous masses ranging in size from 2 to 10 mm. The tumors are often tan in color or may be hyperemic with a dark flaky crust. Tumors may also appear as raised, ulcerated areas, or as diffuse areas of erythema and crusting. Pruritis may be present at the site.
- Mast cell tumors have occasionally been associated with diffuse or generalized areas of alopecia that resolve with surgical removal of the tumor.
- Metastasis is rare but has been reported in the lung and gallbladder (see Chapter 28 for information about mast cell tumors in dogs and cats).

Sebaceous Epitheliomas

- These tumors may also be referred to as haral cell tumors or sebaceous adenomas and are common in the ferret.
- Tumors may appear as wart-like, ulcerated, or cystic masses ranging in size from 0.5 to 2 cm.
- Excision is usually curative. Recurrence is rare, and metastasis is not reported.

Other Neoplasms

- Other, less common neoplasms of the skin and subcutaneous tissues include: basal cell carcinoma, basi-squamo-sebaceous carcinoma, hemangioma, histiocytoma, leiomyosarcoma, lymphoma, myxosarcoma, neurofibrosarcoma, perianal gland adenocarcinoma, sebaceous gland adenocarcinoma, and squamous cell carcinoma.
- Adenocarcinomas often metastasize to regional lymph nodes, liver, and lungs.
- Diagnosis, treatment, and prognosis for these tumors in ferrets are the same as for dogs and cats (see Chapter 30).

Cardiovascular Diseases

CHARACTERISTICS OF THE NORMAL FERRET HEART

- Cardiac auscultation is centered more caudally in the thorax than are auscultations in cats.
- The heart extends from the sixth rib to the caudal border of the seventh or eighth rib (compared with cats, where it extends from the second to the sixth rib).
- The heart rate averages 180 to 250 beats per minute.
- A pronounced sinus arrhythmia and pronounced bradycardia are common during auscultation.
- Cardiac disease is relatively common in the ferret. Quality of life and long-term prognosis for ferrets with cardiac disease depends on the type and severity of cardiac disease present, and the initial response to treatment. Many ferrets do well for months on the appropriate medications.

Congestive Heart Failure

Clinical Signs

- Ferrets appear to compensate well for early cardiac insufficiency, perhaps because a slight decrease in activity is not readily apparent to owners.
- Ferrets with congestive heart failure (CHF) may present with clinical signs that resemble symptoms associated with other disease entities, such as anorexia, ascites, coughing, dehydration, dyspnea, exercise intolerance, generalized weakness, hindlimb weakness, hypothermia, lethargy, tachypnea, and weight loss.
- Pale or cyanotic mucus membranes and a prolonged capillary refill time (CRT) may be noted on physical examination.
- Jugular pulses may be present when right-sided CHF is present.
- Femoral pulses may be weak, irregular, or normal.
- Ascites, hepatomegaly, or splenomegaly may be noted on abdominal palpation.
- Murmurs may be noted on auscultation, and are typically associated with valvular insufficiency.

Diagnosis

- History and physical examination findings are important in the diagnosis of heart disease.
- Perform a complete physical examination, including auscultation of the heart, and evaluation of the capillary refill time. Observe for tachypnea or dyspnea and auscult the lungs. Palpate the abdomen and examine for ascites.
- Key Point Proceed with further testing only if the ferret is stable. Otherwise, administer furosemide and oxygen therapy.
- Diagnosis requires information obtained by radiography, ECG, and echocardiography.
- Obtain whole-body radiographs. The cardiac silhouette typically appears enlarged and globose in shape with rounded right and left ventricles. Ascites, hepatomegaly, pleural effusion, and pulmonary edema may be present as well.
- Evaluate a CBC, serum biochemical profile, and urinalysis to determine if azotemia, electrolyte abnormalities, or other systemic diseases are present. Perform a heartworm test if the history is supportive for potential exposure.
- If thoracic or abdominal effusion is present, perform thoraco- or abdominocentesis and submit fluid for
Perform centesis as described for cats; take into consideration the relatively caudal position of the heart in ferrets. Sedation is usually necessary. A modified transudate is typically associated with CHF.

**Key Point**

Sedation with isoflurane is recommended when necessary. Sedation with ketamine or a ketamine-diazepam combination raises the heart rate. The heart rate tends to decrease with ketamine-xylazine sedation; therefore, avoid using xylazine in ferrets with suspected cardiac disease.

Echocardiography is the most useful diagnostic tool in the ferret. The same echocardiographic changes observed in the dog and cat are seen in the ferret. (Table 175-7).

### Treatment

**Key Point** Treatment of acute CHF should focus on improving oxygenation and reducing cardiac preload and afterload.

### Table 175-6. Electrocardiographic Data for 52 Clinically Normal Ferrets

| Parameter | Mean ± SD (Range) | Value† |
|-----------|-------------------|--------|
| Age (mo)  | 10–20 Average, 5.2| 233 ± 22 |
| Male:female ratio | All male | 1.25 |
| Body weight (kg) | 1.4 ± 0.2 | NA |
| Heart rate (beats/min) | 196 ± 26.5 (140–240) | 233 ± 22 |
| Rhythm | NA | 67% |
| Normal sinus | NA | 33% |
| Sinus arrhythmia | 86.13 ± 2.5 (79.6–90) | 77.22 ± 12 |
| Frontal plane MEA (degrees) | 0.122 ± 0.007 |
| Lead II | 0.024 ± 0.004 |
| P amplitude (mV) | 0.056 ± 0.0086 (0.04–0.08) | 0.047 ± 0.003 |
| P duration(s) | 0.044 ± 0.0079 (0.035–0.06) | 0.043 ± 0.003 |
| PR interval(s) | 0.04 ± 0.018 (0.08–0.14) | 0.12 ± 0.04 |
| QT interval(s) | 2.21 ± 0.42 (1.4–3) | 1.46 ± 0.84 |
| R amplitude (mV) | 3.3 mm |
| QT interval(s) | 3.5 mm |
| Fractional shortening | 42% |
| End-point septal separation | None |

NA, not available; MEA, mean electrical axis.

*All ferrets were sedated with ketamine-xylazine.

†Data from Bone L, Battles AH, Goldfarb RD, et al: Electrocardiographic values from clinically normal, anesthetized ferrets (Mustela putorius furo). Am J Vet Res 49:1884–1887, 1988.

‡Data adapted from Fox JG: Biology and diseases of the ferret. Philadelphia, Lea & Febiger, 1988, p 170; and Edwards J: Unpublished data, 1987.

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**Table 175-7. Mean Echocardiographic Values for 34 Normal Adult Ferrets**

| Parameter | Mean Value |
|-----------|------------|
| Left ventricle, end-diastolic | 11.9 mm |
| Left ventricle, end-systolic | 6.4 mm |
| Left ventricular posterior or free wall | 3.3 mm |
| Fractional shortening | 42% |
| End-point septal separation | None |

From Sitinas N, Beeber N, Skeels M: Unpublished data, 1992.

- Place the ferret in an oxygen-rich environment. Administer supportive care such as subcutaneous fluids (e.g. 0.45% saline and 2.5% dextrose), and provide nutritional support for ferrets that are anorexic.
- Administer diuretics such as furosemide (1–4 mg/kg) IM or IV bid–tid.
- Nitroglycerin 2% ointment may be applied to the skin in the axilla, inguinal area, or on a hairless body surface.
- Angiotensin-converting enzyme (ACE) inhibitors may be given to reduce afterload and preload. Give enalapril (Enacard, Merck Agvet Division) (0.5 mg/kg) PO q48h, then titrate up to q24h if possible. ACE inhibitors may cause hypotension in ferrets, titrate to effect.
• When diuretics and ACE inhibitors are used together it is important to monitor for azotemia.
• Perform thorocentesis or abdominocentesis when indicated.
• Monitor body weight, CRT, heart rate and rhythm, hydration status, mucous membrane color, respiratory rate, respiratory effort, BUN, creatinine, and serum electrolytes.

▼ **Key Point** Chronic therapy typically includes the use of ACE inhibitors, and diuretics with the addition of digoxin in ferrets with dilated cardiomyopathy. Whenever possible, try to titrate the diuretic dose to the lowest possible dose without recurrence of pleural effusion or pulmonary edema.

• Administer digoxin elixir (0.01 mg/kg) PO sid–bid to ferrets with dilated cardiomyopathy.
• Side effects associated with digoxin include anorexia, arrhythmias, diarrhea, lethargy, and vomiting.
• Serum digoxin levels should be monitored every 4 to 8 weeks. Normal values have not been published for the ferret; reference values for dogs and cats are used for interpretation.
• Use of antiarrhythmic drugs such as atenolol or diltiazem is not well documented in the ferrets, but may be useful in the treatment of ferrets with hypertrophic cardiomyopathy.
• Salt-free diets may be beneficial; however, they are often unpalatable to ferrets. Instruct the owner to avoid feeding snacks, treats, or food items with a high-salt content.
• Management includes periodic reevaluation of heart rate and rhythm, serum electrolytes, and renal values. Radiographs should be used to monitor for the development of pulmonary edema or changes in the cardiac silhouette. ECG and echocardiography should be repeated periodically as well.

**Cardiomyopathy**
Cardiomyopathy may occur in ferrets 2 years of age or older. Dilated (congestive) and hypertrophic forms can occur; the dilated form is more common.

**Dilated Cardiomyopathy**

**Etiology**
The cause of dilated cardiomyopathy (DCM) in the ferret is unknown.

**Clinical Signs**
• Abdominal enlargement secondary to ascites, anorexia, dyspnea, lethargy, and weight loss are often noted.
• Ascites, heart murmur, pale mucous membranes, tachycardia, and weakness may be noted on physical examination.

• Moist rales and increased respiratory sounds may be noted when pulmonary edema is present.
• Pleural effusion may be present, and may cause an increased inspiratory effort. The heart may sound muffled on auscultation.
• Coughing generally is not noted.

**Diagnosis**
• See the CHF section in this chapter.

**Treatment**
• Treatment is the same as that described for CHF.
• Taurine supplementation does not appear to have any effect on DCM in the ferret.

**Follow-Up Care**
• See the CHF section in this chapter.

**Hypertrophic Cardiomyopathy**

**Etiology**
The cause of hypertrophic cardiomyopathy (HCM) is unknown.

**Clinical Signs**
• Clinical signs may be compatible with those described for CHF or DCM (see above).
• Other clinical signs are similar to those described for the cat, and include acute onset of congestive heart failure and/or sudden death.

**Diagnosis**
• Follow the same guidelines described for DCM.
• Include HCM on the rule-out list when evidence of cardiac disease is noted on the physical examination or diagnostic evaluation.
• Radiographs may not be beneficial in the diagnosis of HCM.
• Echocardiography should be used for definitive diagnosis.

**Treatment**
• Treatment should be aimed toward alleviating signs of CHF and improving the diastolic efficiency of the left ventricle.
• Administer beta-adrenergic blocking drugs such as atenolol (3.125–6.25 mg) PO sid. Titrate to effect.
• Administer calcium channel blockers such as diltiazem (3.75–7.5 mg) PO bid. Titrate to effect.
• Diuretics are indicated if symptoms of CHF are present (see above).

**Follow-Up Care**
• Follow-up is the same as that described for CHF and DCM.
Heartworm Disease

Natural and experimental heartworm infections have been reported in ferrets (see Chapter 152). The clinical presentation of heartworm disease typically resembles that of cats; however, the life cycle of *Dirofilaria immitis* in ferrets is similar to the life cycle present in the dog. Reported adult worm burdens range from 1 to 10. The presence of only one adult worm in the heart can be lethal.

**Etiology**

- Heartworm disease is caused by the canine heartworm *Dirofilaria immitis*, a filarial nematode that is transmitted via mosquitoes.
- Ferrets that are housed outdoors in endemic areas are at greatest risk of infection; however, ferrets kept indoors also can become infected.

**Clinical Signs**

- Clinical signs include coughing, dyspnea, hepatomegaly, inappetence, lethargy, melena, weakness, and symptoms associated with right-sided CHF (pulmonary edema, pleural effusion, ascites). Sudden death due to pulmonary artery obstruction may also occur.
- Microfilaria may be present in the blood of approximately 50% of infected ferrets.

**Diagnosis**

- Diagnosis is based on the history, clinical signs, heartworm test results, radiographs, and echocardiography.
- If the history is compatible with cardiac failure, inquire about possible mosquito exposure.
- Physical examination findings resemble those of heart failure (see above).

▼ Key Point Minimize stress in ferrets suspected of heartworm disease. If symptoms of congestive heart failure are present, delay further diagnostic evaluation until the patient is stabilized (see “Treatment of Congestive Heart Failure”).

- Obtain whole body radiographs. Thoracic changes may include cardiomegaly with enlargement of the right atrium, caudal vena cava, and right ventricle. Pleural edema and pleural effusion may be present as well. Radiographic changes in the peripheral pulmonary arteries are not typically noted because the worms tend to reside in the right side of the heart and in the main pulmonary artery. Abdominal changes often include hepatomegaly, splenomegaly, and ascites.
- If possible, draw blood for the modified Knott’s test for microfilaria. Microfilaria are identified in approximately 50% of infected ferrets.
- Submit blood for an enzyme-linked immunosorbent assay (ELISA) for *Dirofilaria* antigen. Antigen is produced by adult female heartworms; there is a potential for false negative test results in ferrets with low worm burdens. A commercial assay (Snap Heartworm Antigen Test Kit; IDEXX Laboratories Inc., Portland, ME) has been used to detect heartworm infection in the ferret.
- Perform a CBC, serum biochemical profile, and urinalysis to rule out the presence of other systemic diseases.
- If pleural or abdominal effusion is present, submit fluid for cytology. A modified transudate is typically noted when CHF is present.
- Echocardiography may be used to visualize heartworm(s) in the pulmonary artery, right ventricle, and right atrium; dilation of the right ventricle and right atrium may be assessed as well. Doppler echocardiography may be used to evaluate the patient for the presence of pulmonary hypertension.

**Treatment**

- Treatment of heartworm disease in ferrets is difficult. Success is dependent on early diagnosis, diligent supportive care, and long-term antithrombotic therapy in conjunction with adulticide therapy.
- If signs of CHF are present, treat this first, and stabilize the patient (see the CHF section).
- If the patient is symptomatic and microfilaremia positive:
  - Administer microfilaricidal therapy: Ivermectin (50 µg/kg) SC every 30 days until clinical signs and microfilaremia resolve.
  - Follow with adulticide therapy: melarsomine (Immiticide, Rhone Merieux, Athens, GA) using a two-stage protocol:
    - Stage 1: Administer a single dose of melarsomine (2.5 mg/kg) IM.
    - Stage 2: 1 month later, administer two injections of melarsomine (2.5 mg/kg) IM given 24 hours apart.
- Transient swelling at the site of injection is common.
- Administer prednisone (0.5 mg/kg) PO sid–tid during adulticide treatment and for as long as clinical signs persist.
- If pleural effusion is present administer diuretics (see the CHF section).
- Cage confinement is important for 4 to 6 weeks after treatment.
- Perform a post-treatment ELISA for heartworm antigen 3 months after adulticide therapy. Repeat every 30 days if results are positive. Most ferrets become seronegative 4 months after treatment.
- Begin heartworm prevention 1 month after adulticide treatment.
- If ferrets are microfilaria negative, administer adulticide therapy as described above.
Prevention

**Key Point** Because of the high mortality associated with heartworm disease, recommend preventive therapy for all ferrets in heartworm-endemic areas.

- Ivermectin may be given as preventive therapy beginning 1 month before and continuing 2 months after mosquito season. Liquid ivermectin 1% may be diluted in propylene glycol (0.3 ml ivermectin in 30 ml propylene glycol) and administered at a dose of (0.2 ml/kg) PO every month. This solution must be stored in an amber glass bottle out of sunlight.
- Feline Heartguard (Merck Agvet) may be administered using the dose appropriate for a 1- to 5-lb cat.
- If possible, house all ferrets in endemic areas within structures with mosquito-proof screening.
- Follow the same recommended guidelines for heartworm prevention in dogs and cats.

Valvular Heart Disease

Valvular heart disease may occur in ferrets >3 years of age.

**Clinical Signs**

- Clinical signs depend on the severity of the underlying disease process.
- Mitral regurgitation may be ausculted as a systolic murmur in the left apical region.
- Tricuspid regurgitation is ausculted in the right parasternal region.
- Dyspnea and moist rales may be noted on auscultation of the lungs if CHF is present.

**Diagnosis**

- Obtain thoracic radiographs to evaluate the size of the heart and to determine if CHF is present. Pulmonary edema typically appears as a mixed alveolar and interstitial pattern in the caudodorsal lung lobes.
- Electrocardiography (ECG) may be normal or may demonstrate evidence of atrial arrhythmias.
- Echocardiography typically demonstrates thickening of affected valves and atrial enlargement.
- Doppler echocardiography may be used to identify and quantify the degree of regurgitation present. Aortic regurgitation is often noted in ferrets and is considered an incidental finding.

**Treatment**

- Treatment is recommended if CHF is present, or if cardiac enlargement is significant (see the CHF section).

Myocarditis

Myocarditis occurs when the myocardium is infiltrated with inflammatory cells, resulting in the development of reduced myocardial function, arrhythmias, and replacement of the normal myocardial tissue with fibrous tissue.

**Etiology**

- Causes include sepsis, systemic vasculitis, parasitic, bacterial or viral infection, and autoimmune disorders.
- Aleutian disease can cause fibrinoid necrosis and mononuclear cell infiltration of the arterioles of the heart.

**Clinical Signs**

- Antemortem diagnosis is difficult.
- Suspect myocarditis if arrhythmia and/or acute myocardial dysfunction is noted in association with multisystemic illness.
- Definitive diagnosis is made by histopathological evaluation of affected myocardial tissue.

**Treatment**

- Treatment should be directed at identifying and treating the underlying systemic disease.
- Cardiovascular support should be provided and may include the use of diuretics or antiarrhythmic drugs (see the CHF section).

Other Cardiac Diseases

As clinical experience with pet ferrets increases, other types of cardiac disease are likely to be recognized. Third-degree heart block (of unknown etiology) and various forms of valvular disease, including mitral and tricuspid insufficiency and endocarditis, have been seen in ferrets.

- The approach to these conditions in ferrets is the same as for other companion animals; use the drug dosages given previously for cardiac myopathies.

Gastrointestinal System

**Characteristics of the Normal Ferret Digestive Tract**

**Teeth**

- The permanent teeth erupt between 50 and 74 days of age.
- The dental formula is 2 (I3/3, C1/1, Pm3/3, M1/2).
- The third upper premolar (carnassial tooth) has three roots. The second lower molar has one root. All other premolars and molars have two roots.

**Gastrointestinal Tract**

- The ferret is an obligate carnivore with a simple stomach, short intestinal tract, no cecum or ileocolic valve, and a short colon.
The duodenum terminates at the jejunoileum; there is no gross anatomic distinction between the jejunum and the ileum.

The junction of the jejunoileum and the colon is determined by evaluating the pattern of anastomosis between the jejunal artery and the ileocolic artery.

GI transit time is approximately 3 to 4 hours.

**Anal Sacs**

The anal sacs are located between the external and internal anal sphincter muscles at 4 and 8 o’clock. The ducts are located near the mucocutaneous junction.

**Diet**

The exact nutritional requirements of the ferret have not been determined.

The diet of the ferret must contain predominantly animal protein and fat.

Due to the short digestive tract and rapid GI transit time, the ferret requires a concentrated maintenance diet high in protein (30–35%) and fat (15–18%), and low in fiber. The protein quality should be 85% to 90% digestible.

Breeding ferrets and kits may require diets higher in protein and fat.

Meat, poultry, meat and poultry meals, and other animal-based proteins should appear first, then several more times on the food ingredient list.

Complex carbohydrates (starch, fiber) are not readily digested by the ferret. High-fiber diets can induce a relative protein-calorie deficiency; the ferret cannot eat enough of a low-density food to meet its high maintenance requirements.

Premium cat foods and ferret diets typically meet the ferret’s nutritional requirements for growth and reproduction.

Treats and supplements should not exceed more than 10% of the daily diet. Acceptable treats include meat baby foods, and moist cat or ferret diets. High-sugar or carbohydrate treats should be limited, especially if insulinoma is present.

Fatty acid supplements should be given in measured amounts (a few drops per day). Administration of large quantities of fatty acid supplements may reduce the intake of the balanced diet.

Canine diets should not be fed to ferrets; the protein, fat, and carbohydrate content is not appropriate, and the diets often contain high percentages of grain and vegetable matter.

The long-term effect of formulated dry and canned diets on the long-term health of ferrets is controversial among some practitioners.

Some practitioners feel that feeding commercial diets containing large quantities of plant-based ingredients contributes to the development of eosinophilic gastroenteritis, inflammatory bowel disease, insulinoma, urolithiasis, and general untriftiness. For example, most ferrets in the United States are fed dry kibbled diets, and the incidence of insulinoma is high. Many ferrets in Europe and Australia are fed whole prey items (e.g., a “natural diet”), and the incidence of insulinoma is low.

A correlation between diet and the development of certain diseases in ferrets is hypothetical at this time; however, this controversy demonstrates the need for longer-term diet studies in the ferret.

**DENTAL DISEASE**

The canine teeth are often worn or broken at the tips due to biting and gnawing.

Broken canine teeth typically are not painful unless the dental pulp is exposed.

Dental tartar and periodontal disease are common in ferrets over 2 years of age.

Soft, moist diets may predispose ferrets to the development of dental disease.

Tartar typically accumulates first on the second and third upper premolars.

Dental abscesses are not common, but may be noted, even in young ferrets.

Follow the basic medical and surgical treatment principles described for dental diseases in dogs and cats (see Chapter 64).

**SALIVARY MUCOCELE**

Ferrets have five major pairs of salivary glands: the parotid, submandibular, sublingual, molar, and zygomatic.

Salivary mucocele occurs secondary to trauma or infection of a salivary gland.

Salivary mucocele typically presents as a soft to firm swelling in the region of the orbit, oral commissure, or mandibular lymph node. Aspiration of the swelling often yields a clear to serosanguinous or mucinous fluid; microscopic examination demonstrates amorphous debris and occasional RBCs.

Treatment of choice is surgical excision of the affected gland (see Chapter 64).

Advise clients that recurrence is possible.

**MEGAESOPHAGUS**

Megaesophagus is rare in ferrets.

**Etiology**

The etiology of megaesophagus in ferrets is unknown (see list of possible causes in dogs in Chapter 65).
Clinical Signs

- Clinical signs resemble those described for the dog and include: lethargy, anorexia, dysphagia, coughing, choking, dyspnea, weight loss, and regurgitation.
- Clients may indicate that the ferret vomits up large boluses of food.

Diagnosis

- Diagnosis may be based on clinical signs and radiographic evidence of megaesophagus.
- Obtain thoracic radiographs. The esophagus is often dilated and filled with air in the cervical and thoracic regions. Food may be present within the lumen of the esophagus.
- Perform a barium contrast study to delineate the esophageal mucosa and to identify potential mural lesions, strictures, or obstructions.
- Aspiration pneumonia may be visible radiographically.

Treatment

- Follow canine treatment protocols. The prognosis is poor; response to therapy is usually not successful.
- GI promotility agents such as metoclopramide (Reglan, AH Robins Company, Inc., Richmond VA) (0.2–1mg/kg) PO tid–qid may be helpful.
- Administer H2-receptor blocking drugs such as cimetidine, ranitidine (Zantac, Glaxo Pharmaceuticals, Research Triangle Park, NE), or famotidine (Pepcid AC, Johnson and Johnson, Fort Washington, PA).
- Administer antibiotics if indicated for aspiration pneumonia.
- Supportive care includes feeding high-calorie, high-protein slurried diets 3 to 4 times per day, and elevating the ferret for 15 to 30 minutes immediately after feeding.

NAUSEA AND VOMITING

- Ferrets, like other carnivores, are able to vomit.
- Differential diagnoses to consider for vomiting include esophageal and gastroenteric disorders (see below).
- Ferrets often demonstrate symptoms associated with nausea or vomiting when gastroenteritis, GI disease, gastric ulcers, Helicobacter mustelae gastritis, or GI foreign bodies are present. Hypoglycemia may cause signs of nausea as well (see discussion of Insulinoma in this chapter).
- Signs of nausea include hypersalivation and pawing at the mouth.
- Ferrets may demonstrate bruxism (grinding of the teeth) when abdominal discomfort is present.

GASTROINTESTINAL PARASITES

- GI parasites are uncommon in the ferret. Coccidiosis and giardiasis are occasionally seen. Nematodiasis is rare.
- Routine fecal testing is still recommended, especially in young animals and ferrets with diarrhea or rectal prolapse.
- Young ferrets with coccidiosis may have diarrhea and may be severely dehydrated.
- Cryptosporidiosis may occur in ferrets, but typically does not result in clinical disease. The zoonotic potential is unknown; however, it may be prudent to warn immunosuppressed owners of the potential for zoonosis.

Treatment

- Treat with appropriate anthelmentics following the protocols and dosages used for cats (see Chapter 69).

GASTRITIS, AND GASTRIC AND DUODENAL ULCERS

Gastric and duodenal ulcers have been documented in laboratory ferrets and reported occasionally in pet ferrets. Clinical signs are often vague, making the diagnosis difficult.

Etiology

- The etiology is unknown but may include stress, GI foreign body, H. mustelae gastritis, administration of ulcerogenic drugs, GI neoplasia, and azotemia secondary to renal disease.
- H. mustelae is similar to H. pylori, the bacteria associated with gastritis and ulceration in humans. H. mustelae infection in the ferret can be an incidental finding, or can induce gastritis, duodenitis, and GI ulceration.

Clinical Signs

- Gastritis and ulcers may be acute or chronic.
- Clinical signs include anorexia, lethargy, hypersalivation, bruxism (tooth grinding), weight loss, vomiting, and melena.

Diagnosis

- Presumptive diagnosis may be made based on the history and clinical signs.
- Perform a CBC and serum biochemistry profile to rule out systemic and metabolic disease.
- Obtain fasting whole-body radiographs to help rule out the presence of a GI foreign body or trichobezoar.
A barium study may be used to demonstrate GI ulceration.

Exploratory laparotomy/gastrotomy is often required for a definitive diagnosis.

Diagnosis of H. mustelae gastritis is often a diagnosis of exclusion. Definitive diagnosis requires the finding of organisms along typical histological lesions on gastric biopsy specimens.

Treatment

- Debilitated, anorexic ferrets may require hospitalization for supportive care.
- If the patient is vomiting, withhold food for 6 to 12 hours. Administer IV fluids containing dextrose, and monitor for signs of hypoglycemia. When vomiting has resolved begin to offer small, bland meals.
- Feed small meals of a bland, moist diet tid–qid (see diet recommendations in “Insulinoma” section). Avoid feeding high-fiber dry foods.
- Administer broad spectrum antibiotics if the ferret is debilitated.
- Administer a gastric protectant. Options include:
  - Bismuth subsalicylate (Pepto Bismol, Procter & Gamble) (1 ml/kg) PO tid.
  - Sucralfate (Carafate, Marion Merrell Dow, Inc., Kansas City, MO) (100 mg/kg) PO tid–qid.
  - Systemic H₂-receptor antagonists such as cimetidine and famotidine.
  - Omeprazole (Prilosec, Astra Merck, Inc., Wayne PA) (_ the contents of a 10-mg capsule mixed with soft food) PO sid-bid.
- If H. mustelae infection is suspected, administer the following three drugs concurrently for at least 2 weeks (“triple therapy”):
  - Amoxicillin (10 mg/kg) PO, SC bid.
  - Metronidazole (20 mg/kg) PO bid.
  - Bismuth subsalicylate (Pepto-Bismol, Procter & Gamble) (see dosage information above).
- If H. mustelae infection is suspected, administer the following three drugs concurrently for at least 2 weeks (“triple therapy”):
  - Amoxicillin (10 mg/kg) PO, SC bid.
  - Metronidazole (20 mg/kg) PO bid.
  - Bismuth subsalicylate (Pepto-Bismol, Procter & Gamble) (see dosage information above).

GASTROINTESTINAL FOREIGN BODIES

GI obstruction caused by foreign body ingestion or hairballs is one of the most common problems in pet ferrets.

Etiology

- Foreign bodies typically occur in ferrets younger than 1 year of age; trichobezoars (hairballs) are common in ferrets older than 2 years of age.

Key Point Suspect the presence of a GI foreign body in any young ferret presented for anorexia, even if no vomiting is reported.

- Rubber and foam objects are the most common foreign bodies. Obstruction with a hairball (older ferrets), cloth, or plant material also may occur.

Clinical Signs

- Lethargy, partial or total anorexia, hypersalivation, bruxism, pawing at the mouth, weight loss, and diarrhea are the most common clinical signs of GI foreign body. Hindlimb weakness, dehydration, and melena may be noted as well.
- Vomiting is uncommon; however, if the ferret is vomiting, be suspicious that a GI foreign body may be present.

Diagnosis

- Diagnosis is based on the history, physical examination findings, radiographs, or exploratory laparotomy.
- History: Identify possible types or causes of foreign body ingestion. Ask the owners if hairball preventative is used routinely.
- Physical Examination: Large gastric foreign bodies are often palpable. Small foreign bodies in the small intestine may be associated with localized pain.
- Obtain fasting (4–6 hours) plain whole body radiographs. Radiographs may reveal segmental ileus, and marked gaseous distention of the stomach and/or bowel. Occasionally a foreign body or trichobezoar can be identified.
- Obtain a GI barium contrast study to identify small foreign bodies and to rule out GI ulceration.
- Perform a CBC and serum biochemical panel to rule out hepatic lipidosis and other systemic diseases.

Treatment

Surgical removal is the treatment of choice. If the ferret is debilitated, begin supportive therapy, and perform surgery as soon as possible.

- Surgery: Follow routine preoperative, operative, and postoperative procedures for gastrotomy or enterotomy (see Chapters 68 and 70). Ferret tissues are more delicate than those of a puppy or kitten of equivalent weight. Use 4-0 or 5-0 suture material to close the GI tract.
- Perform gastric biopsy to rule-out underlying H. mustelae infection, and other GI diseases. Perform biopsy of the liver.
- Evaluate the entire abdominal cavity prior to closure. Older ferrets often have concurrent diseases such as insulinoma or adrenal gland disease.
- The prognosis following gastrotomy is good with prompt therapy.

Prevention

Key Point Instruct owners to “ferret proof” the house if ferrets are allowed to roam. In particular, restrict access to rubber toys and rubber objects.

- To prevent trichobezoars, administer a feline hairball laxative product (2–4 cm) PO 2 to 3 times per week.
EPIZOOTIC CATARRHAL ENTERITIS

Epizootic catarrhal enteritis (ECE, “green slime disease”) is a highly infectious diarrheal disease that first appeared in 1993.

Etiology

The etiological agent is thought to be a coronavirus. ECE can spread rapidly through a ferret population, often affecting 100% of ferrets within 48 hours. Histological examination of intestinal biopsy samples reveals lymphocytic enteritis with villous atrophy and blunting, and degeneration of the apical epithelium.

Clinical Signs

- The history often includes recent exposure of an older ferret to a new or young ferret that appears healthy. Often within 48 hours the older ferret becomes anorexic and lethargic.
- Four clinical syndromes are typically seen:
  1. ECE may cause relatively mild diarrhea that lasts several days in young ferrets with no underlying disease.
  2. ECE may cause severe diarrhea lasting for several days that may be followed by an acute onset of severe bloody diarrhea in older ferrets or ferrets with concomitant disease. Anemia may develop as a sequelae.
  3. A wasting disease with abnormal stools that have the appearance of bird-seed or of being grainy. These stools may develop in ferrets that initially appear to have recovered from the diarrheal phase.
  4. Voluminous green, watery diarrhea and occasional vomiting followed by chronic wasting may occur in some ferrets.
- The clinical course of disease can be prolonged in some ferrets, and may last weeks to months. Affected ferrets typically appear to recover, but continue to have persistent, intermittent diarrhea.

Treatment

- No one specific treatment is consistently effective.
- Supportive care, including fluid therapy and nutritional support, is very important in the treatment of ECE.
- Treat sick ferrets with aggressive fluid therapy. Administer fluids IV, IO, PO, or SC depending on the ferret’s status.
- Administer broad-spectrum parenteral antibiotics.
- Feed a bland, high-calorie diet, such as a mixture of Science Diet A/D (Hills Science Diet, Topeka, KS) mixed with Deliver 2.0 (Mead Johnson Nutritional). Intestinal adsorbents or protectants (e.g., Kaopectate, UpJohn) may help in some ferrets.
- Loperamide (Imodium A-D, McNeil Consumer) (0.2 mg/kg) or (1 ml/kg) PO bid every 1 to 3 days may be helpful.
- Administration of prednisone (1 mg/kg) PO bid every 14 days may alleviate the chronic, intermittent diarrhea in ferrets with long-term symptoms.
- The disease may recur in previously affected ferrets; an asymptomatic carrier state appears to be possible.
- Do not house ferrets that have had ECE with ferrets that have not had the disease.

ROTAVIRUS

- Rotavirus has been associated with several outbreaks of diarrhea and high mortality in ferret kits 2 to 6 weeks of age; it is often referred to as “ferret kit disease.”
- Rotavirus also causes diarrhea in the young of several other species, including humans, cattle, swine, sheep, and rats.
- In adult ferrets, rotavirus infection is rarely fatal, but may cause bright green mucoid diarrhea that lasts for several days.
- There is no readily available antemortem test for the rotavirus infection; rotavirus particles can be identified in feces by electron microscopy.

Treatment

- Treatment consists of supportive care. Administer fluids, antibiotics, and nutritional support.

SALMONELLA

Salmonellosis is rare in the ferret, and is typically associated with exposure to contaminated raw meat and meat by-products. Salmonella typhimurium, S. newport, and S. choleraesuis may be associated with clinical disease.

- Clinical signs include anorexia, lethargy, fever, and diarrhea (usually bloody). Conjunctivitis and anemia have also been reported.
- Diagnosis is based on clinical signs and a positive fecal culture. Multiple fecal samples must be collected, and selective media is used for culture.
- Treatment includes aggressive supportive care and antibiotic therapy.
- Ferrets may be presented in shock. IV fluids and administration of rapidly acting intravenous corticosteroids may be necessary for treatment of these patients.
- Other details of salmonellosis, including its public health significance, are discussed elsewhere in this text.
EOSINOPHILIC GASTROENTERITIS

Eosinophilic gastroenteritis (EGE) is an inflammatory bowel disease that occurs in ferrets and other animals.

Etiology

No specific etiological agent has been identified in the ferret, but food allergy is implicated in humans and other animals.

Clinical signs

- Chronic diarrhea with or without mucus or blood, and weight loss are the most common signs. Inappetence, intermittent vomiting, and skin lesions may be seen as well.
- On physical examination, the mesenteric lymph nodes may be enlarged and the intestines may feel thickened.
- A marked peripheral eosinophilia is often present on the CBC differential.

Diagnosis

- Presumptive diagnosis is based on history, clinical signs, physical examination findings, the presence of a peripheral eosinophilia, and/or the presence of eosinophils on fecal cytology.
- Definitive diagnosis is based on histological examination of intestinal biopsy specimens. Mild to extensive eosinophilic infiltration of the mucosa, submucosa, and muscularis of the stomach and small intestines are noted. Focal eosinophilic granulomas maybe identified in the mesenteric lymph nodes.

Treatment

- Treatment is similar to that described for treatment of dogs and cats.
- Begin corticosteroid therapy with prednisone (1.0–2.5 mg/kg) PO sid every 14 days. Perform a recheck examination and CBC 2 weeks after the last dose. If the ferret has improved clinically and the peripheral eosinophilia is resolving, decrease the prednisone dose by 50% every 14 days, and recheck again. Continue the prednisone taper at 2-week intervals until the ferret is tapered to the lowest possible dose, or withdrawn from the steroids altogether.
- Although food allergy has not been identified as a definitive etiological cause of EGE, changing the ferret to a hypoallergenic diet, such as a feline lamb and rice-based diet may be helpful in resolution of signs.
- There have been reports of ferrets with peripheral eosinophilia (up to 40%), and erythema and crusting of the feet, ears, and face. Histological lesions in biopsy specimens from affected skin were consistent with allergic dermatitis. These ferrets were treated with corticosteroids, and did respond to treatment. One also responded to diet change.

INFLAMMATORY BOWEL DISEASE

Inflammatory bowel disease (IBD) can occur in the ferret.

Etiology

The etiology is unknown; dietary factors, hypersensitivity reactions, or an immune-mediated cause have been considered.

Clinical Signs

- Clinical signs can be subtle and include diarrhea; nausea; occasional vomiting; soft, malformed stools that resemble bird seed; and weight loss. These signs often resemble ECE, EGE, and Helicobacter gastroenteritis.
- Affected ferrets are often young or middle-aged adults.
- Elevation of liver enzymes and serum globulins may be noted on serum biochemistry analysis.
- Lymphocytosis may be noted on the CBC.

Diagnosis

- Diagnosis is based on the history, clinical signs, and diagnostic work-up.
- Definitive diagnosis is made by histological examination of gastric and intestinal biopsy samples. Mild to severe lymphoplasmacytic gastritis and enteritis are noted on histopathology.

Treatment

- Administer corticosteroids such as prednisone (1.0–2.5 mg/kg) PO sid every 14 days initially, and taper in a manner similar to that described for EGE. Some ferrets respond poorly to steroid therapy.
- Azathioprine (Imuran, Prometheus Laboratories, San Diego, CA) (0.9 mg/kg) PO q24–72h may be used as an alternative to steroid treatment.
- Hypoallergenic diets may offer some benefit.

PROLIFERATIVE BOWEL DISEASE (PBD)

Proliferative bowel disease (PBD) in ferrets was first reported in 1982, and is similar to the PBD that occurs in swine and hamsters. PBD was a commonly encountered disease in the late 1980s and early 1990s, but is relatively uncommon now.
Etiology
PBD is caused by *Lawsonia intracellularis*, an intracellular bacteria that cannot be propagated by routine culture methods.

Clinical Signs
- This disease affects primarily young ferrets 4 to 14 months of age.
- Acute and chronic forms of the disease can occur.
- Diarrhea is present and often contains mucus and blood. Defecations are frequent and small; ferrets often cry out when they defecate. The rectum may be partially prolapsed.
- Other signs include lethargy, depression, inappetence, weight loss, dehydration, and pyrexia.
- Neurologic signs such as ataxia and muscle tremors may be present.
- The intestines may feel firm or thickened on abdominal palpation.

Diagnosis
- A tentative diagnosis of proliferative bowel disease is based on clinical signs and physical examination. Definitive diagnosis requires intestinal or colonic biopsy, but this rarely is warranted because response to therapy usually is good if initiated early.
- A polymerase chain reaction (PCR) assay specific for the swine isolate, and an indirect fluorescent antibody test (IFA) are available.
- Necropsy lesions include gross thickening and discoloration of the small intestine and/or colon. Ridges of proliferative tissue that are distinct from normal adjacent tissues are present on the mucosal surface.
- Histological examination of biopsy samples or necropsy specimens typically demonstrate epithelial proliferation, hypertrophy of the muscularis, and infiltration of the bowel wall with monocytic or granulocytic inflammatory cells. Silver-stained tissues reveal intracellular, comma-shaped organisms in crypt epithelial cells. Glandular hyperplasia consisting of irregular, branching proliferative glands that lack goblet cells, and necrotic debris may be identified in the crypts. Severe glandular hyperplasia may resemble neoplasia and can metastasize.

Treatment
- Treat mild cases on an outpatient basis.
- Hospitalization for supportive care (fluid therapy, nutritional support) may be necessary when severe disease is present.
- Administer Chloramphenicol (50mg/kg) q12h, PO, IV, IM, or SC as the drug of choice. Treat for at least 2 weeks; longer therapy often is necessary to prevent relapse.
- Metronidazole (20mg/kg) q12h, PO may be effective.

Prevention and Prognosis
- The prognosis is good with timely therapy.
- Some ferrets improve temporarily and then relapse at the end of the treatment period. Use a long-term course of antibiotic therapy in these animals.

RECTAL PROLAPSE
- Rectal prolapse is usually a disease of young ferrets, and is often associated with diarrhea.
- Possible causes of rectal prolapse include colitis, diarrhea, GI parasitism (e.g., coccidiosis), PBD, and other diseases that may cause straining or diarrhea.
- Other differentials include GI lymphoma, benign intestinal polyps, and postoperative complications of anal gland removal.
- Perform direct fecal and fecal flotation tests to screen for parasites.
- Medical treatment is similar to that described for other species. Administer anthelmentics and antibiotics when indicated.
- The prolapse may resolve without surgical intervention when the underlying disease process is resolved.
- Surgical correction is usually unnecessary (see below).
- If indicated, perform a biopsy of the prolapsed tissue to rule out lymphoma.

Surgical Therapy
- Flush the prolapsed tissues with sterile saline and replace them into the rectum.
- Place a purse-string suture in the anus with a small opening to allow passage of feces. Keep the purse-string suture in place for 2 to 5 days.
- In ferrets with chronic prolapse, surgery may be necessary to reduce the size of the anal opening. Excise a small triangular wedge of anal mucosa and routinely close the defect by suturing. Alternatively, consider abdominal exploratory surgery and colopexy (see Chapter 75).

ANAL SAC ABSCESS
- Clinical signs and physical examination findings in ferrets with an abscessed anal sac are the same as those described in dogs and cats.
- The recommended treatments include antibiotic therapy, lancing and drainage of the abscess, or surgical removal of both anal sacs (see Chapter 75).
Anal Sacculectomy

Anal sacculectomy is performed as a treatment for anal sac abscesses, or to decrease the musky “ferret” odor. For odor reduction, neutering should be performed simultaneously because the apocrine, perianal, sebaceous, and scent glands in the skin are under hormonal control and contribute to the overall musky odor. Some clinicians believe that neutering is sufficient to decrease odor and that routine anal sacculectomy should be discouraged.

Surgical Technique

1. Grasp the anal sac duct and hold it closed with mosquito forceps. Make a circumferential skin incision around the duct opening.
2. Apply gentle caudal traction to the anal sac, and use a scalpel blade or gauze to tease away the surrounding fascia.
3. Leave the surgical sites open, and allow to heal by second intention.

Alternative Technique

1. Make small, arc-like incisions just lateral to the duct openings.
2. Dissect the subcutaneous tissues bluntly to reveal the neck of the anal sac; grasp the opening and hold it closed with mosquito forceps.
3. Dissect the sac free of surrounding tissues, using gentle traction.
4. Do not suture the incisions.

Urogenital System

REPRODUCTIVE SYSTEM

Characteristics of the Normal Ferret Reproductive System

Ferrets reach sexual maturity during the first breeding season after birth. The breeding season runs from March to August under natural lighting conditions.

Males

- The opening of the prepuce is located just caudal to the umbilical area.
- Males (hobs) have a J-shaped os penis.
- During the breeding season (March–August), testicle size is twice that noted in the fall and winter months.
- Prostatic tissue is located at the base of the urinary bladder and surrounds the urethra. Prostatic disease associated with adrenal gland disease may occur in middle-aged and geriatric male ferrets (see “Adrenal Gland Disease” and “Prostatic Disease”).

Females

- Female ferrets (jills) are seasonally polyestrous and are induced ovulators. Ovulation typically occurs 30 to 40 hours after mating.
- The vulva is located in the perineal region ventral to the anus. In non-estrous females the vulva is small, and looks like a slit; during estrus (or when adrenal gland disease is present), the vulva becomes swollen and is easily visualized.
- If mating is unsuccessful, pseudopregnancy results and lasts 41 to 43 days.
- Approximately 50% of females remain in estrus if they are not bred. The resultant prolonged elevation of serum estrogens can cause bone marrow toxicity and pancytopenia (see the discussion of anemia under “Hematopoietic System” in this chapter).
- Submit blood for a CBC and platelet count if the ferret has been in estrus for more than 28 days.
- Termination of estrus is recommended (see “Termination of Estrus in the Hematopoietic”).

Castration

- Most pet male ferrets in the United States have already been neutered prior to 8 weeks of age.
- Castrate intact male ferrets at 6 to 8 months of age in order to reduce aggressive behavior and odor.
- Castration is performed using techniques similar to those used in cats (see Chapter 87).
- Make an incision in the scrotum over each testicle.
- Remove the testicles using an open or closed technique.
- Incisions may be left open to heal by second intention.

Ovariohysterectomy

- Most pet female ferrets in the United States have already been spayed prior to 8 weeks of age.
- Spaying intact female ferrets is recommended to prevent estrogen-induced bone marrow hypoplasia.
- Ovariohysterectomy is similar to the procedure performed in cats (see Chapter 91).
- The ventral midline incision is made approximately 1 cm caudal to the umbilicus, and may be extended as necessary.
- The uterus is bicornuate, and is located dorsal to the bladder.
- Ovarian vasculature may be difficult to locate due to the large amount of body fat typically present in this region.

Pyometria/Metritis

- Pyometra and metritis are uncommon in pet ferrets in the United States because they are usually spayed prior to being sold as pets.
• Clinical signs may include anorexia, lethargy, pyrexia, and vulvar discharge. Polyuria and polydipsia are not usually noted.
• Persistent estrus may predispose ferrets to pyometra.
• Preoperatively perform a CBC and a serum biochemical analysis to rule out estrogen-induced bone marrow hypoplasia (see “Anemia” in “Hematopoietic System”).
• Provide appropriate supportive care pre- and postoperatively.
• Perform ovariohysterectomy when the patient is stable (see Chapter 91).
• Start the ferret on broad-spectrum antibiotic therapy preoperatively, and continue postoperatively for 10 to 14 days. Use broad-spectrum antibiotics. Organisms commonly associated with pyometra include Staphylococcus spp, Streptococcus spp, Corynebacterium spp, and E. coli.

Vulvar Swelling in Spayed Females
• Vulvar swelling is an external sign of estrus in female ferrets.
• In a spayed female, a swollen vulva indicates a remnant of ovarian tissue, or another source of estrogens and estrogen precursors such as adrenal gland disease (see “Adrenal Gland Disease”).
• Ovarian remnants typically induce signs of estrus in ferrets younger than 2 years of age.
• Administer HCG (100 IU) IM. Vulvar swelling should subside if an ovarian remnant is present. If no changes occur, adrenal gland disease is probably the cause of the clinical signs.
• Perform exploratory laparotomy to remove the ovarian remnant (see Chapter 91). Evaluate for uterine remnants and adrenal gland disease as well.
• Preoperatively evaluate a CBC to rule out estrogen-induced bone marrow hypoplasia.

Pregnancy Toxemia
• Pregnancy toxemia is a potentially life-threatening condition that occurs in late pregnancy. Primiparous females are most commonly affected.
• The disease results in high mortality of jills and kits.
• Toxemia can be induced if an accidental fast occurs in the last week of gestation.
• Pregnancy toxemia may also develop in primiparous jills that are carrying large litters due to nutritional compromise induced by the size of the gravid uterus and the resultant reduced capacity of the stomach.
• Advise owners that pregnant jills must have access to food and water at lib during pregnancy.
• Suspect pregnancy toxemia if acute lethargy develops in the last week of gestation. Other clinical signs include dehydration, melena, hypoglycemia, ketonuria, and azotemia.
• Affected ferrets usually are presented in an acute state of shock.

• Treatment includes aggressive supportive care including IV or IO fluids containing dextrose. Perform an immediate cesarean section (see Chapter 91).
• Postoperative care includes continued supportive care, including frequent feedings of high-caloric critical care diets.
• Jills that survive pregnancy toxemia often do not produce milk. Kits can be difficult to hand rear; if a foster jill is not available, attempts may be made to hand rear the kits using a kitten milk replacer (see below). Kits born before 40 days of gestation often do not survive.
• The prognosis is usually poor, even with aggressive treatment.

Mastitis
• Do not breed females with a history of mastitis.
• Abrasions to the mammary tissue and nipples can cause mastitis. Prevent trauma from occurring by providing a large nest box opening with smooth edges that allows the jill to pass through easily.
• Mastitis may be acute or chronic.
• Acute mastitis typically occurs immediately after whelping or during the third week of lactation.
• Affected glands appear swollen, firm, red to purple in color, and are painful. Gangrene can develop within hours of clinical signs.
• Treatment must be aggressive. Administer broad-spectrum antibiotics, and apply hot packs to the affected area 2 to 3 times per day for 2 days. Debride necrotic tissue if present. Provide supportive care and analgesic therapy.
• Submit a sample for bacterial culture and sensitivity testing. Modify antibiotic therapy based on test results.
• If there is no clinical response to medical therapy in 2 days, or if gangrene rapidly develops, consider surgical removal of the affected mammary tissue. Because of the potential for severe toxicity and life-threatening disease, do not delay surgery if gangrene is already present, or if there is no improvement with medical therapy.
• If the jill continues to lactate, leave the kits with her. Supplement feed the kits with a kitten milk replacer if necessary. Do not foster the kits with another jill because this may result in mastitis in the foster jill.
• Ingestion of infected milk may cause gastroenteritis in the kits; kits may need to be treated with antibiotics as well.
• Chronic mastitis is often difficult to diagnose. The affected jill often appears normal, while the kits lose weight or fail to thrive.
• Mammary glands appear firm but are not painful or discolored; often the glands are presumed to be full of milk.
Foster Care of Kits
- Hand-rearing kits from birth is difficult. Prognosis is poor for survival.
- It may be necessary to provide supplemental feeding for kits if the jill’s milk production is reduced, or if the litter size is large.
- Whenever possible, foster kits with another lactating jill. Most jills will accept kits of any size or age.
- Kits require a milk supplement that contains a high fat content (20%). Kitten milk replacers mixed with cream may be used.
- Feed kits as much as they will eat 4 times per day with a dropper or small pet nurser.
- Begin to mix solid food with the enriched milk replacer when kits are 4 weeks of age. This mixture may be offered in a shallow dish or bowl.
- Kits may be weaned onto a solid diet at 5 to 6 weeks of age. Feline or ferret growth diets are recommended.

URINARY SYSTEM
Characteristics of the Normal Ferret Urinary System
- The right kidney lies cranial to the left kidney. The cranial end of the right kidney often lies under the caudate lobe of the liver.
- The bladder is small, and can hold up to 10 ml of urine.
- Male ferrets have a small prostate gland that surrounds the urethra at the base of the bladder.
- Urinalysis:
  - The normal urine pH is 6.0 for ferrets on a meat-based diet.
  - Normal values for urine-specific gravity have not been reported.
  - There is evidence that proteinuria may be normal in ferrets (7–33 mg/dl in males; 0–32 mg/dl in females) and that bilirubinuria can occur in the absence of liver disease.

Renal Disease
Renal disease is not common in ferrets, but may occur.

Clinical Signs
- Clinical signs are similar to those described in other animals, and include ataxia, bruxism, halitosis, hindlimb weakness, inappetence, melena, mucus membrane ulceration, polyuria/polydipsia, vomiting, and weight loss.
- Physical examination findings may include cachexia, dehydration, irregularity in the shape and size of the kidneys, pale mucous membranes, and oral ulceration.

Diagnosis and Treatment
- Diagnosis is based on clinical signs, physical examination, and CBC, serum biochemical analysis, and urinalysis results.
  - Key Point Hyperphosphatemia, hypocalcemia, and high BUN may be noted on serum biochemical analysis. Serum creatinine concentration is often normal or only moderately elevated.
- Treatment should address the underlying cause, if possible.
- Nonspecific treatment includes fluid therapy, nutritional supportive care, and antibiotic therapy based on culture and sensitivity when indicated.
- Prognosis is guarded, depending on laboratory findings and response to treatment.

Renal Cysts
Unilateral or bilateral renal cysts are relatively common in ferrets (see Chapter 77 for a description of this disease in dogs and cats). The condition is usually an incidental finding in middle-aged and older ferrets, although clinical signs associated with this condition can occur at any age.

Etiology
- The cause of renal cysts in the ferret is unknown.
- Heredity does not appear to be a factor. Renal cysts are not associated with hepatic or biliary cysts.
- Renal cysts typically present as one or more smooth masses on the surface of the kidney. On abdominal palpation affected kidneys feel smoothly enlarged or irregular.
- Polycystic disease is unusual in the ferret. When present, affected kidneys appear rough and irregular; multiple cysts are often distributed throughout the renal tissue. Cysts may be present in other organs as well.

Clinical Signs
- Usually there are no clinical signs associated with renal cysts.
- Rarely, there may be enough disruption of normal renal parenchyma to lead to renal failure, and subsequent clinical signs.

Diagnosis
- Palpate the kidneys for irregular shape.
- Perform a CBC, serum biochemical profile, and urinalysis.
- Abdominal radiography usually is not helpful unless the kidneys are very irregular.
Perform an abdominal ultrasound to detect renal cysts, to evaluate renal architecture, and to rule out other conditions such as renal neoplasia.

Intravenous pyelography or nuclear scintigraphy may be used to evaluate renal function.

Renal cysts may be an incidental finding during abdominal surgery.

**Treatment**

- There is no specific treatment for renal cysts. No treatment is necessary in asymptomatic animals.
- Monitor affected ferrets by periodic abdominal palpation, serum biochemical profile, urinalysis, and ultrasound, if indicated.
- If an affected kidney becomes very large, consider unilateral nephrectomy (if the opposite kidney is functional) (see Chapter 78).
- Symptomatic ferrets may be managed using the same supportive care methods used in dogs and cats with chronic renal failure.
- The prognosis is grave for ferrets in renal failure.

**Hydronephrosis**

- Hydronephrosis is uncommon in ferrets. Iatrogenic hydronephrosis may occur as the result of inadvertent ligation of a ureter during ovariohysterectomy (see Chapter 77 for information about hydronephrosis in dogs and cats).

**Cystitis**

- Bacterial cystitis without urinary calculi is rare in pet ferrets. Follow treatment protocols for cystitis in dogs (see Chapter 79).

**Urolithiasis**

Urinary calculi was a common cause of stranguria in ferrets at one time; improvement in the quality of ferret diets has decreased the incidence of calculi. Calculi are usually composed of calcium oxalate or struvite (magnesium ammonium phosphate hexahydrate). Cysteine calculi also have been reported.

**Etiology**

- The cause of urinary calculi is unknown; however, diet is believed to be a factor.
- Diets containing plant proteins or poor quality meat-based proteins may be associated with the development of urinary calculi. Urolithiasis is uncommon in ferrets maintained on a high-quality feline or ferret diet containing high-quality animal-based proteins.
- Other factors may include urinary tract infection, metabolic, genetic, and congenital factors.

**Clinical Signs**

Clinical signs depend on the location of the urolith(s) and may include dysuria, stranguria, hematuria, persistent wetness in the perineal region, and frequent licking of the perineum.

- Urethral calculi may cause obstruction in both male and female ferrets.
- Ferrets with urethral obstruction often strain and cry as they attempt to urinate.
- If complete obstruction is present ferrets often appear lethargic and anorexic, and may not demonstrate obvious signs of dysuria.

**Diagnosis**

- Palpate the bladder to identify cystic calculi. The urinary bladder wall may be thickened; in ferrets with urethral obstruction, the bladder is distended and firm.
- Obtain abdominal radiographs to confirm the presence of radiopaque urinary calculi. Cysteine calculi are not radiopaque and require contrast radiography or ultrasonography for diagnosis.

**Treatment**

- Stabilize non-obstructed ferrets by providing supportive care, fluids, analgesics, and antibiotics (if indicated) prior to performing cystotomy to remove the urolith(s).
- Cystic calculi may be removed surgically via cystotomy; the procedure is similar to that used in cats and dogs (see Chapter 80). Close the bladder wall with 4-0 or 5-0 absorbable sutures.
- Submit a urolith sample for analysis and bacterial culture/sensitivity testing.
- Administer antibiotics for a minimum of 10 to 14 days. Use results of follow-up urinalysis, and urine culture/sensitivity testing to determine when to discontinue antibiotic therapy.
- Begin conversion to a high-quality–animal, protein-based feline or ferret diet. Urinary acidifies are not usually necessary once the ferret is on a high-quality animal, protein-based diet, since this diet alone will cause the urine to be acidic.
- Feline calculi-dissolving diets and preventative diets may be offered to ferrets; however, many ferrets do not find these diets palatable.
Renal calculi can often be managed medically by administering antibiotic therapy and changing the diet.

**Urethral Obstruction**

* Key Point The bladder is very fragile in ferrets. Handle ferrets with obstruction gently to avoid bladder rupture.

- Urinary obstruction in the male ferret can be difficult to manage. Catheter placement is challenging due to the small size of the urethra and the J-shaped os penis. (See Urinary Catheterization in the Techniques section of this chapter.)
- To facilitate placement of the urinary catheter, empty the bladder via cystocentesis prior to catheterization. Submit urine samples for urinalysis and bacterial culture/sensitivity testing.
- Use either a ferret urinary catheter (Slippery Sam Ferret Urinary Catheter, Cook Veterinary Products), a standard tom cat catheter, or a 3.5-Fr red rubber catheter for catheterization.
- Inhalant anesthesia with isoflurane or sevoflurane is strongly recommended to facilitate catheter placement.
- If the urinary catheter placement is not successful, consider emergency cystotomy, and attempt to perform anterograde flushing of the urethra via the cystotomy site.
- Perineal urethrostomy may be considered if cystotomy is unsuccessful (see Chapter 82).

**Prevention**

- Feed a high-quality, animal protein-based feline or ferret diet.

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**Prostatic Disease/Prostatic Cysts**

Prostatic disease and subsequent urethral obstruction is a potentially life-threatening condition of middle-aged and geriatric male ferrets. This condition typically occurs in association with adrenal gland disease.

**Etiology**

- Prostatic disease and prostatic cyst formation are presumed to be the effect of excessive androgens on the prostate. Excessive androgen production occurs with adrenal gland disease.
- Squamous metaplasia of prostatic glandular epithelium occurs and may subsequently lead to the development of cysts ranging in size from 1 to 6 cm or larger. Secondary bacterial infection and abscessation may occur.
- Prostatic abscesses associated with transitional cell tumor of the bladder, prostatic seminoma, and prostatic carcinoma have also been reported in the ferret, but are rare.

**Diagnosis**

**History and Clinical Signs**

- Clinical signs associated with prostatic disease may include symptoms associated with a urinary tract infection, urethral obstruction, or urinary incontinence.
- Signs of adrenal gland disease are often present (see “Adrenal Gland Disease”).

**Physical Examination**

- On physical examination, a large, firm, often painful caudal abdominal mass is usually palpable. With careful palpation, this mass is found to be bilobed, representing the urinary bladder and a cystic structure. Ferrets with mild to moderate prostatic disease may appear to have a normal-sized prostate on abdominal palpation, yet are still symptomatic.

**Diagnostic Tests**

It is important to remember that adrenal gland disease is usually the cause of prostatic disease. Perform a complete diagnostic work-up that includes whole-body radiography, CBC, serum biochemistry analysis, and urinalysis. A plasma steroid hormone assay, and abdominal ultrasound may be indicated as well.

- Obtain abdominal radiographs; prostatic enlargement or prostatic cysts appear as mass lesions dorsal to the bladder.
- Perform abdominal exploratory surgery for a definitive diagnosis.

**Treatment**

- Address urethral obstruction if present (see “Urolithiasis”).
- Manage medically until the ferret is stable for adrenalectomy and surgical drainage of the cysts.
- Medical management includes maintenance of urinary catheterization for several days, administration of fluids, antibiotic therapy, anti-inflammatory and analgesic therapy, and nutritional support as needed.
- Consider administration of an androgen receptor blocker (see “Adrenal Gland Disease”).
- Consider administration of leuprolide acetate 30-day depot formulation (Lupron Depot, Bristol-Myers-Squibb Oncology, Princeton, NJ) (250 mg/kg) IM; prostatic tissue shrinkage may occur within 48 hours in some individuals. Some ferrets have been maintained successfully on monthly injections of this drug, although results are highly variable.
- Perform adrenalectomy and drainage of the cysts. Large cysts may require debulking.
Perform bacterial culture and sensitivity testing of the cyst contents.

Key Point Omental pull-through procedures and marsupialization have been described as means of prostatic abscess management in the ferret. These procedures should be used with some caution. Prostatic abscesses and prostatic cysts can be difficult to differentiate from paraurethral cysts. Paraurethral cysts communicate with the urethra or bladder neck. Consider performing contrast radiography to determine if there is communication between the cyst/abscess and the bladder prior to performing these procedures.

Administer postoperative antibiotic therapy for a minimum of 10 to 14 days, along with androgen receptor blockers or leuprolide acetate.

Base the decision to discontinue antibiotic therapy and androgen receptor blocker/leuprolide acetate therapy by monitoring changes on physical examination, follow-up radiography, and follow-up urinalysis.

The long-term prognosis is good if prostatic changes regress, and if subsequent adrenal gland disease does not occur in the remaining adrenal gland.

Some ferrets may need to be maintained on androgen receptor blockers or leuprolide acetate indefinitely.

Paraurethral cysts are thin-walled single or multiple cysts present on the dorsal aspect of the bladder and proximal urethra. These cysts appear to also be associated with adrenal gland disease and can cause urethral obstruction.

It is important to differentiate between prostatic cysts and paraurethral cysts when planning the surgical protocol.

Paraurethral cysts have been reported in male and female ferrets. Clinical signs are similar to those described for prostatic disease, and include symptoms associated with a urinary tract infection, urethral obstruction, urinary incontinence.

Clinical signs of adrenal gland disease are usually present (see “Adrenal Gland Disease”).

**Diagnosis**

**Physical Examination**

A large, firm caudal abdominal mass is often palpable dorsal to the bladder, just cranial to the pelvic inlet.

**Diagnostic Tests**

- Perform a complete diagnostic work-up that includes whole-body radiography, CBC, serum biochemical analysis, and urinalysis.
- Because adrenal gland disease is usually the underlying etiology, consider performing a plasma steroid hormone assay.
- Radiographically, paraurethral cysts appear as mass lesions dorsal to the bladder.
- Ultrasonography may be useful in evaluation of the paraurethral cysts and adrenal glands.

**Treatment**

- Surgical drainage and debulking of the cysts is the treatment of choice.
- Marsupialization is an alternative, but may lead to a formation of a permanent cystotomy.
- Do not perform an omental pull-through procedure.

**Supplemental Readings**

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