Surgery of the Adrenal and Pituitary Glands

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Adrenalectomy is the removal of one or both adrenal glands. Hypophysectomy is the removal of the pituitary gland (hypophysis). Hyperadrenocorticism (HAC) is a multisystemic disorder caused by an excess of glucocorticoids. Cushing disease refers to HAC caused by a pituitary adenoma. Addison disease is caused by a deficiency of glucocorticoids or mineralocorticoids or both.

PREOPERATIVE MANAGEMENT

Adrenocortical insufficiency may be primary, secondary to other diseases, or iatrogenic (i.e., due to administration of glucocorticoids, progestins, or drugs that suppress the adrenal glands [e.g., trilostane, mitotane, etomidate]). The history should include the dosages of glucocorticoids or other drugs given, type of glucocorticoids, duration of administration, and time since the last dose. It is easier to inhibit secretion of glucocorticoids than mineralocorticoids (see the discussion of anatomy on p. 587). When glucocorticoid secretion is severely suppressed, the patient may experience depression, inappetence, lethargy, collapse, and/or weakness without electrolyte abnormalities. If mineralocorticoid secretion is suppressed, hyponatremia, hyperkalemia, acidosis, and/or azotemia may occur. Diminished ability to retain sodium results in volume depletion, diminished cardiac output, and reduced vascular tone, which may cause acute vascular collapse. Gastrointestinal disturbances and prolonged vomiting may contribute to electrolyte abnormalities and volume depletion. Electrolyte concentrations should be corrected before surgery. Some dogs with hypoadrenocorticism are hypoalbuminemic. A protective steroid release normally occurs during surgery that prevents circulatory collapse; however, animals with hypoadrenocorticism may be unable to respond appropriately and often require glucocorticoid supplementation before and during surgery. When minor elective surgery is performed in animals with adrenocortical insufficiency, glucocorticoid therapy may be given intravenously before induction of anesthesia (Box 22.1). The same dose can be given intravenously or intramuscularly after recovery from anesthesia, and the animal is returned to its oral maintenance glucocorticoid therapy the day after surgery. A similar protocol is used for major surgery, except that glucocorticoid therapy is continued at approximately five times the maintenance dose for 2 to 3 days (Box 22.2). Normal maintenance doses are then reinstituted. Once the animal is eating, medications can be given orally (Box 22.3).

HAC is found primarily in dogs; it is rare in cats. Iatrogenic HAC is the most common type. Spontaneous HAC is usually caused by excessive pituitary secretion of adrenocorticotropic hormone (ACTH), resulting in bilateral adrenocortical hyperplasia or pituitary-dependent hyperadrenocorticism (PDH) (80%–90% of noniatrogenic cases). Functional adrenocortical tumors (adrenal-dependent hyperadrenocorticism [ADH]) are less common (10%–20% of noniatrogenic cases). Differentiation of PDH from ADH is potentially complex, and the reader is referred to a medical text for further information.

Patients with HAC are catabolic and often protein depleted; this may adversely affect wound healing. They may have connective tissue abnormalities and muscle wasting, resulting in a pot-bellied appearance, redistribution of fat, and thin, fragile skin. Pyodermas are common in affected dogs, which may cause postoperative suture line healing to be compromised. Affected dogs may pant because of their catabolic state, but intraabdominal fat deposition plus abdominal muscular weakness sometimes causes ventilatory abnormalities. Hypernatremia, hypokalemia, and alkalosis may be present; substantial abnormalities should be corrected before surgery. Concurrent abnormalities (e.g., congestive heart failure, diabetes mellitus [DM]) increase the patient’s anesthetic risk. Cardiovascular abnormalities may occur secondary to hypervolemia and hypertension; a thorough preoperative cardiac examination including blood pressure measurement is appropriate.

Animals with HAC are at increased risk for postoperative pulmonary thromboembolism (PTE). If hypercoagulability is suspected, preventive measures may be indicated before surgery. For prevention of PTE, animals may be started on heparin during surgery and continued on it postoperatively (see Box 29.1) or they may be administered clopidogrel (2–3 mg/kg per day in dogs); however, prospective studies are needed to determine the relative benefit of these therapies. Many animals with HAC have clinically silent urinary tract infection; therefore urine culture is indicated in all patients, regardless of urinalysis findings.

ANESTHESIA

A variety of anesthetic protocols may be used in adrenocortical-insufficient or hyperadrenal animals. Etomidate causes transient adrenal suppression and should be avoided in patients with hypoadrenocorticism and those in which postoperative hypoadrenocorticism is anticipated. Steroid replacement should be provided in animals showing signs of adrenal insufficiency. Maintenance of electrolyte and glucose concentrations is important. Glucocorticoid supplementation is often necessary in animals with adrenocortical insufficiency undergoing surgery (see previous
ABSTRACT
Organs of the endocrine system that may require surgical intervention are the pituitary gland, adrenal glands, thyroid and parathyroid glands, and the pancreas. Conditions affecting the endocrine system often result in abnormal production and release of hormones into the circulation. Excessive production of hormones, most often due to a neoplastic process, may require removal of the affected gland (e.g., adrenalectomy, thyroidectomy). Diseases of the pancreas can be challenging as they may be diffuse, or a neoplastic lesion requiring resection may be in an anatomically challenging area. Thorough diagnostic evaluation for suspected endocrinopathies is critical to determine the best mode of treatment.

KEYWORDS
adrenal gland
adrenalectomy
pituitary gland
hypophysectomy
hyperadrenocorticism
pheochromocytoma
pancreas
insulinoma
partial pancreatectomy
neoplasia
abscess
pseudocyst
gastrinoma
thyroid gland
parathyroid gland
hyperthyroidism
hyperparathyroidism
Perioperative prophylactic antibiotics are recommended for these patients.

**SURGICAL ANATOMY**

The adrenal glands are near the craniomedial pole of the kidneys (Fig. 22.1). The left adrenal is slightly larger than the right. The left gland lies ventral to the lateral process of the second lumbar vertebra; the right adrenal is more cranial, lying ventral to the lateral process of the last thoracic vertebra. Because of the proximity of the right adrenal to the caudal vena cava, surgical removal of neoplastic glands can be difficult. The phrenicoabdominal (cranial abdominal) vessels cross the ventral surface of the adrenal. The adrenal glands are composed of two functionally and structurally different regions. The outer cortex produces mineralocorticoids (e.g., aldosterone), glucocorticoids, and small quantities of androgenic hormones. Mineralocorticoids regulate sodium and potassium concentrations. Aldosterone causes transport of sodium and potassium through the renal tubular walls and also causes hydrogen ion transport.

**BOX 22.2** Protocol for Glucocorticoid Administration in Animals With Adrenocortical Insufficiency Undergoing Major Elective Procedures

1. Administer preoperative steroids as described in Box 22.1.
2. Administer dexamethasone (0.05–0.1 mg/kg IV) during surgery.
3. Taper the dexamethasone dose by 0.02 mg/kg (IV bolus) every 12 hours (but do not give <0.02 mg/kg).
4. As soon as the patient can reliably be given oral medications, switch to oral prednisolone (see Box 22.3).

**ANTIBIOTICS**

Animals with HAC are at increased risk of developing postoperative infection owing to high levels of circulating glucocorticoids. Discussion). Glucocorticoid therapy should be instituted before surgery in patients with HAC that are undergoing adrenalectomy. Because of the close association of the adrenals and the caudal vena cava, retraction of the caudal vena cava is often necessary for adrenalectomy. Vascular pressures should be closely monitored during surgery, and retraction should be done carefully to avoid obstructing venous return. Special anesthetic considerations are required for animals with pheochromocytomas to prevent complications associated with excessive secretion of catecholamines (see p. 594).

**SURGICAL TECHNIQUE**

Adrenalectomy is usually performed for adrenal tumors. Bilateral adrenalectomy for canine PDH is controversial and uncommonly performed, but it has been effective for feline PDH. One of two open approaches can be used, or alternatively a laparoscopic approach can be performed. A ventral midline approach allows the entire abdomen to be explored for metastasis and bilateral adrenalectomy to be performed with a single surgical incision.
if necessary. However, exposure and dissection of the adrenal may be difficult with this approach, particularly in large dogs. A paracostal incision provides better access to the adrenal gland but does not allow evaluation of the liver or other organs for metastasis. It may be considered in animals with unilateral lesions that have no evidence of metastasis on ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI). Concurrent DM might be a contraindication to bilateral adrenalectomy because lack of endogenous catecholamines may make it difficult to regulate the diabetes. For laparoscopic adrenalectomy, dogs are positioned in lateral recumbency, with the adrenal gland to be removed on the up side. Dogs may also be positioned in sternal recumbency with the thorax and pelvis elevated, such that the abdomen is not in contact with the surgical table. This allows for gravitational displacement of the abdominal viscera and better visualization of the affected adrenal gland.

Adrenalectomy via a Midline Abdominal Approach

Prepare the entire ventral abdomen and caudal thorax for aseptic surgery. Make a ventral midline abdominal incision that extends from the xiphoid cartilage to near the pubis. Identify the affected adrenal gland and carefully inspect the entire abdomen, including the other adrenal gland, for abnormalities or evidence of metastasis. Palpate the liver for evidence of nodularity and biopsy if indicated. Palpate the caudal vena cava near the adrenal glands for evidence of tumor invasion or thrombosis. If additional exposure is necessary for adrenalectomy, extend the incision paracostally on the side of the affected gland by incising the fascia of the rectus abdominis muscle and the fibers of the external abdominal oblique, internal abdominal oblique, and transversus abdominis muscles, respectively. Use self-retaining retractors to improve visualization of the abdominal cavity. Retract the liver, spleen, and stomach cranially, the kidney caudally, and the caudal vena cava medially to expose the entire adrenal gland. Identify the blood supply and ureter to the ipsilateral kidney, and avoid these structures during dissection. Ligate the phrenicoabdominal vein and divide it between sutures. Using a combination of sharp and blunt dissection, carefully dissect the adrenal gland from surrounding tissue (Fig. 22.2). Numerous vessels may be encountered. Obtain hemostasis with electrocautery, a vessel-sealing device, or with hemoclips. If possible, do not invade the adrenal capsule. Remove the adrenal in one piece, if possible, to reduce the chances of leaving small pieces of neoplastic tissue in the abdominal cavity. If tumor thrombosis is present in the caudal vena cava medially. Ligate the phrenicoabdominal vein and divide it between sutures. Carefully dissect the adrenal gland from surrounding tissue. Figure 22.2 To resect the right adrenal gland, retract the vena cava medially. Ligate the phrenicoabdominal vein and divide it between sutures. Carefully dissect the adrenal gland from surrounding tissue.

Adrenalectomy via a Paralumbar Approach

Place the patient in lateral recumbency with a rolled towel or a sandbag between the abdomen and the operating table. Make an incision just caudal to the 13th rib, extending it from the lateral vertebral processes to within 3 to 4 cm of the ventral midline (the incision will be approximately 10–14 cm long, depending on the animal’s size; Fig. 22.3). Incise the abdominal muscles individually and identify the adrenal gland cranial to the kidney. Retract the kidney ventrally and ligate any vascular structures that cross its surface. Dissect the gland free from surrounding tissue (Fig. 22.4). Suture each muscle layer of the paracostal incision in a continuous suture pattern of synthetic absorbable (i.e., 2-0 or 3-0) material. Close the skin and subcutaneous tissues routinely.

Laparoscopic Adrenalectomy

When no evidence of caudal vena cava involvement is found, laparoscopic adrenalectomy may be an option. Laparoscopic adrenalectomy has been described in veterinary patients; however, the procedure is challenging and requires careful patient selection. Removal of the right adrenal gland in dogs is especially challenging as the adrenal gland capsule can be continuous with the tunica externa of the caudal vena cava. Preoperative diagnostic imaging is important in the workup for adrenal masses and is the basis for deciding whether a laparoscopic approach might be feasible. The dimensions of the mass are vital, as are relationships to surrounding organs and vascular structures. Approximately 25% of adrenal neoplasms invade the caudal vena cava, phrenicoabdominal

FIG. 22.2 To resect the right adrenal gland, retract the vena cava medially. Ligate the phrenicoabdominal vein and divide it between sutures. Carefully dissect the adrenal gland from surrounding tissue.

FIG. 22.3 Adrenalectomy may be performed via a paralumbar approach. Place the animal in lateral recumbency with a rolled towel or sandbag between the abdomen and the operating table. Make an incision just caudal to the 13th rib, extending it from the lateral vertebral processes to within 3 to 4 cm of the ventral midline.
Keeping the adrenal capsule intact. Following completion of the space using dissecting forceps, electrocautery, or a vessel-sealing device, remove the vein. Carefully dissect the adrenal gland out of the retroperitoneal hemoclips or a vessel-sealing device to ligate the phrenicoabdominal vein and periadrenal tissue dorsolateral to the gland. Use large hemoclips or a vessel-sealing device to ligate any vascular structures before dissection, place the adrenal gland in a specimen retrieval bag and remove it from the body. Close the port sites routinely following evacuation of the pneumoperitoneum.

HEALING OF THE ADRENAL AND PITUITARY GLANDS

Because adrenal or pituitary biopsies are rarely performed, little information is available about the healing of these glands after surgery.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

HAC may cause delayed wound healing; therefore abdominal closure should be performed with strong, slowly absorbed, or nonabsorbable suture material (e.g., polydioxanone, polyglyconate, polypropylene, nylon). Self-retaining retractors, such as Balfour abdominal retractors, are recommended to improve abdominal visualization. Malleable retractors covered with moistened sponges are used to retract viscera from the adrenal glands. Hemostasis is easier to achieve with vessel-sealing devices, electrocautery, and hemoclips than with suture ligation of vessels (see Chapter 7).

POSTOPERATIVE CARE AND ASSESSMENT

After adrenalectomy, the patient’s hydration status and electrolyte balance should be monitored carefully and corrected as necessary. Bilateral adrenalectomy causes permanent adrenal insufficiency, and these animals require lifelong glucocorticoid (e.g., prednisolone) and/or mineralocorticoid (e.g., deoxycorticosterone) replacement (see Box 22.3). Animals should be closely monitored for hypoadrenocortical collapse. They are generally less likely to have an Addisonian crisis after they have been released to the owner’s care. Owners must be advised to watch for malaise, inappetence, vomiting, weakness, and other clinical signs suggesting decompensation. Temporary adrenal insufficiency occurs after unilateral removal of functional adrenal tumors because the tumor has suppressed the function of the contralateral adrenal. Glucocorticoids should be supplemented postoperatively (see Box 22.3) but may be discontinued when the remaining adrenal begins to function normally, as determined by the results of an ACTH stimulation test.

PTE is a potentially life-threatening complication of adrenal surgery, particularly in dogs with adrenal neoplasia. Sudden, severe postoperative respiratory distress may indicate PTE. Lung perfusion scans may help identify lung regions that are underperfused. To help prevent this have the dog walk every 2 to 3 hours to promote circulation. Sufficient analgesics should be provided so that the dog can be coaxed to walk 4 hours after it wakes up. Treatment with strict cage rest, oxygen, anticoagulants (e.g., clopidogrel, aspirin, heparin), and thrombolytic agents (e.g., streptokinase, tissue plasminogen activator) has been proposed in the past, but the value of these pharmacologic agents is uncertain (see Box 29.1). Animals treated for PTE with thrombolytics should be assessed frequently for evidence of hemorrhage, and the hematocrit should be checked every 2 hours. If the packed cell volume (PCV) drops or hemorrhage is noted, the thrombolytic infusion should be discontinued.

COMPLICATIONS

The main complications of adrenalectomy are hemorrhage, fluid and electrolyte imbalance, pancreatitis, wound infection, delayed veins, or renal vasculature; pheochromocytomas are more likely to invade than are adenocortical tumors. Vascular invasion is an indication for an open surgical approach. Ultrasonography and CT are used most often for preoperative imaging. Ultrasonography has a sensitivity and a specificity of approximately 80% and 90%, respectively, for detection of tumor thrombus. A 2015 study found excellent agreement between signs of vascular invasion in CT images and finding vascular invasion at surgery or necropsy with sensitivity, specificity, and agreement measuring 92%, 89%, and 94%, respectively. Animals with functional tumors causing clinical signs, and those with tumors measuring more than 2 cm that do not exhibit vascular invasion may be candidates for laparoscopic adrenalectomy. Animals that are systemically unstable and those that have uncontrolled metabolic or acid-base disturbance, uncontrolled coagulopathies, untreated severe arrhythmias, or hypertension should undergo an open approach. Animals that may be poorly tolerant of pneumoperitoneum (e.g., severe cardiorespiratory disease, diaphragmatic herniation) are also poor candidates for a laparoscopic approach. Vascular invasion of the mass into surrounding vessels and large masses (>7 cm) are indications for open adrenalectomy. An upper limit of 5 to 7 cm maximal diameter of the adrenal mass has been recommended for laparoscopic removal of adenocortical tumors. Place the animal in lateral recumbency with elevation of the vertebral column or sternal recumbency with the thorax and pelvic elevated. Use a three-port technique in the paralumbar fossa, caudal to the last rib, triangulating the approximate position of the adrenal gland. Place an additional port dorsally if additional retraction is needed. Isolate the adrenal gland by careful dissection of the peritoneum and periadrenal tissue dorsolateral to the gland. Use large hemoclips or a vessel-sealing device to ligate the phrenicoabdominal vein. Carefully dissect the adrenal gland out of the retroperitoneal space using dissecting forceps, electrocautery, or a vessel-sealing device, keeping the adrenal capsule intact. Following completion of the...
uncommon, nonfunctioning masses. Postoperative hemorrhage is usually associated with incomplete occlusion of small vessels surrounding an enlarged, highly vascular tumor. Judicious use of vessel-sealing devices, electrocautery, and hemoclips helps prevent this complication. Delayed wound healing is often encountered in animals with HAC because of the adverse effect of steroids on wound healing; therefore care should be taken with abdominal closure in these animals (see previous discussion). Strong monofilament absorbable (e.g., polydioxanone or polyglyconate) or nonabsorbable (e.g., polypropylene) suture should be used.

**SPECIAL AGE CONSIDERATIONS**

Animals with adrenal neoplasia are usually older and often have concurrent abnormalities, such as hypertension or cardiovascular abnormalities; therefore extreme care should be exercised during anesthesia. These animals also require extensive postoperative monitoring. If the animal is debilitated, anorexic, or vomiting, placement of an enteral feeding tube during surgery (see p. 101) or parenteral nutrition (see p. 94) is advised.

**SPECIFIC DISEASES**

**ADRENAL NEOPLASIA**

**DEFINITIONS**

Adrenal carcinomas are autonomously functioning, malignant tumors of the adrenal cortex; adrenal adenomas are benign adrenocortical tumors. Pheochromocytomas are catecholamine-secreting tumors of the chromaffin tissue, which usually arise in adrenal medullary tissue. Pheochromocytomas are also known as paragangliomas. “Incidentalomas” (incidental adrenal masses) are adrenal masses that are fortuitously found during imaging in animals that are not suspected of having adrenal disease. They may be carcinomas or pheochromocytomas, or they may be undefined, nonfunctioning masses.

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

Adrenal gland tumors include adrenal adenomas, carcinomas, and pheochromocytomas. Most adrenal tumors are nonfunctional, and clinical signs are caused by local invasion of the tumor into surrounding tissue, distant metastases, or both. Functional cortical tumors secrete excessive amounts of cortisol or aldosterone. In dogs, functional tumors typically secrete cortisol, which inhibits pituitary ACTH secretion and causes atrophy of the contralateral adrenal. Adrenocortical adenomas and carcinomas appear to occur with equal frequency. They are usually unilateral, although bilateral adrenocortical neoplasia rarely occurs. History, physical examination, and laboratory findings do not differentiate between bilateral and unilateral adrenal neoplasia. Ultrasonographic evaluation of the adrenals often identifies adrenomegaly on one side and adrenal atrophy on the other, which localizes the tumor. Colonic perforation is a rare sequela of excessive glucocorticoid secretion. Corticosteroids may inhibit collagen synthesis and increase collagen breakdown. They may also cause breakdown of the mucosal barrier and inhibit normal immune responses.

**NOTE** Most animals with hyperadrenocorticism have pituitary (rather than adrenal) tumors.

Unilateral or bilateral adrenal gland tumors may also occur in cats. In a 2016 study of 33 feline adrenal tumors, 17 were carcinomas, 13 were adenomas, and 3 were pheochromocytomas. Nineteen of these cats were found to have functional tumors, with 16 of 19 causing hypersecretion of aldosterone. Common clinical signs include weakness (most often due to significant hypokalemia), blindness, respiratory issues, and gastrointestinal signs.

Pheochromocytomas are tumors of the adrenal medulla that secrete excessive amounts of catecholamines (primarily norepinephrine, but also epinephrine and dopamine) and other vasoactive peptides (e.g., vasoactive intestinal polypeptide, somatostatin, enkephalin, corticotropin). Excessive catecholamine and vasoactive peptide levels may manifest as cardiovascular, respiratory, or central nervous system (CNS) disease. Although these tumors have classically been reported as benign, recent reports suggest that regional invasion and distant metastases (liver, regional lymph nodes, lungs, spleen, ovaries, diaphragm, and vertebrae) occur in as many as 50% of affected dogs. Invasion of the caudal vena cava, phrenicoadominal (cranial abdominal) artery or vein, renal artery or vein, or hepatic vein may cause signs of ascites, edema, or venous distention. Pheochromocytomas are usually unilateral, although bilateral tumors occur. These masses are usually reddish-tan, multilobulated, firm, or friable, and may be completely or partly encapsulated. Occasionally, pheochromocytomas may be associated with neoplastic transformation of multiple endocrine tissues of neuroectoderm origin (e.g., pituitary, adrenocortical, or thyroid adenomas; or pancreatic islet cell tumors). Extra-adrenal pheochromocytomas have been reported in dogs and cats. Other tumors rarely arising from the adrenal medulla include neuroblastoma and ganglioneuroma.

**DIAGNOSIS**

**Clinical Presentation**

**Signalment**

Adrenocortical tumors usually occur in older, large-breed dogs and appear to be diagnosed more commonly in females. A definitive breed predisposition has not been identified. Pheochromocytomas usually occur in older dogs but have been reported in dogs as young as 1 year; both genders appear to be equally affected.

**History**

It is critical to understand that HAC can be diagnosed only in animals with clinical signs consistent with HAC. To diagnose HAC, there must be abnormalities (e.g., polyuria-polydipsia, polyphagia, pendulous abdomen [i.e., “pot belly”], endocrine alopecia, hyperpigmentation, and/or calcinosis cutis) that are typical of HAC. One might also see muscle wasting, weakness, lethargy, and/or panting in some patients, but these are not strongly suggestive of HAC. No imaging or laboratory findings (including adrenal gland function tests) allow diagnosis of HAC in the absence of obvious clinical signs. Pheochromocytoma and nonfunctional tumors will not cause these signs.

Vomiting has been associated with intestinal perforation in a dog with adrenocortical adenoma, but this is rare. High circulating levels of glucocorticoids may make diagnosis of intestinal perforation difficult because signs of peritonitis (abdominal discomfort, restlessness, panting, weakness, and/or dyspnea) are initially obscured. Cats reported with primary hyperaldosteronism present with a history of weakness, vomiting, inappetence, dehydration, diarrhea, and cervical ventroflexion.
Pheochromocytomas may cause vague, intermittent signs of weakness or panting due to episodic hypertension and tachycardia. Many times they are incidental findings on CT, ultrasonography, or necropsy. Signs of nonfunctional adrenocortical tumors may include anorexia, abdominal enlargement, abdominal pain, diarrhea, vomiting, and lethargy; however, they too may be incidental findings on CT, ultrasonography, or necropsy. In a study of 270 dogs undergoing abdominal CT for reasons other than adrenal gland disease, incidental adrenal gland masses were identified in 9.3%.

**Physical Examination Findings**

Clinical findings in animals with adrenocortical tumors depend on whether the tumors are functional. Dogs with ADH should have obvious signs of HAC (see previous discussion). Ascites, abdominal pain, edema, diarrhea, and vomiting are more common with nonfunctional tumors, although many are asymptomatic. Cats with hyperaldosteronism may have pelvic limb weakness, cervical ventroflexion, or a plantigrade stance. For cats presenting with blindness, there may be hyphema, retinal detachment, or intraocular hemorrhage. A palpable abdominal mass may also be noted in cats.

Clinical findings in animals with pheochromocytomas may include tachycardia or cardiac arrhythmia, acute collapse, polypnea, panting, cough, lethargy, anorexia, dyspnea, weakness, abdominal distention, congestive heart failure, ataxia, incoordination, polyuria-polydipsia, and alopecia. Hypertension (paroxysmal or sustained) is also frequently present. However, pheochromocytomas can be incidental findings in dogs that have no clinical signs associated with the tumor.

**Diagnostic Imaging**

Adrenal tumors are difficult to detect radiographically unless they are associated with significant adrenal enlargement (≥20 mm) or calcification. Food should be withheld for 24 hours before radiography to allow the gastrointestinal tract to empty. In some dogs, mineralization of tissue cranial to the kidney may be seen on survey radiographs and may or may not be associated with obvious adrenal enlargement. This finding is suggestive of adrenocortical neoplasia (adenoma or carcinoma). Nonneoplastic mineralization of adrenal glands is rare in dogs; however, bilateral adrenal calcification may occur with PDH. Conversely, adrenal gland mineralization is considered an incidental finding in cats (Fig. 22.5). Hepatomegaly, calcinosis cutis, or osteoporosis may be seen with PDH and ADH. Enhanced abdominal contrast caused by increased abdominal fat may occur. Dogs with HAC are more likely to have calcium-containing uroliths than dogs without clinical evidence of HAC. Although pheochromocytomas may be detected radiographically if sufficiently large, ultrasonography and CT are more sensitive.

**NOTE** If you note mineralization of an adrenal gland radiographically in a dog, consider the possibility of neoplasia.

Ultrasonography is useful for assessing the adrenal size, echogenicity, and shape of the gland and whether invasion into adjacent structures has occurred. The normal canine adrenal gland size in mature dogs is dependent on body size. Several methods can be used to ultrasonographically assess the size of the adrenal gland. One method is to compare the maximal width with the length of the gland; this should be less than approximately 20 cm, whereas adrenal thickness in dogs more than 12 kg should not exceed 0.62 cm, whereas adrenal thickness in dogs more than 12 kg should not exceed 0.72 cm.

Both adrenals should be routinely imaged in dogs with HAC because one normal adrenal gland does not exclude the existence of a contralateral functional adrenocortical tumor. Bilateral adrenocortical tumors are rare. Pheochromocytomas have been reported to have mixed echo patterns and cannot be definitively differentiated from adrenocortical tumors. Although bilateral adrenal enlargement is suggestive of PDH, atrophy of the contralateral adrenal gland in dogs with functional adrenocortical tumors may not be apparent ultrasonographically. Adrenal metastasis may be diagnosed ultrasonographically. In cats, unilateral or bilateral adrenal masses may be identified by abdominal ultrasound.

**NOTE** It is not possible to definitively differentiate between benign and malignant adrenal lesions using ultrasonographic criteria alone unless invasion of the vena cava has occurred.

CT and MRI allow accurate localization of adrenal neoplasia, but they do not allow differentiation of tumor type. Adrenal carcinomas may appear as well-demarcated, homogeneous masses, or they may be poorly demarcated, with irregular texture and contrast enhancement. Masses that are poorly demarcated, irregularly shaped, and nonhomogeneous with mineralization are usually carcinomas. Ultrasound can detect caudal vena cava invasion, but contrast-enhanced CT is more sensitive. Administration of contrast agents in patients with pheochromocytoma may produce severe hypertension; thus it should be done cautiously. If contrast-enhanced CT and ultrasound are not performed, caudal vena cava angiography (note precautions in previous discussion) may be considered before surgery if caudal vena cava thrombosis is suspected (Fig. 22.6). Excretory urography...
may help identify tumor invasion requiring nephrectomy (e.g., ureteral obstruction or renal invasion).

**NOTE** CT and MRI do not differentiate adrenal adenomas, carcinomas, or pheochromocytomas. Clinical signs, biochemical testing, and a tissue sample are necessary for a definitive diagnosis.

**Laboratory Findings**

No laboratory changes are reliably seen in all animals with HAC; however, common laboratory abnormalities include substantially increased serum alkaline phosphatase, neutrophilic leukocytosis, lymphopenia, eosinopenia, mild polycythemia, modestly increased alanine aminotransferase, hypophosphatemia, and hypercholesterolemia. Mild hypernatremia and mild hypokalemia are rarely seen. Urinary tract abnormalities may include pyuratea (urine specific gravity <1.007) or isosthenuria (1.008–1.012). Urinary tract infections are common, even when bacteriuria and pyuria are absent.

Diagnosis of HAC requires clinical signs consistent with the disease. Abnormal adrenal function test results in the absence of clinical signs are not diagnostic of HAC. The ACTH stimulation test (Box 22.4) and the low-dose dexamethasone suppression (LDDS) test are the primary adrenal function tests used. The ACTH stimulation test is easy and quick to perform, and is the best test to look for iatrogenic HAC (Box 22.5). However, the ACTH stimulation test has disadvantages. Dogs with ADH can have almost any response (i.e., normal, exaggerated, or diminished) to ACTH, and many clinically ill dogs without HAC have exaggerated test results that mimic HAC. Classically, one measures cortisol concentrations before and after administration of ACTH, but one may also measure sex steroids (e.g., 17-hydroxyprogesterone). The sensitivity and specificity of sex steroid concentrations for diagnosing HAC are uncertain. The LDDS test requires 8 hours to perform, but it is a better test to look for spontaneous HAC. Furthermore, the LDDS test often permits differentiation of PDH and ADH.

Functional adrenocortical carcinoma may cause decreased aldosterone concentrations and increased deoxycorticosterone concentrations in the presence of hypokalemia. These metabolic abnormalities have been shown to resolve with resection of the carcinoma. Elevated serum aldosterone levels have been found in all cats with hyperaldosteronism secondary to an adrenal tumor. Other laboratory findings include hypokalemia, increased creatine kinase, alkalemia, hyperglycemia, hypophosphatemia, and hypernatremia, as well as evidence of chronic kidney disease.

It is critically important to note that HAC cannot be diagnosed simply by performing an adrenal function test; these tests may be substantially altered by nonadrenal disease or drugs. Exaggerated test results often occur in chronically stressed or ill dogs. In particular, animals with abnormal adrenal function tests but normal-sized adrenal glands should be carefully scrutinized before beginning therapy. PDH and ADH very rarely coexist. Because of the complexity of diagnosing HAC and differentiating PDH from ADH, readers are referred to a medical text for further information.

Laboratory abnormalities are inconsistent and nonspecific in animals with pheochromocytomas. However, measurement of urine and plasma catecholamines and metanephrines have been used to help differentiate between dogs with HAC and dogs with pheochromocytomas. Dogs with pheochromocytomas classically have significantly higher urinary normetanephrine- and metanephrine-to-creatinine ratios, and significantly higher plasma-total and -free normetanephrine and plasma-free metanephrine concentrations compared with dogs with HAC and dogs with nonadrenal disease.

**DIFFERENTIAL DIAGNOSIS**

Pheochromocytomas and adrenocortical tumors must be differentiated because operative management is different. Generally, clinical signs and laboratory analysis allow preoperative differentiation (see previous discussion). At surgery, pheochromocytomas may be identified grossly by application of Zenker’s solution (potassium dichromate or iodate), which oxidizes catecholamines, forming a dark brown pigment within 10 to 20 minutes after application to the surface of a freshly sectioned tumor. Although adrenal carcinomas are apt to be large and invasive, differentiation of adenomas and carcinomas is impossible without histopathology. Apparent metastatic lesions in the liver or draining lymph nodes may suggest malignancy, but care should be taken to differentiate benign hepatic nodules from neoplastic disease.

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**BOX 22.4 Adrenocorticotropic Hormone Stimulation Test in Dogs**

1. Obtain serum to determine pretest cortisol concentration.
2. Administer 2.2 IU/kg of ACTH gel or 5 μg/kg of synthetic ACTH (Corotroyn) IM or IV
3. Obtain serum for testing 1 hour after ACTH administration.

**ACTH**, Adrenocorticotropic hormone; **IM**, intramuscular; **IV**, intravenous.

**BOX 22.5 Patterns of Adrenocorticotropic Hormone Stimulation Tests (Post–Adrenocorticotropic Hormone Cortisol)**

- <24 μg/dL: suggestive of hyperadrenocorticism
- 19–24 μg/dL: equivocal for hyperadrenocorticism
- 8–18 μg/dL: normal
- <4 μg/dL: potentially consistent with iatrogenic Cushing disease
- <1 μg/dL: consistent with hypoadrenocorticism, spontaneous or iatrogenic

*aSubstantial variation between laboratories may occur. To convert μg/dL to nmol/L, multiply μg/dL x 27.59.
*bSevere nonadrenal disease can be associated with stress, causing values this high or higher; hyperadrenocorticism is not diagnosed simply by performing an adrenal function test.

**FIG. 22.6** Color Doppler evaluation of the caudal vena cava. A hyperechoic mass is seen within the lumen of the cava from invasion of an adrenal tumor. Note the disruption of blood flow around the mass (blue).
MEDICAL MANAGEMENT

Adrenergic blockage (e.g., phenoxybenzamine, phentolamine, prazosin) is used to control blood pressure in patients with pheochromocytoma. These drugs are also used preoperatively and intraoperatively (see discussions of Preoperative Management and Anesthesia on p. 586). If tachycardia or cardiac arrhythmias are present, β-adrenergic blockade may be used; however, unopposed β-blockade may cause severe, life-threatening hypertension. β-Blockade should not be started until α-adrenergic blockade has been determined to be adequate. In one study, phenoxybenzamine-treated dogs undergoing adenalecromy for pheochromocytoma had a significantly decreased mortality rate compared with untreated dogs (13% vs. 48%, respectively).8

In general, it is recommended to pretreat dogs for 2 weeks with phenoxybenzamine (see p. 594) prior to surgery for a pheochromocytoma.

Medical therapy for HAC is potentially complex and can have significant side effects. Therefore the reader is referred to a current medical text for a more complete discussion. Trilostane inhibits one of the synthetic enzymes, thus blocking production of cortisol and other adrenal steroids; it usually does not cause adrenal necrosis. Trilostane appears to be as safe or perhaps safer than other drugs used to treat HAC and is effective in most patients (Box 22.6). The major disadvantages are that it must be given daily and it has occasional side effects (e.g., hypoadrenocorticism, hypertension).

Mitotane (o,p′-DDD) destroys the adrenal cortex in a dose-dependent fashion. It was the major drug used to treat canine HAC before the advent of trilostane. Mitotane can often control clinical signs in animals with ADH; however, tumors are more resistant to the adrenocorticotytic effects of mitotane than are normal or hyperplastic adrenal cortices. Larger doses (see Box 22.6) are often required to attain and maintain control in ADH dogs than in PDH dogs, and stronger side effects (e.g., gastric irritation, vomiting) can be expected. Major advantages of mitotane include the following: (1) it is effective, and (2) after an induction therapy (usually 4–14 days), maintenance therapy consists of one to two treatments per week. Major disadvantages are: (1) it can easily destroy the entire adrenal gland, resulting in temporary or permanent hypoadrenocorticism (or death), (2) some dogs are resistant to its effects, and (3) some dogs are very sensitive to its effects. The last two disadvantages mean that there is substantial variation in how patients respond. Some patients respond well and are easy to treat, whereas others are extremely difficult to control using mitotane. For this reason, trilostane has become the more popular treatment for HAC.

Ketoconazole causes reversible inhibition of adrenal steroid production and has little effect on mineralocorticoid production. Therefore ketoconazole may be used (1) in dogs with ADH who are not surgical candidates, (2) before surgery to reduce anesthetic and surgical risks in animals with uncontrolled HAC, and (3) as a diagnostic trial in dogs in which equivocal test results make the diagnosis of HAC difficult. If used for diagnostic purposes, the drug should be given for a minimum of 4 to 8 weeks. Major advantages of ketoconazole include the following (1) it is relatively safe, and (2) it is very effective. Major disadvantages are: (1) it works only while there are blood levels of drugs, (2) it is relatively expensive, and (3) it is so effective at decreasing cortisol concentrations that it is easy to make patients feel sick when first starting the drug. Furthermore, adverse reactions to ketoconazole (e.g., anorexia, depression, vomiting, diarrhea, icterus) may occur, necessitating that the drug be dropped or the dosage reduced. If an overdose is suspected of causing acute illness or collapse, one should administer glucocorticoids and stop the ketoconazole. As previously noted, it is seldom used for HAC since the introduction of trilostane.

Amlodipine, spironolactone, and potassium gluconate are used in the medical management of primary hyperaldosteronism associated with adrenal tumors in cats. Aldosterone is released from adrenal glands via the action of the renin-angiotensin-aldosterone system. Aldosterone stimulates renal sodium reabsorption, resulting in volume expansion. Signs of hyperaldosteronism typically result from systemic hypertension. Aldosterone also stimulates potassium excretion and may result in a hypokalemic myopathy.

SURGICAL TREATMENT

The overall health of the animal, the presence of nonresectable metastases, and the apparent invasiveness of the tumor (i.e., evidence of caudal vena cava thrombosis on CT or ultrasound) should be considered when the appropriateness of surgery for adrenal tumors is determined. Long-term survival (>1 year) may be possible, even in dogs with widespread metastatic lesions. If the tumor appears invasive, a midline abdominal approach is preferred to allow evaluation of the caudal vena cava and other abdominal structures. Thrombus removal may require that the midline incision be extended into the caudal thorax through a caudal median sternotomy approach (see p. 891). Thrombi more commonly occur because of intraluminal extension via the adrenal or renal vein, and less commonly by direct invasion. Cava thrombi occur in approximately one-fourth of dogs with adrenal gland tumors. They are more common with pheochromocytoma than with adrenocortical tumor, but they may occur with either. Venotomy may be used to remove tumors extending into the caudal vena cava. If venotomy cannot be performed, gradual

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**BOX 22.6 Medical Treatment for Adrenal Tumors**

**Trilostane (Primary Drug Used for Adrenal Tumors)**

1. Start with 1 mg/kg bid or 2 mg/kg once daily (twice daily is probably more effective). If that dose is insufficient, then gradually increase the dose orally with food. Observe for signs of lethargy, vomiting, diarrhea, or decreased appetite.
2. Perform a clinical examination, serum biochemistry profile, and ACTH stimulation 4–6 hours after the morning capsule on days 10–14.
3. Increase the dose until the owner reports a good clinical response and the ACTH serum cortisol is <9 μg/dL.

**Mitotane**

1. Administer 50–75 mg/kg once daily with food plus 0.2 mg/kg prednisolone/day. Observe for signs of lethargy, vomiting, diarrhea, or decreased appetite. Recheck dog in 10–14 days. If no response, then increase dose by 50 mg/kg/day.
2. Perform ACTH stimulation test. Appropriate response is ACTH-stimulated cortisol <1 μg/dL. If patient is responding, decrease mitotane to 75–100 mg/kg/day and continue prednisolone. If no response at 14 days, then increase mitotane by 50 mg/kg/day (continue prednisolone) for another 14 days and recheck.

**ACTH**, Adrenocorticotropic hormone.

*A A total cumulative dose of up to 3000 to 5000 mg/kg may be needed. Treatment for ablation generally is 10 days to 11 weeks (mean, 24 days). Two-thirds of dogs eventually relapse.
occlusion of the caudal vena cava may allow removal of adrenal gland tumors with vascular invasion that would otherwise be difficult or impossible to resect; en bloc resection of the caudal vena cava during removal of a pheochromocytoma may also be performed. Small tumors and those that do not appear invasive may be removed through a paralumbar approach (see p. 588). Laparoscopic adrenalectomy has also been performed in dogs and cats (see p. 588).

Preoperative Management
Preoperative management for a function adrenal tumor causing hyperadrenocorticism should include 3 to 4 weeks of trilostane therapy (see Box 22.6). Renal function should be determined before surgery in case ipsilateral nephrectomy is necessary. Substantial electrolyte or acid-base abnormalities, blood glucose concentrations greater than 200 mg/dL, and hypertension should be corrected before surgery, if possible. Fluid therapy should be initiated before induction of anesthesia. Animals with HAC are at increased risk of postoperative PTE (see p. 586). Fresh-frozen plasma transfusions, aspirin, and/or plasma incubated with heparin may be indicated in patients with disseminated intravascular coagulation (DIC) (Box 22.7). Aspirin, heparin, and clopidogrel have been used preoperatively in patients with adrenocortical tumor to prevent thromboembolism (see Box 22.7), but the value of these preventative measures is controversial. Heparin (75–100 U/kg) may be added to plasma during surgery, with the animal continuing to take heparin for 3 to 4 days. Low-molecular-weight heparin (150 U/kg) may be more effective than unfractionated heparin but requires different monitoring than unfractionated heparin (see Box 22.7). Perioperative antibiotics should be administered and continued in the immediate postoperative period in hyperadrenal animals. Do not give steroids to animals with functional adrenal tumors before surgery as they may increase blood pressure and promote PTE. Wait until surgery to give steroids in these animals.

Particular emphasis should be placed on preoperative examination of the cardiovascular system for evidence of arrhythmias or congestive heart failure in animals with pheochromocytomas. If cardiac arrhythmias are present, a β-blocker may be added, but only after the phenoxybenzamine dosage (see under Anesthesia) has been determined to be adequate and blood pressure has returned to normal. Both α- and β-blockade will allow the return of a normal fluid volume; however, they may unmask renal insufficiency and anemia. α-Adrenergic blockade has been shown to drastically reduce the incidence of severe perioperative hypertension, thereby reducing mortality (Table 22.1).

Anesthesia
Anesthetic complications are common during adrenalectomy for pheochromocytoma, and wide fluctuations in heart rate (HR) and blood pressure are typical in unblocked animals. Even if hypertension is well controlled, patients can be extremely difficult to manage under anesthesia. Appropriate monitoring is critical and includes cardiac rhythm, arterial blood pressure, end-tidal carbon dioxide (EtCO₂), and pulse oximetry. Treatment for several weeks before surgery with an α-adrenergic blocker (e.g., phenoxybenzamine; see Table 22.1) is recommended. An initial dose of phenoxybenzamine, 0.25 mg/kg given orally every 12 hours is gradually increased every 2 to 3 days until blood pressure is within the normal range. This process may take 1 to 2 weeks before the patient is adequately blocked. Maximum dose is 2 mg/kg. HR can be controlled with a β-blocker (e.g., metoprolol, esmolol); however, this treatment should not be initiated until adequate α-blockade has been established (i.e., normal blood pressure). Intraoperative β-blockade with esmolol (see Table 22.1) is preferred because of its short half-life and can be given as boluses or constant-rate infusion (CRI). Cardiac arrhythmias may be treated with lidocaine (Box 22.8) or esmolol (see Table 22.1). Hypertension may result from tumor manipulation and can be minimized by isolating the blood supply of the tumor before manipulating the tumor. Hypertension may be treated with phentolamine given as an intravenous (IV) bolus (see Table 22.1). Sodium nitroprusside or nitroglycerin may also be infused if hypertension is present. Hypotension frequently occurs after tumor removal; large doses of crystalloids should be given to replace estimated blood loss, as well as third-space fluid loss. If hypotension persists, dobutamine could be administered (2–10 μg/kg per minute IV). In human pheochromocytoma patients, the vasopressors of choice are phenylephrine and norepinephrine. Because removal of the tumor initiates cessation of norepinephrine release into the bloodstream, infusion of an alpha-1 agonist (phenylephrine or norepinephrine) provides

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**BOX 22.7 Therapeutics for Disseminated Intravascular Coagulation**

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma (Fresh Frozen)*</td>
<td>10–20 mL/kg, then reassess plasma ATIII activity. Repeat as needed to increase ATIII to near-normal concentrations. NOTE: Large amounts of FFP may be necessary to increase ATIII, but it is thought that adequate levels of ATIII are critical in these patients.</td>
</tr>
<tr>
<td>Aspirin*</td>
<td>0.5 mg/kg PO q24h (efficacy not proven in dogs or cats)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>Loading dose of 2–3 mg/kg (some suggest as high as 10 mg/kg), then 1–2 mg/kg q24h (efficacy not proven in dogs or cats)</td>
</tr>
<tr>
<td>Heparin-Activated Plasma*</td>
<td>Place the first heparin dose (50–100 U/kg) into the plasma and incubate for 30 minutes before administration. Once ATIII levels are above 60%, continue the heparin subcutaneously. If additional plasma is needed, incubation with heparin is not necessary (efficacy not proven in dogs or cats).</td>
</tr>
<tr>
<td>Heparin (Unfractionated)**</td>
<td>50–300 U/kg SC q8–12h; adjust dose based on monitoring</td>
</tr>
<tr>
<td>Low-Molecular-Weight Heparin (Dalteparin)**</td>
<td>100–150 U/kg SC q8–24h (dogs) 180 U/kg SC q12h (cats)</td>
</tr>
</tbody>
</table>

*Replacing clotting factors and ATIII is probably the best therapy for DIC; however, large doses of FFP are required to raise ATIII. Thus the efficacy of this treatment has been questioned.

**Is believed effective in DIC associated with immune-mediated hemolytic anemia; efficacy in DIC caused by other diseases unproven.

**Heparin is controversial in the treatment of DIC. Low-molecular-weight heparin is probably more effective than unfractionated heparin, but its effectiveness for treating DIC is questionable. Substantial differences have been noted between the two forms, and the reader is referred to a medical text for a more complete discussion, including differences in monitoring the effectiveness of therapy. As more becomes known regarding low-molecular-weight heparin, this dosage recommendation may change.

**Heparin is no longer considered to be an optimal treatment for DIC. There are no adequately designed publications in veterinary medicine critically evaluating heparin therapy in patients with DIC. Rightly or wrongly, current thinking at the time of this writing is that heparin should not be used for patients with DIC that have concurrent inflammatory conditions, those that are actively bleeding, or those that appear hypocoagulable. Although heparin seems beneficial in patients that are at risk for thromboembolism, there is no study that confirms this hypothesis. Readers are referred to a medical text for advances in the treatment of DIC.

**ATIII, Antithrombin III; DIC, disseminated intravascular coagulation; FFP, fresh-frozen plasma; SC, subcutaneous.
### CHAPTER 22  Surgery of the Endocrine System

#### Preoperative Considerations

**Associated conditions**
- Anemia (may be occult)
- Hypovolemia
- Hypertension
- Tachydysrhythmias
- Ventricular ectopy
- Cardiac dysfunction
- Renal insufficiency (may be occult)
- Cardiogenic pulmonary edema

**Bloodwork**
- HCT
- Electrolytes
- BUN
- Cr
- TP
- Urinalysis

**Physical examination**
May be hypovolemic, tachycardic, and hypertensive if untreated with phenoxybenzamine. May have orthostatic hypotension if treated.

**Other diagnostics**
- Blood pressure is essential
- ECG
- Radiographs (thoracic, abdominal)
- Ultrasound

### Premedications

Give:
- Phenoxybenzamine until the morning of surgery
- Diazepam (0.2 mg/kg IV), or
- Midazolam (0.2 mg/kg IV, IM), or
- Hydromorphone (0.05–0.2 mg/kg IV, IM), or
- Oxymorphone (0.1–0.2 mg/kg IV, IM), or
- Morphine (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM)
- Avoid ketamine, xylazine, medetomidine, dexmedetomidine, atropine, glycopyrrolate, and acepromazine

### Intraoperative Considerations

#### Induction

- Titrate propofol (2–4 mg/kg IV if sedated or 4–8 mg/kg IV if unsedated), or
- Give alfaxalone (2–3 mg/kg IV if sedated or 3–5 mg/kg IV if unsedated)
- If CHF, titrate etomidate (0.5–1.5 mg/kg)

#### Maintenance

- Isoflurane or sevoflurane plus
  - Fentanyl (1–5 μg/kg IV loading dose, then 2–30 μg/kg/h IV), or
  - Hydromorphone (0.05–0.2 mg/kg IV PRN), or
  - Oxymorphone (0.1–0.2 mg/kg IV, IM), or
  - Morphine (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM) if minimal hypotension, plus
  - For hypertension (to keep MAP 70–90 mm Hg)
  - Phentolamine (0.02–0.1 mg/kg IV bolus and/or CRI (0.5–3 μg/kg/min IV) and
  - Nitroprusside (1–5 μg/kg/min IV) and
  - Esmolol (0.05–0.25 mg/kg IV boluses every 2–5 min to effect and/or CRI (50–200 μg/kg/min IV) to maintain normal heart rate
  - For hypotension (to keep MAP 70–90 mm Hg)
  - Phenylephrine (20–200 μg IV boluses and/or CRI 0.1–1 μg/kg/min IV), or
  - Norepinephrine CRI (0.05–2 μg/kg/min IV), or
  - Dopamine (5–15 μg/kg/min IV)
  - For hypotension with CHF
  - Epinephrine (0.1–1 μg/kg/min IV) or
  - Dobutamine (2–15 μg/kg/min IV)

#### Fluid needs

- 5–10 mL/kg/h to replace evaporative losses plus 3 x EBL
  - If CHF, then 5–10 mL/kg and slower replacement of fluid losses over 3–4 h if necessary

#### Monitoring

- BP: essential
- ECG
- Respiratory rate
- SpO₂
- EtCO₂
- Temperature
- Arterial line
- U/O

#### Blocks

**Epidural**
- Morphine (0.1 mg/kg preservative free) or
- Buprenorphine (0.003–0.005 mg/kg diluted in saline)

**Incisional**
- Lidocaine (<5 mg/kg), or
- Bupivacaine (<2 mg/kg)

### TABLE 22.1 Anesthetic Considerations in the Canine Pheochromocytoma Patient

<table>
<thead>
<tr>
<th>Associated conditions</th>
<th>Bloodwork</th>
<th>Physical examination</th>
<th>Other diagnostics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia (may be occult)</td>
<td>HCT</td>
<td>May be hypovolemic, tachycardic, and hypertensive if untreated with phenoxybenzamine. May have orthostatic hypotension if treated.</td>
<td>Blood pressure is essential</td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>Electrolytes</td>
<td></td>
<td>ECG</td>
</tr>
<tr>
<td>Hypertension</td>
<td>BUN</td>
<td></td>
<td>Radiographs (thoracic, abdominal)</td>
</tr>
<tr>
<td>Tachydysrhythmias</td>
<td>Cr</td>
<td></td>
<td>Ultrasound</td>
</tr>
<tr>
<td>Ventricular ectopy</td>
<td>TP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac dysfunction</td>
<td>Urinalysis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal insufficiency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(may be occult)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic pulmonary edema</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Premedications</th>
<th>Intraoperative Considerations</th>
</tr>
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<tbody>
<tr>
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</tr>
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</tr>
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<td>Hydromorphone (0.05–0.2 mg/kg IV, IM), or</td>
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</tr>
<tr>
<td>Oxymorphone (0.1–0.2 mg/kg IV, IM), or</td>
<td>Oxymorphone (0.1–0.2 mg/kg IV PRN), or</td>
</tr>
<tr>
<td>Morphine (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM)</td>
<td>Morphine (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM) if minimal hypotension, plus</td>
</tr>
<tr>
<td>Avoid ketamine, xylazine, medetomidine, dexmedetomidine, atropine, glycopyrrolate, and acepromazine</td>
<td>For hypertension (to keep MAP 70–90 mm Hg)</td>
</tr>
<tr>
<td>phenylephrine (20–200 μg IV boluses and/or CRI 0.1–1 μg/kg/min IV), or</td>
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<td>Nitroprusside (1–5 μg/kg/min IV) and</td>
</tr>
<tr>
<td>Dopamine (5–15 μg/kg/min IV)</td>
<td>Esmolol (0.05–0.25 mg/kg IV boluses every 2–5 min to effect and/or CRI (50–200 μg/kg/min IV) to maintain normal heart rate</td>
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</tr>
<tr>
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<td>Dopamine (5–15 μg/kg/min IV)</td>
</tr>
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<td>For hypotension with CHF</td>
<td>Epinephrine (0.1–1 μg/kg/min IV) or</td>
</tr>
<tr>
<td>Epinephrine (0.1–1 μg/kg/min IV) or</td>
<td>Dobutamine (2–15 μg/kg/min IV)</td>
</tr>
<tr>
<td>Dobutamine (2–15 μg/kg/min IV)</td>
<td></td>
</tr>
<tr>
<td>Fluid needs</td>
<td>Fluid needs</td>
</tr>
<tr>
<td>5–10 mL/kg/h to replace evaporative losses plus 3 x EBL</td>
<td>5–10 mL/kg/h to replace evaporative losses plus 3 x EBL</td>
</tr>
<tr>
<td>If CHF, then 5–10 mL/kg and slower replacement of fluid losses over 3–4 h if necessary</td>
<td>If CHF, then 5–10 mL/kg and slower replacement of fluid losses over 3–4 h if necessary</td>
</tr>
</tbody>
</table>

### Continued
etCO₂, TP, total protein; CO₂ causes an increase in catecholamine release, EtCO₂ monitoring circulating levels of catecholamines. Because an increase in arterial should be avoided because it increases HR, blood pressure, and an undesirable addition to the anesthetic protocol. Ketamine to catecholamines, changes in blood pressure make alpha-2 agonists hypotension. Although they may increase myocardial sensitivity typically cause transient hypertension followed by prolonged and dexmedetomidine are primarily alpha-2 agonists. They in patients with pheochromocytoma. Xylazine, medetomidine, the sympathetic nervous system to be unopposed. Tachyarrhythmias and glycopyrrolate block the parasympathetic pathways, allowing of having a pheochromocytoma. As anticholinergics, atropine and medetomidine, and ketamine should not be used in patients suspected with arrhythmias, the need for perioperative steroid replacement should be anticipated. When etomidate is used, the advantages of cardiovascular stability should be weighed against the possibility of transient adrenal suppression, which may occur in patients undergoing unilateral adrenalectomy. Isoflurane and sevoflurane are the inhalation agents of choice because they do not sensitize the myocardium to epinephrine-induced arrhythmias; halothane should be avoided. In addition, drugs that release histamine such as morphine and meperidine should be avoided. Other opioids such as hydromorphone and/or fentanyl provide good pain control without histamine release.

### Surgical Anatomy

See p. 587 for the discussion of the surgical anatomy of the adrenal gland.

### Positioning

The animal is positioned in dorsal recumbency or in lateral recumbency with the affected side up, depending on the operative approach chosen. With large or invasive tumors, a generous area should be clipped and prepared for surgery to allow a caudal thoracotomy (median sternotomy) to be performed if necessary.

#### SURGICAL TECHNIQUE

Adrenalectomy via a midline abdominal or a paralumbar approach is described on pp. 587 to 588. Laparoscopic adrenalectomy is discussed on p. 588. Concurrent nephrectomy may be necessary in some patients with invasive tumors. Surgical resection of adrenal tumors should be aggressive to ensure complete tumor removal. En bloc resection should be performed if possible to prevent leaving behind small fragments of neoplastic tissue. The vascular supply to pheochromocytomas should be isolated before tumor manipulation to reduce catecholamine release and help prevent shedding of tumor cells. Venotomy may be required to remove tumor thrombi. The entire abdomen should be explored, with special attention paid to the bladder, pelvic canal, kidneys, and aorta near the junction of the caudal mesenteric artery, where extra-adrenal neoplasia is reported to occur.

### SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Care should be taken when selecting the appropriate suture because delayed wound healing may occur in any debilitated

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**TABLE 22.1 Anesthetic Considerations in the Canine Pheochromocytoma Patient—cont’d**

<table>
<thead>
<tr>
<th>Postoperative Considerations</th>
<th>Analgesia</th>
<th>Monitoring</th>
<th>Bloodwork</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Analgesia</strong></td>
<td>Fentanyl CRI (1–10 μg/kg loading dose, then 2–20 μg/kg/h IV, or</td>
<td>SpO₂, Blood pressure is essential</td>
<td>Estimated pain score Moderate to severe</td>
</tr>
<tr>
<td></td>
<td>Morphine (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h) if minimal hypotension or</td>
<td>ECG, HR, Respiratory rate, Temperature, U/O</td>
<td>Moderate to severe</td>
</tr>
<tr>
<td></td>
<td>Hydromorphone (0.05–0.2 mg/kg IV, IM q5–4h), or</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Oxymorphone (0.1–0.2 mg/kg IV q2–4h)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Monitoring</strong></td>
<td>IV, CRI</td>
<td>IV, CRI</td>
<td>IV, CRI</td>
</tr>
<tr>
<td></td>
<td>Blood pressure</td>
<td>Blood pressure</td>
<td>Blood pressure</td>
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<tr>
<td></td>
<td>end-tidal CO₂</td>
<td>end-tidal CO₂</td>
<td>end-tidal CO₂</td>
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<tr>
<td></td>
<td>MAP</td>
<td>MAP</td>
<td>MAP</td>
</tr>
<tr>
<td><strong>Bloodwork</strong></td>
<td>HCT</td>
<td>HCT</td>
<td>HCT</td>
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<tr>
<td></td>
<td>TP</td>
<td>TP</td>
<td>TP</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Estimated pain score</strong></td>
<td>Moderate to severe</td>
<td>Moderate to severe</td>
<td>Moderate to severe</td>
</tr>
</tbody>
</table>

*Give slowly to prevent histamine release.*

**BOX 22.8 Lidocaine Administration for Ventricular Arrhythmias**

**Dogs**

Give IV (2 mg/kg bolus, up to 8 mg/kg total dose) to determine responsiveness to this drug. If the arrhythmias decrease or stop, lidocaine should be given by IV CRI of 50–75 μg/kg/min for 50 μg/kg/min, place 500 mg lidocaine in 500 mL of fluids and administer at maintenance rate (66 mL/kg/dj).

**Cats**

Give IV (1 mg/kg bolus, up to 4 mg/kg total dose); if necessary administer 25–50 μg/kg/min by CRI.

CRI, Constant-rate infusion; IV, intravenous.
animal (see p. 589). Self-retaining retractors (e.g., Balfour abdominal retractors) and malleable retractors are useful for improving visualization of the adrenal glands. With vascular tumors, hemostasis is attained more easily with electrocautery, Ligasure, and hemoclips than with suture ligation of vessels.

**POSTOPERATIVE CARE AND ASSESSMENT**

Animals with HAC caused by ADH often develop hypoadrenocorticism postoperatively as a result of atrophy of the contralateral gland. These animals require glucocorticoid therapy postoperatively (see discussion of postoperative care on p. 589). If HAC continues postoperatively, medical therapy should be considered. Fluid therapy should be continued until the animal is able to maintain hydration. Blood pressure, HR, and heart rhythm should be carefully monitored after surgery. Blood transfusions may be required intraoperatively or postoperatively in some patients. Dogs should be reevaluated periodically for tumor recurrence. The most common complications after adrenal tumor removal include dyspnea, hemoperitoneum, ventricular arrhythmias, anuric acute renal failure, and coagulopathies. Dogs with adrenocortical tumors are at increased risk of developing thromboembolism (see p. 586).

**PROGNOSIS**

Although there can be significant perioperative complications, median survival for dogs that survive to discharge exceeds 10 months. In a study of 52 dogs undergoing adrenalectomy, survival time was significantly shorter in dogs with carcinoma, tumors with a major axis length greater than 5 cm, thrombosis, metastasis, and when adrenalectomy was combined with another abdominal surgical procedure. Dogs in this study had a median survival of 953 days, with more than 65% living for more than 1 year.

Perioperative mortality ranges from 10% to 20%. Risk factors that have been identified for poor short-term survival (death <14 days after surgery) include caval invasion, extent of invasion (extension beyond the hepatic hilus), receipt of intraoperative transfusion, dogs with pheochromocytoma, and postoperative complications such as DIC, pancreatitis, hypotension, hypoxemia, and renal failure. In a study of nine dogs undergoing bilateral adrenalectomy, median survival of the eight dogs that survived to hospital discharge was 525 days; no dogs died of metastatic disease or from complications of hypoadrenocorticism.

Outcomes following laparoscopic adrenalectomy have been reported in several studies. When comparing perioperative morbidity and mortality rates for laparoscopic versus open adrenalectomy in dogs with noninvasive adenocortical tumors, laparoscopic adrenalectomy was associated with shorter surgical and hospitalization times, with no perioperative deaths. In a study of 10 dogs that had laparoscopic removal of noninvasive pheochromocytomas, only one dog required conversion to an open approach. Prognostic factors for improved survival for pheochromocytoma include preoperative use of phenoxybenzamine, younger age, lack of intraoperative arrhythmias, and decreased surgical time.

Surgical treatment of adrenal gland tumors in cats has good long-term survival regardless of tumor type. Perioperative mortality (death <14 days after surgery) was 23% in a 2016 study; median survival time was 50 weeks.

**PITUITARY NEOPLASIA**

**DEFINITIONS**

Pituitary tumors arise from the hypophysis in the sella turcica. Hypophysectomy is the surgical removal of the pituitary gland. The pituitary gland is also called the hypophysis.

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

Functional pituitary tumors are the most common cause of canine HAC. However, 40% of pituitary tumors are nonfunctional. Clinical signs are usually caused by hypersecretion of ACTH from tumors in the pars distalis (adenohypophysis) or pars intermedia. Large pituitary tumors often grow dorsally into the brain because the diaphragm of the sella is incomplete. Such tumors may cause clinical signs by impinging on adjacent brain tissue (e.g., optic chiasm, hypothalamus, thalamus, infundibular recess, and third ventricle). Size of the tumor and development of neurologic signs do not always correlate. Adenomas and carcinomas may arise from pituitary tissue; however, carcinomas represent less than 3% of all pituitary neoplasms. Adenomas are usually classified as microadenomas (<1 cm in diameter) or macroadenomas (>1 cm in diameter). Microadenomas are most common, accounting for nearly 70% of all pituitary tumors.

**DIAGNOSIS**

**Clinical Presentation**

**Signalment**

Poodles, Malteses, dachshunds, and boxers may be predisposed to PDH. Middle-aged and older dogs are most commonly affected; however, young dogs may occasionally develop pituitary tumors.

**History**

Most dogs are presented for evaluation of typical signs of HAC (polyuria, polydipsia, polyphagia, abdominal enlargement, endocrine alopecia, muscle wasting, weakness, lethargy, panting, and/or hyperpigmentation). Concurrent neurologic signs (e.g., seizures, visual deficits, ataxia, incoordination, facial hemiplegia, head tilt, somnolence, compulsive walking, depression) may be noted. The diversity of neurologic signs in dogs with pituitary tumors is probably a result of impingement on various parts of the brain responsible for differing functions. Mental depression and stupor were reported as the most common abnormalities in two studies describing dogs with large pituitary tumors. In animals with nonfunctional macroadenomas or carcinomas, neurologic signs may be the only presenting abnormality, and some owners report only anorexia with no obvious CNS abnormalities.

Cats with insulin-resistant DM secondary to pituitary adenoma present with a ravenous appetite, increased body weight, polyuria/polydipsia, and a dull hair coat.

**NOTE** Large, nonfunctional pituitary tumors may cause neurologic abnormalities. Functional tumors usually cause hyperadrenocorticism but may also cause neurologic signs.

**Physical Examination Findings**

Typical signs of HAC (see previous discussion) are expected in animals with functional pituitary tumors. Neurologic

**NOTE** Warn owners that animals with pheochromocytoma may die suddenly as a result of arrhythmias and hypertension.
abnormalities (e.g., papillary edema, ataxia, incoordination) and poor appetite occasionally occur as the only signs.

**Diagnostic Imaging**

Diagnosis of pituitary neoplasia is best made with CT or MRI (Fig. 22.7). Pituitary adenomas and carcinomas cannot be differentiated with CT; however, animals with microadenomas, which might benefit from hypophysectomy, can be differentiated from animals with macroadenomas. The latter seldom benefit from surgery. Bilateral adrenal enlargement is usually indicative of PDH.

**Laboratory Findings**

Laboratory abnormalities are generally consistent with HAC. Small, nonfunctional pituitary tumors seldom cause laboratory abnormalities. Large tumors may cause increased intracranial pressure. See p. 592 for differentiation of PDH from ADH. In a diabetic cat with insulin resistance secondary to somatotroph pituitary adenoma, plasma concentrations of growth hormone (51 μg/L, reference range 0.8–7.2 μg/L) and insulin-like growth factor 1 (3871 μg/L, reference range 39–590 μg/L) were highly elevated.

**DIFFERENTIAL DIAGNOSIS**

Animals with PDH must be differentiated from those with iatrogenic HAC or ADH (see p. 592). Once a diagnosis of pituitary dysfunction has been made, pituitary neoplasms must be differentiated from other lesions that may arise in the pituitary (e.g., cysts, abscesses, and craniopharyngiomas); however, such lesions are rare.

**MEDICAL MANAGEMENT**

Clinical signs of HAC may be treated medically (see p. 593). External-beam radiation therapy (RT) appears to be an effective treatment for large pituitary tumors when combined with concurrent adrenal-suppressive treatment (e.g., trilostane, mitotane, ketoconazole). Long-term survival may be possible with RT. Single-fraction modified radiosurgery is a safe and effective approach to the treatment of pituitary tumors in cats. In one study, 11 client-owned cats referred for treatment of pituitary tumor causing neurologic signs or poorly controlled DM secondary to acromegaly or pituitary-dependent HAC underwent MRI of the brain to manually plan RT. Modified radiosurgery was performed by delivering a single large dose (15 or 20 Gy) of radiation while arcing a linear accelerator–generated radiation beam around the cat’s head with the pituitary mass at the center of the beam. Eight cats were treated once, two cats were treated twice, and one cat received three treatments. Five of nine cats with poorly regulated DM had improved insulin responses, and both cats with neurologic signs had clinical improvement. No confirmed acute or late adverse radiation effects were reported. Overall median survival was 25 months (range, 1–60 months), and three cats were still alive at the time of the report.

RT in dogs with pituitary masses may enhance survival and control neurologic signs. In a retrospective study, 19 dogs with pituitary masses identified on CT or MRI were irradiated with 48 Gy given in daily-dose fractions of 3 Gy. Twenty-seven dogs with pituitary masses that received no RT were used for comparison. Mean survival time in the treated group was 1405 days (range, 1053–1757 days) with 1-, 2-, and 3-year estimated survival of 93%, 87%, and 55%, respectively. Median survival in the nonirradiated group was 359 days (range, 48–916 days), with a mean of 551 days (range, 271–829 days). The 1-, 2-, and 3-year estimated survival was 45%, 32%, and 25%, respectively. Dogs that received RT for their pituitary tumors had significantly longer survival times than untreated dogs. Treated dogs with smaller tumors (based on maximal pituitary-to-brain height ratio or area of tumor to area of brain) had longer survival than those with larger tumors ($P < .001$). Stereotactic radiosurgery and stereotactic radiotherapy have been shown to be effective treatments for reducing tumor volume, particularly in dogs with pituitary tumors.

**SURGICAL TREATMENT**

Hypophysectomy can be performed in animals with pituitary microadenomas and functional adenohypophyseal hyperplasia (rare); however, it is seldom performed by veterinary surgeons. Advocates of this procedure suggest that most dogs with PDH are surgical candidates for hypophysectomy, and that this technique is preferable to long-term medical management. The transoral paramedian approach to the pituitary gland has been advocated to remove pituitary tumors, even though some normal tissue may be left behind. Dogs with normal-sized pituitary glands may have fewer postoperative complications than dogs with enlarged pituitary glands. However, if concurrent neurologic signs are present, or if the tumor has extended intracranially or transsphenoidally, hypophysectomy is not indicated. Hypophysectomy should not be considered in animals intended for breeding purposes because it renders them infertile. Transphenoid cryohypophysectomy has been performed safely and effectively in cats. Cats may present with acromegaly caused by elevated growth hormone and insulin-resistant DM.

**NOTE** Hypophysectomy should be performed only by surgeons familiar with the regional anatomy and experienced in the technique.
**Preoperative Management**

If surgery is considered for a pituitary neoplasm, extensive preoperative workup is indicated to confirm and localize the lesion. CT or MRI of the pituitary fossa (including a contrast-enhanced image) is used to determine the height and width of the pituitary gland and to localize pertinent landmarks. Animals with HAC are at increased risk of developing postoperative infection because of high levels of circulating glucocorticoids. Perioperative prophylactic antibiotics are recommended. See p. 594 for additional comments on the preoperative management of animals with HAC.

**Anesthesia**

Most animals with pituitary tumors do not require special anesthetic consideration; however, patients with large masses that increase intracranial pressure need special precautions. Fluid therapy should be restricted to the volume required to maintain adequate circulation. Isoflurane and sevoflurane are the inhalants of choice because they interfere less with autoregulation of cerebral blood flow than does halothane. The faster recovery that sevoflurane offers provides some advantage over isoflurane. Patients with increased intracranial pressure should be modestly hyperventilated (EtCO₂ approximately 30 mm Hg) during surgery. For further discussion of increased intracranial pressures and preoperative management and anesthetic considerations, see Chapter 39, pp. 1338–1342, and Table 39.2.

**Surgical Anatomy**

The pituitary is a small appendage of the diencephalon (Fig. 22.8). It occupies a shallow, oval recess in the basisphenoid bone called the *sella turcica*. The gland varies greatly in size among breeds of dogs and within the same breed, but it is usually approximately 1 cm long. The pituitary is composed of the adenohypophysis and the neurohypophysis, and the adenohypophysis is further subdivided into the pars proximalis, the pars intermedia, and the pars distalis. The arterial supply of the pituitary arises from the internal carotid arteries and caudal communicating arteries.

**SURGICAL TECHNIQUE**

Transsphenoidal, intracranial, and peripharyngeal approaches have been described for hypophysectomy.

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**BOX 22.9 Postoperative Drug Therapy After Hypophysectomy**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Formulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desmopressin Acetate*</td>
<td>Nasal preparation: give 1–4 drops of 100 μg/mL intranasally or in conjunctiva q12–24h</td>
</tr>
<tr>
<td></td>
<td>Parenteral form: give 0.5–2 μg/dog SC or IV q12–24h</td>
</tr>
<tr>
<td></td>
<td>Tablets: 0.05–0.1 mg/kg q12h PO; can increase to 0.2 mg/kg if needed</td>
</tr>
<tr>
<td>Hydrocortisone (Solu-Cortef) or Prednisolone</td>
<td>Hydrocortisone: 1 mg/kg IV q6h</td>
</tr>
<tr>
<td></td>
<td>Prednisolone: 0.2 mg/kg q24h</td>
</tr>
<tr>
<td>Levothyroxine (Soloxide, Thyro-Tabs, Synthroid)</td>
<td>18–22 μg/kg PO q12h</td>
</tr>
</tbody>
</table>

*Can often be discontinued 2 weeks after surgery. IV, Intravenous; PO, orally; SC, subcutaneous.

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**SUTURE MATERIALS AND SPECIAL INSTRUMENTS**

Delayed wound healing may occur in animals with HAC; therefore incisions should be closed with strong, slowly absorbed or nonabsorbable suture material (e.g., polydioxanone, polyglyconate, polypropylene, nylon).

**POSTOPERATIVE CARE AND ASSESSMENT**

See p. 597 for the postoperative management of animals with HAC. See also Box 22.9.

**COMPLICATIONS**

Diabetes insipidus–like symptoms have been reported after hypophysectomy in dogs, although these symptoms tend to resolve in approximately 2 weeks. It is unclear whether return of arginine vasopressin secretion is involved. The presence of increased ACTH concentration after hypophysectomy is a risk factor for recurrence of HAC.

**PROGNOSIS**

Long-term survival is possible after hypophysectomy, RT plus chemotherapy, or chemotherapy alone in dogs with PDH caused by microadenomas. Long-term survival has also been reported in dogs with large, functional tumors after RT. Successful radiation treatment for pituitary tumors has been reported in cats. In a study of 306 dogs with PDH undergoing transsphenoidal hypophysectomy, median survival was 781 days and median disease-free interval was 951 days.¹⁷

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**Surgery of the Pancreas**

**GENERAL PRINCIPLES AND TECHNIQUES**

**DEFINITIONS**

Pancreatectomy is surgical removal of all or part of the pancreas. Insulinoma is a functional tumor of pancreatic β-islet cells; excessive insulin production commonly causes hypoglycemia in affected animals (see p. 609). Zollinger-Ellison syndrome is a...
condition caused by non-β-islet cell tumors, in which excess gastrin is secreted.

**PREOPERATIVE MANAGEMENT**

Although acute onset of anorexia, vomiting, and anterior abdominal pain have been considered the hallmarks of canine pancreatitis, affected dogs may have a wide range of clinical signs, ranging from peracute to chronic and including ascites, shock, dyspnea, and melena. Clinical signs in cats are even more variable, with the primary signs being lethargy, poor appetite, and dehydration; vomiting is much less common or much less noticeable in this species. It is important to try to diagnose pancreatitis before surgery because (1) these animals often neither require nor benefit from surgery if pancreatitis is the major problem (dogs with abscesses may be different; see later discussion) and (2) poor visceral perfusion due to anesthesia and/or unnecessary manipulation of the pancreas during surgery can exacerbate the disease.

Abdominal ultrasonography has been an important tool in the diagnosis of pancreatitis. Ultrasonography is useful in diagnosing pancreatic disease (especially pancreatic and peripancreatic abscess) and in guiding aspirates and biopsies. Sensitivity has been suggested to be in the 40% to 60% bracket. However, it is worth noting that the ultrasonographic appearance of the pancreas in a dog with pancreatitis can change remarkably in hours; therefore repeating ultrasonography a day later might increase its sensitivity. Pancreatitis, pseudocysts, abscesses, neoplastic lesions, nodular hyperplasia, exocrine pancreatic insufficiency (EPI), pancreatitis, pancreatitis, acinar insufficiency, and pancreatic edema cannot always be reliably differentiated.

CT is the technique of choice for diagnosing pancreatitis in humans. In a 2015 pilot study, abdominal CT angiography was performed in 10 dogs under sedation to confirm clinically suspected pancreatitis. Serum pancreatic lipase immunoreactivity as measured by Spec cPL (canine) or Spec fPL (feline) is widely used for the diagnosis of pancreatitis. Sensitivity of Spec cPL is reported to range from 64% to 94%, whereas sensitivity of Spec fPL ranges from 54% to 100%. The reader is referred to a medical text for a more detailed discussion of the diagnosis of pancreatitis.

**NOTE** Spec cPL and Spec fPL tests are sensitive enough to detect histologic pancreatitis that is not clinically important; therefore one should not blindly use these tests as a “litmus” test.

Vomiting animals often require correction of fluid, electrolyte, and acid-base abnormalities before surgery. Diabetic animals may be prone to pancreatitis and are often anesthetized for elective and nonselective procedures. Diabetics should be carefully evaluated before surgery, including complete blood cell count, serum biochemical panel (including fasting blood glucose, blood urea nitrogen, and creatinine), urinalysis, and urine culture. Severe hyperglycemia (>300 mg/dL), ketoadsisis, major electrolyte abnormalities (e.g., hypokalemia, hypophosphatemia), and urinary tract infection should be corrected before surgery. Animals with pancreatic tumors may have a wide variety of metabolic disorders.

**ANESTHESIA**

Many protocols have been described for anesthetic management of diabetic animals. Blood glucose concentrations should ideally be maintained between 100 and 300 mg/dL during surgery. Hypoglycemia may occur if animals are given their regular insulin dose and if food is withheld before surgery; however, the stress of surgery usually results in hyperglycemia. Animals should be fed their normal diet the day before surgery, and their regular dose of insulin should be administered. Food should be withheld 6 to 8 hours before surgery or a small meal given after the morning insulin. Surgery should be performed in the morning. Blood glucose concentrations should be measured the morning of surgery. One to 2 hours before surgery, if the blood glucose concentration is between 150 and 300 mg/dL, the animal should receive one-half of its usual morning dose of insulin subcutaneously. Blood glucose should be checked at induction and hourly thereafter. If the blood glucose level is low, 0.45% saline and 2.5% dextrose (10–15 mL/kg for the first hour, then 5 mL/kg thereafter if blood and evaporative fluid losses are small) should be administered. If the blood glucose level is normal, administer lactated Ringer’s solution (at the same rate). Fluids should be changed to 5% dextrose and an additional small dose of regular insulin given if the blood glucose concentration is greater than 300 mg/dL. The key to management of the diabetic patient is frequent blood glucose checks and an appreciation of the variability of patient responses to insulin therapy.

**NOTE** Be sure to maintain excellent perfusion during surgery to help prevent postoperative pancreatitis.

Selected anesthetic protocols for animals with pancreatic disease that are in stable condition are provided in Table 22.2. These animals may be premedicated with an anticholinergic and opioid, induced with thiopental or propofol, and maintained on isoflurane or sevoflurane inhalants. If the animal is shocky, dehydrated, or hypovolemic, anesthesia must be induced and maintained with greater care. Suggested anesthetic protocols are provided in Table 22.3. An alternative, animals that are not vomiting may be induced with a mask or placed in a chamber, or they may be given thiopental or propofol at reduced dosages. If ketamine is not contraindicated, reduced dosages of diazepam and ketamine may be used.

**NOTE** It is important to realize that dehydration is often not appreciated or is underestimated, especially in obese animals.

**ANTIBIOTICS**

Antibiotics have not been shown to benefit dogs with pancreatitis, which is almost exclusively a nonseptic disease. They are often administered in an effort to prevent secondary infection in necrotic pancreatic and peripancreatic tissue, but no evidence suggests that they benefit the patient. Nonetheless, prophylactic antibiotic therapy (e.g., cefazolin 22 mg/kg IV) is often administered in animals undergoing pancreatic biopsy or partial pancreatectomy to prevent pancreatic abscessation. Treatment with imipenem or ciprofloxacin has been shown to reduce early and late septic pancreatic complications and to improve survival in experimental pancreatitis in rats; however, no evidence indicates that these drugs benefit dogs or cats with pancreatitis. Antibiotic therapy should be based on the results of culture and sensitivity testing of infected tissue in animals with septic pancreatic abscessation.
## TABLE 22.2 Anesthetic Considerations in the Stable Patient With Pancreatic Disease

<table>
<thead>
<tr>
<th>Preoperative Considerations</th>
<th>Intraoperative Considerations</th>
<th>Postoperative Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preoperative Considerations</strong></td>
<td><strong>Induction</strong></td>
<td><strong>Analgesia</strong></td>
</tr>
<tr>
<td>Associated conditions</td>
<td>If premedicated, give:</td>
<td>Fentanyl CRI (1–10 μg/kg IV loading dose, then 2–20 μg/kg/h IV), or</td>
</tr>
<tr>
<td>Diabetes</td>
<td>• Propofol (2–4 mg/kg IV), or</td>
<td>Morphine (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), or</td>
</tr>
<tr>
<td>Often healthy patients before pancreatic disease</td>
<td>• Alfaxalone (2–3 mg/kg IV), or</td>
<td>Hydromorphone (0.05–0.2 mg/kg IV or IM in cats), or</td>
</tr>
<tr>
<td>HCT</td>
<td>• Etomidate (0.5–1.5 mg/kg IV)</td>
<td>Oxymorphone (0.005–0.02 mg/kg IV, IM)</td>
</tr>
<tr>
<td>Electrolytes</td>
<td>If no premedication given, then:</td>
<td>Morphine CRI (0.005–0.02 mg/kg/h IV in dogs), or</td>
</tr>
<tr>
<td>BUN</td>
<td>• Propofol (4–8 mg/kg IV), or</td>
<td>Buprenorphine (0.005–0.02 mg/kg IV, IM)</td>
</tr>
<tr>
<td>Cr</td>
<td>• Alfaxalone (3–5 mg/kg IV), or</td>
<td>Buprenorphine (0.003–0.005 mg/kg diluted in saline)</td>
</tr>
<tr>
<td>TP</td>
<td>• Ketamine (5.5 mg/kg IV) with diazepam (0.28 mg/kg IV)</td>
<td>Lidocaine (&lt;5 mg/kg in dogs; 2–4 mg/kg in cats), or</td>
</tr>
<tr>
<td>Blood glucose, often serial glucose checks</td>
<td>• Isoflurane or sevoflurane plus</td>
<td>Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), or</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>• Fentanyl (2–10 μg/kg IV PRN in dogs; 1–4 μg/kg IV PRN in cats) for short-term pain relief, plus</td>
<td>Oxymorphone (0.01–0.02 mg/kg IV, IM)</td>
</tr>
<tr>
<td>Physical examination</td>
<td>• Hydromorphone (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), or</td>
<td>Buprenorphine (0.005–0.02 mg/kg IV, IM)</td>
</tr>
<tr>
<td>Often middle-aged or elderly patients</td>
<td>• Morphine (0.1–0.2 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), or</td>
<td>Buprenorphine (0.003–0.005 mg/kg diluted in saline)</td>
</tr>
<tr>
<td>Other diagnostics</td>
<td>• Ketamine (low dose) (0.5–1 mg/kg IV once), or</td>
<td>Lidocaine (&lt;5 mg/kg in dogs; 2–4 mg/kg in cats), or</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>• Ketamine CRI (0.5 mg/kg IV loading dose, then 10 μg/kg/min IV) plus</td>
<td>Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), or</td>
</tr>
<tr>
<td>ECG</td>
<td>• For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, or dopamine as needed</td>
<td>Oxymorphone (0.005–0.02 mg/kg IV, IM)</td>
</tr>
<tr>
<td>Bloodwork</td>
<td>Fluid needs</td>
<td>+/− Ketamine CRI (2 μg/kg/min IV). If no previous loading dose, give 0.5 mg/kg IV prior to CRI</td>
</tr>
<tr>
<td>HCT</td>
<td>5–10 mL/kg/h if minimal EBL and minimal evaporative losses, or 10–20 mL/kg/h if open abdomen with higher evaporative losses, plus 3 × EBL</td>
<td>Continued</td>
</tr>
</tbody>
</table>
### TABLE 22.2 Anesthetic Considerations in the Stable Patient With Pancreatic Disease—cont’d

<table>
<thead>
<tr>
<th>Monitoring</th>
<th>Bloodwork</th>
<th>Estimated pain score</th>
</tr>
</thead>
<tbody>
<tr>
<td>• SpO₂</td>
<td>• HCT if significant blood loss</td>
<td>Moderate to severe if open abdominal surgery or if underlying pancreatitis</td>
</tr>
<tr>
<td>• Blood pressure</td>
<td>• Repeat abnormal preoperative bloodwork</td>
<td></td>
</tr>
<tr>
<td>• HR</td>
<td>• Serial blood glucose checks if necessary</td>
<td></td>
</tr>
<tr>
<td>• Respiratory rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Temperature</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• U/O</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• ECG if electrolyte abnormalities</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Preoperative Considerations**

- Associated conditions: Dehydration, Electrolyte abnormalities, Hypotension, Abnormal blood glucose, Adrenal suppression may be present in the critically ill patient.

**Bloodwork**

- HCT
- Electrolytes
- BUN
- Cr
- TP
- Blood glucose, often serial glucose checks
- Urinalysis

**Physical examination**

- Often elderly patients
- May be dehydrated, tachycardic or bradycardic, hypotensive, and/or hypothermic
- Painful abdomen if pancreatitis

**Other diagnostics**

- Blood pressure
- ECG

**Premedications**

- Rehydrate over 4–6 hours if possible; if emergent, may have to give more rapid boluses to expedite time to surgery
- Correct electrolyte abnormalities
- Avoid sedatives in sick patients
- Avoid alpha-2 agonists and acepromazine
- If patient is anxious, give:
  - Midazolam (0.1–0.2 mg/kg IV, IM) or
  - Diazepam (0.1–0.2 mg/kg IV)
- If patient is not depressed, then give:
  - Hydromorphonea (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), or
  - Morphineb (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), or
  - Oxymorphone (0.1–0.2 mg/kg IV, IM), or
  - Buprenorphinec (0.005–0.02 mg/kg IV, IM)

**Intraoperative Considerations**

**Induction**

- If dehydrated, give the following:
  - Etomidate (0.5–1.5 mg/kg IV); if possible, avoid its use in critically ill patients, or
  - Propofol (1–4 mg/kg IV) slowly, or
  - Alfaxalone (2–3 mg/kg IV)
- If hydrated, give the following:
  - Propofol (2–6 mg/kg), or
  - Alfaxalone (3–5 mg/kg IV)

**Maintenance**

- Isoflurane or sevoflurane plus
- Fentanyl (2–10 μg/kg IV PRN in dogs; 1–4 μg/kg IV PRN in cats) for short-term pain relief, plus PRN
- Fentanyl CRI (1–5 μg/kg IV loading dose, then 2–30 μg/kg/h IV), or
- Hydromorphonea (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), or
- Buprenorphinec (0.005–0.02 mg/kg IV PRN, plus
- Ketamine (low dose) (0.5–1 mg/kg IV), or
  - Ketamine CRI (0.5 mg/kg IV loading dose, then 10 μg/kg/min IV)
- For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, or dopamine as needed

---

*Monitor for hyperthermia in cats.
*Give slowly to prevent histamine release.
*Buprenorphine is a better analgesic than morphine in cats.

**BUN, Blood urea nitrogen; Cr, creatinine; CRI, constant-rate infusion; EBL, estimated blood loss; ECG, electrocardiogram; EtCO₂, end-tidal CO₂; HCT, hematocrit; HR, heart rate; IM, intramuscular; IV, intravenous; MAP, mean arterial pressure; OTM, oral transmucosal; PRN, as needed; SpO₂, hemoglobin saturation with oxygen; TP, total protein; U/O, urine output.

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**TABLE 22.3 Anesthetic Considerations in the Compromised Patient With Pancreatic Disease**

<table>
<thead>
<tr>
<th>Preoperative Considerations</th>
<th>Bloodwork</th>
</tr>
</thead>
<tbody>
<tr>
<td>Associated conditions</td>
<td>HCT</td>
</tr>
<tr>
<td>Dehydration</td>
<td>Electrolytes</td>
</tr>
<tr>
<td>Electrolyte abnormalities</td>
<td>BUN</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Cr</td>
</tr>
<tr>
<td>Abnormal blood glucose</td>
<td>TP</td>
</tr>
<tr>
<td>Adrenal suppression</td>
<td>Blood glucose, often serial glucose checks</td>
</tr>
<tr>
<td>patients</td>
<td>Urinalysis</td>
</tr>
<tr>
<td>Adrenal suppression</td>
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<tr>
<td>may be present in the</td>
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<tr>
<td>critically ill patient</td>
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<tr>
<td>Physical examination</td>
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<tr>
<td>Often elderly patients</td>
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<tr>
<td>May be dehydrated,</td>
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<tr>
<td>tachycardic or bradycardic</td>
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<tr>
<td>hypotensive, and/or</td>
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<tr>
<td>hypothermic</td>
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<tr>
<td>Painful abdomen</td>
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<td>if pancreatitis</td>
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<tr>
<td>Other diagnostics</td>
<td>Blood pressure</td>
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<tr>
<td>Blood pressure</td>
<td>ECG</td>
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<td>Premedications</td>
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<tr>
<td>Rehydrate over 4–6 hours</td>
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<td>if possible; if emergent,</td>
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<td>may have to give more</td>
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<td>rapid boluses to expedite</td>
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<td>time to surgery</td>
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<tr>
<td>Correct electrolyte</td>
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<tr>
<td>abnormalities</td>
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<tr>
<td>Avoid sedatives in sick</td>
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<td>patients</td>
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<tr>
<td>Avoid alpha-2 agonists</td>
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<tr>
<td>and acepromazine</td>
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<td>If patient is anxious, give:</td>
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<tr>
<td>Midazolam (0.1–0.2 mg/kg IV, IM) or</td>
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<tr>
<td>Diazepam (0.1–0.2 mg/kg IV)</td>
<td></td>
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<tr>
<td>If patient is not depressed, then give:</td>
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<tr>
<td>Hydromorphone (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), or</td>
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<tr>
<td>Morphine (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), or</td>
<td></td>
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<tr>
<td>Oxymorphone (0.1–0.2 mg/kg IV, IM), or</td>
<td></td>
</tr>
<tr>
<td>Buprenorphine (0.005–0.02 mg/kg IV, IM)</td>
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**Intraoperative Considerations**

**Induction**

- If dehydrated, give the following:
  - Etomidate (0.5–1.5 mg/kg IV); if possible, avoid its use in critically ill patients, or
  - Propofol (1–4 mg/kg IV) slowly, or
  - Alfaxalone (2–3 mg/kg IV)
- If hydrated, give the following:
  - Propofol (2–6 mg/kg), or
  - Alfaxalone (3–5 mg/kg IV)

**Maintenance**

- Isoflurane or sevoflurane plus
- Fentanyl (2–10 μg/kg IV PRN in dogs; 1–4 μg/kg IV PRN in cats) for short-term pain relief, plus PRN
- Fentanyl CRI (1–5 μg/kg IV loading dose, then 2–30 μg/kg/h IV), or
- Hydromorphone (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), or
- Buprenorphine (0.005–0.02 mg/kg IV PRN, plus
- Ketamine (low dose) (0.5–1 mg/kg IV), or
  - Ketamine CRI (0.5 mg/kg IV loading dose, then 10 μg/kg/min IV)
- For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, or dopamine as needed
### Anesthetic Considerations in the Compromised Patient With Pancreatic Disease—cont’d

| Fluid needs | • 5–10 mL/kg/h if minimal EBL and minimal evaporative losses, or 10–20 mL/kg/h if open abdomen with higher evaporative losses, plus 3 × EBL.  
| Monitoring | • Blood pressure  
| Blocks | • ECG  
| | • Respiratory rate  
| | • \(\text{SpO}_2\)  
| | • \(\text{EtCO}_2\)  
| | • Temperature  
| | • U/O  
| | • Consider colloids if persistent hypotension  
| | | Epidural:  
| | | • Morphine (0.1 mg/kg preservative free) or  
| | | • Buprenorphine (0.003–0.005 mg/kg diluted in saline)  
| | | • Avoid local anesthetics for spinals and epidurals in hypotensive patients  
| | Incisional:  
| | | • Lidocaine (< 5 mg/kg in dogs; 2–4 mg/kg in cats), or  
| | | • Bupivicaine (< 2 mg/kg)  
| Postoperative Considerations | • Fentanyl CRI (1–10 μg/kg IV loading dose, then 2–20 μg/kg/h IV), or  
| | • Morphine\(^{\text{a}}\) (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, or  
| | • Hydromorphone\(^{\text{b}}\) (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), or hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), or  
| | • Oxymorphone (0.1–0.2 mg/kg IV, IM), or  
| | • Buprenorphine\(^{\text{c}}\) (0.005–0.02 mg/kg IV, IM q4–8h; 0.01–0.02 mg/kg OTM q6–12h in cats), plus  
| | | +/− Ketamine CRI (2 μg/kg/min IV). If no previous loading dose, give 0.5 mg/kg IV prior to CRI  
| | • Avoid NSAIDs in patients with hypotension  
| Monitoring | • \(\text{SpO}_2\)  
| | • Blood pressure  
| | • HR  
| | • Respiratory rate  
| | • Temperature  
| | • U/O  
| | • ECG if electrolyte abnormalities  
| Bloodwork | • HCT if significant blood loss  
| | • Repeat abnormal preoperative bloodwork  
| | • Serial blood glucose checks if necessary  
| Estimated pain score | Moderate to severe if open abdominal surgery or if underlying pancreatitis  

\(^{\text{a}}\)Monitor for hyperthermia in cats.  
\(^{\text{b}}\)Give slowly to prevent histamine release.  
\(^{\text{c}}\)Buprenorphine is a better analgesic than morphine in cats.

**SURGICAL ANATOMY**

The pancreas of dogs and cats is composed of a right and a left limb and a small central body (Fig. 22.9). The right limb of the pancreas lies within the mesoduodenum and is closely associated with the duodenum, particularly at its cranial aspect. The dorsal aspect of the right pancreatic lobe is visualized by retracting the duodenum ventrally and toward the midline; the ventral aspect of the right pancreatic lobe is examined by retracting the duodenum laterally. The pancreatic body (angle) lies in the bend formed by the pylorus and the duodenum. The left pancreatic lobe is viewed within the deep leaf of the greater omentum by retracting the stomach cranially and the transverse colon caudally.

**NOTE** Visualize the left lobe of the pancreas by looking in the deep leaf of the greater omentum while retracting the stomach cranially.

The main blood supply to the left pancreatic lobe is provided via branches of the splenic artery; however, branches from the common hepatic and gastroduodenal arteries also supply portions of it. The main vessels of the right lobe of the pancreas are the pancreatic branches of the cranial and caudal pancreaticoduodenal arteries that anastomose in the gland. The cranial pancreatico-duodenal artery is a terminal branch of the hepatic artery; the caudal pancreatico-duodenal arises from the cranial mesenteric vessel. These vessels also provide branches that supply the duodenum. Because they are closely associated with the proximal portion of the right lobe of the pancreas, care must be taken not to damage these vessels during pancreatic surgery, or devitalization of the duodenum may occur.

**NOTE** The proximity and shared blood supply of the pancreas and duodenum make duodenal resection difficult if pancreatic function is to be maintained.
The pancreas has both endocrine (insulin) and exocrine (digestive secretions) functions. Digestive secretions enter the duodenum via one of two ducts. These ducts may communicate within the gland or may cross each other. When the two ducts do not communicate, the pancreatic duct drains the right lobe and the accessory pancreatic duct drains the left lobe. The accessory pancreatic duct is the largest excretory pancreatic duct in dogs. It opens into the duodenum at the minor duodenal papilla. The smaller pancreatic duct is occasionally absent. The latter usually enters the duodenum on the major duodenal papilla, adjacent to the common bile duct. The pancreatic duct is the principal and oftentimes the only duct in cats.

**NOTE** Extrahepatic biliary obstruction may occur secondary to pancreatic swelling or masses because of impingement of the common bile duct as it enters the major duodenal papilla. Attempting to resect such masses is typically contraindicated because it can easily result in laceration and/or rupture of the bile duct.

**SURGICAL TECHNIQUE**

A ventral midline abdominal incision is made, extending from the xiphoid cartilage caudal to the umbilicus, and the pancreas is examined using a combination of gentle palpation and visual inspection. The free portion of the greater omentum is retracted cranially and is covered with moist sponges. The omental leaf overlying the pancreas can be bluntly separated to allow direct visualization of the left pancreas. When neoplasia is suspected, lymph nodes that lie along the splenic vessels and portal vein and those at the hilus of the liver and head of the pancreas should be examined for evidence of metastasis.

Pancreatic biopsy and partial pancreatectomy are performed in dogs and cats. Because of the difficulty in diagnosing feline pancreatitis, biopsy may be indicated more often than currently performed. Laparoscopic biopsy of the canine and feline pancreas is generally well tolerated. Pancreatic biopsy is occasionally performed in dogs to differentiate benign pancreatic conditions (e.g., pancreatitis, pancreatic fibrosis) from neoplastic disease. Although ultrasound-guided biopsies of large pancreatic lesions may be possible, exploratory laparotomy and direct visualization of pancreatic tissue are usually indicated. Partial pancreatectomy is indicated in animals with insulin-secreting or gastrin-secreting tumors and in those with pancreatic adenocarcinoma (see p. 613). Total pancreatectomy is infrequently performed in veterinary patients. Removal of the pancreas without duodenectomy requires that pancreatic tissue be bluntly dissected from the pancreaticoduodenal vessels without damage to branches supplying the duodenum. This is difficult in animals with pancreatic disease because of adhesions, fibrosis, and edema. Therefore total pancreatectomy is usually performed in conjunction with resection and anastomosis of the proximal duodenum (i.e., Billroth II procedure), ligation of the common bile duct, and cholecystojjunostomy (see p. 574) and is associated with high rates of morbidity and mortality. Pancreatic drainage or omentalization is indicated in conditions (e.g., large abscesses or cysts) in which pancreatectomy is not feasible.

**NOTE** Handle the pancreas gently to avoid causing pancreatitis.

**NOTE** Total pancreatectomy is difficult because it usually necessitates cholecystoenterostomy and removal of the duodenum.

**Laparoscopic Pancreatic Biopsy**

This is generally performed during two-port laparoscopy. As previously noted, diagnosing pancreatitis in cats can be more difficult than in dogs; therefore pancreatic biopsy is more commonly done in cats. *Use a punch type of biopsy forceps (as opposed to ‘double spoon’ forceps).* Obtain biopsies from the edge of the pancreas and take care to examine both sides (a large vein typically runs under the pancreas, near the edge). If you see obviously diseased pancreatic tissue, take a biopsy; however, some cats with pancreatitis do not have grossly diseased pancreatic tissue. Because canine pancreatitis can be a focal or multifocal lesion, take multiple biopsies from such animals.

**Surgical Pancreatic Biopsy**

If obvious, diffuse pancreatic disease is present, a biopsy is best obtained by removing a small portion of the caudal aspect of the right pancreatic limb (see the discussion of Partial Pancreatectomy in the next section). Focal lesions near the extremity of the pancreas may be removed in a similar fashion. If no obviously diseased tissue is noted, then multiple biopsies should be taken, as pancreatitis can be a localized or multifocal disease. *For focal lesions within the pancreatic parenchyma, use a TruCut (Cardinal Health) or Vim-Silverman needle (see p. 544), or take a portion of the lesion using a guillotine ligature of absorbable suture to obtain a small sample of pancreatic tissue. Take care not to damage adjacent blood vessels or pancreatic ducts.*

**FIG. 22.9** Vascular supply to the pancreas.
CHAPTER 22  Surgery of the Endocrine System

HEALING OF THE PANCREAS

The fibrous stroma of the pancreas allows healing to occur by protein synthesis, epithelialization, and fibrin polymerization. Obstruction of the pancreatic duct is seldom caused by wound contraction; rather, parenchymal edema or obstruction at the duodenal papilla is usually the cause. The main concern associated with pancreatic healing after surgery is the effect of healing on the flow and drainage of pancreatic secretions. If the duct to the remaining portion is left intact, as much as 80% of the pancreas can be removed without causing deleterious decreases in exocrine or endocrine function.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Duct ligation is performed with absorbable suture material (e.g., polydioxanone, polyglyconate, glycomer 631, or poliglecaprone 25) in animals with inflammatory, aseptic, or neoplastic conditions; braided suture material should be avoided.

POSTOPERATIVE CARE AND ASSESSMENT

Medical treatment of canine and feline pancreatitis is replete with opinions, but as of this writing, no well-designed, prospective, controlled studies have been performed. The following recommendations are generally agreed upon, but the reader is referred to a current medical text for a more complete discussion.
Classically, enteral feeding has been delayed for 2 to 5 days after extensive pancreatic surgery is performed, or if pancreatitis is present. It is now generally agreed that keeping dogs with pancreatitis off food and putting them into a negative nitrogen balance is deleterious. Patients with pancreatitis should be fed as soon as they can tolerate enteral alimentation (per os or via esophagostomy tube). Feeding should be initiated with small amounts of low-fat (probably less than 2–3 g fat/100 kcal), bland food (e.g., rice, defatted white chicken, or white turkey without the skin). Animals with severe or prolonged pancreatitis that cannot accept enteral nutrition without obviously becoming worse may benefit from parenteral nutrition (see Chapter 10). Hydration and electrolytes (especially potassium) should be maintained with IV fluid therapy because pancreatic perfusion is probably paramount in healing the diseased pancreas. Visceral perfusion may be inadequate in animals that subjectively appear normally hydrated; dehydration is often not obvious clinically. Plasma oncotic pressure should be maintained by replacing colloid (e.g., hetastarch or pentastarch) if hypoalbuminemia (i.e., serum albumin <2 g/dL) occurs (see Chapter 4). Plasma is much less effective in raising plasma oncotic pressure. Treating patients with DIC consists of maintaining an adequate blood pressure and thus perfusion pressure, as well as replacing clotting factors with fresh-frozen plasma transfusions; heparin therapy may be given in addition. There is general consensus that early intervention in animals with DIC increases their chances of survival. Antiemetics and analgesics may be administered as needed. If sepsis is identified, appropriate antibiotic therapy should generally be continued for 10 to 14 days after surgery, but this is exceedingly rare in dogs.

**COMPLICATIONS**

The most common complication of pancreatic surgery is pancreatitis; this can be minimized by maintaining good visceral perfusion and gentle tissue handling. In a study of postoperative complications following pancreatic biopsy in dogs and cats, postoperative pancreatitis occurred in 11.6% of patients.²⁰ Anecdotally, octreotide (1–2 μg/kg given subcutaneously before surgery) has been used to try to prevent postoperative pancreatitis.

Severe, acute pancreatitis puts the patient at risk for mortality from multiorgan failure. EPI may occur if pancreatic drainage is completely obstructed. EPI is treated with pancreatic supplements of commercial pancreatic extract (e.g., Viokase; Box 22.10) and feeding of low-fat, highly digestible meals. Endocrine pancreatic insufficiency (DM) may result when more than 80% to 90% of the pancreatic tissue is removed. Supplementation with insulin may be necessary.

**SPECIAL AGE CONSIDERATIONS**

Pancreatic disease is usually found in middle-aged or older animals. Special care must be taken to meet the nutritional and metabolic needs of geriatric patients, particularly when disease may have caused inappetence or chronic vomiting. Parenteral hyperalimentation may be necessary before and after surgery in these patients.

**SPECIFIC DISEASES**

**PANCREATIC ABSCESSES AND PSEUDOCYSTS**

**DEFINITIONS**

Pancreatic abscesses are a collection of purulent material and necrotic tissue within and extending from the pancreatic parenchyma. Pancreatic pseudocysts are collections of pancreatic secretions and cellular debris enclosed within a wall of granulation tissue.
tissue or fibrous sac that lacks an epithelial wall. Pancreatic pseudocysts have also been called pancreatic cysts.

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

Pancreatic abscesses (Fig. 22.12) are pancreatic or peripancreatic collections of purulent, necrotic, and hemorrhagic tissue that probably occur as a consequence of acute pancreatitis. Pancreatic abscesses may also occur in human beings as a consequence of chronic ductal obstruction.

Pancreatic pseudocysts (Fig. 22.13) are a common complication of acute pancreatitis in human beings but are rarely diagnosed in small animals. They may be associated with recurrent bouts of pancreatitis or trauma. Fluid in the cysts is a combination of blood, pancreatic fluids, and enzymes. These are not true cysts because the fluid is thought to leak from damaged pancreatic ducts and vessels rather than being secreted by the lining of the cyst. Pancreatic pseudocysts may be incidental findings or may be associated with nonspecific abdominal signs, such as pain and vomiting. Complications such as infection, rupture, or acute hemorrhage may occur in people and are associated with a high mortality rate.

**NOTE** Large pancreatic masses in symptomatic or asymptomatic animals may be pseudocysts or sterile abscesses; cancer is a much less common event. Do not recommend euthanasia solely on the basis of a radiographic finding of an abdominal mass.

**DIAGNOSIS**

**Clinical Presentation**

**Signalment**

Pancreatic abscesses/pseudocysts probably arise after acute pancreatitis; therefore the signalment of these animals closely parallels that of animals diagnosed with acute pancreatitis (see p. 600). Most animals are middle-aged or older, and dogs are more commonly affected than cats.

**History**

Animals with pancreatic abscesses/pseudocysts may have a previous history of acute onset of anorexia, depression, diarrhea, or vomiting; some have previously been treated for gastroenteritis that was probably pancreatitis. Other clinical findings may include ataxia, anorexia, abdominal pain, or pyrexia. These patients may have dramatic acute signs, or vague, smoldering, chronic signs, or they may be asymptomatic.

**Physical Examination Findings**

Typical findings with pancreatic abscesses/pseudocysts may include pain during abdominal palpation, depression, icterus, pyrexia, palpable cranial abdominal mass, or abdominal distention. Some animals may be weak and reluctant to stand. Pyrexia is an uncommon finding. However, some animals are asymptomatic.

**Diagnostic Imaging**

The most consistent finding with pancreatic abscess/pseudocyst on survey abdominal radiographs is an ill-defined increase in soft tissue density in the right cranial abdominal quadrant. If peritonitis is present, a generalized increase in soft tissue opacity and loss of visceral detail in the right quadrant or throughout the abdomen may be observed. Abdominal ultrasonography is more sensitive and usually reveals a mass in the area of the pancreas. Gallbladder and bile duct distention may also be noted. Ultrasonography may also identify pancreatitis (Fig. 22.14). Gastric outflow obstruction is rarely observed on contrast studies.

**FIG. 22.12** Pancreatic abscess in a dog.

**FIG. 22.13** Pancreatic pseudocyst. (Courtesy H.P. Hobson, Texas A&M University.)

**FIG. 22.14** Ultrasound parasagittal image of the pancreas. It is hypoechoic (arrows), enlarged, and surrounded by hyperechoic fat consistent with pancreatitis.
of the upper gastrointestinal tract. Ultrasonography is the best tool for identifying pancreatic abscess/pseudocyst; however, differentiation of pseudocysts from other fluid-filled masses is not possible without evaluation of the fluid. Percutaneous fine-needle aspiration of masses is reasonable in dogs because of the extremely low incidence of septic pancreatitis in this species. Risk must be weighed against the advantage of a preoperative diagnosis. Resolution of pancreatic abscess/pseudocysts after percutaneous, ultrasound-guided drainage is possible.

NOTE Fine-needle aspiration of cavitary pancreatic masses may involve some risk. Perform with caution and observe these animals carefully after the procedure.

**Laboratory Findings**

Hematologic and serum biochemical findings with pancreatic abscess/pseudocyst are inconsistent but may include leukocytosis, neutrophilia with or without a left shift, lymphopenia, or monocytosis. Serum biochemical abnormalities may include hyperbilirubinemia and high serum alkaline phosphatase caused by extrahepatic cholestasis, high alanine aminotransferase, hypocholesterolemia, hyponatremia, hypokalemia, and hyperkalemia. Serum lipase and serum amylase are insensitive and nonspecific; they should not be requested. Bilirubinuria is often present.

NOTE The absence of neutrophilia does not exclude pancreatic abscessation.

**DIFFERENTIAL DIAGNOSIS**

Pancreatic abscess/pseudocyst must be differentiated from other causes of vomiting and cranial abdominal pain (e.g., pancreatitis, gastric foreign bodies, intestinal foreign bodies, gastritis, cholecystitis, pancreatic neoplasia, gastrointestinal neoplasia). Ultrasound evaluation of the pancreas is the most useful test for differentiating these abnormalities preoperatively; however, exploratory surgery may be required in some animals to make a definitive diagnosis.

**MEDICAL MANAGEMENT**

Pancreatic abscesses/pseudocysts have classically been considered surgical diseases. However, we now realize that in some patients they can be resolved with percutaneous drainage, and others (typically those fortuitously diagnosed on abdominal ultrasound for some other reason) may need no treatment. These lesions are almost invariably sterile in dogs, but septic abscesses can occur in cats. The mortality rate in human beings with pancreatic abscesses is nearly 100% when medical therapy without drainage is used; with surgical treatment, mortality has been reduced. Similar studies have not been reported in dogs or cats. Some pancreatic abscesses/pseudocysts may resolve spontaneously without therapy.

**SURGICAL TREATMENT**

Symptomatic dogs with abscesses or pseudocysts probably benefit from drainage procedures; however, many dogs with pancreatitis have hypoechoic areas in the pancreas that do not represent fluid accumulations that can be drained. Generalized, sterile peritonitis is present in some dogs with pancreatic abscess/pseudocyst; if septic peritonitis or pancreatitis is present, a thorough search should be made for a primary cause such as a perforated duodenum. On opening the abdomen, a mass is observed originating from the pancreas in the cranial portion of the abdomen. The mass may be firm and fibrotic or friable. Multiple adhesions to omentum and adjacent loops of small or large intestine are often present. These lesions can look malignant; however, a vast majority of pancreatic lesions and masses are inflammatory without malignancy, regardless of how bad they appear. Adhesions may be present if the lesion has ruptured and reformed. If the abscess or pseudocyst is surgically resolved, omentalization is preferred over external drainage (see later discussion).

**Preoperative Management**

If clinically apparent pancreatitis is present, medical management should be initiated before surgery (percutaneous drainage may proceed if deemed important). See the discussion on p. 606 for information on the treatment of pancreatitis. A broad-spectrum antibiotic can be administered intravenously before surgery if sepsis is believed to be present, but this is seldom necessary. If infection is found by cytology or culture, then antibiotics should be used for at least 10 to 14 days postoperatively.

**Anesthesia**

See the discussion of the anesthetic management of animals with pancreatic disease on p. 600.

**Surgical Anatomy**

See the discussion of the surgical anatomy of the pancreas on p. 603.

**Positioning**

The animal is positioned in dorsal recumbency, and the caudal thorax and the entire abdomen are prepared for aseptic surgery.

**SURGICAL TECHNIQUE**

**Pancreatic Abscesses/Pseudocysts**

Perform a midline abdominal laparotomy that extends from the xiphoid cartilage caudally to distal to the umbilicus. Gently explore the abdomen. Locate the pancreatic mass and obtain cultures of infected tissue. Gently break down adhesions in the intestine and omentum as needed to visualize the lesion. Preserve the pancreatic ducts, common bile ducts, and adjacent vascular structures during dissection. Debride necrotic or purulent areas of the pancreas using a combination of sharp and blunt dissection. Resect as much of the necrotic pancreas as possible without damaging adjacent blood vessels or tissue. Once the lesion has been debrided, place a piece of omentum in it and secure it with sutures. If possible, loop the omentum through a tunnel in the pancreatic tissue and suture it back to itself. Determine common bile duct patency by gently expressing the gallbladder. If the common bile duct is not patent, catheterize the duct and try to obtain flow or perform a cholecystoenterostomy (see p. 574). Make sure you do not ligate the common bile duct. If generalized peritonitis is present, lavage the abdomen thoroughly with warm, sterile saline or lactated Ringer’s solution. If peritonitis is present close the abdomen, insert a drain, or leave it open for drainage (see p. 534).
tumors, adenomas, adenocarcinomas of the pancreatic islets. 

postoperative complications may be required occasionally. Blood cultures are warranted if bacteremia is suspected. Pancreatitis is a potential complication of any surgery involving the pancreas (see p. 606).

**NOTE** Warn owners that repeat surgery may be necessary in some animals with pancreatic abscesses/pseudocysts.

**DIAGNOSIS**

**Clinical Presentation**

**Signalment**

Insulinomas generally occur in middle-aged or older dogs; no gender predilection has been noted. Medium- to large-breed dogs (e.g., Irish setters, German shepherds, Labrador retrievers, standard poodles, boxers) appear to be more commonly affected.

**History**

The clinical signs are attributable to hypoglycemia and include muscle tremors, muscle weakness, ataxia, mental dullness, disorientation, collapse, and/or convulsions. Dogs may be easily agitated and may have intermittent periods of excitability and restlessness. These clinical signs suggest hypoglycemia from any cause, not just insulinoma. Owners may notice clinical signs for months before presenting the animals for evaluation. The clinical signs are often intermittent initially but occur more frequently as the disease progresses. Owners often report that clinical signs diminish or resolve with feeding. Animals are sometimes treated for seizures with anticonvulsant agents before the diagnosis is made.

**Physical Examination Findings**

Physical examination findings may reveal a normal or ataxic animal, muscle weakness (usually seen as shaking or collapse in the rear), mental dullness, or disorientation. Affected dogs are usually normal between hypoglycemic episodes, a fact that may help differentiate insulinoma from other causes of hypoglycemia. Withholding food before and during the evaluation may precipitate seizures in affected animals. Neuronal demyelination and axonal degeneration may result from chronic hypoglycemia. Although the cause is not known for certain, direct toxic effects of hypoglycemia on peripheral nerves or a paraneoplastic neuropathy has been postulated. Signs of peripheral polyneuropathy, such as ataxia and weakness, may continue despite appropriate therapy.

**Diagnostic Imaging**

Thoracic and abdominal radiographs do not contribute to the diagnosis; however, the location of the tumor in the pancreas can sometimes be determined using ultrasound. Unfortunately, tumor masses are often so small that they are difficult to identify. Ultrasonography may reveal metastasis to the liver and regional lymph nodes in some affected animals. Thoracic radiographs are indicated to look for metastasis, although pulmonary metastasis is rare. Abdominal and thoracic CT can be used to identify thoracic and lymph node metastasis, but identification of false-positive lesions is possible. Therefore intraoperative inspection and palpation of the pancreas are superior to CT. Positron emission tomography/CT may be more effective than ultrasound or CT in identifying canine insulinomas, but this has yet to be fully evaluated.

**NOTE** More than 90% of canine insulinomas are malignant. They nearly always metastasize even though they may lack histologic criteria of malignancy.

**SUTURE MATERIALS AND SPECIAL INSTRUMENTS**

Absorbable suture material should be used for partial pancreatectomy in animals with pancreatic abscesses/pseudocysts. Aerobic and anaerobic culture swabs should be available. Copious amounts of warmed fluids should be available for abdominal flushing; suction allows complete removal of instilled fluid and facilitates dilution of infected fluids in the abdominal cavity.

**POSTOPERATIVE CARE AND ASSESSMENT**

The patient should be treated for pancreatitis, as described previously. Antibiotic therapy should be continued if infection is found. Animals should be monitored postoperatively for signs of worsening inflammation. Clinical assessment of these patients (e.g., presence or absence of fever, abdominal pain, anorexia, vomiting, and icterus) is more important than the complete blood cell count or ultrasonographic appearance. Repeat operations may be required occasionally. Blood cultures are warranted if bacteremia is suspected. Pancreatitis is a potential complication of any surgery involving the pancreas (see p. 606).

**NOTE** Warn owners that repeat surgery may be necessary in some animals with pancreatic abscesses/pseudocysts.

**PROGNOSIS**

The prognosis in animals with inflammatory pancreatic lesions is guarded. Mortality rates appear to be higher with necrotic mass lesions of the pancreas and pancreatic abscesses than with pancreatic pseudocysts. Cats undergoing surgery because of extrahepatic biliary obstruction secondary to severe acute pancreatitis that does not respond to medical management may have a good prognosis. Postoperative complications that have been described include progression of DM, septic peritonitis, local gastrostomy tube stoma inflammation, local gastrostomy tube stoma infection, and mild dermal suture reaction.

**INSULINOMAS**

**DEFINITION**

Insulinomas are functional tumors of the β-cells of the islets of Langerhans. These tumors secrete insulin despite the presence of hypoglycemia. They have also been called pancreatic β-cell tumors, adenomas, or adenocarcinomas of the pancreatic islets.

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

Insulinomas are pancreatic islet cell tumors that secrete excessive amounts of insulin, causing hypoglycemia. They are more commonly recognized in dogs than in cats. Unlike human beings, in whom up to 90% of insulinomas are benign, malignant tumors predominate in dogs. They typically metastasize to the regional lymph nodes, liver, and omentum. Occasionally nodules may be found in the lung. They are slow-growing tumors that compress adjacent pancreatic parenchyma. Because they are typically sharply delineated and encapsulated, palliative surgical excision often prolongs survival. Tumor state may correlate with survival time following surgery and medical management (see Prognosis section).
Laboratory Findings
A tentative diagnosis of insulinoma is based on demonstration of Whipple's triad (Box 22.11). Fasting or nonfasting blood glucose concentrations are often below 70 mg/dL. If blood glucose concentrations initially are within the normal range, most affected dogs can be made hypoglycemic by fasting for 12 to 24 hours. Blood glucose measurements should be determined every 2 to 3 hours in these animals until hypoglycemia is detected. Serum fructosamine may be useful for diagnosing chronic, occult hypoglycemia.

Once hypoglycemia has been confirmed, blood for serum insulin measurement should be obtained immediately. If food has been withheld from the animal to induce hypoglycemia, serum insulin concentrations should be measured on the first hypoglycemic sample (i.e., <55 mg/dL). Normal fasting serum immunoreactive insulin concentrations range from 5 to 26 μIU/mL, whereas insulin levels in affected animals often exceed 70 μIU/mL. Evaluating the absolute insulin concentration when the patient is hypoglycemic while considering the history, physical examination, and other clinical pathology data is the best approach. In some cases, definitive diagnosis of insulinoma may require exploratory surgery.

Differential Diagnosis
Insulinomas should be considered a differential diagnosis in any dog with persistent and progressive seizures. Once hypoglycemia has been verified, these tumors must be differentiated from other causes of hypoglycemia, including extrapancreatic neoplasms, hunting dog hypoglycemia, sepsis, hepatic failure, hypoadrenocorticism, glycogen storage disorders (very rare), and hypopituitarism.

Medical Management
Dogs with insulinomas should be fed frequent, small meals. Three to six meals a day of a diet high in protein and complex carbohydrates but low in refined sugar reduces clinical signs. Exercise restriction may help alleviate clinical signs. Glucocorticoid therapy (Box 22.12) may help prevent hypoglycemia caused by islet cell tumors by increasing hepatic glucose production and decreasing cellular glucose uptake. The lowest possible dose that controls hypoglycemia should be used to prevent iatrogenic HAC (e.g., polyphagia, polydipsia, bilateral symmetric alopecia, thin epidermis). If clinical signs of HAC occur, glucocorticoid therapy may be reduced and alternate drugs used; however, HAC may be preferable to hypoglycemia. Diazoxide (see Box 22.12) is an oral hyperglycemic agent that inhibits pancreatic insulin secretion and glucose uptake by tissue. It raises blood glucose concentrations in some dogs with insulinomas; however, side effects, such as anorexia, vomiting, aplastic anemia, cataracts, bone marrow suppression, thrombocytopenia, anorexia, diarrhea, tachycardia, and fluid retention, may occur. Diazoxide should be used cautiously in animals with hepatic dysfunction. If hypoglycemia is severe and unresponsive, IV administration of 5% or 10% dextrose may be necessary to maintain blood glucose concentrations in the normal range until surgery can be performed. Alloxan and a somatostatin analog (octreotide) have been used in a few dogs with insulinomas; however, too little information is available at this time to recommend their use.

Streptozotocin may be efficacious in dogs with insulinomas that have metastatic disease. It has been suggested that it can be given safely to dogs at a dosage of 500 mg/m² IV every 3 weeks when combined with a protocol for induction of diuresis. Give 0.9% NaCl administered at a rate of 18.3 mL/kg per hour IV for 3 hours before streptozotocin administration. Dilute the streptozotocin to an appropriate volume and give over 2 hours at the same rate as the fluid administration. Then give 0.9% NaCl for an additional 2 hours. To reduce vomiting, butorphanol or antiemetics may be given intramuscularly immediately after streptozotocin administration; however, these drugs are usually ineffective. Repeat the treatment every 3 weeks until tumor progression is evident (i.e., the tumor increases significantly in size), recurrence of hypoglycemia is observed, or toxicity (e.g., renal, hepatic) is noted that is unresponsive to supportive care. Other abnormalities that may occur in association with streptozotocin administration include neutropenia, thrombocytopenia, anorexia, diarrhea, and DM.

Surgical Treatment

Preoperative Management
Fluid therapy with 5% glucose should be initiated 12 to 24 hours before surgery. Food is withheld 6 to 8 hours before surgery. Blood glucose concentrations should be measured immediately before surgery and additional glucose given if the concentration is below 75 to 100 mg/dL.

Anesthesia
The goal of surgery is to maintain blood glucose concentrations above 75 to 200 mg/dL. Thiopental, propofol, or alfaxalone may be used for induction of anesthesia because they reduce cerebral glucose metabolism. Etomidate should be avoided because it may cause adrenal suppression. After intubation, anesthesia should be maintained with isoflurane or sevoflurane. Isoflurane and sevoflurane reduce the cerebral metabolic rate more than does halothane. Blood glucose concentrations should be monitored regularly during surgery (i.e., every 20–40 minutes) to prevent intraoperative hypoglycemia.

Surgical Anatomy
See the discussion of the surgical anatomy of the pancreas on p. 603.
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Methylene blue may be given intravenously to help identify primary and metastatic nodules (see previous discussion).

Postoperative Care and Assessment

Blood glucose concentrations should be measured frequently during the first 24 hours after surgery. Pancreatitis may result from surgical manipulation of the pancreas and should be treated aggressively, as described on p. 605. Small amounts of water may be administered the day after surgery, and if vomiting does not occur, feeding of small, frequent meals may be initiated. Once the blood glucose concentration stabilizes at 75 to 100 mg/dL or higher, the glucose infusion can be discontinued (Box 22.14).

If persistent hypoglycemia continues, medical therapy (glucocorticoids, diazoxide; see Box 22.12) should be initiated. Prolonged hypoglycemia may cause cerebral laminar necrosis. Neurologic signs (e.g., ataxia, bizarre behavior, coma, seizures) may persist in such animals despite normoglycemia. Transient hyperglycemia occasionally occurs and may persist for years after surgery. Insulin therapy may be indicated if blood glucose concentrations above 180 mg/dL persist for longer than 3 to 5 days.

Complications

Complications of surgery in animals with insulinomas include persistent hypoglycemia, pancreatitis, DM, epilepsy, and diffuse polyneuropathy. The most common causes of postoperative hypoglycemia are unrecognized or nonresectable metastases and multiple or incompletely resected primary tumors. Persistent hyperglycemia occurs in up to one-third of dogs undergoing surgical removal of insulinomas and is thought to be a result of suppression of normal β-cells by tumor insulin, resulting in loss of insulin production.

Prognosis

The median survival times for dogs with insulinoma has ranged from 12.3 to 18.2 months, but survival times between studies are difficult to compare because of differences in treatment and data reported (Table 22.4). In a study of 28 dogs with insulinoma, median survival time was 547 days. The median survival of 19 dogs undergoing partial pancreatectomy was 785 days, and for those subsequently receiving prednisone therapy on relapse, median survival was 1316 days. Median survival of dogs with insulinoma treated with medical therapy alone was 196 days.

Prognostic biomarkers have been studied to facilitate optimal patient management. The Ki67 index has proved prognostically significant for both the disease-free interval and overall survival time. In addition to known factors such as tumor size and stage,
Ki67 can act as a biomarker of insulinoma that may be used to predict clinical outcome.

**NOTE** If metastasis is not apparent at surgery, survival of longer than 1 year may occur, even though cures are unlikely.

### GASTRINOMAS

**DEFINITIONS**

Gastrinomas are tumors that secrete excessive gastrin. Zollinger-Ellison syndrome is the term used to describe a syndrome of gastric acid hypersecretion, gastrointestinal ulceration, and non-β-cell pancreatic tumors. Gastrinomas are also called non-β-cell tumors and gastrin-secreting tumors. The terms gastrinoma and Zollinger-Ellison syndrome are often used interchangeably; however, gastrinomas can arise in other parts of the alimentary tract. Zollinger-Ellison syndrome refers specifically to gastrinomas arising in the pancreas.

### GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gastrinomas are rare tumors in dogs and cats. They are derived from ectopic amine precursor uptake decarboxylase cells in the pancreas and produce an excess of the hormone gastrin. Gastrin is normally secreted by cells of the antral and duodenal mucosa in response to antral distention and stimulation by amino acids. Excess gastrin causes hyperacidity, which produces multiple duodenal ulcerations and/or erosions. Pancreatic gastrin-secreting tumors are usually locally invasive into adjacent parenchyma and frequently metastasize to regional lymph nodes or the liver or both.

### DIAGNOSIS

**Clinical Presentation**

**Signalment**

Dogs and cats may be affected. Too few cases have been reported to determine breed or gender predisposition. Affected animals are usually middle-aged to older.

**History**

Most animals have clinical signs of anorexia, vomiting (which is occasionally blood tinged), regurgitation, intermittent diarrhea, weight loss, and/or dehydration. Clinical signs may be present for several days or months before diagnosis. Animals may have been treated for gastric ulcers for months with poor response.

**Physical Examination Findings**

Clinical findings are nonspecific and may include dehydration, diarrhea, melena, hematemesis ("coffee grounds" appearance), steatorrhea, and/or weight loss. Abdominal pain is inconsistent. Gastric ulcer perforation may cause generalized peritonitis (see p. 527).

**Diagnostic Imaging**

Radiographs and ultrasonography are nondiagnostic for gastrinomas because pancreatic masses are generally too small to be visualized. Endoscopy is the most useful technique for diagnosing esophagitis, gastric mucosal hypertrophy, or duodenal ulceration in dogs with suggestive clinical signs. Ulcers are most commonly located in the proximal duodenum.

**Endoscopy**

On endoscopy, patients typically have esophagitis (because of profuse vomiting of acid) and duodenal ulcers or erosions. Duodenal biopsies typically have minimal inflammation. Gastric ulceration is much less common, but erosions and/or mucosal hypertrophy may be seen.

**Laboratory Findings**

Nonspecific laboratory abnormalities noted in animals with gastrinoma include anemia, hypoproteinemia, elevated serum alkaline phosphatase activity, and/or leukocytosis. Electrolyte and acid-base abnormalities (e.g., hypochloremia, hypokalemia, metabolic alkalosis, metabolic acidosis) may occur if vomiting has been severe. Preoperative diagnosis of gastrinoma is based on demonstration of hypergastrinemia. Blood samples for serum gastrin analysis should be obtained after a 12-hour fast and before treatment with any antacid drug. Serum gastrin levels of animals with Zollinger-Ellison syndrome may exceed 1000 pg/mL.

### TABLE 22.4  Reported Survival Times for Dogs With Insulinomas

<table>
<thead>
<tr>
<th>Reference</th>
<th>Number of Cases</th>
<th>Approximate Median Survival Time (Months*)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kruth et al., 1982**</td>
<td>25</td>
<td>12.3</td>
<td>Mean survival time; medically and surgically treated cases; peri-mortality rate associated with owners euthanizing due to presence of inoperable lesions or presence of presumed metastatic lesions</td>
</tr>
<tr>
<td>Mehlhaff et al., 1985**</td>
<td>35</td>
<td>14.2</td>
<td>Mean survival time; medically and surgically treated cases; peri-mortality rate associated with owners euthanizing due to presence of inoperable lesions or presence of presumed metastatic lesions</td>
</tr>
<tr>
<td>Leifer et al., 1986**</td>
<td>18</td>
<td>14.5</td>
<td>Cases surviving surgery, no relapse</td>
</tr>
<tr>
<td>Caywood et al., 1988**</td>
<td>47</td>
<td>18</td>
<td>Stage I; surgically and medically treated cases</td>
</tr>
<tr>
<td>Tobin et al., 1999**</td>
<td>26</td>
<td>12.7</td>
<td>Surgically treated cases only</td>
</tr>
<tr>
<td>Polton, 2006</td>
<td>28/19</td>
<td>18/16.5</td>
<td>Surgically and medically treated cases</td>
</tr>
<tr>
<td>Northrup et al., 2013**</td>
<td>19</td>
<td>10.3</td>
<td>Medical management with biweekly streptozotocin; serious adverse events reported</td>
</tr>
</tbody>
</table>

*Statistically significant.
DIFFERENTIAL DIAGNOSIS

Gastrinomas must be differentiated from other causes of gastrointestinal tract ulceration, including nonsteroidal antiinflammatory drugs, dexamethasone, local neoplasia, infiltrative disease, mast cell tumors, DIC, hepatic failure, circulatory shock, and septic shock (see also p. 427). Other causes of hypergastrinemia include renal failure, gastric outflow obstruction, chronic gastritis, and recent proton pump inhibitor therapy.

MEDICAL MANAGEMENT

Because of the aggressive biological behavior of this malignant neoplasm, the prognosis for long-term cure is poor; however, aggressive medical management includes the use of proton pump inhibitors (Box 22.15). Proton pump inhibitors (e.g., omeprazole) are the most potent known inhibitors of gastric acid secretion. Other agents that may be used to help treat ulcers in dogs with gastrinomas include those that protect the gastric mucosa from damage (see Box 22.15). The effectiveness of these drugs in animals with gastrinoma can be limited, and beneficial effects vary from short to long periods of time.

Sucralfate forms a protective coating over the ulcer or erosion (see Box 22.15). Cimetidine, ranitidine, and famotidine are H₂-receptor blockers that reduce acid secretion; they are much less effective than proton pump inhibitors.

SURGICAL TREATMENT

Exploratory laparotomy is often required to confirm the diagnosis. Surgical resection of the pancreatic mass may provide a cure if metastasis is not present. If metastasis is present, surgical debulking of the mass and removal of operable metastatic lesions may improve the efficacy of medical therapy and prolong survival. The gastrointestinal tract should be closely inspected during surgery for evidence of ulcerations that may perforate. Any such lesions should be removed or should have a serosal patch (see p. 447). Total gastrectomy has been recommended for animals in which the condition is unresponsive to medical therapy; however, because of long-term complications (e.g., malnutrition, dysphagia, bile reflux), this procedure is seldom performed.

Preoperative Management

If possible, the animal’s condition should be stabilized before surgery. Whole blood should be given if the animal is severely anemic (i.e., PCV <20% [see Box 4.1 and Table 4.5]), and anemic animals should be oxygenated before induction of anesthesia.

Electrolyte and acid-base abnormalities should be corrected and fluid therapy initiated before surgery.

Anesthesia

See p. 600 for anesthetic recommendations for animals undergoing pancreatic surgery. Preoperative volume resuscitation may be necessary.

Surgical Anatomy

See p. 603 for discussion of the surgical anatomy of the pancreas and p. 399 for discussion of the surgical anatomy of the stomach.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The caudal thorax and the entire ventral abdomen should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Perform a thorough abdominal exploration. Inspect the draining lymph nodes, liver, duodenum, and mesentery for evidence of metastasis. Inspect the entire pancreas for a mass lesion. Perform a partial pancreatectomy (see p. 605) and resect metastatic lesions that are accessible. Submit excised tissues for histopathologic examination.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

If the animal is severely hypoproteinemic or anemic, wound healing may be delayed. In such cases, polydioxanone or polyglyconate suture is preferred to close gastrotomy and abdominal incisions (see p. 415). These sutures may also be used to perform a serosal patch.

POSTOPERATIVE CARE AND ASSESSMENT

Anemic animals benefit from nasal oxygen given postoperatively. The patient must be monitored, and if signs suggestive of pancreatitis are seen, aggressive therapy is indicated (see p. 605 for treatment of pancreatitis). Small amounts of water should be given the day after surgery, and the patient observed for vomiting. If vomiting does not occur, small amounts of food can be given 24 hours postoperatively. The diet should be low in fat and fiber and should contain moderate amounts of protein and carbohydrates to aid gastric emptying. Fluid therapy should be continued until the animal is eating and drinking. Medical therapy for ulcers should be continued until clinical signs resolve. Long-term medical therapy may be necessary to control gastric hypersecretion caused by hypergastrinemia and to reduce the incidence and severity of ulcers.

PROGNOSIS

Because of the malignant nature of this tumor and its high propensity for metastasis, the long-term prognosis is generally grave.

EXOCRINE PANCREATIC NEOPLASMA

DEFINITION

Exocrine pancreatic carcinomas are malignant tumors that arise from acinar or ductular epithelial cells. They are also called pancreatic adenocarcinomas.
GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Exocrine pancreatic tumors are slightly more common than tumors of the pancreatic islet cells in dogs and cats. Pancreatic tumors are more common in human beings than in dogs and are associated with an extremely high mortality rate (approximately 90% within 1 year of diagnosis). Most pancreatic tumors are malignant (adenocarcinoma); they are aggressive tumors that invade locally and metastasize readily. The most common sites for metastasis are the liver, lungs, peritoneum, and regional lymph nodes. Metastatic pancreatic carcinoma has been diagnosed in a dog with diabetes insipidus. Benign pancreatic tumors (i.e., adenomas) are rare.

NOTE Most pancreatic masses are caused by pancreatitis, not neoplasia. Never euthanize a patient with a pancreatic mass without a histologic diagnosis, no matter how “bad” the mass looks grossly.

DIAGNOSIS

Clinical Presentation

Signalment

Pancreatic adenocarcinomas occur more commonly in older animals; Airedale terriers and boxers have been reported to be at higher risk for this tumor. A gender predisposition has not been proven in dogs, although pancreatic carcinoma seems to be more common in males.

NOTE Clinical signs or gross appearance does not help differentiate pancreatic adenocarcinoma from benign pancreatic disease.

History

Animals with pancreatic adenocarcinoma may have vomiting, abdominal pain, anorexia, weight loss, lethargy, abdominal distention, and/or diarrhea. The history may be acute or chronic. Adenomas are usually incidental findings at surgery or at necropsy and are not associated with clinical signs.

Physical Examination Findings

Physical examination findings for exocrine pancreatic carcinoma may include abdominal pain on palpation and/or ascites occurring secondary to compression of the portal vein or other vessels or as the result of widespread abdominal metastasis. Some animals may have a palpable abdominal mass; others may have icterus secondary to common bile duct obstruction.

Diagnostic Imaging

An ill-defined increase in soft tissue opacity in the right cranial abdominal quadrant may be noted on survey abdominal radiographs. If ascites is present, loss of visceral detail throughout the abdomen may be observed. Abdominal ultrasonography often reveals a mass in the area of the pancreas, but it is not necessarily easy to distinguish from pancreatitis. Distention of the gallbladder and bile ducts may be noted with obstruction of the extrahepatic biliary tract. Obstruction of gastric outflow may be seen on contrast studies of the upper gastrointestinal tract.

Laboratory Findings

Laboratory abnormalities have not been well defined in animals with exocrine pancreatic neoplasia. Abnormalities consistent with extrahaepatic cholestasis (i.e., elevated alkaline phosphatase and hyperbilirubinemia) are often present. Some animals may show mild leukocytosis, dehydration, and hemoconcentration. Extremely high serum lipase values might suggest pancreatic carcinoma, but the test is not generally recommended.

DIFFERENTIAL DIAGNOSIS

Exocrine pancreatic carcinoma must be differentiated from benign and metastatic pancreatic disease. Nodular pancreatic hyperplasia, a condition seen in older animals, is characterized by multiple small, white lesions that protrude minimally from the pancreatic surface. Adenomas are usually small masses that may contain cysts. These conditions are not associated with clinical signs. Pancreatic carcinomas are usually well advanced at the time of diagnosis, and it may be difficult to determine the site of origin for neoplastic masses.

MEDICAL MANAGEMENT

Although numerous treatments have been used in human beings in an attempt to improve the survival of patients with pancreatic adenocarcinoma, only those with resectable lesions at the time of laparotomy have a fair prognosis. Chemotherapeutic agents have not prolonged the life of people or animals with this tumor.

SURGICAL TREATMENT

Surgical resection is the treatment of choice; however, many animals are presented with advanced disease and surgical resection is not possible.

Preoperative Management

The animal’s condition should be stabilized before surgery with administration of IV fluids and correction of acid-base and electrolyte abnormalities.

Anesthesia

See the discussion of anesthetic management of animals with pancreatic disease on p. 600.

Surgical Anatomy

The surgical anatomy of the pancreas is described on p. 603.

Positioning

The animal is prepared for a ventral midline exploratory procedure. The entire abdomen and the caudal thorax should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make an abdominal incision that extends from the xiphoid cartilage as far caudally as necessary to allow complete exploration of the abdominal cavity. After identifying the pancreatic mass (Fig. 22.16), explore the abdominal organs, peritoneum, and regional nodes for evidence of metastasis. Euthanasia should be considered in animals with widespread metastasis. Perform a partial pancreatectomy, if possible. Confirm the patency of the common bile duct before closing the abdomen.
CHAPTER 22  Surgery of the Endocrine System

SUTURE MATERIALS AND SPECIAL INSTRUMENTS
A standard soft tissue pack or a general surgery pack is usually all that is required. See p. 605 for requirements for partial pancreatectomy.

POSTOPERATIVE CARE AND ASSESSMENT
These animals may have pancreatitis secondary to tumor at the time of diagnosis, and this may require therapy (see p. 605). Animals presented for treatment that have pancreatic carcinomas are often debilitated and require special attention to ensure that their nutritional needs are met postoperatively. Enteral or parenteral hyperalimentation should be considered. See also p. 605 for postoperative care of patients with pancreatic disease.

PROGNOSIS
The prognosis is extremely poor for animals with pancreatic carcinomas. Most have widespread disease at the time of diagnosis, and many are euthanized at surgery. Survival of less than 3 months should be expected for most of the remaining animals.

FIG. 22.16 Pancreatic carcinoma in a dog.

Surgery of the Thyroid and Parathyroid Glands

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS
Thyroidectomy is removal of a thyroid gland. Hypothyroidism is deficient secretion of thyroxine. Goitrous hypothyroidism is caused by an abnormal iodine uptake or by defects in iodine uptake, organification, or thyroglobulin formation. Nongoitrous hypothyroidism is spontaneous hypothyroidism that may be immune mediated (i.e., lymphocytic thyroiditis) or may result from idiopathic atrophy. Hyperthyroidism is excessive secretion of thyroxine. Primary hyperparathyroidism is excessive secretion of parathyroid hormone (PTH) by one or more abnormal parathyroid glands.

BOX 22.16  Treatment of Canine Hypothyroidism

<table>
<thead>
<tr>
<th>Maintenance</th>
<th>Levothyroxine (Soloxine) 18–22 μg/kg PO q12h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before Surgery (If Not on Maintenance Therapy, Which Is Preferred)</td>
<td>1. Oral: liothyronine (T₃; Cytobin or Cytomel) 4.4 μg/kg PO q6–8h, or 2. Intravenous: L-thyroxine 4–5 μg/kg (1 dose) (use with caution)</td>
</tr>
</tbody>
</table>

PO, Orally.

PREOPERATIVE MANAGEMENT
Hypothyroidism is a common endocrinopathy in dogs. It is usually due to thyroid dysfunction (primary hypothyroidism), although pituitary and hypothalamic causes are occasionally diagnosed. Three weeks of administration of trimethoprim-sulfamethoxazole (14.1–16 mg/kg orally, twice daily) will depress total thyroxine and free thyroxine and will elevate canine thyroid-stimulating hormone (TSH) concentrations, mimicking hypothyroidism. Thyroid function tests should be interpreted carefully in dogs that are receiving glucocorticoids, phenobarbital, and carprofen. Secretion of thyroid hormones triiodothyronine (T₃) and thyroxine (T₄) from the thyroid is controlled by a feedback mechanism between the hypothalamus, pituitary, and thyroid glands. Thyrotropin (TSH) is produced in the pars distalis of the pituitary gland. It stimulates the synthesis and release of thyroglobulin, a precursor of T₃ and T₄, as well as T₃ and T₄. Release of thyrotropin is controlled by a neuropeptide produced in the hypothalamus called thyrotropin-releasing hormone (TRH). TRH secretion is inhibited by high circulating levels of glucocorticoids (e.g., HAC) or thyroid hormone. Primary hypothyroidism is usually caused by idiopathic follicular atrophy or lymphocytic thyroiditis. Dogs with lymphocytic thyroiditis often have circulating thyroglobulin antibodies that form antigen-antibody complexes in the gland, causing functional glandular tissue to be replaced by fibrous tissue. Feline hypothyroidism is usually caused by thyroidectomy, damage to the blood supply during parathyroidectomy, or destruction by iodine-131 (¹³¹I) therapy. Congenital hypothyroidism may occur in Abyssinian cats. The disease is inherited as an autosomal recessive trait and appears to be the result of a defect of iodide organification. Congenital hypothyroidism has also been reported in dogs.

Hypothyroidism may be manifested as lethargy, exercise intolerance, weight gain, constipation, nonpruritic symmetric alopecia, peripheral neuropathies (e.g., laryngeal paralysis, vestibular deficits), reproductive problems, cardiovascular changes (i.e., bradycardia and weak apex beat), and/or coagulopathies. Hypothyroidism may also result in diminished activity of factor VIII or of factor VIII-related antigen, which may predispose animals with von Willebrand disease (vWD) to spontaneous bleeding or serious hemorrhage during surgery. The mean von Willebrand factor/antigen (vWF) concentration in hypothyroid dogs has been found to be significantly reduced compared with that in euthyroid dogs. It appears that reduced concentrations of plasma vWF can be found in dogs in association with congenital vWD or with vWD acquired through hypothyroidism. Animals with untreated severe hypothyroidism and bleeding tendencies undergoing emergency procedures should be given oral L-triiodothyronine (Box 22.16) three or four times a day, or a
single IV dose of l-thyroxine. Elective procedures should be postponed until replacement therapy has been maintained for a minimum of 2 weeks. If excessive bleeding is noted despite thyroid supplementation, whole blood, plasma, or cryoprecipitate should be given (see Box 4.1 and Table 4.5).

**NOTE** Hypothyroid animals may bleed excessively during surgery. Monitor hemostasis carefully.

Animals with hyperparathyroidism are often brought in because of signs caused by hypercalcemia. PTH is synthesized by chief cells of the parathyroid glands. PTH stimulates renal reabsorption of calcium, mobilizes calcium from bone, and promotes intestinal calcium reabsorption. PTH also controls hydroxylation of 25-hydroxyvitamin D₃ to 1,25-dihydroxyvitamin D₃ in the proximal renal tubules. 1,25-Dihydroxyvitamin D₃ regulates PTH secretion through a negative feedback mechanism. PTH is synthesized and secreted in response to decreases in circulating calcium levels. Functional parathyroid neoplasms (primary hyperparathyroidism; see p. 625) cause hypercalcemia through excessive secretion of PTH; this causes increased renal reabsorption of calcium and increased renal excretion of phosphorus, increased release of calcium and phosphorus from bone, and increased intestinal absorption of calcium and phosphorus.

The preoperative management of animals with functional parathyroid tumors is described on p. 616. Primary hypoparathyroidism is rare in dogs and cats. It primarily affects middle-aged female dogs, occurring secondary to lymphocytic parathyroiditis. Most affected animals have a history of neurologic abnormalities (particularly seizures) or neuromuscular disease.

Cystic thyroid and parathyroid lesions have been reported in older cats but are uncommon. These lesions may be benign (thyroid cysts or cystadenomas) or malignant (parathyroid adenocarcinomas). Surgical resection may be curative, and long-term survival is excellent. Differentials for a cystic ventral cervical mass should include branchial cyst, thyroglossal cyst, thyroid cyst, thyroid cystadenoma, parathyroid cyst, parathyroid cystadenoma, thyroid carcinoma, salivary mucocele, and abscess.

**ANESTHESIA**

Hypothyroidism may prolong recovery from anesthesia. Dosages of premedications and anesthetics should be reduced and titrated to effect in moderately or severely affected animals. Blood pressure and hematocrit should be closely monitored during anesthesia and in the early postoperative period. Blood should be available in case excessive bleeding occurs intraoperatively. Hypothermia may be of greater concern in these patients because of their inability to regulate body temperature normally; care should be taken to maintain body temperature during surgery and to rewarm these patients after surgery. See pp. 621 and 628 for anesthetic recommendations for animals undergoing thyroidectomy.

**NOTE** Anesthetize hypothyroid animals with care; these patients may require reduced dosages of anesthetics.

**ANTIBIOTICS**

Guidelines for appropriate use of perioperative antibiotics should be followed in hypothyroid patients (see Chapter 9). Prophylactic antibiotic therapy should be considered in animals that are debilitated or obese, have pyoderma, or that have concurrent HAC.

**SURGICAL ANATOMY**

The thyroid gland (with two lobes) is a dark red, elongated structure attached to the outer surface of the proximal portion of the trachea (Fig. 22.17). The lobes are usually positioned laterally and slightly ventral to the fifth to eighth cartilage rings. The left lobe is usually located one to three tracheal rings caudal to the right lobe. In adult dogs, they are approximately 5 cm long and 1.5 cm wide; in cats, they are 2 cm long and 0.3 cm wide. Occasionally the right and left lobes are connected by a ventral isthmus. Unlike most glandular organs, they can often be palpated when enlarged. Thyroid secretions (T₄, T₃, and calcitonin) excrete a major effect on metabolism. Thyroid hormone is synthesized by follicular cells, stored intercellularly, and released into the circulation. In adults, it causes an increase in the overall metabolic rate; in juveniles, it stimulates growth. Calcitonin (formed by parafollicular C cells) lowers blood calcium by stimulating calcium uptake. Functional accessory thyroid tissue is common along the trachea, thoracic inlet, mediastinum, and thoracic portion of the descending aorta. Thyroid follicular cells arise from a midline outpouching known as the thyroid diverticulum on the ventral pharyngeal floor. The pharyngeal connections of the diverticulum usually separate completely; however, a persistent connection that has functional glandular epithelium and cysts along its course may remain (thyroglossal duct).

The cranial and caudal thyroid arteries are the principal blood supply of the thyroid. The cranial thyroid artery arises from the common carotid artery; the caudal thyroid artery typically arises from the brachiocephalic artery. The cranial and caudal thyroid arteries anastomose on the dorsal surface of the gland, where they send numerous vessels that supply the gland. The cranial thyroid artery in dogs usually sends a branch that supplies the
Therefore most surgeons recommend implanting an inadvertently excised parathyroid gland into surrounding muscle rather than discarding it. Ectopic parathyroid tissue may hypertrophy after removal of the parathyroid glands, resulting in normal parathyroid function.

**SUTURE MATERIALS AND SPECIAL INSTRUMENTS**

Delayed wound healing may occur in animals with hypothyroidism; care should be used in closing surgical wounds in these patients. See pp. 623, 626 and 628 for a discussion of the instruments for thyroidectomy and parathyroidectomy, respectively.

**POSTOPERATIVE CARE AND ASSESSMENT**

Postoperative care and assessment of animals undergoing thyroidectomy for hyperthyroidism or neoplasia are provided on pp. 627 and 629, respectively.

**SPECIFIC DISEASES**

**FELINE HYPERTHYROIDISM**

**DEFINITIONS**

Hyperthyroidism is a multisystemic disease that results from excessive production and secretion of T₄. Goiter is an enlargement of the thyroid gland. Graves disease describes an autoimmune disorder of human beings in which circulating autoantibodies stimulate thyroid tissue. It is the most common cause of human hyperthyroidism.

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

Hyperthyroidism may occur in dogs or cats; however, it is much more common in cats, in which it is generally associated with adenomatous hyperplasia of one or both thyroid glands. Approximately 90% of affected cats have bilateral thyroid lobe involvement, although the enlargement is usually asymmetric. In 5% to 10% of cats, the thyroid mass is ectopic (i.e., at the thoracic inlet or in the cranial mediastinum). Feline hyperthyroidism secondary to malignant thyroid carcinoma is rare. The cause of feline hyperthyroidism is unknown. Suggested causes have included circulating thyroid-stimulating immunoglobulins, dietary goitrogens, and viral causes. Studies suggest that cats that (1) consume food packaged in cans, (2) have diets containing fish, (3) eat more than 50% wet cat food, or (4) use a litter box have increased risk of hyperthyroidism. It has been suggested that iodine recommendations for commercial cat foods in the past may have inadvertently contributed to the recent epidemic of feline hyperthyroidism, because changes in the recommended iodine concentration of foods may have resulted in a reduction in iodine supplementation since the late 1970s. Canned food may predispose to hyperthyroidism because toxic chemicals such as bisphenol-A are released into the food from the plasticizer linings placed in cans during the canning process. Exposure to fertilizers, herbicides, plant pesticides, flea products, and smoke did not seem to be associated with increased risk of this disease. Advanced age is a risk factor, and purebred cats have a decreased risk of developing hyperthyroidism.

Excessive circulating T₄ causes multisystemic organ dysfunction. Thyrotoxicosis increases the metabolic rate and sensitivity...
to catecholamines and causes significant cardiovascular and metabolic abnormalities. Up to 80% of affected cats may have thyrotoxic heart disease; approximately 20% of these may have congestive heart failure. Hypertension is sometimes identified but does not appear to be as common as cardiac disease. Multifactorial mechanisms may cause neuromuscular and CNS dysfunction in some hyperthyroid cats. Neurologic signs associated with feline hyperthyroidism are listed in Box 22.17. T₄ and T₃ bind to receptor sites in the sarcoplasm that increase skeletal muscle heat production and mitochondrial oxygen consumption. The hyperthyroid state may reduce muscle contraction by uncoupling oxidative phosphorylation. Thyroid hormones may lower the threshold for cerebral tissue activation, alter the activity of some brain enzymes, and may interact with catecholamines to alter the mental state of some affected animals. Abnormalities of the CNS may include hyperexcitability, irritability, aggression, seizures, confusion, and stupor.

DIAGNOSIS

Clinical Presentation

Signalment
Hyperthyroidism generally affects cats older than 8 years of age (mean age, 13 years); however, it may rarely occur in young cats. No gender predisposition has been noted. Siamese and Himalayan cats may be at decreased risk of developing hyperthyroidism. Cats fed primarily canned food and those using cat litter may be at increased risk. Cats that prefer to eat canned cat food of fish or liver and giblet flavor may have an increased risk of developing this condition.

History
Most affected cats are presented for treatment because of weight loss despite a normal or voracious appetite, restlessness, and/or hyperactivity. Occasionally, a small mass is noted in the ventral cervical region. Vomiting, diarrhea, polyuria, polydipsia, aggression, and/or a rough hair coat may occur. Frequency of defecation is sometimes increased. Body temperature may be slightly elevated. Approximately 10% of hyperthyroid cats are depressed, lethargic, inappetent, and/or weak (i.e., “apathetic” hyperthyroidism).

Physical Examination Findings
A palpable cervical mass is present in most affected cats. The weight of the enlarged gland often causes it to gravitate ventrally because the thyroid is loosely attached to tracheal fascia. Occasionally the gland may descend into the thoracic inlet, where it can no longer be palpated. Additional physical examination findings may include emaciation, a thin and/or roughened hair coat, and cardiac abnormalities (e.g., tachycardia, gallop rhythms, murmurs, left anterior fascicular block, and/or atrial and ventricular tachyarrhythmias). Electrocardiographic abnormalities may include tachycardia, prolonged QRS duration, increased R-wave amplitudes in lead II, and ventricular preexcitation.

Diagnostic Imaging
An enlarged heart consistent with hypertrophic cardiomyopathy is often found on thoracic radiography and echocardiography. If the cat is in congestive heart failure, pleural effusion or pulmonary edema (or both) may occur. Ectopic thyroid tissue is rarely visible radiographically. Thyroid scintigraphy is the optimal diagnostic test because it can both definitively identify hyperthyroidism and locate functional ectopic thyroid tissue. With this procedure, technetium-99m is administered intravenously or intramuscularly. The radionuclide is trapped in functional thyroid tissue but is not organified. Delayed phase imaging of the body using a gamma camera provides functional and rudimentary anatomic locations of the hyperfunctioning thyroid tissue (Figs. 22.19 and 22.20). A ratio of uptake in thyroid tissue compared with that in salivary glands of greater than 2 is diagnostic of hyperthyroidism.

Laboratory Findings
Most affected cats have high serum total T₄ (TT₄) and free T₄ (T₄) by equilibrium dialysis concentrations. However, the

### BOX 22.17 Neurologic Abnormalities in Hypokalemic, Hyperthyroid Cats

- Generalized weakness
- Neck ventroflexion
- Fatigue
- Muscle tremors
- Ataxia
- Incoordination
- Inability to jump
- Muscle atrophy
- Breathlessness (due to weakness of intercostal muscles)
- Collapse

**NOTE** Approximately 20% of hyperthyroid cats have multiple areas of hyperfunctional thyroid tissue and/or intrathoracic hyperfunctional thyroid tissue where surgical thyroidectomy would not be curative. Warn owners of the possibility of ectopic thyroid tissue when performing thyroidectomy.

**NOTE** Before anesthetizing animals for a thyroidectomy, perform echocardiography and obtain thoracic radiographs to identify thyrotoxic heart disease.
diagnosis of hyperthyroidism cannot be excluded on the basis of a normal TT4 concentration, and cats without hyperthyroidism may have increased fT4 concentrations. In general, fT4 is measured only if a cat that is suspicious for hyperthyroidism has a normal TT4. Hyperthyroidism increases glomerular filtration rate (GFR), which may lessen azotemia and thereby mask clinical signs of chronic kidney disease (CKD). This is important because successful therapy of hyperthyroidism may result in clinical manifestation of CKD. At the same time, CKD may lower serum TT4 concentrations (i.e., euthyroid sick syndrome) into the reference range, making diagnosis more difficult. The combined measurement of fT4 and TT4 may be necessary when trying to determine if cats with moderate to severe CKD have hyperthyroidism.

Other abnormalities may include mild elevations in red blood cell numbers, increased PCV, neutrophilic leukocytosis, eosinopenia, lymphopenia, and elevated alanine aminotransferase and alkaline phosphatase. Serum creatinine and blood ionized calcium concentrations are often decreased, and serum phosphorous concentrations are often increased.

If baseline serum thyroid hormone concentrations are normal in a cat with appropriate clinical signs, or if a ventral cervical mass is palpable, serum TT4 and fT4 concentrations should be remeasured in 3 to 4 weeks, or nuclear scintigraphy should be performed. As an alternative, a T3 suppression test (Box 22.18) or a TRH stimulation test may be performed. In normal cats, the serum T4 concentration should decline by more than 50% after administration of sodium liothyronine (i.e., <1.5 mg/dL), whereas in hyperthyroid cats, a minimal decrease in the serum T4 concentration is seen. The T3 concentration should increase in both hyperthyroid and euthyroid cats if the medication was given appropriately. For the TRH test, serum T3 and T4 concentrations are measured before and 4 hours after IV administration of TRH (0.1 mg/kg). Hyperthyroid cats usually have a relative increase in T4 of less than 50%; normal cats have a relative increase of greater than 50%. As an alternative, the response to oral antithyroid drugs may help confirm the diagnosis.

**NOTE** If total T4 fails to diagnose hyperthyroidism, measurement of free T4 should be requested.

Cats with hyperthyroidism usually have an increased GFR compared with normal cats; resolution of hyperthyroidism may reduce the GFR, resulting in signs of renal failure. Significant changes in kidney function typically occur within 4 weeks post-treatment, but are normal afterwards. Measurement of GFR and/or urine specific gravity and serum TT4 may help identify those animals that will develop evidence of renal azotemia after treatment. If in doubt as to the safety of thyroidectomy or 131I therapy, one may first administer a course of methimazole and monitor serum creatinine and blood urea nitrogen concentrations to see how the patient will tolerate euthyroidism. For cats that develop overt renal failure after establishment of euthyroidism, withdrawal of methimazole should result in improved renal function. Hypokalemia and concurrent muscle weakness may occur in cats with hyperthyroidism.

**DIFFERENTIAL DIAGNOSIS**

Cats presented for treatment with weight loss or vomiting caused by hyperthyroidism must be differentiated from those with intestinal lymphoma or inflammatory bowel disease. Those with neurologic signs must be differentiated from cats with primary CNS abnormalities. Cardiac dysfunction that occurs secondary to hyperthyroidism should be differentiated from that resulting from other acquired or congenital causes.

**MEDICAL MANAGEMENT**

Treatment of feline hyperthyroidism may include long-term administration of antithyroid drugs (see Preoperative Management later) or 131I, or surgical removal of the affected glands. The choice of treatment for the individual cat depends on the age and condition of the animal (i.e., presence of cardiovascular or renal disease) and the therapies available to the practitioner.

Propylthiouracil is an effective oral antithyroid drug in cats; however, it is not recommended because of severe side effects (i.e., autoimmune hemolytic anemia and immune-mediated thrombocytopenia). Long-term administration of methimazole or carbimazole (not available in the United States) can cause remission; however, clinical signs return once the drug is discontinued. Methimazole inhibits several steps in thyroid hormone synthesis and is effective in restoring a euthyroid status to most cats; however, up to 20% of cats experience gastrointestinal upset.
Important to detect other diseases before treating with 131I, so cat must be confined for days to weeks (depending on specific however, facilities are required to safely handle the isotope. The once daily after 3 and 53 weeks, respectively. 22 pro-drug to methimazole) has been evaluated. Treatment was administration does not appear to be a significant factor when response administration does not appear to be as high as that of the oral route. Timing of blood sampling after oral methimazole administration does not appear to be a significant factor when response to methimazole treatment is evaluated. If thyroid carcinoma is suspected, medical therapy with antithyroid drugs may palliate clinical signs while allowing tumor growth. The safety and efficacy of a novel controlled-release formulation of carbimazole (a pro-drug to methimazole) has been evaluated. Treatment was started at 15 mg once daily; response was assessed after 10 days and 3, 5, 8, 26, and 53 weeks thereafter; and the dosage was adjusted. The median dose used in these cats was 10 and 15 mg once daily after 3 and 53 weeks, respectively. 22 131I is a safe and effective method of treating hyperthyroidism; however, facilities are required to safely handle the isotope. The cat must be confined for days to weeks (depending on specific state laws), during which time it is a human health hazard. It is important to detect other diseases before treating with 131I, so that minimal contact with the cat is required during treatment. Radioactive iodine is trapped in the thyroid gland and causes tissue destruction. However, normal thyroid tissue is spared because it is suppressed and thus does not uptake the radioactive iodine. In the past, recent administration of antithyroid medications has been proposed to decrease the efficacy of radioactive iodine treatment because of decreased uptake. In normal cats methimazole may cause an increase in uptake as the result of upregulation of TSH. A definitive conclusion will require evaluation in hyperthyroid cats. If carcinoma is present, larger doses of 131I may be necessary, requiring longer isolation periods.

NOTE Percutaneous ethanol ablation of bilateral thyroid nodules is not recommended as a treatment for hyperthyroidism in cats.

during treatment (Box 22.19). In rare cases, drug-induced hepatopathy, thrombocytopenia, and agranulocytosis occur with long-term therapy. Although in general the drug is well tolerated and many side effects resolve with continued therapy, administration of an oral compound may be difficult in fractured cats and in those with impaired or elderly owners. A pluronic organogel formulation for transdermal application has recently been reported. The incidence of adverse gastrointestinal signs is less with the transdermal application; however, the efficacy of this route of administration does not appear to be as high as that of the oral route. Timing of blood sampling after oral methimazole administration does not appear to be a significant factor when response to methimazole treatment is evaluated. If thyroid carcinoma is suspected, medical therapy with antithyroid drugs may palliate clinical signs while allowing tumor growth. The safety and efficacy of a novel controlled-release formulation of carbimazole (a pro-drug to methimazole) has been evaluated. Treatment was started at 15 mg once daily; response was assessed after 10 days and 3, 5, 8, 26, and 53 weeks thereafter; and the dosage was adjusted. The median dose used in these cats was 10 and 15 mg once daily after 3 and 53 weeks, respectively. 22 131I is a safe and effective method of treating hyperthyroidism; however, facilities are required to safely handle the isotope. The cat must be confined for days to weeks (depending on specific state laws), during which time it is a human health hazard. It is important to detect other diseases before treating with 131I, so that minimal contact with the cat is required during treatment. Radioactive iodine is trapped in the thyroid gland and causes tissue destruction. However, normal thyroid tissue is spared because it is suppressed and thus does not uptake the radioactive iodine. In the past, recent administration of antithyroid medications has been proposed to decrease the efficacy of radioactive iodine treatment because of decreased uptake. In normal cats methimazole may cause an increase in uptake as the result of upregulation of TSH. A definitive conclusion will require evaluation in hyperthyroid cats. If carcinoma is present, larger doses of 131I may be necessary, requiring longer isolation periods.

NOTE Percutaneous ethanol ablation of bilateral thyroid nodules is not recommended as a treatment for hyperthyroidism in cats.

**BOX 22.19 Possible Side Effects of Methimazole Therapy**

- Anorexia
- Vomiting
- Pruritus
- Lethargy

**Polyarthritis**

- Development of serum antinuclear antibodies
- Hepatopathy
- Thrombocytopenia with or without bleeding

**Anemia**

- Agranulocytosis
- Leukopenia
- Positive Coombs’ test result

*Fewer adverse effects are seen when using transdermal gel as opposed to oral administration. Also see text.

**BOX 22.20 Preoperative Drug Therapy for Cats With Hyperthyroidism**

**Methimazole (Tapazole)**

1.25–2.5 mg/cat PO q12h for 7–14 days. If needed, increase dose by 2.5 mg/d until control is achieved up to 5–10 mg/cat PO q12h. 2 Transdermal application (to the hairless skin of the pinna) is also available.

**Carbimazole**

5 mg/cat PO q8–12h, then adjust to 5 mg/cat q12h as needed

**Carbimazole (Controlled-Release Tablet Formulation)**

15 mg/cat PO q24h, then adjust as necessary (10–25 mg/cat)

**Propranolol (Inderal)**

2.5–5 mg/cat (0.4–1.2 mg/kg) PO q8–12h

*Use lower dose in small or debilitated animals. If long-term administration is considered, this dose should be adjusted to maintain the T4 concentration within the normal range.

*The overall efficacy of transdermal methimazole may not be as high as the orally administered drug; however, it is associated with fewer gastrointestinal adverse effects.

*Vidatta, Merck Animal Health, Intervet International, The Netherlands. Carbimazole is not available in the United States but is available through compounding pharmacies (e.g., Diamondback Drugs; www.diamondbackdrugs.com, 1-866-678-4428).

*Propranolol should be used with care in hyperthyroid cats. Administration of propranolol to hypokalemic cats may cause sudden death.

PO, Orally; T4, thyroxine.

**SURGICAL TREATMENT**

Surgical treatment of hyperthyroidism involves thyroidectomy. Complications of thyroid surgery include intraoperative hemorrhage and clinical signs associated with damage to the recurrent laryngeal nerves, the parathyroid blood supply, or parathyroidectomy.

The major complication of bilateral thyroidectomy is hypoparathyroidism, which occurs secondary to removal or damage of the parathyroid glands. The procedure must be performed carefully to prevent this complication. If the parathyroid gland is inadvertently removed, it should be transferred to a nearby muscle belly (e.g., sternohyoideus muscle) so that the gland may revascularize and become functional again (parathyroid gland autotransplantation). To prevent the complication of hypocalcemia, some surgeons recommend a two-stage procedure in which one thyroid lobe is removed and its associated parathyroid is reimplanted into adjacent musculature during the first surgery. Two to three weeks later, the other thyroid lobe is removed and its associated parathyroid is similarly reimplanted. Although this may reduce the risk of postoperative hypocalcemia, the added risk of a second anesthetic event is introduced.

**Preoperative Management**

Metabolic and cardiovascular abnormalities associated with hyperthyroidism make anesthesia risky; therefore cats should be made euthyroid preoperatively by administering methimazole (Tapazole) (Box 22.20). Generally, administration for 1 to 3 weeks before surgery is sufficient; however, measurement of the TT4 concentration should be repeated to ensure that it is within the normal range before surgery is performed (see comment on side effects earlier). If preoperative therapy with methimazole is not tolerated, propranolol, a β1- and β2-blocker, may be given for 1 to 2 weeks before surgery (see Box 22.20) to reduce the HR. Propranolol may be discontinued 24 to 48 hours before surgery.
because of its β-blocking effects, which may interfere with treatment of hypotension; however, this increases the risk of tachycardia and hypertension, especially at induction. Another preanesthetic choice would be to use metoprolol, a specific β1-blocker, and continue antihypertensive therapy until the morning of surgery. Although atenolol (β-blocker) effectively reduces HR in most cats with hyperthyroidism, elevated systemic blood pressure is poorly controlled, and the addition of another vasodilator such as amlodipine or an angiotensin-converting enzyme inhibitor is often necessary to treat associated hypertension. Hypotension secondary to vasodilation induced by the anesthetic gases can be treated with small boluses of phenylephrine.

Because cardiac abnormalities are common, an electrocardiogram, blood pressure readings, and chest radiographs should be obtained before surgery. An echogram needs to be obtained if a cardiac murmur has been auscultated. Many hyperthyroid cats have concurrent renal disease, hypokalemia, and/or azotemia. If clinical signs of hyperthyroidism have not been resolved with the preoperative methimazole, hypovolemia may persist secondary to increased catecholamine production. These cats should be given fluids before, during, and after surgery, and care should be taken to ensure that uremia does not occur during surgery (see the discussion of anesthetic management of animals with renal disease on p. 650) or after surgery, when cardiac output drops because the cat becomes euthyroid. Fluid therapy should be adjusted if the cat is in congestive heart failure.

Anesthesia
Cats with cardiomyopathy may be premedicated with butorphanol or buprenorphine (Table 22.5). As soon as an IV catheter is placed, diazepam or midazolam can be given to minimize the patient’s stress. Because mask induction may stress the animal and cause increased catecholamine release, its use is generally not recommended; the animal can be reoxygenated by holding the end of the circuit near the animal’s face. These patients have a high oxygen requirement, and adequate oxygenation should be maintained throughout the perioperative period. Anesthesia can be induced with propofol or etomidate. Ketamine and thiopental can cause tachycardia and should be avoided during induction. A couple of drops of lidocaine given via a tuberculin syringe may aid intubation and reduce the elevations in HR and blood pressure associated with intubation. Maintenance on isoflurane or sevoflurane with oxygen should be used; halothane should be avoided. Tachyarrhythmias usually can be controlled with esmolol intravenously. If elevated HR and/or blood pressure continues, low doses of metoprolol can be titrated to effect. If the arrhythmias are ventricular in nature, lidocaine can be given as an IV bolus (see Box 22.8).

It bears repeating that these patients often have high catecholamine production with underlying hypovolemia and renal insufficiency, masked anemia, abnormal cardiac output, tachyarrhythmias, and increased metabolic rates, drug metabolism, and oxygen consumption. Therefore they need to be sedated with benzodiazepines and must be adequately hydrated before surgery. Elevated blood pressure and HR need to be controlled with β-blockers. If hypotension arises during surgery and blood losses are minimal, small boluses of phenylephrine are usually sufficient to maintain mean arterial pressures between 60 and 80 mm Hg. Intraoperative fluid rates may need to be 5 mL/kg per hour in patients that have decreased cardiac function, and 10 mL/kg per hour in patients with normal cardiac function. Thyroid storm is a condition caused by excessive thyroid hormone production due to excessive catecholamine release. Clinical signs of thyroid storm may include marked elevations in HR, blood pressure, and temperature, as well as cardiac arrhythmia and shock.

Surgical Anatomy
See p. 616 for a description of the surgical anatomy of the thyroid gland.

Positioning
The animal is placed in dorsal recumbency with the neck slightly hyperextended and the forelimbs pulled caudally. The entire ventral neck and the cranioventral thorax should be prepared for aseptic surgery.

SURGICAL TECHNIQUE
Intracapsular Thyroidectomy
Make a skin incision from the larynx to a point cranial to the manubrium. Bluntly separate the sternohyoid and sternothyroid muscles. Use a self-retaining (e.g., Gelpi) retractor to maintain exposure. Identify the enlarged thyroid gland and the external parathyroid gland (Fig. 22.21). Make an incision on the caudoventral surface of the gland in an avascular area (Fig. 22.22), and extend it cranially with small scissors (e.g., iris scissors). Using a combination of blunt and sharp dissection, carefully remove the thyroid tissue from the capsule. Perform the dissection carefully to prevent damage to the parathyroid gland or its blood supply. Use bipolar cautery to achieve hemostasis, but avoid damaging the gland’s blood supply. After the thyroid parenchyma has been removed, excise most of the thyroid capsule; however, do not excise the capsule that is intimately associated with the external parathyroid gland. If the parathyroid gland is inadvertently excised, or if its blood supply is damaged, transplant the gland to a nearby muscle belly (see p. 613). Close subcutaneous tissue in a simple continuous suture pattern (e.g., 3-0 or 4-0 absorbable). Close the skin in a simple continuous or simple interrupted suture pattern (e.g., 3-0 nonabsorbable).

NOTE Sterile cotton-tipped swabs are useful to help separate the gland from the capsule.
## TABLE 22.5 Anesthetic Considerations in the Feline Hyperthyroid Patient

### Preoperative Considerations

**Associated conditions**
- Anemia (may be occult)
- Hypovolemia
- Hypertension
- Tachydysrhythmias
- Ventricular ectopy
- Cardiac dysfunction
- Renal insufficiency (may be occult)
- Cardiomyopathy
- Renal insufficiency (may be occult)
- Cardiomyopathy

**Bloodwork**
- HCT
- Electrolytes
- BUN
- Cr
- TP
- Urinalysis

**Physical examination**
May be hypovolemic, tachycardic, and hypertensive if untreated with methimazole

**Other diagnostics**
- Blood pressure is essential
- ECG
- Radiographs (thoracic)
- Echocardiography

**Premedication**
Give:
- Diazepam (0.2 mg/kg IV), or
- Midazolam (0.2 mg/kg IV, IM), or
- Buprenorphine *a* (0.005–0.02 mg/kg IV, IM) or
- Butorphanol (0.2–0.4 mg/kg IV, IM) or
- Morphine *b* (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM)
- Avoid ketamine, xylazine, medetomidine, dexmedetomidine, atropine, glycopyrrolate, and acepromazine

### Intraoperative Considerations

**Induction**
- Titrate propofol (2–6 mg/kg IV), or
- Give alfaxalone (2–3 mg/kg IV)
- If CHF, titrate etomidate (0.5–1.5 mg/kg IV)
- Isoflurane or sevoflurane
  - Fentanyl (1–4 μg/kg IV PRN) for short-term pain relief, plus
  - Buprenorphine *a* (0.005–0.02 mg/kg IV PRN), or
  - Hydromorphone (0.05–0.1 mg/kg IV PRN), or
  - Morphine *b* (0.05–0.2 mg/kg IV PRN) if minimal hypotension
  - For hypertension (to keep MAP 70–90 mm Hg)
  - Esmolol (0.05–0.25 mg/kg IV) boluses every 2–5 min to effect and/or CRI (50–200 μg/kg/min IV) to maintain normal heart rate, plus
  - Nitroprusside (0.5–5 μg/kg/min IV) or
  - Nitroglycerin (1–5 μg/kg/min IV)
  - For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine or dopamine as needed

**Fluid needs**
- 5–10 mL/kg/h if minimal blood loss

**Monitoring**
- BP: essential
- ECG
- Respiratory rate
- SpO₂
- Temperature
- +/– Arterial line

### Postoperative Considerations

**Analgesia**
- Buprenorphine *a* (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h)

**Monitoring**
- SpO₂
- Blood pressure
- ECG
- HR
- Respiratory rate
- Temperature
- U/O

**Bloodwork**
- Serial calcium for 48–72 h
- Serial BUN/Cr for 2–4 wk

**Estimated pain score**
Mild; however, these patients need to have minimal stress and to be kept pain free for approximately 24 h to assist in avoiding thyroid storm (see text)

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*a* Buprenorphine is a better analgesic than morphine in cats.

*b* Give slowly to prevent histamine release.

Monitor for hyperthermia.

BP, Blood pressure; BUN, blood urea nitrogen; CHF, congestive heart failure; Cr, creatinine; CRI, constant-rate infusion; ECG, electrocardiogram; EtCO₂, end-tidal CO₂; HCT, hematocrit; HR, heart rate; IM, intramuscular; IV, intravenous; MAP, mean arterial pressure; OTM, oral transmucosal; PRN, as needed; SpO₂, hemoglobin saturation with oxygen; TP, total protein; U/O, urine output.
Modified Extracapsular Approach for Thyroidectomy

Position the animal as previously described. Locate the thyroid gland as described before, and ligate or cauterize the caudal thyroid vein. Using fine-tipped, bipolar cautery forceps (Fig. 22.23A), cauterize the thyroid capsule approximately 2 mm from the external parathyroid gland. With small, fine scissors, cut the gland at the cauterized area, and use sharp and blunt dissection to remove the gland from the parathyroid gland (see Fig. 22.23B). Carefully dissect all thyroid gland from the surrounding tissue and parathyroid gland (see Fig. 22.23C). Do not damage the cranial thyroid artery or its branches to the external parathyroid gland. If the parathyroid gland is inadvertently excised, or if its blood supply is damaged, transplant the gland to a nearby muscle belly (see p. 613). Close as previously described.

**NOTE**  To prevent hypocalcemia, take special care to avoid damaging the cranial thyroid artery.

Modified Intracapsular Approach for Thyroidectomy

Position the animal as described previously. Locate the thyroid gland and remove the gland as described previously in the intracapsular thyroidectomy section. Using a No. 15 scalpel blade or fine scissors, create a peninsula of capsular tissue containing only the parathyroid gland and its blood vessel. Excise the remainder of the capsule. If the parathyroid gland is inadvertently excised, or if its blood supply is damaged, transplant the gland to a nearby muscle belly (see p. 613). Close as previously described.

**SUTURE MATERIALS AND SPECIAL INSTRUMENTS**

Small, fine instruments, such as iris scissors and Bishop-Harmon thumb forceps, facilitate removal of the thyroid glands. Bipolar cautery forceps are advantageous for providing hemostasis because they allow finer control of coagulation than do unipolar forceps.
Sterile Q-tips are useful for dissecting the thyroid glands from the parathyroid glands.

**POSTOPERATIVE CARE AND ASSESSMENT**

Complications may include hypocalcemia, hypothyroidism, recurrence of hyperthyroidism, worsening of renal disease, Horner syndrome, and/or laryngeal paralysis. Hypocalcemia (serum calcium level <9 mg/dL in adult dogs and <8.5 mg/dL in adult cats) is the most important, acute, life-threatening complication of thyroidectomy. Hypocalcemia may occur owing to removal of or trauma to the parathyroid glands or their blood supply. Transient hypocalcemia may be caused by local edema of the parathyroid gland associated with trauma (including use of electrocautery) to the gland’s vascular supply. Hypocalcemia appears to occur in 6% of cats undergoing bilateral thyroidectomy; surgeon experience may be an important factor in determining whether hypocalcemia occurs. Most animals do not develop clinical signs until the serum calcium level is below 7.5 mg/dL, depending on acid-base status. Animals should be closely observed for 2 to 4 days for signs of hypocalcemia (e.g., panting, nervousness, facial rubbing, muscle twitching, ataxia, seizures). In cats, early signs may include lethargy, anorexia, panting, and facial rubbing. Clinical signs are usually noted within 24 to 96 hours, although delayed signs have been reported up to 5 or 6 days later. Acute signs of hypocalcemia may be treated with IV 10% calcium gluconate; calcium chloride should never be administered. The 10% calcium gluconate should be given slowly intravenously (Box 22.21), and the cardiac rate and rhythm should be monitored during administration. Calcium administration should be discontinued if bradycardia develops. Calcium gluconate can also be added to the fluids, or the IV dose can be diluted in an equal volume of saline (or for greater safety, dilute 1:3 or 1:4) and given subcutaneously every 6 to 8 hours (see Box 22.21) until the animal is eating and can be given oral medications. Administration of undiluted calcium gluconate may be associated with abscess formation (Fig. 22.24). Subcutaneous or IV administration of calcium should be discontinued when the serum calcium level is above 8 mg/dL.

Maintenance therapy consists of oral calcium and vitamin D administration (see Box 22.21). The form of vitamin D most commonly used is dihydrotachysterol. It does not accumulate in fat and has a more rapid onset of action than vitamin D₃. Serum calcium levels should be monitored weekly and the dosage of calcium changed accordingly. Vitamin D supplementation can often be discontinued once the parathyroid gland revascularizes. Some animals must be maintained on vitamin D for months before the dose can be reduced; others require lifelong therapy.

Recurrence of hyperthyroidism is likely associated with the presence of ectopic hyperplastic thyroid tissue (EHTT). In a study of 2096 cats undergoing thyroid scintigraphy, ectopic thyroid tissue was found in 81 (3.9%). This reinforces the importance of performing preoperative thyroid scintigraphy in affected cats.

**NOTE** Do not administer calcium chloride (especially intravenously); you can overdose the animal too easily.

**PROGNOSIS**

Hypocalcemia due to iatrogenic hypoparathyroidism may be permanent or temporary. Persistent hypocalcemia may occur if all four parathyroid glands are removed, or if their blood supply is irreversibly damaged. Temporary or transient hypocalcemia is usually caused by disruption of the parathyroid blood supply, which may be associated with edema and swelling of the gland or its blood supply. Hypocalcemia should not occur after unilateral thyroidectomy. Recurrent hyperthyroidism may result from hypertrophy of adenomatous tissue not removed during thyroidecmy or from adenomatous changes in EHTT (see earlier). Hyperthyroidism may occur within months or years in a small portion of cats undergoing bilateral thyroidectomy. If possible, a thyroid scan should be performed to localize hyperfunctioning tissue in these animals before surgery is repeated.

**NOTE** Hypocalcemia is extremely rare after unilateral thyroidectomy for feline hyperthyroidism.

![FIG. 22.24 A dog that received undiluted calcium gluconate subcutaneously and developed skin necrosis and tissue sloughing.](image-url)
In a study of 101 hyperthyroid cats undergoing thyroidectomy, two cats died within 72 hours of surgery, one from laryngospasm and the other from unknown causes. In cats treated with 131I, methimazole, or both, cats with preexisting renal disease were shown to have significantly shorter survival times than cats without preexisting renal disease. When cats with preexisting renal disease were excluded, median survival time for cats treated with methimazole alone was 2 years, and median survival time for cats treated with 131I alone was 4 years. Treatment with both resulted in a median survival of 5.3 years. Age, preexisting renal disease, and treatment type affected survival times.

In another study, it was found that iatrogenic hypothyroidism appeared to contribute to the development of azotemia after treatment of hyperthyroidism. In the aforementioned study, cats with azotemia had reduced survival time compared with cats with normal renal function. The authors suggested that restoration of hypothyroid cats to euthyroidism may be indicated in cats with normal renal function. The authors suggested that cats with azotemia had reduced survival time compared with cats with normal renal function. The authors suggested that restoration of hypothyroid cats to euthyroidism may be indicated to normalize renal function and improve survival.

### Hyperparathyroidism

**Definition**

Primary hyperparathyroidism is a disorder resulting from excessive secretion of PTH by the parathyroid gland or glands.

**General Considerations and Clinically Relevant Pathophysiology**

Primary hyperparathyroidism is uncommon in dogs and rare in cats. It is usually caused by parathyroid adenomas, although parathyroid carcinomas and parathyroid hyperplasia have also been reported. Parathyroid adenomas are typically small, well-encapsulated tumors that appear brown or red and are located near the thyroid glands; however, ectopic adenomas may be located near the thoracic inlet or in the cranial mediastinum. Clinical signs are caused by PTH increasing calcium absorption and phosphorus excretion in the kidneys and enhancing bone resorption. The net result is an increase in serum calcium levels and a decrease in serum phosphorus levels. Clinical abnormalities caused by hypercalcemia may include dystrophic calcification, impaired renal tubular concentrating ability, renal failure, and calcium oxalate nephrolithiasis and urolithiasis.

**Diagnosis**

**Clinical Presentation**

**Signalment**

Parathyroid tumors usually occur in older dogs. No gender predisposition has been noted. Keeshonden (and possibly German shepherds and Norwegian elkhounds) may be predisposed to the disorder. Primary gland hyperplasia has been reported in young dogs.

**History**

Dogs may be asymptomatic or may be presented because of polyuria-polydipsia or nonspecific signs (e.g., vomiting, weakness, constipation, lethargy, inappetence). Clinical signs may be insidious at onset. The most common clinical signs in cats with primary hyperparathyroidism are anorexia, lethargy, vomiting, weakness, and weight loss; polyuria and polydipsia are less common in cats than in dogs. Occasionally, bone and joint pain and pathologic fractures may occur secondary to skeletal demineralization. Cystic calculi may develop secondary to hypercalcemia.

NOTE Consider primary hyperparathyroidism as a differential diagnosis in animals with hypercalcemia, dystrophic calcification, calcium oxalate urolithiasis, and/or nephrolithiasis.

**Physical Examination Findings**

The physical examination findings usually are nonspecific. The enlarged parathyroid gland can seldom be palpated in dogs; however, a cervical mass may be palpated in some cats.

**Diagnostic Imaging**

Cervical radiographs rarely identify the neoplasm. Notable demineralization of the skeleton, nephrolithiasis, and/or nephrocalcinosis may be seen radiographically. Ultrasonography is an excellent modality to evaluate the parathyroid glands. Ultrasonographic detection of a parathyroid gland exceeding 4 mm in diameter is highly suspicious for parathyroid adenoma or carcinoma. Parathyroid scintigraphy does not appear to be a sensitive or specific indicator for definitive identification of abnormal parathyroid glands in dogs with hypercalcemia.

**Laboratory Findings**

Serum biochemical abnormalities in dogs with primary hyperparathyroidism include hypercalcemia and hypophosphatemia. Hypercalcemia is the most consistent finding in affected cats. Measurement of PTH in animals with normal renal function is a sensitive test. High-normal or increased serum concentrations of PTH in hypercalcemic animals with normal renal function are strongly suggestive of hyperparathyroidism. Other causes of hypercalcemia (see later discussion) are usually associated with low or low-normal levels of PTH. Renal dysfunction, which may occur secondary to hypercalcemia or may be a primary disorder, may also elevate serum concentrations of PTH. If renal function is abnormal, serum PTH concentrations should be evaluated in conjunction with the serum ionized calcium concentration. Serum ionized calcium levels are increased with hyperparathyroidism, but are usually low to low-normal with renal failure (Table 22.6). Definitive diagnosis of primary hyperparathyroidism requires surgical exploration of the parathyroid glands. Dogs can have uniglandular or multiglandular disease. Systemic and local PTH concentrations decrease by more than 50% in all dogs after excision of the parathyroid gland(s), and serum calcium concentration eventually returns to normal, although rebound hypocalcemia is common.

Careful physical examination (including rectal examination), thoracic and abdominal radiography, abdominal ultrasonography,

<table>
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<tr>
<th>TABLE 22.6 Serum Parathyroid and Calcium Levels With Primary Hyperparathyroidism and Renal Disease</th>
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<tbody>
<tr>
<td><strong>PTH</strong></td>
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<tr>
<td>HPTH</td>
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<td>Renal failure</td>
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*Serum ionized calcium.

Ca²⁺, Calcium; HPTH, hyperparathyroidism; PTH, parathyroid.
skeletal radiography, routine bloodwork, and/or lymph node aspirations should be performed in hypercalcemic animals to identify neoplastic causes of hypercalcemia of malignancy (e.g., lymphosarcoma, apocrine gland adenocarcinoma) before a diagnosis of hyperparathyroidism is pursued. Other causes of hypercalcemia include granulomatous disease, chronic renal failure, hypoadrenocorticism, and hypervitaminosis D. Thyroglos- sal cysts (formed when the embryonic thyroglossal duct fills with fluid) may be confused with parathyroid masses on palpation.

**NOTE** Paraneoplastic hypercalcemia of malignancy is a more common cause of hypercalcemia than primary hyperparathyroidism. Hypercalcemia of malignancy can rapidly cause renal failure if diagnosis and therapy are delayed, but hypercalcemia caused by hyperpara-throidism is often not as high as that seen in primary hyperpara-throidism and is less likely to cause renal failure.

### MEDICAL MANAGEMENT

Hypercalcemia may be treated by diuresis (see later discussion on Preoperative Management). Surgical removal of the neoplastic parathyroid tissue is the definitive treatment for primary hyperparathyroidism. Glucocorticoid therapy is usually transiently effective in lowering the serum calcium concentration in animals with lymphosarcoma and may also lower the calcium concentration in animals with other disorders. In confusing cases, administration of L-asparaginase may be used as a therapeutic trial to rule out occult lymphosarcoma as a cause of hypercalcemia.

### SURGICAL TREATMENT

Parathyroidectomy is the treatment of choice for hyperparathyroidism caused by parathyroid neoplasia and primary hyperplasia. If the parathyroid glands are uniformly enlarged, secondary hyperparathyroidism should be suspected and other diagnostic tests performed to identify the cause (e.g., renal secondary hyperparathyroidism); however, enlargement of all four glands may occur with primary hyperplasia. If one or several glands are slightly enlarged, parathyroid adenomas or primary hyperplasia should be suspected. Most dogs with primary hyperpara-throidy have a single parathyroid adenoma. If the parathyroid glands appear normal, ectopic parathyroid tissue may be located adjacent to the thyroid or as far caudal as the base of the heart. Alternatives to surgical parathyroidectomy include percutaneous ultrasound-guided ethanol ablation and percutaneous ultrasound-guided heat ablation.

**NOTE** If you cannot find any other cause of hypercalcemia in a patient with increased PTH concentrations, and if the parathyroid glands appear normal, look for ectopic parathyroid tissue.

### Preoperative Management

Before induction of anesthesia, diuresis should be instituted with 0.9% saline solution to help lower serum calcium concentrations (Box 22.22). In severe hypercalcemia, salmon calcitonin can be used to lower the serum calcium. Fluids should be used with caution in animals with severe renal dysfunction. Once the animal has been appropriately hydrated, furosemide administration may promote further calciuresis. Electrolytes should be monitored to prevent iatrogenic hypokalemia. Volume status and electrolytes should be normal before anesthesia.

### Anesthesia

Theoretically, notable hypercalcemia may cause bradycardia, peripheral vasoconstriction, and hypertension. Hypotension may occur during anesthesia associated with relaxation of peripheral vascular tone. Hypercalcemia also may predispose to cardiac arrhythmias. Anesthetic agents that potentiate arrhythmias (e.g., thiobarbiturates, halothane) should be avoided. Respiratory and metabolic acidosis should be avoided because elevations in pH will increase the free fraction of calcium, worsening hypertension and bradycardia.

### Surgical Anatomy

A discussion of the anatomy of the parathyroid glands is provided on p. 616.

### Positioning

The animal is placed in dorsal recumbency with the neck slightly hyperextended and forelimbs pulled caudally. The entire ventral neck and cranioventral thorax should be prepared for aseptic surgery.

### SURGICAL TECHNIQUE

All four parathyroid glands should be carefully inspected. If the external parathyroid gland is involved, the gland can be removed without removal of the thyroid gland; however, removal of the internal parathyroid gland requires that thyroidectomy be performed (see p. 617). The external parathyroid gland should be spared when the internal parathyroid gland is neoplastic, if possible. Visualization of the abnormal parathyroid gland may be facilitated by infusion of IV methylene blue in saline solution (see Box 22.13). Abnormal parathyroid tissue may stain dark blue with this procedure. A potential side effect of methylene blue administration is hemolytic anemia caused by Heinz body formation. Severe and occasionally fatal Heinz body anemia has been reported after the use of methylene blue. If carcinoma is suspected on the basis of apparent invasiveness of the tumor, complete thyroidectomy and removal of draining lymph nodes are indicated.

### SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Small, fine instruments, such as iris scissors and Bishop-Harmon thumb forceps, facilitate removal of the parathyroid glands.

### BOX 22.22  **Diuresis of Hypercalcemic Dogs**

<table>
<thead>
<tr>
<th><strong>0.9% Physiologic Saline Solution</strong></th>
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<tr>
<td>90 mL/kg/d IV</td>
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<tr>
<th><strong>Furosemide (Lasix)</strong></th>
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<td>2–4 mg/kg IV q8–12h, or can give as CRI (load with 0.66 mg/kg bolus, then give 0.66 mg/kg/h for 4–5 h; alternatively, can estimate the IV or PO dose to be given over the course of the next 24 h, then give that amount as CRI over 24 h. Be sure patient is volume replenished before administering), or give total daily dose as CRI.</td>
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CRI, Constant-rate infusion; IV, intravenous; PO, orally.
Bipolar cautery forceps are advantageous for providing hemostasis because they allow finer control of coagulation than do unipolar forceps. Sterile Q-tips are useful for dissecting the parathyroid glands from the thyroid glands.

**POSTOPERATIVE CARE AND ASSESSMENT**

Hypocalcemia is the most common postoperative complication in dogs; it may be less common in cats. Hypocalcemia may occur after removal of a single parathyroid adenoma because negative feedback from high circulating levels of PTH suppresses function in the other normal glands. PTH has a functional half-life of 20 minutes; therefore PTH levels fall rapidly once neoplastic tissue has been removed. Hypocalcemia may be most pronounced in animals with higher preoperative serum calcium levels and in those with notable skeletal demineralization. However, prediction of the severity of postoperative hypocalcemia using preoperative factors has proven challenging.26,27 Treatment of hypocalcemia is presented in Box 22.21; chronic treatment should not be necessary in these patients. Renal function should be monitored postoperatively in patients with hypercalcemia.

**PROGNOSIS**

The prognosis for long-term survival after parathyroidectomy for hyperparathyroidism secondary to adenomas or hyperplasia is excellent if severe renal damage has not occurred. Following surgical removal of parathyroid carcinomas, 1-, 2-, and 3-year survival rates were estimated to be 72%, 37%, and 30%, respectively.28 In recent studies, percutaneous ultrasound-guided radiofrequency heat ablation was successful in 69% of dogs, whereas percutaneous ultrasound-guided ethanol ablation was successful in 85% of dogs.29,30

**THYROID CARCINOMAS IN DOGS**

**DEFINITIONS**

Thyroid neoplasms may be carcinomas (malignant) or adenomas (benign). Carcinomas may arise from follicular cells and may be classified as follicular, compact, papillary, or mixed, or they may arise from parafollicular or C cells (medullary thyroid carcinomas).

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

Thyroid neoplasms make up slightly more than 1% of all canine tumors. Canine thyroid carcinomas are more common than adenomas, whereas functional adenomas prevail in cats (see the discussion of feline hyperthyroidism on p. 617). Carcinomas and adenocarcinomas represent nearly 90% of thyroid cancers; adenomas represent approximately 9% in dogs. Carcinomas are further divided into differentiated follicular cell carcinomas and medullary thyroid carcinomas, although outcomes following surgical removal are comparable between the two.29 Carcinomas are generally rapidly growing, highly invasive tumors that frequently metastasize to the draining lymph nodes and lungs. Reportedly, large tumors (i.e., those larger than 100 cm³) are typically associated with pulmonary metastasis. Although histologic classification of thyroid tumors based on the predominant microscopic pattern has been done (e.g., compact cellular or solid, follicular, mixed solid follicular, anaplastic), the histologic pattern has been thought to correlate poorly with prognosis. However, medullary thyroid carcinomas are more apt to be well circumscribed and resectable and to have gross and histologic characteristics of a less malignant nature compared with other thyroid carcinomas. Ectopic thyroid tumors have been reported at the heart base, caudal mediastinum, and tongue.

**NOTE** Warn owners that surgical excision of canine thyroid tumors is usually difficult because of the invasiveness of the tumors and the tendency toward substantial hemorrhage.

Tumors arising in cystic remnants of the thyroglossal duct are rarely reported in dogs. They are usually well-circumscribed, fluctuant, movable enlargements in the ventral midline cervical region. Histologically, they are usually well-differentiated papillary carcinomas. Because more than a third of dogs with thyroid carcinomas have multiple malignancies, thorough staging should be performed before surgery.29

**DIAGNOSIS**

**Clinical Presentation**

**Signalment**

Dogs between 10 and 15 years of age have a significantly increased chance of developing thyroid cancer. Breeds most commonly affected are golden retrievers, beagles, and Siberian huskies. A gender predisposition is not apparent.

**History**

Affected animals are often presented for evaluation of a palpable cervical enlargement, dysphagia, dyspnea, coughing, voice change, and/or exercise intolerance. Respiratory abnormalities may be the result of tracheal compression or pulmonary metastasis, and regurgitation may be caused by compression and/or invasion of the esophagus. In rare cases, hyperthyroidism (i.e., polydipsia, polyuria, weakness, restlessness, and a propensity to seek cool places) is caused by canine thyroid carcinomas.

**Physical Examination Findings**

A ventral cervical mass is often palpable. Carcinomas usually appear firm and poorly encapsulated; adenomas are typically small and freely movable. Abnormal lung sounds may occur secondary to pulmonary metastasis. Bilateral ptosis and prolapse of the nictitating membrane may be associated with paralysis of the extraocular and intraocular muscles, secondary to thyroid adenocarcinoma invasion of the cavernous sinuses in dogs.

**Diagnostic Imaging**

Cervical radiography or ultrasonography may reveal diffuse cervical edema and soft tissue swelling caudal to the mandible and surrounding the trachea. The mass may be partly mineralized. Thoracic radiographs should be taken to identify pulmonary metastasis. Thyroid imaging (see p. 618) may reveal abnormal thyroid gland uptake (heterogeneous uptake with “hot” and “cold” regions compared with normal thyroid or salivary gland uptake) and focal accumulations of the radiopharmaceutical in the lungs, indicative of pulmonary metastasis. CT angiography can be beneficial in the differentiation of thyroid versus nonthyroid neck masses, aids in the staging of thyroid carcinomas, and is used to determine the degree of invasiveness to aid surgical planning.
Laboratory Findings
Cytologic evaluation of a fine-needle aspirate of the cervical mass may reveal bizarre, pleomorphic cells consistent with neoplasia. Nondiagnostic samples may be obtained if the sample is contaminated with blood or is hypocellular. Additionally, neoplastic follicular epithelial cells are fragile and are often broken during sample preparation. Hyperthyroidism and hypothyroidism are occasionally associated with thyroid carcinomas; therefore measurement of serum fT4 and endogenous canine TSH concentrations is warranted. Hematologic and serum biochemical results are often normal. Hypocalcemia has been reported in a dog with a thyroid medullary carcinoma.

DIFFERENTIAL DIAGNOSIS
Cervical swelling caused by thyroid neoplasia must be differentiated from abscesses, lymphadenopathy, or sialadenopathy. This can usually be done by cytologic evaluation of fine-needle aspirates.

MEDICAL MANAGEMENT
Dogs with thyroid carcinomas, particularly if hyperthyroid, may be palliated with radioactive iodine ($^{131}$I); however, much larger doses of $^{131}$I appear to be necessary in dogs than in cats with thyroid adenomas, and this option is not routinely used. Chemotherapy with doxorubicin may benefit animals in which complete excision is not possible. External beam RT appears beneficial for reducing tumor volume in animals after debulking procedures; however, large doses are required. Fractionated, definitive RT using multiple, moderate doses of radiation may be effective in providing local control of invasive thyroid carcinoma in dogs. Linear accelerators have replaced cobalt therapy for treatment of these tumors.

SURGICAL TREATMENT

Surgical excision of thyroid adenomas is the treatment of choice. Surgical removal of thyroid carcinomas is often difficult because of their invasive nature and pronounced vascularity (Fig. 22.25) but should be considered if metastasis is not evident and if the lesion is localized. Marginal excision (i.e., outside the tumor pseudocapsule) in tumors that are freely movable results in fewer complications than more extensive resection and does not appear to affect the local recurrence rate. Adjunctive RT and/or chemotherapy may be warranted after marginal excision, or if complete surgical excision is not possible. Chemotherapy may be indicated if debulking is done in animals with metastasis.

NOTE: Have blood available during surgery because hemorrhage is often excessive.

Preoperative Management
Substantial electrolyte and acid-base abnormalities should be corrected before surgery. Fluid therapy should be initiated before surgery in geriatric patients with reduced renal function and in those that are dehydrated.

Anesthesia
In human beings, life-threatening thyroid storms are reported intraoperatively and postoperatively for thyroid tumors. Clinical signs of tachycardia or arrhythmias may occur because of catecholamine release, and treatment should be anticipated. It may be wise to avoid drugs that are arrhythmogenic (e.g., barbiturates, halothane) in these patients. This complication is more likely to occur in cats than in dogs. Most thyroid tumors in dogs are nonsecreting; however, it is beneficial to be prepared to treat hypertension and tachycardia with β-blockers in the perioperative period.

Surgical Anatomy
The surgical anatomy of the thyroid glands is discussed on p. 616. Important structures that may adhere to or surround the tumor include the carotid artery, the internal jugular vein, the recurrent laryngeal nerve, and the esophagus. These structures should be identified and preserved, if possible, during the dissection.

Positioning
The animal is placed in dorsal recumbency with the neck slightly hyperextended. The front limbs should be tied back, away from the neck. The entire neck, cranial thorax, and caudal intermandibular space should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE
Make a ventral midline incision over the thyroid glands. Identify the neoplastic mass and adjacent structures. If necessary, ligate the carotid artery and jugular vein. Remove the mass (thyroid and parathyroid glands) by a combination of sharp and blunt dissection. Identify and remove abnormal cervical lymph nodes. Use electrocautery, vascular clips, Ligasure, and/or ligation to provide hemostasis. Inspect the contralateral thyroid and biopsy or remove if indicated. Close the incision routinely. Submit tissue for histologic evaluation (Fig. 22.26).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS
These tumors are frequently very vascular, and electrocautery is useful for obtaining hemostasis.
REFERENCES


POSTOPERATIVE CARE AND ASSESSMENT

A light pressure wrap may be used postoperatively to help reduce hemorrhage and swelling; however, it should be placed with care and monitored to prevent airway obstruction. Hematocrit should be monitored postoperatively and transfusions given as necessary. If unilateral thyroparathyroidectomy is performed, the animal should be observed for hypocalcemia or hypothyroidism, but supplementation is not usually necessary. If bilateral thyroparathyroidectomy is performed, vitamin D, calcium, and thyroid supplementation should be initiated postoperatively (see p. 624).

PROGNOSIS

The prognosis is good following surgical treatment of mobile thyroid tumors and irradiation of fixed thyroid carcinomas, with median survival times greater than 3 years. Median survival time for dogs with local or regional tumors (i.e., stage II or III) was significantly longer (839 days) than median survival time for dogs with metastasis (366 days). Vascular invasion, either grossly or histologically, has been shown to be a negative predictor for disease-free interval.31 Following simultaneous bilateral thyroidec- tomy for discrete, mobile thyroid tumors, median survival was reported as 38.3 months; parathyroid gland tissue was preserved or reimplanted in 40% of dogs.55


