that underwent ovariohysterectomy years before developing Cushing's syndrome, the metal sutures have caused bruising years later. These abnormalities are due to decreased subcutaneous tissue secondary to the hypercortisolism. Wounds that do heal do so tenuously, with fragile, thin scar tissue equivalent to the striae seen in humans (see Fig. 118–9). Healing skin lesions often undergo dehiscence because of the limited amount of fibrous tissue present.

Calcinosis Cutis. Calcium deposition in the dermis and subcutis is an uncommon but well-described sign associated with Cushing's syndrome. On examination, these areas feel like firm plaques in or under the skin, almost as if a collar stay were inserted into these areas. Common locations for this calcium deposition, called calcinosis cutis, include the temporal area of the head and the dorsal midline, neck, ventral abdominal, and inguinal areas (Fig. 118–12). The exact pathogenesis is not known.

Obesity

Owners of hyperadrenal dogs usually comment on their pets' apparent weight gain. In fact, dogs with hyperadrenocorticism do not usually gain a large amount of weight. Rather, these dogs have fat redistribution, as mentioned previously, and a potbellied appearance, which mimics weight gain. Truncal obesity is a classic symptom of Cushing's syndrome. In dogs and humans, it appears to occur at the expense of muscle and fat wasting from the extremities and subcutaneous stores; true obesity is present in less than one-half the dogs.

Respiratory Signs

Panting. Dogs with hyperadrenocorticism often are noted to be short of breath or to have a rapid respiratory rate while at rest. These animals have increased fat deposition over the thorax plus the wasting and weakness of the muscles involved in respiration. The increased pressure placed on the diaphragm due to fat accumulation in the abdomen and liver enlargement further accentuates disturbances in ventilatory mechanics. Coughing is not a common owner complaint.

Signs of mild respiratory distress are believed to be exaggerated by a marked reduction in expiratory reserve volume and decreased chest wall compliance, which increase the work of breathing. If such a dog also has a collapsing trachea (a common problem in smaller breeds), the combination of expiratory distress associated with the tracheal problem and the changes seen with obesity can cause marked respiratory signs. Similar problems can easily be appreciated if the obese dog also has chronic mitral and/or tricuspid valvular fibrosis. Signs become further exaggerated with the stress of excitement or exercise.

Thromboembolism. Thromboembolism is a recognized problem in humans and dogs with Cushing's syndrome. Dogs with pulmonary thromboembolism can have chronic signs or develop acute severe respiratory distress (described elsewhere in this chapter).

Testicular Atrophy or Failure to Cycle

A male dog with Cushing's syndrome usually has bilaterally small, soft, spongy testicles. A female dog with Cushing's commonly ceases estrus cycle activity. The duration of anestrus often reflects the duration of subclinical or clinical hypercortisolism. These would be unusual owner concerns because so many pets are old, neutered, or both. If the pet is intact, the owner either is unaware of the problem or associates the change with age (see Physical Examination).

Myotonia (Pseudomyotonia)

Rarely, dogs with hyperadrenocorticism develop a distinct myopathy characterized by persistent active muscle contraction after cessation of voluntary effort (this has been noted in only 5 of more than 800 dogs with Cushing's syndrome). Historically, these dogs have had a stiff gait (especially in the pelvic limbs) that was present from the time the other signs of hyperadrenocorticism developed. One of our dogs could not ambulate with its rear legs. Pelvic limb muscle stiffness is obvious on physical examination. Myotonic, bizarre high-frequency discharges are noted on electromyography. Histologic, electron microscopic, and histochemical findings in the musculature of several dogs with Cushing's myotonia are characteristic of noninflammatory degenerative myopathy. Clinical signs may improve after successful therapy for hyperadrenocorticism. The cause for this unusual phenomenon in hyperadrenocorticism is not known.

Figure 118–12. *A* and *B*, Areas of skin altered dramatically by calcinosis cutis.

Neurologic Problems

See section, Central Nervous System Signs.

Physical Examination

General Review

The physical examination on a typical dog with Cushing's reveals an individual that is stable and hydrated, has good mucous membrane color, and is not in distress. Veterinarians typically observe many of the signs seen by owners on physical examination of hyperadrenocorticism dogs. Among these abnormalities are abdominal enlargement, increased panting, truncal obesity, bilaterally symmetric alopecia, skin infections, and comedones (hair follicles filled with keratin and debris that usually are black and easily expressed). Hyperpigmentation, ectopic calcification, testicular atrophy, clitoral hypertrophy, hepatomegaly, and easy bruisability are common (Table 118–4). There is a remarkable variation in the number and severity of these signs. These dogs may have a single dominant sign, or 10 signs.

Hyperpigmentation

Hyperpigmentation may be diffuse or focal (see Fig. 118-8*A*). Histologically, there are increased numbers of melanocytes in the stratum corneum, basal epidermis, and dermal tissues. Because hyperpigmentation has been observed in dogs with either pituitary or adrenal causes for Cushing's syndrome, the likelihood of excess secretion of alpha-MSH, as a by-product of ACTH production (see Fig. 118–2), being the sole cause of hyperpigmentation is not strongly supported.[1]

Hepatomegaly

An enlarged liver is typical of hyperadrenocorticism, contributing to the abdominal enlargement previously discussed. The liver typically is swollen, large, and pale. Hepatomegaly is easily palpated because of the weak abdominal muscles. The liver may be so large in some dogs that the veterinarian may become suspicious of a large abdominal tumor or tense ascites.

Liver biopsy samples from animals with hypercortisolism usually reveal steroid hepatopathy: centrilobular hepatocytic vacuolation with few, often single, large vacuoles displacing the nucleus to the periphery of the cell. Hepatocellular glycogen accumulation is concentrated in periportal hepatocytes. Lipid deposits are not demonstrable with Sudan III stains, and hepatocellular necrosis, although present, is not a significant feature. Vacuolization alone can be caused by various problems. Steroid hepatopathy does imply chronic elevation in circulating glucocorticoids.

TABLE 118–4. PHYSICAL EXAMINATION FINDINGS IN DOGS WITH HYPERADRENOCORTICISM

Thin skin	Hepatomegaly
Bilaterally symmetric alopecia	Panting
Acne (skin infection, comedones)	Bruising
Cutaneous hyperpigmentation	Exophthalmos
Calcinosis cutis	Testicular atrophy
Abdominal enlargement	Clitoral hypertrophy
Muscle wasting of extremities	

Testicular Atrophy, Anestrus, and Clitoral Hypertrophy

The negative feedback effects of hypercortisolism result in decreased pituitary gonadotropin secretion. This explains the testicular atrophy, decreased libido, and depressed plasma testosterone concentrations typically seen in male dogs. Testicular androgen secretion is reduced, whereas adrenal androgen secretion is increased. The physiologic effect of adrenal androgens, however, is negligible in males, and the reduction in testicular androgren is significant. The final result is that these males are feminized. Plasma testosterone concentrations averaged 4.7 ng/ml in normal males versus the significantly lower 1.2 ng/ml in male dogs with Cushing's.[19]

In female dogs, negative feedback effects of hypercortisolism depress pituitary secretion of gonadotropins, as in males. This results in prolonged anestrus. Abnormal adrenal function in Cushing's results in excessive secretion of adrenal androgens, and their peripheral conversion results in clinical androgen excess (virilization). A small number of these dogs have clitoral hypertrophy. The average plasma testosterone concentration in normal female dogs was 20 pg/ml, whereas in females with Cushing's, it was 30 pg/ml.[19]

Ectopic Calcification

In addition to hyperadrenocorticism's causing the previously described calcinosis cutis, ectopic calcification has been seen involving the tracheal rings and bronchial walls, kidneys, and, rarely, major arteries and veins. This calcification may be noted only histologically in some dogs but occasionally will be visible radiographically. Calcific band keratopathy, a syndrome characterized by a gray-white superficial corneal opacity horizontally oriented in the interpalpebral opening, was reported in two dogs with hyperadrenocorticism.[34]

Bruisability

Easy bruisability is common after venipuncture in dogs and cats with Cushing's (see Fig. 118–11). This reflects the poor wound healing associated with suppressed tissue granulation secondary to glucocorticoid excess (see p. 1546.)

Sudden Acquired Retinal Degeneration Syndrome

A retinal disorder of unknown etiology, sudden acquired retinal degeneration syndrome, causes sudden and permanent blindness in adult dogs. The syndrome is characterized by noninflammatory degeneration and loss of retinal photoreceptors. An association with hyperadrenocorticism has been suggested.[35] Strong evidence has yet to be presented that confirms the presence of hyperadrenocorticism in a significant number of these dogs.

In-Hospital Evaluation

General Approach

A dog or cat suspected of having hyperadrenocorticism should be thoroughly evaluated before specific endocrine procedures are undertaken. Initial tests should include clinicopathologic studies (complete blood count [CBC]; urinalysis with culture; and a chemistry profile, including liver en-

zymes, renal function tests, calcium, phosphorus, sodium, potassium, cholesterol, blood glucose, total plasma protein, plasma albumin, and total bilirubin). In addition to blood and urine testing, abdominal ultrasonography (less ideally radiography) should be completed in these dogs and cats. Finding a large percentage of abnormalities on initial screening tests that are consistent with hyperadrenocorticism allows the veterinarian to establish a presumptive diagnosis (Table 118–5). The more expensive and sophisticated studies needed to confirm a diagnosis and localize the cause of the syndrome can then be presented to the client.

The initial results not only ensure that the veterinarian is pursuing the correct diagnosis but also alert the clinician to concomitant medical problems. These problems may be common (urinary tract infection) or unexpected (congestive heart failure), but in either case, they should not be ignored.

Complete Blood Count

Excessive production of cortisol results in neutrophilia and monocytosis caused by steroid-produced capillary demargination of these cells and by the subsequent prevention of normal egress of cells from the vascular system. Lymphopenia is probably the result of steroid lympholysis, and eosinopenia results from bone marrow sequestration of eosinophils. These changes are seen as a stress response in the white blood cell differential. About 80 per cent of hyperadrenal dogs have reduced lymphocyte and eosinophil counts, and 20 to 25 per cent have increased total white blood cell numbers. The red blood cell count usually is normal, although mild polycythemia occasionally may be noted because of the previously described ventilatory problems or, in females, because of androgen stimulation of the bone marrow.

TABLE 118–5. HEMATOLOGIC, SERUM BIOCHEMICAL, URINE, AND RADIOGRAPHIC ABNORMALITIES TYPICAL OF HYPERADRENOCORTICISM*

TEST	ABNORMALITY
Complete blood count	Mature leukocytosis
	Neutrophilia
	Lymphopenia
	Eosinopenia
	Erythrocytosis (females)
Serum chemistries	Increased alkaline phosphatase (sometimes extremely elevated)
	Increased ALT
	Increased cholesterol
	Increased fasting blood glucose
	Increased or normal insulin
	Abnormal bile acids
	Decreased BUN
	Lipemia
Urinalysis	Urine specific gravity <1.015, often <1.008
	Urinary tract infection
	Glycosuria (<10% of cases)
Radiograph/Ultrasound	Hepatomegaly
	Excellent abdominal contrast
	Pot belly
	Distended bladder
	Osteoporosis
	Calcinosis cutis/dystrophic calcification
	Adrenal calcification (usually adrenal tumor)
	Congestive heart failure (rare)
	Pulmonary thromboembolism (rare)
	Calcified trachea and mainstem bronchi
	Pulmonary metastasis of adrenal carcinoma
Miscellaneous	Low T_4/T_3 concentrations
	Response to TSH that parallels normal but both pre and post values are low
	Hypertension

*It would be unusual for an individual animal to have all these abnormalities.

Blood Glucose and Plasma Insulin

Dogs and cats with hyperadrenocorticism occasionally have mild increases in fasting plasma glucose concentrations and, less commonly, overt diabetes mellitus. Glucocorticoids increase gluconeogenesis and decrease peripheral utilization of glucose by antagonizing the effects of insulin. Glycosuria may be manifested if the renal threshold for plasma glucose (180 to 220 mg/dl) is exceeded. In comparing fasted normal dogs with fasted non-diabetic dogs with Cushing's (those without glucose in their urine), the average morning plasma insulin concentration was 12 μU/ml in the controls and 38 μU/ml in the dogs with naturally occurring hyperadrenocorticism. Plasma glucose concentrations averaged 85 mg/dl in the control dogs and 111 mg/dl in the hyperadrenal dogs.[19] In most dogs with Cushing's, the increased secretion of insulin partially controls carbohydrate intolerance but may not be adequate to normalize glucose parameters. These abnormalities usually dissipate with successful therapy for Cushing's syndrome.

Blood Urea Nitrogen

The diuresis stimulated by glucocorticoids causes a continual urinary loss of blood urea nitrogen (BUN). Because a differential diagnosis for polydipsia and polyuria in an older dog would be renal disease, the normal-to-decreased BUN values (similar results are seen in the creatinine concentration) quickly dismiss that concern.

Quite uncommonly, a dog is diagnosed with renal failure and concurrent hyperadrenocorticism. If the Cushing's diagnosis is certain, major concerns remain regarding treatment. Such a dog may be helped by the Cushing's (i.e., the appetite) and well-being may be enhanced by the cortisone excess. Further, renal perfusion may be enhanced by or hindered by the Cushing's condition.

Alanine Aminotransferase

The alanine aminotransferase (ALT) concentration commonly is elevated in dogs with Cushing's. This usually is a mild elevation believed to occur secondary to liver damage caused by swollen hepatocytes, glycogen accumulation, or interferences with hepatic blood flow. Hepatocellular necrosis, a minor but significant feature of steroid hepatopathy, is seen with enough frequency to account for mild increases in serum ALT.[19]

Alkaline Phosphatase

Sources. An increase in serum alkaline phosphatase (ALP) activity is the most common routine laboratory abnormality in canine hyperadrenocorticism.[26] ALP is increased in 95 per cent of dogs with hyperadrenocorticism. The serum ALPs are a group of enzymes that catalyze the hydrolysis of phosphate esters. The main source of ALP is the liver, with bone ALP contributing smaller amounts to the circulation. Both have serum half-lives of about 3 days. Intestinal, placental, and renal ALPs are not detectable in serum because their half-lives are only 3 to 6 minutes.[27]

Corticosteroid-Induced ALP. The major contributor to

the increased ALP in canine hyperadrenocorticism is induction of a specific and unique (to this species) isoenzyme of ALP by either endogenous or exogenous glucocorticoids.[28] In dogs with hyperadrenocorticism, 70 to 100 per cent of their ALP is specifically the steroid-induced fraction (SIALP). The subcellular source of this isoenzyme was found to be on the bile canalicular membrane of hepatocytes. ALP is one of the most common biochemical measurements used to screen for the presence of liver disease, and the ability to discriminate between steroid-induced and liver isoenyzmes of ALP is important. Heat inactivation is a reliable method for distinguishing between these two isoenzymes of ALP.[26] If a steroid-induced increase in ALP is established, further laboratory investigation to characterize a liver problem could be omitted. Alternatively, can identification of the steroid-induced isoenzyme be used to diagnose hyperadrenocorticism?

Several groups have evaluated the clinical application of assaying for SIALP. These studies have demonstrated rather uniform agreement. The greater the increase in ALP, the more reliable the results of SIALP measurement. SIALP is present in increased concentrations in most dogs with Cushing's. As such, the test is considered quite sensitive. The finding of increases in SIALP, however, is nonspecific. SIALP may be abnormal in dogs with primary hepatopathies as well as in those being treated with anticonvulsants, such as phenobarbital, diphenylhydantoin, and primidone. A major concern with an abnormal SIALP is the inability to distinguish three disorders commonly confused with naturally occurring hyperadrenocorticism: iatrogenic Cushing's, diabetes mellitus, and hepatopathies (Fig. 118–13). The conclusion reached by most groups has been that finding no SIALP in the serum may have diagnostic value in ruling out the diagnosis of hyperadrenocorticism but that an increase in SIALP can be caused by a variety of disorders and must be considered nonspecific.[28–32]

Cholesterol and Lipemia

Glucocorticoid stimulation of lipolysis causes an increase in blood lipid and cholesterol concentrations. Ninety per cent of dogs with Cushing's have increased plasma cholesterol concentrations. Lipemia is at least as frequent, and it may interfere with the accurate assessment of several clinicopathologic test results.

Serum Phosphate

Hypophosphatemia has been reported to occur in about one-third of dogs with hyperadrenocorticism.[23] This has been explained as resulting from a glucocorticoid-induced increase in the urinary excretion of phosphate.

BSP and Bile Acids

Bile acid measurements are more sensitive than the sulfobromophthalein (Bromsulphalein [BSP]) excretion and equivalent to ammonia tolerance tests in many conditions. These test results frequently are abnormal in dogs with Cushing's and do not aid in separating dogs with primary liver disorders from those with Cushing's.

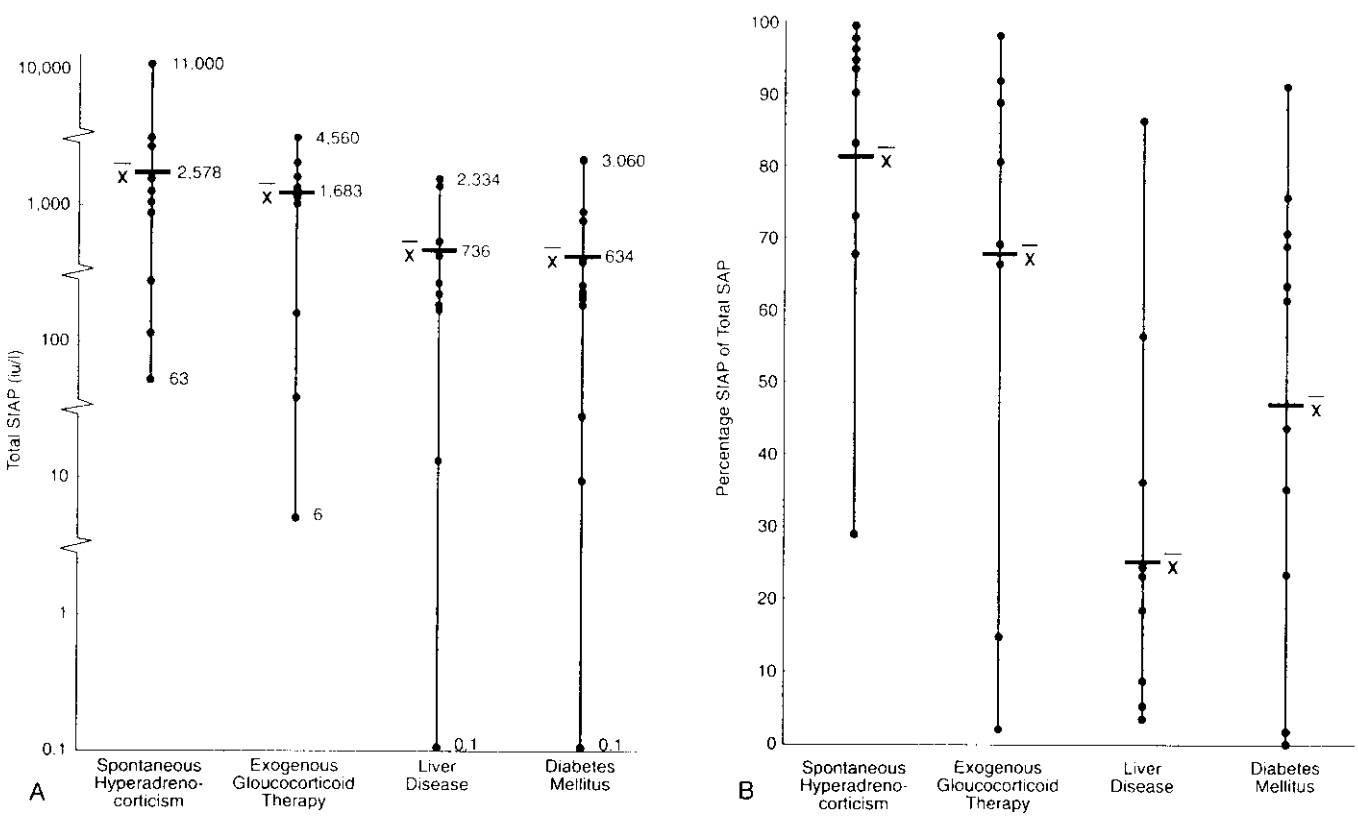

Figure 118–13. *A*, Group means and ranges of total serum steroid-induced alkaline phosphatase (SIAP) isoenzyme concentrations showing that this parameter lacks the specificity necessary to be a reliable screening test for canine hyperadrenocorticism. *B*, Percentage of total serum alkaline phosphatase (SAP), which is the steroid-induced isoenzyme of alkaline phosphatase (SIAP), by group.

Serum Electrolytes

Although of little diagnostic or clinical significance, mild abnormalities in the serum sodium (elevation) and potassium (depression) concentrations are seen in about one-half the dogs with Cushing's. Assessment of serum electrolyte concentrations becomes extremely important if a dog with hyperadrenocorticism develops anorexia, vomiting, or diarrhea because exaggerations of these abnormalities may become life-threatening.

Amylase and Lipase

If pancreatitis occurs, it is likely to be secondary to the lipemia or to the fact that such polyphagic dogs may eat garbage or large quantities of fat. In these instances, the lipase levels are elevated and may be an important diagnostic aid.

Urinalysis

Concentration. The urinalysis is perhaps one of the most important initial studies in the evaluation of a dog for hyperadrenocorticism. It is strongly recommended that owners obtain a urine sample by clean-catch before bringing the pet to the hospital or that a urine sample be collected at the time of initial examination. The most frequent abnormality is the finding of dilute urine (specific gravity less than 1.013), which occurs in 85 per cent of our cases. Other investigators have found dilute urine less frequently, perhaps because samples were obtained after the dogs had been hospitalized for hours or even days. It is less reliable to measure water intake in the hospital. Most water-deprived or frightened dogs with Cushing's can concentrate their urine to an osmolality well above plasma osmolality, although their concentrating ability usually remains less than normal.

Glucose. In addition to determining specific gravity, the veterinarian can assess the urine sample for the presence of glycosuria. Such a finding has been noted in 5 to 10 per cent of cases and would indicate that overt diabetes mellitus is present.

Infection. Because urinary tract infection is a common sequela to Cushing's, cystocentesis urine for culture should be obtained. About 50 per cent of dogs with Cushing's have a urinary tract infection at the time of initial examination. There are several potential explanations for this worrisome incidence of infection. First, glucocorticoid excess does increase the risk for infection. Second, the polyuria combined with muscle weakness in housebroken dogs creates a potential for bladder retention of urine, despite urination. Finally, dilute urine increases susceptibility to lower urinary tract infection.[33] Thus, the bladder constantly has dilute urine, which acts as a ready site for infection in an immunosuppressed dog. Control of these infections is important, although in some dogs, the infection is difficult to resolve because of pyelonephritis.

Thyroid Function Tests

Some of the clinical signs of hypothyroidism overlap with those of Cushing's (i.e., listlessness, bilateral symmetric nonpruritic alopecia, apparent weight gain, hypercholesterolemia). Chronic hypercortisolism (iatrogenic or naturally occurring) suppresses pituitary secretion of TSH, leading to secondary hypothyroidism.[34] Hypercortisolism may also change thyroid hormone binding to plasma proteins, enhance the metabolism of thyroid hormone, and decrease peripheral deiodination of thyroxine (T_4) to triiodothyronine (T_3). About 70 per cent of dogs with naturally occurring Cushing's have decreases in basal serum T_4, free T_4, and/or T_3 concentrations. Administration of TSH increased serum T_4 concentrations in a manner parallel to normal but usually not to normal concentrations (Fig. 118–14).[23, 35, 36]

Radiographs

General Approach. Radiographs of the chest and ultrasonography of the abdomen (the preferred tool for evaluating the abdomen) should be used in looking for changes consistent with the diagnosis of Cushing's. Veterinarians should also remember that most of these dogs are older and may have serious concurrent (perhaps subclinical) diseases that may be revealed radiographically.

Abdominal Detail and Hepatomegaly. Good contrast usually is observed in dogs and cats with Cushing's because of abdominal fat deposition. The potbellied appearance (60 per cent of dogs with Cushing's) and hepatomegaly may be obvious. About 80 to 90 per cent of these dogs have hepatomegaly. There is no obvious association between the duration of illness and the degree of hepatomegaly.[37]

Urinary Bladder. Distention of the urinary bladder may be seen radiographically. Some of these dogs have atonic bladders and may not be capable of voiding completely, maintaining large, partially filled bladders (Fig. 118–15).

Visualizing the Adrenals. Perhaps the most important but least common finding on abdominal radiographs is an adrenal mass. Positive identification of such a mass occurs infrequently because only 10 to 20 per cent of dogs with

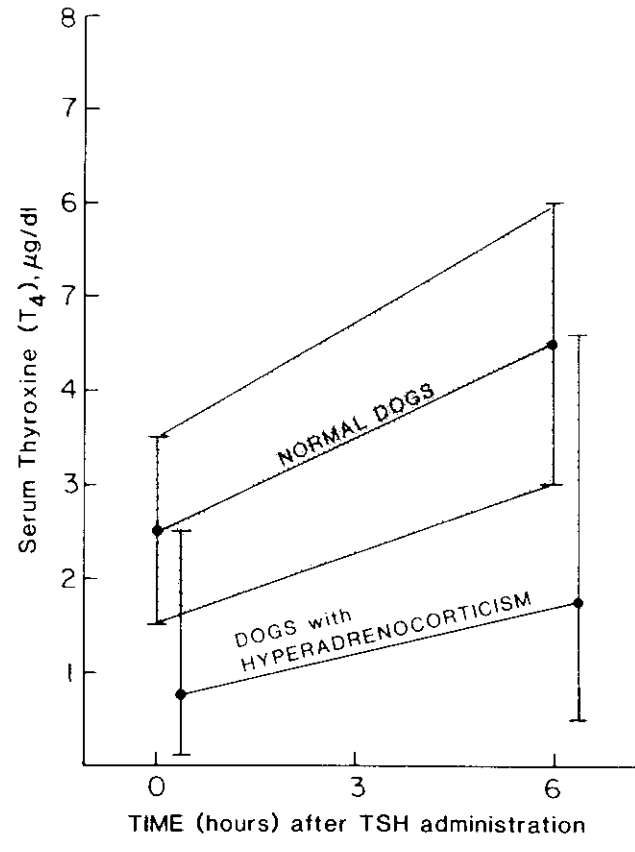

Figure 118–14. T_4 concentrations before and after TSH administration in normal dogs and those with hyperadrenocorticism. The Cushing's syndrome dogs may have normal or below normal values which parallel normal increases in serum T_4 concentrations.

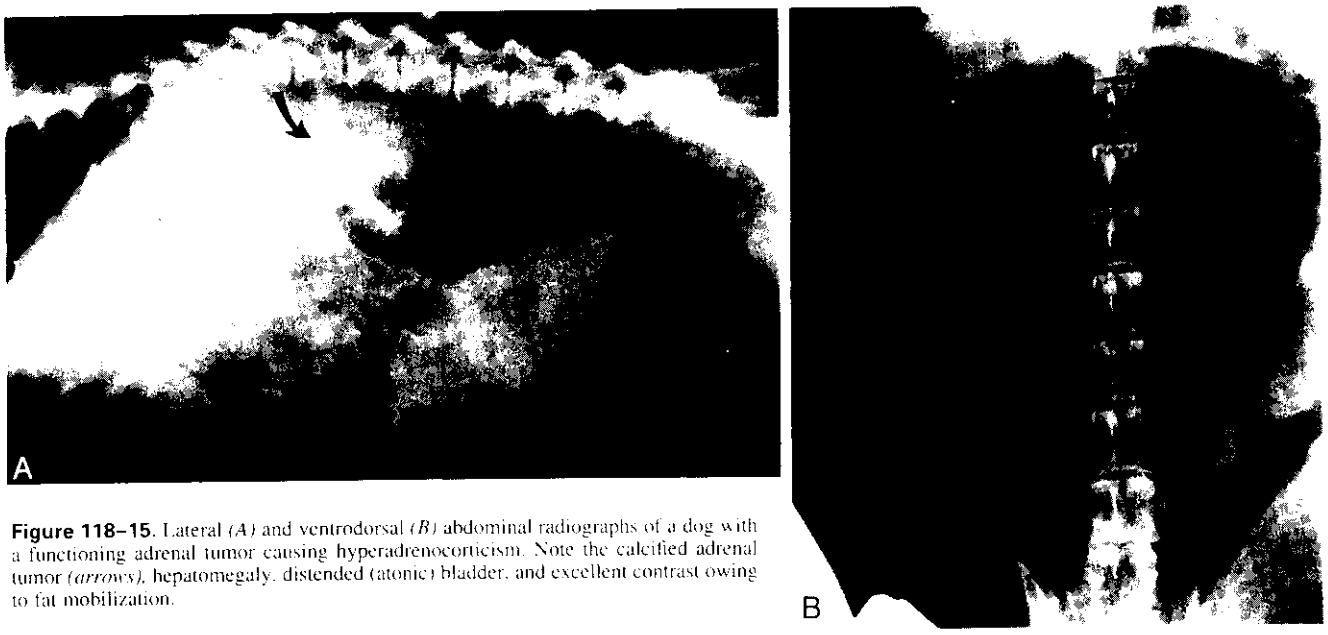

Figure 118–15. Lateral *(A)* and ventrodorsal *(B)* abdominal radiographs of a dog with a functioning adrenal tumor causing hyperadrenocorticism. Note the calcified adrenal tumor *(arrows)*, hepatomegaly, distended (atonic) bladder, and excellent contrast owing to fat mobilization.

naturally occurring hyperadrenocorticism have an adrenocortical tumor, and only about 50 per cent of these can be visualized radiographically because of calcification. Adenomas and carcinomas are calcified in relatively equal numbers (see Fig. 118–15).[12, 37, 38]

Osteoporosis. A distinct reduction in the radiographic density of the lumbar vertebral bodies relative to vertebral end plates may be detected in about 15 per cent of dogs with Cushing's syndrome. Glucocorticoids have a catabolic effect on bone matrix, increase urinary calcium excretion, and inhibit gastrointestinal absorption of calcium by interfering with the action of vitamin D. Thus, depletion of matrix accompanied by loss of mineral may be the cause of osteoporosis.

Dystrophic (Ectopic) Calcification. Radiographic signs of calcinosis cutis are seen in 10 to 20 per cent of dogs with Cushing's syndrome; a smaller number have dystrophic calcification that involves the renal pelvis, liver, gastric mucosa, or branches of the abdominal aorta. Ectopic calcification frequently is seen involving the tracheal rings and mainstem bronchi. Calcification of these structures, however, can be seen in normally aging dogs.

Thoracic Radiographs. The most common finding is calcification of tracheal rings. Osteoporosis may be suspected from the appearance of the thoracic vertebrae. Most important, radiographs must be evaluated for evidence of adrenocortical carcinoma lung metastasis, which occurs in a small percentage of these dogs. Another major concern is pulmonary thromboembolism.

Skull. Radiographs of the skull are not recommended. The studies usually are normal and require anesthesia. The bone destruction seen in the area of the sella turcica of some people with expanding pituitary tumors is not seen in the dog.

Ultrasonography

Background. Perhaps more than with any other tool, the value of ultrasonography directly correlates with the skill of the operator. Transverse, longitudinal, and oblique scanning from the ventral abdomen must be performed to thoroughly evaluate the adrenals.[39] Most (75 to 85 per cent) normal dog adrenals and 50 to 60 per cent of normal cat adrenals can be visualized.[40-42] In our recent experience, these estimates are quite conservative.[40] In both species, the left adrenal is easier to visualize than the right because of overlying bowel and several other factors.

Dogs or Cats with Cushing's. In hyperadrenocorticism, abdominal ultrasonography serves three major functions. First, it is part of the routine data base used to evaluate the abdomen for unexpected abnormalities (e.g., urinary calculi, masses, cysts). Second, if an adrenal tumor is identified, ultrasound is an excellent screening test for hepatic or other organ metastasis, tumor invasion of the vena cava or other structures, and compression of adjacent tissues by a tumor. Third, the study is used to evaluate the size and shape of the adrenals. If bilaterally normal-sized or large adrenals are visualized in a dog or cat otherwise diagnosed as having Cushing's, this is considered strong evidence in favor of adrenal hyperplasia caused by pituitary-dependent disease. Visualization of a normal or slightly enlarged left adrenal is nonspecific evidence that weakly points toward pituitary dependence. Visualization of only the right adrenal is considered suspicious because this adrenal usually is more difficult to see. If either adrenal is remarkably enlarged, irregular, or invading or compressing adjacent structures and the opposite adrenal cannot be visualized, suspicion of an adrenal tumor is heightened (Fig. 118–16).

Incidentally Discovered Adrenal Mass. With the increased use of sophisticated aids for evaluating the abdomen, unsuspected abnormalities are being identified with increased frequency. As many as 2 per cent of humans evaluated with ultrasonography and imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) have an adrenal mass.[43, 44] Dogs with clinical evidence of hyperadrenocorticism that have an adrenal mass should be evaluated for an adrenocortical tumor. If the dog or cat with an adrenal mass has no historical or physical examination findings suggestive of Cushing's, endocrine evaluation is not recommended. Some adrenal masses are normal. Other differential diagnoses include adrenal cysts, myelolipomas, hemorrhage, non-functioning (non–hormone-producing) tumor, pheochromocytoma, metastatic tumor, and granuloma.