Hypoadrenocorticism (“Addison’s disease”) is an uncommon disease in dogs. However, because of the potential for acute death in dogs with severe acid/base and electrolyte abnormalities, and the excellent prognosis with treatment, prompt diagnosis is crucial.

In dogs, Addison’s is most commonly caused by adrenocortical failure, usually secondary to immune-mediated destruction. Most patients exhibit signs of both cortisol and aldosterone deficiency. Clinical signs often include lethargy, decreased appetite, weight loss, vomiting, and diarrhea.

Clinical presentation and clinicopathologic abnormalities
Clinical presentation of hypoadrenocortical patients varies from patients with chronic “failure to thrive” (ADR) and/or gastrointestinal signs, to patients that present acutely in hypovolemic shock. Both groups of patients may have a history of improvement with fluid administration and/or glucocorticoid therapy.

Physical examination findings can also vary from almost normal to hypovolemic shock. Hyponatremia and hyperkalemia are the classic laboratory findings in dogs. However, these findings may be absent early in the disease process, or in dogs with “atypical hypoadrenocorticism,” which have signs of cortisol deficiency, but normal sodium, potassium, and chloride concentrations. Additional laboratory abnormalities may include azotemia, hypoglycemia, hypochloremia, hypocholesterolemia, hypercalcemia and metabolic acidosis (decreased tCO2/bicarbonate). Hypoadrenocorticism should be considered in patients that present for signs of hypoglycemia (such as seizures) and hypercalcemia. Because most patients also have a specific gravity <1.030, azotemic patients can be incorrectly diagnosed with primary renal failure. In these cases, the patient’s history and rapid response to fluid therapy should increase suspicion of hypoadrenocorticism.

Patients exposed to cortisol often exhibit neutrophilia and lymphopenia (“stress leukogram”). In the absence of cortisol, such as with hypoadrenocorticism, patients may be predicted to have neutropenia, lymphocytosis, and eosinophilia. In fact, these specific changes don’t occur very frequently in Addisonian patients. However, a number of Addisonians do have a “lack of a stress leukogram,” meaning that they do not have neutrophilia or lymphopenia. In a clinically ill patient, the findings of normal neutrophil and/or lymphocyte counts, with or without eosinophilia, are unexpected, and may raise suspicion of hypoadrenocorticism.

Additional diagnostics
In cases of moderate to severe hyperkalemia, an ECG may reveal spiked T-waves, absent p-waves, increased P-R interval, and/or bradycardia. Other basic diagnostic findings in hypoadrenocortical dogs are non-specific. Thoracic radiographs may reveal microcardia (consistent with hypovolemia). Abdominal ultrasound may reveal small adrenal glands.

Definitive diagnosis
Definitive diagnosis relies on results of an ACTH-stimulation test. Post-stimulation cortisol samples of <2 µg/dL are consistent with hypoadrenocorticism. Steroids given days prior to the test may blunt the response, and it is not uncommon for a dog with a history of recent glucocorticoid administration to have a post-stimulation cortisol of 2.5 – 5.0 µg/dL. Most synthetic glucocorticoids (including prednisone and methylprednisolone) will interfere with the cortisol assay itself, and may cause a falsely elevated cortisol result. However, dexamethasone does not interfere with the cortisol assay, and may be given prior to or during the ACTH stimulation test, if necessary.

Baseline cortisol: for rule-out purposes only!
Although definitive diagnosis of Addison’s requires an ACTH stimulation test, the disease can be RULED-OUT by checking baseline cortisol values. If the baseline cortisol is >3 µg/dL, the dog does not have hypoadrenocorticism. If the baseline cortisol is <2ug/dL, an ACTH stimulation test MUST be run to confirm the diagnosis. The baseline cortisol is most useful in patients without electrolyte abnormalities, that may be suspected of atypical hypoadrenocorticism because of chronic GI signs. Since it does not require the purchase of synthetic ACTH, it is much less expensive that the ACTH stimulation test.

Treatment
Treatment of hypoadrenocorticism depends on the presentation of the patient. If they present in hypovolemic shock (“Addisonian crisis”), diagnosis is usually unknown initially, and treatment is generally similar to that for any patient in hypovolemic shock. The first priorities in stabilizing a patient in Addisonian crisis are to correct the hypovolemia and the hyperkalemia, since these conditions are most likely to be fatal if not treated immediately. Although 0.9% NaCl is recommended because of its sodium content, isotonic
crystalloids such as Normosol-R and Lactated Ringer’s Solution may also be used. Hypoglycemic patients should be treated with dextrose.

If the dog is moderately hyperkalemic, the hyperkalemia will likely be corrected with fluid therapy alone. However, if the hyperkalemia is severe (>8.5 mEq/L) or causing ECG changes, additional therapy may be warranted. A 10% solution of calcium gluconate (2 to 10 mL/dog) may be administered intravenously over 10 to 15 minutes while monitoring for ECG changes associated with hypercalcemia. Although the effect is almost immediate, it lasts for only about 10 to 30 minutes. This treatment is cardioprotective and does NOT lower the potassium concentration. Simultaneous intravenous administration of dextrose (1 g/unit of insulin) and regular (R) insulin (0.2 U/kg) will decrease potassium levels within 15 to 30 minutes. A 5% dextrose solution in 0.9% sodium chloride should be administered after insulin treatment to alleviate hypoglycemia.

Glucocorticoids should be given to a patient during the crisis. Dexamethasone, 0.2 mg/kg, may be given initially. Although this dose is lower than recommended in some drug resources, it is equivalent to approximately 1.5 mg/kg of prednisone and is more than adequate. This dose is often given twice the first day, and then cut in half for the next two days. As soon as the patient is eating, he may be given oral prednisone. While in the hospital, the patient needs more than the normal physiologic dose (~0.2 mg/kg/day); approximately 1 mg/kg/day is commonly used. The dog may go home on an increased dose of 0.5 mg/kg/day for a couple of days, and then be tapered to around 0.1 – 0.3 mg/kg/day. This dose is adjusted based on the clinical signs of the dog (activity level, appetite, gastrointestinal signs), combined with the avoidance of side effects from the prednisone, such as PU/PD.

Following confirmation of hypoadrenocorticism, the dog should also be started on a mineralocorticoid replacement. The author’s preference is desoxycorticosterone pivalate (DOCP), 2.2 mg/kg, q25-28d. Although the first dose may be given IM in case dehydration impedes SQ absorption, subsequent doses can usually be given SQ. Electrolyte values should be checked 14 days after injection to assess the dose, and immediately prior to the next injection (25-28d) to assess duration of activity. Dose and frequency should then be modified based on these electrolyte values.

It is IMPERATIVE that owners be cautioned not to try to space out or skip DOCP injections for financial reasons. This almost always leads to an Addisonian crisis eventually, which risks the patient’s life and increases overall cost of treatment. Alternatively, fludrocortisone may be used as a mineralocorticoid supplement. It is oral and initially given at 0.02 mg/kg/d, It also has some corticosteroid activity.

Management of a chronic Addisonian involves the administration of prednisone and a mineralocorticoid, as described above. Additional glucocorticoids, 2-3 times the normal dose, should be given when the dog is stressed, such as prior to veterinary visits (even for DOCP injections), or when there are visitors to the home.

The prognosis for good quality of life is excellent with prompt treatment of hypoadrenocorticism. Even hunting dogs can return to normal activity (with adjustment of prednisone dose), and patients have a normal life expectancy.