

RECOMMENDATIONS

conversations with a cardiologist

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Diagnosis and Management of Systemic Hypertension in Cats and Dogs

“Systemic hypertension” (HT) refers to the persistent elevation of systemic blood pressure. Chronic elevation of systemic blood pressure results in damage to multiple end-organ systems; some of this damage may be clinically obvious (e.g. detached retinas) but other injuries may be hard to detect clinically (e.g. slowly progressive renal damage). Organs at highest risk for detectable damage include the eyes, brain, kidneys and heart. Because capillary blood pressure management is so critical in these organs, some of them have their own “local” blood pressure control systems (e.g. brain and kidney), but local control in these systems may be overwhelmed by systemic HT. Additionally, elevated systolic blood pressure requires increased left ventricular pressure work and results in secondary left ventricular hypertrophy and eventually cardiac dysfunction.

Systemic hypertension is typically classified as primary (“essential”) HT or secondary HT. Some veterinary clinicians have attempted to distinguish essential HT from idiopathic HT (i.e. systemic HT in the absence of overt, clinically apparent causal disease). Use of the term “idiopathic” acknowledges that there may be a causal disease (e.g. renal disease) that is responsible for the HT but that the causal disease is in a pre-clinical phase. In cases where the underlying disease is rare (e.g. adrenal tumors such as pheochromocytoma or aldosterone secreting adenoma), the discovery of the cause of the HT is dependent on the thoroughness of the diagnostic testing.

From diagnosis to treatment, keeping a few important points in mind when evaluating the patient will prevent the clinician from overlooking critical information.

DIAGNOSIS

Diagnosis of systemic hypertension involves 4 steps: identifying the correct population to test, correct measurement/assessment of blood pressure, careful screening for target organ damage, and evaluation for (and management of) any causative disease.

► Identification of the correct patient population:

Routine evaluation of blood pressure during wellness exams is generally not recommended in dogs and cats, with a few exceptions. Dogs and cats with diseases known to be associated with HT, including any renal disease, any adrenal disease, diabetes mellitus and hyperthyroidism (in cats) are candidates



for blood pressure evaluation regardless of the presence or absence of clinical signs. Cats with hyperthyroidism should be assessed for HT as part of their initial diagnostic evaluation, but because renal disease and hyperthyroidism occur at high prevalence in older cats, feline patients ≥ 10 years of age should have their blood pressure measured at each yearly check-up.

► Correct measurement of blood pressure:

References are available describing correct measurement of blood pressure in dogs and cats using the non-invasive Doppler or oscillometric techniques¹⁻³. When measuring blood pressure in a clinical setting, the so-called “white coat effect” should be neither over- nor under-estimated. The term “white coat effect” has been used to describe increases in blood pressure that occur as a result of (typically) nervous responses during blood pressure measurement in the doctor’s office. This response has been documented in normal cats⁴ but it is unclear how often this effect occurs in typical daily practice in cats or in dogs. In both species, however, there is an advantage in measuring blood pressure in a quiet and preferably dimmed environment after the patient has had a chance to acclimate to the situation. It is often quite helpful to have the owner hold the patient if this is feasible. When assessing the readings, multiple readings (3-5 per session) should be averaged to deliver a “representative” value. The most common blood pressure value used to diagnose HT in dogs and cats is systolic blood pressure (SBP).

► Careful screening for target organ damage:

“Target organ damage” or TOD, refers to organ injury that may be detectable in some HT patients. TOD can be hidden (left ventricular hypertrophy with no outward clinical signs), subtle (mild depression), or catastrophic (retinal detachment

leading to blindness). The body systems most likely to sustain detectable damage as a result of systemic HT include the eyes, the nervous system, the kidneys and the heart. **If a patient is known or suspected to have HT, the patient should be screened for TOD of each of these systems (see below). Conversely, discovery of any of these findings in a patient should lead to blood pressure assessment.**

- Ocular damage secondary to systemic hypertension is referred to as “hypertensive choroidopathy” and occurs in approximately 50-60% of dogs and cats with HT. The amount of damage does not appear to be directly related to the severity of HT in either species, but the risk of hypertensive choroidopathy increases at SBP \geq 180 mmHg.

Hypertension rarely leads to congestive heart failure in dogs and cats unless it is superimposed on pre-existing heart disease.

Typical findings in patients with hypertensive choroidopathy include retinal hemorrhage, hyphema, subretinal edema and retinal detachment. Presence of any of these findings in a hypertensive patient indicates that the HT is an emergency situation and HT should be treated immediately before any additional diagnostic testing is performed.

- Neurologic abnormalities due to systemic HT likewise indicate an emergency situation. Typical signs of HT-related neurologic abnormalities include intracranial signs: depression or obtundation and photophobia. Grand mal seizures are not often seen as a result of HT, but focal facial seizures may be noted, especially in cats with severe HT. Neurologic abnormalities attributable to HT are considered an emergency and in all cases where cerebral edema is suspected, blood pressure should be measured to be sure HT is not the cause of the signs prior to any administration of mannitol that may be considered.
- Renal evidence of hypertensive damage may be diagnosed as progression of known renal disease (e.g. progressive azotemia) or diagnosis of proteinuria, an indication of glomerular damage. In cases in which renal disease/proteinuria are considered the likely cause of the HT, it may be difficult to differentiate the natural progression of the disease from additional damage actively caused by intraglomerular HT occurring as a result of systemic HT. As a result, renal disease/proteinuria discovered in the course of a patient’s evaluation should trigger assessment of blood pressure, but in most cases (i.e. except acute renal failure), this type of TOD is not treated as a HT emergency.

- Cardiac TOD can be identified as left ventricular hypertrophy by echocardiography, but may be evident clinically only if a heart murmur or a gallop is detected by auscultation. Discovery of a left-sided heart murmur or gallop in a dog or cat is an indication for blood pressure measurement and is necessary exclusion before diagnosing hypertrophic cardiomyopathy in cats. Hypertension rarely leads to congestive heart failure in dogs and cats unless it is superimposed on pre-existing heart disease. However, patients with significant hypertensive heart disease can progress to congestive heart failure if excessive fluids are administered. Thus, any patient (especially cats) who unexpectedly develops pulmonary edema after administration of fluids should be screened for HT.

► **Evaluation for causative disease: In most cases, HT is a complication of systemic disease rather than a disease in itself.** In dogs and cats, renal disease, adrenal diseases and diabetes mellitus may be associated with the development of HT. Additionally, in cats, hyperthyroidism is commonly associated with HT, and is often co-existent with chronic renal disease in older cats. When TOD due to HT is the diagnosis, SBP measurement (and acute therapy, if necessary) should be followed by testing for an underlying disease. This testing typically includes biochemical analysis and urinalysis, with special attention to quantification of proteinuria (if present) via a urine



protein/creatinine ratio. Cats should be carefully evaluated for physical signs of hyperthyroidism, such as palpation for a goiter, identification of tachycardia and hyperdynamic pulses, weight loss, and other relevant signs. Abdominal ultrasound can provide additional information regarding renal disease and allows for examination of the adrenal glands. Common findings are those typical of hyperadrenocorticism or pheochromocytoma (in dogs) or an aldosterone secreting tumor (in cats). Additional endocrine testing for hyperadrenocorticism, hyperthyroidism or measurement of blood aldosterone concentrations may be required in some patients.

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TREATMENT

Currently, most authors recommend antihypertensive therapy in patients with reliable SBP readings \geq 160-170 mmHg, especially if a causal disease condition has been identified. The presence of TOD in patients with SBP \geq 160 mmHg is an indication for immediate therapy. In patients without known causal disease conditions, SBP readings of 160-170 mmHg that may be situational can be confirmed with a repeat measurement before therapy is instituted.

Chronic systemic HT is often easier to control in cats than in dogs. In cats, oral amlodipine is the current drug of choice to treat HT (0.625 mg PO every 24 hrs for cats \leq 5 kg and 1.25 mg PO q 24 hrs for cats $>$ 5 kg). If proteinuria is documented, therapy with an angiotensin-converting enzyme inhibitor (ACEI, benazepril or enalapril, 0.5 mg/kg PO every 12-24 hrs) should be added. The target SBP in HT patients is $<$ 160 mmHg, and there is evidence that TOD may be better controlled if SBP is maintained $<$ 140 mmHg. Blood pressure and funduscopy findings should be rechecked in 3-7 days, depending on the severity of the initial HT and clinical signs, then every 3 months (approximately) in stable patients. In cats in which the original dose of amlodipine does not deliver adequate blood pressure control, the dose may be incrementally increased with careful recheck measurements to assure that blood pressure is within the target range (SBP \sim 110-150 mmHg) in the treated animal.

The blood pressure of hypertensive dogs may be somewhat more challenging to control. Most dogs benefit from ACEI therapy, especially if proteinuria is present. However, therapy with ACEI alone can be expected to result in a very modest (approximately 10% decrease) in SBP. The dogs with mild HT – blood pressure between 160-180 mmHg – may be controlled with ACEI alone (enalapril or benazepril, 0.5 mg/kg PO q 12 hrs). However, in the dog with initial SBP \geq 180 mmHg, dual therapy with ACEI and amlodipine (0.2-0.4 mg/kg PO q 24 hrs) likely will be needed. In most cases, the lower end of the amlodipine dosage range can be used initially, with recheck BP evaluations dictating increases in the dose. HT dogs often require increases in medical management over time, so they should have their blood pressure and funduscopy findings rechecked routinely (every three months if clinically stable). If patients are receiving ACEI and doses of amlodipine that are at the higher end of the dosage range and remain hypertensive, additional medications can be added and a phone consultation with or referral to a specialist is recommended.

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