

Systemic hypertension is among the most consistently identified and readily modifiable risk factors associated with canine cerebrovascular accidents (CVAs). When blood pressure remains chronically elevated, autoregulation of cerebral blood flow fails, increasing the risk of both ischemic and hemorrhagic stroke.^{1,2} Hypertension was the most common comorbidity in a multicenter cohort of 125 dogs with ischemic stroke.⁶

STROKE MECHANISMS

Ischemic stroke: sustained hypertension is suspected to drive small vessel disease through atherosclerosis and lipohyalinosis of small penetrating cerebral arteries, predisposing to luminal narrowing, occlusion, and focal infarction.^{2,5}

Hemorrhagic stroke: chronic pressure weakens cerebral vessel walls, increasing the risk of rupture, intracranial hemorrhage, and elevated ICP.^{1,2,5}

PREDISPOSING CONDITIONS: SCREEN EVERY 6 MONTHS¹

- Chronic kidney disease (CKD)^{1,3}
- Protein-losing nephropathy (PLN)^{1,6}
- Hyperadrenocorticism (Cushing's disease)^{1,3,7}
- Hypothyroidism (via dyslipidemia / atherosclerosis)^{1,5,7}
- Diabetes mellitus^{1,7}
- Pheochromocytoma¹
- Primary hyperaldosteronism¹
- Idiopathic (primary) hypertension¹
- Drug-induced (glucocorticoids)¹

MINIMUM STANDARD OF CARE

Any dog presenting with acute vestibular signs should receive both of the following before discharge. These are low-cost steps that identify the conditions most likely to cause repeat events.^{1,3}

- Blood pressure measurement¹
- Senior blood panel^{1,3}

Canine hypertension is usually secondary to an underlying condition.¹

Blood pressure screening is the most accessible preventative step against hypertensive **Target Organ Damage (TOD)** in the brain. Measure at every visit in the risk categories above.¹

Recognizing Neurologic Target Organ Damage: Do Not Wait for a Catastrophic Cerebrovascular Accident

- Altered mentation: obtundation, disorientation, stupor^{1,2}
- Vestibular signs: head tilt, spontaneous nystagmus, ataxia^{2,3,4}
- Acute focal deficits: sudden tetraparesis, facial asymmetry^{2,3}
- Unexplained acute behavioral change^{2,3}
- Cluster of transient neurologic episodes preceding a major vascular event^{2,5}
- Seizure activity: new onset, especially middle-aged to older dogs^{2,5,6}

ACVIM Diagnostic & Risk Guidelines¹

Classification is by systolic blood pressure (SBP): average 5–7 consecutive, consistent readings after acclimatization.¹

SYSTOLIC BP (MMHG)	CLASSIFICATION	TOD RISK	CLINICAL ACTION
< 140	Normotensive	Minimal risk	Routine annual monitoring
140–159	Prehypertensive	Low risk	Repeat measurement; investigate underlying disease
* Secondary prevention target: dogs with confirmed neurologic TOD are treated toward an SBP below 140 mmHg when safely achievable.¹			
160–179	Hypertensive	Moderate risk	Repeat twice over 8 weeks; treat if TOD present
≥ 180	Severely hypertensive	High risk	Treat; immediately if TOD present. Repeat BP within 2 weeks

First-line treatment: amlodipine besylate 0.2–0.4 mg/kg PO q24h is the preferred calcium channel blocker; up to 0.6 mg/kg may be used cautiously. Recheck BP within 1–2 weeks of initiating.¹ **Not as sole therapy in CKD or proteinuria:** a RAAS inhibitor is preferred first (enalapril or benazepril 0.5–2.0 mg/kg PO q12h), alone or with amlodipine.¹ All treatment must be individualized to the patient.¹

Refer to neurology: multiple vestibular events, acute focal deficits, or vertical nystagmus, all of which favor a central rather than peripheral localization.^{4,5} Refer for refractory hypertension, suspected pheochromocytoma, or primary hyperaldosteronism.¹
Refer for MRI: MRI is the imaging modality of choice for suspected cerebrovascular accident. It confirms the infarct, defines its territory, and distinguishes ischemic from hemorrhagic stroke, none of which can be established from clinical signs alone.^{3,5,6}
Refer for CT: if hemorrhage is suspected. CT detects acute bleeding but frequently misses ischemic infarcts, so a normal CT does not rule out a stroke and MRI remains indicated.^{2,5}

References

Clinical Quick Reference: Blood Pressure Screening & Cerebrovascular Risk in Dogs

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6. Danciu CG, Gonçalves R, Caldero CJ, et al. Comorbidities, long-term outcome and poststroke epilepsy associated with ischemic stroke: a multicenter observational study of 125 dogs. *J Vet Intern Med.* 2025;39(1):e17291. doi:10.1111/jvim.17291
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Dogs with Confirmed Neurologic TOD Require a Tighter Target

Neurologic injury from a CVA, including vestibular events, focal deficits, and seizure clusters, constitutes **target organ damage**.^{2,3} If TOD is present, start treatment right away on one reading; don't wait to repeat the measurement first.¹ The treatment goal for every hypertensive patient is under 140 mmHg; 160 mmHg is only the minimum fallback if 140 can't be reached.¹

A note on scope. Blood pressure thresholds, classification, target organ damage definitions, and antihypertensive dosing on this sheet follow the ACVIM consensus statement. Cerebrovascular mechanisms and the clinical signs of neurologic target organ damage draw on the peer-reviewed canine cerebrovascular disease literature; referral criteria draw on the vestibular localization literature. All seven sources are peer-reviewed journal articles: one consensus statement, three reviews, and three clinical studies (30, 33, and 125 dogs). Hypothyroidism and diabetes mellitus appear in the consensus statement's own table of conditions causally associated with secondary hypertension; a case-control study additionally found dogs with atherosclerosis roughly 51 and 54 times more likely to have concurrent hypothyroidism or diabetes, which is a stroke pathway rather than a blood pressure one. The consensus recommends blood pressure evaluation every 6 months for dogs in known risk categories and does not recommend routine screening of otherwise healthy animals; the referral thresholds and the pairing of blood pressure with a senior panel before discharge are Canine Stroke Foundation practice recommendations built on those sources rather than a single published protocol. Every treatment decision must be individualized to the patient, the comorbidities present, and the clinical response.